Ulcerative Sarcoidosis: A Prototypical Presentation and Review

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PRACTICE **POINTS**

- Sarcoidosis can present as a primary ulcerative disease.
- Suspect ulcerative sarcoidosis when ulcerations are seen on the leg.
- Systemic corticosteroids may be the most effective treatment of ulcerative sarcoidosis.

Although rare, ulcerative sarcoidosis is an acknowledged morphologic variant of cutaneous sarcoidosis encountered in both the United States and worldwide, particularly in patients with skin of color. Herein, we present a patient with prototypical ulcerative sarcoidosis to highlight this unusual presentation of a relatively rare cutaneous condition. We also review 34 additional cases drawn from the English-language literature to define historical presentation, associated findings, treatments, and outcomes.

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Sarcoidosis is a multisystem granulomatous disorder of unknown etiology that primarily affects the lungs and lymphatic system but also may involve the skin, eyes, liver, spleen, muscles, bones, and nervous system.[1] Cutaneous symptoms of sarcoidosis occur in approximately 25% of patients and are classified as specific and nonspecific, with specific lesions demonstrating noncaseating granuloma formation, which is typical of sarcoidosis.[2] Nonspecific lesions primarily include erythema nodosum and calcinosis cutis. Specific lesions commonly present as reddish brown infiltrated plaques that may be annular, polycyclic, or serpiginous.[1,3] They also may appear as yellowish brown or violaceous maculopapular lesions. However, specific lesions may present in a wide variety of morphologies, most often papules, nodules, subcutaneous infiltrates, and lupus pernio.[4] Additionally, atypical cutaneous manifestations of sarcoidosis include erythroderma; scarring alopecia; nail dystrophy; and verrucous, ichthyosiform, psoriasiform, hypopigmented, or ulcerative skin lesions.[3-5] Among these many potential clinical presentations, ulcerative sarcoidosis is quite uncommon.

We report a case of a patient who presented with classic clinical and histopathological findings of ulcerative sarcoidosis to highlight the prototypical presentation of a rare condition. We also review 34 additional cases of ulcerative sarcoidosis published in the English-language literature based on a PubMed search of articles indexed for MEDLINE using the term *ulcerative sarcoid.*[4-32] Analyzing this historical information, the scope of this unusual form of cutaneous sarcoidosis can be better understood, recognized, and treated. Although current standard-of-care treatments are most often successful, there is a paucity of definitive clinical trials to justify and verify comparative therapeutic efficacy.

Case Report

A 49-year-old black man with known pulmonary sarcoidosis, idiopathic (human immunodeficiency virus–negative) CD4 depletion syndrome, and chronic kidney disease presented with persistent bilateral ulcers of the legs of 1 month's duration. The lesions first appeared as multiple "dark spots" on the legs. After the patient applied homemade aloe vera extract under occlusion for 1 to 2 days, the lesions became painful and began to ulcerate approximately 3 months prior to presentation. The patient applied a combination of a topical first aid antibiotic ointment, Epsom salts, and hydrogen peroxide without any improvement. A current review of systems was negative.

The patient's medical history was notable for sarcoidosis diagnosed more than 10 years prior. During this time, he had intermittently been treated elsewhere with low-dose oral prednisone (5 mg once daily), hydroxychloroquine (200 mg twice daily), and an inhaled steroid as needed. He had a history of human immunodeficiency

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virus–negative, idiopathic CD4 depletion syndrome, which had been complicated by cryptococcal meningitis 7 years prior to presentation. He also had renal insufficiency, with baseline creatinine levels ranging from 1.4 to 1.7 mg/dL (reference range, 0.6–1.2 mg/dL). There was no personal or family history of known or suspected inflammatory bowel disease.

On physical examination, numerous discrete, coalescing, punched out–appearing ulcerations with foul-smelling, greenish yellow, purulent drainage were present bilaterally on the legs (Figure 1). The ulcers had a rolled border with a moderate amount of seemingly nonviable necrotic tissue. A number of hyperpigmented round papules, patches, and plaques also were present on the proximal legs. Laboratory evaluation revealed a CD4 count of 151 cc/mm^3 (reference range, 500–1600 cc/mm^3) and mildly elevated calcium of 10.7 mg/dL (reference range, 8.2–10.2 mg/dL).

