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THE RELEVANCE OF CIRRHOSIS OF THE LIVER

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Annotation: Cirrhosis is the final stage of hepatic fibrosis, which leads to a widespread violation of hepatic architectonics. Cirrhosis is characterized by the formation of regeneration nodes surrounded by dense fibrous tissue. Symptoms may be absent for many years and are usually non-specific (e.g., anorexia, weakness, weight loss). Late manifestations include portal hypertension, ascites, hepatic encephalopathy and, when decompensation occurs, hepatic insufficiency. The diagnosis is usually made using non-invasive imaging techniques, although in rare cases a liver biopsy is needed. Treatment includes supportive therapy and treatment of the underlying cause of liver disease.

Key words: cirrhosis, fibrosis, necrosis, hepotocyte, hepatitis, liver, carcinoma, obstruction, glycogen, autoimmune.

Cirrhosis of the liver (CP) is a chronic diffuse liver disease characterized by a violation of its normal structure as a result of destruction (necrosis) and a decrease in the mass of functioning cells (hepatocytes), the development of connective tissue (fibrosis) and regeneration nodes. These processes in CP lead to the appearance of clinically important symptoms — liver failure (violation of detoxification, protein synthetic and other liver functions) and portal hypertension.

CP is a fairly common disease, occurs significantly more often in men, leads to a deterioration in the quality of life, early disability and mortality of patients. Diagnosis and treatment of this disease are associated with significant economic costs.

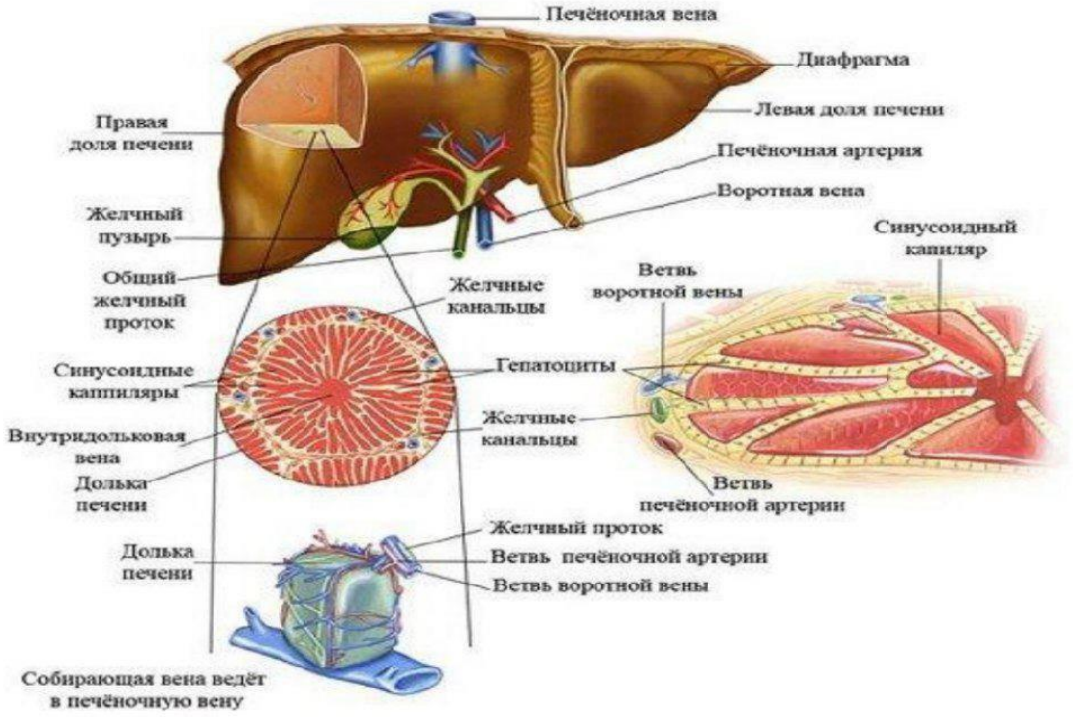

Рис. 1. Схема строения печени

Therapeutic tactics for cirrhosis of the liver (CP) consists of the treatment of the underlying disease that led to its development (antiviral drugs are prescribed only if there are markers of active viral replication in the blood; immunosuppression in autoimmune processes, D-penicillamine in Wilson-Konovalov disease, chelate therapy for hemochromatosis, etc.), and therapy aimed at eliminating complications. In cases of ineffectiveness of these methods and the progression of the disease, liver transplantation is indicated. In recent years, the development of liver cancer against the background of liver cirrhosis has been frequent. Thus, viral hepatitis is detected in anamnesis in 28.6% of patients with hepatocellular carcinoma, alcoholism – in 14.4%, medicinal hepatitis – in 2.8%, gallbladder and bile duct disease – in 5.6%, etiological factors remained unknown in 48% of patients with cirrhosis-cancer.

Cirrhosis ranks 14th among the leading causes of death worldwide.

According to A. F. Bluger and I. N. Novitsky (1984), mortality from CP in different countries ranges from 14 to 30 cases per 100,000 population.

