Persistent Trigeminal Artery Associated with Brainstem Infarct

—Case Report—

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Abstract

A 47-year-old male presented with a sudden onset of right hemiparesis and numbness of the left face. Magnetic resonance imaging demonstrated a lacunar infarct in the ventral pons. Cerebral angiography demonstrated a persistent trigeminal artery (PTA) anastomosing the left internal carotid artery to the distal basilar artery. Bilateral vertebral arteries and the basilar artery below the PTA junction were extremely hypoplastic. The bilateral posterior communicating arteries were embryonic. The posterior fossa circulation was almost independent from the circle of Willis. The poor vascular supply to the posterior fossa probably caused the brainstem infarct.

Key words: persistent trigeminal artery, brainstem infarct, magnetic resonance imaging

Introduction

Persistent trigeminal artery (PTA) occurs in only about 0.2% of patients undergoing cerebral angiography. However, PTA is present in 23% of cases with a history of subarachnoid hemorrhage.¹⁾ PTA is also associated with a higher incidence of posterior fossa transient ischemic attacks (TIAs).⁶⁾ Here, we report a case of PTA associated with severe hypoplasia of the basilar artery resulting in a lacunar brainstem infarction. The significance of PTA in occlusive cerebrovascular disease in the posterior fossa is discussed.

Case Report

This 47-year-old hypertensive male developed a sudden onset of right hemiparesis and numbness of the left face on July 25, 1989. Neurological examination on admission revealed right hemiparesis including the face, paresthesia of the left lower face, and dysarthria. Auscultation disclosed no carotid or subclavian artery bruits. Both initial and repeat computed tomographic scans taken on admission and the next day showed no abnormalities.

Serial angiography was performed by the

transfemoral catheter technique. Left carotid angiograms demonstrated a PTA supplying the upper basilar and superior cerebellar arteries (SCAs). The posterior communicating artery was embryonic and supplied the only ipsilateral posterior cerebral artery. No stenotic or ulcerative lesions were detected in the proximal portion of the internal carotid artery (Fig. 1). Right carotid angiograms revealed almost

Fig. 1 Left carotid angiogram, lateral view, showing the PTA (arrowheads).

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Fig. 2 Left vertebral angiogram, right anterior oblique view, showing hypoplastic bilateral vertebral arteries and the extremely thin basilar artery to the PTA junction (*arrows*). Collaterals supply the SCAs (*arrowheads*).

Fig. 3 T₂-weighted MR images, revealing a lacunar infarct in the upper medial pons as a high-intensity area (*arrow*).

identical findings except that the PTA was absent. Left vertebral angiograms showed bilateral hypoplastic vertebral arteries, especially in the distal portion. The basilar artery was extremely hypoplastic from the origin to the junction with the PTA. The SCAs were partly filled by collaterals from the posterior inferior cerebellar artery (PICA), suggesting that the blood supply from the PTA was insufficient for the SCA distribution (Fig. 2).

The right upper extremity weakness deteriorated during the 2 weeks after onset. Magnetic resonance (MR) imaging (1.5 T, Shimadzu SMT-150) detected a high-intensity area in the left upper medial pons on the T₂-weighted image (TR/TE/excitations, 2500/100/1) (Fig. 3). This lesion appeared as a low-inten-

sity area on the T₁-weighted image (500/23/2-4). The diagnosis was a lacunar infarct.

He received intravenous urokinase (24,000 U/day) for 7 days after admission and thereafter rehabilitation. The weakness gradually improved. He was discharged with hemiparetic gait on January 31, 1990.

Discussion

PTA is usually associated with hypoplastic basilar artery below the junction. The incidence of hypoplastic or atresic vertebral artery was 85% in 400 PTA cases.¹⁾ This indicates that PTA cases have a decreased vascular supply to the posterior fossa. The association of PTA with TIA in the vertebrobasilar distribution is therefore probably due to increased susceptibility to steal phenomena such as subclavian steal syndrome or reversed flow from the basilar to carotid arteries through the PTA.³⁾ An experimental study has established a relationship between the pyramidal conduction and progressive ischemia in the ventral pons in primates.²⁾ The clinical features in our case may have a similar relationship.

Our case demonstrated the common features of decreased blood supply to the posterior fossa. The bilateral vertebral arteries were hypoplastic and the basilar artery was extremely thin below the PTA junction. The posterior cerebral artery was filled from the ipsilateral embryonic posterior communicating artery bilaterally. The blood supply to the posterior fossa was, therefore, almost independent from the circle of Willis except for the upper brainstem. Khodadad³⁾ reported that PTA might compromise the vertebrobasilar circulation. Severe stenosis at the origin of the internal carotid artery increased the reversed blood flow through the PTA from the vertebrobasilar system to the ipsilateral carotid circulation in his case. Our case demonstrated no such stenotic lesion in the internal carotid artery proximal to the PTA origin, so the posterior fossa circulation was protected.

Microemboli originating at an ulcerative lesion at the carotid bifurcation and traveling through the PTA have caused TIAs involving the brainstem.⁵⁻⁷⁾ This is unlikely in our case both clinically and angiographically. Therefore, the lacunar infarct of the pons was probably caused by thrombotic occlusion of the perforators arising from the hypoplastic basilar artery or anastomotic channels supplying the brainstem between the PICA and SCA. Kunishio *et al.*⁴⁾ suggested a similar mechanism in a PTA case associated with Wallenberg's syndrome. PTA associ-

ated with abnormal vertebrobasilar circulation can therefore cause occlusive vascular lesions of the posterior fossa.

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