ON THE MATTER OF STRESS PANCREATITIS

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Emotions usually pass after some time. But what they did stays.

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Key words: acute stress pancreatitis, stress gastroduodenal ulcers, pathogenesis, clinical picture, treatment

On the background of a difficult situation in Donbass, which is stressful for many people, we have encountered cases of stress pancreatitis, which was previously read about only in the literature [4, 5, 6].

Many doctors are familiar with the pathogenesis and clinical features of stress gastroduodenal ulcers. Stress is a common defensive reaction to any strong stimulus, a kind of adaptive syndrome. The protective nature of stress is relative: it causes disorders of varying degrees of severity of the number of organs and systems, including the mucous membrane of the stomach and duodenum, heart, kidneys, liver, pancreas, and so on. It is known that the formation of stress gastroduodenal ulcers happens mostly due to ischemia of the mucosa and, as a result, the suppression of reparative processes in the predominance of aggressive factors (hydrochloric acid, pepsin, bile acids, lysolecithin). Different in strength and duration emotional or physical injury, mental or physical exhaustion, surgery, sepsis, burns, severe somatic diseases can be the stress factors [3].

Pathogenesis of stress ulcers involves not only a violation of microcirculation, increased secretory and motor functions of the stomach, but also increased production of corticosteroids, neurohumoral changes, reducing resistance of the gastroduodenal mucosa. The variety of mechanisms of formation of stress ulcers and their insufficient studying explain the low efficiency of their prevention and treatment [3]. Scheme of modern concepts of the pathogenesis of stress ulcers is shown on Fig. 1.

Spasm of small arteries (Fig. 2), unfibring vessel walls (Fig. 3), shedding endothelium, cell infiltration, pictute of vasculitis, invagination of arteries (Fig. 4), alternation of spastic and atonic fragments in microvessels (Fig. 5), hemorrhagic infiltration paraulcer tissues (Fig. 6).

The pathogenesis of stress pancreatitis has been studied in a much lesser degree. First of all, it should be recalled that the first phase of the exocrine pancreatic secretion is the brain phase. It has a compound reflex mechanism, i.e. it is realized through the central nervous system by the conditioned and unconditioned reflexes. The proportion of the first phase in the total postprandial pancreatic secretion is evaluated differently by various authors (from 10 to 50%). Emotional states, pain and other effects affect the brain phase of pancreatic secretion [2].

According to Prof. G. F. Korot'ko, pathogenesis of stress pancreatitis depends largely on the type of stress and stressor. The main mechanism is insufficient hemoperfusion of the pancreatic tissue due to violation of systemic and local blood circulation in the body (vasoconstriction — Fig. 7, arteriolar invagination, microcirculation disturbance — Fig. 8, thrombogenesis, hemorrhagic infiltration of pancreatic tissue — Fig. 9), it ischemia. Under these conditions, the barrier ability of ducts wall, their structure, antitryptic system (protease activation and inhibition of antiproteolytic activity) are broken, "deviation" of hydrolases in periductal space happens, lymph and blood flow increases. Intraductal important factor in increasing the pressure, leading to a violation of transport secret act, increasing the viscosity ekzosekreta and its outflow obstruction of the duct system dilated ductal cancer valves. The last reason is universal in the genesis of acute pancreatitis. Disorder of the mechanisms of multilevel self-regulation of functions under stress is also important, including pancreatic function, which is typical the effect of extreme influence [2].

We observed 12 patients with stress pancreatitis, i.e. with such a pancreatitis that didn't have any other causes besides stress. Of particular note is the absence of alcohol intake, which confirms not only the patient, but also the results of the study γ-glutamyl transpeptidase. In 5 patients, stress pancreatitis was combined with stressful

gastroduodenal ulcers, and the remaining 7 patients had pancreatitis without erosive and ulcerative lesions of the stomach and/or duodenum. Course of stress pancreatitis coincided with normal course of acute edematous pancreatitis: patients complained of classical pancreatic pain, nausea, vomiting, abdominal distension. All the patients had varying degrees of hyperthermia, symptoms of intoxication. We identified the pancreatic enzymes rise in blood and urine in more than 3 times. Sonography also detected changes typical for acute edematous pancreatitis (Fig. 10A, B).

