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# MR Imaging of Dural AV Fistulas at the Cavernous Sinus

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**Abstract:** The magnetic resonance appearance of dural arteriovenous fistulas (AVFs) at the cavernous sinus (CS) was studied in six angiographically verified cases. Magnetic resonance clearly demonstrated shunted blood as an area of signal void both in the CS and in the superior ophthalmic vein. The relationship between shunted blood, internal carotid artery, and extraocular nerves, as well as proptosis, enlargement of the extraocular muscles, and bulging of the lateral wall of the CS were also depicted in the images. Normal venous flow in the involved CS was shown as a low signal area that enhanced after gadolinium administration. Magnetic resonance is useful for screening and follow-up examinations of dural AVFs at the CS. It is essentially a noninvasive procedure that may be repeated and obviates the need for follow-up angiography. However, it should be noted that a signal void in the CS sometimes represents normal venous flow. A definite diagnosis should rely on angiography, which is essential for therapeutic planning. **Index Terms:** Fistulas, arteriovenous—Brain, blood flow—Vascular system, abnormalities—Magnetic resonance imaging—Angiography.

Dural arteriovenous fistulas (AVFs) at the cavernous sinus (CS) represent abnormal connections between the dural branches of the internal and/or external carotid arteries and the CS (1). They commonly occur in elderly women presenting with proptosis, conjunctival injection, chemosis, retroorbital pain, bruit, ophthalmoplegia, diminished visual acuity, and increased intraocular pressure (1–4). Spontaneous cure of AVFs is not uncommon, but some necessitate interventional procedures (3–7). Definite diagnosis so far has depended on angiography, which is also essential for therapeutic planning.

The usefulness of magnetic resonance (MR) for the study of the CS and its pathology has been previously discussed (8,9). To evaluate the usefulness of MR for the management of dural AVFs at the CS, we retrospectively reviewed six cases of this pathological entity.

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## MATERIALS AND METHODS

The patients' age, gender, involved side, arterial supply, venous drainage, and clinical symptoms are summarized in Tables 1 and 2. Our patients were six women, 49–71 years, with a mean age of 65 years. Clinical symptoms were predominantly localized to one eye (right eye, four cases; left eye, two cases). The common symptoms were oculomotor nerve palsy (two cases), abducent nerve palsy (five cases), objective bruit (four cases), proptosis (six cases), chemosis (five cases), conjunctival injection (five cases), retroorbital pain (one case), and decreased visual acuity (four cases).

Five millimeter thick axial and coronal MR images were obtained using a 0.5 T superconductive magnet (Picker International, Cleveland, OH, U.S.A.) with a T1-weighted spin-echo (SE) pulse sequence (600 ms repetition time, 40 ms echo time, one average, 256 × 256 matrix). T2-weighted SE images or gradient echo images were usually not used due to their poor anatomical delineation of the CS. Gadolinium diethylenetriamine pentaacetic acid (Gd-DTPA) was administered intravenously at a dosage of 0.1 mmol/kg body weight in four cases (Cases 1–4). Correlative angiographic and CT studies were also reviewed.

TABLE 1. *Case summary*

| Case no./ sex/age (years) | Side | Arterial supply | Drainer |
|---|---|---|---|
| 1/F/49 | R | R-IC,EC | R-SOV |
| 2/F/57 | L | L-IC,EC (R-IC,EC) | L-SOV |
| 3/F/71 | R | R-IC,EC (L-IC,EC) | Cortical |
| 4/F/71 | R | R-IC,EC (L-IC) | R-SOV |
| 5/F/71 | L | L-IC,EC | L-SOV,IOV L-IPS |
| 6/F/69 | R | R-IC,EC (L-IC,EC) | R-IPS,BP L-IPS |

BP, basilar plexus; EC, external carotid system; IC, internal carotid system; IOV, inferior ophthalmic vein; IPS, inferior petrosal sinus; SOV, superior ophthalmic vein; parentheses, less contributory than above.

## RESULTS

Feeding arteries, abnormal draining pathways of shunted blood, normal venous pathway in the CS, bulging of the lateral wall of the CS, proptosis, and enlargement of the extraocular muscles were evaluated separately.

