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Aplastic Basilar Artery without a Primitive Carotid–Vertebrobasilar Anastomosis
—Case Report—

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Abstract：Aplastic basilar artery (BA) without a primitive carotid–vertebrobasilar (CA–VBA) anastomosis is exceedingly rare. A 56–year–old woman presented with sudden progressive vertigo and drastic back neck pain at our hospital. Magnetic resonance imaging (MRI) did not demonstrate any organic change, thus suggesting an ischemic lesion in the brain parenchyma, but a magnetic resonance angiogram (MRA) showed no distal part of the BA. Cerebral angiography revealed an aplastic BA and a tortuous bilateral posterior communicating artery (Pcom) including the bilateral superior cerebellar artery (SCA) to supplement the perfusion of the area. When the basilar artery shows aplasia or hypoplasia, the posterior circulation is supplied from primitive segmental arteries in most cases. We herein describe a case of aplastic BA without any primitive CA–VBA anastomosis which was related to an organic pathology.

Key words：**Aplastic basilar artery, Primitive carotid–vertebrobasilar anastomosis, Digital subtraction angiography, Non–ischemic lesion**

Introduction

Numerous anatomic variations of the vertebrobasilar arterial system have been reported. Among them, there have been thirteen reports of a partial aplastic or hypoplastic basilar artery (BA) without a primitive carotid–vertebrobasilar (CA–VBA) anastomosis.[1)–5)] We encountered a patient who had an aplastic BA without a primitive CA–VBA anastomosis. However, the patient did not continue to demonstrate any symptoms similar to those seen in the other cases, but instead experienced transient symptoms. We herein describe the findings of a patient with an aplastic BA.

Case Report

A 56–year–old woman suddenly experienced progressive vertigo, severe nausea and drastic neck pain. In the examination, we found no anomaly of the vital signs nor any distinct neurological local findings. Her symptoms were so severe, that her activities of daily living was remarkably impaired. As we suspected ischemic disease, we immediately performed magnetic resonance imaging (MRI). On the T2–weighted imaging (T2–WI), no organic change to suggest an ischemic lesion in the brain parenchyma was observed, and the main vascular lumina were visualized (Fig. 1). However, magnetic resonance angiograms (MRA) did not reveal a distal part of the basilar artery (BA) (Fig. 2). Based on the images and the clinical time course

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(at three hours from the onset of symptoms), we suspected an acute BA occlusion or vertebrobasilar insufficiency.[6] We decided that thrombolytic therapy would thus be an appropriate treatment, as a result, we performed emergency digital subtraction angiography (DSA).

Bilateral common carotid angiograms (CAG) showed a tortuous bilateral posterior communicating artery (Pcom) which supplied the bilateral posterior cerebral artery (PCA) and superior cerebellar artery (SCA), as well as the distal segment of the BA (Fig. 3a, b). A right vertebral angiogram (VAG) demonstrated the posterior inferior cerebellar artery (PICA) filling from the vertebral

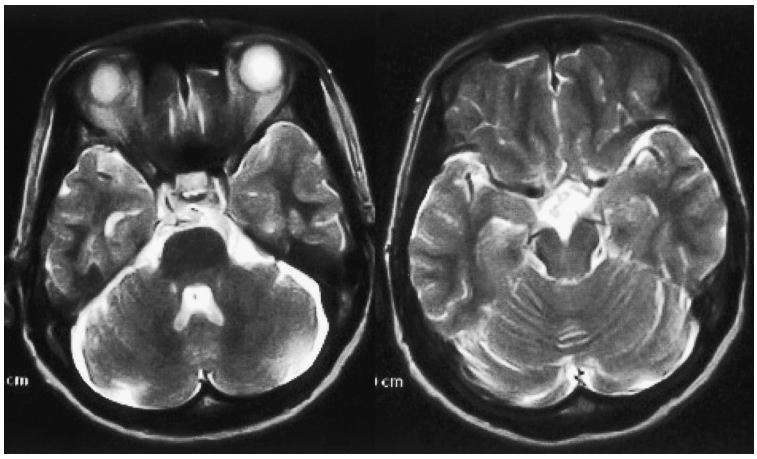

Fig. 1 The magnetic resonance imaging (T2-weighted images) findings indicated no ischemic change in the brain parenchyma. In addition, the main vascular luminal visualizations could be identified.

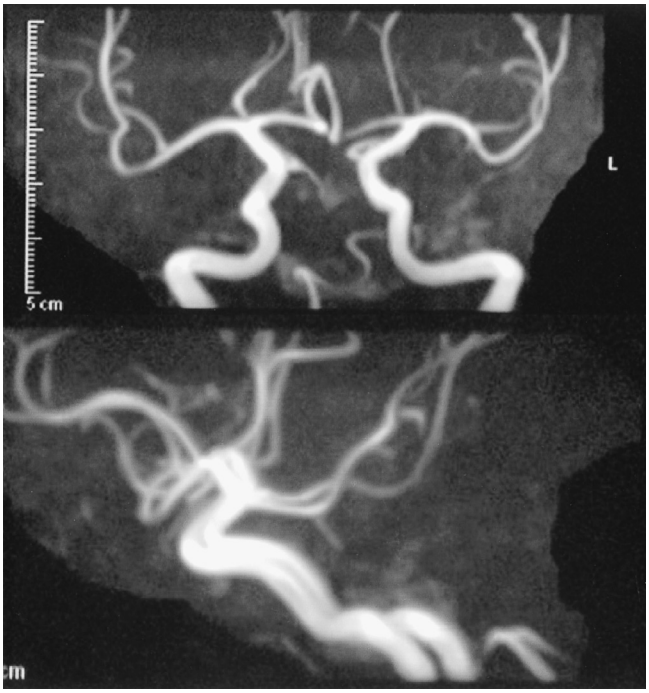

Fig. 2 Magnetic resonance angiograms (Upper : A-P view, Lower : Lateral view) do not clearly depict the distal part of the basilar artery. However, MRI images clearly show the bilateral posterior cerebral arteries via the posterior communicating arteries.

