Detection of Enlarged Cortical Vein by Magnetic Resonance Imaging Contributes to Early Diagnosis and Better Outcome for Patients With Anterior Cranial Fossa Dural Arteriovenous Fistula

Mohammad A. JAMOUS, Koichi SATOH, Junichiro SATOMI, Shunji MATSUBARA, Norio NAKAJIMA, Masaaki UNO, and Shinji NAGAHIRO

Department of Neurosurgery, School of Medicine, The University of Tokushima, Tokushima

Abstract

Twelve patients (10 men, 2 women) with anterior cranial fossa dural arteriovenous fistula (AVF) were treated at our institute between January 1976 and March 2002. Intracranial hemorrhage was the presenting symptom in six patients. Magnetic resonance (MR) imaging findings identified abnormal cortical veins as flow voids in four of five patients. Angiography was the basis of the diagnosis in all patients. Surgery was the primary treatment in nine patients. The other three patients refused intervention and managed conservatively. Surgical morbidity was negligible and the treatment outcome was highly dependent on the clinical status at presentation. In contrast to the reported high incidence of intracranial hemorrhage in patients with dural AVF in the anterior cranial fossa, only half of our study population presented with hemorrhage. Enlarged cortical veins in the frontobasal area could be detected as flow voids on MR images. This finding contributed to the early diagnosis and treatment of patients treated at our institution for dural AVF in the anterior cranial fossa, and to the better outcomes we obtained in these patients.

Key words: anterior cranial fossa dural arteriovenous fistula, flow void, enlarged cortical vein

Introduction

Dural arteriovenous fistulae (AVFs) are abnormal arteriovenous shunts within the dura mater. The clinical course is usually benign, but depends on the fistula location and the venous drainage pattern. Most patients with dural AVF in the anterior cranial fossa presented with intracranial hemorrhage and had poor outcome. Therefore, dural AVFs located in the anterior cranial fossa are considered to be a distinct type with specific angiographic characteristics and aggressive clinical behavior.

We present our experience with 12 patients with dural AVF in the anterior cranial fossa, discuss the clinical and diagnostic features, and stress the value of early diagnosis and treatment of this rare lesion.

Materials and Methods

Between January 1976 and March 2002, 12 patients, 10 males and two females aged from 47 to 71 years (mean 60 years), were treated for dural AVF in the anterior cranial fossa at our institute. The diagnosis was based on angiography in all 12 patients. A summary of the symptoms is presented in Table 1. All patients underwent computed tomography (CT) and five patients underwent magnetic resonance (MR) imaging. CT found no abnormalities in six of the 12 patients, and intracranial hemorrhage in six patients (Table 2). MR imaging detected abnormal cortical flow voids in the frontobasal area in four of five patients. Angiography showed evidence of early filling of the frontal cortical vein draining into the superior sagittal sinus in 11 of 12 patients. The Rosenthal vein was involved in two patients and the sylvian vein in one patient. Six patients had venous lakes. The main feeder was the anterior ethmoidal artery in eight patients. Other feeders included the posterior ethmoidal artery, the inferior lateral trunk of the internal carotid artery,
Table 1  Clinical data

