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REVIEW

Steatotic liver disease: a dynamic spectrum, not a static state

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Background

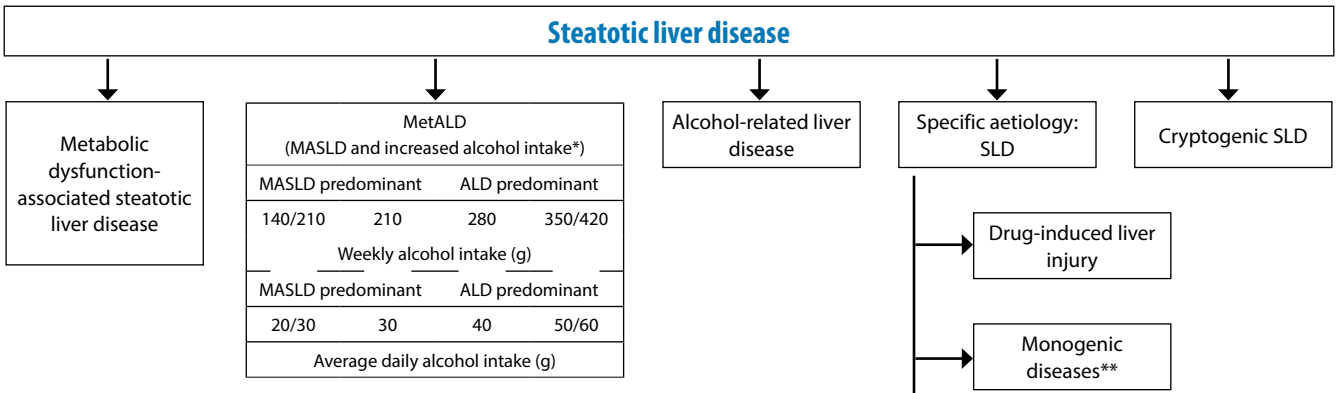
Steatotic liver disease (SLD) is a growing global health threat affecting more than 1 in 3 individuals and encompasses the spectrum of metabolic dysfunction-associated steatotic liver disease (MASLD), metabolic alcohol-related liver disease (MetALD), and alcohol-related liver disease (ALD) (Figure 1).[1] MASLD, MetALD and ALD are driven by the rising prevalence of type 2 diabetes (T2M) and obesity and increased alcohol consumption has led to a rising incidence of liver cirrhosis and hepatocellular cancer. SLD is now the leading indication for liver transplantation in many regions globally.[2]

In a meta-analysis of 44 studies (11 282 575 participants), the pooled prevalence of SLD in the global general population was 37.5% (95% confidence interval, 31.4%–44.1%; $I^2 = 99.8\%$).

Subtype analysis showed a prevalence of 33.6% (95% CI, 28.1%–39.5%; $I^2 = 99.9\%$) for MASLD, 4.1% (95% CI, 3.1%-5.3%; $I^2 = 100\%$) for MetALD, and 2.2% (95% CI, 1.5%–3.1%; $I^2 = 98.0\%$) for ALD. SLD prevalence was particularly elevated in individuals with type 2 diabetes (70.2%; 95% CI, 66.1%–73.9%; $I^2 = 65.4\%$) and in overweight/obese populations (70.7%; 95% CI, 43.2%-88.4%; $I^2 = 99.0\%$).[3]

Prevalence rates of MASLD, MetALD, and ALD are also consistently higher in men (18.5%, 3.2%, 1.7%) than in women (10.3%, 1.2%, 0.3%).[4] However, women are more vulnerable to alcohol-related liver injury and are twice as likely as men to develop severe ALD and progress to cirrhosis at lower levels of alcohol consumption over shorter exposure periods, possibly due to sex differences in gastric alcohol dehydrogenase (ADH) activity and body fat percentage.[5]

According to the 2023 Multisociety Delphi Consensus statement, MASLD, MetALD and ALD are clearly defined according to the presence of cardiometabolic risk factors and the amount of alcohol consumed (Figure 1).[6] MASLD, defined as SLD with



* Weekly intake: 140–350 g (F), 210–420 g (M); average daily intake: 20–50 g (F), 30–60 g (M).
** E.g. lysosomal acid lipase deficiency, Wilson's disease, hypobetalipoproteinaemia, inborn metabolic errors.
*** E.g. hepatitis C, malnutrition, coeliac disease.

