Review Article

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Metabolic dysfunction-associated steatotic liver disease: Current perspectives

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Metabolic dysfunction-associated steatotic liver disease (MASLD), formerly known as non-alcoholic fatty liver disease (NAFLD), represents the most prevalent chronic liver condition worldwide. Affecting approximately one-third of the global population, MASLD is strongly associated with obesity, insulin resistance, type 2 diabetes mellitus (T2DM), dyslipidemia, and metabolic syndrome. The renaming to MASLD underscores the central role of metabolic dysfunction in its pathogenesis and clinical spectrum. The disease ranges from simple hepatic steatosis to metabolic dysfunction-associated steatohepatitis (MASH), fibrosis, cirrhosis, and hepatocellular carcinoma (HCC). Non-invasive biomarkers and imaging modalities have improved risk stratification, but liver biopsy remains the gold standard for diagnosis and staging. Current management strategies emphasize lifestyle interventions, weight loss, and cardiometabolic risk control, with emerging pharmacotherapies showing promise. MASLD poses a major burden on healthcare systems due to its progressive nature and extrahepatic associations with cardiovascular disease, chronic kidney disease, and malignancies. This review provides an updated overview of epidemiology, pathogenesis, diagnosis, management, and future directions in MASLD, highlighting evolving therapeutic opportunities and research priorities.

Keywords: Fatty liver; Liver cirrhosis; Metabolic dysfunction-associated steatotic liver disease (MASLD); Non-alcoholic fatty liver disease (NAFLD).

Introduction

Metabolic dysfunction-associated steatotic liver disease (MASLD) is a chronic liver disorder characterized by hepatic fat accumulation in individuals with cardiometabolic risk factors in the absence of significant alcohol consumption or other secondary causes of hepatic steatosis.[1, 2] The term MASLD was recently adopted to replace the long-standing nomenclature of non-alcoholic fatty liver disease (NAFLD), in an effort to better reflect the underlying pathophysiology and reduce ambiguity in diagnosis.[3] Unlike NAFLD, the MASLD definition requires the presence of at least one metabolic risk factor, such as overweight/obesity, type 2 diabetes mellitus (T2DM), hypertension, dyslipidemia, or insulin resistance, in addition to hepatic steatosis.[4]

The spectrum of MASLD ranges from isolated hepatic steatosis, generally considered benign, to metabolic dysfunction-associated steatohepatitis (MASH), which can progress to fibrosis, cirrhosis, and hepatocellular carcinoma (HCC).5 Importantly, liver-related morbidity and mortality correlate strongly with the stage of fibrosis rather than the presence of steatohepatitis alone.[6]

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MASLD is not confined to the liver; it is increasingly recognized as a multisystem disease linked with increased risk of cardiovascular disease (CVD), chronic kidney disease (CKD), extrahepatic malignancies, and overall mortality.[7,8] With a global prevalence approaching one-third of the adult population and rising incidence in children, MASLD has become a major public health and economic burden.[9]

Epidemiology and Global Burden

MASLD is the most prevalent chronic liver disease worldwide, affecting approximately 25–30% of the global adult population.[10] Its prevalence parallels the increasing rates of obesity, T2DM, and sedentary lifestyles. Regional variation exists: the highest prevalence is reported in the Middle East and South America (30–35%), while lower prevalence is observed in sub-Saharan Africa (13–18%).[11] In Asia, prevalence has risen sharply over the last two decades, reflecting dietary westernization and urbanization.[12]

MASLD is strongly associated with obesity, with up to 70–90% of obese individuals demonstrating hepatic steatosis on imaging.[13] Among patients with T2DM, prevalence exceeds 50%, with advanced fibrosis present in 15–20%.[14] Importantly, MASLD also occurs in lean individuals, particularly in Asian populations, underscoring the role of genetic and environmental factors.[15]

Pediatric MASLD is increasingly recognized, with global prevalence estimated at 7–10% among children and up to 30–40% in obese adolescents.[16] Early onset MASLD may progress more rapidly, increasing lifetime risk of cirrhosis and HCC.

The disease imposes a substantial healthcare and economic burden. In the United States alone, MASLD-related healthcare costs are projected to exceed $100 billion annually.[17] Moreover, MASLD is now a leading indication for liver transplantation in Western countries, surpassing viral hepatitis.[18]

Pathogenesis and Risk Factors

The pathogenesis of MASLD is multifactorial, involving a complex interplay between genetic, metabolic, environmental, and gut microbiome-related factors.

Insulin Resistance and Lipotoxicity

Insulin resistance is central to disease development. Impaired insulin signaling leads to increased lipolysis, elevated free fatty acid flux to the liver, and de novo lipogenesis.[19] Excess lipid accumulation induces lipotoxicity, mitochondrial dysfunction, oxidative stress, and hepatocellular injury, promoting inflammation and fibrosis.

