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Metabolic dysfunction-associated steatotis liver disease (MASLD) in overweight and obese children: literature review

Abstract

Childhood obesity is a growing public health problem worldwide and is associated with multiple metabolic disorders. Metabolic dysfunction-associated steatotic liver disease (MASLD) is the hepatic manifestation of metabolic syndrome and the most common cause of chronic liver disease in pediatric populations in developed countries. MASLD encompasses a variety of clinical and pathological entities, ranging from simple hepatic steatosis to metabolic dysfunction-associated steatohepatitis (MASH), which can progress to fibrosis, cirrhosis, and terminal liver disease. Pediatric MASLD prevalence ranges from 3-10% in the general population but increases to 34-38% in children with obesity and up to 70-80% in severe obesity. Insulin resistance is the central pathogenic factor, creating a bidirectional cycle with steatosis. Genetic variants such as *PNPLA3* and *GCKR* modulate individual susceptibility to hepatic fat accumulation and fibrosis.

Keywords: metabolic dysfunction-associated steatotic liver disease, insulin resistance, metabolic syndrome, obesity, associated factors, pediatric

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Abbreviations: MASLD; metabolic dysfunction-associated steatotic liver disease; IR, insulin resistance; PRISMA, preferred reporting items for systematic reviews and meta-analyses; MRI, magnetic resonance imaging.

Introduction

Childhood obesity affects 340 million children and adolescents between 5 and 19 years worldwide. Simultaneously, steatotic liver disease is recognized as the most common chronic liver disease across all age groups.

Note on Nomenclature: Following the 2023 multi-society consensus (AASLD, EASL, ALEH), the medical community has transitioned from the term NAFLD to **MASLD** and from NASH to **MASH**. This update reflects the metabolic and endocrine risk factors discussed in this review.

Global MASLD prevalence affects 5.5-10.3% of children. These figures increase significantly in children and adolescents with obesity, reaching values up to 44.0%. The geographical distribution shows marked heterogeneity, reflecting the influence of environmental and metabolic factors.

Pathogenesis

The understanding of MASLD development has evolved from the classical **"two-hit hypothesis"** to a **"multiple-hit hypothesis"**. Steatosis begins with an imbalance between synthesis, flow, oxidation, and lipid export in the liver. Insulin resistance (IR) in adipose tissue prevents the suppression of lipolysis, sending a massive flow of free fatty acids into the liver. This excess exceeds mitochondrial oxidation capacity, generating free radicals that cause inflammation and lipoapoptosis.

Dietary factors, especially high fructose intake from sugary drinks, correlate with the epidemic increase in MASLD. Furthermore, gut dysbiosis increases intestinal permeability and the translocation of endotoxins. Genetic factors such as *PNPLA3*, *GCKR*, and *TM6SF2* significantly modulate the risk of fat accumulation and progression to fibrosis.

Diagnosis and evaluation

MASLD usually remains asymptomatic until significant damage occurs. Clinical signs include *acanthosis nigricans* (a marker of hyperinsulinemia), increased waist circumference, and hepatomegalia.

Analytical alterations

Pediatric MASLD is characterized by several biomarkers:

A. ALT: According to the **NASPGHAN** (North American Society for Pediatric Gastroenterology, Hepatology, and Nutrition) clinical practice guidelines, sex-specific cut-off values for screening are **22 U/L for girls and 26 U/L for boys**.[1] However, ALT levels are not always a sensitive marker of disease severity and may be normal in early stages.

B. AST/ALT Ratio: An increase may reflect progression to fibrotic MASH.

C. GGT: Elevated levels represent a risk factor for advanced fibrosis.

Imaging tools like ultrasound are widely used for their non-invasive nature, though they have limited sensitivity for mild steatosis. Liver biopsy remains the reference method for differentiating simple steatosis from MASH, but its use is limited in children due to its invasive nature.

Treatment strategies

There are no specifically approved drugs for MASLD in children; lifestyle modification remains the primary treatment. Recommendations include limiting carbohydrate and fructose intake, performing at least 60 minutes of daily exercise, and limiting screen time to less than 2 hours. While metformin treats insulin resistance, it is not superior to placebo for improving liver histology. Probiotics and omega-3 fatty acids are promising options currently under investigation.

