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Semaglutide as a promising treatment for metabolic dysfunction-associated steatotic liver disease

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ABSTRACT

Metabolic dysfunction-associated steatotic liver disease (MASLD) is the most prevalent liver disorder globally, and if left untreated, it may progress to liver cirrhosis and even hepatocellular carcinoma. As of 2024, the European Association for the Study of the Liver Guidelines have recommended resmetirom as the only pharmacological treatment for adults with non-cirrhotic MASLD who have significant fibrosis (stage ≥2). However, lifestyle interventions and management of comorbidities, such as type 2 diabetes and obesity, remain the cornerstone of treatment. Glucagon-like peptide-1 receptor agonists, particularly semaglutide, have shown emerging promise in treating MASLD. Notably, the Phase III ESSENCE trial, presented in late 2024, demonstrated semaglutide's potential in improving liver fibrosis, confirmed through histological evaluation, marking a possible breakthrough for MASLD management. This review aims to synthesise current evidence on the efficacy of semaglutide in treating MASLD, highlighting its potential to fill a significant gap in the therapeutic options available for this growing global health concern.

MASLD

Definition and epidemiology

Metabolic dysfunction-associated steatotic liver disease (MASLD) is the most prevalent liver disease globally, affecting approximately 30% of the population, with rates continuing to rise [1]. In 2023, a new consensus published by several international societies emphasised the link between metabolic disorders and nonalcoholic fatty liver disease, introducing the term MASLD to describe liver steatosis associated with cardiometabolic risk factors. A more severe form of MASLD called metabolic dysfunction-associated steatohepatitis (MASH), is characterised by lobu-

lar inflammation and hepatocyte ballooning and can progress to hepatic fibrosis, cirrhosis, and hepatocellular carcinoma [2]. MASH is currently the fastest-growing cause of liver cancer among individuals who are potential candidates for liver transplantation [3].

Aetiology

MASLD is a multifactorial disease primarily driven by lipotoxicity, insulin resistance, and activating inflammatory and immune pathways. It is strongly associated with metabolic conditions, particularly type 2 diabetes (T2DM) and obesity [4]. The presence of T2DM and its related comorbidities, such as visceral obesity, hypertension, and dyslipidemia, can accelerate the progression from MASLD to MASH and cirrhosis. On the contrary, MASLD can impair hepatic insulin sensitivity, which may worsen glucometabolic control [4,5].

