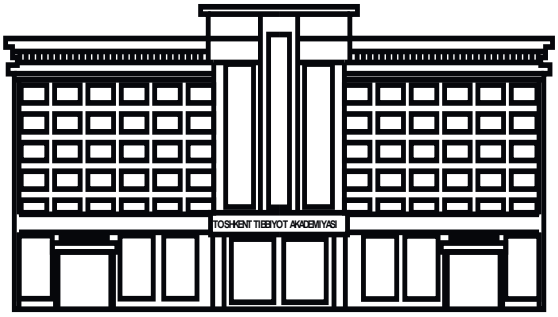
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CURRENT CONCEPTS OF COMORBID CONDITIONS IN METABOLICALLY ASSOCIATED FATTY LIVER DISEASE

Parpibaeva D.A., Buvamukhamedova N.T.

СОВРЕМЕННЫЕ ПРЕДСТАВЛЕНИЯ О КОМОРБИДНЫХ СОСТОЯНИЯХ ПРИ МЕТАБОЛИЧЕСКИ АССОЦИИРОВАННОЙ ЖИРОВОЙ БОЛЕЗНИ ПЕЧЕНИ

Парпибаева Д.А., Бувамухамедова Н.Т.

METABOLIK ASSOTSIRLANGAN JIGAR YOG‘ KASALLIGIDAGI KOMORBID HOLATLAR HAQIDA ZAMONAVIY TASAVVURLAR

Parpibaeva D.A., Buvamuxamedova N.T.

Ташкентский государственный медицинский университет

Проанализированы современные научные данные о роли метаболических нарушений в развитии и прогрессировании метаболически ассоциированной жировой болезни печени, а также об оценке существующих подходов к их коррекции. Показано, что дислипидемия, инсулинорезистентность и ожирение являются не только ведущими факторами риска формирования метаболически ассоциированной жировой болезни печени, но и способствуют повышению сердечно-сосудистой заболеваемости и смертности у данной категории пациентов. Рассмотрены современные стратегии немедикаментозной и медикаментозной терапии, направленные на коррекцию метаболических нарушений и снижение риска прогрессирования заболевания.

Ключевые слова: *метаболически ассоциированная жировая болезнь печени, метаболически ассоциированный стеатогепатит, дислипидемия, кардиометаболические риски, сахарный диабет 2-го типа, ожирение.*

Jigarning metabolik yog‘ kasalligining rivojlanishi va avj olishida metabolik buzilishlarning roli, shuningdek, ularni tuzatish bo‘yicha mavjud yondashuvlarni baholash bo‘yicha zamonaviy ilmiy ma’lumotlar tahlil qilingan.

Dislipidemiya, insulin qarshiligi va semirish nafaqat metabolik jihatdan bog’liq yog’li jigar kasalligining rivojlanishi uchun asosiy xavf omillari, balki ushbu bemorlar populyatsiyasida yurak-qon tomir kasalliklari va o’limning oshishiga ham hissa qo’shishi ko’rsatilgan. Metabolik buzilishlarni tuzatish va kasallikning rivojlanish xavfini kamaytirishga qaratilgan hozirgi dori-darmonsiz va dori-darmonli terapiya strategiyalari muhokama qilinadi.

Kalit so’zlar*: metabolik assotsirlangan jigar yog‘ kasalligi, metabolik assotsirlangan steatogepatit, dislipidemiya, kardiometabolik xavflar, 2-tir qandli diabet, semizlik.*

Nonalcoholic fatty liver disease (NAFLD) is a chronic hepatic disorder characterized by excessive lipid accumulation in more than 5% of hepatocytes (hepatic steatosis) in the absence of secondary causes of fat deposition, such as significant alcohol consumption, medication-induced liver injury, prolonged starvation, hereditary monogenic disorders, and other identifiable etiologies. NAFLD comprises a broad pathological continuum ranging from isolated hepatic steatosis to nonalcoholic steatohepatitis (NASH) and ultimately liver cirrhosis. Simple steatosis and NASH are considered distinct pathological phenotypes associated with differing clinical outcomes and prognostic implications [1,25].

Over recent decades, the prevalence of metabolically associated fatty liver disease (MAFLD) has increased substantially and is currently estimated to affect approximately 25% of the adult population worldwide. The disease has now reached pandemic-level prevalence and is recognized as one of the most common chronic liver disorders globally. Data from large-scale epidemiological studies conducted in the United States demonstrate that MAFLD represents the leading chronic liver disease among Caucasian populations and the second most common etiological factor contributing to liver cirrhosis [2,22].

