

Skin in HIV

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Photosensitive Disorders in HIV

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Cutaneous photosensitivity is an inflammatory skin reaction caused by an abnormal response to non-ionizing radiation and has been associated with HIV disease [1]. The clinical presentation of patients with photosensitivity may vary significantly. In general, manifestations are more prevalent on exposed skin and tend to be more severe in the spring and summer [2].

EPIDEMIOLOGY

Photosensitivity is an increasingly recognized disorder in HIV-infected individuals. It is believed that as the immunodeficiency worsens, photosensitivity reactions become chronic and increase in severity [3]. HIV photodermatitis encompasses a variety of clinical manifestations. It occurs in approximately five percent of HIV-seropositive patients [4]. Photosensitivity may be the initial manifestation of HIV disease [5]. Photosensitivity typically occurs in patients who have a CD4 T lymphocyte count ≤ 50 cells/ μl and worsens as the immunodeficiency progresses [4,6,7]. The propensity to develop HIV photodermatitis varies by race. In one study, African-American patients with HIV were 6.68 times more likely to develop photodermatitis than all other races combined [4,8]. Native Americans are also thought to be at higher risk to develop the disease [7].

CLINICAL MANIFESTATIONS

HIV photodermatitis encompasses a variety of clinical manifestations, including polymorphic light eruption, actinic prurigo, chronic actinic dermatitis (CAD), porphyria cutanea tarda, photosensitive granuloma annulare, lichenoid photoeruption, widespread vitiligo-like depigmentation and photodistributed hyperpigmentation [6,8,9]. Of these, CAD and lichenoid photoeruption are the most common associations [9]. Sun-exposed areas, such as the face, ears, scalp, posterior neck, upper back, “V” area of the chest, extensor arms, and dorsal hands, are involved [4,8]. [Figure 6.1]



Figure 6.1: Chronic actinic dermatitis.

(Photocourtesy: Department of Dermatology, Bowring and Lady Curzon Hospital, Bangalore)

Photosensitivity in HIV-infected individuals can manifest as two distinct morphologic groups: lichenoid and non-lichenoid [4]. Non-lichenoid cases seem to fall within a spectrum of eczematous disease, with varying evolution through acute/subacute eczematous, chronic eczematous, and hyperpigmented dependent on immune status and UV exposure. It is also been postulated that, patients develop photo-induced acute/subacute eczema early in the course of their HIV infection, whereas chronic eczematous lesions evolve as HIV infection progresses and CD4 + counts are still low [4]. Finally, hyperpigmented lesions may appear with recovery of the immune system or from prolonged subclinical UV light-induced inflammation, indicated by the lower viral load, higher CD4+ count, and longer interval between both CD4 nadir and viral load zenith to visit. Potentially, patients may also develop hyperpigmented lesions after an acute eczematous eruption, similar to postinflammatory hyperpigmentation, although a burnt out dermatitis of lichenoid origin can also be the cause [4].

A dose-response association with increasing UV light exposure and photosensitivity in HIV-infected individuals has been observed. Exposure to more intense UV light may therefore

be necessary to initiate the inflammatory process presenting clinically as an acute eczematous eruption. Less intense exposure would suffice to maintain the reaction and the evolution of the other morphologies [4].

Chronic actinic dermatitis (CAD) may be an early presenting feature of advanced HIV infection, preceding the onset of clinical AIDS. Another feature common to CAD patients found to have HIV infection is the earlier age of onset of CAD compared with non-HIV-associated CAD [10]. HIV infection may play a role in hastening the onset of CAD in predisposed individuals, or may be an aetiological factor in its pathogenesis. Pappert et al. hypothesized that the immune response in CAD is regulated by CD8 cells reacting to some photoinduced self-antigen, with loss of control of antiself response due to decreased CD4: CD8 ratio in HIV, and a selective defect in non-HIV CAD [1].

Certain medications, such as trimethoprim-sulfamethoxazole, NSAIDs, and HAART, can be photosensitizing and may increase the risk of photodermatitis, but the skin eruption often does not improve even when the medication is discontinued [5,8]. HAART medications, such as protease inhibitor, nucleoside and non-nucleoside reverse transcriptase inhibitor classes, have a high porphyrinogenic risk, so patients taking these medications should be monitored [11].

HISTOPATHOLOGY

Histology varies based on clinical presentation, but may reveal subacute or chronic dermatitis and abundant eosinophils. Histology identical to PLE, lichen planus or lichen nitidus may also occur [5,8]. HIV photodermatitis mimicking widespread vitiligo-like depigmentation vary from vitiligo by the presence of spongiotic dermatitis with eosinophils [8].

TREATMENT

Treatment is often difficult. Initially, sun protection and topical steroids can be used. Treatment with thalidomide has been successful in refractory cases [5,6,8]. It is important to note that thalidomide has been reported to increase the HIV viral load, although the clinical significance of this phenomenon is unknown [8]. Therefore, the HIV RNA level should be monitored after the 1st and 3rd months of thalidomide treatment and subsequently every 3 months [12].

CONCLUSION

In conclusion, a plethora of clinical manifestations are associated with HIV photodermatitis, which should be considered when a patient with HIV/AIDS presents with a pruritic, photodistributed eruption.

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