### Feeding Arteries

According to the Barrow classification (2), all six cases belonged to type D, which means both the external and internal carotid arteries were contributory to the AVFs. Feeding arteries were detected solely by angiography (Figs. 1 and 2). In this series the artery of the foramen rotundum, the middle meningeal artery, the accessory meningeal artery, and the ascending pharyngeal artery were the main contributory arteries in the external carotid system, whereas the inferolateral trunk and the meningohypophyseal trunk were the contributory arteries in the internal carotid system. Even with knowledge of the angioarchitecture as demonstrated by angiography, it was impossible to depict the feeding arteries by either CT or MR in any of the cases.

### Draining Pathways

Postcontrast CT demonstrated the superior ophthalmic veins (SOVs) of the involved eye in four cases (all cases in which the SOVs were the main draining pathways) as tortuous, dilated high density areas. With MR, the SOVs were demonstrated as areas of signal void in these four cases (Fig. 1c–e). In Case 5 the inferior ophthalmic vein was also demonstrated as signal void. In two cases (Cases 3 and 6) that had no SOV involvement, the SOV was not demonstrated and only a modest proptosis was shown. In Case 6 the inferior petrosal sinus was demonstrated as an area of signal void by MR (Fig. 2).

Computed tomography could not demonstrate the draining pathways in the CS because the contrast medium enhanced the whole CS in addition to the internal carotid artery (ICA) in all cases. On the MR images the draining pathways in the CS were shown in all cases as areas of low signal intensity, usually slightly more intense than the ICA (Figs. 1f and 2c and e). In Case 6, in which the patient experienced oculomotor palsy, partial abducent palsy, and retroocular pain, the draining pathway was located just superolateral to the ICA (Fig. 2c). In Cases 1–4, which presented with abducent nerve palsy, chemosis, and conjunctival injection and none or minimal oculomotor nerve palsy, this low intensity area was located lateral or inferolateral to the ICA (Fig. 1f). In Case 5, in which the patient had full extraocular movement, this area was medial to the ICA. These draining pathways were better depicted on the coronal than on the sagittal images.

No MR evidence of thrombosis in the CS or in the SOV was noted in this series.

### Normal Venous Pathways in the CS

Only MR was able to demonstrate the normal venous pathways in the CS, abutting the abnormal venous draining of the shunted blood. These normal venous pathways were heterogeneous in intensity but generally had a low signal intensity slightly higher than the intensity of the abnormal shunted blood. These normal pathways were markedly en-

TABLE 2. *Summary of clinical signs*

| Case no. | 3rd N | 6th N | Bruit | Proptosis | Chemosis | CI | Pain | DV |
|---|---|---|---|---|---|---|---|---|
| 1 | − | + | + | + | + | + | − | (+) |
| 2 | − | + | + | + | + | + | − | (+) |
| 3 | − | + | − | (+) | (+) | + | − | (+) |
| 4 | (+) | + | + | + | + | (+) | − | (+) |
| 5 | − | − | − | + | (+) | (+) | − | − |
| 6 | + | (+) | − | (+) | − | − | + | − |

3rd N, oculomotor nerve palsy; 6th N, abducent nerve palsy; CI, conjunctival injection; DV, decreased vision; parentheses, not prominent.

