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# From NAFLD to MASLD: A narrative review of the nomenclature paradigm shift and its practical implications for internal medicine

**Muhammad Salman Asghar*, Muhammad Hamza Mubarak**
Department of Internal Medicine, Faculty of Medicine, Universitas Islam Indonesia, Yogyakarta, Indonesia
**Corresponding Author:** Muhammad Salman Asghar
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**Abstract**
For nearly three decades, the hepatic manifestation of metabolic syndrome was described by the exclusionary and stigmatizing term nonalcoholic fatty liver disease. In 2023, an international consensus process replaced it with metabolic dysfunction-associated steatotic liver disease (MASLD), a diagnosis anchored directly in cardiometabolic risk. This narrative review synthesizes the rationale for that change and translates it into a practical framework for internists, covering diagnostic criteria, genetic and epidemiologic context, special populations, non-invasive fibrosis risk stratification, hepatocellular carcinoma surveillance, and the expanding pharmacologic landscape. MASLD is diagnosed when hepatic steatosis coexists with at least one of five cardiometabolic risk factors, in the absence of excessive alcohol intake. The fibrosis-4 index remains the recommended first-line tool for risk stratification in non-specialist settings. Since 2024, resmetirom and semaglutide have become the first two pharmacologic agents approved for steatohepatitis with fibrosis. Internists occupy a pivotal position across this care pathway, from opportunistic case finding to coordination of multidisciplinary management, making familiarity with the updated framework essential to timely and effective care.

**Keywords:** Metabolic dysfunction-associated steatotic liver disease, nonalcoholic fatty liver disease, internal medicine, fibrosis-4 index, cardiometabolic risk factors, nomenclature

## Introduction

Fatty liver disease associated with metabolic dysfunction is now the most common chronic liver condition encountered in internal medicine and primary care. For nearly thirty years, the field relied on the term nonalcoholic fatty liver disease, a label defined by what it excluded rather than by the pathophysiology it represented. This exclusionary structure required clinicians to rule out alcohol use and other competing causes before a diagnosis could be assigned, and both patients and clinicians increasingly viewed the words nonalcoholic and fatty as stigmatizing (Rinella *et al*., 2023a) [40].

An interim proposal in 2020, metabolic-associated fatty liver disease, attempted to reframe the condition around metabolic dysfunction using inclusive, positive diagnostic criteria rather than exclusion, but it did not achieve broad international consensus (Eslam *et al*., 2020) [12]. In 2023, a coordinated, multisociety Delphi process involving more than two hundred panelists from hepatology, endocrinology, and patient advocacy organizations converged on a new nomenclature: steatotic liver disease as an overarching term, with metabolic dysfunction-associated steatotic liver disease (MASLD) replacing NAFLD as its principal subtype (Rinella *et al*., 2023a) [40]. This review outlines that transition and translates it into a practical framework for internists, who are typically the first physicians to encounter these patients, often through incidental findings on abdominal imaging or routine liver enzyme testing.

## Methods

This is a narrative review rather than a systematic review or original research report. Sources were identified through targeted searches of consensus statements, clinical practice guidelines, and peer-reviewed articles published primarily between 2019 and 2026 [5], with priority given to guidance issued or endorsed by the American Association for the Study of Liver Diseases, the European Association for the Study of the Liver, the European Association for the Study of Diabetes, and the European Association for the Study of Obesity (Rinella *et al*., 2023b; Tacke *et al*., 2024) [1, 13]. Because the purpose of this review is educational and clinically oriented rather than to generate new primary data, no original patient data, biological samples, or institutional review board approval were required.

## The Nomenclature Transition: From NAFLD Through MAFLD to MASLD

The 2023 Delphi consensus was convened by three pan-national liver associations and drew participation from seven societies and patient advocacy groups, achieving response rates above 75 percent across four voting rounds (Rinella *et al*., 2023a) [40]. Consensus was predefined as a supermajority of at least 67 percent. Among the external expert committee, 75 percent favored metabolic dysfunction-associated steatotic liver disease as the replacement term for NAFLD, and 88 percent favored metabolic dysfunction-associated steatohepatitis (MASH) as the replacement for NASH (Rinella *et al*., 2023a) [40].

Rather than a single renamed entity, the new framework establishes steatotic liver disease as an umbrella term encompassing five subgroups, distinguished by the presence of cardiometabolic risk factors and alcohol intake: MASLD, MetALD (metabolic dysfunction plus moderate alcohol intake), alcohol-related liver disease, specific-etiology steatotic liver disease, and cryptogenic steatotic liver disease.

