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Hyperuricemia in Obese Children: Diagnostic Challenges, Pathophysiological Mechanisms, and Therapeutic Approaches - systematic review of current literature

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Abstract:
Hyperuricemia in children with obesity is an increasingly common health issue, strongly associated with the development of various metabolic and cardiovascular diseases. It serves as a significant risk factor for conditions such as hypertension, type 2 diabetes, and chronic kidney disease. This study aimed to conduct a comprehensive review of the existing literature on the relationship between hyperuricemia and childhood obesity, adhering to the PRISMA guidelines for systematic reviews.

The primary mechanisms leading to hyperuricemia in this context include insulin resistance, lipid disorders, and impaired renal function. This article discusses several risk factors contributing to elevated uric acid levels in obese children, including a diet high in purines, excessive fructose intake, and a lack of physical activity. Additionally, it presents available diagnostic methods, such as measuring serum uric acid concentration and supporting tests like lipid profiles and renal function assessments. The article also explores preventive interventions, which encompass dietary and lifestyle modifications, as well as the early implementation of pharmacological treatments, such as allopurinol or metformin. Furthermore, it emphasizes the necessity for ongoing clinical research to develop more effective strategies for the treatment and prevention of hyperuricemia in obese children.

Key words: hyperuricemia, obesity, child obesity, uric acid, hypertension, type 2 diabetes

1. Introduction

Hyperuricemia is a condition characterized by elevated levels of uric acid (UA) in the bloodstream, typically exceeding 6 mg/dL or 7 mg/dL, depending on the specific study. This condition is significant for health because it is linked to several medical issues, including gout, kidney stones, cardiovascular disease, metabolic syndrome, and chronic kidney disease (CKD) (1–3). UA is the final product of purine metabolism and serves a dual role in the body. On one hand, it acts as an antioxidant; on the other hand, when present in high levels, it can contribute to metabolic and cardiovascular diseases such as hypertension, non-alcoholic fatty liver disease, type 2 diabetes mellitus (DM2), and gout. Additionally, UA plays a significant role in metabolic syndrome as it helps regulate oxidative stress, inflammation, and enzymes related to glucose and lipid metabolism (4–6).

Over the past four decades, the number of children and adolescents with obesity has increased by more than tenfold. Currently, approximately 1 in 3 children in the United States is overweight or obese. This rise in obesity contributes to the early onset of various physical illnesses, as well as mental, psychological, and psychosocial disorders (7–9) . Obesity and hyperuricemia are relatively common among children, with the prevalence of hyperuricemia ranging from 9.4% to 55.12%. It is notably more prevalent in obese children, especially in boys and older age groups (10–12). Elevated UA levels can be found in up to half of children who are overweight or obese (13). A significant body of research suggests a positive correlation between obesity and hyperuricemia, with obesity frequently occurring prior to the onset of hyperuricemia. Furthermore, both conditions play a crucial role in the development of metabolic syndrome and its associated complications (14,15).

This paper aims to discuss the mechanisms that lead to hyperuricemia in the context of obesity. It will review current research on this phenomenon and analyze the associated risks and potential health complications resulting from hyperuricemia in children with obesity.

2. Materials and methods

A systematic review followed PRISMA guidelines to identify all relevant papers considering hyperuricemia in childhood obesity. The final search took place on April 5, 2025, using search engines such as PubMed, Scopus, and Web of Science. We used the following search terms: "child," "obesity," and "hyperuricemia," along with their variations. Additionally, we examined the reference lists of relevant articles for more related studies.

In the research, we included studies involving children aged 2 to 18 years with obesity and hyperuricemia (defined as a UA level greater than 5.5 mg/dl) published since 1978. After removing duplicate entries, we reviewed the titles and abstracts of all obtained results, and ultimately assessed potentially relevant studies in full text. The detailed selection process is illustrated in Figure 1.

