CORRESPONDENCE

Sweet syndrome in a patient with cervical lymphadenitis caused by Mycobacterium abscessus

Dear Editor,

A 64-year-old Chinese man in general good health had a slowly enlarging, painful, hard mass over the left side of his neck, which he had noticed 2 months ago. Chest roentgenogram revealed consolida-
dation over the left lower lung. Computed tomography (CT) showed multiple lymph nodes in the left carotid space, posterior triangle, and supraclavicular fossa. In addition, he had experienced intermit-
tent low-grade fever in recent weeks. He was admitted to our insti-
tute due to a high-grade fever of 39°C lasting for 1 week and multiple painful skin eruptions over the dorsal aspect of both hands, the forehead, chest, and back. On physical examination, there were multiple itchy and painful erythematous-to-violaceous edematous annular plaques with pustulations and some ulcerations (Figure 1A and 1B).

Laboratory investigations revealed a white blood cell count of 37,700/mm³ with 78% polymophonuclear cells, hemoglobin 11.6 g/dL, platelet count 637,000/mm³, C-reactive protein 31.47 mg/dL, and an erythrocyte sedimentation rate of 113 mm in 1 hour. Serum chemistries, serum lactic dehydrogenase, urin-
alysis, and serologic evaluations of thyroid, renal, and hepatic functions were all within normal ranges. No reversed ratio of albumin versus globulin was noted. Abdominal sonography and sero-
logic tests for rapid plasma reagin and human immunode-
nity test. The fever and skin lesions subsided promptly after treatment with 20 mg of prednisolone daily (0.4 mg/kg), therefore, prednisolone was tapered gradually. However, fever with numerous tender indurated erythematous papules, pustules, and nodules recurred on the patient’s face and on four limbs 1 month later. Repeated skin biopsy showed pathological changes that were similar to the previous findings. Thus, 100 mg of dapsone daily was prescribed as monotherapy, and the fever and skin lesions resolved quickly. Four months after ATD, chest roentgenogram revealed no infiltration, and a CT scan showed resolution of the previous enlarged lymph nodes over the left neck. Dapsone at 100 mg/ d was maintained for a further 6 months until the ATD treatment course was completed. No further relapses of cutaneous eruptions occurred.

Sweet syndrome, also known as acute febrile neutrophilic dermatosis, was first described by Dr Robert Douglas Sweet in 1964.1 Three variants of Sweet syndrome were classified: classic, malignancy-associated, and drug-induced. Classic Sweet syndrome is a form of hypersensitivity. It may be associated with pregnancy, inflammatory bowel disease, and infections.2

Nontuberculous mycobacteria (NTM) are distributed in the environ-
ment and can be isolated from water, soil, wild animals, and medical devices. In Taiwan, the most frequently reported patho-
genic NTM are Mycobacterium avium-intracellulare complex, rapidly growing mycobacteria (Mycobacterium chelonae, Mycobac-
terium fortuitum, and M. abscessus), and Mycobacterium kansasi.3,4 M. abscessus can cause a variety of clinical diseases, including skin infection, keratitis, soft-tissue infection, pulmonary infection, and osteomyelitis. It usually follows penetrating trauma in the skin and soft-tissue infection and usually occurs in immunocompetent individuals. In our patient, there was no evidence of immunosuppression even after thorough investigations.5 Only two cases of NTM-related Sweet syndrome in Taiwan have been reported in the literature. Both of them were associated with cervical lymphadenitis caused by M. fortuitum.5,6 To the best of our knowledge, this is the first reported case of M. abscessus-related Sweet syndrome in Taiwan.

Little is known about the pathogenesis of the association be-
tween Sweet syndrome and NTM infection. The underlying patho-
physiological mechanism may be an infection-mediated imbalance of proinflammatory cytokines, such as interleukin (IL)-1, IL-3, IL-8, or granulocyte/macrophage colony stimulating factor, which pro-
 motes the maturation of myeloid cells and induces migration of inflam-
matory cells into the skin.2,7 However, the exact mechanism has not been established.2 Systemic corticosteroid remains the treatment of choice for Sweet syndrome.2,7 Spencer et al8 reported

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a case of histiocytoid Sweet syndrome associated with Crohn disease, which responded well to dapsone therapy.8 Our patient was also treated successfully with dapsone.

Because of the increasing frequency of NTM infections worldwide, we postulate that there will be an increase in such infections concomitant with Sweet syndrome. Physicians should be aware of the clinical features of NTM infection-associated Sweet syndrome.

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References


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