Anomalous branch of the internal carotid artery supplying circulation of the anterior cerebral artery

Case report

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Angiographic and operative investigations revealed an anomalous branch of the internal carotid artery (ICA) in a patient with an anterior communicating artery (ACoA) aneurysm. The anomalous vessel originated from the right ICA at the level of the ophthalmic artery, and pursued an infraoptic and prechiasmatic path to supply both pericallosal arteries. The clinical features and possible genesis of this anomaly are discussed. This irregularity is frequently associated with intracranial aneurysms, especially those of the ACoA, and with other anomalies.

KEY WORDS • cerebral aneurysm • subarachnoid hemorrhage • arterial anomaly • internal carotid artery • optic chiasm

Only nine cases of an anomalous branch of the internal carotid artery (ICA) have been reported previously. The anomalous vessel originates from the ICA at the level of the ophthalmic artery, and passes below the ipsilateral optic nerve and anterior to the optic chiasm to supply the circulation of the anterior cerebral artery (ACA). We have encountered another such case, and this report describes our findings.

Case Report

This 60-year-old woman was admitted to our hospital on March 30, 1982, 5 days after the sudden onset of occipitalgia, unconsciousness for 15 minutes, nausea, and vomiting. She had suffered from weakness in her right upper extremity six times in the preceding several years. Complete recovery occurred within 15 minutes after each attack.

Examination. Neurological examination revealed mild disorientation and a moderately stiff neck. Computed tomography (CT) on the day of admission revealed hemorrhage in both Sylvian and interhemispheric fissures, interpeduncular fossa, and the third and fourth ventricles. An arachnoid cyst in the left temporal region, and dilated lateral ventricles on both sides, were also detected. Bilateral carotid and left vertebral angiography was performed on March 31. A saccular aneurysm, which filled only on the right carotid injection, was found in the region of the anterior communicating artery (ACoA). The anomalous branch, which originated from the ICA at the level of the ophthalmic artery, was found to run superiorly and medially to supply both pericallosal arteries. There was an anastomotic vessel between the two pericallosal arteries (Fig. 1). Despite the presence of the arachnoid cyst in the left temporal region and a history of transient ischemic attacks, there were no remarkable findings in the left carotid angiograms except for a narrow, tortuous carotid artery system and a hypoplastic A1 segment of the left ACA. The right posterior cerebral artery did not fill on left vertebral angiography because it was supplied only by the right ICA (Figs. 1 and 2).

Enhanced CT performed on April 3 also revealed the anomalous vessel, the ACoA aneurysm, and the arachnoid cyst in the left temporal region (Fig. 3). Later the same day, a second subarachnoid hemorrhage occurred, and the patient became comatose for 2 days, at which time ventricular drainage was instituted. By April 22, she had gradually recovered to a drowsy state, but was suffering from severe quadriplegia.

Operation. Despite the poor condition of the patient, an operation to clip the neck of the aneurysm...
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FIG. 1. Right carotid angiograms, anteroposterior view (left), and oblique view (right), with the head rotated about 30° to the left. An anomalous vessel (thick arrow) originates from the right internal carotid artery (ICA) at the level of the ophthalmic artery (double arrows), and runs superomedially to supply both pericallosal arteries. The posterior cerebral artery originates from the ICA. Note the anterior communicating artery aneurysm (arrowheads) and an anastomotic vessel between both pericallosal arteries (fine arrow).

FIG. 2. Left vertebral angiogram, anteroposterior view. The right posterior cerebral artery does not fill because it is supplied only by the right carotid artery (see Fig. 1).

was deemed necessary. Therefore, on April 23, a right frontotemporal craniotomy was performed, with placement of a ventriculoperitoneal shunt and a Holter valve on the left side. The anomalous branch of the right ICA ran beneath the right optic nerve, ascended the prechiasmatic cistern, and supplied the ACoA system (Fig. 4). The hypoplastic left A1 segment entered the anomalous vessel at a site just proximal to the aneurysm neck. The right A1 branch could not be found, however. The aneurysm neck, located on the medial side of the junction of the anomalous vessel and the ACoA, was clipped with Heifetz clip No. 695-117 (Fig. 4). A plexiform network of the ACoA, which could not be identified in the right carotid angiograms, was found (Figs. 1 and 4).

Postoperative Course. The postoperative course was uneventful except for a recurrent urinary infection. Repeat right carotid angiography revealed no filling of the aneurysm (Fig. 5). Follow-up review on July 31 revealed that the patient was alert, but still had severe quadriparesis.

Discussion

Ten cases with an anomalous branch of the ICA, including our case, are summarized in Table 1. In all
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FIG. 3. Enhanced computerized tomography scans taken on April 3, just before the second subarachnoid hemorrhage, at levels 25 mm (left), 33 mm (center), and 37 mm (right) above the orbitomeatal line. The anomalous vessel (fine arrow), right internal carotid artery (double arrows), anterior communicating artery (ACoA) (arrowhead), ACoA aneurysm (thick arrow), and arachnoid cyst in the left temporal region (arrows, right) can be seen.

