

Stenting for vertebrobasilar artery stenosis

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Summary

We report our experience with stenting for symptomatic vertebrobasilar artery stenosis. One hundred and sixteen patients with vertebrobasilar artery stenosis (101 vertebral ostial stenosis, 15 intracranial vertebrobasilar artery stenosis) were treated with stenting. Indication criteria of treatment were 1) symptomatic lesion, 2) angiographical stenosis more than 60%. Under local anesthesia, pre-dilatation was first performed, then stents were placed to the lesion. Successful dilatation was obtained in 115 cases. The stenosis rate reduced to 2% post-stenting in ostial lesions and 16% in intracranial lesions. Transient neurological complications developed in 2 patients. Follow-up angiographies more than 6 months after stenting were performed in 94 patients with ostial lesions and all patients with intracranial lesions. Of these, 8 patients (9.5%) with ostial lesions and 4 patients (27%) with intracranial lesions developed restenosis. All patients with restenosis were treated successfully with PTA (percutaneous transluminal angioplasty). During the follow-up period, 3 patients developed recurrence of VBI (vertebro-basilar insufficiency) symptoms due to restenosis. One patient developed brain stem infarction due to in-stent occlusion 8 months after stenting.

Conclusion. Stenting for vertebrobasilar artery stenoses is feasible and safe. Prevention of restenosis, especially in intracranial arteries, is the next problem to be solved.

Keywords: Stent; vertebrobasilar artery stenosis.

Introduction

In the treatment of vertebrobasilar artery insufficiency, medical therapy had been the mainstay of treatment because of the high morbidity rate associated with surgical treatment [1, 7, 12]. Recently, percutaneous transluminal angioplasty (PTA) has evolved to a viable treatment of these lesions. PTA, however, sometimes induces wall dissection and elastic recoil, which results in insufficient dilatation, or, on rare occasions, abrupt closure. Furthermore, the restenosis rate after PTA is high [3, 6, 10, 14]. Using stents may resolve these problems. We investigated the feasibility, safety and outcome of stenting for vertebrobasilar artery stenosis.

Stenting for vertebral artery ostial stenosis

Patients and methods

A total of 101 patients with symptomatic vertebral artery ostial stenoses were treated with stenting in our hospital between October 1997 and April 2004. These patients consisted of 85 males and 16 females with a mean age of 71.5 years (range 46–84 years). Indications for stenting were clinically symptomatic patients having over 60% angiographical stenoses.

Procedure

Aspirin and ticlopidine were administered for at least one week before treatment and one month after treatment. In all but one case, the endovascular technique was performed via a transfemoral approach under local anesthesia. After placement of femoral artery access sheaths (8F-25 cm or 6F-90 cm), heparin was administered. During the procedure, the patient's activated clotting time was adjusted to greater than 250 seconds. A guiding catheter (90–100 cm in length, 8F in diameter) or an ultra-long sheath (90 cm in length, 6F in diameter) with a coaxial catheter (125 cm in length, 6F in diameter) and a guidewire was guided to the subclavian artery just proximal to the ostium of the vertebral artery. Firstly the lesion was dilated with an undersized semicompliant balloon. After predilatation, we evaluated the characteristics of the plaque and the normal size of the vessels using intravascular ultrasound (IVUS). Then, an appropriate stent was applied to the lesion. Balloon-expandable type stents were used in all cases. After careful positioning, the stent was deployed. Figure 1 illustrates what we consider to be the best position for stents. Re-

