Acute pancreatic necrosis

Acute pancreatitis is an inflammatory pancreatic disease of a wide range of severity – from mild to severe with signs of multiorgan failure or severe local complications such as abscessesy, pseudocysts and pancreatic necrosis and adjacent tissues. It is undoubtedly one of the most serious and prognostically worst**sudden abdominal events**.

Classification of acute inflammation

Form interstitial edematous (70-80%)

It characterizes her edema interstitia, inflammation parenchyma, increase in concentration pancreatic enzymesin the pancreas and systemically. It is usually associated with pathologybilary systems.The course is light or moderate.

Form necrosis – sterile necrosis, infected necrosis

Within a few days, pancreatic cells will become massive and intra- and extrapancreatic adipose tissue necrosis will occur. Calcified fat forms Necrosis **(balzer necrosis)**. The course determines the extent of necrosis, retroperitoneal involvement and bacterial contamination.

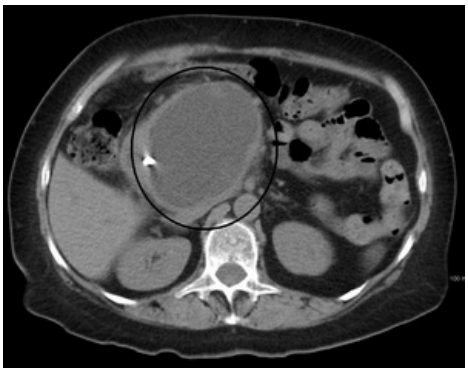
Pancreatic abscess

It is a late form that occurs about 3 – 6 weeks after necrotizing pancreatitis. It takes place under the image of a serious oneseptic wall.

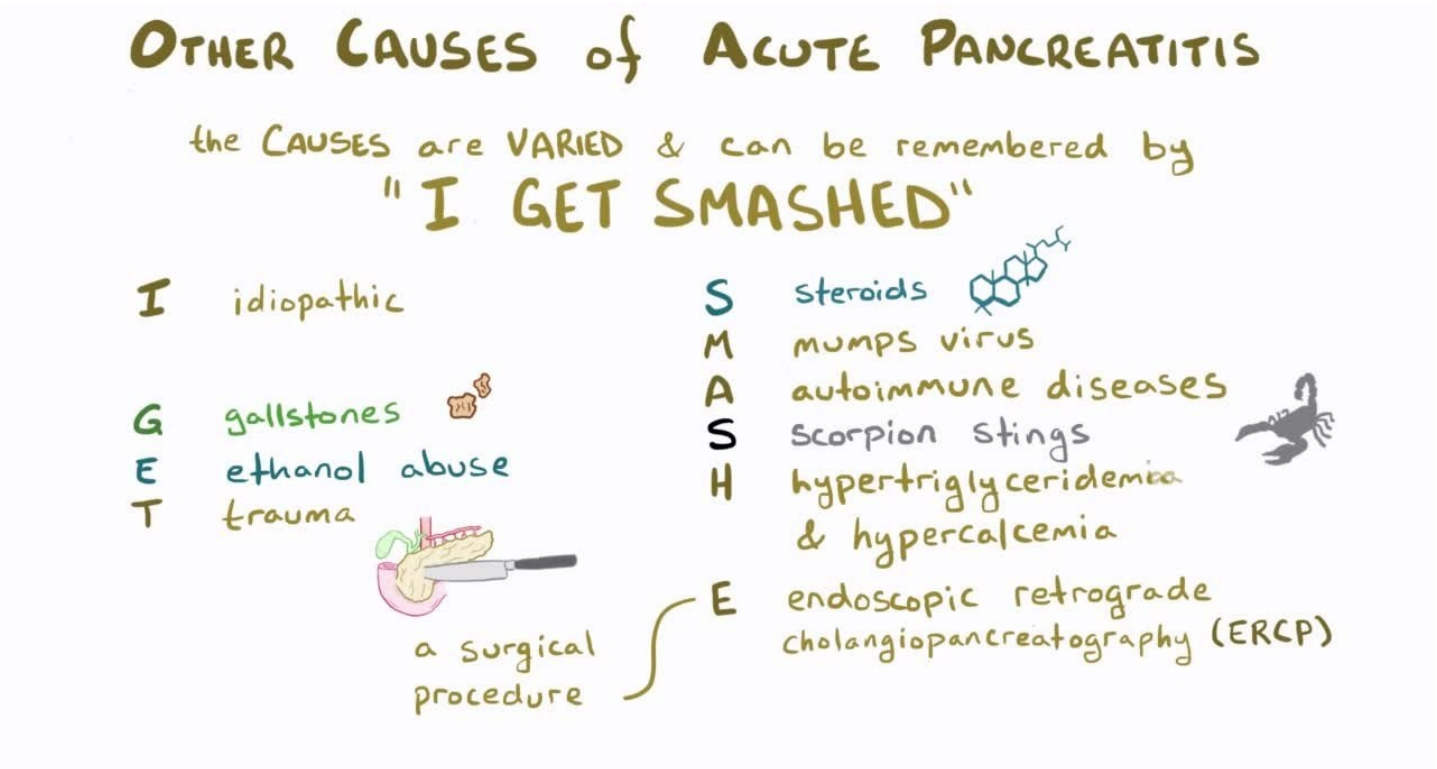
Pseudocysts

They develop several weeks after acute exposure. They are limited collections of fluid with a high concentration of enzymes.

thumb|250px|Pancreas.



Pancreatic pseudocyst.



