

Carotid Artery Stenting Successfully Prevents Progressive Stroke Due to Mobile Plaque

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Key Words

Carotid artery stenting · Mobile plaque · Stroke in evolution

Abstract

We report a case of progressive ischemic stroke due to a mobile plaque, in which carotid artery stenting successfully prevented further infarctions. A 78-year-old man developed acute multiple infarcts in the right hemisphere, and a duplex ultrasound showed a mobile plaque involving the bifurcation of the left common carotid artery. Maximal medical therapy failed to prevent further infarcts, and the number of infarcts increased with his neurological deterioration. Our present case suggests that the deployment of a closed-cell stent is effective to prevent the progression of the ischemic stroke due to the mobile plaque. © 2015 S. Karger AG, Basel

Introduction

A mobile plaque (MP) in the carotid artery is an uncommon condition which can be detected with duplex ultrasound, and it is associated with a high risk of ischemic stroke [1]. Medical treatment alone is sometimes not enough to treat ischemic strokes due to MP [2]. Carotid endarterectomy (CEA) is an option for patients who are refractory to medical treatment; however, the risks of intraoperative embolization and general anesthesia should be considered carefully. Here, we report a case of ischemic stroke in evolution due to MP in which carotid artery stenting (CAS) successfully prevented further infarctions.

Case Report

A 78-year-old man was admitted for a nonketotic hyperosmolar coma. He had been treated for diabetes mellitus, chronic renal and heart failure. After admission, his comatose state had improved by treating him with insulin for several days. He was then able to walk by himself. One month after admission, he suddenly developed right hemiparesis, and an MRI disclosed acute multiple infarcts in the left hemisphere (fig. 1a–c). An MRA failed to show any lesions in the intracranial arteries. A carotid ultrasound disclosed an MP located at the bifurcation of the left common carotid artery (fig. 2a–c; online suppl. video), and the MP was considered to be the cause of the ischemic lesions. A maximal medical therapy consisting of heparin, statin, and cilostazol had been undertaken; however, the number of infarcts had increased along with his neurological deteriorations (fig. 1d–f). As the antithrombotic therapy was not effective to treat progressive stroke, CEA was considered. However, ²⁰¹Tl scintigraphy suggested a cardiac ischemia involving the anterior and inferior walls, making it difficult to induce general anesthesia. Thus, we finally decided to perform CAS.

Operating Procedure

A total of 6,000 U of heparin was infused to maintain anticoagulation. A 9-Fr sheath was placed in the right femoral artery. We used a telescoping technique to advance a guide catheter (9-F Optimo; Tokai Medical, Kasugai, Japan). A 0.038-inch wire was parked in the left common carotid artery (CCA) just proximal to the MP; however, a 5-Fr Simmons diagnostic catheter could not track the wire. Then, under roadmap guidance, a 0.038-inch wire was navigated carefully to the left external carotid artery (ECA) while paying attention not to touch the MP. The 5-Fr Simmons catheter could be advanced over the 0.038-inch wire into the left ECA, followed by tracking along the guide catheter into the distal CCA with a telescoping technique. We inflated the balloon of the guide catheter to obtain the control of the proximal flow. Although ECA was not blocked, favorable flow reversal was archived only by blocking the CCA. During this proximal flow control, spider FX (ev3, Plymouth, Minn., USA) along with a microguidewire was advanced to the internal carotid artery (ICA), and once the spider was deployed, the balloon of the guide catheter was deflated. Carotidwall Wallstent (Stryker, Mass., USA) was deployed in the ICA down to the CCA (fig. 3). Intravascular ultrasound after stenting did not show any MP, and the procedure was completed after the retrieval of spider FX.

No new infarcts were detected after stenting. Postoperative carotid ultrasound clearly showed the disappearance of the MP in the stented CCA and ICA (fig. 4a, b). There was an MP noted in the left ECA which was located outside the stented vessel (fig. 4a). One month later, the MP in the ECA could not be observed by ultrasound. The patient died after 130 days in the hospital because of pneumatosis cystoides intestinalis. A postmortem examination disclosed the patency of the stented carotid artery, and no plaque had prolapsed into the stent (fig. 4c).

Discussion

MP in the carotid artery is a rare condition observed by carotid ultrasound, showing mobile components in a carotid plaque [1]. These types of plaque with mobility have been described as ‘mobile plaque’, ‘floating thrombus’, ‘free floating thrombus’, or ‘mobile thrombus’. The nature of ischemic strokes due to an MP has not been elucidated well because of the small number of reported cases.

Recently, Delgado et al. [2] reviewed 22 reported cases of MP including 3 cases of their own; only 7 out of the 22 patients were treated solely with antithrombotic therapy, and the remaining 13 patients were treated with surgical interventions. Antithrombotic therapy alone seems to be insufficient to treat the ischemic stroke due to MP. Funaki et al. [3] studied 21 MPs removed by CEA, and reported that progressive ischemic strokes were more frequent in patients with MP. According to their histological analysis, the ratio of the necrotic core compared to that of the entire plaque was significantly larger than those in non-mobile plaques [3]. Thus, MP is considered to be more embologenic than non-MP.

