GENERAL SURGERY EMERGENCIES

Editor
Vahit Mutlu

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PREFACE

In the field of medicine, diagnosis and treatment methods are updated and developed day by day thanks to technological innovations. For this reason, we need to follow up-to-date information in the field of medicine and apply it in surgical clinics.

Under the guidance of innovative and up-to-date approaches, minimally invasive techniques have started to be used more frequently in emergencies just as in elective situations. Thus, a shorter and more comfortable hospitalization process, faster diagnosis and more accurate treatment are provided for the patients.

Every general surgeon should know the emergency situations and the details regarding general surgery very well, and these situations are of vital importance. In this regard, we were motivated to write the book "general surgical emergencies" from a more specific and new perspective, using up-to-date information, techniques and practices.

Evaluation, diagnosis and treatment of patients who apply to the emergency department or polyclinics with complex situations should be proceeded from all aspects. The multiplicity of differential diagnoses, the severity of the patient's complaints, the hospital facilities and the surgeon's knowledge and skills are the determining factors of this process. Such emergencies apply to almost every general surgery clinic. The management of the current situation covers a long process from the patient's anamnesis to discharge.

The general surgical emergencies discussed in this book form the basis of many surgical practices. Therefore, we believe that this book will be useful in updating our knowledge and bringing a new dimension to our surgical approaches. The chapters of this book have been systematically addressed and will be a useful guide in the management of general surgery emergencies.

As the editor of the book, I would like to thank all my physician friends who worked in busy clinics and contributed to this book, my teacher Cem Melik for their most valuable support, and all the team.

CONTENTS

CHAPTER I

ETIOLOGY OF ACUTE ABDOMEN

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1. Introduction

Abdominal pain is one of the most common complaints that individuals face several times in their lives throughout human history. When we say acute abdomen, pathologies that manifest themselves with pain in the abdominal region and are usually independent of causes such as trauma should come to mind. When we grade pain, it can range from mild to severe, resulting in medical or surgical treatment. In such cases, the duration and quality of the approach to the patient are of great importance. Ultimately, abdominal pain is one of the most preventable and treatable symptoms. Aside from the possibilities offered by technology towards the end of the century, anamnesis, which is still a classic in diagnosis and treatment, continues its importance. And as a result of these, the opinion of the physician will reveal a preliminary diagnosis, and then a conclusion will be reached with supportive laboratory and imaging procedures that will lead to the diagnosis.

A comprehensive examination and correct diagnosis of the patients who apply to the emergency services due to abdominal pain will both prevent unnecessary interventions and prevent the patient from being harmed. Even if the physician approaches here make a difference, every physician who has basic knowledge will make a diagnosis to a large extent.

2. Etiology

Although there are many diseases that come to mind when the etiology of acute abdomen is mentioned, acute appendicitis, acute cholecystitis, acute pancreatitis, peptic ulcer perforation, acute diverticulitis, intestinal obstructions, strangulated hernias, volvulus, peritonitis, etc. are encountered in surgical

emergencies. In addition, pelvic inflammatory diseases, tuba ovarian abscesses, ovarian cyst ruptures, etc. are seen in gynecological emergencies, as well as urinary tract infections and stones, and can be counted among the causes of acute abdomen in urological emergencies such as testicular torsion.

3. Diagnosis

There must be a way that the surgeon will adopt as an approach. Is the patient's pain acute? Or is it chronic? It must be defined. Afterwards, if it is acute, the etiology has been revealed, and if the diagnosis is made after the necessary tests are performed, it should be decided whether surgical intervention is required. Although there are many approaches, surgical intervention should be avoided if medical treatment will be beneficial to the patient.

When we look at the diseases that will create the picture of acute abdomen, physiological changes cause the same diseases to appear with different symptoms in patients. The fact that human physiology creates some different effects on itself in the body can cause our complex body to be diagnosed in a short time with a set of simple symptoms and clues. For this reason, being able to make an early diagnosis always strengthens the hand of the physician and causes us to get beneficial results for the patient. After taking a good anamnesis, it should be accepted as a classical approach that the physical examination should be done properly, otherwise, we may wander in unnecessary and wrong places by being condemned to laboratory and imaging.

4. Reasons

Inflammation is the most common cause of acute abdomen. And when we look at the following situations, occlusive causes, bleeding, ischemic tables can be counted. They can also occur simultaneously. For example, an occluded inguinal hernia will cause ischemia, and as a result, an inflammation picture will appear. For this reason, it should be kept in mind that it is necessary to approach the patient from different angles, not just from a single window. While an inflammatory picture emerges gradually increasing its effect, perforation and ischemic tables reveal their effects in a short time. If these conditions to be kept in mind are kept in mind in the approach to the patient, ultimately the winner will be the physician and the patient.

Considering the patients who applied to the hospitals, the socio-cultural and generational differences affect the approach to the patients significantly.

Identifying the underlying errors and different approaches and tendencies in self-expression are often one of the situations that weaken the hands of physicians. Here, it is very important that the doctor-patient relationship is calm, reassuring and at the same time understanding each other in order to be able to go towards the diagnosis. Undoubtedly, the art of medicine is practiced in this way.

While taking anamnesis, the person's age, gender, educational status, intellectual structure, profession and lifestyle should be examined very well. It should be questioned whether the patient is constantly taking any medication or whether he has a chronic disease. It should be kept in mind that a person using anticoagulants will have a hematoma, and a person using oral contraceptives will have a haptic adenoma and related rupture. The family history of the patients should be questioned. People with a family history may have similar complaints. Familial Mediterranean Fever should be considered in abdominal pain occurring at regular intervals.

In previous abdominal surgery, the cause of the obstruction is most likely an adhesion. Again, the travels of the patient should be questioned. It will give an idea in terms of explaining the underlying infectious tables.

The most important symptom in acute abdomen is abdominal pain. When this pain is questioned, it is important how it started and what kind of character it acquired. If there are accompanying gastrointestinal complaints, such as nausea, vomiting, loss of appetite, gas and inability to pass stool should be investigated.

At the stage of revealing the clinical picture, the onset, development and characteristics of the pain must be revealed, since the patient's application will usually be abdominal pain in the foreground.

It should be revealed whether there is a visceral pain caused by the irritation of hollow lumen organs or a parietal pain originating from somatic nerves on the basis of the pain.

Embryological development of intra-abdominal organs plays an important role in the spread of visceral pain. bowel. Since the liver and pancreas are in the midline of the embryo, the pain is in the midline. Segmental development of organs constitutes the second important part in the spread of pain. Therefore, foregut lesions cause pain in the epigastrium, midgut lesions in the umbilical region, and hindgut lesions in the hypogastric region.

Distension, contraction, ischemia and inflammation in the organ wall cause visceral pain. They are usually slowly increasing, blunt and not well localized pains.

Parietal pain is caused by irritation of the parietal peritoneum innervated by the somatic nerves. They are transmitted very rapidly by both the C and myelinated A fibers of these nerves. These are transmitted unilaterally in the spinal cord and their localization is much better. Direct contact of the parietal peritoneum with pus, bile, urine, and luminous organ contents may reveal pain.

5. Features of Pain

The characteristic features of pain in a patient presenting with acute abdomen are important in defining the disease. These features are; reflection, diffusion, progression, violence and displacement.

To explain in detail; Pain may be reflected in a different area from the organ where the disease is present. Reflection pain occurs with wide junctional connections formed by the afferent fibers in the posterior horn of the spinal cord. A typical example of this is pain in the shoulder ipsilaterally due to subdiaphragmatic irritation (Kehr's sign). This occurs over the phrenic nerve. Again, biliary and biliary tract pains are reflected to the right scapula. It has different innervations in kidney, ureter and bladder. The celiac, thoracic, lumbar splanchnic nerves, inter mesenteric plexus, superior hypogastric plexus innervate the kidneys and upper ureters. The lower ureters, bladder, and testicles are innervated by the pelvic autonomic ganglia. For this reason, renal colic pain can be reflected to the inguinal regions, testis and vulva.

Acute appendicitis is an example of radiating pain. First of all, the pain that starts in the epigastric region will be a visceral pain in the periumbilical region, and then it will turn into a sharp parietal pain that sits in the right lower quadrant.

The onset of pain will also indicate the severity of the disease. Pain that starts in a short time and is of high intensity should suggest a perforation, rupture of aneurysm, ectopic pregnancy, or rupture of an abscess. Increasing pain within a few hours will suggest acute cholecystitis, acute pancreatitis, ileus, mesenteric ischemia.

A slowly progressing pain, on the other hand, reflects a situation that the body is trying to control. Plastron appendicitis, duodenal perforation turning into closed and limited can be given as examples.

The severity of pain is significant in terms of the process. Constantly increasing and persistent pain is the common form. While sharp and specific pain occurs in peritoneal irritation, inflammatory and ischemic pathologies are effective in both pain types, while colic pains are more common in obstructive

pathologies. Small bowel obstructions, ureteral stones, intestinal pathologies such as volvulus can be given as examples.

Blunt abdominal pain in peptic ulcers, stinging pain in acu pancreatitis, peptic ulcer perforation, mesenteric thrombosis, burning-predatory pain in aneurysm ruptures are terms that can be used. Of course, this type of pain may differ from person to person, but in general, they appear in this way.

Apart from the displacement of the pain, the patient's positioning in different positions due to pain also gives an idea to the physician. Especially in peritoneal irritation, breathing and walking increase the pain, so these patients try to reduce the pain by taking tiny breaths without moving. Pain in the shoulder when lying down is encountered in diaphragm irritation. Again, pancreatitis patients try to reduce the pain more in the forward sitting position. In patients presenting with renal colic, they are usually on the move because the relief is not positional.

Apart from pain, patients also come with a number of different complaints. Nausea and vomiting are among these complaints. Peritoneal and mesenteric nerve irritation as well as obstruction of channels containing smooth muscle cause these complaints. The stimulated visceral afferent nerves irritate the medullary vomiting center and vomiting is observed as a reflex. Pain is usually caused by acute abdominal problems that will require surgical intervention, followed by nausea and vomiting. Nausea often precedes vomiting. Vomiting may be the main symptom in cases of Boerheave syndrome, Mallory-Weiss syndrome, acute gastritis and acute pancreatitis. Vomiting in pancreatitis develops after irritation of the celiac plexus, which it is in close proximity to. In occlusive diseases that cause colic pain, vomiting is more common in cases of severe pain.

Vomiting may be bile, non-bilious, bloody or fecaloid. Biliary and adrenal vomits are seen in high-level ileus tables. Vomiting occurs later in more distal obstructions.

Another symptom is anorexia. Although it is a nonspecific finding, it can be seen in the foreground in tables such as acute appendicitis. Again, most gastrointestinal system diseases in children are accompanied by anorexia.

Another picture is the questioning of changes in bowel habits. Constipation and diarrhea attacks should be questioned.

Jaundice can also be seen in mechanical icterus and suppurative cholangitis.

Gastrointestinal bleeding may also present itself as a picture. It can be seen as hematemesis, melena and hematochezia. These definitions are important in

terms of the location of the bleeding. It should not be forgotten that inflammatory bowel diseases, invaginations and gastroenteritis can cause bleeding.

In addition to these, hematuria, vaginal discharges and vaginal bleeding may present with acute abdomen. Urinary symptoms are important in the differential diagnosis. They can be seen in tables ranging from a simple cystitis to pyelonephritis. Stones and tumoral masses can also be caused by an inflammatory event. Again, the color of the urine is important in the differential diagnosis.

Another important issue in the history of the patient who applied with the picture of acute abdomen is knowing the menstrual picture. Ectopic pregnancy, ovarian cyst rupture, endometriosis, pelvic inflammatory diseases should be questioned and kept in mind.

6. Clinical Examination and Imaging

The examination of the patient presenting with acute abdomen should be done without breaking the rules. The anatomy of the abdomen should be well known, and the position of the patient should be in a way that makes the examination comfortable while examining the patient. It should be checked whether there is a scar on the abdomen due to previous surgery. A well-done inspection sometimes makes us go a long way in making the diagnosis. Roughly, the four main quadrants are divided into 6 zones. The abdomen is divided into longitudinal and transverse imaginary lines. Again, a line passes from both arcus costa and a line from both iliac crests, and the process is completed.

The general appearance of the patients gives important clues. Whether it is active or stationary, important clues such as skin color, tone, facial expression, agitation, and the look in their eyes should be carefully examined. Fever, pulse, blood pressure, respiratory rate should be carefully monitored. In particular, this first opinion creates an opinion in the physician, and then the next steps are taken.

In the abdominal examination, when the inspection is started, conditions such as distension, scarring, abdominal mass, presence of abdominal wall vessels, hernia are observed.

Auscultation should be done before palpation. Bowel sounds are important. A ringing sound indicates a bowel obstruction. As there will be a silent abdomen in paralytic ileus, an increase in number suggests gastroenteritis. Silent abdominal peritonitis, murmur suggests vascular pathologies.

Percussion may show us that the liver dullness has disappeared in the ascites or perforation in the abdomen.

There are a number of special maneuvers and findings in palpation. Abdominal tenderness, voluntary or involuntary defense, rebound, rowsing are the important ones. Here the tone of the abdominal muscles is perceived. Involuntary contraction is an important finding of peritonitis. If there is a wooden abdomen, perforation is considered in the foreground. The presence of rebound suggests a high probability that a surgical intervention will be required. Murphy's sign is tenderness with deep breathing in the right hypochondrium and is often suggestive of acute cholecystitis. The ileopsoas test can be used for retroperitoneal inflammations, and the obturator test for pelvic inflammation. Costovertebral angle tenderness is an important finding in acute pyelonephritis.

Inguinal and femoral hernia examinations should not be overlooked. Missing a small hernia during examination will delay the diagnosis.

Rectal examination should definitely be done in the aqua abdomen picture. Here, sphincter tone, pelvic mass, tenderness, color and content of stool smeared on the finger are important.

Again, genital examinations should be done. While sensitivity in the testicles will suggest that it may be an orchitis, testicular torsion, epididymitis, women may have gynecological pathologies.

After the history and physical examination are completed, diagnostic procedures are started. Here, first of all, there should be orientations towards the suspected diseases. Hemogram, broad biochemistry, coagulation tests, urine and stool tests should be seen.

In imaging, direct abdominal X-rays, chest X-rays and, if necessary, radiopaque examinations should be performed.

Today, ultrasonography is a reliable method in acute cholecystitis, acute appendicitis and pelvic inflammatory diseases and similar tables. Again, it is very valuable in solid organ evaluations.

Computed Tomography (CT) and Magnetic Resonance (MR) are other methods used. Although CT is used more frequently, MR is more prominent in some specific cases. For example, while an abscess mass in the abdomen is visualized with CT, a detailed liver, pancreas and biliary tract are better visualized with MRI.

Conventional Angiography is used especially in intraperitoneal bleeding, intestinal ischemia and gastrointestinal bleeding.

Again, scintigraphic procedures are used to find the bleeding focus.

While colonoscopy is used in the treatment of rectal bleeding and volvulus, gastroduodenoscopy is also used in conditions such as bleeding control.

While paracentesis was one of the most frequently used methods in the past, it is still a method that is used today, although not so frequently. It determines whether the aspiration material is bile, blood or pus and directs the procedure.

Diagnostic laparoscopy should be performed when the patient's surgery is decided, but in cases where the diagnosis cannot be fully differentiated.

7. Conclusion

Acut abdomen is a symptom caused by a number of different conditions. Its etiology refers to the study of the underlying causes of acut acute abdomen.

These causes may include conditions such as appendicitis, gallbladder diseases, intestinal obstructions, ulcer perforations, pancreatitis and kidney stones. However, a detailed medical evaluation is required to make an accurate dianosis in each case.

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CHAPTER II

APPROACH TO AN ACUTE ABDOMEN PATIENT IN THE EMERGENCY DEPARTMENT

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1. Introduction

Abdominal pain is a challenging diagnostic problem for physicians working in the emergency department. The differential diagnosis ranges from benign diseases to life-threatening conditions. The causes can be medical, surgical, intra-abdominal, or extra-abdominal. Associated symptoms are generally atypical, making the issues more complicated.

Diagnosis and diagnostic methods may be limited in elderly patients, immunosuppressed individuals, and pregnant women. Unclear, nonspecific symptoms can occur due to life-threatening diseases in elderly diabetic patients, particularly. [1, 2]

2. Epidemiology

Abdominal pain accounts for 5-10% of emergency department visits.[3] Despite the availability of numerous diagnostic methods, a diagnosis is not reached in 25% of emergency department visits for abdominal pain and in 41% of outpatient visits.[4-7] However, 80% of these patients become pain-free within two weeks.[6]

In elderly patients with abdominal pain, mortality is 6-8 times higher compared to younger patients.[1, 8] Patients aged 65 and over constitute 20% of emergency department visits, and 3-4% of these visits are due to abdominal pain.[1, 8, 9]Studies suggest that mortality rates increase in this patient group when a diagnosis is not reached in the emergency department.[10]

The incidence of abdominal pain in HIV+ patients ranges from 12% to 45% (16-17). Among HIV+ patients presenting with abdominal pain, 38% require hospitalization. In 11% of HIV+ patients who require surgery, there is the presence of an AIDS-related opportunistic infection.[11]

3. Differantial Diagnosis

3.1. Life-Threatening Conditions

The first things that should come to mind in patients presenting with abdominal pain are life-threatening conditions. Possible life-threatening causes are listed below.

- ✓ Abdominal aortic aneurysm
- ✓ Volvulus
- ✓ Ectopic pregnancy
- ✓ Splenic rupture
- ✓ Placental abruption
- ✓ Mesenteric ischemia
- ✓ Perforation of gastrointestinal tract(appendix, esophagus, bowel etc.)
- ✓ Bowel obstruction
- ✓ Myocardial İnfarction

3.2. Common Conditions

Common gastrointestinal, genitourinary and extraabdominal causes of abdominal pain are listed below.

- ✓ Appendicitis
- ✓ Biliary disease
- ✓ Pancreatitis
- ✓ Diverticular disease
- ✓ Peptic ulcer disease
- ✓ Incarserated hernia

- ✓ Gastroenteritis
- ✓ Inflammatory bowel disease
- ✓ Hepatitis
- ✓ Urinary tract infections
- ✓ Pyelonephiritis
- ✓ Nephrolithiasis
- ✓ Adenexial torsion
- ✓ Ruptured ovarian cyst
- ✓ Pelvic inflammatory disease
- ✓ Tuba-ovarian abscess
- ✓ Testicular torsion
- ✓ Diabetic ketoacidosis
- ✓ Pneumonia
- ✓ Pulmonary embolus
- ✓ Herpes zoster[12]

4. Patient History - Anamnesis

A careful history and physical examination usually distinguish the causes of abdominal pain and establish an appropriate differential diagnosis. When trying to determine the etiology of abdominal pain, it is very important to ask about age, gender, past illnesses and surgeries, and medications used and to characterize the pain.[12]

In elderly patients, the likelihood of serious diseases appearing with atypical symptoms is high. The risk of certain diseases, such as abdominal aortic aneurysm(AAA) rupture, mesenteric ischemia, and atypical presentations of myocardial infarction and intraabdominal cancers, increases significantly after the age of 50. It should be kept in mind that elderly patients show different symptoms and signs than younger patients, and those who use steroid-like medications can hide classic symptoms and signs. For example, in studies conducted on elderly patients diagnosed with cholecystitis, nausea and vomiting were seen more frequently than pain, and 84% of these patients had no abdominal pain.[13]

It should be determined whether women of reproductive age are pregnant or not. In the case of pregnancy, pregnancy complications (such as ectopic pregnancy), HELLP syndrome, and miscarriage should be considered in the differential diagnosis[14]

When questioning about abdominal pain, not only intra-abdominal organs but also extra-abdominal causes should be considered. Upper abdominal pain can be caused by lung and heart problems, especially in elderly patients. Therefore, questioning symptoms such as shortness of breath, cough, and palpitation is important.[12]

The medical and surgical conditions in the patient's medical history carry increased risks for certain diseases. For example, patients with a history of coronary artery or peripheral artery disease are at increased risk of abdominal aortic aneurysm and mesenteric ischemia. Patients with a history of atrial fibrillation and heart failure are at increased risk of embolism-induced mesenteric ischemia. Patients with a history of abdominal surgery are at risk of bride-related obstruction. The use of certain antibiotics increases the risk of C.difficile colitis. [12]

Questioning about social life and habits is also very important. For example, excessive alcohol consumption increases the risk of pancreatitis.[15]

5. Features Of Pain

5.1. Types of Pain

5.1.1. Visceral Pain

Visceral pain fibers originate from the walls of hollow organs and capsules of solid organs, and enter the spinal cord from various points. Stimulation of visceral nerves produces a dull, weak, and localized pain felt in the midline of the abdomen. Visceral pain caused by a diseased organ produces pain in the region corresponding to the embryological formation of that organ. Visceral pain originating from foregut organs (stomach, pancreas, liver, gallbladder, and proximal duodenum) is felt in the epigastric region. Visceral pain originating from midgut organs (distal duodenum, small intestine, and proximal large intestine) is felt around the umbilical region. Visceral pain originating from hindgut organs (middle and distal large intestine, pelvic genitourinary organs) is felt in the suprapubic region.

5.1.2. Parietal Pain

After stimulation, parietal pain is transmitted to the dorsal root ganglia on the same side and at the dermatomal level where the pain originates. Therefore, the pain is more distinct, sharp, and localized. Ischemia or stretching of the parietal peritoneum causes parietal pain.

5.1.3. Referred Pain

Refers to pain felt away from the affected organ. For example, shoulder pain caused by irritation of the diaphragm is an example of this type of pain. The common transmission of afferent pathways from different locations to the central nervous system causes this phenomenon. (uptodate)

5.2. Pain Characterization

- Onset (e.g. sudden, gradual)
- Aggravating and relieving factors (e.g. increase or decrease after meals
- Pain quality (e.g. sharp, colicky, increasing, decreasing)
- Spread (e.g. to the shoulder, back, sides, etc.)
- Location (e.g. specific quadrant or diffuse)
- Associated symptoms.[12]

The location of pain can be helpful in making a diagnosis (see table 1). Pain caused by abdominal organs produces pain in certain abdominal regions according to the embryological origin of the organ (see visceral pain).[12]

Table 1: Causes of abdominal pain by location[12]

Right upper quadrant	**Right lower quadrant**	**Diffuse**
Cholesystitis	Appandicitis	Gastroenteritiis
Cholangitis	Salpingitis	Mesenteric ischemia
Pankreatitis	Ectopic pregnancy	Familial mediterranean fever
Biliary colic	Inguinal hernia	Bowel obstruction
Hepatitis	Nephrolithiasis	Peritonitis
Pneumonia	Inflammatory bowel disease	Irritable bowel syndrome
Subdiaphragmatic abcess	Mesenteric adenitis	**Epigastric**
Left upper quadrant	**Left lower quadrant**	Peptic ulcer disease
Splenic abcess	Divertivulosis	Gastrititis
Splenic infarct	Salpingitis	Gastroesophageal refkux syndrome
Gastritis	Ectopic pregnancy	Pancreatitis
Gastric ulcer	Inguinal hernis	Myocardial infarction
Pancreatitis	Inflammatory bowel disease	

Pain in the upper right quadrant is generally related to the hepatobiliary system, but biliary colic pain may be poorly localized and patients may complain of upper chest, epigastric, or back pain.[16]Another cause of pain in the upper right quadrant may be myocardial infarction, pneumonia in the right lower lobe of the lung, or pulmonary embolism in the right lung.(30) Pain in the upper left quadrant may be due to pancreatitis, stomach problems, or splenomegaly. Non-abdominal causes of pain in the upper left quadrant may include left lower lung pneumonia or myocardial infarction. Acute appendicitis and ectopic pregnancy may cause pain in the lower right quadrant. Diverticulitis generally manifests with pain in the lower left quadrant. .[12]

Physicians should not rely solely on the localization of pain in differential diagnosis, as sometimes the diagnosis and location of pain may be atypical. (31,32). Retrocecal appendicitis may present with pain in the upper right quadrant. If this is not kept in mind, the diagnosis may be missed. In a study on patterns of abdominal pain, typical examination findings were found in 60-70% of patients, while atypical examination findings were found in 30-40%.[17]

The location of pain may change with the progression of the disease. As a classic example, appendicitis starts in the umbilical region (since it originates from the midgut, visceral pain is felt in this area), but with irritation of the parietal peritoneum, it turns into parietal pain and becomes localized in the lower right quadrant. The spread of pain can be helpful in diagnosis. [12]

The intensity, severity, and duration of pain can provide information about the severity of the disease.[17-19]Pain with the highest intensity at the beginning may be due to a vascular emergency (such as aortic rupture or dissection, mesenteric ischemia, etc.).[20] Sudden onset severe pain may be due to perforation or ischemia. Gradual onset pain may suggest an inflammatory or infectious process or bowel obstruction. Severe pain that starts suddenly and lasts more than 6 hours continuously or worsens should generally be considered as having a surgical cause. Non-surgical causes of pain are usually less severe.[12]

The factors that increase or decrease pain are important. Pain in peptic ulcer decreases after meals, while biliary colic worsens after meals. Pain in pancreatitis can improve when sitting upright, but can worsen when leaning back.[8]

Burning pains may be associated with ulcers, tearing pain may be associated with aortic dissection, and colicky pain may be due to the stretching of a hollow organ such as the ureter. Sharp pain can occur when inflammation or harmful contents (such as bowel contents) touch the parietal peritoneum.[12]

Symptoms that accompany pain are very important in the diagnostic process. Pneumonia, pulmonary embolism, and myocardial infarction may present to the emergency department with abdominal pain, so fever, cough, shortness of breath, and chest pain should be specifically asked about.[21] Nausea and vomiting may not be specific symptoms, but they can provide some clues. Vomiting before the onset of pain may raise suspicion of bowel obstruction.[18] The content of the vomit is important. Vomiting of bile may be caused by an obstruction distal to the duodenum. Diarrhea is generally caused by infection, but can also be seen in mesenteric ischemia.[14]

Genitourinary symptoms associated with abdominal pain should also be questioned. In women, vaginal bleeding, discharge, and the status of menstruation should be asked, while in men, penile discharge, scrotal pain, and swelling should be asked.[12]

6. Physical Examination

Physical examination should be started by evaluating vital signs. Although fever may increase in cases of infection, it may not increase in elderly and immunosuppressive patients. Elderly patients with intra-abdominal infections are four times more likely to present with hypothermia than the young population.[2] Increased respiratory rate may be a compensatory mechanism. In this case, the clinician should be careful about metabolic acidosis.[12]

Then, inspection is performed. Even if inspection is a few seconds, it can reveal many clues. For example, someone who is lying motionless by bending their knees raises suspicion of peritonitis in the clinician. Surgical scar marks in the patient's medical history can be observed.[14]

After inspection, auscultation is performed. Bowel sounds should be listened to for at least 2 minutes during auscultation. Normally, bowel sounds are heard in a moderately rumbling form between 2 and 12 per minute. The absence of bowel sounds for 2 minutes suggests peritonitis. Hyperactive bowel sounds are associated with blood or inflammation in the gastrointestinal tract. Periodic high-pitched bowel sounds may be present with abdominal distension. A murmur can be heard in abdominal aortic aneurysm.[22]

After auscultation, palpation is performed. Palpation determines the location and degree of sensitivity, and detects signs of peritoneal irritation such as defense and rebound. Palpation starts from areas far from the painful abdominal region and the painful area is examined last. After a sensitive area is detected, the examination is diversified. If sensitivity is not found with light palpation, deep palpation can be performed.[14]

It is valuable to palpate the aorta in older patients over 50 years old. If the pulse of the aorta is felt over a wide area during this examination, abdominal aortic aneurysm should be considered.

In Carnett's sign, when the abdominal wall muscles contract, sensitivity increases. In this case, a pathology originating from the abdominal wall is more likely in worsening abdominal pain. In a study, it was found that Carnett's sign provided a 95% accuracy in distinguishing between abdominal wall pain and visceral pain.

Although the sensitivity is low, the specificity of the Rovsing, obturator, and psoas tests is high for appendicitis.[12]

Murphy's sign occurs when deep inspiration suddenly stops after deep palpation of the right upper quadrant. This test is important for the diagnosis of acute cholecystitis, but its sensitivity may decrease in elderly patients.[23, 24]

Physical examination cannot predict or rule out life-threatening diseases in elderly patients.[25] Due to changes in the nervous system that affect pain sensation, there may be no abdominal pain. Due to the relaxation of the abdominal wall structures with age, rebound or defense may not be felt. In a study of elderly patients with peritonitis, only 34% of patients had defense or rebound.[26]

Pregnant patients may become less sensitive to peritoneal irritation over time and may have fewer clinical signs and peritoneal irritation findings due to the expansion of the intraperitoneal space. Therefore, physical examination may be more difficult in pregnant women.[27]

Since abdominal pain may have extra-abdominal causes, it is important to examine not only the abdomen but also other systems. For example, pyelonephritis may be considered in a patient with costovertebral angle tenderness.

Skin examination is also important in patients with abdominal pain. This is particularly important for diagnosing herpes zoster. Cullen's sign or Grey Turner's sign is an important indicator of hemorrhagic pancreatitis.[12]

Serial physical examinations can increase diagnostic accuracy in patients with suspicious physical examinations.[28]

7. Laboratory Tests

Laboratory tests should be ordered in healthy adults to make a diagnosis in suspected cases or to evaluate patients with unknown etiology. A wider range of tests should be ordered in patients who have difficulty in obtaining a medical history, those with additional diseases such as diabetes, and elderly patients.[12]

Beta HCG should be ordered in women of reproductive age to determine pregnancy status. Determining a patient's pregnancy status with a declaration is not reliable.[29]

Although complete blood count is often ordered, it is not a specific test. [30-32] In acute appendicitis, white blood cell counts increase by 80%, while in other causes of right lower quadrant pain, they increase by 70%.[32, 33] Normal white blood cell counts may be present in immunosuppressed and elderly patients presenting with acute abdomen, whereas leukocytosis may be present in a healthy pregnant woman.[34]

Liver and pancreatic enzyme concentrations should be measured in patients with pain in the right upper quadrant and epigastric region. An increase in serum amylase levels is not specific or sensitive for pancreatitis, as it may also increase in cases of small bowel perforation and mesenteric ischemia. Serum lipase levels are more specific and sensitive than amylase levels in pancreatitis. An increase in bilirubin and alkaline phosphatase levels may be seen in complicated cholecystitis and biliary obstruction.[35]

Urinalysis can be helpful in diagnosis, but can also be misleading. Pyuria, proteinuria, and hematuria suggest a urinary tract infection, but can also be seen in acute appendicitis. In one study, bacteria, blood, or leukocytes were detected in the urine of 20-48% of patients diagnosed with acute appendicitis. [36, 37]

8. Radiological Examinations

8.1. Plain Radiographs

Plain abdominal radiographs are typically taken for patients presenting with abdominal pain. Only a small fraction of the obtained images are abnormal. A plain radiograph can be helpful in the case of gastrointestinal system perforation, bowel obstruction, or the presence of a radiopaque foreign body.[38]

The diagnosis of gastrointestinal perforation can be made by detecting intraperitoneal free air on an upright chest radiograph. In cases of perforation of the stomach and duodenum, free air is found in two-thirds of cases. However, this ratio drops to one-third in cases of distal small bowel and large bowel perforation. If the patient has a history of abdominal surgery, this ratio drops even further.[39] If free air is not detected on a posterior-anterior chest radiograph, a lateral vertical film can be taken. In patients unable to stand, a left lateral decubitus radiograph can be taken.[40]

Approximately 5 mL of air can be detected with a plain abdominal radiograph. Even small amounts of air, ranging from 1 to 2 mL, can be detected on upright films. If it cannot be detected and is suspected, 50 mL of air can be given through a nasogastric tube, and then detected on an X-ray.[39, 41]

The specificity of plain radiographs for the diagnosis of bowel obstruction is 69%, and the sensitivity is 57%. In cases of bowel obstruction, gas distension and air-fluid levels are seen on the radiograph.[42]

8.2. Ultrasound

Ultrasound is one of the first tools to evaluate patients because it can be done quickly, at the bedside, and by most branch physicians. It is the first choice especially for pregnant and trauma patients.It can be useful in evaluating ovaries, uterus, and scrotal pathology, detecting gallbladder pathology, diagnosing ectopic pregnancy, assessing for hemoperitoneum, renal colic, and appendicitis, among other conditions.[43]

8.3. Computed Tomography

Two-thirds of patients presenting with acute abdominal pain to the emergency department are diagnosed using computed tomography. (68) One study found that the accuracy of diagnosis based on history and physical examination alone was 78%, but this rate increased to the 90s when computed tomography was added.[44] It is particularly useful in elderly patients with nonspecific symptoms.[1]

Intravenous contrast administration is beneficial in detecting abdominal pain that cannot be localized. Oral contrast is controversial with the improvement of CT scanners.[45] (72) Contrast-enhanced CT is highly sensitive for detecting free air. In cases where contrast administration may harm the patient (e.g. renal failure), the decision to administer contrast should be made by the patient's physician, weighing the risks and benefits.[46]

8.4. Angiography

It appears to be useful in diagnosing mesenteric ischemia.[12]

9. Results

Patients diagnosed with acute abdomen constitute one of the most common clinical encounters throughout the entire professional life of physicians.

Although the approach to these patients may appear simple, it requires a vast amount of theoretical knowledge and practical experience. Particularly, indicators carrying life-threatening implications should not be overlooked, and the diagnostic approach must be applied without delay. The importance of physical examination should be acknowledged as much as imaging techniques during the diagnostic process. Treatment protocols should be promptly initiated based on preliminary diagnoses. This ensures the successful implementation of patient treatments.

ALGORITMS IN ACUTE ABDOMEN PAIN

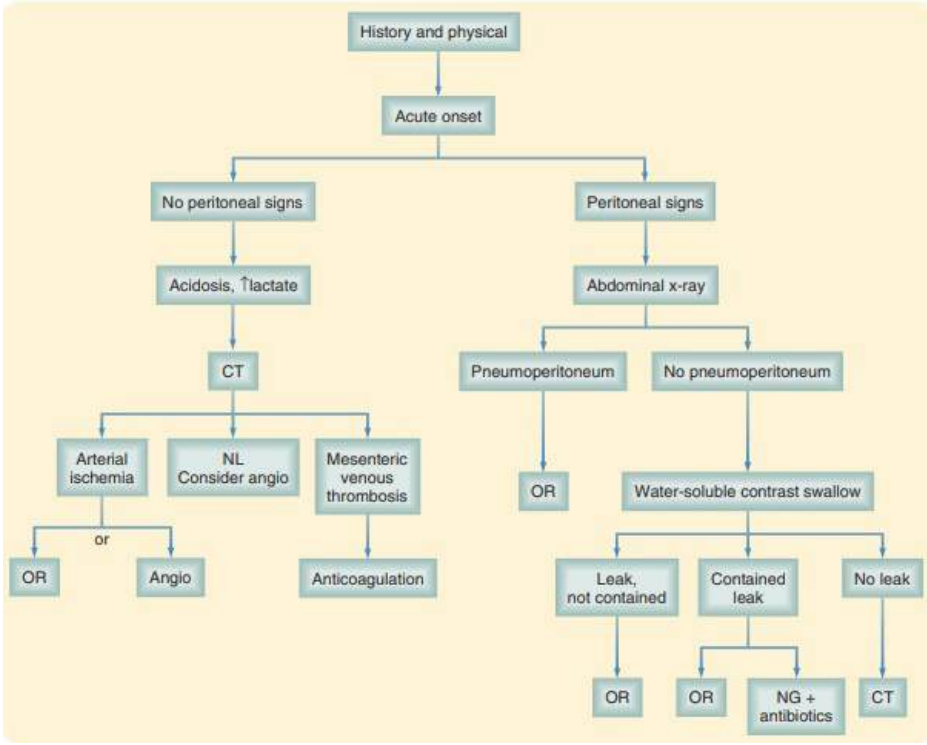

Figure 1: Algorithm for the treatment of acute-onset severe, generalized abdominal pain. CT, computed tomography; NG means nasogastric tube; NL means normal study; OR means operation[14]

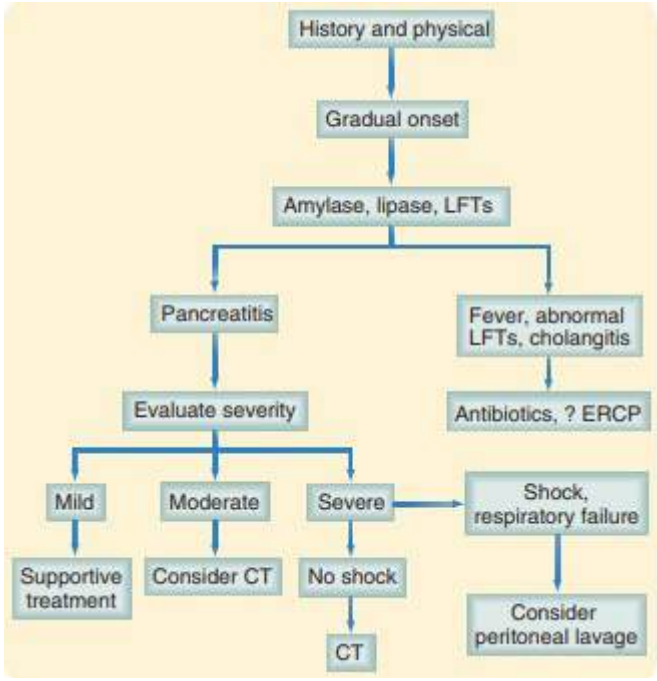

Figure 2: Algorithm for the treatment of gradual-onset severe, generalized abdominal pain. ERCP means endoscopic retrograde cholangiopancreatography; LFTs means liver function tests[14]

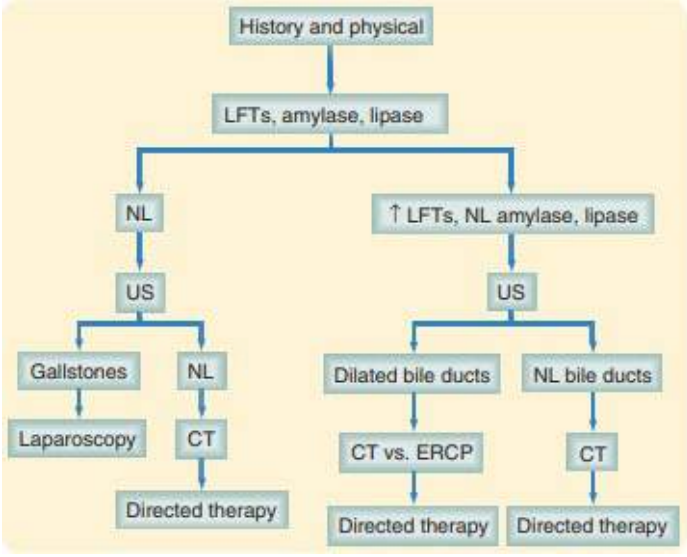

Figure 3: Algorithm for the treatment of right upper quadrant abdominal pain. US means ultrasound[14]

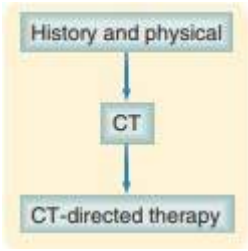

Figure 4: Algorithm for the treatment of left upper quadrant abdominal pain[14]

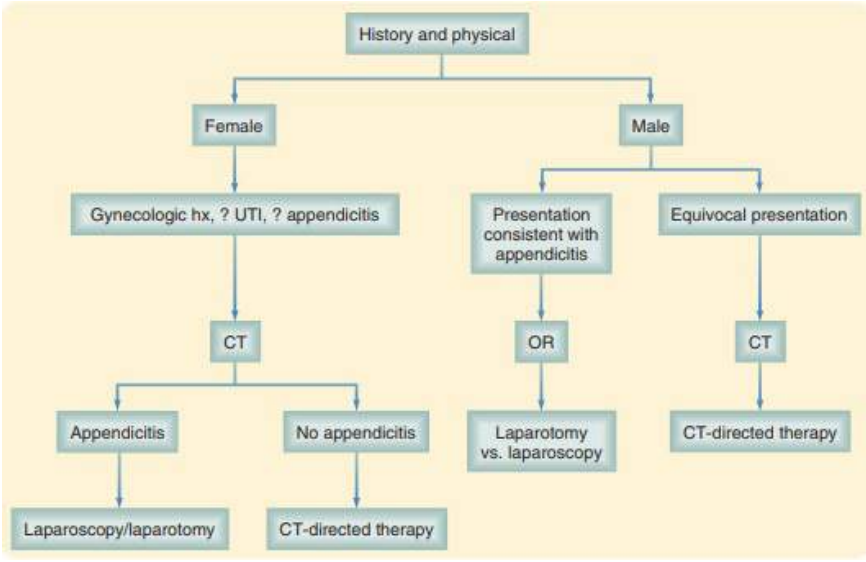

Figure 5: Algorithm for the treatment of right lower quadrant abdominal pain. hx means history; UTI means urinary tract infection[14]

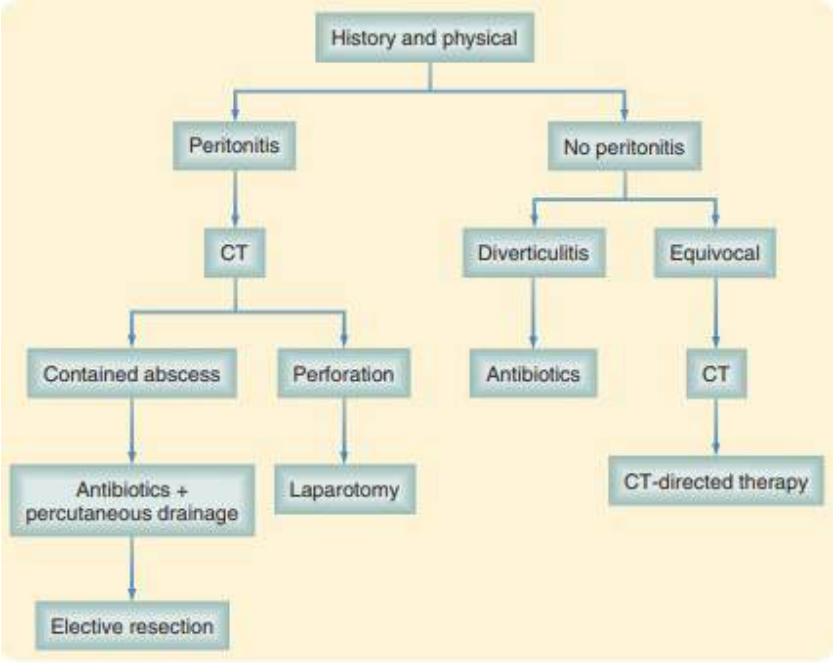

Figure 6: Algorithm for the treatment of left lower quadrant abdominal pain.

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CHAPTER III

FLUIDS, ELECTROLYTES AND SHOCK

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1. Introduction

Surgical diseases are frequently closely related to acid-base balance and fluid-electrolyte balance. Surgeons must be skilled in fluid and electrolyte management and treatment. It is one of the most critical issues in patient management. It is essential to differentiate between dehydration, anemia, hemorrhage, and excessive fluid resuscitation. The purpose of this chapter is to explain the diagnosis and treatment of emergency pathologies in fluid-electrolyte balance.

2. Body Fluids

Approximately 60% of the human body weight consists of water. However, this percentage may vary based on factors such as age, gender, and body weight. The water in our body is divided into two compartments, namely intracellular (which constitutes about 67% of total body water) and extracellular (which constitutes about 33% of total body water). The extracellular compartment is further subdivided into interstitial fluid (which constitutes about 22% of total body water) and intravascular fluid (which constitutes about 8-10% of total body water). Water moves between these compartments via osmosis. In pathological conditions, a third space may be formed, which is caused by an increase in

interstitial fluid due to capillary leakage resulting from injury or illness. The size of this third space is directly proportional to the severity of the injury or illness. The distribution of electrolytes in the different compartments is illustrated in the table below (1).

Table 1. *Electrolyte Concentrations of Body Fluid Compartments (mEq/L).*

Solution	Plasma	Interstitial	Intracellular
Cations			
Sodium	142	144	10
Potassium	4	4,5	150
Magnesium	2	1	40
Calcium	5	2,5	-
Total Cations	153	152	200
Anions			
Chloride	104	113	-
Phosphates	2	2	120
Sulfates	1	1	30
Bicarbonate	27	30	10
Proteins	13	1	40
Organic Acids	6	5	-
Total Anions	153	152	200

Potassium and magnesium are the primary cations found intracellularly, with phosphates and proteins serving as the primary anions. In contrast, the predominant cation extracellularly is sodium, with chloride and bicarbonate being the predominant anions (1, 2). The normal daily water requirement for an adult is approximately 2000cc, with 1500cc obtained orally and the remainder from ingested food. Fluid loss occurs via urine, feces, and sweating, as well as through unnoticed losses from the skin and lungs. The body strives to maintain plasma volume through osmoreceptors and baroreceptors. When a patient presents with symptoms, a rapid examination should be performed to evaluate volume status. Symptoms that suggest hypovolemia:

- ✓ Cold, pale skin
- ✓ A capillary refill time longer than 1.5 seconds
- ✓ Dry mucous membranes

- ✓ Urine output less than 0.5 mg/kg/h
- ✓ Delirium and confusion
- ✓ Tachycardia

However, it should also be kept in mind that intravascular fluid may have shifted to the interstitial space in the presence of these symptoms. Several factors may contribute to fluid depletion, including vomiting, diarrhea, nasogastric suctioning, diuretic use, diabetes mellitus or insipidus, osmotic agents, adrenal insufficiency, third-spaced fluid mobilization, recovery phase of acute tubular necrosis, open wounds, hyperventilation, increased sweating, systemic infection, pancreatitis, ileus, burns, gastrointestinal bleeding, and trauma (3).

3. Electrolytes

3.1. Sodium

The normal range for serum sodium in peripheral blood is 135-147 mEq/L. The concentration of sodium in the serum does not necessarily indicate the total amount of sodium in the body, but rather provides information about fluid balance. Sodium is the primary extracellular cation. Symptoms related to changes in serum sodium levels are more dependent on the rate of change rather than the actual sodium level. Therefore, any correction of serum sodium levels should be done slowly.

➢ Hyponatremia

Hyponatremia is characterized by a serum sodium level that is below 135 mEq/L. This can occur in different forms, including hypervolemic, hypovolemic, or normovolemic hyponatremia.

Table 2. *Classification for the assessment of hyponatremia (1).*

Hypovolemic	Normovolemic	Hypervolemic
✓ Decreased sodium intake ✓ Gastrointestinal losses ✓ Renal losses ✓ Diuretics ✓ Primary renal disease	✓ Hyperglycemia ✓ Increased plasma lipids and proteins ✓ SIADH ✓ Water intoxication ✓ Diuretics	✓ Drugs ✓ Increased intake ✓ Postoperative ADH secretion

The most significant effects of hyponatremia are on the brain, and the symptoms can range from moderately severe to severe depending on the plasma sodium level (4, 5). Moderately severe symptoms such as nausea, disorientation, ataxia, confusion, headache, agitation, and areflexia may occur when plasma [Na^+] is below 130 mEq/L. Severe symptoms such as intractable seizures, vomiting, respiratory arrest, and coma due to brainstem herniation may develop when [Na+] levels fall below 120mEq/L. Brain injury can become irreversible at this point (6).

Rapid correction of sodium levels can make the brain vulnerable due to its protective mechanisms against swelling. This can lead to the development of osmotic demyelination syndrome (known as central pontine myelinolysis) which usually occurs in patients with a sodium level below 120 mEq/L (7). Therefore, a slow correction is necessary to prevent cerebral damage, and the infusion rate should not exceed 2 mEq/L/hour. The rate of infusion should be adjusted based on the severity of symptoms, and it typically ranges between 0.5 to 2 mEq/L/hour (3).

➢ **Hypernatremia**

Hypernatremia is described as a serum sodium level above 145 mEq/L, which can result from either excessive water loss or sodium gain. However, hypernatremia is more commonly associated with fluid loss.

Table 3. *Classification for the assessment of hypernatremia (1).*

Hypovolemic	Normovolemic	Hypervolemic
✓ Nonrenal water loss ✓ Skin ✓ Gastrointestinal ✓ Renal water loss ✓ Renal tubular disease ✓ Osmotic diuretics ✓ Diabetes insipidus ✓ Adrenal failure	✓ Nonrenal water loss ✓ Skin ✓ Gastrointestinal ✓ Renal water loss ✓ Renal disease ✓ Diuretics ✓ Diabetes insipidus	✓ Iatrogenic intake ✓ Mineralocorticoid excess ✓ Aldosteronism ✓ Cushing's disease ✓ Congenital adrenal hyperplasia

Symptoms of hypovolemia, such as mental changes, edema, coma, and seizures, can be observed in hypernatremia patients. In hypovolemic patients, isotonic fluids can be administered, while hypotonic solutions with free water supplementation are used for patients who are not hypovolemic. However, it is important to note that correcting sodium levels too rapidly in asymptomatic

patients can lead to cerebral edema. In acute hypernatremia, the rate of correction is mostly limited to 1 to 2 mEq/hr (2).

3.2. Potassium

Potassium is an essential mineral that plays a crucial role in numerous bodily functions. The normal range of serum potassium levels is between 3.5 and 5 mEq/L. Potassium is primarily an intracellular ion and is necessary for maintaining proper cellular function, including muscle contraction and nerve impulses. The daily requirement of potassium is around 1mEq/kg/day. Potassium levels can be affected by acid-base imbalances, with levels decreasing in alkalosis and increasing in acidosis.

- **Hypokalemia**

Hypokalemia is described as a serum potassium level below 3.5 mEq/L and is more common than hyperkalemia. It can be caused by reduced intake, medications, hyperaldosteronism, renal losses, gastrointestinal losses, or metabolic alkalosis (1).

Hypokalemia can lead to weakness, paresthesia, decreased deep tendon reflexes, and paralytic ileus. It can also cause changes in the ECG such as T wave flattening, prolonged QT interval, and U wave appearance. Life-threatening cardiac rhythm disturbances can occur, including atrial fibrillation, ventricular tachycardia, torsade de pointes, and ventricular fibrillation, and even cardiac arrest (4).

Oral potassium supplementation ranges from 40 to 100 mEq/day, divided into two to four doses. The rate of intravenous potassium administration is 10 to 20 mEq/hr, and if infused at rates over 10 mEq/hr, cardiac monitoring is required. In emergency situations, the rate can be increased to as high as 40 mEq/hr, but it is important to use a central vein due to the potential for peripheral vein irritation from high concentrations of potassium in IV fluids. In patients reduced potassium excretion or renal dysfunction, hypomagnesemia should be considered as a possible cause of resistant hypokalemia (2).

- **Hyperkalemia**

Hyperkalemia is described as a serum potassium level greater than 5 mEq/L and it is most commonly seen in patients with renal failure. Increased potassium intake, blood transfusion, hemolysis, rhabdomyolysis, crush injury, gastrointestinal bleeding, acidosis, hyperglycemia, mannitol use, and potassium-sparing diuretics can all cause hyperkalemia (1).

The symptoms range from nausea, vomiting, and diarrhea to ascending paralysis and coma. The main clinical problem is its cardiac effects. EKG findings may include tall T waves, flattened P waves, widened QRS complex, and prolonged PR interval. Death usually occurs due to cessation of the heart during diastole. If EKG changes are present, immediate treatment should be initiated. The patient should be monitored closely. Glucose and insulin infusion can be used to move potassium inside the cell, and calcium infusion can be used to reduce its cardiac effects. In cases of renal failure, dialysis should be considered as a treatment option (2).

3.3. Calcium

Calcium is an essential mineral in the human body, with a total calcium level 8.5- 10.5 mg/dL and an ionized calcium level 4.2- 4.8 mg/dL. It is primarily absorbed from the intestines under the control of vitamin D, and approximately 99% of calcium in the body is stored in the bones. The remaining 1% is distributed in the bloodstream and extracellular fluid, with about 80% bound to albumin.

In cases of hypoalbuminemia, corrected calcium levels are calculated as ***[Total Ca + (0.8 x (4.5 – albumin level))]*** to account for the effect of low albumin levels on calcium binding. Calcium plays a critical role in various including muscle and nerve function, physiological processes, bone formation, and blood clotting.

- **Hypocalcemia**

Hypocalcemia is described as a serum calcium level below 8.5 mg/dL or an ionized calcium level below 4.2 mg/dL. Various conditions such as pancreatitis, massive tissue infections, renal failure, small intestine and pancreatic fistulas, short bowel syndrome, tumor lysis syndrome, hypomagnesemia, vitamin D deficiency, and hypoparathyroidism, as well as certain medications, can lead to hypocalcemia. Resistant hypocalcemia usually occurs due to atrophy of the remaining glands after parathyroid adenoma removal (1). Hypocalcemia can manifest with various symptoms, including papilledema, diplopia, paresthesia, Chvostek’s sign, Trousseau’s sign, tetany, and prolonged QT interval on ECG without U wave (3).

When serum calcium falls below 7 mg/dL, oral or IV replacement should be initiated. Calcium gluconate should be given at a rate of 1-2 g over 10-20 minutes, followed by a continuous infusion at a rate of 0.5-1.5 mg/kg/hr. Rapid administration of calcium gluconate can increase the risk of cardiac arrest (2).

