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SPECIAL ARTICLE

Metabolic dysfunction–associated steatotic liver disease: Update and impact of new nomenclature on the American Association for the Study of Liver Diseases practice guidance on nonalcoholic fatty liver disease

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Abstract

This commentary discusses how clinicians and various stakeholders can utilize the recently published American Association for the Study of Liver Diseases nonalcoholic fatty liver disease (AASLD NAFLD) Practice Guidance in light of the change in the nomenclature to steatotic liver disease and its subcategories. The new terminologies explained in this commentary make it easier for the readers to interchangeably use metabolic dysfunction-associated steatotic liver disease (MASLD) in place of NAFLD and metabolic-dysfunction associated steatohepatitis (MASH) instead of non-alcoholic steatohepatitis (NASH), respectively, as they read the NAFLD Practice Guidance. The guidance document is relevant and can be utilized for the diagnosis, risk stratification, and management of patients with MASLD. This commentary serves as an accompanying article to the NAFLD Practice Guidance and helps it clinical application in the light of the new nomenclature.

INTRODUCTION

A global Delphi consensus process co-led by the American Association for the Study of Liver Diseases (AASLD) and the European Association for the Study of the Liver (EASL), in collaboration with the Latin American Association for the Study of the Liver (ALEH), recommended a new nomenclature inclusive

Abbreviations: ALD, alcohol-associated liver disease; ALEH, Latin American Association for the Study of the Liver; CMRF, cardiometabolic risk factor; LALD, lysosomal acid lipase deficiency; MASH, metabolic dysfunction–associated steatohepatitis; MASL, metabolic dysfunction–associated steatotic liver; MASLD, metabolic dysfunction–associated steatotic liver disease; MetALD, metabolic dysfunction and alcohol-associated steatotic liver disease; SLD, steatotic liver disease.

