**MINISTRY OF THE HEALTH CARE OF UKRAINE**
**LVIV DANYLO HALYTSKY NATIONAL MEDICAL UNIVERSITY**
**CHAIR OF GENERAL SURGERY**

# GUIDELINES
# TO THE PRACTICAL LESSON

for 3rd-year students of Dentistry faculty

Topic:

**Acute peritonitis. Clinic, diagnostics, treatment.**

Academic discipline:
S u r g e r y
3-rd year dentistry faculty
Specialty: 8. 12010005
"Dentistry"

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**1. Background.**

Acute peritonitis is one of the most severe and widespread complications in abdominal surgery. Mortality in acute peritonitis reaches up to 10-40% and depends on its type and the condition of the body's defense forces. About 16-20% of acute surgical diseases of the abdominal cavity are complicated by acute peritonitis.

**2. Study objective.**

To study the etiology, pathogenesis, classification of acute peritonitis.
To know the typical clinic of acute peritonitis.
To get acquainted with the methods of laboratory and instrumental tests in acute peritonitis.
To study therapeutic tactics in acute peritonitis.
To learn the principles of current antibacterial and intravenous therapy in acute peritonitis.
To plan the examination of a patient with acute peritonitis.
To interpret the results of laboratory and instrumental tests in acute peritonitis.

**3. Basic knowledge, abilities and skills necessary for studying the topic (interdisciplinary integration).**

| Discipline | Obtained skills |
|---|---|
| Normal and topographical anatomy. | Physical examination of abdominal organs. |
| Normal and pathological anatomy. | Ability to interpret clinical manifestations in various forms of acute peritonitis. |
| Biochemistry, pharmacology. | Ability to interpret laboratory tests (CBC, biochemical assay etc.). |
| Radiology. | Ability to interpret radiological data. |
| In-patient surgery, anesthesiology, intensive care, clinical pharmacology. | Application of basic knowledge to further study the problem and pathogenetically justify the need for the use of pain management and other treatment. |

**4. Tasks for independent work in preparation for the class.**

4.1. List of basic terms, parameters. characteristics to be learned by the student in preparation for the class:

| Term | Definition |
|---|---|
| Peritoneum. | A thin translucent connective tissue lining covering the walls of the abdominal cavity (lamina parietalis peritoneum) and the surface of abdominal organs (lamina visceralis). |
| Peritonitis. | Acute or chronic inflammation of the peritoneum as a result of the influence of pathogenic microorganisms, physical and chemical factors, manifesting itself as local and general disorder of the functional state of the organism. |
| Ascitic peritonitis (spontaneous bacterial peritonitis). | The most difficult complication of ascites without evident source of intra-abdominal infection. Most often complication of liver cirrhosis, less often – nephrotic syndrome, systemic lupus erythematosus and cardiovascular failure. |
| Peritoneal dialysis. | Constant lavage of the abdominal cavity with a special solution. |
| Relaparotomy. | The method of repeated surgical treatment used in treatment of peritonitis to evaluate the condition of peritoneum and abdominal organs. |

| | |
|---|---|
| Functions of cytokines. | Role in regulation of the systemic and local inflammatory response, regulation of growth and differentiation of immune cells, control of cell regeneration. |
| Classifications of peritonitis. | By etiology; by original causing factor; by spread; by clinical course; by type of the exudate; by stages of development; by severity. |
| Methods for evaluation of the severity of the patient's condition and prognosis in peritonitis. | Mannheim Peritonitis Index, APACHE III System, Altona Peritoneal Index, IAPI Score. |

## 4. Contents of the topic:

**Anatomic and physiological features of the peritoneum.**

Peritoneum is a thin translucent type of connective tissue lining, which is covering the walls of the abdominal cavity (lamina parietalis peritoneum) and the surface of abdominal organs (lamina visceralis). The square of peritoneum approximately equals the square of the skin and reaches 2-3 $m^2$.

