Aneurysm rupture at an anomalous collateral artery that extended from the proximal A2 segment to the middle of the M1 segment, bypassing atresia of the internal carotid artery bifurcation

Case report

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The authors report the first known case in which an anomalous collateral artery was found to connect the proximal A2 segment with the middle of the M1 segment. This rarity was associated with atresia of the T-shaped internal carotid artery bifurcation. Two aneurysms had developed on a tortuous and tangled portion of the anomalous artery; one of them had ruptured, producing a subarachnoid hemorrhage and an intracerebral hematoma in the area of the putamen. The aneurysms were clipped and the intracerebral hematoma was removed via an emergency craniotomy.

Possible causes of the anomaly and the differences between it and accessory and duplicated middle cerebral arteries are reviewed.

Key Words • anomaly • collateral artery • atresia • middle cerebral artery • aneurysm

ACCESSORY, duplicated, and fenestrated MCAs are relatively well-known congenital anomalies.1,3,5 Aside from these malformations, anomalous collateral vessels may develop in association with aplasia or hypoplasia of the proximal MCA to maintain blood flow to distal segments of that artery.

To our knowledge, this is the first report in which an anomalous collateral artery bordered by the ipsilateral A2 segment and the middle of the M1 segment was confirmed by surgical exploration.

Case Report

This 74-year-old woman presented on May 8, 2003, with sudden mental deterioration and left hemiplegia.

Examination. A CT scan revealed an intracerebral hematoma in the right putaminal area and diffuse subarachnoid hemorrhage. A left CA angiogram demonstrated normal vasculature on the patient’s left side and blood flow to the right MCA via an abnormal vascular structure that was later identified as an anomalous collateral artery on surgical exploration (Fig. 1). The right CA injection demonstrated an obstruction of the terminal ICA and blood flow to the vertebrobasilar circulation through a posterior communicating artery (Fig. 2). A CT angiogram demonstrated the same findings as the digital subtraction angiograms (Fig. 3). A magnetic resonance image did not reveal any associated brain anomaly.

Abbreviations used in this paper: ACA = anterior cerebral artery; CA = carotid artery; CT = computerized tomography; ICA = internal CA; MCA = middle cerebral artery.

Fig. 1. Preoperative left CA angiogram, anteroposterior view, revealing an anomalous collateral artery arising from the right proximal A segment (curved arrow) and joining the middle of the right M segment (straight arrow).
Operation. Surgical exploration revealed atresia of the T-shaped right ICA bifurcation, which exhibited a thin, white, cordlike rudimentary appearance. We identified an anomalous collateral artery that arose from the right proximal A2 segment and joined the middle of the right M1 segment just beyond the end of the atretic portion of the vessel (Fig. 4). Two saccular aneurysms were noted on the tortuous portion of the anomalous collateral artery. Both aneurysms—one ruptured and the other unruptured—were treated by clip occlusion. An intracerebral hematoma in the putamen, which originated from the ruptured aneurysm, was removed.

Discussion

Teal, et al., 6 have proposed that we use the term “accessory MCA” to describe an anomalous vessel that originates from the ACA and the term “duplicated MCA” to characterize an anomalous artery that originates from the distal end of the ICA. Both accessory and duplicated MCAs proceed laterally in the sylvian fissure and do not participate in any anastomosis with the trunk or divisions of the main MCA. The accessory MCA terminates in orbitofrontal and prefrontal areas. The duplicated MCA is distributed to the temporopolar region and to anterior and middle temporal areas.1,3,6

Some cases in which an accessory MCA has been reported may have been cases in which an anomalous collateral artery had formed between the ACA and the MCA. Han and associates2 reported a case in which the accessory MCA was associated with MCA aplasia. Their operative exploration revealed a cordlike rudimentary structure, which arose at the ICA bifurcation and an artery that originated at the proximal A1 segment and coursed into the sylvian fissure. The artery filled all the trifurcation branches of the MCA on the CA angiogram. If the sylvian fissure had been dissected further, those authors might have found an anastomosis between the anomalous artery and the trunk of the MCA. Yaşargil, et al.,7 reported on a case in which an accessory MCA was associated with stenosis around the ICA bifurcation. Operative exploration revealed that a large anomalous artery arising from the ACA lay adjacent to the ipsilateral hypoplastic A1 segment and coursed laterally to the sylvian fissure. Additional dissection of the sylvian fissure might have revealed anastomosis between the anom-
alous artery and the MCA trunk in that case, because cerebral angiography demonstrated findings similar to those in our case. The CA injection revealed an anomalous artery that originated from the region of the anterior communicating artery and filled the MCA trunk and its major divisions.

All cerebral arteries are relatively primitive and plexiform at the gestational age of between 35 and 40 days. In Padget’s illustration of a 14-mm-long embryo, a plexiform anastomosis between the ACA and the MCA can be appreciated. We assume that aplasia or atresia of the ICA bifurcation, which affected the proximal MCA, provoked the formation of the anomalous collateral artery from the plexiform vascular structure between and around the ACA and the MCA.

Atresia of the ICA bifurcation resulted in hemodynamic stress on the anomalous collateral artery, leading to vessel tortuosity and aneurysm growth.

Conclusions

We report on a case in which an anomalous collateral artery connected the proximal A2 segment to the middle of the M1 segment, bypassing an atretic T-shaped ICA bifurcation. One should recollect this type of anomaly before granting a diagnosis of an accessory or a duplicated MCA when one encounters an anomalous artery that supplies all divisions of the MCA and is associated with aplasia or hypoplasia, affecting the ICA bifurcation or the M1 segment.

References