PAPER

Diagnosis and treatment of dural carotid–cavernous fistulas: a consecutive series of 27 patients

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Objectives: To report clinical characteristics, angiographical findings and results of endovascular treatment of patients presenting with dural carotid–cavernous fistulas (DCCFs).

Method: Retrospective analysis of 27 consecutive patients with DCCF referred to a specialised interventional neuroradiology department.

Results: Orbital and neuro-ophthalmological symptoms were the most common clinical presentation at diagnosis (n = 25). The venous drainage of the fistula involved the ipsilateral superior ophthalmic vein in 24 patients, the contralateral cavernous sinus in 6 and a leptomeningeal vein in 5 patients. Thrombosis of at least one petrosal sinus was found in 23 patients. 7 patients did not receive endovascular treatment: 3 had spontaneous DCCF obliteration, and 4 had only minor clinical symptoms and no leptomeningeal venous drainage on an angiogram. 20 patients received endovascular treatment via either a transvenous (n = 16) or a transarterial approach (n = 4). Complete occlusion of the fistula was obtained in 14 of 16 (87%) patients treated by the transvenous approach and in 1 of 4 (25%) patients treated by the transarterial approach. 16 patients had early clinical improvement after endovascular treatment. One patient had a cerebral haemorrhage after transvenous embolisation of a DCCF with leptomeningeal drainage. On follow-up, all patients treated by the transarterial route remained symptomatic, whereas 10 of 14 (71%) patients cured by the transvenous route were asymptomatic.

Conclusions: Transvenous embolisation is a safe and efficient endovascular approach to treat patients with DCCF. However, this technique requires a long learning curve.

Dural carotid–cavernous fistulas (DCCFs) constitute a rare condition caused by abnormal communications between meningeal branches of the internal or external carotid artery and the cavernous sinus, typically diagnosed in postmenopausal women.¹ Its underlying pathophysiology is yet unknown, although a link between DCCF and sinus thrombosis has been suggested.^{2–3} Patients with DCCF are often diagnosed with delay, because the clinical symptoms seem to be usually mild and non-specific. In addition, spontaneous occlusion of the fistula may occur.^{4–5} However, some patients with DCCF may have more severe symptoms such as proptosis, ophthalmoplegia, visual impairment or intracranial haemorrhage. The clinical presentation of a DCCF has shown to be closely related to the venous drainage pattern: drainage via the superior ophthalmic vein is associated with orbital and neuro-ophthalmological symptoms, whereas leptomeningeal drainage correlates with neurological symptoms or intracranial haemorrhage.^{6–8}

For many years, DCCFs have been almost exclusively treated by endovascular embolisation. The endovascular technique has dramatically changed over the past 15 years since the development of the transvenous approach in association with controllable detachable coils.

The aim of this study was to analyse the clinical presentation, angiographical findings and treatment outcome in a retrospective series of 27 consecutive patients with DCCF who were referred to the Department of Interventional Neuroradiology, Hôpital Lariboisière, Paris, France.

MATERIAL AND METHODS

Patients and material

We retrospectively reviewed the records of 27 consecutive patients with DCCF referred to the Department of

Interventional Neuroradiology, Lariboisière Hospital, between 1993 and 2004. We excluded the patients treated before 1993 as no detachable coils were used at our institution for this indication before this date.

Clinical presentation was classified into orbital (chemosis, conjunctival injection, raised intraocular pressure or proptosis), neuro-ophthalmological (ocular palsy or decreased visual acuity), neurological (headaches, focal neurological symptoms or intracranial haemorrhage) or others. The diagnosis of a DCCF was based on standard transfemoral six-vessel angiography using a digital subtraction technique (Advantix, GEMS, Buc, France) with the selective visualisation of both external and internal carotid arteries, and both vertebral arteries (fig 1).

Two independent reviewers (EH and CS) analysed all angiograms following a predefined protocol. The venous drainage pattern was categorised into ipsilateral superior ophthalmic vein, contralateral superior ophthalmic vein via a coronary sinus, leptomeningeal veins, superior petrosal sinus or inferior petrosal sinus. Lack of opacification of the ipsilateral inferior or superior petrosal sinuses was considered to be due to thrombosis. Underlying thrombosis was also assumed in cases where dural venous structures descending from the cavernous sinus towards the internal jugular vein were not opacified.

