# Epidermotropism of lepra bacilli in a patient with histoid Hansen's disease

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## ABSTRACT

Histoid leprosy is a rare form of multibacillary leprosy with distinct clinical and histopathological features. It is a variant of lepromatous leprosy with a very high bacillary load. It appears in patients as relapse after dapsone monotherapy and resistance or rarely, "*de novo*." Although leprosy is slowly declining the exact mode of transmission is unclear. At least until recently, the most widely held belief was that the disease was transmitted by contact between cases of leprosy and healthy persons. Transmission by the respiratory route is also gaining ground. There are other possibilities such as transmission through insects, which cannot be completely ruled out. However, the present case report possibly suggests the role of skin as a portal of both exit and entry for the bacillus in histoid leprosy transmission. *De novo* form of histoid leprosy has numerous solid staining bacteria inside the epidermis. The reports show that these bacilli can be eliminated from the intact epidermis, which indicate an unusual role of the skin in the transmission of leprosy.

**Key words:** Dapsone resistance, *de novo*, histoid leprosy, transepidermal transmission

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## INTRODUCTION

Histoid leprosy is an uncommon variant of lepromatous leprosy characterized by cutaneous/subcutaneous nodules and plaques present over apparently normal skin, with unique histopathological findings, characteristic bacterial morphology and very high bacillary load. The term histoid leprosy was coined by Wade as a histological concept of bacillary-rich leproma composed of spindle-shaped cells in the absence of globi (so conspicuous in an ordinary leproma). It exhibits a fibromatoid tendency in the chronic form.[1] It occurs in lepromatous patients who relapse after the dapsone monotherapy and resistance, or even *de novo*. It is occasionally seen in unstable borderline and intermediate type of leprosy. Responsible factors may include resistance to dapsone, irregular and inadequate therapy, or mutant organism (histoid bacillus).

## CASE REPORT

A 21-year-male presented with papular lesions on his face since 20 days. He had no complains of fever, epistaxis or slippage of sandals. On examination, multiple shiny papulonodular lesions were found over his forehead, ears, and cheeks [Figure. 1] Nasal stuffiness, congestion and watering from eye were present. Bilateral ulnar, greater auricular, common peroneal, anterior tibial, and posterior tibial nerves were thickened. Glove and stocking anesthesia was present up to the elbows and knees. Corneal and conjunctival sensations were normal. Motor examination was normal. All routine investigations were normal. On histopathological examination, sections showed extensive cellular infiltration in the dermis mainly composed of macrophages, lymphocytes and plasma cells. There are clear Grenz zones below the flattened epidermis. Hematoxylin and eosin stain showed histiocytes arranged in a storiform pattern with classical histiocytic granulomas [Figure. 2] and heavy bacillary load on Wade Fite stain. Slit skin smear revealed acid fast bacilli with a bacteriological index of 6.[Figure. 6] Wade Fite stain revealed numerous solid staining acid fast bacilli arranged discretely and in clumps inside the dermis, including the Grenz zone, and surprisingly also inside the epidermis, inside vacuoles in the prickle cell layer and in the stratum granulosum.[Figure. 3-5) The presence of bacilli at various levels inside the

