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Case Report

Bilateral Facial Nerve Palsy in Leprosy without Lepra Reaction: An Uncommon Presentation

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Bilateral facial nerve palsy, a rare condition accounting for 0.3–2% of all facial palsy cases, is often associated with several systemic diseases. Leprosy, caused by *Mycobacterium leprae*, commonly involves the facial nerve; however, bilateral cases are exceedingly rare. We report a case of a 52-year-old male with subpolar lepromatous leprosy presenting with progressive, complete bilateral facial nerve palsy, notably without any lepra reaction. The patient showed hypopigmented skin patches, lagophthalmos, glove-and-stocking anaesthesia, and peripheral neuropathy, with slit skin smears and biopsies confirming leprosy. Unlike the rapid onset and partial palsy often seen in lepra reactions, this gradual, complete palsy suggests chronic nerve infiltration.

Keywords : Bilateral Facial Nerve Palsy, Leprosy, Tarsorrhaphy, Lepra Reaction

Introduction

Bilateral facial nerve palsy is a rare but significant form of facial nerve palsy, accounting for 0.3 to 2% of all cases (Teller & Murphy 1992). While unilateral facial palsy is relatively common in conditions such as Bell's palsy or trauma, the simultaneous involvement of both facial nerves is uncommon and often associated with systemic diseases.

Leprosy, also known as Hansen's disease, is a chronic infectious disease caused by *Mycobacterium leprae*, which primarily affects the skin, peripheral nerves, upper respiratory tract, and eyes. Although facial nerve, its zygomatic branch in particular, is the most common cranial nerve involved in leprosy, most cases are subclinical and occur most commonly in the borderline spectrum (Philip et al 2022). Cranial nerve involvement in leprosy typically occurs within average disease duration of less than five years (Gopinath et al 2004). This report presents a case of bilateral facial nerve palsy in leprosy, distinguished by its slow progression and absence of reactional symptoms, and highlights how delayed presentations may still occur due to delay in early recognition and referral.

Case Report

A 52-year-old male presented with complaints of inability to close his eyelids, which had been progressively worsening over the past one year, accompanied by excessive watering from both eyes and drooling of saliva. He initially noticed

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weakness and difficulty in closing his eyes, which gradually progressed to a complete loss of blinking. He also reported a painless ulcer over his left foot, present for the past nine months. On further questioning, he gave a history of reduced sensation in both hands and legs, which had preceded the development of the ulcer. The patient mentioned seeking treatment from multiple practitioners for these symptoms, during which he received multiple medications including antibiotics along with supportive care. However, detailed treatment records were not available, and the patient did not recall any specific diagnosis being communicated to him. Multiple hypopigmented patches were noted over the arms, neck, trunk, and thighs. These were asymptomatic and detected incidentally. Upon enquiry, the patient recalled having first noticed them approximately two years ago. There was no history suggestive of a lepra reaction.

On examination, multiple (~ > 15) bilaterally asymmetrical discrete to coalescent hypo-pigmented macules with well-defined margins and of varying sizes noted over bilateral arms, neck, anterior aspect of chest, buttocks and thigh (Fig. 1). The lesions over the thigh appeared shiny and infiltrated. A trophic ulcer was noted over the lateral aspect of sole of left foot (Fig. 2). Temperature and touch sensations over the hypopigmented lesions were intact. Bilaterally symmetrical glove-and-stocking anaesthesia - including loss of temperature, pain, fine touch sensation involving the hands and legs below knees - was present. Bilateral foot

