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Case Report

Unruptured internal carotid-posterior communicating artery aneurysm splitting the oculomotor nerve: A case report and literature review

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ABSTRACT

Background: Although it is well known that internal carotid-posterior communicating artery (ICA-PcomA) aneurysms compress the oculomotor nerve and cause nerve palsy, cases of ICA-PcomA aneurysms splitting the oculomotor nerve are extremely rare.

Case Description: We present the rare case of an asymptomatic, growing, left-sided ICA-PcomA aneurysm that was confirmed to split the oculomotor nerve. We report the clinical course and discuss the underlying mechanism. The oculomotor nerve, which is an aggregate of multiple fibers, exhibits age-related loss of compactness in the arrangement of its nerve fibers.

Conclusion: We speculate that injury to the nerve fibers by aneurysmal compression was avoided because of the rare phenomenon of splitting of the oculomotor nerve.

Keywords: Clipping, Internal carotid-posterior communicating artery aneurysm, Nerve fibers, Oculomotor nerve, Splitting

INTRODUCTION

It is well known that internal carotid-posterior communicating artery (ICA-PcomA) aneurysms compress the oculomotor nerve and cause nerve palsy, which is one of the important clinical signs of an ICA-PcomA aneurysm.[6] However, splitting of the oculomotor nerve by an ICA-PcomA aneurysm is an extremely rare phenomenon. We present a rare case of an unruptured ICA-PcomA aneurysm that splits the oculomotor nerve. The patient did not present with any preoperative symptoms of oculomotor nerve palsy. Herein, we report the clinical course and discuss the mechanism underlying this phenomenon.

CASE REPORT

The patient was a 78-year-old woman who first presented with a mild decrease in cognitive function at the age of 75 years. She did not exhibit signs of neurological deficits such as anisocoria or diplopia. However, magnetic resonance imaging revealed a left-sided ICA-PcomA

aneurysm. Three-dimensional computed tomography (CT) angiography revealed an irregularly shaped aneurysm measuring 7 mm [Figure 1a]. We limited treatment to follow-up observations of the aneurysm in the outpatient department without surgery, considering her age and deteriorated cognitive function. She had not experienced any episodes of oculomotor nerve palsy or sudden thunderclap headaches. The aneurysm gradually grew to 10 mm in size by the time she was 78 years old [Figure 1b]. We planned to clip the neck of the aneurysm to firmly preserve the fetal-type PcomA, which seemed to arise from the aneurysmal body, due to an increased risk of rupture [Figure 1c]. We performed a pterional craniotomy and approached the aneurysm through the transsylvian route. The aneurysm had adhered strongly to the oculomotor nerve and surrounding tissue. We clipped the neck of the aneurysm [Figure 2a and b] followed by careful separation of the aneurysmal wall and inspection of the oculomotor nerve. We observed that the dome of the aneurysm was splitting the oculomotor nerve [Figure 2c and d] (surgical video). Temporary anisocoria and diplopia were observed postoperatively. These oculomotor nerve palsy symptoms improved in approximately 10 days. The patient was discharged with a Modified Rankin Scale score of 1.

The patient provided written informed consent for the publication of this report.

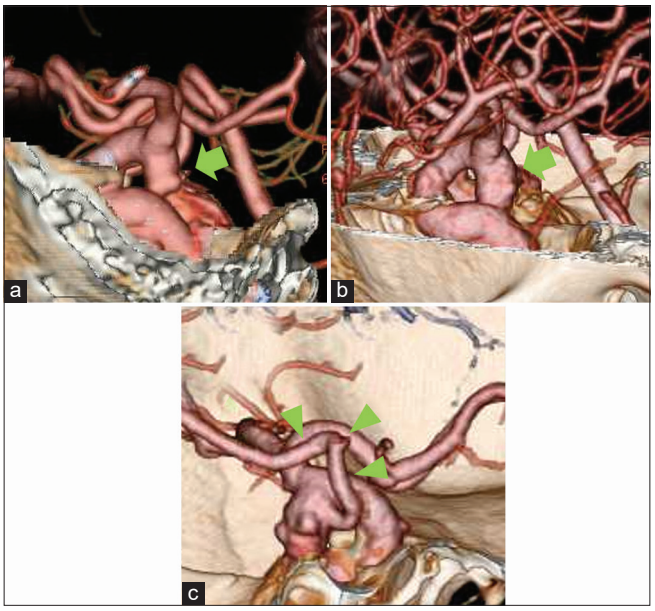

Figure 1: (a) Three-dimensional CT angiography showing the aneurysm (7 mm, arrow) when the patient was 75 years old. The aneurysm originated from the distal side of the bifurcation of the left internal carotid artery and posterior communicating artery. (b) The aneurysm enlarged to 10 mm at the age of 78 years (arrow). (c) CT showing the posterior communicating artery appearing to arise from the aneurysmal body (arrow heads)

