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Evolution of Different Therapeutic Strategies in the Treatment of Cranial Dural Arteriovenous Fistulas – Report of 30 Cases

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Summary

30 cases of cranial dural arteriovenous fistulas, treated between 1983 and 1992, are reported. Twelve presented with an aggressive clinical couse including intracranial haemorrhage, progredient neurological deficit, medically intractable seizures, and cerebellar symptoms. The other 18 patients had a more benign clinical presentation with audible bruit, exophthalmus, chemosis, and cranial nerve dysfunction. One of the latter had symptoms of pseudotumour cerebri due to sinus occlusion with contralateral sinus stenosis. The most common location was at the transverse sinus, followed by the cavernous sinus, the tentorial ring, and the orbita. Four vessel angiography verified the diagnosis and demonstrated all fistulas, mainly supplied by branches of the external carotid artery.

16 of 18 benign lesions were treated by endovascular therapy alone. Two recent patients received adjuvant stereotactic radiosurgery. Among these 18 patients 2 remained untreated, one because of spontaneous fistula thrombosis prior to therapy and one because of poor medical condition.

12 of 16 treated benign dural fistulas were partially occluded. in 6 of them spontaneous fistula thrombosis occurred during the following months. Total endovascular obliteration was achieved in the remaining 4 patients.

7 of 12 aggressive fistulas were embolized only, one of them having additional radiosurgery. Two of them were totally obliterated and five partially. Surgery was performed in the remaining 5 aggressive fistulas. Complete microsurgical excision was achieved in 2 and partial in further two, who presented initially with a life-threatening intracerebral clot. In one early case ligation of the external carotid artery was done, which is now obsolete. Over all 20 of 28 treated patients became asymptomatic or improved clinically.

3 of the remaining 8 patients were unchanged, two deteriorated despite therapy, and 3 worsened after therapy. All of the latter complications occurred early in our series due to thomboembolic events during the procedure. One surgical patient suffered from a new facial nerve palsy postoperatively. Follow up time in all treated patients was between 1 and 139 months with a mean of 48,3 months.

Keywords: Dural arteriovenous fistulas; intracranial haemorrhage; endovascular therapy; operative approach.

Introduction

Dural arteriovenous fistulas (d'AVFs) are rare lesions [14]. Their frequency is reported to be between 10–15% of all arteriovenous malformations of the brain [2]. In our series the incidence was 11%. They are found more frequently in the posterior fossa than supratentorially [1, 2, 6, 7]. d'AVFs are located most commonly at the transverse sinus, followed by the cavernous sinus (CS) and other locations [1, 2]. They occur typically in middle aged women [1, 2].

Little is known about their natural history and their clinical presentation may vary from mild tinnitus and a cavernous sinus syndrome to epilepsy and intracranial haemorrhage [8, 10].

In the clinical course of d'AVFs spontaneous cure due to sinus thrombosis, progressive neurological deterioration or severe disability after initial intracranial bleeding may occur [1, 2, 8, 10, 13, 14]. Depending on the clinical presentation and angiographic features of the draining venous system (i.e., leptomeningeal venous drainage, pure Galenic drainage, venous aneurysms) a more or less invasive therapy may be chosen [1–3, 11].

Manual percutaneous compression, transvascular embolization, surgery or radiosurgery may be used alone or in combination in the treatment of those unusual lesions [1–3, 7, 10, 11].

We report our experience made in the treatment of 28 out of 30 d'AVFs encountered during an eight year

period. The clinical behaviour, the pathogenesis and the different therapeutic strategies will be discussed.

Patients and Methods

Patients

Between 1983 and 1992 30 d'AVFs were seen at the Neurosurgical Department of the Medical School, University of Vienna. Table 1 summarizes clinical and morphological features. There were 11 male and 19 female patients. Age ranged between 18 to 85 years with a mean of 54 years.

Benign clinical presentation (Case 13–30) with tinnitus, exophthalmus and cranial nerve dysfunction was noted in 18 patients, whereas 12 had an aggressive clinical course (Cases 1–12) with haemorrhage, epilepsy, progressive neurological dysfunction, and cerebellar symptoms. One patient (Case 29) had symptoms of pseudotumour cerebri. In the medical history 3 cases of previous meningitis, 2 of head trauma and 1 of middle ear inflammation were found.

Four vessel angiography, including the examination of subclavian artery branches, if an additional supply was suspected, verified the diagnoses in all cases. Two patients remained untreated, one because of cardiac failure and the other had spontaneous thrombosis of her d'AVF.

