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Intracranial Dural Arteriovenous Fistulas: Natural History and Rationale for Treatment with Stereotactic Radiosurgery

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Abstract

Dural arteriovenous fistulas (DAVFs) are abnormal arteriovenous communications within the dura. The symptoms depend on their location and the pattern of the venous drainage. Patients with cavernous sinus DAVFs often present with ocular manifestations such as exophthalmos, chemosis and diplopia. Patients with transverse or sigmoid sinus DAVFs frequently experience headache and tinnitus on the affected side. DAVFs with anterograde sinus or cortical venous drainage (CVD) have been clinically regarded as benign, whereas DAVFs with retrograde CVD are considered aggressive in behavior. Similar to other cerebral arteriovenous malformations, DAVFs can hemorrhage, with an estimated annual risk of approximately 1.8%. The recommended therapeutic intervention for a DAVF is dependent on the anticipated natural history of the lesion. Management options include surgical resection, embolization and radiosurgery. Radiosurgical treatment has been used for DAVFs in various locations including the anterior cranial fossa, cavernous sinus, transverse/sigmoid sinus, superior sagittal sinus and tentorium. We present an update on 321 DAVF patients treated at the Taipei Veterans General Hospital using Gamma Knife radiosurgery. The prescribed mean margin dose was 17.2 Gy. In our series, 98% of patients had a stable or improved clinical condition after radiosurgery. Stereotactic radiosurgery using the Gamma Knife is a safe and effective alternative for the treatment of DAVFs.

Intracranial dural arteriovenous fistulas (DAVFs) are abnormal arteriovenous communications within the dura, in which meningeal arteries shunt blood directly into the dural sinus or leptomeningeal veins [1, 2]. The incidence of DAVFs has been estimated at 5–20% of all intracranial vascular malformations [3–5]. DAVFs comprise only 6% of supratentorial vascular malformations whereas they constitute 35% of infratentorial malformations [6]. The mean age of presentation for a DAVF is

50–60 years of age with no sex preference, though there is a wide range seen [7, 8]. Unlike the more common intracerebral or parenchymal arteriovenous malformations (AVMs), DAVFs are thought to be acquired in origin due to inflammation, thrombosis or trauma of the dural sinus. However, in many cases the exact etiology and underlying disease are difficult to trace and those DAVFs are considered idiopathic [9, 10]. DAVFs most commonly occur in the regions of the cavernous sinus (CS), transverse/sigmoid sinuses, tentorium/torcula, or cerebral convexities with drainage to the superior sagittal sinus [1, 11, 12].

A thorough understanding of DAVF hemodynamics requires a detailed cerebral angiographic investigation. Drainage of the venous flow from a DAVF can be antegrade or retrograde through a dural sinus, through a cortical vein or both. The pattern of the venous drainage is not necessarily static though. Gradual alternation in the venous flow from antegrade to retrograde, and delayed recruitment of arterial feeders into the nidus (sump effect) have been observed in some patients [3, 8]. This is hypothesized to occur as a result of progressive sinus hypertension with redirection of the blood flow into cortical veins [2, 11, 13]. The gradual venous hypertension and reflux of the cortical veins may eventually predispose to the risks of cerebral hemorrhage and/or other neurological deficits [1].

Not all DAVFs demonstrate such a progressive clinical course though. Although not frequently seen, some DAVFs can regress and thrombose gradually resulting in a spontaneous cure [14, 15]. The factors predisposing to DAVF progression or involution have not been clearly clarified.

Natural Course

The clinical presentation of a DAVF is dependent on its location and pattern of the venous drainage. Patients with CS DAVFs often have ocular manifestations (exophthalmos, chemosis, visual impairment and diplopia). Those with lateral tentorial (transverse/sigmoid sinus) lesions frequently complain of headache and pulse-synchronous tinnitus on the affected side. DAVFs with anterograde sinus or cortical venous drainage (CVD) have been clinically regarded as benign, whereas DAVFs with retrograde CVD are considered aggressive in behavior [8, 16, 17]. In 1990, Awad et al. [1] reported a meta-analysis of 377 DAVFs and defined 100 aggressive cases as those with hemorrhages or progressive focal neurological deficits; the other 277 DAVFs were defined as benign cases. They concluded that no location of the DAVFs was immune from the aggressive neurological behavior. The factors that predict aggressive neurological presentation included leptomeningeal venous drainage, variceal or aneurysmal venous dilation, and galenic drainage of the DAVFs.

Similar to other cerebral AVMs, DAVFs can hemorrhage, with an estimated annual risk of approximately 1.8% [7]. Van Dijk et al. [17] in 2002 reported

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that persistence of the cortical venous reflux in DAVFs yields an annual hemorrhage rate of 8.1% and a mortality rate of 10.4%. Duffau et al. [18] reported a high risk of early rebleeding (35% within 2 weeks) after the first episode of hemorrhage, with graver consequences from the second bleed. Söderman et al. [19] in 2008 evaluated the hemorrhage rate in their 85 cases of DAVFs with retrograde CVD. They found a lower hemorrhage rate compared to those of the other previous reports. In their patients already presenting with an intracranial hemorrhage, the annual risk for the recurrent hemorrhage was 7.4% while in those patients not presenting with a hemorrhage, the bleeding rate was approximately 1.5% per year [19].

Classifications

Two main classification systems have been proposed. The Borden-Shucart system distinguishes DAVFs depending on the site of drainage and the presence of CVD [13]. Type I DAVFs drain directly into the sinus or meningeal veins with antegrade flow, whereas type II DAVFs have retrograde flow through the sinus into the subarachnoid veins. Type III DAVFs directly drain into the subarachnoid veins in a retrograde fashion.

