

Cranial and spinal dural arteriovenous malformations and fistulas: an update

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Summary

Awareness of a potential arteriovenous fistula is critical for diagnosis of cranial as well as spinal fistulas. The natural history of cranial and spinal dural arteriovenous fistulas has been clarified during the last decade and interdisciplinary therapies have experienced a substantial development recently. The classification of Cognard & Merland is now the most widely accepted one for cranial dural AVF. It is based on the degree of flow reversal in the sinuses and cortical veins and reflects well the natural history of the different lesions and serves as basis for therapeutic indications. Several studies have defined the annual bleeding risk of cranial dural fistulas between 1.8 and 15%, depending on the pattern of venous drainage and initial symptomatology. Surgical, endovascular and radiosurgical methods must be selectively chosen for the treatment. The risk associated with surgical or endovascular treatment of benign fistulas is higher than the risk of eliminating fistulas that have already led to cortical venous reflux. Transvenous endovascular occlusion or surgical disconnection of draining veins is the treatment of first choice for cranial and spinal dAVF with venous flow reversal. Benign cranial dural arteriovenous fistulas are a developing indication for radiosurgery.

Keywords: Dural arteriovenous fistula; classification; microsurgery; embolisation; radiosurgery.

Cranial arteriovenous fistulas

Cranial and spinal arteriovenous shunts correspond to two types of lesions: (1) Direct fistulas (carotid-cavernous, carotid-jugular, vertebro-vertebral) and (2) indirect or dural arteriovenous fistulas (dAVF). Direct arteriovenous fistulas most commonly affect the cavernous sinus. According to the angiographic classification of Barrow *et al.* these direct internal carotid artery-cavernous sinus fistulas (CCF) are classified as a CCF Type A [2]. These direct fistulas are usually the result of a head trauma, with or without clinically evident basilar skull fracture, or penetrating injuries. Rupture of an intracavernous ICA aneurysm can also produce a direct Type A fistula. The other

types of CCF are indirect truly dural arteriovenous fistulas in the dura around the cavernous sinus. Barrow *et al.* described three types of indirect fistulas depending on the dural vascular supply from the ICA and ECA respectively (Types B, C, D).

More generally, intracranial dAVFs represent arteriovenous shunts from a dural arterial supply to a dural venous drainage channel (see Fig. 1). The nidus of arteriovenous shunting is contained within the leaflets of the dura mater, the wall of a dural sinus, the falx, or the tentorium [1]. Unlike pial arteriovenous malformations, dAVFs seldom have a discrete nidus. Instead, they are composed of numerous arteriovenous



Fig 1. (left) MRA of a dural arteriovenous fistula of the confluens sinuum, showing varicose dilatation of the confluens, venous drainage mainly via the transverse sinuses and arterial supply by both carotid systems and the vertebral arteries; (right) arterial and venous phase of external carotid angiography showing multiple dilated scalp arteries, retrograde leptomeningeal venous flow and flow reversal and thrombosis in the superior sagittal sinus. This 18-year-old man presented with intracranial hypertension. He was cured by multiple endovascular and surgical interventions resulting in complete resection of the confluens

microfistulas with thickened dural arteries and dilated draining channels. The feeding arteries are dural arteries and therefore mainly branches of the external carotids system. Most frequent feeders are the occipital, tentorial, and middle meningeal arteries. Dural AVFs occasionally obtain blood also from the internal carotid or vertebral system.

Epidemiology and clinical characteristics

Most frequently the transverse or sigmoid sinus (60%) is affected. The next common sites are the cavernous sinus (15%), the tentorial incisura (10%), and the superior sagittal sinus and dural convexity (10%), followed by fistulas in the anterior and middle cranial fossa (10%).

