Adoption of the New Nomenclature of Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD) by the Indian National Association for Study of the Liver (INASL): Implications for the INASL Guidance Paper on NAFLD

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The transition from nonalcoholic fatty liver disease (NAFLD) to metabolic dysfunction-associated steatotic liver disease (MASLD) reflects a paradigm shift in hepatology, emphasising metabolic dysfunction as the central driver in patients with MASLD. This inclusive terminology, endorsed by over 70 international organisations including the Indian National Association for Study of the Liver (INASL), reduces stigma of 'fatty and alcohol' and allows the co-existence of other liver disease etiologies along with MASLD. In the present commentary, we discuss the implications of the adoption of new nomenclature of MASLD on the INASL guidance paper on NAFLD, which was published in 2023, before the Delphi consensus on MASLD. (J Clin Exp Hepatol 2025;15:102590)

MASLD

The debate on the nomenclature of nonalcoholic fatty liver disease (NAFLD) was sparked off by a panel of international experts in 2020 who proposed the rechristening of NAFLD to metabolic dysfunction-associated fatty liver disease (MAFLD).[1] This led to a fierce debate in the hepatology community and several issues were raised with both nomenclatures.[2–4] The Indian National Association for Study of the Liver (INASL)

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Abbreviations: ALD: alcohol-related liver disease; BMI: body mass index; CAP: controlled attenuation parameter; HCC: hepatocellular carcinoma; HDL: high-density lipoprotein; ICOM-D: Indian Consortium on MASLD; INASL: Indian National Association for Study of the Liver; LSM: liver stiffness measurement; MASL: metabolic dysfunction-associated steatotic liver; MASH: metabolic dysfunction-associated steatohepatitis; MASLD: metabolic dysfunction-associated steatotic liver disease; MetALD: MASLD with increased alcohol intake; NAFL: nonalcoholic fatty liver; NAFLD: nonalcoholic fatty liver disease; NASH: nonalcoholic steatohepatitis; NITs: noninvasive tests; SLD: steatotic liver disease; USG: ultrasound
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Guidance Paper on Nomenclature, Diagnosis and Treatment of Nonalcoholic Fatty Liver Disease (NAFLD) in 2023 suggested NAFLD as the preferred terminology instead of MAFLD due to a lack of compelling evidence and wide consensus to back a name change.[5] Subsequent to the publication of the guidance document by INASL, a Delphi consensus meet of multiple international hepatology societies was conducted in 2023 which suggested the new name of metabolic dysfunction-associated steatotic liver disease (MASLD).[6] In brief, the putative advantages of this new nomenclature of MASLD include emphasis on metabolic dysfunction as the central pathophysiologic driver of the disease process, an inclusive positive definition based upon well-established cardiometabolic risk factors without needing specialised investigations, recognition of the possible co-existence of additional etiologies, and avoidance of stigmatising terms like "fatty" and "alcoholic".[6] Unlike MAFLD criteria, the MASLD definition does not include investigations like high-sensitivity C-reactive protein and Homeostatic Model Assessment of Insulin Resistance, which are seldom performed in Indian patients with NAFLD. This is particularly relevant in non-diabetic patients with normal body mass index (BMI), so called lean NAFLD who are better categorised using MASLD definition than MAFLD criteria.[7,8]

After the change in the nomenclature from NAFLD to MASLD, there was speculation that it may not be appropriate or possible to extrapolate the NAFLD data on epidemiology, noninvasive tests (NITs), biomarkers, natural history and clinical trials to the MASLD population, and new studies using proposed MASLD criteria would be required after adoption of the MASLD nomenclature. However, data from USA, Europe, Asia–Pacific region, and India suggest that there are more than 90–99% similarities between NAFLD and MASLD cohorts and repeat studies are not required after the change in nomenclature.[8–16]

After the publication of the Delphi consensus on MASLD,[6] more than 70 societies across the globe, including INASL, have endorsed the new nomenclature of MASLD. The present commentary addresses the implications of the adoption of MASLD on the clinical utility of INASL Guidance Paper on Nomenclature, Diagnosis and Treatment of Nonalcoholic Fatty Liver Disease (NAFLD),[5] which was published before the international Delphi consensus. Using the Delphi technique, additional recommendations based on the differences in the INASL Guidance paper on NAFLD and the newly suggested MASLD criteria were made by the working party which have been detailed in this short commentary. The GRADE system was followed to make the grade of recommendations and level of evidence as shown in Table 1.

