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Case Series

The Great Imitator: A Case Series of Leprosy Mimicking Other Diseases

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ABSTRACT

Leprosy, caused by Mycobacterium leprae, is a formidable spectral disease with heterogeneous clinical manifestations that often overlap with dermatologic, neurologic, and systemic disorders. This case series illustrates leprosy's capacity to masquerade as systemic lupus erythematosus (SLE), acute inflammatory demyelinating polyradiculoneuropathy (AIDP), and cutaneous lymphoma, emphasizing the challenges in timely diagnosis. Three patients demonstrated characteristic features of these distinct conditions: a young female presenting with malar rash and alopecia mimicking SLE, an elderly male with progressive neuropathy initially linked to AIDP following hepatitis B infection, and a middle-aged female with chronic indurated plaques and constitutional symptoms suggestive of cutaneous lymphoma. Despite initial diagnostic ambiguity guided by clinical and laboratory findings, histopathological evaluation with Fite-Faraco staining confirmed leprosy across all cases, revealing lepromatous to borderline tuberculoid subtypes. These cases reinforce leprosy's reputation as a "great imitator" and underscore the necessity of including it in differential diagnoses for atypical presentations, even in non-endemic regions. Histopathology remains indispensable for definitive diagnosis, particularly when classic signs such as sensory loss are subtle or not clinically apparent, or when nerve thickening is overlooked due to human error—underscoring the importance of sharpening clinical touch and thorough examination. Enhanced clinical suspicion and collaborative multidisciplinary evaluation are crucial to prevent diagnostic delays and mitigate long-term complications in this protean disease.

Keywords: Autoimmune disease, Leprosy, Lymphoma, SLE

INTRODUCTION

Leprosy, with its diverse clinical spectrum, often mimics other dermatologic and systemic diseases, leading to diagnostic challenges. In endemic regions, maintaining a high index of suspicion is crucial to prevent misdiagnosis. While classic presentations are well-recognized, atypical manifestations can cause significant delays in diagnosis. Histopathology and slit skin smear examination, play a pivotal role in the diagnosis of leprosy. [1] This article presents three intriguing cases that emphasize the protean nature of Hansen's disease.

CASE-1

A 23-year-old female was referred for suspected systemic lupus erythematosus (SLE) due to facial skin lesions resembling a malar rash, along with widespread skin lesions, hair loss, and weakness persisting for five months. She fulfilled five clinical SLICC (Systemic Lupus International Collaborating Clinics) criteria, including photosensitivity, malar rash, oral ulcers, non-scarring alopecia, and multiple joint involvement (Figure-1). Although she had a positive ANA titer, her ANA profile, including anti-dsDNA and anti-Sm, was negative, making the diagnosis uncertain. Histopathology revealed features of lepromatous leprosy, including epidermal thinning, a Grenz zone, and dermal infiltration of foamy macrophages. Fite-Faraco staining and a slit skin smear confirmed acid-fast bacilli.

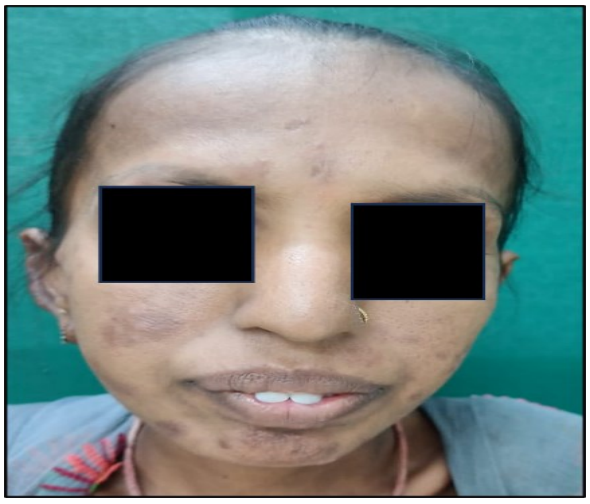

Figure-1: Non-scarring alopecia and photosensitive facial rash

CASE-2

A 60-year-old male with active hepatitis B and suspected acute inflammatory demyelinating polyradiculoneuropathy (AIDP) was referred for evaluation of Hansen's disease after inconclusive MRI findings. He presented with a burning sensation, decreased hand grip, and numbness in the left hand for 15 days. Nerve conduction studies showed absent ulnar, median, and sural sensory nerve action potentials. Initial leprosy screening was inconclusive, with a negative slit skin smear for acid-fast bacilli. However, re-evaluation revealed faint erythematous patches on the palms (Figure-2).

