

## Concurrent Lepra Reactions; Type 1 and Type 2 in a young male patient with borderline leprosy: Clinical and Histopathological correlation

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### Abstract:

Leprosy reactions are acute exacerbations of the signs and symptoms of leprosy occurring during the natural course of the disease and during or after treatment. Left untreated or improperly managed, reactions can lead to severe nerve function impairment and subsequent disabilities. Leprosy reactions continue to pose a significant and enduring challenge. Type 1 leprosy reaction and type 2 leprosy reaction are substantial contributors to nerve impairment and the subsequent development of enduring impairments. We are reporting a case of Concurrent Lepra reactions (Mixed type 1&2) in an 18-year-old male patient and its histopathological correlation.

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## Introduction

Leprosy is a chronic disease that remains active for a long time. During the long course of activity of the disease, in several patients, there are acute bouts of exacerbation characterized by a sudden increase in the activity of the disease. Reactions can be defined as sudden tissue responses resulting from the liberation of the bacilli or their products into the tissues.

**Type 1 Lepra Reaction [1]**

Immunological basis: Cell-mediated immunity ↑ (delayed hypersensitivity).

Seen in: Borderline spectrum (BT, BB, BL).

Clinical features: Acute inflammation of pre-existing lesions (red, swollen, tender). New lesions may appear. Nerve pain, tenderness, neuritis → can lead to sudden nerve function loss.

**Type 2 Lepra Reaction (ENL) [2]**

Immunological basis: Immune complex deposition (Type III hypersensitivity).

Seen in: Lepromatous (LL) and borderline lepromatous (BL).

Clinical features:

Painful erythematous nodules (ENL lesions), Fever, and malaise. Systemic involvement: lymph nodes, joints, eyes, testes, and kidneys.

**Both Reactions in the Same Patient [3]**

Possible but highly uncommon.

Usually occurs in Borderline Lepromatous (BL) patients, since they sit in the “overlap” between Type 1 (borderline instability) & Type 2 (because of higher bacterial load).

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Mechanism:

Type 1: due to a shift in cell-mediated immunity.

Type 2: due to immune complex-mediated inflammation.

### Case Report

An 18-year-old male patient presented to our OPD with complaints of multiple erythematous annular plaques with sharply raised inner margins and outer margins merging with normal skin over bilateral upper & lower limbs, back (3 lesions), buttocks (5 lesions), and face. The lesions were prominent, erythematous, edematous, and warm to the touch. There was hypoanaesthesia over lesions except lesions over the face. Infiltration was present over the left pinna.

There were multiple ulcers over the face (as seen in figure 3), buttocks, and bilateral upper and lower limbs (as seen in figure 1). Some ulcers were associated with central crusting (as seen in figure 2) and necrosis over the floor. Ulcers were tender on palpation.

There was also a complaint of fever, which was intermittent, low-grade, and associated with an

evening rise of temperature. There was pitting edema of the bilateral forearms associated with joint pain.

There was a history of nasal crusting not associated with epistaxis. The patient also gave a history of a single hypoanaesthetic patch over the left elbow for 14 years (as seen in Fig.4). There was a history of infiltration of the left pinna 1 year back, for which the Patient took MDT for a duration of 1 month.

On examination:

The general appearance was ill-looking. There was lymphadenopathy of the submental and jugulo-diaphragmatic lymph nodes.

The right ulnar and radial nerves were thickened more than the left with grade III neuritis. The left superficial radial nerve was thickened with grade III neuritis. The right common peroneal nerve was thickened more than the left common peroneal nerve with Grade III neuritis. The right posterior tibial nerve was thickened with grade II neuritis.

On Motor examination, the Froment sign and card test were found to be positive on bilateral side. Wartenburg sign was present in both hands and there was wasting of bilateral thenar muscles.

HPE findings: see Fig.5&6



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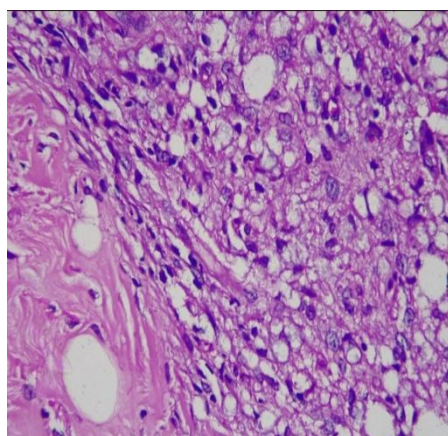
**Fig 1: Multiple erythematous, tender plaques with areas of ulceration, crusting and PIH distributed symmetrically over thighs and knees, s/o Type 2 reaction**



