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Case Report

Immune Zones Involvement in Borderline Leprosy with Type 1 Lepra Reaction

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Leprosy is a chronic granulomatous infectious disease caused by *Mycobacterium leprae*, which typically affects cooler areas of the body. Certain regions such as the palms, soles, scalp, genitalia, groins, lumbosacral region, and axillae are considered relatively immune or spared zones. This case report describes an unusual presentation of mid-borderline leprosy (borderline borderline BB) with type 1 lepra reaction involving the palm and scalp.

Keywords: Borderline Leprosy, Immune Zones, Palm, Scalp, Type 1 Lepra Reaction.

Introduction

Leprosy is known for its clinical diversity, influenced by the host's immune response and the bacillary load. Traditionally, the disease tends to spare certain areas of the body known as "immune zones" including the scalp, palms, soles, genitalia, axillae, and lumbosacral regions owing to their relatively higher surface temperature, thicker epidermis, and subcutaneous insulation, which are believed to inhibit the growth of *Mycobacterium leprae* (Rajashekar et al 2009). However, this traditional understanding is increasingly being challenged by reports of atypical involvement in these so-called protected regions, especially during lepra reactions. This report presents a case of mid-borderline leprosy with involvement of palm and scalp.

Case Report

A 35-year-old male presented to our outpatient department 5 months ago with complaints of multiple erythematous skin lesions involving the face, occipital scalp, chest, and back, along with sensory loss over the right hand for 7 months. Cutaneous examination revealed asymmetrically distributed four erythematous, infiltrated plaques over the face; a solitary 2×2 cm erythematous infiltrated plaque over the left side of occipital scalp; and similar asymmetrically distributed plaques over the chest and back. Sensory examination revealed hypoesthesia over the plaques on the chest and back, as well as reduced sensation in the ulnar nerve distribution over both the palmar and dorsal surfaces of the right hand. On palpation, the right ulnar

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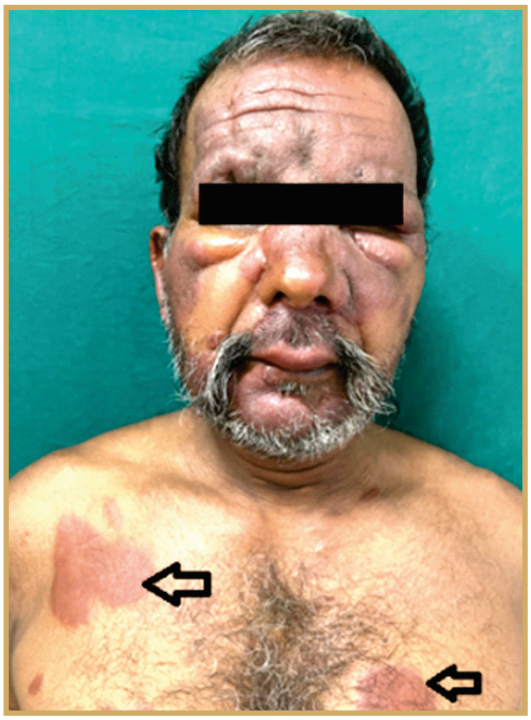

Fig. 1 : Erythematous inflamed plaques covering whole face with periorbital and upper lip oedema. Inflamed erythematous plaques over chest (Black arrows).

nerve was noted to be grade 1 thickened, non-tender, and without motor deficit at the time of presentation. Slit skin smear examination was negative for acid-fast bacilli. A 3 mm punch biopsy taken from a lesion on the trunk revealed epithelioid cell granulomas with occasional giant cells. Based on the histopathology, a diagnosis of borderline tuberculoid leprosy was made. The patient was initiated on multibacillary multidrug therapy (MDT-MB) for adult, comprising rifampicin 600 mg once monthly, clofazimine 300 mg once monthly and 50 mg daily, and dapsone 100 mg daily for 12 month**s**. He was also counselled on the importance of limb protection to prevent trauma-induced trophic ulcers due to

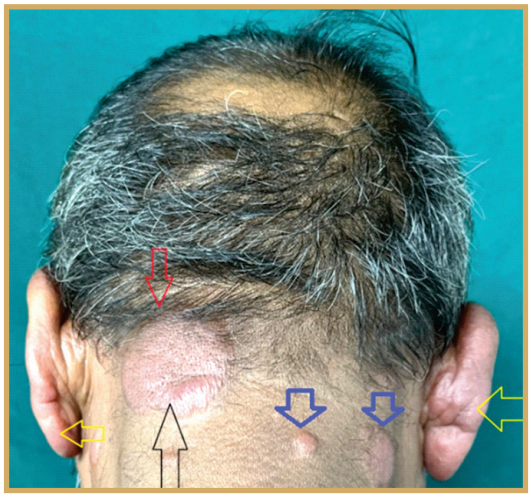

Fig. 2: Erythematous infiltrative tender plaque measuring around 5x6 cm was seen on lower occipital area extending down to neck (Red and Black arrows), Bilateral ear pinna with lobules erythematous infiltration (yellow arrows) (new onset), Newer erythematous papules and plaques over back of neck (blue arrows).

