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RESEARCH ARTICLE

# Metabolic Dysfunction Associated Steatotic Liver Disease (MASLD): A General Overview and Approach in Clinical Practice

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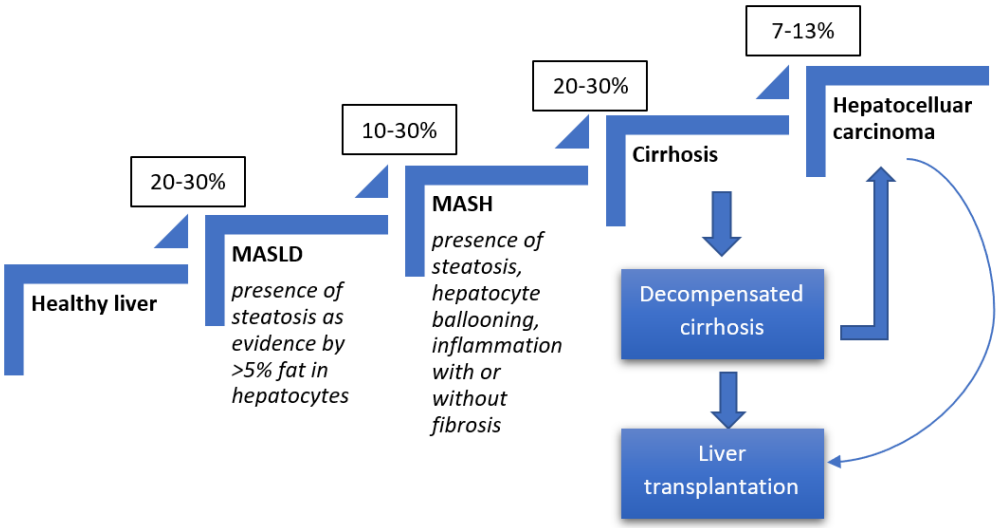
ABSTRACT
Metabolic dysfunction associated steatotic liver disease (MASLD) or previously known Non-alcoholic fatty liver disease (NAFLD) is a common condition with an estimated global prevalence of around 30%. It is becoming a public health concern due to its close association with type 2 diabetes mellitus and obesity. It is important to screen for those with inflammation and fibrosis to halt the progression to cirrhosis. Cirrhosis is associated with liver related complications and liver cancer. Currently, there are no targeted treatments for MASLD at this stage and most treatments are currently in clinical trials. The focus of treatment had been on managing underlying risk metabolic risk factors.
The purpose of this review to inform the readers of the change in the nomenclature from NAFLD to MASLD. This review will also focus on the background of MASLD, the pathogenesis as well as assessment and treatment of patients with MASLD.
**Keywords:** Non-alcoholic fatty liver disease (NAFLD), Metabolic dysfunction associated steatotic liver disease (MASLD), steatosis, steatohepatitis, fibrosis, cirrhosis

## Introduction

Metabolic dysfunction associated steatotic liver disease (MASLD), previously known as non-alcoholic fatty liver disease (NAFLD) is defined by the presence of steatosis or an accumulation of fat (>5%) in hepatocytes, in the absence of significant alcohol consumption or other causes of liver injury. NAFLD encompasses steatosis (without inflammation), steatohepatitis (with presence of hepatic inflammation with or without fibrosis) and ultimately cirrhosis.[1]The presence of an inflammation in the liver as a result of fatty changes (known as steatohepatitis) are at high risk for liver-related complications and hence, it is important to screen patients for the presence of advanced fibrosis.

The Global Burden of Disease (GBD) study showed that MASLD is the most rapidly increasing disease burden of chronic liver disease. [2] The rapid incline in the prevalence of MASLD is driven by the rising in obesity and type 2 diabetes mellitus (T2DM). A recent systematic review showed that the overall global prevalence is around 30% and there is going to be a 50.4% increase in the prevalence of NAFLD over about 3 decades.[3] A recent study predicated that the prevalence of NAFLD was going to be over 55% by 2040. [4] NAFLD can be a progressive disease leading to inflammation or non-alcoholic steatohepatitis (NASH), fibrosis, cirrhosis and subsequently to development of hepatocellular carcinoma. Around 20% of those with NAFLD will progress into NASH. [5] The progression of MASLD is shown in Figure 1.

