CLINICAL VIGNETTE

Pyoderma Gangrenosum Complicating Venous Stasis Ulcer?

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Case Report

A 57-year-old obese man with hypertension and uncontrolled diabetes was admitted to the hospital for non-healing right leg ulcers. The patient reported the lesions started several years ago as single "bed bug bites" that were pruritic. These small recurrent ulcerations involved both legs for the past 4 years, but for 2-3 months prior to admission the right leg ulcers enlarged dramatically and began to weep. The ulcerations now involved the majority of his right lower leg circumferentially. The ulcers were not painful to palpation but became painful when he elevated his leg and improved in a gravity dependent position. The patient denied tingling, numbness, fever, chills, nausea, vomiting, sweating, claudication, abdominal pain, weight loss, diarrhea or any other bowel complaints.

As an outpatient, he was seen and evaluated by both dermatology and podiatry. A plain x-ray did not show evidence of osteomyelitis. Wound cultures grew *proteus, enterococcus* and *klebesiella* species. A course of oral ciprofloxacin and clindamycin did not improve the ulcers.

Dermatology performed biopsies of the ulcer which returned “consistent with pyoderma gangrenosum” (PG) with secondary bacterial infection. The patient was subsequently admitted for inpatient care.

On admission the patient was afebrile, not toxic or distressed. His chest was clear, heart rate was regular without murmur, abdomen was large, soft and nontender. His legs were edematous, with the right lower extremity more edematous than the left. Multiple large, deep weeping ulcerations were present circumferentially around the right calf and tibia with necrotic yellowish surface and scattered areas of punctuate erythema. The leg was malodorous but not tender to palpation and had no fluctuance. The dorsalis pedis pulses were present in both feet but slightly weaker on the right foot.

Laboratory results: WBC 6.1 , INR 1.1, PTT 35.2, EGFR 87, ESR 27, A1c 10.6, normal serum protein electrophoresis, CRP 1.54, ANA (EIA) negative, RH factor 20.5 (0-20), Hepatitis A, B, and C serologies were negative.

The biopsies of the ulcer were re-reviewed and were felt to have two pathological findings: 1) Non-specific ulceration with abscess formation consistent with PG; 2) Non-specific ulceration with papillary dermal vascular proliferation consistent with venous stasis ulceration.

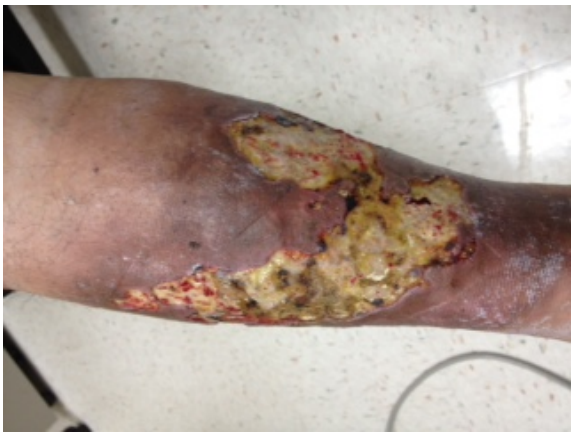

Figure 1

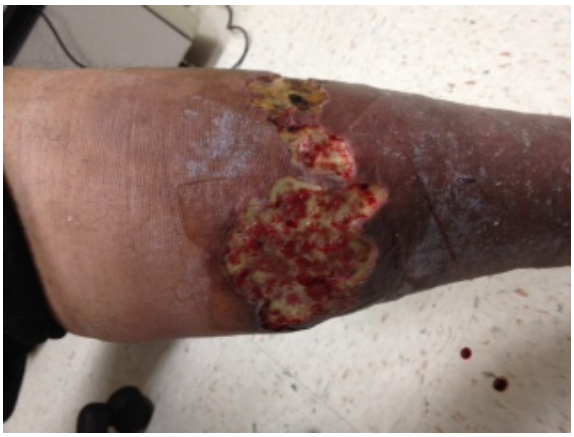

Figure 2

pDoppler arterial studies of both lower extremities showed small vessel disease and no hemodynamically significant arterial stenosis. Venous ultrasound in the standing position showed valvular venous reflux of the right greater saphenous vein at the level of the mid/distal calf and left greater saphenous vein at the proximal calf.

