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Eczema Herpeticum in Cutaneous T-Cell Lymphomas

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To the Editor:

Disseminated skin infection due to herpes simplex virus, also known as eczema herpeticum or Kaposi varicelliform eruption (cutaneous dissemination of herpes simplex infection in patients with generalized skin disease), has been reported in numerous dermatoses, the most frequent being atopic dermatitis, followed by other dermatoses such as Darier disease, ichthyosis, and bullous diseases. Few cases of disseminated herpes have been reported in cutaneous T-cell lymphomas (CTCL).

We present the case of a 61-year-old man diagnosed with stage IVB mycosis-fungoides-type CTCL (T4N3M1B2) in 2006, with erythroderma, widespread lymph node involvement, and a count of circulating cells with abnormal $CD3^+$ $CD4^+$ $CD7^+$ phenotype of 702/μL (78% of the total count); the absolute lymphocyte count was 800/μL, of which 720 were $CD4^+$. During his illness, the patient was treated with phototherapy, methotrexate, prednisone, bexarotene, interferon, and a histone deacetylase inhibitor.[1] In January, 2008, whilst on treatment with monthly liposomal doxorubicin, he presented an exacerbation of the lesions, with more marked skin infiltration and pruritus, and developed superficial crusted lesions on the dorsum of the nose that spread to the rest of the skin (Figures 1-3); there were associated vesicles and pustules, and he had a fever of 39°C. A diagnosis of disseminated eczema herpeticum was made, with a positive culture for herpes simplex virus type 1 from a pustule and positive blood cultures for *Staphylococcus aureus* sensitive to cloxacillin. Treatment was started with intravenous acyclovir 10 mg/kg/d, vancomycin 1 g/12 h, and meropenem 1 g/8 h, and washing with 1:1000 zinc sulfate solution 3 times a day. The lesions disappeared practically completely from the face within 10 days; at discharge, a few lesions remained on the palms of the hands. There was a parallel improvement in the general state and in the fever.

In 1978, Segal and Watson[2] described a patient with mycosis fungoides who developed a vesicular rash during treatment with psoralen-ultraviolet (UV) A; the rash extended to cover the whole skin and was associated with fever. A further 2 cases were published by Brion et al[3] in 1981; these patients also developed the infection during an advanced stage of the lymphoma while receiving treatment with corticosteroids and cyclophosphamide and, in another case, with leukapheresis. Later, Hayashi

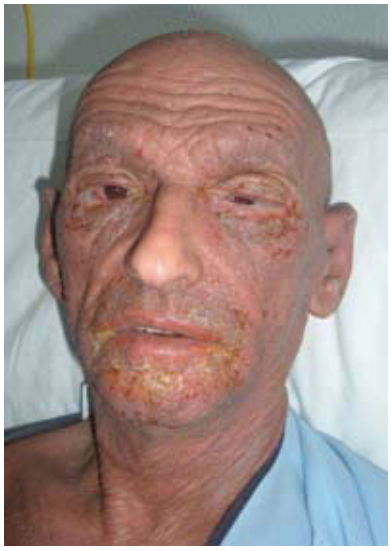

Figure 1. Crusted, erosive lesions affecting the whole face.

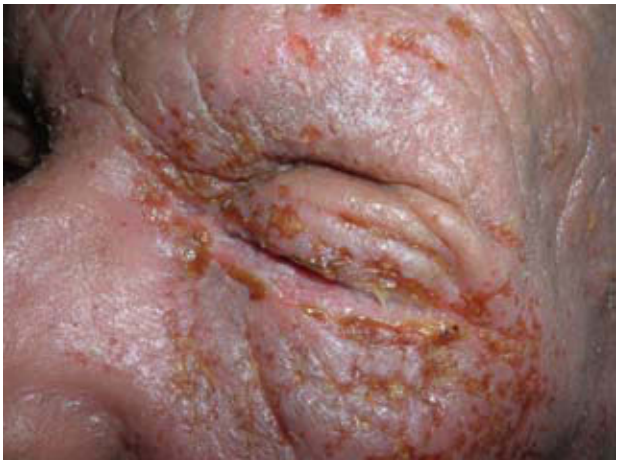

Figure 2. Eye lesions showing erosions and purulent secretions on the eyelids.

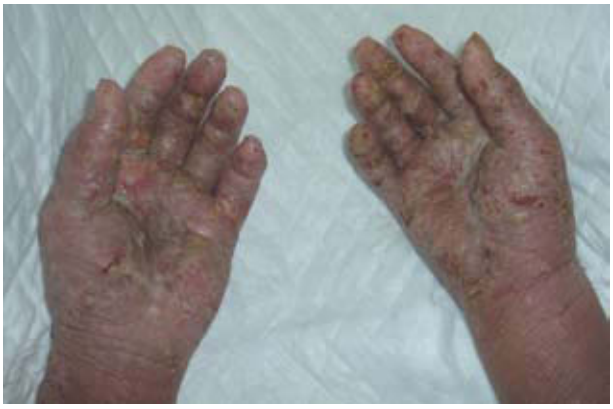

Figure 3. Lesions on the palms of the hands. There are a large number of pustules with the formation of pustular lakes.

et al,[4] Masessa et al,[5] and Taulbee and Johnson[6] presented isolated cases of disseminated herpes in patients with CTCL. Axelrod et al[7] published a series of 166 patients with mycosis fungoides; 64 of these patients developed viral infections, 9 of which were disseminated herpetic infections in patients with advanced CTCL. Epstein[8] presented another series of 144 patients with CTCL with 4 cases of viral infection, though none of these was due to disseminated herpes simplex infection.

