Dual cerebral and meningeal supply to giant arteriovenous malformations of the posterior cerebral hemisphere

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Seven cases of giant posterior hemisphere arteriovenous malformations are described. The significance of meningeal feeding vessels from the external carotid artery in addition to the primary cerebral supply through the internal carotid and vertebral arteries to these malformations is discussed. The necessity of bilateral selective external carotid arteriography is stressed, and the value of preoperative embolization is questioned.

Key Words • arteriovenous malformation • selective external carotid angiography • embolization

The diagnosis and management of cerebral arteriovenous malformations (AVM's) has progressed dramatically in recent years as a result of new developments in the field of neuroradiology. Most notably, computerized tomography (CT) scanning has assisted in early detection of these lesions and in demonstrating those that are angiographically occult, 6, 8 and embolization techniques have aided attempts to decrease the vascularity of intracranial AVM's as a preoperative adjunct to surgery, 4, 6, 8, 10, 15 or as a definitive mode of therapy. 4, 10, 18

We have routinely used magnification and subtraction techniques, and, more recently, selective external carotid arteriography to visualize additional meningeal feeding vessels from the external carotid artery (ECA) in cases of large cerebral AVM's. A description of seven consecutive cases of large malformations of the posterior temporal and parieto-occipital regions with a dual cerebral-meningeal supply forms the basis of this report.

Review of the Literature

In the Cooperative Study, Perret and Nishioka 15 reviewed 545 cases of craniocerebral AVM's and found no cases of intracranial AVM's with contribution from extracranial and meningeal branches of the ECA. The first in-depth study of intracranial AVM's with reference to their dural blood supply is found in a report of the 129 cases by Newton and Cronqvist in 1969. 12 They found that 21% of supratentorial AVM's (103 cases) had "dural involvement as judged by meningeal arterial contribution to the malformation." This included cases with "pure dural" (6%), or "mixed pial dural" (15%) malformations. In the case of posterior fossa AVM's, they corroborated their own experience, 11, 13, 17 and that of others, 2, 7, 14 that meningeal supply was more common; 50% of their cases demonstrated dural contribution, with 15% of their cases having a "pure dural" supply. They stressed the value of selective external carotid arteriography and the need to study the contralateral side when the ipsilateral side showed dural contribution to the malformation.

In 1974, Dahl and Kline 8 reported two cases of intraparenchymal AVM's with predominant contribution from the ECA. They added 299 additional cases from the literature and reported that only 23 of these cases, including their own two cases, had any significant flow via the ECA. They concluded that significant ECA supply to intraparenchymal AVM's was unusual.
TABLE 1

Angiographic findings in seven cases of giant arteriovenous malformation (AVM)*

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Location and Description</th>
<th>Blood Supply</th>
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<tbody>
<tr>
<td>1</td>
<td>AVM, 5½ × 5½ cm in size, in left temporoparietal region; rapid shunting into large cortical veins draining primarily into SSS</td>
<td>cerebral: Lt MCA &amp; PCA; dural: Lt ECA via posterior branch of MMA</td>
</tr>
<tr>
<td>2</td>
<td>large AVM in rt occipital area</td>
<td>cerebral: predominant blood supply from rt PCA; dural: large meningeal branches off both ECA's &amp; posterior meningeal branch off rt VA; also supply via a large rt occipital artery</td>
</tr>
<tr>
<td>3</td>
<td>large AVM in lt temporoparietal region</td>
<td>cerebral: posterior temporal branch of lt MCA &amp; lt PCA, also lt AChA; dural: from enlarged posterior branch of lt MMA</td>
</tr>
<tr>
<td>4</td>
<td>large AVM in lt posterior parietal region with large draining vein entering SSS</td>
<td>cerebral: primarily from large branch of lt MCA, &amp; vertebrobasilar system via left PCoA; dural: from posterior branch of lt MMA</td>
</tr>
<tr>
<td>5</td>
<td>giant AVM of rt parieto-occipital region</td>
<td>cerebral: major feeder from lt MCA and PCA, also from lt ACA; dural: from posterior branch of rt MMA</td>
</tr>
<tr>
<td>6</td>
<td>giant AVM in posterior temporal &amp; angular gyrus region of lt hemisphere</td>
<td>cerebral: multiple feeders off lt MCA &amp; lt PCA, also lt AChA; dural: from enlarged anterior &amp; posterior branches of lt MMA, also from posterior branch of lt STA</td>
</tr>
<tr>
<td>7</td>
<td>large AVM in lt parieto-occipital region with extension into thalamus</td>
<td>cerebral: lt ACA, MCA, &amp; PCA, with contribution from rt ICA as well; dural: from branches off both lt &amp; rt MMA's &amp; both ECA's</td>
</tr>
</tbody>
</table>

