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Pathogenesis, Clinical, Diagnosis, Treatment, and Complications are Acute Pancreatitis

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Annotation: Lecture on the current state of the problem of acute pancreatitis. The pathogenesis, clinical picture, diagnosis and principles of treatment of the disease are considered. Particular attention is paid to indications and contraindications for conservative and surgical treatment of pancreatic necrosis, depending on the stage and severity of the disease. It is emphasized that stopping the acute inflammatory process in the gland does not mean complete recovery and morphological restoration of the pancreas. As a rule, pancreatic necrosis leads to the development of chronic pancreatitis, therefore, after discharge from the hospital, patients are subject to long-term restorative treatment aimed at restoring tissue trophism and gland function.

Keywords: Pancreatic necrosis, clinic, diagnosis, treatment.

In recent decades, acute pancreatitis has begun to occupy a leading position in the practice of emergency abdominal surgery, second in frequency only to acute appendicitis [8, 10, 14]. However, we have not yet been able to significantly improve the results of his treatment. We can list the main and most typical reasons that cause high mortality, the frequency of complications and disability of patients, which impede tangible progress in the treatment of this disease:

- ✓ untimely detection of necrotizing pancreatitis;
- ✓ untimely assessment of the severity of the disease, its prognosis and untimely initiation of adequate intensive care;
- ✓ a high frequency of early surgical interventions due to diagnostic errors or progressive deterioration of the patient's condition;

- ✓ use of inadequate methods of detoxification and antibacterial therapy;
- ✓ difficulty in distinguishing between a systemic response to inflammation and a purulent process;
- ✓ late detection of purulent complications;
- ✓ limited use of minimally invasive drainage methods;
- ✓ inadequate frequency of planned sanitation relaparotomies;
- ✓ lack of a rehabilitation system for patients who have suffered pancreatic necrosis.

Pathogenesis and clinic

In 1992, a group of experts on the problem of acute pancreatitis adopted in Atlanta (USA) a classification of acute pancreatitis, which is now recognized in our country. It includes edematous and necrotic pancreatitis [8, 23]. Necrotizing pancreatitis can be aseptic and infected. Infected pancreatic necrosis can be presented in the form of purulent pancreatitis, retroperitoneal phlegmon. As a result of an acute disease, a pseudocyst or abscess may form. In the most common and mild form – edematous (interstitial) acute pancreatitis – the degree of enzyme activation and their systemic effect are low [18, 23]. Macroscopically, one can note swelling of the parenchyma of the gland, the disappearance of lobulation and clear boundaries, the appearance of isolated fat necrosis and minor hemorrhages, and in the omental bursa - a small amount of serous-hemorrhagic effusion. Such changes are often mistakenly regarded as focal pancreatic necrosis.

With pancreatic necrosis, the macroscopic picture is brighter. The pancreas is usually sharply enlarged in size and distinctly hardened due to inflammatory edema, with multiple and confluent spots of fat necrosis. The surface of the gland and even the surrounding tissue, root the mesenteries of the small and large intestine may have hemorrhagic imbibition without distinct boundaries, often black in color (the effect of proteolytic enzymes on blood components) [24]. It is important to emphasize that in the presence of retroperitoneal hemorrhagic imbibition, it is often almost impossible to determine the localization and distribution of necrosis, and under the altered peritoneum and surrounding cells the less altered pancreas may be located at the tip. That is why a conclusion about the extent of gland damage based only on a visual assessment often does not correspond to the truth. In almost all cases, pancreatic necrosis is accompanied by pronounced changes in the abdominal cavity and its organs [18]. In the abdominal cavity there can be from 500 to 3000 ml of serous, serous-hemorrhagic or intensely hemorrhagic effusion containing high levels of pancreatic enzymes. The parietal and visceral peritoneum is dull, brightly hyperemic, multiple and confluent foci of fat necrosis and hemorrhage appear in the mesentery and omentum [24]. As a rule, there is extensive serous or hemorrhagic imbibition of the retroperitoneal tissue. The loops of the small and large intestines are dilated and peristalt sluggishly. The gallbladder is usually distended and difficult to empty. With pancreatic necrosis, massive activation of pancreatic enzymes (trypsin, elastase, lipase, phospholipase A) occurs, which leads to the appearance of a cascade of interconnected local and general enzymatic reactions. Thus, the pancreas is one of the powerful sources of kallikrein. Activation of the kallikrein-kinin enzyme system with powerful vasoactive properties leads to profound disorders of microcirculation and blood rheology, as well as systemic hemodynamics [18]. Microcirculation disorders in the celiac trunk and portal vein cause further progression of the autolytic and ultimately necrotic process in the pancreas and surrounding tissues. In parallel, systemic exposure to vasoactive and proteolytic enzymes can lead to the development of foci of necrosis in almost all organs [24].

