MR Detection of Dilated Deep Medullary Veins in Intracranial Dural Arteriovenous Fistulas with retrograde Leptomeningeal Venous Drainage

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Summary

Patients with dural arteriovenous fistula (DAVF) are at higher risk of developing neurological deficits when there is retrograde leptomeningeal venous drainage. Our aim is to demonstrate the presence of dilated deep medullary veins in the brain on magnetic resonance imaging (MR) in this group of patients, and to assess their clinical significance. Nine patients with angiographically proven DAVF associated with leptomeningeal venous drainage who had MR before treatment were studied. MR was performed in at least two orthogonal planes before and after gadolinium administration. The dural fistula was located at the cavernous sinus in five patients, at the transverse-sigmoid sinus in three and at the tentorium in one. Dilated deep medullary veins were noted in six patients. Of these, four showed parenchymal abnormalities which included intracerebral haematoma, venous infarction, brain oedema and T2 hyperintensity in brainstem. Venous varix was present in one patient. No neurological complication or parenchymal change was observed in the three patients without dilated deep medullary veins. Therefore, in patients with intracranial DAVF associated with leptomeningeal venous recruitment, the MR finding of dilated deep medullary veins suggests a more severe degree of venous

hypertension and congestion in the brain. This subgroup of patients has a much higher chance of neurological complications and warrants urgent intervention.

Introduction

Intracranial dural arteriovenous fistulas (DAVFs) are abnormal vascular connections located within the walls of a dural sinus or an adjacent cortical vein¹. While many of the patients have a benign clinical course, others may suffer from severe neurological complications and have a debilitating outcome. Retrograde leptomeningeal venous drainage is a dominant angiographic feature associated with neurological complications. Whereas it has been shown that dilated leptomeningeal veins associated with DAVFs can be detected by magnetic resonance imaging (MR)², the appearance of dilated deep medullary veins on MR in DAVFs and its clinical significance have not received wide discussion. We have observed this feature in some of our patients presenting DAVFs with retrograde leptomeningeal venous drainage. Our purpose is to demonstrate dilated deep medullary veins within the brain on MR in this group of patients and to try to correlate with the clinical severity of DAVFs.

Figure 1 Patient 7, left transverse-sigmoid sinus DAVF with left temporal lobe venous infarction. A) Axial T1-weighted spin-echo MR image (560/15) at the level of transverse sinus shows thrombosis of the left transverse sinus. B,C) Axial T1- and turbo spin-echo T2-weighted (4000/90) images reveal acute venous infarction of the left temporal lobe with extensive white matter edema and parenchymal haematoma. Note that there are no discernible vessels within the white matter. D,E) Axial contrast-enhanced T1-weighted images (560/17) demonstrate dilated pial veins over the left temporal lobe in D, parenchymal enhancement and dilated deep medullary veins (arrowheads) traversing the white matter towards the ventricular wall in E. F) Lateral projection left external carotid angiogram shows a DAVF involving the thrombosed left transverse-sigmoid sinus with retrograde drainage in the vein of Labbé (arrow).

Patients and Methods

From July 1998 to June 2001, 45 patients were referred to our institution for angiography and diagnosed to have intracranial DAVF. Fourteen patients had associated leptomeningeal venous drainage by angiography. Nine out of these 14 patients had MR imaging before treatment and were selected for this study. The clinical records were reviewed by a neurosurgeon.

Selective angiograms were obtained in two orthogonal planes. MR imaging was performed on a 1.0 T imager (Magnetom Expert; Siemens Medical Systems, Erlangen, Germany) using a standard quadrature head coil. Standard pre-contrast MR imaging consisted of axial T1 and T2-weighted images of 5 mm slice thickness, and sagittal T1-weighted images.

Contrast enhanced T1-weighted images were also obtained in all patients with gadopentetate dimeglumine (0.1 mmol/kg) given intravenously. Imaging was performed in both axial and coronal planes.

The angiograms and MR images were interpreted by two independent experienced neuroradiologists.