The histological structure of the peritoneum is presented by 6 layers that have different structure:

1. mesothelium – the uppermost layer; when its cells are irritated, a fibrin film is produced, which protects the deeper layers of the peritoneum and isolates the causing factor/structure from intact areas.
2. basal membrane – contains delicate plexuses of the reticular fibers.
3. superficial wavy collagen layer – thin, compact collagen layer located along the gut.
4. superficial diffuse elastic mesh – consists of tightly spaced fibers.
5. deep elastic mesh – presented by thick fibers joined by thin connections.
6. deep lattice collagen – mainly elastic layer.

In various parts of the abdominal cavity, the peritoneum has a different number of layers: their amount directly influences the intensity of absorption and secretion of the fluid by peritoneum.

Physiological functions of the peritoneum:

1. absorptive – the peritoneum absorbs up to 3-6 liters of liquid per hour and up to 70 liters per day.
2. exudative – pus formation.
3. protective – peritoneal fluid has bactericidal and bacteriostatic properties.
4. plastic – ability of fibrin formation.

**Etiology and pathogenesis.**

Microbes leading to the development of acute peritonitis are:

1. Gram-negative aerobes – Escherichia coli, Pseudomonas aeruginosa, Proteus spp., Enterobacteriaceae, Citrobacter spp.
2. Gram-positive aerobes – Staphylococcus spp., Streptococcus spp.
3. Gram-negative anaerobes – Bacteroides spp., Fusobacteria spp., Veilonella.
4. Gram-positive anaerobes – Clostridium spp., Lactobacillus spp., Peptococci, Peptostreptococci, Eubacterium spp.

Pathogenetically, in response to microbial contamination and subsequent production of large volume of toxins, biologically active substances are activated. Under their action, comes the stimulation of intrareceptors, the permeability of capillaries is impaired with the development of stasis, disorders of water-electrolyte and acid-base balance arise, metabolism of proteins, carbohydrates and fats is disrupted. This leads to the development of tissue hypoxia, acidosis, increased blood clotting, development of DIVC syndrome.

Later on, a decrease in the muscular tone of the intestine occurs followed by the paresis and

sequestration of large amount of fluid, salts, trace elements, proteins, carbohydrates, fats and blood cells both into the lumen of the intestine and abdominal cavity. As a result of ischemic changes in the wall of the intestine, the latter becomes permeable for germs.

In acute peritonitis, a significant amount of cytokines is produced, leading to impaired function of all organs and systems of the body, especially immune and cardiovascular. Particularly pronounced mediator aggression is associated with the response to endotoxins of Gram-negative microbes.

**Classification of peritonitis.**

*By etiology:*

• infectious – non-specific or specific. Specific peritonitis is caused by germs that are not normal residents of the gastrointestinal tract (Gonococci, Pneumococci, Tuberculosis). Non-specific refers mainly to autochtonous germs.

• non-infectious – due to the action of chemicals and/or organ secretions (bile, gastric juice, urine, blood) as a result of destructive process/trauma.

*By origin:*

• primary – due to the entry of germs into the abdominal cavity by hematogenous and/or lymphogenous way, as well as by microbial translocation, but absent destructive process/trauma.

• secondary – due to the entry of germs into the abdominal cavity during acute destructive surgical diseases of the abdominal organs, abdominal trauma or postoperative complications (dehiscence of inter-intestinal anastomosis etc.) by leakage of the contents of hollow organs alongside exudate into abdominal cavity.

• tertiary – a low-symptomatic form of postoperative peritonitis, the severity of which is caused by highly resistant nosocomial flora without macroscopically evident source.

*By spread:*

• local (delimited, not-delimited) – the inflammatory process is localized to 1-2 quadrants of the abdominal cavity.

• diffuse – the inflammatory process spreads to 3 or more quadrants.

*By the type of the exudate:*

• serous
• serous-fibrinous
• fibrinous-purulent
• purulent
• bile
• hemorrhagic
• fecal
• chemical.

*By phases of development:*

• reactive - <24 hours after the onset of the disease.
• toxic - 24-72 hours after the onset of the disease.
• terminal - >72 hours. from the onset of the disease.

