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The Accessory Middle Cerebral Artery as a Collateral Blood Supply

Masaki Komiyama, Misao Nishikawa, and Toshihiro Yasui

Summary: We describe two cases of acute embolic occlusion of the internal carotid artery and the middle cerebral artery in association with a patent accessory middle cerebral artery. Because of the presence of the accessory middle cerebral artery, the frontal lobe was salvaged to some extent, but it did not provide sufficient collateral blood supply to the middle cerebral artery territory to spare the rest of the frontal lobe, the temporal lobe, and the basal ganglia.

Index terms: Arteries, collateral; Arteries, stenosis and occlusion

The accessory middle cerebral artery (MCA) was shown angiographically by Handa et al in 1968 (1). In 1973, Teal et al (2) differentiated two types of accessory MCA: the one originating from the ipsilateral anterior cerebral artery near the anterior communicating artery (the true accessory MCA) and the one originating from the distal internal carotid artery (ICA) between the anterior choroidal artery and the terminal bifurcation of the ICA, designated as a duplication of the MCA. In this report, we refer to the former anomalous vessel as the *accessory MCA*.

We report two cases of acute embolic occlusion of the ICA and MCA in association with an accessory MCA, in which the accessory MCA was the sole collateral blood supply to the MCA territory.

Case Reports

Case 1

A 63-year-old man experienced a sudden disturbance of consciousness and left-sided hemiparesis. Ten hours later, he was drowsy but responded correctly to simple commands. He was hemiparetic (2/5 in the manual muscle strength test) on the left side with left-sided central facial palsy. Left-sided homonymous hemianopsia and anosognosia were also noted. It was not possible to examine the patient precisely for sensory disturbance. He had paroxysmal supraventricular tachycardia, which became a sinus rhythm, suggesting that the cause of the stroke was cardioembolism. Computed tomography (CT) showed a mild low-density area in the right temporal lobe and basal ganglia with preservation of the right frontal lobe (Fig 1A). A dynamic flow CT scan showed decreased blood flow in the area in which the conventional CT scan had revealed low density. Blood flow in the frontal lobe, however, was preserved through the accessory MCA (Fig 1B).

Cerebral angiography disclosed an embolic occlusion of the right ICA at the level of the anterior choroidal artery. Left carotid angiography revealed the right accessory MCA, which was the sole contributor of collateral blood supply to the right MCA territory (Fig 1C). This artery had several perforating arteries to the basal ganglia. The patient had been examined in our hospital 10 months before this admission after a transient ischemic attack. The angiogram at that time was interpreted as normal, and the accessory MCA had been overlooked (Fig 1D).

The patient was treated conservatively. Two months later, the hemiparesis improved to some extent, especially in the lower extremity (to 3/5 in the upper extremity and 4/5 in the lower extremity), but hemianopsia remained unchanged. The patient had moderate hypalgesia and hypesthesia in the left half of the body. He could walk with a cane.

Case 2

A 71-year-old man with atrial fibrillation was admitted with a visual disturbance of 2 hours' duration. On admission, he was alert and well oriented. He had no motor weakness or sensory disturbance, but left-sided homonymous hemianopsia was observed. CT findings were normal. Hexamethylpropyleneamine oxime (HMPAO) was administered intravenously immediately after CT examination, followed by scanning with a gamma camera. Owing to the nature of HMPAO, the results of the cerebral flow mapping represented the blood flow when the radioisotope was injected. Single-photon emission CT showed decreased blood flow in the right frontotemporooccipital region, sparing the anterior frontal lobe (Fig 2A).

Cerebral angiography showed the right ICA occlusion at the level of the posterior communicating artery. The distal portion of the right posterior cerebral artery was also occluded, which explained the left-sided homonymous hemi-