The following MR findings were noted: dilated leptomeningeal veins, deep medullary veins, and parenchymal abnormalities. Dilated deep

medullary veins were recognised as linear vessels traversing the deep white matter of the brain towards the wall of a ventricle. Angiographic features recorded included the sites of fistulas, the leptomeningeal venous drainage pathways, and the presence of venous outflow obstruction. The distribution of the dilated venous structures and parenchymal abnormalities seen on MR were correlated with the retrograde venous drainage pattern denoted on angiograms.

Results

The clinical features and radiological findings of the patients are summarised in table 1. There were three men and six women ranging in age from 39 to 79 years, with a mean age of 65 years.

The clinical presentations were chemosis and proptosis ($n = 5$), decreased vision ($n = 3$), ophthalmoplegia ($n = 2$), headache ($n = 1$), homonymous hemianopia ($n = 1$), impaired cognitive function ($n = 1$), and tinnitus ($n = 1$).

Angiography showed that the DAVFs were located at the cavernous sinuses ($n = 5$), at the transverse-sigmoid sinus ($n = 3$) and at the tentorium ($n = 1$). All of the carotid cavernous fistulas were of the indirect type with arterial sup-

ply from the dural branches of the carotid system.

The arterialised blood drained into the Sylvian vein ($n = 5$), other leptomeningeal veins ($n = 6$), superior petrosal sinus ($n = 2$), and pontomesencephalic vein ($n = 1$). Five patients had occlusion at the antegrade venous drainage pathway.

On MR, dilated leptomeningeal veins were identified in all patients. Dilated deep medullary veins were noted within the underlying brain in six patients (figure 1). In four of these six patients (cases 1, 4, 5 and 7), the antegrade venous drainage was obstructed and there were parenchymal abnormalities. Parenchymal changes included intracerebral haematoma (case 1), venous infarction (case 7), brain oedema (case 4) and T2 hyperintensity in the brainstem (case 5) (figure 2). One patient (case 5) had a venous varix over the cerebral convexity.

The locations of the venous dilatation and brain pathology corresponded closely to the routes of retrograde venous drainage demonstrated angiographically in most patients. Two patients having transverse-sigmoid sinus DAVF (cases 1 and 7) with recruitment of the vein of Labbé had dilated pial and deep medullary veins as well as intracerebral haemorrhage and venous infarction of the ipsilateral temporal lobe (figure 1).

In one patient (case 4) with transverse-sigmoid sinus DAVF draining to a cortical vein over the ipsilateral occipital lobe, MR depicted dilated pial and deep medullary veins of the edematous occipital lobe. Two patients (cases 1 and 2) with fistulas draining to the superior petrosal sinus showed dilated pial veins of the cerebellum. In five patients with DAVF involving the cavernous sinus and Sylvian vein recruitment (cases 2, 3, 5, 6 and 8), dilated pial veins were noted in the Sylvian fissure or over the adjacent brain in four patients and dilated deep medullary veins in frontoparietal regions of the brain in two. The venous drainage pattern of the last patient with DAVF of the tentorium was more complicated. In addition to drainage to the vein of Galen, basal vein of Rosenthal and dural veins within the posterior falx depicted on angiogram, MR also demonstrated dilated deep medullary veins in the periventricular white matter of both cerebral hemispheres.

Discussion

Intracranial DAVFs are uncommon clinical conditions, accounting for about 10-15% of all intracranial vascular malformations³. The clinical presentation varies from mild and non-specific symptoms to acute and even life-threatening cerebrovascular accident. The symptomatology and pathophysiology of DAVFs are closely related to the site of involvement of dural sinus(es) as well as the venous drainage pattern⁴⁻⁸.

While many of the patients have a benign clinical course, others may present with aggressive symptomatology and have a poor clinical outcome. Treatment of these lesions, either endovascularly or surgically, can be a challenging task and is not without risks. Therefore, it is recommended that most benign lesions should be treated conservatively and treatment is directed for symptom relief⁹. On the other hand, it is crucial to identify those patients who are at risk of neurological complications and thus demand more aggressive treatment. Currently, a number of classification systems have been advocated aiming at stratifying the aggressiveness of DAVFs¹⁰⁻¹³. In essence, these grading systems are based on the angiographic picture of the venous drainage pattern of the fistulas. Fistulas showing retrograde leptomeningeal venous drainage are considered as aggressive type and are associated with high risk of neurological complications^{1,5-7,14,15}. In fact, Castaigne et al reported that up to 42% of fistulas with cortical venous drainage were associated with haemorrhage. In the series of Malek et al of more than 90 patients with transverse sinus DAVFs, more than 33% of the cases were complicated by haemorrhage.