THE NEW NOMENCLATURE IN A NUTSHELL

Steatotic liver disease (SLD) has been suggested as an overarching term for replacing the stigmatising term "fatty liver disease". SLD may be attributed to various causes including MASLD, alcohol-related liver disease (ALD), other etiologies (e.g. drug-induced liver injury, hepatitis C genotype 3, Wilson's disease, monogenic causes, etc), a combination of different etiologies [including MASLD with increased alcohol intake (MetALD)] or may be cryptogenic SLD (Figure 1).[6]

Table 1 Level of Evidence and Grade of Recommendations (Adapted From GRADE System).

Level of evidence[a]		**Confidence in the evidence**
High	Data derived from meta-analyses or systematic reviews or from (multiple) randomised trials with high quality.	Further research is unlikely to change our confidence in the estimate of benefit and risk.
Moderate	Data derived from a single Randomized controlled trial (RCT) or multiple non-randomised studies.	Further research (if performed) is likely to have an impact on our confidence in the estimate of benefit and risk and may change the estimate.
Low	Small studies, retrospective observational studies, registries.	Any estimate of effect is uncertain.
Recommendations—grade[b]		**Wording associated with the grade of recommendation**
Strong Factors influencing the strength of the recommendation included the quality of the evidence, presumed patient-important outcomes, and cost		''must", ''should", or ''INASL recommends"
Weak Variability in preferences and values, or more uncertainty. Recommendation is made with less certainty, higher cost or resource consumption		''can", ''may", or ''INASL suggests"

INASL, Indian National Association for Study of the Liver.
[a]Level was graded down if there was a poor quality, strong bias, or inconsistency between studies; Level was graded up if there was a large effect size.
[b]Recommendations reached by consensus of the 'Working Party' and included the quality of evidence, presumed patient-important outcomes, and costs.