FIG. 4. Drawing of the operative field as seen through a right frontotemporal craniotomy. An anomalous vessel appears from below the right optic nerve, and passes the prechiasmatic cistern. The aneurysm neck is clipped with a Heifetz clip. The plexiform network of the anterior communicating artery (ACoA), which was not identified on right carotid angiography, is shown.

In cases, the anomalous vessel arose from the ICA at the level of the ophthalmic artery and took an infraoptic and prechiasmatic path to supply the circulation of the ACA. These anomalies were detected at autopsy in two cases, by angiography in three, and by both angiography and operation for an aneurysm in five.

Five of these patients were males and four were females, and the mean age was 47.7 years; the sex and age of one patient is unknown. Anomalous vessels were demonstrated on the right side in six cases, on the left side in two cases, and on both sides in two cases. This anomalous vessel was associated with other vascular abnormalities. These included intracranial aneurysms in five cases, coarctation of the aorta in two cases, carotid-basilar artery anastomosis in two cases, a plexiform ACoA in one case, an anastomotic vessel between both pericallosal arteries in one case, a fused pericallosal artery in one case, agenesis of the ICA in one case, and moyamoya disease in one case.

FIG. 5. Postoperative right carotid angiogram. The aneurysm at the anterior communicating artery has disappeared. The aneurysm clip is not seen due to the subtraction technique.
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**TABLE 1**

*Summary of cases with an anomalous branch of the ICA supplying the circulation of the ACA*

<table>
<thead>
<tr>
<th>Authors, Year</th>
<th>Age (yrs), Sex</th>
<th>Presenting Symptoms</th>
<th>Diagnosis</th>
<th>Anomalous Vessel: Side &amp; Study</th>
<th>Other Anomalies</th>
</tr>
</thead>
<tbody>
<tr>
<td>Turnbull, 1962</td>
<td>81, M none</td>
<td>carcinoma of stomach; died</td>
<td>rt; autopsy</td>
<td>agenesis of lt ICA, lt MCA arose from BA</td>
<td></td>
</tr>
<tr>
<td>Decker, 1966</td>
<td>unknown unknown</td>
<td>unknown</td>
<td>rt; angiography</td>
<td>rt opera artery originated from rt MCA</td>
<td></td>
</tr>
<tr>
<td>McCormick, 1969</td>
<td>67, M none</td>
<td>unknown</td>
<td>rt; autopsy</td>
<td>plexiform ACoA, fused distal ACA, rt SCA arising from PCA proximal to PCoA, duplication of rt PCA distal to PCoA, plexiform network of vessels in area of rt artery of Heubner</td>
<td></td>
</tr>
<tr>
<td>Isherwood &amp; Dutton, 1969</td>
<td>59, F SAH</td>
<td>no source of SAH found</td>
<td>lt; angiography</td>
<td>none</td>
<td></td>
</tr>
<tr>
<td></td>
<td>37, F SAH</td>
<td>two saccular aneurysms: one from normal lt ACA site, &amp; one at rt MCA bifurcation</td>
<td>bilat; angiography &amp; operation</td>
<td>coarctation of aorta</td>
<td></td>
</tr>
<tr>
<td>Teal, et al., 1973</td>
<td>41, M SAH</td>
<td>ACoA aneurysm</td>
<td>rt; angiography &amp; operation</td>
<td>lt PICA arose from distal rt cervical ICA</td>
<td></td>
</tr>
<tr>
<td>Nutik &amp; Dilenge, 1976</td>
<td>22, F SAH</td>
<td>ACoA aneurysm</td>
<td>lt; angiography &amp; operation</td>
<td>each oph artery filled on injection of ipsilateral ECA, lt AICA originated from cavernous portion of lt ICA</td>
<td></td>
</tr>
<tr>
<td>Brismar, et al., 1977</td>
<td>39, M lt-sided hemiparesis, sensory loss, hyperreflexia</td>
<td>lipoma extending to rt cerebellopontine angle</td>
<td>rt; angiography</td>
<td>posterior temporal branch originated from proximal part of rt MCA, multiple congenital defects: rt anosomia, agenesis of rt mandible, harelip with cleft palate, nose deformity</td>
<td></td>
</tr>
<tr>
<td>Lehmann, et al., 1980</td>
<td>23, M crisis of nocturnal syncope†</td>
<td>ACoA aneurysm</td>
<td>bilat; angiography &amp; operation</td>
<td>coarctation of aorta, moyamoya disease</td>
<td></td>
</tr>
<tr>
<td>Fujimoto &amp; Murakami, 1983</td>
<td>60, F SAH</td>
<td>ACoA aneurysm</td>
<td>rt; angiography &amp; operation</td>
<td>anastomotic vessel between both pericallosal arteries, arachnoid cyst in lt temporal region</td>
<td></td>
</tr>
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</table>

*Abbreviations: ICA = internal carotid artery; ACA = anterior cerebral artery; SAH = subarachnoid hemorrhage; MCA = middle cerebral artery; ACoA = anterior communicating artery; BA = basilar artery; Oph = ophthalmic; SCA = superior cerebellar artery; PCA = posterior cerebral artery; PCoA = posterior communicating artery; PICA = posterior inferior cerebellar artery; ECA = external carotid artery; and AICA = anterior inferior cerebellar artery. † Due to myocardial decompensation.

