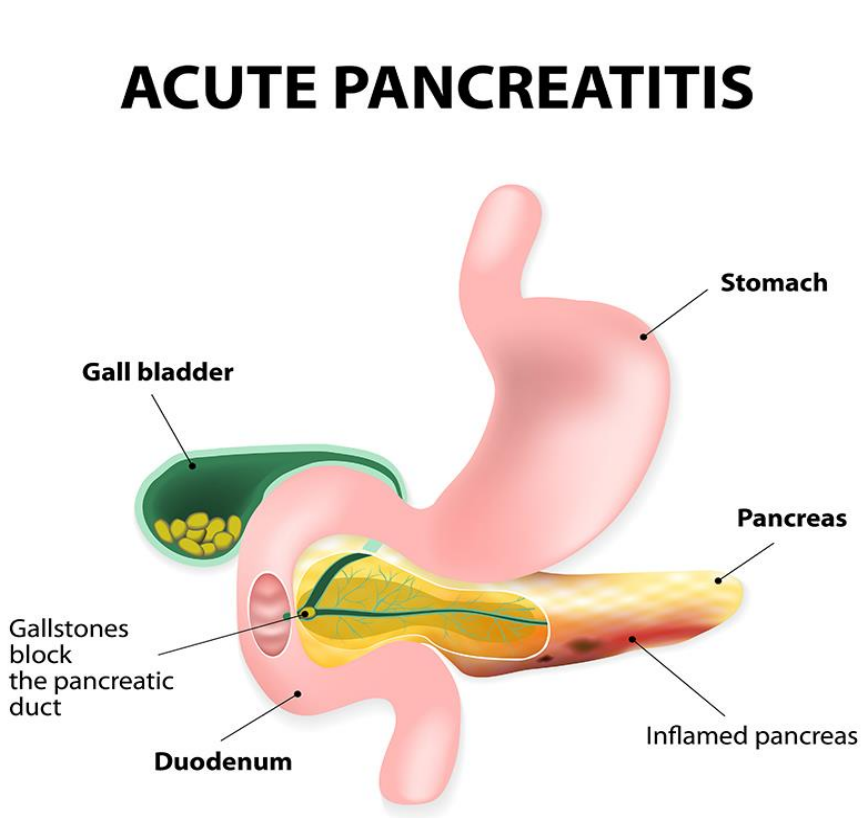
ROYAL METROPOLITAN UNIVERSITY

Department of Clinical Disciplines

# ACUTE PANCREATITIS

EDUCATIONAL AND METHODOLOGICAL MANUAL

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The educational and methodical manual is devoted to acute pancreatitis. Objectives and tasks are spelled out in a form understandable to students. The presentation of the material is intended to provide students with a unified picture, since this material is based on a universally recognized academic structure in the world, starting with definition, etiology / pathogenesis and ending with diagnosis and treatment. In order to check the level of students' knowledge, control questions, tests and tasks, and situational problems are given.

The educational and methodical manual is written in English at the highest level and is intended for international students of the 6th semester of the Faculty of "Medical Science" of the RMU.

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# 1. Introduction

## 1.1. Relevance.

Currently, the prevalence of acute pancreatitis is 32-389 people per 1 million population, the mortality rate from this disease ranges from 6 to 12 people per 1 million population. At the present stage, there have been significant changes in the statistics of the frequency of diseases of large megacities that pass through the ambulance under the brand “acute abdomen": for the period from 2000 to 2009, acute pancreatitis confidently occupied the first place. In recent years, the number of patients with acute pancreatitis has decreased, but despite the decrease in the total number of patients, the share of AP in the structure of diseases of the "acute abdomen" remains at a fairly high level (25% - 35%), ranks second and is second only to acute appendicitis. During this period, there was a tendency to reduce the overall mortality rate in acute pancreatitis from 4.0% - 4.5% to 2.5%-3.5%, but postoperative mortality remains at a fairly high level (20% -25%) [1].

## 2.1. The purpose of the lesson

To familiarize students with the etiology, the main links of the pathogenesis of acute pancreatitis, to explain the classification, syndromic diagnosis of various clinical forms of this disease, to demonstrate patients with typical manifestations of acute pancreatitis, to analyze the issues of tactics, medication and surgical treatment.

## 3.1. Lesson objectives

**The student should know:**

-Etiology and pathogenesis of acute pancreatitis.

-Classification of acute pancreatitis.

-Clinical manifestations of various variants of acute pancreatitis (edematous form, sterile pancreatic necrosis, infected pancreatic necrosis).

-The main methods of clinical, laboratory and instrumental diagnostics of acute pancreatitis and its infectious complications.

-Features of drug therapy of acute pancreatitis, depending on its clinical form.

-Indications for surgical treatment of pancreatic necrosis, technologies of surgical interventions used for the treatment of pancreatic necrosis.

**The student must be able to**

-Conduct a survey of patients with acute pancreatitis, identify anamnestic, clinical features inherent in various clinical forms of acute pancreatitis.

-Perform an objective examination of patients with the determination of specific symptoms.

-Interpret the main clinical indicators and data of X-ray examinations used for the diagnosis of acute pancreatitis.

-Develop a programme of medical treatment of patients with edematous form of pancreatitis and pancreatic necrosis.

**Requirements for the initial level of knowledge**. For a successful and complete development of the topic, you must repeat:

1. Anatomy and topographic anatomy of the pancreas.
2. Normal and pathological physiology of the pancreas.
3. The concept of the systemic inflammatory response syndrome (SIRS) and the main mediators of inflammation.

## 2. Definition

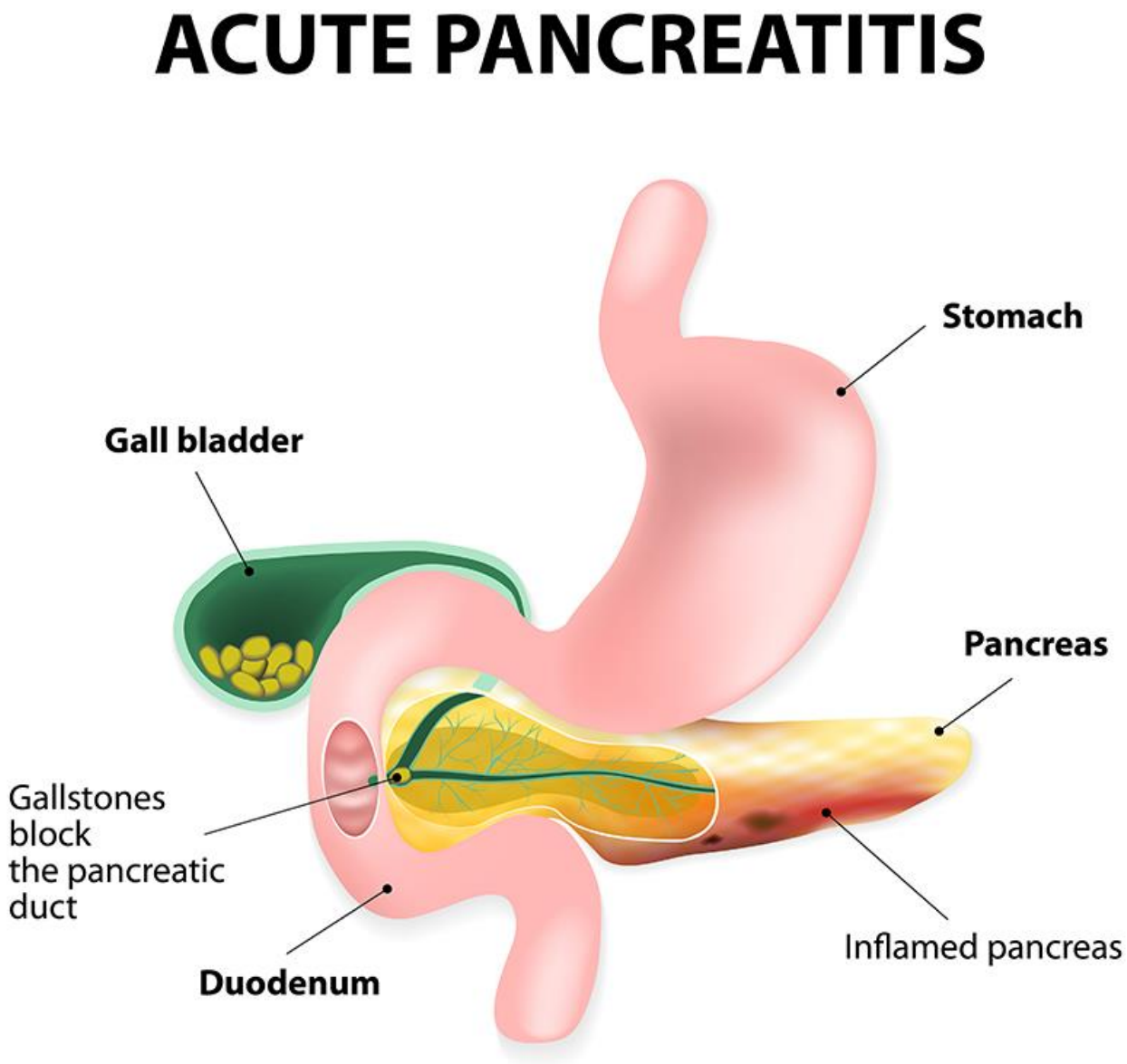


Fig. 1.1 Acute pancreatitis

Acute pancreatitis is initially an aseptic inflammation of the pancreas, in which it is possible to damage the surrounding tissues and distant organs, as well as systems.

## 3. Etiology and pathogenesis

The following etiological forms of acute pancreatitis are distinguished:

1. Acute alcoholic-alimentary pancreatitis-55%.

2. Acute biliary pancreatitis (occurs due to bile reflux into the pancreatic ducts in biliary hypertension, which occurs, as a rule, due to cholelithiasis, sometimes-from other causes: diverticula, papillitis, opisthorchiasis, etc.) - 35%.

3. Acute traumatic pancreatitis (due to pancreatic injury, including surgery) 2-4 %.

4. Other etiological forms of the cause: autoimmune processes, vascular insufficiency, vasculitis, medications (hypothyazid, steroid and non – steroidal hormones, mercaptopurine), infectious diseases (viral mumps, hepatitis, cytomegalovirus), allergic factors (lacquers, paints, odors of building materials, anaphylactic shock), dyshormonal processes during pregnancy and menopause, diseases of nearby organs (gastroduodenitis, penetrating ulcer, tumors of the hepatopancreatoduodenal region) – 6-8%.