Dyslipidemia is regarded as one of the major pathogenic contributors to MAFLD development. Consequently, the clinical management of affected patients extends beyond the field of gastroenterology and requires multidisciplinary involvement from internists and cardiologists. The natural progression of MAFLD generally includes several sequential stages, beginning with hepatic steatosis, which remains stable in the majority of patients, followed by metabolic dysfunction associated steatohepatitis (MASH), which demonstrates a stable course in approximately 65-75% of cases. In approximately 25-35% of patients, metabolic dysfunction-associated steatohepatitis (MASH) progresses to liver fibrosis, while cirrhosis develops in 9-20% of cases. Furthermore, nearly 10% of individuals with MAFLD may develop the terminal stage of the disease – hepatocellular carcinoma (HCC) – within a period of seven years.

A revised classification system for MAFLD has recently been proposed by the American Association for the Study of Liver Diseases, European Association for the Study of the Liver, and Latin American Association for the Study of the Liver, with contributions from international experts in hepatology, gastroenterology, endocrinology, pediatrics, pathology, obesity, and public health. The updated nomenclature was developed using the Delphi consensus methodology and published in 2023 in the statement entitled A multisociety Delphi consensus statement on new fatty liver disease nomenclature [3,18,24].

The consensus endorsed the umbrella term “steatotic liver disease” (SLD), under which several distinct clinical categories are recognized, including:

- metabolic dysfunction-associated steatotic liver disease (MASLD);
- alcohol-associated steatotic liver disease (AASLD);
- metabolic dysfunction and alcohol-related steatotic liver disease (MetALD);
- steatotic liver disease of specific etiology;

- cryptogenic steatotic liver disease.

Most international experts agreed that incorporating the term "metabolic dysfunction" into the disease nomenclature would improve understanding of the condition among both healthcare professionals and patients. In addition, 95% of respondents supported retaining the term metabolic dysfunction associated steatohepatitis because of its important prognostic value. Resolution of MASH remains a principal therapeutic objective in clinical practice and serves as a major endpoint in clinical trials evaluating treatment efficacy. The updated classification also introduced the concept of metabolic dysfunction-associated steatohepatitis combined with excessive alcohol consumption (MetABP) [4,19].

Russian researchers have evaluated the updated fatty liver disease nomenclature and discussed the feasibility of its implementation within the healthcare system of the Russian Federation. Based on these discussions, several recommendations were proposed: the optimized diagnostic criteria for NAFLD should be incorporated into routine clinical practice and included in the revised national clinical guidelines scheduled for publication in 2024. At the same time, the conventional terms "NAFLD" and "NASH" are recommended for use in official medical documentation because of the continued application of the ICD-10 classification system. For scientific and academic purposes, however, the updated terminology may be adopted following standardized and approved Russian translations. Accordingly, the term MAFLD may replace NAFLD, while metabolically associated steatohepatitis (MASH) may be used instead of NASH.

The main new criteria of the MAJBP include:

The revised diagnostic criteria for MAFLD require evidence of hepatic steatosis identified either by imaging techniques (including ultrasonography and other visualization methods) or by histological examination of liver biopsy specimens, in combination with at least one cardiometabolic risk factor.

The principal cardiometabolic risk factors associated with the development of MAFLD (currently termed NAFLD in existing classifications) include the following:

- body mass index (BMI) >25 kg/m^2 in Caucasian populations or >23 kg/m^2 in Asian populations, waist circumference >94 cm in men and >80 cm in women, or equivalent ethnicity-specific indicators;

- fasting plasma glucose >5.6 mmol/L, postprandial glucose >7.8 mmol/L, glycated hemoglobin (HbA1c) >5.7%, established type 2 diabetes mellitus, or ongoing antidiabetic therapy;

- arterial blood pressure ≥130/80 mmHg or current antihypertensive treatment;

- plasma triglyceride concentration ≥1.70 mmol/L or the use of lipid-lowering therapy;

- reduced plasma high-density lipoprotein (HDL) cholesterol levels (<1.0 mmol/L in men and <1.3 mmol/L in women) or treatment with lipid-lowering agents [5,16].

