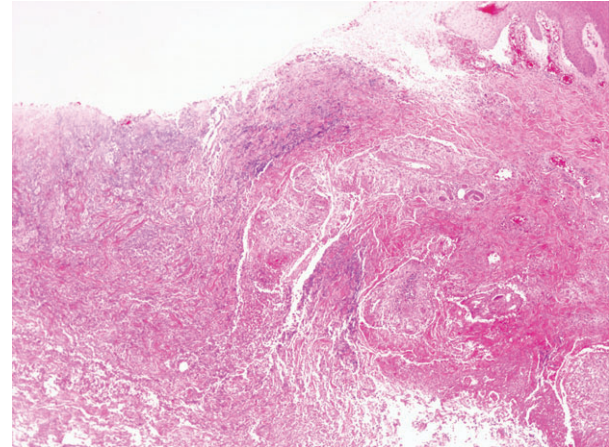


## LETTER TO THE EDITOR

# Necrotising fasciitis by steroid-induced Sweet's syndrome: a case report

Dear Editors,

In November 2011, a 58-year-old woman visited the clinic with complaints of fever, painful swelling and induration with multiple erythematous skin lesions on both her lower extremities (Figure 1). She had a history of rheumatoid arthritis of left fingers and has taken high dose of steroid for 2 years, which could have lead to her weak immune status. We diagnosed necrotising fasciitis based on the symptoms and computed tomography angiography of the lower extremity, and the patient was managed with emergent fasciotomy, surgical debridement, negative pressure wound therapy (NPWT, Vacuum-assisted closure device: Kinetic Concepts, Inc., San Antonio, TX) and antibiotic therapy. Additionally, the patient was diagnosed with Sweet's syndrome by skin biopsy, which was performed at the dermatology department. Microscopic observation of the skin showed severe inflammatory infiltrates with extensive necrosis and neutrophilic microabscess, suggesting necrotising fasciitis (Figure 2). Overall, the features are those of neutrophilic dermatosis and are most consistent with Sweet's syndrome. The presence of eosinophils suggests a drug-related aetiology. Review of prior surgical specimens by the histopathology department demonstrated morphology of Sweet's syndrome (acute necrotising neutrophilic dermatosis). We performed surgical treatment and wound dressing and administered a reduced dose of steroid, with appropriate maintenance. After 2 days of



**Figure 2** Inflammatory neutrophilic infiltrates with extensive necrosis and microabscess.

surgical management and antibiotic therapy, the patient's fevers completely abated, and 1 week later, her lower leg swelling and induration were improved. Skin defect of the left lower leg was covered by split-thickness skin graft (STSG), 0-012 inch thickness, and secondary intension after 2 weeks (Figure 3). At the follow-up, the patient achieved good cosmetic result



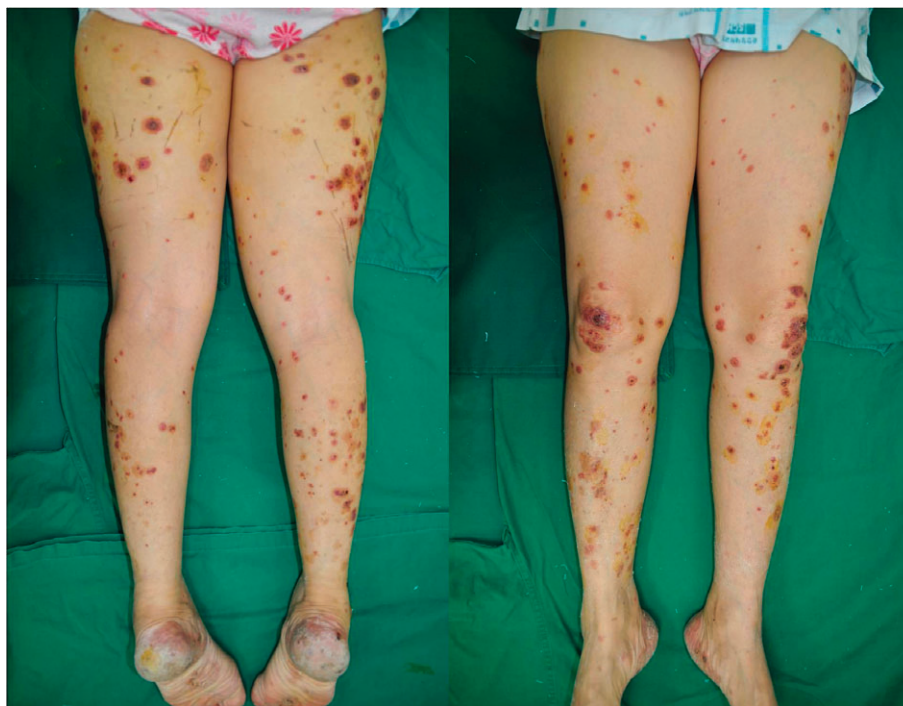
**Figure 1** Multiple erythematous skin lesion with abscess formation on both lower extremities.



