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Dural Arteriovenous Fistulas of the Posterior Fossa Draining into Subarachnoid Veins

Laurent Pierot,¹ Jacques Chiras,¹ Jean-François Meder,² Michèle Rose,¹ Maurice Rivierez,³ and Claude Marsault¹

Purpose: To describe the clinical presentation, angioarchitecture, and risks and problems of therapy in patients with dural arteriovenous malformations of the posterior fossa draining into subarachnoid veins. **Patients and Methods:** Twelve patients with dural arteriovenous malformations of the posterior fossa draining into subarachnoid veins were studied. **Results:** These fistulas often presented with intracranial hemorrhage (eight cases) and myelopathy (two cases). They were located in the tentorium (six cases) or at the skull base (six cases). The arterial supply was provided by branches of the external carotid artery (nine cases), by the posterior meningeal branch of the vertebral artery (nine cases), and by the meningohypophyseal trunk (three cases). The fistulas drained directly into a cortical vein (six cases) or into a venous lake (six cases). In two cases, perimedullary draining veins were observed. The treatment modalities were endovascular embolization alone (two cases), surgery alone (five cases), and embolization followed by surgery (three cases). Despite the treatment, four patients died; in two cases, intracranial hemorrhage recurred. **Conclusions:** Subtotal occlusion of a fistula by surgery or embolization alone is not protective against further complications, especially hemorrhage. The goal of treatment is to achieve a rapid and complete anatomical cure; combined endovascular and neurosurgical treatment seems to be the therapeutic choice.

Index terms: Fistula, arteriovenous; Arteriovenous malformations, cerebellar; Arteriovenous malformations, cerebral; Fistula, therapeutic blockade

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Dural arteriovenous fistulas (DAVFs) account for 10% to 15% of all intracranial arteriovenous malformations (1). 35% of arteriovenous malformations located in the posterior fossa are purely dural and often involve the transverse and sigmoid sinus (2). The clinical presentation is in close relationship with the type of venous drainage of the DAVF (3). Intracranial DAVFs have been classified according to the venous return: group I: immediate venous drainage into a sinus, with normal direction of flow; group II: normal drainage, but accompanied by reflux into cortical veins; group III: direct drainage into a cortical

vein, with retrograde flow; group IV: drainage into a venous lake, which can behave as an expanding process (4). DAVFs with cortical venous drainage are often associated with subarachnoid or intracerebral hemorrhage (5–7). Therapeutic methods include vascular compression (8, 9), transarterial embolization (8–15), transvenous embolization (16–18), surgical excision (13, 19), clipping of the draining vein (20), and embolization followed by surgery (8, 21–23). We report 12 cases of DAVFs of the posterior fossa draining into subarachnoid veins, describe their clinical presentation and angioarchitecture, and discuss their treatments.

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Subjects and Methods

The clinical and radiographic findings of 12 patients with DAVF of the posterior fossa draining into subarachnoid veins were reviewed. The patients (three females and nine males) varied in age from 35 to 68 years (mean: 50 years). The clinical presentation and results of computed tomography (CT) and magnetic resonance (MR) imaging are summarized in Table 1. All patients underwent selective, bilateral vertebral, internal, and external carotid angiogra-

TABLE 1: Dural arteriovenous malformations of the posterior fossa draining into subarachnoid veins: clinical presentations, CT, and MR

No.	Age (years), Sex	Clinical Presentation	CT	MR
1	65, F	Headache	Cerebellar hematoma	
		Cerebellar signs	SAH	
2	56, M	Headache	SAH	
3	59, F	Headache	SAH	
			DVS in PF	
4	68, F	Headache	SAH	DVS in PF
			DVS in PF	
5	58, M	Headache	Hydrocephalus	
			DVS in PF	
6	35, M	Progressive myelopathy	Normal	
7	40, M	Diplopia		
8	50, M	Headache	SAH	
9	60, M	Progressive myelopathy		Cervical ^a
10	51, M	Headache	Cerebellar hematoma	DVS in PF
			DVS in PF	
11	56, M	Headache	SAH	
12	43, M	Headache	Cerebellar hematoma	

Note.—DVS = dilated vascular structures; PF = posterior fossa; SAH = subarachnoid hemorrhage.