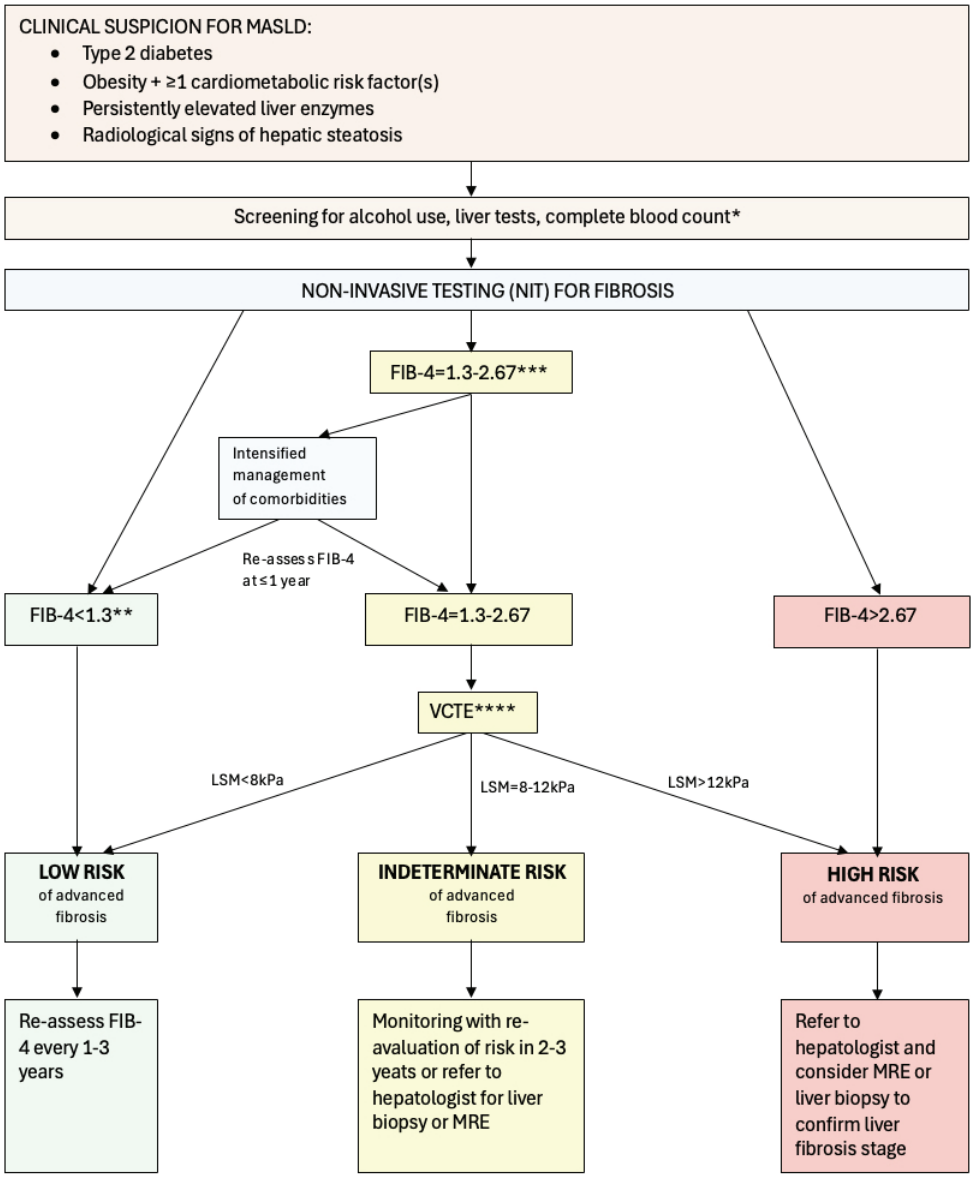

Figure 1. The recommended strategy for MASLD risk stratification [7,8]. *These initial laboratory tests can also identify patients with elevated aminotransferases, all of whom should be evaluated for other chronic liver and biliary diseases. ** The FIB-4 thresholds apply to individuals aged 65 years or younger (for those older than 65, a lower FIB-4 cut-off of 2.0 is used). *** Management of patients with FIB-4 scores between 1.3 and 2.67 depends on medical history, clinical context, and local resources. ****or alternative test, e.g. magnetic resonance elastography (MRE), shear wave elastography (SWE), enhanced liver fibrosis (ELF), with adapted thresholds

Diagnostic process

The diagnosis of MASLD requires the presence of at least one cardiometabolic risk factor in an individual with documented steatosis. Non-invasive screening for steatosis should be considered for patients with obesity/T2DM or raised liver enzymes. B-mode ultrasonography (US) and MR-based techniques such as MRI proton density fat fraction (MRI-PDFF) or proton magnetic resonance spectroscopy (1H-MRS) may be used to assess liver lipid content. Assessing liver fibrosis is essential in MASLD patients, as it is a critical indicator of liver-related outcomes. A multi-step approach is recommended, starting with the fibrosis-4 (FIB-4) score, followed by measurements of liver stiffness measurement (LSM) (vibration-controlled transient elastography [VCTE] or magnetic resonance elastography [MRE]) to assess advanced fibrosis risk. This strategy helps with risk stratification and guides interventions, including speciality referral, for those at intermediate or high risk [6–8] (**Figure 1**).

Treatment

The primary goal of MASLD treatment is to prevent cirrhosis progression to hepatocellular carcinoma and liver failure. Evaluating cardiovascular risk and implementing a multidisciplinary approach is crucial for comprehensive care. First-line therapy for MASLD focuses on lifestyle changes to achieve safe weight loss; however, long-term compliance is often challenging. Resmetirom is the only MASH-targeting drug with positive results from a registrational phase III clinical trial. The latest recommendations for MASLD treatment are shown in **Figure 2** [6–8].

