

Simultaneous Presentation of Two Cerebral Aneurysms

—Case Report—

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Abstract

A 48-year-old woman experienced sudden onset of severe headache. Computed tomography showed subarachnoid hemorrhage (SAH) and intracerebral hematoma in the right frontal lobe. Digital subtraction angiography revealed three aneurysms in the anterior communicating artery (AcomA), the right posterior communicating artery (PcomA), and the right middle cerebral artery. The AcomA aneurysm was treated with endovascular coiling. However, her oculomotor nerve palsy was aggravated after the procedure. Embolization of the right PcomA aneurysm was conducted immediately and her oculomotor nerve palsy recovered completely 3 months later. Simultaneous presentation of multiple aneurysms with separate symptoms is rare. We speculate that the progressive oculomotor nerve palsy was caused by tiny enlargement or morphological change of the aneurysm caused by elevated blood pressure and pulsatile effect after SAH.

Key words: simultaneous presentation, cerebral aneurysm, oculomotor palsy, subarachnoid hemorrhage, endovascular treatment

Introduction

Multiple aneurysms are present in 19% of patients with ruptured cerebral aneurysms.⁸⁾ The purpose of acute surgery is to prevent rebleeding from the ruptured aneurysm, so simultaneous treatment of concomitant unruptured aneurysms is not always necessary.⁹⁾ We report a case of simultaneous presentation of two symptomatic cerebral aneurysms successfully treated with the endovascular technique in the acute stage.

Case Report

A 48-year-old woman experienced sudden onset of severe headache and was transferred to the nearest local hospital by ambulance. On arrival, she was comatose but her consciousness recovered gradually. No lateralizing motor deficit was evident and her pupils were isocoric. Computed tomography (CT) showed subarachnoid hemorrhage (SAH) and intracerebral hematoma in the right frontal lobe. She was then transferred to our hospital by ambulance.

On admission, her neurological status was World Federation of Neurosurgical Societies grade I and systolic blood pressure was 170 mmHg. We immediately started continuous infusion of nifedipine to decrease blood pressure and mild sedation with dexmedetomidine. Her pupils were

anisocoric (right 3 mm > left 2.5 mm), but blepharoptosis was not seen. Further detailed ophthalmological evaluation was not conducted. CT revealed no signs of rebleeding (Fig. 1).

Digital subtraction angiography showed multiple aneurysms arising from the left A₁-A₂ junction (5 mm in diameter), the right internal carotid artery-posterior communicating artery (PcomA) junction (2.5 mm), and the

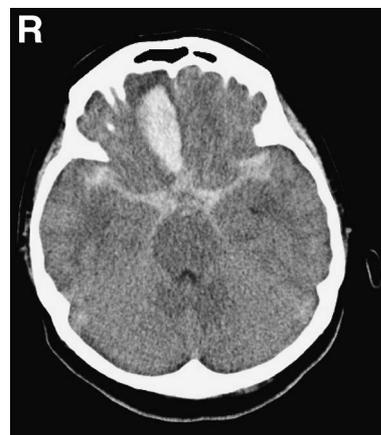


Fig. 1 Computed tomography scan on admission showing subarachnoid hemorrhage and intracerebral hematoma in the right frontal lobe.

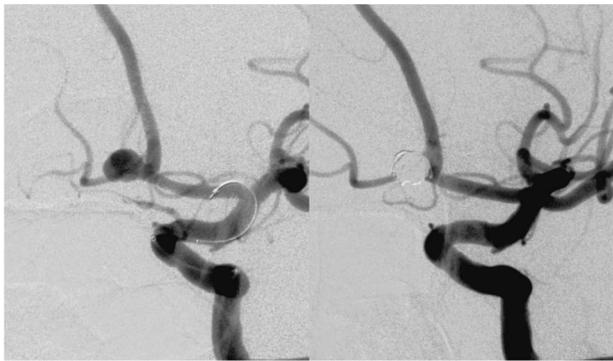


Fig. 2 Left carotid angiograms, anterior oblique view, obtained during coil embolization of the anterior communicating artery aneurysm showing complete obliteration of the aneurysm.

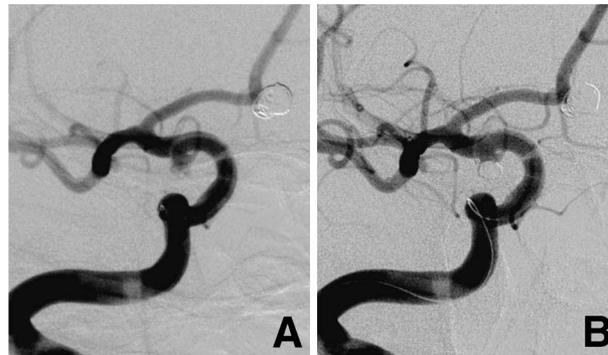


Fig. 4 Right carotid angiograms, anterior oblique view, obtained just before (A) and after (B) the embolization revealing complete obliteration of the right internal carotid-posterior communicating artery aneurysm.