Although angiography still remains the gold standard for analysing the angioarchitecture of DAVFs, it is an invasive procedure not without inherent risk. Previous reports have shown that MR is useful for the evaluation of DAVF, especially in detecting dilated leptomeningeal veins. De Marco et al reviewed their series of 12 patients with DAVFs. They found that eight patients showed dilated cortical veins on MR due to retrograde venous drainage. MR also readily demonstrated associated complications including cerebral infarction and intracranial haemorrhage which might not be apparent on angiography. In Willinsky's series of 13 patients with DAVFs associated with retrograde venous

drainage, MR depicted dilated cortical veins in ten¹⁶. Four patients presented with neurological deficits.

While the interest of these reports focuses on the involvement of leptomeningeal veins, in our literature search there is a paucity of articles describing the MR appearance of dilated deep medullary veins within the brain in this group of patients.

Medullary veins are intracerebral veins

which are located within the white matter of the cerebral hemispheres. The radiological anatomy of these veins has been described in the literature^{17,18}. Depending on the location and direction of blood flow, medullary veins are divided into superficial and deep groups. There is a third group of veins that traverse the whole thickness of the cerebral hemisphere from the cortical surface down to the ependymal lining of the lateral ventricles. These are termed tran-

Table 1 Clinical presentations and imaging findings of patients with DAVFs

Case No.	Sex, age	Symptom/sign	Angiography:			MR: dilated leptomeningeal/medullary v	MR: parenchymal changes
			fistula site	venous drainage	Status of venous outflow		
1	F, 69	Headache	R TSS	R SPS, v of Labbé	Occluded R TSS	R temporal pial v, R cerebellar v, R temporal medullary	v R temporal ICH
2	F, 75	L chemosis	Bil CS	R SPS, R cerebellar v,	Stenotic R IPS,	R cerebellar v	None
3	F, 65	Bil chemosis, impaired L visual acuity	Bil CS	L Sylvian v, R Sylvian v	occluded L IPS Patent bil IPS	R Sylvian fissure v	None
4	M, 60	L homonymous hemianopia	R TSS	R occipital cortical v	Occluded R TSS	R occipital pial and medullary v	R occipital lobe edema
5	F, 68	Bil chemosis, impaired visual acuity	Bil CS	L Sylvian v, APMV	Occluded bil IPS	L Sylvian fissure v, varix, L frontoparietal medullary v	T2 hyperintensity in brainstem
6	F, 79	Bil chemosis, R 6 th nerve palsy	Bil CS	Bil SOV, L Sylvian v, BVR	Patent bil IPS	L Sylvian fissure v	None
7	M, 77	Impaired cognitive function	L TSS	L v of Labbé	Occluded L TSS	L temporal pial and medullary v	L temporal venous infarct
8	F, 54	R chemosis, R 3 rd nerve palsy, impaired visual acuity	R CS	R SOV, R Sylvian v	Patent R IPS	R Sylvian fissure v, R frontoparietal medullary v	None
9	M, 39	L tinnitus	Tentorium	V of Galen, BVR, dural v in posterior falx	Patent straight sinus	V of Galen, BVR, posterior interhemispheric v, bil parietal medullary v	None

R, right; L, left; v, vein(s); TSS, transverse-sigmoid sinus; ICH, intracerebral haematoma; bil, bilateral; CS, cavernous sinus; SPS, superior petrosal sinus; IPS, inferior petrosal sinus; APMV, anterior pontomesencephalic vein; SOV, superior ophthalmic vein; BVR, basal vein of Rosenthal and v, vein

