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REVIEW

Common dermatological conditions in the HIV patient

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The introduction of antiretroviral medication has changed the epidemiology, morbidity and mortality of HIV disease. Nevertheless, cutaneous disorders due to HIV infection remain a major problem in HIV-infected patients. These disorders are mainly infections, inflammatory and neoplastic in origin. Some of these disorders occur at normal CD4 cell counts, while others occur typically at low CD4 cell counts. Clinicians should be aware of various presentations of these disorders and their treatment as they can impact negatively on patient's quality of life.

Keywords: HIV, immunocompromised

Viral Infections

Herpes simplex virus infections

Herpes simplex virus (HSV) infection is very common in the setting of HIV.[1] Herpes labialis caused by HSV type 1 occurs around the nose and lips. In the HIV setting it tends to be more aggressive and lesions last longer (Figure 1).

Herpes genitalis caused by HSV type 2 is the most frequent genital ulcer disease in HIV patients.[2] Herpes genitalis presents as vesicles, erosions, and ulcers on the anogenital area.

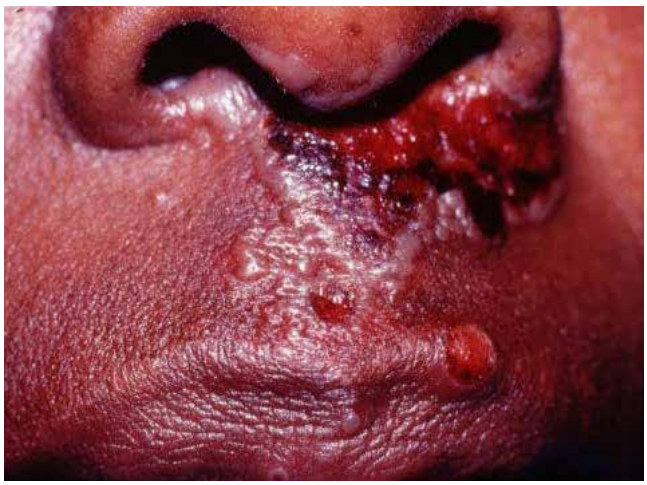

Figure 1. Herpes labialis in an HIV- infected patient

Herpes zoster (Shingles)

Herpes zoster is common in HIV and tends to be multidermatomal (Figure 2). In HIV, it may cause disseminated cutaneous disease and HIV patients tend to have recurrent episodes.[3] It presents as painful vesicles following a dermatome. Treatment is aimed at speedy healing of skin lesions, limiting disease progression, reduction of pain and prevention of complications like post-herpetic neuralgia.

For patients who present within 72 hours of these painful vesicles appearing, oral acyclovir 800 mg five times a day for seven days, or valacyclovir 1 000 mg three times a day for seven days is adequate. Pain should be controlled with analgesics and anti-inflammatory drugs.[1]

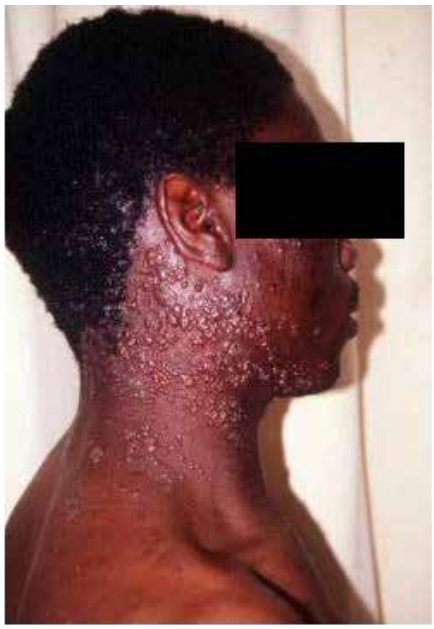

Figure 2. Multidermatomal herpes zoster

Molluscum contagiosum

It is caused by a poxvirus and is common in HIV patients. Typical lesions are skin-coloured, dome-shaped papules or nodules, often with a central umbilication. Lesions may be quite extensive in immunocompromised patients (Figure 3). In HIV, lesions may be atypical and resemble other conditions such as bacillary angiomatosis.[1] Treatment of molluscum contagiosum in HIV patients includes restoration of immune competence by highly active antiretroviral therapy (HAART). In some patients lesions respond to immunomodulators like imiquimod 5%.

