Case Report

Miliarial Gout – An Uncommon Presentation of Chronic Tophaceous Gout

Silvino Rey Hipolito Pino, Maria Mercedes Siayngco Cauilan, Lalaine Rabe Visitacion

Department of Dermatology, Southern Philippines Medical Center, Davao City, Philippines

Abstract

Chronic tophaceous gout is a manifestation of longstanding, untreated hyperuricemia, characterized by monosodium urate crystal deposition in soft tissues and joints. While commonly presenting as nodular or papular tophi, rare morphologic variants such as miliarial gout may occur. We present a case of a 35-year-old Filipino male who presented with classic cutaneous manifestations of chronic tophaceous alongside numerous widespread, milia-like papules containing chalky tophaceous material, consistent with miliarial gout. This distinct and uncommon variant has only been reported in a few cases worldwide. Notably, the patient had a history of prolonged unsupervised corticosteroid use, renal insufficiency, and poor compliance to urate-lowering therapy, all of which may have contributed to the rapid and extensive cutaneous involvement. This case highlights the importance of early recognition of atypical gout presentations and emphasizes the potential role of corticosteroids in modifying disease progression and morphology.

Keywords: Chronic tophaceous gout, cutaneous gout, miliarial gout, tophi

Address for correspondence: Dr. Silvino Rey Hipolito Pino, Department of Dermatology, Southern Philippines Medical Center, Davao City, Philippines.
E-mail: silvinoreypinomd@gmail.com

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Introduction

Gout is a clinical syndrome characterized by deposition of monosodium urate crystals in synovial fluid, articular, and nonarticular structures. The etiology of gout is multifactorial which includes genetic risk factors, medical comorbidities, and dietary factors. A high serum rate concentration, termed hyperuricemia (uric acid level of ≥7 mg/dL), is the most important risk factor. Other risk factors include older age, male sex, obesity, a purine diet, alcohol consumption, medications, and comorbid diseases.[1]

Chronic tophaceous gout is a manifestation of untreated gouty arthritis characterized by urate crystal deposition within subcutaneous tissues, tendons, articular structures, or bursas. Multiple morphologic variants exist, namely bullous, fungating, miliarial, nodular, papular, and pustular.[2] In this case report, we describe a case of miliarial gout, a unique variant that presents with widely distributed milia-like papules containing chalky tophaceous material. This is an uncommon and unique entity with only a few reported cases worldwide.

Case Report

A 35-year-old Filipino male presented with a 1-year history of multiple asymptomatic yellowish-white papules and nodules involving the ears, trunk, and extremities. Over time, some lesions became erythematous, ulcerated spontaneously, and exuded chalk-like material.

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At age 31, he began experiencing recurrent bouts of arthralgia and swelling of the bilateral first metatarsophalangeal joints, later involving the ankles and knees. He was diagnosed with gouty arthritis by a private internist and was prescribed diclofenac 50 mg tablet and dexamethasone 500 µg tablet, both taken as needed for joint pain. Despite initial symptomatic relief, he began self-medicating daily with these drugs and discontinued follow-up. Urate-lowering therapy (ULT) was not initiated at the time.

Over the years, his lesions progressively increased in number and distribution, accompanied by worsening polyarthralgia and joint swelling. These symptoms became only partially responsive to diclofenac and dexamethasone. The persistence and progression of both cutaneous and joint manifestations prompted consultation at our institution.

The patient had a family history of gouty arthritis, with his maternal uncle diagnosed with gout and reportedly succumbed to complications associated with the condition. He works as an electric maintenance employee with a sedentary lifestyle, a cigarette smoker for 3 pack years, and consumes alcoholic beverages occasionally, averaging 2–4 bottles of beer per month. His diet is rich in fat and protein.