The alcohol thresholds separating these categories were retained from prior definitions at more than 20 grams per day for women and more than 30 grams per day for men (Rinella *et al*., 2023a; Tacke *et al*., 2024) [40, 46]. Fig 1 illustrates this classification schema. The pediatric hepatology and gastroenterology community subsequently issued its own multisociety statement endorsing the new nomenclature while highlighting features distinct to children, discussed further in Section 7 (Journal of Pediatric Gastroenterology and Nutrition, 2024) [27].

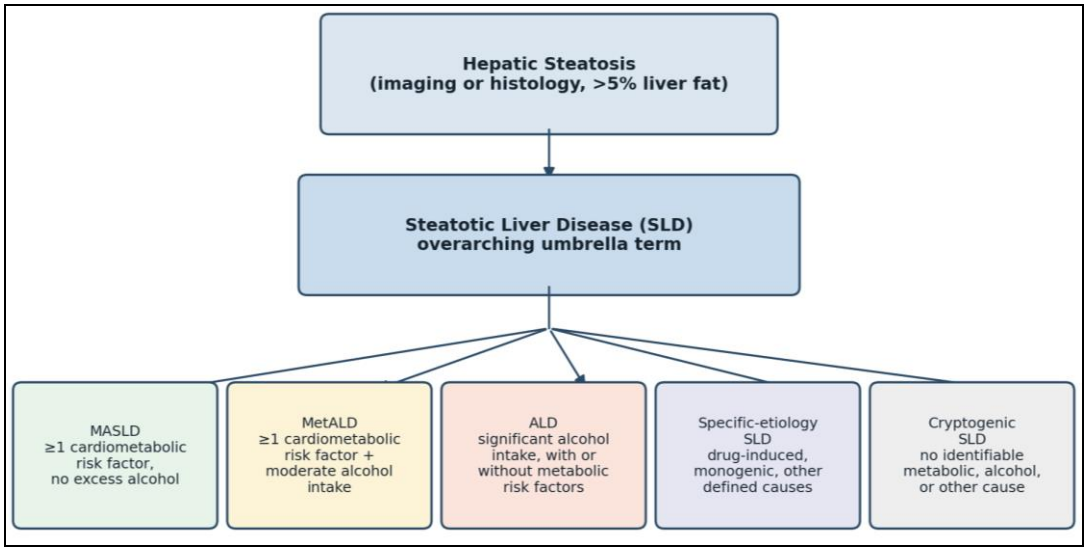



**Fig 1:** Classification schema of steatotic liver disease and its subtypes under the 2023 [1] multisociety nomenclature.

Retrospective concordance studies applying both definitions to the same cohorts have found that the overwhelming majority of patients previously labeled NAFLD, on the order of 99 percent, also meet MASLD criteria, supporting the reusability of existing longitudinal data and biobank cohorts under the new terminology (Rinella *et al*., 2023a) [40].

**Diagnostic Criteria and Cardiometabolic Risk Factors**

A diagnosis of MASLD requires two elements: radiologic or histologic evidence of hepatic steatosis, and the presence of at least one of five defined cardiometabolic risk factors, in the absence of alcohol intake above the defined thresholds or another identifiable competing cause of steatosis (Rinella *et al*., 2023a, 2023b; Tacke *et al*., 2024) [40, 46]. These five criteria, summarized in Table 1, were deliberately designed to be simple enough for use outside specialist hepatology clinics, since most patients with steatotic liver disease are identified and initially managed in internal medicine and primary care settings (Postgraduate Medicine, 2024; Rinella *et al*., 2023b) [38, 40].

**Table 1:** Cardiometabolic Risk Factors Required for the Diagnosis of MASLD

| No. | Cardiometabolic Risk Factor | Diagnostic Threshold |
|---|---|---|
| 1 | Increased adiposity | Body mass index ≥ 25 kg/m² (≥ 23 kg/m² in Asian populations), or waist circumference > 94 cm in men / > 80 cm in women |
| 2 | Impaired glucose metabolism | Fasting plasma glucose ≥ 100 mg/dL (5.6 mmol/L), 2-hour post-load glucose ≥ 140 mg/dL, HbA1c ≥ 5.7%, established type 2 diabetes, or treatment for type 2 diabetes |
| 3 | Hypertension | Blood pressure ≥ 130/85 mmHg, or specific antihypertensive drug treatment |
| 4 | Hypertriglyceridemia | Plasma triglycerides ≥ 150 mg/dL (1.70 mmol/L), or lipid-lowering treatment |
| 5 | Low HDL cholesterol | HDL-C ≤ 40 mg/dL in men or ≤ 50 mg/dL in women, or lipid-lowering treatment |

Note. Diagnosis of MASLD requires hepatic steatosis on imaging or histology plus at least one of the five criteria above, in the absence of alcohol intake exceeding 20 g/day (women) or 30 g/day (men) and without another competing cause of steatosis. Adapted from Rinella *et al*. (2023a) and Tacke *et al*. (2024) [40, 46].

