

An autoimmune quandary: discoid lupus erythematosus and HIV coexistence in an Indian patient

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Abstract

The development of autoimmune diseases in the backdrop of an immune dysfunction in patients with human immunodeficiency virus (HIV) infection is intriguing. An infectious trigger for immune activation is one of the postulated mechanisms and derives from molecular mimicry. Progression from HIV infection to AIDS is associated with a gradual loss of immune-competence. Immune dysregulation and persistent, prolonged immune activation leads to autoimmune phenomena such as lupus and serological abnormalities. We report this sui generis case of discoid lupus erythematosus in HIV positive Indian male for its sheer clinical charm. We also discuss on the probable etiopathogenesis which has not been confounded together so far.

Key words

Human immunodeficiency virus, discoid lupus erythematosus.

Introduction

The fact that autoimmune and inflammatory diseases can occur despite an apparent loss of immunocompetence caused by HIV infection is a true paradox. Association of discoid lupus erythematosus (DLE) with human immune deficiency virus (HIV) infection in the same patient is extremely rare. HIV infection appears to have a favorable impact in the clinical course of DLE. On the other hand, immunosuppressive therapy given for lupus tends to worsen HIV infection.¹ It is well documented that HIV infection can mimic the presentation of autoimmune disorders, although there have been various conflicting reports on how exactly HIV

infection affects the course and prognosis of diseases like DLE.² We report a diacritic case of an HIV-infected patient, who developed extensive DLE.

Case Report

A 49-year-old male, truck driver by occupation, detected to be HIV positive 6 months back with CD4 count 546 cells/mm³, presented to dermatological outdoor with history of depigmented, asymptomatic skin lesions over the face, trunk, both upper limbs since last 2 months. These started as small papules and gradually evolved into larger, depigmented plaques with dry scaly surface. These were associated with photosensitivity. General physical examination and systemic examination were normal. Mucocutaneous examination revealed multiple, depigmented plaques measuring 2×1 cm² to 5×4 cm², with dry surface covered with thick, grayish adherent scales,

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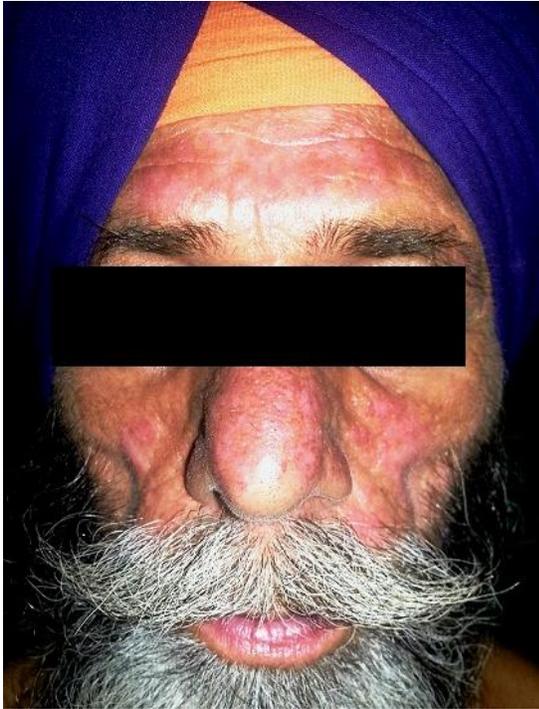


Figure 1 Erythematous to depigmented plaques over the cheeks, nose and malar area of face.



Figure 2 Multiple erythematous to depigmented plaques with scaling on the back.