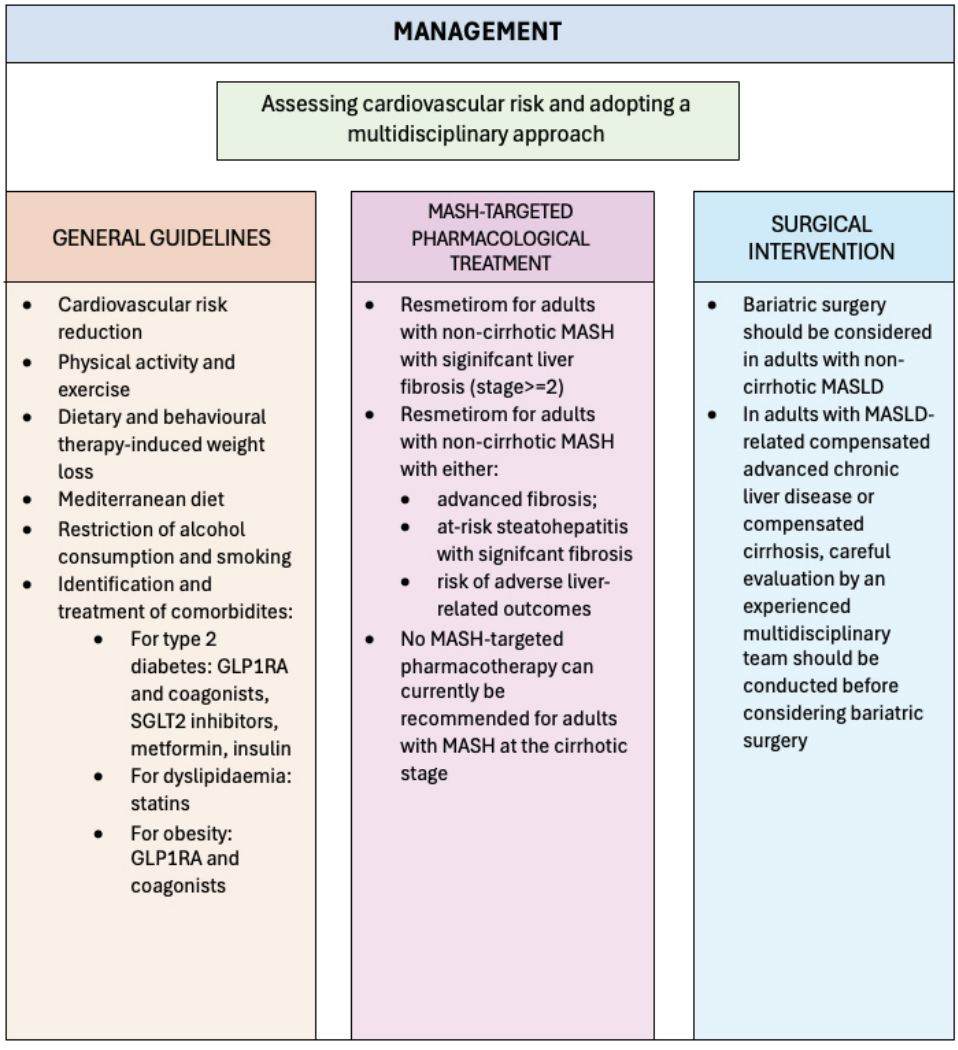

Figure 2. Recommended management of MASLD [6–8]. SGLT2 – sodium/glucose cotransporter 2.

