

Hansen's Disease Borderline Tuberculoid Presenting with Bilateral Facial Nerve Palsy, Trigeminal and Ulnar Nerve Palsy

Cinderella X¹, S Pradeep Nair², Shima KT³, Akhilendu SN⁴, Kabilan T⁵

Received: 07.02.2026

Revised: 22.04.2026

Accepted: 04.05.2026

Bilateral facial nerve palsy is an uncommon neurological presentation, accounting for less than 2% of all facial palsy cases and very rare in borderline tuberculoid (BT) leprosy. We report a 63-year-old hypertensive female referred from the Neurology department for assessment of skin lesions with suspected brachial-facial palsy. CT-Scan and MRI imaging procedures did not reveal any evidence of cerebrovascular accident or any other CNS abnormality. On examination the patient had multiple discrete and confluent hypopigmented annular patches with loss of all modalities of sensation distributed on the trunk, abdomen and thighs. Nerve examination demonstrated bilateral lower motor neuron (LMN) facial nerve palsy, right trigeminal nerve palsy and right ulnar nerve palsy. Ear lobe and slit skin smear were negative for AFB. Skin biopsy showed multiple round epithelioid cells and Langhan giant cell granulomas diagnostic of borderline tuberculoid Hansen's disease (HD- BT). The patient was started on multibacillary multidrug therapy (MB-MDT) and systemic prednisolone, and there was prompt response. HD can mimic many neurological diseases, and thus early recognition is essential to prevent grade 2 disability.

Keywords: Bilateral Facial Nerve Palsy, Hansen's Disease, Neurological Disorders

Introduction

Hansen's disease (HD) is a chronic granulomatous disease caused by *Mycobacterium leprae* primarily involving the peripheral nervous system and skin. It does not usually involve the brain and spinal cord due to the predilection of the bacilli for the cooler regions of the body. However, HD can mimic many neurological disorders and is often misdiagnosed. This is more often for the pure neuritic leprosy as there are no skin lesions. Any patient presenting with multiple palsies,

HD should always be in the differentials. Palsies in leprosy usually follow lepra reactions, the commonest being ulnar palsy (Nair & Mathew 2017). Here we are reporting a case of HD with multiple nerve palsies diagnosed as brachial facial stroke by the neurologist in the absence of lepra reaction.

Case Report

A 63-year-old hypertensive female was evaluated in the neurology ward for sudden onset of headache and facial asymmetry and weakness

¹ Cinderella Xson, MBBS, Resident

² S Pradeep Nair, MD, Professor

³ Shima K T, MBBS, Resident

⁴ Akhilendu S N, MBBS, Resident

⁵ Kabilan T, MD, Resident

Department of Dermatology and Venereology, Government TD Medical College, Alappuzha-688005, Kerala, India.

Corresponding Author: Dr. S Pradeep Nair, Email: dvmchtm@yahoo.co.in



Fig. 1 (a, b, c): a. Annular anaesthetic plaques on breast, b. Hypopigmented patches on posterior trunk with erythematous indurated borders, c. Annular plaques on right thigh with erythematous indurated borders.

of right hand. She was provincially diagnosed as brachial facial stroke and investigated. Since the patient also had concomitant skin lesions she was referred to the dermatologist. Upon history taking the patient revealed that she had hypopigmented skin lesions on the trunk and extremities of 6 months duration. The lesions first started on

the posterior trunk, then later involved the breast and thighs. The lesions were asymptomatic. She was treated from a local hospital with emollients. Three weeks ago she first developed headache and then facial asymmetry and inability to close eyes. One week later she also noticed weakness of right arm. She also had difficulty in squeezing

clothes after washing, difficulty in holding objects and slipping of chappals while walking.

On dermatological examination the patient had multiple (15-20 lesions) discrete and confluent

partly well-defined and partly ill-defined hypopigmented patches and annular plaques (Figs.1a, b, c), distributed on the anterior and posterior trunk, breast and thighs. Some of