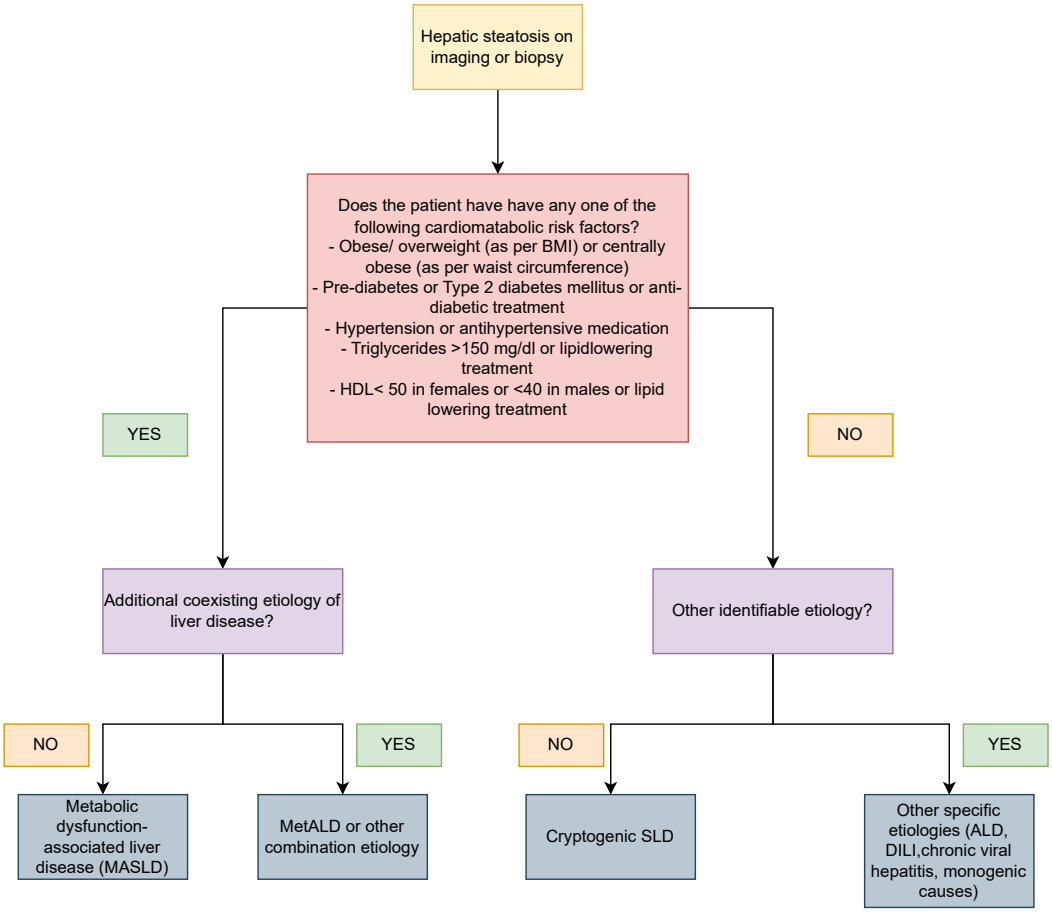

Figure 1 Framework for classifying metabolic dysfunction-associated steatotic liver disease (MASLD) under the overarching umbrella of steatotic liver disease (SLD).[6]

MASLD should be diagnosed as a cause of SLD in the presence of any one of the five cardiometabolic risk factors shown in Table 2 and in the absence of significant alcohol intake. MASLD is thus no longer a diagnosis of exclusion and can co-exist with other etiologies. The terms metabolic dysfunction-associated steatotic liver (MASL) and metabolic dysfunction-associated steatohepatitis (MASH) have been proposed to replace nonalcoholic fatty liver (NAFL) and nonalcoholic steatohepatitis (NASH), respectively.[6]

IMPLICATIONS OF THE NEW NOMENCLATURE IN CLINICAL PRACTICE

Diagnosis

It is important to realise that unlike NAFLD, MASLD is not a diagnosis of exclusion.[5,6] Thus, the presence of at least one of the five cardiometabolic risk factors mentioned in Table 2 is mandatory for making the diagnosis of MASLD in a patient with hepatic steatosis who does not

Table 2 Cardiometabolic Risk Factors for Defining MASLD in Indian adults.

At least one of the following five criteria should be present:
1. Overweight/obese (as per BMI[a]) or central obesity (as per waist circumference[b])
2. Fasting serum glucose ≥100 mg/dL or 2-h post load serum glucose ≥140 mg/dL or HbA1c ≥ 5.7% or type 2 diabetes mellitus or anti-diabetic treatment
3. Blood pressure ≥130/85 mm of Hg or antihypertensive medication
4. Plasma triglycerides ≥150 mg/dl or lipid lowering treatment
5. Plasma HDL ≤50 in females or ≤40 in males or lipid lowering treatment

BMI, body mass index; HDL, high-density lipoprotein; MASLD, metabolic dysfunction-associated steatotic liver disease.
[a]BMI cut-off for overweight and obesity in Indians: ≥23 kg/m^2 and ≥25 kg/m^2, respectively.
[b]Waist circumference cut-offs for central obesity in Indians: >90 cm in males and >80 cm in females.

consume significant amounts of alcohol. In the Delphi consensus paper, non-significant alcohol intake has been defined as <20 g/day in females and <30g/day in males, in accordance with western cut-offs. Traditionally, non-significant alcohol intake in Indian patients has been defined as <20g/day irrespective of gender.[5,17] However, this figure was based on expert opinion without substantial supporting data. To maintain conformity with the globally accepted criteria, we suggest retaining the cut-offs of non-significant alcohol intake as suggested in the Delphi consensus (i.e. <20 g/day in females and <30 g/day in males). In the Indian context, MASLD can thus be diagnosed in a patient with hepatic steatosis in the presence of at least one of the five cardiometabolic risk factors and alcohol consumption <20g/day in females and <30g/day in males. Patients without cardiometabolic risk factors should be evaluated for other causes of SLD including ALD, hepatitis B and C, Wilson's disease, celiac disease, HIV, and other monogenic diseases depending upon the clinical scenario.