Genetic Susceptibility

Genome-wide association studies have identified key genetic variants influencing susceptibility and disease progression. The **PNPLA3 I148M** polymorphism is strongly associated with hepatic fat accumulation and fibrosis progression.[20] Other variants, including **TM6SF2, MBOAT7, and HSD17B13**, modulate risk and clinical phenotype.[21]

Gut Microbiota and Intestinal Permeability

Dysbiosis of gut microbiota contributes to disease pathogenesis through increased intestinal permeability, endotoxin release, and activation of hepatic inflammatory pathways.[22] Microbiome-derived metabolites, such as short-chain fatty acids and bile acid derivatives, further influence hepatic lipid metabolism.

Dietary and Lifestyle Factors

Western-style diets rich in fructose, saturated fat, and processed foods promote hepatic fat deposition and inflammation.[23] Sedentary lifestyle exacerbates insulin resistance and metabolic dysfunction, while physical activity confers protective effects.

Additional Risk Factors

Other contributors include endocrine disorders (e.g., polycystic ovary syndrome, hypothyroidism), obstructive sleep apnea, and certain medications such as corticosteroids and amiodarone.[24]

Clinical Spectrum

MASLD encompasses a wide histological and clinical spectrum:

Simple Steatosis (MASLD without MASH): Characterized by hepatic fat accumulation without significant inflammation or fibrosis; generally benign with low risk of progression.[25]

MASH: Defined by steatosis, lobular inflammation, and ballooning degeneration; carries higher risk of fibrosis and adverse outcomes.[26]

Fibrosis and Cirrhosis: Progressive fibrosis can culminate in cirrhosis, portal hypertension, and liver failure. Fibrosis stage is the most important predictor of liver-related outcomes.[27]

Hepatocellular Carcinoma: MASLD-related cirrhosis increases HCC risk, but HCC can also develop in non-cirrhotic MASLD, complicating surveillance strategies.[28]

Extrahepatic Manifestations: Cardiovascular disease, CKD, and certain malignancies (colorectal, breast) are major causes of mortality in MASLD patients.[29]

Diagnosis

Accurate diagnosis and staging are critical for prognosis and management.

Clinical and Laboratory Evaluation

Diagnosis requires evidence of hepatic steatosis in the presence of metabolic dysfunction and exclusion of secondary causes (significant alcohol intake, viral hepatitis, Wilson's disease, etc.).[5] Liver enzymes may be normal in many patients, limiting their utility.

Imaging Modalities

Ultrasound: Widely available and inexpensive, but limited sensitivity in detecting mild steatosis or differentiating fibrosis stages.

Controlled Attenuation Parameter (CAP, FibroScan): Provides quantitative assessment of steatosis and simultaneous fibrosis measurement using transient elastography.[30]

Magnetic Resonance Imaging-Proton Density Fat Fraction (MRI-PDFF): Highly accurate for quantifying hepatic fat content; increasingly used in clinical trials.

MR Elastograp hy: Superior accuracy for fibrosis staging compared to other non-invasive modalities.[31]

Non-Invasive Biomarkers

Several scoring systems aid in fibrosis risk stratification, including the **Fibrosis-4 (FIB-4) index** and **NAFLD fibrosis score (NFS).**[32] Serum biomarkers such as cytokeratin-18 fragments and novel fibrosis panels are under investigation.

Liver Biopsy

Despite limitations, biopsy remains the reference standard for diagnosing MASH and staging fibrosis. However, its invasiveness, cost, and sampling variability restrict routine use.[33]

Management

Currently, no approved pharmacological therapy exists for MASLD. Management is centered on treating the underlying metabolic drivers (weight, insulin resistance, dyslipidaemia) and on preventing progression to fibrosis and cirrhosis. Current guidance emphasizes lifestyle intervention as first-line therapy, with pharmacologic and procedural treatments reserved for selected patients with advanced disease or when lifestyle measures fail.[34, 35]

Goals of therapy

Primary goals are: (1) reduce liver fat and hepatic inflammation (MASH), (2) halt or reverse fibrosis progression, and (3) treat cardiometabolic comorbidities to reduce overall morbidity and mortality. Management must be individualized by fibrosis stage and cardiometabolic risk.[34]

Lifestyle interventions (cornerstone)

Lifestyle change remains the foundation of MASLD treatment. Structured programs that produce sustained weight loss result in improvements in hepatic steatosis, necroinflammation and—when weight loss ≥7–10% is achieved—histologic improvement in

MASH and fibrosis regression in some patients.[35, 36]

Practical recommendations

Weight loss target: Aim for 7–10% body weight loss to improve steatosis and MASH; greater loss provides greater benefit.[34, 35]

Diet: Calorie reduction with emphasis on Mediterranean-style dietary patterns (high in vegetables, whole grains, lean protein; low in refined sugars and saturated fats) is supported by guidelines and trials.[35, 37]

Physical activity: At least 150–200 minutes/week of moderate aerobic exercise plus resistance training as tolerated.[35]

Alcohol: Minimize or avoid alcohol; even modest intake may worsen outcomes in some patients with MASLD.[36]

Pharmacologic approaches

No single "universal" drug is recommended for all patients with MASLD; therapy is selected by disease severity (especially presence of MASH with fibrosis) and comorbidities. Recent guideline panels and trials have updated recommendations and expanded available options.[34-36]

