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Long-term changes in intracranial dural arteriovenous fistulae leading to worsening in the type of venous drainage

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Abstract We review seven patients with intracranial dural arteriovenous fistulae (ICDAVF), each altering the initial type of venous drainage to one with a higher grading during long-term follow-up. Five were discovered due to symptoms of intracranial hypertension, two due to changes in tinnitus and one case following subarachnoid haemorrhage. In five cases, cortical venous drainage developed during the follow-up period. Three different mechanisms were observed: stenosis or thrombosis in the draining veins in 4 cases: increased arterial flow in 2; and the appearance o a new fistula site or extension of the initial shunt in 2. Type I and type II a fistulae which are not completely cured, require both close clinical observation and Doppler examinations in the follow-up period. Any charge in the clinical pictures indicates a repeat angiogram. Stenosis of the venous drainage, forecasting later worsening in the venous outlet, requires more thorough angiographic follow-up.

Key words Arteriovenous fistula dural · Embolisation · Haemorrhage, Subarachnoid

Introduction

The neurological symptoms and signs and risks associated with intracranial (IC) dural arteriovenous fistulae (DAVF) depend on the venous drainage pattern. The classification based on differences in venous drainage [1] (Table 1) is useful when considering a therapeutic approach (Table 2). Fistulae draining into a sinus with normal antegrade flow (type I) are treated, in our institution, only when symptoms are severe. Treatment is by manual arterial compression by the patient, or embolisation, using particles or glue, of the external carotid meningeal feeding vessels. Treatment is similar for fistulae draining backwards into another main sinus (type IIa), to prevent or treat symptoms or signs of IC hypertension. DAVF with cortical venous drainage (types IIb–V) call for complete, durable cure.

Thus, the treatment of types I and IIa DAVF aims mainly at reducing flow, since attempted complete cure may be associated with an increased risk of complications. However, the possible evolution of IC DAVF from one type to another may modify the therapeutic decision. We present seven patients with IC DAVF, in whom, a change in venous drainage type was observed during long-term follow-up.

Materials and methods

We studied three women and four men, mean age 58 years (range 42–77 years). The initial clinical findings, angiographic venous pattern and treatment are summarised in Table 3.

In six cases the first symptom was tinnitus; in one, it was the sudden disappearance of tinnitus which had been present for 6 months. All the fistulae were on the transverse or sigmoid sinuses. There were five type I (cases 1–5) and two type IIa fistulae (cases 6 and 7). In five cases the venous drainage was abnormal; in cases 1–3 the jugular bulb was stenosed and in cases 6 and 7 the transverse or sigmoid sinus was occluded. In cases 4 and 5 the sigmoid sinus and jugular veins were normal.

Cases 1, 2, 4, 6 and 7 were initially treated by embolisation, using particles, of the meningeal branches of the external carotid artery. This produced a reduction of the bruit in cases 1 and 4 and the complete disappearance in cases 2 and 7. In case 6, tinnitus reap-

Table 1 Classification of dural arteriovenous fistulae according to venous drainage [1]

Type	Pattern of venous drainage
Type I	Into a sinus, with normal antegrade flow
Type II	Into a sinus with insufficient antegrade venous drainage and reflux: IIa: into sinus(es) only IIb: into cortical vein(s) only IIa + b: into sinus(es) and cortical vein(s)
Type III	Directly into a cortical vein, without venous ectasia
Type IV	Into a cortical vein, with venous ectasia
Type V	Into spinal perimedullary veins

Table 2 Types of venous drainage: risks and therapy

Type	Risks	Treatment
I	Functional symptoms No neurological risk	Goal: reduction of flow no treatment vascular compression arterial embolisation (particles or glue)
IIa	Intracranial hypertension (visual impairment 20 %)	Goal: reduction of flow arterial embolisation (particles or glue) sinus occlusion
IIb, IIa + b	Bleeding risk 10 % Focal neurological signs	Goal: complete cure sinus occlusion
III–V	Bleeding risk 40 % type III, 65 % type IV Focal neurological signs and myelopathy	Goal: complete cure endovascular treatment, arterial or venous (catheterisation of cortical veins) neurosurgery radiotherapy

peared a few days after each of three embolisation procedures; subsequent ligation of both occipital and middle meningeal arteries did not alter the tinnitus.

Case reports

We have selected three patients to illustrate different mechanisms of worsening of the type of venous drainage.

