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Pathophysiology and clinical manifestations of fatty liver in children and adolescents: A systematic review

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ABSTRACT:
Background: *Non-alcoholic fatty liver disease (NAFLD) has become one of the most common liver diseases in children and adolescents, and its prevalence is increasing worldwide.*
Objective: *This study aimed to update and synthesize the latest findings on the pathophysiology and clinical manifestations of children and adolescents with NAFLD, focusing on publications in the last five years.*
Method: *The present study used the PRISMA 2020 Protocol (Preferred Reporting Items for Systematic Reviews and Meta-Analysis), the descriptors "Fatty Liver" and "Child" and "Adolescent", in the PubMed, Cochrane and LILACS databases, and with the filters: language (English, Spanish and Portuguese), year of publication 2019 to 2024 and full and free availability of articles.*
Results: *535 articles were identified, 11 articles were included in the study according to the inclusion and selection criteria of independent examiners. Of the articles included, data analysis showed that hepatic steatosis is related to increased levels of ALT and AST and obesity, in addition to metabolic risk factors, such as insulin resistance, dyslipidemia, cardiovascular diseases and visceral adiposity, resulting from the complex interaction between metabolic, genetic, epigenetic and environmental mechanisms.*
Conclusion: *NAFLD in children and adolescents is a multifactorial condition strongly linked to metabolic and demographic factors. Early recognition of at-risk individuals is critical to prevent disease progression and long-term complications. The findings reinforce the need for improved screening strategies and multidisciplinary approaches to pediatric NAFLD care.*
Keywords: *Fatty Liver, NAFLD, Systematic Review, Child and Adolescent.*

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I. INTRODUCTION

Non-alcoholic fatty liver disease (NAFLD) has become one of the most common liver diseases in children and adolescents[1,2], and its prevalence rate is rising all over the world[3]. This condition is characterized by excessive accumulation of fat in the liver in absence of heavy alcohol intake and is associated with several metabolic and cardiovascular complications[4]. If it is not treated, it may progress to fibrosis, cirrhosis, and hepatocellular carcinoma[5,6]. The occurrence of NAFLD in pediatric patients is increasing, which poses a significant challenge for children and adolescents and highlights the need for specific prevention and treatment strategies[3,7,8]. There is a strong link between NAFLD, children's metabolic dysfunction, childhood obesity and disease development.

The prevalence of NAFLD differs across ethnic groups, predominating among Hispanics[6,7,9,10], suggesting that these variations may be influenced by both genetic and environmental factors. Recent analyses, such as those by Hegarty *et al*. (2023)[11], emphasize the importance of several serum biomarkers in distinguishing the stages of the disease, which may also contribute as diagnostic criteria to improve early identification and individualized management of pediatric NAFLD. A review by Chen *et al*. (2021)[7] emphasized the importance of accurate and early diagnosis, and proposed advanced imaging methods and specific biomarkers as the key tools for early identification of NAFLD.

Regional analyses, such as those conducted by Mujlli *et al*. (2023)[1] in Saudi Arabia, provide detailed descriptions of the epidemiological and clinical characteristics of pediatric NAFLD. Studies such as those by Neri *et al.* (2022)[6] highlight the strong association between NAFLD and metabolic dysfunction in children, reinforcing

the key role of behavioral and nutritional interventions in the prevention and treatment of the disease. As emphasized by Furthner *et al.* (2022)[12], effective management of pediatric NAFLD often requires a multidisciplinary approach, involving pediatricians, nutritionists and psychologists.

This study aimed to update and synthesize the latest findings on the pathophysiology and clinical manifestations of NAFLD in children and adolescents, focusing on publications from the past five years. The main pathophysiological mechanism and the most common clinical manifestations were reviewed. To ensure the rigor and transparency of the review process, this study followed the PRISMA 2020 (Preferred Reporting Items for Systematic Reviews and Meta-analysis) guidelines.

II. THEORIC REFERENCE

Nonalcoholic fatty liver disease can be defined by the histological changes of the liver, which resemble those of alcoholic liver disease but occur in individuals who do not drink alcohol. The disease presents a wide spectrum of changes ranging from simple steatosis to progressive steatosis, fibrosis, and cirrhosis[7-10,13,14]. The exact pathophysiology of the disease is not fully understood, but insulin resistance is one of the main factors influencing the development of NAFLD[8].