As detailed in the INASL guidance document on NAFLD,[5] abdominal ultrasound (USG) is the initial screening modality of choice for detection of hepatic steatosis despite higher accuracy of controlled attenuation parameter (CAP) and magnetic resonance imaging–based techniques, primarily because of the widespread availability and low cost of USG. However, the major limitation for USG as the initial screening modality for steatotic liver disease (SLD), which is the new umbrella term in the Delphi consensus,[6] is that it starts detecting hepatic steatosis after it has reached close to 33%, whereas the definition of hepatic steatosis as defined on liver histology is when more than 5% of hepatocyte get laden with fat. The caveats in establishing the etiology of NAFLD in patients with cirrhosis with or without hepatocellular carcinoma (HCC) are also applicable for MASLD. These include the tendency for hepatic steatosis to decrease with advancing fibrosis and the variable impact of cirrhosis *per se* on cardiometabolic comorbidities.[5] For instance, weight loss is common in cirrhosis and systemic vasodilatation in end-stage liver disease may mask years of hypertension. Cirrhosis itself may lead to the onset of hepatogenous diabetes and it also impacts lipid profile with decrease in serum triglycerides and high-density lipoprotein (HDL). Thus, the past history and duration of metabolic comorbidities should be considered and not just the current status. As such, the diagnosis of MASLD cirrhosis or HCC should be considered in patients with presence or history of metabolic comorbidities without significant alcohol intake even in the absence of demonstrable steatosis.[5,6,17,18]

Recommendations

1. INASL endorses and recommends the terminology of SLD, MASLD, MASL, and MASH instead of NAFLD, NAFL, and NASH. The presence of at least one cardiometabolic risk factor is required for establishing the diagnosis of MASLD in a patient with hepatic steatosis and absence of significant alcohol intake (i.e. <20 g/day in females and <30g/day in males). (*Grade of Recommendation—Strong)*
2. INASL suggests that, in patients with current or past history of multiple cardiometabolic risk factors, the diagnosis of MASLD as the etiology of cirrhosis or HCC may be made even in the absence of demonstrable hepatic steatosis. *(Level of Evidence—Low, Grade of Recommendation—Weak)*

Possibility of Multiple Etiologies

The non-exclusionary nature of the new MASLD definition permits its possible co-existence with additional etiologies of hepatic steatosis or deranged liver functions. For instance, MASLD may co-exist with chronic viral hepatitis or autoimmune hepatitis. A noteworthy corollary is that depending upon the clinical scenario, the diagnosis of MASLD should not preclude a search for alternate etiologies. At the very least, it would be prudent to look for chronic viral hepatitis with HBsAg and anti-HCV in all patients, particularly in those with elevated transaminases. Additional etiological investigations should be tailored according to the individual clinical setting.

Among the various possible combinations, specific mention needs to be made about SLD patients with both cardiometabolic risk factors and significant alcohol consumption. It is well-known that alcohol consumption and metabolic comorbidities, the two most common drivers of hepatic steatosis, often co-exist and may have a synergistic role in promoting liver disease progression.[19–22] Patients with hepatic steatosis and significant but moderate alcohol consumption (males: >30g/day to <60 g/day or >210 g/week to <420 g/week; females: >20g/day to <50 g/day or >140 g/week to <350 g/week) who have at least one cardiometabolic risk factor are categorised as MetALD.[6] However, patients with more than moderate alcohol consumption have been categorised as ALD in the Delphi consensus even if cardiometabolic risk factors are present. Nonetheless, it should be recognised that the presence of cardiometabolic risk factors, particularly obesity, in patients with ALD has been associated with more advanced liver disease and worse outcomes including in those presenting with ALD-related acute-on-chronic liver failure and severe alcoholic hepatitis.[22] These patients with heavy alcohol intake and presence of metabolic risk factors are defined as ALD with metabolic dysfunction.