**Figure 1: The progression of metabolic dysfunction associated steatotic liver disease (MASLD)**




Non alcoholic steatohepatitis (NASH) is characterized by the presence of steatosis with lobular inflammation, hepatocyte ballooning and fibrosis. Since theses changes associated with NASH is only seen in the liver histology, the biopsy is essential in differentiating between simple steatosis vs steatohepatitis. However, liver biopsy carries significant risk and in clinical setting, non-invasive assessment of fibrosis was used, and the most commonly used is transient elastography (TE) or Fibroscan. TE is helpful in assessing patients at risk of fibrosis as well as in the monitoring of patients longitudinally over time. In most hospital, 2 step fibrosis assessment are used. The most common indirect markers used are either FIB-4 or NAFLD Fibrosis score (NFS) followed by second stage assessment using either TE or serum markers known as ELF (Enhanced Liver Fibrosis).

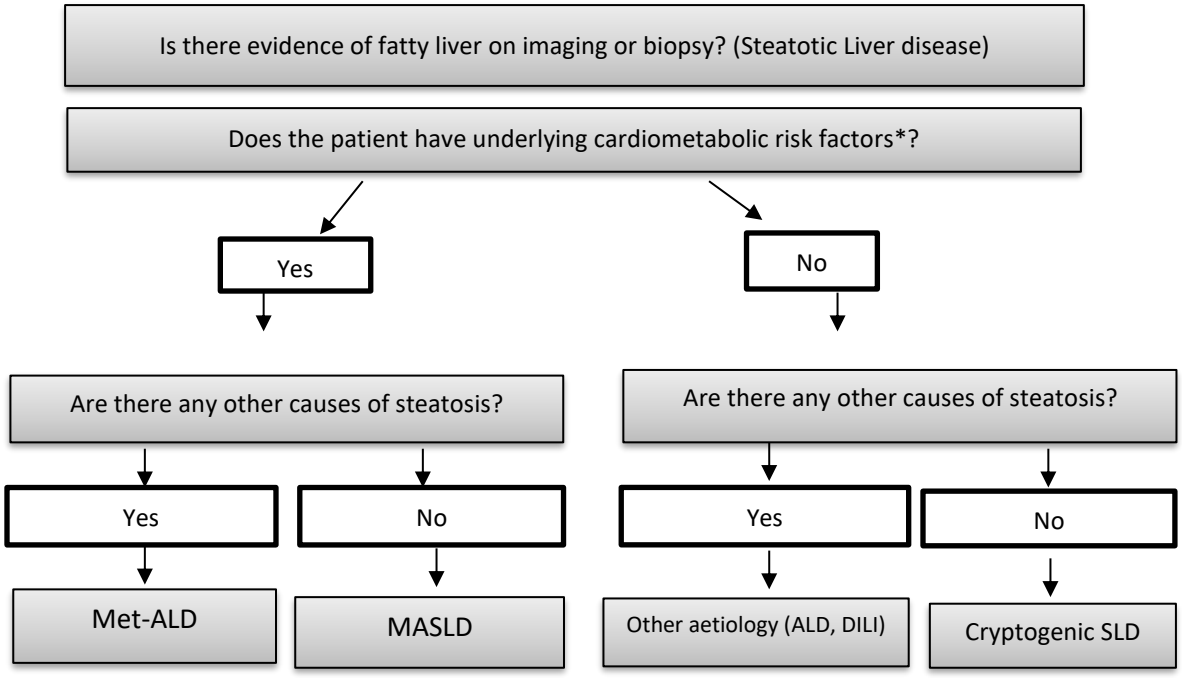
The main step in development of MASLD is an abnormality in the metabolism of hepatic fatty acids (FA). [6] The pathophysiology is complex and involves multiple hits to the liver caused by lipotoxicity, insulin resistance and activation of inflammatory and immune pathways.[7] Genetic factors play a role in the pathway in the pathogenesis of MASLD. A genome-wide association studies (GWASs) of European NAFLD cases showed that PNPLA3 had significant for steatosis and fibrosis.[1] The other genes identified in this study include TM6SF2, GCKR and HSD17B13. [1]

There is also increasing evidence of a link between the gut microbiome and the development of insulin resistance and MASH. It is presumed that an increased in the intestinal permeability results in the transport of microbial metabolites from the gut into the liver contributing to the development of NAFLD or MASLD by dysregulation of the gut-liver axis. [8] Many of the studies are currently looking at the alteration of this pathogenic bacterial population by using probiotics, prebiotics, synbiotics and faecal microbiota transplantation (FMT).[8] The beneficial effects of probiotics on NAFLD have been demonstrated in animal models, and the most

widely used microorganisms are those of the *Lactobacillus* and *Bifidobacterium* genera. [9]