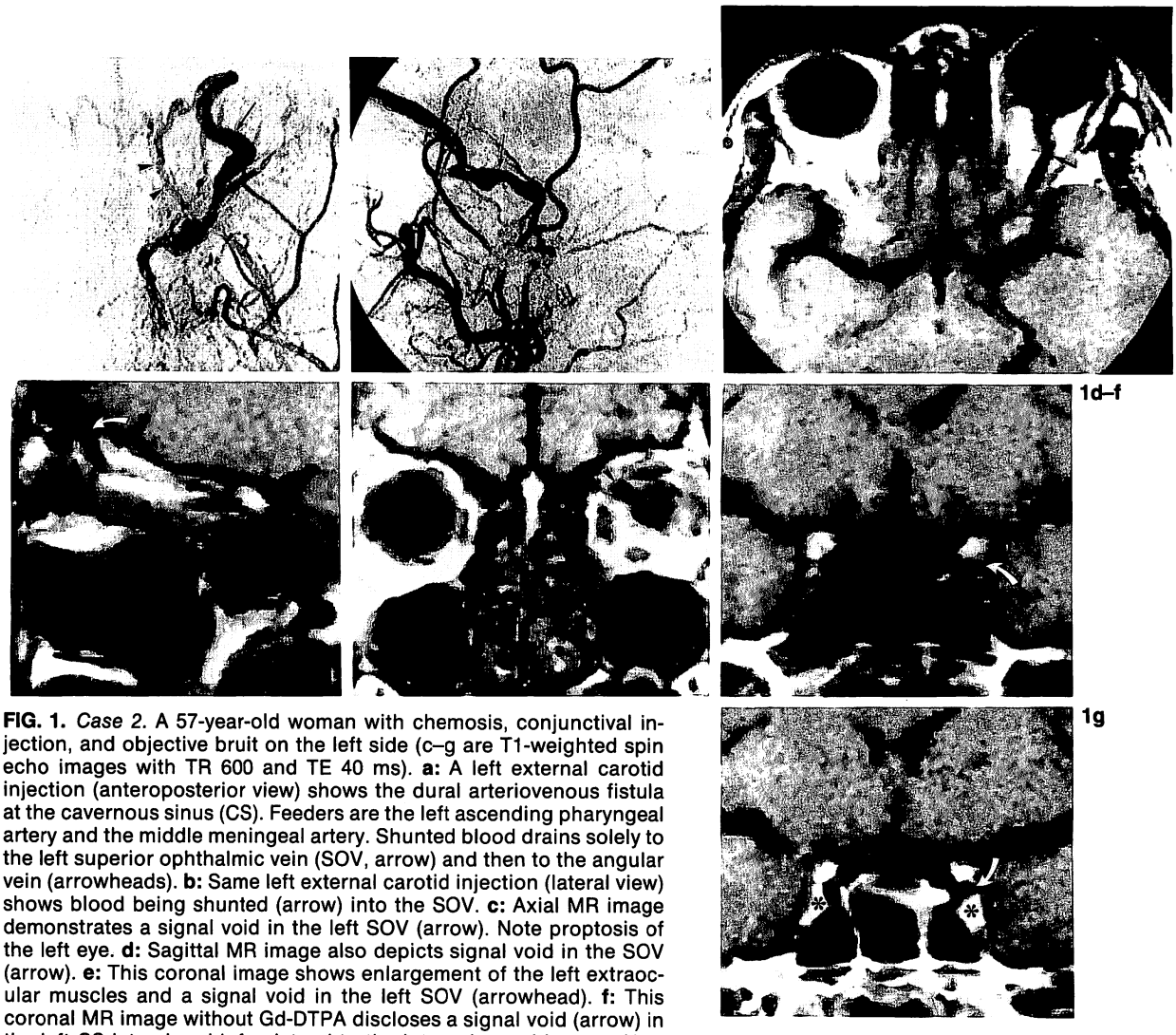


**FIG. 1.** *Case 2.* A 57-year-old woman with chemosis, conjunctival injection, and objective bruit on the left side (c–g are T1-weighted spin echo images with TR 600 and TE 40 ms). **a:** A left external carotid injection (anteroposterior view) shows the dural arteriovenous fistula at the cavernous sinus (CS). Feeders are the left ascending pharyngeal artery and the middle meningeal artery. Shunted blood drains solely to the left superior ophthalmic vein (SOV, arrow) and then to the angular vein (arrowheads). **b:** Same left external carotid injection (lateral view) shows blood being shunted (arrow) into the SOV. **c:** Axial MR image demonstrates a signal void in the left SOV (arrow). Note proptosis of the left eye. **d:** Sagittal MR image also depicts signal void in the SOV (arrow). **e:** This coronal image shows enlargement of the left extraocular muscles and a signal void in the left SOV (arrowhead). **f:** This coronal MR image without Gd-DTPA discloses a signal void (arrow) in the left CS lateral and inferolateral to the internal carotid artery. Normal venous flow in the CS has low intensity. Note mild bulging of the left lateral wall of the CS. The bilateral high intensity areas at the superior portion of the CSs are due to bone marrow in the anterior clinoid processes. **g:** Gd-DTPA enhanced image on the same coronal plane as (f) shows the same signal void (arrow) in the left CS. The normal venous spaces of the CSs are markedly enhanced (asterisks).

hanced by Gd-DTPA, allowing easy recognition of the venous compartments (Fig. 1f and g).

