Dermatological Manifestations : A Clinical Predictor of AIDS

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Abstract

AIDS (Acquired Immune Deficiency Syndrome) was first described as a distinct clinical entity in 1981. Since then, AIDS, which is caused by HIV (Human Immunodeficiency Virus) infection, has been spreading it's tentacles and has come to assume the status of a global pandemic at present. HIV is associated with a wide spectrum of clinical disorders ranging from an asymptomatic infection to AIDS-related complex. The virus produces a panorama of muco-cutaneous manifestations. In resource-poor settings where methods for early detection and management of HIV/AIDS are not readily available, muco-cutaneous disorders may be used as reliable predictors of disease progression and the underlying immune status of the patients, thus, allowing initiation of appropriate antiretroviral therapy and modifying the clinical course of the disease.

Introduction

Ever since its recognition in 1981, HIV/AIDS continues to ravage humanity across all the continents of the world. Within two years of AIDS being defined as a distinctive syndrome, human immunodeficiency virus (HIV) was identified as the causative agent [1]. According to the National AIDS Control Organization (NACO) annual report 2014-15, the total number of people living with HIV/AIDS in India was estimated at

around 20.9 lakh in 2011, 86% of whom were in 15-49 years age-group [2].

Role of Skin Diseases as a Marker of HIV/AIDS

Since the discovery of HIV infection, a number of skin diseases are described to be associated with it. Dermatological manifestation can serve as a window to other systems. Hence, a thorough examination can guide to a diagnosis with severity or stage of affliction. Cutaneous manifestations can be classified into five groups: infectious, auto-immune, drug-induced, HIV-related, and cutaneous malignancies. The prevalence of muco-cutaneous manifestations varies between 68.8% and 90% [3].

Skin diseases have proved to be sensitive and useful measures through which HIV progression can be monitored. Although skin lesions may be seen in the general population, their occurrence in HIV-infected patients is often atypical and more severe, explosive, extensive or resistant to therapy. The number and severity of dermatoses increase with the advancement of AIDS and at a relatively low CD4 count. However, with the advent of HAART in 1995, there has been a temporal reduction in muco-cutaneous manifestations [4].

The various clinical manifestations are tabulated in Table 1.

Table 1. Cutaneous Manifestation in HIV-Positive Patients

INFECTIOUS DERMATOSES	NON-INFECTIOUS DERMATOSES
1. VIRAL • Herpes simplex virus • Herpes zoster virus • Human papilloma virus • Epstein bar virus • Molluscumcontagiosum virus • Cytomegalovirus	1. PAPULOSQUAMOUS • Psoriasis • Seborrheic dermatitis • Reiters disease • Ichthyosiform dermatoses • Xerosis • Prutitus • Eosinophilic folliculitis • Popular pruritic eruption • Eczemas • Papularurticaria
2. BACTERIAL • Pyodermas: staphylococcal, streptococcal, pseudomonas. • Mycobacterial infections • Bacillary angiomatosis • Treponema pallidum • Others	2. PIGMENTARY CHANGES • Diffuse pigmentation • Pigmentation of oral cavity

3. FUNGAL • **Superficial Fungal Infections** Candida Dermatophytoses Pityrosporum infections • **Deep Fungal Infections** Cryptococcus neoformans Histoplasma capsulatum Penicilliummarneffei Pneumocystis jiroveci Others	3. NEOPLASMS • Kaposi sarcoma • Melanomas • Lymphomas • Squamous cell carcinomas
4. PARASITIC INFESTATIONS • Sarcoptesscabiei • Demodicidosis • Toxoplasmosis • Leishmaniasis	4. HAIR CHANGES • Diffuse alopecia • Alopecia areata • Sparseness of hair • Long eye lashes
	5. ORAL CHANGES • Oral candidiasis • Oral ulcerations • Oral hairy leucoplakia • Xerostomia • Melanotic hyperpigmentation • Salivary gland enlargement • Linear gingival erythema • Necrotising ulcerative gingivitis • Necrotising ulcerative periodontitis • Acute necrotising ulcerative gingivitis
	6. NAIL CHANGES • Onychomycosis • Melanotic band

Spectrum of Skin Conditions

1. Acute seroconversion syndrome

Patients may present with fever, sore throat, cervical adenopathy. A symmetrical maculopapular erythematous rash is found in 75% of people [1]. Painful oral ulceration, genital ulceration, erythema multiforme and Stevens-Johnson Syndrome may occur [5,6].

