



UNUSUAL PRESENTATION OF GOUT

Rheumatology

Dr. Lohitha Malipeddi 2nd year resident Department of General Medicine, Sree Balaji medical college and Hospital, Chennai.

Dr. K Shanmuganadan* Professor Department of General Medicine, Sree Balaji medical college and Hospital, Chennai. *Corresponding Author

Dr. Suresh Kanna Assistant professor Department of General Medicine, Sree Balaji medical college and Hospital, Chennai.

KEYWORDS

INTRODUCTION :

Gout is a metabolic disease, which is characterized by acute or chronic arthritis, and deposition of monosodium urate crystals in joint, bones, soft tissues, and kidneys. Gout can either manifest as acute arthritis or chronic arthropathy, which is also called tophaceous gout. The prevalence of gout and hyperuricemia is on the rise in developing countries probably related to population aging, alcohol intake, hypertension, obesity, metabolic syndrome.

CASE REPORT:

50 year old male known alcoholic and diabetic and hypertensive on medication presented with complaints of pain in both knees for last one year, aggravated since last 3 days. No other comorbidities. Vitals were stable. On local examination, tenderness of knee with local raise of temperature are present. Systemic examination found to be unremarkable. Investigations revealed total WBC count of 11,800 cells/cumm, HbA1C of 8.5%, serum uric acid of 7.8mg/dl, CRP of 4.8mg/dl, ESR of 72mm and anti CCP is negative. Ultrasound of bilateral knee joints shows effusion noted in suprapatellar region extending into joint spaces of both knees with reactive synovial thickening, features suggestive of inflammatory arthritis. Diagnostic arthrocentesis was performed on both the knees, and revealed negatively birefringent needle shaped crystals using polarized microscopy in both samples. Patient is managed with intra-articular triamcinolone, inj. methyl prednisolone 80mg, tab. colchicine 0.5mg bd, tab. medrol 16mg od, insulin and antibiotics. Patient improved symptomatically and hence, patient is discharged. On follow up patient was symptom free with serum uric acid of 5.8mg/dl.

Ultrasound Bilateral Knee- features suggestive of inflammatory arthritis

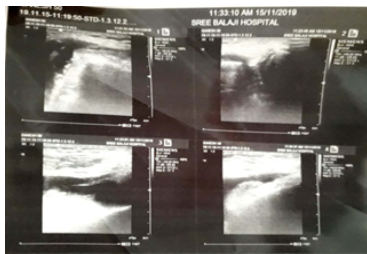


Table 1: Synovial fluid analysis.

Test	Value	Normal
Clarity	Translucent	Transparent
Color	Yellow	Clear
WBC (per mm ³)	1635	< 200
PMNs (%)	54	< 25
Gram stain	No organisms	No organisms
Culture	Negative	Negative
Total protein (g/dL)	1.7	3.1
LDH (IU/L)	386	105 – 333
Glucose (mg/dL)	57	70–110
Crystal	Monosodium urate crystals	None

DISCUSSION :

Gout is a metabolic disease that can manifest as acute or chronic arthritis, and deposition of urate crystals in joint. It can be due to excessive excretion of uric acid, crystal formation, or systemic inflammatory state [2,3]; however, exact mechanism is still unknown. A diagnosis of gout is most accurate when supported by visualization of uric acid crystals in a sample of joint or bursal fluid, or demonstrated histologically in excised tissue. Synovial fluid analysis of our patient was consistent with inflammatory arthritis. Mild leucocytosis in this patient was due to systemic inflammatory response.

Visible or palpable tophi are usually noted only among those patients who are hyperuricemic and have had repeated attacks of acute gout, often over many years. However, presentation of tophaceous deposits in the absence of gouty arthritis is also reported [1,4]. Pain and inflammation are manifested when uric acid crystals activate the humoral and cellular inflammatory processes [5].

During an acute illness, if systemic inflammatory state prevails, such as in an acute infection, cytokines and chemokines triggers inflammation and cause arthritis in the presence of urate crystals [6,7]. Phagocytosis of these crystals by macrophages in the synovial lining cells precedes influx of neutrophils in the joint. This process releases various mediators of inflammation locally [8,9].

Hyperuricemia is often present in patients with tophaceous gout, and they can benefit from uric acid lowering therapy early during the course [10,11].

Our patient presented with pain in bilateral knee joint as an initial presentation of gout, which is very rare, but has been reported.

Investigational studies due to acute elbow joint pain deciphered the underlying mystery of chronic swelling. Systemic inflammatory response secondary to diverticulitis exposed the joints to the effects of urate.

First-line treatments for an acute flare are either oral colchicine and/or non-steroidal anti-inflammatory agents. Systemic or intra-articular corticosteroids can also be used, and are equally effective, but with more side effects [12,13]. Interleukin-1 inhibitors are still under investigation, and are not approved for an acute attack of gout [14].

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