Neuroradiology (1993) 35: 388–393

Neuro radiology



# Dural arteriovenous malformations at the base of the anterior cranial fossa: report of nine cases

**J. Reul[1], A. Thron[1], G. Laborde[2], H. Brückmann[1]**

[1] Department of Neuroradiology, RWTH, Aachen, Germany
[2] Department of Neurosurgery, RWTH, Aachen, Germany

Received: 30 June 1992/Received in revised form: 11 September 1992

**Abstract.** Nine men with dural arteriovenous malformations (DAVM) at the base of the anterior cranial fossa are described. Four patients had intracerebral haemorrhage and four had seizures, associated with haemorrhage in two. In three cases the fistula was an incidental finding. In five cases the diagnosis could be made before angiography, on the basis of CT findings. Angiographically, venous drainage was always seen into ascending cortical veins. Five cases demonstrated drainage via the olfactory vein into the basal vein of Rosenthal; in four this way was the principal route of drainage. Five patients underwent surgery, the therapy of choice. One fistula closed spontaneously after angiography. Two patients refused treatment and one was not treated because of his poor general condition. Because arterial supply was usually bilateral, from small branches of the ophthalmic artery, embolisation seemed to be more dangerous. Compared to dural fistulae in other locations the DAVM of the anterior cranial fossa have a higher risk of complications and should be treated even if asymptomatic at the time of diagnosis.

**Key words:** Dural arteriovenous malformation (DAVM) – Dural arteriovenous fistula – Ethmoidal dural fistula – Anterior cranial fossa – Spontaneous DAVM

There are few reports of dural arteriovenous fistulae at the base of the anterior cranial fossa. In comparison to those in the posterior cranial fossa and the cavernous sinus region, frontobasal dural arteriovenous malformations (DAVM) are less common. Since first description in 1963 [1] about 40 cases have been reported.

Nine cases of frontobasal dural fistulae were diagnosed in our department between 1988 and 1992, suggesting that they are not as rare as previously thought. The clinical and neuroradiological features are described and compared to those in published cases.

*Correspondence to:* J. Reul, Abt. Neuroradiologie, Klinikum der RWTH Aachen, Pauwelsstrasse, W-5100 Aachen, Germany

## Patients and methods

A DAVM of the anterior cranial fossa was diagnosed in nine men admitted to hospital between 1988 and 1992. Their age ranged from 24 to 69 years with highest incidence in the 6th decade (mean age 55 years).

All patients underwent CT using a high performance scanner (512 × 512 matrix). Scans before and after contrast medium were obtained in five cases. Four patients had only an unenhanced study. Bilateral selective intra-arterial digital angiograms of the carotid arteries were performed in all cases. One patient (case 2) was examined with MRI using a special Helmholtz head coil. T1-weighted spin echo images (TR 500 ms, TE 15 ms) before and after gadolinium-DTPA were obtained in axial, coronal and sagittal plane. Axial T2-weighted (TR 2400 ms, TE 90 ms) spin-echo images were also performed. Slice thickness was 5 mm.

Five patients underwent surgery.

## Results

### *Clinical features*

A summary of the symptoms and signs on admission is given in Table 1.

In three patients the fistula was an incidental finding; they had no symptoms or signs which could be related to the fistula. In four patients one or more seizures led to admission. One patient had progressive coma.

In the patients with an incidental DAVM, the indication for CT in case 7 was atypical headache and vertigo; these symptoms were related to degenerative change in the cervical spine. Case 6 suffered from depression of the elderly, while case 3 was admitted because of an acute stroke with a right hemiparesis and global aphasia due to occlusion of the left middle cerebral artery (MCA).