➢ **Hypercalcemia**

Hypercalcemia is a condition characterized by serum calcium levels greater than 10.5 mg/dL or ionized calcium levels greater than 4.8 mg/dL. The most common causes of symptomatic hypercalcemia are primary hyperparathyroidism in outpatient settings and malignancy in hospitalized patients, which can result from bony metastasis or secretion of parathyroid hormone–related protein. Other causes include vitamin D or A intoxication, granulomatous diseases, kidney failure, Paget's disease, and immobilization (1). Symptoms of hypercalcemia may include bradycardia, insomnia, delirium, coma, corneal calcification, vomiting, anorexia, pancreatitis, muscle weakness, hyporeflexia, bone pain, and pathological fractures. To increase volume, the patient may be given fluid loading. Furosemide can be administered at a dose of 20-40 mg every 2-4 hours. The calcium level in the blood should be closely monitored. To treat hypercalcemia, it is necessary to eliminate the underlying cause (3).

3.4. Magnesium

Magnesium is a mineral that is essential for many biochemical processes in the body. Its normal range in the blood is 1.5-2.0 mEq/L. Magnesium is the second most plenty intracellular cation, after potassium, and plays a crucial role in many physiological functions, including muscle and nerve function, protein synthesis, and energy metabolism. About 30% of magnesium in the blood is bound to proteins, 10% is bound to anions, and 60% is present in its ionized form (2). When there are changes in potassium and calcium concentrations, magnesium should be considered.

➢ **Hypomagnesemia**

Hypomagnesemia is a condition characterized by a magnesium level of less than 1.5 mEq/L. It is often caused by urinary or gastrointestinal losses. Chronic diarrhea, vomiting, high-output fistula, malnutrition, and certain medications can also contribute to this condition. Proton pump inhibitors have been reported to cause hypomagnesemia (8). Hypomagnesemia can lead to tetany, muscle weakness, nystagmus, vertigo, paresthesia, seizures, nausea, and heart failure. In acute treatment, magnesium sulfate 2g IV can be given over 2-5 minutes, followed by a continuous infusion of 10 g IV over 24 hours (3).

➢ **Hypermagnesemia**

Hypermagnesemia is described as a serum magnesium level above 2.0 mEq/L. It is a rare condition that can occur in severe kidney failure. It

rarely causes symptoms, but when present, it can lead to nausea, loss of deep tendon reflexes, heart block, and cardiac arrest (4). If the patient is taking magnesium supplements, they should be discontinued. Calcium gluconate is administered as treatment. In cases of severe renal failure, dialysis may need to be considered.

4. Acid Base Disorders

The normal pH of the body is within the range of 7.35-7.45. The normal value of PCO2 in arterial blood gas is 40 mmHg, and the normal value of HCO3 is 22-26 mEq/L. The body tries to keep these values within a narrow range through regulation of the kidneys and lungs.

Chemoreceptors located in the carotid body and brain stem are sensitive to hydrogen levels and regulate changes in the body's ventilation to address metabolic abnormalities. Acidosis stimulates an increase in ventilation, while alkalosis reduces chemoreceptor activity, leading to a decrease in ventilation. The kidneys adjust the amount of bicarbonate reabsorbed in response to respiratory acidosis or alkalosis to compensate for respiratory abnormalities. However, the compensatory response in the kidneys to respiratory abnormalities is delayed compared to the immediate change in ventilation seen with metabolic abnormalities. It may take up to 6 hours before significant compensation begins and may continue for several days (1).

4.1. Metabolic Acidosis

Metabolic acidosis can consist of as a result of increased acid intake, increased acid production, or increased bicarbonate loss. It can be classified into two categories: normal anion gap and increased anion gap. The anion gap is calculated using the following equation:

$$*\text{Anion gap} = (Na+ + K+) – (Cl- + HCO3-)$$

The normal range for the anion gap is 10-12 mEq/L. For patients with hypoalbuminemia, the following corrected value formula should be used:

$$*\text{Corrected anion gap} = \text{actual AG} – (2.5(4.5 – \text{albumin}))$$

Causes of metabolic acidosis with an increased anion gap include ethylene glycol, salicylate, and methanol intake, as well as endogenous acid production such as ketoacidosis, lactic acidosis, and renal insufficiency. Causes of metabolic acidosis with a normal anion gap include acid administration (HCl),

gastrointestinal losses (diarrhea, fistulas), loss of bicarbonate, renal tubular acidosis, and carbonic anhydrase inhibitor use (1).

Lactic acidosis is a common cause of acidosis in surgical patients and occurs due to impaired tissue perfusion. In these patients, increasing perfusion with fluid replacement is necessary. Intervention with bicarbonate from external sources may cause further lowering of pH. In clinical studies of lactic acidosis and ketoacidosis, the administration of bicarbonate has not reduced morbidity or mortality (9).

4.2. Metabolic Alkalosis

Metabolic alkalosis results from decreased bicarbonate elimination or increased bicarbonate generation. Most patients with metabolic alkalosis also have hypokalemia. Hypochloremic hypokalemic alkalosis is caused by the loss of gastric contents. In patients with hypokalemic alkalosis, paradoxical aciduria is observed due to an increase in H+ excretion in the urine. Milk-alkali syndrome is caused by excessive milk or antacid intake, which binds stomach acids. Calcium in milk, and Al and Mg in antacids, bind with H^+ ions. This leads to metabolic alkalosis. Treatment involves volume replacement and correction of electrolyte imbalances.

4.3. Respiratory Acidosis

Respiratory acidosis consists when carbon dioxide accumulates in the blood due to an inability to breathe adequately. This can be caused by a wide range of conditions, such as hypoxia, respiratory depression from medications, pneumonia or chronic obstructive pulmonary disease. It is most commonly seen after an obstruction in the airways. Respiratory acidosis can also occur after surgical procedures due to postoperative narcotic analgesia (3). Treatment focuses on addressing the underlying cause but, if necessary, intubation may be required to assist breathing.

4.4. Respiratory Alkalosis

Respiratory alkalosis consists when there is a decrease in carbon dioxide in the blood, typically due to hyperventilation. This can be caused by a variety of factors, such as anxiety, fear, or certain medications. In surgical patients, respiratory alkalosis is often due to alveolar hyperventilation. Treatment focuses on addressing the underlying cause, such as anxiety management, and adjusting medications as needed.

Table 4. *Acute and chronic acid-base disorders.*

	Acute (uncompensated)			Chronic (partially compensated)		
ACID-BASE DISORDER	**pH**	**PCo2 (Respiratory component)**	**Plasma HCO3 (Metabolic component)**	**pH**	**PCo2 (Respiratory component)**	**Plasma HCO3 (Metabolic component)**
Respiratory acidosis	↓↓	↑↑	Normal	↓	↑↑	↑
Respiratory alkalosis	↑↑	↓↓	Normal	↑	↓↓	↓
Metabolic acidosis	↓↓	Normal	↓↓	↓	↓	↓
Metabolic alkalosis	↑↑	Normal	↑↑	↑	↑	↑

5. Shock

Shock is defined as cellular damage due to impaired tissue perfusion and oxygenation. There is no single parameter that is used to diagnose shock. The patient is evaluated as a whole. Shock is initially a reversible process, but over time it becomes irreversible. Changes in consciousness can occur in shock. Other signs and symptoms of shock include:

- ✓ Hypotension, with a systolic blood pressure of less than 90 mmHg
- ✓ Tachycardia, with a heart rate greater than 120 beats per minute
- ✓ Oliguria, with urine output of less than 30 ml per hour

It is not necessary for all three criteria to be present. The most recent and accurate indirect diagnostic method for shock is tissue pH measurement. Treatment for shock involves identifying and addressing the underlying cause, and supporting the patient's cardiovascular function through measures such as fluid resuscitation, vasopressors, and inotropic agents.

5.1. Classification of Shock

There are different classifications of shock. Hinshaw and Cox proposed a four-part classification involving hypovolemic, cardiogenic, distributive, and obstructive subsets (10). However, shock can be broadly divided into two types: hyperdynamic shock and hypodynamic shock.

➢ Hyperdynamic Shock

In hyperdynamic shock, cardiac output is high and vasodilation occurs. Blood flow may be normal or decreased, but organ dysfunction is due to incorrect distribution of blood. Sepsis, anaphylaxis, drug intoxications, spinal shock and adrenal insufficiency are among the common causes of hyperdynamic shock (11).

✓ Septic Shock

Septic shock is characterized by the inability of the vascular smooth muscle to contract properly due to inflammatory mediators and endothelial dysfunction, leading to vasodilation and hypotension. It is unresponsive to vasopressor therapy. While endotoxin (lipopolysaccharide) is a major trigger in gram-negative bacilli, which causes up to 50% of septic shock cases with an identified pathogen, various exotoxins (all bacteria), peptidoglycans (streptococci), teichoic acid (Staphylococcus aureus), lipoarabinomannan of mycobacteria, bacterial DNA and ribonucleic (RNA), and mannoproteins and β-glucan of fungi are other major initiators of the systemic inflammatory response characteristic of sepsis (14).

✓ Neurogenic Shock

Neurogenic shock is the least common type of shock. It results from vasodilation and pooling in the vessels below the affected level due to impaired neural signaling. Venous return and cardiac output decrease. It is usually caused by spinal cord injury due to vertebral bone fractures. Injuries to the spinal cord resulting from penetrating trauma are more likely to cause hemorrhagic shock than neurogenic shock (15). Symptoms include warm extremities, bradycardia, and hypotension.

➢ Hypodynamic Shock

In hypodynamic shock, cardiac output is low and vasoconstriction is present. Decreased blood flow directly causes organ dysfunction. Bleeding, dehydration, and massive capillary leakage are common causes of hypovolemic shock.

✓ Hypovolemic Shock

In surgical and trauma patients, the most common cause of hypovolemic shock is hemorrhagic shock. As a conclusion of developing hypovolemia, baroreceptors are stimulated and vasoconstriction occurs. Hypovolemia

also activates the sympathetic system, stimulates the release of adrenaline and norepinephrine, activates the renin-angiotensin system, and secretes vasopressin (1).

✓ **Cardiogenic Shock**

Conditions that impair the pumping function of the heart cause cardiogenic shock. The most common cause of cardiogenic shock is MI, and a death rate of 50 to 80% in cardiogenic shock (1). Furthermore, the most common cause of death in acute MI is cardiogenic shock. There are three parameters in diagnosis (12):

✓ Hypotension
✓ Low cardiac index (<2.2L/min)
✓ High pulmonary artery wedge pressure (>15mmHg)

It is important to distinguish right ventricular failure or infarction-related cardiogenic shock from other types of cardiogenic shock because fluid therapy and inotropes are used in this type of cardiogenic shock. Conditions that endanger right ventricular function, such as restrictive cardiomyopathy, cardiac tamponade, constrictive pericarditis, and pulmonary embolism, are also included in the differential diagnosis. Treatment response is better in this type of cardiogenic shock (13).

Obstructive shock is most commonly caused by tension pneumothorax, acute pulmonary embolism, and pericardial tamponade. It is difficult to distinguish obstructive shock from cardiogenic shock (11).

6. Fluid Types

6.1. Crystalloids

Crystalloids are commonly used fluids due to their availability, affordability, and ability to replace volume in the body. They do not carry the risk of infection, but may cause edema as they do not stay within the blood vessels. Crystalloids don't increase the oxygen-carrying capacity of the blood. Studies on fluid kinetics have shown that 70% of crystalloids remain in the intravascular space within the first 20 minutes of continuous infusion, but this percentage drops to 50% after 30 minutes (16).

Normal saline, a type of crystalloid, does not contain bicarbonate and can cause dilutional metabolic acidosis as a result of a decrease in bicarbonate levels

in the blood. There is no clinical evidence that shows this type of acidosis has a significant impact on patient outcomes (2).

6.2. Hypertonic Saline

Hypertonic saline contains a high concentration of sodium, which draws water into the vascular space and increases volume without the need for large amounts of fluid. It has been shown to reduce the inflammatory response and can be used as an immunomodulatory agent (2). It has also been demonstrated to improve brain perfusion and decrease intracranial pressure in closed head injuries, while preventing cerebral edema (18).

Table 5. *Content of fluids (17).*

Fluid	Na^+	K^+	Cl^-	Ca^{2+}	Mg^{2+}	HCO_3^-	Lactate	Acetate	Gluconate	Glucose(g/L)	Osmolarity	pH (In Vitro)
Plasma	140	5	100	4.4	2	24	1	—	—	—	285	7.4
0.9% NaCl	154	—	154	—	—	—	—	—	—	—	308	6.0
0.45% NaCl	77	—	77	—	—	—	—	—	—	—	154	
5% dextrose	—	—	—	—	—	—	—	—	—	50	252	4.5
5% dextrose in lactated Ringer solution	130	4	109	3	—	—	28	—	—	50	525	5.0
Hartmann solution	131	5	111	4	—	—	29	—	—	—	275	6.5
Normosol-R	140	5	98	—	3	—	—	27	23	—	294	4-6.5
Normosol M with 5% dextrose	40	13	40	—	3	—	—	16	—	50	389 / 363	3.5-6

6.3. Colloids

Colloids are fluids that contain large molecules that can hold onto other fluids. Some commonly used synthetic colloids include plasma, albumin, dextran, gelatin, and starch-based colloids. Albumin is the most commonly used colloid and has several advantages over synthetic colloids. It is an anti-inflammatory substance, has a lower volume load, and does not have any coagulopathy side effects. It has also been proven to be safe in patients with infections.

Gelatin, which is derived from the hydrolysis of collagen, is a cheaper option with fewer renal side effects than other colloids (19).

There is no clear evidence that shows whether colloids are better or worse than crystalloids. The choice of fluid in treatment ultimately depends on the patient's condition, fluid requirements, and individual factors (20).

7. Conclusion

This section delineates the repercussions of surgical diseases on fluid-electrolyte equilibrium and acid-base homeostasis, underscoring the imperative for surgeons to possess adept knowledge and skills in this domain. It accentuates the propensity for disturbances in the homeostasis of the substantial aqueous component of the human body during surgical procedures, underscoring the vital necessity for surgeons to demonstrate proficiency in fluid modulation and electrolyte equilibrium. The allocation of electrolytes within the body is expounded upon, with a specific focus on pivotal ions such as sodium, potassium, magnesium, and calcium, elucidating the physiological significance of sustaining normative levels. Furthermore, the import of acid-base equilibrium is underscored, coupled with an exhaustive analysis of the etiology, manifestations, and therapeutic approaches for disorders like acidosis and alkalosis. This elucidation equips surgeons to judiciously manage fluid-electrolyte equilibrium in their patients, thereby contributing to the efficacious execution of surgical interventions.

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CHAPTER IV

DISASTER, TRAUMA AND WAR SURGERY

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1. Introduction

Disasters are defined as "causing huge amounts of damage, destruction, human suffering, an unpredictable and generally sudden event requiring national and international aid in overwhelming regional capacity." [1]. While these events may be originated by the earth's natural procedures like earthquakes, hurricanes and floods, they might also be caused by human -originated disasters and deliberate actions or their negligence such as armed conflicts, industrial and nuclear activities and fires. The majority of the uncovered needs for urgent and primitive surgery have been concentrated on low and medium paid countries: one third of the world's poorest population benefit from solely around 4% of all surgeries carried out [2-4]. While surgery treatment is expected to be much more critical than normal occasions, surgery/ obstetric situations are additionally continued to require ordinary burden and urgent care [5]. Life and limb saver surgery treatment is the first priority in disaster, trauma and war surgery. However, exclusive surgery options will be presented if the best possible standards are available [6].

2. Blunt and Penetrant Abdominal Traumatized Patients

In disaster and war occasions, if both blunt and penetrant abdominal traumas aren't diagnosed and treated early enough, they could lead to deadly

hidden injuries. Considering injury time and it's mechanism, the treatments adopted and carrying out all systematic examination, in particular, physical examination will be the most reliable part in penetrant injuries. The most important decision is determining whether the patient is in need of urgent surgery or not. Blunt abdominal traumatized patients may appear with various pathological mechanisms [7]. Sudden and distinct increase in inner abdominal pressure, the organs inside the abdomen, laceration and rupture; similarly, the strengths imposed to the front walls of the blunt might lead to the organ injuries since the organs inside the abdomen are compressed between vertebral and rib cage. Injuries caused by gun fire carry higher mortality risk than the ones caused by sharp penetrating objects [8, 9]. Additionally, gun fire injuries relate to eight times higher death tolls than the abdominal knife injuries due to higher kinetic energy [10]. Bullets, grenades, as well as postexplosion dashing objects might result in various kinds of injuries. In these kinds of injuries, abdominal traumas are often the ones requiring surgical interference. Surgical methods in patients with blunt and penetrant Abdominal trauma are arguable in compared with others.

3. The First Evaluation of Blunt and Penetrant Abdominally Traumatized Patients

The first evaluation is determined by the most life- threatening injuries. Having limited resources and in the situations in which the injuries are not able to be evaluated and managed more than once, primary detective injuries must be managed. In multi -traumatized patients, if there's collective trauma, a quick evaluation and triage is required. Extreme bleeding, as a preventable Primary death cause, needs to be controlled. Primary evaluation includes air way evaluation, maintenance of respiratory and ventilation continuity, and evaluating the circulation. All patients have to be undressed completely and examined systematically. Hypothermia must be prevented if possible and has to be treated as soon as it's detected. It shouldn't be forgotten that hypothermia can result in both multiple organ failure [11] and coagulation [12].

In conscious patients with blunt abdominal trauma, especially when risk factors for abdominal injury are present, the most reliable symptoms and signs are abdominal pain, abdominal tenderness, and peritoneal irritation. Ecchymosis in abdomen walls, abdominal distention and reduction in intestines noise can be the abdominal trauma symptoms. In a hemodynamically stable patient

lacking extra abdominal injury and abdominal examination being normal are the indication of low possibility of the injury inside the abdomen [13, 14].

Peritoneal irritation symptoms, hemodynamic instability ve hypotension, hematoma or rectal hemorrhoids are the indicators of urgent laparotomy in patients with evisceration. In the patients with no urgent laparotomy indications, physical examination is both susceptible and specific in order to detect serious intra abdominal injuries. For laparotomy in the patients with no distinct indications, abdominal scans can be used or local wound exploration, serial physical examinations, diagnostic peritoneal lavage or diagnostic laparoscopy can be applied. It's believed that the evisceration of omentum and abdominal organs require laparotomy due to increased gastrointestinal perforation [15, 16]. The rate of intra-abdominal injury is higher in these patients than in those without [17]. Additionally, there is a risk of retro peritoneal and peritoneal in back and flank penetrant traumas.

4. Radiological Evaluation

Hemodynamically unstable patients are required to carry out a few limited tests. Direct radiographs of the chest and abdomen should only be performed if there is sufficient time. Getting these done mustn't postpone the surgery. The stable patients must be checked out by taking direct abdominal graphs ambulantly or lateral decubitally to see if there is a shrapnel, bullet or free air inside abdomen.

Sonographically focused trauma evaluation (FAST) is generally a significant way for unstable patients [18, 19]. FAST is primarily used to determine pre cardiac and infra peritoneal blood and it's more reliable to determine the intra abdominal injury signs than any physical examination ones. In unstable patients, FAST has several benefits in guiding which cavity (thoracic or abdominal) to be entered in, before planning the surgery strategy. It identifies the pre cardiac liquid and can be helpful to diagnose hemopnomotorracsin as well as tamponage.

For intra abdominal injuries, in low clinical suspicion situations, tomography is used instead of laparotomy to evaluate the penetrantally injuried patients. To eliminate intra abdominal injury and Manage the penetrant abdominal trauma without surgery, There is a potentiality for the injuries to be abducted in occasions in which relying on the diagnostic methods resources is restricted. Applying Tomography and ultra sonography in penetrant abdominal

trauma are considered in stable patients and is an injury mechanism which can be thought to be an abdominal injury with no distinct surgery indication.

These diagnostic methods must merely be trusted in situations in which there is a fine follow up available and the patients aren't in need of long transplantation and quick surgery interference. Patients transfer shouldn't be delayed in The situations in which the first facility provided needs higher amount of care. If transfer is required the process must commence as soon as possible.

5. Thorax Injuries

Thorax traumas are connected with hemothorax and pneumothorax. The injuries with O2-co2 transition of lung parenchyma lead to sudden function loss meanwhile trigger the sytokine storm. It may result in hematoma in main veins and myocard [20, 21] . Mortal injuries are mainly related to heart, vein injuries and tension pneumothorax [22]. In blast types of injuries, it causes alveolocapillar membrane and by bronchioles pressure breaking up alveoli vascular fistula and air emboli. Hepatisation or known as wet lung syndrome creat dyspnea or hypoxia [23, 24].

90% of patients needing surgery treatment can be treated by tube thoracotomy and closed chest drainage. Early diagnosis is extremely essential in patients suffering from the remained 10% . These are the life threatening injuries, some of which may require thoracotomy. If chest tube is attached, it must be applied to an area apart from the wounding area and isolation pneumothorax rate being low, wide lumen tubes should be preferred considering hemothorax presence.

Massive hemothorax (1500 ml bleeding and 20 ml/kg as soon as it's attached, and subsequently 200-300 ml/h or 500 ml bleeding per hour), massive air leakage, heart tamponade and open pneumothorax, esophagus and large vein injuries and systematic air emboli are the urgent thoracotomy indication of cardiac tamponade[25]. Clamshell thoracotomy in the supine position by making a 4 cm thoracotomy incision in the 5th intercostal space or starting from the sternum, extending the incision from the 4th intercostal space to the posterior axillary line and anterolateral or posterolateral thoracotomy can be performed.

6. Amputation Surgery

In natural disasters or some of ballistic injuries, limbs may be required to be amputated traumatically. Decision on Amputation must be made during the wound evaluation. Skin and bone are relatively more resistant to trauma

than muscle tissues. Contamination can be followed along with facial leveling. Contamination and devitalization of the tissues are generally more comprehensive to be observed at the beginning. However, it should not be forgotten that the important factor determining the level of amputation is soft tissue injury, not bone injury.

It creates serious non-reversible diseases in tissues, deterioration in vascular circulation, ischemic phenomenon, uncontrolled bleeding, and inflectional amputation indications. Before deciding on the amputation, the surgeon should take lots of factors into consideration and clarify the levels to be held if amputation is done. Amputation must always be done under torniquet to reduce blood loss as much as possible. Surgery strategy involves the excision of dead and contaminated tissue, and preservation of the best functional level of the tissue. The wound shouldn't be left open and it must be kept dry and bulky and dressed sterilized- like[26].

7. Approach to Scalp Traumas

Examination of the patients with scalp trauma begins with identification of Glasgow coma scale score. Clinic Glasgow coma scale (GCS) is used in any kind of acute medical and trauma diseases to identify the impaired consciousness level[27]. Glasgow coma scale is comprised of three parameters: eye (E), verbal (V), and motor response. The first examination of the patients after the trauma should include the scalp inspection, evaluation of the skin cuts and sedimentation if available, periorbital ecchymosis and otorhea/rhinorrhea evaluation and attention should be paid to facial asymmetric.

Scalp traumas have lots of different mechanisms. Generally speaking, they can be divided into two main categories: closed and penetrant traumas. Injuries related to closed scalp traumas are more common. The blast kind of scalp traumas are the ones in which the patients have been exposed to explosive forces [28].

Fast evaluation, the common area of use, contradiction and lacking accurate diagnosis, brain tomography analysis has provided the first preferred visualization method [29]. Additionally, computational tomography enables to evaluate the simultaneous epidural and subdural hematoma, intracranial hemorrhage and edema.

Epidural and subdural hematoma are common urgent traumatized brain surgery requiring collection discharge to reduce the pressure impact [30, 31]. The subdural hematoma with the thickness of 10mm or more than 5mm average

line dislocation should be surgically discharged independent of GKS [32, 33] . The Burrhole technique involves opening a burr hole from the area above the maximum hematoma thickness after the patient's head is rotated approximately 45° from the side of the hematoma and fixed. Hematoma cavity is aplenty irrigated until the drainage liquid flow is clear. Henceforward, the wound is closed double-like to prevent any contact with hematoma-atmosphere [34].

8. Approach to Neck Traumas

Due to its complex anatomical structure, neck is vitally important since the post trauma methods are troublesome. The significant injury risk in vital structures of the neck are connected with the trauma formation. In the gunshot injuries approximately 50% of victims (higher in high-velocity weapons) have significant injuries, whereas in stab wounds this risk may be only 10% to 20% [35] .

For clinical approach and diagnostic aims, neck is divided into three parts: 1,2 and 3. Site definitions in penetrating trauma have anatomical, diagnostic, and management implications.

Zone 1: It's the area between the Collarbone and cricoid cartilage. This area includes nameless veins, joint carotid artery's exit, subclavian veins and vertebral artery, biracial plexus, trachea, esophagus and the vital structures such as lung apexes. In addition, surgical exploration and access may be difficult in this region due to the presence of the clavicle and bone structures of the thoracic inlet..

Zone 2: it's the area between the cricoid cartridge and mandibula angle. This area includes the carotid and vertebral arteries, internal jugular veins, trachea and esophagus. It's relatively easy for this area to be accessed for clinical examination and surgical exploration. Neck is the widest and most frequently injured area.

Zone 3: it's the area between the mandibula angle and the skull base. This area includes the distal carotid, vertebral arteries and pharynx. It's less convenient to examine this area physically and is difficult to be explored during the surgical evaluation due to being too close to the skull base [36].

The death toll in penetrant neck injuries is 10%. Vein injuries are the most common causes of death. Zone 1 is the highest risk-bearing area for neck base injuries. Prominent causes of delayed mortality are the esophagus injuries which might not be distinct at the first application [36]. Second area, which

is the most frequently injured part, is the easiest to be accessed surgically. However, exploration and vascular control are more difficult in 1st and 3rd region injuries [37]. If surgical treatment is necessary in area 1 traumas, thoraxic and cervical approaches are generally applied simultaneously; sternotomy, anterior throracotomy, clavicle resection or first rib resection might be required [38-40]. Since it's easy to access, traumas in the second area, apart from the injuries or the situations in which more than one injury is accompanied, surgical exploration is recommended in wholesymptomatic patients without the need of subsidiary tests [41]. There is an injury potentiality in massive blood vessels and cranial nerves close to skull base in the third area. Attention should be paid to the asymptomatic possibility in The first reference of the patients suffering arterial injuries. It shouldn't be forgotten that the interventional radiology in vessel injuries can be beneficial and craniotomy may be required in the patients in need of surgical exploration.

9. Fasciotomy and Compartment Syndrome

Acute compartment syndrome (ACS) consists of the post trauma extreme bleeding in patients, leading to edema- like ischemic pain and probable soft tissue damage which cause the reduction of tissue perfusion and circulation. It's often observed in legs. It usually develops after fracture or crush injuries.

Compartment syndrome clinical features were defined as pain, pallor, paresthesia, paralysis, and pulselessness. The compartment pressure endangering the perfusion develops when the diastolic pressure is increased between 10 and 30 mmHg; muscle oxygenation is reduced when the tissue pressure has approached the average artery pressure [42, 43]. Intra compartment pressure measurement is used in diagnosis [44].

Acute compartment syndrome may appear in long bone breakage, crush injuries, thermal burn, and the injuries related to the penetrant traumas or post vascular injuries. Compartment pressure Measurement is a significant coadjutor to diagnose the acute compartment syndrome. If possible, the compartment pressure measurement is required while deciding on applying or not applying fasciotomy.

Extremity fasciotomy is the only treatment known for acute compartment syndrome. Leg is the most frequently affected area in low extremity that requires fasciotomy [45]. In acute compartment syndrome, if the muscular compartment is not diagnosed timely and decomposition is not done, they may result in extremity necrosis and mortality.

Rhabdomyolysis and acute renal insufficiency are commonly related to muscle necrosis; intravenous liquids and dialysis are the treatments [46]. Deficient fasciotomy may create the necessity to revision fasciotomy for lengthening the fascial space or opening an unnoticeable compartment.

10. Approach to Retroperitoneal Traumas

Retro peritoneal traumas are among the main causes of mortality and morbidity at any age range. The post trauma injuries in this area hold high mortality rates [47] .Considering its complicated anatomy, retro peritoneal injuries treatment may differ at a vast scale. The retroperitoneum includes the gastrointestinal, vascular, musculoskeletal, visceral, and vascular structures. [48]. It's been divided into three parts;

Zone 1: it's the central area which reclines from the diaphragm to aorta bifurcation. It includes the aorta, vena cavity, the origins of renal visceral vascular structures, duodenum and pancreas.

Zone 2: it's the area between aorta bifurcation and diaphragm with its lateraled toldt fascia, it's restricted by medialde renal veins. It includes the adrenal glands, kidneys, renal arteries, ureter ve ascendant ve descents colons.

Zone 3: it's the retro peritoneal area of aortic bifurcation of distalis. It includes the internal and external iliac vessels, the last part of ureter, distal sigmoid colon and the rectum.

The first aim of the surgical treatment is carried through three points: controlling the bleeding area, controlling the gastrointestinal contamination and diagnosis-purposed abdomin emptinesses review. The isolated vascular injuries which aren't urgent laparotomy indications can benefit from the non-surgical treatment including intra vascular treatment; surgical indication is available in bleeding or arterial thrombosis situations. To access the supramesocolon area, with both pancreas tail and spleen, Mattox maneuver is applied in kidney and the left colon containing medial mobilization. Kocher maneuver can be alternatively applied. On the contrary, the access to infra mesocolon area, known as cattle-braasch maneuver, eases the portal vein and inferior cavity evaluation, requiring the thin intestine and right colon cephalic and medial mobilization [49].

There are two treatment approaches to traumatic retro peritoneal hematoma operatively and conservatively [50]. Retro peritoneal hematoma originates from ruptures relating to injuries in solid organs, blood vessels or intra peritoneal organs. In penetrant injury cases, in most traumatic retro peritoneal hematoma, abdominal visceral injury can be accompanied and laparotomy must be done. In

blunt traumas, if there's growth in hematoma, surgical exploration is required providing there's pulsatile mass.

11. Approach to Pelvic Traumas

Pelvic trauma is one of the most complicated managements regarding the trauma and is observed in 3% of skelton injuries [51, 52]. Mortality remains high, especially in patients with hemodynamic instability due to massive blood loss, difficulty in achieving hemostasis, and related injuries. As a result, A multi-disciplinary approach is greatly significant for managing the resuscitation, taking bleeding under control, and managing bone injuries, particularly in the first hours of the trauma [53].

Pelvis correlates anatomically with a range of vascular structures. Aorta separates the common iliac arteries in approximately L4 level. Superior gluteal artery is the most frequently injured vessel in pelvic trauma; it separates from the internal iliac artery. In pelvic traumas, the nerve injuries are observed less than the vascular ones. Plexus injuries creat most of the post pelvic trauma nerve injuries.

World society of emergency surgery divides pelvic traumas in three categories: mild, medium and severe [54]:

- First degree (mild) : the patient is hemodynamically stable with stable fractures
- Second and third degrees (medium): the patient is hemodynamically stable but unstable fractures are available
- Fourth degree (severe): traumas in which the patient is hemodynamically unstable

In hemodynamically stable pelvic fractures, if there's an evidence of arterial bleeding in pelvic tomography, they must directly approach the angioembolization. 85-100% of the angioembolization cases are successful [55]. Angioembolization is generally carried out by the femoral artery approach. Unless femoral artery is used, left brachial or axillary artery can be used.

In hemodynamically unstable patients with pelvic trauma, bleeding control and stabilization of the hemodynamic status, followed by temporary mechanical stabilization, should be controlled for complications and accompanying injuries. In hemodynamically unstable pelvic traumas, bleeding, in 80-90% of the cases, is created by the presacral and paravasical

venous plexus and as a result of venous bleeding of spongiform bone surface depending on sacral and iliac fractures [56]. Preperitoneal pelvic packing is a commonly used technique to take bleeding under control hemodynamically unstable pelvic fracture. Pfannenstiel, paramedical or infra umbilical incision can be used for packing.

Sigmoid colon and intra peritoneal rectal injuries should be debrided and repaired whenever possible injuries consisting less than 50% of its surrounding, are appropriate for primer repair. Injuries in which the primer repair is not appropriate, anastomosis resection is generally applied with or without faecal diversion [57]. In Injuries consisting more than 50% of its surrounding, resection might be required in colon or rectum. Afterwards, the primer anastomosis or diversion can be done.

Bladder injuries may require repairing. The Injury place emerges the management. Extra peritoneal bladder injuries are generally are treated non surgically; intra peritoneal bladder injuries require surgical repair.

12. Conclusion

The management of patients with traumatic injuries presents a variety of challenges. Patients require multidisciplinary evaluation, securing the airway and breathing, hemorrhage control, resuscitation, and stabilization in the emergency department and possible operative intervention prior to inpatient admission. For the combined burn/trauma patient, the immediate management priority is stabilization and resuscitation of the patient from a trauma/management of injury perspective.

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CHAPTER V

BLUNT ABDOMINAL TRAUMA

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1. Introduction

Blunt traumas occur after traumatic energy affects a large area. It is divided into two: high-energy trauma and low-energy trauma. Examples of high-energy trauma are out-of-vehicle traffic accidents, motorcycle accidents, high-speed in-vehicle traffic accidents, falls from height, and traumas related to construction machinery. Examples of low-energy traumas are being beaten with a stick, falling off a bicycle, slipping and falling.

Blunt trauma often affects multiple systems like abdomen, thorax, spinal cord, extremites or cranium. Therefore, multidisciplinary management is required for these patients. The patient with blunt trauma is first initiated with airway, respiratory and circulatory system control (ABC). Then all systems are checked one by one. In the meantime, a detailed anamnesis is taken from the patient if the patient is conscious, or from the witnesses if the patient is not conscious. If anyone has seen the accident, information about the energy of the trauma can be obtained from him. However, in cases where there is no witness, the situation of being thrown out of the vehicle in traffic accidents, the death of other accident victims, and the delay in the arrival of the rescue team provide information about the severity of the situation. While less injuries are expected in low-energy traumas, the possibility of fatal injuries should not be forgotten.

In traffic accidents, vehicle speed, impact angle, location of the victim in the vehicle, ejection from the vehicle, presence of seat belt, airbag status or steering wheel damage should be questioned. In other blunt traumas, the type of trauma, its severity, and the affected body area are questioned in detail.

In in-vehicle traffic accidents, use of seat belt should suggest pancreatic injury, not using it and steering wheel damage should suggest liver spleen injury. We should consider rupture of the diaphragm in falling from a height or abdominal blows that cause sudden increase in intra-abdominal pressure. If the blow was taken from the lateral side, the possibility of injury to the organs in that anatomical location is higher. It should not be forgotten that all organ injuries can occur in comlex trauma such as being thrown from a vehicle.

In blunt abdominal trauma, organs with limited intra-abdominal mobilization are more likely to be injured. Therefore, the liver, spleen, kidney are injured more often.

2. Approach to the Patient with Blunt Abdominal Trauma

The patient with blunt abdominal trauma is first started with ABC control, as in all emergency approaches. Then the patient should be monitored. Simultaneously, the patient's blood tests including hemogram, brief biochemistry, coagulation, blood gas, blood group and cross-mach are studied and fluid replacement is started.

In a patient presenting with blunt abdominal trauma, wooden abdomen, positive DPL or intra-abdominal fluid on FAST with hemodynamic instability are indications for laparotomy, directly. However, a possible alcohol or drug use, spinal cord injury that causes sensory loss may cover up the examination findings. Therefore, hemodynamic instability is more significant. If necessary, it should not be too late for further examination and surgery.

With the advancement of technology, the order of radiological evaluation has changed. While DPL was previously recommended as the first examination, it has now been replaced by FAST. FAST is a mobile method that gives rapid results, especially in patients with unstable hemodynamics. However, it has some limitations. Abdominal CT with IV contrast is very useful in demonstrating intraperitoneal and retroperitoneal events.

FAST is used to detect intraabdominal bleeding in patients presenting with blunt abdominal trauma. Three areas are examined: Morrison's pouch, left upper quadrant, and pelvis. It is sensitive to show 250 ml and more liquid. However, it cannot distinguish the focus of bleeding or fluid. In a patient with FAST positive, if the hemodynamics is stable, a focus is sought with further examination, and if unstable, laparotomy is performed.

In a patient with FAST negative, DPL can be performed if there is still suspicion of an intra-abdominal event. A blood flow of 10 ml or more is

considered positive as soon as the DPL catheter is inserted. If less than 10 ml of blood comes out, 1 lt of saline is given from the catheter and washed. Parameters and positivity criteria in the aspirate are given in the table.

Table 1. Criteria for "positive" finding on diagnostic peritoneal lavage in abdominal trauma(1)

Red blood cell count	>100000/mL
White blood cell count	>500/mL
Amylase level	>19 IU/L
Alkaline phosphatase level	>2 IU/L
Bilirubin level	>0.01 mg/dL

IV contrast CT is performed in patients with stable hemodynamics. High-quality CT images reveal bleeding foci, extravasation and perforation more clearly. This is very important for patient management. Because, recently, due to the developing technology and treatment options, non-operative follow-up rates have increased especially for solid organ traumas, and there have been some changes in operation techniques. Survival has increased dramatically with the development of transfusion protocols. Patients who are clinically appropriate can be intervened with angiographic interventions such as embolization and stenting.

If there are findings such as luminal organ perforation, stage 4-5 spleen injury, pancreatic injury, and fluid suggestive of excessive bleeding into the abdomen in the CT scan of patients without peritonitis, laparotomy is performed. Wide resections and ostomy rates decreased and anastomosis rates increased in patients who underwent surgery for intestinal pathologies.

There are some things that should not be forgotten when the patient with blunt abdominal trauma comes to the emergency room. If the patient is conscious, the last tetanus vaccination time should be learned, and if the patient is not conscious, tetanus prophylaxis should be performed at that time.

There are many reasons to develop hypothermia in patient. Some of these are hemorrhage, peeling the patient for examination, cold transfusion of blood products and fluids. So that, the patient should be warmed with warm blankets and heaters to prevent hypothermia from the moment of admission, and the replacement products should be warmed before transfusion.

Antibiotic prophylaxis should be applied if operation is planned. Antibiotic selection should be based on the type of injury. Anaerobic antibiotics should be added in gastrointestinal lumen injuries.

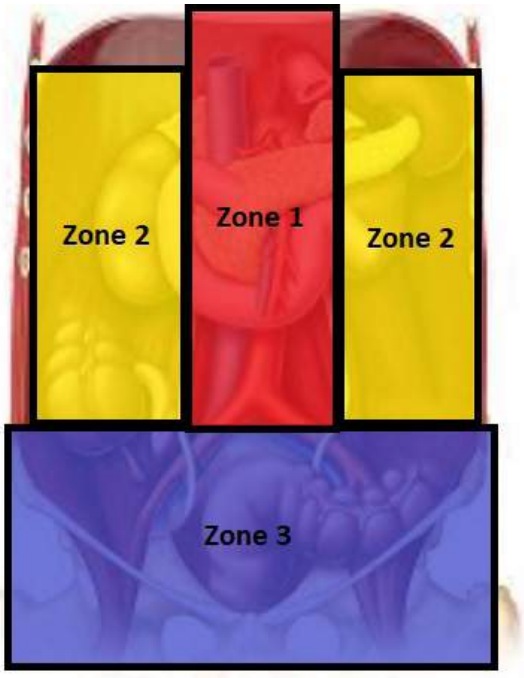
If there is no extremity fracture, compression stockings should be worn and LMWH should be started as soon as bleeding control is achieved for the DVT prophylaxis.

3. Emergency Abdominal Exploration

The midline is chosen for abdominal exploration. In patients with unstable hemodynamics, a scalpel should be preferred for rapid access to the abdomen. Bleeding from the incision is not important at this time and bleeding control is done at the end of the surgery. As soon as the surgeon enters the abdomen, the existing hematoma and blood are quickly evacuated with the help of laparotomy pads and aspirator. If the patient's systolic pressure is 70 mm or less, clamping or compression is applied to the aorta at the level of the hiatus. Then, the spleen and liver, which are the most injured organs in blunt abdominal trauma, should be checked at first. If there is any bleeding in these organs, they are first wrapped with laparotomy pads. If there is persistent bleeding in the liver, turning the hilum with a Pringle maneuver and tourniquet provides a more active bleeding control. The spleen is first mobilized and exposed. For this, blunt dissection is performed over Gerota's fascia, it is mobilized together with the tail of the pancreas. Then, it is checked whether there is bleeding in the mesenteries in the inferior of the transverse colon. After this stage, vascular injuries should be controlled first.

4. Approach to Vascular Injuries

Large intra-abdominal vessels are located in the retroperitoneum or mesentery. If the patient is stable, the injured vessel is detected with CT first. If the vessel cannot be detected, treatment is planned according to the area with the hematoma. For this purpose, the retroperitoneum is divided into three regions: Centromedial region (Zone I), lateral regions (Zone II) and pelvic region (Zone III). Zone I injuries are usually treated with surgery. For Zone II injuries, angiographic intervention or surgery may be required in the presence of a growing hematoma. In stable patients, conservative follow-up is performed. Zone III injuries are mostly due to pelvic fractures and are usually venous bleeding. If there is ongoing extravasation, angioembolization or surgery is needed.(2)



Picture 1. Retroperitoneal zones

If vascular injury is present, it should be demonstrated whether it is supracolic or infracolic. In intra-abdominal vascular injuries, repair is required for the aorta, superior mesenteric artery (SMA), a.hepatica propria, renal artery, iliac arteries, vena cava, renal vein, portal vein, and generally superior mesenteric vein (SMV).

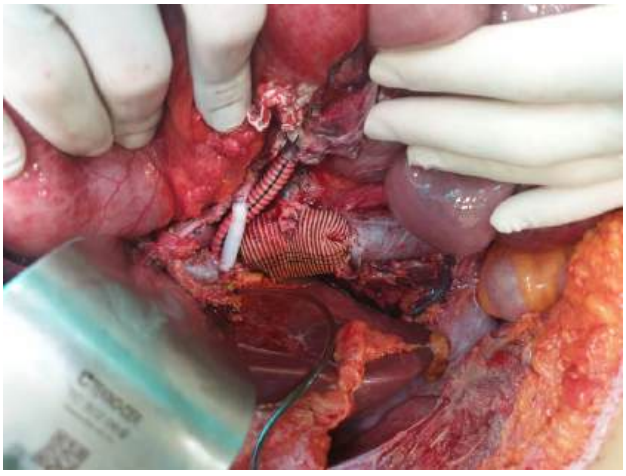
The left colon, spleen, pancreatic tail and stomach are deviated to the right but the left kidney is hold on its place to exhibit supracolic aorta, SMA, left renal artery, truncus coeliacus and its branches. This procedure is called the left medial visceral rotation-Mattox maneuver.

For aortic injury present in the superior part of the celiac truncus, the left diaphragm is opened and a thoracotomy is performed. The aorta is clamped to control bleeding. It is repaired as soon as possible and then the clamp is opened. The clamping time should not be longer than 30 minutes. In case of infrarenal aortic injury, midline hematoma is observed. Similarly, the aorta can be clamped and repaired with the left approach.

Ischemic complications are less common in celiac trunk injuries. To control bleeding, the artery may be ligated first. Then anastomosis can be made to an artery such as the aorta, SMA.(3)

Intervention for SMA injuries differs according to the Fullen classification. Fullen zone 1 is between the aortic root and the inferior pancreaticoduodenal artery (IPD), zone 2 is between the IPD and the middle colic artery, zone 3 is the intramesenteric part after the middle colic artery but before the branching, and zone 4 is distal of zone 3.(4) The best approach for zone 1 is left medial visceral rotation. Zone 2 is reached from the Lasser sac region between the transverse colon mesentery and the pancreas. Zones 3 and 4 are located infracolic and can be reached from the mesentery. Proximal SMA injuries have high mortality and morbidity. If there is a chance for intervention at that time, end-to-end anastomosis to itshelf or anastomosis to renal artery or splenic artery can be performed.(5)

To intervene in the inferior vena cava (IVC) injury, the right colon and the duodenum are exposed by mobilizing medially. This is called right medial visceral mobilization or the Catell-Braash maneuver. Repair is essential in suprarenal injuries, but ligation can be performed in infrarenal injuries. The superior and inferior of the injury and, if necessary, the renal vein are clamped and repair is perforemed. If there is an injury to the posterior wall, it is repaired bt rotating of VCI. If needed, grafts can also be used for repair. In retrohepatic vena cava injuries, the liver must be mobilized for repair after bleeding control is achieved.(3) It was observed that there was no significant difference in mortality between infrarenal vena cava ligation and repair, but morbidity increased in ligation. Therefore, ligation may be preferred in cases of uncontrollable and mortal bleeding.(6) Elevation and elastic bandages should be applied to the lower extremities, since edema will develop.



Picture 2. Anastomosis with graft to portal vein and vena cava

Injuries to the iliac vessels, which can be observed in pelvic fractures, present with hematoma in the pelvis or lateral compartments. Ligation may be preferred when there is an isolated internal iliac artery injury. However, in the presence of external or common iliac artery injury, repair is necessary. It is recommended to clamp the vessel first and then repair it. If necessary, grafts can be used in cases such as tissue loss.(3)

In portal vein injuries, hematoma is observed in the hepatic hilum and in the right region. Injuries close to the hilar region are treated with the Catell-Braasch maneuver. Transection of the pancreas from the trunk may be required in SMV and retropancreatic portal vein injuries. Ligation may be preferred for uncontrollable bleeding.(3) Severe intestinal edema and associated abdominal compartment syndrome may develop in patients undergoing ligation.

5. Approach to Liver and Biliary Tract Injuries

The liver is the second most frequently injured organ in blunt abdominal trauma.(7) We can classify liver injuries into three: parenchymal injuries, vascular injuries and biliary tract injuries. Recently, non-operative follow-up is more preferred in patients presenting with blunt abdominal trauma. Because, angioembolization, ERCP, vascular or biliary stents and their widespread use have increased non-operative treatment. However, surgical treatment is required if there is hemodynamic instability, bleeding which require massive transfusion, persistent bleeding that cannot be managed non-operatively, and peritoneal irritation findings.

The liver injury scale made by the American Association for Trauma Surgery (AAST) is given in the table.(8)

Table 2. The liver injury scale of AAST(8)

Grade I	Subcapsular hematoma <10% surface area
	Parenchymal laceration <1 cm depth
Grade II	Subcapsular hematoma 10-50% surface area; intraparenchymal hematoma <10 cm in diameter
	Laceration 1-3 cm in depth and ≤10 cm length
Grade III	Subcapsular hematoma >50% surface area; ruptured subcapsular or parenchymal hematoma
	Intrparenchymal laceration >10 cm
	Laceration >3 cm depth
	Any injury in the presence of a liver vascular injury or active bleeding contained within liver parenchyma
Grade IV	Parenchymal disruption involving 25-75% of a hepatic lobe
	Active bleeding extending beyond the liver parenchyma into the peritoneum
Grade V	Parenchymal disruption >75% of hepatic lobe
	Juxtahepatic venous injury to include retrohepatic vena cava and central major hepatic veins

Stage I-II injuries can be followed non-operatively under service conditions. Patients with injuries above stage II should followed up in intensive care units by monitoring. Abdominal examination, fluid intake and urine output level, blood analysis are followed up at regular intervals. Conservative follow-up and non-operative interventional procedures are evaluated in patients without an indication for surgery. Angiographic intervention may be considered in patients with contrast extravasation in CT angiography who are hemodynamically stable and need 4 units of erythrocyte replacement in 6 hours or 6 units in 24 hours. Angioembolization can also be considered in patients who had undergone surgery and had applied damage control surgery. Patients who are planned for non-operative follow-up should be followed up in centers that have emergency surgery feasibility.

When laparotomy is performed in patients who is going to treated with surgery, damage control must be performed first. In patients with isolated liver injury, if the parenchymal laceration is small and the gleason capsule is preserved, absorbable parenchymal matrix sutures can be applied with a size 0 blunt needle. However, packing with laparotomy pads is applied in

patients with diffuse liver injury. The second surgery must be planned 24-48 hours later in patients whom applied packing. In patients whose bleeding persists despite packing application, they can be operated earlier than 24-48 hours.

Pringle maneuver can be applied in patients with intraoperative massive bleeding. If the bleeding stops, it is understood that the bleeding originates from the hepatic artery or portal vein. In this case, ligation can be applied if the patient is unstable. If the bleeding continues despite the Pringle maneuver, we must consider that the bleeding originates from the retrohepatic vena cava or hepatic vein. In this case, the injury must be found immediately. If the process will take a long time, the suprahepatic and infrarenal vena cava can be clamped to find the injury. If primary repair is possible, primary repair is performed. In patients with tissue loss, graft can be applied. It should not be forgotten that the Pringle maneuver should be released every 20-30 minutes.(9)

The approach to injuries of the extrahepatic portal vein, vena cava and hepatic artery has been described previously. If the patient is stable, the hepatic artery, portal vein, and infrarenal vena cava can be repaired. In unstable patients, ligation can be applied. Liver necrosis may develop in patients undergoing hepatic artery ligation. Cholecystectomy should be performed in patients undergoing right hepatic artery ligation.(3)

Primary repair is not suitable for extraheptatic bile duct injuries. In this case, biliary drainage with T-tube or Roux-en-Y choledocheojejunostomy can be applied. Biliary drainage is a better choice if the patient is unstable.

Bilioma, pseudoaneurysm, liver abscess due to hepatic artery ligation may develop in patients with liver injury. Small biliomas can be followed if they are not infected. Infected and large biliomas and liver abscesses can be treated by percutaneous drainage.(10) In large necrosis, hepatectomy or segmentectomy can be planned after the patient is stable.

6. Approach to Spleen Injuries

The role of the spleen on the immune system is enormous. Infections related to encapsulated bacteria after splenectomy are mortal. The spleen is the most injured organ in blunt abdominal trauma.(7) Splenectomy was previously performed in all splenic injuries, but now, if possible, non-surgical follow-up is performed due to these conditions. The spleen injury scale made by the American Association for Trauma Surgery is used to determine the treatment protocol.(11)

Table 3. The spleen injury scale of AAST(11)

Grade I	Subcapsular hematoma <10% surface area
	Parenchymal laceration <1 cm depth
	Capsular tear
Grade II	Subcapsular hematoma 10-50% surface area; intraparenchymal hematoma <5 cm
	Parenchymal laceration 1-3 cm
Grade III	Subcapsular hematoma >50% surface area; ruptured subcapsular or intraparenchymal hematoma ≥5 cm
	Parenchymal laceration >3 cm depth
Grade IV	Any injury in the presence of a splenic vascular injury or active bleeding confined within splenic capsule
	Parenchymal laceration involving segmental or hilar vessels producing >25% devascularization
Grade V	Any injury in the presence of a splenic vascular injury with active bleeding extended beyond the spleen into the peritoneum
	Shattered spleen

While the risk of rebleeding is higher in the presence of extravasation or grade III-V injuries, rebleeding rates have been reported as 20% for grade I-II injuries.(12) Non-operative follow-up and angioembolization are increasing day by day. Many grade I-III injuries are followed non-operatively, provided that the patient is stable and the center have emergency surgery feasibility. Even some selected grade IV-V injuries can be followed non-operatively.(13)

Surgical treatment is preferred in cases such as the presence of additional injury, hemodynamic instability, high grade injury, diffuse hemoperitoneum. Splenectomy, partial splenectomy and splenorrhaphy operations can be applied in spleen injury. However, splenectomy is almost always performed in adult patients.

Pseudoaneurysm, late bleeding can be seen in patients followed non-operatively; pancreatic tail injury, gastric injury during short gastric vessel ligation, pancreatic fistula, subdiaphragmatic abscess and infection complications due to encapsulated bacteria can be seen in patients applied splenectomy. Percutaneous drainage is recommended for subdiaphragmatic abscesses. The ideal time for vaccination against encapsulated bacteria is 14. day after surgery.(14)

7. Approach to Pancreas and Duodenum Injuries

Pancreatic and duodenal injuries are rarer due to their deep and retroperitoneal location. However, it should be kept in mind especially in patients who use belts or have a steering wheel impact in traffic accidents. Due to their location, injuries often occur together. It is difficult to diagnose. The most useful examination is CT with contrast, but repeated imaging may be required.