Typical for acute pancreatitis changes were identified upon CT (Fig. 11A, B).

Treatment was successful in all cases. It included infusion therapy, octreotide, proton pump inhibitors (predominantly, pantoprazole parenterally), antibacterial agents, if necessary, analgesics and antispasmodics, enteric sorbents. Treatment was carried out in conjunction with the psychotherapist, who prescribed psychotropic drugs, depending on the peculiarities of each patient.

Although the clinic and course of stress pancreatitis in our patients did not have clear features, we consider it necessary to draw the attention of physicians to the possibility of such a variant of pancreatitis in our difficult times.

We finish the article with the words of Leonid Yakubovich: "... only work relieves stress. If you have a favorite job, there is no stress". We wrote this article during the difficult political situation in Donbass. Indeed, when we were working on the article, it helped us dive into our favorite work and reduce psychological burden.

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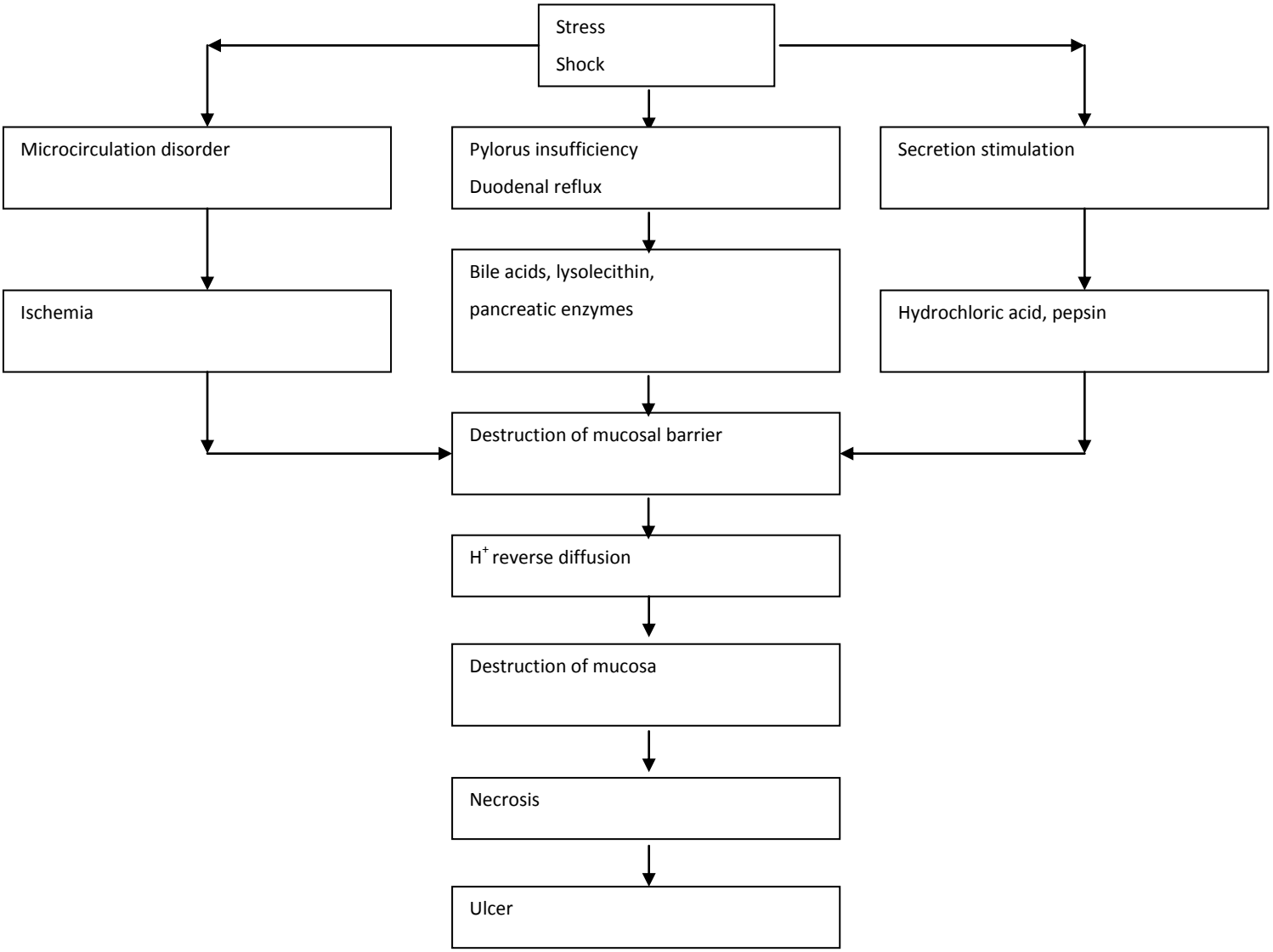
Stress
Shock
Microcirculation disorder
Pylorus insufficiency
Duodenal reflux
Secretion stimulation
Ischemia
Bile acids, lysolecithin,
pancreatic enzymes
Hydrochloric acid, pepsin
Destruction of mucosal barrier
H+ reverse diffusion
Destruction of mucosa
Necrosis
Ulcer