Case 4

The patient was admitted in 1989 with right-sided tinnitus for a few weeks. Carotid angiography disclosed a type I sigmoid sinus fistula (Fig. 1 a). Embolisation of the meningeal external carotid artery branches, using particles, reduced the bruit. Four years later, the patient was admitted because of chemosis and proptosis on the right. Examination also disclosed cognitive impairment. Carotid angiography then showed a very different situation, with a spread of the shunts along the sinuses: one to the right sigmoid sinus draining into the jugular vein (type I); fistulae to the right transverse sinus (TS), torcular (Fig. 1 b) and left TS (Fig. 1 c) draining backwards into the superior sagittal sinus (SSS), straight sinus (SS) and cortical veins respectively (types IIa + b), and a fistula to the right superior petrosal sinus (Fig. 1 d, e) draining into cortical veins (type IIb). Because of the arterialisation of the SSS, SS and left TS, the cerebral venous drainage was very slow, with bilateral drainage into the cavernous sinus, pterygoid plexus, and via transmeningeal and transdiploic veins. Arterial embolisation was performed with glue, and endovascular occlusion of the right TS is planned.

Case 6

The patient presented with right-sided tinnitus in 1986. Angiographic examination disclosed a fistula to the right TS (Fig. 2 a, b) with occlusion of this sinus distal to the fistula and drainage into the left TS (type IIa). Tight stenosis of the left TS and jugular vein was seen. Arterial embolisation was performed with particles, stopping the tinnitus. One year later, the patient suffered transient visual obscurations and decreased visual acuity and was found to have bilateral papilloedema. Three embolisation procedures, using particles, were performed, following each of which the tinnitus reappeared after a few days. In 1994, due to continued deterioration of visual acuity, angiography was repeated, showing a fistula to the right TS (Fig. 2 c) with reflux into the SSS, SS, left TS and cortical veins (type IIa + b). Doppler sonography showed an increase in flow rate, from 560 cm^3/min before initial treatment in 1986 to 750 cm^3/min in 1994. Despite the age of the patient, the right TS was occluded through the ipsilateral jugular vein and the thrombosed sigmoid sinus (Fig. 2 d). This resulted in the complete obliteration of the fistula with normalization of the cerebral venous drainage and disappearance of all symptoms and signs.

Case 7

A fistula on the left TS, producing tinnitus, was discovered in 1989. The left sigmoid sinus was occluded and the fistula drained backwards into the right TS (type IIa) (Fig. 3 a). Arterial embolisation with particles resulted in reduction of the tinnitus. Five years later, the patient was admitted as an emergency for an intraventricular and subarachnoid haemorrhage. Angiography showed a fistula on the left TS (Fig. 3 b) draining backwards to the SS and cortical veins (type IIb) with an occlusion at the level of the junction between the SSS and the proximal portion of the left TS. The left TS was occluded with coils via the thrombosed ipsilateral jugular vein.

Results

The symptoms and signs leading to follow-up angiography were: intracranial hypertension with visual obscurations and papilledoema (case 1); reappearance of (case 2) or increase in (case 3) tinnitus; chemosis and proptosis (case 4); cognitive decline (cases 4 and 5); deterioration of visual acuity (case 6), intraventricular and subarachnoid haemorrhage (case 7).

The angiographic diagnosis of worsened venous drainage was made 1 month to 20 years (mean 7 years) after initial angiography (Table 4).

In two patients, follow-up angiography showed development of one (case 1) or two fistulae (case 4) in ad-

Table 3 Initial venous drainage in seven patients (*SS* straight sinus, *TS* transverse sinus)

Case	First symptom(s)	Sinus/type	Abnormalities of the venous drainage	Treatment	Results
1	Spontaneous disappearance of right tinnitus	Right TS; I	Stenosis right jugular bulb	Particles	Reduction in tinnitus (7 years)
2	Left tinnitus; retroauricular pain	Left TS; I	Stenosis left jugular bulb	Particles	Disappearance of tinnitus (1 month)
3	Right tinnitus (one week after cranial traumatism)	Right TS; I	Stenosis right jugular bulb	Occipital, middle meningeal artery ligation	Tinnitus unchanged (20 years)
4	Right tinnitus	Right SS; I	None	Particles	Reduction in tinnitus
5	Left tinnitus	Left TS; I	None	None	
6	Right tinnitus	Right TS; IIa	Occlusion right TS; stenosis left TS + jugular vein	3 embolisations with particles	Reappearrannce of tinnitus after each embolization
7	Left tinnitus	Left TS; IIa	Occlusion left sigmoid sinus	Particles	Disappearance of tinnitus

Table 4 Changes in type of venous drainage (*SPS* superior petrosal sinus, *SS* straight sinus, *TS* transverse sinus)