The leading role in the pathogenesis of toxemia in acute pancreatitis belongs to the enzymes of the pancreas: trypsin, lipase, phospholipase-A2, lysosomal enzymes that cause oxidative stress, lipid distress syndrome, capillary thrombosis, hypoxia, acidosis,hypermetabolism, damage to cell membranes and endothelium.

Primary factors of aggression:

a) pancreatic enzymes: trypsin, chymotrypsin, - cause proteolysis of tissue proteins;

b) phospholipase A2 destroys cell membranes;

c) lipase hydrolyzes intracellular triglycerides to fatty acids and, when combined with calcium, leads to lipolytic necrosis in the pancreas, retroperitoneal tissue and mesentery of the small and large intestine;

d) elastase destroys the vascular wall and interstitial connective tissue structures, which leads to necrosis.

Secondary factors of aggression. Pancreatic enzymes activate the kallikrein-kinin system with the formation of biologically active substances: bradykinin, histamine, serotonin, which lead to increased vascular permeability, microcirculation disorders, edema, increased exudation and microthrombosis, ischemia, hypoxia and tissue acidosis.

Tertiary factors. Macrophages, mononuclear cells, neutrophils against the background of microcirculation disorders, hypoxia produce cytokines (interleukin 1,6

and 8, tumor necrosis factor, platelet activation factor, prostaglandins, thromboxane, leukotrienes, nitric oxide, inhibition of the immune status.

Factors of aggression of the fourth order. Cytokines, enzymes, and metabolites of various natures formed in the pancreas, adipose tissue, intestinal wall, and abdominal cavity increase the permeability of the intestinal wall, translocation of the intestinal flora occurs, and promote the entry of toxins into the portal and systemic blood flow and lymphatic channel with damage to the target organs: liver, lungs, kidneys, heart, brain, intestines, stomach and intestinal mucosa.

Factors of aggression and organ dysfunction create a "mutual burden" syndrome.

Phases of acute pancreatitis. Edematous (interstitial) pancreatitis occupies 80-85% of the disease structure in frequency. It is characterized by a mild degree of severity of the disease and the rare development of local complications or systemic disorders, it does not have a phase course.

Necrotic pancreatitis (pancreonecrosis) occurs in 15-20% of patients, is always clinically manifested by a moderate or severe degree of the disease, has a phase course of the disease with two peaks of mortality – early and late. The early phase, which usually lasts for the first two weeks, is followed by a second or later phase, which can last for a period of weeks to months. It is advisable to consider these two phases separately, since each phase corresponds to a certain clinical form, and, consequently, a certain therapeutic diagnostic algorithm.

Phase I – early, in turn, is divided into two periods:

Phase IA, usually the first week of the disease. During this period, the formation of foci of necrosis in the pancreatic parenchyma or the surrounding tissue of various volumes and the development of endotoxicosis occurs. Endotoxicosis is manifested by mild or deep systemic disorders in the form of organ (multi-organ) insufficiency.

The maximum period of formation of necrosis in the pancreas is usually three days, after this period it does not progress further. However, in severe pancreatitis, the period of its formation is much shorter (usually 24-36 hours). In the abdominal cavity, there is an accumulation of enzymatic effusion (enzymatic peritonitis and

parapancreatitis), which is one of the sources of endotoxicosis. The average severity of the disease is manifested by transient dysfunction of individual organs or systems. In severe forms of the disease, the clinical picture may be dominated by the phenomena of organ (multi-organ) insufficiency: cardiovascular, respiratory, renal, hepatic, etc.

Phase I, usually the second week of the disease. It is characterized by the reaction of the body to the formed foci of necrosis (both in the pancreas and in the parapancrealtissue). Clinically, the phenomena of resorptive fever prevail, peripancreatic infiltrate is formed.

Phase II – late, sequestration phase (usually begins with the 3rd week of the disease, can last several months). Sequesters in the pancreas and in the retroperitoneal tissue usually begin to form from the 14th day from the onset of the disease. With the rejection of large large fragments of necrotic pancreatic tissue, depressurization of its ductal system and the formation of an internal pancreatic fistula can occur. The configuration of pancreatic necrosis (localization, depth, relation to the main pancreatic duct, etc.) and the volume of the remaining viable pancreatic parenchyma depend on: the number, extent and rate of spread of fluid formation in the retroperitoneal space, the risk of infection and the development of other complications.

There are two possible variants of the flow of this phase:

aseptic sequestration-sterile pancreatic necrosis is characterized by the formation of an isolated accumulation of fluid in the area of the pancreas and postnecroticpseudocysts of the pancreas;

septic sequestration occurs when necrosis of the pancreatic parenchyma and parapancreal tissue is infected with further development of purulent complications.

The clinical form of this phase of the disease is infected pancreatic necrosis, which can be delimited (abscess) or non-delimited (purulent-necrotic parapancreatitis). With the progression of purulent complications, infected pancreonecrosis may have its own complications (purulent-necrotic occlusions, abscesses of the retroperitoneal space and abdominal cavity, purulent peritonitis,

arrosive and gastrointestinal bleeding, digestive fistulas, sepsis, etc.) with the development of endotoxicosis of infectious genesis, organ (multi-organ) insufficiency.

## 4. Classification.

Classification of acute pancreatitis of the Russian Society of Surgeons (2014) designed with a due account of the Classification of Atlanta–92 and its modifications, offered in the city of Cochin in 2011 (international Association of Pancreatology, International Association of Pancreatology) and the International Working Group on the Classification of the Acute Pancreatitis (Acute Pancreatitis Classification Working Group) in 2012 [2-4].

1. Acute pancreatitis thanks to an extent. Pancreonecrosis in this form of acute pancreatitis is not formed (edematous pancreatitis) and organ failure does not develop.

2. Moderate acute pancreatitis. It is characterized by the presence of either one of the local manifestations of the disease: peripancreatic infiltrate, pseudocyst, delimited infected pancreonecrosis (abscess), or/and the development of general manifestations in the form of transient organ failure (no more than 48 hours).

3. Severe acute pancreatitis. It is characterized by the presence of either undifferentiated infected pancreonecrosis (purulent-necrotic parapancreatitis), or/and the development of persistent organ failure (more than 48 hours). The diagnosis of acute pancreatitis of a mild, moderate or severe degree is established upon the fact of a completed case of the disease.

## 5. Diagnostics

Diagnosis of AP refers to emergency measures. Patients with suspected AP are subject to emergency hospitalization in the surgical department of a multidisciplinary hospital.

Complaints and anamnesis

The basis for the diagnosis of acute pancreatitis at the initial examination of the patient is the classic triad of symptoms – severe pain in the epigastrium with radiation in the back or shingles, repeated vomiting and muscle tension in the upper half of the abdomen. Most often, the appearance of symptoms is preceded by a large meal or alcohol, the presence of cholelithiasis. A typical pain syndrome occurs in acute pancreatitis always. Usually it is intense, persistent, not stopped by antispasmodics and analgesics. The onset of acute pancreatitis should be determined by the time of occurrence of abdominal pain, and not by the time of admission of the patient to the hospital. It is possible to identify the moment of the onset of the disease with a thorough collection of anamnesis. With severe pain syndrome, the injection of antispasmodic and non-steroidal anti-inflammatory drugs is permissible. When transferring a patient from one hospital to another, the onset of the disease should be considered the time when the pain syndrome appears at the initial request for medical help. The clinical manifestations of acute pancreatitis depend on the morphological form, the phase of the disease, the severity of the systemic inflammatory response syndrome and the development of organ (multi-organ) insufficiency. Each phase of the disease corresponds to a certain clinical and morphological form of AP, so it is advisable to consider the diagnosis of AP in the corresponding phases of the disease.

Primary protocol of diagnosis and tactics in acute pancreatitis in the IA phase of the disease.

As a rule, it is carried out in the emergency department or emergency department.

To establish the diagnosis of acute pancreatitis (after exclusion of other surgical pathology), it is recommended to use a combination of at least two of the following revealed features:

a) the typical clinical picture (intense antispasmodics intractable pain zoster character, indomitable vomiting, bloating, alcohol, spicy foods or the presence of cholelithiasis

in history, etc.);

b) the characteristic features of the ultrasound data: the increase in size, decrease in echogenicity, necdat contours of the pancreas, the presence of free fluid in the abdominal cavity;

c) hyperfermentemia (hyperamylasemia or hyperlipasemia), exceeding the upper limit of the norm by three times or more.

The level of credibility of the recommendation "B"

Comments: If the diagnosis of acute pancreatitis is established on the basis of methods a), b) and c), then performing multi-spiral computed tomographic angiography (MSCTA) or magnetic resonance imaging (MRI) for the diagnosis of acute pancreatitis is not recommended.

The level of credibility of the recommendation "B".

Computed tomography.

Fig.5.1 CT of acute pancreatitis

Early MSCTA (MRI) is recommended in the following cases:

- Uncertainty of the diagnosis and differential diagnosis with other diseases.
- The need to confirm the severity of the identified clinical prognostic signs of severe AP.
- Lack of effect from conservative treatment.

The level of credibility of the recommendation "C".

For the diagnosis of pancreatic necrosis in the optimal time (and to assess the total volume of pathological changes in the chest, abdominal cavity and retroperitoneal tissue), it is recommended to perform MSCTA (MRI) on the 4th – 14th day of the disease.

The level of credibility of the recommendation "B".

MSCTA (MRI) is recommended to be performed on the eve of an invasive intervention.

The level of credibility of the recommendation "C".

Subsequent MSCTA (MRI) is recommended to be performed with the progression of the disease, in the absence of the effect of treatment and to clarify the location of the foci of suppuration before performing drainage interventions.

The level of credibility of the recommendation "C".

Comments: The use of the Balthazar CT-index of pancreatitis severity in clinical practice is not a mandatory diagnostic study. It is desirable to use it to predict the severity of the disease.

Protocol for the diagnosis and monitoring of peripancreatic infiltrate in phase I of the disease.

The second week of the disease is characterized by the onset of a period of aseptic inflammatory reaction to the foci of necrosis in the pancreas and surrounding tissue, which is clinically expressed by the appearance of infiltrate in the epigastric region (local component) and resorptive fever (systemic component of inflammation). Peripancreatic infiltrate (PI) and resorptive fever are natural signs of severe or moderate-severe pancreatitis, whereas in mild pancreatitis these signs are not detected.

