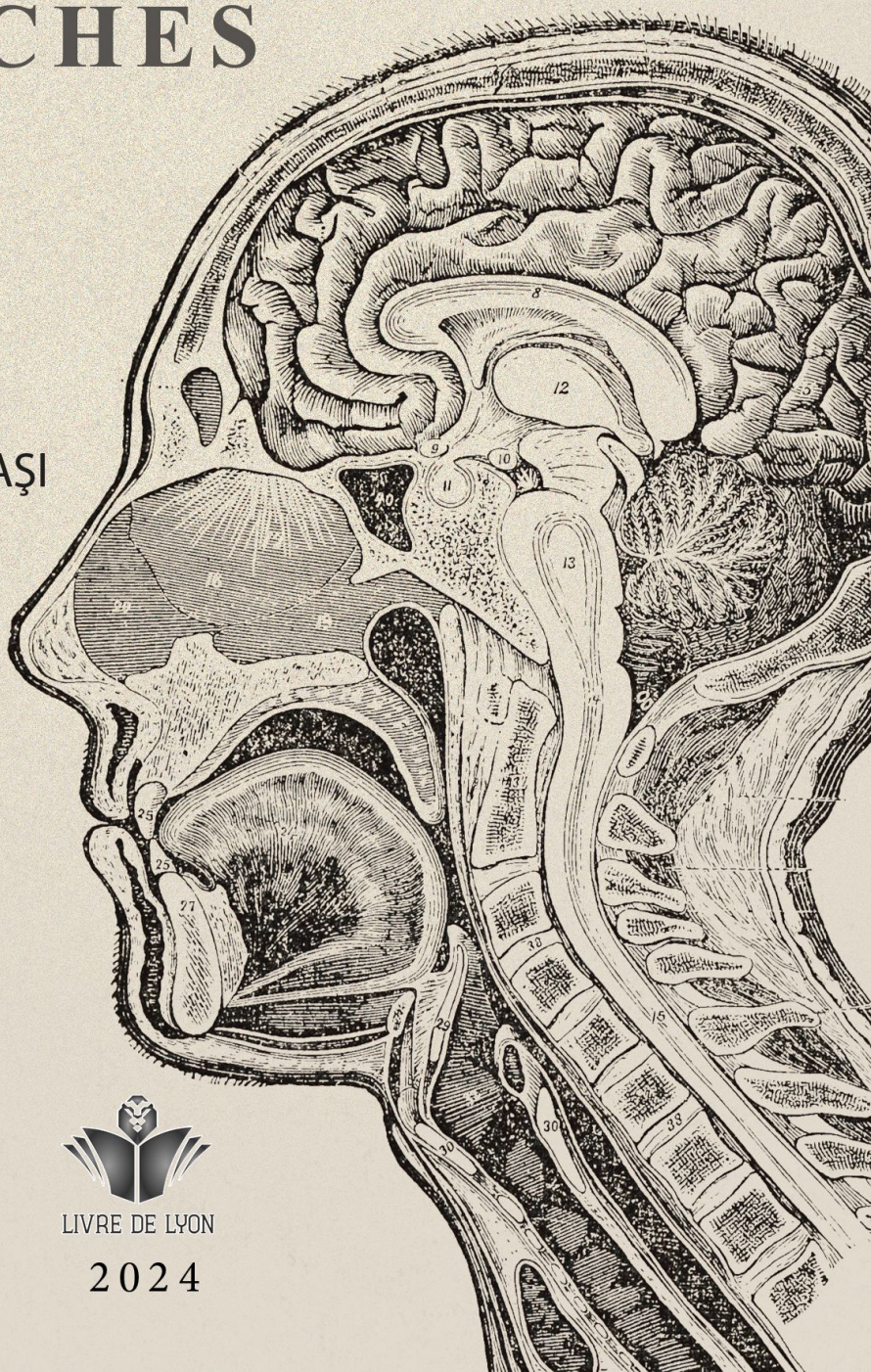
REVIEW OF
CLINICAL ANATOMY
RESEARCHES

Editors
Gülay AÇAR
Aynur Emine ÇİÇEKCİBAŞI



LIVRE DE LYON
2024

Anatomy

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Review of Clinical Anatomy Researches

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Cover Design • Motion Graphics
Book Layout • Motion Graphics

First Published • March 2024, Lyon

e-ISBN: 978-2-38236-662-2

DOI Number: 10.5281/zenodo.10875967

Publisher • Livre de Lyon
Address • 37 rue marietton, 69009, Lyon France
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PREFACE

Dear readers; In this review book, a total of 15 chapters are involved in different anatomic structures in relation to clinical and surgical anatomy, up-to-date and covering current studies. A comprehensive clinically oriented anatomical overview have a key role in making a diagnosis and treating a patient for clinicians and researchers. Each chapter consists of contemporary issues that offer a comprehensive anatomical review based on current studies. Through theoretical knowledge with detailed literature support, this book may contribute to the advancement of both academic researches and clinical applications. Each section emphasizes the anatomy, embryology, clinical significance, and the anatomic "pitfalls" commonly encountered by clinicians. The authors prepared this book with the awareness of the basics of clinical anatomy.

We hope that the scientific content of this book offers a valuable anatomical resource for medical students and researchers who want to make new studies. We would like to thank everyone (especially our authors) who contributed to the preparation of this book.

Editors

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CONTENTS

CHAPTER I

CLINICAL ANATOMY OF THE PANCREAS

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1. Introduction

The pancreas is an organ located in the posterior wall of the abdomen, extending transversely. It produces both endocrine and exocrine secretions and is situated retroperitoneally. While 1% of the pancreas consists of endocrine-secretion Langerhans islets, the remaining portion is composed of exocrine-secreting acinar cells, along with blood vessels, nerves, and extracellular tissue. The external secretion produced by acinar cells is emptied into the second part of the duodenum through a duct, known as the pancreatic duct. This external secretion, called saccus pancreaticus, contains enzymes such as amylase, lipase, and trypsin, facilitating the digestion of carbohydrates, fats, and proteins. The internal secretion of the pancreas is formed by the Langerhans islets and includes hormones that play crucial roles in carbohydrate, fat, and protein metabolism, such as insulin, glucagon, and gastrin (1,2,3).

A good knowledge of the anatomy of the region enhances the chances of success in surgical procedures involving the pancreas. Studies on anatomical relationships, the anatomy of pancreatic ducts, and variations will guide surgical treatments in this field.

2. Pancreatic Embryology

The pancreas begins as two bud-like structures from the portion of the endoderm lining the inner surface of the duodenum. Two buds, known as the dorsal pancreatic bud and the ventral pancreatic bud, are formed. The bud located at the back emerges earlier than the one in the front and rapidly develops between the leaves of the mesentery dorsale. The ventral pancreatic bud, on the other hand, passes near the papilla of the major duodenal papilla and develops between the leaves of the mesentery ventrale (4,5).

During development, the duodenum takes on a 'C' shape through a rightward rotation movement. Due to this movement, the ventral pancreatic bud, along with the ductus choledochus, shifts backward. Later on, it moves behind the dorsal pancreatic bud and merges with it. From the ventral pancreatic bud, parts of the uncinate process and the head of the pancreas develop. Finally, with the rotation of the stomach, duodenum, and mesentery ventrale, the pancreas assumes its position on the posterior abdominal wall (5).

With the rotational movement, the canals of the merging buds also anastomose with each other (5). The Wirsung duct is the main duct of the pancreas. This structure is formed by the junction of the distal part of the duct developed from the dorsal pancreatic bud and the duct developed from the ventral pancreatic bud. The proximal part of the duct from the dorsal bud either gets blocked or forms an accessory pancreatic duct called the Santorini duct. The Wirsung duct, along with the ductus choledochus, opens into the major duodenal papilla, and if present, the Santorini duct opens into the minor duodenal papilla (4).

3. Macroscopic Anatomy of the Pancreas

The location of the pancreas can be roughly described as behind the stomach and the lesser omentum. The pancreas is situated at the level of the L1-L2 vertebrae, extending from the curved portion of the duodenum to the spleen. Due to its transverse position, a portion of it is located in the epigastric and hypochondriac regions (1,3,6).

The pancreas is a secondary retroperitoneal organ. In embryonic development, the pancreas is initially found as an intraperitoneal organ. During the rotation movement that occurs during development, as the pancreas, along with the stomach and duodenum, turns to the right, the posterior leaf of the dorsal mesentery adheres to the parietal peritoneum and transforms into connective tissue. As a result, the posterior surface of the pancreas remains without peritoneum. Organs that later remain outside the peritoneum are referred to as secondary retroperitoneal (1).

Its thin capsule covering sends inward partitions, allowing the formation of a lobular structure. The color of the pancreas is between gray and pink. It is 12-20 cm in length, weighs 75-100 grams, and is slightly larger in males compared to females. Anatomically, the pancreas is examined in four parts: caput pancreatis, collum pancreatis, corpus pancreatis, and cauda pancreatis (2).

3.1. Head of The Pancreas

This portion of the pancreas is located within the curve formed by the first three segments of the duodenum. The protrusion extending to the left beneath the head of the pancreas is called the processus uncinatus. The pancreas is connected to the second segment of the duodenum by the pancreatic duct and vessels. It attaches to the entire curvature of the duodenal loop with peritoneum and connective tissue. Sometimes, the processus uncinatus separates from the pancreas and develops as the pancreas accessorius (Winslow). A notch called incisura pancreatis is present between the processus uncinatus and the body of the pancreas. Through this notch, the superior and inferior pancreaticoduodenal arteries pass, with an anastomosis occurring in the groove formed between the duodenum and the pancreas (1,2).

The pancreas is adjacent to a portion of the small intestine on the anterior side, along with the terminal branches of the gastroduodenal artery, namely the right gastroepiploic artery and the superior pancreaticoduodenal artery. The common bile duct (ductus choledochus) sometimes passes through a canal within the head of the pancreas, and at other times, it courses through a groove on the posterior surface. In cases where the common bile duct passes through the posterior surface, it neighbors the pancreas in the following order from right to left: common bile duct, hepatic portal vein, superior mesenteric artery, and right renal artery (1,3).

Due to the presence of the retropancreatic portion of the common bile duct behind the head of the pancreas, caution is required during pancreaticoduodenectomy. In the upper right part of the head of the pancreas, branches of the gastroduodenal artery are distributed, and just below it is the point where the peritoneum attaches to the transverse colon. Variations in vascular branching and peritoneal attachment points in this region during pancreaticoduodenectomy can lead to various surgical complications (3,7,8).

3.2. Neck of The Pancreas

It is the part located between the head (caput) and body (corpus) of the pancreas. It extends to the left in an anterior and upward direction. The neck

of the pancreas, known as collum pancreatis, is the shortest segment of the pancreas and is approximately 2 cm in length (1,2).

The anterior surface of the neck of the pancreas is adjacent to the pylorus and the gastroduodenal artery. The posterior surface is in proximity to the superior mesenteric vein and the portal vein. The portal vein is formed by the convergence of the superior mesenteric vein, the hepatic portal vein, and the splenic vein (2,6). While the neck of the pancreas is located in front of the superior mesenteric vein and the portal vein, the processus uncinatus is situated behind these venous vessels. A protrusion on the left side of the neck of the pancreas is referred to as tuber omentale (1,2).

3.3. Body of The Pancreas

The largest part of the pancreas, the "corpus pancreatis," extends from right to left and slightly upwards at the level of the L1-L2 vertebrae (1). When extracted from a fixed cadaver, the corpus pancreatis is likened to a triangular prism. Its anterior surface is adjacent to the posterior surface of the stomach, covered by the peritoneum that also covers the back of the lesser omentum. The edge between the anterior and inferior surfaces is referred to as the "margo anterior," where the transverse mesocolon attaches. The peritoneal leaflet forming the posterior surface of the transverse mesocolon also covers the inferior surface. The inferior surface of the corpus pancreatis is narrow and lies adjacent to the folds of the small intestine and the flexura coli sinistra. The posterior surface is without peritoneum and attaches to neighboring structures with connective tissue. Moving from right to left, the posterior surface is in proximity to the abdominal aorta, the beginning of the superior mesenteric artery, the left renal artery and vein, the crus sinistrum of the diaphragm, the left adrenal gland, and the left kidney. Additionally, the splenic artery and vein are adjacent to the posterior surface. The superior margin sometimes extends along the left side of the aorta. The superior margin is also adjacent to the celiac trunk and the celiac ganglia. A prominent area called "tuber omentale" is located on the right side of the superior margin, in proximity to the omentum minus (1,3,6).

3.4. Tail of The Pancreas

This portion of the pancreas is entirely covered by the peritoneum. It extends between the body (corpus pancreatis) and the hilum of the spleen. Typically, it reaches up to 3-4 cm to the left of the spleen. When it terminates before reaching the spleen, it attaches to the spleen through the ligamentum pancreaticosplenicum. Within this ligament, the splenic artery and vein

course. On the posterior side, it is adjacent to the left renal hilum. On the left, it is in proximity to the splenic hilum, and below, it is adjacent to the flexura coli sinistra. The cauda pancreatis contains more lobules compared to other parts (1,2).

3.5. Pancreatic Ducts

The pancreatic ducts are two channels that convey the exocrine (external) secretion of the pancreas to the duodenum (3).

3.5.1. Pancreatic Duct (Wirsung Duct)

The Wirsung canal extends from the tail of the pancreas to the head of the pancreas. It runs close to the posterior surface, branching off during its course. This canal serves as the main drainage channel of the pancreas, transferring the majority of the exocrine secretion of the gland to the duodenum, except for the lower part of the processus uncinatus and the head of the pancreas. In the head of the pancreas, it descends downward and backward, sometimes approaching the posterior surface before exiting, either before or after reaching the ductus choledochus. The descending part of the duodenum's wall runs alongside them, and they may sometimes merge to form an expansion called the ampulla hepatopancreatica (ampulla of Vater). Occasionally, they remain separate. Both of these ducts open into the major duodenal papilla. As they run within the wall of the duodenum, they cause folding in the duodenal mucosa known as plica longitudinalis (1,2).

The Wirsung canal starts as approximately a 1.5 mm diameter canal in the tail of the pancreas. Along its course, it receives contributions from 20-30 ducts, and by the time it reaches the body, its diameter is approximately 2.5 mm, increasing to about 3.5 mm in the head. It is about 15-20 cm long, and in its final part, it runs together with the ductus choledochus for 2-10 mm. They enter the second part of the duodenum from the posteromedial aspect, at a level where the duodenum is crossed by the transverse colon. Finally, these two ducts open into the major duodenal papilla (3,7-9).

The retropancreatic portion of the common bile duct has five types of variations (7,9):

1. Partially surrounded by unilateral pancreatic tissue (%42.5)
2. Completely surrounded by unilateral pancreatic tissue (%30)
3. The common bile duct runs without being surrounded by pancreatic tissue in its own groove (%16.5)

4. Completely surrounded by bilateral pancreatic tissues (%9)
5. Other variations (%2)

When the Wirsung canal and the common bile duct enter the wall of the duodenum, they narrow, and their diameters decrease to 1.4 mm and 3.4 mm, respectively. The septum that separates these two ducts disappears after a short course, with a 70% chance, resulting in the formation of a single common duct. The dilated distal part of this common duct is termed the ampulla hepatopancreatica (ampulla of Vater). The fold created by the ampulla hepatopancreatica in the duodenal mucosa is referred to as the major duodenal papilla. The plica longitudinalis duodeni, formed by segments of these two ducts in the duodenal wall, extends perpendicularly to the papilla. These folds are useful in identifying the papilla and are utilized in hepatobiliary interventions (1,3,7,8).

Ampulla of Vater variations in formation (7,10):

a. Common canal present, long/short ampulla, and a single orifice (%70-85)
b. Septum between the two ducts, no ampulla, and two orifices (%5-10)
c. No septum between the two ducts, no ampulla, and two orifices (%9-15).

There are sphincters composed of smooth muscles that control the passage of bile and pancreatic secretions into the duodenum. In 1957, Boyden identified four types of sphincters. The m. sphincter ductus pancreatici surrounds the distal part of the pancreatic duct; the upper and lower m. sphincter ductus choledochi surround the common bile duct; m. sphincter ampulla (Oddi sphincter) is around the ampulla of Vater. The Oddi sphincter is approximately 5-15 mm in length and has a resting pressure of 5-35 mmHg. Dysfunction of the Oddi sphincter can lead to recurrent pancreatitis (3,7,10).

3.5.2. Accessory Pancreatic Duct (Santorini Duct)

The Santorini duct develops from the dorsal bud of the pancreas during embryonic development. This duct carries exocrine secretion from the lower part of the processus uncinatus and the head of the pancreas (portions not drained by the Wirsung duct) and delivers it to the duodenum. In the head of the pancreas, this duct is connected to the pancreatic duct on the left and opens into the minor duodenal papilla on the right. In adults, the connection to the duodenum is usually severed (1-3).

During the rotational process of pancreas development, various canal anomalies may occur. For example, the anomaly resulting from the curved extension of the Santorini duct over the Wirsung duct is called ansa pancreatica (4,11).

4. Microscopic Anatomy of the Pancreas

The pancreas is a vital organ that produces both internal (endocrine) and external (exocrine) secretions. Its endocrine secretion primarily plays a role in regulating blood glucose levels. The exocrine secretion, transferred to the duodenum through pancreatic ducts, contains enzymes (amylase, lipase, trypsin) responsible for digesting carbohydrates, fats, and proteins (1,3).

Within the lobules formed by the connective tissue surrounding the pancreas, three types of cells are present: acinar cells responsible for exocrine secretion (85%), Langerhans islets that produce endocrine secretion (1-2%), and loose fibrous stroma (10%) that supports structures like interlobular vessels and nerves. The differentiation of endocrine and exocrine cells takes place during the intrauterine 12-14th weeks (4,12).

4.1. Pancreatic Cells with Exocrine Secretory Function

The exocrine secretory units of the pancreas are referred to as acini. The secretions contain enzymes responsible for the digestion of proteins, carbohydrates, and fats, as well as various ions (Na, K, Cl, Mg, HCO3). The daily volume of exocrine secretion is approximately 1500-2000 ml. The acinus structure consists of a lumen surrounded by a basal lamina supported by a reticular framework. The lumen is encircled by pyramid-shaped acinar cells, and the inner surface is lined by centroacinar cells (13,14).

Proteolytic enzymes produced by acinar cells, rich in granular endoplasmic reticulum, are stored by binding with calcium ions. The low calcium concentration in acinar cells, along with the pancreatic trypsin inhibitor (PTI) produced and the acidic environment, helps keep proteolytic enzymes in an inactive state (12,14).

Inactive proenzymes produced and secreted by acinar cells include:

1. Proenzymes involved in protein digestion (zymogens): trypsinogen, chymotrypsinogen, proelastase, procarboxypeptidase A and B, proribonuclease, prodeoxyribonuclease (14).

2. Enzymes involved in lipid digestion: lipase, phospholipase A2, colipase, cholesterol esterase, and the enzyme responsible for carbohydrate digestion, amylase, are secreted. Lipase and amylase are secreted as active enzymes (14).

Mutations in the gene responsible for the production of pancreatic trypsin inhibitor (serine protease inhibitor Kazal type 1), effective in inhibiting pancreatic trypsin, can lead to chronic pancreatitis in childhood. Trauma to the pancreas or blockage of the ducts increases pressure in the pancreatic ducts. As a result, proteolytic enzymes accumulate, and pancreatic trypsin inhibitor (PTI) becomes insufficient. Consequently, enzymes activated within the pancreas, along with cytokines such as interleukin-1 (IL-1) and TNF-α secreted in the pancreas, lead to the development of pancreatitis. These substances increase vascular permeability, disrupt circulation within the pancreas by causing edema and thrombosis in the blood vessels, contributing to pancreatitis (12-14).

Centroacinar cells are found only in the acini of the pancreas. These cells contain carbonic anhydrase and are effective in bicarbonate transfer. They are located within the lumen, surrounded by intercalated ducts as extensions. They secrete a large amount of fluid and HCO3 (14).

Intercalated ducts, like centroacinar cells, secrete a fluid rich in HCO3. These channels are short ducts covered with cuboidal epithelial tissue. The canal cells of the exocrine tissue constitute 4% of the pancreas in terms of weight and 10% in terms of cell count. These channels combine to form columnar intralobular ducts and interlobular ducts, ultimately opening into the Wirsung duct (12,14).

The amount of HCO3 in the fluid secreted by centroacinar and intercalated cells is 113 mEq/L, compared to 24 mEq/L in plasma. CO2 entering with the blood passes through the membranes of centroacinar and intercalated cells, resulting in the formation of HCO3 and H+. HCO3 is delivered to the lumen on the luminal side of the membrane, Cl- is taken into the cell, and on the basolateral side of the membrane, H+ is released into the blood, and Na+ is taken into the cell. The cystic fibrosis transmembrane conductance regulator (CFTR; 1450 amino acids) in the epithelial cells of the duct regulates Cl- exchange. CFTR also increases Na+ absorption from the basolateral Na/K ATPase pumps. In patients with cystic fibrosis pancreatic involvement, approximately 60-70% lack phenylalanine at position 508 in the synthesis of the CFTR protein. Due to the abnormal synthesis of the CFTR protein, there is insufficient Cl- exchange, and the secretion from acinar cells becomes dense and sticky, blocking the pancreatic ducts. Pancreatic insufficiency can occur when ≥80% of the pancreatic exocrine tissue is severely damaged due to acinar cell damage and fibrosis (12,15,16).

The exocrine pancreas has several stages in its secretion process:

The cephalic phase involves the parasympathetic effect (via the vagus nerve) on the secretion of enzymes by acinar cells. In this process, external

stimuli such as the smell of food activate the parasympathetic system, leading to the release of acetylcholine and the secretion of enzymes. This phase accounts for 20-25% of daily pancreatic secretion.

With the passage of food into the stomach, the gastric phase begins. The vago-vagal reflex initiates, leading to the secretion of enzymes from acinar cells. (This secretion constitutes approximately 10% of daily pancreatic secretion).

Subsequently, the intestinal phase begins, and during this phase, 65-70% of the daily pancreatic secretion occurs. The vagus nerve increases the enzyme secretion from acinar cells. Hormonal control in this phase is influenced by secretin and cholecystokinin (CCK). (13,14).

When the chyme with a low pH enters the duodenum, secretin is released from the 'S' cells in the duodenum and jejunum. This secretion reaches the pancreatic ducts through the bloodstream, stimulating the secretin receptors on the basolateral membrane. This results in a secretion that is rich in bicarbonate but weak in enzymes. The high-bicarbonate content of this secretion reaches the duodenum and neutralizes with the hydrochloric acid released from the parietal cells, producing carbonic acid (H2CO3) and sodium chloride. Thus, the acidic duodenum is raised to the pH necessary for the activation of pancreatic enzymes (10,14).

Proteins and long-chain fatty acids entering the duodenum activate the release of CCK (cholecystokinin) from 'I' cells. CCK travels to the pancreas through the bloodstream and, via phospholipase C, induces a secretion that is high in enzymes but low in bicarbonate (Fisher 2015, Townsend 2018, Köylü 2019). Additionally, insulin contributes to the increase in secretion, while glucagon, pancreatic polypeptide, and somatostatin lead to a decrease. VIP, gastrin, glucagon, and neurotensin have minimal effects on secretion (10,14).

The secreted CCK causes contraction in the gallbladder and bile ducts, relaxes the Oddi sphincter, and empties the exocrine secretion of bile and pancreas into the duodenum. The Oddi sphincter prevents the backflow of these secretions. Enterokinase released from the duodenum and jejunum activates trypsinogen to trypsin, which, in turn, activates other proenzymes. As trypsin levels increase, negative feedback inhibits the release of CCK and secretin (10,13,14).

4.2. The pancreatic cells responsible for endocrine secretion function

The pancreas, unlike other endocrine tissues, directly transfers its endocrine secretion produced in the Langerhans islets to the portal vein. Its secretion is responsible not only for the endocrine function but also for controlling the

exocrine secretion. Langerhans islets consist of cells with the APUD (amine precursor uptake and decarboxylation) characteristic, responsible for the secretion of different hormones, arranged around small capillaries. The islets are located among the acini, the structures responsible for exocrine secretion. There are approximately 1-2 million islets with a diameter of about 150-300 μm. Langerhans islets, originating from the endoderm in the intrauterine third month, start secreting insulin by the fifth month. Cells responsible for secreting glucagon and somatostatin develop from parenchymal cells. In the early postnatal period, Langerhans islets constitute about 10% of the pancreas. In adults, this ratio is 1-2% (4,12-14).

Langerhans islets are of two types: compact (90%) and diffuse (10%). The compact islets, mainly located in the body and tail sections, develop from the dorsal bud, and about 70-80% of them consist of beta cells. Alpha cells come second in number. Diffuse islets, on the other hand, develop from the ventral bud. They are mostly located in the lower part of the pancreas head, fewer in number, and larger in size. About 70-80% of them are composed of pancreatic polypeptide (PP) cells. Knowing which cell group is more concentrated in which region is useful in predicting complications that may occur after surgery (4,12).

The Langerhans islets, responsible for the endocrine function in the pancreas, have four types of cells with different functions. Alpha (A) cells, one of these types, secrete the glucagon hormone, leading to an increase in blood glucose levels. They are located around beta cells and constitute approximately 10-20% of islet cells. The most common cell group in Langerhans islets is the Beta (B) cells, constituting about 60-70% and located at the center of the islets. These cells produce insulin hormone, which lowers blood glucose levels, and store and release it. Additionally, they secrete C-peptide, proinsulin, and amylin. Another cell group is the Delta (D) cells, making up 3-10% of islets. These cells inhibit the secretion of insulin and glucagon with the somatostatin hormone they release. Gastrin hormone is also secreted from Delta cells. The fourth type of cell found in Langerhans is the PP (pancreatic polypeptide) cells, which secrete pancreatic polypeptide. Pancreatic polypeptide enters the blood after meals and regulates the release of digestive enzymes from the pancreas. In fasting conditions, it inhibits exocrine secretion (14).

The insulin hormone secreted by the Beta cells in the pancreas is released under parasympathetic influence. With the effect of insulin, glucose is converted into glycogen and stored in the liver. Under sympathetic influence, the secretion of glucagon from Alpha cells increases, leading to the conversion of previously stored glycogen into glucose (3).

The endocrine part of the pancreas is 5-10 times more vascularized than the exocrine part. Langerhans islets receive 20% of the blood flow reaching the pancreas, and blood flow in the islets occurs from the center to the periphery. Venous blood collects around the islets and is ultimately transferred to the portal vein. While 70-80% of the blood to the exocrine pancreas comes directly from the arteries feeding the pancreas, 20-30% comes from venous vessels from the endocrine part. This phenomenon is called the insuloacinar portal system and contributes to the regulation of secretion (17).

5. Pancreatic Arterial Blood Supply

The head of the pancreas, caput pancreatis, is supplied by the superior pancreaticoduodenal artery (a branch of the gastroduodenal artery) and the inferior pancreaticoduodenal artery (a branch of the superior mesenteric artery). The body (corpus pancreatis) and tail (cauda pancreatis) are nourished by branches called pancreatic branches originating from the splenic artery. The most well-vascularized part is the head of the pancreas, caput pancreatis. The arterial supply to the tail of the pancreas is relatively weak. The anastomosis between the superior pancreaticoduodenal artery from the celiac trunk and the inferior pancreaticoduodenal artery from the superior mesenteric artery, along with branches from the splenic artery, plays a crucial role in the blood supply to the concave part of the duodenum and the head of the pancreas. Approximately 40% of the branches on the posterior surface of the pancreas arise from the splenic artery, 22% from the celiac trunk, and 14% from the superior mesenteric artery (1,3,8,15).

The anastomosis between the anterior superior pancreaticoduodenal artery (from the common hepatic artery branch of the celiac trunk) and the anterior inferior pancreaticoduodenal artery (from the superior mesenteric artery) forms the Bühler arcade. Variations in the Bühler arcade and the vessels forming it can affect surgical interventions in the region. Understanding possible variations in the arteries in the region can reduce surgical complications. Studies on the variations of vessels forming the Bühler arcade are available in the literature (18,19). Table 1 and Table 2 summarize the origins of the anterior superior pancreaticoduodenal artery and the anterior inferior pancreaticoduodenal artery as identified in different studies.

Table 1: Origin of the Anterior Superior Pancreaticoduodenal Artery

Study and Year	Origin
Kageyama et al. 2016	Celiac Trunk
Schumacher et al. 2022	Common Hepatic Artery
Şelaru et al. 2020	Common Hepatic Artery
Abouzaid et al. 2023	Celiac Trunk
Padar et al. 2023	Celiac Trunk
Foyaka et al. 2019	Celiac Trunk
Ochoa et al. 2016	Celiac Trunk

Table 2: Origin of the Anterior Inferior Pancreaticoduodenal Artery

Study and Year	Origin
Venieratos et al. 2018	Middle Mesenteric Artery
Sanampudi and Raissi 2019	Middle Colic Artery

It is mainly drained by the splenic vein (v. splenica), and a portion of the venous blood is also drained by the portal vein (v. portae hepatis) and the superior mesenteric vein (v. mesenterica superior) (1).

6. The Lymphatic Circulation of The Pancreas

Lymphatic capillaries beginning around the acini primarily drain into the superior pancreatic lymph nodes, inferior pancreatic lymph nodes, and splenic lymph nodes. Additionally, the lymph nodes of the superior pancreaticoduodenal, inferior pancreaticoduodenal, and pyloric also contribute to the pancreatic lymphatic drainage. The collected lymph then drains into the celiac lymph nodes, hepatic lymph nodes, and superior mesenteric lymph nodes (1,3).

As is known, pancreatic cancers have a poor prognosis, attributed to their extensive lymphatic circulation network (3).

7. The Innervation of The Pancreas

The sympathetic and parasympathetic nerve fibers originating from the celiac plexus reach the pancreas through the splenic plexus. Sympathetic innervation is provided by the splanchnic nerves, which are postganglionic fibers. Parasympathetic innervation, on the other hand, is supplied by fibers from the vagus nerve. These fibers are preganglionic and synapse in ganglia located within the pancreas (1,3).

The activity of the pancreas increases under parasympathetic influence. Additionally, pancreatic secretions are also controlled by hormones (1,3). Pain

sensation from the pancreas's nociceptors is conveyed to the spinal cord through the splanchnic nerves. Referred pain is typically felt in the back (3).

8. The Surgical Procedure

In a living organism, the stomach, transverse colon, and omentum minus are located in front of the pancreas. Therefore, when the abdominal cavity is opened from the front, the pancreas cannot be directly seen. There are three different approaches to reach the pancreas in both living organisms and cadavers.

The first approach is achieved by cutting the omentum minus. The omentum minus is cut at the level of the curvatura minor, and when the stomach is pulled down, the corpus pancreatis can be visualized. The second approach can be made through the ligamentum gastrocolicum. The ligamentum gastrocolicum extends between the stomach and the transverse colon. If this ligament is cut, a significant portion of the pancreas can be seen. Ligamentum gastrocolicum forms the anterior two leaves of the omentum majus. Care should be taken to avoid damaging the arteries supplying the stomach during the cutting of this structure. With careful execution, this approach is the most suitable for living organisms. The third approach is made through the mesocolon transversum. The stomach and transverse colon are lifted upward, and then the mesocolon transversum is cut. In this way, the pancreas can be accessed. During this approach, attention should be paid to the branches of the arteria colica media, as damage to this artery can lead to necrosis in the transverse colon (1,3).

Another way to reach the pancreas is through the Kocher maneuver. With this method, the caput pancreatis, a large portion of the duodenum, pancreatic ducts, common bile duct, and even the nodi lymphatici pancreaticoduodenalis can be examined. Moreover, entering between the v. mesenterica superior and v. splenica with a finger allows for a complication-free resection (3,7,8).

9. Congenital Anomalies and Variations of the Pancreas

9.1. Portal Annular Pancreas

Caput pancreatis'in alt tarafında bulunan proc. uncinatus'un, v. portae hepatis ve v. mesenterica superior'u içine alacak şekilde pankreas'ın dorsal tarafı ile kaynaşması durumudur (27).

İlk olarak Suguira tarafından hipertrofik proc. uncinatus şeklinde tanımlanmıştır. Bu durum genellikle başka bir nedenle yapılan bilgisayarlı tomografi (BT) ile tesadüfen saptanır. İnsidansı %0,8 ile %2,5 arasında bildirilmiştir.

Portal anüler pankreas anomalisi, pankreas'ın ana kanalının v. portae hepatis'in önünden ya da arkasından geçmesine göre anteportal ya da retroportal olarak isimlendirilir. Benzer şekilde v. splenica'ya göre konumuna bağlı olarak da suprasplenik ve infrasplenik olarak isimlendirilir. Tedavisinde duodenum'u saran pankreas bölümünün rezeksiyonu pankreatit ve pankreas fistüllerine neden olabilir. Bu nedenle daha çok duodenojejunostomi tercih edilir (28,29).

9.2. Pancreatic Divisum

In this variation, in 60% of patients, the Wirsung and Santorini ducts merge, while in 30%, the Santorini duct becomes blind, and the Wirsung duct opens alone into the duodenum. In approximately 4-10% of cases, insufficient fusion of the ventral and dorsal buds of the pancreas results in the failure of the pancreatic ducts to merge, providing separate drainage. In this situation, the Santorini duct may be longer and more functional than the Wirsung duct (10,15).

Pancreatic divisum cases are generally asymptomatic. However, due to the longer Santorini duct draining into the smaller papilla duodeni minor, in the long term, complications such as pancreatitis and obstruction can occur due to increased back pressure. Additionally, idiopathic recurrent pancreatitis and other fusion anomalies may accompany pancreatic divisum (15,30).

9.3. Annular Pancreas

In the 7th week of intrauterine life, the abnormal rotation (counterclockwise) of the ventral bud of the pancreas, attached to the second part of the duodenum, partially or completely encircles the second part of the duodenum. The pancreatic tissue surrounding the duodenum may include the pancreatic duct. Anular pancreas can develop in two ways; extramural and intramural types.

In the extramural type, the pancreatic tissue and the pancreatic duct within it encircle the duodenum. Here, the duct papilla has not opened into the major duodenal papilla, leading to obstruction. In the intramural type, pancreatic tissue mixes with the fibers of the duodenum, opening directly into the duodenum with small ducts, and these patients exhibit duodenal ulceration.

This condition, which is asymptomatic in 70% of cases, leads to duodenal obstruction in the majority of symptomatic patients. It is observed in childhood in most cases and in one-third of cases in individuals aged 20-50. In pediatric patients, congenital deformities such as Down syndrome, esophageal, duodenal, and anal atresia accompany the condition in approximately 75% of cases (15,27,29).

9.4. Ektopik (Heterotopic) Pancreas

The pancreas is in a position outside its normal anatomical location. This ectopic structure is also called heterotopic or accessory pancreas and is observed in the population at a rate of 0.6-13.7%. Moreover, the detection rate in autopsies is also 0.5-14%. It is generally asymptomatic and shows a submucosal nodular arrangement. It protrudes towards the lumen and can cause complications such as hemorrhage, intussusception, and obstruction (11,31).

9.5. Dorsal Pancreatic Agenesis and Hypoplasia

A mutation in the PTF1 protein resulting in the varying degrees of absence of the dorsal bud of the pancreas characterizes a condition. Agenesis and hypoplasia of both the ventral and dorsal pancreatic sections together are extremely rare. This condition is usually accompanied by other issues, severe fetal growth restriction, and leads to neonatal death. In imaging, the portions of the pancreas other than the head adjacent to the duodenum are either missing or completely absent (30,31).

9.6. Abnormal Pancreatobiliary Junction

"Ortak Kanal Sendromu," Wirsung kanalı ve ductus choledochus'un birleşerek oluşturduğu kanalın 15 mm'den uzun olması durumu, genellikle konjenital koledok kisti ile birlikte %90-100 oranında görülür.

Pankreas salgısının ductus choledochus'a doğru reflüsü, safra yolu tümörlerinin sıklığını artırırken, Wirsung kanalına doğru geçmesi akut pankreatit insidansını (%31) artırır (30,31).

9.7. The accumulation of pancreatic fluid

After various surgical interventions on the pancreas, various complications can arise, sometimes leading to life-threatening situations. Peripancreatic fluid collection accounts for approximately 50% of the complications observed after pancreatic surgeries. This condition is caused by postoperative pancreatic leakage, leading to bleeding, necrosis, and abscess formation in adjacent vessels and organs. In cases of fluid collection, minimally invasive methods such as percutaneous drainage are often preferred over surgical methods. However, this method also requires external drainage post-discharge, thus being associated with a lower quality of life. Additionally, it has been mentioned in the literature that the percutaneous drainage method increases the risk of permanent pancreatic fistula.

In recent studies, attention has been drawn to endoscopic ultrasound-guided drainage for treatment. This method has low morbidity and is considered the best approach for the treatment of pancreatic cysts and fluid accumulation. It creates trauma in a limited area, preserves surrounding tissues, and shortens the hospital stay. Despite these advantages, its superiority over other treatment methods has not been proven, as there is no study comparing its results with other approaches and investigating its reliability. In a study comparing endoscopic ultrasound and percutaneous drainage methods, 264 articles and 695 cases were examined, and no significant difference in success rates between these two methods was observed (32).

9.8. Intrapancreatic Accessory Spleen

It is a congenital anomaly, observed in approximately 10% of the population. The most common location for an accessory spleen is at the hilum of the spleen, and the second most common location is in the tail of the pancreas (30).

9.9. The Variations in Pancreatic Contour

With aging, degeneration in pancreatic tissue leads to lobulations, especially in the head and neck regions, causing disruption of tissue contours and an irregular appearance. It is observed in approximately 34% of the population and may be perceived as a tumor. Based on the location in the head and neck region, it is classified into three types: Type I (anterior) is observed in 10%, Type II (posterior) in 19%, and Type III (horizontal) in 5%. On MR images, the tissue appears with normal density, but the contours of the tissue are irregular (31).

9.10. Pancreatic Steatosis

Obezite ya da ileri yaşa bağlı olarak gelişen yağlanma genellikle caput pancreatis'in ön bölümünde fokal veya diffüz olarak ortaya çıkar. Bu duruma "yağlı pankreas," "lipomatöz pseudohipertrofi," "non-alkolik yağlı pankreas hastalığı," "pankreatik steatoz" gibi farklı isimler verilmiştir. Pankreasın değişen durumları ve çeşitli anomalileri, bazı hastalıklarla yakından ilişkilidir. Örneğin; Tip 2 diabetes mellitus, akut pankreatit ve pankreas kanseri ile sık sık bağlantılıdır. Ayrıca Schwachman-Diamond sendromu ve kistik fibrozis durumunda hastalarda ciddi diffüz pankreatik lipomatosis gözlemlenmiştir. Pankreas yağlanması sonucu dokular, ultrason görüntülerinde böbrek ve karaciğer dokusundan daha yüksek ekojeniteye sahip olarak gözlemlenir (8,15,31).

10. Conclusion

When considering the functions of both internal and external secretion, the pancreas is a vital organ. It is deeply located, in close proximity to structures of the digestive system, and adjacent to the abdominal aorta and its main branches. There are common variations observed in the relationship between pancreatic ducts and bile ducts. These variations play a crucial role in the diagnosis of both benign and malignant conditions of the pancreas. Additionally, there are various variations in the pancreatic ducts and arteries that supply the pancreas. Particularly during pancreaticoduodenectomy, potential variations in the vessels forming the Bühler arcade should be taken into consideration. In conclusion, a thorough understanding of its normal function, anatomical location, and structure will significantly reduce surgical complications, morbidity, and mortality.

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CHAPTER II

THE ANATOMY AND CLINICAL IMPORTANCE OF THE PERICARDIUM

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1. Introduction

The pericardium is an important structure that surrounds and protects the heart. Consisting of two layers, this membrane not only shields the heart from external influences but also limits and regulates its movements. The pericardium comprises two main parts: the inner layer, called the pericardium serosum, which surrounds the myocardium with a thin membrane, and the outer layer, called the pericardium fibrosum. Between them lies a space called the pericardial cavity, which contains pericardial fluid, allowing for frictionless movements of the heart. The pericardium can be affected as part of heart diseases or can cause various conditions on its own, such as pericarditis. Therefore, a better understanding of the structure, function, and potential diseases of the pericardium plays a significant role in cardiovascular diagnosis and treatment processes. Therefore, understanding topics such as the anatomy, diseases, imaging methods, and treatment options of the pericardium is of vital importance surgically. (1,2)

2. Embryology of the Pericardium

2.1. Formation of the Pericardium

The pericardium is a membranous structure surrounding the heart and forms during embryonic development. Embryologically, the formation of the pericardium is a complex process. In the early stages of embryonic development, the division of the zygote cell results in three embryonic layers: ectoderm, mesoderm, and endoderm. The mesoderm layer forms the middle layer of the embryo. Within the mesoderm lies a specific region called the cardiogenic mesoderm, which is crucial for the development of the heart. The cardiogenic mesoderm gives rise to a structure known as the heart tube. During the development of the heart tube, surrounding mesoderm cells organize to form the pericardial cavity. Spaces develop among these mesoderm cells, creating the pericardial cavity. This cavity forms the foundation of the pericardium and protects the heart from external influences. As the embryo progresses, the development of the pericardium continues, eventually forming a complex membranous structure consisting of various layers. (3)

2.2. Formation of the Transvers Sinus and Oblique Sinus

With the degeneration of the mesoderm cells surrounding the heart tube, a transitional area within the pericardial cavity called the transvers sinus is formed. The transvers sinus remains as a distinct space between two regions where the epicardium transitions to the pericardial covering, between the entrance and exit pathways in the adult heart. The oblique sinus, on the other hand, arises from the anatomical repositioning of the venous entrance pathway upward and to the right. (3)

3. Anatomy of the Pericardium

The pericardium is a serofibrous sac. This sac is described as cone-shaped, encompassing the entire heart and the root portions of the great vessels. This particular structure resides within the mediastinum medius region, behind the body of the sternum and the 2nd to 6th costal cartilages, as well as in front of the 5th to 8th thoracic vertebrae. The apex of the pericardium corresponds to the arch of the aorta, while its base aligns with the upper surface of the diaphragm. (4,5)

3.1. Layers of the Pericardium

The pericardium consists of two distinct layers. The outer layer, named the fibrous pericardium, is characterized by its fibrous structure, whereas the inner layer, known as the serous pericardium, possesses a serous structure. The

fibrous pericardium covers the outer surface of the pericardial cavity, providing stability, while the serous pericardium lines the inner surface of the pericardial cavity, creating a smooth surface and allowing the heart to move freely. (4,5)

3.1.1. Fibrous Pericardium

The fibrous pericardium is a thick, sturdy, and matte-looking membrane layer that surrounds the roots of the heart and major vessels. This structure, which protects and supports the heart, is composed of dense connective tissue and serves to stabilize the position of the heart and prevent excessive expansion. It has an average length of 13 cm, a width of 8 cm at the apex, and 14 cm at the base. Its average volume ranges from 500-800 cm^3 and can reach a volume of 1000-1200 cm^3 when expanded. This layer limits the heart's filling and sudden expansion due to its lack of flexibility and its adjacency to the large vessels entering from its upper part. It is traversed by the aorta, pulmonary artery, pulmonary veins, and superior vena cava at its apex. Its base is influenced by the movements of the diaphragm due to its union with the central tendon of the diaphragm. (3,4,5)

The inferior vena cava is a structure that is part of the pericardial sac and comes into contact with the pericardium in an area close to the posterior surface of the heart. This point of contact contributes to the stability of the heart and major vessels and helps maintain the integrity of the pericardium. However, the inferior vena cava is not directly enveloped by the fibrous pericardium; therefore, there is no direct connection between the pericardial cavity and the inferior vena cava. The inferior vena cava enters the heart from the posterior aspect of the pericardium and is partially covered by the pericardial sac at its entry point. (3,4,5)

Regarding the neighboring structures of the fibrous pericardium, it is reported to be closely related to the sternum anteriorly, the thoracic aorta, oesophagus, main bronchi, and the mediastinal surfaces of the lungs posteriorly, and the anterior margins of the 2nd to 6th costal cartilages, pleura, and the anterior edges of the lungs laterally. The thickness of the fibrous pericardium varies from 0.8 to 1 mm depending on its location, although it may appear slightly thicker on imaging. (3,4,5)

3.1.2. Serous Pericardium

The serous pericardium consists of two layers. The outer layer is called the parietal layer, while the inner layer is called the visceral layer (epicardium). (3,4,5)

The parietal layer is a shiny serous membrane that lines the inner surface of the fibrous pericardium and, together with the pericardium, is referred to as the external pericardium. Studies have reported that the visceral layer, which covers the heart, continues onto the inner surface of the fibrous pericardium, forming the parietal layer, particularly where it surrounds the major vessels. (3,4,5)

The parietal layer is easily visualized on imaging, usually delimited by epipericardial and epicardial fat. Studies have shown that in newborns, epicardial fat tissue is absent, and it tends to develop along the atrioventricular and interventricular grooves with age. (5)

The visceral layer, also known as the epicardium, is a thin layer that covers the surface of the myocardium. The smoothness of the heart's outer surface is ensured by the thin connective tissue layer between the epicardium and myocardium. (3,4)

3.2. Pericardial Cavity

The space between the visceral layer and parietal layer of the serous pericardium is called the pericardial cavity. This space, which is a narrow gap, contains a fluid called pericardial fluid, with a volume ranging from 20 to 30 cm^3. This fluid reduces friction that may occur during the contraction of the heart. It helps the heart to function smoothly and prevents unnecessary energy loss. Additionally, the fluid forms a protective layer around the heart, shielding it from external factors and serving as a buffer against potential injuries. Another function of the fluid is to serve as an ultrafiltrate plasma from the capillaries of the epicardium and parietal layer. (3,4,5)

In cases of pericardial diseases, the volume of fluid can increase to 250 cm^3 or even 500 cm^3. When the fluid in the pericardial cavity increases excessively, the fibrous membrane cannot expand, resulting in pressure on the heart. In a supine position, most of the fluid accumulates in the aortic reces and transversus sinus. (3,4,5)

3.3. Pericardial Sinuses

There are three sinuses identified within the cavitas pericardialis. These are the superior sinus, transvers sinus, and oblique sinus. (6)

3.3.1. Sinus Superior

The superior sinus, located just above the transvers sinus, is referred to as the aortic recess superior by anatomists while radiologists term it as the superior sinus. There are various definitions regarding the nomenclature of this sinus in

the literature. The superior sinus extends along the ascending aorta to the level of the pulmonary artery. The transvers sinus, joining the superior sinus behind the aorta on the right side, extends to the level of the aorta. (6)

3.3.2. Transvers Sinus (Thiele Canal)

The transvers sinus is a space located behind the pericardium, between the aortic arch, the roots of the pulmonary arteries, and the upper portion of the right atrium. This space lies behind and above the pericardium and typically appears triangular when viewed externally. Its dimensions may vary from person to person but are generally described as sufficiently wide. The transvers sinus is significant during cardiac surgery because it is located near the aortic root and pulmonary arteries, requiring careful attention to this area. When procedures involving the aortic root or pulmonary arteries are performed during cardiac surgery, careful examination and, if necessary, manipulation of this space are important. The transvers sinus is an important anatomical region of the pericardium and plays a critical role in the arrangement of major structures around the heart. (3,4)

3.3.3. Oblique Sinus

The oblique sinus (Haller's cul-de-sac) is a space located in the posterior part of the pericardium, between the upper parts of the right atrium and the left atrium. It is the largest and widest cavity of the pericardium and possesses significant anatomical features. The oblique sinus typically appears oval when viewed externally. Its dimensions may vary from person to person but are generally described as quite wide. The shape of the oblique sinus can range from triangular to square depending on the symmetry and depth of the indentations of the pulmonary veins. This cavity allows the heart to move freely during respiration and cardiac movements. The oblique sinus typically appears oval when viewed externally. Its dimensions may vary from person to person but are generally described as quite wide. This cavity allows the heart to move freely during respiration and cardiac movements. (3,7)

From the left pulmonary artery to the left superior pulmonary vein, there extends a pericardial fold in a triangular shape called the plica venae cavae superioris sinistrae. Beneath this fold lies the remnant of the oblique left common cardinal vein. This remnant extends backward to the left atrium and is called the lig. venae cavae sinistrae (Marshall ligament), continuing with the v. obliqua atrii sinistri (Marshall vein). Dissections of adult cadavers have reported that within the Marshall ligament, there is myocardium and neural tissue, with

Marshall's vestigial fold absent in about 7% of bodies, and in approximately 13% of cases, an oblique vessel has been detected. (8)

3.4. Pericardial Recesses

The pericardial cavity has three recesses: the postcaval, left pulmonary, and right pulmonary recesses. The shapes, depths, and widths of these recesses vary. (6)

3.4.1. Postcaval Recess

The postcaval recess is observed as a lateral extension of the superior sinus. This recess, extending around the superior vena cava, is bordered by the right pulmonary artery superiorly and the right upper pulmonary vein inferiorly. In the literature, the average depth of this recess is reported to be 17 ± 4.3 mm. (6)

3.4.2. Pulmonary Venous Recesses

Pericardial recesses extend between the superior and inferior pulmonary veins. In a study involving fresh cadaver dissections, it was reported that the pulmonary recesses were symmetric in 48% of cases, present bilaterally in 56% of cases, and absent bilaterally in 10% of cases. The average depth and width were measured as 19.5 mm and 18.83 mm, respectively, in the left recess, and 12 mm and 16 mm, respectively, in the right recess. Additionally, in 5% of the cases included in the study, an acquired anomaly was observed around the right inferior pulmonary vein. (6,7)

3.5. Ligaments of the Pericardium

The fibrous layer of the pericardium contains various ligaments that extend from the sternum, diaphragm, and vertebral column. The ligament extending between the posterior aspect of the sternum and the pericardium fibrosum is called the superior sternopericardial ligament, while the ligament extending in the lower portion is termed the inferior sternopericardial ligament. The phrenicopericardial ligament extends between the fibrous pericardium and the central tendon of the diaphragm, while the vertebropericardial ligament extends between the prevertebral fascia covering the vertebral column. (3)

3.6. Vessels of the Pericardium

The arterial circulation of the pericardium is supplied by the coronary vessels in the visceral layer, while in other layers, it is provided by the pericardiacophrenica artery and musculophrenica artery, branches of the internal

thoracic artery. In venous circulation, the veins primarily drain into the azygos vena, but they also open into the pericardiacophrenica vena and thoracica interna vena. The pericardium is associated with certain lymph nodes, including the nodi prepericardiaci and nodi prepericardiaci laterales, while its lymphatic drainage mainly directs towards the nodi lymphatici tracheobronchiales. (3)

3.7. Innervation of the Pericardium

The parietal layer of the fibrous pericardium and serous pericardium is innervated by the phrenic nerve, while no nerves are found in the visceral layer. Pain originating from this area is transmitted by the phrenic nerve. Parasympathetic innervation comes from the vagus nerve and the left recurrent laryngeal nerve, as well as branches of the oesophageal plexus. Sympathetic innervation is provided by the first dorsal ganglion, stellate ganglion, and the aortic, cardiac, and diaphragmatic plexuses. (4,5)

3.8. The Functions of the Pericardium

The pericardium performs various crucial functions such as protecting, stabilizing, and regulating the function of the heart. It shields the heart from impacts and blows to the chest region, preventing damage. Additionally, it prevents excessive expansion of the heart, thus averting overfilling and excessive enlargement. Between the two layers of the pericardium lies a small amount of fluid called pericardial fluid. This fluid reduces friction between the two pericardial surfaces during the heart's expansion and contraction, facilitating smooth heart function. It also stabilizes the heart's position within the chest cavity, ensuring it remains anatomically correct and preventing excessive movement, thereby enabling efficient blood pumping. Moreover, the pressure of the pericardial fluid can affect the heart's filling dynamics. The pericardium regulates pressure relationships between the heart and major blood vessels, assisting in effective blood pumping. Acting as a barrier, the pericardium prevents infections from reaching the heart, contributing to its protection against infections. These functions of the pericardium support the healthy functioning of the heart and contribute to the effectiveness of the body's overall circulatory system. (9,10)

4. Pericardial Syndromes

Pericardial diseases are frequently encountered in clinical practice and can have high mortality and morbidity rates in terms of incidence. These diseases may occur congenitally or may develop later, either in isolation or in association

with certain systemic illnesses. Some forms of pericardial diseases are critically important due to the disruption of hemodynamic balance and require urgent intervention. (11)

4.1. Pericarditis

Pericarditis is the inflammation of the layers of the pericardium. Depending on its duration, it can be classified into four different types: acute, incessant, recurrent, and chronic. If pericarditis lasts between 4-6 weeks, it is classified as acute; if it persists without remission for 4-6 weeks, it is termed incessant; if symptoms reappear after a symptom-free period of 4-6 weeks, it is categorized as recurrent; and if pericarditis persists for more than 3 months, it is considered chronic. (12)

It is reported in the literature that approximately 20-30% of acute pericarditis cases progress to recurrent pericarditis. Of these recurrent cases, about 50% eventually present as chronic pericarditis upon returning to the clinic. (12)

4.1.1. Acute Pericarditis

Acute pericarditis is the most commonly encountered pericardial disease in clinical practice. The etiology of the disease includes infectious and non-infectious conditions. Approximately 80-85% of all pericarditis cases are attributed to infectious causes when considering all pericarditis cases in the literature. Furthermore, it is reported that about 40% of patients diagnosed with infectious acute pericarditis have viral pericarditis. Therefore, an increase in pericarditis cases associated with common colds during the winter months is observed. (13,14)

Besides viral infections, other factors such as autoimmune diseases, renal failure, or chest trauma can also lead to acute pericarditis. (15)

There is reported variation in the etiologies of acute pericarditis between developed and developing countries. In developing countries, tuberculosis is found to be the most common cause of acute pericarditis. In developed countries, it has been identified that certain surgical interventions such as cardiac surgery, pacemaker implantation, or aortic valve transplantation can lead to acute pericarditis. (12,15)

To establish the diagnosis of the disease, at least two of the following conditions are necessary: chest pain, pericardial friction rub, electrocardiogram changes, and pericardial effusion. The hallmark symptom is chest pain, which typically presents as sudden onset and is often described by patients as a sharp or dull pain, alleviated by sitting up or leaning forward. Other

accompanying symptoms of chest pain include fever, fatigue, and shortness of breath. (12,15)

4.1.2. Constrictive Pericarditis

Constrictive pericarditis is characterized by the stiffening and thickening of the pericardium, leading to restriction of cardiac filling. This condition represents the final stage of chronic inflammation, affecting the diastolic filling of the heart. Pathological changes in constrictive pericarditis typically affect the parietal layer. However, over time, these changes can extend to the visceral layer and epicardium, leading to adhesion formation between the parietal layer and epicardium. (16,17)

There are three different types of constrictive pericarditis: transient constrictive, chronic constrictive, and effusive constrictive. Transient constrictive pericarditis is a type that occurs as a result of inflammation and may resolve spontaneously or with treatment. Chronic constrictive pericarditis occurs due to chronic inflammation and fibrosis, leading to fibrosis and calcification. Effusive constrictive pericarditis is characterized by the presence of pericardial effusion along with constriction of the visceral layer. (18)

Constrictive pericarditis impairs the normal function of the heart, affecting the circulatory system and may lead to symptoms of heart failure. Typical symptoms include jugular vein distention due to increased pressure, shortness of breath, especially leg swelling, fatigue, and abdominal distension. Chest pain is typically less prominent. Diagnosis is made based on clinical findings along with imaging tests such as echocardiography, magnetic resonance imaging, and sometimes cardiac catheterization. Treatment typically involves surgical pericardiectomy, which involves removal of the stiffened pericardial tissue surrounding the heart, improving cardiac filling. (17,18)

4.2. Pericardial Effusion

Pericardial effusion is the abnormal accumulation of fluid within the pericardial space surrounding the heart, and it is one of the commonly encountered pericardial syndromes in clinical practice. The formation of effusion can result from various pathologies affecting the production or drainage of pericardial fluid. Most studies in the literature report that pericardial effusion occurs in association with pericarditis. There are also studies indicating that it can develop as a consequence of various systemic diseases such as heart, kidney, or liver failure, as well as trauma, inflammation, myocardial infarction, among others. (19,20,21)

The signs and symptoms of effusion generally depend on the size and rate of accumulation. In mild effusions, symptoms may be minimal or absent, but large or rapidly increasing effusions can lead to symptoms such as chest pain, shortness of breath or difficulty breathing, tachycardia, and prominent physical findings such as swelling or fullness in the neck veins. The diagnosis of pericardial effusion is typically based on the patient's symptoms, physical examination findings, and imaging tests (e.g., echocardiography, computed tomography, or magnetic resonance imaging). (19,20,21)

Treatment varies depending on the cause and severity of the effusion. Mild effusions are generally asymptomatic and do not require treatment. However, if symptoms are present or if the effusion indicates an underlying condition that needs to be treated, intervention may be necessary. Treatment options include pericardial fluid drainage, medication (e.g., antibiotics for infections), treatment of the underlying disease, and rarely surgical intervention. (19,20,21)

4.3. Pericardial Cyst

Pericardial cyst is a rare condition that occurs within the pericardial cavity. These cysts typically arise as a result of a congenital anomaly and can be defined as a fluid-filled sac within the pericardium. However, in rare cases, other factors such as infections, trauma, or inflammatory diseases can also lead to the formation of pericardial cysts. Cysts are often small and asymptomatic, and they are most commonly detected incidentally during other medical evaluations through radiological imaging tests. However, large cysts can cause symptoms by compressing the heart. These symptoms may include chest pain, shortness of breath, palpitations, and rarely a feeling of fainting. Pericardial cysts are often observed in the right cardiophrenic angle. The presence of a pericardial cyst in a different area other than this region poses a diagnostic challenge in distinguishing it from other intracardiac or mediastinal abnormalities. (22,23)

Small and asymptomatic cysts are typically monitored and do not require treatment. However, cysts causing symptoms or complications may require treatment. Treatment options may include surgical removal of the cyst, drainage of the pericardial cavity, or endoscopic intervention. (22,23)

