

Research Article

Metabolic dysfunction associated steatotic liver disease in patients with cardiometabolic risk

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Abstract: Metabolic dysfunction-associated steatotic liver disease has become one of the most common liver disorders encountered in general medicine, endocrinology, cardiology, obesity medicine, and primary care. The change from non-alcoholic fatty liver disease to metabolic dysfunction-associated steatotic liver disease reflects an important clinical shift: hepatic steatosis is now understood less as an isolated liver finding and more as part of a wider metabolic disorder. Patients with obesity, type 2 diabetes, hypertension, dyslipidaemia, cardiovascular disease, and chronic kidney disease carry a particularly high burden of steatosis, metabolic dysfunction-associated steatohepatitis, and clinically significant fibrosis. This review examines metabolic dysfunction-associated steatotic liver disease in patients with cardiometabolic risk, with specific attention to obesity, type 2 diabetes, cardiovascular disease, screening pathways, fibrosis assessment, and referral criteria. PubMed-indexed guidelines, systematic reviews, meta-analyses, cohort studies, and randomized trials were reviewed. Current evidence supports a risk-based approach rather than indiscriminate population screening. Patients with type 2 diabetes, obesity with additional metabolic risk factors, persistently abnormal liver enzymes, imaging evidence of steatosis, or clinical suspicion of advanced liver disease should undergo structured case-finding for fibrosis. Simple blood-based scores such as fibrosis-4 can be used as first-line tools, followed by elastography or enhanced liver fibrosis testing when results are indeterminate or high. Fibrosis stage remains the strongest predictor of liver-related outcomes and is also associated with broader mortality risk. Management should combine weight loss, treatment of diabetes and dyslipidaemia, cardiovascular risk reduction, alcohol assessment, and timely referral for specialist evaluation when advanced fibrosis or cirrhosis is suspected. Metabolic dysfunction-associated steatotic liver disease should no longer be treated as an incidental radiology phrase. In patients with cardiometabolic risk, it is a marker of systemic disease and a practical opportunity for early risk stratification.

Keywords: Metabolic dysfunction-associated steatotic liver disease, MASLD, MASH, obesity, type 2 diabetes, cardiovascular disease, fibrosis assessment, FIB-4, transient elastography, cardiometabolic risk

Introduction

A fatty liver report is often brief. The ultrasound may say “hepatic steatosis.” The liver enzymes may be normal. The patient may have no symptoms. For years, this combination encouraged therapeutic delay. The liver finding was noted, the diabetes or blood pressure was treated, and fibrosis risk was often left unmeasured.

That approach is no longer adequate.

Metabolic dysfunction-associated steatotic liver disease, or MASLD, describes hepatic steatosis occurring in the presence of at least one cardiometabolic risk factor and in the absence of other dominant causes of steatosis. The 2023 multisociety Delphi consensus replaced the older term non-alcoholic fatty liver disease with a nomenclature that better reflects the metabolic basis of the condition and avoids defining the disease mainly by what it is not (1). The updated terminology also introduced metabolic dysfunction-associated steatohepatitis,

Received: 12 January 2026

Accepted: 16 May 2026

Published: 27 May 2026

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or MASH, for the inflammatory and progressive form previously called non-alcoholic steatohepatitis.

The change is more than cosmetic. It matters because the patient in front of the clinician usually does not have a liver-only disorder. MASLD commonly sits beside visceral adiposity, insulin resistance, type 2 diabetes, hypertension, dyslipidaemia, obstructive sleep apnoea, chronic kidney disease, and established cardiovascular disease. It is therefore not surprising that cardiometabolic clinics, primary care practices, and diabetes services see many of these patients before hepatology services do.

The global burden is substantial. A systematic review estimated that approximately 30% of the global population has NAFLD, with prevalence increasing over recent decades (5). Since many of those studies used the older nomenclature, the exact figures do not translate perfectly to MASLD, but the clinical message remains clear. Steatotic liver disease associated with metabolic risk is common, underdiagnosed, and closely linked to the modern burden of obesity and type 2 diabetes.

