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Transvenous embolization of dural caroticocavernous fistulae: technical considerations

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Abstract. Sixteen patients with symptomatic dural caroticocavernous fistulae were treated by transvenous embolization, via the jugular vein and inferior petrosal sinus. The fistula was occluded by thrombogenic coils. Complete resolution of symptoms and signs was achieved in 14 patients, and complete angiographic resolution was also obtained in 14 patients. Failures to achieve angiographic cure were attributed to failure to reach the fistula within the cavernous sinus precisely. Factors which make placement of the catheter at the fistula difficult are trabeculae within the cavernous sinus, a specific configuration of the superior ophthalmic vein and venous thrombosis. To improve the efficacy of transvenous embolization, every possible venous route to the cavernous sinus therefore should be tried, to facilitate reaching the fistula and the possibility of transvenous embolization should not be thwarted by venous thrombosis.

Key words: Dural arteriovenous fistula – Cavernous sinus – Transvenous embolization

Dural caroticocavernous fistulae (DCCF) occur predominantly in middle-aged women [1] and present with chemosis, proptosis, bruit and ophthalmoplegia [2]. The symptoms and signs are usually mild and not life-threatening, because the shunt is usually low-pressure and low-flow [1, 2]. The fact that spontaneous resolution is not infrequent [1] makes a minimally invasive form of therapy with a high cure rate desirable. Compression of the carotid artery has been reported to be effective in some patients [3]. However, when the symptoms are intolerable or the signs progressive, more aggressive therapy is warranted. This may take the form of irradiation [4], surgery [5], electrothrombosis [6], and transarterial embolization of the feeding arteries from the external [7] and internal carotid arteries [8]. Transvenous embolization [9–13] has recently been added to the range of treatments. However, it still seems difficult to obtain a complete cure in every patient [10, 12, 13]. We report our experience of transvenous embolization and discuss the ways of improving its efficacy.

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Materials and methods

Thirteen women and three men with a DCCF were treated with via a transvenous approach. Their ages ranged between 23 and 78 years, with an average of 54.4 years. The symptoms were chemosis (16 patients), proptosis (16), bruit (14), deterioration of visual acuity (1), double vision (16) and intolerable pain (9). Seven patients had undergone treatment prior to transvenous embolization: external compression in 5, ligation of the external carotid artery and electrothrombosis in 1 and embolization of the external carotid artery in 1.

Angiography included bilateral selective injection of the external and internal carotid arteries and vertebral artery. The blood supply of the DCCF was from the external and internal carotid arteries on one side in 5 patients, both external and one internal carotid artery in 10, and both external and internal carotid arteries in 1. Venous drainage was to the superior and inferior ophthalmic veins in 16 patients, the inferior petrosal sinus in 14, the contralateral cavernous sinus in 2, and to cortical veins in 9. Partial thrombosis of the superior ophthalmic vein was observed in 6 patients, and stenosis of the inferior petrosal sinus in 3. In 2 patients, the posterior portion of the cavernous sinus and the inferior petrosal sinus were not demonstrable. The cavernous sinus was partially thrombosed in 3 patients.

Feeding arteries from the external carotid artery were embolized transarterially in 8 patients prior to transvenous embolization.

Transvenous embolization was performed via the ipsilateral jugular vein: under local anaesthesia, the vein was punctured and a 5-F sheath was introduced. A 5-F catheter was placed at the junction of the jugular vein and inferior petrosal sinus. Through it, a Tracker-18 catheter was navigated through the inferior petrosal sinus to the cavernous sinus. Thrombogenic coils were placed at the fistula within the cavernous sinus. Coils were first placed in the anterior portion of the cavernous sinus to prevent outflow to the superior ophthalmic vein, then in the middle and posterior portions to prevent outflow to cortical veins. An angiogram was obtained to check for occlusion of the fistula (Fig. 1).

Angiography was repeated 1–8 months after embolization in all patients, who underwent ophthalmological and neurological examinations at 1- and 3-month intervals. Angiographic cure was defined as complete angiographic obliteration of the DCCF, and a clinical cure as total resolution of signs and symptoms.

