Research Article **ISSN 2639-944X**

Journal of Medical - Clinical Research & Reviews

Metabolic Dysfunction–Associated Steatotic Liver Disease in Children and Adolescents

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Received: 16 Mar 2026; **Accepted:** 19 Apr 2026; **Published:** 27 Apr 2026

Citation: Des Bharti, Saanvi Sharma. Metabolic Dysfunction–Associated Steatotic Liver Disease in Children and Adolescents. J Med - Clin Res & Rev. 2026; 10(4): 1-3.

ABSTRACT

Metabolic dysfunction–associated steatotic liver disease (MASLD), previously known as nonalcoholic fatty liver disease (NAFLD), has emerged as the most common chronic liver disease affecting children and adolescents worldwide. The rising prevalence of childhood obesity, sedentary lifestyles, and poor dietary habits has contributed significantly to the growing burden of this disease. MASLD represents a spectrum of liver pathology ranging from simple hepatic steatosis to metabolic dysfunction–associated steatohepatitis (MASH), fibrosis, cirrhosis, and, in rare cases, hepatocellular carcinoma [1-4].

Because many children remain asymptomatic during early stages, the disease often goes undetected until advanced liver damage develops. Therefore, early recognition and intervention are essential to prevent long-term complications. Histologic diagnosis of MASLD typically requires at least 5% macrovesicular steatosis with lobular inflammation and hepatocyte injury characterized by ballooning degeneration [1,5].

Keywords

Metabolic dysfunction–associated steatotic liver disease, MASLD, Pediatric fatty liver disease, Childhood obesity, Metabolic dysfunction–associated steatohepatitis, MASH, Pediatric liver disease, Insulin resistance, Pediatric obesity, Nonalcoholic fatty liver disease.

Introduction

Metabolic dysfunction–associated steatotic liver disease in children and adolescents has become a major global health concern. Over the past two decades, the prevalence of pediatric MASLD has increased dramatically in parallel with the global obesity epidemic. Current estimates indicate that approximately 7% to 10% of children in the general population are affected, while prevalence increases to 30% to 40% among children with obesity [2,6]. The disease is more common in males and adolescents, although recent studies suggest increasing incidence among younger children and even preschool populations [3,7].

Risk factors for pediatric MASLD include obesity, insulin resistance, dyslipidemia, type 2 diabetes mellitus, sedentary lifestyle, and genetic predisposition. In addition, diets high in fructose, refined carbohydrates, processed foods, and saturated fats have been strongly associated with the development of MASLD [4,8]. Environmental factors, including reduced physical activity and increased screen time, further contribute to metabolic dysfunction and hepatic steatosis.

MASLD represents a broad spectrum of liver disease. The earliest stage involves simple hepatic steatosis characterized by accumulation of fat in hepatocytes. Although simple steatosis may remain stable, it can progress to metabolic dysfunction–associated steatohepatitis, which includes inflammation and hepatocyte injury. MASH may further progress to fibrosis, cirrhosis, and end-stage liver disease. Fibrosis stage remains the most important predictor of long-term outcomes and disease progression in pediatric populations [5,9]. Although rare, children with advanced disease may eventually require liver transplantation.

Subcategories

The recent reclassification of fatty liver disease introduced the term MASLD and associated subcategories that better reflect metabolic dysfunction as the primary disease driver. These include metabolic dysfunction–associated steatotic liver disease, metabolic dysfunction–associated steatohepatitis, metabolic dysfunction-associated alcohol-related liver disease (MetALD), and cryptogenic steatotic liver disease [1,10]. This updated classification improves diagnostic clarity and supports more individualized patient management.

Pathophysiology

The pathophysiology of pediatric MASLD is complex and multifactorial. The most widely accepted mechanism is the "multiple-hit" hypothesis, which includes insulin resistance, oxidative stress, inflammatory cytokines, mitochondrial dysfunction, and gut microbiome alterations [3,11]. Insulin resistance plays a central role by increasing lipolysis and free fatty acid delivery to the liver. This accumulation of lipids leads to hepatocyte injury, inflammation, and progressive fibrosis.

Recent research highlights the importance of gut microbiota in disease development. Alterations in intestinal permeability and microbial composition may contribute to hepatic inflammation through endotoxin-mediated immune activation [12]. Genetic susceptibility also plays an important role. Variants in genes such as PNPLA3 and TM6SF2 have been associated with increased disease severity and fibrosis in pediatric populations [7,13].

Clinical Presentation

Children with MASLD are frequently asymptomatic, making early diagnosis challenging. When present, symptoms may include fatigue, vague abdominal discomfort, or hepatomegaly detected during routine examination. Some children may exhibit features of metabolic syndrome, including central obesity, hypertension, insulin resistance, and dyslipidemia [4,14].

Laboratory abnormalities typically include mild elevation of alanine aminotransferase and aspartate aminotransferase. However, normal liver enzymes do not exclude MASLD, and disease severity does not always correlate with aminotransferase levels [2,15]. Therefore, additional evaluation including metabolic risk assessment is often necessary.

Diagnosis

Imaging studies play an important role in the diagnosis of MASLD. Ultrasound remains the most widely used initial screening modality because of its availability and low cost. However, ultrasound sensitivity is limited, particularly in early disease [6,16]. Magnetic resonance imaging and transient elastography provide more accurate assessment of liver fat and fibrosis and are increasingly utilized in pediatric populations [9,17].

Despite advances in imaging, liver biopsy remains the gold standard for diagnosis and staging. Histologic findings include macrovesicular steatosis, lobular inflammation, hepatocyte ballooning, and fibrosis of varying severity [5,18]. Liver biopsy is particularly useful when advanced disease is suspected or when alternative diagnoses must be excluded.

Screening

Recent 2024–2025 clinical guidelines recommend screening children with obesity beginning between ages 9 and 11 years. Earlier screening is recommended for children with severe obesity, type 2 diabetes, or multiple metabolic risk factors [19,20]. These updated recommendations reflect increasing recognition that MASLD develops early in childhood and may progress silently.

Management

Lifestyle modification remains the cornerstone of treatment for pediatric MASLD. Weight reduction through improved diet and increased physical activity has been shown to reduce hepatic steatosis and inflammation. Studies demonstrate that weight loss of approximately 7% to 10% can significantly improve liver histology [8,21].

Pharmacologic therapy remains limited, and no medications are currently approved specifically for pediatric MASLD. However, emerging therapies including glucagon-like peptide-1 receptor agonists, vitamin E, insulin sensitizers, and thyroid hormone receptor agonists have shown promising results in recent clinical trials [22-24]. These therapies may improve insulin sensitivity, reduce hepatic fat, and slow disease progression.

Prognosis

The prognosis of pediatric MASLD varies depending on disease severity and fibrosis stage. Early-stage disease is often reversible with lifestyle intervention. However, advanced fibrosis may lead to long-term complications including cirrhosis and liver failure. Children with MASLD are also at increased risk of cardiovascular disease, type 2 diabetes, and metabolic syndrome later in life [4,25].

Conclusion

Metabolic dysfunction–associated steatotic liver disease is an increasingly common chronic liver disease in children and adolescents. Rising obesity rates and metabolic risk factors have contributed to the growing prevalence of this condition. Early screening, lifestyle intervention, and multidisciplinary care remain essential for preventing disease progression. Emerging therapies offer promising future treatment options. Increased awareness among healthcare providers and families is essential to reduce long-term morbidity and improve outcomes in pediatric MASLD.

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