A 20-year-old man presented with a non-healing ulcer on the dorsum of the left foot that initially appeared after the patient fell and hit his left foot on the edge of a door. An ulcer had formed and grown in size over the past several weeks after repeated physical insults and despite home wound care, as seen in Figure 1. Additionally, the patient had a non-healing ulcer inferior to his left knee. He had a notable past medical history of acute myelogenous leukemia (AML) in remission after a haploidentical stem cell transplant, uncontrolled diabetes type 1, and graft-versus-host disease of the skin. Both wounds showed no signs of osteomyelitis or cellulitis on MRI.

The patient’s ulcers most closely resembled pyoderma gangrenosum (PG), and his history was consistent with the pathergy associated with this disease. Pathergy is defined as the evidence of new skin lesions arising at sites of intradermal trauma due to an inflammatory response mediated largely by neutrophils. Pathergy is also seen in bowel associated dermatosis-arthritis syndrome, Behcet’s disease, and rheumatoid arthritis. PG occurs most frequently in the lower extremities and can be divided into five subgroups: classic, bullous, pustular, vegetative, and peristomal types. Lesions usually start as tender nodules, plaques, or sterile pustules that become larger with trauma and debridement. Over several days to a week, the lesion often enlarges into a sharply demarcated ulcer with violaceous borders and a zone of erythema. PG lesions are extremely painful. They are thought to arise from loss of innate immune regulation and altered neutrophil chemotaxis. AML is the most common hematologic malignancy that underlies PG, with the bullous subtype of PG being the predominant type found in these cases. PG lesion development foreshadows a poor prognosis in AML, and its presence in myelodysplastic syndromes may herald a malignant transformation. Chronic and atypical cases have a higher association with underlying AML and myelodysplastic syndromes than typical cases.

PG is treated with anti-inflammatory agents such as steroids and tumor necrosis factor-alpha inhibitors. If PG appears as a dermatologic manifestation of a systemic disease, treating the systemic disease is necessary.

REFERENCES