

# Case Letter

## Psoriasis and Leprosy: A Mystifying Coexistence

To the Editor:

Psoriasis and leprosy exist mutually exclusively with only a few cases being reported regarding their coexistence. Among the various forms of psoriasis associated with leprosy, the coexistence of erythrodermic psoriasis with leprosy is rare. Herein we describe a case of erythrodermic psoriasis in a patient with leprosy.

A 35-year-old man presented to our outpatient department with asymptomatic, red, scaly lesions all over his body of 1 month's duration. He had been diagnosed with borderline tuberculoid leprosy with a leprosy type 1 reaction 4 months prior to presentation. An associated history of fever was present. In the last 4 months he was regularly being treated with a multibacillary dose of multidrug therapy along with systemic steroids, which were administered for the first 2 months. He did not have a history of diabetes mellitus, hypertension, or any other drug use.

On examination he was febrile (temperature, 38.92°C). Cutaneous examination revealed generalized erythema with thickly adherent, silvery white scales all over his body including the scalp. The Auspitz sign was positive. No mucosal, nail, or joint involvement was seen. A trophic, punched-out ulcer over the left olecranon process with sloughing at the base was present (Figure). He experienced a loss of sensation over the plantar and lateral aspect of his right foot. Nerve examination revealed thickening of both ulnar nerves, right anterior tibial nerves, right common peroneal nerve, and right posterior tibial nerve. Other systemic examination was unremarkable. Complete hemogram, liver function tests, renal function tests, chest radiograph, and ultrasonography of the abdomen revealed no abnormalities. A skin biopsy from the scaly areas revealed features of psoriasis. He was started on oral methotrexate 7.5 mg once weekly with folic acid on the remaining days. He also was given oral antibiotics and topical corticosteroids. Antileprosy treatment was continued. Weekly follow-up visits showed good improvement after 3 weeks.

Since biblical times, the term *lepra* included both leprosy and psoriasis. It was in the 19th century that these 2 diseases were considered distinct entities.<sup>1</sup> Toward the end of the 20th century, a report of a large number of leprosy patients followed for approximately 40 years indicated no clinical evidence of psoriasis.<sup>2</sup> The increasing knowledge of the etiopathogenesis of both of these diseases has made their coexistence an interesting rarity, as only a few cases have been reported.<sup>3-5</sup> In a survey conducted by Kumar et al<sup>6</sup> of 145,661 cases of leprosy, only 20 individuals had psoriasis. Among the various forms of psoriasis associated with leprosy, coexistence of erythrodermic psoriasis with leprosy is rare.



Scaling over the body with a punched-out lesion over the left olecranon process.

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The authors report no conflict of interest.

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The exact cause of this association is unknown. The factors implicated in the rarity of this coexistence are genetics, immunology, and the role of neuropeptides and apoptosis. One hypothesis suggests that the prevalence of psoriasis in different populations mainly results from differences in natural selection for gene polymorphisms associated with more vigorous immunity against infectious agents. Therefore, resisting infection by lepra bacilli may have been the evolutionary advantage that favored the expansion of some psoriasis-associated genotypes.<sup>7</sup>

Genetic factors play a role in the pathogenesis of psoriasis and leprosy. Population studies have noted the association of HLA-DR2 and HLA-DQW1 with leprosy but HLA-B13 and HLA-B17 with psoriasis. One of the important susceptibility factors for psoriasis is the presence of HLA-Cw\*0602.<sup>3,8</sup> Psoriasis is associated with activated T cells in a predominantly T<sub>H</sub>1 (helper T cell) pattern, whereas in leprosy, suppressor T cells (CD8<sup>+</sup>) are generated with a lack of IL-2 production by the host because of immunosuppression.<sup>9</sup>

The cutaneous nerves and neuropeptides released play a role in the pathogenesis of psoriasis. The psoriatic plaque may remit denervation following surgery or application of capsaicin. The local depletion of neuropeptides such as substance P, vasoactive intestinal peptide, protein gene product 9.5, and calcitonin gene-related peptide may remit psoriasis plaques.<sup>9</sup> Hence, the neuropathy caused by *Mycobacterium leprae* infection may result in structural and functional alterations in the cutaneous sensory nerves so that the process of neurogenic inflammation, which seems to be an integral part of the psoriatic disease process, is inhibited.<sup>3</sup>

It has been found that a markedly thickened epidermis in psoriatic lesions is likely the result of both hyperproliferation and decreased cell death (apoptosis),<sup>10</sup> rather than an increase in the level of spontaneous apoptosis seen in leprosy patients,<sup>11</sup> which also may contribute to the rare coexistence of psoriasis and leprosy.

Over the decades different forms of psoriasis have been described in association with leprosy.<sup>3-5</sup> Erythrodermic psoriasis in our patient could have been caused by the discontinuation of steroids or an

overall improvement in the immune response following initiation of multidrug therapy. Our case report documents a rare presentation of erythrodermic psoriasis in an enigmatic relationship with leprosy.

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