Isolated Horizontal Gaze Palsy as a Presenting Symptom in Multiple Sclerosis

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Abstract
Two cases of isolated horizontal gaze palsy resulting from a demyelinating lesion are described. A brain magnetic resonance image confirmed in both cases that the lesions affected the region of the abducens nucleus in the posterior and medial part of the lower pons, either bilaterally or unilaterally. The patients were treated with intravenous methylprednisolone (1000mg/day for 5 days) and demonstrated marked improvement of horizontal gaze palsy. These results indicate that isolated horizontal gaze palsy may be the initial manifestation of relapse in cases of suspected multiple sclerosis.

Keywords: Abducens nucleus, horizontal gaze palsy, pons, multiple sclerosis

Multiple sclerosis (MS) is a chronic autoimmune, demyelinating disease of the central nervous system. In multiple sclerosis, although eye movement abnormalities and bilateral internuclear ophthalmoplegia are frequent, a “one and a half syndrome” or a complete unilateral or bilateral horizontal gaze paralysis are rare.\(^1,2\)

We report two patients of acute selective bilateral or unilateral horizontal gaze palsy associated with a small demyelinating lesion in the region of abducens nucleus.

Case 1
A 20 old woman with unremarkable medical history presented for sudden onset of diplopia and weakness of the left face muscles. Neuro-ophthalmological examination revealed that visual acuity, light reflex and fundoscopy were normal. Complete bilateral horizontal gaze paralysis was seen, with absence of horizontal saccades, pursuit and oculosephalic movements. Vertical eye movements were normal. There was also a left peripheral facial paresis, but the rest of the neurological examination was normal.

Brain Magnetic Resonance Imaging (MRI) on T2 weighted image showed a few bilateral white matter signals in the lateral ventricular regions and a lesion in the brainstem, affecting bilaterally posterior part of the lower pons, the region of the bilateral abducens nucleus (Fig. 1). The investigation included routine blood tests and cerebrospinal fluid analysis were unremarkable, apart from oligoclonal immunoglobulin G (IgG) bands. Results of visual, auditory and somatosensory were normal. Electromyography and edrofonium test were completely normal. The diagnosis of possible MS was made after further tests had revealed no other inflammatory and infectious etiology. MS was also confirmed clinically when the patient presented with an acute left hemiparesis relapse 9 months after bilateral horizontal gaze paralysis. Treatment with intravenous methylprednisolone (1000mg/day for 5 days) was started and after two weeks, improved in bilateral horizontal gaze palsy, followed by gradual restoration of left facial palsy. One month later, left facial palsy had recovered completely.

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Case 2

A 34 year old women presented for sudden onset horizontal diplopia. Neuro-ophtalmological examination revealed that visual acuity, light reflex and fundoscopy were normal. On rightward gaze, she had an abduction deficit in the right eye and adduction deficit in the left eye with absence of saccades, pursuit, and oculocephalic movements. On leftward gaze was normal. Vertical eye movements were normal in both eyes. Brain MRI showed a few bilateral white matter signals on T2 weighted images in the lateral ventricular regions and a single lesion in the left posterior part of the pons, the region of the left abducens nucleus (Fig. 2).

Analysis of CSF showed normal cell and glucose levels, but intrathecal IgG production and positive oligoclonal bands. Visually, auditory and somatosensory evoked potentials were normal. The patient was treated with intravenous methylprednisolone (1000mg/day for 5 days) showing marked improvement of unilateral horizontal gaze palsy after two weeks.

Discussion

Horizontal gaze palsy is a rare clinical finding and most often due to pontine ischaemic, haemorrhagic or neoplastic lesions.[3, 4] Although clinically isolated syndromes often present as brainstem symptoms, while isolated horizontal gaze palsy is rare in MS patients.[5, 6, 7] Complete unilateral or bilateral horizontal gaze palsy implies lesions of the pontine tegmentum involving the abducens nucleus with or without involvement of the paramedian pontine reticular formation (PPRF). The neural signals encoding saccadic, pursuit, vestibular and optokinetic eye movements project independently to the abducens nucleus. In addition, the PPRF seems important for the generation of horizontal and vertical saccades.[8] The common pathway of horizontal gaze movements begins in the abducens nucleus, which contains two populations of neurons: the motor neurons of the sixth nerve controlling abduction and the internuclear neurons controlling adduction via the medial longitudinal fasciculus to the rectus medialis subnucleus of the contralateral oculomotor nucleus complex.[9] A lesion of the abducens nucleus therefore results in an ipsilateral horizontal gaze palsy, and not an isolated sixth nerve palsy. In our cases, complete bilateral or unilateral horizontal gaze palsy implies a lesion of abducens nucleus in dorso-medial tegmental pontine area.

The abducens nucleus is located in the facial colliculus on the floor of the fourth ventricle at the level of the mid to lower portion of the pons.[10] Furthermore, in case 1, the bilateral horizontal gaze paralysis with a left peripheral facial paresis and was the initial clinical signs, likely due to multiple sclerosis. Peripheral facial paresis may be explained by the damage of facial fibres, somewhere in their course just anteriorly or around the abducens nucleus.[10]

Isolated horizontal gaze palsy, which may be part of the clinical abducens nucleus syndrome can rarely occur as the sole manifestation of demyelinating CNS disease, which resulted in a definite diagnosis of relapsing-remitting MS in young adults.

Disclosures

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