Fig. 1. The pathogenesis of stress gastroduodenal ulcers (by A. A. Ponomarev et al., 2003 [3]).

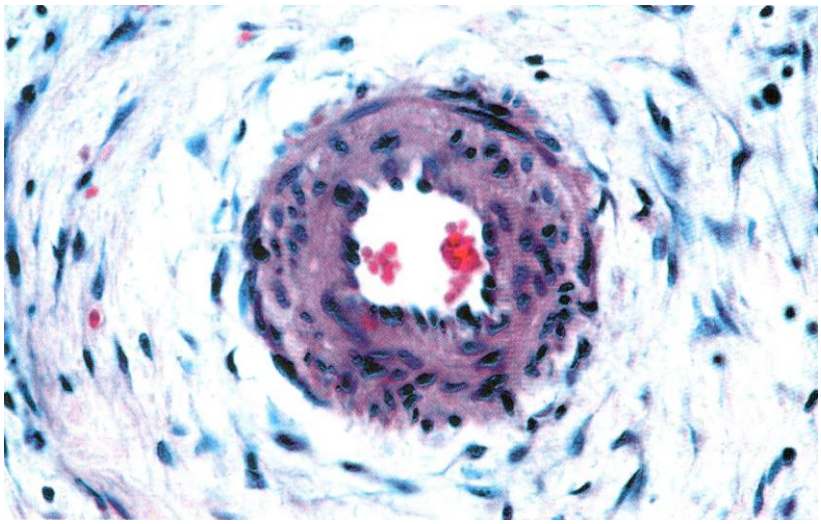

Fig. 2. Spastic state of the small artery (imb. hematoxylin and eosin, enl. × 400) (by L. A. Faustov, 2005 [1]).