2a

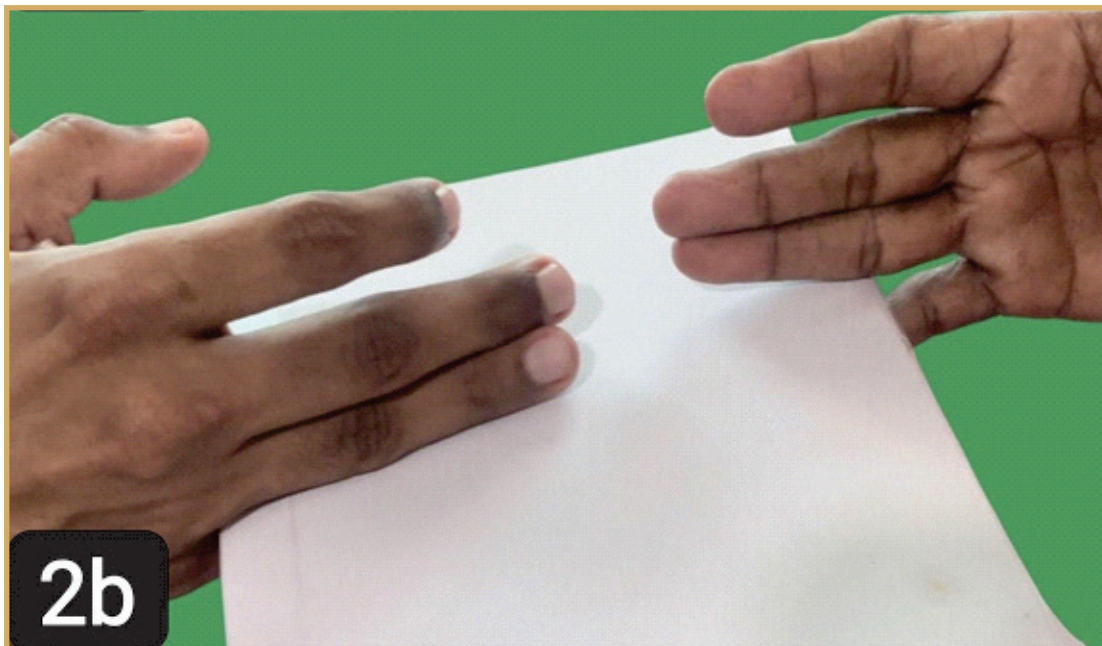


Fig. 2 (a, b): a. Bilateral Bell's phenomenon, loss of forehead wrinkles and deviation of right angle of mouth indicating bilateral facial palsy, b. Positive card test of right hand indicating ulnar palsy.

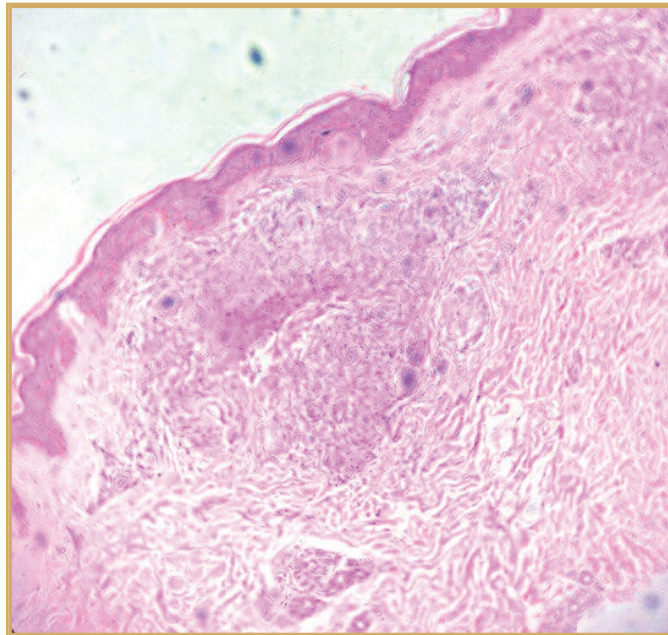


Fig. 3: Skin biopsy in low power showing multiple confluent round granulomas in upper dermis, H&E x100.