Etiological factors of liver cirrhosis

1. Hepatitis B, C and D viruses
2. Alcohol
3. Metabolic disorders:
- hemochromatosis;
- Wilson-Konovalov disease;
- deficiency of a,-antitrypsin;
- mucopolysaccharidoses
4. Autoimmune diseases:
- autoimmune hepatitis;
- primary biliary cirrhosis;
- primary sclerosing cholangitis
5. Disorders of venous outflow from the liver:
- Budd-Chiari syndrome;
- veno-occlusive disease of drugs
6. Cryptogenic cirrhosis

Etiology

Viral hepatitis

Viral hepatitis is the cause of the development of viral CP in 10-23.5% of cases (S. D. Podymova, 1993). Chronic hepatitis B, C, D, and probably G. In 30% of cases (and according to some data — in 50%), chronic active viral hepatitis evolves in the CPU. According to Regillo (1990), among chronic carriers of HBsAg, CP is formed in 10% of cases, and according to morphological examination of biopsies — in 20-60% of cases.

According to Kage (1997), CP develops in 20-25% of patients with chronic hepatitis C, and with histological control of biopsies — in 50% (Dienstag, 1995). HCV genotype 1B is the most cirrhogenic. HCV cirrhosis of the liver remains compensated for many years and is not recognized.

Autoimmune hepatitis

Autoimmune hepatitis is characterized by a severe course, the frequency of its transition to the CPU is higher, and the prognosis is much more serious than with viral hepatitis (S. D. Podymova, 1993).

Chronic alcohol intoxication is the cause of the development of CP in 50% of cases. The disease usually develops 10-15 years after the onset of alcohol abuse. According to Thaler, CP develops in men with a daily intake of 60 g of alcohol, in women — 20 g during the specified period.

Galactose-1-phosphate-uridyltransferase deficiency

Congenital deficiency of galactose-1-phosphate-uridyltransferase leads to the development of galactosemia. At the same time, early childhood cirrhosis of the liver is formed. The mechanism of development of this cirrhosis is unknown.

Diseases of glycogen accumulation

Congenital deficiency of the enzyme amyl-1,6-glycosidase leads to the development of glycogen accumulation diseases and liver cirrhosis.

Chemical toxic substances and medicinal products

Cirrhosis of the liver can be formed under the influence of the following toxic substances:

• industrial poisons (carbon tetrachloride, dimethylnitrosamine, chloroform, benzene, nitro- and amino compounds, etc.);
• heavy metal salts (chronic mercury intoxication, etc.);
• mushroom poisons (phalloidin, phalloin, β-amanitin) cause massive liver necrosis with subsequent cirrhosis;
• aflatoxins (contained in overwintered grain, corn, rice).

In addition, some medicinal substances with prolonged use can cause the development of cirrhosis of the liver:

• methyldof;
• isoniazid;
• paraaminosalicylic acid (PASC);
• iprasid;
• preparations containing arsenic;
• inderal in large doses;
• cytostatics (in particular, methotrexate);
• steroid anabolic drugs and androgens.

Obstruction of extrahepatic and intrahepatic biliary tract

Intrahepatic biliary obstruction of the autoimmune gene leads to the development of primary biliary cirrhosis of the liver (see chapter "Primary biliary cirrhosis of the

liver"). Secondary biliary CP develops due to a prolonged violation of bile outflow at the level of large intrahepatic and extrahepatic bile ducts.

Prolonged venous stagnation in the liver

Prolonged venous stagnation in the liver contributes to the development of cirrhosis of the liver. Venous stasis is most often caused by heart failure (especially with tricuspid insufficiency), less often by constrictive pericarditis and endophlebitis of hepatic veins (Budd-Chiari disease).

Classification

There is no single CPU classification. Most experts consider it appropriate to classify CP depending on the etiology, morphological characteristics, the stage of portal hypertension and hepatic cell insufficiency, the activity of the inflammatory process, the course variant. An example of such a classification is the classification of A. S. Loginov and Yu. E. Blok (1987). It has become widespread (Table 45).

Табл. 45. Классификация цирроза печени (А. С. Логинов, Ю. Е. Блок, 1987)

Этиологические варианты	Морфологические варианты	Стадия портальной гипертензии	Стадия печеночно-клеточной недостаточности	Активность и фаза	Течение
1 Вирусный 2 Алкогольный 3 **Аутоиммунный** 4 Токсический 5 Генетический 6 **Кардиальный** 7 Вследствие **внутри- и внепеченочного холестаза** 8 **Криптогенный**	1 **Микронодулярный** 2 **Макронодулярный** 3 **Смешанный** 4 Неполный **септальный** 5 **Билиарный**	1 **Компенсированная** 2 Стадия начальной **декомпенсации** 3 Стадия выраженной **декомпенсации**	1 **Компенсированная** (начальная) 2 **Субкомпенсированная** 3 **Декомпенсированная**	1 **Обострение (активная фаза)** активность **минимальная, умеренная, выраженная)** 2 Ремиссия **(неактивная** фаза)	1 **Медленно прогрессирующее** 2 Быстро **прогрессирующее** 3 **Стабильное**

Clinical picture

Cirrhosis of the liver is more common in men. The clinical picture of the disease is characterized by a variety of symptoms. According to S. D. Podymova (1993), 60 patients have a pronounced clinical picture, in 20% of patients cirrhosis of the liver occurs latently and is detected accidentally during examination.

