CASE REPORT

Monoarticular gouty arthropathy of the acromioclavicular joint: a rare manifestation

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SUMMARY

Gouty arthritis is a common cause of inflammatory arthropathy affecting joints of the extremities. However, involvement of the acromioclavicular joint is almost unheard of and only a handful were reported in the literature. If not approached prudently, inaccurate diagnosis could lead to suboptimal management. A thorough history taking correlating a positive past episode of gout, ultrasound findings and joint aspiration contribute to the diagnosis of gouty arthritis involving atypical joint. Like gout of any joint, medical treatment is the cornerstone of management, while surgical drainage is reserved for specified cases only.

INTRODUCTION

Gout is a disease due to the accumulation of monosodium urate crystal that predominantly affects synovial joints, although it does have an extraarticular predilection such as subcutaneous tophi and urolithiasis. Known risk factors include genetic, obesity, high alcohol consumption, high purine diet, diuretic usages and comorbidities such as hypertension and chronic kidney disease. As the burden of non-communicable diseases such as hypertension and obesity are on the rise, in addition to the purine-rich dietary practices, gout has become a common inflammatory arthritis we frequently see in our day-to-day clinic.

Rarely does gout involve the acromioclavicular joint.[1] Like gout of any other joints, there are a variety of other diagnoses that can masquerade as an acromioclavicular gouty arthritis. Since they share common signs of joint inflammation, diagnosis of gouty arthritis relies on a detailed history taking. Small joint aspiration may be challenging but detection of monosodium urate crystal on microscopy is confirmative. Ultrasonography examination is useful to elicit characteristic features of gout, though it requires both a skilful operator and a capable device.[2]

In all cases of arthritis, it is always pertinent to rule out septic arthritis as the delay in its treatment may result in devastating consequences. Urgent drainage and joint washout are indicated for septic arthritis, but surgical treatment for gout tends to be associated with poor wound healing hence only recommended in indicated cases such as infection or ulceration.[3] Here, we describe our encounter and management of an enigmatic case of a monoarticular acromioclavicular joint pain that was initially suspicious of septic arthritis and eventually confirmed to be a tophaceous gouty arthritis.

CASE PRESENTATION

A 68-year-old man presented with a painful swelling over his left shoulder of 1 month duration. The pain occurred insidiously, throbbing in nature and aggravated primarily upon shoulder motion. He denied any preceding trauma, fever or any other articular involvement. He visited a health clinic a fortnight before and was treated for an immature abscess. He completed a 2-week course of oral cloxacillin with some symptom abatement, albeit temporarily. He has underlying hypertension, hyperlipidemia, ischemic heart disease and stent placement on aspirin and life-long warfarin. He also gives positive history suggestive of gouty arthritis involving the great toes, which he self-manages with over-the-counter analgesics only. His last gouty attack was 3 years ago.

On clinical examination, there was a small, localized, firm swelling over the left acromioclavicular region (Figure 1(a)) with no overlying erythema, punctum or discharge. It was mildly tender, not warm, and non-fluctuant. His shoulder motion was quite preserved. There were no gouty tophi involving other joints. Plain radiograph examination of the left shoulder appeared unremarkable (Figure 1(b)). We initially treated him as an exacerbation of left acromioclavicular joint osteoarthritis. His concurrent antibiotics were discontinued, and he was given a review in 2 months with some analgesia.

However, he presented again after 2 days with worsening localized symptoms of pain and swelling. Examination showed increased swelling, tenderness and now, erythema. Biochemical investigation revealed normal white counts of 10.9×10^9/L, elevated erythrocyte sedimentation rate of 87 mm/hour and C-reactive protein of 66.2 mg/L. Bedside sports ultrasound using a *Philips Lumify L12-4* linear transducer demonstrated a large, well-circumscribed swelling overlying the anterior and superior aspect of the left ACJ, suggesting a possible effusion with mixed echogenic substance enclosed within the distended lesion with the absence of increased