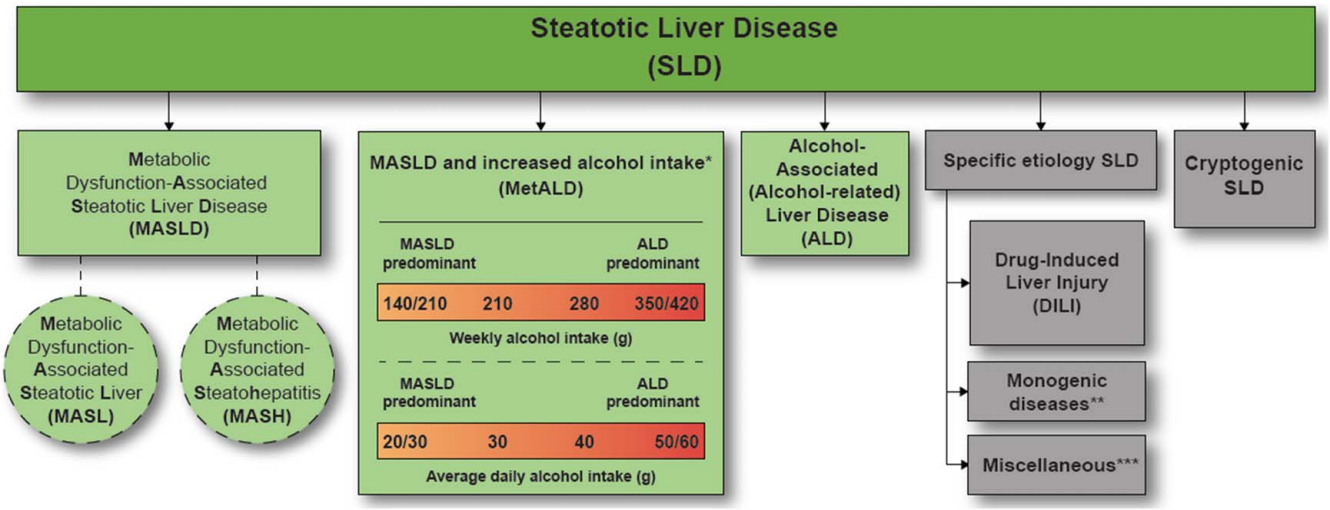

FIGURE 1 Schema of SLD and subcategories. SLD, diagnosed histologically or by imaging, has many potential etiologies. MASLD, defined as the presence of hepatic steatosis in conjunction with one CMRF and no other discernible cause, ALD, and an overlap of the two (MetALD), comprise the most common causes of SLD. Persons with MASLD and steatohepatitis will be designated as MASH (previously NASH). MASL refers to the presence of MASLD in the absence of steatohepatitis. Within the MetALD group, there exists a continuum across which the contribution of MASLD and ALD will vary. To align with current literature, limits have been set accordingly for weekly and daily consumption, understanding that the impact of alcohol intake varies between individuals. Other causes of SLD need to be considered separately, as is already done in clinical practice, given their distinct driving factors. Multiple etiologies of steatosis can coexist. If there is uncertainty and the clinician strongly suspects metabolic dysfunction despite the absence of CMRF, early MASLD may be considered pending additional testing (eg, HOMA-IR and oral glucose tolerance tests). Those with no identifiable cause (cryptogenic SLD) may be recategorized in the future pending developments in our understanding of disease pathophysiology or the emergence of CMRFs. Finally, the ability to provide an affirmative diagnosis allows for the coexistence of other forms of liver disease with MASLD, for example, MASLD + autoimmune hepatitis or viral hepatitis. *Weekly intake 140–350 g female, 210–420 g male (average daily 20–50 g female, 30–60 g male). **For example, LALD, Wilson disease, hypobetalipoproteinemia, inborn errors of metabolism, environmental toxins. ***For example, HCV, malnutrition, celiac disease, HIV. Adapted with permission from[2] Rinella ME, Lazarus JV, Ratziu V, Francque SM, Sanyal AJ, Kanwal F, et al. A multi-society Delphi consensus statement on new fatty liver disease nomenclature. Hepatology. 2023;78:1966-86. Abbreviations: ALD, alcohol-associated liver disease; CMRF, cardiometabolic risk factor; HOMA-IR, Homeostatic Model Assessment for Insulin Resistance; LALD, lysosomal acid lipase deficiency; MASH, metabolic dysfunction–associated steatohepatitis; MASL, metabolic dysfunction–associated steatotic liver; MASLD, metabolic dysfunction–associated steatotic liver disease; MetALD, metabolic dysfunction and alcohol-associated steatotic liver disease; SLD, steatotic liver disease.

of updated definitions for the conditions formerly encompassed by the term nonalcoholic fatty liver disease (NAFLD). In this compendium, we briefly review these changes to explain how they may impact the recommendations included in the recently published AASLD Practice Guidance on the Clinical Assessment and Management of NAFLD and further clarify how a change in the nomenclature from NAFLD to metabolic dysfunction–associated steatotic liver disease (MASLD) may impact clinical practice.[1] In addition, we provide updates to figures related to the new nomenclature.

The Nomenclature Consensus Initiative was designed to address some of the limitations of the terms NAFLD and NASH. Among them were the exclusionary nature of the diagnosis, the lack of recognition of the root cause of the condition, and the use of potentially stigmatizing terms. This global process involved various national hepatology societies, endocrinology societies, and patient advocacy organizations, representing 56 countries in a robust, representative Delphi process that objectively examined the need for revisiting the NAFLD nomenclature and for a name and/or definition change. This process resulted in a new nomenclature and a change in the definition, summarized below and in Figure 1.[2]

THE OVERARCHING TERM: STEATOTIC LIVER DISEASE

The overarching term of steatotic liver disease (SLD) was chosen to classify individuals with hepatic steatosis due to various etiologies. The Delphi panel recommended the term steatosis in lieu of the term fatty because the latter was considered to be stigmatizing. Hence, the overarching term and its derivatives were based on “steatotic liver disease.” This overarching term encompasses MASLD (using specific criteria detailed below) and a new overlap category that includes individuals with cardiometabolic risk factors (CMRFs) and a spectrum of alcohol consumption (metabolic dysfunction and alcohol-associated steatotic liver disease, MetALD), while continuing to recognize other causes of hepatic steatosis including alcohol-associated