2. Bacterial infections

Pyodermas

Staphylococcus aureus (including methicillin-resistant S. aureus), Pseudomonas species, Escherichia coli and Streptococcus pyogenes are the commonest isolates in that order; polymicrobial infection may be present in up to 40% of cases [7]. Besides folliculitis, manifestations of staphylococcal infections in HIV include bullous impetigo, ecthyma, cellulitis, abscesses, botromycosis and staphylococcal scalded skin and toxic shock syndromes [1]. Subcutaneous abscesses due to staphylococci may complicate injection or intravenous line sites. Group A Streptococcus erysipelas and lymphadenitis has been reported [1]. Pseudomonas aeruginosa causes Ecthyma gangrenosum and panniculitis in advanced HIV [8].

Bacillary Angiomatosis

The causative agents are B. quintana and B. henselae. It is most commonly seen in patients with CD4 <100/µL, but cases occurring in the first year following seroconversion with CD4 count > 500/µL have been reported [8, 9]. It presents as single or multiple red-purple nodules on the eyelids, mucosae, liver or spleen. Histology shows lobular capillary proliferation. Warthin-Starry stain demonstrates clumps of tangled bacilli [8, 9].

Mycobacterial Infections

HIV-TB co-infection is a serious problem worldwide, but especially of concern in India where background rates of TB are the highest in the world [10]. Prevalence of HIV among patients with radiologic or bacteriologic confirmation of TB in India ranges from 2.8 to 9.4 percent [11]. According to an estimate, around 5.1 million people are infected with HIV and about half of these cases are co-infected with tuberculosis [12].

The clinical presentation is diverse as shown in Table 2 and Figure 1.

Table 2. Clinical Presentation of Cutaneous Tuberculosis

1.	Scrofuloderma
2.	Palmoplantar keratoderma
3.	Acute military tuberculosis of skin
4.	Keratotic papules
5.	Tuberculids
6.	Scattered violaceous papules
7.	Tuberculous lymphadenitis
8.	Atypical mycobacterial infection
9.	Buruli ulcer

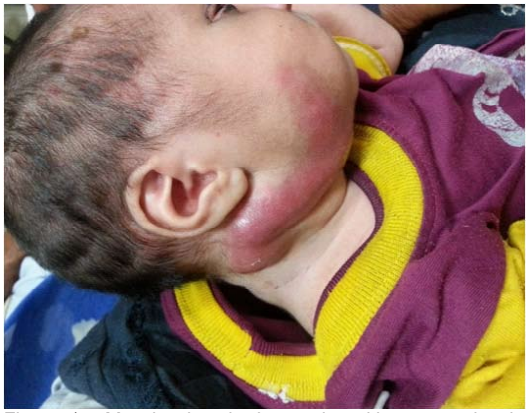

Figure 1. Massive lymphadenopathy with suppuration in HIV-positive infant.

Mycobacterium avium intracellular complex (MAC) is one of the most frequent atypical mycobacterial infections in HIV-infected patients. This occurs as part of a disseminated infection

in up to one-third of patients at CD4 T-cell counts below 50 × 106/L (rare below 200 × 106/L)[1]. Cutaneous manifestations include violaceous papule, nodule and ulcer. M. ulcerans induced aggressive multifocal Buruli ulcer has also been reported [1].

3. Viral infections

Herpes simplex virus

There is a synergistic relation between Herpes Simplex Virus(HSV) and HIV infection. Although ano-genital involvement is frequent, any site can be affected. Atypical presentations include large deep ulcers extending to perineum, buttocks and abdomen, nodular, hyperkeratotic and condylomatous lesions [1]. According to CDC guidelines, any non-healing ulcers of HSV lasting for more than 1 month is an AIDS-defining illness and indicate active HIV [13].

