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Stent Deployment Resolves a Petrous Carotid Artery Angioplasty Dissection

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Summary: A patient with a symptomatic petrous carotid artery stenosis underwent balloon angioplasty, which was complicated by a flow-limiting dissection. The problem was resolved by deploying a balloon-expandable coronary stent. The patient suffered no neurologic deficit, was discharged within 12 hours, and was asymptomatic at last follow-up, 5 months after the procedure, without warfarin therapy. This therapeutic option may prove useful as a means to correct endovascular procedural complications.

Percutaneous transluminal balloon angioplasty has been used to dilate the common carotid artery, extracranial internal carotid artery (ICA) (1–4), and intracranial and subarachnoid obstructive lesions (5, 6). The intent of these procedures has been to open stenotic lesions to prevent progression to occlusion or to enable the controlled angioplasty endothelial injury, the source of recurrent emboli, to undergo neoendothelialization. Recently, obstructive lesions of the extracranial ICA have been managed with stent-supported angioplasty, not only to attain a more predictable result but also to avoid a flow-limiting intimal dissection (7, 8). These devices have not been deployed in the petrous or subarachnoid vessels. Symptomatic intracranial stenoses of the ICA have been associated with a high morbidity and mortality, and they are surgically inaccessible (6). When balloon angioplasty has been attempted in the intracranial subarachnoid vessels, the associated complications have included a 33% stroke rate and a 17% mortality rate. Thus, the reluctant use of balloon angioplasty in this situation reflects not only the extent of disease but also the technological limitations of balloons and guidewires, especially the lack of device(s) specifically designed to resolve any untoward complications.

We describe a case in which the deployment of a Palmaz-Schatz coronary stent (Johnson & Johnson Interventional Systems, Warren, NJ) resolved a flow-limiting intimal tear within the petrous portion of the ICA.

Case Report

A 57-year-old man with recurrent episodes of left-sided monocular blindness was referred for balloon angioplasty of a stenosis within the petrous portion of the left ICA. The patient had diffuse atherosclerotic cerebrovascular disease, and his history included a left carotid endarterectomy (1987), coronary artery bypass surgery (1988), a right carotid endarterectomy (1993), a left common carotid-to-subclavian artery bypass to relieve an ostial left common carotid artery stenosis (1993), an aortobifemoral bypass (1993), and bilateral femoral popliteal bypasses (1993). In 1994, angiography defined a severe ostial left common carotid artery stenosis, a severe left ICA lesion at the level of the petrous bone, and a patent carotid subclavian bypass. Medical management with antiplatelets and anticoagulants was initiated. However, while on aspirin, warfarin, and persantine therapy, additional episodes of ipsilateral monocular blindness occurred, which prompted the referral for endovascular recanalization.

The patient's pertinent physical findings included bilateral carotid bruits, a left supraclavicular bruit, and normal neurologic examination. He was considered a candidate for this unusual angioplasty procedure, and consent was obtained from the patient and his spouse after the unusual circumstances and the risk of major stroke and/or death were clearly delineated.

Methods

The therapeutic strategy was to resolve the problem of recurrent emboli by dilating the ICA lesion. Balloon angioplasty would injure the endothelium, enabling neoendothelialization to occur and precluding the site as a nidus of future emboli.

Technique

The left common carotid artery ostial stenosis was opened via a femoral approach, then a guide sheath was placed into the common carotid, enabling balloon angioplasty of the ICA lesion. With the patient under local anesthesia, the right common femoral artery was cannulated and an arterial sheath was placed. Heparin (10 000 U) was administered, and aortic arch angiography was performed (Fig 1A). Selective cannulation of the left common carotid artery was attained with a 6F headhunter catheter. A 0.035-inch Amplatz (Amplatz-Cook, Bloomington, Ind) extra-stiff exchange guidewire (260 cm long) was then placed into the distal common carotid artery, and a 6.5F 90-cm long sheath (Daig Corp, Minneapolis, Minn) was introduced across the left common carotid ostial lesion. The ostial stenotic lesion was opened using a primary (without predilatation) stent deployment technique with two P204 Palmaz stents on PE-MT balloons (6 mm in diameter, 2 cm long; catheter, 125 cm long; Meditech, Natick, Mass). High-pressure (12 atm) poststent deployment balloon dilatation was accomplished with a Blue Max balloon angioplasty catheter (7 mm in diameter, 3 cm long; Meditech). No residual narrowing was present (Fig 1B).