This article was accepted: 20 November 2023
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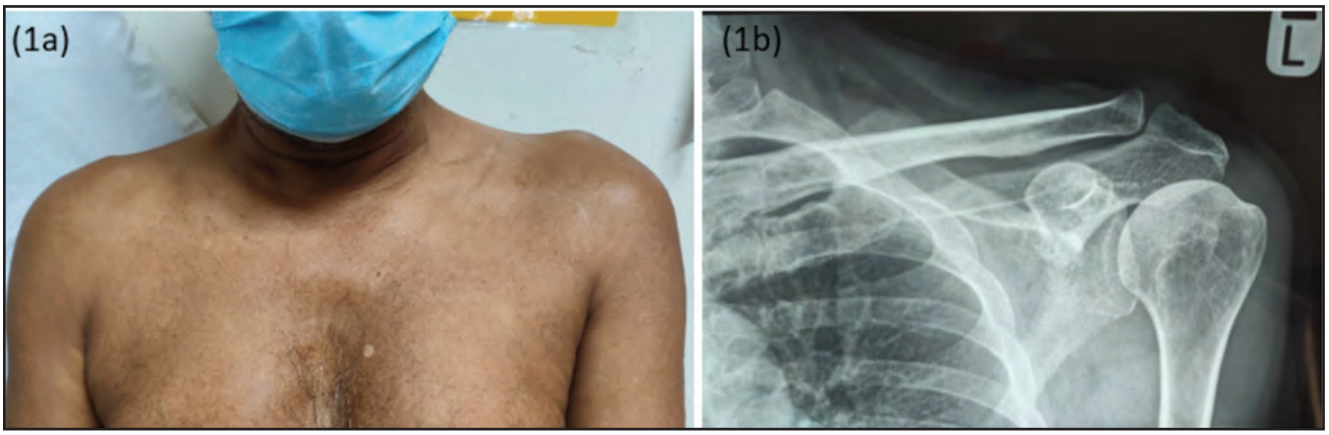

Fig. 1: (A) A localised swelling over the left ACJ with no overlying erythema.* (B) Plain radiograph of the left shoulder with no discernible intra- or peri-articular abnormality. There was a mild increase in soft tissue shadow over the ACJ.
*Patient's consent is obtained prior to publication of this article.

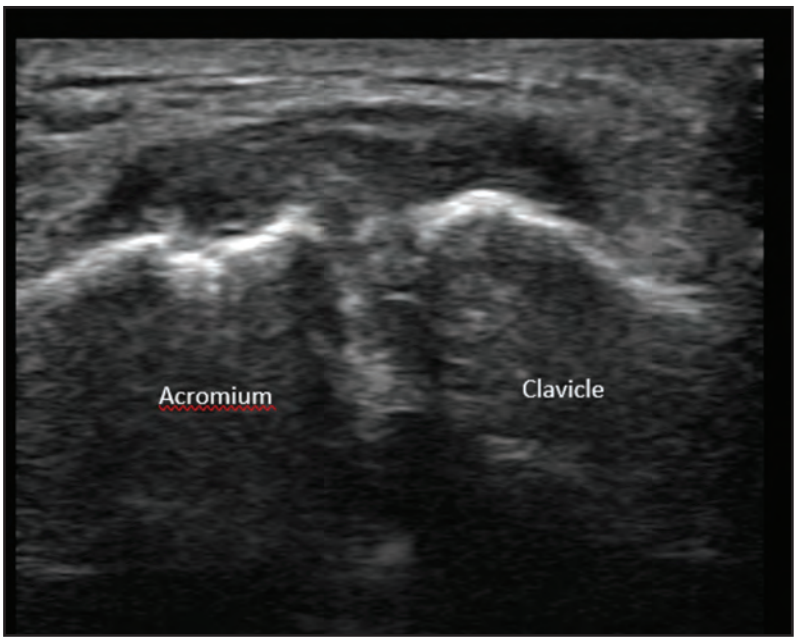

Fig. 2: Bedside sports ultrasound demonstrated a large, well circumscribed swelling overlying the superior and anterior aspect of the acromioclavicular joint suggesting a possible effusion with mixed echogenic substance enclosed within the distended lesion. There was also presence of juxta-articular sclerotic margins.

vascular flow. There was the presence of suspicious juxta-articular sclerotic margins overlying the joint (Figure 2). Our working diagnosis was possible septic arthritis involving the left acromioclavicular joint, and the patient was counselled for a joint drainage and washout procedure.

Intra-operatively, there appeared to be a subcutaneous oedema but no localized collection. There were tophaceous materials deposited onto the otherwise intact joint capsule (Figure 3a). Arthrotomy was not done. The wound was irrigated with normal saline and left open fearing the presence of infection. He was immediately commenced on colchicine 1mg thrice daily post-operatively. A review of his uric acid which was only available the following day showed a borderline high result (426 umol/L, normal range: 208–428umol/L) which further supported our clinical suspicion of an acute gouty arthritis.

The patient was discharged well the following day. Intra-operative tissue cultures were negative for any organism, and at day 7 post-operation, he underwent secondary suturing for wound closure. After 1 month post-operatively, the wound appeared well-healed. His left shoulder range of motion was full, and patient had returned to his daily activities. However, the patient's serum uric acid level only showed minor decrement. A review of the histopathological results showed fragments of crushed fibrotic tissue containing occasional irregular nodules of amorphous eosinophilic material, with areas of mixed inflammatory cell infiltrates and aggregates of histiocytes and urate crystals, which were suggestive of tophaceous gout. The patient was later given a 3-monthly appointment and urate-lowering therapy was continued.