Varicella zoster virus

Varicella may run a prolonged course (>10 days) in HIV-infected children and is frequently associated with complications like pneumonitis, bacterial superinfection and meningitis [3,14]. Von Seidlein et al. documented an association between increasing numbers of episodes of VZV infection and a low CD4 count at the time of primary infection [15].

Herpes zoster occurs in about 6–31.5% patients with HIV-AIDS [16]. Multi-dermatomal lesions are more frequent in advanced HIV disease. Atypical features such as necrotic punched-out ulcers or hyperkeratotic ulcerated nodules have been reported [17]. Systemic complications including fulminant hepatitis and acute meningoencephalitis may occur [3].

Human papilloma virus

Human papilloma virus (HPV), which causes oral, genital and anal warts, has been reported in 29% of buccal mucosal cells and 63% of cervical cells in female sex workers in Kolkata [18].

Both verruca vulgaris and condylomata acuminate are common in HIV disease. There is an increased incidence of facial and intra-oral warts. The extent of disease and the number of HPV types tends to increase as the CD4 count drops. In the anogenital area, condylomata acuminate may form large vegetating masses (Figure 2) or may extend into the anal canal where squamous cell carcinoma may develop. Recent study suggests routine anal cytology to all HIV-infected men especially in those with low CD4 [19].

The most important known determinant of human papilloma virus (HPV) persistence and progression to cancer is viral type, notably the presence of HPV16 [19]. Immune suppression by HIV infection also appears to worsen the outcome of HPV infection. Women infected with HIV are at significantly increased risk for invasive cervical cancer [20].

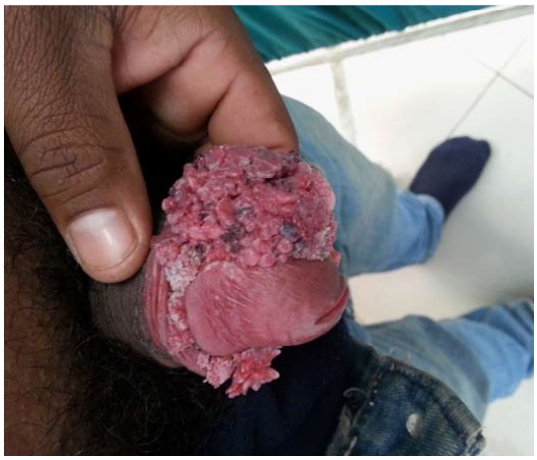

Figure 2. Condyloma Acuminate in a 25-year old HIV-positive male.

Molluscum contagiosum

The incidence of Molluscum contagiosum(MC) in HIV varies from 5% to 18% in adults and 21% in children [21]. MC should be considered as a first sign of HIV infection, especially extragenital and eruptive MC [22]. Typical lesions are shiny, umblicated, pearly, dome-shaped papules ranging from 2 to 5 mm [22]. However, atypical lesions including florid, extensive, genital lesions with cellulitis, several papular and nodular lesions lacking the characteristic central umblication, giant lesions larger than 1 cm, periorbital and intraoral lesions have been reported [1,3]. Disseminated Penicillium marneffei infection, which can be confused with molluscum contagiosum has been reported in Manipur [23].

Cytomegalo virus infection

Reactivation of Cytomegalo virus (CMV) in HIV infection occurs with a CD4 count below 50 × 106/L. Skin involvement with CMV is relatively uncommon in HIV, but when CMV affects the skin, the mortality can be about 85% in 6 months [1]. Muco-cutaneous manifestation can be variable including painful ulcers of the perineum, thigh, buttocks & oral cavity, purpura, papules, nodules, verrucous plaques, coagulopathies and nodular prurigo [1].

4.Fungal infections

Superficial fungal infections

Candidiasis

Oral candidiasis occurs frequently in individuals with HIV infection; it has been reported as the most common HIV-associated condition, occurring in up to 70% of cases [24]. Candida albicans is the most frequent species isolated, however, other species like C. glabrata, C. tropicalis, C. krusei have also been isolated and these may have decreased susceptibility to fluconazole [25].

