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STUDYING THE CLINICAL AND LABORATORY COURSE OF NON-ALCOHOLIC FATTY LIVER DISEASE

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Relevance: Non-alcoholic fatty liver disease (NAFLD) is a pathological excessive deposition of fatty droplets in hepatocytes. The importance of NAFLD among gastrointestinal diseases has been growing for years. Currently, it accounts for 20-30% of liver disease. Despite the achievements of pharmacotherapy for this pathology, the main problem is the nonspecificity of clinical manifestations in the initial stage of NAFLD, leading to untimely treatment and progression of the disease.

Keywords: hepatocytes, steatosis, cardiovascular diseases, diabetes mellitus, magnetic resonance spectroscopy.

Introduction. In recent years, non-alcoholic fatty liver disease (NAFLD) has taken a leading position among chronic liver diseases and its prevalence continues to grow, which cannot but cause concern, since this pathology is associated with a high risk of developing cardiovascular diseases, type 2 diabetes mellitus (DM) and progression liver pathologies. NAFLD is the excess accumulation of fat in the liver associated with insulin resistance (IR) and is defined as the presence of steatosis in more than 5% of hepatocytes by histology or a fat proton density of >5.6% by proton magnetic resonance spectroscopy (PMRS). or quantifying the fat-to-water ratio by magnetic resonance imaging (MRI) (EASL-EASD-EASO, 2016). NAFLD includes two distinct pathological conditions with different prognoses: nonalcoholic steatosis and nonalcoholic steatohepatitis (NASH); the latter covers a wide range of

diseases of varying severity, including fibrosis, liver cirrhosis (LC) and hepatocellular carcinoma (HCC) [20]. The natural history of NAFLD is quite dichotomous: non-alcoholic steatosis is usually benign, whereas NASH can progress to cirrhosis, liver failure and liver cancer (Figure 1). Diagnosis of NAFLD requires a comprehensive assessment of the medical history, laboratory and instrumental studies, which makes it possible to exclude other liver diseases. When collecting anamnesis, it is important to assess alcohol consumption (AUDIT, CAGE questionnaires) to exclude alcoholic liver damage, but a combination of these nosologies is also possible. It is also necessary to exclude drug-induced liver damage (information about taking new medications over the past 6 months) and assess the presence of risk factors for the development of viral hepatitis (contact with blood, blood transfusions, visits to the dentist, tattoo parlor) with mandatory examination for markers of viral hepatitis. Information about hereditary diseases suggests the presence of secondary NAFLD; metabolic syndrome (MS) argues in favor of NAFLD. Early diagnosis of NAFLD is difficult because symptoms in patients are usually either absent or nonspecific and do not correspond to the severity of liver damage. Often, hepatic steatosis is diagnosed accidentally when examining a patient for another reason, in particular about arterial hypertension (AH), coronary heart disease (CHD), peripheral vascular diseases, obesity, type 2 diabetes, etc. The main nonspecific clinical symptoms of NAFLD include: asthenic syndrome (weakness, increased fatigue, and sleep disturbance), dyspeptic syndrome (flatulence, nausea, stool disorders), pain syndrome (dull pain and/or heaviness in the right hypochondrium), hepatomegaly and/or splenomegaly [7]. Upon examination, as a rule, signs of increased body weight or obesity are revealed. Objectively, in patients with steatosis and NASH, moderate painless enlargement of the liver is detected,its edge is rounded, the consistency is doughy, and with severe fibrosis the liver becomes dense. With the progression of the disease and the development of cirrhosis, symptoms of liver failure and/or portal hypertension appear: “liver signs”, ascites, edema, hemorrhagic syndrome, encephalopathy, etc. Data from a meta-analysis of the prevalence of NASH in the population presented in 2016 demonstrate that morphological signs are determined in 59.10% of patients with clinical symptoms of liver damage (weakness, hepatomegaly, changes in liver enzyme levels), but also in 6.67–29.85% of patients without clinical symptoms of liver damage [49]. The most characteristic laboratory manifestation of NAFLD is a slight or moderate increase (no more than 4–5 times the upper limit of normal) in the activity of serum transaminases (ALT, AST), which are markers of necrotic inflammatory and fibrotic changes in the liver. At the same time, in most patients with NAFLD, ALT activity predominates. However, biochemical blood test data do not always correlate with the severity of liver damage. According to research, increased activity of ALT and AST is observed in only 20% of patients with hepatic steatosis and in 70% of patients with NASH [6]. The AST/ALT ratio, the de Ritis coefficient, also has diagnostic significance; in liver fibrosis it exceeds 1 and increases with the progression of fibrosis. When carrying out differential diagnosis, it is important if this coefficient is more than 2 (indicates alcoholic liver disease) or above 4.5 (typical of Wilson’s disease) [7]. In the work of S. McPherson et al. showed a high predictive value of the AST/ALT ratio in detecting severe liver fibrosis (F≥3) with a sensitivity of 74% and specificity of 78%, AUROC 0.83, which allows using this coefficient as an independent predictor of severe liver fibrosis, as well as primary screening to exclude severe liver fibrosis [35, 39]. However, when assessing the results of the AST/ALT ratio, one should remember the age-related features of these indicators: with age, the ALT level gradually decreases, and the AST level remains stable, which can lead to a false increase in the de Ritis coefficient in patients without severe fibrosis [24]. In 30% of cases, there is an increase in GGTP activity (no more than 1.5–2 times), while in some patients this may be the only deviation in the biochemical blood test. In a third of patients with NAFLD, examination reveals an increase in alkaline phosphatase activity (does not exceed normal values by more than 2 times) and in approximately 20% of patients there is a moderate (1.5–2 times) increase in the content of total bilirubin due to the direct fraction [4] . As the disease progresses, laboratory signs characteristic of cirrhosis are observed: hypoalbuminemia, hypoprothrombinemia, hyperbilirubinemia,