^a Cervical MR revealed enlarged spinal cord with central high-signal.

TABLE 2: Dural arteriovenous malformations of the posterior fossa draining into subarachnoid veins: arteriography, treatments, and results

No.	Arterial Feeders	Fistula Location	Medullary Venous Drainage	Treatment	Results
1	Left VA,OA	TENT	No	EMB	Cured
2	Left VA,OA,MMA,MHT	TENT	No	SURG	Death
	Right OA,MMA				
3	Left OA,MMA,AP	TENT	No	EMB + SURG	Cured
	Right OA,MMA				
4	Left OA,MMA	TENT	No	EMB + SURG	Cured
	Right OA,MMA,AP				
5	Left VA,OA,MMA	TENT	No	SURG	Death
	Right VA,OA,MMA,MHT,AP				
6	Left MMA,MHT	TENT	Yes	EMB + SURG	Death
7	Left VA	BASE	No	No	Unknown
8	Left VA	BASE	No	SURG	Cured
9	Right VA,OA	BASE	Yes	SURG	Cured
10	Right VA,OA,AP,PAA	BASE	No	No	Unknown
11	Right VA,OA,AP,PAA	BASE	No	EMB	Death
12	Right VA	BASE	No	SURG	Cured

Note.—AP = ascending pharyngeal artery; BASE = skull base; EMB = embolization; MHT = meningo-hypophyseal trunk; MMA = middle meningeal artery; OA = occipital artery; PAA = posterior auricular artery; SURG = surgery; TENT = tentorium; VA = vertebral artery.

phy. Results of arteriography, treatments and therapeutic results are summarized in Table 2. Two patients were treated by transarterial embolization, five by surgery, and three by embolization followed by surgery. Two patients were untreated. Materials used for embolization were dura mater or polyvinyl alcohol (Ivalon). Results were evaluated by angiography and clinical follow-up. All but one of the treated patients were followed for at least 6 months.

Results

Clinical Presentations (Table 1)

The patients presented with intracranial hemorrhage (eight cases), progressive myelopathy (two cases), headache and behavioral disorders (one case), and V and VI nerves palsy (one case).

