

Case Report

Crateriform and ulcerated lesions of nodular lepromatous leprosy masquerading as histoid Hansen's disease

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ABSTRACT

Nodular lepromatous leprosy (LL) classically presents with diffuse infiltration, nodules, and plaques. However, unusual morphologies may lead to diagnostic confusion, particularly with histoid Hansen's disease. We report a rare presentation of nodular lepromatous leprosy manifesting as crateriform and ulcerated papules and plaques, clinically simulating histoid leprosy. Detailed clinical evaluation, histopathology, and slit-skin smear examination aided in establishing the correct diagnosis. This case highlights the importance of recognizing atypical cutaneous manifestations of leprosy to avoid misclassification and ensure appropriate management.

Keywords: Lepromatous leprosy, Nodular leprosy, Crateriform lesions, Ulceration, Histoid Hansen's disease, Atypical leprosy

INTRODUCTION

Leprosy is a chronic infectious disease caused by *Mycobacterium leprae*, exhibiting a wide clinical spectrum determined by the host's immune response. Lepromatous leprosy represents the multibacillary pole and is characterized by symmetric skin lesions, diffuse infiltration, nodules, and a high bacillary load.¹ Histoid leprosy is a well-recognized variant of lepromatous leprosy, typified by firm, shiny nodules and plaques, spindle-shaped histiocytes on histopathology, and an abundance of acid-fast bacilli. In contrast, crateriform and ulcerated lesions are distinctly uncommon in nodular lepromatous leprosy and may pose a diagnostic challenge by closely mimicking histoid morphology.

We describe an unusual case of nodular lepromatous leprosy presenting with crateriform and ulcerated lesions, clinically resembling histoid Hansen's disease, emphasizing the role of histopathology and bacteriological examination in reaching an accurate diagnosis.

CASE REPORT

A 45-year-old male who is a manual labourer with no known co-morbidities, admitted in the department of orthopaedics for a fracture of the right femur, sought opinion for multiple ulcerated skin lesions in the trunk and extremities for the past 6 months. It was found that the lesions were spontaneous in origin, asymptomatic in nature, and not associated with pain or itching.

Initially, all these lesions started as tiny papules and nodules, then gradually increased in size, and some of them were ulcerated. He mentioned the history of anaesthesia of the bilateral hands and feet (glove and stocking area). There was no history of any hypopigmented patch elsewhere in the body.

No history suggestive of type 1 and type 2 lepra reactions. There was no history of previous treatment for leprosy. The family members had no similar complaints.

On examination, there were multiple skin colored to erythematous, non-tender, umbilicated papules and nodules, and crateriform plaques of varying sizes, ranging from 0.25×0.25 cm to 2×1 cm, distributed predominantly over the trunk, upper and lower extremities, and ear lobes. Some of the lesions were ulcerated and crusted (Figure 1). Nerve examination revealed symmetrically thickened, non-tender ulnar, radial cutaneous, and common peroneal nerves. Clinically, perforated granuloma annulare, papulo-necrotic tuberculid, histoid Hansen, and opportunistic deep mycosis were considered as differential diagnoses.



Figure 1: Multiple skin colored to erythematous, non-tender, ulcerated, umbilicated and crateriform nodules and plaques over the (a) trunk, (b) upper, (c) lower extremities, and (d) ear.

All blood investigations, including random blood sugar, complete blood count, liver function test, and renal function test, were within normal limits. Serology for syphilis and human immunodeficiency virus 1 and 2 turned out to be non-reactive. The Mantoux test was negative. Slit skin smear (SSS) was taken, which showed numerous solid stained acid-fast bacilli (Figure 2). Based on the clinical findings and the presence of acid-fast bacilli in SSS, the diagnosis was narrowed down to histoid leprosy.

Further proceeding with the biopsy, histopathological examination showed ulcerated epidermis and dermis showing plenty of sheets and aggregates of foamy histiocytes surrounding the blood vessels, adnexal structures, and nerve bundles. The Grenz zone was seen (Figure 3).

Fite faraco staining showed numerous globi inside the foamy macrophages, and some of the bacilli were observed in the stratum corneum (Figure 4). Hence, a final diagnosis of lepromatous leprosy with transepidermal elimination (TEE) was made. The patient was started on multibacillary multidrug therapy, and he was under regular follow-up.

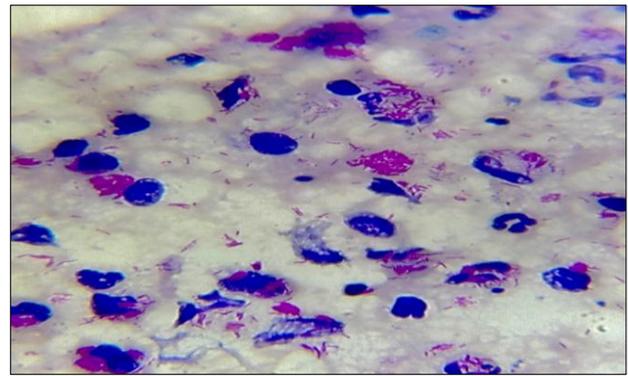


Figure 2: Numerous solid stained acid-fast bacilli in clusters.

