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

Prem Prakash Pravakar[1], Md Mobarak Hussain[2], Hemant Kumar Singh[3], Sumit Shaw[4]
[1]HOD & Associate Professor, Dept of Skin and V.D, Anugrah Narayan Magadh Medical College and Hospital, Gaya
[2]Senior Resident, Dept of Skin and V.D, Anugrah Narayan Magadh Medical College and Hospital, Gaya
[3]PG-3, Dept of Skin and V.D, Anugrah Narayan Magadh Medical College and Hospital, Gaya
[4]PG-2, Dept of Skin and V.D, Anugrah Narayan Magadh Medical College and Hospital, Gaya

Corresponding Author

Prem Prakash Pravakar
HOD & Associate Professor, Dept of Skin and V.D, Anugrah Narayan Magadh Medical College and Hospital, Gaya

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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: Heightened 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 Type-1 &Type-2 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 is due to a shift in cell-mediated immunity.
Type2 is mostly 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 hypoesthesia over the lesions, except for those on 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 Figure 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-diagastric 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 both sides. Wartenberg's sign was positive in both hands, and there was wasting of the bilateral thenar muscles.
HPE findings: Shown in Fig.5&6

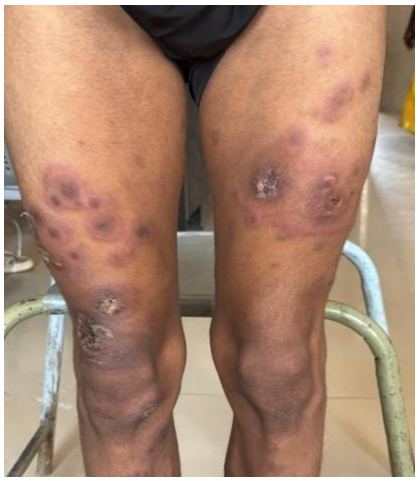

Fig 1: Multiple erythematous, tender plaques with areas of ulceration, crusting and PIH distributed symmetrically over thighs and knees, s/o - Type 2 reaction

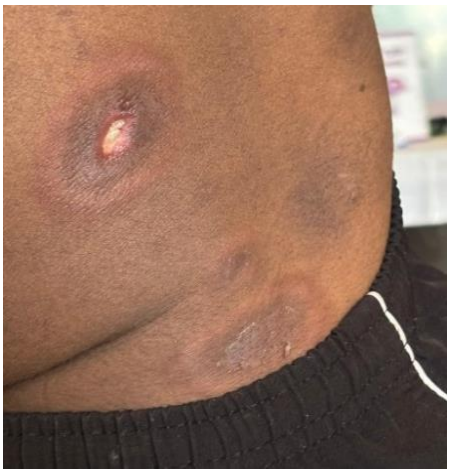

Fig 2: Erythematous annular plaque with central ulceration surrounded by PIH of old healed lesion and erythematous margin, s/o - Type 2 reaction.

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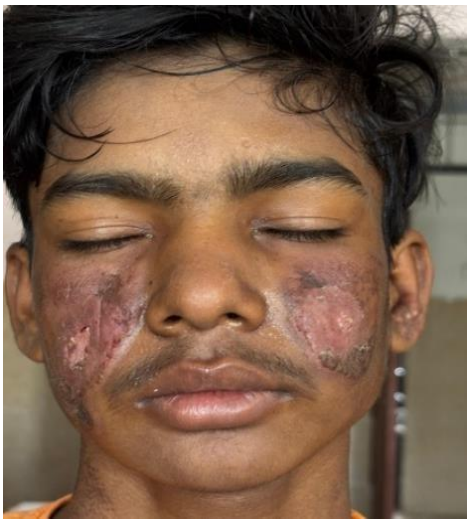

Fig 3: Pre-existing lesions over cheeks have become more erythematous, edematous, prominent, shiny, warm, s/o Type 1 reaction