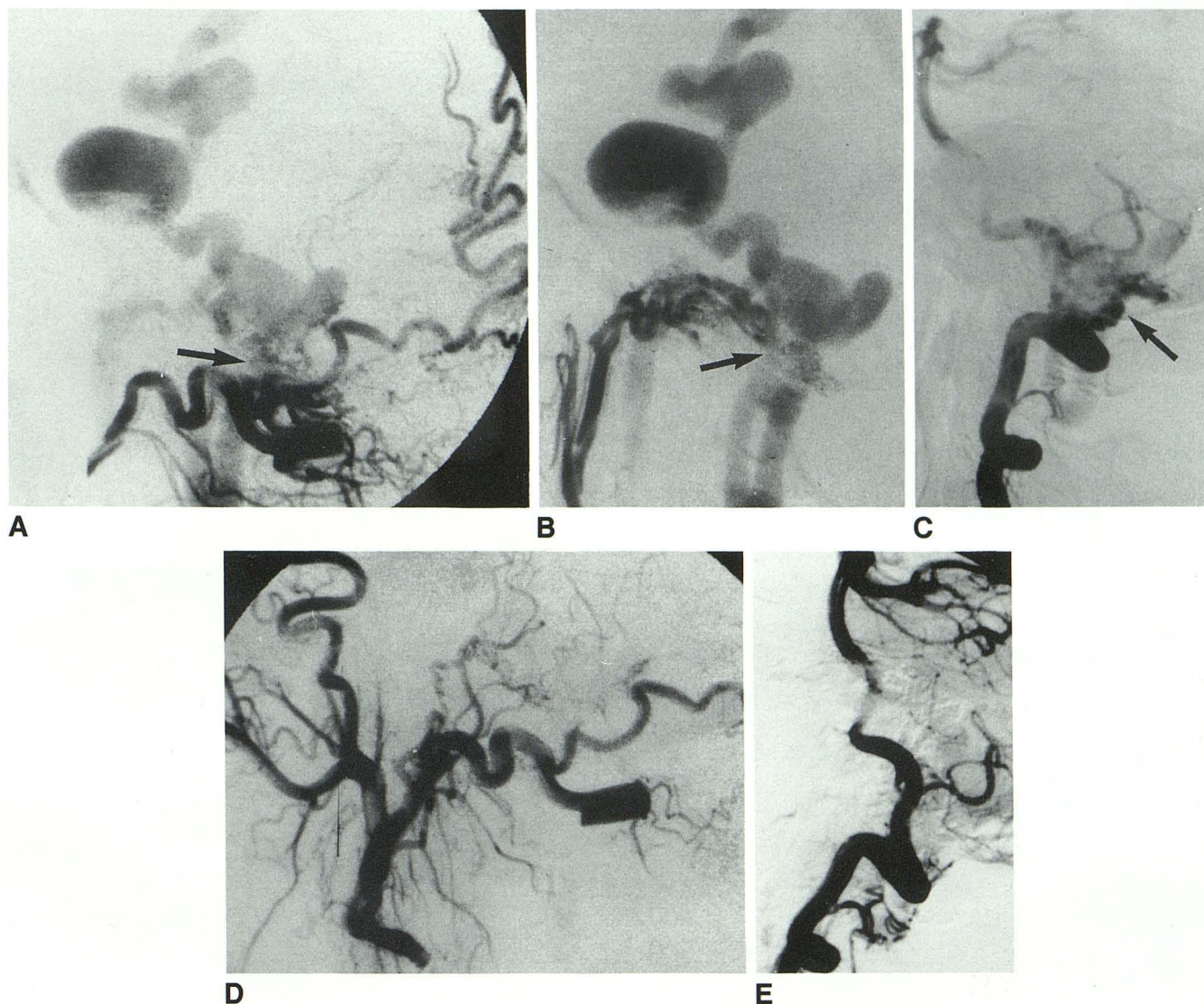


Fig. 1. Case 11.

A, Selective injection of the right occipital artery shows a foramen magnum fistula (*arrow*).

B, Selective injection of the right ascending pharyngeal artery shows the fistula (*Arrow*).

C, Right vertebral angiogram shows supply to the fistula by the meningeal branch of the vertebral artery, which is dysplastic (*arrow*).

D, Right external carotid angiogram performed after particulate embolization shows obliteration of the external branches feeding the fistula.

E, Right vertebral angiogram performed after the placement of a detachable balloon in the meningeal branch.

Location (Table 2)

The location of the fistulas were: tentorium (six cases), and skull base (six cases). The fistulas located at the skull base were all at the edge of the foramen magnum (Figs. 1 and 2). Fistulas located in the tentorium can be divided in two groups: posterior tentorial fistulas, which are in close relation with the torcular (cases 1, 2, 3, 4,

and 5; Fig. 3), and anterior tentorial fistula, located near the free edge of the tentorium (case 6).

Arterial Supply (Table 2)

Arterial supply was unilateral in eight cases and bilateral in four cases. Fistulas were supplied by one feeder in three cases and by multiple pedicles

