Internuclear ophthalmoplegia due to brainstem infarction

Jong Kim describes 30 patients presenting with INO as the sole or predominant sign of brainstem infarction. Etiologies included penetrating artery occlusion, atheromatous branch disease, or major vessel occlusion. Although the prognosis was excellent, INO tended to persist when there were other neurologic deficits.

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Double trouble: Internuclear ophthalmoplegia and stroke

Commentary by Steve L. Galetta

The medial longitudinal fasciculus (MLF) is a richly myelinated white matter tract that travels prominently in the dorsal ponto-mesencephalic region. Injury to the MLF produces one of the most discrete localizing signs in neurology: internuclear ophthalmoplegia (INO). Lesions involving the MLF produce a syndrome characterized by partial or complete failure of adduction and associated abducting nystagmus of the fellow eye. Classic teaching states that when the INO is bilateral and occurs in a young patient, multiple sclerosis is the most likely etiology. On the other hand, a unilateral INO in an elderly patient implies a vascular basis.

Injury to the MLF may be associated with other signs, including a skew deviation. Patients with INO usually have an ipsilateral hypertropia because the otolithic pathways responsible for ocular counter roll travel in the MLF. In some patients with unilateral INO, a dissociated vertical nystagmus will be evident. Interruption of the posterior canal pathways ascending in the MLF may result in nystagmus that is downbeat in the ipsilateral eye and torsional in the contralateral eye. Fibers responsible for vertical gaze stabilization also travel in the MLF. Injury to the MLF bilaterally is typically associated with vertical gaze holding deficits and

impaired vertical pursuit. The gaze holding deficits are most evident in attempted upgaze where an upbeat nystagmus may be observed.

Neuroimaging has greatly improved our ability to resolve the lesions responsible for INO. Frohman et al. have demonstrated that virtually all MLF lesions in patients with multiple sclerosis may be detected by directed MR imaging.[1] Diffusion tensor imaging with its ability to resolve white matter tracts may hold even greater promise to detect lesions involving the MLF.

Since INO of vascular origin may occur in isolation or with minimal signs of other neurologic dysfunction, most authorities have felt that small vessel ischemia was the likely mechanism of injury. Kim's study demonstrates that there is more than one cause of INO in stroke. INO may result from small vessel ischemia or large vessel disease involving the posterior cerebral artery, superior cerebellar artery, or even the basilarartery. This observation may have increasingly important implications as therapeutic options for stroke expand. As thrombolytic drugs and vascular stenting techniques evolve, it may be important to determine whether a large vessel or small vessel process is responsible for a given brainstem stroke.

This study also has potential important implications for other microvascular palsies such as those producing III, IV, and VI nerve dysfunction. It has become recognized that some microvascular ocular motor palsies thought to be peripheral in origin may result from intramedullary lesions.[2] As new therapeutics emerge, neuroimaging may take on a more important role in defining the type and location of vascular injury. Such technological advances may define more precisely the management of patients with ischemic internuclear ophthalmoplegia and other small brainstem strokes.

References

1. Frohman EM, Zhang H, Kramer PD, et al. MRI characteristics of the MLF in MS patients with chronic internuclear ophthalmoparesis. Neurology 2001;57:762–768.
2. Chou KL, Galetta SL, Liu GT, et al. Acute ocular motor mononeuropathies: prospective study of the roles of neuro-imaging and clinical assessment. J Neurol Sci 2004 (in press).

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