Acta Neurochir (2005) [Suppl] 94: 123–126

Printed in Austria

Hemodynamic status and treatment of aggressive dural arteriovenous fistulas

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Summary

In this study the hemodynamic status and treatment modality of aggressive dural arteriovenous fistulas (dAVFs) was evaluated.

Of 145 intracranial dAVFs treated in our clinic, there were 38 aggressive lesions presenting with hemorrhage, infarction, seizures, and symptoms of increased intracranial pressure. They included 3 (5% of all cavernous sinus lesions) cavernous sinus, 24 (44%) transverse-sigmoid and superior sagittal sinus, and 11 (46%) direct cortical types of dAVFs.

Of these 38 aggressive lesions, retrograde leptomeningeal venous drainage was disclosed in 35 lesions, and retrograde sinus drainage in 3. Eighteen cases were treated only with endovascular procedures, 7 with surgical interventions, and 13 with combined endovascular and surgical procedures. Angiographic results were complete obliteration in 66% of the cases, subtotal and partial obliteration in 34%. Clinical outcome was GR (good recovery) in 58% of cases, MD (moderate disability) in 18%, SD (severe disability) in 13%, VS (vegetative state) in 8%, and D (death) (due to acute cardiac infarction) in 3%. Symptomatic procedural complication occurred in 3 cases.

In conclusion, aggressive dural AVF resulted from retrograde leptomeningeal venous drainage. Combined surgical and endovascular treatment played the leading part in the management of this aggressive type of lesion.

Keywords: Dural arteriovenous fistula; endovascular treatment; surgical treatment.

Introduction

Dural arteriovenous fistulas (AVFs) are a rare clinical entity which accounts for 12% of intracranial arteriovenous malformations (AVMs) [14].

Unlike cerebral AVMs, dural AVFs are now considered an acquired lesion [4]. The etiology, however, still remains unknown in most cases. Clinical symptoms of the patients relate greatly to the increased venous pressure, which is considered to be the essential pathophysiology of this disease. It is well known that dural AVFs sometimes behave aggressively depending on the pattern of venous drainage [1], resulting in intracranial hemorrhage, venous infarction, increased intracranial pressure, and status epilepticus.

The purpose of this study is to evaluate the hemodynamic status and treatment modality of these aggressive dural AVFs.

Patients and method

Of 145 intracranial dAVFs which were treated in our clinic from 1990 to 2003, there were 38 aggressive lesions (26% of all cases) which presented with intracranial hemorrhage, venous infarction, convulsions, or progressive neurological deficits resulting from increased intracranial pressure.

They included 3 cavernous sinus lesions (5% of all cavernous sinus cases), 24 transverse-sigmoid and superior sagittal sinus lesions (44% of cases in these locations), and 11 direct cortical type dAVFs (46% of cases with this type) which included fistulas involving the convexity (2 cases), anterior cranial base (4 cases), craniocervical junction (4 cases), and tentorium (1 case).

Patterns of venous drainage were evaluated, focusing particularly on the retrograde leptomeningeal venous and retrograde sinus drainage (RLVD, RSD). Treatment modalities and results were also evaluated retrospectively.

Results (Table 1)

RLVD or RSD was observed in the preoperative angiogram in 60 of the 145 patients.

Among these 60 patients, 38 lesions showed the aggressive course including intracranial hemorrhage (24 cases), venous infarction (5 cases), seizures (3 cases), and symptoms of increased intracranial pressure (6 cases). Thirty-five lesions were associated with RLVD, and 3 lesions with RSD. No aggressive clinical courses were observed in the patients not associated with RLVD or RSD.