The system of Cognard et al. [11] similarly separates DAVFs depending on the site of drainage and the presence of CVD, but also considers the direction of flow through the draining vein as well as the presence of cortical venous ectasia. Cognard type I DAVFs have solely antegrade sinus drainage, similar to the Borden-Shucart system. Cognard type II DAVFs demonstrate retrograde drainage and are subdivided depending on whether drainage is through the sinus (IIa), cortical vein (IIb) or both (IIa + b). Cognard type III DAVFs drain directly into cortical veins similar to Borden-Shucart types, but the Cognard classification gives lesions with venous ectasia a separate designation of type IV. DAVFs that drain into spinal perimedullary veins are designated Cognard type V. The authors present their series of 258 patients and demonstrate a correlation between DAVF type and rate of aggressive clinical symptoms and risk of hemorrhage.

Therapeutic Methods

The recommended therapeutic intervention for a DAVF is dependent on the anticipated natural history of the lesion. For lesions with antegrade sinus drainage (Borden type I) and benign clinical manifestations, intervention is usually palliative or observational unless the patient's symptoms are intolerable [5, 8]. Vigilance is prudent though as conversion in flow pattern can occur in approximately 2% of patients, elevating the risks of developing new neurological deficits [8, 11]. For DAVFs with

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intolerable symptoms, progressive neurological deficits or elevated risk of hemorrhage, intervention is recommended [11, 13, 20].

Advances in the field of interventional neuroradiology have increased treatment options for patients with DAVFs. Obliteration of the fistula can be attempted through a transarterial or transvenous route. Transarterial embolization alone rarely leads to a complete obliteration of the DAVF, because there are usually numerous arterial feeders to the nidus. The purpose of a transarterial approach is mainly the reduction of arterial feeders and the palliative symptomatic relief [21]. For curative treatment, additional treatment through a retrograde transvenous approach may be necessary. In transvenous embolization, superselective disconnection of the refluxing vein is preferred over sacrifice of the dural sinus, although this sometimes becomes necessary to achieve a cure [21]. Endovascular therapy can also be combined with surgery or radiosurgery when it is not feasible to completely obliterate a DAVF alone.

An open surgical approach is indicated for DAVFs with aggressive features that are not amenable to comprehensive endovascular treatment. Typically, lesions involving the anterior cranial fossa or tentorial incisura are associated with hemorrhage, and surgical intervention is indicated. Surgical strategies include ligation of the fistula at the junction with the drainage vein, interruption of arterial feeders, coagulation and/or excision of the fistula in the dura, and resection of the involved sinus [22–24]. Recent studies have suggested that disconnection of the draining vein alone without resection of the sinus is equally efficacious as resection of the fistula. The former can avoid risks of venous hypertension associated with the sinus removal, particularly where the sinus is patent [25–28]. Reported morbidity and mortality of surgical intervention have ranged from 0–13% [29].

Stereotactic radiosurgery has long been used for treatment of intraparenchymal AVMs, and treatment of DAVFs would be a natural extension of this [30–32]. Radiosurgical treatment has been delivered for DAVFs in various locations including the anterior cranial fossa, CS, transverse/sigmoid sinus, superior sagittal sinus and tentorium [33–42]. Guo et al. [35] and Pollock et al. [40] have separately reported an approximately 80% obliteration rate for CS DAVFs treated by Gamma Knife alone or combined with embolization. More recently, Söderman et al. [5] reported on 49 patients with 52 DAVFs, with a 68% obliteration rate and another 24% with flow regression at 2 years. Radiosurgery is often combined with endovascular therapy to provide immediate relief of symptoms and possibly reduction in hemorrhagic risk [12, 34, 36, 37, 43–45]. Complete and partial obliteration rates are similar to those reported for radiosurgery alone, as are the rates of symptomatic improvement. While the early experience at the Taipei Veterans General Hospital using Gamma Knife radiosurgery (GKRS) to treat DAVFs have been published before [35, 39, 42], we present here an update to our experience treating DAVFs with radiosurgery with an additional 83 patients and longer length of follow-up.

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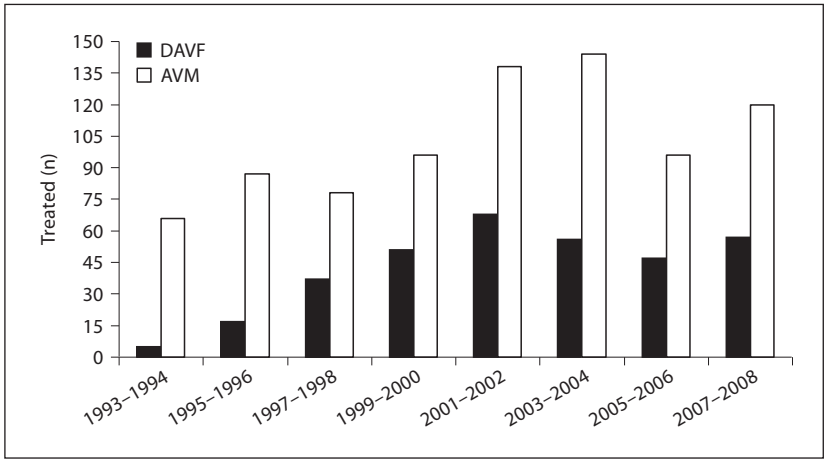

Fig. 1. Number of AVMs and DAVFs treated biennially by GKRS at the Taipei Veterans General Hospital between 1993 and 2008.