Main complaints:

• pain in the right hypochondrium and the epigastric region, which increases after eating (especially after taking spicy, fatty foods), physical activity. The pain is caused by enlargement of the liver and stretching of its capsule, concomitant chronic gastritis, chronic pancreatitis, cholecystitis, biliary dyskinesia. With hyperkinetic dyskinesia of the biliary tract, the pain in the right hypochondrium is colic, with hypokinetic dyskinesia they are usually not intense, pulling, often bothered by a feeling of heaviness in the right hypochondrium;
• nausea, sometimes vomiting (bloody vomiting is possible with bleeding from varicose veins of the esophagus and stomach);
• feeling of bitterness and dry mouth;
• itching of the skin (with cholestasis and accumulation of a large amount of bile acids in the blood);
• fatigue, irritability;
• frequent loose stools (especially after eating fatty foods);
• bloating;
• weight loss;
• sexual weakness (in men), menstrual cycle disorders (in women).

Objective signs of liver cirrhosis detected by physical examination:
- hepatomegaly and/or splenomegaly
- jaundice, subicterицity.
- Erythema palmar, Dupuytren's contracture.
- vascular asterisks
- hemorrhagic rashes (ecchymoses, petechiae, hematomas).
- systemic manifestations (in autoimmune liver diseases)
- ascites
- gynecomastia

Classification of cirrhosis of the liver
To determine the stage of liver cirrhosis, the Child–Pugh criteria are used (Table 2). According to the sum of the indicators, 3 stages of the disease are distinguished: the first (class A, and compensated) — 5-6 points, the second (class B, subcompensated)

— 7-9 points, the third (class C, decompensated) — more than 9 points.

Классификационные признаки цирроза печени по Чайлд-Пью

Признаки	Баллы		
	1	2	3
Протромбиновое время, сек	1-4	>4-6	>6
Билирубин, мкмоль/л	<34	34-51	>51
Альбумин, г/л	>35	35-28	<28
Асцит	Нет	Мягкий	Напряженный
Энцефалопатия	Нет	Стадия 1-2	Стадия 3-4

GENERAL PRINCIPLES OF LABORATORY AND INSTRUMENTAL DIAGNOSTICS

- Virological examination.
- Clinical blood test at the stage of cirrhosis: thrombocytopenia, leukopenia, increased ESR may be observed.
- In the biochemical study of blood, 4 syndromes are distinguished: cytolytic, mesenchymal inflammatory, cholestatic and hepatic cell insufficiency.
- Liver cell insufficiency syndrome: hyperbilirubinemia due to unconjugated fraction, decreased blood levels of albumin, prothrombin, transferrin, cholesterol esters, proconvertin, proaccelerin. Detoxification function of the liver is significantly impaired. Increasing the concentration of ammonia, phenols.
- Fibrogastroduodenoscopy: reveals the degree of varicose veins of the esophagus and stomach,
- Liver scanning, which determines the size and position of the organ, the uniformity of the distribution of RFP, the activity of its accumulation in the spleen as a sign of portal hypertension and hypersplenism.
- Ultrasound of the abdominal cavity, where hepatomegaly and splenomegaly are detected, an increase in the diameter of the portal and splenic veins, a change in the echostructure of the liver and extrahepatic bile ducts, gallbladder, the presence of fluid in the abdominal cavity.
- Computed tomography is advisable to exclude focal organ damage.
- Liver biopsy, histological examination of a biopsy is an important method for diagnosing and monitoring the effectiveness of treatment, determining the activity and stage of the process, the severity of structural changes in the liver. The same

method is used for differential diagnosis between congenital metabolic, viral and other liver diseases and HCG.

CONSERVATIVE TREATMENT

The main type of cirrhosis treatment is conservative therapy, including:

Drug treatment (antiviral, immunosuppressive, detoxifying, diuretic drugs, drugs that reduce pressure in the portal vein system, laxatives, normalizing the microflora of the colon and others, depending on the clinical situation). Drug treatment eliminates the etiological factor and prevents the development of complications of cirrhosis of the liver. Our doctors, choosing treatment tactics, are based on the principles of evidence-based medicine and authoritative Western and Russian recommendations.

Diet therapy. Nutrition should be high in protein and high in calories, but not in the case of severe hepatic encephalopathy. When fluid accumulates in the abdominal cavity, a low–salt diet is necessary, protein intake is 1.5 g per 1 kg of weight.

Complete abstinence from alcohol. The exclusion of alcohol-containing beverages contributes to the speedy improvement of the condition.

Limitation of physical activity. Allows you to prevent the development of complications.

Conservative treatment is usually carried out on an outpatient basis. At the same time, the patient is under dispensary supervision and should regularly visit a doctor. If necessary, the patient can undergo a course of intravenous drip infusions in our comfortable hospital.

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