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Case Report

Lepromatous leprosy Hansen's mimicking histoid Hansen's: a case report

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ABSTRACT

Lepromatous leprosy (LL) represents the anergic pole of the leprosy spectrum and may rarely mimic histoid leprosy (HL). A 45-year-old man presented with multiple shiny umbilicated papules and nodules over normal skin resembling HL. Slit-skin smear revealed numerous solid-staining bacilli (BI 5+), and histopathology showed diffuse foamy macrophages without spindle cells, confirming LL. The patient responded to multibacillary multidrug therapy. This case emphasizes the clinical overlap between LL and HL and the importance of bacteriological and histopathological evaluation for accurate diagnosis and management to prevent misclassification and ensure effective treatment.

Keywords: Lepromatous, Leprosy Hansen's, Histoid Hansen's

INTRODUCTION

In lepromatous leprosy (LL), the first clinical manifestations are cutaneous dermal with macules, diffuse papules, infiltration or nodules, or all four. Macules are small, multiple, red or faintly hypopigmented, with vague edges and shiny surface.[1]

Papules and nodules usually have a normal skin colour but sometimes are red with a bilaterally symmetrical distribution on the face, arms, legs and buttocks, but may be anywhere apart from the hairy scalp, axillae, groins and perineum. In histoid leprosy (HL), lesions are distinctive round, regular, cutaneous nodules that stand out on normal skin. It usually occurs in patients on long-term diamino-diphenyl sulfone, with initial improvement followed by relapse.[2]

Presentation of LL Hansen's with predominantly papular and nodular lesions clinically mimicking HL is infrequent. Here in a case of LL Hansen's who presented with umbilicated papules and shiny papules over normal skin closely resembling histoid Hansen's (HH).

CASE REPORT

A 45-year-old male patient came with complaints of skin-colored vivid lesions over trunk and upper limbs since 6 months. Initially, patient developed skin-colored lesions over the upper back followed by lower back and upper limbs. There was no loss of sensations, no bleeding or stuffiness of nose. There was no association with swelling over ankles, dryness over lower limbs, fever, joint pains, malaise or any eye involvement. There was neither history of slipping of chappals while walking nor any feeling of walking on cotton wool, no difficulty in doing fine movements like buttoning of shirt. No deformity was noted such as claw hand. He was not a known diabetic or hypertensive with no history of thyroid dysfunction. There was no relevant past history and family history. The patient took mixed diet. There was no loss of appetite, no Sleep disturbances and no addictions. Bowel and bladder movements were normal. General examination and systemic examination were normal.

On dermatological examination multiple, discrete, skin-colored papules and few nodules over normal skin were seen on the trunk and upper extremities. Few papules

showed central umbilication. Few grouped papules were seen over trunk. In few areas erythematous infiltrated plaques with overlying shiny papules were observed. Ear lobe infiltration present. Lateral loss of eyebrows was seen. On nerve examination, right ulnar nerve was thickened. Other nerve trunks and cutaneous nerves such as median, radial, superficial peroneal, anterior tibial, posterior tibial, supra trochlear and supraorbital nerves were not thickened. Sensory nerve examination was normal. Voluntary muscle testing showed no abnormality and no deformities were found. Based on the clinical picture, differentials of HH, papular mucinosis, papular sarcoidosis and cryptococcosis were considered.