In addition to clinical signs (peripancreatic infiltrate and fever) in the second week early phase of AP, it is recommended to determine Laboratory parameters of systemic inflammatory response syndrome: a leukocytosis with a left shift, lymphopenia, increased ESR, increased concentrations of fibrinogen, C-reactive protein, etc.; - ULTRASONIC signs of PI (continued increase in the size of the pancreas, necdat its contours and appearance of fluid in parapancreatic tissue).

The level of credibility of the recommendation "D".

To monitor peripancreatic infiltrate, it is recommended to perform a dynamic study of clinical and laboratory parameters and repeated ultrasound (at least 2 studies in the second week of the disease). The level of credibility of the recommendation "D".

At the end of the second week of the disease, it is recommended to perform a computed tomography of the pancreatic area,

The level of credibility of the recommendation "C".

Comments: By this time, the vast majority of patients have one of the three possible outcomes of phase IB:

Resorption, in which there is a reduction of local and general manifestations of an acute inflammatory reaction.

Aseptic sequestration of pancreatic necrosis with a possible subsequent outcome in the pancreatic pseudocyst: the preservation of the size of the PI with the normalization of well-being and the subsiding of the SIRS against the background of persistent hyperamylasemia.

Septic sequestration (development of purulent complications)

Protocol for the diagnosis and monitoring of pancreatic pseudocysts in phase II of the disease (in the phase of aseptic sequestration).

The clinical form of acute pancreatitis in the phase of aseptic sequestration is a postnecrotic pseudocyst of the pancreas, the formation period of which is from 4 weeks and on average up to 6 months.

In the aseptic sequestration phase, it is recommended to use the following criteria for verifying a pancreatic cyst:

- Subsiding of the syndrome of a systemic inflammatory reaction against the background of persistent hyperamylasemia.

The level of credibility of the recommendation "D".

- An increase in the size of the fluid accumulation in the parapancreal tissue by the 5th week of the disease and the appearance of a wall in it according to ultrasound, CT. The level of persuasiveness of recommendation "C".

- In the absence of complications, the patient can be discharged for outpatient treatment. The size of the cyst should be monitored according to ultrasound data (1 time in 2-4 weeks).

The level of credibility of the recommendation "D".

Comments: If the aseptic sequestration does not open the ductal system of the pancreas, then the formation of a cyst does not occur. In this case, as a rule, there is a resorption of the peripancreatic infiltrate (reduction of fluid accumulation in the pancreas) in terms of up to 4 weeks. This period is recommended for patients to be carried out under dynamic medical supervision (it is permissible on an outpatient basis).

Protocol for the diagnosis of purulent complications of acute pancreatitis in phase II of the disease (in the phase of septic sequestration).

Infection of the focus of pancreatogenic destruction occurs, on average, at the end of the 2nd – beginning of the 3rd week from the onset of the disease. However, with late admission of the patient, inadequate treatment, or after too early and hasty surgery, infection of the areas of pancreatic necrosis and purulent-destructive complications can develop earlier, bypassing the period of aseptic destruction ("phase crossing"). The clinical form of acute pancreatitis in the phase of septic sequestration (the third week from the onset of the disease and more) is infected pancreonecrosis: delimited-pancreatic abscess (PA) or non – delimited-purulent necrotic parapancreatitis (GNPP) of various degrees of prevalence. An important point is the timely diagnosis of infection and verification of clinical and morphological forms of pancreatogenic infection.

For the verification of pancreatic abscess or purulent-necrotic parapancreatitis, it is recommended to use:

1). Clinical and laboratory manifestations of a purulent focus:

- Progression of clinical and laboratory parameters of acute inflammation in the third week of AP.

The level of credibility of the recommendation "C".

Markers of acute inflammation (increased fibrinogen 2 times and a higher "C"-reactive protein, procalcitonin, etc.).

The level of credibility of the recommendations of the "S".

2)MSCTA, MRI, ultrasound (the increase in the monitoring process liquid formations, identification of devitalized tissue and/or the presence of gas bubbles).

The level of credibility of the recommendation "B".

3). Positive smears and cultures of the aspirate, obtained by fine-needle puncture.

The level of credibility of the recommendation "B".

Comments: If the methods fail to detect signs of infection, it is recommended to make a decision on the presence of purulent complications in patients and indications for surgical treatment on the basis of the laboratory-clinical minimum.

## 6. Treatment

### 6.1.Surgical treatment.

Before giving clinical recommendations, we will give a general overview of existing methods of surgical treatment for acute pancreatitis.

#### 6.1.1. Minimally invasive methods.

Minimally invasive methods can be the main and auxiliary in the treatment of necrotizing pancreatitis, in some cases they allow you to postpone surgical treatment to a more favorable later date. The methods of minimally invasive surgery are:

### 6.1.2. Endoscopic methods.

Intense pain syndrome, not relieved by narcotic analgesics, rapidly progressing jaundice, the absence of bile in the duodenum in FGDS, signs of biliary hypertension according to ultrasound data indicate the insertion of a gallstone in the LDP. In this case, it is urgently necessary to restore the passage of bile and pancreatic juice. The optimal method is endoscopic papillosphincterotomy (EPST).

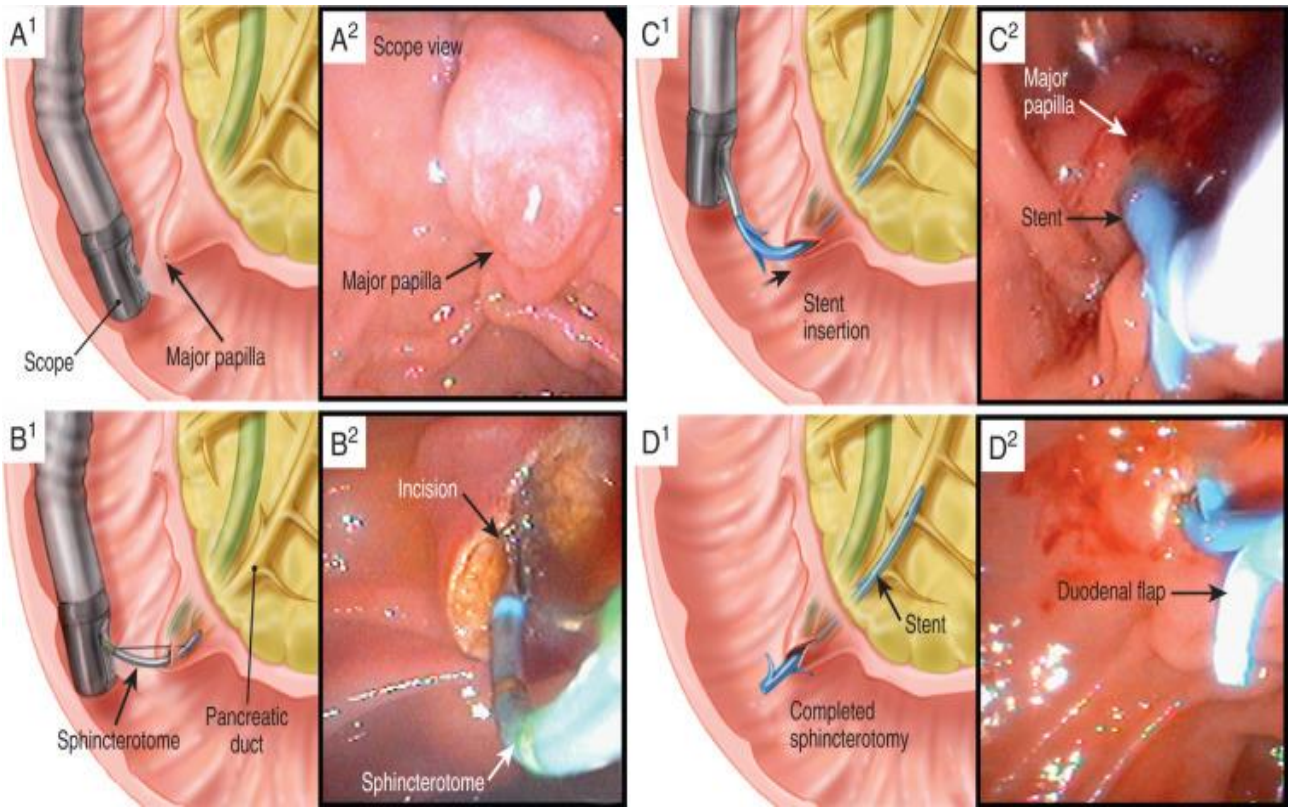


Fig. 6.1.2.1 Endoscopic papillosphincterotomy (EPST)

With AP and a stone embedded in the LDP, EPST is performed without endoscopic retrograde cholangiopancreatography.