MASLD adult diagnostic criteria
Hepatic steatosis plus at least 1 out of 5 Cardiometabolic risk factors:
- BMI ≥ 25 kg/m^2 (23 kg/m^2 for Asian ethnicity) **OR** waist circumference > 94 cm (M), 80 cm (F) **OR** ethnicity-adjusted equivalent.
- Fasting serum glucose ≥ 5.6 mmol/L **OR** two-hour post-load glucose levels ≥ 7.8 mmol/L **OR** HbA1c ≥ 5.7% **OR** T2D **OR** treatment for T2D.
- Blood pressure ≥ 130/85 mmHg **OR** specific antihypertensive drug treatment.
- Plasma triglycerides ≥ 1.70 mmol/L **OR** lipid-lowering treatment.
- Plasma high-density lipoprotein cholesterol ≤ 1.0 mmol/L (M), ≤ 1.3 mmol/L (F) **OR** lipid-lowering treatment.

Figure 1: Steatotic liver disease subclassification (adapted from: Hepatology 2023; 1;78(6):1966 and J Hepatol 2023;79(6):1542)
BMI – body mass index, F – female, M – male, MASLD – metabolic dysfunction-associated steatotic liver disease, MetALD – metabolic alcohol-related liver disease, SLD – steatotic liver disease, T2D – type 2 diabetes

at least one cardiometabolic risk factor, is increasingly being recognised as a comorbidity and potentially severe complication of the metabolic syndrome, with T2D and obesity being the main driving factors.[7] Importantly, alcohol use fluctuates across an individual's lifespan, driving dynamic transitions between MASLD, MetALD and ALD.

However, MASLD, MetALD and ALD are not distinct conditions but overlapping phenotypes, with alcohol and cardiometabolic risk factors (CMRFs) synergistically influencing disease progression, complicating diagnosis, and altering therapeutic strategies over time.[8]

The relationship between SLD and metabolic dysfunction and/or metabolic syndrome (MetS) is multifactorial and often bidirectional. SLD is associated with metabolic dysfunction preceding MetS, but may also reinforce or develop into MetS or its individual components.[7]

Most patients exhibit multiple CMRFs, including obesity, T2D, hypertension, and dyslipidaemia, and these coexisting drivers amplify fibrosis risk and cardiovascular mortality, irrespective of SLD subtype classification.[1] The median number of CMRFs per individual across the SLD spectrum is approximately three risk factors per individual. The risk of advanced fibrosis and mortality increases in proportion to the number of CMRFs present.[9,10] Not all CMRFs carry equal prognostic weight for developing liver-related outcomes in MASLD. T2D and abdominal obesity are stronger predictors of insulin resistance and fibrosis progression compared to elevated BMI alone.[11-13] In an NHANES-based analysis, individuals with elevated alcohol use and ≥ 3 CMRFs had more than a two-fold higher risk of advanced fibrosis, with T2D and waist circumference being the most influential determinants.[9] Adverse muscle-fat phenotypes such as sarcopaenic obesity and myosteatosis are increasingly recognised to be associated with advanced fibrosis and poor outcomes.[14,15]

Limitations of the current steatotic liver disease definitions

There are a number of limitations to the current definitions of SLD. The definitions rely on rigid thresholds for alcohol consumption and CMRFs, maintaining a siloed approach to MASLD, MetALD, and ALD, that often delay appropriate intervention for alcohol misuse and aggressive holistic management of CMRFs.[6] Misclassification is common, particularly due to underreporting of alcohol use, underscoring the need for objective alcohol biomarkers such as phosphatidylethanol (PEth).[16] Over 95% of individuals with ALD also exhibit CMRF, and alcohol itself can induce metabolic changes, such as hypertension and dyslipidaemia.[17] Concerningly, moderate alcohol consumption in the context of obesity or T2D synergistically increases the risk of advanced fibrosis and mortality, emphasising that there is no safe level of alcohol in SLD.[18] Individuals with MASLD and MetALD appear to be at higher risk for cardiovascular-related mortality, whereas those with ALD have a higher risk of liver-related mortality, cancer-related mortality, and overall mortality.[19]