The main clinical danger is not steatosis by itself. Many patients with simple steatosis will not develop liver failure. The problem is the subgroup with MASH, progressive fibrosis, advanced fibrosis, or cirrhosis. Fibrosis stage is the most important liver-related prognostic marker in this disease spectrum (11,12). A patient with steatosis and no significant fibrosis requires metabolic care and periodic reassessment. A patient with bridging fibrosis or cirrhosis requires a different pathway, including hepatology review, surveillance for complications, and more intensive risk management.

This review focuses on MASLD in patients with cardiometabolic risk. It discusses the relationship with obesity, type 2 diabetes, and cardiovascular disease, then turns to practical screening pathways, fibrosis assessment, referral criteria, and current management.

Literature approach and scope

This review was developed from PubMed-indexed literature on MASLD, NAFLD, MASH, NASH, obesity, type 2 diabetes, cardiovascular disease, non-invasive fibrosis testing, clinical practice guidance, and treatment trials. The selected literature included multisociety nomenclature statements, clinical practice guidelines, systematic reviews, meta-analyses, cohort studies, diagnostic studies, and randomized controlled trials.

Because the terminology changed recently, older studies using NAFLD and NASH were included when they were directly relevant to the condition now commonly described as MASLD and MASH. This is unavoidable in a clinically useful review because most epidemiological, diagnostic, prognostic, and therapeutic evidence was published before the nomenclature change. Where appropriate, the older terminology is used when referring to the original study population.

The review is narrative rather than statistical. It aims to provide a practical synthesis for medical journal readers who may work in general medicine, hepatology, endocrinology, cardiology, obesity medicine, and primary care.

A disease defined by metabolic context

MASLD is diagnosed when hepatic steatosis is present along with cardiometabolic risk. This framing makes clinical sense. Steatosis is not merely a storage issue in hepatocytes. It reflects insulin resistance, adipose tissue dysfunction, altered free fatty acid flux, inflammatory signalling, mitochondrial stress, lipotoxicity, and changes in gut-liver metabolism. The liver becomes a visible site of a systemic metabolic disorder.

The newer nomenclature also helps clinicians speak more directly with patients. The older term “non-alcoholic” was often confusing and sometimes stigmatising. Some patients interpreted it as an accusation about alcohol. Others assumed that if they did not drink alcohol, the condition had little relevance. MASLD shifts attention toward metabolic risk and

therefore toward modifiable factors such as weight, glycaemic control, diet quality, physical activity, blood pressure, lipids, and alcohol exposure where relevant.

MASH is the subgroup with steatohepatitis, usually defined histologically by steatosis, lobular inflammation, and hepatocellular ballooning, with or without fibrosis. Liver biopsy remains the reference method for diagnosing steatohepatitis and staging fibrosis, and the NASH Clinical Research Network scoring system has been widely used in clinical trials (13). In daily practice, however, biopsy is not suitable as a first-line test for the large number of patients at risk. The practical challenge is therefore to identify who is likely to have advanced fibrosis without sending every patient with steatosis for invasive testing.

Obesity and visceral adiposity

Obesity is one of the strongest clinical drivers of MASLD. The association is not simply a matter of body mass index. Visceral adiposity, waist circumference, ectopic fat, insulin resistance, and adipose tissue inflammation are central to risk. Two patients with the same body mass index may have different liver risk depending on fat distribution, diabetes status, genetics, physical activity, alcohol intake, and metabolic health.

Weight gain increases hepatic fat accumulation. Weight loss can reduce liver fat and improve steatohepatitis. In a prospective lifestyle intervention study of patients with biopsy-proven NASH, Vilar-Gomez et al. found a dose-response relationship between weight loss and histological improvement. Among those who lost at least 10% of body weight, 90% had resolution of NASH and 45% had regression of fibrosis (24). This finding remains clinically important because it gives both clinicians and patients a concrete therapeutic target. Small changes may improve steatosis and liver enzymes, but larger and sustained weight loss is more likely to affect steatohepatitis and fibrosis.