Parenchymal injury and ductal injury may occur in pancreatic injuries. It is suspected by observing retroperitoneal hemorrhage, peripancreatic fluid or contrast extravasation, and insufficient perfusion of the pancreas on CT. Almost all patients with suspected pancreatic injury undergo surgery. Retroperitoneum must be opened completely and pancreas must be explored totally. It should be followed with a drain to remove corrosive pancreatic enzymes in parenchymal injuries. If we suspect ductal injury in stable patients, we can check with MRCP and ERCP. If the ductal injury is at the left side of the middle colic artery, spleen-sparing distal pancreatectomy can be performed. If the ductal injury is at the right side of the middle colic artery, damage contral surgery is applied in the first surgery and pancreaticoduodenectomy can be applied if the patients condition is suitable. Reconstruction of whipple operation must be completed in the second surgery. Non-invasive methods such as endoscopic pancreatic stent or percutaneous drainage can be applied in selected patients and in appropriate centers, especially for missed injiruies.(15, 16)

If duodenal hemorrhage is considered without perforation, conservative treatment with NG decompression and parenteral nutrition is recommended. If there is a possibility of perforation, laparotomy is performed. Retroperitoneal hemorrhage, free air, or contrast extravasation on CT suggest duodenal perforation. Primary repair can be applied in first, third and fourth part injuries and anterior of second part injuries that do not affect the papilla of Vater. Resection is performed in first-party injuries with large tissue loss or impaired perfusion. The pylorus and antrum are mobilized and anastomosis is applied. Duodenojejunostomy can be performed if primary repair is not possible in third and fourth part injuries. There is no standard treatment for second part injuries. Primary repair, primary repair with gastrojejunostomy, tube of duodenostomy, choledochejunostomy, pancreaticojejunostomy, pancreaticoduodenectomy can be applied due to condition.(17)

Reconstruction of pancreatic and duodenal injury can take times. If patient is unstable we must only damage control surgery in the firsth operation. So we

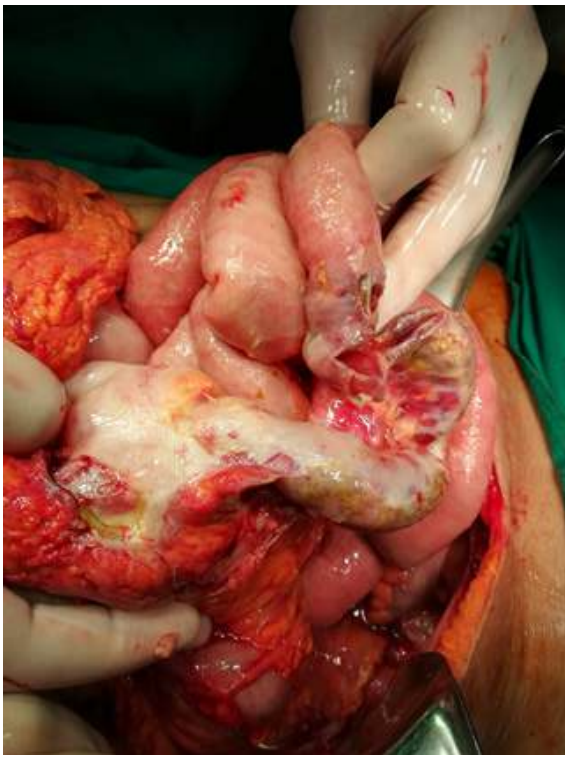
must aim that extraction of gastrointestinal, biliary or pancreatic fluids from the abdomen. Second operation must perform for reconstruction after patient become stable.

8. Approach to Stomach Injuries

Gastric injuries in blunt abdominal trauma occur due to sudden pressure increase. Laparotomy is applied in full-thickness perforation of the stomach wall. These patients are operated for acute abdomen or intraperitoneal free air or contrast extralumination on CT. The gastrocolic ligament should be opened and the posterior wall of the stomach should be checked in the operation. There may also be mesenteric injuries, but these can be followed conservatively. The repair depends on the location and size of the injury. Primary repair, wedge resection is usually sufficient. Very rarely, total or distal gastrectomy may be required.

9. Approach to Small Bowel Injuries

It has two forms: lumen damage or mesentery damage. Free air is observed in the CT if there is lumen damage. For these patients, laparotomy is indicated. The small intestine should be explored carefully, starting from the ligament of Treitz and up to the colon. If the lumen injury covers one third or less of the lumen diameter, primary repair in the transverse plane can be performed. Segmentary resection and anastomosis can be planned in case of larger injury or several injuries that close to each other. End-to-end anastomosis can be applied, or in unstable patients, anastomosis can also be performed using stapler to be faster.



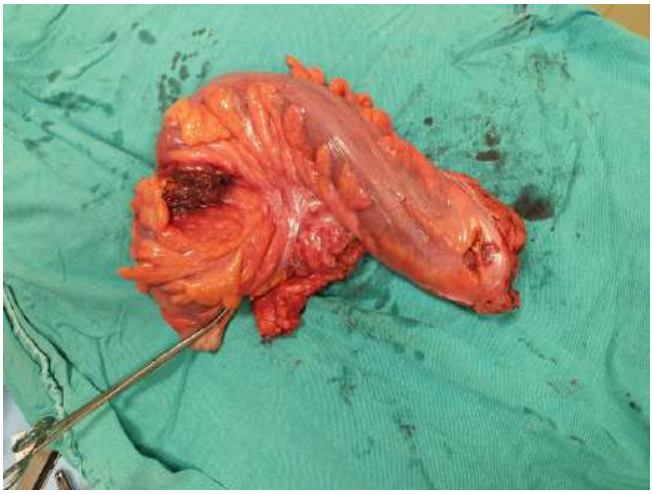
Picture 3. Late laparotomy for small bowel perforation after blunt abdominal trauma

If there is mesenteric injury, hematoma in the mesentery and intraperitoneal blood can be observed on CT. Conservative follow-up can be planned for patients who do not have free air in the abdomen and who have stable hemodynamics. If the injured vessel is thin, there wont going to be problem in the patient, but if a large vessel is injured, the bowel may go to ischemia and necrosis in the late period. Because of that, perforation may develop 3-5 days after trauma. So, we should consider that mesenteric injury may occur in patients with intra-abdominal hemorrhage but no bleeding in solid organs. The necrotic bowel segment should be resected. If the abdomen isn't contaminated and the perfusion of bowel is enough, anastomosis can be applied. Stoma may be preferred in patients with the possibility of persistent ischemia.(18)

10. Approach to Colon and Rectum Injuries

The approach to colon injuries is similar to small bowel injuries. For luminal injuries, primary repair, resection and anastomosis (end-to-end, end-

to-side or side-to-side/sutured or stapled/ileocolic or colocolic), primary repair and proximal fecal diversion (loop colostomy, loop ileostomy), end colostomy can be performed. In patients with mesenteric injury, the presence of ischemia should be carefully controlled, resection should be performed if necessary, and it should be evaluated for the requirement for stoma.(19)



Picture 4. Perforation in two areas of the sigmoid colon

Rectal injuries are slightly different from colon injuries. Since it is located in the pelvic cavity and retroperitoneum, it does not immediately contaminate the intraperitoneum, but its repair is difficult. Simple rectal injuries can be treated with rectal lavage and repair. Fecal diversion with sigmoid loop colostomy can be applied to the patient if needed. In case of fecal contamination to the retroperitoneum, a drain can be placed with a perineal approach. If there is another injury at the proximal of rectum, it may require the Hartman procedure and, rarely, abdominoperineal resection.

The necessity of stoma in colon injuries is decided according to the clinical condition of the patient rather than repair. It is preferred in patients with additional injuries, septic patients, hypotension, mesenteric injury, in whom blood supply is uncertain, or if possible anastomotic leakage will cause serious morbidity.

Complications such as anastomotic leaks, fistulas, stoma complications, intra-abdominal abscesses, retroperitoneal abscess, and necrotizing fasciitis may develop in colon and rectal injuries. Localized abscesses that do not cause

to deterioration of general condition can be drained by percutaneous drainage. Controlled fistulas can be followed conservatively or delayed reoperation can be applied. However, serious complications may require urgent surgery.

11. Approach to Pelvic Injuries

Pelvic injuries in blunt abdominal trauma occur by two mechanisms: injury due to pressure increase, injury due to fractures. If trauma occurs while the bladder is full, bladder rupture may develop due to increased pressure. Bladder rupture should be considered when there is non-hemorrhagic fluid in the abdomen and absence of urine when presence of a foley catheter.

If there is no pelvic fracture, the reltum, vagina, urethra or vessels usually preserved due to the retroperitoneal location of these organs and the strong structure of the pelvis. In the presence of pelvic fracture, these organs may be injured due to the injury of the broken bones. Therefore, vaginal and rectal examination should be done in the detailed examination of the patient, and the possibility of vascular injury should not forgotten in patients with pelvic fracture.

Suggestive findings of urethral injury are bleeding from the eksternal meatus, hematoma in the scrotum or perineum, and palpation of the prostate upper than normal on rectal examination. If these findings exist, a urethrogram should be taken first. If urethral injury is not anticipated, a Foley catheter should be inserted and it should be monitored the amount and quality of urine.

12. Damage Control Surgery and Abdominal Compartment Syndrome

A vicious cycle occurs that leads to death in patients who have severe post-traumatic injury. There are three reasons for this: hypothermia, acidosis and coagulopathy. Etiological reasons for this can be listed as blood loss, massive transfusion, environmental factors, co-morbidities, and cytokine activation due to immune system activation. If this vicious circle is not resolved quickly, it will result in the death of the patient. So, the aim of abdominal damage control surgery is that: control bleeding and to remove gastrointestinal contents from the abdomen. Then, it is aimed to quickly transfer the patient to the intensive care unit and stable it. If the patient becomes stable, he is taken to the second surgery.

It should be repaired as previously mentioned if bleeding due to vascular injury such as the aorta, SMA, iliac artery, suprarenal vena cava inferior, etc. Ligation can be applied if reparing will takes times and it wont be mortal when ligation is performed. Splenectomy and nephrectomy can be applied if needed

in spleen and unilateral renal injuries. Packing is recommended in uncontrolled liver lacerations. Primary repair can be performed if the gastrointestinal injury is minor. In injuries that will require resection, rapid repair should be performed with the help of staplers. In patients with risky anastomosis, ostomy may be preferred. In injuries with bile or pancreatic leakage, the aim should be to provide external drainage to prevent peritonitis.

Primary closure of the abdomen may cause abdominal compartment syndrome in blunt abdominal trauma. Because intraperitoneal hemorrhage or seroma accumulation, intestinal edema, packing application cause to increase of intra-abdominal pressure. On the other hand, it is beneficial to observe the inside of the abdomen in patients whom applied damage control surgery. In these cases, it would be more appropriate not to close the abdomen. The application of an open abdomen and choosing transparent Bogota bag for this application is the most appropriate choice if we think possibility of re-operation.

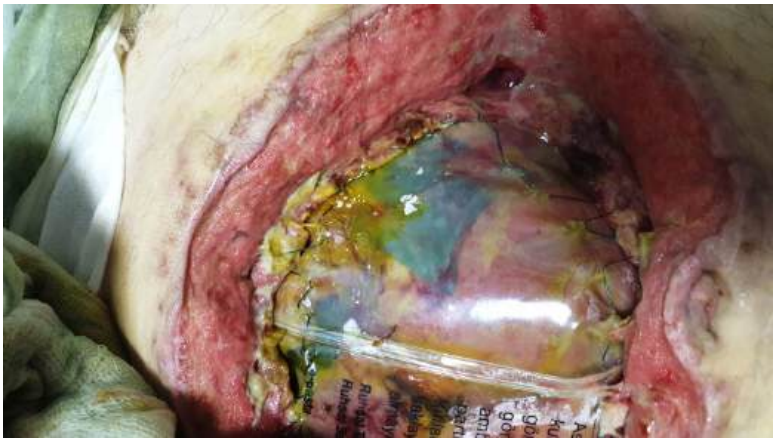
13. Closure of Abdomen

Intra-abdominal hypertension may develop in patients with blunt abdominal trauma treated both operatively and non-operatively. If it is overlooked and this condition progresses, abdominal comartment syndrome may devolop. This is associated with high mortality and morbidity.

In non-operated patients, massive intra-abdominal bleeding, ileus, and fluid loss to intraperitoneum are causes of intra-abdominal hypertension. It should be suspected in patients with signs such as oliguria, hypotension, acidosis and elevated CVP. Intra-abdominal pressure measurement should be performed to these patients. We should not forget that conditions such as pelvic hematoma, bladder injury may cause inaccurate measurement. Patients who develop compartment syndrome should be operated quickly.

In operated patients, intra-abdominal organs may swell in the following situations: intestinal edema, retroperitoneal or intraperitoneal hematoma, ileus, especially those who have been ligated due to venous injury and venous return is impaired. In this situation, the abdomen is not suitable for the primary closure that targeted in every surgery. It would be more appropriate to apply skin closure without fascia clorsure or open abdomen techniques. However, in more severe injuries, open abdomen techniques are applied when the intestines are very swollen. Some of these techniques are: skin closure with a towel forceps, loose suture to the fascia by protecting the intestines with laparotomy pads, Bogota bag or VAC applications. The recent development of intra-abdominal VAC

applications has contributed significantly. Gradual closure of the abdomen and aspiration of the intra-abdominal fluid were achieved. If the abdomen cannot be closed in patients with large abdominal wall defect, the integrity of the abdomen can be provided with dual mesh.



Picture 5. With the Bogota bag method, which is one of the open abdominal techniques, complications that develop in the abdomen such as intestinal fistula that is seen in the picture can be followed.

14. Conclusion

Blunt abdominal trauma management is difficult. Because, it is variable that which organ is exposed to how much damage. And it is usually encountered as multi-trauma. This situation requires multidisciplinary treatment. While some patients can be followed without surgery, some patients may die within minutes due to serious injury. Treatment of patients who undergo surgery or not can be long, tiring and complex. This treatment process definitely requires close monitoring and intervention if necessary. The conditions of the health center are also important in terms of the type and benefit of the intervention.

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CHAPTER VI

PENETRATING ABDOMINAL TRAUMA

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1. Introudiction

Trauma or injury can be defined as cellular damage and ischemia reperfusion injury leading to cell death, occurring when the body encounters an energy level that exceeds what it can cope with.(1) Trauma is among the top causes of mortality across all age groups. Therefore, the condition of trauma patients should be assessed accurately and promptly. Resuscitation and stabilization of patients, interventions related to their current condition, or ensuring that they are transported to an appropriate healthcare center with necessary conditions, and reviewing the steps for ensuring that sufficient interventions have been performed, are all essential.(2)

Abdominal region, which contains important organs and vessels, has a high incidence of trauma due to its wide surface area. Therefore, abdominal traumas should be evaluated quickly and systematically, vital problems should be identified, and appropriate treatment approach should be applied to treat them. Thus, morbidity and mortality rates that may occur due to trauma will decrease. Various approach algorithms and systems have been developed for rapid and systematic evaluation of the patient, and their implementation has a significant positive impact on patient outcomes.(3,4)

The classification of abdominal traumas into blunt and penetrating traumas is based on the mechanism of injury, with different diagnostic and therapeutic approaches required for each.

Penetrating trauma refers to injury caused by foreign objects penetrating the tissue. Weapons that cause penetrating injuries are classified according to the amount of energy they produce as low-energy (knife, arrow), medium-energy

(handgun), and high-energy (shotguns, military rifles). Their velocity is the most important determinant of injury potential.(1)

2. Evaluation of Penetrating Abdominal Trauma

In cases of penetrating abdominal trauma, initial evaluation is crucial. The patient should be promptly taken to the emergency department and vital signs should be checked immediately. These signs may include difficulty breathing, bleeding, hypotension, tachycardia, dehydration, and shock. The patient should be stabilized and necessary medical interventions should be performed.

The secondary evalluation of the patient should include a detailed medical history, physical examination and necessary imaging studies. Patients who have experienced abdominal trauma may present with symptoms such as abdominal tenderness, distension and peritonitis. Imaging techniques such as radiological tests, ultrasonography, computed tomography and laparoscopy can also be used.

After the injuries caused by the trauma have been identied,an appropriate treatment approach should be determined. Minor injuries can usually be controlled with conservative treatment, while major injuries may require surgical intervention. The treatment plan may vary depending on the type, location, extent, and severity of the injury.

2.1. Primary Evaluation

The initial evaluation of penetrating abdominal trauma is based on fundamental principles, as with all types of trauma.

The purpose of the initial evaluation is to determine if there is an immediate life-threatening condition and provide appropriate treatment. In Advance Trauma Life Support, this is defined as “ABCDE” (A: securing the cervical spine and ensuring airway patency, B: breathing, C: circulation, D:Disability; E: Exposure and environmental control)

Life-threatening conditions during the primary evaluation are listed in Table 1 and should be treated before moving on to the secondary evaluation. The first hour after a traumatic injury, also known as the golden hour, requires prompt evaluation and stabilization, following the basic principles of Advanced Trauma Life Support, which emphasize the importance of rapid assessment and resuscitation. Bleeding that is not properly managed or identified in cases of abdominal and pelvic injuries can lead to avoidable fatalities.

Table 1. Acute Life-threatening Injuries During The Initial Evaluation	
Airway	• Airway obstruction • Airway damage
Breathing	• Tension Pneumothorax • Open Pneumothorax • Massive air leak in tracheobronchial injuries • Flail chest with underlying pulmonary contusion
Circulation	• Hemorrhagic Shock o Massive hemothorax o Massive hemoperitoneum o Mechanic unstable pelvic fracture with bleeding o Loss of blood from the extremities • Cardiogenic Shock o Cardiac tamponade • Neurological Shock
Disability	• Intracranial hemorrhage/mass lesion • Cervical spinal injury

A: Assessment of the airway eith protection of the cervical spine the goal is to ensure a safe airway, and the available options include evaluating the airway, nasotracheal or orotracheal intubation, cricothyroidotomy and tracheostomy.

1. Evaluation of airway patency in a trauma patient
2. Applying a non-rebreathable mask to maximize oxygenation.
3. Pulse oximetry monitoring
4. Performing jaw thrusts to maintain an adequate airway.
5. Airway aspiration
6. Nasopharyngeal airway and oropharyngeal airway interventions
7. Performing balloon-mask ventilation
8. Insertion of a supraglottic or extraglottic device.
9. Determination of indications for a definite airway.
10. Oral endotracheal intubation.
11. Determination and application of needle cricothyrotomy indications
12. Determination and application of surgical cricothyrodotomy indications

B: Respiration and Ventilation After securing a safe airway, the aim is to achieve sufficient oxygenation and ventilation. The options include supplemental oxygen therapy, monitoring pulse oximetry, emergency decompression with needle thoracostomy, tube thoracostomy, and direct.

1. Assessment of proper breathing and oxygen saturation in a patient with trauma.

2. Recognition of trauma patients who are experiencing difficulty in breathing.

3. Evaluation of chest X-rays of trauma patients

4. Needle decompression or thoracostomy tube insertion

5. Proper management of pain following chest trauma

6. List the steps necessary to safely transport a trauma patient with respiratory problems.

C: Circulation and Control of Bleeding After achieving adequate oxygenation and ventilation, the primary focus is on maintaining circulation. The options include establishing peripheral intravenous access, intraosseous access, femoral catheterization, subclavian catheterization, jugular catheterization, cut-down saphenous vein catheterization, manual compression, tourniquet application, pelvic stabilization, performing a focused assessment with sonography for trauma (FAST), pericardiocentesis and placement of a pericardial drain, and performing a resuscitative thoracotomy.

1. Diagnosing the presence of both compensated and decompensated shock

2. Determining the type of shock present

3. Choosing the appropriate fluid resuscitation

4. Applying a staged approach to control external bleeding using direct pressure, wound packing, and tourniquet application

5. Considering alternative options for vascular Access

6. Applying a pelvic stabilization device for pelvic fractures and understanding the indications and contraindications for the use of traction devices for femur fractures

7. Recognizing the need for reassessment of the patient's response to treatment and the need for additional resuscitation

8. Identifying which patients require definitive hemorrhage control (i.e., operative and/or catheter-based) and/or transfer to a higher level of careD:

D: Disability and conscious evaluation

1. Conducting a brief neurological assessment, which involves evaluating the patient's Glasgow Coma Scale (GCS) score, checking the pupils for abnormalities, and assessing the patient for signs of lateralization

2. Defining the advantages and drawbacks of IT decision support tools for chief decision-making

3. Explaining the usefulness and limitations of decision-making tools for cervical spine imaging

4. Conducting an appropriate assessment of the spine while maintaining spinal stability, including inspecting the spine, immobilizing the patient, removing the backboard, and reviewing cervical spine and head CT scans

5. Describing the symptoms, signs, and treatment options for neurogenic shock, which include low blood pressure, bradycardia, hypothermia, and peripheral vasodilation

6. Describing the symptoms and signs of a spinal cord injury, including loss of sensation or movement below the level of the injury, muscle weakness or paralysis, difficulty breathing, and loss of bowel or bladder control.

E: Exposure and environmental control

1. Undressing the patient completely, usually by cutting off their clothing, to facilitate a thorough examination and evaluation

2. After completing the assessment, cover the patient with warm blankets or an external warming device to prevent hypothermia in the trauma recipient area.

3. Heating intravenous fluids before infusion and maintaining a warm environment(1)

2.2. Secondry Evaluation

The secondary evaluation cannot start until the primary evaluation (ABCDE) is finished, the patient's vital functions have been stabilized, and resuscitation has begun. The secondary evaluation consists of a comprehensive review of the patient's medical history and a physical examination from head to toe, with reassessment of vital signs. Each area of the body is thoroughly examined to avoid missing an injury or underestimating its importance, which is especially important in an unresponsive or unstable patient.

2.2.1. History: The history of the mechanism of injury is an important component of every medical assessment. However, in cases of trauma, the patient may not be able to provide this information. In such cases, prehospital personnel and family members can provide details about the circumstances surrounding the injury. This information can be crucial in determining the appropriate course of treatment and ensuring the best possible outcome for the patient.

The AMPLE history is a mnemonic commonly used to obtain important information about a patient's medical history, especially in cases of trauma. The letters stand for:

A: Allergies to medications, foods, or other substances

M: Medications currently being taken, including prescription, over-the-counter, and herbal supplements

P: Past medical history, including any chronic illnesses, surgeries, or hospitalizations, and pregnancy status for female patients

L: Last meal or drink, which can be important in cases where surgery or sedation may be necessary

E: Events and environment related to the injury, including how the injury occurred, the time of day, and any environmental factors that may have contributed to the injury.

Obtaining this information can help guide treatment decisions and avoid complications during medical procedures.

2.2.2. Physical examination: In most cases, the patient must undress completely to allow for a thorough examination. Abdominal examination is done in a systematic order: inspection, auscultation, percussion, and palpation. This is followed by pelvis and hip examination; urethral, perineal, rectal and vaginal examinations should be done. All findings, positive or negative, should be documented in the patient's medical record. (exposure) The anterior, flank and posterior abdominal region, lower chest and perineum, abrasions, bruises, tears, penetrating wounds, stuck foreign bodies (pebbles, glass, etc.) are quickly examined in terms of pregnancy status. In the perineum, the scrotum, labia, urethral meatus, vagina, and rectum are examined for hematoma, swelling, and bruising. In obese patients, under the skin folds should not be overlooked. While examining the patient's back, the Logroll maneuver can be rotated in a coordinated and safe manner. At the end of the rapid physical examination, the patient is covered with warm blankets to help prevent hypothermia.

Even though listening to bowel sounds is an essential part of the examination, it's important to note that the presence or absence of bowel sounds doesn't always indicate injury, and in a busy emergency room, it may be difficult to hear them clearly.

Percussion of the abdomen can result in a slight movement of the peritoneum, which may manifest as signs of peritoneal irritation. However, if rebound tenderness is already present, further examination for additional signs of irritation may cause unnecessary pain to the patient. It should be noted that if the patient exhibits voluntary guarding, it can affect the reliability of the abdominal examination. On the other hand, involuntary guarding is a reliable indication of peritoneal irritation.

Palpation; It is the most important part of the examination in penetrating abdominal traumas. Peritonitis findings such as diffuse abdominal tenderness, rebound and defense indicate the presence of fluids in the abdomen that may irritate the peritoneum such as blood, bile, intestinal contents, and urine

Penetrating abdominal trauma requires completion of all necessary interventions in the primary evaluation, followed by completion of all steps of the secondary evaluation if possible.

Penetrating abdominal trauma patients should be evaluated based on the mechanism of injury, which can be classified as stab wounds or gunshot wounds. These two types of injuries have different mechanisms, where stab wounds and low-energy gunshot wounds cause tissue damage through shearing and tearing, whereas high-energy gunshot wounds transfer more kinetic energy, causing increased damage surrounding the bullet's trace due to transient cavitation. As a result of these differences, the approaches to these injuries are also different. However, it should be noted that any patient with an abdominal injury from direct impact, explosion, sharp object or impact should be considered to have a visceral, vascular, or pelvic injury until proven otherwise.

Considering the mechanism of penetrating abdominal injury can help guide healthcare providers to have an idea about which organs are likely to be injured, what examination and diagnostic tools may be required for their evaluation, and it can even determine if the patient needs to be transferred to another medical center.

Stab wounds in the abdomen tend to penetrate the nearby structures and cause injury to adjacent organs and tissues. On the other hand, firearm injuries can cause additional intra-abdominal injuries due to the angle of entry, the effect of cavitation, and possible fragmentation of the bullet. The degree of tissue injury is significantly determined by the type of weapon, muzzle velocity, and characteristics of the bullet used. In addition, the severity of injury from shotgun wounds is influenced by the distance of the shot.

Abdominal anatomy and suspected injury patterns in trauma patients

Injuries that penetrate the trunk between the nipple and perineum can potentially cause intraperitoneal damage and should therefore be carefully evaluated. The appropriate approach for assessing the abdomen and pelvis should be determined based on various factors, including the location and force of the injury, as well as the patient's hemodynamic status. Additionally, it's important to note that the lower rib cage partially encloses the abdominal area.

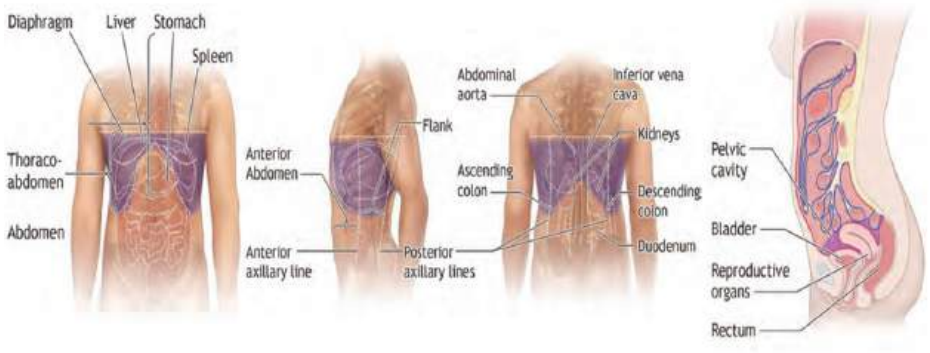
The anterior abdominal region is anatomically defined as the area bounded by the superior margin of the costal arches superiorly, the inguinal ligaments and pubic symphysis inferiorly, and the anterior axillary lines laterally. Trauma to the anterior abdominal wall poses a significant risk to the hollow visceral organs contained within.

The toracoabdominal region is situated between the infrascapular line at the back and the lower edge of the nipple line, and superior to the costal margins. This area is home to vital organs such as the diaphragm, liver, spleen, and stomach, which are partially shielded by the bony thorax. Since the diaphragm ascends to the level of the fourth intercostal space during full expiration, injuries such as rib fractures and penetrating wounds below the nipple line can potentially harm the abdominal organs.

The flank is the lateral part of the torso between the lower ribs and the iliac crest, while the back is the posterior aspect of the torso, extending from the base of the neck to the buttocks. The retroperitoneal space lies deep to the peritoneal cavity and contains vital structures, including major blood vessels, the kidneys and ureters, and parts of the gastrointestinal tract. The musculature in the flank and paraspinal regions provides some degree of protection to these structures but may not completely prevent injury in the event of trauma.

The area of the flank and back is situated posteriorly and includes the retroperitoneum, a space located behind the peritoneum lining the abdominal cavity. The retroperitoneum encompasses several vital structures, including the abdominal aorta, inferior vena cava, duodenum, pancreas, kidneys, ureters, colon, and pelvic cavity. Injuries to the retroperitoneal area can be challenging to diagnose as they occur deep inside the abdomen, and there may not be immediate signs or symptoms of peritonitis.

The pelvic cavity is an area surrounded by pelvic bones and includes the lower part of retroperitoneal and intraperitoneal cavities. It contains the rectum, bladder, iliac vessels, and female reproductive organs. Injuries to pelvic organs may cause significant blood loss directly from the pelvic bones.



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Describing the symptoms and signs of a spinal cord injury, including loss of sensation or movement below the level of the injury, muscle weakness or paralysis, difficulty breathing, and loss of bowel or bladder control.

3. Diagnostic Methods of Penetraitin Abdominal Injury

3.1 Gastric Tubes and Urinary Catheters

The therapeutic objectives of early placement of a gastric tube during the primary examination include relieving acute gastric distension and decompression of the stomach, if necessary, before diagnostic peritoneal lavage is performed. Gastric tubes can reduce the incidence of aspiration in these cases. However, they can trigger vomiting in a patient with an active gag reflex. If blood is present in the stomach contents and it is not of nasopharyngeal and/or oropharyngeal origin, it suggests an injury in the upper gastrointestinal system. If a patient has severe facial fractures or possible basal skull fracture, a gastric tube should be passed through the mouth to prevent passage.

In patients with midline facial injuries, the nasogastric tube may inadvertently enter the sinuses or the cranial cavity. Therefore, an orogastric tube is preferred over the nasogastric tube. The passage of a gastric tube can be difficult or impossible in patients with hiatal hernia, and radiological or other assistance may be needed to avoid causing an iatrogenic injury. The placement of a urinary catheter during resuscitation serves several purposes, including relieving retention, detecting bleeding, monitoring urine output as an index of

tissue perfusion, and decompressing the bladder before diagnostic peritoneal lavage.

A full bladder will help make FAST more effective. If FAST is considered, insertion of the urinary catheter is delayed.

Visible blood in the urine (gross hematuria) is a strong indicator of trauma to the genitourinary system, which includes the kidneys, ureters, and bladder. However, the absence of hematuria does not rule out the possibility of injury to these structures. When a trauma patient presents with an inability to urinate, needs a pelvic binder, or has signs such as blood in the meatus, scrotal hematoma, or perineal ecchymosis, a retrograde urethrogram is necessary. It is important to ensure that the urethra is intact before inserting a urinary catheter to avoid aggravating any possible urethral injury. In cases where a disrupted urethra is identified during primary or secondary examination, an experienced physician may need to place a suprapubic tube.

3.2. Local Wound Exploration

Sterilized around the injury site and local anesthesia is applied to wound edges.The wound can be evaluated directly.If it cannot be evaluated, assesment can be provided by expanding the wound.

3.2.1. Diagnostic Peritoneal Lavage

Diagnostic peritoneal lavage has been applied to detect luminal organ injury and hemoperitoneum in penetrating trauma. Although highly sensitive (96% to

99%) and specific (98%), DPL is an invasive procedure with a complication rate of 1%.(5,6,7)

The DPL should be performed by the surgical team responsible for the patient, as it can significantly alter the subsequent examinations. It should be noted that the DPL requires gastric and urinary decompression to prevent complications. It is useful in patients with multiple cavities or penetrating trauma with obvious tangential trajectories. Finally, hemodynamically stable patients requiring abdominal evaluation in environments where FAST and CT are not available may benefit from the use of DPL. DPL is rarely used in environments where CT and/or FAST are available, as it is invasive and requires surgical expertise. Relative contraindications to DPL include previous abdominal surgery, morbid obesity, advanced cirrhosis, and pre-existing coagulopathy. Open, semi-open, or closed (Seldinger) infraumbilical techniques can be acceptable in the hands of trained clinicians. An open supraumbilical approach is preferred in patients with pelvic fractures to avoid entering the anterior preperitoneal pelvic hematoma. In patients with advanced pregnancy, an open suprarenal approach is more suitable to avoid damaging the expanded uterus. Aspiration of gastrointestinal contents, plant fibers, or bile from the lavage catheter necessitates laparotomy.

In patients with thoracoabdominal penetrating injuries, the cut-off values for diagnostic peritoneal lavage are different.

Table 2. Positive Diagnostic Criteria For Diagnostic Peritoneal Lavage

	Abdominal Trauma	Thoracoabdominal penetrating injury
Red Blood Cell	>100,000 /mL	***>10,000 /mL***
White Blood Cell	>500/**mL**	>500 /mL
Amylase	>19 IU/L	>19 IU/L
Alkaline phosphatase	>2 IU/L	>2 IU/L
Bilirubin	>0,01 mg/dL	>0,01 mg/dL

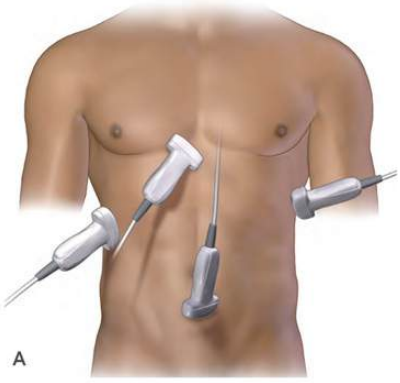
3.2.2. Focused Assessment With Sonography For Trauma(FAST)

Focused assessment with sonography in trauma is an ultrasound protocol developed to evaluate hemoperitoneum and hemopericardium. Numerous studies have shown sensitivities of 85% to 96% and specificities exceeding 98%.(8)

When performed by appropriately trained individuals, focused assessment with sonography in trauma is a widely accepted, rapid, and reliable study for

detecting intraperitoneal fluid. It offers the advantages of being at the bedside, repeatable, easy to use, noninvasive, and free of radiation and contrast agents. It can also detect pericardial tamponade, which is one of the non-hypovolemic causes of hypotension.

FAST involves examining four areas: the pericardial sac, hepatorenal fossa, splenorenal fossa, and Douglas pelvis or pouch. After the initial screening, clinicians may perform single or multiple repeated scans to detect progressive hemoperitoneum. FAST can be performed at the bedside in the resuscitation room simultaneously with other diagnostic or therapeutic procedures. It has no known contraindications. However, the use of ultrasonography is limited in puncture and cutting tool injuries. FAST is used to detect free intraperitoneal fluid in the Morison pouch, left upper quadrant, and pelvis. Although it is highly sensitive in detecting fluid volumes of 250 cc or greater, it does not show the source of bleeding or classify solid organ injuries.



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3.2.3. Computed Tomography

Computed Tomography (CT) is a diagnostic procedure that requires the patient to be transported (i.e. taken out of the resuscitation area), administered IV contrast, and exposes them to radiation. CT should only be used in hemodynamically stable patients without a clear indication for emergency laparotomy (even though it is less time-consuming with modern CT scanners). Do not perform a CT scan if it delays the patient's transfer to a higher level of care.

Table.3 Comparison of DPL, FAST and CT Abdomen Trauma			
	DPL	FAST	CT
Advantage	1. Early operation detection 2. Fast performed 3. Can detect intestinal damage 4. No need to move from the resuscitation area	1. Early operative determination 2. Noninvasive 3. Fast performed 4. Repeatable 5. No need to move from the resuscitation area	6. Anatomic diagnosis 7. Noninvasive 8. Repeatable 9. Visualizes retroperitoneal structures 10. Visualizes bony and soft-tissue structures 11. Visualizes extraluminal air
Disadvantages	• İnvasive • Risk of procedural injury • Requires gastric and urinary decompression to prevent complications • Not repeatable • Interferes with interpretation of subsequent CT or FAST • Low specificity • May miss diaphragm injuries	• Depends on the operatör • Intestinal gas and subcutaneous air distort the images • May miss diaphragm, bowel and pancreatic injuries • Does not fully evaluate retroperitoneal structures Does not visualize extraluminal air • Body build can limit image clarity	• Higher cost and longer duration • Radiation and IV contrast exposure • May miss diaphragm injuries • May bypass some bowel and pancreatic injuries • Requires transfer from the resuscitation area
Indications	• Penetrating abdominal trauma without other indications for emergency laparotomy	• Penetrating abdominal trauma without other indications for emergency laparotomy	• Normal hemodynamics in blunt or penetrating abdominal trauma • Penetrating back/ flank trauma without other indications for emergency laparotomy

Computed tomography (CT) scans are an important diagnostic tool for identifying specific organ injuries and their severity. They are especially useful for detecting retroperitoneal and pelvic organ injuries that may not be apparent

during physical examination, Focused Assessment with Sonography in Trauma (FAST), or Diagnostic Peritoneal Lavage (DPL). However, there are some relative contraindications to using CT, such as delays in obtaining the scan, an uncooperative patient who cannot be safely sedated, or a known allergy to contrast material. It's also worth noting that CT scans may miss certain injuries to the gastrointestinal system, diaphragm, or pancreas. If there are no injuries to the liver or spleen, the presence of free fluid in the abdominal cavity is a concerning sign and may suggest an injury to the gastrointestinal system or mesentery. Many trauma surgeons view this finding as an indication for early surgical intervention. (10)

3.2.4. Rectosigmoidoscopy

Rectosigmoidoscopy is a diagnostic method in the evaluation of extraperitoneal rectal injuries. Injuries to this area usually do not show signs of peritoneal irritation and may lead to complications such as pelvic sepsis or necrotizing fasciitis that may cause mortality. Intraoperative or peroperative rectosigmoidoscopy will provide information about the level of injury and will guide the decision of transanal or transabdominal approach.

3.2.5. Diagnostic Laparoscopy

Diagnostic laparoscopy is a method accepted for evaluating hemodynamically stable penetrating trauma patients with potential tangential injury and no indication for laparotomy. Laparoscopy is useful for diagnosing diaphragm injury and peritoneal penetration. Its utility is limited by the need for general anesthesia. It has a definite indication in left thoracoabdominal penetrating injuries.

When evaluating a patient with penetrating abdominal trauma; if there are signs of altered consciousness, mental confusion, confusion, agitation or drowsiness, if there are symptoms such as loss of sensory sensations, false or altered perception of sensations such as pain, temperature, pressure, or touch, if there is injury to the lower ribs, pelvis, and lumbar spine, on physical examination If there are suspicious findings, general anesthesia for extra-abdominal injuries, or if patient contact is required for long-term radiographic studies, more detailed abdominal evaluation is required to identify or exclude intra-abdominal injury.

3.2.6. Contrast Studies

Contrast studies can be particularly helpful in diagnosing suspected injuries, but should not delay the care of hemodynamically abnormal patients. These studies include:

- Urethrography
- Cystography
- Intravenous pyelogram
- Gastrointestinal contrast studies

When there is a suspicion of a urethral injury, it is crucial to conduct a urethrogram before inserting a urinary catheter. During the urethrogram, an 8 F urinary catheter is placed in the meatus and fixed with balloon inflation to 1.5 to 2 mL. Approximately 30 to 35 mL of undiluted contrast medium is then instilled under gentle pressure. In men, X-rays are taken with an anterior-posterior projection while gently stretching the penis towards one of the patient's shoulders. A proper study should show the reflux of the contrast into the bladder, indicating an intact urethra. This process is important to avoid further damage to the urethra and to ensure proper placement of a urinary catheter in case it is necessary.

To diagnose intraperitoneal or extraperitoneal bladder rupture, a cystogram or CT cystography is recommended. During this procedure, a syringe barrel is attached to the patient's indwelling bladder catheter, and 350 mL of water-soluble contrast is allowed to flow into the bladder. Additional contrast may be given to maintain bladder distension. The patient may void spontaneously or report discomfort, and radiographs are taken before, after, and after drainage to definitively exclude bladder injury. CT evaluation of the bladder and pelvis (CT cystogram) is an alternative study that can provide more information about the kidneys and pelvic bones..

Suspected urinary tract injuries are best evaluated with a contrast-enhanced CT scan. If CT is not available, an intravenous pyelogram (IVP) offers an alternative. A high-dose, rapid injection of renal contrast ("screening IVP") is performed using 200 mg of iodine/kg body weight. The appearance of the renal calyces should be visible on the abdominal radiograph 2 minutes after the injection is complete. It presents with unilateral kidney vision, kidney absence, thrombosis or renal artery avulsion and massive parenchymal deterioration. Imaging may warrant further radiological evaluation. Isolated

injuries of retroperitoneal gastrointestinal structures (eg, duodenum, ascending or descending colon, rectum, biliary tract, and pancreas) may not immediately cause peritonitis and may not be detected on DPL or FAST. Contrast-enhanced CT, specific upper and lower gastrointestinal intravenous contrast studies, and pancreaticobiliary imaging studies may be helpful when injury to one of these structures is suspected. Ultimately, however, the surgeon dealing with the patient will guide these studies.

Late recognition of intra-abdominal or pelvic injury leads to early death from bleeding or late death from visceral injury. For this reason, knowing the injury mechanisms that may result in intra-abdominal injury (eg, high-energy gunshot wounds), recognizing the factors that may limit the usefulness of physical examination (eg substance use), and using additional auxiliary techniques (eg FAST, Diagnostic peritoneal lavage, CT) should be considered.

Physical examination and evaluations with aids such as ultrasound and X-ray can be difficult in obese patients. Therefore, obese patients with the potential for abdominal injury should be approached with high suspicion for abdominal injury.

It is necessary to be aware of the potential limitations of imaging attachments. Seemingly minor abdominal and pelvic injuries can cause severe bleeding in people who are taking anticoagulant therapy, as well as in older, frail people. The degree of coagulation disorder should be determined early and necessary interventions should be made to reverse it when appropriate. Early and aggressive treatment is essential for optimal results.

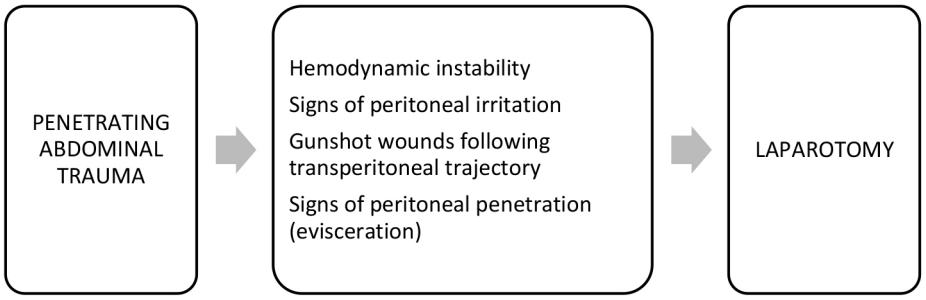
4. Diagnostic Approaches to Penetraiting Abdominal Trauma

The etiology of injury, anatomical location, and available resources influence the evaluation of penetrating abdominal trauma in the patient.

In the last quarter century, nonoperative approaches have gained attention and various studies have shown that anterior penetrating injuries can be followed without requiring surgical intervention in 50-70% of cases, and in patients initially decided to be followed, the need for late surgical intervention remained between 10-15%.(19,20)

First of all, it should be determined whether emergency laparotomy is necessary in the patient. In patients with penetrating abdominal trauma, there is an indication for emergency laparotomy in the presence of hemodynamic instability, signs of peritoneal irritation, signs of peritoneal penetration

(evisceration), and in gunshot injuries following a transperitoneal trajectory. Among these, in patients with signs of peritoneal penetration and no other indication for laparotomy, further evaluation can be considered. (12,13,14,15)



4.1. Stab Wounds

Stab wounds that do not require emergency laparotomy should undergo follow-up and further examination to ensure proper evaluation.

In the evaluation, the time of injury, type of weapon, number of injuries, and amount of blood lost at the scene should be determined. The severity and location of abdominal pain should be inquired from the patient. Then, physical examination should be performed.

Systematic abdominal examination and detailed patient history should be taken and recorded.

In stable patients with anterior abdominal penetrating injuries, local anesthesia should be administered in the emergency department to explore the wound site and observe whether the peritoneum has been penetrated. If the peritoneum has not been penetrated and there are no additional injuries, the patient can be discharged without further testing. If the peritoneum has been penetrated, further evaluation should be performed for potential intra-abdominal organ injuries. Diagnostic approaches may include periodic abdominal exams, diagnostic peritoneal lavage, FAST or CT scan. Recent studies have shown that serial examination and laboratory monitoring are the best diagnostic approaches. (16,17)

If a hemodynamically stable patient has an anterior abdominal stab wound and positive local wound exploration, serial physical examination, FAST, and laboratory follow-up should be performed, and if there is clinical suspicion, an indication for laparotomy should be considered.

Nonoperative management can be considered in cases of anterior abdominal injuries with hemodynamic stability and without peritoneal signs or evisceration. However, approximately 55% to 60% of patients with knife wounds penetrating the anterior peritoneum may present with serious complications such as hypotension, peritonitis, or internal organ evisceration, necessitating urgent laparotomy. Diagnostic methods such as serial physical examinations, DPL, CT scans, or diagnostic laparoscopy can be used for patients considered for nonoperative management. A positive FAST indicates the presence of intra-abdominal fluid accumulation and possible internal organ injury, while a negative FAST does not rule out the possibility of injury. Diagnostic laparoscopy can confirm or exclude peritoneal penetration, but is less useful in identifying specific injuries. The decision to perform laparotomy or other invasive treatment options is at the discretion of the surgeon.

The reliability of local wound exploration is low in stable patients with penetrating injuries from the back and flank regions. No instrument should be inserted into the wound during exploration. Three-phase contrast-enhanced CT scan should be used to evaluate for urinary system, colon, duodenum, and other retroperitoneal organ injuries. Patients with negative CT scans should be followed up with periodic abdominal examinations. Exploratory laparotomy/laparoscopy is indicated for patients with evidence of solid or lumenal organ injury. However, for isolated penetrating injuries in the right upper quadrant that are hemodynamically stable and confirmed by CT scan to have passed through the liver, nonoperative management can be attempted.(18)

The thickness of the lateral and back muscles provides protection against damage caused by penetrating sharp objects and some gunshot injuries to the underlying internal organs. For those who do not have indications for emergency laparotomy, less invasive diagnostic options include serial physical examinations (with or without serial FAST examinations), double or triple contrast CT scans, and DPL. In patients with wounds behind the anterior axillary line, serial examinations are useful in detecting retroperitoneal and intraperitoneal injuries to prevent the development of peritonitis. Double or triple contrast CT is a time-consuming study that can more fully evaluate the retroperitoneal colon on the side of the wound and has an accuracy comparable to serial physical examinations. However, when performed appropriately, CT should allow for earlier diagnosis of injuries. Rarely, retroperitoneal injuries may be missed by serial examinations and contrast-enhanced CT. Due to the mild presentation

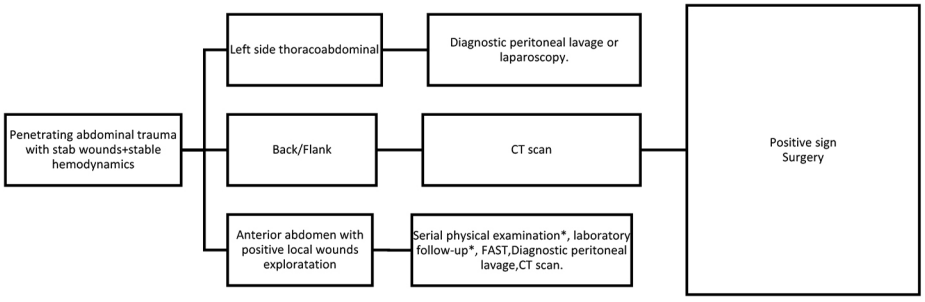
of some colonic injuries, early outpatient follow-up after 24 hours of hospital observation may be necessary.

DPL (Diagnostic Peritoneal Lavage) can also be used as an early screening test for the detection of injuries in the abdominal region of creatures. A positive DPL result may indicate the need for urgent laparotomy. However, DPL may not detect retroperitoneal colon injuries.

In stable patients with penetrating thoracoabdominal injuries, diaphragmatic injury may be overlooked. Especially in left lower thorax penetrative injuries, diagnostic laparoscopy or diagnostic peritoneal lavage should be considered for evaluation. Right diaphragmatic injury due to penetrative injury can be neglected if there is no significant liver damage. Diagnostic laparoscopy may be preferred in patients with hemothorax and pneumothorax, or those who cannot tolerate diagnostic peritoneal lavage.

4.2. Gunshot Wounds

While selective conservative treatment has become more common in penetrating stab wounds, laparotomy continues to be routinely performed in many centers for abdominal gunshot injuries. This is due to series reporting up to 90% of intra-abdominal organ injury related to moderate to high energy. However, in recent years, large trauma centers with many patients have shown that selective conservative treatment is possible for civilian gunshot injuries. (21)



When assessing a patient with a gunshot wound, it is crucial to gather detailed information about the circumstances surrounding the injury, such as the time and location of the incident, the type of firearm used, the distance between the shooter and the victim, and the number and location of the wounds. In cases of shotgun wounds, the distance between the shooter and the victim is particularly relevant, as major organ injuries tend to decrease beyond a certain

distance. It is also important to obtain data on the patient's vital signs and any pre-hospital treatment received. Furthermore, the patient's reported symptoms, especially the magnitude and location of any abdominal pain, can provide valuable insights into the extent of the injury. Explosions can cause significant internal organ damage due to increased pressure, especially when they occur in enclosed spaces or in close proximity to the patient..

In hemodynamically stable gunshot injuries in the anterior abdomen, laparotomy is indicated.

In hemodynamically stable gunshot injuries in the right upper quadrant, tangential, posterior, and flank regions, a non-operative approach can be attempted with the increasing use of computed tomography.(22)

If the bullet trajectory is thought to remain in subcutaneous tissue in obese patients with gunshot injuries, peritoneal entry can be detected with CT. Laparoscopy may be considered in tangential injuries. High-energy tangential firearm injuries can cause intraperitoneal organ injury due to their high kinetic energy.

Three-phase CT evaluation can determine bullet trajectory, peritoneal passage, retroperitoneal effect, and associated injuries.

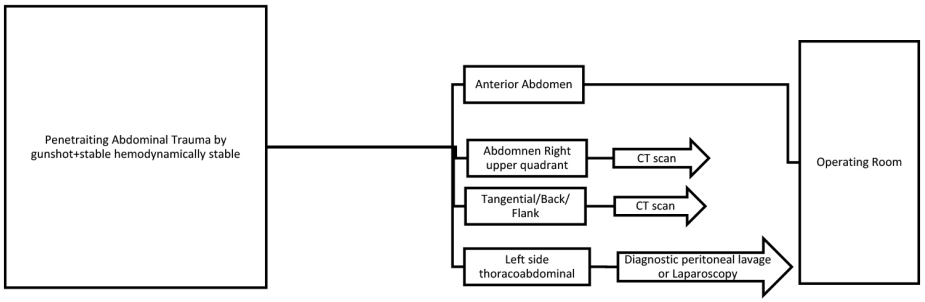
Diaphragmatic injury may be overlooked in stable patients with penetrating thoracoabdominal injuries. Diagnostic laparoscopy or diagnostic peritoneal lavage should be considered, especially in left lower thoracic penetrating injuries. Damage to the right diaphragm due to penetrating injury can be ignored if there is no major liver damage. Diagnostic laparoscopy may be preferred in patients with hemotorax and pneumothorax or those who cannot tolerate diagnostic peritoneal lavage.

In hypotensive patients, the goal is to quickly evaluate abdominal or pelvic injury and determine if it is the cause of hypotension. Patient history, physical examination, and additional diagnostic tools can identify abdominal and pelvic injuries requiring urgent bleeding control. Hemodynamically normal patients without signs of peritonitis may undergo a more detailed evaluation to identify injuries that may cause delayed morbidity and mortality. This evaluation should include repeated physical examinations to detect any signs of bleeding or peritonitis that may develop over time.

4.3. Considerations:

Surgery may be required for diagnosis and treatment in all cases of penetrating trauma.

Transfer to another center for abdominal CT scanning should not be delayed. If CT can change the care at the referring facility or facilitate stabilization for transfer, it should be performed.



In a patient with tangential gunshot wounds to the abdomen, the diagnosis of intra-abdominal injury may be delayed. This is because tangential gunshot wounds may not be truly tangential (e.g., they may penetrate the peritoneal cavity), or high-velocity penetrating wounds may cause injury without penetrating the peritoneum due to the blast effect.

5. Laparotomy Indications

Surgical judgment is necessary to determine the timing and need for laparotomy**.**

The following indicators are commonly used to facilitate the decision-making process in this regard:

In cases of abdominal trauma, certain signs and symptoms may indicate a need for immediate surgical intervention, including low blood pressure with an anterior fascia-penetrating wound, gunshot wounds that pass through the peritoneal cavity, evisceration, bleeding from the stomach, rectum, or genitourinary system, peritonitis, and the presence of free air, retroperitoneal air, or hemidiaphragm rupture. CT scans that show ruptured gastrointestinal systems, intraperitoneal bladder injuries, renal pedicle injuries, or severe visceral parenchymal injuries may also indicate the need for urgent surgical intervention. In hemodynamically unstable patients, aspiration of 10 cc or more blood during DPL or after blunt or penetrating abdominal trauma may indicate aspiration of gastrointestinal contents, plant fibers, or bile.

5.1. Operation Preparation

All trauma patients planned for surgery should receive antibiotics. The choice of antibiotic should be based on the degree of wound contamination. Tetanus prophylaxis should not be overlooked and should be documented.

During inspection of the trauma patient, clothing should be removed and all areas should be examined. It is important to avoid hypothermia at this stage. Wrapping the patient in warm blankets will help reduce heat loss. In patients with bleeding and a need for transfusion of blood products and fluids, these should be warmed to prevent hypothermia.

Patients admitted to the operating room should be warmed with a "bair hugger" heating device, or covered with warm sterile drapes if this is not possible. Heating can also be achieved by providing warm air from the ventilator machine.

5.2. Damage Control Surgery

Acute traumatic coagulopathy (ATC) may develop in trauma patients with life-threatening bleeding.(23,24) Another important component of acute trauma coagulopathy is fibrinolysis. Hyperfibrinolysis and the cessation of fibrinolysis are associated with increased mortality.(25) Post-traumatic coagulopathy, hypothermia, and metabolic acidosis are called the "bloody vicious cycle" and are known as the lethal triad.