The natural history of dAVFs is highly variable [16, 17]. Fistulas can be asymptomatic or present with benign or aggressive symptoms. Cognard *et al.* reviewed the symptoms and progression of dAVFs, and correlated the findings with various angiographic patterns [4]. They found that lesions that drain into a sinus with antegrade flow rarely cause aggressive symptoms, such as intracranial hypertension, epilepsy or neurological deficits (see Table 1). They observed an increasing percentage of aggressive symptoms with retrograde flow in the sinus and/or cortical veins, and that the risk of haemorrhage was associated with cortical venous

drainage. Particularly dAVFs with direct cortical venous drainage and venous ectasia were likely to present with aggressive symptoms (97%) and haemorrhage (66%). The bleeding rate of dAVFs was clarified by analysis of major case series only recently. Brown and collaborators defined in 1994 by a prospective non-selected follow-up study the average bleeding risk as 1.8%/year [3]. Satomi and coworkers from Toronto confirmed in 2002 that benign cranial dural arteriovenous fistulas have indeed a good outcome under conservative management with only 2% conversion to cortical drainage during follow-up [23]. Van Dijk and coworkers showed in 2002 that the clinical course of cranial dural arteriovenous fistulas with long-term persistent cortical venous reflux is unfavourable with a 10%/year mortality and a total of 15% annual events [31]. Duffau *et al.* described in 1999 a subgroup of intracranial dural arteriovenous fistulas which is prone to early re-bleeding [7].

Treatment options

When patients present with minor complaints of noise in the head or headaches, often no treatment needs to be offered. However, pulsatile tinnitus, headache or mastoid pain can be so severe that they limit quality of life. In these cases the potential risk and benefit of treatment has to be considered carefully. In patients with progressive disabling symptoms and in those who are at increased risk for haemorrhage according to the angiographic pattern of the dAVF, definite treatment is warranted.

Many treatment strategies for dAVFs have been described in the literature. Accepted treatment options include microsurgery, embolisation and radiosurgery or combinations of these methods [24, 25].

Microneurosurgery

Sundt and Piepgras published the first systematic series regarding surgical treatment of arteriovenous malformations of the lateral and sigmoid dural sinuses [27]. The preferred surgical treatment was complete excision coupled with packing of the sigmoid sinus. The operative approach was illustrated and discussed in detail. Results and complications were reviewed in 27 patients whose symptomatology had progressed under conservative management; 22 of these cases harboured primary lesions and five had recurrences. There were 22 excellent, one good, and two poor re-

Table 1. Cognard & Merland classification of cranial dural arteriovenous fistulas [4, 6]

	Haemodynamic characteristics	Risk of aggressive symptoms	Risk of haemorrhage
Type I	antegrade flow in sinuses and bridging veins	1%	0%
Type II a	retrograde flow in sinus	37%	0%
Type II b	retrograde flow in bridging veins	30%	20%
Type II a+b	retrograde flow in sinus and bridging veins	67%	6%
Type III	direct fistula in bridging vein	76%	40%
Type IV	direct fistula in varicose bridging vein	97%	66%
Type V	spinal venous drainage	100%	46%

sults (both of the latter from blindness that preceded surgery). There were two deaths, both in patients previously operated on with incomplete removal or obliteration of the dAVF by attempted embolisation.

Surgical excision, with or without prior embolisation, remains the most versatile and effective therapeutic option for complete elimination of dAVFs. Simple ligation of feeding vessels produced success rates of only 0 to 8% and can no longer be recommended [15]. It is agreed that curative treatment of dAVFs generally involves excision of the diseased venous segment. However, surgical excision of dAVFs is associated with notable mortality and morbidity, especially when the transverse-sigmoid sinus or the tentorium are involved. Surgical resection of these dAVFs carries the risk of aggravating the underlying venous hypertension. Elimination of the arterial supply has been advocated if the sinus continues to provide a major route of venous drainage. However, arterial occlusion alone usually leads to recurrence. En bloc resection of the diseased sinus along with the nidus can be performed when the sinus is thrombosed or there is prominent collateral venous drainage. Arterialized red draining veins can be taken with impunity, but radical resection may be limited when disease involves major dural sinuses or bridging veins, especially when these venous channels are still patent with antegrade drainage [24].