For patients with MP in which maximal medical therapy is not effective, the surgical removal of an MP (CEA) or coverage of an MP (CAS) should be considered. CEA surgically removes an MP, and it seems to be the ideal treatment; however, requirements for general anesthesia sometimes make it difficult to perform CEA.

When CAS has to be conducted, a large-sized guide catheter has to be placed in the CCA. Usually, a 0.035-inch wire is advanced to the ipsilateral ECA in order to provide enough support for the tracking of the guide catheter. In patients with MP, toughing of the MP with the wire should be avoided when a guide catheter is placed. In our case, a 0.038-inch wire was placed distal to the CCA; however, the guide catheter could not be advanced along the wire. The advancement of a 0.038-inch wire to the ECA was needed for the placement of a guide catheter to the CCA. Iwata et al. [4] reported 62 cases of successful transbrachial CAS using a novel sheath guide (MSK-guide; Medikit, Tokyo, Japan). The system did not require the advancement of wires to the ECA, and the use of this system is considered to be effective in patients with MP; however, a flow reversal cannot be achieved with it.

The use of embolic protection devices is generally undertaken in the CAS procedure. There are 2 types of protection: distal and proximal. Distal protection is achieved by placing a filter or balloon to block the debris. Proximal protection, which is known as the Parodi anti-embolic system, is achieved by establishing a reverse flow in the ICA by blocking the flow of the CCA and ECA [5]. In distal protection, the devices have to pass a stenosis without any protection. This passage of the protective devices could be risky in patients with MP. Proximal protection can overcome this drawback; however, the patients should be tolerated to flow reversal, and the injection of contrast medium during the procedure is not allowed, resulting in a difficulty in stent deployment. In our case, a filter wire passed the lesion under proximal flow control. Once the filter was deployed, distal protection was undertaken. After the procedure, no ischemic lesions were noted.

There are 2 types of stent design: open- and closed-cell. Open-cell stents have a better conformability compared to closed-cell stents. They are easy to deploy even in tortuous vessels. However, the free cell area of closed-cell stents is smaller than those of open-cell stents. The use of closed-cell stents with a smaller free cell area is expected to cover a plaque more efficiently than open-cell stents [6]. Histological studies suggest that MP is more embologenic than non-MP, and the use of closed-cell stents should be encouraged in terms of plaque coverage.

To our knowledge, 2 cases of MP in which CAS was undertaken have been reported [7]. Both cases were asymptomatic, and this is the first reported case of successful CAS in a patient with stroke in evolution due to MP.

In conclusion, this present case suggests that CAS can prevent ischemic stroke in evolution. However, the accumulation of cases is required to elucidate the nature of CAS for MP in terms of prevention of ischemic stroke.

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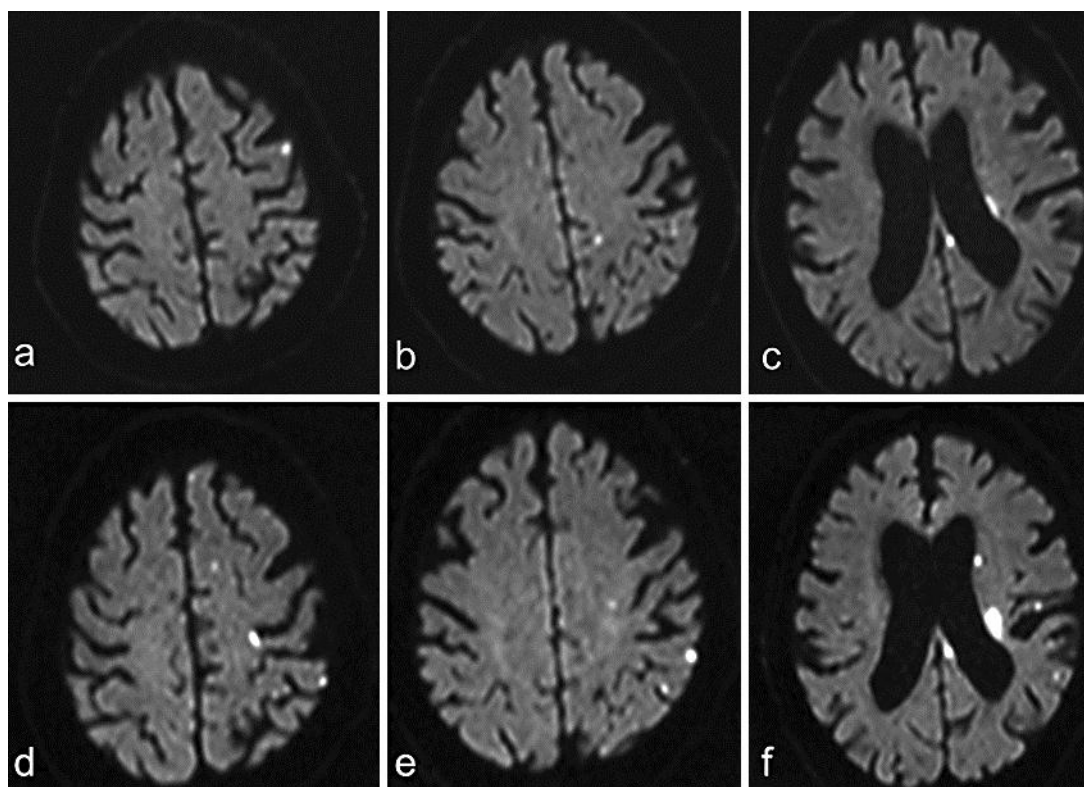


Fig. 1. Diffusion-weighted MRI 1 month after admission showed multiple acute infarcts in the left hemisphere (a–c). After 2 weeks, the number of acute infarcts increased, even though maximal medical therapy was undertaken (d–f).

