anechoic and comma- or mushroom-shaped unilocular on ultrasonography. In rare cases, it shows signs of a multilocular cystic mass with linear septa [3]. Although the CT scan showed the hydrocele of the canal of Nuck in the form of a homogenous fluid-filled unilocular cyst extending to the labia, its communication with the abdominal cavity could not be determined. MRI revealed well-defined, thin-walled, sausage-shaped cystic lesions in the hydrocele of the canal of Nuck. Low- and high-signal intensities were observed on the T_1- and T_2-weighted images, respectively [2].

The final diagnosis was made during surgery and confirmed by histopathological examination. The standardized therapeutic approach is to surgically remove edema and to ligate the processus vaginalis peritonei. Due to the rarity of hydrocele of the canal of Nuck, there are no reports on the recurrence rate of masses or simultaneous occurrence on the opposite side. The hernial sac can include peritoneal fluid, omental fat, bowel loops, the ovary, the fallopian tube, or the urinary bladder in the inguinal canal. Therefore, surgeons can avoid causing damage in the process of examining the anatomy around the inguinal canal by using preoperative imaging.

Despite the fact that hydrocele of the canal of Nuck occurs mainly in adolescence, we strongly suspected hydrocele of the canal of Nuck in this case based on the clinical symptoms and imaging diagnosis, and confirmed the diagnosis by surgical exploration without delay. This case suggests that hydrocele of the canal of Nuck should be included in the differential diagnosis of masses occurring in the inguinal area in adult females.

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Acute Febrile Neutrophilic Dermatosis after Deep Inferior Epigastric Perforator Flap Breast Reconstruction

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Acute febrile neutrophilic dermatosis (AFND), or Sweet syndrome, is a rare neutrophil-mediated hypersensitivity reaction of unknown etiology. There are four sub-classifications of AFND: (1) idiopathic,

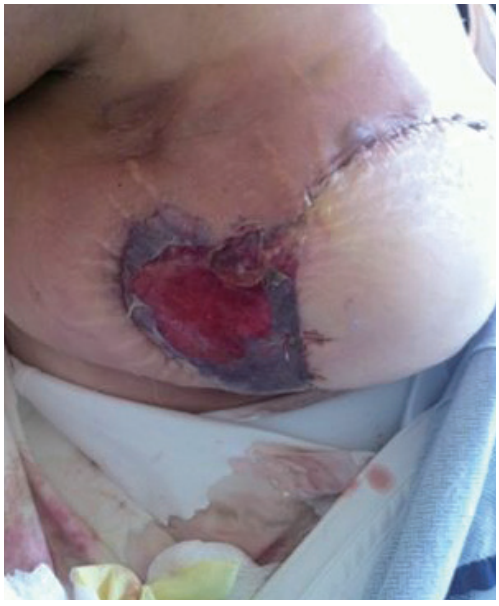

Fig. 1.
Postoperative day three: the neutrophilic dermatosis lesions are seen in evolution, ranging from violaceous papules to partial-thickness skin loss and exposed dermis. Of note, there was no purulence or malodorous drainage.

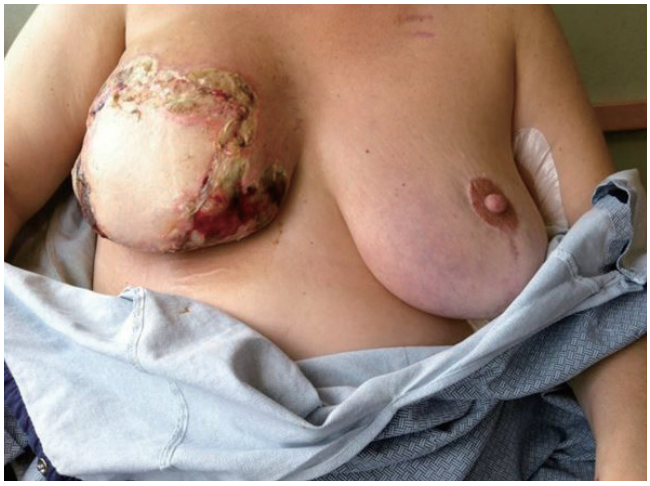

Fig. 2.
Postoperative day 13: bullous lesions progressed to complete desquamation and partial-thickness skin loss in areas of the right mastectomy flap in the deep inferior epigastric perforator free flap. However, no new areas of violaceous lesions developed after initiation of corticosteroid therapy.