Recommendations

1. MASLD is not a diagnosis of exclusion and can co-exist with additional etiologies (*Grade of Recommendation—Strong).*

2. INASL endorses the terminology of MetALD. INASL recommends that patients with SLD and significant but moderate alcohol consumption (males: >30g/day to <60 g/day or >210 g/week to <420 g/week; females: >20g/day to <50 g/day or >140 g/week to <350 g/week) who have at least one cardiometabolic risk factor should be categorised as MetALD. Patients with higher alcohol intake (males: >60 g/day or >420 g/week; females: >50 g/day or >350 g/week) should be categorised as ALD and in the presence of cardiometabolic risk factors should be defined as ALD with metabolic dysfunction. *(Level of Evidence—Low, Grade of Recommendation—Weak)*

Investigations and Management

Despite the differences inherent in their definitions, practically speaking, there is an almost complete overlap between NAFLD and MASLD in the real world. Data from multiple cohorts have shown that >89–99% of patients with NAFLD fulfil the MASLD criteria.[8–16] Furthermore, the long-term natural history of NAFLD and MASLD is likely to be similar. Indeed, in a Swedish cohort, liver-related outcomes and mortality at 10 years were seen in 7.9% and 10.4% of patients with NAFLD, respectively, compared with 7.8% and 10.3% of patients who satisfied the MASLD criteria.[9] Finally, noninvasive tests like fibrosis-4 (FIB-4) and liver stiffness measure (LSM) with

