**MINISTRY OF HEALTH OF UKRAINE**

**ODESA NATIONAL MEDICAL UNIVERSITY**

**Faculty** *Medicine*

**Department** *Surgery, Radiological Diagnostics, Radiation Medicine, Therapy and Oncology*

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# METHODOLOGICAL RECOMMENDATIONS FOR PRACTICAL CLASSES OF THE ACADEMIC DISCIPLINE

Faculty, course Medical 6th year

Academic discipline Surgery

*(name of the discipline)*

## PRACTICAL CLASSES

*Practical class № 10*

**Topic: "Local and widespread purulent and inflammatory processes of the abdominal cavity and abdomen. Etiology, pathogenesis, clinic, diagnostics and treatment"**

**Approved:**
At the meeting of the Department of Surgery, Radiation Diagnostics, Radiation Medicine, Therapy and Oncology of Odesa National Medical University

**Odesa National Medical University**
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## PRACTICAL CLASSES

*Practical class № 10*

**Topic of the practical class:**
**"Local and widespread purulent and inflammatory processes of the abdominal cavity and abdomen. Etiology, pathogenesis, clinic, diagnostics and treatment"**

1. **Relevance of the topic.**

Acute surgical diseases of the abdominal cavity occupy an important place among all surgical diseases. Acute surgical diseases of the abdominal cavity are diseases that occur suddenly, proceed acutely, are accompanied by pain of varying intensity, are sooner or later complicated by peritonitis if treatment is not started in a timely manner, and have a number of common symptoms. Depending on the causes of their occurrence, all these diseases can be divided into the following **subgroups**:

- acute diseases of inflammatory origin (acute appendicitis, acute cholecystitis, pancreatitis, peritonitis, etc.);
- acute diseases caused by organ destruction (perforative ulcers of the stomach and duodenum, gastrointestinal bleeding, ruptures of the fallopian tube in ectopic pregnancy, ovarian ruptures, intestinal infarctions);
- all types of acute intestinal obstruction;
- diseases of the female genital area;
- diseases of mixed genesis from the influence of enzymes and bacteria (some forms of cholecystitis and pancreatitis);
- open and closed injuries of the chest and abdomen;
- diseases simulating the syndrome of "acute abdomen".

A large number of patients with acute surgical diseases of the abdominal organs, the complexity of diagnosis and treatment, significant mortality - all this determines the relevance of the problem of adequate treatment of patients in this category.

**2. Objectives of the lesson:**

**2.1. General objectives:**

A higher education student must learn to:

1. Identify anamnestic and clinical objective signs of diseases that led to the development of local and widespread purulent-inflammatory processes of the abdominal cavity and abdomen. Level II
2. Anatomical data and physiological properties of the peritoneum. Level II
3. Basic principles of diagnosis and differential diagnosis of acute surgical diseases of the abdominal cavity. Level II
4. Assign an examination plan using laboratory, radiological, endoscopic examination methods. Level III
5. Provide emergency conservative care to patients with acute surgical diseases of the abdominal cavity. Level III

6. Determine indications for surgical intervention and theoretically know the methodology for their implementation. Level II
7. To learn the principles of clinical examination of patients with acute peritonitis, to be able to summarize the obtained data for diagnosis, differential diagnosis, and choice of treatment tactics. Level II.
8. To learn the principles of diagnosis, differential diagnosis, and surgical tactics in the treatment of common forms of acute peritonitis and abdominal abscesses. Level II

**2.2. Educational objectives**:

1. Formation of a professionally significant personality of a doctor.

To emphasize the achievements of the national surgical school of surgeons in the development of modern methods of treatment of local and widespread purulent-inflammatory processes of the abdominal cavity and abdomen.

**2.3. Specific objectives:**

to know:

- Anatomy of the abdominal cavity and abdomen;
- Clinical picture of acute surgical diseases of the abdominal cavity;
- Differential diagnostic signs of acute surgical diseases of the abdominal cavity;
- Methods of instrumental and laboratory examination of patients with acute surgical diseases of the abdominal cavity;
- Conservative and surgical treatment of patients with acute surgical diseases of the abdominal cavity.
- To know the principles of clinical examination of patients with acute peritonitis, to be able to summarize the data obtained for diagnosis, differential diagnosis, and choice of treatment tactics.
- Know the principles of diagnosis, differential diagnosis, surgical tactics in the treatment of common forms of acute peritonitis, abdominal abscesses

**2.4. Based on theoretical knowledge on the topic**.

Be able to (master the techniques):

- Collect a medical history.
- Conduct a clinical examination of patients with acute peritonitis.
- Conduct differential diagnostics between various acute surgical diseases of the abdominal cavity;
- Determine the diagnosis of the disease.
  Formulate a detailed clinical diagnosis according to the ICD classification, justify it on the basis of the differential diagnosis.
- Prescribe conservative therapy for the disease.
- Determine surgical tactics. Formulate indications for conservative and surgical treatment.

- Provide emergency care to a patient with acute peritonitis at the prehospital stage.
- Probe and wash the stomach
- Draw up a medical history with a justification for the patient examination plan and indications for surgical intervention.
- Perform a digital rectal examination.
- Read and interpret the results of general clinical examinations, instrumental examinations, and X-ray results, taking into account age characteristics and semiotics of the disease.
- Assess the wound process and perform dressing taking into account the phase of the wound process, assess drainage discharge.

## 3. Materials for classroom self-study (interdisciplinary integration).

| № | Disciplines | To know | To be able to |
|---|---|---|---|
| | 2 | 3 | 4 |
| | **I. Previous disciplines** | | |
| . | Anatomy | The structure of the abdominal cavity and stomach | To learn to perform adequate sanitation of the abdominal cavity during operations. |
| . | Physiology and pathophysiology | Features of pathogenetic mechanisms of development of endogenous intoxication in peritonitis | To be able to interpret disorders due to endogenous intoxication in patients with acute surgical diseases of the abdominal organs. |
| . | Biochemistry | Biochemistry of inflammation. | To be able to interpret laboratory test data. |
| . | Pharmcology | Mechanism of action of antibacterial drugs and drugs for symptomatic therapy | |
| | **II. Intra-subject integration** | | |
| . | acute inflammatory diseases | Stage of disease course, clinical picture of complications, differential diagnosis | Apply the acquired knowledge when studying etiology and pathogenesis during differential diagnosis and treatment |
| . | acute diseases caused by organ destruction | Stage of disease course, clinical picture of complications, differential diagnosis | Apply the acquired knowledge when studying etiology and pathogenesis during differential diagnosis and treatment |

| | | |
|---|---|---|
| . all types of acute intestinal obstruction | Stage of disease course, clinical picture of complications, differential diagnosis | Apply the acquired knowledge when studying etiology and pathogenesis during differential diagnosis and treatment |
| . Diseases of the female genital area | Know the features of the clinical picture and differential diagnosis | Apply the acquired knowledge when studying etiology and pathogenesis during differential diagnosis and treatment |
| diseases of mixed genesis from the effects of enzymes and bacteria. | Know the features of the clinical picture and differential diagnosis | Apply the acquired knowledge when studying etiology and pathogenesis during differential diagnosis and treatment |
| . open and closed injuries of the chest and abdomen, diseases simulating the "acute abdomen" . syndrome. | Know the features of the clinical picture and differential diagnosis<br>Know the features of the clinical picture and differential diagnosis | |

**4. Topic content.**

**Local and widespread purulent and inflammatory processes of the abdominal cavity and abdomen. Etiology, pathogenesis, clinic, diagnostics and treatment**

**Diagnostics**

**Diagnostics** of acute surgical diseases of the abdominal cavity is urgent. At the prehospital stage of examination, acute disease of the abdominal cavity is not always possible to recognize accurately and quickly enough. Continuing to observe the patient for a long time to clarify the diagnosis is unacceptable. This inevitably leads to complications, including peritonitis, necrosis of organs and tissues, and irreparable blood loss.

At the prehospital stage, the family doctor does not have a number of additional research methods, without which clarification of the diagnosis is extremely difficult and impossible; it is impossible to provide the patient with the full range of necessary assistance even before the diagnosis is clarified.

You cannot achieve clarification of the diagnosis at any cost. It is enough to establish that the patient has an acute surgical disease of the abdominal cavity and requires urgent hospitalization.

It is in such cases that the not very precise, not very specific, but useful definition of "acute abdomen" comes to the rescue.

It is distinguished by its conciseness and expressiveness, carries information about an acute surgical disease, in which immediate hospitalization in the surgical department is indicated.

The term "acute abdomen" is not a diagnosis, but only a collective concept and at the same time a signal for action, and for specific, decisive and responsible action - to direct the patient to the hospital.

It is necessary to clearly imagine that delay in this condition is like death.

At present, it is precisely established that the results of treatment of patients with an acute abdomen depend on the timing of their admission to the surgical department.

One of the constant signs of an acute abdomen is pain, which can occur from the very beginning of the disease or develop gradually, slowly, reaching a maximum within many hours, but more often occurs suddenly (with perforated gastric and duodenal ulcers, some types of intestinal obstruction, acute pancreatitis).

G. Mondor wrote that artificial pain relief does not eliminate the causes of its occurrence, and temporary pacification of the patient only postpones targeted treatment and brings enormous and irreparable harm.

The decision to hospitalize a patient with an acute abdomen is the only correct one.

It is necessary to know firmly what should not be done and what supposedly useful, but dangerous help can lead to.

Following the decrease in the patient's condition, the patient refuses hospitalization and surgery and only after some time after the patient's condition has returned and the condition worsens, he again seeks help, which turns out to be late.

Taking laxatives brings enormous harm.

With nausea, vomiting, and bursting pains, gastric lavage is performed in the epigastric region. However, it is impossible to probe and wash the stomach until the diagnosis is clarified.

In some cases, such manipulations lead to a sharp increase in intra-abdominal pressure and increased bleeding (ectopic pregnancy, ovarian rupture), in others - contribute to the rupture of the inflamed organ (acute appendicitis), the progression of peritonitis (perforating ulcers of the stomach and duodenum).

It is impossible to prescribe a hot heating pad to the abdomen until the diagnosis is fully clarified, because bleeding or the spread of the inflammatory process may increase. A patient with an acute abdomen must be provided with rest, it is impossible to allow him to move independently, he must be transported lying down, on his back or on his side.

**Anamnesis**

The **anamnesis** of the disease is of great importance for diagnosis. It is necessary to clarify the time of onset of the disease and find out not only the day, but, if possible, the hour and even the minute. Specify the nature and primary localization of pain, irradiation.

Dizziness, fainting, prolonged loss of consciousness, vomiting (single, multiple, profuse or scanty), the nature of vomit (food, bile, blood, stagnant contents, "fecaloid", etc.), nausea, belching, hiccups should not be ignored.

Special attention should be paid to the characteristics of the stool, the nature of the feces (color, consistency, presence of blood), the passage or retention of gases, urination (normal, accelerated, difficult, complete retention, pain during urination).

From the anamnesis, it is necessary to find out whether there have ever been similar attacks, whether he was hospitalized, what is the diagnosis; whether he has undergone surgical operations and which ones, for women - the time of the last menstruation, the number of births, abortions, miscarriages, gynecological diseases.

**Objective examination of the patient.**

1. General condition of the patient: a) degree of severity of the condition (extremely severe, very severe, severe), b) behavior (calm, groaning from pain), c) typical facial appearance, "Hippocrates mask", forced position - on the back, half-sitting, with legs drawn to the stomach, d) type of physique and degree of compliance, e) condition of the skin (color, pallor, pale blush), turgor (normal, reduced), humidity (cold sweat, dry), e) body temperature (increase, decrease - in degrees), g) condition of the cardiovascular system (decreased HELL, tachycardia, pericardial friction noise) and lungs (pleural friction noise, presence of effusion in the pleural cavity, high diaphragm), h) tongue (dry, coating), i) state of consciousness.

2. Examination of the abdominal organs:

Examination: shape, retraction, swelling, asymmetry, degree of participation in the act of breathing (does not participate, participates superficially, does not participate in the right, left or lower half of the abdomen).

Palpation: soreness (localization and degree of severity), tension of the abdominal muscles (localization, degree of severity), symptoms of peritoneal irritation - Shchetkin-Blumberg and Mendel (their localization and degree of severity), presence and size of the alpinic infiltrate (delineate the borders on the skin), its localization, nature of the surface, mobility, palpation of the liver and spleen, their borders, surface, consistency, edge, soreness.

percussion: high tympanitis, dullness of percussion sound over the infiltrate with limited peritonitis, dullness of percussion sound in gentle places, liver size according to Kurlov (increasing the size of hepatic dullness upwards).

