



## Case Report

# Microvascular decompression of the posterior cerebral artery for treatment of oculomotor nerve palsy

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**J Cerebrovasc Endovasc Neurosurg.**  
**2020 June;22(2):85-89**

Received: 12 June 2015

Revised: 27 March 2016

Accepted: 14 June 2016

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\* This work was previously presented  
as a poster abstract at the American  
Association of Neurological Sur-  
geons 84<sup>th</sup> Annual Scientific Meet-  
ing, April 30-May 4, 2016; Chicago,  
IL, USA

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Oculomotor nerve palsy resulting from non-aneurysmal vascular compression is extremely rare. Microvascular decompression (MVD) has been previously shown to improve oculomotor nerve palsy (ONP) secondary to arterial compression. A 71-year-old female, with a history of Cushing's disease previously treated with two transsphenoidal resections and Gamma Knife radiosurgery, presented with one year of progressive left eye diplopia and was diagnosed with a partial left oculomotor nerve palsy. We performed an orbitozygomatic craniotomy for MVD of the left posterior cerebral artery, which was found to be compressing the oculomotor nerve against the tentorium. Unfortunately, the patient's partial ONP remained unchanged at one year follow-up. The present case suggests inconsistent outcomes of MVD for ONP. Patients with prior sellar or parasellar irradiation may be less likely to benefit from this treatment approach.

**Keywords** Microsurgery, Microvascular decompression, Oculomotor nerve, Posterior cerebral artery, Skull base

## INTRODUCTION

Oculomotor nerve palsy (ONP) secondary to non-aneurysmal vascular compression is extremely rare.<sup>1-3)9-11)13)14)19)</sup> Microvascular decompression (MVD) has been previously demonstrated to improve ONP secondary to arterial compression, although in a very limited number of cases.<sup>18)</sup> The aim of this case report is to describe the technical aspects of MVD of the posterior cerebral artery (PCA) for the treatment of ONP.

## CASE REPORT

A 71-year-old female with a history of Cushing's disease, previously treated with two transsphenoidal resections and subsequent Gamma Knife radiosurgery (GKRS), presented with one year of progressive diplopia. Neuro-ophthalmologic examination revealed limitations in elevation, adduction, and depression as well as ptosis, anisocoria and lid retraction on attempted downgaze of her left eye. The patient was diagnosed with a partial left ONP. The possibility of a delayed cranial neuropathy from GKRS of pituitary adenoma was considered as a possible etiology of ONP. However, analysis of the GKRS treatment plan noted that the left lateral cavernous sinus wall was outside of the radiation field. Magnetic resonance imaging (MRI), 3D-sampling perfection with application optimized contrasts using different flip-angle evolution (SPACE) sequence, demonstrated compression of the left oculomotor nerve between the ipsilateral PCA and superior cerebellar artery (SCA) at the P1-P2 junction (Fig. 1A). In order to prevent further progression of her ONP and in an attempt to facilitate recovery, we elected to proceed with MVD of the left PCA.

We performed a left-sided modified orbitozygomatic craniotomy for an approach to the anterior cranial fossa. The Sylvian fissure was widely split from a distal to proximal approach. After proceeding down the subfrontal corridor, the optic nerve was identified and overlying arachnoid was sharply opened and subsequently connected to the proximal Sylvian fissure. The opticocarotid cistern was opened and cerebrospinal fluid was drained to aid in brain relaxation. The carotid bifurcation was identified, as well as the proximal A1 and M1 segments. The temporal lobe was then retracted posteriorly to expose the tentorial incisura along with the inferior surface of the left supraclinoid internal carotid artery (ICA). The infundibulum of the left posterior communicating artery (PCOM) was identified, as well as the anteriorly displaced loop of the left PCA. This was lifted up, and the oculomotor nerve appeared to be significantly flattened by the pulsations of the left PCA against the tentorial edge (Fig. 1B). Further whitish

