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Oculomotor Nerve Palsy Due to a Slightly Displaced Posterior Communicating Artery, Manifested by Twitching Ptosis: A Case Report and Review of Literature

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ABSTRACT

A 20-year-old woman presented with left eye ptosis without any headache and pupillary dysfunctions. After the radiological examination, the oculomotor nerve compression was detected in the interpeduncular space by the posterior communicating artery (PCoA) with normal size and shape. The patient underwent frontotemporal craniotomy, and during the surgery, the nerve was detached from the PCoA. Immediately after surgery, all symptoms disappeared. Although oculomotor nerve palsy (ONP) owing to internal carotid-PCoA aneurysm is common, vascular compression due to a non-aneurysmal PCoA is very rare. To the extent of our knowledge, this is the first case in which a slightly displaced, otherwise normal, PCoA causes ONP without any pupillary involvement. After ruling out an aneurysmal artery, this should be considered as one of the possible causes of the ONP.

Introduction

Third cranial nerve palsy appears with different constitutional, neurological, and more frequently, ocular symptoms like diplopia, ptosis and mydriasis. [1]

Third nerve compression, heralded by oculomotor nerve palsy (ONP), is usually a sign of aneurysmal vessels in the cranium on the way of the oculomotor nerve, compressing the nerve. Nevertheless, non-aneurysmal vascular reasons for compression are rarely reported in the literature. [2, 3]

The oculomotor nerve exits the midbrain at the level of the cerebral peduncle, and after moving between the posterior cerebral artery (PCA) and the superior cerebellar artery (SCA), it is placed laterally to the

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posterior communication artery (PCoA). Due to this anatomy, changes in posterior intracranial circulation may lead to ONP. [4, 5] PCoA aneurysm is a prevalent cause of oculomotor nerve dysfunction, whereas non-aneurysmal compression of the third cranial nerve by PCoA is very rare. [6]

In the current study, we are going to report an extremely rare case presented with eye ptosis as a result of ONP, which is caused by mild aberrant PCoA compressing the nerve, treated utterly after surgery. Although aberrant PCoA is documented in the literature, the aberrancy was concurrent with other problems like an aneurysm [2]. However, according to the available literature, this is the first case in which a slightly displaced PCoA, which is not associated with other vascular problems, has caused the compression of the oculomotor nerve without the involvement of parasympathetic fibers.

Case presentation

A 20-year-old female patient, otherwise healthy, presented to the neurology clinic in November 2019 with two months progressively established left eye twitching ptosis. No headache and double vision were observed, and her symptoms did not get worse during the day. There was no change with the heat and cold. On the physical examination, the pupil size was normal, and there was no ophthalmoplegia and gaze abnormality. No deviation of the eye was observed. Both lower eyelid movement and eye closure force were within normal limits. The patient then underwent a thorough investigation with brain magnetic resonance imaging (MRI) using FIESTA (fast imaging employing steady state-acquisition) sequence followed by MR angiography (MRA) for ruling out the abnormalities putting the third cranial nerve under compression. The right posterior communicating artery was normal in shape and size, with a typical trajectory towards the right cavernous sinus (Fig. 1). The right third cranial nerve was somewhat compressed in the middle of its course by PCoA superiorly, caused by a mild displacement in its ordinary course (Fig. 2).

Considering these medical findings, the patient underwent a right frontotemporal craniotomy—the carotid artery exposed via the Transylvanian approach using the microdissection of the arachnoid. Once the supraclinoid carotid artery exposed, its branches identified. The PCoA was normal in size and shape without atherosclerotic changes.

The superomedial aspect of the oculomotor nerve was adherent to the artery with a thick arachnoid band. With taking care of all perforators under meticulous inspection, the entire artery was separated from the nerve and released from arachnoid adhesions by using the sharp microdissection technique. After careful