Auscultation: peristaltic sounds, their degree of expressiveness, absence of peristalsis, "splashing sound" and "falling drop sound".

3. Vaginal or rectal examination - overhang of the posterior vaginal fornix, swelling in the lumen of the anterior wall of the rectum, bimanually, the infiltrate in the small pelvis is determined, its size, mobility, soreness, the presence of fluctuations, relationship with the organs of the small pelvis.

**Additional investigations**

1. Laboratory a) complete blood count, leukocyte index of intoxication - LII; b) biochemical - glucose, amylase, bilirubin, transaminases, residual nitrogen, urea, blood

creatinine, lactic acid, medium weight molecules, coagulogram, protein and its fractions, BUN), c) general urinalysis, daily diuresis. 2. X-ray: a) high standing of the diaphragm, limitation of its mobility, b) Kloiber's bowl. 3. Instrumental examination: a) laparocentesis with bacteriological examination of effusion and the presence of pancreatic enzymes, b) laparoscopy. 4. Bacteriological examination of abdominal effusion with determination of microflora sensitivity to antibiotics.

**ACUTE APPENDICITIS**

Acute appendicitis is a nonspecific inflammation of the ileum caused by gram-negative microbes such as streptococci, staphylococci, enterococci, E. coli, etc.

It ranks first among acute surgical diseases of the abdominal cavity (58-72%). 99% of patients undergo surgery. The overall and postoperative mortality rate is the lowest, and the number of patients dying annually with this disease is higher than with perforated ulcer.

The main cause of mortality is delayed surgery, and the main cause of delayed surgery is late hospitalisation.

As for the main reasons for delayed hospitalisation, the first place is occupied by late recognition of acute appendicitis and refusal of patients to be admitted. It would seem

it would seem that the main issues of diagnosis and treatment have been resolved in our country, but doctors still make many diagnostic and tactical errors at different stages, which negatively affect the results of treatment of this disease.

I.I. Grekov called appendicitis due to the extraordinary variety of clinical manifestations

‘a chameleon-like disease’.

Naturally, diagnostic errors are possible at all stages of the patient's examination. The position of the cecum and ileum is variable.

Most often, the appendix is located in the right iliac region, but in the case of a mobile cecum, it can be located in different parts of the abdominal cavity (under the liver, in the pelvis, in the left iliac region, and occupy a retrocecal position - behind the cecum).

According to V.Kolesov's **classification**, there are 4 forms of acute appendicitis:

- appendicular colic;
- simple (superficial or catarrhal) appendicitis;
- Destructive appendicitis: a) phlegmonous, b) gangrenous, c) perforated (perforating) gangrenous;
- Complicated appendicitis: a) appendicular infiltrate, b) appendicular abscess, c) spilled purulent peritonitis, d) other complications (sepsis, pylephlebitis).

Appendicular colic occurs in 3% of cases and is characterised by minor inflammatory phenomena that can resolve or become severe.

At the pre-hospital stage, in case of mild symptoms of acute appendicitis, the patient should not be kept for observation - hospitalisation is necessary.

The rupture of the cecum is a consequence of a violation of the wall integrity under the influence of a necrotic process. It can occur under the influence of a foreign object

that has entered the lumen (fish bone, shell, roundworm, etc.). The pathological process is often not limited to the appendix, but extends beyond it (limited or diffuse peritonitis).

One of the types of complicated acute appendicitis is appendicular infiltrate

- is an inflammatory conglomerate consisting of an inflamed appendix and its mesentery, the cecum and part of the ascending colon, the great omentum and the loops of the small intestine. Appendicular infiltrates on the cob are very dense, inactive, occupy the entire right iliac region, and resolve very slowly. In some cases, the infiltrates are small, mobile, and resolve quickly.

The infiltrate can increase in size, soften and threaten to break through into the abdominal cavity, leading to spilled peritonitis.

A minor complication of destructive appendicitis is limited abscesses (appendicular abscesses) of various localisations (ileal, intestinal, pelvic, subhepatic, subdiaphragmatic, retrocecal).

Very rarely, intrahepatic abscesses (pylephlebitis) are observed due to the transfer of infection from the appendix through the veins.

**Clinic of acute appendicitis**

Abdominal pain in acute appendicitis is caused by irritation of nerve endings located in the wall of the peritoneal appendage covering it and, in the mesentery, and the more severe the inflammation, the more severe the pain. The pains are diverse: more often they appear suddenly and are permanent.

Cramping, cutting, stabbing, girdling pains are atypical for appendicitis. In the first 6-12 years from the onset of the disease, pain is most often localised in the right iliac region. Irradiation to the right thigh is observed in 10% of patients.

The onset and final localisation of pain in the right iliac region is a characteristic symptom of acute appendicitis.

In typical cases (30-35 % of patients), the disease begins with moderate pain in the epigastrium, which shifts to the right iliac region after 4-6 years (Kocher-Wolkowitz symptom). There may be a prolonged epigastric phase, which should be noted.

Nausea affects 60 % of patients, in 40-45 % it is accompanied by vomiting, which is reflexive and does not bring relief.

Destructive forms of acute appendicitis are accompanied by repeated vomiting.

A feeling of stomach fullness and heaviness in the abdomen is disturbing.

One of the typical variants of the severe toxic form of appendicitis is a violent cob of the disease, severe nausea, repeated vomiting, abdominal distension and a feeling of overflow in the epigastric region.

Intestinal dysfunction is expressed in stool retention or loose stools. Sometimes there is gas retention.

Urinary disorders occur in the pelvic or retrocecal location of the process.

The general condition of patients in the initial period is satisfactory.

In destructive forms, the condition is severe. At the onset of the disease, patients prefer to lie on their backs with their legs bent at the hip and knee joints, or on their right side with their legs brought to the abdomen.

Body temperature ranges from normal to 39 °C and above. In elderly and old patients, body temperature is normal or subfebrile.

In 45-50 % of patients, the pulse does not increase, in 40 % - up to 100 beats per 1 min and in 10-15 %- more than 100 beats per 1 min.

Increased pulse rate corresponds to increased temperature, and a discrepancy between them indicates intoxication and a destructive form of appendicitis (peritonitis).

In 60 % of patients, the tongue is moist but coated, in 10 % - dry. The abdomen of patients is often unremarkable.

Auscultation of the abdomen reveals intestinal noises.

Percussion of the abdomen reveals dullness in the low abdominal areas in case of advanced peritonitis with a significant amount of exudate.

Abdominal palpation should be performed with warm hands, starting farther away from the place where the patient says he feels pain.

It is necessary to determine whether there is pain and where it is localised, or whether there is tension in the abdominal wall.

Respiratory movements and coughing cause an increase in pain in the right hypochondrium.

region.

The main symptoms characteristic of the pathology of the celiac

process:

- Shchetkin-Blumberg symptom (80-85 %) - a sharp increase in pain at the moment of rapid removal of the hand from the abdominal wall with moderate pressure in the right hypochondrium;
- Rovzing's symptom (60-70 %) - pain in the right hypochondrium when pushing with a vise on the descending colon;
- Voskresensky's symptom (sliding symptom) - the appearance of cutting pain in the right hypochondrium when the palm of the hand is quickly drawn along the anterior abdominal wall from the right hypochondrium down to the right hypochondrium;
- Sytkovsky's symptom - the appearance of pain in the right hypochondrium when the patient lies on the left side.

Blood changes in acute appendicitis are expressed in an increased number of leukocytes with a shift in the leukocyte formula to the left. Hyperleukocytosis is noted

more often in severe appendicitis and in children. Body temperature measurement and rectal temperature measurement are mandatory (a difference of 1 °C should alert the doctor).

The clinic of the typical location of the cecum is described, but atypical positions are also found: retrocecal, pelvic, and subhepatic.

The retrocecal position of the process is characterised by lancing pain radiating to the right thigh. The right lateral abdominal wall is slightly tense and painful. The other classical symptoms listed above are not pronounced.

The pelvic location of the cricoid process has some peculiarities. Pain occurs above the right inguinal ligament, in the lower part of the right hypochondrium; rapid and painful urination, frequent stools with mucus and blood are indicative of bladder and rectum involvement in the inflammatory process. When the uterus and appendages are involved, pain in the lower abdomen is noted.

In case of subhepatic localisation of the process, stomach pain first appears, then localises in the right hypochondrium, where tension is detected. There is no radiation of pain to the right shoulder and scapula. Positive Shchetkin-Blumberg, Rovzing,Sytkovsky symptoms.

Various clinical manifestations of acute appendicitis are explained not only by different positions of the appendix, but also by the patient's age.

**Age-related features of acute appendicitis**

Acute appendicitis in children can develop at any age, more often after 5 years of age.

It occurs with pronounced general and local symptoms (fever up to 38-39 °C, tachycardia, discrepancy between heart rate and temperature).

Abdominal tenderness and tension in the right hypochondrium, positive Shchetkin-Blumberg symptom. Rapid progression of the disease, a tendency to spread the pathological process.

In older children, it is less acute.

Acute appendicitis in the elderly is characterised by an indolent course, mild local and general symptoms, which is associated with reduced reactivity.

Appendicitis occurs without a pronounced painful attack, with a normal temperature, without leukaemia and tachycardia. The tension of the anterior abdominal wall muscles and the symptom of peritoneal irritation are not sufficiently pronounced, only local soreness in the area of the appendix is determined.

Acute appendicitis in pregnant women after 2-3 months of pregnancy is peculiar due to a change in the position of the cecum caused by an enlarged uterus. Due to the upward displacement of the cecum, local symptoms of appendicitis are localised higher, with larger gestational ages - in the right hypochondrium.

Muscle tension is mild. A pregnant woman should be referred to the gynaecological department with a mandatory examination by a surgeon.

Acute appendicitis must often be differentiated from diseases of the kidneys, uterine appendages, ectopic pregnancy, acute cholecystitis, pancreatitis, gastritis, enterocolitis, food toxicity and other diseases. Diagnosis is difficult in children, women and the elderly, with its atypical location. The slightest suggestion of appendicitis is enough to immediately refer the patient to the surgical department.

**Treatment of acute appendicitis**

The generally recognised method of treating acute appendicitis is an urgent appendectomy.

The main task facing the family doctor is to establish or assume acute appendicitis and refer the patient for hospitalisation to the surgical department. The patient is prohibited from eating and drinking and from taking painkillers.

Causes affecting mortality: late hospitalisation due to untimely referral for medical care, and therefore late surgery (later than 24 hours).

A contraindication to urgent surgery is the presence of a dense, immobile appendicular infiltrate, which is formed on the 2nd-4th day after the onset of the disease.

**Postoperative treatment of acute appendicitis**

In case of uncomplicated appendicitis after appendectomy, patients receive antibacterial therapy. During the first 2 days, the patient is given broth, kefir, liquid porridge, and jelly. On the 3rd-4th day - mashed soups; on the 5th day - steamed cutlets, then transferred to a common diet. Patients are allowed to get out of bed on the 2nd day.

**ACUTE PANCREATITIS**

Acute pancreatitis is an acute disease of the pancreas, which is based on destructive and inflammatory processes caused by autolysis of the gland tissues by its own enzymes. Currently, the proportion of acute pancreatitis among other surgical diseases of the abdominal cavity is 10-12%. Acute pancreatitis is second only to acute appendicitis and acute cholecystitis in terms of frequency. The overall mortality rate in acute pancreatitis is 5-10% and reaches 35% in case of complications.

**Etiology and pathogenesis of acute pancreatitis**

Factors that can be considered causative for the development of acute pancreatitis can be presented in the form of several syndromes.

- Pancreatic hyperinfection (food, alcohol, drug).

- Pancreatic ductal hypertension (spasm of the large duodenal papilla, papillary edema in gastroduodenitis, gallstones); duodenal diverticula; increased intraduodenal pressure in duodenostasis, intestinal paresis, peritonitis, intestinal trauma (postoperative pancreatitis).

- Pancreatic ischaemia (vascular pathology).

- Toxic effects on the pancreas (nitro paints, varnishes, solvents, scorpion venom, medicines (corticosteroids, hypothiazide), toxic-allergic and infectious-toxic effects (more often in children).

- Injuries of the pancreas.

