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Hyperuricemia in Childhood: Review of Literature from a Rare Case of Gout Arthritis in a Teenage Boy

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Abstract Gout is the most common inflammatory arthritis among the adult population. Rare cases of the disease are reported in childhood. The rarity of gout in children prompts the following case report with a review of literature. This is a clinical case of a 13-year-old non-obese boy admitted to the nephrology department for repeated migratory joint pain with hyperuricemia and renal dysfunction, where gout seems to be the most likely underlying cause.

Keywords: *uric acid, gout, child, kidney failure*

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1. Introduction

As children and adolescents are often involved in physical activities, joint pain is usually linked to a mechanical origin [1].

However, children can also experience joint pain of the inflammatory type. We report a case of joint pain in a boy with elevated uric acid levels and impaired kidney function. Gout is one of the rare causes of inflammatory pain in childhood [2]. In this case, we will talk about symptomatic hyperuricemia with renal function impairment, as well as the causes and treatment of acute gout in children.

2. Case Report

For nearly eight months, he complained of migratory joint pain with redness, swelling, and heat, without fever or other associated symptoms, according to his parents. He never took any medication, and the pain went away on its own after a few hours of rest. There is no known history of trauma or accident. One week prior to this admission, he described left metatarsophalangeal joint pain, which resolved on its own; left interphalangeal joint pain then developed moderately.

Two days later, he reported sharp and severe pain in the right medial malleolus ankle, with no triggering factor. This pain was worse with movement, intense in the early morning, permanent, and accompanied by intermittent swelling, and the inability to walk. There was no mention of fever, sore throat, morning stiffness, photosensitivity, or Raynaud's phenomenon. His parents took him to a nearby hospital, where he received symptomatic treatment consisting of "diclofenac sodium, dexamethasone, and sodium bicarbonate." His blood tests revealed that he had elevated uric acid and creatinine levels, so he was transferred to our department for evaluation.

He does not engage in regular physical activity. He had a palatoplasty for cleft lip when he was one year old. His parents are both healthy. The family history was negative for gout, suspected genetic conditions, autoimmune diseases, and other chronic conditions. His immunizations were up to date. His psychomotor development was normal. He was physically fit.

On physical examination, the temperature was 36.6°C with heart rate of 70 bpm, respiratory rate of 20 bpm and a blood pressure of 117/75 mmHg.There was no skin rash, no palpable superficial lymph nodes, and no bone deformity. There were vertical scars on the right side of the nasolabial sulcus and white stripes of purulent secretions on the posterior pharyngeal wall. The right internal ankle was swollen and hot. The rest was unremarkable.

An X-ray of the right foot revealed no significant bone abnormalities. On ultrasound, the structures of both kidneys were not clear. The size of the right kidney was atrophic, about 82 mm x 28 mm, with enhancement in parenchyma and renal cyst. The echogenicity of the left renal parenchyma was slightly stronger. There was no separation of the double kidney collection system. Laboratory findings were notable for the following. WBC: 9.26 G/ L. Hb: 14 g/dl. PLT: 406 G/L. CRP: 12.5 mg/l. Creatine: 125 umol/L. Uric Acid: 1039 umol/L. ESR: 9. Proteinuria 2+, Hematuria+-. Urine micro albumin: 31.8mg / L, urine albumin / creatine ratio: 45.2mg / g.

ANA, dsDNA, ANCA, GBM, HLA-B 5801 antibodies, IgA, IgG immunoglobulin, C3 and C4 complements, liver function tests, HIV, HBV, and HCV serology were all unremarkable.

He was initially treated for acute gouty arthritis with oral celecoxib and a low-purine diet. The evolution in the ward was marked on day 4 of admission by the inflammation of the back of the right hand, with skin redness, heat, and limited joint movement at the third metacarpophalangeal joint. Because of the persistence of pain, oral prednisolone 5 mg, one tablet daily was added. The pain was successfully controlled and he was discharged from the ward with a regular follow-up in the outpatient department.

3. Review and Discussion

3.1. Definition

Hyperuricemia is the increasing level of the uric acid level in the blood. The upper limit for uricemia has been set at 70 mg / l (420 µmol / l) in men and 60 mg / l (360 µmol / 1) in women for adult population [3]. High levels can lead to gout [4]. Uric acid serum in childhood reflects physiological changes and structural development (Table 1).