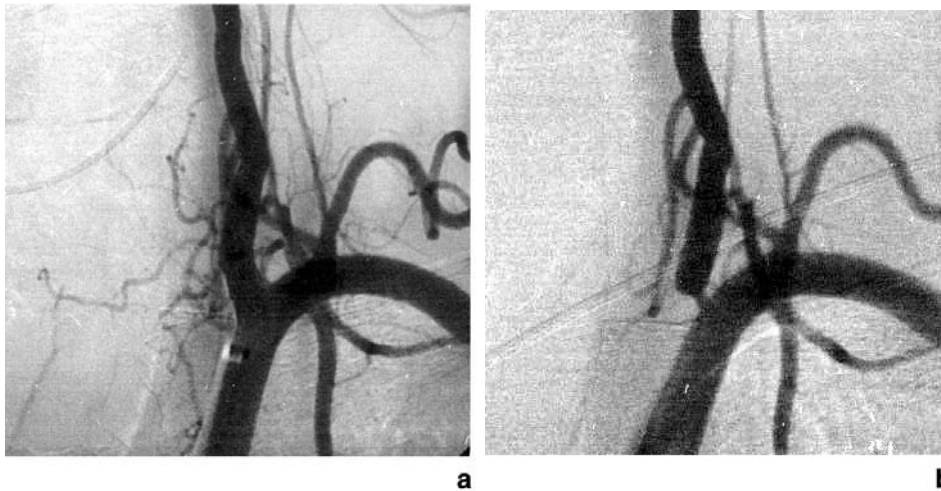


Fig. 1. (a) Anteroposterior left subclavian angiogram demonstrating left vertebral artery ostial stenosis. (b) Angiography after stenting showed excellent dilatation of the lesion

cently, we moderately over-dilated stents, ranging from 0.3 mm to 1 mm. Follow-up angiography was performed six months after stenting. Restenosis was defined as greater than 50% stenosis.

Results

Successful dilatation, defined as less than 30% residual stenosis, was obtained in 100 of 101 cases (99%). The stenosis rate, which was 81% pre-stenting, reduced to 2% post-stenting. The angiograms just after stenting did not show any wall dissections of lesions or any distal occlusions of major intracranial branches. Transient neurological complications occurred in 2 patients (hemiparesis, visual acuity disturbance). No patients experienced permanent neurological complications. Follow-up angiographies more than 6 months after stenting were performed in 94 patients. Of these, 8 patients developed restenosis (9.5%). All patients with restenosis were treated successfully with PTA. As for clinical symptoms, 58% of total patients improved 30 days after stenting. During the follow-up period, two patients developed recurrence of transient VBI symptoms due to restenosis. No patients developed any strokes in the posterior circulation.

Representative case

A 71-year-old man with a history of hypertension and angina developed vertigo and hemiataxia and

consulted our hospital. MRI showed a left cerebellar infarction and angiography showed right vertebral artery occlusion and left vertebral artery ostial stenosis (Fig. 1 a). Ten days after admission, we performed stenting for left vertebral artery stenosis. A guiding catheter was positioned in the left subclavian artery. After predilatation, a Palmaz stent (1 cm in length) was applied to the lesion. Angiography after stenting showed excellent dilatation of the lesion (Fig. 1 b). A follow-up angiogram 6 months after stenting showed no restenosis. The patient's symptoms almost disappeared 3 months after stenting.

Stenting for intracranial vertebrobasilar artery stenosis

Patients and methods

Twelve patients with intracranial vertebral artery stenosis and 3 patients with basilar artery stenosis were treated with stenting. These patients included 12 males and 3 females, with a mean age of 68.2 years (53–79 years). Indication for endovascular treatment of intracranial arterial lesions was clinically symptomatic patients with over 60% angiographical stenoses. In the treatment of intracranial artery stenosis, PTA was firstly performed in all patients. Stenting was performed only in cases with insufficient dilatation, dissection or restenosis after PTA.