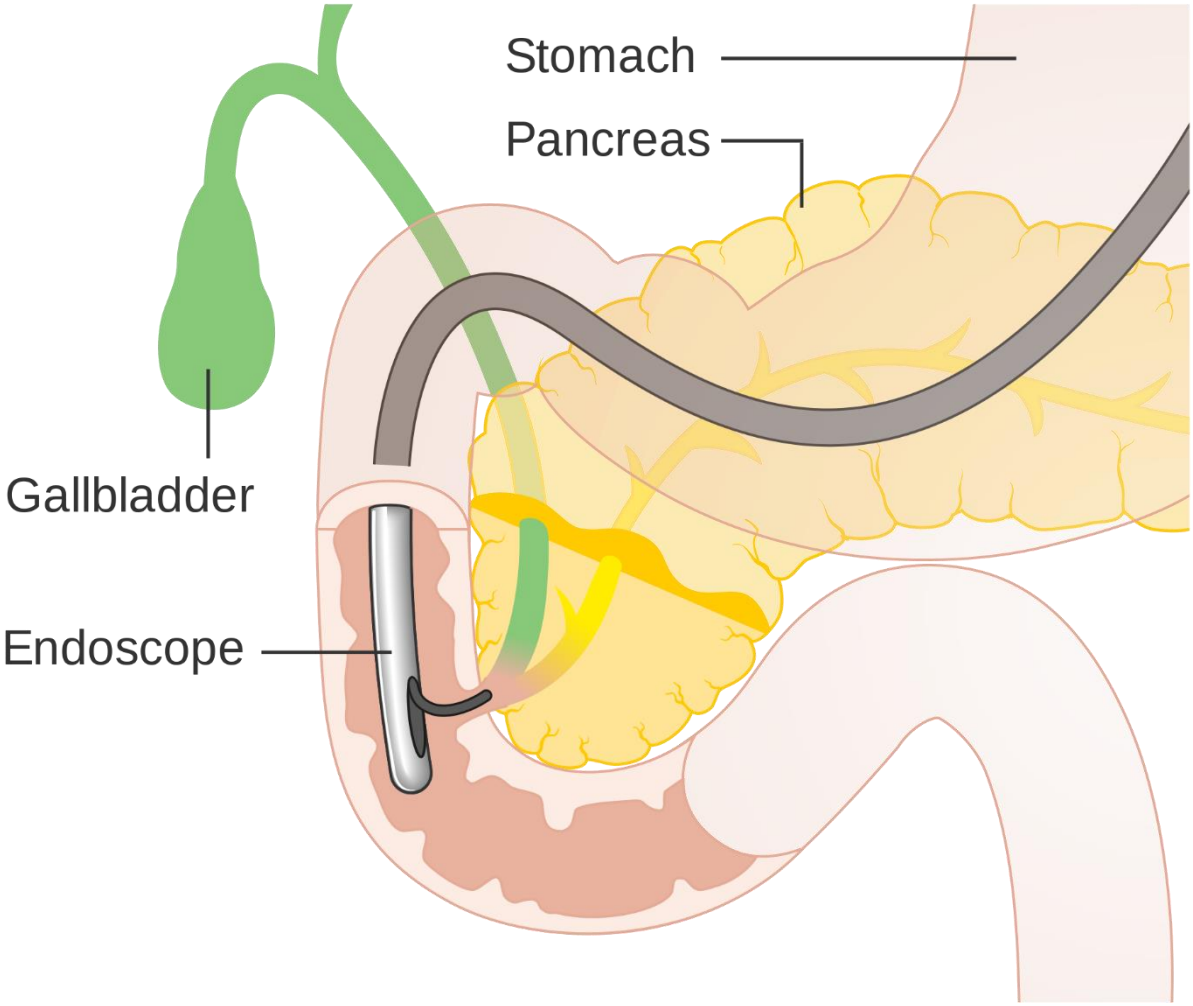


Fig. 6.1.2.2. Endoscopic retrograde cholangiopancreatography.

### 6.1.3. Puncture and drainage of pancreatogenic fluid formations under the control of ultrasound (or CT).

Ultrasound-guided drainage is used to treat acute pancreatic fluid accumulations, acute pseudocysts, and pancreatic abscesses. The method provides for puncture and placement of catheters of various diameters in the pathological focus, followed by its sanitation with antiseptic solutions.

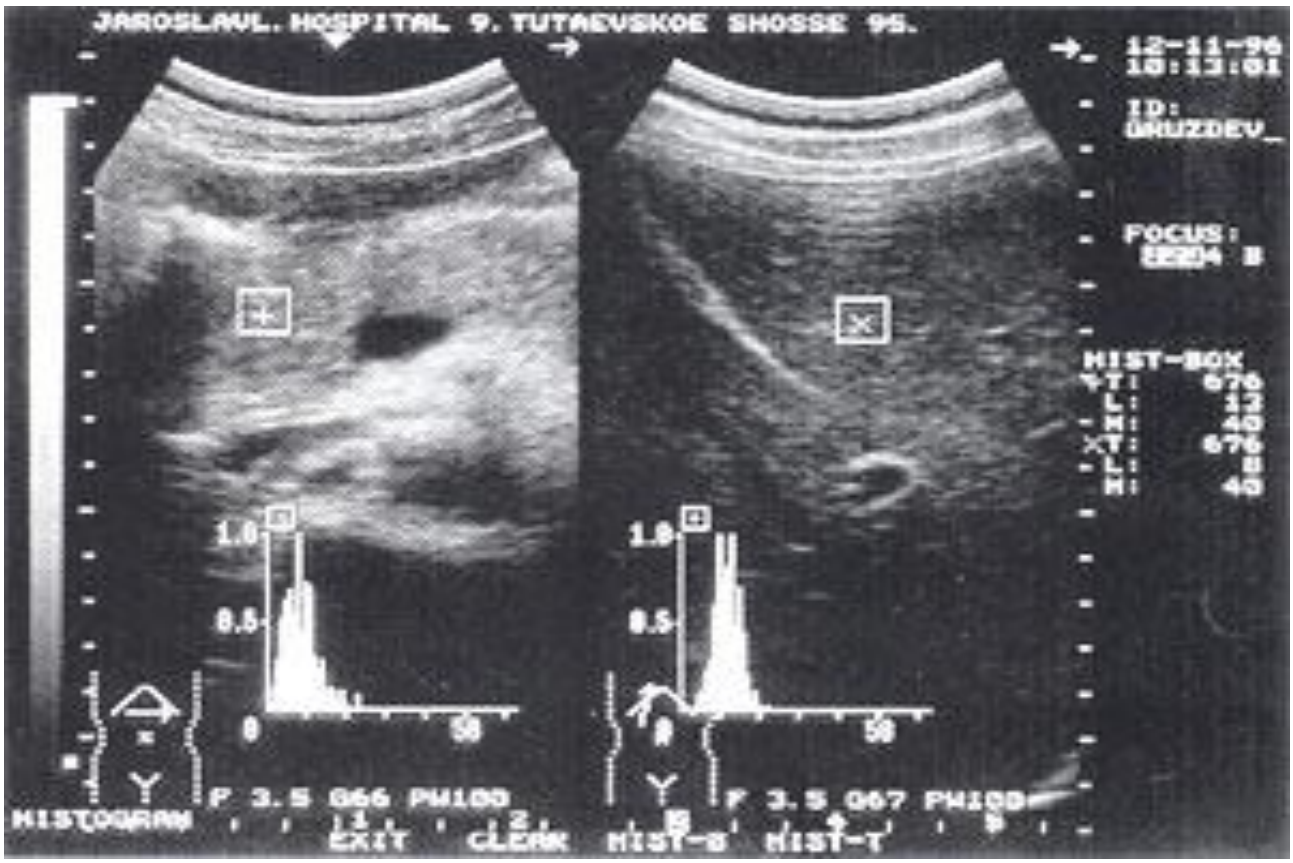


Fig. 6.1.3.1 Ultrasound of acute pancreatitis

13. Percutaneous drainage of the omentum abscess under ultrasound control

In cases that require the removal of areas of necrotic pancreatic tissue and surrounding tissue, this method is ineffective.

3. Laparoscopic drainage and retroperitoneoscopic drainage and necrectomy.

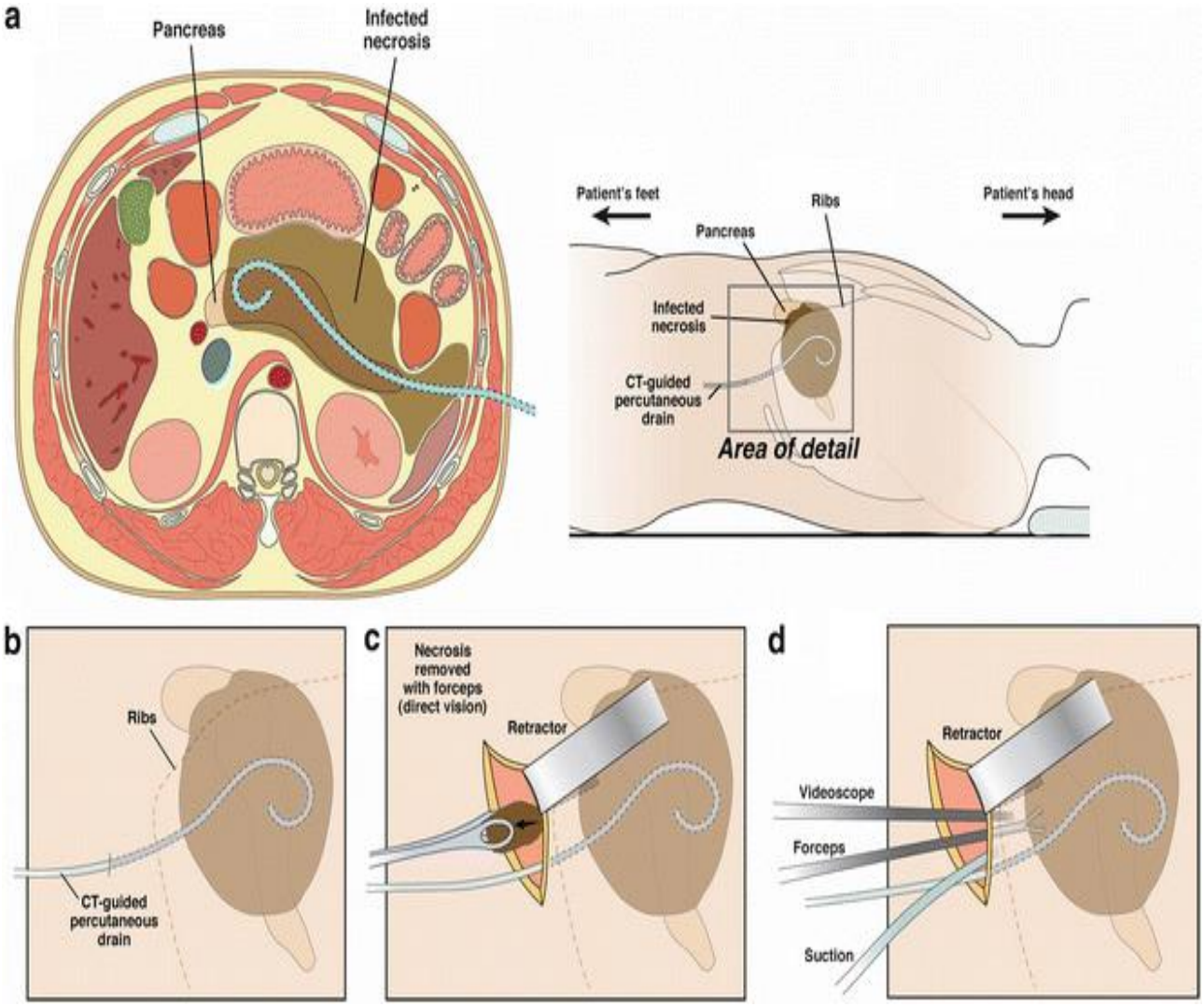


Fig. 6.1.3.2 Laparoscopic drainage and retroperitoneoscopic drainage and necrectomy.

