

Persistent Ulcerated Tophaceous Gout Complicated by Fungal Infection: A Case Report

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ABSTRACT

Gout is a common inflammatory joint disease resulting from hyperuricemia, leading to the deposition of urate crystals in joints and soft tissues, forming gouty tophi. Gouty tophi ulceration in the skin and subcutaneous tissue is a relatively rare clinical occurrence. Currently, there is no universally accepted optimal protocol for treating ulcerative tophi. This report describes a case of persistent, ulcerated, tophaceous gout complicated by fungal infection, intending to increase clinical awareness of ulcerative tophi and propose a practical therapeutic approach that may be considered in future management strategies.

Keywords:

ulcerated tophaceous gout, Candida parapsilosis, vacuum sealing drainage

INTRODUCTION

Hyperuricemia can lead to the formation of urate crystals when serum uric acid levels exceed their saturation point. Moreover, these crystals can cause joint pain and accumulate in locations such as the kidneys, subcutaneous tissues, and cartilage. When gouty tophi are present in the skin and subcutaneous tissue, their ulceration can significantly impact quality of life and greatly increase the risk of infection¹.

Ulcerated tophaceous gout is relatively uncommon in clinical practice, and its prevalence has not been well established. Available evidence suggests that excessive glucocorticoid use, a prolonged duration of tophi formation, and a higher number of tophi are associated with an increased risk of developing ulcerative tophi².

CASE REPORT

A 68-year-old male with a 10-year history of untreated gout presented with recurrent joint swelling and pain, as well as multiple gouty tophi throughout his body. Six months before presentation, a gouty tophus at the olecranon of his left elbow spontaneously ulcerated, leading to the exudation of a white, curd-like substance. The patient initially disregarded the condition and, due to financial constraints, did not seek medical care. As the wound progressively worsened, his family urged him to visit the hospital. His medical history was notable for hypertension.

Hematological tests were performed on the patient, revealing a white blood cell count of $9.5 \times 10^9/L$ (normal: 3.5–9.5), a neutrophil count of $8.43 \times 10^9/L$ (normal: 1.8–6.3), and a C-reactive protein level of 13.3mg/L (normal <10). Biochemical analysis showed a uric acid level of $645 \mu\text{mol/L}$ (normal: 149–416), creatinine of $141 \mu\text{mol/L}$ (normal: 59–104), and glycated haemoglobin of 5.6% (normal: 4.3–6.5). Other evaluations, including screening for infectious diseases, did not reveal any significant abnormalities. On physical examination, an ulcerated gouty tophus measuring approximately $4 \times 4\text{cm}$ was observed at the left olecranon, with white chalky material, purulent discharge, surrounding erythema and swelling (Fig. 1). The patient's elbow joint demonstrated preserved range of motion and function. However, radiograph imaging revealed swelling and disruption in the surrounding soft tissues. Magnetic resonance imaging (MRI) of the left elbow revealed subcutaneous gouty tophi at the olecranon, triceps tendonitis, joint effusion, and bone marrow edema of the ulnar olecranon (Fig. 2).

Discharge from the wound was collected for culture. Given that skin infections are most commonly caused by Gram-positive cocci, amoxicillin-clavulanate was initiated. On the



Fig. 1: (a) Before treatment. (b) After debridement surgery. (c) After VSD therapy. (d) The fully healed wound.

third day, culture results identified *Candida parapsilosis* (*C. parapsilosis*). Based on susceptibility testing, the patient was prescribed a daily intravenous dose of 400mg fluconazole. On the 4th day, debridement surgery was performed, during which necrotic tissue was excised. Histopathological analysis revealed gouty nodules, and culture of the necrotic tissue confirmed the presence of *C. parapsilosis* (Fig. 3). Post-operatively, vacuum sealing drainage (VSD) therapy was applied for three days. Upon removal of the VSD device, the ulcer had significantly reduced in size, with decreased erythema and pain. Following this, continuous anti-infective therapy was administered in combination with regular wound dressing changes. The patient underwent a total of six weeks of anti-infection treatment, consisting of 400mg of intravenous fluconazole daily for the first three weeks, followed by 400mg of oral fluconazole daily for the remaining three weeks. Wound dressings were changed daily during the initial three weeks using the following procedure: the wound and cavities were disinfected with iodophor, petroleum jelly gauze were utilised for packing cavities and covering wound surface, then covered with sterile gauze, and an elastic bandage was applied. As the cavity gradually reduced in size, dressing changes were performed every two days until complete wound healing was achieved.

Furthermore, microbial cultures of the wound secretions were repeated on treatment days 10, 15, and 20, all of which returned negative results. By the 50th day of treatment, the

wound had completely healed. Since uric acid-lowering therapy often triggers acute gout attacks, and the patient had no previous exposure to such treatment. Therefore, urate-lowering therapy was initiated after complete wound healing had occurred.

