SASLT
الجمعية السعودية لأمراض وزراعة الكبد
Saudi Society for the Study of Liver Disease and Transplantation

MARCH | 2026

SASLT NEWSLETTER

15TH ISSUE | 2026

TABLE OF CONTENT

WELCOME TO THE 15TH EDITION OF THE SASLT NEWSLETTER!

Dear SASLT Community,
I am pleased to welcome you to the 15th issue of the SASLT Newsletter. This edition holds particular significance as we address a pressing challenge in our country: the increasing prevalence and epidemic of MASLD (Metabolic Associated Steatotic Liver Disease) in our region. As this health concern escalates, it profoundly affects individuals and communities, highlighting its importance as a topic for discussion and research.

In this issue, we present a unique perspective of articles, research findings, and expert opinions that examine the rising rates of MASLD, its implications, and strategies for prevention and intervention. We are honored to feature contributions from our distinguished authors who share their insights and expertise on MASLD, providing a special understanding of the epidemic and approaches for a collective response.
Additionally, we are pleased to introduce two new members to our Editorial Board: Dr. Saleh Alghsoon and Dr. Mohammed Ayoub. Their experience and knowledge will help to further enhance the mission of our newsletter.

Thank you for engaging in this important conversation. I hope you find this issue both informative and impactful as we work together to address one of the most significant health challenges of our time.

Warm regards,

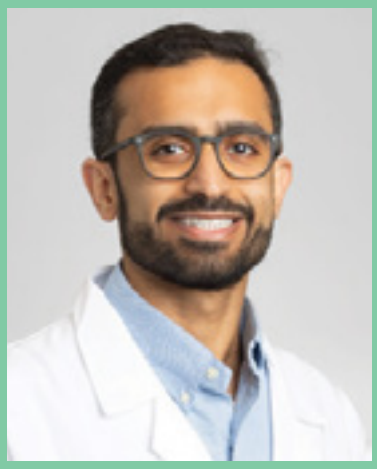
Dr. Saad Alghamdi, MD

- Editor-in-Chief, SASLT Newsletter.
- Consultant, Adult Transplant Hepatology.
- Liver & Small Bowel Health Centre Department.
- Organ Transplant Center of Excellence KFSHRC, Riyadh.

EDITORIAL BOARD

Dr. Saad Alghamdi, MD

- Editor-in-Chief, SASLT Newsletter.
- Consultant, Adult Transplant Hepatology.
- Liver & Small Bowel Health Centre Department.
- Organ Transplant Center of Excellence
- KFSHRC, Riyadh.



Dr. Saleh Alghsoon

- Assistant Professor at KSAU-HS.
- Transplant Hepatologist at MNGHA, Riyadh, Saudi Arabia.

Dr. Mohammed Ayoub

- MBBS, FRCPC, FAAP.
- Consultant Pediatric Gastroenterologist, Hepatology, and Transplant. Assistant Professor of Pediatrics, King Abdulaziz University, Rabigh, Saudi Arabia.

PREVALENCE OF MASLD IN SAUDI ARABIA

DR. FAHD ALMALKI[1]

CHRONIC LIVER CONDITIONS

are on the rise and the increase is driven mainly by the rising incidence of non-alcoholic fatty liver disease. The global incidence of NAFLD rose from 88,180 in 1990 to 172,330 in 2019, while the prevalence increased from 561,370,000 in 1990 to 1,235,700,000 in 2019, predominantly affecting young adults1. In 2023, based on expert consensus, the terminology was changed from NAFLD to MASLD to emphasize the importance of metabolic disturbance in the pathophysiology of the disease and avoid the bad connotation associated with the old terminology2. Multiple studies have confirmed the identical nature of the two conditions, natural history and future consequences of the these entities 3,4. MASLD has many risk factors including obesity, type 2 diabetes, hypertension dyslipidemia among other metabolic risk factors 5. MASLD may progress from simple hepatic steatosis (MASLD) to steatosis with hepatitis and varying degrees of fibrosis (MASH) to cirrhosis and liver cancer6. MASLD also is associated with an increased risk of mortality secondary to cardiac disease and malignancy 7.In a meta- analysis published in 2023 , the global prevalence of MASLD is around 38%8. According to an analysis of the global burden of disease date between (2010 -2021) in 2021, the global age-standardized point prevalence rate of MASLD was 15,018.1 cases per 100,000 people and the annual incidence rate was 608.5 cases per 100,000 confirming the global public health threat of MASLD9. MENA region has one of the highest global

Dr. Fahd Almalki

prevalence rates of MASLD across the globe with over 141.51 million cases of that have been progressively rising over the last decades as evidenced by the rise in prevalence from 35.42% in the period between (2008–2016) to 46.20% in the period between (2017–2020)10. In a study from Saudi Arabia , that utilized CT scan to determine the presence of MASLD in 100 adult hospitalized patients in 2012, the reported prevalence ranged between 18–54% based on the criteria used to make the diagnosis11. In a study conduced among diabetic patients in a hospital in Jazan , the prevalence of MASLD was estimated to be around 47.8%12.

In a study by Abalkhail et al which analyzed the Global Burden of Disease (GBD) data in 2019, MASLD prevalence in Saudi Arabia increased with an annual percent change (APC) of +2.43% between 2012 to 2019 and MASLD related mortality showed an increase with an APC of +1.15%. The prevalence of MASLD increased from 8.34 millions to 11.83 millions representing an increase from 28.02% to 33.11 %13 In a modeling study by AL-Swat et al , the prevalence of MASLD is projected to increase to 12.5 million cases by 2030 in Saudi Arabia associated with a concomitant increase in the burden of advanced liver disease, including cirrhosis and hepatocellular carcinoma14. In another study by AL-Swat et , a similar trend of increased prevalence of DM , hypertension , obesity and other risk factors of MASLD was observed15.All these statistics demonstrating the rapidly rising incidence / prevalence of MASLD in the kingdom highlight the growing public health threat of MASLD which should inform public health policy intended to halt and slow down it's progression. Targeted educational programs with the goal of enhancing public awareness about MASLD and it's metabolic risk factors is of utmost importance. Encouraging a healthy life style by promoting a healthy diet and physical activity will help reduce and curb the rising rates of obesity and MASLD consequently. The healthcare system should focus on early screening, diagnosis and risk stratification of at risk patients.

References:

1.Chen H, Zhan Y, Zhang J, Cheng S, Zhou Y, Chen L, et al. The global, regional, and national burden and trends of NAFLD in 204 countries and territories: An analysis from global burden of disease 2019. JMIR Public Health Surveill 2022;8:e34809.