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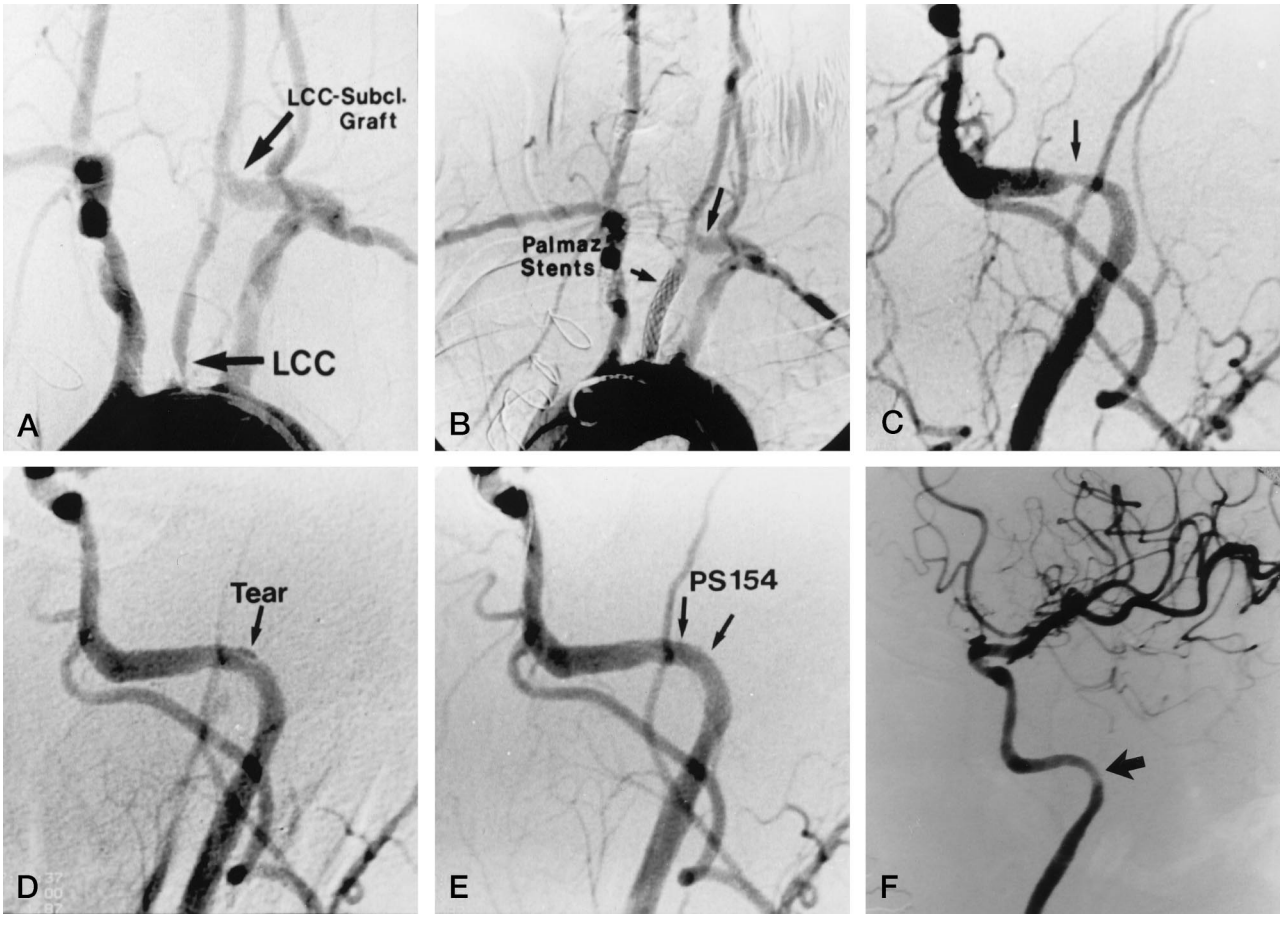

