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Editorial

Beyond the Liver: Why MASLD Demands a Unified, Interdisciplinary Care Model

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Metabolic dysfunction-associated steatotic liver disease (MASLD), affecting nearly 40% of the global population, represents a paradigm shift that views liver fat as a primary hepatic manifestation of systemic metabolic dysregulation rather than an isolated liver condition. MASLD and its progressive form, metabolic dysfunction-associated steatohepatitis (MASH), drive substantial extrahepatic mortality through cardiovascular disease, chronic kidney disease, and extrahepatic malignancies. However, current care models remain fragmented, as non-hepatology specialties frequently overlook hepatic manifestations and primary care often underestimates fibrosis risk in patients with normal transaminase levels. Addressing this systemic metabolic crisis requires health systems to implement an integrated, interdisciplinary framework combining standardized primary care screening protocols using non-invasive tools (e.g., FIB-4 index), multi-target pharmacotherapies (such as GLP-1 receptor agonists, SGLT2 inhibitors, and resmetirom), and dedicated co-managed clinics across primary care, cardiology, endocrinology, nephrology, oncology, and hepatology to detect fibrosis earlier and prevent irreversible cardiorenal and hepatic outcomes.

KEYWORDS: MASLD, Fatty liver disease, NAFLD, MASH, NASH, CAD, CKD, Metabolic syndrome

Metabolic dysfunction-associated steatotic liver disease (MASLD) (formerly known as nonalcoholic fatty liver disease, NAFLD) affects nearly 40% of the global population, making it the most prevalent chronic liver condition in history.[1,2] The recent nomenclature shift from NAFLD to MASLD highlights a fundamental paradigm shift: the medical community no longer views liver fat in isolation, but as a primary hepatic manifestation of systemic metabolic dysfunction.[2] Treating MASLD and its progressive form, metabolic dysfunction-associated steatohepatitis (MASH) (previously nonalcoholic steatohepatitis, NASH), purely within hepatology clinics is an outdated model of care. Because MASLD and MASH drive extrahepatic mortality, including cardiovascular disease,[3] chronic kidney disease,[4] and extrahepatic malignancies,[5] healthcare systems must adopt an integrated, interdisciplinary care framework across primary care, cardiology, nephrology, endocrinology, and hepatology.

MASLD exists on the spectrum of steatotic liver disease (SLD) (Figure 1). Current diagnostic criteria from the American Association for the Study of Liver Diseases (AASLD) require hepatic steatosis

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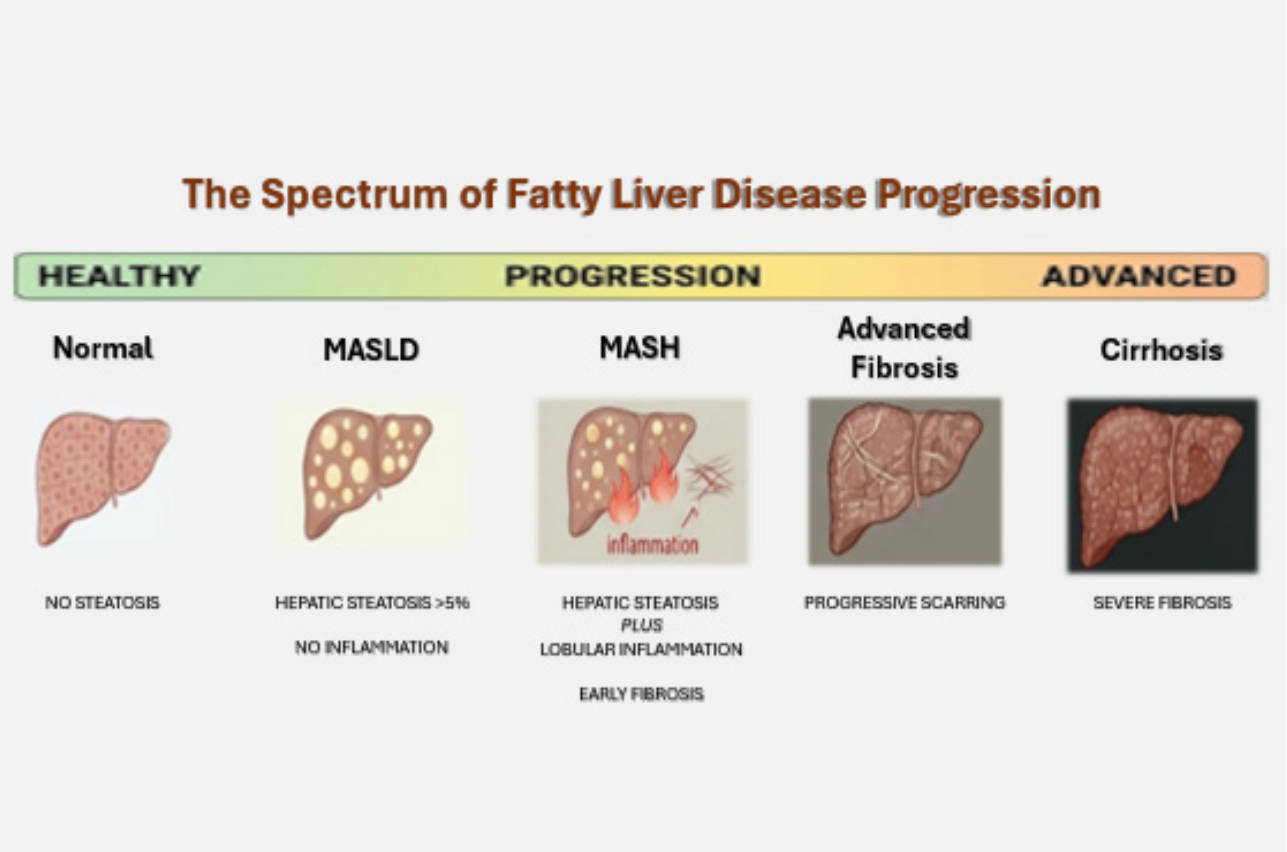