On physical examination, the patient's height was 1.78 meters and weight was 74 kg, corresponding to a body mass index of 23.34 kg/m^2 (normal). Multiple firm, nontender, movable nodules were noted over the joint surfaces of elbows and knees [Figure 1a]. There were multiple yellowish to cream-colored minute papules on the external ear, anterior and posterior trunk, upper and lower extremities, alongside erythematous to hyperpigmented macules and papules [Figure 1b-d]. Few erythematous to hyperpigmented plaques with w/punched-out ulcerations, hemorrhagic crusting, and chalky-white discharges on the posterior aspect of the lower extremities [Figure 1e] were also seen. In addition, there were incidental findings of cushingoid features, including a rounded face, proximal muscle weakness, protuberant abdomen, and thin arms and legs [Figure 1g]. Polarized contact dermoscopy revealed yellowish-white structureless areas with arborizing vessels arising from the periphery. These characteristics bear a striking resemblance to those of milia [Figure 1f].

Blood tests revealed hyperuricemia (11.3 mg/dL), hypercholesterolemia (6.1 mmol/L), and elevated creatinine levels (161 µmol/L), with an estimated glomerular filtration rate of 42.5 mL/min/1.73 m^2, classifying the patient as having chronic kidney disease stage IIIB. Urinalysis showed microalbuminuria. The patient's rheumatoid factor was negative. Imaging work-up, including radiography of hands and feet and a kidney, ureters, and bladder ultrasound, yielded unremarkable results with no evidence of calcifications.