Seizures leading to admission occurred in four patients (cases 1, 2, 4, 5), in two of whom (cases 2, 4) they accompanied intracerebral haemorrhage. Case 1 was admitted because of a first generalised seizure. Three patients (cases 2, 4, 5) had recurrent seizures. Four patients (cases 2, 4, 8, 9) presented with an acute bleed. In cases 4 and 8 haemorrhage was in the basal portion of the frontal

**Table 1.** Clinical features

| Case/sex/age (years) | History | Examination |
|---|---|---|
| 1 m 64 | First seizure | Normal |
| 2 m 56 | Seizures, recurrent left parietal haemorrhage | Left hemiparesis |
| 3 m 57 | Left hemispere stroke due to MCA occlusion | Global aphasia, right hemiparesis |
| 4 m 24 | Seizures, recurrent headache | Disorientation, memory disturbance |
| 5 m 35 | Seizures, recurrent vertigo | Normal |
| 6 m 66 | Headache, depression for 1 year | Normal |
| 7 m 69 | Headache, vertigo | Normal |
| 8 m 66 | Acute headache with disorientation | Left hemiparesis, progressive coma |
| 9 m 59 | Headache, memory disturbance | Impairment of reading and writing |

MCA, Middle cerebral artery

**Table 2.** Initial CT findings

| Case | Before | After intravenous contrast medium |
|---|---|---|
| 1 | Left frontal oedema | Brightly enhancing basal vein |
| 2 | Parietal haemorrhage | Enhancing vein frontobasal |
| 3 | Ischemia left MCA territory | Not performed |
| 4 | Frontobasal blood | Not performed |
| 5 | Normal | Enhancing vein frontobasal |
| 6 | Normal | Enhancing vein frontobasal |
| 7 | Normal | Enhancing vein frontobasal |
| 8 | Frontobasal and intraventricular haemorrhage | Not performed |
| 9 | Left temporal haemorrhage | Not performed |

MCA, Middle cerebral artery

lobe, while in cases 2 and 9 it was distant from the shunt, in the parietal region (case 2) and in the left temporal lobe (case 9). Case 8 developed progressive coma due to intracerebral and intraventricular haemorrhage with increasing intracranial pressure.

The histories revealed no predisposing factors. Most of the patients had previously been asymptomatic, and none had a history suggestive of sinus thrombosis, head trauma, surgery or chronic inflammation in the paranasal sinuses. None had visual disturbance.

### *Computed tomography*

Unenhanced scans showed no abnormality in three cases (nos. 5–7). After administration of contrast medium a markedly enhancing frontobasal linear or tortuous hyperdensity was seen in five patients (cases 1, 2, 5–7), corresponding to the dilated draining veins of the fistula (Figs. 1a, 2a). The unenhanced scan in case 1 showed slight oedema in the basal part of the left frontal lobe. In the patient admitted because of an acute stroke (case 3) CT demonstrated an infarct in the left MCA territory; contrast-enhanced CT was not performed. In two patients (cases 4 and 8) CT showed a frontobasal intracerebral haematoma without signs pointing to an underlying malformation (Fig. 3); contrast-enhanced CT was not performed.

In case 2 recurrent haemorrhage was found in the left parietal region (Fig. 4a), without close topographic relation to the frontobasal AVM. CT in case 9 showed haematoma in the left temporal lobe, also distant from the frontobasal fistula.

The CT findings are summarised in Table 2.

### *Angiography*

The angiographic appearances (Table 3) were very similar in all cases. The fistula was at the level of the foramen caecum.

Arterial supply was by numerous small ethmoidal branches of the ophthalmic artery in all nine cases (Figs. 2c, 4b), with bilateral supply in six. Two showed an additional supply from the middle meningeal artery via the anterior falx artery (Fig. 2b), and two were supplied by ethmoidal branches of the internal maxillary artery (Fig. 2b).

Venous drainage always included ascending cortical veins (Fig. 2c). Variceal dilatation of the veins and pseudoaneurysms were seen in three cases (Fig. 2a, c). Five patients had drainage via olfactory veins into the basal vein of Rosenthal (Figs. 1b, 4b), and in four this was the principal route of drainage.