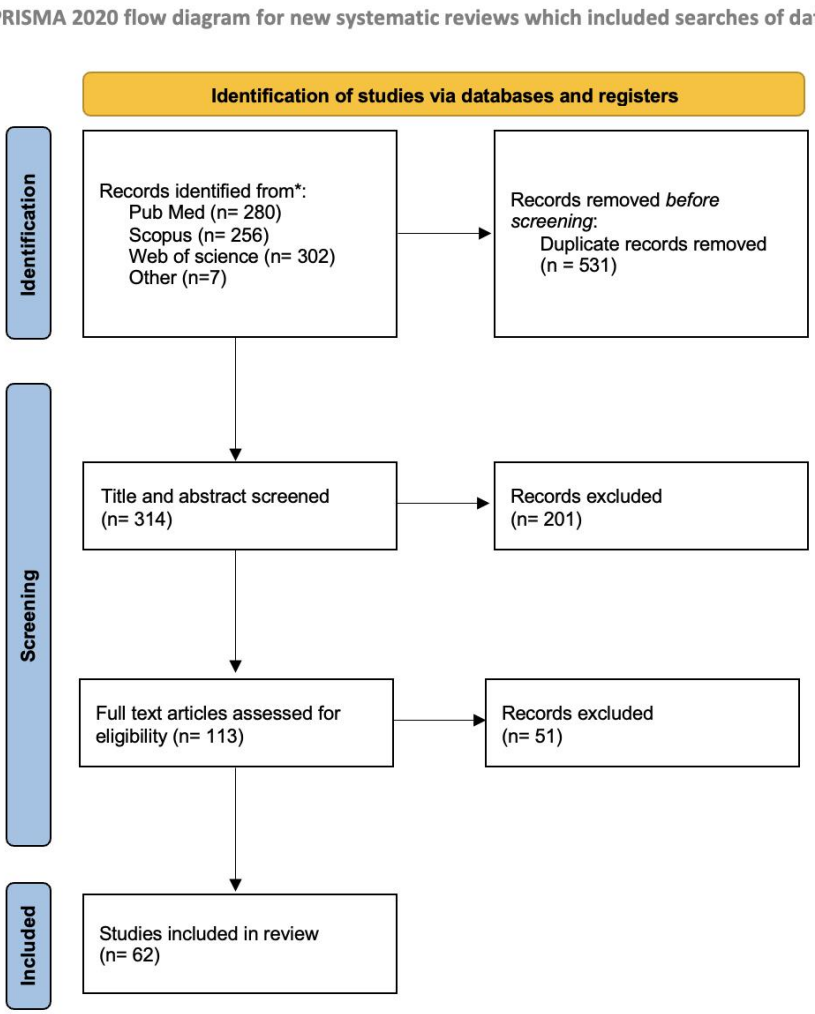

Figure 1. A diagram of screening and identifying relevant research papers, following PRISMA guidelines. From: Page MJ, McKenzie JE, Bossuyt PM, Boutron I, Hoffmann TC, Mulrow CD, et al. The PRISMA 2020 statement: an updated guideline for reporting systematic reviews. BMJ 2021;372:n71. doi: 10.1136/bmj.n7.

3. Hyperuricemia: mechanisms and pathophysiology

In children, UA is produced as a final product of purine metabolism and is primarily excreted by the proximal tubules of the kidneys. The levels of serum UA and the patterns of its excretion vary with age due to changes in the transport mechanisms within the renal tubules (16,17). The levels of UA in the blood are affected by genetic factors, metabolic syndrome, dietary choices (including fructose and alcohol consumption), exercise intensity, sex, age, and several components of metabolic syndrome such as obesity, insulin resistance, and hypertension (6,18–20). UA metabolism is regulated by the kidneys, small intestine, and liver, which helps prevent gout and protects against kidney damage (21) . Increased production of UA is often caused by consuming too many purines in the diet, resulting in their accumulation in the body. Purines are found in foods such as red meat, organ meats, seafood, and alcohol (especially beer). When these purines are metabolized, they produce UA. Excessive levels of UA can lead to hyperuricemia and related conditions, such as gout and kidney stones (22,23) . Additionally, high fructose intake may increase de novo purine synthesis, resulting in higher UA production and

leading to metabolic dysfunctions such as insulin resistance and hypertension (24). Elevated serum UA levels are often caused by reduced renal excretion. The kidneys eliminate approximately 70% of UA mainly through glomerular filtration, reabsorption, and tubular secretion (25). Disturbances in the expression and function of renal transporters, such as URAT1 and GLUT9, can lead to excessive reabsorption and retention of UA in the body (26). Furthermore, insulin resistance and activation of the renin-angiotensin-aldosterone system lead to sodium and UA retention, which exacerbates their excretion (27–29). Additionally, inflammation and oxidative stress contribute to kidney dysfunction and impaired UA excretion mechanisms (29,30). Obesity is a major risk factor for hyperuricemia, and its effects stem from various underlying mechanisms. Insulin resistance, which frequently occurs alongside obesity, enhances the reabsorption of UA in the kidneys through the URAT1 and GLUT9 transporters, leading to decreased UA excretion (31). Moreover, the dyslipidemia associated with obesity increases oxidative stress and inflammation, which can impair kidney function and further contribute to the retention of UA (12,29,32,33). Additionally, adipose tissue influences hyperuricemia in obese individuals through hormones and cytokines like leptin and adiponectin, leading to a pro-inflammatory state and insulin resistance (34).

