CLINICAL AND DIAGNOSTIC ASPECTS OF LIVER CIRRHOSIS.

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Annotation. In recent years, there has been a clear trend towards an increase in the number of patients with diffuse liver lesions. Against this background, the frequency of the formation of cirrhosis of the liver (CP) with portal hypertension (PG) has significantly increased, and among their complications, the primary importance is given to the treatment and prevention of bleeding from varicose veins of the esophagus and stomach (VRVPIZH), the mortality from which can reach 30-80% [3]. This is due not only to the high incidence of viral hepatitis, but also to factors such as ecology, land cultivation with hepatotoxic poisons, the standard of living of the population, etc. The article examines the clinical and diagnostic features of the course of the disease in cirrhosis of the liver.

Keywords. Diagnosis, cirrhosis, liver, clinic, etiology of the disease.

КЛИНИКО-ДИАГНОСТИЧЕСКИЕ АСПЕКТЫ ЦИРРОЗА ПЕЧЕНИ .

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Аннотация. За последние годы отмечена явная тенденция к росту числа больных с диффузными поражениями печени. На этом фоне значительно возросла частота формирования цирроза печени (ЦП) с портальной гипертензией (ПГ), а среди их осложнений первостепенное значение отводится лечению и профилактике кровотечений из варикозно расширенных вен пищевода и желудка (ВРВПиЖ), летальность от которых может достигать 30-80% [3]. Это обусловлено не только высокой заболеваемостью

вирусными гепатитами, но и такими факторами, как экология, обработка земель гепатотоксичными ядами, уровень жизни населения и т.д. Статья рассматривает клинические и диагностические аспекты при циррозе печени.

Ключевые слова. Диагностика, цирроз, печень, клиника, этиология болезни.

Cirrhosis is characterized by fibrosis and nodule formation of the liver secondary to chronic injury, leading to alteration of the normal lobular organization of the liver. Various insults can injure the liver, including viral infections, toxins, hereditary conditions, or autoimmune processes. With each injury, the liver forms scar tissue (fibrosis), initially without losing its function. After a chronic injury, most of the liver tissue becomes fibrotic, leading to loss of function and the development of cirrhosis. This activity reviews the causes, evaluation, and management of hepatic cirrhosis and highlights the interprofessional team's role in the management of patients with this condition.

Chronic liver diseases usually progress to cirrhosis. In the developed world, the most common causes of cirrhosis are hepatitis C virus (HCV), alcoholic liver disease, and nonalcoholic steatohepatitis (NASH), while hepatitis B virus (HBV) and HCV are the most common causes in the developing world.[1] Other causes of cirrhosis include autoimmune hepatitis, primary biliary cholangitis, primary sclerosing cholangitis, hemochromatosis, Wilson disease, alpha-1 antitrypsin deficiency, Budd-Chiari syndrome, drug-induced liver cirrhosis, and chronic right-sided heart failure. Cryptogenic cirrhosis is defined as cirrhosis of unclear etiology. Patients with cirrhosis can be asymptomatic or symptomatic, depending on whether their cirrhosis is clinically compensated or decompensated. In compensated cirrhosis, patients are usually asymptomatic, and their disease is detected incidentally by labs, physical exams, or imaging. Portal hypertension can cause ascites, hepatosplenomegaly, and prominence of the periumbilical abdominal veins resulting in caput medusa. Esophageal varices are another complication of cirrhosis secondary to increased blood flow in the collateral circulation, with a mortality rate of at least 20% at six weeks after a bleeding episode.[1] Patients with

alcoholic cirrhosis are at increased risk of small bowel bacterial overgrowth and chronic pancreatitis, and patients with chronic liver disease have a higher rate of gallstones formation.[2] Anemia can occur due to folate deficiency, hemolytic anemia (spur cell anemia in severe alcoholic liver disease), and hypersplenism. There can be pancytopenia due to hypersplenism in portal hypertension, impaired coagulation, disseminated intravascular coagulation, and hemosiderosis in cirrhosis patients due to different causes.Patients with cirrhosis are prone to develop hepatorenal syndrome secondary to systemic hypotension and renal vasoconstriction, causing the underfilling phenomenon. Splanchnic vasodilation in cirrhosis leads to decreased effective blood flow to the kidneys, which activates the RAAS system, leading to retention of sodium and water and renal vascular constriction.[3] However, this effect is not enough to overcome the systemic vasodilation caused by cirrhosis, leading to renal hypoperfusion and worsened by renal vasoconstriction with the endpoint of renal failure.[4] Spider nevi (central arterioles surrounded by multiple smaller vessels that look like a spider, hence the name) are seen in cirrhosis patients secondary to hyperestrogenemia. Liver dysfunction leads to a sex hormone imbalance, causing increased estrogen to free testosterone ratio and the formation of spider nevi.[4] Palmar erythema is another skin finding that is seen in cirrhosis and is also secondary to hyperestrogenemia. Jaundice is a yellowish discoloration of the skin and mucous membranes seen when the serum bilirubin is greater than 3 mg/dL and in decompensated cirrhosis. A number of imaging modalities are used alongside labs to help in the diagnosis of cirrhosis. These include ultrasound, CT, MRI, and transient elastography (fibroscan). Ultrasonography is a cheap, noninvasive, and available modality for the evaluation of cirrhosis. It can detect nodularity and increased echogenicity of the liver, which are seen in cirrhosis; however, it is nonspecific as these findings can be seen in fatty liver as well.[6] It can also determine the ratio of the caudate lobe width to right lobe width, which usually increases in cirrhosis.[6] Moreover, it is a useful screening tool for HCC in cirrhotic patients. Duplex Doppler

ultrasonography helps to assess the patency of hepatic, portal, and mesenteric veins.

CT and MRI with contrast can detect HCC and vascular lesions, with MRI being superior to CT.[5] MRI also can be used to detect the level of iron and fat deposition in the liver for hemochromatosis and steatosis, and biliary obstruction if an MRC (magnetic resonance cholangiography) is obtained.[5] MRI, however, is expensive and not readily available. Transient elastography (fibroscan) is a non-invasive method that uses high-velocity ultrasound waves to measure liver stiffness, which correlates with fibrosis. In cirrhosis, a colloid liver spleen scan using technetium-99m sulfur colloid may show increased uptake of colloid in the bone marrow and spleen when compared to the liver. The presence of varices in the esophagus or stomach on esophagogastroduodenoscopy (EGD) suggests portal hypertension. Liver biopsy is the gold standard for diagnosing cirrhosis as well as assessing the degree of inflammation (grade) and fibrosis (stage) of the disease. Nevertheless, it can miss the diagnosis at times due to sampling errors.[6] The diagnosis of cirrhosis by biopsy requires the presence of fibrosis and nodules. The nodular pattern can be micronodular, macronodular, or mixed with the micronodular pattern representing an independent risk factor for elevated hepatic venous pressure gradient (HVPG) and more severe disease.[6]

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