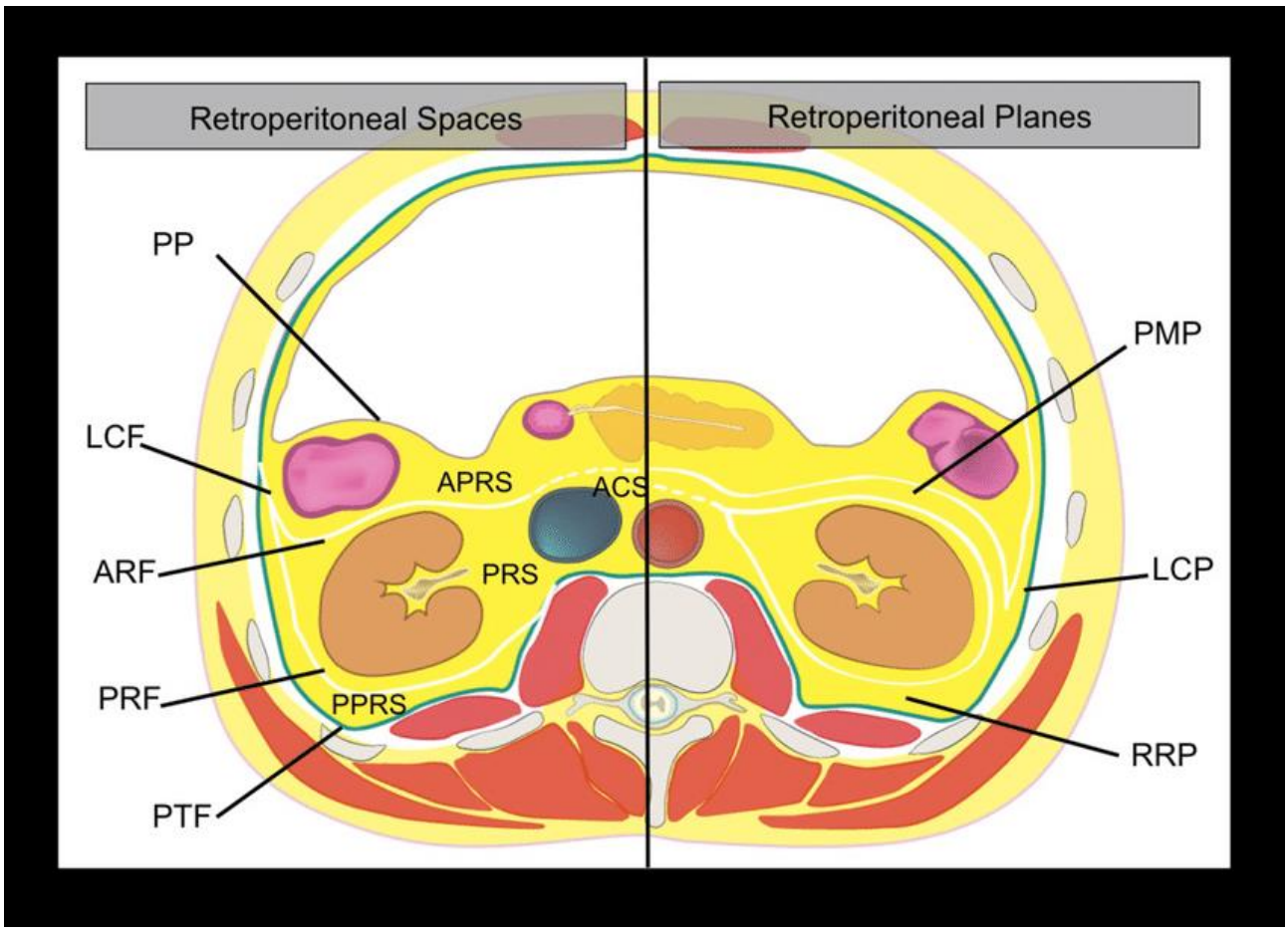


Fig. 6.1.3.3 Retroperitoneal space in surgery

Laparoscopic method in rare cases, it is possible to perform a full-fledged necrectomy and drainage of the omentum bag. Retroperitoneal access involves the installation of a laparoscope and instruments in the retroperitoneal space to perform necrectomy, drainage of the omentum sac and paracolic fiber. However, there is no clear opinion on the indications for the use and effectiveness of such methods in acute pancreatic necrosis, so traditional surgical treatment is the main one.

## 6.2.Surgical interventions in AP.

Given the extreme variety of options for the clinical course and complications of AP, it is necessary to focus on the principal approaches to surgical treatment.

1. The edematous form of AP is not an indication for surgical treatment.

2. Surgical treatment for sterile necrotizing pancreatitis is also not indicated in the first 14 days from the onset of the disease, if there are no complications that require urgent interventions.

3. Indications for early surgical treatment (the first 2 weeks) are a combination of pancreatitis with destructive cholecystitis, perforation of the hollow organ, the presence of infected pancreatogenic peritonitis, massive pancreatogenic bleeding, biliary pancreatitis with the failure of endoscopic papillosphincterotomy, etc.

4. After 2-3 weeks from the onset of the disease, a demarcation line of pancreatic lesion with the formation of sequesters is formed in large-scale pancreatic necrosis. The risk of bleeding during the removal of devitalized pancreatic tissue (pancreatogastrostomy) is much lower. In these terms shows pancreatoduodenectomy and drainage of the packing bags.

5. When infected pancreatic necrosis is typically 2-3 weeks from the onset of the disease develop suppurative complications (abscess packing bags, cellulitis retroperitoneal left / right localization, etc.). Surgical treatment includes necrosectomy, opening, sanitation and drainage of the purulent foci abdominal cavity and retroperitoneal space. The main type of surgical access is a median laparotomy, which, after performing the main surgical procedure, is supplemented by a left - or right-sided lumbotomy.