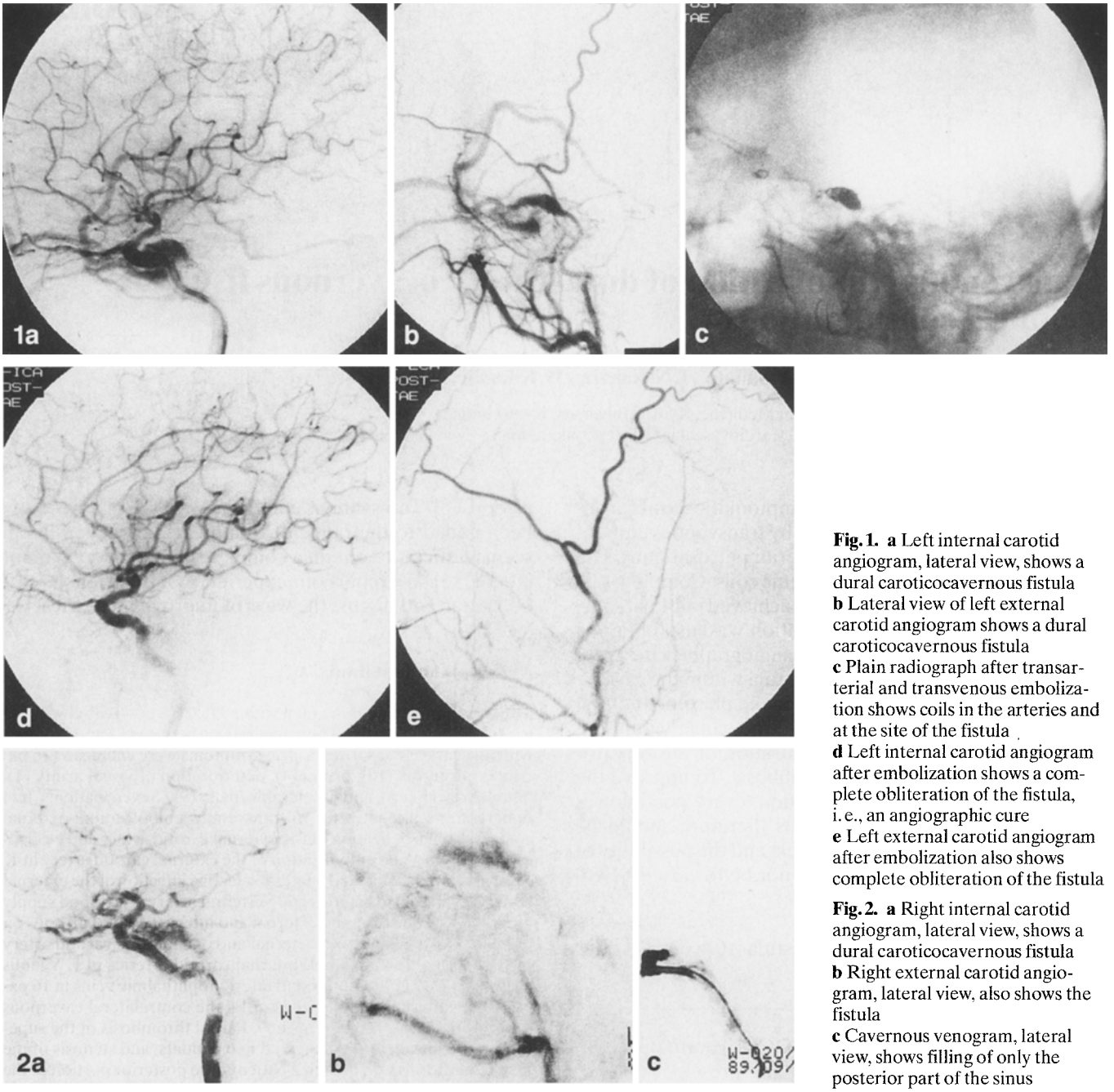


Fig. 1. **a** Left internal carotid angiogram, lateral view, shows a dural caroticocavernous fistula
b Lateral view of left external carotid angiogram shows a dural caroticocavernous fistula
c Plain radiograph after transarterial and transvenous embolization shows coils in the arteries and at the site of the fistula
d Left internal carotid angiogram after embolization shows a complete obliteration of the fistula, i.e., an angiographic cure
e Left external carotid angiogram after embolization also shows complete obliteration of the fistula