2. Rinella ME, Lazarus JV, Ratziu V, Francque SM, Sanyal AJ, Kanwal F, et al. A multisociety Delphi consensus statement on new fatty liver disease nomenclature. J Hepatol 2023;79:1542-56

3. Younossi ZM, Paik JM, Stepanova M, Ong J, Alqahtani S, Henry L. Clinical profiles and mortality rates are similar for metabolic dysfunction associated steatotic liver disease and non alcoholic fatty liver disease. J Hepatol 2024;80:694 701.

4. Paik JM, Duong S, Zelber-Sagi S, Lazarus JV, Henry L, Younossi ZM. Food Insecurity, Low Household Income, and Low Education Level Increase the Risk of Having Metabolic Dysfunction-Associated Fatty Liver Disease Among Adolescents in the United States. Am J Gastroenterol 2024;119:1089-101. doi: 10.14309/ ajg.0000000000002749.

5. Li Q-Q, Xiong Y-T, Wang D, et al. Metabolic syndrome is associated with significant hepatic fibrosis and steatosis in patients with nonalcoholic steatohepatitis. iLIVER 2024;3:100094.

6. Feng G, Fan Y-F, Li R-X, et al. Unraveling the epidemiology of metabolic dysfunction-associated liver cancer: insights from mixed etiologies, regional variations, and gender disparities. iLIVER 2024:100113.

7. Ginès P, Serra-Burriel M, Kamath PS. Metabolic Dysfunction-Associated Steatotic Liver Disease-The New Epidemic of Chronic Liver Disease. JAMA Netw Open. 2025;8(6):e2516381. Published 2025 Jun 2. doi:10.1001/jamanetworkopen.2025.16381

8. Younossi ZM, Golabi P, Paik JM, Henry A, Van Dongen C, Henry L. The global epidemiology of nonalcoholic fatty liver disease (MASLD) and nonalcoholic steatohepatitis (NASH): A systematic review. Hepatology 2023;77:1335 47.

9. Feng G, Targher G, Byrne CD, et al. Global burden of metabolic dysfunction-associated steatotic liver disease, 2010 to 2021. JHEP Rep. 2024;7(3):101271. Published 2024 Nov 14. doi:10.1016/j.jhepr.2024.101271

10. Younossi ZM, Golabi P, Paik J, et al. Prevalence of metabolic dysfunction-associated steatotic liver disease in the Middle East and North Africa. Liver Int. 2024;44(4):1061-1070. doi:10.1111/liv.15852

11. Alshumrani GA, Shawky KA, Assiri YI, Algathradi MA, Mahfouz AA, Mostafa OA. Fatty LiverDisease among Adults in Southwestern Saudi Arabia. Med J Cairo Univ. 2013;81:205–9.

12. Elmakki E, Aqeely H, Bani I, Omer H, Solan Y, Taher A, et al. Nonalcoholic Fatty Liver Disease(NAFLD) in Saudi Patients with T2DM in Jazan Region: Prevalence and Associated Factors. BJMMR.

13. Alqahtani SA, Abaalkhail F, Alghamdi S, Bzeizi K, Al-Hamoudi WK, Paik JM, et al. The burden of metabolic dysfunction-associated steatotic liver disease and viral hepatitis in Saudi Arabia. Saudi J Gastroenterol 2024;30:310-8.

14. Alswat K, Aljumah AA, Sanai FM, Abaalkhail F, Alghamdi M, Al Hamoudi WK, et al. Nonalcoholic fatty liver disease burden-Saudi Arabia and United Arab Emirates, 2017-2030. Saudi J Gastroenterol 2018;24:211-9.

15. Alswat K, Sanai FM, Al-Hamoudi W, Ismail M, Dahlan Y, AlGhamdi HS, et al. Clinical and metabolic characteristics of non-alcoholic fatty liver disease patients in Saudi Arabia: Data from the Systematic Observatory Liver Disease (SOLID) registry. Diabetes Metab Syndr Obes 2021;14:1167-75.

Affiliation :

1.Department of Medicine , College of Medicine , Umm Al-Qura University , Makkah , Saudi Arabia

ARTICLES

MASLD TREATMENT UPDATES: WHAT IS IN THE PIPELINE FOR 2026?

DR. MOHAMMED ALSAGER

[1] Department of Gastroenterology, King Saud Medical City, Riyadh, Saudi Arabia

Metabolic dysfunction-associated steatotic liver disease (MASLD), previously known as nonalcoholic fatty liver disease (NAFLD), encompasses a spectrum from simple steatosis to metabolic dysfunction-associated steatohepatitis (MASH) and progressive fibrosis. The primary goals of MASLD management are resolution of MASH, prevention of fibrosis progression, and reduction of liver-related morbidity and mortality, while also addressing associated cardiometabolic comorbidities such as obesity, type 2 diabetes, hyperlipidemia, and hypertension1. In addition, MASLD is a major contributor to cirrhosis, hepatocellular carcinoma (HCC)2, and liver transplantation. The inflammatory phenotype, metabolic dysfunction–associated steatohepatitis (MASH), is the principal driver of fibrosis progression and liver-related morbidity. Recent years have witnessed substantial advances in disease nomenclature, risk stratification strategies, non-invasive diagnostics, and pharmacotherapy. This review will focus on novel treatments for MASLD and pipeline production in 2026.

Lifestyle Modification and Weight Loss

Lifestyle intervention remains the cornerstone of MASLD treatment, with sustained weight loss as the initial intervention to improve hepatic steatosis, inflammation, and fibrosis. Clinical evidence demonstrates that a reduction of at least 5% of body weight is necessary to decrease liver fat, 7–10% to improve steatohepatitis and fibrosis, and ≥10% for significant fibrosis regression 3,4. The most widely adopted guidelines recommend caloric restriction, increased physical activity (150–300 minutes/week of moderate or 75–150 minutes/week of vigorous intensity), and reduction of commercially produced fructose intake5. Both low-carbohydrate and low-fat hypocaloric diets are effective, but the Mediterranean diet is specifically recommended due to its favorable impact on liver fat and metabolic parameters, emphasizing fruits, vegetables, whole grains, fish, and olive oil while limiting ultra-processed foods, saturated fats, and refined sugars. Long-term adherence is critical, and tailoring dietary recommendations to individual preferences, cultural, and economic factors may enhance compliance1. The challenge with lifestyle modification and sustaining weight loss is the high rate of failure.

