skin lesions did not have the classic findings of edema, warmth, pain, purulent drainage, or response to antibiotic therapy. The abdominal donor site was closed without tension or ischemic signs like the mastectomy flaps; therefore, the development of identical lesions on the breast caused suspicion of a non-infectious etiology.

A high index of suspicion for skin pathergies (pyoderma gangrenosum, AFND, etc.) is necessary for prompt diagnosis. Early diagnosis can lead to faster treatment and help avoid potential morbidities of surgical debridement and skin trauma. However, it is critical to avoid immunosuppressive therapy before infection is ruled out because the early phases of AFND are similar in presentation to cellulitis and necrotizing fasciitis. We recommend prompt skin biopsy for tissue diagnosis, and we recommend non-adherent dressings and infrequent changes to minimize skin trauma. A multidisciplinary team approach including dermatology, hematology, and internal medicine is recommended to expedite diagnosis and treatment of the underlying disease.

We report the first case of AFND in a DIEP free flap for breast reconstruction that was successfully treated with corticosteroids and non-surgical management. Further research and long-term follow-up is warranted to develop consensus recommendations and treatment protocols for skin pathergy and wound breakdown in breast reconstruction.

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Calcifications on the Entire Legs of a Systemic Lupus Erythematosus Patient

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Systemic lupus erythematosus (SLE) is a relatively common entity in rheumatology. Skin involvements such as nodules, vasculitis, and calcifications are commonly reported in patients with SLE [1,2]. In our center, we encounter many rheumatoid patients referred from our rheumatology clinic. However, the case shown here was very unusual even to our experienced eyes.

A 37-year-old female patient, who had suffered from SLE for 16 years, had acquired progressive calcifications on the entirety of both of her legs. The calcification lesions were initially not severe and were

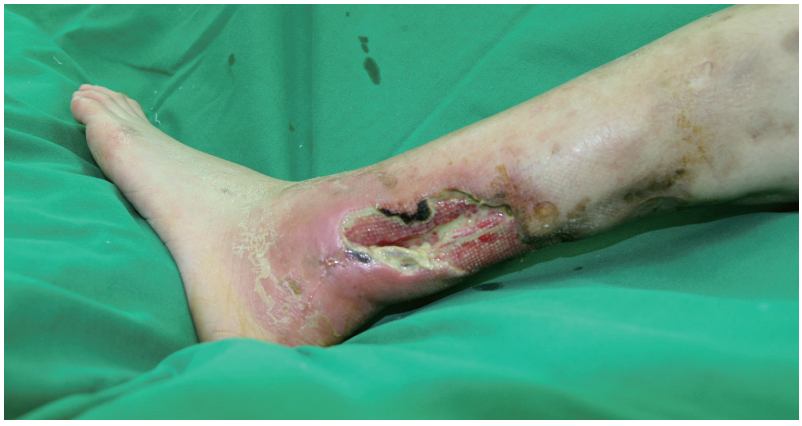

Fig. 1.
Multiple left leg defects. The left leg defects shown here are arose from calcinoses, which occurred in both legs.

Images

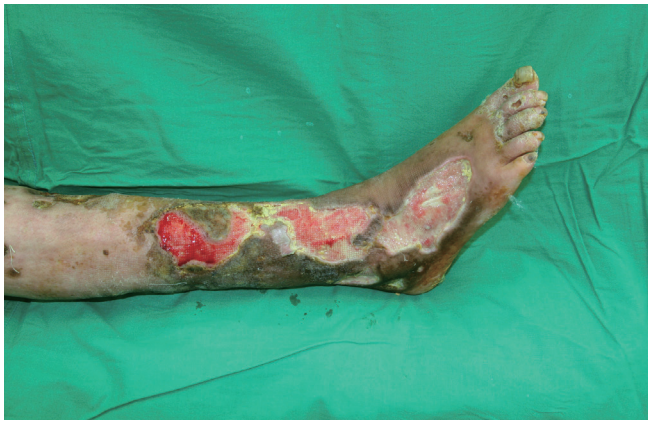

Fig. 2.
Multiple right leg defects. The right leg defects shown here are arose from calcinoses, which occurred in both legs.

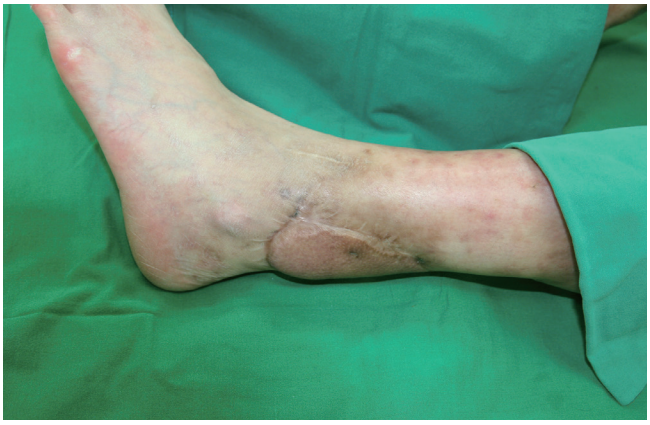

Fig. 4.
Postoperative view of the left leg. The left leg is shown after coverage of the defect with a thoracodorsal perforator free flap.

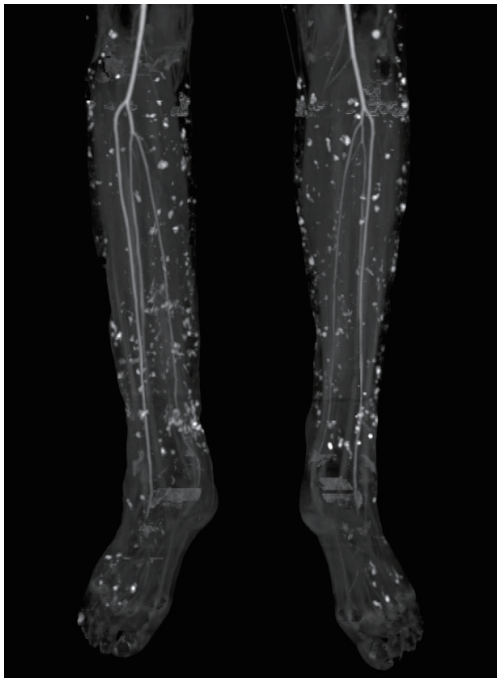

Fig. 3.
Computed tomography (CT) angiography of the legs. CT angiography clearly demonstrated the extent of the calcifications.

limited to the lower leg area. However, the progression accelerated into groups of calcifications on both legs, which resulted in multiple leg defects (Figs. 1, 2). Her computed tomography (CT) angiography clearly showed the extent of the disease (Fig. 3). To help heal the wounds, immune suppressive drugs were administered, but the patient's condition was serious. Though administration of intravenous immunoglobulin was temporary effective to control the SLE, the wounds worsened, which led to sepsis. The wounds progressed despite our efforts. We serially debrided the infected defects and covered the defects with free flaps and 0.010-inch split-thickness skin grafts from the thighs (Fig. 4). Up to the present, a total of 18 surgical procedures have been performed. The wounds are now stable with conservative treatment.

Calcifications with this extent of progression in a SLE patient are extremely rare [3]. Nevertheless, this case reminds us that we should not overlook skin involvements associated with SLE, especially progressive calcifications, which can lead to serious morbidities.

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