Classification and Treatment

Depending on the symptoms and angiographic analysis d'AVFs were classified as benign or aggressive according to the literature [1, 2]. Out of 18 benign d'AVFs, 16 were treated by endovascular embolization, whereas 2 of them had adjuvant stereotactic irradiation with the Gammaknife. Of 12 aggressive d'AVFs 6 were treated only by embolization and 5 had subsequent surgery. Adjuvant Gammaknife therapy was performed after several endovascular procedures in one patient.

Endovascular Embolization

Standard percutaneous arterial access to the cervical carotid or femoral artery was performed using Seldinger's technique [7]. Before 1986 a 4 french angiography catheter was used for selective injection of dura, fibrin and coils into branches of the ECA supplying the fistula, as close as possible to the nidus [4].

Our superselective technique currently used the 3.2 french Tracker 18 microcatheter* supported by a microguidewire (0.014–0.016 platinum tipped guidewire). This microcatheter is introduced coaxially into a 7 french catheter, positioned in the ECA trunk. After superselective studies of the vascular territory to be embolized, the fistula compartment is occluded with polyvinylalcohol (PVA) particles**. If anastomotic vascular channels to physiological territories are excluded, glue*** is used. In early cases the feeding artery was occluded with fibremicrocoils* to avoid the pulsating flow to the mixture of particles and thrombosed blood.

Standard transcutaneous venous access through one femoral vein was performed in 2 patients. A 6 or 7 french angiography catheter was positioned in the jugular vein with its tip just below the skull base [7]. This manoeuvre provided access for the Tracker 18 microcatheter to the envolved segment of the transverse/sigmoid sinus to block the fistula from the venous side with microcoils.

Surgery

Microsurgical isolation and resection of the pathological sinus segment and interruption of all its arterial input was performed according to previous descriptions [2, 11]. In addition to surgical resection haematoma evacuation was done in 1 case of a tentorial and 1 of a fronto-ethmoidal d'AVF.

ECA-ligation, which is now obsolete, was performed after distal exposure of all ECA branches in the neck supplying a d'AVF at the transverse sinus in one early case in 1983.

Radiosurgery

3 patients were treated radiosurgically within the 40 to 60% isodose line with a marginal dose of 19.3 Gy (±1.5) and a maximal dose of 39.7 Gy (±9.1) to the nidus. 3 to 9 isocentres were used and all had preradiosurgical embolization.

Results

Benign d'AVFs

Total early obliteration was achieved in 4 of 16 benign d'AVFs treated by embolization (Table 1). In a further 6 delayed complete thrombosis of the d'AVFs, including 2 after adjuvant radiosurgery, was verified angiographically several months later.

9 of these 10 totally occluded fistulas were cured, one developed a major neurological deficit during the embolization due to a thromboembolic event. He died several months later of a pulmonary embolus.

Partial obliteration was seen in 6 patients. Three improved, one had a persistent 6 nerve palsy and 2 had poor outcomes with one acute unilateral visual deterioration progressing to blindness in a case of an orbital d'AVF. The other patient died 3 months after therapy of an intracerebral haemorrhage elsewhere. Autopsy was unfortunately not performed.

Aggressive d'AVFs

Endovascular therapy: Total obliteration was seen angiographically after embolization in 3 of 12 aggressive d'AVFs with one delayed fistula thrombosis. Two had an excellent outcome and one remained disabled because of severe persisting dizziness. Partial obliteration was seen in 3 patients, all of them improved clinically and refused further angiography or therapy.

Surgery: Complete microsurgical resection was performed in 2 patients with good results in both. Partial fistula resection was verified by follow-up angio-