Assessing alcohol intake

It is important to accurately assess not only the amount of alcohol intake, particularly hazardous drinking and the pattern of drinking (daily or binge), but also the past history of drinking and the possibility of an alcohol use disorder (AUD), which requires appropriate behavioural intervention.[16] Alcohol intake of < 20 g/day (females) and < 30 g/day (males) may accelerate MASLD, whilst a moderate intake of 20–50 g/day (females) and 30–60 g/day (males) may act additively or synergistically with metabolic comorbidities to increase the risk of progressive liver disease.[16,17]

Higher levels of alcohol consumption are associated with an increased risk of adverse outcomes, including advanced fibrosis, cirrhosis, cancer, cardiovascular disease, chronic kidney disease, and overall mortality.[20] Alcohol is the leading risk factor for liver fibrosis progression in SLD, particularly in MetALD and ALD.[21,22] Importantly, abstinence can potentially reverse disease progression across the spectrum of SLD.[23]

Several tools have been developed to screen for alcohol use and AUD, including a combination of patient-reported questionnaires, direct biochemical tests that detect ethanol or its non-oxidative metabolites, and indirect biomarkers that signal alcohol-related tissue injury.[16] Whole blood phosphatidylethanol (PEth) is a sensitive and specific biomarker capable of detecting a single drinking episode for up to 12 days and is a semi-quantitative measure of chronic heavy use for up to 6 weeks. Recommended PEth thresholds are 20–200 ng/ml for MetALD and ≥ 200 ng/ml for ALD.[16]

Current alcohol intake can initially be assessed using the single-item questionnaire, the National Institute on Alcohol Abuse and Alcoholism (NIAAA) Single Alcohol-Screening Question, *"How many times in the past year have you had ≥ 5 drinks in a day (men) or ≥ 4 drinks in a day (women)?"*. The Alcohol Use Disorders Identification Test (AUDIT) is a 10-item questionnaire that has been validated in multi-ethnic cohorts to screen for AUD; however, it is time-consuming and difficult to implement in clinic settings. AUDIT scores from 8 to 14 suggest hazardous or harmful alcohol consumption and a score of 15 or more, indicates the likelihood of alcohol dependence (moderate-severe alcohol use disorder).[16] The AUDIT-C, which includes the first three items of the 10-question AUDIT, is particularly useful for screening for hazardous drinking (≥ 3 points for women had 73% sensitivity and 91% specificity; ≥ 4 points for men had 86% sensitivity and 89% specificity) in general medical and liver clinics.[24]

However, reliance on patient-reported alcohol intake has proven problematic. Phosphatidylethanol (PEth) testing has shown that 20–30% of MASLD-patients previously labelled as "non-alcoholic" were consuming significant amounts of alcohol.[25,26] A recent study of 2 924 SLD participants confirmed that when using the PEth threshold of ≥ 20 ng/ml), 39.0% of MASLD patients could be re-classified as having MetALD or ALD.[26]

Importance of diagnosing and risk-stratifying individuals with steatotic liver disease

Screening for liver steatosis in the general population is not currently recommended, as isolated steatosis does not confer a risk of liver-related events, and there is no evidence to support general population-based fibrosis screening.[16,27]

However, it is important to risk-stratify individuals with at-risk SLD, stage fibrosis, and determine the need for tertiary-level care under a hepatologist or gastroenterologist. This involves the accurate assessment of CMRFs and alcohol consumption and developing diagnostic strategies and referral pathways for at-risk MASLD.[6,16,28,29] MASLD patients with T2D, pre-diabetes, and obesity with ≥ 1 CMRF are at high risk of developing cirrhosis (Figure 2).[30]

Non-invasive tests (NIT) are recommended to screen for fibrosis in SLD. Fibrosis-4 (FIB-4) is the preferred NIT for initial risk stratification, followed by vibration-controlled transient elastography (VCTE) or the enhanced liver fibrosis (ELF).[16,31,32] FIB-4 performance is suboptimal in patients aged < 35 or ≥ 65 years, with the specificity of FIB-4 for advanced fibrosis significantly decreasing with age, leading to higher rates of false positives in older patients. A FIB-4 threshold of > 2 for patients aged ≥ 65 years is recommended to enhance diagnostic accuracy. The two-step approach (FIB-4 followed by VCTE) for outpatients with T2D in a diabetes clinic resulted in only 12% of T2D outpatients needing hepatology referrals.[33]

