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Diuertic induced hyperuricemia presenting as malignant gout in a 19-year old male with moderate rheumatic mitral stenosis

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A 19-year old male presented with swelling of multiple joints of upper and lower limb. On detailed evaluation, he was diagnosed as rheumatic moderate mitral stenosis 1 year back. He was receiving frusemide-40 mg, spirinolactone- 50 mg, metoprolol-50 mg, and erythromycin-250 mg twice daily for past one year. As he was minimally symptomatic, he lost the follow up. Once he noticed his swelling which was gradually progressive, and nontender, he attended the clinic for evaluation. During evaluation, he was diagnosed as secondary gout as it was frusemide induced. Drug was stopped, and allopurinol- and urate lowering agent was started. On follow-up, his swelling started regressing.

Gout, an inflammatory arthritis, is caused by accumulation of monosodium urate crystals (MSU) in joints and soft tissues when serum uric acid concentrations rise above the physiological saturation limit (≥ 6.4 mg/dl) [1]. Imaging tools and clinical presentation together helps in establishing the diagnosis, in absence of histological diagnosis and also helps in assessing the burden of disease.

The acute form of the gout is characterized by sudden onset intense pain, swelling, warmth and erythema of multiple joints. The great toe is characteristically affected, however almost all joints may be affected. During an acute phase of gout, soft tissue swelling and effusions may be seen by conventional radiography, however these findings are nonspecific.

Chronic tophaceous gout results from chronic hyperuricemia. Continued deposition of MSU crystals leads to increased frequency of acute attacks, progressive shortening of intercritical phase and development of tophi due to crystal deposition in soft tissues, bones and joints. The typical radiological findings in chronic tophaceous gout, which differentiate it from other inflammatory arthritides, include well defined, ‘punched-out’ erosions with overhanging edges, soft tissue nodules (tophi), calcification of tophi and asymmetric involvement [3] (Fig.). Radiographic damage is virtually always present in patients with subcutaneous tophi [5]. The erosions are typically extra-articular, but may be intra-articular or para-articular. The joint space is usually preserved until late in the disease and there is lack
of periarticular osteopenia. The most common site affected is the first metatarsophalangeal (MTP) joint, followed by the fifth MTP joint, mid-foot, hand and wrist.

The gold standard for diagnosis of gout is demonstration of negatively birefringent, needle-shaped MSU crystals in tissue or synovial fluid by polarizing microscope. Obtaining a histological diagnosis is not always feasible, and whilst application of international consensus definitions may assist in the diagnosis of gout in the absence of a crystal diagnosis, at times a definitive clinical diagnosis can be difficult [2]. These radiological changes are fairly diagnostic, having a specificity of 93% using clinical diagnosis as the gold standard [4]. The radiographic changes are usually delayed but in our case, it became apparent within nine months. Therefore, the lag to developing radiographic limits its role in the diagnosis or monitoring of this disease [3].

The proximal tubule is the major site of urate handling; both secretion and reabsorption occur in this segment, with the net effect being reabsorption of most of the filtered urate. Hyperuricemia is a relatively common finding in patients treated with a loop or thiazide diuretic and may, over a period of time, contribute to new-onset gouty arthritis or, more promptly, recurrence of established gout. Loop and thiazide diuretics decrease urate excretion by increasing net urate reabsorption. Asymptomatic hyperuricemia does not require any treatment. If diuretic-induced gout where patient is symptomatic, a urate-lowering drug such as allopurinol should be started.

**Key words:** Thiazide diuretic; Monosodium urate; Malignant gout; Rheumatic mitral stenosis; Tophi

**References**


**Figure Legend**

**Figure 1.** Photograph showing both the feet, and hands showing joint swelling and tophi (white arrow)

**Figure 2.** Fluoroscope in anteroposterior (AP) view of the feet where multiple punched out juxta-articular erosions with overhanging edge (red arrow) with the relatively preserved joint space (white arrow) are visible in the 1st metatarsophalangeal (MTP) and interphalangeal joint. Also visible is subchondral bone density (white square) involving the 1st MTP and interphalangeal joint. Multiple soft tissue tophi are also visible (white asterix)

**Figure 3.** Fluoroscope in anteroposterior (AP) view of the both hands showing subchondral deposition (white asterix), and associated erosive changes appearing as multiple punched out lesions (red arrow). Multiple soft tissue tophi are also visible (white arrow)