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# THE EFFECT OF THE LEVEL OF LIVER FAT METABOLISM ON THE SEVERITY OF ACUTE PANCREATITIS.

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**Abstract:** The review article provides information on the pathogenesis and mechanisms of development of acute pancreatitis, provides information on the influence of lipid metabolism and the level of fatty liver on the severity of acute pancreatitis.

**Keywords:** acute pancreatitis; organ failure, fatty liver, non-alcoholic fatty liver disease.

Acute pancreatitis (OP) is an inflammatory disease of the pancreas, affecting all ages, with annual incidence 10-50 cases per 100,000 people [1]. It is slightly open and is resolved without serious complications in 80% of patients, but has complications and significant mortality up to 20% of patients. So far, many causes of acute pancreatitis have been identified, but the pathogenetic theories remain contradictory. In 1856, Claude Bernard suggested that reflux of the bile to the common pancreatic duct could cause acute pancreatitis. Several subsequent studies led to theories that fueled controversy until 1901 [2] when Eugene Opie suggested that the migration of bile stones into the common bile duct is the main cause of acute pancreatitis. Many other causes of pancreatitis have been discovered since then. However, the pathogenicity of acute pancreatitis remains controversial.

The most common cause of acute pancreatitis is the entrance of a gallstone into the distal common bile duct. Most researchers recognize that the main factors of acute biliary pancreatitis are hyperstimulation of the pancreas and obstruction of bile ducts, which increase the pressure in the pancreatic duct and active reflux tripsin subsequent to unregulated activation of tripsin in the acinar cells of the pancreas. Activation of the enzyme in the pancreas leads to auto-redigestion of the gland and local inflammation. Acute pancreatitis occurs when the intracellular protective mechanisms preventing tripsinogen activation or reducing tripsin activity are suppressed. However, little is known about other causes of acute pancreatitis. We have suggested that acute biliary pancreatitis and other causes of acute pancreatitis have common pathogenesis [3].

Acute pancreatitis (OP) is a high-volume acute inflammation and is often accompanied by multiple organ dysfunction syndrome (MOD). The liver, one of the most vulnerable SPB organs in OP, is the main organ involved in the development of the disease and closely correlates with the occurrence of OD[4].

Almost all pancreatic mediators released from the pancreas into the bloodstream can pass through the liver before being reared in the systemic circulation. Once pancreatic mediators reach the liver, they strongly activate Kupfer cells, resident macrophages, greatly increasing the release of cytokines into the bloodstream and thus contributing to systemic manifestations of acute pancreatitis [5].

According to the report, the mortality of patients with acute pancreatic disease associated with liver failure is 83% [6]. So it is crucial to understand the relationship between liver damage and OP.

We searched PubMed, EMBASE and the Cochrane Library to identify all acceptable studies. There are no results yet of the study on the influence of fatty liver tissue on severity and outcomes of acute pancreatitis. We have conducted a meta-analysis to investigate this issue.

Fatty liver deficiency is often found in patients with acute pancreatic disease, as both diseases have common factors contributing to the development of the disease such as obesity, type 2 diabetes mellitus, alcohol abuse and hyperlipidemia [7].

Retrospective analyses determined that compared to patients without a fatty liver, pancreatitis severity and serum C-reactive protein levels in the blood were higher in patients with a fatty liver. The prevalence of local complications, persistent organ failure and mortality were also higher in patients with fatty liver degeneration. Even after adjustment for age, sex, body mass index and pancreatitis etiology, liver fat deficiency was reliably associated with moderately severe or severe acute pancreatitis. Patients with OP who had liver fat deficiency, clinical manifestations were more severe than patients without liver fat deficiency, there was a higher number of local complications, persistent organ failure and mortality. Consequently, it can be assumed that liver fat deficiency is a significant risk factor for severe disease even after correction of numerous factors including age, BMI and the cause of acute pancreatitis.

The data obtained suggest that liver fat deficiency may itself be an independent risk factor for severe acute pancreatitis. Fatty liver changes in early stages of CT can be used as early predictors and can be usefully included in future prognostic models for OT [8].

Finally, fat deficiency in the liver led to a higher frequency of local complications, persistent organ failure and death from acute pancreatitis. The study of the level of distrofia and liver condition can play a prognostic role in acute pancreatitis. The fatty tissue of the liver can be included in future prognostic cortical models.

This simple grading system is a potentially valuable predictor that should be considered for use in future prognostic systems of acute pancreatitis severity

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