- Endogenous factors (congenital and acquired): increased viscosity of pancreatic juice (during pregnancy, chronic alcoholism, disorders of water and electrolyte metabolism); allergic status; chronic liver diseases (hepatosis, hepatitis, cirrhosis).

According to the most common enzymatic theory of the pathogenesis of acute pancreatitis, the disease is based on damage to the pancreatic cells under the influence of a particular factor.

**Classification of acute pancreatitis**

There are 4 forms of acute pancreatitis:

- acute edema of the pancreas;

- acute haemorrhagic pancreatitis;
- acute pancreatic nephrosis;
- acute cholecystopancreatitis.

The main structural feature of the classification should be the extent of pancreatic necrosis.

- Edematous (interstitial) pancreatitis (85 % of cases) is characterised by a mild course. The clinical picture is dominated by tissue edema.
- Small-focal fatty necrosis of the pancreas (moderate severity). Complications (peritonitis, jaundice), mortality - 6.7%.
- Large-focal widespread necrosis of the gland with a pronounced haemorrhagic component (severe condition). Initially, pancreatogenic shock develops, followed by purulent complications. The mortality rate is 36.4%.
- Total-subtotal pancreatic necrosis (mortality rate - 80%).

**Clinical picture of acute pancreatitis**

The clinic of acute pancreatitis depends on the phase, location and extent of pathological changes, the degree of enzyme activity, and complications.

The disease occurs at any age. Mostly women are affected (90%).

According to the literature, acute pancreatitis in children is a rare disease.

The most characteristic signs of acute pancreatitis are pain, vomiting, and dynamic intestinal obstruction.

Abdominal pain, a constant and leading symptom, can be very severe and can lead to shock. More often it is constant, dull or cutting. The pain is localised high in the epigastric region and is of a girdling nature; in some patients, the pain is felt in the right hypochondrium. Due to severe pain, patients moan, even scream and take different positions in bed. Some lie motionless, others roll around. Severe abdominal pain is associated with the involvement of the abdominal nerve plexus in the pathological process. The patient's radiation is varied: to the lower back on the right or left, to the back and spine, to the chest, to the heart, etc.

Vomiting is a common symptom. It can be repeated, uncontrollable, painful, frequent and does not bring relief. Bile is present in the vomit. In severe pancreatitis, the vomit looks like ‘coffee grounds’, which may indicate a poor prognosis.

Painful hiccups and belching with bitterness.

The general condition is severe. The skin is pale, often there is jaundice of the skin and sclerae. In some patients, cyanosis appears early, sometimes with a ‘marble’ pattern, which is associated with impaired microcirculation.

Mondor's symptom (purple spots on the skin of the face and trunk).

Halstead's symptom (cyanosis of the abdominal skin), Grieve-Turner's symptom (cyanosis of the lateral abdominal walls). In more severe cases, intoxication and shortness of breath increase. The skin is covered with fine sweat, the body temperature rises, the pulse increases, the tongue is dry, blood pressure gradually decreases, the abdomen is distended, especially in the supra-abdominal region, due to paresis of the transverse colon.

Abdominal distention is observed in case of development of spilled peritonitis. The abdominal wall is involved in breathing. Coughing impulse always increases pain in the stomach. Palpation of the anterior abdominal wall is painful in the supra-abdominal region. The area of tenderness and stiffness corresponds to the projection of the pancreas 6-7 days from the pleural cavity.

**Differential diagnosis of acute pancreatitis**

The possibility of correct and timely diagnosis of acute pancreatitis at the pre-hospital stage lies in the knowledge of the peculiarities of pancreatic colic and changes in symptoms.

To properly assess diagnostic errors in the prehospital stage, they should be divided into 2 groups:

- Surgical errors;
- therapeutic errors.

If patients with an erroneous diagnosis of acute pancreatitis and suffering from other acute surgical diseases are referred to the surgical department, such an error is not gross, because the diagnosis is quickly clarified and they receive timely care.

Patients with the same therapeutic errors, suffering from acute therapeutic diseases (myocardial infarction, pleuropneumonia, stroke), when admitted to the surgical department, where the diagnosis of therapeutic diseases is less perfect and untimely, do not receive timely treatment, such an error is gross.

Acute pancreatitis should be differentiated from acute cholecystitis, acute gastritis, perforated gastric or duodenal ulcer, intestinal obstruction, renal colic, and cardiovascular disease.

Acute cholecystitis, unlike acute pancreatitis, is characterised by pain in the right hypochondrium, radiating to the right shoulder blade, shoulder, fever, severe tenderness and tension in the right hypochondrium. Acute cholecystitis is usually not accompanied by rapidly increasing symptoms of intoxication and collapse. Symptoms of paralytic intestinal obstruction appear gradually with the development of peritonitis.

An attack of biliary colic is characterised by the absence of abdominal changes that are expressed in acute pancreatitis (painfulness during palpation in the pancreas, stiffness of the anterior abdominal wall muscles, intestinal paresis).

The pain radiates to the right shoulder, scapula, and is relieved by drugs. In acute pancreatitis, drugs do not affect the intensity of pain.

Acute gastritis is accompanied by a less severe pain syndrome than pancreatitis, although the pain is identical in its location. Acute gastritis occurs when taking caustic substances, alcohol surrogates or spicy foods, accompanied by severe heartburn, belching and vomiting, after which there is a temporary improvement. In case of acute pancreatitis, deterioration is observed after vomiting.

Chronic gastroduodenitis is accompanied by less intense, inconsistent pain. They occur either at night or on an empty stomach.

In contrast to perforated ulcer, acute pancreatitis occurs with rapidly increasing symptoms of intoxication and repeated vomiting. Patients have no history of ulceration

and pneumoperitoneum. On X-ray examination, perforation is characterised by the presence of free gas under the dome of the diaphragm.

Acute intestinal obstruction

Mechanical intestinal obstruction is characterised by cramping pain, repeated vomiting of gastric and then intestinal contents in later stages, severe abdominal distention, and Valyachy and Sklyarov symptoms. Symptoms of acute intestinal obstruction in acute pancreatitis are functional and disappear quickly with conservative treatment. On X-ray examination, mechanical intestinal obstruction is characterised by the presence of Kloiber's bowls.

Renal colic is characterised by severe pain in the lumbar region, radiating to the right side of the abdomen, groin, and rapid, difficult urination. During an attack, patients cannot find a place in bed. The urine test shows protein, fresh red blood cells in large numbers.

It is very difficult to differentiate mesenteric vascular thrombosis from acute pancreatitis due to the presence of general symptoms of intoxication, cardiovascular disorders, intestinal paresis and severity of pain. In case of thrombosis, loose stools with blood admixture may be observed, and rarely vomiting. The pain is widespread, there is uniform abdominal distention, no local tenderness on palpation in the projection of the pancreas. Acrocyanosis and marbling of the skin are characteristic.

In these cases, it is necessary to find out whether the patient has heart disease (heart disease, atrial fibrillation, thrombophlebitis of the lower extremities, etc.)

Acute pancreatitis

It can be accompanied by severe pain in the supraperitoneal region, but not by bile vomiting, local abdominal pain in the projection of the pancreas, severe intestinal paresis and abdominal muscle stiffness. The ECG data indicate severe cardiac dysfunction.

Diagnostic difficulties arise in elderly patients. In them, acute pancreatitis occurs against the background of concomitant diseases, cardiovascular diseases, chronic diseases of the digestive system contribute to its development. These diseases can contribute to the occurrence of not one, but several acute abdominal diseases (a combination of acute pancreatitis of vascular origin and mesenteric vascular thromboembolism with intestinal infarction).

**Laboratory diagnostics of acute pancreatitis**

- Complete blood count and urine test.
- Determination of blood and urine amylase.
- Blood sugar.
- Bilirubin.
- Total protein.
- Sulemic test.
- Determination of the activity of aminotransferases and alkaline phosphatase in blood.
- Blood coagulation system.
- Determination of blood calcium.

- Determination of urea, creatinine.
- Determination of electrolytes.
- Study of the acid-base state

**Instrumental diagnostics**

- X-ray (chest X-ray), abdominal cavity to detect indirect signs of pancreatitis (high standing and restriction of diaphragm mobility, pneumatic small intestinal arches on the left side at the level of the II-III lumbar vertebrae, horizontal fluid level due to duodenostasis).
- ERCP (endoscopic retrograde cholangiopancreatography).
- ULTRASONOGRAPHY.
- Endoscopic techniques: laparoscopy (collection of exudates for examination).
- Selective angiography of the abdominal trunk.
- Computed tomography.

**Treatment of acute pancreatitis**

The main treatment for acute pancreatitis is conservative. Conservative treatment is aimed at creating maximum functional rest, inactivation of pancreatic enzymes, detoxification, normalisation of cardiovascular and central nervous system functions, respiration, and disorders of water-salt, carbohydrate and protein metabolism.

I. Inhibition of pancreatic secretion:

local hypothermia (cold on the epigastric area);

- starvation for 3-5 days with a ban on ingestion of not only food but also water;
- suctioning of gastric contents;
- atropinisation (0.5 mg 2-3 times a day); administration of alkalising agents through an almagel tube; intravenous cimetidine - an H2 receptor antagonist;
- intragastric hypothermia (gastric lavage with cold water).

II. Pain management:

- blockade of the abdominal nerves or paraneural block (0.25% novocaine solution);
- nitroglycerin under the tongue, antispasmodics (2% solution of papaverine, no-shpa, halidor, 0.2% solution of platyphylline);
- narcotic analgesics (promedol), droperidol - 2.0 ml for 4-6 h. Morphine is contraindicated because it has a vagal effect, causes spasm of the large duodenal papilla, which leads to stagnation of bile and pancreatic juice;
- non-narcotic analgesics (analgin 50% solution - 4-5 ml, baralgin - 5 ml, dimedrol 1% solution - 2.0 ml);
- novocaine therapy (0.5% solution - 20-40 ml of novocaine intravenously at once or 0.25% solution with the addition of 5% glucose, 400 ml slowly).

III. Antienzyme therapy.

- Antienzyme therapy consists of the administration of drugs that block trypsin and kallikrein (trazylol, contrical, gordox), or drugs that inhibit protein synthesis in the gland and enzyme production (cytostatics - 5-fluorouracil, fluorafur):
- - Contrical 100,000-120,000 units in saline 3-4 times a day;
- - Gordox. Initially, 500 thousand units should be administered slowly intravenously, and then 5 thousand units every hour;
- - epsilonaminocaproic acid (EACA) is a potent inhibitor of fibrinolysis, which has an inhibitory effect on the activation of plasminogen into plasmin. Administer a 5% solution intravenously in an amount of 200-300 ml.

IV. Correction of water and electrolyte balance:

- - Ringer's solution up to 2000 ml per day;
- - 5% glucose solution, 1500 ml + insulin;
- - 10 ml of 10% calcium chloride solution;
- - dry plasma, albumin;
- - haemodesis.

V. Antishock therapy:

- Rheopolyglucin or polyglucin;
- 10% albumin solution;
- 125 ml of hydrocortisone intravenously;
- norepinephrine, 2-3 mg per day.

VI. Antiallergic therapy: pipolphene, dimedrol.

VII. Anti-inflammatory therapy: tetracycline drugs, penicillin.

VIII. Symptomatic therapy.

In case of failure of conservative treatment within 24 hours, laparoscopic drainage of the abdominal cavity is performed.

In the treatment of destructive pancreatitis, cytostatics are used (5-fluorouracil, average daily dose - 350 mg, in mesenteric vascular thrombosis 1-4 days; fluorafur - 4% solution, 10 ml).

The high mortality rate and the development of various complications in patients with acute pancreatitis explain the urgency of finding new methods of treatment.

Currently, sandostatin has been proven to be highly effective. The latter is a synthetic octapeptide that is a derivative of the natural hormone somatostatin, produced by the anterior pituitary gland and the digestive tract. The drug inhibits the secretion of peptides such as gastrin, glucagon, insulin, cholecystokinin, vasoactive integrin peptide, pancreatic polypeptide, as well as pancreatic enzymes (amylase, lipase, trypsin), reduces gastric acidity and the volume of pancreatic secretions.

Sandostatin has a longer half-life, which allows for an extended dosing interval. In acute pancreatitis, sandostatin reduces the intensity of pain (it disappears on the 2nd day of treatment) and reduces the incidence of

complications, especially when high doses of the drug (600-1500 mcg per day) are used.