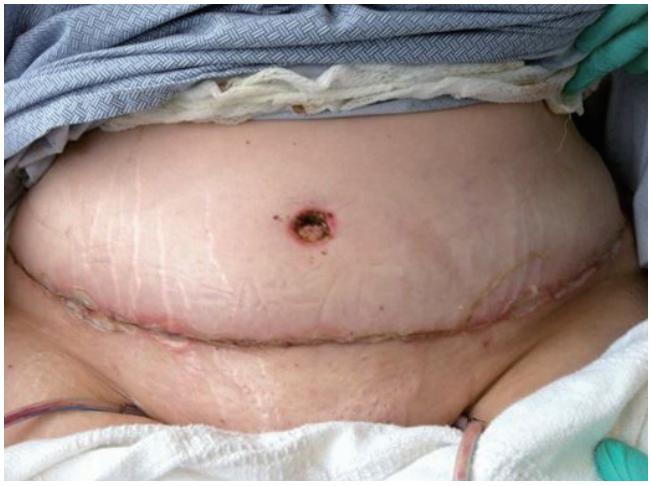

Fig. 3.
Postoperative day 13: smaller areas of bullae that progressed to desquamation and partial-thickness skin loss from the abdominal donor site.

(2) para-inflammatory, (3) paraneoplastic, and (4) pregnancy-associated [1]. Patients with AFND typically present with fever, leukocytosis, subepidermal edema, and red, blue, or violet papules that may coalesce into painful plaques or bullae [1-3].

Diagnosis of AFND must include two major and two minor clinical findings. The major criteria consist of an abrupt onset of erythematous lesions and a neutrophilic infiltration in the dermis without vasculitis. The minor criteria include nonspecific infection, hemoproliferative comorbidities, solid tumors, pregnancy, fever, erythrocyte sedimentation rate (ESR) > 20 mm/hr, elevated C-reactive protein (CRP), neutrophilia, bands > 70%, leukocytosis > 8,000/μL, and response to corticosteroid therapy [4]. Serologic findings of AFND include proteinuria, hematuria, and decreased creatinine clearance [5]. Histopathologic findings show papillary dermis edema without evidence of vasculitis with dense, mature neutrophilic infiltrates within the epidermis, perivascular dermis, or adipose tissue [1]. Thus, the diagnosis of AFND is by exclusion, but infectious etiologies must be ruled out before initiating corticosteroid therapy. The differential diagnosis of AFND should exclude pyoderma gangrenosum, herpes simplex, erythema nodosum, erythema multiforme, and Behçet disease.

Treatment for AFND is targeted towards suppressing neutrophil activity. Corticosteroids have a rapid effect, and prednisone is recommended at a dose of 1 mg/kg/day [1].

A 42-year-old Caucasian female presented to our clinic with a history of right metastatic pleomorphic lobular breast carcinoma. Her past surgical history was significant for thyroidectomy for papillary thyroid carcinoma in 2011, bilateral breast reduction in 2003, knee surgery, and two spinal disc surgeries. In February 2011, she had undergone a right mastectomy, axillary lymph node dissection, and immediate tissue expander reconstruction with acellular dermal matrix, followed by adjuvant radiotherapy. She underwent deep inferior epigastric perforator (DIEP) free flap reconstruction without incident in December 2011.

Her post-operative course was complicated by an acute onset of fever reaching 40°C on postoperative day (POD) two, accompanied by ecchymosis and erythema limited to the mastectomy flaps; the free flap skin paddle remained uninvolved. She was therefore treated for presumed cellulitis with trimethoprim/sulfamethoxazole and cefazolin. Her ESR and CRP were 100 mm/hr and 231 mg/L, respectively; however, the leukocyte count remained normal, and urine, blood, and wound cultures from POD two were all negative. On POD three, the ecchymotic mastectomy flaps developed areas of bullae, but the DIEP flap skin paddle remained viable upon a normal hand-held Doppler exam (Fig. 1). By POD four, the skin lesions progressed to epidermolysis and partial-thickness skin breakdown.

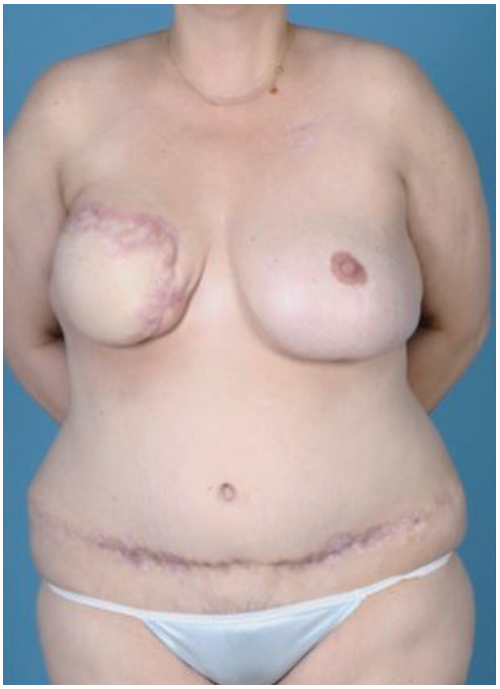

Fig. 4.
Follow-up at 10 months after surgery shows complete resolution of the lesions with mild scarring and discoloration.

