



Letter to the Editor

Leprosy presenting as eczema

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Eczemas that have been commonly reported in patients with leprosy are asteatotic eczema (which may, in turn, be secondary to ichthyosis associated with the disease or due to treatment with clofazimine) and contact dermatitis due to ill-informed application of topical agents.^[1]

A 17-year-old male presented with a few asymptomatic, mildly exudative and crusted lesions on the right upper limb of 2 months duration [Figure 1]. It was diagnosed and treated as nummular eczema previously. He had no prior history of eczema or atopy. There was no history of any topical applications. Examination in bright daylight revealed that the exudative lesions were in the center of hypopigmented macules. There were six partly defined hypopigmented macules of size 1 × 1 cm to 4 × 2 cm on the flexor aspect and lateral border of the right forearm. Some of these lesions had well-defined eczematous plaques in their centers. Sensations (touch, pain, and temperature) on the hypopigmented macules and the eczematous plaques were impaired. Infraorbital, ulnar, ulnar cutaneous, radial cutaneous, and posterior tibial nerves were thickened on the right side. He did not have diffuse infiltration of normal skin including face and ears, mucosal lesions, pedal edema or glove and stocking type of anesthesia.

Slit skin smear (SSS) from lesions showed bacteriological index of 3 + and morphological index of 15. SSS from normal skin was negative. Histopathological examination of an eczematous plaque showed orthokeratosis, focal parakeratosis, spongiosis, and irregular acanthosis [Figure 2a]. Dermis showed mild edema and periappendageal and perineural lymphocytic infiltrate. Wade Fite staining was positive for acid-fast bacilli. Histopathological examination from a hypopigmented macule revealed atrophic epidermis with blunting of rete ridges, grenz zone, and dermal macrophage granulomas [Figure 2b]. Wade Fite staining was positive for acid-fast bacilli. There were no globi. A diagnosis of borderline lepromatous leprosy was made. He was treated with multibacillary multi drug therapy (MDT) for 1 year. The eczematous plaques subsided following application of mid-potency corticosteroids.

A 34-year-old male presented with exacerbation of oozing from a recurrent asymptomatic erythematous exudative lesion on the medial aspect of the left leg of 2 years duration [Figure 3]. There was no history of atopy, ichthyosis, or contact with allergens. On examination, he had an 18 × 15 cm hypopigmented macule with erythematous borders, within which there was a 6 × 4 cm well-defined erythematous plaque with oozing and crusting. Touch, pain, and temperature sensations were impaired on the lesion. Peripheral nerves were normal. Skin smears showed no bacilli. Histopathology of the lesion showed atrophic epidermis, clear subepidermal zone, and granulomas around nerves in the dermis consisting of epithelioid cells, a few Langhan's type giant cells and lymphocytes consistent with borderline tuberculoid leprosy. He was treated with 6 months of paucibacillary MDT following which the hypopigmented macule became less evident. His eczema continued to recur periodically even after completion of treatment and is

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Figure 1: Plaques of nummular eczema on the right upper limb of a patient with borderline lepromatous leprosy.

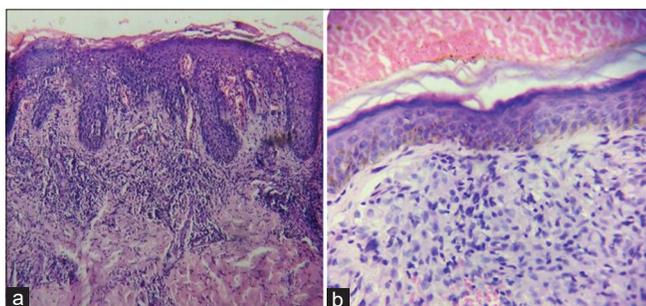


Figure 2: (a) Histopathological image of the eczematous plaque of the first patient showing orthokeratosis, irregular acanthosis, and spongiosis (H and E, 100×) (b) Histopathological image of the hypopigmented macule of the same patient showing thinned out epidermis with blunting of rete ridges, Grenz zone, and a macrophage granuloma in the dermis (H and E, 1000×).



Figure 3: Eczematous plaque on a hypopigmented macule with erythematous border on the leg of the second patient with borderline tuberculoid leprosy.

being managed with topical mid-potency corticosteroids and emollients.

Recurrent pruritic oozing coin-shaped plaques on the extremities are often the most common presentation of nummular eczema and are usually associated with atopy.^[2] Both our patients had no personal or family history suggestive of atopy.

There is a view that nummular dermatitis is a reaction pattern to an underlying infection.^[3] In the first case of

borderline lepromatous leprosy with little dryness of lesions, this seems possible. We had also considered if the eczematous lesions were due to type 1 lepra reaction, but there were no clinical features such as pain or tenderness of lesions or histopathological features such as marked edema or increased number of inflammatory cells such as lymphocytes or macrophages. Another rare possibility considered was lazarine leprosy, which is a rare ulcerating type of type 1 lepra reaction.^[4] Our patient had severe exudation from the plaques, but there was no ulceration. Furthermore, the plaques healed without atrophy on treatment.

Nummular eczema can occur in association with ichthyosis. This can explain our second case of borderline tuberculoid leprosy, in which dry skin lesions could have led to the development of eczema. The irreversible atrophy of sweat glands and sebaceous glands due to the granulomas of leprosy may explain why the ichthyosis in those areas can persist and lead to recurrence of eczema. The non-pruritic nature of lesions in our cases is probably due to the neurological deficit associated with leprosy. These cases suggest that when one encounters non-pruritic eczema, it would be prudent to evaluate those patients thoroughly, for lesions suggestive of leprosy, especially in regions endemic for the disease. Our cases also highlight the importance of examination in good light, preferably adequate daylight, to pick up skin lesions.

Declaration of patient consent

Patient's consent not required as patients identity is not disclosed or compromised.

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Conflicts of interest

Dr. N. Asokan and Dr. Betsy Ambooken are on the editorial board of the journal.

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