Association between MAFLD and Cardiovascular Disease in High-Risk Patients

In addition to its progressive hepatic manifestations, metabolically associated fatty liver disease is closely linked to cardiovascular disease (CVD). Cardiovascular complications represent one of the principal causes of adverse clinical outcomes and mortality among patients with MAFLD, ranking second only to liver cirrhosis as a cause of death and constituting the leading non-hepatic cause of mortality in this population. These findings emphasize the important role of cardiovascular pathology in determining both the prognosis and natural progression of MAFLD. Nevertheless, considerable uncertainty persists regarding the precise mechanisms underlying the association between MAFLD and cardiovascular disease, as well as the overall magnitude of this relationship. From an epidemiological standpoint, MAFLD and CVD share numerous common risk factors, particularly unhealthy lifestyle patterns and metabolic dysfunction. Multiple previous investigations have consistently demonstrated a significant association between MAFLD and an increased risk of several major cardiovascular conditions, including myocardial infarction (MI), ischemic stroke (IS), atrial fibrillation (AF), and heart failure (HF). Although the pathophysiological basis of this relationship has not been fully elucidated, it is believed to involve complex bidirectional interactions among several mechanisms, including endothelial dysfunction, chronic vascular inflammation, and disturbances in glucose and lipid metabolism.

Understanding the contribution of MAFLD to the development of CVD is essential for the design of targeted preventive and therapeutic strategies aimed at reducing cardiovascular morbidity and improving the overall prognosis of patients with MAFLD.

A systematic review and meta-analysis demonstrated that individuals with MAFLD have a significantly elevated risk of myocardial MI, IS, AF, and HF compared with individuals without MAFLD. Although moderate-to-high heterogeneity was identified across the analyzed studies, the associations remained statistically significant in approximately 95% of analyses for all outcomes except MI. Meta-regression analyses further suggested that mean patient age and male sex may act as important modifiers of the association between MAFLD and MI, whereas study design influenced the observed risks of both MI and IS among patients with MAFLD [6,20].

MAFLD represents a component of a broader spectrum of metabolic dysfunction and is considered a contributing factor to the development of an atherogenic lipid profile, endothelial dysfunction, and oxidative stress. The severity and stage of MAFLD appear to directly influence the magnitude of these pathological processes. Patients with MAFLD frequently present with chronic systemic inflammation in combination with overweight or obesity, all of which may substantially increase the risk of cardiovascular complications, particularly myocardial infarction and ischemic stroke.

The relationship between MAFLD, HF, and AF remains less clearly defined. Nevertheless, MAFLD has been associated with structural and functional cardiac remodeling, including alterations in left ventricular architecture and enlargement of the left atrium, both of which may predispose patients to the development of HF and AF. In addition, oxidative stress, systemic inflamma-

tion, and insulin resistance associated with MAFLD may contribute to the pathogenesis of HF, especially HF with preserved ejection fraction. MAFLD may also increase susceptibility to atrial fibrillation through the accumulation of epicardial adipose tissue, which has been implicated in the initiation and maintenance of AF.

Key Pathophysiological Mechanisms Linking MAFLD and Cardiovascular Disease

MAFLD and CVD represent major organ manifestations of metabolic syndrome, making it challenging to determine the independent contribution of MAFLD to cardiovascular risk because of the substantial overlap in shared metabolic risk factors.

In MAFLD, hepatic fat accumulation develops as a consequence of an imbalance between lipid influx and lipid clearance. This imbalance is mediated through several interrelated mechanisms, including excessive uptake of circulating lipids, increased hepatic de novo lipogenesis (DNL), insufficient compensatory fatty acid oxidation, and impaired export of lipids via very low-density lipoproteins (VLDL). Enhanced lipid uptake together with accelerated hepatic DNL contributes to excessive triglyceride accumulation within hepatocytes. Simultaneously, the liver increases the synthesis and secretion of large triglyceride-rich VLDL particles, which facilitate the mobilization and transport of hepatic lipids to peripheral tissues. Excessive production of low-density lipoprotein (LDL) particles in MAFLD contributes to profound alterations in plasma lipoprotein composition and promotes the development of atherogenic dyslipidemia. This dyslipidemic profile is characterized by elevated serum triglyceride concentrations, reduced high-density lipoprotein (HDL) cholesterol levels, and the predominance of small, dense LDL particles with enhanced atherogenic potential. In addition, MAFLD is associated with the accumulation of triglyceride-rich lipoproteins and their remnants, including intermediate-density lipoproteins (IDL), further contributing to cardiovascular risk. Apolipoprotein B-containing lipoproteins play a central role in the pathogenesis of atherosclerosis and are considered major contributors to cardiovascular disease development in patients with MAFLD [7,23].

