A Case of Suspected Metallic Embolism after Carotid Artery Stenting

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A case in which metallic embolism was suspected after carotid artery stenting (CAS) is described. A 79-year-old woman was referred to our hospital because of a severe stenosis of the left cervical internal carotid artery (ICA). Carotid ultrasound revealed that the plaque was fibrous and was accompanied with partial calcification. The carotid stenosis was treated by CAS. The magnetic resonance imaging (MRI) taken in the following day of the CAS demonstrated that a new abnormal spot at the left frontal lobe. The spot appeared as a signal void on T1, T2, diffusion, susceptibility-weighted image (SWI), and fluid attenuated inversion recovery (FLAIR) image, and was surrounded by a high-signal halo on T2 and diffusion-weighted images (DWIs). The spot also demonstrated “blooming” appearance on SWIs. Despite the lesion she was asymptomatic all through the postoperative course, and she left our hospital on postoperative day 6. Follow-up MRI obtained 27 months after the CAS demonstrated that the lesion remained at the left frontal lobe without any signal changes. The patient remained asymptomatic at the last follow-up. Considering the location of the new abnormal spot (in the vascular territory of the catheterized vessel), these imaging characteristics and asymptomatic clinical course, the spot likely suggested metallic embolism. This is the first case in which the metallic embolism was suspected after CAS.

Keywords: metallic embolism, carotid artery stenting, magnetic resonance imaging, asymptomatic embolism

Introduction

Clinically asymptomatic emboli are well-known complications after cerebral angiography and neuroendovascular surgery, of which incidence was reported to be approximately 20% of the cases.³ Although foreign body emboli are a less common complication after endovascular procedure, its frequency is increasing as neuroendovascular surgery becomes popular.² Previously, we reported abnormal signals suggesting metallic emboli on the postprocedural magnetic resonance imaging (MRI) in several cases of endovascular coil embolization for cerebral aneurysms.¹ In this report, we describe a case accompanied with the similar MRI findings following carotid artery stenting (CAS). This is the first case in which the metallic embolism was suspected after CAS.

Case Presentation

Before writing this case report, a written informed consent was obtained from the patient.

A 79-year-old woman was referred to our hospital because of severe stenosis of her left cervical internal carotid artery (ICA) that was incidentally found in screening carotid ultrasound for diabetes. Besides the diabetes, she had a history of hypertension and dyslipidemia. Carotid ultrasound revealed fibrous plaque accompanied with partial calcification at the origin of the left ICA and increased peak systolic velocity of 418 cm/s. Magnetic resonance angiography (MRA) demonstrated a decreased signal of the left intracranial ICA due to the severe stenosis.

MRI did not show any old cerebral infarction, ischemic changes, nor microbleeds. The left carotid angiogram revealed that the degree of the stenosis was 90% by the North American Symptomatic Carotid Endarterectomy Trial⁴ (Fig. 1A), and that the stenosis caused the prolonged cerebral circulation time in the left cerebral hemisphere. For the severe carotid stenosis, CAS was planned.

CAS procedure

Under local anesthesia, a 9 French (F) and a 4F sheath were placed in the right common femoral artery and the left common femoral vein, respectively. Then, 4000 units of heparin were intravenously injected for systemic heparinization to maintain activated clotting time over 250 seconds. A 9F 90cm Optimo (Tokai Medical Products, Aichi, Japan) was advanced to her left common carotid artery using a coaxial system of a 4-5-6F 125cm JB2 (Medikit, Tokyo, Japan) and a 0.035-inch 200cm Radifocus (Terumo, Tokyo, Japan). A GuardWire PS (Medtronic, Dublin, Ireland) was then guided to the left external carotid artery and flow reversal was established by inflating both the Optimo and the Guard-Wire.⁵ The Optimo and the 4F sheath were connected using a filter chamber so that regurgitated blood from the left carotid artery could return to the left common femoral vein. Under the flow reversal, a FilterWire EZ (Boston Scientific, Marlborough, MA, USA) was advanced toward the distal ICA. However, the lesion of the left cervical ICA was too narrow for the FilterWire EZ to pass. Therefore, the lesion was pre-dilated using a SHIDEN (2.5 mm × 40 mm; Kaneka Medix, Osaka, Japan) and the FilterWire EZ could finally advance to the distal ICA. An intravascular ultrasound (IVUS) catheter (Eagle Eye Gold; Volcano, San Diego, CA, USA)

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³Received: January 24, 2020; Accepted: March 30, 2020
Online September 17, 2020

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revealed an iso-echoic plaque. A Precise (8 mm × 40 mm; Cordis, Miami Lakes, FL, USA) was placed, and then an Rx-Genity (3.5 mm × 20 mm; Kaneka Medix) was used for post-dilatation at nominal pressure of 8 atm for 30 seconds. The IVUS revealed no obvious in-stent plaque protrusion and well deployment of the stent. No breakage was observed for the retrieved devices. Final left carotid angiogram demonstrated a well-dilated lesion (Fig. 1B) and significant improvement of cerebral circulation in the left cerebral hemisphere.