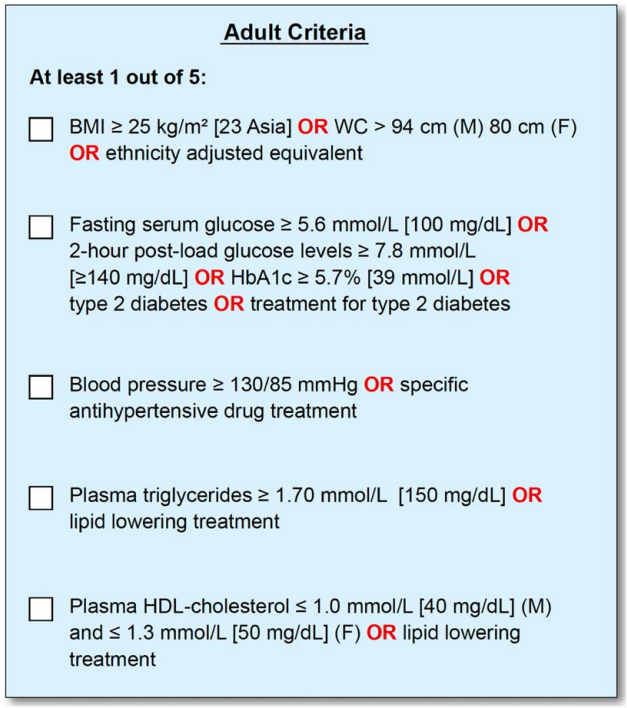

FIGURE 2 Criteria to define MASLD. In the presence of hepatic steatosis, the finding of any CMRF would confer a diagnosis of MASLD if there are no other causes of hepatic steatosis. If additional drivers of steatosis are identified, then this is consistent with a combination etiology. In the case of alcohol, this is termed MetALD or ALD, depending on the extent of alcohol intake. Adapted with permission from[2] Rinella ME, Lazarus JV, Ratziu V, Francque SM, Sanyal AJ, Kanwal F, et al. A multi-society Delphi consensus statement on new fatty liver disease nomenclature. Hepatology. 2023;78:1966-86. Abbreviations: ALD, alcohol-associated liver disease; BMI, body mass index; BP, blood pressure; CMRF, cardiometabolic risk factors; MASLD, Metabolic dysfunction–associated steatotic liver disease; MetALD, metabolic dysfunction and alcohol-associated steatotic liver disease; WC, waist circumference.

liver disease (ALD) with or without metabolic risk factors, drug-induced liver injury, monogenic diseases, and other etiologies (Figure 1).

RATIONALE BEHIND THE TERM MASLD TO REPLACE NAFLD

A supermajority of the Delphi panel concluded that a term that referenced the underlying disease pathophysiology was preferable. The panel considered multiple terms other than MASLD including visceral adiposity-associated steatotic liver disease, lipotoxic liver disease, nutrition-associated steatotic liver disease, and insulin-resistance–associated steatotic liver disease. The top 3 acronyms that emerged from the 4th Delphi round were MASLD, MetSLD, and metabolic steatotic liver disease. These results were presented to an independent multidisciplinary external committee which opted for the term MASLD to replace NAFLD. The definition of MASLD was designed to be broad to avoid the selection of a population that differed from NAFLD in its natural history. Under the new definition, patients with hepatic steatosis, one or more CMRFs, and no other discernible cause of steatosis would be classified as having MASLD. The CMRFs are based on well-established and validated criteria in the context of cardiovascular disease and are adjusted by ethnicity (Figure 2).[3]

TABLE 1 Key points regarding the new nomenclature and the 2023 AASLD practice guidance on the clinical assessment and management of NAFLD

• MASLD replaces the term NAFLD and MASH replaces NASH.
• NAFL can be replaced with the term metabolic dysfunction–associated steatotic liver (MASL)
• Studies suggest a near complete overlap (99%) between the MASLD-defined population and the historical NAFLD-defined populations.
• All recommendations in the AASLD Practice Guidance on the clinical assessment and management of NAFLD can be applied to patients with MASLD and MASH.
• Results from natural history and biomarker validation studies among patients with NAFLD and NASH are applicable to patients with MASLD and MASH, respectively until further guidance.
• The new nomenclature includes the MetALD category to identify patients with hepatic steatosis, cardiometabolic risk factors, and increased alcohol consumption (20/30 g to 50/60 g daily in females and males, respectively) instead of classifying them as patients with both MASLD and more than mild alcohol consumption.
• Future studies of MetALD are needed to provide insight into outcomes, biomarker performance and response to therapeutics.

Abbreviations: MASH, metabolic dysfunction–associated steatohepatitis; MASLD, metabolic dysfunction–associated steatotic liver disease; MetALD, metabolic dysfunction and alcohol-associated steatotic liver disease.