Pharmacologic Therapies

While lifestyle modification is foundational, pharmacotherapy is increasingly important, particularly for patients unable to achieve sufficient weight loss or those with advanced disease. Several classes of drugs are under investigation or have recently received regulatory approval:

- **GLP-1 Receptor Agonists:** The ESSENCE trial is a pivotal phase 3 randomized

Dr. Mohammed Alsager

study evaluating once-weekly semaglutide (2.4 mg) in adults with biopsy-confirmed metabolic dysfunction-associated steatohepatitis (MASH) and moderate to advanced fibrosis (F2–F3). Over 72 weeks, semaglutide significantly increased rates of improvement in liver fibrosis without worsening steatohepatitis (36.8% vs 22.4% with placebo) and of resolution of steatohepatitis without worsening fibrosis (62.9% vs 34.3% with placebo). Key secondary outcomes included combined resolution of MASH and fibrosis improvement (32.7% vs 16.1% placebo) and substantial weight loss, with an overall safety profile characterized mainly by mild-to-moderate gastrointestinal adverse events. Semaglutide is the first GLP-1 receptor agonist with phase 3 evidence and regulatory approval for MASH within MASLD. Imitations include exclusion of patients with cirrhosis and the need for longer-term outcome data from part 2 of ESSENCE to determine effects on clinical endpoints such as progression to cirrhosis or hepatocellular carcinoma 6.

• THR-β Agonists:
Resmetirom, a thyroid hormone receptor-β agonist, is FDA-approved for adults with noncirrhotic NASH (MASH) and moderate to advanced fibrosis (F2–F3), in conjunction with diet and exercise. Resmetirom targets hepatic lipid metabolism and has shown histological improvement in fibrosis and steatohepatitis in phase 3 trials[4].

• PPAR Agonists and FXR Agonists:
These agents modulate metabolic, inflammatory, and fibrotic pathways. Lanifibranor (a pan-PPAR agonist) and FXR agonists are in advanced clinical development, with evidence of histological improvement in MASLD4,7.

• SGLT2 inhibitors:
Currently, sodium–glucose cotransporter 2 inhibitors (SGLT2i) have established or emerging indications across nearly every major field of medicine. In a recently published multicentre, double blind, randomised, placebo-controlled trial, the efficacy and safety of dapagliflozin were evaluated in 154 adults with biopsy-confirmed metabolic dysfunction-associated steatohepatitis (MASH) over a 48-week period. Patients treated with dapagliflozin 10 mg once daily were significantly more likely than those receiving placebo to achieve the primary endpoint of MASH improvement without worsening of fibrosis (53% vs 30%; risk ratio 1.73, 95% CI 1.16–2.58; P=0.006).). In addition, dapagliflozin was associated with higher rates of MASH resolution without fibrosis progression and fibrosis improvement without worsening of MASH. Significant reductions were also observed in NAFLD activity score, fibrosis stage, liver stiffness, hepatic steatosis, body weight, and multiple metabolic parameters. The therapy was well tolerated, with comparable adverse event rates between the two groups. Collectively, these findings highlight dapagliflozin's capacity to improve both histological and metabolic outcomes in MASH, reinforcing its potential as a valuable therapeutic option in this population[8].

• Liver-directed therapies:
Fibroblast growth factor 21 (FGF21)

analogues—including efruxifermin, pegozafermin, and efimosfermin alfa—are engineered to overcome the short half-life and fibroblast activating protein-mediated cleavage of native FGF21, enabling extended activity and resistance to degradation. These agents act via FGFR1/2/3 and β-klotho co-receptors to enhance hepatic fatty acid oxidation, reduce de novo lipogenesis, and improve insulin sensitivity, with additional metabolic effects in adipose tissue.

Efruxifermin, a weekly IgG Fc-FGF21 fusion protein, demonstrated in the HARMONY phase 2b trial that 41% (50 mg) and 39% (28 mg) of patients with biopsy-proven MASH and F2–F3 fibrosis achieved ≥1-stage fibrosis improvement without worsening of MASH, compared to 20% with placebo; MASH resolution rates were also significantly higher9. Pegozafermin, a glycopegylated FGF21 analogue, showed in the ENLIVEN phase 2b trial that 25–44% of patients achieved fibrosis improvement and 23–37% achieved MASH resolution, with ongoing phase 3 studies (ENLIGHTEN-fibrosis [NCT06318169])10. Efimosfermin alfa, with a novel disulfide bond and IgG1 Fc fusion, offers monthly dosing and demonstrated significant reductions in hepatic fat and improvements in fibrosis biomarkers in phase 2a, with ongoing phase 3 evaluation (ZENITH-1 [NCT07221227])[10].

VK2809, a liver-specific thyroid hormone receptor-β agonist, acts by upregulating hepatic lipid metabolism and reducing steatosis. In the phase 2b VOYAGE trial, VK2809 in biopsy-proven MASH (F1–F3) resulted in significant reductions in liver fat and improvements in histologic endpoints, including MASH resolution and fibrosis improvement versus placebo[10].

Denifanstat, a fatty acid synthase inhibitor, targets de novo lipogenesis and palmitate formation. In the FASCINATE-2 phase 2b trial in MASH with F2–F3 fibrosis, denifanstat achieved ≥2-point NAS improvement without worsening of fibrosis and increased MASH resolution rates compared to placebo; however, subsequent phase 3 trials (FASCINATE-3 [NCT06594523], FASCINIT [NCT06692283]) were withdrawn, limiting further development[10,11].

ION224, a diacylglycerol O-acyltransferase-2 (DGAT2) inhibitor, reduces hepatic triglyceride synthesis and steatosis. In the phase 2 ION224-CS2 study, both 90 mg and 120 mg doses led to significant improvement in NAS and regression of fibrosis compared with placebo, supporting further investigation[12].

• Surgical therapies:
Bariatric-metabolic surgery offers a sustainable, durable intervention for substantial weight loss, with the potential to reverse hepatic inflammation, halt fibrosis progression, and reduce cardiometabolic risk. Recent high-quality evidence, including the BRAVES13 and SPLENDOR14 studies, has provided robust data supporting the role of surgical approaches in altering both histological and long-term clinical outcomes in MASLD.

The BRAVES trial was a randomized study comparing bariatric-metabolic surgery with intensive lifestyle and medical therapy in patients with obesity and biopsy-confirmed MASH. At 52 weeks, approximately 56–57% of patients undergoing surgery achieved MASH resolution without worsening fibrosis, compared with 16% in the lifestyle group. In the per-protocol analysis, nearly 70% of surgical patients met the primary endpoint, compared with 19% with lifestyle therapy. Surgery was generally safe, with no mortality and low rates of serious adverse events. Overall, bariatric surgery was markedly more effective than lifestyle intervention in achieving histological remission of MASH[13].

The SPLENDOR trial was a large observational study comparing bariatric surgery (Roux-en-Y gastric bypass or sleeve gastrectomy) with nonsurgical management in patients with MASLD without baseline cirrhosis. The study evaluated long-term clinical outcomes,

focusing on major adverse liver events—including progression to cirrhosis, hepatic decompensation, and liver-related mortality—as well as major adverse cardiovascular events. Bariatric surgery was associated with substantial reductions in liver-related outcomes (adjusted hazard ratio, 0.12) and cardiovascular events (adjusted hazard ratio, 0.30) compared with nonsurgical care. Despite these significant benefits, a small proportion of patients in the surgical group still experienced liver-related complications14. Overall, the findings support bariatric surgery as a powerful intervention for reducing both hepatic and cardiovascular risk in patients with MASLD.