Clinical data on the reduction of edema of the pancreas and surrounding tissues, disappearance of pancreatic ascites, and activation of the retinal endothelial system of the liver and spleen under the influence of sandostatin suggest the presence of a cytoprotective effect of this drug.

Treatment with high doses of sandostatin (200-500 mcg 3 times a day) significantly improved the clinical course of pancreatitis and reduced the number of deaths (8.3% vs. 14.8%).

**Surgical treatment of acute pancreatitis**

Indications for early surgery:

- increasing signs of peritonitis;
- increasing jaundice and signs of gallbladder destruction;
- no effect of the therapy within 24-48 hours. Early surgeries are performed in the first 2-3 days of the disease.

Delayed surgery (2-3 weeks after the onset of the disease)

Indications

- lack of effect from conservative treatment;
- clinical and radiological signs of septic destruction of the gland;
- arrosive bleeding from the pancreaticoduodenal vessels.

The scope of surgical treatment may vary depending on the morphological changes in the gland and the extent of the process.

Basically, the operations are limited to drainage of the choledochal pouch, abdominal cavity, and biliary tract decompression.

**Complications:**

Early complications of acute pancreatitis:

- enzymatic spilled peritonitis;
- pleurisy and pericarditis;
- serous haemorrhagic pancreatitis;
- early acute ulcers, gastric and duodenal erosion;
- early thrombosis of abdominal vessels;
- myocardial infarction;
- pulmonary oedema.

Late complications of acute pancreatitis:

- abscesses of the gland;
- purulent parapancreatitis, omentobursitis, peritonitis;
- abdominal abscesses;
- abscesses of the liver, lungs;
- purulent pleurisy;
- sepsis;
- fistulas of the gland;
- late erosions and ulcers of the gastrointestinal tract;

- arrosive bleeding.

**ACUTE CHOLECYSTOPANCREATITIS**

Acute cholecystopancreatitis is a combination of two mutually aggravating processes cholecystitis and acute pancreatitis. In 85% of cases, cholecystopancreatitis is a complication of gallstone disease, and in 15% of cases it is secondary, enzymatic cholecystitis.

**Acute cholecystopancreatitis clinic**

In acute cholecystopancreatitis, the symptoms of gallbladder and bile duct damage come to the fore. Pancreatitis is indicated by a high prevalence of pain in the epigastrium, right hypochondrium, and right-side canal, and by the encircling pain. The greatest soreness is noted in the right hypochondrium, painful vomiting and early jaundice are characteristic.

Tapping on the right hypochondrium is accompanied by sharp pain.

**Diagnosis of acute cholecystopancreatitis**

Diagnosis is based on anamnesis, clinical examination, and laboratory data.

**Treatment of acute cholecystopancreatitis**

Indications for urgent surgical treatment:

- severe peritonitis;
- presence of a clinic of destructive cholecystitis or pancreatitis;
- failure of conservative treatment.

**ACUTE CHOLECYSTITIS**

Among the main forms of acute abdomen, acute cholecystitis ranks second in terms of the total number of patients, second only to acute appendicitis. Most patients are women (90%). The age varies, with 80% being over 30-40 years old. It is rarely observed in children.

The term ‘acute cholecystitis’ should be understood as a situation when a patient with chronic (calculous) cholecystitis has an attack with a characteristic clinical picture for the first time in his/her life or suffering from chronic (calculous) cholecystitis. Cholecystitis combines all inflammatory diseases of the gallbladder and bile ducts. In the development of acute cholecystitis, the leading role is played by the stagnation factor, aggravated by the addition of infection against the background of a violation of the chemical composition of bile. The cause may be acute obstruction of the bile duct (a stone or its kink), swelling of the mucosa, foreign bodies (roundworms), and the consequence is the development of hypertension in the bladder.

**Classification of acute cholecystitis**

I. Uncomplicated cholecystitis:

- catarrhal (simple) cholecystitis (calculous or stone-free), primary and exacerbation of chronic cholecystitis;
- destructive (calculous or calculus-free), primary or exacerbation of chronic cholecystitis: phlegmonous, phlegmonous-ulcerative, gangrenous.

II. Complicated cholecystitis:

- Obstructive (dropsy, phlegmon, empyema, gangrene of the bladder);
- perforating (local or spilled peritonitis);
- acute, complicated by bile duct damage: a) choledocholithiasis, cholangitis; b) choledochal stricture, papillitis, stenosis of the fauces nipple;
- acute cholecystopancreatitis;
- acute cholecystitis complicated by biliary peritonitis.

**Acute cholecystitis clinic**

Acute cholecystitis clinic depends on changes in the gallbladder, the duration of the disease, the presence of complications and the body's reactivity. Involvement of the organs surrounding the gallbladder (liver, stomach, bile ducts, pancreas, duodenum) in the inflammatory process affects the clinical picture and course.

Acute cholecystitis is violent, occurs suddenly, after a dietary error (eating fatty, fried, salty foods, etc.), but it is preceded by long-term cholelithiasis, chronic calculous cholecystitis. The disease begins with an attack of pain in the right hypochondrium, radiating to the right shoulder and shoulder blade, to the right supraclavicular region.

Vomiting is observed in 60% of patients and is most often repeated. Abundant or scanty vomiting never brings relief. A feeling of bitterness in the mouth is characteristic. The body temperature rises to 38-39 °C, sometimes with chills.

In the elderly and senile, severe destructive cholecystitis can occur with a slight increase in body temperature and leukocytosis.

The general condition of patients suffers significantly. Appetite disappears, aversion to any food, thirst, and dry mouth appear. These phenomena are expressed in destructive forms of acute cholecystitis with various complications.

During an attack, the patient is lying on the right side, but can take a different position.

The pulse in simple cholecystitis increases in accordance with body temperature, in destructive and perforative cholecystitis with the development of peritonitis, tachycardia up to 100-120 beats per 1 min is noted.

Blood pressure is most often low and can be sharply reduced only in case of painful shock developing at the height of an acute cholecystitis attack.

Breathing is slightly rapid and shallow. The tongue is dry, covered with a white or brown coating.

Abdominal palpation reveals sharp tenderness in the right hypochondrium and tension of the anterior abdominal wall. It can be expressed in different ways, but never reaches the state of a ‘board-shaped abdomen’. In 60 % of patients, it is possible to feel

a slightly enlarged liver protruding from under the edge of the rib arch, and in 85-92 % - an enlarged painful gallbladder.

Palpation of the gallbladder in acute cholecystitis may be impeded by

- intense tension of the abdominal muscles;
- excessive development of subcutaneous tissue;
- low position of the rib arch.

The Ortner's symptom (painfulness when tapping the right rib arch) is important.

Murphy's symptom - the patient cannot take a deep breath with deep palpation in the right hypochondrium.

Mussi's symptom (phrenicus symptom) - painfulness on palpation in the right supraclavicular region at a point located between the legs of the right sternocleidomastoid muscle.

In the initial stages of the disease, an enlarged, distended gallbladder can be detected. In severe destructive cholecystitis, there is a sharp tenderness on superficial palpation in the right hypochondrium. Blood tests show leukocytosis, and hyperbilirubinaemia in case of jaundice.

Acute cholecystitis can be very severe with rapid development of gangrene and perforation of the gallbladder within 1-2 days, especially in the elderly and senile.

Difficulties in recognising acute cholecystitis in elderly and senile patients are due to both altered reactivity and the presence of comorbidities. While in the whole group of patients with acute cholecystitis, sharp abdominal pain was noted in 68 %, in the elderly - in 7.5 % of cases. Dull abdominal pain is observed in 93 % of cases.

Concomitant diseases that complicate diagnosis are diverse: cerebrovascular disorders, diseases of the cardiovascular system (heart disease, myocardial infarction, heart aneurysm). Biliary colic usually develops when there is a gallstone in the gallbladder. In 74% of cases, biliary colic ends in the development of inflammation.

The cause of biliary colic is acute obstruction of the bile outflow from the gallbladder by a stone wedged into the bile duct. Disruption of the bile outflow causes contraction of the gallbladder muscles and causes severe pain.

There are pains of varying intensity in the right hypochondrium, radiating to the right shoulder, shoulder blade, repeated vomiting, which does not bring relief. An enlarged gallbladder is often detected palpably in a soft abdomen. The absence of the gallbladder is associated with sclerosis of its walls. The temperature is normal.

A combination of acute cholecystitis and mechanical jaundice is possible.

Jaundice is one of the common symptoms of various diseases of the liver and biliary tract. Diagnostic difficulties lead to the fact that patients with parenchymal jaundice may be admitted to surgical wards, and those with mechanical jaundice - to infectious diseases wards.

Jaundice of mechanical origin causes significant changes in the liver, heart, and kidneys.

Obstructive jaundice is most often caused by cholelithiasis, inflammatory and scarring lesions of the bile ducts, and tumours.

Mechanical jaundice in gallstone disease is caused by bile duct stones. As a rule, stones from the gallbladder enter the duct.

Symptoms. The pain is very intense (hepatic colic) with typical radiation to the back, right shoulder blade, and shoulder.

The onset of pain is preceded by an error in diet.

Feeling of weight, bloating, vomiting. After 12-24 hours, jaundice of the sclerae and skin appears. Urine is dark, stools are light grey in colour (acholic). Patients develop skin itching. In case of prolonged jaundice, there is a scaling on the skin.

Examination of the abdomen reveals tension of the abdominal wall muscles, enlargement of the liver, its edge is blunt and painful. Deep palpation reveals tenderness in the gallbladder and epigastrium.

Accompanying cholangitis is caused by an outbreak of infection in the bile ducts and is manifested by a high temperature rise (up to 39-40 °C), chills, and heavy sweating.

In contrast to choledocholithiasis, jaundice develops slowly, gradually progressing, and the skin has an olive-green colour. The pain is constant and occurs later. In the pre-jaundice period, fatigue, weight loss, and weakness are common. Women are more often affected than men. An abdominal examination reveals an enlarged, slightly painful liver.

In case of pancreatic head tumours, an enlarged, tense, painful gallbladder can be palpated (Courvoisier's symptom).

**Acute cholecystitis in children**

Among the conditions that contribute to the onset of inflammation in the gallbladder in children, the most common are bile stasis in the gallbladder, the introduction of parasites and the penetration of infection by enterogenous, haematogenous or lymphogenic means.

Infectious cholecystitis is characterised by an acute onset of the disease, most often associated with a particular illness suffered by the child (sore throat, infectious hepatitis, upper respiratory tract catarrh). Abdominal pain appears in the abdomen.

Older children complain of pain in the right hypochondrium, and then throughout the abdomen, but more on the right. In younger children, it is not possible to determine the location of the pain. The temperature rises, and subcharacteristic sclerae are noted.

Abdominal palpation reveals tension in the right upper quadrant. The liver is painful and enlarged. Positive symptoms of peritoneal irritation. In the period of exacerbation - leukaemia, increased ESR, eosinophilia.

In phlegmonous and gangrenous types, the clinical picture is characterised by more pronounced symptoms: pain is sharp, vomiting, high temperature. The patient's condition is severe, symptoms of intoxication are pronounced, the tongue is dry. The abdomen is evenly distended, the abdominal wall muscles are tense throughout the right side.

Duodenal probing allows to differentiate infectious cholecystitis from giardiasis.

Surgical treatment of acute cholecystitis in children is rarely used. This is due to the fact that destructive forms of gallbladder inflammation in childhood are the exception, as are biliary stones. In rare cases, surgical intervention is required.

Differential diagnosis of acute cholecystitis

Acute cholecystitis is most often differentiated from renal colic, acute pancreatitis, gastric and duodenal ulcer perforation, and acute appendicitis.

Renal colic, unlike acute cholecystitis, is characterised by acute pain in the lumbar region with downward radiation to the genitals and thigh, as well as the development of dysuric disorders. Temperature was normal, leukocytosis was absent. Abdominal changes in renal colic are rarely observed.

In severe cases of renal colic, with ureteral stones, abdominal distention, abdominal wall muscle tension and repeated vomiting may occur.

In case of renal colic, a positive Pasternatsky's symptom is noted and there are no symptoms of peritoneal irritation.

In the urine test for kidney disease, red blood cells, leukocytes, and salts are found.

Acute appendicitis with high localisation of the cecum can simulate acute inflammation of the gallbladder.

Acute cholecystitis occurs with repeated vomiting of bile, characterised by irradiation of the patient's right shoulder blade and shoulder. Acute appendicitis is characterised by a more severe course with rapid development of purulent peritonitis.