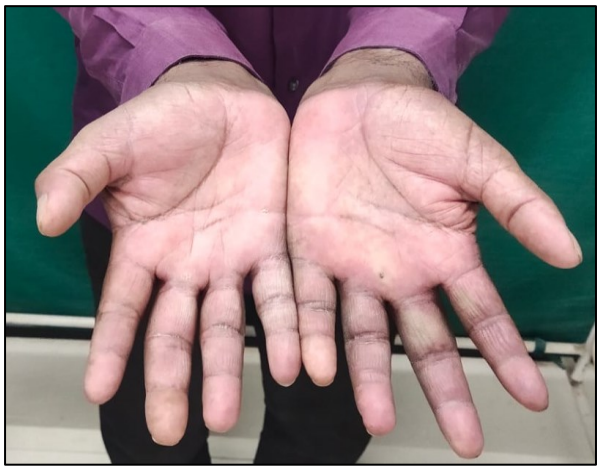

Figure-2: Inconspicuous erythematous patches over palms

A skin biopsy from the left palm showed epidermal hyperplasia, fibro-collagenous changes, lymphohistiocytic infiltrates, and a well-formed granuloma with histiocytes and epithelioid cells, confirming borderline tuberculoid leprosy. The patient responded well to multidrug therapy (MDT).

CASE-3

A 38-year-old female presented with progressive and persistent skin lesions over three years along with bilateral pedal edema, and significant weight loss. The lesions, distributed over the trunk, back, and extremities, appeared as multiple, ill-defined, symmetrical patches and plaques with a smooth and mildly scaly surface (Figure-3). The chronicity, distribution, clinical history and morphology raised suspicion for cutaneous T-cell lymphoma, alongside other differentials like eczema, Hansen's disease, and secondary syphilis.

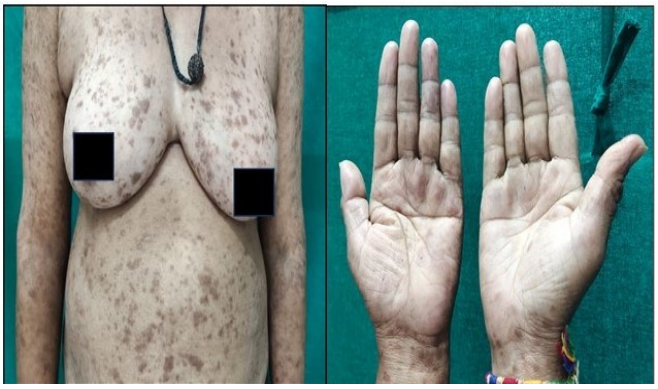

Figure-3: Multiple brown to coffee-colored, macules, patches and plaques over the trunk and palms

However, a leprosy work-up revealed no sensory or motor deficits. Histopathology demonstrated epidermal thinning, a preserved Grenz zone, and dense dermal infiltration of foamy macrophages and lymphohistiocytes (Figures 4 & 5). Fite-Faraco staining confirmed a high bacillary load (Ridley's Bacteriological Index: 6), establishing a diagnosis of lepromatous leprosy (Figure 6).