The disease begins with the appearance of hepatic steatosis, a consequence of increased adipose tissue in organs, characteristic of the first stage of injury. Secondary to excessive lipid accumulation in hepatocytes, lipotoxicity, systemic inflammation, and oxidative stress stimulate liver tissue through cytokine secretion, contributing to fibrotic remodeling of the liver[2]. In patients with insulin resistance, lipid dissolution, fatty acid, and triglyceride synthesis increase in hepatocytes while VLDL export decreases. Elevated triglyceride and fatty acid absorption by the liver, a result of insulin resistance, reduces insulin effectiveness in adipocytes, stimulating lipolysis and increasing hepatic free fatty acid influx. Consequently, hyperinsulinemia leads to increased fat accumulation within hepatocytes[5,10].

In recent decades, the global rise in obesity has been accompanied by a parallel increase in NAFLD and its complications. The disease has therefore become one of the most common causes of chronic liver disease in children and adolescents[3,6,9,11-13,15].

The prevalence of NAFLD depends on multiple risk factors, including maternal obesity, gestational diabetes, metabolic syndrome during pregnancy, cesarean section, intrauterine growth restriction, low or high birth weight, antibiotics exposure during pregnancy and infancy, obesity, hyperinsulinemia, and high dietary intake of fructose, carbohydrate or fats[3,6,12,16]. Thus, beyond postnatal habits, the perinatal and intrauterine environment play crucial roles in influencing metabolic outcomes.

Although more common in obese individuals, NAFLD may also occur in patients with normal body mass index (BMI)[7,16]. Maternal obesity is strongly associated with childhood BMI[17], and children born to diabetic or obese mothers have increased subcutaneous fat at birth and higher risk of developing metabolic syndrome as early as six years old[11]. Breastfeeding is an important protective factor, also associated with reduced risk of steatohepatitis and fibrosis[3,16], likely due to its influence on gut microbiota composition[6].

NAFLD is more common in boys than in girls, with some studies reporting a prevalence rate up to twice higher in boys. One explanation for this finding is that men accumulate excessive adipose tissue in the abdomen and the influence of sex hormones themselves[9].

It is worth emphasizing the strong clinical correlation between NAFLD and metabolic syndrome, which led to the proposal of renaming the condition as metabolic dysfunction-related fatty liver disease (MAFLD), a term that better reflects the disease pathogenesis by highlighting the coexistence of metabolic dysfunction[3,4,6,11].

At present, there are no available data on the natural history and prognosis of pediatric patients with lean NAFLD, and hepatic steatosis in these individuals is often underestimated due to normal body weight[16]. Lifestyle interventions remain the mainstay treatment, including weight management methods, healthy diet, and exercise[8,11,12,15]. Although no effective pharmacological therapy has been established, different drugs can be used as modifiers of steatosis, inflammation, and liver fibrosis. However, there is an urgent need for further research in the pediatric population. Therefore, as long as defining pathology and its subtypes are accepted worldwide, given the heterogeneity of disease mechanisms, it is appropriate to consider fatty liver disease as a broad category because of the multiple etiological triggers.

The consequences of childhood obesity have been manifested in all aspects of health, both in childhood and adulthood. Quality of life tends to worsen as obesity progresses, accelerating deterioration of the overall clinical situation and speeds up establishing chronic diseases in this group[18]. Therefore, body composition is associated with cardiovascular risk factors, blood pressure, increased prevalence of abdominal adiposity, acanthosis nigricans, and other metabolic changes[19]. A strong correlation between insulin resistance and BMI was observed in selected pediatric groups[20].

Childhood and early adolescence represent important windows of opportunity, especially when considering that the likelihood of overweight children becoming overweight adults is >80%[17]. Moreover, early childhood is associated with an easier age to modify eating habits and prevent disease progression and

development of comorbidities[15]. Additionally, another very important and often underestimated aspect is that obesity in childhood is strongly associated with impaired mental health, with high prevalence of anxiety and depression[11].

Because NAFLD is often asymptomatic and progresses slowly, its consequences may be severe, just like cirrhosis and subsequent end-stage liver disease[9,18]. Scheen and Luyckx (2002)[14] noted that fat reduction helps decrease liver damage; however, the sudden loss of fat may lead to harmful hepatic changes, such as a transient increase in the degree of hepatocellular degeneration and focal necrosis.

