CASE REPORT

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Polyarticular tophaceous gouty arthritis: A case report

Sheikh Javeed Ahmad, Sumyra Khurshid

ABSTRACT

Introduction: Gout is a disorder of purine metabolism, affecting men 40–50 years of age resulting in recurrent bouts of arthritis and subcutaneous tophi in patients with long standing disease. We report a case of a 45-year-old male with symmetric, deforming polyarticular arthritis, affecting all the joints of hands, wrists and feet with diffuse subcutaneous nodules over his body. The radiographic findings of the patient were atypical. Following clinical evaluation and additional investigations, the patient received a diagnosis of chronic tophaceous gouty arthritis mimicking rheumatoid arthritis.

Keywords: Gout, Arthritis, Tophi, Hand, Foot

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INTRODUCTION

Gout is a metabolic disorder of purine degradation pathway usually affecting middle aged and elderly men and postmenopausal females. Usually, it results in acute, monoarticular arthritis, intercritical period and chronic tophaceous gout associated with hyperuricemia and characterized by the presence of monosodium urate (MSU) crystals in connective tissues and kidneys [1, 2]. However, some patients develop chronic polyarthritis mimicking rheumatoid arthritis [3–6]. Both disease entities have been reported to occur in adult population to the extent of approximately 1% [2, 7, 8] with symmetric polyarthritis. Symmetrical presentation or positive rheumatoid factor (RF) can be seen in both the diseases. A few cases of polyarticular tophaceous gout have been reported in literature [9, 10]. We report a rare case of a 45-year-old male with polyarticular tophaceous gout with atypical involvement of hand and feet with disabling effects of untreated hyperuricemia.

CASE REPORT

A 45-year-old male (from Kashmir) was presented to our department of physical medicine and rehabilitation at Sher-i-Kashmir Institute of Medical sciences (Srinagar) a multispecialty hospital with long standing history of pain, swelling and deformity of small and large joints of both hands and feet for approximately 12 years. The patient reported with the history of polyarthralgia which had improved over a week's time. This was followed by frequent intermittent episodes of arthritis of small and large joints of hands, knees, wrists, ankles and feet without morning stiffness. The disease had been treated symptomatically with non-specific non-steroid anti-inflammatory drugs (NSAIDs) leading to the improvement over a period of time. Thereafter, patient developed recurrent episodes of polyarthritis with painless nodules on hand and feet. He was put on allopurinol for 6 months and dietary restriction of protein on account of hyperuricemia with serum uric acid levels of 12.43 mg/dL. Patient intentionally interrupted the treatment following his betterment of health.

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When the patient visited the outpatient department of our clinic on first physical examination, he was conscious, afebrile, and normotensive. Cardiovascular and respiratory system parameters were normal. Physical examination of Locomotor system revealed muscle atrophy of all four limbs and interossei muscles of both hands; multiple deformities of wrists, metacarpophalangeal joints (MCP), proximal interphalangeal joints (PIP) of hands (Figure 1) and metatarsophalangeal joints of feet (Figure 2). Skin examination showed subcutaneous nodule of different sizes, measuring 1–2 cm along metacarpophalangeal joints (MCP) and proximal interphalangeal joints (PIP) of hands (Figure 1) metatarsophalangeal joints feet (Figure 2). These nodules were not showing any signs of inflammation and were fixed to deeper tissues.

Laboratory workup revealed hemoglobin 12.1 g/dL, Leukocyte counts of 4,300/ μ L, platelet counts of 150×10^3 / μ L, ESR of 14/mm 1st hr, uric acid of 14 mg/dL, creatinine of 1.35 mg/dL, rheumatoid factor (RF) negative, blood glucose fasting of 110 mg/dL. Urinary uric acid levels were 1.19 g/24 hr and creatinine clearance of 40 mL/min/1.73 m², C-reactive protein 15 mg/L. X-ray of hands showed narrowing of joint space, subarticular cysts at proximal interphalangeal joint of middle finger of right hand with asymmetric soft tissue swelling (Figure 3). X-ray of feet showed the evidence of narrowing of joint spaces bilaterally with subarticular cysts and overhanging edges. There is complete osteolysis of little toe of right foot (Figure 4). Histopathological examination of nodule removed from left MCP joint revealed presence of uric acid crystals (tophus) with no atypical cells (Figure 5).

