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Case Report

Transvenous Embolization of a Dural Arteriovenous Fistula of the Anterior Cranial Fossa: Preliminary Results

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Summary: We report two cases of a dural arteriovenous fistula of the anterior cranial fossa, one causing subarachnoid hemorrhage and one detected accidentally. The first case was incompletely treated by neurosurgery, and the second one was referred for endovascular therapy. Both fistulas were successfully occluded by transvenous embolization by using electrolytically detachable coils.

Intracranial (ie, subarachnoid, intracerebral, or subdural) hemorrhage represents approximately 65% of the clinical spectrum of dural arteriovenous fistulas (AVFs) of the anterior cranial fossa. These fistulas are therefore unanimously considered to be aggressive and call for radical curative treatment. The transfrontal or interhemispheric approach with disconnection of the arterial and venous structures at the fistula site has been very successful, but carries the risks inherent to frontal craniotomy.

We report two cases of dural AVFs of the anterior cranial fossa that were successfully occluded by transvenous embolization of the draining fistula vein with Guglielmi detachable coils (GDCs).

Case Reports

Case 1

A 40-year-old man consulted a neurologist for a persisting headache of 3 weeks' duration. Clinical examination revealed mild neck stiffness without neurologic deficit. There was no fever, and heart rate and blood pressure were within normal limits. Fundoscopy revealed a small choroidal hemorrhage in the right eye. Findings on cranial CT scans without and with contrast medium were normal. As clinical symptoms were suggestive of recent subarachnoid hemorrhage, a lumbar puncture was done. Liquor analysis showed siderophores pointing to a passed subarachnoid hemorrhage. Cerebral digital subtraction angiography (DSA) did not reveal an aneurysm; however, a dural AVF of the anterior cranial fossa was found (Fig 1A).

The neurosurgeons chose a transorbital approach to attack the distinct feeding anterior ethmoidal artery, which was electrocoagulated at its entry in the ethmoid bone. On postoperative control DSA, however, the dural AVF was reinjected by small collaterals branching from the proximal ipsilateral anterior ethmoidal artery and from the ipsilateral sphenopalatine artery (Fig 1B). The patient declined a frontal surgical approach, and he was referred for endovascular therapy.

With the patient under general anesthesia, transarterial embolization of the sphenopalatine artery was attempted but abandoned because of arterial spasm. Then, a transvenous route was decided upon. A catheter was inserted in the right jugular vein transfemorally and a Tracker-38 (Target Therapeutics, Fremont, CA) was placed into the transverse dural sinus. Coaxially, a stem-shaped 2-marker Fast-Tracker-10 (Target) was pushed up over the guidewire (Seeker Lite 10, Target) to the frontal part of the superior sagittal sinus. The 155-cm-long microcatheter, however, was some 2 cm too short to reach the dural AVF. The technical problem was solved by direct retrograde puncture of the right internal jugular vein. The same coaxial Tracker-38 and Fast-Tracker-10 system were introduced for retrograde catheterization of the superior sagittal sinus. The microcatheter was gently pushed into the draining frontopolar vein. Once the tip of the microcatheter reached the last curve, some 5 mm from the fistula point, embolization was performed with two soft GDC-10 2-mm-diameter 2-cm-long coils and one soft GDC-10 2-mm-diameter 3-cm-long coil (Target) (Fig 1C). Control DSA of the right and left internal and external carotid arteries showed immediate occlusion of the dural AVF. The patient was discharged 1 day later in perfect condition. One month later, he consulted again for a persistent headache. Findings on MR images of the brain were normal. Bilateral internal and external carotid DSA was repeated, but no abnormalities were apparent (Fig 1D). The dural AVF of the anterior fossa was still obliterated.

