Ruptured Cerebral Aneurysm Complicated With Rebleeding Following Thrombolysis During Endovascular Embolization
—Two Case Reports—

Kentaro HAYASHI, Hideaki TAKAHATA*, and Naoki KITAGAWA

Department of Neurosurgery, Nagasaki University School of Medicine, Nagasaki; *Department of Neurosurgery, National Hospital Organization Nagasaki Medical Center, Omura, Nagasaki

Abstract

A 68-year-old woman and a 42-year-old woman presented with subarachnoid hemorrhage due to rupture of cerebral aneurysm. Both patients were treated with endovascular coil embolization. Thromboembolic complications occurred during the procedure and local thrombolysis was performed for recanalization. One patient developed massive rebleeding immediately after the procedure and the other suffered minor hemorrhage adjacent to the embolized aneurysm 2 days later. Local thrombolysis during treatment of ruptured aneurysm by coil embolization carries a significant risk of rebleeding. Prevention of thromboembolic complication by adequate heparinization is important.

Key words: subarachnoid hemorrhage, cerebral aneurysm, endovascular coiling, thrombolysis, rebleeding

Introduction

The introduction of endovascular treatment of intracranial aneurysms using Guglielmi detachable coils (GDCs) has resulted in a great increase in the number of aneurysms treated by endovascular embolization. Large series of aneurysms treated in this way suggest low technique-related morbidity and excellent short-term outcome. Thromboembolic and ischemic complications remain the major risk of this procedure, occurring in 2–6% of cases. Periprocedural thromboembolism can be treated with selective intra-arterial administration of thrombolytic agents, but this procedure carries the risk of bleeding. However, the rate of rebleeding in these patients is very low.

Here we report two cases of ruptured cerebral aneurysm complicated with rebleeding after thrombolysis during endovascular embolization.

Case Reports

Case 1: A 68-year-old woman was referred to our hospital for treatment of ruptured aneurysm 3 days after the onset. Computed tomography (CT) showed diffuse subarachnoid hemorrhage (SAH) (Fig. 1). CT angiography revealed a 3-mm aneurysm arising from the anterior communicating artery and another 2-mm aneurysm at the left internal carotid artery (ICA)-anterior choroidal artery bifurcation (Fig. 2A).

Endovascular coil embolization was performed under general anesthesia. Heparin (3,000 units) was given at the beginning without monitoring of activated coagulation time (ACT). The aneurysm of the left ICA was embolized with a 2 mm × 4 cm GDC. Then, a microcatheter was introduced to the aneurysm of the anterior communicating artery and a 3 mm × 4 cm GDC was placed. Although a 2 mm × 6 cm GDC was inserted without difficulty, the bilateral anterior cerebral arteries (ACAs) were occluded during insertion of a 2 mm × 4 cm GDC.

Since slight coil protrusion to the anterior communicating artery was seen, the third coil was withdrawn. However, the coil protrusion persisted...
indicating that a portion of the first or second coil extended into the arterial lumen and the arteries remained occluded (Fig. 2B). We speculated that the obstruction of the parent vessel was possibly caused by coil protrusion with additional local thrombus formation. Local thrombolysis was performed using 3.3 mg of recombinant tissue plasminogen activator (tPA) injected through a microcatheter in the anterior communicating artery, then the left ACA was recanalized (Fig. 2C).

The patient recovered from the anesthesia and responded to simple commands after the procedure. However, she deteriorated suddenly with agonal breathing in the intensive care unit and CT showed massive SAH (Fig. 3). She died 6 days after the treatment.

**Case 2**: A 42-year-old woman was referred to our hospital for treatment of ruptured aneurysm 1 day after the onset of symptoms. CT demonstrated diffuse SAH (Fig. 4). Diagnostic angiography revealed a 6-mm aneurysm on the anterior communicating artery (Fig. 5A).

Endovascular embolization was performed under general anesthesia. A 6 mm×10 cm three-dimensional GDC was placed into the aneurysm via the left ACA followed by a 4 mm×8 cm GDC. After placement of these two coils, heparin was administered to maintain an ACT of approximately 250 seconds during the procedure. Placement of GDCs was continued to a total length of 36 cm (6 mm×10 cm, 4 mm×8 cm, 3 mm×10 cm, 2 mm×4 cm, 2 mm×4 cm). The aneurysm was subtotally occluded with minor residual filling of its neck (Fig. 5B). Angiography revealed occlusion of the left ACA at the end of the procedure.

