Pyoderma gangrenosum (PG) is a neutrophilic dermatosis that is a rare complication of surgery. Its diagnosis and treatment often are delayed in the postsurgical setting; by the time a dermatologist is consulted, lesions often are quite large and may be accompanied by systemic symptoms. Prompt treatment of severe PG with systemic therapy is warranted; however, in patients with contraindications to systemic immunosuppressive agents, treatment with superpotent topical corticosteroids may be considered. We present a case of PG following gastric bypass surgery along with a review of the literature on postsurgical PG.

**Case Report**

A 52-year-old woman with a history of hypertension, lymphedema, and morbid obesity presented 1 week after undergoing elective, minimally invasive, laparoscopic gastric bypass surgery with pain and swelling at one of the port sites on the abdomen. On physical examination, the port site was erythematous and warm with ulceration over the incision and minimal purulent drainage. The patient also reported generalized abdominal pain. Her symptoms were associated with leukocytosis of 15,000 cells/mm³ (reference range, 4000–11,000 cells/mm³), but she was afebrile. The patient was admitted to general surgery with a presumed postoperative wound infection. The incision was reopened, but there was no evidence of abscess or collection of fluid. Computed tomography of the abdomen and pelvis were unremarkable. Wound and blood cultures were obtained with negative results. After consulting the infectious disease department, the patient was started on empiric broad-spectrum antibiotics. The lesion and the surrounding erythema increased in size over the next few days. Cultures remained negative. Despite broad-spectrum antibiotic coverage, the lesion continued to drain purulent material and developed a necrotic center. The necrotic tissues were debrided several times; however, the lesion continued to enlarge. The dermatology department was consulted and the possibility of pyoderma gangrenosum (PG) was raised. At that time the ulcer had grown to a size of 15×20 cm; it had a purulent exudate and purple undermined borders with surrounding erythema (Figure 1). The other 3 port sites had healed well. The abdomen was tender to palpation with no rebound tenderness. The patient had severe nonpitting edema of the bilateral lower extremities.

A biopsy of the lesion was obtained and sent for hematoxylin and eosin staining as well as tissue cultures. Bacterial, acid-fast bacilli, and fungal tissue cultures were negative. The cultures were followed for

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**Practice Points**

- Pyoderma gangrenosum (PG) is a rare postsurgical complication that should be considered in the setting of nonhealing wounds with negative cultures.
- Prompt diagnosis of PG is important, as continued debridement by the surgical team will worsen the condition.
- Once diagnosis is established, the patient should be evaluated for any associated conditions, most importantly malignancy.
- In cases in which systemic immunosuppressive therapy is contraindicated, treatment of severe PG with superpotent topical corticosteroids may be attempted.
2 months and remained negative. Histology revealed epidermal ulceration; dermal necrosis; and a massive, predominantly neutrophilic infiltrate extending to the panniculus (Figure 2). Stains for fungi, acid-fast bacilli, and bacteria remained negative.

Shortly after the diagnosis of PG was made (15 days after initial presentation), the patient was started on clobetasol ointment 0.05%. Systemic therapy, such as prednisone or infliximab, was not used due to patient and surgical team preference. The patient began to show slow improvement and finally was discharged from the hospital after 29 days with continued use of topical clobetasol. On follow-up 1 month later, the lesion was completely healed with an erythematous cribriform scar and minimal striae. The patient continued to do well with no evidence of recurrence 4 months following the initiation of treatment (Figure 3).

Comment
Pyoderma gangrenosum is a rare neutrophilic dermatosis that typically affects patients aged 20 to 50 years, with an annual incidence of approximately 3 to 10 cases per million.1 The etiology of PG remains unknown; however, approximately 50% of cases are associated with an underlying disease. Pyoderma gangrenosum is most commonly associated with inflammatory bowel disease, but it also is a rare cutaneous manifestation of Crohn disease and ulcerative colitis. Other associations include seronegative spondyloarthropathies, rheumatoid arthritis, hepatitis C virus, and a variety of hematologic disorders (eg, malignancies, paraproteinemias, myelodysplastic syndrome, myelofibrosis).1,2 Drug-induced PG also has been reported.3

Clinically, there are 4 variants of PG: ulcerative, pustular, bullous, and vegetative. The most common site of presentation is the lower extremities, but any part of the body may be involved, including the

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Figure 1. Ulceration at a laparoscopic port site following gastric bypass surgery.