Pseudomembranous disease is the commonest oro-pharygeal manifestation but erythematous (atrophic), chronic hyperplastic, papillary hyperplasia, median rhomboid glossitis may also be seen [1]. Candida can also be responsible for paronychia, onychodystrophy, angular cheilitis and intertriginous candidiasis [26]. It may present as a generalized cutaneous eruption of papules and nodules [27].

Dermatophytoses

Tinea corporis, tinea capitis, tinea faciale, and onychomycosis are particularly common. Cases of severe and recurrent tinea capitis have been observed. Widespread dermatophytosis, atypical presentation, and unusual forms of nail infection in the form of proximal subungual white onychomycosis (PSWO) have been described in association with HIV infection [28]. Kumarasamy et al. in their study from south India, found 8% of HIV-infected patients to be having dermatophytosis [29]. In severely immuno-suppressed patients with AIDS, lesions have little inflammation and often lack the elevated border and central clearing typical of tinea (Anergic tinea). They are recognized as sharply marginated areas of hyperkeratosis resembling dry skin. Onychomycosis in HIV infection commonly involves the toe nails. Proximal subungual onychomycosis and superficial white onychomycosis are commonly observed in HIV patients [30].

Deep fungal infections

Histoplasma capsulatum, Cryptococcus neoformans, Coccidio desimmitis, Aspergillus fumigatus, Penicillium marneffei, Sporothrix schenckii, and

others can cause opportunistic infections in HIV-infected adults.

Extra pulmonary infection with histoplasmosis is an indicator condition in the case definition of AIDS [1]. A wide morphological spectrum of lesions is seen. Macules, crusted/eroded/ulcerated papules and plaques mainly located on the face and chest, as well as oral involvement with erosions and ulcers, is the commonest presentation [1].

Cutaneous involvement can occur in 6–20% of cases of disseminated cryptococcosis [31]. It presents as papulonodular necrotizing skin lesions with central umbilication, like molluscum contagiosum in the context of neurological or pulmonary disease [1].

Penicilliosis is an AIDS-defining diagnosis, caused by dimorphic fungus Penicillium marfneffei. Patients with CD4 count less than 100/µl are at an increased risk [32]. The characteristic skin lesions of disseminated infection are umbilicated papules with or without central necrosis. Nevertheless, penicilliosis can manifest as ulcers, nodules, maculopapules, acneiform lesions or folliculitis [32].

5. Parasitic infestations

Scabies

Scabies occurs frequently in HIV-infected patients. Clinical presentation often depends on the degree of immunosuppression. Atypical clinical features such as involvement of head and neck which is highly unusual in non-HIV-infected adults, may be encountered with progressive immunosupression. Norwegian/crusted scabies is highly contagious and its diagnosis should arouse suspicion of underlying HIV infection. Lesions may be in the form of classical thick-crusted plaques, psoriasiform plaques or hyperkeratotic yellow papules resembling Darier's disease [3]. Crusted scabies in HIV infection may be localized to the soles or genitals [1].

Demodex

Demodex folliculorum is a saprophytic mite of human pilosebaceous unit. During HIV infection, demodiciosis occurs with CD4 count lower than 200/µl. Manifestations range from follicular pityriasis, rosacea like demodiciosis, pustular folliculitis, blepharitis and granulomatous rosacea [3].

6. Seborrheic dermatitis

The prevalence of SD in HIV-positive and AIDS patients is 34–83% as opposed to 3% in the general population [33]. In these patients, SD tends to occur early in the course of the disease (CD4+ T-cell count range 450–550 cells/µL) and is usually more severe and difficult to diagnose and treat than in the general population [34].

