

Human Immunodeficiency Virus Infection Presenting with Chronic Actinic Dermatitis: A Case Report

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Abstract

Introduction: We describe a case illustrating a rare but increasingly recognised association of chronic actinic dermatitis (CAD) with human immunodeficiency virus (HIV) infection. **Clinical Picture:** A 40-year-old Chinese man of skin type III presented with a photodistributed rash of 3 years' duration. Histology, phototesting and subsequent clinical course were consistent with CAD. HIV serology was positive. CD4 count was 26 cells/ μ L, with absence of acquired immunodeficiency syndrome (AIDS)-defining illnesses. **Treatment:** Topical steroids, sun-protection and trimethoprim-sulphamethoxazole prophylaxis were prescribed. **Outcome:** Improvement was documented over the arms, but the facial plaques persisted. **Conclusion:** CAD may be a presenting feature of advanced HIV infection.

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Key words: AIDS, Photodistribution, Photosensitivity

Case Report

A 40-year-old Chinese man of skin phototype III (Table I), formerly a welder in a shipyard, presented with pruritic papules on his face, neck and arms of 3 years' duration. He had noted sun-aggravation and had ceased working for 3 months. He had bronchial asthma, for which he had been receiving theophylline for many years. Apart from medicated soaps, there was no contact with chemicals.

Examination revealed erythematous chronic lichenified plaques over his face (Figs. 1 & 2), and eczematous papules and plaques over the sides of his neck, the V of his chest, and the extensor aspects of his forearms. Clubbing was noted. Systemic examination was otherwise unremarkable.

Chronic actinic dermatitis (CAD) was suspected and a series of tests were carried out. Patch test to a standard battery, to exclude concomitant contact dermatitis in CAD, was positive for potassium dichromate and hexachlorophene. The former was attributed to previous exposure, and the latter to exposure to medicated soap, which the patient had ceased using. Photopatch test was negative. Phototest with Dermaray ultraviolet B (UVB) source at 1 mW/cm² revealed decreased minimal erythema dose (MED) to UVB (40 mJ/cm²). Phototest with Murtzhas light source at 36 mW/cm² revealed MED to ultraviolet A (UVA) of 75 mJ/cm² that was at the lower range of normal for his skin type (normal range 77 to 145 mJ/cm²). Histology of a biopsy taken from the forehead showed intracorneal

TABLE I: CLASSIFICATION OF SKIN PHOTOTYPES

Skin phototype	Experience with sun exposure
I	Always burns, never tans
II	Usually burns, sometimes tans
III	Sometimes burns, usually tans
IV	Never burns, always tans
V	Moderate constitutive pigmentation
VI	Marked constitutive pigmentation

pustules with neutrophils, acanthosis, spongiosis, moderately dense perivascular inflammatory infiltrate with eosinophils, and large stellate fibroblasts in the upper dermis. There were no lymphoma-like changes to suggest actinic reticuloid. The histological findings were consistent with photodermatitis (Figs. 3 & 4). Full blood count showed a white cell count of 6.5×10^9 /L, haemoglobin of 16.3g/dL and platelet count of 204×10^9 /L. Eosinophilia was present, with absolute eosinophil count of 1.3×10^9 /L. The following investigations were negative or normal: blood and urine porphyrin, liver function tests, anti-nuclear antibody titre, creatinine kinase, aldolase and chest X-ray.

He was treated with topical steroids, a physical sunscreen and advice on sun protection. The rash over his arm improved after 1 month, but he had persistent plaques on his face and subsequently, scrotum.

