Use of Intravascular Stents in the Treatment of Internal Carotid and Extracranial Vertebral Artery Pseudoaneurysms

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Summary: The management of extracranial carotid or vertebral artery pseudoaneurysms is controversial. Although some of these lesions resolve spontaneously, many clinicians opt to treat them with trapping procedures that result in vessel sacrifice. We describe two cases in which an intravascular stent was used to obliterate an aneurysm of the extracranial vertebral artery and the internal carotid artery, respectively, while maintaining the patency of the parent vessel. This technique, which has been successful in experimental animal models, shows promise for application in humans.

Index terms: Aneurysm, therapeutic blockade; Interventional instruments, stents

Arterial dissections may occur spontaneously or be associated with traumatic injuries. Traumatic dissections are generally extracranial and result from penetrating or blunt injury, spinal subluxation or fracture, or irritating rotational neck movements that appear innocuous. Spontaneous dissections may occur intracranially or extracranially. If the lesion occurs between the intima and media, luminal narrowing and vessel occlusion may develop. If the dissection occurs between the media and adventitia, an aneurysmal defect may appear. Signs and symptoms of dissection include headache, neck pain, ischemic events, and hemorrhage. Treatment may consist of watchful waiting in the case of asymptomatic lesions, anticoagulation for asymptomatic or symptomatic lesions, and, depending on the physician, vessel ligation with or without bypass for lesions that fail to resolve or that remain symptomatic despite anticoagulant therapy.

In 1994, Geremia et al (1) used intravascular stents placed across the lumen of experimentally created side wall aneurysms in dogs to induce thrombosis in the sac while maintaining parent vessel patency. Also in 1994, Marks et al (2) published a report of two human internal carotid artery pseudoaneurysms that were successfully treated with the use of intravascular stents. We report the human application of intravascular stents in the treatment of pseudoaneurysms of the internal carotid and vertebral arteries.

Case 1

A 50-year-old right-handed man suffered a fall in June 1994. Six months later he underwent an anterior cervical disectomy and fusion at C5-6 that was performed through a right-sided incision. Despite excellent postoperative alignment and decompression, pain in the left arm persisted. A cervical magnetic resonance imaging study suggested the presence of a left-sided vertebral artery aneurysm at C-7. Angiography confirmed the presence of an ovoid-shaped pseudoaneurysm at the level of C-7. Results of a neurologic examination were normal, although the patient reported subjective paresthesias and pain in the left C6-7 distribution and episodes of disequilibrium. Medical therapy was begun with 650 mg per day of aspirin, and the patient was referred for obliteration of the aneurysm. Repeat arteriography (8 months after the injury) with 30-minute temporary balloon occlusion of the left vertebral artery was performed (3). The patient passed, and the decision was made to attempt treatment of the aneurysm with a stent (1). Although vertebral artery sacrifice was an option, extensive discussion with the patient regarding the option of vertebral sacrifice versus an experimental therapy geared toward vertebral artery preservation revealed a strong preference for stent placement. Permission to do the procedure was obtained from our Institutional Review Board, and 2 weeks later the patient returned for stent placement. The vertebral arteriogram, however, revealed an apparent reduction in the aneurysm's size. We were hopeful that the lesion would resolve on its own, and the procedure was cancelled. Plans were made to perform a
Fig 1. Fifty-year-old man with an aneurysm of the left vertebral artery.
A, Digital subtraction angiogram of the left vertebral artery shows a cervical extracranial aneurysm.
B, The expanded stent is seen straddling the aneurysm. The balloon is collapsed, and the balloon tip markers can be seen proximal and distal to the stent margins.
C, Digital subtraction subclavian artery angiogram after stent placement and balloon withdrawal shows that the vertebral artery aneurysm that was visible in A is no longer opaque.
D, Follow-up arteriogram at 3 months shows no stenosis at the level of stent placement. No aneurysm is present.

repeat arteriogram 2 months later. If the aneurysm did not show progressive resolution, definitive therapy would be undertaken at that time. The patient was discharged on 650 mg per day of aspirin.

The patient returned 2 months later, describing continuing left C6-7 paresthesias and episodes of disequilibrium that could not be definitively related to the lesion. Interestingly, and without explanation, the pain in the left side of the neck, the arm, and the hand had permanently resolved immediately after the temporary balloon occlusion test. A repeat arteriogram revealed no change in aneurysmal morphology (Fig 1A). The decision was made to proceed with stent placement.

