Intravascular stent and endovascular coil placement for a ruptured fusiform aneurysm of the basilar artery

Case report and review of the literature

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The authors demonstrate the technical feasibility of using intravascular stents in conjunction with electrolytically detachable coils (Guglielmi detachable coils [GDCs]) for treatment of fusiform, broad-based, acutely ruptured intracranial aneurysms and review the literature on endovascular approaches to ruptured aneurysms and cerebral stent placement. A 77-year-old man presented with an acute subarachnoid hemorrhage of the posterior fossa. A fusiform aneurysm with a broad-based neck measuring 12 mm and involving the distal vertebral artery (VA) and proximal third of the basilar artery (BA) was demonstrated on cerebral angiography. The aneurysm was judged to be inoperable. Six days later a repeated hemorrhage occurred. A 15-mm-long intravascular stent was placed across the base of the aneurysm in the BA and expanded to 4 mm to act as a bridging scaffold to create a neck. A microcatheter was then guided through the interstices of the stent into the body and dome of the aneurysm, and GDCs were deposited for occlusion.

The arteriogram obtained after stent placement demonstrated occlusion of the main dome and body of the aneurysm. The coils were stably positioned and held in place by the stent across the distal VA and BA fusiform aneurysm. Excellent blood flow to the distal BA and posterior cerebral artery was maintained through the stent. There were no new brainstem ischemic events attributable to the procedure. No rebleeding from the aneurysm had occurred by the 10.5-month follow-up evaluation, and the patient has experienced significant neurological improvement.

Certain types of intracranial fusiform aneurysms may now be treated by combining intravascular stent and GDC placement for aneurysm occlusion via an endovascular approach. This is the first known clinical application of this novel approach in a ruptured cerebral aneurysm.

KEY WORDS • intravascular stent placement • endovascular coil placement • basilar artery aneurysm • endovascular therapy • interventional neuroradiology • subarachnoid hemorrhage
Case Report

History. This 77-year-old right-handed man with a history of hypertension and hypercholesterolemia presented with the sudden onset of severe headache after sexual intercourse; the headache was followed by vomiting and confusion. On evaluation he complained of severe neck pain and headache and was stuporous. His cranial nerve examination revealed a complete left- and a partial right-sided sixth nerve palsy and left-sided hemiparesis. His condition was assessed as Hunt and Hess Grade III.

Examination. A computerized tomography (CT) scan of the head revealed areas of high density predominately within the prepontine cistern and both lateral ventricles that were consistent with acute SAH. Mild hydrocephalus was also present. The patient’s mental status declined, requiring tracheal intubation and placement of an extra-ventricular drain.

Admission cerebral angiography demonstrated a fusiform, ectatic aneurysm involving the distal right VA and the proximal BA. The inflow to the aneurysm from the distal VA was clearly separate from the outflow, which was at the level of the anterior inferior cerebellar artery (AICA). The aneurysm was triangular, with the base of the lesion measuring 12 mm, and the apex protruding into the brainstem (Fig. 1 left) The contralateral left VA was occluded proximal to the origin of the left posterior inferior cerebellar artery, and there was poor collateral flow from the right and left internal carotid arteries to the posterior cerebral circulation, across the posterior communicating arteries.

The aneurysm was judged to be unsuitable for surgical clipping or endovascular coil placement because of its location and configuration: there was no neck for clipping or for retention of the coils. The coils would simply protrude into the BA, and proximal occlusion of the distal right VA would result in BA thrombosis caused by poor collateral circulation from the internal carotid arteries.

Treatment. The patient was treated medically with nimodipine and sedation, and his blood pressure was controlled with intravenously administered labetalol. His mental status improved over the next several days, allowing extubation. On the 6th hospital day, the patient experienced sudden worsening of his headache and concurrent mental deterioration. A repeated CT scan confirmed a recurrent SAH.

Outcome. The follow-up cerebral angiogram demonstrated severe vasospasm of the distal right VA and an extra protrusion of the dome of the BA aneurysm, probably representing the site of recurrent SAH. The aneurysm at its base measured 12 mm, and the dome and body measured $7 \times 9$ mm (Fig. 1 right). After full and informed consent was received from his family, we decided to treat the patient by using a novel endovascular technique combining an intravascular stent and detachable coil placement.

