Modified Borden Grading System For Cranial And Spinal Dural Arteriovenous Fistulas In Neuroendovascular Era

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Abstract

Objective:

Dural arteriovenous fistulas (DAVFs) is a complex condition in neurovascular surgery. Many DAVF classifications have been reported and have changed over time in the literatures. The purposes of this study was to propose a practical and easy-to-follow grading system for DAVFs.

Methods:

From a retrospective analysis of our database, 143 DAVF patients were consecutively collected. Patients were grouped into modified Borden types I, II and III. Patients' characteristics, treatment and outcomes were analyzed between 3 types. Patients' pre-treatment status(pre-mRS) were analyzed between Borden, Cognard and modified Borden grading systems.

Results:

Male and non-sinus locations were statistically significantly correlated with the type III DAVF type ($p < 0.001$). More than 3 pedicle suppliers and pial arterial suppliers were associated with high grade (type II and III) DAVFs ($p = 0.003$). Worse symptoms were present in most type II and type III patients ($p < 0.001$). Type III DAVF was associated with TAE and type II DAVF was associated with TVE treatment modalities ($p < 0.001$). The results of one-way ANOVA indicated that pre-mRS was significantly different within modified Borden types and Cognard types ($p = 4.3 \times 10^{-6}$ and $p = 1 \times 10^{-4}$, respectively). In terms of pre-mRS, patients were not separated well using Cognard grading systems.

Conclusions:

A modified grading system of cerebral and spinal DAVFs was promoted according to understanding of angioarchitectures in order to evaluate risk of DAVFs and guide the therapies of these lesions. The modified Borden grading system was informative by providing an effective assessment for the risk of patients with simple but precise results.

Background

Dural arteriovenous fistulas (DAVFs) are abnormal connections between dural arteries and dural/pial veins, accounting for 10–15% of all intracranial arteriovenous malformations but they are the most common type of spinal vascular malformations (AVMs)(8). DAVF can develop at everywhere of the dural mater, most common placed transverse-sigmoid sinus and cavernous sinus, following tentorial, sagittal sinus, torcula, anterior cranial fossa, clivus, occipital foramen or dural sleeve of spinal nerve root(8, 23). DAVFs generate a set of clinical signs and symptoms arising from venous hypertension and pial venous rupture(3, 5, 8). Patients with unruptured DAVFs may remain asymptomatic or develop headache, diplopia, proptosis, epilepsy, dementia, neuropsychiatric disturbances, or myelopathy(1, 2, 10, 12, 19, 20,

24). The importance of venous drainage in the clinical presentation of DAVFs is well known when they distinguished between drainage directly into the dural sinuses and the pial veins (Table 1) (3, 5, 8).

Table 1
Previous classifications of DAVFs and 3 modified Borden types.

Djindjian-Merland type (1977) (8)	Borden type (1995) (3)	Cognard type (1995) (5)	modified Borden types (2021)	Symptoms
I, drainage into a sinus	I, venous drainage directly into dural venous sinus or meningeal vein	I, venous drainage into dural venous sinus with antegrade flow	I, DAVFs drain directly into dural venous sinuses; spinal extra-dural AVM without perimedullary vein reflux	No symptom or tinnitus
		IIa, venous drainage into dural venous sinus with retrograde flow	II, DAVFs drain into dural sinuses but also have retrograde drainage into ophthalmic and bridging veins; spinal extra-dural AVM with perimedullary vein reflux	Proptosis, chemosis, headaches, epilepsy, cognitive dysfunction, dementia, intracranial hemorrhage, myelopathy
II, sinus drainage with reflux into cerebral veins	II, with cortical vein reflux	IIb, venous drainage into dural venous sinus with cortical vein reflux		
III, drainage solely into cortical veins	III, cortical vein drainage	III, venous drainage directly into cortical veins	III, DAVFs drain into pial veins and do not have dural sinus drainage; spinal DAVF drained with perimedullary vein only	intracranial hemorrhage, myelopathy
IV, with supra or infra tentorial venous lake		IV, type III with venous ectasias of the draining subarachnoid veins		
---	---	V, spinal perimedullary vein drainage	II or III, cranial or spinal DAVF	intracranial hemorrhage, myelopathy
Cavernous fistula-modified Borden type I (petrosal sinus drainage) and II (ophthalmic vein drainage or cortical reflux)				
Spinal DAVF-modified Borden type II (extradural dural fistula) and III (intradural dural fistula)				