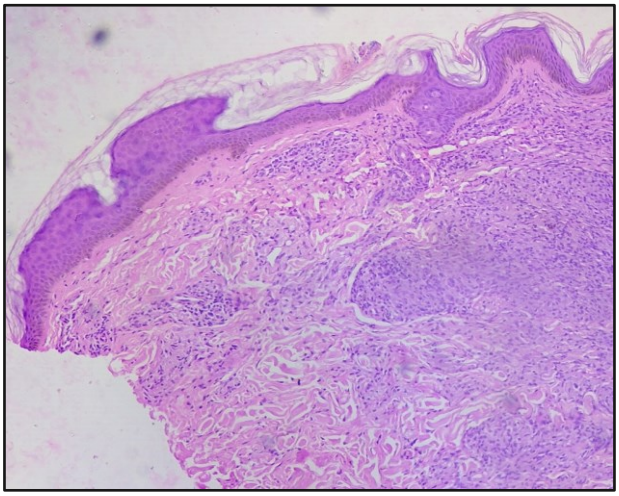

Figure 4: Histopathology demonstrating epidermal thinning, a preserved Grenz zone, and a dense dermal infiltration of foamy macrophages and lymphohistiocytes (H & E scanner view)

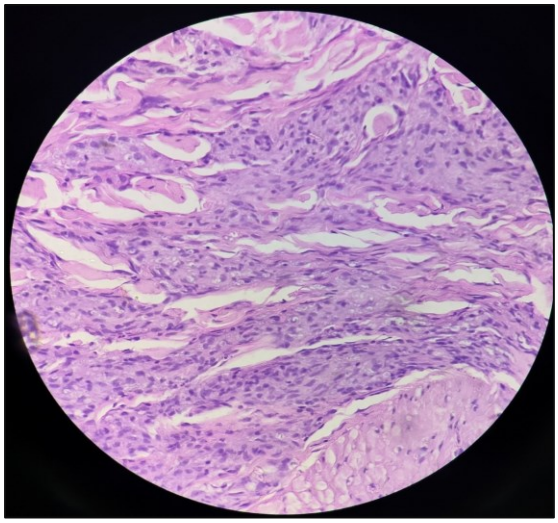

Figure-5: Histopathology demonstrating perivascular & peri-adnexal infiltration of foamy macrophages and dense lympho-histiocytes in the dermis (H&E 40 X)

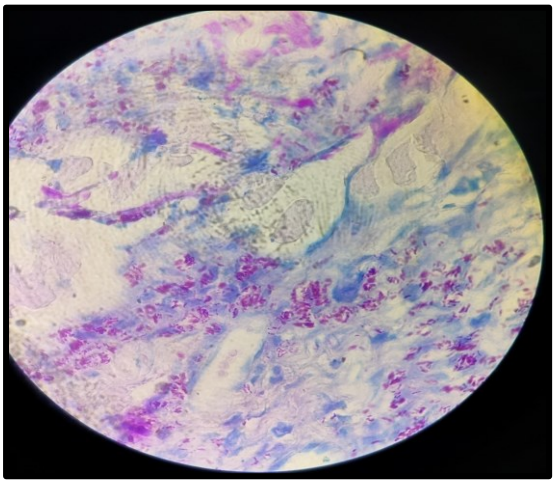

Figure-6: Fite-Faraco stain showing a high bacillary load (Ridley's Bacteriological Index = 6)

DISCUSSION

Leprosy, caused by Mycobacterium leprae, exhibits a broad spectrum of clinical manifestations, often leading to misdiagnosis. While classic features such as hypopigmented patches with sensory loss and peripheral nerve thickening are well recognized, atypical presentations can mimic autoimmune, neurological, and neoplastic conditions. It ranks alongside syphilis, mycosis fungoides, cutaneous tuberculosis, and sarcoidosis as one of the major clinical mimickers.[2]

In Case 1, the patient was initially suspected of having SLE. However, a negative ANA profile prompted further evaluation. Histopathology confirmed lepromatous leprosy, highlighting the diagnostic challenge posed by its atypical presentations.

Leprosy has long been misdiagnosed as vasculitis, lupus, systemic sclerosis, adult-onset Still's disease, rheumatoid arthritis, sarcoidosis, eczema and other autoimmune and autoinflammatory diseases, leading to delayed treatment. [3–10] This case emphasizes the need for heightened clinical suspicion to prevent inappropriate immunosuppressive therapy.

