Pancreatitis (paediatrics)

Pancreatitis is an autodigestive, inflammatory process of the pancreas that can spread to surrounding tissues and distant organs. Laboratory indicators are increased amylase and lipase in the serum and increased amylase in the urine. Pancreatitis is not common in children.

Causes of pancreatitis in children

- **obstructive/anatomical**: choledocholithiasis, tumors, parasites, pancreas divisum, anomalies of the pancreaticobiliary tree, choledochal cysts, periampullary diverticula of the duodenum;
- **toxins**: azathioprine, mercaptopurine, valproate, estrogens, corticosteroids, metronidazole, nitrofurantoin, furosemide, sulfonamides, erythromycin, methyldopa, ranitidine, salicylates,...
- **trauma**: blunt trauma to the abdomen, iatrogenic damage during abdominal surgery or during ERCP;
- **metabolic abnormalities**: hypertriacylglycerolemia, hypercalcemia, organic acidemia, rapid hyperalimentation, malnutrition, cystic fibrosis, uremia, diabetes mellitus, antitrypsin deficiency;
- **infections *viral***: mumps, rubella, hepatitis A, B, Coxsackie B, ECHO viruses, adenoviruses, EBV, HIV; ***bacterial***: *Mycoplasma pneumoniae, Campylobacter jejuni, Mycobacterium tuberculosis*; ***parasitic****: ascariasis, malaria;*
- **vascular changes**: ischemia, hemolytic uremic syndrome, Henoch-Schönlein purpura, Kawasaki syndrome, systemic lupus erythematosus, polyarteritis nodosa, malignant hypertension;
- **miscellaneous**: penetrating peptic ulcer, Crohn's disease, Reye's syndrome, hypothermia, hereditary pancreatitis;
- **idiopathic**.[1]

Acute pancreatitis

Etiology

- the most common cause of severe pancreatitis: blunt trauma to the abdomen (falling on the handlebars of a bicycle, kicking the abdomen, etc.);
- anomaly of the pancreaticobiliary tree;
- more rarely, metabolic diseases;
- causes of mild pancreatitis: multisystem diseases, toxins and drugs, viral infections.

Pathogenesis

- activation of trypsinogen → uncontrolled activation of inactive zymogens of pancreatic acinar cells → enzymatic cascades → local parenchymal lesions (interstitial edema, necrosis, hemorrhage – autodigestive inflammatory process) and activation of kallikrein-kinin system → damage to extrapancreatic tissues → systemic changes: hypovolemia, hypotension, shock, ascites, pleural effusion, acute respiratory distress syndrome, renal failure, hemocoagulation changes);
- antiproteases and the immune system try to stop enzymatic cascades and the inflammatory response.

Clinical picture

1. mild pancreatitis
   - uncomplicated course, minimal or no overall symptoms, resolve within 10 days;
   - pain in the epigastrium, loss of appetite.
2. severe acute pancreatitis
   - severe course and general symptoms or local complications in the pancreas (necrosis, pseudocyst, abscess);
   - sudden and sharp progressive pain in the epigastrium, worse after eating, sometimes shooting into the back;
   - loss of appetite, nausea, vomiting and flatulence, disorders of the intestinal passage;
   - tachycardia, hypotension, jaundice, etc.;
   - rarity is Cullen's, event. Grey-Turner's sign - blue-violet coloration around the navel, possibly. in the groin;
   - in children exceptionally: shock, infection, sepsis, DIC, hypocalcemia, hyperglycemia, MODS.

Diagnosis

- increased serum amylase and lipase (possible increase in urine as well);
- a rise in CRP, neutrophilia, hypocalcemia and hyperglycemia is alarming;
- ultrasound: interstitial changes, enlargement of the pancreas, necrosis, pseudocysts (the wall consists of granulation tissue, the lining is without epithelium; often palpable);
- CT with contrast and MR: focal changes;
- MRCP (magnetic resonance cholangiopancreatography): anomalies of the hepatobiliary tree.