### Bulging of the Lateral Wall of the CS

In all cases, bulging of the lateral wall of the CS on the side of the prominent clinical signs was demonstrated to some extent by MR and postcontrast CT. The coronal MR images were superior to the axial images because they allowed easier comparison of the lateral walls on both sides (Fig. 2c).

### Proptosis and Enlargement of the Extraocular Muscles

Proptosis and enlargement of the extraocular muscles were demonstrated by plain CT and MR in all cases. However, MR was superior to CT for demonstration of the extraocular muscles due to its higher resolution and multiplanar capability (Fig. 1e).

## DISCUSSION

Magnetic resonance has been reported as a useful technique for studying the CS (8,9). Understanding the signal intensity of blood flow in the CS is not straightforward due to the complex anatomy and flow features present in this area (10). Although arterial flow in the intracavernous ICA yields no signal due to "high velocity signal loss" (11), venous flow in the CS is characterized by some signal intensity. Factors that influence MR signal intensity

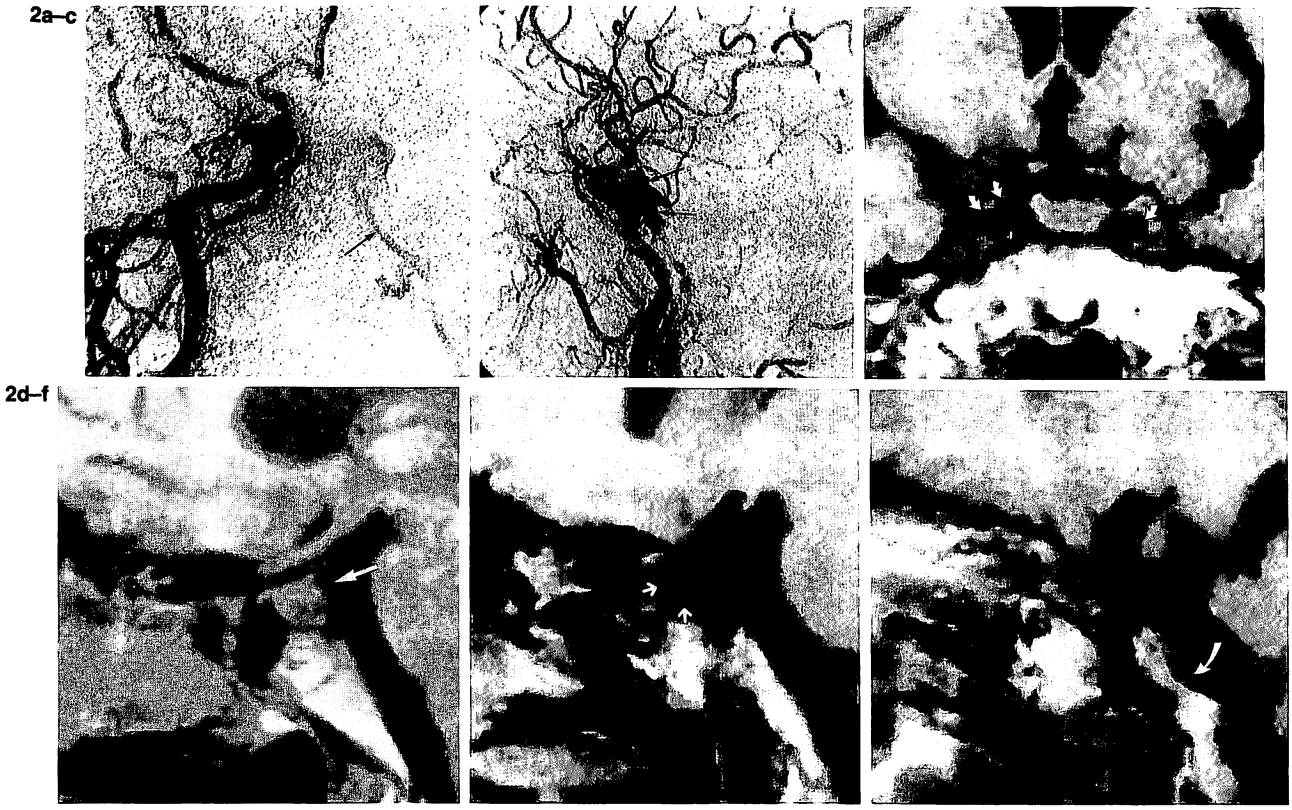


**FIG. 2.** *Case 6.* A 69-year-old woman with right retroorbital pain and oculomotor and abducent nerve palsy (c–f are T1-weighted spin echo images with TR 600 and TE 40 ms). **a:** This right common carotid injection (anteroposterior view) demonstrates the dural arteriovenous fistula at the cavernous sinus (CS). Feeders are the artery of the foramen rotundum, the middle meningeal artery, the ascending pharyngeal artery, and the meningohypophyseal trunk. Shunted blood drains into both inferior petrosal sinuses (arrows) and into the basilar plexus. **b:** Lateral view of same arteriogram shows shunted blood draining into the basilar plexus (arrows) and both inferior petrosal sinuses. **c:** Coronal image demonstrates signal void (arrows) around both intracavernous carotid arteries. **d–f:** These sagittal images are contiguous from the midline to the right side. d: Arrow indicates signal void in the basilar plexus. e: Arrows indicate right internal carotid artery, surrounded by signal void due to shunted blood in the CS. f: Arrow points to signal void in the right inferior petrosal sinus.

include flow velocity, flow direction, flow distribution, turbulence, the anatomy of the cavernous sinus, as well as the MR scanning parameters. In the normal CS a high flow component shows up as no signal or as a signal of very low intensity on T1-weighted SE images. In contrast, stagnant or very slow flow appears as modest or high signal intensity, probably due to "flow-related enhancement" (11). Thus, the signal intensity of the venous pathways in the CS is heterogeneous (10). It is essential to be familiar with these signal characteristics when interpreting pathological states of the CS.