### *Surgical findings and clinical course*

In the five surgically treated patients the fistula was always found at the cribiform plate at the level of the foramen caecum. Feeding vessels were occluded by coagulation, with complete obliteration of the fistula. In case 4 the fistula closed spontaneously during angiography. Case 3 was not operated upon because of his poor neurological condition. Two patients (cases 7 and 9) refused treatment.

## Discussion

DAVM of the anterior cranial fossa are thought to be rare. Since the first description [1] the available literature comprises only about 40 cases. Detailed reviews of the published cases were given by Kobayashi et al. [2] and Halbach et al. [3].

There is widespread controversy about their development [5, 7, 9]; their etiology and pathophysiology are unexplained. They are mainly considered to be acquired lesions secondary to surgical manipulation, trauma, infection or venous obstruction [4–7]. One hypothesis [5] suggests that in the presence of increased venous pressure, due for example to sinus occlusion [8] physiological dural

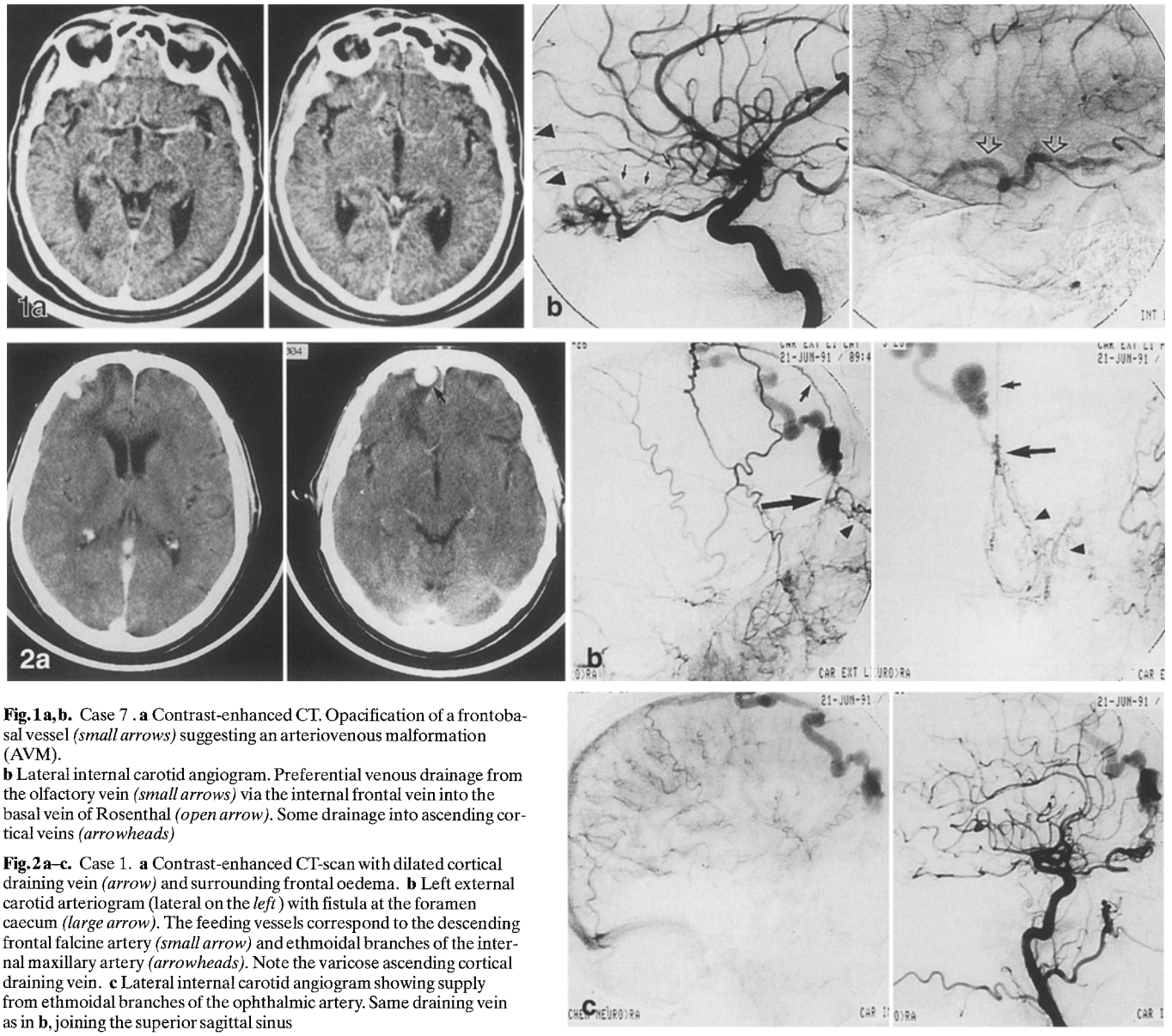


**Fig. 1a, b.** Case 7 . **a** Contrast-enhanced CT. Opacification of a frontobasal vessel *(small arrows)* suggesting an arteriovenous malformation (AVM).
**b** Lateral internal carotid angiogram. Preferential venous drainage from the olfactory vein *(small arrows)* via the internal frontal vein into the basal vein of Rosenthal *(open arrow)*. Some drainage into ascending cortical veins *(arrowheads)*

**Fig. 2a–c.** Case 1. **a** Contrast-enhanced CT-scan with dilated cortical draining vein *(arrow)* and surrounding frontal oedema. **b** Left external carotid arteriogram (lateral on the *left*) with fistula at the foramen caecum *(large arrow)*. The feeding vessels correspond to the descending frontal falcine artery *(small arrow)* and ethmoidal branches of the internal maxillary artery *(arrowheads)*. Note the varicose ascending cortical draining vein. **c** Lateral internal carotid angiogram showing supply from ethmoidal branches of the ophthalmic