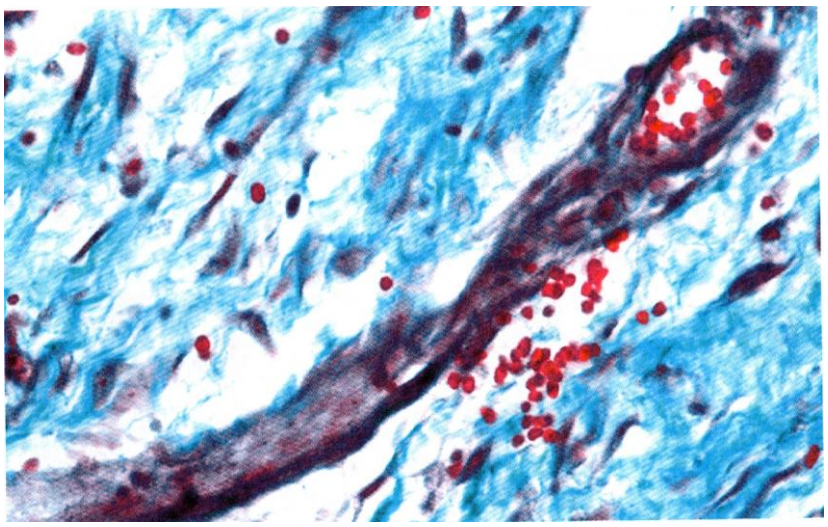

Fig. 3. The splitting and homogenization of the basal membrane in the arteriole (imb. aldehyde-fuchsin, chromotrope, aniline blue, enl. × 400) (by L. A. Faustov, 2005 [1]).

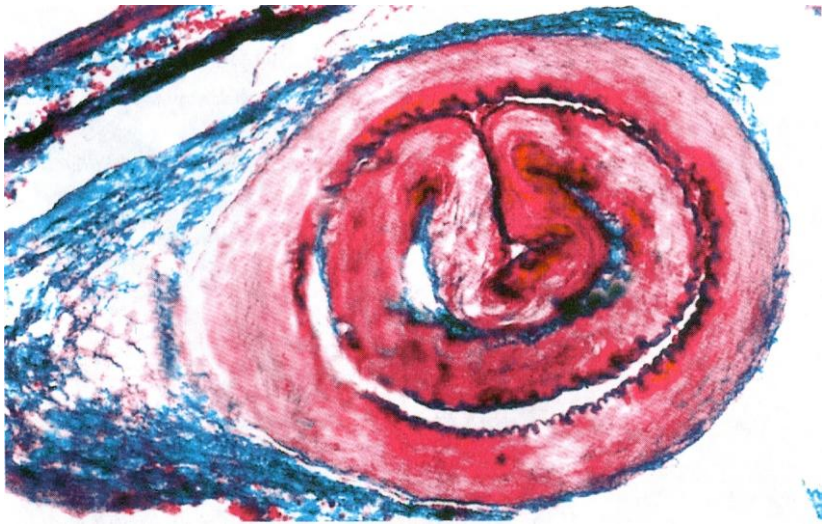

Fig. 4. Invagination of the artery. Internal elastic membrane is located outside in invaginate (imb. aldehyde-fuchsin, chromotrope, aniline blue, enl. × 200) (by L. A. Faustov, 2005 [1]).

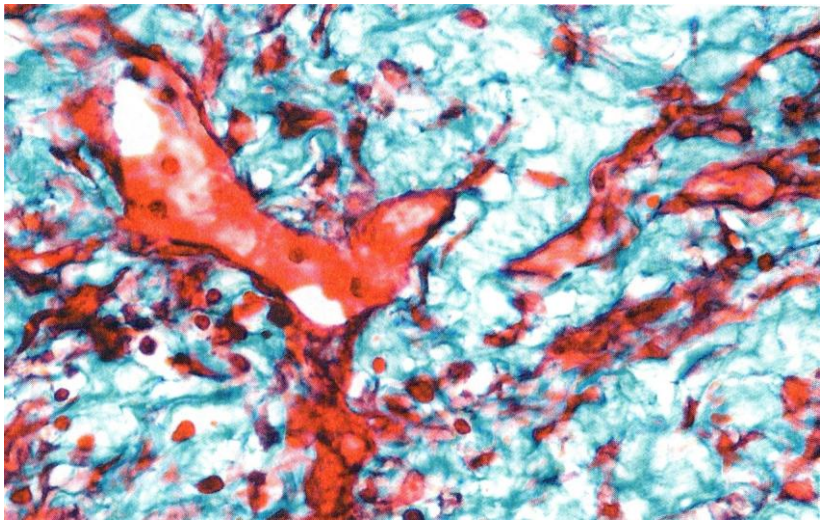

Fig. 5. Alternating spastic and atonic fragments in microvessels (imb. alcian blue, neutral red, enl. × 400) (by L. A. Faustov, 2005 [1]).