Novel Treatment for Rebleeding

Endovascular Technique

A No. 8.0 French guide catheter (Cordis Endovascular J. Neurosurg. / Volume 87 / December, 1997
Systems, Miami, FL) was selectively placed via a trans-femoral approach into the proximal right VA. The patient received 5000 U of heparin to prevent thrombus formation on the microcatheters. An 0.85-mm nondetachable silicone balloon (Interventional Therapeutics Corp., Fremont, CA) was flow directed to the distal VA stenosis, and balloon angioplasty of the vasospastic segment was performed to restore the normal diameter of the vessel and improve perfusion to the posterior cerebral circulation. A rapid transit microcatheter with a 0.016-in steerable microguidewire (Instinct; Cordis Endovascular Systems) was carefully navigated through the distal VA beyond the BA aneurysm, and the tip was directed into the distal branch of the left posterior cerebral artery (PCA). A 0.014-in, 300-cm exchange nitinol guidewire was placed in the distal PCA and the microcatheter was removed. A coronary balloon-expandable stent (Palmaz–Schatz PS 1540; Johnson & Johnson Interventional Systems, Warren, NJ) with the sheath removed (Fig. 2) was then guided over the exchange guidewire and across the fusiform aneurysm, with the proximal segment placed in the distal VA at the aneurysm inflow and the distal segment of the stent placed at the level of the AICA vessels at the aneurysm outflow (Fig. 3). The length of the fusiform aneurysm was 12 mm, and the stent length was 15 mm. The balloon catheter expanded the stent to 4 mm by using 6 atm of pressure and was then removed (Fig. 4 left).

The rapid transit microcatheter with an 0.016-in microguidewire was inserted by means of the No. 8.0 French guide catheter and navigated with the aid of careful fluoroscopic observation through the distal VA, through the interstices of the stent, and into the body of the aneurysm. With the stent remaining in position as a bridging scaffold, a GDC-10 measuring 8 mm × 30 cm (Target Therapeutics Corp.) was deployed within the body and dome of the aneurysm. Two additional coils measuring 7 mm × 30 cm and 5 mm × 15 cm were detached within the aneurysm. The microcatheter was removed along with the exchange guidewire. An angiogram obtained after stent and GDC placement demonstrated occlusion of the dome, body, and daughter aneurysm, with excellent blood flow through the BA and posterior cerebral circulation. After stent placement, the patient received ticlopidine (250 mg twice/day) for 6 weeks and aspirin (5 grains/day) for 6 months to prevent platelet aggregation on the BA stent. A follow-up arteriogram obtained 10 days postoperatively confirmed continued excellent flow through the posterior cerebral circulation and continued stability of the
coils in the aneurysm, which were held in place by the intravascular stent. There was no evidence of distal branch occlusions caused by thromboembolic clots (Fig. 4 right).

Neurological Follow Up

Following the procedure, the patient experienced transient pharyngeal dysmotility requiring a tracheotomy, most likely as a result of his second SAH and associated vasospasm. The patient also remained obtunded as a result of hydrocephalus caused by the previous SAH and therefore a second ventricular drain was placed. The ventricular catheter used in this procedure inadvertently migrated into the genu of the right internal capsule, resulting in left upper-extremity weakness. After removal of the drain and eventual placement of a low-pressure ventriculoperitoneal shunt his mental status and arm paresis recovered over the next several weeks. His palate motor function also improved and the tracheostomy was removed. The patient did not develop any new signs or symptoms of brainstem ischemia in the distribution in which the stent and coils were placed.

At 8 weeks after his initial SAH he had only mild encephalopathy. He was ambulatory with the aid of a walker, had minor left upper-extremity weakness, and mild persistent dysphagia. At his 10.5-month follow-up evaluation his clinical and neurological function continued to improve, with improved cognition and ambulation, and he has not suffered from recurrent hemorrhage or brainstem dysfunction as a result of his treatment.

Discussion

Acute SAH from ruptured cerebral aneurysms accounts for 30,000 new cases of SAH per year in the United States and results in significant neurological disability and death. Aneurysm rebleeding is one of the most critical and disabling events associated with ruptured aneurysms, with a reported mortality rate of between 70% and 90%.20,21 The highest incidence of rebleeding occurs within the first 7 days, and in the absence of surgical or endovascular treatment, there is a 20 to 30% incidence of rebleeding within 2 weeks of the initial ictus.12,24 It has been estimated that 17% of patients with aneurysmal SAH will die or become severely disabled from rebleeding within the first 48 hours after the initial hemorrhage. Thus, early surgical or endovascular treatment of the aneurysm is recommended to decrease that risk.2,13,14,16

Viñuela, et al.,25 recently reported on the use of GDCs for occlusion of acutely ruptured intracranial aneurysms. In a series of 403 patients presenting with acute SAH from a ruptured aneurysm and treated within 15 days of their primary bleed, the 6-month rebleeding rate was reduced to 2.2%. It was believed that by mechanically blocking blood flow into the aneurysm with detachable coils the risk of aneurysm rerupture would be significantly reduced. In that series the mortality rate caused by technical complications of the procedure was 1.7%, with an 8.9% technical morbidity rate. An additional 4.5% mortality rate was attributed to complications caused by the severity of the primary hemorrhage. The incidence of rebleed-
ing after GDC placement was decreased, even in patients with incomplete aneurysm occlusion. In 57.1% of large aneurysms and 50% of giant aneurysms, the coil occlusion was subtotal. Patients were treated with endovascular therapy because of anticipated surgical difficulty in clipping the aneurysm in 69.2% of cases, failed surgery in 12.7%, poor neurological or medical status in 16.9%, or refusal of surgery in 1.2% of cases.

