[Research Article]

COMPLEX MORPHOLOGICAL CHARACTERISTICS OF ALTERED PANCREATIC SIGNS IN CHOLEDOCHOLITHIASIS WITH OBSTRUCTIVE JAUNDICE

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Abstract: In obstructive jaundice caused by choledocholithiasis, the pancreas undergoes significant structural changes due to biliary hypertension, retrograde bile reflux, and activation of proteolytic enzymes. The study revealed pronounced destructive and inflammatory changes primarily in the exocrine portion of the gland, including acinar destruction, edema, plasmorrhea, and vascular stasis. Morphometric analysis revealed a significant decrease in the area of acinar structures, correlating with the duration of biliary stasis. It was established that timely drainage of the common bile duct contributes to a significant reduction in the severity of morphological abnormalities.

Keywords: choledocholithiasis; obstructive jaundice; pancreas; biliary pancreatitis; morphology; morphometry; acinar destruction; biliary hypertension; common bile duct drainage; microcirculation.

In obstructive jaundice resulting from choledocholithiasis, the pancreas is inevitably involved in the pathological process, being part of the functional unity of the biliary-pancreatic system. The anatomical and physiological proximity of the common bile duct and the main pancreatic duct, as well as their morphological relationships, make pancreatic tissue sensitive to obstruction of the biliary tree. Mechanical obstruction of the common bile duct leads to the development of a complex of pathogenetic disorders affecting both the exocrine and vascular components of the gland.

Retrograde bile reflux activates proteolytic enzymes in the duct lumen and acinar structures, triggering cellular autolysis. Concurrently, edema develops, interstitial infiltration by leukocytes occurs, and areas of coagulative and liquefactive necrosis develop. These morphological manifestations were documented in detail during microscopic analysis in the work of Mazur A.I. et al., which emphasizes the increasing destruction of acinar elements depending on the degree and duration of biliary hypertension [1, p. 65].

Macroscopically, the pancreas under such conditions is characterized by an increase in size, pastosity, and discoloration, indicating inflammatory and vascular changes. A section often reveals edematous tissue with foci of necrosis, predominantly in the head and body area. Against the background of persistent biliary tract obstruction, areas of fatty necrosis and hemorrhagic inclusions may form, reflecting the severity of vascular disorders.

Microscopically, a marked disruption of the structural organization of the exocrine pancreas is revealed. Acinar elements undergo vacuolation and destruction, with manifestations of karyopyknosis and karyorrhexis. The interstitial tissue is infiltrated with lymphocytes, macrophages, and neutrophils,

indicating the development of an active inflammatory process. The vascular component deserves special attention. Morphological examination revealed signs of plasmorrhea, perivascular edema, stasis, and microthrombosis, confirming the ischemic origin of some of the changes [1, p. 66].

A characteristic of the pathological process is the relative preservation of the islet apparatus, even with severe destruction of the exocrine tissue. This may indicate a lower sensitivity of the endocrine component to biliary aggression in the early stages, as well as the possibility of functional compensation in the case of partial damage to the gland. However, with prolonged obstruction and the development of pancreatic necrosis, the islets of Langerhans also become involved, which can lead to impaired glycemic control. Morphometric studies comparing diseased and intact tissue revealed a significant decrease in the average area of acinar elements, a reduction in their distribution density, and a change in the ratio of interlobular and parenchymal components. These changes are particularly pronounced in the absence of common bile duct drainage, as highlighted in the clinical study by E.I. Vlasov, which demonstrated an increased incidence of biliary pancreatitis in patients without surgical decompression [2, p. 2].

Table of morphological changes in the pancreas in choledocholithiasis with obstructive jaundice

Based on data from sources, particularly Vlasov's clinical article and morphological studies, the following summary table can be compiled, reflecting the main morphological findings:

Parameter	**Normal (control)**	**In obstructive jaundice (associated with choledocholithiasis)**	**Note**
Acinar structures	Dense, clear, uniform	Destruction, vacuolization, karyopyknosis	Signs of autolysis and enzymatic aggression
Interstitial tissue	Weakly defined	Edema, lymphocytic and neutrophilic infiltration	Especially pronounced with prolonged biliary obstruction
Ducts	Passable, with smooth epithelium	Destruction of the epithelium, deposition of bilirubin pigments	Retrograde bile reflux
Vessels	Full capillaries, moderate blood filling	Stasis, perivascular edema, plasmorrhea	Microcirculatory disorders and ischemia
Endocrine apparatus (islets of Langerhans)	Intact, unchanged	Relatively preserved	Not primarily affected
Acinar morphometry (area)	100%	Decrease to 55–60%	Data are conditional, according to the authors' assessment

The impact of obstructive jaundice on the morphological state of the pancreas is confirmed not only by microscopy data but also by clinical observations. Patients who have experienced episodes of obstructive jaundice without timely drainage significantly more often exhibit signs of a pronounced inflammatory response, a systemic reaction, and, in some cases, multiple organ failure. These clinical features correlate with the morphological picture: autopsy analysis revealed foci of coagulative necrosis, periductal fibrosis, and destructive changes in the capillary network.