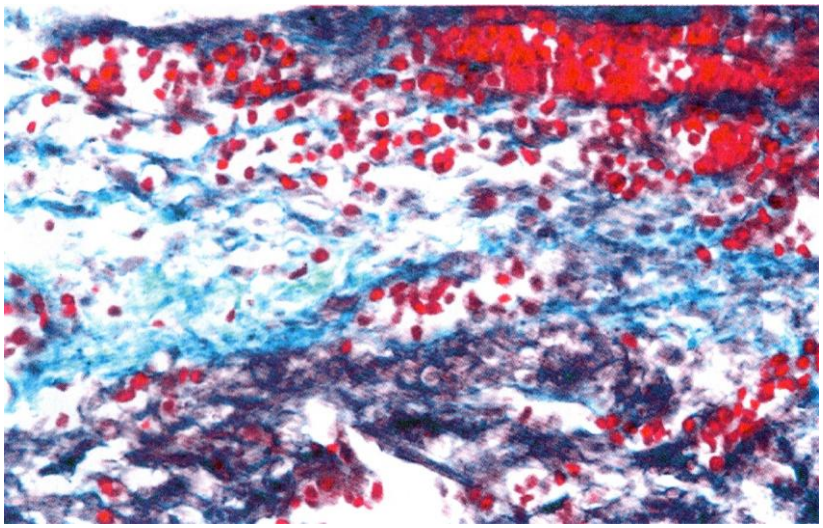

Fig. 6. Haemorrhagic infiltration of paraulcer tissues (imb. aldehyde-fuchsin, chromotrope, aniline blue, enl. × 400) (by L. A. Faustov, 2005 [1]).

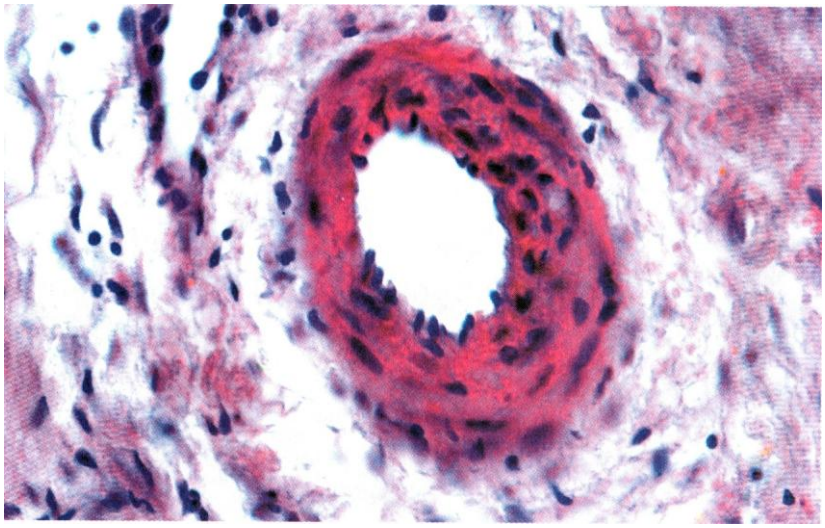

Fig. 7. Spasm of the small arteries of the pancreas (imb. hematoxylin and eosin, enl. × 400) (by L. A. Faustov, 2005 [1]).

