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Embolic Stroke in the Territory of a Cerebellar Arteriovenous Malformation

—Case Report—

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Abstract

A 63-year-old male presented with an embolic stroke in the right medial inferior cerebellum. Angiography at 2 and 8 months after the stroke revealed arteriovenous shunts from the vermian branch of the right posterior inferior cerebellar artery to the right inferior vermian vein. The shunts mimicked the arteriovenous shunts of post-recanalization in acute ischemic stroke, but were finally diagnosed as a pre-existing congenital arteriovenous malformation based on their persistence. Arteriovenous shunts that persist more than 2 weeks after ictus should be differentiated from the post-recanalization arteriovenous shunts of ischemic stroke, as the different etiology may affect the ultimate prognosis and course of treatment.

Key words: arteriovenous shunt, arteriovenous malformation, brain infarction, luxury perfusion, recanalization

Introduction

Arteriovenous (AV) shunt and capillary blush are well-known angiographic characteristics of acute ischemic stroke.[1,2,4,10-12] AV shunts may persist up to 2 weeks after an acute ischemic stroke, but rarely after 15 days.[11] However, angiography cannot easily differentiate post-recanalization AV shunts from pre-existing AV malformation in the acute stage of ischemic stroke. We describe a case of unusually persistent AV shunt demonstrated by angiography 2 and 8 months after acute ischemic stroke.

Case Report

A 63-year-old male experienced left transient monocular blindness for approximately 1 minute on about 10 occasions within 2 months. He suddenly experienced general fatigue, dizziness, and nausea in August 1996, which gradually subsided within several days. Computed tomography 4 days after ictus showed a low density region in the right medial cerebellum. He was admitted to our hospital for further evaluation in October 1996, 2 months after ictus. Neurological examination was completely normal. Magnetic resonance (MR) imaging disclosed a right medial inferior cerebellar infarction appearing as moderately high intensity on the T_1-weighted image and high intensity on the T_2-weighted image, indicating subacute or chronic infarction with cortical laminar necrosis[8] (Fig. 1). Asymptomatic ischemic changes in the bilateral periventricular regions were also observed. Cerebral blood flow (CBF) examination using technetium-99m-labeled hexamethylpropyleneamine oxime (^{99m}Tc-HMPAO) showed slight hypoperfusion in the right cerebellum. Transfemoral right vertebral angiography revealed AV shunts in the territory of the vermian branch of the right posterior inferior cerebellar artery (PICA) which drained to the right inferior vermian vein. There was an intervening capillary blush-like lesion and circulation in this vascular territory was accelerated (Fig. 2). A total of 4 ml of the contrast material (iopamidol 300 mgI/ml; Schering AG, Berlin, Germany) was injected into the vertebral artery at 3 ml/sec without catheter wedging using a 4-F diagnostic catheter. The left vertebral artery mostly supplied the left PICA, with some communication with the basilar artery. Angiography was performed without any complication. We interpreted the unusually persistent AV shunts as due to ischemic stroke, but pre-existing AV malformation could not be ruled out. Electrocardiography and cardiac echo examination

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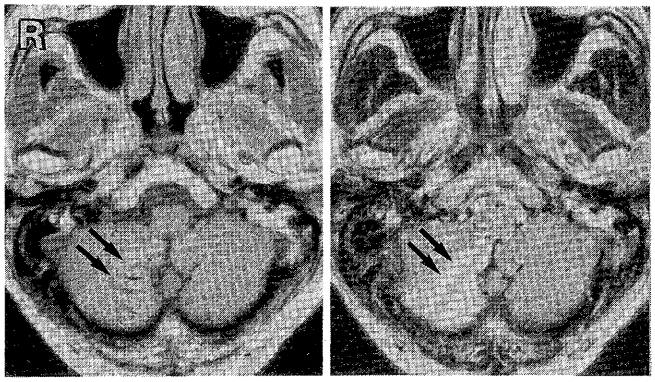

Fig. 1 **Magnetic resonance images at 2 months after ictus showing cortical laminar necrosis in the right medial inferior cerebellum (*arrows*). *left*: T_1-weighted spin-echo image (repetition time 632 msec, echo time 16 msec, number of excitations 2). *right*: T_2-weighted fluid-attenuated inversion recovery image (repetition time 7500 msec, echo time 145 msec, inversion time 1750 msec, number of excitations 4).**

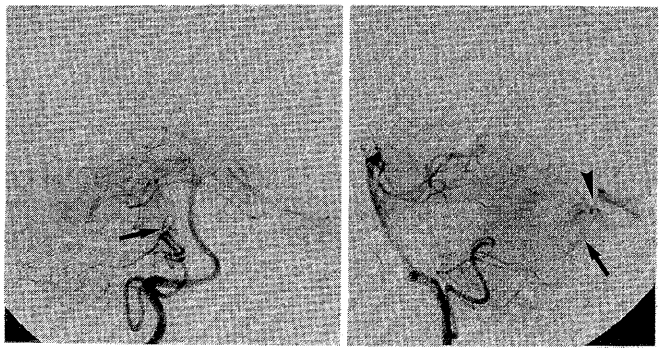

Fig. 2 **Right vertebral angiograms 2 months after ictus, anteroposterior view (*left*) and lateral view (*right*), showing arteriovenous shunts from the vermian branch of the right posterior inferior cerebellar artery (*arrow*) to the right inferior vermian vein (*arrowhead*) with an intervening capillary blush-like lesion.**

were within normal limits, but cardioembolism was suspected based on his clinical history. He had a persistent gastric ulcer, which caused severe gastric bleeding in December 1996, so neither anticoagulants nor anti-aggregants were administered.

