Management of dural arteriovenous malformations of the anterior cranial fossa

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Eight patients with dural arteriovenous malformations (AVM's) of the anterior cranial fossa are presented, and the pertinent literature is reviewed. Unlike cases of dural AVM's in other locations, sudden massive intracerebral hemorrhage was the most frequent reason for presentation. Other symptoms included tinnitus, retro-orbital headache, and a generalized seizure. The malformations were supplied consistently by the anterior ethmoidal artery, usually in combination with other less prominent feeding vessels. The lesion's venous drainage was through the superior sagittal sinus via a cortical vein; in addition, in two cases a subfrontal vein drained the AVM. A venous aneurysm was encountered near the site of anastomosis with the dural feeder in most cases, and was found in all patients who presented with hemorrhage. The AVM was obliterated surgically in six patients, with favorable results achieved in five. One patient died postoperatively from a pulmonary complication. Because of their anatomy and proclivity for hemorrhage, these vascular malformations represent a unique group of dural AVM's. Surgical management of anterior fossa dural AVM's carries low morbidity, and is indicated when the lesions have caused hemorrhage or when there is an associated venous aneurysm.

KEY WORDS • anterior ethmoidal artery • anterior fossa • dural lesion • arteriovenous malformation • cerebral hemorrhage

Dural arteriovenous malformations (AVM's) are rare vascular anomalies that most frequently involve the cavernous, transverse, or sigmoid sinuses. Most intracranial dural AVM's present with relatively benign symptoms such as headache or subjective bruit, with only a small percentage of these lesions causing intracranial hemorrhage. Some remain asymptomatic. Those in the anterior cranial fossa, however, form a distinct subgroup that has an unusually high incidence of intracerebral hemorrhage. Anterior fossa dural AVM's were first reported in 1963, and have been reported only sporadically since that time. In this report, we review eight new cases, with a focus on the unique anatomic features that have relevance to their clinical presentation, natural history, and surgical management.

Summary of Cases

The features of eight patients with dural AVM's of the anterior cranial fossa were collected from a retrospective review of clinical, radiological, and surgical records (Table 1). There were seven men and one woman, ranging in age from 44 to 61 years, with a mean age of 52 years.

Presenting Symptoms

The presenting symptom in four patients was apoplectic. In these cases, computerized tomography (CT) showed evidence of an acute frontal hemorrhage with mass effect (Fig. 1). All of these patients were comatose. One patient presented with a small spontaneous occipital hemorrhage; cerebral angiography performed to evaluate the cause of the hemorrhage failed to show a vascular lesion in the occipital region, but demonstrated an incidental small dural AVM of the anterior fossa. One patient presented with a generalized seizure, and was found on CT to have a small frontal hemorrhage without mass effect. No preceding neurological symptom was elicited from any of the patients who sustained hemorrhages, and there was no history of trauma. Two patients had no evidence of intracranial hemorrhage. One of these patients presented with tinnitus, and dur-
TABLE 1

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (yrs), Sex</th>
<th>Clinical Presentation</th>
<th>Feeding Artery</th>
<th>Draining Vein</th>
<th>Aneurysmal Dilatation</th>
<th>Surgery</th>
<th>Surgical Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>61, M</td>
<td>ICH, obtundation</td>
<td>AEA (bilat)</td>
<td>superficial, SSS</td>
<td>yes</td>
<td>yes, AD excised</td>
<td>died</td>
</tr>
<tr>
<td>2</td>
<td>54, M</td>
<td>ICH, obtundation</td>
<td>AEA (bilat), STA</td>
<td>superficial, SSS subfrontal</td>
<td>yes</td>
<td>yes</td>
<td>good</td>
</tr>
<tr>
<td>3</td>
<td>48, M</td>
<td>ICH, seizure</td>
<td>AEA, ACA, STA</td>
<td>superficial, SSS</td>
<td>yes</td>
<td>yes, falk excised</td>
<td>good</td>
</tr>
<tr>
<td>4</td>
<td>50, M</td>
<td>ICH, incidental (occipital hemorrhage)</td>
<td>AEA</td>
<td>superficial, SSS</td>
<td>no</td>
<td>no</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>55, M</td>
<td>tinnitus</td>
<td>AEA, STA, IMA (bilat)</td>
<td>superficial, SSS subfrontal, BV</td>
<td>yes</td>
<td>no</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>54, M</td>
<td>retro-orbital head-ache</td>
<td>AEA, STA, IMA (bilat)</td>
<td>superficial, SSS</td>
<td>no</td>
<td>yes</td>
<td>good</td>
</tr>
<tr>
<td>7</td>
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<td>ICH, obtundation</td>
<td>AEA</td>
<td>superficial, SSS</td>
<td>yes</td>
<td>yes</td>
<td>good</td>
</tr>
<tr>
<td>8</td>
<td>50, F</td>
<td>ICH, obtundation</td>
<td>AEA, MMA</td>
<td>superficial, SSS</td>
<td>yes</td>
<td>yes, AD excised</td>
<td>good</td>
</tr>
</tbody>
</table>

* Abbreviations: AVM = arteriovenous malformation; ICH = intracerebral hemorrhage; AEA = anterior ethmoidal artery; ACA = anterior cerebral artery; STA = superficial temporal artery; IMA = internal maxillary artery; MMA = middle meningeal artery; SSS = superior sagittal sinus; BV = basal vein; AD = aneurysmal dilatation.

