Sarcoidosis — a chronic, multisystem disease of unknown etiology characterized by noncaseating granulomas — may cause ulcerative lesions, particularly in African American women. A case of ulcerative sarcoidosis mimicking a venous ulcer is presented. The patient is a 44-year-old African American hypertensive, obese woman with a nonhealing medially based lower leg ulcer of 3 years’ duration clinically consistent with a venous ulcer. The ulcer did not heal with compression therapy and pentoxifylline. Subsequent biopsies showed granulomatous inflammation consistent with sarcoidosis. When intralesional triamcinolone was added to compression therapy, the ulcer resolved after 3 months. Given its propensity toward formation on the lower extremities and ulcerative and atrophic appearance, ulcerative sarcoidosis should be considered in the differential diagnosis of a venous ulcer refractory to standard therapy, especially in African American women.

**Abstract**

Sarcoidosis is a chronic, multisystem disease of unknown etiology characterized by noncaseating granulomas. Cutaneous lesions are seen in 20% to 35% of patients and are categorized as *specific* and *nonspecific*. Specific lesions have granulomas on biopsy and include maculopapules, lupus pernio, scar infiltration, and ulcerative lesions. Nonspecific findings are reactive phenomena without sarcoidal granulomas; erythema nodosum is the most common. Although cutaneous sarcoidosis is not uncommon, ulcerative atrophic lesions are very rare. A retrospective study of 147 patients with cutaneous sarcoidosis demonstrated an ulcerative-atrophic sarcoidosis prevalence rate of 4.8% (seven cases).

Ulcerative lesions typically are seen in African Americans and in women. The most commonly affected areas are the lower extremities — more specifically, the pretibial areas. Clinically, ulcerative sarcoidosis has been reported to present as lesions with atrophic bases resembling necrobiosis lipoidica, punched-out ulcers, and ulcers with necrotic bases, raised hyperpigmented borders, and serosanguinous fluid. Ulcerative sarcoidosis is likely due to trauma superimposed on atrophic plaques. Diagnosis is associated with systemic involvement. A case of ulcerative sarcoidosis mimicking a venous ulcer is presented to elucidate differential diagnosis.

**Case Report**

Ms. J was a 44-year-old African American woman with history of hypertension and obesity. She presented with painful, nonhealing leg ulcers on the left medial lower leg of 3 years’ duration. In the past, she had undergone compression treatments and debridement for clinically suspected venous ulcers. However, the ulcers remained recalcitrant to treatment. A review of her symptoms and past medical history were otherwise unremarkable.

Physical examination of Ms. J’s left medial leg showed an indurated hyperpigmented plaque with an overlying 1.5-cm x 1.5-cm partial-thickness ulcer (see Figure 1). A 1.5-cm x 1.5-cm shallow ulcer with surrounding hyperpigmentation was present lower on the leg. Ms. J had minimal...
lower extremity edema and normal distal pulses. Physical examination was consistent with a venous insufficiency ulcer; Ms J was treated with pentoxifylline (Trental, Sanofi-Aventis, Bridgewater, NJ) 400 mg three times daily, topical cadexomer iodine (Iodosorb, Smith & Nephew, Largo, FL), and multilayer compression bandages (Profore, Smith & Nephew, Largo, FL dressings) twice weekly.

At follow-up 1 month later, Ms. J’s ulcer had increased in size to 4 cm². A biopsy revealed granulomatous dermatitis with features consistent with subcutaneous sarcoidosis (see Figure 2). Ms. J’s comprehensive metabolic panel was within normal limits, her angiotensin-1 converting enzyme level was low (5 U/L, normal range 9–67 U/L), and chest radiograph was normal. The combination of Ms. J’s history and histological findings led to the diagnosis of subcutaneous sarcoidosis. No evidence of systemic involvement was noted.

The ulcer received a single injection of 3 cc of intraleisonal triamcinolone 10 mg/cc. Mycobacterial and fungal cultures were negative. Ms. J was treated with compression wraps and healed at follow-up when seen 3 months later. The ulcer remained healed during the subsequent 6 months of follow-up (see Figure 3).

Discussion
Ulcerative lesions are a rare manifestation of sarcoidosis; however, Ms. J, an African American woman, had demographic features typical of the ulcerative form of the disease. Unlike most reported cases of ulcerative sarcoidosis, Ms. J did not have any evidence of systemic involvement, which may have complicated and delayed her diagnosis. Unlike previous cases that were refractory to steroidal treatment with intraleional triamcinolone, topical steroids with aluminum subacetate soaks, and hydroxychloroquine with topical corticosteroids, Ms. J had complete resolution of the ulcer with intraleional triamcinolone and compression wraps.

Ulcerative sarcoidosis also has been reported to be recalcitrant to nonsteroidal therapy with isotretinoin, allopurinol, x-ray, and hydroxychloroquine. Use of methotrexate, split-thickness skin grafting, and oral prednisone all have been met with varying success. Combination therapy with prednisone, hydroxychloroquine, and either mycophenolate mofetil or thalidomide has been reported to be an efficacious treatment regimen.

Conclusion
Based on the clinical findings, ulcerative sarcoidosis masqueraded as a venous ulcer in this patient for years. She underwent standard therapy for venous ulcers including compression therapy with pentoxifylline and experienced no improvement of symptoms, which prompted further diagnostic work-up. Whether she had concomitant venous disease is not known. Given the propensity to form on the lower extremities and its ulcerative and atrophic appearance, ulcerative sarcoidosis should be considered in the differential diagnosis of a venous ulcer recalcitrant to standard therapy, especially in African American women.
References


**Figure 2a,b.** Low power histology (2A) reveals pseudocarcinomatous hyperplasia, noncaseating well demarcated granulomatosus infiltrate in the dermis. Higher magnification (2B) shows that the infiltrate is composed mainly of histiocytes, lymphocytes, and many foreign body-type giant cells. There is mild dermal fibrosis.

**Figure 3a,b.** Indurated atrophic plaque with central pink scar, white scale, and surrounding hyperpigmentation on the left medial leg.