**Figure 3** Split-thickness skin graft (STSG) performed on the skin and soft tissue defect on lower leg.

and stable coverage of the skin defects. There were no signs of infection and the symptoms were relieved. The patient was satisfied with the final results (Figure 4).

Sweet's syndrome was originally described by Dr. Robert Douglas Sweet in the August–September 1964 issue of the *British Journal of Dermatology* as an 'acute febrile neutrophilic dermatosis' (1). Sweet's syndrome is characterised by fever, neutrophilia and painful erythematous cutaneous nodules. Based upon aetiological classification, cases of Sweet's syndrome may be subdivided into classical or idiopathic Sweet's syndrome, malignancy-associated Sweet's syndrome that is usually haematologic and drug-induced Sweet's syndrome (1,2). Su and Liu reported the first patient with drug-induced Sweet's syndrome in 1986; the associated medication was trimethoprim-sulfamethoxazole (3). A decade later, criteria for drug-induced Sweet's syndrome were established by Walker and Cohen (4). The most frequently implicated drug is granulocyte colony-stimulating factor (G-CSF) (1). Many other drugs including all-trans retinoic acid, nitrofurantoin, minocycline, hydralazine, furosemide and oral contraceptive pills have also been associated with Sweet's syndrome (5–10). However, several other medications have been observed to promote the development of drug-induced Sweet's syndrome. Recurrence of the dermatosis is often noted when the patient is re-challenged with the associated drug. However, once the causative agent has been discontinued, the disease manifestations frequently improve (8). We reported a case with an initial diagnosis of necrotising fasciitis that was found to represent a new, unusual variant of acute febrile neutrophilic dermatosis induced by steroid therapy. Necrotising fasciitis is a potentially fatal, rapidly spreading, exuberant inflammation caused by bacterial infection involving deep fascia that leads to necrosis of



**Figure 4** There are no induration, swelling, and necrotic skin lesions at the follow-up.

subcutaneous tissues. Symptoms usually begin with cutaneous oedema, tenderness, warmth, erythema and inflammation that rapidly extend horizontally and vertically with potential for development of septicaemia. It is an emergency requiring proper surgical debridement, fasciotomy and antibiotic therapy. There are only few reports about necrotising fasciitis induced by Sweet's syndrome. Most often, it is associated with haematologic and solid organ malignancies, G-CSF infections and pregnancy. Less commonly, Sweet's syndrome has been associated with arthralgia, arthritis, myalgia, conjunctivitis and medications. Most subcutaneous forms of Sweet's syndrome have been associated with haematologic malignancies, which are immunocompromised and demonstrate variable neutrophilic responses and can involve deeper elements of the soft tissue. Generally, Sweet's syndrome rapidly responds to systemic corticosteroid treatment (11). But in our case, the Sweet's syndrome was induced by inhibitory effects on wound healing and the immunocompromising status with long-term use of high-dose steroid. In this case, proper surgical management with controlled administration of appropriate steroid dose can be one of the choices. As this is a rare case, the diagnosis of necrotising Sweet's syndrome should be considered in all cases of suspected necrotising fasciitis, which fails to respond to general therapy and lacks a causative organism.

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