

## Case Report:

### Silent HIV infection masquerading behind psoriasis

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#### ABSTRACT

Psoriasis is a chronic inflammatory autoimmune skin disease characterized by immune-mediated cutaneous inflammation and keratinocyte hyperproliferation. Psoriasis can be the early presenting cutaneous manifestation in patients with human immunodeficiency virus (HIV) infection, acquired immunodeficiency syndrome. We report three patients presenting with severe extensive cutaneous psoriatic lesions, psoriatic arthritis, cachexia, oesophagitis, “Tambaram tongue” in whom initial serological testing for HIV was negative, but Western blot test confirmed the diagnosis of HIV infection.

**Key words:** Human immunodeficiency virus infection, psoriasis

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#### INTRODUCTION

Psoriasis is a chronic papulosquamous disorder that is commonly seen on an everyday basis in almost all dermatology clinics in India. The human immunodeficiency virus (HIV) infection has emerged as a major health problem that is being seen across all fields of medicine. Compared to immunocompetent individuals, atypical clinical presentation of these various infectious and inflammatory conditions is more frequently evident in HIV-seropositive individuals.

Fulminant cutaneous manifestations are common in patients with HIV/AIDS. Psoriasis can be the presenting cutaneous manifestation in a HIV infected individual and sometimes is found to be the early manifestation in patients having advanced HIV infection, acquired immunodeficiency syndrome (AIDS).<sup>1</sup> In this report, we describe three patients presenting

with severe exacerbation of psoriatic lesions in whom initial serological testing for HIV was negative, but Western blot test confirmed the diagnosis of HIV infection.

#### CASE REPORTS

During 2015, three patients had presented to the Dermatology out-patient service at our medical college teaching hospital at Moradabad with severe fulminant cutaneous psoriatic lesions. All three patients were males in their fourth decade of life. All of them gave a history of promiscuity and intravenous drug abuse. They had also complained of excruciating retrosternal pain, stabbing in character, that worsened on food intake, nausea and retching and were not able to eat or even drink any fluid suggestive of oesophagitis. Salient physical examination findings are shown in (Figures 1, 2, and 3). All three patients were diagnosed to have extensive fulminant cutaneous psoriasis.

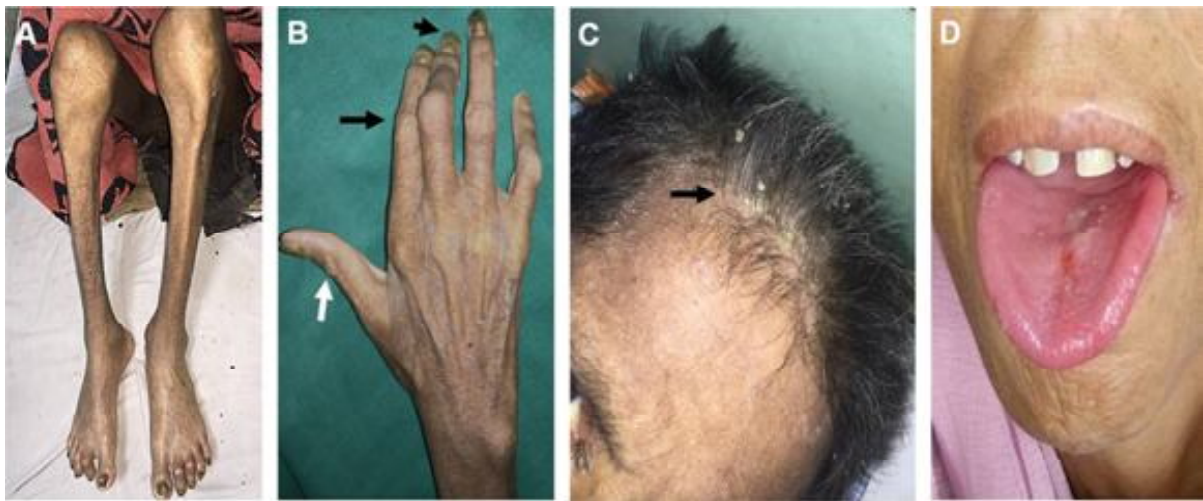
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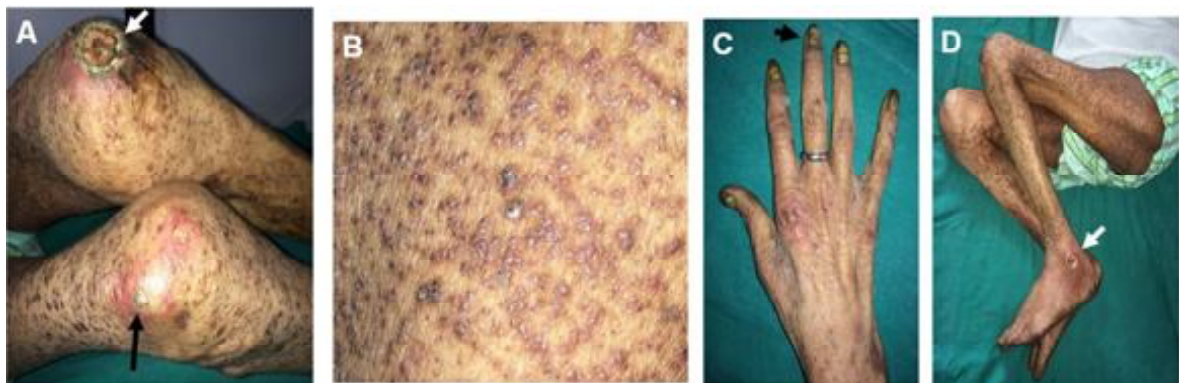
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**Figure 1:** Clinical photograph of patient #1 showing HIV-associated cachexia (A); onychomycosis (arrow head), psoriatic arthritis (black arrow) and psoriatic pustule (white arrow) (B); hairline involvement (C); and angular cheilitis, magenta coloured tongue showing macroglossia, deep furrowing, loss of papillae, candidiasis suggestive of the classical “Tambaram tongue” (D)