Of these 38 patients with an aggressive lesion, 18 were treated with endovascular procedures (transarterial embolization (TAE), transvenous embolization (TVE), or combination of these procedures), 7 were treated solely with surgical intervention (discontinuing the draining veins), and 13 with combined endovascu-

Table 1. *Angiographic and clinical features and results of treatment of 38 patients with aggressive dAVF*

Case	Age	Sex	Location	Presentation	Lraining pattern	Treatment	% Obliteration	Complication	GOS	Remarks
1	60	M	ACB	SDH	RLVD	operation	100		GR	
2	65	M	ACB	ICH	RLVD	operation	100		GR	
3	68	M	ACB	ICH	RLVD	operation	100		GR	
4	58	M	ACB	ICH	RLVD	operation	100		VS	poor basic condition
5	73	M	CCj	SAH	RLVD	TAE	20		VS	poor basic condition
6	52	F	CCj	SAH	RLVD	TAE	100	permanent #1	VS	retroperitoneal bleeding
7	65	M	CCj	SAH	RLVD	TAE	50	permanent #2	SD	NBCA migration
8	52	M	CCj	CI	RLVD	operation	100		SD	
9	70	M	CV	CI	RLVD	TAE	100		MD	
10	41	M	CV	ICH	RLVD	operation	100		MD	
11	60	M	Tent	SAH	RLVD	operation	100		MD	
12	48	M	CS	ICH	RLVD	surgical TVE	100		SD	
13	82	F	CS	ICH	RLVD	TVE	90		MD	
14	69	M	CS	ICH	RLVD	TAE	90		GR	
15	69	M	SSS	ICH	RLVD (bil)	surgical TVE	100		GR	
16	60	M	TS	ICH	RLVD	surgical TVE	100		GR	
17	70	M	TS	ICH	RLVD	surgical TVE	100		MD	
18	47	M	TS	ICH	RLVD	surgical TVE	100		SD	
19	63	M	TS/SSS	ICH	RLVD (bil)	surgical TVE	90		D	AMI 10 days after treatment
20	66	M	TS	ICH	RLVD	surgical TVE	100		MD	
21	56	F	TS	ICH	RLVD	TAE/operation	100		GR	
22	78	F	TS	ICH	RLVD (bil)	TVE	100		SD	poor basic condition
23	72	M	TS	ICH	RLVD	TAE	100	transient #3	GR	lower cranial nerve palsy
24	78	M	TS	ICH	RSD	TVE	100		GR	
25	61	M	TS	ICH	RLVD	TAE/operation	90		GR	
26	87	F	TS + confluence	SAH, dementia	RLVD	TAE/TVE	50		MD	poor basic condition
27	64	F	TS	CI	RLVD	surgical TVE	100		GR	
28	67	M	TS	CI	RSD	TVE	90		GR	
29	60	M	SSS	drop attack (CI)	RLVD (bil)	surgical TVE	100		GR	
30	70	M	SSS/TS	dementia	RLVD (bil)	TAE/TVE	50		GR	
31	51	M	TS (bil)	dementia	RLVD (bil)	TAE	50		GR	
32	82	F	TS (bil)	dementia	RLVD/RSD	TVE	50		GR	
33	71	F	TS	dementia	RLVD	TAE/TVE	100		GR	
34	53	M	TS	VI	RLVD	TAE	100		GR	
35	74	M	TS + confluence	VI	RSD	TAE/TVE	50		GR	
36	57	M	SSS	SE	RLVD	TAE	90		GR	
37	75	M	TS	SE	RLVD	surgical TVE	100		GR	
38	61	F	TS	SE	RLVD	surgical TVE	100		GR	

SDH Subdural hematoma, *ICH* intracerebral hematoma, *SAH* subarachnoid hemorrhage, *CI* cerebral infarction, *SE* status epilepticus, *VI* visual impairment, *RLVD* retrograde leptomeningeal drainage, *RSD* retrograde sinus drainage, *TAE* transarterial embolization, *TVE* transvenous embolization, *AMI* acute myocardial infarction, *#1* Retroperitoneal bleeding, *#2* PCA embolism due to NBCA migration, *#3* lower cranial nerve palsy.

lar and surgical procedures (11 surgical TVEs, and 2 TAEs followed by surgical intervention).

Angiographic results were complete obliteration in 25 patients (66% of aggressive lesions), subtotal obliteration in 6 (16%), partial obliteration in 7 (18%). Clinical outcome was GR in 22 patients (58%), MD in 7 (18%), SD in 5 (13%), VS in 3 (8%), and D in 1 (3%, due to acute myocardial infarction). Thus, favorable

angiographic results were obtained in 31 (82%), and favorable clinical outcome in 29 (76%) patients.