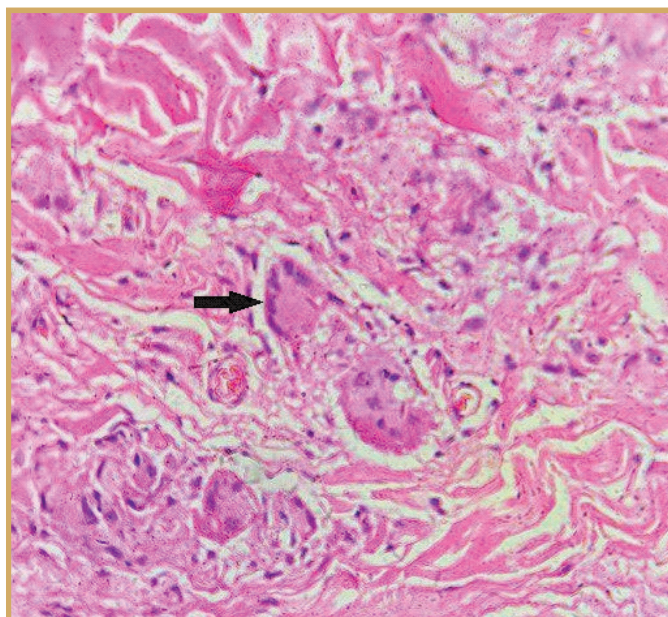


Fig. 4: Skin biopsy findings in high power showing granulomas with epithelioid cells and Langhans giant cell (arrow), H&E x400.

the annular plaques on the thighs and trunk showed erythematous infiltrated borders. Sensory modalities to temperature, touch and pain were lost on the patches on the breast and posterior trunk. A callosity was present over the plantar aspect of the left great toe. On cranial nerve examination there was bilateral Bell's phenomenon indicating paralysis of lower eyelid muscles due to facial nerve involvement, bilateral absence of forehead wrinkles, and bilateral lagophthalmos, indicating bilateral facial nerve involvement (Fig. 2a). Corneal sensation was absent on right side, indicating ophthalmic branch of trigeminal nerve involvement. The blink-reflex was absent on the right- side indicating both trigeminal (sensory) and facial (motor) nerve involvement. The patient also had deviation of the right side of jaw with absent jaw jerk, indicating trigeminal nerve involvement (Fig. 2a). Peripheral nerve examination showed right ulnar nerve thickening (Grade 1) with positive Wartenberg's sign, positive card test and Froment's sign all suggestive of right ulnar palsy (Fig. 2b) and left posterior tibial nerve thickening (Grade 1). The peripheral nerves were non tender and there were no signs of type 1 reaction.

Ear lobe and slit skin smear from the hypopigmented patches were negative for acid fast bacilli (AFB). A CT-Scan and MRI scan did not

show any evidence of stroke. Blood routine, liver and renal function tests and chest X-ray were all normal. Nerve conduction study showed low compound muscle action potentials suggestive of motor predominant neuropathy. A skin biopsy in low power showed epidermal atrophy with multiple round and confluent granulomas in the upper dermis (Fig. 3), while high power showed the granulomas to be epithelioid cells and Langhan giant cells (Fig.4). Thus, we made a final diagnosis of Hansen's disease borderline tuberculoid with bilateral facial palsy, right trigeminal palsy and right ulnar palsy with EHF (Eye, hand, feet) score of 6. The patient was started on multibacillary multidrug therapy (MB -MDT) and 60 mg prednisolone which was tapered gradually within a period of 6 months. The patient was put on prednisolone as the palsies were of 3 weeks duration and we attributed it as a consequence of silent neuritis. At the end of one month the patient showed excellent recovery of facial palsy (Fig.5). The trigeminal palsy showed moderate response, while the ulnar palsy showed tardy response. The patient was also sent to the physical medicine department for further management of trigeminal and ulnar palsy. The Ophthalmology Department advised protective eye shields during sleep and methycellulose lubricant eye drops. The patient was followed up



Fig. 5: Prompt recovery of facial palsy and lagophthalmos with prednisolone.

to 6 months at the end of which, the steroids were completely tapered, with excellent improvement of trigeminal palsy and moderate improvement of ulnar palsy.

Discussion

The 7th cranial nerve – the facial nerve is a “silent victim” of HD due to the propensity of *Mycobacterium leprae* to prefer the peripheral nerves due to its cooler temperature. The prevalence of facial palsy in leprosy ranges from 14.96% (Nair & Mathew 2017) to 20.7% (Thompson et al 2006). Our patient had four nerve palsies: bilateral facial palsy, trigeminal palsy and ulnar palsy. Although multiple nerve involvement is more commonly described in lepromatous disease, it can occasionally occur in borderline tuberculoid cases. In spite of multiple nerve palsies our patient did not show any signs and symptoms of Type 1 lepra reaction. Hence this could qualify for the “silent neuritis”. A classical neuritis presents with acute onset with pain and tenderness along the course of the nerves and sudden neurological deficits like facial, ulnar palsy or foot drop, while silent neuritis is insidious in onset and presents with sensi-motor deficits but without the classical presentations of pain, and tenderness along the course of the nerves (Boku et al 2010). Silent neuritis is unique to leprosy and has also been described in leprosy in pregnancy. Silent neuritis, due to it being asymptomatic is ominous for the patient as the patient may not seek medical advice until too late when irreversible grade 2 disability occurs, in contrast to acute neuritis in a type 1 lepra reaction where the patient seeks immediate medical advice due to the severe pain and tenderness of the nerves. Unilateral facial nerve palsy is relatively common in HD, especially following type 1 lepra reactions, while bilateral facial nerve palsy is very rare and even rarer in the absence of reactions, as in the present