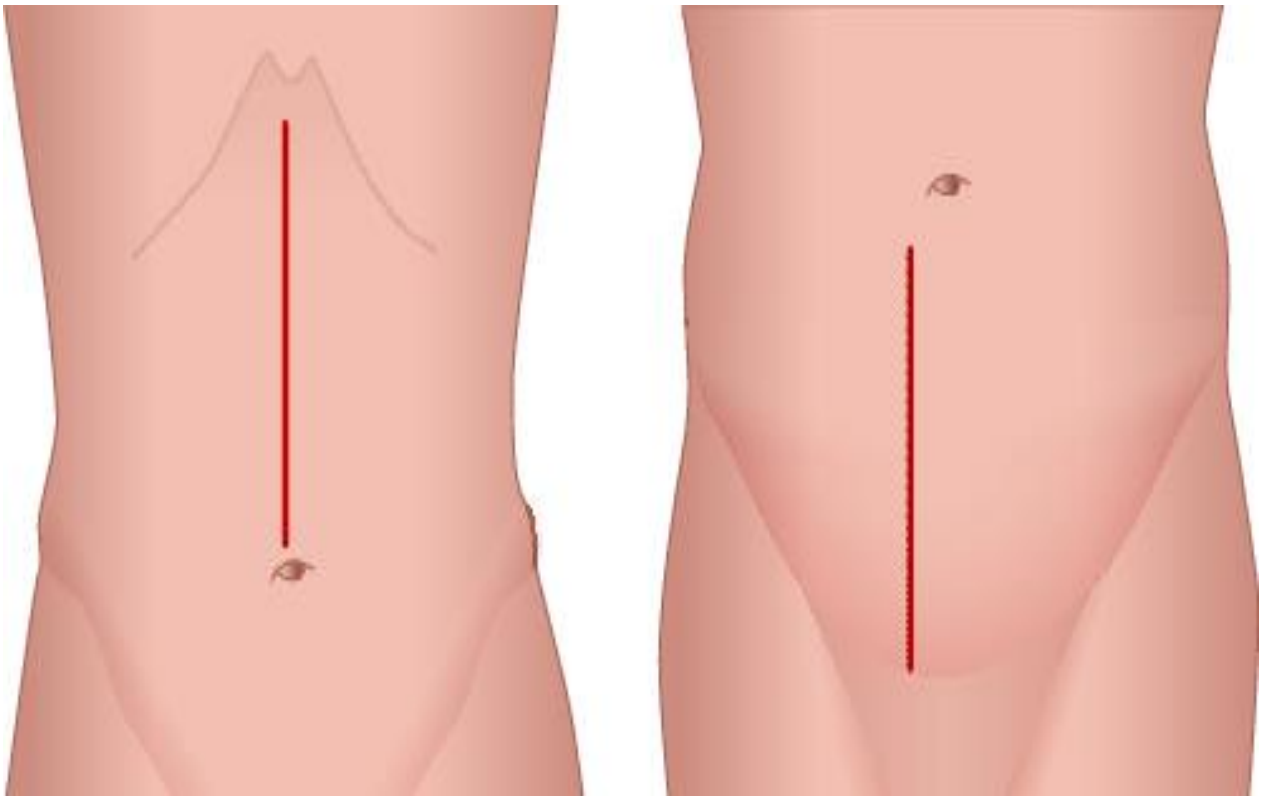

Fig. 6.2.1 Median laparotomy

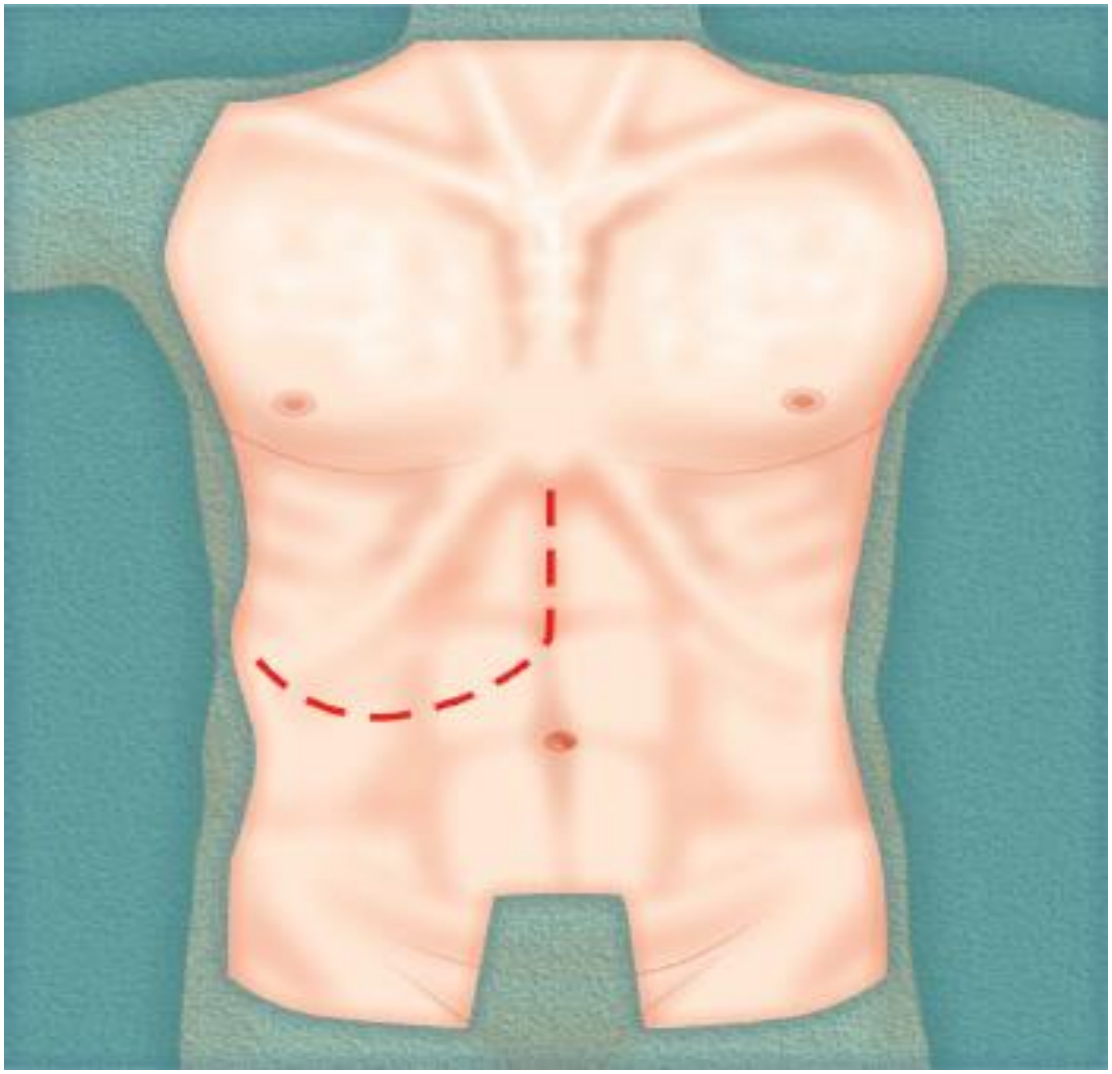

Fig. 6.2.2 Bicostal laparotomy

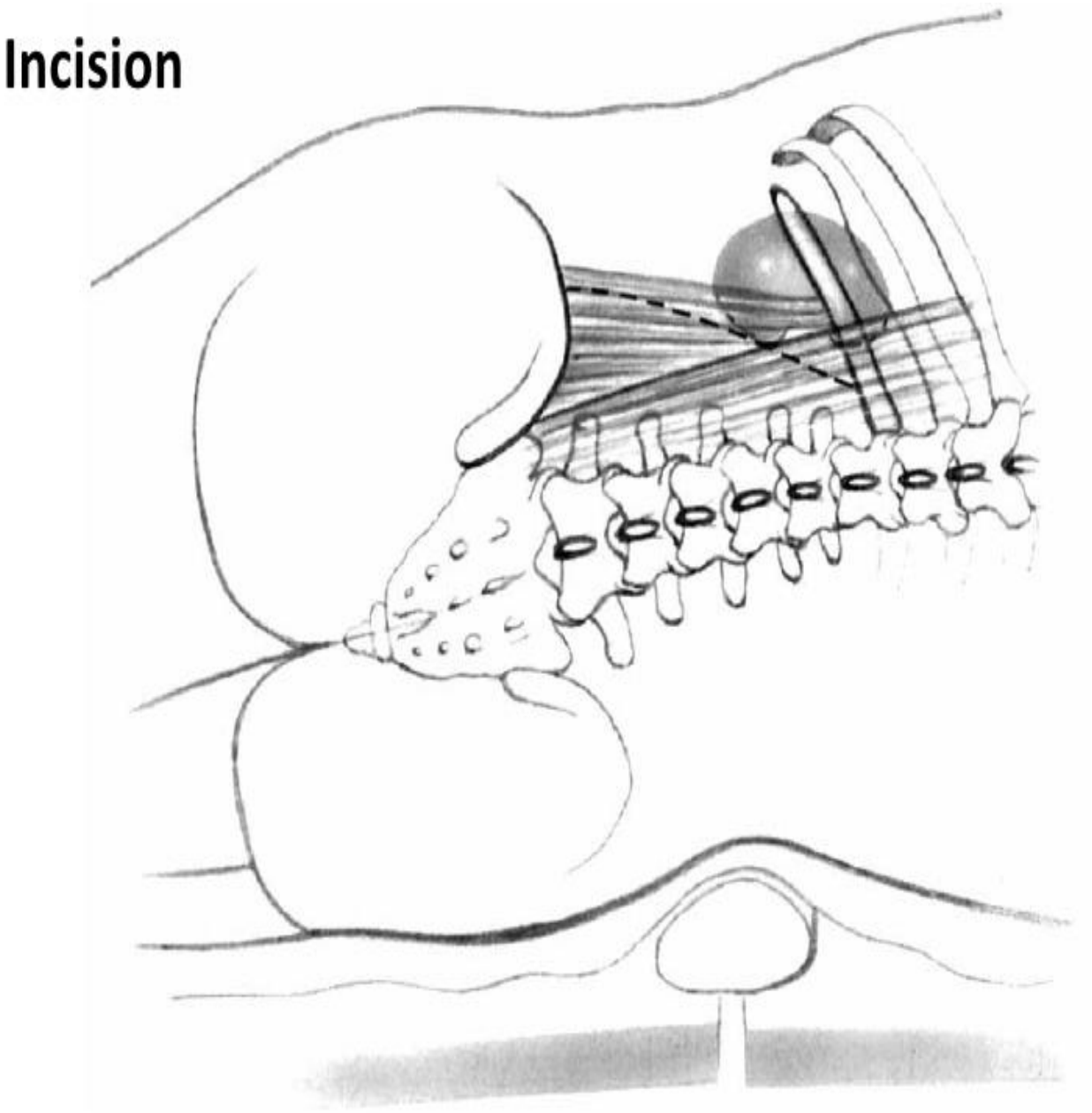


Fig. 6.2.3 The main surgical approaches in the treatment of AP (median laparotomy, bicostal laparotomy, lumbotomy)

The operation usually ends with the drainage of the omentum and retroperitoneal space. The main tasks of surgical intervention are: removal of pancreatogenic and inflammatory exudate from the abdominal cavity, omentum and retroperitoneal space, removal of infected and necrotic pancreatic tissue and pancreatic fiber, preservation of viable pancreatic tissue, postoperative lavage.

In the postoperative period, prolonged irrigation sanitation of purulent cavities is carried out through tubular drainage systems with the use of various solutions of antiseptics in cold form.

In pancreatogenic abscesses of the omentum sac, it is possible to form a pancreatoomentobursostomy by suturing fragments of the gastrointestinal ligament to the parietal peritoneum in the upper third of the laparotomic wound according to the marsupialization type. In large-scale pancreatic necrosis, the processes of sequestration and rejection of large arrays of necrotic tissues, as a rule, are stretched over time (up to 8 weeks), so the program of surgical treatment of such patients provides for repeated necrosequestrectomies. Other complications of severe forms of AP include the formation of intestinal and pancreatic fistulas, cysts, pleurisy, arrosive bleeding, diabetes mellitus, etc.

And now we will give you the data of the clinical protocols.

Early (I) phase. Treatment protocols for acute pancreatitis in phase IA of the disease.

Laparoscopic surgery protocol

Laparoscopy is recommended:

- patients with peritoneal syndrome, including in the presence of ultrasound signs of free fluid in the abdominal cavity.

The level of credibility of the recommendation "C".

- if necessary, differential diagnosis with other diseases of the abdominal cavity.

The level of credibility of the recommendation "C".

Comments: The tasks of laparoscopic surgery can be diagnostic, prognostic and therapeutic.

