

Case Report

Decoding skin deposits: a rare case of miliarial gout

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ABSTRACT

Gout, metabolic disorder characterized by hyperuricemia and deposition of monosodium urate crystals in joints, soft tissue, and peri articular tissues causing recurrent arthritis. Here, we present a case of disseminated gout with distinctive cutaneous lesions and systemic comorbidities. A 50-year-old chronic alcoholic male presented with multiple white to yellowish papules with extrusion of chalky white materials all over body with soft tissue swelling over right elbow and distal joints for 3 years with joint pain. On clinical examination, multiple yellowish-white, non-tender, hard papules and nodules on an erythematous base present along the forearms, shoulders, and upper back with discharge of white chalk-like material. There were multiple soft tissue swellings over the right elbow and interphalangeal joints of hands and feet with flexion deformity and limitation of movement. Diffuse grain like aggregations of whitish gritty substance noted over the palms and soles. Musculoskeletal examination revealed swelling, tenderness, and warmth over both ankles, elbows, metatarsophalangeal (MTP), and metacarpophalangeal (MCP) joints. Other systemic and neurological examinations were unremarkable. Investigations showed increased uric acid level. With X-ray and histopathological examination, diagnosis of tophaceous gout (miliary) was made and managed accordingly. Various forms of chronic tophaceous gout have been described: classic periarticular subcutaneous tophi, disseminated intradermal tophi, ulcerative form, and gouty panniculitis. Miliarial gout is an extremely rare form of tophaceous gout that manifests as "milia-like" widely distributed papules containing white to cream-colored material on an erythematous base.

Keywords: Miliary gout, Tophaceous gout, Skin deposits, Calcinosis cutis

INTRODUCTION

Gout, a metabolic disorder characterized by hyperuricemia and deposition of monosodium urate crystals in joints, soft tissue, and peri articular tissues causing recurrent arthritis.¹ Common cutaneous manifestations include intradermal or subcutaneous collection of urate crystals known as Tophi. Tophaceous gout can present in various forms, including papular, pustular, ulcerative, bullous, fungating, post-traumatic, nodular, and the rare miliarial form.²

Here, we present a case of disseminated miliarial gout with distinctive cutaneous lesions and systemic comorbidities.

CASE REPORT

A 50-year-old male visited our OPD with complaints of gradual eruption of multiple white to yellowish skin lesions over bilateral arms and legs, back, palms, and soles for the past 5 months. He also had soft tissue swellings over the right elbow and distal joints. Few skin lesions were ulcerated and exuded chalky white material. The patient had a history of recurrent episodes of painful red, swollen large joints and small joints over the past 3 years, initially involving large joints such as the knee, and ankle and later involving the elbow, metacarpophalangeal joints, and metatarsophalangeal joints with the restriction of joint movement. He had self-medicated with NSAIDs for the

joint pain and had not obtained medical consultation until the eruption of skin lesions. He is chronic alcoholic and a hypertensive on treatment. He has no other comorbidities and no significant family history.

On clinical examination, multiple yellowish-white, non-tender, hard papules and nodules on an erythematous base from 1×1 cm to 4×4 cm along the forearms, shoulders, and upper back with discharge of white chalk-like material were noted. There were multiple soft tissue swellings measuring 6×5×3 cm over the right elbow and 2×2 cm to 4×4 cm over interphalangeal joints of hands and feet with flexion deformity and limitation of movement (Figures 1a-e). There were multiple grain-like aggregations of whitish gritty substance over the palms and soles. Musculoskeletal examination revealed swelling, tenderness, and warmth over both ankles, elbows, MTP, and MCP joints. Other systemic and neurological examinations were unremarkable.



Figure 1 (a-d): Multiple yellowish white nodules with underlying erythema over back, forearm, palms and soles, involving the peri-articular and non-articular regions; and (e) firm subcutaneous nodule in right elbow showing yellowish-white discoloration in the center.

Investigations

Complete blood count indicated anemia and leucocytosis, kidney function test: urea–56, creatinine–1.7 (raised), serum calcium and parathormone levels were normal, vitamin D3–3 ng/ml (too low), ANA was negative, RA factor was positive, CPK MB–15.3 U/l, and serum uric acid–16.2 mg/dl (elevated).

Echocardiogram revealed left ventricular systolic dysfunction. Ultrasound abdomen and pelvis and chest X-ray were – normal. X-rays of distal joints revealed intraarticular joint space reduction, subcutaneous

calcifications, and joint deformities consistent with gouty arthritis (Figure 2).



Figure 2 (a and b): Intraarticular joint space reduction, subcutaneous calcifications, and joint deformities consistent with gouty arthritis.

Histopathology

Skin biopsy revealed the presence of intradermal deposits of amorphous eosinophilic substance surrounded by multinucleated and mononuclear histiocytes and lymphocytes indicating a foreign body-type reaction. Within these eosinophilic deposits, subtle, needle-shaped, loosely packed, empty spaces were observed, indicating previous sites of crystals characteristic of monosodium urate.⁴ Von Kossa staining was negative for calcium. (Figure 3) Based on the histopathology and clinical findings, the diagnosis of intradermal and subcutaneous tophaceous gout was made.

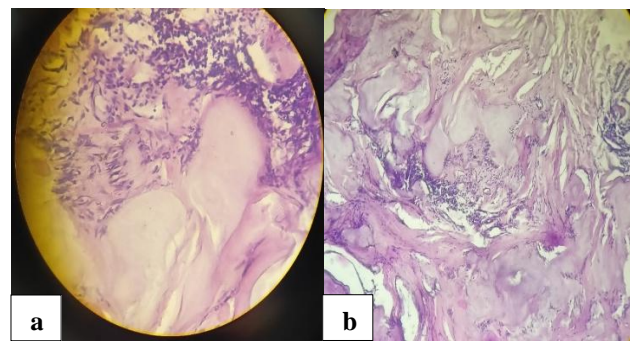


Figure 3 (a and b): Intradermal deposits of amorphous eosinophilic material with needle shaped, loosely packed empty spaces.