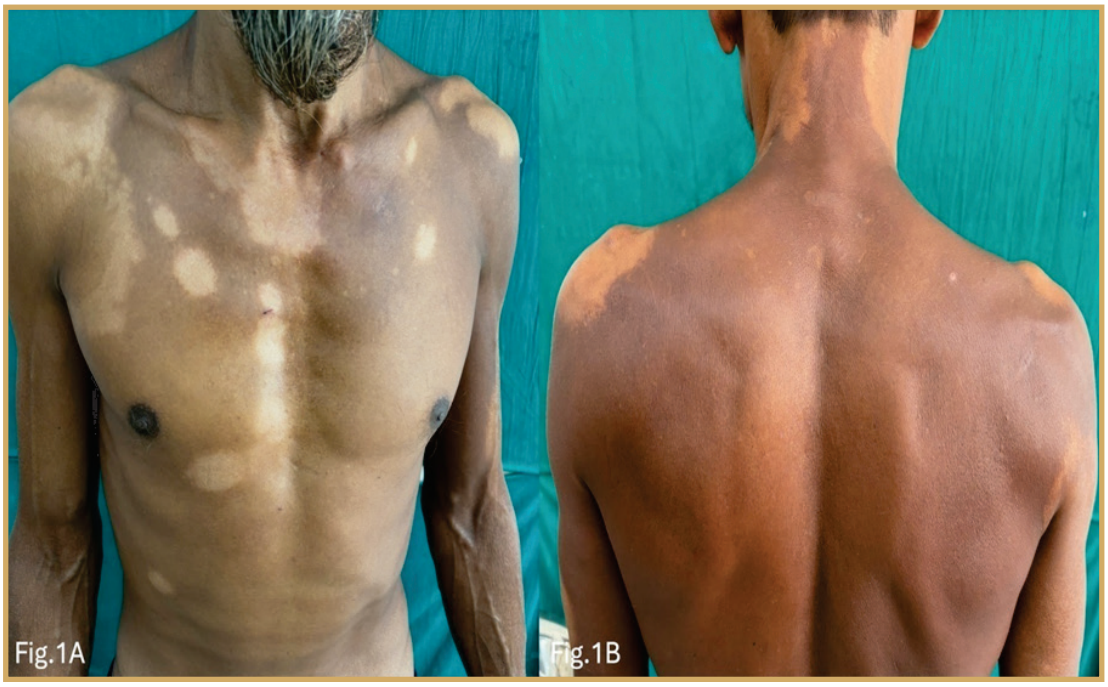

Fig. 1 : Multiple discrete to coalescent hypopigmented macules with well-defined margins and of varying sizes noted over bilateral arms, neck, anterior aspect of chest and upper back. Few satellite lesions were noted over the periphery of the larger lesions.

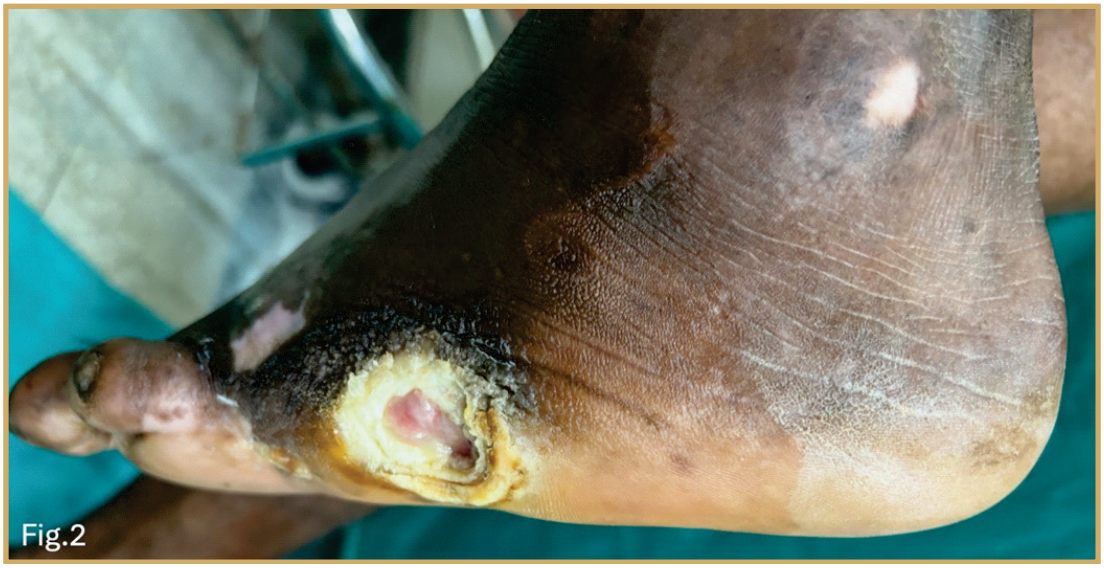

Fig. 2 : A trophic ulcer of size 4 cm □ 4 cm noted over the lateral aspect of left foot with hyperkeratotic margins, sloping edges and floor covered with minimal slough.