Figure 1: Tophaceous nodule on metacarpale and interphalangeal joint of middle figure of right hand.

Figure 2: Tophaceous nodule index toe and little toe.

A diagnosis of polyarticular tophaceous gout was made and patient was treated with colchicine 0.5 mg/day and febuxostat 40 mg/day, which was increased to 1 mg/day and 80 mg/day respectively till acute stage. Currently, patient is showing good response with febuxostat 80 mg/day alone.

Figure 3: Bilateral soft tissue swelling, narrowing of space and subarticular cysts.

Figure 4: Subarticular sclerosis, cysts bilaterally and osteolysis of little toe of right foot.

DISCUSSION

Gout is an inflammatory arthropathy affecting 2.13% of population in United States of America in 2009 [11] with high preponderance for old age, male gender, postmenopausal females and black race [12]. Gout is a metabolic disorder which is characterized by elevation

Figure 5: Monosodium urate crystals of tophi on microscopy.

of uric acid levels above 6.8 mg/dL resulting from impaired renal uric acid excretion. High uric acid level can be attributed to uric acid elevating drugs, genetic predisposition and dietary factors [13]. Gout is a metabolic disorder in which needle shaped crystal of monosodium urate from super saturated fluids are deposited in tissues resulting in gouty arthritis, tophi formation, uric acid nephrolithiasis and renal impairment [14, 15]. The tophi formation usually occurs over a mean period of 10 years [16]. Tophi are present commonly as subcutaneous, sharply circumscribed nodular collection of monosodium urate crystals [17] at periarticular site in and around bursae and in soft tissue overlying tendon and cartilage [18–20]. The most common locations are skin overlying joints and helix of the ears. Rarely, these are found in eyes, nose, larynx, breast and heart valves, penis, spinal cord, tongue, epiglottis [21–31]. Adel and Janitzia have reported location of intradermal tophi on legs, forearm, buttock, abdominal wall, palm and sole [32, 33].

The optimal serum urate level necessary for elimination of tissue deposits of monosodium urate in patients of chronic gout is controversial. Some studies reveal the decrease in serum urate levels achieved by urate lowering drugs and the fast reduction in tophaceous deposition [34]. Surgical treatment is seldom required for gout and is usually reserved for cases of recurrent attacks with deformities, severe pain and joint destruction [35]. The main indication of surgical intervention in gout is sepsis or infection of ulcerated tophi followed by mechanical problems, confirmation of diagnosis and pain control [36]. The radiologic changes in gouty arthritis are asymmetrical, erosive arthritis with preserved articular surface except in late cases. Bone erosions are usually caused by tophi deposits [10].

Almost 30% of patients of rheumatoid arthritis have subcutaneous nodes and all these patients are usually

seropositive. Thus polyarthritis with subcutaneous nodules with negative rheumatoid factor should be investigated for tophaceous gout [37]. Early diagnosis and initiation of early treatment of gouty arthritis will halt the progression of disease to tophaceous state. The current medical management of gouty arthritis has changed a lot. At present management of gout includes non-steroid anti-inflammatory drugs (NSAIDs), colchicines and steroids. The intraarticular steroid has been mainstay to alleviate acute gouty arthritis. It is pertinent to mention interleukin 1 beta inhibitors are the future for amelioration of gouty syndrome. Rilonacept, an interleukin 1 antagonist has proven to be of great advantage in chronic gout and has been helpful in refractory gout but randomized control trails need to be done for its effectiveness.