Ethiology

Main reasons for origin

- Gall bladder, bile ducts and Vater papillae (= acute biliary pancreatitis);
- alcoholism (= acute ethyl pancreatitis);
- postoperative pancreatitis, after ERCP (= acute iatrogenic pancreatitis);
- hyperlipidemic pancreatitis;
- post-traumatic pancreatitis;
- pancreatotoxic pancreatitis (ATB – tetracyclines, diuretics – furosemide, immunosuppressants – cyclosporine, ACE inhibitors, etc.);
- pancreas divisum ((embryonic development disorder – ventral and dorsal junction – is predisposing to acute pancreatitis in adulthood););
- infection – parotitis virus, viral hepatitis.

Mnemonic:: I Get Smashed. **I:** idiopathic, **G:** gallstones, **E:** ethanol, **T:** tumorous, **S:** scorpion stings, **M:** microbiological/mumps, **A:** autoimmune, **S:** surgery or trauma, **H:** hyperlipidemia, hypercalcemia, hypothermia, **E:** emboli or ischemia, **D:** drugs (azathioprine, mercaptopurin, furosemide, estrogen, methyldopa, H2-blockers, antibiotics, salicylates, organophosphates, steroids)[1].

Clinical picture

- Acutely created pain in the abdomen, band-like character, radiate to the back;
- they are accompanied by nausea and subsequent **vomiting without feeling relieved**, weakened peristalsis, meteorism, abdominal wall tension;
- the condition gradually progresses to shocktachypnoea, tachycardia, often hypotension,culminates in circulatory failure, kidneys ARDS;
- left-hand pleural effusion may also be present;
- in biliary genesis – often signs jikteru.

Laboratory

Rise amylase, lipase and CRPin serum, increase glycemia,, hypocalcemia, leukocytosis. However, there may not be deviations from the standard for the most difficult forms.

For**differential diagnosis** is the most important **rise of amylase**.If it rises to more than three times the standard, the diagnosis is almost certain. On the contrary, for **prognosis** the disease is a significant increase **CRP**.It is around 10 mg / l for light forms, and increases to values higher than 200 mg / l (in severe forms, this signals necrosis infection).

Hypocalcaemia is the result of uptake of calcium ions in fat necrosis (so-called. Balzer necrosis, soap).

🔎 *For more information see Acute pancreatitis (laboratory diagnostics).*

Imaging methods

X-ray chest and abdomen

- effusion in the left pleura;
- so-called „ sentinel loop “ – air in the upper loops of the small intestine;
- „ colon cut-off sign “ – level in the lienal bend of the columns.

USG

- the best method is to monitor the course of (resizing, onset of necrosis, concrement in the gallbladder, dilation of the bile ducts).

ERCP ± PST

- especially if biliary genesis is suspected, we can perform papillosphinxotomy (PST) and improve the condition.

CT

- monitoring the course of the disease and determining the stage, especially in obese people, where the USG does not tell us much;
- angio CT – evidence of necrosis.

Differential diagnostics

Acute cholecystitis, gastroenteritis, biliary and left-hand renal colic, ileus, perforation ulcer, rupture aneurysmabdominal aorta, AIM, volvulus, embolism into mesenteric vessels.

Therapy

- Initial phase – intensive conservative therapy with close monitoring of these parameters in the ICU.
- The basis is effort **reduce pancreatic activity** – absolute carcass of oral supply of anything, gastric decompression with nasogastric tube (aspiration of content), the patient's nutritional options are two:
  - nasojeunal probe (currently appears to be better – maintains the natural function of the intestine, which probably reduces the risk of infection with necrosis in the pancreas – does not overgrow bacteria),

parenteral nutrition.

- Intensive volumotherapy (i.v.) – starts at 1 l / h, later decreases to 0.5 l / h (a total of a maximum of 10 – 15 l / day, provided that the patient's cardiovascular system can withstand)..
- Analgesics – Template:HVLP, ev. Template:HVLP (morphineincreases the tone of Oddi's sphincter, they are **KI**).
- ATB – are contraindicated tetracyclines (are pancreatotoxic) preferably cephalosporins, carbapenemy, metronidazole.
- Monitoring CVT, diuresis,...

Indications for surgery

Currently **surgical treatment is not** at the acute stage of this disease **preferred.**. The indications were limited to::

1. **failure of intensive conservative therapy** (failure of intensive conservative therapy
2. **necrosis infection** (is detected by thin needle puncture).

Necrectomy is performed.

Additional procedures are performed after successful conservative therapy (elimination of necrosis, cysts, ...) or in severe complications such as perforation or intestinal stenosis.

Operational therapy

- Removal of necrotic tissue, evacuation and drainage of bacterially infected necrosis, evacuation of pancreatogenic tissue ascit;
- basic rule – gently remove necrosis with maximum maintenance of functional tissue;
- necrosectomy (debridement) associated with continuous postoperative lavage ornamental changes;
- in the most severe cases – open-abdominal treatment (laparostomy);
- resection technique is rarely used – are large blood losses, large lethality.

Systemic complications of pancreatitis

- Pulmonary –atelectasis, pneumonia, hypoxia, ARDS;

- KVS – tachycardia, hypotension, arryhytmia, shock;
- renal – oliguria, azotemia;
- hematological – DIC;
- metabolic – hyperglycemia, hypocalcemia, acidosis, hyperTAG;

prognosis – **Ranson criteria** – are determined by age, leukocytosis, glycemia,LDH and AST.

Links

Related articles

- Acute hemorrhagic necrosis of the pancreas (preparation)
- Acute pancreatitis (laboratory diagnosis)
- Chronic pancreatitis
- Chronic pancreatitis (laboratory diagnosis)
- Pancreatic tumors
- Pancreatic carcinoma
- Acute necrosis of the pancreas/case report

External links

- Template:Acute

Literature used

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Source

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1.