

Psoriasis in HIV patients: A paradox

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Abstract Human immunodeficiency virus (HIV) infection is associated with increased susceptibility to skin disorders, including psoriasis. The exacerbation of psoriasis in severe advanced HIV is paradoxical. A 26-year-old man with HIV who had been diagnosed with psoriasis ten months before the diagnosis of HIV. The skin biopsy results indicated psoriasis vulgaris with a tissue CD4/CD8 ratio of 0/34. The initial CD4 count was 130 cells/L. The patient received ARV and topical steroids. There was a clinically significant rise in blood CD4 and tissue CD4/CD8 ratio in three-month follow-up. There are several hypotheses proposed to explain the pathophysiology of psoriasis in HIV. The main therapy of psoriasis with HIV is ARV. HIV can induce or as a risk factor of psoriasis. It's important to do a search further for risk factors in sexually transmitted diseases in patients with severe and recalcitrant psoriasis. The main therapy of psoriasis with HIV is ARV.

Key words

Psoriasis, HIV, paradoxical exacerbation, ARV.

Introduction

Human immunodeficiency virus (HIV) infection is a global epidemic and is associated with increased susceptibility to mucocutaneous disorders including psoriasis. The clinical course may be similar or different to those without HIV.¹ The reported prevalence of psoriasis in HIV patients is from 1.3-5 percent.^{1,2}

Case report

A 26-year-old male with HIV came with the chief complaint of red scaly patches on his chest and legs. He is a known case of psoriasis vulgaris for the last ten months with a psoriasis area score and severity index (PASI) of 17.9.

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The patient's HIV status was unknown at that time of psoriasis diagnosis. He was treated with methotrexate (MTX) and topical corticosteroids. The patient had routine follow-up for the next five months before he discontinued the treatment due to no improvement in the lesions.

Ten months after diagnosis of psoriasis, He was diagnosed with HIV and started antiretroviral therapy (ARV) fixed drug combination (FDC) of tenofovir 300, lamivudine 300, efavirenz 600 (THE), and cotrimoxazole 1x960mg, with CD4 of 130 cells/mm³ at the time of diagnosis. Two weeks after being diagnosed with HIV, red scaly patches on the chest and legs had thickened and became an itch.

There were well-defined, erythematous to hyperpigmented plaques that varied in size, multiple, and scattered covered white scales over the chest, upper abdomen, back, upper arms, and legs showed. The patient was diagnosed with a case of psoriasis with a PASI score of 16.8. Histopathological features were consistent with



Figure 1 26-year-old man with psoriasis (A) First Admission (B) After use of MTX 5 months (HIV status was unknown).

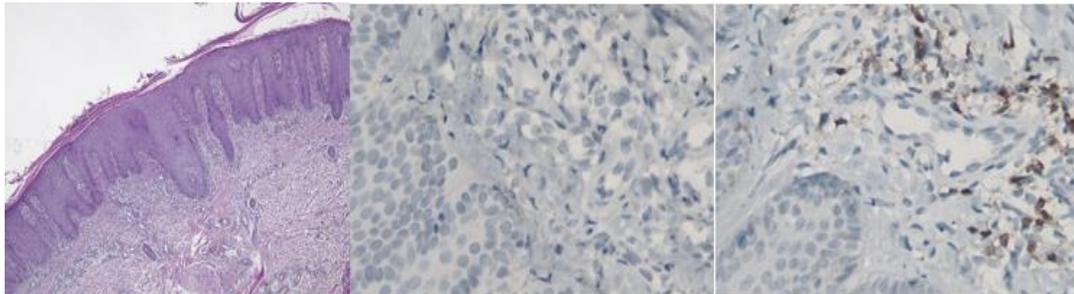


Figure 2 (A) Histological examination (B) CD4 Stain (C) CD8 stain.

psoriasis vulgaris while the tissue CD4/CD8 ratio was 0/34. The patient was diagnosed with psoriasis vulgaris in HIV and was offered phototherapy, which he denied. He was given a topical combination while continued the ARVs regimen. The lesions improved with the PASI score of 75 over the three-month follow-up visit. A repeat biopsy revealed histopathological features were consistent with post-inflammatory hyperpigmentation and might support the diagnosis of psoriasis in remission. Blood CD4 count increased to 218 cells/mm³ while CD4/CD8 ratio increased to 1/5. He continued ARV and topical therapy.

