Delayed deformation of self-expanding stents after carotid artery stenting for postendarterectomy restenoses

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Carotid artery stenting has become an acceptable alternative for treating patients with severe atherosclerotic lesions, particularly those with significant surgical risks, such as recurrent stenosis after endarterectomy. Nonetheless, we herein report two patients who presented with delayed Wallstent deformation after carotid artery stenting for postendarterectomy restenosis. Our cases highlight the need for caution because delayed deformation of self-expanding stents can occur, particularly during treatment of patients with postendarterectomy stenosis. Furthermore, poststent surveillance is imperative in identifying patients with severe restenosis after carotid artery stenting who need reintervention. (J Vasc Surg 2006;44:412-5.)

Since DeBakey performed the first successful carotid artery endarterectomy (CEA) in 1953, CEA has become the gold standard for treating patients with high-grade carotid stenoses. However, this technique is not without complications: infection, stroke, hemorrhage, and cranial nerve injuries have been reported after CEA. Furthermore, increased risks have been demonstrated in the subgroups of patients with significant medical comorbidities and difficult surgical anatomies, such as recurrent stenosis after endarterectomy, prior neck irradiation, and inaccessible lesions above the C2 level. Therefore, carotid artery stenting (CAS) has evolved as a valuable alternative to CEA, particularly in high-risk patients. Although the field of carotid stenting is rapidly advancing and its efficacy has been proven in multiple clinical trials, we herein relate the need for caution when treating post-CEA restenosis, because deformation of self-expanding Wallstents (Boston Scientific, Natick, Mass) occurred in two of our patients.

CASE REPORTS

Patient 1. A 64-year-old man with a history of right CEA with patch angioplasty for a high-grade carotid artery stenosis presented with recurrent symptoms 6 months later. Duplex ultrasonography revealed severe recurrent stenosis, which was successfully treated with a Wallstent placement. A completion angiogram showed a resolution of stenosis with a brisk antegrade flow. However, increased internal carotid artery (ICA) velocities with a significant color-flow disturbance consistent with significant in-stent restenosis were detected 3 and 6 months later on follow-up duplex ultrasound scans (369 peak systolic/147 end diastolic cm/s and 513 peak systolic/151 end diastolic cm/s, respectively). Each stenosis was confirmed by an angiogram (Fig 1, A) and was respectively treated with a cutting balloon angioplasty at 3 months and a cutting balloon angioplasty with, subsequently, a second Wallstent placement at 6 months. The patient again presented with several episodes of right hemisphere transient ischemic attacks 3 months afterward. An ultrasound evaluation revealed an increased peak systolic velocity of 434 cm/s, an end-diastolic velocity of 212 cm/s, and a severe color flow disturbance indicating a high-grade stenosis, which was confirmed by an angiogram (Fig 1, B). The angiogram also showed a deformed second Wallstent with a dilated proximal portion against the patched carotid bulb and a significant narrowing of the distal end located within the first stent that was flow limiting. We therefore decided on an endovascular approach in treating this lesion.

After administration of intravenous bivalirudin, the right carotid artery was cannulated. A 7F, 90-cm carotid guiding sheath (Boston Scientific) was placed in the mid common carotid artery by tracking over a 0.035-inch stiff GlideWire (Boston Scientific). A selective digital carotid angiogram via the side port of the guiding sheath confirmed the severe stenosis due to stent deformation and a patent intracranial ICA. A FilterWire (Boston Scientific) embolization protection device was then manipulated to cross the lesion. After activation of the embolization protection device, a third Wallstent was deployed across the stenosis and dilated with a 5-mm-diameter angioplasty balloon. With satisfactory angiographic results, the embolization protection device was then captured and the shuttle sheath subsequently removed (Fig 1, C). The patient was discharged home the next day after an uneventful hospital stay. He remains symptom free 6 months later with good stent apposition.

Patient 2. A 72-year-old man underwent left CEA with a patch angioplasty for symptomatic carotid artery stenosis 4 years ago. The patient presented with amaurosis fugax and recurrent high-grade stenosis at the proximal end of the patch (Fig 2, A) that required a repeat CEA 1 year later and subsequent CAS with a
Wallstent 3 years later (Fig 2, B). He again presented with symptoms of transient ischemic attack 1 year after the CAS with an ultrasound scan indicating severe stenosis (velocity ratio of ICA/common carotid artery >4). A magnetic resonance image illustrated a high-grade recurrent stenosis, and a carotid angiogram showed a deformed stent with a widened middle portion apposed to the patched carotid bulb and a significantly narrowed distal end located in the native ICA, which substantially limited ICA blood flow (Fig 3, A). To relieve his symptoms and repair the deformed stent, an endovascular approach was chosen.

