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Case Reports

Successful Management with 2 Overlapping Bare Stents for Post–Carotid Endarterectomy Carotid Pseudoaneurysm Secondary to Carotid Shunt

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Pseudoaneurysms are a rare complication of carotid endarterectomy. We successfully excluded with 2 overlapping bare stents a distal carotid artery pseudoaneurysm very likely induced by a Pruitt-Inahara shunt 2 months after carotid endarterectomy.

Pseudoaneurysms (PAs) of the carotid artery can have many different causes, including trauma, arterial dissection, vasculitides, infection, and post–carotid endarterectomy (CEA). A rare complication of CEA, PA occurs in about 0.3% of interventional procedures.[1] In the following case, the PA arose from the distal internal carotid artery 2 months after CEA, likely because of the use of a Pruitt-Inahara shunt. The PA was successfully excluded by inserting 2 overlapping bare stents.

CASE REPORT

A 72-year-old male patient underwent CEA for symptomatic left carotid artery stenosis 6 days after a transient ischemic attack, resulting in aphasia and unilateral amaurosis fugax. At operation under local anesthesia, the patient lost consciousness a few seconds after carotid clamping. An 8F Pruitt-Inahara carotid shunt (LeMaitre Vascular Inc., Burlington, MA) was inserted. No other intraoperative complications occurred; the procedure was not converted to general anesthesia; and the plaque was removed, and angioplasty with a Dacron patch was performed. Intraoperative echo color Doppler (ECD), as we routinely do, showed a normal flow pattern in the distal internal carotid artery, without evidence of arterial wall damage, residual stenosis, or intimal flaps.

Follow-up ECD examination 2 months later showed a PA of the internal carotid artery measuring 23 mm in diameter arising a few centimeters cranially to the distal portion of the patch (Fig. 1). The patient was asymptomatic and no pulsatile mass was palpable. Computed tomography (CT) angiography showed a sacciform PA measuring $25 \times 20\ mm^2$ (antero-posterior × latero-lateral; Fig. 2); the arterial wall breach measured 0.5 mm and was about 4 cm from the distal end of the patch, in the portion of the vessel in which the distal balloon of the Pruitt-Inahara shunt had been inflated. No parietal thrombus was present. Given the size of the PA, follow-up with watchful waiting was not considered; open surgery was ruled out because a safe distal clamping zone looked hard to achieve, so endovascular exclusion of the PA was preferred.

Full heparinization was instituted before the procedure. A 7F sheath (Pinnacle Destination; Terumo Medical Corporation, Somerset, NJ) was inserted via transfemoral arterial percutaneous access into the left common carotid artery, and a diagnostic angiogram was performed (Fig. 3). Lesion crossing was achieved with a microcatheter (SIM 2 catheter; Cook Inc, Bloomington, IN) over a 0.014-in microwire (HI-TORQUE Pilot 200 Guide Wire; Abbott Vascular, Santa Clara, CA). The microcatheter was then exchanged for a carotid Wallstent (Bolton Medical Inc., Sunrise, FL) delivery system. A $7 \times 40\ mm^2$ stent was

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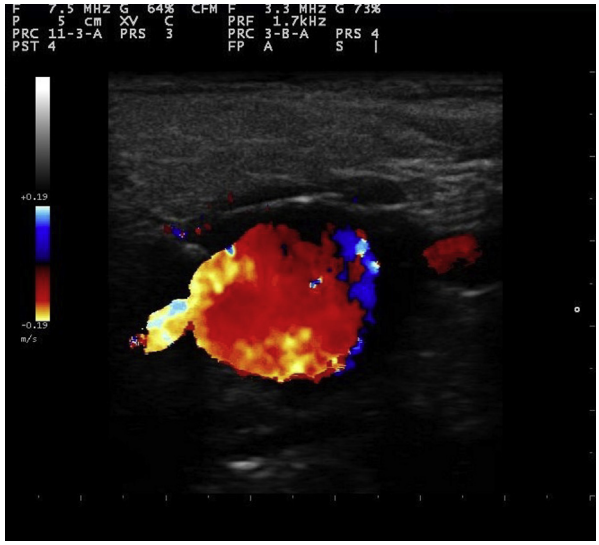

Fig. 1. Preoperative echo color Doppler showing the completely perfused pseudoaneurysm.