Fig. 2. **a** Right internal carotid angiogram, lateral view, shows a dural caroticocavernous fistula
b Right external carotid angiogram, lateral view, also shows the fistula
c Cavernous venogram, lateral view, shows filling of only the posterior part of the sinus

Results

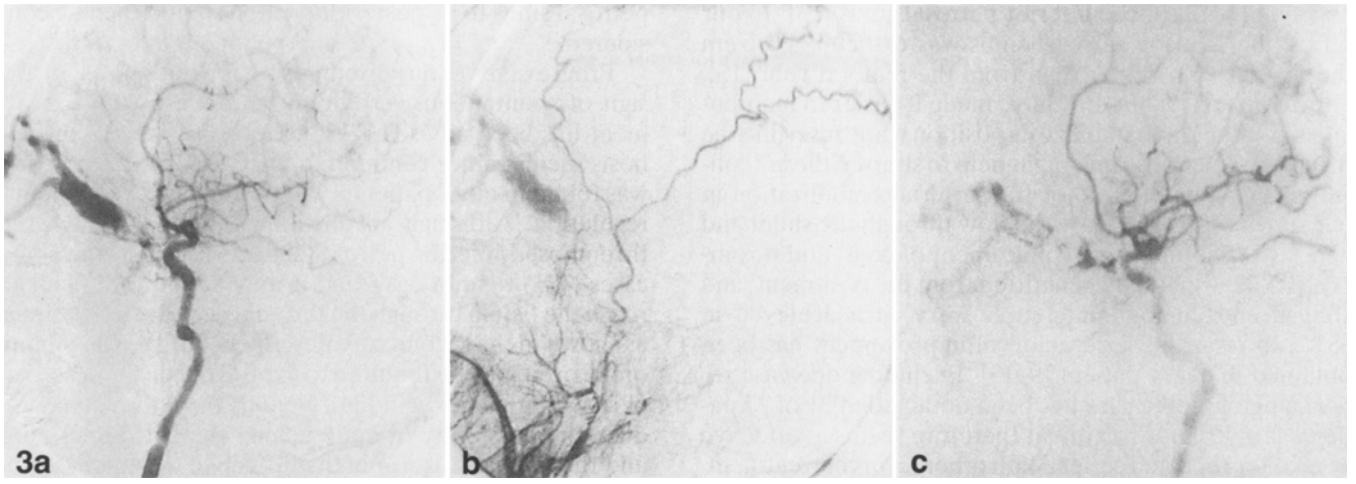
In 9 patients, the symptoms were less marked immediately after transvenous embolization. Disappearance of the DCCF was shown angiographically in 14 patients.

The follow-up period ranged from 6 to 28 months, mean 15.7 months. Clinical cure was achieved in 14 patients, and angiographic cure in the same number. Clinical cure occurred in 1 patient with a residual DCCF on angiography. Anisocoria, present before treatment, persisted in 1 patient, although angiographic cure was achieved. Symptoms were greatly alleviated in 1 patient with a residual DCCF. Recurrence of the DCCF was not observed.

Complications related to transvenous embolization occurred in 7 patients: transient aggravation of chemosis, recovering within 2 weeks (3 patients), aggravation of a sixth nerve palsy, recovering within 2 months (2 patients), aggravation of a third nerve palsy, recovering within 2 months (1 patient), and epidural extravasation from perforation of the inferior petrosal sinus, healing spontaneously and fortunately causing with no clinical manifestations (1 patient).

