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Case Reports & Case Series

Traumatic oculomotor nerve palsy: Unique case of isolated mydriasis and ptosis following head injury

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ABSTRACT

A rare case of partial cranial nerve 3 (CN3) paresis following a traumatic brain injury presented with isolated ptosis and mydriasis due to a hemorrhagic lesion in the ipsilateral tegmentum of midbrain. All the extraocular muscles that control eye movements were spared. The anatomy of CN3 nuclei and their fascicular organization is reviewed.

1. Introduction

The oculomotor nerve (CN3) arises from two nuclei in the midbrain: the oculomotor nucleus and Edinger-Westphal nucleus. The subdivisions of oculomotor nucleus give rise to differentiated fascicles that each separately innervate the skeletal muscles of levator palpebrae superioris and 4 of the 6 extraocular muscles (i.e., superior rectus, medial rectus, inferior rectus, and inferior oblique). The paired Edinger-Westphal nucleus supplies the parasympathetic portion of the CN3 responsible for pupillary constriction and lens accommodation. A variety of clinical presentation of CN3 palsy can be the consequence of CN3 dysfunction depending on the location of the lesion. All previously reported cases of post-traumatic CN3 paresis presented with some degree of ocular motility deficits. Here we report a rare case of partial CN3 paresis presented with unilateral isolated mydriasis and ptosis following a traumatic brain injury.

2. Case report

A 17-year-old previously healthy female was involved in a pedestrian versus motor vehicle accident. She presented to the hospital with a reduced level of consciousness (GCS 6/15), seizure, and left-sided non-reactive pupil dilation. Her head imaging studies (CT and MRI) identified evidence of diffuse axonal injury involving the left-sided cerebrum, cerebellum and brainstem. There was no finding of restricted diffusion to indicate focal acute ischemic change, mass effect, midline shift or herniation. No injury to orbits or eyes was identified. Her brain injury was managed conservatively without surgical intervention.

Approximately 6 weeks post the injury this patient was admitted to our Acquired Brain Injury Rehabilitation Community Reintegration Unit. Physical examination on admission did not reveal focal neurological deficit other than a left-sided incomplete ptosis and mydriasis with sluggish light reflex. The right side was normal. Interestingly, eye movements were intact bilaterally and she denied diplopia following the injury. There was no nystagmus or saccadic oscillation. All earlier medical records from the acute medical unit indicated intact oculomotor function, with the exception of the above-mentioned findings of ptosis and pupil dilation. The brain MRI study 2 days after the injury demonstrated a small intra-axial traumatic hemorrhage in the left-sided midbrain at the level of superior colliculus, as shown in Fig. 1.

3. Discussion

Common causes of CN3 dysfunction are diabetic neuropathy, aneurysm, and head trauma. Partial cranial nerve 3 palsy with isolated mydriasis sparing the extraocular muscles is usually associated with an extra-axial lesion, particularly with vascular compression. Typical signs of complete CN3 palsy include a completely closed eyelid, dilated and nonreactive pupil, and oculomotor palsy with the outward and downward deviation of the eye. Diplopia is associated with ocular motility impairment. The clinical manifestation of CN3 palsy resulting from an intra-axial lesion varies and depends on the location of the lesion.

The oculomotor nucleus and Edinger-Westphal nucleus locate at the level of the superior colliculus in the midbrain tegmentum. The nerve fibers from the CN3 nuclei run ventrally and exit through the interpeduncular fossa. The topographic organization of oculomotor nerve fascicles within the midbrain is not completely elucidated in humans. However, the oculomotor nucleus appears to be organized into