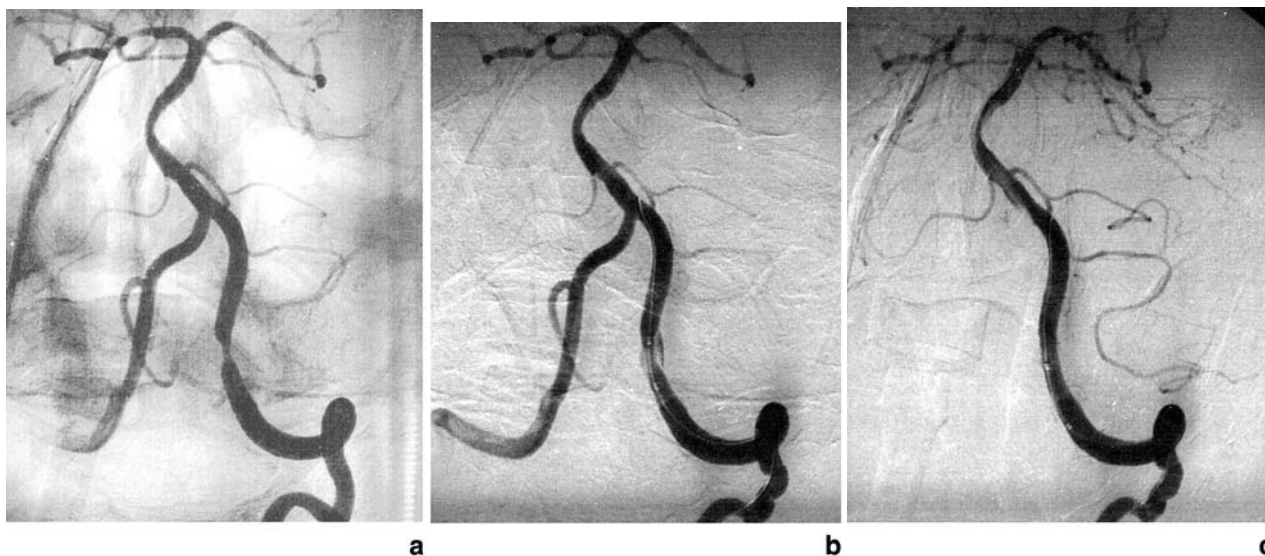


Fig. 2. (a) Anteroposterior left VA angiogram demonstrating stenosis of the left intracranial vertebral artery. (b) Angiography after PTA showed wall dissection (arrow). (c) After stenting, the lesion was sufficiently dilated

Procedure

Stenting methods for these lesions were basically the same as those for ostial lesions. Percutaneous access was achieved via a femoral artery, where a 6F or 8F sheath was introduced. A 6F guiding catheter was advanced up to the level of the second cervical vertebra. Using a load-mapping technique, a 0.014 inch wire was advanced to the second portion of one of the posterior cerebral arteries. PTA was performed with a 2.0–3.0 × 9.0 mm coronary balloon. The stents we used in intracranial lesions were flexible and had a low profile (S670, S660 stent). Stents were expanded to a diameter slightly less than the diameter of the lumen of the normal distal artery. Angiographical follow-up was performed 1 month, 3 months and 6 months after stenting.

Results

Technical success rate was 100% and complication rate was 0%. The stenosis rate, which was 84% pre-stenting, reduced to 16% post-stenting. Four patients (27%) developed restenosis. All patients with restenosis were treated with PTA successfully. In all, 80% of patients were clinically improved 30 days after stenting. Two patients developed transient episodes of VBI due to restenosis within six months of stenting. One patient developed in-stent occlusion 8 months after stenting and 2 months after the 2nd PTA for restenosis. This patient suffered a major stroke.

Representative case

A 78 year-old male with a history of hypertension and diabetes mellitus presented with dizziness and ataxia. MRI imaging revealed a small area of infarction within the left cerebellar hemisphere. His angiography revealed left vertebral artery stenosis (Fig. 2 a). Endovascular treatment was performed 3 weeks after onset. A 6F guiding catheter was positioned at the left vertebral artery. The lesion was crossed with a 0.014-inch wire. PTA was performed with an undersized semicompliant balloon (2.5 mm × 9 mm). Angiography just after PTA showed wall dissection (Fig. 2 b). Stenting was performed using an S660 stent with a nominal diameter of 2.5 mm and a length of 9 mm. The stent-deployment balloon was inflated to a pressure of 10 atm (unconstrained diameter of 2.5 mm). After stenting, the lesion was sufficiently dilated without complications (Fig. 2 c). Follow-up angiography 6 months after stenting revealed no restenosis.