The difficulty is not knowing that weight loss helps. The difficulty is achieving and maintaining it. Many patients with obesity and MASLD have long histories of weight cycling, pain-limited mobility, depression, sleep disturbance, shift work, food insecurity, or medications that promote weight gain. A liver diagnosis may create motivation, but motivation alone rarely produces durable metabolic change. Care needs to move beyond advice to “lose weight” and toward structured support, nutrition counselling, exercise planning, anti-obesity pharmacotherapy where appropriate, and bariatric or metabolic surgery in selected patients.

Bariatric surgery has shown substantial liver benefits in patients with severe obesity. A long-term study reported that five years after bariatric surgery, NASH had resolved without worsening fibrosis in 84% of patients with paired biopsies (29). This does not mean surgery is appropriate for every patient with MASLD. It does show that major and sustained weight loss can alter the course of steatohepatitis in selected patients.

Type 2 diabetes and high fibrosis risk

Type 2 diabetes is one of the most important clinical signals in MASLD. It increases the likelihood of steatosis, MASH, clinically significant fibrosis, and fibrosis progression. In a systematic review and meta-analysis, Younossi et al. reported a high global burden of NAFLD, NASH, and advanced fibrosis among patients with type 2 diabetes (7). An updated meta-analysis in people with type 2 diabetes estimated a high prevalence of NAFLD and found that clinically significant fibrosis and advanced fibrosis were common within this population (8).

The practical implication is direct: type 2 diabetes should trigger active liver fibrosis case-finding. Waiting for high aminotransferases is unsafe. Many patients with significant fibrosis have normal or only mildly abnormal liver enzymes. Alanine aminotransferase can also decline as cirrhosis develops, giving false reassurance if interpreted without clinical context.

In diabetes practice, MASLD should be approached as part of complication screening. Retinopathy, nephropathy, neuropathy, cardiovascular risk, and foot disease are already embedded into diabetes care. Fibrosis risk is less consistently assessed, yet the evidence supports structured evaluation in high-risk patients. This does not require every diabetes clinic to

become a hepatology clinic. It requires a simple first step, usually FIB-4, followed by clear second-line testing and referral thresholds.

Glycaemic control remains important, but the choice of diabetes therapy also matters. Pioglitazone has evidence for histological benefit in patients with NASH, including those with prediabetes or type 2 diabetes, although weight gain and other risks must be considered (26). GLP-1 receptor agonists and dual incretin-based therapies have become particularly relevant because they address weight, glycaemia, and cardiometabolic risk. Semaglutide improved NASH resolution in a phase 2 trial, although fibrosis improvement was not significant in that study (27). Tirzepatide improved MASH resolution without worsening of fibrosis in a phase 2 trial involving patients with moderate or severe fibrosis, but longer and larger studies are needed (31).

Cardiovascular disease remains central

Patients with MASLD often die from cardiovascular disease before they develop liver failure. This is one of the reasons the condition must not be managed as a narrow hepatology problem. In an updated systematic review and meta-analysis of 36 longitudinal studies including more than 5.8 million individuals, NAFLD was associated with increased risk of fatal or non-fatal cardiovascular events, and risk was higher with more advanced liver disease, especially greater fibrosis stage (9).

The mechanisms are overlapping rather than single. Insulin resistance, atherogenic dyslipidaemia, hypertension, systemic inflammation, oxidative stress, endothelial dysfunction, altered coagulation, and visceral adiposity all contribute. MASLD may be a marker of this risk, a mediator of part of it, or both. The distinction matters scientifically, but clinically the response is similar: cardiovascular risk must be assessed and treated.

This includes blood pressure control, lipid management, smoking cessation, glycaemic control, weight management, physical activity, and treatment of sleep apnoea when present. Statins should not be withheld simply because a patient has MASLD or mildly elevated liver enzymes. Patients with MASLD often have a strong indication for lipid-lowering therapy because their cardiovascular risk is high.

The liver assessment should therefore run in parallel with cardiovascular prevention. A clinician who detects MASLD should ask two questions at the same visit. First, does this patient have significant or advanced liver fibrosis? Second, is cardiovascular risk being treated as aggressively as it should be?

