Int J Burn Trauma 2025;15(4):171-176
www.IJBT.org /ISSN:2160-2026/IJBT0163538

Case Report

From suspected joint tuberculosis to gouty arthritis: a diagnostic journey

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Received January 24, 2025; Accepted August 11, 2025; Epub August 15, 2025; Published August 30, 2025

Abstract: Gout is a metabolic disorder characterized by hyperuricemia, leading to the deposition of monosodium urate crystals in joints and soft tissues. It commonly affects the first metatarso-phalangeal (MTP) joint, but atypical presentations can pose significant diagnostic challenges. In this report, we describe a rare case of gouty arthritis affecting the proximal interphalangeal (PIP) joint of the second toe, which was initially suspected to be a case of joint tuberculosis. A 38-year-old male presented with a painful swelling over the second toe for two months, with imaging and laboratory findings suggesting an infectious etiology. Despite clinical suspicion of tuberculosis, histopathological examination of the lesion confirmed the presence of amorphous eosinophilic material with chronic inflammatory infiltrate and giant cell reaction, indicative of gout. Subsequent serum uric acid evaluation revealed hyperuricemia, leading to a definitive diagnosis. The patient underwent surgical evacuation of the tophaceous deposits followed by medical management with colchicine, nonsteroidal anti-inflammatory drugs (NSAIDs), and allopurinol. Postoperative follow-up demonstrated complete resolution of symptoms with no recurrence. Aim of the study: This case report aims to highlight the diagnostic challenges of atypical gout presentations, particularly when mimicking infectious conditions such as tuberculosis. It underscores the importance of maintaining a broad differential diagnosis, utilizing histopathology for confirmation, and considering gout even in uncommon anatomical locations.

Keywords: Gout, atypical presentation, PIP joint, second toe, tuberculosis mimicry, hyperuricemia

Introduction

Gout is an inflammatory arthritis caused by the deposition of monosodium urate (MSU) crystals in joints and periarticular tissues due to chronic hyperuricemia. It has been historically referred to as "The Great Mimicker" because of its ability to resemble various musculoskeletal and infectious conditions, including joint infections, soft tissue tumors, and inflammatory arthritis [1, 2]. While the first metatarso-phalangeal (MTP) joint is the most commonly affected site, atypical presentations in the wrist, shoulder, and small joints of the hands and feet have been reported [3]. These atypical manifestations pose significant diagnostic challenges, especially when gout mimics infectious conditions such as joint tuberculosis. The diagnosis of gout relies on a combination of clinical features, laboratory investigations, and imaging studies. The gold standard for diagnosis is the identification of negatively birefringent monosodium urate (MSU) crystals in synovial fluid or tophaceous deposits under polarized light microscopy [4]. However, in cases where joint aspiration is not feasible, The American College of Rheumatology (ACR)/European League against Rheumatism (EULAR) 2015 Gout Classification Criteria is used [5].

The distinction between gout and tuberculous arthritis is critical due to their overlapping clinical and radiological features [6], while both conditions can present with chronic monoarthritis, several factors help in differentiating them:

1. Etiology and risk factors: Gout being more common in individuals with metabolic syndrome, obesity, chronic kidney disease, and high-purine diets [7] while tubercular arthritis are usually associated with immunosuppression, malnutrition, or prior tuberculosis exposure [8].

https://doi.org/10.62347/PBRZ2450

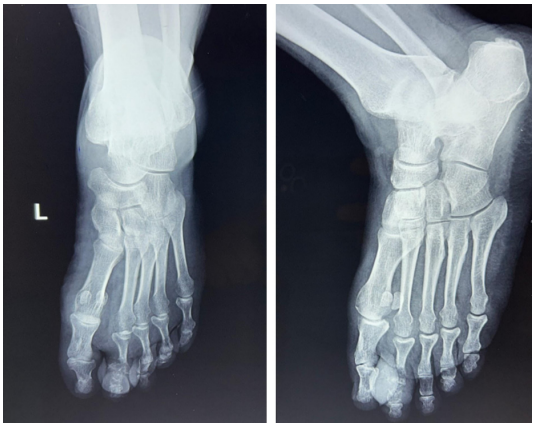

Figure 1. 38-year male, presented with swelling over 2nd toe left foot. X-ray showing bony destruction of the head of the proximal phalanx with cortical erosion of the base of the middle phalanx and complete collapse of the PIP joint.