Perforated duodenal ulcers, mostly covered, can be misdiagnosed as acute cholecystitis.

Acute cholecystitis, unlike perforated ulcer, is characterised by the absence of a peptic ulcer history, the presence of previous attacks of cholecystitis and cholelithiasis.

Acute cholecystitis is characterised by repeated vomiting, irradiation of the patient's pain to the shoulder, scapula, fever, and leukaemia.

Concealed perforations occur with an acute onset and marked muscle tension of the anterior abdominal wall in the first few hours after the onset of the disease; local pain in the right hypochondrium due to leakage of gastric and duodenal contents is noted, which is not typical for acute cholecystitis.

The diagnosis is facilitated by the presence of free gas in the abdominal cavity, which is characteristic of ulcer perforation.

Acute pancreatitis occurs with rapidly increasing symptoms of intoxication, tachycardia, and intestinal paresis. The stomach area is characterised by girdling pain and uncontrollable vomiting.

Increased content of diastase in blood and urine is typical for acute pancreatitis.

There may be a combination of acute cholecystitis and mechanical jaundice due to obstruction of the common bile duct by a stone (described above).

For the differential diagnosis between cholecystitis and viral hepatitis, it is important to determine the activity of transaminases in the blood serum. In mild forms of viral hepatitis, the activity of transaminases increases 10-fold, in severe forms - 40-fold. A significant rise in ALT.

**Diagnostics of acute cholecystitis**

- Plain radiography of the right hypochondrium. In 10-70% of cases of acute cholecystitis, radiopaque stones and the shadow of an enlarged gallbladder are detected.
- Oral cholecystography is ineffective, the gallbladder is usually not contrasted due to blockage of the vesicular duct.
- Intravenous cholecystocholangiography.
- Infusion-drip cholecystocholangiography facilitates faster and more accurate diagnosis, facilitates the choice of treatment tactics.
- ULTRASOUND.
- ERCP (endoscopic retrograde cholangiopancreatography).
- Laparoscopy.

Laparoscopy allows you to clarify the diagnosis, assess the degree of destruction of the gallbladder, the severity of peritonitis, and carry out a number of therapeutic measures.

**Laboratory diagnostics of acute cholecystitis**

- Complete blood count and urine test.
- Total protein.
- Bilirubin.
- Transaminase.
- Alkaline phosphatase.
- Sulemic test.
- Prothrombin test.
- Determination of aminotransferase activity.
- Blood sugar.

**Treatment of acute cholecystitis**

If a diagnosis of acute cholecystitis is made, the patient should be hospitalised in a surgical department. Treatment at home or in the therapeutic department is unacceptable. Antispasmodics (0.2 % platyphylline solution, atropine, nitroglycerin, etc.) can be administered before transport. After confirmation of the diagnosis, not all patients are offered immediate surgery. The choice of treatment method depends on the severity of the attack, the severity of inflammation symptoms, and the nature of complications.

Conservative therapy for acute cholecystitis is aimed at improving or restoring the outflow from the gallbladder and extrahepatic bile ducts, relieving inflammatory effects of intoxication, and correcting various disorders (diabetes, hypertension, heart rhythm disorders).

Removal of spasm from the muscle wicks of the vesicular duct and terminal choledochus. Administration of antispasmodics (no-shpa, atropine, platyphylline,

nitroglycerin). Novocaine blockade of the circular binding of the liver and paraneural blockade. Intravenous drip - 0.25% novocaine solution, 200 ml.

Detoxification therapy (5% glucose solution, Ringer's solution, haemodesis).

For the prevention of liver failure, cocarboxylase and ascorbic acid are prescribed.

In case of heart rhythm disturbance or chronic coronary insufficiency, panangin is indicated.

Antibiotics are not initially prescribed. They are indicated for destructive forms of cholecystitis and peritonitis.

**Surgical treatment of acute cholecystitis**

Surgical interventions for acute cholecystitis are divided into emergency, urgent and delayed operations.

Emergency surgeries are performed within the first day of admission (perforated cholecystitis, peritonitis and in case of severe and increasing intoxication due to jaundice, purulent cholangitis).

Urgent surgery is performed within 24-48 hours of admission if the initiated conservative therapy is ineffective.

Delayed surgeries are performed 2-3 weeks after an acute attack and conservative treatment.

The main operation for acute cholecystitis is cholecystectomy. Surgery for acute cholecystitis is aimed at eliminating the focus of inflammation in the abdominal cavity and restoring the patency of the biliary tract.

Choledochotomy (opening of the choledochus)

Indications: presence of jaundice, stones in the bile duct, narrowing of the bile ducts, wide choledochus with a diameter of 1.5 cm, presence of small stones, history of jaundice.

Choledochoduodenostomy

Indications: wide choledochus, non-removable stricture, stone embedded in the large duodenal nipple, small stones of the common bile duct, cholangitis

In very severe cases, patients undergo cholecystostomy.

Meals after surgery begin on the 2-3rd day. From 4-5 days after the operation, you are allowed to sit down and then get up.

The family doctor should remember that early hospitalisation is a prerequisite for the successful treatment of patients with acute cholecystitis. In case of acute abdominal pain, no painkillers should be administered. Morphine is contraindicated.

Delay in hospitalisation is dangerous because dystrophic changes are detected in the liver tissue within the first day after the onset of acute cholecystitis.

Patients with obtundative jaundice should be hospitalised earlier.

Phytotherapy for cholelithiasis is possible only in the absence of complications, if there are no direct indications for surgical treatment. Barberries, immortelle, strawberries, corn stigmas, acorns, tansy, celandine, rosehip root, etc. are used.

## PERFORATED GASTRIC AND DUODENAL ULCER

Perforation is one of the most dangerous and common complications of peptic ulcer disease. It ranks 4th after acute appendicitis, strangulated hernias and acute intestinal obstruction. Perforation complicates the course of gastric and duodenal ulcers, according to various authors, in 6-20% of cases, and the absence of a peptic ulcer history occurs in 5-10% of young patients. Among patients with perforated ulcers, 95 % are men. It is more common in people aged 20-50 years (80 %), over 60 years - in 4.2 %.

Perforation can occur at any age, even in infants. Ulcer breakthrough occurs at any time of the year, but more often in spring and winter. It is possible that this seasonality is related to dietary habits.

The breakthrough can occur at any time of the day.

**Factors contributing to ulcer breakthrough:**

- alcohol consumption;
- abundant food;
- physical tension
- nervous tension (stress);
- after gastric probing.

Since a perforation is not an independent disease, but a complication of peptic ulcer disease, it is to be expected that in the history of a patient with a perforated ulcer, it is easy to find indications of complaints typical of peptic ulcer disease in the past. There are asymptomatic leaking ulcers of the stomach and duodenum (‘dumb’ ulcers) that manifest themselves clinically at the time of breakthrough.

Patients over the age of 60 often have no or a short history of ulcers. When taking anamnesis, you should persistently seek information about the period preceding the breakthrough: was there heartburn? Did the patient take soda or not? The course of the disease and symptoms depend on the location of the breakthrough. The perforation in gastric ulcer is larger and less frequently covered than the perforation in duodenal ulcer.

Patients with perforated ulcers are classified as surgical patients, because to save their lives, an emergency operation is required to eliminate the developing peritonitis and the cause of its occurrence.

**Clinic of perforated gastric and duodenal ulcer**

According to N. Neimark (1972), in the clinical picture of perforated gastroduodenal ulcer it is advisable to distinguish three periods.

I. Period of ‘acute abdomen’ (shock or perforation). Duration 6-8 h. According to Mondor, this stage is the easiest to diagnose and the most favourable for treatment in case of urgent surgery.

The pain is sudden, severe, excruciating. Each patient describes the pain in his or her own way, but most often: ‘It was like a knife’, ‘it stabbed me’, ‘it was a terrible stabbing’, ‘something burst’, ‘a dagger in the stomach’. Patients often say that they fainted and fell down because of the unbearable pain.

The pain is usually localised in the epigastrium or right hypochondrium, lasts 2-3 hours, and radiates to the shoulder, scapula, clavicle (Elecker's symptom, or ‘phrenicus symptom’).

Examination. The patient lies on the back or on the right side with the legs brought to the abdomen. The skin is pale, covered with cold sweat, the expression is frightened.

The patient moans, the pulse in the first hours is rare (vague pulse), up to 50-60 beats per minute.

At the end of the shock phase, bradycardia begins to be replaced by an increase in the pulse.

Blood pressure remains low during the first phase, but can return to normal in 1.5-2 hours. The decrease in pressure depends on the rapid development and severity of peritonitis. Blood pressure is unstable in elderly and old patients.

The temperature is normal.

Respiration in the first phase is accelerated to 25-30 per minute. The patient's attempt to breathe deeper leads to a sharp increase in abdominal pain.

After breakthrough, the patient is thirsty. The anterior abdominal wall at the beginning of the disease is immobile, does not participate in breathing, and is retracted.

Palpation. Touching the abdomen causes significant soreness in the supra-abdominal region, right hypochondrium and closer to the midline. The main symptom is a sharp tension of the abdominal wall, ‘muscle defence’.

Palpation of a tense abdomen is painful. Tension of the abdominal wall and the Shchetkin-Blumberg symptom are related to the very initial period, later, as the clinical picture changes, the degree of tension and the area of the Shchetkin-Blumberg symptom change.

Abdominal percussion reveals severe tenderness in the epigastrium and right hypochondrium. Very often, it is possible to establish the disappearance of hepatic dullness, tympanitis, and a high boxy sound over the liver.

This symptom is caused by the fact that at the moment of ulcer rupture, not only the liquid contents, squeaking, but also air rushing upwards, comes out through the hole in the stomach or duodenum into the abdominal cavity. The air is located under the diaphragm, above the liver.

The more air that has entered the abdominal cavity, the more pronounced this symptom will be. But the absence of this symptom in no way can serve as a reason to exclude a breakthrough. In a later period, abdominal percussion reveals dullness in the gentle parts.

When examining the rectum with a finger, the painfulness of the rectal vesicle in men and the recto-uterine recess in women (Kuhlenkampff's symptom) is determined.

II. The phase of ‘apparent well-being’ (8-12 hours), improvement. It is in this phase that diagnostic errors are particularly common, which leads to late hospitalisation. This phase is quite rightly called the ‘treacherous’ phase.

The most acute, unbearable pain has decreased and become less severe.

The patient seems to be coming to his senses, he begins to think that in a little while ‘everything will be fine’. Breathing is free, deeper. The face does not look pale. Subjective improvement is deceptive. The process in the abdominal cavity continues and spreads, as evidenced by many signs. After 5-6 hours, the temperature rises to 37.5-38 °C.

The more time has passed, the faster the pulse, the more noticeable the discrepancy between the pulse rate and temperature (‘scissors’).

Blood pressure decreases. Breathing is rapid, the tongue becomes dry. There is abdominal distention.

On palpation, there is a distinct stiffness, but no longer a board-like abdomen. Tension is expressed in the right hypochondrium no less than in the upper abdomen. Positive Shchetkin-Blumberg symptom. Rectal examination always reveals sharp tenderness.

III. Third phase (peritonitis) - 24 hours or more. The patient's condition is severe: sunken eyes, cyanosis of the lips, face, frequent and shallow breathing, thirst, continuous abdominal pain. The rate of peritonitis development depends on the number of gastric contents, its acidity, type of bacteria, size of the perforation, location, age of the accompanying pathology.

The body temperature is up to 38-39 °C, the pulse is frequent, weakly filled.

Blood pressure is low.

Distended, tense, painful abdomen. Positive symptoms of peritoneal irritation.

In some patients, it is possible to detect pre-breakthrough preoperative symptoms, characterised by a significant increase in spoon pain and nausea, and the appearance of vomiting. These symptoms are evidence of an exacerbation of the inflammatory process in the area of the ulcerative defect. This circumstance predisposes to a breakthrough. An increase in intragastric pressure caused by vomiting and physical exertion is a favourable factor for this complication.

There are known cases of gastric and duodenal ulcers (‘silent’ ulcer), which first manifest themselves as a breakthrough. The opinion that there is no history of ulcers reflects not the actual incidence of silent ulcers, but the incidence of ineptly collected data.

In 3-4 % of all observations, there are cases of atypical perforations (rupture of an ulcer located retroperitoneally, on the posterior wall of the duodenum, in the cardiac section of the stomach or on its posterior wall).

