Use of an External Tissue Expansion Device as a Minimally Traumatic Closure Method for Pyoderma Gangrenosum Following Total Hip Arthroplasty

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Background	Pyoderma gangrenosum (PG) is a rare but serious postoperative complication. Diagnosis and treatment are often delayed due to confusion with surgical site infection. PG should be considered in the differential diagnosis for all early postoperative skin changes, particularly when lesions are progressive despite appropriate antibiotic therapy, exacerbated by surgical debridement, and in the setting of systemic disease.
Summary	A 68-year-old female with degenerative joint disease underwent total hip arthroplasty. One week postoperatively, there was drainage, inflammatory skin changes at the incision, and increasing pain. She was treated with antibiotics and surgically debrided twice; however, she continued to have skin changes extending beyond the edges of the incision. Cultures were negative and biopsy revealed neutrophilic infiltrates with ulceration of dermal and subcutaneous components, dermal edema and intra-epidermal pustules, confirming a diagnosis of PG. Antibiotics were discontinued and the patient was started on IV methylprednisolone and topical tacrolimus ointment. The wound stabilized and ten days later, staged wound closure was performed using an external tissue expansion device (DermaClose RC). The device was applied for 10 days to apply controlled, continuous traction on the skin edges. Afterwards, the device was removed, and the wound was primary closed. At one year follow-up, the patient had a healed wound and successful salvage of her prosthetic joint.
Conclusion	Treatment of PG is challenging. Closure methods often cause more trauma and may exacerbate the condition. We present a case of postoperative pyoderma gangrenosum following total hip arthroplasty treated successfully with systemic corticosteroids, local wound care, and an external tissue expander device.
Keywords	Pyoderma gangrenosum, tissue expander, pathergy, Dermaclose, surgical site infection, total hip arthroplasty

DISCLOSURE:
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Case Description

Pyoderma gangrenosum (PG) is a rare skin condition within the spectrum of neutrophilic and autoinflammatory dermatoses. The disease is characterized by pustules that progress into sterile, ulcerative cutaneous lesions. Most eruptions are spontaneous and occur in association with hematologic, rheumatologic, or gastrointestinal disease. Lesions can also develop at sites of trauma. Postsurgical PG is most commonly reported after breast and orthopedic surgery[1-10] and is often confused with wound infections.[2,11,12] The disease may be rapidly progressive if not identified and treated appropriately. Additional surgical trauma exacerbates the disease process.

Treatment of PG is challenging. Lesions are often large at the time at diagnosis. We present a case of postoperative pyoderma gangrenosum following total hip arthroplasty treated successfully with systemic corticosteroids, local wound care, and an external tissue expander device (DermaClose RC).

A 68-year-old female with degenerative joint disease underwent total hip arthroplasty. One week postoperatively, there was drainage, inflammatory skin changes at the incision (Figure 1), and increasing pain. She was treated with antibiotics and surgically debrided twice; however, she continued to have skin changes extending beyond the edges of the incision. Cultures were negative and biopsy revealed neutrophilic infiltrates with ulceration of dermal and subcutaneous components, dermal edema and intra-epidermal pustules, confirming a diagnosis of PG (Figure 2). The resultant soft tissue defect measured 20cm x 25cm (Figure 3).

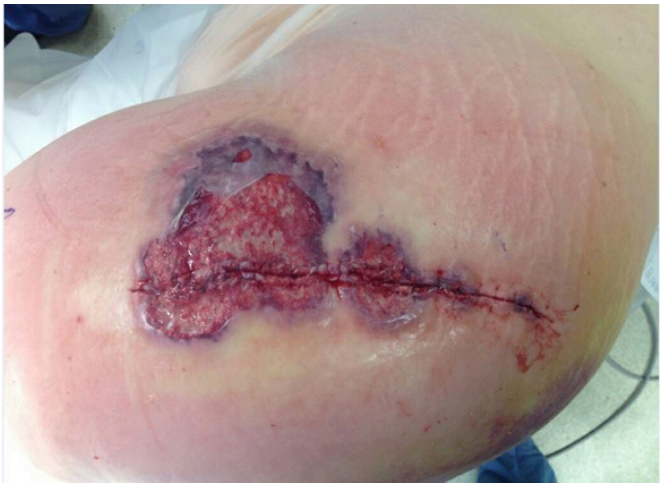

Figure 1. Surgical incision one week postoperatively.

