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Original Research Paper

Orthopaedics

CASE OF GOUTY ARTHRITIS OF RIGHT GREAT TOE

Dr Rajesh K Ambulgekar — H.O.D, Department Of Orthopaedics, Dr S.C.G.M.C Nanded.

Dr Rahul S Agrawal* — Junior resident, Department of orthopaedics, Dr S.C.G.M.C, Nanded. *Corresponding Author

ABSTRACT Gout is a metabolic disorder caused by the deposition of MSU (monosodium urate crystals) in joints and associated inflammatory reactions to it. It usually affects elderly men and postmenopausal women. Chronic tophaceous gout takes usually 10 years to appear due to recurrent episodes of gouty arthritis. Our case is a 50 year male patient with multiarticular tophaceous gouty arthritis. Pharmacological treatment has a high remittance rate. Surgical treatment is rarely required for gout and is usually reserved for cases of recurrent attacks with deformities, severe pain, infection, and joint destruction. Our case is interesting due to the fact that it is early onset and treated only by pharmacological means.

KEYWORDS : gout, uric acid, tophaceous

INTRODUCTION

Gout is a common pathology due to defective uric acid metabolism characterized by a spectrum of presentations such as acute gout, recurrent episodes of inflammatory arthritis, tophaceous soft tissue deposits of monosodium urate crystals, uric acid renal calculi, and chronic nephropathy. Manifestations commonly appear as firm, pink nodules or fusiform swellings [1, 2]. We present a case of multiarticular chronic tophaceous gout in a 50-year-old adult male.

Case Report

Our patient, a 50-year male presented to us with a history of swelling over both feet for 1 year, insidious in onset & gradually progressive. There was a history of discharge of chalky white material from the swelling over right feet which burst into an ulcer. (Figure 1) The swellings developed gradually over a 1-year duration. The patient had no family history but, the patient was a known case of diabetes mellitus & there was a history of high protein diet and alcohol for the past 15 years. He had taken analgesics from multiple hospitals for the condition but it never resolved. He had difficulty walking and performing routine daily activities.

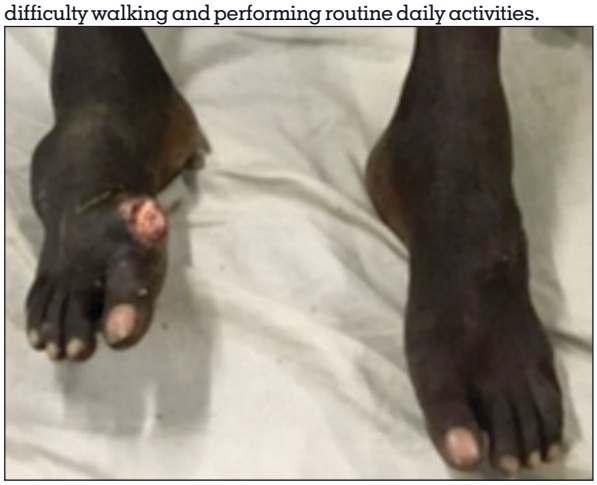

Figure 1. Ulcer over right 1st metatarsophalangeal joint & swelling over both feet.

Blood investigations showed raised serum uric acid levels (10.7 mg/dl) and ESR (20 mm/hr), Serum urea- 145mg/dl, Serum creatinine- 4.1 mg/dl, C-Reactive protein-positive, HbsAg - positive, HIV- negative. Radiographs of the foot showed multiple lytic punched-out lesions at the tarsometatarsal joint, the base of 2nd,3rd,4th metatarsal & multiple tarsal. (Figure 2)

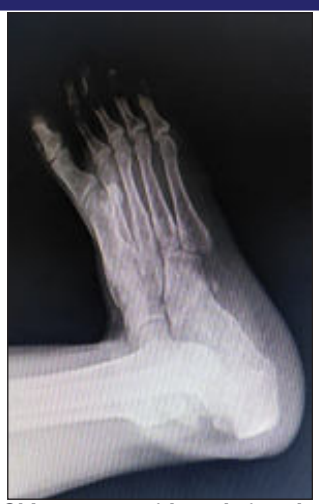

Figure 2. AP & Oblique views of the right foot showing multiple lytic punched-out lesion.

