

Color Doppler Flow Imaging of the Superior Ophthalmic Vein in Dural Arteriovenous Fistulas

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Background and Purpose—This article evaluates the intracranial venous hemodynamics of dural arteriovenous fistula (DAVF) on the basis of data from color Doppler flow imaging (CDFI) findings of the superior ophthalmic vein (SOV) and discusses the clinical application of the SOV CDFI to the DAVFs.

Methods—We examined the diameter, flow direction, flow waveform, and flow velocity of the SOV using CDFI in 20 patients with intracranial DAVF. Six patients were asymptomatic; the other 14 patients were symptomatic. Angiographic retrograde cortical venous filling was seen in 14 patients.

Results—In the DAVFs, the average SOV diameter was 2.95 ± 1.15 mm, which was significantly broad compared with that of the control subjects ($P < 0.05$). The flow direction was reversed in 2 patients and normal in the other 18 patients. Three patients showed an abnormal waveform. A reversed pulsatile waveform was observed in 2 patients, and a normograde pulsatile waveform was seen in 1 patient. The other 17 patients showed normal waveform. The average SOV diameter and resistance index values were significantly higher ($P < 0.05$) in patients with clinical symptoms, angiographic retrograde cortical venous fillings, or large DAVFs compared with those in the other patients.

Conclusions—The SOV CDFI findings in DAVFs correlated well with the patient's clinical symptoms, angiographic findings, and DAVF size. These findings were useful to evaluate the intracranial venous hemodynamics in DAVFs. (*Stroke*. 2002;33:2009-2013.)

Key Words: central nervous system vascular malformations ■ echocardiography, Doppler, color ■ fistula

Dural arteriovenous fistulas (DAVFs) constitute 10% to 15% of all intracranial arteriovenous shunts¹ and may present with an aggressive neurological clinical course or a fatal cerebral hemorrhage.² The specific clinical symptomatology and prognosis are thought to be determined by the pattern of venous drainage rather than by the arterial blood supply.³⁻⁸ Therefore, the gold standard for clarifying the management strategy is angiography. However, angiography is an invasive examination, and MR angiography (MRA) cannot demonstrate hemodynamics. Therefore, we analyzed and monitored the intracranial venous hemodynamics of the DAVFs using color Doppler flow imaging (CDFI) findings of the superior ophthalmic vein (SOV) in 20 patients with intracranial DAVF and then correlated these findings with clinical features and angiographic findings. Here, we describe and discuss the clinical usefulness of the SOV CDFI in intracranial DAVFs.

Subjects and Methods

Subjects

CDFI studies of the SOV were performed on 20 patients with intracranial DAVFs diagnosed by angiography. These 20 patients made up a consecutive series of DAVF diagnosed at Nara Medical University Hospital between January 1997 and June 2001. Patients

with DAVF located in the cavernous sinus region were excluded from this study, as were patients with any lesion causing clinical symptoms or disturbance of venous circulation on angiography other than the DAVF.

Among 20 cases, 5 were reported previously.⁹ Patients underwent neurological examination, CT scan, and angiography. Of the 20 patients, 11 were male and 9 were female. The age distribution ranged from 37 to 76 years, with a mean age of 62 years. Clinical symptoms were the following: pulsatile tinnitus in 5 patients, dizziness in 4 patients (2 of whom presented with tinnitus; in the other 2 patients, dizziness was resolved after surgery), intractable headache in 2 patients, intracerebral hemorrhage in 1 patient, transient ischemic attacks in 1 patient, and dementia in 1 patient; 6 patients were asymptomatic. Tinnitus, headache, and intracerebral hemorrhage were ipsilateral to the location of the DAVFs. Transient ischemic attack caused tetraparesis in 1 patient with DAVF located in the superior sagittal sinus. Among 6 asymptomatic patients, 4 showed positive findings for DAVF on MRI and MRA. In 1 patient who had undergone carotid endarterectomy 5 years earlier, asymptomatic DAVF was found on the MRI and MRA as follow-up examination for carotid endarterectomy. In 3 patients without any neurological complaint, MRI and MRA studies were performed as the "brain dock" examination, which is performed in Japan to screen for any asymptomatic central nervous system disease. In the last 2 patients, DAVF was seen on angiographic investigation of a brain tumor located in a different region. Neither of these patients showed any neurological symptoms such as headache, dizziness, and tinnitus at the time of diagnosis of DAVF and examination of SOV CDFI.

