

Delayed Complications after Uneventful Coil Embolization of Unruptured Aneurysms - Case Report -

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● ABSTRACT

Coiling of intracranial aneurysms is a generally safe treatment. However, despite increasing clinical experience and technological improvements, endovascular treatment still has inherent risks of morbidity and mortality. Recently, we have experienced two cases of delayed complications that developed after uneventful coil embolization of unruptured aneurysms. (**Kor J Cerebrovascular Surgery 10:519-523, 2008**)

KEY WORDS : Unruptured aneurysms · Coil Embolization

Introduction

During the past two decades, there has been a significant evolution in endovascular treatment. Especially, coil embolization has become an important alternative for the treatment of cerebral aneurysms. However, coil embolization is related to various complications such as intra-procedural aneurysm rupture and thromboembolism. Although coil complications mostly occur during the intra-procedural period, they can be occurred after. We report two cases of delayed post-procedural complications after uneventful coil embolization of unruptured paraclinoid aneurysms.

Case1

A 78-year-old woman was referred to our institution due to left third nerve palsy. Angiography showed a large paraclinoid aneurysm in the left internal carotid artery (ICA) measuring 16 mm in maximal diameter and saccular in shape (Fig. 1-A & B). The patient had no more neurological deficits, except third nerve palsy. We perform endovascular treatment considering of the age and location of the aneurysm. Through a 6 French (Fr) guiding catheter, a 2 Fr microcatheter was introduced into the aneurysm via the ICA. The aneurysm was completely occluded with detachable coils. Embolization was successfully but a coil loop protruded into the lumen of the parent artery. Neuroform stent (Boston Scientific; Natick, MA) was deployed into the parent artery in order to prevent thrombus formation. Consequently, the packing density was about 28%. Postoperative angiography could not detect the remnant aneurysm sac (Fig. 1-C & D). After treatment, the patient received intravenous heparin with monitoring of aPTT to prevent thrombosis. Immediate postoperative course was uneventful until 10 hours after embolization, when her mentality deteriorated. Emergent computerized tomography (CT) revealed subarachnoid hemorrhage in the basal cisterns

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and multiple intracerebral hemorrhages in the parietal lobe (Fig. 2). Digital subtraction angiography (DSA) was then performed promptly and did not show reconfiguration of the coil at the aneurysm neck when compared to the configuration immediately after embolization. Until two months after embolization, the patient had severe disability and neurological condition was not remarkably improved.

Case 2

The other patient was 71-year-old woman who had unruptured bilateral ICA aneurysms. One aneurysm with a size of 15x11 mm was located at the right distal ICA and the other originated from the junction of the ICA-superior hypophyseal arteries (Fig. 4-A & B). Both aneurysms were completely occluded with coils (Fig 4-C & D). The patient awoke from anesthesia without neurological deficits and the postoperative course was uneventful. She suffered from sudden onset of left side weakness 8 hours after the

operation. CT detected no cerebral infarction and hemorrhage, but immediate DSA revealed occlusion of the cortical branches of the middle cerebral artery (MCA) and anterior cerebral artery (Fig. 5). Emergent intra-arterial urokinase thrombolysis was tried but failed to recanalize occluded vessels. MRI scan performed on the next day showed hyperintense areas in the right motor cortex (Fig. 6). The patient had a severe left hemiparesis postoperatively. Six weeks after the procedure, her neurological status had slightly improved.

Discussion

The incidence of unruptured aneurysms is increasing in aging society, since imaging technique is more advanced. In the past decade, endovascular treatment of intracranial aneurysms has proven to be a safe and alternative treatment. However, despite technological improvement and advanced

