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Case Report

Chronic tophaceous gout – A case report

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ABSTRACT

Background: Gout is an inflammatory arthritis associated with functional impairment and impact on quality of life. It arises when the tissues are supersaturated with urate leading to the formation of mono sodium urate crystals in and around the joints. This causes an inflammatory cascade resulting in secretion of several proinflammatory cytokines and neutrophil recruitment into the joint.
Case Report: A 60 years old male patient presented with complaints of swelling in the posterior aspect of left elbow since 2 years and pain since 15 days. Examination revealed soft tissue swellings in multiple joints including metatarsophalangeal joint. Renal function tests show increased Uric acid (8.6mg/dl). On Gross examination, we received fibrofatty mass measuring 3.5x1.5x1 cm. Cut-section showed white flakes ? Calcifications. On microscopy, sections studied show fibrocollagenous tissue with several tophi made up of pink eosinophilic material surrounded by foreign body giant cell reaction and lymphocytes. There are several cystic spaces filled with calcareous debris.
Conclusion: Hyperuricemia is the key risk factor for gout and is considered prerequisite for mono sodium urate crystal formation. Urate under excretion through kidney is the major mechanism for hyperuricemia. Tophus formation is the cardinal feature of advanced gout.

1. Introduction

Gouty arthritis is a condition that presents with devitalizing clinical symptoms and impaired functionality, all which have a significant impact on the quality of life. Initially triggered by the deposition of monosodium urate crystals into the joint space, it results in an inflammatory cascade causing the secretion of several proinflammatory cytokines and neutrophil recruitment into the joint.[1] Normal uric acid levels are between 3.5 to 7.2 mg/dl in adult males and postmenopausal women and between 2.6 to 6.0mg/dl in premenopausal women.[2] However uric acid level of greater than 6.8mg/dl, also known as hyperuricemia, is the key risk factor for gout.[3,4] While it is generally asymptomatic, when the concentration of uric acid exceeds the body threshold, monosodium urate crystals precipitate in the cooler areas of the body which leads to gout.[5] The presentation of gout varies from an acute arthritis manifesting as severe pain and inflammation around the affected joint to a chronic presentation as deposition of uric acid crystals in the joint called tophi. Clinical features of gout include sudden onset arthritis, excruciating joint pain, hyperaemia and rise in local temperature around the involved joint. Pharmacological therapies include uricosuric drugs (eg. Probenicid), xanthine oxidase inhibitors (eg. Allopurinol), urate oxidases (eg. Uricases), NSAIDS, and colchicine. Non pharmacological therapies include life style changes and dietary changes.[6]

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2. Case Report

A 60 years old male patient came with a complaint of swelling at posterior aspect of left elbow joint since 2 years and pain since 15 days. He was a smoker, alcoholic and takes non-vegetarian diet. There is no significant personal, past and family history.

On physical examination, there was a tender swelling noted on the left elbow joint measuring 3.5 x 1.5 x 1cm. Other non tender swellings noted on bilateral first tarso-metatarsal, knee, and right elbow joints. All the swellings are firm in consistency and showed restricted mobility. Clinically the case was diagnosed as Olecranon bursitis and further investigations were done. (Figure 1)

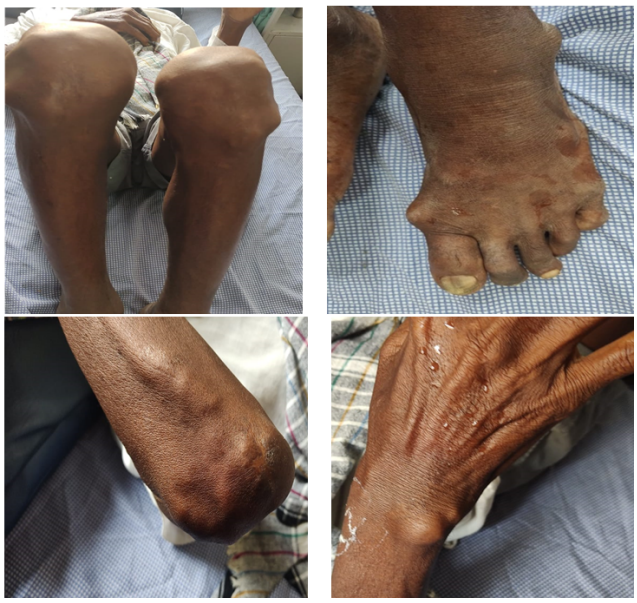

Figure 1: Clinical images of the swellings in different joints

On investigation, Hemoglobin is 9.4 gm/dl, ESR: 80mm/1st hr. Renal function tests showed increased uric acid – 8.6 gm/dl. Ultrasound abdomen showed bilateral Grade 1 Renal parenchymal changes. Intra operatively calcifications were noted over the soft tissue mass and triceps tendon. The mass over triceps tendon was excised and sent for histopathological examination.

