The Role of Transvenous Embolization in the Treatment of Intracranial Dural Arteriovenous Fistulas

ISSN: 0148-396X
Accession: 00006123-199706000-00004

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Issue: Volume 40(6), June 1997, pp 1133-1144

Publication Type: [Technique Assessment]

Publisher: Copyright © by the Congress of Neurological Surgeons
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Institution(s): Received, August 16, 1996. Accepted, January 9, 1997.
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Keywords: Fistula, arteriovenous, Fistula, dural, Interventional procedures, Sinuses, dural

Abstract

OBJECTIVE: To evaluate the role of transvenous embolization in the treatment of intracranial dural arteriovenous fistulas (DAVFs), including its efficacy and safety.

METHODS: We retrospectively studied the charts of 24 patients (21 women and 3 men) treated for an intracranial DAVF since 1990 in whom a transvenous approach was attempted either alone (16 patients) or in combination with arterial embolization (8 patients). There were 12 cavernous sinus, 9 transverse-sigmoid sinus, 2 inferior petrosal sinus, and 1 intradiploic fistulas. Three fistulas were Type I, 12 were Type IIa, and 9 were Type IIa+b, according to the revised Djindjian's classification. Transvenous embolic agents included coils (17 patients), detachable balloons (6 patients), bucrylate (2 patients), and silk sutures (1 patient).

RESULTS: Anatomic cure was proven in 21 patients (87.5%). Clinical cure was obtained in 23 cases (96%), as follows: 15 patients with a single transvenous approach, 6 with a combined arteriovenous approach, and 2 with an arterial approach after failure of venous access. There was one persistent cavernous fistula despite coil packing of the cavernous sinus. Complications were as follows: five transient and one permanent sixth nerve palsies in cavernous DAVFs, two transient labyrinthic dysfunctions in transverse sinus DAVFs, and one subarachnoid hemorrhage without sequelae.

CONCLUSION: Transvenous embolization is a useful and safe approach in the management of intracranial DAVFs.
The treatment of intracranial dural arteriovenous fistulas (DAVFs) is difficult and many therapeutic approaches have been proposed. Manual compression of the common carotid or occipital artery has been successful in a minority of cases (11). Surgery is difficult and may be accompanied by massive blood loss (2, 28). It is most commonly used with DAFVs harboring direct leptomeningeal drainage because these are difficult to treat by endovascular embolization. Endovascular techniques are now frequently used as the first treatment for most DAVFs. Complete cure by arterial embolization is difficult (31) because of the multiplicity of the feeding arteries in most cases. Embolization with particles may be used for the palliation of symptoms, but recanalization occurs with these materials (6, 11, 14, 15, 31). Bucrylates provide more stable results but demonstrate a higher risk because of the numerous dangerous anastomoses involved in these areas and because the vascular supply of the cranial nerves may be jeopardized (2, 6, 31).

Transvenous embolization has been advocated more recently (10, 12, 13, 26, 29-31) and may be combined with arterial embolization in selected cases (26). The role of this approach in the management of DAVFs is still not well established. We present our experience in 24 patients with DAVFs in whom transvenous embolization was at least attempted in their overall management.

PATIENTS AND METHODS

The charts of 24 patients with a diagnosis of a DAVF, in whom a transvenous embolization was attempted since 1990, were reviewed. The patients included 21 women and 3 men (range, 27-78 yr; mean, 56 yr). All patients underwent complete cerebral angiography, including both internal and external carotid for cavernous DAVFs, as well as bilateral vertebral injections for the other DAVFs. There were 12 cavernous sinus, 9 transverse-sigmoid, 2 inferior petrosal sinus, and 1 intradiploic fistulas in the torcular area. Three fistulas were Type I, 12 were Type IIa, and 9 were Type IIa + b, according to the revised Djindjian's classification (8) (Table 1). The transvenous approach was used alone in 16 patients and in combination with an arterial embolization in 8 patients. All venous approaches were from the femoral vein. Fibered platinum coils (Target Therapeutics, Fremont, CA) were used in seven patients, IDC coils (Target Therapeutics) in seven patients, detachable latex balloons (Nycomed-Ingenor, Paris, France) in six patients, GDC coils (Target Therapeutics) in five patients, Gianturco coils (Cook, Stouffville, Ontario, Canada) in three patients, bucrylate (N-butyl cyanoacrylate[NBCA]; Braun, Melsungen, Germany) in two patients, and silk sutures in one patient. Detachable balloons were used via a transvenous route to occlude transverse sinus DAVFs. After inflation of the balloon into the sinus at the level of the lesion and before detachment, an angiographic assessment of the venous phase of the ipsilateral internal carotid injection, as well as of the posterior fossa, was performed to exclude the potential impairment of normal tissue drainage. Balloon test occlusion was performed in Type I and IIa transverse sinus fistulas. Bucrylate was used transvenously in combination with coils in two cases of high-flow cavernous DAVFs. For arterial embolization, NBCA was used in seven patients, and particles (Contour Emboli; Interventional Therapeutics Corporation, Fremont, CA) were used in one patient. In early cases, the transarterial route was attempted in the first place, whereas later it was used when the transvenous route was not possible (one patient) or in case of a positive venous test occlusion (one patient). Follow-
up ranged from 2 to 44 months (mean, 14 mo). All patients except five underwent a control angiogram from 1 to 5 months after embolization. Of these five patients, one refused angiography, and three demonstrated a Type IIa cavernous DAVF that was clinically and angiographically cured immediately after treatment. The patient with the intradiploic fistula was also cured angiographically immediately after treatment. An anatomic cure was defined as the disappearance of all symptoms with the angiographic demonstration of the obliteration of the fistula. The clinical cure group included the anatomic cures, as well as patients in whom the disappearance of all symptoms was obtained despite a persistent shunt. Only lesions of a benign type (Type I and IIa) were considered clinically cured if asymptomatic without anatomic obliteration.