Failures

In a 23-year-old man with right chemosis, proptosis, bruit, and abducens nerve paresis, angiography revealed a right DCCF, fed by both external carotid arteries and the right internal carotid artery, draining into the supe-



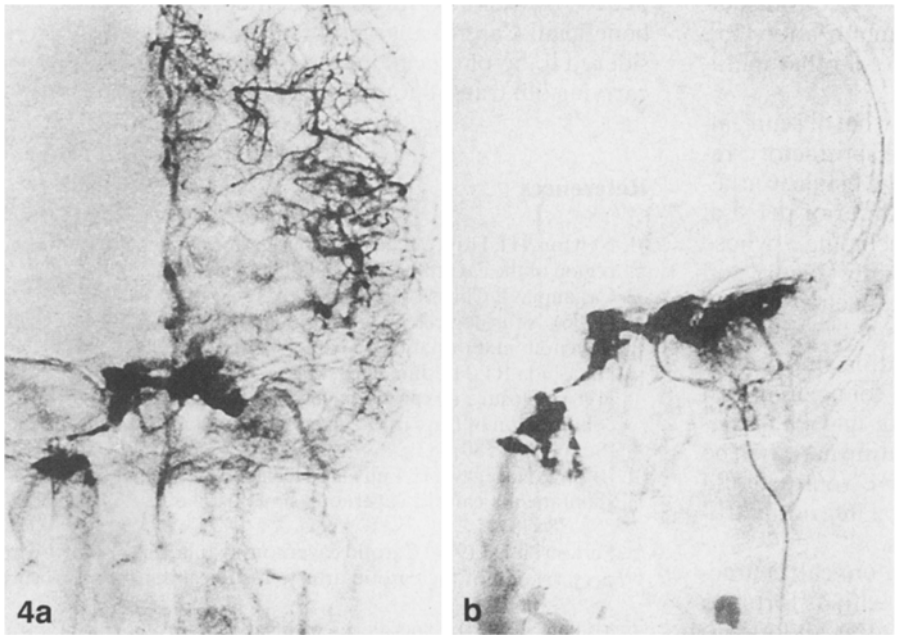


Fig. 3. **a** Left internal carotid angiogram, lateral view, shows dural caroticocavernous fistula with anterior venous drainage only
b Left external carotid angiogram, lateral view, after embolization of the branches feeding the fistula, shows residual fistula
c Left internal carotid angiogram, after transvenous embolization, shows coils in the ophthalmic vein and the residual fistula

Fig. 4. **a** Left common carotid angiogram, anteroposterior view, does not show the left inferior petrosal sinus
b Cavernous sinogram via a catheter passed through the non-opacified sinus

rior ophthalmic vein and the inferior petrosal sinus; no venous anomalies were observed. A catheter could be navigated into the posterior part of the cavernous sinus via the inferior petrosal sinus. Injection showed opacification of the posterior part of the sinus (Fig. 2). Despite manipulation of a thin guide wire, it was impossible to advance to the anterior part of the sinus. The feeding arteries from the external carotid arteries were occluded, leaving the DCCF fed by only the right meningohypophyseal artery. Thrombogenic coils were then placed in the posterior part of the cavernous sinus. A final angiogram showed the DCCF still to be patent. Clinical cure was obtained at 1 month later, but the DCCF, fed by the right artery of the foramen rotundum was demonstrated on a 6-month follow-up angiogram. The patient declined further treatment.

In a 43-year-old woman with left chemosis, proptosis, bruit, and abducens nerve paresis, angiography revealed a left DCCF fed by the left external and internal carotid arteries, draining into the superior and inferior ophthalmic veins; the cavernous and inferior petrosal sinuses were not demonstrated. The feeding arteries from the external carotid artery were occluded. A catheter could be navigated into the posterior part of the cavernous sinus via the inferior petrosal sinus, but it was impossible to advance to the anterior part or the ophthalmic veins. A catheter was then navigated into the superior ophthalmic vein, through the superficial temporal vein from the external jugular vein. However, it was impossible to pass through the superior orbital fissure, and the thrombogenic coils were placed in the ophthalmic vein. A final angiogram showed the DCCF still patent (Fig. 3). Although greatly alleviated, the symptoms were still present at a 6-month follow-up. Follow-up angiography has not yet been performed.