2. Clinical presentation: Gout commonly present as acute, recurrent attacks of severe pain, swelling, and erythema, often resolving spontaneously. Chronic cases may show tophi formation while in tubercular arthritis; chronic, insidious onset with progressive swelling and stiffness, often without acute inflammatory symptoms is the usual complaint.

3. Laboratory and synovial fluid analysis: Presence of negatively birefringent MSU crystals in synovial fluid is typically found in gout but in tuberculosis there is presence of Mycobacterium tuberculosis in synovial fluid culture or positive PCR (CBNAAT). The fluid is usually lymphocyte-predominant with high protein and low glucose levels [9].

4. Imaging features: Gout typically presents as punched-out erosions with overhanging edges; preserved joint space early in the disease while periarticular osteopenia, joint space narrowing, and subchondral bone destruction ("Phemister's triad") is usually found in tubercular arthritis [10].

5. Histopathology: Amorphous eosinophilic deposits with foreign body giant cell reaction is frequently found in gout while in tuberculosis, granulomatous inflammation with caseating necrosis and Langhans giant cells is mostly present [11].

In this report, we present a unique case of gouty arthritis involving the proximal inter-phalangeal (PIP) joint of the second toe, initially misdiagnosed as joint tuberculosis. The rarity of this presentation, coupled with radiological findings suggestive of an infectious process, led to diagnostic uncertainty. A definitive diagnosis was only achieved after histopathological confirmation of MSU crystal deposition. This case highlights the importance of considering gout in the differential diagnosis of chronic arthritis, even in atypical locations, and underscores the role of histopathological examination in challenging cases.

Case presentation

A 38-year male came to our clinic with complaints of painful swelling over second toe of the left foot over a period of 2 months (**Figure 1**). The patient had no record of trauma, insect bite, loss of weight and appetite or previous similar attacks. However, there was history of on and off fever unrecorded for the last 2 weeks. There was no history of gout or any chronic illness in the family. Nutritionally, patient was obese with unhealthy dietary pattern. On examination, a spherical swelling involving PIP joint was seen with marked joint deformity. The lump was firm to hard and painful measuring 3×4 cm. No local rise of temperature and visible skin changes were observed. Furthermore, there was painful limitation of the ROM of PIP joint. Laboratory investigations included CBC, urea and electrolyte, ESR, CRP and urine analysis. All were normal except ESR which was marginally raised (27 mm/hr). Moreover, the rheumatoid factor was also negative. X-ray of the foot showed bony destruction of the head of the proximal phalanx with cortical erosion of the base of the middle phalanx and complete collapse of the PIP joint. MRI showed bony destruction of adjacent proximal and middle phalanx which was isointense on T1 images and hyperintense on PDFS images. There was also minimal collection and edema on the dorsal aspect of the second toe which was more in favour of infection (**Figure 2**). Core needle biopsy was done and the collected sample was sent for histopathological examination, gram stain, AFB stain and culture, cartridge based nucleic acid amplification test (CBNAAT) and fungal culture. It was advised that the patient return for further evaluation with all the test reports after 2 weeks. Following a two-week