On gross examination, we received a fibrofatty mass measuring 3.5 x 1.5 x 1cm. (Figure 2)

Cut section showed white flakes (? Calcifications). (Figure 3)

2.1. Microscopy

Out of our curiosity, we immediately made a wet film and a crush smear from white flakes. Wet film (Figure 4) and crush smear (Figure 5) showed needle shaped crystals.

Hematoxylin and eosin sections showed fibrocollagenous tissue with several tophi made up of

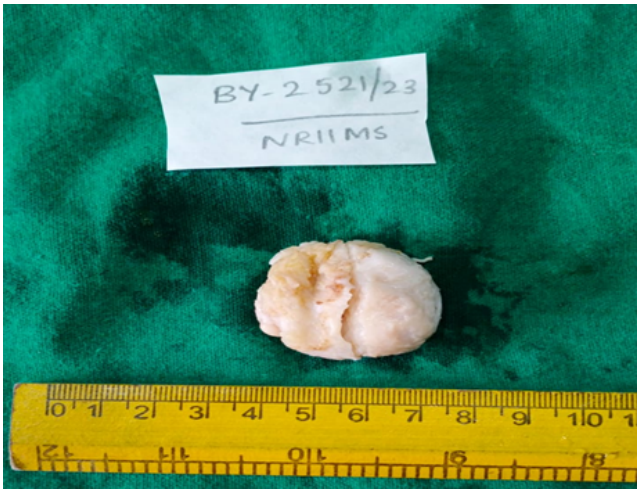

Figure 2: Excised swelling from elbow measuring 3.5 x 1.5 x 1cm

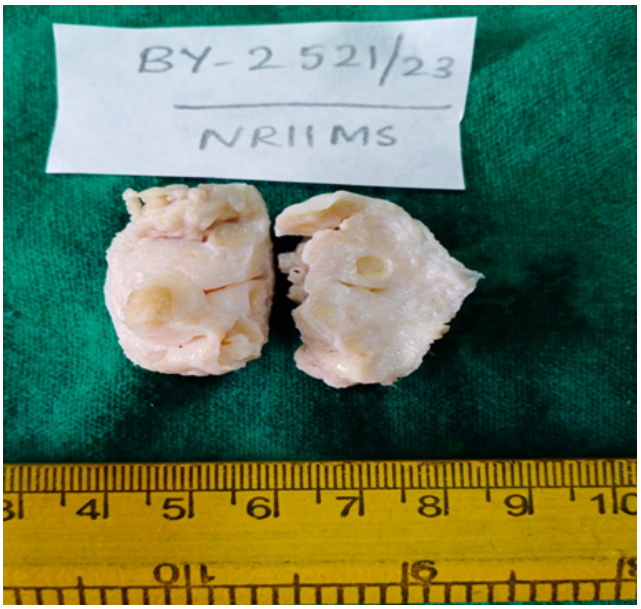

Figure 3: Cut-section showing small spaces with calcified areas

pink eosinophilic material surrounded by foreign body giant cell reaction and lymphocytes. There are several cystic spaces filled with calcareous debris. (Figure 6)

3. Discussion

A tophus is accumulation of large amounts of uric acid crystals in chronic untreated gout. This progresses into destruction of joints with formation of palpable tophi. Tophi leads to joint destruction and deformity.[7] Formation of tophi is the cardinal feature of advanced gout. Microscopically, tophi are foreign body granuloma-like structures containing collections of monosodium urate (MSU) crystals surrounded by inflammatory cells and

Figure 4: Photomicrograph of wet saline mount showing needle shaped crystals. (10X)

