Neurological manifestations of intracranial dural arteriovenous malformations

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✓ The authors describe their experience with four cases of dural arteriovenous malformation (AVM) which led them to analyze the clinical aspects of these lesions in an attempt to understand their pathophysiology. An additional 191 previously reported cases of dural AVM's were reviewed with special attention to the mechanism of intradural, central, and peripheral nervous system manifestations. Apart from the peripheral cranial nerve symptoms, which are most likely due to arterial steal, the central nervous system (CNS) symptoms appear to be related to passive venous hypertension and/or congestion. Generalized CNS symptoms can be related to cerebrospinal fluid malabsorption due either to increased pressure in the superior sagittal sinus, to venous sinus thrombosis, or to meningeal reaction resulting from minimal subarachnoid hemorrhages. These phenomena are not related to the anatomical type of venous drainage. On the other hand, focal CNS symptoms are specifically indicative of cortical venous drainage. Seizures, transient ischemic attacks, motor weakness, and brain-stem and cerebellar symptoms can be encountered depending on the territory of the draining vein or veins. Therefore, the localizing value of focal CNS symptomatology relates to the venous territory and not to the nidus or to the arterial supply characteristics of dural AVM's. Furthermore, the venous patterns of various dural AVM's at the base of the skull are expressed by differences in their clinical presentation. Dural AVM's of the floor of the anterior cranial fossa and of the tentorium are almost always drained by the cortical veins and, therefore, have a high risk of intradural bleeding.

The remarkable similarities in the manifestations of dural and brain AVM's and the differences in the manifestations of dural and spinal dural AVM's are pointed out. High-quality angiograms and a multidisciplinary approach to the study of dural AVM's will provide the best understanding of their symptoms and, therefore, the most appropriate treatment strategy.

KEY WORDS · dural lesions · arteriovenous malformation · intracranial hemorrhage

LTHOUGH dural arteriovenous malformations (AVM's) are well known among the neurosurgical community, their symptomatology remains puzzling. The problem is further confused because additional symptoms can be expected if other vascular malformations, such as aneurysms or AVM's of the brain, cutaneous tissue, or bone, are associated with dural AVM's.^{6,15,26,35} The purpose of this paper is to review and analyze the neurological manifestations of dural AVM's and, therefore, to distinguish them from brain AVM's fed by dural arteries and immediate posttraumatic arteriovenous fistulas. Although the findings presented here may provide a better understanding of the pathogenesis of these lesions⁵ and assist in proper diagnosis or therapeutic planning,²⁴ these topics will not be specifically considered in this clinical survey.

Although early case reports appeared in the 1930's, the concept of spontaneous dural fistulas in adults was first introduced in 1951¹¹ and in children in 1964.³⁹ The 1960's were marked by topographic grouping of these lesions, including those in the cavernous sinus,¹⁴ sigmoid and transverse sinuses,^{21,34} and anterior cranial fossa.²⁷ The diagnosis of dural AVM's^{2,16} was also significantly improved due to advances in angiographic techniques,^{7,8,29-31} which led to a better understanding of vascular anatomy and endovascular treatment. Dural AVM's draining into the cortical veins were categorized as a separate entity in 1976,⁴ and were described in detail and classified in 1978.9 Some recent reports have provided a better understanding of dural AVM's located at the floor of the anterior cranial fossa^{17,37} and of the associated intracerebral pressure

