



Pretreatment Reversal Reaction in Borderline Tuberculoid Leprosy Presenting as a Unilateral Anesthetic Plaque on the Dorsum of the Foot: A Case Report

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Abstract

Background: Leprosy is a chronic infectious disease caused by *Mycobacterium leprae*, primarily affecting the skin and peripheral nerves. Type 1 leprosy reaction, also known as reversal reaction, is an acute immunological event commonly observed in borderline forms of leprosy. Although reversal reactions frequently occur during treatment, they may also develop before the initiation of multidrug therapy. Early recognition is essential to prevent nerve damage and disability. This report describes a case of borderline tuberculoid leprosy with a pretreatment reversal reaction and peripheral nerve involvement.

Case illustration: A 50-year-old woman presented with progressive redness on the dorsum of the left foot for one week, accompanied by pain and numbness persisting for one month. The lesion initially appeared as a small hypopigmented patch one year earlier, subsequently enlarging and becoming elevated, erythematous, warm, and painful. Dermatological examination revealed unilateral, well-defined erythematous anesthetic plaques on the dorsum of the left foot and toes. Neurological assessment demonstrated enlargement of the left common peroneal nerve, impaired pain and touch sensation, and reduced motor strength. Slit-skin smear examination was negative, with a bacterial index of 0/3.

Discussion: The clinical findings supported a diagnosis of borderline tuberculoid leprosy with a pretreatment reversal reaction. A negative slit-skin smear was consistent with paucibacillary disease. Early initiation of corticosteroid therapy was essential for controlling inflammation and preventing irreversible nerve impairment.

Conclusion: Pretreatment reversal reaction should be considered in patients with borderline tuberculoid leprosy, even before the initiation of multidrug therapy. Early recognition and prompt corticosteroid treatment are essential to prevent irreversible nerve damage.

Keywords: *borderline tuberculoid leprosy, corticosteroid, paucibacillary leprosy, reversal reaction.*

Background

Leprosy, also known as Hansen disease, is a chronic granulomatous infection caused primarily by *Mycobacterium leprae* and, less frequently, by *Mycobacterium lepromatosis*. The disease predominantly affects the skin and peripheral nerves, leading to a wide clinical spectrum that is determined by the host immune response. Borderline tuberculoid leprosy is defined by relatively robust cell-mediated immunity, a limited number of lesions, asymmetric nerve involvement, and frequently negative slit-skin smear results.

Although patients with borderline disease exhibit a lower bacillary load, they remain immunologically unstable and are at risk of developing acute inflammatory episodes referred to as leprosy reactions.^{1,2,3,4}

Type 1 leprosy reaction, also known as reversal reaction, is a delayed-type hypersensitivity response to *M. leprae* antigens. This reaction frequently occurs in borderline leprosy and may present as erythema, edema, tenderness, and elevation of pre-existing lesions, with or without acute neuritis.



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While reversal reactions are often identified after the initiation of multidrug therapy, they can also arise prior to treatment as part of the natural immunological variability in borderline leprosy. Timely diagnosis and intervention are essential, as nerve inflammation may progress to irreversible sensory and motor deficits. This report describes a case of borderline tuberculoid leprosy with a pretreatment reversal reaction, presenting as unilateral erythematous anesthetic plaques on the dorsum of the left foot and involving the common peroneal nerve.

Case illustration

A 50-year-old woman presented to the Dermatology and Venereology outpatient clinic with progressive redness on the dorsum of the left foot, which had enlarged over the past week. She reported pain and numbness in the same region for one month. Approximately one year prior, she observed a small hypopigmented patch on the dorsum of the left foot, which gradually increased in size and distribution. One month before presentation, the lesion became more elevated, and one week before presentation, it developed increased erythema, warmth, and pain.

The patient described the pain as a pricking sensation, especially when running water touched the dorsum of the foot during ablution. She also reported numbness on the middle toe and the dorsum of the left foot. Initially, the middle toe was painful, but the pain gradually decreased and was replaced by numbness. She had previously sought care at an internal medicine clinic, but no systemic abnormality was identified, and the symptoms did not improve.

There was no history of similar complaints, diabetes mellitus, hypertension, renal disease, hypercholesterolemia, or hyperuricemia. She denied any history of drug or food allergies. The patient reported that her mother-in-law had experienced a similar illness five years earlier and had completed treatment. Although they resided in separate households, the patient had previously assisted in caring for her mother-in-law and in cleaning her mother-in-law's utensils.