Ruptured fusiform aneurysms of the BA represent one of the most difficult challenges of therapy. When the inflow and outflow of an aneurysm is not contiguous, treatment options are limited. Previous authors have reported surgical ligation or endovascular balloon occlusion of both distal VAs, ligation of the inflow or outflow of the BA, or direct occlusion of the aneurysm and parent vessel. However, these modalities are useful only if there is adequate collateral circulation to the distal BA and PCA and the perforating brainstem vessels. This patient had poor collateral filling of the PCAs bilaterally, occlusion of his left VA, and acute vasospasm of his right VA with concomitant basilar ischemia. He was therefore not a good candidate for parent vessel occlusion therapy.

We decided to treat his right VA vasospasm with balloon angioplasty and to use an intracranial implantable stent to act as a scaffold across the wide fusiform neck of the aneurysm, bridging the inflow and the outflow. This would hold the GDCs in place within the aneurysm. The Palmaz–Schatz balloon-expandable stent is composed of a stainless steel metal alloy, has a nominal length of 15 mm, and consists of two 7-mm-long segments connected by a 1-mm articulation. It is premounted on balloons available in 3-, 3.5-, and 4-mm sizes. The articulation facilitates negotiation of the stent through tortuous small vessel anatomy. The stent is used to bridge the vessel and maintain patency of small vessels after balloon angioplasty for atherosclerosis and acute vessel dissection. The interstices are wide enough to allow adequate blood flow to perfuse brainstem-perforating vessels of the BA without producing occlusion. The stent was used in this case to create a “neck” in the aneurysm that would hold the GDCs in place. The interstices of the stent were also suitable to allow a microcatheter to be placed through it to deposit the GDCs between the stent and aneurysm, inducing occlusion of the dome and body.

After the procedure, and at 3 and 10.5 months of clinical follow up, the patient had not experienced recurrent hemorrhage, continued to make progressive improvement in his neurological function related to the initial bleeding episodes, and had no signs of brainstem or posterior fossa ischemia or infarction related to the procedure. The patient will continue to be monitored clinically and radiographically at 6- to 12-month intervals to ensure that there are no further changes in his treated aneurysm. Blood flow through the stent will be monitored by transcranial Doppler ultrasonography and a follow-up angiogram will be obtained 12 months postoperatively. Treatment of the remaining cephalad portion of the aneurysm is possible if there is any further change in the size or configuration of the lesion.

Previous reports have discussed the feasibility of using stents in both the extra- and intracerebral circulation for aneurysms in laboratory animals. Both Szikora, et al., and Massoud, et al., reported on the biomechanical feasibility of treating experimental fusiform aneurysms in animal models with a combination of stents and coils. Stent patency was maintained while allowing strategic coil placement in an experimentally induced aneurysm sac. Geremia, et al., also reported on the efficacy of metallic stents in the treatment of experimentally created carotid–jugular fistulas in dogs, with fistula closure in three of five cases. In a second study involving silicone-covered metallic stents, Geremia, et al., reported on their successful deployment and occlusion of the lesions in six mongrel dogs with surgically created arteriovenous fistulas.

Wakhloo, et al., reported on the feasibility in canine VAs of treatment with self-expanding nitinol stents, which did not produce significant risk for thromboembolic events after implantation. Although stents are now being used more frequently in the coronary, peripheral, and extracerebral circulation for atherosclerotic disease after balloon angioplasty, as far as we know this is the first reported case of intravascular stent implantation with concomitant coil occlusion of a fusiform BA aneurysm in a patient presenting with acute SAH.

Conclusions

The technique of using an intracranial stent to create a bridging scaffold followed by endovascular placement of GDCs through the interstices of the stent into an intracranial aneurysm may provide another treatment option in the therapeutic armamentarium for patients who present with fusiform aneurysms in which direct surgical clipping or conventional endovascular therapy would be difficult, and in whom parent artery occlusion is not a viable option. It should be considered as a last resort, when no other surgical or endovascular techniques are feasible for therapy.

References

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