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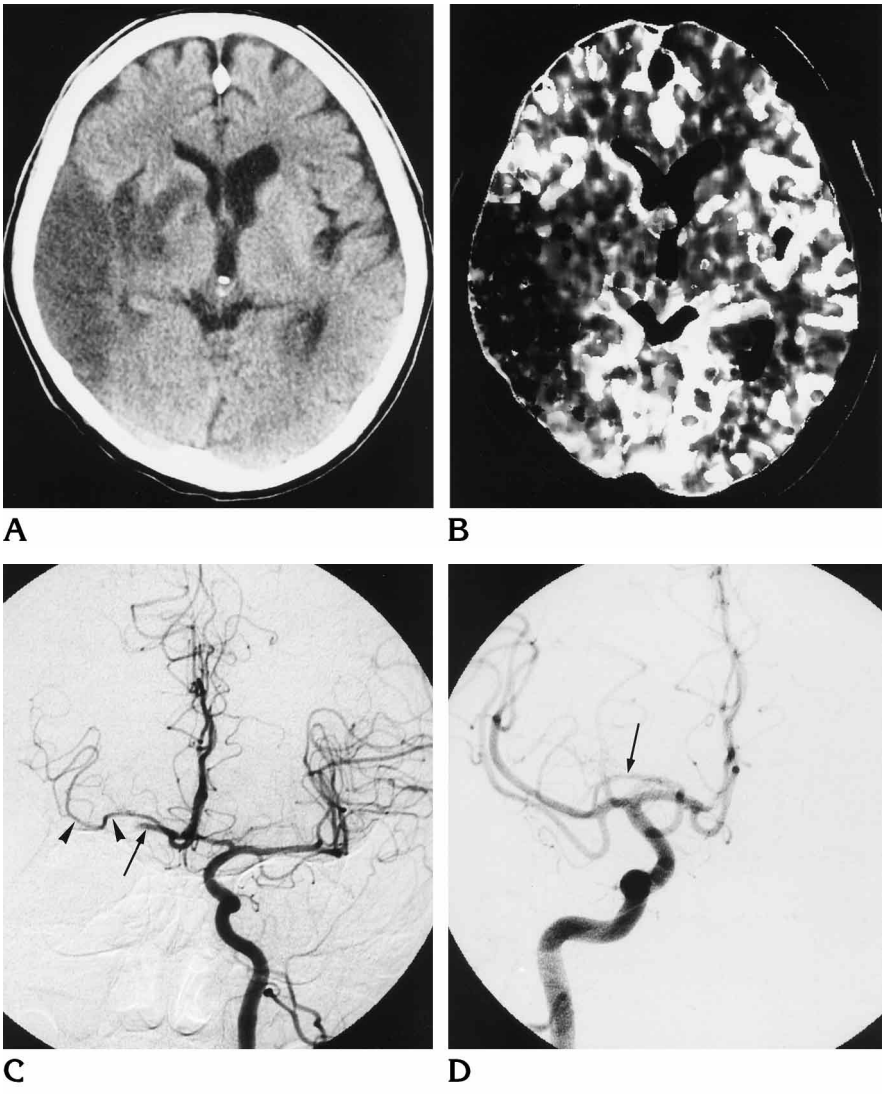


Fig 1. Case 1: 63-year-old man with acute embolic occlusion of the right ICA.

A, CT scan 6 days after ictus shows a low-density area in the right temporal lobe and the right basal ganglia, but the frontal lobe is spared by the accessory MCA. (A CT scan obtained on admission shows essentially the same low-density area, but this scan depicts the lesion more clearly.)

B, Dynamic flow CT scan on admission shows decreased flow in the right temporal lobe and basal ganglia with preservation of the right frontal lobe. Low intensity indicates low perfusion.

C, Left carotid angiogram (anteroposterior view) shows the occluded stump of the right anterior cerebral artery (*arrow*) and the right accessory MCA (*arrowheads*), which have several perforating arteries to the basal ganglia.

D, Right carotid angiogram obtained 10 months before this admission. *Arrow* indicates the right accessory MCA.

anopsia, while the small artery originating from the proximal posterior cerebral artery supplied the parietooccipital region. Left carotid angiography revealed the occluded stump of the A1 portion of the right anterior cerebral artery as well as the patent right accessory MCA, contributing the sole collateral blood supply to the right MCA territory (Fig 2B). This accessory MCA had several perforating arteries to the basal ganglia.

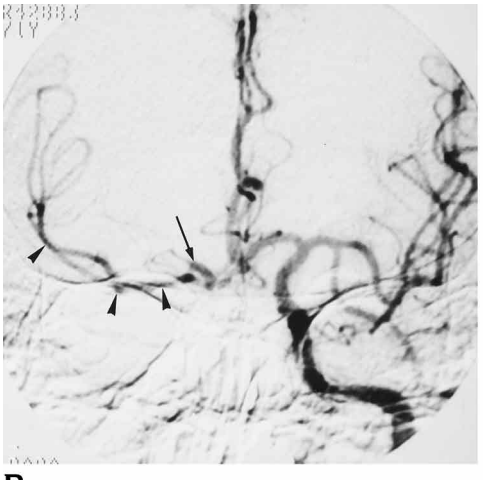
Since CT findings were normal and the patient arrived within 2 hours of ictus, local fibrinolysis was attempted. Because there was no hemiparesis or sensory disturbance and only hemianopsia was noted on admission, intervention was directed to the occluded right posterior cerebral artery. This intervention, however, did not improve the occlusive state.