Figure 2. A biopsy of the ulcer showed a dense inflammatory pandermal infiltrate that was primarily neutrophilic (A and B)(H&E; original magnifications ×4 and ×20, respectively).

Figure 3. At 4 months’ follow-up after initiating treatment with clobetasol ointment, the lesion was completely healed.
mucosa and internal organs. Pyoderma gangrenosum may exhibit pathergy, meaning it may occur in areas of prior trauma or surgery. The course may be acute, as seen in PG associated with lymphoproliferative disorders, which presents with fever, malaise, and a painful, rapidly expanding ulcer. Conversely, PG seen in patients with inflammatory bowel disease follows a more chronic indolent course. Relapses of up to 70% have been reported.1

Pyoderma gangrenosum has been reported to occur postoperatively, with the majority of cases associated with breast surgery.2 It also has been described following hernia repair, coronary artery bypass grafting, abdominal surgery, and orthopedic surgery. Some of the patients in these case reports had underlying medical conditions, which is known to be associated with PG, while other patients had no known risk factors, as seen in our case.3,4,6

A PubMed search of articles indexed for MEDLINE using the search terms surgery, laparoscopy, pyoderma gangrenosum, and complications revealed 5 cases of PG occurring in a port site following laparoscopic surgery.4,5,7,8 In one of these cases, the patient’s medical history was remarkable for an isolated episode of PG that recurred 1 year after a laparoscopic cholecystectomy.1 In another report, a patient with myelodysplastic syndrome developed PG after a laparoscopic appendectomy.5 Finally, similar to our case, Noblett and Woodcock reported a patient with no predisposing conditions and no known associated diseases who developed PG after a laparoscopic hernia repair.

The onset of PG in most cases has been noted in the first week following surgery, but the range of onset was between 3 days and 6 weeks.3 Many cases of postsurgical PG were associated with systemic symptoms including fever, malaise, leukocytosis, and an elevated C-reactive protein.2,4,7 Two cases had severe life-threatening systemic manifestations of culture-negative sepsis and respiratory failure.5,6 Thus it appears that PG in a postsurgical setting follows an acute and fulminant course, similar to hematologic malignancies. In the majority of the postsurgical cases of PG in the literature, the diagnosis was delayed; the initial etiology was thought to be infectious, leading to a delay in treatment and remarkable morbidity.2,7,9

Pyoderma gangrenosum in a postsurgical setting is difficult to diagnose for several reasons, primarily because the initial presentation can be suggestive of a wound infection. Clinically, the patient may appear ill with a postsurgical wound that is painful and erythematous with purulent discharge and often is secondarily infected, leading to an initial misdiagnosis. Pathology is not helpful, as the results generally are nonspecific and cannot differentiate from infection. Lastly, many general surgeons do not consider PG in the differential diagnosis because it is quite rare; however, prompt diagnosis is important because a delay in treatment can cause considerable morbidity and mortality.1 Once the diagnosis is made and treatment with systemic therapy is initiated, the patients do well and improve rapidly. Our case is unique in that our patient responded extremely well to topical corticosteroid therapy alone, which is quite unusual in such severe cases. Topical treatments, including corticosteroids, tacrolimus, and general wound care, commonly are used for mild cases of PG or as an adjunct to systemic therapy in severe cases.9

Conclusion
Our case emphasizes the importance of including PG in the differential diagnosis for nonhealing postsurgical wounds that have either been culture negative or unresponsive to appropriate antibiotic therapy. In these cases, prompt instigation of therapy can substantially reduce morbidity and prevent mortality. In addition, we underscore the role of topical corticosteroid therapy in patients with contraindications to systemic immunosuppressive agents.

REFERENCES