4.4. Pericardial Diverticulum

Pericardial diverticulum resembles a pericardial cyst in terms of various histological and radiological findings. One of the differences between the two is that a pericardial diverticulum establishes a connection with the pericardial cavity. Another distinguishing feature is that the size and shape of the pericardial

diverticulum change with various body movements and respiration, while in a pericardial cyst, the shape changes while the size remains constant. Both conditions are generally asymptomatic. (24)

4.5. Pericardial Tamponade

Pericardial tamponade is a condition characterized by an abnormal accumulation of fluid, blood, or gas in the pericardial cavity to the extent that it disrupts the normal function of the heart. It is a life-threatening clinical syndrome that increases pericardial pressure to the point of reducing cardiac output, necessitating urgent intervention. There are many causes of this clinical condition, including trauma, cardiac surgeries, myocardial infarction, cancer, infections resulting from pericarditis, and certain systemic diseases. Symptoms may include chest pain, shortness of breath or respiratory distress, tachycardia, jugular vein distention or swelling, hypotension, dizziness, or fainting sensation. (19,25)

The capacity for expansion of the pericardial cavity and pericardium is limited, hence the symptoms vary depending on the volume or rate of accumulation of the substance. A small amount of accumulation can lead to pericardial tamponade, while a larger accumulation may be asymptomatic by not affecting ventricular filling. The goal of treatment is to reduce the compression on the heart by draining the effusion. In cases of rapid development of effusion with a small amount of accumulation, surgical drainage is necessary. In cases of larger and slower-developing effusions, drainage can be performed using a subxiphoid approach or by placing a catheter percutaneously. (19,25)

4.6. Pericardial Congenital Anomalies

Pericardial congenital anomalies refer to a condition where the pericardium is completely or partially absent. It is rare, with an incidence rate reported to be approximately one in every 10,000 autopsies. (6) Due to its asymptomatic nature, it can be confused with various clinical conditions such as mitral valve disease, cardiac aneurysms, acute coronary syndromes, and various tumors in the lungs or heart. (10) Although most congenital anomalies do not show any symptoms, they can lead to serious health problems such as ventricular herniation and sudden death. Therefore, the importance of pericardial defects cannot be overlooked. (23)

In cases reported in the literature, approximately 70% have been left-sided partial defects and 17% have been right-sided, with complete absence of the pericardium reported in 9% of cases. Some studies have shown that in 30% of

cases, other congenital anomalies such as atrial septal defects, bronchogenic cysts, and bicuspid aortic valves accompany pericardial defects. (6)

In cases where the pericardium is partially or completely absent, no treatment is necessary. Some studies in the literature have reported similar ejection fractions in patients with pericardial absence compared to those with intact pericardia. Some authors claim that this congenital absence could pose a risk for traumatic aortic dissection. (10)

Studies in the literature indicate that variations in pericardial arteries and veins are rare, but pericardial venous varices have been reported in a few patients with superior vena cava and azygos vein occlusion. The visceral pericardium is more vascular compared to the parietal pericardium. Therefore, in a case of pericardial varices, significant bleeding may occur as a result of any catheter manipulation within the pericardial cavity. (6)

5. Imaging Methods in Pericardial Diseases

5.1. Chest X-Ray

Chest X-ray is the most commonly preferred method for imaging pericardial diseases. However, it is not sufficient alone for the definitive diagnosis of the disease. Additional imaging and diagnostic methods such as echocardiography, computed tomography (CT), and magnetic resonance imaging (MRI) may be required for a conclusive diagnosis. (21)

Chest X-ray is the initial imaging method preferred to detect conditions such as pericardial calcification, which may occur in chronic and constrictive pericarditis, presence of cardiomegaly, and various pleuropulmonary diseases such as lung cancer, tuberculosis, pneumonia, and pleural effusion, in cases where pericarditis or pericardial effusion is suspected. (21)

In cases where pericarditis is diagnosed but there is no structural heart disease present, chest X-ray images appear normal. If the amount of pericardial effusion is more than 300 mL, chest X-ray images may show an increase in the silhouette of the heart and a bottle-shaped appearance. (21)

5.2. Echocardiography

Echocardiography is often preferred as a cost-effective and easy-to-implement imaging method for the diagnosis of various pericardial diseases, especially constrictive pericarditis, pericardial effusion, pericardial defects, cardiac tamponade, or pericardial cysts. Among these diseases, echocardiography is considered the standard diagnostic method for pericardial effusion. With significant advancements in recent years, echocardiography has also seen some

developments. Among these, 3D echocardiography is particularly valuable in the diagnosis of pericardial diseases due to its more accurate detection of pericardium and surrounding anatomical structures compared to 2D echocardiography. Additionally, 3D echocardiography can determine the most suitable area for certain interventions such as pericardiocentesis. (21,26)

5.3. CT

In CT scans, the normal pericardium is observed as a thin fibrous line, surrounded by dense mediastinal and epicardial fat layers. To visualize the pericardium more clearly and separate it from these fat layers, a higher amount of X-rays should be used in CT scans. Additionally, CT images of the pericardium clearly show pericardial sinuses and recesses. Some studies report the thickness of the pericardium in CT scans to be between 0.7-2 mm. (21,27,28)

CT is particularly valuable for the accurate and detailed visualization of calcification in the pericardium. CT scans performed before pericardiectomy provide useful information regarding the extent of calcification. Furthermore, CT can provide information on whether pericardial fluid is transudative or exudative. Congenital complete or partial absence of the pericardium, pericardial tumors, and masses can also be easily detected with CT scans. (21,27,28)

5.4. MRI

In the diagnosis and evaluation of pericardial diseases, MRI plays an important role. MRI can visualize the heart and surrounding tissues in detail, providing important information for diagnosing pericardial diseases, assessing their severity, and guiding treatment. The normal structure of the pericardium appears similar to CT on MRI. In T1-weighted MR images, a thin, less dense, and dark-colored line is observed between dense mediastinal and epicardial fat layers. Its thickness is typically reported to be less than 2 mm. (21,27,28)

MRI can evaluate structural changes in the pericardium and the accumulation of fluid in the pericardial space (pericardial effusion). Additionally, it is valuable for determining whether the pericardium is infected, assessing the degree of inflammation, and detecting pericardial masses. Specifically, certain techniques used in MRI (e.g., contrast-enhanced imaging) can further assist in characterizing pericardial diseases. (21,27,28)

MRI can also aid in evaluating the clinical symptoms of pericardial diseases such as constrictive pericarditis and monitoring the course of the disease. Furthermore, MRI can be used to assess the patient's condition before and after pericardial surgery. (21,27,28)

5.5. Positron Emission Tomography (PET)

PET, especially using 18-fluorodeoxyglucose (FDG), is a type of nuclear medicine imaging method that visualizes metabolic activities in the body. FDG is used as a tracer to monitor how cells utilize glucose because active cells, particularly rapidly growing cancer cells, consume large amounts of glucose. (21,27)

In the evaluation of pericardial diseases, PET imaging, particularly with FDG, can be useful in the diagnosis and management of inflammatory or neoplastic pericardial diseases. Inflammatory processes typically show mild to moderate FDG uptake, while neoplastic diseases may exhibit significantly increased FDG uptake. This difference can be important in understanding the nature of pericardial disease and especially in distinguishing malignant conditions. (21,27)

PET can help determine the etiology of pericardial disease, evaluate response to treatment, and potentially predict the prognosis of the disease. However, PET scanning is usually used in conjunction with other imaging methods such as echocardiography, CT, MRI, as these methods provide better anatomical details and help correlate PET findings with anatomical structures. This integrated approach is particularly important in the evaluation of complex pericardial diseases. (21,27)

6. Interventional Procedures for the Pericardium

Interventional procedures performed on the pericardium play a significant role in the diagnosis and treatment of pericardial diseases. These interventions vary from obtaining a sample for analysis of pericardial fluid (pericardiocentesis), creating a window to provide permanent access to the pericardial space (pericardial window), to removing a part of the pericardium (pericardiectomy) to reduce its restrictive effects. (9,29)

6.1. Pericardial Window

The pericardial window approach is a surgical procedure used for managing recurrent pericardial effusions. This method creates a permanent opening in the pericardium to facilitate the absorption of pericardial fluid by the body and prevent complications caused by pericardial effusion. The pericardial window is preferred especially in cases where effusion is recurrent or cannot be completely drained by pericardiocentesis. Through this surgical technique, biopsy of pericardial tissue can also be performed. (9,29)

There are different methods for creating a pericardial window, with the most commonly used ones being the subxiphoid and transpleural approaches. (9,29)

The subxiphoid approach involves making a small incision just below the xiphoid process at the lower end of the sternum. The surgeon then reaches the pericardium through this incision and creates a small opening in the pericardium. This opening allows continuous drainage of pericardial fluid. This approach is typically used in the treatment of postoperative or infection-related pericardial effusion. (9,29)

In the transpleural approach, pericardial drainage is achieved using thoracoscopic instruments after making a small incision. This approach can be performed in two different ways: anterolateral thoracotomy or thoracoscopic (video-assisted thoracic surgery). When comparing the two, the thoracoscopic approach allows for faster patient recovery, while the anterolateral approach provides a larger access area to the chest wall. If pericardial biopsy or other procedures related to the heart are needed, the anterolateral approach may be preferred. (9,29)

6.2. Pericardiocentesis

Pericardiocentesis is a medical procedure performed to remove an abnormal accumulation of blood or fluid in the pericardial cavity. This procedure aims primarily to determine the presence, cause, and nature of pericardial fluid. It is performed in cases of symptomatic pericardial effusion other than posterior pericardial effusion and conditions like cardiac tamponade. (21,30)

The standard practice for pericardiocentesis is performed under local anesthesia with the assistance of fluoroscopy, echocardiography, or CT scan. There are two different application areas for pericardiocentesis: through the left 4th and 5th intercostal spaces or the subxiphoid region. The patient, in a semi-sitting position, is inserted with a 16-18F needle at a 30–45° angle into the intended area. Pericardiocentesis can also be performed from a parasternal approach. However, during procedures from this approach, care must be taken not to damage the internal mammary artery while entering through the 4th or 5th intercostal space. (21,30)

In all pericardiocentesis procedures, it is essential to ensure that the needle passes over the rib while preserving the neurovascular structures beneath the rib. Additionally, since the needle passes through important structures such as the xiphoid process of the sternum, liver, right ventricle, right atrium, right and left

coronary arteries, and pericardium, attention should be paid to the direction of the needle during the procedure. (21,30)

6.3. Pericardioscopy

Pericardioscopy is a minimally invasive procedure used for examining the pericardium. This method is typically employed in the diagnosis of pericardial diseases, especially when determining the cause of pericardial effusion or examining pericardial masses. Pericardioscopy can be utilized to establish the diagnosis of specific pericardial diseases, assess response to treatment, and sometimes even administer treatment. (21,31)

During pericardioscopy, a small incision is made in the chest wall by the physician, and then an endoscope is advanced into the pericardial space. This endoscope contains a camera and a light source to visualize the inside of the pericardium in detail. Using these images, the physician evaluates the condition of the pericardium and identifies any abnormal findings. If necessary, biopsies or other minor interventions can be performed using endoscopic instruments. (21,31)

One of the significant advantages of pericardioscopy is its ability to provide direct visualization of the pericardial region. This allows for more accurate diagnosis and treatment planning. Additionally, due to its minimally invasive nature, the patient's recovery time is shorter, and the risk of complications is lower. (21,31)

6.4. Pericardial biopsy

Pericardial biopsy is the process of obtaining tissue samples from the pericardium. This procedure plays a significant role in the diagnosis of pericardial diseases, particularly in helping to determine the causes of conditions such as pericarditis. Pericardial biopsy is typically performed in conjunction with the analysis of pericardial fluid and provides critical information for the diagnosis of various conditions including infection, inflammation, and cancer. (21,32)

6.5. Cardiac catheterization

Cardiac catheterization is an invasive medical procedure used to diagnose or treat conditions such as cardiac tamponade and constrictive pericarditis. During this procedure, a thin and flexible catheter is typically advanced through an artery or vein in the groin area and guided into the heart or major blood vessels. During cardiac catheterization, pericardial pressure measurement can be performed. For example, in cases such as pericardial tamponade, where

pericardial pressure may be elevated, useful information can be provided through catheterization. Additionally, pericardial biopsy can be performed during catheterization. This is necessary to confirm the diagnosis of pericardial diseases and determine appropriate treatment. However, caution must be taken to preserve the integrity of the pericardium during pericardial biopsy. (21)

7. The Relationship Between COVID-19 and Pericardial Diseases

In some studies conducted in the last two years, two different etiologies have been identified in patients diagnosed with acute pericarditis. One is the infectious SARS-CoV-2 virus, while the other is non-infectious mRNA vaccines developed against COVID-19. According to these studies, the incidence rate of pericarditis within the first 28 days in cases with a positive SARS-CoV-2 test result is reported as 2.79%, while the incidence of pericarditis in individuals vaccinated against COVID-19 within the same period is reported as 0.001%. (33)

Pericardial effusion is rare in COVID-19 patients, but cases associated with COVID-19 infection have been reported in the literature. Pericardial effusion associated with COVID-19 typically occurs in the advanced stages of the disease. It can vary depending on the severity of the infection, the intensity of the inflammatory process, and the overall health status of the patient. Pericardial effusion may occur in conjunction with other complications of COVID-19 infection or may be seen as an independent manifestation. (33)

8. Conclusion

Understanding the anatomy of the pericardium and its various variations is becoming increasingly important for innovative approaches to heart interventions. In addition, recent studies have highlighted the significance of pericardial spaces in facilitating catheter manipulation for approaches other than endovascular procedures. Therefore, the incompletely understood anatomy of the pericardium should be emphasized more to provide alternative perspectives for surgeries related to the heart and directly associated with the pericardium.

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CHAPTER III

SURGICAL ANATOMY OF THE LIVER

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1. Introduction

The liver, which plays a central role in the regulation of metabolic functions and the functioning of the hepatobiliary system, possesses regenerative capabilities and exhibits both exocrine and endocrine secretion abilities. It is the largest organ in the body and has both hepatic and portal vascularization.

Variations in the vascular structure, intra-abdominal location, organ relationships, and neurovascular connections of the liver have led to a detailed examination of liver anatomy, resulting in the emergence of various hepatic resection methods. The regenerative capacity of the liver contributes positively to the success of these methods. Functional classification based on the segmental anatomy of the liver is crucial for the success of surgical techniques related to the liver.

2. Liver Development

The initial liver bud emerges as an endodermal epithelial projection in the distal part of the foregut during the middle of the third week of embryonic development. This projection is known as the hepatic diverticulum. The hepatic diverticulum consists of cells originating from the mesodermal plate called the septum transversum. As liver cells infiltrate the septum, the connection between the hepatic diverticulum and the foregut narrows, leading to the formation of bile ducts. Ventral growth resulting from this process gives rise to the gallbladder and cystic duct. In the later stages of development, hepatic sinusoids form. Kupffer cells, hematopoietic cells, and other connective tissue cells originate from the

mesoderm of the septum transversum. As development progresses, the organ grows caudally within the abdominal cavity.

Derived from the mesoderm between the liver and the foregut, the lesser omentum and the falciform ligament between the liver and the anterior abdominal wall develop. The surface mesoderm of the liver, except for a small area at its apex, transforms into visceral peritoneum. This surface is never covered by the peritoneum and is known as 'area nuda'.

From the 12th week of development onwards, the liver begins to produce bile. Additionally, during this period, with the formation of the gallbladder, cystic duct, and common bile duct, the produced bile can flow into the intestines. (21)

3. Anatomy of the Liver

The liver is located in the right upper quadrant of the abdomen, extending to the left midclavicular line. Weighing between 1200-1400 grams, the liver's dimensions are approximately 25-30 cm in width, 14-16 cm in height, and 8-10 cm in anterior-posterior length. Rich in vascular supply, the liver possesses an elastic and easily damaged tissue. It ranks second in the risk of rupture after the spleen.

The position of the liver may vary depending on the body's posture; it can descend 3 cm during inspiration and rise 3 cm during expiration. The projection of the liver on the body surface is crucial for physical examination, especially for palpation and biopsy procedures. The liver's projection is roughly defined as a triangle located below the right nipple by 1 cm, 2 cm below the left nipple at the 5th intercostal space on the left, and between the intersection of the right costal margin with the anterior axillary line on the right side. This positioning can vary in pathological conditions leading to hepatomegaly. In cases of hepatomegaly, the lower edge of the liver can be palpable when it extends 1-2 cm below the right costal margin. (2,3)

3.1. Macroscopic Anatomy of the Liver

The liver is an intraperitoneal organ with a limited amount of peritoneal space. The peritoneal layer surrounding specific structures and closed embryonic vessels in the liver thickens to form hepatic ligaments and surgical folds. The liver has two surfaces: the diaphragmatic surface facing the diaphragm and the visceral surface facing the internal organs.

The anterior part of the diaphragmatic surface of the liver is entirely covered by the peritoneum, except where the falciform ligament is located. The

falciform ligament is formed by the convergence of peritoneal sheets covering the right and left lobes of the liver, extending to the anterior abdominal wall and the underside of the diaphragm. The lower edge of the falciform ligament is free and sickle-shaped. Between this free edge and its two sheets lies the round ligament, which contains the obliterated umbilical vein and extends to the umbilical fissure. (3,4,5)

The upper part of the diaphragmatic surface of the liver is mostly covered by the peritoneum. Here, a slight depression formed by the heart is called cardiac impression. When the falciform ligament reaches the upper part of the liver, its leaves open to both sides, forming the anterior leaves of the coronary ligament. (3,4)

The posterior part of the diaphragmatic surface faces the vertebral bodies and is separated from the diaphragm by the lower ribs. This surface is largely devoid of peritoneum. In the middle of the back surface, slightly to the right, there is a sulcus formed by the inferior vena cava. A little to the left of this sulcus, approximately 2-3 cm, lies the lig. venosum, which contains the obliterated ductus venosus from the embryonic period. Slightly to the right of the posterior end of lig. venosum and partly on the visceral surface, there is a groove formed by the adrenal gland. To the right of the sulcus formed by the inferior vena cava and slightly to the right of lig. venosum's posterior end, there is a trace of the esophagus.

The peritoneum extending from the posterior wall of the abdomen to the posterior part of the diaphragmatic surface of the liver, opening to the sides, forms the posterior leaves of the coronary ligament. The non-peritoneal area, approximately 15 cm in diameter, known as the "area nuda" extends forward between the anterior and posterior leaves and loosely attaches to the diaphragm. The anterior and posterior leaves join on both sides to form the right and left triangular ligaments. The posterior leaf of the coronary ligament extends along the anterior surface of the right kidney and adrenal gland, forming the hepatorenal ligament and fixing the liver to the right retroperitoneal region.

Structures that anatomically connect the liver to the right upper quadrant of the abdomen, in addition to peritoneal folds such as the falciform ligament, round ligament, coronary, and triangular ligaments, include the common iliac veins and hepatic veins. To prevent bleeding from an open left umbilical vein during surgical procedures, the ends of the falciform ligament must be tied before cutting. The right lobe is adjacent to the costodiaphragmatic recess between ribs 7-11 and is an extension of the posterior part, covered by peritoneum. (3,6,7)

On the visceral surface of the liver, among the traces of adjacent internal organs are: colic impression, representing the trace of the colon in the right lobe; renal impression, the trace of the right kidney; suprarenal impression, the trace of the right adrenal gland; and duodenal impression, representing the traces of the first and second parts of the duodenum. In the middle of this surface is a section called the porta hepatis, where the portal vein enters the parenchyma along with surrounding nerve structures, while the bile duct and lymphatic vessels exit. Located between the right sagittal groove on the right and the left sagittal fissure on the left, the porta hepatis, together with the other two structures, forms an "H" shape. These three structures divide the liver into four lobes: right, left, caudate, and quadrate. The structures known as the portal triad or pedicle, consisting of the portal vein, hepatic artery, bile duct, and the thin Glisson capsule surrounding them, branch to the right and left lobes from the porta hepatis, providing branches to each of the 8 segments. (3,5,7)

Visceral surface forms a right sagittal groove on the right, the anterior half of which creates the gallbladder fossa accommodating the gallbladder, and the posterior half forms the groove for vena cava located on the posterior part of the diaphragmatic surface. In the functional liver anatomy, right sagittal groove divides the liver into right and left lobes. The anterior half of the left sagittal fissure on the left contains the round ligament, and the posterior half contains the lig. venosum. The left portal triad progresses through the gap where the round ligament is located. The liver section behind the porta hepatis between these two formations is called the caudate lobe, while the anterior part is called the quadrate lobe. The prominence in the section of gastric impression adjacent to lig. venosum is called tuber omentale, and the atrophic fibrous remnant at the end of the left lobe is called appendix fibrosa hepatis. Except for the bilateral extending gaps and sulci around the porta hepatis, the entire visceral surface is covered with peritoneum. (3,5,6)

3.2. Microscopic Anatomy of the Liver

The liver tissue is characterized by a structure consisting of approximately 80% small lobules with hexagonal-shaped hepatocyte cords, forming the fundamental physiological unit. The remaining 20% is composed of supporting connective tissue containing the Glisson capsule. Structures surrounding the liver from the outside to the inside include visceral peritoneum, subserous layer, and fibrous layer (Glisson capsule) (3). The Glisson capsule is a thin membrane surrounding hepatocytes and other cells, connecting to a network

containing blood vessels and bile ducts. Extending from the porta hepatis towards the parenchyma, the Glisson capsule forms triangular spaces called Glisson triangles, Kiernan spaces, or portal distances at the corners of the hexagonal shape of the lobules. The portal triad formations in the Kiernan space include the hepatic arteriole, portal venule, bile canaliculus, nerves, and lymph vessels. In the center of the lobule, radially arranged sinusoids extend from the periphery to the central vein, providing venous drainage. Interlobular arteries and veins, radially arranged from the periphery to the center around these sinusoids in the portal triad, nourish the lobule. Thin bile ducts form between hepatocyte cord layers around these sinusoids, creating interlobular canals in the portal triad, transporting bile secreted by liver cells from the center to the periphery. (2,3,7)

4. Liver Surgical Anatomy

4.1. Peritoneal Structures Around the Liver

The upper supracolic region of the transverse colon is divided into two continuous spaces known as subphrenic and subhepatic. The subphrenic space, situated between the anterior part of the diaphragm and the diaphragmatic surface of the liver, is further divided into right and left portions by the falciform ligament. Additionally, the subhepatic space, located between the visceral surface of the liver and the transverse mesocolon, is subdivided into right (Morison's pouch) and left (lesser sac) compartments.

Peritoneal formations around the liver are summarized in Table 1. The spaces and recesses formed by the peritoneal layers surrounding intra-abdominal organs can lead to fluid accumulation in traumatic or pathological conditions. Occasionally, clinical situations arise due to adhesions between these peritoneal layers. (3,5,6)

Table 1: Peritoneal structures around the liver

Peritoneal structures around the liver	
Omental bursa	Anterior wall of the peritoneal cavity is formed, from top to bottom, by the caudate lobe, lesser omentum, the posterior surface of the stomach, and the upper part of the anterior two leaves of the greater omentum. The posterior wall is formed, from top to bottom, by the adrenal gland, upper part of the left kidney, anterior surface of the pancreas, transverse mesocolon, transverse colon, and the upper part of the posterior two leaves of the greater omentum. The foramen of Winslow is located on the right side, and the hilum of the spleen is found on the left side.
Lesser omentum:	The peritoneal structure that extends between the inner surface of the liver and the lesser curvature of the stomach, as well as the beginning of the duodenum, is called the lesser omentum. It forms the anterior wall of the omental bursa.
Hepatoduodenal ligament:	It extends from the porta hepatis to the beginning of the duodenum and forms the right border of the lesser omentum. It contains the porta hepatis, proper hepatic artery, and common bile duct.
Foramen of winslow:	It is the entrance opening of the lesser sac, bounded by the hepatoduodenal ligament above and the beginning of the duodenum below, and limited by the caudate lobe above and the beginning of the duodenum below, between the hepatoduodenal ligament and the inferior vena cava.
Gastrohepatic ligament:	On the visceral surface of the liver, the tuber omentale begins between the ligamentum venosum and the porta hepatis, attaching to the lesser curvature of the stomach. Between its two leaves, the right and left gastric arteries extend, anastomosing with each other.

4.2. Functional Liver Anatomy

The model of functional liver anatomy was initially described by Cantlie in 1898 and later developed through the studies of Lawrence, Healey, Schroy, Goldsmith, and Woodburne. In 1957, Couinaud further refined the model by considering the vascularization of the liver, dividing it into sectors and segments. In 1982, Bismuth's revisions led to the emergence of an independent, 8-segment

functional liver classification, each with its own portal pedicle. To establish a universally accepted terminology, the Brisbane 2000 IHPBA (International Hepato-Pancreato Biliary Association) committee organized and modified this classification. (5,8) Key structures related to the functional anatomy of the liver are summarized in Table 2.

Table 2: Functional liver anatomy

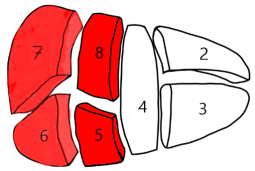
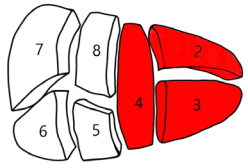
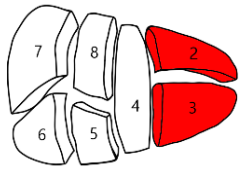
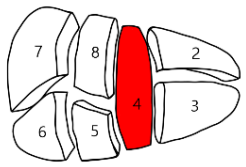
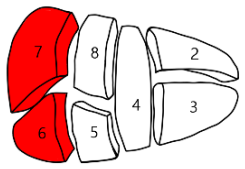
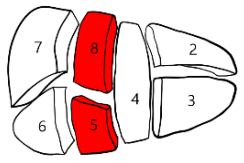
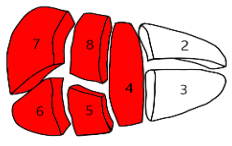
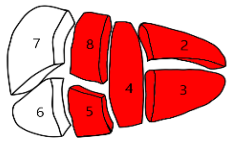
Functional liver anatomy	
Cantlie's line:	It is the main cleft located on the visceral surface. The middle hepatic vein, which divides the liver into right and left lobes, runs from this point.
Right intersegmental fissure:	It contains the right hepatic vein and is located within the right lobe. It divides the right lobe into anterior and posterior sectors.
Left intersegmental fissure:	It separates the lateral sector of the left lobe, divided by the falciform ligament, into anterior and posterior segments. It corresponds to the left hepatic vein.
Hypothetical horizontal fissure:	This fissure, passing at a 75° angle from the portal bifurcation line to the Cantlie line, divides the liver into upper and lower segments.
Caudate lobe:	It lies on the visceral surface of the liver, in the posterior part, between the ligamentum venosum fissure and the Cantlie line. It is numbered as the 1st segment, with the other segments numbered clockwise.

As a result of the horizontal fissure parallel to the portal pedicles dividing the sectors into upper and lower, in the left lateral sector, the 2nd (posterior) and 3rd (anterior) segments are separated by the left hepatic vein, in the left medial sector, segments 4a and 4b, in the right anterior sector, the 5th and 8th segments, and in the posterior sector, the 6th and 7th segments are located.

Each segment has its independent portal pedicle, allowing for the isolation and removal of a specific segment in surgical interventions. This segmental classification plays a significant role in clinical applications in the fields of liver surgery and anatomy. (5,7,9)

The surgical treatment of primary and metastatic liver tumors and the terminology used in transplantation according to the Brisbane 2000 classification are provided in Table 3.

Table 3: Brisbane 2000 hepatic resection terminology

Right hepatectomy/ hemihepatectomy	Removal of segments 5, 6, 7, and 8	
Left hepatectomy/ hemihepatectomy	Removal of segments 2, 3, and 4	
Left lateral sectionectomy/ bisegmentectomy	Removal of segments 2 and 3	
Right lateral sectionectomy/ bisegmentectomy	Removal of segment 4	
Right posterior sectionectomy	Removal of segments 6 and 7	
Right anterior sectionectomy	Removal of segments 5 and 8	
Right trisectionectomy/ extended right hepatectomy	Removal of segments 4, 5, 6, 7 and 8	
Left trisectionectomy/ extended left hepatectomy	Removal of segments 2, 3, 4, 5, and 8	

5. Liver Anomalies and Vascular Variations

5.1 Accessory Liver Lobes

Accessory liver lobes, unlike ectopic liver lobes that do not have anatomical continuity with the normal liver, are defined as additional liver lobes that maintain continuity with the normal parenchyma of the liver. The presence of accessory liver lobes is rare. These lobes are considered as morphological variations of the liver and are associated with excessive development. They are generally asymptomatic, but if they have pedicles, symptoms often manifest in a torsion-like manner. Accessory liver lobes can vary in shapes, sizes, and locations. Depending on how they are connected to the liver, they can be pedunculated or sessile. The Riedel lobe is the best-known type of accessory liver lobe. Although the Riedel lobe is defined as the tongue-like extension of hepatic segments V and VI on the right, this accessory lobe can also be found in the left liver. In addition to the Riedel lobe, other accessory liver lobes include pedunculated lobes and ectopic lobes. Symptoms associated with accessory lobes, especially the Riedel lobe, can mimic a mass presenting with pain, vomiting, constipation, or bloating in the right upper quadrant. Primary hepatocellular or metastatic tumors have been reported in accessory or ectopic lobes. (11)

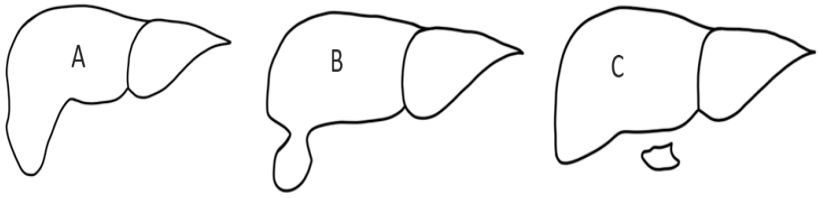


Figure 1: Accessory liver lobes, A Riedel's lobe,
B Pedunculated lobe, C Ectopic lobes.

5.2 Portal Vein Variations

Ischemia and necrosis due to both portal vein and hepatic artery supply to the liver are extremely rare, as the portal vein can balance intrahepatic pressure even at low pressure, providing 50% of oxygen demand and 75% of blood flow without valves. The portal vein, formed by the union of the splenic and superior mesenteric veins, progresses to the porta hepatis and divides into right and left branches. The cystic vein, paraumbilical veins, superior pancreaticoduodenal vein, and right and left gastric veins directly drain into the portal vein. Normally, the portal vein courses behind the first part of the duodenum. Preduodenal portal

vein denotes an abnormal location of the portal vein in front of the duodenum and is often associated with various congenital anomalies, most commonly intestinal obstruction.

In the normal branching pattern of the portal vein, when the left portal vein enters the left lobe, it gives branches to segments 2, 3, and 4. The right portal vein bifurcates into an anterior branch supplying the right lobes and a posterior branch supplying segments 5 and 8. The caudate lobe is nourished by branches from both the right and left portal veins. (12)

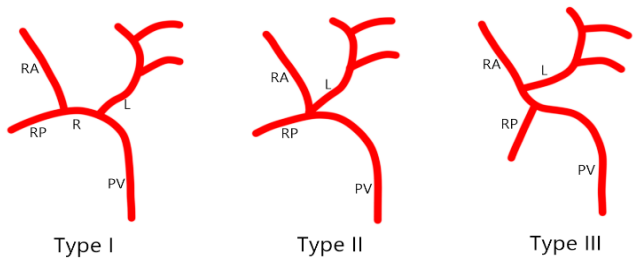


Figure 2: Schematic diagram showing three common types of anatomy of the portal ven, RA right anterior, RP right posterior, PV portal ven, R right, L left.

Approximately 25% of the population exhibits variations in portal vein anatomy, with three main types described in the literature. Type 1 represents normal anatomy. Type 2 and Type 3 indicate situations where the portal vein trifurcates into right anterior, right posterior, and left branches (9-10%) and where the right posterior portal vein separates early from the main portal vein (13–24%), respectively. Another rare variation involves early branching of the right portal vein. Hypoplasia or absence of the right or left portal vein is extremely rare.

Portal vein embolization is performed 4-6 weeks before surgical procedures such as major hepatectomies or hepatic tumor resections to increase the size of the remaining liver tissue for postoperative success. Identifying portal vein variations is critical for the success of these surgical procedures. (5,9,13)

5.3 Hepatic Arter Variations

The main hepatic artery, one of the three main branches of the celiac trunk, gives rise to the proper hepatic artery, which courses within the hepatoduodenal ligament. The proper hepatic artery supplies 25% of the arterial blood to the liver. Before bifurcating near the porta hepatis, the proper hepatic artery divides into right and left branches. The classical branching pattern observed in 60-75%

of the population involves the left hepatic artery coursing within the umbilical fissure and giving off branches to the 2nd, 3rd, and 4th segments. The left hepatic artery bifurcates into medial and lateral branches in 40% of cases, with the lateral branch further dividing into upper and lower branches in 35% of cases. After supplying the cystic artery in the Calot triangle, the right hepatic artery bifurcates into anterior branches (5th and 8th segments) and posterior branches (6th and 7th segments). (14-16)

There is a variation rate of 20-40% in the branching pattern of the hepatic artery. Ten types of hepatic artery anatomy have been identified (Table 4).

Table 4: Hepatic artery branching patterns

Hepatic artery branching patterns	
Type I	Normal anatomy
Type II	The left hepatic artery branching from the left gastric artery
Type III	The right hepatic artery branching from the superior mesenteric artery
Type IV	Combined replaced right and left hepatic arteries
Type V	Accessory left hepatic artery branching from the left gastric artery
Type VI	Accessory right hepatic artery branching from the superior mesenteric artery
Type VII	Combined accessory right and left hepatic artery
Type VIII	Replacement of the right hepatic artery and accessory left hepatic artery or replacement of the left hepatic artery and accessory right hepatic artery
Type IX	Replacement of the main hepatic artery branching from the superior mesenteric artery
Type X	Replacement of the main hepatic artery branching from the left gastric artery

Beyond the normal branching pattern, the most common variations are classified as types II, III, and V. Determining whether type III and IV variations are present is crucial for liver transplantation donors and recipients. In types II and V, the presence of an accessory or modified left hepatic artery in the donor is not significant. However, an increase in length and diameter complicates thrombus formation, necessitating mandatory anastomosis. Type IV variations are observed in 1-2% of cases. Intrahepatic collateral circulation is established

within 24 hours after ligating the right and left hepatic arteries, utilizing the right inferior phrenic, pancreaticoduodenal, and subcostal arteries. If the 4th segment artery arises from the left hepatic artery and has not been identified preoperatively, clamping the right hepatic artery during resection induces ischemia in the medial segment of the left lobe, and regeneration is not observed in the remaining segment in the donor. (5,7,9,14,15,16)

5.4 Hepatic Vein Variations

Hepatic veins are the major intrahepatic vessels draining into the inferior vena cava. Typically, there are three of them: right, left, and middle. Despite the liver being supplied by both the portal vein and hepatic artery, blood drainage occurs solely through the hepatic veins. In approximately 65-85% of the population, the left and middle hepatic veins converge after a short extrahepatic course, forming a common trunk approximately 2 cm long that drains into the inferior vena cava adjacent to the lig. venosum on the left side. Apart from these three main hepatic veins that empty the liver parenchyma, some small accessory veins also directly drain into the inferior vena cava. Generally, the right hepatic vein drains segments 6-7 entirely and a portion of segments 5 and 8, while the middle hepatic vein provides drainage for the entire segment 4 and a portion of segments 5 and 8. The left hepatic vein in the left intersegmental fissure drains the entire segments 2-3 and a portion of segment 4. (5,7,13,17)

The intrahepatic venous system has widespread intersegmental anastomoses. Unlike the protective fibrous sheath surrounding the portal pedicle, there is no such sheath around the hepatic veins, making them susceptible to injury during surgical operations. Therefore, in surgical resections, the resection line is determined to run parallel to the hepatic veins.

Table 5: Hepatic vein variations

Hepatic vein variations	
RHV variations	Single RHV
	Early branching of RHV
	2 RHV: common trunk; independent drainage
	3 RHV: common trunk; independent drainage
	Accessory inferior RHV
	Small RHV with well-developed MHV
MHV and LHV variations	Common trunk with LHV
	Independent drainage of LHV and MHV into IVC
Segmental hepatic vein variations	Segment IV vein draining into MHV
	Segment IV vein draining into LHV
	Segment IV vein draining into IVC
	Umbilical vein draining into MHV
	Umbilical vein draining into LHV
	Umbilical vein draining into IVC
	Common trunk of segment IV vein and umbilical vein draining into LHV
	Common trunk of segment IV vein and umbilical vein draining into MHV
	Common trunk of segment IV vein and umbilical vein draining into LHV
	LMV draining into LHV
	LMV draining into MHV
	ASSV draining into MHV
	ASSV draining into RHV

ASSV, anterior superior segment vein; IVC, inferior vena cava; LHV, left hepatic vein; MHV, middle hepatic vein; RHV, right hepatic vein.

Understanding the detailed anatomy of hepatic vein variations is crucial to define the surgical resection line and prevent venous congestion in the transplanted segment. Table 5 illustrates the most common hepatic vein variations, including the fusion of the left and middle hepatic veins to drain into the inferior vena cava, the drainage of a small accessory right inferior hepatic

vein from the anterior surface of the inferior vena cava (40-65%), and the early branching pattern of the right hepatic vein, where two or three branches unite or separately drain into the inferior vena cava. (5,7,13)

6. Liver Lymph Drainage

Most of the deep lymph vessels of the liver converge at the porta hepatis, where they open into the hepatic nodes. The lymph vessels leaving the hepatic nodes then drain into the celiac nodes and subsequently into the thoracic duct. Some of the deep lymph vessels follow the hepatic veins, reaching the foramen venae cavae through the openings in the diaphragm. These lymph vessels then drain into the superior phrenic nodes and from there into the parasternal nodes. The superficial lymph vessels of the liver join the deep lymph vessels heading towards the porta hepatis, opening into the hepatic nodes and then into the celiac nodes. Lymph vessels originating from the peritoneum-free area nuda pass through the diaphragm. They later pass into the thoracic cavity, draining into the superior phrenic nodes, anterior and posterior mediastinal nodes. Lymph vessels in lig. coronarium and lig. triangulare directly open into the thoracic duct. (1,3)

7. Liver Innervation

The liver is an organ innervated by the autonomic nervous system. Sympathetic fibers originate from the splanchnic nerves, while parasympathetic fibers come from the right and left vagus nerves. These fibers first form the celiac plexus. Subsequently, they enter the liver as the hepatic plexus around the proper hepatic artery and portal vein. Sensory fibers extend along with sympathetic fibers and reach the spinal cord segments of thoracic 8-11. Therefore, pain originating from the parenchymal tissue of the liver is perceived as referred pain in the distribution area of the 10th intercostal nerve and in the upper abdominal region. The branches of the right phrenic nerve distribute over the peritoneum surrounding the liver, so pain originating from the liver peritoneum is felt in the right shoulder. (1,3)

8. Conclusion

Liver surgery has become increasingly effective and safe with advancements in technological and surgical developments in modern medicine. Minimal invasive surgical techniques have marked significant progress in shortening the postoperative recovery process and enhancing the quality of life for patients.

However, the complex anatomy and vascularization of the liver play a crucial role when surgical teams plan interventions for this organ. Understanding the segmental structure and vascularization of the liver is a critical foundation for planning and implementing surgical interventions and conducting specific interventions for lesions.

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CHAPTER IV

CLINICAL ANATOMY OF THE KIDNEY

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1. Introduction

The kidneys are organs located on both sides of the spinal column at the very top of the posterior abdominal wall, tasked with regulating the body's water and salt balance. As the fundamental organs of the excretory system, the kidneys have three primary functions: filtration, secretion, and absorption. They play a vital role in regulating the acid-base balance in the body, excreting waste products such as urea, creatinine, and uric acid that result from metabolic activities, detoxifying and eliminating toxins, drugs, and drug metabolites, and managing the body's fluid and electrolyte balance. In addition to these critical functions, the kidneys also perform endocrine functions, such as secreting renin, erythropoietin, and components of the kallikrein-kinin-prostaglandin system, controlling electrolyte metabolism through hormonal mechanisms, and synthesizing certain growth factors.

2. Kidney Embryology

The kidney and urinary tract develop from the intermediate mesoderm of the urogenital ridge on the posterior abdominal wall of the fetus. The development of the primitive kidney goes through three stages: pronephros, mesonephros, and metanephros. The first stage, pronephros, occurs around the 22nd day (3rd week) of pregnancy. Pronephros has no filtration function and disappears by about the 28th day, after which the mesonephros stage begins. Although mesonephros has some functional filtration capability, it also significantly affects the development of the neighboring adrenal and gonadal glands. The final stage, metanephros,

results in the formation of the permanent kidneys. The calices, collecting ducts, renal pelvis, and ureters originate from the metanephric diverticulum, while the glomeruli and Bowman's capsule derive from the metanephric mesoderm.

The kidneys appear in the fetal pelvis at the end of the 4th week of embryonic life and migrate to their post-birth location in the retroperitoneum of the lumbar region at approximately the L2 level around the 8th week. The fetal kidneys and bladder filled with urine can be observed during the 12th and 15th weeks of pregnancy. Ultrasound scans for anomalies are conducted between the 18th and 22nd weeks of regnancy. Kidney maturation is completed between the 34th and 36th weeks of pregnancy.

3. Kidney Anatomy

3.1. Macroscopic Anatomy of the Kidneys

The inner and outer edges of the kidney are called the medial border and lateral border, respectively. The renal hilum, a notch located in the middle part of the medial border at the level of the L1 vertebra, forms the entrance to a cavity known as the renal sinus. Just behind the renal hilum, 2-3 branches of the renal artery (a. renalis) enter, while the renal vein (v. renalis) exits at the front. The ureter exits from the renal hilum behind the a. renalis. A branch of the a. renalis enters the kidney hilus from behind the ureter. Additionally, perirenal fat tissue enters the renal sinus from the kidney hilus, filling the spaces between the structures there.

The anterior and posterior surfaces of the kidney are called anterior surface and posterior surface, respectively. The posterior surface is located within loose connective and fat tissue, and directly lies on the psoas major muscle, quadratus lumborum muscle, transversus abdominis muscle, and diaphragm without any separating membrane. Some nerves and vessels pass between the kidneys and these muscles.

The upper and lower ends of the kidneys are called superior end and inferior end, respectively. The lower ends are smaller and thinner than the upper ends and are located farther apart from each other. The superior end is adjacent to the 12th rib on the right and the 11th and 12th ribs on the left.

3.1.1. Anatomical Location of the Kidneys

The kidneys are the largest and fundamental organs of the urinary system. Due to the pressure exerted by the liver, the right kidney is positioned slightly lower than the left kidney. Moreover, the left kidney is morphologically a bit

narrower and longer. The kidneys have two surfaces (front and back), two ends (upper and lower), and two edges (medial and lateral). Positioned in the retroperitoneal space and located at the very top of the posterior abdominal wall, the lower ends of the kidneys are at the level of the T12-L3 vertebrae. During deep inspiration, the kidneys move about 3 cm downward from their position. The upper ends of the kidneys tilt medially, the lower ends tilt anteriorly, and the medial surface tilts forward at an angle of approximately 30º.

The position of other organs adjacent to the kidneys, the surrounding pararenal and perirenal fat tissues, and peritoneal membranes (renal fascia (Gerota's Fascia), perirenal fat capsule, fibrous capsule), along with the blood circulation provided by the renal arteries and veins (A.V. renalis), and the pressure from the anterior abdominal wall created by the tone and tension of the muscles in the area, all help maintain the kidneys in their position. Gerota's Fascia is the most crucial structure holding the kidney in place and also encompasses the adrenal gland.

3.1.2. Structures Adjacent to the Kidneys

The upper end of the right kidney is adjacent to the Inferior Vena Cava (IVC), the upper-anterior surface is adjacent to the right lobe of the liver, and the upper-inner surface is adjacent to the right adrenal gland. The outer edge of the lower end is adjacent to the right colic flexure, and the renal hilum is in contact with the second part of the duodenum.

The upper-inner surface of the left kidney is adjacent to the left adrenal gland, and the rest of the kidney is adjacent to the stomach. The inner edge of the left kidney is close to the abdominal aorta. The spleen, pancreas, loops of the jejunum, and the left colic flexure are other organs that are adjacent to the left kidney.

In the middle, the kidneys are directly in contact with the psoas major muscle, the quadratus lumborum muscle (m. quadratus lumborum), the diaphragm, and on the sides with the transversus abdominis muscle (m. transversus abdominis).

3.1.3. Important Structures Surrounding the Kidneys and Peritoneal Membranes

The kidneys, which are vital organs of the urinary system, are encapsulated from the outside inward by the pararenal fat pad, Gerota's fascia (renal fascia), the perirenal fat capsule, and the fibrous capsule. Gerota's fascia is particularly crucial as it maintains the localization of the kidney and prevents the spread of kidney-originated pathologies.

Gerota's Fascia (Renal Fascia)

The subserosal fascia splits into two layers near the convex outer edge of the kidney, enveloping both surfaces of the kidney and extending to its inner edge. The thicker posterior layer, the retrorenal fascia, merges with the fascia of the psoas major muscle and subsequently with the prevertebral fascia at the upper end, continuing upwards as the diaphragmatic fascia. It also connects laterally to the transversalis fascia. The anterior layer, the prerenal fascia, encloses the renal vessels (A.V. renalis) at the kidney hilum and leaps forward from the anterior parts of the IVC and the aorta, merging with the anterior layer of the opposite side. These layers join more loosely at the lower end, integrating into the iliac fascia. Some fibrous fibers progress within the corpus adiposum, linking the renal fascia to the fibrous capsule. A thin layer separating from the renal fascia wraps around the adrenal gland.

Perirenal Fat Capsule

This is the perirenal fat tissue. While it is more abundant on the posterior surface of the kidney, there are no fat layers in the peritoneum-covered areas on the anterior surface.

Fibrous Capsule

This is a thin fibrous capsule that surrounds the kidney externally. Made of sturdy collagen fibers, it is slightly expandable. At the renal hilus, this sheath splits into two layers: the outer layer covers the structures there, forming their adventitia, while the inner layer enters through the renal hilum, lining the interior surface of the renal sinus up to the papillae. Between this sheath and the kidney tissue lies a thin layer called the subfibrous layer, which contains smooth muscle fibers. This layer is more tightly adhered to the kidney tissue, and the smooth muscle fibers within it gather around the papillae in the part covering the renal sinus, forming muscular rings. The fibrous capsule is adherent to the structures at the renal hilus, making it easy to separate from the more loosely attached subfibrous layer, i.e., from the kidney itself.

3.2. Microscopic Anatomy of the Kidneys

Upon examining a coronal section of the kidneys, the outer region that envelops the kidney like a shell and extends projections into the inner region is referred to as the renal cortex. This area is lighter in color. The inner region, characterized by its striated appearance and darker color, is known as the renal medulla. The kidney-shaped space between this section and the renal hilum

is called the renal sinus (renal sinus). The inner surface of the renal sinus is lined with the fibrous capsule and is filled with blood vessels, renal calyces, and the fat tissue between these structures. The renal cortex, which originates from nephrogenic tissue, contains capillary clusters known as glomeruli that are responsible for the filtration of urine. The renal medulla, which develops from the ureteric bud, comprises structures called renal pyramids that consist of collecting ducts.

3.2.1. Renal Cortex

The renal cortex, derived from nephrogenic tissue, encompasses the structures responsible for urine formation and is divided into two parts based on its location. The first part is situated near the outer surface of the kidney, extending from beneath the fibrous capsule to the base portions of the Malpighian pyramids (renal pyramid) with an approximate thickness of 1 cm; this is the area known as the renal cortex. The second part, known as the renal columns (columna renalis or Bertin's columns), extends between the renal pyramids.

Within the renal pyramid, there are extensions called radiate part (also known as medullary stria or Ferrein's extensions) that stretch from the medulla renalis towards the renal cortex. Between these extensions, there are structures known as renal corpuscles (Malpighian bodies) that have the function of filtering blood to form urine, and parts of the urinary tubules named convoluted part. Compared to the radiate part, the convoluted part appears more complex and lighter in color. The renal corpuscles contain a capillary bundle (glomerulus) that filters urine from the blood and appears as a red dot.

3.2.2. Renal Medulla

The renal medulla is characterized by its cone-shaped, striped appearance and dark red color. It typically consists of 8-10 (sometimes 18-20) renal pyramids, each shaped like a pyramid with the apex pointing towards the renal sinus (renal sinus) and the base facing the renal cortex. The apex of the renal pyramids is referred to as the renal papilla (papilla renalis). Except for the parts where the renal papillae are located, the renal cortex wraps around the renal pyramids and sends extensions known as renal columns (columna renalis or Bertin's columns) between the pyramids.

Together, the renal pyramid and the surrounding renal cortex are termed a renal lobe (lobus renalis). While the outer surface of the kidney appears smooth in adults, the boundaries of the renal lobes are distinctly marked by grooves on the kidney's surface in newborns.

Typically, 1-3 renal papillae converge to drain into a minor calyx (calyx minor). Two to three minor calyces then join to form a major calyx (calyx major). The major calyces merge to create the funnel-shaped renal pelvis (pelvis renalis), which opens into the ureter at the level of the L1 vertebra.

3.2.3. Renal Tubular System (Tubulus renalis)

The renal tubular system begins with a cluster of capillaries known as the glomerulus, which is enclosed within the nephrogenic tissue-derived Bowman's capsule (capsula glomerularis). The tubular system, referred to as the renal tubules (tubulus renalis), transports the urine filtered at the glomerulus to the calyces through openings in the renal papilla known as foramina papillare. The renal corpuscle, consisting of the Bowman's capsule and the glomerulus, marks the starting point of this system. Each renal corpuscle has two poles: the vascular pole (polus vascularis), where blood vessels enter and exit, and the tubular pole (polus tubularis), where filtered urine exits.

The filtered urine in the glomerulus undergoes reabsorption of water and some minerals back into the bloodstream. The collecting tubules (tubulus collectivus), originating from the ureteric bud, are solely responsible for conveying urine. These collecting tubules merge to form thicker collecting ducts located in the renal cortex's radiate part and the renal medulla's renal pyramid. These ducts eventually open into the minor calyx through the foramina papillare.

The descending thin limb of the loop of Henle is permeable only to water due to the presence of aquaporin 1. The loop of Henle extends deep into the medulla, makes a U-turn, and reaches back towards the renal cortex. The ascending thick limb of the loop of Henle is permeable to electrolytes (salts), resulting in the dilution of urine within its lumen. Tamm-Horsfall protein is produced and secreted into the lumen in the ascending limb of the loop of Henle. The final segment of the distal tubule passes between the afferent and efferent arterioles and transforms into a group of densely packed, large-nucleated cells known as the macula densa, which are involved in detecting the concentration of Cl- in the lumen. Together, the macula densa, extraglomerular mesangial cells, and the cells in the walls of the afferent and efferent arterioles formthe juxtaglomerular apparatus. The juxtaglomerular cells in the wall of the afferent arteriole are specialized smooth muscle cells capable of secreting renin. The collecting ducts, extending from the distal tubule, are divided into three sections: cortical, inner medullary, and outer medullary.

3.2.4. The Kidney's Smallest Functional Unit: The Nephron

The kidney's morphologically smallest unit, which forms urine, is called the nephron. Each urine tubule, along with the renal corpuscle that precedes it, constitutes a nephron. The structures that make up a nephron, from proximal to distal, are the renal corpuscle, tubulus proximalis, Henle's loop, and tubulus distalis.

The arteriola glomerularis afferens, upon entering the Bowman's capsule, immediately forms a complex capillary network called the glomerulus (rete capillare glomerulare). Each glomerulus and the Bowman's capsule surrounding it are referred to as the renal corpuscle. The structure of these capillaries includes a single layer of endothelial cells and a glomerular basal membrane that surrounds them. Most of the epithelial cells called podocytes (visceral layer of Bowman's capsule) have extensions projecting towards the Bowman's space. These three structures positioned from inside to outside—endothelial cell, glomerular basal membrane, and podocyte—together create the filtration membrane. The arteriola glomerularis afferens, after forming the glomerulus within the Bowman's capsule, exits the capsule as arteriola glomerularis efferens. The efferent arterioles, like the afferent arterioles, form a capillary network around the tubules called rete capillare peritubulare. The kidney's blood circulation features a specialized circulation with two separate capillary networks. Due to the high hydrostatic pressure in the glomerulus, it is prone to filtration. However, the pressure in the peritubular capillary network is quite low, facilitating the reabsorption of fluid from the tubule back into the capillaries.

The single-layer endothelial cells lining the capillary vessels have large fenestrations (windows). The glomerular basal membrane's structure, containing type 4 collagen (most abundant), laminin, heparan sulfate, agrin, and perlecan-like proteoglycans, creates a strong negative charge on the membrane. Podocytes, located outside the basal membrane, are polarized cells with interdigitating foot processes. The filtration slits between neighboring podocytes are covered with proteinaceous structures. Only albumin can pass through these filtration slits, which are quite small in diameter. Between podocytes on the visceral layer of Bowman's capsule and the flat epithelium on the parietal layer lies the Bowman's space. Plasma filtered from the capillary vessels accumulates in this space and progresses inside the proximal tubule (14-17).

4. Kidney Nutrition and Segmental Anatomy

The segments of the kidney are formed according to the distribution areas of the segmental arteries. From the bottom upwards, they can be listed as the

lower pole, the middle part of the posterior surface, two on the middle part of the anterior surface, and the upper pole (2-16).

The renal arteries originate directly from the abdominal aorta. The blood brought to the kidney by the arteries not only nourishes the organ but also plays a role in the filtration function**.**

4.1. Renal Arteries

Branches of the Renal Artery (A. renalis):

1. anterior suprarenal artery
2. ureteric branches
3. segmental artery
   - superior segmental artery
   - superior anterior segmental artery
   - inferior anterior segmental artery
   - inferior segmental artery
   - posterior segmental artery
4. intrarenal artery
   - interlobar artery
   - arcuate artery
   - straight artery
   - interlobular artery
   - deferential artery
   - capsular artery

The left renal artery, originating from the abdominal aorta at the level of the discus intervertebralis between the L1-L2 vertebrae, branches off at a right angle and is located slightly higher than the right one. The right renal artery passes behind the inferior vena cava (IVC) and is longer than the left. The renal arteries, which supply the kidneys, are relatively thick in diameter compared to the size of the kidney because they carry a large amount of blood in a short time. Before entering the renal hilum, the renal artery gives off branches to the inferior suprarenal artery and the ureteric branch. After entering the renal hilum, it gives off segmental branches, dividing the kidney into 5 segments (2,16).

Just before entering the renal hilum, the renal artery gives off a branch to the posterior segment. The segmental arteries, which form and supply the segments, are known as end-arteries and do not form anastomoses. Therefore, a blockage in a segmental artery can cause ischemia and infarction in the renal

parenchyma supplied by that artery. The area known as Brödel's line or the white line is an avascular line recommended for percutaneous nephrostomy interventions and is formed as a result of the segmental branching of the renal artery, representing the upper border of the lower segment in the posterolateral part of the kidney. In the upper segment, arteries and veins cross over; this crossing can give the appearance of a filling defect in urograms and sometimes lead to dilation in the upper major calyx, causing superior infundibulum syndrome (11-13).

The branches of the segmental artery divide again in the renal sinus, passing around the minor calyx and giving off branches of the interlobar arteries that travel through the renal columns. The interlobar arteries then give off arcuate branches. The arcuate arteries extend between the renal cortex and medulla. The arcuate arteries then give off interlobular branches, which travel between the renal lobules. From these branches, arterioles known as afferent glomerular arterioles extend laterally in a thin structure to form the glomerular capillary network, entering the glomerulus from the vascular pole. After forming the capillary network, they exit the glomerulus as efferent glomerular arterioles. These capillaries, descending around the tubular system into the depths of the medulla, form the vasa recta. Thin branches diverging from the ends of the interlobular arteries within the adipose capsule and fibrous capsule form a network and anastomose with branches from the lumbar, testicular, and suprarenal arteries. Similarly, in the renal hilum, branches diverging from the renal artery to supply the adipose capsule, renal pelvis, and calyces also anastomose with branches from the lumbar, testicular, and suprarenal arteries. The renal medulla has much poorer circulation compared to the renal cortex, making it highly susceptible to ischemic damage. Furthermore, blood that cannot be filtered in the glomerulus can directly pass into the venous system through arteriovenous anastomoses formed between the fine vessels of the renal cortex and around the calyces and between the interlobular arteries and veins (14-15).

4.1.1. Renal Artery Variations

In the arterial vascularization of the kidneys, approximately 70% of kidneys are supplied by a single renal artery, but variations are present in the remaining 30%.

Understanding renal artery variants is crucial for safe and effective uro-radiological interventional procedures and surgical operations. Many researchers have conducted studies on renal artery variations based on CT images and cadaveric studies, making statistical inferences.

In one study, it was observed that the aortic path between the upper border of L1 and the lower border of L2 constituted 96% of the main renal arteries and 72% of the extra-renal arteries. The most common site of origin of the renal artery was found to be at the level of the L1-L2 intervertebral disk. In 69% of patients, a single renal artery was observed, multiple arteries in 31%, bilateral multiple arteries in 11% of cases, and early branching in 6% of cases. Additional renal arteries were identified in 5% of cases on the right side and 12% on the left side.

Various researchers in different populations have conducted studies on renal artery variations using CT images or cadavers. They have obtained different results regarding the frequency of double hilum artery, triple hilum artery, superior polar artery, and inferior polar artery (Table 1).

Table 1: Comparison of the frequency of multiple renal arteries in different population groups.