The systemic toxic and damaging effects of activated pancreatic enzymes and products of its autolysis manifest themselves primarily in increasing dysfunction of vital organs and systems. From the central nervous system, phenomena of toxic encephalopathy are observed, up to the

development of pancreatogenic delirium. There are signs of respiratory failure associated with interstitial pulmonary edema, the development of pulmonary distress syndrome and effusion pleurisy [3, 5, 7].

Disorders of hemodynamics and function of the cardiovascular system manifest themselves most early and clearly. Against the background of a decrease in circulating blood volume due to impaired vascular permeability and sequestration of the plasma part of the blood in the intercellular space, free abdominal cavity, and fluid in the intestinal lumen, hypovolemia increases, cardiac output decreases, tachycardia and clear signs of disorders occur. peripheral circulation – cyanosis and hypothermia of the skin of the extremities. Against the background of hypovolemia, disorders of systemic and regional hemodynamics, clinical and biochemical signs of cardiovascular, respiratory and hepatic-renal failure develop and rapidly progress. Symptoms of dynamic intestinal obstruction increase. Dysfunction of the gastrointestinal tract is one of the causes of endogenous infection of foci of necrosis [10, 14]. Studies show that with pancreatic necrosis, by the end of the first week of the disease, up to 40% of patients have infected necrosis.

By the end of the second week of the disease, the frequency of infection of necrosis increases to 80% [21, 22, 24].

This is the most important factor in the pathogenesis of pancreatic necrosis. If initially the foci of autolysis are sterile, then in the process of progression they are almost inevitable become infected and become a source of local purulent-inflammatory complications [21, 24]. Due with this, during pancreatic necrosis it is possible to distinguish trace two successive phases: phase 1 – enzymatic toxemia with hemodynamic and multiorgan disorders (5–9 days) and phase 2 – postnecrotic and purulent complications (after 7–12 days day). In clinical manifestations and timing of these phases have no clear boundaries and complement each other.

Diagnostics

It is important to emphasize that, in addition to abdominal examination data and even instrumental data, the slightest signs of intoxication and organ dysfunction are of utmost importance in the early diagnosis of necrotizing pancreatitis.

Priority in the instrumental diagnosis of pancreatic necrosis and assessment of the dynamics of the process (Balthazar system) belongs to computed tomography with intravenous bolus enhancement, in which it is possible to assess areas of ischemia (necrosis). Ultrasound examination has predominantly screening or differential diagnostic value [20].

Based on the main macroscopic characteristics of various forms of acute pancreatitis, it should be noted that their differences are not so much qualitative as quantitative. Therefore, already at the early stages of diagnosis, patients with acute pancreatitis should be separated according to some assessment system (Ranson, APACHE, SAPS, ASA, etc.). into two flow groups – light and heavy. Mild, as a rule, corresponds only to edematous pancreatitis, and severe - only pancreaonosis [8].

The dynamics of symptoms are also fundamentally different for certain forms of acute pancreatitis. During conservative treatment only for edematous form, already in the first day there is a decrease in pain syndrome, tachycardia decreases, there are no symptoms of peritoneal irritation and gastrointestinal disorders. Within 7–14 days, almost complete normalization of well-being and basic laboratory parameters occurs [9, 16]. Edematous pancreatitis is practically not accompanied by the development of local or systemic complications. The course of pancreatic necrosis at the first stage of the disease (6–9 days) is accompanied by pronounced and progressive symptoms of enzyme toxemia, which are difficult to relieve even with multicomponent intensive care [3]. They manifested by profound hemodynamic disorders, symptoms of multiple organ failure, increasing signs of peritonitis. Any of the listed complications may cause deaths during these periods of the disease. It is the ineffectiveness of intensive care that often serves as one of the indications for emergency surgery. If the patient's

condition can be stabilized pancreatic necrosis, then almost 80% of them on average by the end of the second week of the disease already occurs endogenous infection of foci of necrosis in the gland and surrounding tissues. The purulent-inflammatory process progresses, and the symptoms of intoxication [10, 21]. It shows up increased pain, increased tachycardia, the appearance of hyperthermia, often disorder evacuation from the stomach and the formation of an inflammatory infiltrate in the epigastric region. In the diagnosis of forms of acute pancreatitis, except computed tomography and ultrasound often require laparoscopy, in which it is also possible to perform abdominal drainage, biliary decompression. Plays an important role in assessing the dynamics of pancreatic necrosis and early detection of infection. Determination of C-peptide and fine-needle biopsy of the gland. Patients with pancreatic necrosis should initially receive treatment and monitoring of vital functions in the intensive care unit.