Table 1: Cardiometabolic risk groups in whom MASLD and fibrosis case-finding should be considered

Risk group	Why this group matters	Practical first step
Type 2 diabetes	High prevalence of steatosis, MASH, clinically significant fibrosis, and advanced fibrosis	Calculate FIB-4 using age, AST, ALT, and platelet count
Obesity with metabolic risk factors	Visceral adiposity and insulin resistance increase steatosis and fibrosis risk	Check liver enzymes, platelet count, metabolic profile, and calculate FIB-4
Imaging evidence of hepatic steatosis	Steatosis is often found incidentally and may be the first clue to MASLD	Assess metabolic risk and perform fibrosis risk stratification
Persistently abnormal aminotransferases	May indicate steatohepatitis, fibrosis, viral hepatitis, alcohol-related liver disease, or another liver disorder	Exclude common alternative causes and calculate FIB-4

Risk group	Why this group matters	Practical first step
Established cardiovascular disease	MASLD is associated with higher cardiovascular risk and shared metabolic drivers	Assess liver fibrosis risk while optimising secondary prevention
Metabolic syndrome or multiple risk factors	Risk rises when obesity, hypertension, dyslipidaemia, and impaired glucose metabolism coexist	Use a structured liver and cardiometabolic risk pathway
Clinical signs of chronic liver disease	Suggests possible advanced fibrosis or cirrhosis	Refer directly for specialist assessment

Screening should be risk based rather than universal

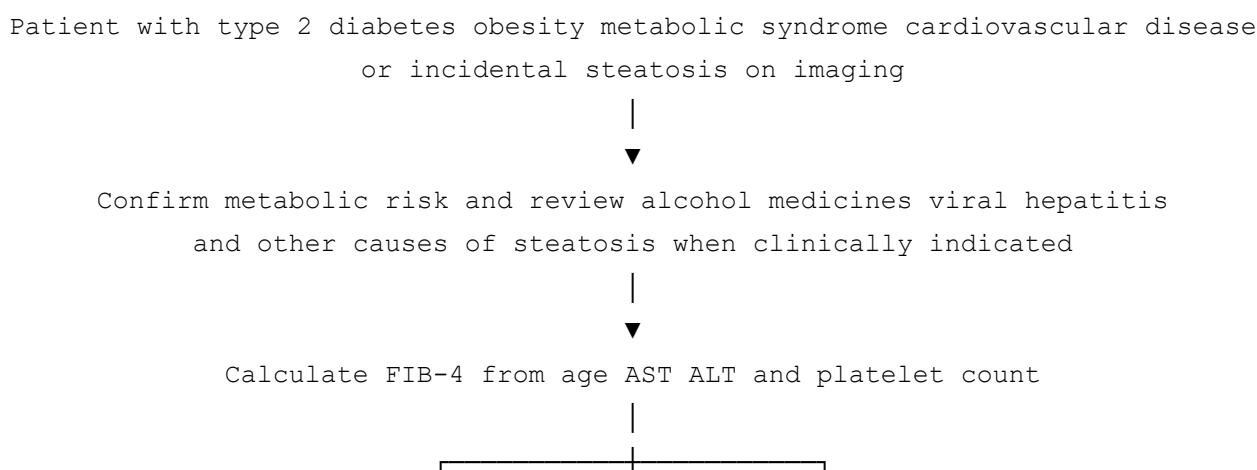
The word “screening” is often used loosely in MASLD. In practice, the best-supported approach is targeted case-finding in high-risk groups rather than universal screening of all adults. Current European guidance recommends case-finding for MASLD with fibrosis using non-invasive tests in individuals with cardiometabolic risk factors, abnormal liver enzymes, or imaging evidence of steatosis, especially in those with type 2 diabetes or obesity with additional metabolic risk factors (2). The AASLD guidance also supports risk stratification using non-invasive tests in appropriate clinical contexts (3).