Authors	DHA	THA	SPA	IPA
Sampaio and Passos (1992)	7.9%	1.9%	6.8%	5.3%
Çiçekcibaşi et al. (2005)	11.1%	-	3.3%	10.5%
Weld et al. (2005)	12.3%	-	9.6%	15.1%
Talovic et al. (2007)	9%	1%	2%	10%
Guan et al. (2011)	45.5%	18.8%	9.4%	3.2%
Budhiraja et al. (2012)	22.6%	11.8%	13.1%	7.1%
Pradhay et al. (2021)	7%	-	6%	9%

(DHA: double hilum artery, THA: triple hilum artery, SPA: superior polar artery, IPA: inferior polar artery)

Similarly, the distribution of the extrahilar branch of the renal artery for the right and left kidneys has been studied in different population groups, and the results have been statistically evaluated (Table 2).

Table 2: Percentage distribution of the extrahilar branch of the renal artery for the right and left kidneys in different population groups.

Authors	Extrahilar superior polar		Extrahilar inferior polar	
	Right kidney	Left kidney	Right kidney	Left kidney
Talovic et al. (2007)	10%	2%	2%	0
Palmieri et al. (2011)	28.60%	11.60%	0	1.40%
Budhiraja et al. (2012)	21.40%	19%	11.90%	2.40%
Pradhay et al. (2021)	8%	2%	8%	4%

In another study, renal artery variations were investigated according to laterality, gender, symmetry, and data collection method (Table 3) (33).

Table 3: Variations in renal arteries depending on laterality (left vs. right), gender (female vs. male), symmetry (unilateral vs. bilateral), and data collection method (CT vs. cadaver).

Variations	Percentage of variation
Left kidney	37/297 (12.5%)
Right kidney	41/297 (13.8%)
Female	25/164 (15.2%)
Male	37/136 (27.2%)
Single side	49/294 (16.7%)
Both sides	10/294 (3.4%)
CT study	53/272 (19.5%)
Cadaver study	13/28 (46.4%)

4.2. Renal Veins

The vasa recta form a U shape in the renal medulla before returning to the renal cortex. They give rise to fine branches around the tubules, forming a capillary network called the rete capillare peritubulare corticale. Since the urine in the tubules is more dilute while the blood in this network is concentrated, some electrolytes and minerals from the tubules, along with excess water, are reabsorbed into the blood. Some vasa recta arise from the arcuate artery, while others arise from the efferent arteriole to supply the renal medulla with blood. Via the venula recta and then the interlobular vein, the vasa recta drain into the

arcuate vein. After opening into the interlobar vein and then the segmental vein, they exit the kidney as the renal vein through the renal hilum. While there are typically three segmental veins (53.8%), this number can vary up to five. The renal vein is seen to bifurcate in about 28.8% of cases. The right renal vein is usually 2-4 cm long and opens directly into the lateral aspect of the inferior vena cava. Before joining the inferior vena cava, the left renal vein crosses in front of the aorta, receiving the left testicular vein, left lumbar vein, and left suprarenal vein, and then drains into the inferior vena cava slightly higher than the right renal vein. Unlike the renal arteries, there can be anastomoses between the veins forming the renal venous network.

4.2.1. Renal Venous Variations

The morphology of renal vessels has been studied by many researchers due to its significant implications in abdominal surgeries such as nephrectomy and kidney transplantation. Understanding the morphology and frequency of vascular abnormalities is crucial, especially in laparoscopic surgeries, where repairing renal vessels is often more challenging than in open surgery and can lead to bleeding, transfusion requirements, or conversion.

There are three main types of anatomical variants of renal veins: firstly, multiple renal veins where two or more renal veins can be identified unilaterally or bilaterally; secondly, the retroaortic left renal vein (RLRV), where the renal vein follows a retroaortic course before entering the inferior vena cava; and thirdly, the circumaortic left renal vein (CLRV), where two or more renal veins form a ring around the aorta.

The prevalence of the main anatomical variants of renal vessels varies in the scientific literature. The prevalence of RLRV ranges from less than 1% to nearly 10% in different studies. Reported prevalence for CLRV ranges from less than 1% to over 15%. The prevalence of multiple renal veins (MRVs) ranges from less than 2% to over 40%.

In a study evaluating images from 921 consecutive patients (503 females, 418 males; mean age, 54 years) obtained with multidetector computed tomography (MDCT) for various purposes, it was found that 89.8% of patients had a single renal vein, 10.2% had multiple veins, and 23.8% had a retro-aortic course of the renal vein in terms of venous drainage.

5. Kidney Lymph Drainage

There are three lymph plexuses responsible for the lymph drainage of the kidney: one beneath the renal fascia, one in the perirenal adipose tissue

(pararenal fat pad), and one around the tubular system. The plexus surrounding the tubular system is formed by merging vessels, which then give rise to 3-4 main branches. These branches later join the vessels of the other two plexuses at the renal hilum and follow the renal vein to reach the lumbar lymph nodes (Lumbar lymph nodes or lateral aortic lymph nodes) located lateral to the aorta, from where they drain into the cisterna chyli.

6. Innervation of the Kidney

The vagus nerve carries parasympathetic fibers, while nerves from the lumbar sympathetic trunk bring sympathetic fibers to the kidney, including the lesser splanchnic nerve and the least splanchnic nerve. Within this sympathetic nerve network that reaches the kidney via the celiac plexus around the celiac trunk and additionally the renal plexus around the renal artery, there are several ganglia, the largest being the aorticorenal ganglion. Parasympathetic and sympathetic fibers reach the cells forming the tubular system via the renal arteries and veins. Sympathetic fibers cause vasoconstriction, narrowing the blood vessels and reducing the amount of blood passing through them, consequently decreasing the amount of urine filtered from the blood. Kidney pain is sharp, stabbing, colicky in nature, and is felt throughout the entire lower back region.

7. Clinical Importance of the Kidneys

The kidneys have significant clinical importance and can be classified under several general headings, including congenital kidney anomalies, acute kidney injury, chronic kidney disease, glomerular kidney diseases, tubulointerstitial nephritis, renal vascular diseases, and renal cystic diseases.

Anatomical variations and metabolic disorders (such as hypercalciuria, hyperuricosuria, hyperoxaluria, and hypocitraturia) can lead to the formation of kidney stones (1-3%). Kidney tumors, which occur in approximately 2-3% of cases, are 50% more common in men than in women and are the third most common tumors of the urinary system.

7.1. Congenital Kidney Anomalies

Congenital anomalies of the kidneys and urinary tract are defects arising from abnormalities in the development of the kidneys and their exit pathways, accounting for 20% of congenital defects. Kidney maturation begins at week 3, and nephrogenesis continues until week 36 of gestation, making the kidneys and their exit pathways susceptible to environmental risk factors that disrupt

development during pregnancy. These anomalies can be unilateral or bilateral and may lead to consequences such as urinary tract infections, hypertension, vesicoureteral reflux, and end-stage kidney failure.

Variations in pelvicalyceal structures, presence of accessory renal arteries (30%), and variations in branching patterns of renal arteries and veins are other anomalies observed in the kidneys.

7.1.1. Renal Agenesis

Renal agenesis refers to the absence of kidney and exit system formation unilaterally or bilaterally. It can be detected with high diagnostic rates on second-trimester ultrasonography and generally has a good prognosis. Features observed on ultrasound include lack of bladder filling, absence of kidney tissue, and absence of colored Doppler flow from renal arteries. Studies have found the general prevalence of renal agenesis in the population to be 0.03%. Additionally, hyperuricemia (99.15%), hematuria (39.94%), and proteinuria (39.87%) are the most common symptoms in patients with unilateral renal agenesis. Bilateral renal agenesis is incompatible with life.

7.1.2. Renal Hypoplasia

Renal hypoplasia refers to kidneys being smaller than normal with a reduced number of nephrons. There are three commonly known types of renal hypoplasia characterized by a decrease in the number of kidney lobes: simple hypoplasia, oligomeganephronic hypoplasia (oligomeganephrony), and segmental hypoplasia (Ask-Upmark kidney). A rare and less-known fourth type is cortical hypoplasia, where nephrogenesis is normal but there is a decrease in the number of nephron generations.

7.1.3. Renal Dysplasia

Renal dysplasia is characterized by the presence of cartilage or primitive mesenchymal structures in the kidney tissue, which should not be there. It is an abnormal developmental disease typically diagnosed in the perinatal and childhood years. The prevalence is estimated to be 0.1% in babies (via ultrasound screening) and 4% in fetuses and babies (via autopsy studies). The routine treatment method for dysplastic kidneys is nephrectomy, although conservative treatment requiring close monitoring is preferred. If the patient is asymptomatic and only one kidney is affected unilaterally, periodic ultrasound monitoring is usually conducted, and the healthy kidney on the opposite side should also be closely monitored.

7.1.4. Horseshoe Kidney

Renal fusion anomaly, where two kidneys fuse in various positions, most commonly results in a horseshoe kidney. The majority of cases involve fusion at the lower pole. Fusion is twice as common in males. The fused kidneys are often located lower than normal. Sometimes horseshoe kidneys are associated with ureteropelvic junction obstruction and can clinically manifest as urinary tract infections, abdominal mass, and hematuria in children. Transperitoneal laparoscopic pyeloplasty is one of the alternative treatments that allows better exploration of the pelvicalyceal system and identification of anatomical anomalies such as cross vessels more commonly found in horseshoe kidneys.

7.1.5. Ectopic Kidney

Ectopic kidney occurs when the kidney is displaced to a location other than its normal position due to delayed or absent completion of rotation during the embryonic period. The pelvis is the most common location, while it is very rare to be found in the thoracic cavity. Crossed ectopy refers to the condition where the kidney is located on the opposite side.

7.1.6. Ureteropelvic Junction Obstruction

Ureteropelvic junction obstruction is the most common cause (40-64%) of hydronephrosis, which is observed in approximately 0.2-0.5% of newborns. There are many causes of ureteropelvic junction obstruction, including abnormalities in ureteral adherence, ureteral muscle hypertrophy, peripelvic fibrosis, and abnormal blood vessels passing over the ureter or renal pelvis. Prolonged obstruction can lead to pyelonephritis, hydronephrosis, and renal failure.

7.2. Acute Kidney Injury (AKI)

Acute kidney injury (AKI) is a condition characterized by impaired kidney function or permanent kidney dysfunction associated with irreversible loss of kidney cells and nephrons, which can lead to chronic kidney disease. It encompasses a range of conditions summarized as acute kidney diseases and disorders.

In low- and middle-income countries, infections and hypovolemic shock are the leading causes of AKI. In high-income countries, it predominantly occurs in elderly hospitalized patients and is associated with sepsis, medications, or invasive procedures. AKI related to infection and trauma is common in all regions.

Acute kidney injury is a frequent and serious complication following major surgery, increasing both mortality and morbidity. Identifying high-risk patients, monitoring hemodynamics preoperatively and postoperatively, and controlling hypotension and hypovolemia can reduce the risk of acute kidney injury.

7.3. Chronic Kidney Disease (CKD)

The most common cause of chronic kidney disease is hypertension following type 2 diabetes. Chronic kidney disease is highly prevalent, irreversible, progressive, and is associated with a higher cardiovascular risk.

7.4. Renal Cystic Diseases

Renal cystic diseases are common disorders that can be hereditary, acquired, or developmental. Their differential diagnoses are challenging. They can lead to end-stage renal diseases and may be mistaken for malignant tumors. Examples of renal cystic diseases include asymptomatic simple cysts, multicystic renal dysplasia characterized by large irregular cysts in the kidneys, autosomal dominant polycystic kidney disease (the most common hereditary cystic kidney disease), autosomal recessive kidney disease presenting with sponge-like appearance in the kidney and usually appearing in childhood, and acquired cystic kidney disease characterized by sporadic multiple small cysts developing in patients undergoing long-term dialysis.

7.5. Glomerular Diseases

Examples of primary glomerular diseases include minimal change disease, membranous nephropathy, focal segmental glomerulosclerosis, acute post-infectious glomerulonephritis, IgA nephropathy, dense deposit disease, and C3 glomerulonephritis. Glomerulopathies secondary to systemic diseases include lupus nephritis, diabetic nephropathy, multiple myeloma-associated secondary glomerulopathy, Goodpasture syndrome, microscopic polyangiitis (PAN), Wegener granulomatosis, Henoch-Schönlein purpura, bacterial endocarditis-associated glomerulonephritis, and thrombotic microangiopathy. Hereditary disorders such as Alport syndrome and Fabry disease can also cause glomerular diseases.

7.6. Tubular and Interstitial Diseases

Tubulointerstitial nephritis refers to a group of inflammatory diseases affecting the tubules and interstitium of the kidney. Various factors can cause these diseases, including infections such as acute bacterial pyelonephritis and

chronic pyelonephritis, viral and parasitic infections, toxins such as medications, analgesics, heavy metals, physical factors like chronic urinary tract obstruction and neoplasms, immunological reactions such as transplant rejection, sarcoidosis, and many others.

7.7. Renal Vascular Diseases

Nephrosclerosis, which results from sclerosis of renal arterioles and small renal arteries often associated with hypertension, is an example of vascular diseases. Renal artery stenosis, which occurs most commonly due to atheromatous plaques in the renal artery and, secondarily, due to fibromuscular dysplasia of the renal artery, is more frequent in men and its prevalence increases with age and diabetes.

7.8. Kidney Tumors

Benign tumors of the kidney include renal papillary adenoma, angiomyolipoma, renal oncocytoma, and medullary fibroma. The most important malignant tumor among kidney tumors in adults is renal cell carcinoma, with smoking being the most significant known risk factor. Urothelial carcinoma of the renal pelvis is another example of malignant kidney tumors, with an increased incidence in Lynch syndrome.

7.9. Urolithiasis

Kidney stones can occur at any level but most commonly develop in the renal pelvis. The concentration of the substance forming the stone in the urine is important. Accumulations of substances above a soluble concentration lead to stone formation. Four types of stones are defined: calcium oxalate stones, which account for 70% of renal stones; magnesium ammonium phosphate stones, which can form in alkaline urine; uric acid stones, which develop due to acidic urine; and cystine stones.

7.10. Kidney Surgery and Partial Nephrectomy

In the last few decades, the incidence of kidney tumors has been increasing. Most of these tumors are diagnosed in clinical stage T1 and are suitable for surgical treatment, particularly partial nephrectomy (PN). Nowadays, minimally invasive PN is being applied as an alternative to open PN (OPN).

New approaches based on anatomical surgery include early clamping, segmental clamping, tumor-specific clamping (zero ischemia), and clampless partial nephrectomy. With these new methods of treatment, cancer control can

be achieved while reducing ischemic time, and efforts are made to preserve kidney function to the maximum extent by increasing the remaining kidney volume and quality.

7.11. Kidney Transplantation

With advancements in surgical techniques and immunosuppression, kidney transplantation has become the preferred treatment method for patients with end-stage kidney disease. The management of kidney recipients, both preoperatively and postoperatively, is complex, and due to the need for expensive drugs that suppress the immune system, the patient population must be closely monitored.

7.11.1. Complications of Kidney Transplantation

Complications of kidney transplantation can generally be classified into anatomical and functional complications. While anatomical complications can be easily diagnosed with imaging studies, allograft biopsy is the "gold standard" for functional complications.

Kidney transplant complications are typically classified over time as early (including hyperacute and acute complications), intermediate, and late. Among these complications are perioperative or iatrogenic complications, peri-nephric fluid collections, vascular complications, urinary complications, generalized abdominopelvic complications, allograft rejection, infections, and malignancies.

Early detection of renal allograft complications is crucial for the long-term survival of the graft. Imaging techniques are used in routine surveillance and in the treatment of acute or chronic complications. During the postoperative period, imaging is initially performed using Doppler ultrasound, but additional imaging studies are often necessary. Noninvasive and comprehensive renal functional MRI guides physicians in the early diagnosis and monitoring of renal allograft dysfunction, helping to delay or prevent irreversible kidney damage.

Among the functional complications of kidney transplantation are acute tubular necrosis, rejection (hyperacute, acute, or chronic), and drug toxicity. Hyperacute rejection occurs intraoperatively when the allograft suddenly becomes deprived of circulation. Acute tubular necrosis (ATN) is the most common cause of "delayed graft function" requiring dialysis in the first week. Prolonged ischemia (cold or warm) and reperfusion injury cause ATN. Excised kidneys can be stored at 4°C for 48 hours after perfusion, but cold ischemia lasting more than 24-30 hours increases the risk of ATN. ATN is typically seen in cadaveric grafts and gradually improves within the first 2 weeks. In transplantations from living donors, the shorter duration of cold ischemia reduces

the risk of ATN. The duration of cold ischemia is a significant determinant of delayed graft function.

Acute rejection (AR) typically occurs 5-7 days later and is a result of T-cell activation. Accelerated AR may occur within the first 5 days due to antibody-mediated rejection in patients receiving blood transfusions or with a history of transplantation. Chronic rejection involves a gradual progressive decline in allograft function starting months to years after transplantation.

8. Conclusion

Understanding the variations in the tubular and vascular structure of the kidney, as well as its segmental anatomical features, is crucial for the diagnosis of renal pathologies and determining surgical intervention methods. A detailed understanding of surgical anatomy is necessary to appropriately plan preoperative preparations and select the most suitable surgical approach to achieve beneficial outcomes. In procedures such as partial nephrectomy and kidney transplantation, which are vital for both donors and recipients, the preoperative identification of anatomical variations through radiological methods plays a major role in minimizing intraoperative and postoperative complications.

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CHAPTER V

ANATOMY AND CLINICAL SIGNIFICANCE OF THE ACHILLES TENDON

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1. Introduction

Tendo calcaneus, also known as the Achilles tendon, enables plantar flexion of the foot. Achilles (Greek: Achilleus) is a semi-mortal mythical war god in Greek mythology. His father is mortal and his mother is a water goddess. It is known that Achilles can only be killed if struck on his left heel, which remains untouched by the waters of immortality. Ultimately, he was killed during the Trojan War by an arrow poisoned and shot into his heel. Inspired by this mythological event, the name of Achilles, the strongest warrior of the time, was given to tendo calcaneus, the most robust tendon in the body. (1)

The Achilles tendon (AT) was first described by Hippocrates and named "Tendo Magnus". Hippocrates remarked that if this tendon were injured or ruptured, it would lead to a high fever to the point of unconsciousness, a sensation of suffocation and eventually death. For many years the tendon was referred to as "chorda Hippocraticis" by later writers. In 1693, during a meeting of Anatomy and Surgical Sciences at the University of Louvain in Belgium, Flemish anatomist Philippe Verheyen introduced the term "Achilles tendon" for the first time, officially establishing it in the literature. (2)

The Achilles tendon, the thickest and most robust tendon in our body, can bear loads of up to one ton. However, according to the literature, it is the most frequently injured tendon. Research conducted worldwide indicates that dense soft tissues containing collagen, such as tendons and ligaments, constitute more than half of all musculoskeletal system injuries. Of these injuries, half are related to sports, with the Achilles tendon being the most commonly damaged tendon in sports-related injuries to the lower extremities. In North America, the rate of Achilles tendon rupture can reach up to 9.9%, while the rates of Achilles tendinopathy rise to as high as 2.35 per 1000 individuals. (3-6)

Despite being one of the well-designed structures in our body, there is no universally accepted method for the treatment of Achilles tendon injuries and ruptures. The prevalence of desk jobs leading to a sedentary lifestyle, the increase in obesity, the growing interest in sports activities in modern life, and excessive loading on the Achilles tendon in professional athletes contribute to injuries and ruptures in people of all ages and demographics. Particularly in the young adult group (ages 35-45), the prolongation of the immobilization period for tendon healing leads to loss of workforce and decreased productivity. Therefore, ongoing research focuses on alternative treatment methods, the pros and cons of conservative and surgical treatments, and ways to enhance tendon healing speed. The exploration of new potential treatment avenues maintains the significance of research on the Achilles tendon and paves the way for further studies. (7)

The microcirculation, innervation, internal structure, and structural organization of tendon fibers of the Achilles tendon should be thoroughly investigated, aided by computerized imaging models, to understand the causes of tendon damage in detail. Comprehensive studies should be conducted on the anatomically less understood aspects of the region. (8)

2. General Structure of Tendons

Tendons are fibrous connective tissue structures that connect muscles to bones. Their primary function is to transmit the force generated in muscles to the bones, contributing to the formation of movement around joints. While tendons are generally considered passive tissues, they exhibit a certain amount of elongation during isometric contractions, allowing the muscles to generate force. This elongation is typically about 4% of the tendon's length.

Tendons are stronger than muscles; they can withstand both tensile and high compression forces, supporting loads up to 10-12 times the body weight. Additionally, they contribute to maintaining position by absorbing shock forces,

possessing energy storage capabilities, and playing a role in the perception of proprioceptive sensation. (9,10)

Miyotendinous junction is the term given to the transition zone between the muscle fibers at the proximal end of the tendon and the collagen fibers of the tendon. This junction allows the transmission of the tensile force generated in the muscle fibers to the collagen fibers. During muscle contraction, the miotendinous junction is subjected to significant mechanical stress because the muscle force is transmitted to the tendon. The intricate structure of this region mitigates the tensile forces on the tendon, enhancing its durability. However, this area represents the weakest point in the muscle-tendon structure.

The point of connection between the distal end of the tendon and the bone is called the osteotendinous junction or the tendon insertion site. The tendon can adhere to cartilage or bone tissue. The osteotendinous junction region consists of four parts: the dense tendon region, fibrocartilaginous portion, calcified fibrocartilaginous area, and bone. This unique structure prevents damage and tearing of collagen fibers. (11,12)

3. Embryology of the Achilles Tendon

Extremity development occurs between the 4th and 8th weeks of the embryonic period. Initially, limb buds emerge around the end of the fourth week as protrusions from the ventrolateral body wall. While upper limb buds become visible around the 26th to 27th days, lower limb buds begin to differentiate 1-2 days later.

Each limb bud is surrounded by a tissue known as the mesenchymal mass, which is enveloped by the ectoderm. While the upper limb buds form in the region corresponding to cervical segments, the lower limb buds develop in the regions corresponding to lumbar and upper sacral segments.

During this four-week period, limb buds further develop to form the basic structures of the limbs. The skeletal system develops from the paraxial mesoderm, the lateral plate mesoderm, and the neural crest. This period of extremity development is crucial for forming the bones, muscles, joints, and other tissues. (13)

In the embryonic period, muscles and tendons develop independently. Muscles are formed from myoblasts, where muscle fibers originate, while tendons develop independently within the mesenchyme. Therefore, the connections between tendons and muscles are considered secondary.

In 11 mm embryos, differentiation of the common flexor muscle mass towards the muscle foundations begins to be observed. In 15 mm embryos,

two distinct muscle groups can be easily seen. The first one is the superficial group consisting of gastrocnemius muscle, soleus muscle, and plantaris muscle. This group is located in the lateral proximal region of the leg. The second group includes flexor hallucis longus muscle, m. flexor digitorum longus muscle, popliteus muscle, and tibialis posterior muscle, situated in the medial region. (11)

Later, the lateral and medial heads of the gastrocnemius muscle establish a connection with the blastemal cells of the calcaneus. Simultaneously, the flexor hallucis longus muscle and the flexor digitorum longus muscle interact with the blastemal cells of both the calcaneus and the toes. Subsequently, the gastrocnemius muscle and soleus muscle spread from the lateral to the medial side of the leg to attach to the tibia.

In the second half of the second month of embryological development, especially the lateral head of the gastrocnemius muscle, completes its development by separating. Subsequently, the plantaris muscle separates from the lateral head of the gastrocnemius muscle, completing its development. As the lower ends of the gastrocnemius muscle and soleus muscle enter the tendonization process, mesenchymal cells transform into tendon tissue. Over time, these tissues form the fused Achilles tendon. (14,15)

4. Histological Structure of the Achilles Tendon

The histological structures of all tendons in our body are similar. The Achilles tendon, composed of regular, dense connective tissue, has a bright white color. Due to its fibroelastic structure, it can withstand tensile forces. (16)

It contains a significant amount of collagen fibers. Collagen fibers, comprising approximately 85-90% of the tendon's dry weight, connect to each other to form collagen fiber bundles. The majority of collagen fibers (about 90-95%) involved in fiber organization are type I collagen. Additionally, another common collagen type is type III collagen, an immature variant of type I collagen. Type I collagen has a high stretching ability and can provide elongation of up to about 4% of the tendon length. In damaged and degenerated tendons, type III collagen begins to proliferate. However, type III collagen is not as flexible as type I collagen and cannot stretch as much. (17)

In immunohistochemical studies, apart from these, type II collagen has been identified in the osteotendinous and myotendinous junctions of the tendon. Another study revealed the presence of type X collagen in the tendon structure. Elastin constitutes approximately 2% of the non-collagenous portion, while glycoproteins make up 2-5%. (18)

Between the collagen fibers, there is an extracellular matrix that fills and supports them. This structure provides durability to the tendon and protects it against external damage. The extracellular matrix consists of glycosaminoglycan, glycoprotein, and proteoglycan. The majority (approximately 90-95%) of the cellular structure of the tendon is made up of tenocytes and tenoblast cells responsible for synthesizing collagen and extracellular matrix content. Tenoblasts are the immature form of tenocytes, and when their maturation is complete, they elongate and transform into tenocytes. Tenoblasts have a significantly higher metabolic activity capacity than tenocytes. Tenocytes provide the energy required for their metabolic activities through the citric acid cycle (Krebs cycle).

The remaining part of the cellular structure of the tendon is composed of chondrocytes, endothelial cells of blood vessels, synovial cells, and muscle cells. Collagen fibrils, the fundamental structure of tendons, are wrapped together with nerve and vascular structures. The smallest unit of collagen fibers, the basic building block of this structure, is tropocollagen, a triple helical polypeptide chain. Tropocollagens cross-link with each other to form the insoluble collagen molecule. Collagen molecules come together to form collagen fibrils, which then cluster and come together to create collagen fibers. Collagen fibers run longitudinally and transversely, forming spirals and weaves, which are essential for the strength of the tendon.

All collagen fibers are wrapped with endotenon. These fascicles wrapped with endotenon later form composite collagen bundles. These collagen bundles are enveloped with a double-layered epitenon structure rich in blood vessels and nerves. Finally, all these bundles are packaged with a sheath called paratenon, located on the most superficial part of the tendon. Unlike other structures in the foot and ankle, the Achilles tendon does not have a synovial sheath, making it prone to degeneration. The tendon is surrounded only by the paratenon rich in blood vessels. The paratenon structure allows the tendon to move freely as a whole by separating it from surrounding structures. Typically, there is a mesotenon between the epitenon structure and the paratenon, facilitating the entry of blood vessels into the tendon. (19)

5. Anatomy of the Achilles Tendon

5.1. Muscles

The muscles contributing to the Achilles tendon are located in the posterior compartment of the leg. The muscles, also known as the calf muscles, found in the posterior compartment of the leg (flexor compartment) are divided into two layers by the deep and superficial layers of the fascia transversa profunda cruris.

In the superficial layer, the triceps surae muscle formed by the gastrocnemius muscle and soleus muscle, and the plantaris muscle are present, while in the deep layer, the tibialis posterior muscle, flexor digitorum longus muscle, flexor hallucis longus muscle, and popliteus muscle are located. The Achilles tendon is the common tendon of the soleus and gastrocnemius muscles in the superficial layer. It begins attached to the gastrocnemius muscle proximally in the middle of the leg. Approximately 12-15 cm proximal to the termination of the tendon, both heads of the gastrocnemius muscle start to merge with the soleus muscle. The Achilles tendon receives fibers from the soleus muscle up to its lower end. Tendons originating from both muscles, approximately 5-6 cm above the calcaneus, completely fuse. Thus, the Achilles tendon terminates as a single tendon on the posterior surface of the calcaneus. (11,20)

5.1.1. Gastrocnemius Muscle

The prominence at the back of the leg and the superficial part of the triceps surae muscle form the Achilles tendon. It is the superficial muscle that constitutes the Achilles tendon. It has two heads, medial and lateral. The medial head originates from the medial epicondyle of the femur, while the lateral head originates from the lateral epicondyle of the femur. Generally, the medial head of the gastrocnemius muscle is larger and longer in diameter than the lateral head. Forming a narrow angle and combining, the lateral and medial heads demarcate the popliteal fossa from the lower sides and terminate in the middle of the leg with a broad aponeurosis. This aponeurosis, called the tendon of the gastrocnemius muscle or tendo musculi gastrocnemi, combines with the fibers of the soleus muscle to form the calcaneal tendon, ending at the calcaneus.

While strongly causing plantar flexion of the foot, it also weakly flexes the leg due to its action of crossing the knee joint. Inside the lateral head of the gastrocnemius muscle, there may be a bone called fabella that extends parallel to the tendon fibers. This sesamoid bone, found in approximately 20% of the population, is approximately 15 mm in diameter and round-shaped. Its anterior surface is covered with hyaline cartilage. It serves as an attachment point for various structures in the posterolateral region of the knee (arcuate ligament, oblique popliteal ligament, etc.). It is noticed randomly in imaging procedures. It can cause atypical knee pain and be mistaken for osteochondritis dissecans (joint mouse). (11,21)

5.1.2. Soleus Muscle

Soleus muscle, located in the depths of the gastrocnemius muscle, overflows from the sides of both parts of the gastrocnemius muscle due to its

broader structure. It is separated from the deep-layer muscles by the deep fascia of leg, which is a part of crural fascia. The tibial and fibular portions of the muscle are almost symmetrical. Some fibers of the tibial and fibular parts of the soleus muscle come together to form the arch of the soleus muscle. The tibial nerve, posterior tibial artery, and posterior tibial vein pass through this arch.

The tibial nerve may undergo compression when passing through the arch of the soleus muscle. The compression mechanism is similar to other entrapment neuropathies in the body. Pain and numbness increase during passive dorsiflexion and active plantar flexion. The shape, size, and position of the fibers forming the arcus of the soleus muscle play a role in etiology. The goal of treatment is to relieve the tibial nerve from the compressed area. The examination of the different placements of the muscle fibers of the soleus muscle is a subject of study. A thin connective tissue structure formed by similar fibers in structure and location is called septum mediale solei. Functionally, it is similar to the gastrocnemius muscle but carries different muscle fibers.

The tibial part of the soleus muscle originates from the posterior surface of the head of the fibula, 1/3 proximal to the corpus fibula, the soleal line on the posterior surface of the tibia, and the arch of the soleus muscle between the tibia and fibula. As a variation, the tibial part of the soleus muscle may sometimes be absent, or the accessory soleus muscle may be present between the tendon of the soleus muscle and the tendon of flexor hallucis longus muscle. The fibers of the soleus muscle terminate in the aponeurosis on the posterior surface of the muscle. This aponeurosis, narrowing as it descends, merges with the tendon of the gastrocnemius muscle and forms the Achilles tendon, ending at the calcaneus. The part of the soleus muscle contributing to the Achilles tendon is shorter and thicker. It strongly causes plantar flexion of the foot. (11,22)

The Achilles tendon, formed by the combination of the tendons of the gastrocnemius and soleus muscles, continues to narrow distally and makes an insertion into the posterior part of the calcaneus before ending, slightly widening. The most powerful flexor (plantar flexion) of the foot, the triceps surae muscle is stimulated by the tibial nerve. These muscles are antigravity muscles that contribute to the body's upright posture. They also facilitate the forward propulsion of the body, standing on the toes, and support walking. The formation of the Achilles tendon occurs in different ways. The most common is the fusion of the tendons of the gastrocnemius and soleus muscles approximately 11-15 cm above the point where the Achilles tendon attaches to the calcaneus. In another case, the aponeurosis of the gastrocnemius muscle fuses directly with the fibers of the soleus muscle, forming the tendo calcaneus. Rarely, the tendons of the gastrocnemius and soleus muscles attach separately to the calcaneus.

Sometimes, lateral and medial of the heasd of gastrocnemius muscle attach separately to the tuber calcanei, independent of each other and from the soleus muscle. (23)

5.1.3. Plantaris Muscle

Generally, the plantaris muscle is found just below the gastrocnemius muscle in most individuals. While the plantaris muscle is not a part of the Achilles tendon, it terminates at a common point with the tendon on the calcaneus. It has also been found to penetrate the Achilles tendon, contributing to mid-portion Achilles tendinopathy. (24)

The size of the plantaris muscle can vary, and it may be absent in 7-10% of the population. Originating from lateral lip of linea aspera and oblique popliteal ligament, it continues between the gastrocnemius and soleus muscles in the form of a long tendon, ending on the medial part of the calcaneus in front of the Achilles tendon. It is approximately 5-10 cm long and can be used as a tendon graft in clinical settings. (23)

5.2. Morphological Structure of the Achilles Tendon

The Achilles tendon is described in the literature as a common tendon formed by the gastrocnemius and soleus muscles. However, cadaver dissection studies have shown that anatomists define this structure as individual fascicles attaching to the calcaneal surface. These fascicles come together to form the Achilles complex, undergoing rotational movements as they merge. As a result of this rotation, fibers from the medial head of the gastrocnemius muscle contribute to the posterior, or superficial, part of the tendon. Fibers from the lateral head of the gastrocnemius and the soleus muscle form the deep, or anterior, part. Specifically, the lateral fibers of the gastrocnemius constitute the lateral portion of the anterior region, while fibers from the soleus contribute to the central and medial portions.

The degree of bending of the tendon varies from person to person. Tendon fibers exhibit longitudinal orientation proximally, with noticeable spiraling towards the middle. Rotation is more pronounced about 4-5 cm proximal to the tendon attachment site, and the rotation angle can increase up to approximately 90°. As the tendon approaches the insertion area, fibers progress laterally and distally. Some of the superficial fibers pass beneath the calcaneus, merging with the plantar fascia on the sole of the foot. Additionally, some fibers of the plantaris muscle penetrate into the fibers of the Achilles tendon. The rotational structure of the tendon fibers is a complex arrangement that, when the tendon

is stationary, prevents friction between fibers, hinders deformation, facilitates comfortable elongation of the tendon, enhances durability, and allows the release of stored energy during movement.

Studies examining the rotation of tendon fibers have classified them into three groups based on the degree of spiraling: minimal, appropriate (moderate), and excessive. In cases where the rotation degree is minimal, the tendon may struggle to absorb forces, inefficiently utilize energy, experience earlier wear, and become vulnerable to rupture. A moderate rotation degree is biomechanically advantageous. Conversely, excessive rotation, like insufficient rotation, is clinically disadvantageous. The excessive spiraling of tendon fibers triggers adaptation in the blood vessels responsible for vascularization, leading to compromised circulation. Gender-based distinctions have not been observed in studies investigating the spiraling of tendon fibers, and spiraling is present in all fibers to varying degrees (25-29).

The Achilles tendon is, on average, approximately 15 cm in length, 0.5-0.7 mm in thickness, and around 2 cm in width. However, these values may vary from person to person and depend on daily life and sports activities. The thickness decreases from proximal to distal, but it flattens and widens slightly towards the insertion point, making the narrowest area just above the attachment site. (30,31)

The attachment site of the tendon to the calcaneus is a distinctive region that conforms to the posterior calcaneal prominence in the shape of a crescent. The radii of this region vary between approximately 13.8 and 43.6 mm. The medial part of the attachment site is wider than the lateral part, with an average distance of 1-3.5 mm between them. The unique shape of this region helps to reduce the load and tension on the tendon. Because it is a point where ligaments, bones, bursae, and fat tissues come together, forming a functional integrity, it is considered an “enthesis organ” or “end organ.” (32)

The actual location of the musculotendinous junction differs from the distal attachment point on the tuber calcanei and can be assessed by measuring this difference. In 12.5% of cases, the distance was found to be 0-2.54 cm, in 70% it was 2.54-7.62 cm, and in 17.5% it was more than 7.62 cm. It is also noteworthy that the soleus tendon may extend to a more distal point than the origin, which could be perceived as a partial tear. (33)

The Achilles tendon, unlike other ankle tendons, is enveloped by a paratenon consisting of two layers. It lacks a synovial sheath. The paratenon, an elastic structure, ensures the integrity of the tendon and facilitates its movements. It separates the tendon from surrounding structures, particularly the fascia cruris

located just behind it. Together with the epitenon sheath, this structure, rich in blood vessels and nerves, is referred to as the peritenon, which wraps around the tendon. The peritenon structure exhibits the ability to contract and expand parallel to tendon movements. (34)

To minimize friction between the Achilles tendon and surrounding structures and enable smooth movement, bursae are present around the tendon. The subcutaneous bursa is located between the subcutaneous skin and the Achilles tendon, allowing the skin to slide over the tendon. Another cushion, known as the tendinis calcanei or retrocalcaneal bursa, is situated between the calcaneus and the tendon, preventing the degeneration of the tendon by reducing friction against the bone. The retrocalcaneal bursa has an average thickness of 1.43 mm. In situations where the foot is excessively dorsiflexed or when activities such as prolonged uphill climbing are performed, the retrocalcaneal bursa may become compressed between the tendon and the calcaneus. Damage to this bursa has been associated with Achilles tendinitis. (35)

The limited area between the tibia and the anterior surface of the Achilles tendon is referred to as Kager's triangle. The boundaries of the triangle are formed by the m. flexor hallucis longus in the front and the calcaneus below, with the Achilles tendon at the back. The interior of the triangle is sometimes filled with synovial fluid, extending to the m. soleus and retrocalcaneal bursa, and covered by Kager's fat tissue. The ratio of fat tissue in this region is not the same in newborns and adults. This tissue provides biomechanical support to the tendon, reduces the load on the tendon during loading, ensures the preservation of blood vessels in the area, and helps with proprioception through its nerve fibers.

Kager's fat tissue moves from the triangular area during the plantar flexion movement of the foot, entering between the Achilles tendon and the calcaneus. This action extends the lever arm of the tendon, allowing the load to be transported with less energy expenditure. As a result, Kager's fat tissue plays a role in facilitating the movement of the Achilles tendon and optimizing energy efficiency in the region. (36)

5.3. Vascularization of the Achilles Tendon

Achilles tendon vascularization is provided through musculotendinous junction, osteotendinous junction, and vessels entering the tendon through the paratenon. The paratenon accomplishes a significant portion of the tendon's vascularization. Therefore, it plays a critical role in the healing and overall health of the Achilles tendon.

Apart from the major vessels entering through the paratenon, there are numerous small vessels in the osteotendinous junction that also contribute to nourishing the tendon. These vessels often form a mesh-like structure in the osteotendinous junction.

Since the vessels enter the tendon from the anterior surface, and the number and diameter of vessels on the anterior surface are greater, the anterior part of the tendon is better nourished than the posterior part.

The vascularization of the tendon is primarily divided into three parts. The proximal and distal portions are supplied by the tibial posterior artery, while the middle part is supplied by the peroneal artery. The vascular supply to the middle part of the tendon is weaker compared to the proximal and distal parts. This poorly vascularized area is approximately 5 cm above the calcaneus and is the most vulnerable to issues, making it the region most prone to tendon rupture.

While the vascularization in the middle part of the tendon may be insufficient, studies have focused on the rich vascularization in the osteotendinous junction. (37,38)

Vessels progress in three fundamental directions within the tendon: transverse, longitudinal, and sagittal (toward the depth of the tendon). The rotation of tendon fibers can lead to the rotation of vessels. Surface transverse vessels, being the most numerous in terms of diameter and quantity, contain numerous small longitudinal branches parallel to the tendon fibers.

The portion of the skin structure covering the Achilles tendon that is most vascularized is the medial (inner) section, followed by the lateral (outer) section. The weakest vascularization occurs in the posterior (back) region. While the medial part is nourished by the tibial posterior artery, the lateral part is supplied by the peroneal artery. This circumstance is crucial in clinical practice. In Achilles tendon surgery, to facilitate faster healing and minimize incision marks, the medial section is preferred initially, followed by the lateral section. (39)

5.4. Achilles Tendon Relationship with Sural Nerve

The primary innervation of the Achilles tendon is provided by nerve fibers originating from the sural nerve. The sural nerve is formed by the union of the medial sural cutaneous nerve from the tibial nerve and the lateral sural cutaneous nerve from the common fibular nerve in the distal 1/3 of the leg. It courses along the posterior aspect of the leg, often accompanied by the small saphenous vein. The sural nerve receives sensory input from the distal posterior-lateral third of the leg, as well as from the skin over the medial aspect of the calcaneus and the little toe, known as the lateral dorsal cutaneous nerve. (40)

The sural nerve travels superficially over the Achilles tendon. Consequently, there is a significant risk of injury to the nerve during surgical procedures on the Achilles tendon. Despite awareness of its course over the tendon, postoperative injuries are frequently reported. Considering that damage to the sural nerve can lead to sensory loss and paresthesia, the course of the nerve and its location in the region should be taken into account during surgical interventions on the tendon. Attention should be paid to the various courses and variations of the sural nerve, especially to prevent nerve damage during minimally invasive surgeries. (41)

5.5. Innervation of the Achilles Tendon

The Achilles tendon receives innervation primarily from the tibial nerve, which innervates the gastrocnemius and soleus muscles constituting the tendon, with only minimal innervation from the sural nerve. Due to the deep-seated location of the tibial nerve within the tendon, the likelihood of iatrogenic injury is very low. In contrast, the paratenon surrounding the tendon has a much stronger innervation than the Achilles tendon itself. This is because a significant portion of nerve fibers cannot reach the body of the tendon, terminating as nerve endings on the superficial part of the tendon. Abundant myelinated nerve endings are found in the attachment regions of the tendon, carrying sensations of pressure and tension. Nociceptive sensation, on the other hand, is perceived by unmyelinated nerve endings. In the osteotendinous region, various sensory components are plentiful, including Ruffini corpuscles detecting temperature changes, Pacinian corpuscles sensing pressure, nociceptors perceiving pain, and Golgi tendon mechanoreceptors. These components collectively contribute to the sensory feedback related to cold and heat perception, pressure changes, pain sensitivity, and mechanical tension within the osteotendinous region. (42,43)

6. Biomechanics of the Achilles Tendon

Tendons act as viscoelastic musculotendinous bridges, transferring the force generated by muscle contraction to bones, bridging the gap between muscles and bones. The Achilles tendon, the strongest and thickest tendon in the human body, transmits the force generated by the most powerful plantar flexors of the foot.

The structural characteristics of the muscles, forming the triceps surae, which constitutes the Achilles tendon, are distinct. The soleus muscle contains predominantly slow-contracting (Type I) muscle fibers. This allows it to undertake the role of an antigravity muscle by preventing forward movement of the body in the neutral position (anatomical position). In contrast, the gastrocnemius

muscle contains more fast-contracting (Type II) muscle fibers and contributes to some extent to knee flexion due to its action in traversing the knee joint. Gastrocnemius muscle, with its fast-contracting fibers, provides propulsive force in challenging movements such as jumping and leaping. Therefore, the Achilles tendon is active in maintaining and continuing posture, in all phases of the walking cycle, and during various movements such as running, jumping, and hopping. It is also responsible for stabilizing the foot and ankle joints during movement. (44)

The Achilles tendon can withstand forces 10-12 times greater than body weight. Despite this excessive load, its energy requirement is 7-8 times less than that of muscles, functioning through anaerobic mechanisms. Hence, its metabolism rate is low. The disadvantage lies in the slow healing of the tendon compared to muscles. In vitro and in vivo studies have indicated that the Achilles tendon is subjected to loads of 2600 N during walking, 3800 N during jumping, 1900 N during leaping, and 2200 N during jumping in a squat position. In fast running, the Achilles tendon bears a load of more than 12 times body weight, exceeding approximately 9000 N. This situation can provide insights into the occurrence of Achilles tendon rupture in runners. (45)

The twisted structure of the muscle fibers constituting the Achilles tendon appears clockwise in the left leg and counterclockwise in the right leg when viewed from proximal to distal. This rotational structure provides a mechanical advantage to the Achilles tendon. Consequently, the tendon's durability increases, stored energy within the tendon is released forcefully during movement, and the tendon elongates with less energy expenditure. (27)

Excessive rotational force on the tendon can lead to damage and degeneration. One of the most crucial factors affecting the tendon's durability is the number, volume, organization of collagen fibers, and the condition of the cross-links between the fibers. In a relaxed state, the spiral arrangement of collagen fibers in the tendon straightens when exposed to tensile forces. This response varies based on the stretching ability of collagen fibers. With gradually increasing force, elongation occurs in both the tendon and the fibers. If the tensile force on the tendon is less than 4% of its length, the force is eliminated, and collagen fibers return to their original state. Forces above 4% lead to microdegeneration between collagen fibers. From 4% to an 8% force application, connections between collagen fibers start to break. The tendon can stretch without rupturing up to 8% of its length and return almost the entire force applied to it (92%). Forces exceeding 8% result in visible damage to the fibers. Beyond this point, complete ruptures occur, and the tendon tears. (46)

7. Clinical Significance of the Achilles Tendon

Despite being the thickest and strongest tendon in the body, the Achilles tendon is the most frequently injured tendon according to the literature. This is due to being under very high physiological load and tension. Intrinsic factors such as lower limb imbalances, improper foot positioning, obesity, hyperlipidemia, genetic diseases, varus-valgus deformities, aging, and extrinsic factors like sudden mechanical loading, incorrect or excessive training, sedentary lifestyle, long-distance running, climbing activities, and inappropriate shoe choices are influential in the injury mechanism of the Achilles tendon.

Tendon damage occurs with the loss of mobilization-stabilization balance. An increase in tendon lesions has been reported with a sedentary lifestyle, significant disruption in Achilles tendon circulation, and an increase in cases of tendon degeneration. Tendon injuries constitute more than fifty percent of all soft tissue injuries, with the Achilles tendon being the most affected tendon. Approximately 59% of reported lower extremity lesions are sports-related. (4)

To examine different parts and structures of the Achilles tendon in detail, provide specificity in describing tendon disorders, and establish a common clinical language, the tendon is divided into regions and named accordingly:

Calcaneal Insertion Region: The area where the tendon attaches to the calcaneus, including the closest part of the Achilles tendon to the heel.

Pre-Insertion Region: Located approximately 2 cm above the calcaneal insertion region, just above the area where the tendon attaches to the calcaneus.

Non-Insertional Region: The region that includes the middle part of the tendon, coming after the pre-insertion region.

Intramuscular Region: This region includes the top part of the tendon and is usually covered by muscle tissue.

Free Tendon Region: This region comes before the intramuscular region and represents the uppermost part of the tendon. The free tendon region is further divided into proximal, middle, and distal regions.

This categorization is used to assess different regions in imaging techniques such as MRI or ultrasound when diagnosing Achilles tendon injuries. (29)

7.1. Achilles Tendinopathy

Creating a precise consensus on the nomenclature for pain and degeneration in the Achilles tendon can be challenging. The terminology is often complex, but histologically, the condition characterized by degeneration without inflammation

within the tendon and a mucoid appearance in the degenerative region is referred to as Achilles tendinosis. In cases less frequently observed, inflammation in the tendon body is termed Achilles tendinitis, while inflammation of the paratenon surrounding the tendon is known as Achilles paratendonitis. In recent years, the term "tendinosis" has been used to describe intratendinous matrix disruption. In clinical research and colloquial language, the term "tendonitis" is mostly used. However, the absence of findings of inflammation (inflammatory mediators or cells) in the culture taken from the tendon indicates that the process is non-inflammatory. Tendon pathology may result from overuse, sudden mechanical loading, or other causes. Therefore, the collective use of "tendinopathy" is recommended for tendon degenerations.

In Achilles tendon pathologies, there is a differentiation in the shape and dimensions of the tendon along with pain. Pain in the acute phase is mostly present at the beginning and partially at the end of the activity, while in chronic cases, the pain extends throughout the entire action process. (43)

7.2. Retrocalcaneal Bursitis

Retrocalcaneal bursitis is the inflammation of the bursa located between the calcaneus and the Achilles tendon (bursa tendinis calcanei or retrocalcaneal bursa). It can occur in conjunction with Achilles tendon degenerations, inflammatory arthritic pathologies, or independently. In this pathological condition, the bursa undergoes hypertrophy and adheres to the Achilles tendon.

This condition typically arises due to excessive friction, pressure, or repetitive trauma. Particularly during repetitive dorsiflexion movements of the foot and ankle (such as uphill running), the bursa becomes compressed between the Achilles tendon and the calcaneus. It commonly manifests as pain, swelling, redness, and tenderness in the heel. Pain typically intensifies during movements that stress the foot. Treatment may involve rest, ice application, anti-inflammatory medications, physical therapy, and, when necessary, injections. Surgical methods may be employed in chronic cases. (29,47,48)

7.3. Achilles Tendon Shortening

Achilles tendon shortening can be congenital or acquired. The tendon may be inherently short, or it can become shortened after orthopedic or neurological defects. The most common causes of this condition include cerebral palsy, chronic tendinitis, birth defects, spinal cord injury, leg-foot deformity, repeated trauma to the tendon, and excessive use of high-heeled shoes. This condition can impact daily activities such as walking, running, and jumping, leading to

discomfort. Treatment options include physical therapy, exercise programs, special shoes and orthotics, and surgical interventions. In surgery, the goal is to lengthen the tendon, alleviate symptoms, and facilitate a return to daily activities. Individuals with a slightly short Achilles tendon often find comfort in using heeled shoes. (49)

7.4. Achilles Tendon Ruptures

Achilles tendon rupture is the partial or complete tearing of the Achilles tendon. This type of injury usually occurs due to a sudden and excessive load on the tendon. Degeneration often happens when the foot is dorsiflexed with sudden loading on the front part of the foot, especially during knee extension.

Partial or complete rupture of the Achilles tendon typically begins to occur in young adults around the age of 35-45. It is more common in men than women. In contemporary times, with the increase in sedentary lifestyles, especially among office workers, and the growing interest in sports, the incidence of Achilles tendon injuries has risen. However, the injury rate is significantly higher in athletes compared to sedentary individuals. Sports such as running, walking, athletics involving throwing and jumping, and soccer are the most common areas where Achilles tendon injuries occur. (3,50)

In cases of partial or complete rupture, individuals often experience a sudden shock sensation, intense pain in the heel area, swelling, and restricted movement. Some patients describe a bursting sound in the back of the leg when the tendon ruptures. Many cases of complete ruptures have a history of previous Achilles tendon degeneration. The Thompson test is commonly used for diagnosis. A positive result is the absence of plantar flexion in the foot after compressing the triceps surae muscles. Although this finding is quite reliable, in cases of complete rupture, some degree of plantar flexion may still occur due to the functioning of the tibialis posterior and flexor digitorum longus muscles. To avoid potential misunderstandings in such cases, imaging methods (MRI, ultrasound, etc.) may be employed. Except for these exceptional situations, imaging methods are generally preferred in scientific studies. (50)

7.5. Treatment of Achilles Tendon Ruptures

The treatment of Achilles tendon ruptures varies based on the size and severity of the rupture, the patient’s age, overall health, and whether they are an athlete. Conservative or surgical procedures may be preferred in treatment. In conservative treatment, the foot is immobilized in slight plantar flexion or a neutral position using a cast or orthosis. Surgical intervention can be either

percutaneous or open, depending on the case. In surgical treatment, the presence and superficial course of the sural nerve should be considered to prevent potential complications. Variations in the course of the sural nerve along the Achilles tendon must also be taken into account. (28,51)

Tendon grafts are a crucial modality in the treatment of tendon ruptures. The purpose of the graft is to anatomically repair and restore the tendon physiologically. If the gap between the severed ends of the tendon is less than 3 cm, end-to-end repair is performed. Grafts are preferred for gaps between 3-7 cm. Preferred grafts for Achilles tendon repair include nearby muscle and tissue grafts such as the fascia of the gastrocnemius muscle, the tendon of the plantaris muscle, the tendon of the flexor digitorum longus muscle, the tendon of the flexor hallucis longus muscle, and peroneal tendons. Distant structures involve free-flap applications of the fascia lata or bone strips from the patellar tendon. In cases of delayed diagnosis, treatment, or recurrent complex conditions, allografts or artificial tendon implants are often preferred. Incisions on the skin are made longitudinally in the medial region where the blood supply to the tendon is robust. (52,53)

8. Conclusion

Despite being the largest and strongest tendon in the body, the Achilles tendon is also the most frequently injured. While there are various procedures for the treatment of tendon damage and ruptures, ongoing research on the Achilles tendon continues to be essential, paving the way for new potential treatments for better and more effective healing. Understanding how the Achilles tendon developed to bear loads of up to one ton, its vital importance in body biomechanics, its unique anatomical structure that supports this biomechanics, its relationship with surrounding tissues, and the factors that render the tendon vulnerable to injury should be investigated. A thorough and comprehensive understanding of the anatomy from a clinical perspective is essential for comprehending the pathophysiology of its diseases, diagnostic approaches, and treatment modalities, thereby accelerating the healing process and preventing complications.

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CHAPTER VI

CLINICAL ANATOMY OF PES PLANUS

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1. Introduction

Pes planus is defined as an anomaly of the foot structure characterized by the lowering or complete loss of the medial longitudinal arch (MLA) of the foot. This condition is characterized by symptoms such as valgus (outward turning) of the foot, eversion (inward turning) of the calcaneus, and abduction (opening) of the midtarsal joint. Pes planus deformity can disrupt the mechanical balance of the foot, adversely affecting the biomechanical balance of the entire body and leading to problems in other joint regions. Since the human body is a closed kinetic chain, it should be noted that structural changes in the foot can also affect other joint areas. Therefore, foot anomalies such as pes planus can have significant effects not only on the foot but also on the overall posture of the body. (1,2)

2. Embryological Development of the Foot

The embryological development of the lower extremities begins with a swelling on the ventrolateral surface of the embryo three weeks after fertilization. By the fourth week, buds formed from mesenchymal cells appear on both sides of the embryo's lower part, which are divided into three parts:

thigh, leg, and foot. The foot progresses from the trunk towards the toes during the development process. By the fifth week, paddle-like buds form as growth continues. The footplate develops in a round and flat shape during this period. From the sixth week onwards, joints differentiate from mesenchymal spaces between regions of bone development. By the seventh week, the talus begins to articulate with the tibia, and by the ninth week, all toe digits are fully formed. During the embryological period, the foot begins to turn towards a mid-supination posture. (3)

3. Anatomy of the Foot

3.1. Ossa Pedis

The foot consists of a complex structure of 26 bones and 55 joints. It is divided into three main parts: the forefoot, midfoot, and hindfoot-ankle complex. The forefoot comprises 5 metatarsals and 14 phalanges, while the midfoot is shaped by the fusion of the cuneiform, cuboid, and navicular bones. The hindfoot-ankle complex is formed by the distal ends of the talus, calcaneus, and tibia-fibula bones. Various joints between these bone structures provide connection and movement. (4)

3.2. Articulations of the Foot

3.2.1. Talocrural Joint

The talocrural joint is a hinge-type joint between the distal ends of the tibia and fibula and the talus. This joint has a range of motion of 10-30 degrees of dorsiflexion and between 40-65 degrees of plantar flexion in the sagittal plane. (5)

3.2.2. Subtalar Joint

The subtalar joint is a planar joint between the talus and calcaneus. It allows pronation and supination movements along an oblique axis. (6)

3.2.3. Transverse Tarsal Joint

The transverse tarsal joint (Chopart joint) is a double-planar joint formed by the articulation of the talocalcaneocuboid and calcaneocuboid joints. Eversion and inversion occur in the longitudinal plane, while flexion and extension occur in the oblique plane. Pronation and supination movements occur in both planes. (7)

3.2.4. Tarsometatarsal Joint

The tarsometatarsal joint (Lisfranc joint) involves the proximal cuneiforms and cuboid bones and the distal metatarsal bones. Minimal extension, pronation, and supination movements occur at this joint. (8)

3.2.5. Intertarsal Joints

The talocalcaneonavicular joint is a planar joint formed by the talus, calcaneus, and navicular bones. Sliding movements occur at this joint. The calcaneocuboid joint is also a planar joint with minimal sliding movements. The cuneonavicular joint is a planar joint between the three cuneiform bones and the navicular bone with sliding movements. The cuboidonavicular joint is a fibrous joint with limited movements. The cuneocuboid joint is a planar joint between the three cuneiform bones and the cuboid bone with restricted sliding movements. Intermetatarsal joints are planar joints located between all metatarsal intervals with limited movements. The metatarsophalangeal joints are ellipsoid-type joints located between the distal ends of the metatarsals and the proximal ends of the phalanges. Interphalangeal joints are hinge-type joints between all phalanges except the first, allowing flexion and extension movements. A hinge-type joint is also present between the phalanges of the hallux, allowing flexion and extension movements. (8)

3.3. Foot Ligaments

3.3.1. Ligaments of the Talocrural Joint

In the ankle, there are three groups of ligaments: lateral, medial, and syndesmotic. The lateral collateral ligament complex consists of three important structures: the anterior talofibular ligament, posterior talofibular ligament, and calcaneofibular ligament. The deltoid ligament complex (medial collateral ligament) may show variations and is composed of superficial and deep layers. The superficial layer includes the tibionavicular ligament and tibiocalcaneal ligament, while the deep layer contains the anterior tibiotalar ligament and posterior tibiotalar ligament. The syndesmotic ligament structure is formed by the distal tibiofibular syndesmosis, anterior tibiofibular ligament, posterior tibiofibular ligament, transverse ligament, and interosseous ligaments. (9)

3.3.2. Ligaments of the Subtalar Joint

The joint capsule, weakened by ligaments such as the medial talocalcaneal ligament, lateral talocalcaneal ligament, interosseous talocalcaneal ligament, and posterior talocalcaneal ligament, is preserved. Except for the interosseous

talocalcaneal ligament, the other ligaments are also known as cervical ligaments. This ligament is subjected to traction forces during inversion. (10)

3.3.3. Ligaments of the Transverse Tarsal Joint

The plantar calcaneonavicular ligament (spring ligament) is located between the sustentaculum tali and the middle of the navicular bone. It plays a significant role in supporting the head of the talus from below, aiding in the even distribution of weight. (10)

The long plantar ligament runs between the plantar surface of the calcaneus and the cuboid bone, extending fibers under the metatarsal bones. It contains the tendon of the fibularis longus muscle within its fibers. (10)

The plantar calcaneocuboid ligament (short plantar ligament) extends from the undersurface of the calcaneus to the cuboid bone. (10)

All these ligaments play a crucial role in maintaining the longitudinal arch of the foot.

3.4. Foot Muscles

3.4.1. Intrinsic Muscles of the Foot

During dissection, the muscles are grouped into four layers, depending on the encounter (Table 1-5).

