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Original Research Paper

Nephrology

THE PARADOX OF PSORIASIS FLARE POST-ART CESSATION: A CASE REPORT

Samrawit Zinabu	Department Of Internal Medicine, Howard University, Washington Dc, Usa
Arsema Zadu	Department Of Dermatology, Howard University, Washington Dc, Usa
Ahmad Mohammed	Department Of Internal Medicine, Howard University, Washington Dc, Usa
Jamon J. Thomas	Department Of Dermatology, Howard University, Washington Dc, Usa
Bezawit Gebre	Department Of Dermatology, Addis Ababa University, Addis Ababa, Ethiopia
Zelalem Kenea	Department Of Internal Medicine, St. John, Medical Center, Cleveland, Ohio
Yemisrach Abawaji	Department Of Dermatology, Menelik Hospital, Addis Ababa, Ethiopia
Huda Gasmelseed	Department Of Internal Medicine, Howard University, Washington Dc, Usa
Miriam Michael*	Department Of Internal Medicine, Howard University, Washington Dc, Usa Department University Of Maryland Rehabilitation And Orthopedic Institution Baltimore, Md, Usa *Corresponding Author

ABSTRACT Psoriasis is a chronic inflammatory skin disease characterized by papulosquamous lesions resulting from T-cell-mediated dysregulation of keratinocyte proliferation. It can be triggered by HIV infection and may be the first sign of HIV in some individuals. Treating HIV-associated psoriasis is challenging because it is a T-cell-mediated disease that occurs in patients with weakened immune systems. We present a case of an HIV-positive patient with well-controlled psoriasis on HAART who experienced relapse upon discontinuation of therapy, emphasizing the importance of finding a balance between treating psoriasis and preserving immune function.

KEYWORDS : Psoriasis, HIV, HAART, immunosuppression, papulosquamous lesions

INTRODUCTION

A distinct relationship exists between psoriasis and significant immunosuppression in patients with HIV infection, although the prevalence of HIV in psoriatic patients is statistically similar to that of the general population. HIV itself doesn't typically cause psoriasis, but the condition can be more aggressive in individuals with both diagnoses (1). Psoriasis falls under the category of papulosquamous disorders, which are marked by scaly papules and plaques (2). It is an immune-mediated disorder caused by both genetic and environmental factors. It affects approximately 3% of the US adult population. Various environmental factors can trigger or worsen psoriasis in genetically susceptible individuals. These factors encompass infections, trauma, psychological stress, smoking, alcohol consumption, and certain medications (3).

Psoriasis is characterized by inflammation that leads to uncontrolled keratinocyte proliferation and dysfunctional differentiation. Microscopic examination of the psoriatic plaque shows epidermal hyperplasia, with underlying inflammatory infiltrates composed of dermal dendritic cells, macrophages, T cells, and neutrophils. Antimicrobial peptides (AMPs), tumor necrosis factor (TNF), IL-23, Th17, IL-17, IL-22, and signal transducer and activator of transcription (STAT)3, contribute substantially to its pathogenesis (4).

The pathogenesis associating psoriasis with HIV is paradoxical and explained by the profound immune dysregulation between immune cellular components (1). It is paradoxical that although drugs targeting T lymphocytes effectively treat psoriasis, the condition can worsen with HIV infection. Psoriasis exhibits a Th1 cytokine profile, indicating excessive T cell activity and inflammation. Conversely, HIV infection is characterized by Th2 cytokine dominance alongside T cell depletion, leading to heightened susceptibility to opportunistic infections (1).

Psoriasis worsens with lower CD4 counts; this suggests that despite being a T-cell-mediated disease, psoriasis severity might be linked to a specific T-cell imbalance rather than overall T-cell levels. Understanding the involvement of T-cell subsets, autoimmunity, genetic predisposition, and infections related to immune dysregulation can elucidate the interplay between psoriasis and HIV (5). Although it is not a characteristic presentation of acquired immunodeficiency syndrome (AIDS), the presentation of psoriasis can have a higher incidence, atypical presentation, and pronounced clinical features. Clinically psoriasis is present in various forms such as plaque, flexural, guttate, pustular, or erythrodermic (6). The most common form is plaque psoriasis, characterized by well-demarcated, salmon-pink plaques with a silvery-white scale (6). It typically appears symmetrically on the extensor surfaces. Managing HIV-associated psoriasis is challenging, though patients generally improve with highly active antiretroviral therapy and retinoids (7).

Case Presentation

The patient is a 41-year-old female with a past medical history of HIV, noncompliance with her medications, and end-stage renal disease on dialysis, presented with a generalized skin lesion of two months' duration. She had a chronic psoriasis rash well-controlled after starting HAART five years ago. She reported that the lesions worsened after she stopped her HIV medication (Bictegravir-Emtricitabine-Tenofovir) two months ago. She had no pruritis. She denied fever, chills, headache, shortness of breath, nausea, vomiting, and new medication use. Her medical history includes depression, hypothyroidism, vitiligo, and COPD, for which she was on 3 L home O2 at baseline and used albuterol. Other treatments include levothyroxine 100 microgram, fluconazole 100 mg PO daily, and 0.1% triamcinolone ointment. Her family history revealed psoriasis in her maternal uncle and mother. She has smoked 0.8 packs/day for ten years.