Semaglutide in MASLD treatment

Glucagon-like peptide 1 receptor agonists (GLP-1 RAs), such as semaglutide, are effective glucose-lowering agents for managing T2DM. Treatment with semaglutide leads to weight reduction, decreased cardiovascular risk, and lowered systolic blood pressure, all with minimal incidence of hypoglycemia [9]. In subcutaneous and oral forms, semaglutide demonstrated the greatest glycemic and weight loss benefits from all GLP-1 RAs and has shown reduced rates of major adverse cardiovascular events in patients with T2DM [10,11]. Many recent studies have evaluated GLP-1 RA's potential benefits in MASLD management. GLP-1RAs are thought to provide indirect liver benefits through improved metabolism, weight loss, and their multiorgan effects [12,13]. (**Figure 3**). A recent meta-analysis concluded that daily semaglutide may be the most effective treatment for MASLD and T2DM compared to other GLP-1 RAs [14].

Newsome et al. conducted one of the first trials on semaglutide's efficacy in MASLD patients in 2021 and showed promising results. In the phase IIb study involving 320 patients with MASH and liver fibrosis, daily semaglutide 0.4 mg for 72 weeks led to more patients achieving MASH resolution without worsening fibrosis than placebo. While the trial did not show significant improvements in fibrosis, it was suggested that a more extended treatment period might be needed to evaluate its effectiveness [15] entirely. A 2024 post-hoc analysis using Artificial Intelligence machine learning models found a significant reduction in biopsy slides from the mentioned trial, suggesting it was a more sensitive method than traditional histopathology [16]. Subsequent trials on semaglutide for MASLD have been conducted, and their findings are summarised in **Table 1**.

After showing inconsistent results in improving MASH-related fibrosis, a significant breakthrough occurred at the end of 2024. The first

