Sequential development of psoriasis, alopecia universalis, and vitiligo vulgaris in a human immunodeficiency virus seropositive patient: A unique case report

Sir,

We report a Person living with human immunodeficiency virus (HIV)/AIDS developing psoriasis, vitiligo vulgaris, and alopecia universalis in a sequential manner. Although, psoriasis and psoriatic arthritis have been classically associated with HIV, its association with, vitiligo vulgaris and alopecia is rare. Vitiligo and alopecia areata (AA) are considered to be T-cell mediated autoimmune disorders their paradoxical coexistence in the setting of immunocompromised host is intriguing.

A 60-year-old male patient was diagnosed HIV positive in April 2010, while screening as a preoperative work up for hernia surgery. All the relevant baseline investigations such as complete blood count, liver function test, renal function test, lipid profile, and urine routine were within normal range.

Base line CD4 count was done and 561 cells/cmm. As the patient was asymptomatic and CD4 count
was 561, [Figure 1] antiretroviral therapy (ART) was not started.

Patient developed early skin lesions in January 2010. Initially, patient had scalp scaling for few months, which was followed by typical annular scaly patches with silvery white scales over back, sacroiliac region, and extensor aspects of limbs. Patient was diagnosed as psoriasis vulgaris in July 2010.

In the month of November 2010 patient developed patches of AA on the temporal scalp which spread rapidly to involve entire scalp, eyebrows, eyelashes, axillary, pubic hair were lost rapidly and within a period of 10 days part landed in alopecia universalis [Figures 2 and 3]. Patient also had trachyonychia or rough nails.

Within few months in January 2011 patient developed vitiligo vulgaris over dorsum of hand and face, which spread rapidly to involve scalp, face trunk, upper limb, and lower limb [Figure 4]. Patient landed in generalized vitiligo by March 2011.

Subsequent CD4 counts of the patient were 392/cmm on 7/1/12 and 611/cmm on 14/7/11. Patient is on regular follow-up until date and has not developed any other dermatological or systemic illness, still ART naive.

The cutaneous presentations of HIV can be infections, noninfectious, inflammatory and neoplasms. In noninfectious manifestations seborrheic dermatitis, psoriasis vulgaris, Reiter's syndrome, xerosis and acquired ichthyosis, papular pruritic eruption of HIV, etc., are seen frequently.

Other dermatologic manifestations have been associated primarily with HIV-1 infection. Photodermatitis,[1] vitiligo and other pigmented alterations of the skin,[2] porphyria cutanea tarda,[3] granuloma anulare,[4] pityriasis rubra pilaris,[5] pemphigus vulgaris and many other autoimmune reactions[6] have been reported, but a clear association between the pathogenesis of each of these disorders and the retrovirus has not yet been established.

It has been well-established that psoriasis, psoriatic arthritis, and Reiter's syndrome can occur in patients with HIV infection. These arthocutaneous diseases tend to occur in temporal proximity to the development of AIDS and AIDS related complex, and their clinical manifestations are unusually
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Severe. The appearance or exacerbation of psoriasis, arthritis, or Reiter’s syndrome in a high-risk person should alert the clinician to possible underlying HIV infection.[7]

In fact, in patients with known risk for HIV exposure, new onset of psoriasis may sometimes be a marker of HIV infection.[8]

Persistent viral infections have been postulated to be trigger factors for the development of autoimmune disease. There have been reports of development of vitiligo in patients with HIV.[9] Vitiligo may be an example of an autoimmune disease triggered by viral infection in a genetically predisposed host.

Alopecia areata is thought to be a tissue-specific autoimmune disease, which occurs most probably, following a breakdown of the hair follicles immune privilege. Genetic susceptibility, role of human leukocyte antigen, autoimmune associations, endocrine and psychogenic factors, role of cytokines including tumor necrosis factor-α, interferon-γ have been implicated in the pathogenesis of AA.[5] Role of infectious agents including viruses have been proposed but not proven.[9] The most consistent histological feature of AA is a perifollicular lymphocytic infiltrate comprised primarily of CD4+ cells associated with a CD8+ intrafollicular infiltrate, as well as a T-helper 1 cytokine profile.[10,11]

Human immunodeficiency virus induced immune dysregulation and altered CD4/CD8 ratio appears to be most plausible explanation for occurrence of AA, vitiligo and psoriasis in HIV.

All the primary dermatologic complications in HIV infected patients are also seen in immunocompetent patients. Conditions such as atopic dermatitis, psoriasis, and seborrheic dermatitis, AA, vitiligo are extremely common dermatologic problems expressed in the general population and are seen in HIV seropositive individuals in extensive and unusual forms. However, the direct role of the HIV virus in the pathogenesis of these manifestations is still to be discovered. Better animal models, which may include humanized rodents, might represent a more suitable approach for the study of the pathogenesis of HIV related disorders and the development of more effective forms of treatment.

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