The thrombus initially appeared at the A2 portion but migrated spontaneously into the callosomarginal artery (Fig. 5C). A microcatheter was introduced to the occlusion site and local thrombolysis was performed using 240,000 units of urokinase. The artery
Fig. 4 Case 2. Axial computed tomography scan on admission revealing subarachnoid hemorrhage in the interhemispheric fissure.

Fig. 5 Case 2. A: Pretreatment left carotid angiogram demonstrating an aneurysm arising from the anterior communicating artery. B: Left carotid angiogram during embolization showing subtotal obliteration of the aneurysm. C: Left carotid angiogram at the end of the embolization showing occlusion of the callosomarginal artery. D: Left carotid angiogram after thrombolysis showing recanalization of the artery.

Fig. 6 Case 2. Axial computed tomography scan after the rebleeding episode demonstrating a minor interhemispheric hematoma adjacent to the aneurysm.

Rebleeding Following Coil Embolization of Cerebral Aneurysm

was recanalized (Fig. 5D). Postoperatively, neurological examination showed no deficit and CT revealed washout of the hematoma without ischemic lesion. Anticoagulation and antiplatelet therapies were employed for the prevention of recurrence of thromboembolic complication as well as cerebral vasospasm.

The patient suffered severe headache 2 days after the embolization and CT demonstrated hemorrhage adjacent to the embolized aneurysm (Fig. 6). Angiography after the episode did not show any significant recurrence. Both anticoagulation and antiplatelet therapies were stopped and blood pressure was controlled to avoid hypertension. She recovered and was discharged with mild mental disturbance 1 month later.

Discussion

The low frequency of thromboembolic events after GDC embolization of aneurysms does not contraindicate the use of periprocedural anticoagulation. Heparinization was started at the beginning in Case 1 or after second coil placement in Case 2. In addition, the guiding catheter and microcatheter were continuously perfused with heparinized saline to prevent thromboembolism. In Case 1, we consider that the thromboembolism was caused by coil protrusion. However, the ACT was not monitored at that time, so heparinization might not have been enough to prevent thrombus formation. In Case 2, the placement of the microcatheter into the small caliber ACA or sharp takeoff of the A1 segments probably caused hypoperfusion of the distal ACA, which might have eventually induced the thrombo-
sis.

Thromboembolic complications are usually managed with local or systemic administration of fibrinolytic or antiplatelet agents. One of the most important problems is the risk of bleeding complication. The International Subarachnoid Aneurysm Trial identified five patients who received thrombolytic therapy to treat thromboembolic complication after endovascular treatment and all five patients died of rebleeding. Another five patients received intra-arterial thrombolytic agents to treat acute vascular occlusion associated with endovascular coil placement of intracranial aneurysm. Two patients suffered hemorrhage resulting in severe morbidity or death. Although thrombolysis may be relatively safe for unruptured cerebral aneurysm, or incidental aneurysm, a patient suffered rupture of an anterior communicating artery aneurysm during tPA thrombolysis for acute myocardial infarction. Our Case 1 suffered massive rebleeding and subsequent sudden deterioration. A total length of 10 cm GDCs was placed into the 3-mm diameter ACA aneurysm. Although dome filling was not observed at the end of the procedure, the embolization was probably not complete. Therefore, administration of tPA induced rebleeding. A potent inhibitor of glycoprotein IIb/IIIa receptor (abciximab) is used for thrombotic complication during aneurysm coiling in Western countries, but this agent is not available in Japan. Our Case 2 suffered minor rebleeding 2 days after the embolization. The second hemorrhage occurred predominantly in the interhemispheric fissure, suggesting that the source of bleeding was possibly from the part of the dome adhering to the frontal lobe. We considered that local thrombolysis in combination with postoperative anticoagulation therapy permitted recanalization or prevented thrombosis of the aneurysm dome, resulting in rupture 2 days after the coil. Histological studies have shown that aneurysms have a weak fibrin layer with only thin thrombus at the rupture site and thrombolysis will dissolve such clots. The decision to perform thrombolysis should be based on the involved vascular territory and the blood flow pattern including possible collateral supply, and mechanical clot disruption using a microcatheter or guide wire should be carried out before chemical thrombolysis to avoid rebleeding. In particular, prevention of thromboembolism by methods such as adequate heparinization under ACT monitoring is important.

Endovascular treatment of aneurysms with GDCs carries the risk of thromboembolism. The present cases illustrate the risks of rebleeding after local thrombolysis during endovascular embolization.

References


Address reprint requests to: Kentaro Hayashi, M.D., Department of Neurosurgery, Nagasaki University School of Medicine, 1–7–1 Sakamoto, Nagasaki 852–8501, Japan.
e-mail: kenkuni@net.nagasaki-u.ac.jp