Impairment of Vertical Saccades From an Acute Pontine Lesion in Multiple Sclerosis

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Abstract: A 62-year-old woman with relapsing-remitting multiple sclerosis suddenly complained of diplopia associated with bilateral adduction impairment, nystagmus of the abducting eye bilaterally, and sparing of abduction, convergence, and vertical eye movements, consistent with bilateral internuclear ophthalmoplegia. Within 1 week, she had developed a complete horizontal gaze paralysis even with the oculocephalic maneuver. Vertical saccades were slow and convergence was preserved. There was a right lower motor neuron seventh cranial nerve palsy. Brain MRI showed a new enhancing lesion involving the pontine tegmentum. Clinical and MRI follow-up showed recovery after 6 months. The slowing of vertical saccades may have been due to spread of the demyelinating lesion to the adjacent paramedian pontine reticular formation, which contains omnipause neurons lying in the raphe interpositus nucleus thought to inhibit excitatory burst neurons for horizontal and vertical saccades. Our patient verifies the fact that vertical saccadic abnormalities may occur from a lesion apparently confined to the pons.

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Nearly 30% of patients with multiple sclerosis (MS) experience internuclear ophthalmoplegia (INO) at some time during the course of disease (1). This eye movement disorder is caused by a lesion of the medial longitudinal fasciculus (MLF). In some patients, adjacent structures may be involved, causing a more complex clinical picture, including complete horizontal gaze paralysis (2). Recently, unusual ocular motor findings have been described in patients with MS, including bilateral third nerve palsy, opsoclonus, and isolated sixth cranial nerve palsy (3). Here, we report the case of a patient with MS who presented with acute bilateral INO followed 1 week later by complete transient horizontal gaze paralysis associated with slow vertical saccades. This patient verifies the fact that a lesion apparently confined, by imaging criteria, to the pons can cause a vertical saccadic impairment.

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CASE REPORT

A 62-year-old woman with relapsing-remitting MS diagnosed at age 44 complained of diplopia for 4 days. She denied previous episodes of diplopia and previous examinations had revealed no ocular motor abnormalities. She was not undergoing immunomodulatory or immunosuppressive treatment.

Neurologic examination showed complete adduction loss and nystagmus of the abducting eye bilaterally. Abduction, convergence, and vertical eye movements were spared. A diagnosis of bilateral INO was made. Examination 48 hours later showed complete horizontal gaze paralysis even with the oculocephalic maneuver. The vertical vestibular-ocular reflex (VOR) was clinically normal, but vertical pursuit was interrupted by saccades. Vertical saccades appeared slow, but convergence was preserved. There was also a right lower motor neuron seventh cranial nerve palsy.

Treatment with intravenous methylprednisolone (1000 mg/day for 5 days) was started and, after a few days, adduction dramatically improved in both eyes, followed by gradual restoration of vertical eye movements.

Brain MRI, performed 21 days after the onset of complete horizontal gaze paralysis, showed a T2 lesion located at the right pontine tegmentum with extension to the left side. This lesion had not been present on an MRI performed 7 months earlier. Only the right side of the lesion enhanced on T1. No enhancing lesions were observed in the midbrain or elsewhere in the brain (Fig. 1).