DISCUSSION

The rising prevalence of gout, as well as ulcerated tophaceous gout, poses a growing treatment challenge. However, management guidelines for ulcerated tophi remain limited. Previous case reports have described various treatment modalities, including surgical excision with free flap transplantation, multiple debridements with autologous platelet-rich gel applications, and the use of silver dressings and freeze-dried collagen, resulting in healing times ranging from 20 to 60 days³. In this case, considering the established effectiveness of VSD in managing diabetic foot ulcers, we applied this technique post-debridement, a strategy not previously reported for ulcerated gouty tophi. VSD facilitates wound healing by providing continuous negative pressure, reducing edema, and promoting the formation of granulation tissue. Following its application, the patient's ulcer demonstrated significant improvement. Further wound care involved the use of cost-effective petrolatum gauze and sterile gauze. Lastly, after 50 days of treatment, the patient achieved complete recovery.

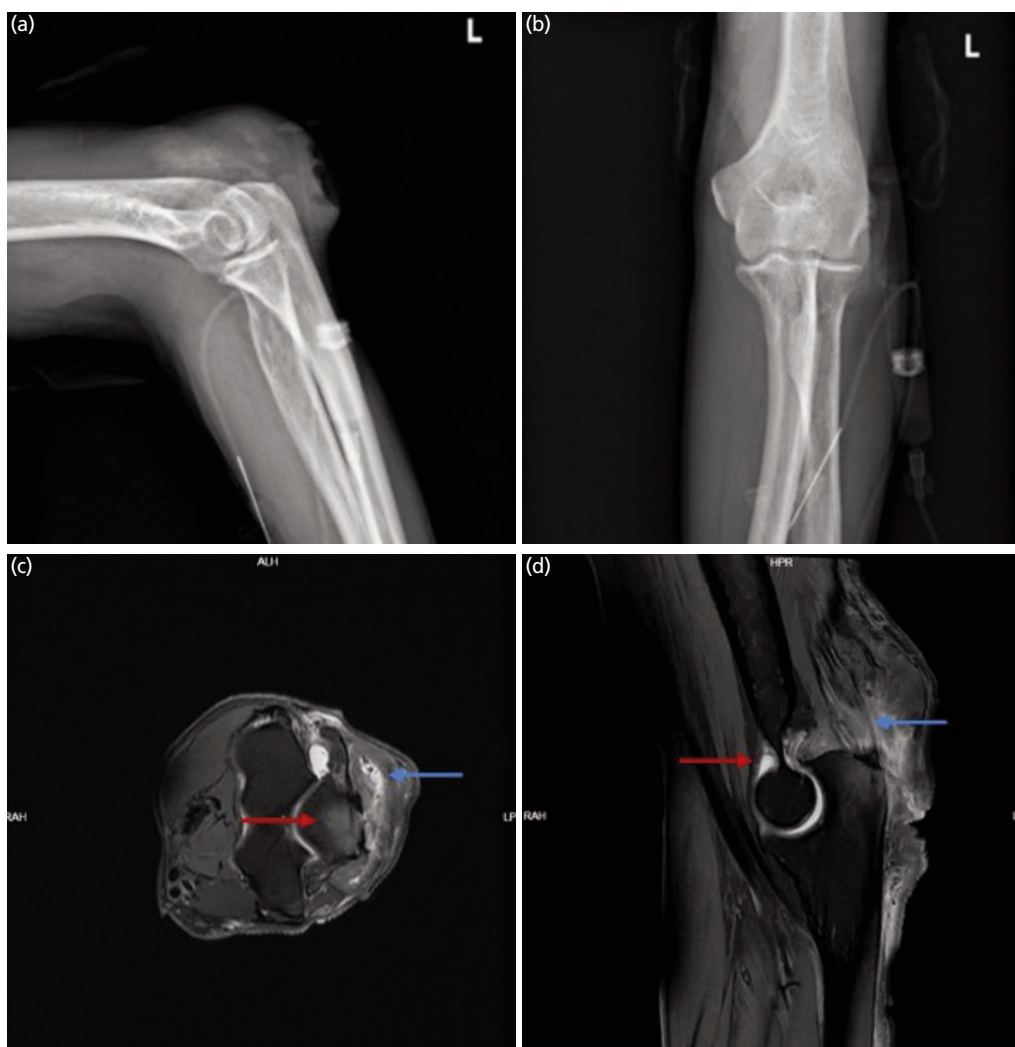


Fig. 2: Radiograph of the left elbow, (a and b) Soft tissue swelling around the elbow joint. MRI of the left elbow, (c) Red arrow: bone marrow edema of the olecranon, and blue arrow: subcutaneous gouty tophi. (d) Red arrow: joint effusion, and blue arrow: triceps tendonitis.

