PTA and Stenting of Extracranial VAS

Percutaneous angioplasty and stent placement should be the first treatment proposed for VBI caused by vertebral artery stenosis.

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Vertebral artery stenosis (VAS) is frequently encountered in patients undergoing coronary or aortic arch angiography.[1,2] The true incidence is unknown in the general population, but approximately 25% to 40% of patients with cerebrovascular disease are affected.[1,3] One quarter of all ischemic strokes occur in the territory of posterior circulation.[4] Twenty percent of posterior circulation infarcts are thought to be cardioembolic in origin, and a further 20% are due to intracranial embolism usually from the vertebral artery (VA).[5] In a series of 35 patients with occipital infarction, six had vertebral atheroma with presumed distal embolism.[6] So, there is evidence that posterior circulation atherosclerosis is implicated in ischemic events. Intra-arterial embolism is the most frequent mechanism of brain infarction[7-9] in patients with VA occlusive disease. Plaques in the VA show the same degenerative features as plaques that appear elsewhere, such as ulceration, intraplaque hemorrhage, and surface thrombus. The growth of a plaque may ultimately result in thrombosis of the VA. VA lesions may lead to insidious ischemic neurologic complications with potentially disabling or deadly consequences.

In an angiographic study of 4,748 patients with ischemic stroke, some degree of proximal extracranial VAS was seen in 18% of cases on the right and 22.3% on the left.[3] The most common site for a symptomatic lesion is the extracranial VA. Among the 407 New England Medical Center Posterior Circulation ischemic event registry patients, 52 patients (15 bilateral) had extracranial VA lesions.[7]

VAS can be asymptomatic, and the stenosis is often bilateral. Asymptomatic lesions are an uncommon cause of stroke and rarely warn of a transient ischemic attack (TIA). VAS can be symptomatic, with embolization of ruptured plaque, which creates a high risk of stroke. Clinically, patients may present with posterior fossa TIA or stroke and/or repetitive symptoms of vertebrobasilar insufficiency (VBI). Symptoms of VBI are often not well recognized by physicians, potentially leading to delay in treatment and other mismanagement. A large number of patients with VA disease may remain asymptomatic, but 50% present with a stroke alone, and 26% present with TIA rapidly followed by stroke.[10] Patients suffering from TIA have a 22% to 35% stroke risk over 5 years,[11,12] and the mortality associated with a stroke is 20% to 30% higher than for a carotid stroke.[13,14]

The risk of disease progression and development of a posterior fossa stroke within 5 years is estimated to range between 20% and 60%.[10] Clinically significant VAS needs to be treated. Medical treatment (antiplatelet or anticoagulation medications) has been proposed, but its efficacy is uncertain at best. Surgical procedures are associated with relatively high rates of mortality and complications. Endovascular procedures have been proposed and can now be performed with safety and efficacy; it is our therapy of choice, as it is in many centers. Balloon angioplasty alone is limited by severe elastic recoil with a high propensity for restenosis. The use of stents seems to improve immediate and long-term results. Drug-eluting stents (DES) have been proposed recently to reduce the restenosis rate.[15-19] As with carotid angioplasty and stenting, there is always a risk of brain embolization during the procedure. Recently, protection devices have been used to reduce this risk.[15,20,21]

DIAGNOSTIC IMAGING WORK-UP

The diagnostic imaging work-up of patients suspected of VBI should begin with a Duplex examination and should include magnetic resonance imaging (MRI) with or without magnetic resonance angiography (MRA) or computed tomographic (CT) scan of the brain with close evaluation of the posterior fossa and brain stem and CT angiography. MRA is useful as a screening test to evaluate stenoses of both intracranial and extracranial vessels. Angiography is essential, but given the inherent risk of invasive angiographic imaging, this technique should be limited to the interven-

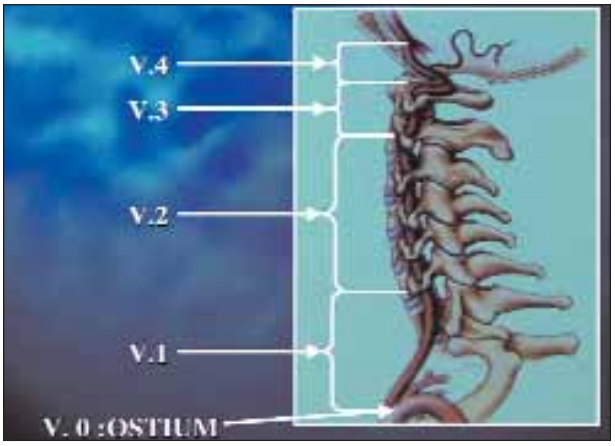

Figure 1. VAS location.

