# Miliarial Gout in an Immunocompromised Patient

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## PRACTICE **POINTS**

- Miliarial gout is a rare intradermal manifestation of tophaceous gout and often presents as multiple small papules containing a white- to cream-colored substance.
- Immunocompromised status may be a risk factor for miliarial gout, especially in patients with a history of gout or hyperuricemia.
- Effective treatments for miliarial gout include allopurinol and colchicine.

To the Editor:

Miliarial gout is a rare intradermal manifestation of tophaceous gout. It was first described in 2007 when a patient presented with multiple small papules with a red base containing a white- to cream-colored substance,[1] which has rarely been reported,[1-6] according to a PubMed search of articles indexed for MEDLINE from 2007 to 2023 using the term *miliarial gout*. We describe a case of miliarial gout in a patient with a history of gout, uric acid levels within reference range, and immunocompromised status due to a prior orthotopic heart transplant.

A 59-year-old man presented with innumerable subcutaneous, firm, popcornlike clustered papules on the posterior surfaces of the upper arms and thighs of 5 years' duration (Figure 1). The involved areas were sometimes painful on manipulation, but the patient

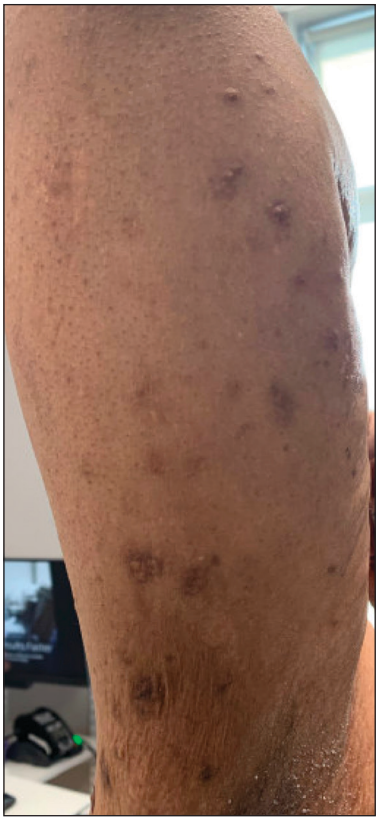

**FIGURE 1.** Miliarial gout. Multiple subcutaneous, firm, popcornlike papules on the right posterior upper arm.

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was otherwise asymptomatic. His medical history was notable for tophaceous gout of more than 10 years' duration, calcinosis cutis, adrenal insufficiency, essential hypertension, and an orthotopic heart transplant 2 years prior to the current presentation. At the current presentation he was taking tacrolimus, colchicine, febuxostat, and low-dose prednisone. The patient denied any other skin changes such as ulceration or bullae. In addition to the innumerable subcutaneous papules, he had much larger firm deep nodules bilaterally on the elbow (Figure 2). A complete blood cell count with differential and comprehensive metabolic panel results were within reference range. A 4-mm punch biopsy of the right posterior arm revealed dermal deposits consistent with gout on hematoxylin and eosin staining (Figure 3) but no calcium deposits on von Kossa staining, consistent with miliarial gout.

He was treated with 0.6 mg of colchicine daily, 80 mg of febuxostat twice daily, and 2.5 mg of prednisone daily. Unfortunately, the patient had difficulty affording his medications and therefore experienced frequent flares.

Gout is caused by inflammation that occurs from deposition of monosodium urate crystals in tissues, most commonly occurring in the skin and joints. Gout affects 8.3 million individuals and is one of the most common rheumatic diseases of adulthood. The classic presentation of the acute form is monoarticular with associated swelling, erythema, and pain. The chronic form (also known as tophaceous gout) affects soft tissue and presents with smooth or multilobulated nodules.[2] Miliarial gout is a rare variant of chronic tophaceous gout, and the diagnosis is based on atypical location, size, and distribution of tophi deposition.

In the updated American College of Rheumatology criteria for gout published in 2020, tophi are defined as draining or chalklike subcutaneous nodules that typically are located in joints, ears, olecranon bursae, finger pads, and tendons.[3] The term *miliarial gout*, which is not universally defined, is used to describe the morphology and distribution of tophi deposition in areas outside of the typical locations defined by the American College of Rheumatology criteria. *Miliarial* refers to the small, multilobulated, and disseminated presentation of tophi. The involvement of atypical locations distinguishes miliarial gout from chronic tophaceous gout.