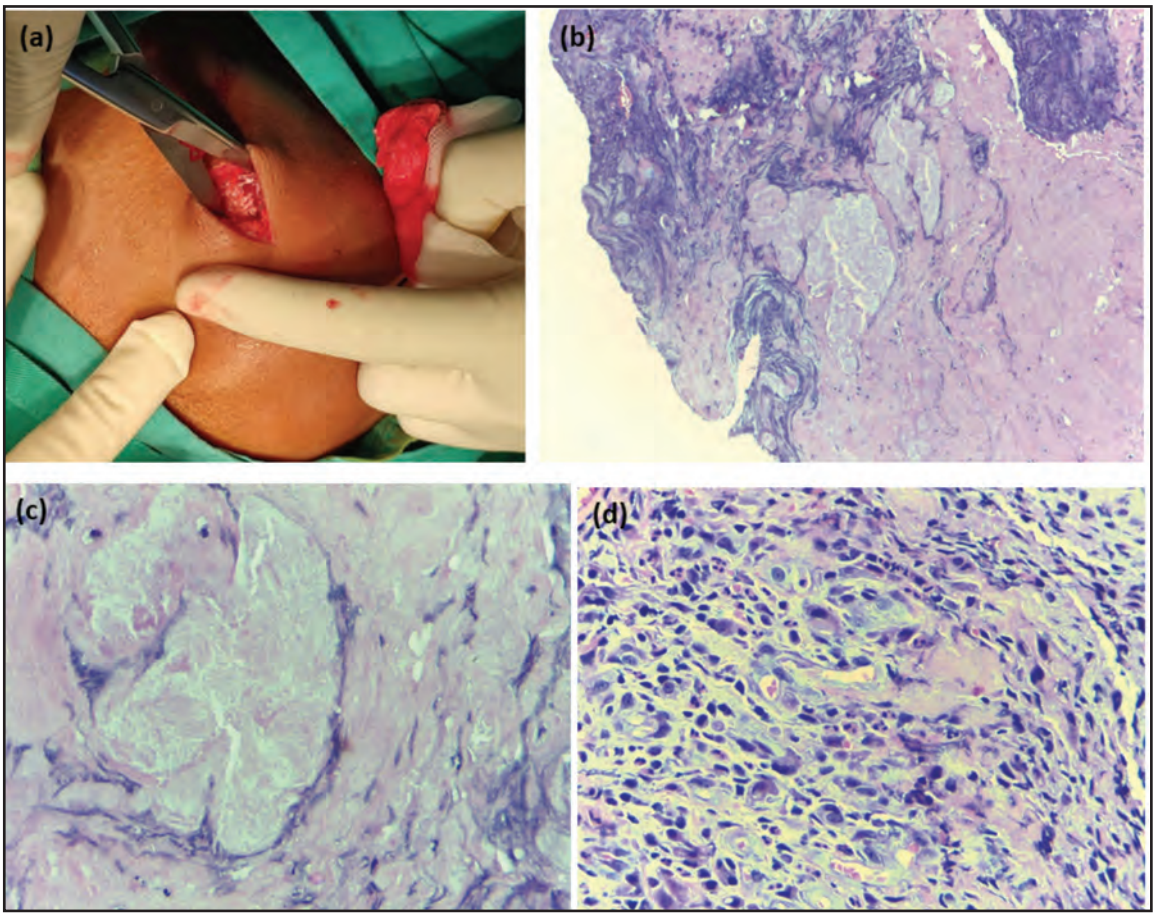

Fig. 3: (A) Intraoperative picture showing tophaceous deposition on the acromioclavicular joint capsule.
(B) Crushed fibrotic tissue containing irregular nodules of amorphous material (H&E ×4)
(C) Aggregates of urate crystals (long and needle-shaped), dissolved after the processing (H&E ×10)
(D) Mixed inflammatory reaction consisting of foreign body giant cells, histiocytes, neutrophils and lymphocytes. (H&E ×10)

DISCUSSION

Gouty arthritis is a crystal-induced arthropathy most commonly affecting the first metatarsophalangeal joint. Nevertheless, it also affects other small and large joints of the appendicular skeleton. The diagnosis of gouty arthritis can sometimes be perplexing in view of potential overlapping characteristics with other differential diagnoses such as pseudogout, septic arthritis, osteoarthritic exacerbation or autoimmune arthritis. In contrast to the other differentials, patients with gout may manifest, for example with a previous history of similar joint pain after certain trigger, sinus with tophaceous discharge or subcutaneous tophi.