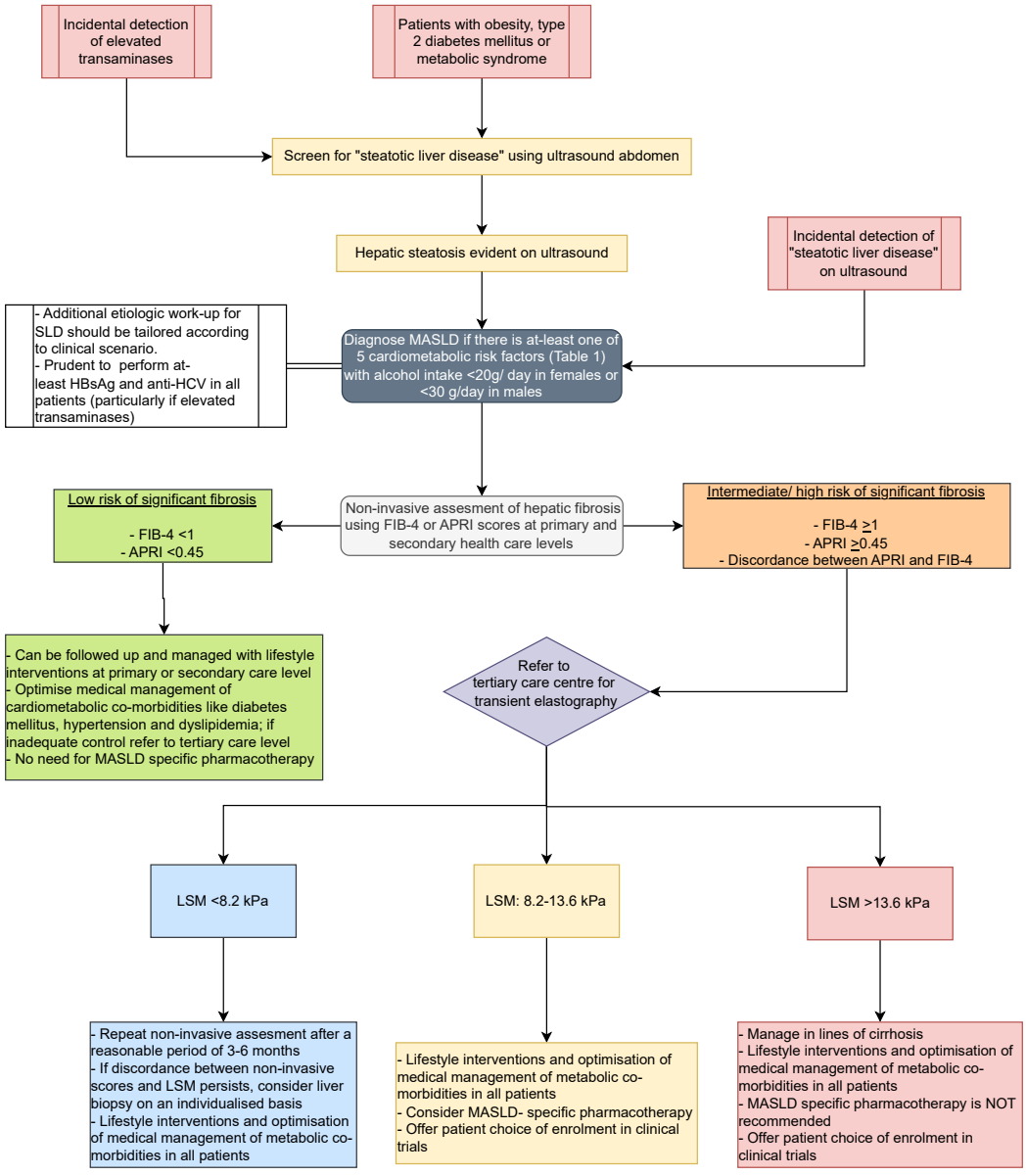

Figure 2 Algorithm for screening, diagnosis, noninvasive risk stratification, referral pathways, and management of MASLD in Indian settings. MASLD, metabolic dysfunction-associated steatotic liver disease.

vibration-controlled transient elastography or FibroScan (Echosens, Paris) were shown to have comparable diagnostic accuracy for stratifying fibrosis risk in patients with NAFLD and those fulfilling MASLD definition in a multicentric review from France.[14] Similar observations have been corroborated in Indian patients. A study of the Indian Consortium on MASLD (ICOM-D) showed that among 7721 patients with MASLD, 7568 (98%) fulfilled MASLD criteria. This study also reported similar clinical phenotypes, disease severity, and diagnostic accuracy of commonly used noninvasive tests like FIB-4 and LSM among patients with NAFLD and MASLD.[8] The vast majority (>85%) of Indian patients with lean NAFLD (i.e. patients with NAFLD who have normal BMI) also fulfil MASLD criteria due to the presence of either central obesity or one of the other four cardiometabolic risk factors (i.e. type 2 diabetes mellitus or pre-diabetes, hypertension, hypertriglyceridemia, or low HDL).[7,8]

Against this background of near universal overlap, noninvasive assessment, need for liver biopsy, risk stratification, referral pathways, and management of Indian adults with MASLD should be done along similar lines as that for NAFLD as detailed in the INASL guidance document.[5] Figure 2 summarises the tenets of screening, noninvasive assessment, risk stratification, referral, and management of MASLD in the Indian setting. The new nomenclature of MASLD emphasises metabolic dysfunction as the fundamental pathophysiological driver thereby highlighting the need for optimising management of metabolic comorbidities along with lifestyle interventions as the central pillars in the management of MASLD. Indications of disease-specific pharmacotherapy and drugs for NAFLD can be extrapolated to patients with MASLD without additional etiologies.

Needless to say, the alternate etiology should be appropriately tackled in patients with additional co-existent etiologies. This includes alcohol abstinence in patients with MetALD. While lifestyle interventions and control of metabolic comorbidities remain a priority even in patients with combination etiologies, the role of MASLD-specific pharmacotherapy like pioglitazone, vitamin E, saroglitazar, etc. in MetALD or other combination etiologies is uncertain and needs further research. Thus, routine use of MASLD-specific pharmacotherapy cannot be currently recommended in patients with MetALD or other co-existing etiologies in addition to MASLD.[23,24]

Recommendations

1. Given the near complete overlap between patients fulfilling NAFLD and MASLD criteria, INASL recommends that the work-up, risk stratification and management of MASLD should be along similar lines as NAFLD. *(Level of Evidence—High, Grade of Recommendation—Strong)*
2. INASL recommends holistic management with lifestyle interventions and adequate control of metabolic comorbidities in all patients with MASLD with or without concomitant additional etiologies. *(Level of Evidence—High, Grade of Recommendation—Strong)*
3. Appropriate management of additional etiology (if any) should be pursued including alcohol abstinence in MetALD. *(Level of Evidence—High, Grade of Recommendation—Strong)*
4. INASL recommends that indications of disease-specific pharmacotherapy and drugs for NAFLD can be extrapolated to patients with MASLD without additional etiologies. *(Level of Evidence—Low, Grade of Recommendation—Strong)*
5. Pending further evidence, INASL cannot make any recommendations for pharmacotherapy in patients with MetALD. *(Level of Evidence—Low, Grade of Recommendation—Weak)*