Case 2

A 39-year-old man presented with complaints compatible with a recurrent cholesteatoma. A first MR imaging study of the head (formerly, he had been examined only with CT) confirmed recurrence of a previously operated cholesteatoma, but also showed vessel flow voids in the anterior cranial fossa that were highly suggestive of an arteriovenous malformation. Cerebral angiography showed a dural AVF of the anterior cranial fossa, supplied by both ophthalmic arteries and draining frontally into the superior sagittal sinus and posteriorly via a lateral anastomic mesencephalic vein into the superior petrosal sinus. Angiography revealed a severe stenosis with prestenotic ectasia on this deep venous route (Fig 2A and B). As the patient was asymptomatic and reluctant to undergo a second skull operation, an endovascular approach was proposed.

A decision was made to perform transvenous embolization, analogous to the first case. With the patient under general an-

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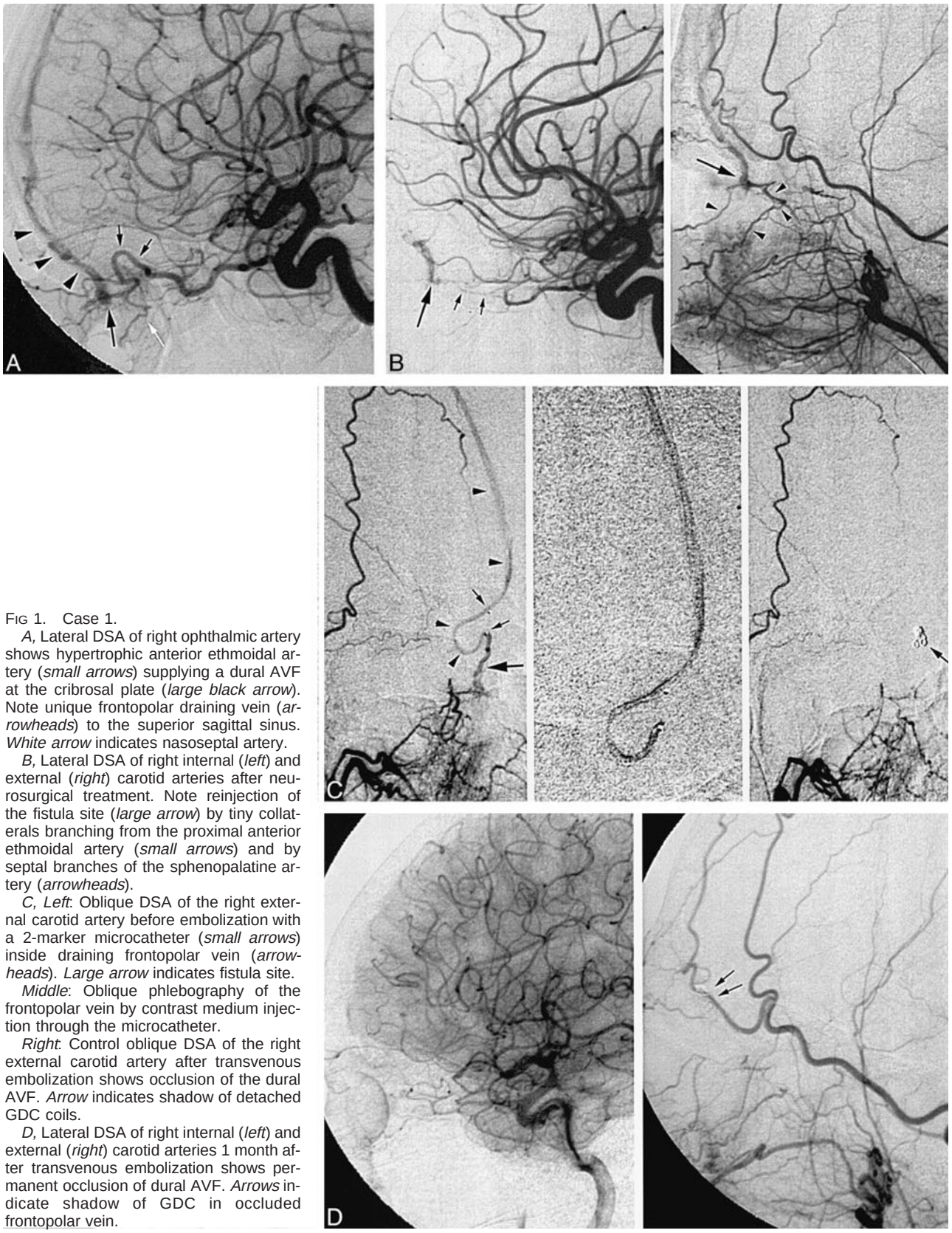