The stomach contents do not enter the free abdominal cavity, but retroperitoneally or into the cephalic pouch. There is no severe pain and abdominal wall tension, as in the typical form. Only in cases of an abscess or its breakthrough into the abdominal cavity are there indications for surgery.

Diagnostic errors are usually found in atypical perforations that are covered. Errors are the result of a superficially collected history, careless examination of the patient, and the inability to compare anamnesis and symptoms.

Covered perforations of gastroduodenal ulcers occur in 5% of cases.

The perforation is covered by a lump of food or by adhesion of neighbouring organs (liver, gallbladder, lumbar colon, omentum). Covering is possible under certain conditions: a small perforation, its location on the back wall of the stomach or duodenum, an empty or slightly filled stomach.

The onset of the disease is no different from perforated abdominal ulcers. The shock phase lasts for 15-30 minutes, and then the disease progression is interrupted. Only tension of the abdominal wall in a limited area and soreness are noted.

In addition to the renewal of the disease (breakthrough), it is possible to form an abscess in the area of the covered perforation, which can lead to spilled peritonitis.

**Differential diagnosis of perforated gastric and duodenal ulcer**

In elderly and senile patients, perforated ulcer is difficult to differentiate from angina pectoris and myocardial infarction, acute cholecystitis, appendicitis, and intestinal obstruction.

In angina pectoris and myocardial infarction, abdominal pain is observed in the area of the xiphoid process, and not throughout the supraperitoneal region, as in perforated ulcer, the patient does not complain of nausea, vomiting; soreness in the upper abdomen is not pronounced; there is no tension, symptoms of peritoneal irritation are negative.

Acute cholecystitis is more common in women, and perforated ulcer - in men. Severe pain in the right hypochondrium is characteristic of acute cholecystitis, there is no shock-like state, less pronounced tension, less sharp

the Shchetkin-Blumberg symptom is pronounced, and the temperature rises early.

If a patient with a perforated ulcer is immobile, then in acute cholecystitis he is restless. After vomiting, a patient with a perforated ulcer feels relief, which is not the case with cholecystitis. In acute cholecystitis, an enlarged gallbladder may be detected.

In acute appendicitis, pain and tenderness in the retroperitoneal region disappear or are mild within 6 hours of the onset of the disease.

Tension in acute appendicitis involves the ileum, and in perforated ulcer it is localised in the epigastrium and right side of the abdomen.

The Shchetkin-Blumberg symptom in acute appendicitis is detected only in the ileum.

Intestinal obstruction is characterised by cramping pain, while perforated ulcers are characterised by constant pain. Vomiting in case of intestinal obstruction is persistent and has a faecal character. Abdominal distension and asymmetry are characteristic of obstruction. The symptom of splashing noise is detected in intestinal obstruction.

**Diagnosis of perforated gastric and duodenal ulcer**

- Anamnesis of the disease (peptic ulcer).
- Clinical examination.

- Examination by fluoroscopy or radiography of the abdominal cavity in the patient's upright position or in the left side position (laterography).
- X-ray examination in 70% of patients reveals the presence of free gas in the abdominal cavity, under the right dome of the diaphragm.
- Pneumogastrography or injection of a contrast agent through a gastric tube, followed by an examination radiography of the abdominal cavity. The detection of gas under the diaphragm or contrast medium in the free abdominal cavity on the radiograph indicates perforation of the ulcer.
- Fibrogastroduodenoscopy. In case of occult perforations, intragastric air injection during fibrogastroduodenoscopy can provoke the appearance of air under the diaphragm and help to detect the ulcer. During the examination, severe abdominal pain may occur during air injection, which is also a diagnostic symptom.
- Laparocentesis using the technique of a balloon catheter to detect peritonitis and abdominal effusion.
- Neumark's diagnostic test (2-3 ml of exudate from the abdominal cavity and 4-5 drops of 10% iodine tincture).
- If there is an admixture of gastric contents in the fluid, then under the influence of iodine tincture, it acquires a dark dirty blue colour (due to the remaining starch).
- Laparoscopy, which allows to detect signs of peritonitis.
- Additional methods of examination to exclude heart disease include an ECG, examination by a therapist.

**Treatment of perforated gastric and duodenal ulcers**

The method of treatment of patients with perforated gastroduodenal ulcers is surgical.

The purpose of surgery is to stop the communication between the stomach cavity and the abdominal cavity and to rehabilitate the latter.

The world literature describes about 40 methods and their modifications for the treatment of perforated gastric and duodenal ulcers. However, surgical treatment should be approached in a differentiated manner, i.e., one approach for duodenal ulcers and another for gastric ulcers.

An important role is played by the time interval from the moment of perforation to the start of surgical intervention. The patient's age and condition also play a role in the choice of surgery method.

The degree of surgical risk in a patient can be influenced by concomitant somatic pathology and its severity, as well as the surgeon's professional training.

During the operation, great importance is attached to the weight and prevalence of peritonitis.

Three types of surgeries are used for perforated ulcers:

- resection of the ulcer
- resection of the stomach;
- organ-preserving surgeries in combination with vagotomy.

Indications for ulcer suturing

- Young people with a ‘fresh’ ulcer without morphological signs of chronic and ulcerative stenosis.
- In common forms of peritonitis.
- High degree of surgical risk (advanced age, severe comorbidities).
- More than 6 hours after perforation.

Due to the fact that after suturing a perforated ulcer, more than half of patients experience peptic ulcer progression, a significant number of complications are observed, mostly radical operations (antrumectomy, or excision of the ulcerative defect with pyloroplasty and vagotomy).

Indications for gastric resection in case of perforated ulcer

- The period from the moment of perforation is not more than 6 hours.
- A history of a prolonged ulcer process.
- Absence of severe concomitant pathology.
- Suspicion of malignancy, stenosis, bleeding, penetration.

Indications for organ-preserving operations

- Young age.
- No prevalence of peritonitis.
- Combination of perforation and bleeding.
- Perforation of the anterior wall of the pyloroduodenal canal in the absence of a large ulcerative infiltrate spreading to the surrounding organs.

Vagotomy with ulcer excision and vagotomy with ulcer excision and pyloroplasty, loroplasty

- In case of duodenal ulcer (anterior wall) or pyloric ulcer, not accompanied by a large infiltrate, scar deformity.
- In case of combination of perforation and bleeding, stenosis, penetration.

**Medical and tactical mistakes**

- At the pre-hospital stage (the doctor hesitates between a diagnosis of a surgical disease and a therapeutic one), referring a patient to a therapeutic hospital is wrong.
- It is also wrong to perform complex surgeries in patients aged 70 years and older with severe comorbidities.

Late hospitalisation (later than 6-24 hours) is associated not only with late turnover of patients for care, their refusal to be admitted, but also with untimely diagnosis and errors in recognising the disease.

The reasons for these mistakes are varied:

- a superficial familiarisation with the anamnesis;
- insufficiently thorough examination at the time of the doctor's examination at home and in the clinic;
- cases of atypical perforations;
- covered perforations;
- changes in the clinical picture that are associated with time that has passed since the onset of the disease.

Early surgical treatment of chronic ulcers should be considered an important preventive measure.

A timely diagnosis, timely surgery, modern anaesthesia and postoperative therapy contribute to the improvement of the results of treatment of patients with perforated ulcers.

**GASTROINTESTINAL BLEEDING**

Gastrointestinal bleeding is divided into ulcerative and non-ulcerative bleeding. Ulcerative haemorrhages account for 60% of all acute gastrointestinal bleeding. The mechanisms of gastroduodenal bleeding are closely related to the pathogenesis of gastric and duodenal ulcers. Currently, more than a hundred diseases are known to cause acute gastrointestinal bleeding. Acute digestive ulcers can occur at any age - both in infants and in the elderly. Sudden development of complications, recurrent nature and difficult to predict outcomes lead to high mortality - 32-33% in patients with advanced bleeding recurrence.

Several major pathogenic factors are important in the occurrence of bleeding from a gastric or duodenal ulcer:

- increased activity of the acid-peptic factor;
- decreased resistance of the digestive tract mucosa to hydrochloric acid, enzymes, food, and medicines;
- Primary damage to the vascular wall caused by its varicose veins, increased permeability and fragility, atherosclerotic lesions;

The source of bleeding in gastric and duodenal ulcers is most often an arterial vessel, less often a vein.

The following disorders occur during peptic ulcer bleeding:

- hypovolaemic shock;
- brain hypoxia;
- renal failure;
- liver failure;
- myocardial hypoxia;

- intoxication by blood decay products in the intestine. The most common location for acute ulcers is the stomach.

In 30 % of patients, the onset of bleeding symptoms is the debut of peptic ulcer disease. Clinic of gastrointestinal bleeding.

The clinic of gastric duodenal bleeding does not have any peculiarities. It is typical for internal bleeding of any localisation.

At the very beginning of the development of the complication, patients notice the appearance of general weakness and dizziness. The skin is pale, cold sweat, cyanosis of the lips. There is a feeling of fear, hearing impairment in the form of ringing in the ears, tachycardia up to 100-120 beats per minute, and a decrease in blood pressure.

During physical activity or the act of defecation, some patients suddenly faint and fall down for a short time.

At the same time, sometimes later, bloody vomiting or vomiting of the colour of 'coffee grounds' appears.

The family doctor should be interested in the vomit, its quantity, and colour.

Bloody vomit and black liquid faeces - melena - are absolute signs of bleeding.

Bloody vomiting is more typical for diseases with a source of bleeding located in the oesophagus and stomach.

The ingestion of blood in the amount of 50 ml or more into the upper part of the digestive tract is sufficient to produce tar-like faeces. This colouration is caused by the formation of ferrous sulphate from the haemoglobin of the lost blood under the influence of intestinal enzymes.

In case of profuse bleeding, bloody vomiting and melena may occur simultaneously.

The clinical picture of peptic ulcer bleeding depends on the amount of blood that has been spilled into the gastrointestinal tract, the rate of blood loss, and the individual response of the body to blood loss.

The most accurate method of determining the weight of blood loss is to examine the volume of circulating blood and its deficiency (BCC).

There are three grades **of blood loss severity**:

- Grade I - mild; blood loss of 20% of the circulating blood volume. This figure does not exceed 500 ml;
- Grade II - moderate; blood loss is from 20 to 30% of the BCC (from 500 to 1000 ml);
- III grade - severe. Circulating blood volume deficit of 30 to 50 % (over 1000 ml).

**Differential diagnosis**

Differential diagnosis of the causes of gastroduodenal bleeding is a difficult task. It is necessary to correctly collect anamnesis of the disease, past illnesses, information about the bleeding that has occurred and their treatment.

Peptic ulcer bleeding is preceded by increased pain in the upper abdomen associated with exacerbation of the ulcer process.

The second place is occupied by erosive haemorrhagic gastritis.

It can occur acutely and on the basis of exacerbation of chronic gastritis. Ulcers are found in the body and antrum of the stomach.

Bleeding is not profuse and is not accompanied by collapse.

The third place as a cause of bleeding from the upper part of the digestive tube belongs to the decaying cancer of the stomach. Elderly age, lack of appetite, rotten belching, sudden weight loss, fatigue suggest that this bleeding is of a tumour nature.

Repeated vomiting in patients with atrophy of the gastric mucosa after alcohol consumption leads to the development of Mallory-Weiss syndrome: longitudinal tears of the cardioesophageal mucosa appear.

An abdominal examination reveals ascites and dilated saphenous veins of the anterior abdominal wall - the 'jellyfish head'. Palpable increase in the size of the liver or spleen should suggest bleeding from the dilated esophageal veins.

In case of liver cirrhosis, the blood flow through the portal vein is disturbed. This leads to the formation of large venous anastomoses between the portal and vena cava systems.

**Diagnostics of gastrointestinal bleeding**

Complete blood count: Hb, blood group, Rh factor, coagulation system, acid-base status. If a significant amount of blood and clots are found in the gastric lumen, all patients should be rinsed with cold water.

All patients with bleeding or suspected bleeding undergo an emergency endoscopic examination (FGS).

As a result of the examination, an important role is played by methods of physical and pharmacological influence on the source of bleeding.

The effectiveness of haemostasis is monitored by a permanent nasogastric tube and repeated endoscopic examination.