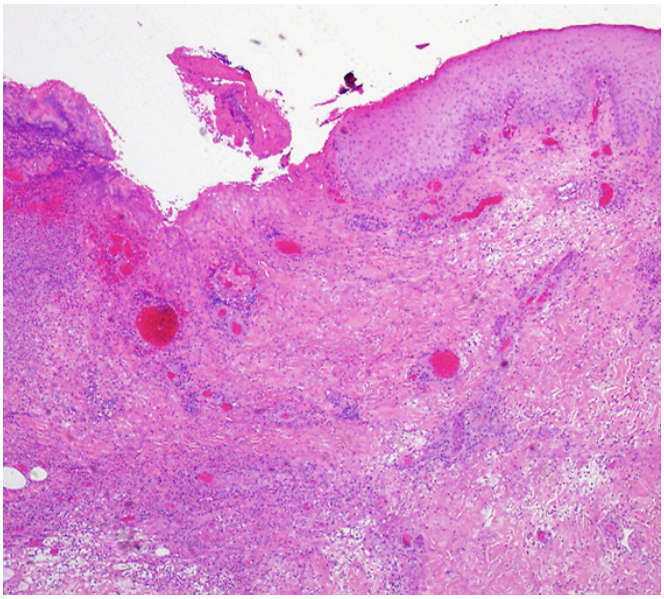

Figure 2. Hematoxylin and eosin stain of wound biopsy on lower power. Skin ulceration, necrosis and inflammatory infiltrate present.

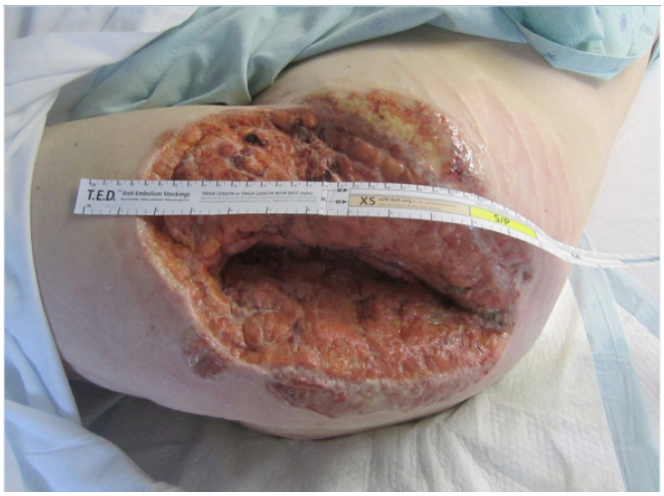

Figure 3. Wound after second debridement, 20 x 25cm.

During the first debridement, the prosthetic joint was removed and replaced with a new device due to presumed contamination. The joint capsule was not violated during the second debridement and the prosthetic was left in place.

No additional debridements were performed once the diagnosis of PG was made. Antibiotics were discontinued and the patient was started on IV methylprednisolone and topical tacrolimus ointment. The wound stabilized, and ten days later, staged wound closure was performed using an external tissue expansion device (DermaClose RC). The device was in place for 10 days, applying controlled, continuous traction on the skin edges (Figure 4); it was then removed and the wound was primary closed (Figure 5). At one year follow-up, the patient had a healed wound and successful salvage of her prosthetic joint (Figure 6).