Once the vicious cycle starts, each component triggers the others, leading to fatal arrhythmias. The aim of damage control surgery is to limit the operation time and to break this cycle by providing necessary interventions in intensive care conditions as soon as possible for the patient. Indications for damage control surgery are to keep the operation time short and refractory hypothermia (<35 degrees), deep acidosis (arterial pH <7.2, base deficit <15 mmol/L), and the combination of refractory coagulopathy.(26,27)

The aim of damage control surgery is to stop bleeding and limit gastrointestinal spread. The techniques used are temporary solutions, and definitive treatment is postponed until the patient is physiologically stable.

Damage control surgery aims to stop bleeding and limit gastrointestinal spread in order to save the patient's life. The techniques used are temporary solutions, and definitive treatment is delayed until the patient is physiologically stable.

1-Damage control surgery follows a three-stage approach

2-Limited surgery: Initial interventions control bleeding, clean wounds, preserve open organ wounds, and create temporary intestinal connections.

3-ntensive care treatment: Vital functions such as respiration, blood pressure, and fluid balance are supported. Blood and fluid transfusions are performed.

Definitive surgery: After the patient's condition is stabilized, a more comprehensive surgical treatment is planned and implemented.

6. Surgery in Penetrating Abdominal Trauma

Abdominal exploration is more convenient with an incision above and below the umbilicus opened from the midline. Free abdominal fluid and clotted blood are removed with abdominal compresses or pads. In penetrating trauma, the route of the penetrating sharp object or bullet should be followed, and a plan should be made based on possible injuries.

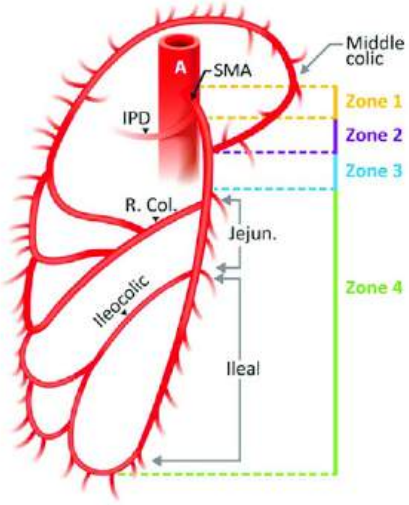
If the systolic blood pressure is <70 mmHg when the abdomen is opened, digital pressure or clamping of the aorta at the diaphragmatic hiatus level should be applied.

If the source of bleeding is seen, vascular injury is controlled with finger pressure, or solid organ injury is controlled by packing with abdominal compresses. If bleeding is from the liver, vascular clamping of the hepatic pedicle or the Pringle maneuver can be performed. If bleeding is from the spleen, the splenic hilum can be clamped.

Exploration may be difficult in the presence of massive hemorrhage. Control of the aorta from the proximal end is achieved at the diaphragmatic hiatus level, and if aortic injury is suspected to be from the proximal area (suprarenal, supra-celiac), the left crus can be cut or the laparotomy can be extended to the left side (left subcostal, left transverse from the umbilical level).

It is necessary to determine whether the vascular injury is supra-colonic or infra-colonic because supra-colonic injuries are best evaluated with a left medial visceral rotation.

In the left medial visceral rotation, the lateral peritoneal reflection is started from the distal end of the descending colon, passes behind the spleen posterior and gastric fundus, and is extended to the splenic flexure of the colon, terminating in the esophagus. The stomach, spleen, pancreas, and left colon are pulled to the midline. If the left kidney is also included, it is called the Mattox maneuver.(28,29)



The level of injury is important in the approach to superior mesenteric artery injuries. Fullen's anatomical classification of superior mesenteric artery injuries according to the region and degree of injury is a guide.

Zone 1 includes the segment from the proximal main body to the inferior pancreaticoduodenal artery. It is located posterior to the pancreas and can be accessed best through left medial visceral rotation.

Zone 2 is defined as the area between the pancreaticoduodenal artery and the middle colic artery, and can be reached by following the inferior border of the pancreas from the root of the transverse colon through the Lesser sac. To access its proximal portion, the body of the pancreas can be divided.

Zone 3 and 4 comprise the distal body segment and its segmental branches from the middle colic. They are accessed directly through the mesentery.(30)

Venous injuries behind the pancreas can be accessed by cutting the neck of the pancreas to reach the junction of the superior mesenteric, splenic, and portal veins.

Inferior vena cava injuries can be accessed through a right medial visceral rotation. This involves a widened Kocher mobilization and continuing a lateral peritoneal incision to the colon that comes out of the hepatocolic ligament. The retroperitoneal membrane is separated from the ileocecal region towards the Treitz ligament and the abdominal organs are medialized towards the abdominal midline. This maneuver is known as Cattell-Braasch maneuver.(31)

Proximal control is achieved by applying a clamp over the iliac bifurcation. In anterior injuries, a Satinsky clamp can be used.

In iliac artery injuries, placing a clamp at the proximal infrarenal level of the aorta controls bleeding. However, iliac vein injuries cannot be controlled with an aortic clamp. Therefore, pressure is applied on the bleeding site to control the bleeding. If adequate visibility cannot be obtained, complete pelvic vascular isolation is achieved by placing clamps on the aorta, vena cava, and bilateral external iliac arteries, and dissection is performed to locate the bleeding focus. While the dissection continues, the clamps are moved closer to the bleeding focus. A right iliac artery division may be required to explore the right iliac vein. After venous injury treatment to avoid ischemia, the artery should be repaired immediately.

After controlling the abdominal bleeding, the colon and small intestine should be examined quickly, the anterior and posterior sections of the stomach should be examined, and a wide Kocher maneuver should be performed to explore the duodenum. The anterior and posterior sections of the pancreas should be examined. After identifying the injury, treatment options should be evaluated based on the patient's physiological status.

6.1. Organ Specific Approaches

6.1.1. Approaches in Abdominal Artery and Venous İnjuries

In abdominal artery injuries; first, bleeding is controlled by applying sufficient pressure with a finger, and the injury site is identified by sharp dissection towards the proximal and distal parts. Thromboembolectomy is performed with a Fogarty catheter. Saline infused with heparin is injected into the ends to prevent clot formation in the intima and media, and fragmented ends are debrided with sharp dissection. If there are multiple injuries or arterial injuries requiring reconstruction with saphenous vein interposition, an intravascular shunt is used.

Continuity of flow should be ensured in the aorta, superior mesenteric artery, hepatic artery proper, renal artery, and iliac artery. Ligature of the celiac artery and right and left hepatic arteries may be tolerated. Approaches for the superior mesenteric artery differ depending on the level of injury. If the opposite kidney is normal on palpation, nephrectomy is an option for renal artery injuries. End-to-end anastomosis is generally the first option for vascular repair. If a tension-free end-to-end anastomosis cannot be achieved, interposition grafts are used. Polytetrafluoroethylene (PTFE) grafts may be used for the repair of large arteries. PTFE grafts are preferred over Dacron because the risk of infection is lower.

Table.4 Intervention in Abdominal Artery Injuries	
Celiac artery:	Ligation
Renal artery:	Nephrectomy if ligation and contralateral kidney are palpable
Aorta:	Intraluminal shunt with thorax tube, other repair options
Superior Mesenteric Artery:	Intraluminal shunt
Common iliac artery and iliaca externa:	Intraluminal shunt, ligation+fasciotomy
Bleeding in the pelvis that cannot be stopped with packing:	Fogarty catheter inserted through iliaca interna, microfib collagen, topical thrombin injection

In abdominal venous injuries, if technically feasible, the injured vein should be repaired. Small injuries without tissue loss can be repaired with primary suturing. There is a risk of thrombosis in treatments with interposition grafts, but this appears to be a sufficient time for collateral development after one to two weeks. The use of PTFE in suprarenal vena cava and superior mesenteric vein injuries can reduce potential morbidity.For common iliac vein, external iliac vein, internal iliac vein, superior mesenteric vein, or portal vein injuries, ligature (after inferior vena cava ligation and fasciotomy), primary repair, and interposition grafts are possible treatments. For bleeding from defects in the pelvis and sacral wall, tamponade of the defect, fibrin adhesive, and Foley catheter can be used.

For bleeding from presacral veins, omental patch and sterile sutures can be used.

Treatment options in vascular injuries
Observation
Ligation of the vessel
Stitch repair
End-to-end primary anastomosis
Interposition grafts
• • Autogenous veins
• • Polytetrafluoroethylene (PTFE) graft
• • Dacron graft
Transposition
Extra anatomical bypass
Interventional radiology
• • Stenting
• • Embolization

In **pancreatic** injuries, should packing and drainage be used regardless of the involvement of the pancreatic duct.

For **gastrointestinal system** injuries, closure or resection can be performed using staplers or sutures.

In **gastric** injuries, the full thickness of the gastric wall is taken and sutured with a continuous single-layer suture or closed with staplers. To avoid missing gastric injuries, the pyloric area is closed and sterile diluted methylene blue or saline is administered through a nasogastric or orogastric tube, and the stomach is filled with air through the same tube in the abdomen. Partial gastrectomy may be necessary in destructive injuries.

In injuries to the **ileum** and **jejunum**, if the affected area is less than one-third of the surrounding area, it can be repaired with a transverse continuous 3/0 PDS suture. In cases of destructive or multiple injuries that are close to each other, resection is performed and a single-layer 3/0 polypropylene suture is used to perform an end-to-end anastomosis.(33)

In cases of mesenteric injuries, the approach is determined based on the blood supply of the intestine.

Duodenal injuries may include isolated injuries or combined pancreaticoduodenal injuries. Small duodenal perforations can be repaired with continuous single-layer primary closure using 3/0 monofilament sutures.(34)

The wound should be closed to obtain the widest lumen. In cases of large injuries in the first part of the duodenum, resection and end-to-end anastomosis can be used for repair. Injuries in the second part can be repaired by Roux-en-Y duodenojejunostomy.

Approaches to colon and rectal injuries include primary repair, end colostomy, and diversion ileostomy with primary repair. Primary repair involves lateral suturing or segmental resection of the damaged colon, followed by ileocolic or colocolic anastomosis, depending on the location. Anastomoses are made using a continuous single-layer anastomosis technique.(33)

Numerous large prospective and retrospective studies have indicated that primary repair can be safely used in patients with penetrating injuries.

In patients with destructive injuries in the left colon and requiring damage control surgery, there is a 40% risk of anastomotic leakage, and they are definitely candidates for temporary colostomy.(36*)*

In cases of rectal injuries, loop colostomy or loop ileostomy are preferred for the purpose of intestinal diversion. If the injury site can be reached, primary lateral suturing is performed. If the injury site is too large, the rectum is separated

from the distal pouch and closed with staplers or sutures, and opening of a terminal colostomy is another option.

Splenic injury can be managed by splenectomy, application of microfibrillar collagen or fibrin glue, placement of absorbable mesh, and packing around the mesh

Approach to penetrating injuries of the spleen involves splenectomy as an indication for grade 2 or higher injuries with splenic hilum injury, shattered splenic parenchyma, multiple spleen injuries, and coagulopathy.

Partial splenectomy can be used in isolated superior and inferior pole injuries. Gentle compression and horizontal matrix sutures can control bleeding.

Topical electrocautery, argon beam coagulation, thrombin-soaked gelatin foam sponges, fibrin sealants, or BiogGlue can be applied. The spleen can be wrapped in an absorbable mesh and repaired with pledgeted sutures.

Approach to Penetrating Liver Injuries:

Due to their large size, liver injuries are at high risk of being affected by trauma. In patients who are being managed non-operatively and do not require laparotomy, close monitoring of hemodynamics, hemoglobin levels, and periodic abdominal examination should be performed in the intensive care unit.

Surgical intervention is indicated in cases of hemodynamic instability. If the patient is hemodynamically stable but requires transfusion of 4 units within 6 hours or 6 units within 24 hours, angiography is required for hepatic bleeding control.

In liver injuries, the first step for controlling bleeding is perihepatic packing and manual compression. If there is excessive or large-scale bleeding, the Pringle maneuver should be performed immediately, and the bleeding focus should be located.

If a penetrating injury caused a clean transsection, end-to-end anastomosis can be performed. If the trauma is destructive, interposition should be performed using a saphenous vein graft after temporary shunting. The right and left hepatic arteries and portal vein can be selectively ligated. If the right hepatic artery is ligated, cholecystectomy should be performed.

If massive venous bleeding is observed behind the liver despite the Pringle maneuver, there is a high possibility of hepatic vein or retrohepatic vena cava injury. If bleeding is controlled with packing, a stent can be placed in the hepatic vein by interventional radiology after surgery. If bleeding cannot be controlled with packing, the vena cava can be clamped above and below the liver to expose the injury site for direct repair or stapler-assisted parenchymal resection.

Temporary shunting and veno-venous bypass of the hepatic vein and vena cava are also among the other options.

For bleeding within an organ's tissue, simple measures like applying manual compression at the site of injury can be effective for minor lacerations. In more severe cases, medical professionals can use various techniques to stop the bleeding, including electrocautery, argon beam coagulation, microfibrillar collagen, thrombin-soaked gelatin foam sponges, fibrin glue, and Bioglue. Suturing the parenchyma with blunt no. 0 chromic sutures can also be used to achieve hemostasis

Omentum can be used for large defects in the liver parenchyma. In penetrating liver injuries that extend into the parenchyma, the extent of damage may not be fully determined. In such cases, intraparenchymal tamponade or balloon occlusion with a Foley catheter can be used for damage control surgical principles.(37)

Cholecystectomy should be performed in cases of gallbladder injury or when ligation of the right hepatic artery is required. Injuries to the extrahepatic bile ducts often accompany vascular injuries due to their anatomical proximity.

If there is no tissue loss, T-tube drainage or lateral primary repair with 6/0 absorbable monofilament sutures can be considered. In cases where there is significant tissue loss or complete transection of the extrahepatic bile duct, Roux-en-Y choledochoduodenostomy should be performed. The anastomosis should be made with 5-0 absorbable monofilament sutures.

For injuries to the hepatic duct, intubation or stenting with ERCP can be done for external drainage.

7. Conclusion

Trauma is an important cause of death in all age groups. There are algorithms and classifications for rapid and systematic evaluations in trauma. The first step of this algorithm begins with determining which factor caused the trauma. Penetrating trauma refers to injury caused by foreign objects ((knife,arrow (low-energy handgun (medium-energy), and shotguns, military rifles (high-energy))entering tissue.After the causative agent is identified, initial evaluation of penetrating abdominal trauma should be made and vital signs should be checked immediately. In cases of penetrating abdominal trauma, initial evaluation is crucial, the first hour after a traumatic injury is the golden hour. Following the first evaluation, the second evaluation should be carried out and the patient's medical history should be determined and from

head to toe physical examination should be performed. The factor that causes penetrating trauma must be evaluated in which part of the abdomen the trauma is caused. Considering the mechanism of penetrating abdominal injury can help guide healthcare providers to have an idea about which organs are likely to be injured, what examination and diagnostic tools may be required for their evaluation, and it can even determine if the patient needs to be transferred to another medical center.As well as imaging techniques such as radiological tests, ultrasonography, computed tomography ,rectosigmoidoscopy,laparoscopy in the diagnosis of penetrating abdominal trauma; gastric tube, urinary catheter, local wound exploration, diagnostic peritoneal lavage methods can be also used.After the injuries caused by the trauma have been identied,an appropriate treatment approach should be determined. Minor injuries can usually be controlled with conservative treatment, while major injuries may require surgical intervention. Conservative treatment and surgical treatment practices may vary depending on the organ. That's why, in penetrating abdominal trauma patients, where every minute is precious, mastering all these treatment methods and applying diagnosis and treatment methods quickly and accurately will be life-saving.

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CHAPTER VII

GASTROINTESTINAL SYSTEM PERFORATIONS

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1. Introduction

The clinic of the patient can give us a clue to diagnose the perforation of the gastrointestinal tract, or the presence of free gas or fluid accumulation in the abdomen outside the lumen in diagnostic imaging may guide us. Clinical symptoms are seen due to contents spreading into the abdomen due to perforation.

Although the diagnosis of intestinal perforations is usually made by radiological examinations, surgical explorations for diagnostic purposes may also be required.

2. Pathophysiology

While perforation is mentioned in full-thickness injuries of the intestinal wall, it should be kept in mind that non-full-thickness injuries due to trauma during cautery or dissection may also turn into perforation in the future.

In general, full-thickness injuries and the associated spread of intestinal contents to the abdomen; it is due to etiologies such as trauma, perioperative thermal damage, during the endoscopy procedure, ileus or tumors that can cause perforation due to increased intestinal pressure causing intestinal obstruction.

Intestinal perforation precedes the obstruction with the increase of perfusion pressure in the intestine and subsequent ischemia and necrosis. The perforation proximal to the occlusion in the colon usually occurs in the cecum if there is a well-functioning ileocecal valve that does not allow backward decompression of the cecum (1).

Free gas accumulating in the peritoneum may disrupt the functioning of the diaphragm and cause respiratory failure. It should also be kept in mind that inflammation secondary to perforation may also lead to abdominal compartment syndrome (2,3).

3. Risk Factors

The main cause of iatrogenic perforation is surgical interventions. Some of these are endoscopy, colonoscopy, stent procedures, nasogastric tube placement, and operative applications that result from adhesion separation or thermal damage during surgery (4,5). In addition, procedures such as chest tube insertion, insertion of a catheter for dialysis, percutaneous gastrostomy, percutaneous drainage, and paracentesis may result in perforation (6,7).

Although perforation in gastrointestinal tract traumas is usually the result of penetrating injury, it can also occur as a result of blunt injury due to acute pressure effects.

Aspirin and nonsteroidal anti-inflammatory drugs, caustic substances and foreign bodies can also cause perforation (8). Severe nausea and vomiting can lead to spontaneous esophageal perforation known as Boerhave syndrome (9).

While perforation may occur secondary to intra-abdominal hernias and intestinal obstructions, it should be kept in mind that perforation may also occur in untreated appendicitis cases, in patients with peptic ulcer, which is the most known cause of gastric and duodenal perforation, and in intestinal diverticulitis (10,11).

Conditions that reduce blood flow to the intestine, such as mesenteric ischemia, embolism and heart failure also increase the likelihood of perforation. Finally, tumors can directly penetrate or obstruct and perforate.

4. Clinical Findings

It is very important to take a good anamnesis in patient evaluation. Conditions such as previous abdominal or chest pain complaints, presence of any intervention procedure, operation history, foreign body ingestion and drug intake that may cause perforation should be effectively questioned. Although most patients go to the emergency services for medical help with the onset of abdominal or chest pain, fewer patients unfortunately present as delayed cases after their clinical worsening.

The patient with perforation usually knows the time of perforation. They describe that their pain starts suddenly, worsens gradually, and there are also

temporary relief attacks. Persistent pain is caused by gastrointestinal contents irritating the peritoneum.

The first indication of perforation is usually sepsis. It should be known that the ability of the perforated area to be closed by the body may be impaired in weak, immunosuppressive and non-young patients who are often comorbid. Thus, gastrointestinal contents secondary to perforation easily spread into the abdomen and cause infection and sepsis (12). Such patients may be hemodynamically unstable and have normal or high fever.

During the physical examination, the vital signs of the patient should be checked. On examination, the neck, chest, abdomen and rectal region should be evaluated. Although patients with perforation may have normal vital signs at first, they may also be mildly tachycardic. As the inflammatory process increases, septic symptoms may become more pronounced.

Crepitations may occur with palpation in patients with esophageal perforation (13). In addition, if there is an esophageal rupture, swelling may be seen in the patient's face area.

Although the abdominal examination may be normal at first, there may also be tenderness and distension in the abdomen. As a result of perforation in the small intestine, ileus secondary to the contents that has spread into the abdomen may develop, and the patient may present with symptoms of distension or peritonitis secondary to intra-abdominal perforation.

With rectal exam, it can be seen that there is tenderness in inflammatory conditions in the lower region.

In such patients, hemogram and biochemical parameters are examined as a laboratory. Especially C-reactive protein (CRP) is very helpful in demonstrating gastrointestinal leak after colorectal or metabolic surgeries.

5. Diagnosis

Although gastrointestinal perforation may be suspected based on anamnesis and physical examination, it may be desirable to support the suspicion with tests. However, if the suspicion is very strong, abdominal exploration may be required as a diagnostic surgery without waiting for the examination.

Evaluation of the patient with abdominal complaints should generally begin with radiographs of the lungs and abdomen. If the patient is bedridden, an abdominal X-ray should be taken in the lateral decubitus position. Although the rate of help of such examinations is around 90 percent, unfortunately, perforation cannot be ruled out (14). While the sensitivity in detecting extraluminal air in

direct abdominal films is around 70 percent, this rate increases when the patient is in an upright or supine position for about 20 minutes before filming (14,15).

Although direct abdominal films say that there is perforation, they cannot tell its focus. Direct exploration is the best approach for a patient with free air on the film, tenderness in the abdomen, and no recent surgical history.

If subcutaneous emphysema is seen on the chest X-ray, the neck area should be carefully examined.

Computerized tomography (CT), which is specific for showing extraluminal air, is the most commonly used examination in emergency departments. CT is superior to direct abdominal films in the evaluation of the suspected patient. CT is very helpful in demonstrating continuity and thickening of the intestinal wall, material causing perforation, extraluminal leakage, occlusion site, or intestinal-related inflammatory conditions (16).

Although the presence of free air in the abdomen is seen as a sign of perforation, it should be kept in mind that after laparoscopic operations, free air in the abdomen can be seen for about a week in half of the patients (17).

Endoscopic examination, esophagogastric X-ray, intravenous and oral films may be required to find the suspected perforation while evaluating the organs, and additional examinations with rectal contrast material may be required (18). Barium should not be given as an oral contrast agent at first, but if it is shown that there is no leakage out of the lumen in the examinations taken with contrast material and if perforation is suspected, barium can be given instead of control tomography (19).

Endoscopic examination is a good evaluation especially for the patient with suspected esophageal perforation (20). However, since the process itself has the ability to perforate, CT should still be the first choice in such cases. Apart from this, the presence of blue color in the tube helps in the diagnosis of esophageal perforation in a patient with methylene blue chest tube given with a carefully inserted nasogastric tube.

6. Approach and Treatment of Perforation

Evaluation of patients with gastrointestinal perforation should be to turn off oral intake and give intravenous fluid support, and receive antibiotic therapy under intensive care unit conditions. Proton pump inhibitors should also be started.

Electrolyte disturbances and volume loss in the body are high in such patients. All kinds of surgical treatment should be done quickly.

Antibiotic selection should be made according to the suspected perforation. If the perforation site is unknown, a broad-spectrum antibiotic is a better choice.

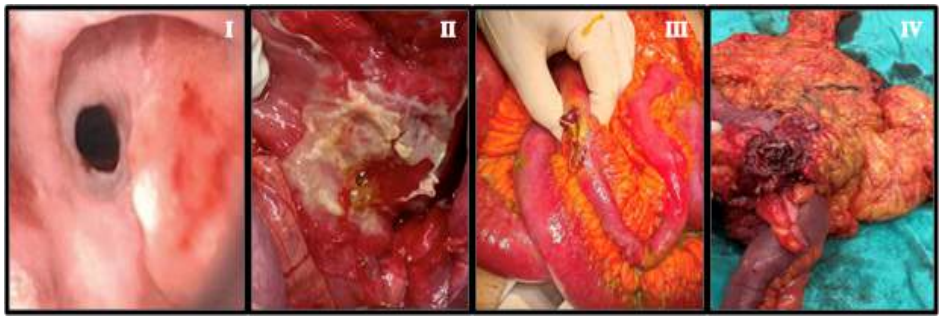
Such patients may not require an urgent operation, and they can be followed conservatively. When we look at who is followed up non-operatively, we are confronted with conditions such as perforated area and intra-abdominal fistula formation in a limited manner (21). In addition, the conservative approach in this way shows us a lower morbidity and mortality rates compared to the operative approach.

It should be kept in mind that in diagnoses such as esophageal perforation, colonic diverticulitis and perforated appendicitis, the use of antibiotics along with drainage, procedures such as enterostomy or stenting may be an appropriate approach for patients (22).

Immediate abdominal exploration will be required if the perforated location cannot be determined and the contamination situation cannot be limited.

If perforation is suspected in cases such as persistent and increasing abdominal pain or signs of peritonitis, ischemia in the intestinal organs in the examinations, and intestinal obstruction, they will benefit from emergency surgery.

Treatment approaches are also important in the case of perforations of solid organs (Picture 1).



Picture 1. Perforation examles

I: esophagus perforation
II: stomach perforation
III: small bowel perforation
IV: colorectal perforation

6.1. Esophagus Perforation

Esophageal perforations usually present with perforations in the wall secondary to biopsy or sclerotherapy. The patient feels pain during swallowing (23). Morbidity is most common in thoracic injuries. Esophageal perforation is mostly seen as iatrogenic or secondary to trauma. Rarely, ingestion of a foreign body, ingestion of caustic substances or a disease in the esophagus may also be the cause of perforation. In such cases, stent placement by endoscopic methods has become increasingly common. Thus, the perforated place is closed and the patient enters the healing process. However, complications such as stent-related bleeding, fistula development, and stent displacement should not be forgotten. In cases where the stent does not help, open surgery is also an option. Primary repair as open surgery, repair over drain and esophagectomy in cases of obstruction or cancer can be mentioned (23).

6.2. Stomach Perforation

Although peptic ulcer disease is the most common cause of gastric and duodenal perforation, iatrogenic, trauma, foreign body ingestion and tumors also play a role in the etiology. Most stomach perforations usually require surgical repair using the Graham technique. However, a biopsy should be taken from the ulcer margin to rule out tumor-related perforation.

6.3. Small Bowel Perforation

Small bowel perforation may be iatrogenic, secondary to trauma, or associated with ileus. Injuries can be overlooked, especially in surgeries performed with the closed entry technique. Perforations are also seen due to tumor, secondary to foreign body ingestion, and displaced stent. In the treatment, the perforated ans is sutured in full thickness or resections can be performed.

6.4. Colon and Rectal Perforation

Colon and rectal perforations are due to tumor, diverticulum, iatrogenic or trauma. Most patients with diverticulitis are followed conservatively unless there are severe complications (24). It is known that perforation may occur during colonoscopy as well as after the use of enemas for colon cleansing. As a treatment, if the perforation is not large, it can be sutured with closed surgery, if it is large, resection is required (25).

7. Conclusion

Gastrointestinal system perforations are among the serious health issues that can lead to emergency situations. A perforation refers to a hole in the walls of the stomach or intestines, often caused by factors such as ulcers, diverticulitis, trauma or surgical complications.

Perforations can increase the risk of infection in the abdominal cavity and may cause damage to vital organs over time. The severity of this condition depends on the location and size of the perforation, as well as how quickly intervention occurs.

Treatment typically involves surgical intervention, repairing the perforated area and controlling infection. Successful outcomes hinge on early diagnosis of the perforation anf the implementation of an appropriate treatment plan.

In conclusion, gastrointestinal system perforations can lead to serious complications requiring urgent medical intervention. Early diagnosis and effective treatment can positively impact the patient's health.

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CHAPTER VIII

GASTROINTESTINAL BLEEDING

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Gastrointestinal bleeding (GIB) is among the most common causes of emergency hospitalizations. It is classified into two main groups, namely upper and lower GIB, depending upon whether the bleeding point is above or below the ligament of Treitz. The annual number of hospital admissions in the United States related to upper gastrointestinal bleeding (UGIB) is 350,000 (1). UGIB is less common among women compared to men. The spectrum of symptoms ranges from life-threatening bleeding to iron deficiency anemia, and although many diagnostic and treatment methods have been developed, GIB still has significant mortality rates. The most common causes of UGIB are peptic ulcers, esophageal varices, and erosive esophagitis. It is generally examined in four different forms: upper GIB, lower GIB, obscure occult (hidden) bleeding, and obscure overt (obvious) bleeding.

Anamnesis is highly important in treating GIB, as it is in every patient. Vomiting that produces fresh, red blood (hematemesis) or coffee grounds-colored vomit is a sign of the presence of older, pending blood in the stomach. Dark tar-colored, foul-smelling stool (melena) from the rectum typically indicates upper gastrointestinal bleeding. 50 to 100 mL of blood is sufficient for the development of melena in a patient. Oral iron or bismuth intake may dye the stool green-black, but no foul odor is observed in such cases. Light red bleeding from the rectum (hematochezia) is generally the result of a colorectal hemorrhage. However, UGIB can also be observed in bleeding episodes that produce 1,000 mL or more blood.

The medical history of the patient is also important in relation to GIB. For instance, insights regarding the presence of peptic ulcers, nonsteroidal anti-

inflammatory (NSAID) use, cirrhosis, anticoagulant use, etc. provide pivotal information concerning the bleeding site of the patient.

UPPER GASTROINTESTINAL BLEEDING

Bleeding that is proximal to the ligament of Treitz is defined as upper GIB. In the United States, the annual number of patients who present to emergency departments with upper GIB is 350,000. (1) Increasing age (particularly in the 5th to 8th decades of life), comorbidities, and recurrent bleeding are associated with increased morbidity and mortality. It is observed two times more frequently among men compared to women (2). Its mortality rate is around 10% in both men and women. However, the mortality rate increases in patients over 60 years of age (3, 4). Mortality reaches 30% in patients with varicose bleeding, and the rate of re-bleeding reaches 50% to 70%. The most common cause UGIB is peptic ulcer. Other causes are gastric erosions, esophageal varices, Mallory-Weiss tears, esophagitis, erosive duodenitis, and malignancies. Advanced age (>60) is an independent factor that increases mortality in relation to upper GIB. According to the American Society for Gastrointestinal Endoscopy (ASGE), mortality rates are 3.3% for those between 21 and 31 years old, 10.1% for those between 41 and 51 years old, and 14.4% for those between 71 and 80 years old. Risk factors related to high mortality, bleeding recurrence, and the need for endoscopic or surgical hemostasis in bleeding are as follows: being older than 60 years of age, the presence of severe comorbidity (cardiopulmonary failure, renal failure), the presence of active bleeding signs (hematemesis, fresh blood in the rectum), hypotension, shock, the need for more than six units of blood transfusion, and severe coagulopathy.

When evaluating a patient with upper GIB, it is important to first determine whether the bleeding originates from varicose veins or non-variceal bleeding. For this reason, cirrhosis-related findings should be accounted for while taking the patient's anamnesis and physical examination.

A patient with suspected acute bleeding presents with a decrease in hemoglobin in the form of normochromic normocytic anemia. If the patient has underlying chronic bleeding or thalassemia, low mean corpuscular volume (MCV) can also be observed. Locopenia and thrombocytopenia as the result of hypersplenism and portal hypertension should be considered, which relates to varicose bleeding. The increase in blood urea nitrogen (BUN) is not correlated with creatine.

The approach to the bleeding patient begins with securing the airway, opening the vascular accesses in both arms, and stabilizing the hemodynamics.

Notably, in patients presenting with hypovolemic shock, 20 to 25% of the intravascular volume is lost, and extensive bleeding occurs. If orthostatic hypotension and tachycardia are present, the amount of loss should be estimated at 10 to 20%. No clinical signs are observed in patients with a loss of < 10%. For every mL of blood loss, 3 mL of crystalloid should be administered. In patients with high comorbidity, central venous pressure should be monitored by inserting a central catheter, and a urinary catheter should be inserted to monitor urine output.

Erythrocyte suspension (ES) should be initiated in patients with low hematocrit and non-recovering hemodynamics despite fluid resuscitation. The lower limit for starting ES has been established to be 7 g/dL. However, this threshold can be higher in patients with a history of coronary artery disease (5). Platelet suspensions should be given to patients with a platelet count of less than 50,000 x 103 /mL. In patients who need an emergency endoscopy, the INR should be <2.5; if the patient is using warfarin, warfarin should be discontinued and four-factor prothrombin complex should be used.

Immediately after this evaluation, an endoscopy should be performed in the first 24 hours to determine The origin of the bleeding, to stop the active bleeding, to identify the underlying anomaly, and to prevent bleeding (6). An endoscopy should be performed within the first 12 hours in patients with varicose bleeding and high risk.

Peptic ulcers are the most common cause of upper GIB. Proton pump inhibitors (PPIs), H2 receptor blockers, somatostatin analogs, and prostaglandins are used in patients with ulcers caused by bleeding. Proton pump inhibitors (PPI) increase gastric pH, decrease pepsin activity, diminish clot dissolution, and increase platelet aggregation. Patients with peptic ulcers with active bleeding should be treated with an IV bolus of 80 mg before an endoscopic treatment followed by an infusion of 8 mg/hour for 72 hours (7). After the hemodynamic stability is achieved, 2-40 mg/day maintenance therapy should be initiated. Some drugs, such as aspirin, NSAIDs, tetracycline, doxycycline, clindamycin, anticoagulants, and steroids, may cause bleeding by causing erosive gastropathy.

PEPTIC ULCER BLEEDING

Peptic ulcer bleeding accounts for half of all GIB. Although the incidence of duodenal ulcers is higher than that of gastric ulcers, bleeding rates are similar. The most common causes of peptic ulcers are Helicobacter pylori and aspirin or NSAID use. HP destroys the gastric and duodenal mucus barrier, causing inflammation and mucosal erosions. Ulcerating erosions deepen mucosa,

particularly in the gastroduodenal region, causing necrosis, pseudoaneurysms, and bleeding in the gastroduodenal artery serosa there. Helicobacteria pylori (HP) colonization is observed in 95% of non-bleeding ulcer patients and in 71% of bleeding patients. Therefore, HP eradication reduces ulcer recurrence and bleeding recurrence.

NSAID use causes a 40-fold increase in gastric ulcers and an eight-fold increase in duodenal ulcers. NSAIDs cause mucosal barrier defects by diminishing mucosal prostaglandin synthesis and inhibiting cyclooxygenase enzymes.

Peptic ulcer bleeding ceases spontaneously in 80% of cases. Ulcers observed in endoscopies after the first approach to the ulcer are classified using Forrest classification and risk classification. Hemorrhage of LGIB signs of ulcers are defined as follows: clean floor (Forrest III), superficial spots (Forrest IIc), adherent clot at the base of the ulcer (Forrest IIb), non-bleeding visible vessel (Forrest IIa), and active bleeding (gushing Forrest Ia, leaking Forrest Ib) (8). Using this classification, the prognosis for the ulcer and the possibility of rebleeding are evaluated. The most risky group in terms of rebleeding is characterized by active arterial bleeding and a protuberant visible vessel at the base of the ulcer. Such ulcers require an urgent endoscopy. The issue of intervention in relation to ulcers with adherent clots on the surface is controversial. Ulcers characterized by clean-based spots do not require intervention.

Rockall scoring, which includes age, systolic blood pressure, heart rate, comorbid diseases, endoscopic findings, and new bleeding focus images, is most commonly used to determine the prognosis of the bleeding. Patients with a score of 0 to 2 are considered low-risk and are discharged from the emergency department in the early period (9).

Endoscopic treatment methods for peptic ulcer bleeding are most commonly used by the heater probe. The most common injection treatment is a 1/10,000 adrenaline injection of 0.5-1 cc into four quadrants.

ESOPHAGUS VARICOSE BLEEDINGS

Esophageal bleeding is the second most common cause of UGIB bleeding. It is a significant complication of portal hypertension and is present in 30 to 70% of cirrhosis cases. Mortality rates for hemorrhages without treatment are around 50 to 70%. While mortality is 30% in isolated medical treatment, one-year survival rates are 40% (10). Gastric varices may accompany esophageal varices in 20% of cases. Today, esophageal variceal bleeding is regarded as a stand-alone criterion for decompensated cirrhosis, according to the Boveno

consensus. Increased risk parameters for variceal bleeding include high-hepatic vein pressure gradient (HVPG>12 mmHg), varicose diameter, endoscopic red color sign, Child C, active alcohol intake in chronic alcoholic liver disease, distal esophageal regional changes, bacterial infections, and aspirin or NSAID intake. 12 mmHg is the critical value for varicose bleeding. Varicose bleeding frequently stops spontaneously. However, due to the 50% probability of re-bleeding within 5 to 7 days, it requires intervention.

PRIMARY PROPHLAXY

These are treatments to prevent the development of varicose bleeding in patients who have not experienced varicose bleeding. While the annual probability of recurrence in a patient who has had varicose bleeding is 70%, it is 20% in a patient who has not. The most commonly used non-selective beta-blockers are propranolol and nadolol. A decrease of 1/5 of the pressure is sufficient for prophylaxis. Isosorbid-5-mononitrate can be used in cases in which beta-blockers are contraindicated.

TREATMENT IN ACUTE VARICOSE BLEEDING

In these patients, volume overload should be avoided, excretory enemas should be utilized to reduce the risk of hepatic encephalopathy, and prophylaxis with antibiotics should be applied to reduce the risk of spontaneous bacterial peritonitis. During bleeding, endoscopic interventions should aim to decrease portal tension. For this, pharmacological treatments, shunt operations, transjugular intrahepatic portosystemic shunts (TIPS), Sengstaken–Blakemore balloon applications, sclerotherapy, and band ligation are applied. Among potential pharmacological agents, vasopressin, somatostatin analogues, terlipressin, and nitroglycerin are used. These reduce portal blood flow by vasoconstricting the splanchnic area. Somatostatin is administered as a 250 μg IV bolus followed by 250-500 μg/hour, and octreotide is administered as a 100 μg bolus followed by an infusion of 50-100 μg/hour. Terlipressin is a vasopressin analog and is the only drug that has been demonstrated to increase survival in patients with active varicose bleeding. It is recommended to administer 2 mg intravenously every four hours and to continue at a dose of 1 mg every four hours for five days after bleeding is controlled. Nitroglycerin, on the other hand, causes systemic vasodilation as well as splanchnic vasoconstriction, and it is generally recommended to be used in conjunction with vasopressin.

Endoscopic treatments include sclerotherapy and band ligation. Due to the complications of sclerotherapy, band ligation is preferred more than two weeks apart.

The double-balloon Sengstaken Blakemore tube can also obliterate varicose veins. The gastric balloon is inflated with 200-300 mL of air and weighed at 500 mg, and half of the stomach is given air through the esophagus tube. The balloon is deflated after 12 hours, and bleeding is controlled.

Transjugular intrahepatic portosystemic shunts (TIPS) and surgical shunt operations are used in patients who are unresponsive to medical and endoscopic treatment. While the success rate of TIPS is 90%, the probability of occlusion is 40%. For this reason, it is done for the purpose of bridging prior to transplantation in necessary cases. A surgical shunt is applied only in the child A group.

SECONDARY PROPHLAXY

It is prophylaxis to prevent secondary bleeding, and the most effective method involves beta blockers and band ligation.

OTHER REASONS FOR UPPER GIB

Other causes of upper GIB include Mallory-Weiss tears, Cameron lesions, erosive gastritis, gastric antral vascular ectasia (GAVE), erosive esophagitis, Dielulefoy lesions, gastric-duodenal cancer, and gastricleiomyomas. Aortoenteric fistulas are rarely observed and is mostly seen in the 3rd and 4th continents of the duodenum. It occurs after continent and aortic graft surgeries.

The bleeding of Cushing's ulcers after head trauma and Curling ulcers after burns are evident. Angiodysplasias constitute 2 to 4% of upper GIBs.

Upper GIB associated with vomiting is also observed in the lower esophagus and upper stomach. The severity of bleeding is proportional to the depth of the tear. In rare cases, esophageal rupture (Boerhave's syndrome) may occur as a result of severe vomiting. Bleeding, air in the mediastinum, left pulmonary infiltration, and subcutaneous emphysema are signs of Boerhave's syndrome.

Mallory-Weiss syndrome, which is responsible for 15% of cases of upper GIB, may present with severe vomiting, burping, and massive hemorrhages caused by linear deep tears in the gastric cardia.

Cameron's lesions manifest as erosions and ulcers that develop as a result of ischemia and trauma in the part of the stomach that is proximal large hiatal hernia.

Dieulefoy's lesion refers to a vascular malformation located along the lesser curvature of the stomach. Due to the wide veins at the base of the lesion, it may cause massive GIB.

LOWER GASTROINTESTINAL BLEEDING

Lower GIB increases with expansions in the elderly population and the use of antithrombolytics (11, 12).

ESOPHAGUS VARICOSE BLEEDING

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LOWER GASTROINTESTINAL BLEEDING

Lower GIB has increased with expansions in the elderly population and the use of antithrombolytics (11, 12).

Colonoscopy, radionuclide scans, and angiography are used for diagnosis. After the patient's hemodynamic stability is ensured, it is appropriate to start with a colonoscopy by performing rapid bowel cleansing. It is important to observe the presence of bleeding, the bleeding focus, and the lesions that do not bleed but that have the potential to bleed (stigma of the recent hemorrhage) in a colonoscopy. If coagulation was not applied in patients with stigmata of the hemorrhage (SRH), re-bleeding was observed within 30 days in 66% of patients, and bleeding was not observed in patients without SRH (20). The lower gastrointestinal system bleeding (LGIB) guidelines define a colonoscopy performed within the first 24 hours after admission as an early colonoscopy. While there is no significant difference in the mortality and morbidity between an early colonoscopy and an elective colonoscopy, an early colonoscopy is more useful in terms of achieving hemostasis (21). However, there is no definite

order regarding when an endoscopy will be performed and who will receive it. Studies have demonstrated that CT angiography is 85.2% sensitive and 92.1% specific in determining the bleeding focus (22). In patients with severe bleeding in whom hemodynamic stability cannot be achieved, and in patients who cannot tolerate colon cleansing, CT angiography should be performed first to detect the bleeding focus before surgery or angiography (23). Notably, the sensitivity of CT angiography is low, particularly in cases in which diverticular hemorrhages are intermittent and the bleeding rate is low. For this reason, the sensitivity of CT angiography, especially in the first four hours after hematochezia, is the highest (24).

The greatest advantage of angiography and embolization is the sudden cessation of 40% to 100% bleeding in cases of severe bleeding. After this procedure, the rate of re-bleeding was found to be 15% (25). The LGIB guideline recommends the use of angiography embolization in cases involving severe hemorrhages whose hemostasis cannot be corrected. For angiography to detect the focus, 0.5 mL/min bleeding is required. The most important complications of this procedure are contrast nephropathy and local ischemia necrosis. In highly severe bleeding, diagnostic laparotomy and intraoperative colonoscopy can be performed.

For diverticular hemorrhages, bipolar coagulation, adrenaline injection, and hemoclips can be used with a colonoscopy. In cases of recurrent bleeding, the divertilar segment should be resected. Electrocoagulation or argon plasma coagulation can be used in angiodysplasias. In ischemic colitis, the regimen is closed, and IV hydration and appropriate antibiotic therapy are initiated. Vasopressin can be used in cases in which the bleeding focus cannot be determined. When this is ineffective, highly selective embolizations may be attempted.

The complication rate of surgeries for LGIB is 60%, and the mortality rate is 16% (26). For this reason, it should be used for patients whose surgical hemodynamics did not improve and if other methods could not produce results. LGIB surgical indications include when bleeding cannot be controlled with non-surgical interventions despite the detection of bleeding focus, when patients have undergone more than six units of ES replacement since hospitalization, and when patients' bleeding focus cannot be detected despite all procedures. While the rate of rebleeding after surgery in LGIB was 0 to 4% in total colectomy patients, it was found to be 4 to 18% in limited resections. However, while the mortality rate after total colectomy was 20 to 40%, it was found to be 7 to

22% in limited resections (27). In previous studies regarding surgical treatment for acute LGIB [68–71], the rate of rebleeding was higher after limited colon resection (4–18%) than after total colon resection (0 to 4%). In most of these studies [68–70], the mortality rate after limited colon resection (7 to 22%) was lower than after total colon resection (20 to 40%).

MEDICATION USE FOR LOWER GASTROINTESTINAL BLEEDING

The use of antithrombotic drugs in relation to LGIB should be considered. Although there are not sufficient studies regarding the discontinuation of these drugs, the general opinion is that drugs are discontinued until hemostasis is achieved and resume as soon as hemostasis is achieved (23).

NON-STEROIDAL ANTI-INFLAMMATORY DRUGS

In the studies performed, a significant recurrence rate was observed within 12 months in patients with diverticular bleeding compared to those who did not use NSAIDs (28).

ANTIPLATELET AGENTS

Antiplatelet agents are known to increase both direct LGIB and recurrences (29). However, while 37% of the patients who discontinued aspirin experienced a cardiovascular event, it was 23% in those who did not (30). For this reason, aspirin should not be discontinued for secondary prophylaxis in a patient with a known history of cardiovascular events. However, the discontinuation of aspirin in primary prophylaxis in patients with low cardiovascular risk does not increase or decrease the risk of GIB. The discontinuation of dual antiplatelet therapy during the first 90 days following coronary artery stenting strongly increases the risk of myocardial infarction and death (31). Dual antiplatelet therapy should be continued in such patients. However, in stents made in more peripheral arteries, the second antiplatelet without aspirin can be discontinued for up to seven days (32).

ANTICOAGULANTS

Existing studies do not adequately discuss the effects of bridging with anticoagulants and heparin.

PROTHROMBIN TIME-INTERNATIONAL NORMALIZED RATIO (INR)

Calvus reports that a moderate increase in INR (<2.5) does not affect endoscopic treatment and rebleeding for non-variceal UGIB (33). It recommends the use of reversing agents before endoscopy in cases with an INR >2.5, but the evidence is inconsistent. In fact, some studies have demonstrated that a high INR value does not increase rebleeding in all cases of GIB (34). However, a high INR value is an indicator of thromboembolism risk within 90 days in all gastrointestinal bleedings (35). This is attributed to the underlying comorbid diseases that cause the high INR value.

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CHAPTER IX

ILEUS

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Ileus is the obstruction of the movement of intestinal contents in the peristalsis direction within the lumen. Obstruction may occur at the level of the small intestine or large intestine. If mechanical causes such as adhesions, hernias, tumors, and inflammatory diseases play a role in the etiology of ileus, this situation is defined as mechanical intestinal obstruction. In adynamic ileus -associated with decreased intestinal motility, functional obstruction develops without a mechanical obstruction.

1. Small Intestine Obstructions

Small bowel obstruction constitutes an important part of applications to the emergency department due to abdominal pain and therefore of emergency surgical interventions. (11) Small bowel obstruction, which is generally a surgical disease, is divided into three classes according to its relationship with the intestinal wall. Adhesions, hernias and metastatic lesions prevent the passage of intestinal contents by external compression. While bezoar and gallstone ileus are among the intraluminal causes, Crohn's disease, tumors and other infective events cause mechanical obstruction with their effects on the intestinal wall. Although its incidence has decreased in parallel with the widespread use of laparoscopy, postoperative adhesion remains the most common cause of small bowel obstruction today. (2) The most important risk factor leading to adhesions is previous surgeries. It has been observed that adhesions develop more frequently after pelvic region surgeries such as colorectal and gynecological surgeries. (12) Adhesions occur as a result of fibrous bands formed during wound healing after surgery. (5) Other common causes in the etiology are hernias, tumors and inflammatory bowel diseases.

Table 1. Factors involved in the etiology of small bowel mechanical obstruction.

	Common causes in the etiology
Intraluminal	Bezoar, gallstone ileus, intussusception
Intramural	Crohn's disease, infective conditions, tumors
Extraluminal	Adhesions, hernias, metastatic lesions

1.1. Pathophysiology

In small bowel mechanicalobstructions, dilatation occurs as a result of air and fluid accumulation in the intestinal segment proximal to the obstruction area. Although an increase in intestinal motility is observed in the beginning to overcome the obstruction, bowel movements slow down in the course of time. The main source of accumulated gas is swallowed air. However, there is a change in the small intestinal flora proximal to the obstruction and an increase in bacterial translocation is observed. (8) These bacteria also cause gas accumulation through the fermentation of intestinal contents. (7) Fluid and gas accumulation increases intraluminal pressure. This increase in pressure causes edema by blocking venous return and disrupts vascular perfusion, leading to ischemia and subsequently necrosis in the intestinal mucosa, which is fed by the end-arterials and is very sensitive in terms of blood supply. (6) This condition, which is also defined as strangulated bowel obstruction, compromises the integrity of the wall in the area of ischemia and necrosis, and there is a predisposition to perforation.

In partial obstructions, the clinical presentation is milder than in complete obstructions, since there is a slight passage of air and intestinal contents to the distal. One of the most dangerous situations encountered is closed loop obstructions in which the proximal part of the intestine is also closed. In this context, the development of ischemia and necrosis will be faster, since gas and liquid cannot progress in the proximal and distal directions. Because closed loop obstruction is a strong predictor of ischemia and necrosis, it is considered a surgical emergency. (10)

In short, small bowel obstruction is a serious condition that can progress to mucosal ischemia, necrosis and perforation, regardless of its etiology. The most important factor determining the prognosis is obstruction being partial or complete. While partial obstructions have a better prognosis, the treatment of complete obstructions is more difficult and requires more frequent surgical solutions. (13)

1.2. Clinical Presentation

The degree of symptoms and clinical complaints of patients with small bowel mechanical obstruction differ depending on whether the obstruction is partial or complete and its localization. Patients often experience nausea-vomiting, abdominal distension, and cramping abdominal pain. Vomiting is prominent in proximal occlusions and since undigested food is removed, fluid-electrolyte loss is greater than in distal occlusions. Therefore, proximal SBOs have an acute course and patients become symptomatic more quickly. However, the nature of the vomiting also gives the surgeon an opportunity to guess about the part of the bowel where the problem originates. It is possible to talk about vomit containing bile in proximal SBOs and fecaloid vomit in distal SBOs. Since the distally located obstruction allows for a large intestinal reservoir, abdominal distension due to the dilatation of the intestines often develops in these patients. On the other hand, added to the clinical picture; fever, tachycardia, signs of peritoneal irritation, persistent abdominal pain are signs that raise suspicion in terms of ischemia and necrosis.

1.3. Diagnosis

1.3.1. Laboratory Parameters

There is no specific laboratory test to indicate mechanical small bowel ileus. Laboratory parameters vary depending on the degree, location, and duration of the obstruction. Electrolyte imbalance is common due to fluid loss after vomiting. Due to dehyrdration ,deterioration in kidney function tests and an increase in hematocrit are expected results. Elevated white blood cell count, increase in lactate level with acute phase reactants, and metabolic acidosis are among the markers that indicate poor prognosis associated with ischemia and necrosis. In addition, there are studies supporting that the increase in intestinal fatty acid binding protein levels secreted from necrotic enterocytes is a precursor to ischemia. (14) Although laboratory parameters do not carry a diagnostic value on their own, they are significant when interpreted together with clinical findings.

1.3.2. Radiological Tests

1.3.2.1. Abdominal X-ray

Abdominal radiography is the first option recommended in patients with clinical symptoms of ileus and suspicious laboratory findings. It has high

specificity and sensitivity. (16) However, not providing sufficient information about etiology stands out as its biggest handicap. Air-fluid levels are characteristic radiographic signs of SBO. The presence of air-fluid level in the same small intestine segment, small bowel loops larger than 2.5 cm, and air-fluid levels differing more than 5 mm are the main findings on abdominal X-ray of SBO. (16) The absence of gas in the distal segments of the colon and rectum also supports SBO. Free air in the abdomen can also be detected on abdominal radiographs and indicates perforation. However, abdominal radiographs taken with contrast material also play an important role in the diagnostic phase of ileus. Detection of orally administered water-soluble contrast agent in the colon in the abdominal X-ray after 24 hours is important in showing that the small bowel obstruction will be resolved. (17, 18) In addition, contrast material shortens the length of hospital stay. In summary, radiological tests should be started with abdominal X-ray in patients with suspected ileus. However, it should not be forgotten that abdominal X-ray may be normal in proximal occlusions.

1.3.2.2. Ultrasonography (USG)

Ultrasound; Despite its advantages such as rapid application, protection from the negative effects of radiation and low cost, its role in the diagnostic process of ileus is limited except for the evaluation of incarcerated hernias. Although it has high sensitivity and specificity in the diagnosis of SBO (15), it provides limited information to the surgeon about strangulation and etiology. (21) As it shows peristalsis, enlargement of the intestinal loops and free fluid in the abdomen, the presence or absence of ileus can be judged with the help of ultrasound. In addition, USG can be used in the follow-up of patients who are followed conservatively due to SBO.

1.3.2.3. Computed Tomography (CT)

Computed tomography (CT) is a radiological diagnostic method with high sensitivity and specificity, which is very useful to the clinician in the diagnosis of SBO and in determining the etiology and passage point. (21, 23) One of the most important benefits of CT compared to other radiological examinations is that it reveals strangulation. Because late recognition of strangulation due to SBO is closely associated with increased morbidity and mortality. (26) Therefore; Patients whose physical examination findings and laboratory parameters are suspicious for strangulation should be evaluated with CT without delay. Presence of mesenteric fluid and decreased contrast enhancement in the intestinal wall

are the main findings pointing to strangulation on CT. (19, 20, 22) In addition, mesenteric fluid, decreased intestinal wall contrast and lack of small bowel feces sign on CT are predictive of failure of conservative treatment. (24) CT scans containing these symptoms may guide the surgeon to make an operation decision. On the other hand, the other advantage of CT is that it answers questions about etiology. Most of the post-adhesion surgeries are treated with conservative methods. (25) In this context, the presence of adhesion revealed by CT may lead the surgeon to conservative follow-up. In summary, CT is an important method that helps the surgeon in the diagnosis of SBO, determining the etiology and passage zone, evaluating the strangulation and predicting which patients will be operated.

1.3.2.4. Magnetic Resonance Imaging (MRI)

MR imaging has 95% sensitivity and 96% accuracy in the diagnosis of SBO. (30) There is no significant difference in the diagnostic accuracy of SBO and CT. (28) However, despite these data, SBO has no place in routine use in the diagnosis process due to its high cost, need for experience and long scanning time. It is preferred only in selected patients. In particular, MR imaging offers a good alternative to CT in order to minimize the radiation load in children and pregnant women.