Although hepatocellular carcinoma can occur in patients with MASLD and stage F3 fibrosis, routine screening and surveillance are not recommended in the absence of cirrhosis, but should be determined on a case-by-case basis depending on additional risk factors.[29]

Overview of steatotic liver disease management

A multidisciplinary, holistic approach is crucial for optimising SLD management. This includes patient education, behavioural strategies, and social support to enhance motivation and adherence to therapeutic programmes. Although the therapeutic landscape of SLD is rapidly evolving, particularly in the context

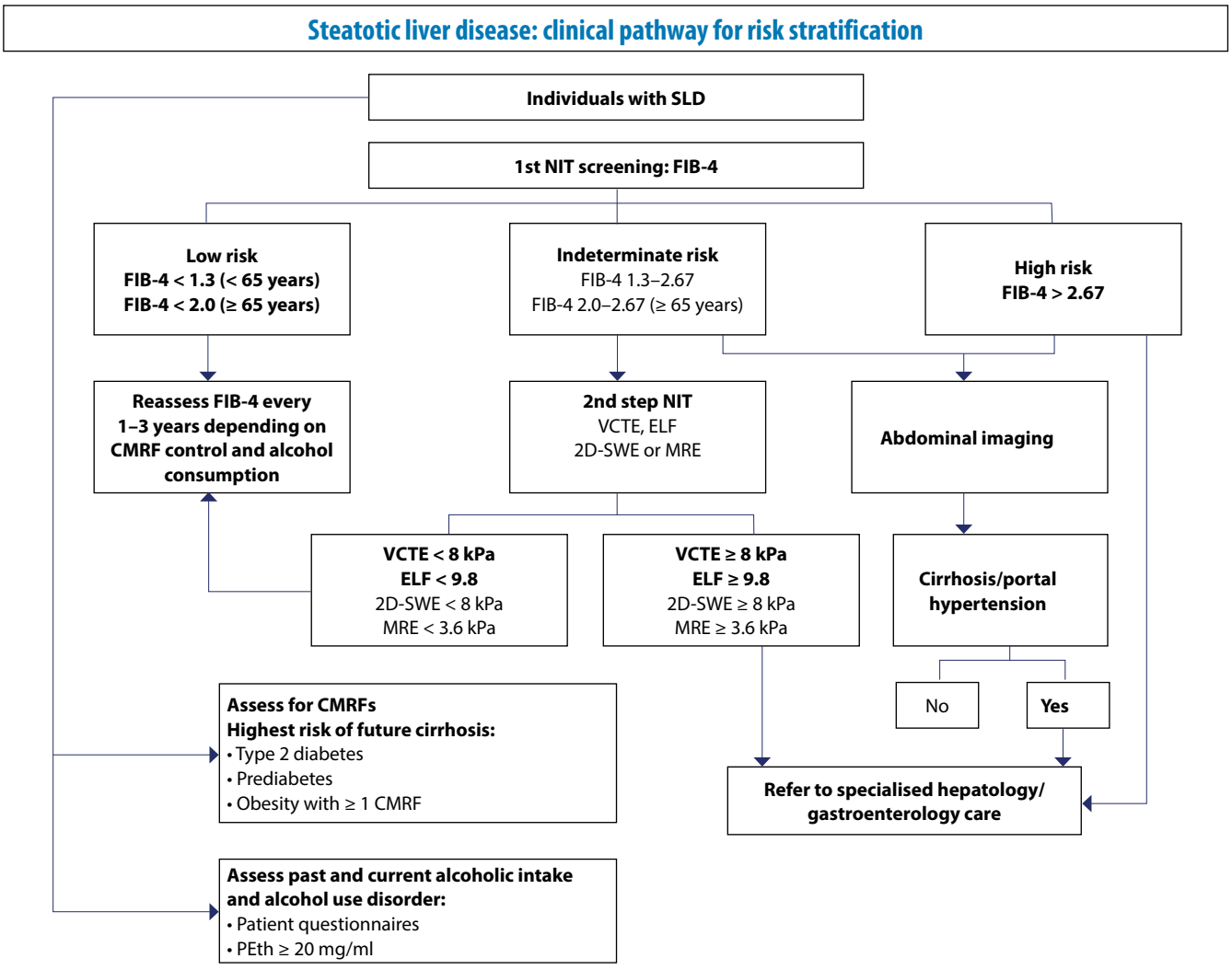


Figure 2: Clinical pathway for steatotic liver disease risk stratification
2D-SWE – two-dimensional shear wave elastography, CMRF – cardiometabolic risk factor, ELF – enhanced liver fibrosis, FIB-4 – Fibrosis-4, MRE – magnetic resonance elastography, NIT – non-invasive test, PEth – phosphatidylethanol, SLD – steatotic liver disease, VCTE – vibration-controlled transient elastography

of metabolic dysfunction-associated steatohepatitis (MASH), lifestyle modifications remain the cornerstone of management.[29]

Weight loss

MASLD and overweight/obesity:

- ≥ 5% total body weight loss to reduce hepatic steatosis.
- 7–10% total body weight loss to improve hepatic inflammation.
- ≥ 10% total body weight loss to improve liver fibrosis.
- Consider bariatric surgery for people with class 2 and 3 obesity.

MASLD and normal weight:

Reduction of 3–5% of total body weight to reduce hepatic steatosis.

Diet:

- Follow a culturally appropriate Mediterranean diet with increased consumption of fruits, vegetables, nuts, legumes, olive oil, and unprocessed fish and poultry.
- Limit intake of ultra-processed foods and saturated fats.
- Avoid sugar-sweetened beverages.
- Coffee: 2–3 cups of black coffee per day.
- Stop all alcohol and smoking.

Exercise:

- Increase daily physical activity: 150–300 minutes of moderate exercise or 75–150 minutes of vigorous exercise.
- Limit sedentary lifestyle.

Therapeutic landscape of metabolic dysfunction-associated steatotic liver disease

