Pyoderma Gangrenosum Following Bilateral Deep Inferior Epigastric Perforator Flaps

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Background

Pyoderma gangrenosum (PG) is an atypical ulcerative cutaneous condition, with an estimated six cases per million people per year in the United States.¹ One-half of patients have idiopathic disease, and it is most commonly associated with underlying systemic inflammatory conditions or hematologic malignancies.^{1,2} There have been several reported cases of PG following breast reduction procedures; however, postsurgical PG (PSPG) is rarely seen following autologous tissue breast reconstruction.³

In this article, the authors report the clinical course, treatment, and outcome of a patient diagnosed with PSPG following a delayed breast reconstruction with bilateral deep inferior epigastric perforator (DIEP) flaps. The objective of reporting this case is to further the understanding of the clinical presentation and treatment of PSPG as a complication in patients who present with cutaneous ulcerations following autologous tissue breast reconstruction.

Case Report

A 58-year-old Caucasian female with a history of right breast invasive lobular carcinoma who was status-post bilateral mastectomy and right chest wall radiation presented for delayed breast reconstruction. Due to her history of radiation, body habitus, and personal preference, she was found to be a good candidate for a bilateral DIEP flap. The patient had a 25-pack-year history of smoking that she quit 15 years ago, a history of hepatitis A, gastroesophageal reflux disease, hyperlipidemia, osteopenia, vitamin D deficiency, ocular hypertension, and migraines. She denied a history of autoimmune diseases or colitis.

The patient's intraoperative course was uneventful. The internal mammary artery and vein were used as the recipient vessels (►Fig. 1). Her postoperative course was uneventful

and she was discharged on postoperative day 5 with aspirin, analgesics, antibiotics, stool softeners, and drains in her abdomen and chest.

On postoperative day 7, the patient noticed sudden onset of right breast pain and erythema within the flap and along her incisions. She presented to the emergency room and her breast flap was noted to be swollen, erythematous, weeping serous fluid with blistering along the superior flap incision (►Fig. 2). Clinical examination and Doppler assessment of perforators demonstrated excellent perfusion of both flaps. Radiographs showed no evidence of retained foreign objects. Laboratory evaluation demonstrated a white blood cell count of 10.4 with a slight left shift. Urinalysis, basic metabolic panel, and blood cultures were all negative. She did have an elevated erythrocyte sedimentation rate of 111 (normal: 0–30). The patient was initially diagnosed with a suspected necrotizing soft tissue infection, and was placed on broad spectrum intravenous antibiotics and followed by infectious disease (►Fig. 3). Due to minimal improvement of the erythema and swelling, a computed tomography (CT) scan was performed in hospital at day 6. CT showed no evidence of abscess, hematoma, or seroma, but there was evidence of soft-tissue edema and stranding.

The negative cultures, lack of response to antibiotics, and negative CT scan led to concern for pyoderma gangrenosum (PG). Dermatology was consulted, and the patient was started on PO prednisone at 80 mg/day and clobetasol 0.05% cream. A biopsy was avoided to prevent worsening of disease. After noting immediate improvement in erythema and regression of the blisters, the patient was given the official diagnosis of PSPG. She was subsequently discharged on POD 16 with oral and topical steroids, and her follow-up on POD 18 revealed continued improvement of the PG. Six weeks later, all of the patient's wounds were healed with resolution of the right flap erythema (►Fig. 4).

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Fig. 1 POD4. One day prior to discharge from hospital.

Discussion

PG is a rare, sterile, inflammatory, ulcerative disease that causes painful, progressive necrosis of the skin.^{4,5} The cause of PG remains unclear; however, one-half of patients with PG have underlying systemic inflammatory conditions, including inflammatory bowel disease and rheumatoid arthritis, or hematologic diseases.⁶ The remaining one-half of patients with PG appear to have idiopathic disease with no apparent underlying disease process associated with PG, as was the case in this patient.²