TABLE 1. Revised Djindjian's Classification

<table>
<thead>
<tr>
<th>Type</th>
<th>Description</th>
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</thead>
<tbody>
<tr>
<td>Type I</td>
<td>Antegrade drainage into a sinus</td>
</tr>
<tr>
<td>Type IIa</td>
<td>Reflux into the sinus (retrograde flow)</td>
</tr>
<tr>
<td>Type IIb</td>
<td>Reflux into cortical veins</td>
</tr>
<tr>
<td>Type IIa+b</td>
<td>Reflux into both sinus and cortical veins</td>
</tr>
<tr>
<td>Type III</td>
<td>Direct cortical venous drainage without venous ectasia</td>
</tr>
<tr>
<td>Type IV</td>
<td>Direct cortical venous drainage with venous ectasias</td>
</tr>
<tr>
<td>Type V</td>
<td>Spinal venous drainage</td>
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</tbody>
</table>

RESULTS

Patient data, including age, sex, site and type of DAVF, approach (venous, combined arteriovenous, or arterial), results, complications, and time of follow-up are summarized in Table 2. Mean follow-up of the patients was 12.9 months.
TABLE 2. Patient Data

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Sex</th>
<th>Age (yr)</th>
<th>Site</th>
<th>Type(^b)</th>
<th>Approach</th>
<th>Result(^c)</th>
<th>Complications</th>
<th>Follow-up (mos)</th>
<th>Control Angiogram</th>
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<td>Transient sixth nerve palsy</td>
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<td>Ila(a)+b</td>
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<td>Ila(a)+b</td>
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<td>Ila(a)+b</td>
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<td>Venous</td>
<td>Cure (A)</td>
<td>None</td>
<td>3</td>
<td>Yes</td>
</tr>
</tbody>
</table>

\(^a\) SAH, subarachnoid hemorrhage.
\(^b\) Revised Djindjian’s classification.
\(^c\) Cure (A), anatomic cure proven by control angiogram. Cure (C), clinical cure.
Anatomic and clinical results

Clinical cure was obtained in 23 of the 24 patients (96%). In 21 patients (87.5%), this was associated with the complete obliteration of the fistula (anatomic cure), as confirmed by immediate (4 patients) or late angiograms (17 patients). In one patient (Patient 13), a significant reduction of flow was obtained with the disappearance of symptoms (clinical cure without anatomic cure) via an arterial approach that was chosen because of a positive balloon test occlusion of the transverse sinus. In one patient (Patient 10), a transvenous embolization led to an important reduction of flow visible at the end of treatment with the disappearance of her bruit in the ensuing days, but she refused the late control angiogram (clinical cure without proof of anatomic cure). Finally, there was one failure of treatment (4%) despite transvenous coil packing of a cavernous fistula (patient is still symptomatic).

Of the 23 patients who experienced clinical cures, 15 experienced cures with a single venous approach, 6 with a combined arteriovenous approach, and 2 with a single arterial approach. These two last patients are included because a transvenous approach was attempted at first. In Patient 3, the transvenous catheterization of a cavernous fistula from the inferior petrosal sinus failed, and the lesion was subsequently cured by arterial embolization using bucrylate. This is included as an example of a failure of transvenous embolization. In one case of a transverse sinus Type IIa DAVF (Patient 13), the balloon test occlusion of the transverse sinus demonstrated impairment of cerebellar venous drainage. We decided to treat the patient by a partial arterial embolization with bucrylate. Clinical cure was obtained at a 6 month follow-up. A control angiogram obtained at 6 months revealed persistent shunt but with important reduction of flow compared with the pretreatment state.

Complications

Six patients with a cavernous fistula demonstrated transient ophthalmoplegia resulting from sixth nerve dysfunction. In three patients, this problem lasted from 4 to 6 months. In one of these cases (Patient 12), the patient recovered from sixth nerve palsy, which then recurred after 2 months. An angiogram obtained at the time of recurrence revealed no fistula. The patient underwent surgery for her external oculomotor nerve dysfunction. This was the only permanent complication of the present series. There were two patients who demonstrated transient labyrinthine dysfunction after a sigmoid sinus occlusion. Both patients presented with an acute vertigo that rapidly subsided under steroids and betahistine therapy. One of these patients also sustained a partial hearing loss on the treated side that lasted for several days. One patient presented a subarachnoid hemorrhage resulting from a sinus perforation by the guidewire. This patient made a complete recovery and is now asymptomatic. Overall, there were eight transient complications (33%) and one permanent complication (4%).
ILLUSTRATIVE CASES
Patient 5