OVERLAP BETWEEN POPULATIONS IDENTIFIED AS HAVING MASLD AND NAFLD

While the definition of MASLD is distinct from that of NAFLD, several studies report a nearly complete overlap between the MASLD-defined population and the historical NAFLD-defined populations. For example, Song et al[4] recently found a minimal difference in the population prevalence between NAFLD (25.7%) and MASLD (26.7%) in a random subset of 1016 persons from Hong Kong examined with proton-magnetic resonance spectroscopy. Similar results were reported from the Association Française pour l'Etude du Foie—French Association for Study of the Liver Group for the Study of Liver Fibrosis that included 2187 patients seen in 5 French tertiary care centers, with >98% of their data set meeting MASLD criteria.[5] Diagnostic accuracy

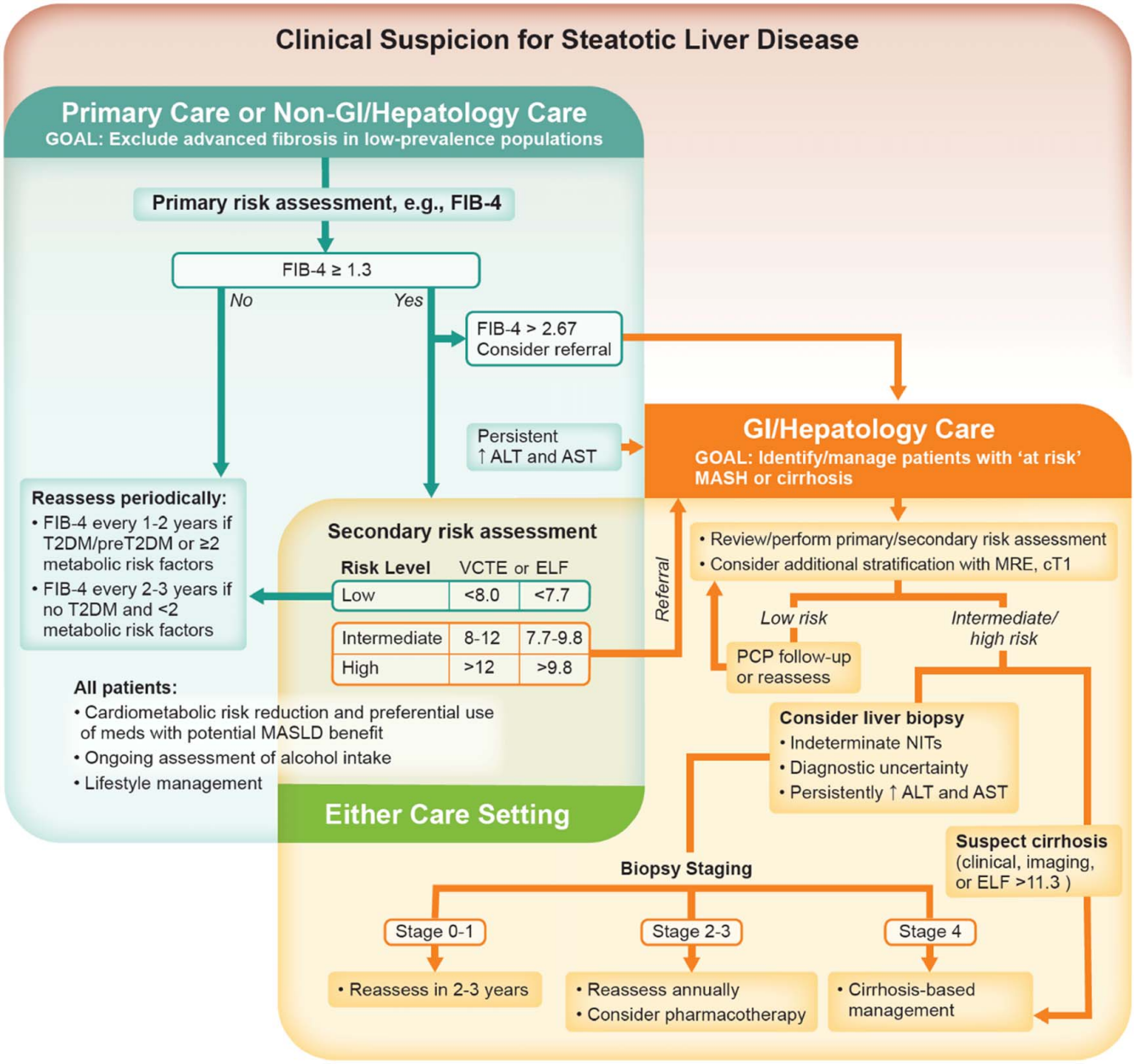

FIGURE 3 Algorithm for the evaluation of patients at risk for or with established SLD across practice settings. Patients with steatosis noted on imaging or for whom there is a clinical suspicion of MASLD, such as those with metabolic risk factors or unexplained elevation in liver chemistries, should undergo further evaluation. In low-prevalence settings, such as in the primary care setting, the emphasis is on excluding advanced fibrosis using a test with a high negative predictive value. When the FIB-4 is <1.3, patients can be followed in the primary care setting and reassessed periodically. Patients without prediabetes/T2DM and < 2 metabolic risk factors can be reassessed every 2–3 years. Patients with prediabetes/T2DM or 2 or more metabolic risk factors are at higher risk for disease progression, and more frequent FIB-4 monitoring (eg, every 1–2 y) should be considered. In patients older than age 65, an FIB-4 cutoff of > 2.0 should be used. FIB-4 has low accuracy in those under the age of 35 years; thus, secondary assessment should be considered in those <35 years of age with increased metabolic risk or elevated liver chemistries. FIB-4 should not be used in acutely ill patients. In patients with FIB-4 ≥ 1.3, a secondary assessment should be done (preferentially VCTE or ELF initially) or the patient referred for further risk stratification (if being seen in a non-gastroenterology/hepatology setting). Direct referral to gastroenterology/hepatology should be considered in those with aminotransferases persistently (> 6 mo) above normal to exclude other causes of liver disease or when FIB-4 > 2.67 due to the increased risk of clinically significant fibrosis. In higher prevalence settings, such as gastroenterology/hepatology clinics, additional risk assessment with MRE may be appropriate when NITs are indeterminate or there is clinical suspicion of more advanced disease. Identification of cirrhosis should prompt screening for HCC and esophageal varices. Additionally, MRE or corrected T1 (cT1) may help identify patients with "at-risk" MASH (MASH with NAFLD activity score ≥ 4 and fibrosis stage ≥ 2) who may benefit from a therapeutic intervention as they become available. If cirrhosis is suspected based on NITs, clinical data, or imaging findings, then cirrhosis-based management may be initiated without a liver biopsy. Liver biopsy should be considered when NITs suggest significant fibrosis (≥ F2), especially if additional evaluation suggests the presence of "at-risk" MASH (eg, using FAST, MEFIB, MAST, or cT1), NIT assessment is indeterminate, aminotransferases are persistently elevated (> 6 mo), or additional/alternate diagnoses are suspected. Note that in patients with confirmed or suspected advanced fibrosis, an ELF ≥ 11.3 is a predictor of future liver-related events and is approved for this purpose; the use of other ELF cutoffs in secondary risk assessment is based on expert option. Patients

at all stages of disease should be counseled on lifestyle modifications, and those with ≥F2 fibrosis targeted for pharmacologic interventions as they become available. Specific threshold values of NITs are approximations supported by current evidence and are meant to guide clinical management through primary care to gastroenterology/hepatology practices rather than be interpreted in isolation. Adapted with permission from[1] Rinella ME, Neuschwander-Tetri BA, Siddiqui MS, Abdelmalek MF, Caldwell S, Barb D, et al. AASLD Practice Guidance on the clinical assessment and management of nonalcoholic fatty liver disease. Hepatology. 2023;77:1797–835. Abbreviations: ALT, alanine aminotransferase; AST, aspartate aminotransferase; ELF, Enhanced Liver Fibrosis; FIB-4, fibrosis-4 index; MASH, metabolic dysfunction–associated steatohepatitis; MASLD, metabolic dysfunction–associated steatotic liver disease; MRE, magnetic resonance elastography; NIT, noninvasive test; PCP, primary care provider; SLD, steatotic liver disease; T2DM, type 2 diabetes mellitus; VCTE, vibration-controlled elastography.

of vibration-controlled transient elastography and FIB-4 for risk stratification into low, intermediate, or high risk for advanced fibrosis in the MASLD-defined population was almost identical to their performance in the NAFLD population. In another study of 1333 patients with NAFLD seen at 3 Swedish university hospitals, only 4 patients (0.3%) did not meet the criteria for MASLD.[6] The most common cardiometabolic conditions were a BMI ≥25 kg/m^2 (88.5%) and hypertension (83.6%), and the least prevalent condition was low serum HDL -cholesterol (58.7%). Outcomes were essentially identical: at 10 years after diagnosis, 7.9% of patients with NAFLD had developed a liver-related outcome compared with 7.8% of patients with MASLD, respectively. Furthermore, at 10 years, 10.4% of patients with NAFLD had died compared with 10.3% of patients with MASLD, respectively. Collectively, these data suggest that results from natural history and biomarker validation studies among patients with NAFLD may be applied to patients with MASLD (Table 1).