Figure 2 Patient 5, bilateral cavernous sinus DAVF. A) Axial turbo spin-echo T2-weighted image (4000/90) shows a flow void adjacent to the dilated left cavernous sinus and a prominent Sylvian vein (arrowhead). Note the hyperintense signal in the brainstem. B,C) Contrast-enhanced T1-weighted axial (560/17) and coronal (532/17) images show dilated deep medullary veins in the cerebral white matter running towards the lateral ventricle. Note the relatively straight course and uniform caliber of the vessels. Dilated pial veins are also evident within the cerebral sulci. D) Arterial phase postero-anterior projection right common carotid angiogram confirms cavernous sinus DAVF with predominant shunting to left cavernous sinus and Sylvian vein. E) Late phase lateral projection right common carotid angiogram reveals retrograde venous drainage to left Sylvian, pontomesencephalic and spinal perimedullary veins (arrow). Note the varix (arrowhead) over the frontal region.

scerebral veins. The medullary venous system serves to drain the white matter of the brain, whereas the transcerebral anastomotic veins may be concerned with the regulation of reabsorption of the cerebrospinal fluid¹⁹.

Superficial medullary veins are short and small. They are difficult to recognise on angiogram¹⁷. Deep medullary veins are larger and longer. They originate 1 to 2 cm below the cortical gray matter and run deeply through the

deep white matter. They converge towards the lateral ventricle, draining into subependymal veins that course along the ventricular wall. In situations where they become dilated, deep medullary veins can be recognised on imaging as vascular structures that are arranged in a wedge-shaped pattern in the deep white matter of the brain. They run a straight course towards the ventricle and change relatively little in caliber.

Deep medullary veins may become dilated in the following situations:

- 1) developmental anomaly^{18,20},
- 2) increased blood flow and venous drainage through the brain¹⁷,
- 3) venous congestion or development of collateral venous drainage^{21,22}.

In our series, dilated leptomeningeal veins could be identified on MR in all nine patients, whereas dilated deep medullary veins were noted in six.

The deep medullary veins were readily recognised on contrast-enhanced T1-weighted images as enhancing linear structures traversing the deep white matter of the brain towards the lateral ventricular wall. They may not be seen on pre-contrast images, since they are small and the flow is slow. In four out of these six patients, the drainage pathway of the dis-

eased dural sinuses was obstructed. All four patients had brain parenchymal abnormalities shown on MR: venous infarction, intracerebral haematoma, brain and brainstem edema. The brain parenchymal changes and neurological complications were most likely related to venous hypertension and congestion as a result of the incoming arterialised blood being diverted into the leptomeningeal veins. Although our series is small, we believe that dilated medullary veins in association with leptomeningeal venous recruitment indicates a more severe degree of venous congestion, which can be aggravated by dural sinus outflow obstruction. Four of our six patients (67%) with dilated deep medullary veins had either haemorrhagic or non-haemorrhagic neurological complications. On the contrary, the three patients without dilated medullary veins did not have neurological complications. Based on our findings, we can divide patients into two groups based on the presence of deep medullary veins on MR. Patients with dilated medullary veins demands prompt and aggressive intervention.

Medullary veins are located within the brain parenchyma. Dilatation of these veins is a reflection of high venous pressure being transmitted to the brain, and is a sign of venous congestion within the brain. The location of the di-

lated deep medullary veins within the brain corresponded to the territory of the arterialised leptomeningeal veins (figure 1). In addition, leptomeningeal venous recruitment in DAVF is known to be associated with intradural haemorrhage⁴. While subdural or subarachnoid haemorrhage is believed to be due to rupture of a dilated cortical vein or varix, intracerebral haemorrhage is likely to be the result of ruptured deep medullary veins. In figure 1, both the dilated deep medullary veins and haematoma are located in the same temporal lobe.

Conclusions

Deep medullary veins are small draining veins located within the white matter of the brain parenchyma. Our results show that these veins may become dilated in patients with DAVF associated with leptomeningeal venous recruitment and are best seen on post-contrast T1-weighted MR images. Their presence reflects a more severe degree of venous hypertension and congestion. This subgroup of patients has a much higher chance of neurological complications and warrants urgent treatment.