Table 1. Serum uric acid levels in children and adolescents [5,6,7,8]

Age	Uric acid (mg/l)	
	Mean	SD
Children		
<5 year	36	9
5-10 years	41	10
Adolescents		
Male		
12 years	44	11
15 years	56	11
18 years	62	8
Female		
12 years	45	09
15 years	45	09
18 years	40	07

3.2. Pathophysiology

The metabolism of purine nucleotides leads to uric acid with an apparent complexity [5]. It comes from the catabolism of endogenous and exogenous nucleic acids and de novo biosynthesis [6]. In humans, the ultimate catabolite of purine is uric acid. The kidney is the main route of elimination for uric acid. Excessive production of uric acid or insufficient elimination is responsible for hyperuricemia [7]. This can develop at low noise or lead to acute gout with its typical monoarticular pain attack, or to chronic gout with its deleterious renal effects. The absence of coexisting diseases or drugs that alter uric acid production or excretion leads to primary hyperuricemia [8]. Overproduction of urate or under excretion by the kidney can cause secondary hyperuricemia.

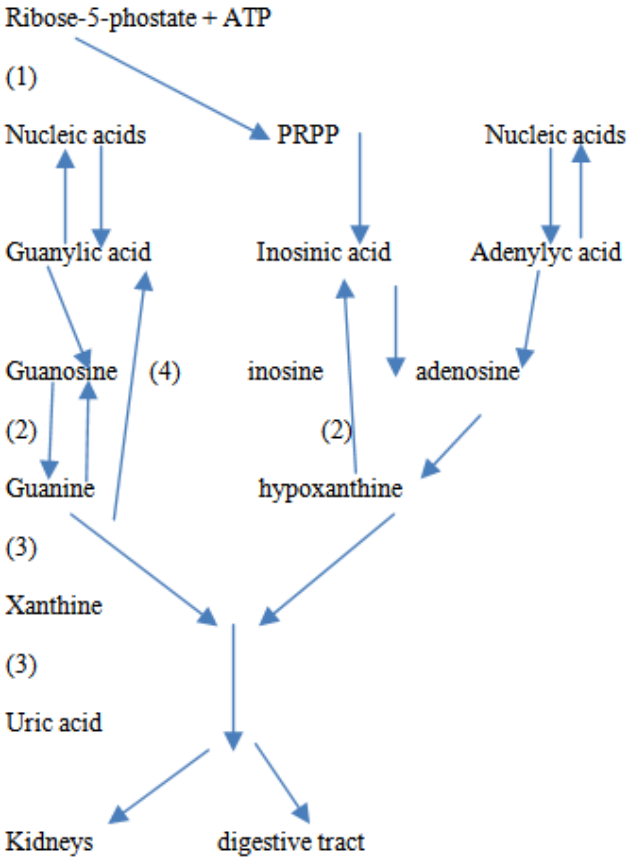

Figure 1. Production and excretion of uric acid. 1, PRPP synthetase; 2, purine nucleoside phosphorylase; 3, xanthine oxidase; and 4, hypoxanthine-guanine phosphoribosyl transferase. Adapted from Wilcox W.D., 1996

3.3. Prevalence

Hyperuricemia and gout have been widely reported in China and across the world. In 2010, Song et al have estimated the prevalence of hyperuricemia at 6.4% in general middle-aged and older Chinese using the baseline data from the China Health and Retirement Longitudinal Study (a nationally representative survey). [9]. This result was not as prevalent as in developed countries. The prevalence varied greatly according to demographic, socioeconomic, and geographic factors of the Chinese mainland. The pooled prevalence of hyperuricemia and gout was 13.3% and 1.1%, respectively, based on a systematic review of relevant chinese articles from 2000 to 2014 identified through electronic databases searches [10]. In that review, the authors also suggested that the geographical region might be associated with prevalence.

The prevalence of hyperuricemia in children has been poorly studied. Many studies reported hyperuricemia in conjunction with other risk factors such as obesity, cardiovascular disease, and metabolic syndrome [11,12]. In a cross-sectional survey using data from the seventh Korea National Health and Nutrition Examination Survey (2016-2017), the prevalence of hyperuricemia was estimated at 9.4% among Korean children from 10 to 18 years old [13]. Metabolic syndrome, abdominal obesity, and BMI z scores were significantly associated with hyperuricemia in that study population.

A cross-sectional survey in six urban districts of Tianjin in 2015 estimated the prevalence of hyperuricemia and its major risk factors among Chinese preschool kids [14]. Based on the results, the prevalence of hyperuricemia among children aged 3 to 6 years was 10.1%. This prevalence was significantly higher among boys (11.8%) than girls (8.3%). Several metabolic syndrome components were associated with the risk of hyperuricemia: BMI and triglycerides. This result is similar to the previous one.

