Classic ulcerative pyoderma gangrenosum in Fitzpatrick V skin type

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To the Editor:

We describe a 46-year-old man with Fitzpatrick V photo skin type and a history of obesity and hepatic steatosis likely secondary to non-alcoholic steatohepatitis, who presented with a rapidly enlarging ulcer on the left upper abdomen. The patient had experienced minor occupational trauma to the abdomen while carrying and moving heavy packages. An extremely painful papule progressed to an ulcer within less than 10 days.

On a physical examination of the left upper abdomen, he was noted to have a 5cm ulcer with an irregular, undermined border with a violaceous hue and subtle surrounding peripheral erythema. Multiple satellite pustules and surrounding small ulcers with yellow debris were also present (**Figure 1**). The differential diagnosis was broad but included pyoderma gangrenosum (PG), deep fungal or atypical mycobacterial infection, and other neutrophilic dermatoses such as Sweet syndrome, given the presence of pustules. Of note, several firm, indurated papules and nodules with scarring were also appreciated in the bilateral axillae, but no acneiform lesions or scarring were evident on examination of the face, chest, and back. Prior workup was notable for a computed tomography scan that showed an exophytic, irregular collection of fluid centered within the skin overlying the anterior left upper quadrant which was associated with soft tissue stranding and skin thickening. A recent colonoscopy and esophagogastroduodenoscopy had been undertaken without significant findings.

A complete blood count, urinalysis, chest X-ray, serum and urine protein electrophoresis, antinuclear antibodies, rheumatoid factor, viral hepatitis panel, antineutrophil cytoplasmic antibodies, and antiphospholipid panel were all within normal limits.

Punch biopsy of the ulcer edge demonstrated a pandermal perivascular and interstitial neutrophilic infiltrate. Stains for infection (periodic acid-Schiff stain, Gram, Steiner, acid-fast bacilli, and Fite) and tissue cultures were all negative. Given no evidence

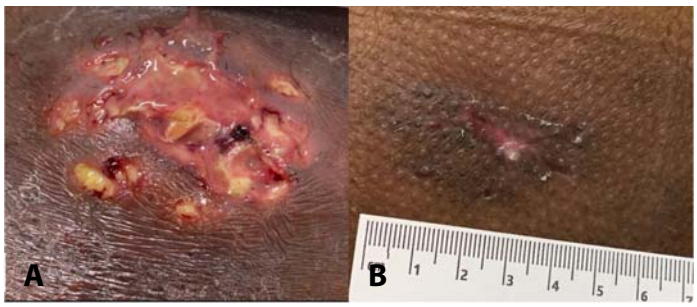

Figure 1. *Classic ulcerative pyoderma gangrenosum in a patient with photo Fitzpatrick V skin.* ***A)*** *Patient presented with a 5cm ulcer on his left upper abdomen after minimal trauma to the site, with an irregular, violaceous, and undermined border and subtle peripheral erythema. Punch biopsy of ulcer edge showed a neutrophilic infiltrate, with negative infectious stains (PASd, Gram, Steiner, AFB, and Fite) and negative tissue culture. The cause of the pyoderma gangrenosum was determined to be idiopathic, after a thorough infectious, autoimmune, and gastrointestinal work-up for underlying disease.* ***B)*** *After 6 weeks of wound care, the patient healed with a hyperpigmented scar with a subtle cribriform pattern.*

of infection, along with our clinicopathologic correlation, our leading diagnosis became pyoderma gangrenosum, as our patient met the histologic and four minor diagnostic criteria that have been published for PG. One of these included: 1) history of papule, pustule, or vesicle that rapidly ulcerated, 2) peripheral erythema, undermining border, and tenderness at site of ulceration, 3) pathergy, and 4) exclusion of infection [1]. He met one major criterion for Sweet syndrome (neutrophilic infiltrate on histology), but the abdominal lesion was atypical for Sweet syndrome. Furthermore, he failed to meet any of the minor criteria for Sweet syndrome, including fever, abnormal laboratory values, and underlying malignancy or preceding viral or gastrointestinal infection [2].

Treatment was initiated with Di-Dak-Sol soaks for 15 minutes a day to the ulcer, followed by a twice daily regimen of clobetasol 0.05% ointment to the ulcer rim. Complete healing was noted within 2-3 weeks. The initial appearance of the several satellite small ulcers surrounding the main ulcer likely contributed to the ultimate cribriform appearance of the patient's scar. This cribriform healing pattern is classic for pyoderma gangrenosum. Significant post-inflammatory hyperpigmentation was also observed. The patient remained in remission at his subsequent 3-month follow-up visit.

Pyoderma gangrenosum can be associated with autoimmune disease, inflammatory bowel disease, myeloid dyscrasia, malignancy, or IgA monoclonal gammopathy [3]. It may also be a component of genetic conditions or autoinflammatory syndromes, including PASH (PG, acne, and hidradenitis suppurativa) or PAPA (pyogenic arthritis, PG, acne), [3]. Our patient likely had concurrent hidradenitis suppurativa but did not have other stigmata of autoimmune disease or autoinflammatory syndromes. Sigmoidoscopy and colonoscopy ruled out underlying inflammatory bowel disease for our patient. In addition, serum protein electrophoresis was unremarkable and quantitative immunoglobins for IgA, IgG, and IgM were all normal.

In patients with darker skin, surrounding erythema can be challenging to appreciate when evaluating for PG. Furthermore, PG is more likely to result in excessive scarring and/or skin dyspigmentation (hypo- or hyperpigmentation) in darker-skinned patients.

Potential conflicts of interest

The authors declare no conflicts of interest.

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