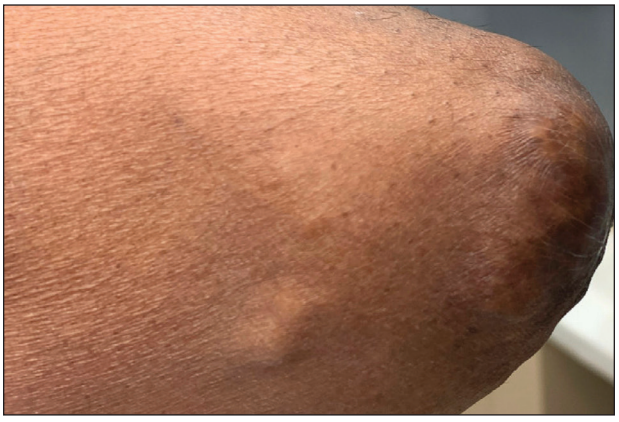

**FIGURE 2.** Firm nodules consistent with miliarial gout on the right elbow.

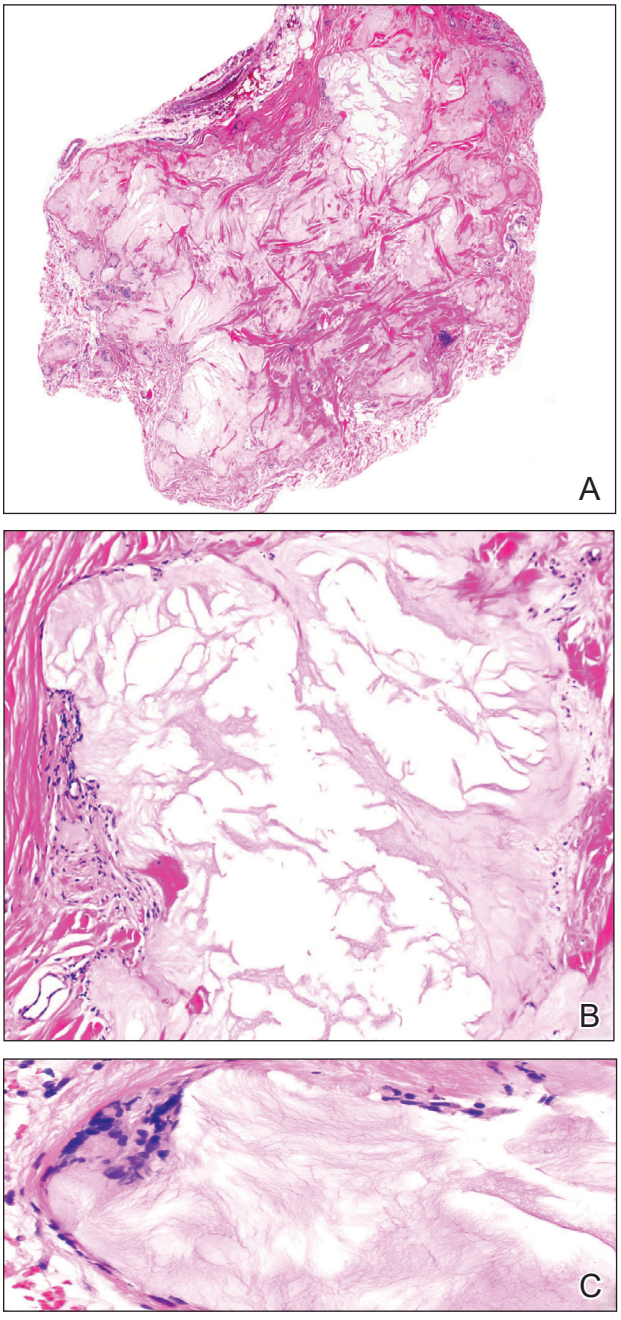


**FIGURE 3.** A, Low-power histopathology revealed nodular aggregates of acellular material with areas varying in color from pale to eosinophilic (H&E, original magnification ×2). B, On closer inspection, the acellular material showed a feathery appearance with prominent clefts and empty spaces (H&E, original magnification ×10). C, There was a multinucleated (foreign body–type) giant cell reaction around the amorphous material (H&E, original magnification ×40).

The cause of tophi deposition in atypical locations is unknown. It is thought that patients with a history of sustained hyperuricemia have a much greater burden of urate crystal deposition, which can lead to involvement of atypical locations. Our patient had innumerable, discrete, 1- to 5-mm, multilobulated tophi located on the posterior upper arms and thighs even though his uric acid levels were within reference range over the last 5 years.

Miliarial gout is a rare entity.[1] In 2007, Shukla et al[1] coined the term *miliarial gout* when reporting the first known presentation of a patient with multiple tiny papules containing a white or creamlike substance scattered on an erythematous base. Other cases of miliarial gout have commonly involved the metacarpophalangeal joints of the hands, knees, abdomen, extensor forearms, and thighs.[5] Similarly, our patient had disease involvement of the posterior upper arms and thighs. Furthermore, miliarial gout has been associated with carpal tunnel syndrome; monosodium urate crystal deposition in this space can lead to a clinical diagnosis of this condition.[6]

With a history of orthotopic heart transplant, it is possible that our patient's immunocompromised status could have increased his susceptibility for the miliarial form of chronic tophaceous gout. Gout reportedly is the most common inflammatory arthritis in transplant recipients, with the highest prevalence following renal and heart transplantation.[7] Pretransplant hyperuricemia is correlated with higher probabilities of posttransplant gout.[8] In patients with a heart transplant, hyperuricemia may be due to diuretic use. Additionally, the presence of a gout diagnosis before transplant nearly triples the likelihood of posttransplant gout, which often is more severe than de novo gout, as seen in our patient. Calcineurin inhibitors, including tacrolimus, also can predispose patients to hyperuricemia and more severe forms of gout in the posttransplant phase by limiting fractional urate excretion within the first 3 months of therapy.[7] Treatment with oral steroids, as in our patient, also has been identified as a potential inciting factor for the development of cutaneous tophaceous gout.[9]

Treatment with allopurinol and colchicine has been effective in patients with miliarial gout. Obesity and long-term treatment with furosemide (which our patient was not taking) are considered risk factors for the deposition of dermal and hypodermal urates.[9] Our patient had a body mass index of 35 (≥30 indicates obesity); therefore, he also should be counseled on lifestyle modifications for optimal disease control.

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