

Case Reports

Successful treatment of severe stenosis of the posterior cerebral artery with percutaneous transluminal angioplasty and stenting

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ABSTRACT

يعد تضيق الشريان المخي من الأسباب الرئيسية لحدوث السكتة الدماغية وعدم تدفق الدم الكافي إلى الجهاز الفقري القاعدي. تُستخدم طريقة رأب الشرايين المخية عبر البالعة وعبر الجلد (PTCAS) لعلاج التضيق القاعدي الفقري، غير أن جدوى استخدام هذه الطريقة لعلاج تضيق الشريان المخي الخلفي لم يتم إثباتها. نستعرض في هذه التقرير حالة مريض يبلغ من العمر 64 عاماً يُعاني من تضيق شديد في الشريان المخي الخلفي، وقد نجحت طريقة رأب الشرايين المخية عبر البالعة وعبر الجلد في شفاؤه من هذا التضيق.

Cerebral arterial stenosis is a major cause of stroke and of insufficient blood supply to the vertebral basilar system. Percutaneous transluminal cerebral angioplasty and stenting (PTCAS) have been used to preliminarily treat vertebrobasilar stenosis. However, the feasibility to treat the posterior cerebral arterial stenosis by PTCAS has not been fully established. We report a case of a 64-year-old man with a severe stenosis of the posterior cerebral artery that was treated successfully using a PTCAS procedure.

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Stenosis is a major cause of stroke and of insufficient blood supply to the vertebral basilar system. Atherosclerosis is the most common cause of stenosis. Atherosclerosis plaques can cause considerable vascular stenosis, or occlusion, which frequently occurs in the vertebral and basilar arteries.¹ Percutaneous transluminal

cerebral angioplasty and stenting (PTCAS) has been used preliminarily to treat vertebrobasilar stenosis.²⁻⁴ However, a literature review found that reports on the treatment of posterior cerebral artery (PCA) stenoses by PTCAS are rare. Here, we report the case of a 64-year-old man with a severe stenosis of the PCA that was successfully treated with a PTCAS procedure.

Case Report. A 64-year-old man was admitted to the hospital with a 6-year history of asthenia in his right limbs; the symptoms had worsened significantly 3 days before admission. Aggravation of the asthenia was concomitant with the onset of transient dizziness, drop attack, and aphasia. He also had a history of hypertension, and experienced intracerebral hemorrhage of the left basal ganglia, which lead to asthenia of the right limbs for 6 years. His BP was 170/87 mm Hg and his mental status was alert and cooperative. A neurologic examination identified right central faciolingual paralysis, symmetrical sensation in all 4 limbs, no muscle atrophy, muscle strength of the right upper and lower limbs 3+, hyper muscle tension, and normal muscle strength in the left upper and lower limbs. A head MRI showed subcortical arteriosclerotic encephalopathy, multiple infarctions, and encephalomalacia in the pons cerebelli, the periphery of both lateral cerebral ventricles, and the semioval areas of both hemispheres. An ultrasound of the neck showed extensive thickening of the intimal and medial tunicae of the left common carotid artery and of the posterior wall of the right common carotid artery adjacent to the carotid sinus, as well as slowed blood flow in vertebral arteries on both sides. A digital subtraction angiography (DSA) showed local stenosis of the P1 segment of the left PCA with up to 85%

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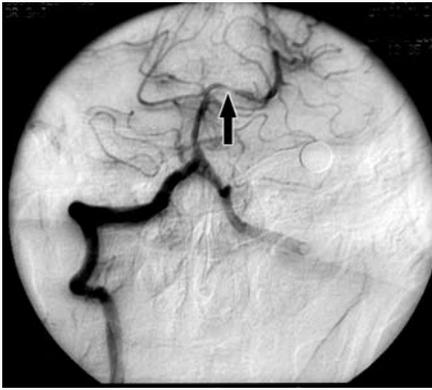


Figure 1 - The digital subtraction angiography shows severe stenosis of the P1 segment of the left posterior cerebral artery.

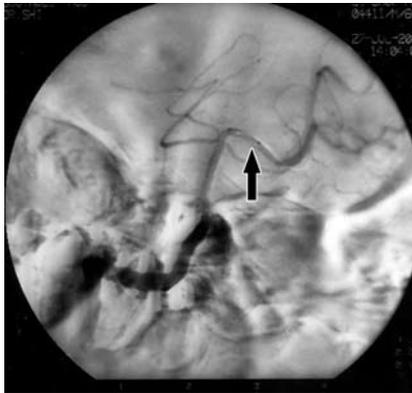


Figure 2 - The digital subtraction angiography shows the precise location of the stent.

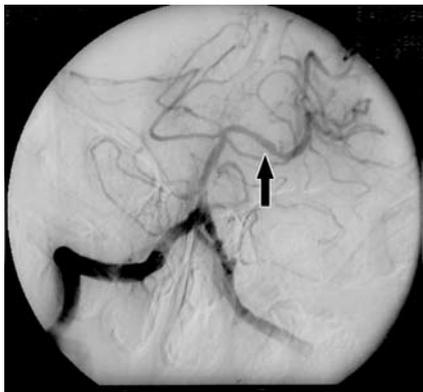


Figure 3 - Postoperative digital subtraction angiography indicates the disappearance of the stenosis in the P1 segment of the posterior cerebral artery.

