ACUTE PANCREATITIS

Pancreatitis is initially aseptic inflammatory process in which active lipolytic and proteolytic pancreatic enzymes autodigest the gland. Recurrent attacks are referred to as chronic pancreatitis. It is characterized by long run of fibrosis, calcification with disorder or complete loss of its external and internal secretory functions.

ANATOMY

Location

The pancreas lies posterior to the stomach and small omentum in the retroperitoneal space. It is lies anterior to the inferior vena cava, aorta, splenic vein, and left adrenal gland.

Parts

The pancreas is divided into four parts: the head, neck, body, and tail. The head lies within the duodenal C loop. The neck of the gland extends medially from the head and lies anterior to the portal and superior mesenteric vein and superior mesenteric artery. The body extends from the neck toward the spleen, whereas the tail extends into the splenic hilum.

Blood Supply

The celiac trunk and the superior mesenteric artery provide the arterial supply to the pancreas.

The body and tail are supplied by branches of the splenic artery.

The head receive its supply through branches of the gastroduodenal artery that is branch of the celiac trunk and from the branches of the superior mesenteric artery.

Venous drainage is to the splenic, superior mesenteric, and portal veins.

Innervation

The pancreas is innervated by both sympathetic and parasympathetic components of the autonomic nervous system.

Ducts

The main pancreatic duct, or duct of Wirsung, begins in the tail of the pancreas and terminates at the papilla of Vater in the duodenum. The duct within the head is 3.1 to 4.8 mm in diameter and gradually tapers to 0.9 to 2.4 mm in the tail.
The duct of Santorini (i.e., the minor, or accessory, pancreatic duct) is smaller than the main duct. It extends from the main duct to enter the duodenum at the little papilla, that lies about 2 cm proximal and slightly anterior to the major papilla.

**Normal pancreatic function**

It is responsible for insulin production (endocrine pancreas) and the manufacture and secretion of digestive enzymes (exocrine pancreas) leading to carbohydrate, fat, and protein metabolism. Approximately 80% of the gross weight of the pancreas supports exocrine function, and the remaining 20% is involved with endocrine function.

Digestive enzymes are produced within the pancreatic acinar cells.

**ETIOLOGY**

Acute pancreatitis is the polyetiological, but monopathogenic disease. The main factors of starting of acute pancreatitis are:

- diseases of extrahepatic cholic ways with the disorder of bile flow;
- obturative acute pancreatitis;
- excessive loading on pancreas;
- long-standing alcohol consumption;
- endoscopic retrograde cholangiopancreatography
- acute and chronic disorders of circulation of blood with the disorders of microcirculation in pancreas;
- diseases of pancreas (tumours and chronic pancreatitis and etc.);
- traumas of pancreas;
- postoperative pancreatitis;
- acute poisoning by some poisons, use of toxic doses of medicinal preparations.

**THEORIES of ARISING**

1. Theory of bile reflux into the pancreatic duct (E. Opie, 1901). Regurgitation of bile into the system of the pancreatic ducts increases the intraductal pressure, which results in destruction of the glandular cells.
2. Theory of **hypertension** of the pancreatic duct (A. Rich, G. Duff, 1936). The increase of pressure in the ductal system of the pancreas results in rupture of its acinoses and small ducts and leads to the damage of cells.


4. **Allergic** theory. Role of allergy in development of acute edema and hemorrhagic necrosis of the pancreas is noted by many authors.

5. Role of **infection** in development of acute pancreatitis is confirmed by the fact, that acute hemorrhagic pancreatitis may arise in acute inflammatory process in the gallbladder, in acute parotitis and other infectious diseases. Infection penetrates the pancreas through the blood and lymph vessels.

**PATHOGENESIS**

Distinguished two stages:

**I. Tripsin stage.**
Cytokine activates tripsinogen, transforming it in tripsin. Tripsin and tripsinogen affect on the interstitial tissue and vessels of the pancreas, which result in edema, stasis and hemorrhage. In such conditions there is cell death and death of the glandular tissue.

**II. Lipase stage.**
Lipase initiates the development of fat necrosis. It is activated by salts of fatty acids. The presence of edema, hemorrhage and fat necrosis result in destruction of the pancreatic tissue, extension of edema to surrounding tissues, to transudation of fluid into the abdominal and pleural cavities, and sometimes into the pericardial cavity and into the retroperitoneal space.

In acute pancreatitis the following "local" **pathomorphologic changes** are noted:

1. Edema of the pancreas and surrounding tissues.
2. Fat necrosis.
3. Formation of hemorrhagic foci.
4. Necrosis of the pancreatic parenchyma.
5. Suppurative inflammation.