*By severity:*

• I - mild: no manifestations of endotoxicosis are seen.

• II - moderate: elimination of endotoxicosis is possible by removal/localization of the primary cause of peritonitis.

• III - severe: after removal/localization of the primary cause of peritonitis, endotoxicosis can be managed only by the use of auxilliary methods of detoxification (hemosorption, lymphosorption, plasmophoresis, intensive therapy etc.) and/or relaparotomy/-ies.

• IV - terminal: development of multiple organ failure, frequently lethal.

**5. Clinical symptoms of acute peritonitis.**

Subjective symptoms include abdominal pain, nausea, vomiting, digestive disorders (gas retention, bloating, delayed bowel movements).

Objective signs to be evaluated:

1. General:
   - patient's behaviour.
   - patient's appearance.
   - cardiovascular activity, respiratory functions.
   - morphological and biochemical blood parameters.

2. Local (related to objective changes in the abdominal cavity and, specifically, anterior abdominal wall):
   - abdominal wall, its participation in the act of breathing.
   - results of palpation, percussion and auscultation.
   - results of rectal and vaginal examination.
   - results of radiological (X-ray, ultrasound) and instrumental (laparocentesis, laparoscopy) methods of examination.

**Diagnosis of acute peritonitis.**

The diagnosis of acute peritonitis is based on the analysis of patient's case history, complaints, data of objective examination of the abdomen, as an additional measure – evaluation of function of vital systems, changes in the blood and other bodily secretions. Auxilliary methods of examination are chosen to prove/disapprove the diagnosis, especially in doubtful cases.

**Differential diagnostics.**

Differential diagnostics of acute peritonitis should be done primarily with diseases that resemble its clinical course, but do not require surgical treatment: acute thoracic (lower-lobe pneumonia, basal pleurisy etc.), cardiovascular (myocardial infarction, cardiac tamponade, abdominal manifestations of rheumatic disease etc.), uro-gynecological (acute adnexitis, renal colic, acute urine retention, extra-uterine pregnancy etc.) pathology as well as acute food poisoning.

**Treatment of acute peritonitis.**

Current treatment of acute peritonitis is based on 3 principles:

1. Intensive, pathogenetically-oriented pre-operative preparation.
2. Emergency surgery (principles of "source-control" amd "damage-control").
3. Rational antibacterial therapy (targeted on the locally proven sensitive microbes).
4. Complex intensive therapy aimed at correction of disorders of homeostasis.

Pre-operative preparation should be performed in short terms. Its purpose is correction of the basic vital parameters (oxygenation, control of volemia, electrolyte and acid-base balance, nutritional status etc.). Pre-operative preparation of patients in the toxic and terminal stages of acute peritonitis should be performed in the Intensive care unit. Narcosis is the method of choice of anesthesia in most cases of acute peritonitis.

**<u>Materials for self-control:</u>**

**Theoretical questions to the topic:**

1. Anatomical and functional features of the peritoneum.
2. Etiology and pathogenesis of acute peritonitis.
3. Classification of acute peritonitis.
4. Methods of examination of patients with acute peritonitis.
5. Clinic of acute peritonitis.
6. Differential diagnosis of acute peritonitis.
7. Diagnostic program in a patient with acute peritonitis.
8. Therapeutic tactics in patients with acute peritonitis.
9. Peculiarities of preoperative preparation for acute peritonitis
11. Patient care in the postoperative period.

**Tests for self-control.**

**1. Main physiological functions of the peritoneum are:**

a. absorptive, exudative, protective, plastic.
b. antiseptic, exudative, protective, plastic.
c. exudative, hemostatic, hormonal, detoxicative.
d. protective, exudative, cushioning, respiratory.

