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HEPATOSIS

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Hepatosis is a liver disease characterized by dystrophy and necrosis of hepatocytes; it can be hereditary and acquired. A large group of hereditary hepatoses are metabolic liver diseases that occur due to metabolic disorders:

- proteins and amino acids – cystinosis and aminoaciduria, or Debre-de Toni-Fanconi syndrome;
- fats - hereditary lipidoses;
- carbohydrates – glycogenoses;
- pigments – hereditary pigment hepatosis, porphyria;
- minerals – hemochromatosis, hepatocerebral dystrophy, or Wilson-Konovalov disease.

Many of the hereditary hepatoses are accumulation diseases and end in the development of liver cirrhosis. Acquired hepatoses, depending on the nature of the course, can be acute or chronic. The most important among acute hepatoses is toxic dystrophy, or progressive massive necrosis of the liver, among chronic ones - fatty hepatosis.

Massive progressive liver necrosis Massive progressive liver necrosis is an acute, less commonly chronic disease characterized by progressive massive liver necrosis and liver failure.

Etiology and pathogenesis. Massive liver necrosis often occurs in cases of intoxication:

- exogenous – poisoning by poor-quality food products, mushrooms, phosphorus, arsenic, etc.;
-endogenous – toxicosis of pregnancy, thyrotoxicosis.

It develops in viral hepatitis as an expression of its malignant (fulminant) form. In pathogenesis, the main significance is given to the hepatotoxic effect of the poison (virus). Allergic and autoallergic factors play a certain role.

Pathological anatomy. Liver changes vary at different stages of the disease, which usually lasts about 3 weeks.

In the first days, the liver is somewhat enlarged, dense or flabby, and acquires a bright yellow color on the surface and on the cut. Then it progressively decreases, becomes flabby, and the capsule becomes wrinkled; on the cut, the liver tissue is gray, clay-like.

Microscopically, in the first days, fatty degeneration of hepatocytes in the center of the lobules is noted, quickly followed by their necrosis and autolytic decay with the formation of fat-protein detritus, in which crystals of leucine and tyrosine are found.

By the end of the 2nd week of the disease, necrosis affects all parts of the lobules, and only on their periphery remains a thin layer of hepatocytes in a state of fatty degeneration. These liver changes are characteristic of the yellow degeneration stage. In

the 3rd week of the disease, the liver continues to shrink and becomes red. These changes are associated with phagocytosis and resorption of the fatty protein detritus of the liver lobules; as a result, the reticular stroma with sharply dilated, blood-filled sinusoids is exposed; cells are preserved only on the periphery of the lobules. Liver changes in the 3rd week of the disease are characteristic of the red degeneration stage.

In massive liver necrosis, jaundice, hyperplasia of the periportal lymph nodes and spleen, multiple hemorrhages on the skin, mucous and serous membranes, in the lungs, necrosis of the epithelium of the renal tubules, dystrophy and necrobiosis in the pancreas, myocardium, and central nervous system are observed. In progressive liver necrosis, the patient usually dies from acute liver or kidney (hepatorenal syndrome) failure. The outcome of progressive massive necrosis may be postnecrotic cirrhosis of the liver. Chronic toxic liver dystrophy is rarely observed in relapses of the disease. Postnecrotic cirrhosis of the liver develops in the end.

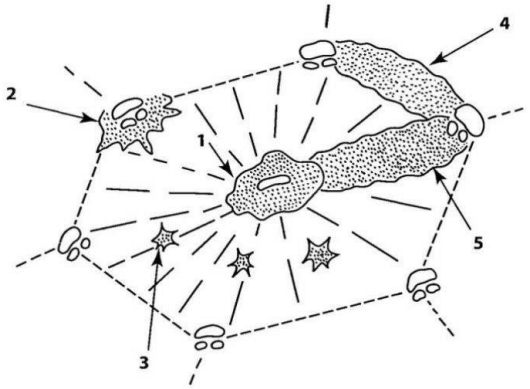

Fig. 2. Variants of hepatocyte necrosis:

1 - centrilobular; 2 - periportal; 3 - focal (spotted); 4 - bridging porto-portal; 5 - bridging portocentral

Fatty hepatosis

Fatty hepatosis (fatty liver dystrophy, liver steatosis) is a chronic disease characterized by increased accumulation of fat in hepatocytes. Etiology and pathogenesis. Causes of fatty hepatosis:

- toxic effects on the liver (alcohol, insecticides, some medications);
- endocrine-metabolic disorders (diabetes mellitus, general obesity);
- nutritional disorders (deficiency of lipotropic factors, kwashiorkor, excessive consumption of fats and carbohydrates);
- hypoxia (cardiovascular and pulmonary failure, anemia).

