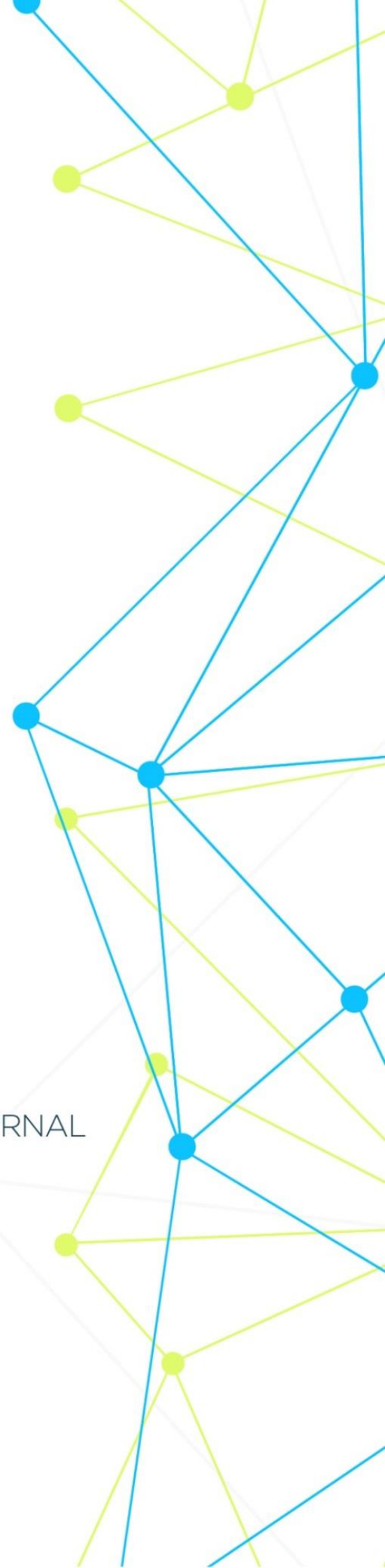


INTERNATIONAL MEDICAL SCIENTIFIC JOURNAL

ART OF MEDICINE



Founder and Publisher **North American Academic Publishing Platforms**

Internet address: <http://artofmedicineimsj.us>

E-mail: info@artofmedicineimsj.us

11931 Barlow Pl Philadelphia, PA 19116, USA +1 (929) 266-0862

Chief Editor

Dr. Pascual Izquierdo-Egea

Prof. Dr. Francesco Albano

Dr. Catherine J. Andersen

Prof. Dr. Sandro Ardizzone

Dr. Dmitriy Atochin

Prof. Dr. Antonio Aversa

Prof. Dr. Tamam Bakchoul

Prof. Dr. Pierre-Grégoire Guinot

Prof. Dr. Rainer Haak

Prof. Henner Hanssen

Roy G. Smith

Department of Molecular and Cellular Biology/Department of Medicine

Baylor College of Medicine

Houston, TX 77030, USA

Kalpesh Patel, MD

The Sydney Kimmel Comprehensive Cancer Center

Johns Hopkins Medical Institutions

Baltimore, MD, 21231, USA

Roy G. Smith

Department of Molecular and Cellular Biology/Department of Medicine

Baylor College of Medicine

Houston, TX 77030, USA

Khamdamov Bakhtiyor Bukhara State Medical Institute

Khamdamova Mukhayokhon Bukhara State Medical Institute

Available at <https://www.bookwire.com/>

ISBN: [978-0-578-26510-0](https://www.isbn-international.org/product/9780578265100)

MORPHOLOGICAL CHANGES IN THE PANCREAS IN CHOLEDOCHOLITIASIS

Rakhimov I.R.

Babajanov K.B.