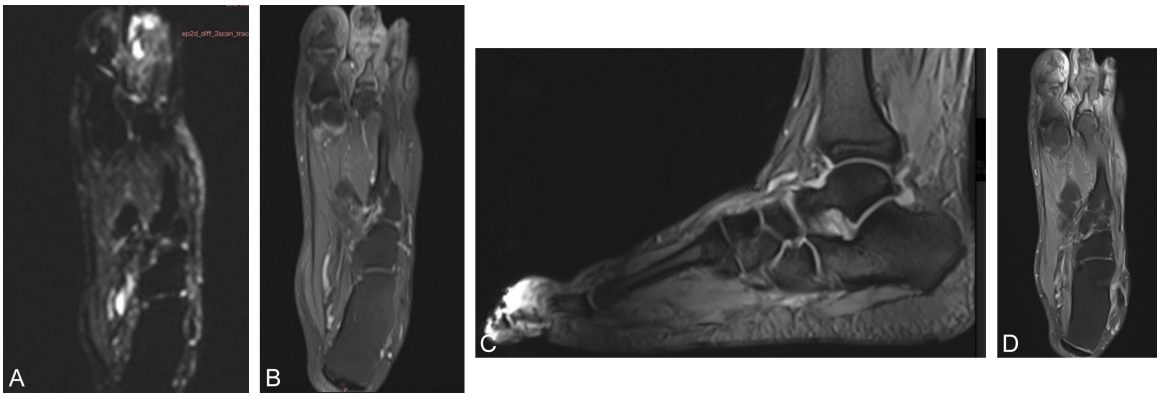

Figure 2. A-D. Altered signal intensity area involving PIP joint of the 2nd toe with destruction of adjacent proximal and middle phalanx appearing T1 isointense and PD hyperintense showing contrast enhancement with minimal collection and adjacent edema on the dorsal aspect of the 2nd toe.

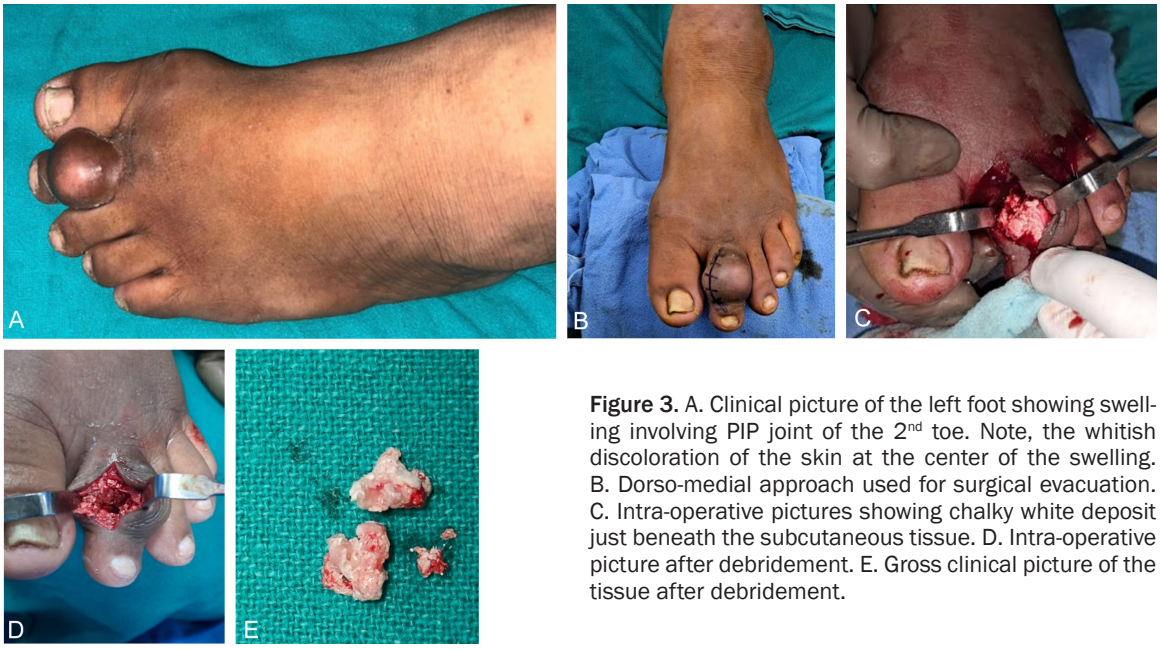

Figure 3. A. Clinical picture of the left foot showing swelling involving PIP joint of the 2nd toe. Note, the whitish discoloration of the skin at the center of the swelling. B. Dorso-medial approach used for surgical evacuation. C. Intra-operative pictures showing chalky white deposit just beneath the subcutaneous tissue. D. Intra-operative picture after debridement. E. Gross clinical picture of the tissue after debridement.

