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Pyoderma gangrenosum flare complicated by cellulitis and impaired wound healing

Suneel Kumar Parvathareddy*

***Corresponding Author: Suneel Kumar Parvathareddy**
Wake Forest Baptist School of Medicine, USA.
Email: suneel.parvathareddy@advocatehealth.org

Abstract

Pyoderma Gangrenosum (PG) is a rare ulcerative neutrophilic dermatosis often associated with systemic disease, specifically inflammatory bowel disease [1]. Chronic liver dysfunction may impair the wound healing process and complicate management of PG flares. This report focuses on a 57-year-old female with extensive liver disease and long-standing bilateral lower extremity ulcers due to PG, who presented with a PG flare of her left lower leg, complicated by cellulitis and *Streptococcus pyogenes* bacteremia. Imaging ruled out an abscess or necrotizing infection. This patient's management was guided by a multidisciplinary team that included internal medicine, wound care, general surgery, infectious disease, and virtual dermatology. She received empiric IV antibiotics, wound care, and corticosteroids, and was discharged with oral antibiotics and close outpatient follow-up appointments. This case demonstrates the challenges of managing PG in patients with chronic liver disease, where impaired hemostasis and tissue perfusion delays proper wound healing. A multidisciplinary team is imperative for optimizing patient outcomes, with balancing immunosuppression and infection control.

Case Presentation

A 57-year-old female with a history of IV drug use, chronic hepatitis B and C with cirrhosis, coronary artery disease, hypothyroidism, obesity, chronic Pyoderma Gangrenosum (PG), lipodermatosclerosis, and elephantiasis nostras verrucosa who presented to the ED with a flare of her PG. She had chronic bilateral lower extremity ulcers for 2 years that had not fully healed (Figure 1A & 1D).

On presentation, she was afebrile, tachycardic (116 bpm), tachypneic (22 breaths/min), and hypertensive (147/67 mmHg). The left leg was markedly swollen, tender, warm, with chronic ulcers oozing purulent discharge, extending proximally to the groin (Figure 1B & 1C). The patient denied any fevers, use of blood thinners, or nausea/vomiting.

Diagnostics

Initial labs revealed leukocytosis (11,900 cells/μL; reference range 4,000-10,000 cells/μL), hypomagnesemia (1.3 mg/dL; reference range 1.7-2.3 mg/dL), hypokalemia (3.2 mEq/L; reference range 3.5-5.0 mEq/L), elevated ESR (252.7 mm/hr; reference range 0-20 mm/hr), elevated CRP (93 mg/L; reference range <10 mg/L), and an acute kidney injury evidenced by elevated serum creatinine (1.92 mg/L; reference range 0.6-1.1 mg/dL). Liver enzymes are chronically elevated and were near baseline, with prolonged PT (16.5 seconds; reference range 11-13.5 seconds) and INR (1.6; reference range 0.8-1.1). A urine drug screen was positive for amphetamines, benzodiazepines, and opiates, though the latter reflected ED-administered opiates. The rest of her lab results were unremarkable.

A 12-lead electrocardiogram revealed sinus tachycardia, but otherwise unchanged from previous. Doppler ultrasound of bilateral lower extremities ruled out deep vein thrombosis. CT left lower extremity with contrast demonstrated extensive soft tissue edema and chronic ulcers without abscess or necrotizing infection. CT pelvis with contrast revealed reactive lymphadenopathy without drainable fluid.

Despite the patient reportedly completing treatment for both chronic hepatitis B and C, her HCV RNA was detected with a viral load of 2 million IU/mL and HBV DNA was detected with a viral load of >83 million IU/mL. She was restarted on her previous entecavir and was advised to follow up closely with her gastroenterologist for further evaluation and long-term treatment.