Chronic alcohol intoxication plays the main role in the development of fatty hepatosis. Alcoholic steatosis of the liver develops. Direct action of ethanol on the liver has been established. Direct oxidation becomes the most adequate under these conditions. As a result, the synthesis of triglycerides in the liver is enhanced, the mobilization of fatty acids from fat depots is increased, and the use of fatty acids in the liver is reduced. Triglycerides are formed - inert compounds that do not interfere with the synthetic

processes occurring in hepatocytes. This determines the duration of liver steatosis in alcohol intoxication.

Pathological anatomy. The liver in steatosis is large, yellow or red-brown, its surface is smooth. Fat related to triglycerides is determined in hepatocytes. Obesity of hepatocytes can be dust-like, small- and large-droplet (Fig. 3). A drop of lipids pushes relatively intact organelles to the periphery of the cell, which becomes signet-ring-shaped. Fatty infiltration can involve single hepatocytes – disseminated obesity, groups of hepatocytes – zonal obesity, or the entire liver parenchyma – diffuse obesity. In some cases (intoxication, hypoxia), obesity of liver cells develops predominantly centrilobularly, in others (protein-vitamin deficiency, general obesity) – predominantly periportally. With pronounced fatty infiltration, liver cells die, fat drops merge and form extracellularly located fat cysts, around which a cellular reaction occurs, connective tissue grows.

Fig. 4. Fatty hepatosis, large droplet obesity of hepatocytes

There are three stages of fatty hepatosis:

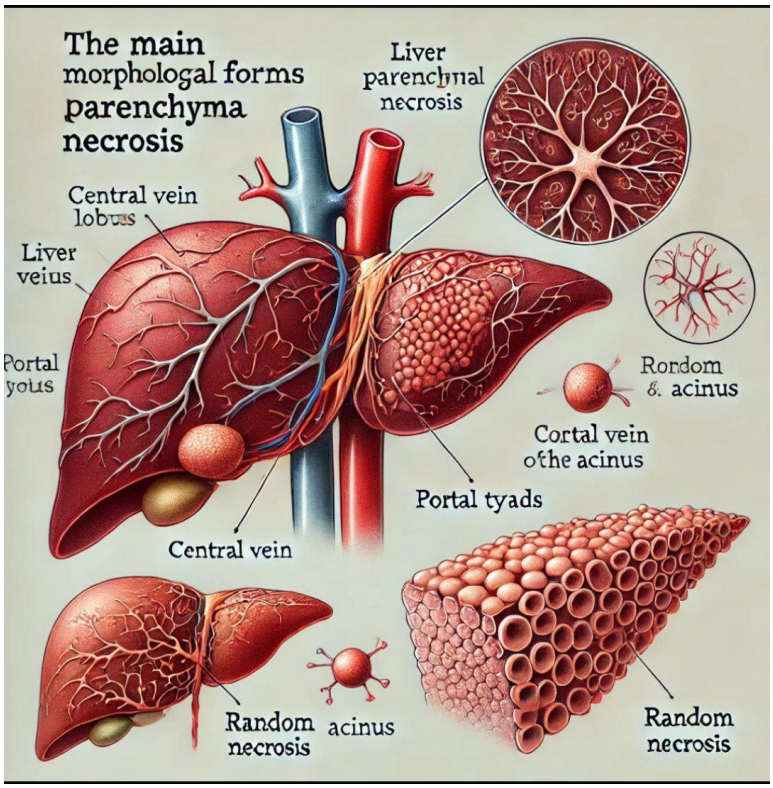

Fig. 3. Fatty hepatosis, large droplet obesity of hepatocytes

There are three stages of fatty hepatosis:

- simple obesity in which the destruction of hepatocytes is not expressed, the mesenchymal-cellular reaction is absent;

- obesity combined with hepatocyte necrobiosis and mesenchymal cell reaction;

- obesity with the beginning of the reorganization of the lobular structure of the liver. The third stage of liver steatosis is irreversible - the precirrhotic stage. The evolution of fatty hepatosis into portal cirrhosis has been traced in repeated liver biopsies and proven experimentally. In cirrhosis against the background of steatosis, fats disappear from hepatocytes. With liver steatosis, hepatic jaundice is possible. In some cases, fatty hepatosis is combined with chronic pancreatitis, neuritis.

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