**2. Pathological conditions that lead to the development of acute peritonitis are:**

a. acute destructive diseases of abdominal organs, acute ischemic conditions of abdominal organs, abdominal trauma, intraperitoneal postoperative complications, portal hypertension.
b. acute destructive diseases of abdominal organs, acute ischemic conditions of abdominal organs, abdominal trauma, intraperitoneal postoperative complications, ischemic heart disease.
c. acute destructive diseases of abdominal organs, acute ischemic conditions of abdominal organs, abdominal trauma, intraperitoneal postoperative complications, urolithiasis.
d. acute destructive diseases of abdominal organs, acute ischemic conditions of abdominal organs, abdominal trauma, intraperitoneal postoperative complications, diabetes mellitus.

**3. Pathogenetic stages of of acute peritonitis are:**

a. effect of etiological factor, endotoxicosis, activation of biologically-active substances, increase of capillary permeability, stasis, hypercoagulation, metabolic acidosis, systemic disorders, tissue hypoxia, multiorgan failure.
b. effect of etiological factor, inflammation of the peritoneum, exudate formation, hyperthermia, hypotension.
c. effect of etiologic factor, inflammation of the peritoneum, exudate formation, hepato-renal failure, enteritis.
d. effect of etiological factor, inflammation of the peritoneum, exudate formation, intoxication syndrome, chronic peritonitis.

**4. Classification of acute peritonitis by the spread of inflammatory process in the abdominal cavity is:**

a. local, non-delimited, diffuse.
b. local, delimited.
c. local, non-delimited.
d. local, diffuse.

**5. Stages of clinical course of acute peritonitis are:**

a. reactive, toxic, terminal.
b. reactive, toxic, cardiovascular.
c. reactive, toxic, renal.
d. reactive, toxic, recovery.

**6. The main clinical signs of the toxic stage of acute peritonitis are:**

a. abdominal pain and swelling, vomiting, dry tongue, fever, tachycardia.
b. abdominal pain and swelling, vomiting, dry tongue, fever, tachycardia, loss of consciousness.
c. abdominal bloating, vomiting, dry tongue, fever, tachycardia, hypertension, anuria.
d. abdominal bloating, vomiting, dry tongue, fever, tachycardia, acute urinary retention.

**7. The main clinical signs of terminal stage peritonitis are:**

a. abdominal bloating, intoxication, multiple organ failure.

b. abdominal bloating, intoxication, multiple organ failure, hypertension, pyuria.

c. abdominal bloating, intoxication, multiple organ failure, hyperglycemia, glucosuria.

d. positive Bloomberg's symptom, abdominal wall asymmetry, intoxication, multiple organ failure.

**8. Signs of local peritonitis are:**

a. pain, muscular tension, positive Bloomberg's symptom above primary source.

b. pain, muscular tension, positive Bloomberg's symptom above primary source, abdominal wall asymmetry.

c. muscular tension, positive Bloomberg's symptom above primary source, abdominal bloating.

d. pain, muscular tension, positive Bloomberg's symptom above primary source, arterial hypotension, proteinuria.

**9. X-ray signs of acute peritonitis include:**

a. aeroentery, horizontal fluid levels in the small intestine lumen, free gas under the right diaphragmatic dome.

b. horizontal fluid levels in the small intestine lumen, fuzzy diaphragmatic contours, acceleration of passage of barium sulphate through colon.

c. free gas under the right diaphragmatic dome, acceleration of passage of barium sulphate through small intestine, pneumatization of colon.

d. dilation of the stomach, aerocolia, dolichocolon.

**10. Additional instrumental methods of diagnostics in acute peritonitis could be:**

a. X-ray, ultrasound, laparocentesis, laparoscopy.

b. X-ray, ultrasound, laparocentesis, laparoscopy, retrograde cholepancreatography.

c. X-ray, laparocentesis, laparoscopy, virtual colonoscopy, biopsy.

d. X-ray, ultrasound, laparocentesis, laparoscopy, biopsy, angiography.

**11. Basic principles of treatment of acute peritonitis:**

a. emergency surgery, antibacterial therapy, intravenous therapy, peridural anesthesia.

b. emergency surgery, antibacterial therapy, intravenous therapy, diet.

c. emergency surgery, antibacterial therapy, intravenous therapy, correction of blood sugar.

d. emergency surgery, antibacterial therapy, intravenous therapy, drainage of abscess.