thrombocytopenia, an increase in prothrombin time and international normalized ratio.Considering the bidirectional associations between NAFLD and the components of metabolic syndrome, attention should be paid when examining changes in the lipid and carbohydrate spectrum (hypertriglyceridemia, increased levels of low- and very low-density lipoproteins, decreased concentrations of high-density lipoproteins, impaired fasting glycemia/or impaired glucose tolerance, hyperinsulinemia, insulin resistance).

Purpose of work: To study the clinical and laboratory course of non-alcoholic fatty liver disease (NAFLD) in the Samarkand region.

Materials and methods of research: 45 patients with NAFLD will be examined to solve the assigned problems. The following research methods are used in the study: clinical, laboratory. The subjects of the study will be patients with NAFLD in the stage of steatosis and cholelithiasis. A general clinical examination is carried out according to a standard scheme, including identifying complaints, collecting anamnesis, and assessing the condition of organs and systems. All patients were re-examined in the clinic and hospital. A comprehensive examination includes: general blood test, urinalysis, bilirubin, ALT, AST, G-GT, hydroxide phosphatase, cholesterol, triglycerides, albumin.

Study results: Clinical and laboratory features of the pathology were studied for the first time in NAFLD patients with cholelithiasis. At the stage of steatosis, a comprehensive clinical and laboratory assessment of the characteristics of the course of NAFLD is carried out. Those examined had weakness (20%), decreased ability to work (18%), heaviness in the hypochondrium (29%), bitterness in the mouth (11%), nausea (7%). In a comprehensive study, the prevalence was studied, complaints, clinical features, and functional features of the hepatobiliary system, reflecting the main features of NAFLD in the steatosis stage, were analyzed and compared. The presence of concomitant pathology of gallstones in 100% of cases is accompanied by NAFLD, which justifies the need for adequate hepatoprotective therapy.

Conclusion: The results obtained can be used for early and differential diagnosis of NAFLD in the steatosis stage. A prognosis system will be developed to identify patients at risk of developing NAFLD and its transition to the development of liver fibrosis, which will allow timely correction of functional disorders of the hepatobiliary system and reduce the incidence of liver cirrhosis. The developed diagnostic and treatment methods make it possible to optimize and increase the effectiveness of treatment and reduce possible complications.

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