Discussion

HIV infection is considered a risk factor for psoriasis and causes significant exacerbations in

people who had psoriasis before HIV infection.^{2,3} More than 80% of HIV-positive psoriasis patients develop active psoriatic lesions when their CD4 cell count reaches 450 cells/mm³.^{3,4} In our case, when the patient was diagnosed with severe psoriasis vulgaris, his HIV status was still unknown. The patient was diagnosed with HIV six months after the diagnosis of psoriasis, with an initial CD4 cell count of 130 cells/mm³. The CASCADE study, including 2,820 HIV-positive individuals, discovered an average CD4 cell count reduction of 61 cells/L each year.⁵ This low CD4 cell count finding at the time of diagnosis raised suspicion of prolonged undiagnosed HIV infection. HIV-associated psoriasis appears contradictory considering psoriasis is an inflammatory disease associated with increased

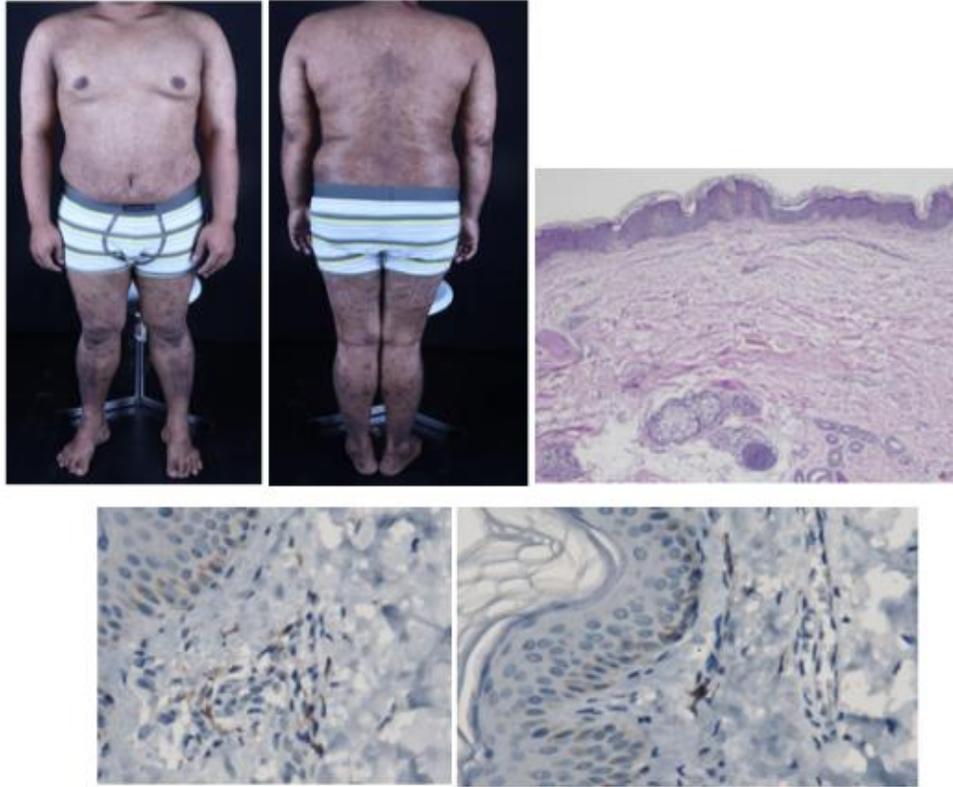


Figure 3 Follow up after 12 weeks of therapy (A). Clinical manifestation (B) HE staining (C) CD4 staining (D) CD8 staining.

T cell activation, while HIV is related to a reduction in the number of T cells.¹ Typically, HIV infection targets and destroys memory CD4 cells and naïve CD8 T cells. The progression of HIV induces depletion of naïve CD8 cells, resulting in a relative increase of the memory CD8 cell population to more than 85 percent of the total CD8 count in HIV patients. The ratio of naïve to memory CD8 cells in the non-HIV population was 50 percent. This creates an imbalance in the CD4/CD8 ratio, which disrupts homeostasis and induces the onset of psoriasis. The CD8 cell count and the subset of memory CD8 cells are found to be increased in the epidermis and dermis of the affected skin compared to non-lesional skin. It is similar to that seen in classic psoriasis without HIV.^{2,6,7} Another distinction is that psoriasis is generally mediated by type 1 cytokines, whereas HIV is typically mediated by type 2 cytokines. Further studies on the disposition of the T cell subset,

cytokine profile, and antigen presentation in HIV and psoriasis may shed light on this paradox.^{3,5}

Antiretroviral therapy is the first-line recommendation for HIV patients concurrent with mild to severe psoriasis. The first-line treatment for moderate to severe psoriasis was a combination of ARVs and ultraviolet (UV) radiation.^{6,7} MTX and topical therapy was not effective in our patient, however, a combination of ARVs and topical agents had significant therapeutic benefits. This further supported that psoriasis in this patient began with a low CD4 count/ HIV so that immunosuppressive therapy without ARV did not result in significant improvement. A follow-up visit after three months showed satisfactory results with PASI score of 75, and a repeat biopsy revealed improvement in psoriasis lesions and increased the CD4/CD8 ratio to 1/5.

Conclusion

HIV infection in this patient might exist long before the diagnosis of psoriasis and was a risk factor for the severity of psoriasis since we found a low CD4 cell count at the time of HIV diagnosis. The physicians should ask regarding the sexually transmitted infections and HIV if finding cases of severe psoriasis which recalcitrant to therapy. The main treatment for psoriasis in HIV is antiretroviral therapy.

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