The Taipei Veterans General Hospital Experience in Treating Dural Arteriovenous Fistulas with the Gamma Knife

Patients and Classifications

Between 1993 and 2008, a total of 1,395 patients with intracranial vascular lesions were treated using GKRS at the Taipei Veterans General Hospital. 23% of these vascular malformations (321 patients) were DAVFs, a rate higher than that reported by others [4, 5, 12, 46, 47]. One possible explanation for the higher incidence at our institution is a greater awareness of the possibility of a DAVF and a higher rate of early detection of these lesions by radiological imaging [20]. This finding may thus reflect both the patient referral pattern and treatment bias at our institution. Figure 1 demonstrates the number of AVMs and DAVFs treated biennially at our institution between 1993 and 2008.

Of our 321 DAVF patients, 141 (44%) were male and 180 (56%) were female. The age at the time of GKRS ranged from 17 to 81 years (mean 57.8 years). Two potential inciting factors for DAVF formation could be traced: 31 patients (13.4%) had a history of head trauma and 14 patients (6%) had a history of head and neck surgery prior to their DAVF diagnosis. Table 1 shows the anatomical localization of the DAVFs in our patients. The most common locations are the CS (206 cases) followed by the transverse/sigmoid sinus (72 cases), which together account for 86.6% of the cases. Owing to the unique clinical characteristics of DAVFs involving the CS, we divided the DAVFs into two groups: the CS group and the non-cavernous-sinus (NCS) group. For the CS group, we applied Barrow's classification to describe the feeding arteries to the CS DAVF (table 2). All DAVFs were also classified based on their angiographic venous drainage pattern using the systems of Borden-Shucart

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Table 1. Location of DAVFs in 321 patients treated by GKRS

	Number	Percentage
Cavernous sinus	206	64.2
Transverse/sigmoid sinus	72	22.4
Petrosal sinus	9	2.8
Superior sagittal sinus	8	2.5
Others		
Tentorium	9	2.8
Frontal base (anterior fossa)	6	1.9
Sphenoparietal	4	1.2
Vein of Galen	2	0.6
Jugular foramen	2	0.6
Clivus	2	0.6
Foramen magnum	1	0.3
Total	321	100.0

Table 2. Barrow classification of CS DAVFs

	Number of cases	Percentage
A (direct)	0	0.0
B (indirect, ICA feeding)	19	9.2
C (indirect, ECA feeding)	12	5.8
D (indirect, both ICA and ECA feeding)	175	85.0
Total	206	100.0

ICA = Internal carotid artery; ECA = external carotid artery.

and Cognard (table 3) [11, 13]. Of the 115 NCS DAVFs, 63 cases were Borden type I (Cognard types I and IIa) with solely dural sinus drainage, and thus considered clinically benign [8, 16, 21]. The remaining 52 cases were Borden type II or III (Cognard type IIb, III, IV, V), demonstrating retrograde CVD and thus considered clinically 'aggressive' [14, 17].

Patients with CS DAVFs frequently presented with symptoms related to ocular functions including red eyes, proptosis, chemosis, visual impairment and diplopia. The duration of clinical symptoms prior to diagnosis of CS DAVFs ranged from 1 to 38 months (median 4 and mean 6 months). Seven of the CS DAVF patients (3.4% of CS DAVFs) experienced an intracranial hemorrhage before treatment.

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Table 3. Cognard and Borden classifications of 115 patients with NCS DAVFs

	Number	Percentage
Cognard type		
I	25	21.7
IIa	38	33.0
IIb	9	7.8
IIa + IIb	26	22.6
III	6	5.2
IV	8	7.0
V	3	2.6
Total	115	100.0
Borden type		
I (sinus drainage)	63	54.8
II (sinus drainage with retrograde venous filling)	35	30.4
III (retrograde drainage to cortical veins only)	17	14.8
Total	115	100.0

In the NCS DAVFs, the clinical manifestations and severity of symptoms were more varied depending on location of the fistula and pattern of the venous drainage. For the 72 patients with transverse/sigmoid sinus DAVFs, pulsatile tinnitus and headache were the most common symptoms. Hemorrhage before treatment occurred in 8 (11.1%) of the 72 patients and nonhemorrhagic neurological deficits developed in 27 (37.5%) patients. For the 9 patients with DAVFs involving the tentorium, 6 (67%) were Borden type II/III, and 2 (22.2%) had history of previous hemorrhage. Arteriovenous shunts involving the anterior skull base were all aggressive and harbored a high risk of hemorrhage (3 of 6 patients). For the 115 patients with NCS DAVFs, 16 (14%) had hemorrhagic events prior to diagnosis, and 43 (37%) suffered from persistent or slowly progressive neurological deficits (table 4).

We also analyzed the relationship between DAVF types and duration of symptoms in NCS DAVFs (table 5). The average duration of symptoms before diagnosis in Borden type I (Cognard type I and type IIa) patients was 19.2 months (range 3–168 months). For Borden type II (Cognard type IIb and type IIa + b) patients, the average duration of symptoms was 39.1 months (range 2–180 months). For Borden type III (Cognard type III, IV and type V) patients, the average duration of symptoms was 23.7 months (range 1–144 months). This longer duration of symptoms seen with a higher grade DAVFs suggests that DAVFs with retrograde CVD may be present for longer prior to diagnosis (p value <0.0001 between Borden type I and type II/III, independent t test).