FIG 1. Case 1.

A, Lateral DSA of right ophthalmic artery shows hypertrophic anterior ethmoidal artery (*small arrows*) supplying a dural AVF at the cribrosal plate (*large black arrow*). Note unique frontopolar draining vein (*arrowheads*) to the superior sagittal sinus. *White arrow* indicates nasoseptal artery.

B, Lateral DSA of right internal (*left*) and external (*right*) carotid arteries after neurosurgical treatment. Note reinjection of the fistula site (*large arrow*) by tiny collaterals branching from the proximal anterior ethmoidal artery (*small arrows*) and by septal branches of the sphenopalatine artery (*arrowheads*).

C, Left: Oblique DSA of the right external carotid artery before embolization with a 2-marker microcatheter (*small arrows*) inside draining frontopolar vein (*arrowheads*). *Large arrow* indicates fistula site.

Middle: Oblique phlebography of the frontopolar vein by contrast medium injection through the microcatheter.

Right: Control oblique DSA of the right external carotid artery after transvenous embolization shows occlusion of the dural AVF. *Arrow* indicates shadow of detached GDC coils.

D, Lateral DSA of right internal (*left*) and external (*right*) carotid arteries 1 month after transvenous embolization shows permanent occlusion of dural AVF. *Arrows* indicate shadow of GDC in occluded frontopolar vein.