Because overweight or obesity is present in the large majority of patients who otherwise meet MASLD criteria, some authors have questioned whether adiposity should remain a required criterion versus a near-universal comorbidity; the other four cardiometabolic markers, particularly dysglycemia and hypertension, appear more strongly and independently associated with all-cause and cardiovascular mortality within MASLD cohorts (Atherosclerosis, 2025) [20]. A large real-world analysis further found that many patients labeled lean MASLD cluster just below conventional obesity thresholds rather than representing a truly non-adipose phenotype, reinforcing that body mass index alone is an imperfect risk marker (Epic Research, 2026) [11].

**Genetic Susceptibility and Precision Risk Assessment**

Beyond cardiometabolic risk factors, common genetic variants materially influence both susceptibility to MASLD and the likelihood of progression to steatohepatitis, fibrosis, and hepatocellular carcinoma. The PNPLA3 rs738409 (I148M) variant is the most extensively studied genetic risk factor, present in roughly 30 to 50 percent of individuals, and is associated with a substantially higher risk of liver-related mortality among homozygous carriers (Impact of

PNPLA3 I148M on Clinical Outcomes in Patients With MASLD, 2025) [33]. The TM6SF2 E167K variant independently increases the risk of hepatic fibrosis and hepatocellular carcinoma, and appears to act through impaired transfer of polyunsaturated fatty acids that promotes hepatic lipid accumulation (Deep Metabolic Phenotyping of Humans with Protein-Altering Variants in TM6SF2, 2024) [9].

Combining genetic information with routine fibrosis scores may improve risk prediction beyond either approach alone. In a multicenter cohort of Japanese patients with biopsy-proven MASLD followed for a median of 8.1 years, PNPLA3, TM6SF2, and GCKR genotypes were each independently associated with liver-related events, and combining FIB-4 with genetic polymorphisms improved discrimination for future events compared with FIB-4 alone (Seko *et al*., 2025) [44]. Among patients with coexisting type 2 diabetes, cumulative PNPLA3 and TM6SF2 risk alleles were independently associated with cirrhosis, its complications, and extrahepatic cancer over more than a decade of follow-up (Lavrado *et al*., 2024) [29]. While polygenic risk scores are not yet part of routine internal medicine practice, current guidance suggests they may eventually refine risk stratification for patients whose FIB-4 or elastography results are indeterminate (Rinella *et al*., 2023b) [40].

**Epidemiology and Global Burden**

Estimates of the global burden of MASLD vary depending on methodology and diagnostic definition, but all point to a substantial and rising burden. The 2023 Global Burden of Disease analysis estimated that approximately 1.3 billion people, or 16.1 percent of the global population, were living with MASLD in 2023, representing a marked increase since 1990 [17] (Institute for Health Metrics and Evaluation, 2026) [19]. Other systematic analyses using slightly different case definitions have placed overall adult prevalence closer to 30 to 38 percent, with regional prevalence ranging from roughly 20 to 80 percent depending on local rates of obesity and type 2 diabetes (Internal and Emergency Medicine, 2025) [24]. MASLD has become the leading cause of chronic liver disease and, in some regions, the leading indication for liver transplantation, and cardiovascular disease remains the most common cause of death among affected patients (Frontiers in Medicine, 2025) [24].

Regional variation is particularly relevant to practice in Indonesia and Southeast Asia. Pooled estimates place overall NAFLD or MASLD prevalence in Indonesia at approximately 51 percent, among the highest reported worldwide, compared with roughly 38 to 40 percent in neighboring Malaysia and Singapore (Li *et al*., 2019). A recent occupational cohort study among university staff in Indonesia similarly reported a high burden of fatty liver disease linked to obesity, dyslipidemia, and lifestyle factors, underscoring the relevance of active case finding in Indonesian internal medicine practice (Frontiers in Public Health, 2026) [19].

**Lean and Pediatric MASLD: Special Populations**

Although MASLD is closely associated with obesity, a clinically important minority of patients develop the disease without meeting conventional obesity thresholds, a phenomenon termed lean MASLD. Pooled estimates suggest a prevalence of approximately 19 percent for lean and 41 percent for broadly defined nonobese MASLD among affected populations, with substantial variation by country (Diagnosis and Management of Lean Metabolic Dysfunction-Associated Steatotic Liver Disease, 2024) [13]. Compared with higher-body-mass-index counterparts, lean patients with MASLD generally show a milder metabolic and histologic profile but can still progress through the full spectrum of steatohepatitis and fibrosis, and diagnosis is often delayed because clinicians do not expect fatty liver disease in the absence of visible obesity (Nutrition & Metabolism, 2025) [34].