CT Scan of the bilateral ankle with foot showed multiple punched out lytic destruction with overhanging sclerotic margins giving rat bite lesion appearance predominantly in juxta articular location with adjacent calcified soft tissue component with preserved joint space and bony mineralization involving predominantly bilateral first metatarsophalangeal joints & also in bilateral 2nd to 5th tarsometatarsal joints, intertarsal joints, and tibio-talar joints with adjacent tendon & soft tissue involvement likely inflammatory arthritis-gouty arthropathy with tophi formation. (Figure 3)

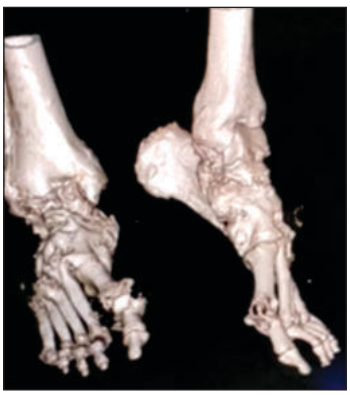

Figure 3. CT Scan Of Bilateral Foot Showing Multiple Punched Out Lytic Lesions.

Plain MRI right foot was done suggestive of multiple large juxta articular erosion associated with extensive mass like lobulated hyperintense synovial and periarticular soft tissue thickening involving multiple joints of foot representing chronic inflammatory arthritis of these joints mostly gouty arthropathy, extra articular soft tissue deposits representing tophi. (Figure 4)

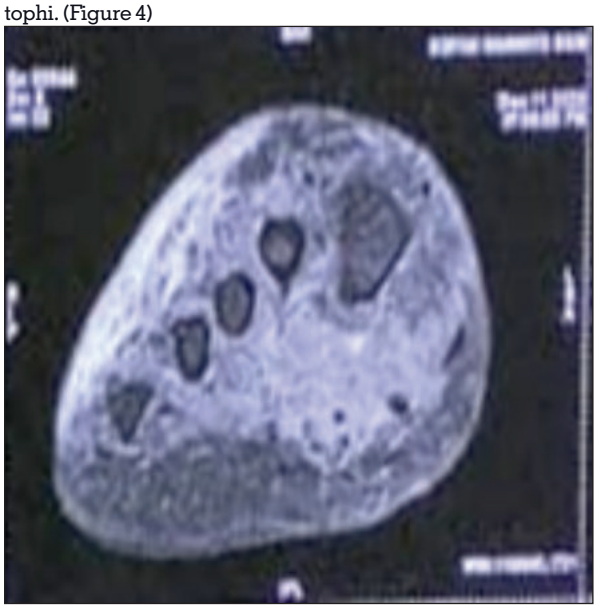

Figure 4. T2 weighted MRI (axial & coronal section) of the right foot showing extra articular soft tissue deposits representing tophi.

Excisional biopsy was done & a sample was sent for histopathology. It showed predominantly needle like crystals in acellular background suggestive of tophaceous gout while pus for culture & sensitivity showed no organism after 72 hours.

USG (abdomen + pelvis) was done & it showed right kidney of size 6.7 x 3 cm & left kidney of size 7.1 x 2.9 cm with bilateral raised cortical echoes with maintained corticomedullary junction.

The patient was put on modified diet with restriction of high protein intake & reduced intake of alcohol with tablet colchicine 1mg/ day in divided doses, short term steroids for a few days in the tapering dose and NSAIDS for a week. It was followed by febuxostat 40 mg/ day for the next four weeks. After 4 weeks uric acid levels came upto 6.7 mg/dl. After 1-month pain reduced gradually and swelling reduced in size. (Figure 5) Patient was able to do his routine daily activities.