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age/Sex</th>
<th>Presentation</th>
<th>Intracranial hemorrhage</th>
<th>Treatment</th>
<th>Outcome</th>
<th>Follow-up period (year)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>69/M</td>
<td>loss of consciousness</td>
<td>+</td>
<td>microsurgery</td>
<td>SD</td>
<td>&gt;10</td>
</tr>
<tr>
<td>2</td>
<td>52/M</td>
<td>headache, confusion</td>
<td>+</td>
<td>microsurgery</td>
<td>MD</td>
<td>&gt;10</td>
</tr>
<tr>
<td>3</td>
<td>47/M</td>
<td>headache, confusion</td>
<td>+</td>
<td>conservative</td>
<td>GR</td>
<td>10</td>
</tr>
<tr>
<td>4</td>
<td>66/M</td>
<td>headache, confusion</td>
<td>+</td>
<td>microsurgery</td>
<td>MD</td>
<td>&gt;10</td>
</tr>
<tr>
<td>5</td>
<td>55/F</td>
<td>loss of consciousness</td>
<td>+</td>
<td>microsurgery</td>
<td>MD</td>
<td>9</td>
</tr>
<tr>
<td>6</td>
<td>58/M</td>
<td>loss of consciousness</td>
<td>+</td>
<td>microsurgery</td>
<td>SD</td>
<td>1</td>
</tr>
<tr>
<td>7</td>
<td>63/M</td>
<td>visual symptoms</td>
<td>-</td>
<td>TAE and microsurgery</td>
<td>GR</td>
<td>8</td>
</tr>
<tr>
<td>8</td>
<td>64/M</td>
<td>dizziness</td>
<td>-</td>
<td>microsurgery</td>
<td>GR</td>
<td>5</td>
</tr>
<tr>
<td>9</td>
<td>48/M</td>
<td>dizziness</td>
<td>-</td>
<td>TAE and microsurgery</td>
<td>GR</td>
<td>7</td>
</tr>
<tr>
<td>10</td>
<td>71/M</td>
<td>incidental</td>
<td>-</td>
<td>conservative</td>
<td>GR</td>
<td>2</td>
</tr>
<tr>
<td>11</td>
<td>63/F</td>
<td>visual symptoms</td>
<td>-</td>
<td>TAE and microsurgery</td>
<td>GR</td>
<td>4</td>
</tr>
<tr>
<td>12</td>
<td>60/M</td>
<td>dizziness</td>
<td>-</td>
<td>conservative</td>
<td>GR</td>
<td>3</td>
</tr>
</tbody>
</table>

GR: good recovery, MD: moderate disability, SD: severe disability, TAE: transarterial embolization.

Table 2  Radiological findings

<table>
<thead>
<tr>
<th>Case No.</th>
<th>CT findings</th>
<th>MR imaging findings</th>
<th>Feeding arteries</th>
<th>Draining veins</th>
<th>Venous lake</th>
<th>Arteriovenous shunt*</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>ICH, SDH</td>
<td>n.d.</td>
<td>AEA</td>
<td>cortical vein</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>2</td>
<td>ICH</td>
<td>n.d.</td>
<td>AEA, PEA</td>
<td>cortical vein</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td>3</td>
<td>ICH, SAH</td>
<td>n.d.</td>
<td>PEA</td>
<td>cortical vein</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td>4</td>
<td>SAH</td>
<td>n.d.</td>
<td>ILT</td>
<td>sylvian vein</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>5</td>
<td>ICH, IVH</td>
<td>n.d.</td>
<td>IMA</td>
<td>cortical vein</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td>6</td>
<td>SDH</td>
<td>n.d.</td>
<td>AEA, PEA</td>
<td>cortical vein</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>7</td>
<td>normal</td>
<td>flow void of cortical vein</td>
<td>AEA, IMA</td>
<td>cortical vein, Rosenthal vein</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td>8</td>
<td>normal</td>
<td>n.d.</td>
<td>AEA</td>
<td>cortical vein</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>9</td>
<td>normal</td>
<td>flow void of venous lake</td>
<td>AEA, PEA</td>
<td>cortical vein</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>10</td>
<td>normal</td>
<td>normal</td>
<td>AEA</td>
<td>cortical vein</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td>11</td>
<td>normal</td>
<td>flow void of cortical vein</td>
<td>MMA</td>
<td>cortical vein, Rosenthal vein</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>12</td>
<td>normal</td>
<td>flow void of cortical vein</td>
<td>AEA</td>
<td>cortical vein</td>
<td>-</td>
<td>+</td>
</tr>
</tbody>
</table>


the internal maxillary artery, and the middle meningeal artery.

Three patients refused surgical intervention and were managed conservatively. One of these patients presented with headache, and CT revealed a small right frontal intracerebral hematoma. The other two patients showed no evidence of hemorrhage. Surgical obliteration of the fistula was performed in nine patients. Low frontal craniotomy was performed, and the shunt site between the dural arteries in the region of the cribriform plate and the pial cortical veins in the anterior-inferior aspect of the frontal lobe was identified, coagulated, and divided. Following division of the shunt site, the dilated cortical vein collapsed. Three patients underwent preoperative transarterial embolization using 5-mm fibered platinum coils (Target Therapeutics, Fremont, Calif., U.S.A.) to reduce the blood flow through the arterialized vein.