Case	Interval	New symptom(s) or signs	Sinus/type	Changes in venous drainage	Treatment	Evolution
1	7 years	Visual obscurations; bilateral papilloedema	Right TS: IIa Torcular, left TS: IIa	Occlusion right sigmoid sinus	Particles; TS occlusion planned	Visual obscurations decreased
2	1 month	Left tinnitus; vertigo	Left TS: IIa	Occlusion left sigmoid sinus	TS occlusion (coils)	Cured, asymptomatic
3	20 years	Worsening of tinnitus	Right TS: IIb	Proximal occlusion + distal right TS	TS recanalisation and occlusion (coils)	Cured, asymptomatic
4	4 years	Right chemosis and proptosis: cognitive decline	Right SS: I Right TS, torcular, left TS: IIa + b; SPS: IIb	None	Arterial embolisation (glue); right TS occlusion planned	Partial improvement
5	3 years	Seizures; cognitive decline	Left TS: IIa + b	None	TS occlusion (coils)	Cured, asymptomatic
6	8 years	Decreased visual acuity; bilateral papilloedema	Right TS: IIa + b	None	TS recanalisation and occlusion (coils)	Cured, asymptomatic
7	5 years	Intraventricular haemorrhage	Left TS: IIa + b	Occlusion proximal left sigmoid sinus; reflux into SS	Percutaneous TS puncture and occlusion (coils)	Cured, asymptomatic

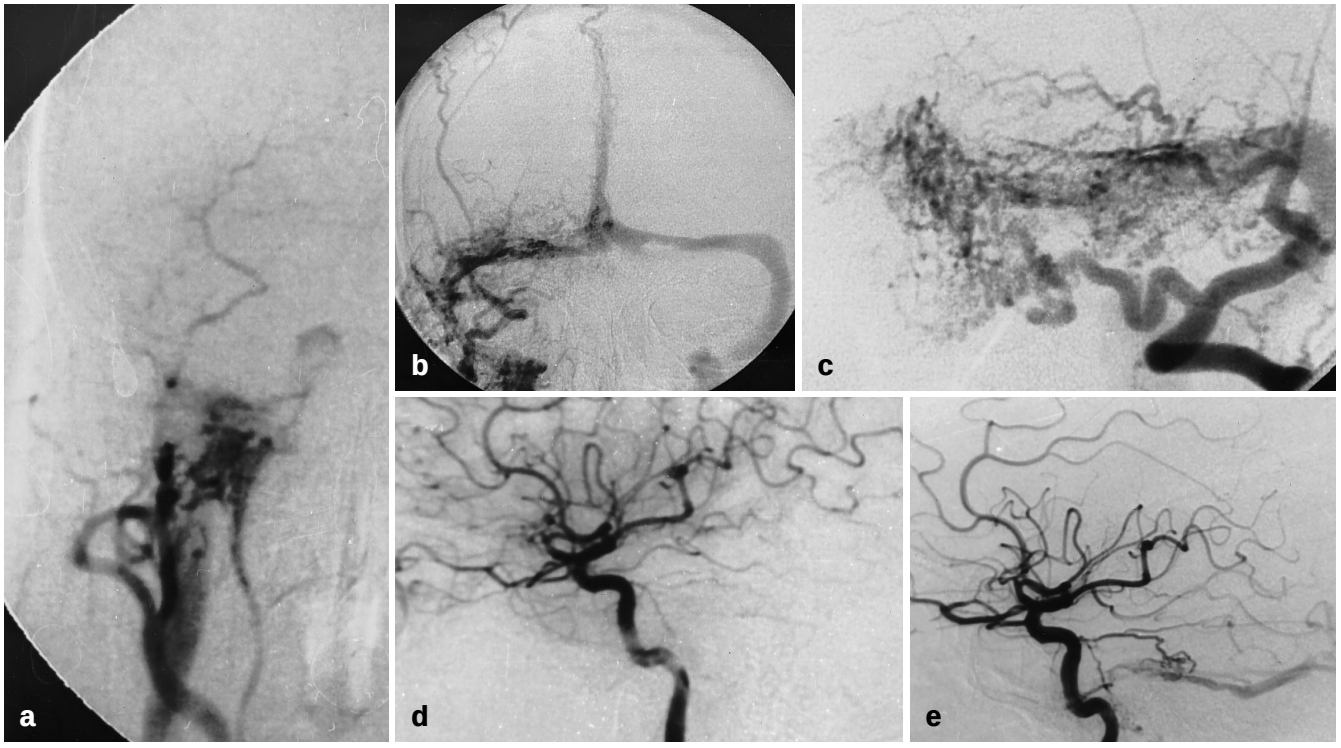

Fig. 1 a–e Case 4. **a** Right external carotid angiogram in 1989; anteroposterior view prior to first embolisation. **b** 1993: note extension of the shunt to the proximal right transverse sinus (TS) and torcular with reflux into the left TS and superior sagittal sinus (SSS). **c** Left external carotid angiogram (1993): development of new fistula on the left TS. **d, e** Right internal carotid angiogram in 1989 and 1993: development of a new fistula on the right superior petrosal sinus, with cortical venous drainage

dition to the previously existing one. In case 1, the first fistula had changed from type I to type IIa.