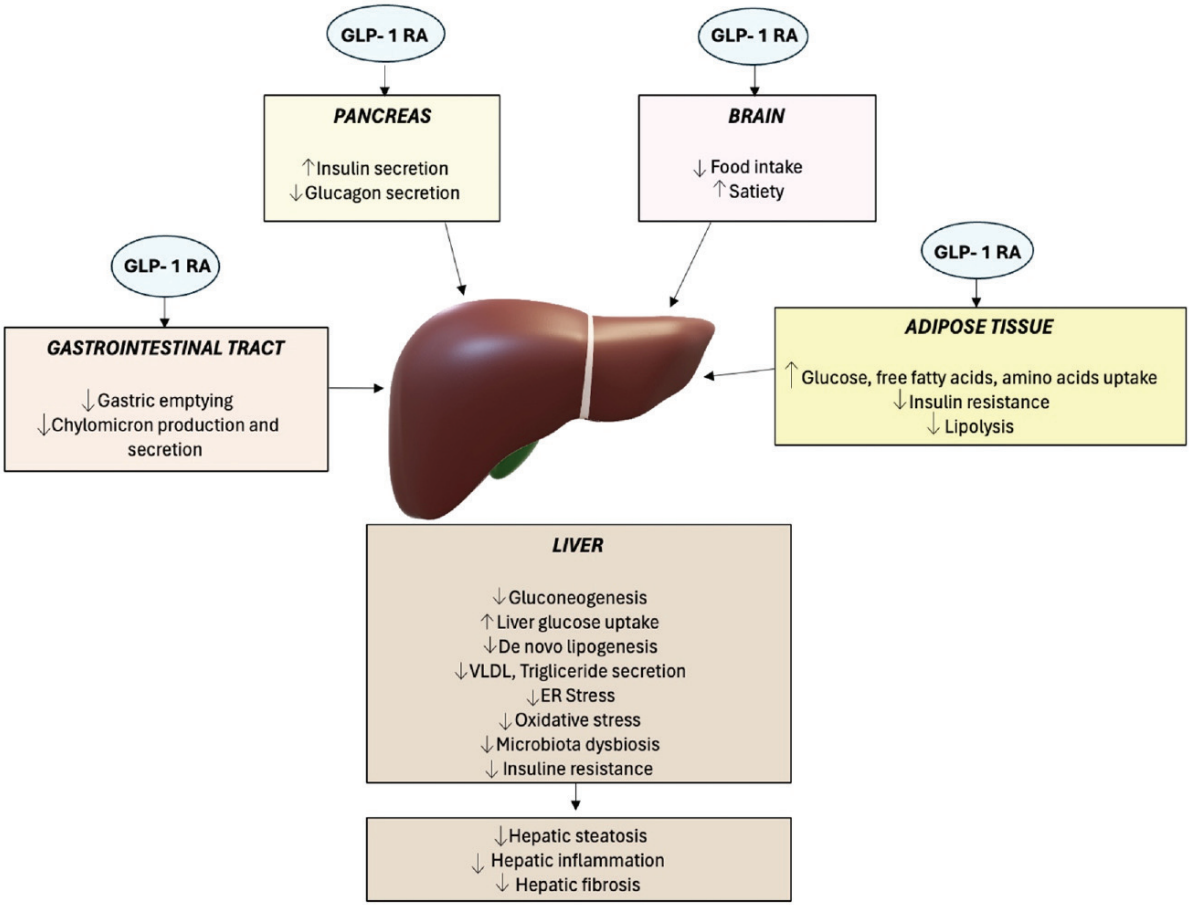

Figure 3. Overview of the pleiotropic effects of GLP-1RAs in managing MASLD/MASH [12,13]. ER – endoplasmic reticulum.

Table 1. The latest publications on the efficacy of semaglutide in MASLD [19–25].

Publication	Methods	Results
Gad et al. 2024	An open-labelled intervention study with 180 patients classified into three parallel groups (1:1:1): – group I received oral semaglutide (up to 14mg) daily – group II patients received injectable semaglutide (up to 2mg) once-weekly – group III received pioglitazone and/or vitamin E	– A substantial improvement in lipid profile, liver enzymes, and body mass index, especially in group II – Only group II showed a consistent increase in HDL – Group II had significantly lower scores of the fibrosis-4 score (FIB-4), liver stiffness measurement (LSM), and controlled attenuation parameter (CAP) at 6 and 12 months ($p < 0.001$)
Loomba et al. 2023	A double-blind, placebo-controlled phase 2 trial with 71 patients with biopsy-confirmed MASH-related cirrhosis and body-mass index (BMI) of 27 kg/m2 or more, who were randomly assigned (2:1) to receive either once-weekly subcutaneous semaglutide 2.4 mg or placebo	– After 48 weeks, there was no statistically significant difference between the two groups in the proportion of patients with an improvement in liver fibrosis of one stage or more without worsening of MASH – There was also no significant difference between groups in the proportion of patients who achieved MASH resolution ($p = 0.29$)
Volpe et al. 2022	A prospective, single-arm, real-life study with 48 patients treated with subcutaneous semaglutide (up to 1mg) once-weekly in add-on to metformin for 52 weeks	– A significant decrease in anthropometric and glucometabolic parameters, insulin resistance, liver enzymes, and laboratory indices of hepatic steatosis during treatment. – Fat mass and visceral adipose tissue (VAT) decreased – Ultrasound-assessed VAT thickness and the 12-point steatosis score also declined at T3 up to T12 – Liver steatosis improved in most patients (70%), showing a reduction by at least one class in the semiquantitative ultrasound staging
Flint et al. 2021	A randomised, double-blind, placebo-controlled trial with 67 patients with liver stiffness 2.50-4.63 kPa by magnetic resonance elastography (MRE) and liver steatosis ≥10% by MRI proton density fat fraction (MRI-PDFF), randomised to once-daily subcutaneous semaglutide 0.4 mg (n = 34) or placebo (n = 33).	– Decrease in liver enzymes, body weight and HbA1c with semaglutide – Reductions in liver steatosis were significantly greater with semaglutide (estimated treatment ratios: 0.70 [0.59, 0.84], $P = 0.0002$; 0.47 [0.36, 0.60], $P < 0.0001$; and 0.50 [0.39, 0.66], $P < 0.0001$) and more subjects achieved a ≥ 30% reduction in liver fat content with semaglutide at weeks 24, 48 and 72, (all $P < 0.001$) – Not signifcant differences in liver stiffness with semaglutide
Kitsunai et al. 2025	– A secondary analysis of a multicenter, retrospective, observational study, analyzing oral semaglutide up to 14 mg once-daily – Subjects with suspected MASLD were placed in an overall group; a subpopulation from an overall group at high risk for hepatic fibrosis was placed in a high-risk group (n = 67); and the remaining subjects were placed in a low-risk group (n = 102)	– Oral semaglutide significantly improved the hepatic steatosis index (from 46.1 to 44.6, $p < 0.001$) and FIB-4 (from 1.04 to 0.96, $p < 0.001$) – Improvement in the FIB-4 index was significantly negatively correlated with the baseline FIB-4 index. – HbA1c, body mass index, systolic blood pressure, and lipid profile decreased in the overall cohort – The mean values of liver enzymes showed a significant improvement
Arai et al. 2022	A single-arm, open-label pilot study with 16 patients receiving semaglutide initiated at a dose of 3 mg once daily, which was sequentially increased to 7 mg at 4 weeks and 14 mg at 8 weeks (maintenance dose)	– Semaglutide decreased body weight, levels of liver-related biochemistry, plasma glucose, HbA1c, HOMA-IR, triglyceride, CAP and liver fibrosis markers (fibrosis-4 index, ferritin, and type IV collagen 7 s) – Changes in body weight were correlated with those in levels of ALT (alanine aminotransferase) and CAP – Semaglutide did not decrease liver stiffness measurement
Alkhouri et al. 2022	A phase II, open-label, proof-of-concept trial involved patients with mild-to-moderate fibrosis due to MASH, who were randomized to three groups: A. Semaglutide 2.4 mg/week (n = 21) B. Semaglutide 2.4 mg/week and once-daily, cilofexor 30 mg (n = 22) C. Semaglutide 2.4 mg/week and once-daily, cilofexor 100 mg (n = 22)	Compared with semaglutide monotherapy, combination treatments resulted in greater improvements in liver steatosis measured by MRI-PDFF (least-squares mean of absolute changes: ranging from −9.8% to −11.0% vs.−8.0%; the difference was statistically significant only between the semaglutide and semaglutide + firsocostat groups) as well as in non-invasive tests of liver fibrosis and liver biochemistry