• Combination therapy:

MASLD is a multifactorial disease driven by insulin resistance, excess hepatic fatty acid delivery, and increased de novo lipogenesis, leading to toxic lipid accumulation, hepatocyte injury, inflammation, and progressive fibrosis[4]. These processes are further shaped by genetic susceptibility and inflammatory signaling from adipose tissue and the gut–liver axis. Because MASLD involves overlapping metabolic, genetic, and inflammatory pathways, monotherapy often cannot address all key mechanisms[15]. Therefore, combination therapy that targets multiple drivers (metabolic dysfunction, inflammation, and fibrogenesis) is being further explored.

In a recent randomized, open-label phase II trial[16] in 108 patients with NASH and mild-to-moderate fibrosis, semaglutide 2.4 mg weekly alone versus combinations with cilofexor (30/100 mg daily) and/or firsocostat (20 mg daily) for 24 weeks. The primary endpoint was safety, and all regimens were generally well tolerated, with mostly gastrointestinal adverse events and only mild pruritus in cilofexor-treated groups. Despite similar weight loss across arms (~7–10%), combination therapy achieved greater reductions in liver fat by MRI-PDFF (about −9.8% to −11.0% vs −8.0% with semaglutide alone) and larger improvements in ALT and several non-invasive fibrosis/activity measures.

In another combination therapy study[17], a double-blind, placebo-controlled phase 2b study, 31 adults with T2D and MASH with fibrosis (F1–F3) who were already on stable GLP-1RA therapy were randomized 2:1 to add efruxifermin 50 mg weekly or placebo for 12 weeks. The primary endpoint was safety, and efruxifermin added to GLP-1RA was generally well tolerated, with mostly mild-to-moderate gastrointestinal adverse events, one discontinuation due to nausea, and no treatment-related serious adverse events. Efruxifermin produced a markedly greater reduction in hepatic fat fraction than placebo (about 65% vs 10%) and normalized liver fat (≤5%) in ~88% of patients versus 10%. It also improved multiple noninvasive markers of liver injury and fibrosis, as well as glycemic and lipid parameters, while preserving GLP-1RA–associated weight loss.

• Emerging Biotherapeutics:

Novel approaches include RNA interference, mRNA-based gene therapies, monoclonal antibodies, PROTACs, peptide-based strategies, cell-based therapies (e.g., CAR-modified immune cells, stem cells), and extracellular vesicle-based delivery systems. These are designed to enhance target specificity, optimize biodistribution, and reduce systemic exposure, addressing limitations of current therapies[7].

• **Emerging Biotherapeutics:**
Novel approaches include RNA interference, mRNA-based gene therapies, monoclonal antibodies, PROTACs, peptide-based strategies, cell-based therapies (e.g., CAR-modified immune cells, stem cells), and extracellular vesicle-based delivery systems. These are designed to enhance target specificity, optimize biodistribution, and reduce systemic exposure, addressing limitations of current therapies7.

Conclusion:

The management of MASLD and MASH has entered a transformative era. Advances in discovering novel therapies, approval of fibrosis-targeted pharmacologic agents, the availability of surgical therapy, and the potential benefit of combination therapy have redefined clinical practice. Treatment paradigms are shifting from passive observation of steatosis to proactive, stage-based intervention aimed at preventing fibrosis progression and liver-related outcomes.
Future directions will emphasize precision medicine, combination therapy, and multidisciplinary integration across hepatology, endocrinology, cardiology, and transplant services. As therapeutic options expand, early identification and structured care pathways will be critical in mitigating the global burden of metabolic liver disease.

References:
1. Tilg H, Petta S, Stefan N, Targher G. Metabolic Dysfunction–Associated Steatotic Liver Disease in Adults A Review. JAMA. 2026;335(2):163-174. doi:10.1001/jama.2025.19615
2. Rivera-esteban J, Muñoz-martínez S, Higuera M, et al. Phenotypes of Metabolic Dysfunction – Associated Steatotic Liver Disease – Associated Hepatocellular Carcinoma. Published online 2024:1774-1789.
3. Israelsen M, Francque S, Tsochatzis EA, Krag A. Steatotic liver disease. The Lancet. 2024;404(10464):1761-1778. doi:10.1016/S0140-6736(24)01811-7
4. Targher G, Valenti L, Byrne CD. Metabolic Dysfunction–Associated Steatotic Liver Disease. New England Journal of Medicine. 2025;393(7):683-698. doi:10.1056/nejmra2412865
5. Wajcman DI, Byrne CJ, Dillon JF, et al. A narrative review of lifestyle management guidelines for metabolic dysfunction-associated steatotic liver disease. Hepatology. Published online 2024. doi:10.1097/HEP.0000000000001058
6. Sanyal AJ, Newsome PN, Kliers I, et al. Phase 3 Trial of Semaglutide in Metabolic Dysfunction–Associated Steatohepatitis. New England Journal of Medicine. 2025;392(21):2089-2099. doi:10.1056/nejmoa2413258
7. Hong J, Kim YH. Cutting-edge biotherapeutics and advanced delivery strategies for the treatment of metabolic dysfunction-associated steatotic liver disease spectrum. Journal of Controlled Release. 2025;380(September 2024):433-456. doi:10.1016/j.jconrel.2025.02.008
8. Lin J, Huang Y, Xu B, et al. Effect of dapagliflozin on metabolic dysfunction-associated steatohepatitis: Multicentre, double blind, randomised, placebo controlled trial. Bmj. 2025;389:1-10. doi:10.1136/bmj-2024-083735
9. Noureddin M, Rinella ME, Chalasani NP, et al. Efruxifermin in Compensated Liver Cirrhosis Caused by MASH. New England Journal of Medicine. 2025;392(24):2413-2424. doi:10.1056/nejmoa2502242
10. Li W, Alazawi W, Loomba R. Current and emerging therapeutic landscape for metabolic dysfunction-associated steatohepatitis. Lancet Gastroenterol Hepatol. 2026;11(2):150-162. doi:10.1016/S2468-1253(25)00260-2
11. Loomba R, Bedossa P, Grimmer K, et al. Denifanstat for the treatment of metabolic dysfunction-associated steatohepatitis: a multicentre, double-blind, randomised, placebo-controlled, phase 2b trial. Lancet Gastroenterol Hepatol. 2024;9(12):1090-1100. doi:10.1016/S2468-1253(24)00246-2
12. Loomba R, Morgan E, Yousefi K, et al. metabolic dysfunction-associated steatohepatitis (ION224-CS2): results of a 51-week , multicentre ,. Published online 2025:821-831.
13. Verrastro O, Panunzi S, Castagneto-Gissey L, et al. Bariatric–metabolic surgery versus lifestyle intervention plus best medical care in non-alcoholic steatohepatitis (BRAVES): a multicentre, open-label, randomised trial. The Lancet. 2023;401(10390):1786-1797. doi:10.1016/S0140-6736(23)00634-7
14. Aminian A, Al-Kurd A, Wilson R, et al. Association of Bariatric Surgery with Major Adverse Liver and Cardiovascular Outcomes in Patients with Biopsy-Proven Nonalcoholic Steatohepatitis. JAMA - Journal of the American Medical Association. 2021;326(20):2031-2042. doi:10.1001/jama.2021.19569
15. Stefan N, Yki-Järvinen H, Neuschwander-Tetri BA. Metabolic dysfunction-associated steatotic liver disease: heterogeneous pathomechanisms and effectiveness of metabolism-based treatment. Lancet Diabetes Endocrinol. 2025;13(2):134-148. doi:10.1016/S2213-8587(24)00318-8
16. Alkhouri N, Herring R, Kabler H, et al. Safety and efficacy of combination therapy with semaglutide, cilofexor and firsocostat in patients with non-alcoholic steatohepatitis: A randomised, open-label phase II trial. J Hepatol. 2022;77(3):607-618. doi:10.1016/j.jhep.2022.04.003
17. Harrison SA, Frias JP, Lucas KJ, et al. Safety and Efficacy of Efruxifermin in Combination With a GLP-1 Receptor Agonist in Patients With NASH/MASH and Type 2 Diabetes in a Randomized Phase 2 Study. Clinical Gastroenterology and Hepatology. 2025;23(1):103-113. doi:10.1016/j.cgh.2024.02.022.

