SHORT COMMUNICATION

A Rare Case of Leprosy in Sweden Mimicking Drug Eruption

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Leprosy (morbus Hansen), caused by *Mycobacterium leprae*, remains a global health concern in endemic regions, with approximately 200,000 new cases reported annually (1, 2). In contrast, it is exceedingly rare in Northern Europe, where only sporadic cases are seen (2, 3). We report a patient with widespread cutaneous lesions diagnosed as lepromatous leprosy in Sweden. This case underscores the crucial role of healthcare professionals in nonendemic regions to maintain a high index of suspicion for leprosy when evaluating patients from endemic areas who present with unexplained skin lesions or neuropathy.

CASE REPORT

We report a case of a physically active 49-year-old male patient, originally from Afghanistan, who had lived in Sweden for more than 25 years but continued to travel frequently to Pakistan. He was referred to a dermatology clinic with widespread, asymptomatic pink patches, papules and plaques of 6–7 months' duration. The main clinical differential diagnosis was a suspected drug eruption related to recently initiated metformin for type 2 diabetes, but despite discontinuing metformin for several months, the lesions persisted.

Clinical examination revealed innumerable erythematous macules, convex plaques and nodules on the trunk and extremities (**Fig. 1**). On dermoscopy, yellow structureless areas and prominent vessels were observed. In the nodules, crown vessels were noted, whereas an anaesthetic patch demonstrated an absence of visible skin appendages.

All lesions retained normal sensory perception, with one exception. A solitary 2 cm macule on the lower leg exhibited total loss of tactile, nociceptive and thermal sensation, remaining stable for at least 4 decades. Subtle madarosis of the lateral eyebrows was noted. Neurological examination, including palpation of peripheral nerves, was otherwise normal.

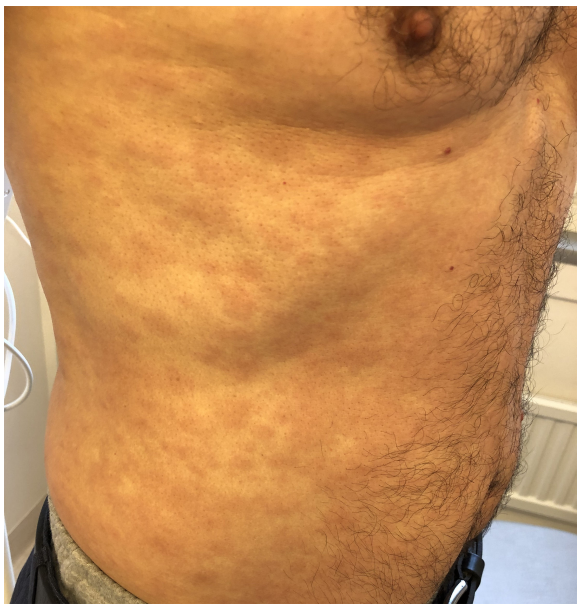

Fig. 1. Forty-nine-year-old male with widespread erythematous macules and convex plaques on the trunk with 6–7 months' duration.