Discussion

Transient ischemic attacks of posterior circulation are associated with a 22% to 35% risk of stroke in five years, and infarction of the vertebrobasilar artery carries a serious prognosis [2, 5, 16]. Although open surgery, such as carotid-vertebral transposition, endarterectomy and bypass surgery, have been performed for obstructive lesions of vertebrobasilar arteries, they are technically demanding and fraught with complica-

tions [12]. Medical treatment such as anticoagulation and antiplatelet aggregation therapy does not consistently benefit patients [24]. PTA for vertebrobasilar artery stenosis was introduced in the early 1980s as an alternative to surgery by Sundt *et al.* [13]. Its usefulness has been limited by immediate complications including elastic recoil, wall dissection and vessel rupture [3, 6, 10, 14]. These limitations have fueled interest in treating these lesions by using stents. Theoretically, stenting improves acute and long-term patency and reduces the risk of acute closure from dissection by trapping plaque material between the stent and arterial wall.

Stenting for vertebral artery ostial stenosis

The vertebral artery origin is easily accessible with an endovascular technique, so PTA has become the treatment of choice for these lesions since the 1980s [3, 6]. The usefulness of PTA has been limited by some factors. Arterio-ostial stenoses involving vertebral artery ostial lesions are more elastic than non-arterio-ostial lesions. Therefore, these lesions are resistant to PTA. The use of larger balloons in an attempt to overcome recoil often results in dissection. The difficulty in achieving an adequate dilatation of the vertebral artery origin with PTA only and the potentially high restenosis rate have encouraged the use of stents at this site. In our series, lesions were accessible to stents in all cases. Successful dilatation was obtained in all patients except the initial case. Since 2001, we have performed moderate over-dilatation of stents without any complications. Between 1997 and 2000, the restenosis rate after stenting was 12%. Since 2001, restenosis rate has been reduced to 4.5%. Moderate over-dilatation of stents can be effective in preventing restenosis at this site. The low restenosis rate and low complication rate of stenting for ostial lesions may justify performing primary stenting for vertebral artery ostial stenosis.

Stenting for intracranial vertebrobasilar artery

The tortuous nature of atherosclerotic intracranial vertebrobasilar arteries has limited the use of stents in this area. Although stent devices are less flexible and trackable than PTA balloon catheters, the advent of new-generation, flexible stents has enabled reliable and atraumatic access to intracranial arteries [4, 8, 9, 11]. In our series, technical success was achieved in all

patients. The mean stenosis rate was reduced to 16% after stenting in our patients, whereas other reported series had greater than 40% mean stenosis after balloon angioplasty [10]. Although this residual stenosis rate after stenting was smaller than that of PTA for intracranial arteries, it was greater than that of the ostial lesions in our patients. Intracranial arteries are delicate and thin-walled vessels, with a greater risk for rupture, which can result in lethal bleeding [10]. Therefore, we only under-dilate in these arteries, the relative under-dilatation can prevent procedural complications, such as subarachnoid hemorrhage. Indeed, the complication rate was 0% in our patients. This result was better than that of reported series after PTA or stenting [4, 8–11]. On the other hand, the restenosis rate after stenting for intracranial lesions was high, accounting for 27% of total patients. We speculated that reasons were due to the small diameter of intracranial arteries and under-dilatation of stents. Prevention of restenosis in the intracranial artery is the next problem to be solved. Using self-expandable stents or drug-eluting stents may be an answer to this problem.

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