Discussion

The goal of transvenous embolization of DCCF is to promote thrombosis within the cavernous sinus, which may be the mechanism of spontaneous cure. Transvenous embolization has been carried out via the superior ophthal-

mic vein [9–12] or the inferior petrosal sinus [13]. In our series, the inferior petrosal sinus was catheterized from the jugular vein rather than from the femoral vein [13]. Shortening its length may have made it easier to manipulate the catheter, to stabilize its position while inserting the thrombogenic coils, and might help to shorten the embolization session. The role of transarterial embolization in our series was to decrease the flow through the shunt and hence to facilitate precise placement of coils, and thrombosis. All our patients benefitted from the treatment, and clinical and angiographic cures were each achieved in 88%. In reported series, clinical improvement has been obtained in every patient [9–13]. Excluding one case report, angiographic cure has been obtained in 21 of 27 patients [10–13]; our treatment therefore seems as effective as in other reported series. With other forms of treatment, an cure rates of 88% have been obtained by electrothrombosis [14], and of 77% by transarterial embolization [15]. Our results do not seem inferior to those of other methods.

Even so, the causes of failure are somewhat obscure, although, our experience suggests that unsatisfactory results may be ascribed to failure to reach the fistula. Catheterization of the cavernous sinus via the inferior petrosal sinus was possible in all our patients, including 2 whose sinus was not demonstrated angiographically (Fig. 4), and 3 patients whose sinus was stenosed. Halbach et al. [13] have reported similar experience and suggest that recent thrombosis might not make catheterization impossible. The inferior petrosal sinus may have no connection with the jugular vein (in 7% of individuals), or may be immature plexiform (in 24%) [16]. Catheterization may then be difficult or impossible and an alternative route should therefore be selected, via the contralateral inferior petrosal sinus or the superior ophthalmic veins.

It has been suggested that the anatomical characteristics of the superior ophthalmic vein, with its tortuous course, with abrupt angulations, in the orbit and a constant narrowing at the superior orbital fissure [17, 18], may make retrograde catheterization difficult [13]. However, these anatomical features have never been reported to impede catheterization [12], although fragility of the vein has been stressed as a cause of complications [10, 19]. Catheterization of the superior ophthalmic vein from the external jugular vein is feasible, but the tortuosity of the veins leading to the superior ophthalmic vein may have made it more difficult. It may be advisable not to proceed with catheterization when it is difficult, and to adopt other methods, such as surgical exposure or direct puncture under angiographic control of the superior ophthalmic vein [9–12].

Even though a catheter can be advanced to the cavernous sinus via the inferior petrosal sinus or superior ophthalmic vein, it can not always reach the fistula, as shown in the cases described, but, this has never been reported previously [9–13, 19]. We agree that the cavernous sinus is an unbroken venous channel with trabeculae [20, 21]; if it compartmentalized by thick trabeculae, it may be difficult or impossible to reach the fistula from any one approach. Combined approaches via the superior ophthalmic vein to the anterior portion of the sinus and through the inferior petrosal sinus to its posterior portion should then be considered.

Progressive venous thrombosis has been suggested as a sign of spontaneous resolution, and conservative treatment has been advised [7, 22]. However, venous thrombosis seems rather common in DCCF [7, 13, 22, 23], and was found in our 8 patients, without signs of spontaneous resolution. Although advancing a catheter through the thrombosed inferior petrosal sinus is successful in some cases [13], it is not easy, and it may be more difficult to reach the fistula through the thrombosed cavernous sinus, as shown in the second case described. Surgical unroofing of the orbit may be required to expose the thrombosed superior ophthalmic vein [24]. Venous thrombosis may reduce the possibility of approaching the cavernous sinus and the fistula. It is ironic that effective treatment is impeded by venous thrombosis, heretofore considered beneficial. Careful angiographic study is therefore, considered to be of crucial importance, if the opportunity of carrying out transvenous embolization is not to be lost.