Apart from formal resection permanent clinical and angiographic cure can be expected by complete obliteration of the venous outflow. Thompson and co-workers as well as Collice *et al.* described interruption of leptomeningeal drainage for cranial dural AV fistulas with pure leptomeningeal drainage [5, 12, 30]. For fistulas with a pure leptomeningeal drainage this mode of therapy has become the method of choice (see Figs. 2 and 3). If only the retrograde leptomeningeal outflow is obliterated in sinus fistulas with secondary cortical reflux (Cognard Type 2 b and 2 a+b) conversion of an aggressive fistula into a benign one can be expected from obliteration of the leptomeningeal drainage [32].

In our opinion, surgery is indicated when embolisation is impossible or results in subtotal occlusion and when life-threatening or debilitating symptoms such as haemorrhage, retrograde venous drainage, intracranial hypertension, focal deficit, or epilepsy are present. Pulsatile tinnitus, headache, and mastoid pain may also indicate the need for therapy. Subtotal occlusion of a fistula by surgery or embolisation alone is not protective against further complications, especially

haemorrhage. The goal of treatment is to achieve a rapid and complete anatomical cure.

Endovascular treatment

Successful endovascular transarterial occlusion of dAVFs has been reported. However, long-term follow-up review of patients often was lacking. If followed long enough, embolised dAVFs have been shown to recanalise [24, 25, 33]. Only for lesions of the cavernous sinus, embolisation was already prior to the advent of transvenous techniques recommended as the primary treatment by almost all authors. The reported success rates are some 60% for transarterial approaches. For lesions in other locations, it is now quite clear that transarterial embolisation may significantly decrease flow through a dAVF, but arterial embolisation is unlikely to result in complete and permanent obliteration of the fistula. Transvenous or combined transvenous-arterial approaches seem to be more efficacious for dAVFs that remain patent after transarterial therapy or that cannot be reached arterially. Halbach and co-workers pioneered transvenous embolisation of dural fistulas involving the transverse and sigmoid sinuses [11]. Today the higher efficacy of transvenous embolisation compared to transarterial embolisation is generally accepted. However, the risks and long-term results of transvenous obliteration are still unknown. Roy and Raymond emphasized that the occlusion must be precise [22]. An erroneous occlusion could reroute the fistulous output and overload an intracerebral vein. Cavernous dural fistulas with a venous access route can be treated by this technique without significant risk of harm. However, dAVFs in other locations must be carefully evaluated to ensure that the embolisation does not interfere with normal venous drainage of the adjacent brain. This is particularly true for low-risk fistulas, in which the brain still uses the involved sinus for venous drainage.

Radiosurgery

Lewis *et al.* reported on nine patients with tentorial dAVFs treated with transarterial embolisation combined with stereotactic radiosurgery. Five of the seven patients who underwent radiosurgery had a residual dAVF after a mean follow-up period of 24 months and one patient suffered transient radiation injury to the brain stem [13]. Link *et al.* developed a treatment strategy that includes radiosurgery followed by partic-