Methodology

A systematic literature search was conducted following the **PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses)** guidelines to ensure transparency and reproducibility. The search was performed across databases including PubMed (MedLine) and Google Scholar for studies published between 2015 and 2025.

The selection process was divided into four stages:

1) **Identification:** Initial search using keywords such as "MASLD", "pediatric obesity", and "insulin resistance" yielded **408 records**.
2) **Screening:** Titles and abstracts were screened for relevance, resulting in the exclusion of 360 records that did not meet the thematic criteria or were duplicates.
3) **Eligibility: 48 full-text articles** were assessed for eligibility.
4) **Included:** After excluding 28 articles (reasons included: focus on adult populations, lack of primary data, or restricted access), **20 studies** were ultimately selected for the final qualitative synthesis.

PRISMA flow diagram data (Figure 1)

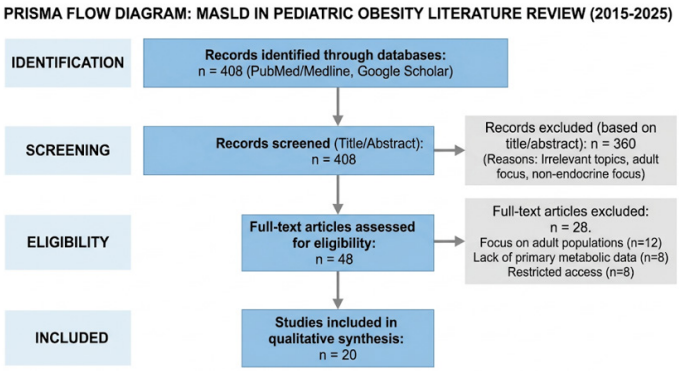

Figure 1 PRISMA flow diagram of the study selection process. Out of the 408 records identified, 20 studies met the full inclusion criteria and were included in the qualitative synthesis.

Identification: * Records identified through database searching: **408**

a) **Screening**

Records screened: **408**

Records excluded (based on title/abstract): **360**

b) **Eligibility**

Full-text articles assessed for eligibility: **48**

Full-text articles excluded: **28**

Reasons: Adult population (n=12), lack of full-text (n=8), irrelevant outcomes (n=8).

c) **Included**

Studies included in qualitative synthesis: **20**

Results

The overall prevalence of MASLD is estimated at 7.6% in the general pediatric population, increasing to 34.2% in children with obesity.[2] In cases of severe obesity, studies using magnetic resonance imaging (MRI) report that up to 70-80% of patients exhibit some degree of steatosis.[3,2]

Clinical data confirm a clear male predominance; in overweight populations, prevalence rates have been recorded at 36.05% in males versus 26.84% in females.[4] Ethnicity also plays a significant role, with Hispanic children showing a prevalence of 40-45%, representing a risk four times higher than non-Hispanic white populations.[4,1]

Regarding endocrine factors, insulin resistance (IR) remains the central pathogenic axis. Approximately 23.4% of children with MASLD present with prediabetes and 6.5% with type 2 diabetes.[5,6] Furthermore, the imbalance of adipokines—specifically high leptin and low adiponectin levels—is a key driver of the transition from simple steatosis to MASH and advanced fibrosis.[7,8]

Discussion

The results indicate that MASLD is the leading cause of chronic liver damage in children today. The transition to a "multiple-hit" model emphasizes the convergence of IR, adipokine dysfunction, and gut dysbiosis. However, methodological heterogeneity and the invasive nature of biopsies remain significant limitations in the current literature.[9–21]

Conclusion

MASLD is a major global public health challenge linked to the obesity epidemic. Significant underdiagnosis persists due to the limitations of current screening tools. Future research must focus on the validation of non-invasive biomarkers and birth cohort studies to better understand the natural history of the disease from childhood to adulthood.

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Conflicts of interest

The authors declare that there are no conflicts of interest regarding the publication of this study.

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