**CLASSIFICATIONS**

**Classification** of acute pancreatitis (Saveliev, 1983)

I. **Clinical - anatomical forms:**
1. Edematous pancreatitis (abortive pancreonecrosis).
2. Fatty pancreonecrosis.
3. Hemorrhagic pancreonecrosis.

II. **Prevalence:**
1. Local (focal) process.
2. Subtotal process.
3. Total process.

III. **Current:**
1. Abortive.
2. Progressive.

IV. **Periods of illness:**
1. Period of hemodynamic disturbances and pancreatogenic shock (1-3 days).
2. Period of functional insufficiency of parenchymal organs (4-7 days).
3. Period of degenerative suppurative complications (8-10 days).

**Classification** of acute pancreatitis (Shalimov, 1990)

I. **On the morphological changes:**
1. Edematous pancreatitis:
   a) Serous;
   b) Serous-hemorrhagic.
2. Necrotic pancreatitis (pancreonecrosis):
   a) Hemorrhagic (small-focal, large-focal, subtotal, total);
   b) Fatty (small-focal, large-focal, subtotal, total);
   c) Mixed (small-focal, large-focal, subtotal, total).
3. Purulent pancreatitis:
   a) Primary purulent;
   b) Secondary purulent;
   c) Intensification of chronic festering pancreatitis.

II. **On the degree of severity:**
1. Easy degree;
2. Middle degree;
3. Heavy degree;
4. Extremely severity of illness (lightning form).

III. **On the clinical current:**
1. regressing;
2. progressing;
3. recidivous.

IV. **On the presence of complications:**
1. Local complications, complications from the side of gland;
2. Intraperitoneal complications;
3. Extraperitoneal complications.

**CLINICAL PICTURE**

Clinical picture of acute pancreatitis depends on form of the pathologic process and stage of the disease.
The cardinal symptom of acute pancreatitis is **abdominal pain**, which is dull, boring, and steady. Usually, the pain is sudden in onset and gradually intensifies in severity until reaching a constant ache. Most often, it is located in the upper abdomen, usually in the epigastric region, but it may be perceived more on the left or right side, depending on which portion of the pancreas is involved. The pain radiates to the back.

Pain in acute pancreatitis may be moderate in edematous form of pancreatitis and unbearable in pancreonecrosis.

The cause of pain in acute pancreatitis is compression of nerve plexuses, which are located around the pancreas; they may occur in enlarged pancreas and in case when edema extends on the parapancreatic fat.

**Nausea and vomiting** are often present, along with anorexia. Vomiting is appears simultaneously with pain or accompanies it, may be recurrent and persistent, and sometimes becomes uncontrollable. Diarrhea can also occur. Some patients with acute pancreatitis notes distension of abdomen and retention of gas.

Fever and tachycardia are common abnormal vital signs. Hypotension may be noted.

Abdominal tenderness, muscular guarding are observed in most patients; guarding tends to be more pronounced in the upper abdomen.

When examining the **skin integuments** may be revealed:

1. Mondor's symptom — violet spots on the body and face alternating with the sites of the pale skin;
2. Halsted's symptom — cyanosis of the abdomen skin;
3. Turner's symptom — cyanosis of the lateral surfaces of the abdomen and the lumbar region;
4. Grunwald's symptom — petechial skin rash in the navel area.

**LABORATORY STUDIES**

**Serum amylase and lipase** levels are typically elevated. These elevations may only indicate pancreastasis. Elevated lipase levels are more specific to the pancreas than elevated amylase levels.

A complete blood count demonstrates **leukocytosis**.
Increase of **urinary amylase (diastase)** activity (over 128 units according to Volgemuth).

**SPECIAL METHODS OF INVESTIGATION**

**Ultrasonography** of the abdomen is the most useful initial test in determining the etiology of pancreatitis.

**Computer tomography** is "a gold standard" in topical diagnostics and the most sensing method of investigation in acute pancreatitis and its complications. It reveals the enlargement of the pancreas, which shadow has well defined outlines in edematous form of acute pancreatitis, but in hemorrhagic, necrotic and suppurative pancreatitis the outlines of the pancreas become blurred. By means of CT it is possible to reveal pancreatogenic abscesses and fluid masses in the retroperitoneal space at early stage of the disease.

**BASIC PRINCIPLES OF TREATMENT OF ACUTE PANCREATITIS**

Patients with acute pancreatitis should be hospitalized in the intensive care unit.