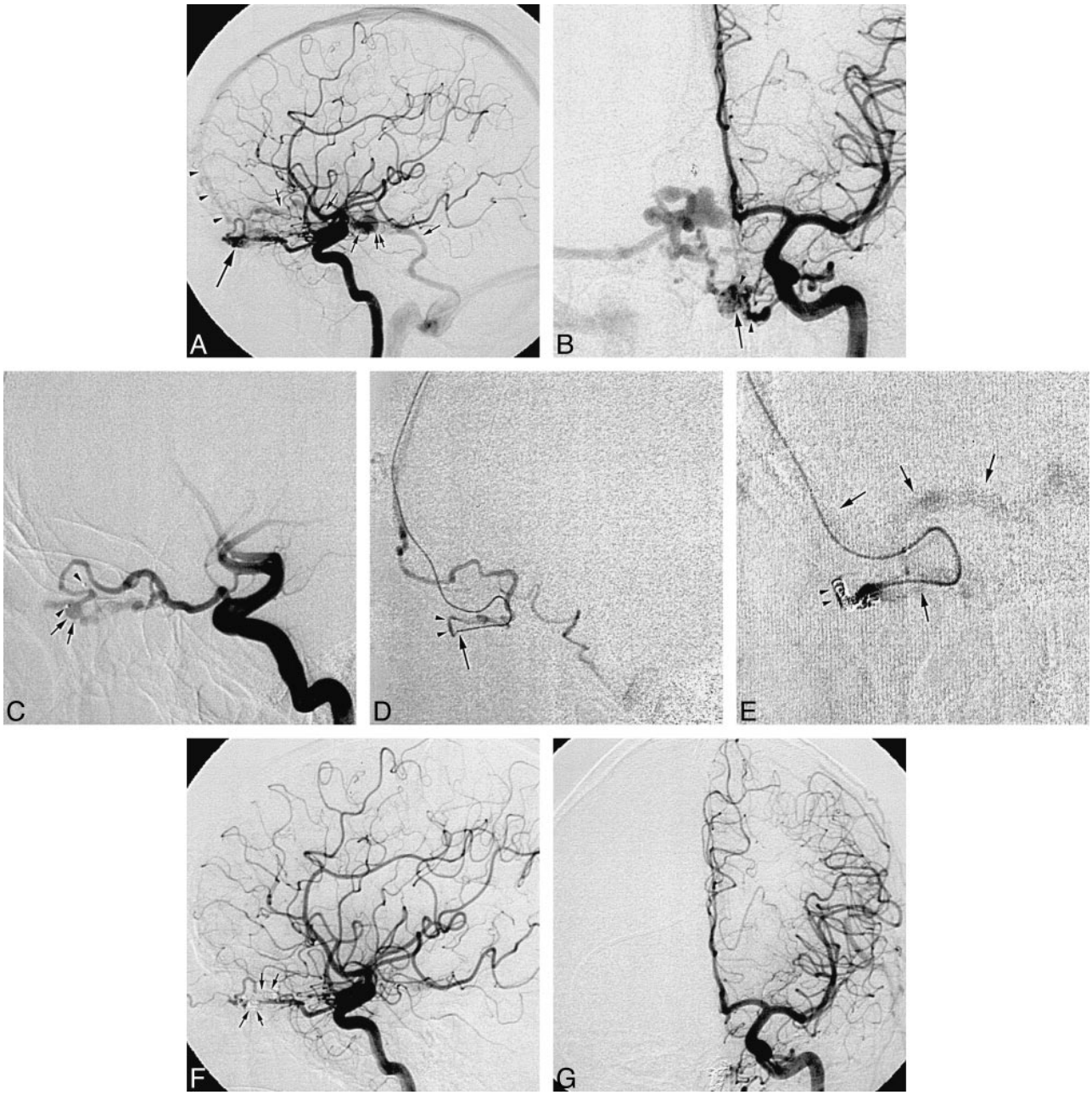

FIG 2. Case 2.

A, Lateral DSA of right internal carotid artery shows a hypertrophic anterior ethmoidal artery feeding a dural AVF of the anterior cranial fossa (*large arrow*). Venous drainage follows a frontalpolar route to the superior sagittal sinus (*arrowheads*) and a deep route via the olfactory vein to the lateral anastomotic mesencephalic vein to the superior petrosal sinus (*single small arrows*). Note severe stenosis at the petrosal junction with venous ectasia (*double arrow*).

B, Frontal DSA view of the left internal carotid artery shows supply of the dural AVF (*arrow*) by the contralateral anterior ethmoidal artery (*arrowheads*). Note stenosis and multiple ectases of the draining mesencephalic vein.

C, Magnification DSA of the left ophthalmic artery: early arterial phase depicts both arterial and venous sides of the fistula point. Both radiopaque markers of the microcatheter (*arrowheads*) are visible within the immediate extension of the venous pouch (*arrows*).

D, Superselective contrast medium injection through the microcatheter confirms its tip (*arrow*) location inside the origin of the first branching vein (*arrowheads*), draining predominantly frontally to the superior sagittal sinus.

E, Superselective phlebography after occlusion of the first draining vein (*arrowheads*) now shows opacification of the venous pouch. Subsequently, both the large frontal vein in which catheterization was performed and the lateral mesencephalic vein became faintly visible (*arrows*).

F, Postembolization lateral DSA of right internal carotid artery shows occlusion of the dural AVF. Eight soft GDC-10 coils (diameter, 2–4 mm; length, 3–10 cm) and one GDC-10 coil (diameter, 5 mm; length, 15 cm) were delivered (*arrows*).

