Chronic gout and rheumatoid arthritis are common medical manifestations with debilitating effects on patients. However, these conditions are not typically identified concomitantly and can be hard to distinguish from one another. We report a rare case of a 50-year-old white woman with a history of chronic gout and rheumatoid arthritis who presented with intradermal tophaceous gout. Physical examination and laboratory results are described.

Gout, which is caused by the overproduction of uric acid, is a common medical diagnosis that can have a presentation similar to rheumatoid arthritis (RA).1,2 As a chronic, systemic, inflammatory disease, RA affects the synovial membranes and causes muscle atrophy and bone weakness. This condition can lead to significant debilitation in patients if not identified early and managed appropriately.3 Although both gout and RA are commonly observed in the general population, they rarely coexist in the same patient.

Tophaceous gout is a less common condition that causes skin lesions called tophi. This disease occurs in patients with chronic gout and has been reported on finger pads, arms, thighs, buttocks, and the abdomen.4 Although typically painless, these lesions may ulcerate and drain with a white, chalky, urate matter.5

We describe a rare occurrence of concomitant gout and RA in a patient with multiple intradermal tophi. Although a few concurrent cases of gout and RA have been described,2,4,5 the present report is, to our knowledge, the first published incident of coexistent intradermal tophaceous gout and seronegative RA.

Report of Case
A 50-year-old white woman with severe anemia was transferred from an outlying medical center to the Oklahoma State University (OSU) Medical Center in Tulsa. The patient complained of shortness of breath and severe fatigue. Her medical history was significant for RA, which was diagnosed 16 years earlier, as well as gout, depression, and hypertension, which were of unknown durations. The patient was on various medications, including adalimumab injections for RA (40 mg every other week), allopurinol tablets for gout (300 mg twice daily), amitriptyline hydrochloride tablets for depression (150 mg/d at bedtime), clonidine tablets for hypertension (0.2 mg daily), and acetaminophen tablets as needed for pain. In addition, she was administered 4 units of packed red blood cells for anemia just before she was transferred to the OSU Medical Center.

On admission at the OSU Medical Center, the patient’s blood pressure was 160/96 mm Hg; heart rate, 105 beats per minute; body temperature, 102.9°F; and respiratory rate, 24 breaths per minute. On physical examination, the patient was noted as obese, alert and oriented, and in no acute distress. She had multiple pustules with a yellow center on her abdomen and fingers (Figure 1). Her wrists were bilaterally swollen and tender, and she complained of decreased range of bilateral motion in her metacarpophalangeal joints. She also had bilateral swelling and pain in her knees.

Laboratory test results on admission revealed a creatinine value of 1.8 mg/dL; erythrocyte sedimentation rate, 106 mm/h; white blood cell count, 15,200/μL; and hemoglobin, 9.6 g/dL.

Radiographic scans of the patient’s hands revealed bilateral juxta-articular osteopenia involving the metacarpophalangeal regions and bilateral symmetric radial and ulnar carpal joint space loss with associated erosive changes consistent with RA (Figure 2). The first metacarpophalangeal joint in the patient’s left hand was subluxated and loss of the ulnar styloid was visible. Radiographic scans of the patient’s feet also uncovered symmetric joint space narrowing with osteopenia (Figure 3). A wet mount specimen with polarized light microscopy of the punch biopsy for an abdominal skin lesion revealed monosodium urate crystals, which are indicative of tophaceous gout (Figure 4).

The patient’s daily dose of amitriptyline was changed to 75 mg—50% of the dose prescribed to her at home—because of her previous noncompliance with prescribed medications. On day 2 of admission, a gastroenterology consultation was obtained and an esophagogastroduodenoscopy was performed.
formed, which showed mild antral gastritis. The patient was administered pantoprazole sodium, 40 mg/d, for gastritis and severe anemia and to prevent gastrointestinal bleeding. The patient was also administered ceftriaxone sodium, 1 g/d, as empiric therapy as a result of the elevated white blood cell count and fever. All other medications were continued as previously described.

Because the patient had a history of gout and RA as well as an elevated serum creatinine level, consultations were obtained from the rheumatology and nephrology services 4 days after admission. Laboratory results from these consultations revealed a rheumatoid factor of $<20$ IU/mL; reticulocyte count, 3.08%; lactate dehydrogenase, 263 U/L; serum uric acid, 6.8 mg/dL; 24-hour urine protein, 2074 mg; and 24-hour urine uric acid, 432 mg. The low rheumatoid factor value indicated seronegative RA. The patient received prednisone, 40 mg/d, and colchicine, 0.6 mg/d, combination therapy for her rheumatologic conditions.

Although the patient’s uric acid levels were within the normal range, tophaceous gout was also diagnosed as a result of the presence of monosodium urate crystals. In addition, the patient’s elevated lactate dehydrogenase level and reticulocyte count indicated hemolytic anemia. A Coombs test was not performed because the patient had already received corticosteroids. Results from a peripheral smear suggested normocytic anemia with granulocytosis and thrombocytosis. However, this result may be attributed to the fact that the smear was performed after administration of steroids and packed red blood cells.

The patient was discharged 6 days after admission in stable condition. She had decreased shortness of breath, fatigue, and joint pain, as well as improved but not completely resolved tophi lesions. Because of the high serum creatinine level and the patient’s noncompliance with her previous home medication regimen, the daily dose of allopurinol was decreased to 100 mg. Similarly, colchicine (0.6 mg/d) was used as an acute therapy and as a prophylactic agent with the “initiation” of allopurinol therapy (i.e., the colchicine was used as if the patient was just started on allopurinol therapy in an acute flare). The patient’s other medications on discharge included prednisone, 40 mg/d for 2 weeks, and weekly doses of alendronate, 70 mg, and etanercept, 50 mg.

**Comment**

Symmetric arthritis and swelling in the patient’s metacarpophalangeal joints confirmed her history of RA. The suspected hemolytic anemia also supported a diagnosis of RA. The radiologic findings of periarticular osteopenia and symmetric

Figure 1. *Intradermal gout lesions on the (A) abdomen and (B) fingers of a 50-year-old woman. Punch biopsy of an abdominal skin lesion revealed monosodium urate crystals.*

Figure 2. *Radiographic image of a 50-year-old woman’s right hand. Bilateral juxta-articular osteopenia in the metacarpophalangeal regions is visible. Bilateral symmetric radial and ulnar carpal joint space loss with associated erosive changes consistent with rheumatoid arthritis are observed.*
however, typically presents with asymmetric erosive lesions that are “punched out” in appearance and have sclerotic margins.10 Although the patient’s uric acid level was normal, the history of gout was confirmed through skin biopsy by the presence of monosodium urate crystals.

The reason for the lack of coexistence between these two diseases is still disputed. Hyperuricemia may produce an immunosuppressive effect on RA because rheumatoid factor is decreased in these patients.11 High concentrations of uric acid may function as an antioxidant and a free radical scavenger.12 There is also evidence to suggest that monosodium urate crystals may bind antigens such as immunoglobulin G and may block the activation of B and T cells, which are prominent in patients with RA.12

Conclusion
Tophaceous gout is a rare clinical manifestation that has been observed in patients with a history of chronic gout. Although chronic gout and RA are common clinical entities, they seldom coexist.2-8 In understanding the specific—yet sometimes similar—presentations of both conditions, physicians can better diagnose and treat their patients.

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References