In early 2020, an international panel of experts led a consensus-driven process to develop a more appropriate term for NAFLD.[10] From this process, a recent Delphi consensus statement for the nomenclature of fatty liver disease proposed new definition which was now known as Metabolic Dysfunction Associated Steatotic Liver Disease (MASLD). [11] Although fatty changes in the liver is mostly related to the underlying metabolic risk factors such as obesity, diabetes mellitus or dyslipidaemia, it can also be seen in those with excess alcohol intake. In this new nomenclature, there is sub-category for those with combination of metabolic risk factors and alcohol excess and now known as MetALD. The new diagnostic criteria for patients with fatty liver disease are shown in Figure 2.

**Figure 2: New diagnostic criteria for those with liver steatosis**[11] *(adapted from Rinella et al; Hepatol. 2023 Jun 20:101133.)*




***Cardiometabolic risk factors (Adult criteria)**

At least 1 out of 5 criteria

1. Weight:
    a. Asian (BMI ≥ 23 kg/m²) or BMI ≥ 25 kg/m² for other ethnicity OR
    b. Waist circumference (WC): >94cm in male and 80cm in female
2. Glucose:
    a. Fasting serum glucose ≥ 5.6mmol/L (100 mg/dL) OR
    b. 2-hour post load glucose level ≥ 7.8 mmol/L (≥ 140mg/dL) OR
    c. HbA1c ≥ 5.7% (39 mmol/L) OR
    d. Type 2 diabetes OR
    e. On treatment for type 2 diabetes
3. Blood pressure
    a. BP ≥ 130/85mmHg OR
    b. Specific antihypertensive medication
4. Lipids
    a. Plasma triglycerides ≥ 1.70 mmol/L (150 mg/dL) OR
    b. Plasma HDL-cholesterol ≤ 1.0 mmol/L (40 mg/dL) in male and ≤ 1.3 mmol/L (50 mg/dL) OR
    c. Lipid lowering medication

## How do we risk stratify for liver fibrosis in patients with MASLD?

It is important to identify patients at risk of liver fibrosis to prognosticate liver-related risk. Fibrosis risk assessment can be challenging due to multiple profiles available in clinical setting. Some are more widely used in day-to-day clinical practice. A meta-analysis evaluating NAFLD cohort studies showed that the patients with fibrosis stage of 2 or above are at greater risk of disease progression and liver-related mortality. [12]

There are non-invasive assessments using either biomarkers or elastography or combination of both to rule out patients with advanced liver disease without needing for a liver biopsy. There are direct and indirect fibrosis biomarkers and they are shown in Table 1. The most commonly used indirect and direct biomarkers in the United Kingdom (UK) are FIB-4, NAFLD Fibrosis score (NFS) and ELF (Enhanced Liver Fibrosis). In the UK, most of the liver centres use a 2-step referral pathway which increased the identification of patients with probable high risk of liver fibrosis. This leads to appropriate secondary care specialist referral. Having a 2-step referral pathway reduced unnecessary referrals to the specialist care by 80%.[13] Transient elastography or Fibroscan is widely used in the secondary care setting to identify those with liver fibrosis.

**Table 1: Indirect and Direct serum markers used in assessment of liver fibrosis**

| Indirect Fibrosis Biomarkers | Direct Fibrosis Biomarkers |
|---|---|
| AST: ALT ratio | ELF (Enhanced Liver Fibrosis): an extracellular matrix (ECM) marker set consisting of tissue inhibitor of metalloproteinases 1 (TIMP-1), amino-terminal propeptide of type III procollagen (PIIINP) and hyaluronic acid (HA) |
| AST to platelet ratio index (APRI) | FibroTest: Alpha-2-macroglobulin, Haptoglobin, Apolipoprotein A1, GGT, Total bilirubin, and ALT |
| BARD score (BMI, AST/ALT ratio, T2DM) | FiboMeter: Platelets, Prothrombin level, AST, ALT, Urea/BUN. Alpha-2-macroglobuin, GGT, Hyaluronic acid, age, gender |
| FIB-4 score (Age, AST, ALT, Platelet count) | ADAPT: pro-C3 (a marker of type 3 collagen formation), platelet count, age, the presence of diabetes |
| NAFLD Fibrosis Score (Age, BMI< Impaired fasting glucose or diabetes, AST, ALT, Platelet count, Albumin) | FIBC3: age, BMI, T2DM, platelet count, Pro-C3 |
| | ABC3D: Age, BMI, Platelet count, Pro-C3, T2DM |