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Summary of 20 Patients With Intracranial DAVF

Case	Age, y	Sex	Clinical Presentation	Neuro-ophthalmic Finding	DAVF Location	Angiographic Retrograde Cortical Venous Filling	DAVF Size	SOV CDFI Findings		
								Diameter, mm	Flow Direction	Waveform
1	58	M	Intracerebral hemorrhage	...	L TSS	Recognized	Large	4.3	Normal	Normograde pulsatile
2	37	F	Pulsatile tinnitus	...	L TSS	Recognized	Small	3.0	Normal	Normal
3	66	M	Transient ischemic attack	Papilledema with conjunctival congestion	SSS	Recognized	Large	6.1	Reversed	Reversed pulsatile
4	51	M	Pulsatile tinnitus	...	L TSS	Recognized	Large	4.8	Normal	Normal
5	68	M	Asymptomatic	...	TI	Not recognized	Small	1.7	Normal	Normal
6	72	F	Asymptomatic with brain tumor	...	R AF	Recognized	Small	1.7	Normal	Normal
7	58	M	Intractable headache	...	R AF	Recognized	Small	2.9	Normal	Normal
8	65	F	Pulsatile tinnitus	...	L TSS	Recognized	Large	2.8	Normal	Normal
9	68	F	Asymptomatic	...	SSS	Not recognized	Small	2.1	Normal	Normal
10	73	M	Asymptomatic	...	L AF	Not recognized	Small	1.6	Normal	Normal
11	76	F	Pulsatile tinnitus	...	L TSS	Recognized	Small	2.8	Normal	Normal
12	59	F	Asymptomatic	...	R MF	Not recognized	Small	1.8	Normal	Normal
13	55	M	Dizziness	...	SSS	Recognized	Large	3.2	Normal	Normal
14	69	F	Asymptomatic with brain tumor	...	R TSS	Not recognized	Small	2.3	Normal	Normal
15	62	F	Pulsatile tinnitus	...	L TSS	Not recognized	Large	3.1	Normal	Normal
16	51	M	Intractable headache	...	L AF	Recognized	Large	3.8	Normal	Normal
17	64	M	Dizziness	...	L TSS	Recognized	Large	2.4	Normal	Normal
18	76	M	Dizziness, tinnitus	...	L TSS	Recognized	Large	2.6	Normal	Normal
19	64	F	Dementia	...	L TSS	Recognized	Small	2.2	Normal	Normal
20	55	M	Dizziness, tinnitus	Papilledema	L TSS	Recognized	Large	3.7	Reversed	Peverse pulsatile

L indicates left; R, right; TSS, transverse sigmoid sinus; SSS, superior sagittal sinus; TI, tentorial incisula; AF, anterior fossa; and MF, middle fossa.

One was found during postoperative follow-up study for tumor recurrence without neurological symptoms, and the other was found during routine screening examination for the metastatic intracranial lesion without neurological symptoms. These 2 patients did not demonstrate neurological symptoms even from the brain tumor. On angiography, neither of these 2 patients demonstrated disturbed venous circulation resulting from the tumor. The distribution of DAVFs was as follows: transverse sigmoid sinus in 11 patients, anterior fossa in 4 patients, superior sagittal sinus in 3 patients, tentorial incisula in 1 patient, and middle fossa in 1 patient. Angiographically, retrograde cortical venous filling was observed in 14 patients, although no retrograde venous filling could be found in the other 6 patients. As the neuro-ophthalmic manifestation, cork-screwing of the conjunctival vasculature (conjunctival congestion) was seen in 1 patient, and papilledema was seen in the 2 patients.

Methods

CDFI was performed with Computed Sonography 128XP/10 (Acuson Corp) using a 5-MHz linear-array probe. CDFI studies produced transverse color flow images and a pulsed Doppler recording on SOV. These CDFI studies provided information regarding the size, flow direction, pulsed Doppler wave curve (waveform), and flow velocity of the SOV. From flow velocity, the resistance index (RI) was calculated. The RI describes the ratio of peak systolic velocity to end-diastolic velocity. The SOV CDFI study was performed on both eyes in all patients; we evaluated data from the side showing more severely abnormal SOV CDFI findings.