Resistant lesions may also be treated with cryotherapy, which involves application of liquid nitrogen onto the lesions for cold-induced cell destruction, however, this may not be possible in patients with extensive disease.[1]

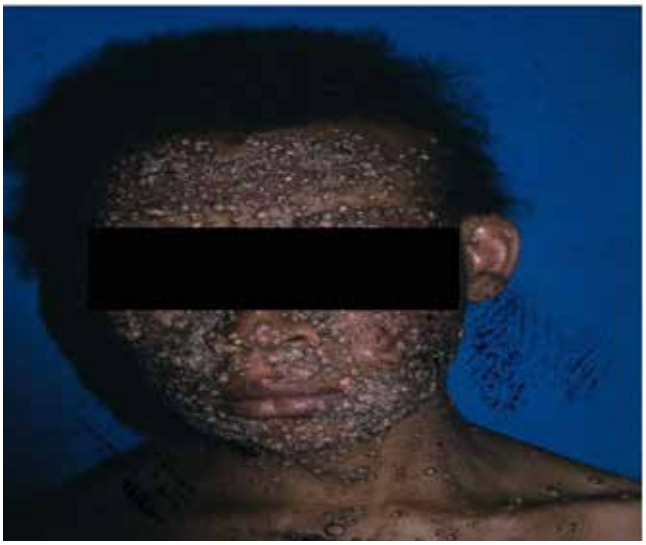

Figure 3. Extensive molluscum contagiosum in an HIV –infected patient

Viral warts

These are caused by the human papilloma virus (HPV). Both verruca vulgaris, verruca plana, and genital warts (condyloma accuminata) are common in HIV patients. Verruca vulgaris presents as verrucous, fungating, cauliflower-like lesions, whilst verruca plana are flat (Figure 4). Topical therapies like trichloroacetic acid and podophyllin resin in a compound tincture of benzoin may help. Ablative therapies such as cryotherapy and curettage may be helpful in patients with fewer lesions.[1]

Figure 4. Extensive verruca plana

Bacterial Infections

Staphylococcus aureus infections

Staphylococcus aureus is the commonest bacterial pathogen in HIV, causing folliculitis, impetigo, ecthyma and skin abscesses (Figure 5). Treatment of staphylococcal skin disease involves the use of systemic antibiotics like cloxacillin and application of topical antibiotics like mupirocin or fucidin. Antiseptic solutions in bath water help to prevent recurrent episodes.[1]

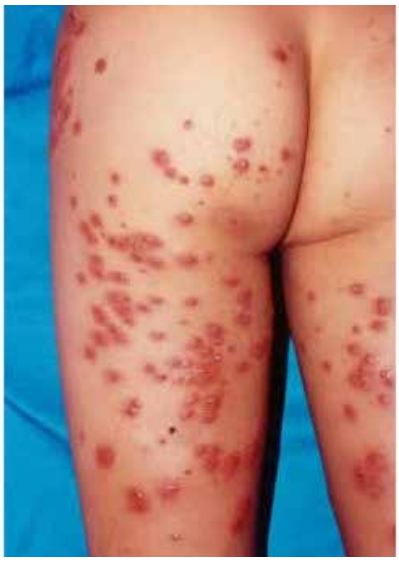

Figure 5. Staphylococcal folliculitis in HIV

Bacillary angiomatosis

This is a vascular proliferative disease common in HIV.[4] It is caused by Gram-negative bacilli *Bartonella quintana*.

The proliferative vascular lesions commonly involve the skin but may be present in many other tissues including lymph nodes, bone, brain, respiratory tract, gastrointestinal tract, cardiac valves and bone marrow.[4]

It has been suggested that cutaneous lesions of bacillary angiomatosis may be a marker of systemic infection, especially in HIV-positive patients.[5] The disease responds well to erythromycin, doxycycline, ceftriaxone, and fluoroquinolones can also be used.

Syphilis

Syphilis is common in HIV, especially in HIV-positive men who have sex with men.[6] Syphilis and HIV frequently tend to occur together because both are sexually transmitted and the risk factors for acquisition are the same, and furthermore, ulcers of primary syphilis tend to facilitate the transmission of HIV.[7] On the other hand, HIV is known to modulate both the manifestations of syphilis and the serological response to therapy.[7]

HIV-infected patients with primary syphilis tend to have multiple chancres compared with non-HIV patients.[8] HIV-infected patients with secondary syphilis tend to have genital ulcers of primary syphilis thus they have an overlap of primary and secondary syphilis (Figure 6).