period, the patient came back to our clinic with heightened pain in the swelling. Assessment revealed the swelling to be tender and warm with increase in its size. There was whitish discoloration of the skin over dorsolateral aspect however there was no discharge (**Figure 3A**). On further evaluation, all the tests were normal (no growth) except HPE which showed amorphous eosinophilic material surrounded by chronic inflammatory infiltrate with giant cell reaction favouring the possibility gout (**Figure 4**). After biopsy report, we checked the uric acid level which was found to be elevated (11.3 mg/dl) [Normal: 3.5-7.2 mg/dl]. Based on the severity of symptoms, surgical evacuation of the swelling was planned under tourniquet and digital block. After incising the subcutaneous tissue, chalky white deposits were found oozing out from the wound. After thorough debridement, wound was washed and primarily closed (**Figure 3B-E**). For stabilization of the PIP joint, one intramedullary K-wire was placed. The chalky white deposits were sent for polarised microscopy examination which validated the existence of monosodium urate crystals. Postoperatively, apart from intravenous antibiotic, which was given for one day, we started tablet colchicine 500 microgram thrice daily, NSAIDS (ibuprofen) 600 mg four times a day and tablet allopurinol 300 mg once a day. At 2 weeks after

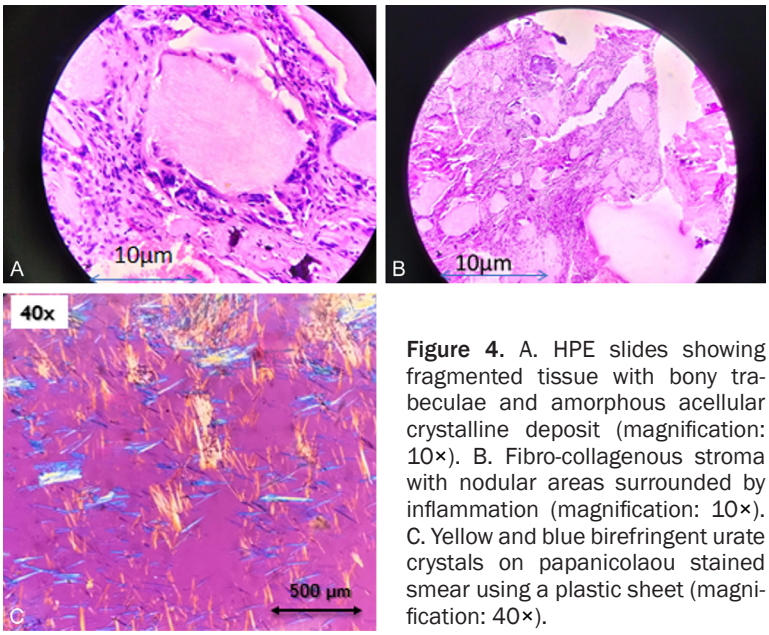

Figure 4. A. HPE slides showing fragmented tissue with bony trabeculae and amorphous acellular crystalline deposit (magnification: 10×). B. Fibro-collagenous stroma with nodular areas surrounded by inflammation (magnification: 10×). C. Yellow and blue birefringent urate crystals on papanicolaou stained smear using a plastic sheet (magnification: 40×).

surgery, stich removal was done and at 4 weeks, K-wire was removed (**Figure 5**). At 2 months of follow-up, the medications were discontinued and the swelling largely subsided and further surgical intervention was not required. At the 6 months follow-up, the patient reported no recurrence of symptoms with complete subsidence of pain (**Figure 6**).

