A Case of Ostraceous Psoriasis with Psoriatic Arthritis in an AIDS Patient

Minkee Park, Myeong Jin Park, Mi Soo Choi, Chan Hee Nam, Byung Cheol Park, Seung Phil Hong, Myung Hwa Kim

Abstract
Human immunodeficiency virus (HIV) infection may present with severe and atypical cutaneous diseases. Psoriasis also can develop in HIV patients associated with immune dysfunction and be presented as more severe and atypical manifestation. Furthermore, treatment of psoriasis in HIV patients can be complex and challenging. Herein, we report the case of a 50-year-old male with a 9-year history of HIV infection who developed an uncommon clinical variant of psoriasis with psoriatic arthritis and we discuss a possible pathogenesis of this autoimmune disease and possible treatment.

Key Words: AIDS, human immunodeficiency virus, psoriasis, psoriatic arthritis

Introduction
The immune dysfunction associated with human immunodeficiency virus (HIV) results in several cutaneous disorders. Dermatological disorders such as psoriasis can be the presenting feature of HIV infection and can provide clinical clues to the degree of immune dysfunction. Psoriasis is a chronic inflammatory skin disorder of presumed autoimmune origin found in 2% of the general population. Although the prevalence of HIV-associated psoriasis is similar to that of general population, psoriasis in HIV patients tends to be more severe, extensive, recalcitrant, and atypical. We herein report a patient with severe uncommon presentation of ostraceous type of psoriasis along with psoriatic arthritis.

Case Report
A 50-year-old male with a history of 9 years of HIV presented to our clinic with thick scaly hyperkeratotic plaques on the distal part of extremities and face. After 1 year of highly active antiretroviral therapy (HAART), he was not treated for 8 years. Physical examination showed diffuse areas of greasy scales on face and scalp [Figure 1a]. The patient's extremities revealed firmly adherent thick scales [Figures 1b-d]. He complained of swelling of hands and feet, arthralgia, walking difficulty, and poor oral intake. Laboratory data revealed C-reactive protein – 8.23 mg/dL, hemoglobin – 9.0 g/dL, and HLA B27 positive. T lymphocyte subsets revealed that CD4+ T-cell count was 66 cell/μL, CD4/CD8 ratio was decreased to 0.19, and HIV RNA-titer was 224,701 copies/mL. Rheumatologist confirmed psoriatic arthritis on both shoulders, wrists, and left knee. Skin biopsy showed spongiform pustule of Kogoj formed by collections of neutrophils which was consistent with psoriasis [Figure 2].

With the biopsy report, the patient was diagnosed as ostraceous psoriasis with psoriatic arthritis. Treatment was initiated with oral acitretin, low-dose steroid, topical calcipotriol with betamethasone, and reinitiation of HAART therapy. After 3 weeks of treatment, there was marked improvement in skin lesions [Figure 3a and b], arthralgia, and walking difficulty.

Discussion
Skin manifestation in HIV/AIDS patients is complex and ranges widely. Although psoriasis may develop at any stage of HIV infection, the severity of psoriasis tends to correlate with worsening immune function. All clinical variants of psoriasis may manifest in HIV/AIDS patients, with guttate, inverse, and erythrodermic types being more common and severe. Often, more than one form of psoriasis may coexist. In addition, the prevalence of psoriatic arthritis is much higher.
Psoriasis may present with intensely hyperkeratotic lesions, as in this case, thus being classified as ostraceous, rupioid, and elephantine psoriasis. Lesions with firmly adherent thick scales, varying color, and surface resembling an oyster shell are typical features of ostraceous psoriasis.

Psoriasis is thought to be a chronic inflammatory papulosquamous skin disorder of T-cell-mediated keratinocyte proliferation. Although psoriasis can present throughout the range of immunodeficiency, it tends typically to present late with increasing immunodysfunction, and CD4-cell counts are <100 cells/µL. The pathogenesis of psoriasis in patients with HIV is considered as a medical paradox that revolves around three main quandaries. First, this T-cell-mediated disease manages to flourish in an environment of decreasing T-cell counts. Second, although various therapies targeting T lymphocytes are effective in psoriasis, the condition worsens with decreasing CD4 T-cell counts in patients with HIV. Third, HIV is characterized by a strong Th2 cytokine profile and psoriasis is characterized by a strong Th1 secretion pattern. Recent theories on the effects of the HIV virus on T-cell populations have begun to explain how an imbalance in the CD4:CD8 ratio can be responsible for the immune dysregulation in HIV-associated psoriasis. The majority of studies have shown that the virus preferentially infect memory CD4 T-cells and naive CD8 T-cells. As HIV progresses and naive CD8 T-cells become depleted, there is a disproportional relative expansion of the CD8 memory T-cell population that comprises >85% of the total CD8 T-cell count in patients with HIV in contrast to 50% in healthy controls. The overall decrease in naive CD8 T cells not only diminishes the ability to fight off new infections but also allows autoimmune diseases such as psoriasis to become established. In patients with HIV without psoriasis, the cytokine profile is characterized by a strong propensity of Th2 cytokines, especially IL-4 to IL-6 and IL-10 as the HIV infection progresses. However, the cytokine pattern found in psoriatic patients with HIV is not characterized by a clean shift in cytokines to a complete Th2 profile. Instead, due to the increased subpopulation of memory T cells, there is a distinctive increase in the production of IFN-γ, the cytokine most responsible for creating and maintaining psoriatic phenotype.