Fig. 3A : Examination of the face showing bilateral lagophthalmos with palpebral aperture of ~ 2 cm, lower lid ectropion, hollowing of cheeks, and deformity of nasal columella and left ala.
Fig. 3B : Demonstration of Bell's phenomenon.

drop along with deformity of the toes was also noted. On examination of the face, no madarosis / facial lesions were observed; however, there was a nasal deformity with destruction of the left ala and atrophy of the nasal septum. Nerve examination revealed bilateral symmetrical cord-like, non-tender thickening of the zygomatic branches of the facial nerve, as well as the supraclavicular, greater auricular, ulnar, radial cutaneous, common peroneal and posterior tibial nerve. Ophthalmological examination showed bilateral lagophthalmos with palpebral aperture of ~ 2 cm, lower lid ectropion, epiphora and left corneal opacity (Fig. 3A). Facial nerve examination revealed complete bilateral facial nerve palsy (House-Brackmann grade V on both sides; House & Brackmann 1985), lagophthalmos with Bell's phenomenon (Fig. 3B), loss of facial expression, absence of forehead wrinkling, drooping of bilateral angle of mouth, hollowing of cheeks, and weakness on attempted eyebrow elevation, teeth baring, and cheek puffing.

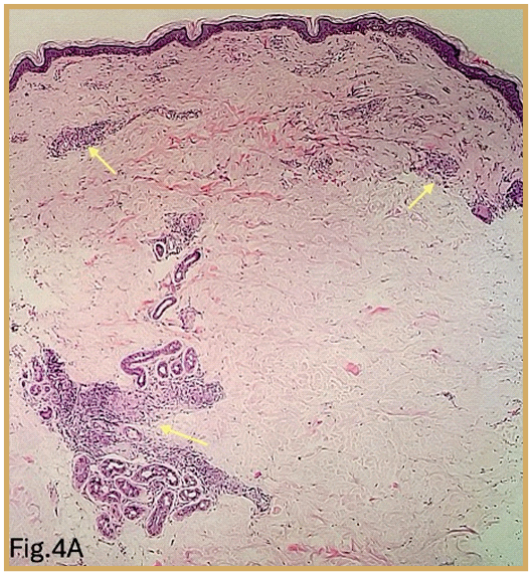

Fig. 4A : Histopathology image showing peri-adnexal and peri-vascular inflammatory infiltrate highlighted by yellow arrows. (H&E stain, 10x magnification).

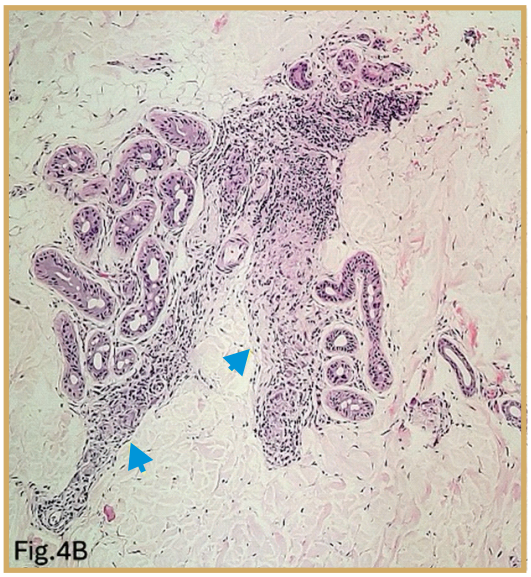

Fig. 4B : Histopathology image showing peri-eccrine epithelioid granulomas with histiocytes, lymphocytes, plasma cells and few Langhans type giant cells highlighted arrows. (H&E stain, 40x magnification).