In Case 2, suspected AIDP was referred for evaluation of Hansen's disease after inconclusive MRI findings. Despite initial negative slit skin smears, re-evaluation revealed faint erythematous patches, and histopathology confirmed borderline tuberculoid leprosy. Notably, involvement of the palms is uncommon, as these regions are traditionally considered "relatively spared zones" due to the thick epidermis and abundant fibro-fatty tissue, which provide insulation and maintain a higher nerve bed temperature, making M. leprae localization less likely. [11–13] However, exceptions occur, emphasizing the need for a thorough skin examination, even in areas typically considered resistant to infection. This case highlights the importance of considering leprosy in unexplained neuropathy, where clinical and electrophysiological findings may mimic other neurological disorders.

In Case 3, skin lesions resembled mycosis fungoides or chronic dermatoses like eczema. The absence of classical sensory impairment delayed suspicion of leprosy. However, Fite-Faraco staining demonstrated a high bacillary load, confirming lepromatous leprosy. Atypical leprosy can mimic autoimmune diseases, neuropathies, chronic dermatoses and skin malignancies, causing diagnostic delays. Early identification requires a high index of suspicion. Timely diagnosis and MDT are vital to prevent progression and disability.

CONCLUSION

Atypical leprosy presentations pose significant diagnostic challenges. A high index of suspicion, combined with clinical, histopathological, and microbiological evaluation, is crucial for timely diagnosis. Increased awareness among healthcare providers can prevent unnecessary immunosuppressive therapy, and reduce the disease burden by ensuring prompt initiation of MDT.

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REFERENCES

1. Jindal R, Shirazi N. Uncommon clinical presentations of leprosy: apropos of three cases. Lepr Rev 2016;87(2):246–51.

2. Kundakci N, Erdem C. Leprosy: A great imitator. Clin Dermatol 2019;37(3):200–12.

3. Fragozo-Ramos MC, De-León-Benito-Revollo Y, Sierra-Merlano RM. Case Report: Lucio's Leprosy Mimicking Vasculopathy: Severe Leprosy Reaction Due to Mycobacterium leprae. JCR J Clin Rheumatol 2021;27(8S):S396–7.

4. Rath D, Bhargava S, Kundu BK. Leprosy Mimicking Common Rheumatologic Entities: A Trial for the Clinician in the Era of Biologics. Case Rep Rheumatol 2014;2014:1–5.

5. Simeoni S, Puccetti A, Tinazzi E, Codella OM, Sorleto M, Patuzzo G, et al. Leprosy Initially Misdiagnosed as Sarcoidosis, Adult-Onset Still Disease, or Autoinflammatory Disease. JCR J Clin Rheumatol 2011;17(8):432–5.

6. Sampaio L, Silva L, Terroso G, Pimenta S, Brandão F, Pinto J, et al. Hansen's disease mimicking a systemic vasculitis. Acta Reumatol Port 2011;36(1):61–4.

7. Lee JY, Park SE, Shin SJ, Kim CW, Kim SS. Case of lepromatous leprosy misdiagnosed as systemic sclerosis. J Dermatol 2014;41(4):343–5.

8. Horta-Baas G, Hernández-Cabrera MF, Barile-Fabris LA, Del S Romero-Figueroa M, Arenas-Guzmán R. Multibacillary leprosy mimicking systemic lupus erythematosus: case report and literature review. Lupus 2015;24(10):1095–102.

9. Salvi S, Chopra A. Leprosy in a rheumatology setting: a challenging mimic to expose. Clin Rheumatol 2013;32(10):1557–63.

10. Rahman N, Asokan N, Ambooken B. Leprosy presenting as eczema. J Skin Sex Transm Dis 2021;3:202–3.

11. Kaur I, Indira D, Dogra S, Sharma VK, Das A, Kumar B. "Relatively Spared Zones" in Leprosy: A Clinicopathological Study of 500 Patients. Int J Lepr Other Mycobact Dis 2003;71(3):227.

12. Indira D, Kaur I, Sharma VK, Das A. Palmoplantar lesions in leprosy. Indian J Lepr 1999;71(2):167–72.

13. Anish SA. The relationship between surface temperature and dermal invasion in lepromatous leprosy. Int J Lepr Mycobact Dis Off Organ Int Lepr Assoc 1971;39(4):848–51.

Declaration of patient consent
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