AST: ALT, aspartate transaminase: alanine transaminase; ELF, enhanced liver fibrosis; FIB-4, Fibrosis-4 index; NAFLD, non-alcoholic fatty liver disease; T2DM: Type 2 diabetes mellitus; GGT: Gamma-glutamyl transpeptidase, BMI: Body mass index

Liver biopsy is the gold standard reference to identify those with underlying inflammation and fibrosis (simple steatosis vs steatohepatitis vs fibrosis). Liver biopsy can be used to assess the severity of the disease (grade) and the stage (fibrosis) of the disease.[7] However, the liver biopsy is not a risk-free procedure and the sample variability can be challenging.

In the UK, there had been a quality standard recommendation of NAFLD which has 2 parts.[14] The first part is how to manage patients with or at risk of NAFLD in the primary care setting before being reviewed in the gastroenterology or liver clinic and the second part is for assessment and investigations of NAFLD in the secondary care setting. These quality standards are shown in table 2.

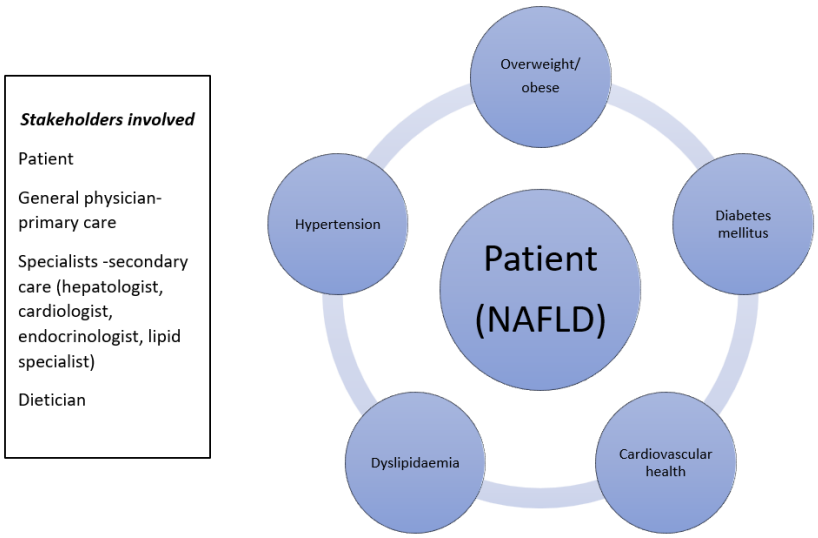
**Table 2:** Summary of the NAFLD quality standard recommendations [14]

