◆ CASE REPORT ——

Use of the Parodi Anti-Emboli System and Transient Subclavian Steal for Cerebral Protection During Emergent Vertebral Artery Recanalization

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Purpose: To report the use of the Parodi Anti-Emboli System (PAES) for cerebral protection during emergent vertebral artery recanalization.

Case Report: A 56-year-old chimney sweep was referred with recurrent episodes of vertigo and gait ataxia. Left vertebral artery (LVA) flow was barely detectable on duplex Doppler, and brain computed tomography revealed a small infarct in the posterior inferior cerebellar artery territory. Angiography showed subtotal ostial stenosis of the LVA with poor distal flow and possible thrombus. Due to a high risk of distal embolization with percutaneous treatment, anticoagulation was initiated, and the lesion was to be re-evaluated in 2 to 3 weeks. However, 2 days later, the patient developed severe, aggravating headache, gait and left-limb ataxia, horizontal nystagmus, and vomiting. Emergent angiography showed a total ostial LVA occlusion. The PAES was employed to elicit a temporary subclavian steal during percutaneous LVA recanalization, thus protecting the brain from embolization. The ostial LVA was successfully recanalized and stented, with immediate symptom cessation. **Conclusions**: The PAES can be successfully applied in the subclavian artery to prevent distal embolization during emergent vertebral artery recanalization. Since a significant proportion of vertebral strokes are embolic, PAES may play a novel role in the treatment of acute cerebellar stroke.

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Key words: stroke, vertebral artery occlusion, endovascular treatment, stent-assisted angioplasty, neuroprotection devices

Despite extensive research, optimal management of acute stroke remains controversial. Thrombolysis requires careful patient selection and needs to be initiated early.^{1,2} The benefits of thrombolysis outweigh the risk of intracranial hemorrhage; however, the 10% to

15% rate of hemorrhagic transformation causing neurological deterioration is substantial.² Although endovascular treatment strategies, such as balloon angioplasty/stenting or thrombus removal, have been reported in acute stroke,^{2,3} they have not yet undergone

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any systematic evaluation and are not routinely recommended.²

Although evidence from randomized trials is pending, recent data suggest that cerebral protection devices can significantly reduce the complication rate during elective stent-assisted carotid angioplasty in both symptomatic and asymptomatic patients.^{4,5} Vertebral angioplasty, however, is believed to be associated with only a small risk of distal embolization. Thus, any routine use of mechanical neuroprotection during elective endovascular treatment of vertebral artery stenosis is currently considered unnecessary.

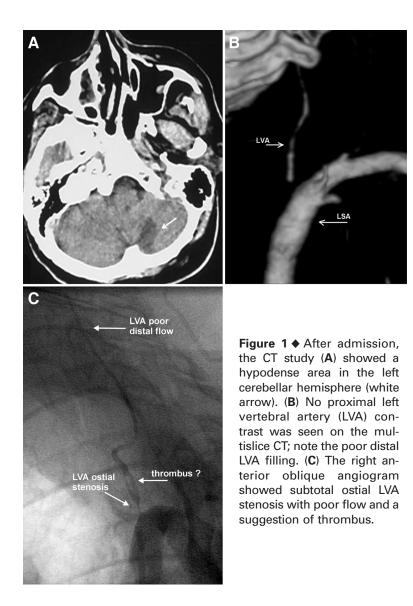
In a patient with acute cerebellar ischemia caused by vertebral artery occlusion with a thrombus component, we performed emergent vertebral artery recanalization using the Parodi Anti-Emboli System (PAES)⁶ to elicit a transient subclavian steal to protect the brain from embolization.

CASE REPORT

A 56-year-old chimney sweep with hypertension and hyperlipidemia was referred to our Institution from the Department of Neurology. The patient gave a history of recurrent episodes of vertigo and gait ataxia over the past several weeks, the most recent event requiring hospitalization. Brain computed tomography (CT) was consistent with a left cerebellar hemisphere stroke in the posterior inferior cerebellar artery (PICA) territory (Fig. 1A). The patient had been managed conservatively on aspirin and ticlopidine.