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Table 4. Incidence of intracerebral hemorrhage (ICH) and nonhemorrhagic neurological deficit (NHND) before GKRS in 321 patients with DAVFs

	Patients	ICH	NHND
Cavernous sinus	206	7 (3.4%)	9 (4.4%)
Transverse/sigmoid sinus	72	8 (11.1%)	27 (37.5%)
Petrosal sinus	9	1 (11.1%)	4 (44.4%)
Superior sagittal sinus	8	0 (–)	3 (37.5%)
Tentorium	9	2 (22.2%)	4 (44.4%)
Frontal base (anterior fossa)	6	3 (50%)	3 (50%)
Sphenoparietal	4	2 (50%)	0 (–)
Vein of Galen	2	0 (–)	1 (50%)
Jugular foramen	2	0 (–)	0 (–)
Clivus	2	0 (–)	0 (–)
Foramen magnum	1	0 (–)	1 (100.0%)
Total	321	23 (7.2%)	52 (16.2%)

Nonhemorrhagic neurological deficits include symptoms of hemiparesis, hemiparesthesia, cerebellar sign, dementia and mental confusion.

Table 5. Duration of symptoms before treatment in 115 patients with NCS DAVFs stratified by Borden classification

Borden type	Patients n	Average duration of symptoms months	Range months
I	63	19.2	3–168
II	35	39.1	2–180
III	17	23.7	1–144

Before radiosurgery, some of the patients had attempted other therapies for treatment of the DAVF. Thirteen of the CS DAVFs and 28 of the NCS DAVFs had undergone prior endovascular embolizations from 1 to 4 times in attempts to obliterate the lesion. Thirteen patients who suffered from intracranial hemorrhage from NCS DAVFs had undergone craniotomy for hematoma evacuation and clipping of feeding vessels. GKRS was performed as secondary treatment in these 54 patients due to their residual angiographic filling of the fistula. In 3 patients with CS DAVFs (1.5% of CS DAVFs), spontaneous remission of symptoms or angiographic regression of the DAVFs was seen and no further intervention was taken. The remaining patients underwent radiosurgery as the primary treatment. The indications for radiosurgical treatment are intolerable symptoms (ocular symptoms, headache, pulsatile tinnitus), focal neurological deficits and presence of a residual shunt after other therapeutic modalities.

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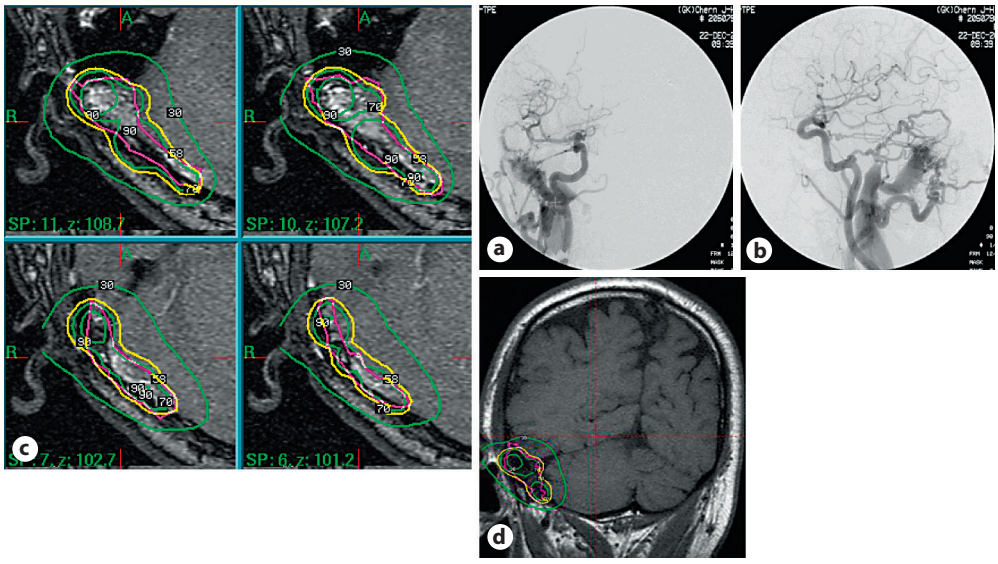

Fig. 2. A case of Borden type I DAVF at the junction of the right transverse and sigmoid sinuses, diagnosed by anteroposterior view (**a**) and lateral view (**b**) of the common carotid angiogram. The Gamma Plan® dose-planning program illustrated a 58% isodose line covering the target volume on serial transaxial time-of-flight MRA images (**c**) and coronal T_1-weighted MRI (**d**). In this case, the peripheral and maximum doses were 17.5 and 30.2 Gy, respectively, with a mean dose of 23.4 Gy. The target volume was 16.1 cm^3.

Radiosurgical Method

The standard GKRS procedure was performed using a Gamma Knife (Elekta AB, Stockholm, Sweden) either with model B (from 1993 to 2006) or model 4C (after 2007). Target localization was performed by integrating imaging data from stereotactic noncontrast magnetic resonance imaging (MRI) including thin-cut axial views time-of-flight magnetic resonance angiography (MRA), and a cerebral X-ray angiogram. Our goal of the treatment was to occlude the fistulous shunts completely. Proper delineation of the treatment target to include all abnormal arteriovenous shunts on the dural sinus wall is crucial for a successful treatment. The target volume was defined along the involved dural sinus wall where the true arteriovenous fistula occurs [2, 3, 48, 49]. The remote arterial feeders and drainage veins distal to the sinus were excluded from the treatment volume, as they were not considered part of the nidus (fig. 2). In our patients, the mean treatment target volume for CS DAVFs was 4.7 cm^3 (range 0.2–28.4 cm^3), whereas NCS DAVFs demonstrated a larger mean treatment volume of 16.9 cm^3 (range 0.8–52 cm^3).