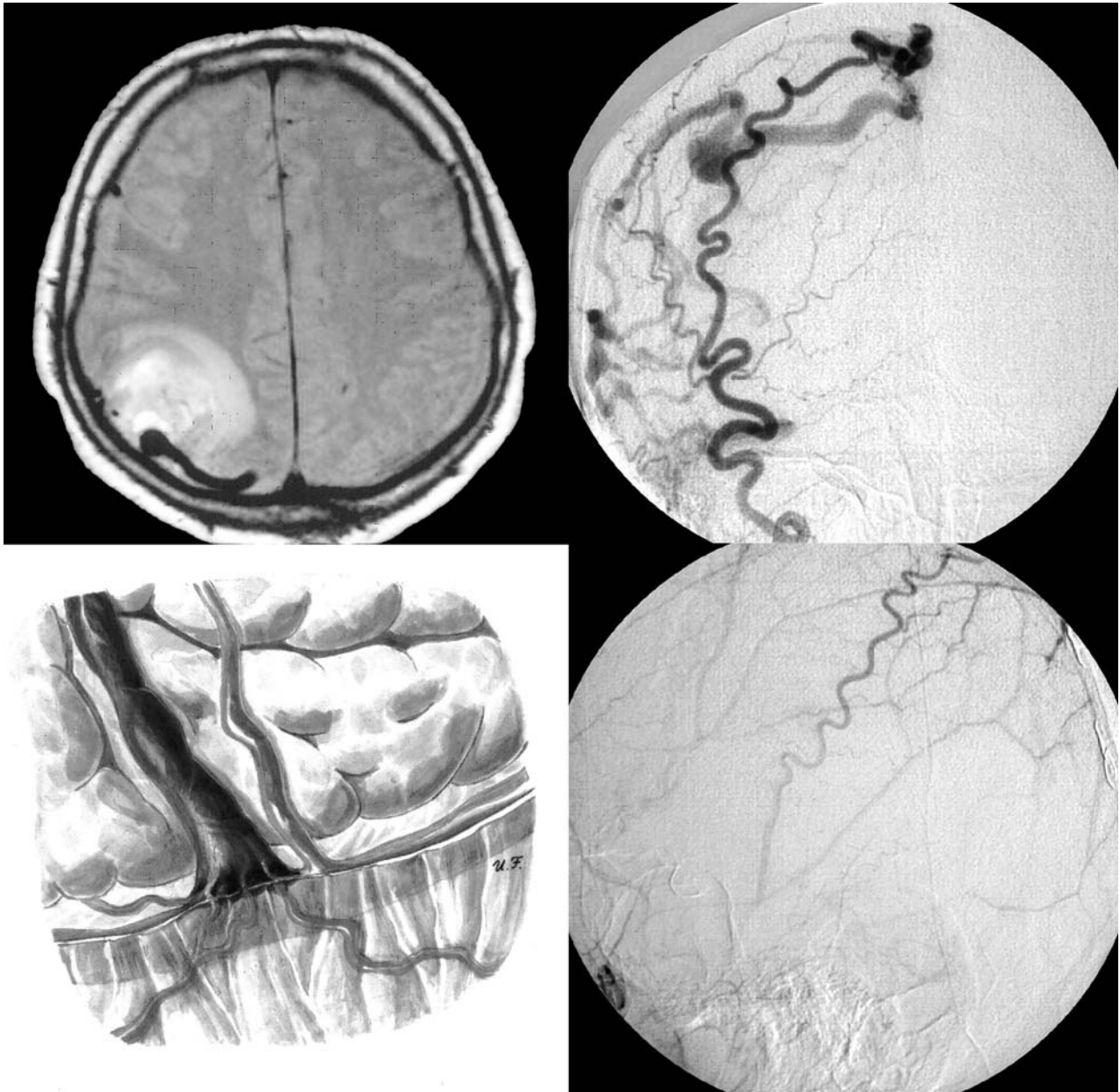


Fig. 2. A 59-year-old man presented with sudden headache and hemianopia. MRI showed a right parietal haemorrhage and a dilated bridging vein. Right external carotid angiography showing direct fistula into a varicose leptomeningeal vein and cortical reflux. Outflow of the leptomeningeal vein into the superior sagittal sinus is thrombosed. Intraoperative view after right parietal craniotomy showing multiple dural arteries entering the leptomeningeal vein. Curative therapy consisted of coagulation and division of this vein. Postoperative right external carotid angiography showing elimination of the fistula

ulate embolisation for selected patients with symptomatic dAVFs who are not good surgical candidates [14]. They treated 29 patients with dAVFs using a Leksell Gamma-Knife unit. Within 2 days after radiosurgery, 17 patients with AVFs that exhibited retrograde venous drainage (12 patients) and/or produced intractable bruit (eight patients) underwent particulate

embolisation of external carotid feeding vessels. Angiography 1 to 3 years posttreatment in 18 patients showed total obliteration of 13 fistulas (72%) and partial obliteration of five (28%). No lesion had bled after treatment. Since these pioneering reports other series have confirmed the high efficacy and the low risk of radiosurgery for dural arteriovenous fistulas [8–10,

