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LIVER CIRRHOSIS DISEASE AND DISEASE SYMPTOMS

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Abstract: This article provides information about the disease "Liver cirrhosis". In the article, you will get information about the symptoms and causes of the disease. Using scientific sources, it is explained in detail about disease prevention, disease protection, different stages of disease and disease treatment measures. The human body is a whole organism, in which every organ has its own importance. The liver is one of the most important organs in our body. It performs many important biological functions, such as detoxification of the body, synthesis of proteins and biochemical substances necessary for digestion and growth. Its other metabolic roles include carbohydrate metabolism, hormone production, conversion and storage of nutrients such as glucose and glycogen, and breakdown of red blood cells. After all, if the liver stops working, it causes serious problems for the whole body. Many liver diseases are insidious at a treatable stage. Symptoms begin to be felt when the liver is already significantly damaged and serious disorders occur. In severe cases, liver disease can lead to the death of the patient. Among the common and dangerous pathologies of this organ, cirrhosis takes one of the leading places. But cirrhosis rarely develops "by itself". Exceptions are primary biliary cirrhosis, in which liver cells are damaged by the immune system. In most cases, it occurs as a result of untreated chronic problems.

Key words: liver, cirrhosis, liver diseases, chronic disease, hepatitis, symptom, the most important organ, gallbladder (bile), biliary cirrhosis, alcohol.

JIGAR SERROZI KASALLIGI VA KASALLIK ALOMATLARI

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Annotatsiya: Ushbu maqolada “Jigar serrozi” kasalligi haqida ma’lumot berilgan. Maqolada kasallikning alomatlari, kelib chiqish sabablari haqida ma’lumot olasiz. Bunda ilmiy manbalardan foydalangan holda kasallikni oldini olish,kasallikdan himoyalanish,kasallikning turli bosqichlari va kasallikni davolash chora-tadbirlari haqida batafsil yoritilgan.Inson tanasi yaxlit organizm ,bunda har bir a’zoning o’z ahamiyati bor.Jigar-tanamizdagi eng katta ahamiyatga ega bo’lgan organlardan biridir.U organizmni detoksifikatsiya qilish, hazm qilish va o‘sish uchun zarur bo‘lgan oqsillar va biokimyoviy moddalarni sintez qilish kabi ko‘plab muhim biologik funksiyalarni bajaradi. Uning boshqa metabolik rollariga uglevod almashinuvi, gormonlar ishlab chiqarish, glyukoza va glikogen kabi ozuqa moddalarini aylantirish va saqlash ,qizil qon hujayralarining parchalanishi kiradi. Mobodo,jigar ishlashdan to’xtasa bu butun organizm uchun jiddiy muommolarni keltirib chiqaradi. Ko’pgina jigar

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kasalliklari davolash mumkin bo'lgan bosqichida yashirin ravishda kechadi. Alomatlar jigar allaqachon sezilarli darajada shikastlanganda va jiddiy buzilishlar yuzaga kelganida sezila boshlaydi. O'ta murakkab holatlarda jigar kasalligi bemorning o'limiga olib kelishi mumkin.

Ushbu a'zoning keng tarqalgan va xavfli sanaladigan patologiyalari orasida sirroz yetakchi o'rinlardan birini egallaydi. Ammo sirroz juda kamdan-kam hollarda «o'z-o'zidan» rivojlanadi. Istisno holatlari - birlamchi biliar sirroz, bunda jigar hujayralariga immun tizimi tomonidan shikast yetkaziladi. Aksariyat hollarda esa u davolash choralari ko'rilmagan surunkali muammolar natijasida yuzaga keladi.

Kalit so'zlar:jigar,serroz,jigar kasalliklari,surunkali kasallik,gepatit,symptom,eng muhim organ,o't pufagi(safro),billiar serroz,alkagol.

