Pyodermagangrenosum (PG) is rarely reported as a complication following breastsurgery.1-8 Itisan uncommon ulcerativecutaneous disorder ofunknownetiology. Itmayoccurinotherwise healthy personsor in association with asystemic disease. Overall, approximately 50% ofpatients have a systemic disease. The most common diseases associatedwith PG are ulcerativecolitis, Crohn’s disease and rheumatoidarthritis.9-13 Management ofPG involves medical control of theinflammatory phase ofPG and local wound care.

CASE REPORT

A 36-year-old woman with no history of systemicillness, allergy orautoimmune disease, underwent a bilateral inferior pedicle reduction mammoplasty. The patient complained of pain after two weeks following...
surgery. Erythema, edema, heat and superficial ulcers were noted along the breast incisions. There was no fever or other systemic symptoms. The blood count showed moderate leukocytosis. Initial diagnosis was wound infection, even though bacteriologic cultures were negative for microorganisms, the patient was sent home with oral antibiotic. The wounds failed to heal over the next month despite repeated courses of antibiotics and conservative wound care (Figure 1). She was then referred to the dermatology unit for further evaluation. PG was suspected and a wound biopsy was advised. Histopathologic evaluation of the biopsy specimen revealed fibroblast proliferation, neutrophils, eosinophils and mononuclear inflammatory cells along the ulcer site compatible with PG. No associated disease was detected. Antibiotics were discontinued and the treatment was continued with oral cyclosporin A 250 mg daily and a mixture of silverdine, bepanthene and prednol cream topically. Wounds on both breasts started to heal within two weeks and over the next three months; they completely healed by secondary intention (Figure 2).

**DISCUSSION**

PG was first described by Brunsting, Goeckerman, and O’Leary in 1930. They considered infection as an etiology of this disorder. However, no infectious cause was identified. The cause of PG still remains unknown. It may be idiopathic or it may be associated with various systemic disorders including inflammatory bowel diseases, arthritis, paraproteinemias and hematologic malignancies. Hepatitis C or other viral infections, Wegener’s granulomatosis, systemic lupus erythematosus and other autoimmune diseases are other likely causes. Twenty-three percent of cases are induced and aggravated by minor trauma or surgery and this points out the pathergy phenomenon in the PG. It was also reported as a complication of various surgical interventions, such as hernioplasty, cardiac surgery, cesarean section and breast surgery.

Since postoperative infection or synergistic gangrene can be clinically very similar to PG, initially the problem was thought to be the result of an infection. Necrotizing wound infections require immediate and aggressive surgical debridement. Conversely, in patients with PG, operative intervention can be complicated by pathergy and it may result in new ulcer formations on traumatized skin regions and may also be resistant to local wound care and antibiotics. Therefore, PG has to be considered in the diagnosis and management.

When PG is suspected, other infectious causes should be ruled out. Treatment of PG with immunosuppressive therapy would be disastrous since PG is a serious condition. Culture from the wound should be taken for bacteria, mycobacteria, atypical mycobacteria, and deep fungal infection because those conditions can mimic PG. When the accurate diagnosis is established by clinical presen-
PG usually requires aggressive local and systemic treatment. Local therapy is directed to relieve the pain, to prevent or to treat the secondary bacterial infection and to provide a convenient environment for wound healing. Local therapy includes cleaning the wound with saline or antibacterial agents such as hydrogen peroxide or benzoyl peroxide. Use of wet compresses and nonsensitizing topical antibacterial creams may be beneficial. In many cases, the wound can be left to heal by secondary intention. However, larger wounds may require skin grafting or flaps for closure. Operative management should be performed when medical therapy has controlled the inflammatory phase of PG. Long et al. suggested that the risk of pathergic PG was reduced when subcuticular sutures were used for skin closure, rather than sutures going through the skin. Systemic treatments include sulfones and other antimicrobials such as dapsone, clofazimine, minocycline, which have been used with different success rates. However, the recognized first line treatment of this disorder is immunosuppressive medication with steroids. Prednisone has proved to be the most consistently successful agent. It is crucial to control the disease rapidly; so high-dose prednisone at initial treatment is preferred with slow tapering to prevent recurrence. Immunosuppressive therapy includes the use of cyclophosphamide, melphalan, chlorambucil, imuran and cyclosporine. Cyclosporine has shown to be the most promising agent especially in severe recalcitrant cases of PG. In addition, long-term maintenance therapy may be required in some patients because of persistent and recurrent nature of the disease.

In summary, this case emphasizes the difficulty of distinguishing PG from an acute infection. Delay in diagnosing PG may lead to prolongation of therapy and extensive scar formation. This condition should always be considered in patients who develop rapid progressive nonhealing ulcers after surgery.

REFERENCES