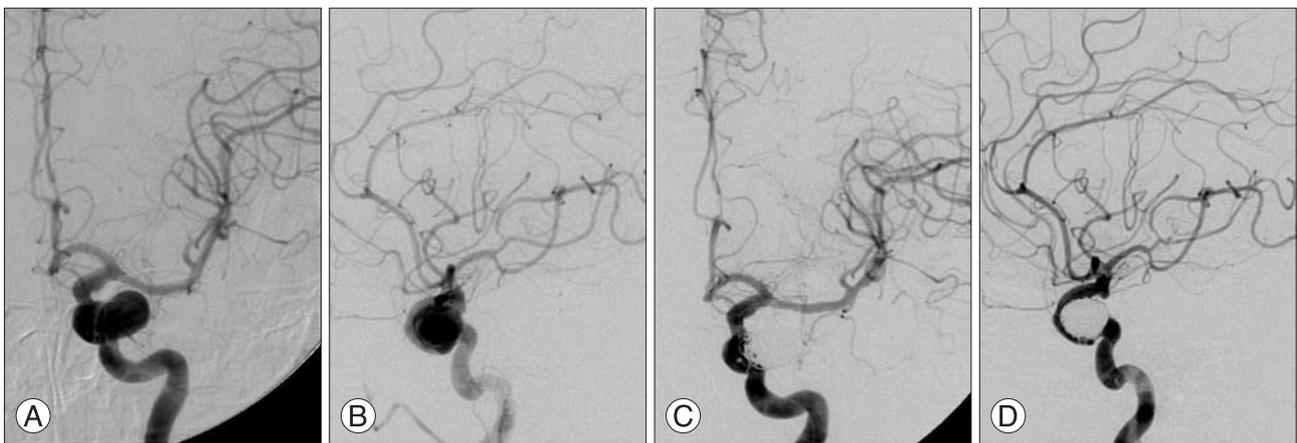


Fig. 1. Initial left ICA angiogram (A, B) shows a large paraclinoid aneurysm postero-laterally projected. Immediate postembolization angiogram (C, D) demonstrates no aneurysmal remnant.

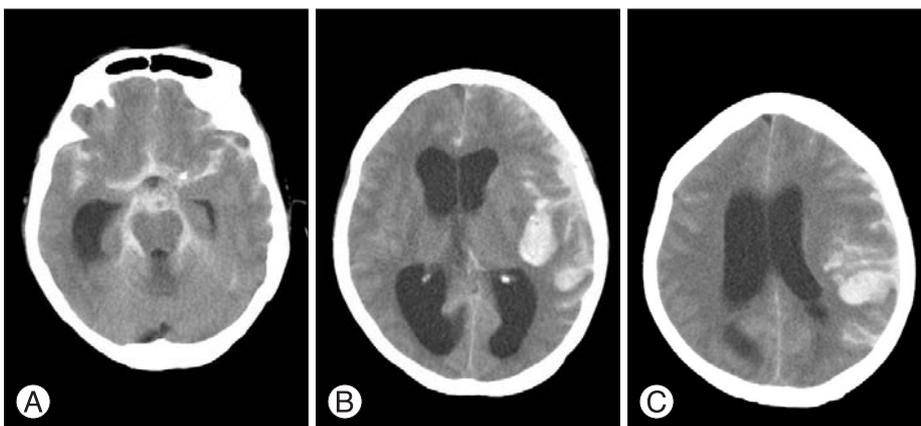


Fig. 2. Emergent brain CT after ictus reveals thick subarachnoid hemorrhage in the basal cisterns and left temporoparietal convexity.

equipment, endovascular treatment of intracranial aneurysms has unavoidable innate risks. Several groups have reported procedural complication rates related to coil embolization.⁷⁾⁽⁸⁾⁽¹⁴⁾ In the unruptured group, procedure related morbidity and mortality rates were 3.7~9.5% and 1.1~9.1%, respectively.²⁾⁽¹⁴⁾ Two main causes of procedure related morbidity and mortality in this group were thromboembolism and intra-procedural rupture. Especially, thromboembolism was the major cause of procedure related morbidity and mortality in unruptured aneurysms. Despite routine use of heparin in conjunction with this procedure, complications that related to thromboembolic events occurred in 2.5~28% of patients treated.⁵⁾⁽¹⁴⁾⁽¹⁸⁾ Large

aneurysm, especially wide necked aneurysm, is a reliable predictor of high risk of thrombus formation. The risk of thromboembolic complication increased with aneurysm diameter.⁹⁾ Klotzsch et al. used transcranial doppler to monitor embolic events after coil embolization in 35 patients with large or giant (diameter > 15 mm) aneurysms and 20 patients with small (diameter < 15 mm) aneurysms. Embolic signals were observed in 11 of the 35 patients with large or giant aneurysms. Five of the seven patients with large or giant aneurysms and subsequent TIA or stroke had postprocedure embolic signals. No embolic signals and no ischemic events were noted in the 20 patients with small aneurysms.⁹⁾

The other independent risk factor of postprocedure ischemic event was prolapse of coils into the lumen of parent or branch arteries. Previous investigators have suggested that prolapsed loops increase the risk for subsequent stroke.⁹⁾⁽¹²⁾ It is likely that these loops serve as a site for platelet aggregation, leading to local thrombosis or distal thromboembolism. It is important to note that these patients very likely received more aggressive postprocedure treatment (antiplatelet and/or anticoagulation) than patients in whom there was no coil prolapse.