Symptomatic procedural complications were observed in 3 patients including one showing a transient ischemic cranial nerve symptom after glue injection, one suffering a permanent hemiparesis due to glue migration to the parent vertebral artery, and the other one with neurologic deterioration caused by a massive retroperitoneal bleeding.

Discussion

The recent development of neuroendovascular techniques enables us to treat dural arteriovenous fistulas very safely and effectively. However, we have sometimes experienced complicated lesions which were not easy to treat. These lesions sometimes behave aggressively causing intracranial hemorrhage, venous infarction, seizures, and symptoms of increased intracranial pressure. Hemodynamic features of these aggressive dAVFs are quite essential to consider the symptoms and evaluate the treatment modalities of this disease.

Aggressive clinical course

Dural AVF is a disease of which pathophysiology is thought to be based on the abnormality of the venous side [13]. Its symptoms, therefore, are also related with the abnormal venous conditions. Antegrade venous drainage usually does not behave aggressively, only sometimes causing vascular bruit (pulsatile tinnitus) which is not basically life-threatening. As clearly shown in this series, aggressive clinical courses resulted from venous hypertension caused by retrograde leptomeningeal venous or retrograde sinus drainage (RLVD, RSD).

In a review of the literature, Awad *et al.* [1] have clarified that leptomeningeal venous drainage, variceal or aneurysmal venous dilatation, and galenic drainage were significant factors as predisposition for this aggressive neurological course. Brown, *et al.* [3] reported a long-term follow-up study of 54 patients with dural AVFs and concluded that a significant predictor for intracranial hemorrhage was venous varix on a draining vein. Lesions draining into leptomeningeal veins (RLVD) also had an increased occurrence of hemorrhage. There have been several reports [2, 5, 9, 15, 16] where pure leptomeningeal drainage without involvement of dural sinus (direct cortical type in our series) was emphasized as a potential risk of bleeding. Sinus occlusion is another potential factor to modify the venous drainage. Ishii *et al.* [11] identified a subgroup of patients with a high risk of hemorrhage and dementia due to a severe venous overload through occlusive changes of transverse-sigmoid sinus. A recent report clarified the annual risk of hemorrhage (8.1%) and annual mortality rate (10.4%) in cases with long-term persistent cortical venous drainage [6]. Rebleeding risk (35% within 2 weeks) has also been emphasized in the lesions with RLVD [7].

Treatment

Transarterial embolization (TAE) with glue is effective to reduce the arterial inflow, but it is sometimes difficult to obtain complete and permanent obliteration of the shunt in this way. Transvenous embolization (TVE) [10, 17] is now recognized as one of the most effective and radical treatments of dural AVFs and may be an alternative to standard surgical treatment for many patients, particularly for the majority of the cases with cavernous sinus lesion. However, microcatheters sometimes cannot access the lesions transvenously with isolated sinus and those with pure leptomeningeal venous drainage without sinus involvement (direct cortical type). These are exactly the lesions causing the aggressive neurological course. We reported the efficacy of the direct sinus packing [8] for patients with isolated transverse-sigmoid sinus dural AVFs, and surgical transvenous embolization [12] for patients with cavernous sinus lesion draining only into the cortical veins. It is well known that transvenous catheterization is sometimes successful for lesions in which no draining access route is opacified on the angiogram. But in cases of unsuccessful percutaneous transvenous approach, the surgical TVE as reported by us is very effective to access and cure the lesion. We believe it plays the leading role in the management of aggressive dAVFs.

Conclusions

1) Of 145 cases with intracranial dural AVF, there were 38 aggressive lesions which presented with hemorrhage, infarction, seizures, and symptoms of increased intracranial pressure.

2) Retrograde leptomeningeal venous drainage was disclosed in most cases and thought to be a causative factor of the aggressive behavior.

3) Half the patients were treated solely with endo-

vascular procedure, and the remaining patients were managed surgically alone or with combined endovascular and surgical procedures.

4) Treatment results and clinical outcomes were favorable in most cases of multimodal treatment.

5) The aggressive dural AVF should be treated flexibly with endovascular, surgical, or combined procedures. The importance of teamwork between neuroradiology, neurology, neuroanesthesiology, and neurosurgery should be emphasized.

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Cerebral revascularization