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A Hyperplastic Anterior Choroidal Artery with Double Persistent Anastomotic Channels

Chi-Jen Chen and A. James Barkovich

Summary: We describe a case of a hyperplastic anterior choroidal artery (AChA) with double persistent anastomotic channels. This hyperplastic AChA, which was detected at vertebral angiography, is different from previously reported cases, because it has an unusual, secondary anastomotic channel.

In 1907, Blackburn (1) was the first to describe patients in whom the territories of the posterior cerebral artery (PCA) were supplied mainly by the anterior choroidal artery (AChA). Since then, such arteries have been described by many authors and have been termed hyperplastic anomalies of the AChA (1–6) or anomalous temporal, calcarine, parietooccipital arteries originating directly from the internal carotid artery (ICA) (7–9) or from two separate trunks of the PCA (10). Although many variations of the hyperplastic AChA have been reported, all have had just one persistent anastomotic channel detected at carotid angiography (6). We report a case of a hyperplastic AChA with two persistent anastomotic channels, seen on vertebral angiography.

Case Report

A 20-year-old man was admitted because of sudden-onset left hemiparesis. CT revealed a small hematoma in the right putamen. Cerebral angiography showed no obvious vascular malformation or tumor stain. A hyperplastic left AChA was noted on left common carotid angiograms (Fig 1A and B) that fed the left calcarine artery by a persistent anastomosis between the left AChA and left PCA distal to branching of the temporal artery. This hyperplastic AChA was also seen on vertebral angiograms because of an enlarged anastomotic channel between temporal branches of the left PCA and uncus branches of the left hyperplastic AChA (Fig 1C and D).

Discussion

The normal AChA has potential anastomoses with its neighboring arteries, especially with the posterior communicating artery (PCoA)-PCA system (2, 4, 11). Five anastomotic channels are recognized: 1) near the optic tract with branches from the PCoA; 2) near the

cerebral peduncle with the proximal PCA; 3) near the piriform cortex (uncus branches) with the temporal and hippocampal branches of the PCA; 4) over and around the lateral geniculate body with PCA branches, including the lateral posterior choroidal artery; and 5) in the choroid plexus with posterior choroidal branches (6) (Fig 2). Besides these anastomotic channels, there is also a reciprocal relationship between the territories supplied by the AChA and the PCoA-PCA system (5, 6). For example, if the proximal segment of the PCA is hypoplastic, the AChA will enlarge and supply the remaining distal segment of the PCA via the anastomoses. Because of this reciprocal relationship, most authors believe the anomalous AChA is a hyperplastic variant rather than a true vascular anomaly (1–6). Different phenotypes of the hyperplastic AChA may evolve depending on which segments of the PCoA-PCA system are hypoplastic and which anastomotic channels remain (6). The result may be any of a wide spectrum of such arteries, representing transitional forms between a normal AChA and extreme hyperplasia that totally replaces the PCoA-PCA system on the same side.

Although many variants of a hyperplastic AChA have been reported, all seem to have a single persistent anastomotic channel from the ICA-AChA, with hypoplasia of the segment of the PCoA-PCA just proximal to this persistent anastomosis (6). No double persistent anastomotic channel with the second channel fed from the vertebrobasilar system has been mentioned before. In our case, besides the usual persistent anastomotic channel (Fig 2, anastomotic branch 4), a second persistent anastomosis connected the temporal branches of the PCA with the uncus branches of the hyperplastic AChA (Fig 2, anastomotic branch 3). More interesting was that the PCA segment proximal to this anastomotic branch 3 (temporal-uncus anastomosis) persisted without hypoplasia. This seems to break the rule of a reciprocal relationship: that one anastomotic channel persists and the receiving vessel involutes proximal to the anastomosis (6). Does the second anastomosis have a

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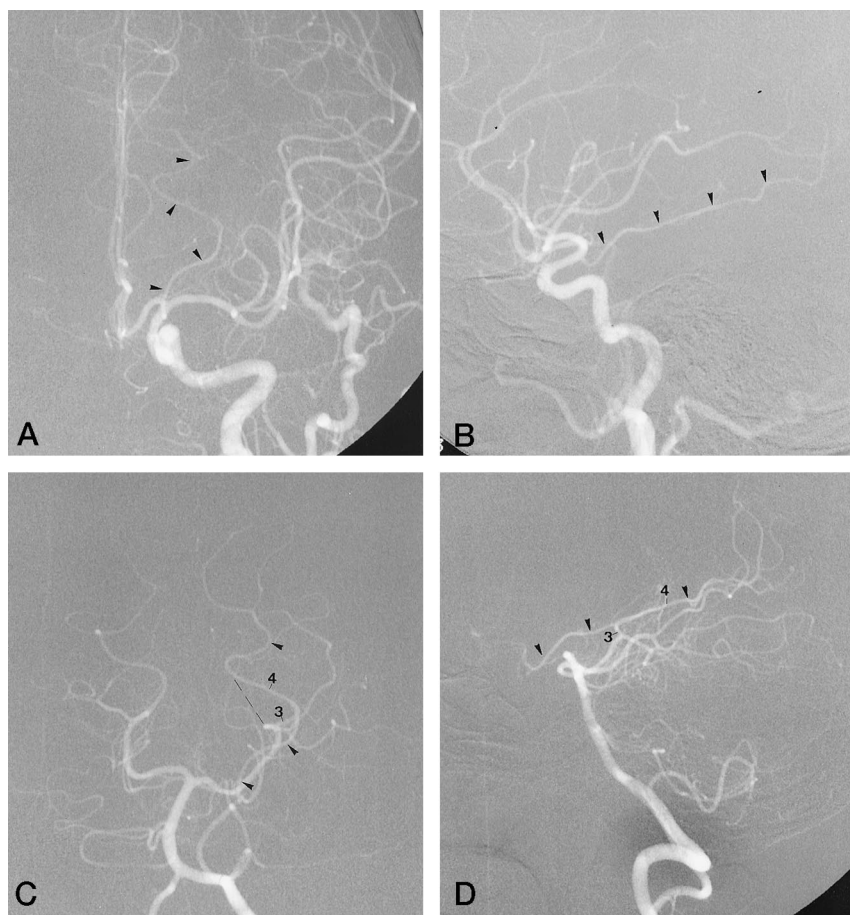


