ETHIOPIAN INTERNATIONAL JOURNAL OF MULTIDISCIPLINARY RESEARCH

eISSN: 2349-5715 pISSN: 2349-5707

Volume: 12, Issue 05, May-2025 SJIF 2019: 4.702 2020: 4.737 2021: 5.071 2022: 4.919 2023: 6.980 2024: 7,662

METABOLIC ASSOCIATED FATTY LIVER DISEASE (MAFLD)

Palvanova Matlyuba Satvaldievna

Ferghana medical institute of public health

Annotation. Metabolic Associated Fatty Liver Disease (MAFLD) is a chronic non-infectious condition characterized by structural changes in liver tissue. The primary manifestation of this pathology is the excessive accumulation of fat (mainly triglycerides) in liver cells—hepatocytes. As the disease progresses, inflammation (steatohepatitis), the formation of fibrous tissue, and in severe cases, cirrhosis may develop. In everyday language, MAFLD is [1,2] often referred to as "fatty liver" or hepatic steatosis. Although the disease is usually asymptomatic or mild in its clinical presentation, it is closely associated with metabolic disorders, significantly increasing the risk of cardiovascular complications. This, in turn, negatively affects the patient's overall health, reducing both life expectancy and quality of life.

Keywords: hepatocyte, steatohepatitis, hepatosis, triglycerides, steatosis, insulin resistance.

INTRODUCTION

Fat accumulation in liver cells—hepatocytes—leads to disruption of membrane integrity, disturbances in cellular metabolism, and redox processes. These pathological changes contribute to the destruction of hepatocytes. As a result, connective and scar (fibrous) tissue forms in place of the damaged cells, impairing normal liver function. This leads to changes in blood biochemical parameters, increases the risk of developing metabolic syndrome, and may result in cirrhosis[3,4].

Metabolic Associated Fatty Liver Disease (MAFLD) can develop in individuals over the age of 30. Those who consume high-calorie, fatty foods and lead a sedentary lifestyle are especially at risk. Although the disease often progresses without noticeable symptoms, structural changes in the liver continue to intensify, and metabolic processes become increasingly impaired—these changes can be detected through laboratory and instrumental examinations. According to global statistics, the prevalence of this condition is approximately 20%.

Depending on the extent of fat accumulation in liver cells (hepatocytes), MAFLD is classified into four grades (stages):

- Grade 0: Small lipid droplets are found on the surface of individual hepatocytes.
- Grade I: Medium and large fat droplets accumulate in certain areas of the liver, located on the surface of functional cells.
- Grade II: Fat (triglycerides) penetrates deeply into liver cells and is evenly distributed inside them.
- Grade III: Lipids affect the entire organ diffusely, accumulating both within and between cells, forming fatty cysts.

In the early stages of the disease, there are no external signs. A patient may discover the condition incidentally during a routine check-up or while seeking care for an unrelated issue.

eISSN: 2349-5715 pISSN: 2349-5707

Volume: 12, Issue 05, May-2025 SJIF 2019: 4.702 2020: 4.737 2021: 5.071 2022: 4.919 2023: 6.980 2024: 7,662

Hepatocytes have a significant capacity for regeneration, so liver function often remains compensated even after the onset of MAFLD.

However, over time, the functional activity of affected cells declines, and characteristic symptoms begin to appear: a feeling of heaviness or discomfort in the right upper quadrant or epigastric region, nausea, belching, bitter taste in the mouth, general weakness, fatigue, decreased work capacity, weight gain, unexplained low-grade fever, and the appearance of vascular "spider" marks on the skin.

With significant disease progression and the development of complications, symptoms may include yellowing of the skin, sclerae, and mucous membranes, itching, and fluid accumulation in the abdominal cavity (ascites)[5,6,7].

Objective of the study: To assess the clinical and laboratory features of Metabolic Associated Fatty Liver Disease (MAFLD) and determine the impact of risk factors such as obesity, insulin resistance, and metabolic syndrome on the severity and progression of the disease.

MATERIALS AND METHODS.