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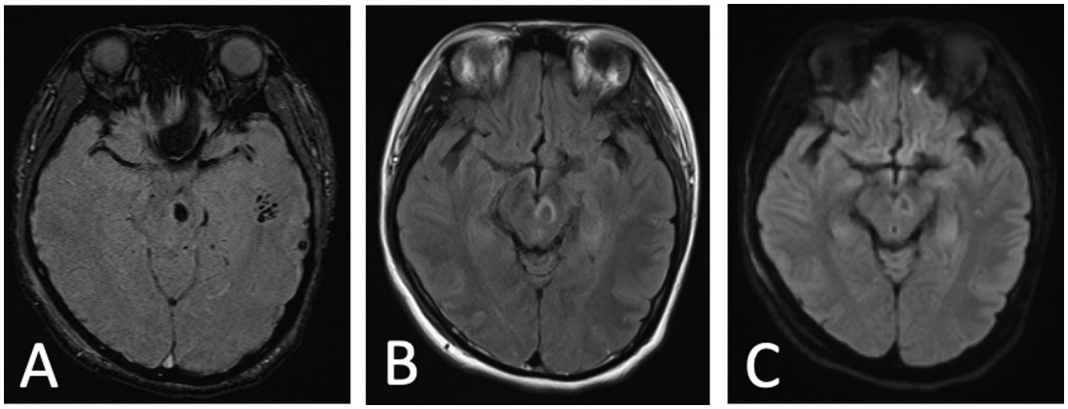

Fig. 1. The MRI imaging studies demonstrate a hemorrhagic lesion in the medial aspect of the midbrain tegmentum on the left side involving the oculomotor fascicles. Multiple microbleeds in the left temporal lobe are also demonstrated in keeping with diffuse axonal injury. A: susceptibility weighted imaging (SWI); B: fluid attenuation inversion recovery (FLAIR) imaging; C: diffusion weighted imaging (DWI).

subdivisions, with the fascicles of each being involved in the control of corresponding ocular muscles. Brainstem lesions were reported to lead to intra-axial divisional oculomotor nerve paresis indicating a topographical arrangement of the CN3 fascicles in the midbrain [1–5]. The clinical presentation of the previously reported cases suggests a fascicular organization into the superior and inferior divisions in human brainstem [1].

A literature search for traumatic oculomotor dysfunction identified a few case reports of CN3 palsy due to one lesion in midbrain following a head injury, and all those cases presented with some degree of ocular motility deficits, either with or without other symptoms of Benedikt's syndrome. Only one case of isolated unilateral mydriasis and ptosis sparing oculomotor muscles was reported, having been caused by a midbrain infarct [2]. By contrast, the patient in the present case report is the first case report that demonstrates traumatic pathogenesis for this rare presentation of partial oculomotor paresis, with an ipsilateral lesion in the tegmentum of midbrain comparable to that seen in the case reported by Chen and colleagues [2].

Ksiazek and colleagues proposed the proximity of fascicles that innervate inferior rectus and pupillary muscles, and proximity of fascicles innervating medial rectus and inferior rectus muscles [3]. Castro and colleagues proposed the lateral to medial organization of oculomotor fascicles in the ventral midbrain tegmentum as follows: inferior oblique, superior rectus, medial rectus, levator palpebrae, inferior rectus and pupillary fibers [4]. Saeki and colleagues proposed a rostral to caudal order of oculomotor fascicles in the midbrain as follows: pupillary fibers, extraocular movement and eyelid elevation, which were analogous to the nuclear arrangement in monkey proposed by Warwick [5]. These models were based on the case reports of neuro-ophthalmological impairment and imaging findings. However, the clinical presentation of this case cannot be completely explained by these models.

4. Conclusion

The patient in this study is the second reported case with isolated mydriasis and ptosis resulted from a single lesion in the ipsilateral tegmentum of midbrain, this site being similar to Chen et al.'s patient with infarction [2]. The uniqueness of this case is the clinical presentation of partial CN3 palsy following a traumatic brain injury. The combination of mydriasis and ptosis sparing eye movement muscles cannot be explained by the aforementioned speculative anatomic models. This case report provides evidence that may shed further light on the intra-axial oculomotor fascicular organization in human brainstem. The future study may be utilizing the MRI-based technique of diffusion tensor imaging and fiber tractography to illustrate the neural tracts and fascicles in the cases of CN3 palsy. This advanced neuroimaging technique would provide insight about the oculomotor fascicular organization in midbrain tegmentum, but is not typically available for the clinical use yet.

Declaration of interest

None.

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Disclosures

Zhihui Deng, John Davis, Flor Muniz-Rodriguez have no conflicts to disclose.

Compliance with ethics guidelines

The authors received consent from the patient included in this case report.

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