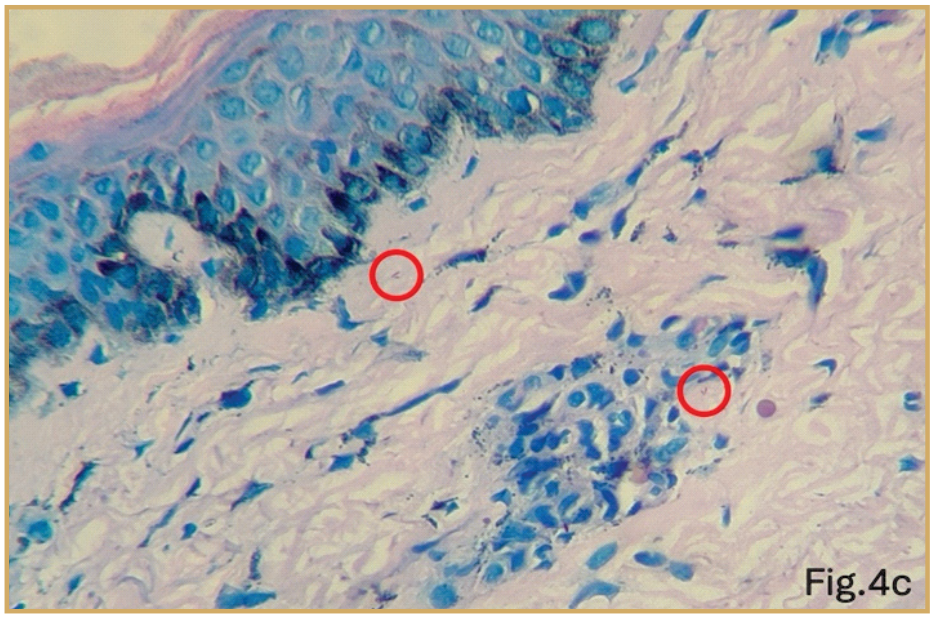

Fig. 4C : Histopathology image showing few scattered acid- fast- bacilli in Fite-Faraco staining highlighted by red circle. (Fite-Faraco stain, 100x magnification).

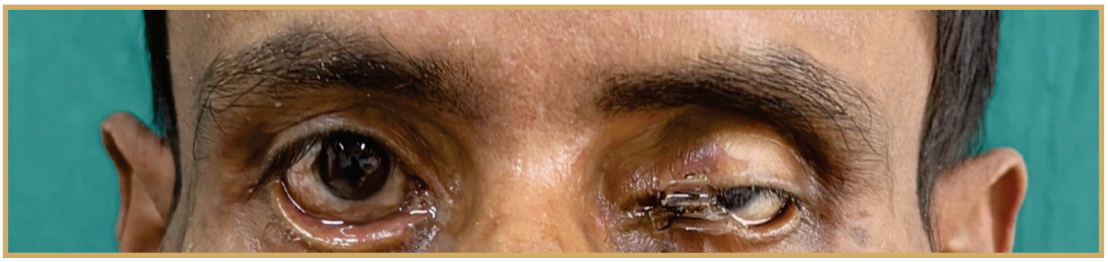

Fig. 5 : Post-operative image showing tarsorrhaphy done over the left eyelids.

Table 1 : Comparison of findings noted in previous reports of bilateral facial nerve palsy in Hansen's disease with the current case report.

Author	Type of palsy	Immunological pole	Concomitant lepra reaction	Presence of facial lesion
Inamadar et al. (2 cases)	Incomplete	BT	+	+
	Incomplete	BT	+	+
Singal et al.	Incomplete	LL	–	–
Jaiswal et al. (3 cases)	Incomplete	BT	+	+
	Incomplete	BT	+	+
	Incomplete	Not mentioned	–	–
Lalla et al.	Incomplete	Not mentioned	+	+
Current case	Complete	LL (subpolar)	–	–

Abbreviations: BT – Borderline tuberculoid leprosy, LL – Lepromatous leprosy

Olfactory and facial sensations were intact, and examination of the remaining cranial nerves revealed no abnormalities.

