Acute necrosis of pancreas

Acute pancreatitis is an inflammatory disease of the pancreas of a wide range of severity - from mild to severe with signs of multiorgan failure or severe local complications such as abscesses, pseudocysts and necrosis of the pancreas and adjacent tissue.

It is without a doubt one of the most serious and prognostically worst **acute abdominal events.**

Classification of acute inflammation

Interstitial edematous form (70-80%)

It is characterized by interstitial edema, parenchymal inflammation, increased concentration of pancreatic enzymes in the pancreas and systemically. It is usually associated with the pathology of the biliary system. The course is mild or moderate.

Necrotizing form - sterile necrosis, infected necrosis

Within a few days, a massive death of pancreatic cells occurs and there is a necrosis of intra- and extrapancreatic adipose tissue. Calcified fat necrosis (Balzer necrosis) is formed. The course determines the extent of necrosis, retroperitoneal involvement and bacterial contamination.

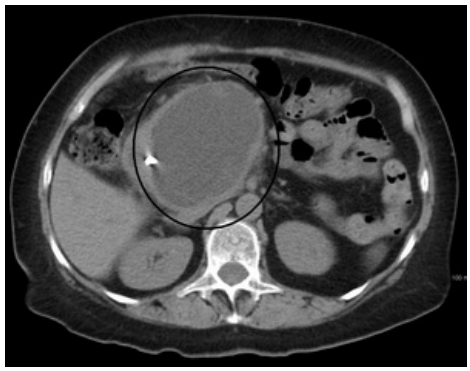
Pancreas abscess

It is a late form that occurs about 3-6 weeks after necrotizing pancreatitis. It manifests by a form of severe septic condition.

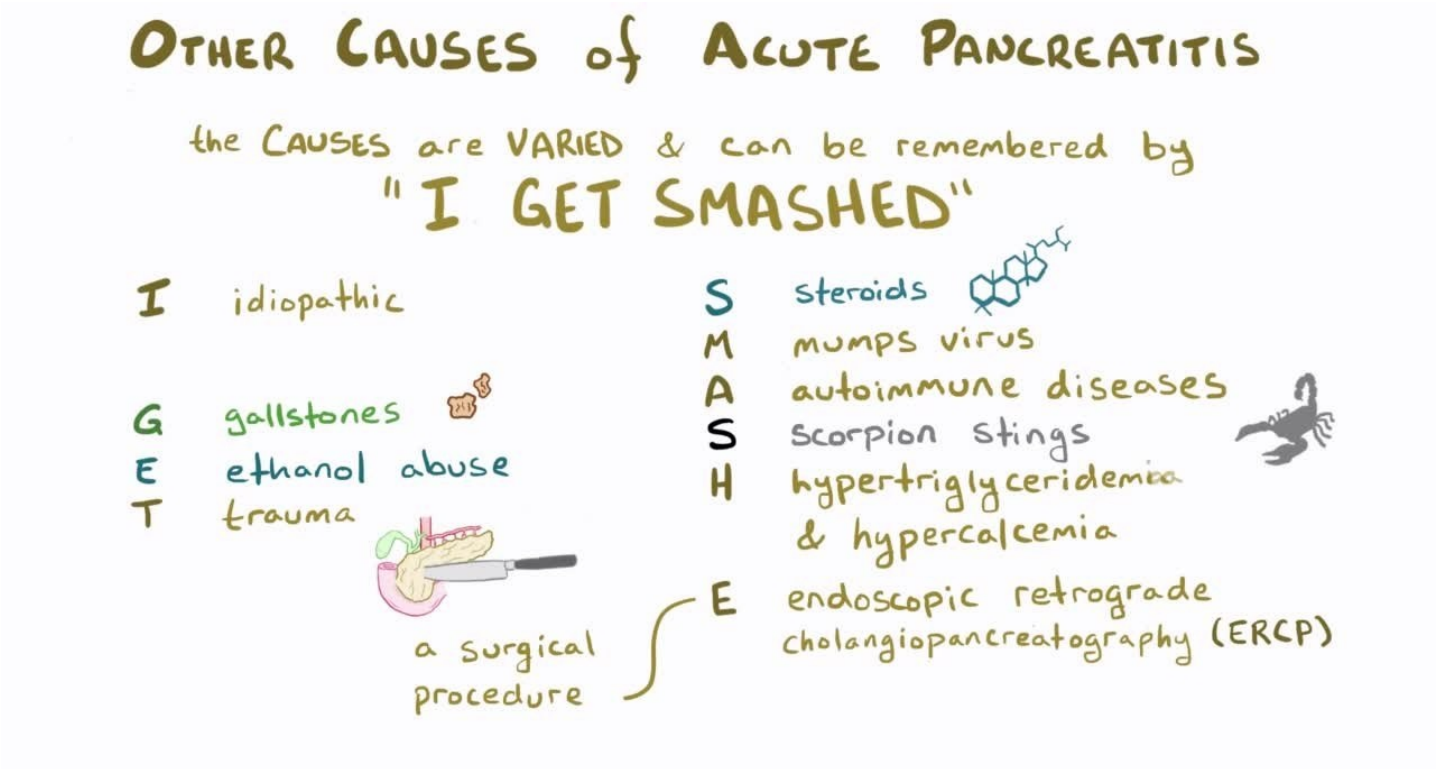
Pseudocysts

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

thumb|250px|Pankreas.