All values are reported as mean \pm SD. The physiological data were compared by use of 2-tailed unpaired Student's *t* test and Kruskal-Wallis test. A value of $P<0.05$ was taken as the threshold for

statistical significance. Before we examined the SOV in 20 patients with DAVF, we examined the SOV on the 47 control subjects consisting of normal volunteers without intracranial lesion on CT and/or MRI in each side. Of the 47 control subjects, 31 were male and 16 were female. The age distribution ranged from 38 to 76 years, with a mean age of 64 years. There was no significant difference in sex or age distribution between the 20 DAVF patients and 47 control subjects. In the 47 control subjects, the SOV can be identified superiorly and nasally in the orbit on orbital CDFI. The average SOV diameter was 2.08 ± 0.26 mm. The flow direction of the SOV was toward the orbital apex in all control patients, and the average SOV RI was 0.37 ± 0.16 . The waveform may be a continuous waveform with or without cardiac or respiratory variation.¹⁰ Concerning the waveform, there were 2 abnormal types that were typed in our DAVF series: the reversed pulsatile waveform and the normograde pulsatile waveform, which was a continuous waveform influenced by the arterial waveform. The RI was evaluated only in patients with normograde-flow SOV.

Results

SOV CDFI Findings

In the 20 patients, the average SOV diameter was 2.95 ± 1.15 mm (range, 1.6 to 6.1 mm), and this value was significantly broader than that of the control subjects ($P=0.003$) (Table). The flow direction was reversed in 2 patients and normal in the other 18. Waveforms were reversed pulsatile in 2 patients, normograde pulsatile in 1

patient, and normal in 17 patients. The average RI of SOV (n=18) was 0.38 ± 0.18 (range, 0.17 to 0.86).

Clinical Symptoms

In 6 asymptomatic patients, the average SOV diameter was 1.87 ± 0.27 mm, and the average RI was 0.23 ± 0.07 . The waveform was normal in all patients. In 14 symptomatic patients, the average SOV diameter was 3.41 ± 1.06 mm, and the average RI of the 14 patients with normograde SOV flow was 0.46 ± 0.16 . Both of these 2 values were significantly ($P < 0.05$) higher than those of the asymptomatic patients. Two symptomatic patients showed an abnormal flow direction. Three symptomatic patients showed abnormal waveforms: reversed pulsatile in 2 patients and normograde pulsatile in 1 patient.

Angiographic Retrograde Cortical Venous Filling

In the 6 patients without retrograde cortical venous filling on angiography, the average SOV diameter was 2.10 ± 0.56 mm. Flow direction and waveform of SOV were normal in all 6 patients. The average RI was 0.25 ± 0.13 . In the 14 patients with retrograde cortical venous filling, the average SOV diameter was 3.31 ± 1.15 mm, and the average RI of the 12 patients with normograde SOV flow was 0.45 ± 0.17 . Concerning the SOV waveform, 2 patients showed reversed pulsatile waveform, 1 patient showed normograde pulsatile waveform, and the remaining 11 patients showed normal waveform. There was a significant difference in the average diameter and RI of the SOV between the patients with and those without retrograde cortical venous filling as indicated by angiography ($P < 0.05$).

Localization of DAVF

According to the localization of DAVF, 20 patients were divided into 3 groups. Group A consisted of 4 patients with DAVF in the anterior fossa; their average SOV diameter was 2.50 ± 1.05 mm and average RI value was 0.45 ± 0.29 . Group P consisted of 11 patients with DAVF on the transverse sigmoid sinus; their average SOV diameter was 3.09 ± 0.84 mm and average RI value was 0.42 ± 0.12 . Group M consisted of 5 patients with DAVF on superior sagittal sinus, tentorial incisula, or middle fossa; their average SOV diameter was 2.98 ± 1.84 mm and average RI value was 0.23 ± 0.05 . Between these 3 groups, there was no significant difference in average SOV diameter and RI. Therefore, there was no significant difference in SOV diameter and RI according to the localization of DAVF.