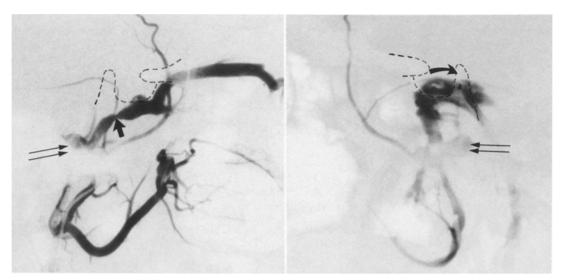


FIG. 1. Selective right middle meningeal arteriograms, lateral view. Left: Case 1. A dural arteriovenous malformation (AVM) can be seen in the right cavernous sinus. Note the arterial ectasia at the level of the petrous branch of the middle meningeal artery (double arrow) and the venous drainage into the cavernous sinus (arrow). There is no evidence of posterior drainage toward the inferior petrosal sinus (compare with Fig. 1 right). The major complaint was proptosis. Right: Case 2. A dural AVM can be seen in the left cavernous sinus. Note that the arterial supply and the arterial ectasia (double arrow) are the same as shown left. However, the major complaint was pulsatile tinnitus. The posterior veins drain into the inferior petrosal sinus (curved arrow) with no ophthalmic vein involvement. This accounts for the difference in symptoms between Cases 1 and 2.

changes.³⁶ A series of posterior dural AVM's in pediatric patients has been carefully analyzed,¹ and new surgical¹² or endovascular therapeutic techniques^{26,40} have been described.

We recently treated four cases that emphasized the need for an understanding of the mechanisms responsible for the symptoms and complications of dural AVM's and of their morphological relationships. These cases are presented briefly. We then summarize our review of 191 cases of dural AVM's that we selected from the literature to find a reliable link in the behavior of these lesions.

Case Reports

Case 1

This 37-year-old woman suffered from a right Bell's palsy during the last trimester of pregnancy. The symptoms regressed spontaneously in a few weeks time. Two years later, she presented with migraine-type headaches and right proptosis, but no pulsatile tinnitus. Angiographic examination showed a dural AVM of the right cavernous sinus fed by the middle meningeal artery (Fig. 1 *left*).

Case 2

This 28-year-old woman presented with a left pulsatile tinnitus during the last trimester of pregnancy. On examination, she had no orbital symptoms, but an objective bruit was noted in the left auricular area. Angiographic examination showed a dural AVM of the left cavernous sinus fed by the middle meningeal artery (Fig. 1 *right*). Following selective embolization with particles, she experienced a left facial nerve paresis which cleared spontaneously in a few hours.

Case 3

This 65-year-old man was admitted with progressive temporal aphasia and temporal disorientation. Neurological examination was unremarkable apart from the presenting symptoms. An average-quality computerized tomography (CT) scan was considered normal. Angiographic examination showed a dural AVM of the right sigmoid sinus draining into the vein of Labbé (Fig. 2).

Case 4

This 52-year-old man was admitted with the sudden onset of severe occipital headache followed by vomiting and collapse. Physical examination revealed a stiff neck. His level of consciousness was moderately altered, but no motor deficit was noted. A CT scan confirmed subarachnoid hemorrhage (SAH) in the basal cisterns with an enhancing vascular-like picture in the area of the left cerebellopontine angle. Angiographic studies showed a dural AVM of the tentorium draining into the petrous vein and later into the basal vein of Rosenthal (Fig. 3).

Analysis of Cases

We reviewed 191 cases of dural AVM selected from the literature. Significant clinical findings are summa-