In four of the five patients where the initial angiographic studies had shown abnormalities of the venous pathways (cases 1–3, 7), additional abnormalities were seen on the later angiograms: occlusion of the sigmoid sinus distal to the fistula (cases 1 and 2), indicating evolution from type I to type IIa; occlusion of the TS both proximal and distal to the fistula (case 3); i. e. evolution from type I to type IIb; occlusion of the sinus proximal to the fistula (case 7), converting type IIa to type - IIa + b. In the two patients (cases 4 and 5) with previously normal drainage from the fistula, the later angiogram showed no change.

There were two patients (cases 5 and 6) in whom venous drainage changed angiographically (types I and IIa to type IIb). Although no change in venous outflow, nor any new fistula sites could be found, Doppler sonography revealed an interval increase in flow rate.

In five cases, treatment consisted of occluding the sinus with coils. This was performed through the ipsilateral jugular vein, when the sinus was normal (case 5), or by endovascular recanalisation, when the TS or sigmoid sinus were thrombosed (cases 3 and 6). In case 2, because of the thrombosis of the left TS, occlusion was performed through the contralateral jugular vein and TS. In case 7, the TS was occluded on both sides of the fistula, and the coils had to be delivered via direct puncture of the TS. The two patients treated by arterial embolisation with glue (case 4) or particles (case 1) are still not cured and occlusion of the TS is planned.

When it was possible, occluding the sinus induced complete closure of the fistula with complete clinical improvement. Patient 2 experienced diplopia and vertigo a few hours following the occlusion but these symptoms disappeared in 2 days following systemic heparinisation and corticosteroid therapy.

Discussion

DAVF were initially considered more benign than arteriovenous malformations of the brain. However, it was noted that all DAVF carry a potential risk of IC haemorrhage, and bleeding was attributed to pial compartment. The role of cortical venous drainage and particularly of dilated or ectatic veins was first pointed out by Houser et al. [2]. A general classification of DAVF, with regard to their pattern of venous drainage and corresponding symptomatology, was elaborated in 1978 by Djindjian et al. [3]. They assumed that DAVF

