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NON-ALCOHOLIC FATTY LIVER DISEASE: THE PREVALENCE, ASSOCIATION AND IMPACT OF METABOLIC RISK FACTORS IN THE DEVELOPMENT AND PROGRESSION

Introduction

Non-alcoholic fatty liver disease (NAFLD) is currently one of the most common chronic liver diseases worldwide and represents a major public health problem. It is characterized by excessive fat accumulation in hepatocytes in the absence of significant alcohol consumption or other causes of liver disease. According to epidemiological studies, the global prevalence of NAFLD reaches approximately 25–30% of the adult population [1].

NAFLD encompasses a wide spectrum of pathological conditions ranging from simple hepatic steatosis to non-alcoholic steatohepatitis (NASH), liver fibrosis, cirrhosis, and hepatocellular carcinoma [2]. The disease is closely associated with metabolic disorders such as obesity, dyslipidemia, insulin resistance, and type 2 diabetes mellitus [3].

Metabolic syndrome plays a key role in the pathogenesis of NAFLD. Several studies have demonstrated that abnormalities in lipid metabolism, impaired glucose regulation, and arterial hypertension significantly increase the risk of hepatic steatosis and disease progression [4].

Early identification of metabolic risk factors associated with NAFLD is essential for timely diagnosis and prevention of disease progression. Ultrasound examination remains one of the most widely used non-invasive diagnostic methods for detecting hepatic steatosis in clinical practice [5].

Therefore, investigating metabolic risk factors associated with NAFLD in clinical practice is important for improving early diagnosis, identifying high-risk patients, and preventing the progression of liver disease and related metabolic complications.

Goal

To investigate the association between metabolic risk factors and the presence of NAFLD among hospitalized patients, and to evaluate the clinical, biochemical, and ultrasound characteristics that may contribute to the development of hepatic steatosis.

Material and methods of the research

A clinical observational study was conducted based on the analysis of medical records of 31 patients who were hospitalized in a therapeutic department of Gomel city clinical hospital № 3 during the period of November 2025 – March 2026. All patients underwent a comprehensive clinical examination, including medical history assessment, physical examination, and laboratory and instrumental diagnostic tests. Laboratory investigations included a complete blood count, biochemical blood analysis (alanine aminotransferase, aspartate aminotransferase, bilirubin, lipid profile, glucose levels), coagulation profile, and urinalysis. Instrumental diagnostics included ultrasound examination of abdominal organs, which was used as the primary method for detecting hepatic steatosis. The diagnosis of non-alcoholic fatty liver disease (NAFLD) was established based on characteristic ultrasound signs such as increased echogenicity of the liver parenchyma, reduced acoustic conductivity, and hepatomegaly, in the absence of significant alcohol consumption or viral hepatitis markers. For the purpose of analysis, patients were divided into two groups: patients with ultrasound-confirmed NAFLD and patients without signs of fatty liver disease. Descriptive statistical methods were used to analyze the collected data, and the results were expressed in absolute numbers and percentages.

The results of the research and their discussion

A total of 31 patients were included in the study. The examined population consisted of 17 women (54.8%) and 14 men (45.2%), with an age range from 22 to 68 years, with the majority of patients (approximately 58%) belonging to the middle-aged group between 40 and 60 years, which corresponds to the age category most commonly associated with metabolic disorders and the development of NAFLD.

Based on ultrasound examination of the abdominal organs, NAFLD was detected in 20 patients (64.5%), while 11 patients (35.5%) had no ultrasound signs of hepatic steatosis.

Among patients with NAFLD, ultrasound examination most commonly revealed increased echogenicity of the liver parenchyma in 20 patients (100%), reduced acoustic conductivity in 18 patients (90%), and hepatomegaly in 16 patients (80%). The size of the right hepatic lobe in patients with hepatomegaly ranged from 160 to 167 mm, exceeding the normal value of approximately 150 mm. These findings indicate structural changes in the liver associated with excessive lipid accumulation within hepatocytes and confirm the diagnostic significance of ultrasound imaging in detecting fatty liver disease.