Fig. 1. Case 8. Left: Computerized tomography scan showing a large intraparenchymal hemorrhage in the left frontal lobe with mass effect and intraventricular extension. Right: Left common carotid angiogram. The venous aneurysm which was the source of hemorrhage is demonstrated (upper arrow). The two smaller arrows indicate the ophthalmic and anterior ethmoidal arteries. The nidus in the dura is located in the region of the open arrow.

ing the course of the evaluation was found to have an unruptured dural AVM of the anterior fossa. The second patient presented with severe persistent retro-orbital headache, which led to identification of the dural AVM by CT and angiography.

**Radiographic Features**

Computerized tomography was performed in all cases. The CT scan showed an acute intracerebral hemorrhage within the frontal lobe in five patients. The hematoma involved the anteromedial aspect of the frontal pole and, in several cases, extended ventrally to the area overlying the cribriform plate. Mass effect was evident except in Case 3 where the hematoma was small. A remote and unrelated occipital hemorrhage was seen in one patient. Significant subarachnoid hemorrhage was not observed in any of the cases. In the two patients without hemorrhage, a serpentine pattern of enhancement, suggestive of vascular structures, was seen in the anteromedial frontal lobe.

Internal and external carotid angiography was performed in all cases. In all eight patients, the primary source of arterial supply was from the ipsilateral anterior ethmoidal artery, which is a terminal branch of the ophthalmic artery (Fig. 2). Contralateral internal carotid artery injections showed filling of the malformation, apparently through interethmoidal arterial anastomoses. In one patient (Case 3), in whom a portion of the malformation appeared to involve the anterior falk, a tiny frontal branch of the anterior cerebral artery participated in the malformation. External carotid artery supply was seen in five cases (Fig. 3). The internal maxillary artery participated by anastomoses with distal branches of the ophthalmic artery in two cases, and the middle meningeal artery participated in one case. In four cases a branch of the superficial temporal artery penetrated the calvaria and supplied blood flow to the malformation through dural anastomoses.

Venous drainage was primarily into the pial veins, which drained medially to the superior sagittal sinus. Additionally, in two cases subfrontal pial veins drained posteriorly toward the region of the sphenoparietal and cavernous sinuses. In patients with hemorrhage from the dural AVM, cerebral angiography consistently demonstrated an aneurysmal dilatation of the venous channels (venous aneurysm) near the site of the dural-to-pial anastomosis (Fig. 1). Two of the patients with unruptured AVM's had direct connections to the sag-
Operative Procedure

Surgical treatment was carried out in the five patients with hemorrhage and in the one patient with retro-orbital headache. The vascular malformation was approached via a low-frontal craniotomy in each case and, upon exposure of the cortical surface, arterialized cortical veins were identified. In one case, the venous aneurysm ruptured during evacuation of the frontal lobe hematoma resulting in moderately brisk arterial bleeding, which was controlled by clipping the fragile sac. After partial evacuation of the frontal hematoma, retraction of the frontal lobe revealed the vascular connection between the dura in the region of the cribiform plate and the pial vessels on the anterior inferior aspect of the frontal lobe (Fig. 5). Once the vascular connection between the dura and frontal lobe was coagulated and divided, the aneurysm and the arterialized pial veins collapsed. In no case was extensive excision of the dura of the anterior fossa floor required. However, the falx was excised in one case when its anterior portion was involved by the AVM. Entry into the orbit was not required in any case, nor was occlusion of the ophthalmic artery.

Operative Results

All of the patients recovered neurologically after surgery except for Case 1. This patient, who was comatose preoperatively, did not recover consciousness after surgery and ultimately died from a pulmonary embolism. Postoperative angiography confirmed complete obliteration of the AVM, even in the patients in whom the arterialized pial venous system was not excised (Fig. 6).

Discussion

Dural AVM’s comprise 10% to 15% of all intracranial AVM’s. The great majority of these lesions involve the dural wall of the cavernous sinus or the transverse and sigmoid sinuses. Dural AVM’s of the anterior cranial fossa are particularly unusual; only approximately 20 cases have been reported, and they make up less than 10% of the cases in the large reviews. Dural AVM’s are typically fed by meningeal arteries and drain into adjacent dural sinuses. Occasionally, pial veins are recruited when venous hypertension or occlusion of the sinus gives rise to retrograde flow into the cerebral veins, which ordinarily empty into the involved sinus.