HIV RNA transcripts have been identified in the skin of patients with HIV-associated psoriasis and within CD4 Factor XIIIa + dermal dendritic cells, which suggest a direct role of HIV and is compatible with worsening disease with the higher viral loads associated with progressive immunodeficiency. HIV might directly trigger psoriasis as a costimulatory factor through antigenic presentation or as a source of superantigens. Management of HIV-associated psoriasis is challenging, as the clinical course is progressive and often refractory to conventional therapies. Many of the systemic treatments for psoriasis are immunosuppressive and can potentially lead to severe complications, such as progression to AIDS and development of opportunistic infections. Topical therapies, such as emollients, corticosteroids, retinoids, and vitamin D analogs, are recommended as first-line treatment for mild-to-moderate disease. For moderate-to-severe disease, phototherapy, oral retinoids, and antiretrovirals are recommended in addition to topical therapies. For more refractory
or severe disease, the use of immunosuppressants or biologic agents is cautiously recommended; these include cyclosporine, methotrexate, and tumor necrosis factor-alpha inhibitors.\(^9\)

In this case, we observed dramatic improvement of psoriatic skin lesions and psoriatic arthritis after 3 weeks of treatment including oral retinoids, low-dose steroid, and HAART therapy. Since we have performed the treatment of HIV and psoriasis at the same time, it is not clear which treatment mainly attributed to this improvement. We present the uncommon presentation of ostraceous psoriasis with psoriatic arthritis as the features of HIV infection. This case highlights the importance of recognizing the unusual serious manifestation of psoriasis associated with HIV.

**Declaration of patient consent**

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

**Financial support and sponsorship**

Nil.

**Conflicts of interest**

There are no conflicts of interest.

**References**

1. Morar N, Willis-Owen SA, Maurer T, Bunker CB. HIV-associated psoriasis: Pathogenesis, clinical features, and management. Lancet Infect Dis 2010;10:470-8.
2. Cedeno-Laurent F, Gómez-Flores M, Mendez N, Aner-Rodríguez J, Bryant JL, Gaspari AA, et al. New insights into HIV-1-primary skin disorders. J Int AIDS Soc 2011;14:5.
3. Patel RV, Weinberg JM. Psoriasis in the patient with human immunodeficiency virus, part 1: Review of pathogenesis. Cutis 2008;82:117-22.
4. Castillo RL, Racaza GZ, Roa FD. Ostraceous and inverse psoriasis with psoriatic arthritis as the presenting features of advanced HIV infection. Singapore Med J 2014;55:e60-3.
5. Mesquita Lde S, Sherlock J, Portugal FM, Mota Lde S, Fakhouri R, Silva SF, et al. Case for diagnosis. An Bras Dermatol 2014;89:841-2.
6. Montazeri A, Kanitakis J, Bazex J. Psoriasis and HIV infection. Int J Dermatol 1996;35:475-9.
7. Mahoney SE, Duvic M, Nickoloff BJ, Minshall M, Smith LC, Griffiths CE, et al. Human immunodeficiency virus (HIV) transcripts identified in HIV-related psoriasis and Kaposi’s sarcoma lesions. J Clin Invest 1991;88:174-85.
8. Mendoza N, Yang B, Patablanda K, Reusser NM. Psoriasis and HIV: Rupioid psoriasis, an uncommon presentation. J Dermatol Clin Res 2015;3:1043.
9. Menon K, Van Voorhees AS, Bebo BF Jr., Gladman DD, Hsu S, Kalb RE, et al. Psoriasis in patients with HIV infection: From the medical board of the national psoriasis foundation. J Am Acad Dermatol 2010;62:291-9.