ЦИРРОЗ ПЕЧЕНИ И СИМПТОМЫ ЗАБОЛЕВАНИЯ

Аннотация: В данной статье представлена информация о заболевании «Цирроз печени». В статье вы получите информацию о симптомах и причинах заболевания. С использованием научных источников подробно рассказывается о профилактике заболеваний, их защите, различных стадиях заболеваний и мерах лечения. Организм человека представляет собой целостный организм, в котором каждый орган имеет свое значение. Печень является одним из важнейших органов нашего организма. Она выполняет множество важных биологических функций, таких как детоксикация организма, синтез белков и биохимических веществ, необходимых для пищеварения и роста. Другие его метаболические функции включают углеводный обмен, выработку гормонов, преобразование и хранение питательных веществ, таких как глюкоза и гликоген, а также распад эритроцитов. Ведь если печень перестает работать, это вызывает серьезные проблемы для всего организма. Многие заболевания печени коварны на излечимой стадии. Симптомы начинают давать о себе знать, когда печень уже значительно повреждена и возникают серьезные нарушения. В тяжелых случаях заболевание печени может привести к смерти больного.Среди распространенных и опасных патологий этого органа одно из ведущих мест занимает цирроз печени. Но цирроз редко развивается «сам по себе». Исключением является первичный билиарный цирроз печени, при котором клетки печени повреждаются иммунной системой. В большинстве случаев это происходит в результате невылеченных хронических проблем.

Ключевые слова: печень, цирроз печени, заболевания печени, хронические заболевания, гепатит, симптом, важнейший орган, желчный пузырь (желчь), билиарный цирроз печени, алкоголь.

Cirrhosis of the liver is a chronic disease of the organ, which is characterized by the irreversible replacement of liver parenchymatous tissue with fibrous connective tissue or stroma. Cirrhotic liver is enlarged or reduced in size, abnormally dense, bumpy. Death occurs at the terminal stage, depending on various circumstances, within 2-4 years, when the patient experiences severe pain and suffering.

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Epidemiology: In economically developed countries, cirrhosis of the liver is one of the 6 main causes of death in patients aged 35-60 years, with 14-30 cases per 100,000 population. Every year, 40 million people die in the world with cirrhosis of the liver and hepatocellular carcinoma developed against the background of hepatitis B virus transport. In the CIS countries, this disease occurs in 1% of the population. The disease is often observed in men: the ratio of sick men to women is on average 3:1. The disease can develop in any age group, but is most often noted after the age of 40.

Causes: Cirrhosis of the liver often develops against the background of long-term alcohol intoxication (from 40-50% to 70-80% according to various data), parasitic infection and viral hepatitis B, C and D (30-40%). Less common causes of cirrhosis are diseases of the biliary tract (inside and outside the liver), congestive heart failure, various chemicals (hepatotoxins) and drugs. poisoning. Cirrhosis can also develop in the presence of genetic disorders of metabolism (hemochromatosis, hepatolenticular degeneration, α1-antitrypsin deficiency) and occlusive processes in the portal vein system (phleboportal cirrhosis).

Infectious factors: chronic viral hepatitis, especially B and C, parasitic infections, especially fungal and trematodes (schistosomiasis, opisthorchosis, candidiasis, aspergillosis). Primary biliary cirrhosis of the liver usually occurs for no apparent reason. In approximately 10-35% of patients, the etiology remains unclear.

Pathogenesis: Over many months and years, the genome of hepatocytes changes and a clone of pathologically changed cells is created. As a result, the immune-inflammatory process develops. The following stages of the pathogenesis of cirrhosis are distinguished:

1. Etiological factors: cytopathogenic effects of viruses, immune mechanisms, hepatotoxic cytokines, chemokines, prooxidants, eicosanoids, acetaldehyde, iron, effects of lipid peroxide oxidation products;

2. Activation of the function of Ito cells, which leads to excessive growth of connective tissue in the perisinusoidal space and pericellular fibrosis of the liver;

3. Violation of blood supply of the liver parenchyma as a result of the narrowing of the vascular space with the development of capillarization of sinusoids and ischemic necrosis of hepatocytes;

4. Cytolysis of hepatocytes, activation of immune mechanisms.

In the bridging necrosis of hepatocytes, T-lymphocytes are attracted to the area of damage, and they activate Ito cells, which acquire fibroblast-like properties: these cells synthesize type I collagen, which in turn eventually leads to fibrosis. In addition, microscopically, false segments without a central vein are formed in the liver parenchyma.