ARTICLES

MASLD IN CHILDREN: A PRACTICAL GUIDE

DR. MUHANAD ALRUWAITHI
AND **DR. MOHAMMED D. AYOUB**

Dr. Muhanad Alruwaithi

• Pediatric Gastroenterology and Hepatology Consultant, King Fahad Medical City, Ministry of Health, Riyadh, Saudi Arabia.

Dr. Mohammed D. Ayoub

• Pediatric Gastroenterology and Hepatology Consultant, Assistant Professor of Pediatrics, King Abdulaziz University, Rabigh, Saudi Arabia

1. Background

Metabolic dysfunction-associated steatotic liver disease (MASLD) is now the most common chronic liver disease in children. The global prevalence is estimated at around 7.6% among all children. It rises substantially in children with obesity or type 2 diabetes, though exact estimates vary depending on how steatosis is confirmed and the population under study. The disease is largely silent. Most children are asymptomatic and many are never investigated. The 2025 AASLD Practice Statement was made to fill that gap. Regionally, Saudi Arabia has one of the highest MASLD burdens in the Middle East. Adult prevalence in Saudi Arabia and the UAE rose from around 28% in 2012 to 33% in 2019. The broader Middle East & North Africa adult estimate sits at 27–30%. Dedicated pediatric data from Saudi Arabia are limited. The only published local pediatric cohort by Mujili et al confirmed that obesity is the main driver, with dyslipidemia and elevated aminotransferases being common findings. A subgroup of affected children had a normal BMI, and as such, a healthy weight does not rule MASLD out.

2. The 2023 Name Change: What It Means in Practice

The renaming from NAFLD to MASLD in 2023 reflects an important shift in how the disease is defined.
First, the diagnostic criteria no longer require elevated liver enzymes. Under NAFLD, a child with hepatic steatosis on imaging but a normal ALT often did not meet diagnostic criteria and was not labelled or followed. This is probably the most underappreciated implication of the name change. It substantially expands the population of children who warrant evaluation.
Second, MASLD may overlap with other liver conditions. The terms MASLD and MASH describe fatty liver disease linked to cardiometabolic risk factors in children, excluding monogenic inborn errors of metabolism. However, MASLD can occur alongside other diagnoses. It may coexist with autoimmune liver disease and Wilson disease, and these possibilities should be carefully considered and ruled out before assigning the MASLD label, especially when the clinical features are atypical.

3. What Drives Disease in Children

Insulin resistance and hepatic lipid dysregulation are the central mechanisms. Around 60% of hepatic fat derives from excess free fatty acid flux from adipose tissue, with de novo lipogenesis contributing a further 30%. The composition of the diet, and particularly dietary fructose, drives hepatic fat through a pathway that is largely independent of total caloric intake.

Genetic susceptibility plays a significant role. The PNPLA3 rs738409 G allele is the most important pediatric risk variant associated with steatosis, lobular inflammation, and fibrosis. TM6SF2 and GCKR variants also increase risk, while HSD17B13 appears protective against progression to steatohepatitis. Genetic background helps explain the variation in disease burden across populations.

Early-life programming is underappreciated. Maternal obesity, gestational diabetes, preterm birth, and in utero nutritional stress drive epigenetic changes that shape hepatic lipid metabolism before birth. These perinatal factors interact with genetic susceptibility to amplify disease risk, directly relevant to Saudi Arabia, given the regional burden of gestational diabetes.

Gut dysbiosis and environmental exposures are emerging contributors. Dysbiotic microbiota profiles and endocrine-disrupting chemicals (bisphenols, phthalates, PFAS) have been associated with MASLD in children. These are not yet actionable in routine clinical practice but help explain interindividual variation in disease severity.

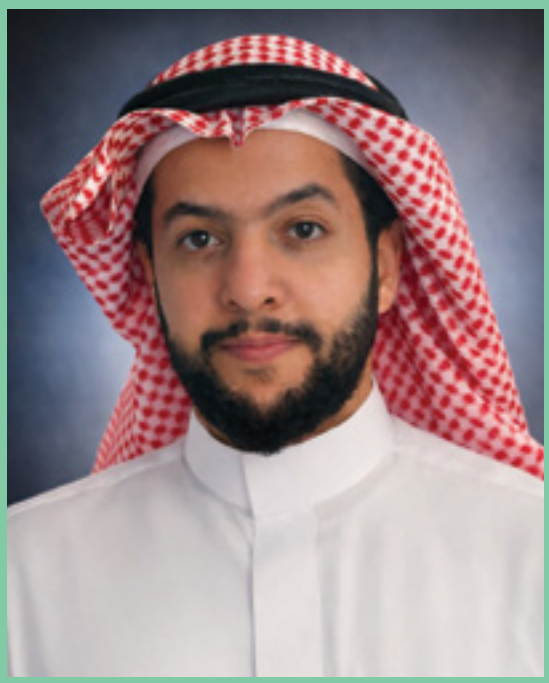
4. Screening

Who to screen: children aged 10 and older with obesity (BMI ≥ 95th percentile for sex and age) , or those with overweight (BMI ≥ 85% for sex and age) plus at least one cardiometabolic risk factor or a family history of MASLD. Screen annually if the risk remains. For children under 10, screen if they are being evaluated for obesity or significant metabolic risk factors.