tional procedure itself and in cases of doubt, with other noninvasive techniques. Angiography is essential with a complete four-vessel arteriogram as well as the intracranial portion of both the posterior and anterior circulation before deciding whether a patient is a suitable candidate for percutaneous transluminal angioplasty (PTA). Angiography is also necessary to determine the extent of the lesion and evaluate it for evidence of ulceration, degree of stenosis, and presence of fresh intraluminal thrombus. Furthermore, angiography is needed to evaluate possible associated lesions in carotid (CA) and subclavian arteries (SA). Imaging of the cervicocerebral arch is also mandatory when planning access for any neurointerventional procedure.

The VA is divided into four segments (Figure 1). The first segment, the V1 segment, includes the origin of the VA (ostium or VO) upward to the level of entry into the foramen of the transverse process of the cervical vertebral body (usually the sixth cervical vertebra, C6). The second segment, the V2 segment or neck segment, courses upward to the level of the foramen of the C2 transverse process. The third segment, the V3 segment, is situated distal to the V2 segment, up to the atlanto-occipital membrane where the VA enters into the subarachnoid space. The V4 segment is the intracranial portion of the VA. The VA are often of asymmetrical diameter, one is hypoplastic and has a one-way end at the posterior inferior cerebellar artery; a rich cervical collateral is frequently present.

INDICATIONS FOR VERTEBRAL ANGIOPLASTY

Classically, patients were managed initially by conventional medical therapy. Only patients who failed to respond to this therapy were considered for PTA. At the present time, our treatment of choice is an endovascular procedure. A complete neurological history and examination must be performed on all patients before and after the procedure

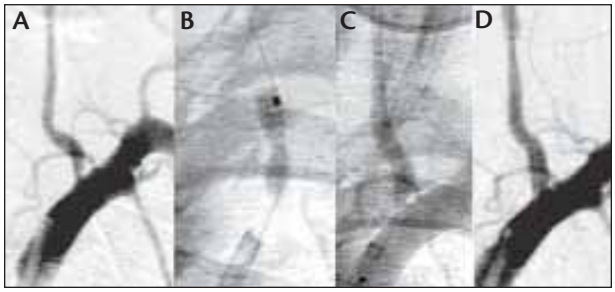

Figure 2. Left VO VAS before angioplasty (A). Balloon angioplasty with coronary balloon (B). Result after balloon angioplasty: residual stenosis (C). Final result after coronary stent implantation (D).

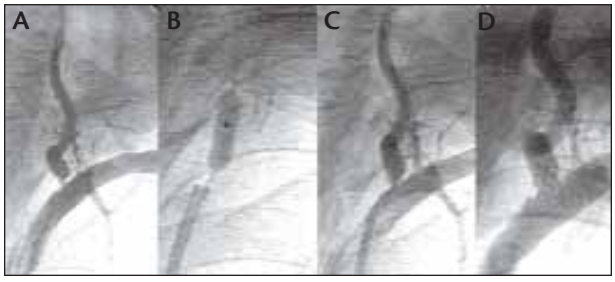

Figure 3. Left VO VAS before angioplasty (A). Balloon angioplasty (B). Result after balloon angioplasty: residual stenosis (C). Final result after peripheral stent implantation (Palmaz stent, Cordis Corporation, Bridgewater, NJ) (D).

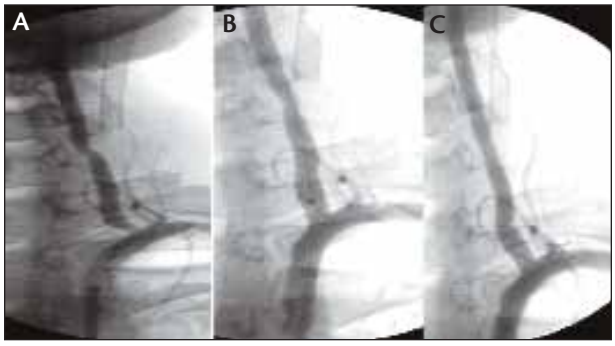

Figure 4. Left VO and V1 VAS before angioplasty (A). After angioplasty and stenting of VO lesion (Corinthian stent, Cordis Corporation) balloon angioplasty of V1 lesion: residual stenosis (B). Final result after stenting of V1 lesion with self-expanding stent (Optimed stent, Optimed Technologies, Inc., Fairfield, NJ) (C).