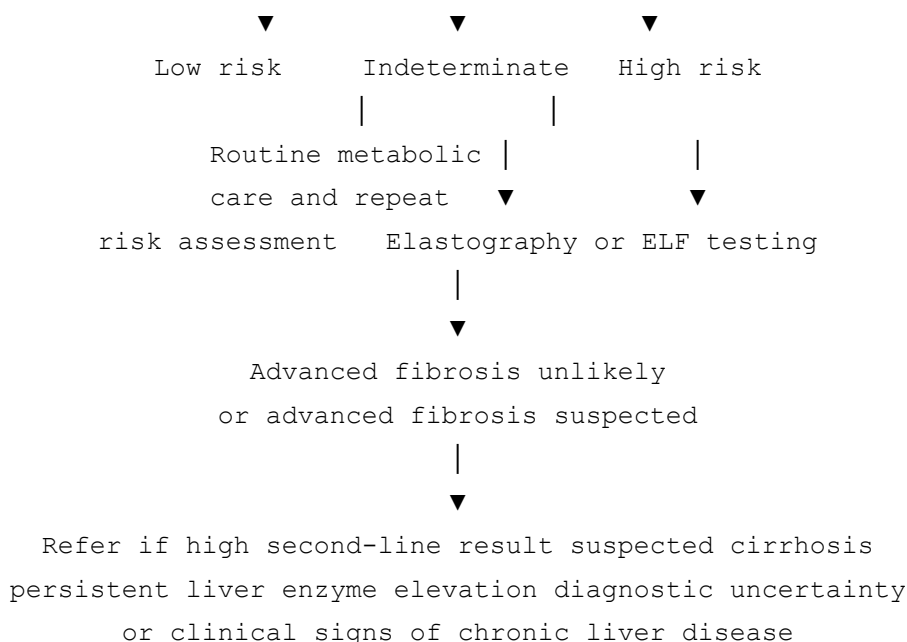
A workable pathway starts with identifying risk. A patient with type 2 diabetes, obesity with metabolic syndrome, incidental steatosis on imaging, persistent liver enzyme elevation, or suspected advanced liver disease should not be left with a passive label. The next question is fibrosis risk.

FIB-4 is commonly used as the first-line test because it requires only age, aspartate aminotransferase, alanine aminotransferase, and platelet count. It is cheap, widely available, and has useful negative predictive value for advanced fibrosis in low-risk results. It was originally developed in patients with HIV and hepatitis C coinfection but has since become widely used in fatty liver pathways (16). The NAFLD fibrosis score is another validated tool, although FIB-4 is often preferred because of simplicity (17).

FIB-4 is not a diagnostic endpoint. It is a triage test. A low result can help exclude advanced fibrosis in many patients. An indeterminate or high result should lead to second-line testing such as vibration-controlled transient elastography, enhanced liver fibrosis testing, or magnetic resonance elastography where available. Results must be interpreted carefully in older adults because age affects FIB-4, and in younger adults because low age can reduce the score despite real disease.

Figure 1 Risk-based pathway for MASLD assessment in cardiometabolic practice





Fibrosis assessment is the pivot point

Steatosis identifies the disease spectrum, but fibrosis identifies much of the risk. This is a central message for clinical practice. In a systematic review and meta-analysis, biopsy-confirmed fibrosis stage was associated with mortality and liver-related morbidity in patients with NAFLD (11). Another meta-analysis found that mortality risk increased with fibrosis stage (12). These findings explain why current pathways are built around fibrosis assessment rather than liver fat alone.

Non-invasive tests are useful because the at-risk population is very large. FIB-4 is suitable for a first step. Transient elastography gives liver stiffness measurement and, when controlled attenuation parameter is included, an estimate of steatosis. Magnetic resonance elastography has strong diagnostic performance but is more expensive and less available (19). The enhanced liver fibrosis test may also help as a second-line blood test in some pathways.

A common error is to stop after detecting steatosis. Another error is to refer every patient with steatosis. Neither approach works. The better strategy is staged risk stratification. Low-risk patients can remain in primary care or metabolic clinics with periodic reassessment. Patients with indeterminate or high fibrosis risk need second-line testing. Patients with suspected advanced fibrosis, cirrhosis, or diagnostic uncertainty should be referred.