Physical examination was normal. Duplex Doppler of the aortic arch vessels showed normal flow in the carotid arteries and in the right vertebral artery, but the flow in the left vertebral artery (LVA) could barely be detected. Multislice CT (Somatom Sensation Cardiac 16; Siemens, Erlangen, Germany) indicated a subtotal ostial stenosis of the LVA (soft plaque of 26 to 68 Hounsfield units), with very poor distal filling of the vessel (Fig. 1B). On the following day, elective angiography confirmed the subtotal LVA ostial stenosis with poor distal flow and suggested the presence of a thrombus (Fig. 1C). At that time the patient was asymptomatic, and the perceived risk of distal embolization with percutaneous treatment was high, so anticoagulation was initiated (enoxaparin 80 mg bid). The plan was to re-evaluate the lesion in 2 to 3 weeks and perform angioplasty if the thrombus was cleaved (if some residual thrombus was evident, warfarin therapy was planned). However, 2 days later, the patient developed severe, aggravating headache, gait and left-limb ataxia, horizontal nystagmus, and vomiting. Emergent re-angiography from the right femoral artery with a 6-F diagnostic right Judkins catheter showed a total occlusion of the LVA ostium (Fig. 2A).

For the attempted LVA recanalization, we decided to employ the PAES system to establish a transient steal effect in the LVA/left subclavian artery (LSA) to ensure optimal brain protection during guidewire navigation through the lesion. First, the femoral arterial sheath was exchanged for 10-F, and a 7-F cannula was placed in the left femoral vein. A stiff 0.035-inch J-tipped guidewire (Emerald Amplatz Super Stiff; Cordis, a Johnson & Johnson company, Miami Lakes, FL, USA) was introduced into the distal portion of the LSA. The PAES (ArteriA, San Francisco, CA, USA) guiding sheath with a 6-F right Judkins guiding catheter ("telescopic" technique) were positioned in the proximal LSA. The low-pressure balloon of the PAES guiding sheath was inflated, halting antegrade flow in the LSA. Then the arterial and venous lines were connected with an external filter supplied by the manufacturer. Efficient retrograde flow was achieved with a back pressure of 48 mmHg (Fig. 2B), without any new symptoms (the headache, nausea, and horizontal nystagmus continued). The lesion was crossed with a 0.014inch coronary J wire (Whisper MS; Guidant Corporation, Indianapolis, IN, USA) (Fig. 2C) and predilated with a 2.5×20-mm balloon catheter (Stormer; Medtronic Vascular, Santa Rosa, CA, USA) at a maximum of 12 atmospheres. A 3.5×16-mm Express coronary stent (Boston Scientific, Natick, MA, USA) was positioned and expanded under 10 atmospheres followed by proximal post-dilation at 16 atmospheres. The stent protruded \sim 2 mm into the LSA. Active aspiration with a 50-mL syringe was maintained as the lowpressure PAES balloon was deflated. After



an 18-minute flow reversal, antegrade flow in the LSA and LVA was restored. The final angiogram (Fig. 3A) documented optimal stent position and expansion, with fully restored LVA flow. Headache, nausea, and nystagmus resolved completely within 30 minutes. The patient was discharged to his home on antihypertensive treatment, aspirin (75 mg/d), ticlopidine (250 mg bid), and a statin. Multislice CT performed 3 months later (Fig. 3B) showed a patent, optimallysized stent in the LVA. The patient remains asymptomatic at 8 months.