CONCLUSION

Polyarticular tophaceous gouty arthritis is uncommon considering pharmacological treatment of hyperuricemia and such cases may be considered as differential diagnosis for rheumatoid arthritis so that early treatment will stop the disability effects in such patients.

Author Contributions

Sheikh Javeed Ahmad – Substantial contributions to conception and design, Acquisition of data, Analysis and interpretation of data, Drafting the article, Revising it critically for important intellectual content, Final approval of the version to be published

Sumyra Khurshid – Acquisition of data, Analysis and interpretation of data, Drafting the article, Final approval of the version to be published

Guarantor

The corresponding author is the guarantor of submission.

Conflict of Interest

Authors declare no conflict of interest.

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REFERENCES

1. Cassetta M, Gorevic PD. Crystal arthritis: gout and pseudogout in the geriatric patient. *Geriatrics* 2004;59(9):25–30.

2. Kuo CF, Tsai WP, Liou LB. Rare copresent rheumatoid arthritis and gout: comparison with pure rheumatoid arthritis and a literature review. *Clin Rheumatol* 2008;27(2):231–5.
3. Baker DL, Stroup JS, Gilstrap CA. Tophaceous gout in a patient with rheumatoid arthritis. *J Am Osteopath Assoc* 2007;107(12):554–6.
4. Gomez MM, Carvajal JJD, Pastor MAAC. Gota tofácea: Indisciplina o desconocimiento? *Medifam* 2002;12(4):289–2.
5. Wooten M. Seronegative rheumatoid arthritis and gout. *Clin Rheumatol* 2005;24(1):91.
6. Reginato AJ. Gota e outras artropatias causadas por cristais. In: Kasper DL, Fauci AS, Longo DL, Braunwald E, Hauser SL, Jameson JL. *Harrison. Medicina Interna*. v. 2, 16 ed., Rio de Janeiro: McGraw-Hill Interamericana do Brasil Ltda., 2006, pp. 2146–51.
7. Bachmeyer C, Charoud A, Mougeot-Martin M. Rheumatoid nodules indicating seronegative rheumatoid arthritis in a patient with gout. *Clin Rheumatol* 2003;22(2):154–5.
8. Khosla P, Gogia A, Agarwal PK, Pahuja A, Jain S, Saxena KK. Concomitant gout and rheumatoid arthritis—a case report. *Indian J Med Sci* 2004;58(8):349–52.
9. Schapira D, Stahl S, Izhak OB, Balbir-Gurman A, Nahir AM. Chronic tophaceous gouty arthritis mimicking rheumatoid arthritis. *Semin Arthritis Rheum* 1999;29(1):56–3.
10. Talbott JH, Altman RD, Yü TF. Gouty arthritis masquerading as rheumatoid arthritis or vice versa. *Semin Arthritis Rheum* 1978;8(2):77–114.
11. Brook RA, Forsythe A, Smeeding JE, Lawrence Edwards N. Chronic gout: epidemiology, disease progression, treatment and disease burden. *Curr Med Res Opin* 2010;26(12):2813–1.
12. Neogi T. Clinical practice. Gout. *N Engl J Med* 2011;364(5):443–52.
13. Lee SJ, Terkeltaub RA, Kavanaugh A. Recent developments in diet and gout. *Curr Opin Rheumatol* 2006;18(2):193–8.
14. Touart DM, Sau P. Cutaneous deposition diseases. Part II. *J Am Acad Dermatol* 1998;39(4 Pt 1):527–44.
15. Emmerson BT. The management of gout. *N Engl J Med* 1996;334(7):445–1.
16. Levinson DJ, Becker MA. Clinical gout and the pathogenesis of hyperuricemia. In: McCarty DJ, Koopman WJ, editors. *Arthritis and allied conditions*. 12th ed. Malvern, PA: Lea & Febiger; 1993: 1773–806.