How to screen:

serum ALT using sex-specific thresholds: >26 U/L for boys and >22 U/L for girls. These limits are lower than standard reference ranges (usually 40–56 U/L), which are based on adult data. The AASLD 2025 thresholds offer over 80–85% sensitivity for detecting histologic MASLD in children. Relying on the standard lab range as a cutoff may lead to missed diagnoses.

If ALT levels are elevated in asymptomatic healthy individuals, repeat the test in three months. Transient increases often occur due to viral illnesses. If the levels remain elevated after three months, further investigation is necessary.



Dr. Muhanad Alruwaithi

5. Making the Diagnosis

Diagnosis requires two points: hepatic steatosis on imaging or biopsy and at least one of five cardiometabolic criteria. Both must be present. Neither alone is enough. The most common practical error is assuming that elevated ALT plus obesity equals MASLD. It does not. Steatosis must be directly confirmed by imaging or biopsy. Conversely, a child with confirmed steatosis but no cardiometabolic criterion, does not meet criteria for the diagnosis of MASLD; an alternative cause should be considered. For steatosis assessment, ultrasound can confirm steatosis qualitatively and is sufficient for making the diagnosis. It cannot grade severity reliably. Only MRI-PDFF is validated in children for steatosis quantification. CAP (controlled attenuation parameter) by transient elastography is not validated for pediatric use and should not be the primary tool for steatosis assessment in children.

The five cardiometabolic criteria should use pediatric-specific thresholds:

Criterion	Pediatric Threshold	Key Note
Body Habitus	BMI ≥85th percentile for age and sex, OR waist circumference ≥95th percentile.	Not the adult BMI of 25. A child can look "normal" by adult standards and still qualify.
Blood Glucose	Fasting glucose ≥100 mg/dL, prediabetes, or type 2 diabetes	Similar to the adult threshold
Blood Pressure	Under 13: ≥95th percentile for age, sex, and height / Age 13+: ≥130/85 mmHg	Using the adult cutoff in younger children will miss hypertension
Triglycerides	Under 10: ≥100 mg/dL / Age 10+: ≥130 mg/dL	Lower cutoff in younger children reflects their naturally lower normal range
HDL Cholesterol	≤40 mg/dL regardless of sex	Adults use sex-specific values. Children use one threshold for all

For persistently elevated ALT, the workup should be comprehensive and systematic. Viral hepatitis serologies, including HBsAg, anti-HBs, anti-HCV antibody, and Hepatitis A and E, depending on clinical context. Autoimmune markers, including ANA, SMA, anti-LKM, and IgG levels, must be checked to exclude autoimmune hepatitis before the diagnosis of MASLD is confirmed. The broader screen should include Wilson disease workup with serum ceruloplasmin and 24-hour urine copper; iron studies; celiac disease antibodies (tTG-IgA and total serum IgA); Serum alpha-1 antitrypsin; and thyroid function.

Medications review is of paramount importance as corticosteroids, anticonvulsants, antipsychotics, antidepressants, methotrexate, and doxycycline, all have effects on hepatic lipid metabolism and cause drug-induced liver injury and can ultimately, quietly, confound the picture.

When the presentation is atypical, such as onset of disease ≤ 3 years of age, rapidly progressive or acute disease, absence of obesity, or neurological and multi-organ involvement, inborn errors of metabolism move to the top of the differential diagnosis. Additionally, glycogen storage diseases, fatty acid oxidation disorders,

lipodystrophy, and lysosomal acid lipase deficiency should all be considered in this setting.

6. How Children Differ from Adults

The histological pattern in children is fundamentally different from that of adults. Adults show zone 3 injury, centrilobular steatosis, hepatocyte ballooning, and perisinusoidal fibrosis. Children predominantly show zone 1 injury periportal steatosis, portal inflammation, and portal or periportal fibrosis with ballooning being minimal or absent. This distinction has direct practical consequences. The standard NAFLD Activity Score (NAS) was designed around adult zone 3 histology and ballooning of weights. In a child with zone 1 disease and no ballooning, NAS will underestimate severity. A child with significant portal inflammation and early fibrosis can score low and appear to have mild disease when they do not. Pathologists reviewing pediatric liver biopsies must recognise this pattern and report accordingly.

Beyond histology, the AASLD statement highlights several other pediatric distinctions. Cardiometabolic thresholds differ from adult cutoffs (as above). Fibrosis staging tools and non-invasive markers validated in adults have not been adequately studied in children and cannot be applied.

When to do a biopsy in childhood MASLD: consider when ALT is persistently ≥2× the upper limit of normal using sex-specific thresholds (≥52 U/L in boys, ≥44 U/L in girls); when advanced fibrosis or MASH is clinically suspected; when autoimmune hepatitis cannot be excluded; when the result will change management; or when the presentation is atypical. Biopsy is generally well tolerated in children but carries procedural risk and requires appropriate patient selection.

7. Comorbidities and Follow-Up

MASLD rarely comes alone. Prediabetes is present in 20–23% at diagnosis, with type 2 diabetes developing in around 11% within four years. Annual screening is recommended for all and vice versa. Dyslipidemia affects roughly half, and hypertension up to a third of MASLD patients. Obstructive sleep apnea is common and underevaluated; depression runs at twice the general pediatric rate. Reduced bone mineral density and Polycystic Ovary Syndrome in post-pubertal girls round out a comorbidity profile that extends well beyond the liver. These are not incidental findings. MASLD diagnosis is an entry point into a full metabolic assessment, not simply a referral to hepatology.
MASLD does not reliably resolve with puberty. Long-term cohort data show elevated rates of premature mortality, liver-related disease, and cardiometabolic events compared with the general population. Histologically, around a third of children in clinical trials show fibrosis progression within 18 months. Simple steatosis at diagnosis does not mean the disease will stay simple. Structured follow-up matters.