Slit skin smear was positive from both lesional and non-lesional (ear lobe) skin with a bacillary index of +4. Skin biopsy from lesional skin was consistent with the findings of borderline tuberculoid leprosy (Fig. 4). Nerve conduction studies demonstrated reduced conduction velocities in the facial, common peroneal and median nerves confirming peripheral neuropathy. A neurologist consultation was sought to evaluate alternative etiologies for the bilateral facial palsy. Routine blood tests revealed normal fasting glucose and HbA1c levels, HIV and syphilis serology were negative, and a chest X-ray was unremarkable for sarcoidosis. Although a brain imaging was advised to exclude central or compressive causes, it was not performed due to financial constraints.

A diagnosis of subpolar lepromatous leprosy not in reaction and grade II disability with bilateral lower motor neuron facial nerve palsy was made based on clinical findings, skin biopsy results, and nerve conduction studies.

The patient was started on multibacillary-multidrug therapy (MB-MDT) for leprosy, consisting of rifampicin, clofazimine and

dapsone, in accordance with WHO guidelines. Ophthalmology opinion was sought and eye care, including the use of artificial tears and eye patches at night was advised. The patient underwent tarsorrhaphy of left eye (Fig. 5) and was planned for the same in the alternate eye at a later period. Unfortunately, the patient did not return for further evaluation and was lost to follow-up.

Discussion

Bilateral facial nerve palsy is an extremely rare manifestation of leprosy, and while the same has been described in longstanding, untreated cases in the past, it remains an exceedingly rare presentation in the current post-integration era, especially in the absence of lepra reaction. Other causes of bilateral facial nerve palsy include Lyme disease, Bell's palsy, Guillain-Barré syndrome, diabetes, sarcoidosis, Parkinson's disease, and multiple sclerosis; leprosy being a rare cause (Keane 1994). The notable features in our case are complete bilateral lower motor neuron facial palsy in a non-reactive state and the delay in the diagnosis despite clear clinical indicators and available healthcare resources.

In the borderline spectrum, facial nerve involvement is usually unilateral and occurs as a part of leprosy reaction (Dastur et al 1966). In lepromatous leprosy, facial nerve palsy develops not due to leprosy reaction but from symmetrical, widespread infiltration and subsequent atrophy, affecting both the branches of the facial nerve and the muscles themselves (Slem 1971).

Most cases of bilateral facial nerve palsy in leprosy show rapid onset with partial facial palsy (sparing one or more muscles) and are often associated with an acute lepra reaction. In contrast, our patient experienced gradual, complete bilateral palsy without any lepra reaction, suggesting chronic nerve infiltration causing silent neuropathy rather than an acute inflammatory process. A summary of findings from other studies compared to our case is provided in Table 1 (Inamadar & Palit 2003, Singal et al 2006, Jaiswal & Subbarao 2010, Lalla et al 2015).

Previous studies have proposed that, unlike other causes of bilateral lower motor neuron facial paralysis, leprosy tends to affect individual terminal branches of the facial nerve. This often results in partial symptoms, such as bilateral lagophthalmos, while other features of facial nerve palsy are usually absent; however, this pattern was not observed in our case.

The discordance between the clinical diagnosis (subpolar lepromatous leprosy) and histopathology (borderline tuberculoid) reflects the spectral nature of leprosy, as histopathological findings vary with lesion stage and site, such mismatches are not uncommon and must be interpreted in the broader clinical context, especially in atypical presentations. Prior studies have reported a 60%–63% clinicopathological correlation, with higher concordance seen in lepromatous cases (Moorthy et al 2001, Giridhar et al 2012, Arunagirinathan et al 2017).

