Multiple sclerosis presenting as isolated peripheral facial nerve palsy

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ABSTRACT

Isolated facial nerve palsy is commonly seen with Bell's palsy. However, it can be associated with a central lesion at the level of the ipsilateral facial nucleus or the facial nerve at the pons. Multiple sclerosis is a chronic autoimmune inflammatory disease characterized by axonal degeneration and demyelination of the central nervous system. Isolated cranial neuropathies are rarely seen with multiple sclerosis. The patient being reported is a 20-year-old female who developed isolated facial nerve palsy, which was initially treated as Bell's palsy. However, she was found to have multiple sclerosis on MRI brain. Isolated facial nerve palsy due to multiple sclerosis is a rare scenario and can often get misdiagnosed and treated as Bell's palsy.

Keywords: *Bell palsy, Facial Nerve, Multiple sclerosis.*

Multiple sclerosis (MS) is a chronic autoimmune inflammatory disease affecting approximately 2.5 million people worldwide. The incidence is more among females, with a female-to-male ratio of 2:1. The disease is characterized by demyelination and axonal degeneration of the central nervous system [1-4]. MS is an uncommon cause of isolated cranial neuropathies, accounting up to 1.6% of all affected patients. The incidence of peripheral facial nerve palsy is only about 0.2% in patients with MS [5].

Bell's palsy, characterized by acute-onset unilateral facial paralysis, is the commonest cause for facial palsy. It has an idiopathic etiology, and the symptoms resolve over a period of 6 months [6,7]. The patient being reported developed unilateral facial nerve palsy and was diagnosed to have Bell's palsy. However, MRI of the brain revealed MS. Therefore, this case is aimed at emphasizing the importance of considering MS as a cause for isolated facial palsy, before attributing it to Bell's palsy.

CASE REPORT

A 20-year-old female presented initially to a local practitioner with 2 days history of sudden onset facial deviation to the right side. She also noticed mild slurring of speech and difficulty in closing her left eye. There were no other associated symptoms like paresis, paresthesia, lack of balance, visual disturbance, bladder problems or lack of coordination. She had a similar episode about 4 months ago which was treated as Bell's palsy with oral prednisolone (no brain imaging was done). Her symptoms got relieved within 3 weeks. This time again, she was told to have Bell's palsy and was started on oral prednisolone and acyclovir. She presented to us in view of recurrent episodes of facial palsy.

On examination, she was conscious, oriented and afebrile with heart rate 80/ minute, blood pressure 120/80 mmHg and respiratory rate 20/ minute with saturation 94% in room air. She had facial deviation to the right (Figure 1), loss of forehead creases and nasolabial fold on the left, inability to close her left eye (Figure 2) and Bell's phenomenon of the left eye. Other systemic examinations were normal.

Blood investigations like complete blood counts (haemoglobin 13 g/dL, total counts 9,200/mm^3 with neutrophils 71% and lymphocytes 29% and platelets 154,000/mm^3), renal (urea 24 mg/dL and creatinine 1.21 mg/dL) and liver function tests (total bilirubin 1.2 mg/dL, direct bilirubin 0.8 mg/dL, aspartate aminotransferase

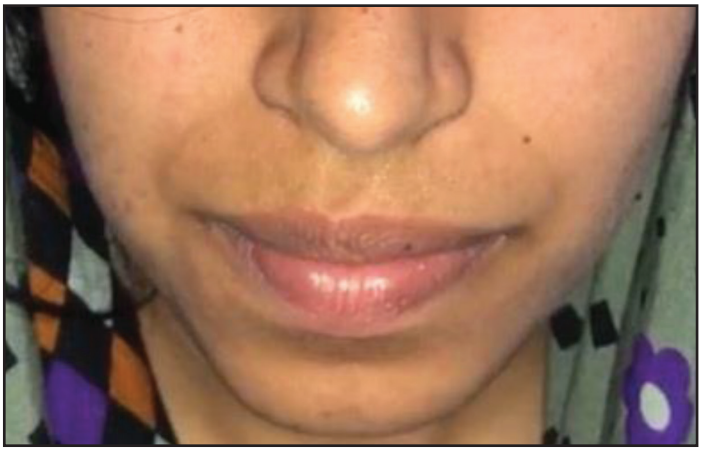

Figure 1: Facial deviation to the right

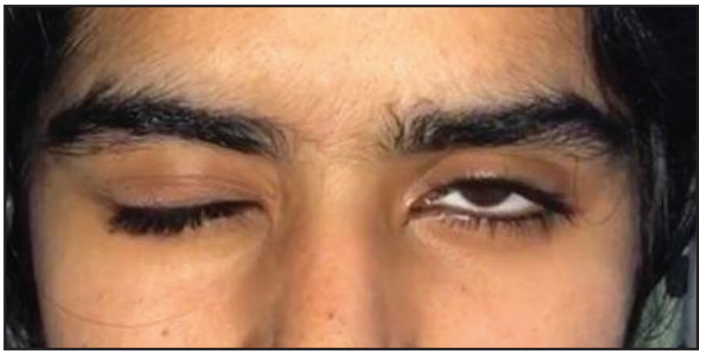

Figure 2: Inability to close the left eye

(AST) 32 IU/L, alanine aminotransferase (ALT) 44 IU/L, alkaline phosphatase (ALP) 175 IU/L and albumin 3.6 g/dL), electrolytes (sodium 138 mEq/L, potassium 4.6 mEq/L), calcium (8.9 mg/dL) and thyroid stimulating hormone (2.56 mIU/L) were normal. Antinuclear antibody (ANA) profile was negative. Viral markers (HIV, HBsAg and Anti-HCV) and Venereal Disease Research Laboratory (VDRL) test were also negative.