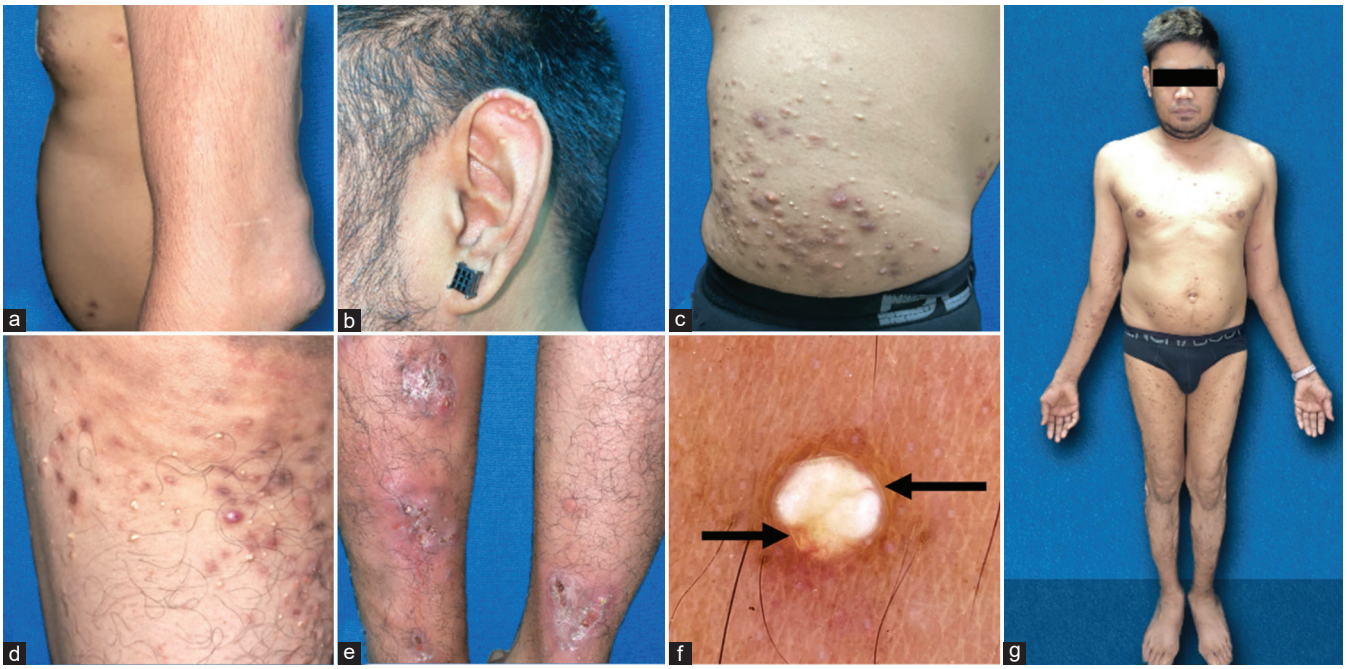

Figure 1: Dermatologic Findings: Multiple firm, nontender, movable nodules overlying joint surfaces (a). Multiple nontender, yellowish to cream-colored papules on the external ear, trunk, upper and lower extremities alongside erythematous to hyperpigmented macules and papules (b-d). Few erythematous to hyperpigmented plaques with punched-out ulcerations, hemorrhagic crusting, and chalky-white discharges on the posterior aspect of the lower extremities (e). Polarized Dermoscopy (×10): yellowish-white structureless areas with arborizing vessels (black arrows) arising from the periphery (f). Cushingoid features: Rounded face, proximal muscle weakness, protuberant abdomen, and thin arms and legs (g).

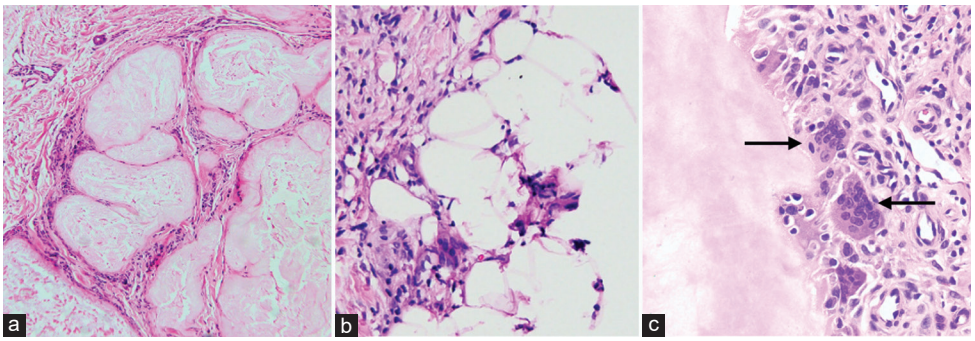

Figure 2: Histopathology: The dermis revealed mild interstitial and perivascular infiltrates composed of lymphocytes, histiocytes, neutrophils, eosinophils, and multinucleated giant cells (a), extending into the subcutaneous fat lobules (b). Multiple amorphous eosinophilic material surrounded by histiocytes and multinucleated giant cells (black arrows) (c). H and E (a), ×40, (b and c), ×400

Two 4 mm punch biopsies were obtained from a papule on the forearm and an ulcerated plaque on the posterior right leg. Upon gross inspection, both sections exhibited yellowish-white, friable materials resembling chalk. On histopathologic examination [Figure 2], the dermis revealed the presence of multiple amorphous deposits of eosinophilic material surrounded by palisading histiocytes, lymphocytes, and multiple multinucleated giant cells forming granulomas located in the deep dermis, extending into the subcutaneous fat lobules. A diagnosis of cutaneous gout and gouty panniculitis was made, and considering its peculiar cutaneous presentation, miliarial gout was also considered.

The patient was referred to the rheumatology service. Dexamethasone was gradually tapered and then discontinued after 2 months. Febuxostat 40 mg twice daily and colchicine 500 μg once daily were initiated. Despite the patient's noncompliance of the prescribed medications due to financial limitations and suboptimal control of urate levels which showed persistently increasing levels, the milia-like papules gradually decreased in both number and size [Figure 3].