Urganch Branch of Tashkent Medical Academy

Abstract: Choledocholithiasis is a stone disease of the bile ducts of the liver, which continues with a chronic course of the process. 10% of the world's population is infected with this disease. This disease occurs in the ratio of 1:8 between men and women, and in terms of gender, women are more common. In choledocholithiasis, the main affected organs are the liver, gall bladder and pancreas. Choledynamic disorders in the biliary tract occur as a result of acute or chronic obturation of the ducts draining from the pancreas to the duodenum, resulting in the accumulation of proteolytic enzymes in the pancreatic ducts and in the pancreatic duct. As a result, depending on the dynamics of the process in the pancreas, it is manifested by the development of pancreatitis in the form of acute or chronic inflammation.

Keywords: morphology, pancreas, acute and chronic pancreatitis, choledynamics, cholelithiasis, gallstone disease.

The relevance of the topic: Gallstone disease is characterized by the fact that it occurs mostly in women in 10% of the world population. The main role in the mechanism of the development of gallstone disease is explained by the constitutional structure of the female body, metabolic disorders, exocrine disorders, and disorders of immunopathological processes. At the same time, indirect or predisposing factors that lead to the development of gallstone diseases also play an important role. In particular, obesity, sedentary lifestyle, sharp consumption of semi-finished foods, smoking, chronic stress and daily partial meals disrupt the smooth functioning of the gastrointestinal tract and liver. In very rare cases, it develops after secondary complications after the development anomalies of the stomach, intestinal tract and biliary tract, pregnancy, major reconstructive surgery procedures in the abdomen. This type of predisposition accounts for 1.3-5.2% of gallstones caused by congenital or secondary factors.

Object and subject of the research: As an object of the research, the biliary tract and pancreas of patients who died with the diagnosis of acute pancreonecrosis in a total of 116 cases at the Bureau of Pathological Anatomy Expertise of Khorezm Region and the Republican Center of Pathological Anatomy are considered. Morphological and morphometric changes of the obtained pancreas were analyzed.

The obtained results: the following changes were found in the morphological changes of the pancreas during the autopsy of patients who died of pancreonecrosis. In particular, acute blockage of stones with a diameter of 0.3-0.5 cm in the interconnected branches of the pancreas and bile ducts, spot hemorrhages on the internal surfaces of the bile ducts with serous parade in these branches, and the presence of erosive damaged foci on the surface were found. In the acute period of

most choledocholithiasis, i.e. in 6-12 hours, stones stuck in the mucous membrane of the common bile duct can cause compression of the mucous membrane surface and the appearance of acute pailitis. In most cases, these changes lead to retention of pancreatic secretions. In most acute processes, this preserved secretion is characterized by the development of various forms of pancreatic necrosis due to the acceleration of autolysis and autophagy processes in the acinar glands of the pancreas.