**Prehospital first aid:**
- Spasmodyltics;
- Antihistamines;
- Infusion therapy;
- Non-narcotic analgesic;
- Oxygen therapy;

In the **intensive care unit:**
- Protease inhibitors;
- Proton pump inhibitors;
- H2 blockers;
- Antibiotics;
- Infusion therapy:
  1. protein preparations (albumin, plasma, solutions of amino acids);
  2. crystalloid to compensate for the need for Na, K, Ca, Cl, Mg;
  3. isotonic and hypertonic solutions of glucose with insulin;
- Parenteral nutrition (lipofundin);
- Bed rest;
- Nasogastric tube - to view the evacuation of gastric contents;
- External and internal hypothermia (To suppress the external function);

**The indications for surgical treatment are:**
- Acute pancreatitis complicated by purulent peritonitis;
- When there is suppurative pancreatitis;
- With increasing of obstructive jaundice (bile flow obstruction);
TYPES OF SURGERY

I. **Minimally invasive intervention:**
   1. laparoscopic drainage of the abdominal cavity;
   2. endoscopic retrograde papillosphincterotomy;
   3. ultrasound guided drainage of fluid accumulation.

II. **Necrectomy** - removal of necrotic gland within the viable tissue;

III. **Sequestrotomy** - removal of necrotic gland within non-viable tissue;

IV. **Omento-pancreatopexy** - moving part of the greater omentum is carried out through a hole in the gastro-colon ligament and fix the seams to separate the peritoneum along the top and bottom edges of the pancreas. Omento-pancreatopexy facilitates delimitation of process, improves blood supply to the pancreas, accelerates the organization and encapsulation necrotic areas. Omento-pancreatopexy shown at small focal necrosis.

V. **Abdominisation of pancreas (mobilization of all surfaces)** - Tissues nearby pancreas (along the bottom and top edges of the body and tail) infiltrated with novocaine, then cut the parietal peritoneum. Under the body and tail of gland is hold a part of the greater omentum and wrap it around gland. This operation is able to prevent the ingress of enzymes and degradation products in the retroperitoneal space.

VI. **Drainage of omental pouch and for retroperitoneal space with small incisions.**

   The early opening of retroperitoneal space and evacuation of exudates helps to avoid the development of heavy forms of pancreatitis and parapancreatitis and to avoid the development of formation of sequesters and cysts.

   At the revealing of signs of hypertension of bilious ways it is necessary to perform cholecystostomy for the external drainage of bilious ways.

   If there are stones in gall bladder even at absence of signs of inflammation of gall bladder, cholecystolytotomy with the applying of cholecystostomy is carried out, or cholecystectomy with draining of choledoch through the stump of cystic duct.

**PANCREATIC DUCT DISRUPTION**

Damage to the pancreatic ductal system may allow the pancreatic juice to leak from the gland. The sudden rapid increase in retroperitoneal fluid on computed
tomography is suggestive of this condition. Fluid amylase or lipase levels in the 10,000s strongly suggest the presence of a ductal disruption.

In the appropriate clinical setting, ERCP confirms the diagnosis and provides a treatment option - transpapillary stent placement or placement of nasopancreatic tube. Occasionally, leaks are associated with downstream stenoses that are also amenable to endoscopic treatment. Refractory cases may warrant surgery.

**PSEUDOCYSTS**

Peripancreatic fluid collections persisting for more than 4 weeks are referred to as acute pseudocysts. Pseudocysts lack an epithelial layer and thus are not considered true cysts. They also differ from true cysts in that they are usually filled with necrotic debris rather than fluid.

Most pseudocysts can be followed clinically. However, when they are symptomatic (ie, associated with pain, bleeding, or infection) or are larger than 7 cm and are rapidly expanding in an acutely ill patient, intervention is indicated.

In selected patients with very large fluid collections, percutaneous aspiration of pancreatic pseudocysts is a reasonable approach.

**Transpapillary drainage** requires the main pancreatic duct to communicate with the pseudocyst cavity, ideally in the head or body of the gland. The proximal end of the stent (which should be smaller than the diameter of the pancreatic duct) is placed into the cavity. However, pancreatic stents are difficult to monitor, are prone to obstruction, and carry an increased risk of infection and ductal injury.

Some noncommunicating pseudocysts may be amenable to transmural enterocystostomy. Technical success requires a mature cyst that bulges into the foregut, and the distance from the lumen to the cyst cavity should be less than 1 cm. The transduodenal approach is associated with fewer complications and recurrences than the transgastric approach.

**Surgical treatment:**

1. excision a part of pancreatic gland that communicate with the pseudocyst;
2. external drainage;
3. internal drainage: cystogastrostomy, cystojejunostomy, cystoduodenostomy.
PANCREATIC ABSCESS

Pancreatic abscesses generally occur late in the course of pancreatitis. Many of these respond to percutaneous catheter drainage and antibiotics. Those that do not respond require surgical debridement and drainage.