CASE REPORT

Incomplete peripheral facial nerve palsy and ulnar neuropathy due to leprosy mistaken as faciobrachial stroke

Rakesh Lalla,¹ Rupal Vijay Mulherkar,² Parag Vijay Misar³

¹Department of Neurology, Chhatrapati Shahuji Maharaj Medical University, UP, Lucknow, Uttar Pradesh, India

²Kaya Skin Clinic, Lucknow, Uttar Pradesh, India

³Department of Medicine, Lokmanya Tilak Municipal General Hospital and Lokmanya Tilak Municipal Medical College, Sion, Mumbai, Maharashtra, India

Correspondence to

Dr Rakesh Lalla,
rakesh_lalla@yahoo.com

Accepted 29 May 2015

SUMMARY

A middle-aged woman was referred to our hospital emergency ward in view of acute onset left faciobrachial weakness. An urgent MRI of the brain was performed, which did not reveal any abnormality and hence a neurology consultation was arranged in order to rule out acute stroke. However, examination and retrospective history taking proved to be a valuable aid in this patient's diagnosis. The incomplete lower motor neuron facial nerve palsy and hand weakness due to leprosy in reaction was confused by the general practitioner as a faciobrachial stroke.

BACKGROUND

Leprosy type 1 reaction is an important cause of acute onset multiple mononeuropathy and the most common cause of multiple mononeuropathy in India. Leprosy can notoriously affect multiple cranial nerves (VII and V most common) and is especially known to cause patchy or incomplete facial palsy. In this case report, we describe a patient of leprosy in reaction with incomplete left facial palsy and coincidental left hand grip weakness mimicking left faciobrachial stroke.

CASE PRESENTATION

A 45-year-old woman, a farmer by occupation, was referred to our hospital emergency ward by a general practitioner for acute weakness of the left half of her face and left hand of 3 days' duration. She also had pain in her left arm and was unable to perform fine movements due to weakness of left hand grip. She had no weakness in the lower limb. She denied any fever, vesicular rash or any major illness such as diabetes mellitus, stroke or ischaemic heart disease.

On general examination, there was hypopigmented and a hypoaesthetic patch on her left cheek, thickened nerves bilateral supraorbital, bilateral greater auricular (figure 1A), left ulnar, right radial and bilateral common peroneal nerves. Tinel sign was positive for left greater auricular nerve and left ulnar nerve. The facial nerve examination revealed incomplete lower motor neuron type (LMN) facial palsy, as a result, she was unable to close her eyes; Bells phenomenon was present and nasolabial fold (figure 1B) and forehead furrowing were intact (figure 1C). There was loss of sensation on the left cheek. The other cranial nerves were normal. Left hand grip was weak. There was no pronator drift, adduction of fingers and thumb was weak (Froment

sign positive), radial deviation was noted on wrist flexion and wrist extensors were normal. Hypoaesthesia was noted in the distribution of left ulnar and left sural nerve distribution. Deep tendon jerks were normal. Hoffmann sign was absent and both plantars had flexor response.

After examination, the patient was specifically asked about a history of Hansen's disease and any treatment received for it. After repeated enquiry, she admitted that she was receiving treatment for leprosy for the past 2 months.

INVESTIGATIONS

A complete haemogram, renal function tests and liver function tests were within normal limits. Erythrocyte sedimentation rate was 24. MRI of the brain was normal. Nerve conduction studies showed absent sensory nerve action potential in bilateral ulnar, median, right radial and left sural along with decreased compound motor action potentials amplitude in left ulnar, right median and left common peroneal nerve, with normal distal onset latencies and conduction velocities suggestive of an axonal type of multiple mononeuropathy. Slit skin smear for lepra bacilli was positive.

DIFFERENTIAL DIAGNOSIS

1. Multiple mononeuropathy due to leprosy type 1 reaction.
2. Acute left faciobrachial stroke.

TREATMENT

The patient was asked to continue her multidrug treatment for leprosy (rifampicin, clofazimine and dapsone). She was started on oral prednisolone 1 mg/kg for 4 weeks and a gradual taper was advised over the next 12 weeks.