After intravenous sedation, an 8F Cook (Cook, Bloomington, Ind) sheath was advanced into the right common femoral artery using the Seldinger technique, and the patient received 10,000 units of intravenous heparin. A Palmaz P294M balloon-expandable stent (30 mm × 2.5 mm diameter when collapsed, 29.2 to 23 mm × 4 to 9 mm diameter when open; Johnson & Johnson, Warren, NJ) was loaded onto a Cordis angioplasty balloon (110-cm, 5F catheter; 6-mm balloon diameter, 40-mm balloon length: Cordis, Miami Lakes, Fla). The loaded balloon catheter was placed in an 8F introducer catheter (Interventional Therapeutics Corp, Fremont, Calif) such that the tip of the angioplasty catheter protruded from the introducer with the stent remaining within the lumen. The entire system was advanced into the left vertebral artery over a 0.035-mm hydrophilic guidewire (Terumo, Tokyo, Japan) until the stent straddled the aneurysm. Following withdrawal of the introducer along the angioplasty catheter to expose the balloon/stent combination to the arterial lumen, the balloon was inflated to 8 atm using a Disilator (Namic, Glens Falls, NY). The balloon was then deflated (Fig 1B). The Disilator was detached and a syringe was attached to the balloon catheter. While applying negative pressure, the entire system including the guiding catheter was removed. A repeat diagnostic arteriogram showed patency and slight overdistention of the left vertebral artery to a diameter of 5.6 mm. The aneurysm no longer opacified (Fig 1C).

The patient was taken directly to the intensive care unit. The heparin was discontinued but not reversed. His only complaint was of mild neck pain during stent inflation. Neurologic examination results remained normal, and the patient was discharged from the hospital the next day on 625 mg of aspirin. Repeat arteriography 3 months later showed absent filling of the pseudoaneurysm with normal patency of the parent artery. No evidence of intimal hyperplasia or concomitant vessel stenosis was present at the time of this first follow-up study (Fig 1D).
Case 2

A 53-year-old woman had a left carotid artery dissection during arteriography for a ruptured aneurysm of the right posterior communicating artery. The aneurysm was clipped successfully. Four months later, a follow-up arteriogram showed carotid patency with a small pseudoaneurysm at the previously injured segment; 2 months later, a repeat study again showed the pseudoaneurysm. All options for treatment were discussed with the patient and the referring physician, and a decision was made to proceed with internal carotid artery stenting.

As described for the previous case, a Palmaz stent was placed across the ostium of a 3-mm pseudoaneurysm that arose from the C1 level of the internal carotid artery (Fig 2A). Immediately following placement, the aneurysm showed no opacity (Fig 2B).

The patient was discharged from the hospital 48 hours later on a 3-month regimen of warfarin therapy. Follow-up arteriography 3 months later revealed obliteration of the aneurysm. The internal carotid artery was slightly narrowed at the proximal end of the stent relative to the stented portion of the vessel. We attributed this to kinking of the vessel as the rigid stent straightened it and also to slight overdistention of the stent in relation to the normal diameter of the vessel (Fig 2C).

Discussion

Extracranial vertebral artery dissections may occur spontaneously or they may result from trauma. They may be accompanied by headache, neck pain, and ischemic attacks. Although it has been shown that 88% of patients with spontaneous dissections recover partially or completely without surgery, and that 76% of involved vessels examined 15 to 115 months after initial arteriography return to normal or show improvement (4), many clinicians fear embolic complications or hemorrhage and prefer to sacrifice the involved vessel if the patient can tolerate such a loss. The natural history of extracranial traumatic vertebral aneurysms without cervical spine injury remains unknown, and although anticoagulation in symptomatic or asymptomatic patients may be safe (5), some clinicians worry about its use while the risk of vessel rupture exists.

In 1985, Palmaz introduced a balloon-expandable metallic stent (6). This device is a stainless steel lattice with rectangular slots that assume a diamond shape when expanded (7). The lattice structure provides a small working
surface area that reduces the mechanism's thrombogenicity. The stent is ultimately covered by a neointimal lining and incorporated into the vessel wall (8).

In 1994, Geremia et al (1) used a self-expanding stent to treat experimentally created side wall carotid aneurysms. Graves et al (9) had previously demonstrated that by changing the flow dynamics in aneurysms, one might be able to induce thrombosis within the sac. Geremia and colleagues thought that by crossing the aneurysmal neck, the stent's wire mesh would interfere with the normal inflow blood pattern and promote formation of thrombus and organized fibrosis within the aneurysm's fundus. Experimental models did in fact show that, after stent placement, blood and contrast material pooled within the dome, leading to fibrotic growth and organization and to eventual obliteration of the aneurysm (1). No parent vessels were occluded, and a thin layer of neointima coated the wire mesh (1).

Although there is a significant rate of occurrence of restenosis following stent placement in the systemic vasculature, the applicability of this statistic to the patients described in this article is uncertain. Restenosis rates reported for the peripheral and coronary arteries following stent placement are most closely related to diameter of the stented vessel's lumen. Vessels with larger diameters have a lower hemodynamically significant restenosis rate than do smaller vessels. Renal arteries would be the closest size match to the vertebral and carotid arteries that were stented in our patients. The restenosis rate in patients treated with renal artery stents is in the range of 10% to 25% (10–12). This restenosis rate, however, applies to patients being treated for atherosclerotic vascular stenoses, most of whom have undergone a technically unsatisfactory balloon angioplasty before placement of the vascular stent. It is probable that the surface characteristics of the diseased and traumatized vessels underlying these stents are more likely to incite an intimal hyperplastic response than is the more normal intima underlying the stents in our patients. A greater range of experience and careful follow-up will be necessary to ascertain the long-term consequences of stent placement in this patient population.

References