HIV patients with syphilis have a higher likelihood of developing neurosyphilis. Serological tests for syphilis are often false-negative and even serologically defined treatment failures have been reported.[8,9]

Treatment of syphilis in HIV-infected patients is the same as in HIV-negative patients. For primary and secondary syphilis the

treatment is benzathine penicillin 2,4 mu imi weekly X3, oral doxycycline is also helpful.[8]

Figure 6. Overlap of primary and secondary syphilis in an HIV-infected patient

Mycobacterial infections

Cutaneous tuberculosis is divided into three categories as follows:

- Inoculation tuberculosis, a primary infection of the skin that is introduced by an exogenous source, e.g. lupus vulgaris and tuberculous chancre.
- Secondary tuberculosis, either by contiguous or haematogenous spread from a primary focus that leads to involvement of the skin, e.g. scrofuloderma.
- Tuberculids which are hypersensitivity reactions to M. tuberculosis components, e.g. papulonecrotic tuberculid, erythema induratum of Bazin and lichen scrofulosorum.

Lupus vulgaris is the most common form of cutaneous tuberculosis and papulonecrotic tuberculid is the commonest tuberculid.

Fungal Infections

Both dermatophytes and deep fungal infections are common in HIV. Dermatophyte infections are caused by fungi that invade the superficial dead layer of the skin as well as keratinised tissues like hair and nails. They include tinea corporis, tinea pedis, tinea cruris and onychomycosis.

Deep fungal infections which are common in HIV are cryptococcosis and histoplasmosis. *Cryptococcus neoformans* has a predilection to the skin and the central nervous system. Skin lesions in cutaneous cryptococcosis may be papules, nodules, subcutaneous nodules, Kaposi sarcoma-like lesions and molluscum contagiosum-like lesions.

Cryptococcal meningitis occurs in 75% of HIV infected patients with cryptococcosis, unfortunately symptoms and signs may be very subtle, making it difficult to make early diagnosis.[10]

Ideally all patients with cutaneous cryptococcosis should have their cerebrospinal fluid examined as some patients may have cryptococcal meningitis with no symptoms and signs.[11]

Histoplasmosis is also common in HIV. The aetiological agent of histoplasmosis associated with AIDS is *Histoplasma capsulatum var capsulatum*.[11]

The clinical presentation includes fever, weight loss, hepatosplenomegaly, skin lesions and pulmonary involvement.

Cutaneous involvement occurs in 11% of patients due to haematogenous dissemination from the pulmonary focus. Skin lesions are non-specific and may be papules, pustules, plaques, nodules, abscesses and ulcers.

Parasitic Infections

The commonest parasitic infection in HIV is Norwegian scabies (crusted scabies) caused by *Sarcoptes scabiei*. This is highly contagious.[12] Affected patients present with generalised, hyperkeratotic, scaly plaques that fall off easily (Figure 7). Unlike in ordinary scabies, pruritus in Norwegian scabies is usually slight or absent.[12]

Norwegian scabies is a clinical diagnosis. Biopsy can confirm the diagnosis in case of doubt. Norwegian scabies has to be managed promptly to avoid dissemination of this highly contagious disease.

Patient's clothes and linen must be soaked and washed with hot water. Patients must bath with anti-scabicide soaps like Tetmosol soap.

Benzyl benzoate cream must be applied daily, but this is a skin irritant that may not be tolerated by children, in which case 10% sulphur in emulsifying ointment can be used.

In many African countries where parasites are a major problem, cases of Norwegian scabies are treated with intramuscular or oral ivermectin.

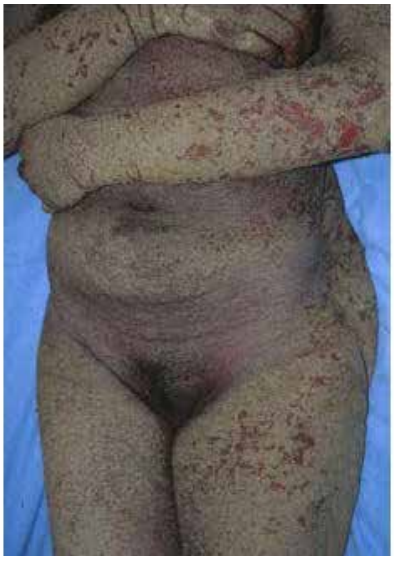

Figure 7. Extensive Norwegian scabies in advanced HIV disease

In South Africa ivermectin is currently only registered for veterinary use.[1]

Inflammatory Dermatoses

Seborrhoeic dermatitis

It is a common chronic inflammatory dermatosis. The incidence of seborrhoeic dermatitis in the general population is 2,35% to 11,3%.[13] In HIV patients the incidence increases to 30% to 80% depending on the population studied.[14,15]

In many studies seborrhoeic dermatitis is the most frequent cutaneous finding in HIV-infected patients. Clinically seborrhoeic dermatitis in HIV patients is usually atypical and more severe (Figure 8).

