

NON-ALCOHOLIC FATTY LIVER DISEASE (NAFLD)

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Abstract: Non-alcoholic fatty liver disease (NAFLD) is a chronic non-infectious disease in which structural changes occur in the liver tissue. The main manifestation of the pathology is excessive deposition of fats (mainly triglycerides) in liver cells - hepatocytes. As the process progresses, inflammation (steatohepatitis), formation of fibrous tissue, and in severe cases - cirrhosis may develop. In everyday life, NAFLD is often called "fatty liver" or fatty hepatosis. Although the disease usually proceeds relatively calmly and without pronounced symptoms, its presence is closely associated with metabolic disorders, which significantly increases the risk of cardiovascular complications. This, in turn, negatively affects the general condition of the patient, reduces the duration and quality of life.

Key words: hepatocyte, steatohepatitis, hepatosis, triglycerides, steatosis, insulin resistance.

Introduction.

Fatty deposits in liver cells — hepatocytes — lead to disruption of the integrity of their membranes, failures in cellular metabolism processes and oxidation-reduction reactions. These pathological changes contribute to the destruction of hepatocytes. Subsequently, connective, scar (fibrous) tissue develops at the site of dead cells, which disrupts the normal function of the liver. This entails changes in biochemical parameters of the blood, increases the likelihood of developing metabolic syndrome and can lead to the formation of cirrhosis.

Non-alcoholic fatty liver disease can occur in people over 30 years of age, even if they do not abuse alcohol, but periodically consume it in small quantities. Those who prefer high-calorie and fatty foods, as well as lead a sedentary lifestyle, are especially at risk. Although most often the disease proceeds without pronounced symptoms, changes in the liver structure continue to increase, metabolic processes are disrupted, which can be detected during laboratory and instrumental examinations. The prevalence of this pathology, according to world statistics, is approximately 20%. At an early stage of the disease, there are no external manifestations. The patient may accidentally learn about the presence of pathology during a preventive examination or when visiting a doctor about another problem. Liver cells (hepatocytes) have a pronounced ability to recover, so liver function remains compensated for a long time even after the onset of NAFLD (non-alcoholic fatty liver disease). However, over time, the functional activity of the affected cells decreases, and characteristic signs of the disease begin to appear: a feeling of heaviness and discomfort in the right hypochondrium or epigastric region, nausea, belching, bitterness in the mouth, general weakness, fatigue, decreased performance, weight gain, an unexplained increase in body temperature to subfebrile numbers, vascular asterisks on the skin.

With pronounced progression of the disease and development of complications, yellowing of the skin, sclera and mucous membranes, skin itching and accumulation of fluid in the abdominal cavity (ascites) may be observed.

Purpose of the study. To assess the clinical and laboratory features of non-alcoholic fatty liver disease and determine the influence of risk factors (obesity, insulin resistance, metabolic syndrome) on the severity and progression of the disease.

Materials and methods

The study used clinical, laboratory, instrumental and anthropometric data. The following served as the material for analysis:

Fasting venous blood samples to determine biochemical parameters (alanine aminotransferase - ALT, aspartate aminotransferase - AST, gamma-glutamyl transferase - GGT, total bilirubin, glucose, cholesterol, triglycerides, high-density and low-density lipoproteins).

Anthropometric parameters, including body weight, height, waist circumference and body mass index (BMI), which were assessed to identify obesity and metabolic disorders.

Ultrasound examination of the liver (ultrasound), used to visualize the degree of liver steatosis. The study was conducted using a standard ultrasound machine in B-scan mode, according to the liver parenchyma echogenicity scale. Survey data collected using a questionnaire, including information on diet, lifestyle, physical activity level, presence of chronic diseases and medication intake.

Calculation of the HOMA-IR insulin resistance index based on the results of fasting glucose and insulin analysis.

Exclusion criteria were: presence of viral hepatitis, alcohol abuse, autoimmune and drug-induced liver damage. All patients were aware of the purpose of the study and gave voluntary consent to participate.

Results and discussion. This pathology develops mainly as a consequence of an unhealthy lifestyle and disorders of lipid metabolism in the body. Factors contributing to the development of the disease include: presence of abdominal obesity (waist circumference over 80 cm in women and over 94 cm in men); endocrine disorders, including hypothyroidism and Itsenko-Cushing syndrome; decreased sensitivity to glucose and type 2 diabetes mellitus; unbalanced diet; long-term parenteral nutrition, in which nutrients are administered intravenously; infectious liver lesions of both acute and chronic nature; congenital liver abnormalities and hereditary lipid metabolism disorders caused by enzyme deficiency; insufficient physical activity; high blood pressure; taking certain medications, such as amiodarone or corticosteroids.

An important role in the diagnosis of NAFLD is the collection of patient complaints, anamnesis, and assessment of possible risk factors for the disease. Before making this diagnosis, other liver diseases, especially those of viral etiology, should be excluded. To examine a patient with possible NAFLD, laboratory and instrumental diagnostic methods are used: general blood and urine tests; fibrosis degree test; coagulogram; biochemical blood test (ALT, AST, cholesterol, total and fractional bilirubin, alkaline phosphatase, sugar, protein fractions); blood test for infections; ultrasound of the abdominal cavity and liver; CT, MRI of the liver. The most important component of treatment for non-alcoholic fatty liver disease is lifestyle modification: physical activity for at least 150 minutes a week, normalization of body weight, refusal of alcoholic beverages and a number of medications, balanced

nutrition, compliance with the work and rest regime. The patient should exclude fatty meat, fish, canned food, smoked meats, easily digestible carbohydrates, cooking fats, chocolate and ice cream, cocoa, black coffee, alcohol from the diet. Preference in nutrition should be given to lean meats, fish, vegetable and fruit dishes, complex carbohydrates (cereals, pasta made from durum wheat).

The optimal rate of weight loss for fatty hepatosis is 1 kg per 7 days. Rapid weight loss will lead to the development of steatosis, steatohepatitis, and acceleration of liver tissue fibrosis processes. A 10% reduction in body weight in obese individuals with non-alcoholic fatty liver disease will significantly improve the values of AST, ALT and other liver enzymes, as well as histological indices.

Drug treatment of this pathology includes:

drugs to overcome insulin resistance;

omega-3-PUFA, statins, fibrates to reduce the risk of cardiovascular complications;

hepatoprotectors.

If other methods of losing weight are ineffective, you can use the method of bariatric surgery - gastric bypass and banding.

Progression of liver failure may be an indication for orthotopic liver transplantation.

Patients suffering from fatty hepatosis should periodically (once every 1-6 months, depending on the condition) visit their doctor to conduct a minimum of examinations to control the course of the disease, timely detection of its complications and correction of therapy.

Conclusion.

Non-alcoholic fatty liver disease is a progressive disease associated with metabolic disorders and lifestyle. According to the results of a systematic review and meta-analysis, primary hypothyroidism is associated with an increased risk of metabolic-associated fatty liver disease, with an increased risk of both steatohepatitis and progressive fibrosis. Early detection, nutritional correction, physical activity and treatment of concomitant conditions play a key role in preventing complications and maintaining liver function.

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