DISCUSSION

Although stenotic lesions at the origin of the vertebral artery are common, they are usually asymptomatic due to the filling of the posterior circulation via the contralateral vertebral artery and posterior communicating arteries.⁷ Strokes related to the atherosclerotic lesion of the vertebral artery are more commonly due to the embolic mechanism (i.e., emboli formation at the site of atherosclerotic plaque) than to hemodynamic flow reduction.^{7,8} Several studies reviewed by Cloud and

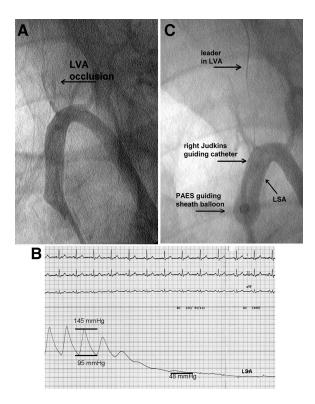


Figure 2 ♦ Emergent angiography (A, 50° right anterior oblique view) 2 days after the initial study showed total left vertebral artery (LVA) occlusion. (B) Pressure recording in the left subclavian artery documenting the transient steal established by the PAES during LVA recanalization. (C) PAES guiding sheath (low-pressure balloon inflated) with a 6-F right Judkins guiding catheter are positioned in the proximal LSA. Under brain protection by flow reversal, the lesion is crossed with a coronary lead.

Markus⁹ have shown that symptomatic patients with vertebral artery stenosis benefit from elective stent-assisted angioplasty. In contrast, there is only anecdotal data on acute endovascular treatment of cerebellar ischemia, whose symptoms are frequently severe.^{3,9,10}

Our patient presented with prior symptoms of recurrent vertigo and gait ataxia, and he had already had a stroke in the left PICA territory. With a high-grade ostial vertebral artery stenosis indicative of a significant thrombus component, we believed that the risk of embolization with acute intervention would not be balanced by its potential benefit, particularly in a person who was not symptomatic at that time. There are no data on optimal management of a nonoccluding thrombus in the carotid or vertebral artery in the absence of acute symptoms of brain ischemia. Our decision to add heparin treatment to the antiplatelet regimen was based on what we believed was a pragmatic extrapolation from the evidence supporting heparin use in unstable angina, in which the thrombus component is known to be substantial.¹¹ Sudden symptoms of acute cerebellar ischemia required emergent intervention, but our main concern with percutaneous revascularization of a soft thrombus-containing plague was the high risk of distal embolization that could extend to vascular territories other than PICA (e.g., the basilar artery).

Several techniques may reduce the risk of distal embolization during vertebral artery angioplasty. Some degree of brain protection can be obtained by increasing the flow to the hand to reduce vertebral artery flow. This is achieved by keeping a blood pressure cuff inflated on the ipsilateral arm and releasing it just prior to lesion crossing, balloon inflation, or stent deployment.¹² Also, Cohen et al.¹⁰ have recently reported the use of a distal protection device during emergent vertebral artery angioplasty. However, faced with a total ostial occlusion, we believed that unprotected crossing with a filter was contraindicated. Finally, we were aware of the earlier reports of a transient subclavian steal^{13,14} achieved during interventions in the vertebral/basilar artery territory by temporary balloon occlusion of the proximal subclavian artery. This, however, required a femoral approach for the transient subclavian artery occlusion and a simultaneous axillary (or radial) access for the guiding catheter. In our patient, we considered such a technique suboptimal because the angle of the LVA takeoff would have made it difficult to cross the occlusion from the axillary or transradial approach. Furthermore, the presence of a large (6 or 7 F) guiding catheter in the axillary or radial artery would greatly reduce blood (and emboli) diversion into the arm during LSA occlusion.