Influencing factors:

- **Alcoholic.** Stages: acute alcoholic hepatitis and hepatic dystrophy with fibrosis and

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mesenchymal reaction. The most important factor is the direct toxic effect of alcohol, as well as necrosis of hepatocytes due to autoimmune processes.

- **Autoimmune.** An important factor is the sensitization of immunocytes to the body's own tissues. The main target of autoimmune reaction is liver lipoprotein.

- **Congestive.** Hepatocyte necrosis is associated with hypoxia and venous damping.

Portal hypertension: Increased pressure in the portal vein system of the liver due to internal or external obstruction of the vessels. This leads to the formation of portocaval shunting of blood, splenomegaly and ascites (accumulation of fluid in the abdominal cavity in cirrhosis). Thrombocytopenia (strong deposition of platelets in the spleen), leukopenia, as well as anemia due to high hemolysis of erythrocytes, is associated with splenomegaly. Ascites can cause the following syndromes:

- Limitation of diaphragm mobility;
- Gastroesophageal reflux with peptic erosion;
- Ulcers and bleeding from varicose veins of the esophagus;
- Ventricular hernia;
- Bacterial peritonitis;
- Hepatorenal syndrome.

Primary biliary cirrhosis: The main place belongs to genetic disorders of immunoregulation. First, there is a violation of the biliary epithelium, and then there is a segmental necrosis of the ducts, and then their proliferation: this is accompanied by violations of the excretion of bile. The steps of the process are as follows:

- Chronic non-purulent destructive cholangitis;
- Ductular proliferation with destruction of bile ducts;
- Scarring and narrowing of bile ducts;
- Large nodular cirrhosis with cholestasis.

The pathologoanatomical picture of primary biliary cirrhosis includes the infiltration of the epithelium with lymphocytes, plasma cells, macrophages. Antimitochondrial antibodies (AMA) are detected in laboratory studies, the most characteristic of which are M2-AMA directed against the E2 subunit of pyruvate dehydrogenase, an increase in serum IgM. In addition, extrahepatic phenomena indirectly caused by immunity - Hashimoto's thyroiditis, Sjögren's syndrome, fibrosing alveolitis, tubulointerstitial nephritis, celiac disease, as well as systemic scleroderma, rheumatoid arthritis, and systemic lupus erythematosus.

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Stages: The disease proceeds in several stages, each stage has its own clinical symptoms. Depending on how advanced it is, not only the patient's condition, but also the therapy methods are different.

1. **Compensated phase:** At this stage of development, the disease does not manifest itself in any way. If the pathology is diagnosed at this stage, it is possible to compensate for the liver failure with the help of drugs. In this phase, liver cells - hepatocytes undergo necrosis (death), and instead of them, fibrous scar tissue begins to form. If the treatment is not started on time, soon the organ will not be able to fully perform its functions. At this time, laboratory analyzes show that the level of bilirubin has increased, and the prothrombin index has decreased to 60. In general, the patient feels healthy, only sometimes the pain under the right rib is annoying.

2. **Subcompensated phase:** In this phase, the symptoms of the disease begin to be felt more clearly. This indicates an increase in the number of dead hepatocytes. At this stage, the patient has symptoms such as weakness, apathy (indifference), reduced work capacity, nausea, and weight loss. In men, the first signs of gynecomastia can be noted. In laboratory indicators, it is noted that the level of albumin decreases and the prothrombin index reaches 40. If the treatment is started on time, the disease can be brought to the compensated stage.