The Borden and Cognard classifications constitute the most well-known classification schemes predicting aggressiveness of intracranial DAVFs (3, 5). Borden has proposed a classification system that

unifies spinal and cranial DAVFs based on their surgical practices(3). The Borden classification designate types I, II, and III lesions as those with dural venous drainage without cortical venous reflux, dural venous drainage with cortical venous reflux, and cortical venous drainage without dural venous drainage, respectively. The Cognard classification designates types I, IIa, IIb, IIa + b, III, and IV lesions as those antegrade dural venous drainage without cortical venous drainage (type I), retrograde dural venous drainage without cortical venous reflux (type IIa), antegrade dural venous drainage with cortical venous reflux (type IIb), retrograde dural venous drainage with cortical venous reflux (type IIa + b), cortical venous reflux without dural venous drainage (type III), cortical venous reflux with venous ectasias (type IV), and cervical perimedullary venous drainage (type V). Borden types II and III and Cognard types IIb, IIa + b, III, IV, and V dural AVFs constitute aggressive lesions which must be treated(5).

Classifications developed by Borden et al and Cognard et al have both focused chiefly on drainage direction of DAVFs excluding direct carotid cavernous fistulas (3, 5). Though adequately predicting extent of lesional flow and properly categorizing the lesions angioarchitecturally, these classifications lack of correlation with clinical presentation, natural history, and hemorrhagic risk. While the Cognard system is more complicated to use, it can be changed. Therefore, we proposed a classification of DAVFs, which was modified from Borden grading system (Table 1).

Methods

The records of 143 patients with cranial and spinal DAVFs admitted to our institutions from 2014 to 2018 were retrospectively reviewed. This study was approved by ethics committee of Beijing Tsinghua Changgung Hospital and patients' consent was obtained. Among 143 patients, 120 treated and 23 untreated patients were followed up from 0 to 50 months (mean, 29 months). We defined patients' symptoms into incidental, pulsatile tinnitus, cranial nerve symptoms(oculomotor nerve palsy, trigeminal neuralgia, facial nerve spasm), venous sinus hypertension (ie, headache, visual deficit, ocular proptosis, focal neurological deficits, seizures, altered mental status, and ascending myelopathy) and intracranial hemorrhage. Worse symptoms were defined as cranial nerve symptom, venous sinus hypertension and intracranial hemorrhage. Benign symptoms were defined as asymptomatic and pulsatile tinnitus. Cure embolization was defined as no contrast radiopaque of the lesion and partial embolization was defined as any residual arteriovenous shunts of lesion after treatment. Patients were divided into 3 types as following.

Modified Borden Classifications of DAVFs

Type I DAVFs are those that drain into the dural sinus with antegrade venous flow, e.g. the flow of the veins draining from the parenchyma or spinal cord into the dural sinuses or epidural veins is anterograde, a CCF without cortical or SOV reflux.

Type II DAVFs drain into dural sinus with retrograde venous flow, a CCF with SOV reflux without cortical reflux. Intracranial Type 2 DAVFs can drain into spinal perimedullary veins.

Type III DAVFs are those that drain into the pial veins and the spinal coronal or perimedullary veins, a CCF with cortical drainage. Type 3 spinal DAVFs can drain intracranially.

Clinical status evaluation

Pre-treatment and follow-up clinical outcomes were graded by modified Rankin Score scale(mRS): 0, no symptoms at all; 1, no significant disability despite symptoms(able to carry out all usual duties and activities); 2, slight disability(unable to carry out all previous activities, but able to look after own affairs without assistance); 3, moderate disability(requiring some help, but able to walk without assistance); 4, moderately severe disability(unable to walk and attend to bodily needs without assistance); 5, severe disability(bedridden, incontinent and requiring constant nursing care and attention) and 6, death. Benign clinical outcome was defined 0 and 1 scores and worse clinical outcome was defined 3 to 6 scores.

Statistical analysis

The modified Rankin Scale before treatment (i.e., pre-mRS) was applied to assess the preoperative risk of the patients. The comparison of pre-mRS was made among three grading systems (i.e., modified Borden, Borden, and Cognard grading system). The violin plots were used to display the distributions of pre-mRS within types of each grading system. For each grading system, pre-mRS within types were compared using one-way ANOVA where the dependent variable was the pre-mRS, and the independent variable was the type. In addition to pre-mRS, we also compared the differences of other variables (such as age, sex, location, supplier, presentation, complication, treatment, result, post-mRS) within types in the modified Borden grading system. Categorical variables were compared using Fisher exact test and continuous variables were compared using one-way ANOVA. Significant difference was defined as $p < 0.05$.