A history of unprotected sexual exposure was elicited following the scrotal rash, and he was screened for syphilis

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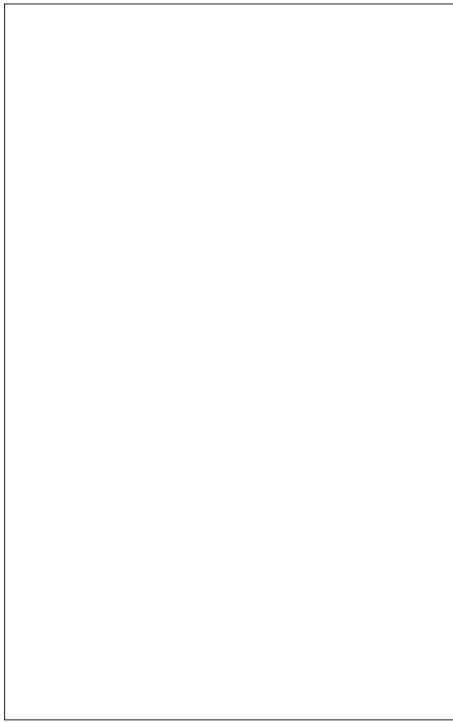


Fig. 1. Erythematous papules and plaques over the face in typical photodistribution, with sparing of nasolabial folds and the areas under the nose and lips.

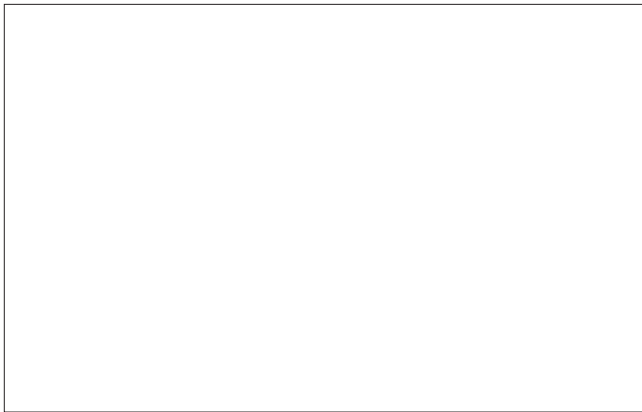


Fig. 2. Close-up of rash reveals eczematous morphology and lichenification.

and human immunodeficiency virus (HIV), which were both positive. His absolute CD4 count was 26.0 cells/ μ L (342.0 to 929.0), CD4/CD8 ratio was 0.04 (1.00 to 2.20), and HIV RNA viral load was 382,000 copies/mL. He does not yet have any acquired immunodeficiency syndrome (AIDS)-defining illnesses. He did not have any stigmata of syphilis but was treated with IM benzathine penicillin 2.4 megaunits weekly for 3 weeks for late latent syphilis. He was commenced on trimethoprim-sulphamethoxazole for *Pneumocystis carinii* prophylaxis but was unable to afford anti-retroviral therapy.

Despite sun avoidance, use of sunscreens and topical steroids for 11 months since diagnosis, he continues to have eczematous plaques over his face.



Fig. 3. Acanthosis and spongiosis of the epidermis. An intracorneal pustule with neutrophils is also seen (haematoxylin and eosin, original magnification x40).

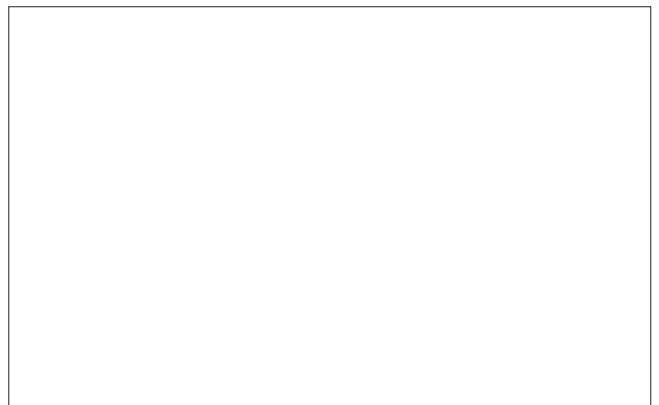


Fig. 4. Perivascular inflammatory infiltrate was present in the upper dermis (haematoxylin and eosin, original magnification x100).