**Treatment of gastrointestinal bleeding**

If signs of bleeding are detected, any specialist in the outpatient clinic or family doctor should take all possible measures to deliver the patient to a surgical hospital in a horizontal position. Put a cold heating pad on the stomach area.

Food and liquid intake by mouth should be completely excluded. In case of severe blood loss, infusion and haemostatic therapy is performed, which consists in the administration of

- rheopolyglucin - 200 ml;
- 5% solution of epsilon-aminocaproic acid, 200 ml;
- 4% sodium bicarbonate solution, 200 ml;
- 10-20% albumin solution,
- 100-150 ml of fresh frozen plasma;
- transfusion of single-group donor blood to patients

It is unacceptable to administer to patients with gastroduodenal bleeding cardiac and vasoconstrictor drugs in the event of a collapse. They can be used in cases of failure of massive infusion therapy. Patients need anti-ulcer therapy (antacids, antispasmodics, sedatives and substances that stimulate repair processes).

An important role is played by local effects on the source of bleeding (ice bubble on the stomach area).

Intraventricularly inject 150 ml of cooled epsilon-aminocaproic acid with 10 g of thrombin, 4 ml of 0.1% norepinephrine solution in 150 ml of saline.

Endoscopic methods include irrigation of the bleeding source with silver nitrate solution, injection of vasoconstrictor drugs into the submucosal layer around the bleeding area. Hyperbaric oxygenation (HBO) is indicated as part of the treatment package.

After stopping peptic ulcer bleeding, bed rest and the Meilengracht diet are prescribed.

**Surgical treatment of gastrointestinal bleeding**

The choice of surgical intervention method in patients with bleeding ulcers depends on the patient's condition, endoscopic examination findings, haemostasis and patient location, and the degree of bleeding activity.

Depending on the timing of bleeding ulcers, the following types of surgery are distinguished: emergency, urgent and delayed.

Surgical intervention for a bleeding ulcer involves stopping the bleeding and treatment. Bleeding control can be achieved by performing both radical and palliative surgical operations. In patients with ongoing

bleeding on the background of concomitant severe pathology, in the elderly, low-traumatic, rapid interventions are advisable.

Cure of peptic ulcer disease is achieved by radical surgery.

If the ulcer is located in the body of the stomach and with known normal or reduced secretory function, in the absence of signs of duodenostasis, pylorus-sparing gastric resection is preferable.

Detection of a bleeding ulcerative defect in the cardiac or subcardiac regions with stable haemodynamics is a staircase gastric resection.

When performing emergency surgery on the small curvature of the stomach, an infiltrate of 3-4 cm in diameter may be detected. If the malignancy of the lesion cannot be excluded with certainty, the operation should be performed according to oncological principles - subtotal gastric resection or gastrectomy.

The detection of a bleeding ulcer of more than 1 cm in diameter in the duodenum requires an antrum or hemigastrectomy in combination with a vagotomy. Continued bleeding in the setting of severe blood loss in elderly or senile patients is an indication for excision of a duodenal ulcer located on the anterior wall.

The localisation of the source of ongoing bleeding on the posterior wall of the duodenum requires stitching of the bleeding vessel in the ulcer. If mechanically possible, it is necessary to remove the crater of the penetrating ulcer beyond the duodenal lumen - extraduodenalisation of the ulcer.

In Mallory-Weiss syndrome, treatment begins with conservative therapy: blood transfusion, plasma, haemostatics; gastric lavage with cold water; therapeutic

endoscopy; application of an aerosol film, photocoagulation of the gap with an Aig laser.

If conservative therapy is ineffective and bleeding continues, surgery is indicated - laparotomy, gastrotomy, suturing of mucosal tears.

In case of bleeding from varicose veins of the oesophagus and stomach due to portal hypertension, the above haemostatic therapy is performed, as well as in case of other causes of bleeding. A Blackmore-type obturator probe is widely used.

## OESOPHAGEAL AND GASTRIC BLEEDING IN CHILDREN

### Causes of oesophageal and gastric bleeding in children

- Portal hypertension.
- Reflux esophagitis.
- Taking acetylsalicylic acid.
- Mechanical injuries.
- Stressful situations.

### Clinic of oesophageal and gastric bleeding in children

Bleeding in children with portal hypertension is more likely to occur before the age of 6. 1-3 days before the onset of bleeding, fever, malaise, pallor of the skin, nausea are recorded; a smell of decomposed blood appears from the mouth.

Against a background of complete health, there is profuse (‘fountain’) vomiting with an admixture of red blood and clots, black faeces.

Blood pressure drops, tachycardia.

Bloody vomiting syndrome occurs in case of bleeding from gastric and duodenal ulcers, acute ulcers, in patients treated with hormones, hernia of the esophageal opening of the diaphragm, etc.

Children with bleeding gastric ulcers have a history of aching, hungry pains, changes in appetite, and weight loss.

In children with hiatal hernia, the resulting bloody vomiting and ‘black faeces’ do not immediately affect the child's general condition. They are hospitalised with anaemia of unclear etiology, and only in case of FGS is a hernia of the diaphragm diagnosed.

### Diagnostics of oesophageal and gastric bleeding in children

- X-ray contrast examination of the oesophagus and stomach.
- Fibrogastroduodenoscopy.

### Treatment of oesophageal and gastric bleeding in children

Treatment should begin with conservative therapy. The child is prescribed strict bed rest. Feeding and drinking by mouth are stopped.

Intravenous haemostatic therapy. According to the indications, transfusions of the same group of blood, fresh frozen plasma are performed. In case of bleeding due

to portal hypertension, pituitrin and hyphotocin are used to selectively reduce portal pressure and stop bleeding (5 units of pituitrin are administered per 10-20 ml of isotonic NaCl solution intravenously).

Local gastric hypothermia is used. Sclerosing therapy through an esophagoscope.

In case of ineffectiveness of conservative therapy, surgical treatment is indicated (gastrotomy with stitching of varices of the cardiac esophagus and stomach).

**ACUTE PERITONITIS.**

**Classification of peritonitis**

1. **By prevalence:**
   - local
   - limited
   - delimited
   - general
   - diffuse (widespread)
   - total
2. **By time from the onset of the disease**
   - reactive phase
   - toxic phase
   - terminal phase.

**Medical and diagnostic care in the emergency department**

The main task is to diagnose peritonitis and hospitalise the patient in an emergency. It is worth remembering that the clinical picture will depend on the time since the onset of the disease (phases of the disease).

Reactive phase. Forced supine position with legs tucked to the abdomen, cold sweat, shallow breathing, retracted and immobile abdomen, sharp tension of the abdominal muscles - 'board-shaped abdomen', extremely sharp pain. The Shchetkin-Blumberg symptom is sharply positive

**Toxic phase.** With increasing intoxication, the following appear: sharpened facial features (Hippocrates face), dry speech, tachycardia, abdominal distension, absence of peristaltic sounds. Pain during digital examination of the rectum.

**Terminal phase** - Presence of symptoms of multiple organ failure

**Laboratory tests**

o Complete blood count with hematocrit.

o Blood group and Rh factor.

o Biochemical blood tests (sugar, bilirubin, protein, urea, amylase, prothrombin).

o Complete urine test

**Instrumental tests**

- ECG - must be performed for all patients.
- X-ray examination - survey radiography of the abdominal cavity - according to indicators.
- In unclear cases, diagnostic laparoscopy is performed.

**Note.** It is not advisable to expand the diagnostic search to clarify the cause of peritonitis and delay the timing of surgery.

**Preoperative preparation**

o Before surgery, a gastric tube is necessarily placed and the stomach contents are evacuated without gastric lavage.
o The bladder is emptied and the surgical area is hygienically prepared.
o In severe conditions (in the stage of diffuse peritonitis), intensive therapy is performed together with an anaesthesiologist for 1-3 hours and then surgery.
o In the preoperative period, it is advisable to administer antibiotics.

**Anesthetic support of the operation**

The operation is performed only under general anesthesia, the means of choice is endotracheal anesthesia.

**Surgical treatment of widespread purulent peritonitis**

• Access - wide median laparotomy.
• Removal, elimination or limitation of the source of peritonitis.
• Sanitation of the abdominal cavity (lavage of the abdominal cavity with antiseptic solutions).
• Intubation of the small intestine with signs of intestinal paresis.
• Drainage of the abdominal cavity.
• With a favorable course of peritonitis (grade I severity) - closing the laparotomy wound, with a severe course of peritonitis (grade II and III severity) - performing programmed relaparotomies.

To assess the severity of the patient's condition and the prognosis of the disease, a 6-point system is used - the Mannheim peritonitis index (MPI). For assessment on this scale, standard information (risk factor) based on clinical data is used (Table 3).
Table

**Mannheim peritonitis index**

| Risk factor | Weight rating (points) |
|---|---|
| Age over 50 | 5 |
| Female gender | 5 |
| Presence of organ failure | 7 |
| Presence of malignant tumor | 4 |
| Duration of peritonitis more than 24 hours | 4 |
| Colon as a source of peritonitis | 4 |
| Diffuse peritonitis | 6 |
| Exudate (only one answer): | |
| • transparent | 0 |
| • turbid-purulent | 6 |
| • fecal-purulent | 12 |

MPI provides 3 degrees of severity of peritonitis. With an index of 20 points (I degree of severity), the mortality rate is 0%, within 20-30 points (II degree of severity) - 29%, more than 30 points (III degree of severity) - 100%. This scale is well-founded, has high sensitivity and accuracy in assessing the severity, treatment and prognosis of the disease.

## Treatment

1) Urgent hospitalization in the surgical department and possibly earlier surgery after minimal preoperative preparation (gastric emptying with a probe, detoxification, infusion therapy, cardiac drugs).
2) Type of anesthesia (combined anesthesia with mechanical ventilation, rarely - local).
3) Operation protocol.
4) Justification of the tactics of further treatment (conservative, planned laparosanation, laparostomy, abdominal lavage).
5) Postoperative management: a) position of the patient in bed (Fowler's); b) local hypothermia (ice on the abdomen); c) antibiotics (selection, method of administration - into the abdominal cavity, intravenously, intramuscularly, into the umbilical vein, into the aorta, into the abdominal trunk, into the abdominal artery); d) detoxification therapy, including extracorporeal detoxification methods; e) combating intestinal paresis; f) cardiac drugs; g) prevention of pulmonary complications; h) vitamin therapy; i) proteolysis inhibitors, anti-inflammatory drugs; j) combating renal failure (forced diuresis); l) drugs that increase the overall reactivity of the body; m) parenteral nutrition, diet; n) postoperative course (time of replacement and removal of drains, removal of sutures, nature of healing of the surgical wound).

6) Discharge time, recommendations upon discharge, period of incapacity for work.

## 5. Lesson plan and organizational structure

| № | Main stages of the lesson. Their function and content. | Learning objectives in learning levels | Control and training methods. | Methodological support materials | Time in minutes |
|---|---|---|---|---|---|
| | 2 | 3 | 4 | 5 | |
| | **Preparatory stage** | | | | |
| . | Organization of the lesson. | | | | min. |
| . | Setting learning goals and motivating the topic. | | | | 0 min. |
| . | Control of the initial level of knowledge, skills, abilities. | | | | |
| | a) Anatomy and physiology of the abdominal organs | II | 1. Individual oral interview. | 1. Tables<br>2. Slides<br>3. Presentation, computer control | |
| | b) Etiology and pathogenesis of abdominal organs | II | 2. Written theoretical survey | | 0 min |
| | c) Diagnostic program for acute abdomen | III | | 4. Level III tasks | |
| | d) Conservative therapy and preparation for surgery | II | 3. Solving non-standard problems | | |
| | e) Surgical interventions for acute abdomen | II | 4. Solution of typical problems.<br>5. Prescribing treatment to a patient. | 5. Equipment, radiographs, medical histories. | |
| | **Main stage** | | | | |
| . | Formation of professional skills and abilities | | | | |
| | 1. To master the methods of objective examination of patients with acute abdomen syndrome | III | Method of forming skills of practical training | Educational equipment, orientation maps. Atypical tasks in the form of: patient, disease histories, test situational tasks, business games, dressings | |

| | | | | | |
|---|---|---|---|---|---|
| | 2. To manage a patient with acute abdominal syndrome | III | Method of forming skills. | | |
| | | | | | 30 min. |
| | **Final stage** | | | | |
| | Control and correction of the level of professional skills and abilities | III | Control method: individual control of practical skills | Equipment | 0 min. |
| | Summing up the results of the lesson. | | | | min. |
| | Homework, educational literature on the topic. | | Oriented maps for independent work with literature | | min. |

## 6. Materials on methodological support of the lesson

## 6.1. Control materials for the preparatory stage of the lesson.

## Questions

- Anatomical data and physiological properties of the peritoneum.
- Classification of peritonitis by etiology, causes, nature of the course, nature of the pathogen, effusion, prevalence, limitation, phase of the course.
- Pathogenesis of acute secondary nonspecific infectious peritonitis and diseases that cause it.
- Clinical picture of acute peritonitis by phases of its course.
- Features of the course of peritonitis in children, elderly, weakened patients, in the postoperative period.
- Differential diagnosis of peritonitis.
- Prevention and surgical treatment of peritonitis (stages of the operation, types of drainage and indications for it, peritoneal lavage, laparostomy, planned laparosanations).
- Postoperative management of patients with acute surgical diseases of the abdominal cavity.

