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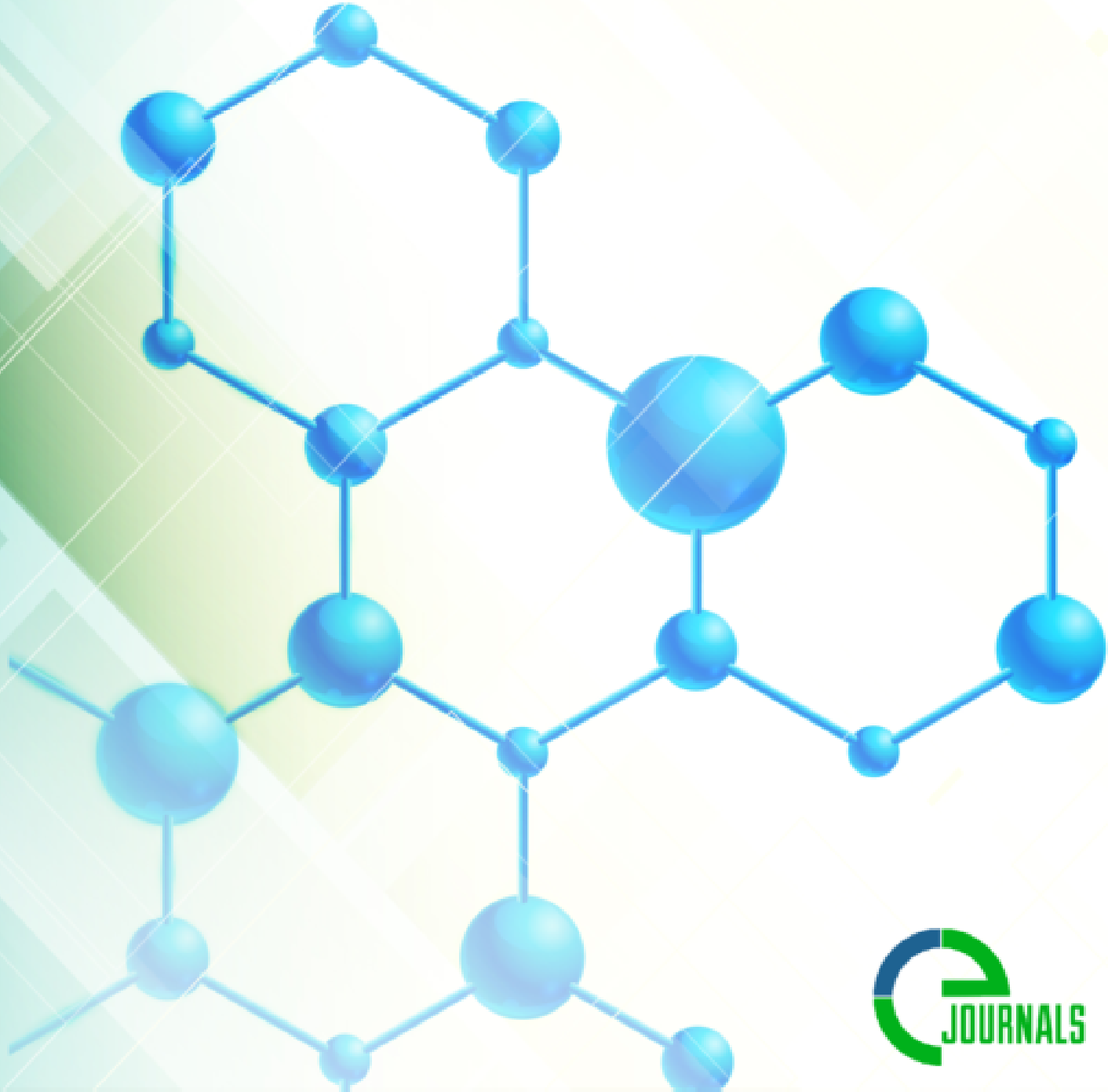

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ACUTE PANCREATITIS

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Abstract: Acute pancreatitis, its symptoms and treatment occur in both adults and children. This is an inflammation of the pancreas, in which the pathological process violates the integrity of pancreocytes, resulting in enzymatic auto aggression with the further development of necrotic changes, degeneration of the gland and subsequent bacterial infection. This article deals with giving a brief overview of the diagnosis and treatment methods of acute pancreatitis.

