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Case Report

Gluteal Abscess Caused by Infected Gouty Panniculitis: A Case Report on Sequalae of Uncontrolled Gout

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Abstract:

Gouty panniculitis is uncommon manifestation of gout associated with monosodium urate (MSU) deposition into subcutaneous tissue. We report our local experience of this disease and its management strategy. Our patient was 39 years old gentleman, known to have gout who had not been compliant to follow up, presented with bilateral gluteal swellings for one month associated with discharge from the left gluteal swelling for 2 days. Clinical diagnosis of gluteal abscess with suspected infected gouty tophi was made. Wound debridement of infected left gluteal swelling was performed, followed by short course antibiotic therapy according to culture and sensitivity. Subsequently our rheumatology colleagues had assisted us with medical management of his gout, in which his right gluteal swelling did not get worsen and he remained well during our follow up.

Keywords: Gouty tophi, Panniculitis, Monosodium urate crystals, Ethiopia

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Introduction

Hyperuricemia associated with monosodium urate (MSU) crystal deposition in the synovial fluid leads to a condition known as gout, a form of inflammatory arthritis (1). Diagnosis of gout is proven by identification of MSU crystal in synovial fluid analysis (1,2). Gout is getting more prevalent related to diet factors (3). Gout affects men more than female (ratio 4:1) (1). The presence of MSU crystals in synovial fluid or tissue biopsy confirm diagnosis of gout (4). Tophi characteristically resembles granuloma where inflammatory cells and connective tissue surrounding the MSU crystals aggregate (4,5), which suggests advanced stage of gout. Common sites of tophi include tendons, ligaments, knee joints and so on (6). There were reports of unusual sites of tophi, namely skin, bones, and axial skeletons (7). Gouty panniculitis occurs due to deposition of urate crystals into subcutaneous tissue, either prior or as a sequalae of chronic tophaceous gout (1,9). It was hypothesized that subcutaneous tissue destruction and localized inflammation leading to increased production and deposition of uric acid into subcutaneous layer (9).

We would like to share our experience of encountering a case of atypical site of gluteal gouty panniculitis which manifested with abscess formation.

Case presentation

This is 39 years old gentleman, with underlying hypertension and gout previously on allopurinol who defaulted follow up for the past 1 year. He presented to Sibu Hospital emergency department with bilateral gluteal swelling of 1 month associated with discharge from the left gluteal swelling for 2 days. Clinical examination revealed multiple indurations on the right gluteal region, ruptured left gluteal swelling about 8cm x 10cm with chalky white material and purulent discharge surrounded by erythematous skin (Figure 1).



Figure 1: Photo of left gluteal region showing erythematous skin and induration with ruptured swelling showing chalky material suggestive of tophi (red arrow)

Total white cell count was raised (36.3 x $103/\mu$ L), with deranged urea (12.5 mmol/L) and creatinine (290 μ mol/L). His serum uric acid was 723 μ mol/L.

and subsequently referred to rheumatology team for uncontrolled gout. He was started on low dose hydrocortisone and allopurinol for severe unstable chronic tophaceous gout.

He underwent wound debridement of left gluteal infected gouty tophi (Figure 2)



Figure 2: Post wound debridement of left gluteal region showing underlying healthy muscle and fascia, with minimal residual tophi. (green arrow)

Post operatively he has had daily saline dressing over his left gluteal wound and his intra operative culture reported as *Staphylococcus aureus*, which he had completed 1 week course of antibiotics. Histology of left gluteal tissue reported palisading granuloma showing aggregates of histiocytes with foreign body type multinucleated giant cells, consistent with panniculitis (Figure 3).



Figure 3: H&E x 200 - The palisading granuloma shows aggregates of histiocytes with foreign body type multinucleated giant **cell**.

On follow up after 3 months, he had his left gluteal wound almost healed with granulation and reepithelialization seen, with no worsening of his right gluteal swelling as he had been compliant to his gout medication (Figure 4).



Figure 4: At 3 months follow up, there is evidence of granulation and re epithelization of left gluteal wound. Right gluteal gouty tophi not worsened compared to previous. (**yellow arrow**)

Discussion

Gouty panniculitis is atypical presentation of gout patients with subcutaneous tissue lesions at any site, related to lobular hypodermis deposition of MSU crystals (8,9). This is uncommon diagnosis unfortunately happened to our patient, with paucity of reported cases till present (10,11). There was postulate that high tendency of deposition in lobular subcutaneous tissue possibly related to microtrauma of the terminal capillary wall and adipose tissue coupled with MSU crystals associated arteriopathy (9). Our patient presented with significantly elevated uric acid level, which might be associated with occurrence of gouty panniculitis (12). Differential diagnoses of gouty panniculitis include cellulitis, pseudogout, calciphylaxis, lupus panniculitis, which need to be excluded (13). Panniculitis is a histological diagnosis, with presence of granuloma with adipocyte necrosis (10), which is found in our patient histopathological specimen. Whenever diagnosis is in doubt or uncertain, imaging modalities (ultrasound, plain radiograph, computed tomography, and magnetic resonance imaging) can be useful to identify severity of disease (14). The presence of chalky whitish material from our patient buttock with background history of gout supported our clinical diagnosis of infected gluteal gouty tophi, thus we did not proceed with any imaging studies, as it is considered as a form of subcutaneous infection of unusual etiology. In our patient, surgical treatment in the form of wound debridement was performed, followed by anti-hyperuricemic medication, with low dose steroid, comanaged with our rheumatology colleagues, aiming to control pain and inflammation of contralateral gouty tophi (9,10). While our case report is limited by lack of structured guideline in managing gouty panniculitis with scarce literatures, we were glad that collaboration between surgical and rheumatology professionals provide good post operative outcome for our patient. We had proven that with adequate wound debridement followed by compliance to anti-hyperuricemic medication by our rheumatology colleagues, provide long term uric acid control and showed no new gouty tophi elsewhere in our patient.

Conclusion

In patients known to have long standing hyperuricemia and gout with subcutaneous erythematous lesions, clinicians should consider gouty panniculitis as a possible diagnosis. Surgical intervention for infected lesion, coupled with medical therapy to control hyperuricemia can be useful treatment strategy to prevent recurrence.

Competing interests

There was no funding for the study and no conflicts of interest to disclose.

Consent

Written informed consent was obtained from the patient participant for publication of this case.

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