# Leprosy in a Psoriasis Patient – Rare Co-existence

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Received: 02.06.2024

Revised: 07.07.2024

Accepted: 17.07.2024

Leprosy and psoriasis are two unique diseases of mankind used interchangeably till modern times. As our understanding of the pathogenesis of both diseases increased, rarer it became its coexistence. This rarity is because of a complex interaction between genetic factors, immunity, cytokines, neuropeptides, and keratinocytes. We report a rare case of psoriasis of 16 years duration, who developed numbness over both legs and hands of 8 months duration, diagnosed as a case of leprosy based on investigation. We report a rare coexistence of pure neuritic leprosy and psoriasis.

Keywords: Psoriasis, Leprosy, Hansen's disease, Co-existence

### Introduction

Leprosy and psoriasis were known to mankind from ancient times and used interchangeably till the 19th century. After identification as a separate entity, the coexistence of both leprosy and psoriasis was found to be very rare (Wahba et al 1980, Kumar et al 1992). We report a case of rare coexistence of leprosy in a patient with psoriasis.

### **Case report**

A 45 years-old-male, known case of psoriasis of 16 years duration on irregular treatment, presented with numbness over both legs and hands for 8 months. One episode of sharp shooting pain over the medial aspect of both elbows to hand was present with slippage of slippers in both legs. On examination, multiple well-defined polysized erythematous plagues with silvery white scales over the trunk and bilateral extremities were present (Fig. 1). Grattage and Auspitz signs were positive. Multiple hypopigmented macules with normal sensation were present at the previous psoriasis sites. Calculated PASI was 11.2. Nails showed pitting, ridging, and focal onycholysis. Patchy hypoesthesia was present over both the medial aspect of the hands and the lower half of the legs. The right ulnar nerve, common peroneal nerve, and left sural nerve were grade II thickened. Card test was positive in the 3rd and 4th web spaces of both hands. The slit skin smear was negative. Biopsies from the hypopigmented lesion and hypoaesthetic areas showed normal histopathology. Biopsy from the plaque showed features of psoriasis (Fig. 2). Left sural nerve biopsy sent for histopathology

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Fig. 1 : Erythematous scaly plaque with hypopigmented areas in the trunk.



Fig. 2 : Histopathology showing parakeratosis, hyperkeratosis, elongated rete ridges and collection of neutrophils in the spinosum layer suggestive of psoriasis. (20x)

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| Essential criteria  | Auxiliary criteria  |
| <ul> <li>Epidemiological features</li> <li>Residence in an endemic area or a history of contact with cases of leprosy.</li> </ul>   | <ul> <li>Nerve biopsy/fine needle aspiration cytology<br/>when done shows definitive (acid-fast bacilli or<br/>caseous necrosis) or suggestive (perineurial or<br/>endoneurial infiltrate and perineural fibrosis)<br/>features of leprosy neuritis.</li> </ul> |
| <ul> <li>Clinical features</li> <li>Thickened peripheral nerve(s) with definitive sensory impairment, with or without motor impairment or loss of function.</li> <li>Absence of any skin patch.</li> </ul>                        | <ul> <li>Nerve conduction studies showing a decline in<br/>amplitude and nerve conduction velocities or<br/>an increase in latency.</li> </ul>  |
| <ul> <li>Laboratory features</li> <li>Slit skin smear from three different sites including the anesthetic area should be negative and</li> <li>No definitive histological features of leprosy in lesional skin biopsy.</li> </ul> |   |
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Table 1 : Diagnostic criteria for pure neuritic leprosy (PNL).

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showed perineural inflammatory infiltrate with negative AFB and *M leprae* PCR RLEP gene amplification was positive. A nerve conduction study showed involvement of both sensory and motor component of lower limbs in the form of reduced velocity and increased latency. He was diagnosed as pure neuritic leprosy based on the essential and auxiliary criteria listed in Table 1 (Narang et al 2016). He was started on WHO based multi-drug therapy for 1 year along with injection secukinumab for psoriasis with which he had significant improvement.

## Discussion

The word 'Lepra' originates from old Greek meaning scaly disease. In the late 18th century, the word 'Lepra' is a generic name for psoriasis among English physicians (Jopling 1990). Though Daniel Turner in 1726 differentiated both as separate entities and mentioned psoriasis as the 'Leprosy of Greeks' and Hansen's disease as 'Leprosy of Arabians', it was used interchangeably till modern times (Lyell 1987).

The rarity of psoriasis among leprosy patients was reported early by Wahba et al. in 1980 by following 309 leprosy patients for 40 years (Wahba et al 1980). Kumar et al. found only 20 cases of psoriasis among 1,45,661 leprosy cases, corresponding to a prevalence of 0.014% which implies co-existence of these two diseases, whereas the prevalence of psoriasis among the world population is 2-3% (Kumar et al 1992). The exact nature of rarity is unknown, and a few proposed hypotheses are discussed below.

Tuberculoid leprosy is characterized by Th1 immune response with compact granuloma formation and lepromatous leprosy is characterised by Th2 immune response. Psoriasis is characterized by strong Th1 immune polarization (Sheikh & Hill 2020). Also, there is hyperreactivity of the reticuloendothelial system and neutrophils have enhanced chemotactic, and phagocytic activities in psoriatic patients in comparison with the normal population (Sugathan & Riyaz 1990). These factors keep the *M leprae* under control and inhibit clinical progression of leprosy.

Genetic factors play a major role in both diseases. HLA- Cw\*06, HLA-B13 and HLA B17 are associated with psoriasis whereas HLA-DR2 and HLA-DQW1 are associated with leprosy. HLA DR B1\*04 provides increased susceptibility to psoriasis while protects against leprosy. HLA-Cw\*06 also has a significant negative association with leprosy in the Indian population (Bassukas et al 2012).

The T cells also play a major role in the pathogenesis. In psoriasis, there is an increase in T helper cells (Th1) and IL -2 production with diminished regulatory T cells (Treg), whereas in all types of leprosy, there is a depressed T-cell functional state and reduced IL-2 production. Therefore, psoriatic patients might have a natural protection against developing leprosy. HLA antigen associated with a T-cell defect may be common to psoriasis and leprosy and only such patients may develop both diseases. (Sugathan & Riyaz 1990, Bassukas et al 2012)

Neuropeptides like substance P, calcitonin, and neuropeptide Y released by cutaneous nerves have been postulated to play a role in the pathogenesis of psoriasis. Destruction of cutaneous nerve fibers in leprosy results in depletion of neuropeptides and inhibits the psoriasis causing clearance of plagues. Toll-like receptors (TLR1&2), IL12, ERBB2-ERK1/2 pathway, and amphiregulin have a role in the pathogenesis of both diseases and need further evaluation (Bassukas et al 2012). Also, heterozygotes to some autosomal heritable diseases may have the selective advantage of increased resistance against common mycobacterial infections and expansion of psoriasis resulting in containment of leprosy epidemics. Psoriatic lesions show hyperproliferation and decreased

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apoptosis of keratinocytes, whereas leprosy cases have increased spontaneous apoptosis of keratinocytes, which also may contribute to the rare coexistence (Sheikh & Hill 2020).

Case reports of leprosy in psoriatic patients on TNF  $\alpha$  blockers were reported (Lydakis et al 2012), however, our patient was not on any biologicals. No pure neuritic leprosy case in psoriasis has so far been reported and we report one such case of rare coexistence.

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**How to cite this article :** Mannu A, Vasudevan B, Lekshmipriya K et al (2024). Leprosy in a Psoriasis Patient – Rare Co-existence. *Indian J Lepr.* **96**: 349-352.

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