Pancreatic pseudocyst



Etiology

The main reasons for this

- Diseases of the gallbladder, bile ducts and papilla Vateri (= acute biliary pancreatitis);
- alcoholism (= acute ethyl pancreatitis);
- postoperative pankreatitis, after ERCP (= acute iatrogenic pancreatitis);
- hyperlipidemic pancreatitis;
- posttraumatic pancreatitis;
- pancreatotoxically induced pancreatitis (ATB - tetracyclines, diuretics - furosemide, immunosuppressants - cyclosporine, ACE inhibitors, etc.);
- pancreas divisum (a disorder in embryonic development - failure of the ventral and dorsal parts - is predisposing to the development of acute pancreatitis in adulthood);
- infection – virus of parotis, hepatitis caused by viruses

Mnemonic aid: 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 (azathioprin, merkaptopurin, furosemid, estrogen, methyldopa, H2-blokátory, antibiotika, salicyláty, organofosfáty, steroidy)[1].

Clinical picture

- Acute abdominal pain, with character of bands, radiates to the back;
- they are accompanied by nausea and **subsequent vomiting without relief**, weakened peristalsis, flatulence, abdominal wall tension;
- the condition gradually progresses to shock - tachypnoea, tachycardia, often hypotension, culminates in circulatory failure, kidney failure, ARDS;
- left-sided pleural effusion may also be present;
- in biliary genesis - often signs jaundice.

Laboratory

Increase in serum amylases, lipases and CRP, increase in glycaemia, hypocalcaemia, leukocytosis. However, the most difficult forms may not deviate from the norm.

The most significant increase in differential diagnosis is amylase. If it rises to more than three times the norm, the diagnosis is almost certain. Conversely, an increase in CRP is significant for the prognosis of the disease. For light forms it is around 10 mg / l, for heavy forms it rises to values higher than 200 mg / l (this signals necrosis infection).

Hypocalcaemia is due to the uptake of calcium ions in fat necrosis (so-called Balzer necrosis, saponification).

Iron

Imaging methods

X-ray of the chest and abdomen

- effusion in the left pleura;
- „sentinel loop“ – air in the upper loops of the small intestine;
- „colon cut-off sign“ – a level of fluid in lienal flexure of the colon .

USG

- the best method is to monitor the course (changes in size, onset of necrosis, gallbladder stones, dilatation of the bile ducts).

ERCP ± PST

- especially when biliary genesis is suspected, we can perform papillosphincterotomy (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 – proof of necrosis.

Differencial diagnostics

Acute cholecystitis, gastroenteritis, biliary and left renal colic, ileus, ulcer perforation, rupture of abdominal aortic aneurysm, AIM, volvulus, embolism into mesenteric vessels.

Therapy

- Initial phase - intensive conservative therapy with consistent monitoring of the above parameters in the ICU.
- The fundamental therapy is an effort to reduce the activity of the pancreas - the absolute deficiency of oral delivery of anything, decompression of the stomach by nasogastric tube, the patient's nutritional options are twofold:
  - nasojejunal probe (currently appears to be better - it preserves the natural function of the intestine, which probably reduces the risk of infection of necrosis in the pancreas - there is no overgrowth of bacteria),
  - parenterální výživa.
- Intensive volume therapy (i.v.) - starts with 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 it).
- Analgesics : tramadol, fentanyl (morphine increases the tone of Oddi's sphincter, they are KI).
- ATB - tetracyclines are contraindicated (they are pancreatotoxic), preferably cephalosporins, carbapenems, metronidazole.
- Monitoring CVT, diuresis,...

Indication to surgery

Nowadays, surgical treatment is not preferred in the acute stage of this disease.

1. failure of intensive conservative therapy (patient in multiorgan failure, usually a delayed procedure - beware of perioperative mortality);
2. **necrosis infection** (detected by thin needle puncture).

A necrectomy is performed.

Further procedures are performed after successful conservative therapy (removal of necrosis, cysts,...) or with serious complications such as perforation or intestinal stenosis.

Surgical therapy

- Necrotic tissue removal, evacuation and drainage of bacterially infected necrosis, evacuation of pancreatogenic ascites;
- basic rule - gently remove necrosis with maximum preservation of functional tissue;
- necrosectomy (debridement) associated with continuous postoperative lavage of the omental bursa;
- in the most severe cases - treatment by the method of open abdomen (laparostomy);
- Resection technique is rarely used - there is great blood loss, great lethality.

Systemic complications of pancreatitis

- Pulmonary - atelectasis, pneumonia, hypoxia, ARDS;
- CVS – tachycardia, hypotension, arytmia, shock;

- renal – oliguriea, azotemia;
- hematologic– DIC;
- metabolic – hyperglycemia, hypokalcemia, acidosis, hyperTAG;

prognosis– **Ranson's rating** – determined by age, leukocytosis, glycemia, LDH a AST.

References

Related articles

- Akutní hemoragická nekróza pankreatu (preparát)
- Akutní pankreatitida (laboratorní diagnostika)
- Chronická pankreatitida
- Chronická pankreatitida (laboratorní diagnostika)
- Nádory slinivky břišní
- Karcinom pankreatu
- Akutní nekróza pankreatu/kazuistika

External sources

- Template:Akutně

Literature

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Source

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Kategorie:Chirurgie Kategorie:Gastroenterologie Kategorie:Vnitřní lékařství Kategorie:Patologie Kategorie:Patofyziologie

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