

Case report:

An uncommon case of gouty arthritis in a teenage boy

Ain Farah Abdul Rhahman¹, Nurul Izaa Mohamad², Soo Huat Teoh³

Abstract:

Joint pain is a common complaint in the paediatric age group with overuse and traumatic injury being the common causes. However, joint pain involving multiple sites, persistent or recurrent, and severe may suggest other diagnoses. One of the possible groups of diagnosis is rheumatology in nature, which is often difficult to diagnose, especially in paediatric patients. Gouty arthritis is a rather uncommon rheumatology problem in children and the diagnosis is often missed at the initial presentation. We report a 16-year-old boy who repeatedly presented with debilitating and alternating polyarthritis, diagnosed and treated as juvenile gouty arthritis after thorough clinical assessment and basic laboratory investigations in a resource-limited primary care clinic.

Keywords: gout; juvenile; arthritis; paediatric

*Bangladesh Journal of Medical Science Vol. 20 No. 01 January'21. Page : 200-203
DOI: <https://doi.org/10.3329/bjms.v20i1.50372>*

Introduction

Gouty arthritis, both acute and chronic, is a clinical diagnosis of joint inflammation due to the deposition of monosodium urate (MSU) crystals.¹⁻³ Other than joints, MSU crystals can also deposit in soft tissues such as cartilage and renal parenchyma, manifesting as tophaceous gout and uric acid nephrolithiasis.^{1,3} Hyperuricemia at serum uric acid (SUA) level around 420 $\mu\text{mol/L}$ is an important predisposing factor for MSU crystals deposition causing the clinical syndromes.^{1,2} Risk factors associated with gout can be divided into nonmodifiable and modifiable. Male gender, advancing age, ethnicities, and genetic inheritance are risk factors from the former, while high purine diet, alcohol ingestion, obesity, and medications use are from the latter.¹⁻³

The prevalence of gout in the United States (US) has been increasing since the year 1970, with an estimated three to eight million citizens diagnosed, which is more than 3% of the US adults.³ This increasing trend was also observed worldwide possibly due to unhealthy eating habits, sedentary lifestyle, and presence of concurrent comorbidities

such as diabetes, chronic kidney disease, and cardiovascular diseases.³ Of note, the prevalence of gout among countries in the Asia-Pacific region has a wide variation, ranging from 5.3% in Micronesia and Cook Islands to 25% in Taiwan.¹

Normal SUA level in children ranges from 180 to 240 $\mu\text{mol/L}$ for both genders which are below the expected threshold for MSU crystals deposition; hence, gout is rare in children.¹⁻³ However, marked hyperuricemia in children can happen due to unregulated urate production and impairment of renal uric acid clearance, which is both due to rare genetic defects.³

Case report

A 16-year-old teenage boy presented with painful and swollen left wrist, left first metatarsophalangeal (MTP) and bilateral second proximal interphalangeal joints for three days. It was acute in onset and affected his daily house chores and school attendance. It was his third episode in three months. His presentation during the last two visits is depicted in Figure 1. For his previous visits, he was treated symptomatically

1. Ain Farah Abdul Rhahman, Medical & Health Officer, Klinik Kesihatan Jepak, Bintulu, Sarawak, Ministry of Health Malaysia
2. Nurul Izaa Mohamad, Family Medicine Specialist, Klinik Kesihatan Bayan Lepas, Penang, Ministry of Health Malaysia
3. Soo Huat Teoh, Medical Lecturer & Family Physician, Lifestyle Science Cluster, Advanced Medical & Dental Institute, Universiti Sains Malaysia, 13200 Kepala Batas, Penang, Malaysia

Correspondence to: Soo Huat Teoh, Medical Lecturer & Family Physician, Lifestyle Science Cluster, Advanced Medical & Dental Institute, Universiti Sains Malaysia, 13200 Kepala Batas, Penang, Malaysia.
e-mail : soohuat@usm.my

with paracetamol and nonsteroidal anti-inflammatory drug (NSAID) for general musculoskeletal pain attributed to traumatic injury presuming an active teenage boy lifestyle. His symptoms resolved after two to three days of NSAID.

On further questioning, consuming red meats (mutton and beef) precipitated and aggravated his joint pain and swelling. He did not experience fever, joint stiffness, joint deformity or rashes, and review of other systems were normal. He has a sedentary lifestyle and indulges in food, especially meats. However, he denied smoking behaviour and alcohol consumption. There was no autoimmune or rheumatology disorder diagnosed among his family members. Examination revealed an obese adolescent boy with a body mass index (BMI) of 28kg/m². He was afebrile and his blood pressure was normal. His left first MTP joint was markedly inflamed, warmth with limited movement as compared to the other joints (wrist and interphalangeal). Other systemic examinations were normal.