Disturbances in glucose metabolism and hepatic insulin resistance represent additional hallmark features of MAFLD and are critically involved in the pathogenesis of both MAFLD and cardiovascular disorders. Disorders of glucose metabolism observed in patients with NAFLD are frequently associated with chronic systemic inflammation, visceral adiposity accompanied by excess body weight, and increased accumulation of dysfunctional ectopic adipose tissue. Hepatic and systemic insulin resistance are typically accompanied by compensatory persistent hyperinsulinemia, which contributes substantially to the formation and maintenance of an adverse metabolic milieu characterized by elevated concentrations of circulating free fatty acids and glucose. This metabolic imbalance further aggravates insulin resistance and subsequently promotes the progression of cardiometabolic complications. Hyperinsulinemia is also associated with increased hepatic glucose production, leading to sustained elevations in plasma glucose and insulin concentrations and thereby establishing a self-perpetuating pathogenic cycle. This process is further intensified by reduced hepatic insulin clearance in patients with MAFLD.

Endothelial dysfunction represents an early and pivotal stage in the development of atherosclerosis and therefore plays a critical role in the pathogenesis of cardiovascular disease. In MAFLD, endothelial dysfunction is associated with oxidative stress mediated by superoxide radicals, lipoprotein-induced vascular inflammation (including mechanisms involving apolipoprotein C3), and selective vascular insulin resistance. These abnormalities are characterized by reduced bioavailability of nitric oxide (NO), a key endogenous vasodilatory and vasoprotective molecule. Elevated concentrations of asymmetric dimethylarginine (ADMA), an endogenous inhibitor of nitric oxide synthase (NOS), further reduce nitric oxide availability and may contribute to impaired regulation of vascular tone, increased vascular permeability, and platelet dysfunction. Increased circulating ADMA levels are frequently observed in patients with MAFLD, partly as a consequence of impaired hepatic metabolism and clearance of ADMA [8].

Beyond hepatic lipid accumulation, ectopic fat deposition within the cardiovascular system – including intramyocardial fat and adipose tissue surrounding the heart – constitutes an important pathogenic mechanism linking MAFLD with CVD. Epicardial adipose tissue plays a significant role in modulation of the cardiac autonomic nervous system, and increased epicardial fat thickness has been associated with the severity of obstructive sleep apnea syndrome, cardiovascular pathology, and liver injury in patients with MAFLD. Under physiological conditions and in the setting of minimal oxidative stress, pericardial adipocytes exert protective effects on the adjacent myocardium by providing metabolic substrates, particularly through fatty acid metabolism, and by secreting cardioprotective adipokines such as adiponectin. Adiponectin possesses anti-inflammatory, antioxidant, antifibrotic, and antiatherogenic properties that contribute to cardiovascular homeostasis. However, in the presence of chronic systemic inflammation and excessive epicardial fat accumulation associated with obesity, MAFLD, or other metabolic and dyslipidemic disorders, the biological properties of epicardial adipose tissue undergo substantial pathological alterations. Production of adiponectin decreases, while synthesis of pro-inflammatory mediators – including leptin, tumor necrosis factor-α (TNF-α), and interleukins-1β and -6 – increases markedly. These mediators promote macrophage infiltration, microvascular injury, and activation of profibrotic signaling pathways. The resulting inflammatory environment contributes to pathological remodeling of the coronary arteries embedded within adipose tissue and induces structural alterations in the adjacent myocardium, including ventricular inflammation and fibrosis. These processes may ultimately facilitate the onset and progression of coronary artery disease, atrial fibrillation, and heart failure, particularly heart failure with preserved ejection fraction. Moreover, the release of pro-inflammatory cytokines from epicardial adipose

tissue into the systemic circulation amplifies systemic inflammatory activity, thereby worsening cardiometabolic dysfunction and promoting further ectopic fat accumulation within epicardial depots, creating a self-perpetuating pathogenic cycle. Since MAFLD itself contributes to systemic inflammation and is strongly associated with increased epicardial adiposity, these mechanisms likely represent an additional critical pathophysiological link between MAFLD and cardiovascular disease [9].