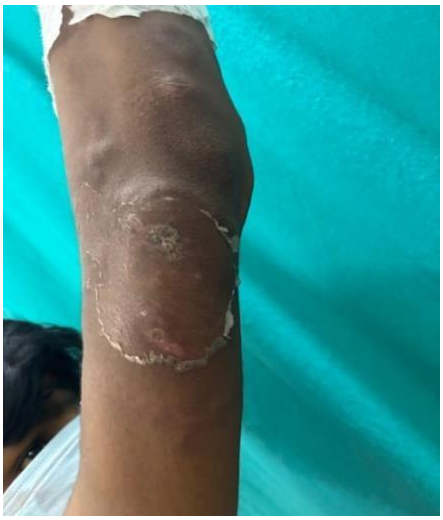

Fig 4: Skin lesion over elbow becomes dry and scaly, flattens to leave behind a wrinkled hypopigmented surface, s/o subsiding Type 1 reaction

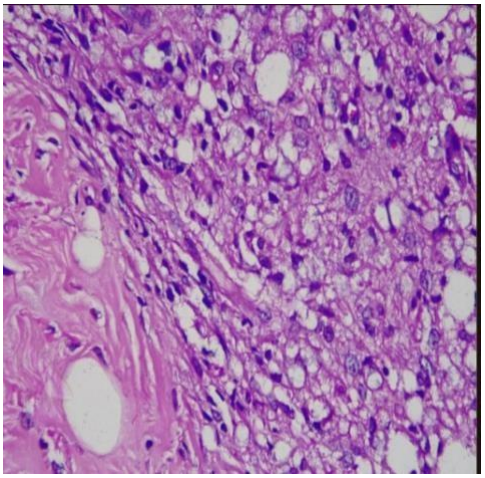

Fig 5: HPE of Skin biopsy (H&E stain, 100X) showing loose aggregates of foamy epitheloid cells around blood vessels and eccrine glands.

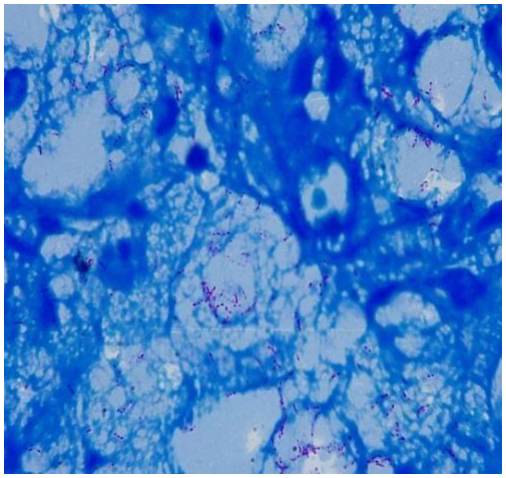

Fig 6: Fite Faraco stain showing both fragmented and intact AFB in plenty.

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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 a 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 spectra. 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 borderline patients have a higher risk than borderline tuberculoid patients. [5] However, reactions occur earlier in Borderline tuberculoid patients. [6] Studies have shown that the patients in the borderline tuberculoid spectrum have presented with a 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 the 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 the maximum rate of reactions. The borderline lepromatous (BL) type can present with both type 1 and type 2 reactions, with a 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 of the cases. Reaction also occurred in 0.8 percent of cases released from treatment. [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, which may be the first presenting sign of the disease and usually lasts for a few weeks to a few months. The general condition of the patient is satisfactory. Usually, there is no fever, and patient does not feel ill. There will be an increase in inflammation in skin lesions or nerves or both. Skin lesions become erythematous and/or edematous and may ulcerate. Edema of hands, feet, and face can also be a feature of a 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, the patient presented with features of reaction before leprosy was diagnosed and before the treatment had started. Most frequently seen in lepromatous leprosy patients. TNF-alpha is known to be a pyrogen that may be responsible for the 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 the morning and high in the 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, and legs. Nodules disappear in a few days, and successive crops appear. Upon resolution, leave a purplish stain and desquamate. Neuritis is less as compared to type 1 reaction.

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