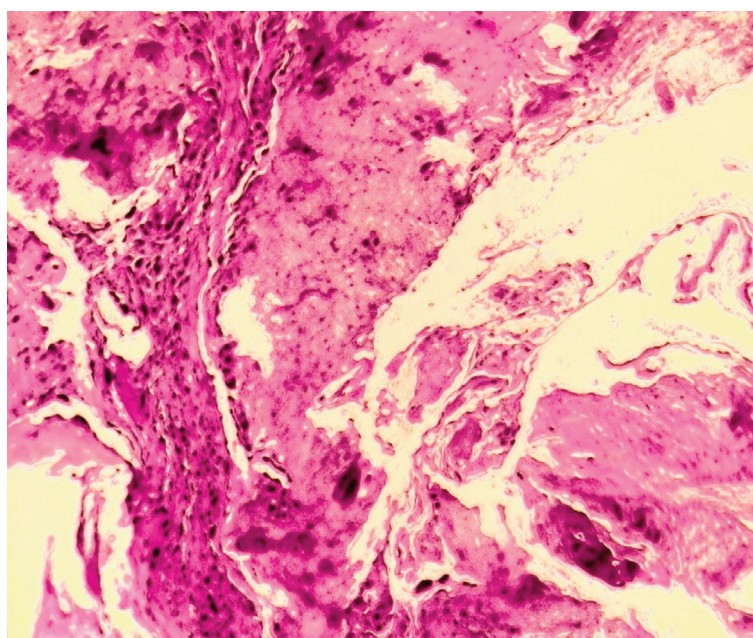


Fig. 3: Gouty tophi, surrounded by inflammatory granulation tissue with areas of necrosis.

In addition to debridement surgery and continuous wound care, anti-infective therapy plays a crucial role in managing ulcerative tophi. Previous reports have highlighted that ulcerative gouty tophi can predispose patients to severe infections, including methicillin-resistant *Staphylococcus aureus* (MRSA), which may progress to sepsis and hemophagocytic syndrome⁴. These findings underscore the importance of vigilant monitoring for secondary infections in patients with ulcerated tophi.

In most reported cases, bacterial infections are the predominant type. A study involving 38 patients with ulcerative tophi found that 18 developed secondary infections, with *Staphylococcus aureus* responsible for nine cases and other pathogens, including *Pseudomonas aeruginosa*, *Acinetobacter baumannii*, and *Shewanella algae*⁵. In comparison, our patient developed a rare secondary infection with *C. parapsilosis*, which is an opportunistic pathogen that typically affects immunocompromised individuals, such as those with neutropenia, HIV infection, diabetes, burns, prosthetic devices, or prolonged antibiotic exposure. Interestingly, our patient did not present any of these known risk factors, and the precise mechanism underlying the fungal infection in this case remains unclear. Infection of the subcutaneous soft tissues caused by *C. parapsilosis* may potentially progress to bloodstream infections, osteomyelitis, or arthritis. In this

patient, MRI findings revealed joint effusion and bone marrow edema, suggesting a potential risk for infectious arthritis and osteomyelitis if the infection were to disseminate. Timely intervention successfully prevented severe complications.

Microbiological culture of necrotic tissue is crucial for identifying coexisting infections, determining the specific pathogens involved, and guiding targeted antimicrobial therapy. Early-stage intervention is essential for preventing the spread of further infection and avoiding serious complications.

With the rising prevalence of gout, an increasing number of patients with poorly controlled disease are likely to develop ulcerated tophi. At present, no standardised management guidelines exist for ulcerated tophaceous gout. Through the presentation of detailed clinical observations, diagnostic considerations, and therapeutic outcomes, this report seeks to offer valuable guidance to healthcare professionals who may encounter similar cases.

CONFLICT OF INTEREST

The authors declare no potential conflict of interest.

REFERENCES

1. Patel GK, Davies WL, Price PP, Harding KG. Ulcerated tophaceous gout. *Int J Rheum Dis*. 2010; 7(5): 423-7. doi: 10.1111/j.1742-481X.2010.00700.x
2. Huang Z, Liu X, Liu Y, Li G, Pan X, Huang Z, *et al*. Clinical characteristics and risk factors of ulceration over tophi in patients with gout. *Int J Rheum Dis*. 2019; 22(6): 1052-7. doi: 10.1111/1756-185X.13581
3. Lam G, Ross FL, Chiu ES. Nonhealing Ulcers in Patients with Tophaceous Gout: A Systematic Review. *Adv Skin Wound Care*. 2017; 30(5): 230-7. doi: 10.1097/01.ASW.0000515456.65405.56
4. Yang W, Yang C. Septic arthritis caused by gout progressed to sepsis and hemophagocytic syndrome. *Heliyon*. 2024; 10(9): e30583. doi: 10.1016/j.heliyon.2024.e30583
5. Xu J, Zhu Z, Zhang W. Clinical characteristics of infectious ulceration over tophi in patients with gout. *J Int Med Res*. 2018; 46(6): 2258-64. doi: 10.1177/0300060518761303

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