| |
|---|
| **Management of people with, or at risk of, NAFLD before the gastroenterology or liver clinic**<br>1. Services should have an agreed local clinical pathway for the investigation of suspected liver disease that includes an assessment for liver fibrosis using available non-invasive liver fibrosis tests.<br>2. Consider the possibility of liver fibrosis due to NAFLD in people with type 2 diabetes or metabolic syndrome<br>3. Do not rely on abnormal liver blood tests to prompt consideration of liver disease. However, persistently unexplained abnormal liver blood tests should always be investigated<br>4. The finding of liver steatosis on ultrasound, or unexplained abnormal liver blood tests, should prompt risk assessment for liver fibrosis<br>5. Use validated widely available non-invasive tests (eg, FIB-4 score or NAFLD fibrosis score) with high negative predictive value to risk assess for clinically significant liver fibrosis in the community<br>6. Refer patients stratified as high risk for advanced fibrosis or cirrhosis to a hepatologist. For patients stratified as indeterminate risk, offer further discriminatory tests (eg, transient elastography or Enhanced Liver Fibrosis test) or refer for further evaluation<br>7. Manage people at low risk of significant fibrosis in the community, with focus on lifestyle advice and cardiovascular risk reduction. Reassess fibrosis using non-invasive tests every 3 years<br>8. Secondary care liver services and community services should collaborate on audits, research, and education to share knowledge, strengthen links, and encourage service and quality improvement, and involve patients as part of this as appropriate |
| **Assessment and investigations in secondary care**<br>1. Patients with NAFLD should be assessed for additional causes of steatosis (eg, drugs and alcohol) and undergo investigations for other causes of liver disease (ie, completion of blood aetiology screen) if these were not already done in primary care<br>2. Patients with NAFLD should have a detailed alcohol (eg, AUDIT-C), illicit drug, and smoking history documented<br>3. Practitioners should document a treatment history and medicines use review. The rationalisation of medicines that may accelerate disease progression should be considered<br>4. An assessment of dietary habits and physical activity levels should be obtained<br>5. Patients with NAFLD should undergo sequential use of a simple non-invasive test (eg, FIB-4) and specialist non-invasive tests (eg, ELF, transient elastography, or acoustic radiation force impulse elastography) to assess the severity of fibrosis<br>6. Patients with NAFLD should be considered for a liver biopsy in the following situations: (A) if there is diagnostic uncertainty (other aetiologies or overlap conditions); (B) to evaluate the severity of NASH and be considered for potential drug therapies (including clinical trials); or (C) to determine the stage of liver fibrosis where non-invasive tests are inconclusive to aid with future management (eg, F4 for hepatocellular carcinoma surveillance)<br>7. Liver biopsies should be processed, stained, and examined according to the UK Royal College of Pathologists guidelines and reported by pathologists who participate in the liver External Quality Assurance scheme using a validated score such as the NASH Clinical Research Network criteria (NAS) or steatosis activity fibrosis (SAF) score<br>8. Patients with NAFLD cirrhosis should be offered surveillance for complications of cirrhosis, including hepatocellular carcinoma and varices, in accordance with national or international recommendations. The Baveno VI exclusion criteria should be considered as a non-invasive tool to rule out the presence of varices requiring treatment<br>9. People with NAFLD should undergo systematic assessment of cardiovascular risk factors including use of an objective risk score (eg, QRISK-3)<br>10. Patients with NAFLD should be screened annually for type 2 diabetes (using HbA1c), hypertension, and dyslipidaemia |

## Treatment of MASLD

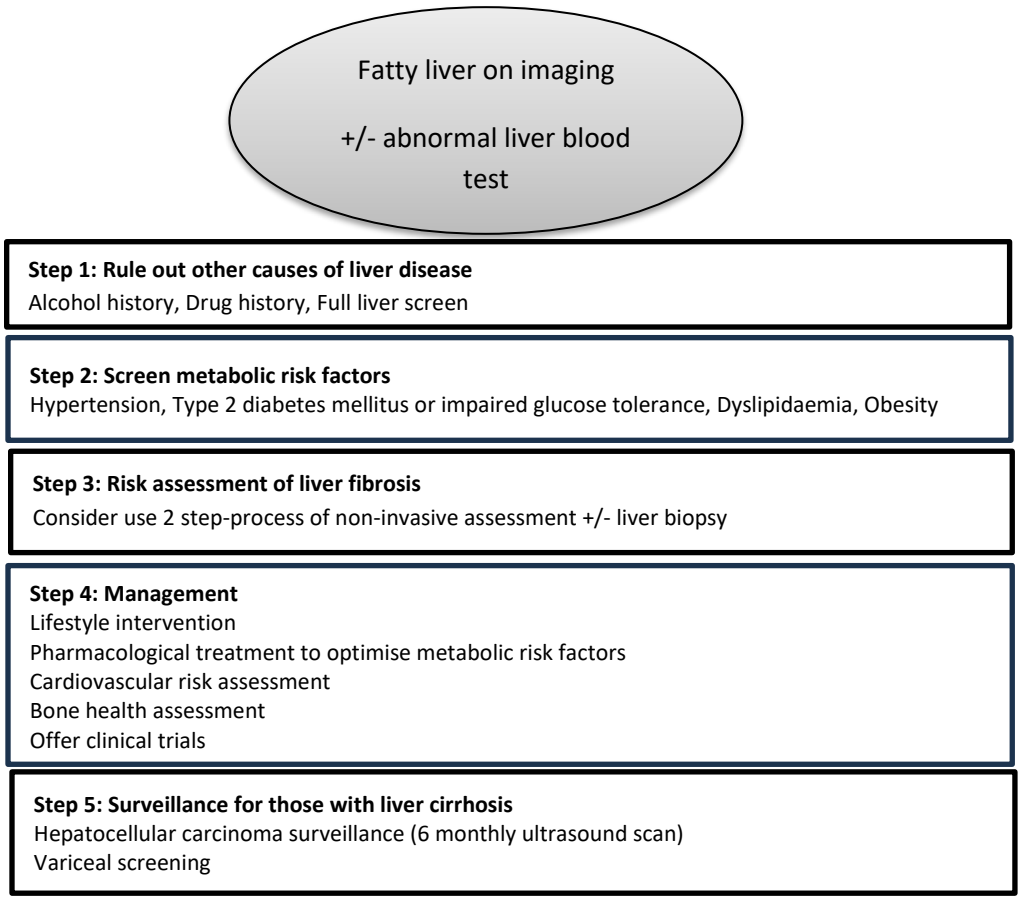
The management of patient with MASLD should be in the settings of a multidisciplinary team (MDT) and the role of the MDT approach is to improve the metabolic risk profile of patients with MASLD. The key state holders are mentioned in Figure 3.

