

Palatal palsy in a case of lepromatous leprosy

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Summary A male patient with lepromatous leprosy developed nasal regurgitation of food due to palatal palsy during Type 2 reaction. Early high-dose administration of corticosteroid achieved a prompt therapeutic response and he completely recovered from palatal palsy. The associated lagophthalmos, foot drop and ulnar paralysis persisted.

Introduction

Mycobacterium leprae commonly affects the sensory fibres of the peripheral nerves, although motor fibres are often affected. The trigeminal and facial nerves are also commonly affected, but as the involvement of other cranial nerves is uncommon,^{1–6} we report a patient with lepromatous leprosy who developed a palatal palsy during Type 2 reaction.

Case report

A 45-year-old man (Figure 1) was diagnosed to have lepromatous leprosy on 3 March 1983 on clinical, bacteriological and histopathological features. His history and progress are summarized in Table 1.

Discussion

The central nervous system remains unaffected in leprosy, though the cranial nerves during a superficial course may be involved. In the patient reported here, two cranial nerves—the facial fibres of the cranial part of the eleventh nerve which travel in the

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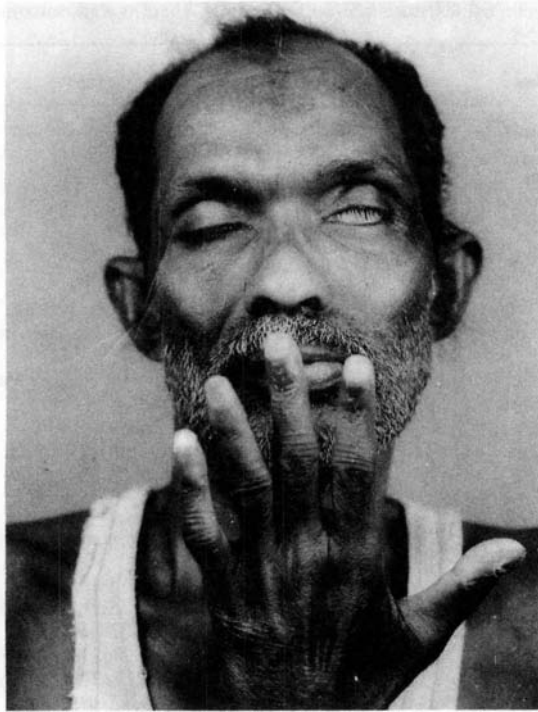


Figure 1. Note lagophthalmos of the left eye, the absence of nasolabial fold on the left side and clawing of the left hand.

vagus—were affected. Involvement of the zygomatic branch alone of the left facial nerve, resulting in lagophthalmos, and obliteration of the nasolabial fold suggested leprous aetiology for facial palsy in our patient. In most other conditions the facial nerve trunk, rather than its isolated branches, becomes affected, causing paralysis of all muscles of one-half of the face. The cranial nerves I, II, V, VII and VIII are affected in the patients reported by Katoch *et al.*² Involvement of the first and eighth cranial nerves has also been reported.^{1,3-6}

Paralysis of the palate is unusual in leprosy. Its occurrence in association during a Type 2 reaction is of interest, especially as the severity of nerve involvement is usually greater in Type 1 reactions. Masashi and Schigenobu⁸ reported bulbar palsy syndrome of leprosy in 6 patients with involvement of cranial nerves VII, IX, X and XI. They suggest an immunological mechanism as an aetiological factor. The most severe focus of inflammation was found in the nucleus ambiguus.⁹ Leprous granulomata may occur in the palate in lepromatous patients.¹⁰ The motor fibres derived from the nucleus ambiguus are distributed through the glossopharyngeal, vagus and cranial accessory nerves to the striated muscles of the palate, pharynx and larynx. With the exception of tensor palati, which is innervated by the mandibular division of the trigeminal nerve, muscles of the palate are supplied by fibres of the cranial part of the eleventh nerve which run in the vagus.¹¹ The normal CSF and isolated palatal palsy unassociated with

Table 1. Summary of the clinical features, laboratory findings, therapy and outcome