Dural AVFs at the CS are abnormal fistulous connections between the dural branches of the internal and/or external carotid arteries and the CS (1). Drainage takes place mostly through the superior and/or inferior ophthalmic veins in the anterior direction, the inferior and/or superior petrosal sinuses in the posterior direction, and the intercavernous veins and the basilar plexus medially, toward the opposite CS and the pterygoid plexus in the inferior direction. Sometimes drainage is directed toward the cortical veins (Case 3). The CS may be either unilaterally or bilaterally involved.

Magnetic resonance clearly shows shunted flow in the CS and the SOV as no signal intensity due to "high velocity signal loss." Interestingly, shunted blood in the CS can coexist with slow or stagnating blood flow, which MR depicts as an area of low signal intensity (slightly more intense than shunted blood), which enhances with Gd-DTPA. One should keep in mind that high venous flow even in a normal CS appears as an area of low or negligible signal intensity (10). This phenomenon is commonly observed at the level of the superior sagittal and transverse sinuses. Thus, a high intensity area in the CS on T1-weighted SE images implies slow or stagnant blood flow, recent thrombosis, or fat. Conversely, no signal implies high and/or turbulent flow. If loss of signal in the CS is seen, the differ-

ential diagnosis must include a dural AVF, a pial arteriovenous malformation with drainage to the CS, and aneurysm (12). However, it should be noted that normal venous flow (rapid component) in the CS can also create signal loss (10).

From an anatomical point of view, the oculomotor nerve in the CS is located superolateral to the intracavernous ICA, and the abducent nerve is lateral to the ICA (13). In this series, oculomotor nerve palsy with partial abducent palsy was noted in Case 6 and abducent nerve palsy was present in Cases 1–4. In the coronal images the signal void in the CS was located near the paralyzed nerves, with a lateral or inferolateral location for the abducent nerve palsy and a superolateral location for the oculomotor nerve palsy. The reason for nerve palsy is unknown, but the MR observations suggest that a mechanical compression of the nerve by shunted blood is more likely to be responsible for the palsy than nerve ischemia due to increased venous pressure.

The etiology of dural AVFs at the CS is not well understood. Rupture of the small dural branch that travels through the CS has been considered the possible (1). On the other hand, the angiographical patterns of abnormal venous drainage, filling defects within the CS and its tributaries, the abnormal shape of the CS, and venous stasis indirectly suggest occurrence within the CS (14). Magnetic resonance demonstrates venous thrombosis as a high intensity area on both T1- and T2-weighted SE images (15). Sergott et al. (16) have reported dural AVFs with thrombosis formation shown by MR in the SOV but not in the CS. Since no thrombosis formation in the CS was detected by MR in our series, we think that further studies are necessary to determine the role of thrombosis formation in the CS as a possible cause of dural AVF.

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