Fig. 3 A: Photograph of the patient's face obtained just after the first embolization showing right blepharoptosis. B: Photograph obtained 4 days after the endovascular treatment revealing improvement of right blepharoptosis.

right middle cerebral artery-anterior temporal artery bifurcation (1.5 mm). These CT findings indicated that the anterior communicating artery (AcomA) aneurysm was the most likely source of bleeding, so we decided to perform embolization of the AcomA aneurysm. Under general anesthesia, a microcatheter was navigated into the aneurysm. After insertion of five Guglielmi detachable coils (total 31 cm; Stryker Neurovascular, Fremont, California, USA), the aneurysm was completely occluded (Fig. 2).

After waking up from general anesthesia, she was alert, but oculomotor nerve palsy was aggravated. Right blepharoptosis became evident (Fig. 3A), with anisocoria (right 5 mm > left 2.5 mm), absence of pupillary light reflex, and disturbance of the right eye movement in the upward, downward, and medial directions. Postoperative CT revealed no findings of rebleeding. We speculated that the aggravation of the third nerve palsy indicated impending rupture of the PcomA aneurysm. Immediate right carotid angiography showed the shape and size of the PcomA aneurysm remained unchanged (Fig. 4A). A microcatheter was advanced into the aneurysm (SL10 J curve type; Stryker Neurovascular), and placement of two

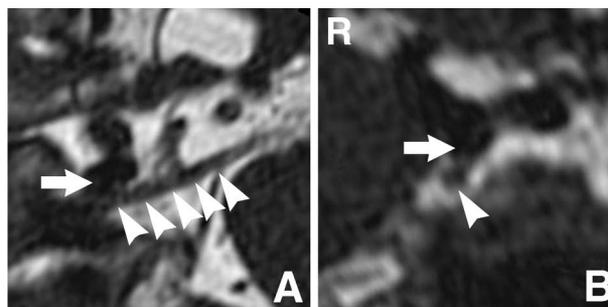


Fig. 5 Constructive interference with steady state magnetic resonance images (A: oblique sagittal view, B: oblique coronal view) revealing that the treated internal carotid-posterior communicating artery aneurysm (arrow) was just adjacent to the upper surface of the right oculomotor nerve (arrowheads).

detachable coils, TRUFILL DCS ORBIT™ MiniComplex Fill 2.5 mm × 45 mm (Codman & Shurtleff, Inc., Raynham, Massachusetts, USA) and ED Coil Extra Soft, 2 mm × 30 mm (Kaneka Medix Corp., Tokyo), resulted in complete obliteration of the aneurysm (Fig. 4B).

The postoperative course was uneventful. Four days after the treatment the blepharoptosis resolved (Fig. 3B), and diplopia was markedly improved at 7 days. Twenty-one days after admission, she was discharged on foot with negligible diplopia and anisocoria. Three months after the treatment she did not complain of diplopia, and her pupils were isocoric. Three-dimensional constructive interference in steady state (3D-CISS) magnetic resonance (MR) imaging showed that the treated PcomA aneurysm was just adjacent to the upper surface of the right oculomotor nerve (Fig. 5).

Discussion

Simultaneous presentation of multiple symptomatic aneurysms is uncommon.^{5,9,11-13} In a few case reports of simultaneous rupture of multiple aneurysms,^{5,13} CT showed multiple separate hematomas, and open surgery

confirmed simultaneous bleeding from multiple aneurysms. Rupture of a concomitant unruptured aneurysm occurred within 2 weeks after surgical repair of a ruptured aneurysm.¹¹⁾ This patient had multiple risk factors (e.g., hypertension, smoking, family history of SAH), and the concomitant aneurysm was multilobular with a bleb. Postoperative hypertensive hypervolemic therapy might have also increased the risk of aneurysm rupture. That case suggested that multiple aneurysms in patients with such risk factors should be treated simultaneously. Enlargement of concomitant unruptured carotid cavernous aneurysm occurred just after bleeding from a middle cerebral artery aneurysm.¹²⁾ These findings suggest that elevation of blood pressure after SAH can induce enlargement and rupture of concomitant aneurysms.

In our case, oculomotor nerve palsy occurred subsequent to rupture of the AcomA aneurysm and progressively aggravated. Such progressive exacerbation of oculomotor nerve palsy may be due to tiny enlargement or morphological change of the aneurysm caused by elevated blood pressure and the pulsatile effect after SAH. Once an aneurysm has enlarged, continuous compression can cause progressive oculomotor palsy in spite of antihypertensive treatment.

Some cases of oculomotor nerve palsy after SAH are known without responsible aneurysms.^{1,3,10)} Subarachnoid clot,³⁾ vascular anomalies,¹⁾ blood flow jet,¹⁰⁾ and elevated intracranial pressure^{1,3,10)} were pointed out as causes of oculomotor palsy. In our case, rapid improvement of symptoms after the treatment and the findings of postoperative 3D-CISS MR imaging suggested that the PcomA aneurysm was responsible for the oculomotor palsy.

Previous reports have recommended early clipping or coiling of PcomA aneurysm with progressive oculomotor palsy to prevent catastrophic SAH.^{2,4,6,7,14)} Fortunately we were able to treat both aneurysms successfully by endovascular procedures. Detailed neurological evaluation is often difficult in the acute phase of SAH. This case underscores the importance of basic neurological examination in addition to detailed neuroimaging.

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