References

1. Newton TH, Hoyt WF (1970) Dural arteriovenous shunts in the region of the cavernous sinus. Neuroradiology 1: 71–81
2. Lasjaunias P, Chiu M, Brugge KT, Tolia A, Hurth M, Bernstein M (1986) Neurological manifestations of intracranial dural arteriovenous malformations. J Neurosurg 64: 724–730
3. Higashida RT, Hieshima GB, Halbach VV, Bentson JR, Goto K (1986) Closure of carotid cavernous sinus fistulae by external compression of the carotid artery and jugular vein. Acta Radiol [Suppl] 369: 580–583
4. Bitoh S, Hasegawa H, Fujiwara H, Nakao K (1982) Irradiation to spontaneous carotid cavernous sinus fistulas. Surg Neurol 17: 282–286
5. Parkinson D (1973) Carotid cavernous fistula: direct repair with preservation of the carotid artery. Technical note. J Neurosurg 38: 99–106
6. Mullan S (1974) Experiences with surgical thrombosis of intracranial berry aneurysms and carotid cavernous fistulas. J Neurosurg 41: 657–670
7. Viñuela F, Fox AJ, Debrun GM, Peerless SJ, Drake CG (1984) Spontaneous carotid-cavernous fistulas: clinical, radiological, and therapeutic considerations. Experience with 20 cases. J Neurosurg 60: 976–984
8. Halbach VV, Higashida RT, Hieshima GB, Hardin C (1989) Embolization of branches arising from the cavernous portion of the internal carotid artery. AJNR 10: 143–150
9. Courtheoux P, Labbé D, Hamel C, Lecoq PJ, Jahara M, Thèron J (1987) Treatment of bilateral spontaneous dural carotid-cavernous fistulas by coils and sclerotherapy. Case report. J Neurosurg 66: 468–470
10. Teng MMH, Guo WY, Juang C, Wu CC, Chang T (1988) Occlusion of arteriovenous malformations of the cavernous sinus via the superior ophthalmic vein. AJNR 9: 539–546
11. Takahashi A, Yoshimoto T, Kawakami K, Sugawara T, Suzuki J (1989) Transvenous copper wire insertion for dural arteriovenous malformations of cavernous sinus. J Neurosurg 70: 751–754
12. Monsein LH, Debrun GN, Miller NR, Nauta HJW, Chazaly JR (1991) Treatment of dural carotid-cavernous fistulas via the superior ophthalmic vein. AJNR 12: 435–439
13. Halbach VV, Higashida RT, Hieshima GB, Hardin CW, Pribram H (1989) Transvenous embolization of dural fistulas involving the cavernous sinus. AJNR 10: 377–383
14. Ishikawa M, Handa H, Taki W, Yoneda S (1982) Management of spontaneous carotid-cavernous fistulae. Surg Neurol 18: 131–139

15. Halbach VV, Higashida RT, Hieshima GB, Reicher M, Norman D, Newton TH (1987) Dural fistulas involving the cavernous sinus: results of treatment in 30 patients. Radiology 163: 437–442
16. Shiu PC, Hanafee WN, Wilson GH, Rand RW (1968) Cavernous sinus venography. Am J Roentogenol 104: 57–62
17. Offret G, Aron-Rosa S, Metzger J, Doyon D (1966) La phlébographic orbitaire. Acta Radiol 5: 441–452
18. Hanafee WN, Shiu PC, Dayton GO (1968) Orbital venography. Am J Roentgenol 104: 29–35
19. Uflacker R, Lima S, Ribas GC, Piske RL (1986) Carotid-cavernous fistulas: embolization through the superior ophthalmic vein approach. Radiology 159: 175–179
20. Bedford MA (1966) The "cavernous" sinus. Br J Ophthal 50: 41–46
21. Miyazaki H, Kurihara M, Ishizaka H, Kawano M, Mori K (1981) Intracavernous aneurysms and carotid-cavernous fistulas. A clinicopathological study. Neurol Med Chir (Tokyo) 21: 111–120
22. Seeger JF, Gabrielsen TO, Giannotta SL, Lots PR (1980) Carotid-cavernous sinus fistulas and venous thrombosis. AJNR 1: 141–148
23. Brismar G, Brismar J (1976) Spotaneous carotid cavernous fistulas. Phlebographic appearance and relation to thrombosis. Acta Radiol 17: 180–192
24. Hanneken AM, Miller NR, Debrun GM, Nauta HJW (1989) Treatment of carotid-cavernous sinus fistulas using a detachable balloon catheter through the superior ophthalmic vein. Arch Ophthalmol 107: 87–92