artery. Same draining vein as in **b**, joining the superior sagittal sinus

microshunts might enlarge, leading to the visible and sometimes symptomatic fistula. A relationship between the existence of a fistula and the embryonic development of the cerebral vasculature is also postulated [9]. Persistance of an enlarged olfactory emissary vein in the cribriform plate may be the anatomical basis for the development in this location of a fistula.

In all our patients, as in most reported cases there was no history of any of the above-mentioned causes nor of venous sinus occlusion. The second assumption therefore seems more likely. Both theories depend on the presence of microshunts in the dura mater.

The observed predominance of males is unexplained. All of our patients were men, as were nearly 90% of cases in the literature. This is in contrast to the posterior cranial fossa and cavernous sinus DAVM, with a predominance of females [3, 5–7, 10, 11]; our own 51 cases of DAVM in these regions show a male to female ratio of 2:3.

Clinically, the DAVM in the posterior cranial fossa is quite different from that of the anterior skull base [12]. In the posterior fossa DAVM there is often no other symptom than a bruit [13]; in a number of cases it is an incidental finding on Doppler ultrasonography. This type of DAVM is less dangerous and produces neurological deficits less often than the frontobasal variety [10, 13].

Houser et al. [5] demonstrated occlusion of the ipsilateral transverse or sigmoid sinus in 60% of their cases of DAVM of the posterior cranial fossa, and concluded that dural vessels within the sinus develop and lead to arteriovenous shunting during the organisation of a venous thrombosis. However, other workers, describing spontaneous closure of DAVM [14–18], suggest that elevation of venous pressure, with concomitant stasis of venous flow upstream results in thrombosis of the venous sinus, leading to occlusion of fistula. The aetiology and pathophysiology of DAVM are unexplained; those in the posterior cranial fossa might be caused to other mechanisms than those

