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Case Report

Development of 'De novo' Aneurysm after Therapeutic Carotid Occlusion

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Carotid occlusion is an inevitable therapeutic modality for the treatment of complex aneurysms such as giant, traumatic, and intracavernous aneurysms. Late complications of carotid occlusion include 'de novo' aneurysm formation at a distant site because of hemodynamic changes in the circle of Willis. We report a case of de novo aneurysm in a vessel that appeared to be normal on initial angiography. The patient developed an anterior communicating artery aneurysm and marked growth of a basilar bifurcation aneurysm 9 years after trapping of the left internal carotid artery for the treatment of a ruptured large saccular aneurysm involving ophthalmic and cavernous segments. We propose that patients who undergo therapeutic carotid occlusion should be periodically followed by magnetic resonance angiography or computed tomographic angiography to evaluate the possibility of de novo aneurysm formation; this advice is in line with previous reports.

KEY WORDS : De novo aneurysm · Carotid occlusion · Late complication · Magnetic resonance angiography · Computed tomographic angiography.

INTRODUCTION

Despite the advances in surgical and endovascular techniques, there remains a group of challenging aneurysms that cannot be treated without sacrifice of the parent feeding artery by surgical or endovascular approaches. Several late complications of carotid occlusion are well recognized, and may even occur many years after the procedure. These are 1) delayed ischemia; 2) aneurysm regrowth and delayed hemorrhage; and 3) formation of a de novo cerebral aneurysm at a location distant from the original aneurysmal site[1,2,4,15,17]. We report a case of de novo aneurysm formation and marked growth of a prior preaneurysmal lesion occurring after therapeutic carotid artery occlusion.

CASE REPORT

A 62-year-old woman presented with sudden-onset severe headache in December 1997. Initial neurological examination revealed a drowsy mentality with no focal neurological deficit. Brain computed tomography (CT) showed a subarachnoid hemorrhage (SAH) at the basal cistern. Left carotid angiography revealed a large saccular aneurysm originating from the ophthalmic segment, and involving the cavernous segment; a small saccular aneurysm at the origin of the posterior communicating segment; and a preaneurysmal dilatation at the bifurcation of the basilar artery (Fig. 1A, B, C). Because no symptoms were seen on a balloon occlusion test (BTO) of the left internal carotid artery (ICA), trapping of the left ICA by clipping of the cervical ICA and the distal ICA proximal to the anterior choroidal artery was performed. Postoperative angiography showed complete occlusion of the left ICA with no opacification of the aneurysm (Fig. 1D).

In February 2006, after a 9-year symptom-free period, our patient again experienced severe headache. A brain CT showed a SAH along the interhemispheric fissure and focal hematoma in the septum pellucidum (Fig. 2A). Brain computed tomographic angiography (CTA) and four-vessel angiography confirmed the occlusion of the left ICA and revealed an anterior communicating aneurysm raising a suspicion of rupture, and prominent growth of a basilar bifurcation aneurysm (Fig. 2B, C, D). The anterior communicating artery aneurysm was successfully treated by detachable coil embolization with no postoperative complications. We planned to treat the basilar top aneurysm by

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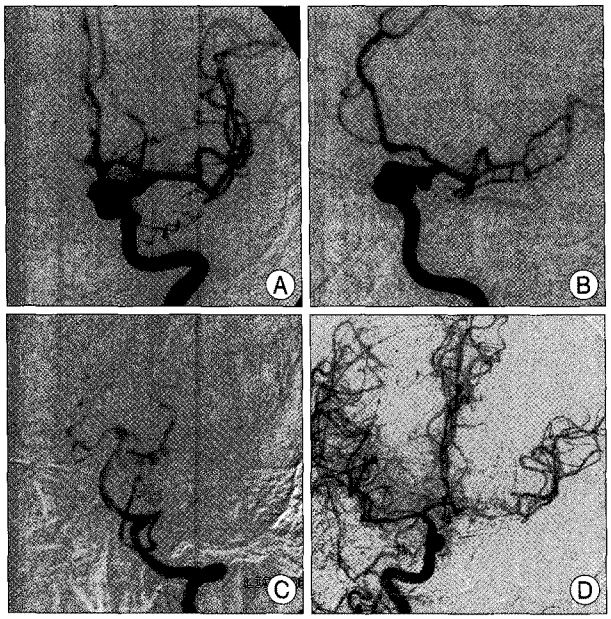

Fig. 1. A and B : Left carotid angiogram showing a large aneurysm at the paraclinoid segment involving cavernous portion of the left internal carotid artery and a small aneurysm at the anterior choroidal artery. C : Left vertebral angiogram revealing a preaneurysmal dilatation at the basilar bifurcation. D : Right carotid angiogram after therapeutic carotid occlusion showing no aneurysmal shadow and anterior communicating artery aneurysm.

