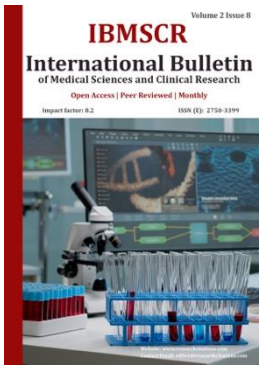

CAUSES, DIAGNOSIS AND PREVENTION OF LIVER CIRRHOSIS

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Abstract:

Liver cirrhosis is a chronic disease characterized by drastic changes in liver tissue and the development of fibrosis, which disrupts normal liver function. This disease may not cause any symptoms at first, but over time it damages the liver, increases pressure in the internal blood vessels, causes changes in the liver mucosa, and leads to poisoning of the body. Liver cirrhosis is often associated with hepatitis, alcoholism, fatty liver disease, or other factors that damage the liver. It is important to detect and begin treatment of the disease early, as cirrhosis can develop early and lead to complete loss of liver function. The disease is diagnosed by laboratory and imaging techniques such as blood tests, liver biopsy and ultrasound.

Keywords: cirrhosis, liver fibrosis, liver failure, hepatitis, alcoholism.

Introduction

Liver cirrhosis (LC) is a chronic diffuse disease of the liver characterized by disruption of its normal structure as a result of destruction (necrosis) and reduction in the mass of functioning cells (hepatocytes), development of connective tissue (fibrosis) and regeneration nodules. These processes in LC lead to the appearance of clinically important symptoms - hepatic insufficiency (impaired detoxification, protein synthetic and other liver functions) and portal hypertension. The latter is caused by a violation of the flow of blood from the liver, coming into it from the abdominal organs through the portal system. Patients with LC have a high risk of developing primary liver carcinoma.

LC 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.

Etiology. Most often, LC is the outcome (end stage) of chronic viral hepatitis B, C and D, significantly less often - autoimmune hepatitis. The cause of LC, in addition to infection with hepatotropic viruses and autoimmune process, may be long-term alcohol abuse (alcoholic cirrhosis), exposure of the liver to toxic substances in the home and at work (heavy metals, chlorinated hydrocarbons and naphthalenes, benzene and its derivatives, etc.), the use of certain drugs (alcoholic cirrhosis).), use of some drugs (cytostatic, narcotic, hormonal contraceptives, hepatotoxic antibiotics, etc.), genetically determined disorders of iron and copper metabolism, biliary tract diseases.

Diagnosis. The clinical course of LC is characterized by great diversity: from complete absence of clinical manifestations to pronounced and rapidly progressive signs of liver damage. Depending on the severity of the clinical picture - absence or presence of hepatic encephalopathy and portal hypertension - and violations of biochemical (functional)

indicators (prothrombin index, bilirubin and albumin levels), compensated, subcompensated and decompensated LC are distinguished.

Patients are often bothered by general weakness, increased fatigue, decreased ability to work, irritability, bitterness in the mouth, decreased or no appetite, nausea, flatulence, discomfort and heaviness in the right subcostal and pancreatic region, skin itching, stool disorders, darkening of urine, decreased diuresis, increased body temperature.

There is significant evidence of persistent or intermittent jaundice coloration of the skin and visible mucous membranes. Liver encephalopathy, caused by damage to the central nervous system as a result of the toxic effect on it of ammonia, phenols, bacterial toxins and other substances in liver failure, characterized by the appearance of low mood, lethargy, drowsiness during the day and insomnia at night, inability to prolonged concentration of attention and thought, indifference to the surrounding, then there may be a loss of consciousness. Exhaled air has a sweetish taste - "liver odor". If emergency measures are not taken, the patient may die. The development of hepatic encephalopathy can be promoted by gastrointestinal bleeding, taking large doses of diuretics (to reduce ascites), an attached infection (acute respiratory viral infections, influenza, pneumonia, cystitis, pyelonephritis, etc.), the content of large amounts of protein in the diet.

Indications of skin hemorrhagic manifestations (bruises, bruises), bleeding of various localizations (nasal, esophageal, gastrointestinal, renal) complement the clinical picture of this disease.

Examination often reveals varying degrees of jaundice coloration of the skin and mucous membranes, traces of combing, bright red coloration of the border of the lips, crimson ("liver") color of the tongue, a decrease in body hair, some increase in breast glands in men (gynecomastia). May be found hemorrhagic manifestations and peculiar vascular formations on the skin in the form of stars and spiders (telangiectasia), redness of the palms (palmar erythema), enlargement of the liver, sometimes its soreness on palpation. As cirrhosis progresses, the liver becomes dense, lumpy, the abdomen increases in size due to the appearance of fluid in the abdominal cavity (ascites), on the anterior and lateral surfaces of the abdomen are seen dilated veins, enlarged spleen, swollen legs.

