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CASE REPORT

A Tale of Two Diagnoses; Gout Vs Chronic Osteomyelitis. A Case Report

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Abstract

Gout can potentially be diagnosed clinically and treated, if classical symptoms are present. In some cases, gout and osteomyelitis can have similar presenting signs and symptoms and it may be difficult to differentiate just on clinical presentation, routine laboratory workup and imaging like radiography or ultrasound. Arthrocentesis can be crucial in such scenarios to differentiate the two entities as missed opportunity to treat infectious etiology can have detrimental outcomes. We present a case of patient with ankle pain and swelling treated as recurrent gout, as there were no risk factors for osteomyelitis. Arthrocentesis confirmed the diagnosis of osteomyelitis and patient was treated with intravenous antibiotics, resulting in resolution of symptoms.

Keywords: Gout, Osteomyelitis, Chronic osteomyelitis, Joint pain

1. Introduction

The presence of bone pain prompts many differentials, and the history, clinical presentation, and lab evaluation are critical to making a precise diagnosis. Two common causes of bony pain are Gout and Osteomyelitis (OM). Both have different predisposing factors and patient histories. Although Gout patients tend to have a classic picture of clinical symptoms and risk factors across the board, this is not the case with chronic OM, which is prone to having a variable presentation depending upon the cause.[1] Features can sometimes overlap with Gout if the cause is through closed injury mechanisms.[2] We present a case of a chronic OM, treated as recurrent gout. A delay in diagnostic investigations like arthrocentesis can have adverse outcomes.

2. Case description

A 57-year-old patient with no known prior comorbid conditions was evaluated for left ankle pain and swelling for over a month at a local walk-in clinic. He first noticed symptoms following a workout session and attributed it to an ankle sprain. Conservative measures such as icing, elevating his foot, and taking Ibuprofen did not provide much relief. He had diffuse tenderness throughout the ankle joint, along with mild edema, mild erythema, and pain on plantar and dorsiflexion, and inversion and eversion. A three-view x-ray of the foot and ankle revealed no obvious acute abnormalities or degenerative changes (Fig. 1). The patient was treated for gout with Methylprednisolone and Diclofenac Sodium for 5 days. He revisited the walk-in clinic 10 days later for persistent symptoms. Physical exam showed persistent left ankle edema, tenderness to palpation, and mild to moderate stiffness with range of motion. Laboratory workup showed white blood cell (WBC) count of 9.6×10^3/uL (4–10.5 10^3/uL), uric acid (UA) of 4.7 mg/dL (3.5–7.2 mg/dL), erythrocyte sedimentation rate (ESR) of 42 mm/h (<20 mm/h), and C-reactive protein (CRP) 16.1 mg/dL (<0.9 mg/dL). Repeat left

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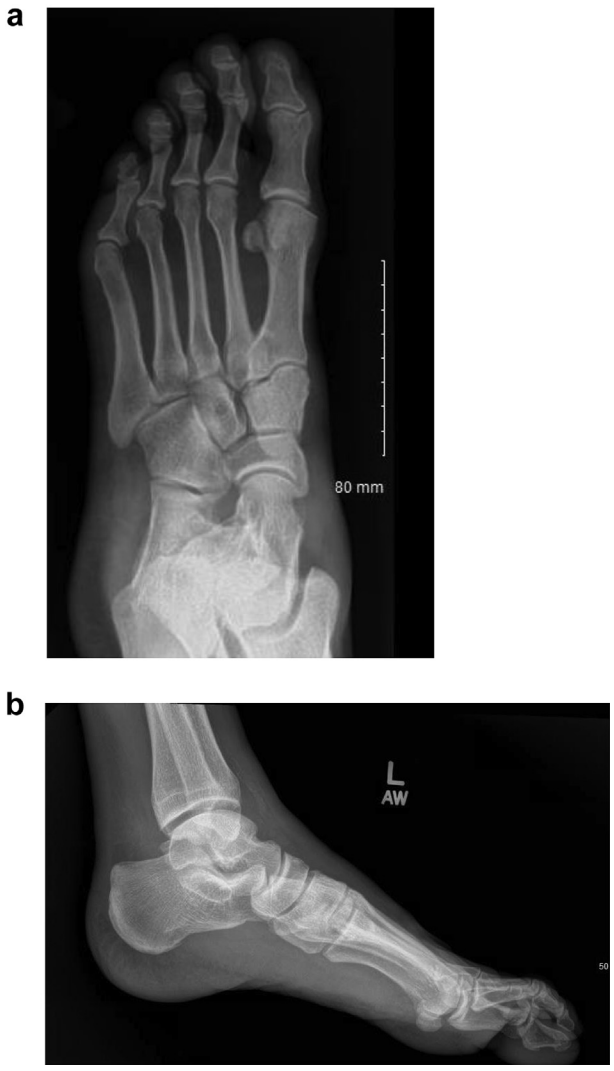

Fig. 1. (a and b): No acute fracture or dislocation was seen on AP and lateral view of left foot and ankle x-rays.