Alterations in gut microbiota composition, referred to as dysbiosis, have been implicated in the pathogenesis of several cardiometabolic disorders, including atherosclerosis, hypertension, type 2 diabetes mellitus, and MAFLD. A key feature of dysbiosis is impairment of intestinal barrier integrity, resulting in increased permeability of the intestinal mucosa. This disruption facilitates translocation of gut microbiota and microbial-derived molecular patterns, also known as microbe-associated or pathogen-associated molecular patterns (MAMPs/PAMPs), such as lipopolysaccharides (LPS) and peptidoglycans, into the systemic circulation. Once in the bloodstream, these molecules activate multiple intracellular signaling cascades, leading to the induction of a systemic inflammatory response associated with intestinal dysbiosis. This dysbiosis-driven inflammatory state is considered a potential mechanistic link between gut microbiota alterations and the development of cardiovascular diseases, particularly atherosclerosis. In addition, microbial metabolites – including short-chain fatty acids, primary and secondary bile acids, and trimethylamine N-oxide (TMAO) – as well as their associated signaling pathways, exert significant influence on cardiovascular pathophysiology. In this context, associations between high dietary protein intake and increased histological disease activity in MAFLD are of particular relevance, as dietary modification, including reduced consumption of red meat, may confer additional metabolic and cardiovascular benefits in patients with MAFLD [10,21].

Cardiovascular Disease as the Primary Cause of Mortality in MAFLD

CVD represents the leading cause of mortality in patients with MAFLD. The mechanisms linking MAFLD to CVD have been extensively investigated and are multifactorial in nature. These mechanisms include enhanced oxidative stress and systemic inflammation, endothelial dysfunction associated with hyperhomocysteinemia, impaired vascular tone regulation, prothrombotic alterations, dysregulation of lipid and lipoprotein metabolism, and insulin resistance. Particular emphasis has been placed on dyslipidemia and both systemic and hepatic insulin resistance, as these processes are central contributors to CVD pathogenesis and represent key therapeutic targets in cardiovascular risk reduction.

Atherogenic dyslipidemia, frequently observed in MAFLD, arises from coordinated disturbances in lipid, lipoprotein, and glucose metabolic pathways. In insulin-resistant adipose tissue, suppression of hormone-sensitive lipase by insulin is impaired, resulting in increased lipolytic activity. In combination with defective fatty acid esterification and re-esterification processes, this leads to enhanced release of free fatty acids into the circulation and their subsequent increased delivery to the liver. This augmented hepatic influx of fatty acids promotes accumulation of diacylglycerol (DAG) within hepatocytes, which further impairs insulin signaling through activation of protein kinase C (PKC) isoforms, thereby exacerbating hepatic insulin resistance. Concurrently, de novo lipogenesis (DNL) is upregulated in individuals with MAFLD. Collectively, these alterations result in increased intrahepatic lipid accumulation, which in turn stimulates hepatic secretion of very low-density lipoproteins, contributing to the development of atherogenic dyslipidemia. Furthermore, the physiological inhibitory effect of insulin on very low-density lipoprotein secretion is attenuated in MAFLD. In addition, hyperglycemia driven by both hepatic and peripheral insulin resistance further enhances hepatic VLDL production. Increased secretion of lipoprotein particles plays a central role in atherogenesis by promoting the formation of small, dense low-density lipoprotein (LDL) particles while simultaneously reducing high-density lipoprotein (HDL) concentrations [2,11].

Hepatic insulin resistance also contributes to hyperinsulinemia through increased hepatic glucose output, which stimulates compensatory pancreatic insulin secretion. Moreover, in individuals with malabsorption-related conditions, hepatic insulin clearance is markedly reduced, further intensifying the hyperinsulinemic state and reinforcing a self-perpetuating metabolic cycle. This metabolic environment promotes atherogenesis through enhanced systemic inflammation and oxidative stress, thereby facilitating vascular plaque formation.

Overall, the increased cardiovascular risk observed in MAFLD is partly attributable to disturbances in glucose, lipid, and lipoprotein metabolism, which collectively promote atherosclerotic plaque development. Therapeutic strategies targeting dyslipidemia and insulin resistance, with the aim of restoring normal lipid and lipoprotein homeostasis, may therefore reduce cardiovascular risk in patients with MAFLD.