*Abbreviations: ACA = anterior cerebral artery; AChA = anterior choroidal artery; ECA = external carotid artery; ICA = internal carotid artery; MCA = middle cerebral artery; MMA = middle meningeal artery; PCA = posterior cerebral artery; PCoA = posterior communicating artery; SSS = superior sagittal sinus; STA = superficial temporal artery; VA = vertebral artery.

Clinical Material and Methods

We have surgically treated 33 cases of intracranial supratentorial AVM's at Emory University and affiliated hospitals from November, 1975, to December, 1978. Seven of the patients had giant posterior temporal and parieto-occipital malformations, with a primary cerebral blood supply via branches of the internal carotid and vertebral artery (Table 1). These patients also had feeding vessels from posterior meningeal branches from the ipsilateral and, in some cases, both ECA's. Angiography for this group of patients always included selective ECA injections, in addition to the routine four vessels visualized in all cases of large cerebral AVM's. Magnification and subtraction techniques were performed routinely. Seven cases of giant AVM with a dual cerebral and meningeal supply were identified. All of these AVM's were located in the posterior temporal and parietal regions and received rapid blood flow. We did not include AVM's of the posterior fossa in this series. Six patients were treated surgically, and underwent preoperative embolization procedures in an attempt to decrease the vascularity of the lesion. Our embolization techniques have been described in a recent publication.8

Case Reports

Case 1

This 37-year-old woman was admitted with severe left-sided headaches, blurred vision in her left eye, and generalized seizures. She had suffered a subarachnoid hemorrhage (SAH) 7 years previously. An audible bruit was heard over the left hemicranium, and she had a mild right-sided hyperreflexia and hemisensory deficit. Cerebral angiography demonstrated an AVM in the left temporoparietal region with dual blood supply (Fig. 1 upper). Embolization was performed on two occasions, with a total of 205 2-mm and 21 1-mm Silastic spheres introduced through the left internal carotid artery (ICA), and this was believed to have decreased the vascularity of the malformation by 40% (Fig. 1 lower). Subsequently, a parieto-occipital craniotomy was performed with total excision of the AVM. Blood loss was estimated at 8000 cc. After surgery, the patient did well except for a residual right superior homonymous quadrantanopsia.

Case 2

This 53-year-old woman was awakened at 4:00 a.m. by a severe headache associated with a stiff neck, and
she was admitted with the diagnosis of SAH. She had had signs of meningismus, a left homonymous hemianopsia, left spastic hemiparesis, and left hemisensory deficit. Cerebral angiography demonstrated a large AVM in the right occipital area (Fig. 2). Embolization was performed with 20 Gelfoam pledgets into the right occipital and internal maxillary arteries, which had provided feeding vessels to the dural component of the malformation. She then underwent right temporoparieto-occipital craniotomy with total excision of the AVM. Blood loss was approximately 1500 cc, most of which occurred during...
separation of the dural component from the cerebral AVM. Three months postoperatively, she was well except for a persistent visual field loss and mild spastic left hemiparesis.