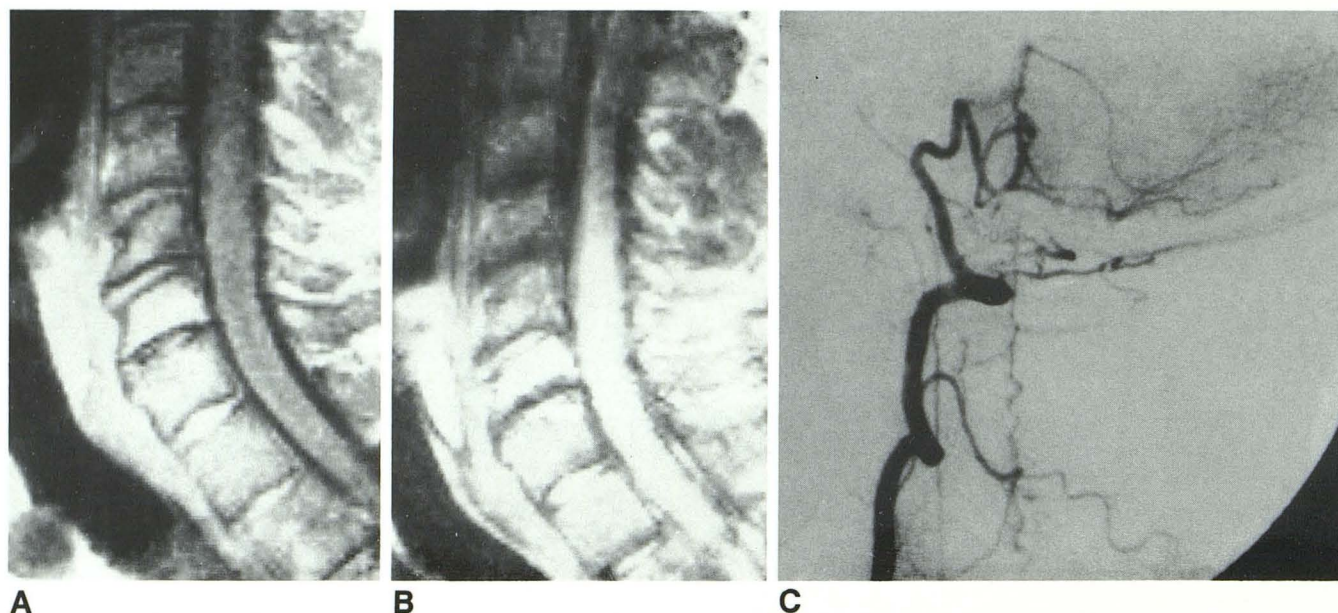


Fig. 2. Case 9.

A, T1-weighted images (TR = 400 msec; TE = 14 msec) shows enlargement of the spinal cord and a central hyposignal, extending from C2 to C6.

B, T2-weighted images (TR = 1800 msec; TE = 60 msec) shows a central hypersignal extending from C2 to C6.

C, Right vertebral angiogram shows a dural fistula located at the foramen magnum and fed by the meningeal branch of the vertebral artery.

D, T1-weighted images (TR = 400 msec; TE = 14 msec) performed after surgical clipping of the draining vein shows a decrease of the size of the spinal cord and disappearance of the central hyposignal.



in nine cases. The feeding arteries were: the vertebral artery (nine cases; Figs. 1C and 2C), the occipital artery (eight cases; Figs. 1A and 3B), the middle meningeal artery (five cases; Fig. 3A), the ascending pharyngeal artery (five cases, Fig. 1B), the meningohypophyseal trunk (three cases) and the posterior auricular artery (two cases).

Tentorial fistulas were more often supplied by bilateral pedicles (4/6 cases). Tentorial fistulas were always supplied by meningeal branches of the external carotid artery (Figs. 3A and 3B). The posterior meningeal branch of the vertebral artery and the meningohypophyseal trunk both supplied

three fistulas. The branches of the external carotid supplying the fistulas were the occipital artery (5/6 cases, Fig. 3B), the middle meningeal artery (5/6 cases, Fig. 3A) and the ascending pharyngeal artery (3/6 cases).

Foramen magnum fistulas were supplied by unilateral pedicles. They were always supplied by the posterior meningeal branch of the vertebral artery (Figs. 1C and 2C) and in three cases by branches of the external carotid artery (occipital artery: three cases, Fig. 1A; ascending pharyngeal artery: two cases, Fig. 1B; posterior auricular artery: two cases).