4. Epidemiology and risk factors

Childhood obesity rates vary significantly across different regions and populations, ranging from approximately 8% to over 40% in some countries. This issue is influenced by factors such as socioeconomic status, education level, and ethnicity. There has been a dramatic increase in the incidence of childhood obesity in the pediatric population. For example, in the United States, the prevalence of childhood obesity has more than tripled, rising from 5% in 1978 to 18.5% in 2016 (35–37). Furthermore, there has been a noticeable increase in hyperuricemia cases among obese children. For instance, in China, the prevalence of hyperuricemia in obese children and adolescents is 54.8%. This rate has shown a consistent upward trend over the past seven years, particularly among boys (28,38–40). There are several risk factors that contribute to the development of obesity and hyperuricemia. These factors include genetics, lifestyle choices, diet, social and environmental influences, and physical inactivity. Specific genetic predispositions to obesity and hyperuricemia are associated with polymorphisms in genes such as ABCG2, SLC22A12, XDH, MSRA, CYP24A1, and FTO. The interaction between these genetic variants and elements such as body mass index, gender, and dietary habits can significantly influence the risk and severity of these conditions (41–43). An unhealthy lifestyle, characterized by a diet high in purines, sugary beverages, and fast food, contributes to the development of hyperuricemia and obesity through various metabolic processes. Consuming high-purine foods increases the production of UA, potentially leading to elevated levels in the bloodstream. Additionally, fructose-sweetened beverages promote the de novo synthesis of purines, which further raises UA levels and can lead to insulin resistance. A diet rich in highly processed foods, which are high in fats and simple sugars, encourages excessive calorie intake and weight gain, heightening the risk of insulin resistance and further increasing UA levels (43–46). Environmental factors, including food availability and safety, as well as home environments, significantly influence dietary choices, physical activity, and obesity rates (28,33,47). Additionally, maintaining a healthy lifestyle can reduce the risk of hyperuricemia by 41%, particularly among individuals with a low genetic risk who also follow a healthy diet (6,48). A lack of physical activity can lead to hyperuricemia by increasing the risk of obesity and insulin

resistance, which results in higher levels of UA in the blood. In contrast, regular physical exercise can help lower UA levels, enhance insulin sensitivity, and support weight management (49).

5. Diagnosis of hyperuricemia in obese children

Hyperuricemia in obese children is a significant clinical concern, as it increases the risk of developing gout and other metabolic disorders. The primary laboratory diagnostic tool is measuring the concentration of UA in the blood serum. Reference values vary based on the child's age and gender; however, levels exceeding 5.5–6.0 mg/dl (327–357 μmol/l) are typically considered elevated and warrant further evaluation. Stiburkova et al. report that hyperuricemia is defined as serum UA levels exceeding 370 μmol/L (6.22 mg/dL) in girls and exceeding 420 μmol/L (7.06 mg/dL) in boys. In addition to UA measurement, other laboratory tests are recommended to gain a comprehensive understanding of the child's health and to identify potential complications associated with obesity and hyperuricemia. These tests include assessing the concentrations of creatinine and urea in the blood serum, which help evaluate kidney filtration function. Elevated levels may indicate impaired kidney function, an important consideration in the context of hyperuricemia. Additionally, analyzing the lipid profile—which includes total cholesterol, HDL (high-density lipoprotein), LDL (low-density lipoprotein), and triglycerides—is crucial due to the frequent occurrence of lipid disorders in obese children. Dyslipidemia can increase cardiovascular risk and necessitates appropriate management (11,50). Furthermore, renal ultrasound examination is crucial for assessing structural changes in the kidneys that may arise from chronic UA retention and obesity (51).