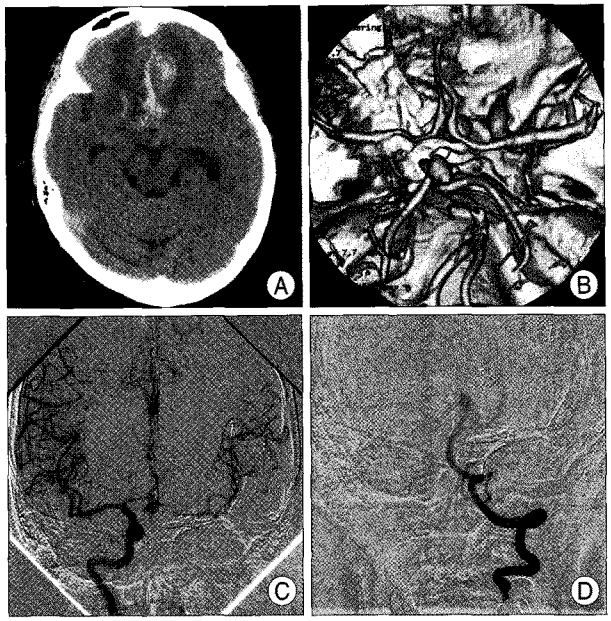

Fig. 2. A : Computed tomography taken 9 years after therapeutic carotid occlusion showing subarachnoid blood in the interhemispheric fissure and hematoma in the septum pellucidum. B : Computed tomographic angiogram revealing a de novo aneurysm at the anterior communicating artery and remarkable growth of the saccular aneurysm of the basilar bifurcation. C : Right carotid angiogram revealing an anterior communicatin artery aneurysm. D : Left vertebral angiogram showing a remarkable enlargement of basilar apex aneurysm.

detachable coil embolization with regular radiological follow-up evaluation. The basilar bifurcation aneurysm responded almost completely to the endovascular technique, using a detachable coil and no stent, 2 years after coil embolization of the ruptured anterior communicating artery aneurysm.

DISCUSSION

The pathogenesis of aneurysm formation remains unresolved. However, it is thought that the complex hemodynamic environment at an arterial bifurcation is characterized by high wall shear stress and a high wall shear stress gradient. The combination of wall shear stress and the wall shear stress gradient at the bifurcation apex is suggested to predispose the local vessel wall to aneurysm initiation[11,12]. Abnormally high flow in intracranial arteries, such as the afferent feeding arteries, draining into low-resistance venous stumps within an arteriovenous malformation, is believed to increase the incidence of cerebral aneurysm[14]. In a study of rabbits, Gao and colleagues[6] demonstrated that in the absence of other known predisposing factors such as hypertension, genetic susceptibility, and vascular wall weakening factors, an increased hemodynamic insult could initiate nascent aneurysms at the basilar bifurcation, as also shown by Hassler[7]. The increased flow at a bifurcation apex caused by vessel ligation would result in further elevation of the wall shear stress and wall shear stress gradient in the hemodynamically vulnerable zone. The bifurcation danger zone for hemodynamic insult can trigger local maladaptive vascular remodeling leading to aneurysm initiation[11,12].

The occurrence of de novo aneurysms is a well known late complication of therapeutic carotid occlusion. The incidence of de novo aneurysm formation following carotid occlusion varies from 0% to 20 % with a mean around 4-11%[1,5,13]. There are more than 30 reports of de novo aneurysm formation after carotid occlusion[1-4,9,10,16,19,20]. The majority of these aneurysms were situated in the anterior circulation, in the anterior communicating artery, the contralateral posterior communicating artery, and the contralateral ICA; these vessels were the sites of the main hemodynamic changes associated with the increased blood flow necessary to supply the contralateral carotid circulation.

Wermer[18] and colleagues reported that important risk factors for de novo aneurysm development and growth of previous aneurysms in a cohort of patients with clipped aneurysms were the occurrence of multiple aneurysms, a history of hypertension, a smoking history, familial aneurysm history, and female sex. Because our patient had several risk factors for the development of new aneurysms and enlargement of previous aneurysms (such as multiple aneurysms,

female sex, and hypertension that occurred after carotid occlusion), new aneurysm formation and enlargement of previous tiny aneurysms might have occurred even if our patient had not undergone therapeutic carotid occlusion.

Multiple aneurysms were observed in about 10% of cases. The de novo aneurysm of the anterior communicating artery and the enlargement of the basilar bifurcation aneurysm may have resulted from hemodynamic changes after carotid artery occlusion, in addition to increased patient susceptibility to the development of new aneurysms and enlargement of existing aneurysms.

The time to de novo aneurysm formation after therapeutic carotid occlusion ranged from 4 to 25 years with a mean of 9.6 years. Although carotid occlusion significantly influences intracranial circulation, mural arterial degeneration likely requires at least several years before an aneurysm begins to grow. Drapkin and Rose[3] reported a case with "serial" development of de novo aneurysms in different sites after different intervals. This suggests that patients with de novo aneurysms, successfully treated by surgical or endovascular approaches, must not be considered as definitively cured. Such patients must be followed up periodically, to at least 25 years after carotid artery occlusion, based on previous reports[1,5,17]. In our opinion, 2 to 3 years is adequate as a follow-up interval after therapeutic carotid occlusion.

If a patient for whom therapeutic carotid occlusion is planned shows risk factors for de novo aneurysm formation and enlargement of previous aneurysms, it is suggested that follow-up of such patients should be performed using only non-invasive imaging modalities, such as magnetic resonance angiography or CTA, because of the potential for morbidity if conventional angiography is employed[8].

CONCLUSION

Therapeutic carotid occlusion remains an inevitable modality in the treatment of specific cerebrovascular disorders. However, it should be remembered that de novo aneurysm formation may occur after this procedure because of hemodynamic changes, especially in patients showing risk factors for the development of de novo aneurysms and enlargement of previous aneurysms. There is no standard protocol for routine follow-up of such patients. To detect de novo aneurysms as early as possible, without aneurysm rupture, we propose that non-invasive modalities such as CTA or MRA should be regularly performed every 2 to 3 years after therapeutic carotid occlusion.

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