A 55-year-old woman presented with a 6-month history of a left pulsatile tinnitus. This had been associated with episodes of left eye redness, proptosis, and diplopia. An angiogram (Fig. 1) revealed a high-flow Type IIa+b DAVF of the left cavernous sinus with multiple feeders from both internal and external arteries. Venous drainage was through the inferior petrosal sinus, the superior ophthalmic vein, and the deep sylvian vein. Under neuroleptanalgesia, a 5-French (F) catheter was introduced into the left common carotid artery for angiographic control. Another 5-F catheter was brought to the left inferior petrosal sinus. A tracker-18 microcatheter (Target Therapeutics) was coaxially brought to the venous pouch of the DAVF. Nine 6-mm fibered coils (Target Therapeutics) were deposited from the junction between the superior ophthalmic vein and the cavernous sinus to the posterior part of the cavernous sinus. A mixture of NBCA and Lipiodol (66% of NBCA) (Guerbet, Paris, France) was also injected during manual compression of the left eye to avoid distal migration of the glue into the superior ophthalmic vein (Fig. 1C). There was immediate disappearance of the bruit. The patient experienced a transient left sixth nerve palsy during the next few days. A control angiogram obtained 1 month later confirmed the complete cure of the fistula. The patient is now completely asymptomatic, with a follow-up of 23 months.
FIGURE 1. Patient 5. A, left common carotid injection (lateral view). Type II cavernous DAVF. Arrowheads, cortical veins; arrow, superior ophthalmic vein; open arrow, inferior petrosal sinus. B, injection into the cavernous sinus via an inferior petrosal sinus approach. Arrow, superior ophthalmic vein; arrowheads, cortical veins. C, same injection during manual compression of the eyeball. Arrow, compressed superior ophthalmic vein. The reflux into the cortical veins is more obvious (arrowheads). During bucrylate injection, this maneuver avoided distal embolization of the ophthalmic vein. D, computed tomographic scan showing coils and bucrylate into the cavernous sinus (arrow). E-F, injection of both common carotid arteries 1 month after embolization confirming anatomic cure.
Patient 7

A 58-year-old man presented with a 9-month history of a right pulsatile tinnitus associated with headaches. The angiogram (Fig. 2) revealed a Type I DAVF of the right transverse-sigmoid sinus fed by multiple branches originating from the right internal carotid, right external carotid, and right vertebral arteries. The venous drainage was antegrade through the right sigmoid sinus, but the sinus was occluded at its junction with the distal transverse sinus. Under neuroleptanalgesia, a 5-F catheter was introduced into the right common carotid artery for angiographic control. A 9-F femoral sheath was introduced into the right femoral vein. An 8-F guiding catheter was brought to the opening of the right sigmoid sinus into the right internal jugular vein. Next, a No. 9 Debrun latex balloon (Nycomed-Ingenor), hand-tied on a tracker-10 microcatheter (Target Therapeutics), was inflated into the right transverse sinus approximately 1 cm distal to the junction between the vein of Labbé and the transverse sinus (Fig. 2B). After successive injections of both the right internal and right vertebral arteries to assess the normal venous drainage of the supra- and infratentorial compartments, the balloon was detached. A second No. 9 balloon was detached just under the first one after reassessment of the phlebography of the posterior fossa during balloon inflation before detachment. There was immediate relief of the tinnitus. A control angiogram obtained 3 months later confirmed the complete cure of the DAVF.
FIGURE 2. Patient 7. A, right external carotid injection (lateral view). Type I transverse sinus DAVF (arrow) fed by multiple meningeal branches. B, venous phase of right carotid artery injection with a No. 9 Debrun balloon inflated into the transverse sinus. Note the position of the balloon (star) in relation with the opening of the vein of Labbeé into the transverse sinus (arrow). C, right common carotid injection 3 months after embolization showing complete obliteration of the fistula. Right vertebral and left common carotid injections (not shown) were also performed. D, cranial radiograph (lateral view) showing the position of the balloons.
Patient 20

A 67-year-old woman presented with an 8-month history of right eye redness and proptosis with diplopia. The magnetic resonance imaging scan revealed dilated cortical veins over the right hemisphere without dilatation of the superior ophthalmic vein. The angiogram revealed a Type IIa+b DAVF of the right cavernous sinus fed by multiple branches from both internal and external carotid arteries (Fig. 3A). The venous drainage was to the deep sylvian vein with venous congestion on the phlebographic phase of the right internal carotid injection. There was no opacification of the inferior petrosal sinus. Under neuroleptanalgesia, a 5-F catheter was introduced from the right femoral artery into the right common carotid artery for angiographic control. A 6-F catheter was brought from the right femoral vein into the lower part of the right inferior petrosal sinus (Fig. 3B). A double-marker Fas-tracker 18 (Target Therapeutics) was introduced into the venous pouch in which the fistula was first draining (Fig. 3C). The catheterization of the venous pouch was easy despite the lack of spontaneous opacification of the inferior petrosal sinus by the fistula. A total of seven IDC coils were put into the pouch. In the ensuing days, there was a transient increase in the diplopia, which rapidly subsided. At the 3-month follow-up, the patient was asymptomatic. The control angiogram revealed complete obliteration of the fistula (Fig. 3, D and E) with a normal venous phase of the right carotid injection.
FIGURE 3. Patient 20. A, left external carotid injection (face view). The right paracavernous DAVF is visible. Arrow shows the primary venous pouch and arrowheads point to the cortical drainage. There is no spontaneous opacification of the inferior petrosal sinus. The fistula was also fed by branches from both internal carotids and right external carotid (not shown). B, right inferior petrosal phlebogram (face view). There is no opacification of the venous pouch at the level of the fistula. C, injection into the venous pouch via the inferior petrosal sinus. Small arrow, proximal marker of the catheter; large arrow, coil into the venous pouch; arrowheads, cortical veins. D-E, injection of right (D) and left (E) common carotid arteries (face view) 3 months after treatment. There is no remaining shunt. Arrows point to the mass of coils.
Patient 22