Treatment

Treatment of acute pancreatitis should be carried out only in a hospital. The scope of therapy and the agents used vary in different forms of the disease. The general principle is maximum abstinence from surgical interventions on the pancreas, complete pain relief, creation functional rest of the organ, suppression of secretion, reduction of intraductal hypertension, compensation of fluid and electrolyte losses [28]. It is necessary to neutralize activated pancreatic enzymes and remove toxic products, maintaining the function of vital organs and systems, as well as the prevention of purulent-inflammatory complications [1, 2]. The most important condition the effectiveness of conservative therapy is its anticipatory nature, prevention of major systemic and local complications. Each of these problems can have several solutions:

1. Pain relief. Novocaine blockade of paranephric or round ligament of the liver, intramuscular or intravenous administration of a 2% solution of promedol, epidural blockade, intravenous infusion of a glucose-novocaine mixture [2, 4];
2. Functional rest of the pancreas. Hunger, continuous nasogastric aspiration, parenteral administration of H2 receptor blockers histamine [7, 12, 13];
3. Suppression of external secretion of the organ. Most currently an effective remedy Octreatide (Sandostatin), which is given subcutaneously every 6 hours at a dose of 50–100 mg. Decrease enzyme synthesis is promoted by 5-fluorouracil (ftorafur) at a dose of 5 ml/kg for 5–7 days [10, 11].
4. Reduction of intraductal hypertension. Antispasmodics are prescribed and, if technically possible, cannulation of the Wirsung duct is performed to aspirate gland secretions [17].
5. Compensation for losses and provision of natural fluid and electrolyte needs. A massive infusion of isotonic solutions is carried out. It is very important to regulate the volume of infusion based on the central venous pressure, cardiac output, hematocrit, diuresis [19]. At pancreatic necrosis, the daily volume of infusion should be five or more liters. To maintain volemic and hemodynamic parameters, isotonic solutions are combined with protein solutions, drugs with high oncotic properties, and rheologically active drugs [9].
6. Combating systemic fermentemia. Achieved forced diuresis, infusion of inhibitors proteases (contrical, gordox). It should be noted that the prescription of these drugs is justified only for pancreatic necrosis, severe hemodynamic, hemorheological and coagulation disorders [4]. During the process in the pancreas, these the drugs have no effect.
7. Prevention of purulent complications. Broad-spectrum antibiotics are administered parenterally, affecting coccal, non-clostridial and anaerobic flora [21]. Taking into account the critical role of enterogenous bacterial contamination in the development of purulent complications foci of necrosis, one should strive for early restoration of the functions of the gastrointestinal tract [15]. Therefore, in addition to drug stimulation intestinal motility, from the 4th–5th day it is advisable start nasoenteral tube feeding.

The volume of therapy varies significantly for edematous pancreatitis and pancreatic necrosis.

For edematous pancreatitis, which is not characterized by systemic disorders, the main components of conservative therapy are pain relief and creation of functional rest of the pancreas gland [5]. The volume and structure of intravenous infusion therapy is determined based on considerations prevention of hypovolemia, hemodynamic disorders and compensation of the body's natural needs for fluid and electrolytes. Usually 1.5–2 liters of isotonic solutions are required. These manipulations somewhat reduce the interstitial compression of the pancreatic parenchyma, as well as the possibility of the spread of enzymatic effusion through the retroperitoneal tissues. spaces. However, most often in the early stages from the onset of the disease, such interventions have little effect effective and aggravate the severity of the patient's condition. Surgical treatment is fully justified if there are clinical signs of purulent inflammation, laboratory and instrumental indications of a purulent focus [22, 24]. An important rule for surgical interventions in the early stages of the disease there should be abstinence from using tampons, which contribute to infection of the lesions necrosis and progression of the purulent process.

The main purpose of the operation in the second phase of the disease is to remove foci of necrosis or loose tissue sequestration. During these interventions, a thorough inspection of all possible areas of tissue necrosis development is necessary [6, 15]. This is not only the pancreas and the surrounding tissue, but also the retroperitoneal tissue in the area of the mesenteric root, the tissue spaces along ascending and descending colon. This opportunity is provided only by a wide laparotomy (transverse or median).

