METABOLIC DYSFUNCTION–ASSOCIATED STEATOTIC LIVER DISEASE: EVIDENCE BASED APPROACH

M. GOLAM AZAM[1]

Metabolic dysfunction–associated steatotic liver disease (MASLD) is the most prevalent chronic liver disease globally, driven by obesity, insulin resistance, and metabolic syndrome. Its progressive form, metabolic dysfunction–associated steatohepatitis (MASH), can lead to fibrosis, cirrhosis, and hepa-tocellular carcinoma. Evidence-based management prioritizes lifestyle interventions, including sustained weight loss of ≥7–10%, dietary modification, and increased physical activity, which remain the cornerstone of therapy and can significantly improve histological outcomes. Pharmacologic therapy is indicated in patients with biopsy-proven MASH and fibrosis or those at high risk of disease progression. Resmetirom, a selective thyroid hormone receptor-â (THR-â) agonist, represents a major advancement as the first FDA-approved therapy (2024) for noncirrhotic MASH with fibrosis. It enhances hepatic lipid metabolism, reduces intrahepatic triglyceride accumulation, and improves atherogenic lipid profiles. The phase 3 MAESTRO-NASH trial demonstrated that resmetirom significantly increased rates of MASH resolution (approximately 25–30%) and fibrosis improvement (≥1 stage in ~24–26%) compared to placebo. Additionally, it reduced liver fat as measured by MRI-PDFF and improved serum lipid parameters, offering both hepatic and cardiometabolic benefits. Adverse effects are generally mild, with diarrhea and nausea being the most commonly reported. Emerging pharmacotherapies target multiple pathogenic pathways in MASLD. Glucagon-like peptide-1 (GLP-1) receptor agonists, such as semaglutide, have demonstrated significant weight reduction and high rates of MASH resolution, particularly in patients with type 2 diabetes. Peroxisome proliferator-activated receptor (PPAR) agonists, including lanifibranor, show promise due to combined metabolic, anti-inflammatory, and antifibrotic effects. Farnesoid X receptor (FXR) agonists and fibroblast growth factor (FGF) analogues (e.g., FGF19 and FGF21 mimetics) are also under investigation, targeting bile acid signaling and metabolic regulation. Additional approaches include anti-inflammatory agents, antifibrotic drugs, and therapies targeting the gut–liver axis. Current evidence supports a personalized treatment strategy based on disease severity and metabolic comorbidities. Resmetirom may be particularly beneficial in patients with dyslipidemia and fibrosis, while GLP-1 receptor agonists are preferred in individuals with obesity and diabetes. Combination therapy targeting multiple mechanisms is likely to represent the future of MASLD management. In conclusion, while lifestyle modification remains foundational, resmetirom marks a paradigm shift as the first approved targeted therapy for MASH. Ongoing development of emerging agents and combination regimens is expected to further improve clinical outcomes in MASLD.

Keywords: Metabolic dysfunction–associated steatotic liver disease, MASLD, Metabolic dysfunction–associated steatohepatitis, MASH, Resmetirom therapy

Date of submission: *11.04.2026*

Date of acceptance: *29.04.2026*

DOI: *https://doi.org/10.3329/bjm.v37i20.89589*

Citation: *Azam MG. Metabolic dysfunction–associated steatotic liver disease: Evidence based approach. Bangladesh J Medicine 2026; Vol. 37, No. 2(1): pp. 191-192.*

Affiliation:

1. Professor, Department of Gastrointestinal, Hepatobiliary and Pancreatic Disorders, BIRDEM, Dhaka, Bangladesh. E-mail: *drgolamazam@gmail.com*