Pediatric MASLD represents a second special population of direct relevance to internists who transition adolescent patients into adult care. The condition now affects an estimated 13 to 14 percent of children globally and up to one-third of children with obesity, and unlike the typically decades-long course seen in adults, steatohepatitis can develop in children within only a few years of the onset of steatosis (Springer Medicine, 2026) [45]. A joint multisociety statement from pediatric hepatology and gastroenterology organizations endorsed the new nomenclature while cautioning that the word metabolic could cause confusion with inborn errors of metabolism in pediatric settings, and recommended periodic reevaluation of children initially classified as cryptogenic steatotic liver disease (Journal of Pediatric Gastroenterology and Nutrition, 2024) [27]. The bidirectional relationship between pediatric MASLD and type 2 diabetes further underscores the need for coordinated surveillance as these patients enter adult internal medicine care (Converging Pathways between Metabolic Dysfunction-Associated Steatotic Liver Disease and Diabetes in Children, 2024; Obesity Medicine Association, 2026) [13, 35].

**Diagnostic Approach in Internal Medicine Practice**

A pragmatic stepwise approach for the internist begins with quantifying alcohol intake, since this determines which steatotic liver disease subcategory applies. The clinician then screens for the five cardiometabolic risk factors described in Table 1, and finally excludes viral hepatitis and other secondary causes of steatosis such as steatogenic medications, including corticosteroids, amiodarone, and methotrexate (Rinella *et al*., 2023b) [40]. Fig 2 presents this pathway as it might be applied at the point of care, whether steatosis is discovered incidentally on abdominal imaging performed for another indication or suspected because of unexplained elevation in liver enzymes.

Guidelines are consistent in recommending against population-wide screening for steatotic liver disease in individuals without cardiometabolic risk factors; the recommended strategy is targeted case finding among adults who already carry one or more of the five risk factors, particularly those with type 2 diabetes or obesity plus an additional metabolic abnormality (Tacke *et al*., 2024) [46].

**Extrahepatic Manifestations**

MASLD is increasingly understood as a systemic, multi-organ condition rather than a liver-confined diagnosis, a framing with direct relevance to internists who manage its comorbidities. Cardiovascular disease and cancer are the leading causes of death among patients with MASLD, with liver-related mortality ranking third overall, and fibrosis severity remains the strongest predictor of both liver-specific and all-cause mortality (Extrahepatic Manifestations of Metabolic Dysfunction-Associated Steatotic Liver Disease, 2025) [44]. Chronic kidney disease

occurs at increased frequency independent of shared classical risk factors, an association attributed in part to lipotoxicity affecting the kidney through pathways parallel to those operating in the liver (MASLD: Lipotoxicity and Imaging Parallels from Liver Steatosis to Kidney Injury, 2025) [44].

Obstructive sleep apnea shares an especially close, likely bidirectional relationship with MASLD; in one cohort of patients with type 2 diabetes, essentially all patients with severe MASLD also had obstructive sleep apnea (Obstructive Sleep Apnea Is Related to Metabolic Dysfunction Associated Steatotic Liver Disease in Type 2 Diabetes Mellitus, 2025) [44]. Additional extrahepatic associations relevant to internal medicine practice include increased risk of colorectal, pancreatic, and breast cancers, endocrine disorders such as hypothyroidism and polycystic ovary syndrome, and musculoskeletal conditions including sarcopenia and osteoporosis (Extrahepatic Comorbidities Associated with Metabolic Dysfunction-Associated Steatotic Liver Disease, 2025; Mitra *et al*., 2020) [32, 44]. This broad comorbidity profile supports the argument that MASLD management belongs squarely within general internal medicine rather than hepatology alone.