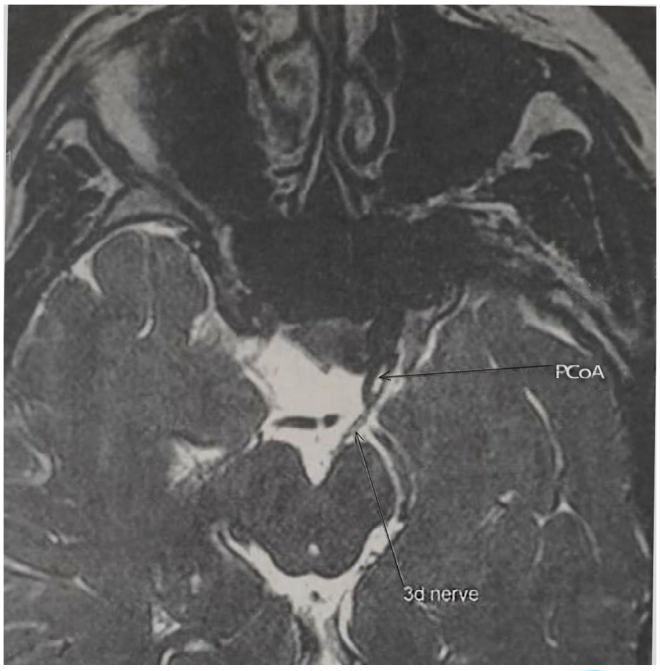

Fig. 1. Oculomotor nerve trajectory. MRI FIESTA sequence showing normal size and shape of the nerve at the site in which the oculomotor nerve exits from the midbrain and also a normal pathway of the nerve inside the interpeduncular space toward cavernous sinus. 3d nerve, third cranial nerve; PCoA, posterior communicating artery

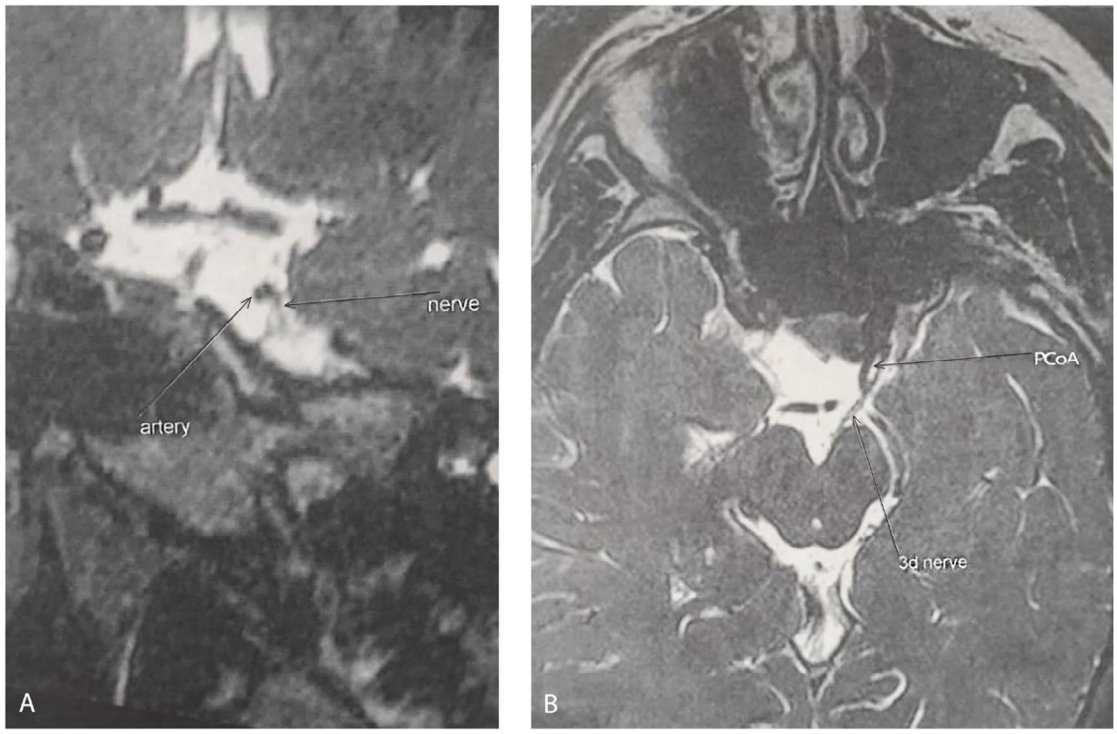

Fig. 2. Oculomotor nerve compression. MRI image shows little aberration of PCoA inside the interpeduncular space (A), causing compression of the oculomotor nerve (B). 3d nerve, third cranial nerve; PCoA, posterior communicating artery

hemostasis, the dura mater repaired using 5-0 prolene watertight sutures. The bone flap was replaced and the wound closed in anatomical layers. The patient tolerated the surgery, and immediately after surgery, all patient's symptoms and signs resolved and were maintained until the last follow-up visit, three months after decompression surgery.

