

Thrombosed Unruptured Cerebral Aneurysm Causing Brain Infarction Followed by Subarachnoid Hemorrhage

—Case Report—

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Abstract

A 71-year-old man presented with right hemiparesis and aphasia due to cerebral infarction in the frontal lobe. Computed tomography (CT) revealed a high-density mass, 12 mm in diameter, in the stem of the left sylvian fissure. Carotid angiography demonstrated occlusion of the left ascending frontal artery complex and retention of contrast medium at the bifurcation of the left middle cerebral artery (MCA). The diagnosis was cerebral infarction caused by occlusion of the ascending frontal artery complex resulting from thrombosed left MCA aneurysm. The patient was managed conservatively and his neurological symptoms gradually improved. One month later, he lapsed into a coma. CT revealed subarachnoid hemorrhage. Carotid angiography showed a large left MCA aneurysm with branch occlusion of the left ascending frontal artery complex. A left frontotemporal craniotomy was performed. The MCA aneurysm was opened and the intramural thrombi removed, and finally neck clipping was performed. The patient made a good postoperative recovery.

Key words: brain infarction, thrombosed aneurysm, subarachnoid hemorrhage

Introduction

Unruptured cerebral aneurysm has an incidence of 3.6–6%.¹³⁾ Unruptured cerebral aneurysm is generally asymptomatic and detected incidentally, but symptoms of mass effect or ischemia are occasionally observed.⁵⁾ Ischemic symptoms were observed in 3–6.3% of unruptured aneurysms.^{8,9,15)} Partially thrombosed cerebral aneurysm is associated with thromboembolic events due to propagation of mural thrombus.^{4,14)} Here we report a case of partially thrombosed middle cerebral artery (MCA) aneurysm with an extremely unusual clinical course of cerebral infarction followed by subarachnoid hemorrhage (SAH).

Case Report

A 71-year-old man presented with sudden onset of right hemiparesis and aphasia. Computed tomography (CT) obtained 2 hours after the onset of symptoms revealed no intraparenchymal abnormality

except for a high-density mass, 12 mm in diameter, in the stem of the left sylvian fissure (Fig. 1A), which was suspected to be a left MCA aneurysm. Magnetic resonance (MR) imaging revealed the aneurysm containing homogeneous isointense thrombus on T₁-weighted imaging (Fig. 1B), and hyperintense anterior and hypointense posterior thrombus on fluid-attenuated inversion recovery imaging, which indicated thrombus in different stages of organization (Fig. 1C). Diffusion-weighted MR imaging showed a hyperintense area around the left frontoparietal operculum (Fig. 1D). He had no history of hypertension or diabetes. Other laboratory tests including the coagulation function test, and electrocardiography and echocardiography revealed no abnormality. Carotid angiography showed occlusion of the left ascending frontal artery complex and retention of contrast medium at the bifurcation of the left MCA in the late arterial phase (Fig. 2A). The diagnosis was brain infarction due to occlusion of the left ascending frontal artery complex, as a result of thromboembolism from the thrombosed aneurysm. His right hemiparesis gradually improved with conservative therapy.