Classical PG presents along previously intact skin as an inflammatory papule or pustule that rapidly progresses to a large, necrotic, painful ulcer with a violaceous border. Unlike classical PG, PSPG presents along the site of surgical incision an average of 7 days after surgery with erythema, tenderness, and cutaneous ulcerations. This initial presentation rapidly progresses to extensive wound dehiscence and ulceration expansion with a characteristic violaceous border sur-

Fig. 3 POD 15. Hospital day 6. Ulceration on superior aspect of flap larger in width and length. Prominent ulceration on superior border of flap. Patient continued on intravenous and PO antibiotics. Dermatology was subsequently consulted for a possible diagnosis of pyoderma gangrenosum and the patient was started on high dose steroids.

rounded by erythema.^{1,6,7} Due to its occurrence within the postoperative period, PSPG is often misdiagnosed as a postoperative wound infection, which leads to antibiotic administration and surgical debridements that are ineffective in preventing wound progression and unfortunately cause rapid ulceration enlargement due to pathergy. Misdiagnosis, surgical debridements, and improper treatment can result in devastating outcomes and unsatisfactory aesthetic results with extensive deformation and scarring.^{7,8} There are several reports in the literature of PSPG following breast surgery; however, PSPG is very rarely seen following autologous tissue breast reconstruction.⁶

Fortunately, our team recognized the patient's diagnosis with PSPG prior to performing any surgical debridements, preventing deformation and possible loss of the DIEP flap. The literature reports successful treatment of PSPG with high-dose systemic corticosteroids (40--120 mg/day) with

Fig. 2 POD 8. Patient admitted to emergency room. Unilateral swelling and inflammation of the right breast flap. Erythema confined to the right deep inferior epigastric perforator flap skin, abdominal, and umbilical incisions. There was no erythema of the left breast.

Fig. 4 Six weeks following initiation of steroid treatment. Pyoderma gangrenosum lesions healed with resolution of erythema.

tapering when there is wound stabilization.^{2,6–8} Treatment with high-dose prednisone promoted regression of the PSPG and allowed wound healing to progress in our patient. While it is a rare complication following autologous tissue breast reconstruction, delaying proper treatment of PSPG may result in devastating complications and outcomes.³ Therefore, it is prudent to include PSPG in the differential diagnosis for a patient presenting approximately 1 week after surgery with erythema and cutaneous ulcerations along surgical incisions, particularly if there are negative blood and wound cultures, an elevated sedimentation rate, and nonresponsiveness to antibiotics and wound care.

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References

- Schadt C. Pyoderma gangrenosum: pathogenesis, clinical features, and diagnosis. Waltham, MA: UpToDate; 2014. Available at <http://www.uptodate.com/contents/pyoderma-gangrenosum-pathogenesis-clinical-features-and-diagnosis>. Accessed on July 8, 2015
- Callen JP. Pyoderma gangrenosum. *Lancet* 1998;351(9102):581–585
- Momeni A, Satterwhite T, Eggleston JM III. Postsurgical pyoderma gangrenosum after autologous breast reconstruction: case report and review of the literature. *Ann Plast Surg* 2015;74(3):284–288
- Brunsting LA, Goeckerman WH, O'Leary PA. Pyoderma (echthyma) gangrenosum: clinical and experimental observations in five cases occurring in adults. *Arch Derm Syphilol* 1930;22(4):655–680
- Farhi D. The clinical and histopathological description of geometric phagedenism (pyoderma gangrenosum) by Louis Brocq one century ago. *Arch Dermatol* 2008;144(6):755
- Zuo KJ, Fung E, Tredget EE, Lin AN. A systematic review of post-surgical pyoderma gangrenosum: identification of risk factors and proposed management strategy. *J Plast Reconstr Aesthet Surg* 2015;68(3):295–303
- Caterson SA, Nyame T, Phung T, Lee B, Tobias AM. Pyoderma gangrenosum following bilateral deep inferior epigastric perforator flap breast reconstruction. *J Reconstr Microsurg* 2010;26(7):475–479
- Tuffaha SH, Sarhane KA, Munding GS, et al. Pyoderma gangrenosum after breast surgery: diagnostic pearls and treatment recommendations based on a systematic literature review. *Ann Plast Surg* 2016;77(2):e39–e44