

Endovascular management of spontaneous carotid artery dissection

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Objective: Despite medical therapy, a subset of patients with spontaneous carotid artery dissection (SCD) experience recurrent or progressive symptoms. In this study, we assessed the safety and efficacy of endovascular stent angioplasty in the treatment of SCD.

Methods: Seven consecutive patients with SCD underwent endovascular stent angioplasty of a total of 12 vessels. Indications included the presence of a large or enlarging pseudoaneurysm, a contraindication to anticoagulation, failure of anticoagulation, and compromised cerebral blood flow.

Results: Five patients had fibromuscular dysplasia. All patients tolerated the procedure well, with no adverse clinical events. All patients showed symptomatic improvement on clinical follow-up except for one patient, who had hemorrhagic conversion of a pre-existing large ischemic stroke 13 days after intervention; he made an eventual recovery to baseline. All stents have remained patent on radiologic follow-up.

Conclusions: This study provides additional evidence that endovascular stent angioplasty is a safe and effective treatment for SCD in patients for whom medical treatment is not adequate. (*J Vasc Surg* 2005;42:854-60.)

The incidence of spontaneous carotid artery dissection (SCD) in the United States is thought to be approximately 2.6 per 100,000.¹ It is one of the leading causes of stroke in young people.² The underlying cause of SCD is usually idiopathic; however, fibromuscular dysplasia is present in approximately 12% of these patients.^{3,4} Although more than 85% of medically treated patients with SCD improve clinically and angiographically,^{4,5} there are at least four circumstances under which medical management may be insufficient: patients with recurrent symptoms despite anticoagulation,⁶⁻⁸ patients in whom anticoagulation is contraindicated because of the risk of bleeding,^{6,7} patients with expanding or symptomatic pseudoaneurysm,^{7,9} and patients with significantly compromised cerebral blood flow due to the involvement of multiple vessels, poor collateral vessels, or both.⁸

When medical therapy is inadequate, surgical repair can be used but is associated with a 9% to 12% incidence of death and stroke.^{3,10} Several small case series have indicated that stenting is an effective alternative to surgery for repair of cervical artery dissection.^{6-9, 11-15} However, no series has focused solely on spontaneous dissections. In this article, we present 7 patients with SCD who had a total of 12 vessels successfully treated with endovascular stent placement.

METHODS

Patients. We performed a retrospective review of seven consecutive patients who underwent endovascular

treatment of SCD performed with stent placement between October 2001 and November 2003. Over this same time period, approximately 135 patients with cervicocephalic dissections were seen at our institution. Approval for the collection of these data was obtained from the institutional review board, and informed consent was obtained from all patients or their surrogates before intervention.

Procedure. All patients were pretreated with clopidogrel (75 mg by mouth daily for at least 4 days before the procedure or a 600-mg oral load if emergently treated) and aspirin (325 mg by mouth daily). Patients underwent four-vessel digital subtraction angiography. Intravenous heparin was administered to achieve an activated clotting time of longer than 250 seconds. All patients were treated under local anesthesia. Stable vessel access was achieved by placing a sheath or guide catheter distally in the common carotid artery. The true lumen of the dissected vessel was identified by using high-resolution angiography with high frame rates (5-10 frames per second) and multiple projections. It was often helpful to perform nonsubtracted angiograms to better visualize the thin luminal flaps, which can be subtracted out with digital subtraction techniques. When there was doubt about the true lumen, a 0.014-inch floppy-tipped coronary microwire was passed distal to the lesion into the presumed true lumen; a microcatheter or 1.5-mm balloon catheter was then inserted distal to the lesion, and the wire was removed. If there was no blood return, the catheter or balloon was withdrawn. If there was blood return, a very gentle manual injection of 0.25 to 1 mL of contrast was injected through the microcatheter or central balloon lumen to confirm that the tip was in the true lumen. When this was confirmed, the wire was replaced, and the procedure was continued. If the catheter tip was in a false channel, then the catheter was withdrawn, and wiring of the true lumen was reattempted. We used the softest wire possible and paid particular attention to good wiring tech-

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Competition of interest: none.

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Figure. **A**, Left intracranial and extracranial internal artery before intervention. **B**, Left intracranial internal carotid artery after intervention. **C**, Left extracranial internal carotid artery after intervention.

