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Noninfectious Dermatoses Among HIV Patients: Clinical Descriptive Study

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Abstract

Introduction: Skin harbours specialized subsets of antigen-presenting dendritic cells, that take up microbial and tissue antigens, migrate to peripheral lymph nodes and present processed antigens to naïve T lymphocytes. The T lymphocytes are thereby induced to become activated and to expand in number, and T cells so activated acquire the capacity to migrate preferentially to skin, directed by specific homing receptors, where they exert their effector functions against relevant antigens *Methodology:* HIV positive patients attending Skin and STD Department and also patients referred from other departments of Hospital were screened for skin diseases by taking detailed history, clinical examination and relevant laboratory investigations. HIV positive patients having skin diseases were included in the study. *Results:* Out of 60 patients included in the study 15 patients (25%) had non-infectious dermatoses. Seven patients (11.7%) presented with pruritic papular eruptions, 4 (6.7%) had adverse cutaneous drug reactions, 2 (3.3%) had photodermatitis and only 1 case (1.7 %) of pityriasisrosea was seen. *Conclusion:* Photodermatitis was seen in 2 patients (3.3%) included in the study. Only 1 (1.7%) patient had pityriasis – rosea with typical clinical features.

Keywords: Noninfectious Dermatoses; PityriasisRosea; HIV.

Introduction

Dermatological involvement in AIDS has been appreciated since the disease was first recognized and even before the causative virus was identified. Mucocutaneous involvement establishes criteria for diagnosis and staging; the prognostic significance of some complications for example pruritic papular eruptions was well established before specific treatments were introduced. The proportion of patients with skin complications increases as HIV progresses and AIDS develops. The incidence and severity of several common cutaneous diseases (such as mollusca, herpes simplex, seborrhoeic dermatitis) are increased in patients with HIV and this correlates in many instances with the absolute numbers of CD4 T-helper cells. The skin can be affected by the immune reconstitution syndrome [1].

Although initially the immune deficit was thought to consist solely of depletion of CD 4 lymphocytes, it is now apparent that the effects HIV induces on the immune system are much more complex. Recently, subsets of T helper cells have been identified and found to play significant roles in HIV infection and AIDS. These two subsets are referred to as Th1 and Th2. Th1 cells promote cellular immunity and produce interleukin IL-2, IL-12, and interferon gamma. They also suppress the Th2 response and their effects are primarily concerned with eradication of infection. Predominant Th1 is the normal state in immunocompetent individuals. In contrast, Th2 lymphocytes promote humoral immunity and produce IL-4, IL-5 and IL-10. Th2 lymphocytes suppress the Th1 response and are associated with allergy. Patients with HIV infection initially have normal CD4 cell numbers, a low viral load, and a Th1-dominant immunologic milieu. With advanced infection, patients have low CD4 cell numbers, a high viral load, and a Th-2 dominant immune state. Therefore, many of the unusual cutaneous manifestations of HIV infection may be a result of the process developing in a Th2 setting. For example, vascular proliferative disorders, which are far more common in HIV infected hosts,

may develop as a consequence of an infection in a patient with predominant Th2 immunity. Kaposi's sarcoma (KS) is now known to develop as a consequence of widespread infection with Human Herpesvirus VIII (KS-HV) with vasoproliferation. Similarily, bacillary angiomatosis may be a manifestation of an unusual response to Bartonella infection in a Th2 milieu [2,3].

Syphilis may also present in unusual fashions when the immunologic milieu is predominantly Th2. Many HIV infected patients with syphilis have presentations and courses that are similar to those in immunocompetent hosts. These individuals most likely develop their disease at a point when their immune response is Th1 predominant. Later, in the setting of Th2, there may be unusual clinical, histologic, and serologic presentations [4].

There have been a number of different effects of HAART on HIV associated skin disorders including regression of KS, molluscumcontagiosum, and warts as well as improvement in a number of other cutaneous disorders. In addition, with true reconstitution of immunity, the need for prophylaxis is lessened and consequently, fewer secondary cutaneous complications ensue. Of interest, cutaneous manifestations that were noted to develop when CD4 cell counts declined to certain levels with advancing HIV infection have been noted to reappear at similar CD4 cell counts when the immune system of the host is being reconstituted.