3. **Decompensated phase:** in the 3rd phase, the number of normally functioning hepatocytes is greatly reduced. This leads to the development of liver failure and increased symptoms of the disease. The patient's skin turns yellow, pains in the abdomen begin to be felt. At this stage, astitis (accumulation of fluid in the abdominal cavity) often develops. Laboratory analysis reveals that the level of albumin and the prothrombin index have decreased significantly. Treatment is ineffective and there is a risk of complications (especially liver coma, cancer, internal bleeding, peritonitis and pneumonia). The patient must be hospitalized under the supervision of a doctor.

4. **Terminal phase:** In the final stage of the disease, the organ is unable to perform its function due to severe damage. The patient suffers from severe pain, because of which he is prescribed strong painkillers. At this stage, there is no way to stop the development of the pathology. The prognosis is generally negative. If a new liver is not transplanted, death will occur due to severe complications of the disease.

Symptoms of cirrhosis of the liver: Many non-hepatic symptoms are associated with increased pressure in the sinusoids, which leads to increased pressure in the portal venous system. Another characteristic symptom of the disease - "Medusa's head" - is the filling of the veins of the anterior abdominal wall with blood.

Common symptoms of cirrhosis include:

- Weakness, reduced working capacity;
- Unpleasant feelings in the abdomen;

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- Dyspeptic disorders;
- Increase in body temperature;
- Pain in the joints;
- Flatulence, pain and heaviness in the upper half of the abdomen;
- Weight loss;
- Asthenia.

During the examination, it is determined that the liver is enlarged, its surface is thickened and deformed, and its edges are sharpened. At first, uniform, moderate enlargement of both lobes of the liver is noted, and later, as a rule, enlargement of the left lobe is more dominant. Portal hypertension is manifested by a slight enlargement of the spleen. A common clinical picture is manifested by liver-cellular failure and portal hypertension syndromes. Abdominal discomfort, intolerance to fatty food and alcohol, nausea, vomiting, diarrhea, heaviness and abdominal pain (mainly under the right rib) are also observed. In 70% of cases, hepatomegaly is detected, the liver is thickened, and its edges are sharp. In 30% of patients, palpation of the surface of the liver is nodular, and in 50%, splenomegaly is noted. Subfebrile fever may be associated with the passage of intestinal bacterial pyrogens that cannot be neutralized by the liver. The fever is resistant to antibiotics and passes only when the liver function improves.

External symptoms observed in liver cirrhosis:

- Palmar (palm) or plantar erythema;
- Veined asterisks;
- Lack of hair in the armpit and groin area;
- Whiteness of nails;
- Development of gynecomastia due to hyperestrogenemia in men.
- Similarity of fingers to "drumsticks".

In the terminal stage of the disease, 25% of cases show a decrease in the size of the liver. Also, due to jaundice, ascites, hyperhydration, peripheral edema (first of all, swelling of the legs), external venous collaterals (varicose dilatation of the esophagus, stomach, intestinal veins) also occur. Bleeding from the veins often leads to death. Sometimes hemorrhoidal bleeding is observed, but their intensity is less. Encephalopathy can be the result of both hepatocellular and portal liver failure.

Complications:

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- Hepatic coma;
- Bleeding from varicose veins of the esophagus;
- Thrombosis in the portal vein system;
- Hepatorenal syndrome;
- Liver cancer — formation of hepatocellular carcinoma;
- Infectious complications — pneumonia, "spontaneous" peritonitis in ascites, sepsis.