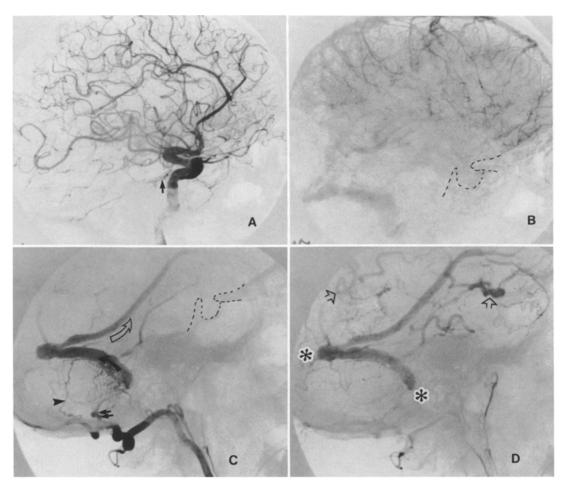


FIG. 2. Case 3. Selective internal carotid (A and B) and occipital (C and D) angiograms in the early (A and C) and late (B and D) phases. A dural arteriovenous malformation (AVM) of the transverse sinus is shown, fed by the basal tentorial artery (*arrow*) and mastoid artery (*double arrow*) and a transosseous branch (*open curved arrow*). Although the primary drainage route of the dural AVM is via the sinus (*black curved arrow*), proximal and distal thrombosis of the sinus (*asterisks*) causes rerouting of the venous outflow toward the cerebral veins (*open arrows*) into the vein of Labbé. Note that at the late phase of the internal carotid arteriogram (B) the remaining venous drainage of the brain is rerouted toward the superior longitudinal sinus and the opposite transverse and sigmoid sinus.

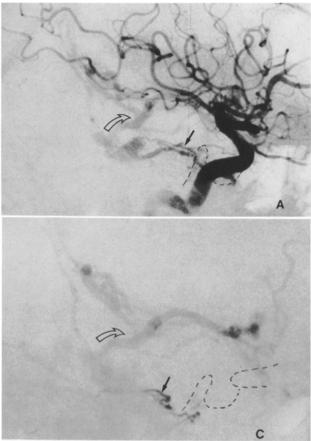
rized in Table 1. Since the explanation for these findings is often unclear in the literature, and sometimes even controversial, we have tried to analyze the cases based on their anatomical relationships (arterial, nidus, and venous), each of which can theoretically be responsible for specific clinical manifestations.

Artery-Related Symptoms

An arterial steal phenomenon has been proposed as a cause of central nervous system (CNS) manifestations. However, most dural AVM's have a slow flow with no angiographic evidence of steal. Selective angiographic studies further demonstrate that the internal carotid or vertebral supply is insignificant, and therefore dural AVM's cannot be considered to have any arterial hemodynamic consequences for the CNS. On the other hand, the subject of meningeal arterial steal has always been neglected, although dural arteries are known to

supply the cranial nerves:²⁵ one of our patients (Case 1) presented with a facial nerve palsy apparently caused by steal from the meningeal artery supplying a middle cranial fossa dural AVM. In addition, cranial nerve symptoms that are either corrected or produced during selective embolization procedures at the base of the skull, as in our Case 2, support the concept of a dural arterial steal mechanism. However, some authors have proposed that venous occlusive factors are responsible for the cranial nerve manifestations in cases of spontaneous cavernous sinus dural AVM's.^{3,40} We believe that, in this particular location, cranial nerve involvement may be due to either or both mechanisms. The most satisfactory explanation of cranial nerve manifestations of dural AVM's in other areas seems to be the dural steal mechanism. These findings correlate with our recent analysis of the ischemic syndrome of the cranial nerves.23

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Nidus- and Vein-Related Symptoms

Two angiographic types of spontaneous dural AVM were identified: the single-feeder arteriovenous fistula and the complex arterial network with multiple arteriovenous shunts. With both types, pulsatile tinnitus is partially related to the nidus. However, this symptom is more commonly associated with some locations in the brain than with others (Table 1), and it appears that the tinnitus is perceived whenever the drainage involves a dural sinus in direct contact with the petrous pyramid (Fig. 1 *right*). The higher the flow in the nidus and the shunt, the more intense the tinnitus.