Neuro-Ophthalmic Findings

There were 2 patients showing abnormal neuro-ophthalmic findings. These 2 patients showed reversed ophthalmic artery flow in each and showed only DAVF without brain tumor. Therefore, these neuro-ophthalmic findings were due to DAVF. The other 18 patients without abnormal neuro-ophthalmic findings showed normograde SOV flow in each. There was a significant difference ($P < 0.05$) in the relationship between the flow direction of SOV and neuro-ophthalmic abnormal findings. The average SOV diameter was 4.90 ± 1.70 mm in 2 patients with abnormal neuro-ophthalmic findings and 2.73 ± 0.89 mm in the other 18 patients without abnormal neuro-ophthalmic findings. How-

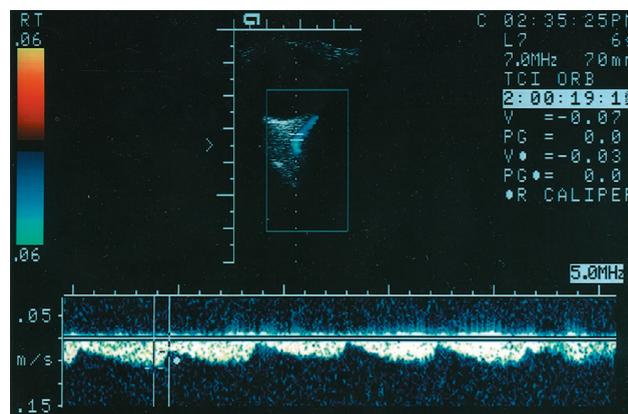


Figure 1. SOV CDFI showing the dilated SOV, normograde pulsatile waveform, and a high RI.

ever, there was no significant difference in SOV diameter between these 2 groups.

DAVF Size

Twenty patients were divided into 2 groups according to DAVF size defined by the number of feeding major vessels shown on angiography. A DAVF fed through arterial branches from only 1 major vessel such as an internal carotid artery, external carotid artery, or vertebral artery on each side was defined as a small DAVF. A DAVF fed through arterial branches from ≥ 2 major vessels was defined as a large DAVF. Group S consisted of 10 patients with small DAVFs; their average SOV diameter was 2.21 ± 0.53 mm and average RI value was 0.29 ± 0.10 . The flow direction and waveform of the SOV were normal in all 10 patients of group S. Group L consisted of 10 patients with large DAVFs; their average SOV diameter was 3.68 ± 1.14 mm, and the average RI value of the 8 patient with normograde SOV flow was 0.50 ± 0.18 . Concerning the SOV waveform in group L, 3 patients showed abnormal waveforms. There were significant differences in SOV diameters and RI values between these 2 groups ($P < 0.05$).

Illustrative Cases

Patient 1

A 58-year-old man experienced sudden-onset, right homonymous hemianopsia and aphasia. A CT scan demonstrated a subcortical hemorrhage in the left temporal lobe. A left external carotid angiogram demonstrated a DAVF at the left transverse sigmoid sinus supplied mainly by the middle meningeal artery and retroauricular artery with drainage into retrograde-fashion cortical veins. A left internal carotid angiogram showed a DAVF through the tentorial branch of internal carotid artery. The DAVF was large. SOV CDFI studies showed a dilated SOV (diameter, 4.3 mm), a normograde pulsatile waveform, and a high RI (0.57) (Figure 1).

Patient 3

A 66-year-old man complained of recurrent transient ischemic tetraparesis. Neuro-ophthalmic findings were papilledema with conjunctival congestion. Left and right external carotid angiograms demonstrated a DAVF at the superior sagittal sinus supplied by the superficial temporal artery,

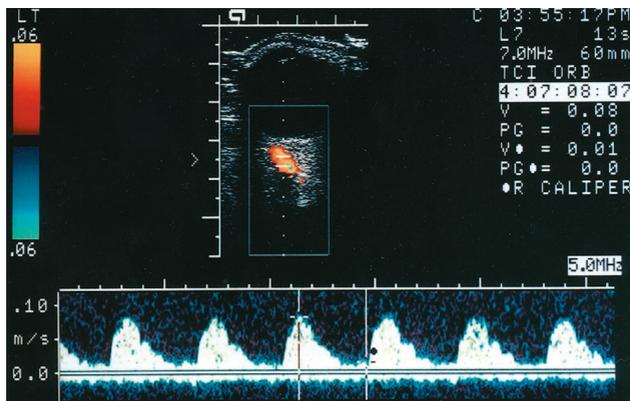


Figure 2. SOV CDFI showing the dilated SOV and the flow toward the probe with reversed pulsatile waveform.

middle meningeal artery, and occipital artery, with drainage into the ascending cortical veins. The DAVF was large in this case. The SOV diameter was 6.1 mm; the flow direction was retrograde; and the waveform was reversed pulsatile flow (Figure 2).