In April 1997, the patient suddenly developed mild speech disturbance and slight mental slowness, which subsided in 1 month. He was admitted for re-evaluation in May 1997. MR imaging disclosed an additional small cortical infarction in the right temporo-occipital region. CBF examination using ^{99m}Tc-HMPAO (1 month after the second ischemic stroke) showed marked hypoperfusion in the right cerebellum and moderate hypoperfusion in the right temporo-occipital region. Repeat angiography (8 months after the initial stroke) revealed the same AV shunt found in the initial examination in the vascular territory of the vermian branch of the right PICA (Fig. 3). No other abnormality was observed. However, the AV shunts were now interpreted as a pre-existing AV malformation which was treated conservatively. Due to repeated infarction in different vascular territories, cardioembolic stroke was strongly suspected. Administration of anticoagulants was begun, since the gastric ulcer had almost completely healed with medication.

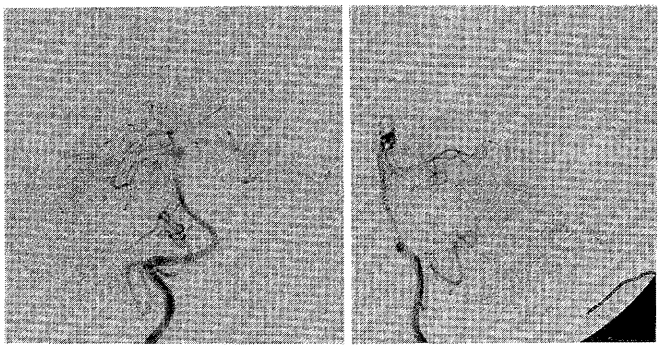

Fig. 3 **Right vertebral angiograms 8 months after ictus, anteroposterior view (*left*) and lateral view (*right*), showing the same persistent arteriovenous shunts as in Fig. 2.**

Discussion

Angiographic indications of early ischemic stroke include: normal angiographical appearance after recanalization, AV shunt (early venous drainage), capillary blush or stain, stagnation of blood flow, prolongation of circulation time, occlusion of the vessel, including visualization of an embolus, narrowing of the vessel, a mass effect, and retrograde filling of the occluded artery through collateral circulation.[3,6,11] AV shunt and capillary blush after a stroke may mimic congenital AV malformation. Differentiation is usually easy based on the clinical course. However, differentiation is more difficult in the acute stage of a stroke, especially when the stroke occurs in the same vascular territory. Other possible causes of AV shunt include neoplasm (either primary or metastatic), contusion, resolving hematoma, seizure (post-ictal), and infection (ab-

scess, meningitis, and encephalitis).[1,2,4,10,12]

Possible interpretations of the AV shunts in our patient included: persistence of post-recanalization AV shunts, which are usually observed only within the first 2 weeks, an error in angiographic technique, a pre-existing AV malformation that induced an ischemic stroke, and cerebral infarction which had occurred in exactly the same territory as a pre-existing AV malformation.

AV shunts may occur within a few seconds of a stroke, and are observed for the first 2 weeks but rarely after 15 days.[11] CBF examination shows this phenomenon as regional hyperemia, also known as "luxury perfusion,"[2,5,9] which can be observed within 16 days of ictus.[13] The region of "luxury perfusion" becomes ischemic about 4 to 5 weeks after the ictus.[13] Such AV shunts or "luxury perfusion" have been explained as lowered oxygen tension and resultant decrease in tissue pH, which causes vasodilatation.[2,5,11] We first thought that the AV shunts in our patient were unusually persistent when they were still observed 2 months after ictus. However, follow-up angiography at 8 months after ictus revealed the same AV shunts, indicating that the AV shunts were not caused by stroke but rather by a pre-existing AV malformation. Our experience of interventional neuroangiography has shown that superselective injection of the contrast material into a small artery can sometimes cause AV shunts and capillary blush, especially in cases of acute ischemia. This phenomenon can be attributed to breakdown of the blood-brain barrier, due to either overpressurization due to catheter wedging or the high osmotic pressure of the contrast material.[7] Repeat angiography showed that this type of error in angiographic technique was not the cause.

AV malformation may be asymptomatic, or may cause intracerebral hemorrhage or subarachnoid hemorrhage, but is highly unlikely to induce embolic stroke. Although there was no histological proof of the exact etiology of the AV shunts in our patient, we believe that the embolic cerebellar infarction occurred in the territory of a pre-existing AV malformation. This unusual case points to the importance of recognizing that AV shunts caused by acute ischemia may persist for 2 weeks, and that persistent AV shunts observed for a longer period have other etiologies.

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