Venous High Flow. Forty percent of cases with pulsatile tinnitus have objective bruits.²⁶ The bruit is found in most pediatric patients,¹ presumably because this population is disposed to high-flow lesions. Although the objective bruit is important, it may not be perceived by the child as an abnormality if it has been present since birth.²⁶ In this age group, high-flow lesions may lead to cardiac complications and ultimately to death.¹ These lesions often produce intracranial mass effect due to secondary dural venous lakes.^{1,13,39} In comparison to the well known relationship between vein of Galen aneurysms and brain AVM's, torcular ectasia must be considered to be the consequence of an adjacent dural AVM, with the same potential systemic

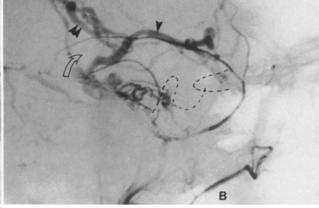


FIG. 3. Case 4. Selective angiograms, lateral projection, of the internal carotid (A), distal external carotid (B), and the ascending pharyngeal (C) arteries. A tentorial arteriovenous malformation (AVM) (at the medial one-third of the tentorium) is demonstrated on the left side, with middle meningeal, ascending pharyngeal, and internal carotid arterial supply (arrows). The dural AVM is draining via the petrosal and lateral mesencephalic veins into the supratentorial cortical veins (open curved arrows). Note the late filling of the temporal vein (arrowhead) and basal vein of Rosenthal (double arrowhead).

complications. In addition, these high-flow lesions may produce increased intracranial pressure (ICP), communicating hydrocephalus, and secondary macrocephaly.⁷ These chronic effects are thought to be due to long-standing high pressure in the superior sagittal sinus. Similar manifestations have been observed in the adult population, with resulting papilledema and secondary visual atrophy.^{20,36} Two factors have been proposed to explain these symptoms: 1) chronic disturbance of cerebrospinal fluid absorption³⁶ due to venous sinus hypertension,²² and 2) repeated episodes of minimal SAH with subsequent meningeal reaction.¹⁹ The former mechanism is the more frequent, since the latter depends specifically on intradural drainage (see below).

Venous Thrombosis. While the previous explanations for increased ICP may be sufficient by themselves, secondary sigmoid sinus thrombosis may create an additional limitation on venous outflow, leading to a further ICP increase.³⁶ On the other hand, it is interesting that, although partial cavernous sinus thrombosis has been frequently³ or constantly⁴⁰ observed in cavernous sinus dural AVM's, increased ICP has never been reported. In fact, thrombosis of the cavernous sinus does not compromise the craniofugal drainage of a dural AVM as is true of sigmoid thrombosis distal to a transverse sinus dural AVM (see our Case 3). Venous thrombosis may even modify the type of drainage of a given dural AVM and, therefore, affect its clinical behavior (see below). Appreciation of the venous disposition requires knowledge of the anatomical variation of the sinus in these areas.¹⁰ In 1974, in a discussion of

TABLE 1

Location and major symptoms in 191 cases of spontaneous dural AVM's reported symptoms in the literature*

	Series		Major Symptoms†		
Location	Reference Nos.	Total Cases	Туре	No.	%
anterior fossa	17, 26, 37	19	intradural bleeding	16	84
			SAH	10	
			ICH	8	
			SDH	4	
cavernous	16, 26, 40	36	intradural bleeding	0	0
sinus			bruit	15	42
			visual deficits	10	28
			proptosis	30	83
			CNS	1	3
			PNS	16	44
tentorium	12, 26	7	intradural bleeding	5	71
			SAH	4	
			ICH	3	
			SDH	1	
			CNS	3	43
			PNS	1	14
lateral sinus,	25, 32, 36	129	intradural bleeding	20	15
sigmoid			bruit	90	70
sinus, tor-			visual deficits	15	12
cular			papilledema	29	22
			headache	59	46
			CNS	17	13
			PNS	9	7

* AVM = arteriovenous malformation; SAH = subarachnoid hemorrahge; ICH = intracerebral hematoma; SDH = subdural hematoma; CNS = focal central nervous system symptoms; PNS = peripheral nervous system deficits. In the large series selected, no bleeding was found; however, intracerebral hematomas have been observed to be indicative of dural AVM's of the cavernous sinus.

† Numbers and percentages of manifestations are based on the total cases at each location.

the arterial steal mechanism of focal CNS symptoms, Kosnik, *et al.*,¹⁹ stated: "Flow problems in these patients are created more by the increased venous pressure and consequent passive congestion of the brain than by arterial steal."