Discussion

HIV infection, particularly AIDS, is associated with a myriad of cutaneous manifestations. The more well-recognised cutaneous manifestations include dermatoses such as seborrhoeic dermatitis, pruritic papular eruptions and eosinophilic pustulosis; neoplasms e.g., Kaposi's sarcoma; florid infections such as molluscum contagiosum, chronic herpes simplex or herpes zoster; and infection with unusual organisms such as atypical mycobacteria.¹ Increasingly, photosensitive disorders have been reported in association with HIV infection.² These include porphyria cutanea tarda,³⁻⁸ CAD,^{9,10} photosensitive granuloma annulare,¹¹⁻¹³ lichenoid photoeruptions¹⁴⁻¹⁶ and photo-distributed hyperpigmentation.² Some, particularly the lichenoid eruptions, have been associated with the use of potentially sensitising drugs (e.g., trimethoprim-sulphamethoxazole, dapsone, non-steroidal anti-inflammatory drugs);¹⁶ but in several cases, there was no history of potential sensitisers.

CAD is an idiopathic photosensitivity of eczematous morphology, which preferentially affects elderly men and persists for months to years in the absence of continued exposure to photosensitisers. The term CAD has over the years been accepted to encompass the previously distinct

entities of actinic reticuloid, photosensitive eczema, persistent light reactivity and photosensitivity dermatitis, as transitions between these conditions have been well documented.^{17,18}

Our patient met the criteria for diagnosis of CAD as proposed by Norris and Hawk¹⁷ (Table II). He had a persistent photodistributed eczematous eruption accompanied by a reduction in MED of covered skin to UVB, and histology consistent with photodermatitis. A concomitant diagnosis of AIDS was made based on the CD4 count of less than 200 cells/ μ L, although he did not yet have any AIDS-defining illness.

There have been 8 reported cases of CAD in association with HIV^{9,10} (Table III), with a majority (7) occurring in Afro-Americans of skin type VI. In 6 patients, photosensitivity was the presenting feature, pre-dating the

diagnosis of HIV and the use of potentially photosensitising medications. Most of them had HIV risk factors, such as our patient, which emphasizes the importance of a good history. However, 2 patients did not report any HIV risk factors.¹⁰ This highlights the importance of recognising this clinical association and screening for HIV when no other cause has been found. CD4 count was markedly suppressed in all of them ($<0.2 \times 10^9/L$), suggesting that photosensitivity is a late feature of HIV infection.

Most were treated with topical steroids and sun protection, with poor to good response. Our patient responded initially with photoprotection and topical steroids, particularly over his arms, but the rash on his face has persisted for 11 months despite treatment. Hydroxychloroquine was used in addition to topicals in 3 patients,⁹ with no improvement. PUVA was tried in 1 patient,¹⁰ with fair to good response. Although immunosuppressive therapy with azathioprine or cyclosporin has been effective in CAD,¹⁹ the additional immunosuppression is undesirable in HIV infection.

The finding of photosensitivity in association with HIV does not necessarily imply causation. However, as CAD and HIV infection are both uncommon conditions, the increasing number of reports of CAD associated with HIV infection suggests that the association may be more than a mere coincidence.

The pathogenesis of CAD is unclear, but it has been

TABLE II: DIAGNOSTIC CRITERIA FOR CHRONIC ACTINIC DERMATITIS (NORRIS AND HAWK¹⁷)

Criteria	
1. Clinical:	Persistent eczematous eruption, predominantly photodistributed
2. Photobiological:	Reduced MED-UVB, often also to longer wavelengths
3. Histological:	Changes consistent with chronic eczema, $\pm$ chronic lymphoma-like changes

