Case Report

Development of Pyoderma Gangrenosum after a Deep Inferior Epigastric Perforator Breast Reconstruction

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ABSTRACT

Pyoderma gangrenosum is a rare skin disorder and a part of the spectrum of neutrophilic dermatoses. However, its etiology remains unknown. It is characterized by ulcer-like active skin lesions and may develop in skin areas subjected to surgical intervention or injury. Here, we report a rare case of a patient who developed pyoderma gangrenosum after an autologous unilateral breast reconstruction and simultaneous contralateral breast augmentation. In our patient, lesions developed on the left breast and on the abdominal donor site with surgical dermal scarring; however, the other breast was unchanged, without scarring. The patient recovered after a systemic steroid treatment and negative pressure wound therapy.

Key words: breast reconstruction, neutrophilic dermatoses, pyoderma gangrenosum

Instruction

Pyoderma gangrenosum (PG) is a rare skin disorder of undetermined etiology, first described by Brunsting et al. in 1930, and characterized by ulcers, bullae, and pustules. Approximately 50%–70% of patients have systemic diseases that are considered idiopathic, such as rheumatoid arthritis, inflammatory bowel disease, and hematologic disease. Approximately 25%–50% of PG cases develop after skin trauma, such as surgical wounds, vessel puncture, or laparoscopy. Post-surgical PG is difficult to diagnose because its appearance is similar to that of a wound infection or necrotizing fasciitis. A diagnosis of PG is not typically considered until the lesions are exacerbated upon debridement.

Recent studies have reported that PG can develop after a breast reconstruction surgery. In previous cases of PG developing after an autologous breast reconstruction, the wound onset typically occurred 5 days postoperatively. Patients were treated with systemic steroids and/or immunosuppressive medications, and complete wound healing occurred at an average of 4 mo. Here, we report a rare case of a patient who developed PG after breast reconstruction and recovered after treatment with systemic steroid administration and negative pressure wound therapy (NPWT).

Case report

A 50-year-old woman with a medical history of cesarean delivery, cervical cancer, and left breast cancer presented to our hospital for delayed breast reconstruction (Fig. 1). Two years after undergoing a left mastectomy and insertion of a tissue expander, the patient underwent left-sided breast reconstruction using the deep inferior epigastric perforator flap, superficial circumflex iliac artery perforator flap, and right-side breast augmentation by performing dermal fat grafting under the mammary gland subcutaneously through the midline tunnel. The total operative time was approximately 6 h.

Postoperative recovery was uneventful until postoperative day (POD) 3, when bullae appeared under the film dressing. The patient developed fever (38–39 °C) at night on POD 6 and was administered levofloxacin (500 mg/d). On POD 8, redness...
and swelling were observed around the drain holes after removal of the drainage tube. Subsequently, a necrotizing wound developed on the surgical scars on both the left breast and abdominal regions; the wound rapidly evolved over the next few days. We did not observe any redness or other changes on the right breast, which had not been subjected to direct skin incision for dermal fat grafting. On POD 13, painful ulcers with necrotic tissue surrounded by pustules and redness were observed (Fig. 2). Laboratory examination revealed leukocytosis (white blood cell count, 29,040/μL) and an elevated C-reactive protein level (23.6 mg/dL) (Fig. 3). Wound bacterial culture results were negative. Computed tomography revealed inflammation localized to the skin and shallow subcutaneous tissues.

The patient was suspected to have postoperative wound infection. On the basis of this probable diagnosis, necrotic tissue debridement on the breast and abdominal wounds was performed on POD 14. However, the necrosis had not progressed beyond the dermis (Fig. 4). Therefore, she was referred to an infectious disease team, who recommended the addition of teicoplanin (400 mg/day) and tazobactam/piperacillin (13.5 mg/day) to her treatment regimen. However, the lesions continued to progress and exacerbated after debridement (Fig. 5).
Finally, on POD 16, the characteristic clinical findings, such as painful necrotic ulcers, progression after debridement, and negative wound cultures led to the clinical diagnosis of PG. However, biopsy was not performed because of concerns about additional pathology.

