NONALCOHOLIC FATTY LIVER DISEASE, MODERN CONSIDERATIONS

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Abstract
The relationship of obesity and diabetes to liver disease (1) (obese-diabetic liver disease) was first reported over three decades ago. In 1980, Ludwig and his collaborators (2) described the condition of a series of patients, who without having a significant history of alcohol consumption, showed histopathological changes that were indistinguishable from those occurring in alcoholic liver disease. This group coined the term "non alcoholic steatohepatitis" (NASH), which has been recognized since then as one of most frequent liver diseases in the world (3). Its potential progression to cirrhosis (4), liver insufficiency (5) and hepatocellular carcinoma (6, 7) has been identified. The real importance of NASH in Colombia is unknown, but it is increasingly the reason for general consultations, and increasingly leads to cirrhosis and liver transplants.

Keywords:liver disease, increasingly, cirrhosis, liver transplants.

Non-alcoholic fatty liver disease (NAFLD) is a chronic disease that occurs in people who do not consume more than the norm of alcohol, i.e. ethanol consumption does not exceed 40 g per day for men and 20 g for women, liver cells due to the accumulation of lipids, it combines clinical and morphological changes, morphologically manifested in the form of steatosis, steatohepatitis, fibrosis, cirrhosis [1,2,3,6].

To date, the concept of JNYoX is manifested by the following pathogenetically related changes of the liver:

• steatosis of the liver, excessive accumulation of triglycerides in the cytoplasm of hepatocytes (if it is more than 5% of the liver mass); Small fat bodies in hepatocytes (if the amount of fat increases to 2-3%) can be detected under a light microscope, and this pathological condition can be assessed as the beginning of liver steatosis [4,7,9].

• Nonalcoholic steatohepatitis (NASG), a chronic diffuse liver disease with necrotic-inflammatory processes leading to fibrosis;
• liver fibrosis, increase in connective tissue without changes in organ structure;
• Liver cirrhosis (JTs) is an irreversible replacement of liver parenchymal tissue with fibrous connective tissue, which is replaced by a special nodular anatomical structure[9,10,].

In the stage of steatosis, JNYoX is characterized by relatively safe and slow progression. But NASG is often not manifested for a long time, and if not treated adequately, it can progress to fibrosis and cirrhosis in 50% of cases. In the general population, there is evidence that one in three patients with NASG will progress to the stage of JTs .

JNYoX is a slowly progressive disease, and patients do not always develop cirrhosis. However, liver fibrosis has been observed in one fourth of patients with hepatic steatosis [9.11]. According to some authors, 10% of cases of hepatic steatosis will progress to NASG within ten years. In 5-25% of cases, NASG turns into liver cirrhosis. Approximately 10% of patients with NASG in the cirrhotic stage develop hepatocellular carcinoma (HCC) within ten years [2,8,11]. Notably, 60–80% of all cryptogenic liver cirrhosis results from JNYoX [17] and 10% of those referred for liver transplantation are due to NASG in the cirrhotic stage. JNYoX is the focus not only of general practitioners and gastroenterologists, but also of cardiologists, endocrinologists, and nephrologists, because JNYoX increases the risk of developing cardiovascular diseases, type 2 diabetes, and chronic kidney disease. [5.8.11,].

Scientific studies on the clinical symptoms and diagnosis of this disease have shown that The main feature of JNYoX is that it is often asymptomatic, and the disease is detected incidentally on the basis of laboratory or instrumental examinations conducted in patients with metabolic syndrome. NASG presents with nonspecific symptoms. Although these signs indicate liver damage, they do not help determine its severity. Most patients with NASG have asthenovegetative syndrome; sometimes there is a short or long-lasting feeling of heaviness under the right rib cage - dyskinetic syndrome. Itching, anorexia, appearance of complaints of dyspeptic syndrome, development of jaundice along with signs of portal hypertension indicate the transformation of NASG into cirrhosis [6].

During physical examination, hepatomegaly is detected in 50-75% of patients with JNYoX [5]. Additional diagnostic tests are performed when the following symptoms are present:

- asymptomatic increase in the amount of aminotransferases;
- presence of unexplained persistent hepatomegaly;
- hepatomegaly in X-ray examination;
- when excluding all other causes leading to hepatomegaly.

As a result of a large number of observations, forecasts indicating a high risk of JNYoX development with steatohepatitis and fibrosis have been identified:

- over 45 years old;
- female gender;
- TMI above 28 kg/m2;
- two or more times increase in ALT activity;
- TG level is more than 1.7 mmol/l;
- presence of arterial hypertension;
- type 2 diabetes;
- IR (NOMA-Sh) index is higher than 5.

The detection of more than two criteria indicates a high risk of liver fibrosis. It should be noted that there is a possibility of reverse development of NAS, NASG against the background of gradual decrease in body weight. However, rapid weight loss helps to progress from one stage to another.

Summary

Thus, JNYoX is a very common pathology, which requires investigation of the mechanisms of its pathogenesis and the search for non-invasive methods to detect and evaluate complex forms of JNYoX (steatohepatitis, fibrosis, cirrhosis). Understanding the multifactorial nature of JNYoX and the formation mechanisms of polymorbidity associated with it allows us to adequately assess its prognosis, determine the priority methods of pharmacological and non-pharmacological treatment.

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