Physical examination revealed a silvery scaling lesion with underlying erythema on the scalp and diffuse thick hyperkeratotic erythematous scaly plaques all over her body, more concentrated on her breasts and extremities (Figure 1). Nail examination showed periungual scaling, pitting, and onycholysis (Figure 1). Other physical findings were unremarkable.

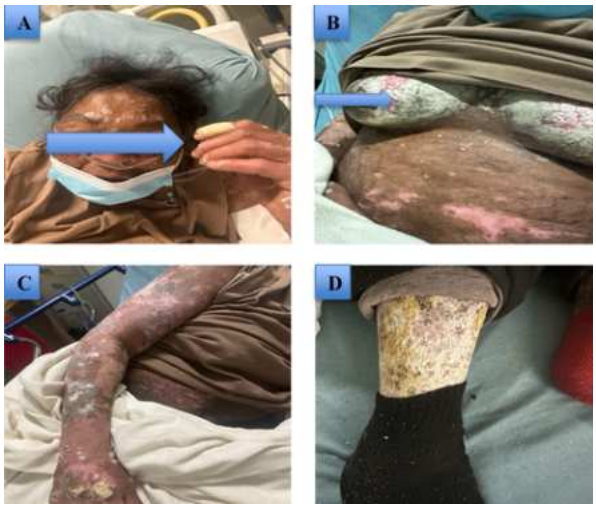

Figure 1: Hyperkeratotic Silvery Plaques With Erythematous Background
A. Face
B. Trunk and breast
C. Arm and forearm
D. Lower Leg

Laboratory examination revealed a white blood cell count of 5,800 x $10^3/\mu L$, hematocrit of 38%, hemoglobin 11.3gm/dl, and platelet count of 124,000/mcl. Blood chemistry was within the reference range except for creatinine, 3.87mg/dl, and BUN 42mg/dl; Rapid plasma reagin test for Syphilis was non-reactive. A punch biopsy showed hyperkeratotic skin with dermal fibrosis and pigment incontinence, with no evidence of vasculitis. The patient was then treated with betamethasone dipropionate 0.05% ointment and restarted on HAART medication for a diagnosis of psoriasis vulgaris but subsequently passed away shortly afterward due to another unrelated cause.

DISCUSSION

Psoriasis within HIV-infected populations poses distinct therapeutic challenges due to increased severity and treatment resistance. Psoriasis may present at any stage of HIV, occasionally as the initial presentation. Pre-existing psoriasis frequently worsens upon HIV contraction. This complex relationship is underscored by the T-cell-mediated nature of psoriasis occurring alongside the T-cell depletion characteristic of HIV (7)

In this case, the patient had a history of well-controlled psoriasis while adhering to HAART but experienced a significant flare following the discontinuation of her HIV medication. This emphasizes the critical role of HAART in managing psoriasis in HIV-positive patients. HAART not only controls HIV infection but also appears to exert a beneficial effect on psoriasis by modulating the immune system.

Managing psoriasis in HIV-positive individuals requires a delicate balance between treating the skin disease and preserving immune function. Treatment options are tiered into topical, phototherapeutic, and systemic modalities (8). Topical therapies (tars, emollients, salicylic acid, corticosteroids, retinoids) remain the first-line treatment for mild to moderate psoriasis, while systemic therapies are considered for more severe cases (8).

Antiretroviral therapy (ART) warrants strong consideration as a first-line treatment for moderate to severe psoriasis, often serving as effective monotherapy (7). Phototherapy (UVB, PUVA) can be adjunctive for severe cases. While UV radiation is immunosuppression and activates HIV in vitro and animal studies (8), clinical studies demonstrate no significant immune deterioration with UVB treatment in HIV-associated psoriasis (9).

Traditional systemic therapies (Figure 2) are reserved for moderate to severe psoriasis but carry heightened immunosuppressive risks in HIV-positive individuals. The literature strongly favors ART as the first-line systemic treatment (10), with open-label studies demonstrating up to 90% improvement rates (11). Oral retinoids can be a second-line option. Highly immunosuppressive agents (cyclosporine, methotrexate, hydroxyurea, TNF- inhibitors) should only be used when all other approaches fail, and the benefits outweigh the significant risks.

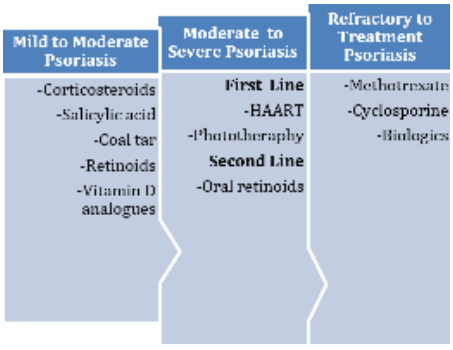

Figure 2: Treatment of HIV-associated psoriasis

CONCLUSION

Individuals with HIV infection often suffer from severe psoriasis, leading to considerable therapeutic challenges. This case report highlights the potential of antiretroviral therapy (ART) in managing psoriasis alongside its primary role in HIV treatment. While treating psoriasis in HIV-positive individuals remains complex, ART demonstrates significant promise in improving both psoriatic symptoms and overall patient outcomes.

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