Management

The patient was started on tablet febuxostat 40 mg BD and tablet colchicine 0.5 mg BD daily, as recommended by rheumatologist. Additionally, the cardiologist prescribed statins, diuretics, and antiplatelet drugs, with tablet cholecalciferol 60,000 IU given weekly for 4 weeks. Antihypertensives were continued, and dietary advice was provided. The patient was counselled regarding regular follow-up and lifestyle modifications.

After two weeks, the patient's serum uric acid levels reduced to 11 mg/dl, and joint pain and swelling around the ankles also reduced. After a month of follow-up, the patient reported that the nodules became softer and smaller in size, with no new nodules observed after initiating the treatment. The patient is currently on a monthly follow-up with an improvement of symptoms and a decreasing trend in serum uric acid levels.



Figure 4 (a and b): Follow up after one month – marked reduction in size of the nodules and healing ulcers in back and soles.

DISCUSSION

Gout is a systemic disorder caused by abnormal uric acid metabolism. Uric acid forms crystals that accumulate in the joints, leading to recurrent episodes of arthritis. If left untreated, gouty arthritis can cause MSU crystal deposits in the skin, typically over joints or on the ears, leading to chronic cutaneous tophaceous gout. The progression of chronic gout occurs in four stages: hyperuricemia, acute gout, resolution of flares, and chronic gout. Tophaceous gout primarily occurs in individuals with chronic gout. Multiple risk factors have been recognized in the emergence of chronic gout, such as being male, having metabolic syndrome, kidney dysfunction, excessive purine intake, alcohol consumption, and the use of certain medications like cyclosporine. Tophaceous gout should be considered when a patient presents with firm, yellow-white, well-defined papules or nodules on the skin. Cutaneous tophi presenting as yellow skin lesions can be mistaken as calcinosis cutis, milia, osteoma cutis, foreign body granuloma, eruptive xanthomas, necrobiosis lipoidica, pseudoxanthoma elasticum or sebaceous tumours.³ Various forms of chronic tophaceous gout have been described: classic periarticular subcutaneous tophi, disseminated intradermal tophi, ulcerative form, and gouty panniculitis. Miliarial gout is an extremely rare form of tophaceous gout that manifests as "milia-like" widely distributed papules containing white to cream-colored material on an erythematous base.³

Our patient was seen during the chronic tophaceous state exhibiting various forms: periarticular subcutaneous tophi, disseminated intradermal tophi, and miliarial tophi. Chronic tophaceous gout frequently occurs after 10 years

or more of recurrent polyarticular gout with tophi, but in our case, extensive tophi in the skin occurred within 3–4 years of the onset of arthritis.⁴ Serum urate levels are usually elevated in gout. However, tophi can even occur in the absence of hyperuricemia as reported by Kholey et al, limiting the diagnostic utility of measuring serum uric acid levels.⁵ A biopsy is required to confirm the diagnosis, and it is advised to use an ethanol-based fixative as formalin fixation can dissolve the characteristic urate crystals.⁴ Even when fixed in formalin, as was the biopsy in our case, the diagnosis of tophaceous gout can be made based on the characteristic foreign body inflammation surrounding aggregates of eosinophilic material. The diagnosis of cutaneous gout involves identifying amorphous crystalline material in histopathology and negatively birefringent needle-like crystals in polarized microscopy. Gouty arthritis affecting large joints can be mistaken for rheumatoid arthritis, and tophi can be confused with calcinosis cutis. However, serological, biochemical, and radiological investigations can differentiate between the two conditions. In our patient, rheumatoid arthritis with calcinosis cutis was ruled out due to normal serum calcium and parathyroid hormone levels, as well as negative von Kossa stain. Gouty arthritis was diagnosed based on elevated serum uric acid and X-ray findings, and tophaceous gout was confirmed by histopathological examination showing amorphous, pinkish, crystalline material in the dermis, surrounded by granulomatous inflammation. Additionally, our patient had systemic hypertension, heart failure, vitamin D deficiency, and early-stage renal insufficiency as identified comorbidities.

Acute inflammatory gouty arthritis attacks are typically treated with colchicine and nonsteroidal anti-inflammatory agent. Managing chronic gout focuses on reducing and maintaining serum uric acid at lower levels, with a target level of <6 mg/dl. Previous reports have suggested that using a xanthine oxidase inhibitor like allopurinol in combination with an anti-inflammatory such as colchicine can be effective. In our patient, a combination of colchicine and febuxostat proved to have better patient compliance and improvement in skin lesions, as allopurinol is considered to have a higher risk of causing drug reactions. Thus, we preferred febuxostat for management. The use of febuxostat for managing miliarial gout has not been reported in the literature so far.

Limitations

Polarized microscopy was unavailable at our center, hence negatively birefringent crystals could not be visualized.

CONCLUSION

We are reporting this case due to its extensive cutaneous involvement with a short duration of gouty arthritis, atypical cutaneous presentation in the form of multiple sinuses, and miliarial tophi in the skin, in addition to periarticular subcutaneous nodules and swellings with successful improvement with the safer xanthine oxidase

inhibitor febuxostat. Clinicians are recommended to consider miliarial gout in patients with a known history of hyperuricemia or gout and request a fixation technique from the histopathologist that facilitates proper diagnosis. Overall, prompt identification and aggressive treatment of hyperuricemia are important in reducing disseminated cutaneous gout and potential systemic sequelae.

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