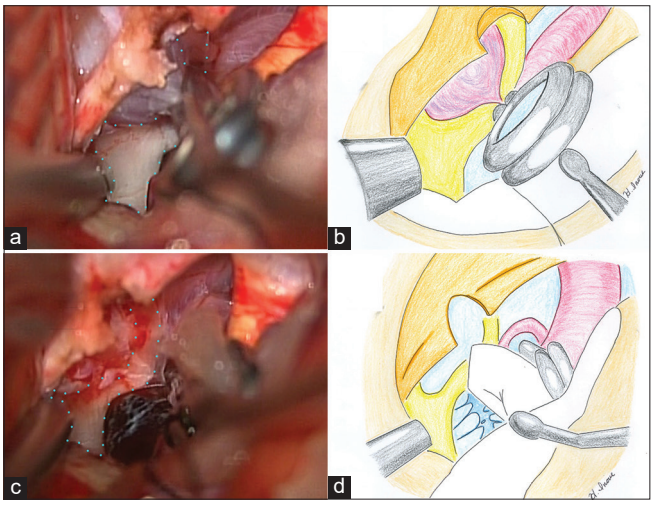

Figure 2: (a) Intraoperative photograph of the left pterional approach. Splitting of the oculomotor nerve by the aneurysm (dotted line). (b) Schematic illustration depicting the aneurysm splitting the oculomotor nerve. (c) The aneurysm is resected. (d) Schematic illustration of the resected aneurysm.

DISCUSSION

Oculomotor nerve palsy is a well-known and important clinical sign of growing ICA-PcomA aneurysms. Thinning of the oculomotor nerve due to aneurysmal compression is often observed during direct clipping. The oculomotor nerve is susceptible to compression by the ICA-PcomA just before it enters the cavernous sinus, which has low mobility.[8] The aneurysm can come into contact with the nerve if its size exceeds 4–5 mm, leading to compression and palsy.[5] However, splitting of the oculomotor nerve by an ICA-PcomA aneurysm is an extremely rare phenomenon. Only five such cases have been reported till date.[2,3,6-9] The clinical information of these cases is presented in Table 1. Four of the reported aneurysms were larger than 5 mm (6 mm, 6 mm, 7 mm, and 10 mm), similar to the present case, except for one case where the aneurysmal size was not specified. Interestingly, four of these six patients did not present with any preoperative symptoms of oculomotor nerve palsy. These studies attributed the absence of palsy to the gradual enlargement of the aneurysm, which retained the functional integrity of the nerve.[6] Although several studies have reported on ICA-PcomA aneurysms, the cause of the rarity of oculomotor nerve splitting by ICA-PcomA aneurysms is unknown. Furthermore, the absence of oculomotor nerve palsy in two-thirds of these cases adds to the conundrum. Cahill *et al*. confirmed that the proximal part of the oculomotor nerve was frequently supplied by the thalamoperforating artery and penetrated by the branches of brainstem vessels in patients without oculomotor palsy.[1] The oculomotor nerve innervates four extraocular muscles

Table 1: Clinical characteristics of patients with oculomotor nerves split by ICA-PcomA aneurysms reported in the literature and present study.

Author (years)	Age, sex	Rupture	Size of aneurysm	Oculomotor palsy	
				Preoperative	Postoperative
Yasargil[9] (1984)	NR	NR	NR	None	NR
Horiuchi *et al*.[2] (1997)	48, F	+, 10 years previously	6 mm	Partial	Complete
Maekawa *et al*.[3] (2010)	71, F	-	7 mm	None	None
Toyota *et al*.[8] (2014)	42, M	-	10 mm	Complete	Improved
Sasaki *et al*.[6] (2018)	71, F	+, ASDH	6 mm	None	None
Inoue (2020)	78, F	-	10 mm	None	Partial (transient)

ICA-PcomA: Internal carotid-posterior communicating artery, PCA: Posterior communicating artery, ICA: Internal carotid artery, ASDH: Acute subdural hematoma, M: Male, F: Female, NR: Not reported

and the levator palpebrae superioris muscle. It consists of preganglionic parasympathetic fibers and is an aggregate of multiple fibers.[4] Sharma *et al*. investigated the cross-sections of the precavernous portion of the oculomotor nerve in cadavers. They found that this segment of the oculomotor nerve was sheathed by thin connective tissue and that the fibers were arranged compactly in younger age groups, while a loss of compactness in fiber arrangement was observed in older age groups.[7] Therefore, we propose the following hypothesis to explain the mechanism by which the aneurysm could have split the oculomotor nerve asymptomatically in this case: the roughly arranged fibers were split by aneurysmal compression, thereby avoiding nerve injury. Further investigation is necessary to elucidate the pathophysiology of aneurysms splitting the oculomotor nerve, considering that only five cases have been reported to date.

CONCLUSION

We reported the unique case of an unruptured left-sided ICA-PcomA aneurysm splitting the oculomotor nerve. Oculomotor palsy was not observed preoperatively in this patient. We speculated that nerve fiber injury by aneurysmal compression may have been avoided, because the oculomotor nerve was split (before the aneurysm could have caused compressive injury).

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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