Anterior Choroidal Artery Variant and Acute Embolic Stroke

Case Report

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Summary

The anterior choroidal artery has the cortical branches to the temporal, parietal, and occipital lobes in the early embryological stage, which later become the posterior cerebral artery distal to the posterior communicating artery (P2-4). Acute embolic stroke occurred in a 57-year-old man with an anterior choroidal artery having such a persistent embryonic branch to the temporal lobe. Recognition of this embryological form of the anterior choroidal artery is clinically important in acute cerebral ischaemia because the cerebral region between the territories supplied by the middle cerebral artery and the anterior choroidal artery is shown on carotid angiography as an avascular area, which could be misunderstood as a region of the acute ischaemia.

Introduction

Embryologically, the primitive internal carotid artery can be divided into the rostral (cranial) and caudal divisions. The former consists of the anterior cerebral artery, middle cerebral artery, and anterior choroidal artery (AchA). The latter consists of the posterior communicating artery, P1 portion of the posterior cerebral artery (PCA) and the basilar artery distal to the primitive trigeminal artery [1-3]. AchA is embryologically an old artery and its telencephalic branches of the temporal, parietal, and occipital lobes are annexed to the diencephalic-mesencephalic arteries or primitive posterior choroidal artery of the caudal division of the primitive internal carotid artery, of whose process is called "distal annexation" [2,3]. Annexed cortical branches of the AchA become the PCA distal to the posterior communicating artery (P2-4).

When the distal annexation does not occur or is incomplete, AchA keeps the cortical branches of the temporo-parieto-occipital lobes in varying degree, which are normally supplied by the P2-4 segments of the PCA. We present a patient having such an embryological form of the AchA, who suffered from acute cardio-embolic stroke. Knowledge of such an AchA variant is important for a proper understanding of cerebral angiography, and we discuss its clinical implications.

Case Report

This left-handed 57-year-old man developed temporary numbness in the left hand lasting three minutes, followed by speech disturbance. The patient was transferred to us within an hour of ictus by ambulance.

At admission, the patient was alert without apparent motor weakness. The eyes were deviated rightward and motor aphasia was ob-