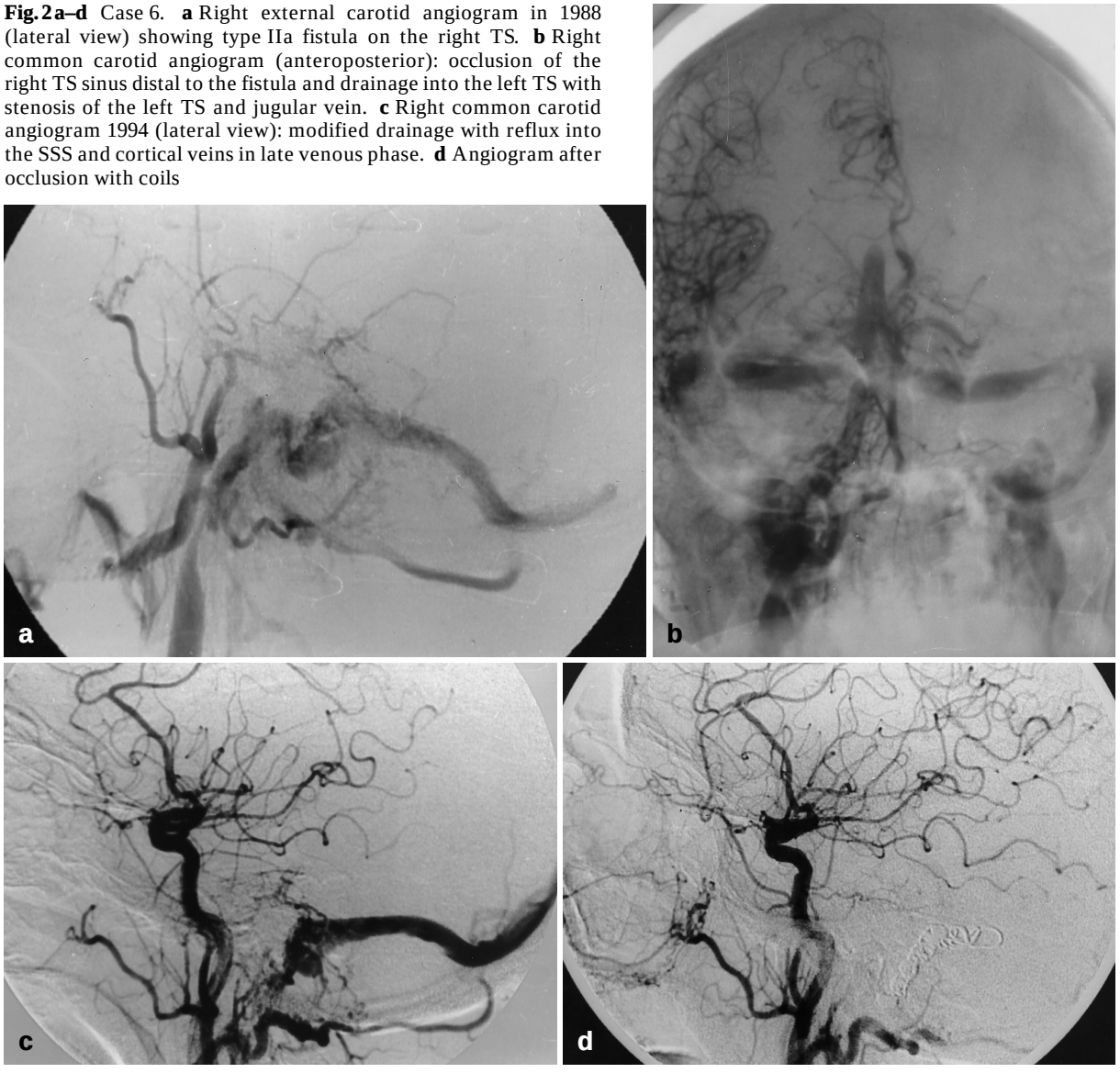

Fig. 2a–d Case 6. **a** Right external carotid angiogram in 1988 (lateral view) showing type IIa fistula on the right TS. **b** Right common carotid angiogram (anteroposterior): occlusion of the right TS sinus distal to the fistula and drainage into the left TS with stenosis of the left TS and jugular vein. **c** Right common carotid angiogram 1994 (lateral view): modified drainage with reflux into the SSS and cortical veins in late venous phase. **d** Angiogram after occlusion with coils

draining freely into a sinus produced only mild symptoms, while cortical venous drainage was associated with more severe neurological disturbance and haemorrhage. Reviews of previously published cases assessed the role of venous patterns in the occurrence of bleeding or focal neurological deficits [4–6]. We recently reviewed 205 patients with DAVF [1], using clinical and angiographic correlations to revise the classification of Djindjian et al. (Table 1). Clinical consequences and risks correlated with different types of venous drainage, thus indicating different therapeutic approaches (Table 2).

However, if type I DAVF are clearly benign lesions and therefore must not be treated aggressively, it must be ascertained that they will not transform into higher grades. Some workers have related clinical progression to worsening of the venous drainage [2, 7–9], but to our knowledge no angiographic proof of such evolution has been published.

In our seven cases, three different mechanisms explaining this worsening venous drainage pattern can be observed.

Stenosis or thrombosis of the venous outflow

In cases 1, 2, 3 and 7 thrombosis at the site of venous drainage led to progression. Two type I fistulae became type IIa by occlusion of the sinus distal to the shunt, and one became type IIb following occlusion on both sides (Fig. 4); one type IIa fistula with an initial distal occlusion became type IIa + b by virtue of a proximal occlu-

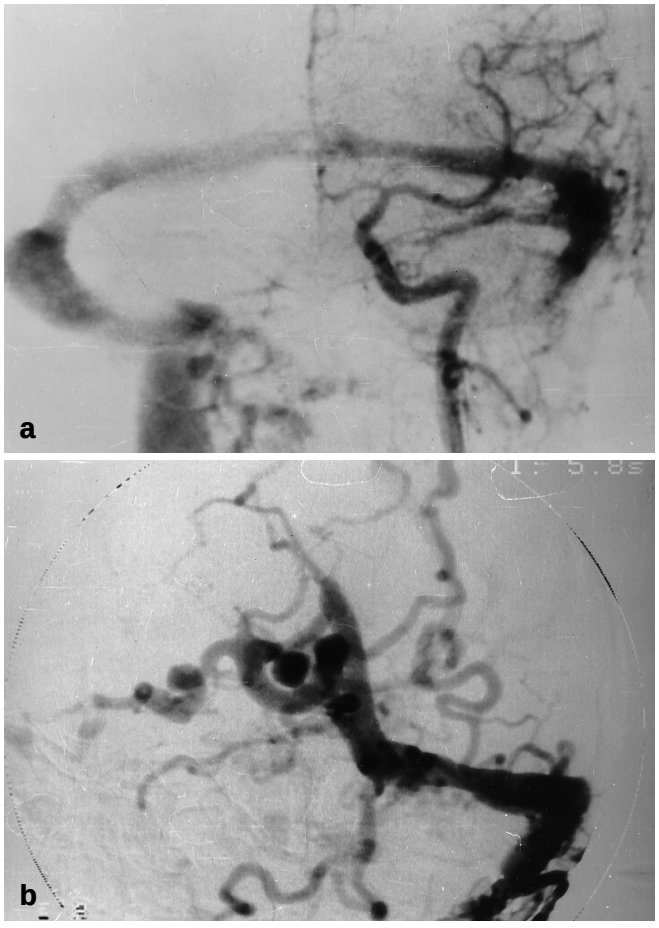

Fig. 3 a,b Case 7. **a** Left common carotid angiogram (anteroposterior): type IIa fistula on the left TS with occlusion of the left sigmoid sinus and retrograde drainage into the right TS. **b** Occipital arteriogram (oblique). Venous phase, showing changes in venous drainage, with occlusion of the proximal portion of the left TS and reflux into the straight sinus and cortical veins