G, Postembolization frontal DSA of the contralateral internal carotid artery shows occlusion of the dural AVF. (DSA of both external carotid arteries confirmed obliteration of the fistula [not shown].)

esthesia, venous access was obtained by retrograde puncture of the right internal jugular vein and placement of a Tracker-38 guiding catheter into the frontal segment of the superior sagittal sinus. After transfemoral catheterization of the right and left carotid arteries, a right oblique projection centered on the left ophthalmic artery was chosen as the best working position. The venous fistula pouch was localized over the right cribrosal plate and then retrograde catheterization was performed with a Tracker Excel (Target) in an over-the-wire technique (Fig 2C). Superselective venography showed that the tip of the microcatheter was located in the first branching vein beyond the fistula point (Fig 2D). This small vein and the venous pouch were then occluded by serial packing with GDC-10 coils (Fig 2E) until injection of contrast medium into the internal and external carotid arteries of both sides showed occlusion of the AVF (Fig 2F and G). The patient left the hospital 2 days after the intervention in perfect neurologic condition. A control angiogram 8 months later showed occlusion of the dural AVF.

Discussion

Among the dural arteriovenous malformations of the brain, AVFs located in the anterior cranial fossa are the most rarely encountered. An article by Halbach et al (1), published in 1990, reviewed 33 new cases since 1963, the year Lepoire et al (2) described their first three encounters with this entity. Since that survey, dural AVFs of the anterior cranial fossa have been reported with increasing frequency, at a rate of six new cases per year and per article, for a total of 83 cases recorded in the literature (3–11 [references 5 and 9 contain three identical patients]). Dural AVFs of the anterior cranial fossa occur more frequently in men than in women, with a ratio of 7:1, and usually in the elderly (70% of patients are older than 50 years; no cases in children have been reported). The most frequent initial symptom is hemorrhage, either subarachnoidal, subdural, or intracranial. Convulsions and psychomotor and oculomotor disorders have also been reported. Dural AVFs of the anterior cranial fossa have occasionally been observed incidentally during imaging for other diagnostic purposes, as in one of our cases (12).

Dilated cortical veins may be visible on CT or MR studies, and suggest dural AVF (1, 13), but bilateral external and internal carotid angiography is still required to establish the diagnosis and to map arterial feeders and venous drainage. Usually, both ophthalmic arteries are hypertrophic and feed the dural AVF via the anterior ethmoidal arteries (therefore also called anterior ethmoidal dural AVF). External carotid feeders from penetrating septal branches of the sphenopalatine arteries are almost always present. The dural AVF usually drains via a frontal cortical vein directly into the superior sagittal sinus. A few cases with venous outflow directly to basal veins or to the cavernous sinus have been described (1, 14–16). As in case 2, these alternative venous outflows do not seem to be associated with frontal cortical outflow obstruction.

Dural AVFs of the anterior cranial fossa are thought to represent a high risk for hemorrhage because of their cortical venous drainage (types III and IV) (17 [this article documents eight more cases of dural AVFs of the anterior cranial fossa]). Even in the absence of venous outflow stenosis and venous ectasia, dural AVFs of the anterior cranial fossa may bleed, as was seen in case 1. In view of their aggressive nature, dural AVFs of the anterior cranial fossa require treatment. Neurosurgical disconnection of the vascular shunt between dural arteries of the cribrosal plate and pial veins of the frontobasal lobe has been the method of choice. Usually, a low frontal craniotomy with electrocoagulation and transection of the vascular connection and eventually excision of the fistula site is performed (1, 3).

Transorbital ligature of the anterior ethmoidal artery should not be done, because distal collaterals will reinject the dural AVF at the level of the lamina cribrosa, as was observed in case 1 and in other reported cases (16). For similar reasons, transarterial embolization has not been very successful in dural AVF. Occlusive agents such as polyvinyl alcohol particles and *N*-butyl 2-cyanoacrylate will be caught in the ethmoidal web of tiny feeders proximally to the fistula site and will not reach the venous portion. Moreover, approaching the dural AVF of the anterior cranial fossa through the ophthalmic artery carries a substantial risk of retinal infarction. Therefore, transarterial embolization has been reserved for the few patients who cannot undergo surgery or for those in whom surgery has failed (1, 16).