1. *Treat cardiometabolic comorbidities*
Pioglitazone: For biopsy-proven MASH, pioglitazone has shown histologic benefit (improved steatosis and inflammation) in multiple trials (useful in patients with and without diabetes, but consider weight gain and fracture risk).[34, 35]
Statins: Safe in MASLD and recommended for atherosclerotic cardiovascular risk management; they do not worsen liver disease and are indicated when cardiovascular indications exist.[34]

Vitamin E: Demonstrated histological benefit in non-diabetic patients with MASH, though long-term risks (prostate cancer, hemorrhagic stroke) limit use.[38]

Sodium-glucose transport protein 2 (SGLT2) inhibitors: Improve hepatic steatosis and metabolic parameters, though histological benefits require further validation.[39]

2. *Glucagon-like peptide-1(GLP-1) receptor agonists and dual incretin agonists*
GLP-1 receptor agonists (semaglutide) and dual Glucose-dependent insulinotropic polypeptide (GIP)/GLP-1 agonists (tirzepatide) produce substantial weight loss and reduce liver fat; trials suggest marked improvements in steatosis and metabolic parameters, with promising signals for inflammation resolution in some studies.[35, 37]

3. *Agents targeting NASH/MASH biology*
Resmetirom (thyroid hormone receptor-β agonist): Developed specifically for MASH; recent regulatory decisions reflect evidence for liver-fat reduction and some histologic benefit in phase 3 programs.[40-42]

Other agents: under investigation or with mixed results include fibroblast growth factor analogues, farnesoid X receptor (FXR) agonists (obeticholic acid), peroxisome proliferator-activated receptor (PPAR) agonists, and combination strategies.[34, 41-44]

4. *When to consider pharmacotherapy for the liver itself*
Most guidance recommends considering MASH-directed pharmacotherapy for patients with biopsy-proven MASH and ≥F2 fibrosis or at high risk of progression, particularly if lifestyle interventions have failed.[34, 42]

Bariatric/metabolic surgery and endoscopic options

For patients with obesity and MASLD, bariatric/metabolic surgery (e.g., sleeve gastrectomy, Roux-en-Y gastric bypass) is highly effective at substantial and durable weight loss and often results in resolution or marked improvement of steatosis and MASH; it is appropriate when surgical criteria for obesity are met. Endoscopic weight loss procedures are emerging options with promising effects.[35, 36]

Monitoring and follow-up

Fibrosis assessment using noninvasive tests (transient elastography, serum fibrosis scores like FIB-4 or NAFLD Fibrosis Score) is essential to

stratify risk and guide therapy intensity. Repeat assessment depends on baseline fibrosis and interventions instituted.[45]

Practical algorithm

1. Screen for metabolic drivers and assess fibrosis stage.[34]
2. Implement structured lifestyle program with weight loss target 7–10%.[35]
3. Treat cardiovascular risk factors (statins, antihypertensives, diabetes therapy).[34]
4. For biopsy-proven MASH with ≥F2 fibrosis: discuss pharmacologic options and trials.[43]
5. For eligible patients with obesity and MASLD: consider bariatric/metabolic surgery.[35]

Nomenclature changes (NAFLD→MASLD) refocus attention on systemic metabolic drivers. Combination therapies (antifibrotic + metabolic) and precision-medicine approaches are in development.[36, 37, 41]
Lifestyle modification with meaningful, sustained weight loss remains the bedrock of MASLD therapy. Pharmacotherapies (e.g., GLP-1/GIP agonists, resmetirom) offer options for selected patients. Management should be individualized, fibrosis-directed, and integrated with cardiometabolic care.[34-36]

Special Populations

Pediatric MASLD

Early detection and lifestyle interventions are critical. Pediatric MASLD may have distinct histological patterns and more aggressive progression.[46]

Lean MASLD

Particularly prevalent in Asia, lean MASLD highlights the contribution of genetic susceptibility and visceral adiposity. Despite normal body mass index (BMI), these patients remain at risk for fibrosis and cardiometabolic complications.[47]

Elderly Patients

MASLD in the elderly is often underdiagnosed due to normal liver enzymes and overlapping comorbidities. Age-related sarcopenia exacerbates disease progression.[48]

Future Directions and Research Gaps

Despite advances, MASLD remains underdiagnosed and undertreated. Key challenges include development of reliable non-invasive biomarkers for steatohepatitis and fibrosis staging, identification of effective, safe, and widely accessible pharmacological therapies, and tailored management approaches for pediatric, lean, and elderly populations. Integration of digital health tools and artificial intelligence in risk stratification and surveillance is needed, besides understanding the long-term safety and efficacy of emerging agents in real-world settings.

Conclusion

MASLD has emerged as a leading cause of chronic liver disease, tightly linked with the global epidemics of obesity and metabolic syndrome. Its multisystem nature, rising prevalence, and association with adverse hepatic and extrahepatic outcomes make it a critical public health challenge. While lifestyle modification remains the cornerstone of management, promising pharmacotherapies are on the horizon. Continued research into disease mechanisms, diagnostic modalities, and therapeutic interventions is essential to reduce the growing burden of MASLD.

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