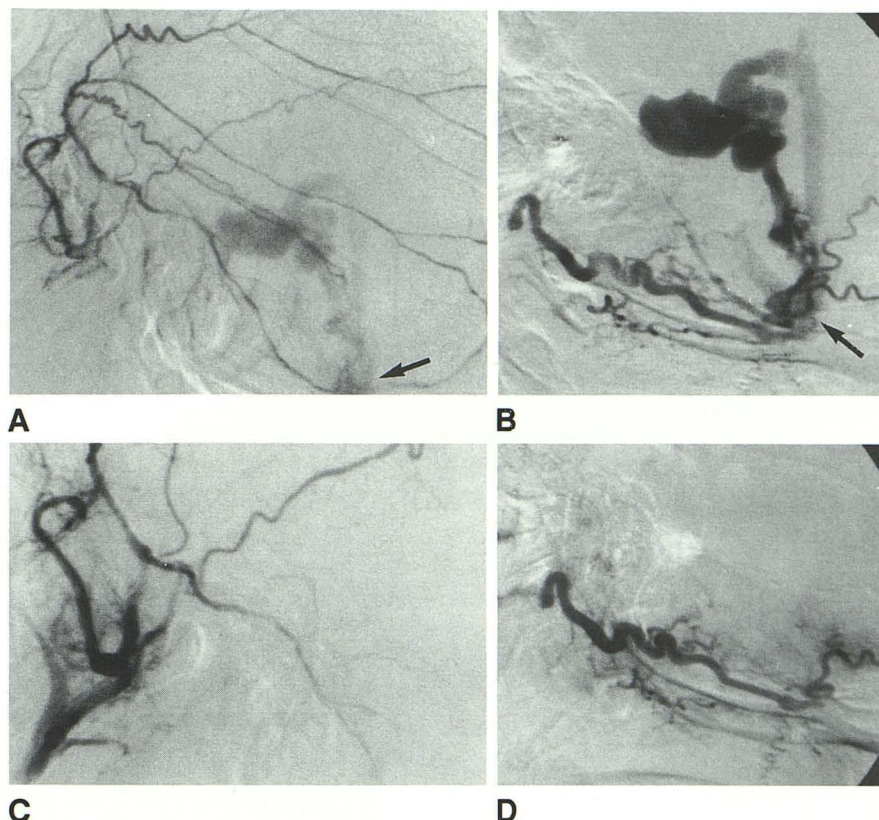


Fig. 3. Case 4.

A, Right internal maxillary angiogram shows a tentorial fistula fed by the middle meningeal artery (arrow).

B, Selective injection of the right occipital artery shows supply to the same fistula (arrow).

C, Control right internal maxillary angiogram performed after particulate embolization shows occlusion of the external branches feeding the fistula.

D, Control right occipital angiogram after particulate embolization shows occlusion of the meningeal branches supplying the fistula.

Venous Drainage (Table 2)

The tentorial fistulas had, in the majority of cases (4/6 cases), a type IV venous drainage. The tentorial fistulas emptied into vermian veins, which joined the vein of Galen or the straight sinus (cases 1, 4, and 5). In case 3, the venous drainage proceeded towards the superior sagittal sinus via temporal veins. In case 6, the venous drainage proceeded towards the anterior and posterior spinal veins. In case 2, the venous drainage was difficult to assess but seemed to be by way of the vein of Galen. The fistulas located at the skull base had a type III venous drainage in the majority of cases (4/6 cases). The draining veins of the skull base fistulas proceeded towards the vein of Galen and the straight sinus via mesencephalic veins (cases 8, 10, 11, and 12; Figs. 1A and 1B) or towards the anterior and posterior spinal veins via a lateral ponto-mesencephalic vein (cases 9; Figs. 2C). In case 7, the venous drainage was difficult to assess.

Treatment and Results (Table 2)

Two patients were treated by transarterial embolization alone. Patient 1 was treated by embolization of the left occipital artery using dura

mater. A follow-up angiogram 1 year later showed complete obliteration of the fistula. Patient 11 was treated by embolization of the external carotid branches feeding the fistula and a detachable balloon was placed in the posterior meningeal branch of the vertebral artery (Fig. 1). The control angiogram showed a decrease of the flow through the fistula. Three months later, subarachnoid hemorrhage recurred and the patient died.