The clinical outcome was correlated with the patient’s status at presentation and with the degree of brain damage resulting from intracranial hemorrhage. Early diagnosis based on abnormal MR imaging findings was associated with good recovery after surgical obliteration of the lesion, but most patients who presented with intracranial hemorrhage had disabilities.
**ILLUSTRATIVE CASE 6:** A 58-year-old male presented with sudden loss of consciousness. Brain CT revealed a left frontotemporal subdural hematoma with midline shift (Fig. 1A, B). Despite emergency decompressive craniectomy and drainage of the hematoma, his postoperative recovery was poor. Digital subtraction angiography showed a dural AVF in the anterior cranial fossa supplied by the left anterior and posterior ethmoidal arteries and draining into the left ascending frontal cortical vein with venous varix (Fig. 1C, D). The dural AVF was ligated and the shunt site in the region of the cribriform plate was divided, resulting in complete obliteration of the fistula. However, the patient's response was slow and he had a poor outcome.

**ILLUSTRATIVE CASE 9:** A 48-year-old male complained of nausea and dizziness. MR imaging revealed loss of flow void in the left frontobasal area (Fig. 2A), indicative of vascular pathology. Cerebral angiography revealed a dural AVF in the anterior cranial fossa supplied by the left anterior and posterior ethmoidal arteries and draining into the left frontal ascending cortical vein, with a venous lake (Fig. 2B, C). After transarterial embolization of the distal left ophthalmic artery, he underwent surgery using the left basal interhemispheric approach and the draining vein was ligated, resulting in complete obliteration of the fistula (Fig. 2D). His postoperative course was uneventful except for left anosmia.

**Discussion**

Intracranial hemorrhage was the presenting symptom in more than 80% of reported cases of dural
AVF in the anterior cranial fossa, and was usually associated with morbidity and mortality, even after successful surgical obliteration of the shunting point.5-10,13,14,16,17 This outcome looks similar to the outcome of patients with cerebral aneurysm following subarachnoid hemorrhage. Preventive clipping of the unruptured aneurysm is the best way to manage such patients.

The high rate of intracranial hemorrhage in patients with dural AVF in the anterior cranial fossa is attributed to the high venous pressure and dilation of the draining cortical veins.2,4,12,13 The absence of warning symptoms that mandates radiological examination may also contribute to the high prevalence of intracranial hemorrhage in patients with this lesion.19

Early diagnosis of dural AVF is very helpful to avoid progression and subsequent rupture of the fistula. We suggest that dural AVF in certain locations will cause symptoms that allow early diagnosis. However, dural AVF located in clinically silent areas, such as the anterior cranial fossa, usually manifests as non-specific symptoms and the lesion may not be detected before progression to intracranial hemorrhage.3,4,6,18-21

Enlarged cortical draining veins were present in the frontobasal area in most patients with dural AVF in the anterior cranial fossa, and could be seen as flow voids on T1- and T2-weighted MR imaging due to the rapid blood flow in the enlarged veins. Demonstration of the actual fistula is extremely difficult with spin-echo MR imaging, but the appearance of dilated cortical vein without parenchymal nidus is suggestive of dural AVF.5 Venous pouching and parenchymal edema, which indicate venous hypertension and are considered risk factors for venous rupture, can also be detected by MR imaging.24 MR imaging is useful for detecting clinically silent or incidental abnormalities, so is now performed in many patients with minor neurological symptoms, such as non-specific headache, dizziness, blurred vision, and behavioral changes.11,22,23 MR imaging was useful for the early diagnosis in four patients treated at our institution for dural AVF in the anterior cranial fossa.