A 46-year-old woman presented with a very uncomfortable pulsatile tinnitus on the right side. The angiogram revealed a Type IIa DAVF of the right transverse sinus fed by multiple branches originating from the right external carotid, right internal carotid, and right vertebral arteries. Figure 4 shows the balloon test occlusion of the transverse sinus. In the first position, the impairment of flow towards the contralateral transverse sinus led to reflux into the superior petrosal sinus and lateral mesencephalic vein (Fig. 4A). This would transform a Type IIa fistula into a Type IIa+b lesion, a more dangerous lesion. The balloon was thus positioned more caudally to avoid this reflux (Fig. 4B). After the embolization, which resulted in significant reduction of flow, the fistula went on complete occlusion, as confirmed on a control angiogram obtained 6 months later (data not shown).

FIGURE 4. Patient 22. A, common carotid angiogram (lateral view) during balloon inflation (star) into the transverse sinus just cranial to the superior petrosal sinus (small arrow). Note the reflux into the lateral mesencephalic vein (arrow). B, same injection with the balloon positioned more caudal (star). There is no more reflux into the lateral mesencephalic vein.
DISCUSSION

DAVFs account for 10 to 15% of all intracranial vascular malformations (20). Any sinus may be involved, but the two most frequent sites are the cavernous sinus and the transverse-sigmoid sinus (2). Symptoms are extremely variable and are closely related to the type of venous drainage (1, 2, 6, 8, 17, 18, 22, 26). Transverse sinus DAVFs typically cause a pulsatile tinnitus that is audible on auscultation of the mastoid area. Cavernous sinus DAVFs most commonly present with ocular symptoms, including chemosis, proptosis, ophthalmoplegia, and a raise in intraocular pressure. If the DAVF drains only into a sinus, the course of the disease is benign, whereas involvement of subarachnoid veins carries the risk of intracranial hemorrhage (1, 2, 6, 17, 18, 22, 23, 26, 28). High-flow DAVFs may also present with signs of intracranial hypertension or cranial nerve deficits (2, 6, 17, 18, 28). Djindjian and Merland (8) were the first to propose a comprehensive classification based on venous drainage, which is the main determinant of prognosis. This classification has been recently revised (6) (Table 1), with a distinction between DAVFs that drained antegradely into a sinus and DAVFs with a reflux into another sinus that were associated with a poorer prognosis in this series (6). It also includes intracranial DAVFs with a spinal drainage. A similar classification was also recently proposed by Borden et al. (2).

Although the exact etiopathogenesis is still debated, most authors now agree regarding the acquired nature of DAVFs (2, 3, 5, 9, 16, 21, 28). The epidemiological features of the clinical manifestations and the many reported cases with previously normal angiograms support this theory (3, 7, 9). A relationship between DAVFs and sinus thrombosis is evident, yet not well understood (3-5, 7, 9, 15, 16, 21, 24, 25, 31). Thrombosis might well be the triggering factor leading to the development of a DAVF, as was demonstrated angiographically in several cases (3, 7, 9). The opening of dural arteriovenous shunts is thought to accompany the recanalization process of a thrombosed sinus (3, 5, 9, 16, 21, 28). Venous occlusion also seems to explain the dynamic behavior of some DAVFs. This is especially true for cavernous DAVFs, in which a changing pattern of symptoms is often observed. The symptoms may change from a prominent bruit when the drainage is mainly posterior to sudden ocular problems caused by an inferior petrosal sinus occlusion and rerouting of the venous drainage to the superior ophthalmic vein. These secondary occlusions of the outflow of the fistula are either thrombotic or caused by a myointimal proliferation, as observed in high-flow angiopathy (24). Venous thrombosis is also involved in the spontaneous cure of this disease (25, 27). Furthermore, this process more commonly occurs with cavernous DAVFs (27). Thus, we are in a paradoxical situation in which the cause of the abnormality, i.e., venous thrombosis, might also be the curing process (19).