OUTCOME AND FOLLOW-UP

At the time of writing, the weakness of the patient's face and hand grip responded partially and mild weakness still persists.

DISCUSSION

In upper motor neuron (UMN) palsy or central facial palsy, the lower half of the face, which has unilateral representation is completely paralysed, whereas eye closure and wrinkling of the forehead remain intact. Bell's phenomenon is a characteristic feature of LMN or peripheral facial palsy. However, LMN facial palsy patients have complete paralysis of the entire half of the face.

CrossMark

To cite: Lalla R, Mulherkar RV, Misar PV. *BMJ Case Rep* Published online: [please include Day Month Year] doi:10.1136/bcr-2015-210060

Figure 1 (A) Hypopigmented and hypoaesthetic patch noted on the left cheek, ear lobule (small arrows) and thickened left greater auricular nerve (bold arrow). (B) Lagophthalmus noted in the left eye and flattening of the left nasolabial fold. (C) The forehead furrows are intact (bold vertical arrow).

In this patient, incomplete peripheral facial palsy appears due to selective involvement of the zygomaticotemporal branch of the facial nerve. Hence the patient is unable to close her left eye, but the wrinkling of the forehead as well as the nasolabial fold is intact. Her general practitioner mistook this patient's facial weakness for UMN facial nerve palsy, as the forehead furrowing was intact. The patient also had difficulty gripping objects with her left hand due to involvement of the left ulnar nerve, which further added to the confusion, hence the mistaken diagnosis of left faciobrachial stroke. Leprosy is a highly stigmatised disease in this country and many fear to reveal it even to their family physicians.

The cranial nerves are frequently involved in leprosy. Facial nerve followed by trigeminal nerve are the most frequently

affected nerves in leprosy. The incidence of facial nerve involvement has been reported to be around 10%.^{1 2} Nerve disease in leprosy is more common in unstable subtypes of leprosy such as borderline tuberculoid (BT) or borderline lepromatous (BL) leprosy, with higher tendency for lepra reactions, thus there is also cranial nerve involvement in these subtypes (BT, BL).³ Cranial nerve involvement has been seen more often during lepra reaction 1 and is more common in BT leprosy.^{4 5} Wani *et al*⁵ showed that lepra reaction was a significant risk factor for new onset facial nerve palsy. Leprosy should always be ruled out in patients presenting with bilateral facial palsy.

Leprosy affects the facial nerve in a scattered pattern.⁶ Some authorities believe that there is preferential involvement of the zygomatic branch to the facial nerve and other superficial

Table 1 Clinical differentiating features between faciobrachial stroke and Leprosy type 1 reaction

Clinical signs	Faciobrachial stroke	Leprosy type 1 reaction
1 Facial weakness	Central type: sparing of upper face, intact furrowing of forehead, normal eye closure Bell's phenomenon absent Usually unilateral	Peripheral type: entire half of face. Patchy peripheral nerve involvement can lead to incomplete facial palsy including selective branches of facial nerve Bell's phenomenon is present Bilateral facial palsy is also common in leprosy
2 Facial sensory loss	Uncommon	Common
3 Thickened nerves	Absent	Present. In reaction, painful neuropathy is present. Nerves are enlarged and tender
4 Skin patches	Absent	Present
5 Arm weakness	UMN pattern: pronator drift, arm flexors stronger than extensors, pronators stronger than supinator. Loss of dexterity of fingers	Depends on the pattern of nerve involvement, for example, ulnar nerve innervated muscles. Common patterns are ulnar claw (ulnar nerve), wrist drop (radial nerve) or ape hand (median nerve)
6 Sensory loss	Rare. If present, entire half of face along with ipsilateral arm	Present in the pattern of nerve involved
7 Deep tendon reflexes	Normal or exaggerated	Normal or hypoactive
8 Hoffmann sign	Present	Absent

UMN, upper motor neuron.

nerves. The zygomatic branch lies superficial to zygoma and provides a lower temperature for proliferation of *Mycobacterium leprae*.⁷ Contrary to this theory, Turkof *et al*⁶ believe that haematogenous spread of lepra bacilli within epineural and perineural vessels may be the reason for skip lesions and selective involvement of branches of the facial nerve.