ankle X-rays were reported without an acute abnormality. He was treated with Indomethacin 50 mg along with a Prednisone taper dose for gout again. After initial improvement, there was a sudden worsening of symptoms. On a third visit to a different walk-in clinic, a joint needle aspiration was performed, synovial fluid was negative for crystals while culture grew Methicillin Sensitive Staphylococcus Aureus (MSSA). The patient was admitted to the hospital and started on intravenous (IV) Ceftriaxone. Left ankle arthrotomy with synovectomy and debridement was done and specimens for bone histopathology and cultures were sent. Gram stain was negative for organisms and WBCs. Surgical specimens of the left ankle revealed gross findings of 2.5 cm aggregate of tan rubbery tissue, diagnosed as prominent granulation tissue associated with acute and chronic inflammation, and variable fibrinoid material and necrosis on histopathologic analysis (Fig. 2). The sample from the left tibia revealed 0.5 cm aggregate of tan white spongy material, probable bone, diagnosed as bone showing reactive changes and features of chronic OM (Fig. 3). Culture grew MSSA. Infectious diseases consulted and antibiotic coverage was switched to IV Cefazolin every 8 h for six weeks. He had resolution of symptoms upon completion of therapy. Inflammatory markers normalized and he had no ambulatory dysfunction.

3. Discussion

OM is defined as the infectious inflammation of the bone and medullary cavity, which can have an acute or chronic presentation. It accounts for more than 50,000 hospitalizations in the United States annually.[3] Inoculation of pathogenic organisms can occur via traumatic mechanism, spread contiguously from an adjacent infection, or hematogenously. The pediatric patient population accounts for most cases of hematogenous OM, whereas trauma is usually the culprit behind adult OM.[2] The most common organism, found in most cases of chronic OM is *Staphylococcus Aureus*.[4] Major risk factors for OM are penetrating trauma, IV drug use, diabetes, immunocompromised state, local soft-tissue infection, vascular insufficiency, and open and closed fractures, with the former being more common.[2,5] The pathophysiology of the development of acute hematogenous OM comprises bacterial inoculation into the body, which then adheres to either bone or implant, subsequently causing infection, with the potential to become chronic.[6] Acute OM presents with local inflammatory signs such as erythema, local tenderness, and swelling, along with systemic symptoms such as fever while chronic OM has nonspecific symptoms such as chronic pain, draining sinuses, malaise, fever, and poor wound healing.[7]

Gout is caused by the deposition of monosodium urate (MSU) crystals in tissues and joints, present with swelling, pain, or tenderness in a joint or bursa.[8] There is significant overlap of the major symptoms associated with both crystal arthritis and septic arthritis.[9] Presence of urate crystals in joint fluid is needed to confirm the diagnosis of Gout, although it can be diagnosed clinically as well if the criteria is met,[10] and a high index of suspicion and familiarity with its various presentations is required to make an astute diagnosis. Early recognition and treatment of septic arthritis is important as destruction of cartilage can begin within days of the onset of infection.[9,11] Routine laboratory investigations and

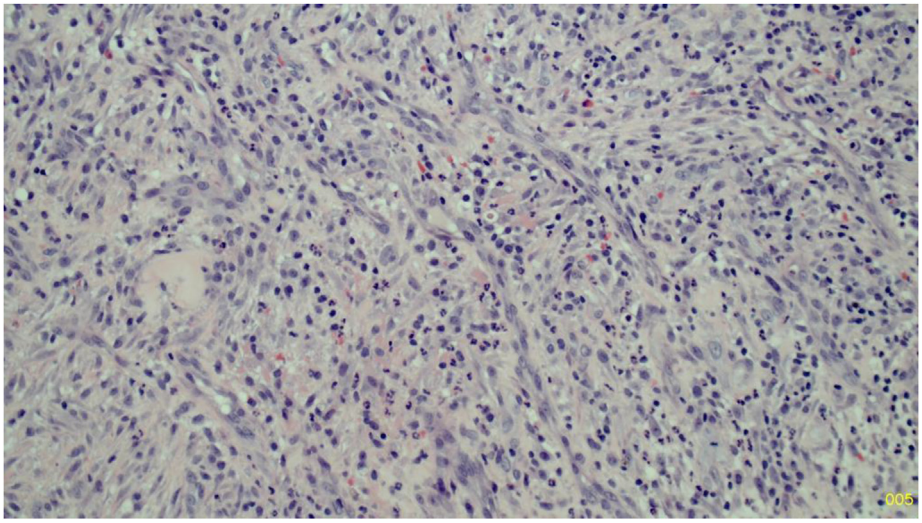

Fig. 2. Left ankle histopathology specimen from debridement showing acute and chronic inflammatory changes.