Consequently, transvenous embolization is logical as long as the diseased segment of the sinus is completely and permanently occluded with the embolic agent and as long as normal venous drainage is rerouted through another pathway (2). One of the drawbacks of venous embolization is the risk of changing the venous drainage of the fistula from a benign one to a more aggressive one with subarachnoid vein involvement (2). There is also a potential risk of venous infarction if the occlusion of a sinus causes the obstruction
of the outflow of a vein draining normal cerebral tissue (i.e., the vein of Labbé for the transverse sinus). The best indications for venous embolization are cases in which the involved sinus is already compromised and no longer contributes to the drainage of normal tissue (2, 31). Caution is recommended when contemplating occlusion of a sinus that is freely communicating with normal venous structures. The use of detachable balloons for occlusion of the transverse sinus is helpful. Angiographic test occlusion can be performed studying the phlebographic phase of both carotid and vertebral injections during occlusion to make sure that the venous outflow of the normal brain or cerebellum is not in jeopardy (31). In agreement with Urtasun et al. (31), we advocate the use of balloon test occlusion for Type I and IIa fistulas and Type IIb and IIa+b lesions in which there is ambivalent flow in the cortical veins. If the venous flow in the cortical veins is already inverted because of the fistula, sinus occlusion need not impair the normal drainage and test occlusion is not mandatory. In their series, Urtasun et al. (31) did not use balloons for the final occlusion itself, probably because of the fear of recurrence after balloon deflation. We prefer to use the same detachable balloon for the occlusion, because it might not be possible to deposit coils in the same position after the test occlusion. We inflate the balloons with contrast only. Even though the contrast-filled latex balloons deflated in the ensuing weeks in all patients in whom they were used, we did not observe any recurrences of the fistula. Technically, we use latex balloons hand-tied on a tracker-18 or tracker-10 microcatheter. This system allows the use of a guidewire to help the positioning of the balloon, the navigation being often difficult because of the postphlebitic changes in the sinus.

With the use of coils, complete occlusion of the fistula is often delayed. This may be troublesome because the end point of coil packing may not be easy to assess. Even if the packing seems complete, there is often some remaining flow and the thrombosis usually goes on to completion in the ensuing days. However, it was not the case in Patient 15 in whom the fistula remained. Dense tight packing of the venous pouch may prevent this problem but might lead to more frequent and more severe iatrogenic ophthalmoplegia in cavernous DAVFs. Urtasun et al. (31) reported a higher percentage of immediate occlusion. This is probably because of the more frequent use of glue in combination with coils. In our series, bucrylate was used during venous approach in two cases of high-flow cavernous DAVFs. In these cases, the thrombogenicity of coils was thought to be insufficient to close the fistula. Bucrylate injection was performed into a basket of coils. In this situation, distal migration of glue to the ophthalmic veins may be avoided by manual compression of the eyeball during injection, as in Patient 5. This technique could have been used in Patient 15, who also harbored a high-flow fistula. A late control angiogram is important if the angiographic proof of the cure is not obtained immediately. The late control angiogram is mandatory for Type IIb or IIa+b lesions, because the patient may be asymptomatic despite a residual shunt with drainage into subarachnoid veins.

We recommend the use of supple microcatheters and microcoils to avoid sinus perforation as in Patient 10, in whom 0.038 coils were used with a 5-F catheter. The occurrence of transient diplopia after cavernous sinus occlusion was frequent. Even though it was transient, it lasted long enough in some cases to cause a significant clinical
problem. It is probably because of the acute thrombus impinging on the oculomotor nerves and not because of the mass effect of the coils. If the latter were the case, the symptoms would be immediate and irreversible. Inflammation may also play a role, producing a situation comparable to the Tolosa-Hunt syndrome. We did not use anticoagulation nor steroids in these cases, but their use might be considered in the prevention of this complication. Patient 12 presented a puzzling problem; after the recovery of transient diplopia, this patient developed a recurrent and permanent sixth nerve palsy despite an angiogram that disclosed no abnormalities and the absence of other symptoms of cavernous sinus thrombosis. We still think that the sixth nerve palsy might be caused by a thrombotic event in a compromised cavernous sinus. We encountered two cases of transient labyrinthine dysfunction after sigmoid sinus occlusion. In both cases, the symptoms appeared more than 18 hours after treatment. This complication had been reported previously (13, 26) and is probably related to hydrops of the saccus endolymphaticus (which is situated in a dural fold in the vicinity of the sigmoid sinus), precipitated by the acute sinus thrombosis (26). A more permanent damage would be expected if a venous infarct of the labyrinth were involved.

Our results are comparable with those of Halbach et al. (10), who reported clinical cures in 81% of patients with cavernous DAVFs and cures or marked improvement in 55 and 35% of patients with transverse-sigmoid DAVFs, respectively. In their series, permanent complications occurred in 4% of patients with cavernous lesions, whereas permanent complications occurred in 5% and transient complications in 15% of patients with transverse-sigmoid fistulas. Urtasun et al. (31) reported anatomic cures in 80% and clinical cures in 90% of patients with transverse-sigmoid fistulas. Results were not as satisfactory for superior sagittal sinus fistulas. We do not have experience with these probably more challenging lesions. Transient complications occurred in 10% and permanent complications occurred in 5% of patients with transverse-sigmoid sinus fistulas.