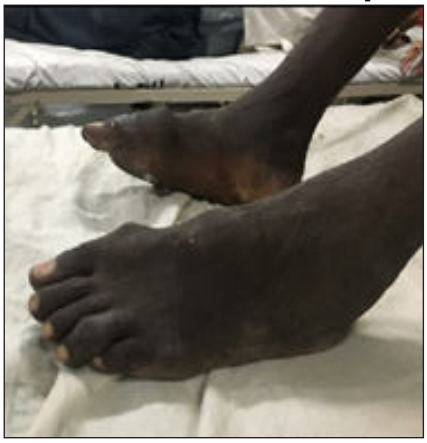

Figure 5. The clinical picture of patient shows healing of ulcer over the right foot with reduced swelling over both feet.

DISCUSSION

Gout is a common disease caused due to abnormal uric acid metabolism. The uric acid gets deposited in the joints due to crystallization resulting in recurrent arthritis. Older age, male sex, postmenopausal women, and black race are at higher risk of developing gout [3] Hyperuricemia and gout can be attributed to uric acid elevating drugs (diuretics, cyclosporine, low doses of aspirin, etc.), genetic Polymorphisms in genes controlling renal urate transport and predisposing dietary factors, such as consumption of red meat, seafood, and alcohol [4] Gout usually starts with affecting the first metatarsophalangeal joint of the foot and less commonly other joints. In untreated and traditionally treated patients tophaceous gout may develop, which is characterized by chronic destructive polyarticular involvement and tophi. Chronic tophaceous gout frequently occurs after 10 years or more of recurrent polyarticular gout [5] Tophi can occur in soft tissue and different organs either in the presence or in absence of gouty arthritis. The prevalence of gout is much higher in men than in women and rises with age due to the uricosuric action of estrogen. In our case, history was quite short with extensive tophi in hands and feet occurring within 4 years of onset of arthritis. Kim et al. reported disseminated miliarial gout in a 34-yearold male, which occurred 4 years after the onset of gouty arthritis [6] . Jung et al. reported a case of disseminated gout in the form of nodules that appeared after 10 years of gouty arthritis [7]. Gout can affect the large joints which can be confused with rheumatoid arthritis and calcinosis cutis. However, it can be differentiated neurologically, biochemically, and radiologically. Treatment of Gout should include dietary modification as well as pharmacological measures. Nonpharmacological measures include diet restriction of purines, reducing intake of alcohol, and reducing weight. An acute attack is treated by NSAIDs, colchicines, or steroids. Normalization of hyperuricemia is the aim of treatment in chronic tophaceous gout. Drugs like probenecid favoring excretion of uric acid or allopurinol and febuxostat for inhibiting the production of uric acid are used. Surgical removal of tophi is attempted only in the case of severe pain, joint deformities, or physical removal of tophi [8].

Pathophysiology

Gout is an inflammatory disease characterized by the deposition of uric acid crystals in and around joints, subcutaneous tissues, and kidneys. Although men and women are equally affected by gout, men are six times more likely to have serum urea concentrations above 7 mg per dL. Gout typically occurs during middle age and is uncommon before the age of 30 years old. Women rarely have gouty arthritis attacks before menopause.

Clinical Presentation

Gout attacks are usually associated with precipitated events. Patients usually present with rapid onset of severe pain, swelling, redness, and warmth in one or totwooints. This pain and inflammation are caused by an inflammatory response. Acute attack untreated attacks usually last two to 21 days depending on cases. There are four clinical stages of gout according to the National Institute of Arthritis and Musculoskeletal and Skin Diseases (NIAMS). The first stage is known as asymptomatic hyperurecemia. During this stage, the patients can have an elevation of uric acid in the blood but they do not have any symptoms yet. After more and more urate deposits around a joint and if any trauma triggers the release of crystal into the joint space, patients will suffer acute attacks of gout. This second stage is known as acute gouty arthritis. The third stage, known as interval or intercritical gout, involves the interval between acute flare gout attacks with persists crystals in the joints. When crystals deposits continue to accumulate, patients are likely to develop chronically stiff and swollen joints. This stage is called chronic tophaceous gout. Some permanent damage to affected joints and

sometimes to kidneys can be seen. This advanced stage is relatively uncommon if patients receive proper treatment.