III. METHOD

This study aims to summarize the latest information on the pathophysiology and clinical manifestations of fatty liver disease in children and adolescents by a systematic search in June 2024 using the descriptors "fatty liver" AND "child" AND "adolescent". These studies were written in English, Spanish, or Portuguese and indexed in the PubMed, Cochrane Library, and LILACS databases. Only articles with free full-text availability and published between 2019 to 2024 were considered. The inclusion criteria were: studies show that the pathophysiology and clinical manifestations of fatty liver in children and adolescents may contain obesity. The exclusion criteria were: studies focused primarily on treatment, other liver or systemic diseases, or steatosis as complications of another primary disease.

Articles selection began with screening the search results from the previously cited databases. First, titles were screened; second, abstracts were evaluated; and finally, the full texts of potentially eligible studies were read in detail. Based on this process, 535 records were identified. After removing 20 duplicates, 515 studies remained for screening. Of these, 22 were selected for full-text review, and 11 met all eligibility criteria and were included in the final synthesis. All stages of selection were performed independently by three reviewers, according to predefined criteria.

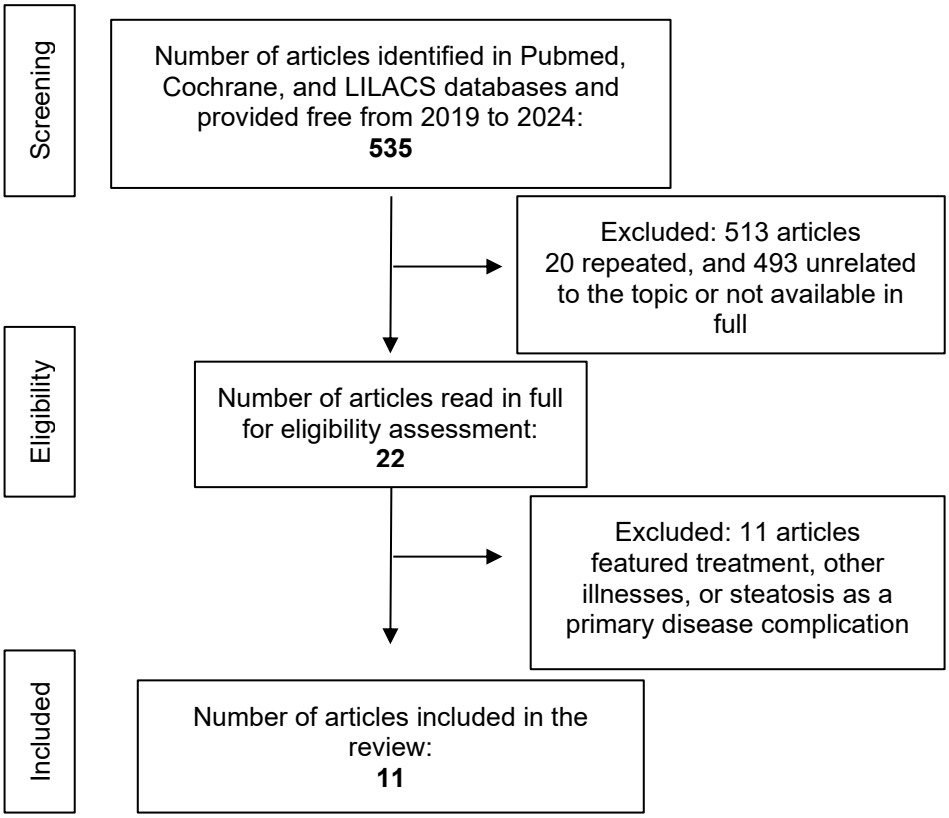
Data collection was completed by critically and completely reading the final articles and extracting parts, which included the pathophysiology and clinical manifestations of fatty liver in children and adolescents, to highlight the cross information between articles.

The identification, selection, and inclusion steps of this systematic review followed the PRISMA 2020 recommendation, and reviewing the selected articles followed the scheme planned by the author, aiming at determining the most common pathophysiology and clinical manifestations in children and adolescents, which may include the risk factors and complications of hepatic steatosis itself. Writing was based on the information extracted from the contained articles, highlighting the cross information between articles, thus enhancing the most common consensus expressions in pathophysiology and scientific circles.

