An uncommon presentation of cutaneous dissemination of gout

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INTRODUCTION

Gout is a common inflammatory disease caused by the deposition of monosodium urate crystals in joints and soft tissues. Classically, gout manifests as joint pain, or collections of urate crystals called tophi. Rarely, it may present as disseminated cutaneous papulonodules and/or ulcerations.[1,2] Here, we present a case of disseminated gout with unique cutaneous morphologies and hyperuricemia, which may have been contributing to systemic comorbidities.

CASE REPORT

A 45-year-old man with a history of gout (on allopurinol 300 mg daily and colchicine 0.6 mg daily), morbid obesity, and osteoarthritis was admitted to the cardiac intensive care unit for unexplained new-onset nonischemic heart failure with an ejection fraction of 10% to 15%, cardiogenic shock, and sustained ventricular tachycardia. Dermatology was consulted for the evaluation of a single multinodular asymptomatic plaque involving the upper portion of the left arm, present for 1 year.

The skin examination revealed asymptomatic lesions with multiple morphologies, covering approximately 25% of the body surface area. The most numerous and prominent lesions were 5- to 12-cm, skin-colored to light brown, indurated dermal to subcutaneous plaques involving the arms, chest, back, and legs, most of which were discernible by palpation only (Fig 1, *A*). There was a single slightly hyperpigmented plaque on the upper lateral aspect of the left side of the back, with numerous superimposed 5- to 10-mm punched-out appearing depressions resulting in a moth-eaten appearance (Fig 1, *B*). There were scattered mildly erythematous plaques studded with 2- to 4-mm, firm rough-surfaced aggregations of a whitish gritty substance (Fig 1, *C*). On the lower portion of the left leg, there was a 2-cm superficial ulceration extruding a whitish gritty substance (Fig 1, *D*). In addition, ventral pterygia of most fingernails (Fig 2) and bilateral enlargement of the lacrimal glands (Fig 3) were noted. No tophi or any joint deformities were appreciated. He denied any gout flares of the joints because these skin findings appeared.

Punch biopsies were obtained from lesions representative of the multiple cutaneous morphologies. Histology from all clinical morphologies revealed many feathery deposits of eosinophilic material surrounded by multinucleated cells in the dermis, characteristic of gout (Fig 4). Serum uric acid levels were 12.6 mg/dL (reference range, 3.8-8.7 mg/dL). X-rays of the hands demonstrated periarticular erosions consistent with gout arthropathy; X-rays of the feet were suggestive of gout.

He received a 1-time dose of 1 g intravenous methylprednisolone followed by prednisone (40 mg daily tapered over 6 days). Allopurinol dosing was increased to 400 mg daily, and colchicine 0.6 mg daily was continued. He reported that the cutaneous lesions became softer (less indurated) after the initiation of this regimen.

Hyperuricemia was considered a contributing factor to his heart failure and ventricular tachycardia. However, his morbid obesity prohibited cardiac

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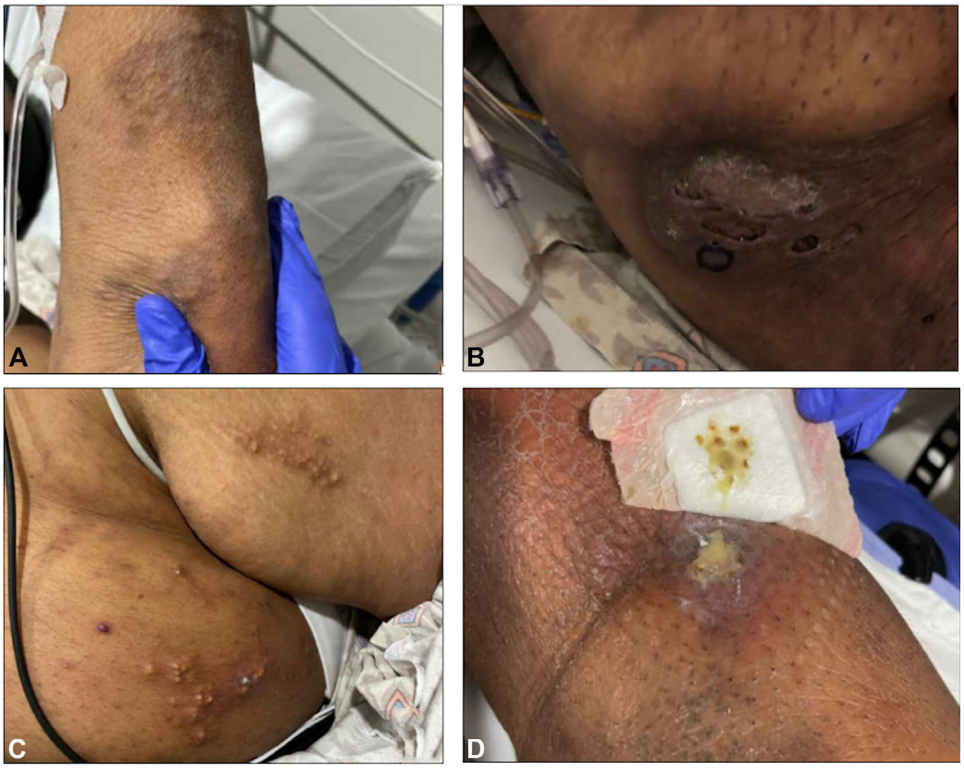

Fig 1. Multiple cutaneous morphologies, which all revealed gout on histopathologic examination. **A,** Skin colored indurated dermal plaques, most discernible by palpation. **B,** Hyperpigmented plaque of the left back with superimposed punched-out appearing depressions. **C,** Mildly erythematous plaques studded with firm aggregations of a whitish gritty substance. **D,** Superficial erosion of the right leg extruding a whitish, gritty substance.