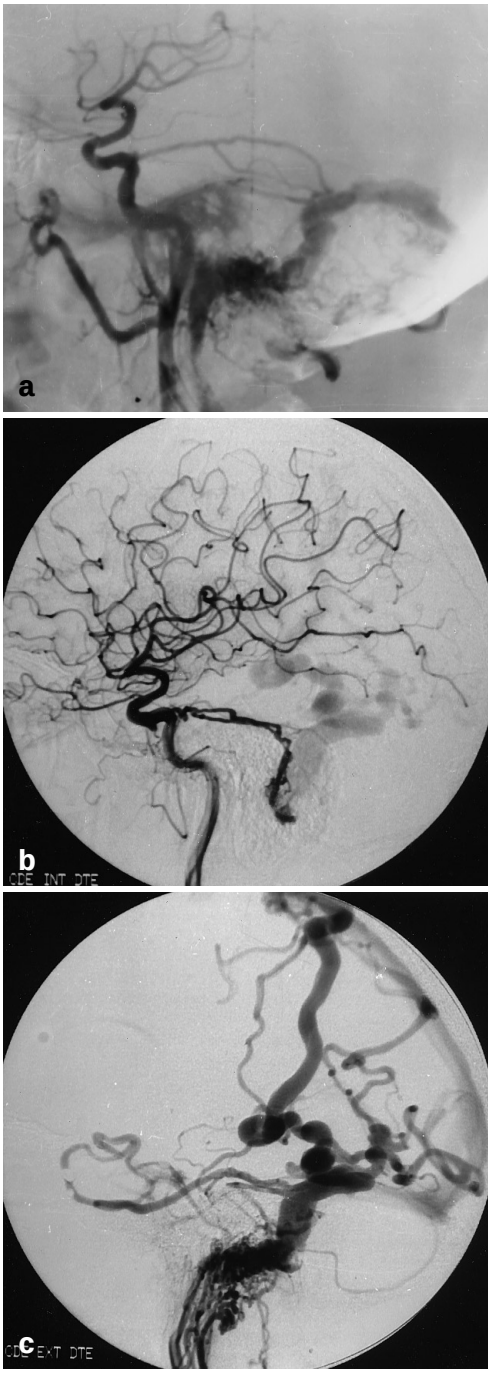

Fig. 4 a–c Case 3. **a** Right common carotid angiogram in 1972 showing type I TS fistula with stenosis of right jugular bulb. **b** Internal carotid angiogram in 1992 showing occlusion of the sinus proximal and distal to fistula. **c** Late venous phase of external carotid angiogram (in 1992) shows extensive cortical venous drainage

sion. The three patients (cases 1–3) with distal occlusion had an obvious stenosis of the sinus or jugular vein on the initial angiogram which progressed to thrombosis. In the two patients (cases 3 and 7) with proximal occlusion, no venous abnormality was visible initially. In case 3, with a type I fistula, it was impossible to analyse the sinus proximal to the shunt because of competition between the drainage of the fistula and that of the brain. In case 7, with a type IIa fistula, the sinus initially appeared normal.

The role of thrombophlebitis in the genesis of DAVF has frequently been discussed [2, 8–19]. Chaudhary et al. [8] suggested that "thrombosis in the dural sinus or vein may be the primary event in the formation of DAVF". Most authors believe that causative factors, such as thrombophlebitis or other involvement of the sinus including surgery, trauma or tumour, may induce opening of the normal arteriovenous shunts described by Kerber and Newton [20]. Thus, underlying thrombotic disease at the origin of the fistula may explain progressive

thrombosis of the sinus(es) and the delayed development of additional shunts.

On the other hand, the role of the fistula itself in producing sinus stenosis or occlusion remains unclear. Piton et al. [7] suggested that the different types of venous drainage probably correspond to stages in the course of the disease. They assume that distention of mural vessels progressively reduces the lumen of the vein and may result in complete obstruction, with subsequent drainage through the contralateral sinus. Afterwards, only one part of the fistula would remain, in the wall of the sinus, near the ostium of a cortical vein, which thus would constitute the sole outlet of the system. Nishima et al. [19] postulated that stenosis of the lumen of the sinus is due mainly to marked thickening of its intima and the development of an abnormal vascular network within its wall. Awad et al. [4] suggested several stages in the natural history of DAVF, due to arterial recruitment – "sump effect" – with secondary venous hypertension. In a recent study, looking at grades of restrictive venous disease in DAVF, Lalwani et al. [21] suggest that the DAVF is a dynamic condition, which can progress at variable rates from minimum restrictive venous disease to more severe outflow impairment.