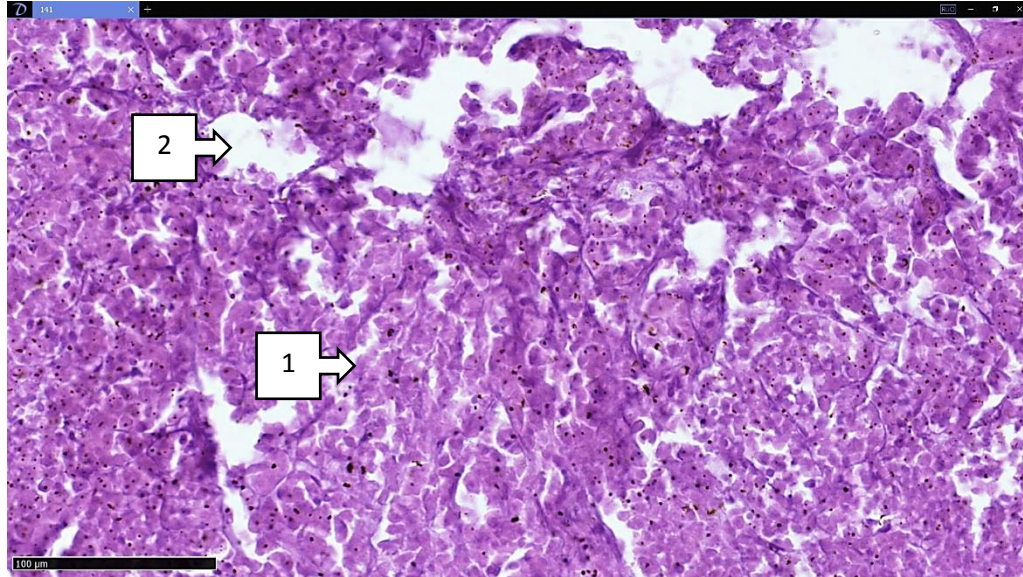


Figure 1. Pancreas. Acute period of common gallstone disease. After 48 hours. Foci of necrobiosis and necrosis are detected in the epithelia of most acinus glands(1). In the focus, detritus is autolyzed and consists of various cellular and interstitial components (2). Paint G.E. The size is 10x20.

It was found that the pancreas was macroscopically brownish-red in color, enlarged in size, and there were foci of hematomalacia with severe autolysis around the common gland ducts. In our microscopic examinations, a sharp reduction of gland secretions in the alveoli of exocrine glands, and dystrophic and necrotic cells in the epithelia of the gland ducts were revealed. In particular, sharp changes are mainly observed around the small glandular structures around the collecting ducts, the interstitial tissue and necrotic gland epithelia, as well as foci of diapedesis hemorrhage in the dilated venous vessels in these areas.

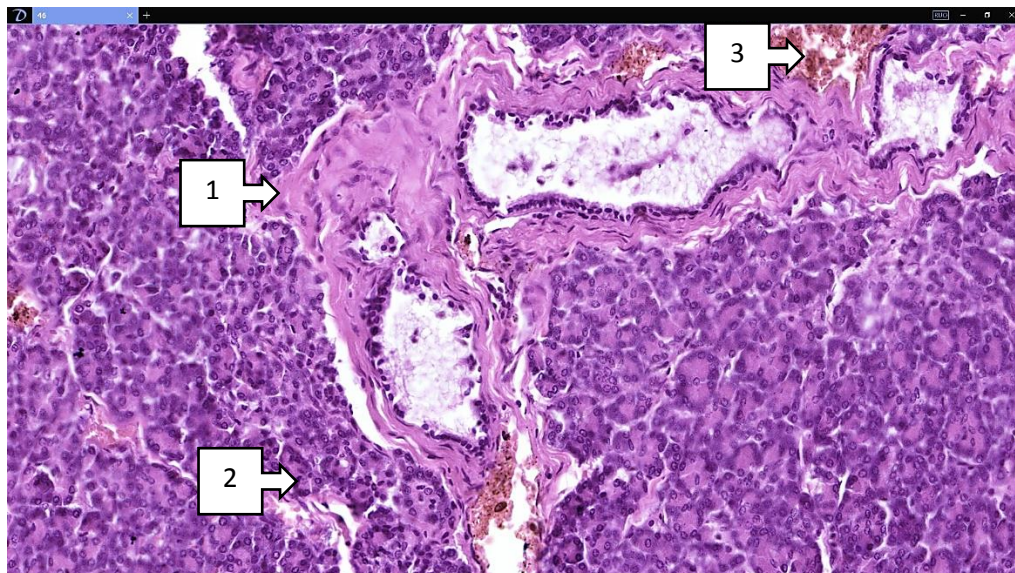


Figure 2. Pancreas. The patient is a 49-year-old woman. Suffered from choledocholithiasis. Sclerosis and hyalinosis foci are detected around the common bile duct (1), hyperplasia foci are detected in the epithelia forming the cavity of the gland duct (2), venous blood vessel dilation and diapedesis hemorrhage foci are detected around the common gland duct (3). Paint G.E. The size is 20x10.