Carotid angiography performed 1 month after the

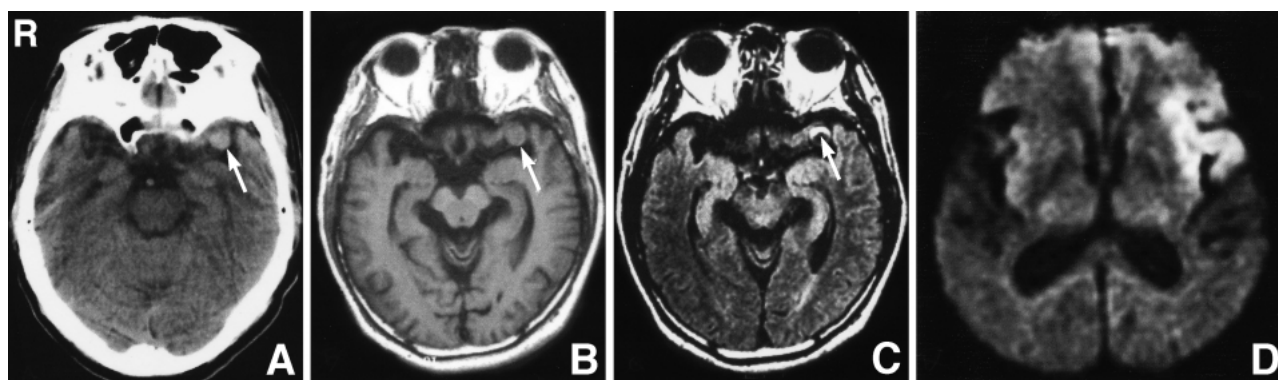


Fig. 1 A: Computed tomography scan obtained 2 hours after the onset of symptoms showing no intraparenchymal abnormality except for a high-density mass of 12 mm diameter in the stem of the left sylvian fissure (arrow). B: T₁-weighted magnetic resonance (MR) image showing a homogeneous isointense thrombus in the middle cerebral artery aneurysm (arrow). C: Fluid-attenuated inversion recovery image showing the thrombus with hyperintense anterior and hypointense posterior parts (arrow). D: Diffusion-weighted MR image showing a hyperintense area around the left frontoparietal operculum.

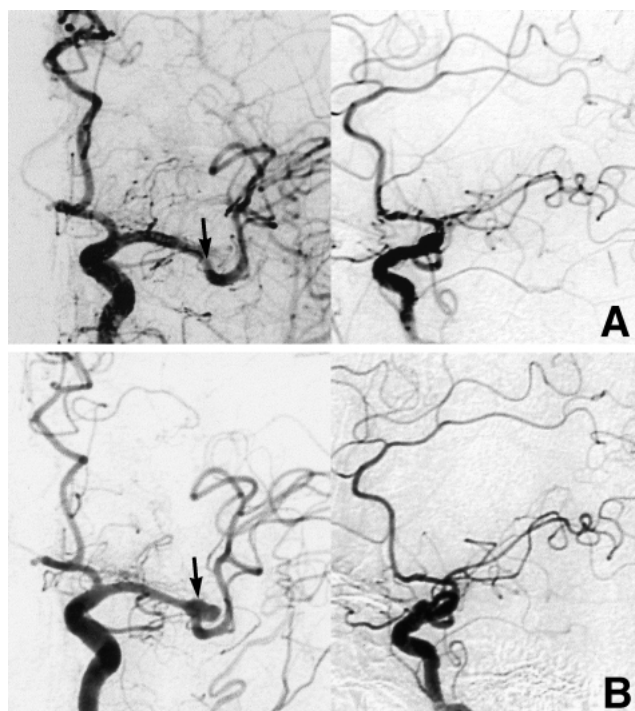


Fig. 2 A: Initial left carotid angiograms showing occlusion of the left ascending frontal artery complex and retention of contrast medium at the bifurcation of the left middle cerebral artery (arrow). B: Left carotid angiograms obtained 1 month after the onset showing occlusion of the left ascending frontal artery complex with partial opacification of the left middle cerebral artery aneurysm (arrow).

onset showed occlusion of the left ascending frontal artery complex with partial opacification of the left MCA aneurysm (Fig. 2B). We considered surgery. However, examination at 1 month after development of the initial symptoms found good progress in rehabilitation, so we decided to treat the patient conservatively until his functional recovery plateaued. Therefore, he was treated with oral aspirin (80 mg/day) in consideration of the risk of further thrombus from the thrombosed aneurysm rather than rupture.

Two months after the initial ischemic insult, he was found comatose. CT revealed SAH (Fig. 3A). Carotid angiography showed a large left MCA aneurysm with occlusion of the left ascending frontal artery complex (Fig. 3B). Left frontotemporal craniotomy was performed. The sylvian fissure was opened widely and the MCA aneurysm exposed. A clot was found adhering to the anteromedial side of the dome of aneurysm, which was considered to be the rupture point (Fig. 4A). The large MCA aneurysm was trapped, the dome of the aneurysm was incised (Fig. 4B), and intramural thrombi at various stages were completely removed, so that optimal clipping could be achieved. No arteriosclerotic change of the ascending frontal artery was seen. As the ascending frontal artery was completely occluded and cerebral infarction had already occurred in the affected area, vascular reconstruction of this artery complex was not attempted. The patient made a good postoperative recovery. He was discharged 1 month later with expression aphasia.

