Intracranial Stenting

An overview of intracranial stents for the treatment of atherosclerotic disease and aneurysms and a glance into the promising future of newer devices.

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In the past few years, intracranial stents have provided a significant advancement in the endovascular treatment of many lesions. Currently, the most common indications for their use are intracranial stenosis/occlusion (intracranial atherosclerotic disease), intracerebral aneurysms (to assist coiling of wide-necked aneurysms), and arterial dissections.

INTRACRANIAL ATHEROSCLEROTIC DISEASE

The Wingspan Stent System with Gateway PTA Balloon catheter (Boston Scientific Corporation, Natick, MA) was developed specifically for treating ischemic atherosclerotic disease. It features a self-expanding design with flexible cells to enhance conformability and vessel wall apposition in curved and tapered vessels. The stent is designed to support the vessel lumen by minimizing vessel recoil after angioplasty and exerting active, controlled, outward radial force using thicker and shorter struts.

The available stent diameters and lengths of the Wingspan range from 2.5 to 4.5 mm and 9 to 20 mm, respectively. The device has also been used in the endovascular treatment of dissecting fusiform or wide-necked intracranial aneurysms with good results. The Wingspan device was granted humanitarian device exemption status by the FDA in 2005 to treat symptomatic patients who have failed medical management.

The NIH-sponsored SAMMPRIS trial[1] (Stenting versus Aggressive Medical Management for Preventing Recurrent Stroke in Intracranial Stenosis) is currently underway (enrollment not yet begun) to assess the efficacy of intracranial angioplasty and stenting using the Wingspan stent combined with intensive medical therapy over maximum medical therapy alone during a 2-year period.

The Wingspan stent[2] is deployed after predilation using a Gateway PTA balloon catheter, which provides controlled, low-pressure inflation (6 atm). The Gateway catheter is compatible with all .014-inch guidewires (300-cm length recommended) and guide catheters with an inner lumen of .064-inch diameter (6 F). The balloon diameters and lengths vary from 1.5 to 4 mm and 9 to 20 mm, respectively, with a burst pressure of 12 atm.

A summary of studies of the use of angioplasty for the treatment of intracranial atherosclerotic disease is shown in Table 1. Angioplasty usually results in subtotal vascular diameter restoration due to the eccentric location of the lesion and the risk for elastic recoil.[2] Multiple small studies have been performed to assess the efficacy of intracranial stents (Table 2).

In a comparison of angioplasty versus stent placement (95 patients with primary angioplasty and 98 intracranial stent placements), Siddiq et al[19] did not find any difference in

TABLE 1. SUMMARY OF CLINICAL PUBLICATIONS ON ANGIOPLASTY FOR TREATING ATHEROSCLEROTIC DISEASE

Study	No. of Patients	Follow-Up	Results	Vessel
Sundt et al[3] (1980)	1	24 mo	No recurrence; no restenosis	Basilar artery
Terada et al[4] (1996)	12	24 mo	Periprocedural complications, 33%; success, 8/12 (75%)	Intracranial vertebral, 8; basilar, 5
Hacein-Bey et al[5] (1998)	12	12 mo	Recurrent events, 16%	–
Marks et al[6] (1999)	23	35.4 mo	Success, 91%; stroke rate, 3.2% per year	Anterior, 10; posterior, 13
Connors et al[7] (1999)	70	3–12 mo	Success, 98%; late restenosis, 9%	Anterior, 44; posterior, 27
Gress et al[8] (2002)	25	Perioperative risk, 16%	Angiographic success, 100%	Posterior circulation
Marks et al[9] (2006)	120; stents in 16 patients when angioplasty failed	42.3 mo	Stroke rate, 3.2% per year; periprocedure stroke/death rate, 3.8%	Anterior, 51%; posterior, 49%

TABLE 2. SUMMARY OF CLINICAL PUBLICATIONS ON INTRACRANIAL STENTING FOR TREATING ATHEROSCLEROTIC DISEASE

Study	No. of Patients	Follow-Up	Stroke Rate (%)	Vessel Stented/Stent Used
Horowitz et al[10] (1999)	3	None	Pontine stroke, 1	Basilar artery/flexible coronary stent
Gomez et al[11] (2000)	12	5.9 mo	None	Basilar artery
Phatouros et al[12] (2000)	2	6.5 mo	No recurrence of symptoms	Basilar artery
Gomez et al[13] (2000)	1	1 mo	None	MCA/coronary stent
Wojak et al[14] (2006)	Angioplasty, 62; angioplasty + stent, 22	>6 mo	Periprocedure event rate, 4.8%; stroke rate, 1.8%	Anterior, 55%; posterior, 45%
Jiang et al[15] (2004)	40	10 mo	Recurrence, 0; restenosis, 1/8	M1 segment of MCA
SSYLVIA[16] (2004)	Neurolink (formerly Guidant, Indianapolis, IN); n=61, single symptomatic stenosis; intracranial arterial, 43; extracranial vertebral, 18	6 mo	Restenosis (>50%): intracranial, 32%; extracranial, 42%; stroke rate, 6.6 % (30 d), 13.1% (1 y)	Intracranial arterial, 43; extracranial vertebral, 18
Henkes et al[17] (2005)	15	6 mo	Stroke, 7.1%	Wingspan
Fiorella et al[18] (2007)	44; success, 95.7%	7 y	Periprocedure event rate, 26.1%; recurrent VBI, 15%	Balloon-mounted coronary stents

MCA indicates middle cerebral artery; VBI, vertebrobasilar territory ischemia.

restenosis rate or follow-up survival (stroke, or stroke and/or death) at 12 months. The stroke- and/or death-free survival rates at 24 months were 92%±4% and 89%±5% for the angioplasty- and stent-treated groups, respectively.