The study utilized clinical, laboratory, instrumental, and anthropometric data. The following materials served as the basis for analysis:

Venous blood samples collected in a fasting state to determine biochemical parameters including alanine aminotransferase[8,9,10] (ALT), aspartate aminotransferase (AST), gamma-glutamyl transferase (GGT), total bilirubin, glucose, cholesterol, triglycerides, and high- and low-density lipoproteins.

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

Ultrasound examination of the liver was used to visualize the degree of hepatic steatosis. The procedure was performed with a standard B-mode ultrasound scanner and assessed according to the echogenicity scale of liver parenchyma.

Survey data collected via questionnaire covering dietary habits, lifestyle, physical activity levels, chronic conditions, and current medication use.

Calculation of the HOMA-IR index (Homeostasis Model Assessment of Insulin Resistance) based on fasting glucose and insulin levels.

Exclusion criteria included: the presence of viral hepatitis, alcohol abuse, autoimmune and drug-induced liver diseases. All patients were informed of the study's purpose and provided written informed consent to participate.

RESULTS AND DISCUSSION.

This pathology primarily develops as a result of an unhealthy lifestyle and disturbances in lipid metabolism. Contributing factors include: Abdominal obesity (waist circumference >80 cm in women and >94 cm in men); endocrine disorders, such as hypothyroidism and Cushing's syndrome; glucose intolerance and type 2 diabetes mellitus; unbalanced diets; prolonged parenteral nutrition, where nutrients are administered intravenously; infectious liver diseases, both acute and chronic; congenital

eISSN: 2349-5715 pISSN: 2349-5707

Volume: 12, Issue 05, May-2025 SJIF 2019: 4.702 2020: 4.737 2021: 5.071 2022: 4.919 2023: 6.980 2024: 7,662

liver anomalies and hereditary lipid metabolism disorders due to enzyme deficiencies; sedentary lifestyle; hypertension; use of certain medications, including amiodarone and corticosteroids. Epidemiological studies indicate a connection between primary hypothyroidism and MAFLD. However, the extent of the risk and disease severity remains unclear. [A systematic review and meta-analysis conducted by a group of researchers based on observational studies from PubMed, Scopus, and Web of Science databases (up to January 2024) supports this correlation

An essential aspect of diagnosing MAFLD includes collecting patient complaints and medical history, and assessing potential risk factors. Before confirming the diagnosis, other liver diseases—especially those of viral origin—must be ruled out[11,12]. Diagnostic evaluation includes laboratory and imaging techniques such as: complete blood count and urinalysis, fibrosis scoring, coagulogram, biochemical blood analysis (ALT, AST, cholesterol, total and fractionated bilirubin, alkaline phosphatase, glucose, protein fractions), blood screening for infections, abdominal and retroperitoneal ultrasound, CT and MRI of the liver.

The most important component in the treatment of Metabolic-Associated Fatty Liver Disease (MAFLD) is lifestyle modification. This includes engaging in physical activity for at least 150 minutes per week, achieving and maintaining a healthy body weight, abstaining from alcohol and certain medications, maintaining a balanced diet, and adhering to a proper work-rest schedule.

Patients should eliminate from their diet fatty meats and fish, canned foods, smoked products, easily digestible carbohydrates, cooking fats, chocolate and ice cream, cocoa, black coffee, and alcoholic beverages. Preference should be given to lean meats and fish, dishes made from vegetables and fruits, and complex carbohydrates such as whole grains and pasta made from durum wheat.

The optimal rate of weight loss for patients with hepatic steatosis is approximately 1 kg per week. Rapid weight reduction may lead to the development of steatosis, steatohepatitis, and acceleration of liver fibrosis. A 10% weight reduction in obese patients with non-alcoholic fatty liver disease significantly improves levels of liver enzymes such as AST, ALT, and others, as well as histological indicators.

Pharmacological treatment of this pathology includes:

- Agents to overcome insulin resistance;
- Omega-3 polyunsaturated fatty acids (PUFAs), statins, and fibrates to reduce the risk of cardiovascular complications;
- Hepatoprotective agents.

If conservative weight loss methods are ineffective, bariatric surgery (such as gastric bypass or banding) may be considered. Progression to liver failure can be an indication for orthotopic liver transplantation.