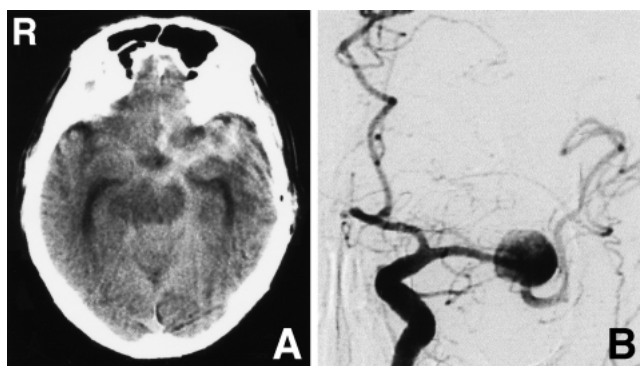


Fig. 3 A: Computed tomography scan obtained 2 months after the initial ischemic insult showing subarachnoid hemorrhage predominantly in the left sylvian fissure. B: Left carotid angiogram obtained 2 months after the initial ischemic insult showing a large left middle cerebral artery aneurysm with occlusion of the left ascending frontal artery complex.

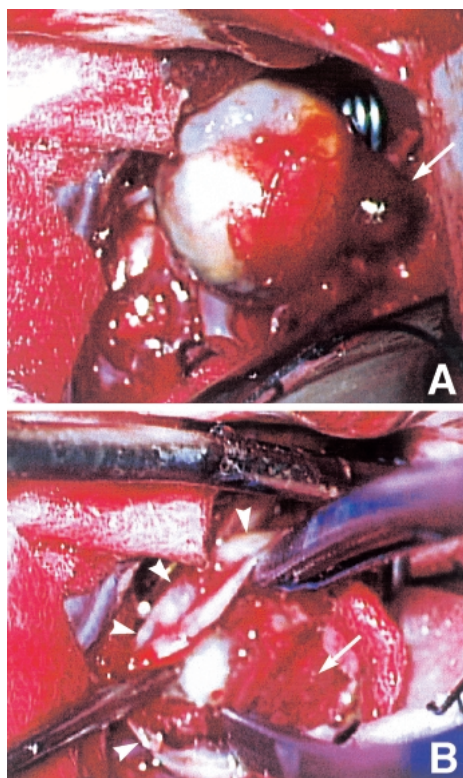


Fig. 4 A: Intraoperative photograph showing a clot adhering to the anteromedial side of the aneurysm dome (arrow). B: Intraoperative photograph showing the aneurysm wall (arrowheads) and intramural thrombus in the aneurysm (arrow).

Discussion

Intramural thrombi are often observed in larger aneurysms and are thought to be one of the causative factors of ischemic symptoms. Formation of thrombus was detected in 48% of aneurysms of 20–25 mm diameter and in 76% of aneurysms of over 25 mm diameter.¹⁰⁾ The average diameter of unruptured aneurysms with ischemic symptoms was 12.5 mm.⁸⁾ In the present case, the aneurysm was 12 mm in diameter. SAH is associated with 33–44% of thrombosed aneurysms,^{3,14)} and ischemic symptoms with 5–8%.^{4,14)} However, thrombosed aneurysm causing brain infarction, followed by SAH, is extremely rare.

Reduction of blood inflow, increased blood viscosity, and turbulent blood flow are important causative factors in the formation of intramural thrombi.^{1,11)} Turbulent blood flow in the aneurysm dome causes serious damage to the endothelium of the aneurysm wall and leads to exposure of the subendothelial matrix that finally induces platelet aggregation.¹²⁾ Intramural thrombus impairs the diffusion of oxygen and metabolites into the aneurysm wall.²⁾ The clinical course of the present case was interpreted as follows. Turbulent blood flow in the aneurysm dome caused significant damage to the endothelium of the dome wall, which probably accelerated formation of the thrombus. Thrombotic emboli from the thrombosed aneurysm caused occlusion of the branches of MCA, resulting in brain infarction. The intramural thrombus may also have caused ischemia and damage of the aneurysm wall with fragmentation of the elastic lamina. Rapid resolution of fresh intramural thrombi associated with the fragile aneurysm walls may have lead to aneurysm rupture.

Treatment of unruptured cerebral aneurysm causing brain infarction is controversial. Some cases have been treated surgically, whereas others were treated conservatively with or without medication.^{7,8)} The prognosis for surgical treatment of unruptured cerebral aneurysm complicated by ischemic cerebrovascular disease is poorer than that of asymptomatic unruptured cerebral aneurysm. The surgical morbidity and mortality in cases of unruptured cerebral aneurysm complicated by ischemic cerebrovascular disease were 18% and 4%, respectively, and conservative therapy is recommended in such cases.⁶⁾ In the present case, the cerebral aneurysm was first detected at the acute stage of brain infarction and conservative therapy was instituted. Cases of thrombosed aneurysm causing brain infarction, followed by SAH, are extremely rare. However, the possibility of aneurysm

rupture should be considered if carotid angiography shows rapid resolution of the intramural thrombus.

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