The Newcastle-Ottawa Scale (NOS) was used to analyze the bias risk of the data extracted from the paper, measuring methodological quality via the number of "+" items received in selecting research groups, comparing groups, and checking exposure/outcomes. Two independent reviewers completed the analysis.

Deterministic evaluation relies on the GRADE (Grading of Recommendations Assessment, Development and Evaluation) protocol; this protocol evaluates the paper's evidence based on the deviation, inconsistency, and inaccuracy of the results, influence, confounding factors, and dose-response gradient.

This systematic review was registered in URL: https://osf.io/h5ckx. DOI: https://doi.org/10.17605/OSF.IO/H5CKX.



Picture 1. Article selection flow chart.

IV. RESULTS AND DISCUSSION

From an initial 535 studies, 22 were selected for full assessment. However, 11 were excluded because they featured treatment, other illnesses, or steatosis as a secondary complication. Therefore, 11 studies remained in the final qualitative synthesis. These papers systematically described the pathophysiology, clinical manifestations, and complications of fatty liver disease in children and adolescents, as well as the correlation between NAFLD, obesity and alterations in ALT and AST levels.

According to Klepper (2023)[15], among which 237 children aged 2 to 6 with fatty liver, 35% presented elevated ALT levels. The study also reported that increased γ-glutamyl transferase was associated with suspected NAFLD, a marker linked to more advanced disease in pediatric population.

Based on the selected materials, pediatric NAFLD is defined as existing as ≥5% macrovesicular steatosis in hepatocytes[8,12,16], in absence of alcohol or other identifiable causes, and may present varying degrees of fibrosis. According to Lee *et al*. (2024)[8], NAFLD prevalence has been increasing in Western countries, affecting approximately 10% of children, and rising to 35% among those with obesity. The clinical spectrum ranges from simple steatosis to non-alcoholic steatohepatitis (NASH), a condition characterized by inflammation and hepatocellular injury[8]. In some cases, the disease progression includes fibrosis, cirrhosis, or even hepatocellular carcinoma[6]. NAFLD is one of the fastest rising indications for liver transplantation in young adults, suggesting that the disease onset likely occurs during childhood[15].

Childhood-onset NAFLD increases the likelihood of developing advanced liver disease and the early appearance of complications[2]. According to all the articles analyzed, the development and progress of fatty liver disease involves a complex interaction of insulin resistance, dyslipidemia, cardiovascular disease, visceral obesity, heredity factors, epigenetic influences, and environmental exposures. Lifestyle and dietary patterns remain major determinants of metabolic imbalance. In addition, intrauterine and perinatal factors may predispose children to metabolic diseases and NAFLD. Therefore, maternal nutrition directly affects the risk of children's illness, inducing changes in hepatocyte metabolism, mitochondrial function, and postpartum intestinal microflora. Fibrosis stage is identified as the most relevant liver complication for prognosis[17]. About 11-15% of children referred for hepatology evaluation already present advanced fibrosis, particularly among obese individuals[11].

The renaming of NAFLD to Metabolic dysfunction-Associated Fatty Liver Disease (MAFLD) emphasizes its link to metabolic dysfunction. This new name better represents the disease's etiology and pathogenesis[3,6,11]. However, on the other hand, the new criteria may exclude patients without metabolic risk factors, who can still have NAFLD[12]. Hepatic steatosis diagnosis requires excluding other potential causes and involves identifying it through imaging, serum biomarkers, or liver biopsy, along with metabolic assessment. This

new definition doesn't fully explain the diverse range of diseases, it offers a way to identify individuals with poor metabolic health and a heightened risk of developing severe liver damage. Consequently, early screening will lead to faster diagnosis and intervention, which may prevent or postpone metabolic and cardiovascular complications associated with NAFLD in youth and later life.

The proportion of nonalcoholic fatty liver disease is more significant in obese people than in non-obese people. In one study, 70% of obese children had NAFLD compared with 30% of non-obese children[1]. Sahota *et al.* (2020)[21] showed that among 36,658 overweight or obese children aged 9 to 18, and the level of alanine aminotransferase exceeded 30 U/L, NAFLD diagnosis increased with time, from 36.0/100,000 in 2009 to 58.2/100,000 in 2018. In addition, in Song's research (cited by Lee HS[8]), the prevalence of NAFLD among Korean children and adolescents increased from 8.17% in 2009 to 12.05% in 2018.