Table I. Clinical characteristics

Patient No.	Age (y)	Sex	Presenting symptoms	Time from first SCD to first stent	FMD?	Infarct location	Vessels with dissection	Indication for treatment
1	41	F	Bifrontal headache, neck pain, tinnitus in R ear, and numbness of R side of face and arm	2 y	Y	R hemisphere watershed infarcts	R ICA L ICA	Expanding pseudoaneurysm (expanding over 3 mo) with recurrent TIA and intractable headaches
2	57	M	Syncope, R homonymous hemianopia, aphasia, R hemiparesis, and obtundation	8 d	N	L parietal-occipital	L ICA R ICA	Highly compromised bihemispheric cerebral blood flow
3	39	F	L Horner syndrome, tinnitus, and headache	1 y	Y	None	R ICA L ICA L Vertebral	Severe flow-limiting dissection and expanding pseudoaneurysm (expanding over 1 y)
4	53	M	R-sided neck pain and severe recalcitrant headache	5 mo	N	None	R ICA L Vertebral	Expanding pseudoaneurysm (expanding over 4 mo) with intractable headaches
5	40	F	L lower extremity numbness	5 d	Y	R insular cortex and internal capsule	R ICA L ICA	Ongoing bihemispheric ischemia (and emboli by TCD) despite heparin and aspirin
6	54	F	Right upper extremity weakness and mild expressive aphasia	3 mo	Y	L frontal-parietal	R ICA R CCA L ICA	Compromised cerebral blood flow
7	50	F	Right frontal headache, L hemianopsia, dysarthria, and L hemianesthesia	5 d	Y	R frontal-temporal	R ICA L ICA	Compromised cerebral blood flow with fluctuating clinical deficits

SCD, Spontaneous carotid artery dissection; L, left; R, right; ICA, internal carotid artery; TIA, transient ischemic attack; CCA, common carotid artery; FMD, fibromuscular dysplasia; TCD, transcranial Doppler ultrasonography.

nique to avoid extending the dissection flap and entering pseudoaneurysms, given the risk of perforation.

All lesions causing luminal narrowing were predilated with a small (2.5- to 4-mm) coronary balloon. A combination of self-expanding or balloon-expandable stents was used in each vessel treated, depending on the vessel size and location: intracranially extending lesions (eg, petrous carotid segment) were treated with balloon-expandable stents, whereas, because of their flexible nature, cervical carotid lesions were treated with self-expanding stents when possible. Stent grafts were used in all cases involving expanding pseudoaneurysms. The external carotid artery orifice was not covered during these procedures (Fig 1).

Postprocedure management. After the intervention, clopidogrel was continued for at least 6 weeks, and aspirin was continued indefinitely. All patients were referred by and evaluated before and after the procedure by a neurologist specializing in cerebrovascular disease. Evaluation of stent patency was obtained with ultrasonography, computed tomographic angiography (CTA), or repeat digital subtraction angiography (Figure).

RESULTS

Patient characteristics. Seven patients (5 women) between the ages of 39 and 57 years (mean \pm SD 47.7 \pm 2.8 years) were treated (Table I). Five of the seven patients had

a stroke before treatment as a result of the initial arterial dissection. The time interval between the initial dissection and the first endovascular procedure is listed in Table I. Six patients had angiographic evidence of dissection in two vessels, and one patient, in three vessels. The indications for endovascular treatment included ongoing ischemia of 12 to 24 hours' duration (despite anticoagulation with heparin) in three patients, expanding pseudoaneurysms in three, and severe reduction in cerebral blood flow in one. One patient had 3 vessels treated, 3 patients had 2 vessels treated, and 3 patients had 1 vessel treated, for a total of 12 treated vessels. Of the patients with two or more treated vessels, two had simultaneous treatment of both internal carotid arteries (ICA). One of these patients was comatose from bihemispheric ischemia, and the other had recurrent strokes and bihemispheric emboli on transcranial Doppler monitoring despite heparin and aspirin therapy. A third patient had the left ICA treated because of recurrent stroke and had the right internal and common carotid arteries treated subsequently because of severe flow-limiting stenosis involving both vessels. The location of the dissections was the right ICA (n = 6), the right common carotid artery (n = 1), and the left ICA (n = 5).