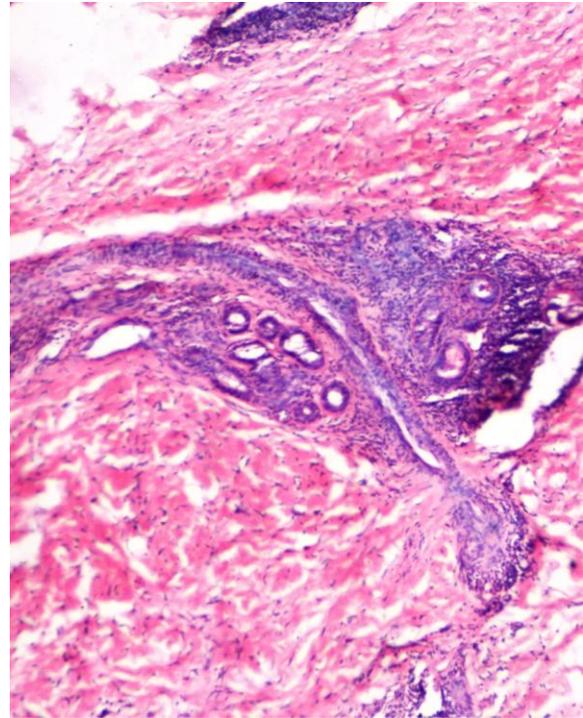


Figure 3 H& E stained section showing dermis with a perivascular and periadnexal lymphohistiocytic infiltrate. (100X).

distributed over the forehead, nose, and cheeks (**Figure 1**), both forearms, and trunk (**Figure 2**). Tin-tack sign was positive. Hair, nail and mucosae were normal. Routine blood and urine investigations were within normal limits. Antinuclear antibody (ANA) profile was negative. A biopsy was performed with the differential diagnosis of DLE, vitiligo, lupus vulgaris, hypopigmented lesions of tuberculoid leprosy, actinic keratoses, coral reef keratoacanthomas.

Histopathological examination confirmed the diagnosis of DLE (**Figure 3**). Patient was started on hydroxychloroquine 200 mg twice a day after a thorough ocular examination. He was also given a topical sunscreen and topical steroid (mometasone furoate 1% cream).

Discussion

Autoimmune diseases are well-documented manifestations of HIV infection and are

generally considered to be a manifestation of late stage of the disease.³ Although HIV infection is often associated with several rheumatic diseases, the coexistence with DLE is extremely uncommon. Generally, HIV-related immunosuppression improves lupus and antiretroviral therapy may lead to an autoimmune disease flare subsequent to the increase in circulating CD4+ cells (immune reconstitution syndrome).⁴

Immunologically speaking, there is no clarity as to how the body reacts to immune alterations in presence of HIV infection. Regulatory T cells (Tregs) are CD4+ CD25^{bright} CD62L^{high} cells that actively down regulate immune responses. Tregs appear to decline at different rates compared with other CD4+ T cells, resulting in an increased regulator to helper ratio in many patients with advanced disease. They were observed to be a major contributor to the immune activation during chronic HIV infection.⁵ Furthermore what is intriguing is the fact that these Foxp3+ Treg cells have been found to play a pivotal role in the specific organ inflammation in lupus.⁶ This underscores beyond doubt that coexistence of lupus and HIV is more than a chance. Now how these Treg cells interplay their role in DLE in a HIV positive patient needs more knowledge at the molecular level and further detailing.

Photosensitivity in HIV-infected individuals appears to be a manifestation of advanced disease. Most patients are sensitive to UVB. Even this can play a role in precipitating DLE in this already altered immune milieu.⁷ The understanding of the impact of immune dysregulation, as well as, the effect antiretroviral therapy-associated immune reconstitution syndrome (IRIS) has on the generation of autoimmune phenomena in HIV infection remains shrouded.

In addition, the question of the validity of the diagnostic criteria of lupus in the setting of HIV positivity would probably require further refinement in future. It may bring a lot of diagnostic difficulties to differentiate the DLE/SLE and HIV infection, since there are many multiorgan symptoms that are common for both of these diseases.⁸

Treatment of such patients remains dicey with few therapeutic options. In view of HIV infection, immunosuppressants are relatively contraindicated. Thalidomide⁹ and hydroxychloroquine¹⁰ holds promise in such situations along with adequate sun protection and topical steroids.⁹

The frequency of reported rheumatological syndromes in HIV-infected patients ranges from 1 to 60%. The list includes SLE, anti-phospholipid syndrome, vasculitis, primary biliary cirrhosis, polymyositis, Graves' disease and idiopathic thrombocytopenic purpura.¹¹ To the best of our knowledge there has been a single case report of chronic cutaneous lupus erythematosus in an Indian patient.¹⁰ We report this case for its rarity and quiddity. In midst of a rising HIV pandemic, an apt diagnosis and precision to treat such conditions is a must.

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