Eosinophilic Folliculitis in 2 HIV-Positive Women

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Background: Human immunodeficiency virus–associated eosinophilic folliculitis (HIV-EF) among homosexual men is a commonly reported dermatologic finding, while only 4 cases in HIV-positive women have been documented in the literature to date. This article describes 2 additional cases of HIV-EF in immunocompromised women and reviews the data on this condition.

Observations: The diagnoses were made on the basis of clinical appearance and microscopic analysis of skin biopsies. The women were not receiving highly active antiretroviral therapy (HAART) and their CD4 cell counts were below 100/µL.

Conclusions: As HIV prevalence continues to increase in the female population, more cases of HIV-EF will be seen among women. Because the etiology of HIV-EF remains elusive, no single treatment stands above the rest although several successful therapies have been demonstrated. However, HAART restores the proper T-cell milieu, which seems to improve the course of this disease.

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HUMAN IMMUNODEFICIENCY VIRUS–ASSOCIATED EOSINOPHILIC FOLLICULITIS (HIV-EF) Is a Chronic Papulovesicular Disease Seen in Late Stages of HIV Infection as CD4 cell counts drop below 300/µL. Nearly all cases have been reported in homosexual men,1 and the first case in a woman was described in 1996.2 Since this initial report, only 3 other cases of women with HIV-EF have been documented in the literature.3-5 We report 2 additional cases in women who presented 1 month apart to our dermatology service.

REPORT OF CASES

CASE 1

A 43-year-old Hispanic woman with HIV infection presented with a 6-month history of an intensely itchy eruption involving the entire face. Her primary care physician had prescribed topical steroids and antifungal creams, which reduced the pruritus but had no effect on the skin lesions. Her medical history was significant only for HIV disease, and she had taken no antiviral medications for several years because of noncompliance.

Cutaneous examination revealed numerous large eriythematous, skin-colored, monomorphic dome-shaped papules over the face (Figure 1), posterior auricular area, and neck. On her anterior chest was a 3-cm eriythematous, urticarial-appearing plaque with a 4-mm central erosion. The oral mucosa, palms, and soles appeared normal. The patient stated that a skin biopsy had been performed 2 months previously at another hospital and that her skin disorder had been diagnosed as an acute allergic reaction. Recent laboratory work revealed a hemoglobin concentration of 9.7 g/dL, a white blood cell count of 4.9 × 10³/µL with 62% neutrophils, 22% lymphocytes, 7% monocytes, and 9% eosinophils, and a CD4 cell count of 35/µL.

The patient underwent a second biopsy. Microscopic examination demonstrated no epidermal changes. In the dermis there was a mild superficial and deep perivascular lymphocytic infiltrate, along with a predominant perifollicular and intrafollicular mixed inflammatory cell infiltrate showing numerous intact and degranulated eosinophils (Figure 2 and Figure 3). Periodic acid–Schiff staining was negative for fungal organisms.

CASE 2

A 41-year-old HIV-positive African American woman was seen in consultation for a 1-year history of pruritic papules on both
collections. She had previously been treated successfully for seborrheic dermatitis of the scalp with therapeutic shampoo (T-Gel; Neutrogena Corp, Los Angeles, Calif). Her medical history was significant for intravenous drug use, hepatitis B and C, and a 14-year history of HIV infection. The patient’s antiviral regimen had undergone frequent changes owing to multiple adverse reactions to her medications, and at the time of consultation she was not receiving highly active antiretroviral therapy (HAART).

Cutaneous examination demonstrated several 1- to 2-mm skin-colored, erythematous, well-defined papules on the malar region and dorsal nose. On the upper arms bilaterally were scattered excoriated skin-colored papules without crusting or pustules. Laboratory results were as follows: hemoglobin, 12.4 g/dL; white blood cell count, 2400/µL with 40% neutrophils, 37% lymphocytes, 14% monocytes, and 7% eosinophils; and a CD4 cell count of 62/µL.

Microscopic examination of a skin biopsy specimen from the cheek demonstrated a flattened epidermis with mild papillary dermal edema. There was a mixed infiltrate of lymphocytes with scattered eosinophils in the dermal perivascular and perifollicular regions. In addition, focal intrafollicular eosinophils were noted. Periodic acid-Schiff staining was negative for fungal organisms.

**COMMENT**

The most frequently seen HIV-associated folliculitis, HIV-EF, classically presents as an erythematous follicular papular eruption with frequent excoriations. Urticarial lesions and erythematous plaques with central clearing, as seen in 1 of our patients, have also been reported. The trunk is the most frequently affected site, and the head, neck, and proximal extremities are the next most commonly affected areas. Peripheral eosinophilia, elevated serum IgE levels, and CD4 cell counts almost uniformly under 300/µL have been repeatedly documented at the time of diagnosis. Both of our patients had mild eosinophilia and CD4 cell counts less than 100/µL.