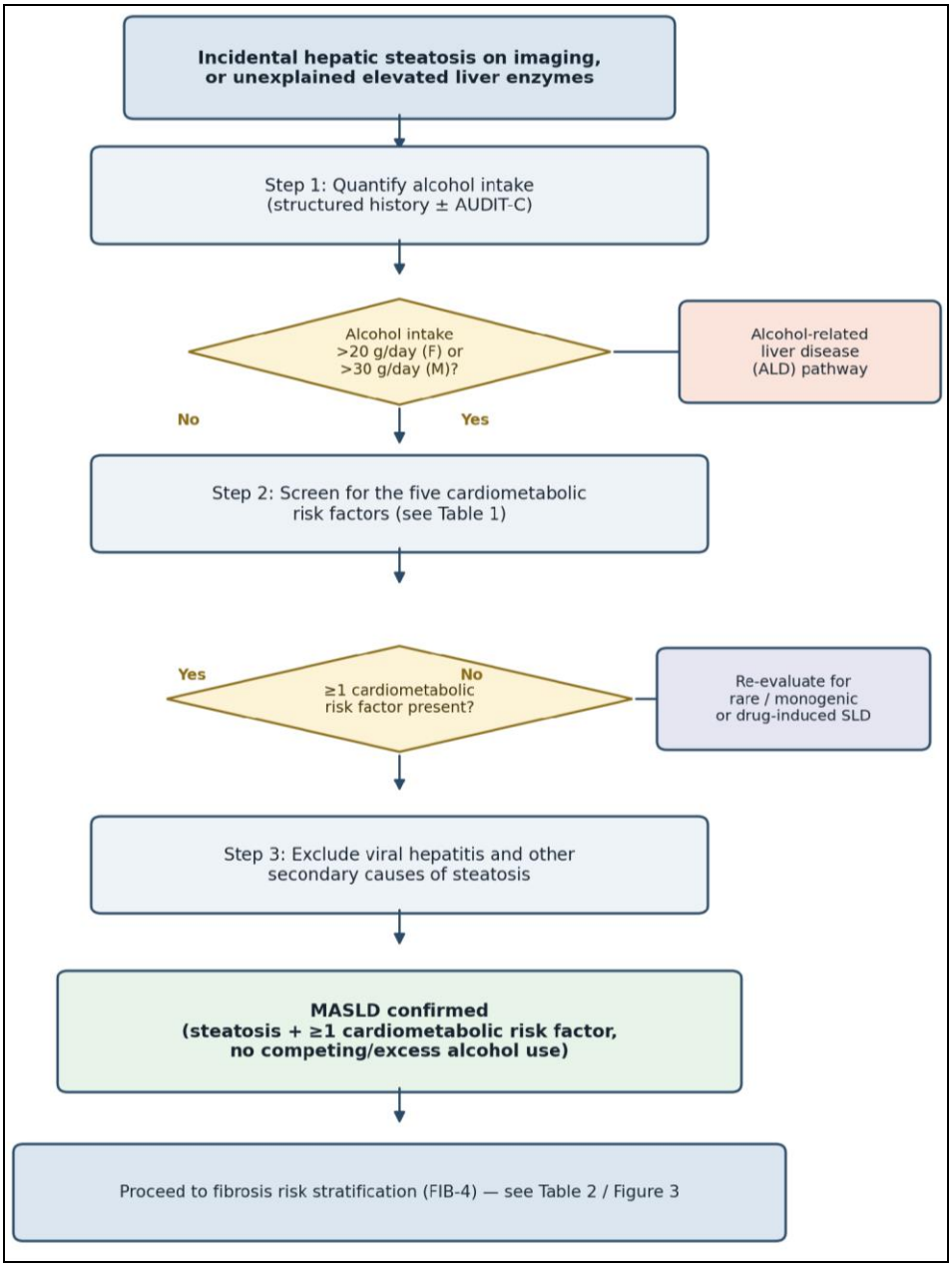



**Fig 2:** Stepwise diagnostic approach to suspected MASLD in internal medicine practice.

**Non-Invasive Fibrosis Risk Stratification**

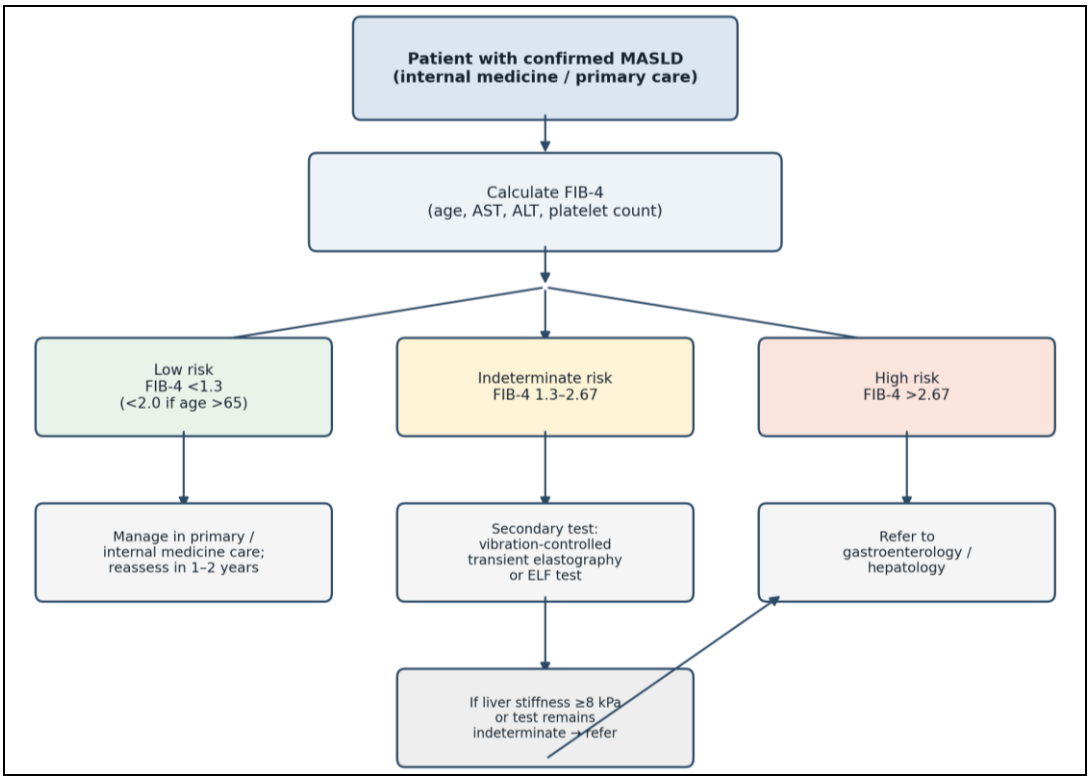
Once MASLD is identified, the clinical priority shifts from confirming the diagnosis to estimating the risk of significant fibrosis, since fibrosis stage is the strongest predictor of liver-related morbidity and mortality (Journal of General Internal Medicine, 2024; Rinella *et al*., 2023b) [26, 40]. The fibrosis-4 (FIB-4) index, calculated from age, aspartate aminotransferase, alanine aminotransferase, and platelet count, is recommended as the first-line non-invasive test because it is inexpensive, requires no specialized equipment, and is well validated across diverse populations (Postgraduate Medicine, 2024; Rinella *et al*., 2023b) [38, 40].

**Table 2:** Stepwise Non-Invasive Fibrosis Risk Stratification Using FIB-4 in Internal Medicine Practice

| Risk Category | FIB-4 Score | Recommended Next Step |
|---|---|---|
| Low risk | < 1.3 (< 2.0 if age > 65 years) | Continue management in internal medicine / primary care; reassess FIB-4 in 1 to 2 years |
| Indeterminate risk | 1.3 to 2.67 (2.0 to 2.67 if age > 65 years) | Second-line testing with vibration-controlled transient elastography or the enhanced liver fibrosis (ELF) test |
| High risk | > 2.67 | Refer to gastroenterology / hepatology for further evaluation and consideration of advanced fibrosis or cirrhosis |

Note. FIB-4 = (age in years x AST [U/L]) / (platelet count [10^9/L] x square root of ALT [U/L]). Cut-off values adapted from Rinella *et al*. (2023b) [40] and subsequent validation cohorts (Postgraduate Medicine, 2024) [38].