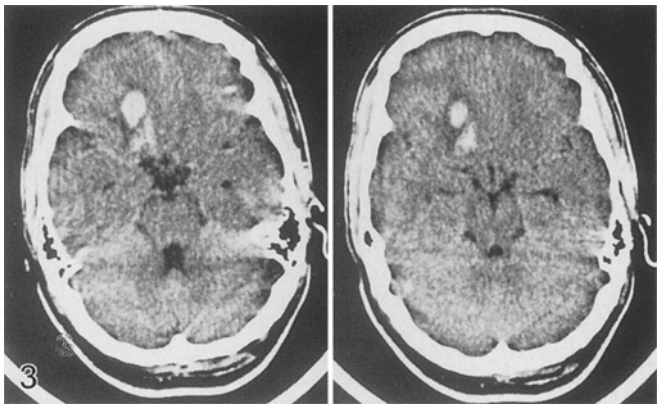

**Fig. 3.** Case 4. Frontobasal intracerebral haemorrhage along the draining vein. No subarachnoid haemorrhage

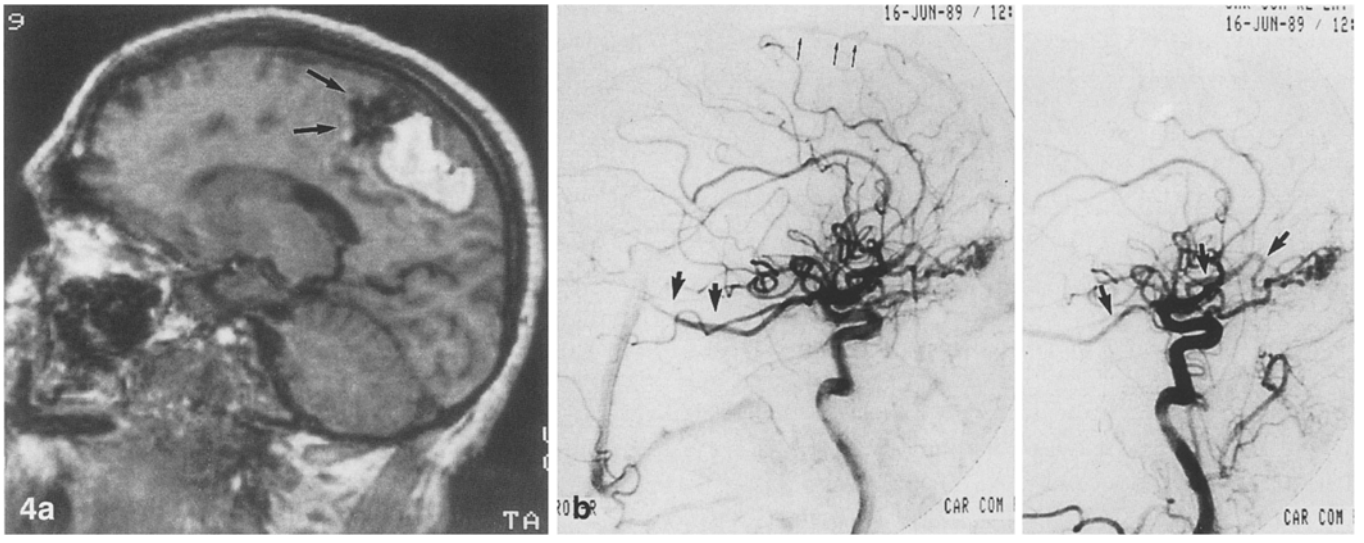


**Fig. 4a, b.** Case 2. **a** T1-weighted MRI demonstrating left parietal parenchymal damage *(arrows)* due to first bleeding distal to the frontobasal fistula and recurrent haemorrhage in this region. **b** Lateral internal carotid angiogram. Frontobasal fistula with same route of drainage as in case 7 into the basal vein of Rosenthal *(arrows)*. A little drainage into ascending cortical veins is seen. There are no signs of an AVM in the parietal region

of the anterior skull base, which may also explain the clinical and radiological differences. Phylogenetic abnormalities may have an influence on the development of DAVM [19].

