Superficial granulomatous pyoderma of the leg improved after conservative management with Unna boot and intralesional steroid injections

Emily C. Milam, MD, and Nooshin K. Brinster, MD
New York, New York

INTRODUCTION

Superficial granulomatous pyoderma (SGP) is a rare condition that some regard as a variant of pyoderma gangrenosum (PG) given their clinical similarities; however, SGP is distinct in its cutaneous presentation, histology, lack of underlying disease, and prognosis. Treatment options have ranged from oral antibiotics to oral and intralesional steroids, immunomodulatory agents, and intravenous immunoglobulin (IVIG), among others. We describe a case of long-standing SGP that responded to conservative management with Unna boot and intralesional steroid injections.

CASE REPORT

A 27-year-old Pakistani woman presented with a 20-year history of an indolent nonhealing, ulcerating eruption extending down her left leg. The lesions began as painful acneiform papules on her lower thigh and progressively tracked toward her ankle, grew in size, ulcerated, and healed as scars. The patient also endorsed pathergy, with new lesions developing upon waxing her leg hair. Numerous courses of antibiotics, colchicine, antituberculosis medications, and dapsone at other institutions did not achieve resolution. A review of systems and medical history were unremarkable.

The physical examination of the left leg revealed confluent, scarred plaques in areas of previous ulcers and multiple punched-out, superficial ulcers with associated tenderness (Fig 1). The ulcers lacked undermined borders. There was no regional lymphadenopathy. The remainder of the physical examination was unremarkable.

Three 3 × 3 × 4 mm punch biopsy specimens showed similar findings, including irregular epidermal hyperplasia containing neutrophilic microabscesses adjacent to an ulcer and suppurative and granulomatous inflammation in the papillary dermis (Fig 2). Leukocytoclastic vasculitis was not present.

Fresh tissue sent for culture and a polymerase chain reaction study were negative for leishmanial, bacterial, fungal, and mycobacterial organisms. Routine laboratory values, erythrocyte sedimentation rate, QuantiFERON Gold (Quest Diagnostics, Secaucus, NJ) tuberculosis tests, autoantibodies (rapid plasma reagin, myeloperoxidase-antibody, proteinase 3 antibody, and hepatitis B and C antibodies), and serum protein electrophoresis were normal. Venous and arterial ultrasound studies were unremarkable. A magnetic resonance imaging scan showed no evidence of sinus tracts or osteomyelitis. The constellation of clinical, biopsy, and laboratory findings supported a diagnosis of SGP.

The patient had failed multiple previous oral treatment courses as outlined above. Cyclosporine was considered; however, conservative management was ultimately pursued. An Unna boot—or zinc oxide-impregnated gauze wrapped from the base of the toes to the popliteal flexure—was applied to the affected leg weekly. Intralesional triamcinolone (ranging in concentration from 10-40 mg/mL) was injected into active ulcers monthly. Within 4 months, all ulcers had resolved (Fig 3).

DISCUSSION

SGP is a rare condition characterized by chronic, superficial sterile ulcerations. Wilson-Jones and

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Correspondence to: Emily C. Milam, MD, New York University School of Medicine, 240 E 38th St, Fl 11, New York, NY 10016. E-mail: emily.milam@nyumc.org.

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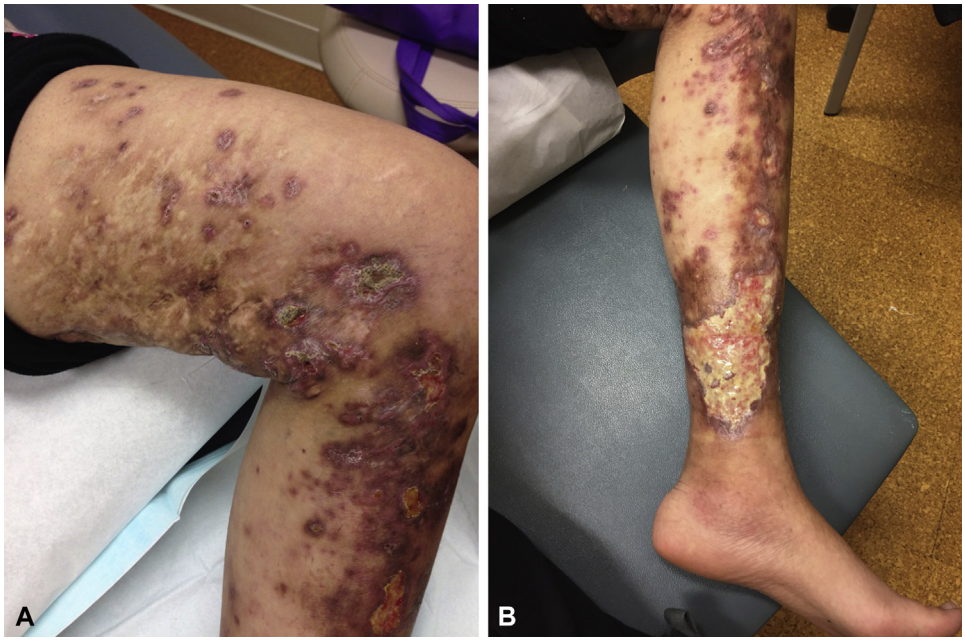

Fig 1. **A**, Extending down the left leg are confluent, scarred plaques in areas of previous ulcers, and multiple punched out, superficial ulcers that lack undermined borders. **B**, The distal medial leg shows a larger, irregularly shaped superficial ulceration with vegetative borders.