Venous Ischemia. Besides the general effects mentioned above, focal CNS symptoms are encountered exclusively in dural AVM's draining to the cortical veins.^{4,26} Depending on the topography of the vein involved, different manifestations such as aphasia, motor weakness, transient ischemic attacks, and seizures can be anticipated, as shown by Miyasaka, *et al.*,²⁸ and in our Case 3. These symptoms appear to be related to chronic passive congestion due to retrograde increased venous pressure toward the venous drainage routes of the normal brain.^{4,19,26,36}

Venous Mass Effect. It has been suggested that the CNS can be mechanically compressed by the venous outlet of an AVM.²⁰ Although it is conceivable that a large venous lake may produce a mass effect,^{4,27,39} an engorged draining vein from a dural AVM is unlikely to cause mechanical compression. However, we have noted a visual field defect in a patient with an enlarged

olfactory vein and basal vein of Rosenthal draining a dural AVM located at the floor of the anterior cranial fossa.²⁶ In this patient, no angiographic evidence of venous hemodynamic constraint could be demonstrated. Therefore, mechanical compression of the visual pathways seemed to be the most satisfactory explanation. A mechanical phenomenon may also be responsible for the pseudo-extraocular motor nerve palsies in cases of cavernous dural AVM's draining into the orbital venous system. In such cases, proptosis and muscle tension could be the cause of diplopia and poor movement of the eyeballs. It is worth noting that a carotid-cavernous fistula may produce "paradoxical" symptoms in the contralateral orbit if ipsilateral cavernous sinus thrombosis reroutes the venous drainage.²⁶

Venous Rupture. Another complication of dural AVM drainage into the cortical veins is intradural hemorrhage.^{4,26} Depending on the type of hemorrhage, specific focal CNS manifestations or sequelae may result. In 1975, Obrador, et al., 32 reported that 20% of dural AVM's were associated with intradural bleeding. but no attention was paid to whether or not cortical venous drainage was present. One year later, Castaigne, et al.,4 reported that 42% of dural AVM's that drained into cortical veins were complicated by intradural hemorrhage. Dural AVM's in certain locations almost always drain into intradural veins such as those of the anterior cranial fossa^{17,37} and tentorium,¹² and thus are associated with a high frequency of bleeding episodes. This was true in our Case 4. Many types of intradural bleeding may be encountered, including SAH, subdural hematoma, and even intracerebral hematoma (Table 1). In a recent case,¹² a hemorrhagic complication occurred twice in a 12-day period. In another of our series,²⁶ only one of three patients with tentorial AVM's presented with hemorrhage: the other two lesions were found incidentally during angiography. Dural AVM's draining into the transverse sinus or middle cranial fossa do not bleed unless they drain into the cortical veins.4,38

Combined Venous Mechanisms. Episodic headaches may result from minor bleeding,³⁶ but hemorrhagic complications may be overlooked and their frequency underestimated. Secondary thrombosis of the venous outlet of a pure sinus dural AVM may reroute drainage into the cortical veins, potentially leading to focal CNS symptoms and hemorrhagic complications. The disappearance of a pulsatile tinnitus preceding the development of a CNS manifestation implies transformation of the draining modality.⁴ However, thrombosis may also represent one of the natural causes of the socalled "spontaneous cures" of dural AVM's. This regression has also been reported following bleeding.^{18,33} To produce spontaneous regression, the thrombosis probably must occur within the nidus and not in the venous outlet. Unfortunately, there is no angiographic pattern that allows us to predict which dural AVM will regress or transform into a more dangerous type. In the

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TABLE 2
Location of dural AVM's related to frequency of common types of drainage*

Leasting of ANNA	Frequency of Drainage Types			
Location of AVM	Sinus	Cortical		
cavernous sinus sigmoid sinus superior sagittal sinus transverse sinus torcular anterior cranial fossa tentorium	almost constant almost constant depending on type depending on type not described rare	rare rare depending on type depending on type depending on type almost constant almost constant		

* AVM = arteriovenous malformation. For a pictorial representation of the intracranial locations of dural AVM's and the routes of venous drainage see Fig. 4.