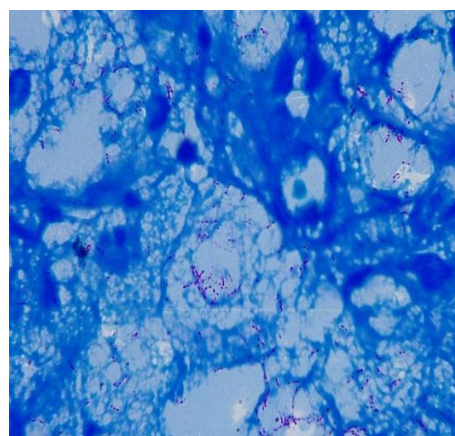
**Fig 2: Erythematous plaque with central ulceration surrounded by PIH of old healed lesions, s/o Type 2 reaction**



**Fig 3: Pre-existing lesions over cheeks have become more erythematous, edematous, prominent, shiny, warm, s/o Type 1 reaction**



**Fig 4: Skin lesion over elbow becomes dry and scaly, flattens to leave behind a wrinkled hypopigmented surface, s/o subsiding Type 1 reaction**



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**Fig 5: Biopsy shows loose aggregates of foamy epithelioid cells around blood vessels and eccrine glands**

**Fig 6: Fite Faraco stain showing both fragmented and intact AFB**

### Discussion

Leprosy is a chronic disease, which remains active for a long time. During the long course of activity of the disease, in several patients, there are acute bouts of exacerbation characterized by sudden increase in the activity of the disease. The word reactions in leprosy are used to describe an episode in a major disease. They can occur in tuberculoid spectra and lepromatous spectrum. Reactions can be defined as sudden tissue responses, resulting from the liberation of the bacilli or their products, into the tissues. Manifestation can be local or systemic. Leprosy is a quiescent disease unless, it is complicated by reactions. Reactions make the patient to seek medical attention. Reactions are responsible for the most permanent nerve damage, deformity and disability. Borderline group of patients are the most vulnerable group with the highest risk of developing Type 1 reaction. [4] Borderline lepromatous and borderline patients have a higher risk than borderline tuberculoid patients. [5] However, reactions occur earlier in Borderline tuberculoid patients. [6] In this study the patient was in borderline tuberculoid spectrum and he presented with reaction thirty years after the appearance of initial lesions. Patients with multiple and disseminated patches involving larger body areas and multiple nerve involvement are at increased risk of developing type 1 reaction. [7], [8], [9] Nerve enlargement, tenderness and paraesthesia on palpation are associated with an increased risk of a reaction. [10] Reaction may be present at the time of presentation or develop during treatment and even after release from treatment. [11] At a fully monitored field control unit at Koraput leprosy eradication project, the data showed that type 1 reaction occurred in 3.9 percent of borderline cases. Of the borderline cases, borderline borderline (BB) type showed maximum rate of reactions. The

borderline lepromatous (BL) type can present with both type 1 and type 2 reactions with total incidence of 12.8 percent. While borderline tuberculoid (BT) type constituted 74 percent of the total cases, Type 1 reaction occurred in only 3.1 percent cases. Reaction also occurred in 0.8 percent of release from treatment cases. [12] Type 1 reaction occurs as a result of increased activity of the body's immune system, particularly cell mediated immune response fighting the leprosy bacillus or remnants of dead bacilli. Clinical features include reaction may be the first presenting sign of the disease and usually last for few weeks to few months. General condition of the patient is satisfactory. Usually there is no fever and patient does not feel ill. There will be increase in inflammation in skin lesions or nerves or both. Skin lesions becomes erythematous and/or edematous, and may ulcerate. Edema of hands, feet and face can also be a feature of reaction. Systemic symptoms are rare. Neuritis is present if the patient has nerve pain, paraesthesia or tenderness, which precedes nerve function impairment. This is seen in our case. If left unnoticed it may lead to silent neuropathy. Immunology of Type 1 reaction is associated with upregulation of interferon-gamma production leading to granuloma formation, enhanced macrophage microbicidal activity and inflammation. This reaction is associated with enhanced bacterial clearance.

Erythema nodosum leprosum or type 2 reaction is an acute inflammatory condition involving a TNF alpha and immune complex mediated immune response. Type 2 reaction occurs mostly during the course of antileprosy treatment. A few cases present for the first time with features of reaction before leprosy is diagnosed and treatment started. Here also the patient presented with features of reaction before leprosy is diagnosed and before the treatment has started. Most frequently seen in lepromatous leprosy patients. TNF-alpha is known to be a pyrogen which may be responsible for the



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rise of temperature and further tissue damage during type 2 reaction.[13] Precipitated by physical or psychological stress, pregnancy, puberty, lactation or alcohol intake. Erythema nodosum leprosum precedes fever, joint pain, malaise, and headache. Subnormal temperature in morning and high in evening. The fresh crops of erythema nodosum leprosum lesions usually appear in the evening when endogenous cortisol production is at its lowest.[14] Skin lesions are brightly erythematous, slightly raised nodules or plaques, variable in size. Nodules measuring 0.5 to 3cm on the face, thigh, legs. Nodules disappear in few days and successive crops appear. Upon resolution, leave purplish stain and desquamate. Neuritis is less compared to type 1 reaction

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