The radiation dose profile is shown in table 6. The prescribed margin doses for both CS and NCS DAVFs were similar, with a mean dose of 17.2 Gy, although the mean maximum dose differed: 25 Gy for CS DAVFs and 30 Gy for NCS DAVFs. For the treatment of CS DAVFs, we preferred to use a large (14 or 18 mm) collimator to

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Table 6. Radiation profile of GKRS for the treatment of 321 patients with DAVFs

	CS group	NCS group
Radiation volume, cm^3	4.7 (0.2–28.4)	16.9 (0.8–52)
Maximum dose, Gy	25.2 (18.1–38)	29.9 (20.6–36)
Marginal dose, Gy	17.2 (14–25)	17.2 (15–21)
Isodose level, %	68.5 (50–96)	57.7 (50–90)
Isocenters, n	3.3 (1–14)	13 (1–27)

Figures within parentheses demonstrate the range.

cover the margin of the CS. The average number of isocenters was 3. For the NCS DAVFs, a greater number of isocenters (several large and many small shots) was used to cover the treatment volume, with a mean number of isocenters of 13 (range 1–27). Care was taken to protect the adjacent critical structures such as the optic nerve and brainstem to receive radiation doses less than 8–9 Gy.

Follow-Up Program and Results

Post-GKRS follow-up studies were available in 156 (76%) of the 206 patients with CS DAVFs, and 108 (94%) of the 115 patients with NCS DAVFs. The mean follow-up period for the CS group was 20.8 months (range 1–149 months), while for the NCS group it was 28 months (range 2–141 months). After the GKRS, both a clinical neurological examination and radiographic imaging study (MRI with MRA) were performed at 6-month intervals. Cerebral X-ray angiography was usually performed between 1 and 3 years after GKRS, if complete regression of the lesion had been shown on the MRI (fig. 3). For CS DAVFs, a noninvasive color Doppler ultrasonography examined through eyeballs was performed every 3 months to evaluate flow direction and velocity in the superior ophthalmic vein [50]. Frequently, normalization of the color Doppler ultrasonography was associated with concomitant findings of complete obliteration of DAVF on MRI and cerebral angiography (fig. 4).

Patient outcomes after radiosurgery were grouped into four categories: (1) complete improvement, indicating complete symptomatic relief with complete obliteration of the DAVF on cerebral angiogram and/or MRA; (2) partial improvement, indicating partial resolution of clinical symptoms with >50% regression of the DAVF nidus on MRA; (3) stationary, indicating no change of the DAVF nidus on the follow-up MRA; (4) progression, indicating enlargement or aggressive change of the DAVF nidus on MRA.

Table 7a summarizes the clinical outcomes in our 264 DAVF patients with follow-ups. For the CS DAVFs, 109 of the 156 patients (70%) showed complete improvement, 47 (30%) were partially improved. No lesions were stationary or progressed after radiosurgery. For the NCS DAVFs, 64 of the 108 (59%) showed complete improvement, 40 (37%) were partially improved, 2 (2%) were stationary, 1 (1%) showed progression.

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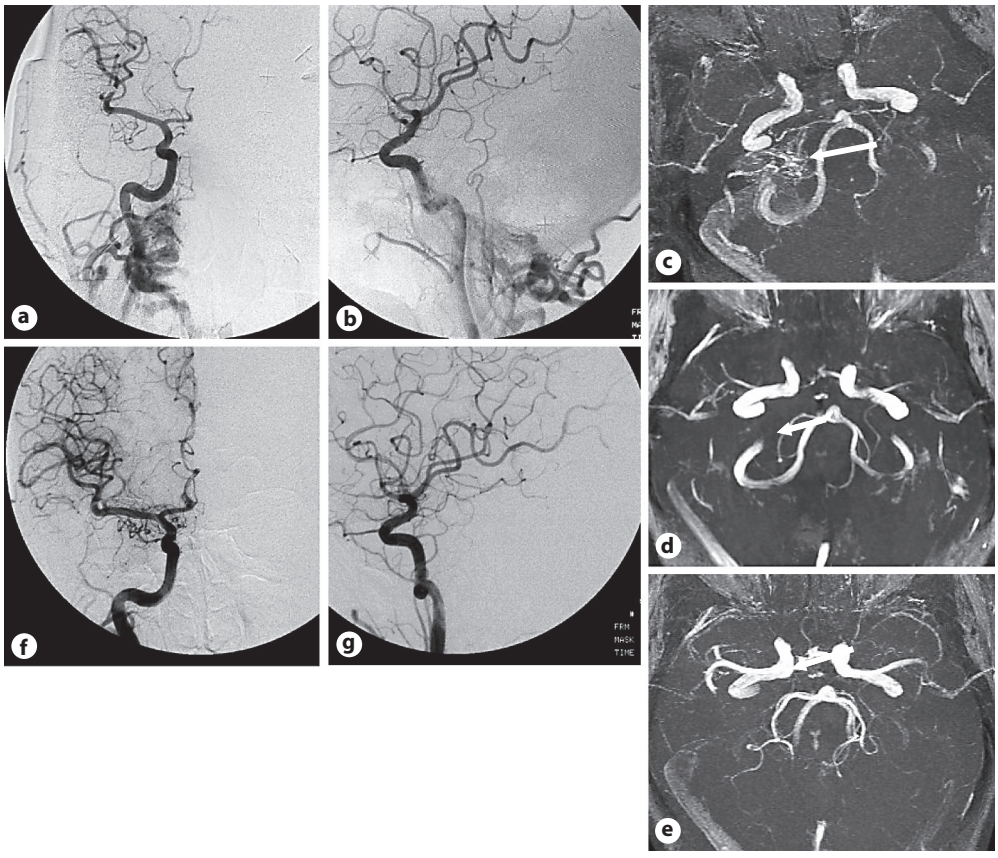

Fig. 3. A case of Borden type I DAVF in the right condylar foramen region on anteroposterior view (**a**) and lateral view (**b**) of the common carotid angiogram. A gradual decrease in the abnormal flow of the DAVF (black arrow) was noted on sequential MRA follow-up images before GKRS (**c**), 6 months after GKRS (**d**) and 20 months after GKRS (**e**). Two years after radiosurgery, the angiogram showed complete obliteration on anteroposterior view (**f**) and lateral view (**g**).