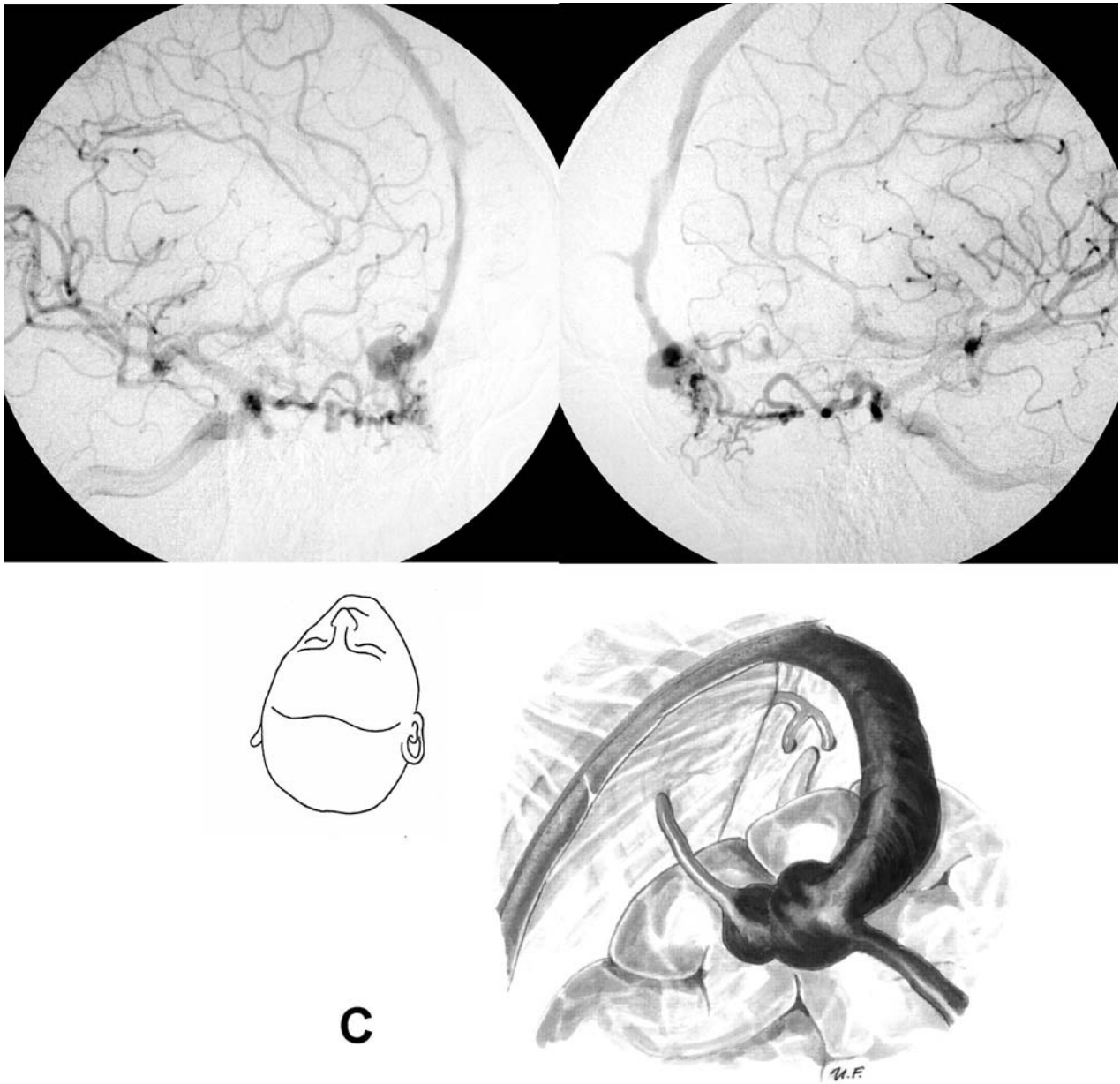


Fig. 3. A 53-year-old man presented with progressive headache. Right and left internal carotid arteriography showed fistula supplied by both anterior ethmoidal arteries and draining into a right frontopolar bridging vein. (C) After right frontal interhemispheric exposure the arterialized retrograde leptomeningeal artery was identified. Disconnection of this vein led to angiographic and clinical cure

20, 21]. The reported cure rates range between 80 and 90%.

Multidisciplinary treatment

The treatment of complex intracranial dAVFs often remains problematic. Combination of all primary treatment modalities must be used with the aim 1) to eliminate venous reflux and 2) to obliterate the fistula completely to prevent recurrence.

Personal experience (Munich/Düsseldorf, 1994–2003)

Our initial experience with treatment of 30 cranial dural fistulas including the transverse and sigmoid sinuses was summarized in 1997 by Olteanu-Nerbe *et al.* [19]. Depending on the venous drainage the fistulas were classified following Cognard's description with 18 patients Type I (main sinus with antegrade flow), 5 Type II a (main sinus with reflux into the contralateral sinus), 5 Type II b (reflux into cortical veins),

1 Type II a+b (both) and 1 of Type III (direct cortical drainage). Bruit, pulsatile tinnitus and headaches were the most common symptoms. 6 patients presented with intracranial haemorrhage, 4 with progressive neurological deficit or seizures and 3 with dementia. Arterial embolisation was performed in all cases except one, where a transvenous approach for balloon occlusion of the transverse sinus was performed. 21 patients were treated by single or repeated embolisation alone. Only in 9/21 cases did arterial embolisation result in complete occlusion of the fistula. In 12/21 patients incomplete occlusion was achieved. Following embolisation 8 patients underwent additional surgery including coagulation of the feeding arteries and arterialized veins, sinus resection and reconstruction of the sinus. Overall, 18 patients were cured, 11 improved and 