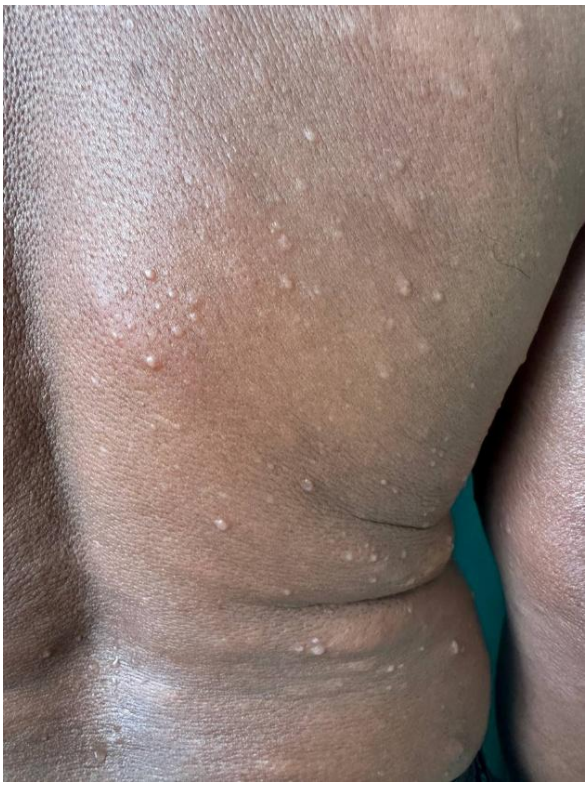

Figure 1: Discrete shiny skin-coloured papules arising over normal skin seen over back.

Complete hemogram, serum biochemistry, viral screening and Imaging studies were normal. Slit skin smear examination showed multiple solid stained bacilli in each field with bacillary index or 5+(100-1000 bacilli in an average field). Histopathology showed basket weave orthokeratosis, preserved granular layer, mild acanthosis and intact basal layer. The superficial and deep dermis show diffuse and dense infiltration with foamy macrophages and few lymphocytes. Fite-faraco stain shows abundant solid stained typical lepra bacilli which were not long and slender.

Based on clinical picture, slit skin smear finding and histopathological examination patient was diagnosed as lepromatous leprosy. Patient was administered MB-MDT along with supportive treatment. Moderate improvement in skin lesions was noticed after 2 months and the patient is under follow up.

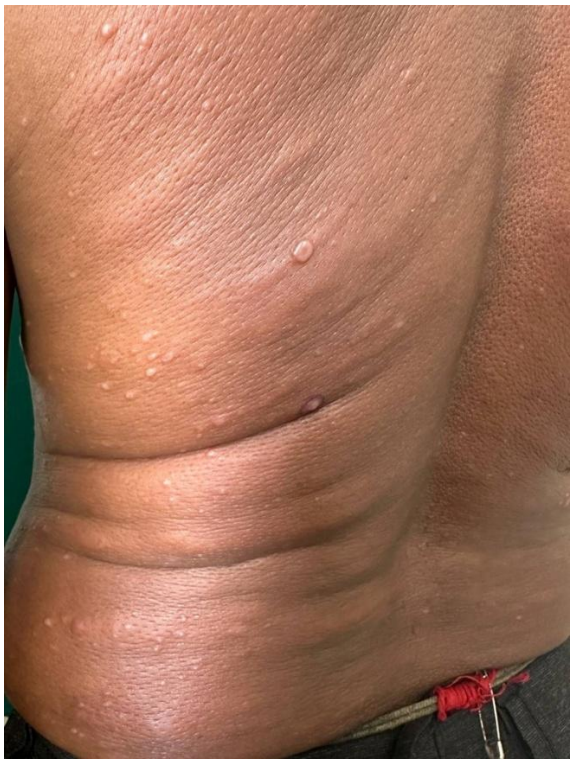

Figure 2: Shiny umbilicated papules, multiple small skin-coloured papules and infiltrated lesions seen over left flank region and back.