**Figure 3: Multi-disciplinary approach in management of MASLD**




There are no licensed treatments for MASLD despite its increasing prevalence. The current treatment is focused on managing underlying metabolic risk factors (diabetes, or insulin resistance, hypertension, dyslipidaemia and obesity). There seems to be a positive associated between T2DM and the prevalence of liver steatosis and fibrosis. [15] Cardiovascular risk assessment and bone health assessment are also essential. A cardiovascular risk assessment can be performed using the QRISK3 calculator (https://www.qrisk.org). It is important to screen for the underlying risk since the cardiovascular related mortality is the most common cause of death in patients with MASLD. A recent meta-analysis showed that NAFLD was associated with decreased bone mineral density (BMD) and increased risk of osteoporosis and osteoporotic fractures.[16] The systemic approach to the assessment and management of MASLD are shown in Figure 4.

**Figure 4: Systematic approach to patients with steatotic liver disease**



## Weight management

Lifestyle interventions, such as dietary calorie restriction and exercise, to promote weight loss are currently the main form of therapy although it can be difficult to achieve and maintain.[17] It had been proposed that weight loss of ≥ 5% is required to improve hepatic steatosis, ≥ 7% to improve liver inflammation and ≥ 10% to improve fibrosis.[18]There are pharmacological, endoscopic and surgical interventions available for weight management. A recent open-label randomised trial (BRAVES) bariatric surgery (Roux-en-Y gastric bypass or sleeve gastrectomy) had been shown to improve histological resolution of NASH without worsening of fibrosis at 1 year follow-up compared to lifestyle modification and best medical care. [19]This study showed that Roux-en-Y gastric bypass seems to have a better reduction of NASH and fibrosis compared to sleeve gastrectomy. [19]

Many novel therapies are in phase 3 and the expected clinical outcomes are due to be available between 2025 and 2028 (shown in Table 3). The four major groups currently in phase 3 trials are FXR agonist, Pan-PPAR agonist and Glucagon like peptide-1 (GLP-1) receptor agonist and THRβ agonist.

**Table 3: Phase 3 clinical trials in treatment of MASLD**

| Medication | Drug class | Trial | Trial ID | NASH resolution | Reduction if Fibrosis |
|---|---|---|---|---|---|
| OCA | FAR-agonist | REGENERATE Study<br>REVERSE Study | NCT 02548351<br>NCT 03439254 | No<br>- | Yes<br>- |
| Elafibranor | PPAR-α/δ agonist | RESOLVE-IT Study | NCT 02704403 | No | No |
| Lanifibranor | Pan-PPAR agonist | NATiV3 | NCT04849728 | | |
| Resmetirom | TSH-β agonist | MESTRO-NASH Study<br>MESTRO-NASH Outcomes | NCT 03900429<br>NCT 05500222 | - | - |
| Cenicriviroc | CCR2/CCR antagonist | AURORA Study | NCT 03028740 | - | No |
| Selonsertib | Antifibrotic | STELLAR-3, 4 Study | NCT 03053050,<br>NCT 03053063 | No | No |
| Semaglutide | GLP-1 RA | ESSENCE Study | NCT 04822181 | | |