In the light of the classification by Djindjian and Merland [4] of DAVM by venous drainage (Table 4) all nine fistulae must be classified as type III, because drainage into cortical veins was seen always. This classification, regardless of the location of the fistula, is of therapeutic importance because DAVM with cortical venous drainage are more often associated with severe complications such as haemorrhage and require a more aggressive therapeutic approach [13].

In a few cases of anterior fossa DAVM venous drainage into basal veins has been reported [20]. We found predominant drainage via olfactory veins into the basal vein of Rosenthal, not only into ascending cortical veins in almost half our cases. It therefore seems useful to divide type III into two subgroups: (a) with drainage into ascending cortical veins and (b) with predominant drainage into deep cerebral veins.

The symptoms and signs in our patients differed from those in the literature [2, 12, 21]. The presenting symptom is said to be haemorrhage, intracerebral or subarachnoid [22] in about 90% of published cases. In nearly half of our patients it was seizures which led to admission. A causal relationship between seizures and alteration or cerebral haemodynamics due to the shunt can be assumed, but could not be proved in the individual cases. Four of our patients had intracerebral haemorrhage but in only two was there a close topographical relationship to the fistula. Subarachnoid haemorrhage and subdural haematomas – according to the literature a common initial finding – did not occur in any of our cases. In three of nine cases the DAVM was an incidental finding.

In five patients without bleeding the diagnosis could be established by careful analysis of the CT (Fig. 5). A contrast-enhanced examination with thin slices through the frontobasal region allows in many cases identification of the draining veins and may establish the diagnosis before angiography.

Cases 2 and 9 differed from the others: the first had repeated haemorrhages at the same site in the left parietal region, distant from the frontobasal fistula, while the second was admitted because of a left temporal intracerebral haemorrhage. Angiography (in both) and repeated MRI (in case 2) did not demonstrate a vascular malformation in the vicinity of haemorrhage. It remains unclear if there was any relationship between the frontobasal fistula and the haemorrhage. Harding et al. [23] described two cases of caroticocavernous fistula complicated by intracerebral haemorrhage, and Fardoun et al. [24] a tentorial DAVM complicated by a temporoparietal haemorrhage. In all these cases a local elevation of venous pressure due to the

**Table 3.** Angiographic findings

| Case | Arterial supply | Venous drainage |
|---|---|---|
| 1 | Left IMA, MMA, OA<br>Right OA | ACV to SSS |
| 2 | Left –<br>Right OA | OV to BVR<br>ACV to SSS |
| 3 | Left OA<br>Right OA | ACV to SSS |
| 4 | Left OA<br>Right – | OV to BVR<br>ACV to SSS |
| 5 | Left MMA, OA<br>Right OA | ACV to SSS<br>OV to BVR |
| 6 | Left –<br>Right OA | ACV to SSS |
| 7 | Left OA<br>Right IMA, OA | OV to BVR<br>ACV to SSS |
| 8 | Left OA<br>Right OA | OV to BVR<br>ACV to SSS |
| 9 | Left OA<br>Right OA | ACV to SSS |

OA, Ophthalmic artery; IMA, internal maxillary artery; MMA, medial meningeal artery; OV, olfactory vein; ACV, ascending cortical vein; SSS, superior sagittal sinus; BVR, basal vein of Rosenthal

**Table 4.** Classification of dural arteriovenous malformations by venous drainage (Djindjian and Merland [4])

| | |
|---|---|
| Type I | Drainage into a sinus (or a meningeal vein) |
| Type II | Sinus drainage with reflux into cerebral veins |
| Type III | Drainage solely into cortical veins |
| Typ IV | With supra- or infratentorial venous lake |

route of drainage might explain the haemorrhage. In our cases the minimal drainage via ascending cortical veins makes a direct relationship questionable. A significant elevation of venous pressure is not very likely, but it would be of interest to discover whether other workers have similar experiences.