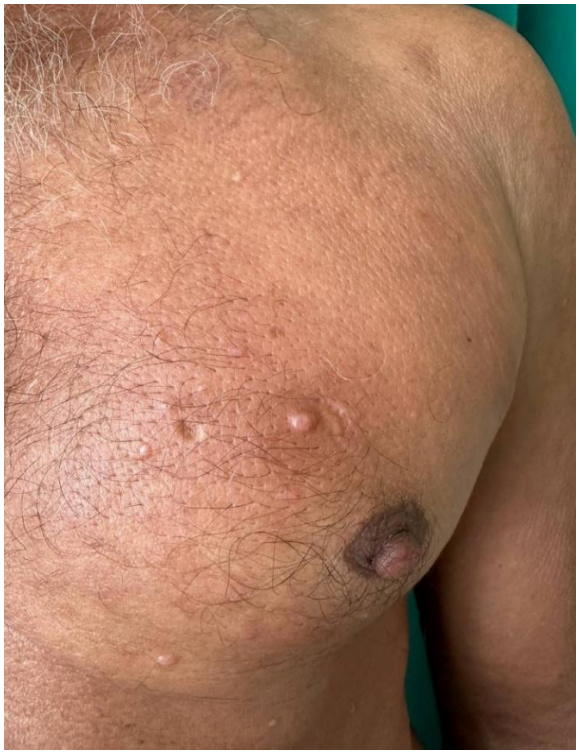

Figure 3: Shiny papules and surrounding infiltration seen over left side of chest.

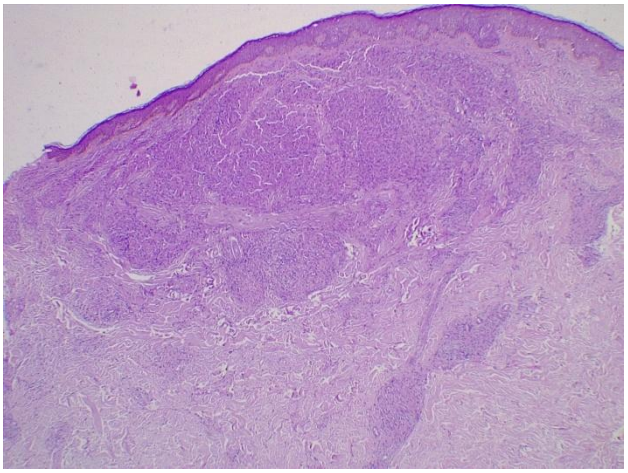

Figure 4: Inflammatory infiltrates in the dermis along with prominent perineural infiltrate.

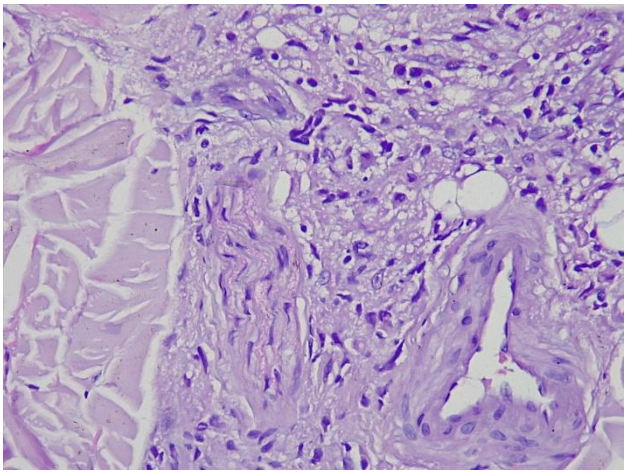

Figure 5: Thin epidermis and diffuse and dense infiltrate with macrophages and lymphocytes throughout the dermis.

DISCUSSION

LL represents the anergic pole of the Ridley-Jopling spectrum and is characterized by a complete absence of cell-mediated immunity to *Mycobacterium leprae*, resulting in uncontrolled bacillary proliferation and widespread disease. It presents with numerous, symmetrical papules, nodules, and plaques that may coalesce to produce diffuse infiltration, giving rise to leonine facies and diffuse nerve involvement.[3] In LL, systemic involvement with involvement of multiple organ systems in the body including nasal mucosa, larynx, ocular involvement, bone and joint involvement and-are commonly seen. In lepromatous leprosy nodules arise among areas of infiltration and its edge merges imperceptibly with normal skin. Histopathologically, LL demonstrates an atrophic epidermis with a grenz zone and a diffuse dermal infiltrate of foamy macrophages (Virchow cells) laden with acid-fast bacilli.[4] The bacillary index (BI) is typically 5+ to 6+, while the morphological index (MI) is low due to fragmented bacilli.[5] Immunologically, LL is governed by a Th2 cytokine milieu with high IL-4 and IL-10 expression and absent delayed-type hypersensitivity.[6] In the present case the patient presented with predominantly papular and nodular lesions and few infiltrated lesions but without any evidence of systemic involvement