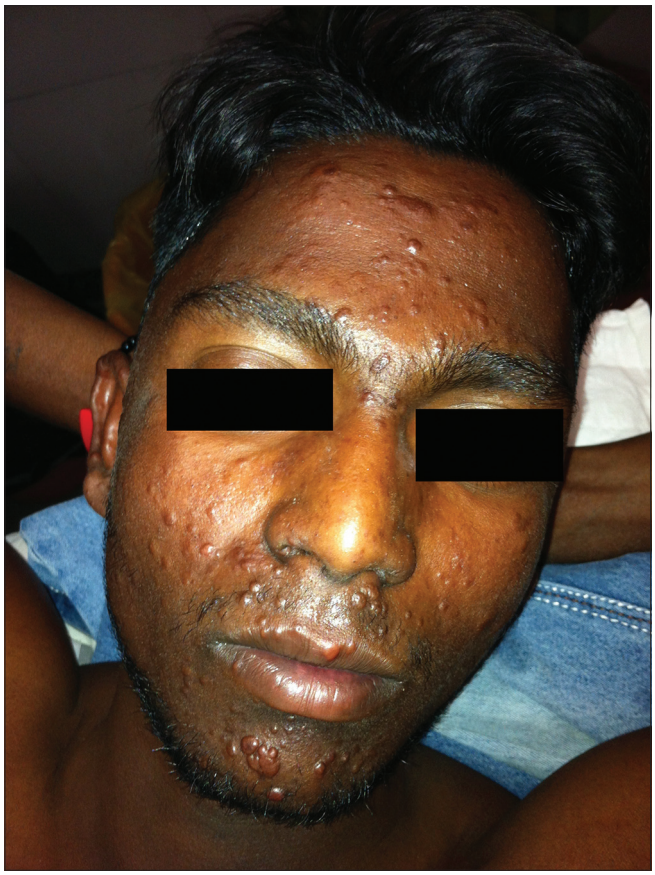

**Figure 1:** Multiple shiny succulent papulonodular lesions over the forehead, cheeks, and chin

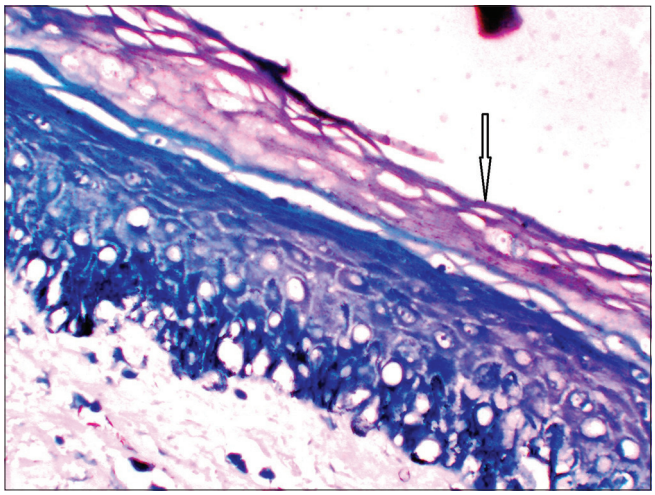

**Figure 3:** Wade Fite stain revealed numerous acid fast bacilli arranged in clumps inside the epidermis

epidermis suggested their movement upward along with epidermal cell maturation.

## DISCUSSION

Histoid leprosy presents with shiny, skin colored or erythematous papules, nodules or plaques arising abruptly over normal skin,

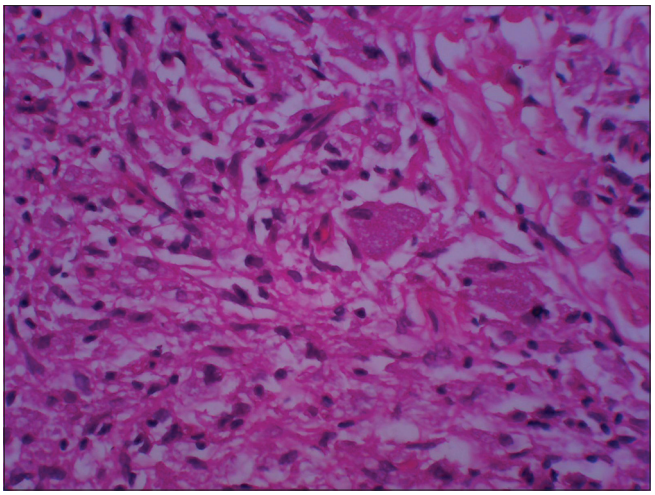

**Figure 2:** Histopathology showing extensive cellular infiltration in dermis mainly composed of macrophages, lymphocytes and plasma cells with histiocytes arranged in a storiform pattern with classical histiocytic granulomas

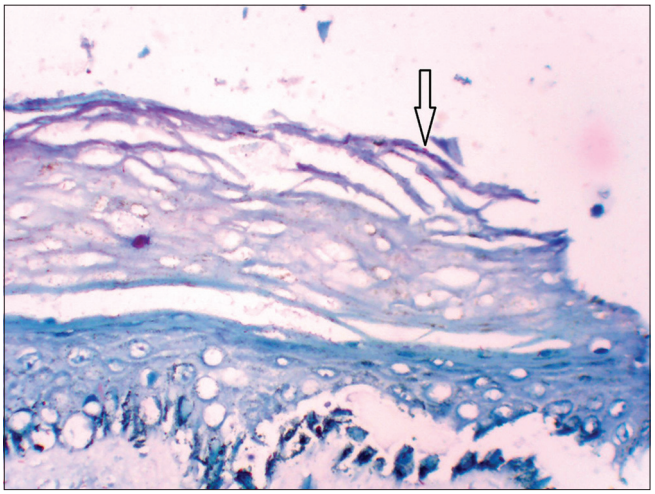

**Figure 4:** Wade Fite stain revealed numerous solid staining elongated slender acid fast bacilli arranged discretely in vacuoles in the stratum corneum, prickle cell layer and in the stratum granulosum

mostly after inadequate or irregular treatment or because of dapsone resistant mutant strains, but may occur *de novo*.[1] In a recent Indian report, histoid leprosy was found to constitute 1.8% of all leprosy cases and occurred *de novo* in 12.5% of cases.[2]

There are scant reports of detection of *Mycobacterium leprae* inside the leprous epidermis[3-5] and they could be missed/underreported unless specifically looked for.[3] Okada *et al*.[5] suggested that dermal bacilli could be gradually transferred to the epidermal layers through phagocytic activity of young basal cells, and finally eliminated, possibly through the intact skin. Namisato *et al*.[6] described "transepidermal elimination" of *M. leprae* in lepromatous leprosy and attributed it to rapidly growing dense granulomas in the upper dermis. The presence of bacilli in all layers of epidermis indicates that the bacilli that