results of the Phase III ESSENCE trial emerged, which evaluated once-weekly 2.4 mg semaglutide in adults with MASH and moderate to advanced liver fibrosis (F2–F3). At The Liver Meeting 2024, Phil Newsome (London, UK) presented an interim analysis from the 72-week data, highlighting promising results in addressing MASH-related fibrosis. Semaglutide demonstrated significant superiority over placebo on both primary endpoints. A greater proportion of patients receiving semaglutide achieved resolution of steatohepatitis without worsening fibrosis. More notably, 37% of semaglutide-treated patients showed improved fibrosis without worsening steatohepatitis, compared to 22.5% in the placebo group. This study confirmed semaglutide's effectiveness in improving liver fibrosis, with the histological improvements assessed through liver biopsies [17].

Conclusions

The ESSENCE trial and AI-driven liver histology assessments highlight semaglutide's potential to transform MASH-related fibrosis treatment. Promising results show semaglutide outperforms placebo in improving liver histology and fibrosis, offering a new therapeutic option for MASH. Following the positive outcomes of the trial, Novo Nordisk expects to file for regulatory approvals in the US and EU in the first half of 2025. Given these findings, continued investment in clinical trials is essential to explore the long-term benefits of semaglutide in MASLD management. There are ongoing trials combining semaglutide with other therapies, such as luseogliflozin (UMIN000045003) and zalfermin (NCT05016882). Recent studies have also highlighted the effectiveness of novel incretin-based analogs, including dual GLP-1/GIP agonists (tirzepatide), dual GLP-1/glucagon agonists (survodutide, pemvidutide, and cotadutide) and GLP-1/GIP/GCGR agonist (retarutide) in reducing liver fat and fibrosis. These innovative therapies offer promising potential for the future management of MASLD [18].

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Conflict of interest statement
The authors declare no conflict of interest.

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