

# ANGIOPLASTY AND STENTING OF BASILAR ARTERY: SHORT-TERM OUTCOMES

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## ABSTRACT

Symptomatic basilar artery stenosis has a poor prognosis. Surgical bypasses are technically demanding and of no proven benefit. A new generation of intravascular stents that are flexible enough to navigate the tortuosities of the vertebral artery may provide a new therapeutic approach. Our two cases, 57 and 52 year-old men experienced a vertebrobasilar ischemia with repeated vertigo and falls. Magnetic resonance angiography from vertebrobasilar arteries revealed severe middle basilar artery stenosis in one case, and severe vertebrobasilar artery stenosis in the other. The patients underwent uncomplicated angioplasty and stenting of the basilar arteries, with excellent angiographic results. The new flexible intravascular stents provide a new therapeutic approach for patients with basilar artery stenosis.

**Key Words:** Basilar artery, Vertebral artery, Angioplasty, Stenting, Case Report  
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**T**he outcome of symptomatic basilar artery stenosis is poor, with most patients experiencing significant morbidity or death from recurrent ischemic events, despite optimal medical therapy<sup>1, 2</sup>. Surgical therapy is technically demanding, with significant rates of complication and failure even in the most experienced hands<sup>3-5</sup>. Angioplasty of basilar artery stenosis has been proposed as a novel therapeutic approach<sup>6-8</sup>. Although, there have been some encouraging results, intraplaque dissection, plaque dislodgment, and vessel recoil with restenosis may occur<sup>9-11</sup>. Another author showed that the restenosis rate after intracranial angioplasty is significant<sup>12</sup>. The advantages of stent-assisted angioplasty over angioplasty alone include the exclusion of dislodged plaque and regions of dissection from the vessel lumen, as well as the prevention of vessel recoil and rupture<sup>11</sup>. We will describe two patients that angioplasty and stenting resolved a symptomatic atherosclerotic stenosis of the basilar arteries for which longstanding medical therapy failed to resolve neurological symptoms.

## CASES REPORT

**Case 1:** The 57 year-old man with history of hypertension and history of recurrent vertigo referred in October 2003 to our institution for angioplasty of a basilar artery. Brain magnetic resonance imaging scans showed no infarction within the distal field territories of both posterior cerebral arteries. Magnetic resonance angiography was performed and showed severe middle basilar artery stenosis. The patient was given warfarin therapy. Our patient didn't recover completely and he continued to experience daily episodes of vertigo that were unrelated to positional changes. Cerebral angiography confirmed the presence of high-grade middle basilar artery stenosis (Fig 1A).

**Case 2:** The 52 year-old man with history of repeat severe vertigo and falls referred in April 2004 for angioplasty of a basilar artery stenosis. Brain magnetic resonance imaging scans showed no infarction within the distal field territories of both posterior cerebral arteries. Magnetic resonance angiography was performed and showed severe stenosis in proximal of basilar artery. The patient was given warfarin therapy. Our patient didn't recover completely by medical therapy. Cerebral angiography confirmed a large plaque with severe stenosis in distal of the left vertebral artery and proximal of the basilar artery (Fig2A).

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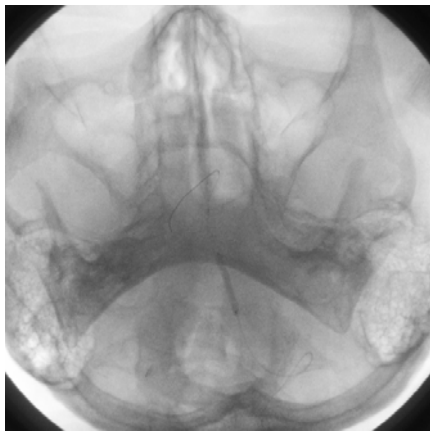
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**Fig1A.** Left vertebral angiogram shows middle basilar artery stenosis.



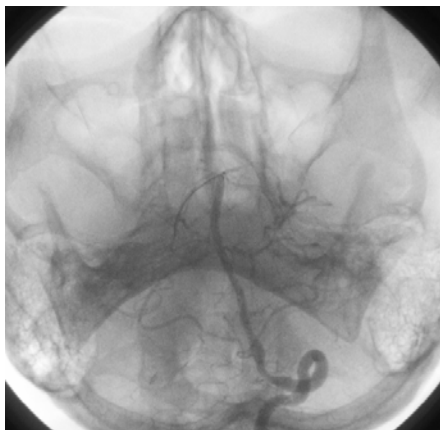
**Fig2A.** Left vertebral angiogram shows distal vertebral artery and proximal basilar artery stenosis.



**Fig1B.** Radiograph shows navigation of 0.014-inch guidewire and AVE S7 coronary stent, Medtronic into left vertebral artery. Stent was smoothly navigated into middle basilar artery.



**Fig 2B.** Radiograph shows navigation of 0.014-inch guidewire and AVE; driver coronary stent, Medtronic into left vertebral artery. Stent was smoothly navigated into proximal basilar artery.



**Fig1C.** Final left vertebrobasilar control angiogram shows stent deployed into basilar artery. No residual stenosis is visible. Proximal



**Fig2C.** Final left vertebrobasilar control angiogram shows stent deployed into distal of vertebral and proximal of basilar artery. No residual stenosis is visible.