Its incidence and severity are closely related to the stage of HIV infection and inversely correlate with the absolute CD4 and helper T cell counts. More severe, widespread disease, with more erythematous, hyperkeratotic and psoriasiform lesions, has led to the suggestion that the clinical picture seen in HIV infection/AIDS should be termed as SD-like dermatitis of AIDS and should be regarded as a distinctive entity caused by immunological defects [33]. Histopathology shows a deeper lymphocytic infiltrate and a more perivascular neutrophilic (with occasional leukocytoclasis) and plasma cell infiltrate in HIV compared with classic SD [1].

7. Papular pruritic eruptions (PPE)

This is a very common cutaneous manifestation

of HIV-AIDS with Indian reports suggesting an incidence varying between 2% and 35.8% [35, 36]. It is usually a diagnosis of exclusion manifesting as chronic, sterile pruritic papules and pustules on the extensor surfaces of the arms, dorsa of the hands, trunk, and face with sparing of the palms and soles. The condition tends to wax and wane. Sometimes lichenified patches and plaques may be seen. It is associated with eosinophilia and elevated IgE levels. Lesions heal with disfiguring scarring and hyperpigmentation. Interestingly, in a study, 75% of these patients had circulating bullous pemphigoid autoantibodies [37].

It has to be differentiated from prurigonodularis, prebullous pemphigoid, scabies, papulo-necrotic tuberculid, drug eruption, photo-dermatitis, secondary syphilis, oncho-dermatitis and eosinophilic, seborrhoeic, bacterial and acneiform folliculitis [1].

8. Eosinophilic folliculitis

Eosinophilic folliculitis (EF) is a HIV-specific disorder occurring at CD4 T cell counts of 250–300/µl [38]. It presents as peri-follicular erythematous papules and pustules, commonly seen over the face and central trunk with sparing of acral sites. The lesions are pruritic and chronic but may display periods of improvement, unlike PPE. Histopathology is an important tool in differentiating PPE from EF and many other conditions that can mimic it. In EF, sterile inflammatory infiltrate consists of perifollicular eosinophils; unlike in PPE, which shows perivascular mononuclear cell infiltrate [38].

9. Psoriasis

The overall incidence of psoriasis is probably not increased in HIV infection, however, its clinical presentation tends to be more severe. The severity varies inversely with the underlying immune status and CD4 counts. A rapid onset of eruptive psoriasis can serve as a clue to underlying HIV infection [1,39]. Palmoplantar, flexural involvement and psoriatic arthritis is common in HIV psoriasis than in the general population of people with psoriasis [1, 39].

10. Pruritus, xerosis and ichthyosis

The incidence of xerosis has been found to be between 22.6% and 100% [3]. Workup of pruritus should include a careful examination of the skin, hair, nails, and mucous membranes to establish a primary dermatologic diagnosis. If no dermatologic cause is found, a systemic cause or medication-related etiology should be sought. Idiopathic HIV pruritus is a diagnosis of exclusion and should only be considered when a specific diagnosis cannot be established. Pruritus and xerosis are also side effects of Anti-retroviral drugs, especially the protease inhibitors [1].

11. Drug reactions

Patients with HIV infection are particularly prone to hypersensitivity drug eruptions.

Table. 3 enumerates various drugs implicated in erythema multiforme, Stevens–Johnson syndrome (SJS), toxic epidermal necrolysis (TEN), hypersensitivity syndrome, and drug reaction with eosinophilia and systemic symptoms (DRESS) syndrome in patients with HIV/AIDS [1].

Table 3: Drugs Implicated In Erythema Multiforme, SJS, TEN, DRESS Syndrome in HIV/AIDS

1.	Abacavir
2.	Allopurinol
3.	Amprenavir
4.	Carbamazepine
5.	Co-trimoxazole
6.	Efavirenz
7.	Fluconazole
8.	Griseofulvin
9.	Indinavir
10.	Nevirapine
11.	Nitrofurantoin
12.	Phenytoin
13.	Probenacid
14.	Pyrimethamine
15.	Saquinavir
16.	Streptomycin
17.	Sulfadiazine
18.	Sulfadoxine
19.	Thioacetazone
20.	Vancomycin
21.	Zidovudine
22.	Traditional Chinese medications

12. Neoplasms

The AIDS-defining malignancies are neoplasms that consistently correlate with the presence of AIDS in HIV-infected persons. Over the years, Kaposi sarcoma, squamous cell carcinoma of the uterine cervix and high grade non-Hodgkin's lymphomas have been listed as AIDS-defining malignancies. The non-AIDS-defining malignancies appear to occur at a much younger age in HIV-infected persons compared to those who are HIV negative, the neoplasms show atypical features, and a higher grade and stage at the time of diagnosis [39].