(Figure 1). No valid collaterals with meningeal arteries were observed. Three days before the operation, he was given Nimodipine (90 mg/d), aspirin (300 mg/d), and Clopidogrel (75 mg/d). Fasting was initiated 6 hours before the operation. Phenobarbital (0.1 g) was given via intramuscular injection 30 minutes before the operation. The following procedures were performed sequentially:

local anesthesia in the right groin area, puncture of the femoral artery, insertion of an arterial sheath, and placement of a 6F guide catheter in the V2 segment. Under the guidance of road roadmap fluoroscopy, a mini-guide wire (Agility® 10, Cordis, Miami, Florida, USA) was placed into the P3 segment of the left PCA through the stenotic segment (Figure 2). We noted that when the tip of the wire reached near the stenosis, arterial spasm occurred. Following administration of 6 mg of Papaverine via the guide catheter, the spasm was immediately prevented. Then, under the guidance of the wire, a cobalt-chromium alloy balloon-expandable stent (ArthosPico, amg International GmbH, Raesfeld-Erle, Germany) of 2.0 mm in diameter and 8.0 mm in length was placed with a release pressure of 6 atmospheres and dilation duration of 10 seconds. Brain angiography indicated that the PCA stenosis had been completely removed (Figure 3). The catheter and guide wire were pulled out and the incision was dressed with a pressure bandage. Postoperatively, he was treated with heparin (500 U/h) for 24 hours, aspirin, and Clopidogrel continued to be administered. The asthenia of the right limbs and the aphasia improved progressively and no further dizziness and drop attacks were observed. At 6-month follow-up, no further transient ischemic attack or stroke was observed. Transcranial Doppler monitoring indicated normal cerebral blood flow.

Discussion. Major vessel occlusion that occurs in the vertebral basilar system usually leads to severe disability or death. The most common lesion found in the vertebral basilar system is atherosclerosis. Atherosclerotic plaques can cause considerable vascular stenosis, or occlusion.¹ The perforating branches of the PCA supply blood to the mesencephalon and thalamus, while the PCA supplies blood to the occipital cortex. The most common clinical symptom of PCA stenosis or occlusion is half blindness that spares the macula on the side opposite the occipital infarctions, although it can vary depending on the location of the stenosis or occlusion. The PCA is the most distal and longest vessel of the posterior circulation. Thus, it is usually difficult to perform PTCAS in this circuit.^{5,6} From our limited experience, we may conclude that it is feasible for angioplasty and stenting of small caliber PCAs to treat the stenosis of the PCA P1 segment and subsequently to prevent infarction of the brain region that receives blood supply from the PCA. Simple angioplasty readily causes intimal laceration and acute occlusion of the PCA, while long-term arterial recoil may lead to restenosis. Therefore, we combined angioplasty with stenting in our case. ArthosPico small vessel (a diameter <2.70 mm) stents are tubiform, of cobalt-chromium alloy and engraved by Laser, which has sound flexibility and compliance to the vessel and

can reach any lesion site through any angulations. Also, due to better biocompatibility of cobalt-chromium alloy relative to stainless steel and the small contact area of the ArthosPico stent with the vessel wall (only 13%), this stent produces little risk of prompting an allergic response.⁷ The most advanced trait of the ArthosPico stent is its wall thickness (65 µm), which is thinner by half than that of ordinary stainless steel stents. The ArthosPico stent has a radial propping force that is 2 times that found with ordinary stainless steel stents, namely, 2070 mN on average. It is thought that thinner stent walls correlate with a lower incidence of restenosis and that a larger radial propping force correlates with better stenting and angioplasty.⁸

As the stent passes through the vertebral-basilar artery junction, serious spasm of the basilar artery may occur, resulting in nonvisualization of the contrast agent in the distal basilar artery. A small dose of Papaverine given via the guide catheter can prevent this problem. In addition, because of the guide catheter “leap forward” phenomenon, which results from passage of the stent through a curvature, we had the stent follow the guide wire. With this technique, the guide wire is not placed too distally, lest it “leap” forward and injures the distal vessel. Because the angulation of the PCA and basilar artery is usually large, the stent should be passed gently and rotated frequently. Its position should be confirmed using angiography instead of roadmap fluoroscopy before the release of the stent. While pushing the stent into position, the guide wire frequently withdraws. Accordingly, during the insertion of the balloon and the withdrawal of the stent, we made sure that part of the guide wire was first withdrawn, followed by the guide wire and the balloon withdrawn. Regarding the different calibers of the vertebral arteries, we propose that when PTCAS of the PCA are carried out, it should be performed via the dominant vertebral artery.

In conclusion, performing PTCAS in the PCA is usually difficult, however, with advances in the development of imaging and angioplasty technology,

and the availability of the ideal stent material, this surgical procedure may be feasible for routine use in clinical practice in certain patients.

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