Treatment can be surgical or by embolisation. Surgically, the DAVM is easily coagulated at the foramen caecum, with complete occlusion of the fistula. Because of the multiple feeding vessels from the ophthalmic artery and inconstantly from the meningeal and maxillary arteries, interventional therapy by embolisation may be more dangerous or ineffective. Spontaneous closure is possible, as in all DAVM, but rare [16, 17, 25]. It occurred during angiography in one of our patients (case 4).

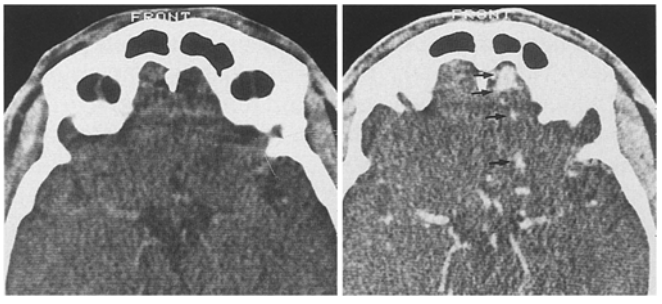

**Fig. 5.** Case 5. CT before and after intravenous contrast medium demonstrating abnormal frontobasal vessels, invisible before injection

## References


1. Lepoire J, Montaut J, Bouchot M, Laxenaire M (1963) Anévrismes artério-veineux infrafrontaux vascularisés par l'artére ethmoidale antérieure. A propos de trois observations. Neurochirurgie 9: 159–166
2. Kobayashi H, Hayashi M, Noguchi Y, Tsuji T, Handa Y, Caner HH (1988) Dural arteriovenous malformations in the anterior cranial fossa. Surg Neurol 30: 396–401
3. Halbach V V, Higashida RT, Hieshima GB, Wilson CB, Barnwell SL, Dowd CF (1990) Dural arteriovenous fistulas supplied by ethmoidal arteries. Neurosurgery 26: 816–823
4. Djindjian R, Merland JJ (1978) Superselective angiography of the external carotid artery. Springer, Berlin Heidelberg New York
5. Houser OW, Campbell JK, Campbell RJ, et al (1979) Arteriovenous malformation affecting the transverse venous sinus – an acquired lesion. Mayo Clin Proc 54: 651–661
6. Houser OW, Baker HL Jr, Rhoton AL Jr, et al (1972) Intracranial dural arteriovenous malformations. Radiology 105: 55–64
7. Chaudhary MY, Sachdev VP, Cho SH, Weitzner I, Puljic S, Huang YP (1982) Dural arteriovenous malformation of the major venous sinuses: an acquired lesion. AJNR 3: 13–19
8. Handa J, Yoneda S, Handa H (1975) Venous sinus occlusion with a dural arteriovenous malformation of the posterior fossa. Surg Neurol 4: 433–437
9. Olivecrona H, Ladenheim J (1957) Congenital arteriovenous aneurysms of the carotid and vertebral system. Springer, Berlin Göttingen Heidelberg
10. Lasjaunias P, Chiu M, Brugge KT, Tolosa A, Hurth M, Bernstein M (1986) Neurological manifestations of intracranial dural arterio-venous malformations. J Neurosurg 64: 724–730
11. Lasjaunias P, Halimi PH, López-Ibor L, Sichez JP, Hurth M, de Tribolet ND (1986) Traitement endovasculaire des malformations vasculaires durales (MVD) pures spontanées: revue de 23 cas explorés et traités entre mai 1980 et octobre 1983. Neurochirurgie 30: 207–223
12. Ito J, Imamura H, Kobayashi K, Tsuchida T, Sato S (1983) Dural arteriovenous malformations of the base of the anterior cranial fossa. Neuroradiology 24: 149–154
13. Lasjaunias P, Berenstein A (1987) Surgical neuroangiography. Springer, Berlin Heidelberg New York, pp 273–315
14. Kataoka K, Taneda M (1984) Angiographic disappearance of multiple dural arteriovenous malformations. Case report. J Neurosurg 60: 1275–1278
15. Landman JA, Braun IF (1985) Spontaneous closure of a dural arteriovenous fistula associated with acute hearing loss. AJNR 6: 448–449
16. Olutola PS, Eliam M, Molot M, Talalla A (1983) Spontaneous regression of a dural arteriovenous malformation. Neurosurgery 12: 687–690
17. Rohr J, Gauthier G (1985) Regression spontanée d'une fistule arterioveineuse dure-mérienne de la fosse postérieure. Rev Neurol (Paris) 141: 240–244
18. Magidson MA, Weinberg PE (1976) Spontaneous closure of a dural arterio-venous malformation. Surg Neurol 6: 107–110
19. Folkman J, Klagsbrun M (1987) Angiogenetic factors. Science 235: 442–447
20. Abumiya T, Kamiyama H, Murata J, Nunomura M, Chono Y, Kobayashi N, Abe H, Saito H, Miyasaka K, Abe S (1987) Dural arteriovenous malformation with uncommon draining veins in the anterior fossa. Case report. Neurol Med Chir (Tokyo) 27: 1195–1200
21. Katayama Y, Tsubokawa T, Moriyasu N, Kotani A (1980) Dural arteriovenous malformation in the anterior cranial fossa. Neurol Surg (Tokyo) 8: 1079–1085