- Mortality depending on the timing of the operation, age, type of peritonitis.
- Delimited peritonitis, its forms depending on localization, treatment.
- Pneumococcal, gonococcal peritonitis.
- Chronic peritonitis, its etiology, forms, treatment.
- Postoperative peritonitis.
- Abdominal sepsis, systems for assessing the severity of the condition.

**Situational tasks**

1. When performing appendectomy using the Mack-Burney-Volkovych-Diakonov approach, 20 ml of muddy exudate was released from the abdominal cavity. The appendix is thickened, hyperemic, covered with fibrinous plaque. Fibrin deposits are located on the distal part of the cecum and at the base of the appendix. The appendix is removed. Its stump is peritonized with a purse-string and Z-shaped suture. Did the patient have signs of peritonitis? Phase of the disease? Formulate the diagnosis. How would you complete the operation?
2. A 44-year-old patient was admitted to the clinic with a perforated gastric ulcer. The perforation occurred 9 hours ago. He has been suffering from peptic ulcer disease for about 10 years with frequent exacerbations. Condition on admission: moderate severity. On examination: a large amount of muddy exudate in the abdominal cavity, especially in the subdiaphragmatic areas. A small amount of exudate in the right flank of the abdomen and pelvis. The perforation hole is on the lesser curvature 0.5-0.3, fibrin deposits are visible in its circumference. The intestinal loops are slightly hyperemic, not distended. What should be the surgical intervention? How should the abdominal cavity be closed?
3. 3. A 30-year-old patient, 5 days after surgery for acute appendicitis, developed abdominal distension, dull, distending abdominal pain, and repeated vomiting. The tongue is dry. Pulse
   120 beats/min. The borders of the distended stomach are sharply defined. The abdomen is painful and tense in the right half. Positive symptoms of Shchetkin-Blumberg, Voskresensky. Gas and stool retention. Leukocytosis increased from 9.0?10 9 /l to 16.0?10 9 /l. During ultrasound, a small amount of fluid is detected in the pelvis and behind the liver. Loops of the small intestine with a diameter of up to 25 mm. What complication can be suspected in the patient? What will be your treatment tactics?

6.2. Methodological support materials of the main stage of the lesson

1. A 56-year-old patient was admitted with a picture of acute recurrent calculous cholecystitis on the second day after the onset of the attack. The

general condition of the patient upon admission: moderate severity. Temperature - 38.1° C. Pulse - 92 beats / min. The abdomen is painful in the right hypochondrium, where mildly expressed protective muscle tension and a positive Shchetkin-Blumberg symptom were detected. Other parts of the abdomen are in order. The patient received conservative treatment. A day and a half after hospitalization, the patient's condition deteriorated sharply: severe abdominal pain, a feeling of fear, suffocation, vomiting, the temperature rose to 40° C. Pulse - 120 beats / min. Leukocytosis increased from 9.0?10 9 /l to 25.0?10 9./l. The abdomen is distended, diffuse tenderness throughout the abdomen and a positive Shchetkin-Blumberg symptom in the entire right half of the abdomen are determined. What happened to the patient? What should be the surgeon's tactics?

2. A young woman suddenly developed severe pain in the lower right abdomen. The pain is constant, radiating to the rectum. The patient's general condition is satisfactory, temperature 38.8°C, pulse 100 beats/min. The tongue is moist. The abdomen is not distended, participates in the act of breathing. When palpating the abdominal wall in the right iliac region, there is tension, sharp soreness. Shchetkin-Blumberg symptom is positive, Sitkovsky symptom is negative, but with the slightest movement of the patient, the abdominal pain intensifies. Leukocytosis 12.3?10 9 /l. What disease could you suspect in the patient? What additional examinations should she undergo? What will be your treatment tactics?
3. A 29-year-old patient was brought to the clinic by helicopter on the third day from the onset of the disease in an extremely serious condition: lethargic, apathetic, adynamic, unable to report the onset and course of the disease. A typical facial appearance, the “Hippocrates mask” is present. Temperature – 39.2° C, pulse – 132 beats/min, weak filling, blood pressure – 80- and 40-mm Hg. Art. Breathing is superficial, up to 36 breaths/min. The tongue is dry, coated with a brown coating. The abdomen is sharply and diffusely distended, on palpation – moderately painful in all departments. The Shchetkin-Blumberg symptom is negative, “hepatic dullness” is absent. Fluid is determined in the free abdominal cavity. Peristalsis is not auscultated. Liquid feces with an unpleasant odor are released through the gaping sphincter. What diagnosis will you give the patient? How will you treat him?

**6.3. Control materials for the final stage of the lesson**

**1. Patient K., 26 years old, fell ill 10 hours ago after the onset of pain in the lower abdomen, in connection with this she went to the hospital, where she was**

**hospitalized with a diagnosis of acute appendicitis. Ectopic pregnancy. Which study is the most informative for differential diagnosis?**

**A. Puncture of the posterior vaginal vault.**

B. Thoracocentesis.

C. Laparotomy.

D. EFGS.

E. Ultrasound of the abdominal cavity.

**2. Patient N., 34 years old, hospitalized with a diagnosis of acute pancreatitis. He has been ill for 9 days, complaining of pain in the epigastrium and in the left hypochondrium, nausea, vomiting. Examination revealed leukocytosis, sharply increased urine diastasis. Which of the following indicators is the main one for immediate surgical intervention?**

**A. Peritonitis.**

B. He has been ill for 9 days.

C. Sharply increased urine diastasis.

D. Leukocytosis.

E. Constant pain in the epigastrium and hypochondrium.

**3. Patient O., 33 years old, was hospitalized with complaints of cutting pain in the epigastric region. On examination of the patient: the abdomen is tense in all departments. Shchetkin's symptom is detected. Diagnosis: perforated gastric ulcer. Which of the following methods will be the most informative for confirming the diagnosis?**

**A. Survey radiography of the abdominal cavity.**

B. Laparocentesis.

C. Ultrasound of the abdominal cavity.

D. ECG.

E. Laparoscopy.

**4. Patient L., 24 years old, came to the hospital with complaints of pain in the lower abdomen, weakness, nausea, fever up to 38°C. She has been ill for 3 days. During the examination in the emergency department, the diagnosis was established: acute appendicitis, peritonitis. The required access for the operation:**

**A. Median laparotomy.**

V. According to Pyrohov.

S. According to Volkovych-Diakonov.

D. Pararectal.

E. According to Pfannenstiel.

**5. A man, 63 years old, considers himself sick for the last six months after complaints of stool retention and weight loss of 20 kg appeared. He came to the hospital a day after the onset of sharp pain in the left half of the abdomen. The examination revealed indications for immediate surgical intervention. During the operation for widespread peritonitis, a perforation of a cecum tumor was detected. What operation will be the most optimal?**

**A. Suturing the perforation site, ileostomy.**
B. Right-sided hemicolectomy.
C. Cecal resection, ileotransverse anastomosis.
D. Ileostomy.
E. Drainage of the abdominal cavity.

**6. Patient M., 38 years old, hospitalized with a diagnosis of pelvioperitonitis. She has been ill for 7 days, temperature 38.5°C. Complains of pain in the lower abdomen. The tongue is dry, pulse 96 beats per minute, the abdomen is sharply painful in the lower sections upon palpation, a positive Shchetkin symptom is determined. What will be your treatment tactics?**

**A. Laparotomy.**
B. Infusion detoxification therapy.
C. Diagnostic cleaning.
D. Ultrasound of the pelvic organs.
E. Anti-inflammatory therapy.

**7. Patient L., 29 years old, was admitted with a strangulated inguinal hernia, gangrene of the intestine, and phlegmon of the anterior abdominal wall. What will be your surgical tactics?**

**A. Laparotomy, resection of the strangulated organ, opening and drainage of the phlegmon of the abdominal wall.**
B. Opening and drainage of the phlegmon.
C. Antibacterial and anti-inflammatory therapy.
D. Laparotomy, revision of the abdominal cavity.
E. Plastic surgery of the anterior wall.

**8. Patient N., 26 years old, complains of epigastric pain, which gradually shifted to the right half of the abdominal cavity, nausea, temperature 37.3°C. He has been ill for 10 hours. He had not been ill with anything before. What will be your most likely diagnosis?**

**A. Acute appendicitis.**
B. Acute pancreatitis.
C. Acute cholecystitis.
D. Perforating ulcer.
E. Renal colic.

**9. A 35-year-old patient came to the hospital with complaints of pain in the right iliac region, fever up to 37.8°C, nausea and vomiting. The pain appeared 12 hours ago and gradually intensified. Palpation of the abdomen is painful, the most pronounced pain is in the right iliac region. What is your previous diagnosis?**

**A. Acute appendicitis.**
B. Acute cholecystitis.
C. Acute pancreatitis.
D. Perforating ulcer.
E. Ectopic pregnancy.

**10. A 40-year-old patient came with complaints of acute abdominal pain, which appeared suddenly 6 hours ago. The pain is constant, accompanied by nausea and vomiting. During examination: tension of the muscles of the anterior abdominal wall, positive Shchetkin-Blumberg symptom. What is your most likely diagnosis?**

**A. Perforated gastric ulcer.**

B. Acute appendicitis.

C. Acute pancreatitis.

D. Acute cholecystitis.

E. Gastric volvulus.

**6.4. Materials of methodological support for self-training of higher education students**

**To Know:**

• Modern methods of clinical, laboratory, instrumental examination of patients with acute surgical diseases.

• Stages of examination of the abdominal cavity, characteristics of each stage.

• Special research methods used in the examination of surgical patients

• Definition of the concept of "anatomical and physiological features of the peritoneum".

• Classification of acute surgical diseases of the abdominal cavity.

• Sources of development of acute purulent peritonitis. Features of the spread of abdominal infection in various acute surgical diseases of the abdominal cavity. Pathoanatomical changes in acute purulent peritonitis, pathogenesis. The significance of impaired absorption from the abdominal cavity, intestinal paresis, endotoxemia, impaired hydroid balance and microcirculation in the development of the clinical picture of peritonitis.

• Clinic, diagnostics, differential diagnosis. Modern principles of complex treatment. Features of surgical intervention. Indications for drainage and tamponade of the abdominal cavity, laparostomy, carrying out program sanitation. The role of antibiotics in the complex treatment of peritonitis. Combating hemodynamic and microcirculatory disorders, hydroid disorders, intoxication and intestinal paresis in the postoperative period.

• Methods of extracorporeal detoxification of the body. Treatment results. The role of emergency care in early diagnosis and treatment of peritonitis.

• Gynecological peritonitis. Clinic, diagnostics, treatment.

• Tuberculous peritonitis. Classification (by course, by morphological forms). Clinic, diagnostics and treatment of various forms.

**To be able to:**

• Conduct a clinical examination of patients with acute surgical diseases of the abdominal cavity.

• Formulate a detailed clinical diagnosis according to the ICD classification, justify it on the basis of a differential diagnosis.

• Determine surgical tactics. Formulate indications for conservative and surgical treatment.
• Provide emergency care to a patient with acute peritonitis at the prehospital stage.
• Probe and wash the stomach
• Draw up a medical history with a justification for the patient examination plan and indications for surgical intervention.
• Perform a digital rectal examination.
• Read and interpret the results of general clinical examinations, instrumental examinations, and X-ray results, taking into account age characteristics and semiotics of the disease.

**Literature:**


1. **https://www.saudedireta.com.br/catinc/tools/e_books/Oxford%20Handbook%20of%20Clinical%20Surgery,%204th%20Edition.pdf**
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