Apart from considerations of the role of the fistula in creating a thickened sinus wall, we may assume that the high arterial flow can produce lesions in the wall of the sinus at a distance from the shunt itself. Occlusion proximal to the fistula may be explained by impaired venous drainage and stagnation, where the normal drainage of the brain and that of the fistula are in competition. Three case reports have indicated the role of fistulae in the genesis of sinus thrombosis [22–24]; in these cases, however, occlusion of the sinus resulted in disappearance of the fistulas.

Thus, even if the supposition that the haemodynamics of DAVF can be modified by venous stenosis or occlusion is generally accepted, these changes have never been shown angiographically. Consequently, it seems difficult to consider progression from one stage to another as the natural course of a DAVF.

In three of our four cases, the patients had initially been treated by arterial embolisation with particles and, in one case, by ligation of external carotid branches. Thus, the role of reduced arterial flow in the development of stenosis or occlusion of the sinus may be questioned. High flow could maintain sufficient pressure in the sinus, avoiding the formation of thrombosis, but embolisation without complete obliteration of the fistula could favour thrombosis and worsened venous drainage.

Increased arterial flow

In cases 5 and 6, the drainage worsened from type I and type IIa respectively, to type IIa + b. In case 5, no venous abnormality was observed on the initial angiogram, or on follow-up. In case 6, initially a type IIa fistula with distal sinus occlusion and stenosis of the controlateral TS and jugular vein, the later angiogram showed similar venous abnormalities, and thus did not explain the worsened venous drainage. It seems plausible that in these cases, the change of venous drainage was due to an increase in arterial flow. This supposition is also supported by the sonographic findings of increased arterial flow rate.

Appearance of new fistulae or extension of the initial shunt

In cases 1 and 4 the later angiogram showed development of new fistulae. In case 1, the initial fistula changed from type I to type IIa by virtue of distal occlusion of the TS. The shunt, initially on the right TS, was found on the later angiogram to extend to the torcular and proximal portion of the left TS. In case 4, the initial type I sigmoid sinus fistula was unchanged, but two other fistulae had developed, one at the torcular and proximal portion of the TS bilaterally and one on the superior petrosal sinus.

It is difficult to determine whether the same pathological mechanism as at the origin of the initial fistula was responsible for the two other shunts, or whether the haemodynamic changes created by the first fistula gave rise to the others. Barnwell et al. [25] reported 7 cases of multiple DAVF. They postulated that a hypercoagulability state may lead to thromboses and to fistulae at different sites. They also stated that impaired venous drainage into the main sinuses, causing stagnation, could give rise to a second fistula.

To our knowledge, there has been no previous reports in the literature of this occurrence, which partly reflects its rarity. Our previous experience [1] did not indicate a natural progression from one type to another. In the present study it is difficult to draw firm conclusions about the frequency of progression into higher grades, since the time between the initial angiogram and demonstration of progression varied from 1 month to 20 years, indicating the necessity of very long-term follow-up in these cases.

At the time of diagnosis, the venous outflow must be analysed carefully and any stenosis of the draining veins must be considered a risk factor for worsening of the fistula. After initial diagnosis and treatment, patients with I or type IIa fistula who are incompletely cured should be told that any change in their symptoms, for better or for worse, is an indication for a new angiogram. We routinely perform annual Doppler sonography to screen for an increase in arterial flow rate.

In our opinion, fistulae with cortical venous drainage (types IIb–V) must be completely obliterated because of the high risk of haemorrhage or focal neurological

deficits. Type IIa fistulae (often very difficult to cure) are frequently treated by arterial embolization, with particles, to reduce the flow and induce clinical improvement. Type I fistulae can be considered benign and treated only if symptoms are severe. The goal of arterial treatment in these cases is to reduce flow, to minimise or, if possible, remove symptoms such as tinnitus. Complete cure, necessitating more hazardous treatment, is not required.

The possibility of progression with more severe venous drainage could change the approach to therapy and lead one to perform more aggressive treatment of types I and IIa fistulae. In cases with abnormalities of venous drainage the venous approach, with occlusion of the sinus, should be performed. However, occlusion of the sinus in types I and IIa fistulae raises two main problems. Firstly, occlusion of a sinus implies the sacrifice of a major route of cerebral venous drainage. Even though arterialised and not currently available for drainage, the sinus could be usable later, after haemodynamic modification or through the development of new fistulae. In case 4, the left TS, even though arterialised, drained the contralateral TS fistula, because of the increased pressure in the SSS. Secondly, treating types I and IIa DAVF by sinus occlusion is more dangerous than in type IIb fistulae. It needs careful study of the cerebral and cerebellar late venous phase, to be sure that normal veins are not draining into the arterialised sinus. Sinus occlusion often leads to a complete cure of the fistula but also runs the risk of being more dangerous than the natural history of the disease.