8. Management

Lifestyle-based Interventions

Diet and exercise remain a core component of the treatment approach for childhood MASLD. As children are in a state of continued growth (in both weight and height), reductions in BMI z-score, rather than absolute BMI values are more reliable to assess treatment response. In a retrospective pediatric MASLD study with over 700. children, a BMI z-score reduction > 0.25 was associated with significant change in aminotransferase levels. Diet is better supervised by an experienced dietician. Most interventions are aimed

at reducing added sugars, which in isolation have shown improved liver steatosis by 6%. Mediterranean diets may confer long-term benefit for cardiovascular risk reduction. Although the effect of screen time is not properly studied in MASLD, it is generally recommended to limit screen time to less than 2 hours per day. Medications that are known to cause drug-induced liver injury (particularly steatosis-type injury) should be avoided. Intensive exercise programs, such as weight management camps has been shown to reduce BMI z-score by up to –0.87, with reductions in ALT by more than 30 points if sustained for at least a year. All children are encouraged to engage in age-appropriate moderate to vigorous physical activity. Overall, greater intensity and sustainability of diet and exercise is associated with greater improvement in hepatic steatosis

Pharmacological Interventions

At the time of writing, no medications are FDA or SFDA approved for treating pediatric isolated MASLD/MASH (age < 18 years). Numerous supplements and medications have been studied, such as vitamin E owning for its antioxidant effects and metformin that helps with insulin resistance. Overall, five randomized double-blind placebo-controlled studied using vitamin E in children, four of these studies showed no major improvement in ALT.
The TONIC trial included 173 children with MASLD/MASH who were randomized to receive high-dose vitamin E, metformin, or placebo for 2 years and included end-of-treatment biopsies. It revealed that neither vitamin E nor metformin was superior to placebo in reducing ALT levels. However, vitamin E and metformin-treated patients with NASH did show improvement in histological NASH score, but that was influenced by improved changes in ballooning degeneration following therapy. Systematic reviews since then however, had shown no significant improvements with vitamin E over placebo regardless of dose or duration. No further studies have investigated metformin further in the

Dr. Mohammed D. Ayoub

context of MASLD. Therefore, both metformin and vitamin E are not considered to provide benefit as per the 2025 AASLD pediatric MASLD practice guidelines. Other medications/supplements that have not been proven to be effective for widespread use include vitamin D, Omega-3-fatty acids, probiotics, and losartan.

In April 2025, semaglutide, a glucagon-like-peptide-1 (GLP1) agonist has been approved in adults (aged ≥ 18 years) with MASH and moderate to advanced fibrosis. This is unfortunately not the case for children. Therapy with GLP1 agonists is not approved for MASLD/MASH but they can be considered for use in children aged ≥ 10 with type 2 diabetes mellitus, and children aged ≥ 12 years with obesity.

GLP1 agonists approved in diabetes include subcutaneous liraglutide and extended-release exenatide. Doses are typically lower than ones used for obesity. Not much is known about liver outcomes in

children undergoing therapy with GLP1 agonists in type 2 DM as they were not reported in GLP1 trials at large. However, they show major promise in smaller studies, with reportable 70% improvement in ALT in 9 children with MASLD and type 2 DM treated with GLP1 agonists for approximately 1 year.

In clinical practice, GLP-1 agonists are frequently prescribed to treat children and adolescents with obesity and comorbid MASLD. In this context, a landmark trial in adolescents aged 12-18 years – a double-blind, randomized, placebo-controlled study published in New England Journal of Medicine in 2022 – reported that semaglutide, administered at maximum weekly doses of 2.4mg produced an 18% reduction in ALT levels by week 68, compared with a 1% reduction in the placebo group.

In summary, the use of GLP-1 agonists in adolescents with obesity is expected to rise in our country. However, because safety and efficacy date in pediatric MASLD remain limited in clinical practice, liver-related outcomes should be monitored closely, as adult findings do not translate to children and adolescents. Nonetheless, emerging clinical experiences among pediatric hepatologists suggests that the effects of these medications on the pediatric steatotic liver are promising. Finally, as endocrinologists are more experienced with these novel agents, patients with MASLD and coexisting obesity or type 2 diabetes mellitus should be referred to an experienced endocrinologist/obesity clinician. This ensures informed pharmacological decision-making and allows treatment to be tailored according to the approved indications of the SFDA

Surgical Interventions

Bariatric surgery should be considered for children and adolescents (aged ≥ 12 years) with severe obesity (BMI of ≥ 120% of the 95th percentile) and comorbidities such as MASLD. It should be particularly considered in adolescent with MASH with advanced fibrosis who have with failed lifestyle intervention and when available, pharmacotherapy. Surgical decision-making should be guided by a multidisciplinary team that includes pediatric hepatologists, surgeons, endocrinologists, and mental health professionals. Safe and effective options include Roux-en-Y gastric bypass and sleeve gastrectomy, both of which have demonstrated BMI reductions of up to 27% and improvement in cardiometabolic risk factors in adolescents. In addition, histological improvement in MASH and liver fibrosis had been observed in 40 adolescents who underwent bariatric surgery compared with those receiving lifestyle interventions alone. Importantly, the safety of bariatric surgery in MASH-related cirrhosis in adolescents remain uncertain. All potential surgical candidates should undergo a comprehensive pre-operative evaluation for cirrhosis and signs of portal hypertension by pediatric hepatologists.

9. Conclusions

MASLD in the new “liver epidemic” in the 21st century particularly in the Middle East and Saudi Arabia. Children and adolescent meeting cardiometabolic criteria should be screened for MASLD with serum ALT and persistently elevated, should undergo imaging or liver biopsy to confirm MASLD and assess for MASH if clinically suspected. Treatment requires a multidisciplinary team and follows a stepwise multi-modal approach, with optimism for use of novel pharmacological agents such as GLP-1 agonists in eligible adolescents.

References:

1. Xanthakos SA et al. AASLD Practice Statement: MASLD in children. Hepatology. 2025;82:1352–1394.
2. Rinella ME et al. Multisociety Delphi consensus: new fatty liver disease nomenclature. Hepatology. 2023;78:1966–1986.
3. Schwimmer JB et al. Long-term mortality in 1,096 children with MASLD (LIVERS cohort). Hepatology. 2025.
4. Lavine JE et al. (TONIC Trial). Vitamin E or metformin for NAFLD in children. JAMA. 2011;305:1659–1668.
5. Kalveram L et al. Noninvasive fibrosis scores in paediatric fatty liver disease. JPGN. 2024;78:27–35.
6. Younossi ZM et al. NAFLD/MASLD in MENA. Liver Int. 2024;44:1061–1070.
7. Mujlli G et al. NAFLD in children in Saudi Arabia. Cureus. 2023;15(10):e46380.
8. AlQahtani SA et al. Burden of MASLD in Saudi Arabia (GBD 2012–2019). Saudi J Gastroenterol. 2024;30(5).
9. Alswat KA et al. NAFLD burden in Saudi Arabia and UAE. Saudi J Gastroenterol. 2018;24:211–219.
10. Hartmann P et al. Call to action: pediatric MASLD. Hepatology. 2025.
11. Rinella ME et al. AASLD Practice Guidance on the clinical assessment and management of nonalcoholic fatty liver disease. Hepatology. 2023;77:1797–835.
12. Khurana T et al. Clinically meaningful body mass index change impacts pediatric nonalcoholic fatty liver disease. J Pediatr. 2022;250:61–66.e1.
13. Nobili V et al. Lifestyle intervention and antioxidant therapy in children with nonalcoholic fatty liver disease: A randomized, controlled trial. Hepatology. 2008;48:119–28.
14. Chan DFY et al. Dietitian-led lifestyle modification programme for obese Chinese adolescents with non alcoholic fatty liver disease: A randomized controlled study. Int J Obes (Lond). 2018;42: 1680–90.
15. Schwimmer JB et al. Effect of a low free sugar diet vs usual diet on nonalcoholic fatty liver disease in adolescent boys: A randomized clinical trial. JAMA. 2019;321:256–65.
16. Vos MB et al. NASPGHAN Clinical Practice Guideline for the diagnosis and treatment of nonalcoholic fatty liver disease in children: Recommendations from the Expert Committee on NAFLD (ECON) and the North American Society of Pediatric Gastroenterology, Hepatology and Nutrition (NASPGHAN). J Pediatr Gastroenterol Nutr. 2017;64:319–34.
17. Zohrer E et al. Efficacy of docosahexaenoic acid-choline-vitamin E in paediatric NASH: A randomized controlled clinical trial. Appl Physiol Nutr Metab. 2017;42:948–54.
18. Sarkhy AA et al. Does vitamin E improve the outcomes of pediatric nonalcoholic fatty liver disease? A systematic review and meta-analysis. Saudi J Gastroenterol. 2014;20:143–53.
19. Choi E et al. Glucagon-like peptide-1 receptor agonists—A potential new medication for pediatric metabolic-dysfunctionassociated steatotic liver disease (MASLD). Children (Basel). 2024;11:275
20. Weghuber D et al. Once-weekly semaglutide in adolescents with obesity. N Engl J Med. 2022;387:2245–57.
21. Alqahtani AR et al. Laparoscopic sleeve gastrectomy in 108 obese children and adolescents aged 5 to 21 years. Ann Surg. 2012;256:266–73
22. Manco M, Mosca A, De Peppo F, et al. The Benefit of Sleeve Gastrectomy in Obese Adolescents on Nonalcoholic Steatohepatitis and Hepatic Fibrosis. J Pediatr. 2017;180:31-37.e2.
23. Hampl SE, Hassink SG, Skinner AC, et al. Clinical Practice Guideline for the Evaluation and Treatment of Children and Adolescents With Obesity. Pediatrics. 2023;151(2):e2022060640

SCIENTIFIC ADVANCES IN MASLD/MASH: SCREENING ALGORITHMS AND MULTIDISCIPLINARY CARE PATHWAYS

Moderator

Dr. Nasser Al-Masri

- Consultant Transplant Hepatologist, Division Head, Adult Transplant
- Hepatology -Prince Sultan Military Medical City – Riyadh
- Assistant Professor in the College of Medicine at Al Faisal University in Riyadh
- Board member, Saudi Society for the Study of Liver Disease and Transplantation.

Speaker

Dr. Adel Al Qutub

- Consultant Gastroenterology and Transplant Hepatology. Director of Patient Safety & Risk Management Administration at King Fahad
- Medical City, Riyadh, Saudi Arabia

IF YOU MISSED THE LIVE WEBINAR, WE RECORDED THE EXCITING TALK FOR YOU!

ACCESS THE LINK TO THE WEBINAR

CLICK TO WATCH

SPONSORED

novo nordisk®

UNDERSTANDING MASH

Metabolic Dysfunction-Associated Steatohepatitis
Formerly: Non-Alcoholic SteatoHepatitis (NASH)

25% Global MASLD prevalence	**5%** Progress to MASH	**80%** MASH cirrhosis within 5–10 years	**#1** Fastest-growing cause of liver transplant

WHAT IS MASH?

MASH is the inflammatory and fibrosing form of Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD). Excess hepatic fat triggers inflammation, hepatocyte injury and progressive fibrosis that can advance to cirrhosis, liver failure and hepatocellular carcinoma (HCC). The 2023 Delphi consensus renamed NAFLD/NASH to MASLD/MASH — emphasising metabolic origin and reducing patient stigma

NEW NOMENCLATURE 2023

NAFLD	MASLD
NASH	MASH
Cryptogenic cirrhosis	MASH-Cirrhosis

DISEASE PROGRESSION SPECTRUM

Steatosis (MASLD)	Steatohepatitis (MASH)	Fibrosis F1–F3	Cirrhosis F4	HCC / Liver Failure

KEY RISK FACTORS

Type 2 Diabetes Obesity (BMI > 30) Metabolic Syndrome

Dyslipidaemia Hypertension Hypothyroidism / PCOS

Genetic variants (PNPLA3)

CLINICAL PRESENTATION

Often asymptomatic until advanced stages
Elevated ALT / AST (may be normal in cirrhosis)
Right upper quadrant discomfort or fatigue
Hepatomegaly on physical examination
Features of metabolic syndrome
Signs of portal hypertension (advanced disease)

WHO SHOULD BE SCREENED?

Screen all patients with Type 2 Diabetes, obesity (BMI > 30), or > 2 metabolic risk factors. Use FIB-4 score as first-line non-invasive test. FibroScan (LSM/VCTE) for intermediate-risk patients. Liver biopsy remains the gold standard for definitive MASH diagnosis and staging.

MANAGEMENT APPROACH

Lifestyle Modification	Pharmaco-therapy	Advanced Disease
>7–10% weight loss reduces steatohepatitis and fibrosis. Mediterranean diet preferred. 150 min/week moderate aerobic exercise	GLP-1 RAs (semaglutide) and SGLT-2 inhibitors show benefit. Resmetirom targets hepatic THRβ. Pioglitazone for T2DM.	HCC surveillance every 6 months in cirrhosis. Liver transplantation for decompensated MASH-cirrhosis with excellent outcomes

"MASH is a silent epidemic — early metabolic risk assessment and fibrosis staging can prevent end-stage liver disease."

References:
AASLD 2023 MASH Guidance · EASL CPG 2021 · Rinella ME et al. Hepatology 2023 (Delphi Consensus) · Loomba R et al. NEJM 2024

SPONSOR

SASLT

الجمعية السعودية لأمراض وزراعة الكبد

Saudi Society for the Study of Liver Disease and Transplantation

SAUDI LIVER MEETING (SLM) 2026

SAVE THE DATE | 8-10 OCT 2026

SASLT

الجمعية السعودية لأمراض وزراعة الكبد
Saudi Society for the Study of Liver Disease and Transplantation

SASLT NEWSLETTER

15TH ISSUE | 2026

MARCH | 2026