Case 3

This 22-year-old man had a previous history of SAH at 12 years of age. He was admitted with progressive headaches and seizures. Cerebral angiography showed a large left temporoparietal AVM with cerebral and meningeal supply (Fig. 3). Embolization was performed with 19 1-mm and 149 1.5-mm Silastic spheres to both ICA and ECA feeding vessels. Embolization resulted in substantial reduction of flow to the AVM only from the ECA circulation. Subsequently, a temporoparieto-occipital craniotomy was performed, with a blood loss of approximately 10,000 cc. Postoperative angiography showed no residual AVM. Recovery was uneventful, and the patient was discharged without deficit except for a right superior homonymous quadrantanopsia.

Case 4

This 57-year-old man had had a history of “blackout spells” since 1942. He was admitted with a 3-day history of progressive dysphasia and right hemiparesis. Cerebral angiography on admission demonstrated a large AVM in the left posterior parietal region with dual blood supply. Embolization was carried out, with 80 1-mm and 121 0.5-mm Silastic pellets introduced through the left ICA; there was no change in flow to the AVM. He underwent left parieto-occipital craniotomy with total excision of the AVM and with a blood loss of 500 cc. The dural AVM was not dissected from the cerebral component until all the major feeding vessels had been interrupted. One month postoperatively, the patient was doing well except for a residual visual field loss.

Case 5

This 34-year-old nurse was admitted with SAH. On examination, she had a left inferior homonymous quadrantanopsia and a faint bruit over the right posterior parietal region. Angiography demonstrated an AVM in the right parieto-occipital region (Fig. 4 upper). The AVM was then embolized via the ICA, with approximately 200 1-mm Silastic pellets, but there was no change in vascularity in the malformation. Subsequently, a right occipital craniotomy was performed with total excision of the malformation (Fig. 4 lower). Blood loss was 3000 cc. The patient’s postoperative course was complicated by a right frontal subdural hematoma which was uneventfully evacuated. She was discharged with a left homonymous hemianopsia but with no other deficits.

Case 6

This 70-year-old man had a history of a previous craniotomy for removal of a “very vascular” tumor. He had had a previous SAH in 1965. He was admitted with an SAH. On admission, he was obtunded and confused. There was a bruit over the left hemicranium, and marked meningismus. He had a right homonymous hemianopsia and spastic right
Dual supply to giant AVM's of the PCA

Fig. 3. Case 3. Left: Lateral view of left internal carotid artery angiogram shows a large arteriovenous malformation (AVM) in the temporoparietal region. Right: Lateral view of a selective injection of the left external carotid artery demonstrates meningeal supply from the posterior branch of the middle meningeal artery.

hemiparesis. Admission CT scan showed a left temporoparietal enhancing lesion with an intraventricular hemorrhage. Cerebral angiography showed a large left posterior temporal AVM. Embolization of all feeding vessels with 194 1.5-mm Silastic spheres demonstrated no change in the vascularity of the malformation except for complete occlusion of the external carotid supply (Fig. 5). Subsequent re-embolization was complicated by a dense but transient right hemiplegia following occlusion of the left anterior choroidal artery, a major feeder to the AVM. Eighteen days from the initial hemorrhage, a large craniotomy was performed, with total excision of the malformation. Blood loss was estimated at 8000 cc. The postoperative course was complicated by an intraventricular hematoma requiring immediate reoperation, but the patient remained comatose and died 2 weeks postoperatively.

Case 7

This 29-year-old man had a long history of a seizure disorder. He was admitted after a seizure episode without SAH. Cerebral angiography demonstrated a left parieto-occipital malformation with dual supply and deep extension involving the thalamus. The patient's seizures were controlled on the appropriate anticonvulsant drugs, and no surgical intervention was undertaken.