## Drugs currently in phase 3 clinical trials for MASLD

1. Farnesoid X receptor (FXR) agonists

FXR plays an important role in bile acid metabolism but also in metabolic, inflammatory and fibrogenic pathways.[20] FXR, also known as a bile acid receptor, is a key nuclear receptor and activated by bile acids and expressed at high levels in the liver and the terminal ileum. [21]There are two generations of FXR agonists. The first generation is obeticholic acid (OCA) and the second generation is cilofexor and tropifexor.[22] OCA has been approved for the treatment of primary biliary cholangitis (PBC). The most common side effects of OCA are pruritus, increase in low density lipoprotein (LDL) cholesterol and cholecystitis. [23] An 18-month interim analysis from a multicentre, randomised, placebo-controlled phase 3 trial of OCA showed improvement in fibrosis with no worsening of non-alcoholic steatohepatitis (NASH) in patients with stage F2 or F3 fibrosis. [24]

2. Peroxisome proliferator-activated receptor (PPAR) agonists

PPAR agonists, ligand-activated transcription factors, are the critical regulators of fatty acid metabolism, glucose metabolism, inflammation and fibrogenesis. [20,22] There are 3 iso-subtypes of PPAR (PPAR α, PPAR β/δ and PPAR γ) [20]. The mechanisms of PPAR agonists are shown in the table 4. [25] PPAR-α are expressed in the liver and hepatic PPAR-α stimulates mitochondrial FFA uptake, β-oxidation, ATP production and ketogenesis. [21]

**Table 4: The mechanism of action of Peroxisome proliferator-activated receptor (PPAR) agonists**

| PPAR-agonists | Mechanism of action |
|---|---|
| PPAR-α | Reduces plasma triglyceride levels |
| PPAR- β/δ | Enhances fatty acid metabolism and had anti-inflammatory effects by inhibiting inflammatory macrophages |
| PPAR- γ | Promotes insulin sensitisation and a role in lipid storage |

Elafibranor is the first member of the PPAR-α/δ agonist family. [22] Lanifibranor is a pan PPAR agonist and has antifibrotic and positive metabolic effects.[26] Saroglitazar is a dual agonist of PPAR-α/δ and had a benefit in reducing transaminase levels and improving liver steatosis. [27]

3. Glucagon-like peptide-1 (GLP-1) analogues

GLP-1 reduces liver steatosis and improve insulin resistance in the murine models of fatty liver disease.[28] There are 3 subclasses of GLP-1; 1) GLP-1 receptor agonists (GLP-1 RA) (liraglutide and semaglutide), 2) Dual glucose-dependent insulinotropic polypeptide (GIP) and GLP-1 RA (tirzapatide), and 3) Dual glucagon and GLP1-RA (cotadutide).[22] GLP-1 is a hormone with an incretin effect that stimulates insulin secretion by intestinal cells after a meal, in addition to glucagon suppression.[22] It also exerts an effect on weight reduction by activation hypothalamic GLP1-receptors, enhancing satiety and delayed gastric emptying time. [22]

These agents are licensed to be use in those with T2DM but not licensed in MASLD. A placebo-controlled phase 2 study (LEAN Trial) showed that liraglutide improved NASH and fibrosis. [28] A phase 2 study of Semaglutide in biopsy proven NASH patients demonstrated a higher efficacy for NASH resolution than placebo. [29] A recent phase 2 study of Semaglutide in NASH cirrhosis did not show any significant improvement in fibrosis or NASH resolution. The data and outcome from phase 3 trial of Semaglutide in NAFLD or MASLD are awaited. [30]

4. Thyroid hormone receptor (THR)

THR-β receptor is a nuclear receptor and a transcription factor that mediates the genomic effects of thyroid hormones.[21] Thyroid hormone receptor β (THR-β) had been shown to have effect in reduction of triglycerides and cholesterol, improving insulin sensitivity, reducing apoptosis and promoting live regeneration.[31] Resmetirom is an oral, liver-targeted, selective thyroid hormone receptor β (THR-β) agonists.

## Dyslipidaemia management

Statin is widely used in managing patients with dyslipidaemia. It should be used in those with high risk of cardiovascular disease. Statin inhibits 3-hydroy-3-methylgutarul-coenzyme A reductase, a key enzyme involved in the synthesis of cholesterol.[32] A case control study showed that statin use in patients with NAFLD or MASLD was significantly associated with a reduced risk of disease progression and significant liver fibrosis compared to those who are not on statin therapy.[32]

## Diabetes management

It is important to control the hyperglycaemia since poor glycaemic control is associated with a risk of liver disease progression in those with MASLD. There are many new agents in treatment of MASLF and patients should be opted for medications that can contribute to either weight neutral or weight loss with less risk of hypoglycaemic effect. There are no dedicated medications that are recommended in treating both T2DM and MASLD and hence, the treatment should be focused on mainly addressing glycaemic control. The summary of diabetes treatment is shown in table 5.