and during the follow-up. The current indications for the correction of extracranial VA lesions are:

- Symptomatic (TIA or no disabling ischemic stroke in the VA system) significant bilateral VAS causing > 60% diameter reduction;
- Asymptomatic unilateral significant stenosis of a dominant VA or significant stenosis with contralateral occlusion;

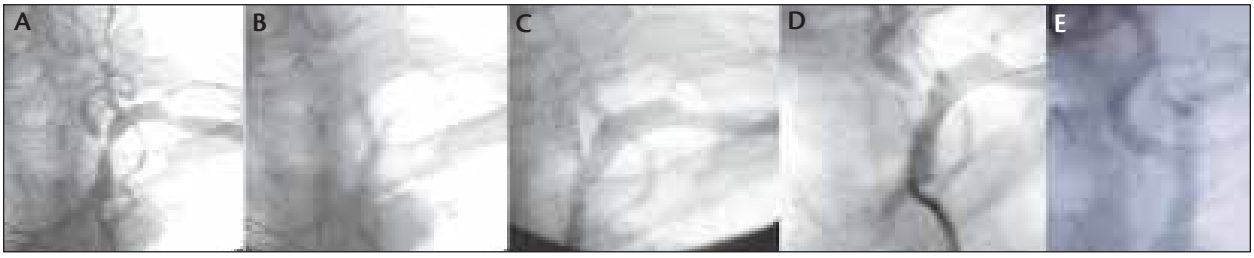

Figure 5. VA and SA stenoses before angioplasty (A). Kissing-balloon techniques (B). Palmaz stent implantation in the VA (C). Final stent result after Palmaz stent implantation in the VA and Wallstent in the SA (D). There was excellent patency of both arteries at 1-year follow-up (E).

- Asymptomatic significant VAS or tandem lesions with evidence of resting posterior fossa hypoperfusion or diminished cerebrovascular reserve may be considered for treatment due to high risk for infarction;
- Significant stenoses in an asymptomatic patient who needs collateral support (eg, concurrent [bilateral] CA occlusion);
- Asymptomatic patients with high-grade (> 70% stenosis) lesions or progressive severity of the stenosis are associated with an increased risk for stroke and would benefit from treatment, especially those with disease of either the dominant VA or a single VA.

ANGIOPLASTY AND STENT PLACEMENT

The procedure is performed under local anesthesia and, if necessary, intravenous conscious sedation and analgesia. Continuous neurological monitoring throughout the procedure is performed to quickly recognize any neurological complication.

Access

Percutaneous access through the femoral artery is used in the majority of cases. Brachial access is used in cases of severe lower extremity atherosclerosis, severe arterial tortuosities, unfavorable anatomy of the aortic arch with severe angulation of the VA origin, and when femoral access fails. The radial approach has recently been proposed.[22,23]

Techniques

The techniques used in angioplasty and stent placement are illustrated in Figures 2 through 5. Using the femoral approach, depending on the artery and the lesion—aortic arch type, SA anatomy, angulation between the VA and SA—a 6- to 8-F guide catheter or long introducer sheath is inserted, and systemic anticoagulation is achieved by administering intravenous heparin starting with 50 to 70 U/kg to attain an activated coagulation time > 250 seconds. An appropriately shaped guide catheter (Multipurpose, VBA catheter, right Judkins) is positioned in the SA just proximal to the ostium of the VA to be treated.

For better control and support and to prevent the guide catheter from losing its position, an 0.018-inch extra-support buddy wire can be placed in the ipsilateral axillary artery. Quantitative angiography is performed to evaluate the lesion, the degree of the stenosis, and measure the diameter of the vessel to size balloons and stents. The stenosis is crossed with a 0.014- or 0.018-inch coronary wire. The wire should be positioned far enough distally so that it is stable. The tips of the wire should be visualized during the entire procedure to reduce the risk of perforation. Two techniques may be performed: (1) predilatation of the stenosis followed by stent implantation or (2) direct stenting. Predilatation is useful in case of a very tight or calcified stenosis. Angioplasty is performed with a coronary balloon (2–3 mm in diameter). The balloon should be inflated for no more than 10 seconds to avoid induction of further ischemia in an already compromised area.