* TARGET Therapeutics, California, USA

** IVALON®, Ingenor, Paris, France or CONTUR®, ITC, South San Francisco, California, USA

*** N-butyl-2cyanoacrylate, HISTOACRYL BLAU®, Braun-MELSUNGEN, Germany

Table 1. *Summary of 30 d'AVFs*

Case no.	Age (yrs). Sex	Presentation	Site of fistula	Venous pathology	Embolization	Surgery/ G. knife	Angio result	Follow-up (months)	Outcome
1	51 m	cn 5, 6, 7 palsy, tinnitus, SAH	R trans.sin., torcular, sag.sin.	R transv.sin occlusion*	transart. 4×, glue, coils	refused	partial	46	G-mild H/A
2	67 m	H/A, seizure	R transv.sin.	R transv.sin. occlusion* ven. aneur.	transart. 3×, Ivalon, coils fibrin	refused	partial	22	G-mild dizziness
3	53 m	cn 6 palsy, dizziness, ataxia	L transv.sin.	L transv.sin. occlusion*	transart. 3×, glue	none	total	99	G-mild dizziness
4	50 f	IVH SAH	L tent.incis.	ven.aneur.	transart. 1×, glue	none	partial**	35	E-no symptoms
5	73 f	ICH	B fronto-ethmoidal	ven.aneur.	transart. 1×, Ivalon, coils	haem.evac, part.excis., refused further therapy	partial	22	G-H/A
6	52 m	SAH, seizure	R transv.sin.	R transv.sin. occlusion* ven.aneur.	transart.2×, Ivalon, coils	refused	partial	24	G-H/A
7	57 f	tinnitus, H/A ataxia, nausea	L transv.sin.	L transv.sin. occlusion*	transart.3×, transven.1×, Ivalon, coils	excision	total	28	G-H/A
8	75 f	hemiparesis, aphasia, H/A, chemosis, exophthalmus	L cav. sin.	partial L cav. sin. occlusion*	transart. 3×, Ivalon, coils	none	total	19	F-severe dizziness, H/A
9	58 f	ICH, hemiparesis, aphasia	L tent.incis.	straight sin. occlusion* ven.aneur.	transart. 1×, Ivalon, coils	haem.evac. part.excis. refused further therapy	partial	51	P-hemiparesis aphasia
10	60 m	seizure	R transv.sin.	R transv.sin. occlusion*	transart.1×, fibrin, dura	excision	total	129	G-H/A
11	72 m	aphasia	L transv.sin	L transv.sin. occlusion*	transart.1×, fibrin, dura	ECA ligation	partial	lost	f-cn 7 palsy
12	18 m	seizure, ataxia, H/A	R transv.sin. R sup. petr.sin	ven.aneur.	transart.3×, transven.2×, fibrin, dura, coils	G.knife	partial	24	P-initially imp. status epilept. died after aspir.pneum.

Case No. 1–12: aggressive clinical course;
* retrograde leptomeningeal drainage; ** delayed fistula thrombosis; *SAH* subarachnoid haemorrhage; *IVH* intraventricular haemorrhage; *ICH* intracerebral haemorrhage; *H/A* headache; *aspir.pneum.* aspiration pneumonia; *transv.sin.* transverse sinus; *sag.sin.* sagittal sinus; *cav.sin.* cavernous sinus; *sup.pet.sin.* superior petrosal sinus; *tent.incis.* tentorial incisura; *transart.* transarterial; *transven.* transvenous; *haem.evac.* haematoma evacuation; *part excis.* partial excision; *ECA* external carotid artery; *E* excellent; *G* good; *F* fair; *P* poor; *R* right; *L* left; *B* bilateral; *G knife* Gamma knife; *ven. aneur.* venous aneurysm.