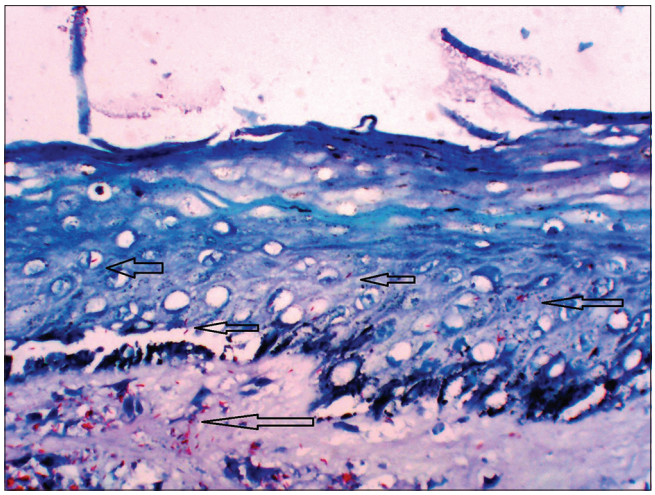

**Figure 5:** Wade Fite stain revealed numerous solid staining elongated slender acid fast bacilli arranged discretely and in clumps inside the epidermis and in in dermis

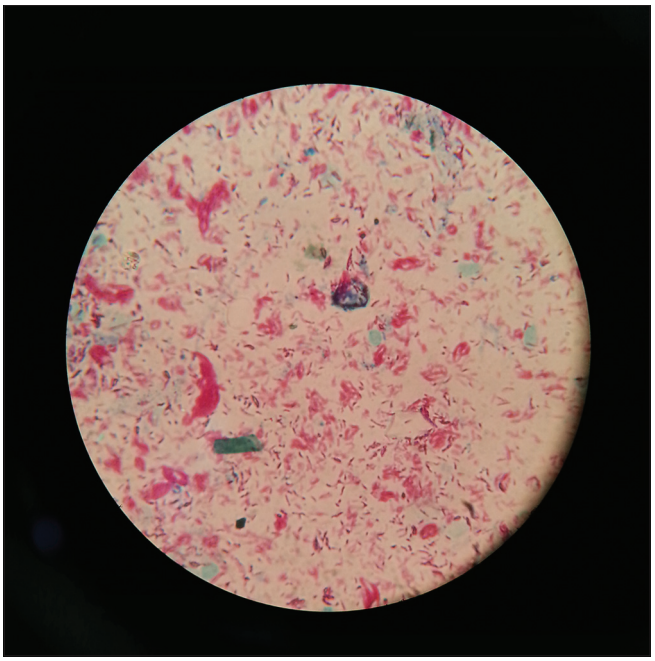

**Figure 6:** Slit skin smear showing numerous acid fast bacilli

are taken up by the basal cells from the upper dermis can move upward inside the epidermal cells and are ultimately eliminated from the stratum corneum into the environment. The very large area of skin of about 1.62 $m^2$ in an adult Indian and the average turnover time of about 28 days seems to provide an opportune environment for bacillary multiplication and elimination, as even 1 g of lepromatous tissue is estimated to carry up to 7 billion bacilli. It is also known that the bacilli once eliminated could remain viable for several days or weeks.[7] As shown in polymerase chain reaction studies, 60% of untreated *Mycobacterium* patients had bacilli in the keratin layer, and 80% had *M. leprae* deoxyribonucleic acid in skin washings.[8]

Interestingly, on the other hand, the reports of initial/single leprosy lesions developing at the site of tattooing, vaccination scar or trauma suggesting that the bacilli could gain entry through traumatized skin. The recent report of linearly distributed skin lesions suggesting pseudo-isomorphic phenomenon of Koebner in a histoid leprosy patient points toward cutaneous inoculation of the bacillus.[9]

Dendritic cells are antigen-presenting cells that phagocytose microorganisms and particles and are represented by Langerhans cells in the epidermis. They have a role in the cutaneous response to leprosy, which may be a determinant of the course and clinical expression of the disease.[10] Future studies on their contribution, if any, to the carriage of and response to the epidermal bacilli might help unravel a few unexplored aspects of leprosy.

The transepidermal exit of the *M. leprae* demonstrated in this case, along with previous reports mentioned above, indicate that skin could possibly be a portal for both exit and entry of the bacillus causing the occurrence of pseudo-isomorphic Koebner phenomena and should be given due recognition for its contributory role in leprosy transmission. Okada *et al*. suggested that upper dermal lepra bacilli in multibacillary patients could be phagocytosed by basal cells and gradually move up through the epidermis to be finally eliminated, possibly also from intact skin.[5] However, scant reports of this phenomenon may reflect underreporting.

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