have undergone gastrectomy or splenectomy, and it should not be used at all in patients with glucose-6phosphate dehydrogenase deficit.

Rhizoma coptidis and its main alkaloid component, berberine, inhibit the proinflammatory activity induced by tumor necrosis factor-alpha (TNF- $\alpha$ )<sup>2</sup> in a dose-dependent manner. Recent studies have investigated the antiacne properties of various groups of Chinese herbs-some containing Rhizoma coptidis-showing their antilipogenic and action antibacterial against Propionibacterium acnes.<sup>3</sup> Higaki et al<sup>4</sup> even suggest these are more effective than antibiotics such as minocycline or erythromycin because, unlike these, the Chinese herbs do not produce an increase in minimum inhibitory concentration.

The fact that the patient had previously ingested this extract without reaction suggests a hypersensitivity mechanism. Another hypothesis would be that the excess from the dose ingested could have produced an imbalance between the formation of reactive metabolites and enzymatic hepatic detoxification, leading to an accumulation of reactive oxidants, which acted as haptenes and provoked an immune response.

In conclusion, we wish to stress the power of Chinese herbs to produce both therapeutic benefit and side effects, including dermatological ones, whilst pointing out that the extensive use of such treatments in the growing Chinese population implies greater consideration must be given to the possibility of their ingestion in cases of toxicoderma.

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# Sweet Syndrome as a Possible Initial Manifestation of Human Immunodeficiency Virus Infection

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## To the Editor:

Acute febrile neutrophilic dermatosis was initially described by Sweet in 1964<sup>1</sup> as reactive dermatoses in middle-aged women following upper respiratory tract infections. These dermatosis had 4 characteristic clinical elements: fever; leukocytosis; eruption of erythematous and edematous plaques on the extremities, face, and neck; and a dense predominantly neutrophilic inflammatory infiltrate in the dermis, with no sign of vasculitis. The reactive nature of this condition is noted for its frequent association with infectious, inflammatory, or neoplastic processes. We present a new case of Sweet syndrome as the initial manifestation of infection with the human immunodeficiency virus (HIV).

A 35-year-old male, with no relevant medical history, was examined for fluidfilled erythematous and edematous lesions that had presented 4 days earlier. These were painful to pressure and were found on the upper lip (Figure 1), outer



Figure 1. Violaceous erythematous and edematous plaques painful to pressure, on the upper lip.

ears, scalp, knees and elbows (Figure 2), and finger pads, with no associated fever or malaise. The patient had no previous catarrhal symptoms, and had taken no medication in the previous month. Test results showed a white



Figure 2. Violaceous erythematous and edematous plaque on the left elbow.

blood cell count of 8200 cells/mL, 70% neutrophils, and left shift (7% band forms). Globular sedimentation rate was 25 mm/h and the chest x-ray revealed no significant abnormalities. Histology of a biopsy sample taken from a lesion on the forearm showed the presence of a subepidermal blister with intense neutrophilic inflammatory infiltrate, but no signs of leukocytoclastic vasculitis. Serology was negative for syphilis, herpes simplex, and hepatitis B and C, but positive for HIV-1 in an enzyme-linked immunosorbent assay. This finding was confirmed in a second test. The skin lesions improved after initiating treatment with a tapering course of oral prednisone (beginning at 50 mg/d) for 6 weeks, with no subsequent relapse. The patient was referred to the infectious diseases department of the hospital, with initial analysis showing a low CD4+ T-cell count (285 cells/mL) and a viral load of 100 000 copies/mL.

The association of Sweet syndrome with HIV infection has rarely been described in the literature,<sup>2-7</sup> and only twice has Sweet syndrome been reported as the first manifestation of HIV infection.<sup>2,5</sup> The CD4<sup>+</sup> T-cell count in the cases described varied between 368 cells/mL,<sup>5</sup> and less than 50 cells/mL,<sup>3</sup> suggesting that immunological status is not the only factor involved in the pathogenesis of the process. Some authors have suggested that the immunological changes induced by HIV could play an important role in triggering dermatoses, through the formation of immunocomplexes, with activation of polymorphonuclear neutrophils.<sup>2</sup> Also, certain HIV proteins, such as transactivating protein, have been reported to be an important factor for inducing neutrophil chemotaxis.<sup>7</sup>

Other suggested pathogenic factors include photosensitivity,<sup>3</sup> reactions to antiretroviral therapies,<sup>6</sup> or those related to phenomena of sudden immune restoration in patients in whom antiretroviral therapy has been recently initiated.<sup>4</sup> Cofactors may have played a role in some of the cases reported, for example the treatment of drug-induced aplasia in HIV-positive patients with granulocyte colony-stimulating factor (G-CSF).<sup>3</sup>

The presence of blister-like lesions in Sweet Syndrome is described relatively frequently<sup>8</sup> as the clinical outcome of intense edema and inflammatory infiltrate in the dermis, which leads to subepidermal detachment.

In this case, factors associated with Sweet syndrome other than infection by HIV were not observed, given that the patient reported none of the traditional indicators (catarrh, new medication). Even if the exact mechanism of the association is uncertain, we must stress the significance of considering infection with HIV in patients with Sweet syndrome, especially in young patients exhibiting associated risk behaviors.

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# Benign Lymphangiomatous Papules and Plaques After Radiotherapy

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#### To the Editor:

Lymphangiomas are tumors that normally appear at birth. They are formed from dilated lymph vessels that may extend to the subcutaneous cellular tissue. A number of causes of acquired lymphangiomas such as radiotherapy and surgery have been reported.

The area irradiated during radiotherapy may develop benign vascular

proliferations such as acquired progressive lymphangioma or malignant processes such as high-grade angiosarcoma, even when low doses of radiation are used.<sup>1,2</sup> Within what are considered acquired