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It Starts With a Dog Scratch

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A 63-year-old female with a history of essential thrombocythemia and hypertension presented with a 4-week history of a worsening ulcer on her right second digit. Initially, the patient attributed the wound to a dog scratch but sought further treatment at an outside clinic when she did not see improvement. She was given a diagnosis of cellulitis and was treated with unknown oral antibiotics and silvadene cream. The ulcer continued to worsen and the patient presented to our hospital. On physical exam, an 8 cm × 3 cm ulcer was observed on the right second digit. It had violaceous rolled up borders, granulation tissue, fibrinous exudates, and areas of necrotic tissue (Figure 1). The remainder of the physical examination was unremarkable. Initial laboratory values included hemoglobin 12.5 gm/dL, white blood cell count 31.2 K/UL, and platelets 625 gm/dL. An x-ray of the hand showed soft tissue swelling with no evidence of osteomyelitis. The ulcer was evaluated and treated as an infected wound. The patient was started on broad spectrum intravenous antibiotics and underwent excisional debridement with biopsy. Blood and wound cultures were negative for aerobic and anaerobic bacteria, fungi, and acid-fast bacilli. Pathology from the biopsy showed extensive necrosis and acute inflammation. The patient was discharged home with 10 days of oral antibiotics, and instructions for wound care. Upon follow-up 1 week later, the patient complained of intense pain and worsening of the ulcer prompting readmission. Dermatology was consulted and diagnosed pyoderma gangrenosum (PG). The patient was started on prednisone, 60 mg daily and azathioprine, 50 mg daily. The ulcer slowly improved (Figure 2) and the steroid dosage was tapered. She was finally discharged home with a 6-week taper of prednisone, azathioprine, and home health consultation for assistance with wound care.

PG is an ulcerative neutrophilic dermatosis. In up to 50% of cases, PG is associated with either inflammatory bowel disease, collagen vascular disease, or hematologic disorders.[1] Although an immune-modulated pathway may be involved, the etiology and pathophysiology of PG is still unknown.[1] Furthermore, PG is a diagnosis of exclusion.[1] However, PG does have clinical findings which favor the diagnosis. There are 4 main subtypes of PG; ulcerative or "classic," pustular, bullous, and vegetative.[1] Although myeloproliferative disorders are more specifically associated with the bullous form, our patient presented with the classic

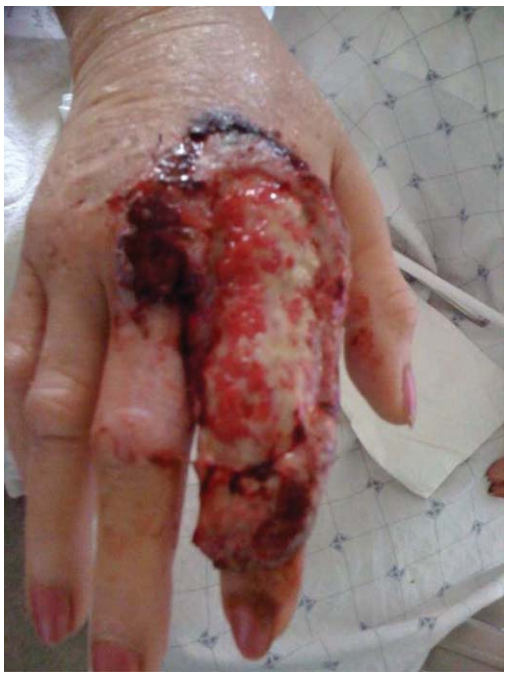

FIGURE 1. Right second digit ulcer with violaceous, irregular, rolled up borders with areas of necrosis.

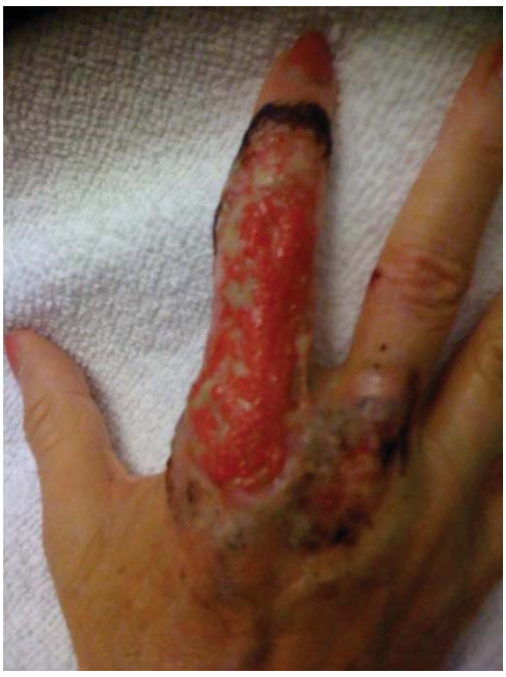

FIGURE 2. Significant regression of ulcer 3 weeks after initiation of immunosuppressants.

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subtype.[2,3] In the classic subtype, patients will often describe an initial pustule which then necroses, forming an ulcer with a reddish/purple or gray undermined border and a red halo surrounding the ulcer. PG can occur anywhere on the body however it is more frequently seen on the legs. A clinically relevant feature of PG, emphasized in this case, is pathergy. Thus, PG can develop or worsen secondary to mild trauma. PG has been reported to form after mild trauma such as an insect bite or dog scratch and has been documented to worsen with debridement, skin grafting, and biopsies.[1] Another feature and clinical clue of PG as manifested by our patient is intense pain. The skin biopsy, however, is usually nonspecific and can reveal findings which include edema, neutrophil infiltration, abscess formation, necrosis, and thrombosis of vessels.[1] In patients with PG associated with myeloproliferative syndromes, no correlation has been shown between the time of diagnosis and the severity of the underlying myeloproliferative syndrome.[2,3] Treatment for PG depends on extent of involvement and association with underlying disease and can include local, oral, or intravenous corticosteroids, immunosuppressants, appropriate wound care, and treatment of associated disease.[4]

PG is a diagnosis of exclusion. Underlying infection, vasculitis, malignancy, and Sweet's syndrome should be considered in the differential. However, one must consider PG in the differential diagnosis of an ulcer in a patient with an underlying predisposing illness, when the ulcer has characteristics of pathergy and intense pain, and is not healing appropriately as illustrated in this case.

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References

1. Ruocco E, Sangiuliano S, Gravina AG, Miranda A, Nicoletti G. Pyoderma gangrenosum: an updated review. *J Eur Acad Dermatol Venereol.* 2009; 23(9):1008–1017.
2. Romano J, Safai B. Pyoderma gangrenosum and myeloproliferative disorders: report of a case and review of literature. *Arch Intern Med.* 1979;139: 932–934.
3. King K, Murray A. Pyoderma gangrenosum in a patient with essential thrombocythemia. *J Cutan Med Surg.* 2000;2:107–109.
4. Crowson AN, Mihm MC, Magro C. Pyoderma gangrenosum: a review. *J Cutan Pathol.* 2003;30:97–107.

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