Discussion

As the oculomotor nerve exits the midbrain, it enters the interpeduncular cistern and continues between PCA and SCA, then passes laterally to the posterior communicating artery. This proximity of the oculomotor nerve to vessels leads to nerve dysfunction because of possible changes in vascular anatomy [7] Vascular compression of the oculomotor nerve due to aneurysm is a prevalent cause of third cranial nerve compression. [8] Among aneurysmal causes of CNIII compression, an aneurysm at the junction of PCoA and internal carotid artery (ICA), which is called ICA-PCoA aneurysm, in most cases, is the leading cause. [9] CNIII compression by the aneurysm of other arteries like PCA, SCA, Basilar artery, and anterior communicating artery has been reported in very rare cases. [9, 10]

Moreover, the compression of the oculomotor nerve can be caused by non-aneurysmal dilation of vessels through subarachnoid space. Ectatic vessels can compress the oculomotor nerve and put it under pressure [8]. Infundibular dilation of PCoA as a cause of compression has been reported in a few cases. In one case, the patient had presented with sudden left oculomotor nerve paresis with a history of chronic headaches. ICA aneurysm and dilated PCoA were detected by cerebral angiography. Frontotemporal craniotomy was operated, and infundibular PCoA was separated from CNIII; paresis and headaches disappeared after a while. [11] In another case, ONP was resolved by clipping the dilated PCoA(6); also, the resolution of ONP by microvascular decompression (MVD) is reported in the literature. [3, 12, 13] Although ONP due to subarachnoid hemorrhage (SAH) usually is caused by the ruptured aneurysm, infundibular PCoA also can change to an aneurysm and cause hemorrhage. [7, 13]

The non-vascular origin of ONP includes reasons such as trauma, diabetes mellitus, tumors, infarction, infections, thrombosis, cavernous hemangioma and demyelination. [1, 7] The parasympathetic part of the oculomotor nerve with smaller fibers starts from Edinger–Westphal nucleus in the midbrain, then passes on the top of the oculomotor nerve and innervates pupillary constrictors and ciliary

Table 1. Cases with Non-Aneurysmal Neurovascular Compression of CNIII

study	Patient/s	clinical manifestations	imaging and diagnosing	cause of non-aneurysmal vascular nerve palsy	Intervention/surgery	results of the intervention
Tan et al., 2014(5)	24-year-old woman	sudden droopy eyelid, anisocoria, and a little headache	CTA, MRI, and MRA	right aberrant PCA without aneurysm	no intervention	complication resolved spontaneously
Cruz et al., 2013(14)	75-year-old woman	eyes were not moving together with later diplopia and anisocoria	MRI and MRA	aberrant PCoA with no mention of other problems	no surgery, carbamazepine	diplopia resolved by carbamazepine, but partial third nerve paresis remained
Demetriou & Bell, 2014(16)	86-year-old female	left-sided retro-orbital pain, ptosis, and diplopia	CT, MRI, MRA	occlusion of the right internal carotid artery and tortuous left internal carotid artery	no surgery and endovascular procedure	N/A
Fukami et al., 2018(13)	70-year-old woman	pain in left forehead, left ptosis, and mild anisocoria	MRI, MRA	infundibular dilation of PCoA	surgery without endovascular treatment	anisocoria and ptosis resolved
Israni et al., 2016(17)	7-year-old boy	ptosis limited upward movement of the right eye	MRI and MRA	tortuous and ectatic distal internal carotid artery	N/A	N/A
Joshi et al., 2017(18)	62-year-old male	diplopia, partial ptosis, and anisocoria	CT, CTA, and MRI	duplicated superior cerebral artery	N/A	N/A
Fukushima et al., 2014(6)	60-year-old woman	left mild ptosis	MRA	infundibular dilation of PCoA	surgery (clipping)	ptosis improved within one month
Kheshaifati et al., 2016(19)	16-year-old male	transient right ptosis, diplopia, ophthalmoplegia, and moderate mydriasis	cerebral angiogram, MRI	compression by trifurcation of PCoA P2 segment	surgery	palsy improved over three months
Mulderink et al., 2001(11)	69-year-old man	sudden onset of third cranial nerve paresis with a history of chronic headaches	MRI and angiography	infundibular PCoA in direct contact with CNIII	surgery	symptoms improved, and headaches disappeared
Silva et al., 2010 (8)	56-year-old man	bilateral ophthalmoplegia and partial ptosis with no pupillary abnormality	CT, CTA, and MRI	right conflict: compression by right PCA and left conflict: left SCA	N/A	N/A
Haider et al., 2019(2)	76-year-old woman	left-sided incomplete ptosis	MRI, MRA, and DSA	tortuous aneurysmal PCoA	surgery (MVD)	ONP completely resolved
Hashimoto et al., 1998 (20)	74-year-old woman	diplopia without ptosis	MRI	tortuous basilar artery	N/A	N/A
Babbitz & Harsh, 2005(3)	36-year-old man	headache with ptosis	MRI and MRA	compression between ecstatic, atherosclerotic PCoA and small tentorial meningioma	surgery (MVD)	patient's headache and oculomotor paresis disappeared