Table 1: Plantar Muscles: First Layer Muscle Name, Origin, Insertion, and Function

Muscle name	Origin	Insertion	Function
M. abductor hallucis	Retinaculum flexorum, tuberosity of the calcaneus, plantar aponeurosis	The medial base of the proximal phalanx of the thumb	Abduction to the thumb
M. flexor digitorum brevis	Tuberosity of the calcaneus, plantar aponeurosis	Divides into four parts, each part splits into two at the base of the proximal phalanges, attaches to the edges of the bodies of the middle phalanges of the lateral four fingers	Flexion to the lateral four fingers
M. abductor digiti minimi	Tuberosity of the calcaneus, plantar aponeurosis	The lateral side of the base of the fifth finger' proximal phalanx	The flexor muscle rather than the abductor muscle of the fifth finger's metatarsophalangeal joint

Table 2: Plantar Muscles: Second Layer Muscle Name, Origin, Insertion, and Function

Muscle name	Origin	Insertion	Function
M. flexor accessorius (quadratus plantae)	Medial head: calcaneus, Lateral head: calcaneus and lig. plantare longum	Lateral border of the tendon of m. flexor digitorum longus	Flexes the lateral four digits by pulling the tendons of the flexor digitorum longus muscle
Mm. lumbricales (4 in total)	Tendon of m. flexor digitorum longus	The dorsal aspect of the phalanges proximalis	Flex the proximal phalanx of digits 2-5 and extend the middle and distal phalanges

Table 3: Plantar Muscles: Third Layer Muscle Name, Origin, Insertion, and Function

Muscle name	Origin	Insertion	Function
M. flexor hallucis brevis (2 head)	Medial head: along the lateral divisions of the m. tibialis posterior Lateral head: cuboideum, cuneiforme laterale	Base of the proximal phalanx of the thumb, both medially and laterally	Flexes the proximal phalanx of the thumb
M. adductor hallucis (It has transverse and oblique heads)	Oblique head: base of 2nd-4th metatarsal bones Transverse head: ligg. metatarsofalangeale of 3rd to 5th ligg. metatarsale transversum profundum, ligg. plantaria, lig. metatarsale transversum profundum	Base of the proximal phalanx of the thumb	Flexes the proximal phalanx of the thumb and stabilizes the basis ossis metatarsalis
M. flexor digiti minimi brevis	Plantar surface of the base of the fifth metatarsale	Lateral aspect of the base of the proximal phalanx of the fifth finger	Flexion of the fifth finger's art. metatarsophalangealis

Table 4: Plantar Muscles: Fourth Layer Muscle Name, Origin, Insertion, and Function

Muscle name	Origin	Insertion	Function
Mm. interossei dorsales (4 in total)	The two heads of the metatarsal bones	Attach to the bases and dorsal expansions of the proximal phalanges	Abduction to the toes and flexion to the metatarsophalangeal joints
Mm. interossei plantares (3 in total)	The bases and medial aspects of the 3rd to 5th metatarsal bones	The bases, medial sides, and dorsal expansions of the proximal phalanges of the corresponding toes	Adduction to the 3rd to 5th toes and flexion to the metatarsophalangeal joints

Table 5: Plantar Muscles: Fifth Layer Muscle Name, Origin, Insertion, and Function

Kas ismi	Origo	Insertio	Fonksiyon
M. extensor digitorum brevis	Calcaneus, lig. talocalcaneum interosseum, retinaculum musculorum inferius	The medial portion of the muscle is named m. extensor hallucis brevis and terminates at the upper portion of the base of the proximal phalanx of the big toe. The lateral portion attaches laterally to the tendons of the m. extensor digitorum longus	The m. extensor hallucis brevis provides extension to the phalanges of the big toe, while the lateral portion provides extension to the phalanges of the second to fourth toes

3.4.2. Extrinsic Muscles of the Foot

In the anterior compartment, the muscles tibialis anterior, extensor hallucis longus, extensor digitorum longus, and peroneus tertius are present; in the lateral compartment, peroneus longus and peroneus brevis; while in the posterior compartment, tibialis posterior, flexor digitorum longus, flexor hallucis longus, medial gastrocnemius, lateral gastrocnemius, and soleus muscles are located. (10,11)

3.5. Vascularization and Innervation of the Foot

The region is supplied by the posterior tibial artery and the dorsalis pedis artery. The posterior tibial artery divides into two branches in the

plantar aspect of the foot: the lateral plantar artery and the medial plantar artery. The lateral plantar artery joins with the terminal branch of the dorsalis pedis artery to form the plantar arch profundus, providing vascularization to the toes. The dorsalis pedis artery, an extension of the anterior tibial artery, courses to the dorsal aspect of the foot, giving off the plantar arch profundus branch to the plantar surface from the midpoints of the first and second metatarsals. There are deep and superficial venous plexuses closely located in the foot. Deep veins accompany arteries and are usually in pairs. Superficial veins begin as a dorsal venous arch over the metatarsals on the dorsal surface of the foot. (12)

On the dorsal surface of the foot, the superficial peroneal nerve provides sensory innervation to a small area between the first two toes; the sural nerve innervates the lateral border, the saphenous nerve innervates the medial region, and the superficial peroneal nerve innervates other areas of the dorsal surface. On the plantar surface of the foot, the tibial nerve provides sensory innervation to the heel, the medial plantar nerve innervates the skin between the fourth toe, the saphenous nerve provides sensory innervation to the skin on the medial side, the lateral plantar nerve innervates the lateral side, and the sural nerve innervates the most lateral part. The tibial nerve also innervates the peroneus profundus muscle. The sural nerve also innervates the lateral gastrocnemius and soleus muscles. (12)

The foot is a highly sensitive structure due to the complexity of its circulatory and nervous systems. Understanding these structures is important for the accurate assessment and treatment of foot and ankle injuries. (12)

3.6. Fasciae of the Foot

3.6.1. Fascia Cruris

The deep fascia of the leg is called the fascia cruris. It extends downward to the foot, forming an extension known as the fascia pedis. The fascia cruris creates fibrous channels through which the tendons of the flexor, extensor, and peroneal muscle groups pass. These channels collectively form five retinacula:

1. Flexor Muscles Retinaculum
2. Superior Extensor Muscles Retinaculum
3. Inferior Extensor Muscles Retinaculum
4. Superior Fibular Muscles Retinaculum
5. Inferior Fibular Muscles Retinaculum (13)

3.6.2. Plantar Fascia

The plantar fascia has a fibrous structure and is divided into three parts: medial, lateral, and central. The central band is divided into five parts to attach to the phalanges. Biomechanically, the plantar fascia is important because during the propulsion phase of the gait cycle, the phalanges extend. During this time, the plantar fascia shortens, elevating the foot arch and reducing energy expenditure. Additionally, it plays an important role in shock absorption and contributes to the stabilization of the foot arches. These muscles, bones, and the plantar fascia support the anatomical structure of the foot arch and can be factors contributing to foot arch deformities such as flat feet and high arches. (14)

3.7. Arch of the Foot

There are three different arches in the foot: the medial longitudinal arch (MLA), the lateral longitudinal arch (LLA), and the transverse arch (TA). The anatomical integrity of the foot arches is important for the effectiveness of foot function and the balanced distribution of loads. Functional impairment of the foot arches can increase the risk of injury. (15)

3.7.1. Transverse Arch

The TA in the foot is examined as the anterior transverse arch (ATA) and the posterior transverse arch (PTA). The PTA extends from the junction of the cuneiform and cuboid bones proximally, while the ATA passes distally between the five metatarsal bones. These arches can also be referred to as the metatarsal arch and transverse arch in different sources, and they are stabilized by the following ligaments and muscles:

- Intermetatarsal Ligament
- Adductor Hallucis Muscle
- Peroneus Longus Muscle
- Tibialis Posterior Muscle (16)

3.7.2. Lateral Longitudinal Arch

The LLA is formed by the junction of the calcaneus, cuboid, and the last two metatarsal bones. It flattens with weight-bearing, increasing the contact area. The ligament plantaris longus plays an important role in maintaining the anatomical integrity of this arch. (10)

3.7.3. Medial Longitudinal Arch

The MLA is the longest arch among the foot arches. It is formed by the junction of the calcaneus, talus, navicular, three cuneiform, and the first three metatarsal bones. The MLA is supported by various muscles, tendons, and fascias. The size of the MLA is important in determining the presence of foot arch deformities such as flat feet and high arches. This arch is actively supported by:

- Tibialis Anterior Muscle
- Tibialis Posterior Muscle
- Peroneus Longus Muscle
- Tibialis Posterior Tendon
- Intrinsic foot muscles

Passively supported by:

- Plantar Fascia
- Plantar Ligaments
- Support from tarsal and metatarsal bones. (17)

3.8. Preservation of Foot Arches

In the preservation of foot arches, the plantar aponeurosis and plantar ligaments play important roles.

Plantar Aponeurosis: It attaches to the tuberosity of the calcaneus and the tuberosity of the fifth metatarsal. It supports the longitudinal arch and assists the plantar ligaments. The attachment of the medial part of the plantar aponeurosis to the sesamoids in the flexor hallucis brevis prevents collapse of the foot arch when standing on the toes. (18)

Plantar Ligaments: The ligamentum plantare longus attaches to the tuberosity of the calcaneus and the plantar surface of the cuboid. It is the primary supporter of the LLA as it entirely encircles the LLA. In the foot, the main supporter of the MLA is the ligamentum calcaneonaviculare plantare (spring ligament). The ligamentum calcaneocuboideum plantare (ligamentum plantare brevis), shorter and deeper than the ligamentum plantare longus, attaches to the anterior end of the calcaneus and the proximal edge of the cuboid. It supports the longitudinal arch while assisting the spring ligament and the ligamentum plantare longus. (18)

3.9. Biomechanical Characteristics of the Foot

The foot and ankle function perfectly through the coordinated action of five distinct biomechanical mechanisms. These mechanisms include: (19)

- Tarsometatarsal mechanism
- Metatarsophalangeal mechanism
- Superior tibiofibular joint mechanism
- Talocrural joint articulation mechanism
- Tarsal mechanism between the distal half of the foot and the lower extremity. (14, 20)

4. Pes Planus

Pes planus is defined as a decrease in the height of the arch during weight-bearing. During weight-bearing, there is a parallel valgus inclination in the hindfoot, a decrease in the arch height, and an increase in the contact area of the foot. The primary issue in pes planus is excessive pronation occurring during standing and walking. Pes planus is observed in 20-25% of adults, with a higher likelihood in individuals with ligamentous laxity and in females. (21)

4.1. Classification of Pes Planus

4.1.1. Classification of Pes Planus in Children

In children, pes planus is classified based on its flexibility into flexible and rigid pes planus. The World Health Organization defines rigid pes planus as a congenital, rigid, or spastic deformity affecting the foot, while flexible pes planus is described as an acquired joint structural abnormality causing valgus deformities in the foot. Flexible pes planus is characterized by a normal MLA when no load is placed on it. Rigid pes planus affects less than 1% of the world's population and typically leads to pain and functional loss requiring surgical intervention. (22)

Although there is no consensus on the mechanism of flexible pes planus formation, abnormal kinematics in the hindfoot (increased range of eversion motion), abnormal kinetics in the foot and ankle (altered loading forces), and changes in physical functions (changes in muscle activation and increased energy consumption) are known to play a role. (23)

Flexible pes planus has been found to be associated with male gender, increased body mass index, and shorter body length. Flexible pes planus

encountered in childhood rarely requires conservative and surgical intervention. However, customized surgical intervention is recommended in patients with congenital vertical talus. (24)

Pathological pes planus has various etiologies such as tarsal coalition, excessive heel valgus, genu valgum, torsional alignment disorders in the lower extremity, excessive body weight, generalized joint laxity, and pain. Therefore, diagnosing pathological pes planus is complex as it varies from child to child. Failure to diagnose this pathology correctly by walking age can lead to difficulties in weight transfer and problems with shoe wear. (24)

4.1.2. Classification of Pes Planus in Adults

The etiological factors of pes planus in adults are generally examined in two main groups: congenital and acquired.

Congenital Pes Planus

Hypermobile pes planus

- Asymptomatic/idiopathic flexible pes planus
- Symptomatic flexible pes planus
- Pes planus associated with generalized dysplasias (Marfan Syndrome, Ehlers-Danlos Syndrome)
- Pes planus associated with accessory navicular bone

Rigid pes planus

- Peroneal spastic pes planus
- Congenital anomalies (Vertical Talus, Tarsal Coalition)

Acquired Pes Planus

Traumatic

- Subtalar joint dysfunction due to fracture or dislocation
- Tibialis posterior tendon rupture Degenerative and inflammatory arthritis
- Rheumatoid arthritis
- Psoriatic arthritis

Neuromuscular imbalance

- Cerebral palsy
- Stroke
- Poliomyelitis
- Diabetes

Other causes

- Occupational factors (prolonged standing, heavy lifting, etc.)
- Obesity
- Inappropriate exercise programs (25)

The most common cause of acquired pes planus in adults is tibialis posterior tendinopathy (PTT). In the etiology of tibialis posterior tendinopathy, inflammatory synovitis, degenerative rupture, repetitive microtrauma, collagen disorders, vascular pathologies, and rarely acute traumas such as ankle fracture, ankle distortion, or PTT rupture after direct impact are involved. In addition, hypertension, diabetes mellitus, and steroid injections around the tendon are identified risk factors. (26, 27)

Tibialis posterior tendinopathy is evaluated in four stages. This classification system was developed by Johnson and Strom, and the fourth stage was later added by Myerson. In Stage I, there is tenderness and paratendinitis findings on the tibialis posterior; no deformity is observed. In Stage II disease, patients exhibit dynamic or flexible flatfoot. The arch is widened, and abduction in the forefoot and valgus alignment in the hindfoot are observed. Stage II is divided into two separate groups, Stage IIA and Stage IIB, depending on the presence of pain in the lateral ankle. Stage IIA is characterized by pain, swelling, and tenderness along the medial aspect of the arch and PTT. In Stage IIB, existing deformities are more advanced, accompanied by increased pain. When progressing to Stage III, the valgus deformity in the hindfoot becomes fixed, often accompanied by compensatory varus alignment in the forefoot. Stage IV, as described by Myerson, involves deltoid ligament insufficiency with valgus alignment within the mortise and potential lateral tibiotalar arthritis. (26, 27)

4.2. Prevalence of Pes Planus

The true prevalence of pes planus is uncertain because it is not defined by specific clinical or radiographic criteria. Studies conducted worldwide indicate that pes planus occurs in 5% to 14% of the adult population. A study conducted in 2009 with male schoolchildren in Istanbul reported a prevalence rate of 0.69% for pes planus. (28)

It is known that pes planus is more common in overweight children in Asian countries and is observed equally regardless of gender. (29)

In a study conducted on 667 children in Iran, the prevalence of pes planus was found to be 19%, but it was observed that 10.5% of all children had pathological findings. However, they also found that the prevalence of pes planus decreases with age and is not influenced by gender. (24)

In the United States, it is mentioned that there are 3 million adult individuals with acquired pes planus, while in the United Kingdom, pes planus is reported to be present in 3% of women over 40 years old. (27)

Tendon problems of the tibialis posterior muscle affect 10% of geriatric patients. It is suggested that the geriatric population may be more prone to acquired flatfoot in adults due to degeneration in muscle mass and bone structure. (27)

In another source, it is stated that the prevalence of pes planus is over 3% in women over 40 years old and over 10% in all adults over 65 years old. (26)

4.3. Biomechanics of Pes Planus

For balanced walking, appropriate weight distribution, sufficient support, wide contact, and stability are required. In standard walking biomechanics, weight transfer occurs parallelly in the subtalar and midtarsal joints. In healthy individuals, the decrease in the arch height during walking is stabilized by muscles and ligaments. However, increased pronation in pes planus leads to locking at the subtalar joint and excessive mobility at the midtarsal joint during walking. In this situation, weakness of the tibialis posterior muscle is observed to provide stability in the supination of the subtalar joint and in the midtarsal joint during the push-off phase. Therefore, the propulsive force produced by the plantar flexor muscles affects the midtarsal joint rather than the metatarsal heads. As a result, compared to the m. tibialis posterior, the weaker m. peroneus brevis brings the heel into eversion and causes the calcaneus to turn into valgus. (30, 31)

The rise in pronation encountered in standing forces the tibia into medial rotation, leading to deformities affecting the lower extremity bones such as the femur, patella, ilium, and sacrum. This affects the lower extremity due to imbalance in weight distribution, increased stress on the foot, and the kinetic chain during walking. Pes planus also affects the tibiofemoral connection by increasing the Q angle. (30, 31)

Instability may occur due to insufficient supination, and the rise in pronation in the foot makes the midfoot hypermobile. Additionally, negative changes in body posture may occur depending on feedback and flow strategies from the hip and knee. Navicular drop results from pes planus, causing stretching of plantar connective tissues. In this case, the weight on the plantar intrinsic muscles and m. tibialis posterior increases, leading to muscle fatigue and unnecessary power usage. (30, 31)

Various methods are used in the evaluation of pes planus, including visual inspection, foot anthropometric measurements, radiography, and footprint

analysis. Among these, the most common are visual inspection, radiography, and footprint analysis. Additionally, special tests such as the navicular drop test and the Feiss line method are used to assess the degree of pes planus. (30, 31)

There are various strategies for treating pes planus, including arch taping, foot exercises and plantar intrinsic muscle training, orthotic use, appropriate shoe selection, and surgical treatment. These treatment methods help reduce pain, correct imbalance, and improve quality of life. However, not all individuals with pes planus may require treatment, and treatment methods should be determined based on individual needs. (30, 31)

4.4. Evaluation of Pes Planus

4.4.1. Visual Inspection

Visual inspection methods are traditional and common approaches used to assess foot structure. These methods include direct visual examination, examination of foot photographs, and evaluation of footprints.Formun Üstü

Formun Üstü

Direct visual examination: Foot morphology and height are observed from anterior, posterior, and lateral views during weight-bearing and movement. Although this method is easily applicable, it is a subjective and non-quantitative evaluation method. (32)

Visual examination of foot photographs: Researchers take photographs of the foot to examine its structure in more detail. These photos can be taken using vertical mirrors or a special foot photography box. This method provides higher validity and reliability compared to other visual inspection methods. (33)

Visual assessment of footprints: Footprints are examined by simple ink imprint analysis on paper. These imprints, formed by bearing weight on the foot, can indicate the size of the foot structure. However, this method may lead to some discrepancies and incorrect classifications compared to caliper measurements. (34)

4.4.2. Anthropological Measurements of the Foot

Anthropological measurements of the foot allow for the measurement of foot structure in the sagittal plane. These measurements are performed using tools such as a measuring tape, ruler, or specially designed arch height index measurement. These measurements are used to assess foot structure in more detail. (35)

4.4.3. Calculation of Arch Height Index (AHI)

The calculation of the arch height index (AHI) is a method used in the evaluation of foot structure. This calculation is performed using various measurement tools during weight-bearing and non-weight-bearing. One of the most reliable methods is AHIMS. (35)

4.4.4. Navicular Drop Test and Feiss Line Method

The navicular drop test and Feiss line method are important methods used to determine the degree of pes planus. The navicular drop test involves measuring navicular height during weight-bearing, while the Feiss line method is used to assess proper foot positioning. If the scaphoid tubercle has dropped by one-third of the distance between the Feiss line and the ground, it is interpreted as first-degree pes planus; if it has dropped by two-thirds, it is second-degree pes planus; and if it reaches the ground completely, it is third-degree pes planus. (36)

4.4.5. Radiographic Examination

X-ray images of the foot are used for observation and/or quantitative analysis and are an important tool for categorization purposes. This method is known for its high reliability level. X-ray evaluations can also be used to assess the appropriateness of data collected from other observation-like methods. However, radiography has some limitations, including radiation exposure, accessibility, and high cost. (37)

4.4.6. Footprint Analysis

Analysis of the portion of the foot in contact with the ground can be performed using simple ink on paper and pressure analysis systems such as the Tekscan Pressure Sensor System. However, there is debate about the reliability of footprint analysis. Some studies show that this method is reliable, while others claim it is not. (38)

4.4.7. Three-Dimensional (3D) Motion System (Oxford Foot Model)

The 3D motion analysis system used to evaluate foot dynamics and foot type is supported by synchronized cameras through markers placed on the foot dorsum. This system was introduced by Wright, Arnold, Coffey, and Pidcoe (2011). (39)

4.4.8. Other Methods

Various methods, including limited foot criteria used to evaluate foot models, are available. Among these methods are more complex techniques such as 3D scanning of the feet and fluoroscopy studies. While these methods may have higher accuracy and sensitivity, they may face practical challenges such as affordability and accessibility. (40)

4.5. Treatment Strategies for Pes Planus

Pes planus is known to negatively affect balance, cause fatigue and pain in the lower extremity muscles, and increase the risk of injury. Therefore, treatment strategies that improve the quality of life in these patients are important. However, not all cases of pes planus may require treatment. (41)

4.5.1. Arch Taping

Arch taping is among the methods that provide continuous support often combined with analgesic medication. Different types of tapes such as elastic, non-elastic, or kinesio tapes can be used. Depending on the type of tape, support in the foot, muscle activation, and plantar pressure distribution may vary. The use of non-elastic tapes may reduce pain but does not alter the amount of pronation in the hindfoot. Kinesio tape may assist in correcting deformities by providing proprioceptive feedback and is considered more suitable than other taping methods. (42)

4.5.2. Foot Exercise and Plantar Intrinsic Muscle Training

It is known that exercises strengthening intrinsic foot muscles support pes planus treatment. Research has shown that these exercises effectively reduce pain by altering pressure distribution in the foot. Additionally, it has been noted that intrinsic muscle strengthening training over a period of 4 weeks significantly improves balance and reduces navicular drop. (43)

4.5.3. Use of Insoles

Insoles support the improvement of balance in cases of pes planus, even in individuals with a normal arch structure. Proper insole use can be beneficial in pes planus treatment as it reduces talocalcaneal joint eversion. It has been found that insoles reduce pain in pes planus cases and positively affect quality of life through static and dynamic plantar pressure analysis. (44, 45)

4.5.4. Foot Orthotics

Foot orthotics ensure proper alignment of foot bones and support foot function. They are commonly used in pes planus treatment, especially for individuals with flexible pes planus. Foot orthotics are used to correct calcaneal eversion and allow the abductor hallucis muscle to reach its physiological length. Thus, they improve bone alignment and foot function. Additionally, these orthotics increase sensory input on the medial side of the foot, thereby maintaining postural stability. (46)

It is known that using foot orthotics for two months improves abductor hallucis muscle strength in pes planus cases. While studies show that foot orthotics reduce pain in pes planus cases, some studies indicate they may not be effective in reducing pain. Orthotic use may be beneficial, but its long-term effects remain uncertain. (46)

4.5.5. Selection of Proper Footwear

The effect of proper footwear selection in controlling pes planus deformity is not clearly understood. However, there are studies that radiologically prove the increase in medial longitudinal arch height with the continuous use of contoured sandals. Individuals with MLA deformities can use specially designed footwear and insoles for pes planus. Further publications are needed to clarify the issue of proper footwear selection in cases with MLA deformities. (47)

4.5.6. Surgical Treatment

Asymptomatic patients do not require any treatment. For symptomatic patients, among the conservative treatment options that can be applied in the first step are activity modifications, stretching exercises, manipulations, serial casting, weight loss, use of analgesics for pain and inflammation, footwear changes, footwear modifications, and use of foot or ankle orthoses. Activity modification, ice applications, and the use of nonsteroidal anti-inflammatory drugs can be beneficial, especially for patients with complaints of pain due to overuse and those with advanced deformities. In cases resistant to conservative treatment, the decision for surgery is made based on the etiology of pes planus. (48)

In symptomatic patients, studies advocate not avoiding surgery if there are no contraindications such as morbid obesity, excessive smoking, or non-cooperative patients, while other studies suggest that any intervention aimed at correcting pes planus should only be performed due to severe pain and only when conservative treatment is inadequate, and never for cosmetic reasons. (48)

Surgical intervention may be necessary in cases of severe pes planus with progressive symptoms and complications. Surgical intervention helps significantly improve foot structure and function in severe cases and prevents the progression of deformity. Relaxation of the plantar fascia and calcaneal osteotomy reduce stress on foot ligaments and bones, improving foot function. (49)

The goal of surgical treatment is not cosmetic but to alleviate resistant and unresponsive pain. It aims to correct Achilles shortening, restore the arch of the foot, and correct excessive abduction of the forefoot. Soft tissue and bone surgeries can be performed. Soft tissue surgeries include:

- Achilles tendon lengthening.
- Tibialis posterior plication.
- Moving the tibialis posterior tendon distally.
- Transfer of the flexor digitorum longus muscle.
- Lengthening of the peroneal tendons.
- Talo-navicular capsulorraphy.

  Bone surgeries applied in flexible pes planovalgus include:
- Medial cuneiform flexion osteotomy.
- Lateral column lengthening surgery.
- Posterior calcaneal sliding osteotomy.
- Transverse calcaneal osteotomy.

  In addition, arthrodesis surgeries may rarely be necessary:
- Extra-articular subtalar arthrodesis (Dennyson Fulford, Green Grice).
- Isolated arthrodesis of the navicula-medial cuneiform.
- Medial column arthrodesis.
- Triple arthrodesis. (48)

5. Impact of Pes Planus Deformity on Other Joints

Looking at the studies in the literature, it has been found that individuals from different occupational groups become more prone to injury when foot biomechanics change and normal walking patterns are disrupted. (50, 51)

One of the most common problems related to pes planus is excessive pronation occurring during standing and walking. While this excessive pronation may manifest with mild discomfort during standing, it has been emphasized that it can lead to pain during walking. Furthermore, patients with pes planus deformity have been shown to experience twice as much knee and back pain caused by arch collapse compared to healthy individuals. (50, 51)

In a study examining the relationship between pes planus and body mass index (BMI) in sedentary female and male individuals, and its impact on quality of life and pain levels, individuals with pes planus deformity were found to have a higher BMI compared to those without pes planus deformity. Additionally, it was observed that static and dynamic body balance was negatively affected in individuals with pes planus deformity. (52, 53)

5.1. Articulatio Metatarsophalangealis

The relationship between pes planus and hallux valgus, a deformity occurring at the first metatarsophalangeal joint, has been a debated topic from the past to the present.

Hallux valgus refers to the lateral deviation of the big toe due to an imbalance between abductor and adductor muscles. Untreated, it progresses and disrupts the biomechanics of the entire foot, starting with medial deviation of the first metatarsal bone, thus becoming an orthopedic condition. (54)

Some angles between the foot joints are used in the detection of hallux valgus deformity. It has been found that the calcaneal pitch angle and talonavicular angle are less in both hallux valgus and pes planus deformity patients compared to patients with only hallux valgus deformity, and a negative correlation between hallux valgus deformity and these two angles has been identified. Therefore, a strong relationship between pes planus and hallux valgus has been demonstrated. (55)

5.2. Articulatio Talocruralis and Subtalaris

Some studies in the literature have shown that longitudinal arch collapse facilitates ankle injuries. (56)

5.3. Articulatio Genus

It is believed that the pes planus deformity can adversely affect muscle contraction mechanics and angle of traction by incorrect sensory input, thereby disrupting lower extremity alignment. (2)

In a study, a relationship between pes planus deformity and the Q angle (patellofemoral angle) at the knee was found, suggesting that the Q angle is an important factor in identifying potential risks affecting the foot sole. (57)

Various studies on sedentary individuals with pes planus in the literature have indicated that the valgus position of the hindfoot changes the contraction pattern of muscles abducting the hip and increases the Q angle at the knee, leading to increased loading on the lateral patellar facet and increased internal

rotation of the hip. Studies have also indicated a negative correlation between decreased ALM height and knee valgus. (57, 58)

In a study on body mass index, pes planus, and knee pathologies, it is believed that knee problems result not from increased load due to excess weight or biomechanical alterations caused by obesity but rather from the greater influence of body weight on the arch of the foot, which disrupts the alignment of the entire lower extremity, potentially leading to pathological problems in the knee. (59)

5.4. Articulatio Coxae

As mentioned earlier, the increased Q angle in the knee due to pes planus deformity leads to increased hip internal rotation angle.

Moreover, increased subtalar joint pronation associated with pes planus deformity has been found to lead to medial traction and lateral compression stress on the knee joint and increased internal rotation angle at the hip joint. Additionally, the increased Q angle in the knee has been shown to be associated with excessive anterior pelvic tilt and excessive tibial torsion. (60, 61)

A study examining the effect of pes planus on lower extremity biomechanics found that individuals with pes planus had a greater pelvic inclination angle on the non-dominant side compared to those without pes planus, which could lead to lower back pain, and they also had higher Oswestry Disability Index scores. (62)

5.5. Articulationes Columnavertebralis

Since pes planus deformity can disrupt the biomechanics of the lower extremity and increase anterior pelvic tilt and hip internal rotation angle, the changes that occur here can also affect the biomechanics of the vertebrae immediately above the pelvis, which are closely related to the pelvis.

According to studies examining the relationship between pes planus and anterior knee pain and back pain, individuals with pes planus deformity tend to experience more frequent anterior knee pain and back pain. (63, 64)

Bilateral foot pronation is known to increase lumbar lordosis. Pes planus is almost always associated with some degree of foot pronation. In a study investigating the relationship between pes planus and lordotic angles in individuals with detected pes planus deformity, postural deformity due to increased lordosis was found to be significantly more pronounced in patients with pes planus deformity. (30)

The lower extremity internal rotation caused by pes planus is believed to lead to an increase in anterior pelvic tilt and, consequently, an increase in lordosis in the lumbar and cervical regions and an increase in kyphosis in the thoracic region. (65)

6. Conclusion

As foot health is relevant to overall body health, it is important to take foot conditions seriously. Disproportionate distribution of load to muscles, tendons, and joints can lead to undesirable consequences over time. Despite appearing simple, pes planus is a biomechanically unfavorable condition. Flat feet, which can impair mobility and negatively impact quality of life, can occur in both children and adults. Failure to provide pes planus treatment, even when symptoms are present, can negatively affect many muscles and joints in the body.

While asymptomatic patients may not require treatment, conservative treatment options may be considered as a first step in symptomatic patients. In symptomatic patients who do not respond to conservative treatment, surgery should not be avoided if there are no contraindications such as morbid obesity, excessive smoking, or noncompliance. The appropriate surgical approach should be determined based on the underlying cause and the degree of deformity.

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CHAPTER VII

KNEE JOINT ANATOMY AND CLINICAL SIGNIFICANCE

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1. Introduction

The knee joint, formed by the femur, tibia, and patella bones, is the largest synovial joint in the body. It is equipped with numerous ligaments, both inside and outside the joint, which provide stability and control movement. Many bursae, both connected and unconnected to the synovial membrane, surround it. With the consideration of menisci and muscles crossing the joint, it exhibits a highly complex structure. It is also one of the most commonly injured joints in clinical practice. Managing its injuries becomes quite challenging due to its complex structure. Understanding injury mechanisms, making accurate diagnoses, providing appropriate treatment, and minimizing surgical complications require clinicians to have a thorough understanding of the joint's detailed anatomy.

2. Knee Joint Embryology

During embryological development, the human body develops from proximal to distal and from cranial to caudal. Accordingly, lower extremity joints develop later compared to upper extremity joints (1).

During intrauterine life, the buds of the lower extremities begin to appear around the 27th to 28th days. These buds contain blastema, a group of cells derived from mesenchymal cells, on the inner side, while they are surrounded by an ectodermal sheath on the outer side. Blood vessels, bones, and tendons

develop from the blastema. By the fifth week, the blastema begins to undergo chondrification to form cartilage for bone formation, and the bone segments enveloped by the perichondrium undergo transformation between the 8th and 12th weeks. Subsequently, to form the interzone (which will later form the joint), mesenchymal tissue proliferates to form organized rings at the ends of developing bones that will constitute the joint (1).

The embryological development described above occurs in the following sequence: condensation, chondrification, formation of interzones, formation of synovial mesenchyme, and formation of the joint cavity. Condensation occurs after the formation of limb buds and involves an increase in the number of mesenchymal cells, leading to their clustering, which is essentially the formation of blastema. During chondrification, the blastema divides into cartilage precursors to form bones. The chondrified areas contain mesenchymal cell regions devoid of blood vessels, which constitute the interzonal mesenchyme that will form the joint (1).

Subsequently, this region of mesenchyme differentiates into a tissue containing fibroblasts by forming three layers. The cells in the middle layer transform into connective tissue and secrete an extracellular matrix rich in polysaccharides. The other two layers form cellular layers to produce joint cartilage in the long term. From the peripheral part of the interzone, the synovial mesenchyme, rich in blood vessels, develops (1).

The formation of the joint cavity (cavitation) begins from the central interzone, where small cavities initially form (1,2). These small cavities then merge to form a complete joint cavity. The process of cavitation can initially be detected in the femoropatellar interzone, followed closely by its appearance in the femoromeniscal interzone (2). The formation of the joint cavity occurs approximately at the same time as the transformation of the synovial cell layer (1).

Knee joint, reaches its adult appearance except for cavitas articularis by the 8th week of intrauterine life. By the end of the second month, synovium villi become visible (1). According to a study conducted in 1997, during the 9th week of intrauterine life, the patella is positioned opposite the lateral femoral condyle and primarily articulates with it. Additionally, menisci can be observed attached to the capsule with ligaments. At weeks 10-11, the tibiofibular joint space is observed, which is connected to the lateral meniscotibial space. By weeks 12 to 13, this connection disappears, and the joint cavity attains its adult appearance. In the same study, it was observed that initially the joint cavity consisted of one femoropatellar and two femoromeniscal cavities, followed by

the development of two additional meniscotibial cavities (2). Additionally, it has been reported that bursae develop between the 3rd and 4th months, and fat pads develop between the 4th and 5th months (1).

3. Knee Joint Anatomy and Biomechanics

The knee joint essentially consists of two articulations: the patellofemoral joint and the tibiofemoral joint (3). The tibiofemoral joint is a highly complex joint formed between the concave articular surfaces of the tibial condyles and the convex articular surfaces of the femoral condyles (4,5). Being the largest joint in the human body, the knee joint has the highest amount of synovial fluid and the largest joint cavity (4). The knee joint is frequently subjected to trauma and pathologies. Factors explaining this include its prominent position in vehicular accidents, being the first area to contact the ground during falls, and the anatomical inadequacy of tibia in protecting the joint due to the bones involved. While the joint surfaces are susceptible to dislocations, the robustness of the ligaments results in fewer occurrences of dislocations (5).

3.1. Tibiofemoral Joint

It is a bicondylar type joint. (4,5). The presence of rotational capability alongside flexion-extension movement distinguishes this joint from the ginglymus group of joints (3,4). The reason for this difference lies in the shape of the convex articular surface. The convex articular surface is formed by the condyles of the femur. This convexity is both transverse and sagittal. When viewed laterally, the posterior part of the joint surfaces is more convex and spherical. The articular surfaces of the condyles, which are divided by the intercondylar fossa at the back, meet in the front. There is a groove that divides this surface, which articulates with the patella, into outer and inner parts. The inner surface is narrower than the outer surface, and the patellar joint surfaces are adapted to these dimensions (5).

The medial condyle of the femur is larger compared to the lateral condyle (3,5). As the femur descends from top to bottom, it follows a slightly inclined course from outside to inside. Additionally, the inner condyle of the bone is more distal compared to the outer one. The articular surfaces of the tibia, which will articulate with it, are located in a horizontal plane. Besides the inclined extension of the femur, the difference in size of the condyles is important for ensuring contact with the articular surfaces of the tibia and for aligning the joint surfaces in the same plane (5).

The concave surface of the joint is formed by the condyles of the tibia. Among these surfaces, the one located on the outside is smaller and more rounded (3). The one on the inside is more oval-shaped, with its long axis extending anterior-posteriorly. Both articular surfaces rise slightly on the medial side and form the lateral and medial intercondylar tubercules (5).

The concavity-convexity degrees of the tibia and femur are not perfectly aligned. Therefore, complete contact of the joint surfaces is not possible. The area where the two bones can come into contact is limited by the free inner edges of the menisci. The menisci fill the areas where contact cannot be achieved (5).

In 90 degrees of flexion, the ligaments are in their most relaxed state, and approximately a 40-degree rotation movement can be performed. In full extension, the ligaments are taut, and no rotational movement is observed. After 20 degrees of flexion, the ligaments begin to loosen, and some rotational movements can be performed (6).

Knee Joint Screw-Home Mechanism: During the final 30 degrees of extension, the tibia undergoes approximately 10 degrees of external rotation. The structures primarily responsible for this movement are the compressive force of the condyles and the ligaments of the joint. Disruption of this mechanism following injury leads to excessive loading on the joint. Conversely, the opposite occurs at the beginning of flexion (6).

3.2. Patellofemoral Joint

The joint between the patella and the femoral condyles is a saddle-type synovial joint. As the largest sesamoid bone in the human body, the patella serves two important functions. One is to extend the lever arm by moving the quadriceps muscle away from the joint center and thereby providing a torque and mechanical advantage. The second function is to distribute the stress on the patellar tendon that begins to contact the femoral condyles after approximately 72 degrees of flexion. Without the patella, the quadriceps femoris would not be able to generate sufficient torque and could not lock the knee joint. During the stance phase of walking, if sufficient extension torque cannot be generated, the individual will try to lock the knee joint either by using their hands or by leaning their body forward to shift the center of gravity forward, thus attempting to lock the knee joint with the effect of the generated moment (6).

The alignment between the patella and femur is crucial during flexion and extension. Initially, during the first 10-20 degrees of flexion, the patellar

articular surface is located distally. As flexion increases, the contact surface shifts proximally and laterally. Any anatomical abnormalities of the two bones and disruption of their alignment can lead to altered distribution of the load on the patellofemoral joint and cartilage degeneration (3).

3.3. The Ligaments of the Knee Joint

The ligaments of the knee joint can be listed as follows:

1. Joint capsule
2. Patellar ligament
3. Oblique popliteal ligament
4. Arcuate popliteal ligament
5. Tibial collateral ligament (Also known as medial collateral ligament)
6. Fibular collateral ligament (Also known as lateral collateral ligament)
7. Anterior cruciate ligament (ACL)
8. Posterior cruciate ligament (PCL)
9. Lateral meniscus
10. Medial meniscus
11. Transverse ligament of the knee

3.3.1. Extracapsular Ligaments

The articular capsule consists of two layers: the fibrous membrane and the synovial membrane. The fibrous membrane is primarily composed of strong and thin fibers but becomes a complex and stronger structure with the addition of certain tendons and ligaments around it. The fibers joining the fibrous capsule from the outside do not distribute evenly in every part. Therefore, the fibrous capsule varies in thickness and strength in different areas (5).

The posterior portion of the fibrous capsule is reinforced by the tendons of the gastrocnemius muscle at their origins and the oblique popliteal ligament (from lateral to medial, from top to bottom). The capsule is fused with the tibial collateral ligament in the posterior-medial area. However, there is no tight fusion with the fibular collateral ligament on the outer side. As it passes from the posterior-lateral to the anterior side, it attaches to the tendon of the popliteus muscle. Between the capsule and the fibular collateral ligament, there are sensory fibers leading to the joint capsule, the lateral inferior genicular artery, and adipose tissue. In the anterior-medial aspect, the fibrous capsule is reinforced by fibers from the tendons of the semimembranosus and sartorius muscles. Here, along with the joint capsule,

the tibial collateral ligament attaches to the medial meniscus and from there to the upper edge of the tibia. This arrangement restricts the movements of the medial meniscus. These fibers between the tibia and the meniscus are also known as the coronary ligament. In the anterior-lateral region, some fibers from the iliotibial tract extend between the oblique popliteal ligament and the fibular collateral ligament (5).

The fibrous capsule is not present at the location of the patella and its upper portion. However, there are two structures known as the lateral patellar retinaculum and medial patellar retinaculum, which extend on both sides of the patella as continuations of the tendons of the vastus medialis muscle and vastus lateralis muscle. These retinacula, which attach to the patellar ligament at one end, are fused with the joint capsule. Additionally, the iliotibial tract sends fibers that strengthen the lateral retinaculum (5).

The synovial membrane attaches to the upper edge of the patella at the front and passes beneath the quadriceps femoris muscle on both sides. It then extends by lining the inside of the fibrous membrane and progresses to where it merges with the menisci. Beneath the patella, there is a mass of adipose tissue called the infrapatellar fat pad, situated between the synovial membrane and the patellar ligament (5). This fat pad can be entirely or partially removed during total knee arthroplasty. Being extracapsular and intracapsular, this fat pad is rich in vascular and neural tissue (3). As the synovial membrane passes through, it folds to form folds on both sides of the patella. Both of these folds are called alar plica (4). These formations later combine to form the infrapatellar plica, which extends to the intercondylar fossa (5). The folds are effective in maintaining the position of the infrapatellar fat pad (3).

3.3.1.1. Patellar Ligament

Some authors consider the patellar ligament as a continuation of the tendon of the quadriceps femoris muscle (4,5). Others, however, discuss this connection separately and mention that fibers from the middle portion of the quadriceps femoris muscle contribute to the structure of this ligament. It extends from the lower part and surroundings of the patella to the tuberosity of the tibia. The lateral and medial patellar retinaculum on the sides of this ligament are formed by the vastus lateralis and medialis, respectively. The joint capsule is weak on both sides of the patellar ligament, and due to the negative pressure inside the joint, this area may appear concave when viewed from the outside. However, in situations where there is an increase in pressure inside the joint, these concave areas disappear and may even be observed as elevations. This

observation is important in clinical inspection as it can provide information about pathology (5).

3.3.1.2. Oblique Popliteal Ligament

It originates from the medial condyle of the tibia at the back of the joint and extends upward and outward. At the top, it attaches to the lateral condyle of the femur and the intercondylar line. Some fibers from the semimembranosus muscle contribute to the superficial portion of the ligament. Just posterior to it lies the popliteal artery (5).

3.3.1.3. Arcuate Popliteal Ligament

It is fused to the joint capsule. It is a Y-shaped ligament. The ends of the Y attach to the head of the fibula, the lateral epicondyle of the femur, and the posterior intercondylar area (5).

3.3.1.4. Tibial Collateral Ligament

It extends from the medial epicondyle of the femur to the medial condyle of the tibia. Additionally, it is connected to the medial meniscus through the fibrous membrane. It may have one or more bursae between it and the joint capsule. It is crossed by the muscles forming the pes anserine, with a bursa located between these muscles and the ligament (5).

3.3.1.5. Fibular Collateral Ligament

It extends from the posterior aspect of the lateral condyle of the femur to the anterior aspect of the head of the fibula. Distally, it merges with the tendon of the biceps femoris muscle. There is a tendon of the popliteus muscle between it and the joint capsule, so it does not have a direct connection with the capsule and the lateral meniscus. Between the capsule and the ligament, there are also branches of the inferior genicular artery and some sensory fibers (5). Additionally, it is adjacent to the common fibular nerve (4).

3.3.2. İntracapsular Ligaments

3.3.2.1. Anterior Crutiate Ligament

Although it is an intraarticular ligament, it is extracapsular in structure (7). It is named according to its attachment site on the tibia. It originates from the anterior aspect of the intercondylar area of the tibia and attaches to the posterior aspect of the medial surface of the lateral femoral condyle. During its course, it twists around itself. Its attachment to the tibia also extends to the

lateral meniscus. The ACL serves as the primary static stabilizer against anterior translation of the tibia on the femur (8). Additionally, it restricts internal rotation of the tibia in extension (7).

Researchers have examined this ligament in two or three-band configurations. However, the two-band model is generally considered the best representation for understanding the function of the ACL (7). These bands are named according to their attachment sites on the tibia. When examined in two bands, they are referred to as the anteromedial and posterolateral bands, while studies examining them as three bands also mention an intermediate band (7,8). The posterolateral band consists of more fascicles compared to the anteromedial band. In full extension, the anteromedial band is longer than the posterolateral band (7).

During flexion, it undergoes internal rotation relative to the femur, which can reach up to 55 degrees. Therefore, during surgery, the ligament is rotated (3). During this rotation, the posterolateral band remains more fixed while the anteromedial band rotates around it (7). As the anteromedial band lengthens and tightens during flexion, the posteromedial band shortens and loosens. The opposite occurs during extension (3,7). Some researchers have found that the anteromedial band shortens up to 30 degrees of flexion and then begins to stretch, reaching its longest state at 120 degrees of flexion (7).

The cruciate ligaments are composed of a highly organized collagen matrix, constituting approximately three-fourths of their dry weight. While the majority of this collagen is type I collagen, a small portion is type III collagen (8).

Type II collagen is the collagen of cartilage tissue and is not typically found in ligaments. However, it is present in the fibrocartilaginous regions, especially at the tibial and femoral attachment sites, of the ACL. The majority of the total weight of the ACL is composed of water (7).

The innervation of the ligament is provided by the tibial nerve (8). Nerves entering the joint capsule from the posterior aspect follow synovial vessels and extend to the infrapatellar fat pad (9). These nerve fibers primarily serve vasomotor functions and are associated with the endoligamentous vascular system. Additionally, smaller myelinated and unmyelinated nerve fibers can progress independently of the vessels and extend individually between the ligament bundles. Within the ligament, there are nociceptors and mechanoreceptors sensitive to movement and tension. It’s been suggested that these receptors have a proprioceptive effect, influencing muscle tone around the knee and altering knee postüre (7).

The ligament is supplied by the middle genicular artery, a branch of the popliteal artery (3). This artery penetrates the capsule posteriorly to enter the joint space and then divides into branches inside. A small portion of the ligament's distal end is nourished by branches of the inferior genicular artery. The proximal portion has better blood vessels compared to the distal part. The fibrocartilaginous tissue located 5-10 mm proximal to the tibial attachment of the ligament is avascular (7,8).

3.3.2.2. Posterior Crutiate Ligament

The ligament, like the ACL, is an intracapsular ligament and is named according to its attachment on the tibia (5). It originates from the posterior intercondylar area of the tibia and attaches to the outer surface of the medial condyle of the femur. It is stronger and thicker than the ACL (3,5). It restricts excessive flexion of the joint and posterior movement of the tibia on the femur (4,5). It merges with the lateral meniscus below (5). It serves as the primary stabilizer for posterior translation of the tibia at all flexion angles. Additionally, it restricts internal rotation at all flexion angles and external rotation at flexion angles above 90 degrees (10).

Injuries to this ligament are less common in clinical practice compared to the ACL. Similar to the ACL, it is composed of two bands. These bands are named according to their attachment on the femur: anterolateral and posteromedial. During flexion, the anterolateral band is stretched, while during extension, the posteromedial band is stretched. The anterolateral band is thicker. The anterolateral band primarily resists posterior translation at flexion angles between 70-105 degrees, while the posterolateral band provides more resistance at flexion angles between 0-15 degrees. This distribution of forces is significant in surgical double-band repair (10).

The synovial membrane comes from the anterior aspect, covering the anterior and lateral surfaces of the cruciate ligaments before jumping onto the fibrous membrane without extending behind the PCL. Therefore, there is no synovial membrane in the posterior part of the PCL (5).

3.3.2.3. Transverse Ligament

The transverse ligament extends between the anterior horns of both menisci. It facilitates the synchronized movement of the menisci (5). Some authors have stated that the exact function of this ligament is not fully understood (3).

3.4. Menisci

The two fibrocartilaginous structures found within the knee joint are called menisci. Their outer parts are thicker and convex, while their inner parts are thinner and concave. They are positioned on the tibia with their lower surfaces, while their upper surfaces are concave, accommodating the femoral condyles. They attach to the anterior intercondylar area with their anterior ends and to the posterior intercondylar area with their posterior ends. They are fused with the fibrous membrane on the outer side. The ligament connecting the anterior horns of the two menisci is called the transverse genicular ligament (4,5). They help to improve the congruence of the joint surfaces that do not perfectly match, thereby facilitating better joint movement. The inner two-thirds of the menisci are composed of radial collagen fibers, while the outer one-third consists of circular collagen fibers (3).

The menisci are primarily nourished by branches of the popliteal artery. However, in adults, the menisci are largely avascular, and only a small portion is directly supplied by blood vessels. This avascularity significantly affects their healing capacity. Vascularization of the menisci is classified into three zones: the peripheral vascularized red-red zone, the central avascular white-white zone, and the partially vascularized red-white zone in between. It is believed that the white-white and red-white zones obtain more than two-thirds of their nourishment from synovial fluid through diffusion or mechanical pumping (11).

The menisci are composed of fibrocartilage, which is a dense extracellular matrix consisting of a high content of water, collagen, and proteoglycans. The distribution of collagen varies according to the zones of the meniscus. In the red-red zone, predominantly type I collagen is found, whereas in the white-white zone, most of the extracellular matrix consists of type II collagen. The collagen fibers are organized in a three-layered, complex arrangement that enables the distribution of vertical compressive loads (11). In a healthy meniscus, compression forces lead to the absorption of water molecules into the meniscal tissue, which then returns to the joint space during decompression and is redistributed back into the meniscal tissue. This mechanism facilitates the nourishment of fibrochondrocytes (12).

The primary functions of the menisci are to ensure joint congruence, stabilization, load transmission, and shock absorption. Some researchers have also suggested that the meniscus plays a role in proprioception and joint lubrication (11).

3.4.1. Lateral Meniscus

Compared to the medial meniscus, the distance between the anterior and posterior horns of the lateral meniscus is narrower. The anterior horn attaches to the anterior intercondylar area, while the posterior horn attaches to the posterior intercondylar area. The tendon of the popliteus muscle is located on the posterior-lateral side of this meniscus and merges with the medial side of the lateral meniscus (5).

In front and behind the PCL, there are two ligaments extending from the posterior horn of the lateral meniscus to the outer surface of the medial condyle of the femur. The one in front is called anterior meniscofemoral ligament, and the one behind is called posterior meniscofemoral ligament (Wrisberg ligament) (3,4,5). These ligaments function to prevent anterior laxity and should be preserved during surgery (3). These ligaments are not always found together, but at least one of them is present in the majority of individuals (11).

3.4.2. Medial Meniscus

The gap between the anterior and posterior horns is greater in the lateral meniscus compared to the medial meniscus. The attachment site of the anterior horn is located in the anterior intercondylar area, anterior to the ACL. The posterior fibers of the anterior horn extend to the lateral meniscus as the transverse ligament. The posterior horn attaches to the posterior intercondylar area between the PCL and the posterior horn of the lateral meniscus (5). Injuries are more commonly observed in the lateral meniscus. This is due to its peripheral portion being attached to the joint capsule and tibial collateral ligament, making it less mobile (4).

3.5. Bursae Around The Knee Joint

Due to its susceptibility to trauma and the presence of strong and thick tendons passing around it, the knee joint is surrounded by numerous bursae. These bursae can be categorized into three groups: those located anteriorly, laterally, and medially.

The main bursae among these are as follows: Subcutaneous prepatellar bursa, subcutaneous infrapatellar bursa, deep infrapatellar bursa, suprapatellar bursa, subpopliteal recess, pes bursa, and semimembranosus bursa (5).

3.6. The Nutrition of the Knee Joint:

Supplied by descending genicular artery, genicular branch of the popliteal artery, anterior tibial recurrent artery, and descending branch of lateral circumflex femoral artery (5).

3.7. Innervation of the Knee Joint:

Innervated by branches from the obturator, femoral, tibial, and common fibular nerves (5).

Kennedy et al. (9) examined the nerve network innervating the knee joint in two divisions: anterior group and posterior group. The anterior group includes articular branches of the femoralis, saphen, and common peroneal nerve, while the posterior group comprises posterior articular branches from the tibial nerve.

4. Knee Joint Clinical Significance

4.1. Varus Deformity

Maquet line or hip-knee-ankle angle can be used for definition. When using the Maquet line, a line is drawn from the center of the femoral head to the center of the talus. In a normal alignment, this line passes through the center of the hip joint, the center of the knee joint, and the middle of the talus. In the presence of varus deformity, the Maquet line will pass medial to the knee joint. When using the hip-knee-ankle angle, the angle between the line drawn from the center of the hip joint to the center of the knee joint and the line drawn from the center of the knee joint to the center of the ankle joint is measured on full-length radiographs. In a normal alignment, this angle is considered to be 180 degrees, whereas in varus deformity, it will be less than 180 degrees (13). In this deformity, contraction of medial soft tissues and elongation of lateral soft tissues can be observed. Additionally, differences in both soft tissue and bone tissue may be present (14).

4.2. Valgus Deformity

Most patients are asymptomatic and have no functional limitations. Intermalleolar distance has been used to evaluate the degree of genu valgum. To find this distance, the length between the medial malleoli is measured in a standing position where the medial femoral condyles are touching. An intermalleolar distance greater than 8 cm is considered pathological (15).

Some studies have defined a tibiofemoral angle of more than 10 degrees as “valgus deformity” (14,17). The tibiofemoral angle is defined as the angle between the anatomical axis of the femur and the anatomical axis of the tibia (18). In normal adults, there is an average of 6 degrees of valgus (16).

The most common cause of this condition is primary osteoarthritis (16). It can be congenital as well as secondary to osteoarthritis, rheumatic diseases, post-traumatic arthritis, and excessive correction secondary to valgus osteotomy (18).

Treatment may involve distal femoral osteotomies and hemiepiphysiodesis in children under 10 years of age (15). Surgically, soft tissue release in the lateral region can be performed, but inadequate release may result in the development of permanent deformity or excessive release may result in mediolateral instability. However, total knee arthroplasty is also one of the most commonly used methods (16).

4.3. Genu Recurvatum

Genu recurvatum refers to the condition where the hyperextension angle is above 5 degrees (19). The tight iliotibial band can contribute to knee hyperextension. This deformity may occur due to laxity of the cruciate and collateral ligaments in patients with rheumatoid arthritis, high tibial osteotomy, or poliomyelitis (20). It can also manifest as a compensatory mechanism when the quadriceps femoris muscle is insufficient to lock the knee (19).

Especially in cases of increased recurvatum due to increased plantar flexion, the use of hinged ankle-foot orthosis (AFO) can help control recurvatum during walking (21).

4.4. Anterior Cruciate Ligament Injury

The incidence of ACL injuries is unknown, but it is estimated that 350,000 ACL reconstructions are performed annually in the United States. Despite surgical repair, approximately 79% of these individuals develop knee osteoarthritis, and 20% experience re-injury within 2 years (22). Most ACL injuries occur with non-contact or minimal contact. They typically occur during landing or deceleration maneuvers in team sports (23). Patients often report hearing a “pop” sound at the time of acute trauma, followed by effusion (24). When compared between male and female athletes in the same sports, the incidence of ACL injuries is reported to be 2-8 times higher in females. This is attributed to factors such as joint laxity, valgus moment, and recurvatum (25).

There are numerous modifiable and non-modifiable risk factors for injury. Any injury to passive stabilizers of the knee can affect joint biomechanics and increase the risk of injury. Additionally, small intercondylar notch dimensions are considered a risk factor for ACL injury (22). Factors such as thinness or excessive length of the ligament also contribute to injury (25).

There are many dynamic and modifiable risk factors as well. Hyperextension of the knee, inadequate hip abductor muscle strength, and increased valgus are among these factors. Internal rotation of the tibia near full extension of the knee is one of the mechanisms that puts the most strain on the ligament. Falling from

a jumping position with valgus stress also increases the load on the ACL and can result in injury (22). Rotation of the trunk with valgus at the knee joint while the foot is fixed on the ground is another mechanism that can cause ligament injury (24).

The simultaneous contraction of the hamstring muscles with the quadriceps femoris muscle during activities such as jumping and changing direction helps to control the anterior translation of the tibia. Weakness in the hamstring muscles, delays in reaction time, and neuromuscular control deficiencies can also be risk factors for injury (25).

There are several tests to assess ligament integrity, such as the Lachman test, pivot-shift test, and anterior drawer test. These tests may often yield negative results in acute injuries due to muscle contraction, so examination under anesthesia is recommended (24).

The ACL is a strong ligament that tears when subjected to loads requiring more than 5% elongation of its length. In physically active individuals, athletes, and those with combined ligament injuries, surgical reconstruction is recommended. Various surgical techniques are available, with hamstring autografts being the most commonly used (26). Allografts and bone-patellar tendon-bone grafts are also used (27).

Although older studies argued that complete ACL ruptures do not heal spontaneously, recent studies have found evidence that the ACL can heal. In a study by Filbay et al. (28), they followed patients with ACL rupture regularly for 2 and 5 years. Spontaneous healing was observed in 53% of 30 patients who were followed up with rehabilitation alone, and in 16 out of 54 patients followed up for elective delayed surgery in the second year.

There are many mechanoreceptors within the ACL known to play a proprioceptive role and influence surrounding muscle tone. It is known that the loss of afferent feedback from these mechanoreceptors in the ACL leads to weakness in the quadriceps femoris, which becomes more pronounced in patients with a ruptured ACL. Indeed, this afferent feedback from the ACL has a significant impact on the maximum voluntary contraction of the quadriceps femoris. Therefore, preserving remnants of the ACL during ACL reconstruction can help preserve proprioception after reconstruction (7).

4.5. Posterior Cruciate Ligament Injury

Injuries to the PCL typically occur as a result of a force applied from anterior to posterior on the proximal tibia, especially when the knee is in

flexion. However, they can also occur during falls onto a flexed knee while the foot is in plantar flexion, hyperflexion, or hyperextension. The posterior drawer test is utilized in clinical settings to assess injuries to this ligament, with a sensitivity of 90% and specificity of 99%. Additionally, the Godfrey test and quadriceps active contraction test are among the tests used to evaluate ligament integrity (10).

The management of PCL tears is a subject of considerable debate. However, most authors agree on conservative treatment for isolated PCL ruptures. Surgical reconstruction is recommended for combined and symptomatic injuries; however, studies have not demonstrated that surgery prevents the development of osteoarthritis (10).