## METHOD

After the insertion of a 7-French femoral sheath, 5000 U of heparin was injected IV followed by a continuous infusion of 3000 U/hr to obtain an activated clotting time at 200 sec. Two hundred fifty milligrams of acetylsalicylic acid and 400mg Clopidogrel were also given.

A 7-French right coronary guide catheter was placed over an exchange wire in the proximal vertebral arteries. A 0.014-inch guide wire was then navigated across the basilar arteries stenosis, and the guide wire tip was secured into the left posterior cerebral artery (Fig1B, 2B).

The pressures immediately proximal to the stenotic lesion were measured (120 mm Hg in case 1 and 140 mmHg in case 2). The microguidewire and microcatheter were then advanced through the stenotic lesions. Pressure measurements distal to the stenotic lesions revealed a significant gradient across the stenosis. Angioplasty dilation was performed with a 2.0- $\times$ 15-mm balloon in case1 and 2.5- $\times$ 20-mm balloon in case 2.

Then the balloon-expandable coronary stent (AVE, S7; coronary stent, Medtronic) with diameter of 3.0 mm and a length of 18 mm in case 1 and ( AVE, driver; coronary stent, Medtronic) a diameter of 3.0mm and a length of 18 mm in case 2 were advanced over the guide wire to cover the stenosed segments and deployed with no residual stenosis.

During balloon inflation in case 2 (which lasted only 10, 15), the patient experienced a syncopal episode; the episode resolved immediately after balloon deflation. A control angiogram showed the stents mesh covering the entire atherosclerotic basilar segments and excellent basilar arteries patency, with no residual stenosis (Fig1C, 2C).

However, patency of the right vertebral artery outflow was not impaired. No complications occurred during or after the stents deployment. Continuous intravenous heparin infusion was administered for 12 hours after the procedure, to maintain a partial thromboplastin time 1.5 to 2.0 times the normal value. The same day, 250 mg of aspirin and 400 mg of Clopidogrel were administered. During follow-up (12 months in case 1 and 8 months in case 2), the patients were well and free of symptoms. At the 12-month follow-up examination in case one, the control angiogram showed excellent patency of the basilar artery.

## DISCUSSION

To prevent acute basilar artery thrombosis and its catastrophic consequences, anticoagulation and antiplatelet therapy has been considered. A recent randomized study suggested that the administration of prescription drugs is inadequate<sup>13</sup>. Further atheromatosis progression was not stalled by long-term anticoagulative therapy, and stroke rates reported for patients with basilar artery stenoses undergoing such therapy were as high as 10% per year, leading to the conclusion that aggressive therapy such as angioplasty should be considered, especially for high-grade stenoses.<sup>14</sup> Until recently, intracranial stenting was limited by the rigidity of available devices, making navigation through the tortuous proximal intracranial circulation difficult. The availability of intravascular stents that can be navigated through tortuous intracranial vessels has initiated a new era in endovascular therapy<sup>11, 15</sup>. Endovascular treatment of basilar artery stenosis has been advocated only for recurrent symptoms that are refractory to medical treatment<sup>6,8,15</sup>.

During angioplasty, vessel tortuosity and compromise of the brainstem perforators originating from the basilar artery are problems that are less common with proximal stenosis<sup>14</sup>. The reasons for a slow and undersized dilation of the stenosis are to protect the vessels that do not have supporting tissue in the event of a sudden rupture, intimal dissection, acute vasospasm, and thrombosis. However, the likelihood of such potentially dramatic complications may be minimized by stent implantation, which also allows a more aggressive anatomic correction<sup>14-17</sup>. Stents have also been used in the treatment of intracranial carotid stenosis, with encouraging results<sup>18-20</sup>. Two reports have suggested that stent-assisted angioplasty may represent a viable therapeutic option for vertebral artery and basilar artery atheromatous stenoses<sup>17, 21</sup>. Our cases suggest that stent-assisted angioplasty may represent a viable therapeutic option for patients with basilar artery stenosis. However, antithrombotic dictation in cerebral angioplasty has not been standardized and antiplatelet regimens administered during and after intracranial stenting remain empirical<sup>14</sup>. The use of acetylsalicylic acid and Clopidogrel and stent implantation may also confer complementary long-term clinical benefits for patients undergoing intracranial angioplasty, similar to benefits for patients undergoing coronary

stenting<sup>22</sup>. There is concern that occlusion of the ostia of small side branches and perforating arteries by stent placement may result in ischemia or infarction in the territory of these vessels<sup>11</sup>.

However, experimental evidence suggests that small lateral carotid branches in dogs, which approximate human intracranial perforating vessels with respect to their diameter and angle of origin, tend to remain patent if less than 50% of the ostial diameter is covered by the stent struts<sup>11, 22</sup>. Similarly, no difficulties involving perforating branch occlusions were encountered after stenting in our cases. Our procedure was balloon angioplasty to predilate the lesions. Then they delivered the balloon-expandable stent. Long-term follow-up data and additional clinical experience are required to effectively assess this novel approach for the treatment of vertebrobasilar occlusive disease.

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