TABLE III: COMPARISON OF OUR CASE WITH 8 REPORTED CASES OF CAD ASSOCIATED WITH HIV^{*9,10}

No.	Skin type	Age/Sex	Risk factors	Duration	MED-A J/cm ²	MED-B mJ/cm ²	Presenting feature?	CD4 count ($\times 10^9/L$)	Treatment	Outcome
1	-	50/M	Homosexual	15 y	$\downarrow$ <1	$\downarrow$ <10	Yes	0.02	Topical steroids; sun protection	Complete clearance
2	VI	52/M	Homosexual	1 y	normal	$\downarrow$ < 20	Yes	0.15	Topical steroids; sun protection	Gradual improvement
3	VI	36/M	None	1.5 y	normal	$\downarrow$ < 20	Yes	0.007	PUVA	Fair to good response
4	VI	49/F	None	2 mo	normal	$\downarrow$ < 60	Yes	0.08	Topical steroids; sun protection	Some improvement
5	VI	58/M	Sex with prostitutes	2 mo	$\downarrow$ 18	$\downarrow$ 72	Yes	0.018	Hydroxy-chloroquine	No improvement
6	VI	44/M	IVDU	3 mo	normal	$\downarrow$ < 6	No; AIDS 1 y	0.004	Topical steroids; sun protection	Improved
7	VI	48/M	IVDU	3 mo	$\downarrow$ 18	$\downarrow$ 24	Yes	0.04	Hydroxy-chloroquine	No improvement
8	VI	50/M	Bisexual	2 hr after UVB therapy	$\downarrow$ 3	$\downarrow$ 36	No; HIV+, eosinophilic pustular folliculitis	0.135	Hydroxy-chloroquine	No improvement
9	III	40/M	Sex with prostitutes	3 y	normal	$\downarrow$ 40	Yes	0.026	Topical steroids; sun protection	Some improvement

CAD: chronic actinic dermatitis; IVDU: intravenous drug use; MED: minimal erythema dose; PUVA: psoralen-ultraviolet A; UVB: ultraviolet B

* Cases 1 to 4 from Pappert et al,¹⁰ cases 5 to 8 from Meola et al⁹ and case 9 is this case report.

postulated that UVB absorption leads to transformation of an endogenous protein to a neoantigen, with subsequent delayed type hypersensitivity as in allergic contact dermatitis. Pappert et al¹⁰ found a markedly decreased CD4/CD8 ratio in the skin biopsy of an HIV patient with CAD, which may reflect the reversed peripheral blood CD4/CD8 ratio in HIV patients. However, decreased CD4/CD8 ratio with predominance of CD8 cells is also found in lesional skin in non-HIV patients with CAD,²⁰ particularly those with severe CAD.¹⁹ Furthermore, a reversal of peripheral blood CD4/CD8 ratio has also been observed in an HIV-negative CAD patient.¹⁰ Pappert et al hypothesised that the immune response in CAD is regulated by CD8 cells reacting to some photoinduced self-antigen, with loss of control of anti-self response due to decreased CD4/CD8 ratio in HIV, and a selective defect in non-HIV CAD.

Whether HIV plays a role in the pathogenesis of photosensitivity disorders in general has yet to be established. The increasing number of reports of photosensitive disorders associated with HIV infection suggests it, but 2 case-control studies studying the skin response to UVB in HIV-infected patients did not find any significant difference in UVB sensitivity between the 2 groups.^{21,22} In the study by Kaporis et al,²¹ 57 HIV patients and 57 controls were receiving UVB therapy for various non-photosensitive skin disorders. Although the MED to UVB was not significantly different between the 2 groups, stratified according to skin type, 1 HIV patient developed CAD 3 months into therapy, with marked decrease in MED. The numbers were small in the second study (12 in each group) involving HIV-infected patients and healthy volunteers.²² Although the MED to UVB was again not significantly different between the 2 groups, there was a trend towards more erythema and pigmentation with 6 MED exposure in the HIV group. This did not achieve statistical significance. However, a repeat study with a larger study population may reveal statistical significance. In both studies, the results were not stratified according to CD4 count, which may be important as CAD, photosensitive GA and photo-distributed hyperpigmentation appear to occur in late-stage HIV infection.

In conclusion, we report a case of CAD that was subsequently found to have advanced HIV infection. Clinicians should be aware of the association between CAD and HIV infection, where photosensitivity is often the presenting feature pre-dating the diagnosis of HIV infection. We recommend HIV testing in patients presenting with photosensitivity where no cause is found, particularly if risk factors for HIV are elicited.

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