Table 1. *Continued*

Case no.	Age (yrs) Sex	Presentation	Site of fistula	Venous pathology	Embolization	Surgery/ G.knife	Angio result	Follow-up (months)	Outcome
13	33 f	tinnitus, ataxia vertigo, H/A	jugular foramen	none	transart. 2×, Ivalon, glue, coils	none	partial	71	G-improved
14	67 f	cn 3, 6 palsy, chemosis, exophthalmus	B cav. sin.	none	transart. 2×, Ivalon, coils	none	partial	72	F-cn 6 palsy, vision poor left eye
15	49 m	H/A, tinnitus	L cav.sin.	none	transart. 2×, Ivalon, coils	none	partial	81	G-improved
16	24 f	H/A, tinnitus	R. transv.sin.	R transv.sin. occlusion	transart. 1×, fibrin,dura,coils	none	partial**	59	E-no symptoms
17	50 f	tinnitus	R transv.sin.	R transv.sin. occlusion	transart. 2×, glue	G.knife	total	12	E-no symptoms
18	64 f	tinnitus	R. transv.sin.	none	transart. 2×, fibrin,dura,coils	none	total	99	E-no symptoms
19	60 m	exophthalmus	L sup.oph.v.	none	transart.2×, glue	none	total	1	P-hemiparesis aphasia, death
20	46 f	cn 6 palsy, chemosis, exophthalmus, blurred vision	R cav.sin.	R transv.sin. occlusion	transart.1×, Ivalon, coils	none	total	18	E-no symptoms
21	36 f	exophthalmus, H/A	R sup. oph. vein	none	transart. 1×, Ivalon, glue	none	partial	67	P-unilateral, blindness
22	55 f	cn 6 palsy, tinnitus	R cav.sin.	none	transart. 2×, Ivalon, glue, coils	none	partial**	33	E-no symptoms
23	55 m	exophthalmus, chemosis	R cav.sin.	none	transart. 1×, Ivalon, coils	none	partial**	44	E-no symptoms
24	47 m	cn 6 palsy, tinnitus	R. cav.sin.	none	transart. 2×, Ivalon,glue, coils	none	partial	48	G-improved, mild tinnitus
25	85 f	exophthalmus	R cav.sin	none	cardiac failure no therapy	none	none	1	P-died
26	77 f	cn 6 palsy, chemosis, exophthalmus	L cav.sin.	none	transart. 1×, Ivalon, coils	none	partial	3	P-initially improved, died of ICH 3 months after therapy
27	67 f	tinnitus	L transv.sin.	none	transart. 1×, Ivalon, coils	none	total	48	E-no symptoms
28	25 f	tinnitus	R transv. sin.	none	transart. 1×, tantalum, fibrin	none	partial**	139	E-no symptoms
29	55 f	tinnitus, H/A, blurred vision	L transv. sin.	L transv. sin. occlusion R trans. sin. stenosis	transart. 3×, glue, coils	G. knife/ LP-shunt	total	10	E-no symptoms
30	50 f	cn 6 palsy, exophthalmus, tinnitus	R cav. sin.	none	angiogram only	none	partial**	6	E-no symptoms

Cases 13–30: benign clinical course;
** delayed spontaneous fistula thrombosis; *ICH* intracerebral haemorrhage; *H/A* headache; *transv.sin.* transverse sinus; *cav.sin.* cavernous sinus; *sup.oph.v.* superior ophthalmic vein; *transart.* transarterial; *E* excellent; *G* good; *F* fair; *R* right; *L* left; *B* bilateral, *LP* lumboperitoneal; *G.knife* Gammaknife.

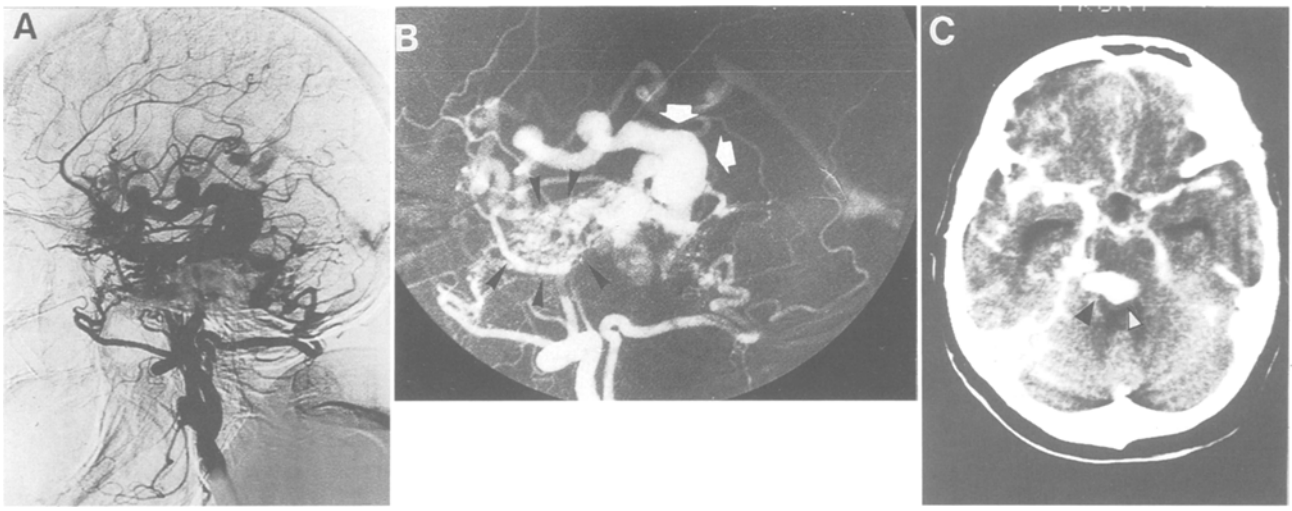

Fig. 1. Unusual aggressive d'AVF (Case 9) located at the tentorial ring (A) on common carotid artery angiography; lateral view. Selective ECA-injection (B) shows the nidus cleaqrly (arrows). Note the venous aneurysm (big arrows). Contrast-enhanced CT-scan (C) demonstrates the venous varix at the left tentorial incisura (arrows). This patient refused treatment initially and presented weeks later with intracerebral haemorrhage