Five patients were treated by surgery alone. In one case (case 2), surgical ligation of the left occipital artery was followed by subtotal surgical excision of the fistula. A few days after surgery, subarachnoid hemorrhage recurred and the patient died. In case 5, the patient initially refused the treatment. His condition dramatically worsened and, 2 months later, CT revealed increasing hydrocephalus. A control angiogram showed partial thrombosis of the venous pouches. Placement of ventriculo-peritoneal shunt was carried out and a surgical excision of the fistula was performed, but he died a few days later. In case 8, surgical excision of the fistula was performed and the postoperative course was uneventful. The patient

remains asymptomatic 1 year after surgery, control angiogram being normal. In case 9, surgical clipping of the draining vein was performed, followed by marked clinical improvement (Fig. 2). Control angiogram performed 6 months after surgery showed complete closure of the fistula. In case 12, clipping of the draining vein was performed with an initial good result.

In three cases, preoperative embolization was performed, followed by surgical excision or clipping of the draining vein. In case 3, embolization with dura mater in both occipital arteries and middle meningeal arteries was performed. Eight days later, surgical ligation of the left ascending pharyngeal artery and removal of the venous pouches were carried out. Control angiogram showed closure of the fistula. In case 4, bilateral feeding arteries provided by external carotid arteries were embolized with dura mater and Ivalon (Figs. 3C and 3D) and 12 days later, surgical excision of the fistula was performed. Follow-up angiogram was normal. In case 6, a first embolization of the left middle meningeal artery was followed by a transient improvement. A control angiogram performed 1 month later showed persistence of the fistula and the left middle meningeal artery was reembolized. One month later, surgical clipping of the draining vein was performed. A rapid worsening of the clinical status of the patient was observed and he died 3 days later in status epilepticus.

Discussion

The pathogenesis of dural fistulas remains unclear. They are considered by most authors to be acquired lesions, following head trauma or thrombophlebitis (4, 5, 10, 24). In our cases, neither head trauma nor thrombophlebitis were documented. DAVFs account for 10% to 15% of the intracranial arteriovenous malformations (1). 50% of the DAVFs are located in the posterior fossa and mainly involve the transverse and sigmoid sinuses (2). Two other groups of posterior fossa DAVFs are less frequent: fistulas located in the tentorium and fistulas located at the skull base near the foramen magnum. DAVFs of the transverse and sigmoid sinuses are, in the majority of the cases, clinically benign, resulting only in intracranial bruits (3, 19, 25). Tentorial and foramen magnum fistulas are marked by a worse prognosis. Indeed, in our series, 67% of these lesions were associated with intracranial hemor-

rhage. In 17%, the lesion was associated with paraparesis related to the presence of perimedullary draining veins. Moreover, 33% of the patients died in spite of treatment. This is related to the type of venous drainage of these fistulas. Indeed, as was previously reported by Picard et al (14), the great majority of the tentorial fistulas drain into subarachnoid veins. All foramen magnum fistulas and the majority of the tentorial fistulas observed in our institution also drained into subarachnoid veins (Figs. 1, 2, and 3). Cortical venous drainage from a DAVF has been shown to be a risk factor for intracranial hemorrhage (5–7). Hemorrhage is observed in 42% to 50% of DAVFs draining into cortical veins (5, 6). For DAVFs of the posterior fossa with subarachnoid venous drainage, the risk of hemorrhage seems to be particularly important. Grisoli et al (20) reported four cases of DAVFs of the posterior fossa draining into subarachnoid veins, all patients presented with intracranial hemorrhage. In our series, hemorrhage was observed in five of six DAVFs with venous drainage of type IV and in three of six cases with type III venous drainage. The type IV DAVFs seems to have a higher risk of bleeding than type III. Four of six fistulas at the skull base and four of six tentorial fistulas were associated with hemorrhage, both having a high risk of bleeding. The occurrence of hemorrhage both near and far from the malformations suggests that the draining veins are most likely responsible for the bleeding episodes (13, 30).