Old age and prolonged procedure time were also independent factors for procedure related thromboembolism. Patients with large aneurysm diameter or prolapsed coil loops are at the greatest risk for postprocedure stroke. Aneurysms treated with GDCs may retain thromboembolic potential for up to 2 months.¹⁷⁾ Efforts at reducing the risk of stroke in patients undergoing treatment of intracranial aneurysms with GDCs should be aimed at this group.

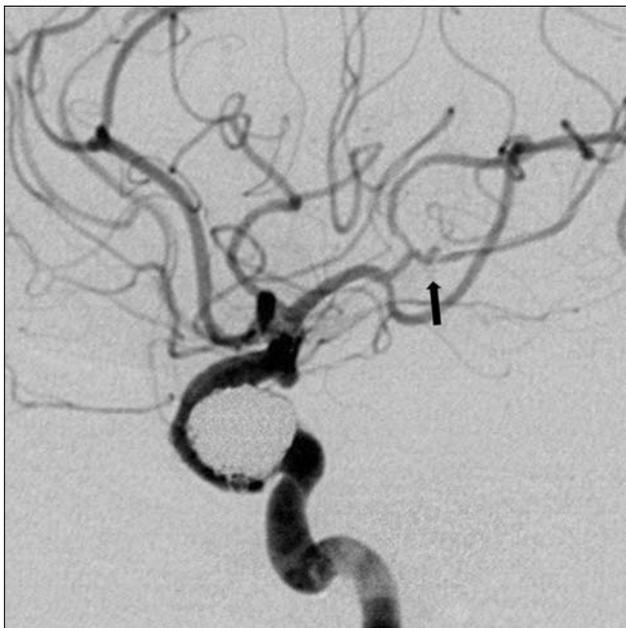


Fig. 3. Left ICA angiogram after coil embolization. The insular branch of MCA is irregular (arrows). It is assumed to be injured and focus of subarachnoid hemorrhage.

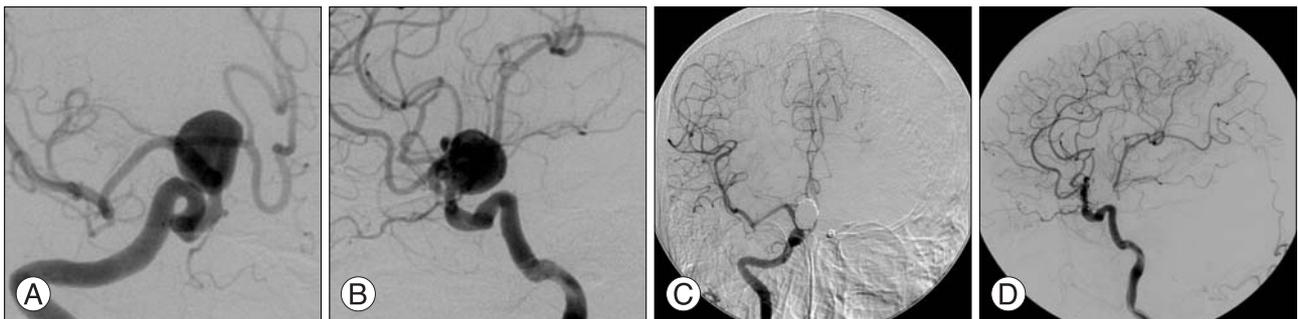


Fig. 4. Case 2, a 71-year-old woman who has an unruptured paraclinoid aneurysm. A and B, angiograms show a large paraclinoid aneurysm measured 15x11mm. C and D, angiograms show complete occlusion after coiling.

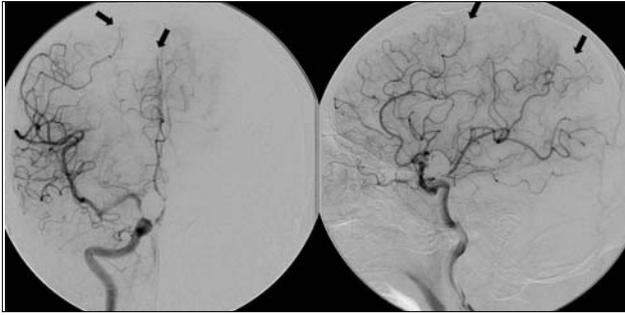


Fig. 5. Right ICA angiograms reveal occlusion of cortical branches of MCA and ACA (arrow).