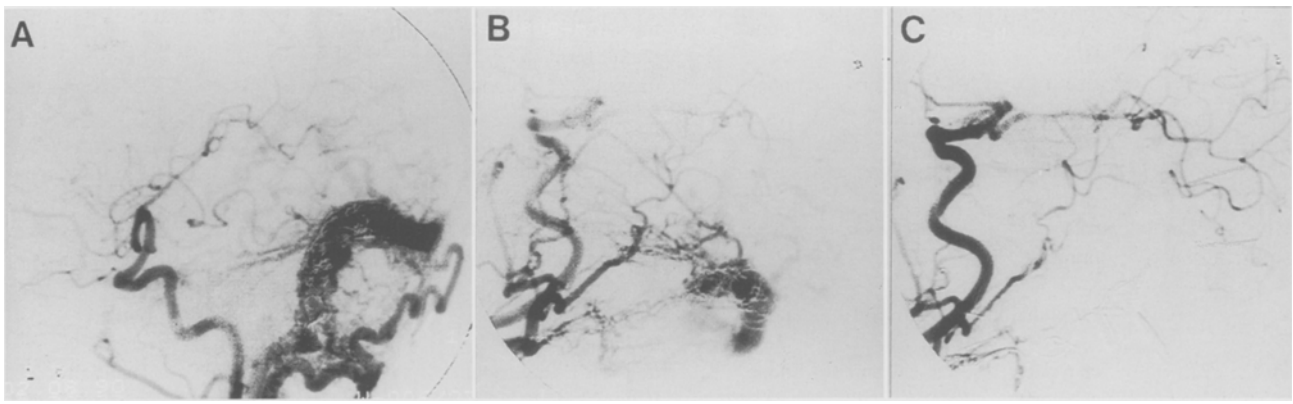

Fig. 2. Aggressive transverse sinus d'AVF (Case 7) before (A), after (B) embolization, and after complete surgical resection (C) on common carotid artery angiography; lateral view

graphy in 2 patients, who initially had undergone emergency haematoma evacuation. One recovered almost completely and the other remained disabled after the haemorrhage. Both patients refused further treatment. The early patient, whose ECA was ligated, suffered a new facial nerve palsy. He was subsequently lost to follow up.

Radiosurgery: The patient with a giant d'AVF suffered a generalized seizure, while awaiting the next transvenous embolization. He aspirated and died several weeks later of sepsis elsewhere.

Thus, in 20 out of 28 treated patients results were excellent or good. 3 of the remaining 8 were unchanged and 2 worse despite therapy. 2 patients had a clinical deterioration due to thrombo-embolic events resulting in hemiplegia and aphasia in 1, unilateral blindness in another. A new facial nerve palsy was seen postoperatively in another patient.

Follow-up

Follow up period ranged from 1–139 months, with a mean of 48.3 months.

Discussion

The natural history of d'AVFs is highly variable and depends on different morphological features [1, 2, 4, 7, 9, 10]. Spontaneous cure by fistula thrombosis

is commonly seen in benign d'AVFs presenting with tinnitus, a cavernous sinus syndrome, headache and cranial nerve dysfunction [1, 2, 7]. Spontaneous disappearance of d'AVFs located at the cavernous sinus, in particular is very common, but not all have a benign clinical course [7, 10].

Djindjan noted that the d'AVFs located at the tentorial incisura and at the base of the anterior cranial fossa were extremly prone to haemorrhage [4]. He drew attention to the specific venous drainage patterns, which were commonly found in d'AVFs that had haemorrhaged [4] (Fig. 1).

Recently, Awad *et al.* emphasized a refined classification, which combined the clinical course and specific angiographic features, like leptomeningeal venous drainage, deep Galenic venous drainage or venous aneurysms [1, 2]. This classification was based on a thorough review of all reported cases until 1989 and concluded that the above mentioned morphological features (venous abnormalities) found in certain d'AVFs significantly increased the natural risk for future haemorrhage [1, 2, 4].