In this case, the premorbid history and tell-tale signs of leprosy-like thickened nerves, and the hypopigmented and hypoaesthetic patch helped us to make the correct diagnosis in this patient. The patient's fear of becoming an outcaste prevented her from revealing the history of leprosy to her treating physician. The patient was referred to our emergency services as stroke. The major difference in the semiology of stroke and peripheral nerve disease is the rapidity of onset of symptoms,

which is seen in stroke. Stroke is a sudden onset (within seconds to minutes) vascular event, whereas a lepra reaction will have an acute onset (within minutes to hours) neurodeficit. This feature was overlooked as the patient was not able to precisely reveal the rapidity of onset and progression of symptoms, and was probably therefore misdiagnosed. Another important lesson from this case is that prejudice should always be avoided and detailed neurological examination should never be overlooked. We have listed important differentiating clinical points (table 1) that counterindicated the diagnosis of stroke and helped us to arrive at the diagnosis of leprosy. This is extremely important in areas where leprosy and lepra reactions are not often seen in day to day practice by general physicians.

Competing interests None declared.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

Learning points

- ▶ There is no substitute for detailed history taking and clinical examination.
- ▶ Semiology usually helps to differentiate sudden onset diseases, including stroke from acute onset pathology such as lepra reaction.
- ▶ Leprosy is still considered a social stigma and hence patients may not voluntarily share this history unless specifically probed.
- ▶ Leprosy is the most common cause of incomplete peripheral facial palsy.
- ▶ Lepra reaction can present as acute leprous neuropathy or worsen the pre-existing neuropathy.

REFERENCES

- 1 Kumar S, Alexander M, Gnanamuthu C. Cranial nerve involvement in patients with leprous neuropathy. *Neurol India* 2006;54:283–5.
- 2 Gopinath DV, Thapa DM, Jaishankar TJ. A clinical study of the involvement of cranial nerves in leprosy. *Indian J Lepr* 2004;76:1–9.
- 3 Thompson K. An unusual presentation of facial palsy in leprosy; a case report. *Int J Lepr* 1995;63:567–9.
- 4 Ramadan W, Mourad B, Fadel W, *et al*. Clinical, electrophysiological and immunopathological study of peripheral nerves in Hansen's disease. *Lepr Rev* 2001;72:35–49.
- 5 Wani AA, Gupta V, Jan N. A clinical study of the cranial nerve involvement in leprosy. *J Egypt Dermatol* 2009;5:3.
- 6 Turkof E, Tambwekar S, Kamal S, *et al*. Leprosy affects facial nerves at the main trunk: neurolysis can possibly avoid transfer procedures. *Plast Reconstr Surg* 1998;102:1565–73.
- 7 Reichart PA, Srisuwan S, Metah D. Lesions of the facial trigeminal nerve in leprosy. *Int J Oral Surg* 1982;11:14–20.

Copyright 2015 BMJ Publishing Group. All rights reserved. For permission to reuse any of this content visit <http://group.bmj.com/group/rights-licensing/permissions>.
BMJ Case Report Fellows may re-use this article for personal use and teaching without any further permission.

Become a Fellow of BMJ Case Reports today and you can:

- ▶ Submit as many cases as you like
- ▶ Enjoy fast sympathetic peer review and rapid publication of accepted articles
- ▶ Access all the published articles
- ▶ Re-use any of the published material for personal use and teaching without further permission

For information on Institutional Fellowships contact consortiasales@bmjgroup.com

Visit casereports.bmj.com for more articles like this and to become a Fellow