We need to remember that even though endovascular techniques are evolving and venous embolization is an appealing way to deal with this disease, Type I DAVF is a benign entity (2, 6). Thus, the aggressiveness of the treatment needs to be tailored to the importance of the symptomatology and to the angiographic pattern that relates to prognosis. For example, occlusion of a patent transverse sinus involved with a low-flow DAVF draining antegradely may not be indicated (2, 31). In low-risk lesions of the transverse-sigmoid area, transvenous embolization is only indicated in selected cases in which the anatomy is favorable, as in Patient 7. If not, a simpler measure such as particle embolization is preferred for symptomatic relief in cases of a disturbing bruit. The possibility of progression from a low-risk type DAVF to a more aggressive one, although rarely reported (6, 31), warrants clinical follow-up of uncured patients. High-flow transverse-sigmoid DAVFs with reflux and retrograde flow were associated with potential neurological complications in a recent series (6), and treatment is indicated. We now first consider the venous approach when possible because of its better efficacy compared with the arterial route. Arterial embolization is added if needed. Whenever cortical venous drainage is present in a DAVF (Type Ib or IIa+b), radiographically demonstrated anatomic cure is the goal of treatment. A combination of arterial and
transvenous embolization as well as surgery, if necessary, is then indicated to prevent intracranial hemorrhage.

CONCLUSION

Transvenous embolization is a useful and safe approach in the treatment of intracranial DAVFs. It has contributed to a higher rate of cure of intracranial DAVFs than was achieved when only the arterial route was available.

REFERENCES


COMMENTS
This study contributes to the growing body of knowledge regarding dural arteriovenous fistulas (DAVFs), i.e., that they are venous in site, venous in pathogenesis, venous in symptomology and complications, and venous in treatment. Fistulas that demonstrate no evidence of retrograde orbital or intracranial venous drainage may not require treatment. There is an increasing logical consensus that percutaneous venous occlusion of the sinus lumen is, in most instances, the therapeutic method of choice. There is increasing recognition that the segment of sinus wall, which now carries the fistula, was at one time thrombosed and that an alternate venous return had, at that time, existed. Thus the segment could be again occluded safely as a permanent therapeutic measure. The authors emphasize, as illustrated in Figure 4 of the article, that the occlusion must be precise. An erroneous occlusion could reroute the fistulous output and overload an intradural vein.

As in any developing field, there is a need to fine-tune the methodology. The authors report on their experience but may not yet be in a position to define clearly the specific indications for balloon, wire, and glue. Balloons may outline a linear length to be occluded, sparing adjacent normal venous flow. In serial fashion, they may directly effect occlusion by impinging on the porous wall. Presently available coils are effective, but thrombosis in a large fistulous sinus is slow and tedious, requiring a great (and expensive) volume of coil. Bucrylate might always be regarded as a potential source of danger. In Patient 5, the authors prevented its entry into the orbit by compressing the eye but did not discuss the serious danger of an unprotected venous cortical infarct in this dominant hemisphere. The literature presents a case of aphasia from an intracavernous injection (1).

On occasion, a single entering vein may sustain simple occlusion. It might be held that an inadvertent occlusion of the superior petrosal vein (together with the fistula) in Figure 4 of the article would have been tolerated because of the very wide anastomosis of the lateral mesencephalic vein. I presume, from the evidence, that the sinus was not itself fistulous. The authors express especial concern about the vein of Labbé. The occlusion of this vein depicted in Figure 2B of the article might have created a problem (all radiographs are not available for review), but in this reviewer's experience, it was always possible to demonstrate preoperatively that a good anastomosis existed between the vein of Labbé and the vein of Trolard or the middle cerebral vein, thereby permitting an occlusion in selected instances. If an entering vein is observed preoperatively to carry fistulous (pressurized) blood away from the sinus, then it will certainly carry away the residual cortical (nonpressurized) blood.

The classification that is used is somewhat confusing because of its use of the word "reflux." "Drainage" would be a better word. Type IIa is retrograde drainage. Type IIb is a true reflux from a fistulous sinus into a normal cortical vein. Type IIa+b is a combination of retrograde sinus drainage and cortical vein reflux. Types III and IV demonstrate a direct sinus wall drainage into a cortical vein without going through the sinus lumen. In general, these veins are spontaneously occluded at their former point of entry into the sinus. Another Type VI would be needed to describe a combination of a direct wall drainage into a cortical vein (usually occluded at the point of sinus contact) and a separate direct wall drainage into the sinus lumen. Types III, IV, and VI do not
have cortical vein access via a trans-sinus catheter. Their primary treatment might be transarterial, provided that the supply was exclusively external carotid. They might also be dealt with by a very limited and simple craniotomy that detaches the vein from the sinus wall. I disagree with the authors in their assigning Sundt's classical reference to intrasinus thrombogenic packing (6). That dealt with sinus excision, a very different procedure, which many surgeons had abandoned long in advance of that 1983 article. The omission of the reference that introduced the concept of prefistulous thrombosis, by Sundt's colleague Houser, leaves the literature review of that aspect incomplete (2).

Regarding patient anxiety, the percutaneous procedure far outweighs surgical packing, but not in terms of patient discomfort or morbidity. The morbidity of the percutaneous procedure is modest, but points the way toward further effort. The reader might wish to have a clearer idea of the morbidity that might be ascribed to any particular treatment component, i.e., the catheter, the balloon, the glue, or the coil. Not enough detail is presented by which a reviewer could evaluate all aspects of the authors' technique. However, I wonder why more use was not made of the transophthalmic vein route of access. Its percutaneous puncture is not as simple as a femoral vein puncture, but a cutdown is a very simple task. I wonder whether the authors had fully explored the contribution that bilateral packing might afford a fistula of the lateral or sigmoid sinuses, using the bilateral femoral approaches.