A disturbance in consciousness and left-sided hemiparesis developed gradually during the 12 hours following angiography and intervention. This was thought to be due to deterioration of the ischemic penumbra supplied by the accessory MCA. Follow-up angiography on day 11 showed recanalization of the right ICA (Fig 2C). At 4 months after ictus, the patient was alert and had left-sided homonymous hemianopsia, hemiparesis (3/5 in the upper extremity and 4/5 in the lower extremity), mild hypalgesia, and hypesthesia. He could walk with a cane. CT showed the right MCA territory infarction with sparing of the anterior frontal lobe (Fig 2D).

Discussion

The accessory MCA is reported as an incidental anatomic finding (3) or in coexistence with cerebral aneurysms (1, 4). An accessory MCA with duplication of the MCA is reported in about 3% to 4% of cases (3, 5–7), but angiographic demonstration might in many cases be overlooked because the accessory MCA is usually smaller than the normal MCA and runs parallel to this vessel.

A



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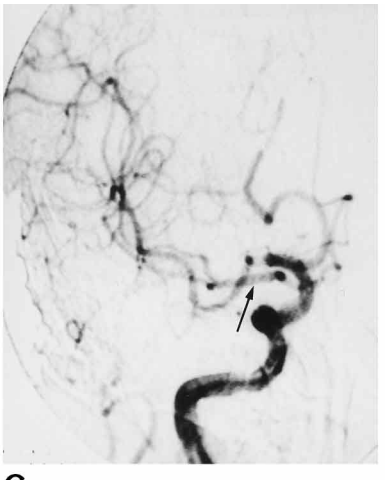

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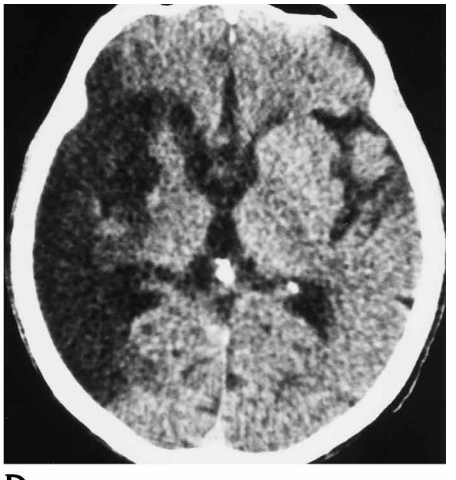

D

Fig 2. Case 2: 71-year old man with acute embolic occlusion of the right ICA.

A, Single-photon emission CT scan with HMPAO shows hypoperfusion of the right frontotemporooccipital region, but the anterior frontal lobe is spared.

B, Left carotid angiogram (anteroposterior view) shows the occluded stump of the right anterior cerebral artery (*arrow*) and the right accessory MCA (*arrowheads*), which has several perforating arteries to the basal ganglia.

C, Right carotid angiogram (anteroposterior view) 11 days after ictus shows recanalization of the right ICA. *Arrow* indicates the accessory MCA.

D, CT scan 4 months after ictus shows the large infarction in the right frontotemporoparietal region with preservation of the anterior frontal lobe.

Handa et al (8) suggested that the accessory MCA is a variant form of the recurrent artery of Heubner. However, there are objections to this idea (2) because (*a*) perforating arteries originate from the accessory MCA only occasionally, (*b*) the recurrent artery of Heubner coexists with the accessory MCA, and (*c*) the recurrent artery of Heubner enters more medially to the anterior perforated substance than does the accessory MCA. Takahashi et al (7) proposed that both the recurrent artery of Heubner and the accessory MCA are persistent anastomoses between the anterior cerebral artery and the MCA over the tuberculum olfactorium. This explains the developmental variants of these anastomoses, which may result in either an accessory MCA or a recurrent artery of Heubner.

Consistent with previously reported cases, in our series, the accessory MCA coursed along the horizontal portion of the MCA and supplied the orbital surface and the anterior frontal lobe. The vessel divided into several branches in the sylvian fissure. The concurrence of ICA occlusion with occlusion of the MCA enabled us to evaluate the true vascular territory of the accessory MCA. Umansky et al (6) showed that the terminal cortical distribution was in the orbitofrontal, precentral, and/or central vessels. In MCA occlusion, the accessory MCA may play an important role in supplying collateral blood flow to the frontal lobe and basal ganglia through the perforating arteries. There are abundant anastomoses on the cortical surface between the MCA and the accessory MCA (5).

Thus, the accessory MCA has the potential to serve as a collateral blood supply to the MCA territory in cases of MCA occlusion.

The accessory MCA is usually smaller in caliber and contributes to a smaller portion of the territory than does the duplicate MCA (4). We believe that this accessory MCA can provide a collateral blood supply but that it cannot furnish sufficient blood flow to the territory that the MCA usually supplies.

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