Date	Clinical features	Laboratory findings	Therapy	Outcome
3.3.83	Bilateral symmetrical multiple, shiny, ill-defined macules and plaques on face, trunk and limbs.	Slit-skin smears AFB. BI 6+, MI 68%. Skin biopsy features typical of LL leprosy. Routine blood and urine tests within normal limits.	Tab. dapsone 100 mg daily till 5.6.86.	Gradual clinical improvement with less infiltration of skin lesions. Gradual fall in MI to 42% on 6.6.86; BI 6+.
6.6.86	Type 2 reaction with fever, neuralgia and ENL lesions. Right and left ulnar and common peroneal nerves thickened and tender at elbow and popliteal fossa, respectively.	Slit-skin smears BI 6+ MI 42%. ESR 60 mm first hour, Blood TC 12,000 cells/c mm; VDRL negative, SGPT 40 IU/L. Chest X-ray normal.	Tab. dapsone 100 mg daily, Cap. Rifampicin 600 mg once a month, cap clofazimine 100 mg tid, ⁷ Tab Ibuprofen 400 mg tid till 23.6.86.	Type 2 reaction persisted till 20.8.1986.
24.6.88	Developed left lagophthalmos and obliteration of nasolabial fold on the left side. Ulnar clawing on the left side.	ESR 42mm/first hour.	Tab. prednisolone 40 mg daily (24.6.86–24.7.86, 30 mg daily till 20.8.86, 25 mg daily till 20.10.86, 20 mg daily till 20.12.86, 15 mg daily till 28.2.87, 10 mg daily till 30.4.87, then 5 mg daily till 16.7.87). Dapsone, rifampicin and clofazimine continued. Clofazimine dose reduced to 200 mg daily on 20.10.86, and then 100 mg daily 16.7.87 onwards. It was further reduced to 100 mg on alternate days from 10.10.1987.	Gradual regression of Type 2 reaction, steroid withdrawn on 16.7.87. Persistent lagophthalmos and ulnar paralysis, BI 6+, MI 10%. Antileprosy drugs continued.

paralysis of the pharynx and larynx in our patient suggest involvement distal to the nucleus ambiguus. Palatal palsy in our patient was probably caused by the bilateral involvement of the peripheral motor fibres that supply the striated muscles of the soft palate. Nasal regurgitation of the food was caused by the failure of the paralysed soft palate to shut off the nasopharynx during swallowing. This might suggest either the involvement of all muscles or that the tensor is too weak to affect sufficient movement. For the same reason his voice acquired a nasal resonance.¹² This report emphasizes the need to also consider leprosy in the differential diagnoses of palatal palsy. It also stresses the importance of administering corticosteroid at the earliest evidence of palatal palsy, which if unilateral may be completely asymptomatic. Another question is also raised by the persistent ulnar paralysis and foot drop. The possibility of early release or constriction at the cubital tunnel and at the neck of the fibula combined with steroid therapy merits consideration.

Table 1. Continued

Date	Clinical features	Laboratory findings	Therapy	Outcome
9.5.88	Recurrence of Type 2 reaction with ENL lesions. Developed foot drop left side, nasal regurgitation of food and nasal resonance of voice. Complete palatal paralysis, with absent reflex. Pharyngeal reflex retained. Normal palatal sensations.	Slit-skin smears BI 5+ MI 4%. CSF—cells, proteins within normal limits. Blood sugar fasting 80 mg %.	Tab. prednisolone 80 mg daily for 1 month, 60 mg daily for 1 month, 40 mg daily for 1 month, 30 mg daily for 1 month, 20 mg daily for 1 month, 15 mg daily for 1 month, 10 mg daily for 2 months, 5 mg daily for 2 months and stopped on 7.3.89. Dapsone, rifampicin and clofazimine continued. Dose of clofazimine 300 mg daily for 3 months, then 200 mg daily for 3 months, then 100 mg daily till 7.3.89. Then 100 mg alternate days.	Complete recovery from palatal palsy when seen on 7.3.89. Foot drop, claw hand and lagophthalmos unchanged.
7.3.89	Skin lesions, regressed leaving atrophic macules. Foot drop, lagophthalmos and claw hand persist.	Slit-skin smears AFB 2+MI.O. Histology of the sural nerve; thickened perineurium, nerve parenchyma replaced by fibrosis and hyaline degeneration. Many granular AFB in Schwaan cells and perineurial cells. No granuloma or amyloid. Skin biopsy. No granuloma. Fibrosis and scanty lymphocytic infiltration in dermis.	Dapsone 100 mg daily rifampicin 600 mg once a month, clofazimine 100 mg alternate days + 300 mg once a month ⁷ till 10.10.89 when the slit-skin smears were negative.	Antileprosy drugs stopped on 10.10.89. Lagophthalmos, foot drop and claw hand persist. Patient is on follow up. No relapse of the disease.

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