In 2022, the American Heart Association issued a scientific statement addressing the relationship between MAFLD and cardiovascular disease risk. The statement concluded that MAFLD represents an independent risk factor for atherosclerotic cardiovascular disease, based on systematic reviews comparing MAFLD populations with individuals affected by obesity alone. Data from large cohort studies indicate that the risks of stroke and acute myocardial infarction in patients with MAFLD are broadly comparable, with hazard ratios of 1.18 and 1.17, respectively, after adjustment for age, sex, and smoking status. However, a more recent meta-analysis reported that in individuals with MAFLD, the risk of stroke (OR = 1.41) is lower than the risk of myocardial infarction (OR = 1.66).

Patients with concomitant type 2 diabetes mellitus constitute a particularly high-risk subgroup for the development of cardiovascular complications. A recent meta-analysis including more than 8,000 participants demonstrated that patients with MAFLD and concomitant type 2 diabetes mellitus have approximately a two-fold higher risk of cardiovascular disease compared with individuals with type 2 diabetes mellitus in the absence of MAFLD. It is noteworthy that the proportion of cardiovascular mortality among patients with MAFLD decreases with increasing disease severity. This trend is large-

ly explained by competing risks, particularly mortality due to HCC and liver cirrhosis, highlighting the need for a comprehensive and multidisciplinary approach to the management of MAFLD.

The European Association for the Study of the Liver, the American Association for the Study of Liver Diseases, and the American Heart Association strongly recommend systematic cardiovascular risk assessment in all patients with MAFLD. Although individuals with MAFLD combined with type 2 diabetes mellitus or advanced hepatic fibrosis represent particularly high-risk subgroups, cardiovascular risk stratification remains an essential component of routine clinical management in all patients with MAFLD.

Several validated risk prediction models are available for cardiovascular risk assessment in this population. The Framingham Risk Score (FRS) and the ASCVD (atherosclerotic cardiovascular disease) risk estimator have been applied in patients with MAFLD, although validation has been limited to relatively small cohorts comprising 309 and 1,262 individuals, respectively. Both models estimate the 10-year risk of cardiovascular events using demographic characteristics, medical history, and standard laboratory and clinical parameters, facilitating their integration into routine clinical practice. Another widely used set of tools, SCORE2 and SCORE2-OP, is designed to estimate the 10-year risk of fatal and non-fatal cardiovascular events in individuals aged 40-69 years (SCORE2) and ≥70 years (SCORE2-OP). These models stratify risk based on age, sex, smoking status, systolic blood pressure, non-high-density lipoprotein (non-HDL) cholesterol levels, and regional risk calibration. Body mass index (BMI) is not included, as it has not been shown to improve predictive accuracy. Similarly, diabetes mellitus is not incorporated as a variable because all individuals with diabetes are already classified as high cardiovascular risk and are generally considered candidates for preventive intervention. Patients classified as having high or very high 10-year cardiovascular risk are recommended to initiate early combination therapy, which typically includes structured lifestyle modification alongside pharmacological interventions such as glycemic control, lipid-lowering agents, antihypertensive therapy, and smoking cessation measures. In contrast, for individuals at low cardiovascular risk, initial management generally prioritizes lifestyle modification as the primary intervention. If pharmacological treatment is required, monotherapy with either statins or antihypertensive agents may be considered depending on the predominant risk profile [4,12].

Emerging Treatment Strategies in MAFLD

Clinical evidence from multiple trials has consistently demonstrated that weight reduction achieved through caloric restriction, with or without increased physical activity, leads to significant improvements in key biomarkers of MAFLD, including serum liver enzymes, hepatic steatosis, metabolic dysfunction-associated steatohepatitis, and liver fibrosis. Interventional studies with histologically confirmed endpoints indicate a dose-dependent relationship between weight loss and hepatic improvement. Specifically, a reduction in body weight exceeding 5% is associated with decreased hepatic fat accumulation, a 7–10% reduction is required to improve hepatic inflammation, and weight loss greater than 10% is necessary to induce regression of fibrosis [13].

Dietary patterns also play a crucial role in disease management. The Mediterranean diet has been shown to reduce hepatic lipid content and improve overall cardiometabolic profiles. In contrast, there is currently insufficient evidence regarding the efficacy and safety of very low-carbohydrate ketogenic diets – defined as carbohydrate intake below 20-50 g/day (approximately 10-25% of total energy intake) with high fat and protein content – in patients with MAFLD. Concerns remain regarding potential adverse effects on cardiovascular, renal, and other organ systems.