The patient was initially treated with IV methylprednisolone, plus Vancomycin and Ertapenim, then transitioned to oral prednisone and amoxicillin/clavulanate. Infectious disease felt the positive wound cultures were likely colonization and not active infection but still recommended a course of antibiotics. During his hospitalization, the patient's leg was elevated and his ulceration improved mildly: the depth of the ulceration became shallower and drier. His leg was placed in an UNNA boot dressing and he was discharged home to complete a course of prednisone and amoxicillin/clavulanate. He was scheduled to follow up visit with both podiatry and dermatology.

Over the next several months the patient continued to get wound care through the podiatry service. They started two additional courses of prednisone with marginal improvement.

Discussion

Chronic lower extremity ulcerations cause major morbidity for patients. The overwhelming majority of leg ulcers are of venous origin. Next most common is arterial occlusive disease followed by neuropathic ulcers[1]. Determining the etiology is important so effective treatment can be rendered.

Pyoderma gangrenosum is usually associated with underlying pathology like inflammatory bowel disease (IBD), monoclonal gammopathy, hematologic malignancy, paraproteinemia, Behcet disease, Sweet syndrome, hepatitis, HIV, systemic lupus erythematosus, rheumatoid arthritis, ankylosing spondylitis, pregnancy, Takayasu arteritis, and others (2-4). These conditions were excluded in our patient. Typically, IBD is the underlying cause in only 15% to 20% of patients with PG[5] but 25% to 50% of PG can be idiopathic in origin[6].

The key in diagnosing PG in a rapidly progressive painful ulcer is excluding other causes of cutaneous ulcers through biopsy, culture, and clinical acumen[7]. Biopsy of an early lesion of PG often demonstrates a dermal neutrophilic abscess, which was present in the above patient. Later-stage lesions show epidermal necrosis and ulceration, superficial dermal edema, and a dense, mixed dermal infiltrate that may extend to the panniculus. Histologic examination of the advancing, inflamed border reveals dense perivascular lymphocytic inflammation, which may at times be associated with vascular destruction[1]. PG should also have at least two of the following: pathergy (lesions are precipitated by cutaneous trauma), systemic disease, histopathologic findings consistent with PG, or treatment response (systemic glucocorticoid)[8].

Stasis ulcers, on the other hand, tend to occur with long-standing venous disease. The skin becomes indurated with fibrosis of the dermis and subcutaneous tissue. The ulcers are usually restricted to the medial leg and are sharply demarcated from proximal normal skin, resulting in the appearance of an inverted bottle. Lipodermatosclerosis, a term used to describe these clinical findings, suggests a greater impairment of the fibrinolytic system and is highly associated with and usually restricted to the legs of patients with venous insufficiency[9,10].

The etiology of the ulcerations in the above patient is likely both venous stasis ulcer and PG. This may explain the reason why his ulcers were so difficult to treat. Venous stasis ulcers represent a chronic type of wound, with microcirculation impairment, which can be the pathergy that triggers PG.[11-13]. Up to 40% of patients with PG exhibit pathergy. Leg ulcers are frequently subjected to minor acute traumas that may be caused by dressing changes and debridements. Also, some topical medications used in wound care could trigger PG, such as iodine. It is necessary to avoid local surgical treatment of venous leg ulcers in patients with a history of PG or with underlying predisposing conditions[14].

We have identified a patient who has components of both venous stasis ulcers and pyoderma gangrenosum. We suggest that patients who have worsening chronic venous stasis leg ulcers be biopsied to evaluate for rare etiologies such as PG.

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Submitted on October 12, 2012