General physical examination indicated stable vital signs and no significant systemic abnormalities. Dermatological assessment identified unilateral, well-demarcated erythematous anesthetic plaques

on the dorsum of the left foot and toes, ranging from lenticular to plaque size. The lesions were sharply circumscribed and confined to the left foot region.

Peripheral nerve examination revealed enlargement of the left common peroneal (popliteal lateral) nerve with elastic consistency. No tenderness was detected upon palpation. Sensory assessment demonstrated reduced touch and pain sensation on the affected side. Motor evaluation indicated moderate strength in the left common peroneal nerve distribution, while other examined nerves remained clinically intact.

Slit-skin smear examination revealed no acid-fast bacilli, with a bacterial index of 0/3. Considering the clinical morphology, sensory impairment, peripheral nerve involvement, and negative slit-skin smear, a diagnosis of borderline tuberculoid leprosy with type 1 leprosy reaction (reversal reaction) was established. Differential diagnoses included borderline leprosy, tuberculoid leprosy, creeping eruption, cellulitis, and tinea corporis.

Histopathological examination was planned but had not yet been performed at the time of this report. The patient was treated with paucibacillary multidrug therapy consisting of rifampicin 600 mg once monthly under supervision and dapsone 100 mg daily for six months, together with methylprednisolone 8 mg three times daily after meals, which was gradually tapered according to clinical response and nerve function assessment. Glycerin lotion, paracetamol, and patient education regarding foot protection were also provided. She was educated regarding treatment adherence, foot protection, routine self-examination for wounds or ulcers, skin hygiene, regular moisturizer use, monthly follow-up, and immediate return to the clinic if symptoms worsened.

The overall prognosis was favorable for survival and cutaneous recovery, but remained guarded for neurological function due to peripheral nerve involvement. At six-month follow-up, erythema and edema had markedly decreased, with residual post-inflammatory hyperpigmentation. Pain had improved; however, residual sensory impairment persisted, indicating only partial neurological recovery. No ulceration or new reactional episodes were observed. Written informed consent was obtained from the patient for publication of this case report and accompanying clinical images.

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Figure 1. An initial hypopigmented-to-erythematous anesthetic plaque was observed on the dorsum of the left foot prior to therapy.



Figure 2. The pre-existing lesion became more extensive, increasingly erythematous, and more elevated, consistent with an acute reversal reaction in borderline tuberculoid leprosy.



Figure 3. Lateral view showing erythematous plaque on the dorsum of the left foot.

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Figure 4. After six-month follow-up, the lesion on the dorsum of the left foot appeared faded, with reduced erythema and residual post-inflammatory hyperpigmentation, suggesting clinical improvement.

Discussion

This case illustrates borderline tuberculoid leprosy presenting with pretreatment reversal reaction and focal peripheral nerve involvement. This case highlights an uncommon diagnostic scenario in which pretreatment reversal reaction mimicked other localized inflammatory dermatoses, emphasizing the importance of careful sensory and peripheral nerve examination in suspected paucibacillary leprosy. The diagnosis was established clinically based on the cardinal signs of leprosy, including anesthetic erythematous plaques, asymmetric distribution, sensory impairment, and enlargement of the left common peroneal nerve. These findings represent cardinal features of leprosy, particularly when skin lesions are associated with peripheral nerve dysfunction.^{1,2,5}

Borderline tuberculoid leprosy belongs to the immunologically unstable borderline spectrum. Patients in this spectrum may experience changes in cell-mediated immunity, resulting in acute inflammation of existing lesions and nerves. Reversal reaction is characterized by increased erythema, edema, tenderness, and elevation of pre-existing lesions, often accompanied by neuritis. In the present case, the patient had a chronic hypopigmented patch that became erythematous, elevated, warm, painful, and associated with sensory and motor impairment before multidrug therapy was initiated. This temporal pattern supports the diagnosis of pretreatment reversal reaction rather than a drug-induced inflammatory event.^{3,4,6} Recent immunopathogenesis evidence further supports that type 1 leprosy reaction is driven by coordinated CD4⁺ T-cell responses and pro-inflammatory cytokine activity that may contribute to neural inflammation and nerve impairment.⁷