References

- 1 Malek AM, Halbach VV et Al: Diagnosis and treatment of dural arteriovenous fistulas. In Drayer BP (ed), *Neurovascular malformations: diagnosis and intervention*. Neuroimaging Clinics of North America 8: 445-468, 1998.
- 2 DeMarco KJ, Dillon W et Al: Dural arteriovenous fistulas: evaluation with MR imaging. *Radiology* 175: 193-199, 1990.
- 3 Newton TH, Cronqvist S: Involvement of dural arteries in intracranial arteriovenous malformations. *Radiology* 93: 1071-1078, 1969.
- 4 Lasjaunias P, Chiu M et Al: Neurological manifestations of intracranial dural arteriovenous malformations. *J Neurosurg* 64: 724-730, 1986.
- 5 Vinuela F, Fox AJ et Al: Unusual clinical manifestations of dural arteriovenous malformations. *J Neurosurg* 64: 554-558, 1986.
- 6 Halbach VV, Hieshima GB et Al: Carotid cavernous fistulae: indications for urgent treatment. *Am J Radiol* 149: 587-593, 1987.
- 7 Awad IA, Little JR et Al: Intracranial dural arteriovenous malformations: factors predisposing to an aggressive neurological course. *J Neurosurg* 72: 839-850, 1990.
- 8 Hurst RW, Bagley LJ et Al: Dementia resulting from dural arteriovenous fistulas: the pathologic findings of venous hypertensive encephalopathy. *Am J Neuroradiol* 19: 1267-1273, 1990.
- 9 Davies MA, Saleh J et Al: The natural history and management of intracranial dural arteriovenous fistulae Part 1: Benign lesions. *Interventional Neuroradiology* 3: 295-302, 1997.
- 10 Djindjian R, Merland JJ, Theron J: *Super-selective arteriography of the external carotid artery*. Springer-Verlag, New York: 606-628, 1977.
- 11 Lalwani A, Dowd C, Halbach VV: Grading venous restrictive disease in patients with dural arteriovenous fistulas of the transverse/sigmoid sinus. *J Neurosurg* 79: 11-15, 1997.
- 12 Cognard C, Gobin YP et Al: Cerebral dural arteriovenous fistulas: clinical and angiographic correlation with a revised classification of venous drainage. *Radiology* 194: 671-680, 1995.
- 13 Borden JA, Wu JK, Shucart WA: A proposed classification for spinal and cranial dural arteriovenous fistulous malformations and implications for treatment. *J Neurosurg* 82: 166-179, 1995.
- 14 Castaigne P, Bories J et Al: Fistules arterioveineuses de la dure-mere, etude clinique et radiologique de 13 observations. *Ann Med Interne* 126: 813-817, 1975.
- 15 Duffau H, Lopes M et Al: Early rebleeding from intracranial dural arteriovenous fistulas: report of 20 cases and review of the literature. *J Neurosurg* 90: 78-84, 1999.
- 16 Willinsky R, Terbrugge K et Al: Venous congestion: an MR finding in dural arteriovenous malformations with cortical venous drainage. *Am J Neuroradiol* 15: 1501-1507, 1994.
- 17 Huang YP, Wolf BS: Veins of the white matter of the cerebral hemispheres (medullary veins): diagnostic importance in carotid angiography. *Am J Radiol* 92: 739-755, 1964.
- 18 Lee C, Pennington MA, Kenney CM: MR evaluation of developmental venous anomalies: medullary venous anatomy of venous angiomas. *Am J Neuroradiol* 17: 61-70, 1996.
- 19 Jimenez JL, Lasjaunias P et Al: The trans-cerebral veins: normal and non-pathologic angiographic aspects. *Surg Radiol Anat* 11 (1): 63-72, 1989.
- 20 Saba PR: The caput medusae sign. *Radiology* 207: 599-600, 1998.
- 21 Friedman DP: Abnormalities of the deep medullary white matter veins: MR imaging findings. *Am J Radiol* 168: 1103-1108, 1997.
- 22 Moody DM, Brown WR et Al: Periventricular venous collagenosis: association with leukoaraiosis. *Radiology* 194: 469-476, 1995.

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