Dural AVM’s of the anterior cranial fossa almost universally involve the dura in the region of the cribiform plate and the anterior falx. The principal arterial supply comes from the ipsilateral anterior ethmoidal artery. These malformations may recruit additional supply through anastomoses with the contralateral anterior ethmoidal artery, distal branches of the internal maxillary artery, or middle meningeal branches or from

Fig. 2. Drawing of a typical anterior fossa dural arteriovenous malformation indicating the anterior ethmoidal artery, nidus, and venous aneurysm.
AVM's of the anterior cranial fossa

Fig. 3. Left common carotid angiograms, early arterial phase, in Case 5. **Left:** This shows a branch of the ophthalmic artery (small arrows) feeding the dural arteriovenous malformation (AVM) nidus (large arrow). An aneurysm clip was previously used to treat a contralateral middle cerebral artery aneurysm. **Right:** Distal branches of the internal maxillary artery (lower arrows) have been recruited by the AVM. The supraorbital branch (arrowheads) and a transosseous branch (upper arrows) of the superficial temporal artery also supply the AVM. In this case drainage is through a subfrontal pial vein.

superficial temporal artery branches through transcalvarial anastomoses.

Dural AVM's of the anterior cranial fossa have a distinct pattern of venous drainage. Drainage is primarily into pial veins of the anterior frontal lobe from which they empty into the superior sagittal sinus or posteriorly toward the cavernous sinus. The configuration of these lesions in this way is reminiscent of dural AVM's of the spine. In 85% of reported cases, the venous side of these malformations has involved a large venous aneurysm (or varix). This varix appears to be the source of the hemorrhage which is a common complication of anterior fossa dural AVM's. Eighty percent of previously reported cases and five (63%) of our cases presented with hemorrhage. The hemorrhage was always into the frontal lobe and a venous aneurysm was present in each case. When an aneurysm was not present, the lesion caused nonhemorrhagic symptoms or was an incidental finding. Hemorrhage may be massive and life-threatening, as it was in four of our patients. Dural AVM's in other locations present with hemorrhage less frequently, typically from pial veins which have been recruited for drainage. Approximately 15% of the patients with dural AVM's reviewed by Houser and associates had intracranial hemorrhage (usually subarachnoid hemorrhage from ruptured pial vessels).

In our series and in previously reported cases of anterior fossa dural AVM's, there has been a striking predominance of males. This contrasts with the female predominance found in dural AVM's of the cavernous sinus. The male:female ratio found among anterior fossa dural AVM's is strikingly similar to that for dural AVM's of the spine. There was no history of basal skull fracture or significant trauma in our cases, and this has been rare in other reports. No other predisposing causes have been identified. It is our belief that

Fig. 4. Case 4. Right common carotid angiogram showing an incidentally found anterior fossa arteriovenous malformation. The malformation drains directly into the superior sagittal sinus (large arrows). The nidus is clearly seen distal to the ophthalmic and ethmoidal arteries (small arrows).

Fig. 5. Surgical drawing with the left frontal lobe elevated to reveal the arterialized vascular connection between the dura in the region of the cribiform plate and the pial venous system.
these malformations are congenital, and that symptoms occur only after long-standing hemodynamic stress has caused dilatation then rupture of a draining venous channel.

Surgical treatment is straightforward. The key step is occlusion of the vascular connection between the dura of the cribriform plate area and the pial vessels. With a low frontal craniotomy, this requires minimal frontal lobe retraction. Evacuation of an intracerebral hematoma may be necessary to allow safe retraction of the frontal pole, but care should be taken to avoid rebleeding from the arterialized veins. If the anterior aspect of the falx is involved, this should be excised. Although we excised the venous aneurysm in two cases, this is probably not necessary. With occlusion of the dural-pial connection, the venous side of the malformation will collapse and thrombose. Again, in this respect, a parallel can be drawn between anterior fossa dural AVM's and dural AVM's of the spine.

Surgery is the treatment of choice. Only one patient in our series did not recover after surgery. This patient died from complications related to his failure to recover from the effects of the initial hemorrhage. Previously reported series have shown a very high success rate for complete AVM obliteration, with minimal morbidity. Embolization or other forms of endovascular therapy, given the present state of technical development, appear to be impractical because of the difficulty of catheterizing the ophthalmic artery, and seem to carry an unacceptable risk of visual compromise due to occlusion of retinal branches.

Conclusions

Drawing upon our experience and that described in the previously reported cases, the following recommendations are made:

1. Dural AVM's of the anterior fossa should be included in the differential diagnosis of spontaneous intracerebral hemorrhage involving the anteromedial frontal lobe (particularly in middle-aged males).

2. Surgical treatment should consist of division of the vascular connection between the dura and the pial vessels, a procedure that carries low risk.

3. Surgery is clearly indicated in cases where there has been intracerebral hemorrhage related to the dural AVM.

4. Surgery is indicated in unruptured cases when a venous aneurysm is demonstrated, because of the apparently high risk of hemorrhage.

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AVM's of the anterior cranial fossa


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