After angioplasty, an angiography control is completed to evaluate its results and select the stent. The choice of the stent is largely governed by the location of the lesion and its anatomy. Balloon-expandable stents are used to treat VA ostial stenoses. For large vessels (> 4 mm in diameter), peripheral stents used for renal stenting mounted on low-profile balloons can be implanted. Most of the time, we use coronary stents.

Precise placement of the stent and good visualization (oblique, cranial projection) of the ostium are required. Contrast media injection through the guide catheter facilitates the precise positioning of the stent. For ostial lesions, the stent must be placed 1 mm (maximum) inside the lumen of the SA. The stent need not be implanted too distal (missing the lesion) or too proximal (protruding too much in the SA); doing so could lead to difficulties for further reaccess. New devices, such as the ArchStent ostial system (SquareOne, Inc., Campbell, CA), have been proposed to treat ostial lesions, allowing an accurate positioning of the stent, good ostial coverage, and an easy reaccess to the vessel. Stents are deployed at high pressure (10–18 atm). Self-expanding stents are reserved for nonostial VAS. Precise placement of these stents could be difficult at the ostium of

the VA. Stents with a monorail design are preferred to over-the-wire stents to simplify the technique. The stent diameter should be sized to the VA distal to the lesion, and the stent length should be sized to cover the entire lesion. Overdilation should be avoided to decrease the risk of dissection or embolization.

Protrusion of the stent into the SA is necessary in cases in which we have to treat plaque within the SA that is contributing to the VA ostial lesions. After balloon placement via the brachial approach, a similar technique is used. A postprocedure arteriography is always performed to evaluate the results of the procedure to exclude complications and distal embolization into the intracranial circulation. Close neurological evaluation is performed during and immediately after the procedure as well as the day after. Patients are followed neurologically for 6 months postprocedure and then annually.

In case of associated VA and SA stenosis (Figure 5), a kissing-balloon technique can be performed by femoral approach. A coronary wire is placed into the VA, and a coronary balloon is advanced over this wire. Another guidewire is placed in the SA with a dilatation balloon over it. The two balloons are placed at the site of the VA and SA stenoses and simultaneously inflated. The decision for stent placement is made by evaluating the angioplasty results after deflation and withdrawal of the balloons. The coronary techniques for "bifurcation" lesions can be used to implant the stents.

Protection Devices

Several studies confirm that VAS, like carotid stenosis, has the potential for embolization. Friable ulcers on atheromatous plaques and thrombi may be responsible.[24-26] Protection devices have been proposed to protect the brain[15,20,21] and could be used at least in specific situations with patients at high risk for brain embolization. The three types of protection devices (protection balloons, filters, and flow-reversal devices) can be used during PTA and stenting of the VA. Nevertheless, difficulties could occur when recovering the device from unfavorable angulations of the VA origin after stent deployment. We recommend using distal protection devices in large VAS (diameter > 3.5 mm) with more favorable geometric orientation of the VA origin. With unfavorable angulations of the left VA, a reversal flow device (Gore Flow Reversal system, W.L. Gore & Associates, Flagstaff, AZ) can be used to create a temporary subclavian steal syndrome[27] and reduce the risk of brain embolization.

Medications

Aspirin (160 mg/day) and either ticlopidine (250–500 mg/day) or clopidogrel (75 mg/day) are given at least 3 days before the procedure, and the same treatment is continued for 4 weeks. Thereafter, only aspirin is given.

PERSONAL SERIES RESULTS

Patient Characteristics

Ninety-three VA angioplasties were attempted in 87 patients (65 men, 22 women age, ranging from 22 [the youngest patient had inflammatory arteritis] to 82 years [mean age, 67.9 ± 7 years]). One patient had three lesions in the same VA at the V0, V1, and V2 segments. One patient had two lesions in the V0 and V1 segments. Three patients had bilateral VAS at the V0 segment. In total, 86 lesions at the V0 segment (ostium), five at the V1 segment, and two at the V2 segment were treated (left, 52; right, 41). Ninety-one lesions were atheromatous, two of which were due to inflammatory arteritis. Twelve lesions were calcified, and nine were ulcerated.