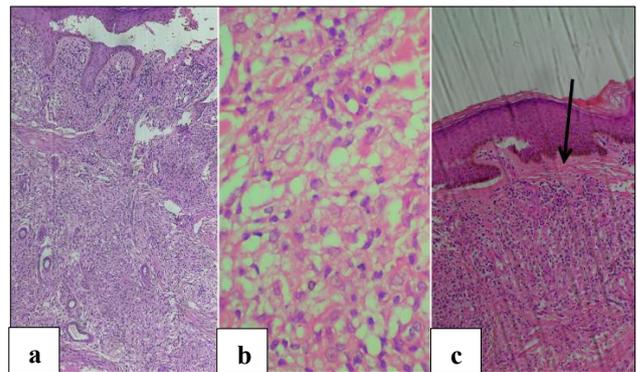


Figure 3: (a) H and E under scanner (4x view) showing ulcerated epidermis and dermis showing plenty of sheets and aggregates of foamy histiocytes surrounding the blood vessels, adnexal structures, and nerve bundles, (b) H and E under high power (40x) showing foamy macrophages, and (c) black colour arrow showing grenz zone.

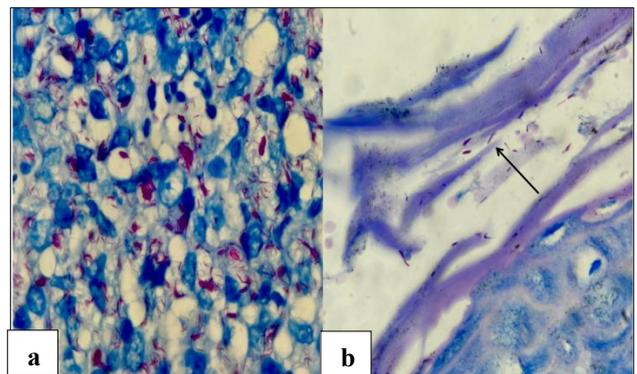


Figure 4: (a) Fite faraco staining showing acid-fast bacilli in globi, and (b) acid-fast bacilli in stratum corneum showing trans-epidermal elimination (TEE).

DISCUSSION

Lepromatous leprosy is a multibacillary form that is characterised by a widespread skin lesion. It presents as

three distinct forms: diffuse, infiltrated, and nodular forms.¹

Nodular lepromatous leprosy is known for its protean clinical manifestations, yet crateriform and ulcerated lesions remain distinctly rare. Such morphology may arise due to pressure necrosis, secondary infection, or rapid bacillary proliferation leading to tissue breakdown.

Ulceration in leprosy can occur in the following conditions like lepra reactions (type 1 and type 2), neuropathic causes and Lucio phenomenon.

Type 1 reaction is ruled out because clinically the patient was asymptomatic and there is no evidence of neuritis, and histopathologically there was no loosely formed granuloma and dermal edema.

Type 2 lepra reaction (erythema nodosum leprosum), it classically presents with tender nodules along with systemic involvement. However, in our case, erythema nodosum leprosum was ruled out because the skin lesions were asymptomatic and non-tender, and confirmed histopathologically, as there was no evidence of panniculitis or vasculitis.

There was no evidence supporting the Lucio phenomenon because clinically there was no retiform purpura with necrotic lesions, and also histopathology did not show any thrombi occluding the dermal blood vessels, and the absence of lepra bacilli in the endothelium.

Spontaneous and de novo ulceration of nodules in lepromatous leprosy is very rare and barely discussed in the literature.²

Ulceration can represent TEE of lepra bacilli. It often indicates a high bacillary load, and various pathomechanisms have been proposed. These bacilli in the dermis are incorporated by the basal cells, transported upwards, and expelled from the stratum corneum into the environment. Namisato et al postulated that TEE occurs as a result of rapidly enlarging dense granulomas in the upper dermis.³ In such conditions, there is a high risk of transmission of leprosy via the transcutaneous route.

Nodular lepromatous leprosy can clinically simulate histoid Hansen, but histopathologically, a true histoid lesion has unique features, such as it does not form globi and contains spindle-shaped histiocytes, creating a fibromatoid appearance in histopathological examination.⁴

While leprosy is a vast domain, arriving at the specific diagnosis is crucial for proper management of the disease because in the case of histoid Hansen, the treatment duration includes 24 months of multi-drug therapy, while treatment of lepromatous leprosy is about 12 to 18 months.⁵ Also, lepra reaction is more prevalent in lepromatous leprosy and is to be anticipated, while it is seldom seen in histoid Hansen, though reported scarcely.

CONCLUSION

Crateriform and ulcerated lesions in nodular lepromatous leprosy are rare and may closely mimic histoid Hansen's disease. Awareness of this unusual presentation, along with careful clinicopathological correlation, is essential for accurate diagnosis and timely initiation of appropriate therapy. This case expands the clinical spectrum of lepromatous leprosy and reinforces the need for vigilance in atypical cutaneous manifestations.

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