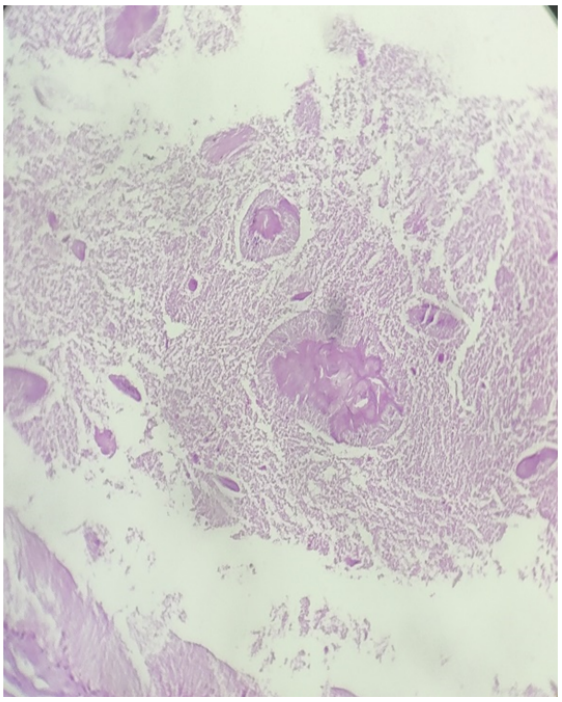

Figure 6: Photomicrograph shows several tophi (H&E,10X)

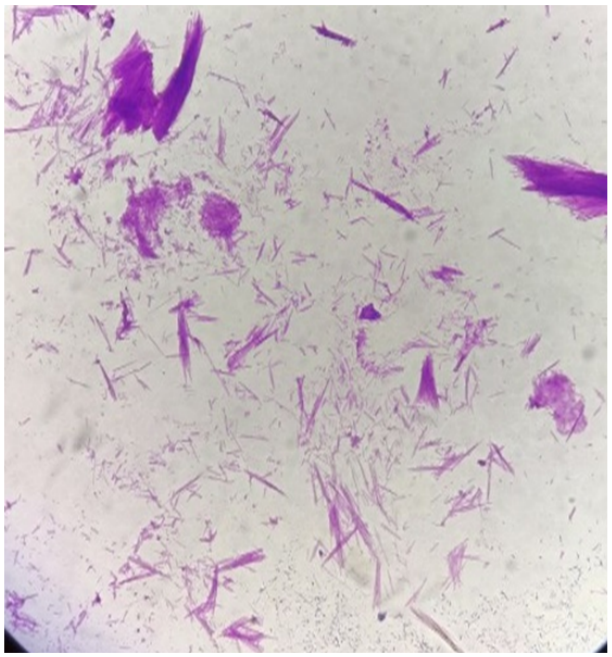

Figure 5: Photomicrograph of crush smear showing needle shaped crystals (Giemsa, 10X)

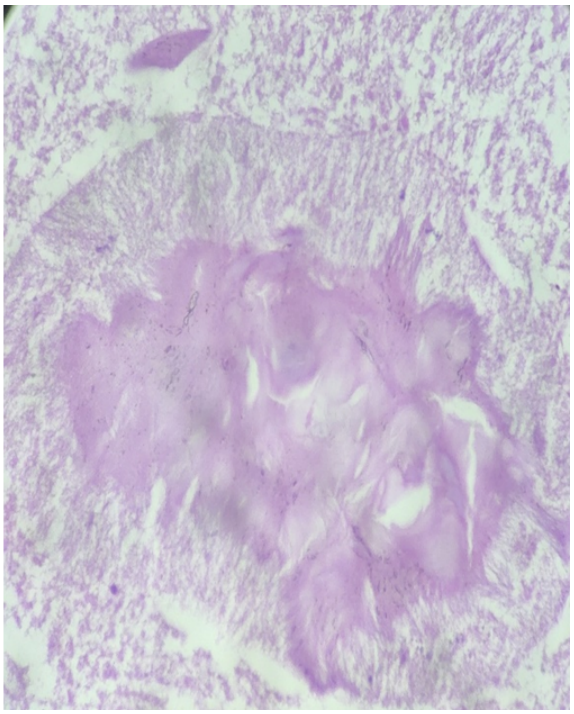

Figure 7: Photomicrograph shows Tophi made up of pink eosinophilic material (H&E,40X)

connective tissue.[8]

In patients with hyperuricemia, the monosodium urate crystals may get deposited in the joints leading to gouty arthritis. This MSU-induced inflammation is driven by components of the innate immune system.[9]