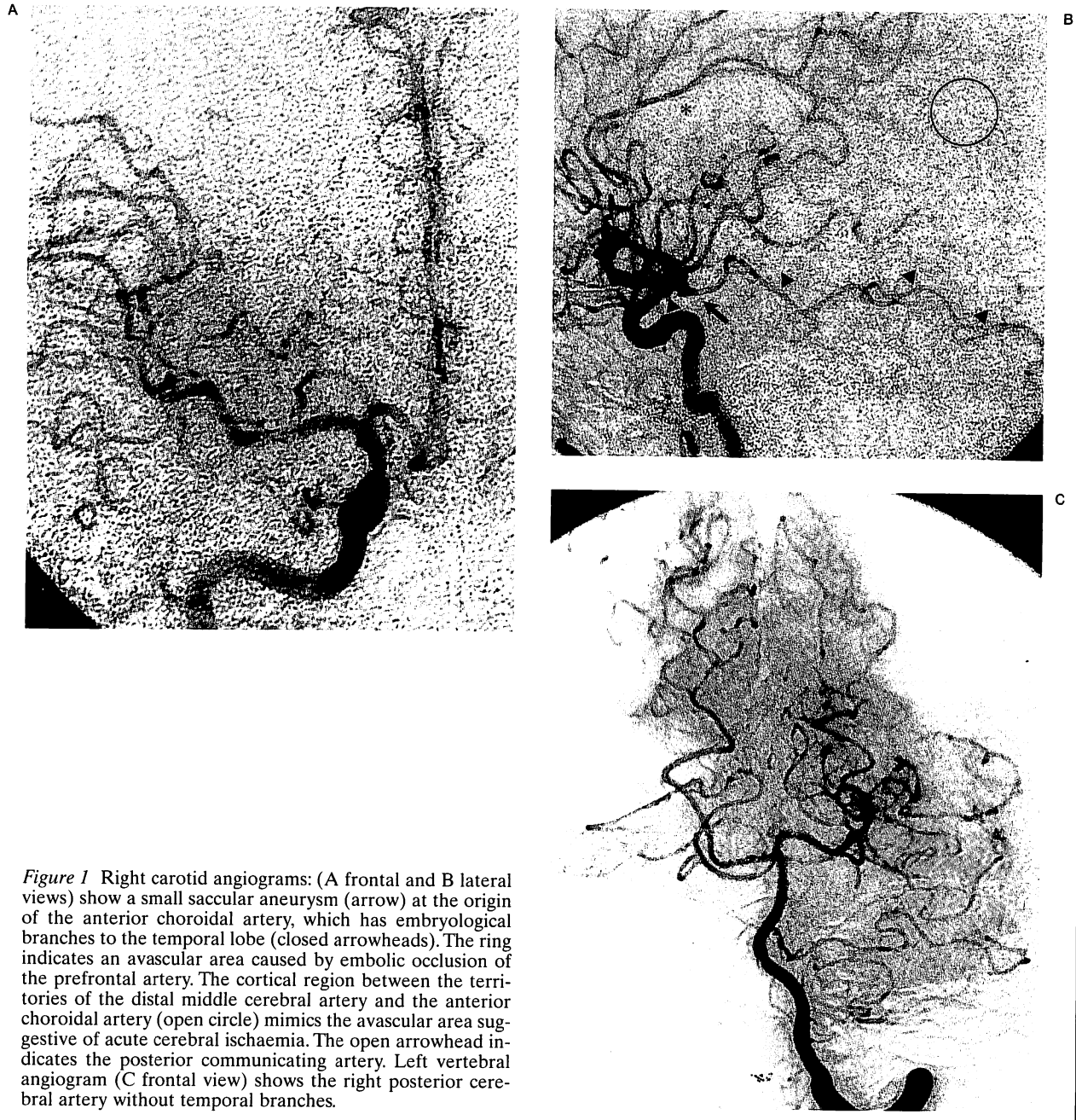

Figure 1 Right carotid angiograms: (A frontal and B lateral views) show a small saccular aneurysm (arrow) at the origin of the anterior choroidal artery, which has embryological branches to the temporal lobe (closed arrowheads). The ring indicates an avascular area caused by embolic occlusion of the prefrontal artery. The cortical region between the territories of the distal middle cerebral artery and the anterior choroidal artery (open circle) mimics the avascular area suggestive of acute cerebral ischaemia. The open arrowhead indicates the posterior communicating artery. Left vertebral angiogram (C frontal view) shows the right posterior cerebral artery without temporal branches.

served. The speech center seemed be located in the right cerebral hemisphere. Six years prior to this admission, this patient had undergone cardiac surgery for stenosis and regurgitation of the aortic valve and for regurgitation of the mitral valve. Warfarin and aspirin had been given thereafter, but thrombo test value on admission was 33.1, which was ineffective for anticoagulation.

Computed tomography of the brain at admission was normal without an apparent low-density area. Emergency cerebral angiography showed occlusion of the right precentral artery of the middle cerebral artery and a small AchA aneurysm at the origin of the internal carotid artery. AchA had the large cortical branches to the temporal lobe. (figure 1). Right carotid angiography showed a non-opacified cortical re-

gion between the territories supplied by the middle cerebral artery and the AchA, which mimicked an avascular region caused by embolus. Careful interpretation of the vertebral angiography showed that this "avascular" region was supplied by the parieto-occipital branches of the right PCA. Selective local fibrinolysis using 180 k units of urokinase to the prefrontal artery resulted in partial recanalisation. Careful manipulation of the microcatheter was required to avoid an injury to the AchA aneurysm. The aneurysm was not treated at this time. This patient developed mild left hemiparesis postoperatively, which subsided in several days. Motor aphasia improved gradually by speech therapy. Intense warfarin administration was give to the patient controlling the international normalised ratio of the prothrombin time around 2.0-2.5.