Overall, pediatric NAFLD is a universal condition, increasing all over the world. Although it shares many characteristics with early adult diseases, pediatric NAFLD shows important differences. In particular, it is necessary to consider the influence and susceptibility of early life, progression and management strategies. Given that the disease often begins silently in childhood, recognizing risk factors and establishing early screening pathways are essential steps for preventing long-term hepatic and metabolic complications. There is a real chance to reverse the course of childhood disease, and pediatricians must be aware and ready to take action.

Article title	Citation	Results
A review of the increasing prevalence of metabolic dysfunction-associated fatty liver disease (MAFLD) in children and adolescents worldwide and in Mexico and the implications for public health.	Ramírez-Mejía *et al*., 2021	MAFLD prevalence in children and adolescents has increased globally and is the most common form of chronic liver disease in this age group. Risk factors include obesity, insulin resistance, and metabolic dysfunction. Early screening for risk factors is necessary.
Childhood and adolescent nonalcoholic fatty liver disease: is it different from adults?	Fitzpatrick & Dhawan, 2019	Pediatric NAFLD has important differences compared to adult disease, with influences and susceptibilities starting early in life. An early approach to diagnosis and treatment is essential to reverse the course of the disease in childhood.
Fatty liver disease in children (MAFLD/PeFLD Type 2): unique classification considerations and challenges.	Hegarty *et al*., 2023	Fatty liver disease is the leading cause of chronic liver disease in children. The term pediatric fatty liver disease should be used until the diagnosis of MAFLD is definitively established.
Incidence of nonalcoholic fatty liver disease in children: 2009-2018.	Sahota *et al*., 2020	NAFLD incidence increased substantially between 2009 and 2018. ALT >30 U/L was associated with suspected NAFLD, especially in overweight/obese adolescents. More attention is needed for the condition in pediatrics.
Liver steatosis: a marker of metabolic risk in children.	Neri *et al*., 2022	Hepatic steatosis is strongly associated with metabolic risk factors and may serve as an early indicator of metabolic dysfunction in pediatric populations.

Non-alcoholic fatty liver disease in non-obese children.	Zdanowicz *et al*., 2020	NAFLD also affects non-obese children, requiring a broader evaluation in pediatrics.
Non-alcoholic fatty liver in children and adolescents in Saudi Arabia.	Mujlli *et al*., 2023	The prevalence of NAFLD among children and adolescents is high, especially among those with obesity. Ultrasound is useful for identifying hepatic steatosis.
Nonalcoholic fatty liver disease in children and adolescents.	Lee HS, 2024	NAFLD prevalence is increasing worldwide. Early interventions are critical to prevent disease progression and long-term complications.
Nonalcoholic fatty liver disease in children with obesity: narrative review and research gaps.	Furthner *et al*., 2022	Pediatric NAFLD differs from adult NAFLD presentation and natural history. There is no consensus on screening; improved biomarkers and imaging tools are needed.
Nonalcoholic fatty liver disease in young children with obesity.	Klepper *et al*., 2023	The suspicion of NAFLD is common in very young obese children. Cardiometabolic markers were equivalent to ALT, and could precede the onset of comorbidities. Thus, early screening is essential.
Pediatric nonalcoholic fatty liver disease - a changing diagnostic paradigm.	Trandafir *et al*., 2020	Conventional ultrasound can be used for screening in the population, but magnetic resonance imaging is the most accurate method for diagnosis and classification. Serum markers and liver stiffness correlate with severity and risk of complications.

Table 1. List of studies included in the review

V. CONCLUSION

The increasing prevalence of hepatic steatosis in the pediatric population accompanies the global rise in childhood obesity. Across the analyzed studies, NAFLD consistently correlated with elevated ALT and AST levels, obesity, and multiple metabolic risk factors including insulin resistance, dyslipidemia, cardiovascular disease, and visceral obesity reflecting a complex interaction between metabolic, genetic, epigenetic, and environmental determinants.

Despite the growing burden of pediatric NAFLD, the review identified a scarcity of studies specifically addressing the pathophysiology and clinical manifestations of hepatic steatosis in children, which made the selection and data-extraction process challenging. This gap highlights the importance of research focused on the pediatric population, as early-life onset may carry unique implications for disease progression. Strengthening evidence in this field is essential to improve early detection, risk stratification, and long-term management strategies aiming at preventing the progression of the disease.

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