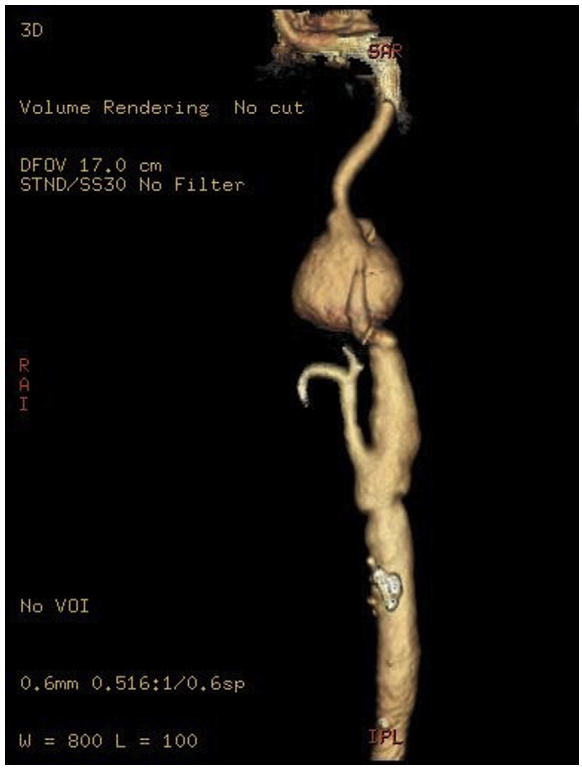

Fig. 2. Preoperative computed tomography scan showing the pseudoaneurysm distal to the endarterectomy zone.

deployed and the delivery system removed. Another carotid Wallstent delivery system was then introduced over the same wire and a second 7 × 40 mm^2 stent was deployed so that it overlapped with the first device by about 2.5 cm. Completion angiography to assess vessel integrity showed marked reduction in blood flow within the PA and unmodified vessel morphology, without kinks (Fig. 4). The arterial access was closed using an Angioseal closure device (St. Jude Medical, St. Paul, MN). A distal embolic protection device was not used because the treated segment of the vessel wall was devoid of plaque or parietal thrombus. Postoperative recovery was uneventful, and the patient was discharged 2 days later, with combined aspirin and clopidogrel therapy for 1 month and then aspirin monotherapy thereafter. An early duplex ultrasound examination on postoperative day 7 documented complete obliteration of the pseudoaneurysmal sac, with a normal flow pattern in the internal carotid artery, no endoleaks, and a slight decrease in diameter (23 mm vs. 25 mm at the preoperative CT scan). Carotid duplex ultrasound scans at 1 and 6 months confirmed the good result (Fig. 5).

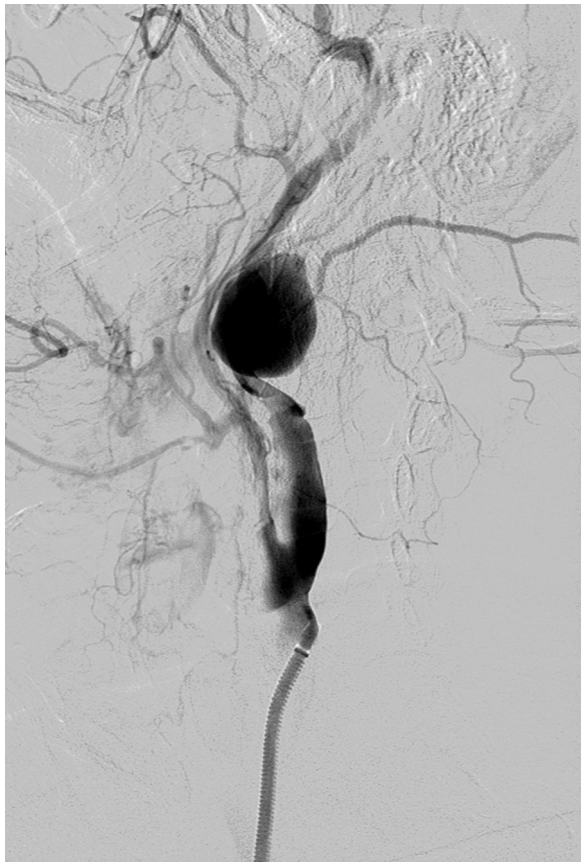

Fig. 3. Diagnostic angiogram before stent deployment.

DISCUSSION

PA of the internal carotid artery is a rare complication of CEA, accounting for 0.3% of cases.[1] It can result from suture degeneration, infection, clamp-induced damage, and shunt use. Surprisingly, however, patch closure does not increase the risk of PA

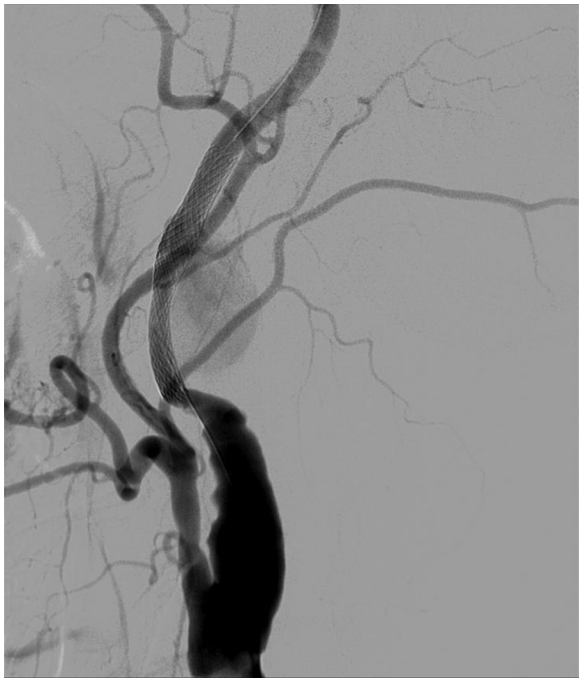

Fig. 4. Completion angiography after stent deployment, with slight perfusion of the sac.