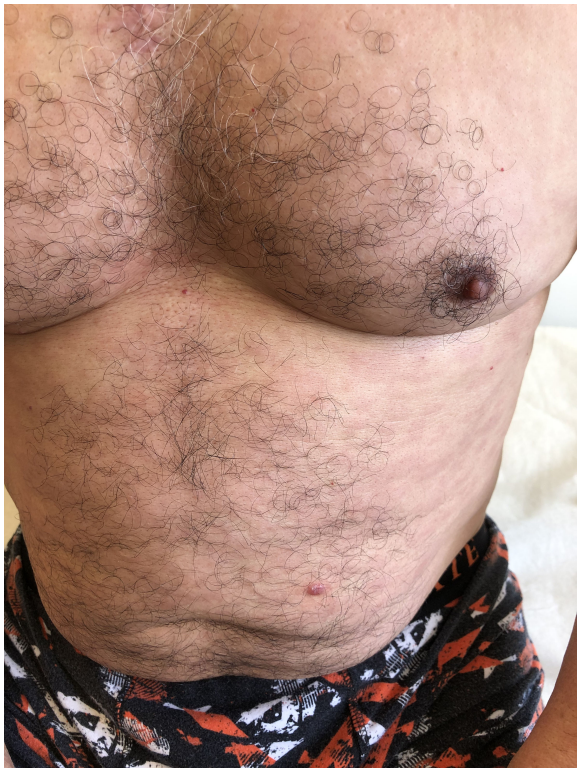

Fig. 2. Abdominal erythematous nodule with a high load of *Mycobacteria*.

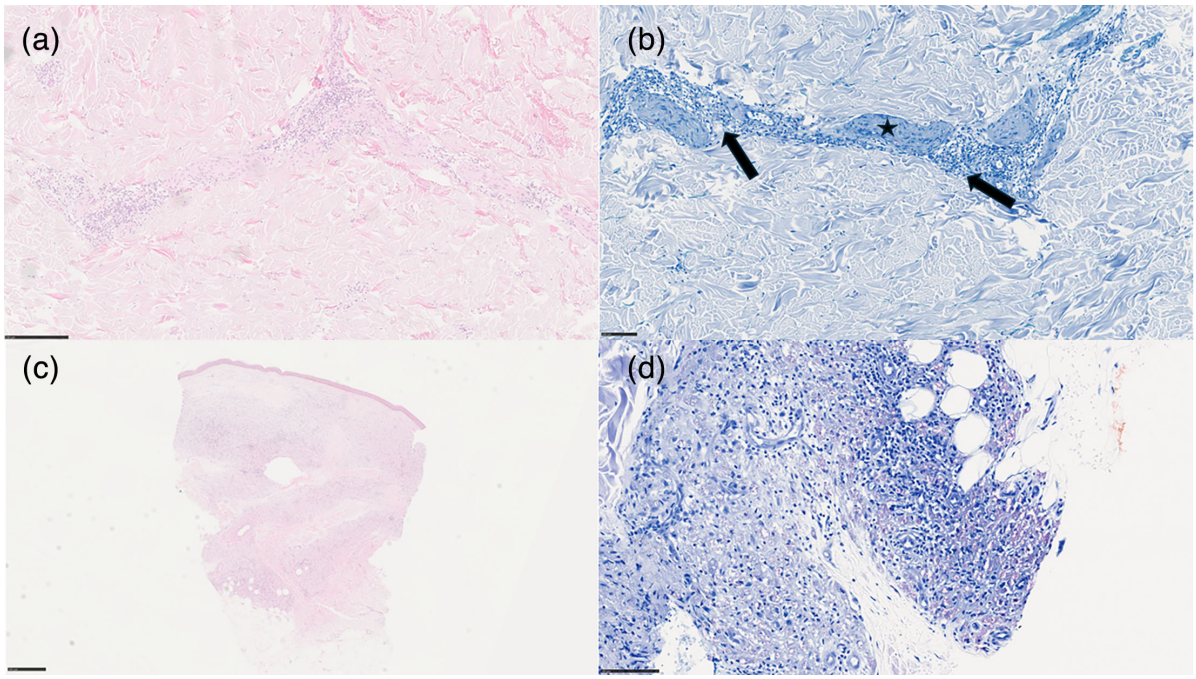

Fig. 3. (a, b) Paucibacillary leprosy on skin biopsy from an abdominal macule stained with H&E (X40) showing single perineural foamy histiocytes in reticular dermis. (b) Fite staining (X40) demonstrating acid fast bacilli in histiocytes (black arrow) surrounding a small peripheral nerve (black star). Multibacillary leprosy (c,d) on biopsy from an abdominal nodule (X2) stained with H&E (X20) (c) showing a dense dermal histiocytic infiltration with numerous *M. leprae* demonstrated by Fite staining (X40) (d).