TABLE 3

Major symptoms of dural AVM's related to common types of venous drainage*

Sinus Venous Drainage	Cortical Venous Drainage	
proptosis cranial nerve palsy visual deficits ICP changes bruits	intradural bleeding focal CNS deficits seizures or other transient symptoms	

* AVM = arteriovenous malformation; ICP = intracranial pressure; CNS = central nervous system.

review by Albright, *et al.*,¹ of dural AVM's in children, no instances of spontaneous regression were described. This observation further suggests that slow-flow dural AVM's are more likely to regress spontaneously. However, we failed to find a satisfactory explanation for the absence of spontaneous regression of dural AVM's with cortical venous drainage.

Discussion

The symptoms presented above are not of equal clinical importance. With the exception of cranial nerve involvement, all symptoms are related to the venous character of the dural AVM (Tables 2 and 3). However, the potential of a dural AVM to bleed depends on the type of venous drainage, and theoretically all dural AVM's can drain into the cortical venous system (and bleed). Bleeding will occur more often at some locations than others, since the only venous outlet for a dural AVM is a cortical vein (Table 2 and Fig. 4). It is interesting that no hemorrhage has been reported in the epidural space, regardless of the venous drainage pattern. As already pointed out,^{1,4} there is a remarkable similarity between the intradural symptoms of dural AVM's with cortical venous drainage and brain AVM's. However, neither the venous drainage of a brain AVM and its hemodynamic effect on the healthy brain nor its potential responsibility for neurological and hemorrhagic manifestations have been evaluated recently.

In comparison, spinal dural AVM's draining into the

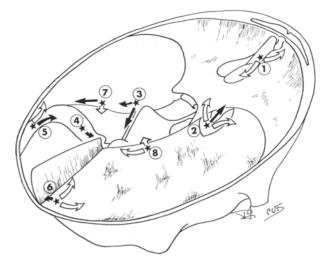


FIG. 4. Schematic representation of the most common locations of dural arteriovenous malformations (asterisks), with their possible routes of venous drainage. Black arrows correspond to sinus drainage and open arrows to cortical (or intradural) venous drainage. 1 = Anterior cranial fossa: frontal and olfactory veins. 2 = Anterior cavernous sinus: ophthalmic vein, Breschet's sinus, and deep sylvian vein. 3 = Posterior cavernous sinus: superior and inferior petrosal sinuses. 4 = Sigmoid sinus: sigmoid sinus and internal jugular vein. 5 = Transverse sinus: sigmoid sinus and temporal veins (vein of Labbé). 6 = Torcular: medial occipital and infratemporal veins. 8 = Marginal tentorium: superior petrosal sinus and petrous vein. 8 = Marginal tentorium: tentorial veins, vein of Rosenthal, and lateral mesencephalic veins.

spinal cord venous system are characterized by the same type of passive retrograde venous congestion as are dural AVM's draining into the cortical venous system. However, normal venous drainage of the cord allows for slow progressive transmission of the retrograde venous pressure, leading to possible dilatation of the entire medullary venous system. This could, therefore, explain the absence of intradural bleeding complications with this type of spinal dural AVM.

Thus, to understand the symptomatology of dural AVM's, proper analysis of the venous pattern is mandatory. Although the arterial feeders are of relatively minor clinical significance, their role in therapeutic strategy requires detailed angiographic studies which should be conducted by a skilled neuroradiologist. While patients can be referred by otolaryngologists, neurologists, neurosurgeons, ophthalmologists, or pediatricians, diagnostic neuroradiology constitutes an essential link in the multidisciplinary approach to investigation and treatment of these interesting, uncommon, and ill-understood lesions.

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