A FIB-4 score below 1.3, or below 2.0 in patients older than 65 years, is generally sufficient to exclude advanced fibrosis and allows continued management in internal medicine with periodic reassessment (Rinella *et al*., 2023b) [40]. Indeterminate scores warrant second-line testing, most commonly vibration-controlled transient elastography or the enhanced liver fibrosis blood test, while scores above 2.67 warrant referral to gastroenterology or hepatology (European Journal of Internal Medicine, 2024; Rinella *et al*., 2023b) [14, 40]. Real-world validation studies have found FIB-4 to have imperfect sensitivity, particularly in younger patients and in detecting earlier stages of significant fibrosis, so clinical judgment and reassessment intervals should not be abandoned even when the score is reassuring (Postgraduate Medicine, 2024 [38]; Unrecognized Fibrosis Risk in MASLD, 2026) [47]. Fig 3 depicts this stepwise pathway.




In all risk categories: treat cardiometabolic comorbidities and reinforce lifestyle modification concurrently

**Fig 3:** FIB-4-based stepwise fibrosis risk stratification pathway for use in internal medicine and primary care.

Large primary care cohort studies illustrate a persistent implementation gap: in one electronic health record based analysis of over ninety thousand patients at increased risk for MASLD, only a small minority of patients with indeterminate or high-risk FIB-4 scores went on to receive appropriate secondary testing or specialist referral, and this gap was not explained by normal aminotransferase levels, which were common even among higher-risk patients (Journal of General Internal Medicine, 2024) [26]. Similar care gaps have been reported in more recent retrospective analyses exploring the potential role of electronic decision-support tools and natural-language processing of radiology reports to improve case capture (Unrecognized Fibrosis Risk in MASLD, 2026) [47].