Results

Among the 143 DAVF patients, 46(32.2%) were female and 97(67.8%) were male. Patients' age ranged from 5 to 74 years (mean, 46 ± 15 years). Aggressive symptoms (cranial nerve symptom, venous sinus hypertension and intracranial hemorrhage) were present in 124 (86.7%) patients. The distributions of DAVFs were cavernous sinus in 40(28.0%) patients, transverse-sigmoid sinus in 39(27.3%) patients, tentorium in 30(21.0%) patients, anterior cranial fossa in 11(7.7%) patients, sagittal sinus in 9(6.3%) patients, occipital foramen or clivus in 4(2.8%) patients and spine in 9(6.3%) patients. Venous drainage was type 1 in 9(6.3%) patients, type 2 in 66(46.2%) patients and type 3 in 68(47.5%) patients according to modified Borden classification. Twenty-three patients were not treated and 120 patients were treated by transarterial embolization(TAE) in 86 patients, transvenous embolization(TVE) in 22 patients, a combination of two modalities in 3 patients, open surgery in 2 patients and radiosurgery in 7 patients. The overall cure rate was 70% (84/120) and with 30%(36/120) resulted in partial embolization. Favorable clinical outcome was presented in 127 (88.8%) patients and unfavorable clinical outcome was presented in 16 (11.2%) patients at follow-up.(Table 2)

Table 2
Symptoms and locations of DAVFs in 143 patients.

	Typel(n = 9)	Typell(n = 65)	Typelll(n = 68)
Symptoms			
Incidental	3	2	5
Pusatile tinnitus	5	5	3
Cranial nerve symptom	0	4	
Venous sinus hypertentiom	0	59	32
Hemorrhage	0	0	24
Locations			
Cavernous sinus	3	36	1
Transverse-sigmoid sinus	2	23	14
Sagittal sinus	2	4	3
Anterior cranial fossa	1	1	9
Tentorium	0	0	30
Occipital foramen or clivus	0	1	3
Spine	0	1	8

Type I

Nonaggressive symptoms (asymptomatic and pulsatile tinnitus) were present in 6/9 patients. However, cranial nerve symptom was present in 2 patients and sinus hypertension in 1 patient. The locations of Type I DAVFs were 3 cavernous sinus, 2 transverse sinus, 2 superior sagittal sinus and 1 anterior cranial fossa. Five patients were not treated, 3 patients were treated by TAE and 1 was treated by TVE to relieve tinnitus symptoms(Fig. 1). Three patients resulted in cure embolization with 1 partial embolization. All 9 patients were favorable in clinical outcome at follow-up.

Type II

In the 66 patients with type II drainage, nonaggressive symptoms were present in 7 patients and aggressive symptoms (cranial nerve symptom and venous sinus hypertension without intracranial hemorrhage) in 59 (89.4%) patients. DAVFs of type II were located: 36 cavernous sinus, 23 transverse sinus, 4 superior sagittal sinus, 1 anterior cranial fossa, 1 occipital foramen and 1 spine. Eleven patients were not treated and 55 patients were treated by TAE in 29 patients(Fig. 2), TVE in 19 patients, a combination of two modalities in 2 patients, and radiosurgery in 1 patient. There was no open surgery performed for type II DAVFs. The overall cure rate was 67.3% (37/55) and with 32.7%(18/55) resulted in

partial embolization. Favorable clinical outcome was presented in 61 (92.4%) patients and unfavorable clinical outcome was presented in 5 (7.6%) patients at follow-up.

Type III

Sixty-eight patients had type 3 DAVFs. Fifty-nine (86.8%) had aggressive symptoms (cranial nerve symptom, venous sinus hypertension and intracranial hemorrhage) and nonaggressive symptoms in 9 (13.2%) patients. DAVFs of type III were 30 tentorium(Fig. 3), 1 cavernous sinus, 14 transverse sinus, 3 superior sagittal sinus, 9 anterior cranial fossa, 3 occipital foramen or clivus and 8 spine(Fig. 4). Seven patients were not treated and 61 patients were treated by TAE in 53 patients, TVE in 3 patients, a combination of two modalities in 1 patients, open surgery in 2 patients and radiosurgery in 2 patients. The overall cure rate was 72.1% (44/61) and with 27.3%(17/61) resulted in partial embolization. Favorable clinical outcome was presented in 57 (83.8%) patients and unfavorable clinical outcome was presented in 11 (16.2%) patients at follow-up.