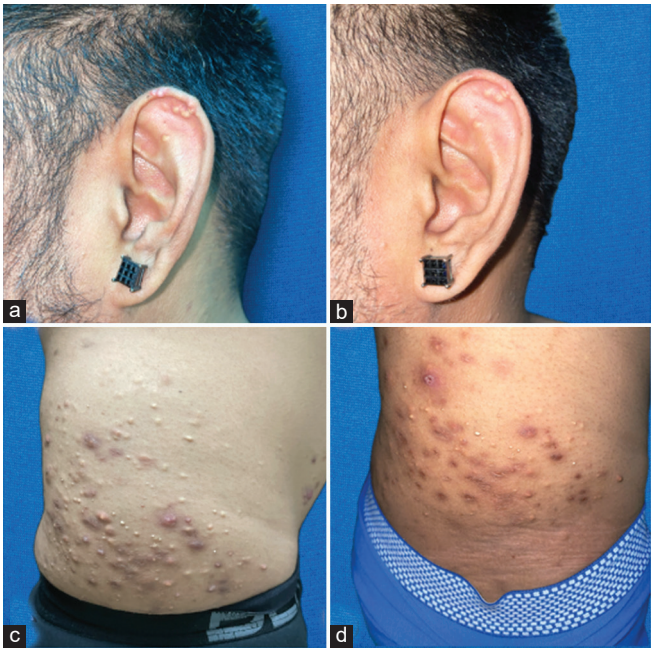

Figure 3: Clinical improvement of lesions with postinflammatory hyperpigmentation after discontinuation of dexamethasone and initiation of febuxostat and colchicine (before treatment: a and c; after treatment: b and d)

Discussion

Chronic tophaceous gout refers to advanced-stage gout characterized by persistent symptoms with infrequent or absent asymptomatic intervals. Patients typically present with tophi deposited around joint surfaces, cartilage, and tendons. On average, chronic tophaceous changes develop approximately 12 years after the initial gout episode, though onset may range widely from 5 to 40 years.[1] In this case, the patient exhibited a classic progression from initial gout flares to advanced tophaceous gout; however, his presentation was distinguished by unusual intradermal tophi resembling milia. In addition, the rapid progression from initial symptoms to widespread tophaceous involvement within just 4 years was notably atypical.

The precise pathogenesis underlying intradermal tophi formation remains unclear; however, prolonged corticosteroid therapy has been hypothesized to accelerate disease progression and intradermal crystal deposition.[2] Rull *et al*. demonstrated corticosteroid-associated promotion of urate crystal deposition in animal studies.[3] Literature review identified seven similar cases of miliarial gout, four associated with prolonged steroid use.[2,4-9] Additional risk factors include renal insufficiency, untreated hyperuricemia, and chronic diuretic therapy, all present in our patient.

Given the potential complexities and atypical presentations, dermatologists should adopt a proactive role in managing cutaneous gout beyond recognition and diagnosis. Collaboration with rheumatologists and nephrologists is

essential. Dermatologists can contribute significantly by initiating early work-up and recommending early initiation of ULT, lifestyle modification, and procedural interventions. Here is a proposed algorithm for dermatologists managing cutaneous gout [Figure 4].[10-12]

The management of chronic tophaceous gout aims to reduce gout flares, reverse tophi formation, and prevent progressive structural joint damage. These can be achieved through a combination of lifestyle modification/risk reduction and pharmacologic therapy. ULT, such as xanthine oxidase inhibitors, uricosuric agents, and uricase agents, is used to decrease and maintain sub-saturating urate levels with a serum urate target level of <6 mg/dL (below the urate solubility limit). Administration of concomitant anti-inflammatory prophylaxis, such as colchicine, oral corticosteroids, and nonsteroidal anti-inflammatory drugs, during initiation of ULT is also recommended for the prevention of gout flares.[13] In our patient, ULT with febuxostat (40 mg twice daily) and colchicine (500 μg daily) were initiated with planned dose escalation if serum urate targets were not met. However, the patient was subsequently lost to follow-up.

In conclusion, dermatologists have a critical role in the comprehensive management of cutaneous gout, including early diagnosis, initiation of therapy, and recommending procedural interventions. Continued interdisciplinary collaboration will optimize patient outcomes, highlighting the importance of dermatology beyond diagnostic recognition alone. Further research is warranted to elucidate the mechanisms linking prolonged steroid use and intradermal tophi formation.