Table II lists the procedural details. Self-expanding stents were used in 11 vessels. The single balloon-expandable stent was placed in a relatively immobile segment: the distal

Table II. Summary of angiographic characteristics and procedural details by vessel treated

<i>Vessel treated</i>	<i>Balloons and stents</i>	<i>Pretreatment/posttreatment degree of stenosis</i>	<i>Complications</i>
L ICA and pseudoaneurysm	4 × 30-mm Maverick* balloon 5 × 26-mm Jomed [†] stent #1 5 × 26-mm Jomed stent #2 5 × 20-mm Viatrac [‡] balloon	50% → 0%, pseudoaneurysm completely occluded	None
R ICA	4 × 40-mm Viatrac balloon 7 × 40-mm Precise [§] stent 5 × 20-mm Viatrac balloon	99% → 0%	Hemorrhagic conversion 13 d after intervention
L ICA	4 × 40-mm Viatrac balloon 6 × 40-mm Precise stent	70% → 0%	None
R ICA	4 × 13-mm Tetra [‡] stent	80% → 5%	None
L ICA pseudoaneurysm	5 × 19-mm Jomed stent 5 × 20-mm Viatrac balloon	50% → 0%, pseudoaneurysm completely occluded	None
R ICA and 3 pseudoaneurysms	8 × 30-mm Wallgraft* 7 × 40-mm OptaPro [§] balloon	80% → 0%, pseudoaneurysms completely occluded	Small, non-flow-limiting, intimal tear R CCA
R ICA	7 × 20-mm Precise stent	90% → 10%	None
L ICA	7 × 40-mm Precise stent 5 × 30-mm Viatrac balloon	90% → 0%	None
L ICA	6 × 30-mm Precise stent 5 × 20-mm Viatrac balloon	70% → 0%	None
R CCA extending into ICA	7 × 8-mm SMART [§] stent 5.5 × 30-mm Aviator [§] balloon 7 × 20-mm Precise stent	90% → 0%	None
R ICA	5.5 × 30-mm Aviator balloon 1.5 × 15-mm Maverick balloon 5 × 20-mm Precise stent 5 × 40-mm Precise stent 4.5 × 20-mm OpenSail [‡] balloon	99% → 0%	None

L, Left; R, right; ICA, internal carotid artery; CCA, common carotid artery.

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[†]Jomed Inc (Helsingborg, Sweden).

[‡]Guidant Corp (Indianapolis, IN).

[§]Cordis Corp (Miami Lakes, FL).

cervical ICA extending into the petrous segment. A total of four stent grafts were placed in the three segments that had pseudoaneurysms; one patient (patient 1) required overlapping Jomed (Jomed International AB, Helsingborg, Sweden) stents to completely cover a long pseudoaneurysm. Angiographic success was achieved in all patients, with no significant residual stenoses or filling of the pseudoaneurysms.

Outcomes. There were no immediate clinical complications, and the three patients with ongoing clinical deficits had immediate improvement or resolution of recurrent transient ischemic attacks (Table III). There was one small, non-flow-limiting, intraprocedural dissection that was asymptomatic. The patient with bihemispheric ischemia and obtundation developed hemorrhagic conversion of a large infarct 13 days after the intervention but has since had continued recovery and is living independently. One patient died after heart transplantation (4 months after stenting).

All of the vessels were patent on follow-up imaging (catheter angiography, carotid duplex scan, or CTA; Table III). The average duration of follow-up was nearly 14 ± 8 (SD) months (range, 3 to 24 months). At their most recent

follow-up visits, all patients showed improvement or resolution of their previous neurologic deficits, and none had recurrent ischemia. The patients with headache had either complete resolution of headaches or marked improvement to the point that they no longer required headache-specific therapy.

DISCUSSION

This case series demonstrates that SCD can be safely and effectively treated with stenting: it is the largest published series to date of patients treated with this technique. All of the patients in this series were treated with stents to maintain vessel patency by using a technique similar to that used for atherosclerotic carotid stenosis.¹⁶ However, we did not use emboli-prevention devices because we believed that the risk of worsening the dissection or creating a new intimal tear was greater than the potential for embolization given the lack of bulky plaque underlying the stenoses. In all of our patients, there was improvement in the symptoms. In addition, three of our patients had ongoing cerebral ischemia that resolved immediately after improvement of cerebral blood flow, with rapid return of neurologic function. This improvement was attributed to a return of