First reported in 1986 by Soeprono and Schinella, HIV-EF was given its name to distinguish it from Ofuji disease (eosinophilic pustular folliculitis). Although these 2 dermatoses can overlap in presentation and distribution, there are several clinical and laboratory features that distinguish them. In HIV-EF, patients typically present with unremitting pruritus, lack palm and sole involvement, and exhibit leukopenia correlating with their acquired immunodeficiency syndrome. In contrast, fewer than 50% of patients with Ofuji disease note persistent pruritus, 20% have palm or sole involvement, and most have a leukocytosis.

The etiology of HIV-EF is currently unknown. Bacterial, fungal, and viral culture results are generally nega-
tive. Several antimicrobial agents, however, have demonstrated success in treating HIV-EF; they include metronidazole,\textsuperscript{10} itraconazole,\textsuperscript{11} and permethrin.\textsuperscript{12} This success has led to postulations that HIV-EF is caused by a pathogen, eg, \textit{Pityrosporum}\textsuperscript{13} or \textit{Demodex} mites.\textsuperscript{14} In contrast, the success of isotretinoin in treating some cases has led to the hypothesis that HIV-EF could represent an eosinophilic immune dysregulation directed at a lipid-soluble factor in the sebum.\textsuperscript{6} Other successful treatments have included UV-B, psoralen–UV-A,\textsuperscript{15} and cetrizine, an antihistamine with anti-eosinophilic properties.\textsuperscript{16}

As advanced HIV infection depletes T cells, the immune system shifts to a \(T_{H2}\) cell–dominant environment\textsuperscript{17} that sets the stage for opportunistic pathogens to cause cutaneous diseases like HIV-EF. Since HAART has become the standard of care, there has been a changing paradigm in the cutaneous manifestations of HIV.\textsuperscript{17} For example, \(T_{H2}\)-cell predominant conditions such as Kaposis sarcoma and bacillary angiomatosis disappear as CD4 cell counts rise.\textsuperscript{18} The notion is that HAART restores the \(T_{H1}/T_{H2}\) cell balance, which allows the immune system to overcome these skin conditions. On the other hand, mucocutaneous herpes simplex and zoster, which are dominated by \(T_{H1}\)-cell responses, often erupt in patients beginning HAART for immune restoration.\textsuperscript{18} There is a paucity of records documenting the effect of HAART on HIV-EF, although there are anecdotal reports of immune improvement. After 6 months of HAART the skin lesions of 1 of our patients were much improved although she had also been specifically treated for her HIV-EF with itraconazole. The other patient had not restarted HAART at follow-up.

Microscopically, HIV-EF demonstrates a perifollicular and perivascular mixed cell infiltrate rich in eosinophils, with spongiosis of the follicular epithelium and sebaceous glands.\textsuperscript{19} In contrast, suppressive folliculitis typically demonstrates microorganisms surrounded by neutrophils and macrophages, along with frequent follicular rupture. In a study of 52 skin biopsy specimens, McCalmont et al\textsuperscript{20} were able to distinguish HIV-EF from other folliculitides based on the above histopathologic findings. Likewise, our 2 patients with HIV-EF presented with the classic picture of a papular follicular eruption and an eosinophil-rich infiltrate by light microscopy.

Most of the documented cases of HIV-EF have been in homosexual men in their third to seventh decade of life. A search of the literature revealed only 4 reports of HIV-EF in women.\textsuperscript{2,3} The first 2 cases were in a 40-year-old Portuguese woman and a 30-year-old Ethiopian woman in whom HIV-EF was the presenting manifestation of acquired immunodeficiency syndrome.\textsuperscript{2,3} The third case was in a 39-year-old African American woman with a very low CD4 cell count (20/\(\mu\)L) as well as numerous other dermatologic conditions including herpes zoster and oral hairy leukoplakia.\textsuperscript{4} The final case was that of a 43-year-old HIV-positive Chinese woman with a 2-week history of pruritic facial eruption diagnosed as HIV-EF.\textsuperscript{3}

The 2 cases of HIV-EF in HIV-positive women that we present here bring the total number of reported cases to 6 and underscore the fact that HIV-EF is no longer a disease found solely in homosexual men with AIDS. Also, the scarcity of documented cases of HIV-EF in women highlights the need to increase reporting of diseases that affect women in general, and HIV-positive women in particular. Physicians, especially those who provide primary care for HIV-positive women, need to be aware of this condition because it is often overlooked, is very difficult to treat, and is a continual source of anxiety and frustration for those who have it.

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REFERENCES