All patients were symptomatic, exhibiting dizziness (n = 87), bilateral weakness (n = 11), visual disturbances (n = 11), diplopia (n = 10), drop attacks (n = 17), TIA (n = 10), ataxia (n = 5), and dysarthria (n = 2). Seventeen VAS were associated with severe SA stenoses. Five lesions involved the origin of the VA, and 12 were located at the ostium of the SA. These SA stenoses were responsible for arm claudication in 17 cases and recurrent angina pectoris in five patients presenting with left internal mammary artery bypasses. These VAS were treated during the same procedure. Other associated diseases were present—60 patients (69%) had carotid stenoses, 59 (68%) had coronary diseases, 23 (26%) peripheral vascular diseases, and 12 (13%) had renal stenoses necessitating interventional procedures.

Other comorbidities included 44 patients (51%) with hypertension, 42 smokers (48%), 40 (46%) with elevated cholesterol (> 200 mg/dL), 20 (23%) with diabetes mellitus, and 13 (15%) with obesity. Seven patients (8%) had congestive heart failure, four (5%) had renal insufficiency, and three (4%) had pulmonary insufficiency.

Technique

Femoral access was used in all cases. Failures to access the VA occurred in three cases (3.2%) due to severe tortuosities of iliac arteries and supra-aortic vessels. In these three cases, catheterization of the VA was attempted by a brachial approach, which also failed in two cases due to the same problem of vessel tortuosities. These two high-risk patients were treated medically. Six lesions were treated by PTA alone: three were V0 lesions (the first three patients), two were V1 lesions, and one was a V2 lesion. One V0 lesion, in the patient presenting with inflammatory arteritis, was treated with a cutting balloon. Eighty-four lesions were treated with stents: peripheral balloon-expandable stents (n = 19), self-expandable stents (n = 4)

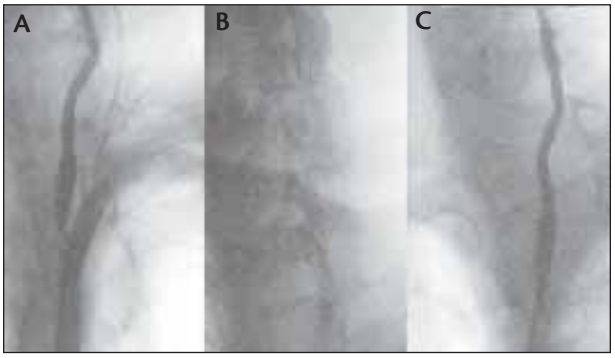

Figure 6. Left VO VAS. Angioplasty stenting under protection. Tight ulcerated lesion (A). FiberNet filter (Lumen Biomedical, Inc., Plymouth, MN) in place (B). Final result after angioplasty and stenting (C).

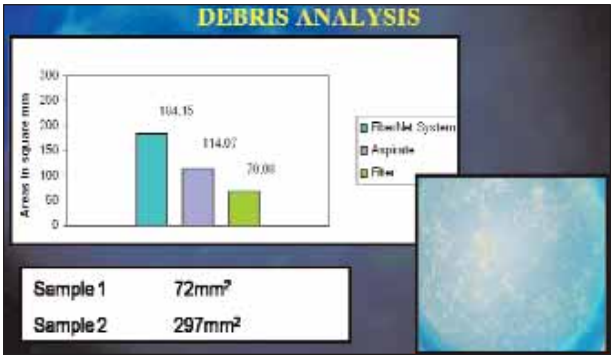

Figure 7. VA angioplasty under protection with FiberNet debris analysis.

for three V1 lesions and one V2 lesion), and 61 coronary stents (nine drug-eluting sirolimus stents). Sixty lesions were treated by direct stenting, 15 after balloon dilatation.

Seventeen SA stenoses were treated during the same procedure. Twelve lesions located at the ostium were treated with balloon-expandable stents, and five lesions involved the origin of the VA using the kissing-balloon technique via the femoral approach and then stenting. A protection device was used in eight patients (six men, mean age 69 years; left, six) (FilterWire EZ embolic protection system [Boston Scientific Corporation, Natick, MA], in six patients; FiberNet embolic protection system [Invatec, Inc., Bethlehem, PA], in two patients) presenting with tight ulcerated ostial VAS. No difficulties were observed to place and remove the filter. Visible debris was seen in six patients (75%)—four treated with the FilterWire and two with the FiberNet filter. With the FiberNet device, a large amount of debris was removed (Figures 6 and 7). The amount of debris removed was comparable to that of carotid angioplasty and stenting.

Angiographic Results

The results are illustrated in Figures 2 through 5. The VA reference size was 4.8 ± 0.6 mm (range, 4–6). The mean percentage of stenosis before the procedure was 82.8 ± 7.9% (70–98); the mean lesion length was 9.6 ± 2.7 mm (range, 5–14), and the minimum lumen diameter (MLD) was 1.11 ± 0.47 mm.