1.3.2.5. Management of Small Bowel Obstructions

SBO constitutes an important part of the applications made to the emergency department due to abdominal pain. This high rate of admission not only makes the management of SBO important, but also necessitates it to be known by physicians other than surgeons. Patients with SBO can be followed conservatively or surgically.

1.3.2.6. Medical Treatment

In the first stage of SBO management, the patient should be evaluated for the need for emergency surgery. This is one of the most critical aspects of patient management with SBO. In this context, if a diagnosis of SBO is made outside the surgical service, surgical consultation should be promptly requested. Then, oral intake should be stopped and intravenous fluid replacement should be started immediately to replace the lost fluid. Since gas and fluid accumulation proximal to the obstruction increases the risk of ischemia, necrosis and perforation, drainage with a nasogastric tube should be applied to decompress these intestinal

segments. (31) The parameters in blood tests should also be reviewed in terms of electrolyte imbalance and electrolyte disorders should be corrected by taking into account kidney functions. (32) In order to evaluate the adequacy of fluid resuscitation, hourly urine output is also measured by performing bladder catheterization.

Patients with SBO frequently experience nausea, vomiting, and abdominal pain. Symptomatic treatment should also be planned for these complaints of the patients. It is aimed to control pain with analgesic drugs. The point that should be underlined here is that analgesics should not be given before physical examination by the surgeon. Because the administration of analgesics may mislead the surgeon by obscuring the physical examination findings. Another annoying condition for patients is vomiting. Antiemetic agents are put into effect at this stage. It has been shown that the use of dexamethasone contributes to the improvement of symptoms in malignant SBOs with resistant vomiting. (37)

Somatostatin analogs are also used to reduce GI secretions for symptomatic relief in SBOs due to malignant causes. (33) The use of somatostatin analogues not only improves symptoms but also reduces the need for NG tubes. (34) There are also studies showing that corticosteroid use reduces intestinal edema, fibrin and helps to resolve intestinal obstruction in this patient group. (36)

1.3.2.7. Surgical Treatment

The duration of follow-up and when the surgical decision will be taken in patients who are followed conservatively due to SBO are a matter of debate. The general opinion is that conservative follow-up can be extended up to 72 hours. (44) Since development of ischemia, strangulation and peritonitis are contraindicated in terms of conservative follow-up, these patients should be operated immediately. In case of clinical deterioration in conservatively followed patients, surgical solutions come into play. Changes in vital signs such as fever, low blood pressure, tachycardia, and laboratory parameters such as leukocytosis, acidosis in blood gas, and lactate elevation are signs that reflect clinical worsening. Therefore, in the presence of these markers in patients under conservative follow-up, the surgeon should not delay in making an operation decision.

In some studies, surgical treatment is recommended if the orally administered contrast does not pass into the colon within 24 hours. (41) In this respect, it can be said that CT helps the surgeon in predicting the need for surgery as well as the diagnosis of SBO.

Contrary to previous knowledge, current studies show that the laparoscopic method can be safely preferred in the surgical treatment of SBO. (38, 39) Postoperative outcomes are better after laparoscopic bridectomy, and the rates of morbidity, mortality, and surgical site infections are lower than with the open approach. In the presence of strangulation, the laparoscopic method can be preferred and the results are similar when compared with the open method. (40) In addition, the return of bowel movements is earlier after laparoscopic surgery. (43) Despite these positive results, iatrogenic bowel injury is more common in laparoscopic surgery. (42) In this context, care should be taken in the selection of patients to be treated with the laparoscopic method. The presence of severely dilated bowel loops and multiple adhesions are the main factors suspending the surgeon from the decision of laparoscopy.

CHAPTER X

PEPTIC ULCER PERFORATION

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1. Introduction

Peptic ulcer disease (PUD) has been among the common diseases all over the world for many years. Innovations in medical technology, the development and advancement of endoscopy have allowed us to better understand the causes and mechanism of this disease. PUD occurs mainly due to many factors such as Helicobacter pylori (H. pylori), nonsteroidal anti-inflammatory drugs (NSAIDs), corticosteroids or stress.

Since the second half of the last century, the incidence of PUD has decreased with the discovery of proton pump inhibitors (PPI) and other anti-acid drugs. (1) In addition, in parallel with the widespread use of h.pylori eradication therapy, the incidence and prevalence of uncomplicated PUD has gradually decreased. (2) Peptic ulcer perforation (PUP) is seen in 2-10% of peptic ulcer patients and accounts for more than 70% of PUD-related deaths. (3)

2. Peptic Ulcer Disease

Complications of peptic ulcer and risk factors for their recurrence include corticosteroid, acetylsalicyl acid (ASA) and NSAID use, helicobacter pylori infection, high age, presence of comorbidity, and ulcer larger than 1 cm. Delay between diagnosis and surgery, high age, presence of comorbidities and septic shock are associated with increased mortality. (4)

As mentioned above, the incidence of PUD decreases with the widespread use of PPIs and H.pylori eradication therapy, but the use of ASA and NSAIDs, which cause side effects in the gastrointestinal tract, is also becoming more common. (5) Interestingly, H. pylori shows a variable prevalence (0-90%) in

perforated ulcers, and ulcers can develop in the absence of H. pylori infection and the use of NSAIDs. (6)

Although the rates vary slightly in various studies, A complication occurs in approximately 10-20% of patients with PUD. (4) The most common complications are bleeding, perforation and obstruction, respectively. (7) Although bleeding was reported 6-7 times more common than perforations; The most common emergency surgery indication for PUD is perforation. (8,9) In some countries such as Turkey, perforation has become the most common complication in recent years. Unlike the literature, bleeding is the complication requiring the least surgery in Turkey. (9)

3.1 Epidemiology

In previous years, perforation due to peptic ulcer was more common mainly in young and male patients, but today the average age is 40-60 years. (11) Similarly, in one study, the mean age of patients who developed perforation was 46.4 years, and more than 80% of patients were male. (9)

The frequency of perforation is relatively high in regions where H. pylori is common. Especially duodenal ulcer perforations; It is much more common in geographies where H. pylori is dominant in ulcer etiology. The circadian rhythm in gastric acid secretion is thought to play a role in the further development of perforation in the morning. (10)

Duodenal ulcer perforation is seen 7 times more frequently than gastric ulcer perforation when both genders are evaluated together. (12) However, the incidence of stomach ulcers is increasing day by day. (13) During this 45-year study, although duodenal ulcer perforation was more common in men, an increase in the proportion of women was observed in the following years. However, there was no proportional change in gender in the patient group with gastric ulcer perforation. (12)

3.2 Etiopathogenesis

The etiology of PUP includes the use of corticosteroids, chemotherapeutics and nonsteroidal anti-inflammatory drugs, H. pylori infection, smoking, prolonged fasting, metabolic and physiological stress, and the current diagnosis of peptic ulcer.

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prolonged fasting, metabolic and physiological stress, and the current diagnosis of peptic ulcer.

3.2.1 Helicobacter Pylori

There is strong evidence that the presence of H. pylori is involved in the etiopathogenesis. However, the presence of H. pylori negative peptic ulcer perforations should also be kept in mind. The prevalence of H. pylori in perforation in resources is variable (0-100%). It is estimated to be 50-80% by histopathological diagnostic methods. (14)

3.2.2 Drugs

Steroid use increases bleeding and perforation in PUD. (15) Bevacizumab (anti-VEGF) has also been reported to be associated with the development of peptic ulcer and perforation. (16) Studies on the relationship between NSAIDs and peptic ulcer have shown that these drugs have a strong role in the development of peptic ulcer and its complications. (17) There is a decrease in the prevalence of H. pylori, especially in developed countries. Along with this situation, the commen use of NSAID are shown as the main reason for the decrease in the frequency of duodenal ulcers and the increase in the frequency of gastric ulcers in the elderly population. (18)

3.2.3 Other Risk Factors

Prolonged fasting increases gastric acid secretion and the gastric mucosa becomes more vulnerable. For example, peptic ulcer perforation rates were found to be slightly higher in those who fasted more than 12 hours a day during Ramadan. (19)

It has been reported that PUP may develop after Roux-en-Y gastric bypass bariatric surgery. Smoking, cocaine or amphetamine use has also been reported to increase the risk of perforation. (20-22)

Gastrinoma (Zollinger-Ellison syndrome) also increases the risk of ulcer and perforation by causing hyperacidity in the stomach. Gastrinoma should be kept in mind in patients with multiple and resistant peptic ulcers. (23)

3.3 Clinical, Laboratory and Imaging

The most common symptom in PUP is severe and acute epigastric pain. Sepsis clinic develops as well as fever, tachycardia, hypotension. Various inflammatory parameters such as white blood cell (WBC), C-reactive protein are elevated. On physical examination, there is usually localized or generalized

peritonitis in the abdomen. (1) This condition, which is caused by stomach acid exposure, can cause 'acute abdomen' symptoms. However, typical peritonitis is seen in only 2/3 of the patients who develop perforation. This causes delay in diagnosis in some patients. (23)

In addition, the typical perforation clinic may not occur in people with impaired cognitive function, such as elderly and children, the obese, immunocompromised or patients using steroids. In these cases, further imaging and additional laboratory tests may be needed. (10)

Demonstration of 'free intra-abdominal air' in diagnostic radiological examination is largely indicative of perforation. (figüre-1)

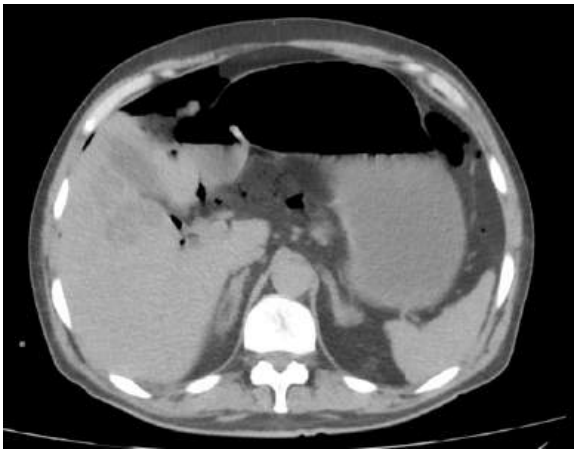

Figure 1. 'Free Intraabdominal Air' Image on Computed Tomography

Although abdominal X-ray is fast and accessible, its sensitivity is low. The use of ultrasonography, which has limited sensitivity in the diagnosis of perforation, has not become widespread. Computed tomography has become the basic imaging method because of its high sensitivity of 98% in imaging intra-abdominal free air and its superiority in determining the etiology of pneumoperitoneum.

3.4 Mortality and Morbidity

Although perforation is 6-7 times less common than bleeding in PUD, 37% of peptic ulcer-related deaths are perforation-related deaths. Mortality due to perforation is approximately 5 times that of bleeding. (8) Perforation is an important emergency that requires emergency surgery and causes 30% mortality and 50% morbidity in the short term. (26)

In critically ill patients, rapid resuscitation and imaging may need to be delayed. Patients presenting with diffuse peritonitis, with or without sepsis symptoms, may often require direct surgery. Because mortality rates in particular increase with every hour that surgery is delayed. (27,28)

High age, presence of comorbidities, development of septic shock, and delay in surgery consistently carry a higher risk of mortality in the literature. (29) But in line with all these data; Despite advances in rapid perioperative care, improved imaging modalities, and surgical techniques, the mortality rate due to PUP in Western countries remains almost stable. (30)

In clinical and laboratory data; The presence of tachycardia, acute renal failure and metabolic acidosis, low serum albumin level, high American Society of Anesthesiologists (ASA) score are other poor prognostic factors. (29)

3.5 Treatment

Among the basic principles of PUP treatment; management of the process by experienced surgeons, determination of mortality and morbidity risks, early diagnosis and then not delaying the surgical decision, initiation of empirical broad-spectrum antibiotics in the early period, close monitoring of vital signs and laboratory parameters. Antacid therapy such as proton pump inhibitors, appropriate fluid-electrolyte replacement, and management of postoperative complications should be implemented immediately in a specific protocol. (26,31)

3.5.1 Surgical Treatment

One of the most critical points in the treatment of patients with PUP is not to delay surgical treatment. (28) Conventional approach applied for many years in the treatment; laparotomy and closure of the perforation -with or without omentoplasty- with primary sutures. (32) A biopsy should also be taken.

The large perforation area (>2 cm) or the presence of densely inflamed tissues may not allow a safe primary repair. In addition, fistulas that occur after primary repair complicate a second repair. In these circumstances, the decision for surgical resection is more accurate. The presence of malignancy should always be kept in mind, especially in large gastric ulcer perforations or persistent fistulas. (33,34)

If surgical resection is decided, distal gastrectomy for gastric ulcer perforations (prepyloric) and total/subtotal gastrectomy for gastric perforations due to malignancy may be preferred. Diverting gastrojejunostomy can be applied

in pyloric duodenal ulcer perforations. T-drains can be used in the presence of persistent duodenal fistula. (35)

In recent years, laparoscopic repair has become increasingly common instead of classical laparotomy in perforated ulcer surgeries. (32) In current studies; It was reported that there was no significant difference between laparoscopic and open surgical treatments in terms of surgical mortality and complication rates. (36,37) Some centers even reported less postoperative pain and hospital stay compared to the open procedure after laparoscopic repair became widespread. However, these reports were studies involving younger patients with low ASA scores and small perforations (<1 cm). (38)

3.5.3 Nonoperative Follow-up

In some centers, successful nonoperative follow-up results were reported in selected patients. Spontaneous closure of the perforation was reported in some of the patients who developed PUP. In the nonoperative follow-up plan; nasogastric tube decompression, intravenous antibiotics, acid-suppressing drugs were included. The process was followed by contrast-enhanced imaging using water-soluble contrast material. (41) It should never be forgotten that non-operative follow-up should be applied carefully, considering the increasing mortality rate for each hour of delay to surgery. (27)

3.6 Postoperative Follow-up

3.6.1 Nasogastric Decompression and Oral Feeding

When the literature is examined, the application of nasogastric (NG) decompression is controversial. It is widely applied for 48 hours after surgery, almost all over the world. It is recommended to remove NG tube and start oral feeding after decompression applied to prevent tension of the surgical repair line. There is no evidence to support routine contrast-enhanced imaging before removing NG tube or initiating oral intake. (1,42) Early initiation of peroral nutrition, especially in patients with limited disease, is supported after removal of the NG tube. (43)

3.6.2 Antibiotherapy

In the successful treatment of PUP, early initiation of broad-spectrum intravenous antibiotics is important. After successful source control with surgical or non-surgical methods, empirical antibiotics are discontinued in 3 to

5 days. If sepsis persists, antibiotic therapy is continued in accordance with the culture results. (44)

3.6.3 Helicobacter Pylori Eradication and Use Of PPI

H. pylori eradication is a standard part of treatment. Appropriate and effective antibiotic combinations should be used. A recent Cochrane review showed that standard triple therapy (amoxicillin, clarithromycin, PPI) eradication rates increased with longer treatment duration (14 days versus 7/10 days). (45) Especially in duodenal ulcer perforations; H. pylori eradication therapy applied after surgery significantly reduces recurrence. (46) PPI use can be discontinued after eradicating H. pylori and limiting NSAIDs. (1)

3.6.4 Postoperative Endoscopic Follow-up

Gastric perforations are associated with approximately 13% of gastric cancer. Therefore, after surgery for gastric ulcer perforation, endoscopy is routinely recommended to rule out malignancy. (34) Endoscopic control is usually scheduled for 6-8 weeks after surgery. Endoscopic follow-up is not recommended for duodenal ulcers because of the very low risk of malignancy. However, it is difficult to distinguish between stomach and duodenum location in the juxtapyloric region and in highly infected tissues. In this case, endoscopy should be performed because the exact localization is uncertain. In addition, endoscopy should be planned even if no biopsy was performed during the surgery. (1,10)

3.6.5 Postoperative Complications

Complications such as sepsis, leakage, fistula, intra-abdominal abscess, surgical site infection, evisceration/eventration, incisional hernia, ileus and pneumonia may develop after surgery. (47) The complication rate has been reported as approximately 30%, and 20% of the patients who undergo surgery require secondary intervention due to complications. (32) The overall recurrence rate for PUP has been estimated at 12.2%. (4)

4. Conclusion

The most important step in peptic ulcer perforations is to prevent complications that may occur by applying early surgical intervention. (27) In cases other than very severe clinical conditions, providing early enteral

nutrition, pain control, early mobilization of the patient, removal of NG tube and abdominal drains as early as possible are the basic principles for the management of patients whose general condition improves. (1)

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CHAPTER XI

ACUTE CHOLECYSTITIS – CHOLANGITIS

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1. Introduction

Acute cholecystitis and cholangitis are the most common benign diseases of the gallbladder and bile ducts. Although the most common cause of both clinical entities is gallstones, they can also occur secondary to other metabolic diseases. In this section, we are going to discuss the clinical presentation, diagnosis, and treatment modalities of both diseases.

1.1. Gallbladder

Gallbladder is an part of the digestive system, which stores and concentrates bile. In response to a meal, the gallbladder contracts and releases the bile into the duodenum, allowing a plentiful amount of bile to flow through. Bile acids help digestion Of dietary lipids in the duodenum and facilitate their absorption (1). The gallbladder consists of three main parts: the neck, the body(corpus), and the fundus. In adults, it is approximately 7-10 cm in length and 3-4 cm in width, with a capacity of about 50 mL. The thickness of the gallbladder wall is approximately 2 mm and can vary in different degrees of distention and contraction. Increase of wall thickness is one of the most important indicators of acute inflammation.

1.2. Bile Ducts

Understanding the anatomy of the bile ducts is important for differentiating clinical conditions such as cholecystitis, cholangitis, cholelithiasis, and choledocholithiasis. It is also critical for successful hepatobiliary surgery (2). The basic structure that makes up the bile ducts is the hepatic canaliculi located within the liver. These structures unite to form intralobular canals, which

drain into interlobular canals. Interlobular canals then drain into two separate intrahepatic ducts in the right and left lobes of the liver. These intrahepatic ducts, the right (RHD) and left (LHD) hepatic ducts, exit the liver separately and merge in the extrahepatic area to form the common hepatic duct (CHD). The CHD then forms the common bile duct (CBD) by joining with the cystic duct of the gallbladder, which connects to the bile duct system. The CBD opens into the duodenum at the major duodenal papilla, where a sphincter structure called the Oddi sphincter regulates the passage of Bile and pancreatic enzymes into the intestinal lumen (3). Cadaver MRCP and intraoperative cholangiogram studies have revealed the presence of different configurations in the bile ducts. The most common variation can be described as a right posterior hepatic duct (RPHD) crossing horizontally and a right anterior hepatic duct (RAHD) oriented vertically, merging to form the right hepatic duct. Then, the RHD joins with the LHD to form the common hepatic duct (CHD). The second variant describes a three-way split configuration where RPHD, RAHD, and LHD converge at a single point to form the CHD. Rarely, an accessory canal that connects the liver directly to the gallbladder (Luschka canal) may be present. Configurations such as abnormal drainage of RPHD or direct drainage from RHD to the cystic duct are also present but less common (4).

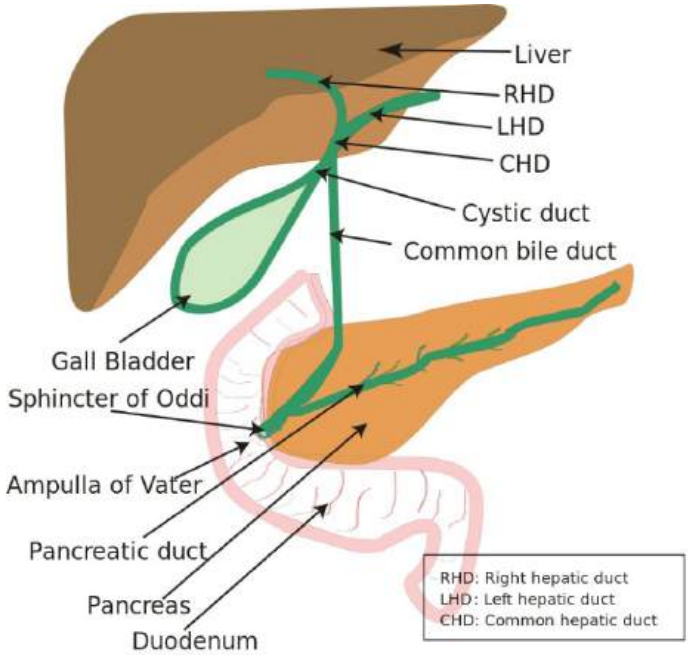

Figure 1. Anatomy of the biliary tract **3**

1.3. Gallstones

Gallstones are the leading cause of acute cholecystitis and cholangitis. Gallstones can be divided into two categories: pigment stones and cholesterol stones. In western countries, 75% of these stones are comprised of cholesterol. Bile secretion contains three distinct components: cholesterol, phospholipids, and bile acids (5).

There are 5 different mechanisms that can play a role in the pathogenesis of cholesterol gallstones. These are LITH gene defect, hypersecretion of hepatic cholesterol, gallbladder hypomotility, increased mucin secretion in the gallbladder lumen, and other intestinal factors. Risk factors for the development of cholesterol stones include increasing age, female gender, pregnancy, metabolic syndrome and insulin resistance, sedentary lifestyle, obesity, rapid weight loss, cholesterol-rich diet, gallbladder stasis, oral contraceptive use, and diabetes (6).

Pigment stones typically occurs from a consequence of bilirubin metabolism abnormalities in the hepatobiliary and intestinal systems. The most common risk factors include liver cirrhosis, hemolytic anemia, cystic fibrosis, Crohn's disease, extensive ileal resection, biliary infections, vitamin B12/folic acid deficiency, and aging. Black pigment stones arise from hypersecretion of bilirubin conjugates into bile and develop in the gallbladder. They contain pure calcium bilirubinate or polymer-like complexes and do not exhibit a regular crystal structure. Excessive increase in unconjugated bilirubin is a major pathogenic factor. Brown pigment stones can occur in the gallbladder and other parts of the biliary tree. These stones are primarily composed of calcium salts of unconjugated bilirubin, varying amounts of fatty acids, cholesterol, pigments, and mucin glycoproteins. They also contain small amounts of phospholipids, bile acids, and bacterial remnants. Biliary stasis is a predisposing factor for the development of brown pigment Stones (7).

2. Acute Cholecystitis

Acute cholecystitis can be defined as inflammation of the gallbladder. Approximately 200,000 people are diagnosed with acute cholecystitis every year in the United States. Systic duct obstruction secondary to gallstones is the most common cause of acute cholecystitis, accounting for 90-95% of cases. The remaining 5-10% of patients are characterized by inflammation of the gallbladder secondary to other metabolic diseases without gallstones, which is clinically defined as "acalculous cholecystitis" (8).

2.1. Clinical History and Physical Examination

During the diagnosis of acute cholecystitis, clinical history and physical examination findings play an important role. However, it should not be forgotten that these findings may not be sufficient for diagnosis, and other diagnostic methods may need to be used in combination. A good clinical history is crucial in the first stage. During the clinical examination, the patient's general condition and vital signs (blood pressure, pulse, respiratory rate, body temperature) are evaluated in the first instance. Subsequently, a detailed abdominal examination should be performed.

Clinical history of patient is crucial in the diagnostic process of acute cholecystitis. Patients with acute cholecystitis typically present with complaints such as right upper quadrant pain, nausea and vomiting, loss of appetite, abdominal bloating, weakness, and sweating. The pain usually starts after a meal or during the night and can be severe. The pain usually lasts for more than 6 hours and is most intense in the right subcostal area. Additionally, some patients may exhibit symptoms related to biliary obstruction such as jaundice, darkening of urine color, and lightening of stool color. During the patient's history, information regarding gallstones, previous biliary infections, family history, obesity, pregnancy, and other risk factors should be collected. Moreover, the patient should be asked about previous surgeries, chronic diseases, and medications used (9).

In medical examinations following a medical condition or trauma, evaluating the general condition of the patient is one of the basic steps. This evaluation includes factors such as the patient's consciousness, respiratory status, mobility, and gait. In addition, other factors such as age, gender, height, weight, chronic illness, or medication use are taken into consideration alongside the expected ranges for each. Measuring vital signs is also very important. Vital signs such as blood pressure, pulse rate, respiratory rate, and body temperature provide important information about the patient's general condition. Vital signs such as fever, tachycardia, and hypotension may indicate a more serious infection or sepsis (10). The most common findings during clinical examination for the diagnosis of acute cholecystitis include tenderness in the right upper quadrant (98%), Murphy's sign (75-85%), and fever (70%) (11). The clinical and physical examination findings are as follows:

• Abdominal pain: Typically, acute cholecystitis patients have right upper quadrant pain. This pain often appears in the form of a painful attack and worsens

after meals. The pain usually lasts for several hours to several days and often spreads to the back.

• Nausea and vomiting: Nausea and vomiting are frequently seen in acute cholecystitis patients.

• Fever: About half of the patients have a fever.

• Abdominal tenderness: The abdominal tissue may be tender when palpated and show increased tenderness upon palpation of the right upper quadrant.

• Jaundice: Jaundice rarely occurs as complications develop due to gallstones or obstruction.

• Subcutaneous emphysema: Acute cholecystitis can rarely result in a serious complication with gas gangrene or perforation, which is accompanied by subcutaneous emphysema.

• Murphy's sign: Murphy's sign is one of the most important physical examination findings in acute cholecystitis. It is the pain felt by the patient when deep breathing after pressing the area of the gallbladder located under the lower rib on the right side with a finger. This sign is considered a typical symptom of gallbladder inflammation and is an important indicator in the diagnosis. Additionally, a positive Murphy's sign accompanied by pain may also indicate the formation of gallstones.

2.2. Etiological Factors and Differential Diagnosis

Gallstones are the cause of more than 90% of cases of acute cholecystitis (12) Gallstones are defined as structures formed by the accumulation of cholesterol, calcium, pigment, and other substances inside the gallbladder. These stones can accumulate in the gallbladder and obstruct the cystic duct, which is the channel that opens into the biliary tree, leading to inflammation of the gallbladder, also known as cholecystitis. The risk of developing gallstones is dependent on various factors, both modifiable and non-modifiable, such as age, gender, family history, weight, and dietary factors (13).

Tumors causing biliary tract obstruction and primary sclerosing cholangitis, an inflammatory disease of the bile ducts, can also cause acute cholecystitis (14). In addition, infections of the gallbladder (acalculous cholecystitis), bacterial infections, parasites, viruses, or fungi can also play a role in the development of acute cholecystitis (12).

It has been shown that gallbladder infections are commonly caused by gram-negative bacteria (E. coli, Klebsiella, Proteus, Pseudomonas, Enterobacter), gram-positive bacteria (Streptococcus, Enterococcus, Staphylococcus), and

anaerobic bacteria (Bacteroides, Clostridium) (15). Escherichia coli is the most commonly responsible microorganism for gallbladder infections (16).

Some drugs can also cause inflammation in the gallbladder wall and lead to the development of acute cholecystitis. For example, drugs such as octreotide, tamoxifen, statins, estrogens, and propofol can cause inflammation in the gallbladder wall (12).

Lastly, the risk of acute cholecystitis may be higher in individuals with immune suppresive patients, obese patients, and those with comorbidities. Risk factors for acute cholecystitis may also include obesity, diabetes, pregnancy, coagulation disorders, excessive alcohol consumption, use of total parenteral nutrition (TPN), and immune deficiency (12). In addition, hospitalization, long-term antibiotic use, and the use of broad-spectrum antibiotics are also among the risk factors for acute cholecystitis (15). All of these factors can lead to an increase in the bacteria causing gallbladder infection and a higher risk of infection.

The diagnosis of acute cholecystitis can be made using classic symptoms (right upper quadrant pain, fever, blood tests indicating cholangitis or gallstone disease) and imaging techniques (ultrasound or computed tomography) (17). However, there are many diseases that can be confused in the differential diagnosis, especially those involving the upper gastrointestinal system, liver, and pancreas. Therefore, advanced imaging and laboratory tests may be required to increase diagnostic accuracy, especially in patients with atypical symptoms or comorbidities (18). Diagnosis of acute cholecystitis can be challenging as there are many diseases that can mimic its symptoms. These include:

Acute coronary syndrome: Although typically presenting as a pressure-like chest pain radiating to the left arm, acute coronary syndrome can rarely cause epigastric pain or pain in the right upper quadrant. Therefore, differential diagnosis is crucial.

- Peptic ulcer: Ulcer formation in the stomach or duodenum can cause similar symptoms.
- Pancreatitis: Due to its anatomical location, the pancreas can cause pain in the same area as the gallbladder. Therefore, pancreatitis should also be considered in the differential diagnosis.
- Extrahepatic biliary obstruction: Obstruction of the bile ducts, whether caused by a tumor or gallstones, can present with symptoms similar to those of gallbladder disease.
- Acute viral hepatitis: Viral infections of the liver can also cause similar symptoms.

2.3. Laboratory Findings

Laboratory tests of acute cholecystitis patients usually show an increase in markers of inflammation. These include tests such as white blood cell count (WBC), C-reactive protein (CRP), and erythrocyte sedimentation rate (ESR) (19). WBC, increases due to inflammation and this is an important indicator for the diagnosis of cholecystitis. CRP and ESR are used to measure the severity of inflammation (20). In some patients, blood urea nitrogen (BUN) and creatinine levels can also be elevated (21). This may indicate damage of renal function and is a factor that needs to be taken into account during the treatment process.

Elevations in serum AST (aspartate aminotransferase) and ALT (alanine aminotransferase) levels may be observed in acute cholecystitis. The cause of this elevation is hepatocyte damage and inflammation caused by biliary obstruction. The elevation in AST and ALT levels is not specific for the diagnosis of cholecystitis, but it can be helpful in differential diagnosis when observed in conjunction with acute cholecystitis (22). A study has shown that serum AST and ALT levels are elevated in patients with acute cholecystitis. In addition, a high AST/ALT ratio may be associated with severe inflammation and worse prognosis in cholecystitis (23). Obstruction of the bile duct can cause an increase in levels of alkaline phosphatase (ALP) and gamma-glutamyl transferase (GGT). These enzymes are primarily found in the liver and biliary tract, and their elevation can be a sign of obstruction of the bile flow. However, elevated ALP and GGT levels are not specific to cholecystitis and can also be seen in other liver and biliary tract diseases (24).

In acute cholecystitis, there may be an elevation in bilirubin levels. This elevation may be due to biliary obstruction. Biliary obstruction causes an increase in the circulation of bile pigments due to the obstruction of bile flow, leading to an increase in bilirubin levels (25). In acute cholecystitis, mild bilirubin elevation is commonly seen and usually returns to normal when treated. However, in cases of severe bile duct obstruction, bilirubin elevation can be significant and may require immediate treatment (26). However, an increase in bilirubin levels is not specific to the diagnosis of acute cholecystitis and may also be associated with different pathological conditions such as other biliary tract or liver diseases. Therefore, elevated bilirubin levels should be taken into consideration in differential diagnosis and evaluated in conjunction with other laboratory tests and imaging methods (27).

The results of these laboratory tests can help in making a diagnosis of cholecystitis and treatment plan. However, the results of these tests can be

misleading and are not sufficient on their own to make a diagnosis. Clinical evaluation and other imaging tests must be used in conjunction with them (28).

2.4. Imaging Methods

During the diagnosis process of acute cholecystitis, imaging methods in addition to laboratory tests and clinical evaluation are also used. These methods include ultrasonography (USG), computed tomography (CT), magnetic resonance imaging (MRI), and gallbladder scintigraphy.

The most commonly used imaging method for the diagnosis of acute cholecystitis is ultrasonography. Ultrasonography is a noninvasive, widely used, low-cost, and radiation-free imaging method. With ultrasonography, the size, wall thickness, content of the gallbladder, and complications such as stones or obstruction can be evaluated. Ultrasonography can also reveal pericholecystic fluid and other findings indicating inflammation of the gallbladder and surrounding tissues. Its sensitivity and specificity are high. In one study, ultrasonography showed a sensitivity of 92.9% for the diagnosis of acute cholecystitis and 97.4% for cholelithiasis (29).

In some cases, ultrasound results may be uncertain or suspicious. In such cases, a computed tomography (CT) scan may be useful for diagnosing cholecystitis. CT scan can also help evaluate biliary obstruction and other biliary pathologies in addition to cholecystitis. Although CT has a lower specificity than ultrasound, it can be considered as a useful alternative for diagnosing acute cholecystitis. CT can detect findings such as thickening of the gallbladder wall, accumulation of pericholecystic fluid, and pericholecystic hyperemia that indicate inflammatory changes in the gallbladder. Additionally, it can also detect biliary pathologies such as obstruction or dilatation in the bile ducts (30). However, its use may be limited, especially in children and young women, due to radiation exposure. In addition, some patients may experience side effects such as allergic reactions to contrast agents or contrast-induced nephrotoxicity (31). As a result, CT can be a useful method for the diagnosis of acute cholecystitis in patients with uncertain or suspicious ultrasound results. However, the patient's radiation exposure and potential side effects of contrast agents should be taken into consideration.

MR and MRCP can be used together with other imaging methods in the diagnosis of acute cholecystitis due to their high sensitivity and specificity (32). In particular, MR and MRCP are more effective in determining the presence of bile duct obstruction and detecting complications compared to other methods

in the diagnosis of acute cholecystitis (33). Additionally, MRCP can also be used to detect anomalies and strictures in the bile ducts (34). However, MR and MRCP also have disadvantages compared to other methods. They are expensive and time-consuming (35).

2.5. Treatment

The treatment approach for acute cholecystitis can be divided into two main categories: medical and surgical. Medical treatment is recommended for patients whose symptoms are mild or whose overall condition does not permit surgical intervention (12). The aim of medical treatment in acute cholecystitis patients is to control the infection and reduce inflammation. Treatment typically includes intravenous antibiotic therapy, fluid support, and pain control. The choice of antibiotic therapy is made with broad-spectrum antibiotics that provide effective coverage and are effective against biliary tract infections (36). According to the Tokyo Guidelines, in patients with acute cholecystitis, a broad-spectrum antibiotic effective against both anaerobic and aerobic bacteria is recommended. For this purpose, it may be appropriate to use a beta-lactam antibiotic such as ceftriaxone, cefotaxime, ampicillin/sulbactam, piperacillin/tazobactam, imipenem/cilastatin, or meropenem in combination with an aminoglycoside. The duration of antibiotic treatment may vary depending on the severity of symptoms and the origin of the infection. Antibiotic treatment is recommended for 4-7 days in hospitalized patients and may continue for 7-14 days in patients treated at home. However, if symptoms improve rapidly, the treatment duration may be shortened (12). Antibiotic treatment can be used prophylactically before and after surgical intervention in patients requiring surgery for acute cholecystitis. In addition, it can be used as an option to control symptoms in patients with a high surgical risk (37).

Medical treatment can help alleviate symptoms, control the infection, and reduce the need for surgical intervention. However, surgical intervention may be necessary in patients who do not respond to medical treatment or who have complications (38).

Based on the literature data, surgical treatment is generally recommended as the first option for appropriate patients with acute cholecystitis. Surgical options include laparoscopic cholecystectomy and open cholecystectomy. Laparoscopic cholecystectomy is the preferred method because it is a minimally invasive approach and is currently accepted as the standard treatment method (39). Although it is generally accepted that surgical intervention carries a higher

risk of complications in cholecystitis lasting longer than 72 hours from the onset of symptoms, there are debates regarding the timing of cholecystectomy. Studies recommending cholecystectomy regardless of the onset time of symptoms also exist in the literatüre (40). Evaluation and stabilization of patients before surgery are important. As part of surgical preparation, electrolyte balance should be corrected, appropriate medication should be given for severe pain and nausea, and fluid replacement should be performed. In addition, if patients have coagulation disorders or if it is a high-risk surgery, appropriate measures should be taken (41). During the postoperative period, it is necessary to control the patient's pain and monitor for postoperative complications. After laparoscopic cholecystectomy, the patient is usually discharged on the same day or the next day and only needs to rest for a few days to return to normal activities (42).

Percutan cholechystostomy is a minimally invasive method used in the treatment of acute cholecystitis to control the infection. It can be performed in patients who are not suitable for surgical intervention. The procedure is performed transcutaneously under ultrasound guidance, and involves draining purulent material from the gallbladder using a catheter. The importance of percutaneous cholecystostomy in the treatment of acute cholecystitis is increasing day by day. This method can quickly control a patient's symptoms and reduce the risk of infection (43). Percutaneous cholecystostomy can also help reduce pressure in the gallbladder and provide pain relief in addition to its role in controlling infection and draining purulent material from the gallbladder (44). Another important benefit of percutaneous drainage in the treatment of acute cholecystitis is that it is a suitable option for patients with high surgical risks (45). In elderly and comorbide patients, the risk of complications during surgical intervention may be higher. Therefore, percutaneous drainage can be used as an effective alternative to reduce the risk of surgical intervention in these patients.

3. Acute Cholangitis

Acute cholangitis is an infection and inflammation of the bile ducts that occurs due to the obstruction of the bile ducts (46). This can occur due to various reasons such as gallstones, tumors, or inflammation that cause obstruction of the bile ducts (47). Acute cholangitis can cause severe symptoms such as abdominal pain, fever, fatigue, loss of appetite, and even sepsis (48). Acute cholangitis can progress to a wide range of serious complications, including septic shock and mortality (49).

3.1. Clinical History and Physical Examination

Acute cholangitis clinical symptoms may vary depending on the severity and etiology of the disease. The most common symptoms of acute cholangitis include abdominal pain, fever, and fatigue (50). Abdominal pain is the most common symptom of acute cholangitis and is usually felt in the right upper quadrant (51). This pain is caused by increased pressure, especially due to gallstones or obstruction. The severity of pain can vary depending on the severity and localization of the infection. Fever is a common symptom of acute cholangitis. Although fever is important for immune modulation, it also carries the risk of dehydration. Fatigue, weakness, loss of appetite, and nausea are other common symptoms. In addition, symptoms such as jaundice, itching, abdominal distension, bloody vomiting, respiratory distress, and altered consciousness can also be observed (52). These symptoms depend on the severity of the infection, bile duct obstruction, liver damage, or the presence of systemic infection.

Physical examination is important in the diagnosis of acute cholangitis and in determining the severity of the disease (53).

During physical examination, the patient's general condition and vital signs (blood pressure, pulse, respiratory rate) are evaluated. In addition, an abdominal examination is performed, and findings such as abdominal pain, tenderness, guarding, rebound tenderness, distention, and the location of liver and spleen dullness are investigated (54). During liver palpation, liver tenderness can be detected in patients and liver size may increase due to bile duct obstruction, which can be reflected in physical examination as liver dullness being taken at a lower level than the intercostal space where it should be (55). In cholangitis secondary to obstruction of the bile ducts, the Murphy sign may be positive due to the presence of gallbladder hydrops (56). Inspection of the skin and sclera is important during physical examination as secondary hyperbilirubinemia can be seen due to biliary obstruction. In addition, symptoms such as pruritus, changes in urine color, and pale stool color can be observed in patients with hyperbilirubinemia (57).

3.2. Etiological Factors and Differential Diagnosis

The most common etiological factor is gallstones. Gallstones cause stasis in the biliary tree, leading to obstruction and increased risk of infection (58). Stenosis, tumors, and parasites that occur in the bile ducts are also among the causes of acute cholangitis (59). In addition, acute cholangitis can also occur

iatrogenically (after invasive procedures such as ERCP). As a result of this procedures, trauma, inflammation, and infection can be occur in the bile ducts. In this case, infection arises from pathogens entering the bile ducts during endoscopic intervention. Therefore, it is important to apply aseptic techniques and administer antibiotic prophylaxis regularly during procedures such as ERCP. Liver and biliary tract diseases such as acute pancreatitis, cholecystitis, and liver abscess can also lead to acute cholangitis (60). Bacterial infections are also an important factor in the etiology of acute cholangitis. Gram-negative bacteria, especially Escherichia coli and Klebsiella pneumoniae, are bacteria that can cause acute cholangitis. In addition, Clostridium difficile infections have also been associated with acute cholangitis. Parasitic infections are also among the rare causes of acute cholangitis. Especially in some regions such as Asia and South America, cases of acute cholangitis due to parasites such as Clonorchis sinensis, Opisthorchis viverrini, and Fasciola hepatica are frequently seen. Infection can occur as a result of these parasites living in the liver of animals and humans consuming the meat of these animals (61,62).

Diagnosis of acute cholangitis can be challenging as its symptoms can be similar to those of other diseases. Therefore, the following diseases should be considered in the differential diagnosis:

• Cholelithiasis - Acute Cholecystitis: Gallstones are small crystalline formations that form in the gallbladder. These formations can block the bile ducts and cause symptoms similar to acute cholangitis. Additionally, this duo can also be the cause of cholangitis. Gallstones that fall into the common bile duct and cause obstruction, and acute cholecystitis that presses on the common bile duct during inflammation can lead to the development of cholangitis (63). The elevation of bilirubin and stasis enzymes can be used as a basis for distinguishing these clinics from cholangitis. While an increase in these values is expected in cholangitis, elevation of these values is rare in cases of cholelithiasis and acute cholecystitis.

• Acute Pancreatitis: Pancreatitis is a condition that occurs as a result of inflammation of the pancreas. Factors such as gallstones, alcohol consumption, or high blood triglyceride levels can lead to the development of pancreatitis. Pancreatitis can show symptoms similar to acute cholangitis (64). Elevation of amylase and lipase levels is important in distinguishing cholangitis. This laboratory value is specific to pancreatitis. Their elevation is not expected in cholangitis.

• Liver abscess: Liver abscess is a cavity formed in the liver as a result of infection. It can be caused by bile duct infections, parasitic infections or trauma. Liver abscess may present symptoms similar to acute cholangitis (65).

• Periampullary tumors: Periampullary tumors are tumors located in the bile duct, pancreas, or ampulla of Vater. Symptoms include abdominal pain, weight loss, jaundice, and fatigue, and can resemble symptoms of acute cholangitis (66). When these diseases progress to the point of causing obstruction, they usually also cause hyperbilirubinemia. Although an elevation in tumor markers is more suggestive of periampullary tumors, the reliability of CA19.9, especially its elevation above a certain level of bilirubin, is questionable.

3.3. Laboratory Findings

Acute cholangitis is diagnosed through a combination of clinical symptoms, radiological and laboratory tests. Laboratory tests play an important role in the diagnosis and management of acute cholangitis (67,68).

• Complete Blood Count (CBC): CBC is a commonly used test for the diagnosis of acute cholangitis. In patients with acute cholangitis, there is an increase in WBC count and thrombocytopenia. However, this test alone is not sufficient to diagnose acute cholangitis and should be interpreted in conjunction with other tests. Thrombocytopenia is particularly seen in advanced disease-septic conditions.

• Liver function tests: Liver function tests (LFT) are used to evaluate hepatic disorders. In patients with acute cholangitis, levels of ALT and AST are elevated.

• Stasis enzymes and bilirubin levels: Since the main cause of cholangitis is expected to be obstruction in a part of the biliary tree, an increase in ALP and GGT levels is generally expected. In addition, bilirubin levels may also be high.

• C-reactive protein (CRP): CRP is an acute-phase reactant protein and an indicator of inflammation. In patients with acute cholangitis, CRP levels may be elevated.

• Procalcitonin: Procalcitonin is a marker of infection. In patients with acute cholangitis, procalcitonin levels may be elevated.

• Blood gas analysis: Blood gas analysis may reveal abnormalities such as hypoxemia, acidosis, and respiratory failure in severe cases of cholangitis.

• Coagulation tests: Coagulation factors and platelet count may be decreased in patients with acute cholangitis. Therefore, coagulation tests such

as prothrombin time (PT), activated partial thromboplastin time (aPTT), and platelet count should also be evaluated.

• Serological tests: Serological tests can be used to detect the infectious agents in patients with acute cholangitis.

3.4. Imaging Methods

Imaging has an important role in the diagnosis of acute cholecystitis, as with other surgical diseases. Ultrasonography (USG) is the most commonly used imaging method in the diagnosis and treatment process. Ultrasonography is a fast and reliable method for the diagnosis of acute cholecystitis. This method can be used to detect dilation in the gallbladder and bile ducts, gallstones, and obstructions in the gallbladder and bile ducts. It can also detect abscesses, hematomas, and cysts in the gallbladder and bile ducts. It is a readily available and cost-effective method (69). In addition, patients do not need to be exposed to toxic factors such as radiation and contrast agents. Therefore, ultrasonography is the preferred method for the diagnosis of acute cholangitis (70). However, there are also disadvantages to USG. In particular, evaluating the biliary tree with USG requires serious experience. Additionally, USG may be difficult to provide accurate results in patients with stenosis or obstruction of the bile ducts. In such cases, other imaging methods, especially computed tomography (CT), magnetic resonance cholangiopancreatography (MRCP), and endoscopic retrograde cholangiopancreatography (ERCP), may be necessary.

Another imaging method used for diagnostic purposes in acute cholangitis is computed tomography (CT). CT can be used especially in cases where ultrasound is insufficient or to confirm the diagnosis (71). CT can create a 3D image of the liver, gallbladder, and bile ducts. It can also be used to detect complications secondary to cholecystitis such as abscess formation. CT can also be used to determine the presence of other diseases (e.g., acute pancreatitis) in conjunction with acute cholecystitis. However, some studies in the literature have reported CT's sensitivity for acute cholecystitis to be approximately 80-90%. It should also be noted that CT is not as sensitive as ultrasound in acute cholecystitis and is often used in conjunction with ultrasound. Additionally, the use of CT is limited because it is an invasive method and can expose patients to radiation, making it impossible to use in special populations such as pregnant women and children (72).

MRCP is a non-invasive imaging method that provides a detailed anatomical view of the bile and pancreatic ducts. MRCP is particularly useful in evaluating obstructions and dilations in the bile ducts in patients with acute cholangitis.

Additionally, in cases of acute cholangitis caused by bile duct diseases such as gallstones, MRCP can help determine the location and size of the stone (72). MRCP also has some disadvantages, like other imaging techniques. MRCP is an expensive imaging method, and patients may experience MR anxiety, which can limit its applicability (73).

ERCP, although used as an imaging method in the past, is now mostly used as a therapeutic procedure, and will be discussed in the next section. While it is currently recognized as an imaging method in the literature, based on my personal clinical experience, I strongly recommend avoiding its use for imaging purposes only, as it can result in serious complications.

3.5. Treatment

The first and most important step in the treatment of acute cholangitis is to make a proper diagnosis. After a correct diagnosis of cholangitis, fluid resuscitation and antibiotic therapy are the most important interventions that should be empirically performed initially. In addition, symptomatic treatments such as analgesia should also be added. Procedures and surgery related to the cause of cholangitis should constitute the next step after resuscitation. This is because patients may have presented to the hospital with a poor clinical condition due to infection and inflammation, and may not be able to tolerate the procedures to be performed at the time of initial admission.

Fluid resuscitation is important for maintaining metabolic homeostasis. During a serious infection, accompanying symptoms such as hyperthermia and vomiting can lead to fluid and electrolyte loss. Additionally, oral intolerance can exacerbate this deficit. The goal of resuscitation is to replace lost fluids and electrolytes. The following factors should be taken into consideration during fluid resuscitation (12).

- Fluid resuscitation should be adjusted based on the patient's body weight and degree of fluid loss. Generally, 2-3 liters of fluid resuscitation are recommended within 24 hours.
- Fluids can be administered orally or intravenously (IV). IV fluid therapy can help patients compensate for fluid loss and electrolyte imbalances more quickly.
- During laboratory tests, electrolyte levels should be monitored and replaced as necessary. Especially potassium, sodium, and magnesium levels should be regularly checked.

• In patients at risk of hyponatremia (decrease in blood sodium levels), fluid therapy should be carefully planned. Avoidance of overly hypotonic and hypertonic solutions and preference for more balanced fluids would be appropriate.

Antibiotherapy is another important step in treatment. Tokyo Guidelines 2018 provides comprehensive recommendations for the use of antibiotics in the treatment of acute cholangitis. Proper administration of antibiotic therapy and avoidance of unnecessary use can increase the success rate of treatment and prevent serious problems such as antibiotic resistance. Antibiotic therapy may vary depending on the severity of the infection and the presence of bile duct stones. Most cases of acute cholangitis are caused by gram-negative bacteria (such as E. coli, Klebsiella pneumoniae) and gram-positive bacteria (such as Streptococcus spp., Enterococcus spp.). Therefore, broad-spectrum antibiotics are preferred. The first-choice antibiotics are third-generation cephalosporins (such as cefotaxime) and carbapenems (such as meropenem). In addition, an antibiotic that covers anaerobic bacteria, such as metronidazole, should be added. Antibiotic therapy is continued until the infection is under control, usually for at least 4-7 days. Treatment response should be monitored by symptoms and laboratory improvements, and the duration of treatment should be evaluated based on the patient's response to treatment (12).

ERCP can be considered as a treatment option in patients with acute cholangitis. It is highly effective in providing drainage in cases of obstruction caused by gallstones in the bile ducts. However, the use of ERCP in patients with acute cholangitis should be carefully evaluated due to the risk of serious complications such as infection, bile duct injury, hemorrhage, and pancreatitis during the procedure. Studies on the role of ERCP in the treatment of acute cholangitis have shown that ERCP alone or compared to conservative treatment does not provide significant benefit (74). However, ERCP can be life-saving in cases of obstruction due to stones, especially those associated with severe cholangitis or pancreatitis (75). In conclusion, ERCP has a limited role in the management of acute cholangitis and should be evaluated on a case-by-case basis. Before deciding to use ERCP in the treatment of acute cholangitis, the patient's clinical condition, biliary obstruction, and risk of complications should be considered.

Percutane Transhepatic Cholangiography (PTC) is generally a preferred treatment option in acute cholangitis when there is the presence of stones or obstruction in the bile ducts. PTC can be used especially in cases where ERCP

has failed. The main purpose of this method, which is performed percutaneously transhepatically, is to provide drainage of the bile ducts that dilate secondary to the obstruction. Although it is not often preferred, it can be a life-saving method for patients who cannot undergo surgery due to their general condition or in cases where drainage with ERCP has failed.

Many international guidelines state that surgical options should be considered in the treatment of acute cholangitis, especially in cases where there is no response to antibiotic therapy and minimal invasive methods such as ERCP and PTK have not been successful. While surgery is not the first-line treatment for acute cholangitis, the patient's condition and the severity of the infection should be carefully evaluated before implementing surgical options, and the most appropriate surgery should be performed at the most appropriate time. In special cases such as Mirizzi syndrome, cholecystectomy can provide definitive treatment for acute cholangitis. In addition, in cases of cholangitis secondary to choledocholithiasis, interval cholecystectomy after conservative treatment in the 4-6 week period is necessary to prevent another attack of cholangitis. It should be noted that inflammation caused by conditions such as acute cholecystitis and cholangitis in this area can make cholecystectomy more difficult. Especially during surgery, it is important to clearly identify anatomy to prevent potential injuries. In suspicious cases, evaluating the biliary tract with intraoperative cholangiography may be appropriate.

4. Result

As a result, acute cholecystitis and cholangitis are among the most common emergencies in general surgery. The most common cause of both clinical entities is gallstones, and a multidisciplinary approach should be adopted in management. Medical treatments, minimally invasive interventions and surgery are treatment options, and a patient-specific path must be followed for each patient.

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CHAPTER XII

ACUTE PANCREATITIS

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1. Introduction

Acute pancreatitis is characterized by acute inflammation of the pancreas. It is characterized by upper abdominal pain and elevated levels of pancreatic enzymes. Gallsotnes and chronic alcohol use are responsible for above %70 of acute pancreatitis (AP) cases. In most patients, inflammation is self-limiting and the disease progresses mildly. However, 20% of the cases have a severe clinical course. Mortality ranges from 5% to 30% owing to pancreatic necrosis and multi-organ failure in the severe form of the disease. (1,2) The main treatment in acute pancreatitis is intravenous fluid resuscitation. The evidence in the current literature has shown that the benefit of enteral nutrition. (3) The role of the open surgical approach in the necrotizing form of the disease is gradually decreasing, and minimally invasive methods have come to the fore.

This review discusses the etiology, pathogenesis, clinical findings, diagnosis, and treatment methods for acute pancreatitis.

2. Epidemiology

The incidence of AP is increasing worldwide owing to an increase in obesity and gallstone disease. (2) It is difficult to diagnose cases of acute pancreatitis with a mild course. Therefore, it is difficult to determine the true incidence. The annual incidence in the United States is 35 per 100,000 people. (4) Acute pancreatitis is the most frequently hospitalized gastrointestinal system disease in the United States, with over 270,000 cases annually. (5) The overall mortality rate of the disease is reported to be between 4-13%. (6,7) Mortality rate of acute necrotizing pancreatitis ranges from 9 to 30%. (3,6) The cause of

mortality during the first 14 days was multiple organ failure. Deaths occurring after the second week were due to complications of necrotizing pancreatitis. (8)

3. Etiology

After diagnosing acute pancreatitis, revealing the etiology provides an effective treatment opportunity and prevents disease recurrence.

