Hypertriglyceridemia - Rare Etiological Condition of Necrotic-Hemorrhagic Pancreatitis

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Hypertriglyceridemia is the most common etiology after alcohol consumption and gallstones. The risk of acute pancreatitis occurrence increases at a triglyceride level of over 1000 mg / dL. We hereby present a rare pathological situation of a young patient of 24 years old with acute, necrotic-hemorrhagic pancreatitis induced by hypertriglyceridemia. The emphasis is on the importance of considering the rare etiologies of acute pancreatitis and on the application of more effective treatments.

Keywords: hypertriglyceridemia, severe pancreatitis

Acute pancreatitis induced by hypertriglyceridemia was found in 1.3-3.8% of patients [1]. Hypertriglyceridemia is the most common etiology after alcohol consumption and gallstones [1,2]. The risk of acute pancreatitis is of approximately 5% at triglyceride levels above 1000 mg / dL and of 10% -20% at values higher than 2000 mg / dL [4]. The pathophysiological mechanisms include hydrolysis of triglycerides by pancreatic lipase, excessive formation of free fatty acids, secondary inflammatory changes and capillary lesions [3-7]. The hyper-viscosity of the blood with tissue's ischemia plays an additional role.

We present a rare pathological situation of a young patient of 24 years old with acute necrotic-hemorrhagic pancreatitis induced by hypertriglyceridemia.

Experimental part

Material and method

A 24-year-old patient with newly discovered insulin-requiring type II diabetes is transferred from the Internal Medicine Department to the Surgery Clinic IIof Sf. Apostol Andrei Emergency Hospital, Galati, with the diagnosis of acute pancreatitis.

The paraclinical laboratory examinations had highlighted the following pathological changes: WBC 20960/mmc, amylasemia 878 U/L, glycaemia 235 mg/dL, TGP 764 U/L, TGO 242 U/L, urea 93 mg/dL, creatinine 1.41 mg/dL, triglycerides 1059 mg/dL, total lipids 880 mg/dL, amylasuria 3986 U/L.

The following were observed at the abdominal ultrasonography: the liver:left lobe = 79 mm, right lobe = 160/151 mm, increased echogenicity, micro-granular structure, steatosis of 3rd degree, VP = 11 mm, CBP = 4 mm free, undiluted CBIH, folded gallbladder with reduced volume, apparently without gravel, invisible pancreas, 105 mm long homogeneous spleen, intraperitoneal free fluid in supra- and inframesocolic average quantity.

The abdominal and pelvic CT with contrast agent had revealed left pleural effusion with a liquid blade with maximum thickness of 26 mm, right pleural effusion with a liquid blade with maximum thickness of 22 mm, pancreas with increased dimensions, low contrast agent capture, infiltrated aspect of peripancreatic tissues, inaccurate fluid clusters delimited in the bilateral peritoneal and retroperitoneal cavity, infiltrated aspect of bilateral lumbar muscle planes.

Results and discussions

Surgery was performed, with the diagnosis of severe acute pancreatitis induced by hypertriglyceridemia and necrotic-hemorrhagic pancreatitis, enzyme peritonitis, hepatomegaly, chronic alithiasis cholecystitis has been reported. Capsulotomy, necrectomy, lavage, multiple peritoneal drainage were performed.

The post surgery evolution was unfavorable, ending with the patient's death 3 days after surgery.

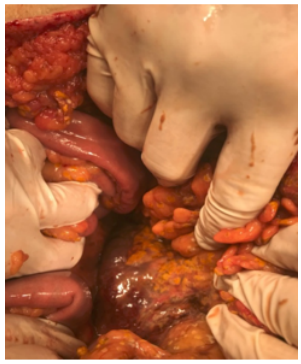

Fig.1. Cytosteatonecrosis - intra-surgical aspect

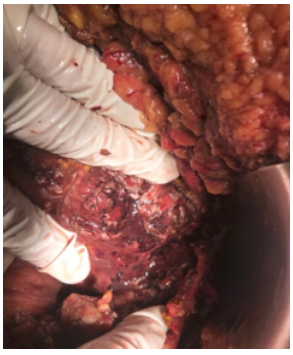

Fig.2.Pancreatic necrosis - intra-surgical aspect

The microscopic examination described the epiploic fragment (6320 / 29.11.2017) with necrosis areas, where the adipocytes lose nuclei, retaining only the contour of the cell membranes, the cytoplasm being replaced by a granular, eosinophilic, opaque sediment, with areas more basophilic, occurred through the fat's hydrolysis followed by the precipitation of calcium salts. Moderate acute granulocytic inflammatory infiltration and diffuse hematic infiltration were also found.

