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METABOLIC DYSFUNCTION–ASSOCIATED FATTY LIVER DISEASE (MAFLD): CURRENT CONCEPTS IN DIAGNOSIS AND MEDICAL MANAGEMENT

Health Science

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ABSTRACT

Metabolic Dysfunction–Associated Fatty Liver Disease (MAFLD) is a recently proposed nomenclature that replaces Non-Alcoholic Fatty Liver Disease (NAFLD), emphasizing the central role of metabolic dysfunction in the pathogenesis of hepatic steatosis [1]. MAFLD has emerged as the most common chronic liver disease worldwide and is closely associated with obesity, type 2 diabetes mellitus, dyslipidemia, and metabolic syndrome [2]. Unlike NAFLD, the MAFLD definition is based on positive diagnostic criteria and does not require exclusion of alcohol intake or other liver diseases [1]. MAFLD encompasses a wide spectrum ranging from simple steatosis to steatohepatitis, advanced fibrosis, cirrhosis, and hepatocellular carcinoma [3]. Early diagnosis and appropriate management are crucial to prevent disease progression and reduce cardiovascular morbidity and mortality [4]. This review discusses current concepts in the diagnosis, pathophysiology, and medical management of MAFLD.

KEYWORDS

MAFLD, Metabolic Dysfunction, Fatty Liver, Insulin Resistance, Steatohepatitis, Fibrosis

INTRODUCTION

Fatty liver disease related to metabolic abnormalities has become a major global health burden worldwide [2]. Traditionally termed as Non-Alcoholic Fatty Liver Disease (NAFLD), this condition was defined by hepatic fat accumulation in the absence of significant alcohol intake or other secondary causes [3]. However, this exclusion-based definition had several limitations, particularly in addressing the underlying metabolic derangements responsible for disease development [1].

To overcome these limitations, the term Metabolic Dysfunction–Associated Fatty Liver Disease (MAFLD) was introduced. MAFLD is diagnosed based on evidence of hepatic steatosis along with overweight or obesity, type 2 diabetes mellitus, or metabolic dysregulation [1]. This paradigm shift aligns fatty liver disease with systemic metabolic dysfunction and improves clinical relevance.

MAFLD affects nearly one-fourth of the global adult population and its prevalence is increasing rapidly, especially in developing countries due to urbanization, sedentary lifestyle, and dietary changes [2].

Pathophysiology of MAFLD

The pathogenesis of MAFLD is complex and multifactorial. Insulin resistance plays a central role, leading to increased free fatty acid flux to the liver, enhanced de novo lipogenesis, and reduced fatty acid oxidation [4]. Hepatic lipid accumulation results in lipotoxicity, oxidative stress, mitochondrial dysfunction, and release of pro-inflammatory cytokines [3].

Chronic inflammation promotes hepatocyte ballooning, cell injury, and activation of hepatic stellate cells, ultimately resulting in progressive fibrosis [3]. Gut dysbiosis, genetic polymorphisms such as PNPLA3, and adipokine imbalance further accelerate disease progression [1].

Diagnostic Criteria of MAFLD

Diagnosis of MAFLD requires evidence of hepatic steatosis detected by imaging, blood biomarkers, or histology, along with one of the following criteria [1]:

1. Overweight or obesity
2. Type 2 diabetes mellitus
3. Evidence of metabolic dysregulation such as hypertension, dyslipidemia, insulin resistance, or elevated inflammatory markers

Investigations

- Laboratory tests: Mild elevation of ALT and AST, dyslipidemia, raised fasting glucose or HbA1c [3]
- Imaging: Ultrasound (first-line), CT scan, MRI-PDFF [2]
- Non-invasive fibrosis assessment: FIB-4, NAFLD fibrosis score, transient elastography [3]
- Liver biopsy: Reserved for selected cases to assess inflammation and fibrosis severity [3]

Clinical Spectrum and Complications

MAFLD represents a continuous disease spectrum including [4]:

- Simple steatosis
- Metabolic steatohepatitis
- Progressive fibrosis
- Cirrhosis
- Hepatocellular carcinoma

Patients with MAFLD are at a significantly increased risk of cardiovascular disease, which remains the leading cause of mortality, followed by liver-related complications [2].

Medical Management of MAFLD

Lifestyle Modification

Lifestyle intervention remains the cornerstone of MAFLD management [3]:

- Weight reduction of 7–10% of body weight
- Regular aerobic and resistance exercise
- Calorie-restricted diet with reduced saturated fats and refined carbohydrates
- Mediterranean-style diet is strongly recommended

Pharmacological Therapy

Currently, no drug is specifically approved for MAFLD; however, certain agents are used in selected patients [3,4]:

- Pioglitazone: Particularly beneficial in MAFLD with diabetes
- Vitamin E: For non-diabetic patients with steatohepatitis
- GLP-1 receptor agonists: Promote weight loss and improve liver enzymes
- Statins: Safe and effective for dyslipidemia and cardiovascular risk reduction

Emerging Therapies

Several novel agents targeting inflammation, fibrosis, and metabolic pathways are under clinical trials and may provide disease-specific therapies in the future [1].

Role of Surgery and Interventional Strategies

Bariatric surgery has demonstrated significant improvement in hepatic steatosis, inflammation, and fibrosis in morbidly obese patients with MAFLD [4]. Liver transplantation remains the definitive treatment for end-stage liver disease secondary to MAFLD.

CONCLUSIONS

MAFLD represents a major paradigm shift in the classification of fatty liver disease by placing metabolic dysfunction at the center of diagnosis and management [1]. Early diagnosis using positive diagnostic criteria allows timely intervention and risk stratification. Lifestyle modification remains the foundation of therapy, while pharmacological and surgical options play an adjunctive role. With the rising prevalence of metabolic syndrome, MAFLD is expected to become the leading cause of chronic liver disease globally.

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