Diagnosis: The disease is characterized by increased activity of alkaline phosphatase, ALT, AST, and leukocytosis. This is determined by biochemical analysis of blood. Hepatolienal syndrome can develop leukopenia, thrombocytopenia, anemia, and hypersplenism, which is manifested by an increase in cellular elements in the bone marrow. Expanded and branched venous collaterals are visible during angiography, computer tomography, ultrasound examination or surgical intervention. If necessary, MRI of the liver and dopplerometry of the liver vessels can be performed.

Child-Pugh liver failure severity rating scale

Liver cell function in liver cirrhosis is evaluated according to Child-Pugh.

Parameter	Points		
	1	2	3
Ascites	No	Mild, treatable	Advanced, difficult to treat
Encephalopathy	No	Light (I-II)	Severe (III-IV)
Bilirubin, μmol/l (mg%)	less than 34 (2.0)	34-51 (2.0-3.0)	over 51 (3.0)
Albumin, g	More than 3.5	2,8-3,5	less than 2.8
PTV, (seconds) or PTI (%)	1-4 (over 60)	4-6 (40-60)	More than 6 (less than 40)

The class of cirrhosis is determined based on the sum of points for all indicators. When the sum of points is 5-6, it is class A, when it is 7-9 it is class B, and when it is 10-15 it is class C.

1. Class A patients' life expectancy is expected to be 15-20 years. The postoperative mortality rate in abdominal surgery is 10%.

2. Class B is an indication for liver transplantation. The rate of postoperative death in abdominal surgery is 30 percent.

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3. The life expectancy of C-class patients is expected to be 1-3 years, and the postoperative mortality rate in abdominal surgery is 82%.

The need for a liver transplant is also assessed based on the Child-Pugh criteria: for class C patients, it is extremely necessary, for class B - moderate, and for class A - low.

SAPS criteria system: In recent years, the SAPS (Simplified Acute Physiology Score) criteria system, which includes basic physiological parameters, has been used to determine the prognosis of patients during the development of gastrointestinal bleeding, coma, sepsis, and other complications. The patient's age, number of heart contractions (HRC), respiratory rate, systolic arterial pressure, body temperature, diuresis, hematocrit, blood leukocytes, urea, potassium, sodium, and plasma are used for classification. The amount of bicarbonates, as well as the stage of liver coma, have a value.

SAPS evaluation criteria:

Unit of assessment	Points								
	4	3	2	1	0	1	2	3	4
Age, in years					≤45	46-55	56-65	66-75	>75
YQS, per minute	≥180	140-179	110-139		70-109		55-69	40-54	<40
Systolic arterial pressure, mm. sim. above.	≥190		150-189		80-149		55-79		<55
Body temperature, ºC	≥41	39,0-40,9		38,5-38,9	36,0-38,4	34,0-35,9	32,0-33,9	30,0-31,9	<30

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Breathing rate, per minute	≥50	35-49		25-34	12-24	10-11	6-9	*O'SV or HDMB	<6
Urine volume, l/milk			≥5,00	3,50-4,99	0,70-3,49		0,50-0,69	0,20-0,49	<0,2
Blood urea, mmol/l	≥55	36 −54,9	29 −35,9	7,5 −28,9	3,5 −7,4	<3,5			
Hematocrit, %	≥60,0		50,0-59,9	46,0-49,9	30,0-45,9		20,0-29,9		<20,0
The number of leukocytes, ×109/l	≥40		20,0-39,9	15,0-19,9	3,0-14,9		1,0-2,9		<1
Blood glucose, mmol/l	≥44,4	27,8-44,3		13,9-27,7	3,9-13,8		2,8-3,8	1,6-2,7	<1,6
Blood potassium, mEq/l	≥7,0	6,0-6,9		5,5-5,9	3,5-5,4	3,0-3,4	2,5-2,9		<2,5
Sodium in blood, mEq/l	≥180	161-179	156-160	151-155	130-150		120-129	110-119	<110

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HCO3, mEq/l		≥40		30,0-39,9	20,0-29,9	10,0-19,9		5,0-9,9	<5,0
Glasgow scale, scores					13-15	10-12	7-9	4-6	3

HDMB — constant positive air pressure, O'SV — artificial lung ventilation.