FIGURE 1. Spectrum of fatty liver disease progression. **Abbreviations:** MASH, metabolic dysfunction-associated steatohepatitis; MASLD, metabolic dysfunction-associated steatotic liver disease.

on imaging or biopsy and at least one cardiometabolic criterion (Figure 2).[2] MASH is characterized by hepatic steatosis, hepatocyte ballooning, and lobular inflammation, and requires a liver biopsy for definitive diagnosis.[1] As MASH progresses, the liver becomes fibrotic, leading to cirrhosis, possible hepatocellular carcinoma (HCC), and the need for liver transplantation.[1] Recent literature even suggests that MASLD and MASH can have extrahepatic multisystemic manifestations, particularly in the cardiovascular, renal, and endocrine systems.[3,4,5] MASLD and disorders of these organ systems share risk factors and a common metabolic milieu with subsequent manifestations of systemic pathology. Evidence supports direct causal pathways linking MASLD to extrahepatic disease.[3,4,5]

Although the direct pathophysiological mechanism of MASLD's multisystemic effects has not yet been fully elucidated, it likely stems from a combination of downstream effects of systemic insulin resistance, a proinflammatory milieu, fatty acid mobilization, and resultant lipotoxicity.[3] In the cardiovascular system, these effects can lead to endothelial dysfunction and atherogenic dyslipidemia.[3] As such, MASLD has been independently associated with up to double the rate of clinically significant adverse cardiovascular events, including acute coronary syndrome and ischemic stroke, and mortality related to these events as compared with patients without MASLD.[3] Cardiologists should be involved early in the diagnosis and management of MASLD to initiate cardiovascular therapies early, prevent major adverse cardiovascular events, and reduce mortality. Given that cardiovascular disease remains the leading cause of death in patients with MASLD,[6] proactive cardiology involvement can help identify high-risk individuals, optimize preventive and therapeutic interventions, and address the systemic cardiometabolic abnormalities that drive both hepatic and cardiovascular disease progression.

Systemic dysregulation associated with

MASLD also affects renal and endocrine feedback loops. MASLD and type 2 diabetes mellitus (T2DM) share a bidirectional relationship: T2DM accelerates progression to MASH and fibrosis, while MASLD worsens glycemic control.[7] Tight regulation of serum glucose in patients with T2DM by an endocrinologist is imperative, as poor glycemic control fuels a vicious cycle in which insulin resistance promotes hepatic steatosis and fibrosis, while worsening liver disease further impairs glucose homeostasis. Early endocrinology involvement can therefore help interrupt disease progression, improve metabolic outcomes, and reduce the risk of both hepatic and extrahepatic complications.