The corn-beam CT obtained just after the CAS detected no abnormal findings.

The MRI performed on the following day of the CAS showed a new abnormal spot at the left frontal lobe, which

![Fig. 1](image-url)
was undetectable on any of the preoperative MRI sequences including SWIs. The abnormal spot appeared as a signal void on T1, T2, diffusion, fluid attenuated inversion recovery (FLAIR), and SWI (Figs. 1C–1G), and it was surrounded by a high-signal halo on T2, DWI, and FLAIR image (Figs. 1D–1F) and demonstrated “blooming” appearance on SWI (Fig. G). Despite the lesion she was asymptomatic all through the postoperative course, and she left our hospital on postoperative day 6.

Follow-up MRI obtained 27 months after the CAS demonstrated that the lesion remained at the left frontal lobe without any signal changes. The patient remained asymptomatic at the last follow-up.

Discussion

Because the abnormal signal spot was newly found just after the endovascular surgery and was in the vascular territory of the catheterized vessel, it was likely that the lesion developed due to embolic phenomena.

Various potential embolic sources can be considered such as thrombus, air, calcified plaque, or iatrogenic foreign body. We believe that the abnormal signal spot represented some metal because the MRI findings of the lesion included no MRI signals resulting in a signal void, a susceptibility artifact resulting in the high-intense halo and the “blooming” appearance, and no signal changes over time, and these characteristics completely consisted with those of cases that we previously reported as suspected metallic embolisms after endovascular coil embolization for cerebral aneurysms.3)

Besides embolism, hemosiderin deposition might be considered because microbleeds were reported to occur after CAS in approximately 10% of the cases6) and the abnormal spot was of low intensity in all the images. However, this was unlikely because the surrounding halo on T2 and DWIs and the “blooming” effect on SWI were inconsistent with hemosiderin. Some metallic embolism may have been misunderstood as microbleeds, and others may have been missed because in not all the CAS cases MRI, especially T2* or SWI, has been routinely obtained preoperatively and postoperatively.

The patient’s clinical course also supports the inference that the abnormal signal spot might represent a metallic embolism. While an embolism due to a hydrophilic coating of interventional tools (e.g., polyvinylpyrrolidone) should cause various clinical symptoms such as headache or convolution accompanied with dynamic changes of neuroimaging findings,8,9) patients with metallic embolisms were asymptomatic all through the clinical course.3)

There are several limitations to this report. The first limitation is lack of histopathological corroboration. Because the patient was asymptomatic, it was not reasonable to perform a brain biopsy. Given that only microscopic metal fragments are required to produce artifacts on MRI10) and that cerebral infarction was not observed in the patient, it was likely not that a cerebral artery was embolized by a metal fragment but that just a metal powder got lodged on the vessel wall. Therefore, the term “metallic deposition” rather than “metallic embolism” may be appropriate. Second, similarly to the previous aneurysm series, it was unclear which device caused the abnormal signal spot. Neither special devices nor techniques were used during the CAS procedure in this case. The common devices that were used in both the present CAS case and the previous aneurysm cases with suspected metallic embolism were the 0.035-inch Radifocus employed in all the aneurysm cases and the 4.5-6F JB2 catheter employed to navigate a guiding catheter in some of the aneurysm cases. It is, however, unlikely that the 0.035-inch guidewire is responsible because this device has been used in almost all the endovascular procedures as well as diagnostic cerebral angiography, and no suspected metallic embolism has been detected after these procedures besides a part of cases of CAS and aneurysm coil embolization. The JB2 catheter is also unlikely for the same reason. During the CAS in this case, a certain device might have been scratched by the carotid plaque and engendered the metallic embolism. However, further investigation is warranted to clarify the mechanisms.

Conclusion

This is the first case in which the MRI findings suggesting a metallic embolism was observed after CAS.

Conflicts of Interest Disclosure

All the authors have no conflicts of interest.

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