Management

In the ED, the patient received IV morphine 4 mg, 2 tablets of oxycodone 5-3 25 mg, 2 L bolus of lactated Ringer's solution, along with empiric antibiotic therapy with IV clindamycin 900 mg, IV Zosyn 4.5 g, and IV vancomycin 2 g. A urine culture and 2 blood cultures were obtained prior to administering antibiotics.

Clinical course/outcome

Upon admission to intermediate care for PG flare with left lower extremity cellulitis, IV Solumedrol (2 mg/kg/day) was initiated, and antibiotic therapy was continued per infectious disease recommendations (Figures 2A - 2E). Due to the patient's current acute kidney injury and chronic liver disease, diuretics and hepatotoxic medications were held. Urine culture was negative, while her blood cultures finalized positive for *Streptococcus pyogenes.* A transthoracic echocardiogram was negative for any valvular vegetation, ruling out infective endocarditis.

Wound care applied dressings to open wounds daily throughout the patient's hospital stay. The general surgery team noted that due to the degree of the infection, there may be a need for above-the-knee amputation, to which the patient refused. Due to the appearance of multiple blisters along the medial and lateral left thigh and the continuous purulent drainage from her open wounds, antibiotic therapy was optimized with the addition of IV clindamycin 900 mg three times daily.

The hospital is in a rural town, so there was no dermatology team on site. Therefore, a virtual dermatology consult was conducted and the provider utilized images that were taken by staff to make appropriate recommendations. The dermatologist suggested a punch biopsy to evaluate for severe bullous cellulitis or cellulitis-like sweet syndrome rather than a PG flare. The results of the punch biopsy revealed changes consistent with stasis ulceration. Leukocytosis and erythema improved with tapering steroid therapy and continuing antibiotics. At discharge, the patient was ultimately transitioned to oral linezolid 600 mg twice daily to extend coverage given lymphedema of tissues and potentially poor blood flow. Dermatology also recommended to increase her daily home-dose prednisone from 10 mg to 20 mg daily. Close outpatient follow-up appointments with her dermatologist, gastroenterologist, and primary care provider were made and the patient was discharged after 13 days in the hospital.

Discussion

Pyoderma Gangrenosum (PG) is a type of ulcerative disorder that is not specifically caused by infection or gangrene, despite its name, but actually associated with systemic disease, most commonly inflammatory bowel disease [1]. It is an uncommon pathology that affects about 10 cases per million people every year, with a seemingly female predominance [2].

Pathogenesis

The etiology and pathophysiology of PG is poorly understood, but it is theorized to be due to an autoimmune process from defects in cell-mediated immunity, neutrophil and monocyte function, and humoral immunity [3]. It has also been found that PG may be induced due to drugs, including isotretinoin, granulocyte colony-stimulating factor, cocaine, and more (Table 1). There appears to be an upregulation of key proinflammatory and neutrophil chemotactic factors within lesional skin, which is also induced in fibroblasts of the ulcers seen in PG [4]. There also seems to be an increase in Matrix Metalloproteinase (MMP) expression which may contribute to poor healing [4].

Variants [5]

Ulcerative (Classic) PG	• Initially presents as single or few small pustules that ulcerate quickly into painful lesions • Occurs in sites of minor trauma of lower extremities
Vegetative PG	• Less aggressive than ulcerate PG • Commonly appears on the trunk
Bullous PG	• Appears as painful superficial bullae that rapidly progress to form ulcers • Usually appear on the face and upper extremities • Less destructive than ulcerative PG • Commonly associated with myeloproliferative disorders
Pustular PG	• Presents as painful and symmetric pustular lesions on the lower extremities and upper trunk • Rare variant
Peristomal PG	• Often occurs near stoma sites • Appear as painful erythematous to violaceous papules that progress to ulcers

Differential

Acute febrile neutrophilic dermatosis, or sweet syndrome, is a neutrophilic dermatosis, like PG, but a diagnosis is made when either 2 of 2 major criteria and 2 of 4 minor criteria [3]. The major criteria

include abrupt onset of tender erythematous plaques with vesicles, pustules, or bullae, and predominantly neutrophilic infiltration in the dermis without leukocytoclastic vasculitis. Minor criteria include fever, leukocytosis, elevated sedimentation rate, and a rapid response to systemic steroid medications [3].