Gallstones are the most common causes of acute pancreatitis. (15) It accounts for more than 40 percent of all cases. (10) However, AP occurs only in 3-8% of patients with symptomatic gallstone disease .It is more common in women aged 50-70 years. The risk of developing acute pancreatitis is high in the presence of small gallstones. (11)

Excessive alcohol consumption is the second most common cause of acute pancreatitis. The incidence is higher in young men aged 30-45 years. Acute pancreatitis develops in 5-10% of alcohol users. This situation is explained by alcohol abuse (>100 g/day for at least five years) and genetic predisposition. (12)

The third most common cause is **drug use**. It is the cause of 5% of all cases. (3). Drug-related pancreatitis has a better prognosis and a lower mortality rate. (13) The the strongest association drugs include azathioprine, sulfonamides, didanosine, thiazides, furosemide, acid, sulfasalazine and valproic acid. (3,13)

Hypertriglyceridemia accounts for 2-5% of acute pancreatitis cases. (3) A serum triglyceride concentration > 1000 mg/dL (11 mmol/L) causes acute pancreatitis attacks. (14) The lipoprotein metabolism disorders that cause hypertriglyceridemia can be primary (genetic) or secondary (acquired). Hypertriglyceridemia-induced pancreatitis is more common in type I, II and V lipoprotein metabolism disorders. (15)

Although **hypercalcemia** is a rare cause, in acute pancreatitis. It is most commonly observed in patients with hyperparathyroidism. (15) Intracellular increase in calcium levels lead to intraductal early trypsinogen activation. Consequently, tissue damage and ductal obstruction occurred. This leads to pancreatitis. (16)

Cases that develop as a result of **genetic factors** are often characterized by recurrent attacks. Genetic factors should be investigated in cases of acute pancreatitis in childhood. In most of these patients, the etiologic cause cannot be identified and the disease progresses to chronic pancreatitis. Mutations in the gene encoding cationic trypsinogen (PRSS1) lead to hereditary pancreatitis.

Furthermore, low-penetrance mutations in SPINK1 block the active binding site of trypsin. In the presence of this mutation, the risk of developing pancreatitis as a result of other etiologic factors is increased. (17)

In 2% of patients with AP, an attack of pancreatitis may be the clinical clue of a possible **pancreatic tumor**. Patients over 50 years of age diagnosed with idiopathic acute pancreatitis should be investigated for pancreatic or periampullary tumors using tomography after the attack has resolved. (19)

In approximately one-third of the patients, the etiology cannot be determined by anamnesis, routine laboratory tests, or ultrasound. Despite extensive investigations 10%-15% of patients are diagnosed with **idiopathic AP**. (20) The WSES guidelines recommend that two consecutive transabdominal ultrasounds followed by magnetic resonance cholangiopancreatography should be performed to exclude biliary etiology in idiopathic pancreatitis. (21) Table.1 summarizes the etiological factors of AP.

Table.1: Etiology factors for acute pancreatitis

CAUSE	MECHANISM	FREQUENCY	CLINIC
Gallstones	Obstructive	>40 %	Women Aged 50-70 Years
Alcohol	Toxins	25-35 %	Young Men Aged 30-45 Years
Drugs	İmmunologic reactions , Direct toxic effect etc.	5 %	Azathioprine Didanosine Valproic acid Mesalamine Diuretics, Azathioprine
Hypertriglyceridemia	Metabolic	2–5%	Fasting triglycerides > 11.3 mmol/L
Autoimmune	Autoimmune	<1%	Elevated IgG4, occurs in younger patients.
Viral infections	Toxins	<1%	mumps, coxsackie B, hepatitis B, herpes simplex
Pancreatic Anomalies	Pressure and obstruction of pancreatic duct	<1%	Pancreas divisum , Biliary cysts
Trauma	Trauma	<1%	History of trauma; most likely to affect midbody of pancreas
Peripancreatic tumour	Obstructive	<1%	Advanced age and etiology uncertain
ERCP	Trauma	Rare, 5–10% of patients undergoing ERCP	History of recent ERCP
Post-surgery	Pancreatic ischemia	Rare	Following biliary, upper gastrointestinal or cardiothoracic surgery

Abbreviations:; ERCP, endoscopic retrograde cholangiopancreatography; IgG4, immunoglobulin G4.

Source: Adapted from Sagar AJ et al. (3)

4. Classification

According to Atlanta consensus, acute can be divided into two main categories. (22)

• Interstitial edematous AP: It accounts for 80% of all cases. The clinical course is mild. The most important feature is the absence of necrosis in the tissue (1).

• Necrotizing acute AP: The most typical feature is the presence of **necrosis** in the parenchyma and/or peripancreatic tissue. Initially, necrosis may not be detected. It accounts for one-fifth of cases.

Acute pancreatitis is divided into three categories according to the severity of the disease. (22)

• Mild AP: Organ failure and complications are not observed. Mortality is very rare.

• Moderately severe AP: Organ failure and complications are transient. Clinical course resolves within 48 h.

• Severe AP: It is characterized by persistent organ failure.

5. Diagnosis

The diagnosis of AP is diagnosed based on history, laboratory studies, and imaging methods. According to the Atlanta consensus, at least two of the three criteria are required to diagnose acute pancreatitis. (22)

1. Abdominal pain compatible with acute pancreatitis (acute onset, usually radiating to the back, persistent, severe, epigastric pain)
2. Serum amylase/lipase level higher than 3 times the normal limit
3. Characteristic imaging findings compatible with inflammation findings of acute pancreatitis (contrast-enhanced computed tomography)

If abdominal pain and laboratory findings meet the diagnostic criteria, imaging is not required. (22)

5.1. Clinical History & Physical Examinaiton

Pain is the cardinal symptom. Most patients present with **acute-onset, persistent**, **severe**, and **epigastric pain.** In acute pancreatitis caused by gallstones, patients experience better pain localization. Pain relieved by leaning forward. Pain radiates to the back in 50% of the patients. (23) Pain worsens after food intake. Nausea and vomiting are often accompanied by pain. Dyspnea may occur secondary to pleural effusion in patients. (24) The patients are restless and agitated. Physical examination findings vary according to the disease severity. There is mild abdominal tenderness. Defense may accompany. Rebound is

rare. If a paralytic ileus develops secondary to intra-abdominal inflammation, abdominal distension is observed. Jaundice can be observed in the sclera and skin in biliary pancreatitis. Fever, tachypnea, hypoxemia, and hypotension indicate disease severity. Although not specific to acute pancreatitis, the Cullen's sign and Gray Turner's sign suggest necrotizing pancreatitis. In some patients, physical examination may be helpful in revealing the underlying etiology. Hepatomegaly in alcoholic pancreatitis and xanthomas in pancreatitis due to hypertriglyceridemia may be present.

5.2. Laboratory findings

Laboratory findings of acute pancreatitis correlate with pancreatic enzyme levels, inflammatory mediators, and hemoconcentration.

Amylase, a pancreatic digestive enzyme, increases within 6-12 hours. The half-life of amylase is approximately 10 hours. Therefore, serum levels return to normal values within 3-5 days after a pancreatitis attack. (25) A serum amylase level three times higher than normal is diagnostic for acute pancreatitis. Although patients have acute pancreatitis, amylase level may be normal. This does not exclude the diagnosis. **Serum lipase** level is a more reliable diagnostic marker than serum amylase level for the diagnosis of pancreatitis. (26) Lipase levels start to rise within 4-8 hours. It reached its peak level within 24 hours. The time to return to normal levels was between 1-2 weeks. It remained high in the blood for a much longer time than amylase did. (27) **C-reactive protein (CRP)** peaked 48 h after the onset of acute pancreatitis. CRP levels> 150 mg/L are favorable for severe acute pancreatitis. The level 48 hours after the onset of the disease is the best laboratory marker of the severity of AP. (25) Fluid loss to the third space in acute pancreatitis leads to hemoconcentration. Consequently, the **hematocrit** level increased. Leukocytosis was often observed. Increased urea and creatine levels, hyperglycemia/hypoglycemia, and hypocalcemia are other laboratory findings that may also be observed. Although trypsinogen activation peptide (TAP) and urine/serum trypsinogen-2 levels are useful during the early phase of AP, they are not routinely used. (28)

5.3. Imaging

Imaging methods are not essential for the diagnosis of AP; they are only helpful in the differential diagnosis. In the abdominal radiograph, a cutoff sign can be seen in the colon, suggestive of ileus and not specific for acute

pancreatitis. Calcified gallstones are rarely detected in biliary pancreatitis. Pleural effusion may be seen on a posteroanterior chest radiograph. Acute pancreatitis cannot be diagnosed using ultrasonography (USG). Possible gallstones that play a role in the etiology were detected by USG, and the diagnosis of acute cholecystitis was excluded. Therefore, USG should be performed for all patients. **Contrast-enhanced computed tomography** (CT) is the most appropriate imaging modality for evaluating the pancreatic parenchyma and presence of peripancreatic inflammation. CT was not required for all patients. CT is necessary when the diagnosis is suspected, in cases of clinical worsening, in cases of unresponsiveness to treatment, when complications are suspected, and to detect necrosis in patients with severe AP. Abdominal magnetic resonance imaging (MRI) has limited use in the diagnosis of AP because of its high cost and difficulty in accessing it. MRI is the best method for imaging the anatomy of the biliary tract and pancreatic duct. For this reason, it is very useful in patients whose etiology cannot be clarified and in recurrent attacks.

6. Prognostic Scoring System

Predicting the severity of APis challenging. Many scoring systems have been developed to predict the severity of AP. However, none have high accuracy in predicting the severity of AP. Scoring systems have low specificity and high false-positive rates. (29).

The Ranson criteria are the oldest scoring system designed to assess AP severity. Ranson criteria consists of 11 parameters. Five of the parameters are evaluated at the time of admission, and six of them are evaluated 48 hours after the application. Ranson score ≥3 defines severe pancreatitis. The mortality rate increases as the number of positive parameters increases. Mortality was 0–3 percent when the Ranson score was <3 and 40 percent when the score was ≥6. (1) Its negative predictive value was 90%. This method was used to exclude the possibility of severe pancreatitis. (15)

Computed Tomography Severity Index (CTSI) (Balthazar score) aims to predict the severity of the disease based on acute pancreatitis CT findings. Scoring was calculated on the basis of the presence of necrosis, inflammation, and fluid collection. A **Balthazar score** ≥ 6 is an indication of severe pancreatitis (cf. Table 2) (30)

Table 2: Computed Tomography Severity Index for acute pancreatitis*

FEATURE	POINTS
Grading of Pancreatic Inflammation	
Normal pancreas	0
Enlargement of pancreas	1
Intrinsic pancreatic alterations with peripancreatic fat inflammatory	2
changes	3
Single fluid collection	4
Two or more peripancreatic fluid collections:	
Pancreatic Necrosis	0
None	2
≤30%	4
30%–50%	6
>50%	

Note: CTSI score: ≥6 = severe disease.
*Adapted from: Balthazar EJ et al. (30)

Harmless acute pancreatitis score can be calculated at the time of first admission to the hospital. It consists of three parameters: absence of rebound tenderness or guarding, normal hematocrit, and normal serum creatinine level. It predicts mild acute pancreatitis with high diagnostic accuracy. This scoring system can prevent unnecessary intensive care hospitalizations. Quickly identify outpatients who can be treated. This reduces the cost of hospitalization. (32)

7. Local & Systemic Complications

According to the revised Atlanta criteria; Peripancratic fluid collections as local complications are defined as: (22)

- **Acute pancreatic fluid collection**: Seen within the first 4 weeks of onset; peripancreatic fluid without associated peripancreatic necrosis. Homogeneous fluid, confined by normal tissue planes, without intrapancreatic extension or defnable encapsulating wall
- **Acute necrotic collection**: Usually seen within the first four weeks of onset, fluid collection(s) with variable amounts of solid and liquid contents associated with pancreatic parenchymal or pancreatic necrosis. No encapsulating wall

- **Pancreatic pseudocyst**: Usually seen at least 4 weeks from onset; well-circumscribed, usually regular oval- or spherical-shaped homogeneous fuid collection without solid components but with a defined, completely encapsulating wall
- **Walled-off necrosis**: Usually seen at least 4 weeks from onset; heterogeneous, liquid, and solid density collection associated with necrosis, completely encapsulated by a well-defned wall

Each fluid collection described above may be sterile or infected. However, the risk of infection is low in collections without necrosis. The systemic and local complications that may develop in AP are listed Table 3.

Table 3: Complications of acute pancreatitis

Systemic	**Local**
More common in the first week	Usually develop after the first week
Cardiovascular	
Shock	
Arrhythmias	Peripancreatic fuid collection
Pulmonary	Pancreatic necrosis
ARDS	Pancreatic ascites
Renal failure	Pseudocyst
Haematological	Pseudoaneurysm
DIC	Portal/splenic vein thrombosis
Metabolic	Pleural effusion
Hypocalcaemia	Pancreatic abscess
Hyperglycaemia	
Hyperlipidaemia	
Gastrointestinal	
Ileus	
Neurological	
Visual disturbances	
Confusion, irritability	
Encephalopathy	
Miscellaneous	
Subcutaneous fat necrosis	
Arthralgia	

Source: Adapted from Satyajit Bhattacharya et al. (23)

8. Treatment

8.1. Initial Management

In patients with AP, the cardiovascular, respiratory, and renal systems should be evaluated for organ failure in the first stage. Therefore, disease severity should be determined. Patients with severe acute pancreatitis should be followed-up in the intensive care unit. In cases of hypotension, tachypnea, tachycardia, electrolyte disorders (hypopotassemia, hypernatremia, etc.), and anuria in patients followed up in the clinic, patients should be transferred to intensive care unit. Serum amylase and lipase levels are useful only in the diagnosis of pancreatitis. Daily measurements of amylase and lipase levels did not affect the course of treatment and were unnecessary during the follow-up of the disease. If local complications are suspected in patients with severe AP, arterial and portal venous phase CT should be performed 72–96 hours after the admission. Patients should be monitored for their vital signs. The urine output should be monitored hourly. Patients' blood electrolyte levels should be monitored daily.

8.1.1. Fluid Replacement

The initial treatment for acute pancreatitis is adequate fluid resuscitation. Systemic hypoperfusion is thought to be effective in the progression of interstitial edematous disease to necrotizing pancreatitis. (33) The superiority of **early fluid resuscitation** is generally accepted in literature. However, there is no consensus on the type, rate, volume, and duration of fluid use in resuscitation. In general, the aim of fluid therapy is to reduce hematocrit and BUN levels within the first 24 hours after hospitalization. The American College of Gastroenterology guidelines recommend **isotonic crystalloid solutions of 250–500 mL/h** in the first 12–24 hours. (34) Initially, all patients should be started on 5-10 ml/kg fluid therapy. Subsequently, the patient's fluid deficit should be evaluated. Minimal fluid requirement is approximately 6 L in 48 hours. Intensive fluid replacement should be done within the first 48 hours. In severe cases, 20 ml/kg/hour Ringer's lactate should be started, followed by an infusion of 150-300 cc/hour for 24 hours. The urine output of the patients should be at least 0.5 mL/kg/hr. (21,35,36) The current guidelines, except the AGA guidelines, recommend **ringer lactate** as the fluid for resuscitation. (33,35,36) Ringer's solution should be avoided if there is hypercalcemia. Excessive fluid may result in acute lung injury and abdominal compartment syndrome (ACS). (21)

8.1.2. Nutrition

In the past, patients with AP were followed-up with 'nil per os' until full recovery. Currently, this approach has left its place for early enteral nutrition. Oral and enteral nutrition have been shown to significantly reduce mortality and risk of infection in patients with acute pancreatitis. (33) Enteral nutrition prevents mucosal atrophy in the intestinal lumen, protects the intestinal integrity, and reduces bacterial translocation. All these factors are effective in preventing the development of SIRS-related organ failure in acute pancreatitis. (37) As a result of inflammation, a catabolic state occurs and metabolic energy requirements increase. If appropriate nutritional support is not provided, malnutrition may develop.

Early oral nutrition should be initiated for patients without nausea, vomiting, or abdominal distention. AGA recommends starting oral nutrition **within 24 h** of mild acute pancreatitis (35) If oral nutrition is not tolerated, enteral nutrition is the first choice and **enteral nutrition should be started within 72 hours**. (2) Contraindications (ileus, gastric outlet obstruction, mechanical intestinal obstruction, aspiration risk, abdominal compartment syndrome, etc.) should be investigated before initiating enteral feeding. The nasojejunal and nasogastric routes are not superior to each other in enteral nutrition for AP (3). If enteral nutrition is contraindicated, parenteral nutrition should be initiated. However, if the calorie needs of patients cannot be met with enteral nutrition, partial parenteral nutrition support can be provided. (21) The benefit of **immunonutrition** in the treatment of AP have not been demonstrated. (3) Probiotics have no place and should not be used. (37)

8.1.3. Anelgesia

Pain management is an important component of the initial management of AP. Sympathetic activation due to pain worsens clinical outcomes in AP. Absolute analgesia should be provided to all patients. In cases of severe pain, **opiates** are preferred, and there is no harm to using them. (3) The WSES guidelines recommend using **oxycodone** over morphine and fentanyl. (21) **Epidural analgesia** can be considered for patients who require long-term opiates.

8.1.4. Antibiotics

The use of **prophylactic antibiotics** in acute pancreatitis has no effect on treatment. Current guidelines **do not recommend** the use of prophylactic

antibiotics, including in patients with sterile pancreatic necrosis. (21,35,36) Although there are reports that the use of antibiotics for selective oral decontamination in patients with severe AP reduces gram-negative sepsis and mortality, there is no consensus on this issue. (3) If there is a clinical focus of extrapancreatic infection, if the blood culture is positive, and if there is evidence of infection in the necrotic pancreatic tissue, antibiotic therapy is initiated. (34)

8.1.5. Biliary Decopmression

Endoscopic retrograde cholangiopancreatography (ERCP) is not routinely recommended for treating acute biliary pancreatitis. However, ERCP reduces the mortality and morbidity of patients with acute cholangitis. Guidelines recommend performing ERCP in patients with acute biliary pancreatitis **and cholangitis/biliary obstructio**n. (21,35,36) It is recommended that ERCP be performed **within 24 hours**. (35) Endoscopic sphincterotomy with ERCP prevents recurrent attacks in patients diagnosed with acute biliary pancreatitis who are unable to undergo cholecystectomy. (3)

8.1.6. Cholecystectomy

Cholecystectomy is necessary to prevent recurrent acute biliary pancreatitis. (19) Therefore, index **laparoscopic cholecystectomy** is **recommended** for patients with **mild** biliary pancreatitis at the **same admission and before discharg**e. (21,36). In addition, cholecystectomy may not be performed because of the peripancreatic inflammation. In these patients, cholecystectomy should be performed after local and systemic complications have been resolved. Cholecystectomy should be added to the procedure if surgery is performed to treat local complications. (23)

8.2. Mangement of Complicaitons

8.2.1. Acute Peripancreatic Fuid Collection

Acute peripancreatic fluid collection (APFC) are fluid collections that occurs early in patients with AP. Necrosis was absent and the fluid content was sterile. It did not have a wall surrounded by a capsule. It occurs in approximately one-quarter of the patients with AP. (38) It was mostly spontaneously resorbed. Rarely, it can persist for four weeks and turn into a pseudocyst. Intervention was not required unless the patient was symptomatic. (23) If it causes compression symptoms, it is drained percutaneously under CT guidance. If percutaneous

drainage fails, transgastric drainage is performed under endoscopic ultrasound (EUS) guidance.

8.2.2. Pseudocyst

Pseudocysts usually occur after mild AP attacks of acute pancreatitis. It occurs in 5–15% of patients with peripancreatic fluid collection. (15) It is surrounded by a capsule consisting of granulation tissue with no epithelium on its wall. **At least four weeks** must pass after an acute pancreatitis attack for a pseudocyst to occur. (3) They are mostly soliters. Approximately half of the pseudocysts associated with the main pankeric duct. (23) Almost half of the patients progressed asymptomatically. Asymptomatic pseudocysts were not treated, and the patients were followed up. This is because two-thirds of the asymptomatic pseudocysts spontaneously resorb and regress. (15) Pseudocysts: if they are located in the tail of the pancreas, have no relationship with the main pancreatic duct, and are less than 4 cm in diameter, they often regress spontaneously. Indications for intervention in patients with asymptomatic pseudocysts are disruption of pancreatic duct integrity, failure to distinguish between pseudocysts and cystic neoplasms, and the risk of rupture and diaphragm compression. (39) The diameter of the pseudocyst diameter alone is not an indication for treatment. However, pseudocysts > 6 cm in diameter tend to be symptomatic and are unlikely to resolve or resorb spontaneously. (15) The development of symptoms such as persistent pain, weight loss, early satiety, and gastric outlet obstruction is an indication for intervention. (39) Pseudocysts can be treated in three ways. Percutaneous drainage with imaging is not recommended unless it is certain that the cyst is not connected to the pancreatic duct. (23) The possibility of cyst recurrence is high with percutaneous drainage. If there is a relationship between the pancreatic main duct and cyst, a pancreaticocutaneous fistula develops after percutaneous drainage. Percutaneous drainage should be preferred only in patients who develop sepsis secondary to a pseudocyst infection. (15) Surgical drainage of the gastrointestinal tract is superior to percutaneous drainage in terms of hospitalization and mortality. (3) If the anatomical location is suitable, endoscopic drainage is preferred. (2,3) If there is a relationship between the pseudocyst cyst and the pancreatic duct, endoscopic transpapillary drainage is preferred. (39) Cysts located in the pancreatic tail could not be drained via the transpapillary route. Therefore, with or without pseudocyst-pancreatic duct relationship, all of them should be drained via the transmural route. (40) Primarily, the endoscopic transmural approach

is preferred. Since transmural or transpapillary access using the endoscopic method is not possible for pseudocysts located in the uncinate process, surgical drainage is required.

The basis of the surgical approach is the drainage of the pseudocyst contents into the gastric or intestinal lumen. Surgical drainage is the only option when the endoscopic treatment fails. Depending on the anatomical location of the cyst, surgical cystogastrostomy, cystoduodenostomy, or Roux-en-Y cystojejunostomy was performed. Each technique can be performed either open or laparoscopically. The overall trend was an open surgical drainage with a large midline incision. (19) Pseudocysts located close to the stomach should be treated using **cystogastrostomy.** (15) **Cystoduodenostomy** should be performed for cysts located in the pancreatic head and adjacent to the duodenum**. Roux-en-Y cystojejunostomy** is recommended for the surgical treatment of pseudocysts that are not adjacent to the stomach and duodenum. (3,15) Possible complications of pancreatic pseudocysts (process outcomes infection abscess ,rupture into the gut, gastrointestinal bleeding, pressure effects obstructive jaundice from biliary compression ,bowel obstruction, erosion into a vessel haemorrhage into the cyst, haemoperitoneum etc.) shoould be surgically treated.(23)

8.2.3. Sterile and Infected Pancreatic Necrosis

An acute necrotic collection is typically an accumulation of peripancreatic fluid containing fluid and necrotic material, without a capsule. If it persists for more than 4 weeks, it turns into Walled-off necrosis surrounded by a fibrotic capsule. (23) While 40% of acute necrotic collections resolve spontaneously, 5% become infected. (3) An infected necrotic collection should be suspected in the presence of progressive clinical worsening, unresponsiveness to treatment, and a prolonged fever. (19) A definitive diagnosis is made by fine-needle aspiration from the collection; however, this is not routine. There was no indication for intervention in the presence of sterile acute necrotic collection. (41) If there is clinical worsening and unresponsiveness to treatment in sterile necrotizing pancreatitis, intervention should be delayed to allow the development of Walled-off. If the patient is stable, necrosectomy may be delayed at least four weeks. (23) If clinically infected necrosis is confirmed, antibiotic therapy should be initiated. Carbapenems are the first choice of treatment because of their high penetration into pancreatic tissue. (15)

The definitive treatments for infected pancreatic necrosis are necrosectomy and debridement. With necrosectomy, all pancreatic/peripancreatic infected

necrotic tissues should be excised and viable tissues should be preserved. It should be aimed to evacuate all exudative material in the retroperitoneal and peritoneal space. Mortality rates increased by up to 50% after open necrosectomy. (41) There are different recommendations in the guidelines regarding the optimal necrosectomy approach. Some patients may recover without the need for drainage. One-third of infected necrosis resolves with antibiotic therapy If anatomically possible, percutaneous or endoscopic drainage is recommended in the first stage. (2,21,36,41) Current guidelines recommend a "***minimally invasive step-up approach***" for the initial treatment. (36,41). In the **step-up** approach, drainage/lavage is performed primarily with percutaneous and endoscopic methods. Subsequently, debridement is performed endoscopically and laparoscopically. Finally, surgical necrosectomy (open or laparoscopic) is performed. In terms of mortality, the step-up approach was not superior to surgical necrosectomy. (42) However, few studies have reported fewer complications and organ failure associated with minimally invasive methods. (3) ***Surgical necrosectomy*** remains a valid treatment method in cases where invasive options are not suitable and when minimally invasive methods are unsuccessful. (21.41)

8.2.4. Venous Thrombosis

Patients with AP are at risk of venous thromboembolic disease. In particular, the **splanchnic venous** system is affected. The clinical presentation was faint. Thrombosis is usually diagnosed using computed tomography (CT) for acute pancreatitis. Significant **thrombocytosis** is in favor of thrombosis in the diagnosis. (23) Treatment is conservative. If systemic anticoagulation is initiated in the early period, it provides recanalization in the vein. However, there is insufficient evidence that systemic anticoagulants are safe for use in patients with AP. For this reason, it is not routinely recommended. (3) A prophylactic dose of **Low molecular weight heparin (LMWH) is** sufficient for isolated splenic vein thrombosis. LMWH is recommended at therapeutic dose for **six months** for portal vein thrombosis. (43)

8.2.5. Haemorrhage

In acute pancreatitis, bleeding may occur in the intestines, retroperitoneum, and peritoneal cavity. Hemorrhages are mostly observed in patients with necrotizing pancreatitis. The most common cause is **pseudoaneurysm rupture** secondary to inflammation of a large peripancreatic vessel. (23) Recurrent bleeding is common in cases of pseudoaneurysm rupture, with mortality rates

are very high. (44) In hemodynamically stable patients, CT angiography should be performed. Embolization was performed for the active arterial hemorrhage. Hemodynamically unstable patients require surgical intervention.

8.2.6. Abdominal Compartment Syndrome

Abdominal compartment syndrome (ACS), often develops within one week of symptom onset. ACS is defined as the **intra-abdominal pressure > 20 mm Hg** and accompanied by **organ failure.**(45) It is associated with an initial aggressive fluid resuscitation in acute pancreatitis. During treatment, fluid infusion was stopped, and diuretics were added. Decompression with a nasogastric tube should be performed.Muscle relaxants for relaxation of the abdominal muscles and rectal enemas are other important elements of conservative treatment. **Surgical decompression** is required if the conservative treatment fails. (3,21)

9. Conclusion

Cases of acute pancreatitis are mostly mild. Despite this, clinicians should accurately assess the severity of the AP at the first admission and be careful of the development of complications. A multidisciplinary treatment approach consisting of a surgeon, gastroenterologist, interventional radiologist, and intensive care specialist is important in the management of complicated cases. All treatment options should be planned together with a multidisciplinary team. This approach can improve the clinical outcomes of the disease.

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CHAPTER XIII

BILIARY COLIC AND OBSTRUCTIVE JAUNDICE

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1. Introduction

When gallstones are symptomatic, they present as biliary colic. Although the treatment and complications of this disease are seen as public health, it creates an economic and social burden. Gallstones are formed in the gallbladder and biliary tract due to irregularity in the levels of cholesterol or bile salts in the bile. Biliary colic usually refers to pain caused by a temporary self-resolving obstruction of the biliary tract. Cholecystitis or cholangitis may occur as a result of the cystic duct being blocked for a long time or causing obstruction in the biliary tract. In this case, the intensity and duration of the pain increases (1). There is wide variation worldwide regarding the known prevalence of gallstones, as most patients may be asymptomatic. Gallstone disease has high incidence rates in the United States and Europe, while it has a low incidence in Asia (2). Although biliary obstruction is rare, it can be considered as one of the diseases that cause morbidity and mortality in general. The most common cause of biliary obstruction is choledochal stones or strictures that cause extrahepatic bile duct obstruction. After this pathology, cholangitis, which causes obstruction in the bile ducts and fatal infection, needs to be treated urgently (3).

2. Pathophysiology and Risk Factors

Precipitation of excess cholesterol and decreased gallbladder motility play a role in the formation of gallstones as a result of the increase in cholesterol concentration in the bile (4). In addition, obesity, diabetes, diet low in fiber,

low physical activity, female gender are among the risk factors for gallstone formation. After meals, cholecystokinin is secreted from the duodenum, which causes contraction of the gallbladder. With the contraction of the gallbladder, stones can fall from the gallbladder into the cystic duct or the common duct. Although rare, stones that form in the common bile duct are called primary bile duct stones. These stones irritate the inner surface of the ducts, causing pain especially due to the contraction of the gallbladder and duct (5). Obstructive jaundice is commonly known as obstruction of the extrahepatic biliary tract. As a result of bile duct obstruction, serious complications such as liver failure, renal dysfunction, bleeding diathesis and infections may occur (6). Choledochal stones are the most common cause of biliary obstruction and account for 31% of all new cases. Malignant disease accounts for 14% of new cases of jaundice, and the most common cause is pancreatic head cancer (7). Other causes of biliary obstruction are strictures caused by iatrogenic damage to the bile ducts, parasitic cholangiopathy (Clonorchis sinensis, Ascaris lumbricoides), chronic pancreatitis, and primary sclerosing cholangitis (8).

3. Symptoms and Diagnosis

When stones in the gallbladder are symptomatic, they present with pain as biliary colic (BC). BC is defined as paroxysmal pain in the right upper quadrant and interscapular area. Patients with BC will usually have tenderness in the right upper quadrant on physical examination. Abdominal distention and acute abdominal findings are rare. Although the symptoms of BC are characteristic of gallstones, they can also occur in the absence of gallstones. In such cases, it is known that the symptoms are caused by millimetric stones, calculi (microlithiasis) or cystic duct anomaly. Rarely, BC is associated with high oddi sphincter pressure. In this case, it is seen that the pain is reduced by the endoscopic sphincterotomy procedure. The decision to perform cholecystectomy in patients with stone-free BC should be made by the hepatobiliary surgeon (9). Jaundice is not seen with obstruction of the cystic duct. Jaundice appears due to the direct rise of bilirubin with obstruction of the common bile duct. This finding suggests a more serious biliary obstruction and should raise suspicion of cholangitis rather than biliary colic. Biliary obstruction has various causes depending on the underlying etiology. It usually presents with acholic stools and dark urine. Patients with chronic biliary obstruction may have itching with chelestasis. In differential diagnosis; Hepatitis, cholangitis, mesenteric ischemia, pancreatitis, kidney stones or biliary dyskinesia should be considered.

3.1. Laboratory Findings

Laboratory tests to be checked in biliary colic and obstructive jaundice usually include hemogram, liver function tests, bilirubin values and bleeding profile. In cases that may develop due to infection such as acute cholecystitis or cholangitis, acute phase reactants and leukocyte values increase in addition to these values. In case of possible pancreatitis, amylase and lipase values may increase at least three times normal. In the case of biliary colic, these values are not usually elevated (Table 1). Laboratory abnormality is seen when stones in the gallbladder cause obstruction in the cystic duct or common bile duct (10).

3.2. Imaging

If biliary obstruction is suspected, upper abdominal ultrasonography (USG) is usually the first choice for examination. The sensitivity of USG is 90% for the detection of dilated bile ducts. In some cases, the common bile duct cannot be fully visualized. Therefore, the sensitivity of USG for common choledochal stones and stenosis is low. CT is not normally used to detect stones because many stones are not radiopaque and you want to avoid exposing the patient to radiation. MRCP (MR cholangiopancreatography) is used in the imaging of choledochal stones and in biliary tract pathologies)(Figure-1). If malignancy is suspected, CT can often be used in the diagnosis of pancreatic cancer, cholangiocarcinoma, and liver metastases. If the clinical and biochemical suspicion of biliary obstruction is high, the next step is usually MRCP or endoscopic ultrasonography (EUS) (11) (Figure-2). Before ERCP, if there is a clinical picture of cholangitis, and if there are signs of obstructive jaundice in laboratory values, MRCP is recommended (12).

4. Treatment and Management

Biliary colic treatment is primarily surgical. Non-surgical treatment of biliary colic includes supportive methods such as a low-fat diet, antiemetics, and pain control. However, the risk of recurrence of biliary colic is high because patients typically have many stones. It is estimated that in 2008, 14.2 million women and 6.3 million men in the USA had gallstone disease and 1.2 million cholecystectomies were performed each year (13). Antibiotics have no place in biliary colic since it is not an infectious etiology as in acute cholecystitis or cholangitis. Surgical intervention with laparoscopic cholecystectomy remains the gold standard. Extracorporeal lithotripsy may be considered in surgically high-

risk patients, but recurrence of cholecystitis is very high. Open cholecystectomy is a rare approach (14). The goal of the treatment of biliary obstruction is to provide adequate drainage of bile. Depending on the underlying etiology, treatment is curative. Endoscopic retrograde cholangiopancreatography (ERCP) is both diagnostic and therapeutic for common bile duct stones. Ampulla Vateri sphincterotomy can help remove stones after ERCP and prevent future stones from settling in the common bile duct.

4.1. Choledocholithiasis

Choledochal stones are primarily treated by sphincterotomy with ERCP and stone extraction using an endoscopic basket or balloon. If the stones are numerous or too large for extraction, temporary stenting may be used to maintain biliary drainage until definitive extraction(15).

4.2. Biliary Stenosis

In benign biliary strictures, sphincterotomy, balloon dilatation or stenting can be performed with ERCP. Plastic stents should be removed or replaced after 6-8 weeks. If endoscopic interventions are unsuccessful, biliary-enteric bypass should be considered. In malignant stenosis of the bile ducts, endoscopic drainage with permanent biliary stenting or biliary drainage from the wig is recommended. Palliative biliary-enteric bypass in unresectable tumors or endoscopic failure, resection of the tumor in resectable disease followed by biliary-enteric anastomosis(16).

4.3. Parasites

After drug treatment, sphincterotomy with ERCP and extraction using a basket are recommended.

4.4. Choledochal Cysts

Malignancy should be ruled out with ERCP or biopsy, and hepaticojejunostomy should be performed when necessary.

4.5. Neoplastic Tumors

Endoscopic bile duct stenting, palliation with chemoradiotherapy or radiotherapy is recommended in advanced disease. The Whipple procedure/ pylorus-sparing pancreaticoduodenectomy is recommended for tumors of the ampulla, duodenum, pancreas, and distal biliary tract. ERCP is an invasive

procedure with a risk of complications. Pancreatitis (3-10%), cholangitis (0.5-3%), bleeding (0.3-2%), perforation of the biliary tract and duodenum (0.08-0.6%) are common after ERCP (17).

5. Conclusions

For the effective management of patients with biliary colic and biliary obstruction; History, clinical examination, laboratory review, and diagnostic imaging should be used. In these cases, which are among the emergent diseases of surgery, the correct use of diagnostic imaging and treatment, starting with the least invasive and most cost-effective, results in effective treatment results and minimizes the risk of complications. Endoscopic and surgical treatment of biliary tract diseases usually requires a multidisciplinary approach. If the pathology is unclear or traditional forms of treatment fail, patients should be referred to specialized centres.

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Table 1: Laboratory Findings of Gallstones

Lab	Biliary Colic	Choledocholithiasis	Cholecystitis	Cholangitis
Wbc	Normal	Normal	Normal or slightly elevated	Normal or Slightly Slevated
Transaminases (AST, ALT)	Normal or Slightly Elevated	Normal or Slightly Elevated	Normal or Slightly Elevated	High
Konjuge Bilirubin	Normal	High	Normal or Slightly Elevated	High
Cholestases (ALP, GGT)	Normal or Slightly Elevated	High	Normal or Slightly Elevated	High

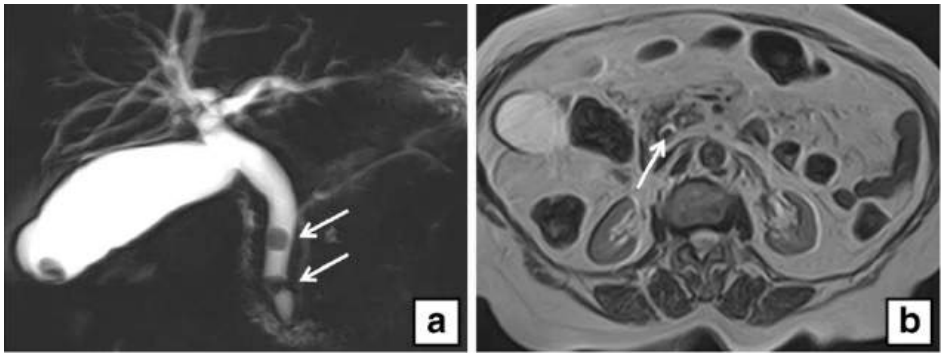

Figure 1: **a**-Coronal MRCP imaging dilated CBD defect in the common bile duct and gallbladder. **b**-Axial MRI defect in a dilated CBD

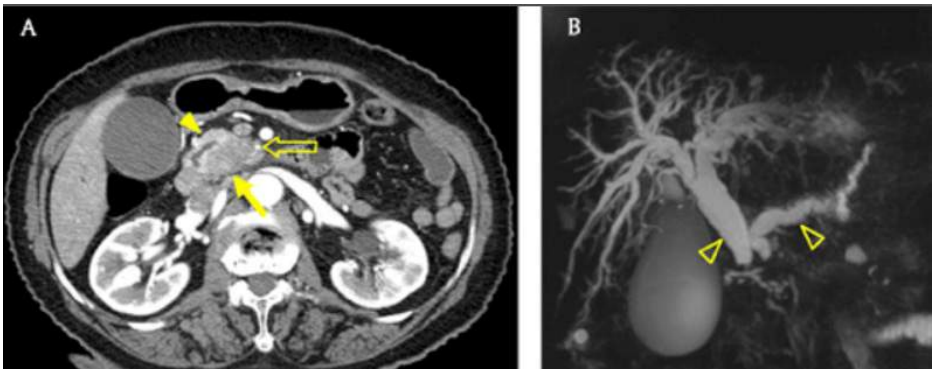

Figure 2: A-Axial MRI pancreatic head adenocarcinoma,
B-Coronal MRCP shows that dilated CBD and dilated pancreatic ducts, the so-called double duct sign.

CHAPTER XIV

MESENTERIC ISCHEMIA

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1. Introduction

Mesenteric ischemia occurs when blood flow to the intestines is interrupted. This interruption can be acute or chronic. This process results in decreased oxygen delivery, cellular damage, intestinal necrosis and thus death. Non-specific clinical findings and laboratory markers delay the diagnosis and treatment of the disease.

Mesenteric ischemia can be divided into two main titles as acute and chronic. Acute mesenteric ischemia is divided into 4 as mesenteric artery embolism, mesenteric artery thrombosis, mesenteric vein thrombosis, and non-occlusive mesenteric ischemia (NOMI).

2. Anatomy and Pathophysiology

The mesenteric arterial system is formed by the celiac artery (CA), superior mesenteric artery (SMA), and inferior mesenteric artery (IMA), originating from the abdominal aorta. The CA and its branches supply the stomach, liver, pancreas, and part of the duodenum. The distal SMA supplies the duodenum, jejunum, ileum, and the proximal and middle portions of the transverse colon. The IMA supplies the distal portion of the transverse colon, the descending colon, the sigmoid colon, and the upper rectum. SMA forms various collaterals with ÇA and IMA. The most important of these collaterals are Barrow arch and Buhler arch between SMA and CA, Drummond Marginal Artery and Riolan Arch

between SMA and IMA. Thanks to the these collaterals, intestinal oxygenation is provided in incomplete obstructions. (1,2) According to the formation processes of occlusion or stenosis in these main vessels, mesenteric ischemia is divided into the 4 main groups that we mentioned above.

The mesenteric circulation is influenced by gastrointestinal contents, hormones, and the nervous system. The hormonal effect is regulated by vasodilator (nictric oxide, glutathione, VIP) and vasoconstrictor (such as vasopressin) agents. Nervous stimulation is carried out by the visceral autonomic system. (3,4) Occlusion in the mesenteric vessels can be tolerated for a long time due to existing collaterals. The decrease in blood flow and oxygen amount reaching the mesenteric system due to occlusions of CA, SMA and IMA causes pain after meals, especially in the organs fed by these vessels. The decrease in blood flow causes anaerobic metabolism and acidosis. Cellular damage occurs as a result of released metabolites. If ischemia progresses, perforation of the intestinal wall due to full-thickness necrosis and a process leading to death occurs.

3. Etiology

Mesenteric ischemia can be classified as mesenteric arterial embolism (50%), non-occlusive mesenteric embolism (20-30%), mesenteric artery thrombosis (15-25%), mesenteric venous thrombosis (5%). (5)

3.1 Mesenteric Arterial Embolism

It is seen in patients with a history of cardiac disease such as myocardial infarction, congestive heart failure, heart valve disorders, arrhythmias, endocarditis, atrial fibrillation. While 15% are at the origin of the SMA, 85% are seen 3-10 cm distal from where the SMA emerges from the celiac trunk. (6,7)

Sudden onset of severe abdominal pain may be accompanied by various symptoms such as nausea and vomiting, bloody diarrhea. Most SMA embolisms occur parts of the small intestine, other than the proximal jejunum, where they are located distal to the celiac trunk. (8,9)

3.2 Mesenteric Arterial Thrombosis

It is seen in patients with a history of coronary artery disease and peripheral artery disease, in advanced age and traumatic situations. It progresses slowly from the background of atherosclerosis. Since it mostly occurs as a result of major visceral artery and collateral occlusion, it has a wide involvement from the duodenum to the transverse arm.

It is accompanied by symptoms such as increasing abdominal pain, abdominal pain after eating, fear of eating, weight loss, nausea/vomiting. (9,10)

3.3 Non-Obstructive Mesenteric Ischemia

It occurs due to reasons such as congestive heart failure, end-stage kidney disease, previous surgery, use of vasopressor medication. Hypovolemia is mesenteric vasoconstriction that occurs in response to decreased cardiac output, hypotension, or vasopressors. (4,11)

Symptoms such as abdominal pain, hypotension, diarrhea, and mental status changes appear. (4,9)

3.4 Mesenteric Venous Thrombosis

It may occur in patients with coagulation disorders (Factor V Leiden, prothrombin mutation, protein C/s deficiency, antiphospholipid syndrome, etc.), previous abdominal surgery, malignancy, intra-abdominal infection. It is usually asymptomatic. Symptoms such as insidious onset, suspicious abdominal pain, nausea-vomiting can be seen. (4,9,10,11)

4. Epidemiology

Mesenteric ischemia is a rare disease. It constitutes 0.1% of those who applied to the hospital. Mesenteric ischemia is common in patients with female gender, advanced age, and cardiovascular disease. The incidence is 34% in AT and AE, 13% in VT and 19% in NOMI. Mesenteric ischemia has mortality rates ranging from 50% to 70%. (8,9,13)

5. Clinic

Since the clinical applications of acute mesenteric ischemia are nonspecific, the physician's skeptical approach and experience are of great importance for early diagnosis and treatment.

Severe abdominal pain inconsistent with physical examination is the classic clinical finding of mesenteric ischemia. This is because ischemia progresses from the mucosa to the serosa. (14,9)

In arterial embolism, severe abdominal pain inconsistent with the examination, diarrhea, nausea-vomiting, and hematochezia as a result of mocosal ischemia may be seen. It should definitely be considered in elderly patients with a cardiac history. (8,9,10)

In ischemia due to thrombosis, increasing abdominal pain may be accompanied by distention, diarrhea, nausea-vomiting, and bleeding, as the process takes months to weeks. Pain after eating, fear of eating, and weight loss are the classic triades. Post-meal abdominal pain history may be confused with chronic ischemia. Due to the difficulty in making the diagnosis, the time spent in the hospital increases. (9,10,11)

The localization and severity of abdominal pain in NOMI varies in patients who are elderly, have a history of many additional diseases, have previous surgeries, and use digital and vasopressor drugs. (14,10)

6. Diagnosis

Early diagnosis in mesenteric ischemia is of great importance in preventing the risk of morbidity and mortality. Since the clinical complaints of the patients are not specific, the skeptical approach of the physician is very important. Past cardiac history with variable abdominal pain should suggest mesenteric ischemia. Intestinal obstruction, acute colitis, pancreatitis, cholecystitis, hollow organ perforation, malignancies should be considered in the differential diagnosis. (1,4,7,14)

There are no specific laboratory markers to determine the diagnosis in AMI. Increased leukocytosis and hemoconcentration may be seen. As a result of anaerobic metabolism, metabolic acidosis and an increase in serum lactate levels occur. In the late period, azotomy may occur with hyperkalemia. More specific markers such as I-FABP, α-GST, D-dimer, citrulline, L-lactate and D-lactate may be beneficial-as shown in recent studies. (20,10,4)

Pneumoperitoneum, pneumotasis and the presence of air in the portal vein on plain abdominal radiographs indicate intestinal necrosis and ischemia. The aim of plain abdominal radiographs is to exclude intestinal obstruction and perforation. If mesenteric ischemia is considered in the diagnosis, barium radiographs are contraindicated because of the risk of perforation. Barium can also compromise the diagnosis of tests such as CT and angiography. (9,10,20)

Doppler USG is a non-invasive method that can be used in diagnosis to evaluate vascular patency, as well as in follow-up and evaluation of recurrence. (4,14)

Multidetector computed tomography angiography (CTA) has now replaced angiography. BTA should be withdrawn immediately if mesenteric

disease is suspected. Despite the presence of acute kidney damage, BTA should be withdrawn by considering the profit-loss ratio. Radiologic findings such as intestinal dilatation, pneumotasis intestinalis, free intraperitoneal fluid, SMV thrombosis, portal vein thrombosis, and splenic vein thrombosis can be seen. (24,21)

Magnetic resonance angiography (MRA) can be used for functional assessment of arterial and venous vascular structures, intestinal insufficiency. However, its use in emergencies is limited.

Since mesenteric angiography takes a long time and is an invasive procedure, it has lost its importance in diagnosis over time. It can be used in the diagnosis of chronic mesenteric ischemia and NOMI. In addition, mesenteric angiography is also used in the treatment. (24,21)

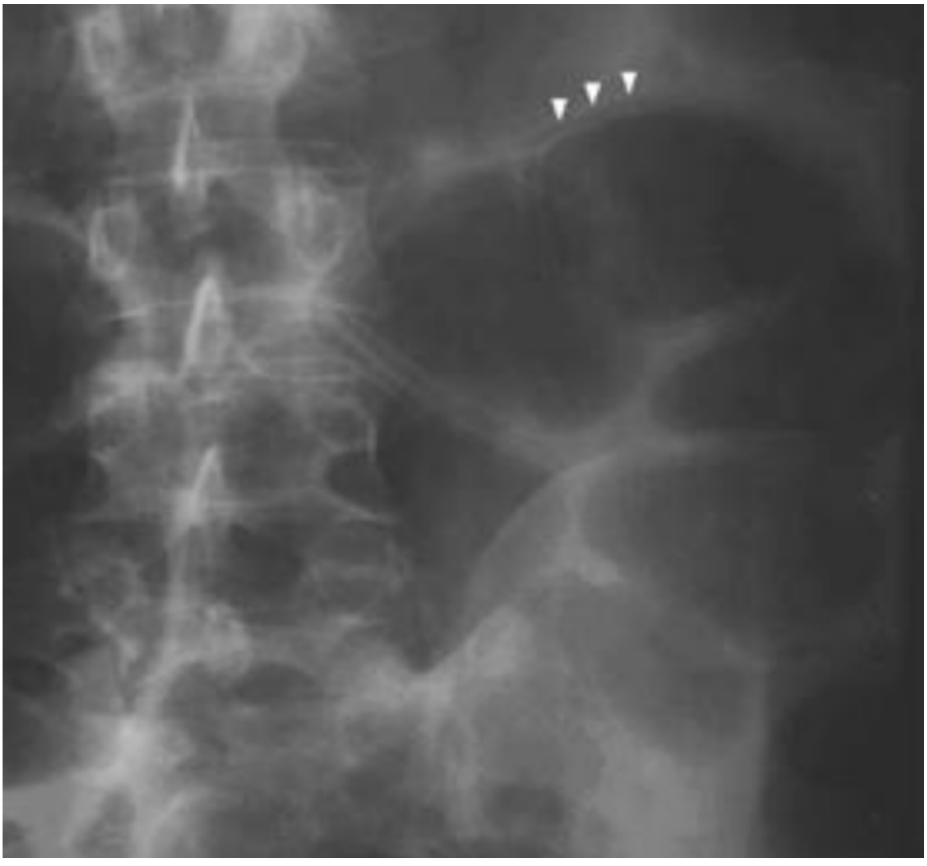

Image 1: Fingerprint View in Direct Graph

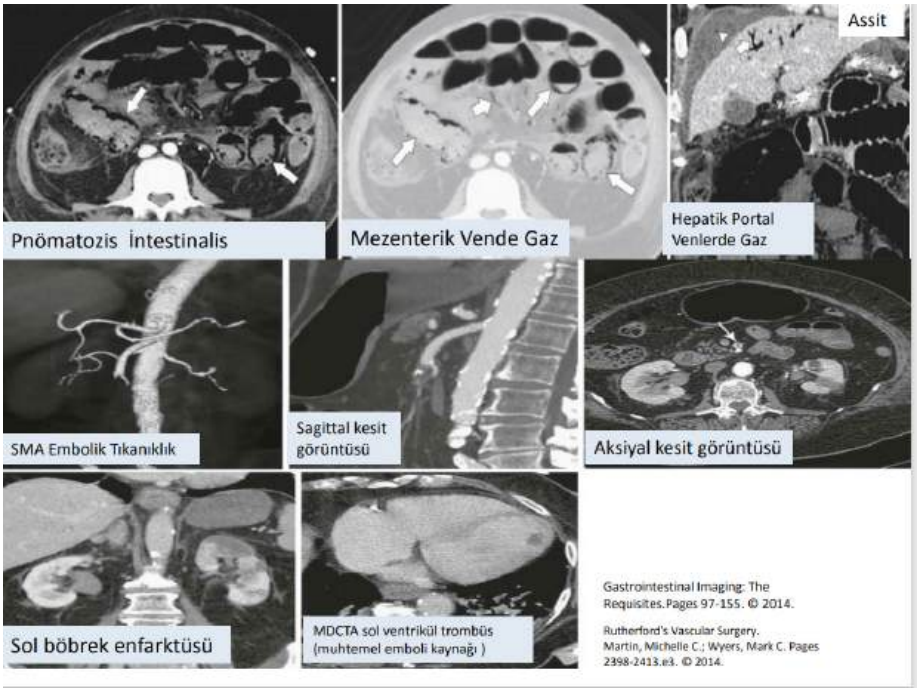

Image 2: CTA Imaging Sections of Mesenteric ischemia

7. Treatment

After diagnosis of AMI, treatment should be started immediately. Urinary catheter, nasogastric tube, central catheter and arterial catheterization should be provided to monitor the hemodynamics of the patient. Appropriate fluids and blood products should be given. Metabolic acidosis and hyperkalemia resulting from ischemia should be corrected. If there are no contraindications, anticoagulation treatment should be started. Broad-spectrum antibiotics should be given because of the high risk of bacterial contamination. (1,3,9)

7.1 Surgical Treatment

Surgery should not be delayed in patients with poor general condition with signs of peritoneal irritation. Revascularization (embolectomy, thrombectomy, endarterectomy or bypass) should be provided, followed by resection of the dead bowel portions. (22,23)

SMA is more involved in AE. Embolectomy with transverse arteriotomy should be performed after SMA is found in the exploration. In the event of

unsuccessful reperfusion, bypass may be necessary. After the revascularization, the condition of the intestines should be evaluated. Necrotic and lifeless intestines should be resected. The state of the intestines can be evaluated using intraoperative Doppler USG or intravenous fluorescein. Another method is the secondary look. Intestinal segments that cannot be evaluated 24-48 hours after revascularization are reevaluated. Necrotic segments are resected. (22,23)

Thrombotic mesenteric ischemia mostly involves an atherosclerotic process in which the SMA and CA arteries are involved. Revascularization is achieved by vascular bypass. Saphenous vein is preferred as graft. (9,14,22)

Transaortic endarterectomy and bypass can be used to prevent intestinal infarction in chronic mesenteric ischemia. The graft used can be drawn from the supraceliac aorta or the infrarenal aorta. (22,23)

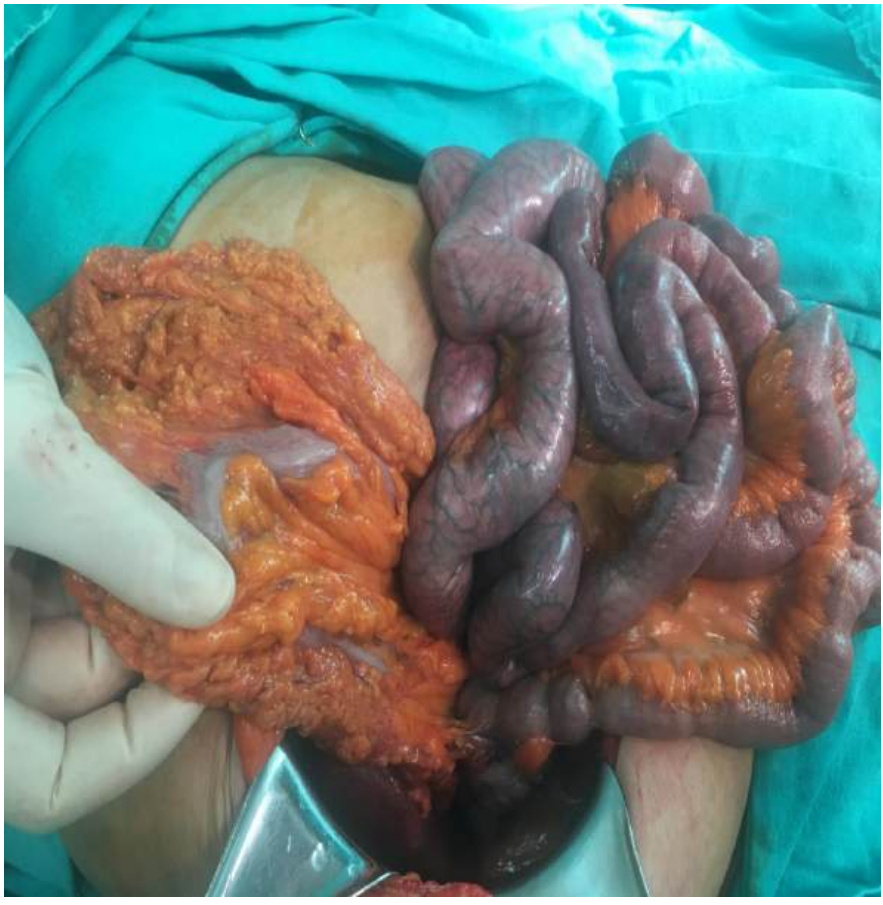

Figure 1: Necrotic Small Intestine Surgery Image

7.2 Endovascular Treatment

Recent advances in interventional radiology pave the way for endovascular procedures in the treatment of mesenteric ischemia. It is preferred in patients with early diagnosis, no intestinal necrosis, high comorbidities, and high-risk surgery. In endovascular procedures, patients are treated using thrombolysis, embolectomy, balloon dilation and stents. (9) Successful results can be achieved with an experienced team in endovascular treatment of AMI, NOMI and chronic mesenteric ischemia, but care should be taken in terms of complications of treatment. Endovascular treatment is a more non-invasive method than surgical procedure and should be the first preferred method if possible.