Biochemical analysis of liver function demonstrated abnormalities in several patients. Elevated alanine aminotransferase (ALT) levels were observed in 9 patients (45%) with NAFLD, based on sex-specific reference values (men: 0–41 U/L; women: 0–31 U/L), elevated ALT was identified in 5 men and 4 women, with recorded values ranging from 65 to 77 U/L, indicating moderate hepatocellular injury. Mild elevation of aspartate aminotransferase (AST) was noted in 6 patients (30%). According to sex-specific reference limits (men: ≤38 U/L; women: ≤37 U/L), elevated AST levels were detected in 3 men and 3 women, with values ranging from 34 to 37 U/L, reflecting mild hepatocellular involvement. Increased levels of gamma-glutamyl transpeptidase (GGT) were identified in 7 patients (35%). Considering sex-specific norms (men: 8–61 U/L; women: 5–36 U/L), elevated GGT was observed in 4 men and 3 women, with values ranging from 90 to 109 U/L, indicating possible cholestatic or metabolic liver dysfunction.

Renal function markers remained largely within reference limits. Serum creatinine levels, taking into account sex-specific ranges (women: 53–97.2 μmol/L; men: 61.8–123.7 μmol/L), were elevated in 3 patients (9.7%), including 2 men and 1 woman, while the remaining patients demonstrated values within normal limits, suggesting generally preserved renal function.

Analysis of lipid metabolism revealed that dyslipidemia was present in 16 patients (51.6%). Among these patients, elevated low-density lipoprotein (LDL) cholesterol above 3.0 mmol/L was

detected in 12 patients (38.7%), while reduced high-density lipoprotein (HDL) cholesterol below 1.0 mmol/L was observed in 8 patients (25.8%). Elevated triglyceride levels above 1.7 mmol/L were detected in 7 patients (22.6%). These disturbances of lipid metabolism contribute significantly to hepatic fat accumulation through increased delivery of free fatty acids to hepatocytes and enhanced hepatic lipogenesis.

Disturbances of carbohydrate metabolism were also frequently identified. Impaired fasting glucose or type 2 diabetes mellitus was detected in 10 patients (32.3%). In several cases, fasting glucose levels ranged from 6.0 to 6.5 mmol/L, exceeding the normal range (<5.6 mmol/L), while glycated hemoglobin (HbA1c) levels in diabetic patients reached 9.1%, reflecting chronic hyperglycemia. These findings support the strong relationship between NAFLD and insulin resistance.

Additional metabolic and clinical risk factors were also observed. Overweight or increased body mass index (BMI >25 kg/m²) was present in 12 patients (38.7%), while arterial hypertension was documented in 9 patients (29.0%). Furthermore, endocrine disorders such as thyroid abnormalities were identified in 5 patients (16.1%). These comorbid conditions further contribute to metabolic imbalance and may accelerate the progression of hepatic steatosis.

The coexistence of multiple metabolic abnormalities among patients with NAFLD confirms the concept that fatty liver disease represents the hepatic manifestation of metabolic syndrome. Insulin resistance plays a central role in this process by increasing lipolysis in adipose tissue, leading to elevated levels of circulating free fatty acids that accumulate in the liver and promote triglyceride deposition within hepatocytes.

Conclusions

Overall, the high prevalence of metabolic risk factors observed in this study underscores their central role in the development of NAFLD. These findings highlight the critical importance of early identification and management of metabolic abnormalities through regular screening, including metabolic assessment and ultrasound monitoring. Early detection of NAFLD and its associated risk factors enables timely intervention through lifestyle modification and clinical management, which is essential to prevent progression to more severe stages such as non-alcoholic steatohepatitis, liver fibrosis, and cirrhosis. Therefore, prioritizing early metabolic screening and sustained monitoring strategies is key to reducing the long-term burden of liver disease and improving overall patient outcomes.

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