Magnetic resonance imaging of the brain (T2W) showed two well defined round hyperintense lesions along the midline of the pons and left middle cerebellar peduncle (Figure 3) suggestive of demyelination. Cerebrospinal fluid (CSF) analysis revealed oligoclonal bands. Auditory, visual and somatosensory evoked potentials were normal. Based on the clinical and brain imaging findings, a diagnosis of MS was made. She was started on pulse dose methylprednisolone (1 gram/ day for 5 days), followed by oral prednisolone in tapering doses. On review after 3 weeks, she was asymptomatic.

DISCUSSION

The facial nerve has two components: the motor fibers, responsible for muscles of facial expression, and the sensory fibers. Bell's palsy is responsible for about 72% of facial palsies and is usually seen following immunisation or viral infection. Other aetiologies include Mobius syndrome, motor neuron disease, cerebellopontine angle mass lesions, pontine lesions, diabetes mellitus, Lyme disease and HIV infection [7-11].

Patients with Bell's palsy have an acute-onset unilateral facial muscles paresis along with numbness of the affected side without any sensory deficit. Loss of lacrimation, ipsilateral hyperacusis, taste disorders, pain behind the ear are some of the other features [12-14]. In cases of Bell's palsy, the recommendations for clinical practice are: (a) assessment of history and physical examination in order to exclude other causes of acute onset unilateral facial paresis or paralysis; (b) administration of oral steroids within 72 hours of symptom onset in patients of age 16 years and above; (c) avoiding oral antivirals in patients with new-onset Bell's palsy and (d) the use of eye protection in those with impaired eye closure.

Other recommendations include (a) avoiding routine laboratory testing in new-onset Bell's palsy; (b) avoiding diagnostic imaging routinely; (c) avoiding electrodiagnostic testing in those with incomplete facial paralysis and (d) reassessment or reference to a facial nerve specialist in case of new or worsening neurological findings, incomplete facial recovery within 3 months following the onset of initial symptom or development of ocular symptoms [15].

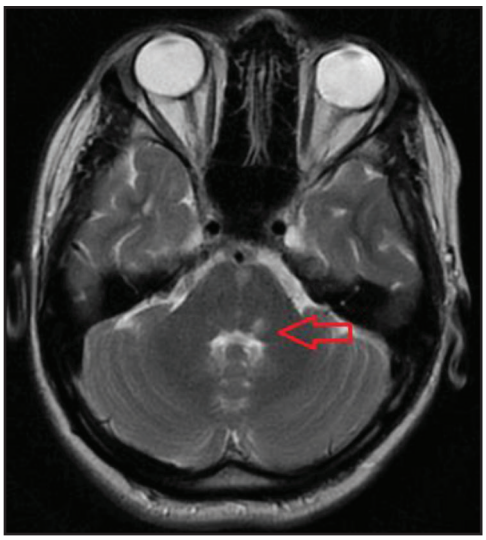

Figure 3: MRI brain with contrast (T2W) showing two well defined round hyperintense lesions along the midline of pons and left middle cerebellar penduncle

MS is defined as a disease of the central nervous system, while Bell's palsy involves the peripheral nervous system. Visual disturbances, paresthesia, impaired coordination, and paresis are the common symptoms of MS. Other features include bladder and sexual dysfunction, Lhermitte's sign and ataxia. These symptoms resolve spontaneously and tend to recur after months or even years [1]. Pontine lesions may present as facial palsy. The facial nerve on the same side of the lesion may get damaged at the fascicular level; thereby causing a lower motor neuron lesion on the same side. However, if the upper motor neuron fibers to the opposite seventh nerve are involved as they decussate, an upper motor seventh nerve lesion can be seen on the opposite side [16]. Though brain stem involvement in MS is common [17], isolated peripheral nerve palsies are rare [5,18-20].

The differential diagnosis for lower motor neuron facial palsy include trauma, infections (atticoantral disease, acute suppurative otitis media, tuberculous otitis media, Ramsay syndrome, malignant otitis externa, acute parotitis), Bell's palsy, tumours (acoustic neuroma, parotid carcinoma, carcinoma middle ear, schwannoma 7th nerve) and Melkersson Rosenthal disease [21]. With less than a handful of cases being reported, MS as a cause for facial nerve palsy is a rare scenario [22,23].

CONCLUSION

Although brain imaging should not be routinely performed in cases of new-onset Bell's palsy, conditions like MS and other pontine lesions, which can rarely present as isolated facial

nerve palsy, can masquerade as Bell's palsy, thereby warranting diagnostic imaging.

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