We have over 2 years' experience with the use of the PAES for proximal brain protection during carotid angioplasty.¹⁵ The particular

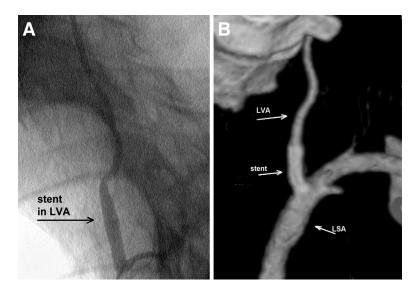


Figure 3 \blacklozenge The final angiogram (**A**) after left vertebral artery (LVA) recanalization with stent implantation and (**B**) the multislice CT performed 3 months later. LSA: left subclavian artery.

strengths of the PAES are its ability to provide protection during lesion crossing and the opportunity to actively drain debris in addition to the continuous flow reversal. Although the PAES has been designed specifically for the carotid system, we have demonstrated that it can be used successfully to achieve subclavian flow reversal with a temporary steal effect in the vertebral artery, a hitherto unreported application of this device. Emergent recanalization of an acute vertebral artery occlusion can be performed safely with PAES in the setting of acute cerebellar ischemia.

REFERENCES

- 1. Wardlaw JM, Zoppo G, Yamaguchi T, et al. Thrombolysis for acute ischemic stroke. *Cocharne Database Syst Rev.* 2003;3:CD000213.
- Adams HP, Adams RJ, Brott T, et al. Guidelines for the early management of patients with ischemic stroke: a scientific statement from the Stroke Council of the American Stroke Association. *Stroke.* 2003;34:1056–1083.
- Eckert B, Kucinski T, Pfeiffer G, et al. Endovascular therapy of acute vertebrobasilar occlusion: early treatment onset as the most important factor. *Cerebrovasc Dis.* 2002;14:42–50.
- Kastrup A, Gröschel K, Krapf H, et al. Early outcome of carotid angioplasty and stenting with and without cerebral protection devices. A sys-

tematic review of the literature. *Stroke.* 2003; 34:813–819.

- EVA-3S Investigators. Carotid angioplasty and stenting with and without cerebral protection. Clinical alert from the Endarterectomy Versus Angioplasty in Patients With Symptomatic Severe Carotid Stenosis (EVA-3S) trial. *Stroke.* 2004;35:e18–21.
- Parodi JC, La Mura R, Ferriera LM, et al. Initial evaluation of carotid angioplasty and stenting with three different cerebral protection devices. *J Vasc Surg.* 2000;32:1127–1136.
- Cloud GC, Markus HS. Diagnosis and management of vertebral artery stenosis. *QJM.* 2003; 96:27–54.
- Koroshetz WJ, Ropper AH. Artery-to-artery embolism causing stroke in the posterior circulation. *Neurology.* 1987;37:292–295.
- Boardman P, Byrne JV. Giant fusiform basilar artery aneurysm: endovascular treatment by flow reversal in the basilar artery. *Br J Radiol.* 1998;71:332–335.
- Cohen JE, Gomori JM, Umansky F. Endovascular management of symptomatic vertebral artery dissection achieved using stent angioplasty and emboli protection device. *Neurol Res.* 2003;24:418–422.
- Hirsh J, Anand SS, Halperin JL, et al. Guide to anticoagulant therapy: heparin. A statement for healthcare professionals from the American Heart Association. *Circulation*. 2001;103:2994– 3018.

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- 12. Wholey MH, Wholey MH, Ferral H. Techniques and results treating lesions of the vertebral and great arteries. In: Marco J, Serruys P, Biamino G, et al., eds. *The Paris Course on Revascularization.* 2003:429–439.
- Nasim A, Sayers RD, Bell PR, et al. Protection against vertebral artery embolization during proximal subclavian artery angioplasty. *Eur J Vasc Surg.* 1994;8:362–363.
- 14. Mayer TE, Hamann GF, Brueckmann HJ. Treatment of basilar artery embolism with a mechanical extraction device: necessity of flow reversal. *Stroke.* 2002;33:2232–2235.
- Pieniazek P, Kablak-Ziembicka A, Pasowicz M, et al. Reversed blood flow during internal carotid artery stenting using Parodi Anti-Emboli System—progress in protection against cerebral stroke. *Kardiol Pol.* 2002;57:322–331.