One patient died due to a new intracerebral hemorrhage after treatment. This was a 70-year-old man, with a Borden type III DAVF involving the tentorial region. GKRS was performed in 2002 and the patient had an initially stable course. However, in 2006 (59 months after GKRS), the patient developed a sudden loss of consciousness with intracerebral hemorrhage. Despite emergency craniotomy for evacuation of the hematoma, the patient expired. Thus, the mortality rate for the entire series is 0.4% (1 of 264 with follow-up available). Postradiosurgical hemorrhage due to uncontrollable venous hypertension was found in another patient with an extensive, aggressive DAVF (Borden type II) involving the transverse/sigmoid sinus. This patient recovered from the hemorrhage and improved after further combined treatment with endovascular embolization and repeated radiosurgery.

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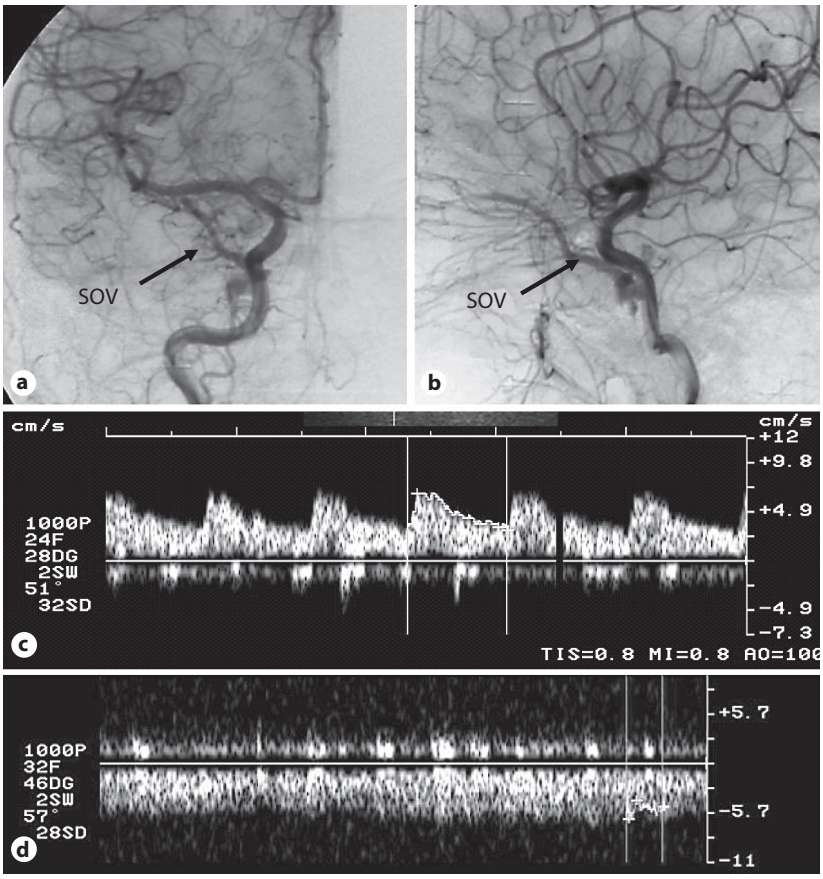

Fig. 4. Color Doppler ultrasound can be used to assess flow patterns of CS DAVF before and after treatment. A case of indirect DAVF at the right cavernous sinus with engorged superior ophthalmic vein (SOV) showing on AP (**a**) and lateral views (**b**) of the common carotid angiogram. (**c**) Color Doppler ultrasonography revealed pulsatile reverse flow of the SOV before radiosurgery. (**d**) Eight months after the treatment, the flow of the SOV returned to normal.

In this series, 98% (260 of 264 patients) had a stable or improved clinical condition after radiosurgery. For the assessment of adverse reactions to radiation on MR images, there was only 1 patient who developed radiation-induced brain edema 6 months after radiosurgery. The edema subsided gradually after steroid treatment.

In order to evaluate the response to GKRS in DAVFs with different venous drainage patterns, we further analyzed treatment results of the 108 patients with NCS DAVFs based on the Borden classification (table 7b). The results show that radiosurgery was effective in treating Borden type I lesions with a 72% complete obliteration rate, while another 28% had partial improvement. However, for Borden type II and III lesions, a lower cure rate was observed. Of the 48 Borden type II and III patients, complete obliteration was observed in 21 (44%), with another 48% showing partial

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Table 7. Clinical outcomes after GKRS

a In 264 patients with DAVFs available for neurological and imaging follow-ups

	CS	NCS
Complete improvement	109 (70%)	64 (59%)
Partial improvement	47 (30%)	40 (37%)
Stationary (no change)	–	2 (2%)
Progression	–	1 (1%)
Death	–	1 (1%)
Total	156	108

b In 108 patients with NCS DAVFs stratified by Borden classification

Borden type	Complete improvement	Partial improvement	Stationary	Progression	Death	Total
I	43	17	0	0	0	60
II	12	18	1	1	0	32
III	9	5	1	0	1	16
Total	64	40	2	1	1	108

improvement, 4% stationary lesion and 2% progression. There was 1 mortality as previously described (2%).

