Dural-cortical anastomosis in pial arteriovenous malformation

Case report

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A patient is reported with a purely pial arteriovenous malformation (AVM) supplied from the posterior parietal artery. The prominent middle meningeal artery contributed to opacification of the angular branches distal to the AVM, but did not contribute to the AVM. After total removal of the AVM, the angular branches became opacified from the middle cerebral artery. Review of the literature suggests that hypertrophied dural arteries which do not contribute to the AVM's but which do opacify the cortical branches distal to the AVM's are rare.

KEY WORDS □ cerebral arteriovenous malformation □ external carotid artery □ dural artery □ cerebral ischemia □ blood flow

I n cerebral arteriovenous malformations (AVM's), preoperative angiograms usually show that the brain is poorly vascularized except for the AVM's, even though the clinical manifestations attributable to ischemia are absent. After total removal of the AVM's, angiograms demonstrate normal visualization of cerebral vessels. However, it is possible that normograde blood flow is preserved and that the contrast material has been diluted so that it is not seen on the preoperative angiograms. It is also reasonable to assume that there may be collateral circulation from other arteries, including the dural arteries. Dural arteries may contribute to the leptomeningeal-cortical blood supply as collateral anastomoses in patients with cerebral occlusive disease. Newton and Troost10 wrote that similar collateral contribution from the dural arteries might occur with AVM's that were supplied solely from the pial arteries. However, we have not found in the literature, including recent articles concerning AVM's,3, 6, 10-12, 14 and superselective2 and selective external carotid angiography,1-7, 9 any cases in which dural arteries contributed solely to the cortical branches distal to the AVM's.

Case Report

This 43-year-old woman was admitted to the University Hospital on May 2, 1977, because of five seizures beginning at the age of 41 years. Episodes began in the left arm.

Examination. On admission neurological examination showed slight symmetrical hyperreflexia and extensor plantar responses on the left. X-ray films of the skull demonstrated a prominent groove of the right middle meningeal vessels. Contrast-enhanced computerized tomography scans revealed a worm-like area of increased density in the right parietal region. Transfemoral selective cerebral angiography showed that the right middle cerebral artery was dilated, and the posterior parietal branch directly fed the AVM in the parietal lobe. Other cerebral vessels were poorly filled. The AVM drained into the superior sagittal and transverse sinuses (Fig. 1). The posterior branch of the right middle meningeal artery was also dilated and filled the angular branches distal to the AVM via a leptomeningeal anastomosis but did not contribute to the AVM itself (Fig. 2).
Dural-cortical anastomosis in pial AVM

**Operation.** The AVM was totally removed through a right parietal craniotomy. The posterior branch of the right middle meningeal artery was coagulated and divided at the time of dural opening. The AVM was purely pial in location and did not receive any dural blood supply.

**Postoperative Course.** Common carotid angiography demonstrated that the circulation of the right cerebral hemisphere was normalized, although the posterior parietal artery was still larger than usual. The angular branches were now opacified from the middle cerebral artery and the middle meningeal artery no longer opacified the angular branches (Fig. 3). The patient was discharged from the hospital on June 9, 1977, without any neurological deficit.

**Discussion**

Newton and Cronqvist\(^8\) reported that supratentorial AVM’s received a contribution from dural vessels in 21% of cases. Dural arteries may participate in mixed pial and dural or dural AVM’s, or may contribute to purely pial AVM’s through leptomeningeal anastomosis. Kusske and Kelly\(^5\) reported that partial embolization of unresectable AVM’s that shunted normal blood away from the brain improved the clinical symptoms by increasing cerebral perfusion. In many AVM’s that are large enough or situated in important regions, such as speech and motor-sensory areas, the distal brain appears angiographically to be poorly vascularized, but ischemic symptoms may not develop.\(^3,4,6,8,12-14\) Adequate distal perfusion to the brain, both normograde and via the collateral circulation, may not be visible on the angiograms because of dilution of contrast material.

In our case, the prominent middle meningeal artery did not contribute to the purely pial AVM, which was
Fig. 3. Postoperative common carotid angiogram, lateral view, demonstrates that the angular branches (arrows) are now opacified from the middle cerebral artery.

fed solely by the posterior parietal artery, but it did contribute to the angular branches. Postoperatively, the angular branches were filled from the middle cerebral artery in an entirely normal fashion. It seems reasonable to assume that the brain distal to the AVM may have been deprived of direct blood supply from the middle cerebral artery by a shunt of the AVM itself and that an anastomotic channel such as the dural-leptomeningeal anastomosis developed as demonstrated on the angiograms. Although this event may occur naturally, we have not found an example of such an occurrence in the literature. In our case, the angular branches might have received blood supply from both the middle cerebral and the middle meningeal arteries, and preoperatively the supply from the former had not been clearly visualized on the angiograms because of dilution of the contrast material.

References


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