Keywords: Acute pancreatitis, metabolic theory, flow-enzymatic theory, Immunological theory, traumatic impact, reflux

Introduction

For the first time, mention of acute pancreatitis is found in scientific works in the 16th century. In 1578, S. Alberti described the clinical picture of the disease. In 1870, acute pancreatitis was singled out as a separate nosological unit. The beginning of the 20th century was marked by a scientific breakthrough in diagnostics, which significantly increased the detection of pathology before surgery. To do this, in patients with a picture of an acute abdomen, the content of diastase in the urine was determined, an increase in which is characteristic of damage to the cells of the pancreas.

In the middle of the last century, the principles of pathogenetic therapy were developed. Treatment of acute pancreatitis has become combined. Depending on the activity of the pathological process, various combinations of drug therapy and surgical correction were used.

Approximately 120 years ago, scientists formulated the first theory of the pathogenesis of the disease.

Methods and literature review

Theories of the pathogenesis of acute pancreatitis:

Metabolic theory - the development of acute pancreatitis is based on significant metabolic disorders within the organ, due to which enzymatic auto aggression occurs.

Flow-enzymatic theory - the trigger for the development of pathology is bile reflux into the pancreas, which provokes aseptic inflammation.

Circulatory theory - circulatory disorders affect the resistance of pancreatic tissue to its own enzymes.

The theory of toxic shocks - scientists have suggested that an increase in the permeability of the walls of blood vessels located in the pancreas stimulates the release of red blood cells from the bloodstream. In this case, they come into contact with enzymes, which provoke the formation of toxic compounds that damage the pancreatic tissue. Subsequently, it was proved that contact with blood cells activates trypsin, which causes necrotic processes in the organ.

Immunological theory - the primary damage to pancreocytes is caused by circulating immune complexes against their own tissues.

Numerous studies have shown that the inflammatory process in the pancreas has a polyetiological nature. The cause of the acute form of pancreatitis is usually combined. There are three groups of etiological factors:

1.mechanical;
2.toxic-allergic;
3.neuro-humoral.

Mechanical Cause Group

This group includes all conditions in which mechanical damage to the pancreas occurs:

Reflux. Throwing into the ductal system is observed with cholelithiasis, oncological pathology of hepatobiliary or duodenal localization, increased pressure in the ampulla of the major duodenal papilla, and ulcerative processes in the intestine.

Traumatic impact is the cause of pancreatitis in 5% of patients, often complicating surgical interventions in the abdominal cavity.

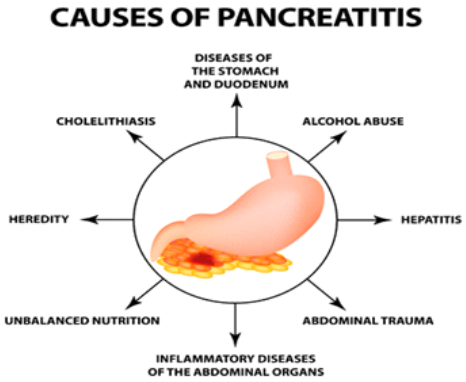

Figure 1

Toxic-allergic group of causes. The most significant etiological factors from this group are:

alcohol abuse;
food allergy;
allergic reactions to medications.

The above factors damage the endothelium of the pancreatic ducts, which is accompanied by damage to its own tissue by enzymes. Alcohol also enhances the secretory activity of the organ and can increase intraductal pressure.

Neuro-humoral group of causes

Symptoms of acute pancreatitis occur as a result of metabolic disorders, most often fat, with the development of hyperlipidemia in the blood. This group of factors also includes pregnancy and the postpartum period, since at this time a pronounced violation of neuro-humoral reactions can be observed.

Results and discussions

Phases of Enzyme Theory

Lipolysis. Under the influence of various combinations of etiological factors, pancreatic enzymes lipase and phospholipase enter the tissue of the organ, causing the death of pancreocytes and necrotic changes in fatty tissue. This is called the autodigestive process. During this period, the first symptoms of acute pancreatitis appear.

Demarcation inflammation. In this phase, necrosis of adipose tissue occurs with the development of a protective inflammatory reaction. This is facilitated by a large number of mediators and biologically active substances released in the area of inflammation.

