Intracranial stent placement for the treatment of a carotid–cavernous fistula associated with intracranial angioplasty

Case report

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The authors report a case of an iatrogenic carotid–cavernous fistula (CCF) associated with intracranial angioplasty. Angioplasty was performed using a 3 × 10-mm Open Sail coronary balloon in a patient with high-grade stenosis of the left cavernous internal carotid artery (ICA). After angioplasty, a perforation developed in the cavernous ICA, resulting in a CCF. A 3.5 × 9-mm S670 coronary stent was used to treat the fistula. To the authors’ knowledge, this is the first reported case in which a CCF developed after angioplasty was performed using a coronary balloon. Long-term angiographic and clinical evaluation is needed to test the suitability and durability of intracranial angioplasty and stent placement in the treatment of symptomatic intracranial stenosis.

KEY WORDS • carotid–cavernous fistula • intracranial stenosis • angioplasty • stent

Intracranial ICA stenosis resulting from atherosclerosis has been associated with significant stroke and mortality rates, particularly if the lesion is symptomatic or exists in tandem with extracranial ICA stenosis. Among patients who have symptoms attributable to intracranial atherosclerosis that fail to improve with antithrombotic therapy, there is a significant rate of recurrent TIA and stroke or death. Intracranial angioplasty has been introduced as an alternative treatment for patients in whom symptoms have persisted despite medical therapy. Although there have been reports on the feasibility and safety of intracranial angioplasty, procedure-related complications such as acute arterial dissection have prompted the adjunctive use of intracranial stent placement. We describe a case in which iatrogenic CCB occurred after angioplasty for symptomatic cavernous ICA stenosis. A coronary stent was used to treat the fistula. Our case report adds to the list of complications of intracranial angioplasty and underscores the technological limitations of angioplasty in which a coronary balloon is used.

Abbreviations used in this paper: ACA = anterior cerebral artery; CCF = carotid–cavernous fistula; ICA = internal carotid artery; MCA = middle cerebral artery; MR = magnetic resonance; TIA = transient ischemic attack.

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History. This 84-year-old right-handed man presented with a transient right peripheral visual deficit that had occurred 3 months before neurosurgical evaluation. The patient had been receiving aspirin (81 mg/day). He denied any speech deficit, motor weakness, sensory loss, ataxia, or previous symptoms of TIA or stroke. No focal neurological deficit was seen on examination. His medical history was significant for hypertension and peripheral vascular disease. He had quit smoking 50 years ago. Cranial MR angiography revealed a high-grade (80%) stenosis in the left cavernous internal carotid artery (ICA). After angioplasty, a perforation developed in the cavernous ICA, resulting in a CCF. A 3.5 × 9-mm S670 coronary stent was used to treat the fistula. To the authors’ knowledge, this is the first reported case in which a CCF developed after angioplasty was performed using a coronary balloon. Long-term angiographic and clinical evaluation is needed to test the suitability and durability of intracranial angioplasty and stent placement in the treatment of symptomatic intracranial stenosis.

Procedure. The patient began aspirin (325 mg/day) and clopidogrel (75 mg/day) therapy 3 days before the procedure; he was fully awake during angioplasty. Percutaneous access was obtained through the right femoral artery. A No. 5 French Simmons 2 Sidewinder catheter (Boston Scientific Meditech, Natick, MA) was used to perform diagnostic cerebral MR angiography, which confirmed the 80%
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The patient was discharged home on the 2nd day after the procedure, and he remained asymptomatic at the 3-month follow-up evaluation.

Discussion

Atherosclerotic stenosis of large intracranial arteries accounts for 5 to 10% of ischemic strokes in the US. In retrospective studies it has been suggested that the annual risk of stroke in patients with intracranial stenosis is in the range of 3 to 15%.2,15 In the prospective Extracranial–Intracranial Bypass Study, recurrent strokes or TIAs occurred in the medical treatment group at a rate of 11.7% per year in patients with symptomatic MCA stenosis or occlusion during the 42-month follow-up period.7 Currently, it is not clear which antithrombotic therapy is most effective for symptomatic intracranial stenosis.

In our patient, other antiplatelet agents such as thienopyridine derivatives (clopidogrel and ticlopidine) or dipyridamole could have been prescribed first, with angioplasty reserved until such time as his symptoms became refractory to this therapy. Studies have demonstrated the superiority of clopidogrel, ticlopidine, and dipyridamole over aspirin in reducing the risk of stroke and other vascular events among patients with previous stroke and TIA.8,17 These studies, however, have not specifically evaluated the population with symptomatic intracranial stenosis.