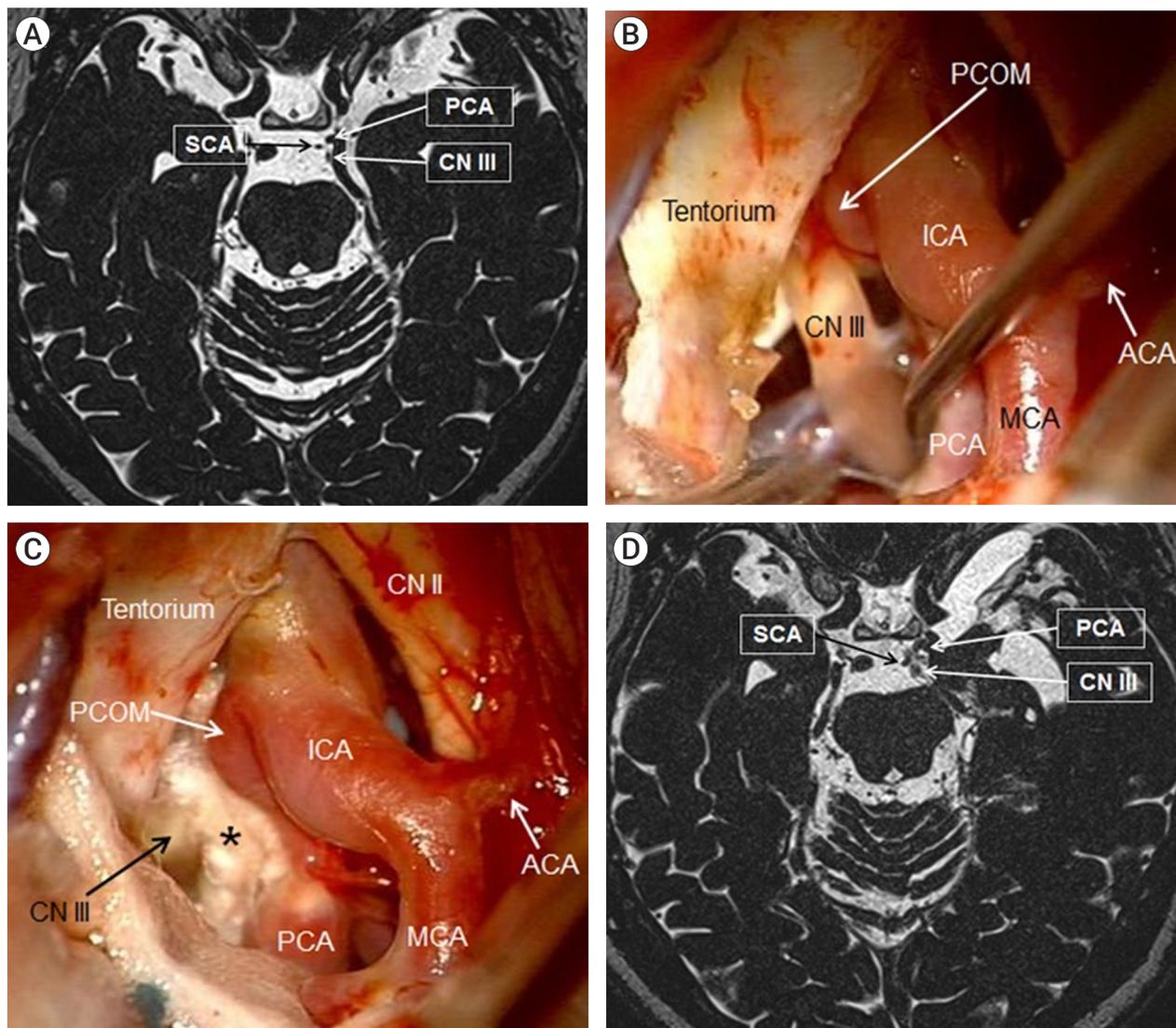
discoloration of the nerve was seen at the distal edge of the compression. The left SCA was visualized below the oculomotor nerve, and the arachnoid bands surrounding this complex were cut and relaxed.

We explored the possibility of ligating the PCOM at its origin from the ICA in order to mobilize the PCA posteriorly. However, we noted too many thalamoperforators arising from the proximal segment of the PCOM to safely perform this maneuver. Instead, cottonoid buffers were placed between the left oculomotor nerve and PCA, which appeared to adequately decompress the nerve (Fig. 1C).

The patient had an uncomplicated postoperative course. Immediately after surgery, the patient had a complete left ONP, which subsequently improved, after a short course of steroids, to her baseline function by the time of discharge on postoperative day two. Postoperative 3D-SPACE MRI showed a small cerebrospinal fluid space between the left oculomotor nerve and the ipsilateral PCA and SCA (Fig. 1D). At one year neuro-ophthalmologic follow-up, the patient's oculomotor nerve function had not significantly improved from her preoperative baseline.

## DISCUSSION

The present case suggest that the patient's ONP primarily resulted from vascular compression by the ipsilateral PCA, and that outcomes following MVD for the treatment of ONP may be more variable than previously recognized. There are few previously reported cases of ONP resulting from non-aneurysmal vascular compression.<sup>1-3)9-11)13)14)19)</sup> Of these reports, only four cases involved compressive effects by the PCA (Table 1).<sup>10)13)14)18)</sup> Two of these cases described acute oculomotor paresis involving compression by the P1 segment that were not treated with surgical intervention; one was a postmortem diagnosis and the other involved a perimesencephalic vascular anomaly with partial ONP.<sup>10)13)</sup> Suzuki et al. reported a case of left oculomotor nerve palsy secondary to compression between an arteriosclerotic



**Fig. 1.** (A) Preoperative brain MRI, axial 3D-sampling perfection with application optimized contrasts using different flip-angle evolution (SPACE) sequence, shows compression of the left CN III between the PCA and SCA at the P1-P2 junction. Beyond this point of compression, the nerve is decreased in volume (not shown). (B) Intraoperative photograph, from a transsylvian approach through a left-sided modified orbitozygomatic craniotomy, shows compression of CN III, which appears flattened, by the PCA (elevated by an instrument) against the tentorium. (C) Intraoperative photograph after placement of a cottonoid buffer (asterisk) between CN III and the PCA. (D) Postoperative MRI, axial 3D-SPACE sequence, shows a small cerebrospinal fluid space between CN III and the left PCA and SCA. MRI, magnetic resonance imaging; PCA, posterior cerebral artery; SCA, superior cerebellar artery; PCOM, posterior communicating artery; ICA, internal carotid artery; MCA, middle cerebral artery; ACA, anterior cerebral artery.