FIG 1. A 57-year-old man with recurrent emboli and episodes of monocular blindness.
A, Aortic arch angiogram shows an ostial left common carotid (*LCC*) stenosis and LCC–to–left subclavian bypass graft.
B, Successful Palmaz stent supported angioplasty of the LCC lesion (*short arrow*) and did not close the LCC–to–subclavian bypass (*long arrow*).
C, Angiogram of the LCC artery shows stenosis (*arrow*) in the petrous segment of the left ICA.
D, Postangioplasty angiogram shows the intimal tear.
E, Repair of tear after placement of the stent is documented in final angiogram.
F, Angiogram 5 months after stent placement (*arrow*) shows patent vessel.

Angiography of the intracranial lesion (Fig 1C) was performed through the guide sheath residing in the common carotid artery. A 0.014-inch hyperflex guidewire (USCI, Billerica, Mass) was navigated across the lesion, and a Bandit balloon catheter (3 mm in diameter, 20 mm long; Scimed Corp, Minneapolis, Minn) was passed distal to the lesion. We then replaced the hyperflex wire with a 0.014-inch, 300-cm-long extra-support guidewire (ACS, Mountain View, Calif). The balloon was positioned across the lesion, and contrast material injected through the sheath confirmed its position. The balloon was inflated to 8 atm for 60 seconds. Angiography delineated an open vessel with a linear tear on the outer curvature of the bend of the ICA (Fig 1D). The flow across the lesion became less and less brisk, diminishing to minimal flow, and the patient exhibited no neurologic impairment. Serious deliberation was given to placement of a coronary stent to seal the tear, as well as to anticoagulation with continuous heparin infusion. The coronary stent had not been designed to be used in the ICA nor to navigate the severe angulation present in this vessel as it crosses the petrous bone, and, therefore, was being used in a nonapproved manner. However, with a recognized decrease in anterograde flow, the decision was made to attempt stent deployment. The patient, having received aspirin (325 mg), was given ticlopidine hydrochloride (250 mg). Under fluoroscopic control and with much difficulty, a Palmaz-Schatz coronary stent (PS154; 15 mm long on a 3.0-mm-diameter balloon) was piloted across the lesion and deployed at 6 atm. The deployed stent was then dilated with a 3-mm-diameter 20-mm-long Bandit coronary balloon to 12 atm. Before inflation, the balloon had been purged with carbon dioxide to remove any oxygen in the event of balloon rupture. An angiogram (Fig 1E) showed repair of the tear, resumption of rapid, brisk flow, and no residual stenosis.

The catheter, guidewires, and sheaths were removed. The patient's neurologic status was unchanged, and independent neurologic observation revealed no alteration from preprocedural status. Computed tomography showed no change from the preoperative study. The patient was discharged within 12 hours on ticlopidine hydrochloride (250 mg twice a day) and aspirin (325 mg daily). At the 5-month follow-up, he was asymptomatic, and angiography confirmed a widely patent stent with normal arterial flow and no evidence of restenosis (Fig 1F).

Discussion

Stenosis within the intracranial ICA may be a marker of extensive cerebrovascular and systemic atherosclerotic disease (1). In a retrospective study of 66 patients with intracranial ICA stenosis ($\geq$ 50% diameter stenosis) who were followed up for a mean of 3.9 years, 18 patients (27%) experienced cerebrovascular ischemic events and 10 patients (15%) had an ipsilat-

eral stroke. Furthermore, 33 patients (55%) died during the follow-up period, 50% of related cardiac events. Another retrospective pooled data study (2) focusing on 58 patients with intracranial ICA stenosis revealed that only one third of the patients were alive and had had no neurologic event at the 30-month follow-up. Twenty-five patients (43%) had died (11 deaths were cardiac related and nine were stroke related) and 25 patients (43%) had had a neurologic event, of which 17 (28%) were strokes, 11 ipsilateral. These data imply that the presence of intracranial ICA stenosis is a good prognosticator of a poor outcome.