For some DAVFs with extensive involvement of dural sinuses and cortical veins, repeated radiosurgery might be necessary for the complete obliteration of the DAVFs. In this current series, a total of 5 CS DAVFs and 14 NCS DAVFs had required repeated radiosurgery 1–3 years after the first treatment. The method and dose selection during the second radiosurgery were similar to the first treatment. Figure 5 shows one such case of repeated radiosurgery for an extensive DAVF involving the transverse/sigmoid sinus.

Discussion

Efficacy of Radiosurgery for DAVF

Due to the greater frequency of intraparenchymal AVMs, the efficacy of radiosurgery for treatment of these lesions has been well established [31, 51–53]. While most case series of DAVFs treated with radiosurgery are smaller than those for intraparenchymal AVMs, sufficient data now exist to support radiosurgical therapy for DAVFs. In 1993, Chandler and Friedman [33] published the first detailed, complete case report of a DAVF treated with radiosurgery with angiographically confirmed obliteration. Since then, a number of publications have appeared which establish a role for radiosurgery in treating DAVFs.

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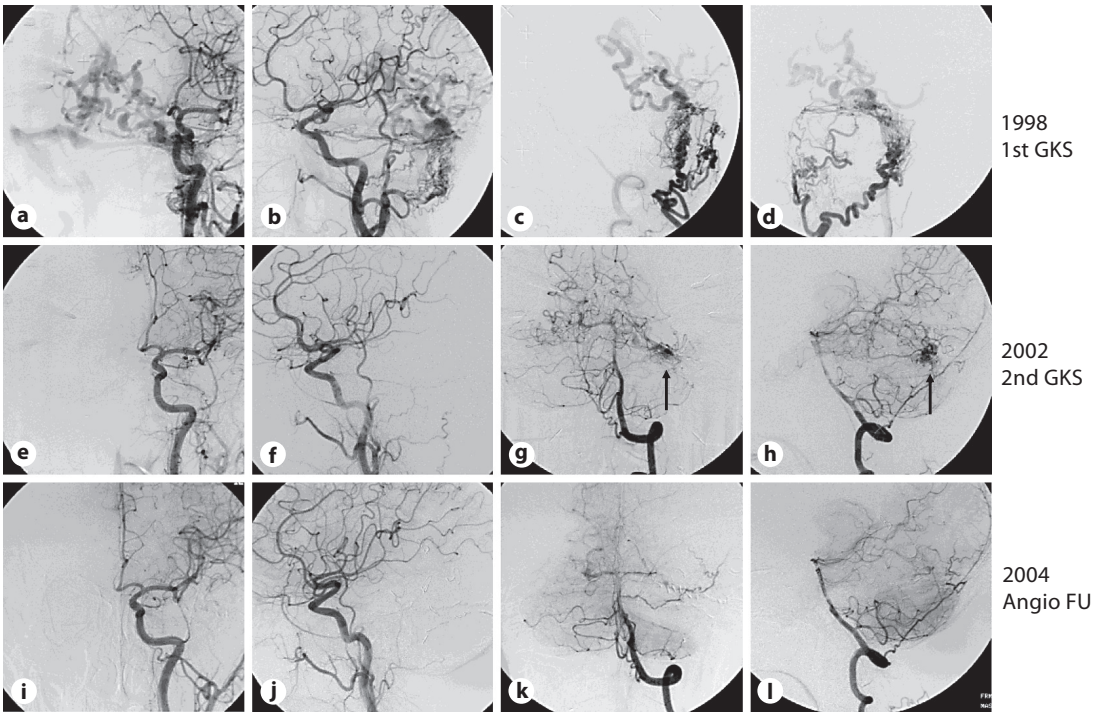

Fig. 5. Common carotid angiogram in anteroposterior (**a**) and lateral views (**b**) showed a Cognard type IIa + b DAVF at the left transverse sinus and torcular region. There were also arterial feedings from muscular branches of the left vertebral artery on anteroposterior (**c**) and lateral vertebral angiograms (**d**). Four years after GKRS, the DAVF was no longer seen on the carotid angiogram (**e**, **f**), but a small residual DAVF nidus (black arrow) supplied by the posterior meningeal artery was noted on the vertebral angiogram (**g**, **h**). A second radiosurgical treatment was performed. Complete obliteration of the DAVF on both carotid (**i**, **j**) and vertebral angiograms (**k**, **l**) was achieved 2 years after the second GKRS. Angio-FU = Angiographic follow-up.

At the Taipei Veterans General Hospital, this current study of 264 DAVF patients treated by GKRS in the past 15 years shows a 66% complete obliteration rate, with another 33% of the patients demonstrating a reduction in DAVF size with improvement in symptoms. These results are similar to those reported by other institutions. Shin et al. [41] reported on 2 patients with tentorial DAVFs treated with dosage greater than 20 Gy to the fistula, with complete obliteration obtained in both patients at 27 and 29 months. Söderman et al. [5] reported on 49 patients with 52 DAVFs, showing a 68% obliteration rate and another 24% with flow regression at 2 years. O'Leary et al. [44] achieved a 77% complete obliteration rate with improvement in another 15% of patients. In the series of Brown et al. [43], of the 50 patients with angiographic follow-up, 68% demonstrated complete obliteration, with another 14% showing near-total obliteration. In that of Koebbe et al. [12], all 18 patients had complete or near complete resolution of their presenting symptoms. Of the 8 patients with angiographic

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follow-up, all patients demonstrated complete obliteration. Although some of the patients in these publications had been treated with surgical resection or endovascular embolization prior to radiosurgery, they were referred for radiosurgery for further management of the residual DAVFs. From these studies we can estimate an overall success rate of complete obliteration associated with radiosurgery of DAVFs at 65–77%, with a greater number of patients gaining symptomatic relief from radiosurgical treatment. Complications directly related to the radiosurgical procedure are rare, as shown in our series.