HH disease is a distinct clinicopathological variant of lepromatous leprosy rather than a separate entity, first described by Wade in 1963.[7] It may occur *de novo* or as a relapse after inadequate or irregular therapy, particularly dapsone monotherapy, or in association with drug-resistant bacilli. Clinically, HH presents with discrete, firm, dome-shaped, shiny papules or nodules over apparently normal skin, commonly distributed on the back, buttocks, face, and extremities.[8] In HH, nodules arise on a normal skin and edge is sharply demarcated. Unlike LL, there is no diffuse infiltration of skin, and the lesions may mimic dermatofibroma, xanthoma, or neurofibroma, which can lead to diagnostic confusion.[9] The disease is epidemiologically significant because histoid lesions contain a massive bacillary load and viable organisms, thus acting as potential reservoirs for transmission. Pathogenesis is thought to involve partial restoration of local cell-mediated immunity within a lepromatous background, resulting in the formation of spindle-cell granulomas. In the case, though the nodular lesions over normal skin and umbilicated papules resembled HH, based on clinico-investigative correlation a final diagnosis of HH was diagnosed.

Investigations reveal distinct differences between LL and HH. On slit-skin smear, HH shows a very high BI (5+ to 6+) with a high MI due to the presence of numerous viable bacilli, whereas in LL, bacilli are fragmented with a lower MI. Morphologically, histoid bacilli are long, slender, solid-staining rods often arranged in parallel bundles or “histoid habitus,” in contrast to the shorter, granular bacilli of LL.[10] Electron microscopy demonstrates that LL bacilli are surrounded by electron-transparent “foam zones,” whereas histoid bacilli lack these spaces, maintaining structural integrity and viability. Histopathologically, LL reveals a diffuse infiltrate of foamy macrophages with globi formation, while HH exhibits a well-circumscribed lesion composed of spindle-shaped histiocytes arranged in whorled or storiform patterns with minimal foamy change. A clear grenz zone and pseudo-capsule are often seen. Immunohistochemical studies indicate relatively increased CD3+ and CD8+ T-cell populations in HH compared with LL, suggesting partial immune restitution (da Costa et al).[11] In the present case because of absence of spindle shaped cells and histiocytes, ABSENCE of long slender bacilli and presence of typical bacteriological and histological features of LL led to the diagnosis of LL Hansen’s.

Treatment of both forms follows the WHO multidrug therapy (MDT) regimen for multibacillary leprosy-rifampicin 600 mg monthly, clofazimine 300 mg monthly

plus 50 mg daily, and dapsone 100 mg daily for at least 12 months.[12] However, HH often necessitates prolonged or intensified treatment due to its high bacillary load and relapse potential. Several experts advocate extending therapy to 18-24 months or until smears are negative. Because HH frequently arises after dapsone monotherapy, drug resistance testing-particularly for rpob and folp1 gene mutations conferring rifampicin or dapsone resistance-is recommended. In resistant or relapsed HH, second-line bactericidal drugs such as ofloxacin, minocycline, or clarithromycin may be added to MDT for 6-12 months. Prolonged follow-up is essential, as viable bacilli may persist even after apparent cure. In the present case patient was started on MB-MDT and moderate improvement in patient's general condition and skin lesions was noticed after 2 months.

CONCLUSION

The case highlights the occurrence of umbilicated papules and shiny nodules over normal skin in a case of Hansen's. The skin lesions over multiple lesions of body closely mimicked HH, but based on slit skin smear examination and thorough evaluation of histopathological examination and clinical examination the case was diagnosed as lepromatous leprosy. Clinically, the skin lesions were suggestive of HH but the histopathology favoured lepromatous leprosy including the presence of globi.

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Ethical approval: Case Report

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