Balloon dilatation or stenting is preferred in chronic mesenteric ischemia. Because of the risk of developing acute ischemia, revascularization should be performed in patients who are asymptomatic or have minimal symptoms (4,14,24)

In acute mesenteric ischemia, catheter-related thrombolytic therapy is the most appropriate treatment. During diagnostic angiography, thrombolytic (urokinase, recombinate tissue plasminogen activators) treatments can be administered into the thrombus. If thrombolytic therapy is performed within the first 12 hours from the moment of symptoms, the success of the treatment increases. If thrombolytic therapy is successful, stenting and angioplasty can be performed electively. While the thrombolytic therapy is continuing, the patient's clinic should be monitored closely and the success of the treatment should be checked with serial angiographies. Surgery may be required when treatment fails and ischemia is suspected. (9,10,18)

Vasodilator (papaverine, tolazoline) drugs are used in the primary treatment of NOMI. During the treatment process, vasoconstrictor drugs are discontinued and heparin is administered to prevent re-thrombosis. After the patient is monitored, clinical and hemodynamics are observed closely. If the patient's clinic deteriorates and acute abdomen develops, the patient may need emergency surgery. Papaverine infusion should be continued intraoperatively and postoperatively. (25,26)

Life-threatening complications in endovascular treatment are very rare. Dissection, thrombosis, hematoma and infection may develop. During and after the treatment, patients should be closely monitored and monitored. One should be vigilant in terms of response to treatment and complications. (27,28)

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CHAPTER XV

ACUTE APPENDICITIS

GÜRKAN DEGIRMENCIOGLU

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1. Introduction

Appendicitis is among the most common diseases that surgeons face in practice. Acute appendicitis is the cause of approximately 25% of hospital admissions because of acute abdomen. The risk of developing appendicitis is highest in the second and third decades (8.6% in men and 6.7% in women). Approximately 250.000 appendectomies are performed annually in the USA (1). Because low-fiber and high-fat diets are more common in Western countries, it is considered that appendicitis is more common than in underdeveloped countries (2). The appendix is among the intestinal lymphoid tissue elements, playing roles in the secretion of immunoglobulin, especially IgA.

2. Anatomy and Embryology

The appendix is a midgut organ and begins to develop as a protrusion at the end of the cecum in the eight-week embryo, and lags because its development is slower than the cecum. Although it is usually between 6-10 cm, it can be longer or shorter. The base of the appendix vermiformis is usually located at the base of the cecum at the junction of the tapeworms. The tip of the appendix is most commonly located retrocecal, pelvic subcecal, preileal, and less commonly retroperitoneal or intracecal. The surgeon needs to know the localities because clinical findings and physical examination findings may vary according to the anatomical locations. For example, patients with retroperitoneal appendicitis may present with back pain or flank pain, and patients with pelvic location have suprapubic pain.

The blood supply to the appendix is provided by the appendicular artery, which passes through the mesoappendix over the ileocolic artery, which is one of the major branches of the superior mesenteric artery. Lymphatics are also found in the mesoappendix and drain into the ileocecal lymph nodes (3).

3. Etiopathogenesis

Luminal obstruction is the first step in the development of acute appendicitis. Although the cause of luminal obstruction is frequently fecaliths (35%), lymphoid hyperplasia, fruit seeds, neoplasms or intestinal parasites may also be the cause of obstruction. The pressure gradually increases with the gas production of the bacteria in the lumen and the secretion of mucus in the lumen of the appendix, which is distal to the obstruction. This increased pressure gradually causes distension. This increased pressure is transmitted over the sympathetic nerve fibers and causes visceral pain. This pain cannot be localized in the periumbilical region. As well as pain, symptoms related to sympathetic activation such as loss of appetite, nausea, vomiting, and sweating are accompanied. If the obstruction continues, inflammation also affects the entire appendix wall, and when it reaches the serosa, symptoms related to parietal peritoneal irritation occur, in other words, severe pain that is localized to the right lower quadrant occurs. Ischemia occurs because the increased pressure will disrupt the arterial circulation, and perforations occur after the entire layer has ischemia. If the obstruction disappears without ischemia during the inflammation process, the event regresses (2).

The flora of the appendix is similar to that of the cecum. Gram-negative bacteria are usually among these in appendicitis-associated infections with commonly detected being Escherichia coli, Bacteroides fragilis, and enterococci.

4. Presentation

4.1. Anamnesis-Symptoms

The first complaint in patients who present with acute appendicitis is usually pain. At first, the pain is non-localized around the umbilicus and turns into somatic pain within 4-8 hours as the inflammation reaches the parietal peritoneum. Although this pain is usually localized to the right lower quadrant, sometimes, it can also be felt in different places depending on the localization of the appendix. Although back or flank pain is prominent in retrocecal appendicitis, there may be suprapubic pain and pain during urination in appendices that

extend to the pelvis. An important symptom seen after the pain and requires the clinician to check the diagnosis again if not present is anorexia. Anorexia may be accompanied by nausea, and sometimes by vomiting. Although some patients have constipation, they may describe a feeling of tenesmus (4).

4.2. Physical Examination

In appendicitis, clinical findings vary according to the stage of the disease. If perforation and peritonitis are not present, the general condition is good, there is subfebrile fever. The absence of fever does not rule out the diagnosis of appendicitis (4).

A localized pain in the right lower quadrant, named after Mc Burney, is typical on physical examination. The Mc Burney point is the 1/3 lateral point of the line on the umbilicus and the anterior superior line of the right spina iliaca (Figure 1). Pain and tenderness are often associated with a rebound, which is the indication of localized peritonitis as the pain occurring as a result of lightly pressing the painful area with palpation and pulling the hand suddenly. An indirect rebound is the feeling of pain in the right lower quadrant if the hand is withdrawn after palpation of another region of the abdomen. Rebound and indirect rebound are signs of parietal peritoneal irritation.

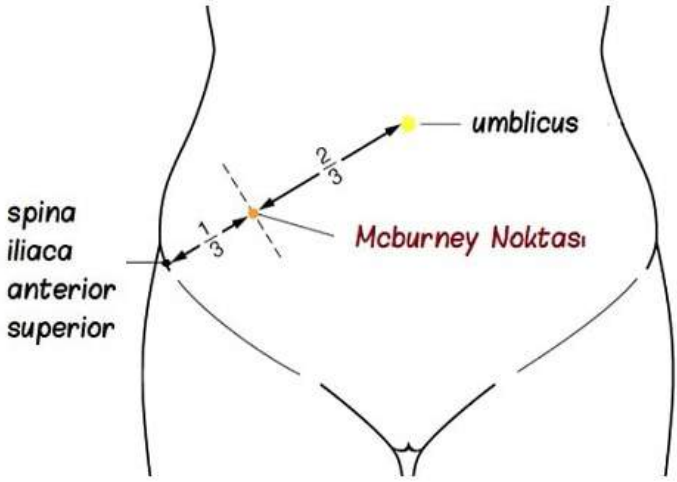


Figure 1. McBurney point

Although not used very often in practice to help the diagnosis of appendicitis, some findings were described in the past. One of these is the Rovsing Sign and pain in the right lower quadrant during pressure palpation

to the left lower quadrant. The Psoas Symptom is the pain associated with inflammation around the stretched iliopsoas muscle when the right thigh is slowly extended and the patient is placed on the left side. The Obturator Sign is the pain when the patient's right thigh is flexed and internally rotated and is indicative of inflammation around the obturator internus muscle.

A mass can be palpated in the right lower quadrant, which may be a sign of plastron appendicitis, or neoplasm may be palpated in appendicitis because of a cecum mass. Although rectal examination is usually normal, tenderness or a palpable mass can be detected in the presence of a pelvic abscess, especially in pelvic appendicitis.

4.3. Laboratory Findings

Laboratory findings are used to support the clinical manifestation rather than to make the diagnosis or exclude it. There is a manifestation of leukocytosis with neutrophil dominance. Leukocytosis may not be seen in 10% of cases. Perforation or abscess must be considered in the presence of leukocytosis over 18.000/mm^3. C-Reactive Protein is not specific to make or exclude the diagnosis but is employed to support the diagnosis. Although urine tests are usually normal, mild leukocytes can be detected in reactions around the ureter or bladder (5).

4.4. Imaging

Standing direct abdominal X-ray is routinely used in all patients evaluated with the preliminary diagnosis of acute abdomen. The only finding that can be specific in the diagnosis of acute appendicitis is the presence of calcified fecaliths in the right lower quadrant. Calcified fecaliths can be seen only in 5% of cases (6). Free air under the diaphragm may rarely be seen in patients with perforated appendicitis.

Ultrasonography is among the most commonly used reliable methods for the diagnosis of acute appendicitis. Although its sensitivity varies according to the experience of the physician, it has a specificity of 85-97%. Inflamed appendicitis typically appears in enlarged, immobile, and incompressible forms (Figure 2 A). It is important in the diagnosis, especially in pregnant and pediatric patients because there is no radion.

Computed Tomography is a very effective and accurate test in the diagnosis of appendicitis. Its sensitivity is between 90-100%, and the specificity is between 91-99%. CT with IV contrast material is sufficient, oral contrast is not recommended (7-8). On CT, signs of inflammation in the thickened

appendix and surrounding mesentery are typical. The appendix is usually larger than 7 mm in diameter and has inflamed walls and mural enhancement with a "target sign" (Figure 2B). The presence of air and free fluid around the appendix suggests perforation. Although CT is not recommended in patients with severe appendicitis based on anamnesis, physical examination, and laboratory findings, it reduces the rate of negative laparotomy in cases where there is doubt.

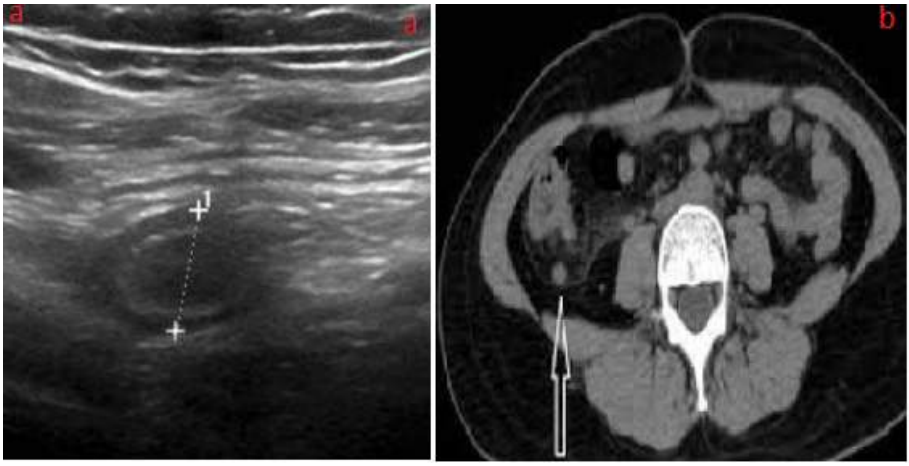


Figure 2. A- Distension and wall thickening in appendicitis on USI.
B- Appendicitis in sagittal imaging on CT.

Non-contrast MRI can be used in cases where the diagnosis of appendicitis cannot be made definitively in pregnant women. The accuracy of the diagnosis of appendicitis is quite high in MRI and its use is limited because of its high cost and difficulty of access.

5. Acute Appendicitis Treatment

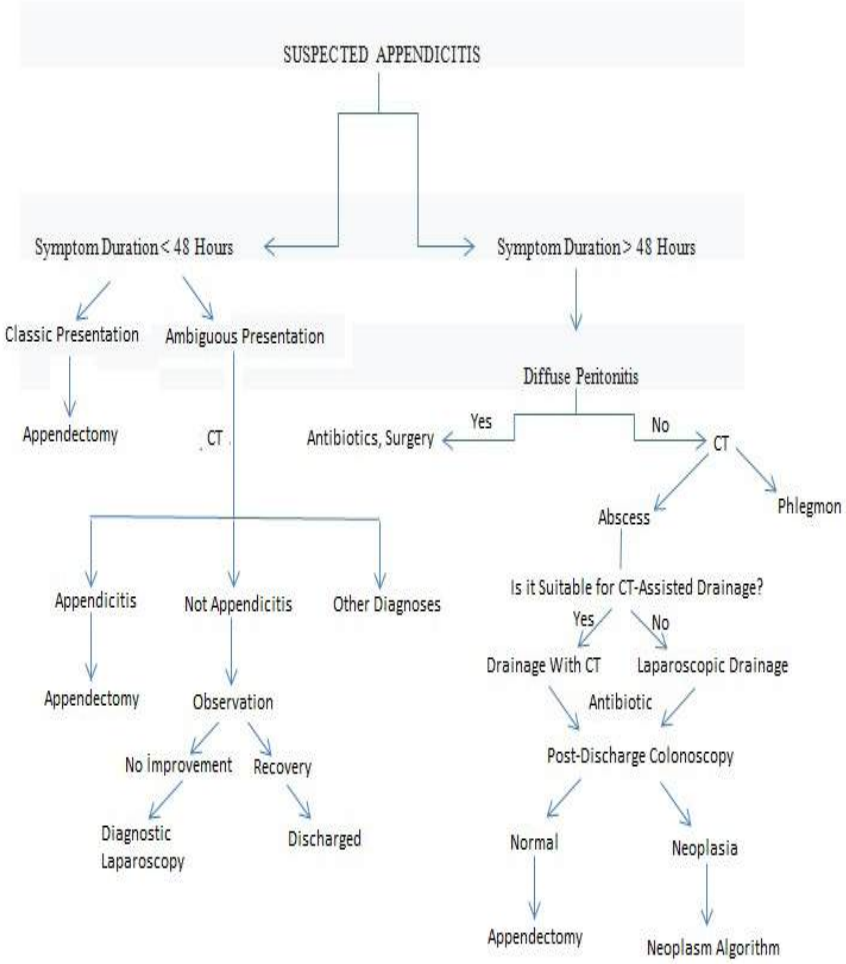
5.1. Acute Uncomplicated Appendicitis

The treatment of acute uncomplicated appendicitis is appendectomy. After the diagnosis of acute appendicitis is made, the period must not be delayed. Patients must be given as much fluid therapy as needed along with broad-spectrum antibiotics for gram-negative and anaerobes (8). Although non-surgical treatment methods are used in uncomplicated acute appendicitis, the patient must be informed about the risk of treatment failure and that appendicitis may recur. Although it is reported in previous studies that the complications of

medical treatment are less in uncomplicated acute appendicitis, it is seen that the treatment failure rate is quite low when compared to appendectomy (9).

Although appendectomy is routinely performed as an open appendectomy, it is replaced by laparoscopic appendectomy in many centers in our present day. Laparoscopic appendectomy provides information on the case, especially in the intraoperative differential diagnosis, and it is completed with therapeutic surgery when the diagnosis of appendicitis is confirmed. The patient must lie on the operating table in the supine position in open appendectomy. Although the choice of the incision varies according to the experience and preference of the surgeon, McBurney (McArthur) or Rockey-Davis incision that has a more transverse incision is preferred in uncomplicated appendicitis. A midline incision is preferred in complicated appendicitis or differential diagnosis, depending on the preference of the surgeon. The patient is placed in the supine position in laparoscopic appendectomy and the diagnosis is confirmed by entering the abdomen through the navel. Then, the surgery is performed by entering 2 more ports from the left lower quadrant or a different area according to the preference of the surgeon.

There is no need for the continuation of antibiotic therapy after surgery. Oral intake of the patient can be initiated at the post-op 6th hour after the surgery and is continued if the patient tolerates it. The patient must be mobilized as soon as possible.



5.2. Perforated Appendicitis

The treatment is surgery. As with uncomplicated appendicitis, antibiotics must be initiated as soon as the diagnosis is made. Closed or laparoscopic surgery can be performed. Although the technique is like uncomplicated acute appendicitis, more careful surgery must be performed because the tissues may be more fragile or gangrenous. If fecal contents come into contact with the abdomen, the inside of the abdomen must be irrigated and washed with fluid. In case of significant abscess cavities, a drain for the right lower quadrant must be placed.

Antibiotic treatment of the patient must continue up to post-operative 7 days (8). Oral must be initiated if mobilization is provided in the early postoperative

period and bowel sounds are heard and gas comes out. Nasogastric insertion is needed in the case of the ileus. If pain, fever, and leukocytosis are seen in the patient in the postoperative period, intra-abdominal abscess must be considered. There is a 10% risk of developing intra-abdominal abscess after perforated appendicitis. Intra-abdominal abscess status must be confirmed with CT. In the presence of an abscess, drainage from the wig is performed peruktan drenaj uygulanır, and if it is not possible, surgical drainage is needed (2).

5.3. Plastron Appendicitis

Patients may be seen days or weeks after the occurrence of acute appendicitis. On physical examination, palpation of a mass in the right lower quadrant must suggest plastron, especially in frail individuals with localized right lower quadrant pain, fever, and a history of pre-existing acute appendicitis. Primary surgical treatment must not be considered in these patients. Because of the fragile nature of the tissues and the risks of post-op complications, abscess drainage must be planned primarily with USI or CT (10-11). The drainage catheter must be kept under control for an average of 5-6 days and antibiotic treatment must continue for 7 days (8). If the acute period can be overcome in this way, surgery must be performed in elective conditions after 6-8 weeks.

5.4. Chronic Appendicitis

Patients rarely have recurrent right lower quadrant pain, but chronic appendicitis can be considered in the case of spontaneous regression of the clinical manifestation and frequent recurrence of the nose, which is consistent with acute appendicitis (1). It is seen that recurrent complaints disappear when elective appendectomy is performed in the treatment of these patients. Although these patients present with the clinical manifestation of acute appendicitis, inflammation associated with acute appendicitis is not detected radiologically.

6. Appendicitis in Special Groups

6.1. Appendicitis in Children

Clinical findings are more prominent because it is not always possible to obtain reliable anamnesis in children. The high frequency of non-specific abdominal pain complaints in children may prolong the time of taking children to the hospital. Peritonitis is difficult to limit because the development of the greater omentum is not fully developed in children. The risk of perforation is

2-fold higher in pediatric patients than in adult patients, and it is necessary to be careful in the clinical examination and to keep the surgical indication wide.

6.2. Appendicitis in Pregnant Women

The most common reason that requires nonobstetric emergency surgery in pregnant women is acute appendicitis (12). Acute appendicitis is seen in 1/1250 - 1/1500 cases in pregnancy. It is more common, especially in the first two trimesters. Symptoms such as nausea and vomiting during pregnancy may delay the diagnosis of appendicitis in the differential diagnosis. Because of the enlargement of the uterus during pregnancy, the appendix is displaced upwards, and therefore, there are difficulties in physical examination. Since leukocytosis and elevated C-Reactive Protein levels may be normal during pregnancy, it creates difficulty in diagnosis when compared to non-pregnant women. As well as these difficulties, the presence of pain because of obstetric reasons during pregnancy strengthens the diagnosis (13). USI is very helpful as an imaging method and is the first-line examination. If USI findings are unclear, MRI without gadolinium is a safe alternative that can be used to confirm or rule out the diagnosis.

In pregnancy, perforation is among the most feared complications, increasing fetal mortality to 16% and the risk of preterm birth to 11% (14). For this reason, early intervention is important in pregnant women. The percentage of negative laparotomy has increased in pregnant women because of the need for early intervention and reasons that strengthen the diagnosis.

In pregnant women, laparoscopic surgery is safe and it is important to use the Hasson Technique routinely (open access) for the first trocar placement not to damage the uterus.

6.3. Appendicitis in Elderly Patients

Although it is not common among the elderly population, the fact that symptoms are not typical and the presence of comorbidities such as hypertension and diabetes might delay the diagnosis, causing increased mortality and morbidity rates. The anamnesis may not be taken adequately because of dementia or altered mental states, and for this reason, physical examination becomes more important. In case of widespread peritonitis, urgent surgical intervention is needed. If there is no evidence of peritonitis, localized pain, and clinical suspicion, CT must be performed primarily for diagnostic purposes. Laparoscopic surgery can be performed in the absence of diseases such as cardiomyopathy or pulmonary

problems that might contraindicate surgical intervention (15). Since general anesthesia may bring a risk to the patient with advanced pulmonary problems, an open appendectomy can be performed with spinal anesthesia.

7. Conclusion

In summary, the significance and prevalence of appendicitis cases constitute a prominent health issue for surgeons. The rapid and effective management of diagnosis and treatment is crucial for patients' health and recovery process. Early diagnosis and surgical intervention can reduce the risk of complications and aid patients in achieving a healthy recovery. Therefore, careful evaluation of patients suspected of appendicitis and timely surgical intervention when necessary are vital. Additionally, the follow-up of patients and support through appropriate rehabilitation services can positively impact long-term health outcomes.

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CHAPTER XVI

DIVERTUCULAR DISEASE AND DIVERTICULITIS

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1. Introduction

Diverticulum is the herniation of the mucosa -including the muscularis mucosal layer, in the colon from the weak area of its wall to the pericolic fat layer with the effect of high pressure in its lumen. Most diverticula are pseudo-diverticula. Diverticulum mostly occurs in the colon and exclusively in the sigmoid colon. Patients with diverticulum are usually asymptomatic. However, it may become symptomatic and present as diverticular disease. Diverticulitis can be classified as acute, chronic, complicated and uncomplicated. While most uncomplicated diverticulitis regresses with medical treatment, complicated diverticulitis may require surgical intervention. The incidence of the disease increases with age. Terms related to diverticulum are shown in table 1. The risk of serious complications is highest in the first episode of diverticulitis and decreases with the number of recurrences. Acute complicated diverticulitis is associated with significant short- and long-term mortality and morbidity. In order to reduce mortality and morbidity in diverticular diseases, prompt diagnosis and appropriate treatment should be applied.

2. Epidemiology

The rate of diverticulosis and diverticulitis is similar between men and women, and increases with age. While diverticulosis is seen in 20% under 40 years of age, it is seen in 60% over 60 years of age, and diverticulitis develops in approximately 10-25% of patients with diverticulosis. The majority of

individuals with diverticulosis remain asymptomatic throughout their lives. The distribution of diverticulosis in the colon varies by geography. It is more common in Western countries than Asian countries. While it is mostly seen in the sigmoid colon in western countries, it is seen in the right colon in Asian countries. Although diverticulum is still common in the advanced age group, its frequency is increasing in the age group below the age of 45. (2-4)

Table 1: Terminology (1)

Diverticulosis coli	Presence of multiple diverticuli in the colon
Pseudo diverticulum	Herniation of mucosal and submucosal layer of kolon
Diverticulum	Herniation of whole colon wall layers
Diverticulosis	Presence of diverticula. Can be symptomatic or asymptomatic
Diverticular disease	Situation where diverticules becomes symptomatic and complicated
SUDD (Symptomatic uncomplicated diverticular disease)	Precense of symptoms without bleeding or enflamation
Diverticulitis	Inflamation of colon wall and preidiverticular area where diverticul is present
Acute non-complicated diverticulitis	Inflamation of colon wall and surrounding tissue without complication
Acute complicated divertucilitis	Diverticulitis where complications (abscess, perforation, fistula, stricture, bleeding) that go beyond the colon wall
Chronic non-complicated diverticulitis	Development of chronic inflammation when acute diverticulitis does not resolve without complications
Chronic complicated diverticulitis	Development of chronic inflammation with complication
Diverticular bleeding	Bleeding due to traumatization of the diverticulum lumen and injury to the vessels

3. Ethiopathogenesis

The pathogenesis of diverticulum formation and diverticular disease is multifactorial and is not yet fully understood. However, the most important event is the increase in intra-colon pressure. In the presence of high pressure,

the mucosa and submucosa herniate outward from the area where the colon wall is the weakest. Thus, a diverticulum is formed (Figure 1-A). Conditions that cause an increase in intra-colonic pressure and a decrease in the strength of the colon wall contribute to the formation of diverticulum. The place where the pressure is highest in the colon is the sigmoid colon, so it is most commonly seen in the sigmoid colon. Diverticulitis is the inflammatory response resulting from microscopic or macroscopic perforation of diverticulum (Fig. 1B). As the diverticulum becomes obstructed by fecalitis, the pressure increases and causes perforation. As a result of microperforation and macroperforation, they can be complicated by abscess, fistula between neighboring organs, peritonitis, bleeding and colonic stenosis. (5)

Reasons that increase the risk of developing diverticulitis and diverticulosis are the same. Diet (low fiber, high fat, rich in red meat), obesity, smoking, drugs (NSAID, corticosteroids, opioids), constipation, lack of physical activity, advanced age, connective tissue diseases (Ehler-Danlos, Marfan syndrome), genetic factors are the factors related with formation of diverticulitis. It is known that physical activity, weight loss, vegetarian diet, fiber-rich diet, statin use, and high vitamin D values have a protective effect. (6-8)

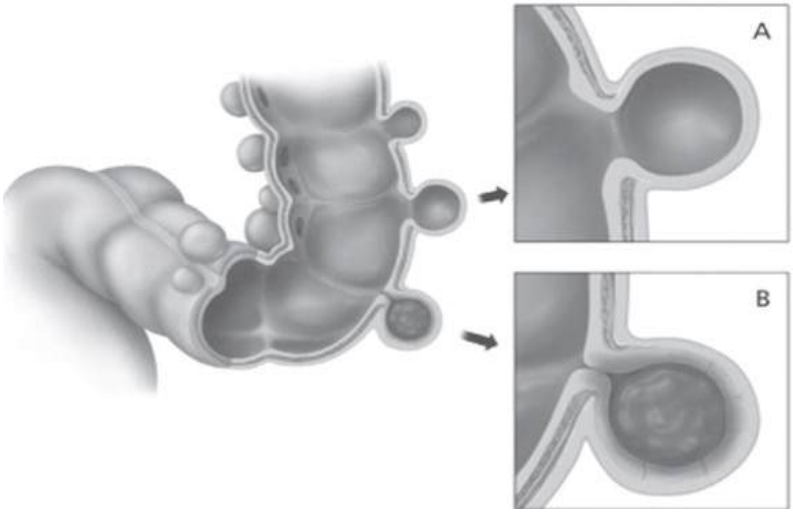

Figure 1: (A) Diverticulosis (B) Diverticulitis (9)

4. Clinical Features

Diverticulum is mostly asymptomatic. Diverticulum may become symptomatic and present as diverticular disease (SUDD (Symptomatic

uncomplicated diverticular disease), diverticulitis, diverticular bleeding). Diverticulitis can be classified as acute, chronic, complicated and uncomplicated (Figure 2-3). When the diverticulum becomes symptomatic, it is called diverticular disease. The term symptomatic uncomplicated diverticular disease is controversial. Because it remains unclear whether this condition is a disease on its own or is associated with irritable bowel disease. In complicated diverticulum; perforation, abscess, stenosis and fistula may develop. Vasa recta in the weakened wall of the diverticulum bleeds as a result of trauma. It is an important factor in lower GI bleeding. Most bleeding stops on its own. Differential diagnosis should be done well.

Clinical manifestations of acute diverticulitis vary according to the severity of the inflammatory process and complications. Abdominal pain is the most common symptom in diverticular disease. Stomachache; It can be mild or severe and continuous or cramping. In general, the pain is in the left lower quadrant and localization may vary depending on the extent of the disease. In patients of Asian descent, abdominal pain is usually in the lower right quadrant. Patients may experience constipation and diarrhea. Fever in case of abscess and peritonitis, nausea-vomiting in case of stenosis, urinary tract infection symptoms in case of fistula to the bladder, stool and gas discharge from the vagina in case of fistula to the vagina, lower gastrointestinal system bleeding after diverticular bleeding can be seen.

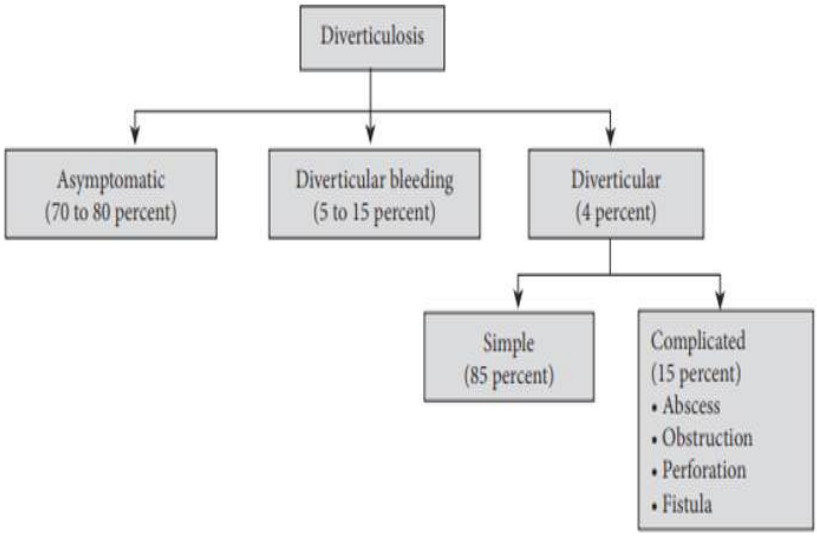

Figure 2: The course of diverticulosis (10)

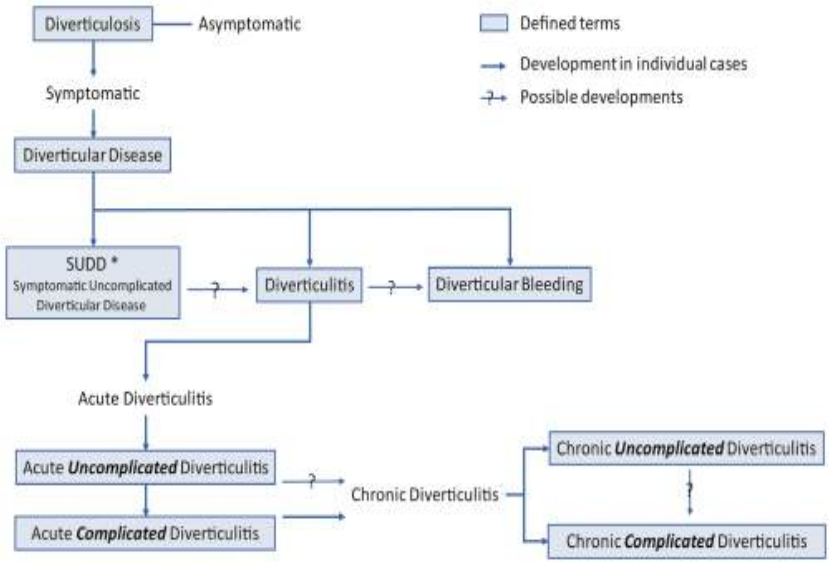

Figure 3: Stages of diverticulosis (1)

There are many classifications of diverticulitis. The classifications can show the stages of the disease and decide our method of treatment. Not all classifications used are completely safe and are not generally accepted. (1)

The first and most widely used classification is the Hinchey classification. According to this classification, which is the intraoperative classification of surgeons, the surgeon determines the severity of diverticulitis and the treatment approach (Table 2). (11) Later, with the increasing use of CT, the Hinchey classification was modified for preoperative use (Table 2). (12)

Table 2. Hinchey and modified Hinchey classification (11, 12)

Hinchey Classification		**Modified Hinchey Classification**	
Hinchey I	Pericolic abcess	Hinchey 0	Mild clinical diverticulitis
Hinchey II	Intraabdominal abcess	Hinchey Ia	Limited pericolic inflamation
Hinchey III	Purulent Peritonitis	Hinchey Ib	Limited pericolic abcess
Hinchey IV	Fecal Peritonitis	Hinchey II	Intraabdominal abcess
		Hinchey III	Generalised purulant peritonitis
		Hinchey IV	Generalised fecal peritonitis

5. Complications

Acute or chronic complications may occur in approximately 12% of patients with acute diverticulitis. Complications may occur at the first admission in patients and may develop later. (13) Acute complicated diverticulitis is associated with significant short- and long-term mortality. In complicated diverticulitis; abscess, fistula, perforation and stenosis may develop.

5.1. Perforation

Perforation develops with the development of necrosis in the inflamed diverticulum wall. There may be microperforation or open perforation. Diagnosis is made with the presence free air on imaging. The course of the treatment is decided according to the general condition of the patient and the degree of peritonitis.

5.2. Abscess

An abscess develops in approximately 17% of patients with acute diverticulitis. Diverticular abscess symptoms are similar to diverticulitis. Abscess may be suspected in patients with abdominal pain, prolonged fever and no improvement despite antibiotic therapy.

5.3. Fistula

Fistula may develop between the colon and adjacent organs with pericolonic inflammation after diverticulitis. Colovesical fistula is the most common fistula. Fistula can also be seen between the colon and the vagina, uterus, ovary, perineum, ureter, urethra and anterior abdominal wall.

5.4. Stenosis

After recurrent attacks, stenosis and obstruction may develop after healing with an inflammatory response.

6. Diagnosis

For correct diagnosis, differential diagnosis and treatment, detailed anamnesis and physical examination, laboratory tests, imaging methods, colonoscopy (except in case of acute attack) should be performed. In the anamnesis, a detailed anamnesis should be taken regarding diverticulitis and its complications. On physical examination there may be tenderness, defense, rebound and palpable fresh blood on rectal examination.

High levels of acute phase reactants such as leukocytosis, erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) in the laboratory are helpful in the diagnosis. While computed tomography (CT) of the lower and upper abdomen is the first choice in imaging methods, ultrasonography (USG) and magnetic resonance imaging (MRI) are alternative imaging methods. CT; detects diverticulitis and its complications. Due to its high sensitivity and specificity, it is the most important imaging method for both diagnosis and staging. It helps not only for diagnostic purposes but also for treatment planning by showing the severity of the disease (Figure 4). USG; It can show diverticulitis and its complications and is easy to access. Since CT is not suitable in pregnant women, USG is the first choice. MRI has the advantage of avoiding radiation. It has high sensitivity and specificity for diagnosis and differential diagnosis. However, there are costs and access difficulties. Colonoscopy; It is not recommended in the acute period due to the risk of perforation. It is recommended after about 6 weeks for diagnosis and differential diagnosis (Figure 5). (1, 14, 15)

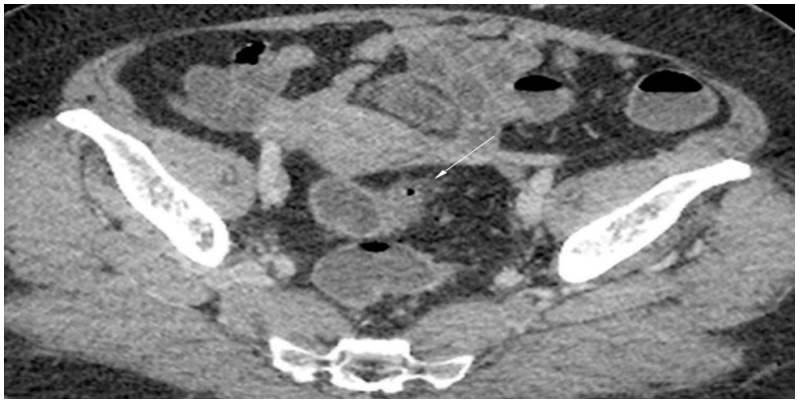

Figure 4: CT image of duverticuli

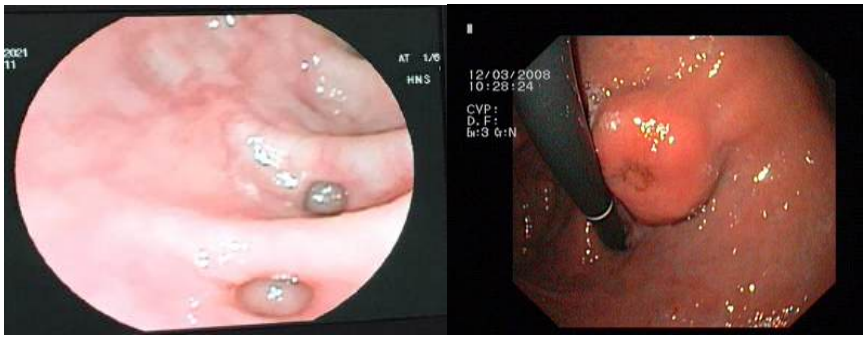

Figure 5: Endoscopic image of diverticulum

7. Differential Diagnosis

Differential diagnosis includes irritable bowel syndrome, colorectal cancer, acute appendicitis, epiploic appendagitis, ovarian torsion, tubovarian abscess, ectopic pregnancy, gastroenteritis, inflammatory bowel diseases, infectious colitis and ischemic colitis. We can clarify the diagnosis with a detailed anamnesis, physical examination, laboratory tests, CT and colonoscopy.

8. Treatment

8.1. Medical Treatment

Antibiotherapy is not recommended in patients with uncomplicated diverticulitis unless there are signs of systemic inflammation and immune deficiency. The patient's immune system can manage this process with its anti-inflammatory response. It can be treated without antibiotics with pain control and a liquid diet. Oral therapy is recommended even if antibiotics are needed. Antibiotherapy (including gram-negative bacilli and anaerobes) is recommended for patients with complicated diverticulitis, signs of systemic inflammation, and immunocompromised patients. (1, 16)

If antibiotics are the choice of treatment to patients after diverticulitis is diagnosed, it should be decided whether they should receive outpatient or inpatient treatment. If there is complicated diverticulitis, the patient is treated as an inpatient and the treatment is shaped according to the complication. . If uncomplicated, the patient should be treated as an inpatient in cases of sepsis and SIRS, age>70, microperforation, immunosuppression, severe abdominal pain and widespread peritonitis, severe comorbidities, inability to tolerate oral feeding, and not having reach for outpatient treatment. In the absence of these conditions, patients who tolerate oral intake can be treated as outpatient with oral antibiotics. In the absence of complications, the vast majority of patients can be treated on an outpatient basis. If the treatment fails in outpatients, inpatient treatment is started and CT is repeated if necessary. Outpatients are treated with oral antibiotics, analgesics and liquid diet for 7-10 days. Antibiotics acting on gram-negative bacilli and anaerobes should be treatment of choice. (1, 17-19)

The treatment of hospitalized patients is decided according to whether it is complicated or not. The treatment of complicated patients is determined according to the complication. Patients with acute diverticulitis are fully restricted orally and treated with broad-spectrum Intravenous antibiotics, IV fluids (Ringer's lactate, saline) and analgesics. After the oral intake is stopped,

clear liquid and then soft diet are started again, depending on the situation. Intravenous antibiotics are given for 3-5 days, then the patient switches to oral antibiotic therapy for 10-14 days. In uncomplicated patients, there is usually a response to treatment within 2-3 days. Patients who respond to treatment (hemodynamically stable, no abdominal pain, no leukocytosis, oral diet tolerance) are discharged to start oral antibiotic treatment, and those who do not respond are operated. (1, 17-19)

During treatment and at discharge, a liquid-soft diet with plenty of fiber is preferred. While a high-fiber diet may be recommended for general health purposes, there is little evidence that it may prevent recurrent episodes or persistent symptoms in patients with acute diverticulitis. There is no evidence to support dietary restrictions. While some guidelines recommend a liquid-soft then free diet, some guidelines have recently recommended an unrestricted diet (when tolerated). There is no evidence of bed rest, and bed rest is not recommended as imposed physical inactivity may impair the general condition of patients. In order to prevent recurrent diverticulitis or persistent complaints after an acute diverticulitis attack, mesalazine, rifaximin and probiotics can be recommended. (20)

8.2. Elective Surgery

Previously, elective colon resection was widely recommended to prevent severe episodes after the second episode of uncomplicated diverticulitis. Surgical; Although it is effective in reducing the risk of attacks, prophylactic surgery is not recommended because complications usually occur in the first attack. Although the number of attacks is high, it is recommended to perform elective surgery. The only surgical indication in patients with frequent relapses and ongoing symptoms is to improve their quality of life. In elective surgery, the indication should be limited and the treatment should be individualized according to the frequency of relapses, duration of symptoms, severity of symptoms and comorbidity of the patient.

Previously, regardless of the number of attacks, emergency surgery was recommended to diagnose and treat complicated diverticulitis. In this case, as in uncomplicated diverticulitis, resection is not routinely recommended and the approach is the same. The decision is made according to the condition of the complication and surgery is the last option.

In elective surgeries for diverticulitis, laparoscopic surgery should be preferred if possible because of its advantages. Inferior mesenteric artery sparing

surgery should be preferred by preserving the vascular and neural structures if malignancy has been ruled out. (1)

8.3. Emergency Surgery

About 25% of hospitalized patients for acute diverticulitis may require emergency surgery. Emergency surgery should be considered in septic patients who are hemodynamically unstable in acute diverticulitis. In an emergency, the aim is to resect the cause of the septic condition and try to control it. In addition, the abscess foci in the region should be destroyed and a drain should be placed in the region. Emergency surgery is often required for patients with generalized peritonitis (Hinchey III-IV). Some patients with Hinchey III can have laparoscopic lavage and drainage. If this procedure does not treat the situation, resection is performed as an alternative. Hartmann procedure was mostly used in Hinchey III and IV. However, recently, primary anastomosis with diverting ileostomy or primary anastomosis without ostomy is performed in patients with Hinchey III and IV who are hemodynamically stable and not immunosuppressed. However, some literature recommends Hartmann to be performed in patients with Hinchey IV due to peritoneal contamination. The Hartmann procedure is more preferred in hemodynamically unstable, septic and immunosuppressed patients. (1, 21)

8.4. Treatment of Complications

8.4.1. Perforation

Microperforation is not considered complicated diverticulitis and is treated with antibiotics and bowel rest. Non-surgical treatment of Hinchey I and II is recommended. Open perforations with free air in the abdomen cause generalized peritonitis (Hinchey III and IV). In this case, emergency surgery is required because of the high mortality and morbidity due to the spread of fecal fluid and bacteria into the abdomen. Anastomosis should be applied primarily to patients with Hinchey III and IV, if not possible, the Hartmann procedure should be applied. (21, 22)

8.4.2. Abscess

For small abscesses (<4 cm), antibiotic therapy alone or percutaneous drainage is recommended. Both treatments have similar morbidity and mortality. If the patient responds to the antibiotherapy, the patient is followed up with control CT, if not, percutaneous drainage or surgical procedure is applied.

Percutaneous drainage is more effective in large abscesses (≥4 cm) and 80% of patients heal with this procedure. Percutaneous drainage and antibiotherapy are used together. If the patient does not respond to treatment, surgical treatment should be evaluated. (23, 24)

In patients who underwent percutaneous drainage, if the clinical and laboratory findings regress, if the abscess does not persist after CT evaluation, the catheter should be removed. If there is no response to the treatment and the abscess persists, the position of the catheter can be changed or a surgical option can be considered. Colonoscopy is recommended after 4-6 weeks in patients with abscess whose treatment can be completed with antibiotics and percutaneous drainage. As the size of the abscess increases, the rate of negative results increases.

Surgery should be the last choice for abscesses. For most abscesses, drainage from the anterior abdominal wall is preferred, while abscesses deep in the pelvis are drained transgluteally. (16)

8.4.3. Obstruction

Surgical resection of the obstructed area is required for both treatment and differential diagnosis.

8.4.4. Fistula

Fistulas associated with diverticulum are very difficult to heal without treatment, so surgical resection of the fistula and primary anastomosis is required. Primary repair is applied to the organ outside the colon.

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CHAPTER XVII

ONCOLOGICAL SURGERY EMERGENCIES

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1. Introduction

1.1. Definition

Oncological surgery can be defined as an emergency, an acute, life-threatening situation that develops due to cancer pathology or as a result of cancer treatment. Its basis is obstruction of hollow organs or bleeding due to a tumor. The urgency is determined according to the localization of the pathology and the severity of the patient's symptoms. Urgent consultations are requested for symptoms due to malignant disease associated with poor prognosis. Obstruction (colorectal, biliary, small intestine) and infection are the most common reasons for oncological surgery emergency consultations. The main difficulty is early diagnosis and determination of the treatment algorithm. The general condition of most of the patients who need emergency oncological surgery is poor. Emergency oncological surgeries are associated with shorter-term mortality compared to elective surgeries (1,2).

Definitive or temporary relief in oncological emergencies. It is unlikely to prepare guidelines for the management of surgical oncological emergencies due to large patient variation and the multifactorial etiology of the diseases. Each patient should be evaluated individually and multidisciplinary objectively, and accordingly, an appropriate decision should be made for the patient. At this stage, the patient's performance, the stage and prognosis of the cancer, the type and severity of the oncological emergency, and the patient's wishes regarding treatment should be considered (3).

2. Obstruction

Cancer continues to be a serious public health problem that causes significant mortality and morbidity all over the world (4). Cancer patients may experience occlusion symptoms depending on different localization and causes (2). Gastrointestinal system obstructions are among the most common oncological emergencies. Intestinal obstruction is a true oncological emergency and is associated with mortality. Early identification of the disease and implementation of treatment protocols are extremely important (5).

2.1. Gastrointestinal System Obstruction

Malignant gastrointestinal obstructions present with clinically significant symptoms. The most common localizations are the distal esophagus, gastric outlet, and colon (6, 7). The main symptoms are oral intake intolerance, nausea, vomiting, abdominal pain, and inability to pass gas and stool. Initial treatment of intestinal obstructions is fluid resuscitation, correction of electrolyte imbalance, and nasogastric decompression. This conservative approach saves time for diagnosis in terms of determining the level of obstruction and the stage of malignant disease. Imaging methods (standing abdominal X-ray, abdominal computed tomography (CT)), endoscopy-colonoscopy, and laboratory tests including tumor markers can be evaluated as diagnostic parameters (Picture 1). Patients are evaluated in terms of surgical treatment (2).

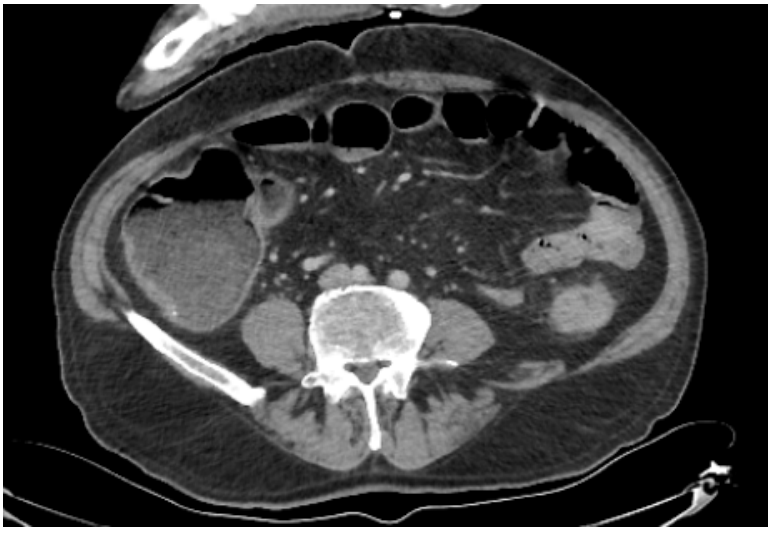

Figure 1: Abdominal CT: Tumor-related obstruction in the rectosigmoid region

2.1.1. Obstruction Causes

The main cause of malignant gastrointestinal system obstructions is the presence or invasion of intraluminal tumors (2) (Table 1).

Table 1: Causes of Gastrointestinal System Obstructions

Localization	Reason
Esophagus	Intraluminal tumor or invasion External pressure due to tumor Treatment-related edema Worsening of obstructive symptoms due to chemoradiotherapy
Stomach	Intraluminal tumor or invasion
Small intestine	Intraluminal tumor or invasion External pressure due to tumor Peritoneal carcinomatosis
Large bowel	Intraluminal tumor or invasion External pressure due to tumor

2.1.2. Disease Management

Malignant gastrointestinal obstruction should be treated in a multidisciplinary environment with an oncologist, gastroenterologist, and surgeon, considering the characteristics of the obstruction, patient expectations, prognosis of the disease, and possible surgical treatments (8).

2.1.2.1. Esophageal and Cardiac Obstructions

The five-year survival rate of patients with esophageal cancer is less than % 10, and dysphagia is the most common symptom. Most patients have unresectable disease at diagnosis, resulting in obstruction.

Stents are used to relieve obstructive symptoms in patients with advanced esophageal, gastroduodenal, and pancreaticobiliary malignancies. Most patients with gastrointestinal cancer present with advanced, inoperable disease. Elderly patients with comorbid factors often have a low chance of surgery. This has led to the widespread use of SEMS (9).

For unresectable esophageal cancer obstruction, clinicians should consider placing SEMS. Routine SEMS should not be placed for patients with resectable esophageal cancer obstruction who are potential candidates for chemoradiotherapy. Clinicians can use enteral feeding tubes for patients with resectable esophageal cancer obstruction and nutritional concerns (10).

2.1.2.2. Gastric Outlet Obstructions

Laparoscopic surgical gastrojejunostomy should be considered in patients with resectable obstructive cancer who have good performance, have a life expectancy of more than 2 months, and have a life expectancy of more than 2 months. The laparoscopic approach is associated with less blood loss and shorter hospital stays. Enteral therapy in obstructive cancer patients who are not candidates for gastrojejunostomy. stenting can be considered (8).

2.1.2.3 Malignant Colonic Obstructions

Colorectal cancers are common cancers all over the world. Patients may develop symptoms such as nausea, vomiting, abdominal pain, and constipation due to obstruction. % 7-29 of patients with colorectal cancer present with intestinal obstruction. Initial treatment is fluid electrolyte replacement, nasogastric decompression, and bowel rest. The traditional treatment of acute colonic obstructions is emergency surgery. Mortality and morbidity rates for emergency colorectal surgeries performed for acute obstruction are around 10-20%. Approximately 66% of patients undergoing emergency surgery results in a permanent ostomy. The prognosis of malignant intestinal obstructions is poor, with a median survival of approximately 26-192 days (9,10).

SEMS insertion or diverting colostomy unresectable are suitable options in patients with malignant colonic obstruction (11).

2.2. Biliary System Obstruction

The most common causes of malignant biliary obstructions are pancreatic adenocarcinoma and cholangiocarcinoma. Other etiological causes can be listed as ampullar/duodenal adenocarcinoma, gallbladder adenocarcinoma, hepatocellular carcinoma, lymphoma, surrounding organ, and lymph node metastasis. The primary treatment of periampullary region tumors is curative resection, but only 20% are resectable at diagnosis because of local invasion and distant metastasis. Surgical bypass, percutaneous drainage and stenting, and endoscopic stenting are used in the palliative treatment of bile duct obstruction (12,13).

2.2.1. Diagnosis

Patients present with symptoms such as anorexia, weight loss, jaundice without pain, itching, nausea-vomiting, fever due to cholangitis, and

darkening of urine color. In the initial evaluation of obstruction, magnetic resonance cholangiopancreatography (MRCP), and endoscopic retrograde methods such as cholangiopancreatography (ERCP) are used. MRCP and ERCP can be used to differentiate malignant and benign strictures (Picture 2). Periampular region tumors can be diagnosed without tissue biopsy, but histological confirmation of the lesion is also important. A tissue biopsy can be taken by ultrasonography (USG) or computed tomography-guided puncture, ERCP or endoscopic USG (EUS) guided fine-needle aspiration. In case of failure in the diagnosis and treatment of the ERCP procedure, percutaneous transhepatic cholangiography (PTC) is used. CT and MR angiography can be used to rule out vascular invasion and to evaluate the patient in terms of resectability (12,13).