FIG 1. 20-year-old man with a small right putaminal hematoma.

A and B, Left common carotid angiogram in anteroposterior (A) and lateral (B) projections show a hyperplastic left AChA (arrowheads) that feeds the left calcarine artery via an anastomotic branch. The left PCoA is hypoplastic. The anastomotic branch 3 between uncinal branches of the AChA and temporal branches of the PCA fills poorly because of the unopacified flow from the PCA.

C and D, Left vertebral angiograms in Towne (C) and lateral (D) projections show the hyperplastic AChA (arrowheads) because of the existence of anastomotic branch 3. The dotted line in C represents the hypoplastic segment of the PCA proximal to anastomotic branch 4, which connects the calcarine artery and AChA.

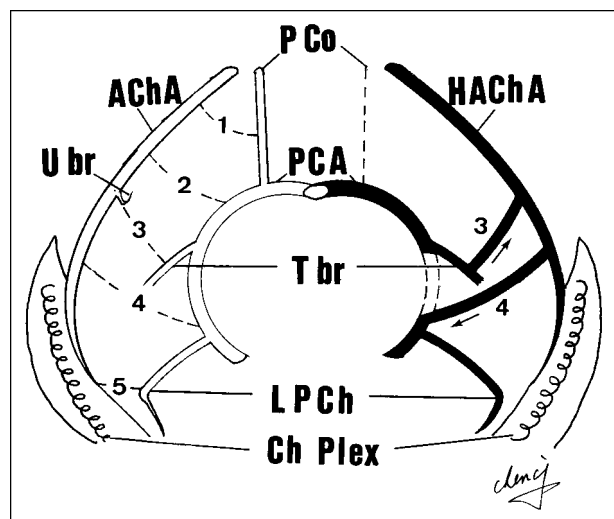


FIG 2. Diagram shows the potential anastomotic channels between the AChA and the PCoA-PCA system on the right side and the actual anastomosis occurring in our case on the left side. 1, 2, 3, 4, 5 represent the locations of these potential anastomotic channels: 1, over optic tract with branches from PCoA; 2, over cerebral peduncle with proximal PCA; 3, over piriform cortex (uncal branches) with PCA branches (temporal and hippocampal branches); 4, over and around lateral geniculate body with PCA branches; 5, in the choroid plexus with posterior choroidal branches. Double dotted line on the left side represents the hypoplastic segment of the PCA proximal to the first anastomotic channel (anastomosis, 4). Arrows on the left side indicate the direction of flow through these anastomotic channels. AChA indicates anterior choroidal artery; HACHA, hyperplastic anterior choroidal artery; U br, uncinal branches of the AChA; T br, temporal branches of the PCA; L P Ch, lateral posterior choroidal artery; Ch Plex, choroid plexus; P Co, posterior communicating artery; PCA, posterior cerebral artery.

pathogenetic mechanism different from that of the first one?

Our explanation is as follows: Originally, the hyperplastic AChA forms from persistent anastomosis 4 (anastomosis over and around the lateral geniculate body with the PCA branches) with hypoplasia of the PCA segment proximal to this anastomosis (broken line in Figs 1C and 2). At this time, the hyperplastic AChA is completely separate from the proximal PCoA-PCA system. After that, the proximal end of

the hyperplastic AChA becomes transiently obstructed, which changes the blood supplier of the distal PCA from the proximal AChA to the proximal PCA by opening a potential channel between the PCA branches (temporal and hippocampal branches) and the piriform cortex (uncal branches) (anastomotic branch 3). In the literature, a similar situation has been reported in one case of hyperplastic AChA with an aneurysm arising from its origin (6). Preoperative vertebral angiograms did not opacify the anomalous

AChA. After aneurysmal clipping, carotid angiograms showed complete occlusion of the hyperplastic AChA at its origin in addition to the disappearance of the aneurysm. Vertebral angiography showed this hyperplastic AChA to be opacified via these potential anastomotic channels, which explains why the patient had no serious symptoms postoperatively.

Conclusion

We speculate that the secondary anastomotic channel (anastomotic branch 3) remained open even after the obstruction at the proximal hyperplastic AChA resolved, resulting in persistent communication of the hyperplastic AChA with both the carotid and vertebrobasilar systems.

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