Balloon angioplasty has been proved successful in the middle cerebral artery (3), the cavernous carotid artery (4), and the petrous ICA (5). However, the limited use of intracranial balloon angioplasty may be directly related to interventional neuroradiologists' familiarity with potential applicable devices, the paucity of specifically designed equipment, and the lack of acceptable solutions to associated complications (eg, thrombus, thrombosis, particulate embolization, arterial dissection, acute vessel closure, vessel rupture). The successful, albeit technically difficult, deployment of the Palmaz-Schatz coronary stent to resolve a flow-limiting dissection of the petrous carotid artery has potential significance, and further defines the need for low-profile, trackable, lubricous, flexible balloons, and for stents that are easily deployable at low pressures on noncompliant balloons and specifically designed for intracranial use.

In this case, the Palmaz-Schatz coronary stent was deployed through a guide sheath positioned within the common carotid artery. The difficulty in negotiating the stent through the acute angulation just proximal to the lesion was overcome with guide sheath support. After deployment, a high-pressure inflation (12 atm) with a noncompliant balloon was used to fully expand the stent and firmly appose it against the arterial wall; this stent resolved the dissection and restored normal blood flow. The 60-second inflation time has been a standard part of our carotid bifurcation angioplasty technique, and has rarely been associated with loss of consciousness, seizures, or severe bradycardia. The postdeployment management was simply aspirin and ticlopidine hydrochloride therapy (no warfarin) and early discharge after independent neurologic assessment.

In this case, successful stent placement probably precluded catastrophic dissection resulting in abrupt vessel closure and stroke. Although the Palmaz-Schatz stent, composed of 316L stainless steel, accomplished its goal, its metallic composition interferes with magnetic resonance (MR) imaging by producing an image void, which is a problem for patients who may need recurrent intracranial MR diagnostic procedures. This obstacle may prompt manufacturers to create stents composed of nonferrous material.

In conclusion, balloon angioplasty of a symptomatic carotid artery lesion within the petrous bone resulted in a significant flow-limiting dissection that was corrected by deployment of a balloon-expandable, stainless steel, articulated Palmaz-Schatz coronary stent. Interventional neuroradiologists might wish to consider using this technique in similar, appropriate situations.

References

1. Marzewski DJ, Furlan PJ, St Louis P, Little J, Modic MT. **Intracranial internal carotid artery stenosis: long-term prognosis.** *Stroke* 1982;13:821–824
2. Craig DR, Meguro K, Watridge C, Robertson JT, Barnett HJM, Fox AJ. **Intracranial internal carotid artery stenosis.** *Stroke* 1982; 13:825–828
3. Purdy PO, Devous Sr MD, Unwin DH, Giller CA, Batjer HH. **Angioplasty of an atherosclerotic middle cerebral artery associated with improvement in regional cerebral blood flow.** *AJNR Am J Neuroradiol* 1990;11:878–880
4. O'Leary DH, Clouse ME. **Percutaneous transluminal angioplasty of the cavernous carotid artery for recurrent ischemia.** *AJNR Am J Neuroradiol* 1984;5:644–645
5. Rostomily RC, Mayberg MR, Eskridge JM, Goodkin R, Winn HR. **Resolution of petrous internal carotid artery stenosis after transluminal angioplasty.** *J Neurosurg* 1992;76:520–523
6. Higashida RT, Tsai FY, Halbach VV, Dowd CF, Hieshima GB. **Cerebral percutaneous transluminal angioplasty.** *Heart Dis Stroke* 1993;2:497–502
7. Marks MP, Dake MD, Steinberg GK, Norbash AM, Lane B. **Stent placement for arterial and venous cerebrovascular disease: preliminary experience.** *Neuroradiology* 1994;191:441–446
8. Eskridge JM. **Neurovascular stents.** *Radiology* 1994;191:313–314