Treatment involves treating the underlying HIV infection with antiretrovirals, antifungal shampoos and topical corticosteroids. Pimecrolimus and tacrolimus can be used on steroid-sensitive areas.

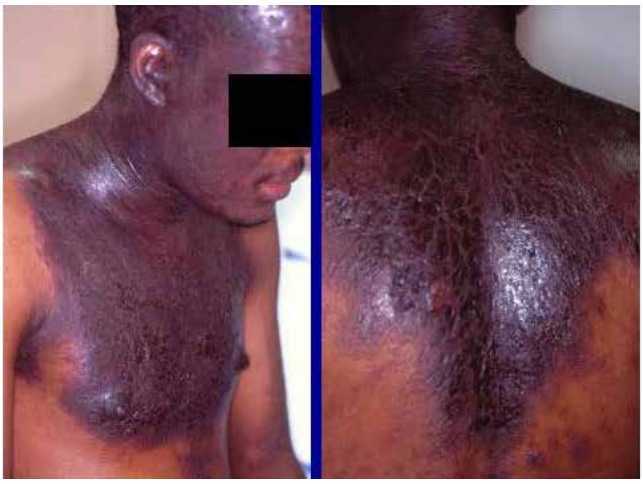

Figure 8. Extensive seborrhoeic dermatitis in HIV

Papulo-pruritic eruption (PPE)

Papulo-pruritic eruption is a common cutaneous manifestation of HIV. The incidence of papulo-pruritic eruption of HIV varies between 12% and 46% depending on the geographic location.[16]

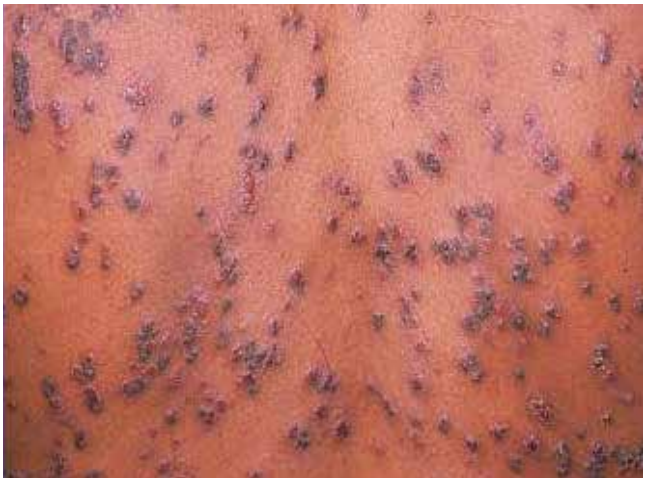

Figure 9. Papulo-pruritic eruption of HIV. Note the extensive excoriations and post inflammatory hyperpigmentation

PPE presents as extremely pruritic, symmetric, skin-coloured to erythematous papules and pustules. Excoriations are common due to intense pruritus (Figure 9).

The cornerstone of treatment is the use of HAART, often resulting in a dramatic improvement of symptoms. Topical corticosteroids, emollients and antihistamines alone, are usually of limited value. In resistant cases, narrow-band UVB phototherapy can be helpful.[17]

Eosinophilic folliculitis of HIV

It is another pruritic eruption occurring in HIV-infected patients, closely resembling papulo-pruritic eruption.[18] It presents as follicular, oedematous papulovesicles or pustules, affecting the face, neck and upper arms. These lesions are extremely pruritic and they become secondarily excoriated.[19] Management is more or less the same as in papulo-pruritic eruption.

Xerosis (dry skin)

It is also a common finding in HIV-positive patients (Figure 10). In some studies it is the most common skin manifestation in HIV and is one of the major causes of pruritus in HIV patients.[20] Xerosis worsens with a decrease in CD4 cell count. Mainstay of treatment is the use of emollients.