Another cross-sectional study in aboriginal children with a high prevalence of gouty arthritis was conducted in 2001, with the objective of exploring the factors influencing serum uric acid concentrations and the prevalence of hyperuricemia [15]. In that study, Children aged 4-13 were requested to fill out a structured questionnaire with the assistance of their parents. 414 children (mean age, 8.9 +/- 2.1 years) were recruited. 40.2% of girls and 29.5% of boys were found to have hyperuricemia. Hyperuricemia was significantly associated with serum creatinine level, BMI, and a family history of gouty arthritis in parents. That conclusion highlight the fact that hyperuricemia may be detectable early in childhood.

While many studies confirmed that hyperuricemia predicts the development of CKD in adults as well as the deterioration of renal function in kidney disease patients, this has not yet been demonstrated in children [11,16,17,18]. According to the CKD in Children (CKiD) study, a cohort of 891 children seen at 55 pediatric nephrology centers across North America, the treatment of children and adolescents with CKD with urate-lowering therapy could possibly slow kidney disease progression [19].

One study compared 51 juvenile gout patients (age ≤ 18 years) and 337 adult gout patients in Guangdong Second Provincial General Hospital (from 2016 to 2018) and described comparative clinical features between adult and children gout [20]. The authors analyzed the Clinical parameters and laboratory data of the two groups. The average age of onset in children with gout was 15.0±1.9 years and the youngest patient was 8 years old [20]. Juvenile gout was associated with higher serum uric acid levels and greater involvement of finger joints. Correlations with BMI and systemic inflammation were less prominent compared to gout in adults [20].

3.4. Clinical Features and Evaluation

Hyperuricemia may be symptomatic or asymptomatic. Most of the time hyperuricemia does not result in apparent clinical manifestations in the early stage. The major manifestations of hyperuricemia are: arthritis, renal lithiasis and tophaceous deposits [21].

The presentation depends mainly on the degree and duration of the hyperuricemia. If gout is the underlying etiology, we can see warm and swollen erythematous joint generally on the big toe, but still possible with any other joint. In our case study, the young patient presented with symptomatic acute ankle arthritis. A complete history taking, including meticulous family history and physical examination, may establish or suggest one of the underlying conditions associated with hyperuricemia. A plan, including appropriate laboratory tests and consultations, can help to confirm the diagnosis. In other patients, the presentation is asymptomatic and the history and physical examination are unrevealing. Serum uric acid, joint aspiration, and demonstration of crystals in cases of gout are important. However, our patient did not present with joint effusion. More than 800 mg of uric acid on 24 hours on a normal diet suppose an overproduction of purine as etiology [21]. It is also useful in assessing the risk of stone formation [21,22]. Increased urinary excretion of uric acid indicates increased production of uric acid, whereas normal or low excretion suggests decreased renal clearance of urate as the cause of hyperuricemia [23]. Unfortunately, urine uric acid excretion, enzyme deficiencies, and gene mutations associated with hyperuricemia and renal insufficiency were not performed in our case. Simple radiography may evaluate acute gout, however radiographic imaging findings like soft tissue masses, cystic changes, and well-defined erosions with sclerotic margins and overhanging bony edges generally appear in advanced chronic gout. In an acute situation, it can help in a routine differential examination.

3.5. Treatment

Early induction of comprehensive therapy can avoid severe clinical conditions [24,25]. The risk of complications and mortality due to cardiovascular event or kidney disease justify the treatment [26]. The following treatment options can be used to lower uric acid levels.

1. *Febuxostat* which is a selective inhibitor of xanthine oxidase recently developed and has not been formally studied in children and further investigation is required to determine the effect and safety of this medicine for children. Nevertheless, it used to prevent tumor lysis syndrome (TLS) in children who received induction chemotherapy for hematologic malignancies. A retrospective study by Kishimoto et al in Japan, performed on 45 pediatric patients with hematological malignancies who received Febuxostat (10 mg daily, n=20) or allopurinol (300 mg/m^2 daily, n=25) had found a notable effect in reducing serum uric acid level in this group with Febuxostat [27].
2. *Allopurinol* is the most famous lowering uric acid drug. It is an old inhibitor of xanthine oxidase and widely prescribed in adult population. However, precaution should be made first by testing the human leukocyte antigen HLA B 5801 like the one we did in our case study to avoid severe secondary skin reaction specially the allopurinol-induced Stevens-Johnson Syndrome [28].
3. *Rasburicase* is uric acid oxidase that reduce serum uric acid level in some tumor lyse syndrome related malignancy but secondary hemolysis reported as serious adverse effect [28,29].