4.6. Osteoarthritis

Osteoarthritis is defined as a heterogeneous group of pathologies characterized by cartilage breakdown and damage to adjacent bones, resulting in symptoms, clinical, and radiological findings in the joint, as defined by the American College of Rheumatology (ACR). While the exact cause is not clear, two theories are proposed. In the first theory, the joint cartilage is unhealthy, and even normal loads lead to degeneration due to cartilage material defects. In the second theory, there is no unhealthy cartilage initially; however, traumas and excessive loads affect the cartilage structure, damage the collagen network, and enzymes released due to chondrocyte injury cause damage. Although primarily affecting joint cartilage and subchondral bone, all joint components are affected by this pathology. Risk factors include increasing age, female gender, obesity, genetic factors, occupation, sports activities, and trauma. The most commonly affected area in the knee joint is the medial tibiofemoral joint. Common findings include joint pain, stiffness lasting less than 15-30 minutes, loss of joint range of motion, osteophytes, crepitus, and quadriceps femoris muscle atrophy. Pharmaceutical methods can be used for pain control. Physical therapy applications are recommended for regaining joint range of motion, restoring muscle strength, and pain control (29).

Ablation of nerve branches carrying pain sensation from the knee is also a method used for pain management. Blocking the popliteal plexus formed by the obturator and tibial nerve branches in the popliteal fossa (without causing any motor weakness) can provide significant analgesic effects after major knee surgery (30,31). Total knee arthroplasty is also a commonly used method for patients with advanced cartilage damage who do not respond to conservative treatment (32).

4.7. Hoffa's Fat Pad Syndrome

First described in 1904 by Albert Hoffa, Hoffa's fat pad syndrome is defined as the inflammatory hypertrophy of the infrapatellar fat pad, causing compression and pain (33). The emerging symptoms are often mistaken for other pathologies and are detected less frequently than they actually occur. The widespread use of magnetic resonance imaging (MRI) techniques facilitates its detection. The etiopathogenesis is not clear but inflammation, acute trauma, and repeated microtrauma may be causative factors. The inflamed fat pad can be compressed in the patellofemoral and tibiofemoral joints, leading to anterior knee pain (34). Pain is felt in the retropatellar and infrapatellar regions along with loading and knee movements. In acute cases, the fat pad is hypertrophic and tender (33).

Clinical examination and radiological imaging are used for diagnosis, and observation compared to the opposite knee is important. Typically, there is effusion and limited movement in the knee. The "Hoffa Test" can be used during examination (34). MRI may visualize calcification, edema, or fibrosis in the corpus adiposum infrapatellare (33).

Treatment may include nonsteroidal anti-inflammatory drugs, corticosteroid injections, or physiotherapy, but these may not be sufficient. Surgical partial resection is performed in cases where conservative treatment is not effective (33).

4.8. Medial Plica Syndrome

Plicae are folds of synovial tissue that are elastic structures and change shape and size with joint movement. The most symptomatic plica is the medial plica. Plicae can become symptomatic due to a structural difference in their composition. It is accepted that their structural characteristics change following an inflammatory process. Therefore, conditions causing synovitis can lead to plica syndrome. It can occur idiopathically or following trauma. Following trauma, there is often a silent period (weeks/months) before symptoms appear. The most common symptom is medial knee pain that worsens with activity. Due to the lack of specific findings, it is often confused with other pathologies. However, a thickened and painful plica can be palpated in the medial aspect of the joint. Clinical tests as well as imaging methods are used for diagnosis. Among these methods, MRI and ultrasound are prominent, with arthroscopy being the gold standard for diagnosis (35).

Treatment options include physiotherapy, corticosteroid injections, and plica resection (35).

4.9. Baker's Cyst

Baker's cyst is a synovial cyst formed by the enlargement of the bursa between the gastrocnemius and semimembranosus tendons, located in the popliteal fossa. It is characterized by swelling, pain, and tightness in the posterior aspect of the knee joint, which may limit joint range of motion. Small cysts may be asymptomatic. The cyst becomes more prominent in extension due to compression by the gastrocnemius and semimembranosus muscles. Conversely, it may not be observable in flexion, a sign known as the "Fouchier sign". The cyst may compress the popliteal artery and vein, tibial nerve, or common peroneal nerve, leading to symptoms associated with these structures. Additionally, the cyst may rupture, causing leg pain and swelling. Treatment often involves aspiration and excision of the cyst (36).

4.10. Discoid Meniscus

A discoid meniscus is a condition where the meniscus, which should normally have a crescent shape, appears disk-shaped. It is quite rare in the medial meniscus and presents symptoms similar to those of meniscal injuries. It is more commonly seen in the lateral meniscus. Pain, tenderness along the joint line, a sensation of popping, giving way, and locking are among the most common symptoms. There is a consensus not to intervene in discoid menisci that do not manifest symptoms. Treatment typically involves total or partial meniscectomy. Since patients who undergo total meniscectomy have a higher risk of developing osteoarthritis, partial meniscectomy is often preferred (37).

4.11. Osteochondritis Dissecans

Osteochondritis dissecans is defined as the detachment of an osteochondral fragment from its bony bed, with the knee joint being the most commonly affected joint. The detached portion maintains its vitality as the cartilaginous tissue continues to be nourished by synovial fluid, while the subchondral bone portion becomes necrotic. When the fragment becomes detached, locking or catching sensations may occur. The most common symptoms include anterior knee pain, pain-related limping, and intermittent swelling (38).

Diagnosis is typically made through direct radiography. If the patient's epiphyses are not closed and the fragment remains in place, conservative treatment may be preferred. Conservative treatment involves the use of pharmaceutical agents for pain control and activity restriction. If there is no response to conservative treatment or in cases of instability, surgical intervention is preferred (38).

4.12. Osgood-Schlatter Disease

Osgood-Schlatter disease (OSD) was first described by Osgood and Schlatter in 1903. It arises from the formation of a traction apophysis due to repetitive contractions of the rectus femoris muscle. Symptoms typically occur in girls aged 8 to 12 and boys aged 12 to 15. However, cases have also been reported in adults. Treatment is generally conservative (39).

Several treatment strategies have been suggested, including modification of physical activity, cold therapy, the use of knee braces that reduce tractional load on the insertion of the patellar tendon, physical therapy, and warm-up and cool-down exercises before and after training/competition. It is recommended to reduce or avoid running, jumping, and directional changes until symptoms improve (39).

Surgery is recommended when conservative treatment fails and bone fragments are observed after ossification is complete (39).

4.13. Meniscal Injury

Meniscal tears lead to an increase in tibiofemoral contact pressure and contact area, which has been shown to result in altered biomechanics and accelerated degenerative changes in the knee joint. The treatment approach for meniscal injuries now primarily focuses on anatomical restoration, as conservative treatment and meniscectomy are associated with worse clinical outcomes and a higher rate of total knee arthroplasty (40)

5. Conclusion

When considering a healthy knee joint as a whole, it is a complex mechanism that functions in perfect harmony. Managing injuries becomes challenging when considering its size, the function of joint components, and their relationship with each other. It is essential for every clinician to have a detailed understanding of the anatomy of each joint element for accurate diagnosis, treatment, and rehabilitation.

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CHAPTER VIII

CLINICAL ANATOMY OF ENTRAPMENT NEUROPATHIES

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1. Introduction

Entrapment neuropathies are the most common cause of the clinical picture characterized by compression of peripheral nerves as they pass through narrow anatomical spaces. Nerve entrapment develops as a result of reduced mobility and flexibility of peripheral nerves due to compression by surrounding tissues. The compression of nerves in tunnels is sometimes accompanied by muscular and vascular structures. Acute or chronic neuropathic pain, numbness, sensory changes, loss of function and trophic disorders are observed. (1)

The most common entrapment neuropathy is “carpal tunnel syndrome” (CTS), which affects 10% of the population. After CTS, the next most common syndrome is cubital tunnel syndrome. Compression of the Sciatic nerve also shows a prevalence between 1.6% and 43%. Despite their high prevalence, they are often difficult to diagnose and treat in the clinical setting. (2,3)

Although the etiology is not fully known, recent studies suggest that genetic predisposition may be the strongest risk factor. However, systemic problems such as body mass index, occupational and physical factors, diabetes and thyroid disorders predispose to entrapment neuropathies. (3)

In addition to patient complaints, provocative tests such as “Tinel’s sign”, “Phalen’s test”, morphological measurements such as body mass index, circumference measurements, neurodynamic tests such as straight leg raising (SLR), electrodiagnostic tests and radiological imaging tools such as ultrasound,

computed tomography (CT), magnetic resonance imaging (MRI) are used to make a clinical diagnosis. (3)

There is a wide range of treatments. Conservative treatments, medical drugs and injections and surgical methods are preferred depending on the course of the disease. (3)

2. Trunk and Lower Extremity Entrapment Neuropathies

Nerve compression in the trunk and lower extremities occupies a large part of the clinical picture. They are difficult to diagnose because they present similar complaints with many other problems. At the same time, this situation often leads to the inability to apply the correct treatment due to the inability to make the correct diagnosis. This is a waste of time for the patient and in some cases may even lead to unnecessary surgical procedures.

Low back pain is seen in almost all trunk and many lower extremity entrapment neuropathies. Spinal stenosis, disc herniations, degenerative joint problems, infection and inflammatory conditions present similarly. Although the clinical symptoms are similar, the treatment methods differ. For these reasons, the anatomical structure and clinical presentation of these regions should be well researched and known. (4)

3. Entrapment Neuropathies of the Trunk

3.1. Notaljia Paresthetica

Notalgia paresthetica is an idiopathic chronic cutaneous benign dysesthesia characterized by pruritus localized on the inferior-medial border of the scapula. Although it is more common in older women, the incidence is 2-3 times higher in women than in men. Geography and ethnicity have no effect on the incidence. Although it is usually unilateral, it may be bilateral and persists for a very long time. It shows a course characterized by periods of remission and exacerbation. There is no primary cutaneous lesion, secondary changes are seen due to chronic scratching. (5)

3.1.1. Anatomy

The dorsal branches of the posterior intercostal nerves in the thoracic region are divided into medial muscular branches and lateral muscular branches. Above the level of T6, the cutaneous branches innervating the skin originate from medial muscular branches and are named medial cutaneous branches. These branches pass between the semispinalis muscle and multifidus muscle

and pierce the rhomboideus muscles and trapezius muscle. These branches, which run at right angles along the multifdus muscle, receive the sensation of the dorsal skin above T6. Lateral muscular branches has only somatomotor branches above T6, while between T5-T12 it has additional branches innervating the skin. These branches, called posterior cutaneous branches, extend along the serratus posterior inferior muscle and its aponeurosis and pierce the latissimus dorsi muscle providing sensory innervation of the skin of the posterior lower back. Somatomotor branches run between latissimus dorsi and iliocostalis muscles. (6,7)

3.1.2. Etiology

Besides physical causes such as muscle spasms, anatomical variations in the course of cutaneous branches predispose to nerve compression. Genetic factors and metabolic diseases such as diabetes are among the etiologies. (5)

3.1.3. Clinical Symptoms and Imaging

In notalgia paresthetica, there is a long-standing, intermittent pruritus of variable intensity located contralateral to the dominant side, medial to the lower two-thirds of the scapula. Patients may complain of pain, warmth, coldness, foreign body, tingling and numbness. There is no loss of function, but pain may limit shoulder and trunk movements. Notalgia paresthetica causes thickening of the skin and areas of darkened pigmentation after inflammation. X-rays, CT and MRI are not necessary for diagnosis. (5)

3.1.4. Treatment

Oral and topical medications as well as transcutaneous electrical stimulation to control pain are the main components of treatment. A strengthening program applied to the paraspinal muscles to prevent muscle atrophy and stretching exercises to reduce itching by changing the course of cutaneous branches are included in the treatment protocol. (5)

3.2. Superior Cluneal Nerve Syndrome

Superior cluneal nerve syndrome is a neuropathy characterized by the compression of the medial branch of the superior cluneal nerves in a tunnel between the thoracolumbar fascia and the superior iliac crest as it passes over the iliac crest, resulting in a pain pattern localized in the crista iliac and radiating to the buttocks. The pain is always unilateral and can mask many gastrointestinal and urinary tract problems. The incidence is high in patients with chronic low

back pain and half of those with nerve compression also have symptoms in the legs. (8,9)

3.2.1. Anatomy

The dorsal branches of the lumbar spinal nerves at the L1-L3 level are divided into medial and lateral branches. Frequently, the lateral branches of these posterior branches is called the superior cluneal nerves and pierces the aponeurosis of the latissimus dorsi muscles and the outer edge of the sacrospinal muscles and extends to the upper part of the buttocks in the skin of the gluteal region. Since it is composed of posterior branches, it does not join the lumbar plexus. During its course, it passes through the psoas major and paraspinal muscles and goes behind the quadratus lumborum muscle. It provides sensory innervation of the area above the gluteus maximus muscle and gluteus medius muscle. (6,7)

3.2.2. Etiology

Diseases and deformities that cause thickening of the thoracolumbar fascia predispose to entrapment. Injections and surgical procedures that may cause nerve irritation may also cause entrapment. (8,9)

3.2.3. Clinical Symptoms and Imaging

The pain almost completely disappears after lidocaine injection. On segmental examination, tenderness is often present at the T12-L1 level and sometimes at the L1-L2 level. In the prone position, there is a trigger point on the posterior iliac crest 7 cm from the midline. Since superior cluneal nerves is a thin nerve, CT and MRI cannot help in terms of diagnosis. (8,9)

3.2.4. Treatment

Thoracolumbar exercise protocols, analgesic or anti-inflammatory drugs and targeted spinal manipulations are the first line of treatment. In the absence of an effective response or when manipulation is contraindicated, corticosteroid injections, neural blockades or surgical procedures are used. (8,9)

3.3. Abdominal Cutaneous Nerve Entrapment Syndrome

Abdominal cutaneous nerve entrapment syndrome is a syndrome characterized by chronic pain after entrapment of the anterior cutaneous terminal branches of the last six intercostal nerves (T7-12) at the lateral edge of the rectus abdominis. Although it is one of the most common causes of abdominal wall

pain, abdominal pain is often attributed to other etiologies. While psychological factors are suggested to be the cause of persistent pain after abdominal surgeries, the possibility of entrapment is often overlooked, leading to a much lower incidence of this syndrome than it should be. Abdominal cutaneous nerve entrapment syndrome is diagnosed in 2% of patients with acute abdominal pain. It is four times more common in women than in men. The incidence peaks in the young and middle-aged population. (10)

3.3.1. Anatomy

The intercostal nerves are located in the lower part of the neurovascular bundle running along the costae. The first six intercostal nerves reach the sternum, while the lower six intercostal nerves pierce the diaphragm, run between the abdominal oblique muscle and transverse abdominal muscle, and reach the linea alba after passing through the rectus sheath. These nerves are composed of motor and sensory fibers that innervate the skin as well as motor stimulation of the abdominal muscles. The anterior abdominal cutaneous branch, terminal branches of the six inferior intercostal nerves, travel between abdominal internal oblique and transverse abdominal muscles, reaching the inner side of the trunk and then crossing the outer layer of the sheath. Under the skin, it divides into medial and lateral branches. The anterior abdominal cutaneous branch provides sensory innervation of the anterior abdominal wall from the level of the xiphosternal line. (6,7)

3.3.2. Etiology

Excessive forward or backward movement of the neurovascular bundle causes compression of the nerve against the fibrous ring to which the bundle is attached. In conditions such as pregnancy, when the volume of the abdominal cavity increases, the distance between the two ends of the nerve increases and can cause it to stretch. Heavy physical activity and hyperactivity of the muscle can cause stretching or compression of the nerve at its insertion into the muscle sheath. Laparoscopic surgeries can put pressure on the abdominal wall and trigger pain. Infectious diseases can cause neural edema and increase pain. (10)

3.3.3. Clinical Symptoms and Imaging

There is acute burning pain in the rectus abdominis muscle region. This pain can be unilateral or bilateral. Changing position, coughing and movements that stretch the muscle, such as the Valsalva maneuver, increase the pain. Intra-abdominal pressure can cause the abdominal wall to protrude in the painful

area. The most painful pressure point is 3 cm lateral to the umblicus. Lying still is usually the most relaxing position. Autonomic nervous system disorders may accompany the syndrome. Sweating, dizziness, loss of appetite, nausea and vomiting are seen in many patients. These visceral symptoms prolong the diagnostic process and expose the patient to incorrect treatment. Imaging modalities are used to exclude other diagnoses causing abdominal pain. (10)

3.3.4. Treatment

Lowering the intra-abdominal pressure or removing the cause usually gives good results. Local anesthesia of the painful area reduces or stops the pain. If the cause cannot be treated, reduced activity is recommended. Corticosteroids and injections are used if there are no positive results. Surgical intervention is rarely indicated. (10)

3.4. Iliohypogastricus Syndrome

It is a syndrome characterized by motor and sensory disturbances in which the iliohypogastric nerves is compressed as it travels along its course along the posterior abdominal wall where it pierces the aponeurosis of the transverse abdominal muscle and abdominal internal obliquemuscles and then travels between the abdominal internal obliquemuscle and abdominal external oblique muscle. (4)

3.4.1. Anatomy

The iliohypogastric nerve, which consists of the anterior branches of the T12 and L1 nerve roots, arises at the upper part of the lateral margin of the psoas major muscle and travels downward along the anterior abdominal wall, crossing the quadratus lumborum muscle until it reaches the iliac crest. In this region, it divides into two branches, the lateral cutaneous branch and anterior cutaneous branch. The lateral cutaneous branches pierces the abdominal internal oblique muscle and abdominal external oblique muscle above the iliac crest before sensing the gluteal region. The anterior cutaneous branch runs medial to the annulus inguinalis subcutaneous , and innervates the skin over the inguinal ligament and symphysis pubis. (6,7)

3.4.2. Etiology

Scars along the line of the nerve, bleeding, tumors, surgeries in this area, trauma or natural processes such as pregnancy are the most likely causes of compression. (4)

3.4.3. Clinical Symptoms and Imaging

Compression of the nerve proximally causes same-sided muscle weakness that increases when the patient stands. The patient may have paresthesias both around the hip and over the inguinal ligament. Or only sensory changes occur near the inguinal ligament. If the nerve is compressed near the iliac crest, only the lateral cutaneous branches dermatome is affected. These sensory changes usually present as burning, pain or pins and needles that increase with hip extension. Electromyographic tests (EMG), MRI and nerve blockades with imaging help to make the diagnosis. (4)

3.4.4. Treatment

Treatment involves eliminating the causative factors if possible. Conservatively, abdominal muscle strengthening, gait correction and postural modifications, transcutaneous stimulation can modify the patient's symptoms and reduce nerve irritation. Nonsteroidal anti-inflammatory drugs (NSAİD), opioids, antiepileptics and antidepressants can reduce symptoms. A nerve block may aim to interrupt neuronal transmission leading to temporary relief of pain. Surgery is used for persistent, intractable pain after conservative measures have failed. (4)

3.5. Ilioinguinalis Syndrome

It is a syndrome characterized by both motor and sensory dysfunction due to compression of the ilioinguinal nerve as it travels through the abdominal wall between the transverse abdominal muscle and the abdominal internal oblique and abdominal external oblique. (11)

3.5.1. Anatomy

The ilioinguinal nerve arises from the anterior branches of the spinal nerves from L1 and passes behind the kidney and descends along the quadratus lumborum muscle and iliacus muscles. It pierces the transverse abdominal and abdominal internal oblique and passes under the abdominal external oblique muscles. While traveling between the oblique muscles distal to the spina iliaca anterior superior (SIAS), it changes direction and passes through the inguinal canal, accompanying the spermatic cord in men and the round ligament of uterus in women. Sensory fibers branch and distribute to the skin of the scrotum as anterior scrotal nerves in men and to the mons pubis and labium majus in women as anterior labial nerves, providing sensory innervation of this area. (6,7)

3.5.2. Etiology

Since ilioinguinal nerve passes through the retroperitoneal space anterior to the abdominal wall, the nerve is at risk of compression from muscular, retroperitoneal, renal and urogenital pathologies. Inguinal hernias and subsequent surgeries, abdominal surgeries, pathologies involving the spermatic cord, trauma and pregnancy are the main etiologies of nerve entrapment. (11)

3.5.3. Clinical Symptoms and Imaging

Burning pain in the scrotal and groin area radiating to the buttocks is the first complaint. This pain may be provoked by activities that stretch the anterior abdominal wall, such as standing more upright. A compression medial and distal to the SIAS reintroduces the pain along the inguinal ligament. Antalgic gait develops due to the syndrome. Sensory changes are seen in the dermatome area. MRI and ultrasound examination may be helpful for diagnosis. (11)

3.5.4. Treatment

Physical activities that relieve pain, daily life modifications, non-steroidal anti-inflammatory drugs, transcuteneal electrical stimulations constitute the first step of treatment. If conservative treatment is not successful, surgical treatment can be applied. Surgical treatments are very effective in eliminating symptoms. (11)

3.6. Genitofemoralis Syndrome

Compression of the genitofemoral nerve where it passes through the psoas muscles or where the femoral and genital branches pass through the abdominal wall. Compression or traction at these sites causes sensory changes in the skin of the medial thigh, scrotum/labium majus and along the abdominal wall below the inguinal ligament. Changing from standing to bending position relieves the pain, while walking or Valsalva maneuvers worsen the condition. (12)

3.6.1. Anatomy

The genitofemoral nerve consists of L1 and predominantly L2 nerve roots. The nerve passes through the psoas major muscle before branching into femoral and genital. The lateral branch of the genitofemoral nerve is the femoral branch, which lies lateral to the femoral nerve. It crosses the deep circumflex iliac artery and passes through the fascia iliaca. It passes through the lacuna vasorum with the external iliac artery and reaches the subinguinal region before branching to anastomose with the genital branch and lateral femoral cutaneous nerve. It

receives sensation from the skin of the upper part of the anterior aspect of the thigh.

The medial branch of the genitofemoral nerve is the genital branch along the psoas major muscle. It runs in front of the common iliac artery and external iliac artery and behind the ureter, spermatic artery and spermatic vein. It enters the deep inguinal ring, passes through the inguinal canal and exits through the superficial inguinal ring, giving fibers to the cremaster muscle and scrotum skin in men. In women, it travels with the round ligament of uterus in the inguinal canal and receives sensation from the skin of the mons pubis and labium majus pudendi. (6,7)

3.6.2. Etiology

Entrapment can be caused by scars and adhesions after hernia surgeries, appendectomy or gynecologic surgery, psoas abscesses, blunt abdominal trauma, tight clothing or cycling. (12)

3.6.3. Clinical Symptoms and Imaging

Patients complain of intermittent pain in the groin area and medial thigh. The pain does not go below the knee. Hypoesthesia is seen in the dermatome area. Absence of cremaster reflex, increased pain with hip extension and rotation and radiation of pain with compression are signs of entrapment. Tinel's sign is positive and there may be weakness in the anterior abdominal wall. CT-guided diagnostic and therapeutic interventions can be performed to block the genitofemoral nerve. (12)

3.6.4. Treatment

Conservative treatment options are limited. It is not always possible to eliminate the causes of the tightness or tension. The combined use of anesthetic drugs and corticosteroids can provide treatment of sensory disturbances. Botulinum toxin is an effective method for controlling pain. In cases that do not respond to conservative treatment, surgery is an option to reduce pain and other complaints. (12,13)

3.7. Alcock Canal Syndrome

Alcock canal syndrome is an impingement syndrome characterized by chronic neuropathic pelvic pain caused by compression of the pudendal nerve as it passes through the infrapiriform foramen, lesser sciatic foramen or Alcock canal area. Alcock canal syndrome is often misdiagnosed and therefore not

treated correctly. The most characteristic symptom found in more than 50% of patients is groin pain that increases with sitting and is relieved by standing or lying down. Alcock canal syndrome can affect up to 1% of the general population and is twice as common in women as in men. (14,15)

3.7.1. Anatomy

The pudendal nerve, one of the longest nerves of the sacral plexus, is a mixed nerve originating predominantly from the ventral branches of the S2-S4 spinal nerves. The plexus is located in front of the coccygeus muscle and at the lower edge of the piriformis muscle. The pudendal nerve leaves the pelvis via the infrapiriform foramen, the distal part of the greater sciatic foramen, makes a loop around the spina ischium behind the pudendal artery internale and re-enters the pelvis with the artery via the lesser sciatic foramen, reaching the lateral wall of the ischioanal fossa. It then extends in the pudendal canal to the perineal region and external genitalia. The pudendal nerve first gives off the inferior anal nerves, and as it approaches the diaphragm urogenitale it divides into two terminal branches, the perineal nerves and the dorsal nerve of the penis. The pudendal canal is located in the obturator fascia, which covers the inner surface of the obturator internus muscle on the outer wall of the ischioanal fossa. (6,7)

3.7.2. Etiology

Primary or metastatic tumors of the urogenital system, cauda equina tumors and trauma to the pelvic region may cause nerve compression. Sports such as cycling and horseback riding, which increase the pressure in the pelvic region, also predispose to pudendal nerve compression. Pregnancy, difficult delivery and strength training are also factors that can cause pudendal nerve compression. (14,15)

3.7.3. Clinical Symptoms and Imaging

Sensory disturbances, neuralgias or chronic pain may be present. Symptoms are exacerbated by factors that increase intrapelvic pressure, such as defecation or sexual intercourse. The discomfort is worse in the morning on waking, intensifies in a sitting position and decreases with upright posture and walking. The pain worsens hours after sexual intercourse. It may result in persistent burning or paresthesias radiating to the pelvic floor. Motor dysfunctions may occur after prolonged compression. In many cases thought to be chronic prostatitis or interstitial cystitis, the real problem is compression of the pudendal nerve. Radiological examinations are often used to detect causes

of compression such as tumors. Diagnostic nerve blocks are performed with ultrasound and CT guidance. (14,15)

3.7.4. Treatment

Trying to eliminate the causative factors, pelvic floor physiotherapy, medication and nerve blockades are the treatment methods used in the first stage. Cryotherapy and sacral neuromodulation are interventional procedures for pain. In persistent pain that does not go away, surgical methods aimed at removing the tissues compressing the nerve are used. (14,15)

4. Entrapment Neuropathies of the Lower Extremities

4.1. Lumbosacral Tunnel Syndrome

Lumbosacral tunnel syndrome is a syndrome characterized by hypoesthesia and pain following compression of the L5 nerve root under the l lumbosacral ligament after it leaves the intervertebral foramen and crosses the ala of sacrum. (16,17)

4.1.1. Anatomy

The lumbosacral ligament starts from the fifth lumbar vertebra and attaches to the upper border and anterior surface of the ala of sacrum. The fifth lumbar spinal nerve leaves the intervertebral foramen and travels inferolaterally, crossing the upper border of the sacrum before reaching the anterior surface of the sacrum. On this route the nerve runs under the lumbosacral ligament. The sympathetic nerve branches pierce the lumbosacral ligament and merge with the nerve. The branch of the fourth lumbar nerve root passes through the anterior surface of the lumbosacral ligament, merges with the fifth nerve root and exits the lower border of the ligament to form the lumbosacral trunk. The lumbosacral tunnel can be seen as a continuation of the L5-S1 intervertebral foramen, through which the L5 spinal nerve and the sympathetic communican branch and branches of the iliolumbal artery and veins pass. (6,7)

4.1.2. Etiology

Factors such as thickening of the lumbosacral ligament, psoas hematoma, aneurysms, vascular problems and local inflammatory processes that cause changes in the volume of the tunnel are factors that cause the syndrome. Similarly, causes of lumbosacral instability such as tumors and pelvic fractures can also cause nerve compression. (16,17)

4.1.3. Clinical Symptoms and Imaging

It presents with sensory disturbances, pain and weakness in the L5 nerve dermatome. Bladder and bowel incontinence may be seen, similar to cauda equina syndrome. EMG studies and MRI can be used to make the diagnosis and exclude other pathologies. (16,17)

4.1.4. Treatment

Conservative treatments are applied to eliminate the causative factors. In the absence of positive responses, surgical loosening operations are performed. (16,17)

4.2. Gluteal Nerve Syndrome

It is a condition in which the superior gluteal nerve and inferior gluteal nerve are compressed while passing through the infrapiriform foramen and suprapiriform foramen, resulting in weakness in the muscles they innervate. Although it can be bilateral, unilateral impingement is more common. Sitting for a long time causes pain and worsens the existing pain. Night pains are common. (18)

4.2.1. Anatomy

The superior gluteal nerve originates from the lumbosacral trunk, which is formed by the L4 and L5 roots that merge with the sacral plexus. This root is proximal and dorsal to the beginning of the inferior gluteal nerve. The nerve leaves the pelvic region through the suprapiriform foramen with the superior gluteal artery. The borders of this foramen are the upper edge of the piriformis muscle, the lower edge of the gluteus medius muscle and the greater sciatic notch. The piriformis muscle bisects the greater sciatic notch, forming a superior and an inferior foramen. As the nerve passes through the suprapiriform foramen is, it branches to innervate the gluteus medius muscle and gluteus minimus muscle and inferior branch innervates the tensor fasciae latae muscle. (6,7)

The inferior gluteal nerve originates from the lumbosacral trunk but also receives innervation from the S1 and S2 roots. The nerve enters the infrapiriform foramen with the sciatic nerve, which runs laterally and inferiorly. After exiting the infrapiriform foramen, it branches to innervate the gluteus maximus muscle and the hip capsule. (6,7)

4.2.2. Etiology

Isolated compression of these nerves is very rare. Nerve irritation may occur following pelvic fractures, hip surgery and intramuscular injections. (18)

4.2.3. Clinical Symptoms and Imaging

Injury to the superior gluteal nerve causes the pelvis to tilt to the opposite side during walking or standing on the affected leg, producing the Trendelenburg sign. İnferior gluteal nerve compression causes difficulty in activities requiring hip extension such as climbing stairs, standing up from a sitting position. CT and ultrasound-guided nerve blocks are helpful for diagnosis. MRI is a sensitive method to confirm the diagnosis. (18)

4.2.4. Treatment

Physical therapy and electrotherapy applications aiming to strengthen muscles and nerve regeneration constitute the first step of treatment. Nerve injuries due to serious causes such as trauma and pelvic fractures may require surgery. (18)

4.3. Iliacus Syndrome

The femoral nerve and iliopsoas muscle pass through the lateral cavity of the lacuna muscularis, whose walls are formed by the ilium bone, iliopsoas muscle, iliopectineal arch and inguinal ligament. Iliacus syndrome is a retroperitoneal compartment neuropathy with both sensory and motor deficits caused by compression of the femoral nerve in this tunnel. The incidence is estimated to range from 1.3 to 6.6% in patients on anticoagulants and from 5.5 to 10.4% in patients with hemophilia. Although it is rare, it can cause serious consequences if left untreated. (4)

4.3.1. Anatomy

The femoral nerve originates from the L2, L3 and L4 spinal nerves. Passing between the iliacus muscle and psoas muscles, the nerve branches immediately after passing through the muscular lacuna under the inguinal ligament. One of the superficial terminal parts of the femoral nerve, muscular branch, provides motor innervation to the pectineus and sartorius muscles. Anterior cutaneous branches provides sensation to the anterior thigh. The deep branch innervates the quadriceps femoris muscles and forms the saphenous nerve, which provides sensation to the medial thigh, leg and foot. All anterior thigh muscles except tensor fasciae latae muscle are innervated by the femoral nerve. (6,7)

4.3.2. Etiology

Surgical procedures in the pelvis are the most common cause of femoral nerve compression. Hematomas, vascular problems, tumors and trauma are other possible causes of nerve compression. (4)

4.3.3. Clinical Symptoms and Imaging

Patients with iliacus syndrome have difficulty in extending the knee. Hip extension aggravates the pain, while the hip is painless in other movements. The patellar reflex disappears. Sensory disturbances are seen in the femoral nerve dermatome. MRI is a reliable tool to detect hemorrhage due to injury of the M.iliacus. It is also a good option to differentiate it from other possible diagnoses. Ultrasound is used to diagnose and especially to treat nerve blocks. (4)

4.3.4. Treatment

Electrotherapy and exercise methods to prevent atrophy of the quadriceps femoris muscle are the primary treatment option due to the lack of risk. Symptoms can be alleviated with nerve blocks. Surgery is inevitable in cases that do not respond to conservative treatment. (4)

4.4. Obturator Tunnel Syndrome

It is an entrapment neuropathy due to compression of the obturator nerve as it passes through the obturator canal. It is often confused with pathologies such as disc herniations with similar findings. It is important to differentiate it from diseases that cause groin pain such as tendonitis, bursitis, osteitis pubis or stress fractures. Radiologic imaging and nerve conduction velocity tests are reliable methods for diagnosis. (19,20)

4.4.1. Anatomy

Originating from the second, third and fourth lumbar nerve roots of the plexus lumbalis, the obturator nervetravels under the psoas major and enters the obturator canal together with the iliac internal artery and vein. As it exits the canal, it divides into anterior and posterior branches. These branches include a part of the adductor brevis and obturator externus muscles. Anterior branches provides motor innervation to pectineus, adductor longus, adductor brevis and gracilis muscles and sensory fibers to the hip joint. Posterior branches innervates adductor magnus and adductor brevis muscles when it is not innervated by anterior branches. It also provides sensation to the knee joint and the medial side of the knee. (6,7)

4.4.2. Etiology

Although the obturator canal is well protected by surrounding structures, natural processes such as pelvic fractures, hematomas, tumoral processes or

pregnancy can cause neuropathy. Obturator nerve is at risk due to its course and is the least tolerant to compression among the entrapment neuropathies. (19,20)

4.4.3. Clinical Symptoms and Imaging

Obturator tunnel syndrome causes severe rest pain radiating from the symphysis pubis to the knee. Hip extension and abduction cause an increase in pain. Adductor paresis may develop if compression is prolonged. Patients have a broad-based gait pattern. There is sensory loss in the middle and lower 1/3 of the thigh, sometimes extending below the knee. MRI is preferred to identify the causative intrinsic factors. It is also effective in differentiating pathologies that produce similar complaints such as tendinopathy. (19,20)

4.4.4. Treatment

While entrapments caused by primary causes are treated with physical therapy, problems due to pelvic pathologies aim to eliminate the causative condition. Surgery is indicated for persistent and persistent pain. Nerve decompression is the first surgical method used. If obturator nerve is too damaged to heal with decompression, nerve repair is performed. (19,20)

4.5. Posterior Femoral Cutaneous Nerve Syndrome

A syndrome in which the posterior femoral cutaneous nerve is compressed while passing through the infrapiriform foramen and traveling under the gluteus maximus muscle, causing sensory disturbances in the dermatome area. (21,22)

4.5.1. Anatomy

The posterior femoral cutaneous nerve consists of two or three roots from the posterior branches of the sacral plexus. It travels between the piriformis muscle and the sacral plexus, exiting the infrapiriform foramen with the inferior gluteal nerve. Deep to the gluteus maximus muscle, it distributes to the perineum, thigh and back of the leg. In the thigh, it runs under the fascia lata between the biceps femoris and semitendinosus muscles and gives off branches of the inferior cluneal nerves and perineal branches, which are distributed in the skin on the posterior aspect of the thigh. (6,7)

4.5.2. Etiology

The most common cause is intramuscular injections. Factors such as piriformis muscle spasm or hypertrophy causing narrowing of the infrapiriform foramen may also cause compression of the cutaneous femoris posterior. (21,22)

4.5.3. Clinical Symptoms and Imaging

Sensory tenderness is seen on the posterior aspect between the hip and knee, distal and medial to the gluteal region, perineum, labium majus or posterior part of the scrotum. It is differentiated from radiculopathies of S1 origin by needle EMG. With MRI imaging, the factor causing the compression can be identified. (21,22)

4.5.4. Treatment

Physical therapy applications, daily life modifications, NSAİD, corticosteroids, PRP injections, nerve blockages and surgeries where the nerves are resected are the methods to be preferred in treatment. (21,22)

4.6. Piriformis Syndrome

It is the clinical picture of compression of the sciatic nerve as it passes through the greater sciatic foramen near the piriformis muscle. A detailed examination is necessary to differentiate it from other pathologies that may cause irritation of the Sciatic nerve. Lumbar spinal stenosis, disc herniation and pathologies of the pelvic region present similar symptoms. Approximately 0.6% of patients with low back pain experience pirifromis syndrome. (23-25)

4.6.1. Anatomy

The sacrospinal ligament and sacrotuberale ligament connect the ischial spine and ischial tuberosity to the sacrum and form the greater sciatic foramen and lesser sciatic foramen. Most of the important structures connecting the gluteal region to the pelvis pass through the greater sciatic foramen. Piriformis muscle divides the foramen into suprapiriform foramen and infrapiriform foramen. The superior gluetal nerve and veins originate from the suprapiriform foramen.

The infrapiriform foramen is formed superiorly by the inferior edge of the piriformis muscle, inferiorly by the sacrospinal ligament and laterally by the edge of the greater sciatic notch. The pudendal neurovascular bundle, sciatic nerve, inferior gluteal nerve, posterior femoral cutaneous nerve and inferior gluteal artery originate from here.

The sciatic nerve is the thickest nerve in the body and is a continuation of the sacral plexus. It is formed by the spinal nerves tibial nerve (L4-L5; S1-S3), the ventral part of the plexus, and common peroneal nerve (L4-L5; S1-S2), the dorsal part of the plexus. (6,7)

4.6.2. Etiology

Spasm of the piriformis muscle, trauma and inflammation in the surrounding tissues, scar tissue and local ischemia are the most common causes of impingement. Anatomical variations and the associated change in the course of the sciatic nerve are important in the development of pirifromis syndrome. (23-25)

4.6.3. Clinical Symptoms and Imaging

Pain and paresthesia may develop throughout the course of Sciatic nerve. Pain increases with sitting or walking and decreases with supine position. Trying to get up from a supine position may provoke pain. Symptoms are strongest when the hip joint is flexed, adducted and internally rotated. Ultrasound, MRI, CT and EMG are often preferred to exclude other diagnoses. In addition, MRI is used to detect irritation of the sciatic nerve. (23-25)

4.6.4. Treatment

Stretching exercises, neurodynamic mobilizations, drugs and injections for inflammation are the preferred treatment methods for piriformis muscle in the first stage. Botulinum toxin injection may be preferred for symptom relief. In the absence of a positive result for at least six months, surgical interventions aimed at displacing the piriformis muscle tendon and relieving the nerve are considered. (23-25)

4.7. Meralgia Parestetika

It is a clinical picture that occurs when the lateral femoral cutaneous nerve is compressed or stretched while passing under the inguinal ligament during its course. It is generally more common in women than in men. The incidence increases in military members due to the use of belts. The most common age is between 40 and 50. The incidence in the general population is 0.03% per year. (26,27)

4.7.1. Anatomy

The lateral femoral cutaneous nerve, a pure sensory nerve, arises from the L2-L3 nerve roots. It exits lateral to the psoas major muscle and runs to the SIAS on the anterior aspect of the iliacus muscle. Passing under the inguinal ligament, it divides into two branches, anterior and posterior branches, on the anterior aspect of the thigh and under the skin. The anterior branch provides

sensation to the anterolateral part of the thigh, while the posterior branch is distributed in the posterolateral skin. (6,7)

4.7.2. Etiology

Obesity, sagging belly, the use of large belts due to occupation, pregnancy, tight clothing, factors that increase intra-abdominal pressure such as tumors and surgical operations are the main causes of the syndrome. (26,27)

4.7.3. Clinical Symptoms and Imaging

Symptoms relieved by pelvic compression test in the side lying position confirm the diagnosis. Symptoms are usually unilateral. Pain increases with walking and standing up from sitting. Causes that increase intra-abdominal pressure, such as the Valsalva maneuver, worsen the symptoms. Paresthesia and burning sensation are the most characteristic symptoms. Trophic changes are also seen with prolonged compression. If the compression is thought to be caused by a tumor, a CT, ultrasound or MRI examination is helpful in the diagnosis. (26,27)

4.7.4. Treatment

The aim of treatment is to eliminate the factors that cause compression and to relax the surrounding muscles to relax the nerve. With patient education, it is possible to reduce complaints by restricting the use of tight clothing and belts, or by dietary recommendations for weight loss. NSAİD, capsaicin and GABA derivatives are effective for neuropathic pain. Surgery is considered as a last option in rare but persistent cases. (4,26,27)

4.8. Saphenous Nerve Syndrome

It is a syndrome characterized by pain along the dermatomes of the saphenous nerve caused by compression or stretching of the saphenous nerve as it passes through the adductor canal or in its subsequent course. (4,28,29)

4.8.1. Anatomy

The saphenous nerve, the longest sensory branch of the femoral nerve, leaves the trigonum femorale to enter the adductor canal together with the femorale artery and femoral vein. After entering the canal, it pierces the lamina vastoadductoria towards the end and exits the canal. It pierces the fascia lata between the tendons of sartorius and gracilis muscles. It divides into terminal branches in the distal part of the leg. One of these branches extends medial to the

tibia up to the ankle. The other branch senses the area between the medial aspect of the dorsum of the foot and the first toe. The infrapatellar branch, which leaves the saphenous nerve medial to the knee and receives sensation of the anterior part of the patella, joins the structure of the patellar plexus. Its medial crural cutaneous branches, which runs with the great saphenous vein provides sensory innervation of the anterior and medial aspect of the leg. (6,7)

4.8.2. Etiology

Inflammations, vascular surgeries, meniscectomy and arthroplasty operations, reconstructive surgeries, traumas and scar tissue formation that cause a decrease in the internal volume of the adductor canal predispose to impingement. (4,28,29)

4.8.3. Clinical Symptoms and Imaging

Pain in the medial part of the leg, especially during walking, is the first symptom patients complain of. Deep thigh pain, knee pain and paresthesias in the dermatome area of the nerve in the leg and foot. Resistance to adduction provokes pain. Sensory disturbances are seen in the infrapatellar region. For the clinician, tenderness on palpation along the saphenous nerve is important for diagnosis. EMG tests are utilized for diagnosis. (4,28,29)

4.8.4. Treatment

Rest and conservative treatment often relieve symptoms. Activity modification and oral analgesics, anti-compression pillows, transcutaneous electrical stimulation are effective in relieving symptoms. Surgery to decompress the nerve is recommended for persistent pain. (4,28,29)

4.9. Peroneal Tunnel Syndrome

It is a syndrome with characteristic clinical features that occurs after compression of the common peroneal nerve in a fibroosseous tunnel at the level of the neck of the fibula. Peroneal neuropathy is the most common compressive neuropathy of the lower extremity, while tunnel entrapment is rare. It can occur at any age, including children, but usually affects athletes and impairs performance. (30-32)

4.9.1. Anatomy

The common peroneal nerveis the branch of the sciatic nerve that leaves proximal to the fossa poplitea and travels downward. It is thinner than the tibial

nerve and runs laterally. After separating from sciatic nerve, it travels between biceps femoris muscle and gastrocnemius muscle and comes to the head of fibula. It then surrounds the neck of the fibula, passes under the origin of the perenous longus muscle and enters the peroneal tunnel. As it enters the tunnel, it divides into two branches as deep peroneal nerve and superficial peroneal nerve.

Superficial peroneal nerve lies between the fibula and perenous longus muscle. Moving downwards, it lies proximally between perenous longus muscle and extensor digitorum longus muscle. Superficial peroneal nerve is responsible for the motor innervation of perenous longus muscle and peroneus brevis muscle. In the distal 1/3 of the tibia, the superficial peroneal nerve pierces the fascia cruris and divides into two cutaneous branches. Medial dorsal cutaneous nerve comes to the dorsum of the foot where it divides into two branches and distributes to the toes as dorsal digital nerves of foot. The medial branch carries the sensory innervation of the medial aspect of the 1st toe, while the lateral branch provides sensory innervation of the opposing sides of the 2nd and 3rd toes. Intermediate dorsal cutaneous nerve divides into two branches on the dorsum of the foot and reaches the roots of the digits and carries the sensation of the faces of the 3rd, 4th and 5th digits facing each other with the branches of dorsal digital nerves of foot. The deep peroneal nerve passes between the peroneus longus muscle and fibula, enters deep into the extensor digitorum longus muscle and travels with the anterior tibial artery on the anterior surface of the interosseous membrane of leg. Tibialis anterior muscle provides somatomotor innervation of the extensor digitorum longus, peroneus tertius and extensor hallucis longus muscles. As it passes under the extensor retinaculum , it divides into two branches. The medial branch travels with the dorsalis pedis artery on the dorsum of the foot and branches into the dorsal digital nerves of foot at the 1st digit interval and distributes on the opposing sides of the 1st and 2nd digits. The lateral branch innervates the extensor digitorum brevis muscle. (6,7)

4.9.2. Etiology

Compression in the peroneal tunnel area is often caused by external factors. The neighborhood of the common peroneal nerve with the fibula may cause compression during trauma to the fibula, fractures and surgeries of the fibula. Repetitive inversion and eversion movements of the ankle also predispose to compression. Anatomical variations, prolonged cross-leg sitting, diabetes, obesity, tumors and ganglion cysts may force the common peroneal nerve to compress. (30-32)

4.9.3. Clinical Symptoms and Imaging

The pain occurs proximal to the dermatome of the common peroneal nerve and may radiate into the thigh, and external compression of the tunnel exacerbates the pain. After the onset of pain, gradual motor weakness and atrophy may lead to low foot syndrome. There is difficulty in dorsiflexion and inversion. Strenuous movements in the direction of inversion further provoke pain. MRI is the most appropriate imaging method for diagnosis. Since it is superficial, ultrasound can also be easily visualized and is useful for diagnosis or exclusion. (30-32)

4.9.4. Treatment

The aim of treatment is to eliminate the external factors and to relieve pain and motor loss with physical therapy applications. Neuromuscular electrical stimulation, nerve shifting exercises and the use of appropriate footwear are important for developing low foot syndrome. Local injections and surgical decompression of the tunnel are preferred in advanced cases. (30-32)

4.10. Peroneus Superficialis Syndrome

It is a syndrome characterized by somatomotor changes following compression of the superficial peroneal nervewhere it pierces the crural fascia at the level between the middle and distal third of the leg. It is more common in dancers and athletes. (33,34)

4.10.1. Anatomy

At the entrance of the peroneal tunnel near the head of fibula, the common peroneal nerve divides into two terminal branches, the superficial peroneal nerve and the deep peroneal nerve. This branching point may vary. The superficial peroneal nerve extends distally between the perenous longus and peroneus brevis muscles and provides motor innervation of these muscles. It pierces the crural fascia in the distal 1/3 of the leg and continues as the medial dorsal cutaneous nerve and intermediate dorsal cutaneous nerve. (6,7)

4.10.2. Etiology

Trauma is the most common etiology. Forced inversion and plantar flexion of the foot causes overstretching of the nerve. Surgical trauma, lipomas, narrow boots with high throat, ganglions, repetitive pressure on the foot and dynamic compression can cause the syndrome. (33,34)

4.10.3. Clinical Symptoms and Imaging

Superficial peroneal nerve compression causes pain and sensory disturbances in the dorsum and dermatome areas of the foot. Motor deficits are rare, with weakness in eversion of the foot. Ultrasound can detect thickening of the nerve. MRI is also a helpful imaging modality in doubtful cases. (33,34)

4.10.4. Treatment

It is important to remove the trauma and eliminate other causative factors. Local injections with physical therapy agents constitute the first step of treatment. Neurodynamic approaches and strengthening programs combined with electrical stimulation for motor loss are preferred. Surgery is indicated in cases where relief and improvement cannot be achieved. (33,34)

4.11. Sural Nerve Syndrome

It is a clinical picture characterized by pain and burning after compression or tension of the sural nerve as it travels posteriorly in the distal part of the leg and behind the lateral malleolus. (35)

4.11.1. Anatomy

The sural nerve is formed by the union of the medial sural cutaneous nerve from the tibial nerve and the lateral sural cutaneous nerve from the common peroneal nerve. Medial sural cutaneous nerve branches from the tibial nerve in the popliteal fossa and travels distally under the crural fascia between the two heads of the gastrocnemius muscle together with the small saphenous vein. In the posterior part of the leg, they merge with the lateral sural cutaneous nerve in the midline to form the sural nerve. Sural nerve runs on the surface of peroneus longus and peroneus brevis muscles tendons and exits behind the lateral malleolus. The cutaneous nerve continues as the lateral dorsalis pedis and carries the sensation of the ankle joint, the heel and the lateral side of the foot up to the fifth toe. (6,7)

4.11.2. Etiology

Sports injuries, external trauma, anatomical differences and inappropriate footwear can affect the mechanics of the ankle and cause impingement. Achilles ruptures and surgeries, casts after lower extremity problems and intraneural hematomas are also the main etiologies. (35,36)

4.11.3. Clinical Symptoms and Imaging

Excessive inversion of the foot may create a tension on the sural nerve and a picture characterized by pain and burning in the dermatome areas may be seen. A compression test with knee extension and dorsi flexion with tension on the sural nerve may be helpful for diagnosis. Ultrasound, MRI and EMG examinations may confirm the diagnosis. (35,36)

4.11.4. Treatment

After elimination of the causative factor, nerve recovery by spontaneous regeneration usually occurs. If long-term conservative treatment does not yield positive results, relief is provided by fasciotomy. (35,36)

4.12. Anterior Tarsal Tunnel Syndrome

The deep peroneal nerve is compressed as it passes under the extensor retinaculum and may cause pain in the dermatome area with its branches leaving distal to the retinaculum. Although the exact incidence of the syndrome, which is often overlooked in the literature, is unclear, it has been reported that 5% of people with foot pain and numbness may have anterior tarsal tunnel tendroma. There is no difference in terms of age and gender. Athletes are exposed more than other occupational groups. (37-39)

4.12.1. Anatomy

Deep peroneal nerve enters the anterior tarsal tunnel after innervating all foot extensors except extensor digitorum brevis muscle. It divides into lateral and medial branches within the tunnel. The lateral branch passes under the extensor digitorum brevis tendon and innervates the tarsal and metatarsal joints. The medial branch terminates between the 1st and 2nd toes, providing sensory innervation. (6,7)

4.12.2. Etiology

The anterior tarsal tunnel consists of a tight retinaculum covering soft tissue structures. Movement of structures within the tunnel can cause stretching of the deep peroneal nerve. Acute traumas or repetitive microtraumas, ganglion cysts, inflammatory processes of the tendons, wearing narrow boots, wearing high heels causing continuous plantar flexion of the foot and anatomical deformities are the main etiologies. (37-39)

4.12.3. Clinical Symptoms and Imaging

The clinical picture varies depending on whether the sensory or motor fibers of the deep peroneal nerve are affected. Burning pain is localized in the area between the 1st and 2nd toes. Burning pain at night may awaken patients from sleep. Pressure on the nerve increases the pain. Electrodiagnostic tests are usually not accurate. Motor fibers are mostly affected in lesions proximal to the anterior tarsal tunnel. Ultrasound, MRI and CT are used to detect bone structures and muscle atrophy. (37-39)

4.12.4. Treatment

Treatment depends on the underlying etiology. Elimination of the causative factors is the first step. Physical therapy, anti-inflammatory drug therapy or local injections may alleviate symptoms. Surgery is the most effective method if the factor causing the impingement is anatomical structures. (37-39)

4.13. Tarsal Tunnel Syndrome

The two terminal branches of the tibial nerve, medial plantar nerve and lateral plantar nerve, pass through the sinus tarsi with the related arteries and veins. Compression of the nerve in this narrow fibro-osseous canal creates a clinical picture. Although the incidence is unknown, it can be seen at any age. The incidence is higher in women than in men. 43% of patients have a history of trauma such as ankle sprain. (40,41)

4.13.1. Anatomy

It is formed by the sinus tarsi, calcaneal sulcus, posterior process of talus and medial malleolus. The flexor retinaculum and the tendon of the abductor hallucis muscle cover the medial side of the tunnel. Almost all neurovascular structures pass through the sinus tarsi on the plantar side. The tibial nerve forms the medial plantar and lateral plantar nerves between the flexor digitorum longus and flexor hallucis longus muscles before bifurcating in the tarsal tunnel. (6,7)

4.13.2. Etiology

Extrinsic causes such as mechanical pressure, trauma, surgical scars, increased longitudinal arch of the foot, diffuse edema and diabetes, and intrinsic factors such as tendinopathies, ganglion cysts, lipomas, intense exercise, tumors and inflammation can cause compression of the tibial nerve in the sinus tarsi. (40,41)

4.13.3. Clinical Symptoms and Imaging

In tarsal tunnel syndrome, pain and paresthesia are the most prominent complaints. It gets worse at night or after standing or walking for a long time. The pain is accompanied by numbness and pins and needles sensation. Radiologic examinations, EMG and nerve conduction studies often produce abnormal responses in patients with tarsal tunnel syndrome. (40-42)

4.13.4. Treatment

Tarsal tunnel syndrome can be treated conservatively and surgically. Which method is preferred is often decided according to the causative factor and the degree of symptoms that develop. In addition to rest, various methods such as avoiding repetitive trauma, immobilization, physical exercise, anti-inflammatory drugs and local corticosteroid injection are used in the treatment process. If conservative treatment does not relieve the patient's symptoms, surgical methods are preferred. (40-42)

4.14. Medial Plantar Nerve Syndrome

Medial plantar nerve syndrome is a condition characterized by shooting pain following compression of the medial plantar nerve in an osseo-fibro-muscular tunnel lying between the abductor hallucis muscles and the navicula bone. (43)

4.14.1. Anatomy

After passing under the flexor retinaculum, the medial plantar nerve passes deep to the abductor hallucis muscle and distally between the abductor hallucis and the flexor digitorum brevis muscles. At the level of the metatarsal bones, it branches into the common plantar digital nerves. At the base of the toes, the common plantar digital nerves gives off the proper plantar digital nerves, which innervates the plantar side of the medial side of fingers 1-3 and the 4th toe. (6,7)

4.14.2. Etiology

Eversion of the foot causes the medial plantar nerve to stretch more within the fibromuscular tunnel. Repeated trauma causes inflammation. Running for long distances with the ankle in valgus can damage the medial plantar nerve. It is described as "jogger's foot" because it is common in runners. (43)

4.14.3. Clinical Symptoms and Imaging

Patients often complain of burning and sharp pain radiating towards the plantar aspect of the 1st and 2nd toe when running. Variations in the bony structure of the foot can be identified by ultrasound. MRI gives an idea of both the extent of the lesion and the severity of denervation. (43,44)

4.14.4. Treatment

Changing the shoes, revising the training program, adapting the running mechanics to the athlete and regular use of anti-inflammatory drugs are important in terms of treatment. Nerve blockages are used in persistent pain. (43)

4.15. Baxter Neuropathy

Baxter's neuropathy is an entrapment neuropathy that causes heel pain after the first branch of the lateral plantar nerve is compressed as it passes through the inferior medial border of the heel. (38,45)

4.15.1. Anatomy

The lateral plantar nerve runs between the flexor digitorum brevis muscles and the quadratus plantae muscle, giving off the deep and superficial branches. Before branching, the first branch of the lateral plantar nerve innervates the abductor digiti minimi muscle. The superficial branch is distributed on the sole of the foot, lateral to the 4th toe and on the skin of the 5th toe. Deep branch is more of a somatomotor branch and innervates all interosseal and lumbrical muscles except the 1st lumbrical and 4th interosseal muscles, quadratus plantae and adductor hallucis muscles (6,7)

4.15.2. Etiology

Chronic inflammatory changes and sporting activities are the most likely causes of Baxter neuropathy. Excessive eversion of the foot, plantar fasciitis, perifoot cysts and muscle hypertrophy can also cause impingement. (38,45)

4.15.3. Clinical Symptoms and Imaging

It is characterized by chronic heel pain. The pain is sharp and radiating. It worsens after activities such as walking and at night. The most obvious sign is intense tenderness in the abductor hallucis fascia. A Tinel's test in this area gives a positive response. There is decreased abduction of the fifth finger due to

entrapment and paresthesia with compression. MRI is used to detect edema and muscle hypertrophy, while ultrasound can be used to monitor neural changes. (38,45)

4.15.4. Treatment

The use of heel pads and insoles, physical therapy to stretch the Achilles tendon and plantar fascia, and anti-inflammatory drugs often relieve symptoms. Surgical decompressions or open operations are preferred after unsuccessful conservative treatment. (38,45)

4.16. Morton's Metatarsalgia

Morton's Metatarsalgia is an entrapment syndrome that causes forefoot pain after compression of the nerves in the metatarsal tunnels. The most common sites of neuromas, which are frequently seen in middle-aged women, are between the 3rd and 4th metatarsal heads. Women are exposed five times more frequently than men. While it is usually seen in one foot, it can also be affected bilaterally. (46,47)

4.16.1. Anatomy

The common plantar digital nerves from the medial plantar nerve and lateral plantar nerve pass through the tunnels between the deep transverse metatarsal ligament and superficial transverse metatarsal ligament, which connect the metatarsal heads. Within the tunnels, they branch into proper digital nerve. (6,7)

4.16.2. Etiology

Factors such as trauma, repetitive joint movements, inflammatory diseases, use of shoes with pointed toes, walking on hard ground cause morton metatarsalgia. (46,47)

4.16.3. Clinical Symptoms and Imaging

The pain is often localized, increasing with hyperextension of the fingers but not radiating. Especially between the 3rd and 4th metatarsal heads there is a point where the pain is felt. Vascular changes can be seen with thickening of the surrounding structures due to compression. Palpation in the affected area reveals symptoms. Lidocaine injection is used for diagnostic purposes and MRI imaging is performed to exclude other pathologies. Ultrasound examination is also helpful for diagnosis. (46,47)

4.16.4. Treatment

Physical therapy, anti-inflammatory drugs and local corticosteroid injections are effective in eliminating symptoms, as well as restricting the use of high-heeled and narrow-tipped shoes. Surgery is indicated in resistant cases. (43,46,47)

5. Conclusion

Reported trunk and lower extremity entrapment neuropathies are much fewer compared to upper extremities. This statistic is probably related to the fact that neuropathic complaints such as pain, loss of strength and numbness are thought to be caused by conditions such as herniated disc and spinal stenosis. Correct diagnosis during the treatment process prevents loss of time in relieving complaints and saves the patient from a financial burden. Therefore, it is very important to examine the anatomical structures that cause entrapment neuropathies and to know the clinical signs and symptoms well in order to make the correct diagnosis. (48)

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CHAPTER IX

SKIN MORPHOLOGY AND CLINICAL IMPORTANCE

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1. Introduction

The skin, which wraps around the body like a dress and serves as a static barrier protecting from the outside world, integrates with various structures such as nerves and vessels just beneath it. Besides different pathologies related to the skin, the importance given to the skin's architecture and biomechanics regarding its cosmetic function is often overlooked. If the skin is to be cosmetically improved, it is crucial not only to focus on the biology of the skin but also to aim to understand its living dynamic structure. Therefore, understanding the dynamic microanatomy of the skin from the deep fascia to the skin surface is of vital importance in dermatological and surgical terms.