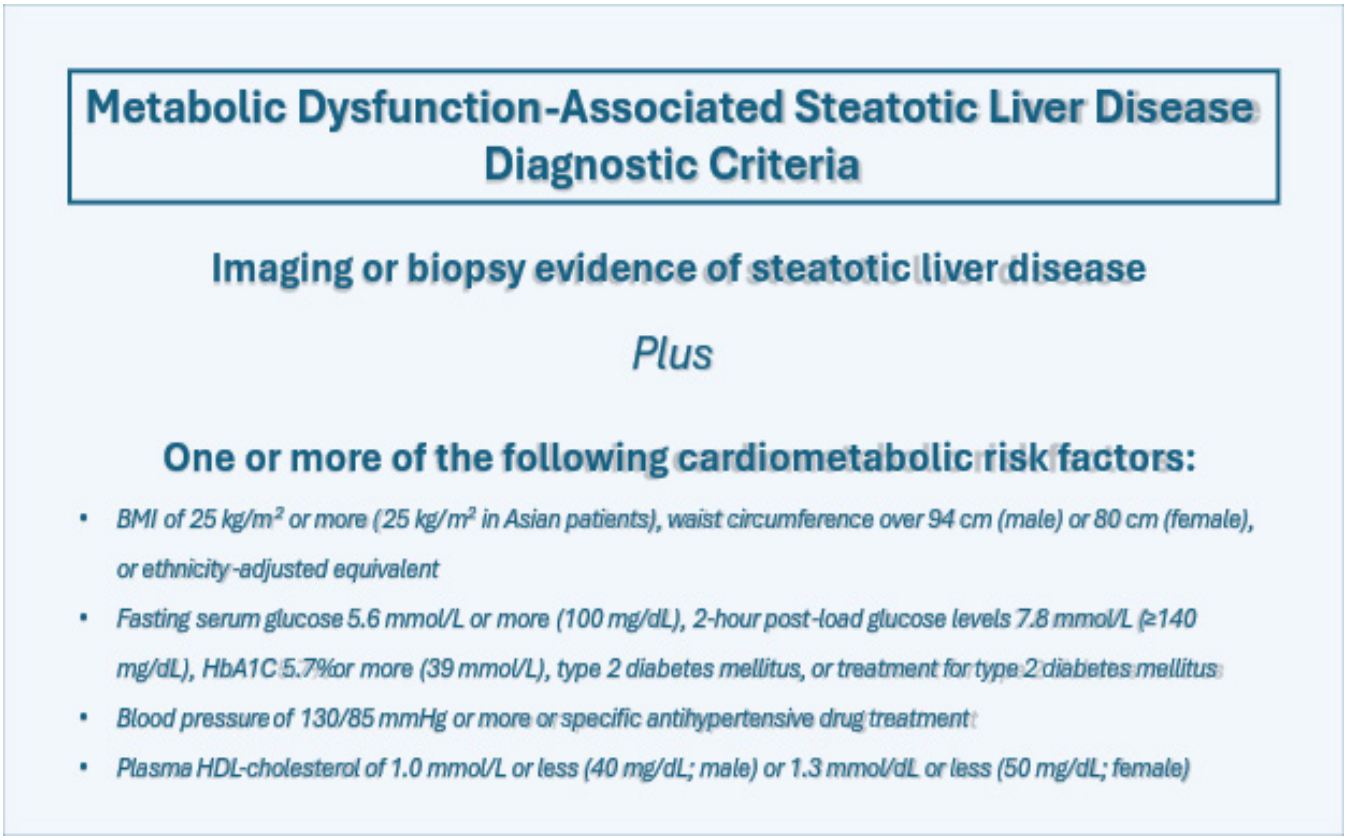

FIGURE 2. Diagnostic criteria for metabolic dysfunction-associated steatotic liver disease (MASLD).