Our case emphasizes the importance of recognizing chronic, non-reactive leprous neuropathy as a potential cause of facial nerve palsy which is rarely emphasized in the literature, and which may require different therapeutic strategies and has a distinct prognosis compared to reactional neuropathy, which responds well to immunosuppressive agents. At present, no novel medical therapies are available for the treatment of chronic facial nerve palsy in leprosy. Management focuses on multidrug therapy to halt disease progression, supportive care (including ocular protection), physiotherapy, and surgical correction when required.

Despite significant advancements in community awareness, the availability of effective multidrug therapy, and improvements in healthcare infrastructure, it is concerning that this patient remained undiagnosed for several years. Advanced disease presentations, as demonstrated in this case, continue to occur primarily due to delayed diagnosis resulting from a low index of clinical suspicion at the primary care level, the first point of patient contact. This underscores persistent systemic gaps, including limited access to specialist consultations and under-utilization of established referral pathways. To address these challenges, regular training of general healthcare providers, reinforcement of referral systems under public health programs, and enhanced coordination between field healthcare workers and program managers are imperative for the effective control of leprosy in the post-integration era.

In summary, this case of bilateral facial nerve palsy due to leprosy differs from other reported cases in terms of its gradual onset, the absence of a lepra reaction, the complete bilateral paralysis. Clinicians should be aware of these variations in presentation and tailor treatment strategies accordingly, with an emphasis on early diagnosis and intervention to prevent and treat permanent nerve damage.

References

1. Arunagirinathan M, Muniswamy V, Jeevirathinam S (2017). Clinical and histopathological correlation in Hansen's disease. *Ann Pathol Lab Med*. **4(4)**: A454-459.
2. Dastur DK, Antia NH, Divekar SC (1966). The facial nerve in leprosy 2. Pathology, pathogenesis, electromyography and clinical correlations. *Int J Lepr Other Mycobact Dis.* **34**: 118-138.
3. Giridhar M, Arora G, Lajpal K et al (2012). Clinicohistopathological concordance in leprosy - a clinical, histopathological and bacteriological study of 100 cases. *Indian J Lepr.* **84(3):** 217-225.
4. Gopinath DV, Thappa DM, Jaishankar TJ (2004). A clinical study of the involvement of cranial nerves in leprosy. *Indian J Lepr.* **76(1)**: 1-9.
5. House JW, Brackmann DE (1985). Facial nerve grading system. *Otolaryngol Head Neck Surg*. 93(2): 146-147.
6. Inamadar AC, Palit A (2003). Bilateral facial palsy in Hansen's disease. *Lepr Rev.* **74(4)**: 383-385.
7. Jaiswal AK, Subbarao NT (2010). Bilateral lagophthalmos in leprosy: Is it a rare phenomenon? *Indian J Lepr.* **82(4)**: 201-203.
8. Keane JR (1994). Bilateral seventh nerve palsy: analysis of 43 cases and review of the literature. *Neurology*. **44(7)**: 1198-1202.
9. Lalla R, Mulherkar RV, Misar PV (2015). Incomplete peripheral facial nerve palsy and ulnar neuropathy due to leprosy mistaken as faciobrachial stroke. *BMJ Case Rep*. **2015**: bcr2015210060
10. Moorthy BN, Kumar P, Chatura KR et al (2001). Histopathological correlation of skin biopsies in leprosy. *Indian J Dermatol Venereol Leprol.* **67(6)**: 299-301.
11. Philip AS, George A, Mathew V et al (2022). Facial nerve and branch dysfunction in leprosy - a study from India. *Lepr Rev.* **93(4)**: 377-391.
12. Singal A, Vij A, Pandhi D (2006). Lepromatous leprosy with bilateral facial nerve palsy and hyperthyroidism. *Indian J Lepr*. **78(3)**:291-296. Slem G (1971). Clinical studies of ocular leprosy. *Am J Ophthalmol.* **71(1 Pt2)**: 431-434.
13. Teller DC, Murphy TP (1992). Bilateral facial paralysis: a case presentation and literature review. *J Otolaryngol*. **21(1)**: 44-47.

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