Evaluation

PG is a diagnosis of exclusion by using both clinical presentation and histology of the ulcers. Although there are no confirmed diagnostic criteria, a Delphi consensus exercise using the RAND/UCLA Appropriateness Method yielded 1 major criterion and 8 minor criteria to help guide clinicians and prevent further misdiagnoses [6]. The major criterion is based off a biopsy result of an ulcer, demonstrating neutrophilic infiltrate. The 8 minor criteria include exclusion of infection, pathergy, history of inflammatory bowel disease or inflammatory arthritis, history of papule, pustule, or vesicle ulcerating within 4 days of appearing, peripheral erythema and tenderness at ulceration site, multiple ulcerations with at least 1 on an anterior lower leg, cribriform scarring at healed ulcer sites, and decreased ulcer size within 1 month of initiating immunosuppressive medications [6].

Treatment

The mainstay of treatment for PG includes systemic immunosuppression, wound care, and pain control. The use of medications for systemic immunosuppression depend on how fast the disease progresses, so if the size of the lesion appears to be growing rapidly, then corticosteroids or cyclosporine may be used [1]. The STOP GAP randomized control trial in 2015 compared immunosuppressive medications, prednisolone and cyclosporine, for treating PG, and the results determined that there did not seem to be a difference in the speed of wound healing with either medication [1]. Debridement of adhered tissue is actually contraindicated as it may cause pathergy, but as the necrotic tissue loosens with re-epithelialization, it can be removed gently with forceps [7].

Prognosis and Prevention

According to a study that followed 23 patients who were admitted to the hospital with pyoderma gangrenosum, several patients had lengthy hospital stays with a mean of 47 days, along with high death (21.7%) and recurrence rates (39%) [8]. Most of the patients had severe and aggressive PG, and it was found that a significant factor in the prognosis of PG includes the type and severity of the associated systemic disease. Unresponsiveness to treatment of the associated disease would result in a poorer prognosis. This study also found that infected PG wounds were associated with a worse prognosis, as most of the patients who died had lower limb PG complicated by infection, with sepsis identified as the primary cause of death [8].

Contributing Factor

The patient discussed in this case study did not have associated inflammatory bowel disease, which is commonly seen with PG. However, she had extensive liver dysfunction, with chronic hepatitis B, chronic hepatitis C, and cirrhosis, among other comorbidities. When hepatocytes are exposed to inflammatory

cytokines and activated, they begin to transform into myofibroblasts and deposit collagen, ultimately leading to fibrosis of the liver [9]. Patients with liver disease may have incompetent hemostasis due to thrombocytopenia, low levels of coagulation factors and inhibitors, and decreased fibrinolytic proteins [10]. It is crucial for the phases of hemostasis, inflammation, proliferation, and tissue remodeling to be initiated promptly to promote healing. Treatments for impaired wound healing include focusing on optimizing controllable healing factors, like nutritional support, mechanical protection, and clearance of infections [11]. In this patient case, due to extensive liver dysfunction, there is a lack of a competent hemostatic process. This is likely a significant contributor to her poor wound healing.

Conclusion

This case study emphasizes the multifactorial relationship between chronic liver disease, impaired wound healing, and PG flares. Prompt recognition and treatment are essential, as multiple factors can adversely influence the prognosis of patients with PG and poorly healing wounds. Clinical benefit can be maximized in these patients by strategically utilizing a multidisciplinary approach with individualized adjustments of immunosuppressive treatment and antibiotic therapy.

Patient consent: Verbal consent was obtained from the patient for publication of this case report and any accompanying images.

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Authors Information: Suneel Kumar Parvathareddy*
Wake Forest Baptist School of Medicine, USA.

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