22. Hayashi T, Tokunaga T, Matusuo M (1986) Dural arteriovenous malformation in the anterior fossa with subarachnoid hemorrhage. Kurume Med J 33: 187–191
23. Harding AE, Kendall B, Leonard TJK, Johnson MH (1984) Intracerebral haemorrhage complicating dural arteriovenous fistula: a report of two cases. J Neurol Neurosurg Psychiatry 47: 905–911
24. Fardoun R, Adam Y, Mercier P, Guy G (1981) Tentorial arteriovenous malformation presenting as an intracerebral hematoma. J Neurosurg 55: 976–978
25. Wilkins RH (1985) Natural history of intracranial vascular malformations: a review. Neurosurgery 16: 421–430
26. Aminoff MJ (1973) Vascular anomalies in the intracranial dura mater. Brain 96: 601–612
27. Aminoff MJ, Kendall BE (1973) Asymptomatic dural vascular anomalies. Br J Radiol 46: 662–667
28. Sutherland GR, King ME, Drake CJ, Peerless SJ, Vezina WC (1988) Platelet aggregation within cerebral arteriovenous malformations. J Neurosurg 68: 198–204
29. Balo J (1950) The dural venous sinuses. Anat Rec 106: 319–325
30. Barnwell SL, Halbach V V, Dowd CF, Higashida RT, Hieshima GB, Wilson CB (1991) Multiple dural arteriovenous fistulas of the cranium and spine. AJNR 12: 441–445
31. Graeb DA, Dolman CL (1986) Radiological and pathological aspects of dural arteriovenous fistulas. J Neurosurg 64: 962–967
32. Willinsky R, Lasjaunias P, Terbrugge K, Pruvost P (1988) Brain arteriovenous malformations: analysis of the angio-architecture in relationship to hemorrhage (based on 152 patients explored and/or treated at the Hospital de Bicetre between 1981 and 1986). J Neuroradiol 15: 225–237
33. Tanaka K, Yonekawa Y, Miyake H (1991) Dural arteriovenous malformation in the anterior cranial fossa: report of a case. Surg Neurol 36: 221–225
34. Kuwayama N, Takaku A, Nishijima M, Endo S, Hirano Y (1989) Multiple dural arteriovenous malformations. Report of two cases. J Neurosurg 71: 932–934
35. Terada T, Kikuchi H, Karasawa J, Nagata I (1984) Intracerebral arteriovenous malformation fed by the anterior ethmoidal artery. Case report. Neurosurgery 14: 578–582