The AchA aneurysm was electively clipped three months later without any sequelae. This patient had minimal motor aphasia at the last follow-up of 1.5 years from the ischaemic attack.

Discussion

Normally, AchA supplies the critical brain structures including the optic tract, the posterior limb of the internal capsule, globus pallidus, cerebral peduncle, uncus, lateral geniculate body, and optic radiation as well as choroid plexus [1,4,5]. AchA has a connection with the lateral and medial branches of the posterior choroidal artery as well as a choroidal branch of the anterior cerebral artery. AchA may have several branches supplying the temporal lobe, and possibly the medial parietal and occipital lobes. The latter two are telencephalic branches normally supplied by the PCA.

AchA originates normally from the internal carotid artery and less frequently from the posterior communicating artery or from the middle cerebral artery [1,5-9]. Transposition of the origins of the AchA and the posterior communicating artery [10,11] and an absence of the AchA [6] are extremely rare. Large AchA supplying the temporal lobe and/or parieto-occipital lobes could be misunderstood as a duplicated posterior communicating artery or as a hyperplastic AchA [9], both of which do not express the embryological backgrounds.

AchA with persistent embryological cortical branches has been reported in patients with arteriovenous malformations [12] and cerebral aneurysms [9,12,13], but no patients had been reported in association with acute cerebral ischaemia. In acute cerebral ischaemia, prompt interpretation of cerebral angiograms is mandatory because local fibrinolysis is indicated in the selected situation. In the patient with embryological form of the AchA, the cerebral region between the frontal and temporal regions, which is not opacified on carotid angiograms, should not be misunderstood as a territory of acute ischaemia. Knowledge of such a primitive form of the AchA is of clinical importance especially to avoid misunderstanding of the territory of the acute ischaemia. Concomitant vertebral angiography may help to understand the AchA variations.

In conclusion, an AchA variant may supply the temporal and/or parieto-occipital lobes, which are normally supplied by the PCA. In patients suffering from0 acute cerebral ischaemia with such an AchA variant, carotid angiograms could be misunderstood as showing an avascular region in the parieto-occipital lobe. Knowledge of the AchA variants is important to avoid such a misinterpretation.

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EDITORIAL COMMENT

The authors describe an embolic cerebral event (stroke) in a 57-year-old man presenting with an anterior choroidal artery (AchA) giving rise to the cortical branches on the medio-basal surface of the temporal and occipital lobes. They emphasize the importance of recognizing this arterial anatomical variation because: "in a patient with this anatomical disposition, one expect only a few vessels present in between the temporal and parieto/frontal lobes in lateral view on the carotid angiogram which should be not erroneously interpreted as a territory of acute ischaemia". If both MCA and AchA arise from the same carotid trunk, they are demonstrated simultaneously during contrast injection of the ICA (internal carotid artery) on lateral view angiogram without any "avascular" areas at that time. Indeed, there is no embryological relationship between MCA and AchA at the cortical level on the convexity surface of the brain justifying an avascular area in the territory of the former in the presence of the latter (AchA giving temporal branches). In fact, a haemodynamic balance between MCA and AchA exists at the striate level (basal ganglia) only, reflecting the embryological arrangement of the MCA in human, grossly, a combination of the striate branch of the lateral olfatory artery of fish and the pyriform artery of amphibians.

On the other hand, in the presence of this anatomical variation - AchA/temporal branches, a "avascular region" is really appreciated only during the vertebral injection on frontal view. Thus, this last piece of information is useful inthe presence of vertebrobasilar ischaemic syndrome avoiding a misdiagnosis of thromboembolic event based solely on the reduced vascular markings when "the missing" temporal branches actually arise from ICA-AchA.

The authors mention that the AchA can originate from MCA which is a misunderstanding because as AchA is a phylogenetically older vesssel than MCA, its existence precedes the latter and would allow, theoretically, a common origin of both but never, conceptually speaking, a AchA arising from MCA. The opposite could probably be true, but this is beyond the present discussion.

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