Extrahepatic lipid deposition, particularly in the perirenal space, is strongly associated with chronic kidney disease and contributes directly to renal dysfunction by compressing the renal vasculature and parenchyma, increasing interstitial hydrostatic pressure and renin release, and reducing eGFR.[4] As a result, MASLD is associated with an increased risk of chronic kidney disease independent of age, sex, obesity, hypertension, T2DM, and other traditional renal risk factors.[4] Furthermore, the risk of chronic kidney disease increases with more advanced liver disease, especially with the severity of hepatic fibrosis.[4] Nephrologists must be involved to help mitigate and manage these risks, as the renal consequences of MASLD extend beyond shared metabolic risk factors and may represent a direct manifestation of systemic disease. Early nephrology consultation can promote timely risk stratification, preserve renal function through targeted interventions, and reduce the likelihood of progression to end-stage renal disease, particularly in patients with advanced fibrosis and multiple cardiometabolic comorbidities.

Substantial epidemiological evidence shows that MASLD is a risk factor for HCC, even in those without cirrhosis.[5] MASLD has also demonstrated a strong association with extrahepatic malignancies, especially uterine cancer, breast cancer, prostate cancer, colorectal cancer, and lung cancer.[5] Interestingly, extrahepatic cancers were over eightfold more frequent than HCC in patients with MASLD.[5]

The role of primary care in encouraging compliance with age-related or high-risk cancer screening as well as aggressive diagnostic workups in patients with MASLD and suggestive malignant symptoms cannot be overstated. Early involvement of oncologic care should also be lauded. Given the growing recognition of MASLD as a risk factor for both hepatocellular carcinoma and several extrahepatic malignancies, proactive oncologic involvement can promote earlier cancer detection, facilitate timely treatment, and improve survival through coordinated multidisciplinary management.

The current problem with metabolic syndrome care is that it is fragmented across many specialties, many of whom do not recognize the significant interplay between MASLD and their respective specialties. Primary care often underestimates fibrosis risk because serum transaminase levels are normal; endocrinology manages glycemic control but rarely screens for liver fibrosis with validated non-invasive tests, including the FIB-4 score or transient elastography; and cardiology treats coronary artery disease without addressing underlying hepatic steatosis or inflammation. The bottom line is that treating the multisystemic ripple effect of MASLD requires a multifaceted, interdisciplinary approach. Critics may argue that specialized multidisciplinary clinics may be resource-intensive, difficult to scale in low-resource environments, and/or place heavy administrative burden on care coordinators. However, the downstream costs of uncoordinated care, including end-stage renal disease, heart failure hospitalization, liver transplants, late-stage cancers, etc., far exceed the upfront investment in integrated screening and early co-management.

The blueprint for the interdisciplinary care of MASLD and its sequelae is multipronged, with each specialty playing a significant and vital role. First, creating and implementing standardized primary care screening pathways can help catch advanced fibrosis early. Universal use of simple risk stratifiers like the FIB-4 index in routine metabolic workups (T2DM, metabolic syndrome, obesity), especially in those with imaging or biopsy evidence of hepatic steatosis, can improve early MASLD diagnosis and shorten the delay to appropriate therapy. Second, all specialties in a health system can adopt consistent therapeutic guidelines, including lifestyle changes (i.e., diet and exercise) and dual-target pharmacotherapies to promote weight loss and optimize metabolic abnormalities. Clinicians should encourage multi-effect drug therapies that target both liver pathology and systemic risk, such as glucagon-like peptide (GLP-1) receptor agonists, glucose-dependent insulinotropic polypeptide (GIP)/GLP-1 receptor co-agonists, sodium-glucose cotransporter 2 (SGLT2) inhibitors, and the new liver-specific thyroid receptor-beta agonist, resmetirom. Finally, and perhaps most importantly, establishing metabolic-liver-cardiovascular clinics can create a setting for dietitians, physical therapists, hepatologists, cardiologists, nephrologists, and endocrinologists to align on a unified management plan, improving patient compliance by offering a "one-stop shop" for all metabolic syndrome-related management.

In conclusion, MASLD is not a silent liver disease; rather, it is a systemic metabolic crisis masquerading as a liver condition. Health system leaders, clinical guidance committees, and primary care networks must reset their standards of care by implementing uniform risk-stratification protocols and formally integrate multidisciplinary care teams across the MASLD spectrum. Failure to act will perpetuate a fragmented model that identifies disease only after irreversible damage has occurred. In contrast, a coordinated multidisciplinary approach offers an opportunity to detect fibrosis earlier, proactively address cardiorenal and metabolic risk, and halt disease progression before MASLD culminates in irreversible multisystem damage.

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