Gouty arthritis of the acromioclavicular joint is clinically a rare entity. Coincidentally, all reported cases happened to occur in patients with underlying gout. Certain cases have been associated with other secondary causes such as immunosuppression, lead toxicity, drug (gemfibrozil) use.[1,4,5] Other reported cases include Santis et al who reported a patient with a painless acromioclavicular cyst secondary to gout, and Maxwell et al who reported a case of gouty polyarthropathy with involvement of acromioclavicular joint.[6,7] In contrast, our case presented with a painful monoarticular acromioclavicular gouty arthritis. Historically, warfarin was postulated to increase serum uric acid production and gout manifestation, however, was debunked by another study that shows no significant increase in uric acid concentration in patients on warfarin therapy.[8,9] We postulated that his prophylactic low-dose aspirin for ischemic heart disease might have been the precipitating cause of this atypical gouty flare. Anecdotally, low-dose aspirin use is known to decrease urinary uric acid excretion and has been associated with gout attacks.[10]

This clinical case was perplexing in view of the subacute presentation and atypical joint involvement. Our patient's gout was thought to be in remission since he was flare-free for the past 3 years. These factors initially did not favour the present diagnosis. The narrative of improving symptoms with antibiotic usage and worsening swelling after antibiotic cessation somehow favoured septic arthritis as our principle working diagnosis and therefore, joint aspiration was not suggested initially.

Retrospectively, consideration to perform an intralesional aspiration with a large bore needle prior may have an added diagnostic value and to avoid morbidities associated with an open excision.[6,7] However, aspiration of such a small joint is

often challenging due to narrow joint space and low synovial fluid volume. In addition, synovial Gram stain from arthrocentesis has a low sensitivity with 45%–71% false-negative rates. Presence of negatively birefringent monosodium urate crystals is specific to gout, but nevertheless, septic arthritis can rarely coexist with gout in about 1.5% of cases.[11] A positive synovial fluid culture would be the gold standard for septic arthritis, but it will take at least a few days to detect bacterial growth. Therefore, in cases with high suspicion of infection, an inconclusive synovial fluid analysis should not delay operative intervention for diagnostic and therapeutic purposes.

Ultrasonography examination did confirm a pathological joint but did not objectively aid the decision-making. Perhaps, utilizing a higher resolution ultrasonography device instead of a hand-held screening device may lead to better diagnostic appreciation of pathognomonic features of gout such as the presence of a 'double-contour sign' or 'snowstorm appearance'. A systematic review reported that ultrasound for diagnosis of gout showed high specificity ranging from 0.65 to 1.00.[2] Thus, when in doubt, ultrasonography assessment by a musculoskeletal radiologist would be beneficial in suspected cases of gout.

Gouty arthritis commonly demonstrates a raised serum uric acid; however, in about half of cases, it may remain normal even within periods of acute flare. Serum leucocytes count, erythrocytes sedimentation rate and C-reactive protein are overall not specific for diagnosis but more relevant to monitor clinical progress and resolution.[12]

Lifestyle modification and medications are the mainstay of treatment of gouty arthritis. Intralesional steroid injection together with colchicine or NSAID may supplant the need for surgery.[12] Maxwell et al reported a similar case in 2009 in which they successfully treated the patient with colchicine, oral prednisolone, local intraarticular steroid injection and was able to avoid surgical drainage.[7] Surgical drainage is often associated with poor wound healing and skin necrosis and therefore applied only to indicated cases of tophaceous gout.[3]

Gout is a systemic disease with a wide range of atypical presentations. It could affect the joints of both the appendicular and axial skeleton.13 Other than atypical joint localization, gout can also have out of the norm presentation such as erythema nodosum, flexor tenosynovitis, giant cell tumour, tumour-like lumps, acute locked knee, finger flexion deformity and carpal tunnel syndrome.[14-17] The causes of such atypical presentations are unclear, possible correlations being genetic, serum uric acid concentration, presence of other systemic illnesses, and usage of drugs interfering with uric acid metabolism.

CONCLUSION

In patients presenting with an atraumatic acromioclavicular joint pain and underlying gout, gouty arthritis should be considered even in normo-uricemia state. Eliciting a detailed past medical history needs to be emphasized. Findings of characteristic features of gouty arthritis on ultrasonography by an experienced operator are helpful. In suspicious cases, diagnostic aspiration may be attempted under ultrasound guidance. Most patients with gouty flare recover well with optimal medical therapy. Surgical drainage remains an option only when infection is suggestive, or when diagnosis remains inconclusive even after a thorough workup. Finally, the link between low-dose aspirin intake and acromioclavicular gouty arthritis is still unclear and requires more understanding by research.

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