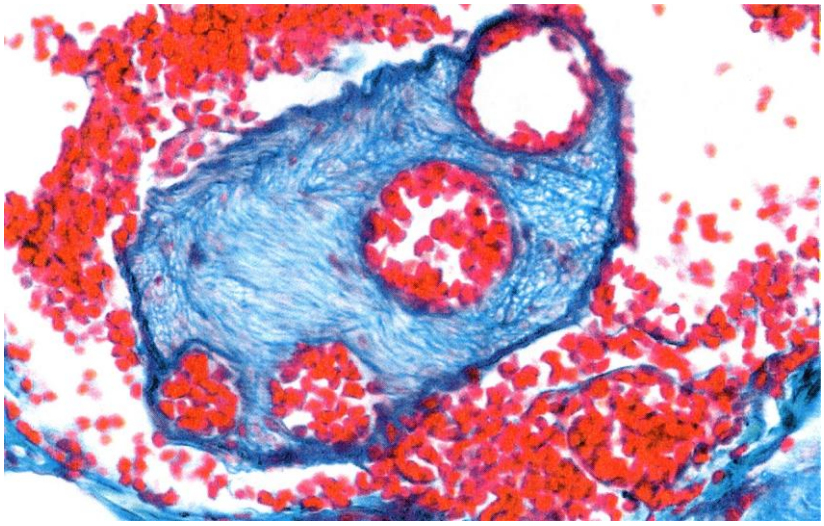

Fig. 8. Sharp congestion of the capillaries in the nerve trunk of parapancreatic tissue (imb. aldehyde-fuchsin, chromotrope, aniline blue, enl. × 400) (by L. A. Faustov, 2005 [1]).

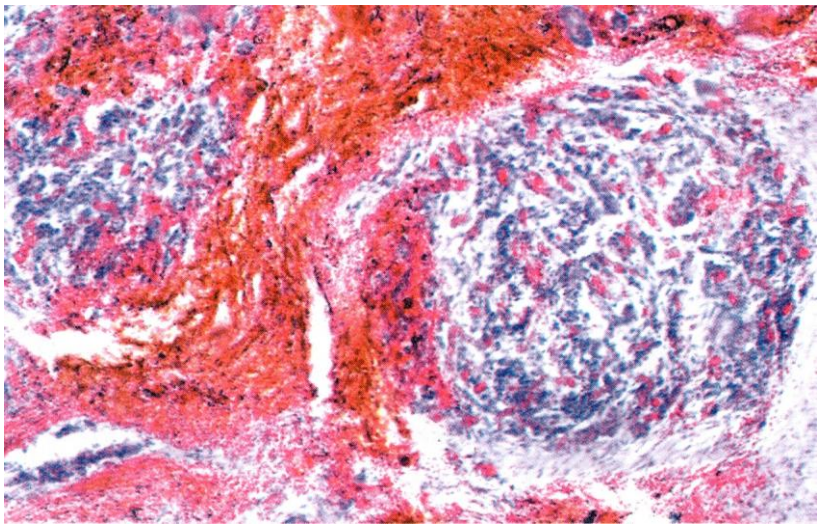

Fig. 9. Haemorrhagic infiltration of pancreatic tissue (imb. hematoxylin and eosin, enl. × 100) (by L. A. Faustov, 2005 [1]).