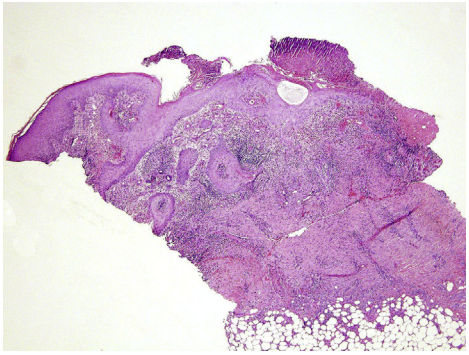

Fig 2. Irregular epidermal and follicular hyperplasia containing neutrophilic microabscesses adjacent to an ulcer, and suppurative and granulomatous inflammation in the papillary dermis. A scar is present in the reticular dermis. (Hematoxylin–eosin stain; original magnification, ×40.)

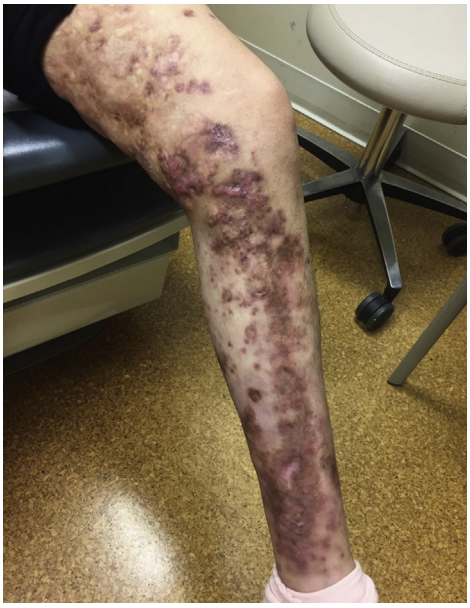

Fig 3. Resolution of superficial ulcerations, with residual cribriform scarring, after 4 months of conservative treatment with Unna boot and intralesional triamcinolone.

Winkelmann[1] first described SGP in 1988, considering it a "localized, limited form of chronic superficial PG" made distinct by its verrucous and vegetative hyperplasia, granulomatous histology, and more indolent course.

While SGP shares similarities with PG, it overall has a distinct clinical and histologic presentation. Like PG, SGP presents as a progressive ulcerating disease that may demonstrate pathergy and heal with cribriform scarring. Unlike classic PG, however, SGP favors a more slowly progressive course, with ulcers that are more superficial, granulomatous, and clean appearing, with vegetative (rather than undermined) borders. SGP often arises in a truncal distribution and has a greater propensity for surgical wounds and injured hair follicles, as we suspect occurred in our patient.

Histologically, SGP consistently demonstrates granulomatous inflammation, typically with 3 layers:

(1) a central neutrophil-dense zone with cellular debris; (2) a surrounding layer of histiocytes and giant cells; and (3) an outer layer of plasma cells and eosinophils.[1,2] Sinus tracts have also been noted. Unlike PG, SGP typically lacks hemorrhage or necrosis. The vegetative granulomas of SGP have been compared to other infectious and noninfectious granulomatous skin diseases, including blastomycosis, tuberculosis verrucosa cutis, and bromoderma.[2] The differential diagnosis also includes atypical mycobacterial infections, vegetative lupus erythematosus, foreign body granuloma, and actinomycosis.

SGP does not always require treatment with immunosuppressive agents and is usually not associated with underlying disease, whereas PG is associated with inflammatory bowel diseases and rheumatoid arthritis, among many other conditions. Unlike PG, it is not unusual for SGP ulcerations to spontaneously heal. When treatment is pursued, the literature demonstrates success with topical therapies, including topical steroids and topical tacrolimus, which may require daily application for upward of 1 year for adequate results. For SGP cases unresponsive to topical medications, a variety of systemic agents have proved effective, including oral corticosteroids, tetracyclines, colchicine, and dapsone.[3,4] SGP rarely occurs on the face, but documented cases mimic granulomatosis with polyangiitis, appear to behave more aggressively (akin to PG), and be treatment refractory, requiring stronger systemic agents, such as cyclosporine, infliximab, and intravenous immunoglobulin.[5]

In our patient, we elected to take a conservative approach with intralesional triamcinolone and compression therapy using an Unna boot. Compression therapy has been successfully used to heal other inflammatory ulcerations of the lower extremities, including erythema nodosum, pyoderma gangrenosum, necrobiosis lipoidica, cutaneous leukocytoclastic vasculitis, psoriasis, and even dissecting cellulitis of the scalp.[6] Cochrane reviews of compression therapy especially support its efficacy in treating ulcers occurring secondary to chronic venous insufficiency.[7] Unna boots—which typically use moisture-retentive zinc oxide-impregnated gauze—represent an inexpensive and low-risk method of promoting wound healing. Compression therapy fosters a favorable wound environment by speeding reepithelialization, stimulating collagen synthesis, and promoting angiogenesis. Compression also reduces superficial vein distention, relieves edema, and covers the skin, thereby protecting from further trauma.[8] Research among patients with venous insufficiency who are receiving compression therapy shows a reduction in the proinflammatory milieu coupled with increased antiinflammatory cytokines.[9] Moreover, zinc oxide itself protects surrounding skin, is antiinflammatory, and may also improve reepithelialization and decrease inflammation.[10]

This case illustrates the clinical and histopathologic distinctions between SGP and PG, which are frequently misconstrued as being part of a diagnostic continuum. Our case also shows that conservative treatment with a focus on wound care can be sufficient for treatment of some cases of SGP.

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