Table III. Immediate and long-term outcomes

<i>Patient No.</i>	<i>Immediate outcome</i>	<i>Long-term outcome</i>	<i>Most recent anatomic follow-up</i>
1	No new symptoms	Resolution of tinnitus, improvement in headache, no recurrent ischemia	L ICA: 12-mo A/G patent
2	Awakened, R hemiparesis resolved	Hemorrhagic conversion 13 d after intervention, with recovery; stable neurologic examination at 2 y after intervention	R and L ICA: 24-mo U/S patent
3	No new symptoms	Resolution of Horner syndrome and tinnitus, improvement in headache severity and frequency	R and L ICA: 24-mo A/G patent
4	No new symptoms	Resolution of intractable headaches	R ICA: 14-mo U/S patent
5	Transient ischemic attacks and TCD emboli resolved; placed on transplant list	Stable neurologic examination; died as a complication of transplantation 4 mo after intervention	R and L ICA: 3-mo U/S patent
6	No new symptoms, subtle R upper extremity weakness	Symptoms resolved	L ICA and R ICA/CCA: 12-mo U/S patent
7	Immediate improvement in hemianesthesia and hemianopsia	Minor residual visual field and sensory deficits	R ICA: 6-mo U/S patent

R, Right; L, left; ICA, internal carotid artery; A/G, angiography; U/S, ultrasonography; CCA, common carotid artery; TCD, transcranial Doppler ultrasonography.

penumbral tissue to normal or near-normal function, the removal of the embolic source, or both. None of these patients received thrombolysis.

There have been 8 previously reported series of patients with carotid dissections (both traumatic and spontaneous) treated with endovascular stent angioplasty (a total of 57 patients with 22 spontaneous dissections). In these series, no deaths occurred, no transient ischemic attacks or strokes occurred that could be attributed to the treated vessel, and patients remained clinically stable or improved during the follow-up period.^{6-9,12-15}

Only a minority of patients with SCD require such treatment. More than 85% of medically treated SCD patients improved clinically and angiographically in one study.⁵ The standard of care for SCD is anticoagulation with warfarin or antiplatelet therapy. Even patients in whom the dissection is severe and persistent have a low risk of recurrent stroke if the dissection is unilateral.¹⁷ Studies using magnetic resonance imaging to follow up on changes in arteries with spontaneous dissections also find stabilization or improvement in most cases.^{18,19} In some circumstances, however, medical therapy is inadequate (eg, patients with recurrent symptoms despite anticoagulation,⁶⁻⁸ patients with expanding or symptomatic pseudoaneurysm,^{7,9} and patients with involvement of multiple vessels or poor collateral vessels⁸) or contraindicated (eg, patients in whom anticoagulation is contraindicated because of a risk of bleeding^{6,7}). Multivessel involvement increases the risk of compromised cerebral blood flow⁸ and, in this series, may explain why our patients all had evidence of multivessel involvement. In addition, there is a group of patients in whom the degree of stenosis does not improve or actually worsens. This may be accompanied by additional symptoms, especially when more than one vessel is involved.²⁰

In the above-mentioned groups and in patients who experience additional symptoms while fully anticoagulated, there is growing evidence that an intervention should be considered. Surgical intervention has been used for the repair of spontaneous dissections, but it is technically challenging and is associated with higher morbidity and mortality than the repair of atherosclerotic lesions of the carotid.²¹ In one recent series, the surgical treatment of SCD in 49 patients was associated with a 12% incidence of stroke and death and a 58% incidence of cranial neuropathy.³ No examples of stroke or death associated with endovascular treatment of SCD have been reported in the literature. In our series, there were no immediate neurologic complications, although one of our patients had hemorrhagic conversion of a large infarct 13 days after the intervention. This may have been related to the tendency of large bland infarcts to develop hemorrhage, particularly in the setting of dual treatment with aspirin and clopidogrel. A second possibility is that the increased cerebral blood flow after endovascular repair and impaired autoregulation caused the rupture of an ischemic vessel wall, ie, hyperperfusion syndrome. This same patient had been nearly comatose before stenting as a result of severe bihemispheric ischemia and is currently alive and living at home. Endovascular treatment may therefore be the treatment of choice for patients with persistently symptomatic SCD,⁴ but because the natural history of most cases of SCD is so good, it is unlikely that most patients with SCD would benefit from endovascular therapy.

The clinical significance of a persistent pseudoaneurysm is uncertain. El-Sabrout and Cooley¹⁰ looked at 12 previously reported series and found that pseudoaneurysms had a combined major stroke and death rate of 21% when treated nonoperatively, whereas Touzé et al²² followed up

33 patients with pseudoaneurysms for 14 months and found no evidence of embolic events or rupture. Nevertheless, all of our patients with pseudoaneurysms were symptomatic: most had recalcitrant headaches, and one had recurrent cerebral ischemia. Several reports have used expanding or symptomatic pseudoaneurysms as a criterion for intervention.^{7,9} We used stent grafts to exclude and prevent expansion of the pseudoaneurysms and to reconstruct a normal lumen. In addition to technical success, this approach led to resolution of symptoms. Others have described stenting with noncovered stents followed by coil embolization for the treatment of pseudoaneurysms.²³ Unfortunately, this approach has been associated with aneurysm recurrence and is more time-consuming.¹⁴ Covered stent placement is relatively straightforward, although the selection of covered stents is somewhat limited. The use of the Jomed stent requires manufacturer approval because the stent is available for use only in coronary arteries on a humanitarian device exemption issued by the Food and Drug Administration.

