

CURBSIDE CONSULTS

A noninfectious “infection” after cesarean delivery

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SCENARIO

A 23-year-old patient with a history of hidradenitis suppurativa presents for an elective repeat

cesarean delivery at 39 weeks 0 days' gestation. Her previous cesarean delivery was notable for a 6-month recovery secondary to a wound infection requiring surgical debridement, prolonged use of wound vacuum-assisted closure (VAC), and multiple courses of antibiotics.

Her cesarean delivery is performed without incident, and the decision is made to empirically place a Prevena closed incision wound VAC.

In the first postoperative days, the patient complains of progressive incisional pain. She is started on triple antibiotics due to febrile morbidity and a diagnosis of endometritis.

On postoperative day 9, dehiscence of the wound and respiratory distress prompt admission to the intensive care unit. Two abdominal wound washouts fail to improve wound healing (**Figure 1**). All wound cultures are negative and microscopy reveals no bacteria, only an abundance of neutrophils. An autoimmune work-up is negative.



FIGURE 1 Patient incision on postoperative day 13 after repeat cesarean delivery. (Image courtesy of Tomlin)

What is going on?

Pyoderma gangrenosum (PG) is a rare, chronic, recurrent cutaneous ulcerative disease with an incidence of 3 to 10 per million in the general population.¹ It is commonly mistaken for infection because of the impressive wounds and marked pu-

rulence. Repeat negative cultures combined with a failure to respond to standard wound care and antibiotics should prompt inclusion of the diagnosis in the differential. Clinically, a rolled blue to violaceous undermined wound edge is classic but not a pathognomonic finding. Con-



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sequently, tissue pathology can often help solidify the diagnosis. In this case, tissue obtained from the washout was stained with standard hematoxylin and eosin, revealing a dense neutrophilic infiltrate and the absence of bacteria.

PG disproportionately affects women aged 20 to 50 years, and approximately half of patients have an associated proinflammatory disease such as inflammatory bowel disease (IBD), hidradenitis suppurativa, or rheumatoid arthritis.² Associated malignancies include acute and chronic myelogenous leukemia and IgA monoclonal gammopathy. Between 1.5% and 5% of patients with IBD develop PG.³

The pathophysiology of PG is poorly understood; however, familial clustering indicates a likely genetic predisposition. Pathergy (defined as an exaggerated skin injury following minor trauma that may be resistant to healing) is noted in approximately 15% of cases, as illustrated with this patient. Ulcerative PG is the most common subtype of PG, typically beginning on the lower extremity as a tender, indurated, erythematous papulopustule that ulcerates over weeks to months, leaving a full-thickness necrosis with a purulent base and an irregular gun-metal gray border. Bullous PG lesions are more superficial and are associated with leukemia or polycythemia vera. Vegetative PG is the least aggressive variant; it presents with elevated cribriform ulcerations and is not linked to systemic disease. Aggressive surgical debridement results in pathergy and a paradoxical worsening of the wound. The development of lesions is usually rapid, and pain may be out of proportion to the appearance of the ulceration. Fever and systemic symptoms

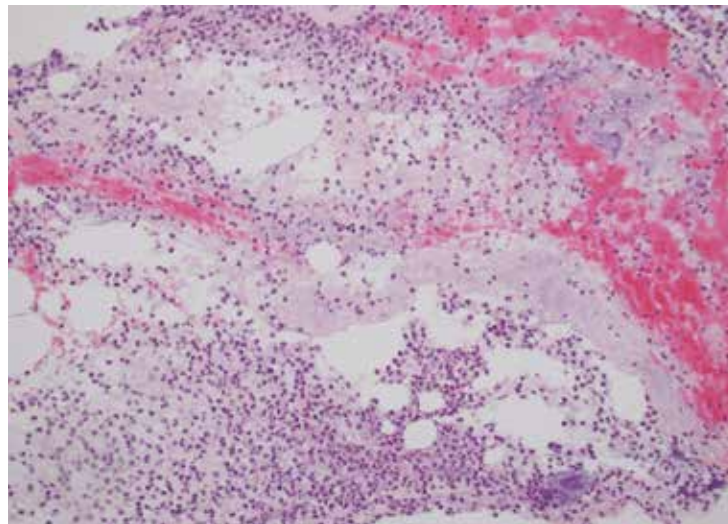


FIGURE 2
Histopathology from abscess biopsy confirming abscess formation with fibrin deposits and an overwhelming neutrophilic response. (Image courtesy of Tomlin)

may accompany this process.⁴ This patient had a typical clinical course, with postoperative dehiscence and systemic symptoms including respiratory distress.