Metformin is considered the first line therapy for the management of T2DM given its ability to improve insulin action and lower plasma glucose level without hypoglycaemia, as well as its potential weight loss benefit. [33,34]

Thiazolinediones (TZDs) are ligands for the transcription factor PPAR-γ that plays a key role in the regulation of glucose and lipid metabolism, as well as in inflammation. [34,35] Pioglitazone and rosiglitazone had been tested in patients with NASH although the results had been mixed due to small and uncontrolled nature of study methods. [36] TZDs are associated with weight gain and at risk of heart failure and should be used carefully in some patient cohort.

**Table 5: Treatment of type 2 diabetes mellitus and their mechanism of action**

| Medications | Route of medications | Mechanism of actions | Effect on weight |
|---|---|---|---|
| Metformin | Oral | Insulin sensitizer | Weight loss/ neutral |
| Thiazolidinediones (pioglitazone) | Oral | PPAR-γ agonist | Weight gain |
| Sitagliptin/ Saxagliptin/Alogliptin/ Linagliptin | Oral | DPP-4 inhibitor | Weight neutral |
| Canagliflozin/ Dapagliflozin/ Empagliflozin | Oral | SGLT2 inhibitor: Inhibits renal glucose reabsorption | Weight loss |
| Exenatide or Byetta<br><br>Exenatide extended release (Bydureon bcise) | Injection (twice daily)<br><br>Injection (once a week) | GLP-1 receptor agonist | Weight loss |
| Liraglutide (Victoza or Saxenda) | Injection (daily) | GLP-1 receptor agonist | Weight loss |
| Dulaglutide (Trulicity) | Injection (weekly) | GLP-1 receptor agonist | Weight loss |
| Semaglutide (Ozempic) | Injection (weekly) | GLP-1 receptor agonist | Weight loss |
| Insulin | Injection | | Weight gain |

GLP-1 receptor agonists are an attractive treatment for those with NAFLD, T2DM and obesity due to its potential benefit of weight loss. Glucose-lowering by GLP-1RA is attributed to enhancement of insulin secretion, reduction of postprandial glucagon concentration, effects at the level of the central nervous system with appetite suppression and induction of weight loss, as well as improvement of insulin action in hepatocytes and adipose tissue. [34,37] Treatment with Semgalutide is currently undergoing phase 3 clinical trial for those with NASH.

Dipeptidyl peptidase-4 (DDPP-4) inhibitors are another therapeutic option for those with NAFLD and T2DM. The treatment action is primarily to block the protein DPP-4 that degrades GLP-1. [34]A study had showed that this treatment had shown improvement in postprandial plasma glucose control and reduced HbA1c by 0.6 to 0.8%. [38]

Sodium-glucose co-transporter 2 (SGLT2) inhibitors inhibit the reabsorption of glucose in the proximal renal tubular system which caused a marked reduction in the plasma glucose levels.[34] The current 3 agents used in T2DM are dapagliflozin, canagliflozin and empagliflozin. [34]

Insulin is still the option of treatment when all other agents had failed. Insulin contributes towards the weight gain and should be considered as the last option if possible.

## Conclusion

MASLD is the common presentation of abnormal liver blood tests being referred to the outpatient clinic for assessment. It is a heterogenous disease that cause a challenge in finding an effective treatment. The assessment of underlying fibrosis is important since fibrosis can progress to cirrhosis which then lead to complications of cirrhosis from portal hypertension and hepatocellular carcinoma. The most commonly used fibrosis assessment in clinical settings is using a non-invasive form of assessment either with serum markers or elastography. Liver biopsy is still the gold standard and required for enrolling patients into the clinical trials. Having a dedicated clinical service with multidisciplinary team is important in patients with MASLD because it will provide careful assessment of the underlying metabolic risk factors alongside the liver disease and stratify the risk for underlying liver fibrosis.

## Declarations

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**Ethical approval:** This is a review article. No human or animals were involved and hence, no ethical approval was required.

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