CASE REPORT

Recurrent Alternating Oculomotor Nerve Palsy: An Unusual Presentation of Parasagittal Meningioma

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ABSTRACT

We present a previously unreported case of 57-year-old man suffering from diplopia. Motility assessment revealed a total right oculomotor nerve palsy that spontaneously resolved in about 10–18 days. Three years later, sudden oculomotor nerve palsy occurred in his left eye and complete resolution without any treatment was observed after a month. Five years from the second episode there was a further recurrence in the right eye. Magnetic resonance imaging demonstrated a parasagittal meningioma.

Keywords: Magnetic resonance imaging, meningioma, oculomotor nerve palsy, peritumoural oedema

INTRODUCTION

Intracranial tumours can affect the oculomotor nerve by direct and indirect mechanisms. Increased intracranial pressure induced by peritumoural oedema may compress the nerve near the brain stem. Significant brain oedema may cause severe neurological deficits. Meningiomas are generally well-circumscribed, slow-growing tumours that account for 13% to 19% of all brain tumours.¹ Peritumoural oedema is a particular feature of some meningiomas, with an incidence of about 40% to 78%.^{2,3} Written consent to publish this report was obtained from the patient.

CASE REPORT

A 57-year-old man suffering from diplopia presented to our ophthalmology department. The medical history and the laboratory work-up of the patient revealed only diabetes, which was medically controlled, and no diabetic microvascular complication was detected. On ophthalmic examination, motility assessment revealed a right exotropia with limited adduction and elevation and normal

abduction (Figure 1A). The affected eye deviated slightly out and down in primary position. The pupil was mildly dilated and did not react to light. Diplopia and ptosis were also detected. Total oculomotor nerve palsy spontaneously resolved in about 10–18 days (Figure 1B). Three years later, an oculomotor nerve palsy of sudden onset occurred in his left eye (Figure 1C) and the patient demonstrated complete resolution without any treatment after a month (Figure 1D). Five years later, the problem recurred in his right eye (Figure 1E). Complete neurological evaluation revealed no other abnormality. Between episodes, all functions of both oculomotor nerves were normal. Supratentorial magnetic resonance (MR) imaging demonstrated a parasagittal homogeneous and round mass with thin capsule and meningioma was diagnosed. The tumour is 24 × 22 mm in size and arising from the left side of the falx cerebri at the level of the centrum semiovale. Mass effect was noted against the truncus of the corpus callosum (Figure 2).

DISCUSSION

Meningiomas are mostly very slow growing tumours and the majority are asymptomatic through life.¹ MR is

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FIGURE 1. (A) The patient's first attack of non-pupil-sparing third nerve palsy of the right eye. (B) Total oculomotor nerve palsy spontaneously resolved in about 3 weeks after the first attack. (C) The patient's second attack of total third nerve palsy occurred 3 years after the first episode in his left eye. (D) Complete resolution of the second episode was observed after a month. (E) The patient's third attack occurred in the right eye 5 years following the resolution of the second.

the preferred imaging method for the diagnosis and evaluation of meningiomas.¹ In our patient, this lesion with typical imaging features of meningioma has been documented to increase in size slowly over 8 years (The patient first presented to our ophthalmology

clinic in 2003 and the lesion was 24×22 mm in size. The most recent MR scan was performed in 2012 when the tumour was 24×29 mm in size). Following diagnosis by neuroradiologists, the neurosurgeons recommended surgical removal but the patient refused.

FIGURE 1. Continued.

FIGURE 2. T2- (A) and T1- (B and C) weighted MR images revealed parasagittal homogeneous, spherical mass attached to the left side of the falx cerebri at the level of the centrum semiovale (arrow). Mass effect is noted against the truncus of the corpus callosum (arrow).

The oculomotor nerve has a long intracranial course and a very narrow diameter, and as a result this nerve is vulnerable to damage in a variety of intracranial pathologies.⁴ The oculomotor nerves exit the midbrain in close approximation through the medial aspect of the cerebral peduncles. At this location there are no other cranial nerves.⁴ Thus, tumour-related bilateral third cranial nerve palsy

tends to occur following damage at this anatomical location. The third nerve enters the cavernous sinus above the fourth and sixth nerves near the trigeminal and sympathetic nerve fibres. Because of the close proximity of these nerves, intracavernous lesions typically give rise to multiple nerve damage rather than an isolated nerve palsy.⁵ It should also be noted that the oculomotor trigone, which is defined by the

anterior and posterior petroclinoid ligaments,⁶ is also a possible site of damage resulting in isolated third cranial nerve palsies.⁷ Correspondingly, the parasagittal tumour in the present case was in the midline plane such that compression of the truncus of the corpus callosum may have caused local entrapment of cerebrospinal fluid, causing increased intracranial pressure indirectly compressing the trigonal or cisternal segments of the third cranial nerves in our patient.

Ophthalmoplegic migraine is an inflammatory cranial neuropathy characterized by recurrent attacks of headache and total ophthalmoplegia that mostly involves the oculomotor nerve. The total oculomotor nerve palsy takes about 10–18 days to resolve completely. The symptom free interval may be from several months to years.⁸ This clinical appearance seems close to our patient's; however, our patient has never complained of headache over the 8-year period. Furthermore, the cisternal portion of the oculomotor nerves were not thickened and neither did they enhance with paramagnetic contrast in MR studies that were performed during the palsy episodes. Ophthalmoplegic migraine mostly occurs in infancy or early childhood, thus the first attack usually occurs before 5 years of age.⁸ Rarely patients experience their first attack in adulthood, but such patients almost always have a history of migraine headaches with and without aura since childhood, which not the case for our patient. In contrast, meningiomas are noticed most commonly at age of 50 or older.¹ Our patient was 49 years of age when his first palsy episode occurred.

CONCLUSION

We conclude that the meningioma itself or episodic occurrence of meningioma-associated peritumoural oedema or meningioma-associated mediators may trigger an inflammatory reaction combined with

vasculopathy leading to oculomotor neuropathy mimicking ophthalmoplegic migraine. However, the exact pathogenesis remains unknown.

Declaration of interest: The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the paper.

Note: Figure 1 of this article is available in colour online at www.informahealthcare.com/oph

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