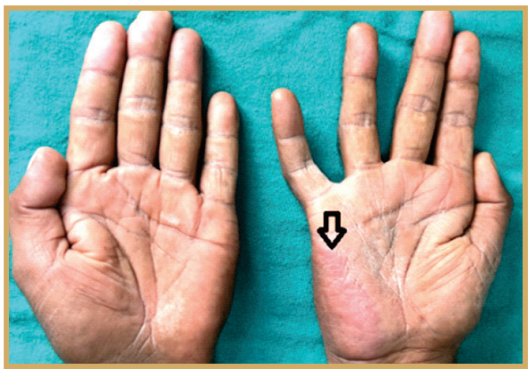

Fig. 3 : Erythematous tender macule over medial side of right palm.

hypoesthesia. After 5 months of compliant MDT, the patient returned with complaints of pain, swelling, and exaggeration of pre-existing lesions over the face, chest, back, and occipital scalp.

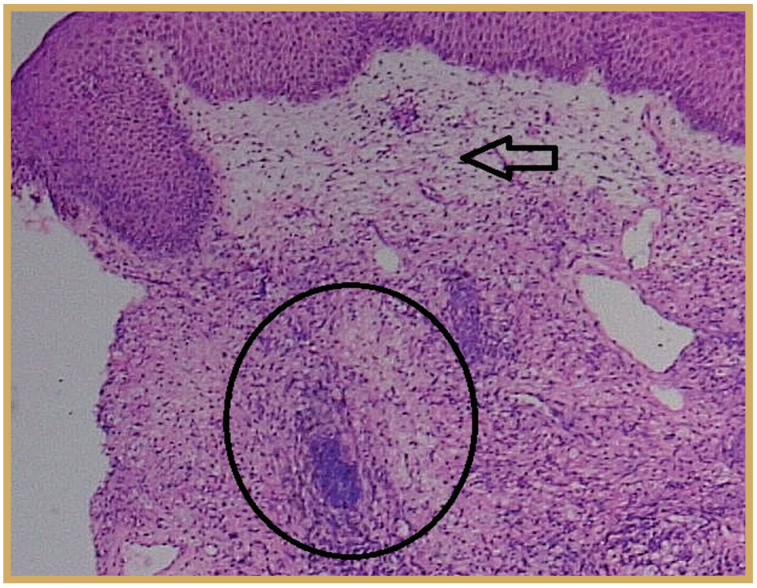

Fig. 4 : Histopathology section on haematoxylin and eosin stain (40X): Mild papillary oedema (black arrows) and lymphocytic adnexal infiltrates (black encircled).

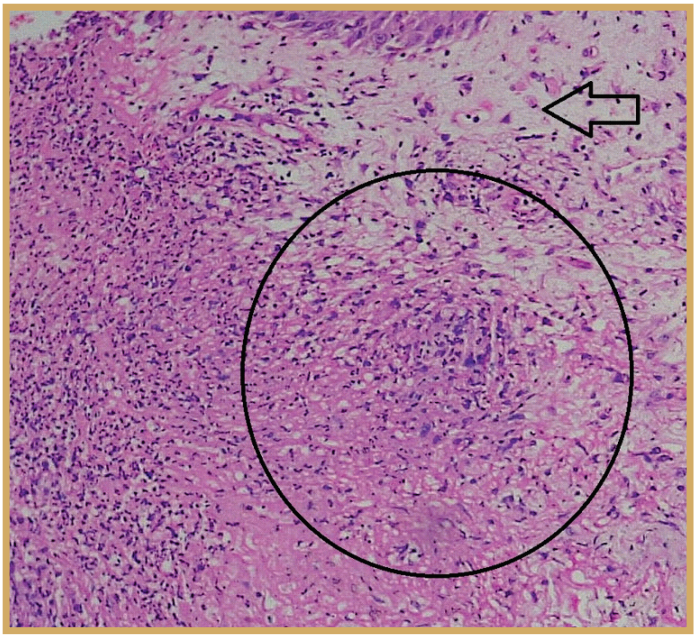

Fig. 5 : Histopathology section on haematoxylin and eosin stain (100X): Mild papillary oedema (black arrows) and ill-defined epithelioid cells associated with necrosis. (black encircled).