Discussion

Gout is a metabolic disorder caused by the deposition of monosodium urate (MSU) crystals in joints and soft tissues due to persistent hyperuricemia. While it commonly affects the first metatarso-phalangeal (MTP) joint, atypical presentations such as involvement of the proximal inter-phalangeal (PIP) joint - are rare and can pose significant diagnostic challenges [1]. This case highlights the complexity of diagnosing gout in an unusual location, where clinical and radiological features initially suggested an infectious etiology, particularly joint tuberculosis.

In the present case, the patient presented with painful swelling over the second toe for two months, without a history of trauma, weight loss, or constitutional symptoms which was against our diagnosis of tuberculosis. However, the initial diagnosis of joint tuberculosis in this case was based on multiple factors. India is endemic for tuberculosis, making tuberculous arthritis a common consideration in chronic monoarthritis. The patient's clinical presentation of a chronic, painful swelling over the proximal interphalangeal (PIP) joint of the second toe, along with X-ray findings of bony destruction with cortical erosion, strongly suggested an infectious etiology, particularly tuberculosis. Additionally, gouty arthritis rarely affects the second toe, further supporting an alternative diagnosis. Given the endemic nature of tuberculosis and the atypical site for gout, tuberculous arthritis was the most plausible initial diagnosis, necessitating further investigations to confirm the etiology. However, histopathological examination ultimately confirmed the presence of eosinophilic amorphous deposits surrounded by chronic inflammatory infiltrate and giant cell reaction, consistent with gouty arthritis.

This case underscores the importance of considering gout in the differential diagnosis of chronic arthritis, even in the absence of classic podagra or tophi. Clinicians should maintain a high index of suspicion for gout when evaluating persistent joint swelling, particularly in patients with risk factors such as obesity and an unhealthy diet, as seen in this case. A definitive diagnosis can only be made through synovial fluid analysis or histopathological examination, as imaging alone may not differentiate gout from other inflammatory or infectious arthropathies [4].

The American College of Rheumatology (ACR)/ European League against Rheumatism (EULAR) 2015 Gout Classification Criteria provides a structured approach to diagnosing gout [5]. The most definitive test is the identification of negatively birefringent MSU crystals in synovial fluid or tophaceous deposits. However, in cases where synovial aspiration is not feasible or the diagnosis remains uncertain, serum uric acid levels, radiographic findings, and histopathology can aid in confirming the diagnosis.

Key diagnostic considerations for atypical gout include: (1) Imaging findings: While X-rays may reveal rat-bite erosions and overhanging ed-

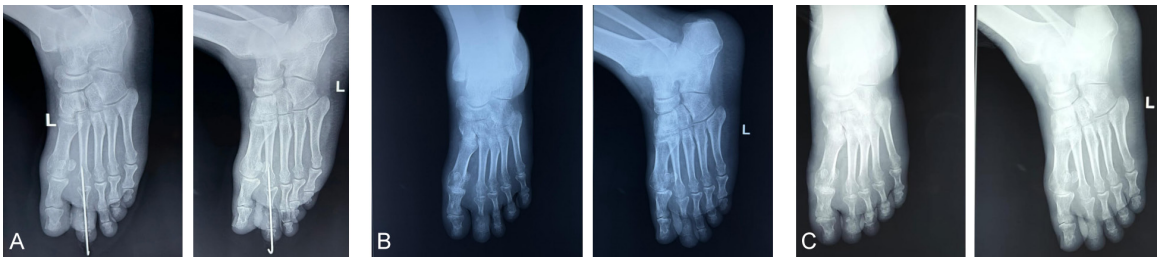

Figure 5. Post-operative X-ray at day 1 (A), at 6 weeks (B), at 6 months (C).