Intracranial DAVF is a rare cause of progressive paraparesis. This clinical presentation was observed in two of our cases (cases 6 and 9; Fig. 2). Eight similar cases have been previously reported (14, 26–28). Myelography often reveals serpentine filling defects and MR of the spinal cord can visualize perimedullary dilated vessels and intramedullary increased signal on T2-weighted images. However, these clinical and radiologic findings are also those of arteriovenous malformations of the spine. For this reason, patients with intracranial DAVFs clinically presenting as myelopathy were often explored by spinal angiography, which is negative. In such circumstances, cerebral angiography should be performed (Fig. 2). The locations of DAVFs with myelopathy include the lateral sinus (26), the petrous area (27), the foramen magnum (28), and the tentorium (14, 27). In our cases, the fistulas were located at the foramen magnum (case 9; Fig. 2C) and the tentorium (case 6). The mecha-

nism of cord dysfunction in these patients is thought to be venous congestion (26–28).

In one case, the fistula was revealed by headaches. Headaches in DAVFs may be due to intracranial hypertension, resulting from the impairment of venous runoff from associated major sinus occlusion and/or from increased venous pressure resulting from high flow into the draining sinuses (19, 29). In our case, CT revealed hydrocephalus. The occurrence of hydrocephalus in DAVFs seems to be rare (25). It could be secondary to meningeal reaction to previous subarachnoid hemorrhage (which was not demonstrated in our case), increased pressure in the venous sinuses resulting in impaired cerebro-spinal fluid absorption, or to midbrain and aqueductal compression by venous structures (30, 31). In our case, the patient initially refused treatment. His clinical condition rapidly worsened, probably because of the occurrence of partial thrombosis of the venous pouches draining the fistula, resulting in increased mass effect and hydrocephalus. In one case, the fistula was revealed by cranial nerve palsies, involving the V and VI nerves. The occurrence of cranial nerve palsies in dural fistulas is often explained by an arterial steal phenomenon (7). However, in our case, the fistula was fed by the meningeal branch of the vertebral artery, and this mechanism seems unlikely. For anatomical reasons, compression of the fifth and sixth nerves by the venous drainage was also unlikely. This palsy was probably related to venous hypertension. The angioarchitecture of the tentorial and foramen magnum fistulas seems to be different. Indeed, tentorial fistulas are more often supplied by bilateral pedicles (4/6 cases); fistulas at the skull base have unilateral pedicles in all cases. Moreover, arterial feeders are not the same. The tentorial fistulas are always supplied by the meningeal branches of the external carotid (Figs. 3A and 3B) and, in half of the cases, by the posterior meningeal branch of the vertebral artery and the meningohypophyseal trunk. The fistulas located at the skull base are always supplied by the posterior meningeal branch of the vertebral artery (Figs. 1C and 2C); external carotid branches supply only half of these fistulas (Figs. 1A and 1B).

DAVFs of the posterior fossa draining into subarachnoid veins are potentially dangerous lesions because they are accompanied in a high percentage of cases by intracranial hemorrhage, which can recur, or by paraparesis, which can

rapidly evolve, leading to tetraparesis. Because of this grave potential, the goal of the treatment is to obtain rapidly a clinical and anatomical cure. Picard et al (14) suggests that the reduction of blood flow in the fistula, without anatomical cure, reduces the risk of hemorrhage. However, it should be observed that in our series, two patients treated by incomplete embolization (case 11, Fig. 1) or partial surgical resection of the fistula (case 2) died due to rebleeding.

Therapeutic methods have included vascular compression (8, 9), transarterial or transvenous embolization (8–18), surgical treatments (19, 20, 25, 29, 32), embolization followed by surgery (8, 21–23). Occipital artery compression can induce a clinical and angiographic cure of dural fistula involving the transverse or sigmoid sinus in 22% of cases (8). This low percentage of cure and the fact that DAVFs of the posterior fossa which drain into subarachnoid veins often have multiple feeders which precludes this treatment for this type of fistula.