Early detection and treatment of dural AVF in the anterior cranial fossa clearly improves patient outcome, as seen in the two illustrative cases presented here. Case 6 represents the classical course of patients presenting with intracranial hemorrhage caused by dural AVF in the anterior cranial fossa, whereas early detection and treatment of the lesion in Case 9 prevented the unexpected consequences of intracranial hemorrhage.

All of our patients treated before 1990 (Cases 1-4) manifested intracranial hemorrhage, whereas only two of our eight patients treated after 1990 (introduction of MR imaging) had intracranial hemorrhage. Abnormal flow void on MR imaging was helpful for the early diagnosis in four of the eight patients treated after 1990. Early treatment resulted in better outcome for the patients who underwent MR imaging.

Cerebral angiography is essential to establish the diagnosis of dural AVF in the anterior cranial fossa. Early filling of the frontal cortical vein that drains into the superior sagittal sinus is a common finding. The most common feeder is the anterior ethmoidal artery, which is a branch of the ophthalmic artery. Other feeders include the middle meningeal artery, the internal maxillary artery, the superficial temporal artery, and the posterior ethmoidal artery. The presence of a venous lake is reportedly associated with a high incidence of bleeding.2,13 However, although a venous lake is indicative of high venous pressure, it is not essential for the development of intracranial hemorrhage. In our series, half of the patients with hemorrhage had a venous lake.

Interventional embolization and radiosurgery have been used successfully to treat dural AVF in the anterior cranial fossa.3,4,6 However, long-term results are not available. The major limitation of radiosurgery is the inherent delay of 1-2 years between irradiation and thrombosis or fibrosis. During this period the patient remains at risk for hemorrhage.

Endovascular techniques are most useful for moderate sized AVMs, with a predominantly external carotid artery supply and preferably no pial drainage. Small, high-flow AVFs with pial drainage, like dural AVF in the anterior cranial fossa, carry the risk of embolizing material passing to the draining veins, resulting in venous hypertension and possibly hemorrhage. Transophthalmic artery embolization is needed for curative endovascular treatment, but this is a risky procedure, and may be complicated by visual loss due to migration of the embolizing material into the central retinal artery.

Surgery is the treatment of choice for dural AVF in the anterior cranial fossa.7,8,12,16 The target is to obliterate the shunt site and drain existing hematoma. We performed low frontal craniotomy and, following hematoma evacuation, explored for a fistula at the cribriform plate. Dividing the vascular connection between the dura at the site of the cribriform plate and the frontal lobe usually resulted in the collapse of the arterialized vein. Surgery carries a very low risk and the success rates are high. In our series, complete disappearance of the malformation was obtained in all patients. However,
the prognosis was highly dependent on the extent of brain damage at presentation.

Our experience with 12 patients with dural AVF in the anterior cranial fossa clearly demonstrates the usefulness of MR imaging for early diagnosis and better prognosis. The clinical behavior of this rare vascular lesion is unknown and surgical obliteration carries very low risks, so we recommend surgical intervention in patients with incidental findings of dural AVF in the anterior cranial fossa.

References

11) Katzman GL, Dagher AP, Patronas NJ: Incidental findings on brain magnetic resonance imaging from 1000 asymptomatic volunteers. JAMA 282: 36–39, 1999

Address reprint requests to: M. Jamous, M.D., Department of Neurosurgery, School of Medicine, The University of Tokushima, 3–18–15 Kuramoto-cho, Tokushima, Tokushima 770-8503, Japan.
e-mail: neuros@clin.med.tokushima-u.ac.jp

Commentary

The natural history of dural arteriovenous fistulae is dependent upon the pattern of venous drainage. Those lesions draining solely into a dural sinus are associated with a relatively benign natural history and rarely present with progressive neurological deficit or hemorrhage. Alternatively, dural arteriovenous fistulae which have leptomeningeal venous drainage

Neurol Med Chir (Tokyo) 44, October, 2004
are associated with a very aggressive natural history. As pointed out by the authors, dural fistulae of the anterior cranial fossa are almost always associated with leptomeningeal venous drainage and are therefore aggressive in nature, most often presenting with intracerebral hemorrhage. I would agree with the authors that the ultimate outcome of the patient following treatment is most dependent upon the condition of the patient at the time of presentation. Therefore, the patient whose fistula is discovered on imaging studies before hemorrhage may be treated by surgical obliteration of the fistula with minimal risk and elimination of the risk of future hemorrhage. Our experience has mirrored that of the authors. In recent years more anterior fossa dural fistulae have been discovered on MRI performed in patients with vague or unrelated symptoms and provided an opportunity for curative treatment.