The first biopsy from a plaque revealed a subtle perineural histiocytic infiltrate in the deep dermis with a few histiocytes containing acid-fast bacilli in Fite special stain, raising a strong suspicion of paucibacillary leprosy despite the extreme rarity of the disease in Sweden. The second biopsy, taken 3 weeks later from a nodule (**Fig. 2**), showed abundant dermal interstitial histiocytic infiltrates with acid-fast bacilli, negative in Ziehl–Neelsen and positive in Fite special stains, confirming multibacillary leprosy (**Fig. 3**a–d) (4, 5). The anaesthetic patch on the leg was clinically consistent with an indeterminate form that had persisted since adolescence. The simultaneous onset of diabetes and skin disease raised the possibility of an immunological trigger.

The patient was started on multidrug therapy with rifampicin, dapsone and clofazimine according to the World Health Organization guidelines (1). Treatment was well tolerated. After 6 months, there was a marked regression of lesions, and at 11 months, only residual post-inflammatory hypopigmentation and a few macules remained. Therapy was planned for at least 12 months, with follow-up for possible relapse or immune reactions.

DISCUSSION

This case emphasizes that leprosy, although rare in Sweden, can still be encountered, with 0–8 cases reported annually in recent years (2, 3). Awareness of the disease is crucial to prevent misdiagnosis, particularly since cutaneous and neurological manifestations may not appear until decades after exposure. In nonendemic settings, the diagnostic process relies heavily on close collaboration and high clinical suspicion among dermatologists, pathologists and microbiologists (6).

Leprosy exhibits a broad clinical spectrum, ranging from self-limited paucibacillary disease with a dominant Th1 response to multibacillary disease with strong humoral responses and high *M. leprae* antibody titres (4, 6). Paucibacillary leprosy is characterized by a low bacterial load, usually presenting with 5 or fewer skin lesions, and early nerve involvement that may cause localized sensory loss. In contrast, multibacillary leprosy shows numerous lesions, extensive nerve damage and abundant bacilli. Both paucibacillary and multibacillary lesions can occur concurrently within the same patient. The macroscopic appearance of leprosy is remarkably diverse. Cutaneous lesions may present as hypopigmented or erythematous macules, plaques, nodules or diffuse infiltration. They can closely mimic other dermatoses such as sarcoidosis, granuloma annulare, psoriasis, vitiligo, syphilis, cutaneous tuberculosis and tinea corporis. Advanced cases may show leonine facies or nerve-related complications such as ulcerations, deformities or blindness (7).

M. leprae cannot be cultured *in vitro*, which is why diagnosis relies on nonculture direct detection methods.

Molecular techniques such as PCR offer a reasonable sensitivity and a high specificity (8), but they are often unavailable, limited by the need for advanced equipment in endemic regions and by the rarity of cases in nonendemic regions. Serological tests targeting phenolic glycolipid-I provide supportive evidence but cannot confirm active disease (9). Traditional methods such as slit-skin smears and histopathology detect acid-fast bacilli but have limited sensitivity, especially in paucibacillary cases (9). In multibacillary leprosy, histopathology is often decisive for establishing the diagnosis (3). In biopsy specimens, the choice of special stain is critical. Unlike *Mycobacterium tuberculosis*, *M. leprae* is fragile, and its lipid capsule is easily damaged by strong solvents used in tissue processing. This makes Ziehl–Neelsen, the routine stain for acid-fast bacilli, unreliable. Instead, the milder Fite stain preserves mycobacterial lipids and is essential for demonstrating *M. leprae* (5).

Leprosy remains an exceptionally rare diagnosis in Northern Europe and its clinical presentation can be highly variable. This case aims to warn and educate the medical community in low incidence countries and illustrates the value of multidisciplinary collaboration in achieving timely diagnosis and appropriate management.

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