CASE REPORT

Two cases of acute ischemic stroke associated with strut exposure in the cerebral artery lumen after stent-assisted coil embolization

Seong-Joon Lee, Hanul Park, Hong Nam Kim, So Young Park, Ji Man Hong, Jin Soo Lee

Department of Neurology, Ajou University Medical Center, Ajou University School of Medicine, Suwon, Korea

ABSTRACT

Stent-assisted coil embolization (SAC) is used for complex wide-necked aneurysms but can expose stent struts to the arterial lumen, leading to thrombosis. Herein, we report two cases of delayed thromboembolic stroke post-SAC. Case 1: A 71-year-old woman had an acute ischemic stroke 2 months after Y-stent SAC for a basilar artery aneurysm, and aspirin was prescribed post-procedure. Diffusion-weighted imaging revealed multiple scattered infarcts of various sizes in the posterior circulation. Case 2: A 72-year-old woman experienced an acute ischemic stroke 3 years post-SAC for a right posterior communicating artery aneurysm. The stroke occurred after discontinuation of anti-platelet therapy. Diffusion-weighted imaging revealed scattered acute infarctions in the right middle and anterior cerebral artery territories. These two cases of delayed thromboembolic stroke after SAC might have been due to stent strut exposure in the arterial lumen and concurrent thrombosis.

Keywords: Coil embolization; Delayed thromboembolic event; Intracranial aneurysm; Stent-assisted coiling

INTRODUCTION

Stent-assisted coil embolization (SAC) is increasingly being used to treat complex and wide-necked aneurysms. Coil embolization is a minimally invasive procedure used to treat aneurysms in which a soft coil is inserted into the aneurysm to block blood flow and reduce the risk of rupture. SAC uses a stent during the procedure. Stents provide a mechanical aid that protects against coil protrusion into the parent arteries and may act as a flow diverter, promoting intra-aneurysmal stasis and thrombosis [1]. However, acute and delayed thromboembolic events are major adverse complications of this technique [2]. Compared with coronary arteries, intracranial vessels are composed of much more acute angles, which can complicate stent placement and hinder stent adherence to the arterial endothelium. As a result, the stent struts may be exposed in the
vessel lumen, causing turbulent blood flow and possibly thromboembolism in the affected vasculature. Unlike coronary artery stent placement, following a SAC procedure, neither the duration of the initial dual antiplatelet therapy nor the risk factors for developing thromboembolic events are well characterized. Here, we discuss two cases in which the patient experienced a delayed thromboembolic stroke following SAC for cerebral artery aneurysms. The case findings support the possibility that exposed stent struts in the vessel lumen are one of the mechanisms underlying delayed thromboembolism in this population.

**CASE REPORTS**

**Case 1**
A 71-year-old woman was referred to the emergency department with an acute decrease in consciousness. She had undergone Y-stent-guided coil embolization of a basilar artery tip aneurysm 2 months before presentation, after which aspirin was prescribed to prevent thromboembolism. On neurological examination, she exhibited a drowsy mental status, left homonymous hemianopsia, right third nerve palsy, left facial palsy, dysarthria, dysphagia, and left hemiparesis. Brain magnetic resonance imaging performed upon admission revealed multiple scattered infarcts of various sizes in the bilateral occipital lobes, cerebellar hemispheres, right pontine, right midbrain, and right thalamus (Fig. 1A). No evidence of hemorrhage exists on the gradient echo images. The patient’s SAC was visualized on digital subtraction angiography, and an intraluminal filling defect was observed in the right P1 and P2 portions, where the stent struts were exposed in the artery lumen (Fig. 1B, C). The results of the additional laboratory tests and cardiovascular evaluations were unremarkable. The patient was admitted to the stroke unit and was administered aspirin, clopidogrel, and argatroban. After the patient’s neurological symptoms stabilized, she was transferred for rehabilitation. Aspirin, ticlopidine (because of clopidogrel unresponsiveness), and cilostazol were prescribed as preventive therapies.