Statistical analysis results

No correlation was observed between age and modified Borden types($p = 0.782$). There was a male predominance of 67.8% in our patients and a correlation was observed between male and type III DAVF($p < 0.001$). Location of the DAVF was statistically significantly correlated with the DAVF types ($p < 0.001$). In our series, type III was associated with non-sinus and spinal DAVFs but type I and II were associated with sinus DAVFs. More than 3 pedicle suppliers and pial arterial suppliers were associated with high grade (type II and III) DAVFs($p = 0.003$). Worse symptoms were present in most type II and type III patients($p < 0.001$). However, intracranial hemorrhage was only presented in type III patients (35.3%). There was no difference in complication rate between 3 DAVF types($p = 0.934$). Type III DAVF was associated with TAE and type II DAVF was associated with TVE treatment modalities($p < 0.001$). There was no difference in complete obliteration rate between 3 DAVF types($p = 0.094$).(Table 3)

Table 3

Statistical analysis of DAVF characteristics, treatment and outcome between 3 modified Borden types.

Characteristics	Value	Type I (n = 9)	Type II (n = 66)	Type III (n = 68)	P-value ¹
Age	Years, mean $\pm$ SD	43 $\pm$ 17	47 $\pm$ 16	46 $\pm$ 13	0.782
Sex	Female	4	32	10	< 0.001
	Male	5	34	58	
Location	Sinus	7	63	18	< 0.001
	Non-sinus	2	2	42	
	Spinal	0	1	8	
Supplier	ECA,internal costal	4	12	21	0.003
	ECA,ICA	1	37	20	
	ICA,VA	2	4	5	
	ICA	2	2	9	
	ECA,ICA,VA	0	6	1	
	ECA,ICA,VA,pial	0	2	0	
	ECA,ICA,pial	0	1	6	
	ECA,pial	0	2	2	
	VA	0	0	1	
	VA,pial	0	0	1	
	Pial	0	0	2	
Presentation	benign	9	7	12	< 0.001
	Worse	0	59	56	
Complication	Yes	1	10	12	0.934
	No	8	56	56	
Treatment	TAE	3	29	53	< 0.001
	TVE	1	19	3	
	TAE + TVE	0	2	1	
	Radio, surgical	0	5	4	
	Untreated	5	11	7	

Characteristics	Value	Type I (n = 9)	Type II (n = 66)	Type III (n = 68)	P-value ¹
Result	Complete	3	37	44	0.094
	Subtotal	1	2	5	
	Partial	1	17	12	
	Untreated	4	10	7	
Post-mRS	Score, mean $\pm$ SD	0.67 $\pm$ 0.5	0.65 $\pm$ 0.83	1.16 $\pm$ 1.89	0.109

¹ The p-values are computed from Fisher exact tests (for categorical variables) and one-way ANOVA (for continuous variables). Bold indicates significant differences ($p < 0.05$) within three modified Borden types in terms of the corresponding variable. ECA, dural branches of external carotid artery; ICA, dural branches of internal carotid artery; VA, dural branches of vertebral artery; TAE, transarterial embolization; TVE, transvenous embolization; mRS, modified Rankin Score scale.

The results of one-way ANOVA indicated that pre-mRS was significantly different within modified Borden types and Cognard types ($p = 4.3 \times 10^{-6}$ and $p = 1 \times 10^{-4}$, respectively) but not significantly different within Borden types ($p = 0.24$). The means (standard errors) of pre-mRS of modified Borden types are 0.78 (0.67), 2.24 (0.75), and 2.35 (0.97) in turn. The means (standard errors) of pre-mRS of Borden types are 2.07 (0.83), 2.1 (0.96), and 2.34 (0.98) in turn. The means (standard errors) of pre-mRS of Cognard types are 0.78 (0.67), 2.22 (0.69), 2.29 (0.92), 2.41 (1), 2.18 (1.02), and 3 (0) in turn. Apparently, in terms of pre-mRS, patients were not separated well using Borden grading system (Fig. 5).

We further compare the modified Borden grading system and the Cognard grading system. The violin plots in Fig. 5 demonstrated that the patterns of the distributions of pre-mRS for 3 grading systems were very different. For different modified Borden types, the pre-mRS concentrated in values of 1, 2, and 3, respectively, and there is very little overlap between the three distributions. In contrast, pre-mRS is not highly distinguishable for different Cognard types, and there is considerable overlap. Nine spinal DAVF patients did not fall into any of the Cognard types (i.e. "none" in Fig. 5). Moreover, the modified Borden grading system was simple since it only had 3 types, while there were 5 types in the Cognard grading system. Pre-mRS was monotonous in the modified Borden grading system but was not in the Cognard grading system. Therefore, the modified Borden grading system was more informative than Cognard's by providing an effective assessment for the risk of patients with simple but precise results.