Surgical interventions for sequestration gland tissue and surrounding tissue are rarely given full effect with a single execution, so removal of necrotic tissue is often must be carried out at least 4–5 or more times with an interval of 3–4 days. If the process is localized only in the area of the omental bursa, then the stage necrosisequestrectomy can be performed through formed omentobursostomy. An operation of this kind is completed with external drainage in the area necrosis with silicone drainages, and extremely rarely Gauze swabs can be used. If there is an omentobursostomy for subsequent revisions cavities of the lesser omentum and pancreas Laparoscopic techniques are also widely used. In recent years, the method of percutaneous puncture to remove fluid (with its examination for microflora) or to conduct drainage during a purulent process under ultrasound supervision has become widespread, and due to its low invasiveness, has a great therapeutic effect. Often this method allows you to avoid surgical intervention. However, the presence of multiple sequestra in the lesion accumulation of aseptic or purulent fluid most often requires traditional surgery.

Complications

Acute pancreatitis at all stages of its course can be accompanied by general and local complications. Typical general complications include toxic encephalopathy, effusion pleurisy and pulmonary distress syndrome, acute cardiovascular failure, hepatic-renal failure. To a greater or lesser extent in acute . In pancreatitis, dysfunction of all these organs is observed, but only in severe forms can we talk about multiple organ failure. Local complications of acute, mainly necrotizing pancreatitis include enzymatic, and subsequently purulent peritonitis, pseudocyst or abscess of the omental bursa, obstructive jaundice, arrosive bleeding, as well as external or internal pancreatic fistulas of the pancreas. The course of pancreatic necrosis usually goes away

stage of infiltrate formation in the epigastric area, which is associated with swelling of the ligamentous-cellular formations in the omental bursa in the upper floor of the abdominal cavity. If infection does not occur or it can be prevented with conservative therapy, the infiltration may resolve without a trace. When infected, purulent omentobursitis develops and most often an abscess of the omental bursa is formed.

The most typical manifestation of infected pancreatic necrosis is retroperitoneal phlegmon,

which can spread through the cellular spaces up to the pelvis. Purulent melting of fiber occurs in the root area mesentery and both paracolic spaces. This a severe complication is accompanied by symptoms of rapidly progressing intoxication, worsening organ dysfunction and is usually one of the leading causes of death in the late stages of the disease. Therefore, symptoms of recurrence deterioration of the condition after its temporary stabilization in the phase of enzymatic toxemia should always suggest the development of purulent complications. The appearance of clinical signs of purulent intoxication and the persistence of the serious condition of patients against the background of intensive therapy should be considered as an absolute indication for surgical intervention. Its essence lies in the obligatory revision of the gland and all retroperitoneal tissues spaces, maximum removal of necrotic and sequestering tissues and drainage zones of their location through separate incisions of the anterior and lateral abdominal wall. But this procedure in itself is not capable of stopping a sluggish purulent-inflammatory process. Therefore, it is necessary to especially emphasize that in treatment of infected pancreatic necrosis with extensive necrosis of retroperitoneal tissue is practically It is never possible to achieve its cure through one or two sanitation laparotomies. One of the severe complications of pancreatic necrosis may be arrosive bleeding from blood vessels, located near the pancreas, splenic, mesenteric, and gastrocolic ligament vessels. Most often, this complication occurs after surgical interventions, necrosis-vestrectomy. In the treatment of arrosive bleeding, tight tamponade of the area is rarely effective source of bleeding. It is necessary to strive for search and ligation of the damaged vessel. Arrosive bleeding can be successfully eliminated by endovascular methods of vascular occlusion. Necrosis of pancreatic tissue and independent or surgical rejection of these areas

in most cases inevitably leads to the formation of external pancreatic fistulas the course of drainage channels. In case of delayed surgical interventions or inadequate drainage zones of necrosisequestrectomy can be formed and internal fistulas with the gastrointestinal tract, free abdominal or pleural cavity and even with bronchi [15].

Relief of acute inflammatory process in the pancreas according to clinical laboratory tests signs after conservative and surgical treatment does not mean complete recovery and morphological restoration of the pancreas. Within several months after pancreatic necrosis, pseudocysts and even abscesses may develop near the pancreas or in its parenchyma itself [25, 26]. Their treatment in most cases is carried out with conservative measures (antibacterial, nonspecific anti-inflammatory therapy, physiotherapeutic treatment). For cysts larger than 5 cm, more often all you have to do is use the percutaneous method their puncture, and with densely formed walls, more than 3–4 months after pancreatic necrosis .Usually, an operation is performed to internally drain the cyst by cystoenterostomy [27]. As a rule, previous pancreatic necrosis leads to the development of chronic pancreatitis due to microcirculation disorders, fibrous replacement of necrosis foci [13, 15, 25]. Therefore, after discharge from the hospital, patients are subject to long-term rehabilitation treatment, aimed at normalizing tissue trophism and gland functions.

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