Subarachnoid hemorrhage occurred in five of the nine fully described cases. A total of six aneurysms were detected in five cases, but the source of the hemorrhage in one case could not be found. The six aneurysms were located on the ACoA in four cases, middle cerebral artery bifurcation in one case, and the ICA bifurcation at the site where a normal ACA would have taken origin in one case. In these 10 cases the incidence of ACoA aneurysms is 67%, which seems to be higher than the incidence in patients without an anomalous vessel (34%). This may be attributable to the change in flow dynamics that results from an anomalous vessel. However, it is difficult to differentiate between a real and a coincidental association because of too few cases. If this is only an incidental association, we could find no other specific symptom caused by this anomalous vessel.

The anomalous branch of the ICA supplies the circulation to the territory of the ACA, either partially or almost completely, and shares the function of the ACA. Isherwood and Dutton inferred that this anomalous vessel functions as a part of the ACA. In two autopsy cases, however, a hypoplastic A1 segment of the ACA was confirmed on the ipsilateral side of the anomalous vessel. Therefore, it seems reasonable that this vessel is not a part of the ACA. Nutik and Dilenge named this anomaly "carotid-anterior cerebral artery anastomosis."

The genesis of this anomaly remains to be clarified; however, previous authors have offered possible explanations for its development. Isherwood and Dutton subscribed to the theory of prechiasmal anastomosis, as reported by Dawson. Prechiasmal anastomosis at the medial aspect of the intracranial segments of the two optic nerves and at the anterior border of the chiasm consists of the prechiasmal branches of the ophthalmic artery, superior chiasmal arteries from the ACA's, and inferior chiasmal arteries from the ICA's. Dawson suggested that the variations in the caliber of the component vessels of this anastomotic system were caused by differences in the components of the arterial circle of Willis. The carotid artery-ACA anas-
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FIG. 6. Difference in the schema of Padget, and Lasjaunias and Moret in embryos from 5 to 20 mm in crown-to-rump length. A and B show normal development, while C shows a possible explanation for an anomalous branch of the internal carotid artery. For details see text. Abbreviations: PVO = primitive ventral ophthalmic artery; PDO = primitive dorsal ophthalmic artery; PM = primitive maxillary artery; PO = primitive olfactory artery; AC = anterior cerebral artery; MC = middle cerebral artery; Ach = anterior choroidal artery; Pcom = posterior communicating artery; AO = adult ophthalmic artery; and AV = anomalous vessel.

This anomalous vessel is frequently accompanied by other vascular irregularities secondary to embryogenic disorders, such as carotid-basilar artery anastomosis, fused pericallosal artery, and a plexiform ACoA. Therefore, a second hypothesis related to the complicated and late emergence of the definitive ophthalmic artery seems to be correct. Padget has reported that the origin of the primitive ventral and dorsal ophthalmic arteries is from sites opposite the future anterior choroidal artery and posterior communicating artery. In her opinion, the adult ophthalmic artery should be formed through caudal migration of the primitive ventral ophthalmic artery, while the primitive dorsal ophthalmic artery regresses. On the other hand, Lasjaunias and Moret have reported the presence of two primitive ophthalmic arteries — one ventral, originating from the future ACA, and one dorsal, originating from the intracavernous part of the ICA. The adult ophthalmic artery should then be formed by caudal migration of the primitive dorsal artery.

Brismar et al. have offered explanations for various anomalous origins of the adult ophthalmic artery (Fig. 6). They suggested that the adult ophthalmic artery originating from the middle cerebral artery and posterior communicating artery could be the result of incomplete migration of the primitive ventral ophthalmic artery (as proposed by both Lasjaunias and Moret) or the primitive dorsal ophthalmic artery (as suggested by Padget). The adult ophthalmic artery could originate from the intracavernous part of the ICA by persistence of the primitive dorsal ophthalmic artery (Lasjaunias and Moret) or the primitive maxillary artery (Padget). Duplication of the adult ophthalmic artery could be explained through caudal migration of the primitive ventral ophthalmic artery and persistence of the primitive dorsal ophthalmic artery (Lasjaunias and Moret), or caudal migration of the primitive dorsal ophthalmic artery and persistence of the primitive maxillary artery (Padget). Brismar et al. have also pointed out that the schema of Lasjaunias and Moret is better than that of Padget in explaining the anomalous branch of the ICA and the origin of the adult ophthalmic artery from the ACA. The former could be readily explained as the persistence of the ventral ophthalmic artery in conjunction with regression of the proximal part of the ACA, and the latter as persistence of the primitive ventral ophthalmic artery, as illustrated by the schema of Lasjaunias and Moret.
References


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