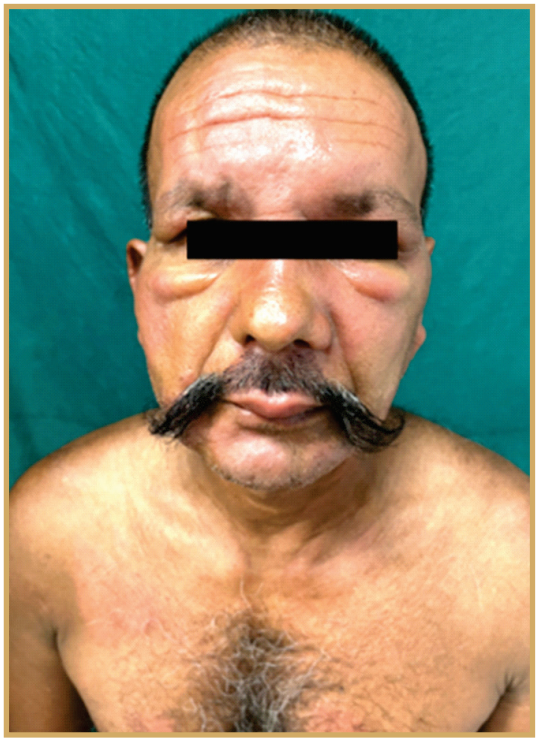

Fig. 6 : Decrease in the inflammation, erythema and oedema of plaques over face and chest after starting the patient on corticosteroids.

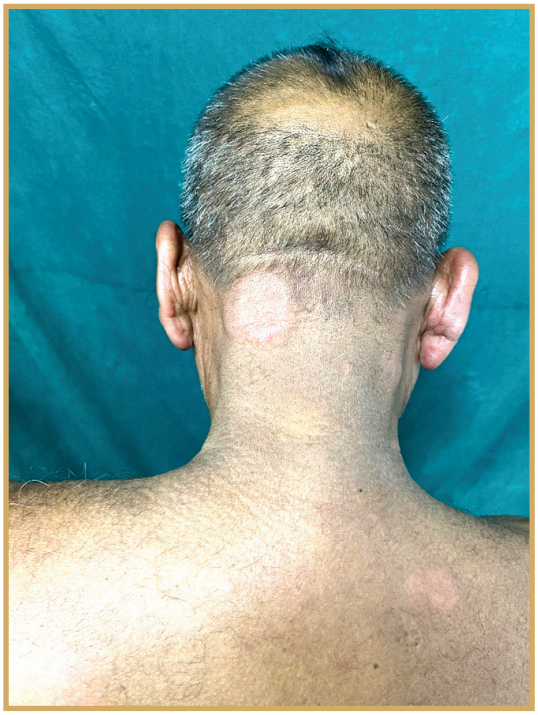

Fig. 7 : Decrease in the inflammation, erythema and oedema of plaques over back of neck, scalp and back after starting the patient on corticosteroids.

He also reported the appearance of new lesions over both ear lobules, back of neck and, the right palm, along with extension of the occipital lesion to the nape of the neck. These symptoms were accompanied by pain near the right elbow radiating to the forearm, suggestive of neuritis. There was no history of fever, systemic illness, recent infections, or emotional or physical stress that could have triggered the onset of these lesions. On examination, the face showed erythematous, infiltrative, and inflamed and tender plaques involving the entire facial area, with associated periorbital and upper lip oedema (Fig. 1). Additionally, infiltrated nodular lesions were present over both ear lobules (Fig. 2, yellow arrows). The occipital plaque had increased in size to approximately 5×6 cm, now extending to the nape of the neck (Fig. 2, red and black arrows), new infiltrative erythematous lesions over back of neck (Fig. 2, blue arrows). The plaques on the chest were inflamed (Fig. 1, black arrows), and a new erythematous macule was noted over the medial aspect of the right palm which was associated with tenderness (Fig. 3). Neurological evaluation revealed hypoesthesia over the inflamed occipital plaque accompanied by hair loss, as well as hypoesthesia over the trunk plaques. In addition, sensory impairment

persisted over the ulnar distribution in the palm extending to right medial side of forearm. The right ulnar nerve was tender and thickened on palpation, with motor examination showing loss of adduction of the right little finger. A skin biopsy obtained from the scalp lesion demonstrated papillary dermal oedema with several ill-defined epithelioid cell granulomas and lymphocytic infiltration of adnexal structures (Figs. 4 and 5). Based on clinical and histopathological findings, a diagnosis of mid-borderline leprosy with type 1 lepra reaction with right ulnar nerve neuritis was established.