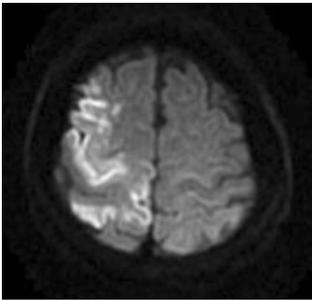


Fig. 6. MRI image on next day after embolization shows high signal intensity on right motor cortex.

Periprocedural thromboembolism can be treated with selective intra-arterial administration of thrombolytic agents (urokinase or tPA). Cronqvist et al reported the results of superselective intra-arterial fibrinolytic therapy in 19 patients to treat thromboembolic complications during endovascular aneurysm treatment. Complete recanalization was achieved in 10, and partial recanalization, in nine. Fourteen patients had a good clinical recovery, and one died of a large intracerebral hematoma.⁴⁾ Several authors have proposed the use of abciximab and tirofiban to manage acute thromboembolism during endovascular coil placement in intracranial aneurysms.¹¹⁾¹³⁾ Both of them are a chimeric monoclonal antibody fragment against the platelet glycoprotein IIb/IIIa receptor complex. These drugs may be more effective and logical for intra-procedural thromboembolism because most patients have already received full heparinization, making it unlikely that a fibrin clot is present.¹³⁾ But, intra-arterial thrombolysis cannot always be available. In case 2, cause of complication was presumed thrombus from large coiled aneurysm. On follow up DSA, neither apparent incomplete coil packing nor extrusion of the coil into the parent artery was seen just after the coil occlusion. However, thromboembolism occurred 8 hours after embolization in spite of continuous

administration of heparin. It is likely that a thrombus from coiled aneurysm migrated to the arterial lumen of the cortical branch of MCA and ACA. We tried to recanalize occluded vessels with intra-arterial urokinase and tirofiban but, didn't achieve. Therefore, the patients who have such risk factors should be administered anti-platelet drug (aspirin, clopidogrel) orally before endovascular treatment to prevent thromboembolic event.

Other serious complication is rupture of aneurysm during procedure, and delayed rupture in incompletely occluded aneurysm. However, the risk for hemorrhage appears to be low.⁷⁾¹⁶⁾ Rates of aneurysmal rupture during coil embolization are 1.9~16% for ruptured aneurysms and 0~1.3% for unruptured aneurysms.¹³⁾¹⁵⁾

Hodgson et al. described that delayed rupture of an unruptured aneurysm was managed endovascularly.⁷⁾ Horowitz et al. reported a case of an anterior communicating artery aneurysm that was occluded with coils and appeared to be stable on follow up angiography, which subsequently ruptured 23 months after initial therapy. In 2007, Ko et al described that early rebleeding occurred after incomplete coil embolization of unruptured basilar apex aneurysm. They suggest that causative factors for delayed rupture included residual aneurysmal sac due to incomplete coiling and a partially thrombosed aneurysm. And they recommended that careful follow-up is necessary in all patients with incomplete occlusion of aneurysm, even in case of unruptured one.⁷⁾⁸⁾⁹⁾

There is rarely microwire induced arterial injury. Arterial injury might also have occurred as a result of the catheter tip applying tension against the arterial wall and the catheter tip eventually eroding through the intima with the passage of time and the cardiovascular pulsatile motion. There may be a slightly elevated risk of iatrogenic dissection in interventional neuroradiologic procedures because of an increased number of vascular manipulations (eg, catheter exchanges, stent placement) and the necessity for more distal placement of catheters compared with diagnostic angiography. There may be a large number of spontaneous or traumatic dissections that are never diagnosed because they remain asymptomatic or because they produce nonspecific symptoms, such as head or neck pain³⁾.

In case 1, although intra-procedural rupture didn't occur and aneurysm was completely occluded and there was no remnant sac, massive subarachnoid hemorrhage was

developed after coil embolization. We didn't find leakage or extravasation of contrast material during procedure but, we realized stretched MCA and distorted insular branch of MCA on follow up angiography (Fig. 3). It is assumed to be induced by microwire. In addition, systemic heparinization might have a detrimental effect on damaged vessel.

Conclusion

After experiencing these two aforementioned cases, we believe although coil embolization of an intracranial aneurysm is a safe technique, high risk patient (elderly patient, large aneurysm, prolonged procedure time) should be carefully observed during procedure as well as after procedure. Also more gentle catheterization will be needed and premedication with antiplatelet agents.

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