PCA and SCA.<sup>18)</sup> A similar case was reported earlier by Nakagawa et al., in which a patient experienced relief with MVD of the left PCA but subsequently developed similar ONP on the contralateral side due to postsurgical arachnoid adhesions at the tentorial edge.<sup>14)</sup> In both cases, ONP caused directly by arterial compression was more reliably detected intraoperatively than with

preoperative MRI, and MVD was associated with near-complete resolution of the cranial neuropathy within three months. Non-aneurysmal arterial compression has also been reported in a case of an enlarged, atherosclerotic loop of the PCOM in a patient with unilateral subacute ONP and retro-orbital headaches in the setting of a small, non-distorting meningioma of the left tentorial

**Table 1.** Summary of case reports of non-aneurysmal compression of the oculomotor nerve by the PCA

Series (year)	Patient age/gender	Laterality and degree of ONP	Vascular anomaly in addition to PCA compression	Treatment	Follow-up duration	Outcome
Current study	71 / F	Left, partial	None	MVD (left)	12 months	No improvement
Suzuki et al. (2008) <sup>18)</sup>	76 / M	Left, complete	None	MVD (left)	1 month	Improved
Nakagawa et al. (1991) <sup>14)</sup>	59 / M	Bilateral (first on left and then two months later on right)	Duplicated PcomA, P1 infundibulum	MVD (first on left and then on right)	3 months	Improved ocular movement and ptosis at one month; improved adduction at two months; improved elevation at three months
Morimoto et al. (1985) <sup>13)</sup>	61 / F	Left, complete	None	Postmortem diagnosis	N/A	Patient developed acute ONP after angiogram complicated by left MCA infarct and died one month later
Imes et al. (1984) <sup>10)</sup>	31 / M	Left, partial	Small perimesencephalic vascular anomaly	No surgical intervention	N/A	No improvement

ONP, oculomotor nerve palsy; PCA, posterior cerebral artery; MVD, microvascular decompression; PcomA, posterior communicating artery; MCA, middle cerebral artery

incisura.<sup>2)</sup>

The natural history of ONP secondary to non-aneurysmal vascular compression is unknown. Previous studies of surgical outcomes of aneurysmal oculomotor nerve compression may serve as a proxy.<sup>2,12)</sup> A review of 319 cases of isolated unilateral oculomotor palsy (70% complete, 30% partial) caused by intracranial aneurysms treated with surgical decompression proposed a correlation between treatment and symptomatic resolution: 64% of patients experienced complete recovery of oculomotor nerve function when surgery was performed within two weeks of symptom onset, 30% when surgery was performed within 14-30 days, and 14% when surgery was performed after one month. Overall, 41% of patients experienced full resolution of symptoms.<sup>12)</sup>

Other studies have proposed that if patients experience complete oculomotor nerve recovery, then this will typically occur within three months of surgery.<sup>7,8,15)</sup>

Our case describes a patient who underwent MVD nearly one year after the onset of ONP. At one year postoperative follow-up, the patient's oculomotor nerve function had not significantly improved from her preoperative baseline. This outcome suggests an inconsistent efficacy of MVD for ONP. While prior reports did

not stratify the timing of postoperative recovery, their findings suggest that longer intervals between diagnosis and surgical management are associated with a lower likelihood of functional recovery. Delayed decompression in the present case may have contributed to lack of symptomatic improvement. Furthermore, the patient underwent prior GKRS for a pituitary adenoma, which may suggest that those who have undergone prior sellar or parasellar irradiation are less likely to benefit from MVD for the treatment of ONP.<sup>4-6,16,17)</sup> Therefore, our case differs from prior reports of successful MVD for ONP in two aspects: (1) the relatively longer time interval between diagnosis and surgery, and (2) a history of prior sellar irradiation.

## CONCLUSIONS

ONP is rarely caused by non-aneurysmal vascular compression. Previous reports suggest that clinical improvement can be achieved after treatment with MVD, particularly when performed early after symptom onset. The present case suggests inconsistent postoperative outcomes from MVD for ONP. Patients who have re-

ceived prior sellar or parasellar irradiation may be less likely to benefit from surgical intervention. Until additional data from larger patient cohorts becomes available, the application of MVD should not be generalized to all cases of ONP secondary to non-aneurysmal vascular compression.

## Disclosure

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

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