None of the patients with disseminated herpes virus infection died in any of the series reviewed. However, it cannot be said that disseminated herpetic skin infection is a mild infection, rather it is just the opposite, considered to be a serious and potentially life-threatening condition in which early intravenous treatment associated with antibacterial prophylaxis is essential as this can be a serious complication.

In our case, treatment was administered with intravenous acyclovir (10 mg/kg/d) and concomitant treatment to prevent bacterial superinfection, which is widely reported as the principal cause of infection and death in this type of patient.[9,10]

In the cases reported by Segal and Watson[2] and by Brion et al,[3] it was suggested that the disseminated herpesvirus infection could have developed because the patients' immune status was compromised by the treatments they were receiving. However, in our case, apart from the chemotherapy, the disease itself could have been the cause as, in the most recent blood cell count, cellular immunity was reduced as almost the whole $CD4^+$ T-cell count had a morphologically incompetent phenotype.

It is noteworthy that none of the cases mentioned had presented cutaneous herpes previously and that the first clinical manifestation of the herpetic viral disease was the appearance of disseminated skin involvement. Herpesvirus type 1 was the causative organism in all cases reported.

A number of predisposing factors for the onset of both viral and bacterial infections were described in the principal series in the literature,[3-5] and these were present in our patient: advanced stage of the disease (T4N3M1B2, stage IVB), presence of erythroderma, marked deterioration of immunity (720 lymphocytes per microliter, of which 702 showed phenotypic abnormalities), and chemotherapy treatment (liposomal doxorubicin) in addition to the previous treatments.

In a comparative study of the infectious complications of various tumors, the cause of death was not found to differ between mycosis fungoides and other lymphomas.[8] In all these diseases, the primary cause of death was infection, mainly bacterial sepsis. Death due to disseminated herpetic infection did not occur in any patient in the study.

In the cutaneous lymphoma database in our hospital, we have 320 cases of mycosis fungoides and Sezary syndrome but this is the first patient to develop disseminated eczema due to herpes simplex virus.

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Conflicts of Interest
The authors declare no conflicts of interest.

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Verrucous Carcinoma of the Foot Associated with Human Papillomavirus Type 18

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To the Editor:

Verrucous carcinoma (VC) is an uncommon and well-differentiated variant of squamous cell carcinoma of the skin and mucosas. The clinical and pathological concept was first described by L.V. Ackerman in 1948.[1]

In the past, VC has been called according to its anatomical location.[2] When found on the oral mucosa, the term florid oral papillomatosis is used whereas, in the anogenital region, it receives the name giant condyloma of Buschke and Löwenstein. VC of the sole of the foot, the most common site, is known as epithelioma cuniculatum. Clinically it resembles a plantar wart, and has an endophytic appearance, with multiple orifices on its surface that correspond histologically to crypts full of keratin and interconnected tunnels. This gives it a certain similarity to a rabbit warren, justifying the use of the term cuniculatum (in Latin, cuniculus means rabbit or tunnel). Less commonly, VC may be situated on other areas of the skin and is then known simply as cutaneous VC.

Although the etiology of VC has not been fully explained, a number of factors have been implicated in its development, including infection by human papillomavirus (HPV).[3,4]

The patient was a 61-year-old white man with no past history of interest, who was seen for a progressively enlarging lesion on the sole of the left foot; it had been present approximately 1 year. Previously, the lesion had been diagnosed as a plantar wart and was treated with a number of cycles of cryotherapy, with no improvement.

Physical examination revealed a hyperkeratotic plaque of 2 by 2 cm, of verrucous appearance, with well-defined margins, and a macerated central tissue with orifices full of purulent material (Figure 1).

There were a number of possibilities in the differential diagnosis, including squamous cell carcinoma, superinfected plantar wart, and amelanotic melanoma.

Biopsy of the lesion revealed a bulbous proliferation of keratinocytes forming nests of different shapes and sizes in the superficial half of the dermis. Some of these nests presented central keratin pearls (Figure 2).

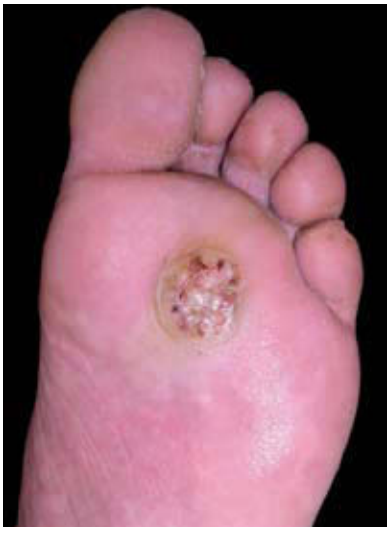

Figure 1. Plaque with a verrucous appearance.