Aerobic, anaerobic, mycobacterial, and fungal cultures of the purulent exudate were obtained. Given a high suspicion for secondary infection of the exogenous wound sites, doxycycline (100 mg twice daily) and topical mupirocin were initiated. Gram stain revealed few to moderate polymorphonuclear cells and many gram-positive cocci in pairs, chains, and clusters, along with many gram-negative rods. Bacterial culture grew *Pseudomonas aeruginosa, Enterococcus* species group G streptococci, and methicillin-resistant *Staphylococcus aureus*–positive staphylococci. Ciprofloxacin (500 mg twice daily) was then initiated, but the ulcers showed absolutely no clinical improvement and in fact worsened both in number and depth (Figure 2) over subsequent clinic visits during the next 3 months, even after amoxicillin (500 mg 3 times daily) was added. The patient was admitted for treatment with intravenous antibiotics after additional wound cultures revealed fluoroquinolone-resistant *Pseudomonas.*

Punch biopsies of the ulcers showed nonspecific acute inflammation and tissue necrosis in the active ulcers with nonnecrotizing granulomatous inflammation extending into the deep dermis, with many Langerhans-type giant cells present in the palpable ulcer borders (Figure 3). Neither birefringent particles nor asteroid bodies were observed. Tissue Gram stains did not reveal evidence of bacterial infection. Special stains for acid-fast and fungal organisms (ie, periodic acid–Schiff, Gomori methenamine-silver, Fite, acid-fast bacilli) were similarly negative. Tissue cultures obtained on deep biopsy revealed

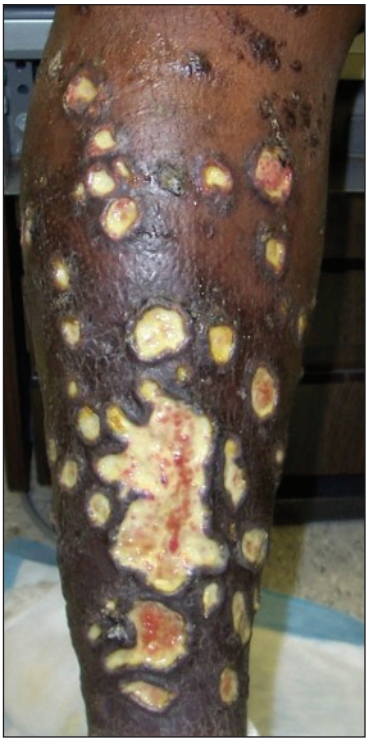

FIGURE 1. Ulcerative sarcoidosis consisting of multiple leg ulcers, with more typical lesions proximally.

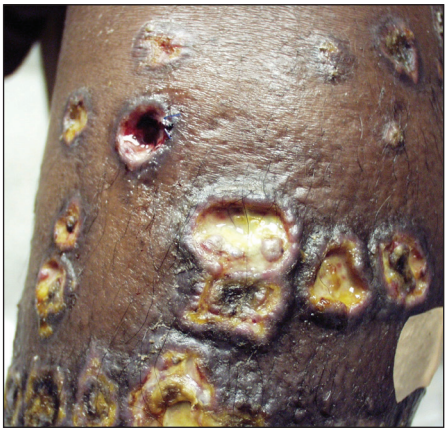

FIGURE 2. Ulcerative sarcoidosis lesions became more numerous and deeper with time.

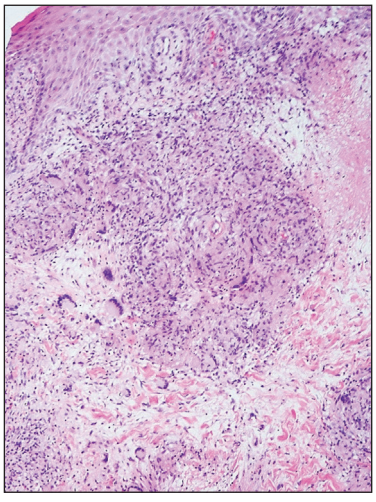

FIGURE 3. Classic noncaseating granuloma in ulcerative sarcoidosis (H&E, original magnification ×40).

only rare colonies of *P aeruginosa* and no isolates on anaerobic, mycobacterial, or fungal cultures. Polymerase chain reaction for mycobacteria and common endemic fungi also was negative. In the absence of infection and considering his history of known sarcoidosis, these histologic features were consistent with ulcerative sarcoidosis. The patient was started on prednisone (60 mg once daily) and hydroxychloroquine (200 mg twice daily). The prednisone was tapered to 20 mg once daily over a 2-year period, at which point 90% of the ulcers had healed. He was continued on hydroxychloroquine at the initial dose, and at a 3-year follow-up his ulcers had healed completely without relapse.