To reduce the risk of developing non-alcoholic fatty liver disease, individuals should:

- Maintain an active lifestyle and avoid physical inactivity;
- Control body weight and keep it within normal limits to prevent obesity;

eISSN: 2349-5715 pISSN: 2349-5707

Volume: 12, Issue 05, May-2025 SJIF 2019: 4.702 2020: 4.737 2021: 5.071 2022: 4.919 2023: 6.980 2024: 7,662

- Follow a healthy diet (the Mediterranean diet is considered optimal);
- Undergo regular medical check-ups for early detection of cardiovascular and endocrine disorders.

Patients with hepatic steatosis should visit their healthcare provider regularly (every 1 to 6 months depending on their condition) for follow-up evaluations in order to monitor disease progression, detect complications early, and adjust therapy as needed.

CONCLUSION

Metabolic-Associated Fatty Liver Disease (MAFLD) is a progressive condition linked to metabolic disorders and lifestyle factors. According to the results of a systematic review and meta-analysis[11], primary hypothyroidism is associated with an increased risk of MAFLD, including a higher likelihood of developing steatohepatitis and progressive fibrosis. Early diagnosis, dietary correction, regular physical activity, and the management of comorbid conditions play a key role in preventing complications and preserving liver function.

REFERENCES:

1. Клинические рекомендации по диагностике и лечению жировой болезни печени Российского общества по изучению печени и Российской гастроэнтерологической ассоциации. Гепатология, журнал. № 2, 2016. С. 24-42.
2. Буеверов А.О., Богомолов П.О., Маевская М.В. Патогенетическое лечение неалкогольного стеатогепатита: обоснование, эффективность, безопасность // Терапевтический архив – 2007. – Т. 79. – № (8). – С. 1-4.
3. Лазебник Л.Б., Радченко В.Г., Голованова Е.В. и др. Неалкогольная жировая болезнь печени: клиника, диагностика, лечение // Экспериментальная и клиническая гастроэнтерология. — М., 2015. — № 7. — С. 85-96.
4. Mantovani A, Csermely A, Bilson J, et al Association between primary hypothyroidism and metabolic dysfunction-associated steatotic liver disease: an updated meta-analysis Gut Published Online First: 23 May 2024. doi: 10.1136/gutjnl-2024-332491.
5. Jurabaev A.A., Eshbaev E.A., Mamasaidov Zh.T. - Pathomorphological changes in necrotic enterocolitis of the lateral layer of the intestinal//New Day in Medicine 11(73)2024 964-970
6. Мамасаидов Ж. Т., Джурабаев А. А., Эркин А. Э. Некротик энтероколитда ингичка ичак шиллиқ қаватининг патоморфологияси //Журнал гуманитарных и естественных наук. – 2024. – №. 17. – С. 200-205.
7. Isaqova, N. R. (2022). Influence of constipation on anthropometric indicators of children. Science and Innovation, 1(8), 888-892.
8. Raxmatjonovna, I. N. (2023). Effects of colonic diseases on children's health. World bulletin of public health, 23, 101-103.
9. Del Campo JA, Gallego-Durán R, Gallego P, Grande L. Genetic and Epigenetic Regulation in Nonalcoholic Fatty Liver Disease (NAFLD). Int J Mol Sci. 2018 Mar 19;19(3) [PMC free article] [PubMed]
10. Basaranoglu M, Neuschwander-Tetri BA. Nonalcoholic Fatty Liver Disease: Clinical Features and Pathogenesis. Gastroenterol Hepatol (N Y). 2006 Apr;2(4):282-291. [PMC free article] [PubMed]

eISSN: 2349-5715 pISSN: 2349-5707

Volume: 12, Issue 05, May-2025 SJIF 2019: 4.702 2020: 4.737 2021: 5.071 2022: 4.919 2023: 6.980 2024: 7,662

11. Lee YH, Cho Y, Lee BW, Park CY, Lee DH, Cha BS, Rhee EJ. Nonalcoholic Fatty Liver Disease in Diabetes. Part I: Epidemiology and Diagnosis. Diabetes Metab J. 2019 Feb;43(1):31-45. [PMC free article] [PubMed]