As described previously, after systemic anticoagulation was initiated, a guidewire was used to cannulate the left carotid artery. However, the tip of the wire was bounced against the extremely narrowed stent opening, thus forming a J-shaped curve, which potentially protected the wire from penetrating through the struts of the stent. A selective catheter was then advanced over the wire to the narrowed stent opening, and the wire was carefully manipulated through the opening. Next, the distal end of the stent was dilated with an angioplasty balloon, followed by a short Express stent (Boston Scientific) for further support. The completion angiogram showed a patent stent with a brisk antegrade flow (Fig 3, B). The patient recovered well from the procedure and remained symptom free 1 year later. Furthermore, the stent was patent on a follow-up ultrasound evaluation.

**DISCUSSION**

CAS has become an acceptable alternative in treating high-grade carotid artery stenoses, particularly in high-risk patients. Stent deformation, a phenomenon primarily associated with balloon-expandable stents, often results in thrombosis and symptom recurrence. Therefore, self-expanding stents with increased flexibility and a radial expanding force have been exclusively used for treating carotid artery stenosis to avoid the occurrence of stent deformation. However, the cases described herein emphasize the potential risk of deformation of self-expanding stents, particularly in treat-
or substantial cardiopulmonary morbidities. There are sis, an irradiated neck, surgically inaccessible high lesions, with high surgical risks, such as postendarterectomy steno-

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tance of poststent surveillance in identifying the patients who require reintervention.

CEA, currently the gold standard for treating symp-
tomatic and asymptomatic carotid artery stenosis, was first introduced in the 1950s. Since then, its efficacy has been demonstrated by numerous clinical trials. However, post-CEA restenosis with more than 50% luminal reduction has been documented at rates of 7% to 22% according to duplex ultrasonography. Therefore, a patch angioplasty in combination with CEA has been widely adopted for potentially improving surgical outcomes, as demonstrated in several clinical investigations. Theoretically, a patch angioplasty eliminates an increased cross-sectional diameter, thereby reducing the rate of restenosis. However, as demonstrated in patient 2, the oversized patch causes a significant size mismatch between the native ICA and the patched carotid bulb, and this raises challenges for future carotid stent selection and placement.

Despite the proven efficacy of CEA, significant cardiopulmonary morbidities and difficult surgical anatomies may increase procedure-related complications in a subset of patients undergoing CEA. Thus, CAS is advocated as a valuable alternative to CEA, particularly among patients with high surgical risks, such as postendarterectomy steno-
sis, an irradiated neck, surgically inaccessible high lesions, or substantial cardiopulmonary morbidities. There are still several ongoing multicenter prospective trials evaluating the efficacy of CAS. Meanwhile, the technology continues to evolve. The first generation of carotid stents involved balloon-expandable stents. The Palmaz (Cordis, Johnson & Johnson International Systems, Warren, NJ) stents have the advantage of accurate placement but have decreased longitudinal flexibility and are plagued by reports of stent deformation secondary to extrinsic compression of the cervical carotid arteries that are not protected by bony structures. Carotid stenting then moved to self-

expanding stents, which improved flexibility and the radial expansion force, thereby increasing the resistance to defor-
mation. However, our cases, for the first time, to our knowledge, demonstrate the deformation of self-expanding stents (namely, Wallstents) in the treatment of carotid artery stenosis in patients with post-CEA restenosis.

Among several theories, we speculate that a large luminal diameter mismatch, decreased compliance of the artery after surgical intervention, and specific characteristics of the stainless-steel Wallstent may encourage the deformation. Both of our patients received carotid stenting for post-CEA restenosis, with the proximal ends of the stents placed within the patched common carotid arteries. A large cross-

section diameter discrepancy between a small ICA and a large patched carotid bulb potentially causes an uneven expansion of the stainless-steel stent, which can store elastic energy. Theoretically, a stent that expands to a markedly larger cross-sectional diameter at one end and a smaller expansion at the other end does not have the same stability as a stent that undergoes a uniform expansion. In addition, the outward force of the expansion at the proximal end of the larger diameter may induce an opposite inward force at the smaller cross section of the distal end (because metal generally transmits force evenly across its surface), thereby leading to a Wallstent deformation and shortening. Furthermore, poor compliance of operated carotid arteries due to a surgery-induced fibrosis formation may further limit the ability of the stent to expand. Finally, both of our patients had aggressive myointimal hyperplasia, as demonstrated by significant restenosis shortly after CEA and CAS. This malignant myointimal hyperplasia may also contribute to stent deformation by generating inward force at the site of stenosis.

In conclusion, the deformation of self-expanding stents is a rare complication in carotid stenting procedures. Although we observed this phenomenon only with Wallstent, this complication may occur with other self-expanding stents in treating post-CEA stenosis. Our cases emphasize the need for caution during stenting of postsurgical carotid arteries with marked diameter mismatches and underline the need for diligent post-CAS surveillance in identifying patients who require reintervention.

REFERENCES


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