Since gallstone disease is a chronic process, it is determined that atrophic and sclerotic changes have developed in the lobular structure of the pancreas. This change, in turn, led to the chronic reduction of proleptic enzymes in the exocrine structures of the pancreas and the development of atrophic and metaplastic changes in the epithelia of the gland. Against the general background, changes in the histioarchitectonics of the pancreas in the form of reparative regeneration were characterized by the appearance of deformations as a result of the sharp formation of coarse fibrous connective tissue in the interglandular barriers.

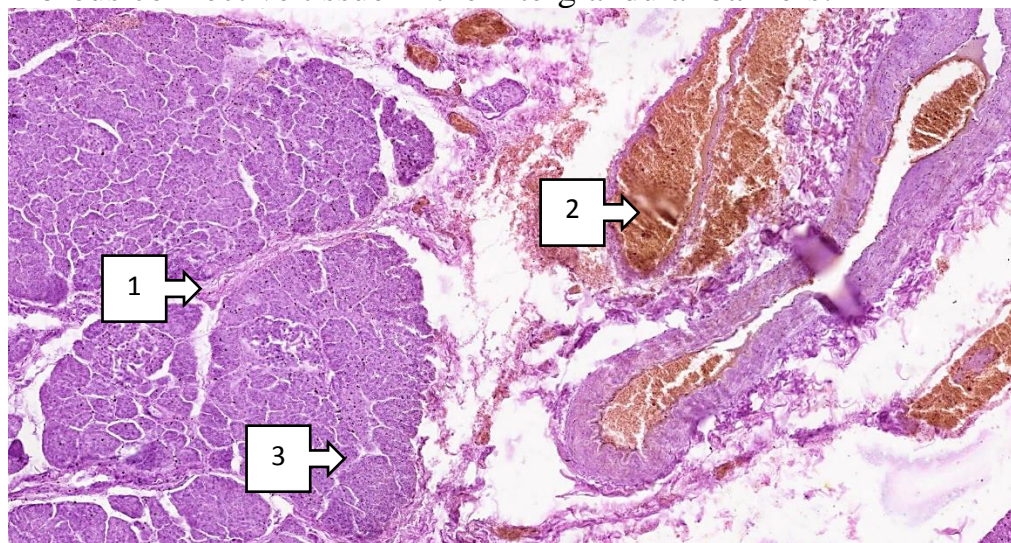


Figure 3. Pancreas. A 48-year-old woman with chronic gallstones. At the perimeter of the lobes, a septa-like connective tissue layer is identified (1). In most venous blood vessels, massive fullness (2), foci of acinar glands undergoing atrophic

and hypertrophic changes in the general background are determined (3). Paint G.E. The size is 20x10.

Dystrophic and necrobiotic changes of acinar gland epithelia in most gallstone diseases depend on the duration of the process, and during most acute attacks, choledynamic disturbances caused by dampness and regurgitation of secretions in the common glandular tract lead to the return of pancreatic fluid in the common glandular tract and from the small branches of the common glandular tract to the entrance gate of the acinar glands. it is manifested by a lot of acute dystrophic and necrotic changes. (See Figure 4). When the process develops acutely, pancreonecrosis develops in approximately 24-48 hours, severe pain in clinical and morphological form, the spread of pain to other organs through a stray nerve leads to reflex narrowing of blood vessels and secondary ischemic changes in the pancreas. Morphologically, it leads to the development of hydropic and yellow dystrophic changes in the epithelia of the acinar glands, and if the process is more continuous, then it continues with necrotic and hemorrhagic hemorrhages in the acinar glands around the common duct (see Fig. 3).