Effective management primarily involves assessing and actively managing the driving CMRFs of SLD, and establishing the amount and pattern of alcohol consumption and the potential of AUD.[16,28-30,34]

Treatment options and considerations

Type 2 Diabetes with or without MASH

- Glucagon-like peptide-1 receptor agonists (GP-1RA): preferred treatment for T2D and/or obesity in individuals with MASH.
- GLP/glucose-dependent insulinotropic polypeptide (GIP) dual agonists: treatment for T2D and/or obesity for individuals with or without MASH.
- Sodium-glucose co-transporter-2 (SGLT-2) inhibitors: consider as treatment for T2D for individuals with or without MASH.
- Dipeptidyl peptidase-4 (DPP-4) inhibitors: consider as treatment for T2D for individuals with or without MASH.

Dyslipidaemia

- Statins are safe in chronic liver disease and reduce cardiovascular mortality.

Metabolic dysfunction-associated steatohepatitis with F2-F3 fibrosis

There are now two Food and Drug Administration (FDA)-approved therapeutic options for the treatment of MASH with F2-F3 fibrosis in the absence of cirrhosis based on NIT criteria: resmetirom, a thyroid hormone receptor-B agonist (MAESTRO-NASH trial) and semaglutide, a GLP-1 receptor agonist (ESSENCE trial).[35,36] Both meet the primary endpoints of MASH resolution without worsening fibrosis and a ≥ 1 stage reduction in liver fibrosis without worsening MASH. Semaglutide has the added benefit of weight loss and reduces the risk of worsening kidney disease, kidney failure, and death due to cardiovascular disease in adults with T2D and chronic kidney disease.

Conclusion

SLD comprises a dynamic spectrum of MASLD, MetALD, and ALD, and it is a global and increasing health burden. A multidisciplinary and multisectoral approach, including primary care physicians, hepatologists, metabolic specialists, behavioural health experts, addiction specialists, dietitians, and exercise trainers, is essential to address the diverse aetiologies of SLD and improve long term patient outcomes.

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