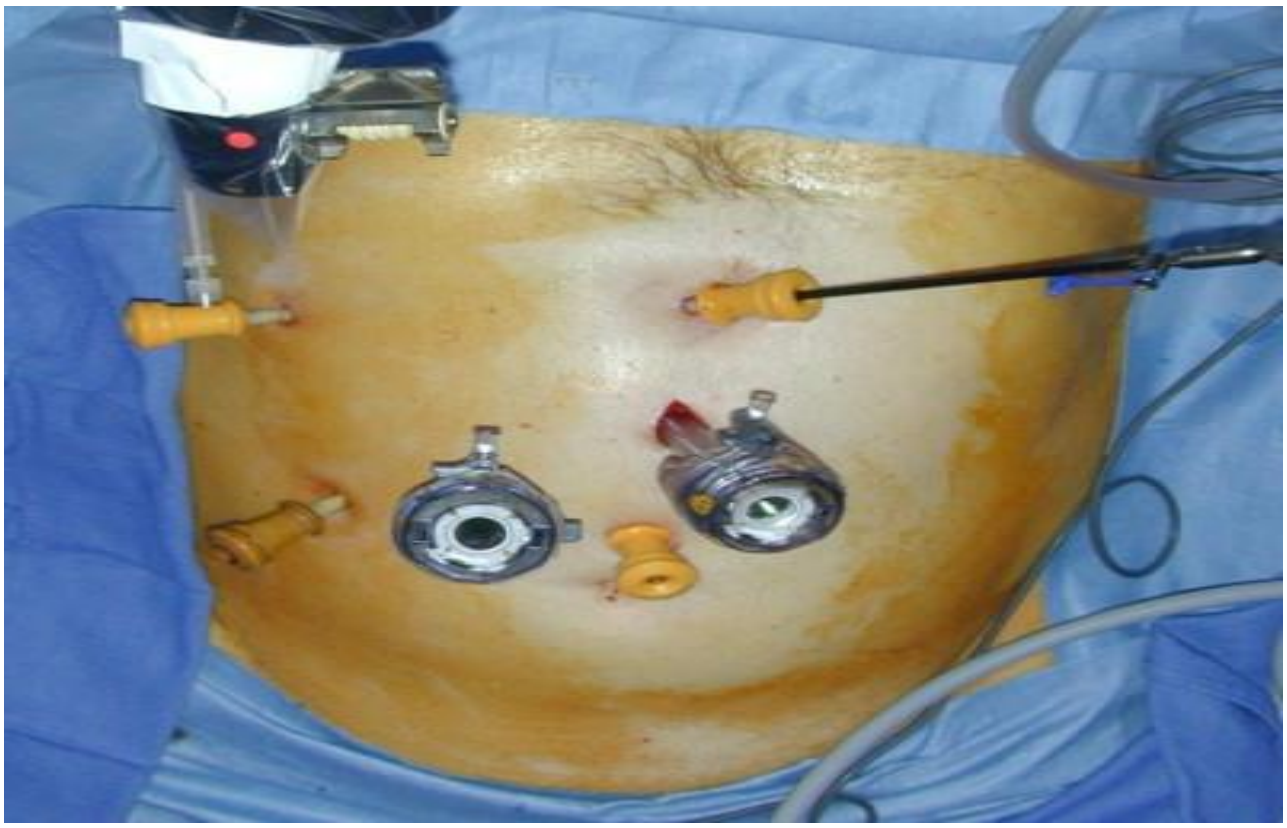

Fig.6.2.4. Laparoscopy

Percutaneous drainage of the abdominal cavity under ultrasound guidance or laparocentesis is recommended.

The level of credibility of the recommendation "C".

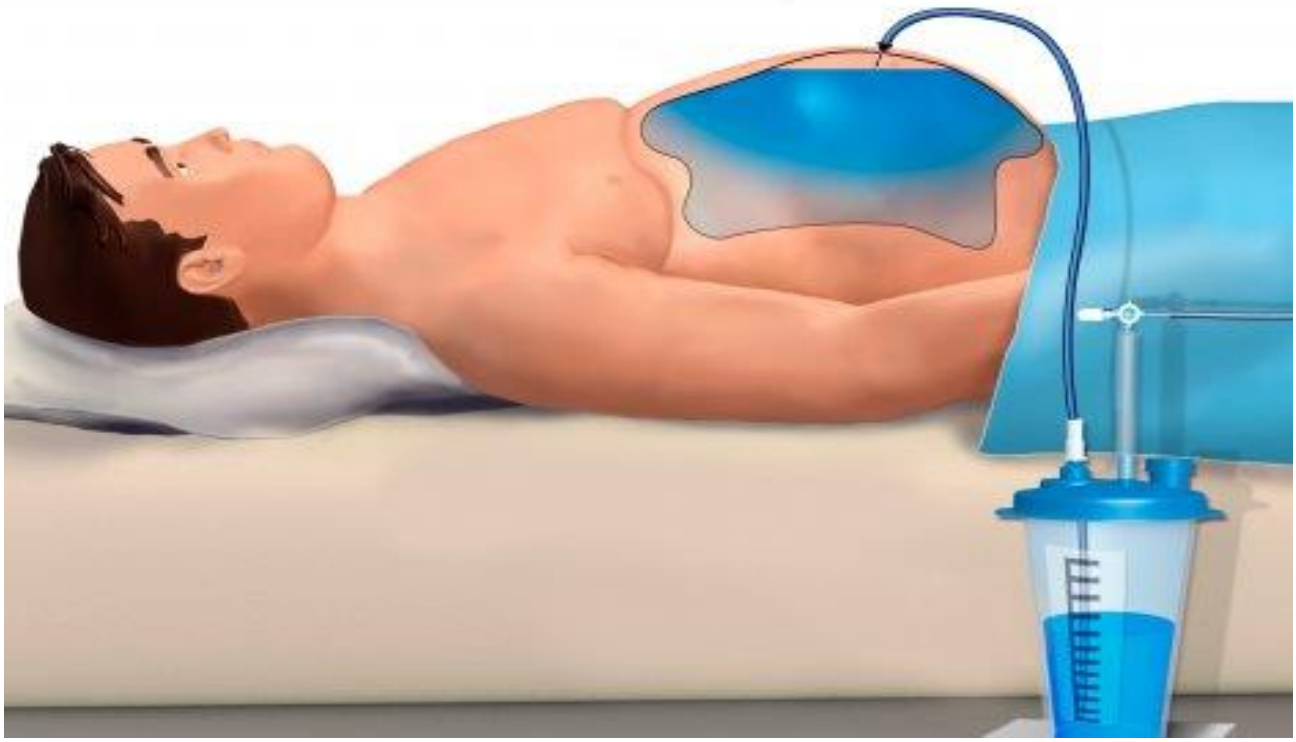

Fig.6.2.5. Laparocentesis

Tasks of laparoscopic surgery:

a) confirmation of the diagnosis of acute pancreatitis (and, accordingly, the exclusion of other diseases of the abdominal cavity, primarily acute surgical pathology-mesenteric thrombosis, etc.); the signs of AP include:

- the presence of an outflow of the root of the mesentery of the transverse colon;
- the presence of an effusion with high amylase activity (2-3 times higher than the activity of blood amylase);
- the presence of steatonecrosis;

b) identification of signs of severe pancreatitis:

- hemorrhagic character of the enzymatic effusion (pink, raspberry, cherry, brown);
- common foci of steatonecrosis;
- extensive hemorrhagic impregnation of the retroperitoneal tissue, extending beyond the zone of the pancreas;

Verification of serous ("vitreous") edema in the first hours of the disease (especially against the background of the patient's severe general condition) does not

exclude the presence of severe pancreatitis, since laparoscopy in the early stages may not reveal signs of severe pancreatitis, i.e. the disease may progress in the future.

c) therapeutic tasks:

removal of peritoneal exudate and drainage of the abdominal cavity

Late (II) phase (sequestration). Protocol for the treatment of acute pancreatitis in the phase of septic sequestration, i.e., the treatment of purulent complications.

In case of purulent complications of AP, surgical intervention is recommended, the purpose of which is to sanitize the affected retroperitoneal tissue.

The level of credibility of recommendation " A "

Comments: The intervention includes the opening, rehabilitation and drainage of the affected retroperitoneal tissue. The main method of rehabilitation of purulent-necrotic foci is necrsequestrectomy, which can be either one-stage or multi-stage, and is achieved by both minimally invasive and traditional methods.

When deciding on the primary drainage of a pancreatic abscess or purulent-necrotic parapancreatitis, it is recommended to give preference to minimally invasive interventions (ultrasound-guided drainage, retroperitoneoscopy, minilaparotomy with a set of "Mini-assistant", etc.).

The level of credibility of the recommendation "B".

Comments: If minimally invasive drainage is ineffective, the operation of choice is a sanation laparotomy with a necrosequestrectomy. Drainage is preferably carried out by extraperitoneal accesses. The optimal timing of the first rehabilitation laparotomy with necrsequestrectomy is 4-5 weeks of the disease. With the development of complications that cannot be stopped with the help of minimally invasive interventions, it is necessary to perform an open operation, including from a mini-access.

After surgery, most patients develop an external pancreatic fistula, which, after the inflammatory process is stopped, is treated conservatively and closes independently in an average of 2-4 months.

In case of persistent pancreatic fistula that does not close for more than 6 months, surgical treatment is recommended as planned.

The level of credibility of the recommendation "C".

Comments: as a rule, the pancreatic fistula in this case is associated with the large ducts of the pancreas.

In the postoperative period, complex therapy is indicated:

Parenteral or enteral nutritional support is recommended (through a probe inserted into the small intestine behind the Treitz ligament) if oral nutrition is not possible.

The level of credibility of the recommendation "B".

Systemic antibiotic therapy is recommended in combination with the prevention of dysbiosis and other complications.

The level of credibility of the recommendation "B".

Comments: The choice of an antibacterial drug depends on the sensitivity of the isolated microorganisms.

Recommended immunocorrection, the options of which are determined individually, depending on the clinical and laboratory parameters.

The level of credibility of the recommendation "D".

## 7. Control questions

1. The concept of AP, definition and frequency of occurrence.

2. What are the most common causes of AP? What etiological factors play an important role in the development of AP (poor nutrition, alcohol abuse, gallstone disease, bile hypertension, congestion in the upper gastrointestinal tract, vascular, toxic and infectious factors, injuries, allergies).

3. What processes underlie the pathogenesis of AP?

4. What pathoanatomical changes can be observed in AP?

5. What is characteristic of the clinical picture of AP? What complaints of patients are characteristic of this acute disease?

6. Describe the nature of the pain syndrome and the features of dyspeptic phenomena in AP. How do peritoneal and inflammatory syndromes manifest in AP?

7. What are the symptoms characteristic of AP? What vascular manifestations (symptoms) are observed in severe forms of AP?

8. What clinical studies should be performed for AP? What changes in the biochemical blood test are characteristic of AP? What laboratory indicator in the analysis of urine should be determined in this disease?

9. With what diseases is it necessary to make a differential diagnosis in AP? What is the basis for the diagnosis of AP?

10. What is related to the complications of AP? Which of the complications of AP are indications for surgery?

11. Signs of functional insufficiency of organs and systems in AP.

12. What are the tactics of managing a patient with AP?

13. What measures are necessary for the conservative treatment of AP? What is the drug therapy of AP?

14. What is characteristic of the clinical picture of pancreatic necrosis?

15. What laparoscopic signs occur in AP?

16. What measures should be taken during the operation for pancreatic necrosis?

## 8. Tests

1. Indications for surgical intervention in destructive pancreatitis:

A) parapancreatic infiltrate;

B) purulent parapancreatitis;

C) swelling of retroperitoneal fat;

D) pancreatogenic peritonitis;

E) severe intoxication.