**Advanced Imaging and Emerging Biomarkers**

When FIB-4 results are indeterminate, second-line testing typically relies on vibration-controlled transient elastography, which quantifies liver stiffness and, through the controlled attenuation parameter, hepatic steatosis in a single outpatient examination (Full Article: Non-Invasive Assessment of Liver Fibrosis in MASLD, 2025) [44]. Magnetic resonance imaging proton density fat fraction offers superior accuracy for quantifying hepatic steatosis compared with ultrasound or computed tomography and correlates closely with histologic steatosis grade, although cost and limited availability restrict its routine use outside specialist or research settings (European Journal of Internal Medicine, 2024) [14].

Composite scores that combine imaging and blood-based markers, such as the MRI-AST score and the combination of magnetic resonance elastography with FIB-4, have shown improved discrimination for fibrotic steatohepatitis compared with simple non-invasive tests alone, particularly in populations with a lower pretest probability of advanced disease (Novel Noninvasive Tests for Liver Fibrosis, 2025) [44]. For internists without direct access to elastography, awareness of these second- and third-line options supports more informed conversations when referring patients with indeterminate FIB-4 results to gastroenterology or hepatology.

**Hepatocellular Carcinoma Surveillance**

Surveillance for hepatocellular carcinoma with twice-yearly ultrasound, with or without alpha-fetoprotein, is recommended for all patients with cirrhosis, including cirrhosis attributable to MASLD, because surveillance in this population improves overall survival (European Association for the Study of the Liver, 2024) [13]. Current guidance does not recommend surveillance in non-cirrhotic MASLD given the comparatively low annual incidence of hepatocellular carcinoma in this group (American Gastroenterological Association, 2022) [3]. This blanket exclusion is increasingly debated, since nearly half of hepatocellular carcinoma cases arising in the context of MASLD occur without cirrhosis and often present with larger, more advanced tumors than cirrhosis-associated cases, prompting proposals for risk-stratified surveillance algorithms in selected non-cirrhotic patients with additional risk factors such as type 2 diabetes or advanced fibrosis on non-invasive testing (Risk-Stratified Hepatocellular Carcinoma Surveillance in Non-Cirrhotic Patients with MASLD, 2025) [20].

For internists, the practical implication is that cirrhosis prevention through timely fibrosis risk stratification and comorbidity management remains the single most effective strategy for reducing hepatocellular carcinoma mortality, since current surveillance tools still perform best once cirrhosis has already developed (Medscape, 2026) [31].

**Therapeutic Landscape: From Lifestyle Modification to Pharmacotherapy**

Lifestyle modification remains the foundation of MASLD management at every fibrosis stage. Current guidance recommends gradual weight loss through caloric restriction and increased physical activity, dietary patterns consistent with a Mediterranean-style diet, moderation of alcohol intake even below diagnostic thresholds, and optimization of coexisting cardiometabolic conditions such as type 2 diabetes, hypertension, and dyslipidemia (Tacke *et al*., 2024) [46]. A randomized controlled trial comparing a twelve-week Mediterranean diet with a low-fat diet found both equally effective at improving hepatic steatosis and fibrosis biomarkers regardless of PNPLA3 genotype, suggesting that dietary pattern can be individualized to patient preference without loss of efficacy (Hepatology Communications, 2025) [21]. Observational cohort data further associate regular coffee consumption with lower rates of obesity, hypertension, dyslipidemia, and diabetes among patients with steatotic liver disease, alongside more favorable elastography parameters (Frontiers in Nutrition, 2026) [18].

For patients with obesity and MASH who do not respond adequately to lifestyle intervention, bariatric surgery has shown superiority to intensive lifestyle and optimized medical therapy for achieving histologic resolution of steatohepatitis without worsening fibrosis in a large multicenter randomized trial (BRAVES Trial Collaborative Group, 2023) [7]. Long-term follow-up from an independent prospective cohort found that resolution of steatohepatitis without worsening of fibrosis after bariatric surgery was associated with survival approaching that of patients without baseline steatohepatitis, supporting its use as a clinically meaningful treatment target rather than a surrogate endpoint alone (Lassailly *et al*., 2024) [28].

The pharmacologic landscape changed substantially beginning in 2024. Resmetirom, an oral, liver-directed, selective thyroid hormone receptor-beta agonist, received accelerated approval from the United States Food and Drug Administration in March 2024 for adults with noncirrhotic MASH and moderate to advanced fibrosis, becoming the first approved pharmacotherapy specifically for this indication (American Association for the Study of Liver Diseases, 2025; Joszt, 2024) [13, 44]. In the pivotal MAESTRO-NASH trial, resmetirom achieved resolution of steatohepatitis without worsening of fibrosis in roughly a quarter to a third of treated patients, compared with about ten percent on placebo, along with improvement in fibrosis without worsening of steatohepatitis in a similar proportion (American Association for the Study of Liver Diseases, 2025 [44]; Resmetirom in the Management of Metabolic Dysfunction-Associated Steatotic Liver Disease and Steatohepatitis, 2025) [44].