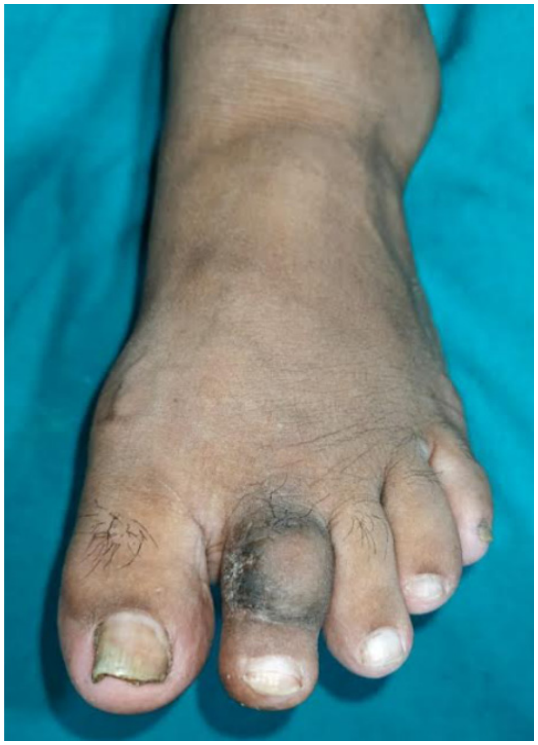

Figure 6. Clinical picture at 6 months showing regression of the swelling.

ges, they are often indistinguishable from other chronic arthritis. Ultrasound (showing the double contour sign) and dual-energy CT (DECT) can enhance diagnostic accuracy [6, 17]. (2) Laboratory markers: Although hyperuricemia supports a diagnosis of gout, it is neither sensitive nor specific, as some patients may have normal serum uric acid levels during an acute attack [7]. (3) Biopsy and histopathology: In cases of diagnostic uncertainty, a biopsy remains the gold standard for distinguishing gout from infectious or neoplastic conditions.

Management of gouty arthritis should be tailored based on disease severity, presence of tophi, and patient comorbidities. Treatment involves a combination of pharmacological therapy, lifestyle modifications, and surgical intervention in selected cases. For acute attack management, first-line therapy includes NSAIDs (e.g., ibuprofen, naproxen) or colchicine (0.5 mg, thrice daily) to reduce inflammation [12, 15]. Corticosteroids (oral or intra-articular) are used in patients with contraindications to NSAIDs or colchicine. Allopurinol (300 mg daily) or febuxostat is recommended for patients with recurrent gout or tophi. The target serum uric acid level should be maintained below 6 mg/dL to prevent future attacks [13]. Lifestyle modifications, including dietary changes (reducing red meat, alcohol, and fructose-rich beverages), weight loss, and increased hydration, play a crucial role in preventing recurrence [14, 18]. In cases of severe joint destruction, recurrent tophaceous deposits, or functional impairment, surgical debridement or joint stabilization procedures may be required [11, 16]. In our case, the patient underwent surgical evacuation of tophi, which resulted in complete resolution of symptoms.

Conclusion

This case emphasizes the diagnostic complexity of atypical gout presentations, especially when mimicking infectious arthritis. A systematic approach, incorporating clinical evaluation, imaging, synovial fluid analysis, and histopathology, is essential for accurate diagnosis. Treatment should focus on rapid symptom relief, long-term uric acid control, and, when necessary, surgical intervention. Clinicians must remain aware of unusual gout presentations to avoid misdiagnosis and ensure timely management.

Learning points

1. Gout has potential to resemble various conditions. 2. Gouty tophi can appear indepen-

dently in any part of the musculoskeletal system. 3. Biopsy is crucial for confirming the diagnosis.

Disclosure of conflict of interest

None.

Abbreviations

PIP, Proximal inter-phalangeal joint; MTP, Metatarsophalangeal joint; MSU, Monosodium urate crystals; EULAR, European League Against Rheumatism; ACR, The American College of Rheumatology; PCR/CBNAAT, Polymerase chain reaction/Cartridge based nucleic acid amplification test; ROM, Range of motion; CBC, Complete blood count; ESR, Erythrocyte sedimentation rate; CRP, C-reactive protein; HPE, Histopathological examination; DECT, Dual energy computed tomography; PDFS, Proton density fat saturated.

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