Skin harbours specialized subsets of antigen-presenting dendritic cells, that take up microbial and tissue antigens, migrate to peripheral lymph nodes and present processed antigens to naïve T lymphocytes. The T lymphocytes are thereby induced to become activated and to expand in number, and T cells so activated acquire the capacity to migrate preferentially to skin, directed by specific homing receptors, where they exert their effector functions against relevant antigens. Impairment of the skin immune system, a well recognized consequence of pharmacological immunosuppression, leads to microbial and malignant invasion [5].

In addition to CD4 T-helper lymphocytes and monocytes, dendritic cells such as Langerhans cells were important targets in HIV-1 infection. Because antigen-presenting dendritic cells are essential for an effective immune response, abnormalities in their function are believed to be critical to the development of immunodeficiency. In addition to directly deleterious effects of HIV-1 on subsets of immune cells, it was shown that physical contact between HIV-1 pulsed dendritic cells and T cells during antigen presentation promotes massive HIV- 1 replication, followed by injury to both the T cells and the dendritic cells. These in-vitro data suggest that antigen presentation in-vivo likewise induces HIV-1 replication and results in T-cell death, leading to the progressive elimination of antigen-specific T lymphocytes from skin and lymph nodes [6].

Impairment of the skin immune system, which may be present early in the course of HIV-1 disease, is believed to be responsible for the frequent occurrence of both infectious and non-infectious skin diseases, even before the development of full immunodeficiency.

Methodology

HIV positive patients attending Skin and STD Department and also patients referred from other departments of Hospital were screened for skin diseases by taking detailed history, clinical examination and relevant laboratory investigations. HIV positive patients having skin diseases were included in the study. Except viral all other sexually transmitted diseases and lesions present over the mucous membranes were excluded from this study. The following investigations were done

Hemoglobin%, total leucocyte count, differential counts, erythrocyte sedimentation rate, random blood sugar, urea, creatinine, bilirubin and standard tests for syphilis.

Urine: Albumin, sugar and microscopy.

Before starting study proper, written/informed consent was taken from every patient included in the study.

Results

Table 1: Age distribution

Age group in years	No of cases	Percentage
15-24	08	13.3
25-34	18	30.0
35-44	23	38.3
45-54	08	13.3
55-64	03	5.0

In this study majority of the patients belonged to the age group between 35 – 44 years, the youngest patient was 15 year old and oldest was aged 56 years (Table 1).

Table 2: Gender

Sex Distribution	No of cases	Percentage
Male	39	65
Female	21	35

Out of sixty patients included in the study, 21 were females (35%) and 39 were males (65%). Male to female ratio was 1.9:1. (Table 2).

Table 3: Marital status

Marital status	No of cases	Percentage
Married	51	85
Unmarried	9	15

Among sixty patients included in the study, 51 patients (85%) were married and 9 (15%) were unmarried. (Table 3).

Table 4: Socio economic status

Socioeconomic status	No of cases	Percentage
Lower	50	83
Lower middle	10	16.7

Table 5: Non – infectious dermatoses

Non infectiousdermatoses	No of cases	Percentage
Pruritic papular eruptions	7	11.7
Adverse cutaneous drug reactions	4	6.7
Photodermatitis	2	3.3
Pityriasisrosea	1	1.7
Psoriasis	1	1.7
Total	15	25

Out of 60 patients included in the study 15 patients (25%) had non-infectious dermatoses. Seven patients (11.7%) presented with pruritic papular eruptions, 4 (6.7%) had adverse cutaneous drug reactions, 2 (3.3%) had photodermatitis and only 1 case (1.7 %) of pityriasisrosea was seen. (Table 4,5).

Table 6: Adverse cutaneous drug reactions

Type	No of cases	Percentage
Maculopapular	2	3.3
Toxic epidermal necrolysis	1	1.7
Erythroderma	1	1.7
Total	4	6.7

Four (6.7%) patients out of 60 included in this study presented with adverse cutaneous drug reactions. Two patients (3.3%) had developed maculopapular drug eruptions, 1 (1.7%) had drug induced erythroderma and 1 patient (1.7%) presented with toxic epidermal necrolysis. (Table 6).

Discussion

Adverse cutaneous drug reactions were seen in four patients (6.7%) out of sixty patients included in the study. Two patients had maculopapular drug eruptions, one had toxic epidermal necrolysis and one patient presented with erythroderma. All 4 patients were on multiple drugs, hence causative drugs could not be identified. Wang Jing et al reported an incidence of 2.1% for drug eruptions in their study [4,5]. An incidence of 14.8% for drug eruptions was reported by Rosatelli et al. [7].