Discussion

In this study, cranial and spinal DAVFs are classified into 3 modified Borden types based on venous drainage and hemodynamic pathology. Type I lesions exhibit a benign clinical course. Patients harboring type II lesions usually present intracranial hypertension. Hemorrhagic ruptured occurred in more than 30% of patients harboring type III DAVFs. Type II and III spinal DAVFs exhibiting drainage into spinal perimedullary veins might present progressive myelopathy in all of cases. Some spinal DAVF patients

characterized by spasticity of muscular hypertonia caused by the upper motor neuron syndrome, an alteration where the inhibitory influence of supraspinal structures is lost.

Type I DAVFs can be treated by TAE and major sinus drainage needs to be preserved. Since type II DAVFs usually have symptoms of venous hypertension, endovascular techniques are option being associated with moderate risk and should be used in seletive patients. When the venous drainage is sacrificed safely, the DAVF can be cured by the TVE occlusion. Type III DAVFs are cured by TAE embolization of the pial draining vein. Contrast to type I DAVFs, which are usually benign, types II and III DAVFs presented a high proportion of worse clinical status before treatment. Their pathophysiology was consistent as venous congestion and hypertension. Type II and III DAVFs can be successfully treated with endovascular treatment.

The cranial and spinal dura consists of two layers: the outer is periosteal layer, and the inner is meningeal layer(3). The dural sinuses are located between the two fused layers of cranial dura. There are 2 specific locations, such as the orbit and the spine. The intra-orbital veins are located in the space between the orbital periosteum and ocular nerves sheath dura and the spinal epidural veins are located in the spinal canal between the spinal periosteum and meningeal dura. Thus, the intraorbital venous plexus and spinal epidural veins correspond to the dural sinuses.

Classifications developed by Borden et al and Cognard et al have both inadequately distinguish between anterior and posterior drainage in the cavernous sinus region, which is the most common DAVF location(4, 11, 16, 25, 27). We define cavernous sinus DAVFs drained only inferior petrosal sinus and spinal extradural DAVFs only involving epidural venous plexus as type I DAVFs. Type I DAVFs are often asymptomatic or bruits. We classify cavernous sinus DAVFs with drainage into ophthalmic veins as type II DAVFs, which could dictate treatment planning and clinical correlation. Cavernous sinus DAVFs that drain retrogradely into the intraorbital/ophthalmic veins with or without reflux to the Sylvian veins are classified as type II DAVFs because the intraorbital veins corresponds to dural sinuses(16). Spinal DAVFs that drain into the epidural venous plexus with reflux to the perimedullary veins are classified as type II DAVFs because the epidural plexus corresponds to dural sinuses and the intradural perimedullary veins are pial veins. Intra-orbital arteriovenous fistula exhibit an exceedingly low incidence and prevalence, presenting chiefly with proptosis and chemosis and also classifying type II.

We classify as type III DAVFs that first drain into pial veins with or without giant venous aneurysm (Cognard's type III and IV and Borden's type III) because their natural history and treatment are the same as for other type III DAVFs. However, those cranial DAVFs draining into spinal perimedullary veins (Cognard type V) are classified as Type II or III because perimedullary veins are drained from intracranial dural venous sinuses or pial brain stem veins(10, 19). The most common group of type III DAVFs are intracranial tentorial DAVFs(6, 18). The most common spinal DAVFs are type III DAVFs supplied by dural branches of a radicular artery and drained into the perimedullary venous plexus(22). Spinal DAVFs drained into intradural spaces causing spinal venous hypertension and myelopathy. In type III patients,

the venous ectasia can cause intracranial hemorrhage, supratentorial ventricular dilatation and trigeminal neuralgia and facial nerve spasm.

When a DAVF drains into a major dural sinus that cannot be safely occluded, it is often difficult to cure the DAVF, especially if it has multiple fistulas(15). The hemorrhagic complication risk of a major dural sinus sacrifice combined with the benign natural history of type I or II DAVFs makes this procedure an unwarranted option. When the sinus must be preserved and the arterial supply cannot be completely eliminated, our treatment goal should aim to restore the hemodynamic of brain or spine circulation, eg. the dural arteriovenous shunts can be significantly reduced by endovascular embolization.