Good lifestyle with physical exercise, low purine diet, alkalization of urine could be important in addition, as we, seen obesity were correlate with hyperuricemia and cardiovascular events [29].

3.6. Etiologies

These are the main causes of hyperuricemia based on the predominant mechanism by which they occur (Table 2).

Table 2. Etiologies of hyperuricemia in children and adolescents [30]

Over production	Under excretion
√ Gout √ Sickle cell anemia √ Cyanotic congenital heart disease √ Hypoxanthine-guanine phosphoribosyl transferase (HGPRT) deficiency • Complete (Lesh-Nyhan Syndrome) • Partial (Kelly-Seegmiller Syndrome). √ Myoadenodenylate deaminase deficiency √ Glycogen storage disease from type I, III, V and VII √ Acetyl-coenzyme A dehydrogenase deficiency √ Reye Syndrome √ Pancreatic enzyme replacement √ Phosphoribosyl pyrophosphate (PRPP) synthetase variant √ Malignancy like myeloproliferative syndromes √ familial juvenile hyperuricemic nephropathy (FJHN)/medullary cystic kidney disease (MCKD)	√ drugs: • ethambutol • salicylates • diuretics • pyrazinamide • levodopa • nicotinic acid √ disease related condition: SLE, HTN √ acute disorders: • volume depletion • acute renal failure • hemolytic uremic syndrome • lead intoxication • metabolic acidosis,

1. Gout is a common medical problem in adult population; it is uncommon in children [17,30]. After all, kids do not eat diets rich in animal protein and do not drink beer. In pediatrics, join pain is mostly associated with physical injury or sport overuse. Nevertheless, today we know that genetics play a big part in the development of gout [30,31]. The disease is extremely rare in children without the presence of contributing factors such as underlying medical conditions or medications [30,31,32]. In this case report it does seem to be the most likely diagnosis with no real alternative explanation and typical history. Genetic and metabolic tests may not always give clear answers. In 2014, a case of gout in a 15-year-old obese boy with juvenile idiopathic arthritis was described. The author concluded that falling to diagnose gout may result in delay of treatment and may cause long-term effects of joints destruction [33]. According to ACR/EULAR criteria, the probability of gout in our patient was very high because of the presence of the following clinical features [34].

√ *More than one episode of swelling, pain and tenderness in a peripheral joint.*

√ *This pattern of joint pain involving first metatarsophalangeal joint*

√ *Inability to use joint and walking, with erythema overlying the joint*

√ *Recurrent typical episodes*

√ *Serum uric acid level more than 100 mg/l*

2. In 2015, the previously known familial juvenile hyperuricemic nephropathy (FJHN), medullary cystic kidney disease type 2 (MCKD 2) and UMOD-associated kidney disease was named by Kidney Disease Global Outcomes (KDIGO) as Autosomal Dominant Tubulointerstitial Kidney Disease (ADTKD) caused by UMOD gene mutation (ADTKD-UMOD) [35]. ADTKD-UMOD is a rare disease, the manifestations are present at adulthood [35,36]. Up to now, no more than 2000 families have been reported worldwide [36]. The clinical manifestations of ADTKD-UMOD include hyperuricemia and gout; some patients have mild urinary abnormalities [36,37]. It usually develops to end-stage renal disease (ESRD) at 30–60 years old [37]. There is no specific therapy of the disease, and to date, more than 100 mutations reported in UMOD gene in the world [38]. A Chinese study in 2019 reported less than 10 cases of this gene mutation in mainland China in population before 25 years old and they found that family history is a key clue for the diagnostic [38]. As described in KDIGO consensus in 2015, positive family history is a very important clue to the diagnosis of ADTKD-UMOD [35,39]. Our patient family history was negative for specific past medical history of rheumatic disorder. Hyperuricemia is usually present with patient with renal disease. In our patient, the serum uric acid is much greater than would be expected for the degree of chronic kidney disease found. It is doubtful that the kidney disease is secondary to the renal hyperuricemia (alone), but many studies confirm uric acid as independent predicting factor for chronic kidney disease [39,40]. At the time of writing this article, genetics tests were not analyzed. So inherited hyperuricemic disorders were not formally excluded.

4. Conclusion

This case report described acute gout with evidence of hyperuricemia from literature indicating causes and management. Multidisciplinary (rheumatologist, nephrologist, pediatrician) follow up will give to our young patient an optimal long-term management. Therefore, we should evaluate a child with episodic joint pain and hyperuricemia for secondary causes, accessed renal function, investigated family history for genetic disorders, and taken out the appropriate management to prevent complications in old age.

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