HIV = human immunodeficiency virus



**Figure 2:** Clinical photograph of patient #2 showing psoriatic arthropathy, ulceration (white arrow), pustular psoriasis (black arrow) (A); extensive cutaneous lesions (B); onychomycosis (arrow head) (C); HIV-associated cachexia, psoriatic arthropathy, ulceration (white arrow) (D)

HIV = human immunodeficiency virus



**Figure 3:** Clinical photograph of patient #3 showing lesions of pustular psoriasis

**Table 1: Body mass index and laboratory findings**

Patient	BMI (kg/m <sup>2</sup> )	Haemoglobin (g/dL)	CD3+ count (/mm <sup>3</sup> ) (Normal = 716-2130 cells/mm <sup>3</sup> )	CD4+ count (/mm <sup>3</sup> ) (Normal = 500-1500 cells/mm <sup>3</sup> )	CD8+ count (/mm <sup>3</sup> ) (Normal = 218-1396 cells/mm <sup>3</sup> )	Serum albumin (g/dL)	Serum potassium (mEq/L)
#1	14	10.1	310	180	234	3.0	3.0
#2	17	10.5	325	116	214	3.0	3.2
#3	13	4.0	711	399	251	2.9	3.0

BMI = body mass index

Due to the extensive fulminant nature of dermatological lesions the patients were referred to Integrated Counselling and Testing Centre (ICTC) for serological testing for HIV infection as per National AIDS Control Organization (NACO) guidelines.<sup>2</sup> All the three patients had tested negative. As the clinical suspicion of HIV infection was very high, we then referred all the three patients to New Delhi for further testing. Out of the three, only two patients managed to get themselves tested at New Delhi with Western Blot test. Both these patients tested positive for HIV and were initiated on antiretroviral treatment (ART). Viral load in the these two patients was more than 5000 copies/mL.

The third patient succumbed to his illness before further testing could be performed. Before the initiation of specific treatment for psoriasis could be undertaken, both the patients were lost to follow-up.

### DISCUSSION

Eventhough there is no consensus regarding the definition of HIV-associated psoriasis, certain traits distinguish it from psoriasis seen in HIV-seronegative immunocompetent individuals. These include, sudden onset, severe, extensive acral, involvement and recalcitrant nature. Most of the autoimmune diseases are driven by a Th1 cytokine profile and the same can be seen in

the case of Psoriasis. Interferon- $\gamma$  (IFN- $\gamma$ ) along with interleukin-17 (IL-17) orchestrates the initiation of the inflammatory reactions underlying the cascade of pathogenesis of psoriasis ultimately leading to the various histopathological changes seen in a psoriatic lesion.<sup>3</sup> A theory postulated to explain the appearance of Psoriasis for the first time in previously non-psoriatic patients involves the surface protein gp120 of the HIV. It was postulated that gp120 acts as a superantigen the same way as the infective organisms as mentioned above leading to keratinocyte expression of HLA-DR but this theory requires further research as no concrete evidence is available to back this hypothesis.<sup>3,4</sup> HIV-associated psoriasis is more severe and this is thought to be because of weakened immunosuppression. Risk of psoriasis has been observed to be nine-fold greater in HIV-seropositive persons with a CD4+ T-lymphocyte count less than 200/mm<sup>3</sup>.<sup>5,6</sup> Thus, HIV-associated psoriasis is considered to be paradoxical as well as a marker of immune suppression.

In comparison to immunocompetent individuals, psoriasis in patients with HIV responds poorly to treatment and is associated with a higher rate of morbidity. The specific treatment of HIV-associated psoriasis depends on the severity of the disease. In mild cases

where less than 2% body surface area (BSA) is involved, topical treatment with emollients, corticosteroids, tar, vitamin D analogues, and retinoids are used. In moderately severe (2%-10% BSA involved) and severe disease (> 10% of BSA involved) systemic therapies, such as phototherapy, acitretin, cyclosporin, hydroxyurea, methotrexate and tumour necrosis factor- $\alpha$  inhibitors (i.e., etanercept, infliximab) have been used.<sup>1</sup>

Thus, it is important to remember that with HIV in the background, there will be exacerbation of the already existing cutaneous lesion which will become fulminant within a short duration of time. The clinical manifestations will be atypical in such patients resulting in a delayed diagnosis. Psoriasis being one of the early dermatological manifestations, any patient presenting with extensive fulminant cutaneous psoriatic lesions should be tested for HIV infection. Further, the presence of severe cachexia, "Tamaram tongue" were valuable clinical clues to underlying HIV infection. Accordingly, we had subjected our patients to serological testing for HIV infection; however, all three patients had tested negative. At this point in time, we had considered false negative serological test result, idiopathic lymphocytopenia as the differential diagnosis and had referred the patients for Western blot

test which yielded positive results confirming the diagnosis of HIV infection in them. Clinicians should be aware that, in these patients, due to the autoimmune nature of psoriasis, serological test results can be falsely negative and further diagnostic testing with Western blot is helpful in confirming the diagnosis of HIV infection.

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