A transcatheter thrombogenic material is needed that is as simple, effective, and complication free as the conventional topical surgical agents (Surgicel and Gelfoam). More detailed comments on this interesting subject have been published previously (3-5). This report is a significant contribution to the literature.

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The authors report their experience during the past 6 years using transvenous embolization techniques in 24 patients with various DAVFs. Transvenous embolization techniques have been used by interventional neuroradiologists for the past 10 years for this indication with great success, and the authors add another impressive series to the literature. These techniques are highly successful in the treatment of selected dural fistulas but cannot be used to treat all dural fistulas. All cavernous dural fistulas with a venous access route can be treated by this technique without risk of harm; however, dural fistulas in other locations must be carefully evaluated to ensure that the embolization does not interfere with normal venous drainage of the adjacent brain. High-risk fistulas, i.e., those that drain into cortical veins, can have the fistula site interrupted by surgery or transvenous embolization techniques without the risk of venous infarction, because the adjacent brain has already rerouted its drainage via venous collaterals. Low-risk fistulas, in which the brain still uses the involved dural sinus for egress of blood, need not be treated with this technique, as the authors have implied. However, many dural fistulas fall between these two extremes. Urtasun et al. (4) have suggested that temporary balloon test occlusion with angiographic evaluation and clinical testing be performed on selected cases. This suggestion has been echoed by the authors of this article. We agree that angiographic evaluation with balloon test occlusion seems to yield useful information; however, we are not confident that the clinical examination during the test occlusion can be a reliable predictor of tolerance to permanent occlusion. In patients who develop venous infarction from surgical interruption of cortical veins, there is often a long interval, usually several hours or more, between the time of occlusion of the veins and the clinical event. We thereby remain unconvinced that a short duration of balloon occlusion of the dural sinus means that permanent occlusion will always be tolerated.

The authors promote the use of the same balloon for test occlusion and for permanent embolic occlusion, a novel concept in this era of cost-effectiveness. Although they report success in six cases, there are several theoretical disadvantages to the use of a balloon for venous sinus occlusion. A series of balloons may leave small gaps between the balloons (as shown in Fig. 2D of the article). If the fistula involves this region of sinus and there is a cortical vein or sinus exiting at the same location (e.g., the superior petrosal sinus), the dural fistula may be converted into a higher risk category. Secondly, balloons may deflate sooner than a few weeks. If the proximal (posterior) balloon (Fig. 4D of the article) deflated shortly after treatment and before the fistula thrombosed, the downstream occlusion produced by the second balloon could divert fistula flow into cortical veins. Our group's experience with more than 100 cases of dural fistulas treated with transvenous embolization with fibered coils alone has demonstrated a high cure rate (1). With the advent of new coil designs that allow greater density of coil packing, the cure
rates have risen significantly in the past years and, thus, fibered coils remain our primary embolic device in these cases.

Lastly, it is important to acknowledge the pioneering neurosurgical work of Hosobuchi (2) and Mullan (3) who, more than two decades ago, taught us that transvenous coils embolization of direct fistulas is possible. Roy and Raymond make a significant contribution to the treatment of a complex and vexing disorder.

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The authors describe the endovascular approach to DAVFs, which uses transvenous embolization to occlude the fistula. The authors well document the usefulness of this approach compared with previously reported series. They provide an honest appraisal of the cranial neuropathies that may result after thrombosis of these lesions, which has probably been underreported in the past. It is encouraging to observe that these cranial neuropathies are almost always temporary. The transvenous embolization of these lesions is well documented in this article and in previous reports. The particular use of transvenous embolization of transverse sigmoid sinus fistulas is evolving as a primary mode of therapy, obviating the need for surgery. Furthermore, the authors described in detail the pitfalls of this approach.
This article confirms the results of previous studies, particularly those published by Halbach et al. (2). Previously published work by Barnwell et al. (1) regarding complex DAVFs also needs to be considered. I have several comments regarding the transvenous embolization route. Concerning occlusion of the cavernous sinus, we almost uniformly observe a temporary period of severe nausea and headache. We routinely treat this with Decadron, volume expansion, and antiemetics. I agree with Roy and Raymond in that there may be a mild transient worsening of ocular symptoms after successful occlusion of the cavernous sinus.

In general, we use transarterial particle embolization in nearly all cases of DAVFs (whether they involve the cavernous or transverse/sigmoid sinuses) to slow the flow through the fistula to prepare it for transvenous coil embolization. Preembolization cerebral arteriography is essential to determine the pattern of ipsilateral cerebral drainage, particularly in relation to the vein of Labbé. If there is no antegrade drainage within the vein of Labbé into the effected transverse sinus, then we are more confident regarding skeletonizing this sinus to treat a DAVF. I am sure that the authors are aware of the potential risks of performing a temporary balloon occlusion of an affected dural sinus, particularly if this sinus provides the only venous drainage from the cerebral hemispheres, because this may precipitate intracranial hemorrhage. I think that the anatomic information from preembolization angiography is more important than balloon occlusion testing.