Differential and initial work-up

Although there are no validated criteria for diagnosis of PG, wound biopsy to assess for a neutrophilic response and rule out other conditions is recommended. This specimen should include the wound edge and ulcer base for permanent histopathology (formalin). The sensitivity of wound culture is insufficient for diagnosis as colonization can be misinterpreted as causative. Bacterial, mycobacterial, and fungal tissue cultures should be obtained to exclude infectious etiology.

Laboratory studies to support the diagnosis may include an erythrocyte sedimentation rate and C-reactive protein, but the low specificity makes the utility questionable. Because approximately 50% of patients with PG have Crohn disease or ulcerative colitis, a thorough gastrointestinal review of systems and a screening

fecal calprotectin serologic test should be performed.² Clinical evaluation for a history of hidradenitis suppurativa should be explored.

Approximately 10% of cases are associated with leukemia, and evaluation for a monoclonal gammopathy with protein and urine electrophoresis could be considered; some authors recommend Bence Jones protein analysis.⁵ Hepatitis, syphilis, and HIV testing are reasonable. Given that approximately 30% of patients have coexisting arthritis, consideration of evaluation with rheumatoid factor and an anti-cyclic citrullinated peptide is reasonable. A hypercoagulability work-up should be considered if a review of systems warrants.

Initial management by an obstetrician

Considering this is a rare diagnosis, it is often missing from the differential. Even more challenging is redirecting the management from antibiotics to anti-inflammatories. Meticulous surgical techniques are key, particularly in a patient with a known or suspect-



FIGURE 3 Patient incision on postoperative day 21, 8 days after beginning oral prednisone therapy. (Image courtesy of Tomlin)

ed history of PG. We recommend closing incisions with sutures as opposed to staples and considering the use of preoperative steroids to minimize the risk of pathergy. If a lesion does develop, initial therapy involves optimizing wound care, minimizing pathergy from further surgical intervention, and initiating topical or intralesional therapy as described below.

First-line therapy in small lesions without systemic symptoms involves topical anti-inflammatories including corticosteroids, tacrolimus, and dapsone. These wounds are effectively mucosal sites, and it should be noted that systemic absorption of these medications has been detected. In the absence of systemic symptoms, small wounds may be managed with intralesional steroids at a concentration of

10 mg/mL at the wound border. Topical clobetasol, tacrolimus, and dapsone have been used with anecdotal success. Consider tacrolimus levels for larger wounds requiring frequent application. When using topical dapsone, monitor the common adverse effects (AEs) of nausea, vomiting, decreased appetite, blurred vision, tinnitus, and insomnia. Wounds worsening or failing to improve with a topical regimen may require systemic therapy such as high-dose corticosteroids or immune modulators. Glucocorticoids, such as prednisone, are typically given at high doses until all the lesions have healed but can be continued at lower doses as maintenance therapy to prevent recurrence. Systemic cyclosporine may be an alternative for patients who cannot tolerate glucocorticoid therapy, but use of other therapies

The authors recommend closing incisions with sutures and the use of preoperative steroids.

such as dapsone and minocycline has also been described.⁶

There is a trend toward biologics as first-line therapy, specifically from the tumor necrosis factor α class, including infliximab and adalimumab, as they are rapid in response and have a tolerable AE profile and finite duration of use. The association with pathophysiologic similarities to hidradenitis suppurativa, inflammatory bowel disease, and psoriasis further support this practice. However, cost is often a rate-limiting factor. Though a 3- to 6-month course of adalimumab is expensive, we propose that it costs less than multiple inpatient stays for debridement, wound VACs, parenteral antibiotics, wound care, and the impaired daily function of the patient.

In this patient, with no clinical improvement and a negative infectious work-up, the differential diagnosis was expanded to include PG. A wound biopsy confirmed the presence of an overwhelming neutrophilic response with a focal area of necrosis, which could be consistent with PG (**Figure 2**). The patient was started on systemic oral corticosteroids, and her symptoms improved rapidly. She was discharged from the hospital 48 hours later, with a prednisone taper with a standard wound VAC in place. One week later, significant improvement of the incision was noted (**Figure 3**). Complete healing occurred by 6 weeks' post hospital discharge. ■

FOR REFERENCES VISIT
contemporaryobgyn.net/cesarean-infection