The inflammatory response in the gouty arthritis begins with phagocytosis of MSU crystals by macrophages. This triggers the formation of a protein scaffold known as an inflammasome within the cytosol of the macrophage. The inflammasome helps in activation of pro-IL-1β, which is then secreted from the cell when there is co-stimulation with free fatty acids or lipopolysaccharide.[10,11] So after a large meal or alcohol consumption, there is increase in free fatty acid concentrations in the blood, triggering release of IL-1β, which plays a key role in the development of gouty arthritis flares.[11]

IL-1β regulates cell proliferation, differentiation, and apoptosis. IL-1β also activates IL-1 receptor on endothelial cells which facilitates the transcription of proinflammatory cytokines and chemokines that drive subsequent inflammatory processes.[10,12] This causes neutrophil influx to the synovium which results in further phagocytosis of MSU crystals, IL-1β release and its associated inflammatory processes.[13] The acute inflammation and repeated flares of gouty arthritis result in pathological joint destruction. Prolonged accumulation of MSU crystals gives rise to tophi which on microscopy show crystals in a matrix of lipids, protein, and mucopolysaccharides.[14] IL-1 is a key molecule in the process of bone and cartilage damage and plays a critical role in osteoclast formation.[14]

4. Conclusion

Gout is an avoidable complication of hyperuricemia. This patient was unaware of his disease and presented with chronic tophi for which he had to undergo excision procedures. So educating the patients about the disease, changes in lifestyle such as weight loss in obese individuals, dietary changes to reduce purine intake particularly non vegetarian diet, reduce alcohol consumption and sweetened beverages, and regular exercises to prevent progression and importance of follow-up can decrease morbidity and financial burden on the patient.

5. Source of Funding

None.

6. Conflict of Interest

None.

References

1. Gonzalez EB. An update on the pathology and clinical management of gouty arthritis. *Clin Rheumatol*. 2012;31(1):13–21.
2. Desideri G, Castaldo G, Lombardi A, Mussap M, Testa A, Pontremoli R, et al. Is it time to revise the normal range of serum uric acid levels? *Eur Rev Med Pharmacol Sci*. 2014;18(9):1295–306.
3. Narang RK, Dalbeth N. Pathophysiology of Gout. *Semin Nephrol*. 2020;40(6):550–63.
4. Sinha S, Rijal R, Shah J, Chaudhary P. A Case of Surgically Intervened Chronic Tophaceous Gout and Review of Literature. *J Orthop Case Rep*. 2019;10(1):66–9.
5. Ragab G, Elshahaly M, Bardin T. Gout: An old disease in new perspective - A review. *J Adv Res*. 2017;8(5):495–11.
6. Kumar V, Abbas AK, Aster JC, Perkins JA. Robbins & Cotran Pathologic Basis of Disease. 10th ed. Netherlands: Elsevier; 2018.
7. Chhana A, Dalbeth N. The gouty tophus: a review. *Curr Rheumatol Rep*. 2015;17(3):19.
8. Shi Y, Evans JE, Rock KL. Molecular identification of a danger signal that alerts the immune system to dying cells. *Nature*. 2003;425(6957):516–21.
9. Martinon F, Pétrilli V, Mayor A, Tardivel A, Tschopp J. Gout-associated uric acid crystals activate the NALP3 inflammasome. *Nature*. 2006;440(7081):237–41.
10. Joosten LAB, Netea MG, Mylona E, Koenders MI, Malireddi RKS, Oosting M, et al. Engagement of fatty acids with Toll-like receptor 2 drives interleukin-1β production via the ASC/caspase 1 pathway in monosodium urate monohydrate crystal-induced gouty arthritis. *Arthritis Rheum*. 2010;62(11):3237–48.
11. Pope RM, Tschopp J. The role of interleukin-1 and the inflammasome in gout: implications for therapy. *Arthritis Rheum*. 2007;56(10):3183–8.
12. Schlesinger N, Thiele RG. The pathogenesis of bone erosions in gouty arthritis. *Ann Rheum Dis [Internet]*. 2010;69(11):1907–12.
13. Torres R, Macdonald L, Croll SD, Reinhardt J, Dore A, Stevens S, et al. Hyperalgesia, synovitis and multiple biomarkers of inflammation are suppressed by interleukin 1 inhibition in a novel animal model of gouty arthritis. *Ann Rheum Dis*. 2009;68(10):1602–8.
14. Werina J, Redlich K, Polzer K. TNF-induced structural joint damage is mediated by IL-1. *Proc Natl Acad Sci*. 2007;104(28):11742–7.

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