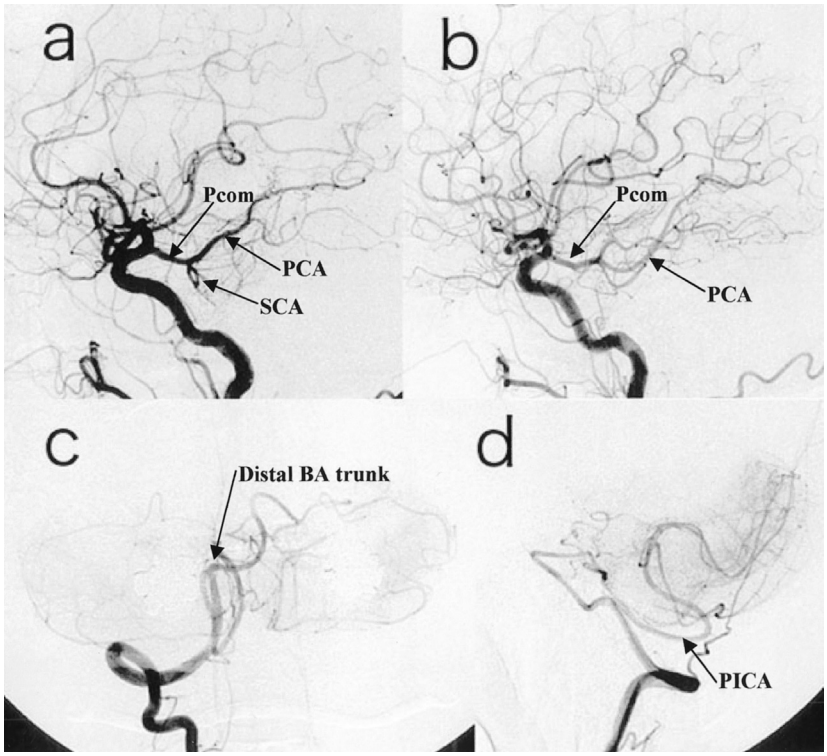

Fig. 3 Bilateral common carotid angiograms (CAG) show a tortuous bilateral posterior communicating artery which supplied the bilateral posterior cerebral artery and superior cerebellar artery (a : right CAG, b : left CAG). Right vertebral angiograms show the bilateral posterior inferior cerebellar artery running from the vertebral artery (c : A–P view, d : lateral view). However, the distal part of the BA is not observed in the angiograms.

artery and aplasia of the distal BA trunk (Fig. 3c, d). The left VAG showed the left hypoplastic vertebral artery at the origin of the left subclavian artery.

No symptoms occurred after the exams and the sequential MRI findings did not show any ischemic lesions. The patient has been regularly followed up at our outpatient clinic.

Discussion

The association between BA hypoplasia and a primitive CA–VBA anastomosis, such as the presence of a persistent primitive trigeminal artery, as well as proatlantal, hypoglossal, or otic artery is well known.[7)–11)] In the early gestational period, the bilateral longitudinal neural arteries form connections with the ICA via the Pcom as well as via the primitive segmental arteries. From approximately the 5th week of gestation, the primitive segmental arteries degenerate. The adult BA develops from the bilateral longitudinal neural arteries after their fusion on the anterior surface of the neural tube. If the BA becomes the main supplier of blood to the developing PCA, then the Pcom becomes smaller. If the latter vessels remain large, however, a "fetal origin" of PCA may thus persist. As a result, an anomaly in the involution of the primitive segmental arteries or in the fusion of the bilateral longitudinal neural arteries may result in either aplasia or hypoplasia of the BA.[7)12)]

In our case, the patient's general status was exceedingly progressive,[6)] the time from onset was short, and there was no depiction of BA on the MRA. Therefore, we worried that the BA occlusion may have caused a complication of a progressive cerebral infarction, and we thus performed emergency cerebral angiography. The posterior circulation with aplastic BA was supplied from Pcom without any primitive segmental arteries.

Since Szdzuy and Lehmann reported the first case based on the findings of conventional CAG,[1] thirteen cases of BA aplasia or BA hypoplasia without a primitive CA−VBA anastomosis have been reported, which were similar to our case.[1)−5)] These cases all had organic lesions. Chaturvedi and colleagues reported posterior circulation ischemia in the territory of the hypoplastic vertebro−basilar system in eight patients,[2] and Befari, et al reported aplastic BA with multiple aneurysms on the dominant Pcom.[5] However, our case did not show any ischemic lesions in the brainstem or the cerebellum due to fact that the distal parts of the BA were supplied from the Pcom, and this case also did not demonstrate any aneurysms. We selected a treatment strategy of natural serial observations for our patient. However, if our case had demonstrated either ischemic disease or cerebral aneurysm, similar to that seen in previous cases, then more drastic treatment would have been required.[2)5)] Therefore, we are presently carefully following up this case by MRI including arterial studies.

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