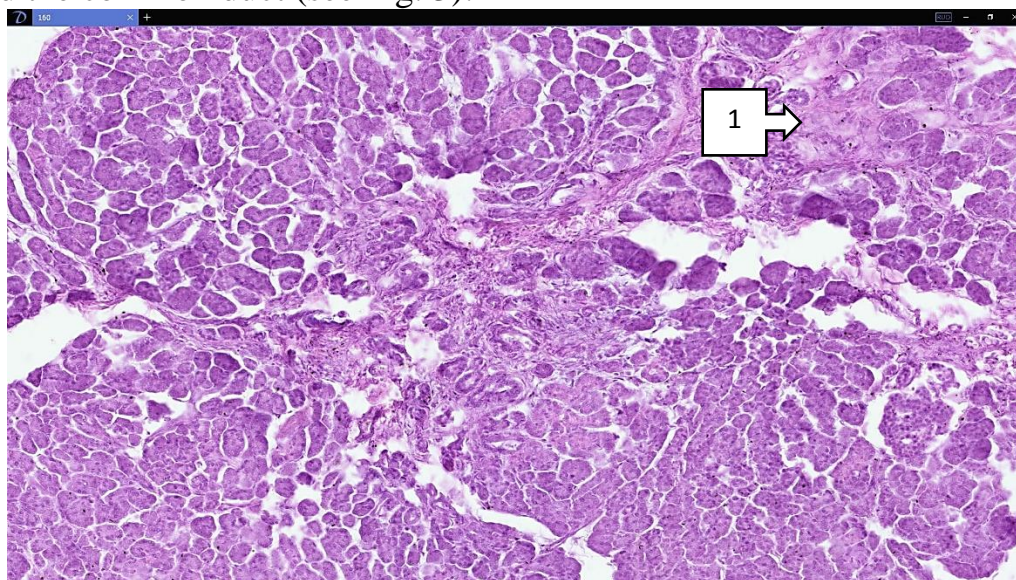


Figure 4. Pancreas. Acinar glands scarred in the center and scarring foci are identified in part of the general glandular tract networks (1). Paint G.E. The size is 20x10.

Summary

In choledocholithiasis, chronically stagnant bile fluid in the bile ducts is a manifestation of choledynamic disorders, which leads to atrophic and sclerotic changes of all glandular structures due to chronic stagnation in the exocrine system of the pancreas. As a result, the dynamic movement of pancreatic secretions is chronically disturbed, and the parenchymatous cells continue with the occurrence of a hypofunctional condition. Clinically morphologically, these changes are manifested in the form of mild dyspepsia.

Used literature

1. Kugaev M. I., Samsonova I. V., Klopova V. A. et al. Relationship between nervous tissue damage and fibrotic changes in the pancreatic head with pain syndrome and quality of life in patients with chronic pancreatitis // *Novosti hir.* 2018. 19, no. 5. pp. 39–45.
2. Maev I. V., Kazyulin A. N., Kucheryavy Yu. A. *Chronic pancreatitis.* Moscow: Medicine, 2015. 504 p.
3. Maev I. V., Kucheryavy Yu. A., Moskaleva A. B. Chronic pancreatitis: myths and reality // *Gastroscopy.* 2016. No. 1. S. 8–10.
4. Maistrenko N. A., Chumasov E. I., Petrova E. S. et al. Features of the pathomorphosis of chronic pancreatitis in the rationale for surgical approaches // *Vestn. hir.* 2018. No. 4. S. 29–39.
5. Minushkin O.N. Chronic pancreatitis, etiology, epidemiology, classification // *Farmateka.* 2017. No. 2. S. 53–57.
6. Nikitin P. N. Morphological changes in the nerve trunks and stroma of the pancreas in chronic pancreatitis with pain syndrome: Abstract of the thesis. dis. ...cand. honey. Sciences. M., 2019. 28 p.
7. Paklina O. V., Chekmareva I. A., Setdikova G. R. et al. Damage to the nerve trunks in chronic pancreatitis // *Ann. hir. hepatol.* 2019. No. 3. P. 95–101.
8. Pryadko A. S., Maystrenko N. A., Romashchenko P. N., Boyko I. Yu. Minimally invasive technologies in the diagnosis and treatment of chronic pancreatitis // *Ann. hir. hepatol.* 2019. No. 2. P. 55–64.