We reviewed our cases according to Awad's proposed classification, and found similar results. All of our d'AVFs with an aggressive clinical presentation had these venous abnormalities. Additionally nearly all venous sinuses adjacent to aggressive d'AVF were occluded (see Table 1).

Factors which most likely play a role in the evolution of d'AVFs include trauma, intracranial surgery or infection [7, 10]. These factors may lead to sinus thrombosis and occlusion with subsequent recanalization via arterioles adjacent to a major sinus [1, 2]. Further recruitment of meningeal arteries progressively enlarges the fistula and increases the venous pressure inside the sinus, resulting in arterialized pial veins [1, 2]. Depending on a co-existing venous outflow obstruction a leptomeningeal drainage may develop, sometimes associated with venous aneurysms, which may rupture or compress important neural structures [1, 2, 8, 13, 14].

This pathophysiological concept and histopathological studies of resected specimens support the theory of an acquired pathology [1, 2, 5, 9]. In pathological studies evidence of a recanalized blood clot was found in parts of resected sinuses [5, 10]. Therefore, sinus thrombosis, which has been demonstrated angiographically preceeding d'AVF development, seems to be an important factor in a pathological process involving the sinus wall [5, 9].

Based on the above mentioned data concerning d'AVFs, different forms of treatment should be proposed to the patient [1–3, 6, 7, 13]. Sometimes a conservative attitude or manual percutaneous compression of feeding arteries may be sufficient in patients with a mild symptomatology [6, 7].

In our series, patients with benign d'AVFs underwent arterial embolizations with the aim of improving or curing their symptoms [7]. In patients with aggressive lesions complete elimination of the fistula by endovascular and/or microsurgery was the aim of treatment [11, 13] (Fig. 2).

In benign d'AVFs partial endovascular occlusion via the arterial route was often sufficient to reduce the symptoms or make them disappear [10, 13]. Partial embolization often initiated progressive thrombosis of the nidus of these low flow arteriovenous shunts [6, 7, 10]. If symptoms reoccurred, further embolizations were done to further reduce flow through the fistula [10]. Surgery should be performed in cases where endovascular therapy failed, but with careful assessment of the "risk to benefit ratio" in each patient [2, 11].

The transvenous route for endovascular fistula occlusion was performed only exceptionally in our series. Several authors regard it as the therapy of choice in cavernous d'AVFs [7, 10]. We achieved only 1 satisfying angiographic result, but unfortunately the patient died later of sepsis (Case 12).

Complete permanent angiographically verified cure achieved either by embolization or/and surgery is the only acceptable result in patients suffering from aggressive d'AVFs [1–3, 7, 11].

Microsurgical resection of the involved sinus segment is reported to have a high mortality and morbidity rate [11]. Therefore, we combine it with endovascular therapy, as in cerebral arteriovenous malformations, to facilitate surgical resection. Careful angiographic analysis is necessary in the pre-operative planning to determine orthograde or retrograde flow in the venous sinus and its associated cortical veins. Only those sinuses should be sacrificed, which have no draining function for the brain, otherwise the patient is at risk from postoperative venous infarction [1, 2, 11]. If the involved sinus segment has to be preserved, sinus skeletonization should be performed disconnecting the sinus from all its arterial input [2]. If a d'AVF drains exclusively through one or a few leptomeningeal veins, an alternative way of treatment is simple surgical interruption of the vein close to the

nidus, which results in complete elimination of the fistula [12].

The preliminary reports of radiosurgical treatment of d'AVFs are encouraging, but late results are not yet available. In 2 of 3 patients, who had adjuvant Gammaknife therapy after embolization, progressive obliteration of the nidus was noted over a one year period.

However, during the waiting period for total obliteration, the patient's risk for further haemorrhage is not reduced [1, 2]. Therefore, Gammaknife therapy can be used primarily in cases with a benign clinical presentation, whereas in aggressive d'AVFs it should be used as an adjuvant therapy [2].

In conclusion, aggressive d'AVFs require total, permanent cure either by embolization alone, or in combination with surgery. Careful angiographic analysis, pre-operative embolization, particulary of the deep arterial supply, and subsequent surgery, can achieve very satisfying results also in the treatment of giant and complex d'AVFs [2, 3].

In the benign group with its mild clinical presentation, a reduction or disappearence of symptoms after incomplete endovascular embolization is possible in the majority of cases [7, 10, 13].

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