2. Skin Embryology

During the embryonic period at the 3rd week, gastrulation occurs, from which the epidermis develops from the surface ectoderm, and the dermis develops from the mesenchyme located beneath the surface ectoderm. The hypodermis, found beneath the dermis, is the subcutaneous tissue and is macroscopically known as the fascia subcutanea. While the epidermis and skin appendages are of ectodermal origin, the dermis and hypodermis are of mesodermal origin. Protrusions in the epidermis and papillae in the dermis start to form at the 10th week. It is observed that the layers of the epidermis can be distinguished after the 4th month, while the stratum corneum is known to form after the 21st week. (1,2)

Around the 10th week of the fetal period, it is observed that the formation of fingernails and toenails begins. By the 17th week, the nail plate has grown to cover approximately the entire nail bed. And by the 32nd week, the fingernails have reached the fingertips, while by the 36th week, the toenails have reached the tips of the toes. (1,2)

Hairs, at the beginning of the fetal period (9-12th week), proliferate from the germinative layer towards the dermis as hair buds, but cannot be distinctly identified until the 20th week. The hair papilla forms with the invagination observed at the terminal ends in the dermis. Later, the dermal root sheath and the arrector pili muscle develop from the mesenchymal cells inside these papillae. The first hairs appear as lanugo hairs on the lips, eyebrows, and chin area at the end of the 12th week. The number of these hairs increases between the 17th and 20th weeks, but they fall out after birth. (1,2)

Sweat, sebaceous, and mammary glands, which are skin glands, begin to form during the 3rd to 4th months of intrauterine life. (1,2)

3. Skin Morphology

The skin (cutis) is the largest organ in the body. It constitutes about 15% of an adult's total body weight and covers an area of approximately 3000 square inches (1,9 m^2). (3,4) Its weight is about twice that of the brain, varying between approximately 3-5 kg. While the skin is generally 1-2 mm thick, this thickness varies according to the function and region of the body. The skin on the eyelids is 0.5 mm thick, whereas the skin on the soles of the feet is 3-4 mm thick. (3)

The skin covers the entire body surface and surrounds natural openings, thereby significantly preventing interaction between the body and the environment and minimizing harmful effects related to mechanical, chemical, thermal, osmotic, and light from the outside. (5) From the surface to the deep, the skin consists of three layers: the epidermis, the dermis (corium), and the tela subcutanea (subcutis).

3.1. Epidermis

The epidermis is composed of multi-layered keratinized squamous epithelium and consists of many layers close to the skin surface. The epidermis contains four main cell types, most of which are keratinocytes, making up 90% of the cells in this layer. Melanocytes constitute 8% of the epidermal cells and are responsible for the production of melanin pigment. Langerhans cells and Merkel cells are also found in the epidermis. The epidermis is avascular, and its

oxygenation, provision of metabolites, and removal of metabolic waste products depend on the blood vessels in the dermis. (5,6)

The epidermis consists of five different layers from superficial to deep: stratum corneum, stratum lucidum, stratum granulosum, stratum spinosum, and stratum basale. Except for the stratum corneum, the other four layers are also referred to as the stratum germinativum. (7) However, there are also different sources in the literature that consider only the stratum basale as the stratum germinativum.

3.1.1. Stratum Corneum

It is the top layer of the epidermis, consisting of 25-30 layers of flattened dead keratinocytes. (6) The cells are arranged in regular, vertical stacks and appear to be tightly connected by cell membranes. The cells contain keratin protein, which helps protect the skin and underlying tissues from heat, microorganisms, and chemicals. Intracellular lipid from the stratum granulosum adheres the cells together and is vital in preventing the cells from drying out. As cells progress through the stratum corneum, they lose their adhesiveness and shed individually or in clusters known as scales. (8) Since the stratum corneum is the outermost layer between the body and the external environment, it is significantly susceptible to abrasion and wear. Cells are constantly lost from this layer. If a part of the skin is subjected to continuous friction, a hard callus, which indicates abnormal thickening of the stratum corneum, can form, while areas not exposed to trauma become thinner. (6)

3.1.2. Stratum Lucidum

This layer between the stratum corneum and stratum granulosum is found only in areas of the body where the skin is thick, such as the palms of the hands and the soles of the feet. It is a thin, clear layer consisting of dead skin cells. Elaidin cells, which give it a homogeneous and transparent structure, are found in this layer. Due to its structure, this layer appears translucent under the microscope and is also called the clear layer. The thickness of the stratum lucidum is controlled by the rate of mitotic division of epidermal cells, while its darkness is determined by melanosomes in the stratum basale. Additionally, this layer provides some degree of waterproofing to the skin. (3,6)

3.1.3. Stratum Granulosum

As cells progress from the epidermal layers towards the skin surface, they elongate and flatten horizontally to form the stratum granulosum. The

stratum granulosum consists of three to five layers of flattened keratinocytes. This layer contains keratohyalin granules, which are not found in other layers of the epidermis and connect keratin filaments to each other. These granules are lipid-producing, membrane-coated, layered granules that spread into the spaces between cells and assist in their adhesion. (6,9)

The thickness of the stratum granulosum is proportional to the thickness of the overlying stratum corneum. There is an inverse relationship between the thickness of this layer and the inadequacy of keratinization in the stratum corneum, which characterizes a nutritional disease called parakeratosis. (6,9)

3.1.4. Stratum Spinosum

The stratum spinosum is 5 to 12 cells thick. The cells of this layer, composed of multiple layers of cells, flatten towards the superficial layers. They come together through cytoplasmic bridges called desmosomes. As cells progress through this layer, they constantly break apart and reassemble desmosomes. These desmosomes have spike-like projections that bring adjacent cells closer together, hence the name spinosum, meaning “thorny” in reference to these spikes. The threads extending from one cell to another through desmosomes are called tonofibrils. Tonofibrils extend from the stratum basale to the stratum lucidum in a perpendicular manner to the skin surface and continue to extend parallel to the skin when reaching this layer. When pressure is applied to the skin, the curved structures of these threads open up, becoming oblique, thereby spreading the applied pressure over a larger area and providing protection against impact. When the pressure is relieved, they return to their original state. With this structure, the stratum spinosum contributes to the elasticity and tensile strength of the skin. Additionally, this layer is also referred to as the Malpighian layer along with the stratum basale. (7,10)

3.1.5. Stratum Basale

The stratum basale is the bottom layer of the epidermis and is also called the stratum germinativum due to its formation from cylindrical cells. The stratum basale consists of a single layer of columnar keratinocytes. Among the other cell types found in this layer are melanocytes and Merkel cells. The stratum basale is the only layer of the epidermis composed of cells capable of division. Keratinocytes in the stratum basale undergo mitosis, producing two daughter cells, one of which remains in the stratum basale while the other migrates towards the surface of the epidermis through other layers. This process takes

about 28 days in an epidermis with an average thickness of 0.1 mm. Mitotic division occurring in this layer prevents skin loss. (7,10)

3.1.6. Skin Color and Factors Affecting It

The color of the skin varies according to race, age, sex, and body region. People are classified into different races based on skin color, including white, black, yellow, and red. In fact, skin color is genetically determined but can also be altered by some environmental and physiological factors. Among genetic factors are the amount of melanin and the size of melanin granules, while environmental factors include sunlight, ultraviolet radiation (UV), and X-rays. Physiological factors include accumulation of carotene and pregnancy. (11)

Skin color is related to the amount of melanin in the epidermal cells. The more melanin produced, the darker the skin becomes. (6) Many studies in the literature report no racial difference in the number of melanocytes. However, the actual number of melanocytes can vary from person to person and from one anatomical region of the body to another; for example, the scalp and forearms have the highest number of melanocytes. (11)

3.1.7. Cells Located in the Epidermis

3.1.7.1. Keratin

The new cells formed in the stratum basale are pushed upward. As these cells move away from the stratum basale, they receive less nutrients and consequently die. When these cells reach the stratum corneum, they lose their nuclei, creating a void in their centers. Around this void, a substance called keratin forms, and the cells are surrounded by a tough outer membrane. These hardened cells accumulate more keratin, a fibrous protein that plays a role in protecting the skin from heat, chemicals, and microorganisms. In healthy skin, there is a balance between the formation of new keratinocytes in the stratum basale and the shedding of dead keratinocytes from the stratum corneum. (5,6,10)

3.1.7.2. Langerhans Cells

Langerhans cells are found in the stratum spinosum, dermis, lymph nodes, and thymus. (5) These cells are produced in the red bone marrow and later migrate to the stratum spinosum where they can participate in immune responses against microorganisms. They function by attracting and phagocytizing microbes, and by activating lymphocytes to destroy appropriate cells. Langerhans cells are responsible for aiding other cells of the immune system in recognizing and eliminating invading microorganisms. (9)

3.1.7.3. Melanocytes

Melanocytes are cells that produce melanin, a pigment that protects the skin from the harmful effects of UV light. Melanocytes have long, thin projections containing melanin granules. These projections extend between keratinocytes and transfer the granules to them. Once inside keratinocytes, the granules gather around the nucleus to form a protective coating, shielding the keratinocytes from damage caused by UV light. (5,6,10)

3.1.7.4. Merkel Cells

Merkel cells are oval-shaped modified epidermal cells found in the stratum basale. These cells serve a sensory function as mechanoreceptors for light touch. Although they are found in the palm, sole, oral, and genital mucosa, they are most abundant in the fingertips. They interact with free nerve endings in the skin. (12)

3.2. Dermis (Corium)

The dermis, located between the epidermis and the subcutaneous tissue (hypodermis), is the thickest layer and is composed of numerous cells. The main proteins found in this layer are collagen and elastin, synthesized and secreted by fibroblasts. Collagen constitutes about 30% of the volume or 70% of the dry weight of the dermis. The main functions of the dermis are providing tensile strength, support, moisture retention, and supplying the skin with nutrients, blood, and oxygen. It also protects underlying muscles, bones, and internal organs. The dermis contains lymph vessels, nerve endings, hair follicles, and glands. Additionally, it includes sebaceous glands that secrete sebum, an oil-rich substance that lubricates the skin, prevents dehydration of the stratum corneum, and preserves its barrier function. Because it forms the main portion of the skin, it is also called the cutis vera, meaning the true skin. (7,9,13)

The dermis consists of two layers: the superficial papillary layer (stratum papillare) and the deeper reticular layer (stratum reticulare). The papillary layer is composed of collagen and reticular fibers. Its distinct and unique pattern allows for the identification of fingerprints for each individual. On the upper surface of this layer, there are projections called papillae, which are cone-shaped, leaf-like, or fine thread-like. These papillae contain sensory nerve endings, Meissner's corpuscles, and capillaries for nourishing the skin. (7,14) The reticular layer is made up of a strong connective tissue containing collagen and elastic fibers, which anchor the dermis to the hypodermis. Sweat glands, hair follicles, nerves, and blood vessels are found in this layer. (9)

3.3. Subcutaneous Tissue (Hypodermis, Subcutis)

The subcutaneous tissue, also known as the hypodermis, separates the dermis from the deep fascia covering the muscles below. Its function is to provide a continuous blood flow (blood reservoir) to the dermis for regeneration. It primarily consists of adipose tissue, which acts as a cushion between the skin layers, muscles, and bones. It supports the mobility of the skin, shapes body contours, and insulates the body. (7,15)

The hypodermis consists of two layers: the superficial layer (lamina superficialis) and the deep layer (lamina profunda). The superficial layer contains abundant adipose tissue, while the deep layer contains elastic tissue. Although these two layers are tightly adhered in most parts of the body, they can be easily distinguished in some areas. The hypodermis is thicker in regions such as the breasts, buttocks, and abdomen, where it contains more adipose tissue. The adipose tissue found in these areas contributes to the formation of feminine body contours. (7,15)

3.4. Appendages of the Skin

3.4.1. Nail (Unguis)

The nails, located on the dorsal surface of the distal phalanges of the fingers, are made of layers of keratin. They provide protection to the fingertips, enhance sensation, and aid in grasping small objects. The edge of the nail, which is connected to the skin on the sides, is called the lateral margin or margo lateralis, while the free edge at the front is called the free margin or margo liber, and the concave narrow area beneath the margo liber is called the hyponychium. The nail plate is composed of matrix keratinocytes, while the underlying nail bed, which contains blood vessels, nerves, and melanocytes, is part of the nail matrix and has parallel ridge projections. Beneath the nail bed, there is a wide network of capillaries, making it generally pink in color. The root part of the nail behind it is called the radix unguis, and the crescent-shaped structure above it is called the lunula. The fold of skin behind the lunula is called the eponychium (cuticle), which is the stratum corneum layer of the skin. The stratum germinativum layer of the skin at the back is the matrix unguis. (7,9)

The fingernails grow at an average rate of 0.1 mm per day; this is two to three times faster than the growth rate of toenails. Due to their slow growth rate, toenails can provide information about toxic exposures or illnesses from months earlier. For example, arsenic poisoning can cause horizontal hypopigmentation known as Mees lines across all nail plates. (16)

3.4.2. Hair (Pili)

From the outside, hair appears as thin, flexible tubes made of dead, fully keratinized epithelial cells. However, within the skin, hair is part of live hair follicles, cylindrical epithelial outgrowths towards the dermis, and the hypodermis expanding towards the hair root surrounding the mesenchymal-derived dermal papilla at the base. The length, diameter, color, and cross-sectional shape of hair vary among different ethnic groups and individuals. (3,17)

Hair consists of two parts: the radix pili located within the skin and the visible hair shaft called the scapus pili (corpus pili) on the body surface. (3,9,17)

The hair shaft, known as the scapus pili, corresponds to the epidermis' stratum corneum layer and consists of medulla, cortex, and cuticle cells arranged from the center to the periphery. The cortex, representing the majority of the hair fiber composition, is a layer comprising approximately 50-60 pieces, playing a significant role in the physical and mechanical properties of the hair. The cuticle layer covers the hair from the root to the tip of the epidermis, consisting of flat, overlapping cells. The integrity of this layer is crucial for protecting the cortex from physical and chemical assaults, maintaining the hair in a clean and untangled state, and greatly influencing its appearance. (3,9,17)

The hair root, known as the radix pili, anatomically consists of three parts: upper, middle, and lower. The upper part comprises two sections: the infundibulum and the isthmus. The portion from the epidermis to the sweat gland duct, known as the sudoriferous duct, constitutes the infundibulum, while the section from the sudoriferous duct to the arrector pili muscle is the isthmus. The part containing the bulge region is the middle section, while the bulb region, forming the lowest part of the hair follicle, constitutes the lower section. (3,17)

The radix pili is located within the folliculus pili, formed as the epidermis extends downward. The folliculus pili contains the other four layers of the epidermis except for the stratum corneum: the outermost layers include the stratum basale and stratum spinosum, while the innermost layers consist of the stratum granulosum and stratum lucidum. If the hair is straight, the folliculus pili follows a straight course, whereas if the hair is curly, it exhibits a curly path. It then extends down to the hypodermis, terminating as the bulb of the hair. Hair formation and growth occur here. The dermis beneath contributes from below, forming the papilla pili. This area is rich in blood vessels. (3,12,17)

The radix pili is divided into two regions: the lower region, which contains undifferentiated cells, and the upper region, which contains differentiated cells. Along the widest part of the papilla pili, the Auber line critically separates these

two regions. Below the Auber line lies the follicular matrix, where each cell is actively mitotic, along with the dermal papilla. Cells move from the matrix towards the upper part of the bulb, where they increase in volume and elongate vertically. (3,17)

On the side with a wide angle between the hair follicle and the epidermis, there is a muscle made of smooth muscle fibers called the arrector pili muscle. Between these two structures lies the sebaceous gland. The innervation of the arrector pili muscle is provided by the sympathetic system. When these muscles contract, the hairs become erect, assisting the nearby sebaceous gland in releasing its secretion by applying pressure to it. (3,7,17)

There are four types of hair follicles in the body: lanugo, vellus, intermediate, and terminal. Lanugo hairs are the hairs that form during pregnancy and shed between the 32nd and 36th weeks of gestation, making way for vellus or terminal hairs. These hairs are very fine, soft, without a medulla, and appear colorless because they lack pigment. Their average length is about 2 mm. Vellus hairs, also known as peach fuzz, are thin, lightly colored hairs that replace lanugo hairs, with an average length of about 1 cm. These hairs are found on the entire body surface except for the palms of the hands, soles of the feet, mucous membranes, and semi-mucous membranes, and they do not have sebaceous glands in their hair follicles. Vellus hairs transition into terminal hairs in the armpits and pelvic region around the ages of 7-9. Intermediate hairs are the first hairs seen on the scalp after birth, appearing between 3 months and 2 years of age. These hairs are soft, contain little pigment, and have a fragmented medulla, with an average length of about 2 mm. Terminal hairs are thick, pigmented, long hairs with a medulla, and their size and diameter vary depending on their location on the body. (3,17)

3.4.3. Skin Glands

3.4.3.1. Sebaceous Glands (Holocrine Glands)

Sebaceous glands are simple, branched acinar glands. These glands are typically located between the dermis of the skin and the hair follicle, and their ducts open into the hair follicle. They are most commonly found on the face, neck, and back. (9) While not all sebaceous glands are associated with hair follicles, many of them are. Sebaceous glands secrete sebum, an oily substance composed of triglycerides, cholesterol, proteins, and organic salts. Sebum coats the surface of the hair, protecting it from drying out and becoming brittle. Additionally, sebum helps prevent excessive evaporation of water from the skin, thus keeping the skin soft and supple. (6)

In addition to its lubricating function, sebum also possesses antifungal and antibacterial properties. (9) Hormonal activity during adolescence can lead to excessive sebum production due to the overworking of the sebaceous glands, which can sometimes result in the presence of open comedones (blackheads) and closed comedones (whiteheads). Sebaceous glands are not under motor stimulation but are influenced by androgens. (7)

3.4.3.2. Sweat Glands

The sweat glands, which extend from the epidermis to the dermis and hypodermis in a tube-like structure, terminate in a coil-like cluster. The coiled parts are called portio terminalis, the openings to the skin are called porus sudorifer, and the canal section in between is called ductus sudorifer. While the ductus sudorifer runs straight in the upper part of the dermis, it is coiled in the lower part, and in thick areas of the epidermis such as the hands and feet, it is spiral-shaped. There are two types of sweat glands in the body: eccrine and apocrine. (7)

Eccrine sweat glands (gll. sudorifer eccrina) are simple, coiled glands distributed throughout many regions of the skin, especially on the forehead, palms, and soles of the feet. Their ducts open onto the skin surface. These glands primarily produce sweat, which consists mostly of water but also contains sodium and chloride ions, urea, uric acid, ammonia, amino acids, glucose, and lactic acid. Approximately 600 ml of sweat is produced per day. Sweat glands play an important role in thermoregulation through evaporation. (6)

Apocrine sweat glands (gll. sudorifer apocrina) are not active during childhood but are activated by sex hormones during puberty. (10) Unlike eccrine glands, these glands have larger terminal portions and thicker ducts, and they empty their secretions into hair follicles. The sweat produced by apocrine glands is slightly viscous and has a milky or yellowish appearance. When it is secreted, it is odorless. However, when it comes into contact with bacteria on the skin surface, these bacteria metabolize the components of sweat, producing a musky odor commonly known as body odor. Unlike eccrine glands, apocrine glands do not contain glycogen. (6,11)

3.5. Fibers Found in the Structure of the Skin

The collagen and elastin in the dermis are organized into a woven network of tissue fibers that provide the skin with its ability to stretch and contract, and they contribute significantly to its tensile strength. Collagen, a protein that

accounts for approximately 70% of the dry weight of the dermis, is crucial in this regard. When the skin is stretched, collagen fibers prevent tearing due to their high tensile strength. (6,16)

Elastin fibers are synthesized by fibroblasts, they are thinner than collagen, and they are found intertwined between collagen bundles. Elastin also possesses elastic properties that allow the skin to return to its normal position after being stretched. As one ages, there is a decrease in the number of collagen fibers, which become hardened and fragmented, losing their shape and becoming entangled with each other. Meanwhile, elastic fibers lose some of their elasticity, thicken into bundles, and become worn out. These changes result in the appearance of wrinkled skin. (6)

3.6. Nutrition of the Skin

There are two main arterial networks involved in the nourishment of the skin. The deep plexus is located where the dermis meets the subcutaneous fat layer. This network supplies blood to the layers of the dermis and subcutaneous tissue. From this plexus, small branches extend to nourish the hair follicles, sweat glands, and other structures within the dermis. At the top level of the dermis, the superficial plexus branches out and carries blood vessels to the border between the epidermis and dermis. (9)

3.7. Receptors Found in the Skin

Receptors in the skin enable the perception of sensations such as touch, pain, pressure, and vibration, providing the skin with the characteristic of being a sensory organ. These receptors are classified into different groups based on their functions, locations, and morphological structures. Based on their location, they are divided into three groups: exteroceptors, interoceptors, and proprioceptors. Morphologically, they are classified into encapsulated and unencapsulated receptors. While some receptors are specialized for only one sensation, others can perceive multiple sensations. For example, the sensation of pain is perceived only by free nerve endings, whereas the sensation of touch is perceived by receptors such as Ruffini corpuscles, Meissner corpuscles, Merkel discs, and hair follicle receptors. (3,7)

Encapsulated receptors include Meissner corpuscles, Vater-Pacini corpuscles, Krause end bulbs, and Ruffini corpuscles. Among these, Meissner corpuscles perceive touch and tactile discrimination in hairless skin. Vater-Pacini corpuscles detect vibrations and rapid mechanical changes. Krause end bulbs perceive pressure and temperatures below 20 degrees Celsius, while Ruffini

corpuscles detect touch, pressure, and stretching sensations at temperatures above 20 degrees Celsius. (7)

Unencapsulated receptors include free nerve endings, Merkel discs, and hair follicle receptors. Among these, free nerve endings perceive pain, pressure, touch, and temperature, while Merkel discs are pressure receptors in hairless skin. Hair follicle receptors, on the other hand, perceive the sense of touch around the entire hair follicle as a nerve network. (7)

3.8. Gender-Based Differences

Gender differences in the skin can be partially attributed to hormonal differences between genders, which regulate the distribution of facial and body hair, sebum production, sweating, and skin pH. Structurally, male skin is thicker compared to female skin. In women, estrogen loss during menopause can further thin the skin, although this can be reversed with estrogen therapy. Studies have reported that men have smaller fat compartments compared to women, with the opposite being true for women. Additionally, in women, the saturation of fibrous compartments with fat leads to the formation of cellulite dimples. (15)

3.9. Age-Related Differences

One of the most studied areas of skin anatomy is related to aging skin. Clinical signs of aging skin include xerosis, melanocytic hyperplasia, telangiectasia, and loss of elasticity. With aging, the number of topographic cutaneous channels decreases, leading to the folding and deepening of visible lines into larger plateau areas. Other causes of wrinkle formation include thinning of the dermis, thickening of the stratum corneum, thinning of the stratum spongiosum, thinning of the epidermis, and loss of type IV and VII collagens in the dermoepidermal junction at the base of wrinkles. This suggests that changes in any of the fibrous layered components of the skin's tissue properties can lead to bending of the surface. Collagen naturally becomes sparser and less soluble in aged skin, while in sun-damaged skin, it thickens and becomes more soluble; elastin accumulates in the stratum papillare of the dermis, making the skin mechanically more fragile. Research has found a general loss of skin volume to increase from 30% at 50 years to 52% at 80 years; thus, there appears to be a relatively increased density of elastic fibers. (3,14,15)

In healthy, undamaged skin, the distribution of elastic fibers is uniform throughout the dermis, while clustering of elastic fibers in the stratum papillare

is a distinctive sign of photoaged skin. Components of the elastic fiber network, such as fibrillin-rich microfibrils, are hypothesized to play a role in absorbing the harmful effects of UV. (3,14,15)

Despite the richness of studies conducted on aged skin, it is remarkable that very little is known about the entirety of structures that visibly degenerate over time. (15)

3.10. Regional Variations in the Body

The characteristics of the skin exhibit significant differences across various regions of the body in terms of topology, pH, temperature, humidity, and microbiology. For instance, the thickness of the skin in areas like the face can range from 0.1 mm on the upper eyelid to 1 mm on the upper lip and nose. Studies have reported significant variations in elasticity among different parts of the body. For example, while the skin of the pelvis is highly lax, the skin of the shoulders, chest, and abdomen, despite having similar collagen concentrations, is less lax. This indicates that the overall elastic modulus of the dermal and hypodermal structure is influenced by these regions. Additionally, the distribution of skin ligaments results in different mechanical properties of the skin in various body regions. (14,15)

3.10.1. Face

The skin on the face is rich in skin appendages such as hair follicles and sebaceous glands, but poor in apocrine sweat glands. Prolonged exposure to the sun can lead to thickening, dryness, and wrinkling of the skin due to an abnormal accumulation of elastin fibers in the dermis. (18)

The forehead is rich in eccrine sweat glands and especially abundant in sebaceous glands in males. The skin on the eyelids, known as the eyelids, is thicker compared to other facial areas and has more prominent dermal papillae. Apocrine sweat glands, known as Moll glands, are located between and behind the eyelashes. Sebaceous glands, known as Zeis and Meibomian glands, open directly onto the epidermal surface. The nose is the region with the highest density of sebaceous glands on the face. Lips are highly mobile due to their role in human speech. Solar elastosis is commonly observed, particularly on the lower lip skin due to frequent sun exposure. While vellus hairs are present on the skin of the lips in individuals of all ages and both sexes, terminal hairs indicate that an individual is an adult male. Eccrine glands are absent at the vermilion border of the lips. The epidermis of the lips also contains numerous Merkel cells. (3,18)

3.10.2. Trunk

The dermis of the skin on the back contains the thickest stratum reticulare layer in the body. Many studies have reported that the average thickness of the skin in this area exceeds 4 mm. Furthermore, these studies have identified the absence of apocrine glands and the presence of only a few eccrine glands, with pilosebaceous units predominantly located in the midline. (3,14,18)

In the female body, the nipple area contains large collecting ducts that open onto the skin surface. These ducts become more prominent, especially during pregnancy. The male nipple has a similar structure to the female nipple but lacks large collecting ducts. (3,14,18)

3.10.3. Perineum

It is reported that the perineal area of the skin contains a large number of melanocytes and apocrine sweat glands. In the anal area, vellus hairs are observed in addition to these sweat glands. In the female genital structures, such as the labia minora, there are no subcutaneous fat tissue, eccrine sweat glands, or hairs. However, it is reported that eccrine sweat glands and sebaceous glands are sometimes seen on the sides of the inner labia in some women. Terminal hairs are found on the lateral part of the labia majora, another structure of the female external genital system. Especially in young women, the skin subcutaneous tissue in this part of the vulva is distinctly visible. (3,18)

In many studies in the literature, it is reported that bundles of the cremaster muscle are seen in the dermis of the scrotum, which are structures of the male genital system. The epidermis of the glans penis is keratinized in circumcised males, whereas it is not keratinized in uncircumcised males. In both cases, there are no hairs on the glans penis. (3,18)

3.10.4. Extremities

The stratum spinosum layer of the epidermis, which is one of the layers of the skin of the extremities, is thicker than that of the body, while the dermis is thinner. Solar elastosis is more pronounced in the lower extremities compared to the upper extremities. There is a slightly verrucous appearance in the epidermis of the knees and elbows. The skin on the palms and soles of the feet is thick to provide protection against constant friction and pressure. It is reported that both areas contain a significantly thickened stratum spinosum and stratum granulosum. These areas are devoid of pilosebaceous units but contain numerous eccrine glands. It is also characteristic for these areas to contain a large number of receptors. The subcutaneous tissue of the fingers

is rich in Pacinian corpuscles and Merkel cells, and there are no hairs on the dorsal aspects. (3,10,18)

3.11. Functions of the Skin

3.11.1. Sensory Function

The skin, being the outermost layer of our body, detects changes occurring in the external environment, thus forming sensations such as cold, heat, pain, touch, and pressure. The skin accomplishes this function through the receptors located on its surface. With approximately one million receptors stimulated by receptors in the skin, with a large amount of them being present in the face and extremities, differences in sensory sensitivity occur. (3,5)

3.11.2. Thermoregulation Function

In terms of maintaining the balance of body temperature, the thermoregulatory function of the skin is of vital importance. In order for internal organs and other structures in the body to function properly, the human body needs to maintain a constant temperature range. To achieve this, mechanisms regulating heat in the skin include sweating, fat insulation, piloerection (hair standing up), and control of blood flow. The temperature perceived by receptors in the skin is transmitted to the hypothalamus, and when the body temperature rises above 37˚C, sweat glands are stimulated to produce sweat. Sweat works as a mechanism to lower body temperature and evaporates when secreted onto the skin, thus cooling the body through the process of evaporation. (3)

The blood vessels in the skin play a role in regulating blood flow by constricting in cold temperatures and dilating in warm temperatures. In situations where body temperature increases, the skin increases blood flow to cool itself down. Conversely, in cold temperatures, vasoconstriction reduces the flow of warm blood from the body's core to the extremities, thus minimizing heat loss. (3)

These regulatory mechanisms for temperature control are not fully developed in younger ages, increasing the risk of hypothermia. With aging, the function of these mechanisms decreases. Therefore, elderly skin experiences difficulty in perceiving temperature changes and responding to them. (19,20)

3.11.3. Protective Function

The skin acts as a barrier for the organs underneath, protecting them physically against various external conditions. It prevents water and fluid loss, allowing internal organs to maintain their normal structure without drying out,

while also preserving the elasticity of the skin and maintaining the body's fluid and electrolyte balance. The skin also contains acidic secretions that prevent the proliferation of harmful microorganisms from the external environment. (5,9)

3.11.4. Vitamin D Synthesis

Vitamin D synthesis is a biological process that occurs as a result of exposure of the skin to sunlight. UVB rays convert 7-dehydrocholesterol in the skin into the active form of vitamin D, known as vitamin D3. This vitamin is later activated in the liver and kidneys, converting into different forms. It is reported that active vitamin D supports bone health by increasing the absorption of minerals such as calcium and phosphorus. It also regulates the activation of immune system cells and controls inflammation. (5,6)

3.11.5. Psychological Function

The skin is an important part of a person's appearance. Additionally, it is known to play a significant role in psychological health, often influencing an individual's confidence and self-esteem. Healthy skin holds cosmetic, aesthetic, and cultural significance. (3,9)

4. The Clinical Importance of Skin

4.1. The Clinical Importance of Skin from a Cosmetic Perspective

4.1.1. Intrinsic and Extrinsic Skin Aging

Skin aging consists of two different biological processes: intrinsic and extrinsic aging. Intrinsic skin aging is genetically determined and occurs as a result of the body's overall aging process. This type of aging is continuous throughout a person's life and progresses with time. Naturally occurring intrinsic aging is closely related to an individual's endocrine system. Extrinsic skin aging, on the other hand, is aging that occurs due to environmental factors. Factors such as alcohol consumption, poor or unhealthy dietary habits, environmental pollution, exposure to various and severe physical and psychological stresses, and excessive UV radiation have been reported to contribute to this type of aging. Among these factors, excessive exposure to UV radiation has been proven to have the most detrimental effect in studies. There are some important differences between intrinsic and extrinsic skin aging. For example, intrinsic skin aging involves thinning of the epidermis and dermis, reductions in the number of cells and fibers such as fibroblasts, Langerhans cells, melanocytes, collagen, and elastin in the skin, dysfunction of skin appendages, and decreased

thermoregulation and sensory function. In extrinsic skin aging, thickening of the epidermis and increases in the number of fibroblasts, mast cells, and neutrophils are observed. If these two different types of aging occur together, the changes observed in the skin and the damage it causes will be more significant. (21)

4.1.2. Collagen Supplements

Collagen is a type of protein found in the skin, tendons, and bones in the body. There are 28 identified types of collagen, with types I and III collagen being the most abundant in the skin, comprising approximately 85-90% and 10-15%, respectively. (22,23) During aging, a decrease in collagen synthesis, elasticity, and fibroblast activity in the skin leads to the formation of wrinkles. Various nutrients and supplements are used to reduce wrinkles, improve skin health, and delay aging. Among the most commonly used supplements today are topical creams, injectable fillers, and orally ingested collagen supplements. Type I collagen is the most commonly used in the cosmetic production of these supplements due to its high similarity to human collagen. (22,23)

In recent years, oral collagen supplements have become increasingly popular as promising anti-aging nutraceuticals. These supplements are typically produced using type I, II, and III collagens obtained from sources such as bovine, porcine, poultry, and marine animals. Type I collagen is primarily derived from marine animals, type II from poultry and bovine collagen, and type III from porcine and bovine collagen. Among these, porcine collagen is reported to cause fewer allergic reactions as it closely resembles human collagen. However, the use of this type of collagen is limited due to religious restrictions. Collagen derived from marine animals is preferred in this field due to lower risk of disease transmission. Despite the increasing use of these supplements, research in this area is still limited. Recent studies have reported that these supplements increase skin hydration and collagen density in the dermis, while reducing collagen breakdown in the dermis. (22,23)

4.1.3. Coenzyme Q10

Ubiquinone, also known as coenzyme Q10 (CoQ10), is a vitamin-like substance synthesized by the body. Found in the epidermis layer, CoQ10 is a powerful antioxidant that prevents oxidative damage in the skin. Playing a crucial role in the repair of oxidative damage and production of new cells, CoQ10 supports the skin's youthful and healthy appearance. CoQ10 helps prevent photoaging caused by exposure to sunlight by fighting free radicals that form after exposure to the sun's rays. It can contribute to the reduction

of wrinkles and fine lines by maintaining the skin's elasticity. The antioxidant property of CoQ10 also helps protect the immune system from oxidative stress. Thus, it enables immune cells to work more effectively, creating a better defense against infections and diseases. Immune cells require high energy to fight pathogens, and CoQ10 supports the energy production processes of these cells, allowing them to work more efficiently. In addition to all these effects, CoQ10 offers some benefits in inflammatory and autoimmune disorders by regulating infection processes. (24,25,26)

Coenzyme Q10 deficiency can occur in two different ways: primary or secondary. Primary CoQ10 deficiency is caused by mutations in genes related to CoQ biosynthesis. Secondary deficiency occurs due to mutations in genes not related to biosynthesis, various complex diseases, and as a side effect of statin therapy used to lower cholesterol levels. Diseases associated with secondary CoQ10 deficiency include fibromyalgia, cardiovascular diseases (chronic heart failure, atherosclerosis, hypertension), neurodegenerative disorders (Parkinson's disease, Alzheimer's disease, Huntington's disease), diabetes mellitus, infertility, cancer, and migraines. (24,25,26) There are several approaches to addressing CoQ10 deficiency, including CoQ10 supplements, dietary changes, and treatment of the underlying disease. CoQ10 supplements are used to provide an additional source of CoQ10, a compound naturally produced by the body but which may decrease with age. These supplements are available in various forms on the market, including oral spray, hard or soft shell gel capsules, and tablets. Consuming more CoQ10-rich foods through a balanced diet can help alleviate deficiency. Foods rich in CoQ10 include meat, oily fish, whole grains, nuts, and liver. (24,25,26)

4.1.4. Vitamins

4.1.4.1. Vitamin C (L-Ascorbic Acid)

There is evidence that Vitamin C plays vital functions in promoting collagen synthesis, exhibiting antioxidant properties, and increasing the proliferation and migration of dermal fibroblasts on the skin. Studies have reported a relationship between Vitamin C deficiency and the loss of many important skin functions. These include slowed wound healing, thickening of the stratum corneum, and increased subcutaneous hemorrhages in those with a Vitamin C deficiency. (24,27)

Vitamin C can be used both orally and topically to benefit the skin. Supplemental Vitamin C is reported to help balance skin tone, reduce sunspots, hyperpigmentation, and other color irregularities. Additionally, according to

information obtained from in vitro studies, as well as animal and human studies, Vitamin C has a protective effect against the harmful UV rays of the sun. Its antioxidant properties are also reported to reduce skin damage following sun exposure and help with the healing of sunburns. (24,27)

4.1.4.2. Vitamin E (Tocopherols)

The Vitamin E complex, referred to as tocopherols, consists of 8 compounds. Tocopherol is a naturally occurring, fat-soluble antioxidant. (24) Vitamin E, although not as strong an antioxidant as Vitamin C, is commonly used for protection against photo damage and for regulating the skin's moisture. (28) Studies have shown that in addition to its antioxidant properties, Vitamin E also possesses anti-inflammatory properties and, when used together with Vitamin C, reduces oxidative damage in the skin. (21,29) Vitamin E has been proven to have neuroprotective effects in clinical trials for various neurological disorders, including traumatic brain injury, Alzheimer's disease, and Parkinson's disease. (29) Additionally, most published studies report that topical applications of Vitamin E reduce erythema, skin damage due to UV radiation, and sunspots. (24)

4.1.4.3. Vitamin A (Carotenoids, Retinol)

Vitamin A has two topical varieties, carotenoids and retinol, both of which are extremely powerful antioxidants. Studies have shown that Vitamin A possesses photoprotective properties and regulates the activity of sebaceous glands. This helps prevent clogging of pores, thereby reducing the formation of acne. This is why Vitamin A-based treatments are often preferred in acne treatment. (21,24)

Retinol is essential for the human body, but the body cannot synthesize it; it must be obtained through diet. Food sources rich in retinol and retinol esters include liver, milk, egg yolk, cheese, and fatty fish. The market offers various retinol products for both topical and pharmaceutical use. Topically applied Vitamin A is reported to exhibit anti-aging properties and affect various processes, including DNA repair. These processes include the stimulation of collagen synthesis and cell proliferation, regulation of keratin composition, reduction of melanocyte activity, and decreased keratinocyte adhesion. (21,24)

4.1.4.4. Vitamin D

Vitamin D not only serves as a barrier function in the skin but also plays a significant role in the formation of hair follicles. Recent studies have reported

that a decrease in the expression of vitamin D receptors is associated with the progression of cutaneous melanoma, indicating that vitamin D receptors act as an effective tumor suppressor. Many studies testing oral vitamin D supplements have reported that they could be effective in preventing, mitigating, or treating skin cancer, which is associated with anti-aging effects. (24,30)

Vitamin D, a secosteroid hormone, plays a crucial role in the regulation of both innate and acquired immune systems. Vitamin D receptors are found in many immune cells, including mast cells, macrophages, T and B lymphocytes, and natural killer cells. Additionally, vitamin D induces the production of cathelicidin, which is necessary for antimicrobial defense, and exhibits immunosuppressive effects through the modulation of Langerhans cells. (24,30) Due to all these important effects in the body, it is recommended that individuals at risk of vitamin D deficiency, such as those with limited exposure to sunlight, obese individuals, those with fat absorption disorders, those with dark skin, the elderly, and those who are light-sensitive, should take higher doses of vitamin D. (24)

4.1.4.5. Pre and Probiotics

Probiotics are defined as live microorganisms that, when consumed in adequate amounts, confer a health benefit on the host. Prebiotics, on the other hand, are non-living food components that confer health benefits on the host where they are present. Clinical studies using pre and probiotics have found that they can contribute to maintaining skin homeostasis by regulating the immune system of the skin, similar to their effects on the gut system. Additionally, some studies report that UV exposure adversely affects immune system functions. Following this, there are studies in the literature indicating that the use of pre and probiotics can be beneficial for cutaneous immunity altered as a result of UV exposure. (24)

4.2. The Clinical Importance of Skin in Terms of Skin Grafts

Skin grafts are pieces of skin that are removed from one part of the body, including the epidermis and dermis, or just the dermis, and transferred to a different part of the body. Skin grafts are divided into two types: full-thickness grafts, if only the dermis is taken, and split-thickness grafts, if taken with the epidermis. Factors to consider when choosing the type of graft include the reason for use (repair or cosmetic improvement), the depth of the defect to be covered, the suitability of the donor area, and the recipient area's ability to accept the graft. (31,32)

There are 4 different classifications of skin grafts based on the individual from whom they are taken or to whom they are applied. If the graft is made between different areas of the same person, it is called an autograft. If it is made between genetically different individuals of the same species, it is called an allograft (homograft). If there is a transfer between individuals of different species, it is called a heterograft. If there is a transfer between genetically identical beings, like identical twins, it is called an isograft. (31,32)

The use of skin grafts in burn treatment is extremely valuable. Allografts, in particular, are commonly preferred in the treatment of burns. These types of grafts serve as a biological dressing. Especially in extensive burn cases where autografts are not available, they provide temporary coverage of the wound area. Additionally, they prepare the wound area for autografting. (33)

4.3. Skin Diseases

Skin diseases refer to various conditions that occur on the skin, mucous membranes of certain organs such as the mouth, nose, eyes, and on skin appendages such as hair and nails. Psoriasis, atopic dermatitis, lichen planus, acne, rosacea, urticaria, dermatophytes (fungal infections) are a few examples of skin diseases.

4.3.1. Psoriasis

Psoriasis is a chronic, inflammatory, and the most commonly encountered skin disease in clinical practice. It is also defined as the shortening of the keratinocyte cycle, typically taking around 28 days from the stratum basale to the stratum corneum. In psoriasis, this cycle is completed within approximately three to five days. Various factors that may trigger or exacerbate the disease have been reported, but the exact cause of the disease has not yet been determined. (3,34) Among the symptoms of the disease, large, flat, palpable lesions in the form of plaques are observed on the elbows, knees, and scalp. It is reported that scaling on the scalp is present in 50% of patients with psoriasis. While in most patients, it is observed to be very thick around the hairline, in some patients, it covers the entire scalp. Another common finding associated with the disease is problems with skin appendages such as pitting of the nails and detachment of the nail from the nail bed. (3,34,35)

Psoriasis has three different types: guttate, pustular, and erythrodermic. In guttate psoriasis, plaques occurring on the body are seen in the form of drops, and a history of streptococcal throat infection is usually found when taking the patient's history. Pustular psoriasis is characterized by pustules that are typically

present on the hands and feet and are more sterile. Erythrodermic psoriasis is a generalized inflammatory form of psoriasis that includes various body areas such as the face, hands, feet, nails, trunk, and extremities, and requires urgent dermatological intervention. If erythrodermic psoriasis affects a large surface area of the skin and interferes with the skin's two important functions of thermoregulation and preventing the entry of harmful microorganisms into the body, it can be life-threatening. (3,34,35)

4.3.2. Atopic Dermatitis (Eczema)

Atopic dermatitis, also known as eczema, is a highly itchy, chronic, inflammatory skin disease. It is reported to affect up to 20% of children worldwide, and these effects can persist into adulthood. Most clinical studies have reported that atopic dermatitis is often associated with high serum IgE levels, conjunctivitis, asthma, and allergic rhinitis. This condition typically follows a course of flare-ups and remissions. Atopic dermatitis is often associated with a genetic predisposition and arises as a result of an abnormal response of the immune system in the skin. (36,37)

Atopic dermatitis is a disease characterized by continuous dryness and itching. In these patients, the skin loses moisture, resulting in a dry, swollen, and cracked appearance. Itching and scratching follow each other in a cycle, resulting in rashes. It has been reported that red and lichenified (thickened) patches develop in areas such as the eyelids, neck, elbows, wrists, and knees as a result of continuous itching. In these irritated areas, in addition to infections associated with continuous itching, some diseases such as warts and contagious skin diseases may develop. (36,37,38)

The first step in the treatment of atopic dermatitis is to address the patient's dryness and itching problems. Patients should be advised not to take excessive showers as this can further dry out the skin. Changes in diet in children are reported to have an impact on the disease. Therefore, it is recommended to eliminate dairy, eggs, fish, nuts, chocolate, and spicy foods from the diet. Topical corticosteroids are prescribed to reduce acute flare-ups, oral antihistamines to reduce itching, and antibiotics by physicians if a secondary infection due to atopic dermatitis has developed. (37,38)

4.3.3. Urticaria (Hives)

Urticaria, characterized by usually itchy, well-defined red welts and temporary fluid accumulation beneath these welts, is a skin reaction. Urticaria is divided into two types based on certain clinical features defined in clinical

practice: acute and chronic. Acute urticaria is a type of urticaria that lasts for less than six weeks, during which angioedema may occur in some cases. If urticaria attacks persist for more than six weeks, this condition is referred to as chronic urticaria. There is usually a triggering factor for acute urticaria, which can be easily identified. Chronic urticaria, on the other hand, presents with a more complex clinical picture, making its treatment more difficult and prolonged. (39,40,41)

There are various conditions that can cause urticaria. Among the most common causes are allergic reactions, stress, medications, infections, food pseudoallergens, insect stings, and changes in temperature. Changes in conditions such as temperature, hot-cold, and pressure among these causes are indicators of urticaria progressing towards chronicity. Agents causing infections include viruses causing hepatitis A, B, and C, herpes simplex, bacteria causing urinary tract infections, Helicobacter pylori, and other bacteria. (39,40,41)

The first step in the treatment of urticaria is to identify the condition that exacerbates the symptoms and prevent its recurrence. During this process, doctors may prescribe some antihistamine medications to alleviate various symptoms. If urticaria is chronic, it is also recommended to use medications such as H2 receptor blockers, anti-inflammatory drugs, and immunosuppressants in addition to these drugs. (39,40)

5. Conclusion

Skin is structurally active throughout life and undergoes many changes with aging. Understanding how the structure of the skin develops to allow the body's dynamic form and function, and how injury, disease, or aging leads to anatomical changes and alters the physical properties of the skin, is crucial for finding therapeutic solutions to these changes. From a surgical perspective, emphasizing the less understood aspects of skin microanatomy is essential for offering alternative perspectives on skin pathologies and skin aging.

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CHAPTER X

ANATOMY AND CLINICAL IMPORTANCE OF THE LUMBAR REGION

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1. Introduction

The skeletal system is divided into two parts: axial and appendicular. The axial skeleton consists of the bones of the head, vertebral column, costae and sternum; the appendicular skeleton consists of the bones of the lower and upper extremities. Vertebral column forms an important part of the axial system. (1)

The vertebral column is mechanical. The vertebrae are controlled by a complex system of muscles, joints and ligaments. This complex mechanism also creates a strong control system. This is due to the protection of the medulla spinalis, which is located in the middle of the vertebral column. (2)

The vertebral column has three main functions. Support of the trunk, movement of the trunk and protection of the medulla spinalis and spinal nerves. (3)

The lumbar region has a kinematic role in carrying the weight of the head and trunk, providing support and directing movement. Due to its role, it is an area vulnerable to trauma and is at high risk of disability and injury. At the same time, low back pain ranks first among the musculoskeletal problems seen in the society and approximately 65% of adult individuals experience low back pain at some point in their lives. The most common age range for low back pain is 35-64 years, but the prevalence also varies between countries. (4)

2. Anatomy of the Columna Vertebralis

The vertebral column is a bony column, approximately 70-75 cm long, formed by the overlapping articulation of 32-34 vertebrae, with anatomical and alignment differences depending on the region. The intervertebral disc between the vertebrae makes up about 25% of this column.

The vertebral column allows movement of the head and trunk, as well as supporting and bearing the weight of many organs in the thoracic and abdominal cavity. It allows movement of the costae during inhalation and exhalation. It also provides a protected space for the medulla spinalis inside the vertebral canal. The first 24 of the 33 vertebrae that make up the vertebral column are connected to each other by joints containing discs. These are called presacral vertebrae. These 24 vertebrae are named according to their location. There are seven cervical (C1-7), 12 thoracic (T1-12) and 5 lumbar (L1-5) vertebrae. Of the remaining vertebrae, 5 form the os sacrum (S1-5) and 4 form the os coccygis (Co1-4). These are called fixed vertebrae. (5,6)

2.1. Anatomy of a Typical Vertebra

A typical vertebra consists of two parts. The anterior part, called the bodyvertebrae, is surrounded by thick, strong and compact bone. The posterior part, made up of thinner bony structures, is called the arcus vertebrae. The bodyvertebrae articulate with the neighboring vertebrae with intervertebral discs between them, forming a solid column of bone. The edges of the vertebral bodies are made of compact bone and the rest of the vertebral bodies are made of spongiose bone. This allows the discs to attach to the vertebrae more easily. Between the bodyand arcus of each vertebrae there is a hole called foramen vertebrale. These holes form a canal when the vertebrae are stacked on top of each other and form a column of bone. This canal is the vertebral canal, which houses the medulla spinalis. On the back of the vertebral bodies facing the vertebral canal, there is a hole called basivertebral foramen through which the basivertebral veins passes. (5,6)

2.2. Regional Characteristics and Differences of Vertebrae

Cervical Vertebrae

There are seven cervical vertebrae and the 1st, 2nd and 7th cervical vertebrae are different from the other cervical vertebrae. The most important feature that distinguishes the cervical vertebrae are the holes on thetransvers process, called for. transversarium, through which the vertebral artery and vein

and vertebral plexus pass. The first cervical vertebra (C1) does not have a spinous process, the spinous process of the other 5 are bifurcated except for the 7th cervical vertebra (C7). On thetransvers process of the cervical vertebrae there are projections called tuberculum anterius corresponding to the caput costae and tuberculum posterius corresponding to the tuberculum costae. On the 6th cervical vertebra, just anterior to the tuberculum anterius, there is a protrusion called the tuberculum caroticum or Chassaignac's tubercle. At the site of this prominence, the pulse is taken from the common carotid artery.

The atlas (C1) is one of the atypical vertebrae that do not conform to the typical characteristics of vertebrae. The most important feature is the absence of the body and spinous process. Transvers process is quite short. The upper articular surface superior articular process articulates with the occipitale bone, not with the vertebrae.

Axis (C2), like the atlas, is an atypical vertebra. It has a section called the dens axis that protrudes upwards from its body. This structure, which is seen as a second body, is the most important difference of the axis. Spinous process is short and bifurcated.

The vertebra prominens (C7) is more similar to the thoracic vertebrae below it than to the cervical vertebrae. The spinous process is long and not bifurcated like the thoracic vertebrae, but it has a for. transversarium, the main feature of the cervical vertebrae. It is the first and only cervical vertebra whose spinous process can be felt by palpation from above to below in the midline.

Thoracic Vertebrae

There are twelve thoracic vertebrae. Their bodies are larger than the cervical vertebrae and the bodies become larger as you descend from the 1st thoracic vertebra to the 12th thoracic vertebra. The most important feature that distinguishes the vertebrae of this region from the other regions is the presence of small articular faces that articulate with the costa on the lateral surface of the body and just in front of the pedicles. Except for the last two thoracic vertebrae, all thoracic vertebrae articulate with the anterior faces of their transvers process and the rib tubercle. These articular faces are called transverse costal facet.

Lumbar Vertebrae

The lumbar region, consisting of five mobile vertebrae and intervertebral discs, makes up 25% of the length of the entire vertebral column. The lumbar vertebrae are larger than all other mobile vertebrae. They differ from the cervical vertebrae by the absence of the transverse foramen and from the thoracic

vertebrae by the absence of the costal facet. These bones, which are very thick due to the excessive force they carry, have two characteristic projections. The protrusion at the posterior part of the superior articular processis called mamillar process and the protrusion at the posterior part of the base of the transvers process is called accessory process.

The bodyvertebrae of the fifth lumbar vertebra is the largest of the vertebral column. Its anterior part is longer than its posterior part, resulting in an increase in the lower lumbar lordosis. It has the smallest and rounder spinous process of the lumbar vertebrae. Itstransvers process is wider in the antero-posterior and supero-inferior directions than the other lumbar vertebrae.

Sacrum

It is a large, triangular bone formed by the fusion of five vertebrae. The fusion points of these vertebrae are clearly visible when viewed from the front; these fusion points are called transverse linea. It forms a large part of the back wall of the pelvis. It articulates with the last lumbar vertebra above, os coccygis below and bones of the coxae on the sides. The surfaces where the os ilium and sacrum articulate are called facies auricularis in both bones. The linea arcuata continues as a faint bony edge on the sacrum up to the midline with the same name. The midline anteriormost point of the articulatio lumbosacralis with the last lumbar vertebra is called the promontorium. The anterior aspect of the sacrum that joins the pelvic wall is concave and called facies pelvica, while the convex posterior aspect is called facies dorsalis. The first three segments of the sacrum, which play an important role especially in load transmission in the pelvis, carry the main load, while the last two segments do not participate in this transmission and are therefore less developed. When the last two segments are viewed from behind, you can see that the bone is thinner and even has an opening, called the hiatus sacralis. In order for the anterior and posterior branches of the spinal nerves to pass through, there are four anterior sacral foramen holes on the anterior face of the bone and four posterior sacral foramen holes on the posterior face. (5,6)

2.3. Intervertebral Disc

They are hydroelastic and semi-flexible joint-type auxiliary formations located between two adjacent bodyvertebrae. The main function of the İntervertebral discs, which make up 1/3 of the length of the vertebral column, is to carry and distribute the load on the vertebral column and allow the muscles to move.

The height of the intervertebral discs varies according to their location and the differences within the disc. The posterior part of the cervical and lumbar intervertebral discs is thinner than the anterior part. This contributes to the formation of cervical and lumbar lordosis. The ratio of disc height to vertebral height is 1/5 in the thoracic region, 1/3 in the lumbar region and 3/5 in the cervical region. This feature is the reason for the high range of motion in the cervical and lumbar regions. The intervertebral disc consists of three main parts: endplate, annulus fibrosus and nucleus pulposus.

The anterior part of the annulus fibrosus is thicker and more prominent, while the posterior part is thinner and well embedded in the vertebral body. It is mainly collagenous and contains a high amount of water (65-70%). The annulus fibrosus, which provides the shape and integrity of the disc, consists of 15-20 anterior and 7-10 posterior multilayered fibrocartilage lamellae located obliquely around the nucleus pulposus. The annulus fibrosus, which carries 75% of the load on the disc, is much more flexible than tendons and ligaments due to the arrangement of the fibers that form it and the high amount of proteoglycan.

The annulus fibrosus is closely connected to the anterior longitudinal ligamen tand posterior by durable sharpey fibers that keep it firmly attached to the outermost vertebral cortex.

The nucleus pulposus is located 1/3 posterior to the intervertebral disc of the annulus fibrosus. It occupies 40-50% of the disc area. Thanks to its compression resistance and shock absorption properties, it transforms forces and spreads them evenly throughout the annulus fibrosus. (5-7)

2.4. Joints of the Lumbar Region

There are two types of joints between the vertebrae that make up the lumbar region. The lumbar region is also connected to the sacrum by a joint between them. (5,6)

2.4.1. Symphysis Intervertebralis

They are symphysis-type semi-floating joints formed by İntervertebral discs between the bodyof neighboring vertebrae. There are a total of 17 of these joints between all bodyvertebrae from the second cervical vertebra to the first sacral vertebra. In relation to these joints, there are two important ligaments that run anterior and posterior to the bodyvertebrae. These ligaments, called ligamentum longitudinale anterius and ligamentum longitudinale posterius, limit the movements of the vertebral column.

Anterior Longitudinal Ligament

It is a strong and broad ligament that runs along the anterior surface of the bodyvertebrae. Starting from the anterior aspect of the os sacrum below, this ligament becomes thinner as it ascends. It is tightly attached to the İntervertebral discs and the edges of the bodyvertebrae and loosely attached to the central parts of the trunk. It has a limiting effect in extension movement.

Posterior Longitudinal Ligament

While the bodyis firmly attached to the vertebrae, the support it gives to the disc is less because it is attached to the posterior surfaces of the İntervertebral discs by merging with the annulus fibrosus fibers and opening to both sides. This situation constitutes one of the most important causes of disc herniations. The width of the Posterior Longitudinal Ligament (PLL) decreases from the L1 level and decreases to half of its original width at the L5-S1 disc level. Weakness due to the opening posterolateral to the PLL is the reason why more disc herniations occur in this region. The function of the PLL is to prevent excessive flexion. (5,6)

2.4.2. Zygapophyseal Joints (Facet Joints)

They are plana joint type fully mobile joints formed between the superior articular process of a lower vertebra and the inferior articular process of an upper vertebra. There are 24 pairs of facet joints in the vertebral column. The articular faces forming this joint differ at each level. The ligaments between the arcus vertebrae, spinous process , transverse process are important for the stabilization of the vertebral column.

The upper surface of the joint is convex and faces lateral, inferior and anterior, while the lower surface is concave and faces medial, inferior and posterior. The capsula articularis is attached to the edges of the adjacent proc. articularis of the joint. The medial side of the capsula articularis consists of ligamenta flava fibers and the lateral side of fibrous tissue. The joint capsule has two recesses, superior and inferior.

In the lumbar region, the facet joints provide flexion and extension movement due to their location in the sagittal plane, and allow slight lateral flexion and rotation in an amount that minimizes the torsional forces on the discs.

It has two movements, translation and angulation distraction. In lateral flexion, translational movement occurs on one side and in forward flexion on both sides. During rotation, angulation occurs in the direction of movement

and compression occurs on the other side. The facet joint carries approximately 16% of the axial load. This load reaches its maximum level when in extension. Rotational movements with flexion movement cause serious risks on the discs. The facet joint is innervated by the medial branches of the r. dorsalis at its own level and the level above it.

Supraspinal Ligament

It adheres to the spinous process posteriorly and continues with the crossing fibers of the erector spinae muscles tendons, terminating at the L4 spinous process. The supraspinal ligament, which also functions against the shearing force on the lumbar vertebrae, is stretched in flexion and prevents excessive flexion.

Ligamenta Flava

It is the strongest ligament in the lumbar region, connecting two adjacent vertebrae.

It covers the posterior aspect of the vertebral canal and extends from the upper edge of the lamina of the lower vertebra to the inner edge of the lamina of the upper vertebra. It increases in thickness from cervical to caudal and is the most fiber-containing structure of the human body with an elastic fiber content of 80%. It supports the facet joints from below with its broadly fanning structure towards the sides and forms the joint capsule in the anterior part. The ligamenta flava, which has an important role in providing antero-posterior stabilization of the body, has a constant tension. It has an important role in maintaining normal posture thanks to its elastic structure that lengthens in flexion and shortens in extension. It also has the function of preventing lumbar hyperflexion.