The combination of these signs in combination with bleeding from varices of the esophagus, stomach and intestine refers to the syndrome of portal hypertension.

Often noted decrease in body weight (weight loss) of the patient, sometimes reaching the degree of cachexia.

Mandatory laboratory tests include:

- serologic markers of hepatitis B viruses (HBeAg; antibodies to HBeAg; HBV DNA), hepatitis C (antibodies to HCV; HCV RNA), hepatitis D (antibodies to HDV; HDV RNA);
- clinical blood analysis with platelet and reticulocyte counting, prothrombin index;
- determination of the concentration of aminotransferases (ALT, AST), alkaline phosphatase, gamma glutamyltranspeptidase, bilirubin, creatinine, cholesterol, total protein and protein fractions, immunoglobulins, potassium, sodium, iron in the blood;
- general urinalysis;
- coprogram, fecal occult blood test;
- blood group, Rh factor.

Instrumental studies include: Ultrasound of the liver, gallbladder, pancreas, spleen and vessels of the portal system, esophagogastroduodenoscopy. Diagnosis of chronic liver disease,

including LC, is facilitated by percutaneous liver biopsy with subsequent histologic examination of the biopsy.

Analyzing the results of serological, biochemical and instrumental tests and verifying LC is the responsibility of the physician. It is useful for the nurse to know that the results of serologic studies can establish or exclude a viral origin of LC, the severity of which is assessed by biochemical blood tests - the degree of increase in bilirubin concentration and decrease in prothrombin index and albumin levels. Increased concentration of amino transferases (ALT and AST) in the blood reflects the activity of the process of destruction (necrosis) of liver cells.

Principles of treatment. Modern treatment of LC provides for slowing the progression of the disease, reduction or elimination of clinical manifestations of complications - hepatic encephalopathy, hemorrhagic syndrome, portal hypertension and its consequences (ascites, bleeding from esophageal, gastric and intestinal veins).

Inpatient treatment is indicated for patients with CP in the decompensation stage: the presence of hepatic encephalopathy, ascites and its complications, marked hemorrhagic syndrome, renal failure. Patients need a gentle (bed rest or bed rest) regimen with restriction of physical activity and psychoemotional stress.

Therapeutic nutrition prevents adverse reactions to drugs or reduces their severity, creates conditions for optimal digestion, contributes to the reduction or disappearance of discomfort and heaviness in the upper half of the abdomen, flatulence, dyspeptic disorders. Meals should be frequent (4-5 times a day), small portions, mechanically and chemically sparing, containing a sufficient number of proteins and carbohydrates with a limited amount of fats. The appearance of signs of hepatic encephalopathy is an indication to limit the protein content in the daily diet to 30-40 g. If fluid retention in the body (ascites, edema of the legs) limit its intake to 900-1000 ml with a simultaneous reduction of table salt in the diet to 2 g per day.

Regardless of the severity of LC cut off the consumption of fatty, fried, spicy dishes, smoked meats, offal, carbonated drinks. Completely exclude alcoholic beverages. Increase the content of cereal products, vegetables and fruits with a sufficient amount of lean beef, veal, fish and poultry. Patients need additional intake of vitamins B1, B2, B6, B12, B12, E, K, ascorbic and folic acids.

Prevention. Prevention of liver cirrhosis involves two groups of measures:

1) early detection and treatment of chronic hepatitis predominantly viral, alcoholic, toxic, including drug origin;

2) prevention of LC progression and the occurrence of its complications - hepatic insufficiency and portal hypertension.

An important role is played by identification of chronic viral hepatitis with the help of serologic (virus markers), biochemical and instrumental studies in risk groups: patients requiring blood transfusion and hemodialysis units, medical workers, drug addicts, prisoners and male homosexuals. Similar tests contribute to the diagnosis of chronic alcoholic hepatitis in people who abuse alcohol. People who come into contact with toxic substances at work or in the home or who use hepatotoxic medicines should be examined.

Prevention of progression of LC with constant dispensary monitoring of the patient in outpatient polyclinic conditions contribute to the patient's compliance with proper (therapeutic) nutrition, long-term use of drugs (polyenzyme, diuretics, hepatoprotectors,

etc.), the exclusion of alcohol consumption and smoking, heavy physical exertion, prevention of constipation.

It is necessary to teach the patient simple methods of self-examination, the results of which at intervals of 2-3 days he enters into a notebook (diary of self-examination). The patient records the following information in the diary: date, body weight, frequency and color of feces. An increase in body weight for 3-4 days by more than 1 kg indicates internal edema (fluid retention). The presence of feces of black color is possible with bleeding in the esophagus, stomach, or upper intestines. Appearance of any of these signs requires urgent appeal to the patient to a doctor. When observing the patient at home, the nurse will carry out a number of nursing interventions as outlined above.

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