inflammatory markers such as CRP and ESR lack diagnostic specificity for either condition.[11,12] An elevated leukocyte count is only seen in less than 50 % of patients with septic arthritis, making it an unreliable indicator.[11] Serum UA level is not used as a diagnostic test as it is often normal, even during an acute attack.[8] Plain film radiography findings for gouty arthritis may not appear until 1 year of uncontrolled disease. Both Dual Energy Computed Tomography (DECT) and radiography are not accurate in detecting gout in its early stages. However, as the disease progresses further, DECT has the ability to calculate the composition of deposits seen, helping it detect MSU accurately. For acute cases, Ultrasound is the preferred method of imaging in point-of-care rheumatology.[13,14] Magnetic Resonance Imaging (MRI) is also helpful in showing cartilage damage, soft tissue inflammation, and bone erosion.[14] Synovial fluid or tophus analysis showing negatively birefringent crystals is the definitive test for Gout, but currently it is only performed in cases of uncertain diagnosis.[8]

In OM, radiography and MRI findings are either normal or non-specific in early disease, making them both unreliable for the purpose of diagnosis.[9] The imaging studies in chronic OM may demonstrate bone sequestra and/or sinus tract formation on X-ray, computed tomography (CT), or MRI, and a positive Ga67 uptake bone scan.[15] Because initial imaging for both diseases are either unreliable or

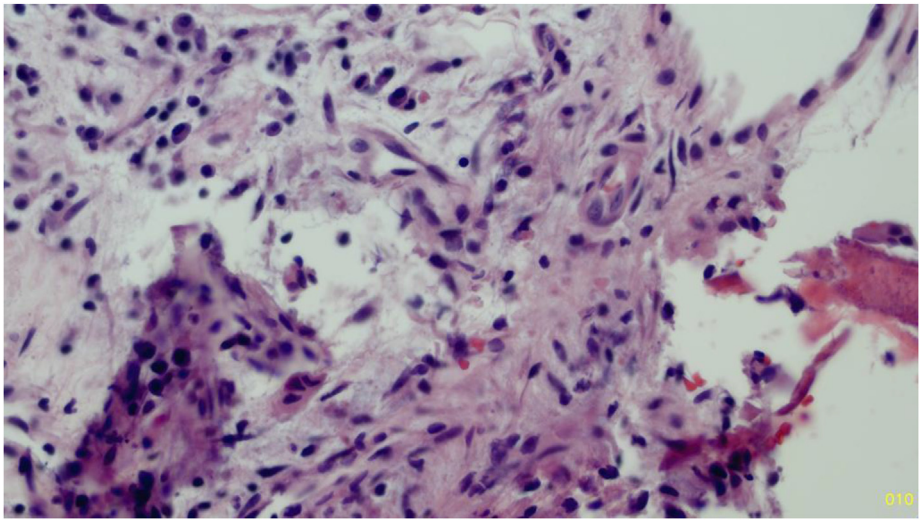

Fig. 3. Biopsy specimen from the left tibia showing segments of bone with reactive features and chronic osteomyelitis including chronic inflammation with plasma cells.

nonspecific, the diagnostic distinction is critical to make, as timely and appropriate treatment can be life-saving. A low threshold for arthrocentesis in cases where imaging studies are normal and laboratory workup is non-conclusive, can confirm the diagnosis and appropriate for septic arthritis and/or OM can be initiated.

Treatment of chronic OM initially involves establishment of the causative organisms and their antibiotic susceptibilities. Along with a crucial four to six weeks of antibiotic treatment, surgical debridement and in some cases, stabilization of the involved bone, are also key components of the treatment. Initial antibiotic treatment after obtaining cultures is started with Nafcillin and either Cefotaxime or Ceftriaxone. Upon becoming available, culture results determine the need for a revision in treatment. MSSA or coagulase-negative Staphylococci are treated with antibiotics like IV Nafcillin or Clindamycin Phosphate, or first-generation Cephalosporins or Vancomycin as alternatives. Methicillin resistant S. Aureus (MRSA) is primarily treated with IV Vancomycin, although alternatives are available.[16] OM is challenging to treat and requires extensive therapy both surgically and medically. It is prone to latency and recurrence, hence making the prolonged antibiotic therapy along with extensive surgical debridement necessary.[17] OM disease course lasting more than three months, the presence of bone exposure, and a treatment regimen not including surgical debridement with muscular flap, run the risk of osteomyelitis relapse.[15]

4. Conclusion

There is a significant overlap in symptoms of gout and septic arthritis/OM. Clinicians should maintain a low threshold for synovial fluid analysis and cytology, especially if the presenting symptoms are sub-acute or chronic, and if symptoms persist or worsen after initial course of treatment. Early diagnosis and treatment initiation is the key to better outcomes.

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Informed consent

Obtained. IRB approval available upon editor's request.

Data availability

All the essential data is included in the draft.

Author contributions

As follows.

Mashal Imtiaz Khan MD: Primary Author. Case description and literature review, and referencing.

Wajiha Ali MD: PGY-II. Data Collection and contributed to discussion.

Ali Javed MD: PGY-II. Contributed to discussion, review and editing.

Saadia Haleema MD: Review of pathology slides and review and editing.

Yasir Ahmed, MD: Supervised the entire process, from conception to finalizing the draft. Literature search, figures' description, referencing, and review and editing and final draft for publication.

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Conflicts of interest

There is no conflict of interest.

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