Liver Cirrhosis Treatment: Liver cirrhosis is treated with medication and a strict diet, but once it develops, it is irreversible. In this case, the liver cannot be treated, the only way to save the patient's life is liver transplantation. In severe ascites, fluid can be removed from the abdomen.

Diet: The diet in liver cirrhosis should be complete, containing 70-100 g of protein (1-1.5 g per 1 kg of body mass), 80-90 g of fats (50 percent of which are plant-based), and 400-500 g of carbohydrates. It is necessary to take into account the patient's habits, tolerance to food and the presence of other diseases of the digestive system. Chemical additives, preservatives and toxic ingredients are excluded from the recipe. The diet changes when there are complications of portal hypertension. Contraindicated products include:

- Any chemical food additives, including preserves;
- Fried, salted, marinated, smoked;
- Confectionery, chocolate, ice cream;
- Animal fats, margarine, fatty meat and meat broths;
- Salty cheeses, dairy products with a high percentage of fat;
- Legumes, radish, spinach, radish, corn, garlic, onion;
- Sour fruits and berries;
- Sweet carbonated drinks, spicy tea and coffee.

Ursodeoxycholic acid: Use of ursodeoxycholic acid (UDXK) is appropriate to replace bile acid deficiency in the intestine caused by biliary insufficiency. Dosage — 1 time a day, in the evening, 10-15 mg per 1 kg of the patient's weight, which helps to restore digestive processes. The impact of UDXK includes the following:

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1. An increase in the amount of pancreatic and bile fluid entering the intestine;
2. Completion of intrahepatic cholestasis;
3. Increased contraction of the gallbladder;
4. Saponification of fats and increase in lipase activity;
5. Improvement of intestinal motility, which improves mixing of enzymes with chyme;
6. Normalization of the immune response.

Homeopathic medicines: The most popular remedies for liver diseases are:

- Phosphorus 6, 12;
- Magnesia Muriatica 6;
- Lycopodium 6;
- Nux Vomica 6;
- Mercur dulcis 6 and others.

However, it should not be forgotten that homeopathy is not recognized by official evidence-based medicine, and its methods are not subjected to serious clinical trials. Today, it has not been confirmed that such preparations have a therapeutic effect due to their components.

Medicinal therapy: It should be noted that there is no specific treatment method for cirrhosis. In compensated or subcompensated cirrhosis, supportive therapy is prescribed - a strict diet and hepatoprotectors (glycyrrhizic acid, phospholipids, amino acids, milk thistle (Silybum marianum) and other components that contribute to the restoration of liver function). In the decompensated stage of the disease, the effectiveness of drug therapy is low, and the issue of liver transplantation should be considered. If the cause of liver cirrhosis is viral in the case of primary disease such as hepatitis C and B, therapy includes anti-viral, as well as anti-fibrosis and cirrhotic treatments.

Cellular therapy: Conventional treatment mainly consists of using pharmaceutical agents to protect liver cells from damage, stimulate bile secretion, and correct metabolic disorders. Undoubtedly, this improves the patient's condition, but cannot stop the development of the disease. If the above treatments do not help, liver transplantation (transplantation) is performed.

Any approach aimed at preventing hepatitis can be included in the preventive measures of liver cirrhosis. In addition, it is recommended to follow the following: treatment of hepatitis with the help of a qualified hepatologist and compliance with the prescribed therapeutic regimen; limit arbitrary intake of drugs, avoid working in harmful industrial enterprises. Take vitamin and mineral complexes according to the doctor's recommendation; do not eat fatty, fried and bitter-tasting, canned and semi-finished products; refrain from bad habits, especially the abuse of

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alcoholic beverages; annual endoscopic examination of the digestive system it is also recommended to take a vaccine against viral hepatitis B.

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