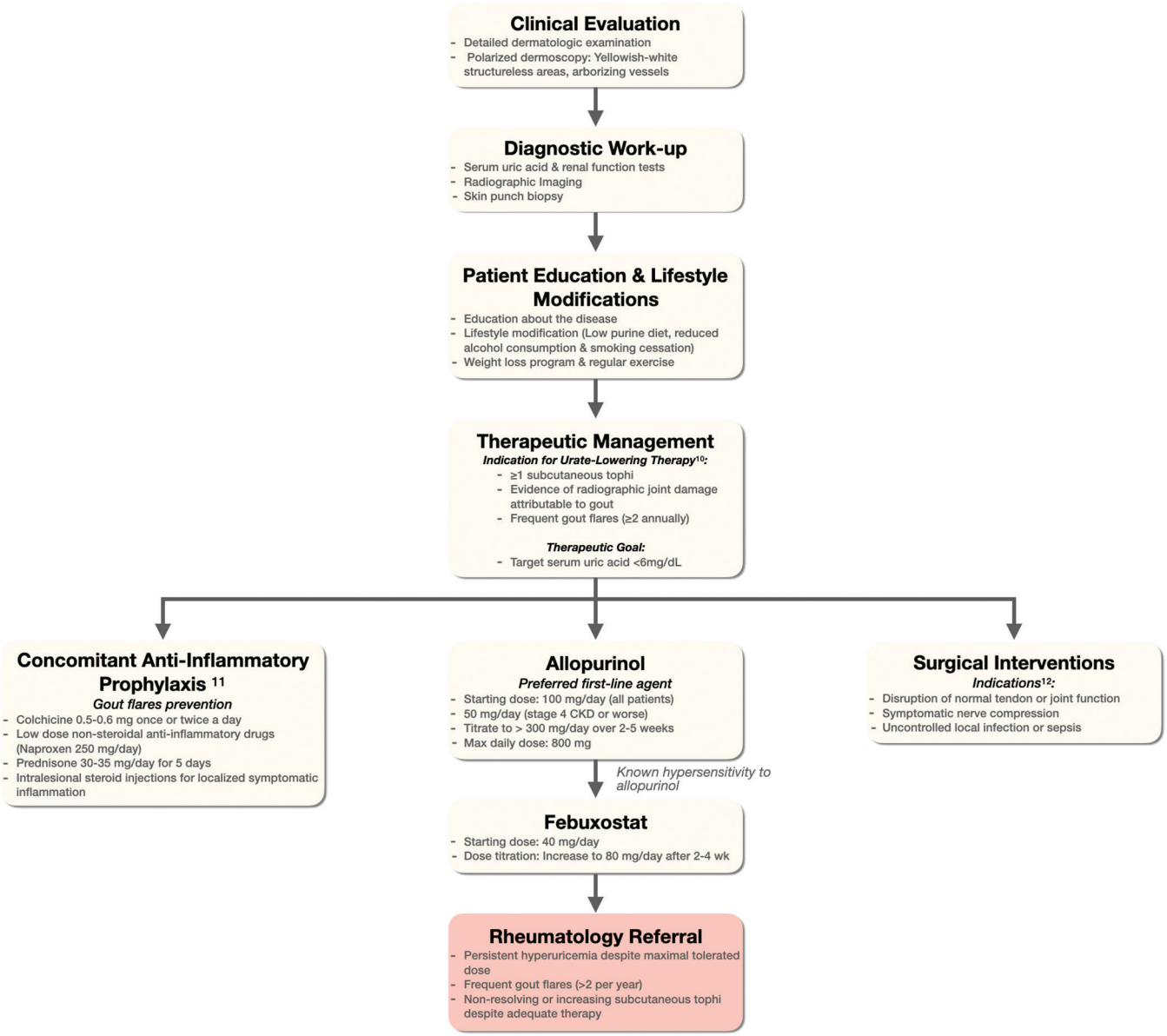

Figure 4: Proposed algorithm for dermatologic management of cutaneous gout. CKD: Chronic kidney disease

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given her consent for her images and other clinical information to be reported in the journal. The patient understands that name and initials will not be published and due efforts will be made to conceal identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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