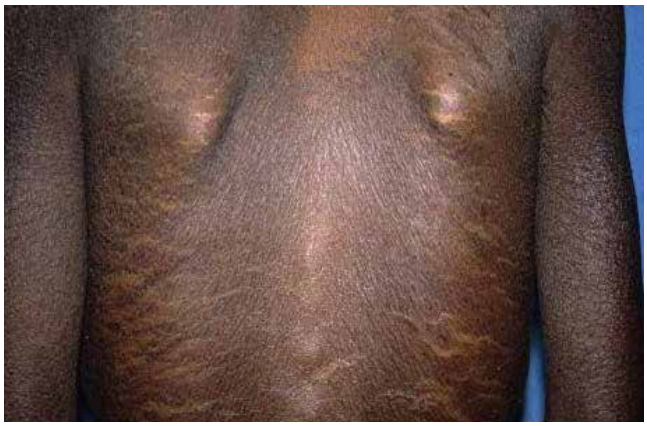

Figure 10. Marked xerosis and acquired ichthyosis in HIV

Photosensitive dermatitis

Photosensitivity occurs in approximately 5,4% of HIV-positive patients, especially in pigmented skin. The virus itself renders the body to be photosensitive. Lesions are distributed on sun-exposed areas with a sharp demarcation on covered areas (Figure 11).

In severe cases it can spread to involve sun-protected skin and even become generalised. Treatment is difficult and involves the use of HAART, sun protection and topical corticosteroids.[2]

Neoplastic Diseases

HIV-associated Kaposi sarcoma

In South Africa, human immunodeficiency virus infection is widespread and HIV-associated Kaposi sarcoma is common.[22] It is the commonest neoplastic disease in HIV patients. Early lesions

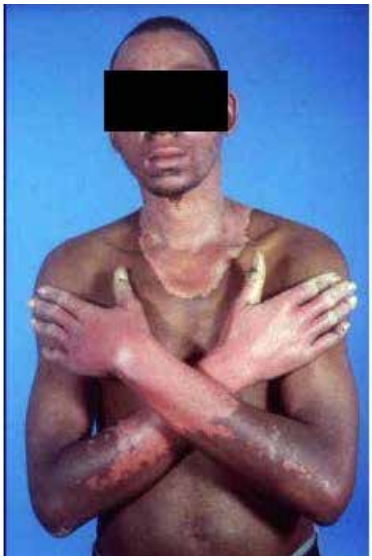

Figure 11. Photosensitive dermatitis in HIV. Note the distribution on sun exposed areas

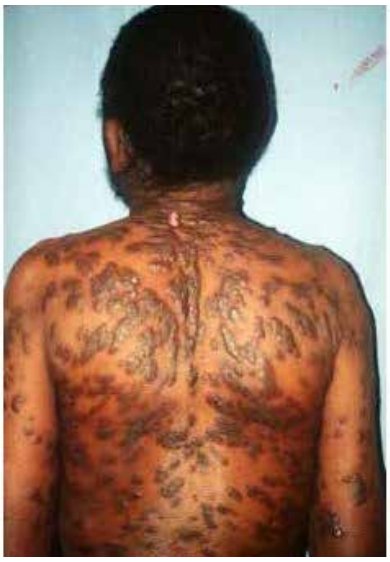

Figure 12. Multiple plaques of Kaposi sarcoma in HIV

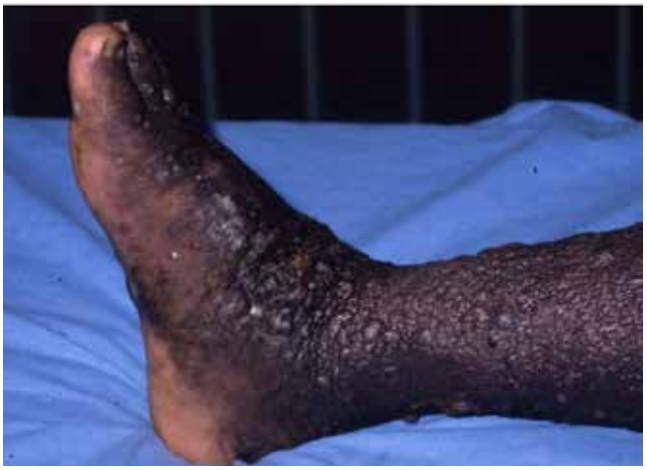

Figure 13. Extensive Kaposi sarcoma with lymphoedeoma and multiple nodules

are erythematous-bluish macules, which develop into plaques and nodules (Figure 12).

With time, massive lymphadenopathy develops on the limbs and results in incapacitation (Figure 13). These patients must be on HAART. Treatment includes radiotherapy for limited skin disease and chemotherapy for extensive skin disease with systemic involvement.

Cutaneous B-cell and T-cell lymphomas are also common neoplastic diseases in the setting of HIV.

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