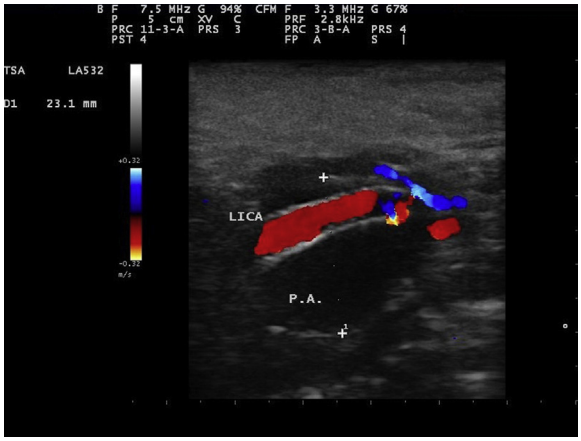

Fig. 5. Duplex ultrasound scan at 6 months, with complete exclusion of the pseudoaneurysm.

formation.[2] PAs of the internal carotid artery distal to the surgically exposed zone are very rare; the main etiology in such cases has been attributed to Pruitt-Inahara shunt balloon inflation.[3,4] This device is commonly used during CEA to provide blood flow to the brain whenever intolerance to vessel clamping occurs. A balloon is inflated in both the common and the internal carotid artery to fix the device during endarterectomy.

Alongside open surgical repair, which remains the gold standard especially in the treatment of infected PAs, endovascular techniques with bare stents plus coil embolization, percutaneous thrombin injection, and stent grafts are being increasingly used.[5] In the present case, the PA most likely occurred secondary to inflation of the distal balloon of the carotid shunt in the distal portion of the internal carotid artery. We use this type of carotid shunt selectively in about 10% of patients. This rationale is borne out by a Cochrane meta-analysis,[6] which found no differences in terms of perioperative strokes and deaths between the routine and selective use of shunts during CEA.

On further investigation into the causes of this event, our database search retrieved only 2 articles describing PA after CEA due to Pruitt-Inahara balloon inflation.[3,4] In the first one,[3] the authors reported that, considering the small size of the PA which remained unchanged during follow-up, they decided against taking an invasive approach. They also suggested 3 explanations for PA formation after CEA: distal balloon overinflation, normal balloon inflation in a previously damaged area of the artery, and local dissection followed by PA formation. In our patient, any likely explanation for its etiology remains speculative. Because the intraoperative ultrasound scan detected no immediate PA, we cannot be certain of the exact mechanism of how or when the PA formed. The patient had uncontrolled hypertension, which could be one of the reasons for late PA development. A similar, more recently published second report[4] described a case of post-CEA PA due to a carotid shunt, treated successfully with a Viabahn endoprosthesis (Gore & Associates Inc., Flagstaff, AZ). However, because its application in the supra-aortic trunks is off-label, available data come from a few case reports or small case series mostly regarding penetrating craniocervical injuries, so that data on medium and long-term patency in the internal carotid artery are needed.[7–9]

We used 2 overlapping bare stents instead of 1 covered stent to exclude the PA. This technique has been described in a few case reports on the treatment of traumatic or dissecting internal carotid artery PAs,[10,11] and it has been discussed in a very recent review by Walsh[12] in the treatment of supraclinoid internal carotid artery blister PAs. Our choice to use this technique appears not only to be more cost effective but also provides for similar good results, at least as seen at the early follow-up imaging examinations. At our institution, for example, a Viabahn stent graft for internal carotid artery repair costs about €2,500, whereas a carotid Wallstent costs €900. Sac thrombosis was achieved very early with complete exclusion of the PA as documented by ECD 1 week after the procedure. Furthermore, 1-month dual-antiplatelet therapy (aspirin plus

clopidogrel) was prescribed as usually recommended after carotid artery stenting.[13] Duplex ultrasound scans at 1 and 6 months confirmed the good result. The follow-up examinations at 12 months and then annually thereafter are planned.

To the best of our knowledge, this is the first case of carotid PA likely because of Pruitt-Inahara shunt use and successfully treated with 2 overlapping bare stents. This approach may be just as safe as cover stent deployment but more cost effective.

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