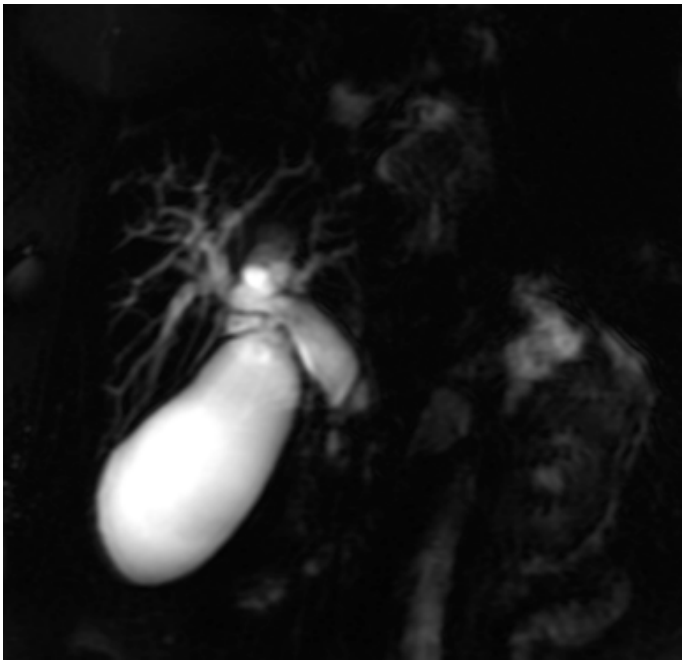

Figure 2: Sudden narrowing of the distal common bile duct (pancreatic head cancer) on MRCP

2.2.2. Treatment

Treatment is planned according to the resectability of the lesion (12,13) (Table 2).

Table 2: Treatment Algorithm of Biliary Obstructions

Operable Patients
Surgical resection
Preoperative biliary drainage
Inoperable Patients
Palliative surgical bypass
Endoscopic biliary drainage
Percutaneous biliary drainage
Stenting (SEMS, plastic stenting)

3. Perforation

In case of detection pneumoperitoneum accompanied by imaging methods, patients should be evaluated for hollow organ perforation and their physical examinations should be done with precision. Colorectal cancers and gastrointestinal lymphomas are malignancies associated with spontaneous perforations (14). Gastrointestinal system perforations are inevitably accompanied by infective and mortal complications. In primary tumor perforations of the gastrointestinal tract, the spread of tumor cells into the peritoneal cavity is associated with the progression of the disease stage and poor prognosis. Long-term obstruction due to intestinal tumors may cause tumor necrosis, disruption of mucosal integrity, and subsequent perforation (15). Malignant perforations can also occur in the esophagus and stomach. Perforations due to gastric tumors are generally an indirect indicator of advanced disease (16) (Picture 3).

Patients with gallbladder tumor perforation present with the clinical picture of acute cholecystitis as a result of long-term involvement of the ductus cysticus. Drainage treatment with percutaneous cholecystostomy can be applied to gallbladder perforations (17).

The primary focus and antimicrobial control are essential in treatment. Perforation should be controlled. Peritonitis and sepsis are inevitable in the patient as a result of the organ contents spreading into the peritoneal cavity. Early

diagnosis of uncontrolled perforations and timing of emergency laparotomy is vital. Oncological surgical principles should be followed in emergency laparotomy. Broad-spectrum antibiotics should be used. Percutaneous drainage may be considered as an appropriate treatment option in self-limiting perforations without generalized peritonitis findings, or abscess formation. If metastasis is not detected in primary gastric and intestinal tumors, surgical resection + primary anastomosis or surgical resection + ileostomy/colostomy may be considered (15).

Peritoneal lavage, ileostomy, colostomy, or percutaneous drainage can be applied in patients with poor general performance and who are not suitable for septic and surgical resection. The later clinical condition of the patient may allow for resection (15).

4. Bleeding

Bleeding in cancer patients may develop secondary to the disease itself or the treatment given. It can manifest as hematemesis, hemoptysis, hematochezia, melena, hematuria, vaginal bleeding, ecchymosis, petechiae, and epistaxis. malignant It can be seen in different stages of patients with varying severity (17). Various factors play a role in the etiology of bleeding (13,17) (Table 3)

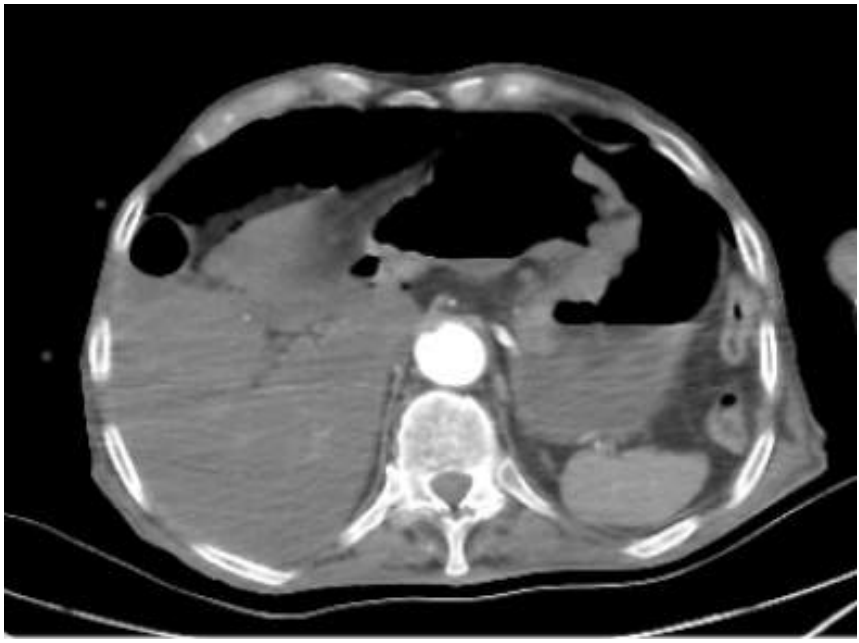

Figure 3: Abdominal CT: Tumor-related perforation in the gastric corpus

Table 3: Etiology of Bleeding in Cancer Patients

Tumor invasion causing local vascular damage
Tumor invasion into hollow organs (stomach, colon, small intestine, bladder) and mucosa
Intra-abdominal bleeding due to tumors (Hepatocellular carcinoma, renal carcinoma, melanoma)
Tumor with increased size and vascularity
Spontaneous rupture of the spleen due to lymphoma
Intra-abdominal bleeding due to leukemia
Chemotherapeutic, anti-angiogenic, non -steroidal anti-inflammatory, anticoagulant drugs
Coagulation and platelet dysfunction
Radiation therapy for pelvic malignancies
Hyperviscosity, disseminated intravascular coagulation

The primary treatment in patients with acute bleeding is close hemodynamic monitoring, intravenous fluid resuscitation, and, if necessary, blood product replacement (18, 19). The etiology of bleeding should be revealed, systemic pathologies should be defined and bleeding localization should be determined. Angiography and endoscopy are the preferred methods in terms of bleeding origin. In bleeding, primary pathology should be corrected first. In the gastrointestinal tract, endoscopy continues to be an effective minimally invasive method in terms of bleeding localization and control of bleeding. Angiography and interventional radiological embolization are effective invasive methods in terms of origin and stopping of bleeding. Despite all minimally invasive interventions, the development of permanent hemodynamic instability in the patient represents severe bleeding and may require emergency surgery (18) (Picture 4).

8. Conclusion

Oncological surgical emergencies present a wide variety of clinical presentations. The most common surgical emergencies are gastrointestinal tract obstructions. Other emergencies are perforation and bleeding.

A patient-specific multidisciplinary approach will be the most accurate management method, considering the patient's performance status, current cancer type, stage and prognosis, the severity of the emergency, treatment options, and management of possible complications.

It should be kept in mind that oncological surgical emergency interventions are associated with mortality and may shorten the life expectancy of patients, and care should be taken when performing invasive procedures.

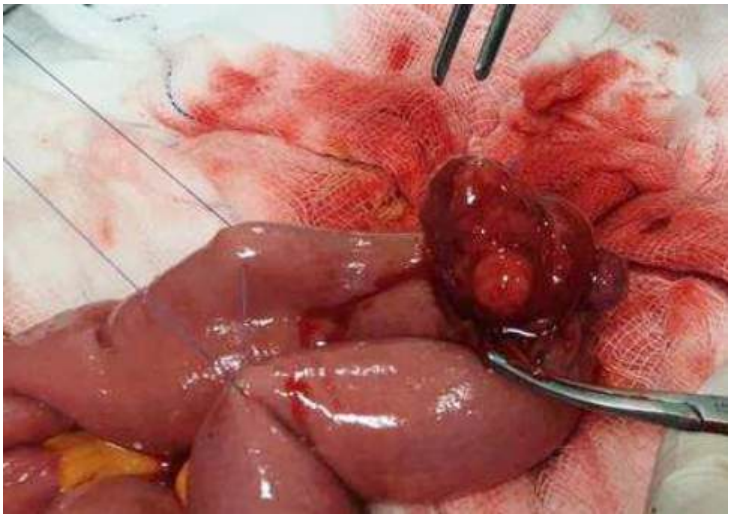

Figure 4: In my jejunum gastrointestinal bleeding due to a stromal tumor

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CHAPTER XVIII

ANORECTAL SURGICAL EMERGENCIES

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1. Introduction

When anorectal surgical emergencies are mentioned, it includes a concept that includes the group of diseases in which a region from the distal rectum to the perianal and sacral region, itself or its complications are treated surgically. This group of diseases includes elective anorectal surgery diseases that are painful enough to take the patient to the emergency room; It can also appear as a hyperacute emergency disease on its own, such as Fourniere's gangrene (1-3).

This chapter; We thought that it should be a guide that will facilitate the evaluation, diagnosis and treatment of anorectal surgery emergencies that both emergency physicians and general surgeons who deal with emergency patients as consultant physicians will encounter during their clinic practice.

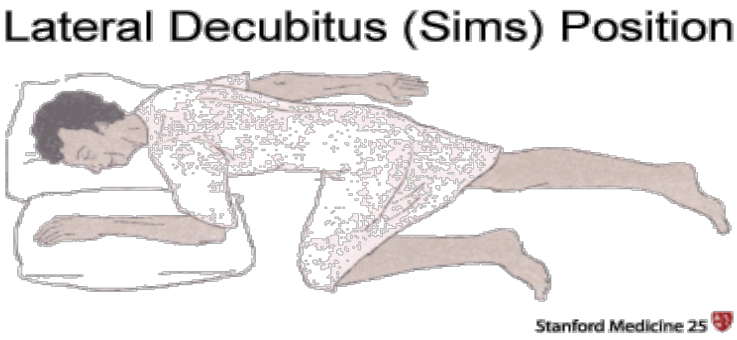
At the time of writing, we have discussed the diseases in order from the rectum to the perianal region. When talking about diseases, we talked in general. We presented the conditions that require emergency evaluation, diagnosis, treatment methods and possible complications of these diseases(4-6).

The general complaints of the patients admitted to the emergency department and the examination findings of these patients vary. The most common complaint is pain in the perianal region. This is followed by discharge and swelling in the perianal region. A small number of patients have isolated rectal bleeding without these findings. In this patient group, patients are mostly referred to the outpatient clinic electively. However more severe forms of these diseases may require urgent intervention. These patients should be carefully

evaluated when they come to the ED and those who need urgent intervention should be determined (4-7).

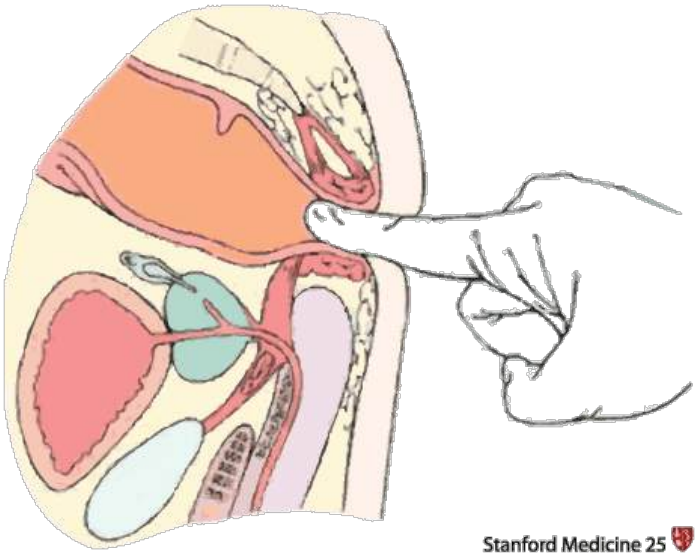
2. Medical Examination in the Emergency Room

First of all, if it is possible to transport patients, they should be taken to an examination room where privacy can be better protected. The emergency physician or consultant surgical physician should introduce himself as the person who will conduct the examination. The patient's complaints, onset of complaints, whether there is bleeding, whether there is any swelling or a palpable mass, whether there is a discharge, whether the complaints increase with defecation should be carefully questioned. During the examination phase, the patient should be given complete information about the perianal examination and rectal touch procedure. Ideal for examination, it is done on the proctology table(8-9). However, since this facility is not available in emergency services and most polyclinics, a normal examination stretcher is often sufficient. The most suitable position for the patient in the examination is the position where the patient is naked from the waist down and lying to the left. It is often sufficient that the dress is simply stripped. Some physicians prefer to examine by bending over their hands and knees. Inspection should be prioritized in the examination. It should be carefully examined starting from the sacral region, where pilonidac cysts are common, and includi the perianal region. Swelling, abscess, fistula mouth, external hermorrhoid, anal fissure. Diseases such as perianal fistula, prolapse or pilonidal cyst abscess or redness due to perianal abscess can be recognized very easily by inspection (8-9). On palpation, abscess infections in the perianal and sacral regions are palpable as hardness (5,6,8,9)



Picture 1: Examination Position (STANFORD MEDİCİNE)

Then rectal touch is passed. After wearing gloves, the anus and internal structures, including the dentate line, were checked 360 degrees with the index finger of the right hand using a liquid-based lubricant. Anus tone, palpable swelling or stiffness, whether there is pain on the touch, whether there is an internal fistula in the dentate line, and finally the nature of the contamination on the glove (bloody, etc.) are evaluated separately. It is important that the patient is not agitated during the examination. If the physician evaluating the patients with severe pain thinks that the patient cannot be evaluated with the full table, he/she may hospitalize the patient to repeat the examination under operating room conditions. As with any examination, the findings should be recorded individually and completely (1,2-5,8-10).

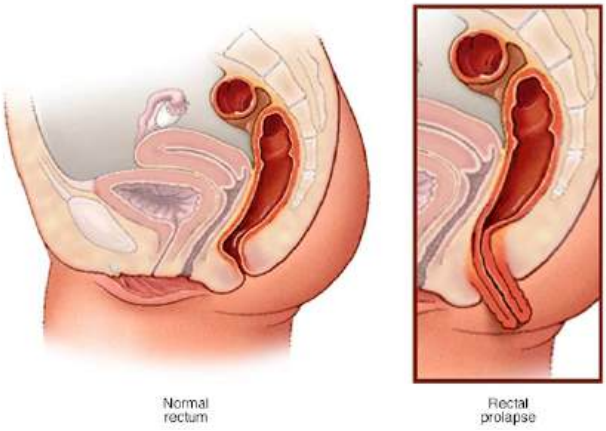


Picture 2: Rectal Touch (Stanford Medicine)

3. Rectal Prolapse

Rectal prolapse can be described as a full-thickness prolapse of the rectum from the anus. Although there is invagination of the mucosa in internal prolapse, there is no prolapse from the anus. Therefore, it would be more accurate to refer to this situation as rectal intussusception. cases where only the mucosa is protruded are mostly associated with external hemorrhoidal disease. treated like hemorrhoids. Complaints of patients are usually manifested by defect in defecation function. Protrusion of the rectum with straining, tenesm, and rectal

pain are the general symptoms. It is highly reductable due to the hypermobility of the rectum. These patients are usually evaluated and treated electively. It should be kept in mind that prolapse may coexist with rectal malignancies in these patients. The patient should be evaluated from this point of view(11).



Picture 3: Rectal Prolapse (Mayo Foundation for Medical Education and Research)

Rectal prolapse patients who should be evaluated in an emergency are those with incarceration and mucosal necrosis. Efforts to reduce often fail due to mucosal edema. Reduction should be avoided in patients with mucosal necrosis. This patient group is the group that should be evaluated in terms of emergency surgery. Since it is mostly seen in elderly patients, the comorbidities present in the patients are effective in determining the appropriate treatment method. Although many surgical methods (more than 100) have been described, the methods are classified in two groups as abdominal and perineal. There are variants of abdominal methods performed with robotic, laparoscopic and laparotomy. While abdominal approaches are generally preferred because of their lower recurrence rate. Perineal approaches may not be preferred in elderly and comorbid patients who should avoid abdominal surgery. The handicap of this approach is its higher recurrence rates (11)

3.1. Moshowitz Repair

This procedure is one of the procedures defined in the early period and includes primarily perineal reduction of the prolapse and then closure of the cul de sac with an abdominal approach. It is a method used in rectocele surgery as well as rectal prolapse. Today, it can be applied mostly laparoscopically and robotically (12,13).

3.2. Ripsten And Wells Rectopexy

In this procedure, the pelvic ligament is opened and the rectum is released. The mesh is laid posteriorly and wrapped around the anterior rectum. Afterwards, the rectum is fixed with sutures on the mesh and strengthened (14).

3.3. Suture Fixation

It is the release of the rectum and fixation to the sacrum with sutures. Its use has decreased after the mesh technique (15).

In many cases, sigmoid resection and anastomosis are added to the case, since a long sigmoid is present in abdominal approaches. This approach significantly reduces recurrence rates.

3.4. Altemeier Procedure

Of the perineal approaches. It is performed by excising the protruding intestine with a perneal approach and anastomosis. It is a preferred method in elderly and patients with high comorbidity (13-15).

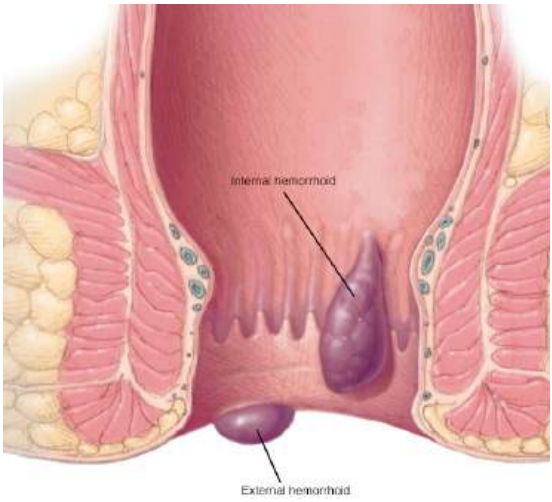
3.5.Delorme Procedure

It is a method that has become popular recently. It involves excising the prolapsed anus mucosa and detecting the remaining mucosa by plicating it together with the muscles. It is a practical surgery that can be used in emergency cases (12,13,15).

4. Hemorrhoidal Disease

Hemorrhoidal diseases are frequently encountered diseases in general surgery clinical practice (16). Structures called hemorrhoids anatomically. They are bubbles located under the mucosa consisting of arterioles and venules. It is thought to help close the anal canal at rest. Hemorrhoid structures, which are anatomically present in every human being, are not felt under normal conditions (16). However, when they are swollen and symptomatic, the disease is mentioned and treated. Hemorrhoidal pouches are divided into internal and external pouches. The external packers are located distal to the dentate line. These packs are covered by the skin of the anus and their nervous innervation is very sensitive. For this reason, external hemorrhoidal diseases are mostly manifested by anal pain. Internal packs, on the other hand, are proximal to the dentate line, covered with mucous membranes, and have no nerve innervation. The causes of hemorrhoidal disease are mostly increased intra-abdominal pressure, severe

and recurrent constipation, etc (16). When staging hemorrhoidal defects, it is evaluated by the prolapse level. Grade 1 hemorrhoids are those that prolapse distal to the dentate line and protrude into the anal canal. Grade 2 hemorrhoids are hemorrhoids that can prolapse out of the anus but are spontaneously reduced. Grade 3 hemorrhoids are hemorrhoids that prolapse from the anal canal and prolapse with mechanical intervention. Grade 4 is hemorrhoids that are not manually reduced and have a risk of strangulation.[16] The continuation of external hemorrhoids in the skin is called skin-tag and is often confused with the hemorrhoidal pack does not need treatment. However, it can be excised with the pack during surgery.



Picture 4: Hemorrhoidal Diseases (Mayo Foundation for Medical Education and Research)

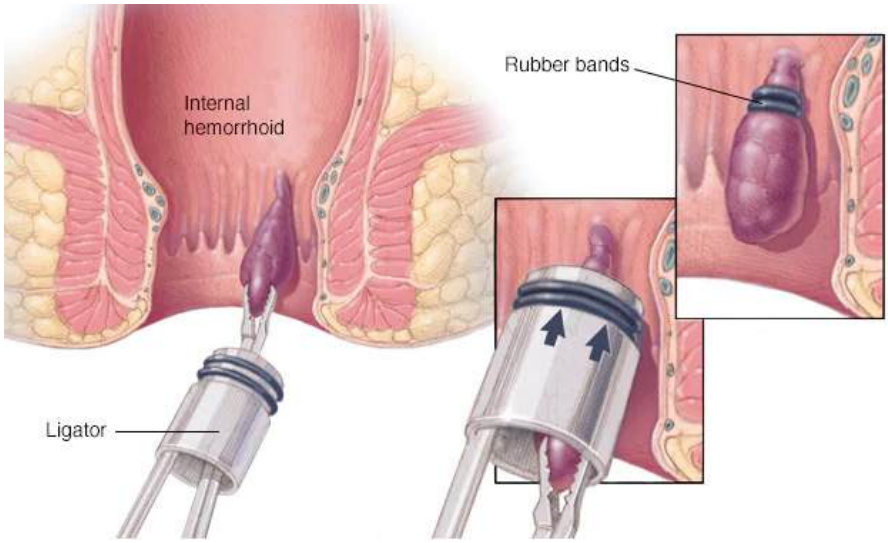
Diagnosis is made by system interrogation and rectal examination. In the interrogation, it is evaluated whether there is pain, bleeding, whether he has used drugs before. In the examination, the presence of hemorrhoids, whether there is thrombosis, and the staging of the hemorrhoidal disease (mentioned above) are revealed in the inspection. In patients with bleeding, it should be clarified that the bleeding is due to hemorrhoids. Sometimes patients may present to the emergency department with massive bleeding, severe low hemoglobin, and severe and analgesic-resistant pain. This is the group of patients who need urgent intervention (4,8,9,16).

In the treatment of hemorrhoidal diseases, treatment options are evaluated according to the stage of the disease. Local evacuation of thrombosis in

thrombosed hemorrhoids can be considered as an emergency intervention. Patients who cannot tolerate the intervention can be evaluated in the operating room. Grade 1 and grade 2 diseases can usually be treated with dietary and habitual changes and medical treatment. Band ligation can be applied in grade 1,2 and selected grade 3 patients with persistent bleeding. Grade 3 and 4 patients often do not benefit from medical treatment. First of all, it is evaluated with the operation (16-20).

4.1. Band ligation

It is based on strangulation of the hemorrhoidal pouch by placing rubber bands on the hemorrhoid root with a special vacuum applicator. It is used in grade 1,2 and selected grade 3 hemorrhoids. It's an element that needs attention. It is used for internal hemorrhoids originating from the proximal dentate line. It is not preferred because of postoperative pain in external ones(20).



Picture 5: Band Ligation (Mayo Foundation for Medical Education and Research)

4.2. İnfrared photocoagulation

It is a widely used method recently. It is applied efficiently and easily in grade 1-2 hemorrhoids. It is treated by penetrating the hemorrhoidal pouch and burning it with a special probe and photocoagulation device produced for this purpose. Its efficiency is lower in larger and thrombosed hemorrhoids (19).

4.3. Sclerotherapy

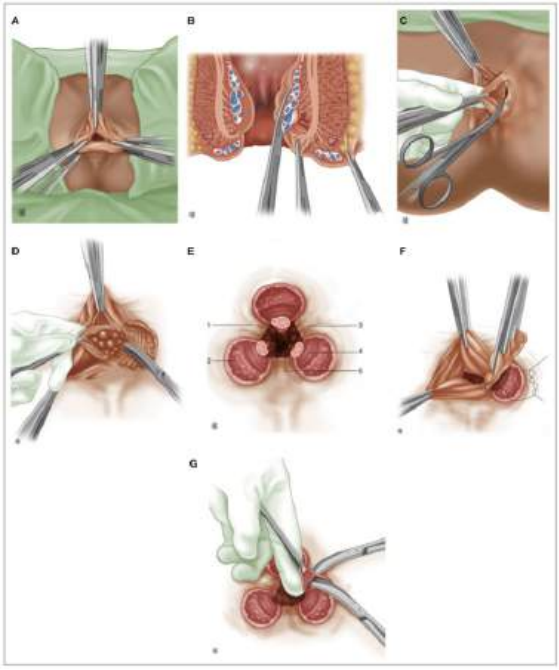
It is applied by injecting 1-3 ml of sclerosing substance into internal hemorrhoids. It is applied in grade 1-2 hemorrhoids. It is a painless procedure (16).

4.4. Ferguson Hemorrhoidectomy

Also called closed hemorrhoidectomy. It is lifted by protecting the external sphincter starting from the anal canal entrance and up to the proximal anal canal. The root, which also provides vascularization on the anal werge side, is ligated and excised. The remaining opening is closed continuously with absorbable sutures. In this technique, all hemorrhoid packs can be resected. However, major resection should be avoided in order to avoid anal stenosis (16-18).

4.5. Miligan and Morgan Hemorrhoidectomy

It is similar in method to Ferguson. The only difference is that the opening is not closed and the wound is left open. It is the gold standard method in the surgical treatment of hemorrhoids. The length of the postoperative recovery period is the most important handicap (16-18).



Picture 6: Milligan&Morgan Procedure (Classical treatment of hemorrhoids. J Visc Surg. (2015).

4.6. Longo (stapler) Hemorrhoidectomy

It is based on suspending the mucosa proxmal to the dentate line to the anal canal mucosa with a circular stapler-like special device and excision of the mucosa, feeding vessels and hemorrhoidal pouches in between. It is a clear and high quality treatment method for all hemorrhoids in the 360-degree anal canal. Its handicap is that it requires a special device and can rarely cause life-threatening complications (anastomotic leakage) (17,18).

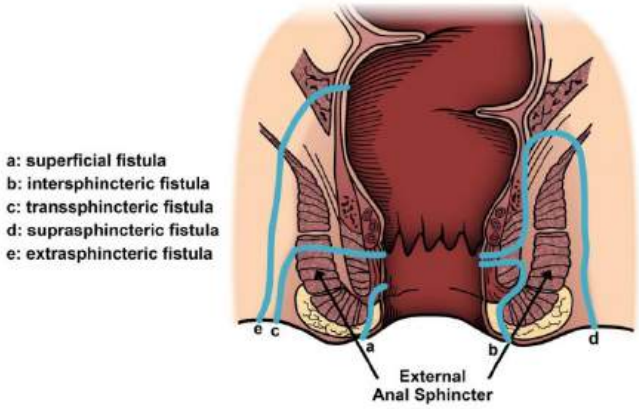
Postoperative pain is the most common complication after hemorrhoidectomy. These patients should be given analgesic support. Urinary retention develops in a significant proportion of patients. This situation can be relieved with analgesic support. Constipation occurs due to pain in the majority of patients cannot be treated with laxative agents. The two most feared complications in clinical practice are fecal incontinence and Fourniere's gangrene. It may develop after operations in which the external sphincter is not properly preserved. The treatment is the detection and repair of the sphincter openings with the eus. Despite treatment, the results are not encouraging. Although wound infection is not very common after hemrooidectomy, Fourniere's gangrene may develop in predisposed patients. Since this group of diseases will be discussed as a separate section in the future, it will not be discussed in detail. Apart from all these, postoperative bleeding is one of the most common complications. The early period is very common. It is mostly resolved by suturation (16-20).

5. Anal Fistula

Perianal fistulas are fistula tracts that occur as a result of spontaneous or drainage opening of perianal abscesses (to be discussed later) arising from the crypts from the skin. In some cases, radiation, chronic disease, and chronic infection also cause fistulas. This should also be considered in fistulas that do not resolve despite treatment (10,21-23).

Patients usually present to the emergency department with fever, discharge, and in some cases, abscess formation due to blockage of the fistula tract. On examination, the external orifice of the fistula tract is seen. If abscess formation has occurred, abscess is considered as hardness. In a small number of cases, the internal orifice may be palpable on the touch. Often the internal orifice cannot be detected and requires detailed anorectal examination under operating room conditions. Patients with abscess formation need to restore drainage. Intervention may be required, especially in patients with infective findings. Patients who need to be intervened in the emergency examination should be carefully evaluated.

In patients whose tract cannot be detected, estimates can be made according to the Googsall rule. while the external orifice is directly connected to the internal orifice anteriorly by a short and non-curved line. posteriorly, it curves from the midline and extends to the external orifice(10,21-23)



Picture 7: Perianal Fistula Staging
(American Society of Colon & Rectal Surgeons)

Fistulas are staged according to their relationship with the anal sphincters. Grade A: simple intersphincteric fistula. It may have little or no connection with the internal sphincter. It has no connection with the external sphincter. Grade B: uncomplicated transsphincteric fistula: These are fistulas that involve a small part of the external sphincter, including the internal sphincter. Grade C: complicated transsphincteric fistula. It includes the entire external sphincter and is located high. Grade D: fistulas passing through the superior of the highly located sphincters (10,21).

In the treatment of emergency patients, if there is abscess formation, drainage and inpatient or outpatient antibiotic therapy can be started. If the clinic is severe, the surgery that can be done elective can be taken to the emergency conditions. Elective or emergency treatment in perianal fistula is always surgery. The important thing in the intervention is the preservation of continence. Therefore, the surgical method is determined according to the type of fistula (10,21-23).

5.1. Simple Fistulotomy

It is performed by passing a stylet from the external sphincter and the internal sphincter under an anoscope, uncovering the top and performing curettage. The open wound is left for secondary healing. Grade A-B fistulas can be treated comfortably in this way (10,21-23).

5.2. Seton Technique

In this technique, since cutting the external sphincter will cause incontinence, the fistula tract is not opened surgically. The tract is exposed with a stylet and tied with an insoluble surgical thread. It is used in grade C-D fistulas involving the external sphincter. There are two types of seton according to its function. The incisors are tied tautly and tightened with intermediate intervention to simplify the fistula by cutting the tract with fibrosis. On the other hand, the drainage seton does not bind tightly, it protects the drainage by keeping the tract open and helps the fistula heal itself with antibiotic therapy (10,21-23).

6. Anal Fissura

Anal fissure is a tear distal to the dentate line. The process becomes chronic due to the hardening of the internal sphincter due to post-tear pain caused by hard stool due to constipation, and the development of ischemia and fibrosis. The fissure is usually in the posterior midline.

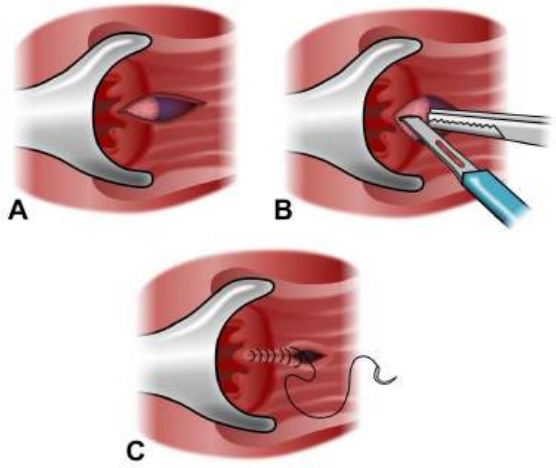
Patients often present to the outpatient clinic or emergency room with severe pain and bleeding during defecation. Fissure can be seen when the hips are separated on inspection. Patients often experience severe pain or cannot tolerate the touch. The internal sphincter is thickly palpable on touch. Also, if it is advanced, the fissure itself is palpable (24).

The basic approach in treatment is to prevent spasm and break the vicious circle. First of all, medical treatment and lifestyle changes, stool softeners, nitrous oxide and diltise preparations to provide sphincter loosening, and warm water sitz baths are tried. NO generally lags behind diltizem preparations because it causes headache. While acute fissures can be treated 100% medically, this rate is low in chronic fissures (25).

In some patients, treatment is applied by providing sphincter relaxation with botulium toxin injection. Its superiority to medical treatment has not been proven in studies (25-28).

6.1. Lateral internal sphincterotomy

As a surgical procedure, lateral internal sphincterotomy is used, which is beneficial in almost all patients. In the lithotomy position, the internal sphincter is isolated and thinned under the anoscope at the 3 o'clock quadrant. In this way, the cycle of spasm and fibrosis is broken and the fissure heals within its own dynamics. Today, the gold standard surgical treatment of anal fissure is lateral internal sphincterotomy. The procedure may cause external sphincter damage and incontinence in inexperienced hands(7,29-30).



Picture 8: Lateral Internal Sphincterotomy
(American Society of Colon & Rectal Surgeons)

7. Condyloma Acuminate

Genital warts are an infection caused by certain types of human papillomavirus (HPV). It is mostly sexually transmitted. Often they can protrude from the surface of the skin and their color may change; There may be several warts or several joined together to appear in a cluster or in the shape of a cauliflower. They may be itchy and feel burning. However, they can sometimes be painful. They typically occur one to eight months after exposure. Warts are the most obvious and common symptom of genital HPV infection.

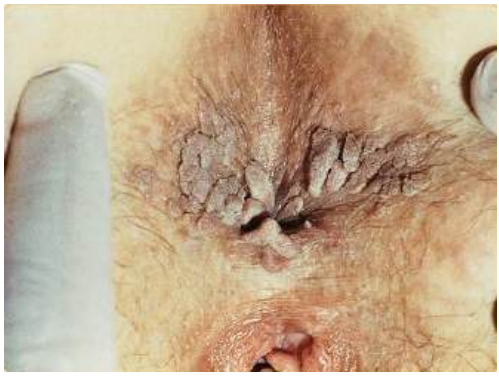
HPV types 6 and 11 are the subgroup that causes most genital warts. It is spread through direct skin-to-skin contact, usually during oral, genital or anal sex with an infected partner. Diagnosis is usually based on symptoms and can be confirmed by biopsy. The types of HPV that cause cancer are not the same as those that cause warts (7,30).

They can be as small as 1-5 mm in diameter, or they can grow in the genital or anal region or spread in large masses.Tthey form a cauliflower-like scaly appearance. They can be hard or soft. Their color may be variable and they may bleed sometimes.

In most cases, there are no signs of HPV infection other than the warts themselves. Sometimes warts can cause itching, redness, or discomfort, especially when they appear around the anus. Although it usually does not show other physical symptoms, a genital wart outbreak can cause psychological distress, such as anxiety, in some people.

The types of HPV that cause warts are highly contagious. It is mostly transmitted by skin-to-skin contact and sexual intercourse. About three out of four unaffected partners of wart patients develop warts within eight months. Other studies of partner compatibility suggest that the presence of visible warts may be an indicator of increased contagiousness; HPV concordance rates are higher in couples where one of the partners has visible warts (7,30).

It is quite easy to diagnose. The presence of scaly and itchy swelling in the peranal region is a serious diagnostic tool. Patients are mostly discharged, hemorrhagic and erosive lesions in the emergency department.



Picture 9: Perianal Condulome (SOA-AİDS Amsterdam)

What is more important than treatment is preventive measures against hpv. Standardizing the HPV vaccine is on the agenda all over the world. Symptomatic treatment is at the forefront of treatment. Start with topical emollients continue with oral treatments. Cryotherapy for lesions is often used. In persistent lesions, cautery ablation and resection may be preferred (7,30).

8. Pilonidal Disease

Pilonidal disease is a frequently recurrent disease with abscesses and fistulas, mostly seen in the sacral region, especially in young adults. The disease is popularly known as ingrown hairs. The general opinion is that the disease is acquired. In traumas occurring in the sacral region, the follicles become abscessed and progress to the subdermal area, forming a fistula tract. The hair follicles reach deeper layers with the vacuum effect that occurs due to the region movement and the abscess effect increases. The resulting sinus usually consists of a primary sinus of about 3 cm and has spontaneous drainage. Patients usually come to the emergency room with abscess formation and severe pain due to blockage of the sinus tract. Deep intergluteal sulcus, hair density and sedentary

life are risk factors. Diagnosis is almost always by clinical examination. MRI is rarely useful in obscure clinics. Most of the patients complain of not being able to sit, and a handful of them have a history of abscess that has been drained before (31).

Treatments for pilonidal disease are divided into surgical and non-surgical treatments. The common opinion is that non-surgical treatment methods are not effective and have high recurrence rates (31).

8.1. Phenol Treatment

Phenol, a naphthalene-like material, can be used for chemical cauterization of sinus. While silver nitrate and fibrib glue can also be used in the treatment, the most common non-surgical treatment is phenol treatment. Generally, 80% solution of phenol is used. It should be kept away from other tissues as it is highly irritating. First of all, the sinus mouth is opened with a clean clamp and the hairs inside are cleaned then phenol is applied. Often repeated sessions may be required. The recurrence rate is high (31).

8.2. Drainage

İs at the forefront of emergency interventions. In case of severe pain, inability to sit, and swelling, draining the abscess and using antibiotics for 10 days is the first intervention. Afterwards, elective surgery can be planned. Antibiotherapy is sufficient for abscesses that drain on their own. If the hairs in the abscess can be reached through the sinus, cleaning can be attempted (31,32).

8.3. Fistulotomy

Sinus tract can be applied if it is simple and small. It is preferred that the fistula tract is superficial. A stylet is placed inside the fistula and it opens up curetted and left for secondary healing. Antibiotherapy can be started (31,32).

8.4. Primary Excision, Secondary Healing

The area containing clean tissue including the sinuses is determined (with stylet or methylene blue if necessary) and removed unblocked up to the presacral fascia and left for secondary healing. Requires regular dressing and has a high recurrence rate. Its use has been almost abandoned (32,33).

8.5. Primary Excision And Marsupialization

Technically the same as before. At the end of the procedure, the skin opening is sutured to the presactal fascia. The disadvantage is that it takes a

long time to heal and requires frequent dressings. However, its advantage is low recurrence rate and short return to normal life (32,33).

8.6. Primary Excision And Midline Closure

It is technically the same as the previous ones and is based on closing the resulting gap in the midline with appropriate surgical techniques. The healing rate is faster than leaving it open, and due to the formation of dead space, infection, separation in the wound, and discharge are more common (31-33).

8.7. Midline Shift Surgeries

Since wound dehiscence and infections are common in surgeries where the sutures remain in the midline, surgeries in which the midline is shifted with flaps have been described. Surgeries such as karydakis and cleft-lift are methods that have increased in use recently. Especially cleft-lift surgery is thought to be the most common surgical method in the future due to low recurrence rates and minimal healthy tissue loss(31-34).

8.8. Sliding Flap Surgeries

Limberg flap and rhomboid flap can be used in relapse cases and closure of the defect in large sinuses. Retraction of the flap used from the gluteal region. The advantages of this type of surgery are that there is no blood supply disorder, less recurrence and reduction of the intergluteal sulcus depth. It is also preferred because the recovery time and the time to rejoin social life are faster (31,33-37).

Picture 10: Limberg Flap Surgery for Diffuse Pilonıdal Sinüs (Dr. H. B. PIYADE)

The reason why there is still no common surgical view on pilonidal sinus is that no treatment method offers a definitive and ideal solution. Wound infections, wound dehiscence, recurrence are very common complications. The ideal treatment is still a subject that many clinics are working on (31).

9. Perianal Abscess

Abscesses of the anal region show a general variety as cryptoglandular abscesses, perianal abscesses, ischiorectal abscesses, and supralevator abscesses. Almost all of them are accompanied by severe pain, fever, sepsis findings and mostly come with emergency admission (5,6).

9.1. Cryptoglandular Abscesses

It occurs with the disruption of drainage and abscessing of the anorectal glands opening to the dentate line. It presents with fever, severe pain and signs of sepsis. It can be detected by touching on suspicion or by rectal examination in operating room conditions. Pelvic MRI is conclusive in patients who are not clear with examination. It should be resisted from the moment it is detected. Drainage is often required in operating room conditions. Antibiotic use is required for up to 10 days after drainage (5,6,10).

9.2. Perianal Abscess

Most perianal abscesses apply to the emergency or outpatient clinic with complaints of pain and can be drained in outpatient clinics and emergencies. More complicated ones should be evacuated in the operating room. Antibiotic therapy is started for 10 days after drainage. Additional treatment is often not required.

Perianal abscesses are very common in immunosuppressed patients. In this group, if there is neutropenia, examination and drainage should be performed, but the patient should be followed closely in terms of Fourniere's gangrene, culture should be taken and antibiotic therapy should be started (5,6,10).

10. Necrotizing Soft Tissue İnfection of the Perineum (Fournier Gangrene)

Fournier gangrene is a type of necrotizing fasciitis or gangrene affecting the external genitalia or perineum. It commonly occurs in older men, but it can also occur in women and children. It is more likely to occur in diabetics, alcoholics, or those who are immunocompromised. Symptoms of Fournier gangrene include swelling or sudden pain in the scrotum, fever, pallor, and generalized weakness.

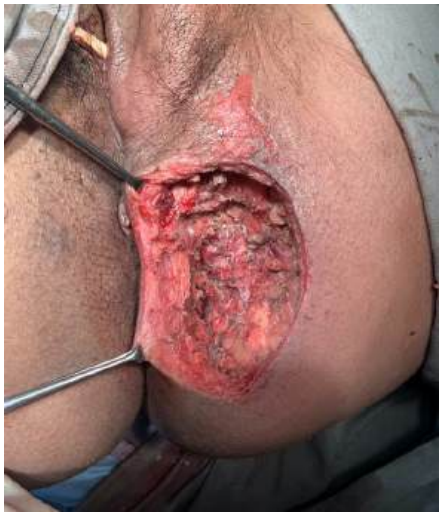
It is characterized by pain that extends beyond the border of the demarcated erythema. Subcutaneous air is often one of the specific clinical signs, but is not seen in >50% of presenting clinical cases. Many cases are characterized by a foul odor and necrotic infected tissue. Crepitus has been seen mainly. It begins as a subcutaneous infection. However, necrotic patches soon appear in the overlying skin, which later develop into necrosis (1,2).

Most cases of Fournier gangrene are infected with Clostridium perfringens. It can also result from infections caused by group A streptococcus (1,2).

Fourniere's gangrene can be seen very frequently in diabetic alcoholic and immunosuppressive patients with low self-care. In some patients, the clinic can progress rapidly, which can be expressed in hours(1,2).

The diagnosis is usually made clinically. Presence of necrotic wound, foul odor, and severe pain are diagnostic. In complex cases, the presence of gas under the skin on tomography is diagnostic.

Surgical debridement and empirical antibiotic therapy are started early in the treatment. Antibiotherapy is revised according to the tissue culture result. May need repeated debridement. It requires a multidisciplinary approach according to the tissues where the necrosis extends. rology in necrosis extending to the scrotum, general surgery in necrosis extending to the anus and anal sphincters, and plastic surgery in the chronic wound process are included in the treatment. Opening a colostomy in cases where anal incontinence will develop repeated debridements. A long-term treatment process is required with supportive treatments such as hyperbaric oxygen therapy. Recently, vac treatments have come to the fore instead of long-term dressings (1,2).



Picture 11: Fourniere's Gangrene Intraoperative Look (American Journal of Emergency Cases)

Despite long-term treatment, the mortality rate is close to 40%. Long term stay heavy and painful dressing series. Debridement and wound closure surgeries one after another strain the patient both physically and mentally and cause additional morbidity (1,2).

11. Conclusion

Anorectal emergencies are common and usually present as subacute emergencies. While most of these diseases require elective examination and treatment, their complications less frequently require evaluation in the emergency room. Since these complications (such as Fourniere's gangrene) can be fatal, a detailed rectal examination and holistic evaluation of the patient is required. The risk of death decreases when treatment is started quickly and correctly. The aim of the department is to help clinicians make the right decisions when faced with anorectal emergencies.

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CHAPTER XIX

BURNS AND TREATMENT

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1. Introduction

Burns are a type of tissue injury that can result from exposure to high temperatures or other factors such as electricity, chemicals, sunlight, radiation, or hot liquids. They are more common in risk groups like children and the elderly. (1) The severity and type of burn determine the appropriate treatment. Prompt first aid is crucial to prevent infection and reduce pain. The treatment process typically involves managing pain, wound care, skin grafting, medication, hygiene, and nutrition. Effective wound care is of great importance to promote quick healing of burn wounds and minimize the risk of infection. (2)

2. Classification of Burns

Burns can be classified according to the depth of tissue damage caused by the burn. First-degree burns affect only the top layer of skin, causing mild symptoms such as redness, swelling, and pain, similar to sunburn and are referred to as superficial burns. Second-degree burns are deeper and affect both the top and lower layers of skin, causing symptoms like redness, blisters, and skin peeling. Further, such burns are painful and cause the affected area to be sensitive to touch. Third-degree burns affect all layers of the skin, causing a range of symptoms such as skin discoloration (white, brown, or black), numbness, hollow skin, hardness, and crusting. This type of burn is the most severe form of burns and can cause permanent skin damage and may require skin grafts. The extent and severity of burns can also be classified by surface area, which is an important factor in determining treatment options. The Wallace rule, developed by Elwood P. C. Wallace, is a commonly used method for calculating the burn

surface area of a specific body part.(3) The Wallace rule estimates the surface area of a burn by proportioning it to the total surface area of the affected body portion, thus helping determine burn severity, extent, and morbidity, and then classifying it into the following categories:

First-degree: 10% or less of the total body surface area is affected.
Second-degree: 10-20% of the total body surface area is affected.
Third-degree: More than 20% of the total body surface area is affected.

This classification utilizes the "rule of nines" to estimate the burn surface area. The rule of nines assigns a specific surface area to different body parts, and the sum of these areas is used to estimate the total burn surface area. This method is very useful in accurately determining the extent of the burn injury.

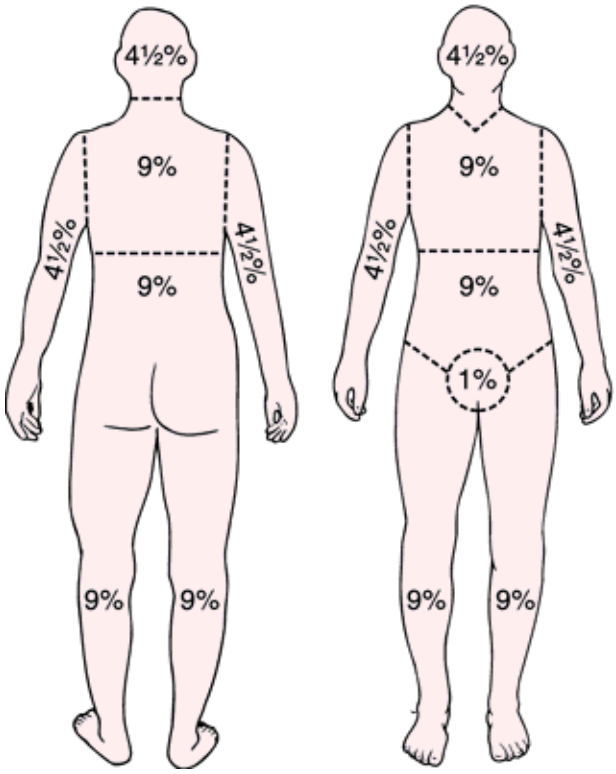

Figure 1: Rule of nines

The Wallace classification is not only used to estimate the surface area of the burn, but also to assess its depth. The depth of the burn is determined based

on whether the underlying tissues are affected. The assessment is conducted as follows:

First-degree burn: Only the outer layer of the skin is affected, with redness and mild swelling. The burn area is painful to the touch.

Second-degree burn: The lower layers of the skin are also burned, and blisters and skin peeling may occur in the affected area. Pain level can vary.

Third-degree burn: All layers of the skin and underlying tissues are burned, resulting in white, black, or brown discoloration of the skin. There is no pain in the affected area.

Although the Wallace classification is a useful tool for estimating burn surface area and depth, it may not be sufficient for developing a treatment plan. Therefore, other factors should also be considered.

3. Physiopathological Changes in Burn

Physiopathological changes that occur after burns are generally proportional to the depth and extent of the damage. After a burn, the local vascular permeability increases, causing fluid, protein, and electrolyte loss at the site of burn injury. Later, hypovolemia and shock may develop with decreased vascular reactivity. Blood coagulation may be impaired as a result of systemic changes after a burn. Fibrinogen and platelet use may be required. Burn-induced coagulopathy is more common in cases of extensive burns or when a large part of the total body surface area is affected. The loss of fluid also leads to dehydration and edema, which can increase tissue pressure at the burn site and cause further damage. In addition, the production of free radicals as a result of inflammation and cell damage in the burn area increases the risk of infection. The immune system plays a vital role in controlling infections after a burn, but factors such as post-burn stress can cause an increase in cortisol and adrenaline levels and suppress immune functions.(4) The formation of granulation tissue and fibrosis are important for post-burn healing, as they promote tissue regeneration and initiate the healing process.(5)

4. Treatment

Burn treatment varies depending on the severity of the burn and the affected body surface area. The goals of burn treatment include pain control, reducing infection risk, minimizing tissue damage, and promoting recovery. The

general approach involves cleaning, sterilizing, moisturizing, and preventing infection of the burn area.

First-degree burns affect only the surface of the skin and can usually be treated at home. Applying cold water for first aid helps relieve pain. Such burns typically heal within a few days to tens of days, and no further treatment is usually required. Topical antibiotics with silver sulfadiazine can help prevent infection.

It is important to note that infection control is critical in all burn treatment and systemic use of antibiotics for infection control is indicated only if infection is confirmed via hemoculture.

Second-degree burns affect the dermis and epidermis layers and may cause blisters and painful lesions. Careful opening of blisters will accelerate wound healing. Treatment of such burns includes moisturizing, sterilizing, and bandaging the skin. Infection control should be provided with local silver-containing agents. Analgesic agents are also used for pain control during wound care.

The healing process of second-degree burns generally takes 2-3 weeks, with attention given to granulation formation, wound dressing, cleaning, hygiene, infection control, and prevention of scarring. During the healing process, the wound dressing should be changed regularly and the risk of infection should be minimized.

Third-degree burns penetrate deep into the tissues and can be fatal if not treated. Treatment requires immediate medical attention and often involves skin grafting.

Before starting treatment, it is important to stabilize the patient and ensure breathing. The main goals are to prevent infection, reduce tissue loss, control pain, and accelerate healing. Treatment of third-degree burns should be conducted in specialized burn centers. After the first intervention, the patient should be referred to the burn center, not exceeding a 7-hour transfer period.

Surgical intervention, skin grafting, and debridement are frequently used. Surgical intervention involves the removal of damaged tissues and prevents the spread of infection. However, in recent years, stem cell therapy, artificial skin grafts, and nanotechnology-based treatments have also been used for the treatment of third-degree burns. These alternative treatments may provide advantages such as accelerating the healing process, reducing scarring, and decreasing the risk of infection. (6)

5. Nutrition

Nutrition is a critical aspect of burn treatment as patients often experience weight loss due to metabolic stress and fluid loss in the body. Therefore, adequate nutrition can significantly speed up the healing process. Burn patients are advised to follow a diet that is high in protein, low in fat, and contains sufficient carbohydrates. Burn injuries cause a spike in metabolism and energy expenditure, which is why patients should follow a balanced and appropriate diet plan.

The nutritional requirements of burn patients may vary depending on the degree of burn, body weight, and overall health status. A diet that is high in calories and protein can help with wound healing and strengthen the immune system by providing the body with the energy and proteins it needs.(7) It is also recommended to consume foods that are rich in vitamin C and zinc, which can accelerate wound healing.(8) Hydration is crucial for burn patients as they often experience fluid loss in the body, which can slow down the healing process. Therefore, it is essential to monitor the fluid intake of burn patients and ensure that they remain adequately hydrated.

6. Complications

Burn injuries can lead to severe complications, such as life-threatening multiple organ failure, infection, hypovolemia, and hypothermia, which can have fatal consequences if left untreated. Life-threatening multi-organ failure is a common complication in the early stages of burn injuries, where systemic inflammation and hypoxemia caused by burn injury can affect other organs in the body. Intensive care therapy and supportive therapies are critical in the treatment of this complication. (9)

Infection is a severe complication that can increase the risk of death in burn patients. Appropriate antibiotic agents are used to treat the infection, and wound care is closely monitored until the infection is controlled.

Hypovolemia and hypothermia are also common complications caused by burn injuries. Hypovolemia can occur due to tissue damage, loss of subcutaneous vascular integrity, and neurohormonal reactions. A burn injury can also disrupt the body's heat regulation mechanisms, leading to hypothermia.

Other serious complications of burn injuries include lung injury, acute kidney failure, hyperglycemia, and septic shock. Therefore, the follow-up and treatment of patients with burn injuries require a multidisciplinary approach.

Urgent intervention, intensive care therapy, and supportive therapies are essential, while there are other important treatment elements such as infection control and nutritional support. (10)

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