Clinical and morphological comparison allowed us to identify the most significant morphological predictors of severe pancreatitis: massive interstitial edema, the presence of plasmarrhea, vascular obliteration, and severe acinar destruction. In cases where early drainage of the common bile duct was performed, the intensity of morphological changes was significantly lower, allowing these signs to be considered objective criteria for the severity of damage and the effectiveness of therapy [2, p. 3].

Thus, the comprehensive morphological characteristics of pancreatic changes in obstructive jaundice caused by choledocholithiasis indicate a multifactorial damaging effect. The main pathogenetic mechanisms of damage are mechanical pressure, bile reflux, protease activation, vascular compression, and secondary ischemia. Each of these factors has its own morphological manifestations, which can be

objectified using light microscopy and morphometric analysis.

The practical significance of the obtained data lies in the possibility of predicting the degree of pancreatic damage based on histomorphological criteria, as well as in justifying the need for early surgical correction to minimize destructive changes. This is confirmed by the fact that timely drainage of the common bile duct reduces the risk of biliary pancreatitis by 2.2 times-from 8.7% to 3.9%, according to observations by E.I. Vlasov [2, p. 4].

The diagnostic value of morphological criteria should also be noted. Identification of specific microscopic signs (acinar destruction, edema, necrosis, vascular pathology) allows for retrospective verification of the biliary etiology of pancreatitis. This is important not only for clarifying the diagnosis but also for developing effective patient management strategies in the postoperative period and during recurrent episodes.

The pattern of changes identified confirms the relevance of morphological analysis as part of a comprehensive assessment of patients with biliary pathology. With the increasing incidence of choledocholithiasis and the tendency for patients to be hospitalized late, especially in those over 60 years of age, morphological verification is becoming not only a scientific but also a clinical necessity. The data obtained can also be used for risk stratification in surgical practice and assessing the need for preventive interventions.

Consequently, the pancreas in obstructive jaundice caused by choledocholithiasis undergoes a wide range of structural changes, the nature of which is determined by the severity, duration, and nature of biliary obstruction. Morphological assessment of these changes provides valuable information both for the pathogenetic understanding of the process and for practical clinical decisions. An integrated approach using morphometry, histological analysis, and clinical and morphological comparison allows for increased diagnostic accuracy, improved risk stratification, and optimized treatment strategies for pathologies of the biliary-pancreatic zone.

An analysis of clinical and morphological data demonstrates a close pathogenetic relationship between obstructive jaundice and pancreatic damage. Biliary hypertension causes pronounced inflammatory and destructive changes in the organ, primarily in the exocrine component. Acinar destruction, interstitial edema, and microcirculatory disturbances are universal manifestations of this pathology. Their severity is determined by both the duration of obstruction and the timeliness of surgical intervention. The data obtained confirm the clinical utility of early common bile duct drainage as a method for preventing biliary pancreatitis and as a factor limiting the severity of morphological disturbances.

References:

1. Мазур А. И., Азнабаев М. Т., Батыров С. А. Особенности морфологии поджелудочной железы при обтурационной желтухе у взрослых // Вестник морфологии. - 2023. - №2. - С. 62–69.
2. Власов Е. И. Предикторы билиарного панкреатита и выбор показаний для дренирования холедоха при остром калькулезном холецистите // Вестник экстренной хирургии. - 2022. - №4. - С. 1–6.
3. Дарвин В.В., Онищенко С.В., Логинов Е.В., Кабанов А.А. Тяжелый острый панкреатит: факторы риска неблагоприятного исхода и возможности их устранения. *Анналы хирургической гепатологии*, 2018. 23(2). С. 76–83. https://doi.org/10.16931/1995-5464.2018276-83
4. Гальперин Э.И., Дюжева Т.Г., Шефер А.В., Котовский А.Е., Семененко И.А., Мудряк Д.Л. Ранние вмешательства при повреждении протока поджелудочной железы у больных острым панкреатитом. *Анналы хирургической гепатологии*, 2021. № 26(2). С. 25–31. https://doi. org/10.16931/1995-5464.2021-2-25-31
5. Шабунин А.В., Бедин В.В., Тавобилов М.М., Шиков Д.В., Колотильщиков А.А., Маер Р.Ю. Эндоскопическое стентирование протока поджелудочной железы в лечении больных панкреонекрозом. *Анналы хирургической гепатологии*, 2021. № 26(2)