Internuclear ophthalmoplegia following head injury

Case report

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The most common causes of internuclear ophthalmoplegia (INO) are multiple sclerosis and vascular disease of the brain stem. Rarer causes are tumor, Arnold-Chiari malformation, and syphilis. Myasthenia gravis has, on occasion, presented with ocular abnormalities indistinguishable from INO. A case is described of brief duration following head trauma. There were no other brain-stem abnormalities. This brings to 11 the number of reported patients in whom head trauma precipitated this abnormality.

KEY WORDS • head injury • brain stem • internuclear ophthalmoplegia

Bilateral internuclear ophthalmoplegia (INO) as an isolated finding is rarely caused by disorders other than a demyelinating disease. We describe a patient in whom bilateral INO developed 10 hours after a closed head injury and resolved 48 hours later. Attention is drawn to 10 previous reports of INO following head trauma.

Case Report

This 19-year-old woman suffered a closed head injury in an automobile accident which rendered her unconscious for 2 hours. The point of impact was uncertain, but she had bruises behind both mastoids and over the left frontal region. Her neck was painful and she preferred not to move it. She had regained consciousness by the time of evaluation in the emergency room, and no neurological abnormalities were described. Her past history was positive only for common migraine. She was discharged after skull and cervical spine x-ray films were ascertained to be normal. Eight hours later, on awaking from sleep she noticed she had double vision.

On examination, approximately 12 hours after the head injury, the patient was alert and of normal intelligence. There was tenderness of the scalp in the areas bruised. The face showed no signs of trauma; the nose had been mildly deviated to the left since childhood. Several areas of tenderness and bruising were scattered over the trunk and limbs. She had suffered no fractures.

The sense of smell was intact. Visual acuity was 20/20 in each eye. Fundoscopic examination was normal. The pupils were symmetrical, reacted normally to light, and attempted accommodation. In the neutral position with no fixation the eyes were horizontally divergent, the right being further deviated than the left and mildly hypotropic. Upward and downward movements in the midline revealed only defective elevation of the right eye. On attempted left lateral gaze, the right eye failed to adduct and there was nystagmus of the abducting left eye. Lateral gaze to the right produced nystagmus of small amplitude in the right eye and a limitation in adduction of the left eye. Attempted convergence produced normal movement of the left eye but severe limitation in movement of the right. The right eye was also limited in upward movement when abducted. There were no other cranial nerve abnormalities. Power was normal in all extremities. The deep tendon reflexes were normal, plantar reflexes were flexor, and the abdominal reflexes were present and symmetrical. Sensory examination was normal, and tests of cerebellar functions were well performed. Computerized tomography scanning was normal. The cerebrospinal fluid (CSF) protein was 32 mg/100 ml, and glucose 65 mg/100 ml; there were seven red blood cells and one mononuclear white blood cell; CSF immunoglobulin G (IgG) was...
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4.2 mg%. Serum VDRL (venereal disease research laboratory) test was negative.

Within 12 hours of the initial examination, the patient's signs were resolving. The red-glass test confirmed persistent bilateral weakness of the medial rectus, worse on the right than the left, and right hypotropia, but nystagmus of the abducting eye on lateral gaze had resolved. Optokinetic nystagmus was of lesser amplitude in the right eye than in the left when the tape was moving from the patient's left to right, and of lesser amplitude in the left eye when the tape moved in the opposite direction.

Caloric stimulation of the left ear with cold water produced deviation of the eyes to the left followed by nystagmus of symmetrical amplitude, but when the right ear was stimulated, nystagmus in the right eye was of lesser amplitude than the left. By 48 hours after the onset no diplopia was present in any direction of gaze. The sole ocular abnormality was slowness of left lateral gaze and a visible increase in the jerkiness of saccades to that side. This problem persisted for 2 weeks.

**Discussion**

This patient's ocular abnormalities fulfill the criteria of bilateral INO, worse on the right than the left, with skewed deviation. This abnormality reflects disturbance of function of the medial longitudinal fasciculus (MLF) bilaterally. Neither the abnormality of convergence nor the exotropia in primary position differentiate a pontine from a midbrain lesion. Bilateral INO has been described as being "virtually pathognomonic" of multiple sclerosis (MS). In Smith and Cogan's review of 29 cases, 28 had MS; the other had tabes dorsalis. Unilateral INO has a striking association with vascular lesions. Cogan stated that bilateral INO is probably present in many diseases of the brain stem but is often masked by other signs. Gonyea's careful search for bilateral INO in stroke cases, however, revealed a surprisingly high incidence. It may be that the reported posttrauma cases represent only those in which the condition was not overshadowed by co-existent cortical and brain-stem disease.

In all the reported cases the INO was a prominent part of the clinical picture, but most patients had other signs of brain-stem impairment. Our patient is unusual in this regard as the INO was her only abnormality. Unconsciousness occurred in six of the 11 trauma cases, but was of less than 24 hours' duration in all but one case (Case 9, Table 1). Severity of head injury was clearly trivial in two cases (Cases 3 and 5, Table 1), suggesting that the trauma may have been coincidental to the brain-stem abnormality, which may have been of a more usual etiology.

Walsh and Hoyt argue that the trivial injury and the bilaterality of lesions in their patient make the diagnosis of disseminated sclerosis unavoidable. The statistical relationship remains controversial, but occasionally an acute attack of MS seems to be precipitated by trauma. In the case described by Walsh and Hoyt, however, no subsequent attack was documented. Dr. David Clark continued to see this boy for a period of 1½ years. His brain-stem signs completely resolved, and he remained neurologically normal. No evidence for demyelinating disease has been put forward in any of the reported cases; however, little follow-up information is available. The patient described in this paper has been neurologically intact for a period of 1 year.