2. Assessment of the severity of acute pancreatitis on the basis of computed tomography is performed according to the system:

A. Ranson.

B. Savelyev V. S.

C. SOFA.

D. Balhtasar.

E. SAPS.

3. Resurrection symptom of acute pancreatitis is:

A. the Presence of girdling pain in the upper abdomen.

B. Tenderness to palpation in the left costal-vertebralcorner.

C. Pneumatization transverse colon under reviewradiography of the abdominal cavity.

D. the disappearance of pulsation of the abdominal aorta duringpalpation.

E. the presence of effusion in the left pleural cavity,confirm radiographically.

4. What kind of pain is characteristic of acute pancreatitis?

A. Cramping in the upper abdomen.

B. Constant intense painof a shingling nature with radiation in the back.

C. Aching pains in the epigastric region that decreaseafter eating.

D. Pains in the right hypochondrium that increase when the patient moves andchanges the position of the body.

E.Pains in the epigastric region that move to the right

iliac region.

5. To combat paresis of the gastrointestinal tract in acute pancreatitis, do not use:

A. Proserin.

B. Ubretid.

C. Washing the intestines with a cooled salinesolution.

D. Sanation laparoscopy.

E. Cerukal.

6. What is not used to eliminate pain in acute pancreatitis?

A. Non-narcotic analgesics.

B. Narcotic analgesics.

C. Novocaine blockade of the round ligament of the liver.

D. Novocaineparanephral blockade.

E. Peridural anesthesia.

7. Among the signs by which acute pancreatitis is classified, one is indicated incorrectly:

A. Clinical and morphological form.

B. The prevalence of the process.

C. The course of the disease.

D. The presence or absence of intoxication.

E.The stages of the disease.

8. What is primarily due to the severity of the course of acute destructive pancreatitis and the severity of the condition of patients:

A. Purulent intoxication.

B. Bacteremia.

C. Hypovolemia.

D. Hyperglycemia.

E. Enzyme toxemia.

9. What type of surgical intervention is the method of choice for advanced pancreatic necrosis with localization of the process in the body and tail of the pancreas:

A. Drainage of the omentum sac and abdominal cavity.

B. Omentopancreatopexy.

C. Abdominization of the pancreas

D. Corporocaudal resection.

D. Marsupialization of the omentum sac.

10. The most common complications of pancreatic necrosis do not include:

A. Pancreatogenic shock.

B. Aseptic enzymatic peritonitis.

C. Pelvic abscess.

D. Abscess of the omentum sac.

E. Cysts and fistulas of the pancreas.

11. The edematous form of AP is characterized by:

A) a significant increase in body temperature;

B) severe abdominal pain;

C) frequent loose stools;

D) yellowing of the skin and sclera;

E) urinary incontinence.

12. Fatty pancreatic necrosis is formed as a result of:

A) the addition of infection against the background of edematous pancreatitis;

B) activation and damaging effect of lipolytic enzymes;

C) activation and damaging effect of proteolytic enzymes;

D) activation and damaging effect of glycolytic enzymes;

E) involution of small-focal pancreatic necrosis.

13. Hemorrhagic pancreatic necrosis is formed as a result of:

A) the addition of infection on the background of edematous pancreatitis;

B) activation and damaging effect of lipolytic enzymes;

C) activation and damaging effect of proteolytic enzymes;

D) activation and damaging effect of glycolytic enzymes;

E) involution of small-focal pancreatic necrosis.

14. The phenomenon of the absence of pulsation of the abdominal aorta in OPis called the symptom:

A) Mayo-Robson;

B) Kerte;

C) Gray Turner;

D) Voskresensky;

E) Mondor.

15. Laparoscopy in AP allows:

A) diagnose the lesion of the pancreas;

B) specify the form of the disease;

C) identify concomitant urgent diseases;

D) diagnose pancreatogenic peritonitis;

E) all of the above is true.

16. Urgent surgical treatment for AP is indicated:

A) for edematous pancreatitis;

B) fatty pancreatic necrosis;

C) hemorrhagic pancreatic necrosis;

D) the development of purulent peritonitis;

E) concomitant acute destructive cholecystitis.

17. The development of retroperitoneal phlegmon in AP is accompanied by:

A) the appearance of vomiting;

B) increased blood and urine amylase levels;

C) swelling of the subcutaneous tissue in the lumbar region;

D) normalization of the temperature reaction;

E) all of the above is true.

18. A 30-year-old patient was admitted to the clinic on the 2nd day of the diseasewith a diagnosis of AP, severe enzymatic intoxication and pancreatogenic peritonitis. Specify the method of removing pancreatic enzymes from the body:

A) local intragastric hypothermia;

B) peritoneal dialysis;

C) peridural anesthesia;

D) catheterization of the umbilical vein;

E) catheterization of the aorta.

19. The patient, 20 years old, has a clinical picture of AP, but can not be excludeda perforated stomach ulcer. It was decided to perform a diagnostic laparoscopy. Name a reliable laparoscopic sign of destructive pancreatitis:

A) pneumatization of the intestine;

B) hyperemia of the peritoneum;

C) edema of the large omentum;

D) the presence of gastric contents in the abdominal cavity;

E) plaques of steatonecrosis on the peritoneum.

20. On the 15th day, a patient with destructive pancreatitis retains pronounced intoxication phenomena, body temperature is 39 °C,chills, sweating, leukocytosis, hyperemia of the skin in the lumbar region. Specify the correct diagnosis:

A) edematous pancreatitis;

B) the abscess of the pancreas;

C) abscess of the omentum bag;

D) purulent peritonitis;

E) retroperitoneal phlegmon.

21. Indications for surgical intervention in destructivepancreatitis:

A) parapancreatic infiltrate;

B) purulent parapancreatitis;

C) swelling of retroperitoneal fat;

D) pancreatogenic peritonitis;

E) severe intoxication.

**Answers**: 1-B, 2-D, 3-A, 4-A, 5-B, 6-E, 7-E, 8-D, 9-D. 11 — B; 12-B; 13-C; 14-D; 15-E; 16-D; 17-C; 18-B; 19-E; 20-E;

## 9. Situational tasks

1.A patient, 34 years old, who had been abusing alcohol for a week, was admitted to the emergency room with complaints of shingling pains in the upper half of the abdomen, repeated vomiting of bile that did not bring relief, an increase in body temperature to 37.5 C, sharp weakness, malaise. Palpation in the epigastrium is determined by a painful infiltrate of 10 x 10 cm. What tests should be prescribed? A presumptive diagnosis?

2. The patient, 42 years old, has a history of gallstone disease, with a clinic of acute pancreatitis, according to the EGDS, there is a "hammered" stone of the large duodenal papilla. The presumed diagnosis and management tactics of the patient?

3. In a patient with pancreatic necrosis, the intraoperative picture is as follows: the pancreas is enlarged, lumpy, heterogeneous, with black necrosis areas all over the surface with purulent discharge, and there is also a necrotic phlegmon of the retroperitoneal fiber of the splenic angle. The presumed diagnosis and what should be the surgeon's tactics?

4. A patient, 49 years old, after taking a plentiful meat, fatty food, suddenly developed an attack of severe shingling pain in the epigastric region, repeated vomiting. The temperature remained normal. Palpation of the epigastric region causes moderate pain. Diastasis of urine 512 units. The conservative treatment carried out during the day led to an improvement in the condition; the intensity of pain significantly decreased, vomiting stopped. The abdomen became soft, and slight soreness in the epigastric region persisted. Symptoms of irritation of the peritoneum 60 were not detected. Urine diastasis decreased to 128 units. Make a diagnosis. What should be the treatment strategy?

5. The patient, 30 years old, was admitted to the surgical department as an ambulance with sharp shingles in the abdomen, accompanied by indomitable vomiting and fever. From the anamnesis, it is known that the patient is a chronic alcoholic. The day before, after taking spicy food and alcohol, there were severe abdominal pains. For the first time, such pains were observed 3 years ago. Conservative therapy resulted in temporary improvement. Upon admission, the body temperature is 38.4°C, the condition is severe, shortness of breath, cyanosis of the skin, jaundice sclera. The abdomen is swollen, with palpation there is a sharp soreness without distinct symptoms of irritation of the peritoneum. Blood pressure is 90/60 mm Hg. On the overview radiograph of the chest, there is an effusion in the left pleural cavity. Urine amylase 2048 units. Make a diagnosis. What should be the treatment strategy?

6. A 41-year-old patient who had been suffering from chronic cholecystitis for many years suddenly developed an attack of sharp pain in the upper abdomen. The pain was shingling in nature, accompanied by repeated vomiting, which did not bring relief. The condition is of moderate severity, pulse 90 per minute, blood pressure 120/80 mm Hg. In the epigastric region, moderate bloating and soreness are determined. There are no symptoms of peritoneal irritation. Diastasis in the urine increased to 1024 units. Conservative treatment, which was carried out for 2 days, did not give any effect. The patient's condition worsened, the body temperature increased to 38.5 °C, the pulse increased to 120 per minute. The abdomen became swollen, increased pain when palpating the lateral parts of the abdomen, there were symptoms of

irritation of the peritoneum. Urine diastasis decreased to 32 units. Make a diagnosis. What should be the treatment strategy?

7. The patient, 27 years old, high nutrition, cook by profession, was taken to the emergency clinic. After an error in nutrition 12 hours ago, there were severe pains in the right hypochondrium, there were several times vomiting. After taking antispasmodics, the pain did not subside. For 4 years, the patient notes paroxysmal pain in the right hypochondrium. On admission, the condition is of moderate severity. There is a slight jaundice of the skin and sclera (blood bilirubin 56 mmol/l). The tongue is dry, overlaid with a white coating. The abdomen increased in volume at the expense of adipose tissue, painful and stressful in the epigastrium and right hypochondrium. In the same place, a sharply painful rounded formation is indistinctly palpated along the mid-clavicular line. Symptoms of peritoneal irritation are indistinct. The body temperature of 38.5 °C. White blood cells in the blood of 13.8 • 10 to the degree of 9 /L. Urine amylase 1024 units. Make a diagnosis. What diagnostic methods can be used? What should be the treatment strategy?

8. A patient, 51 years old, who was abusing alcohol, suddenly had sharp pains in the epigastrium, which radiated into the back. The temperature remained normal. There was repeated vomiting, which did not bring relief. During the inspection it was determined a moderate pain in the epigastric region. The abdomen is soft, there are no symptoms of irritation of the peritoneum. Urine diastasis 1024 units. Conservative measures did not improve. On the next day, the patient's condition worsened: the pulse rate increased to 120 per minute, blood pressure decreased to 70/40 mm Hg. Symptoms of peritoneal irritation appeared. Diastasis in the urine became 64 units. How should the course of the disease be assessed? What should be the treatment strategy?

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