In August 2025, semaglutide, a glucagon-like peptide-1 receptor agonist already established in the treatment of type 2 diabetes and obesity, received FDA approval as a second pharmacologic option for noncirrhotic MASH with moderate to advanced fibrosis, based on results from the phase 3 ESSENCE trial (American Journal of Managed Care, 2025) [4]. At 72 weeks, resolution of steatohepatitis without worsening of fibrosis was achieved in approximately 63 percent of patients treated with semaglutide compared with 34 percent on placebo (American Journal of Managed Care, 2025) [4]. Several additional agents remain in late-stage development: the pan-PPAR agonist lanifibranor met both co-primary histologic endpoints in its phase 3 trial (PatSnap, 2026) [37], while the FGF21 analogue efruxifermin has shown fibrosis regression in earlier fibrosis stages, though it narrowly missed its primary endpoint in a phase 2b trial among patients with compensated MASH cirrhosis (Applied Clinical Trials Online, 2026 [5]; Safety and Efficacy of Efruxifermin in Metabolic Dysfunction-Associated Steatohepatitis, 2025) [6].

For the practicing internist, these developments do not replace the central role of cardiometabolic risk factor management, but they do mean that a confirmed diagnosis of MASH with significant fibrosis is no longer a diagnosis without a specific treatment option, which strengthens the argument for timely referral once risk stratification identifies these patients.

**Practical Implications for Internal Medicine**

Several practical themes emerge from this review for internists and general physicians. First, MASLD should be actively considered, rather than incidentally noted, in any patient with type 2 diabetes, obesity, or multiple

cardiometabolic risk factors, since these are precisely the patients in whom guidelines recommend case finding (Postgraduate Medicine, 2024; Tacke *et al*., 2024) [38]. Second, FIB-4 calculation can be incorporated into routine laboratory review with minimal additional cost, and structured protocols or electronic health record prompts may help close the substantial gap between identification of at-risk patients and completion of appropriate secondary testing that has been repeatedly documented in primary care cohorts (Journal of General Internal Medicine, 2024 [26]; Unrecognized Fibrosis Risk in MASLD, 2026) [47]. Third, extrahepatic cancer and cardiovascular risk should be addressed alongside liver-specific management, since cardiovascular disease, not liver failure, remains the leading cause of death in most patients with MASLD (Extrahepatic Manifestations of Metabolic Dysfunction-Associated Steatotic Liver Disease, 2025; Tacke *et al*., 2024) [44, 46].

Settings such as Indonesia and other countries with rising rates of obesity and type 2 diabetes, and with among the highest reported regional MASLD prevalence, are likely to see a growing burden, and local guideline adaptation, including validation of FIB-4 thresholds and expansion of access to elastography, remains an important area for regional clinical research (Frontiers in Public Health, 2026 [19]; Li *et al*., 2019).

## Limitations

As a narrative rather than a systematic review, this article does not apply formal quality appraisal or meta-analytic synthesis, and selection of sources, while intended to be representative of current major guidance, is not exhaustive. Pharmacologic and epidemiologic data cited from pivotal trials and cohort studies reflect interim or primary results available at the time of writing; longer-term outcome data, including from the ongoing extension phase of the ESSENCE trial and the pending phase 3 readout for lanifibranor, were not yet fully available and may refine the conclusions presented here.

## Conclusion

The transition from NAFLD to MASLD is more than a terminology change. It embeds cardiometabolic risk directly into the diagnostic framework, aligns nomenclature with underlying pathophysiology, and reduces stigmatizing language for patients. For internists, the practical consequences are concrete: targeted case finding among patients with cardiometabolic risk factors, awareness of genetic and population-specific variation in risk, attention to the systemic and extrahepatic burden of the disease, routine use of the fibrosis-4 index for risk stratification, and familiarity with an expanding pharmacologic armamentarium that now includes two approved agents for steatohepatitis with fibrosis. Closing the documented gap between risk identification and appropriate follow-up testing represents one of the most immediate opportunities to improve outcomes in this common condition, particularly in regions such as Indonesia where the burden of disease is especially high.

## Author Contributions

Both authors contributed to the conception of the review, literature synthesis, drafting, critical revision, and approval of the final manuscript. M.S.A. led manuscript drafting and Fig and table design; M.H.M. contributed to literature review, critical revision, and final approval.

## Conflict of Interest

The authors declare no conflict of interest.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## Data Availability

No new data were generated for this narrative review. All sources cited are publicly available in the referenced publications.

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