CASE REPORT

Oculomotor nerve palsy secondary to aberrant posterior cerebral artery

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SUMMARY

The authors report a rare case of an oculomotor nerve palsy attributed to mass effect from an aberrant posterior cerebral artery. The patient presented with complete ptosis, mydriasis and ophthalmoplegia of the affected eye. MRI demonstrated distortion of the oculomotor nerve as it traversed inferior to the non-aneurysmal P1 segment of an aberrant posterior cerebral artery. The oculomotor nerve palsy resolved spontaneously.

BACKGROUND

Isolated oculomotor nerve palsy (ONP) is a common clinical entity. Pupil-sparing ONP is typically caused by diabetes mellitus and giant cell arteritis, while pupil-involved ONP is most often caused by saccular aneurysms or intracranial neoplasms. The oculomotor nerve originates from multiple subnuclei in the midbrain at the level of the superior colliculus.¹ It exits the midbrain at the medial aspect of the cerebral peduncle, inferior to the posterior cerebral artery (PCA), and superior to the superior cerebellar artery (SCA). The cisternal portion of the oculomotor nerve courses ventrally and laterally, and is found inferior and lateral to the posterior communicating artery (PComMA). Parasympathetic pupilloconstrictor fibres are superficial and situated dorsomedially on the cisternal oculomotor nerve. Owing to the intimate relationship between the posterior intracranial circulation and the oculomotor nerve, vascular anatomical variants can be expected to cause ONP.

The authors report a rare case of ONP secondary to an aberrant, non fetal-type PCA. A systematic review of the literature on posterior intracranial vascular variants as a cause of ONP was performed.

CASE PRESENTATION

This 24-year-old right-handed woman noticed an inability to open her right eye due to a droopy eyelid on after waking up from bed. On manual elevation of the eyelid her partner noted anisocoria, with the previously unremarkable right pupil significantly larger than the left. She also presented with subjective blurring of her vision and a mild, right-sided fronto-orbital headache. She denied any history of preceding trauma or any neurological symptoms in her upper or lower limb. She has been previously well and has no medical history. On examination, the right eyelid was partially ptotic. The right pupil was dilated (5 mm) and had sluggish responses to direct and consensual pupillary light reflexes. The left pupil was 3 mm and reacted normally to light stimulation. Accommodation was

impaired with delayed convergence of the right pupil. Elevation and adduction of the right eye was partially impaired. Visual acuity, visual fields and funduscopy were normal. The remainder of the cranial nerve and neurological examination was normal.

INVESTIGATIONS

Routine full blood counts, electrolyte counts, random serum glucose level and vasculitic panel were normal. A CT of the brain and CT angiogram (CTA) of the intracranial vessels demonstrated an aberrant right-sided PCA with no saccular aneurysms. MRI of the brain and MR angiogram (MRA) performed 6 days after initial presentation confirmed the aberrant right PCA and absence of saccular aneurysms or intraparenchymal lesions (figure 1). On thin-section (1 mm) T2-weighted imaging, the right oculomotor nerve was distorted as it coursed inferior to the P1 segment of the right PCA (figure 2).

OUTCOME AND FOLLOW-UP

The patient's headache was managed with paracetamol. Within 24 h, the patient's ptosis, anisocoria, impaired pupillary light reflexes and ophthalmoplegia completely resolved and she was discharged 2 days postpresentation.

DISCUSSION

This case is unique for the fact that it is extremely rare for ONP to occur secondary to distortion by a PCA of basilar origin, that is, non-fetal-type PCA. After a systematic review of the published literature, eight cases.^{2–8} of isolated ONP secondary to posterior intracranial circulation anatomical variants have been published. In 1973, Hopkins *et al*⁵ described an 81-year-old woman with ONP secondary to a tortuous fetal-type PCA. Since then, three^{7 8} additional cases of ONP secondary to tortuous fetal-type PCA have been described. All four cases were conservatively managed and had spontaneous resolution of symptoms. A tortuous fetal-type PCA, due to its intimate relationship with the cisternal oculomotor nerve, can distort the traversing oculomotor nerve, especially when the PCA is directed in a ventrolateral direction.

As the oculomotor nerve exits the midbrain and enters the interpeduncular cistern, it passes and comes into direct contact with the PCA superiorly and SCA inferiorly. The oculomotor nerve may be susceptible to compression at this point. In 2007, Suzuki *et al*⁶ reported the case of a 78-year-old whose oculomotor nerve was intraoperatively found to be compressed at the point between the

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Figure 1 MR angiography of the Circle of Willis (A) and lateral oblique view of the vertebrobasilar arterial system (B) demonstrating normal course of the left posterior cerebral artery. The P1 segment of the right posterior cerebral artery takes an aberrant course, directed acutely dorsolaterally from its origin at the termination of the basilar artery. ACA, anterior cerebral artery; BA, basilar artery; PCA, posterior cerebral artery (right (R) and left (L)); PCommA, posterior communicating artery; SCA, superior cerebellar artery.

Figure 2 (A–C) T2-weighted, non-contrast MRI demonstrating the proximal cisternal course of the right oculomotor nerve (arrows) and its relationship with the right posterior cerebral artery (arrowhead). The oculomotor nerve is distorted by the right posterior cerebral artery, most clearly seen in (A).

arteriosclerotic PCA and SCA. Microvascular decompression was performed resulting in complete symptom resolution.

The classical description of a ‘pupil-sparing’ versus ‘pupil-involved’ ONP serves to aid the clinician in diagnosing a medical (eg, diabetes mellitus) versus surgical (eg, PCommA aneurysm) cause of ONP, respectively. This line of thought is fraught with potential for misdiagnosis. Multiple cases of pupil-sparing ONP have been reported in patients with PCommA aneurysms.⁹ From our literature review, most (7 of 8) cases involved ‘pupil-involved’ ONP. However, Hashimoto *et al*³ reported a case of pupil-sparing ONP in a 74-year-old woman with superolateral displacement of the oculomotor nerve secondary to a tortuous basilar artery. The authors postulate that compression of the inferomedial segment of the cisternal oculomotor nerve resulted in ophthalmoplegia but not pupillary dysfunction due to sparing of the dorsomedial parasympathetic pupilloconstrictor fibres.

With advances in MRI, the complete cisternal course of the oculomotor nerve can be visualised. As shown in the present case, MRI can allow visualisation of a distorted oculomotor nerve secondary to vascular compression. Albrayam *et al*² reported a 24-year-old woman with MRI evidence of a duplicated SCA and prominent PCA compressing the oculomotor nerve. MRI and MRA are useful and sensitive diagnostic tools that enables vascular anatomical variants and oculomotor nerve distortion to be demonstrated.

In conclusion, anatomical variations in the posterior intracranial circulation are rare causes of isolated ONP. MRI and MRA

are sensitive modalities that should be utilised in the diagnosis of vascular compression causing ONP.

Learning points

- ▶ Rare case of third nerve palsy secondary to aberrant non-fetal type posterior cerebral artery is presented.
- ▶ Posterior intracranial vascular variants are rare causes of third nerve palsy.
- ▶ MRI and MR angiography are useful in demonstrating distortion of the oculomotor nerve.

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Competing interests None.

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