Commensurate with the change from NAFLD to MASLD, NASH was replaced with metabolic dysfunction–associated steatohepatitis (MASH). The histologic diagnosis of steatohepatitis remains unchanged. MASLD that does not meet criteria for MASH could be referred to as metabolic dysfunction–associated steatotic liver (MASL) just as NAFL was used to describe NAFLD that was not NASH, though this was not explicitly addressed in the Delphi consensus statements or manuscript.[2] Of note, the definitions of MASH and MASL traditionally required a liver biopsy for histologic identification of the presence or absence of steatohepatitis. However, clinical practice has shifted away from biopsy toward using biomarkers to noninvasively categorize MASLD severity. Given this, in most clinical settings, MASL could be inferred when noninvasive tests suggest the absence of steatohepatitis, though this needs further biomarker validation.

COEXISTENCE OF MORE THAN ONE DISEASE DRIVER

A benefit of using nonexclusionary terminology is that one is able to recognize the existence of more than one disease driver in a given patient. The new nomenclature allows for the coexistence of other forms of liver disease with MASLD, for example, MASLD and autoimmune hepatitis or MASLD and viral hepatitis. These patients are considered to have a dual pathology and should be studied separately in terms of their natural history, diagnostic markers, and treatment.

The definition of MASLD excludes patients with consumption of >20 g/30 g of alcohol per day in females and males, respectively. The Nomenclature Initiative posited several questions to the panelists to better understand the impact of alcohol on the natural history of the disease and also how to characterize various levels of alcohol use in the definition. Panelists in the Delphi process were nearly unanimous in their agreement that consumption of 30–60 g of alcohol daily in the setting of steatosis and CMRFs alters the natural history of the disease (95%) and may alter the response to therapeutic interventions (90%). Under the new nomenclature, patients with hepatic steatosis, CMRF, and alcohol use (at weekly intake of 140–350 g in women and 210–420 g in men or an average daily 20–50 g in women and 30–60 g in men) are classified as having MetALD. The new nomenclature recognizes the impact of the presence of both driving factors, which are often synergistic,[7,8] on disease progression, and patients with MetALD should be studied separately as they may have faster progression of their liver disease than those with MASLD. While precise cutoffs were provided for the purposes of research to better understand disease natural history and response to therapeutics, clinically, the overlay between ALD and MASLD represents a dynamic spectrum ranging from MASLD predominant at the lower level of alcohol use (weekly intake closer to 140 g in women and 210 g in men) to ALD predominant at higher level of alcohol use (weekly intake of up to 350 g in women and 420 g in men) (Figure 1). This explicit recognition of the coexistence of CMRF and alcohol use should also serve to elevate the need to address both cardiometabolic risk and excess alcohol consumption in patient management.[8] Specifically, both excessive alcohol use and CMRFs should be addressed to provide optimal care for the patient. Patients with steatosis who consume alcohol in excess of 50 g (females) or 60 g (males) daily, or weekly equivalent,