Intervertebral Foramen

It is also called a canal or neural foramen. The anterior wall is formed by the discus intervertebralis intervertebral disc and the corpuscles of two adjacent vertebrae, the floor and ceiling by the pedicles, and the posterior wall by the facet joint and ligamenta flava. They are the foramen through which the n. spinalis leaves the vertebral canal and exits.

Interspinal Ligament

It is a membranous ligament lying between the two spinous process processes. It divides the deep muscle groups into two. Although it is the weakest of the spinal ligaments, its strength in the lumbar region is high. Its function is

to create mild resistance during flexion movement and to prevent the shearing force that will occur forward.

Intertransvers Ligament

It lies between the processes oftransvers process. It is membranous in the lumbar region and rounded cord-shaped in the dorsal region, forming the origin of the multifidius muscle. It has the function of preventing excessive lateral flexion.

Capsular Ligament

Capsular ligament consists of fibers located perpendicular to the edges and surfaces of the facet joint. Its length is shorter and tighter in the thoracic and lumbar regions. Its function is to allow sliding of the facet joints during all vertebral movements. Vertebropelvic ligaments consist of iliolumbar ligament, sacroiliac ligament, sacrotuberal ligament and sacrospinal ligament between the lumbosacral vertebrae and pelvis. It extends from the transvers process of L4 and L5 to the posteromedial edge of the iliac crest. It is the main structure that stabilizes the sacrum to L5, preventing L4 and L5 from sliding forward. (5,6)

2.4.3. Articulatio Lumbosacralis

Between the body of the fifth lumbar vertebra and the sacrum, a symphysis-type joint is formed with the L5 disc, while posteriorly they are connected as facet joints between L5 and S1. Since the sacrum is inclined forward, the vertebral bodies of S1 and L5 articulate at an angle called the lumbosacral angle, which is approximately 140°. Ligaments of the other vertebrae are also present at this joint. In addition, the iliolumbal ligament provides support to the joint.

Iliolumbal Ligament

Starting from thetransvers process of the 5th lumbar vertebra, it expands inferiorly and laterally and attaches to the pelvis as two bands. Its upper fibers attach to the iliac crest and continue with the fascia thoracolumbalis. The lower fibers attach to the ala ossis sacri as ligamentumlumbosacrale. (5,6)

2.5. Muscles of the Lumbar Region

In this region, there are muscles that attach directly to the vertebrae and directly affect the vertebral column, as well as muscles that do not attach to the vertebrae but affect the vertebral column when they contract. These are the muscles that participate in many movements to maintain a continuous upright

posture. The muscles are covered by the fascia thoracolumbaris and attach to the costae above, the sacrum below, the fascia of the latissimus dorsi muscle and transversus abdominis muscle on the sides and the spinous process in the middle. The muscles of the lumbosacral region are arranged in superficial and deep layers. The superficial layer begins with the fascia thoracolumbalis, the medial extension of the latissimus dorsi muscle. This structure surrounds the deeper lumbar spinal back muscles starting from the spinous process of the lumbar vertebrae.

The superficial layer of the deep back muscles related to the lumbosacral region is the erector spinae muscles. This muscle consists of iliocostalis, longissimus and spinalis muscles from lateral to medial. The iliocostalis muscles starts from the posterior aspect of the iliac crest and os sacrum and attaches to the 7th-12th costae. The longissimus muscles starts from the transvers process of the lower thoracic and all lumbar vertebrae and attaches to thetransvers process of all thoracic vertebrae. Spinalis muscle is located most medially and starts from the spinous process of the first two lumbar and last two thoracic vertebrae of this group and attaches to the spinous process of the 1st-8th thoracic vertebrae.

Multifidus muscle, rotatores and intertransversarii muscles form the deep layer of deep back muscles related to the lumbosacral region. The multifidus muscle originate from the posterior aspect of the os sacrum and the proc. mamillaris of the lumbar vertebrae and attach to the spinous process, generally 2-4 vertebrae above the vertebrae from which they originate. The rotatores muscles is between the transvers process and the spinous process of the next vertebra. The intertransversarii muscle is located between the transvers process of the lumbar vertebrae.

The muscles of the lumbosacral region are divided into four groups according to their functions: extensor, flexor, lateral flexor and rotator muscles. Especially extensor and rotator muscles support the spine. Extensor muscles; the multilayered erector spinae muscle is located just below the thoracolumbar fascia. They attach firmly to the iliac crest, sacrum, spinous process of the lumbar vertebrae and supraspinal ligament. They form 3 columns with iliocostal muscle at the outermost, longissimus muscle in the middle and spinal muscle at the innermost. The functions of these muscles are extension and lateral flexion of the lumbar region. Below the erector spina muscle is the transvers spinous muscle, which consists of three muscles. These muscles are the semispinalis, multifidus muscle and rotatores muscles, which perform extension and rotation to the opposite side of the lumbar region. In addition, the much smaller interspinal and intertransversales muscles perform extension and lateral flexion movements in this region by working segmentally.The flexor muscles consist of

rectus abdominis, external abdominal oblique and internal abdominal oblique, transversus abdominis and iliopsoas muscles. The rectus abdominis muscle stabilizes the hip and provides trunk flexion. External abdominal oblique muscle provides abdominal pressure and flexion and lateral flexion of the trunk. Internal abdominal oblique muscles provides trunk flexion when contracted bilaterally and rotation and lateral flexion when contracted ipsilaterally. Transversus abdominis provides abdominal pressure and trunk rotation. The iliopsoas consists of two parts, the psoas major and the iliacus, and acts as the strongest flexor of the thigh. It flexes the trunk with bilateral contraction and provides lateral flexion with ipsilateral contraction.

The quadratus lumborum, internal abdominal oblique and external abdominal oblique muscles are also responsible for lateral flexion. For the lumbar region, external abdominal oblique, multifudus and rotatores muscles rotate to the opposite side, while internal abdominal oblique, thoracic intercostal and iliocostalis lumborum muscles rotate to the same side. (5,6)

2.6. Vascularization of the Lumbar Region

The blood supply of the lumbar region is provided by the lumbal arteryand venous drainage by the lumbar vein. The lumbar artery is one of the parietal branches of the abdominal aorta. They are 4 pairs of arteries that branch from the dorsal aspect of the aorta at the level of the upper edges of the first 4 lumbar vertebrae and extend outward.

The lumbal artery has branches, the dorsal and spinal branches. The dorsal branch divides between the transvers process and extends posteriorly, supplying the muscles, joints and skin of the back. The spinal branch enters the vertebral canal through the intervertebral foramen.

The spinal branch, which branches from the first lumbal artery, supplies the conus medullaris, the others supply the cauda equina, the membranes of the medulla spinalis and the vertebrae. The lumbal veinare parietal branches draining into the inferior vena cava.

There are 4 lumbal vein on each side. The posterior branches of the lumbal vein drain the muscles and skin of the lumbar region, while the anterior branches drain the anterior abdominal wall. The main veins of the bodyvertebrae are the basivertebral veins. These are a series of long veins running horizontally towards the center of the bodyvertebrae.

While the nutrition of intervertebral discs is realized by vascular structures in the cartilaginous plaques until adulthood, it is realized by diffusion after 20-30 years of age due to atrophy of these structures. (5,6)

2.7. Innervation of the Lumbar Region

The posterior branches of the lumbar spinal nerves are divided into two branches, r. medialis and r. lateralis.

The r. medialis extends to the articular processes of the vertebrae and terminates in the multifidus muscle. Lateral branch innervates the sacrospinalis muscle. The cutaneous branches of the first three nerves are distributed in the gluteal region up to the great trochanter as superior cluneal nerves. The lumbosacral plexus is formed by the union of the anterior branches of the lumbar, sacral and coccygeal spinal nerves forming the lumbar plexus, sacral plexus and coccygeal plexus.

The lumbar plexus and sacral plexus innervate the lower extremities. The sacral plexus also innervates the perineum via the pudendal plexus and the coccygeal region via the coccygeal plexus.

Lumbar plexus: It consists of all the anterior branches of the first three spinal nerves (L1-3), most of the L4 spinal nerve and a small portion of the T12 spinal nerve. The lumbar plexus is located on the posterior abdominal wall, anterior to the transverse processes of the lumbar vertebrae and within or deep to the psoas major muscle. (5,6)

Branches of the lumbar plexus:
Iliohypogastric nerve (T12-L1)
Ilioinguinal nerve (L1)
Genitofemoral nerve (L1,2)
Lateral femoral cutaneous nerve (L2,3)
Obturator nerve (L2,3,4)
Accessory obturator nerve (L3,4)
Femoral nerve (L2,3,4)

3. Clinical Importance of the Lumbar Region

The lumbar region has a structure in which musculoskeletal system problems are common due to its anatomical structure, as well as being a transition point between the upper and lower parts of the trunk and being important in terms of kinesiology. Pain secondary to disorders occurring in this region is the biggest cause of labor loss after upper respiratory tract disorders all over the world. In addition, it is also the most expensive disease due to treatment costs and the costs incurred due to time away from work, reduced productivity and disability payments. The lumbar region is also critical in the diagnosis of many diseases. (8)

3.1. Lumbar Puncture

Lumbar puncture is a procedure performed to obtain information about cerebrospinal fluid. Using a special needle called a spinal needle or puncture needle, under local anesthesia and sterile conditions, the cavity is entered at the level of L3-4 in adults and L4-5 in children. In any age group, subarachnoid hemorrhage, hydrocephalus, pseudotumor cerebri and many other diagnoses are supported or excluded by lumbar puncture. Lumbar puncture can also be used to detect the presence of malignant cells in the cerebrospinal fluid for intrathecal treatment, especially during spinal anesthesia or chemotherapy, in the presence of carcinomatous meningitis or medulloblastoma. (9,10)

3.2. Lumbal Strain

Lumbar strain, also known as lumbar strain due to mechanical stress, accounts for 60-70% of mechanical low back pain. The etiology is often traumatic episode or ongoing mechanical stress. The pain is felt in a small localized area in the lower back and does not radiate to the lower extremities. Flexion and extension are painful and limited due to reflex contraction of the surrounding muscles. It is classified as mild, moderate and severe according to physical findings. Patients with subjective complaints of pain but no objective findings are considered to have mild strain and recover within a week. Patients with paravertebral muscle spasm accompanied by limitation of movement have moderate strain and recovery time is up to two weeks. Patients' lateral or bent posture and difficulty in ambulation are severe strains and require a recovery period of three weeks. Radiographic evaluation and laboratory tests are normal in these patients. (11,12)

3.3. Lumbar Disc Herniation

Acute, chronic and recurrent low back pain is commonly caused by lumbar disc problems. Disc ruptures usually occur in the third and fourth decade of life when the nucleus pulposus has a gelatinous consistency. Herniation usually occurs in the morning with increased pressure on the disc and in the posterolateral part of the PLL, the weakest part of the disc. It usually occurs as a result of flexion injuries. Since the most trauma during flexion and extension in the lumbar region occurs at the L4-5 and L5-S1 levels, disc herniation occurs at these levels in over 90% of cases.

The resulting disc herniation may compress a single spinal nerve, or it may compress multiple roots or even the cauda equina. Subsequent local inflammatory changes can increase the pressure around the nerve root and

increase low back pain in situations where intraspinal pressure increases, such as coughing, sneezing and laughing. Macnab's classification of disc herniation is based on bulging, protrusion, extrusion and sequestration of the annulus fibrosus consistent with MRI findings. In bulging disc, the disc bulges within normal borders and the annulus fibrosus is normal. It is asymptomatic unless spinal stenosis occurs and neural tissues are damaged. In a protruding disc, the disc material is displaced posteriorly within a ruptured and weakened annulus fibrosus, the PLL is intact. When the disc material completely tears the annulus fibrosus, it is displaced and the PLL ruptures. This is defined as an extruded disc. When a sequestered disc occurs, the disc material can completely separate from the disc as a free fragment and slide up, down or laterally in the for. intervertebral space. Clinically, patients mostly complain of localized pain. The pain is usually of sudden onset, localized to the lower back or radiating to the leg along the nerve line, increasing with prolonged sitting, standing, coughing, straining, driving and bending forward. It may also be described as paresthesia such as pins and needles, numbness, coldness, and pain. In cases of L5 or S1 radiculopathy, the pain is described as sciatica pain following the sciatic nerve trace, often radiating to the gluteal region, back of the thigh and lateral malleolus. In L3 or L4 radiculopathies, pain is felt on the anterior aspect of the thigh. In extruded disc conditions, low back pain decreases while radicular symptoms become more prominent. Midline disc protrusions may cause low back pain without causing significant radiculopathy. Large midline herniations may cause bilateral radiculopathy or cauda equina syndrome, which occurs in only 1% of disc herniations. Patients with disc herniation have normal routine biochemistry test results and electromyography findings if the patient has root involvement. While most patients benefit from conservative treatment, cauda equina syndrome and severe or progressive neurologic deficits may be indications for surgery in patients who do not respond to treatment. (13-15)

3.4. Lumbal Spondylosis

Intervertebral disc degeneration is a clinical condition characterized by degenerative changes such as osteophytosis of the vertebral corpus, hypertrophy of the facet joint processes and laminae, loss of ligamentous flexibility and sometimes segmental instability. It is also called "intervertebral osteochondrosis", "spondylosis deformans" and "osteoarthritis".

Trauma or degenerative disease that causes disruption of the facet joint affects the disc, while lesions that cause disruption of the disc eventually affect the facet joints. Pathologic changes in the three joint complexes in a segment can

also affect the upper and lower levels, leading to the development of multilevel spondylosis. Rotational strains and compressive forces cause the development of lumbar spondylosis by two different mechanisms. Rotational strains mostly affect the L4-L5 segment as the L5-S1 segment is protected by ligaments and bony structures.

While rotational stress causes changes in both facet joints and intervertebral discs, the L5-S1 segment is most frequently affected by compressive forces and the first changes occur in the disc.

Lumbar spondylosis is a disease of wear and tear and aging. The incidence increases with age and after the age of 60 years, normal healthy spine is rarely found. After mild to moderate degenerative changes, the spinal motion segment becomes unstable and symptoms begin to appear. With progressive degeneration, the segment stabilizes again and the decrease in motion reduces the symptoms. Lumbar spondylosis presents with different clinical signs and symptoms depending on the affected structures and period. Symptoms may sometimes be related to the disc and sometimes to the facet joint. Sometimes symptoms are associated with both. (16,17)

3.5. Facet Syndrome

It is a mechanical instability syndrome with degenerative changes in the facet joints. 15-40% of chronic low back pain is caused by facet joints. Patients complain of pain radiating to the gluteal and thigh regions and tenderness with pressure on the facet joints. There is paravertebral muscle tension in the painful segment and pain and restriction of movement in hyperextension and rotation positions. There are no neurologic examination findings. (18)

3.6. Lumbal Spinal Stenosis

It develops as a result of compression of neural elements in the spinal canal, nerve root canal or for. It develops when the neural elements are compressed as a result of narrowing in the intervertebral space. It is divided into central and lateral according to its anatomical position. Central stenosis occurs as a result of narrowing in the sagittal and/or coronal diameter of the spinal canal. The canal diameter may narrow due to facet joint hypertrophy, ligamenta flavathickening, intervertebral disc bulging or spondylolisthesis. Lateral canal stenosis occurs when the lateral canal narrows due to facet joint hypertrophy, decreased disc height, posterolateral disc bulging or spondylolisthesis. It is a clinical picture due to degenerative changes with symptoms that increase and decrease depending on the load and duration of the load affected by posture, usually seen over the

age of 50. Unilateral or bilateral leg pain may radiate from the thigh to the calf or foot. In the forward bending position, the laminae of adjacent vertebrae are separated by trunk flexion. The thickness of the ligamenta flavadecreases and the anteroposterior diameter of the canal increases, causing the patient to relax. An increase in the patient's complaints is observed in the extension position and during loading. During the examination, flexion is painless and open, while in extension there is restriction of movement and pain. In lateral canal stenosis, patients usually complain of single leg pain. Leg pain is predominant without low back pain. (19,20)

3.7. Lumbar Myofascial Pain Syndrome

It is one of the most common causes of local musculoskeletal pain outside the joints. It is characterized by tender trigger points in the muscle on palpation. These trigger points may be active or latent. Active points are sensitive and can cause pain. Latent ones do not cause pain spontaneously although they are tender. Clinical findings are important in making the diagnosis. They are among the common causes of chronic low back pain. (21)

3.8. Marelgia Parasthetica

The lateral femoral cutaneous nerve, which provides anterolateral sensory innervation of the thigh, is most commonly entrapped under the inguinal ligament and this syndrome is called Mareljia Paresthetica (MP). The most common causes of idiopathic MP are obesity due to anatomical position, pregnancy, tight clothing (jeans, military armor, police uniforms), belts, direct trauma, mechanical factors such as muscle spasm, scoliosis, iliacus hematoma and leg length changes, and metabolic factors such as diabetes, alcoholism and lead poisoning. Total hip arthroplasty, spinal interventions, open and laparoscopic appendectomy, cesarean section with epidural anesthesia, obstetric and gynecologic surgery are the iatrogenic causes of MP. Symptoms include pain, burning, numbness, muscle pain, coldness, throbbing pain or vibration in the lateral and anterolateral thigh region, which increases after prolonged standing and walking and decreases with sitting. (22)

4. Treatment Approaches in Lumbal Region Problems

4.1. Surgical approaches

Surgical approaches in the treatment of lumbar region dysfunctions are categorized into two groups as minimally invasive and invasive procedures. In cases requiring urgent surgery such as progressive loss of motor and sphincter

control, microdiscectomy and decompression surgeries are performed. Minimally invasive methods such as sacral epiduroscopic methods, facet joint denervation, epidural steroid injections, intradiscal selective nerve blockages are preferred in persistent low back and leg pain that does not respond to conservative treatment and does not cause motor loss and sphincter loss. (23)

4.2. Medical Approaches

In low back pain problems, local anesthetic drugs, non-steroidal anti-inflammatory drugs or steroid-containing drugs are used to reduce pain, relieve intraneural edema and reduce the pressure around the herniation. At the same time, injection methods such as platelet rich plasma and prolotherapy increase the blood flow to the applied area, allowing the area to repair itself. (24-26)

4.3. Conservative Approaches

Physical therapy applications are of great importance in lumbar region pathologies except in cases where emergency surgery is not required such as progressive motor loss, loss of sphincter control, bladder bowel dysfunction.

The aim of physical therapy applications in lumbar region dysfunctions is to increase tissue health, segmental mobility and functional movement and to maintain continuity.

Healing is a dynamic and complex process that refers to the reconstruction of anatomical integrity in a systematic repair process characterized by inflammation, regeneration, maturation and remodeling.

Therefore, in order for the patient to show a healing reaction in this dynamic process, they need to be informed about the positive and negative aspects of the picture they are exposed to, which is perhaps the most important step in this process.

Controlling the pain and inflammatory process is the second step in this process. Determining whether the pain is nociceptic, peripheral neurogenic or central sensitization type is important for the treatment protocol. The P.OL.I.C.E protocol is a general and valid method for controlling the inflammatory process (P: Protect I: Ice OL: Optimal Loading C: Compression E: Elevation).

Hot and cold pads are modalities frequently used in the inflammatory process due to their effects on nerve conduction velocity and their constrictor and dilator responses on vascular structures.

Progressive movement imagery and progressive movement presentation, functional restoration and therapeutic exercises are also used to treat pain.

Soft tissue mobilizations, manual therapy approaches integrated with joint mobilizations, joint manipulations, symptom modifications, neurodynamic mobilizations, muscle-energy techniques and taping, targeting corrective, inhibitory and or externator effects, prepare the tissues for exercise and bring the tissues to the optimal healing level by recommending the most appropriate exercises considering the age, gender, occupation and sociocultural status of the person. (27-30)

5. Conclusion

The lower back is the center of the human body. It acts as a carrier in activities such as standing, walking or carrying loads. Due to its center of gravity and lordotic structure, it is more susceptible to trauma and herniations.

Since the nerves emanating from the lumbar region provide sensory and motor innervation of the lower extremities, affecting the neural structures of this region may cause hip, knee and foot problems and limit the patient's mobilization.

Low back pain is the most common problem in the world. In 2020, 619 million people complained of low back pain. Since it can occur at any age and most people suffer from low back pain at least once, it is very important for clinicians to know the anatomical structures of this region and the symptoms that occur in order to diagnose and treat it. (31)

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CHAPTER XI

MENISCUS ANATOMY AND CLINICAL SIGNIFICANCE

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1. Introduction

The meniscus is a crescent-shaped fibrocartilaginous structure that partially divides the joint and generally gives concavity to the joint. The meniscus is found in many joints of the body, especially the knee (1). The meniscus was initially defined as an embryonic remnant (2). Today, it is known that the meniscus structure is of great importance for knee joint function (3). It derives from the Greek word "meniskos" meaning "crescent" (4).

2. Meniscus Embryology

Menisci are formed through the condensation of the middle layer of the mesenchymal tissue surrounding the joint capsule and are formed in layers 8-10. It takes on its unique shape within weeks (5). A large number of vascularization and cell increases are observed in all developing parts of the meniscus (6). In children, lymphatics and blood vessels are located throughout the meniscus until they are one year old. As the child begins to walk, load transfer begins to the meniscus. Around 18 months, the meniscus blood supply decreases to 25-33% of the peripheral area, and the number of cells decreases and the amount of collagen increases. Joint movements and loading determine the circular orientation of collagen fibers. The fibrocartilaginous zone in the center remains avascular. The tibial plateau is covered laterally and medially at different rates through the menisci, following the fetal period and continuing until the adult period; The final rates are 51-74% in the medial and 75-93% in the lateral (7).

3. Meniscus Histology

3.1. Cellular and Biochemical Content

72% of the meniscus consists of water. The content of the remaining 28% generally consists of organic substances consisting of extracellular matrix (ESM) and cells. 17% of these organic substances are glycosaminoglycans, 75% are collagens, 2% are DNA, while less than 1% are adhesion glycoproteins and elastin. These rates may vary depending on injury status, age and other pathologies (8).

The basic fibers of the menisci are composite collagen; It also contains a very small amount (0.6%) of elastin. However, the functional and biochemical function of elastin in the meniscus has still not been elucidated. Collagen content varies depending on the zone of the meniscus. In the central zones, 70% of its dry weight consists of collagen. 40% of the collagen in this zone is Type 1 and 60% is Type 2 collagen. Collagen in the peripheral areas constitutes 80% of the dry weight. While it is largely Type 1 collagen, other types (Types 2, 3, 4, 6 and 18) are found in less than 1% (9).

One of the main components of ESM is proteoglycans. These components also have different distributions zoneally. The peripheral 1/3 contains less proteoglycan than the central 2/3. The main function of these components is to provide water in the structure of the meniscus (8). With load, fluid passes into the joint space, and when the load is removed, it passes back into the meniscus. This arrangement provides both joint lubrication and nutrition of fibrochondrocytes (10).

One of the important ESM proteins is adhesion glycoproteins. These provide the connection between other ESM proteins and cells. These are adhesion glycoproteins and are located in the menisci; Type 6 is thrombospondin and fibronectin (11).

The cells within the menisci are not clearly classified. The structure of the white area located in the center contains round cells or fibrochondrocytes that resemble chondrocytes. The structure of the red zone located in the periphery contains spindle cells defined as fibroblasts (12). A third cell type has been identified in the superficial zone. The possibility that these cells, which do not have cellular extensions and have a flat spindle structure, are special progenitor cells is being evaluated (13).

4. Meniscus Anatomy

Menisci are fibrocartilaginous, triangular-sectioned, crescent-shaped structures located on the medial and lateral sides of the knee. It enables the

establishment of a joint between the flat plateau surface of the tibia and the concave joint surface of the femur. It covers approximately 2/3 of the tibial plateau (14). Menisci are connected to the tibia by meniscotibial (coronary) ligaments. Meniscotibial ligaments consist of capsular fibers; It adheres to the rib condyle distally and to the outer edges of the menisci proximally. Meniscotibial ligaments attach a few mm below the articular surfaces of the tibial condyles and form the synovial space there (15). The roots of the meniscus are ligamentous formations that fix the meniscus to the subchondral bone. It transmits tensile and shear forces from soft tissues to bone (16).

4.1. Medial Meniscus

The C-shaped medial meniscus covers an average of 60% of the joint contact area of the medial tibial plateau (6). The front horn is narrower than the rear horn; It grows gradually from front to back and its anteroposterior diameter is larger than its lateral medial diameter. The meniscus, which adheres to the joint capsule in the periphery, also adheres to the tibia with meniscotibial ligaments (17). The medial meniscus is examined anatomically in five zones according to some of its features; anterior root zone (Zone 1), anteromedial zone (Zone 2, 2a, 2b), medial zone (Zone 3), posterior root zone (Zone 5) (18).

4.1.1. Zone 1- Anterior Root

The medial meniscus is divided into four different types depending on where it attaches to the tibia in the anterior. The most common type is the first type, which is the flat intercondylar zone on the tibial plateau, the second type is the area closer to the more medial tibial joint surface, and the third type is the adhesion area extending from the more anterior to the lower part of the tibia. The fourth and last type does not have a solid attachment site and provides stability of the meniscus only with meniscotibial fibers (19). The anterior adhesion area is approximately 110 mm^2. While half of this area consists of support fibers, the other half consists of central fibers (20).

4.1.2. Zone 2-Anteromedial Zone

It starts from the anterior root zone and extends to the anterior border of the medial collateral ligament. It contains two subzones: 2a and 2b. 2a is continuous from the anterior root zone to the transverse ligament. Zone 2b starts from the transverse ligament and continues to the anterior border of the medial collateral ligament. Zones 2a, 2b, 3 and 4 of the medial meniscus are connected to the tibia only through meniscotibial ligaments (19). In zone 2a, the superior part of the

medial meniscus has no connection with the surrounding tissues. In zone 2b, it has a connection only with synovial tissue (18).

4.1.3. Zone 3-Medial Zone

It can also be called the medial collateral ligament zone. It is connected to the tibia inferiorly by meniscotibial ligaments, and superiorly to the joint capsule; It is the only meniscus zone that is completely connected to the joint capsule in the periphery. There is also a medial collateral ligament adjacent to the joint capsule (18).

4.1.4. Zone 4-Posterior Zone

There is no connection with the joint capsule superiorly in this zone. Inferiorly, it is connected to the tibia from the 7-10 mm distal part of the joint surface by meniscotibial ligaments (18). Posteriorly, the zone located between the posterior meniscus and the joint capsule is defined as the posterior femoral space (recess) (21).

4.1.5. Zone 5-Posterior Root

The posterior root of the medial meniscus is located posterolateral to the apex of the medial tibial eminence, anteromedial to the tibial attachment site of the posterior cruciate ligament (PCL), and posterior to the posterior horn attachment site of the lateral meniscus (22).

The anatomy of the first and fifth zones, which include the anterior and posterior roots, is especially important in meniscus transplantation surgeries. In meniscus repairs, the transosseous layer must be identified to ensure stability. In order to ensure the meniscotibial connection in area 2a, an inferior vertical suture must be placed (as there is no attachment in the superior part, it only attaches to the tibia in the inferior part). The junctions superior to zone 2b should also be taken into consideration (18). In order to perform anatomical repair in the third zone, meniscofemoral-meniscocapsular and meniscotibial ligaments must be arranged. Attention should be paid to its close proximity to the medial collateral ligament in this zone. Care must be taken to ensure that the stitches to be placed in the fourth zone do not close the posterior femoral space and restrict joint movement (18).

4.2. Lateral Meniscus

The lateral meniscus is smaller than the medial meniscus. It is also more mobile and rounder in shape. It covers 60-80% of the joint surface of the

lateral tibial plateau (6). The attachment site of the anterior horn of the lateral meniscus is located anterolateral to the anterior cruciate ligament (ACL). It is located posteromedial to the apex of the lateral tibial eminence, anteromedial to the attachment site of the medial meniscus posterior horn, and anterior to the ACL tibial attachment site. In addition to its basic fibers, some fibers also adhere to the posterolateral aspect of the medial tibial eminence (22). Additionally, it is connected to the femoral condyle and PCL via the Wrisberg ligament (posterior meniscofemoral ligament) and Humprey ligament (anterior meniscofemoral ligament). It is also adjacent to the popliteus tendon on the posterolateral side (23).

4.3. Connections Between Medial and Lateral Meniscus

There are four different ligaments between the medial and lateral menisci (24). The transverse ligament (anterior intermeniscal ligament) is located in the knee at a rate of 60-94%. The posterior intermeniscal ligament is located in 1-4% of the knee. The medial oblique intermenischeal ligament is located in 1% of the knee, runs in the center of the anterior root of the medial meniscus and extends obliquely to the superior of the posterior horn of the lateral meniscus. The lateral oblique intermenischeal ligament is located in 4% of the knee, starts from the posterior horn of the lateral meniscus and attaches to the superior part of the posterior horn of the medial meniscus (19, 25).

4.4. Meniscus Root Anatomy

The importance of the anterior and posterior roots of the meniscus in protecting the articular cartilage structure has been demonstrated by previous studies. In their study, Marzo et al. stated that the functional results in posterior root injuries of the medial meniscus correspond to total meniscectomy (26). Injuries to the roots of the meniscus disrupt the structure and biomechanics of the meniscus. As a result of injuries, the properties of the menisci such as circular load distribution, load carrying capacity, transmission and shock absorption are lost. At the end of all this; A decrease in the contact area of the menisci with the joint, an increase in joint stress, accelerated osteoarthritis and overflow are observed (26). Anatomical and stable repair of meniscus roots after injuries in these zones is of great importance in correcting impaired meniscus functions. In anatomical repairs of meniscus root injuries, anterior and posterior root anatomy must be known in detail. Knowing its anatomy is valid not only in the repair of meniscus root lesions, but also in patients who will undergo meniscus transplantation (22, 27).

The center of the anterior root of the medial meniscus, tuberosity is located proximal and medial to the tibia, anterior to the apex of the medial tibial eminence. The anteromedial root is approximately 110.4 mm², of which 56.3 mm² consists of central connections (20). The posterior root center of the medial meniscus is located posterolateral to the apex of the medial tibial eminence, anteromedial to the tibial attachment area of the ACL, and posterior to the posterior root of the lateral meniscus. The adhesion area of the posterior root is 30.4 mm² (22).

The posterior root center of the lateral meniscus is located on the anterolateral side of the ACL, anteromedial to the apex of the lateral tibial eminence. The anterolateral root is approximately 140.7 mm² and 88.9 mm² of it connects with the ACL (20). The posterior root of the lateral meniscus is located posteromedial to the apex of the lateral tibial eminence, anterior to the tibial attachment area of the PCL, and anterolateral to the posterior root of the medial meniscus. The adhesion of the posterior root is 39.2 mm². In addition to the basic fibers, some fibers also adhere to the posterolateral side of the medial tibial eminence (22).

5. Meniscus Nutrition

In children, lymphatics and blood vessels are present in all menisci until they reach the age of one. When there is a load on the meniscus as children begin to walk, 25-33% of the meniscus area that can receive blood remains peripherally for around 18 months (7). The fibrocartilaginous zone in the center is avascular. Another avascular zone is the posterolateral side of the lateral meniscus adjacent to the popliteus (28). In people over the age of fifty, the proportion of blood and lymph vessels decreases to 10-33% of the meniscus (7). There is a very rich blood supply in the anterior and posterior horns. This is necessary for the nerves in the horn areas to be nourished (29).

In adults, the nutrition of the meniscus is provided by blood vessels in the peripheral zones, while it is provided by synovial fluid diffusion in the central zone. In order for nutrition to occur by diffusion from the synovial fluid, intermittent relaxation is required in the meniscus due to muscle contractions and the loading caused by body weight. This loading does not yet occur in children who have not started walking, but in children of this age, all menisci are vascularized (7).

Menisci are more avascular. When the triangular structural sections of the menisci are examined, they are divided into three different zones. The scarlet-red (CC) zone is the peripheral zone that is fully bleedable. The red-white (KB) zone is the zone that lies at the border of the vascularized zone

and can be partially bleed. The white-white (BB) zone is the avascular central zone (30).

Inferior and superior lateral and medial geniculate arteries provide nutrition to the synovium and joint capsule. These blood vessels form the perimeniscal plexus within the synovium and capsule, feeding the peripheral and horn parts of the menisci. The lateral and medial branches of the middle geniculate artery participate in the nutrition of the menisci through the synovial tissues covering the posterior and anterior horns (31).

6. Meniscus Innervation

There are three mechanoreceptors in the 2/3 peripheral part and horns of the meniscus: Ruffini, free nerve endings, Golgi tendon organ and Paccini. There is no innervation in the inner 1/3 (29). It is thought that this situation is due to biomechanical needs. Because, while it is necessary to detect the pressure applied to the peripheral part of the meniscus and to adapt the joint to the pressure, there is no need for such a thing for the pressure applied to the central part of the meniscus. There are more vessels and nerves in the anterior and posterior horns than in their bodies. There are more nerves in the peripheral 1/3 than in the middle 1/3. Posterior horns have greater nerve conduction than anterior horns. This suggests that the posterior horn receives more load (32).

Compression and tension forces can be sensed by mechanoreceptors located in the menisci, and are transmitted to the central nervous system via afferent nerves. Motor impulses carried by efferent nerves in the central nervous system stimulate the muscles around the knee and provide motor control through reflex muscle contractions and changes in muscle tone (33). Afferent nerve fibers located in the menisci also transmit other information such as knee position, movement speed, acceleration and direction of movement to the central nervous system. As the proprioceptive feature decreases as a result of injury to the meniscus, this impairs the positioning ability of the knee joint and causes instability (34).

7. Meniscus Functional Features

Both menisci are of great importance for a healthy knee and perform multiple functions. Most importantly, they undertake more than 50% of the load on the knee (35). The percentage of the force transmitted varies depending on the position of the knee. For every 30° of knee flexion, the contact surface between the tibia and femur decreases by 4% (36). In 90° flexion of the knee, the axial load on the joint is 85% higher than in 0° flexion (37). In full flexion of the

knee, the lateral meniscus completely transmits the load of the lateral part of the knee, and the medial meniscus takes the load of the medial part of the knee by an average of 50% (36). After complete meniscectomy, the contact area is reduced by approximately half and the load per unit area is greatly increased. It has been determined that even only 15-34% meniscectomy increases contact pressure by more than 350% (38).

The menisci increase the joint harmony between the tibial and femoral condyles and ensure the normal flow of synovial fluid circulation within the knee (39). In order for the knee to perform full flexion and extension movements, it must have a normal and intact joint range of motion. The medial meniscus aids in the anteroposterior stability of the knee throughout its full range of motion. This is evidenced by the fact that there is a 33-35% increase in the forces on the PCL of the knee after medial meniscectomy (40). In summary, menisci; It has very important functions such as shock absorption, load transmission, nutrition, stability, joint lubrication and proprioception; It reduces contact stress by increasing the contact area of the knee and ensuring joint harmony (41).

8. Meniscus Pathologies

Meniscus injuries or meniscectomy generally reduce knee joint function. This is followed by the development of osteoarthritis (41). For this reason, meniscus repair is recommended whenever appropriate, and repair practices are increasing day by day (41, 42).

8.1. Traumatic Meniscus Tears

Meniscus tears can occur from traumatic knee injuries or can also occur from degenerative conditions. ESSKA (European Society of Sports Traumatology, Knee Surgery, Arthroscopy) defined traumatic meniscus tears as meniscus tears related to a sufficient amount of knee injury and sudden onset of knee pain (43). The course of injury-related traumatic meniscus tears in athletes or adolescents has not been clarified due to lack of sufficient evidence. Traumatic meniscus tears occur alone, but are often accompanied by ligament injuries (especially ACL). In a cohort study of ACL injuries, meniscus tears were detected in 820 (58.7%) of 1398 ACL injury cases (44).

Morphological tear types of meniscus may include radial, longitudinal, flap and horizontal tears (45). MRI is a very suitable method for evaluating the menisci and provides a 90% correct diagnosis (46). When viewed from the perspective of the hoop function of the menisci, radial tears cutting from the inner free edges of the menisci to their surroundings cause serious losses in the

load distribution on the knee (47). Similarly, posterior root tears also greatly affect knee joint biomechanics. Lateral meniscus posterior root tears are often accompanied by acute ACL injury (48). Medial meniscus posterior root tears typically occur in middle-aged and elderly people, and the first symptom is severe pain. Trauma as simple as tripping over a single step greatly increases the contact pressure on the medial tibial plateau, the same as a complete mesniscectomy. This pathology is a strong risk factor for the progression of knee osteoarthritis (49).

8.2. Degenerative Meniscus Lesions

These traumas are defined as lesions generally seen in individuals over the age of 35 without a history of knee trauma (43). It grows slowly and its typical features are horizontal divisions (50). MR imaging can identify a linear meniscus signal affecting the joint surface in older individuals; This situation is considered a degenerative process. An MRI analysis showed that 35% of people over age 50 had a meniscus tear; but 2/3 of these tears were asymptomatic (41).

8.3. Meniscus Extrusion

Meniscus extrusion is the displacement of the meniscus body beyond the outer edge of the tibial plateau (51). It is often accompanied by osteoarthritis, meniscus posterior root tears or radial tears. Hada et al. showed that medial tibial osteophytes can be detected at a high rate by MRI in patients with early-stage knee osteoarthritis, and this may have a close relationship with medial meniscus extrusion (52). In contrast, Krych et al. reported that disruption of meniscotibial ligaments triggered meniscus extrusion (53).

Meniscus extrusion reduces the coverage of the tibial plateau, causing an increase in cartilage load. Therefore, when compared to meniscus extrusion, meniscus tears and articular cartilage defects, it has a stronger relationship with the narrowing of the joint space (54). The commonly accepted medial meniscus threshold is a displacement of the meniscus body of more than 3 mm (55). However, the amount of displacement considered pathological has not been determined. Some scoring systems have been defined for coronal MRI data: Grade 0 = no extrusion; Grade 1= partial extrusion; and Grade 2= complete extrusion without any contact with the junction area (56). Medial meniscus extrusion also; Grade 0= No extrusion; Grade 1=≤50% extrusion; and Grade 2≥50% extrusion (57). Similarly, as part of the semiquantitative MRI evaluation for osteoarthritis, Grade 0=<2 mm; Grade 1 = 2–2.9 mm, Grade 2 = 3–4.9 mm;

and Grade 3=>5 mm. The use of three-dimensional MRI has also been evaluated to quantify meniscus extrusion (58).

9. Treatment of Degenerative Meniscus Lesions

9.1. Meniscectomy and Conservative Treatment

Most other meniscal tears that are degenerative, significantly traumatized, and/or located in an avascular area of the meniscus are treated with partial meniscectomy (59). It is known that meniscectomy causes symptomatic osteoarthritis in the long term (41). Fairbank was the first person to describe the radiological changes in the knee after meniscectomy (60). Pengas et al. They conducted a follow-up of approximately 40 years (33 to 50) years after total meniscectomy. Kellgren and Lawrence found that the incidence of knee osteoarthritis according to its degree was 81%, and this rate was only 18% in unoperated knees (61).

Clearly, not all meniscus tears can be repaired, and there is no clear guidance in the published literature as to which tears and how many should be repaired. Certainly, partial meniscectomy rates are higher than meniscus repair. Resection of the entire meniscus causes large increases in the load transferred to the articular cartilage and has been associated with a 14-fold increase in osteoarthritis rates after 21 years (62). Therefore, removing as much of the meniscus as possible will give the best results in the long term (63). When partial meniscectomy is performed, if possible, the surrounding collagen fibers, which are important for load bearing and shock absorption, should be preserved (64). These fibers are important in distributing cyclic stresses. Partial meniscectomy has the advantage of faster rehabilitation. This will likely have long-term benefits in reducing the development of osteoarthritis. One study showed the development of osteoarthritis following partial meniscectomy in 19.2% after meniscus repair versus 60% at 8.8 years (65). Other studies have noted that radiographic signs of osteoarthritis are significant for 8 to 16 years after arthroscopic partial meniscectomy, but symptoms of knee osteoarthritis are uncommon (66).

9.2. Meniscus Preservation Surgery

If surgical treatment is required for meniscus tears, meniscus repair should be preferred as the first option to preserve its structure and function (67). The ratio of meniscus repair to meniscectomy has gradually increased with the concept of meniscus preservation (68). Although meniscus repair has a higher rate of repeat surgery than meniscectomy, less degenerative radiographic changes have been

observed with meniscus repair than with arthroscopic partial meniscectomy after more than 10 years of follow-up (69).

9.3. Knee Osteotomy

In the presence of lower leg malalignment as a part of meniscus pathologies, it is very difficult to prevent degenerative changes by performing only meniscus preservation surgery (70). Therefore, knee osteotomy is considered together with meniscus preservation surgery and bone alignment can be corrected to reduce the load on the damaged cartilage. The most common osteotomies for varus deformity are medial open wedge high tibial osteotomy or lateral closed wedge high tibial osteotomy (71).

9.4. Meniscus Change

Replacement of menisci has been applied clinically for meniscus deficiency after meniscectomy. Approvable short- or medium-term clinical results of meniscus allograft transplantation in painful knees that previously underwent subtotal or total meniscectomy have been reported (72). Collagen meniscus implants, composed of collagen type I material, are an option for meniscus placement. Successful clinical and radiological findings have been reported as a result of 10-year follow-up after collagen meniscus implant transplantations. However, the approval for the use of these meniscus materials varies by country (73).

9.5. Orthobiologicals

Recently, orthobiologics have emerged as materials that will increase the weak healing potential of the menisci, especially due to insufficient blood flow at the inner edges of the meniscus (74). Platelet-rich plasma (PRP), which contains growth factors such as transforming growth factor-β, platelet-derived growth factor, vascular endothelial growth factor and basic fibroblast growth factor, is used clinically in meniscus repair. However, its effectiveness is still controversial due to differences in indications or PRP preparation techniques (75).

10. Conclusion

In this section, the anatomical structure, cell content and clinical importance of the menisci in the knee are discussed. Menisci can be damaged for many different reasons and directly affect knee joint function. Therefore, meniscus anatomy should be known in detail by clinicians in treatment approaches.

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CHAPTER XII

OBTURATOR ARTERY ANATOMY AND CLINICAL SIGNIFICANCE

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1. Introduction

The vascular anatomy of the pelvic region is crucial due to the potential for life-threatening bleeding following urogynecologic interventions and pelvic traumas (1). Like all other arteries in the pelvic region, the obturator artery and its branches are at risk of bleeding during surgical interventions and traumas in the area (2). Particularly, complications requiring reoperation due to injuries to the neurovascular structures and the obturator artery (a. obturatoria) in the medial aspect of the ischiopubic ramus have been reported (1). The obturator artery and its branches exhibit significant variation. Consequently, bleeding from the artery may go unnoticed, rendering treatments ineffective (2).

During procedures such as lymphadenectomy, hysterectomy, and stress incontinence treatment, it has been reported that variant branches of the obturator artery may be at risk of injury. Additionally, during bilateral internal iliac artery ligation, it should not be forgotten that obturator artery pubic branches may anastomose with the inferior epigastric artery, which could impact the success of the surgery (3).

In acetabular osteotomies, the possibility of obturator artery injury is high due to the invisibility of the operating field and the unknown location of the chisel tip in the obturator foramen (4). Furthermore, during direct or indirect inguinal, femoral, or obturator hernia repairs, it should be remembered that obturator artery variations may be close to the femoral ring and could be injured (2).

In all the aforementioned surgeries, the knowledge of detailed anatomy and branching variations of the obturator artery is vital for ensuring success, preventing complications, and managing bleeding after pelvic traumas.

2. Anatomy of the Internal Iliac Artery

The common iliac artery extends inferolaterally on both sides and bifurcates again into the internal iliac artery and the external iliac artery at the level of the sacroiliac joint. While the internal iliac artery generally supplies blood circulation to the pelvic parietal and visceral structures, the external iliac artery supplies blood to the lower extremities (5).

The internal iliac artery originates as a terminal branch of the common iliac artery at the anterior aspect of the sacroiliac joint. It then progresses posteriorly and inferiorly towards the greater sciatic foramen within the lesser pelvis. At the upper margin of the foramen, it bifurcates into two branches, anterior and posterior. While the anterior branch heads towards the ischial spine, the posterior branch directs towards the greater sciatic foramen (5, 6). Its length is approximately 3-4 cm, and its diameter does not exceed 8 cm (7). This artery supplies blood to the gluteal region muscles, pelvic visceral and parietal structures, a portion of the inner thigh, and genital organs (5).

The anterior division of the internal iliac artery supplies blood to structures such as the bladder, ureter, ductus deferens (male), rectum, uterus (female), cervix (female), anal canal, superficial transverse perineal muscle, bulb of the penis (male), corpus cavernosum penis (male), and gluteal muscles. Additionally, it supplies blood to the obturator externus muscle, pectineus muscle, gracilis muscle, and adductor muscles through the obturator artery. The posterior division supplies blood to structures such as the psoas major muscle, quadratus lumborum muscle, iliacus muscle, sacral canal, gluteus maximus muscle, and the dorsal surface of the sacrum (7).

The internal iliac artery is a continuation of the common iliac artery in intrauterine life and is a much thicker branch than the external iliac artery. On both sides of the bladder, one branch, one on the lateral side, originates from the internal iliac artery and proceeds towards the anterior abdominal wall. These branches approach the midline on the posterior aspect of the anterior abdominal wall and pass through the navel and distribute in the placenta. The parts of these arteries, called umblical arteries, that remain distal to the superior vesical artery, become obliterated after birth. This closed section becomes a ligament and is called the medial umblical ligament (5).

If the internal iliac artery is ligated unilaterally, certain anastomoses in the arterial system continue the circulation distally to supply the relevant structures. These anastomoses include:

- Anastomoses between the ovarian artery and the uterine artery.
- Anastomoses between the arteries of both sides of the bladder.
- Anastomoses between the branches of the internal iliac artery and the inferior mesenteric artery supplying the rectum.
- Anastomoses between the obturator artery's pubic branch and the same branch on the contralateral side, as well as between the inferior epigastric artery and the medial circumflex femoral artery.
- Anastomoses between the inferior gluteal artery and the circumflex and perforating branches of the deep femoral artery.
- Anastomoses between the superior gluteal artery and the posterior branches of the lateral sacral artery.
- Anastomoses between the iliolumbar artery and the final lumbar artery.
- Anastomoses between the lateral sacral artery and the median sacral artery.
- Anastomoses between the deep iliac circumflex artery and the iliolumbar artery and superior gluteal artery (5).

The branches of the internal iliac artery exhibit considerable variation. Therefore, surgeons operating in the pelvic region must exercise caution during dissection of this area (6). While some sources classify the branches of the internal iliac artery as parietal and visceral, others classify them as those arising from the anterior and posterior divisions. Branches arising from the posterior division include the superior gluteal artery, iliolumbar arteries, and lateral sacral arteries, while branches arising from the anterior division include the umbilical artery, uterine artery, superior vesical artery, vaginal artery, middle rectal artery, obturator artery, internal pudendal artery, and inferior gluteal artery (5).

The internal iliac artery is in contact with the ureter anteriorly, the internal iliac vein posteromedially, the obturator nerve, obturator vein, and external iliac vein posterolaterally, the terminal ileum and cecum (on the right side) anteromedially, the psoas major muscle laterally, and the obturator internus muscle (8).

3. Variations of the Obturator Artery

3.1. Variations in the Origin of the Obturator Artery

Anatomical studies have indicated that the obturator artery may originate from the internal iliac artery, external iliac artery, inferior epigastric artery, common iliac artery, internal pudendal artery, iliolumbar artery, inferior gluteal artery, superior gluteal artery, or femoral artery, or it may have a dual origin. Additionally, in a study conducted by Sañudo et al. (2011) (1), a variation was reported where the artery originated from three different arteries in one case. Furthermore, there is a study reporting the absence of the obturator artery, in which case the area that should be supplied by the obturator artery was found to be supplied by the medial femoral circumflex artery (10). The variation where the obturator artery originates from the external iliac artery is termed "aberrant obturator artery." The frequency of occurrence of an aberrant obturator artery originating from the inferior epigastric artery ranges between 20% and 34%. This artery passes behind the lacunar ligament and enters the obturator foramen perpendicularly through the superior ramus of the pubis. Therefore, the obturator artery originating from the inferior epigastric artery is vulnerable to injury during preperitoneal space dissection. Accessory obturator artery refers to the presence of an additional obturator artery in addition to the normal obturator artery (12). Kostov et al. (2020) (12) included the recognition of piercing the obturator membrane without participating in any anastomosis as part of this definition. In addition, all cases in which the artery does not emerge from the internal iliac artery or the anterior root of the internal iliac artery are called "variant obturator artery" (11). Although some publications report more obturator artery origin anomalies in females, most studies indicate no statistically significant difference between genders (12).

Reviewing the literature, it is observed that the most common origin of the obturator artery is from the internal iliac artery or its branches. This artery most commonly originates from the anterior division of the internal iliac artery. Cases have been reported where both hemipelvises follow the same pattern while having different branching patterns. In cases of dual origin, both origins can arise from the same artery, or one origin can arise from the internal iliac artery while the other arises from the external iliac artery (1).

In a study by Sañudo et al. (2011) (1), it was noted that if the vessel is dual-origin, both arteries merge with a small anastomotic branch and both arteries exit the pelvis through the obturator canal. The same study identified a case with three origins of the obturator artery from the anterior root of the internal iliac artery, the posterior root of the internal iliac artery, and the external iliac artery.

3.2. Variations in the Branches of the Obturator Artery

3.2.1. Foveolar Artery

The foveolar artery is the anterior branch of the posterior root of the obturator artery. This branch enters the fovea capitis femoris through the ligamentum capitis femoris and contributes to the blood supply of the femoral head. However, this artery is not patent in all adults. It is considered an embryonic remnant that does not play a significant vascular role in adult hips (13).

Anatomical descriptions of the foveolar artery vary. Studies provide conflicting results regarding whether the artery persists in the adult hip joint. While some studies suggest continuity of the artery in the adult hip joint, others suggest that it becomes vestigial and contributes little to vascularization (13,14).

3.2.2. Corona Mortis

Corona mortis is a variant anastomosis between the external iliac artery or inferior epigastric artery and the obturator artery. This anastomosis is located behind the superior ramus of the pubic bone (4, 12, 15). The term “crown of death” denotes its significance. Fatal bleeding may occur following trauma or surgical injury to this anastomosis. Due to its proximity to the superior ramus of the pubis, it is vulnerable to injury. The presence of this anastomosis should not be overlooked by radiologists and surgeons during arterial embolization performed to control bleeding in pelvic trauma and especially approaches to pelvic fractures. Furthermore, being aware of potential anatomical variations in the origin and course of bleeding arteries is vital in the treatment of such patients (15). Corona mortis may be in close proximity to the free edge of the lacunar ligament and the neck of the femoral hernia sac. The close proximity of the anastomosis to the lacunar ligament and annulus femoralis can be a major source of concern in surgeries to the area (12).

Based on CT angiography studies, the frequency of corona mortis ranges between 25% and 33%, while cadaveric studies report rates of up to 65% (15).

3.2.3. A Case Report Describing an Abnormal Branching Pattern

In one study, an abnormal branching pattern of the obturator artery was identified during dissection of an adult female cadaver. During examination of the right hemipelvis, the obturator artery gave rise to two abnormal branches on the lateral wall of the pelvis. The first branch divided into three small branches, while the second branch divided into two small branches. The artery itself was observed to continue its course by piercing the obturator membrane (3).

3.2.4. Arteria Nutricia Arising from the Obturator Artery

Arteria nutricia arising from the obturator artery is rarely mentioned in the literature. Bosse et al. (16) identified an arteria nutricia arising from the obturator artery in one right hemipelvis (a 65-year-old female) out of 34 pelvic hemisections examined. Given that this vessel can be injured during intrapelvic surgeries such as pelvic lymph node dissection or procedures requiring arterial embolization of the obturator artery, its size and course should be considered (16).

3.2.5. Anastomosis with the Uterine Artery

Uterine artery embolization for the treatment of uterine fibroids can be used as an alternative to hysterectomy for menorrhagia. However, some studies have reported cases where the uterine artery forms anastomoses with the obturator artery. Anatomical variations in the arteries supplying uterine fibroids can result in incomplete or off-target embolization (17).

4. Surgical Interventions That May Cause Damage to the Arteria Obturatoria

4.1. Use of the Arteria Obturatoria Variant in Autologous Breast Reconstruction

The obturator artery variant arising from the deep inferior epigastric artery is encountered with a frequency that cannot be ignored. In autologous breast reconstruction, it can be detected before the operation or during the operation. The use of this anatomical variant in autologous breast reconstruction provides better size compatibility compared to other anastomosis options used for perfusion of primary and secondary flaps (18).

4.2. Uterine Artery Embolization

One of the most common pelvic tumors in women is uterine myomas. The main methods used in their treatment include GnRH analogs, hysterectomy, myomectomy, and embolization. Uterine myoma embolization is recommended in current studies as an alternative to myomectomy and hysterectomy for symptomatic treatment. Hysterectomy may not be preferred due to its potential to impair fertility. However, myomectomy is a more invasive procedure compared to embolization and has a longer recovery time (19).

In this surgery, a guidewire is first inserted into the uterine artery with the aid of catheterization. Subsequently, contrast material is administered to the

relevant area for visualization of vascularization. Then, microsphere injection is performed. Injection continues until all branches of the uterine artery are embolized. Following the procedure, a sandbag is placed on the inguinal area along with bed rest. There is a possibility of experiencing vaginal bleeding similar to menstruation after the procedure (19).

4.3. Internal Iliac Artery Ligation

Bleeding due to childbirth can reach a critical level and requires immediate intervention. Surgical intervention is necessary in cases where non-invasive treatment is not feasible. Although hysterectomy is the most effective treatment method for such bleeding, it may not be preferred for fertility preservation. Therefore, ligation of the internal iliac artery serves as an alternative method to hysterectomy. This procedure is based on ligating the internal iliac artery using sutures (20).

4.4. Femoral Hernias

The femoral canal is bounded by the femoral vein laterally, the inguinal ligament anteriorly, the lacunar ligament medially, and the pectineal ligament posteriorly. A femoral hernia occurs when a peritoneal sac containing adipose tissue and abdominal or pelvic material protrudes through the femoral canal. Surgery is the only satisfactory treatment for this condition. The objectives of the surgery include reducing the hernia, excising the sac, and closing the femoral canal to prevent recurrence (21).

4.5. Inguinal Hernias

Inguinal hernias observed in the inguinal region are classified into two groups: direct and indirect. A direct inguinal hernia involves protrusion of abdominal tissue from the posterior wall of the inguinal canal, medial to the inferior epigastric artery, while an indirect inguinal hernia involves protrusion of the hernia contents passing through the inguinal canal via the deep inguinal ring, lateral to the inferior epigastric artery (22).

4.6. Pelvic Fractures

Pelvic injuries are potentially fatal and highly morbid traumas. In the early stage of these fractures, injuries to the head, urogenital and gastrointestinal systems, bleeding, and nerve damage may accompany the injury. In the late stage, pain, nonunion, limb asymmetry, and neurological sequelae may occur. To avoid missing any open fractures or accompanying injuries, rectal and

vaginal examinations should be performed on all patients. In men, a high-riding prostate on rectal examination and blood in the urine suggest urethral injury, while vaginal or rectal tears due to bone fragments may lead to highly fatal open fractures (23).

5. Conclusion

The arteria obturatoria is an artery with very common variations. Failure to consider its variations and anastomoses in interventions for its injuries may lead to fatal consequences. Considering its topography, it is vulnerable to injury during hernia surgeries, acetabulum osteotomies, and urogynecological surgeries. Familiarity with the anatomy of the artery and identification of its variations through detailed imaging before surgical interventions and interventions for pelvic bleeding are essential for clinicians.

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CHAPTER XIII

ANATOMY, VARIATIONS AND CLINICAL SIGNIFICANCE OF OPHTHALMIC ARTERY

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1. Introduction

The ophthalmic artery, which nourishes all structures within the orbit including the eyeball, typically arises from the convex anterior surface of the internal carotid artery as it exits the cavernous sinus intracranially (1, 2). It enters the orbit through the optic canal and terminates by branching into terminal branches called the supratrochlear artery and dorsal nasal artery. Depending on its location during its course, it can be divided into three different segments: intracranial, intracanalicular, and intraorbital. It gives rise to orbital branches that supply the orbit and surrounding structures, as well as ocular branches that supply the eyeball and its muscles. Numerous variations in origin, course, branches, and anastomoses have been described in studies. Furthermore, the ophthalmic artery undergoes a complex embryological development, and studies have suggested that these anatomical variations stem from its intricate embryology (1, 3).

Detailed anatomical and variation information regarding the ophthalmic artery is essential for understanding the pathophysiology, diagnostic approach, and therapeutic modalities of various diseases. Therefore, its anatomy has been extensively studied and described in many anatomy and surgical textbooks and articles (2).

For example, understanding the origin of the ophthalmic artery defines the appropriate approach for selective catheterization of the ophthalmic artery for the treatment of central retinal artery occlusion or chemoembolization

for retinoblastoma. Possible anastomoses between the ophthalmic artery and the external carotid artery represent a useful resource or a potential hazard. They may be beneficial when attempting to deliver intra-arterial drugs to the eye and provide a clear advantage when collateral pathways are established following acute ophthalmic artery occlusion, preserving vision in nearly 85% of cases. However, these same pathways may pose a risk of unintended passage of embolic material from branches of the external carotid artery to the ophthalmic artery or into the internal carotid artery region during embolization procedures (4).

Injecting fillers into the periorbital region's vessels can lead to serious complications such as blindness and cerebral embolism. Therefore, a thorough understanding of arterial variations around the periorbital region is crucial in patients undergoing facial filler injections (5).

The clinical significance of the anterior and posterior ethmoidal arteries lies in their roles in supplying vessels for meningiomas and vascular malformations. Additionally, the ethmoidal arteries may contribute to epistaxis by remaining as an effective treatment through embolization (2).