Another alternative therapy in our patient would have been warfarin; however, it is still not clear whether warfarin is superior to aspirin in patients with symptomatic intracranial stenosis. A prospective, randomized, double-blind clinical trial (the Warfarin–Aspirin Symptomatic Intracranial Disease trial) is underway to determine whether warfarin (international normalized ratio 2:3) or aspirin (1300 mg/day) is more effective for preventing stroke and vascular occlusion–related death in patients with symptomatic intracranial large artery stenosis (50–99%).15 Despite the available medical treatment, a significant proportion of patients with symptomatic intracranial stenosis continue to have recurrent TIAs or strokes. In a retrospective study, Thijs and Albers14 reported a 56% risk of recurrent TIA or stroke in patients with symptomatic intracranial stenosis treated with antithrombotic agents. We performed angioplasty because of the high likelihood of stroke in our pa-

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![Fig. 1. A: Lateral MR angiography view of the left cavernous ICA stenosis. B: Anteroposterior view of the intracranial circulation revealing no contrast material filling the left ACA.](image1)

![Fig. 2. Left oblique MR angiography views of the shelf of an atherosclerotic plaque in the cavernous segment of the left ICA.](image2)

![Fig. 3. A: No. 6 French Envoy guiding catheter (Cordis Neurovascular Systems, Miami Lakes, FL) was advanced over a 0.035-in Terumo Glidewire (Boston Scientific Meditech) into the distal left cervical ICA. The catheter was connected to a continuous heparinized saline flush.](image3)

![Fig. 3. B: Anteroposterior view of the intracranial circulation revealing no contrast material filling the left ACA.](image4)
Patient, who was receiving 81 mg of aspirin per day. The complication in our case underscores the controversy surrounding the selection of patients for intracranial angioplasty.

Our case illustrates the technological limitations of balloon angioplasty in the treatment of intracranial stenosis. We selected a balloon size that was smaller than the normal diameter of the cavernous ICA proximal or distal to the stenosis. The balloon was inflated slowly with an insufflator over a period of 30 seconds. Despite these maneuvers, perforation of the artery developed, with subsequent CCF formation. In our case, the CCF was relatively small with slow flow, and we observed no significant pseudoaneurysm or varix of the cavernous sinus. As a result, we could have left the CCF alone because spontaneous closure of CCFs does occur, although it is more common in indirect fistulas.

An alternative endovascular treatment of the CCF in our case would include an intraarterial approach with balloon or coil occlusion of the fistula. Transvenous occlusion of the cavernous sinus with a silicone balloon and platinum microcoils is another option, although it can be a lengthy and costly procedure. We elected to treat the CCF with a highly flexible coronary stent that would allow navigation into a tortuous segment of the ICA and divert the shear stress of the blood into the fistula site. We believed that such a hemodynamic change might promote closure of the fistula. The S670 coronary stent is more flexible than the S7 stent in addition, the shorter and smaller profile of the S670 device may have allowed its navigation into the cavernous segment of the ICA. We did not believe that direct stent placement over the cavernous lesion without initial angioplasty was possible because of the severe stenosis and curvature of the cavernous ICA. If the stent had failed to occlude the fistula, we would have observed the patient and considered the aforementioned options if needed.

This is not the first case of CCF that has been treated with stent placement. Kocer, et al., have reported the use of the polytetrafluoroethylene-covered Jostent (Jomed International, Helsingborg, Sweden) to treat a CCF that resulted from an injury to the ICA during a transsphenoidal surgery for pituitary adenoma. The long-term patency of covered stents is unknown, and this device was not available to us at the time of our procedure. In addition, Weber, et al., have reported a cure of a direct CCF by using two detachable silicone balloons and a porous ACS multilink coronary stent (Guidant, Temecula, CA). In this case, intraoperative manipulation of a Fogarty catheter during surgical thrombendarterectomy of the ICA caused the CCF. To our knowledge, our case represents the first angioplasty-related CCF.

Conclusions

Intracranial angioplasty for symptomatic stenosis is not benign. The endovascular surgeon must be prepared to deal with complications of this procedure. Further experience is needed to evaluate the durability and suitability of intracranial angioplasty and stent placement in the treatment of symptomatic intracranial stenosis.

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