Kaposi sarcoma

Kaposi sarcoma is a multifocal, systemic tumor of endothelial origin. It has four clinical variants, enumerated in Table 4 [1,40]. Human herpes virus 8 (HHV8) is thought to be the initiating factor in the pathogenesis of KS [41]. It is transmitted sexually, more by faeco-oral route or the ejaculate than by blood, in HIV positive homosexual men. However, the predominant mode of HIV transmission in India is heterosexual and this might explain relatively low prevalence of KS in India [1,41].

It generally affects skin, mucous membranes, gastrointestinal tract, lymph nodes, and lungs. The oral mucosa is the initial site of localization in 10–20% of all HIV associated KS, frequently involving palate. The classical lesion in HIV is a purple patch, plaque or nodule, which may ulcerate.

Table 4. Clinical variants of Kaposi sarcoma in HIV positive patients

Variant	Risk Group	Median Survival
1. Classical sporadic KS	Elderly men of Eastern European or Mediterranean descent	Years or decades
2. African endemic KS	African children and adults	Months or years
3. Iatrogenic- immunosuppression or transplantation associated	Organ transplant recipients	Months or years
4. AIDS- related KS	Persons infected with HIV	Weeks or months

Lymphomas

The incidence of intermediate and high-grade B-cell non-Hodgkin's lymphomas in HIV-infected individuals is approximately 60 times greater than in the general population. Extranodal sites are usually involved, including bone marrow, Gastro Intestinal tract, and other sites that are unusual in non-HIV-associated non-Hodgkin lymphoma, such as the Central Nervous System (CNS) and body cavities (pleural, pericardial, peritoneal) [42].

Melanoma and non-melanoma skin cancer

AIDS patients have a three to five fold risk of developing a non-melanoma skin cancer. The ratio of squamous cell carcinoma to basal cell carcinoma in HIV is 1:7 as compared to 1.8:1 in renal transplant patients. Melanoma may present atypically and behave more aggressively. Squamous cell carcinoma may present at unusual sites like the nail folds, are multifocal with a high risk of metastasis and recurrence and thus results in a high mortality rate [1].

13. Oral manifestations

Oral manifestations of HIV disease are common and are among the first signs of HIV infection and immunosuppression. Oral lesions are important not only in early diagnosis but also in monitoring the progress of the disease.

The various oral lesions encountered in HIV include:

a. Oral candidiasis
b. Oral ulcerations
c. Oral hairy leucoplakia
d. Xerostomia
e. Melanotic hyperpigmentation
f. Salivary gland enlargement
g. Linear gingival erythema
h. Necrotising ulcerative gingivitis
i. Necrotising ulcerative periodontitis
j. Acute necrotising ulcerative gingivitis

Conclusion

Early recognition of the muco-cutaneous manifestations of HIV infection is an important challenge. Careful examination of the skin and mucosa may be highly rewarding in evaluating the stages of HIV disease. An increasing number of dermatoses in an HIV-infected individual point toward progression of HIV, and dermatological evaluation may detect prognostic indicators. By recognizing the spectrum of skin conditions associated with HIV infection and performing appropriate diagnostic tests, treatment can be administered in a timely fashion and outcomes optimized.

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If you have scintillating ideas in line with the goals and objectives of IISB, Please do share with us at **sexualityinfo@gmail.com** or write to us at **Indian Institute of Sexology Bhubaneswar,** Sanjita Maternity Care & Hospital, Plot No-1, Ekamra Marg, Unit-6, Bhubaneswar-751001, Odisha, India.