Table 2 Non-invasive tools used in MASLD fibrosis assessment

Tool	What it uses or measures	Main clinical value	Limitations
FIB-4	Age, AST, ALT, platelet count	Simple first-line test to exclude advanced fibrosis in many patients	Less reliable at extremes of age; indeterminate results are common
NAFLD fibrosis score	Age, BMI, diabetes, AST/ALT ratio, platelets, albumin	Validated blood-based fibrosis score	More variables; can be indeterminate; less convenient than FIB-4

Tool	What it uses or measures	Main clinical value	Limitations
Vibration-controlled transient elastography	Liver stiffness and controlled attenuation parameter	Common second-line test for fibrosis and steatosis assessment	Failure or unreliable readings can occur with obesity or technical factors
Enhanced liver fibrosis test	Serum markers of matrix turnover	Useful second-line blood test where available	Availability and cost vary
Magnetic resonance elastography	Imaging-based liver stiffness	High diagnostic accuracy for fibrosis staging	Cost, access, and specialist interpretation limit routine use
Liver biopsy	Histology of steatosis, inflammation, ballooning, and fibrosis	Reference standard for MASH diagnosis and trial endpoints	Invasive, sampling variability, cost, not suitable for broad first-line assessment

Chart 1 Clinical meaning of fibrosis stage in MASLD

F0 No fibrosis

Main priority is metabolic risk reduction and periodic reassessment

F1 Mild fibrosis

Lifestyle and cardiometabolic treatment remain central

F2 Clinically significant fibrosis

Higher-risk disease and closer follow-up are needed

F3 Advanced fibrosis

Specialist assessment is usually appropriate

F4 Cirrhosis

Requires cirrhosis care pathway and surveillance for complications

Referral criteria in routine practice

Referral should be based on risk, not anxiety about the word “fatty.” Most patients with low fibrosis risk can be managed outside hepatology, provided metabolic risk is actively treated and fibrosis risk is reassessed over time. Referral becomes important when non-invasive tests suggest advanced fibrosis, when cirrhosis is suspected, when liver enzymes remain persistently abnormal without clear explanation, or when there is uncertainty about competing liver disease.

Practical referral triggers include high FIB-4, indeterminate FIB-4 followed by abnormal elastography or enhanced liver fibrosis testing, imaging signs of cirrhosis or portal hypertension, thrombocytopenia, splenomegaly, low albumin, elevated bilirubin, prolonged prothrombin time, ascites, varices, hepatic encephalopathy, or hepatocellular carcinoma concern. Patients with mixed risk, such as metabolic dysfunction with substantial alcohol intake, viral hepatitis, autoimmune markers, iron overload, or drug-induced liver injury, may also need specialist input.

Referral should not be seen as a transfer of all care. The hepatologist may clarify fibrosis stage and liver-specific management, but cardiometabolic treatment remains shared. Diabetes,

obesity, hypertension, dyslipidaemia, sleep apnoea, and cardiovascular disease still require co-ordinated management.

Table 3 Suggested referral approach after initial MASLD risk assessment

Clinical situation	Suggested action
Steatosis with low FIB-4 and no clinical concern for advanced disease	Manage cardiometabolic risk in primary care or metabolic clinic; repeat fibrosis assessment periodically
Indeterminate FIB-4	Arrange second-line testing with transient elastography, enhanced liver fibrosis test, or local equivalent
High FIB-4	Proceed to second-line testing and consider hepatology referral, especially if risk factors or abnormal tests persist
Elevated liver stiffness or high ELF result	Refer for specialist assessment of advanced fibrosis risk
Imaging or clinical signs of cirrhosis	Refer to hepatology and begin cirrhosis-related evaluation
Persistently abnormal liver enzymes despite metabolic management	Assess for alternative or additional liver disease and consider referral
MASLD with substantial alcohol intake or other liver disease risk	Refer or discuss with hepatology because disease drivers may overlap
Suspected decompensation	Urgent specialist or hospital assessment

Management should start before referral is complete

A patient does not need to wait months for hepatology review before treatment begins. The first-line treatment of MASLD overlaps with good cardiometabolic medicine. Weight reduction, physical activity, diabetes treatment, lipid lowering, blood pressure control, smoking cessation, and reduction of harmful alcohol intake can begin immediately.

