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USE OF NON-INVASIVE METHODS IN THE DIAGNOSIS OF FATTY HEPATOSIS OF THE LIVER

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Abstract: The results of the first screening study of non-alcoholic fatty liver disease incidence among Andijan residents using the elastometry method are presented. The issues of non-invasive diagnostics and treatment of this pathology are considered.

Keywords: non-alcoholic fatty liver disease screening, non-alcoholic steatohepatitis, biopsy for fatty liver disease.

INTRODUCTION

In recent years, non-alcoholic fatty liver disease (NAFLD), which includes chronic clinical and morphological changes in the liver parenchyma, manifested by steatosis (fatty degeneration), non-alcoholic steatohepatitis (NASH) and liver cirrhosis (LC), has attracted particular interest from hepatologists, endocrinologists and cardiologists around the world.

NAFLD is one of the most important medical and social problems, as it leads to deterioration of quality of life, disability and increased mortality in this category of patients. Thus, if liver steatosis in most cases is benign and asymptomatic, then NASH is characterized by damage to hepatocytes, inflammation and development of connective tissue, which can lead to the formation of cirrhosis, hepatocellular insufficiency and liver cancer.

MATERIALS AND METHODS

The true prevalence of NAFLD is unknown. In North America, Europe and Japan, the incidence of NAFLD reaches 10–40%, with NASH verified in 1.2–4.8% of cases. In the USA, NAFLD accounts for 69% of chronic diffuse liver diseases [1].

The incidence of NASH among patients with long-term elevated serum aminotransferase levels of unspecified genesis is 20–32%.

In overweight patients, various forms of NAFLD are detected in 58–74%, and in morbid obesity – in 95% of patients. Similar data for Uzbekistan as a whole are not available, but it can be assumed that out of more than 2 million patients with type 2 diabetes mellitus (T2DM), 2/3 have NAFLD.

RESULTS AND DISCUSSION

It has been established that the basis for the development of NAFLD is pathological intra- and/or extracellular deposition of fat droplets with a triglyceride content of more than 10%

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of the dry liver mass [2]. The latest ideas about the pathogenesis of NAFLD allow us to distinguish 2 stages of its development (the "two-hits model"):

• Stage I (first "hit") – entry of lipids into hepatocytes – formation of liver steatosis (the "first hit" theory);

• Stage II (second "hit") – development of inflammation – formation of steatohepatitis (the "second hit" theory).

Liver steatosis is an excessive accumulation of lipids in hepatocytes. Fatty liver is said to be present if the amount of lipids exceeds 5% of its mass. In clinical practice, steatosis is usually determined by morphological examination of liver tissue in the form of fat droplets located in hepatocytes [3]. Thus, when diagnosing liver steatosis, the presence of morphological signs of fatty liver is primarily taken into account, and to a lesser extent, clinical manifestations. Liver steatosis usually indicates a violation of the regulation of the synthesis and secretion of triglycerides, the specific mechanisms of which have not been sufficiently studied to date. The factors that act as trigger mechanisms that initiate these disorders have not been determined. The following are discussed as possible pathophysiological mechanisms: excessive intake of fatty acids from adipose tissue or food origin in the liver; increased endogenous synthesis of fatty acids; decreased mitochondrial β-oxidation of fat; impaired inclusion and excretion of triglycerides in the composition of very low density lipoproteins [4].

The clinical picture of NAFLD in most cases is characterized by an asymptomatic course, therefore this disease is most often detected accidentally during laboratory and instrumental examination. Patients with liver steatosis, as a rule, have no complaints. Symptoms of NASH are nonspecific (increased fatigue, weakness, aching pain in the right hypochondrium without a clear connection with food) and do not correlate with its activity. At the stage of cirrhosis, symptoms appear that indicate the development of hepatocellular insufficiency and portal hypertension: an increase in the size of the abdomen, edema, moderate jaundice, increased bleeding, etc. It should be noted that a distinctive feature of NAFLD, in contrast to other chronic diffuse liver diseases, is the absence of skin itching.

Currently, the ICD-10 list of diseases does not have a single code that reflects the completeness of the diagnosis of NAFLD, so it is advisable to use one of the codes:

K 73.0 - chronic persistent hepatitis, not classified in other categories.

K 73.9 - chronic hepatitis, unspecified.

K 76.0 - fatty degeneration of the liver, not classified in other categories.

K 74.6 - other and unspecified cirrhosis of the liver.

When CP is formed, laboratory signs of decreased protein-synthetic function of the liver are revealed: decreased levels of total protein, albumin, cholesterol, cholinesterase and prothrombin index. The presence of hypoalbuminemia in patients with NASH without transformation into CP is possible in patients with diabetic nephropathy.

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CONCLUSION

Thus, the prescription of a dietary regimen, insulin sensitizers and essential phospholipids to a patient with NASH led to a decrease in fibrotic changes and the degree of steatosis, as well as to a regression of the necroinflammatory reaction in the liver tissue, which significantly improved the patient's quality of life and life prognosis.

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