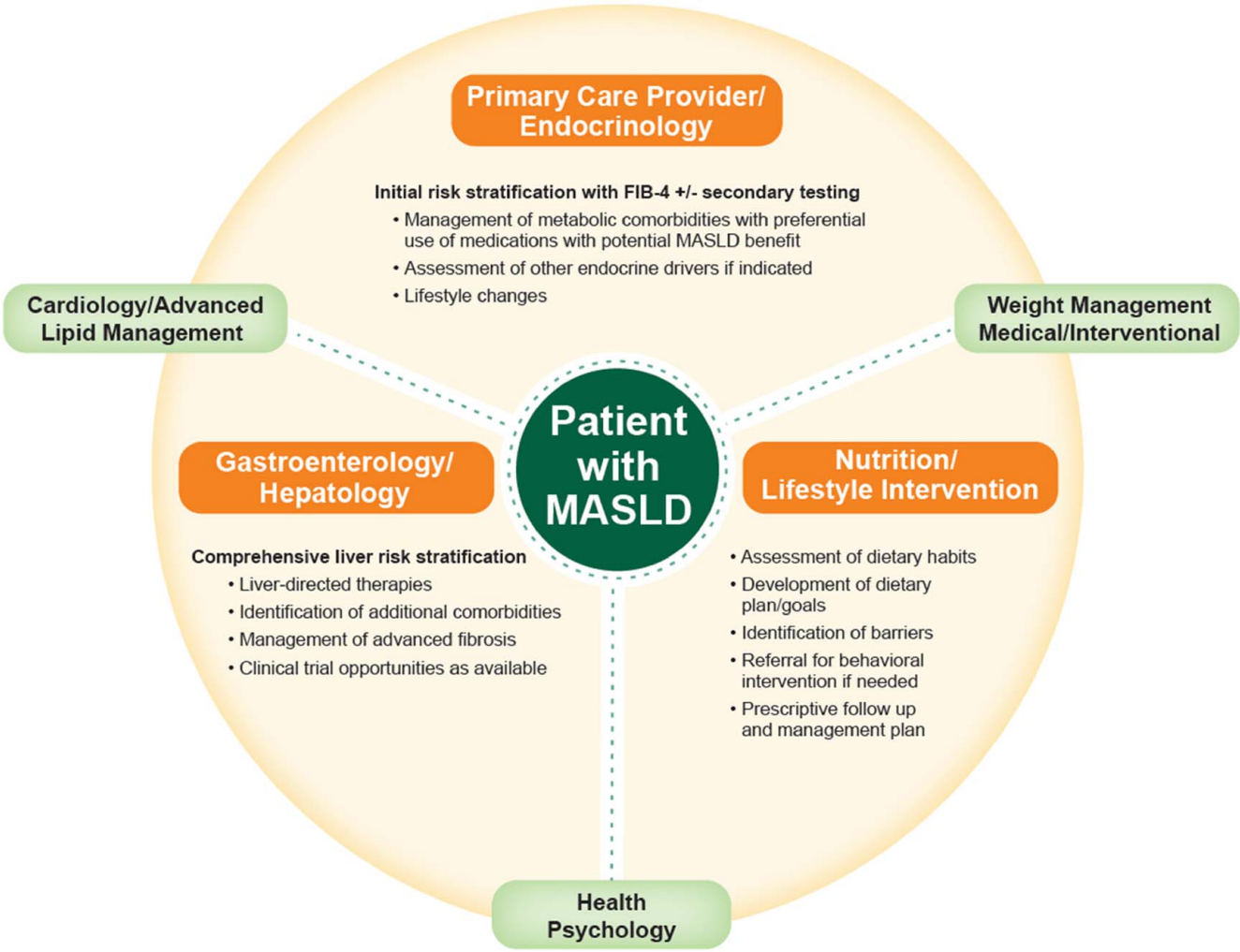

FIGURE 4 A multidisciplinary approach to the management of MASLD. Optimal care of the patient with MASLD requires a multidisciplinary approach. The majority of patients are in the primary care/endocrine setting, in which the management of medical comorbidities should be optimized, with preference given to treatments for type 2 diabetes mellitus, hypertension, or obesity that likely also have beneficial effects on MASLD. In this setting, at-risk patients should be identified and initial risk stratification performed (ie, FIB-4 ± vibration-controlled elastography or enhanced liver fibrosis test (ELF)). The role of the gastroenterologist/hepatologist includes more comprehensive liver risk stratification, exclusion of other liver diseases, and a focus on liver-directed therapy. Close communication between gastroenterology/hepatology and primary care or endocrinology facilitates multidisciplinary management of metabolic comorbidities as well as the prioritization of medications or interventions that may also offer liver benefits (see Treatment section in AASLD NAFLD Guidance).[2] All patients should undergo dietary/nutritional assessment and a plan established for regular follow-up independent of gastroenterology/hepatology visits. The need for more specialized obesity management, including bariatric surgery referral, health psychology, and additional cardiology or lipid metabolic support, should be assessed on an individual basis (dotted arrows). Adapted with permission from[1] Rinella ME, Neuschwander-Tetri BA, Siddiqui MS, Abdelmalek MF, Caldwell S, Barb D, et al. AASLD Practice Guidance on the clinical assessment and management of nonalcoholic fatty liver disease. Hepatology. 2023;77:1797–835. Abbreviations: FIB-4, fibrosis-4 index; MASLD, metabolic dysfunction–associated steatotic liver disease.

are classified as having ALD since alcohol is likely to be the dominant driver of liver disease. Importantly, CMRFs are commonly present in patients with ALD, and both alcohol cessation and CMRF management need to be addressed.[9]

SPECIFIC ETIOLOGY SLD

In the absence of overt CMRFs, other etiologies must be excluded. These include drug-induced liver injury, lysosomal acid lipase deficiency, Wilson disease, hypobetalipoproteinemia, inborn errors of metabolism, HCV genotype 3 infection, malnutrition, celiac disease, and HIV and environmental exposure to agents associated with steatosis such as hydrocarbon inhalation.[10,11]

CRYPTOGENIC SLD

The nomenclature allows for the use of the term cryptogenic SLD if no etiology is identified and no CMRFs are present, although, depending on clinical judgment, it could also be deemed to be possible

MASLD and, thus, these patients could benefit from periodic reassessment on a case-by-case basis for the development of CMRFs. In the setting of advanced fibrosis/cirrhosis, steatosis may be absent, requiring clinical judgment based on CMRFs and the absence of other etiologies.