Weight loss targets should be realistic but not vague. A 5% weight loss may reduce steatosis. A 7% to 10% loss is more likely to improve steatohepatitis. A loss of 10% or more has the strongest association with NASH resolution and fibrosis regression in lifestyle data (24). Exercise has benefits even when weight loss is modest, including improved insulin sensitivity and hepatic fat reduction.

Diet should be framed around sustainable patterns rather than short-lived restriction. A Mediterranean-style diet, reduced intake of sugar-sweetened beverages, lower consumption of refined carbohydrates, adequate protein, and replacement of ultra-processed foods with whole foods are practical starting points. Patients with diabetes may need individualized carbohydrate planning. Patients with cirrhosis or sarcopenia require different nutritional priorities and should not be given aggressive weight-loss advice without specialist input.

Alcohol assessment remains necessary. MASLD does not mean alcohol is irrelevant. Alcohol can worsen liver risk, and the newer nomenclature recognizes overlapping categories such as metabolic dysfunction with increased alcohol intake. The clinical conversation should be precise rather than moralistic: amount, pattern, binge drinking, and coexisting liver risk all matter.

Pharmacological therapy and metabolic drugs

For many years, MASLD care relied mainly on lifestyle intervention and management of associated metabolic disease. That remains the foundation. However, the pharmacological landscape is changing.

Vitamin E improved histological outcomes in non-diabetic adults with biopsy-proven NASH in the PIVENS trial, but its use requires careful discussion of risks and patient selection (25). Pioglitazone has evidence for benefit in NASH, including in patients with prediabetes or type 2 diabetes, but weight gain, oedema, fracture risk, heart failure concerns, and patient preference must be considered (26).

GLP-1 receptor agonists and related agents are clinically important because they target obesity, diabetes, and cardiovascular risk. Semaglutide showed higher NASH resolution than placebo in a phase 2 trial, although fibrosis improvement did not reach significance in that trial (27). A phase 3 semaglutide trial in MASH with fibrosis has since reported histological benefit (28). Tirzepatide, a dual GIP and GLP-1 receptor agonist, showed promising phase 2 results in MASH with moderate or severe fibrosis (31). These agents should be considered within their approved indications and patient context, especially when obesity or type 2 diabetes is present.

Resmetirom, a thyroid hormone receptor beta selective agonist, demonstrated benefit in a phase 3 trial of patients with NASH and fibrosis (30). Its emergence marks a shift toward liver-directed pharmacotherapy for selected patients with non-cirrhotic MASH and fibrosis. Even so, medication should not displace cardiometabolic care. A drug that improves liver histology does not remove the need to treat obesity, diabetes, blood pressure, lipids, and cardiovascular risk.

Cardiometabolic integration is the practical model

MASLD care works best when liver risk stratification is embedded in existing care pathways. Diabetes clinics can calculate FIB-4 annually or at defined intervals. Primary care can add FIB-4 when steatosis appears on imaging or liver enzymes remain abnormal. Cardiology clinics can recognize MASLD as part of broader metabolic risk, especially in patients with obesity, diabetes, and dyslipidaemia. Obesity medicine services can include liver fibrosis assessment before and after major weight-loss interventions.

Fragmented care leads to predictable gaps. The radiologist reports steatosis, the primary care clinician notes it, the diabetes clinician focuses on HbA1c, the cardiologist focuses on low-density lipoprotein cholesterol, and no one stages fibrosis. A simple pathway can prevent this. The patient needs one visible plan: assess fibrosis risk, treat metabolic disease, reduce cardiovascular risk, and refer only when risk warrants it.

Discussion

MASLD has become a practical test of whether modern medicine can manage multisystem metabolic disease without dividing the patient into separate organs. The liver is important, but it is not the whole story. In many patients, the presence of steatosis reveals years of insulin resistance, visceral adiposity, vascular risk, and inflammatory metabolic stress. The diagnosis should therefore prompt two parallel responses: liver fibrosis risk stratification and cardiometabolic risk reduction.