**Situational tasks for self-control.**

1. Male patient, 24, complains of abdominal pain without clear localization, vomiting, diarrhoea. Feels sick for 12 hours. Initially, the pain occurred in the epigastric region. After 4 hours, the pain migrated into right epigastric region and had spread to the entire abdominal cavity. On examination, the tongue is moist, upon palpation the abdomen is not swollen, takes limited part in the act of breathing. Symptoms of peritoneal irritation are not clearly expressed. Body temperature 37,6°C, pulse 90 bpm, BP 120/70 mm Hg, leukocytes $12{,}4 \cdot 10^9$, rods – 9%. Upon radiography of the abdomen, a moderate aeroentery was marked, free gas was not radiologically detected in free abdominal cavity.

Establish and substantiate the preliminary diagnosis. Determine further curative and diagnostic tactics.

2. The patient complains on moderate abdominal pain after falling from height, he had single vomiting. Skin is of normal colour, tongue is moist, belly is moderately bloated and painful on palpation in left half, symptoms of peritoneal irritation are doubtful. Body temperature 37,2°C,

pulse 94 bpm, BP 120/70 mm Hg, leukocytes $14,3 \cdot 10^9$, rods – 11%. Upon radiography of the abdomen, a moderate aeroentery was marked, free gas was not radilogically detected in free abdominal cavity.

Establish and substantiate the preliminary diagnosis. Determine further curative and diagnostic tactics.

3. Patient, 57, suffering from chronic cirrhosis due to chronic alcohol intoxication, experienced moderate abdominal pain. On examination – sclerae and skin are moderately jaundiced. The abdomen is enlarged in size due to ascites. Body temperature is 38,2°C, pulse 104 bpm, BP 110/60 mm Hg, leukocytes $17,3 \cdot 10^9$, rods – 14%. At ultrasound imaging of abdominal organs, signs of cirrhosis of the liver are verified, significant amount of fluid is present in free abdominal cavity.

Establish the preliminary diagnosis. Determine further diagnostic steps.

4. Patient, 32, suffering from peptic ulcer disease of the duodenum in the past, experienced pain in the epigastric region, the intensity of which decreased 3 hours after its onset. After 3 days, the patient began complaining of pain in the right hypochondrium, nausea, fever up to 38,0°C. Palpation in the right hypochondrium had shown positive Bloomberg's symptom. Pulse is 102 bpm, BP 112/70 mm Hg, leukocytes $18,2 \cdot 10^9$, rods – 16%.

Establish the preliminary diagnosis and enumerate possible causes of this condition. Choose additional research methods to confirm the diagnosis and estimate the curative tactics.

5. In a patient of 68 y. o., suffering from gallstone disease, an attack of acute cholecystitis occurred. Initially, conservative antispasmodic therapy was effective. However, on the 3rd day from the onset of the disease, there was recurrent pain in the right hypochondrium, body temperature had increased to 38,1°C. Palpation in the right hypochondrium determined local infiltration. Pulse was 98 bpm, BP 130/80 mm Hg, leukocytes $17,4 \cdot 10^9$, rods – 15%. At ultrasound imaging, gallstones were seen in the gallbladder, as well as edema of its wall.

Make a preliminary diagnosis. Define curative tactics.

6. A patient, after low anterior rectal resection with formation of colorectal anastomosis, on the 5th postoperative day had moderate pain in the lower abdomen. Upon palpation, the abdomen is swollen and painful in its lower part, symptoms of peritoneal irritation are locally positive, there is a fecal exudate out of drainage tubes. Body temperature is 38,2°C, pulse 102 bpm, BP 110/60 mm Hg, erythrocytes $3,8 \cdot 10^{12}$, hemoglobin 94 g/L, leukocytes $17,8 \cdot 10^9$, rods – 18%.

Make a preliminary diagnosis. What surgical operation is supposed to be done ?

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