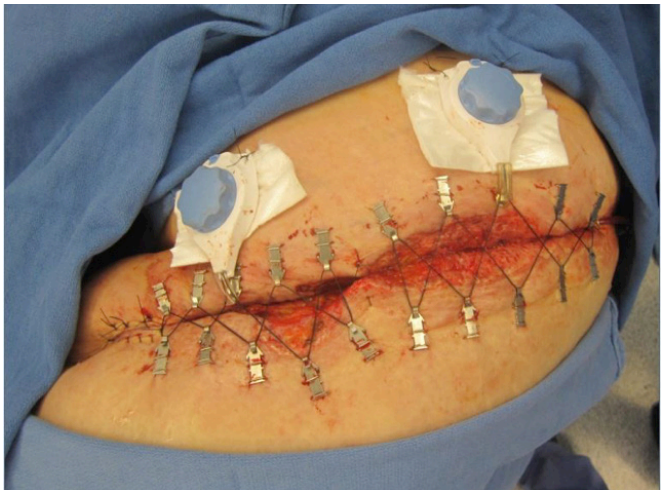

Figure 4. Wound with two DermaClose devices in place after 10 days of expansion

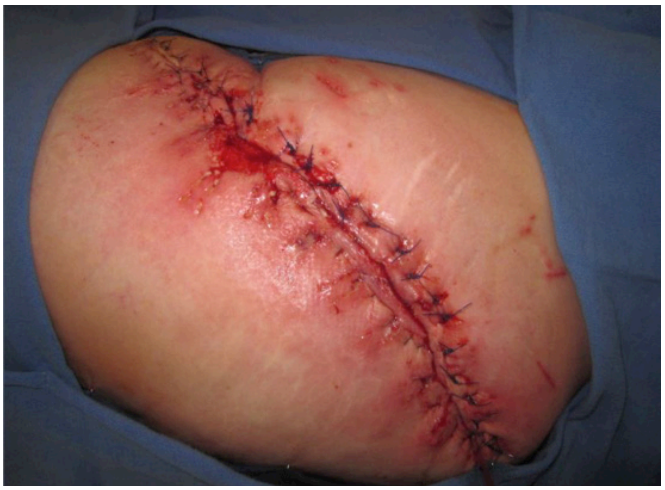

Figure 5. Immediate postoperative photo after removal of external tissue expander.

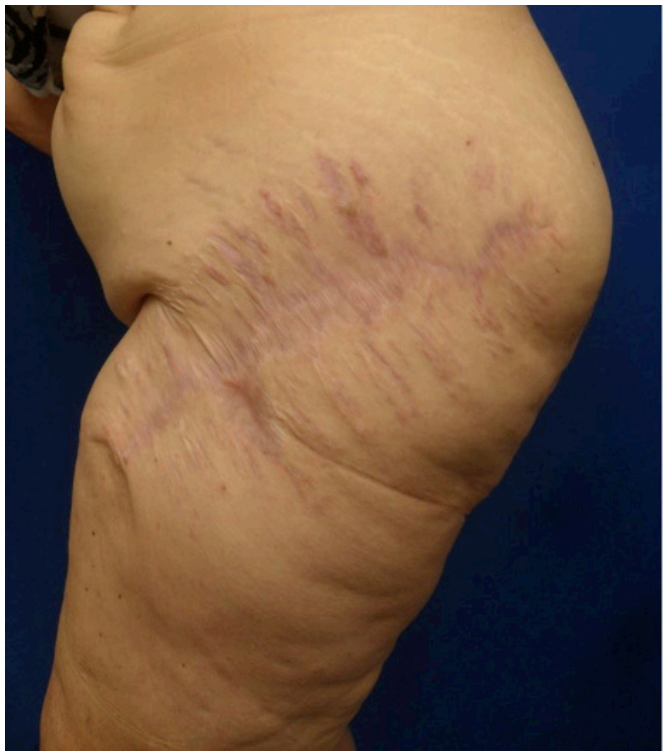

Figure 6. Surgical incision one year postoperatively.

Laboratory work-up for systemic disease, including CBC, CMP, hepatitis panel, ANA, ANCA, SSA/SSB, lupus anticoagulant, anticardiolipin antibodies, and anti-beta 2 glycoprotein antibodies, was negative. Chest Xray revealed no abnormalities. Colonoscopy was negative for inflammatory bowel disease and malignancy.

Discussion

Pyoderma gangrenosum is a rare but serious postoperative complication. Diagnosis and treatment is often delayed due to confusion with surgical site infection.[2,11,12] PG should be considered in the differential diagnosis for all early postoperative skin changes, especially when lesions are progressive despite appropriate antibiotic therapy, exacerbated by surgical debridement, and in the setting of systemic disease (inflammatory bowel disease, hematologic disorders, autoimmune syndromes or malignancy).

Diagnosis is based on characteristic clinical and pathologic features. The hallmark lesion of classical PG is a painful, rapidly enlarging, violaceous ulcer with overhanging or undermined borders and a necrotic base.[13] Histopathology nearly always reveals neutrophilic dermal infiltration. Leukocytoclasia, abscess formation, and leukocytoclastic vasculitis are less frequently observed. Although the pathological findings are not specific, biopsy is important to rule out other causes of ulceration such as infection, vasculitis, or malignancy.[14]

PG is associated with systemic disease in 33 to 84 percent of cases.[14] Inflammatory bowel disease is the most frequent coincident pathology (20 to 30 percent).[14] Other associations include hematologic disorders (monoclonal gammopathy, myelofibrosis, myelogenous leukemia, hairy cell leukemia), autoimmune disease, solid tumors, and infection (HIV, chronic hepatitis). Skin lesions can occur before, after, or coincident with systemic illness. Additional work-up should include laboratory studies (CBC, ESR, liver and kidney profiles, serum and urine protein electrophoresis, coagulation panel, antiphospholipid antibodies, ANA, cryoglobulins), peripheral blood smear, bone marrow aspirate, colonoscopy, and chest X ray.[2] Treatment of the underlying condition is critical.