6. Associations between hyperuricemia and comorbidities in obese children

Hyperuricemia in obese children significantly increases the risk of developing severe chronic diseases, such as hypertension, atherosclerosis, insulin resistance, DM2, CKD, and metabolic syndrome. Hyperuricemia plays a role in developing hypertension, atherosclerosis, and various cardiovascular diseases through mechanisms such as endothelial dysfunction, oxidative stress, inflammation, and impaired nitric oxide synthesis, although the exact molecular pathways remain unclear (6,52–54). Additionally, hyperuricemia is associated with the onset of insulin resistance and DM2 through mechanisms involving metabolic inflammation, impaired insulin signaling, and a reciprocal relationship with metabolic syndrome, which contributes to vascular complications and diabetic nephropathy (55). In addition, hyperuricemia is an independent risk factor for the faster progression of CKD in children and adolescents (56). Elevated serum UA levels are prevalent among children with CKD and are linked to renal dysfunction, hypertension, obesity, and albuminuria (28,57). Additionally, Tang et al. demonstrated that hyperuricemia is linked to metabolic syndrome in obese Japanese children and adolescents, with triglycerides identified as the most frequently abnormal component of the metabolic syndrome (58).

7. Prevention and treatment of hyperuricemia in obese children

Hyperuricemia in children with obesity poses a significant risk for cardiovascular disease and metabolic syndrome. Effective preventive measures primarily involve lifestyle changes, particularly weight loss through the adoption of a healthy diet and regular physical activity. Research shows that even moderate weight loss can lead to lower blood UA levels and an improved cardiometabolic profile in these children. Additionally, health education is crucial for preventing hyperuricemia in both children and their families. It promotes proper eating habits and encourages physical activity. Early intervention, such as dietary changes and increased exercise, can significantly reduce the risk of complications associated with hyperuricemia and obesity (12,28,59,60).

Treatment of hyperuricemia in children involves both non-pharmacological and pharmacological interventions. Non-pharmacological approaches, such as dietary and lifestyle modifications, are crucial in reducing UA levels, particularly in children with obesity. Research indicates that increasing physical activity and adopting a balanced diet can lead to improved lipid and glycemic profiles, as well as a reduction in serum UA concentrations. It is especially important to limit the intake of fructose, as excessive consumption is linked to higher UA levels and an increased risk of non-alcoholic fatty liver disease in children and adolescents. Therefore, promoting healthy eating habits and encouraging regular physical activity can significantly contribute to lowering UA levels and decreasing the risk of metabolic complications in children with obesity (43,61,62).

The pharmacological treatment of hyperuricemia in obese children is considered when lifestyle changes do not yield the desired results. Allopurinol, a xanthine oxidase inhibitor, is the most commonly prescribed medication for lowering UA levels by inhibiting its synthesis. However, using this drug in children requires caution due to the potential for adverse effects. Metformin, primarily used for treating insulin resistance and DM2, also has the ability to reduce UA levels, which can be beneficial for obese children with hyperuricemia. It is important to note that research on the efficacy and safety of pharmacotherapy for hyperuricemia in children is limited, highlighting the need for further studies in this population (6,43,46). To develop new and effective strategies for treating and preventing hyperuricemia in children with obesity, it is essential to address challenges related to the individualization of therapy, patient acceptance, and the monitoring of treatment effects. Consequently, additional clinical and epidemiological studies are needed to identify the best methods for managing this condition.

8. Conclusions

Hyperuricemia in children with obesity is a significant health issue that increases the risk of various chronic diseases, including hypertension, DM2, cardiovascular disease, and chronic kidney disease. Early identification of this condition and the adoption of preventive measures, such as weight loss, a healthy diet, and increased physical activity, are crucial. In cases where lifestyle changes do not yield the desired results, medications like allopurinol and metformin can serve as complementary treatments. However, the challenges associated with treating hyperuricemia in children, such as tailoring therapies to meet individual patient needs, highlight the need for further research. Additional epidemiological studies are necessary to better understand the mechanisms and long-term effects of hyperuricemia in children with obesity. Moving forward, it is essential to develop effective and safe therapeutic strategies that can be widely implemented for this patient population.

9. Disclosures

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The authors confirm contribution to the papers as follows:

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The author declares no conflict of interest.

Declaration of the use of generative AI and AI-assisted technologies in the writing process.

In preparing this work, the author(s) used Open AI Chat Generative Pre-trained Transformer for the purpose of correcting spelling mistakes, punctuation mistakes, grammatical errors and stylistic errors. After using this tool/service, the author(s) have reviewed and edited the content as needed and accept full responsibility for the substantive content of the publication.

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