Discussion

Most DAVFs behave benignly, but the clinical symptoms are most likely attributable to venous ischemia resulting from venous hypertension and/or congestion resulting from the high pressure of direct and retrograde flow into the main venous systems.^{11–15} Therefore, analyzing the venous hemodynamics of DAVFs is quite significant in understanding the pathophysiology of DAVFs and in selecting the best treatment. Usually, venous hemodynamics is evaluated on angiographic findings, and these findings have been the gold standard for treatment selection in DAVFs. However, angiography is an invasive examination, and the patient needs hospitalization for a few days. To analyze the venous hemodynamics of DAVFs, we examined the SOV CDFI, which was a noninvasive examination.

It is easy to identify the SOV on orbital CDFI. Belden et al¹⁶ reported that the SOV was demonstrated in 96% of the normal control subjects through CDFI. In the presenting series, the SOV was detected in all patients on orbital CDFI. CDFI for SOV offers several advantages: (1) it is noninvasive and does not require contrast material; (2) it may be performed quickly, easily, inexpensively, and repeatedly; and (3) it provides specific topographic information regarding the orbital vasculature.¹⁷ The SOV findings reflect the intracranial venous hemodynamics because the SOV reaches the cavernous sinus without a valve through the sphenoidal fissure, and there is also no valve in the intracranial venous system, including the dural sinus. Therefore, we examined the SOV CDFI to analyze the intracranial venous hemodynamics. In the presenting study, the abnormal findings on SOV CDFI in DAVFs correlated well with clinical symptoms and angiographic retrograde cortical venous filling. The SOV CDFI reflected the intracranial venous hemodynamics. Thus, we can use the SOV CDFI as a venous hemodynamics evaluation method in DAVFs. Generally, Doppler imaging studies are dependent on the skill of the examiner. However, MRA does not have this limitation and is not an invasive examination. MRA

also provides information about the size and flow of the DAVF, but venous hemodynamics cannot be evaluated from MRA. Therefore, SOV CDFI and MRA can complement each other in the examination of DAVFs.

Although we routinely examined the SOV CDFI on both sides in each patient, we evaluated SOV CDFI findings on the side showing the more severe abnormalities. However, there was no significant difference in SOV CDFI findings between on the left and right sides regardless of the location of the lesions.

The origin and pathophysiology of intracranial DAVFs remain unknown. However, except for some pediatric lesions and other lesions associated with complex developmental anomalies, the vast majority of DAVFs appear to be acquired.¹¹ In this series, there were no pediatric patients or patients with other developmental anomalies, and most of the patients were >50 years of age. Therefore, we believe that the DAVFs examined in this series were acquired. In pediatric patients, vascular compliance and elasticity differ from those in adult patients. SOV CDFI findings may also differ from those in an adult series. In this study, we avoided confounding factors derived from an excessively wide age distribution. In the future, SOV CDFI studies in pediatric DAVFs are needed for comparison with the DAVFs in this adult series.

In the present series, the overall average SOV diameter was significantly broader than that in the control population. The broad SOV could be identified by the venous congestion resulting from the high volume of shunt flow or occlusion of the dural sinus. In patients with clinical symptoms or angiographic retrograde cortical venous filling, the RI value was significantly higher than in other patients. The SOV RI may reflect the hemodynamics of the distal resistance of the SOV. The significantly increased SOV RI value indicates the high resistance of the intracranial venous system resulting from high intracranial venous pressure. Patients with high intracranial venous pressure present with some clinical symptoms and/or angiographic retrograde cortical venous filling. In the present series, patients with reversed SOV flow or abnormal SOV waveform in addition to dilated SOV and/or high RI showed serious clinical symptoms such as intracerebral hemorrhage or an ischemic event. With progressive venous congestion, the SOV CDFI findings may also advance from dilatation of SOV, increased SOV RI value, and abnormal waveform to reversed ophthalmic artery flow, together with clinical symptoms and angiographic retrograde cortical venous fillings. If attention is given to these findings, SOV CDFI can recognize the advance of venous congestion.