IMPACT OF THE NEW NOMENCLATURE ON RECOMMENDATIONS INCLUDED THE AASLD PRACTICE GUIDANCE ON THE CLINICAL ASSESSMENT AND MANAGEMENT OF NAFLD

We believe that all of the recommendations in the AASLD Practice Guidance on the Clinical Assessment and Management of NAFLD can be applied to adults with MASLD (Figures 3 and 4).[1] Given the >99% overlap between patients identified as having NAFLD with those meeting the criteria for MASLD (Figure 2), it may be only the rare patient with steatosis who does not have any CMRFs who might be considered separately in terms of evaluation and recommendations. Similarly, the terms in the guidance NASH, at-risk NASH, NAFL, and NAFLD/NASH cirrhosis can be replaced by MASH, at-risk MASH, MASL, and MASLD/MASH cirrhosis, respectively. The major update to the Guidance is related to the concept of MetALD. Whereas the Guidance addresses patients with NAFLD and more than mild alcohol consumption (>20/30 g/d in females and males, respectively), the new nomenclature separates these patients under the MetALD category instead of classifying them as patients with MASLD and more than mild alcohol consumption. Future studies of MetALD can stratify and examine patients according to their metabolic dysfunction and amount of alcohol consumption (Table 1). The impact of the nomenclature change on pediatric NAFLD will be covered in the future pediatric Guidance.

FUTURE DIRECTIONS/AREAS FOR ADDITIONAL RESEARCH

The new nomenclature facilitates research across several dimensions. The natural history of the small subgroup of patients with SLD but no overt CMRF requires further clarification to better understand their risk of developing CMRF over time. The liver may be an early target of metabolic dysfunction and the development of SLD could be an early harbinger of subsequent manifestations of metabolic dysfunction.[11] Another important area is to better understand the intersection of CMRF with varying degrees of alcohol consumption to determine the relative roles of CMRFs, underlying genetic variants, patterns of alcohol consumption (eg, moderate daily vs. weekly binges and accounting for the impact of historical alcohol exposure), and lifestyle modifications to alter the impact of CMRFs on the clinical course and outcomes of patients with SLD across its spectrum (Table 1).[12–14]

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CONFLICTS OF INTEREST

Brent A. Neuschwander-Tetri is an advisor or consultant for Akero, Arrowhead, Boehringer-Ingelheim, Corcept, GSK, Hepion, HistoIndex, Madrigal, Merck, Mirum, Sagimet, and Senseion; Stock options: HepGene and HeptaBio; Institutional research grants: BMS, HighTide, Intercept, Inventiva, and Madrigal. Rohit Loomba serves as a consultant to Aardvark Therapeutics, Altimmune, Arrowhead Pharmaceuticals, AstraZeneca, Cascade Pharmaceuticals, Eli Lilly, Gilead, Glympse bio, Inipharma, Intercept, Inventiva, Ionis, Janssen Inc., Lipidio, Madrigal, Neurobo, Novo Nordisk, Merck, Pfizer, Sagimet, 89 bio, Takeda, Terns Pharmaceuticals, and Viking Therapeutics. In addition, his institution received research grants from Arrowhead Pharmaceuticals, Astrazeneca, Boehringer-Ingelheim, Bristol-Myers Squibb, Eli Lilly, Galectin Therapeutics, Gilead, Intercept, Hanmi, Intercept, Inventiva, Ionis, Janssen, Madrigal Pharmaceuticals, Merck, Novo Nordisk, Pfizer, Sonic Incytes, and Terns Pharmaceuticals. Cofounder of LipoNexus Inc. Mary E. Rinella is an advisor or consultant for Boehringer-Ingelheim, Sonic Incytes, GSK, HistoIndex, Madrigal, Intercept, Cytodyn, and Novo Nordisk. Institutional research grants: Inventiva, Madrigal, and Akero. Fasiha Kanwal has no conflicts to report.

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