The most important shift in practice is to move from passive recognition to active staging. Steatosis alone is common and often clinically silent. Advanced fibrosis is less common but far more prognostically important. This is why FIB-4, elastography, enhanced liver fibrosis testing, and referral pathways are central to modern MASLD care. Fibrosis stage predicts liver-related outcomes and mortality more strongly than steatosis or aminotransferase elevation alone (11,12).

Type 2 diabetes deserves special attention. The high prevalence of NAFLD/MASLD and fibrosis in diabetes populations means that liver risk assessment should become part of routine diabetes care, not an occasional reaction to abnormal liver enzymes. Normal

aminotransferases cannot exclude significant fibrosis. This point should be repeated in clinical education because reliance on liver enzymes is one of the reasons MASLD is detected late.

Cardiovascular risk must remain central. The association between NAFLD and cardiovascular events is well documented, and risk increases with more severe liver disease (9). Clinicians should avoid a narrow interpretation of MASLD as a future cirrhosis problem only. For many patients, the more immediate threat is myocardial infarction, stroke, heart failure, or chronic kidney disease. Treating dyslipidaemia, hypertension, diabetes, smoking, sleep apnoea, and obesity is therefore liver care as well as cardiovascular care.

The therapeutic field is also changing. Lifestyle intervention remains foundational, but it must be delivered in a way that patients can actually follow. Newer obesity and diabetes medications offer an opportunity to treat shared metabolic drivers. Liver-directed therapies such as resmetirom may alter the management of selected patients with MASH and fibrosis. The challenge will be choosing patients appropriately, monitoring response, avoiding overmedicalisation of low-risk steatosis, and keeping attention on cardiovascular outcomes.

Several uncertainties remain. Optimal screening intervals are still debated. Non-invasive test cut-offs vary by age, population, and setting. Access to elastography and enhanced liver fibrosis testing is uneven. Many trials use histological endpoints, while patients often care more about fatigue, function, survival, avoidance of cirrhosis, and cardiovascular events. More data are needed in lean MASLD, older adults, South Asian populations, patients with chronic kidney disease, and those with overlapping alcohol and metabolic risk.

Despite these uncertainties, the direction of care is clear. MASLD in a patient with cardiometabolic risk should trigger structured action. The action need not be complicated: identify risk, calculate FIB-4, arrange second-line testing when needed, refer high-risk patients, and treat the metabolic disease seriously.

Conclusion

Metabolic dysfunction-associated steatotic liver disease is common in patients with obesity, type 2 diabetes, cardiovascular disease, and other cardiometabolic risk factors. Its clinical importance lies not only in hepatic steatosis, but in the risk of MASH, progressive fibrosis, cirrhosis, and systemic cardiometabolic complications.

A practical approach should be risk based. Patients with type 2 diabetes, obesity with additional metabolic risk factors, persistent liver enzyme abnormalities, cardiovascular disease, or imaging evidence of steatosis should undergo fibrosis risk stratification. FIB-4 is a useful first-line test, but it should be understood as a triage tool rather than a final diagnosis. Indeterminate or high-risk results require second-line assessment with transient elastography, enhanced liver fibrosis testing, magnetic resonance elastography where available, or specialist review.

Fibrosis stage remains the central prognostic marker. Patients with low fibrosis risk can often be managed in primary care, diabetes clinics, cardiology clinics, or obesity services with periodic reassessment. Patients with suspected advanced fibrosis, cirrhosis, unexplained persistent liver test abnormalities, or overlapping liver disease risks should be referred.

Management should begin early and should not wait for severe liver disease. Weight loss, physical activity, diabetes therapy, lipid management, blood pressure control, cardiovascular prevention, alcohol assessment, and treatment of obesity are all part of MASLD care. New pharmacological options are expanding the field, but they should strengthen rather than replace cardiometabolic management.

MASLD should no longer be treated as an incidental radiology phrase. In patients with cardiometabolic risk, it is a clinical signal that the liver, vasculature, adipose tissue, and glucose metabolism are part of the same disease process. The opportunity is to detect fibrosis earlier, prevent avoidable liver complications, and reduce the wider cardiometabolic risk that often determines the patient's outcome.

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