17. Dacko A, Hardick K, McCormack P, Szaniawski W, Davis I. Gouty tophi: a squamous cell carcinoma mimicker? *Dermatol Surg* 2002;28(7):636–8.
18. Grahame R, Scott JT. Clinical survey of 354 patients with gout. *Ann Rheum Dis* 1970;29(5):461–8.
19. Seegmiller JE. Skin manifestations of gout. In: Fitzpatrick TB, Eisen AZ, Wolff K, Freeberg IM, Austen KF, editors. *Dermatology in general medicine*. 4th ed. New York: McGraw-Hill; 1993:1894–900.
20. Moschella SL. Miscellaneous rheumatic disease that can involve the skin. In: Sontheimer RD, Provost TT, editors. *Cutaneous manifestations of rheumatic disease*. Baltimore: Williams & Wilkins; 1995:243–57.
21. Lefkovits AM. Gouty involvement of the larynx. Report of a case and review of the literature. *Arthritis Rheum* 1965;8(5):1019–26.
22. Marion RB, Alperin JE, Maloney WH. Gouty tophus of the true vocal cord. *Arch Otolaryngol* 1972;96(2):161–2.
23. Magid SK, Gray GE, Anand A. Spinal cord compression by tophi in a patient with chronic polyarthritis: case report and literature review. *Arthritis Rheum* 1981;24(11):1431–4.
24. Simkin PA, Campbell PM, Larson EB. Gout in Heberden's nodes. *Arthritis Rheum* 1983;26(1):94–7.
25. Scalapino JN, Edwards WD, Steckelberg JM, Wooten RS, Callahan JA, Ginsburg WW. Mitral stenosis associated with valvular tophi. *Mayo Clin Proc* 1984;59(7):509–12.
26. Gawoski JM, Balogh K, Landis WJ. Aortic valvular tophus: identification by X-ray diffraction of urate and calcium phosphates. *J Clin Pathol* 1985;38(8):873–6.
27. Ferry AP, Safir A, Melikian HE. Ocular abnormalities in patients with gout. *Ann Ophthalmol* 1985;17(10):632–5.
28. Martínez-Cordero E, Barreira-Mercado E, Katona G. Eye tophi deposition in gout. *J Rheumatol* 1986;13(2):471–3.
29. Fenton P, Young S, Prutis K. Gout of the spine. Two case reports and a review of the literature. *J Bone Joint Surg Am* 1995;77(5):767–1.
30. Holland NW, Jost D, Beutler A, Schumacher HR, Agudelo CA. Finger pad tophi in gout. *J Rheumatol* 1996;23(4):690–2.
31. Fam AG, Stein J, Rubenstein J. Gouty arthritis in nodal osteoarthritis. *J Rheumatol* 1996;23(4):684–9.
32. Fam AG, Assaad D. Intradermal urate tophi. *J Rheumatol* 1997;24(6):1126–31.
33. Vázquez-Mellado J, Cuan A, Magaña M, et al. Intradermal tophi in gout: a case-control study. *J Rheumatol* 1999;26(1):136–40.
34. Perez-Ruiz F, Calabozo M, Pijoan JI, Herrero-Beites AM, Ruibal A. Effect of urate-lowering therapy on the velocity of size reduction of tophi in chronic gout. *Arthritis Rheum* 2002;47(4):356–60.
35. Khandpur S, Minz AK, Sharma VK. Chronic tophaceous gout with severe deforming arthritis. *Indian J Dermatol Venereol Leprol* 2010;76(1):69–71.
36. Kumar S, Gow P. A survey of indications, results and complications of surgery for tophaceous gout. *N Z Med J* 2002;115(1158):U109.
37. Bertolo MB, Brenol CV, Schainberg CG, et al. Atualização do consenso Brasileiro no Diagnóstico e Tratamento da Artrite Reumatoide. *Rev Bras Reumatol* 2007;47(3):151–9.

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