Pretreatment reversal reaction is clinically significant because it can be misdiagnosed as

cellulitis, dermatophyte infection, allergic dermatitis, or other inflammatory dermatoses. In this case, cellulitis was initially considered due to erythema, warmth, and pain. However, the presence of a chronic hypopigmented anesthetic patch, well-defined plaque morphology, unilateral nerve enlargement, and sensory loss were more indicative of leprosy. Tinea corporis was deemed less likely because the lesion lacked a characteristic annular scaly border and was associated with anesthesia. Creeping eruption was also unlikely, as there was no evidence of a serpiginous migratory track or intense pruritus. Both borderline leprosy and tuberculoid leprosy were considered within the leprosy spectrum; however, the clinical presentation was most consistent with borderline tuberculoid leprosy, given the limited, asymmetric lesions, prominent nerve involvement, and negative smear findings.

A negative slit-skin smear in this case did not exclude leprosy. Paucibacillary and borderline tuberculoid leprosy frequently present with low bacillary loads, making acid-fast bacilli undetectable on routine slit-skin smear examination. Consequently, diagnosis depends primarily on clinical assessment, particularly the presence of anesthetic skin lesions and peripheral nerve abnormalities. Histopathological examination can support the diagnosis by revealing granulomatous inflammation and nerve involvement. However, treatment should not be delayed when clinical features are strongly suggestive and nerve function is at risk.^{5,8,9}

Peripheral nerve involvement represents the primary determinant of disability in leprosy. The common peroneal nerve is of particular clinical importance, as its impairment may result in dorsiflexion weakness, altered gait, increased risk of trauma, ulceration, and persistent functional limitations. In this case, enlargement of the left common peroneal nerve, sensory deficits, and reduced motor strength

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indicated active nerve involvement. Consequently, the functional prognosis was guarded. Early recognition and prompt treatment of neuritis are critical to preventing irreversible nerve damage.^{6,10,11}

Management for this patient included paucibacillary multidrug therapy to address the underlying infection and systemic corticosteroids to control reversal reactions and nerve inflammation. Corticosteroids are widely utilized for clinically significant type 1 reactions, especially when pain, edema, neuritis, or nerve function impairment occurs. Analgesics and emollients were provided for symptomatic relief and maintenance of skin barrier integrity. Patient education was emphasized, focusing on adherence to multidrug therapy, foot protection, self-inspection, and prompt reporting of symptom progression.^{10,11,12,13}

This case highlights that reversal reactions can occur prior to the initiation of multidrug therapy and should be considered in patients presenting with acute inflammation of pre-existing anesthetic lesions. In endemic regions, the presence of unilateral inflammatory plaques accompanied by sensory disturbance and peripheral nerve enlargement should prompt evaluation for leprosy, even if the slit-skin smear is negative.¹⁴ Early diagnosis and prompt initiation of corticosteroid therapy are essential to minimize the risk of permanent nerve impairment and disability.^{15,16} Furthermore, additional reviews and regional data underscore the significance of early recognition of type 1 reactions, immunologically informed management, and disability prevention in leprosy care.^{17,18,19}

One limitation of this report is the lack of histopathological confirmation and molecular testing. Nevertheless, treatment was initiated based on clinical findings that strongly indicated leprosy with active nerve involvement, as delaying therapy could increase the risk of permanent disability.

Conclusion

Borderline tuberculoid leprosy may present with pretreatment reversal reaction, characterized by acute inflammation of pre-existing lesions and peripheral nerve involvement. A negative slit-skin smear does not rule out paucibacillary leprosy when clinical features are strongly suggestive. Early recognition of anesthetic inflammatory plaques, careful nerve examination, prompt multidrug therapy, and timely corticosteroid treatment are essential to prevent irreversible nerve damage and disability.

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Author Contributions

All authors act as guarantors of the manuscript. JF contributed to the conception and design of the case report, data collection, clinical assessment, and manuscript drafting. KT contributed to literature review, data interpretation, manuscript drafting, and critical revision. RN contributed to clinical evaluation, diagnosis, patient management, and follow-up assessment. JF supervised manuscript development, provided dermatovenereology expertise, critically revised the manuscript, and approved the final version. All authors have read and approved the final manuscript and agree to be accountable for all aspects of the work.

Conflict of Interests

No conflict of interest

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