The anatomy of the ophthalmic artery is complex. Its intracranial and extracranial course, small size, proximity to many important anatomical structures, particularly the optic nerve, and its vital importance for vision make this artery a significant anatomical and surgical challenge. Advances in neurosurgery, ophthalmology, and radiology have increased diagnostic and treatment options, especially for diseases like retinoblastoma and central retinal artery occlusion. The ophthalmic artery also plays a crucial role in the treatment of intravascular lesions and epistaxis. Therefore, detailed and in-depth knowledge of its anatomy from a clinical perspective is essential for understanding the pathophysiology, diagnostic approach, and therapeutic modalities of its diseases and for preventing complications (3).

2. Embryology of the Ophthalmic Artery

The ophthalmic artery can be considered the most complex embryological development among craniofacial arteries. This complexity arises from several factors. Firstly, there is a lack of knowledge about various embryological stages, and secondly, three different embryological systems are mentioned: the primitive internal carotid artery, stapedial artery, and pharyngeal artery systems. Another factor is the abundance of hypotheses proposed by various authors, which may lack scientific basis and could be misinterpreted as evidence-based information (6). The embryological development of the ophthalmic artery is completed in

the late stages of embryonic development, yet all stages of its development are still not fully understood (7).

During the embryonic stage of 4-8 mm, the orbit is supplied by two arteries: the ventral ophthalmic artery and the dorsal ophthalmic artery. The ventral ophthalmic artery originates from the anterior cerebral artery and enters the orbit through the optic canal. The dorsal ophthalmic artery emerges from the junction of the 5th and 6th segments of the internal carotid artery and enters the orbit through the superior orbital fissure (3, 7).

In the embryonic stage of 7-12 mm, the dorsal ophthalmic artery provides capillary vascularization to the optic cup. At this stage, vascularization of the optic structures is in a mesh-like configuration (3, 7).

During the embryonic stage of 12-14 mm, as the eye moves away from the brain, the lengths of the ventral and dorsal ophthalmic arteries begin to elongate. At this stage, the dorsal ophthalmic artery gives rise to branches such as the hyaloid artery and temporal ciliary arteries. The hyaloid artery is a precursor artery of the central retinal artery, while the temporal ciliary arteries are precursors of the lateral posterior ciliary arteries (3, 7).

In subsequent stages, two anastomoses form. One anastomosis occurs between the ventral and dorsal ophthalmic arteries around the optic nerve within the orbit. The other occurs between the internal carotid artery and the ventral ophthalmic artery near the optic canal in the intradural space (3, 7).

In later stages, the proximal portion of the ventral ophthalmic artery regresses. The primitive ophthalmic artery originates from the supraclinoid segment of the internal carotid artery and is named the “Primitive Ophthalmic Artery”. The origin of the primitive ophthalmic artery marks the junction of the 6th and 7th segments of the internal carotid artery. It enters the orbit through the optic canal. As it enters the orbit, it passes through the ring formed by the three branches of the dorsal ophthalmic artery (temporal ciliary arteries, nasal ciliary arteries, and central retinal artery) (3, 7).

Around the embryonic stage of approximately 18 mm, the dorsal ophthalmic artery begins to regress at the level of the superior orbital fissure. The proximal remnant of the dorsal ophthalmic artery forms the “Inferolateral Trunk”. The distal intraorbital remnant of the dorsal ophthalmic artery forms the “Deep Recurrent Ophthalmic Artery”. The deep recurrent ophthalmic artery provides the connection between the ophthalmic artery and the inferolateral trunk. If the dorsal ophthalmic artery persists or remains dominant, the ophthalmic artery originates from the cavernous segment of the internal carotid artery (3, 7).

3. Anatomy of the Ophthalmic Artery

The ophthalmic artery branches from the convex anterior surface of the internal carotid artery as it typically exits the cavernous sinus. It travels along the inferior-lateral aspect of the optic nerve, passes through the optic canal, and enters the orbit. Within the orbit, it progresses above the optic nerve and reaches the medial wall of the orbit. It then comes to the medial corner of the upper eyelid, between the lower border of the inferior oblique muscle and the medial rectus muscle. Here, it divides into terminal branches called the supraorbital artery and the dorsal nasal artery. The final portion of the artery, where it branches into its terminal branches, runs along with the branch of the nasociliary nerve called the infratrochlear nerve on the medial wall of the orbit. The tortuous course of the artery is important in preventing its interference with eye movements, nourishing all structures within the orbit, including the eyeball itself (1, 8).

3.1. Branches of the Ophthalmic Artery

We can categorize the branches of the ophthalmic artery into two groups: orbital branches, which supply structures within the orbit and its surrounding tissues, and ocular branches, which supply the eyeball and its muscles (1, 9).

Orbital Branches:

a) Lacrimal artery
Lateral palpebral arteries
b) Supraorbital artery
c) Posterior ethmoidal artery
d) Anterior ethmoidal artery
e) Medial palpebral arteries
f) Supratrochlear artery
g) Dorsal nasal artery
Ocular Branches:
h) Central retinal artery
i) Short posterior ciliary arteries
j) Long posterior ciliary arteries
k) Muscular branches
Anterior ciliary arteries
Anterior conjunctival arteries
Episcleral arteries

3.1.1. Lacrimal Artery

The lacrimal artery, a branch of the ophthalmic artery, is the thickest branch and provides nourishing branches to the lacrimal gland, eyelids, conjunctiva, lateral rectus muscle of the eye, cheek, temporal muscle, and dura mater of the brain. Upon entering the orbit, it separates near the optic canal, sometimes even before entering the canal. Alongside the lacrimal nerve, it extends along the upper edge of the lateral rectus muscle to reach the lacrimal gland. Within the gland, it terminates by giving numerous terminal branches. These terminal branches pass through and exit the gland, extending to the conjunctiva and eyelids. Among these branches, two major arteries, one for the upper eyelid and one for the lower eyelid, extend medially. These arteries, called the lateral palpebral arteries, anastomose with the medial palpebral arteries coming from the medial side. The anastomosis on the upper eyelid is called the superior palpebral arch, while the one on the lower eyelid is called the inferior palpebral arch. Together, they form a vascular ring around the eyelid. The lacrimal artery gives one or two zygomatic branches. One of them passes through the zygomaticotemporal foramen to reach the temporal fossa, where it anastomoses with branches of the deep temporal artery. The other branch passes through the zygomaticofacial foramen to reach the cheek, where it anastomoses with the transverse facial artery. One branch of the lacrimal artery turns back and enters the middle cranial fossa from the lateral part of the superior orbital fissure. This branch, called the recurrent meningeal branch, anastomoses with a branch of the middle meningeal artery. Occasionally, the lacrimal gland may be nourished by one of the anterior branches of the middle meningeal artery through this route (1, 10).

3.1.2. Supraorbital Artery

The supraorbital artery provides nourishing branches to the superior rectus muscle, levator palpebrae superioris muscle, skin of the forehead, muscles, and periosteum of the forehead, and the frontal bone. It is a branch of the ophthalmic artery, emerging at the point where the ophthalmic artery crosses the optic nerve. Within the orbit's upper wall, it passes medially from the superior rectus muscle and the levator palpebrae superioris muscle. Here, it runs alongside the supraorbital nerve and extends toward the supraorbital foramen, between the periosteum and the levator palpebrae superioris muscle. Upon passing through the foramen, it divides into superficial and deep branches. These branches nourish the skin, muscles, and periosteum of the forehead. The supraorbital artery anastomoses with the superficial temporal artery and arteries from the

contralateral side. Within the orbit, it gives thin branches to nourish the superior rectus muscle and the levator palpebrae superioris muscle (1, 10).

3.1.3. Posterior Ethmoidal Artery

Entering the anterior cranial fossa through the posterior ethmoidal foramina, this artery provides branches to the posterior ethmoidal cells. In the anterior cranial fossa, it gives rise to the recurrent meningeal branch for the dura mater of the brain. Another branch descends through a hole in the cribriform plate to enter the nasal cavity. The lateral nasal branches, distributed in the upper-back part of the nasal mucosa, anastomose with the sphenopalatine artery (1, 10).

3.1.4. Anterior Ethmoidal Artery

Thicker than the posterior ethmoidal artery, this artery enters the anterior cranial fossa through the anterior ethmoidal foramen alongside the nasociliary nerve. Within the anterior cranial fossa, it gives rise to the anterior meningeal branch for the dura mater of the brain. Along its course, it provides branches to the frontal sinus, anterior ethmoidal cells, and middle ethmoidal cells. Additionally, it passes through a fissure alongside the crista galli to emerge between the nasal bone and the nasal cartilage, giving rise to the anterior nasal arteries. These arteries supply lateral nasal branches to the anterior-outer wall of the nasal cavity, septal branches to the anterior part of the nasal septum, and a terminal branch that nourishes the dorsum of the nose (1, 10).

3.1.5. Medial Palpebral Arteries

Arising near the trochlear cartilage from the ophthalmic artery, one branch extends upward, and the other extends downward toward the upper and lower eyelids. Each of these arteries that enters the eyelids divides into two branches. These branches extend laterally between the orbicularis oculi muscle and the tarsus and along the lateral edge of the tarsus. Two branches converge laterally to form a vascular ring. The portion of this ring located on the upper eyelid is called the superior palpebral arch, while the portion on the lower eyelid is called the inferior palpebral arch. The artery in the upper eyelid anastomoses laterally with the zygomaticoorbital artery (a branch of the superficial temporal artery) and the lateral palpebral arteries. In the lower eyelid, it anastomoses laterally with the lateral palpebral arteries (a branch of the lacrimal artery), the transverse facial artery, and a branch of the angular artery. One branch arising from this anastomosis enters the nasolacrimal duct and nourishes its mucosa (1, 10).

3.1.6. Supratrochlear Artery

One of the terminal branches of the ophthalmic artery, the supratrochlear artery emerges from the frontal foramen at the superior-medial edge of the orbit, alongside the supratrochlear nerve. It ascends on the forehead, nourishing the skin, muscle, and periosteum. The supratrochlear artery anastomoses with the supraorbital artery and the artery from the contralateral side (1, 10).

3.1.7. Dorsal Nasal Artery

This artery is the other terminal branch of the ophthalmic artery. It exits the orbit above the medial palpebral ligament. After providing a branch to the upper part of the lacrimal sac, it bifurcates. One branch crosses the root of the nose to anastomose with the angular artery, while the other extends along the dorsum of the nose, supplying its outer surface. A branch called the lateral nasal artery, a branch of the facial artery, anastomoses with the same branch from the contralateral side (1, 10).

3.1.8. Central Retinal Artery

The first and smallest branch of the ophthalmic artery, the central retinal artery is the primary supplier of blood to the retina. It extends a short distance within the dural sheath surrounding the optic nerve. Approximately 1.25 cm behind the eyeball, it penetrates obliquely through the optic nerve into the eye, termed the pars extraocularis. Upon reaching the optic nerve's center at the optic disc, it bifurcates into lateral and medial branches. These branches give rise to fine vessels that distribute within the layers of the eyeball. This portion is termed the pars intraocularis. These branches further divide into inferior and superior branches, distributing within the retina. Venous counterparts accompany the central retinal artery throughout its course and branching. Occasionally, the central retinal artery may be a branch of the lacrimal artery (1, 10).

3.1.9. Short Posterior Ciliary Arteries

These arteries, numbering from 6 to 12, arise from the ophthalmic artery or its branches. They extend posteriorly around the optic nerve to reach the back of the eyeball. Passing through the holes in the sclera surrounding the optic nerve's attachment to the eyeball, they supply the choroid and ciliary processes (1, 10).

3.1.10. Long Posterior Ciliary Arteries

There are two of these arteries, which pass through holes near the optic nerve before extending to the lateral and medial surfaces of the eyeball and

reaching the ciliary muscles between the sclera and the choroid. They divide into branches here. These branches anastomose near the iris's outer margin, forming the major arterial circle of the iris. This arterial circle sends numerous branches into the iris, which, in turn, anastomose near the pupil, forming the minor arterial circle of the iris (1, 10).

3.1.11. Muscular Arteries

Typically originating as a single trunk, there are two branches: superior and inferior. The superior branch is often absent, but when present, it supplies the levator palpebrae superioris muscle, superior rectus muscle, and superior oblique muscle. The inferior branch is more commonly present, passing anteriorly between the optic nerve and the inferior rectus muscle to supply the lateral rectus, medial rectus, inferior rectus, and inferior oblique muscles. These branches may arise from the lacrimal artery, supraorbital artery, or ophthalmic artery. Most of the anterior ciliary arteries arise from this artery. Its branches include:

Anterior Ciliary Arteries: Most of these arise from the muscular arteries, while some originate from the lacrimal artery. They follow the tendons of the rectus muscles to the front of the eyeball, where they pierce the sclera near the cornea to terminate in the major arterial circle.

Anterior Conjunctival Arteries: Arising from the muscular arteries, these arteries supply the conjunctiva.

Episcleral Arteries: Arising from the anterior ciliary arteries, these arteries distribute within the sclera (1, 8, 10).

4. Variations of the Ophthalmic Artery

The anatomy of the ophthalmic artery exhibits significant variations due to abnormal origins and anastomoses with neighboring arteries. These anatomical variations are attributed to the complex embryology of the ophthalmic artery (3).

To better understand the different origins of the ophthalmic artery, it is necessary to have knowledge of the segments of the internal carotid artery. The Terminologia Anatomica (1998) divides the internal carotid artery into four segments: cervical part, petrous part, cavernous part, and cerebral part. However, Bouthiller et al. (1996) classified the internal carotid artery into seven anatomical segments (C1 to C7) based on microsurgical landmarks and surrounding anatomical structures. This classification is widely used by surgeons and radiologists (12).

C1 (Cervical Part): This segment extends from the bifurcation of the common carotid artery within the carotid sheath to the entry into the skull through the carotid canal (extradural course).

C2 (Petrous Part): This segment traverses through the petrous part of the temporal bone to reach the foramen lacerum (extradural course).

C3 (Lacerum Part): Extending from the upper part of the foramen lacerum to the petroclival ligament (extradural course).

C4 (Cavernous Part): This segment extends from the petroclival ligament to the posterior clinoid process, traversing the lateral aspect of the sphenoid bone and piercing the dura mater to form the roof of the cavernous sinus (extradural course).

C5 (Clinoidal Part): It emerges from the proximal dural ring of the cavernous sinus, extends distally to the distal dural ring, and enters the subarachnoid space (intradural course).

C6 (Ophthalmic Part): This segment extends from the distal dural ring to the origin of the posterior communicating artery (intradural course).

C7 (Communicating Part): It extends from the origin of the posterior communicating artery to the bifurcation of the internal carotid artery (intradural course) (12).

4.1. Variations of the Ophthalmic Artery

4.1.1. Atypical Origins from the Internal Carotid Artery

In over 90% of cases, the ophthalmic artery arises as the first intradural branch (C6 segment) of the internal carotid artery (6). Based on the dissection of 170 human orbits, Hayreh (2006) noted that the origin of the ophthalmic artery can vary from 3 mm proximal to 4.8 mm distal to the proximal edge of the distal dural ring. Matsumura and Nagashima (1999) classified the origin of the ophthalmic artery into four types:

Type A: Originating distal to the roof of the cavernous sinus intradurally.
Type B: Originating intradurally but within the roof of the cavernous sinus.
Type C: Arising from below the roof of the cavernous sinus.
Type D: Intracavernous origin (13).

Intracavernous origin of the ophthalmic artery is a rare variation, estimated to have an incidence of 0.4%. In a few cases (3%), it has been reported that the ophthalmic artery travels in a separate bony canal called the duplicate optic canal before reaching the optic canal (7).

Erdoğmuş and Govsa (2006) stated that the intradural origin of the ophthalmic artery is the most common, with an extradural origin observed in only 5% of dissected orbitals (14).

Hayreh (2006) described that in the majority of cases (57%), the ophthalmic artery arises from the anteromedial aspect of the internal carotid artery. Other possible positions for the origin of the ophthalmic artery are superomedial (35%), medial (7%), or anterosuperior (1%) directions from the internal carotid artery (12).

4.1.2. Double Origins of the Ophthalmic Artery

Double origins of the ophthalmic artery from the internal carotid artery are extremely rare, with an incidence reported around 0.2%, and only a few cases have been reported in the literature (4). The double origin from the internal carotid artery can arise from the C3 and C4 segments or the siphon caroticum, corresponding to the failure of the primitive ventral ophthalmic artery to regress and the migration of the primitive dorsal ophthalmic artery, respectively (12). Additionally, a case of double ophthalmic artery originating from the C3 segment and the middle meningeal artery without any orbital anastomosis has been reported in one case (15). On the other hand, the presence of a variant ophthalmic artery originating from the external carotid artery alongside a normal ophthalmic artery from the internal carotid artery is not so rare (16).

4.1.3. Origin from the Middle Meningeal Artery

In adults, the intracranial portion of the artery of the stapes becomes the middle meningeal artery. Its orbital branch is responsible for vascularization of the extraocular structures of the orbit and enters the orbit through the superior orbital fissure. Variations in the development of the artery of the stapes lead to several possible outcomes.

The incidence of the ophthalmic artery originating from the middle meningeal artery and entering the orbit via the superior orbital fissure has been reported between 1.4% and 2.5% in studies (3, 4, 12). In a Japanese study, Uchino et al. (2013) reported an incidence of 1.45% (26 cases) of variants of the ophthalmic artery arising from the middle meningeal artery among 1652 ophthalmic arteries (17). In this anatomical variation, the orbital arteries are supplied by the anterior part of the middle meningeal artery passing through the superior orbital fissure or the foramen of the sphenoid bone (18). The central retinal artery usually derives its vascular supply from the supracavernous part of the internal carotid artery, but in a few cases, it can be supplied by the

middle meningeal artery without the involvement of the internal carotid artery. The variant ophthalmic artery from the middle meningeal artery can entirely vascularize the orbit in the absence of a normal ophthalmic artery or when a smaller normal ophthalmic artery is present (6).

4.1.4. Other Possible Origins of the Ophthalmic Artery

Limited knowledge of the embryogenesis of the ophthalmic artery does not explain all possible origins of the ophthalmic artery. Typically, these anatomical variations are associated with segmental anomalies.

Two cases of the ophthalmic artery originating from the anterior cerebral artery have been described and angiographically demonstrated (3, 12). This rare variation can be explained by the embryological relationship between the anterior cerebral artery as a branch of the primitive anterior part of the internal carotid artery and the failure of regression of the primitive ventral ophthalmic artery.

The origin of the ophthalmic artery from the posterior communicating artery has been described in one case, but this variant has not been explained by embryological knowledge. It is unclear whether the ophthalmic artery originates directly from the posterior communicating artery or from the supraclinoid segment of the internal carotid artery perfused only by the posterior communicating artery (19).

The origin of the ophthalmic artery from the basilar artery is a rare variation. It has only been described once in this context and was associated with an orbital arteriovenous malformation (20). Sade et al. (2004) also described an ophthalmic artery originating from the basilar artery in a 48-year-old woman. This study represents the first report identifying the basilar artery as the origin of the ophthalmic artery in the absence of vascular malformation. Classical embryological knowledge cannot explain this variation (21).

Table 1: Comparison of ophthalmic artery origin variations in different studies.

Authors	Materials and methods	MMA	ACA	Double OA origin	PcomA	BA
Uchino et al. (2013) (17)	MR 846 patients	1,45%		0,42%		
Tsutsumi et al. (2013) (22)	MR 196 patients	9,2%				
Kam et al. (2003) (15)	Angiography 1 patient	50%		50%		
Sade et al. (2004) (21)	MR 1 patient					50%
Hassler et al. (1989) (23)	Angiography 1 patient		50%			
Naeini et al. (2005) (19)	Angiography 1 patient				50%	
LI et al. (2011) (24)	Angiography 1 patient		50%			
Rivera et al. (2014) (25)	Angiography 1 patient					50%
Mishra et al. (2020)	Angiography 1 patient	100%				

(MMA: Middle Meningeal Artery Origin, ACA: Anterior Cerebral Artery Origin, PcomA: Posterior Communicating Artery Origin, BA: Basilar Artery Origin)

4.2. Variations in the Course of the Ophthalmic Artery

The course of the ophthalmic artery can be divided into three different segments: intracranial, intracanalicular, and intraorbital.

After originating from the internal carotid artery, the ophthalmic artery exhibits a short intracranial course. In approximately 85% of cases, the artery travels in the subdural space, in 10% it travels between the layers of the dura, and in 5% it travels entirely extradurally. To reach the optic canal, it travels infero-laterally to the optic nerve in 70% of cases, infero-medially in 15%, and infero-medially in 15% (14).

During its course in the optic canal, the ophthalmic artery typically travels below the optic nerve, usually at the infero-lateral aspect. Studies have reported

that in about 64% of cases, the artery is positioned infero-laterally to the optic nerve in the optic canal. Cadaveric studies have consistently noted that the ophthalmic artery is always situated below the nerve.

Subsequently, the ophthalmic artery enters the orbit, where its intraorbital portion can be further divided into three segments (27). The first segment extends from the point of entry into the orbit to the point where the artery curves to form the second segment. In the first segment, the ophthalmic artery courses along the infero-lateral aspect of the optic nerve. The second segment involves the artery passing over or under the nerve towards its supero-medial aspect to reach the supero-medial corner of the orbit. The anatomical variations are most commonly encountered in this segment. Approximately 85% of cases show the ophthalmic artery passing over the optic nerve, while around 15% pass under it (7, 27). This anatomical variation is dependent on which part of the primitive arterial circle persists throughout embryological development. The third segment extends from the point where the artery bends again to its terminal point.

In the intraorbital segment, the ophthalmic artery passes medially to the optic nerve and reaches the supero-medial corner of the orbital aperture. After bending (at the end of the second segment), the artery progresses between the medial rectus and superior oblique muscles to reach the medial wall of the orbit near the anterior ethmoidal foramina. At the supero-medial corner of the orbit, the ophthalmic artery bifurcates, giving rise to its terminal branches, namely the supraorbital artery and the dorsal nasal artery, in approximately 83% of cases (27). In other cases, the ophthalmic artery terminates at the level of the anterior ethmoidal foramina, giving rise to another small branch reaching the supero-medial corner of the orbit (7).

4.3. Variations of the Branches of the Ophthalmic Artery

The branches of the ophthalmic artery can be divided into two groups: orbital and ocular. Several variations in the origins of these branches have been identified.

The first two branches of the ophthalmic artery, the central retinal artery and the medial posterior ciliary artery, may arise from a common trunk or have different origins. (27).

Whether the ophthalmic artery passes over or under the optic nerve in its second segment creates a significant difference in the arrangement of its branches. When passing under the optic nerve, the first branch is always the lateral posterior ciliary artery, followed by the central retinal

artery, and then the medial posterior ciliary artery, with other branches following suit. If the ophthalmic artery passes over the optic nerve, the first branch arising from a common trunk with the medial posterior ciliary artery is the central retinal artery, followed by the lateral posterior ciliary artery (7).

4.4 Possible Anastomoses Between the Branches of the Ophthalmic Artery and External Carotid Artery

The ophthalmic artery, belonging to the internal carotid artery, forms numerous anastomoses with branches of the external carotid artery (7). Understanding these natural anastomoses is crucial.

An anastomosis between the lacrimal artery and the anterior branch of the middle meningeal artery, passing through the superior orbital fissure or Hyrtl canal, has been embryologically explained. Further distally, the lacrimal artery may anastomose with the deep temporal artery, transverse facial artery, zygomaticoorbital artery, and infraorbital artery (4, 7).

The dorsal nasal artery may anastomose with the facial artery or infraorbital artery via the angular artery (4). An anastomosis between the dorsal nasal arteries on both sides of the midline may be present (7).

The supraorbital artery and rarely the supratrochlear artery may anastomose with the anterior branches of the superficial temporal artery (7).

5. Clinical Significance of the Ophthalmic Artery

Knowledge of the anatomical variations and potential origins of the ophthalmic artery can be crucial for both neuroradiologists and neurosurgeons in the management of the following pathologies.

5.1. Cribriform Plate dAVF

Cribriform plate dAVFs are primarily supplied by branches of the ophthalmic artery, specifically the ethmoidal arteries. Additionally, they can be supplied by branches of the middle meningeal artery and IMA. The previously described anastomoses between the ophthalmic artery and external carotid artery, even if not always fed by branches of the middle meningeal artery or IMA, explain the possibility of visualizing the fistula after injection from an external carotid artery. A bilateral anastomosis between the dorsal nasal arteries on both sides may explain frequent bilateral feeding of the fistula. In cases of surgical exclusion, knowledge of the anatomy of the ophthalmic artery, including possible variants, is essential to identify the AV shunt and avoid complications.

Several studies have also shown the possibility of managing this pathology with an endovascular approach. Knowledge of the anatomy of the ophthalmic artery is required to select the best injection point (either from the middle meningeal artery or the ophthalmic artery) and limit the risk of arterial reflux into ocular branches (28).

5.2. Sphenoid Wing dAVF

For sphenoid wing dAVF, the inferolateral trunk and branches of the middle meningeal artery and IMA are generally the main arterial feeders. The ophthalmic artery may also play a significant role through its dural recurrent branches. In order to choose the best approach (microsurgical exclusion or endovascular occlusion) and avoid complications, in-depth knowledge of the possible anastomoses between the dural areas of the ophthalmic artery and the middle meningeal artery is required (29).

5.3. Carotid-Ophthalmic Aneurysm

The treatment of these aneurysms includes various possibilities such as surgical clipping and endovascular treatment with simple coiling or flow-diverting stents. In cases of stenting treatment, occlusion or emboli of the ophthalmic artery may occur. When surgically clipping a carotid-ophthalmic aneurysm, great attention should be paid to possible variations in the origin and course of the ophthalmic artery during anterior clinoid process drilling. Before performing anterior clinoidectomy, consideration should be given to the origin of the ophthalmic artery associated with superficial and deep dural rings, double ophthalmic arteries, and other variations (5, 30).

5.4. Olfactory Groove Meningioma

The vascular supply of olfactory groove meningiomas mainly originates from the ethmoidal branches of the ophthalmic artery. Preoperative embolization of this source carries a risk of affecting the ocular branches of the ophthalmic artery and is therefore generally not performed. Although some reports describe superselective catheterization of the ophthalmic artery, preoperative embolization is usually performed via the middle meningeal artery due to the low risk of complications. The most important rule is to place the microcatheter tip as distally as possible. Analysis of the anatomy, origins, and final anatomical variations of the ophthalmic artery is also important. In the case of surgical approach, the first step of resection is to coagulate the feeding vessels on the skull base to limit blood loss (31).

5.5. Resistant and Severe Epistaxis

Resistant or severe nasal bleeding is a good indicator for embolization via branches of the internal maxillary artery. Before performing this treatment, thorough analysis and knowledge of the anastomotic connections between the branches of the sphenopalatine artery and other branches (middle meningeal artery, ethmoidal artery) are essential to avoid uncontrolled embolization. These complications occur in 0-2% of cases and should be considered before the procedure. In rare cases, the ethmoidal arteries are the source of bleeding, and the preferred treatment is usually surgical ligation (32).

5.6. Embolization of Facial Tumors

Although epistaxis is often idiopathic, some cases may be due to neoplastic erosion of vascular structures or tumor necrosis post-treatment. In these cases, symptoms can also lead to hemoptysis due to the frequent nasopharyngeal location of these tumors. Endovascular treatment should be considered for uncontrollable epistaxis or hemoptysis. Preoperative embolization can be used as a preoperative procedure to reduce intraoperative blood loss in benign tumors. In these cases, embolization of the sphenopalatine artery may be insufficient, and embolization of the facial artery and ascending pharyngeal artery is required for devascularization. Neuro-radiologists should pay attention to the anastomoses between the facial artery and dorsal nasal artery for these pathologies. For these pathologies, neuro-radiologists should pay attention to variations to prevent complications arising from anastomoses between the internal carotid artery and external carotid artery (6, 28).

5.7. Intra-arterial Chemotherapy for Retinoblastoma

Retinoblastoma is a childhood ocular tumor. One treatment method is intra-arterial chemotherapy (33). The classical technique involves superselective catheterization of the ophthalmic artery with a microcatheter and subsequent chemotherapy injection. However, direct catheterization of the artery is not always possible due to its small size or anatomical variations, especially in small children, rendering standard technique catheterization impossible. Alternative approaches to indirectly accessing the ophthalmic artery have been described. Specifically, injection can be performed by cannulating the meningo-lacrimal branch via the anterior part of the middle meningeal artery (34).

5.8. Filler Injections

Filler injection is a popular cosmetic procedure, but it can lead to vascular complications. Filler injection around the periorbital region has a high complication rate affecting the glabella (38.8%), nose (25.5%), and forehead (12.2%) and is the highest risk area among all injection sites.

Vessels around the periorbital region are connected to the internal carotid artery system via the ophthalmic artery and the external carotid artery system via the facial artery and superficial temporal artery. Therefore, filler injection into these vessels can lead to serious complications such as blindness and cerebral embolism. Therefore, a comprehensive understanding of arterial variations around the periorbital region is essential for patients undergoing facial filler injections (5).

6. Conclusion

Understanding the embryological development and possible variants in the adult configuration of the ophthalmic artery is highly beneficial. Consequently, knowledge of the anatomy of the ophthalmic artery and its possible variations is required for both neurosurgeons and neuroradiologists approaching previously described pathologies.

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CHAPTER XIV

ANATOMY AND CLINICAL SIGNIFICANCE OF INTERNAL THORACIC ARTERY

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1. Introduction

The circulatory system, which sends blood to all parts of the body, consists of the heart, arteries, veins, and capillaries. The arterial system begins with the aorta, which emerges from the left ventricle of the heart (1, 2).

One of the fundamental principles of circulation is that each tissue determines its own blood flow according to its metabolic needs. The main functions of circulation include transporting oxygen, glucose, amino acids, fatty acids, various hormones, and specific molecules to tissues, removing carbon dioxide and hydrogen ions from tissues, and maintaining the balance of other ion concentrations in tissues (3).

Arteries branch out like the roots of a tree, with their diameters decreasing as they branch. Eventually, they turn into small vessels called arterioles. Arterioles, in turn, transform into microscopic networks of vessels with very thin walls, known as capillaries. Arteries are the vessels that carry oxygenated blood pumped by the heart to all parts of the body. Arteries have three types: arterioles, muscular arteries, and elastic arteries (1, 2).

The internal thoracic artery (ITA) is one of the muscular arteries and is a branch of the subclavian artery. In recent years, due to its anatomical position, the ITA has gained importance in thoracic surgery and coronary revascularization. When looking at postoperative mortality and morbidity in coronary bypass surgery, ITA grafts are among the most commonly used. ITAs with appropriate sizes are suitable recipient vessels for free tissue transfers, especially in breast

reconstruction. Therefore, the artery has attracted considerable attention from surgeons in recent years (4).

In anatomical studies, the difficulty in dissecting thin vessels explains the challenge in identifying the ITA. However, it is a vessel that can be easily identified based on its location. The difficulty in cannulating the fine-caliber ITA in angiographic studies is not a significant limitation. This area is also used for subclavian vein catheterizations. Additionally, it is important for percutaneous transthoracic procedures such as lung needle biopsy. Although most of these procedures are performed safely, complications have been reported. Therefore, it is crucial to be aware of all anatomical details such as the origin, branching pattern, course, and variations in diameter of the ITA (5).

2. Histology of Arteries

Arteries are a series of vessels that carry blood from the heart to tissues and repeatedly branch along their course. Their function is to deliver oxygen, nutrients, and other necessary substances to tissues. As they approach organs, their diameters decrease and they reach large numbers (6).

2.1. Layers of Arteries

The arterial wall consists of three layers: the tunica intima, tunica media, and tunica adventitia (6).

The tunica intima is the innermost layer of the vessel. It consists of three components: the endothelium, which is a single layer of flat epithelial cells, the subendothelial layer composed of the basement membrane of endothelial cells and loose connective tissue. The intima layer, called the internal elastic lamina, is made of elastin and contains pores that allow better diffusion of substances from the blood to the depths of the wall (6).

The tunica media, or middle layer, primarily consists of circularly arranged smooth muscle cells. This layer is relatively thick in arteries and may have a thin external elastic lamina that separates it from the outermost layer (7).

The tunica adventitia, the outermost layer of connective tissue, consists mainly of longitudinally arranged collagenous tissue and a small amount of elastic fibers. These connective tissue elements gradually merge with the loose connective tissue surrounding the vessels. While the tunica adventitia is relatively thin in most of the arterial system, it becomes significantly thickened in venules and veins. Additionally, the tunica adventitia of large arteries and veins contains an autonomic nerve network that controls smooth muscle contraction

in the vessel wall, as well as a vascular system called the vasa vasorum, which nourishes the vessel wall itself (7).

2.2. Classification of Arteries

For instructional purposes, arteries are classified into three groups: large (elastic) arteries, medium-sized (muscular) arteries, and small arteries (arterioles) (6).

Elastic or conducting arteries

These are large vessels that carry blood from the heart to muscular arteries. Examples include the aorta and the pulmonary trunk, which carry blood from the heart to the systemic and pulmonary circulations, respectively (6).

The main branches of these arteries, such as the brachiocephalic trunk, common carotid artery, subclavian artery, and common iliac artery, are classified as elastic arteries (6).

Muscular or distributing arteries

These arteries distribute blood to organs and help regulate blood pressure by contracting or relaxing the smooth muscles in the tunica media. These arteries comprise the majority of the arterial system. Examples include the coronary arteries, brachial artery, radial artery, femoral artery, popliteal artery, and internal thoracic artery (6).

Small arteries and arterioles

They are distinguished based on the number of layers of smooth muscle in the tunica media. Arterioles have only one or two layers in the tunica media, while small arteries have up to eight layers in the tunica media (6).

3. Anatomy of the Internal Thoracic Artery

The internal thoracic artery (ITA) is the first inferior branch of the subclavian artery. This artery arises approximately 2 cm above the articulation of the sternoclavicular joint, immediately opposite the origin of the thyrocervical trunk, and separates from the first part of the subclavian artery. It runs downward behind the lateral border of the sternum, passing behind the brachiocephalic vein and extending downward on the posterior surface of the first six costal cartilages (1, 4).

3.1. Branches of the Internal Thoracic Artery

During its course, the artery gives off branches such as the pericardiacophrenic artery, mediastinal branches, thymic branches, sternal branches, anterior intercostal branches, and perforating branches. It terminates by dividing into two terminal branches called the musculophrenic artery and the superior epigastric artery at the level of the sixth intercostal space (1, 8).

3.1.1. Pericardiacophrenic Artery

It arises from the internal thoracic artery at the level of the first costal cartilage. It extends along with the phrenic nerve on the sides of the heart, between the pleura and the pericardium, reaching the diaphragm. During its course, it gives off fine branches to adjacent structures. It terminates by anastomosing with the musculophrenic artery and the inferior phrenic artery on the diaphragm (1, 8).

3.1.2. Mediastinal Branches

These branches are small twigs that distribute in the structures of the anterior mediastinum. These small branches provide nourishing branches to the pericardium, pleura, thymus, loose connective tissue, lymph nodes, and the transversus thoracis muscle. Thin branches that penetrate deeply follow the bronchi into the lungs (8).

3.1.3. Thymic Branches

These are thin branches that wait for remnants of the thymus (1, 2).

3.1.4. Sternal Branches

This branch extends behind the sternum and supplies the transversus thoracis muscle. The mediastinal branches, sternal branches, fine branches to the pericardium, and some branches of the pericardiacophrenic artery, along with the branches of the anterior intercostal artery and the bronchial artery, form a subpleural mediastinal plexus by anastomosing (1).

3.1.5. Anterior Intercostal Branches

They are located at the lower edge of the ribs of the first 5 or 6 intercostal spaces. The remaining intercostal spaces contain branches of the musculophrenic artery. These small arteries, which are thin, extend laterally along the lower edge of the upper rib in each space. These arteries that nourish the muscles in the region anastomose with the posterior intercostal arteries coming from the aorta.

Initially, the artery runs between the pleura and the internal intercostal muscle, and then between the internal intercostal and innermost intercostal muscles. The branches of arteries in the second, third, and fourth intercostal spaces penetrate the external intercostal muscle and nourish the pectoral muscles. The branches that nourish the breast are called medial mammary arteries (1, 8).

3.1.6. Perforating Branches

These branches are given off near the sternum in the first 5 or 6 intercostal spaces. They penetrate the internal intercostal muscle, external intercostal membrane, and pectoralis major muscle in the anterior chest wall. Then they turn outward and become superficial, supplying the pectoralis major muscle and the skin. Arteries in the second, third, and fourth intercostal spaces supply the breast with branches called medial mammary arteries. These branches widen significantly during lactation (1, 8).

3.1.7. Musculophrenic Artery

It is one of the two terminal branches of the internal thoracic artery. It separates from the internal thoracic artery behind the sixth costal cartilage and extends obliquely downward and outward. It pierces the diaphragm at the level of the eighth or ninth costal cartilage and terminates at the level of the last intercostal space. During its course, it gives off branches to the seventh, eighth, and ninth intercostal spaces. These branches extend like the anterior intercostal arteries derived from the internal thoracic artery and divide into branches. In addition to supplying the lower part of the pericardium, it also supplies the structures on the backside and the front side of the diaphragm, including the abdominal muscles (1, 8).

3.1.8. Superior Epigastric Artery

It is the other terminal branch of the internal thoracic artery and is seen as a continuation of the artery. It passes between the sternal and costal parts of the diaphragm and enters the sheath of the rectus abdominis muscle. Here, it initially extends along the posterior surface of the muscle, then enters its substance to supply the muscle, and anastomoses with the inferior epigastric artery (a branch of the external iliac artery). Some branches arising from this artery penetrate the vagina of the rectus abdominis muscle to distribute to other abdominal muscles and the skin. Some of the branches originating from the artery on the right side enter the liver through the ligamentum falciforme and anastomose with the branches of the hepatic artery. Through these anastomoses, collateral circulation

is established between the branches of the subclavian artery and the external iliac artery around the umbilicus (1, 8).

3.2. Neighboring Structures of the Internal Thoracic Artery

Its initial part is located behind the lateral end of the clavicle, subclavian vein, internal jugular vein, and behind the first costal cartilage. At its entrance into the thorax, it is located just lateral to the brachiocephalic vein, and the phrenic nerve crosses it from outside to inside (sometimes from behind to front). It runs almost vertically from the lower edge of the first costal cartilage to the point where it divides into terminal branches. In its course, the anterior surface is lined by the first six costal cartilages, intercostal muscles between them, intercostal nerves, and internal intercostal membranes. On the posterior surface, the pleura is present up to the third costal cartilage, and below this level, the transversus thoracis muscle (1, 2).

3.3. Variations of the Internal Thoracic Artery

Variations in the internal thoracic artery are quite rare. Karaman et al. (2012) reported that in one of 328 cases, the internal thoracic artery emerged as a common trunk with the thyrocervical trunk, and in two cases, it emerged as a common trunk with the costocervical trunk (5). In their study, Puri et al. (2007) reported that the internal thoracic artery emerged as a single main trunk with other branches of the subclavian artery in 15% of cases (9). Ekim et al. (2003) noted that in 30% of cases on the left side and 5% on the right side, the internal thoracic artery emerged as a single main trunk with another artery from the subclavian artery (10). In a study, it was reported that in 15% of cases, lateral costal arteries of the same caliber as the internal thoracic artery originated from it. The lateral costal branch is found unilaterally in 10% and bilaterally in 5% of cases (10). In the literature, it has been reported that the internal thoracic artery gives branches at different costal levels (13, 14, 15). It has been noted that in 5 of 51 patients, the internal thoracic artery terminates by giving three branches instead of two at the terminal (10). Another variation of the internal thoracic artery is its termination levels. It has been reported that the artery terminates at different levels between the 4th and 6th ribs (9).

Table 1: Comparison of origin variations of internal thoracic artery in different studies.

Authors	Materials and methods	1st part of SA	2st part of SA	3st part of SA	With thyro-cervical trunk	With costo-cervical trunk	With supra-scapular	With transverse cervical	With transverse scapular & transverse cervical	With suprascapular & superficial cervical
Karaman et al. (2012) (5)	MDCT angiography 164 patient	99,4%			0,3%	0,6%				
Vorster et al. (1998) (11)	Cadaver 60 adults			0,83%						
Delmotra et al. (2019) (16)	Cadaver 30 adults	91,7%					1,67%		3,3%	3,3%
Omar et al. (2004) (12)	Cadaver 1 adult			100%						
Uemura et al. (2010) (17)	Cadaver 56 adults				9,8%					
Henriquez-Pino et al. (2005) (18)	Cadaver 100 adult	82,5%					9,5%		3,5%	
Paraskevas et al. (2011) (19)	Cadaver 1 patient				50%					
Daseler and Anson (1959) (20)	Cadaver	79,2%	3,64%	0,78%	8,84%		3,77%	0,78%	0,28%	

Table 2: Comparison of termination patterns of internal thoracic artery in different studies.

Authors	Materials and methods	Termination pattern	Incidence
Gupta et al. (2013) (21)	Cadaver 30 adults	Trifurcation	8,3%
		Bifurcation	91,7%
Paliouras et al. (2015) (22)	Cadaver 50 adults	Trifurcation	7%
		Bifurcation	93%
Agnihotri et al. (2022) (23)	Cadaver 100 adults	Trifurcation	2%
		Bifurcation	98%
Henriquez-Pino et al. (2005) (18)	Cadaver 100 adult	Trifurcation	7%
		Bifurcation	93%

4. Clinical Significance of the Internal Thoracic Artery

Despite the widespread use of effective medical treatment for atherosclerotic risk factors, coronary artery disease remains the leading cause of mortality and morbidity in the United States and worldwide (24). The most effective treatment for coronary artery disease is coronary artery bypass surgery. Therefore, more than 800,000 people worldwide undergo coronary artery bypass surgery annually (25). The aim of coronary artery bypass surgery is to keep the vessel open for a longer period. Although good results are achieved in the short and medium term, the need for reoperation may arise in the long term. The reoperation rate is reported to be more than 3% in the first 5 years, over 10% in 10 years, and 17% in 12 years (26). Coronary reoperations constitute 15% to 20% of all isolated coronary artery bypass surgeries (26). Therefore, appropriate graft selection is crucial. Biological characteristics such as the patient's age, clinical condition, as well as the diameter, length, wall thickness, and incidence of atherosclerosis development of the graft to be used, and graft availability are determinants in graft selection. Graft insufficiencies due to stenosis or occlusion developed in grafts after bypass affect the long-term patency rates of the grafts (27).

The first graft used in coronary artery bypass surgery is the great saphenous vein. In terms of diameter, it is larger than the coronary arteries at the level of the knee joint, and distally, it is more compatible with the coronary arteries. Another characteristic of the great saphenous vein is the insufficient or absence of elastic tissue in the media layer. It is known that elastic tissue prevents intimal hyperplasia. Considering these evaluations, the segments of the great saphenous vein near the knee joint can be used as grafts for all three coronary arteries. The larger diameter of the great saphenous vein will increase blood flow. The great saphenous vein is a vein that carries blood in the opposite direction of gravity, starting from the lower parts of the body. It has approximately 12 to 20 valves in its wall. The least number of valves are found in the 1/3 middle section of the great saphenous vein. With this feature, the knee joint region of the great saphenous vein can be a preference. However, various reasons for developing varicose dilations do not allow the use of the great saphenous vein as a graft. Another disadvantage is the development of "intimal hyperplasia" in great saphenous vein grafts after surgery (28). This occurs when the natural venous structure of the great saphenous vein encounters systemic arterial pressure. It has been reported in the literature that great saphenous vein grafts may undergo intimal proliferation and have a 2% annual occlusion probability on angiography between the 1st and 7th years, while this risk reaches 5% annually between the 7th and 12th years. Accordingly, after a 10-year period, only 30-45% of

great saphenous veins have been found to remain open and exhibit a normal appearance (29).

Another vessel used as a graft in coronary artery surgery is the radial artery. After identifying the disadvantages of the great saphenous vein, attention turned to arteries with similar structures to coronary arteries. Carpentier et al. first demonstrated the use of the radial artery as a graft in myocardial revascularization. Some researchers have recommended the use of the radial artery in coronary bypass operations. This is because these researchers followed some patients' grafts for up to 18 years after initial revascularization and achieved quite satisfactory results. With the introduction of new antispasmodic agents and the development of minimally traumatic harvesting techniques, the use of the radial artery as a graft in coronary artery surgery has become popular. The radial artery is considered a suitable graft due to its sufficient length, more regular lumen compared to the great saphenous vein graft, more suitable diameter for coronary arteries, and rare susceptibility to atherosclerosis. Radial artery dissection is relatively easy, its width and length are quite good, and mid-term results are good. However, the use of the radial artery in coronary bypass surgery has been disappointing due to the initial high perioperative spasm rate and graft occlusion (26).

The internal mammary artery (IMA), known clinically as the thoracic internal artery, was first used in coronary artery surgery in 1968. One of the most important reasons for its preference is the significantly fewer postoperative complications (30). Among alternative grafts, the internal mammary artery has become the standard graft after the advantages were established. In long-term follow-ups, the superior patency rate of the internal mammary artery has also encouraged more distal anastomoses in coronary bypass surgery applications. Because the aim in coronary bypass surgery is to create a graft with long-term patency. As a result, in cases where a high late patency rate is achieved using the internal mammary artery, life expectancy is extended, quality of life improves, and the rate of reoperation or invasive cardiac interventions decreases (31).

Histologically, the internal mammary artery contains a small number of smooth muscle cells and is predominantly elastic in structure. The media, originally composed of elastic lamellae, begins to acquire muscular characteristics in the distal segment and continues as two branches with more muscular properties, the musculophrenic artery and the superior epigastric artery. The amount of elastic lamellae in the media layer influences the degree of intimal hyperplasia. The intima is significantly thicker in the muscular segment compared to the elastic segment, and elastic arteries are more resistant to intimal

hyperplasia than muscular arteries. Damage that may occur in the radial artery during the preparation of arterial grafts is much greater than that in the internal mammary artery, resulting in increased intimal thickening. This situation raises concerns about possible occlusions in the radial artery in the late period. This possibility is lower in the internal mammary artery (27).

In the literature, it has been reported that the internal thoracic artery endothelium produces certain secretions such as nitric oxide and prostacyclin. The endothelium of the internal thoracic artery spontaneously secretes significantly higher amounts of nitric oxide compared to the vena saphena magna. Nitric oxide is a potent vasodilator. Therefore, it inhibits platelet adhesion and aggregation and plays a significant role in keeping the vessel open. Prostacyclin is another mediator in endothelial cells and induces inhibition of platelet activation. The internal thoracic artery secretes prostacyclin in much larger amounts compared to the vena saphena magna. One of the important factors that enables the internal thoracic artery to remain open for a longer period compared to the vena saphena magna is also the secretion of prostacyclin (27).

In practice, the internal mammary artery (IMA) graft exhibits regular, parabolic, laminar flow characteristics. In the internal mammary artery not used as a graft, flow is dominantly systolic proximally and dominantly diastolic distally. On the other hand, the great saphenous vein demonstrates diastolic flow characteristics throughout its entire length. Due to the flow characteristics of the internal mammary artery, the wall shear stress is higher compared to the great saphenous vein. High wall shear stress stimulates endothelial response, resists neutrophil adhesion, and inhibits smooth muscle cell proliferation. Decreased wall shear stress leads to arterial diameter reduction and the development of intimal hyperplasia and atherosclerosis. These are some of the reasons explaining the long-term patency of internal mammary artery grafts (27).

Another advantage of internal mammary artery grafts is their potential for in-situ applications due to their anatomical location. In-situ grafting involves detaching the distal portion of arteries near the aorta along with the surrounding tissue without separating the proximal portions and performing an anastomosis to the coronary artery distal to the occlusion. Studies have reported higher success rates for in-situ internal mammary artery grafts compared to free grafts. When the internal mammary artery is used as a free graft, a second anastomosis is performed between the proximal portion of this artery and the aorta. Intimal damage occurring during proximal anastomosis reduces the success of the graft. However, the length of the internal mammary artery may hinder its reaching the coronary arteries on the posterior surface of the heart as an in-situ graft.

Although literature provides information on anatomical details and variations, existing knowledge is within broad limits. Obtaining more specific information will increase the chances of success (5).

The internal mammary artery (IMA) has been regarded as an ideal graft due to reasons such as adequate length and diameter, thin wall, exhibiting characteristics of an elastic artery, not requiring a second surgery, and being suitable for both free graft and in-situ usage (28, 32, 33, 34).

Because of its anatomical position, the internal mammary artery holds significance in both breast reconstruction and myocardial revascularization (35).

However, despite its advantages in achieving myocardial revascularization, a higher incidence of postoperative infections has been reported. Additionally, cases involving bilateral internal mammary arteries have been associated with a higher likelihood of partial and transient sternal devascularization compared to cases involving a single internal mammary artery (30).

5. Conclusion

Due to its anatomical position, the internal thoracic artery holds significant importance in both breast reconstruction and myocardial revascularization. The internal thoracic artery has been considered an ideal graft for several reasons, including sufficient length and diameter, thin wall, exhibiting characteristics of an elastic artery, not requiring a second surgery, and being suitable for both free graft and in-situ usage. One of the most important reasons for its preference is the significantly low incidence of postoperative complications. Understanding all variations in the origin, branching pattern, course, and diameter of the internal thoracic artery becomes crucial to minimize both intraoperative and postoperative complications.

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CHAPTER XV

ANATOMY, FUNCTIONS AND CLINICAL SYNDROMES OF THE BASAL NUCLEI

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1.Introduction

The basal nuclei(also known as the Basal Ganglia) are five pairs of grey matter masses located deep in the telencephalon. Like mental and emotional state, they are related to routine behaviour and voluntary motor control. They organise planned rough movements. They suppress involuntary muscular activity The basal nuclei consisting of nucleus caudatus, globus pallidus, nucleus subthalamicus, substantia nigra and putamen are located subcortically. Nucleus caudatus and putamen together form the striatal group; globus pallidus, nuc. subthalamicus and substantia nigra together form the nuclear group. The nuclear group usually plays a more intrinsic and regulatory role. They regulate broader motor functions such as inhibition and promotion of movement, motor learning and co-ordination. These groups work together as basal ganglia and play an integrated role in movement control and other cognitive functions. The striatal group, also known as the striatum, is particularly involved in integrating and processing cortical inputs. They play an important role in motor control and influence the planning and execution of movements. Putamen and globus pallidus together form the nucleus lentiformis.

1.1 Caudate Nucleus

It is a C-shaped curved structure located on the sides of the brain. It looks like a comma or comet and consists of three parts: caput, corpus and cauda. It plays a role especially in movement control and learning processes.

1.2. Globus Pallidus

It is a structure located below the nucleus caudatus. It plays an important role in initiating and stopping movement. It is divided into two parts by a lamina formed by myelinated axons. The medial one is called globus pallidus medialis and the lateral one is called globus pallidus lateralis.

1.3. Subthalamic Nucleus

This structure is a nucleus located below the thalamus and is associated with motor control. It plays an important role in the co-ordination of movement.

1.14. Substantia Nigra

It is an important dopaminergic nucleus in the brain. It is effective on movement control, reward mechanism and learning. Substania nigra is seen darker in the mesencephalon in horizantal head sections. This is due to the melanin pigment in its structure. It has two parts, pars compacta and pars reticularis. Pars reticularis consists of gamma amino butyric acid (GABA) containing neurons and goes to the thalamus. The pars compacta, composed of dopamine-containing neurons, is a component of the dopaminergic system and the fibres (efferent) from this part terminate in the neostriatum.

1.5. Putamen

It is a structure located next to the globus pallidus and is important in the regulation of motor control. This shell-shaped nucleus is a basal nucleus located on the lower outer side of the nucleus caudatus. It is especially effective in the regulation of voluntary movements. It is the nucleus mainly involved in passive and active movements.

These structures play important roles in the planning, co-ordination and control of voluntary movements. They also have functions such as suppressing involuntary muscle activity, smoothing movements and contributing to motor learning processes. Some neurological disorders such as Parkinson’s disease occur due to functional disorders of these structures [1,2,3].

2. Functions of the Basal Nuclei

The basal nuclei are structures that function mentally in every direction at every moment. It acts as a kind of filter with the do-not-do code in all our voluntary movements or in a concrete action.

Functional learning, such as learning a new language, learning to drive a car, sewing or It assumes the role of manager in the maintenance of acquired skills. In other words, the subconscious performs all learnt actions automatically.

Basal nuclei play important cognitive and sensory roles along with motor movements. Basal nuclei are related to limbic system and reward mechanism. Although there is no specialised nucleus in the reward mechanism, the activity of doaminergic pathways increases in the basal nuclei.

The basal ganglia play an important role in the regulation of motor control, a complex system of neural circuitry in the brain. These structures are involved in functions such as initiating, stopping and regulating motor movements, as well as emotional functions. The function of the basal ganglia is provided by complex networks of connections. These connections regulate the communication and processing between inputs and outputs. [4,5]

3. Circuit Connections

Cortical Inputs: The most important inputs to the basal ganglia come from various regions of the cerebral cortex called the cortex. Cortical inputs come from brain regions such as the motor cortex and prefrontal cortex and provide information related to the planning and execution of voluntary motor movements.

3.1 Inputs (Afferents)

Almost all afferents entering the basal nuclei terminate in the striatum. These inputs come from sources such as the limbic system, cerebral cortex and thalamus.

The striatum receives and processes these inputs. The striatum receives inputs to the neostriatum (putamen and caudate nucleus) from all sources. The cerebral cortex is the source of these inputs and does not receive any input from the spinal cord.

3.2 Outputs (Efferents)

The outputs of the basal nuclei communicate with specific regions of the thalamus.Outgoing efferents start from the pars reticularis of the substania nigra.

They then reach specialised regions of the thalamus and finally return to the cerebral cortex. These outputs send excitatory impulses to areas of the brain that show motor function.

3.3 Input-Output Loop

The cerebral cortex brings impulses to the striatum via appropriate afferents.The striatum processes these impulses and delivers them to the globus pallidus and specialised segments of the substania nigra.

Outgoing efferents start from the pars reticularis of the substania nigra and reach specific regions of the thalamus. Finally, they return to the cerebral cortex, where they reach motor and prefrontal areas.

This input-output loop plays a critical role in the regulation of voluntary motor control and the management of emotional functions.

3.4 Direct Connections

Direct connections between the cortex and the striatum are excitatory neurones that enhance the function of the cortex.

They start from the cortex by reaching inhibitory neurons in the striatum and then inhibit the globus pallidus and substania nigra. These inhibitions reduce the activity of the thalamus and consequently prevent impulses from travelling to the cortex.

3.5 Indirect Connections

Indirect connections are inhibitory neurones that reduce the excitation of the cortex.

These connections traverse the pathways from the cortex to the striatum and then inhibit the globus pallidus and substania nigra. As a result of these inhibitions, the activity of the thalamus is reduced and impulses are prevented from travelling to the cortex.

The balance and harmony between these connections regulate the motor and cognitive functions of the brain. However, disturbances in any of these pathways can lead to disruption of balance and various diseases. For example, various diseases such as Parkinson’s, Huntington’s, Chorea, Psychosis, Dystonia, Wilson’s disease, autism, tics and Tourette Syndrome are caused by impaired functions of the basal ganglia. Various symptoms of these diseases are the result of disruption of connections in the basal ganglia and their function [6-8].

4. Dysfunction and Clinical Syndromes of the Basal Ganglia

4.1 Parkinson's Disease

Parkinson's disease is a neurodegenerative disorder caused by disturbances in dopamine production in the basal ganglia and characterised by symptoms such as tremors, muscle stiffness, slowed movement and balance problems. This syndrome is associated with reduced dopamine production, particularly in a region of the basal nuclei called the substansia nigra.

4.2 Huntington's Disease

It is a genetic disorder with a dominant inheritance. It leads to degeneration and atrophy, particularly in another region of the basal nucleus called the striatum. Huntington's disease is characterised by various symptoms such as movement disorders, cognitive dysfunctions and psychiatric symptoms.

4.3 Tourette's Syndrome

Tourette syndrome is a neurological disorder that usually begins in childhood and is characterised by motor and vocal tics, such as repetitive tics, vocal tics and sometimes obsessive-compulsive symptoms. This syndrome is believed to be related to dopamine dysregulation in the basal ganglia. Tourette syndrome is a neurological disorder in which motor and vocal tics typically occur. Motor tics involve involuntary and repetitive muscle movements or behaviours, while vocal tics involve involuntary and repetitive vocal sounds or words.

Vocal tics can take the form of sounds that involuntarily come out of a person's mouth, muffled sounds, wheezes, coughs or sometimes involuntarily repeated words or phrases. These sounds can sometimes be strange, repetitive or disturbing. For example, some people may experience vocal tics, such as involuntarily coughing, making muffled sounds, or involuntarily saying certain repetitive words or phrases. Vocal tics can be a prominent feature of Tourette's syndrome, but may differ for each Tourette's patient. In some people, vocal tics may be more prominent, while in others they may be less prominent or rare. The severity and type of these tics may vary from person to person and may change over time.

4.4 Hemibalismus Syndrome

Hemibalismus is a rare movement disorder resulting from lesions of the basal gangli-a. It is usually associated with lesions of the subthalamic nucleus

and is characterised by large movements that occur suddenly, usually affecting the body in a single half [4,9].

5. Conclusion

In conclusion, the basal nuclei, important grey matter masses in the brain, play significant roles in the planning, coordination, and control of voluntary movements. Additionally, they are involved in regulating emotional functions and learning processes. The function of basal ganglia is mediated through complex neural circuits, spanning from cortical inputs to outputs. These connections play a critical role in balancing motor and cognitive functions, with any disruption potentially leading to various diseases. Disorders such as Parkinson's disease, Huntington's disease, and Tourette syndrome are clinical syndromes arising from disruptions in the functions of basal ganglia. These syndromes are typically characterized by symptoms such as movement disorders, cognitive impairments, and psychiatric symptoms. Therefore, further research into the functions and connections of basal nuclei will deepen our understanding of these crucial brain structures and contribute to the treatment and management of neurological diseases.

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ISBN 978-2-38236-662-2