Proteolysis. In some cases, destruction of its connective tissue is observed in the pancreas. This is due to the activation of trypsin and other lysosomal enzymes. As a result,

blood vessels, plasma are damaged, and blood cells sweat the parenchyma of the organ. Necrosis changes from fat to hemorrhagic.

Diagnosis

I/ Complete blood count - high hematocrit (hemoconcentration), leukocytosis, shift to the left, increased ESR.

2/ Serum amylase is increased (more than 7 mg/s/l), absent in pancreatic necrosis.

3/ Amylase (diastase) urine more than 26 mg/s/l.

4/ Blood transaminases are elevated, which is very characteristic (ASAT more than 125, ALAT more than 189).

5/ Bilirubin (norm up to 20.5 mg/l); sugar more than 5.5 mmol / l, 6 / Urea and residual nitrogen in the blood - increased.

7/ Heminic compounds in serum are sharply increased (up to 30-40 units at N - 9 - II).

8/ Prothrombin index - increased especially in the elderly.

9/ Blood calcium decreases (N 2.24 - 2.99 mmol / l), especially in severe forms

10/ Ionogram - decrease in K, chlorides

11 / Analysis of peritoneal exudate - an increase in the content of amylase,

12/ X-ray examinations: a/ increase in the shadow of the pancreas (soft rays) - a direct sign b/ deployed horseshoe duodenum c/ reactive effusion in the sinus, d/ distension of the transverse colon, ^ indirect e/ limitation of diaphragm mobility, / signs e/ blurring contours of the left.b.clar.muscle (symptom of Pchelkina)

13 / Ultrasound diagnostics - changes in the size of the gland, its departments (currently considered the leading additional study).

14/ Computed tomography (if possible).

15/ Laparocentesis with the study of the contents of the abdominal cavity for enzymes.

16/ Laparoscopy - spots of stearic necrosis, bile impregnation, exudate for enzymes. Differential diagnosis is carried out with acute gastritis, food poisoning, ulcer perforation, intestinal obstruction, thrombosis of mesenteric vessels, myocardial infarction, ectopic pregnancy.

With the correct diagnosis, only about a third of patients enter the hospital.

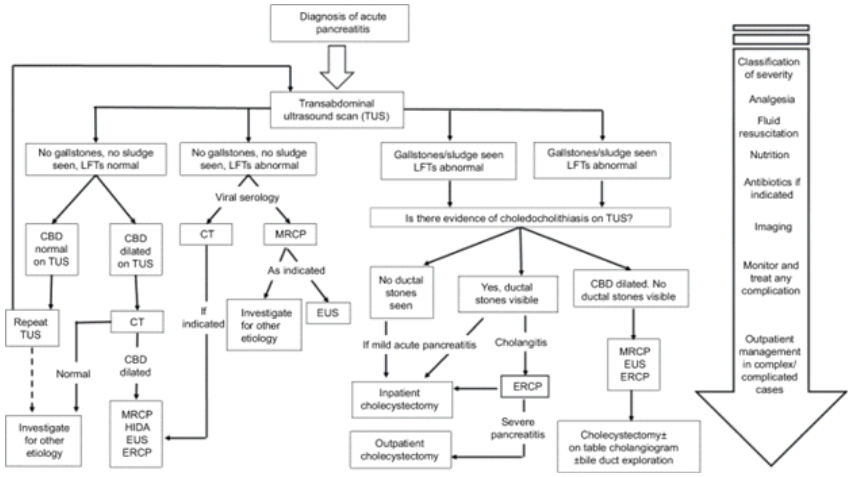

Figure 2 A flowchart encompassing the patient's journey from diagnosis of acute pancreatitis through to further investigation and definitive management.

Conclusion

The prognosis for acute pancreatitis is very serious, despite the development of pathogenetically substantiated conservative and surgical methods of treatment. This is

associated with a high lethal outcome of the disease, which can be up to 85% in patients with complicated forms of pancreatic necrosis. The most common cause of death is multiple organ failure. Acute pancreatitis is usually accompanied by a sharp deterioration in health and requires treatment in a hospital. As a rule, acute pancreatitis is hospitalized in departments and clinics of general surgery or abdominal surgery.

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