Rich, et al., suggest shear forces within the brain stem could be generated by the initial trauma, stretching or tearing the nerve fibers. These forces are known to be maximal near the ventricular system and so the MLF would be in a vulnerable position. Movement of the brain stem has been documented with supratentorial pressure. Both Rich, et al., and Devereaux, et al., quote these studies which disclosed the following two types of brain-stem displacement: 1) downward shift with the posterior portion moving more than the anterior because of anterior fixation by penetrating arterioles from basilar artery, and 2) compression by hippocampal transtentorial herniation with elongation of the midbrain in
TABLE 1
Tabulation of findings in 11 patients with internuclear ophthalmoplegia following head trauma*

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Authors, Year</th>
<th>Type of Injury</th>
<th>Unconscious</th>
<th>Time to Onset and Type of MLF Impairment</th>
<th>Other Signs</th>
<th>Time to Recovery of MLF Function</th>
<th>Proposed Mechanism</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Bielschowsky, 1903</td>
<td>struck head in occipital region</td>
<td>?</td>
<td>1 day, bilat</td>
<td>nausea, dizziness, vertical nystagmus on upward &amp; downward gaze</td>
<td>beginning at several days, still not complete at 1 yr</td>
<td>minute hemorrhages in oculomotor nuclei</td>
</tr>
<tr>
<td>2</td>
<td>Jaensch, 1924</td>
<td>struck on head by heavy object</td>
<td>less than 24 hrs</td>
<td>1 day, rt-sided</td>
<td>generalized seizures, It 7th-nerve palsy, It Horner's syndrome</td>
<td>beginning at several days, not complete at 3 mos</td>
<td>direct injury to MLF, possibly hemorrhage</td>
</tr>
<tr>
<td>3</td>
<td>Walsh &amp; Hoyt, 1969</td>
<td>struck on forehead by fist</td>
<td>none</td>
<td>within 1 wk, first It, then bilat</td>
<td>numbness &amp; ataxia of rt side of body, mild head tilt</td>
<td>not given</td>
<td>disseminated sclerosis</td>
</tr>
<tr>
<td>5</td>
<td>Tanaka, et al., 1973</td>
<td>trivial</td>
<td>none</td>
<td>immediate, It-sided</td>
<td>none</td>
<td>none</td>
<td>brain-stem ischemia</td>
</tr>
<tr>
<td>6</td>
<td>Sato, et al., 1974</td>
<td>struck head on ice while skating</td>
<td>1 hr</td>
<td>2 days, bilat</td>
<td>numbness &amp; hyporeflexia It arm, It ptosis, lack of awareness of bladder filling, limited vertical movements of It eye, unsteady gait, falling to It</td>
<td>4 mos</td>
<td>brain-stem ischemia</td>
</tr>
<tr>
<td>7</td>
<td>Rich, et al., 1974</td>
<td>struck by automobile</td>
<td>less than 24 hrs</td>
<td>present by 24 hrs, bilat</td>
<td>multiple anterior skull fractures with rhinorrhea, loss of smell &amp; skew deviation</td>
<td>began at 6 wks, still improving at 1 yr</td>
<td>shear forces within brain stem resulting in stretched or torn nerve fibers</td>
</tr>
<tr>
<td>8</td>
<td>Zauel &amp; Carlow, 1977</td>
<td>chiropractic cervical manipulation</td>
<td>none</td>
<td>immediate, It-sided</td>
<td>skew deviation, rt hemiparesis</td>
<td>?</td>
<td>brain-stem ischemia</td>
</tr>
<tr>
<td>9</td>
<td>Devereaux, et al., 1979</td>
<td>fell striking head with development of bilat SDH</td>
<td>for 1 day, 1 wk after injury</td>
<td>2 wks after injury, It-sided</td>
<td>signs of transtentorial hematoma preop, rt homonymous hemianopia &amp; impaired recent memory</td>
<td>complete recovery at 6 mos</td>
<td>brain-stem ischemia</td>
</tr>
<tr>
<td>10</td>
<td>frequent falls, chronic SDH</td>
<td>uncertain time</td>
<td>within 1 wk of presumed alcoholic stupor, bilat</td>
<td>It-sided weakness, chronic It SDH</td>
<td>beginning after several mos</td>
<td>brain-stem ischemia</td>
<td></td>
</tr>
<tr>
<td>11</td>
<td>Baker, 1979</td>
<td>closed head injury in automobile accident</td>
<td>2 hrs</td>
<td>8 hrs, bilat</td>
<td>none</td>
<td>48 hrs</td>
<td>brain-stem ischemia</td>
</tr>
</tbody>
</table>

*MLF = medial longitudinal fasciculus; SDH = subdural hematoma.

the anteroposterior dimension. Devereaux, et al.,4 pointed out that, rather than shear forces in the fiber tract itself, the MLF could be rendered ischemic by the brain-stem movement stretching the perforating arteries of the basilar artery. The MLF is in the vascular watershed area of these arteries and would be expected to be preferentially affected by a temporary decrease in flow.

Zauel and Carlow17 added weight to the argument of ischemic etiology with their description of a case following cervical manipulation (Case 8, Table 1). Shear forces would not be expected in this circumstance, but vertebrobasilar insufficiency is readily understandable. The present author finds this proposed mechanism the most attractive in the absence of experimental evidence.
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In summary, although INO is most frequently caused by MS when it is bilateral, and by vascular disease when unilateral, the lesion is less specific than was previously thought. The bilateral lesion is not infrequent in vascular disease, and both unilateral and bilateral MLF lesions have been seen consequent to head trauma. This lack of specificity is not surprising in a relatively long, fast-conducting fiber tract of which the dysfunction is so readily appreciated by both patient and physician.

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References

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