Among 20 patients, 2 (patients 6 and 14) were detected during examination for brain tumor. These 2 patients did not show any neurological symptoms at the time DAVF was diagnosed and the SOV CDFI was examined. Furthermore, angiography did not show any venous circulation disturbance resulting from the tumor in either case. Therefore, we included these 2 patients in this study. If these patients had shown any clinical symptoms such as intracranial hypertension or focal neurological deficit, they would have been excluded from the present study. In these 2 patients, SOV CDFI examination showed normal findings for SOV diameter, flow direction, and waveform.

There was no significant difference in SOV diameter and RI according to DAVF localization in the presenting series. The SOV connect with various intracranial venous systems only via

cavernous sinus, but there was no direct connection with the SOV from the intracranial venous system even from the anterior fossa DAVF. Accordingly, the SOV CDFI findings did not significantly differ according to DAVF localization. This finding also suggested excellent interconnections (right to left and anterior to posterior) of the venous encephalic circulation that were confirmed on SOV CDFI study. Therefore, regardless of DAVF localization, SOV CDFI can monitor the intracranial venous hemodynamics of patients with intracranial DAVF.

Two patients showed abnormal neuro-ophthalmic findings. Only these 2 patients showed reversed SOV flow on SOV CDFI findings. These 2 patients did not show any intracranial mass lesion such as brain tumor; therefore, these abnormal neuro-ophthalmic findings were revealed by venous hemodynamic compromise resulting from DAVF in each patient. There was a significant relationship between neuro-ophthalmic findings and SOV flow direction. Therefore, the flow direction of the SOV on CDFI findings was a good monitor for neuro-ophthalmic findings.

The relationship between DAVF size and intracranial venous hemodynamics is interesting. In this study, we defined DAVF size by the number of major vessels such as an internal carotid artery, external carotid artery, and vertebral artery on each side that fed the DAVF. In this study, there was a clear relationship between DAVF size and SOV CDFI findings. The larger DAVFs showed more severe venous hemodynamic compromise on SOV CDFI findings. SOV CDFI findings could be associated with DAVF size; therefore, those findings also could be used to monitor changes in DAVF size, ie, increased or decreased DAVF during follow-up or after any treatment.

The surgical indication for DAVFs is usually determined according to the angiographic findings and clinical symptoms.^{4,18–20} Awad et al³ reported that leptomeningeal venous drainage, variceal or aneurysmal venous dilatation, and galenic drainage correlate with aggressive neurological presentation. In patients with the above angiographic findings, radical treatment is recommended. According to the authors' data, SOV CDFI findings can demonstrate an aspect of intracranial venous hemodynamics in intracranial DAVFs. From the perspective of SOV CDFI findings, conservative management and strict follow-up are recommended for asymptomatic DAVFs with normal SOV CDFI. Radical treatment is recommended when SOV CDFI shows abnormal findings such as abnormal flow direction, abnormal waveform, and high RI value. However, the treatment for DAVFs should be based on angiographic findings and clinical symptoms. The SOV CDFI findings remain reference values for the choice of treatment plan.

The present study involved a very small series, so the precise usefulness of SOV CDFI findings of intracranial DAVFs remains unclear. However, according to this study, the SOV CDFI findings reflect the intracranial venous hemodynamics and correlate with clinical symptoms and angiographic retrograde cortical venous filling. Therefore, in the future, the SOV CDFI may be useful for evaluating the effect of treatment maneuvers or identifying the recurrence or development of DAVF.

Conclusions

CDFI findings regarding the SOV in patients with DAVFs correlate well the patient's clinical symptoms, angiographic findings, and DAVF size. The patient with a dilated SOV with an abnormal flow direction, abnormal waveform, and high RI showed papilledema resulting from severely compromised venous hemodynamics. The SOV CDFI in DAVFs could be useful as a method to evaluate intracranial venous hemodynamics. DAVFs showing dilated SOVs with an abnormal flow direction, abnormal waveform, and high RI should be considered for radical treatment, because these findings indicate intracranial venous hemodynamic compromise.