He was treated symptomatically, similar to previous visits. However, laboratory and radiological examinations were requested due to suspicion of autoimmune rheumatology disorder (recurrent polyarthritis in an adolescent). During review a month later, his symptoms resolved. Other than markedly elevated serum uric acid (889µmol/L), bilateral hand radiographs, renal profile, full blood count, urinalysis and erythrocyte sedimentation rate were normal. Autoimmune markers were not ordered due to resource-limited primary care health clinic in a rural area.

Diagnosis of juvenile gouty arthritis was made, and he was started on a urate-lowering drug (oral allopurinol 150mg once daily) with acute gout prophylaxis (oral colchicine 0.5mg twice daily for six months). Concurrent advice for low purine diet and weight loss lifestyle interventions were also given. He remained asymptomatic in his next visit two months later with a significant reduction of serum uric acid level (500 µmol/L) and some weight loss.

The patient was started on colchicine and then allopurinol and improved. Over the course of time he did begin to complain of pain in his big toe, a more classic presentation of gout. Uric acid levels however, remained high, running

between 11.7 and 13.5 mg/dL over the following year.

The patient was started on colchicine and then allopurinol and improved. Over the course of time he did begin to complain of pain in his big toe, a more classic presentation of gout. Uric acid levels however, remained high, running between 11.7 and 13.5 mg/dL over the following year.

Discussion

Joint pain is a common complaint in paediatric age group with a broad range of differential diagnoses. It is mainly due to overuse and traumatic injury due to children's active lifestyle.⁴ However, joint pain involving multiple sites, persistent or recurrent, and severe may suggest other diagnoses.^{4,5} Gouty arthritis is one of the rheumatological cause for joint pain. It is a rare diagnosis in children, thus complicating and delaying its' diagnosis as demonstrated in this 16-year-old teenage boy.³ Other than uncommon age of presentation for gout, the presence of polyarthritis in this boy (different joints in each consultation) diverted the clinical assessment towards autoimmune diseases such as systemic lupus erythematosus and juvenile idiopathic arthritis. It was during his third visit that diagnosis of juvenile gouty arthritis (JGA) was made, after considering his unhealthy lifestyle, symptoms precipitated by high purine meals which responded to NSAID, obese physique and sites of arthritis.

The gold standard diagnostic test for gouty arthritis is a joint aspiration to look for needle-shaped, negatively birefringent MSU crystals under polarized light microscope.² However, this investigation is impractical in primary care due to its' invasive nature and lack of appropriate training and equipment. Despite SUA level not being a recommended test to diagnose gout, it was helpful in this case by utilizing the American College of Rheumatology and European League Against Rheumatism new classification criteria (Table I).^{3,6} The boy scored eleven points (highlighted in Table I), which can be classified as gout, whereby at least eight points are required.⁶

Table I: The ACR/EULAR gout classification criteria⁵

Criteria	Categories	Score	
C L I N I C A L	Pattern of joint/bursa involvement	Ankle or midfoot (mono-/oligo-)	1
		1 st metatarsophalangeal (mono-/oligo-)	2
	Characteristics of episode(s) ever	One characteristic	1
		Two characteristics	2
		Three characteristics	3
	Time-course of episode(s) ever	One typical episode	1
Recurrent typical episodes		2	
Clinical evidence of tophus	Present	4	
L A B	Serum uric acid level	<4 mg/dl (<240µmol/L)	-4
		6 -<8 mg/dl (360-<480µmol/L)	2
		8-<10 mg/dl (480-<600µmol/L)	3
		≥10 mg/dl (≥600 µmol/L)	4
	Synovial fluid analysis	MSU negative	-2
I M A G E	Imaging evidence of urate deposition	Present (ultrasound DCS or DECT)	4
	Imaging evidence of gout-related joint damage	Present (X-ray gouty erosion)	4

ACR, American College of Rheumatology; EULAR, European League Against Rheumatism; MSU, monosodium urate; DCS, double contour sign; DECT, dual energy computed tomography

Management of gout in a teenager is the same as an adult. The aims are prevention and management of gout flare, which is debilitating, especially for a teenage boy like this case.⁷⁻⁹ Weight reduction, low purine diet and minimal/elimination of alcohol consumption are some non-pharmacological methods advocated to reduce uric acid production. Multiple clinical guidelines and studies emphasize the importance of weight management in obese

gout patients due to its' effectiveness in reducing SUA level and cardiovascular risks.⁷⁻⁹ Zhu et al reported that male gout patients have a nearly four-fold increase of odds to achieve normal SUA level when they lost at least 10kg.⁷ This evidence is a good motivating factor for healthcare providers and this patient to manage his weight problem adequately in achieving ideal BMI.