Comment

Ulcerative sarcoidosis is rare, seen worldwide in only 5% of patients with cutaneous sarcoidosis.[33] However, cases have been encountered worldwide, with reports emanating from Japan, China, Germany, France, and Russia, among others.[6,34-55] We reviewed 34 cases from the English-language literature based on a PubMed search of articles indexed for MEDLINE using the term *ulcerative sarcoid* and examined patient demographics, clinical presentation, histological findings, treatment type, and outcome. Key references are presented in the Table. Disease prevalence previously has been estimated as being 3-times more common in women than men[1]; in our literature review, we found a female to male ratio of 3.25 to 1. Additionally, ulcerative sarcoidosis is reported to be twice as common in black versus white individuals.[33] In our literature review, when race was reported, 66% (21/32) of patients were black. Disease prevalence has been reported to peak at 20 to 40 years of age.[3] In this review, the average age of presentation was 45 years (age range, 24–79 years).

Ulceration may arise de novo but more commonly arises in preexisting scars or cutaneous lesions. There are 2 distinct patterns seen in ulcerative sarcoidosis.[4] The first is characterized by ulceration within necrotic yellow plaques.[2] The second pattern is characterized by violaceous nodules arising in an annular confluent pattern that eventually ulcerate.[4] This presentation commonly mimics or may be mimicked by multiple disease states, including sporotrichosis, tuberculosis, stasis dermatitis with venous ulceration, and even metastatic breast cancer.[7,46,55,56] Regardless of presentation, the legs are the most common location of ulcer formation.[1,33] In our review, 85% (29/34) of cases presented with involvement of the legs, including our own case. Other locations of ulcer formation have included the face, arms, trunk, and genital area.

On histologic examination of ulcerative sarcoidosis, epithelioid granulomas composed of multinucleated giant cells, histiocytes, and scant numbers of lymphocytes are present.[1,3] These formations are the noncaseating granulomas typical of sarcoidosis (Table). All of the cases in our review of the literature were described as either a collection of epithelioid granulomas with giant cell formation or noncaseating granulomas. There also have been reports of atypical features including necrotizing granulomas and granulomatous vasculitis.[4,8,9,50] The histologic differential diagnosis in this case also would primarily include an infectious granulomatous process and less so an id reaction, rosacea, a paraneoplastic phenomenon, foreign body granulomas, and metastatic Crohn disease. The presence of ulceration, the large number of lesions, and the anatomic distribution help rule out most of these alternate diagnostic considerations. Diligent extensive workup was done in our patient to insure it was not an infection.

The goals of treatment include symptomatic relief, improvement in objective parameters of disease activity, and prevention of disease progression and subsequent disability.[33,57] Fortunately, the majority of sarcoidosis patients with cutaneous symptoms achieve full recovery within months to years.[33] Our literature review indicated that 81% (22/27) of patients with ulcerative lesions experienced full resolution within 1 year of treatment. Of those that did not (19% [5/27]), the patients were either lost to follow-up or died from other complications of sarcoidosis.

The widely accepted standard therapy for cutaneous sarcoidosis includes topical, intralesional, and systemic corticosteroids; antimalarials; and methotrexate.[33,57] Steroids and methotrexate act by suppressing granuloma formation, while antimalarials prevent antigen presentation (presumably part of the pathogenesis).[33] For mild to moderate disease, topical and intralesional steroids may be all that is necessary.[33,57] Systemic steroids are used for disfiguring, destructive, and widespread lesions that have been refractory to local and other systemic therapies.[33,57] Steroids are tapered gradually depending on the patient's response, as it is common for patients to relapse below a certain dose.[33,57] Antimalarials (chloroquine or hydroxychloroquine) and methotrexate are considered adjunct treatments for patients who are either steroid unresponsive or who are unable to tolerate corticosteroid treatment due to adverse events.[33,57]

Standard therapy is complicated by the side effects of treatment. Use of corticosteroids may lead to gastrointestinal tract upset, increased appetite, mood disturbances, impaired wound healing, hyperglycemia, hypertension, cushingoid features, and acne.[57] Antimalarials can cause nausea, anorexia, and agranulocytosis, and chloroquine therapy in particular can lead to blurred vision, corneal deposits, and central retinopathy.[33,57] Methotrexate is associated with hematologic, gastrointestinal tract, pulmonary, and hepatic toxicities well known to most practitioners.