Discussion

Seven cases of giant AVM's of the posterior temporal and parieto-occipital regions have been described. In all instances there was a primary cerebral arterial supply to the lesion, and a small but significant meningeal contribution from the ECA. The cerebral supply was through enlarged branches of the ICA and/or vertebral artery via anterior, middle, or posterior cerebral arteries. The meningeal or dural supply was from the ECA distribution through branches of the middle meningeal artery. One malformation had bilateral supply from both ICA's (Case 7), and two (Cases 2 and 7) had bilateral meningeal supply. Two patients had additional contribution from the ipsilateral posterior branch of the superficial temporal artery (Case 6) and from the occipital artery (Case 2). In one case, the posterior meningeal branch of the vertebral artery was involved in feeding the malformation (Case 2). Selective bilateral ECA arteriography was essential in demonstrating meningeal supply to these AVM's.
At surgery, the dural component of the AVM was in all instances intimately related to the underlying cerebral AVM, making dural opening technically difficult. The importance of preoperative awareness of this entity is twofold. First, in cases where a large component of the malformation is dural, preoperative embolization may be of value to decrease the meningeal supply, despite the fact that in two patients (Cases 2 and 3), in whom the flow through the meningeal vessels had been reduced, the surgical procedure was not technically easier than in the other patients. Second, when a dural component to the
AVM is known to exist, the surgical procedure could be made significantly easier, and blood loss reduced, if the major intracranial feeders were interrupted surgically before the dural component is elevated from the cerebral AVM. It is important that the dural and cerebral incision be made in front of the dural-cerebral malformation, and the dural and cerebral components of the malformation left contiguous and undisturbed until the major cerebral blood supply to the malformation has been interrupted anteriorly and its cerebral feeding vessels cauterized and divided (Fig. 6).

All six patients who were treated surgically underwent preoperative embolization in an attempt to reduce operative blood loss. Silastic spheres, ranging in size from 0.5 to 2 mm in diameter, were used in the five patients in whom the cerebral component of the malformation was embolized via the ICA and/or vertebral artery in addition to the ECA. In one patient (Case 2), embolization was performed with Gelfoam pledgets via the ECA branches only. We found no significant reduction in vascularity at the time of surgery when the primary cerebral supply was embolized, despite the number or size of emboli used. Even in Case 1, which was the only case in which preoperative embolization was thought to have angiographically reduced flow, the blood loss at surgery was not reduced when compared with the other five cases. There was no correlation between preoperative embolization and reduction of blood loss at surgery. Therefore, in this series we have found preoperative embolization to be of limited value in the surgical management of large cerebral AVM's, contrary to the experience of others.

A potentially important observation was noted in Cases 4 and 5. Shortly after complete removal of the AVM, postoperative contrast-enhanced CT scan demonstrated serpiginous structures in the area contiguous to the margin of the previously resected AVM. Initial interpretation of this finding in Case 4 was that it was "residual AVM;" however, repeat angiography revealed complete obliteration of the malformation. The initial CT scan impression in Case 5 was "cerebral abscess vs. residual AVM with mass effect" (Fig. 7). Nevertheless, correlation with the preopera-
Fic 6 Artist’s impression of the intraoperative view of a large arteriovenous malformation (AVM) with dual cerebral and meningeal supply. The dural opening is made anterior to the AVM. No attempt is made to separate the two components of the AVM. The cortical incision is made in front of the malformation with the dural and cerebral components left contiguous until the major cerebral supply to the malformation has been interrupted.

Fig. 6. Artist's impression of the intraoperative view of a large arteriovenous malformation (AVM) with dual cerebral and meningeal supply. The dural opening is made anterior to the AVM. No attempt is made to separate the two components of the AVM. The cortical incision is made in front of the malformation with the dural and cerebral components left contiguous until the major cerebral supply to the malformation has been interrupted.

tive and postoperative angiograms (Fig. 4), demonstrated that the CT scan findings were consistent with residual dilated feeding cerebral vessels which previously had supplied the AVM and now enhanced more impressively as a result of stagnant flow following their surgical interruption. In view of these findings, postoperative CT scans following excision of large AVM's should be interpreted with caution. Postoperative angiography is necessary to demonstrate complete excision of an AVM.

Fig. 7. Case 5. Left: Preoperative computerized tomography scan with contrast enhancement, showing a large arteriovenous malformation (AVM) in the right parieto-occipital region. Right: Postoperative scan with contrast enhancement showing serpiginous structures at the margin of the previously resected AVM associated with mass effect. These findings are consistent with cerebral abscess, or even residual AVM; nevertheless, angiography confirmed that these were postoperative changes (see text).

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