Procedural success—defined as < 20% diameter stenosis without any major neurological event, emergency surgery, or death after angioplasty and stent placement—was achieved in 91 of the 93 cases. The poststent MLD was 4.55 ± 0.8 mm, the mean residual stenosis 2.2 ± 3.5%, and the acute gain 3.44 ± 0.9 mm. The post-PTA MLD was 3.96 ± 0.9 mm, the mean residual stenosis 13.1 ± 6.5%, and the acute gain 2.85 ± 0.9 mm (Table 1).

Procedure-Related Results and Complications

During the procedure, one patient presented with a TIA. No myocardial infarction, stroke, or death was seen during the 30 days after the procedure. None of the patients experienced cranial nerve palsies, wound infection, bleeding requiring transfusion, significant brachycardia, hypotension, or loss of consciousness after treatment with balloon inflations and stent placement. No significant bradycardia hypotension or cranial nerve palsy was seen after carotid stenting. We observed a significant clinical improvement with complete resolution of the clinical symptoms in all patients.

Follow-Up

Patients had a careful follow-up after the procedure. Vertebral duplex sonography and neurological examinations were performed the day after the angioplasty, at 1 month, 6 months, 1 year, and each year thereafter. A control angiography was also performed if a restenosis was suspected. The results of clinical follow-up are available for 75 patients at a mean of 32.4 ± 28.6 months (eight patients died, two were lost to follow-up). During this period, six patients (8%) had neurological symptoms due to a restenosis as confirmed by Duplex scan and angiography. These restenoses included one total occlusion of the VA treated medically and five tight restenoses (stenosis > 70%), which were treated successfully by new angioplasty.

TABLE 1. RESULTS AFTER PTA ALONE AND PTA STENTING

	Baseline	Post-PTA	Poststenting	*P* Value
MLD (mm)	1.11 ± 0.47	3.96 ± 0.9	4.55 ± 0.8	< .001
Diameter (%)	82.6 ± 7.9	13.1 ± 6.5	2.2 ± 3.5	< .001
Acute gain (mm)		2.85 ± 0.9	3.44 ± 0.9	< .01

Abbreviations: MLD, minimum lumen diameter; PTA, percutaneous transluminal angioplasty.

The clinical symptoms resolved after the procedure. Three restenoses appeared in our first three patients treated by PTA alone and three restenoses after PTA and stent. None of the patients treated with DES restenosed, but the follow-up (8 ± 3 months) was not long enough to draw any conclusions.

DISCUSSION

VBI is probably an underdiagnosed clinical condition because patients often have nonspecific symptoms. With new noninvasive techniques (Duplex scan, MRI, CT scan), the diagnosis has become easier, and a stenosis of the VA is more and more suspected, leading to angiography, which remains the main technique to confirm the presence of a lesion in the VA territory.

Medical treatment alone has been the standard treatment for patients suffering from VBI, but its efficacy is not proven to be superior to surgery or endovascular therapy. Several surgical procedures have been proposed (endarterectomy, vertebral reimplantation in the common carotid artery, and bypass), but the mortality, morbidity, and complication rates remain too high.[28] Given the difficulties in operating on the VA, a percutaneous endovascular approach (angioplasty) has been proposed to treat VAS. The initial radiological and clinical results were uniformly reported to be beneficial to the patient.[2,15,20,29-31]However, in most of the cases, the stenosis was not fully dilated to the normal diameter of the VA.[29] If one carefully reviews previous reports on vertebral ostial angioplasties, most illustrations show residual stenosis after the procedure. It has been well recognized that certain lesions, particularly those located at the ostia of coronary and renal arteries, have severe elastic recoil that limits the success of PTA. At the ostial VA level, this may be due in part to adjacent subclavian plaque encroaching on the orifice of the VA. The stenosis at the ostium of a relatively small artery that originates from a sig-