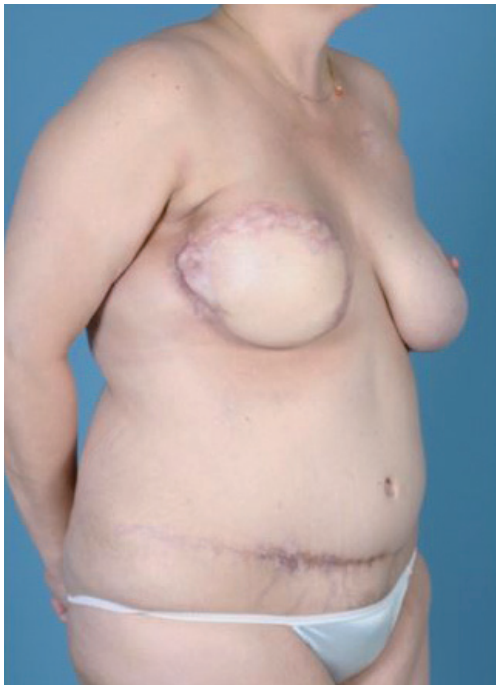

Fig. 5.
Follow-up at 10 months after surgery with only minimal partial-thickness skin loss but no flap volume loss.

She developed leukocytosis to 14.2 K/μL, and the infectious disease service changed antibiotic therapy to vancomycin and meropenem.

On POD five, new lesions were seen at the previously unaffected abdominal donor site. The leukocyte count peaked at 27.1 K/μL with an absolute neutrophil count of 21.8 K/μL, 89% neutrophils, and 23% bandemia. Additionally, ESR and CRP peaked to 135 mm/hr and 420 mg/L, respectively, and the patient developed acute renal insufficiency with a creatinine level of 1.9 mg/dL. In response, the infectious disease service added fluconazole to increase anti-microbial coverage. However, the new lesions, lack of antibiotic response, elevated inflammatory markers, leukocytosis, and multiple negative cultures raised the suspicion of an autoimmune process. Therefore, dermatology sent bedside excisional skin biopsies of the right breast and abdomen for histopathologic examination. Repeat cultures, including mycobacterial and fungal, were negative, and serology studies for autoimmune diseases were also negative (human immunodeficiency virus, hepatitis B/C, antinuclear antibodies, antineutrophil cytoplasmic antibody, antiphospholipid antibodies, rheumatoid factor). Histopathologic findings showed dense neutrophilic infiltrates and papillary dermal edema without signs of vasculitis. A tentative diagnosis of AFND was made on POD five, and the patient was started on a daily dose of 80 mg of prednisone. The leukocyte count decreased to 18.9 K/μL after two doses. Serum creatinine peaked on POD six at 2.4 mg/dL.

On POD seven, the bullous breast lesions began to slough, but the abdominal lesions remained unchanged. The patient had infrequent changes of petroleum-impregnated gauze (Xeroform, Kendall, Mansfield, MA, USA) and non-adherent, absorbent polyethylene terephthalate (Telfa, Covidien, Mansfield) dressings to minimize skin trauma. The patient's renal insufficiency responded to prednisone and resolved without sequelae.

The patient was given a total of eight days of prednisone during which both her breast and abdominal wounds improved (Figs. 2, 3). She was discharged on POD 13 in stable condition with a final white blood cell count of 13.0 K/μL.

At last follow-up 10 months after surgery, her wounds had healed with minimal scarring or discoloration (Figs. 4, 5). The patient is satisfied with her reconstruction and has not elected additional revision surgeries.

AFND is an uncommon autoimmune process that is typically reported in association with hemoproliferative conditions, solid tumors, and granulocyte colony-stimulating factor therapy. In our case, the clinical findings were initially suspicious for cellulitis and mastectomy flap ischemia. However, the

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

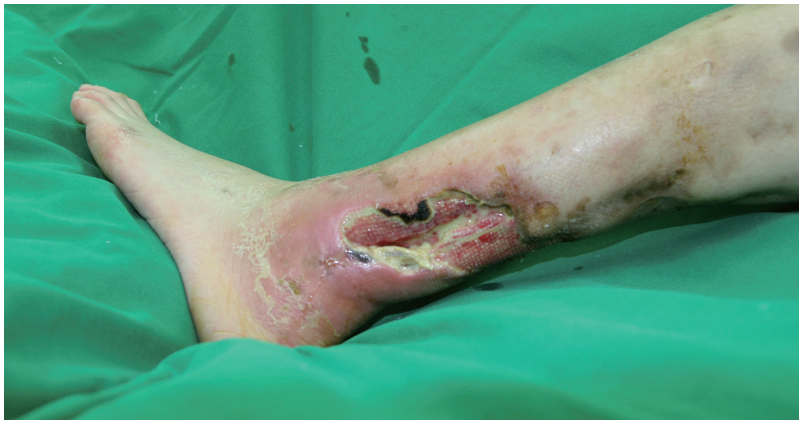

Fig. 1.
Multiple left leg defects. The left leg defects shown here are arose from calcinoses, which occurred in both legs.

Images