Time-restricted eating, also referred to as intermittent fasting, is an emerging dietary approach in which energy intake is limited to a defined daily time window. At present, evidence comparing intermittent fasting with continuous caloric restriction suggests minimal or no additional benefit of time-restricted eating on hepatic fat content in patients with MAFLD [14].

Sedentary behavior is recognized as an independent risk factor for MAFLD and is associated with an increased likelihood of disease progression. In contrast, multiple randomized controlled trials (RCTs) and meta-analyses have demonstrated that exercise alone, even in the absence of dietary modification or significant weight loss, can reduce hepatic steatosis in individuals with MAFLD. For example, an RCT showed that a 3-month program of aerobic exercise (three 90-minute sessions per week) significantly reduced hepatic fat content and liver stiffness independent of weight reduction.

However, due to limited available clinical evidence, it remains uncertain whether exercise alone can induce meaningful improvements in liver histology. Nevertheless, RCT data indicate that physical activity enhances quality of life, improves cardiorespiratory fitness, reduces visceral adiposity, and improves muscle mass and liver-related functional parameters, even in patients with advanced disease stages. Furthermore, observational studies have shown that regular vigorous physical activity is associated with a lower risk of liver fibrosis (assessed using non-invasive biomarkers), cirrhosis, overall mortality from liver-related and non-liver-related causes, as well as HCC [3,15,17].

Conclusion

1. In summary, metabolically associated fatty liver disease is strongly linked with key metabolic comorbidities, including obesity, dyslipidemia, arterial hypertension, and type 2 diabetes mellitus. Severe obesity is associated with hemodynamic alterations that contribute to cardiovascular remodeling and also plays a major role in the natural progression of metabolic dysfunction-associated steatotic liver disease, including progression from MASLD/MASH to advanced fibrosis, cirrhosis, and ultimately hepatocellular carcinoma.

2. Men older than 50 years, postmenopausal women, and individuals presenting with multiple cardiometabolic risk factors are at particularly increased risk for progressive hepatic fibrosis, cirrhosis, and related complications. In adults with MASLD, repeated assessment using non-invasive diagnostic tools is valuable for excluding

fibrosis progression and for estimating the risk of both hepatic and overall adverse clinical outcomes, including mortality. Current clinical guidelines recommend implementation of hepatocellular carcinoma surveillance programs in patients with MASLD-related cirrhosis.

3. Management of patients with NAFLD/MASLD is based on lifestyle and dietary interventions combined with multidimensional pharmacological therapy when indicated. In overweight adults with MASLD, structured dietary and behavioral interventions targeting sustained weight loss are recommended. A weight reduction exceeding 5% is associated with improvement in hepatic steatosis, a 7-10% reduction is required to improve hepatic inflammation, and a weight loss greater than 10% is necessary to achieve regression of fibrosis.

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CURRENT CONCEPTS OF COMORBID CONDITIONS IN METABOLICALLY ASSOCIATED FATTY LIVER DISEASE

Parpibaeva D.A., Buvamukhamedova N.T.

Current scientific data regarding the role of metabolic abnormalities in the development and progression of metabolically associated fatty liver disease, as well as the assessment of existing approaches to their management, have been analyzed. It is demonstrated that dyslipidemia, insulin resistance, and obesity are not only key risk factors for the development of metabolically associated fatty liver disease but also contribute to increased cardiovascular morbidity and mortality in this patient population. Current non-pharmacological and pharmacological therapeutic strategies aimed at correcting metabolic abnormalities and reducing the risk of disease progression are reviewed.

Key words: *MAFLD (NAFLD), MASG (NASG), dyslipidemia, cardiometabolic risks, type 2 diabetes mellitus, obesity.*

About the Authors

Parpibaeva Dinora Ayupovna, MD, Head of the Clinical Modeling Department, TSMU. Tel: +998946046071, e-mail: parpibaeva1970@gmail.com, https://orcid.org/0000-0001-9032-4452

Buvamukhamedova Nasiba Takhirovna, Postdoctoral Fellow, Clinical Modeling Department, TSMU. Tel: +998998190212, e-mail: nasibatohirovna_6569@mail.ru