Type III DAVFs can be fed by vessels that also supply important structures(17). For example, DAVFs at the anterior cranial fossa are fed by the ethmoidal branches of the ophthalmic artery; DAVFs of the tentorium are fed by meningohipophyseal trunk branches of the ICA, and spinal DAVFs are fed by dural branches of the radicular arteries adjacent to the radiculomedullary arteries. When these arteries are embolized, there is a risk of neurological deficits. Inadvertent embolization of the cavernous and petrosal branches of the middle meningeal artery which triumphantly emerges into the middle cranial fossa through foramen spinosum may precipitate infarctions of the trigeminal and/or facial nerve(9, 14, 21). As to the cavernous sinus DAVFs, the cause of post-hemorrhagic complication was attributed to the venous perforations and hemodynamic changes in cavernous sinus flow constituted the chief causes of post-procedural intracranial hemorrhagic rupture(7, 25, 26). Among patients harboring pial artery supply, giant venous pouch and high volume Onyx injection in one session constituted the chief causes post-procedural hemorrhagic complications(13). When collateral arteriovenous shunts exist between dural and pial arterial suppliers, the injection force can cause Onyx reflux across these collateral vessels into the brain, which should give cautions.

Conclusion

Borden and Cognard classifications did not cover cavernous DAVF definitely, which is very common DAVF. In this study, a modified Borden grading system to risk stratify cerebral and spinal DAVFs was promoted according to understanding of angioarchitectures in order to guide the appropriate evaluation of DAVFs and therapies of these lesions.

Declarations

•ETHICS APPROVAL AND CONSENT TO PARTICIPATE

All procedures performed in the studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards.

Informed consent was obtained from all individual participants included in the study.

•CONSENT FOR PUBLICATION

Not applicable

•AVAILABILITY OF DATA AND MATERIALS

Please contact author for data requests.

•COMPETING INTERESTS

The authors declare that they have no competing interests.

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Figures

Figure 1

A 69-year-old man presented with pulsatile tinnitus. A, left external carotid artery angiogram(lateral view) showed a modified Borden type 1 signoid sinus dural fistula supplied by dural branches of the external carotid artery(arrow). B, left internal carotid artery angiogram (lateral view) showed the dural fistula also supplied by dural branches of the internal carotid artery. C, the carotid artery angiogram (lateral view) showed the fistula was accessed through the venous side(arrow). D, control angiogram (lateral view, late arterial phase) showed the dural fistula was completely occluded.

Figure 2

A 56-year-old male patient presented with headaches caused by venous sinus hypertension. A, left external carotid artery angiogram (later view). B, left external carotid artery angiogram (frontal view). Showed a modified Borden type 2 transverse sinus dural fistula(white arrows) supplied by the middle meningeal artery branches with 2 feeding artery aneurysms(2 arrowheads) and transverse sinus reflux(black arrows). C, the left carotid artery angiogram (lateral view) showed the dural fistula was completely occluded.

Figure 3

A 51-year-old patient presented with severe neurological deficits caused by venous sinus hypertension. A, CT scanning showing hydrocephalus and a giant venous pouch at the quadrigeminal cistern (arrow). B, the left external carotid artery angiogram (lateral view) showing a modified Borden type 3 dural fistula supplied by the middle meningeal artery. C, the left vertebral artery angiogram (lateral view) showing the dural fistula supplied by the posterior cerebral arteries and the posterior meningeal arteries. The dural fistula was drained directly by pial veins with a giant venous pouch. D, super selective angiogram (lateral projection) showed the microcatheter was accessed to the fistula through the left middle meningeal artery (arrow). E, control angiogram of the left vertebral artery showed the dural fistula was nearly completely occluded. F, unsubtracted image (frontal projection) showed the Onyx was injected to occlude the fistula. G, CT scanning at 6-month follow-up showing the relieving of the hydrocephalus and the patient returned to independent daily life.

Figure 4

A 57-year-old patient presented with myelopathy caused by spinal venous hypertension. A, frontal view of the intercostal artery angiogram showed a modified Borden type 3 dural fistula supplied by the dural branches of the radicular artery(arrow). B, unsubtracted image showing the Onyx(black arrow) was injected through the microcatheter to occlude the fistula. C, control angiogram of the intercostal artery showed the dural fistula was completely occluded.

Figure 5

Violin plots of modified Rankin Scale before treatment under three classifications. The horizontal line represents the median of the distribution.