1 patient was unchanged. There were a total number of 5 complications including transient stroke, transient facial nerve palsy and a small necrotic skin area following embolisation. Venous infarction of the occipital lobe was induced by transvenous occlusion and surgical resection of the transverse sinus in one patient each, respectively.

Following this initial experience we modified the general policy in so far that we limited venous occlusive surgery or transvenous embolisation to patients with venous reflux. Patients with benign fistulas and orthograde flow in the transverse/sigmoid sinus were treated by arterial embolisation and Gamma-Knife radiosurgery. This change of policy could completely eliminate major complications during in the treatment of transverse/sigmoid fistulas treated between 1997 and 2003.

Carotid-cavernous fistulas were treated during the entire period from 1994–2003 almost exclusively by endovascular embolisation. Transarterial and transvenous methods were combined. The results of the treatment of cavernous sinus fistulas were uniformly satisfactory with the only complication of transient worsening of cranial nerve deficits. Recently, we began to indicate Gamma-Knife radiosurgery for some low-flow dural fistulas of the cavernous sinus.

Direct fistulas into leptomeningeal veins (Cognard Type III–V) were almost exclusively approached surgically by interruption of the venous drainage. The more than 30 patients operated on between 1994 and 2003 were uniformly cured and only minor approach related complications had to be accepted, such as transient temporal lobe swelling after subtemporal

approach or anosmia in a case of a fistula in the olfactory groove.

Spinal dural arteriovenous fistulas

Definition and aetiology

Spinal arteriovenous malformations encompass a variety of lesions with abnormal connections between the arterial and venous circulation. The normal high-resistance capillary system is missing. Spinal arteriovenous malformations have been divided by location into dural AVFs (Type I) and intradural arteriovenous malformations (AVM) (Type II–IV).