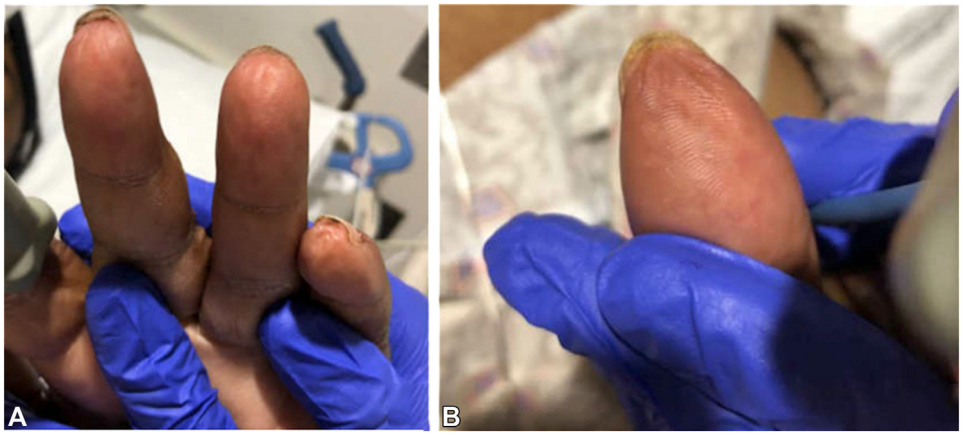

Fig 2. A, Ventral pterygium of multiple fingers. **B,** Close up of ventral pterygium.

magnetic resonance imaging and the medical team determined that the benefits of cardiac biopsy would not outweigh the risks. Thus, the exact etiology of his cardiac comorbidities remains unknown.

Given the findings of lacrimal gland hypertrophy and ventral pterygium, sarcoidosis and other connective tissue disorders were considered. However, the antinuclear antibody was negative, calcium levels were unremarkable (9-10 mg/dL) and the angiotensin-converting enzyme level was 88 U/L (considered to be equivocal). Following hospital discharge, he moved to a different state and was lost to follow-up, so we were unable to follow his clinical course or to further elucidate the etiology of his lacrimal gland hyperplasia, heart failure, and ventral pterygium.

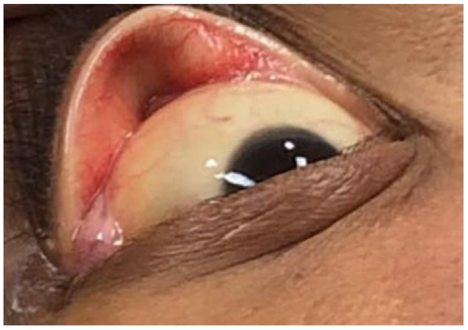

Fig 3. Lacrimal gland hypertrophy.

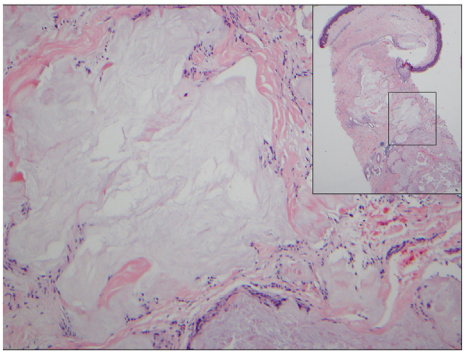

Fig 4. Hematoxylin-eosin stain, 100× magnification (40× magnification inset) revealed gout in all punch biopsy specimens.

DISCUSSION

Cutaneous dissemination is a rare, severe manifestation of gout.[2,3] Such cases generally present with widespread papulonodules and ulcers involving extraarticular sites. This contrasts with classic tophi, which typically involves cartilage, tendons, and periarticular soft tissue. The cutaneous findings in our patient, including indurated morphea form plaques distributed extensively over the trunk and extremities, as well as scattered erythematous plaques punctuated by aggregations of extruding uric acid crystals, appear to be unique, although the morphea form plaques (most of which were palpable but not visible) would have been easy to miss in a cursory skin examination.

Other unique findings, in this case, include that of lacrimal gland hyperplasia and ventral pterygium. It is unclear to our patient whether the ocular findings represented urate crystal deposition. However, gout has been described to affect the periocular soft tissues, canthi, conjunctivae, cornea, lens, retina, and iris.[4]

Ventral pterygium, which occurs when the hyponychium adheres to the nail plate,[5] can be an acquired manifestation of disease, idiopathic, or even familial.[5] To our knowledge, it has not previously been reported in association with gout.

Hyperuricemia has been associated with cardiovascular dysfunction.[1,6-8] A meta-analysis of >400,000 patients found that hyperuricemia was significantly associated with an increased risk of atrial fibrillation with a relative risk of 1.49 (95% CI, 1.24-1.79).[9] In addition, a patient with a uric acid level of 9.4 mg/dL, a left ventricular ejection fraction of 30%, and clinical signs of heart failure was found to have monosodium urate crystals deposition in the myocardium on biopsy.[8] Although we were unable to conclusively demonstrate extracutaneous uric acid deposits in our patient, it is possible that these contributed to his unexplained severe heart failure and sustained ventricular tachycardia. In the appropriate setting, gout may be considered as a potential etiology for unexplained congestive heart failure, and considered in the differential diagnosis of cutaneous disseminated indurated plaques. The presence of erythematous plaques studded with aggregations of a whitish gritty substance (uric acid crystals) is another clue to the diagnosis of disseminated gout. A skin biopsy can confirm the diagnosis. Overall, prompt identification and aggressive treatment of hyperuricemia are important in mitigating disseminated cutaneous gout and potential systemic sequelae.

Conflicts of interest

None disclosed.

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