TABLE 2. VERTEBRAL ANGIOPLASTY AND STENTING

Investigators	Year	Lesions (No.)	Technical Success (%)	Mean Follow-Up (Mo.)	Restenosis (%)
Higashida[31]	1993	34	100	NA	9
Malek[32]	1999	13	100	20.7	25
Chastain[33]	1999	55	98	25	10
Jain[34]	2000	73	97	12	5.6
Mukherjee[35]	2001	12	100	6.4	8.3
Jenkins[36]	2001	73	100	10.6	3
Chiras[37]	2002	13	100	12	7.7
Cloud[38]	2003	10	100	19.7	10
Albuquerque[39]	2003	33	97	16.2	43.3
Mathias[40]	2003	278	97	NA	20–30
Lutsep[41]	2003	14	94	6	43
Ko[42]	2004	25	100	25	16
Janssens[43]	2004	16	100	30	25
Kisilbilic[44]	2004	14	100	NA	0
Hauth[45]	2004	16	87	NA	NA
Lin[16]	2004	68	100	11	25
Hatano[46]	2005	101	99	< 6	9.5
Dabus[47]	2006	28	92.8	24	26
Du[20]	2007	48	97.9	22.4	34.6
Seifert[48]	2009	29	100	24.1	52

Abbreviation: NA, not available.

nificantly larger artery has a natural tendency for elastic recoil. This elastic recoil and the residual stenosis after balloon angioplasty alone favor the restenosis. To overcome this problem of elastic recoil and atherosclerotic plaque overlap into the SA and to improve the long-term results, stenting was proposed for the treatment of VAS. Another advantage of primary stenting is a reduced rate of dissection.

For ostial lesions, balloon-expandable stents seem to be a good option—either peripheral or coronary stents—depending on the diameter of the artery. These stents have a good radial force, short length, good radiopacity, and allow precise positioning. When the proximal V1 segment is very tortuous or kinked, flexible stents may be used, especially coronary stents; a stiffer stent may cause kinking of the artery after stenting. Appropriate stent placement in the ostium of the VA may need multiple angiographic projections or use of three-dimensional rotational angiography to delineate the true ostium, because the artery comes off posteriorly and superiorly from the SA. For lesions in the V2 segment, with bone surrounding the vessel, self-expandable stents such as the Wallstent (Boston Scientific Corporation) are better to avoid compression by the bone during the neck movements. For ostial lesions, the most important technical detail of this technique is proper, precise stent placement. The stent is intentionally placed, minimally projecting into the SA. Meticulous attention to the precise relationship of the proximal edge of the stent to the SA is essential before deployment. The procedure needs experienced interventionists to choose the stent and have appropriate stent placement at the ostium of the VA, because the tortuosity of the VA may be technically challenging. New coronary stents seem to be well suited to treat atherosclerotic lesions of the origin and the proximal VA. To date, the series with primary stenting of VAS have shown high levels of technical success—97% to 100% in most of the published series (Table 2)—and high levels of clinical success; the percentage of asymptomatic patients remains high (> 90%) during the follow-up period in most of the series. Vertebral angioplasty and stenting appears to be a safe procedure. The complication rates and the death-stroke event rates occurring during the in-hospital stay are low (Table 3). Some TIAs were reported.[32,36,46,49] Stroke and death are rarely reported.[6,18,20,32,36]

The essential issues involved in the long-term follow-up of patients who undergo VA angioplasty and stenting are the frequency of in-stent recurrent stenoses and the presence or absence of symptoms of VBI. The rate of restenoses varies from one series to another (3%–52%), but VA recurrent lesions are largely asymptomatic. The most important and recently published series with bare-metal stents (BMS) are reported in Table 2.

In the Stenting of Symptomatic Atherosclerotic Lesions in the Vertebral or Intracranial Arteries (SSYLVIA) trial,[50] 43% of the patients showed evidence of restenoses, and half of them had complete occlusion of the vessel. For Lin et al,[51] the reference vessel diameter is the only independent predictor of restenosis. DES have been recently proposed and used to reduce the restenosis rate. These stents dramatically reduce the incidence of restenoses in coronary artery lesions, especially in diabetic patients. The first published data in VA stenting seem promising. Akins[17] treated five patients with a tacrolimus stent and seven patients with a BMS; at 1-year follow-up, three patients developed restenosis with BMS and none with DES.

Lin et al[51] implanted 11 DES in 11 patients (eight paclitaxel DES and three sirolimus DES) without any periprocedural complications. At a mean follow-up time of 18.7 ± 8.6 months, all patients were asymptomatic. No restenosis was reported, but follow-up angiography was performed only if restenosis was suspected or for later catheterization for other indications. Edgell et al[18] reported five cases of DES without any significant restenosis. Yu et al[19] treated 10 patients with paclitaxel stents. No restenosis was detected at 1-year follow-up. As previously reported, we implanted nine sirolimus stents, and we observed no restenosis at 1-year follow-up. Some stent fractures were recently reported with sirolimus stents.[52] Nevertheless, DES may be the solution to reduce the restenosis rate, as in coronary procedures.