INTRACRANIAL ANEURYSMS

The first intracranial aneurysm stent safety trial was conducted in 2001. The Neuroform (Boston Scientific Corporation, Natick, MA) and the Enterprise Vascular Reconstruction Device (Cordis Neurovascular, Inc., Miami Lakes, FL) stents are available in the US for stenting wide-neck aneurysms.

The Neuroform stent was approved in 2002 as a humanitarian device for use with embolic coils for the treatment of wide-neck, intracranial, saccular aneurysms arising from the parent vessel with diameters ≥2 and ≤4.5 mm. Wide-neck aneurysms are defined as having a neck ≥4 mm or a dome-to-neck ratio ≤2). The Neuroform stent was the first self-expandable microstent specifically designed for the treatment of wide-necked aneurysms to prevent migration of coils deployed in the aneurysm lumen. It is made of nickel-titanium alloy. The Neuroform stent used an open-cell design, which had low radial force, resulting in inadequate support for the coil mass within the aneurysm, and several technical problems with stent delivery were reported, including misplacement of the stent into the aneurysm lumen or stent dislodgement during catheterization.

The Neuroform2 stent was launched in 2003, and the Neuroform2 Treo stent was launched in 2004. These were of an open-cell design as well and were also marred by technical problems with stent delivery and deployment. These problems include the invisibility of the stent meshes despite high-quality fluoroscopy, causing displacement of the struts into the aneurysm.

Launched in 2005, the Neuroform3 stent features a hybrid cell design, optimized for greater scaffolding and coil mass support. The Neuroform3 has an ultrathin, self-expanding design and segmented cell geometry to minimize foreshortening. The stent is designed for flexibility and conformability in tortuous distal anatomy. The stent is highly conformable with a segmented design for vessel apposition in tapered vessels. The interstices of the stent measure slightly <1 mm (2–2.5 F), allowing the placement of a microcatheter through the stent into the aneurysm sac. Also, the stent exerts minimal radial force on the surrounding artery—estimated at 10 mm Hg less than the coronary and carotid stents.

The stent is shown to be safe and compatible with magnetic resonance imaging, including 3 Tesla strengths.[2] More than 24,000 patients have been treated worldwide with this stent as of 2007—with more than 14,000 in the US alone. The Neuroform3 stent is not designed to treat an aneurysm with a neck >22 mm in length. It is available in a range of sizes from 10 to 30 mm in length with diameters from 2.5 to 4.5 mm. The Neuroform stents are not retrievable.

The Cordis Enterprise Vascular Reconstruction Device and Delivery System received humanitarian device exemption status from the FDA and was launched in 2007. The

Cordis Enterprise Vascular Reconstruction Device and Delivery System is used with embolic coils for the treatment of wide-neck, intracranial, saccular, or fusiform aneurysms. It consists of a self-expanding stent and a delivery system. The stent serves as a scaffold for embolic coils to prevent herniation of the coils into the parent vessel. The stent is a self-expanding, metal (nitinol) mesh in the shape of a tube. The delivery system is composed of an introducer and delivery wire and is used to deliver the stent to the treatment site in the neurovasculature. The major features of the device are ease of use, precise placement, wall apposition, and outstanding coil mass support. It is especially known for its navigability in tortuous anatomy. Due to its closed-cell design, the device can be recaptured once and redeployed. The device is available in one diameter, 4.5 mm, which can be used in vessels with a diameter of 2.5 to 4 mm. It is available in four lengths: 14, 22, 28, and 37 mm.

The Pipeline stent[2,20] (Chestnut Medical Technologies, Inc., Menlo Park, CA) is a newer device intended to exclude aneurysms from the parent vessel by reconstruction of the parent vessel. It is a braided, tubular, bimetallic microstent with smaller struts that has been shown to create significant flow disruption along aneurysm necks. It allows the preservation of both parent arteries and small adjacent perforating arteries. It is expected to be a significant improvement in the treatment of broad-neck aneurysms.

ARTERIAL DISSECTIONS

In 2008, Ansari et al[21] reported on nine patients (five men, four women; mean age, 50 y) with distal cervical/intracranial dissections treated with the Neuroform stent. All patients were symptomatic. Spontaneous (n=4) or traumatic/iatrogenic (n=5) dissections involved the internal carotid artery (n=2), vertebral artery (n=5), and vertebral-basilar artery (n=2). Dissections were treated with single (n=4), overlapping (n=2), or tandem (n=3) Neuroform stents. Dissection-related mean stenosis improved from 76% before the procedure to 23% after the procedure, with further reduction to 8% on follow-up imaging. There were no procedure-related complications.

Stent-assisted coil embolization of large dissecting aneurysms was performed in three patients, and spontaneous stent-induced thrombosis was noted in five small dissecting aneurysms. Two patients died due to sequelae—vertebral-basilar artery thrombosis—and delayed in-stent stenosis was seen in two patients. All seven surviving patients reported clinical improvement or resolution of symptoms and were followed with imaging and clinical assessment for a mean of 16.3 months.

The Pipeline stent, owing to its small struts and near covered-cell design, is expected to be a significant improvement in the treatment of intracranial dissections.

CONCLUSION

Management of intracranial vascular disease is still in its infancy. A variety of different stents have been developed in the past few years to help with effective disease management. Studies are still underway to assess their efficacy and usefulness in day-to-day clinical practice. Continued evaluation of the stents and their performance in intracranial disease is required to effectively assess their usefulness in individual patients. ■

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