

Chronic Recurrent Folliculitis with Atypical Hyperpigmented Scarring in an AIDS Patient

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Bacterial, fungal, and viral infections of the skin with extended skin involvement can occur during the early phase of human immunodeficiency virus infection. A significant reduction in circulating CD4+ lymphocytes in the late stage of the disease may cause tumors of the skin such as Kaposi's sarcoma.

A 40-year male patient, a former sailor who had multiple sexual contact with native African women, presented with multiple tender follicular pustules and fibrotic brown patches on both his legs. These had been present for 6 months. The skin lesions were healed leaving brown pigmentation. Laboratory examinations revealed the presence of leukopenia, thrombocytopenia and a reversed T4/T8 ratio. The ELISA and Western blot analysis to human immunodeficiency virus were positive. A skin biopsy from a brown patch showed early stages of scar tissue and perivascular hemosiderin deposition.

We herein report a case of acquired immunodeficiency syndrome patient with atypical dark brown scarring atrophic patches on the lower legs following purulent bacterial folliculitis. This may have been an early manifestation of Kaposi's sarcoma from a preceding skin lesion.

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Key Words : Acquired immunodeficiency syndrome, Folliculitis, Kaposi's sarcoma

INTRODUCTION

The human immunodeficiency viruses (HIV), which belong to the lentivirus subfamily of the retroviruses, cause selective depletion of cells bearing CD4 receptors, and the end result of immunocompromise is recognized as acquired immunodeficiency syndrome (AIDS). The HIV infection causes a profound defect in cell-mediated immunity, which

cause complicating opportunistic infection and neoplastic processes.¹

Numerous cutaneous diseases have been reported to be associated with HIV infection including candidiasis, seborrheic dermatitis, chronic infection caused by herpes simplex, Kaposi's sarcoma (KS), as well as a variety of less common disorders.² So the role of dermatologists in the detection and management of AIDS has been emphasized.

We herein report a case of chronic recurrent purulent bacterial folliculitis leaving atypical dark brown scarring patches in an HIV-infected patient.

CASE REPORT

A 40-year-old Korean male was referred for the evaluation of multiple tender erythematous follicular pustules on his legs. He had suffered from re-

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Fig. 1. Tender follicular pustules and brown pigmented patches on the leg.

Fig. 2. Close-up view of brown atrophic, fibrotic scars.

current multiple tender follicular pustules (Fig. 1) for 6 months. The skin lesions were healed leaving brown hard fibrosing atrophic pigmented scars (Fig. 2).

On physical examination, he was chronically ill and febrile. There were pruritic greasy erythematous patches on his face, scalp and torso. Acneiform eruption with pitting scars on his face and torso, and diffuse scalp alopecia was also noted. Retroauricular and posteroir cervical lymph nodes were palpable.

His past history revealed that he had had multiple

heterosexual contact with native African women 17 years ago while he was a sailor. Herpes zoster along his left chest wall had developed 7 years before his visit. He had an operation to fix a fractured maxillary bone due to a traffic accident one year prior to his visit at the other hospital.

Laboratory examinations revealed mild leukopenia and thrombocytopenia. Many colonies of methicillin-resistant *Staphylococcus aureus* and *Streptococcus pyogenes* were cultured from pus. The ELISA to HIV showed strong seropositive reaction, and a subsequent Western blot reacted to p160 protein of HIV. The T cell count was reversed for the T4/T8 ratio. His wife also showed seropositivity to HIV.

A skin biopsy taken from a brown patch showed the early stage of scar formation. There were increased fibroblasts with collagen bundles, capillaries with hemorrhage in the upper dermis and perivascular hemosiderin deposition from the mid-dermis to subcutis (Fig. 3). Deposition of fibrin in large

Fig. 3. Increased number of fibroblasts with collagen bundles, and increased capillaries with hemorrhage in upper the dermis simulating scar tissue (H&E, $\times 160$).

blood vessels and perivascular lympho-plasma cell deposition was also noted.

The folliculitis was treated with systemic vancomycin, teicoplanin and topical mupirocin ointment with a favorable response. Two weeks after his initial visit, herpes zoster developed along the left C5-7 dermatome, and intravenous injections of acyclovir were started. A few days later, generalized erythematous pruritic maculopapular eruptions developed in the whole body. Meanwhile dyspnea,

diarrhea, mild fever, oral candidial thrush also occurred. After three days of rehospitalization, he died of adult respiratory distress syndrome accompanying with pneumonia.

DISCUSSION

The first report of AIDS in Korea came from a foreigner in 1985.³ Since that case, HIV infected and AIDS patients have been increasing steadily through various routes of infection in this country. Authorities have reported that the total number of HIV infection or AIDS cases at the end of 1997 was 747 of which 145 had died. However epidemiologists estimate that there are at least twice this number registered HIV infected people in this country.

Our patient was unaware that his immune system was compromised until he came to us, so he had not given the chance to take drugs, such as zidobudine, which suppress viral replication. Meanwhile his wife became infected through sexual contact, and he had an operation following a traffic accident, which put medical staff at risk. Bizarre cutaneous manifestations in our patient aroused suspicion of a serious illness at his first visit, and detailed past history and careful physical examination prompted us to check for HIV infection.

We observed some cutaneous manifestations in our patient during a relatively short chronological course of illness. They include purulent bacterial folliculitis, seborrheic dermatitis, acneiform eruptions, diffuse alopecia, pruritic papular eruptions, and recurrent herpes zoster.

Seborrheic dermatitis has been reported to occur with great frequency ranging from 30% to 80% in prevalencies and severity in patients with HIV infection.⁴ Persisting acneiform eruption with pitting scars, diffuse scalp alopecia and recurrent purulent folliculitis in our patient belong to a broad spectrum of seborrheic dermatitis. A recent report⁵ documented that there is a correlation between the level of immunosuppression and the incidence of skin disorders in patients with HIV infection. Seborrheic dermatitis belongs to a category of advanced degree of immunosuppression with CD4 counts below 75/mm³.

Pruritic papular eruptions of AIDS is characterized by generalized pruritic papules and nodules, and is characterized histologically by superficial and mid

dermal perivascular and perifollicular mononuclear cell infiltration.⁶ We observed pruritic papular eruptions which mimic viral exanthem or morbilliform drug eruption at the terminal stage of our patient.

With regards to folliculitis, the eosinophilic pustular folliculitis (Ofuji's disease,) is common in HIV-infected patients.⁷ While eosinophilic pustular folliculitis in AIDS is characterized by culture negativity, unresponsiveness to systemic antibiotic therapy, peripheral leukocytosis and eosinophilia, neutrophilic and eosinophilic infiltration in hair follicles, mainly on the upper trunk, the bacterial purulent folliculitis usually shows staphylococcal and/or streptococcal growth from pus, which responsive to topical and/or systemic antibacterial agents.

The development of Kaposi's sarcoma among the AIDS patients is quite different. The lesions can linear cutaneous arrangements⁸ or masquerade as folliculitis, nevus flammeus, or simply hyperpigmented areas.⁹ In the reported case of AIDS by Won et al.¹⁰ the patient had erythematous or pigmented pruritic nodules on the extremities suggesting KS. However they could not find any evidence of KS in repeated skin biopsies. We also presumed that the unusual clinical features and histopathological findings of our patient may have simulated an early development of KS from preceding skin lesions as reported in a similar account by Rendon et al.⁹ Unfortunately our patient died before the full development of dermatological manifestations of AIDS.

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