Paediatric MASLD

In a recent position statement, multiple paediatric gastroenterology and hepatology societies have endorsed the terminologies of SLD and MASLD.[25] While the overall principles of diagnosing SLD and MASLD in children is similar to that in adults, a few peculiarities need to be highlighted. First, the cut-offs of various cardiometabolic risk factors are less well defined in the paediatric population (Table 3). Secondly, the spectrum of SLD presenting to the paediatrician is different from what is seen by adult hepatologists or gastroenterologists. Thus, alcohol-related liver disease is hardly seen in children or adolescents while inborn errors of metabolism, autoimmune hepatitis, and Wilson's disease are common causes of SLD in them. A relatively more extensive evaluation is required at baseline in children and adolescents to rule out the alternate causes of SLD. At the least, this should include investigations for chronic viral hepatitis, autoimmune

Table 3 Cardiometabolic Risk Factors for Defining Paediatric MASLD.

At least one of the following five criteria should be present:
1 BMI ≥85th percentile for age/gender (BMI z score ≥1) or Waist circumference >95th percentile
2 Fasting serum glucose ≥100 mg/dL or 2-h post load serum glucose ≥140 mg/dL or HbA1c ≥ 5.7% or type 2 diabetes mellitus or anti-diabetic treatment
3 Blood pressure (BP): - Age <13 years: BP ≥ 95th percentile or 130/85 mm of Hg (whichever is lower) - Age ≥13 years: ≥130/85 mm of Hg Or antihypertensive medication
4 Plasma triglycerides: - Age <10 years: ≥100 mg/dl - Age ≥10 years: ≥150 mg/dl Or lipid lowering treatment
5 Plasma HDL ≤40 mg/dl or lipid lowering treatment

BMI, body mass index; HDL, high-density lipoprotein; MASLD, metabolic dysfunction-associated steatotic liver disease.

hepatitis, Wilson's disease, celiac disease, and possibly alpha-1-antitrypsin deficiency.[25] Further investigations should be tailored according to the clinical setting. A low threshold of suspicion for inborn error of metabolism is prudent and should be investigated judiciously depending on the clinical scenario. Finally, "metabolic liver disease" in paediatric hepatology usually refers to hepatic manifestations of inborn errors of metabolism. Thus, "metabolic-dysfunction" in MASLD may be initially confusing to the paediatrician. However, with time and increasing awareness of the terminology, this is not expected to be of much concern.

FUTURE RESEARCH AGENDAS

The new nomenclature of MASLD opens a gamut of research questions. Some of the important grey areas include the differential impact of the various cardiometabolic risk factors on risk and severity of MASLD, and a granular assessment of the interplay between alcohol and cardiometabolic risk factors. Apart from validation of the alcohol quantity for categorising MASLD, MetALD, and ALD, the role of other factors like genetic predispositions, patterns of alcohol intake (binge vs daily drinking), and duration of abstinence in apparently abstinent patients needs to be interrogated in detail. The dynamicity in alcohol intake and the limitation of history regarding quantity of alcohol intake may require the use of direct biomarkers of alcohol intake. The subgroup of lean NAFLD patients who do not fit the MASLD criteria and are likely to be categorised as cryptogenic SLD will also merit special attention to better define the pathophysiological drivers and natural history in this intriguing group. Evidence also needs to be generated on the role of MASLD-specific pharmacotherapy in patients with MetALD or MASLD with other co-existing etiology.

CREDIT AUTHORSHIP CONTRIBUTION STATEMENT

AD - Conceptualization, Writing - Original Draft, Writing - Review & EditingArD - Writing - Original Draft, Writing - Review & EditingFor all other authors - Review and Editing

DECLARATION OF COMPETING INTEREST

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