LIVER AND GASTROINTESTINAL SYSTEM IN CIRRHOSIS

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Abstract. *Cirrhosis is a chronic liver disease characterized by fibrosis, regenerative nodules, and impaired liver function. It significantly affects not only the liver but also the entire gastrointestinal system. This review explores the pathological changes in the liver and gastrointestinal system associated with cirrhosis, focusing on the mechanisms underlying these changes and their clinical implications. The liver and the biliary system are a combined, morphofunctional mechanism. Violation of integrity of one of them directly affects the function of the other. As cirrhotic liver disease is very widespread, it is worth to have a correct idea about diagnostics and treatment of this pathology.*

Keywords. *Cirrhosis, liver fibrosis, portal hypertension, esophageal varices, ascites, hepatic encephalopathy, gastrointestinal motility, malnutrition.*

Introduction. Cirrhosis represents the end stage of chronic liver disease, resulting from various causes such as chronic hepatitis, alcohol abuse, and nonalcoholic fatty liver disease. The condition leads to extensive scarring and architectural distortion of the liver, which impacts its function and affects the gastrointestinal system. Understanding these changes is crucial for managing the complications of cirrhosis and improving patient outcomes. The liver and the biliary system are a combined, morphofunctional mechanism. Violation of integrity of one of them directly affects the function of the other. As cirrhotic liver disease is very widespread, it is worth to have a correct idea about diagnostics and treatment of this pathology.

Purpose of the study: To identify the frequency and type of liver, gallbladder and biliary tract damage in cirrhosis. To learn the results of

comparative possibilities of different methods of investigation of the structure of the liver and biliary system.

Pathophysiology of Cirrhosis.

1. Fibrosis and Nodular Regeneration: Cirrhosis is marked by the replacement of normal liver tissue with fibrous scar tissue and regenerative nodules, disrupting the hepatic architecture and impairing liver function.

2. Portal Hypertension: The fibrotic changes increase resistance to blood flow through the liver, leading to elevated pressure in the portal vein. Portal hypertension is a key driver of many gastrointestinal complications in cirrhosis.

3. Impaired Liver Function: The loss of functional hepatocytes affects various liver functions, including metabolism, detoxification, protein synthesis, and bile production.

Impact on the Gastrointestinal System

1. Esophageal and Gastric Varices: Increased portal pressure leads to the formation of varices in the esophagus and stomach. These varices are prone to rupture, causing life-threatening bleeding.

2. Ascites: Fluid accumulation in the peritoneal cavity, or ascites, is a common complication due to increased hydrostatic pressure in the portal vein and decreased oncotic pressure from hypoalbuminemia.

3. Hepatic Encephalopathy: Impaired detoxification function of the liver allows neurotoxins, particularly ammonia, to accumulate in the blood, leading to hepatic encephalopathy, characterized by altered mental status and cognitive dysfunction.

4. Bacterial Infections: Cirrhotic patients are more susceptible to bacterial infections, including spontaneous bacterial peritonitis (SBP), due to altered gut permeability and immune dysfunction.

5. Gastrointestinal Motility Disorders: Cirrhosis can affect gastrointestinal motility, leading to symptoms such as delayed gastric emptying, small intestinal bacterial overgrowth (SIBO), and altered bowel habits.

6. Malnutrition and Malabsorption: Impaired liver function and gastrointestinal complications contribute to malnutrition and deficiencies in fat-soluble vitamins and other nutrients.

Clinical Implications

1. Management of Portal Hypertension: Beta-blockers, endoscopic variceal ligation, and transjugular intrahepatic portosystemic shunt (TIPS) are used to manage portal hypertension and prevent variceal bleeding.

2. Ascites Management: Sodium restriction, diuretics, and paracentesis are employed to manage ascites. Refractory cases may require TIPS or liver transplantation.

3. Hepatic Encephalopathy Treatment: Lactulose and rifaximin are commonly used to reduce ammonia levels and manage hepatic encephalopathy.

4. Infection Prophylaxis and Treatment: Prophylactic antibiotics can prevent SBP, and prompt treatment of infections is crucial to prevent sepsis.

5. Nutritional Support: Nutritional assessment and support, including supplementation of vitamins and minerals, are essential to manage malnutrition in cirrhotic patients.

Materials and Methods: Case histories of 38 patients (2017-2019) of the therapeutic department of the 1st clinic of SamGosMI Samarkand, aged from 27 years to 60 years with the diagnosis of 'Liver cirrhosis' and 8 patients currently being treated. The results of ultrasound examination, blood biochemistry, liver biopsy, CT of the liver and biliary tract were analysed.

Results: In studies it is proved that, each patient has a peculiar condition of biliary system and organism as a whole. In 3 patients ascites, portal hypertension and liver failure were observed (in one patient is observed). The distribution of 38 patients (by medical history) with cirrhosis by disease etiology and by age will be provided below:

1. Alcoholic CP- 16 (42.1%), mean age 49.3 years,
2. Viral CP- 15 (39.5%), mean age - 52.4 years,
3. Primary biliary CP- 4 (10.52%), mean age - 44.7 years,

4. Mixed CP- 2 (5.26%), mean age - 50.1 years,

5. Cryptogenic CP-1 (2.63%), mean age 53.7 years.

6. (44.7%) of them died due to complications of liver failure.

The main morphological changes in all patients was formation of regeneration nodules and fibrosis. Signs of gallbladder and bile ducts lesions were observed in 27 (71%) patients. Gallbladder wall thickening and changes in its initial volume were common:

* increase in gallbladder volume in 11 (28,9%) patients;

* decrease in gallbladder volume was observed in 5 (13,15%) patients.

In patients with viral cirrhosis, acceleration of blood flow in portal vein was noted (2000 +/- 11ml/min). No dilation or narrowing inside or outside the hepatic ducts was detected.

The diagnosis of liver cirrhosis in 29 (73.3%) was made on the basis of clinical features such as:

* presence of jaundice;

* ascites;

* bleeding from varicose veins of the oesophagus.

In 8 (21%) patients a liver biopsy with a Menghini needle was made for morphological examination. To establish the diagnosis - liver cirrhosis, in other patients required additional investigations, because in them the degree of liver disease was in the initial stage.

The activity of the hepatic process was judged on the basis of ALT, AST, serum gamma globulin and histological changes in the liver.

Conclusions: The correct treatment of the patient requires timely determination of the type of cirrhosis, relying on ultrasound examination, liver CT, changes in ALT, AST and gamma globulin in the blood. After that, a comprehensive, correct therapeutic treatment should be carried out, and depending on the degree of cirrhosis, surgical intervention (resection or transplantation) can be carried out. On biopsy it is important to distinguish fibrosis regeneration nodules, which characterise true cirrhosis from:

* incompletely formed hepatic nodules,
* nodules without evidence of fibrosis,
* nodular regulatory hyperplasia,
* congenital hepatic fibrosis,
* widespread fibrosis without regenerative nodules.

The above-mentioned nodular masses cannot be attributed to true cirrhosis. Cirrhosis significantly impacts both the liver and the gastrointestinal system, leading to a range of complications that require comprehensive management. Understanding the pathophysiological changes in cirrhosis is essential for developing effective treatments and improving patient outcomes. Continued research into the mechanisms underlying these changes will further enhance our ability to manage and treat cirrhosis and its associated gastrointestinal complications.

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