I also agree with the authors that the best treatment for anterior fossa dural fistulae is surgical obliteration. The majority of dural arteriovenous malformations at other sites are amenable to endovascular treatment, particularly transvenous embolization. Anterior fossa dural fistulae are very straightforward to treat surgically. The surgeon only has to disconnect the vein draining immediately from the anterior fossa with no treatment required for the arterial supply. Endovascular treatment, on the other hand, is quite challenging for these lesions. A transvenous approach, which would be more likely to result in complete obliteration, requires lengthy catheterization through tortuous veins to reach the anterior fossa. Transarterial embolization is rarely curative unless a catheter is "wedged" into a feeding artery to allow endovascular glue to be pushed through the fistula into the proximal portion of venous drainage. If the glue polymerizes proximal to the fistula, it will remain open and recruit new arterial supply, and if glue passes too far beyond the fistula, the patient may experience venous hypertension.

In this series, three patients underwent preoperative embolization before surgical obliteration. I question the value of preoperative embolization in these cases. As mentioned earlier, the surgical treatment is very straightforward and involves simply disconnecting the venous drainage from the site of the fistula. Preoperative transarterial embolization "to reduce the blood flow to the arterialized vein" is of questionable value and likely does not justify the need for two procedures on the patient.

The authors are to be commended for increasing the awareness of this entity and reminding neurosurgeons of the aggressive natural history of these lesions as well as their accessibility to curative surgical management.

Daniel L. BARROW, M.D.
Department of Neurosurgery
The Emory Clinic
Atlanta, Georgia, U.S.A.

The authors demonstrated their experience for dural AVF which is located mainly in the anterior cranial fossa. The authors also suggested that asymptomatic frontal dural AVF should be excised due to morbidity or mortality which are associated with bleeding. Enlarged cortical vein in the frontobasal area could be seen as signal void on MR images and this might be helpful for early diagnosis and operation.

It is suggested that transcranial Doppler sonography (TCD) is useful for detection of small or even occult intracranial vascular malformation or AVF. Although many dural AVFs have a cortical drain vein large enough to be seen on MR imaging, some lesions may not appear on MR without dilated cortical vein. ICA or ECA can be assessed easily by TCD, and with flow change, vascular malformation or AVF can be detected before development of dilated cortical vein. TCD is cheaper than MR image. So, if dural AVF is suspected without dilated cortical vein seen on MR, then TCD can be helpful for earlier diagnosis.

In the illustrative cases, the goal of most surgery was obliteration of AVF at the fistula site. This is simple and applicable to frontobasal AVF, because venous drainage around this area is not dependent on the fistula site. However, it is not always easy to find the accurate fistula site in the operation field, due to similar appearance and consistency between artery and vein. When normal venous drainage is compromised by fistula occlusion, catastrophic venous infarction or brain swelling might ensue. This is not common in a frontobasal location, but careful angiographic analysis is necessary when AVF is located at another site. Sometimes, it is necessary to perform provocation test by endovascular procedure to predict the effect of operation and neurological change.

As the authors suggested, there is no need to excise dilated cortical vein or other abnormal appearing vein on the brain cortex. These will disappear after fistula obliteration and, moreover, this vein may contribute to normal venous drainage.

There is also controversy for operation for all detected asymptomatic lesions, because overall hemorrhage rate for dural AVF is only 1.5%/yr. Frontal craniotomy and fistula obliteration is a relatively simple operation, but this operation might not be simple for medically unfit patients. Careful selection of candidates and thorough analysis of angiography are necessary before operation.

References


Dae Hee HAN, M.D.
Department of Neurosurgery
College of Medicine
Seoul National University
Seoul, R.O.K.

Neurol Med Chir (Tokyo) 44, October, 2004