However, we need further knowledge about the frequency of the progression described here to be able to assess whether the type I DAVF is truly benign disease or a more serious condition, which by its natural course or following embolization can evolve to a higher grade.

References

1. Cognard C, Gobin YP, Pierot L, et al (1994) Neurological symptoms of intracranial dural arteriovenous fistulas: clinical and angiographic correlation in 205 cases. A revisited classification of the venous drainage. Radiology 194: 671–680
2. Houser O, Baker H, Rhoton A, Okazaki H (1972) Intracranial dural arteriovenous malformations. Radiology 105: 55–64
3. Djindjian R, Merland JJ, Thèron J (1977) Superselective arteriography of the external carotid artery. Springer, New York Berlin Heidelberg, pp 606–628
4. Awad I, Little J, Akrawi W, Ahl J (1990) Intracranial dural arteriovenous malformations: factors predisposing to an aggressive neurological course. J Neurosurg 72: 839–850
5. Malik G, Pearce J, Ausman J, Mehta B (1984) Dural arteriovenous malformations and intracranial hemorrhage. Neurosurgery 15: 332–338
6. Lasjaunias P, Chiu M, TerBrugge K, Tolia A, Hurth M, Berenstein M (1986) Neurological manifestations of intracranial dural arteriovenous malformations. J Neurosurg 64: 724–730
7. Piton J, Guilleux H, Guibert-Tranier F, Caillé J (1984) Fistules du sinus latéral. J Neuroradiol 11: 143–159
8. Chaudhary M, Sachdev V, Cho S, Weitzner I, Puljic S, Huang Y (1982) Dural arteriovenous malformations of the major venous sinuses: an acquired lesion. AJNR 3: 13–19
9. Watanabe A, Takahara Y, Ibuchi Y, Miukami K (1984) Two cases of dural arteriovenous malformation occurring after intracranial surgery. Neuroradiology 26: 375–380
10. Roland J, Bernard C, Bracard S, et al (1987) Microvascularization of the intracranial dura mater. Surg Radiol Anat 9: 43–49
11. Urdanibia J, Silvela J, Soto M (1974) Occipital dural arteriovenous malformations. Neuroradiology 7: 57–64
12. Lasjaunias P, Halimi P, López-Ibor L, Sichez J, Hurth M, Tribolet ND (1983) Traitement endovasculaire des malformations vasculaires durales pures spontanées. Neurochirurgie 30: 207–223
13. Halbach V, Higashida R, Hieshima G, Mehringer C, Hardin C (1989) Transvenous embolization of dural fistulas involving the transverse and sigmoid sinuses. AJNR 10: 385–392
14. Barnwell S, Halbach V, Higashida CD, Hieshima G (1990) Dural arteriovenous fistulas involving the inferior petrosal sinus. AJNR 11: 511–516
15. Gobin YP, Rogopoulos A, Aymard A, Bailly AL, Casasco A, Merland JJ (1993) Percutaneous transvenous embolization through the thrombosed sinus in two transverse sinus dural fistulas. AJNR 14: 1102–1105
16. Brainin M, Samec P (1983) Venous hemodynamics of arteriovenous meningeal fistulas in the posterior cranial fossa. Neuroradiology 25: 161–169
17. Convers P, Brunon J, Sindou M (1986) Fistules artérioveineuses durales de la fosse postérieure et thrombose du sinus latéral: discussion de leur relations et de leur traitement à propos de deux cas. Neurochirurgie 32: 495–500
18. Handa J, Yoneda S, Handa H (1975) Venous sinus occlusion with dural arteriovenous malformation of the posterior fossa. Surg Neurol 4: 433–437
19. Nishijima M, Takaku A, Endo S, et al (1992) Etiological evaluation of dural arteriovenous malformations of the lateral and sigmoid sinuses based on histopathological examinations. J Neurosurg 76: 600–606
20. Kerber C, Newton T (1973) The macro and microvasculature of the dura mater. Neuroradiology 6: 175–179
21. Lalwani A, Dows C, Halbach VV (1993) Grading venous restrictive disease in patients with dural arterio-venous fistulas of the transverse/sigmoid sinus. J Neurosurg 79: 11–15
22. Magdison M, Weinberg P (1976) Spontaneous closure of a dural arteriovenous malformation. Surg Neurol 6: 107–110
23. Rohr J, Gautier G (1985) Regression spontanée d'une fistule artério-veineuse dure-mèrienne de la fosse postérieure. Rev Neurol 141: 240–244
24. Bitho S, Onishi T, Takimoto N, Sakaki S, Ghoma T, Motozaki T (1978) Dural arteriovenous fistula after removal of a meningioma: a case report. Neurol Surg 6: 397–400
25. Barnwell S, Halbach V, Dowd C, Higashida R, Hieshima G, Wilson C (1991) A variant of arterio-venous fistulas within the wall of dural sinuses. J Neurosurg 74: 199–204