Type I (dural arteriovenous fistula) is a typical long, single-coiled vessel type of malformation that is almost invariably located on the dorsal pial surface of the spinal cord. In 1977, Kendall and Logue first reported that this type of spinal AVM was actually a true dAVF [25]. They identified AV fistulas in the dural sleeves of spinal nerve roots in nine patients who had radiographic and intraoperative findings consistent with lesions that were previously considered pial venous angiomas. Hence, these lesions were recognized as spinal dAVFs. The spinal dAVF, which is a low-flow shunt located proximally in the dural sleeve of a spinal nerve root and the adjacent spinal dura, is supplied by the dural branch of the intervertebral artery. A perimedullary vein, usually the sole venous outflow from the fistula, then carries shunted arterial blood, retrograde to the normal direction of flow, to the coronal plexus.

Spinal dAVFs can be further classified into lesions with a single arterial feeder (Type I-A) and those with multiple arterial feeders (Type I-B). The multiple feeders in Type I-B can originate from either a single or multiple levels, and they are present unilaterally or bilaterally.

Treatment

It is fortunate that the most common type of spinal AVMs is also the easiest to treat. In the past, the traditional treatment has been excision or stripping of the long, arterialized venous complex. This was a dangerous procedure because it required large exposures and carried the risk of spinal cord contusion or infarct. Furthermore, it is now recognized as unnecessary. The present surgical treatment in most cases consists of excision of the dural fistula, if this is easy to accomplish, or interruption of the arterialized veins that

drain the fistula. Treatment limited to simple interruption of the vein draining the dAVF is an adequate therapy in most patients. It eliminates the underlying pathophysiology – venous hypertension – without risk to normal vessels that supply the cord, and carries a negligible risk of symptomatic recurrence. In patients with spinal dAVFs with only intrathecal perimedullary venous drainage, which includes most patients with these lesions, surgical interruption of the intradural draining vein provides lasting and curative treatment. In patients with both intra- and extradural drainage of the AVF, complete excision of the fistula or interruption of the intra- and extradural venous drainage of the fistula is indicated. In patients in whom a common vessel supplies the spinal cord and the dural AVF, simple surgical interruption of the vein draining the AVF is the treatment of choice, as it provides lasting obliteration of the fistula and it is the only treatment that does not risk arterial occlusion and cord infarction. However, it is important to realise that in Type I-B fistulas multiple arterial feeders can originate from multiple levels and also bilaterally. Since complete obliteration of the fistula is the primary goal of surgery, angiographic documentation of all feeders is critical if surgery is planned.

Several authors reported on long-term surgical results in Type I fistulas. Some 60–70% of the patients improve, 25–30% are stabilized and 5–10% appear to further deteriorate [28, 29]. After it had been recognised that excision of the dilated venous complex is not necessary, and that most patients improve after the shunt is obliterated, some authors have recommended that embolisation of the fistula should be attempted first [26]. However, failure of embolisation is not uncommon [18]. In view of these results, many authors consider surgery as the treatment of choice in spinal dAVFs, because surgery is more effective than endovascular embolisation, which should be reserved for nonsurgical candidates.

Radiosurgery is not an established method for spinal dural fistulas. In analogy with the results on cranial arteriovenous fistulas it appears likely that radiosurgery could be an effective modality. Whether introduction of this modality is useful in the light of the relative simplicity of the surgical method appears doubtful.

Personal results

Since 1994 we defended the strategy of a primary surgical therapy of cervical and thoracic dural fistulas.

These fistulas were only embolised if the patients wished a non-surgical mode of therapy and if there was no spinal cord arterial supply from the feeder artery. Lumbar and sacral fistulas were usually embolised with isobutyl-cyanoacrylate (Histarcryl™). A total of some 40 of these patients was managed between 1994 and 2003. The overall rate of recovery corresponded to the data given in the literature. One third of the patients did not recover satisfactorily. Further deterioration was noticed in one patient. Three patients had to be operated for recanalization after embolisation and one surgical patient had to be re-operated for residual fistula after surgery.

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