Regarding the agent to be used for occlusion of a dural sinus, we prefer fibered microcoils. Newer fibered coils, including Vortex coils from Target Therapeutics and Tornado coils from Cook, Inc. (Bloomington, IN), seem to be more thrombogenic; however, complex helical coils from Target Therapeutics may be useful for densely packing a dural sinus. In a number of cases, Roy and Raymond used detachable coils of the IDC and GDC variety, although it is known that these coils are not very thrombogenic. I personally prefer using the fibered coils.

Regarding the use of balloons within the dural sinuses, it is challenging to introduce a balloon loaded on a microcatheter into an arterialized dural sinus, particularly if it is being inserted against the flow within a drainage pathway (i.e., inferior petrosal sinus). Additionally, regarding the use of balloons within the transverse/sigmoid sinus, unless there is complete obliteration of this space within the affected dural sinus, arterialized venous flow may be inadvertently redirected into other venous pathways, including cerebral cortical veins, thus creating the potential for intracranial hemorrhage. I do not recommend the use of balloons for dural sinus occlusion.

Regarding the use of cyanoacrylate within the dural sinuses, a number of hazards are inherent in the use of this material. Roy and Raymond correctly state the risk of allowing flow of cyanoacrylate into the superior ophthalmic vein, in which the flow may block
ciliary veins, leading to blindness. One may avoid flow of the N-butyl cyanoacrylate into these structures by fully coil-occluding the posterior portion of the superior ophthalmic vein (the authors mention placing pressure on the globe to provide the same protection). In the case presented in Figure 1 (Patient 5) of the article, there is, apparently, extensive injection of glue into cortical veins. This is not a benign occurrence and could have caused extensive cerebral venous infarction. This particular complication is to be avoided. In addition, injection of glue into the cavernous sinus may be associated with retrograde flow of glue into minute arterial feeders that collateralize with the cavernous portion of the internal carotid artery. Although the tools and techniques mentioned by Roy and Raymond are very effective and seem to have worked in their patients, caution needs to be used in these procedures, particularly with the use of N-butyl cyanoacrylate.

This information is useful and confirms previous reports of similar techniques. I agree with the authors that the transvenous approach is the most effective means of dealing with DAVFs in which the affected dural sinus compartment can be identified and small fistulas without serious symptomatology or without cortical venous filling probably need to be treated in a more conservative fashion.

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Roy and Raymond report on a retrospective series of 24 patients with DAVFs. Sixteen patients were treated using a transvenous approach, and eight patients were treated with a combined transvenous-arterial approach. Combinations of embolic material were used to treat the fistulas.

The authors differentiate between anatomic and clinical cure. They define cure as angiographic obliteration, symptom resolution, or both. They report an 87.5% anatomic cure rate and a 96% clinical cure rate. Follow-up angiography was performed in 17 patients, supporting the claim of anatomic cure (angiographic obliteration). The authors state that Type I and IIa lesions are cured if they are asymptomatic, despite persistence of a shunt.

The results reported here demonstrate the remarkable ability of endovascular techniques to obliterate these tenacious lesions, yet our lack of knowledge concerning the natural history of these lesions forces us to question the validity of "clinical" cure. Dural fistulas
that are not obliterated on angiograms may continue to grow. It is possible that a clinically cured Type I or Ia lesion could enlarge and develop cortical venous drainage. Therefore, we suggest a rigorous follow-up in these patients that must include cerebral angiography. Even if symptoms do not recur, angiographic follow-up is essential. The authors emphasize the need for venous as well as arterial approaches and the need for both arterial and venous catheters during treatment, so that arterial and venous angiograms may be obtained.

The authors allude to the difficulty of permanently occluding DAVFs that involve the superior sagittal sinus. In our experience, these lesions present significant treatment dilemmas, occasionally defying permanent endovascular occlusion. Aggressive surgical removal is sometimes necessary.

This article is predated by a report by Urtasun et al. (1) of 24 patients with DAVFs. Complete occlusion was achieved in 17 patients, important flow reduction was realized in 3, and moderate flow reduction in 4. Urtasun et al. (1) used temporary balloon occlusion of the venous sinus/fistulous connection before treatment. We agree that the use of temporary balloon occlusion at the site of fistula formation is tantamount to safe treatment of these lesions. Changes in venous outflow need to be carefully studied after temporary occlusion to avoid inducing retrograde cortical venous drainage or occlusion of normal venous drainage (e.g., the vein of Labbé).

I need to mention that this study did not stress the importance of cooperation between endovascular and surgical specialists. When a DAVF remains after endovascular therapy, open surgery can sometimes be used to safely and easily complete the job. Surgery after embolization has reduced vascularity, is usually low risk, and may be as simple as the placement of a clip on a residual draining vein.

Although numerous reports describe the use of endovascular techniques for the treatment of DAVFs, the studies of Urtasun et al. (1) and Roy and Raymond demonstrate that transvenous or combined transvenous-arterial approaches are superior to other therapies. Their results are impressive. As devices, embolic agents, and catheter technology evolve, even the most tenacious DAVFs may be obliterated by endovascular therapy.

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Plate depicting general anatomy of the brain and its coverings.