Because of the variable clinical response of patients to standard therapy and their associated toxicities, other treatment options have been used including pentoxifylline, tetracyclines, isotretinoin, leflunomide, thalidomide, infliximab, adalimumab, allopurinol, and the pulsed dye or CO_2 laser.[10,33,57] In nonhealing ulcers, split-thickness grafting and a bilayered bioengineered skin substitute have been used with good results in conjunction with ongoing systemic therapy.[11,47] Additionally, nanoparticle

Select Cases of Ulcerative Sarcoidosis in the English-Language Literature

Reference (Year)	Age, y/Sex (Ethnicity)	Location of Lesions	Morphology	Histology	Treatment	Outcome[a]
Irgang[32] (1955)	33/F (black)	Face	Ulcerated plaques	EC	Isoniazid	Resolution 8 mo
Simpson[30] (1963)	66/M (black)	Pretibial region	Painful ulcers	NCG	SC	Resolution 6 wk
Bazex et al[28] (1970)	62/F (white)	Pretibial region	Deep ulcers	EC	PV	Resolution 8 wk
Schiffner and Sharma[26] (1977)	79/F (black)	Face, arms	Ulcerated nodules	NCG	C + SC	Resolution 10 mo
Meyers and Barsky[25] (1978)	32/F (black)	Pretibial region	Punched-out ulcers	NCG	SC	Resolution 1 mo
Herzlinger et al[24] (1979)	27/M (black)	Pretibial region	Verrucous ulcers	NCG	SC	Resolution 7 wk
Boyd and Andrews[23] (1981)	24/M (black)	Finger	Ulcer over SQ nodules	NCG	SC	Resolution 14 wk
Schwartz et al[22] (1982)	30/M (black)	Legs	Verrucous nodules	NCG	HC + SC	Resolution 1 mo
Neill et al[20] (1984)	51/F (white)	Breasts	Shallow ulcers	NCG	SC	NR
Saxe et al[21] (1984)	68/F (white)	Legs, face	Verrucous ulcers	NCG	SC	Resolution 2 y
Mitchell et al[5] (1986)	25/M (white)	Legs, arms	Ulcerated nodules	NCG	SC	Resolution 1 y
Verdegem and Sharma[16] (1987)	27, 30, 35, 51/all F (black)	Legs (pretibial region, 3; ankle, 1)	Ulcers	NR	SC	Resolution in 1–4 wk
Albertini et al[14] (1997)	53/F (white)	Face, arms, legs, buttocks, chest, and back	Ulcerated nodules	NCG	MTX	Patient died
Green et al[13] (2001)	62/F (black)	Nose, axilla	Ulcerated plaques	NCG	SC	NR
Philips et al[10] (2005)	55/F (black)	Leg	Ulcer	NR	Adalimumab	Resolution in 9 wk
Poonawalla et al[9] (2008)	45/F (black)	Legs	Ulcers	NNNCG	SC + AZT	Resolution in 3 mo
Joshi et al[7] (2009)	44/F (black)	Legs	Ulcers	NCG	IL-TAC	Resolution in 3 mo
Hunt et al[12] (2012)	35/F (black)	Pretibial region	Painful ulcers	EC	NR	NR
Noiles et al[4] (2013)	47/F (black)	Leg	Deep ulcers	NNCG	SC + CT/MTX	Resolution in 2 mo; patient died of respiratory complications
	62/F (black)	Arm	Ulcerated plaques	NNCG	SC + MTX	NR

Abbreviations: F, female; EC, epithelioid cell; M, male; NCG, noncaseating granuloma; SC, systemic corticosteroid; PV, peripheral vasodilators; C, chloroquine; SQ, subcutaneous; HC, hydroxychloroquine; NR, not recorded; MTX, methotrexate; NNNCG, nonnecrotizing noncaseating granuloma; AZT, azathioprine; IL-TAC, intralesional triamcinolone; NNCG, necrotizing noncaseating granuloma; CT, compression therapy.

[a]After treatment was initiated.

silver burn paste has been used successfully, with resolution of ulcers within 2 weeks in the Chinese literature.[53]

All of these treatment recommendations are based on historically accepted modalities. Controlled trials with longitudinal follow-up are needed to provide justification for the current standard of care.[34]

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