Although SUA is not a recommended diagnostic test, it functions as a monitoring parameter.³ The target value is the same as the adult's level, which is less than 360 µmol/L.⁷⁻⁹ Below this level, the risk of disabling acute gout attack will be reduced. Weight loss and dietary control per se may not be adequate to lower SUA to the desired level in controlling gouty arthritis; thus, urate-lowering treatment such as allopurinol usually is required⁶. Allopurinol can be started two to four weeks after gout flare, as practiced in this case, to prevent worsening of attack due to sudden lowering of SUA.^{8,9} For the same reason, it is also recommended that colchicine be taken concurrently as a gout attack prophylaxis for six months. The dosage of allopurinol should be started at the lowest possible dose and titrated monthly base on the SUA level. However, if there are breakthrough gout flares, acute management by administering a short course of NSAID or oral steroid will ease the symptoms, and allopurinol can be continued concurrently.^{8,9}

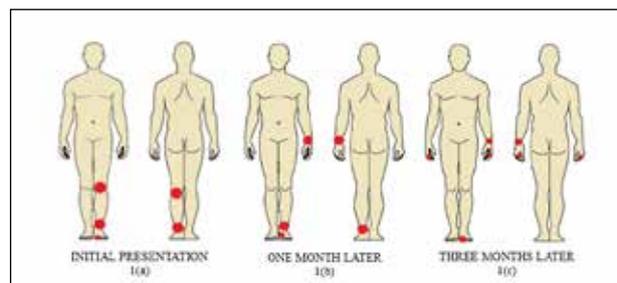


Figure 1: Patient's chronological presentation; (a) Left knee, ankle and first metatarsophalangeal joint (MTPJ); (b) Left wrist, ankle and first MTPJ; (c) Left wrist, first MTPJ and bilateral second proximal interphalangeal joints.

Conclusion

JGA is uncommon. However, increasing prevalence of childhood obesity due to a sedentary lifestyle and unhealthy diet translates into a higher SUA level, which predisposes these children to the risk of gout. A high index of suspicion is required for diagnosis in resource-limited primary care because proper management can prevent recurrent flares and

improve quality of life.

Source of fund: None

Conflict of interest: None

Ethical clearance: Consent from patient's parent and patient's assent obtained. Ethical approval obtained prior submission.

Authors' contribution:

Data gathering and idea owner of this study: Abdul

Rhahman AF, Mohamad NI, Teoh SH

Study design: Abdul Rhahman AF, Mohamad NI, Teoh SH

Data gathering: Abdul Rhahman AF, Mohamad NI, Teoh SH

Writing and submitting manuscript: Abdul Rhahman AF, Mohamad NI, Teoh SH

Editing and approval of final draft: Abdul Rhahman AF, Mohamad NI, Teoh SH

References:

1. Paul BJ, James R. Gout: an Asia-Pacific update. *Int J Rheum Dis.* 2017;**20**(4):407-16.
2. Becker MA, Merriman T. Pathophysiology of gout. In: Dalbeth N, Romain PL, editors. UpToDate. [Internet]. Waltham, MA: UpToDate 2019 [cited July 8, 2019]. Available from: <https://www.uptodate.com/contents/pathophysiology-of-gout>
3. Becker MA, Gaffo AL. Clinical manifestation and diagnosis of gout. In: Dalbeth N, Romain PL, editors. UpToDate. [Internet]. Waltham, MA: UpToDate 2019 [cited July 8, 2019]. Available from: <https://www.uptodate.com/contents/clinical-manifestations-and-diagnosis-of-gout>
4. Kimura Y, Southwood TR. Evaluation of the child with joint pain and/or swelling; In: Sundel R, TePas E, editors. UpToDate. [Internet]. Waltham, MA: UpToDate 2018 [cited July 9, 2019]. Available from: <https://www.uptodate.com/contents/evaluation-of-the-child-with-joint-pain-and-or-swelling>
5. Soomro M, Magsi M, Soomro MA, Akram M, Lahmar O. Patients' knowledge on rheumatoid arthritis presenting with arthralgia in a tertiary care teaching hospital, Pakistan. *Bangladesh J Med Sci.* 2019;**18**(4):808-13.
6. Neogi T, Jansen TLTA, Dalbeth N, Fransen J, Schumacher HR, Berendsen D, et al. 2015 Gout Classification Criteria: An American College of Rheumatology/ European League Against Rheumatism Collaborative Initiative. *Arthritis Rheumatol.* 2015;**67**(10):2557-68.
7. Zhu Y, Zhang Y, Choi HK. The serum urate-lowering impact of weight loss among men with a high cardiovascular risk profile: the Multiple Risk Factor Intervention Trial. *Rheumatology.* 2010;**49**(12):2391-9.
8. Nuki G, Doherty M, Richette P. Current management of gout: practical messages from 2016 EULAR guidelines. *Pol Arch Intern Med.* 2017;**127**(4):267-77.
9. Ministry of Health Malaysia. Clinical practice guidelines: Management of gout. 1st ed. Kuala Lumpur (MY): Ministry of Health; 2008. 25p.