BRIEF ARTICLE

Tophaceous Gout on Ear Imitating Squamous Cell Carcinoma

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ABSTRACT

We report a case of tophaceous gout occurring in a Hispanic 69-year-old immunocompetent man. The patient presented with a 0.8 cm pink, purple, and brown dome-shaped papule on the right superior helix of the ear. The lesion was tender to palpitation. A deep shave biopsy was performed and the histological findings demonstrated pseudoepitheliomatous hyperplasia overlying large dermal deposits of pink, hyalinized feathery material surrounded by mild granulomatous inflammation. This finding supported the diagnosis of tophaceous gout rather than the clinical findings imitating squamous cell carcinoma. Concurrently, the patient received an intralesional Kenalog (ILK) injection to suppress any existing or ensuing inflammation. Treatment of the residual lesion was achieved via curettage and electrodesiccation.

INTRODUCTION

Gout is a common, yet complex form of arthritis that results from the deposition of monosodium urate crystals following normal or increased concentrations of uric acid in the serum. These deposits are most commonly found within joints and typically lead to acute pain and inflammation. Tophaceous gout usually presents as smooth, white, or yellow subcutaneous nodules filled with liquid, pasty, or chalky masses which can be deposited in the dermis or connective tissue layer; however, in rare cases the overlying skin can become hyperkeratotic, thus mimicking a squamous cell carcinoma. We present a case of tophaceous gout where the tophus presented on a patient's superior helix of the ear, clinically mimicking a squamous cell carcinoma.

CASE REPORT

A Hispanic 69-year-old immunocompetent man with a past medical history of coronary artery disease, carpal tunnel syndrome, hyperlipidemia, gout, and hypertension presented with a painful lesion on his right ear, which had been actively growing for the past five years. He denied any bleeding or drainage from the area. He also denied any preceding treatment or procedures for this issue. He had Fitzpatrick skin type IV with no prior history of skin cancer or other skin disorder. His current medications included atorvastatin, ezetimibe, lisinopril, and meloxicam. He reported that he was treated

for gout in the past with medication, diet, and exercise; however, he was not on any specific medication for gout at the time of presentation. He denied a history of HIV or hepatitis and reported that he quit smoking 40 years prior and only drank alcohol on special occasions. He also denied fever, chills, weight loss, nausea, vomiting, diarrhea, shortness of breath, chest pain, or night sweats. Finally, he had no family history of melanoma or other dermatological diseases.

Physical examination revealed a 0.8 cm pink, purple, and brown dome-shaped papule on the right superior helix of the ear (Figure 1). The lesion was tender to palpitation and drainage was observed. Cervical lymph node examination did

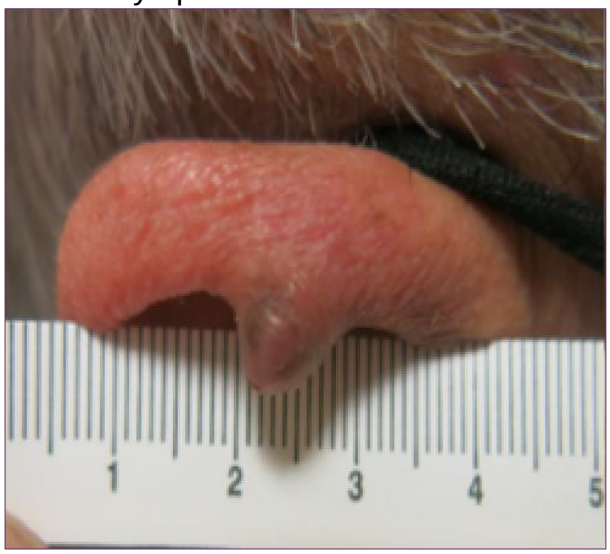

Figure 1. Physical examination revealed thepresence of a pink, purple, and brown dome-shaped papule approximately 0.8 cm in diameter on the right superior helix of the ear

not show any lymphadenopathy. All laboratory findings including uric acid levels were within the normal range. A deep shave biopsy was performed and the histological findings demonstrated pseudoepitheliomatous hyperplasia overlying large dermal deposits of pink, hyalinized feathery material surrounded by mild granulomatous inflammation (Figure 2). PAS was negative for hyphae. Clinical presentation, patient's past medical history, and histological findings were consistent with gout. Concurrently, the patient received an intralesional Kenalog (ILK) injection to suppress any existing or ensuing inflammation. Treatment of the residual lesion was achieved via curettage and electrodesiccation.

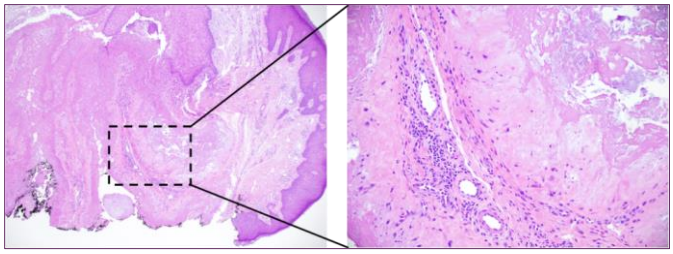

Figure 2. Histological findings showcasing pseudoepitheliomatous hyperplasia overlying large dermal deposits of pink, hyalinized feathery material surrounded by mild granulomatous inflammation.

DISCUSSION

Gout is arguably the most common inflammatory arthritis and occurs in approximately 3% of the United States population [1,2]. The incidence of gout tends to be higher in adult males and occurs rarely in children. One notable exception is in children carrying inherited defects in the enzymes responsible for metabolizing purine, which subsequently leads to hyperuricemia, or in disorders resulting in impaired renal clearance of uric acid [3]. Although gout can result from genetic factors, diet is also a paramount contributor given the fact that the incidence of gout continues to rise concomitantly with rising obesity and diabetes rates [4]. Unsurprisingly, gout is also intimately associated with hypertension and cardiovascular disease—two disease states whose pathogenesis is also exacerbated by obesity and diabetes [4]. Certain medications such as diuretics, methotrexate, cyclosporine, and etanercept

have been associated with an increased incidence of tophaceous gout [2,4].
The most common pathological feature of gout is the deposition of monosodium urate crystals resulting from chronically high serum levels of urate, otherwise known as hyperuricemia (>6.8 mg/dL) [2]. The diagnosis of gout can be verified histologically by the presence of long, needle-shaped crystals of monosodium urate [1,2,4]. Meanwhile, the clinical manifestation of gout results from activation of the inflammatory response to the urate crystals.[2] Therapeutic interventions for gout are centered around strategies that promote the dissolution of urate crystals, and effective uric acid management via dietary and lifestyle modifications.[4] One of the most effective urate-lowering agents is allopurinol, which typically leads to dramatic reductions in gouty arthritis and tophi. When it comes to acute gout flares, some of the most widely recommended drugs include non-steroidal anti-inflammatory drugs and colchicine.[4] Other therapeutic avenues can be pursued depending on the location and severity of the lesion. As presented here, deep shave biopsy followed by ILK injections, curettage, and electrodesiccation is an effective treatment option. Alternatively, resolution of the residual lesion can also be achieved with excisional surgery.

CONCLUSION

Given that gout can be managed or cured by lowering the serum concentration of uric acid below the saturation point of monosodium urate, it is critical that patients are educated about the importance of adhering to the recommended treatment regimen or implementing the appropriate lifestyle and diet modifications.[4] This way, they can be better equipped to understand the treatment of their condition and take an active role in preventing the associated comorbidities that dramatically alter their quality of life.

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