Title: Isolated Oculomotor Nerve Palsy in Patients with Mild Head Injury

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Abstract

Isolated oculomotor nerve palsy following head injury is uncommon. It can only be diagnosed with confidence if it is known to have developed immediately following trauma and if adequate investigations exclude secondary causes. Recovery is only partial and this has repercussion on patients' quality of life.

Clinical details

Oculomotor nerve (CNIII) palsy after a head injury usually occurs secondary to compression from expanding haematomas, orbital wall and sphenoid bone fractures. A primary traumatic oculomotor nerve palsy, on the contrary, is an unusual condition which is not life threatening, but is thought to carry a poor prognosis in terms of recovery[1]. It can only be diagnosed with confidence if it is known to have developed immediately following trauma and if adequate investigations exclude secondary causes.

Three patients with CNIII palsy following mild head trauma presented to our department within a month (mechanism of injury in Table 1). They were all noted to have a dilated, unreactive pupil with the eye being 'down and out' at the scene of the accident. On further examination, in each of the three cases, ptosis with preserved abduction and intorsion, but with no adduction past the midline and no elevation was found, consistent with an isolated CNIII palsy. Non-contrast CT head findings are presented in table 1; CT head angiogram did not identify any vascular abnormalities.

High resolution contrast-enhanced MRI brain was performed in 2 patients 8 and 20 days after injury and revealed contrast enhancement and mild thickening of the cisternal segment of the ipsilateral CNIII in both cases, a finding reported in the subacute phase following traumatic CNIII palsy[1,2,3] and is believed to represent intraneural oedema[3]. Our third patient had a non-contrast MRI brain which revealed a small contusion in the ipsilateral temporal lobe (not visible on CT), but no specific changes of the CNIII. In all three cases, the remainder of the brain appeared normal. Follow up in the neurosurgical brain injury and ophthalmology clinics at 3, 6 and 9 months after injury revealed that the ptosis recovered first and that the recovery can indeed be complete. The range of extraocular movement showed a modest recovery, but pupillary size and reaction did not seem to recover.

Discussion

CNIII palsy in the context of head injury may arise from causes other than uncal herniation, orbital or sphenoid bone fractures which has implications for treatment and prognosis. High-resolution MRI is more sensitive in detecting the subtle changes underlying the isolated CNIII palsy than other imaging modalities.

These patients need regular neuro-ophthalmology input and follow up. We found that the ptosis recovers first and can resolve completely. This was, unfortunately, not the case with extraocular movements which showed only a modest recovery and pupillary size and reaction which did not seem to recover at all. This leaves patients with an unwanted cosmetic defect, double vision and glare. As a

consequence, they are unable to drive for long periods after injury and, possibly, even indefinitely.
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