case. However, bilateral facial nerve palsy in the absence of reactions has been reported previously (Khan et al 2025). Facial palsy as a presenting sign of leprosy has also been reported (Inamadar & Palit 2003). Our patient in addition to bilateral facial palsy also had right trigeminal palsy in the form of lagophthalmos and corneal anesthesia which significantly contributes to morbidity as the exposed eyeballs increases the chance of exposure keratitis and corneal ulcers (Monteiro et al 2015). Leprosy is the rarest cause of bilateral facial palsy, the other common causes being Guillain-Barre syndrome, Parkinson's disease, Bell's palsy, sarcoidosis, Lyme's disease and diabetes (Keane 1994). Studies have shown that facial nerve palsy in leprosy is mild compared to the other aforementioned causes as only the zygomatic and buccal branches are involved in leprosy (Phillip et al 2022). Leprosy, presenting with palsies is more likely to be missed by the non-dermatologists especially cases of pure neuritic leprosy where there are no skin lesions. Thus, a high index of suspicion is to be entertained in all cases presenting with palsies. There are reports in literature of facial palsy and ulnar palsy being diagnosed as facio-brachial palsy (Lalla et al 2015). Facio-brachial palsy is defined as palsies involving the nerves of the facial and brachial plexus - median, ulnar and radial nerves (Lalla et al 2015). Mis-diagnosis of palsies due to leprosy may lead to delay and concomitant progression to irreversible grade 2 disability, keeping in mind that WHO and NLEP are striving for zero grade 2 disability in leprosy within the next decade. This case report highlights the increased need for awareness of leprosy related palsies not only among leprosy workers but also for non-dermatologist physicians.

References

1. Boku N, Lockwood DN, Balagon MV et al (2010). Impacts of the diagnosis of leprosy and of visible

- impairments amongst people affected by leprosy in Cebu, the Philippines. *Lepr Rev.* **81**: 111-120.
2. Inamadar AC, Palit A (2003). Bilateral facial palsy in Hansen's disease. *Lepr Rev.* **74**: 383-385.
 3. Keane JR (1994). Bilateral seventh nerve palsy: Analysis of 43 cases and review of the literature. *Neurology.* **44**: 1198-1202.
 4. Khan SRH, Marak A, Thakur S et al (2025). Bilateral facial nerve palsy in leprosy without lepra reaction: An uncommon presentation. *Indian J Lepr.* **97**: 435-441.
 5. Lalla R, Mulherkar RV, Misar PV (2015). Incomplete peripheral facial nerve palsy and ulnar neuropathy due to leprosy mistaken as facio-brachial stroke. *BMJ Case Rep.* Jun 23; **2015**: bcr2015210060.
 6. Monteiro LD, Martin-Melo FR, Brito AL et al (2015). Physical disabilities at diagnosis of leprosy in a hyperendemic area of Brazil: Trends and associated factors. *Lepr Rev.* **86**: 240-250.
 7. Nair SP, Mathew R (2017). Grade 2 disability in leprosy: Scenario in the post elimination phase of leprosy from a tertiary care center. *Indian J Lepr.* **89**: 127-137.
 8. Philip AS, George A, Mathew V et al (2022). Facial nerve and branch dysfunction in leprosy - a study from India. *Lepr Rev.* **93**: 377-391.
 9. Thompson KJ, Allardice GM, Babu GR et al (2006). Pattern of ocular morbidity and blindness in leprosy – a three centre study in Eastern India. *Lepr Rev.* **77**: 130-140.

How to cite this article : Cinderella X, Nair SP, Shima KT et al (2026). Hansen's Disease Borderline Tuberculoid Presenting with Bilateral Facial Nerve Palsy, Trigeminal and Ulnar Nerve Palsy. *Indian J Lepr.* **98**: 337-343.