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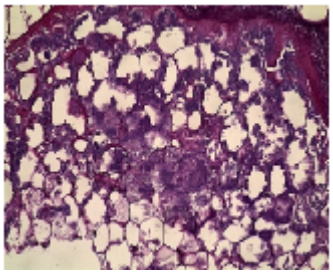

Fig. 3.Cytosteatonecrosisx 10 -bigepiploon

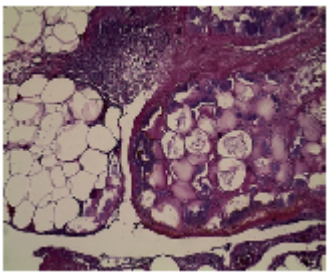

Fig. 4.Cytosteatonecrosis x10 (2) - big epiploon

The histopathological aspects correspond to the changes from cytosteatonecrosis.

The extracted pancreatic tissue (6321 / 29.11.2017) was composed of completely necrotic tissue fragments with rich acute granulocyte infiltration and fibrin deposits.

The pathogenic mechanism of how HTG can cause acute pancreatitis remains unclear and controversial. Several theories have been suggested, most frequently referring to the excess of triglycerides that are hydrolyzed by pancreatic lipase, and free fatty acids (FFAs) are formed in high concentrations [8- 12].

FFA overloads the ability of attachment of albumin and of the self-aggregate to the micellar structures with detergent properties [13- 16]. Thus, acinar cell lesion and pancreatic capillary lesion are formed. The resulting ischemia creates an acidic environment that further enhances the FFA toxicity [17- 19]. Another underlying physiopathologic principle may be hyperviscosity due to high levels of chylomicrons. The hyperviscosity due to hyperchylomicronemia is believed to evolve to ischemia and acidosis in the pancreatic capillaries. In addition, there is evidence that FFA affects the endothelium and thus acute pancreatitis may be associated with microtrombosis and subsequent ischemia [20, 21]. The stress of the endoplasmic reticulum would also be involved in the HTGP mechanism [22]. There are also reports on specific genes associated with HTGP. A Chinese study reported a CFTR mutation and a TNF promoter polymorphism, which were independent risk factors for HTGP. CFTR gene's mutation rates in HTG with and without acute pancreatitis were 26.1% (12 of 46) and 1.3% (one out of 80) [22], respectively. Another recent Spanish study finds that the e4 allele of the APOE gene is more common in patients with HTGP [23, 24]. It is likely that all the theories described contribute to the development of HTGP to a certain degree. However, additional research efforts in this area will be needed to shed more light on HTGP pathophysiology.

Hypertriglyceridemia is also an independent risk factor for acute renal lesions [24]. This hypothesis refers to the action of pancreatic enzymes on the triglycerides accumulated in the kidneys, which may explain acute renal lesions in the early stage of the disease [25-33].

Interestingly, beyond the apparent significance of a triglyceride threshold for the initialization of acute pancreatitis, its stringency does not seem to correlate directly with triglyceride levels. Although the patient is under treatment, the clinician should investigate the etiology of hypertriglyceridemia by verifying the effects of the patient's

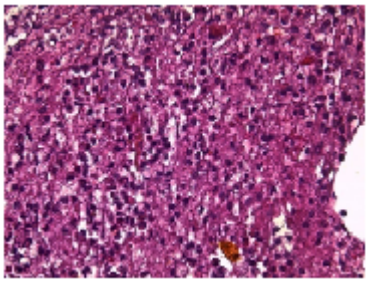

Fig. 5.Pancreatic necrosis x20

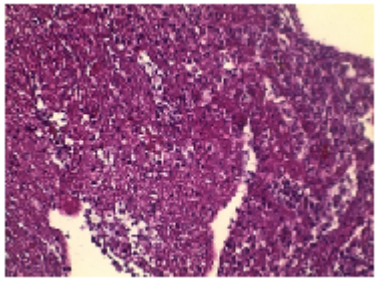

Fig. 6.Pancreatic necrosis x40

chronic medication, of his/her medical history or of his/her family members in his/her search for familial hyperlipidemias.

Conclusions

Acute pancreatitis induced by hypertriglyceridemia remains still a *Pandora's box* with surprising types of patients.

A severe, fatal acute pancreatitis may complicate an asymptomatic, undiagnosed hypertriglyceridemia.

Various diagnostic methods can be very useful in determining the cause of the pancreatic inflammation, but nevertheless, the most valuable approach remains a detailed anamnesis and the clinical examination.

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