CTA, computed tomography angiogram; MRI, magnetic resonance imaging; MRA, magnetic resonance angiogram; PCA, posterior cerebral artery; PCoA, posterior communicating artery; CT, computed tomography; N/A not available; CNIII, third cranial nerve; DSA, digital subtraction angiography; MVD, microvascular decompression; ONP, oculomotor nerve palsy

muscles. Mass lesions compressing CNIII are usually accompanied by pupillary dysfunctions. [1, 6] In our case, while the ONP was due to vascular compression, the patient did not have any pupillary dysfunctions like mydriasis and anisocoria.

Non-aneurysmal vascular compressions are very uncommon and reported in a small number of cases (Table 1). [8] To the best of our knowledge, our case is the first case in which the ONP is caused as a result of direct compression of CNII by a mild aberrant PCoA without any lesions and other problems of the PCoA.

A case of an aberrant vascular source of ONP has been reported by Tan et al. [5]. They have reported a patient presented with ptosis, anisocoria and ophthalmoplegia. The patient underwent imaging for diagnosis. Aneurysm was ruled out by computed tomography angiogram (CTA), and aberrant right-sided PCA was observed. MRA confirmed the aberrant PCA as the cause of the compression. ONP resolved spontaneously.

Haider et al. [2] reported that a female patient presented with left eye partial ptosis without any pupil dysfunction. Based on the MRI and MRA images, a PCoA aneurysm was diagnosed as the probable cause of the compression. The patient underwent craniotomy, and intraoperatively, tortuous PCoA was considered as a more likely origin of compression. Symptoms disappeared shortly after decompression. Although, in this case, the compression was mostly attributed to the aberrant PCoA, it was different from our case. In our case, the aberrant PCoA loop entirely caused ONP by putting pressure directly on the oculomotor nerve.

In another similar case [14], the authors described a female patient with right eye ptosis, subsequently developed diplopia, prolonged left gaze and anisocoria. MRI and MRA imaging revealed a displaced PCoA had put the oculomotor nerve under pressure. In this case, ONP was caused by the displacement of the posterior communicating artery compressing the oculomotor nerve with the involvement of the parasympathetic fibers led to pupil dysfunctions. This case was different from our case because a completely displaced artery caused the compression, and also pupil was involved. In our patient, PCoA did not have any anomalies, and its anatomy almost seemed regular. The only small direct contact with the oculomotor nerve was detected as the cause of compression, and no parasympathetic fibers,thereby; no pupil dysfunctions were involved.

Although dilated vessels because of an infundibular, aneurysmal, or fetal artery are the primary cause of compression of the oculomotor nerve inside the subarachnoid space [11, 15], compression of the oculomotor nerve because of a displaced tortuous artery or a partially displaced normal artery should be taken into account.

Conclusion

Oculomotor nerve compression because of an aneurysmal artery, especially PCoA aneurysm inside the subarachnoid space, is a common etiology of ONP. Furthermore, non-aneurysmal vascular compression of the oculomotor nerve should be taken into account as a possible cause that may have put pressure on the nerve. Herein, we presented a rare case in which an almost normal PCoA, with a little aberrancy, caused pressure on the oculomotor nerve, which was ultimately resolved after surgery. Of note, this case highlights the point that after ruling out aneurysmal etiologies of compression, compression of the oculomotor nerve due to aberrant arteries should be considered.

Ethical Considerations

Consent

The consent was taken from the patient for the case report to be published.

Compliance with ethical guidelines

All ethical principles are considered in this article. The patient was informed about the purpose of the research and its implementation stages.

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Authors' contributions

All authors equally contributed in preparing this article.

Conflict of interest

The authors declare that they have no conflict of interest.

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