Treatment of pyoderma skin lesions consists of local and systemic therapy. Local wound care focuses on maintaining optimum moisture balance in the lesion. Topical medications commonly used include corticosteroids, tacrolimus,[15] and sodium cromoglycate. Intralesional injections of corticosteroids, cyclosporine,[16] and phenytoin[17] have also been effective. Systemic therapy should be considered for aggressive lesions. Corticosteroids and cyclosporine are first line treatments. Immunomodulatory therapy and biologic response modifiers, such as mycophenolate mofetil, tacrolimus, dapsone, azathioprine, and infliximab, are also sometimes used.[14]

Surgery should be avoided on active PG lesions. Debridement may exacerbate the problem due to a pathergic response, in which minor skin trauma results in additional ulceration. If surgery is unavoidable, it should be performed only after inflammation has been controlled with topical and systemic immunosuppresants.[13]

Effective management of soft tissue defects associated with PG is a challenge. Numerous reconstructive options normally exist for large, ulcerative lesions, including healing by secondary intention, delayed primary wound closure, skin grafting, local or regional flaps, and free tissue transfer[18]; however, extensive surgery should be avoided in patients with a history of PG for the above reasons. Split thickness skin grafting has been used with some success but recurrence of PG at the recipient site has been reported in several cases.[13] Donor site morbidity is also a concern as new PG lesions may develop at these locations as well. Amputation is a last resort, but is known to improve quality of life in patients with severe, refractory PG.[19]

External tissue expanders have been used to reduce wound burden in traumatic soft tissue defects,[20] fasciotomy wounds,[21] donor sites after flap harvest,[22] and wounds resulting from oncologic resection.[23,24] Mechanical manipulation of the skin has been utilized since the 1950s[25] and is based on the principals of biological creep, mechanical creep, and stress relaxation. When skin is stretched beyond its physiologic limit, transmembrane mechanoreceptors induce a series of events that result in increased mitotic activity and collagen synthesis, a process termed biological creep. The effect is increased tissue mass. Existing collagen fibers elongate and realign parallel to each other, allowing the skin to stretch. The increase in length of a tissue mass is called mechanical creep. Stress relaxation is defined as the decrease in retractive force exhibited by a material when it is held at a given stretch over time. The end result is an increase in skin surface area when an external force is applied over time.[26]

External tissue expanders facilitate closure of full-thickness defects by applying continuous traction on the skin. A number of methods can be used for external expansion, including fashioning vessel loops into a Jacob's ladder,[21] rubber banding,[27] and retention sutures.[28] An off-the-shelf device (DermaClose RC) is now available to simplify the process of application and control the amount of force applied to the wound edges (Figure 6). Stainless steel anchors are placed in healthy skin 1–3 cm from wound edge and 2–3 cm apart from each other and secured with surgical staples. A nylon monofilament is laced through the anchors and force is applied by a tension controller. The tension controller is spring activated and internally calibrated such that once the device is activated, no additional adjustments are necessary. A constant force is applied to the wound edges while the device is in place.[29]

There are many advantages of external tissue expansion over other reconstructive methods, particularly in the treatment of PG. The technique is noninvasive, causes minimal trauma to the skin, and has no donor site morbidity. Expan-

sion creates phenotypically similar skin that is matched tissue in color, texture, thickness, and hair bearing status, leading to excellent cosmesis.[24] The device is easy to apply and use, achieves wound closure (or significantly reduces wound burden) in a short period of time,[20] and is cost-effective.[30,31] In addition, external tissue expanders may be used in conjunction with NPWT to decontaminate and achieve wound closure simultaneously.[20]

Known complications of external tissue expansion include blistering or maceration of the skin, peri-wound tissue ischemia or necrosis,[22] and scarring. These can be avoided by careful device positioning and protection (e.g., placing soft foam dressings underneath the skin anchors and tension controller),[20] close monitoring of surrounding tissue,[22] and removal of the device after a maximum of seven days.[29]

Conclusion

Effective management of postoperative PG is a challenge. The authors report the safe, effective use of an external tissue expansion device as a possible treatment option for the management of soft tissue defects associated with PG. Further studies are needed to validate applications for external tissue expanders.

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