References

1. Newton TH, Cronqvist S. Involvement of dural arteries in intracranial arteriovenous malformations. *Radiology*. 1969;93:1071–1078.
2. Lucas CP, Zabramski JM, Spetzler RF, Jacobowitz R. Treatment for intracranial dural arteriovenous malformations: a meta-analysis from the English language literature. *Neurosurgery*. 1997;40:1119–1132.
3. Awad IS, Little JR, Akrawi WP, Ahi J. Intracranial dural arteriovenous malformations: factors predisposing to an aggressive neurological course. *J Neurosurg*. 1990;72:839–850.
4. Borde JA, Wu JK, Shucart WA. A proposed classification for spinal and cranial dural arteriovenous malformations and implications for treatment. *J Neurosurg*. 1995;82:166–179.
5. Houser OW, Backer HL Jr, Rhoton AL Jr, Okazaki H. Intracranial dural arteriovenous malformations. *Radiology*. 1972;105:55–64.
6. Iwama T, Hashimoto N, Takagi Y, Tanaka M, Yamamoto S, Nishi S, Hayashida K. Hemodynamic and metabolic disturbances in patients with intracranial dural arteriovenous fistulas: positron emission tomography evaluation before and after treatment. *J Neurosurg*. 1997;86:806–811.
7. King WA, Martin NA. Intracerebral hemorrhage due to dural arteriovenous malformations and fistulae. *Neurosurg Clin N Am*. 1992;3:577–590.
8. Lalwani AK, Dowd CF, Halbach VV. Grading venous restrictive disease in patients with dural arteriovenous fistulas of the transverse/sigmoid sinus. *J Neurosurg*. 1993;79:11–15.
9. Sakaki T, Morimoto T, Hoshida T, Kawaguchi S, Nakase H, Okuno S. Surgical treatment of dural arteriovenous fistulas around the superior sagittal and transverse sigmoid sinuses. *Jpn J Neurosurg (Tokyo)*. 2001;10:47–55.
10. Berges O. Color Doppler flow imaging of the orbital veins. *Acta Ophthalmol*. 1992;204:55–58.
11. Awad IA. Dural arteriovenous malformations. In: Carter LP, Spetzler RF, Hamilton MG, eds. *Neurovascular Surgery*. New York, NY: McGraw-Hill; 1995:905–932.
12. Hurst RW, Hackney DB, Goldberg HI, Davis RA. Reversible arteriovenous malformation-induced venous hypertension as a cause of neurological deficits. *Neurosurg*. 1992;30:422–425.
13. Kurata A, Miyasaka Y, Yoshida T, Kunii M, Yada K, Kan S. Venous ischemia caused by dural arteriovenous malformation. *J Neurosurg*. 1994;80:552–555.
14. Lasjaunias P, Chiu M, Brugge KT, Tolia A, Hurth M, Bernstein M. Neurological manifestations of intracranial dural arteriovenous malformations. *J Neurosurg*. 1986;64:724–730.
15. Nakamura M, Tamaki N, Hara Y, Nagashima T. Two unusual cases of multiple dural arteriovenous fistulas. *Neurosurgery*. 1997;41:288–293.
16. Belden CJ, Abbitt PL, Beadles KA. Color Doppler use of the orbit. *Radiographics*. 1995;15:589–608.
17. Flaharty PM, Lieb WE, Sergott RC, Bosley TM, Savino PJ. Color Doppler imaging: a new noninvasive technique to diagnose and monitor carotid cavernous fistulas. *Arch Ophthalmol*. 1991;109:522–526.
18. Martin NA, King WA, Wilson CB, Nutik S, Carter LP, Spetzler RF. Management of dural arteriovenous malformations of the anterior cranial fossa. *J Neurosurg*. 1990;72:692–694.
19. Sundt TM Jr, Piepgras DG. The surgical approach to arteriovenous malformations of the lateral and sigmoid sinuses. *J Neurosurg*. 1983;59:32–39.
20. Thompson BG, Doppman JL, Oldfield EH. Treatment of cranial dural arteriovenous fistulae by interruption of leptomeningeal venous drainage. *J Neurosurg*. 1994;80:617–623.