Lately, neurosurgeons as well as interventional neuroradiologists have shown specific interest in the venous compartment of dural AVFs. Surgical disconnection of retrograde draining leptomeningeal veins has proved to be a promising procedure for treating aggressive dural AVFs (7, 11). Similarly, a transvenous endovascular approach to dural AVFs involving the cavernous and dural sinuses has been performed successfully with low risk (18, 19). Curiously, transvenous embolization has not been considered in the treatment of dural AVFs of the anterior cranial fossa, probably because neurosurgical access is easy and the operation highly efficacious. Frontal craniotomy, however, is not without risk.

In performing a low frontal craniotomy, the frontal and ethmoidal sinuses may be opened, with the high risk of CSF leakage and intradural infections. If the craniotomy is performed above the frontal sinus, retraction damage to the frontal lobe and trauma to the olfactory nerves may occur. As a way to avoid these dangers, an interhemispheric approach has recently been proposed (9). This technique employs an operative microscope to avoid dural sinus damage and thrombosis or cortical vein rupture. Although the general complication rate is low, patients with minor psychomotor disorders may be reluctant to undergo craniotomy and therefore

will continue to live with a high risk of hemorrhage (4, 9).

As demonstrated by our two cases, transvenous embolization may become a valuable and safe alternative to open surgery. By choosing a transjugular access, problems with catheter length and friction are avoided. Catheter stability in the dural sinus can be ensured by the use of a highly flexible hydrophilic coaxial system, which supports navigation of the microcatheter through frontal bridging vein tortuosities. The microcatheter should be advanced over the guidewire very gently to avoid spasm and rupture of the draining vein or, eventually, a venous aneurysm. As occlusive agents, the fiber helical platinum microcoils seem to have been replaced by mechanically or electrolytically detachable coils (20). Detachable coils have the advantage of retrievability when misplaced or when coil dimensions do not fit the diameter of the vein. Because of the length and circular memory of the detachable coils, packing the venous outflow is quicker and denser. Usually, the stiffer 0.018-inch detachable coils are used because of their greater stability.

In the presented cases, we started the procedure with the smallest and softest GDCs to fit the small diameter of the draining frontal veins. We intentionally did not choose the stiffer GDC-18 (even if the 2-mm GDC-18 had been in production) to minimize the risk of damage and rupture of the tiny frontal veins. By choosing the softer GDC-10, in the first patient, we managed to fill the small venous gap partially by gently pushing the first soft GDC with the delivery wire distally from the tip of the microcatheter toward the lamina cribrosa. This maneuver should be performed with great caution because of the relative stiffness at the junction of the delivery wire and the coil, which could perforate the small vein. After delivery of the second coil, the AVF became occluded, which emphasizes the role of the draining vein in the hemodynamics and treatment of dural AVFs.

Although we achieved distal retrograde venous catheterization in the second case, the frontobasal/olfactory venous complex was too tortuous to allow progress of the microcatheter into the venous pouch immediately adjacent to the fistula point. This would have been the ideal position from which to deliver the occlusive coils; however, we saw that effective embolization can start inside the first venous side branch. Once this branch is packed with GDCs, the microcatheter will automatically be pushed back and fall into the venous fistula pouch, which can then be packed with larger GDCs. Careful study of the angioarchitecture of the dural AVF during arterial injection and with the microcatheter already in place in the venous outflow is mandatory to recognize the level of the first branching dilated vein and to determine the delivery position of the first coil.

Conclusion

Our experience with these two patients indicates that transvenous embolization might offer an efficacious and safe alternative to classical neurosurgical treatment of dural AVFs of the anterior cranial fossa.

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