The patient was initiated on oral prednisolone at 40 mg daily, which was gradually tapered by 5mg every 2nd week to a dose of 10 mg/ day over a course of 12 weeks, while MDT-MB (Adult) was continued. Supportive care and detailed education about disease progression, nerve care, and self-inspection for early signs of disability were provided. The patient showed marked clinical improvement by the 8th week of corticosteroid therapy, with reduction in erythema, oedema, pain, and partial recovery of motor function in the affected limb (Figs. 6 and 7). He continues to be monitored with biweekly follow-ups.

Discussion

Awareness of uncommon presentations is essential to keep the treating physicians/ specialists aware of such forms. Our case presented with infiltrated erythematous plaques over the face, chest, back, and a solitary lesion on the left side of the occipital area of the scalp was diagnosed with borderline tuberculoid (BT) leprosy and was started on MDT-MB (Adult). After 5 months of therapy, the patient returned with swollen and tender pre-existing lesions, along with new lesions over both ear lobules, the back of the neck, and the right palm, as well as extension of the occipital lesion to the nape of the neck. This pattern is similar to a case described by Sharma et al (2019), who reported depigmented lesions extending from the occipital area to the nape of the neck (Sharma et al 2019). On the basis of current clinical features and histopathological findings, patient was subsequently diagnosed with Mid-Borderline leprosy with type 1 lepra reaction. Scalp involvement can also be in form of alopecia (Jadhav & Zawar 2015) or infiltrative plaques which was seen in our patient as well, particularly in BT and borderline lepromatous patients (Dogra et al 2002). Scalp involvement in leprosy is infrequently reported. This might be due to high local temperature. Oteig & Pinegro (1960) classified leprotic alopecia into

1. Diffuse alopecia.
2. Regional alopecia localized to temple.
3. Circumscribed alopecia.
4. Mitsuda's type.
5. Wig-type.

In addition to scalp extension, our patient developed a new erythematous macule on the right medial palm during the type 1 reaction. The involvement of the palms and soles in leprosy are documented in the literature (Dabas et al 2021). Although these areas are cooler than the rest of the body, richly innervated, and more susceptible to trauma factors that would typically increase their vulnerability to leprosy lesions, they possess unique characteristics that offer protection. Firstly, the epidermis of the palmoplantar region is significantly thicker (approximately 1.5 mm) compared to other superficial skin areas, contributing to a relatively higher surface temperature. Secondly, the presence of abundant fibrofatty tissue in these regions provides insulation, maintaining a warmer nerve bed temperature. This elevated temperature in the

palmoplantar nerve beds is thought to reduce the likelihood of *Mycobacterium leprae* localization in these areas (Enna et al 1974). However, studies have noted that in type 1 reactions, previously subclinical lesions in immune zones may become clinically apparent, likely due to heightened cell-mediated immunity (Rajashekar et al 2009). Our findings align with this hypothesis, suggesting that immune reactivation may unmask latent infection even in traditionally spared regions. In our patient, following the onset of the type 1 lepra reaction, the patient developed marked neuritic features including tenderness of the right ulnar nerve and a progressive increase in sensory loss. Initially restricted to the palm, hypoesthesia extended to involve the medial aspect of the forearm, indicating worsening peripheral nerve inflammation. These neurologic changes paralleled the appearance of new lesions and the aggravation of previously existing plaques. Histopathological examination of the scalp lesion, an immune zone, revealed papillary dermal edema with ill-defined epithelioid cell granulomas and lymphocytic infiltration of adnexal structures, consistent with features of a type 1 lepra reaction in borderline leprosy. Although biopsy was performed from the scalp, histological confirmation from the palm was not pursued due to patient preference and the strong correlation between clinical features and response to corticosteroid therapy.

An especially notable and unusual observation in our case was the development of infiltrated nodular lesions over both ear lobules during the type 1 reaction phase. While ear involvement is more commonly associated with lepromatous leprosy due to its bacillary load and cooler temperature preference, its presence in a patient with borderline leprosy experiencing a type 1 reaction is rarely documented. The development of infiltrated nodular lesions over the ear lobules in our patient, may be explained by the immunological instability associated with type 1 lepra reaction. This instability in cellular immune response likely unmasked subclinical lesions in cooler peripheral areas such as the ear lobes, where *Mycobacterium leprae* may have remained sub clinically dormant. Ear involvement in form of plaques in multibacillary mid-borderline leprosy with type 1 lepra reaction has also been described by Chew & Woods (2021).

Conclusion

This case challenges the traditional understanding of immune zones in leprosy by documenting simultaneous involvement of the scalp and palm areas generally considered resistant to *Mycobacterium leprae*. In the current post-elimination era, recognizing such atypical manifestations is essential for accurate diagnosis and timely treatment. Treating doctors including specialist dermatologists need to be aware of such presentations.

Acknowledgements

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