The patient was immediately administered oral prednisolone (50 mg/d), which prevented the development of new lesions (Fig. 3 and 6). Although lesion debridement was performed on POD 39, the lesions had not exacerbated. NPWT (the V.A.C. system, Kinetic Concepts, Inc., Texas, USA) was performed on all lesions for 13 days (Figs. 3 and 7). Prednisolone was tapered off, and the total administration period was 11 weeks. Moist wound healing using bucladesine sodium salt and Vaseline was continued until epithelialization. All breast and abdominal lesions completely healed 4 mo postoperatively (Fig. 8). The right-side breast, which had been subjected to augmentation by dermal fat grafting, was still soft without findings of fat necrosis postoperatively.
Discussion

Up to 50% of PG lesions are located in skin areas that have been subjected to trauma, such as vessel puncture, laparoscopy, and surgical incisions, a phenomenon known as pathergy. The diagnosis of PG is based mainly on clinical findings because biopsies do not show specific diagnostic features. However, a biopsy can help rule out other conditions, such as malignancy, infections, or cutaneous vasculitis in many cases.

Several reports have also described post-surgical PG, which refers to the development of PG at surgical sites in the immediate postoperative period, typically after 1 wk. Post-surgical PG occurs most frequently after breast surgery (25%), followed by cardiothoracic, abdominal, and obstetric surgical procedures. The first operation does not always cause PG, as it usually develops after a “delayed” breast reconstruction in patients without a history of PG. Furthermore, among patients with a history of PG, 5.5% of the procedures led to PG recurrence in 15.1% of patients, showing that surgical operation could be a trigger for pathergy, but not always.

In cases of PG developing after an autologous breast reconstruction, wound onset typically occurs 5 days postoperatively. Systemic steroids and/or immunosuppressive medications (cyclosporine A) are administered as the most common medical therapy for post-surgical PG. Infliximab, tacrolimus, and hyperbaric oxygen therapy were also administered. Using these therapies, complete wound healing occurs in an average of 4–5.1 mo.

Rapid wound improvement upon administration of steroids or immunosuppressive therapy is indicative of PG. After confirming the patient’s response to steroid therapy, local gentle wound care enabled wound healing. Methods, such as moist wound healing, wet-to-dry dressing, and NPWT are used to support wound healing. The use of NPWT is controversial because it can be traumatic and result in pathergy; however, it has been reportedly safe and effective for treating localized wounds in patients undergoing steroid or immunosuppressive therapy. Surgical intervention in PG is controversial because of the risk of triggering pathergy, but minimal debridement as local wound control is relevant if the patient has responded well to steroid or immunosuppressive therapy and gentle local wound care.

In our patient, bullae occurred on POD 3, fever developed on POD 6 (considered an early symptom), and the first debridement (performed since wound infection was suspected) exacerbated the lesions, all of which led to a diagnosis of PG. Unfortunately, the diagnosis was delayed for approximately 2 wks from the first onset of symptoms. Three weeks after commencing systemic steroid therapy, surgical debridement and NPWT were initiated; both were safe and effective, as the patient was being treated with steroids.

In cases of PG after an autologous breast reconstruction, lesions often concurrently occur at several sites, typically at both the donor and recipient sites or on both breasts in case of bilateral breast reconstruction. In our patient, necrotizing wounds developed on both the left breast and abdominal donor site; however, no change was observed on the right breast, which had no scarring, even though both breast sites were connected under the skin. Retrospectively, these observations may have enabled the accurate diagnosis of this case as PG rather than a skin infection. To the best of our knowledge, this is the first report on the association between the onset of PG and skin scarring in the same case regardless of invasion under the skin.

In summary, PG is a rare, but painful skin disorder that may develop after a breast reconstruction surgery. The occurrence of PG lesions is similar to lesions that develop due to wound infection; however, correct diagnosis and early management are important. Steroid treatment or other types of immunosuppressive therapy should be initiated without delay. NPWT and surgical intervention may be effective combined with appropriate medication therapy. In our case, the fact that several lesions simultaneously occurred was an indicator of PG development after an autologous breast reconstruction.

Conflicts of interest

None.

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