Therapy

- light forms: short-term cessation of diet (until the pain disappears) and then a diet;
- more severe forms: intravenous hydration, correction of the internal environment, prevention of shock,

analgesia (morphine derivatives are contraindicated for contraction of the sphincter of Oddi), necrotizing pancreatitis – antibiotics (to prevent infection of necrotic foci, e.g. with meropenem), enteral nutrition (by nasojejunal tube, then by nasogastric and then by mouth), in the most severe stages parenteral nutrition, vomiting – proton pump inhibitors.[1]

Chronic Pancreatitis

Etiology

- cystic fibrosis;
- severe hereditary pancreatitis with AD inheritance,
  - Pancreatitis attacks already in toddlers, more often around 10-12 years old,
  - high risk of pancreatic adenocarcinoma;
- congenital or post-traumatic anomalies of the pancreatic and bile ducts (pancreas divisum – incomplete fusion of the dorsal and ventral parts of the pancreas, drainage through the accessory ductus Santorini, difficult outflow of pancreatic juice);
- hyperlipidemia;
- hyperparathyroidism;
- autoimmune pancreatitis - rare;
- idiopathic.

Pathogenesis

- permanent irreversible inflammation of the pancreas → precipitation of proteins in the pancreatic ducts, atrophy of the epithelium → calcification and stenosis of the pancreatic ducts;
- diffuse destruction of the parenchyma → fibrotization → insufficiency of external secretion → insufficiency of internal secretion;
- takes place continuously or in attacks.

Clinical picture

- chronic or acute recurrent pain in the epigastrium, bloating, nausea, vomiting;
- later manifestations of insufficiency of external secretion: voluminous, smelly, light, greasy or oily stools (steatorrhoea), failure to thrive, secondary malabsorption;
- then manifestations of internal secretion insufficiency: diabetes mellitus.

Diagnosis

- see acute pancreatitis;
- elastase 1 in the stool (examination of pancreatic exocrine secretion).

Therapy

- attacks: same as acute pancreatitis;
- substitution of insufficiency of external secretion: lipase, fat-soluble vitamins, calcium and trace elements;
- diet: low-fat, non-bloating, unseasoned, smaller but more frequent portions, full of energy.

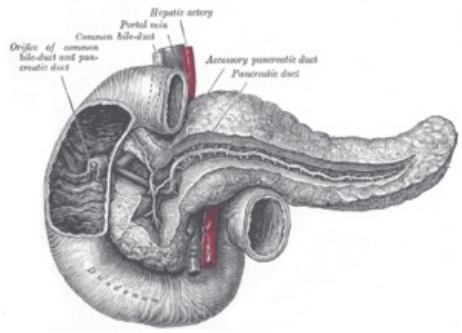
Prognosis

- if fully developed in childhood – unfavorable prognosis.[1]

Pancreatic Physiology

Exocrine function (pancreatic juice)

- ions: bicarbonate (in the duodenum to neutralize HCl from the stomach), Na, Cl, K;
- enzymes: lipase, amylase, ribonuclease, deoxyribonuclease;
- proenzymes: trypsinogen, chymotrypsinogen, procarboxypeptidase, proelastase, phospholipase A2 – activated in the duodenum and small intestine;
  - trypsinogen is activated by enterokinase to trypsin, which further converts other proenzymes;
- mucoproteins, plasma proteins, trypsin inhibitor (antitrypsin);



The pancreas and its ducts.

Endocrine function

- α-cells: glucagon; β-cells: insulin; δ-cells: gastrin.[2]

Diseases accompanied by external pancreatic insufficiency

- cystic fibrosis – bicarbonate secretion disorder in the pancreatic ducts → duodenal juice alkalinization disorder; high protein content in pancreatic secretion → intraductal protein precipitation → obstruction of small pancreatic ducts; fibrotic remodeling of the pancreas;
- chronic pancreatitis;

- Shwachman-Diamond syndrome – AR hereditary defect of microtubular cell elements; progressive degeneration and lipomatous remodeling of the pancreas;
- Pearson syndrome – mitochondrial DNA defect; fibrotization of the pancreas;
- Johanson-Blizzard syndrome – pancreatic hypoplasia;
- enterokinase deficiency of brush border enterocytes of the duodenum (rare) – does not activate pancreatic proteases;
- trypsinogen deficiency (rare).[1]

Videos

Video explanation of acute pancreatitis.

Video explanation of chronic pancreatitis.

Links

Related Articles

- Pancreatitis • Acute pancreatitis • Chronic pancreatitis • Autoimmune pancreatitis

Bibliography

References

1. LEBL, J – JANDA, J – POHUNEK, P, et al. *Clinical Pediatrics.* 1. edition. Galen, 2012. 698 pp. pp. 350-353. ISBN 978-80-7262-772-1.
2. NEČAS, Emanuel, et al. *Pathological physiology of organ systems : Part II.* 1. edition. Prague : Karolinum, 2003. 380 pp. pp. 462-463. ISBN 80-246-0674-7.