We have clinical and imaging follow-up data for all of our patients. All of the patients have remained symptom free. The method of radiologic follow-up was selected on a case-by-case basis. Stents placed in the proximal cervical ICA or distal common carotid artery were well visualized by ultrasonography. More distal cervical and intracranial lesions cannot be visualized by ultrasonography, although both carotid duplex scan and transcranial Doppler ultrasonography can be useful because they can show a proximal high-resistance pattern (duplex scan) or a distal low-resistance pattern (transcranial Doppler ultrasonography) in cases of significant in-stent stenosis. The utility of magnetic resonance angiography as a follow-up technique for the detection of in-stent stenosis is limited by metal artifact caused by the stent and by the difficulty in visualizing the in-stent segment.²⁴ Similarly, CTA is limited by metal and bone artifact.²⁵ In such patients, catheter angiography is the most sensitive means of detecting in-stent stenosis. We did perform angiography in the patients with distal stents and did not find any evidence of restenosis or dissection in any of the patients. Restenosis may not be a major issue in this group of patients, because their stenoses were nonatherosclerotic. Even the patients with stent grafts have not had any in-stent stenosis or recurrence of pseudoaneurysms. Endovascular treatment therefore seems durable, and it is not clear that follow-up catheter angiography is absolutely necessary if patients are asymptomatic, but, again, this must be decided on a case-by-case basis.

The major limitations of this study are the small number of patients and the fact that it was a retrospective study. These limitations notwithstanding, our experience highlights the potential use of endovascular therapy in selected patients.

CONCLUSIONS

Our study is the largest series to date of patients with SCDs treated with endovascular stenting. It has provided

additional evidence for the safety and efficacy of this treatment when medical therapy is insufficient or inappropriate.

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INVITED COMMENTARY

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Drs Edgell, Abou-Chebl, and Yadav report a small but fascinating series of seven patients undergoing carotid stenting for the management of complications of spontaneous carotid dissection. With their experience and meticulous technique, the authors have achieved excellent results in patients facing potentially devastating strokes.

The article should be read carefully. The authors are not reporting the use of stenting in all, or even in most, cases of spontaneous dissection. In their experience only 7 (5%) of approximately 135 patients presenting with spontaneous dissection were selected for intervention. In fact, the title of the article is misleading. The authors are not reporting on the endovascular management of spontaneous carotid dissection, but rather on the endovascular management of complications of spontaneous carotid dissection. It is generally accepted, and the authors re-emphasize, that 85% to 90% of patients with spontaneous dissection recover very well with medical management alone. Furthermore, in most cases, arterial recanalization and remodeling occur after dissection with restoration of a reasonable luminal diameter. Recurrent dissection is unusual, and the overall prognosis usually quite good.

The small subset of patients selected for stenting in this series included those with expanding or symptomatic pseudoaneurysms or with severe flow compromise due to multivessel involvement. Other scenarios in which intervention seems reasonable include

ongoing symptoms despite anticoagulation and contraindication to anticoagulation.

Although this article addresses only patients with complications of spontaneous dissection, it provokes two significant questions. First, what is the role of stenting in patients who present with traumatic carotid dissection? In these patients, neurologic deficits may occur after an asymptomatic period of several hours or even days, and anticoagulation is often contraindicated. Should stenting be used early in these patients to prevent delayed neurologic sequelae and lessen concern over anticoagulation? Second, should we consider changing the default algorithm in the management of uncomplicated spontaneous dissection? The authors' results are so good in very compromised patients that it is tempting to think that stenting for uncomplicated dissection might result in an improvement over the 85% to 90% favorable results with traditional medical therapy. Obviously, only a randomized prospective trial can properly answer this question.

Although they reported only on the management of complicated spontaneous carotid dissections, the authors have demonstrated in very high-risk patients the potential of a new treatment for carotid dissection that calls into question our current management paradigm for lower-risk patients. Aggressive early stenting of traumatic and spontaneous carotid dissections now warrants careful study.