**Case 2**
A 72-year-old woman presented with left-sided weakness 2 days after open reduction and internal fixation surgery for a traumatic left patellar fracture. The patient had a history of

![Image](https://doi.org/10.23838/pfm.2024.00065)
Stroke originated from coil stent diabetes mellitus, hypertension, and dyslipidemia. Additionally, she had undergone a SAC procedure to treat a ruptured aneurysm in her right posterior communicating artery 3 years prior. To ensure surgical hemostasis, antiplatelet drugs such as aspirin and clopidogrel were discontinued before the procedure. A neurological examination revealed mild left hemiparesis without other neurological deficits. Brain magnetic resonance imaging revealed acute scattered infarctions in the right middle and anterior cerebral artery territories without evidence of hemorrhage (Fig. 2A). Visualization of the SAC with computed tomographic volume-rendering angiographic images and digital subtraction angiography revealed segmental luminal narrowing in the right supraclinoid internal carotid artery, where the stent was placed, and a portion of the stent strut was exposed in the parent artery (Fig. 2B-D).

The results of the additional laboratory tests and cardiovascular evaluations were unremarkable. The patient was admitted to the stroke unit, and dual antiplatelet therapy was re-administered. After the patient’s neurological symptoms stabilized, she was transferred for rehabilitation.

As this was a retrospective case report, consents were not obtained.

**DISCUSSION**

Here, we report two cases of delayed thromboembolic stroke after SAC, which may have been due to exposed stent struts in the arterial lumen and concurrent thrombosis. Following SAC, periprocedural thromboembolic complications have been reported in 11.2% of patients [3]. In unruptured aneurysms, although periprocedural complications are less common [4], delayed thromboembolic events have been reported to occur in 7.9% of patients during a mean follow-up of 32.4 months [5]. Short-term dual antiplatelet therapy and long-term maintenance therapy with a single agent are recommended to prevent these complications. Specifically, short-term use (6 weeks to 3 months) of initial dual antiplatelet therapy and a subsequent switch to monotherapy is generally accepted [6-8]. However, while delayed ischemic stroke events have been shown to occur in 3.5% of patients following this regimen, in a mean follow-up of 54 months, no events were observed in patients who made a later switch (\( \geq 9 \) months) from dual antiplatelet therapy to monotherapy, indicating that a longer duration of dual antiplatelet therapy should be prescribed [9].
Additionally, to develop appropriate peri- and postprocedural preventive measures for thromboembolic events following SAC, risk factors need to be established. Previous data suggest that ischemic events are more likely to occur soon after stent deployment (within 40 days) [10] and are associated with clopidogrel discontinuation [11], clopidogrel resistance [12], smoking, and large parental artery diameters [5]. The two cases in our report did not occur in the 40 days; however, both cases were associated with antiplatelet withdrawal, emphasizing the importance of strict adherence to antiplatelet prescriptions even after this period.

Furthermore, it has been identified that incomplete stent apposition due to vascular tortuosity can be an additional risk factor. In these cases, the gap between the stent and vessel acts as a hub for thromboembolism and rarely resolves spontaneously [13]. Similarly, the exposure of stent struts in the arterial lumen may cause stasis or turbulence, increasing the risk of thromboembolism. We consider the thromboembolism in our cases to be due to such mechanisms, for in case 1, the Y-stent created exposed struts in the terminal portion of the basilar artery where the posterior cerebral artery lumens begin, while in case 2, tortuosity of the distal parent artery caused luminal exposure of stent struts. Thus, these patients may require more attention when reducing or discontinuing antiplatelet therapy, even if SAC has not been recently performed. For example, in case 2, a thromboembolic event occurred after antiplatelet drugs were discontinued three years after stent placement.

In conclusion, stent strut exposure in the arterial lumen may be a risk factor for delayed thromboembolic stroke after SAC. Long-term dual antiplatelet therapy and routine screening for antiplatelet resistance are warranted in such patients.

CONFLICTS OF INTEREST

No potential conflict of interest relevant to this article was reported.

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ORCID

Seong-Joon Lee https://orcid.org/0000-0001-9735-6369
Hanul Park https://orcid.org/0009-0005-9474-4725
Hong Nam Kim https://orcid.org/0000-0002-7064-9409
So Young Park https://orcid.org/0000-0002-8625-6968
Ji Man Hong https://orcid.org/0000-0001-6803-1207
Jin Soo Lee https://orcid.org/0000-0002-7225-6166

AUTHOR CONTRIBUTIONS

Conception or design: JSL.
Acquisition, analysis, or interpretation of data: SJL, HP, JSL.
Drafting the work or revising: SJL, HNK, SYP, JMH, JSL.
Final approval of the manuscript: JSL.

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