The efficacy of transarterial embolization has been clearly established in the treatment of DAVFs of the lateral sinus (8, 10, 11, 13), of the cavernous sinus (9, 10, 12), of the superior sagittal sinus (10, 13, 15) and of the inferior petrosal sinus (18). However, this efficacy must be carefully analyzed. Treatment efficacy has been most often reported on the basis of clinical criteria (8, 9, 11). When angiographic data is available, it should be observed that a complete anatomical cure is obtained in a low percentage of cases (34%) (10). An incomplete anatomical cure is often obtained in fistulas that were partially fed by meningeal branches of the vertebral artery or the internal carotid artery, which are dangerous vessels for embolization. Clinical cure of the fistulas was sometimes obtained with two or three embolizations, the interval between embolizations being from 2 months to 5 years with a mean of 8 months (11). Moreover, late recurrences are not exceptional (11). This problem may be related to the type of embolizing agents used. Ferman et al (11) considered that it was not possible to establish a correlation between the type of embolus and the outcome. Lasjaunias et al (10) and Halbach et al (8, 9) considered that results obtained with particulate materials were less consistent than with cyanoacrylates. In case 1, embolization of the occipital artery with dura mater resulted in the anatomical cure of the fistula with a follow-up of 1 year. In case 11, embolization of

branches of the external carotid artery was not able to cure the fistula, because of the supply provided by the vertebral artery. Catheterization of the posterior meningeal artery was impossible, because it was dysplastic. Embolization with particles or glue was, therefore, impossible to perform. Consequently, a detachable balloon was placed in the initial portion of the posterior meningeal branch (Fig. 1). Surgical removal of the fistula was planned but was not performed. Three months later, subarachnoid hemorrhage recurred and the patient died. Given the treatment goal previously defined, the low percentage of fistulas anatomically cured by embolization (10), the risk of recurrence of the fistula after embolization (11), and our results, it seems that transarterial embolization alone must be reserved for fistulas fed by few pedicles from the external carotid artery.

Transvenous embolization has been proposed in the treatment of dural fistulas involving the cavernous sinus (16), the transverse and sigmoid sinuses (17), and the inferior petrosal sinus (18). Platinum coils were placed into the fistula site. This treatment seems to be inappropriate for the tentorial and foramen magnum DAVFs, given the type of the venous drainage, which makes the positioning of the coils difficult and dangerous and increases the risk of intracranial hemorrhage.

Alternative therapies are surgical. Such treatments include proximal ligation of branches of the external carotid, which fails in most cases to control the lesion because of recruitment of new arterial supply (31, 32). Clipping of draining veins can sometimes cure the fistula (20) (cases 9 and 12; Fig. 2), but could increase the risk of hemorrhage by increasing venous pressure. Complete excision of the malformation results in an anatomical cure of the lesion, but is sometimes complicated by massive intraoperative hemorrhage (13, 19). The most suitable treatment seems to be a combination of both methods, embolization and surgery. Indeed, if embolization alone is not able to definitely cure the fistula, it can reduce blood flow into the lesion and the vascularity of the overlying structures. Subsequent surgery can be performed under better conditions with reduced blood loss. Such a combination of neuroradiologic and neurosurgical intervention has been performed in cases of dural fistulas involving the transverse and sigmoid sinuses (8) and the deep cerebral venous system (21). Complex dural arteriovenous fistulas have also been treated by combined endovascular and

neurosurgical techniques (22, 23). In some cases, preoperative embolization was performed, followed by intraoperative placement of liquid adhesives into the fistula; complete cure was obtained in a majority of cases. In other cases preoperative embolization was followed by surgical excision, with good results.

We performed transarterial embolization followed by surgery in three cases. In one case (case 6), the diagnosis of the lesion was established after some delay. Iterative embolizations were not sufficient to close the fistula and surgical clipping of the draining vein was attempted. A few days later, the patient died in status epilepticus. The mechanism of decline in this patient remains unclear. However, diversion of flow into collateral veins can occur after clipping the vein draining the fistula, with resultant acute intracranial hypertension. Resection of the fistula site is probably a more effective treatment than clipping the draining vein. In the two other cases (cases 3 and 4), embolization followed by surgical excision of the fistula was performed and control angiogram showed anatomical cure of the lesion (Fig. 3).

In conclusion, given the potential seriousness of DAVFs of the posterior fossa draining into cortical veins, the goal of the treatment is to obtain a rapid, complete anatomical cure of the lesion. This can be achieved by transarterial embolization in the cases of DAVFs fed by few pedicles provided by the external carotid. In the other cases, embolization followed by surgery is indicated.

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