As previously mentioned, all these series showed that the periprocedural complication rate is low, but potential complications include emboli originating from atheromatous plaques (eg, carotid bifurcation location). When emboli are washed into the VB system, a stroke may result. To reduce the risk of brain embolization, protection devices have been recently proposed just as for carotid angioplasty. Divani et al[53] compared carotid angioplasty and stenting (16 procedures) and vertebral angioplasty and stenting (14 procedures) performed with protection devices. They found that the frequency and amount of captured emboli during stent procedures are comparable, and that there is no significant difference in the characteristics of the debris between the two vascular regions. The investigators concluded that the use of emboli protection devices for vertebral angioplasty and stenting may be advisable.

Qureshi et al[15] treated 12 symptomatic VAS with the EPI FilterWire EZ embolic protection system (Boston Scientific Corporation). Femoral and radial approaches were used in nine and three cases, respectively. Technical success was achieved in 11 patients. Eight devices held macroscopically visible embolic debris (large and small amounts in three and five devices, respectively). No stroke or death was observed in the 1-month follow-up. We reported in our series the use of protection devices in eight patients presenting with high

TABLE 3. COMPLICATIONS

Investigators	Years	Patients (No.)	Complications
Chastain[33]	1999	55	0%
Malek[32]	1999	13	15% (1 stroke, 1 TIA)
Zhang[49]	1999	16	6% (1 TIA)
Jain[34]	2000	54	0%
Jenkins[36]	2001	73	4% (1 TIA, 1 stroke, 1 death)
Mukherjee[35]	2001	12	0%
Maini[54]	2001	14	7.1% (1 subarachnoid hemorrhage)
Lin[16]	2004	58	5.1% (3 strokes)
Ko[42]	2004	25	0%
Hotano[46]	2005	101	2% (2 TIAs)
Dabus[47]	2006	25	0%
Du[20]	2007	48	2% (1 stroke)
Ralea[55]	2008	12	8.3% (1 brain hemorrhage)
Akins[17]	2008	12	0%
Edgell[18]	2008	287 (Meta-analysis)	2.10% (2 strokes, 4 TIAs)

Abbreviation: TIA, transient ischemic attack.

embolic risk and ulcerated lesions. Visible debris was removed in six cases, and with the FiberNet embolic protection device, we removed the same amount of debris that would be removed during carotid angioplasty.[56] The use of protection devices seems promising.

Three controversial issues remain and are well described by Chastain:[33]

- The need for intervention in asymptomatic patients found incidentally to have a stenotic VA lesion. These patients are treated because of the perceived need for the VB system to provide hemodynamic or collateral support. It is also necessary to consider that, although they are believed to be asymptomatic, many of these patients may have nonspecific symptoms, such as dizziness, that may be alleviated by restoring adequate perfusion pressure to the VB system.
- The need for intervention in case of a stenotic VA with a normal contralateral VA. Although the hemodynamic effect of the stenosis on brain stem perfusion can be easily compensated for by the normal contralateral vessel, the risk of in situ thrombus formation and distal embolization is not eliminated. This pathogenic mechanism is reported to be a factor in approximately 25% of patients with VBI more frequently than in those with unilateral lesions.[5]
- Whether the intervention should be reserved for patients in whom medical therapy has failed. We have no prospective randomized data. The treatment with both antiplatelet and anticoagulating drugs is at low risk but does not treat the distal hemodynamic compromise caused by stenotic VA lesions. This is significant enough to have been reported to cause VBI in 16% of the patients with this diagnosis.[10]

CONCLUSION

Percutaneous angioplasty and stent placement seem to be a useful technique for the treatment of VBI caused by VAS and the first treatment to be proposed. This technique appears safe and effective for alleviating symptoms and improving blood flow to the cerebral circulation with a low complication rate and good long-term results. However, this procedure needs experienced interventionists to choose the stent and place it appropriately at the ostium of the VA. The new coronary stents seem to be well suited to treat atherosclerotic lesions of the origin and of the proximal VA. A large variability of restenosis risk has been reported—DES may be the solution. The role of brain protection devices has to be defined at least in some high-risk patients. Further prospective, randomized studies are needed to demonstrate the clinical effectiveness of this procedure in stroke prevention, its durability, and to define more clearly its indications. ■

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