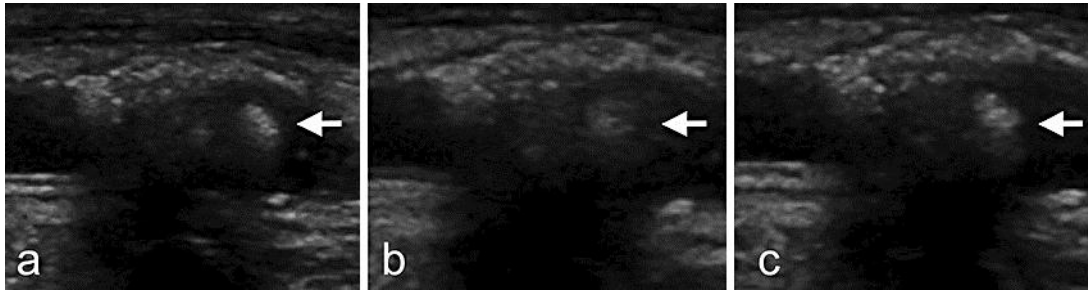


Fig. 2. A preoperative longitudinal view of B-mode carotid ultrasound showing an MP located in the bifurcation of the left common carotid artery, which moves in sequence during systole (a–c, arrow).



Fig. 3. Preprocedural left carotid angiogram showing an MP at the left carotid artery (a, arrow). After the stent had been deployed, the MP disappeared (b).

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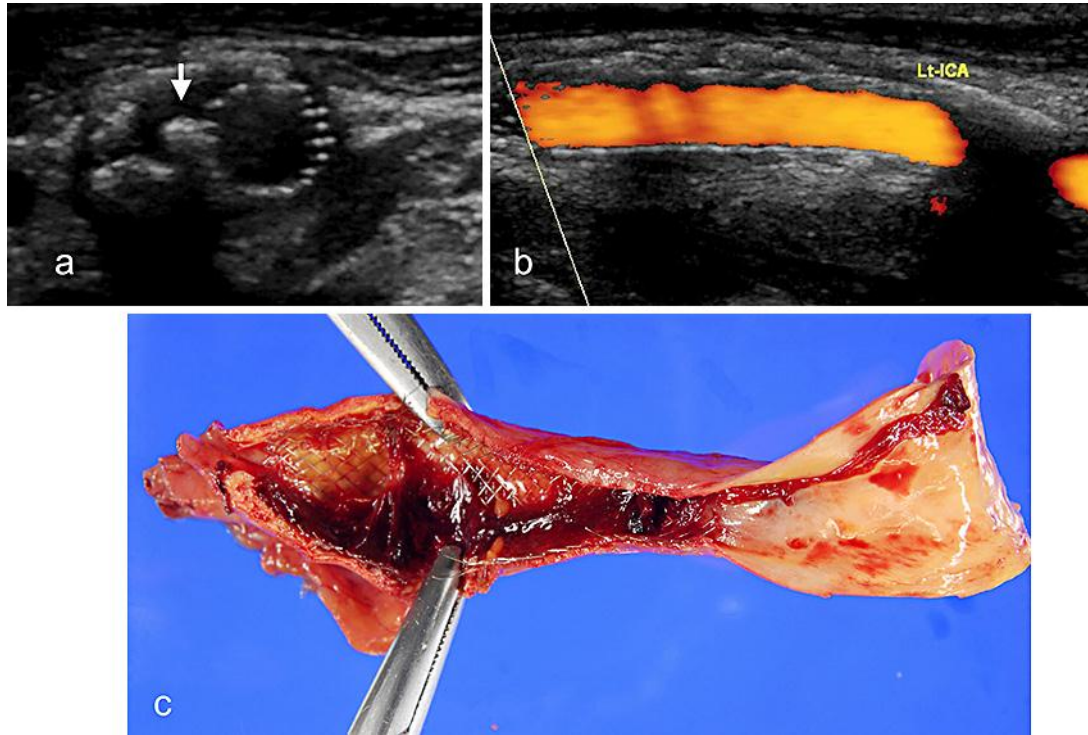


Fig. 4. A postoperative ultrasound showing no MP noted in the stented artery (**a**: transverse view, **b**: longitudinal view). A small residual MP was observed in the left ECA, which was outside of the stented carotid artery (**a**, arrow). A postmortem examination showed the patency of the stented left carotid artery (**c**).