Rationale of DAVF Radiosurgery

The management for a DAVF should be individualized, taking into consideration the clinical presentation of the patient, the anticipated natural course of the lesion based on location and angioarchitecture of the DAVFs, and the benefit and inherent risk of the treatment modality. It is widely accepted that DAVFs presenting with hemorrhage, progressive neurological deficits or increased intracranial pressure require prompt treatment by endovascular embolization, surgery, or a combination of these procedures, to provide immediate relief of the venous congestion.

For Borden type II/III lesions with a single or few CVDs, or DAVFs with an isolated dural sinus and CVDs, complete obliteration of the lesion may be achieved effectively by surgery or endovascular intervention [47, 54, 55]. However, when DAVFs involve dural sinuses with multiple complex feeders and CVDs, surgical and endovascular treatment can be technically challenging. Multisession and/or combined treatment are often needed. The rate of complete obliteration of DAVFs achieved by endovascular procedures differs from center to center, ranging from 27.3 to 81% [56]. It has been well known that partially treated DAVFs may recruit new collaterals to the fistula or redirect the venous outflow [57].

For DAVFs with antegrade sinus drainage alone (Borden type I) and no progressive focal neurological deficits, intervention may not be necessary unless the patient's symptoms (e.g. headache or pulsatile tinnitus) are intolerable. These lesions generally have a benign course and spontaneous obliteration has been reported, particularly with cavernous sinus lesions [58, 59]. Spontaneous regression of transverse/sigmoid sinus DAVFs is rare (maximum 5% of patients) [14, 15, 56]. It is noteworthy that conversion in flow pattern from antegrade to retrograde can occur in approximately 2% of patients, which leads to higher risks of developing new neurological deficits or hemorrhage [8, 11]. Interestingly, we found a significantly longer duration of nonaggressive symptoms (pulsatile tinnitus) in patients presenting with Borden type II/III DAVFs than in those with Borden type I (average 34 vs. 19 months) in our series. One may hypothesize that with a longer follow-up, the conversion rate of venous drainage from anterograde to retrograde may be higher than 2%.

When a treatment is indicated for Borden type I DAVF, its benefit should outweigh its risk. Evidence has shown that injury or increased pressure in the dural sinus could trigger the development of DAVFs or cause neurological deficits secondary to sinus

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hypertension [2]. Thus sacrificing a functioning dural sinus in Borden type I DAVFs by transvenous intervention or surgery may not be justified. Furthermore, it is difficult to achieve a complete obliteration of Borden type I DAVF by transarterial embolization alone due to the frequently complex and tortuous course of the arterial supply [21]. Studies have shown that persistent sinus hypertension or local ischemia caused by incomplete closure of the DAVFs after endovascular and/or surgical intervention can increase the expression of various vascular growth factors [60–65]. Thus, the use of endovascular intervention or surgery as a first-line treatment for Borden type I DAVFs with the intention of palliation rather than cure should carefully balance the risks and benefits of the procedure. Our study and those of others have shown that DAVFs with benign venous drainage can be safely treated using radiosurgery with a high angiographic complete obliteration rate with preservation of the functioning dural sinuses [19, 35, 39, 42].

Retrograde CVD had long been recognized as an angioarchitecture herald of aggressive behavior of DAVFs. However, not all DAVFs with CVDs were symptomatic. In 2009, Zipfel et al. [20] presented a modification to the existing classification scales of DAVFs based on the observation of patients' symptoms and outcome. They divided Borden type II/III patients into two subgroups: symptomatic CVD cases who present with hemorrhage or progressive neurological deficits, and those with asymptomatic CVD who present incidentally or with symptoms of tinnitus or ophthalmological phenomena with a less aggressive clinical course. According to the study of Zipfel et al., the annual rate of intracerebral hemorrhage is 7.4–7.6% for patients with symptomatic CVD, compared with 1.4–1.5% for those with asymptomatic CVD. They suggest that, for patients with asymptomatic CVD, the newly documented lower risk of subsequent hemorrhage or neurological deficits indicates that a more judicious approach toward therapeutic intervention is warranted. In many cases, endovascular or surgical intervention is still indicated, although the timing of treatment may be performed in a more elective nature. In others, particularly in patients who are elderly, medically frail or harbor complex DAVFs, radiosurgery may be a reasonable alternative.

Our results of radiosurgery support the above observation. In our 48 patients with Borden type II/III NCS DAVFs, 21 (44%) achieved a complete obliteration and 23 (48%) showed a >50% reduction of the lesion size with symptomatic improvement. This data indicates that radiosurgery is also suitable for certain DAVFs with CVD but without immediate risk of hemorrhage or focal neurological deficits.

Conclusions

Stereotactic radiosurgery using the Gamma Knife is a safe and effective alternative for the treatment of DAVFs. This method provides a minimally invasive therapeutic modality for patients who harbor less aggressive DAVFs, but who suffer from intolerable headache, bruit or ocular symptoms. For more aggressive DAVFs associated

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with immediate risks of hemorrhage, severe venous congestion or increased intracranial pressure, initial treatment with endovascular embolization or surgery for prompt elimination of aggressive components of DAVFs is necessary. In such cases, radiosurgery may serve as a secondary treatment for the further management of residual nidus after initial intervention. The latent period for the effects of radiation to occur and the longer time for cure compared to surgery and endovascular therapy remain a major drawback for radiosurgery. However, the gradual obliteration of a DAVF after radiosurgery can avoid the immediate risk of aggravated venous hypertension or infarction, which sometimes complicates endovascular embolization and surgery.

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