Differential Diagnosis

Gout in the elderly is often polyarticular and involves upper extremity joints (especially proximal interphalangeal joints and distal interphalangeal joints). Women present 70% of the time with a polyarticular disease rather than the classic monoarticular arthritis seen in men. Gout can be mistaken for rheumatoid arthritis because tophi may resemble rheumatoid nodules and rheumatoid factors often become weakly positive as people age. It may be difficult to differentiate cellulitis or septic arthritis from gout, particularly when a fever, leukocytosis, redness, or desquamation is present. The term pseudogout, for calcium pyrophosphate deposition disease, believes the difficulty in clinically differentiating it from gout. For a definitive diagnosis, joint fluid must be aspirated for culture and a search for urate crystals.

Diagnostic Test

The gold standard diagnostic test for gout is an arthrocentesis. The American College of Rheumatology has established 12 clinical criteria, 6 of which a patient must have for diagnosis.

* Maximum joint inflammation within 1 day
* More than one attack over time
* Monoarticular arthritis (although gout can be polyarticular)
* Redness of joint
* Great metatarsophalangeal pain or swelling
* Unilateral great metatarsophalangeal involvement
* Unilateral tarsal involvement
* Suspected tophus
* Hyperuricemia
* Asymmetrical swelling within the joint on x-ray
* Subcortical cysts without erosion on x-ray
* Joint fluid culture negative for organisms during the attack

Treatment and Management

The gold of treating gout is to minimize or eliminate the urate crystals from the joints and other structures associated with them. Several aspects must be taken into consideration and each treatment regiment is varied from patient to patient. The three main objectives that FNP take into consideration are a treatment for acute attacks, prophylaxis against recurrent attacks, and management of hyperuricemia.

Asymptomatic hyperuricemia: urate-lowering drugs are not recommended to treat patients with asymptomatic hyperuricemia. If hyperuricemia is identified, underlying causes such as obesity, hypercholesterolemia, alcohol consumption, and hypertension should be addressed.

Acute gout: NSAIDs are being used as first-line therapy. Indomethacin (Indocin), ibuprofen (Motrin), naproxen (Naprosyn), sulindac (Clinoril), piroxicam (Feldene) are also effective against gout.

Corticosteroids:

intra-articular, intravenous, intramuscular, or oral corticosteroids are effective in acute gout. When one or two joints are involved, intra-articular injection of corticosteroid can be used.

Intramuscular triamcinolone acetonide is as effective as indomethacin in relieving acute gouty arthritis. Triamcinolone acetonide is especially useful in patients with contraindication to NSAIDs.

Oral prednisone: is an option when repeat dosing is anticipated. Prednisone, 0.5 mg per kg on day 1 and tapered by 5 mg each day is effective.

Colchicine is also an effective treatment for acute gout. However, the majority of patients experience gastrointestinal side effects, including nausea, vomiting, and diarrhea.

Patient Education

If they are obese, they should be advised to begin a concerted program of supervised weight reduction, but to avoid starvation or very low-calorie diets that may only exacerbate the risk of gout. Drinkers should be warned against binges. Maintenance of good hydration needs to be stressed to those at risk for nephrolithiasis. On the other hand, patients will find it comforting to know that severe dietary restrictions are unnecessary. Fasting should be avoided because it may precipitate an attack. The importance of treating an acute attack at the first sign of illness also needs to be stressed. For the patient with interval gout, a discussion of the risks and benefits of prophylactic therapy and the importance of compliance is indicated. Those taking allopurinol should be warned of the risk of a hypersensitivity reaction and advised to cease intake immediately and call the physician at the first sign of a rash, fever, or other manifestation. Pain management is the primary concern during the acute phase of an attack. The patient should be advised to take analgesic medications as scheduled. The joint should be rested as much as possible in a position of comfort. Ice, not heat, may help with reducing discomfort.

CONCLUSION

Gout is one of the most common cause of acute monoarticular arthritis. Primary gout runs in families and follows multifactorial inheritance. The expanded use of agents that decrease uric acid excretion has significantly increased the incidence of secondary gout. The Framingham Study suggested that almost half of new cases were associated with thiazide use.

Consent: Written informed consent of the patient has been taken before publishing the case report.

Conflicts Of Interest: There are no conflicts of interest.

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