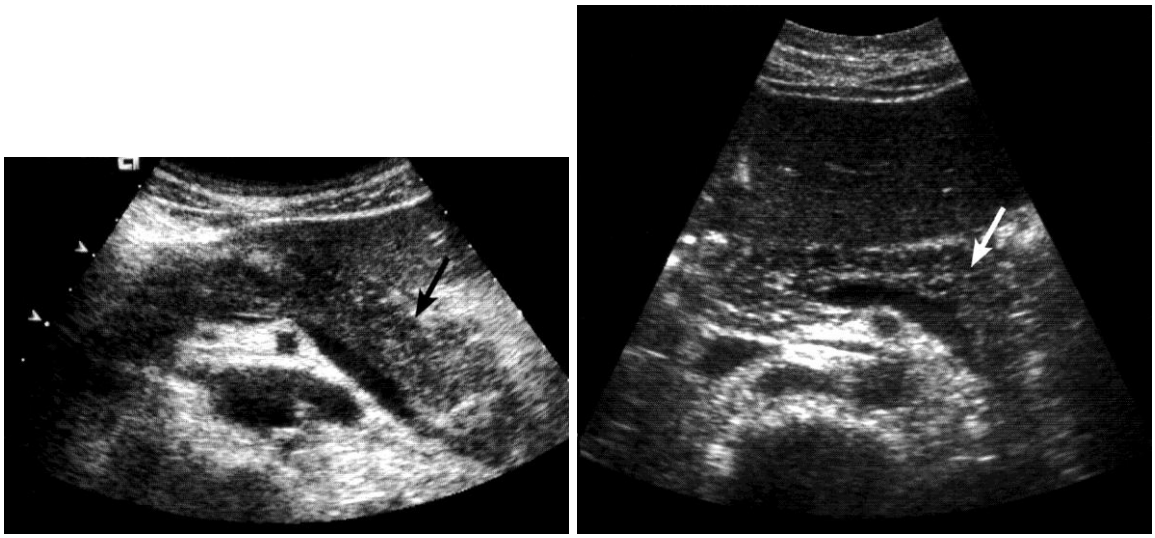

Fig. 10A, B. Acute edematous pancreatitis: hypoechoic enlarged pancreas is detected on transverse scans (by arrow).

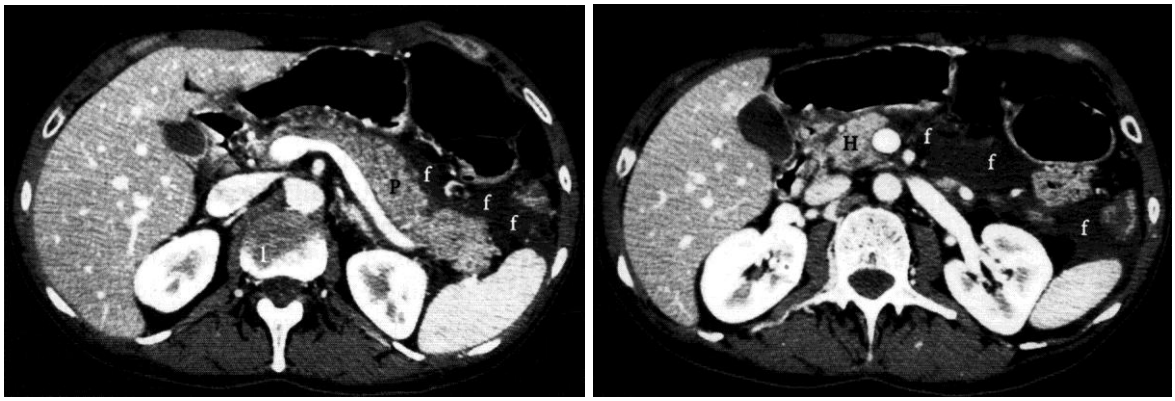

Fig. 11. CT of a patient with acute stress pancreatitis.

A. In the arterial phase diffuse enlargement of the pancreas with a homogeneous density reduction of up to 92 Hounsfield units is revealed due to the

swelling of parenchyma. Normal structure of the pancreas is preserved (P). Fluid around the body and tail of the pancreas is determined (f).

B. Similar change of the pancreatic head (H) and fluid accumulation (f) in the left front pararenal space is determined on a more caudal image.

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Article analyzes the pathogenesis of stress pancreatitis and stress gastroduodenal ulcers. Clinical picture, course and treatment of acute stress pancreatitis are briefly described. The authors represented examples of histological changes of the pancreas and gastric mucosa, the results of sonography and computed tomography.