Pre-existing psoriasis can worsen and become severe with widespread guttate lesions, plaques or pustules or become erythrodermic in HIV infected patients.Psoriasis may (5%) [24] or may not (1%) be more prevalent in HIV infected population. In our study, only one patient (1.7%) had psoriasis which had worsened with palmoplantar and flexural involvement.

Idiopathic photosensitivity is an uncommon phenomenon in HIV disease but may be the presenting complaint of advanced HIV disease. The most common type of photosensitivity in HIV disease are related to drug therapy [8]. Photodermatitis was seen in two patients (3.3%) in this study.

Pityriasisrosea is a papulosquamous disease of young adults with seasonal variations and limited time course. Kaplan et al. have reported an HIV positive patient with a PR like eruption [9]. Only one patient (1.7%) in our study had with pityriasisrosea with typical clinical features.

Eosinophilic folliculitis is an HIV specific disorder, it occurs at CD4 T-cell counts of 250-300x10^6 /L and therefore identifies patients at immediate risk of developing opportunistic infections. It may be part of the same spectrum as papular pruritic eruption of HIV. The cause is unknown but Th2 cytokines (IL-4, IL-5), RANTES and eotaxin are increased in lesional skin.

Eosinophilic folliculitis presents as a centripetal (face and trunk) eruption of pruritic, erythematous, perifollicular papules and pustules. It mimics staphylococcal or pityrosporum folliculitis and

acne vulgaris, with which it can coexist. Histology is characteristic, with degranulatingeosinophils and mast cells in a perifollicular distribution. There may be a peripheral eosinophilia and elevated levels of IgE. Swabs are negative: the lesions are sterile. Phototherapy is the most successful treatment modality. Eosinophilic folliculitis may be an indication for HAART.

Pruritic papular eruption (PPE) is a common cutaneous manifestation of HIV. The prevalence varying between 10 and 45% depending on geographical area. Insect bite hypersensitivity, as in papularurticaria is a speculative pathomechanism.

PPE is a sign of an advanced degree of immunosuppression, occurring at CD4 T-cell counts below 100-200x10^6 /L and may often be the first sign of HIV. PPE presents as excoriated ,erythematous,urticarial papules associated with eosinophilia and elevated IgE. The differential diagnosis includes papularurticaria and eosinophilic folliculitis: it is possible that eosinophilic folliculitis and PPE are part of the same spectrum of disease.

The treatment of PPE is similar to that of eosinophilic folliculitis, with phototherapy the linchpin [3].

As many as 30% of HIV infected individuals experience xerosis or acquired ichthyosis [10]. Eczema is an inflammatory skin condition more common in children that may be exacerbated by dry skin. While both conditions are common in the general population, HIV infected individuals often exhibit severe or unremitting disease.

Fine white scales and cracking skin without erythema typify xerosis, which may be diffuse or preferentially affect the anterior tibia, dorsal hand, and forearm. Ichthyosis is a more severe disorder involving skin thickening and fish like scales. Atopic dermatitis is characterized by erythematous scaling plaques with associated papules or vesicles. Patients may demonstrate the triad of allergic rhinitis, asthma, and eczema. HIV infected individuals often suffer severe disease that may progress to erythroderma. Both conditions are associated with pruritus that can lead to secondary skin changes such as lichenification and excoriations with superimposed bacterial infection.

Both xerosis and eczema benefit from emollients, topical steroids, avoidance of irritants, and antihistamines to relieve pruritus. Acquired ichthyosis may also respond to topical keratolytic agents [10].

Atopic dermatitis is manifest by erythematous patches and plaques with fine papulovesicles associated with scaling, crusting, and lichen simplex chronicus. Patients often have associated hyperlinear palms, allergic rhinitis, and asthma. HIV- infected individuals who develop atopic dermatitis may manifest severe forms of the disorder with erythroderma. Atopic dermatitis has microscopic features of a superficial perivascular infiltrate of lymphocytes and eosinophils with epidermal hyperplasia and foci of spongiosis. Late lesions have a morphology that is primarily that of lichen simplex chronicus.

Conclusion

- Adverse cutaneous drug reactions were seen in 4 patients (6.7%). Two of these had maculopapular drug eruptions, one had toxic epidermal necrolysis and one patient presented with drug induced erythroderma.
- Only one patient (1.7%) had severe form of psoriasis with palmoplantar and flexural involvement.

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