Asymptomatic Penetration of the Oculomotor Nerve by a De Novo Aneurysm Associated with Severe Atherosclerotic Stenosis of the Supraclinoid Internal Carotid Artery

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INTRODUCTION

Cranial nerve splitting and penetration by an intracranial aneurysm has been described in the literature, often with optic apparatus involvement by the ophthalmic artery or anterior communicating artery (AComA) aneurysm. Penetration of the oculomotor nerve by an intracranial aneurysm is rare and has been reported in only 3 cases elsewhere. De novo aneurysm refers the development of a new aneurysm in a location previously observed to be normal by an angiography or direct microsurgical exploration. The main pathophysiology of a de novo aneurysm involves changes in the vascular connective tissue resulting from the presence of hemodynamic stress or change after surgery, and rarely, the formation of a defect in the arterial wall associated with atherosclerotic degeneration could be a causative factor. Herein, we present an unusual case of penetration of the oculomotor nerve due to evolution of a de novo aneurysm associated with atherosclerotic stenosis of the internal carotid artery (ICA) at the supraclinoid portion.

CASE REPORT

A 70-year-old female presented with a relapsing headache and recurrent right-sided clumsiness related to intracranial atherosclerosis and hypertension. Stroke symptoms have been bothering her since 5 years prior, and she was medicated with a dual antiplatelet and antihypertensive regimen indefinitely.

During five years, the patient has been treated for cerebral infarction associated with severe atherosclerotic stenosis of the internal carotid artery. Three-year follow-up magnetic resonance angiography showed a tiny de novo aneurysm arising from the distal part of atherosclerotic internal carotid artery. And 5-year follow-up three-dimensional CT angiogram demonstrated a definite aneurysm enlargement as large as requiring treatment.

During dissection of aneurysm, the oculomotor nerve was found to be penetrated by the growing de novo aneurysm. The authors report a case of a de novo aneurysm, which resulted from atherosclerotic stenosis of the internal carotid artery at the supraclinoid portion, that was found to be penetrating the oculomotor nerve with no ocular palsy.

Key Words: Aneurysm · Atherosclerosis · De novo · Oculomotor nerve · Penetration.
of the ICA and was observed without treatment mainly due to its small size. However, a two-year follow-up three-dimensional (3D) CT angiogram showed a definite aneurysm enlargement, and thus, a transfemoral cerebral angiogram was strongly recommended to the patient. Digital subtraction angiogram (Fig. 1C) and reconstruction of 3D rotational angiogram demonstrated a 6 mm-sized, elongated aneurysm arising from the posterior communicating segment of the atherosclerotic ICA. Surgical intervention, rather than endovascular treatment, was chosen for treatment. A pterional craniotomy was performed, and the aneurysm was found and finally exposed after partial removal of the anterior clinoid process due to the low-lying nature of the aneurysm and tortuousness of the supraclinoid ICA. During neck clipping, the aneurysm was observed with the dome projecting posterolaterally into and penetrating the oculomotor nerve, and neither posterior communicating artery (PCOMA) nor its remnant was confirmed. After careful neck dissection, the aneurysm clip was placed. The dome of the aneurysm was embedded within the oculomotor nerve (Fig. 1E) but left untouched to prevent any unnecessary injury. The patient tolerated clipping well and was discharged with no postoperative neurologic deficits.

**DISCUSSION**

Hemodynamic changes have been regarded as one of the main reasons for de novo aneurysm. Agenesis or hypoplasia of the ICA, as well as therapeutic occlusion, is known to promote hemodynamic stress and subsequent aneurysm development frequently in the AComA or in the contralateral ICA. Few patients exist in whom atherosclerotic stenosis or occlusion has been considered related to de novo aneurysm. Atherosclerosis induces degenerative breakdown of the vascular wall and promotes the formation of fibrous atheroma and resultant luminal stenosis. The narrowed ICA lumen by atherosclerotic plaques might provoke the weakening of the flow and subsequently turbulent effects, resulting in large variations in the wall shear stress that lead to the formation of de novo aneurysms.

Oculomotor nerve palsy is one of the presenting signs of an internal carotid (IC) PCOMA aneurysm. Local compression of the oculomotor nerve during IC-PCOMA aneurysm enlargement is usual, and the fundus of the aneurysm can sometimes adhere to the nerve. However, splitting and penetration of the oculomotor nerve is rare (Table 1). The intracranial oculomotor nerve is divided into proximal (interpeduncular), middle, and distal (intracavernous) parts. Cahill et al. confirmed that the proximal part of the oculomotor nerve was frequently penetrated by thalamoperforating arteries and branches of brainstem vessels, but the middle part (from the point where the oculomotor nerve exits the interpeduncular cistern to where the nerve enters the cavernous sinus) usually did not have any penetration by extraneural vessels. Three explanations for a penetrated cranial nerve by intracranial aneurysm were proposed by Beatty: 1) the aneurysm enlarging along congenital splitting of the nerve, 2) the aneurysm originating from a vessel already penetrating the nerve, and 3) the aneurysm actually penetrating the nerve.

In the present case, several notable findings were given: 1) with no doubt a de novo aneurysm had developed and enlarged over at least a 5-year period associated with symptomatic atherosclerotic stenosis of the ICA; 2) this de novo aneurysm was relatively low-lying compared to usual IC-PCOMA aneurysms, and no PCOMA or its remnant were noted; and 3) neither congenital nerve splitting related to the aberrant vessels nor prior subtle subarachnoid leakage was observed intraoperatively and clinically. Given that the described oculomotor nerve anatomy which is relevant to neurovascular relationship and there was confirmation of no PCOMA during surgery, there is little possibility of congenital oculomotor nerve splitting by intracranial vessels. Instead, we theorize that initially the atherosclerotic ICA was in close contact with the very distal portion of the middle part of the oculomotor nerve, just before entering the cavernous sinus.
As for the absence of cranial nerve symptom in a de novo fenestration, Wang et al. theorized that with a gradual parting of the nerve to its fiber, while the stretch is within physiological limits, nerve integrity could remain in no disruption. The absence of ocular symptoms in the described case was considered to be in line with Wang’s theory. For the best outcomes with operative treatment in this unfamiliar condition, where already the oculomotor nerve is traumatically pierced and penetrated by the pulsatile aneurysm, every effort to preserve the nerve function should be made by avoiding any unnecessary manipulation around the aneurysm dome that is embedded between the nerve bundles in jeopardy.

CONCLUSION

Herein, the authors present a case of a de novo aneurysm, which resulted from atherosclerotic stenosis of the supraclinoid ICA that was found to be penetrating the oculomotor nerve with no ocular palsy. Close and careful radiologic follow-up over a long period in patients with intracranial atherosclerotic stenosis might be justified regardless of symptoms to verify the possibility of de novo aneurysm.

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