Characteristics of patients and angiographic data

Table 1 summarises the clinical and morphological baseline characteristics of the patients (n = 27). The mean age was 64 (standard deviation (SD) 12, range 32–80) years; 18 (66%) were women.

Orbital and neuro-ophthalmic symptoms were the most common modes of clinical presentation. They were ipsilateral to

Abbreviation: DCCF, dural carotid–cavernous fistulas

Figure 1 Different orbital and neuro-ophthalmological presentations of patients with dural carotid-cavernous fistula (DCCF). (A) Isolated conjunctival injection of the right eye associated with drainage via the superior ophthalmic and leptomeningeal veins. (B) Conjunctival injection and proptosis of the left eye in a patient with a thrombosed ipsilateral superior ophthalmic vein. (C, D) Conjunctival injection, chemosis and ptosis of the right eye with a clearly visible right proptosis on a computed tomography scan of the brain in a patient with a right-sided DCCF and thrombosed inferior and superior petrosal sinuses. Informed consent was obtained for publication of this figure.

the fistula in 22 patients, bilateral in three and contralateral to the fistula in two patients. The most common clinical symptoms at diagnosis were diplopia ($n = 12$, 45%), conjunctival injection ($n = 11$, 41%), chemosis ($n = 10$, 37%), proptosis ($n = 10$, 37%) and visual deterioration ($n = 8$, 30%).

In all, 14 of 27 (52%) fistulas were located in the left cavernous sinus. A total of 24 (89%) patients had superior ophthalmic drainage, 6 (22%) had coronary sinus drainage and 5 (18.5%) had leptomeningeal drainage. Angiography showed thrombosis of the inferior petrosal or of the superior petrosal sinus in 23 (85%) patients. Among the four patients whose petrosal sinuses were permeable, one had an ophthalmic superior vein thrombosis.

Treatment

The treatment options were either medical or endovascular. Treatment included aspirin 250 mg/day or a low dose of prednisolone 0.5 mg/kg/day. Endovascular treatment consisted of either transarterial or transvenous embolisation. In patients treated by the transvenous approach, venous access to the cavernous sinus was either through the inferior petrosal sinus the superior petrosal sinus, or the superior ophthalmic vein (via the facial vein).

Transvenous embolisation

Figure 2 describes the technique of transvenous embolisation. All interventions were performed with patients under general anaesthesia. A femoral arterial access was placed to perform cerebral angiography during and after the procedure. Venous access was obtained by a cervical puncture of the internal jugular vein. This access is preferred to femoral access because the guiding catheter can be pushed more easily. After placing a 5 French sheath (Terumo) into the internal jugular vein, a 5 French catheter vertebral curve (Terumo) was introduced and navigated up to the foramen jugulare. For recanalisation of a thrombosed inferior petrosal sinus, the catheter tip was oriented internally and anteriorly just at the level of the foramen jugulare. A channel was then created in a blind manner inside the thrombosed sinus using a 0.035-inch guidewire (Terumo). The guidewire was then slowly pushed forward following the direction of the inferior petrosal sinus.

Once the 0.035-inch guidewire reached the level of the cavernous sinus, its correct position was verified by cerebral angiography on two projections. The guidewire was then withdrawn while keeping its position on a fluoroscopy road map. This trajectory was used to eventually navigate a microcatheter with double markers (Excelsior 14, Boston Scientific Montigny le Bretonneux, France) over a 0.014-inch microguide wire (Transend 14, Boston Scientific). When the cavernous sinus was reached, detachable platinum coils (GDC, Boston Scientific; Detach 10 and 18, Cook Charenton, France) were deposited, starting from the point where the arteriovenous shunts were more visible. The objective was to achieve dense packing of the cavernous sinus. For tortuous facio-ophthalmic access, we used Berenstein liquid coils (Boston Scientific) that are less rigid than detachable coils. Subcutaneous low-weight heparin was given over 72 h to avoid thrombus formation at the site of the jugular vein puncture.

Transarterial embolisation

Figure 3 describes an example for transarterial embolisation. Embolisation was performed under sedation analgesia. A 5 French guiding catheter was placed in the common carotid artery and a microcatheter was coaxially introduced over a 0.014-inch micro-guidewire. All coaxial systems were continuously flushed under pressure with saline. Embolisation was achieved with particles injected in free flow into the internal maxillary artery. We used Embospheres (Biospheres) beginning with a size of 300–500 μm diameter followed by 500–700 μm . Injection was interrupted when the flow inside the internal maxillary artery stopped.

Outcome

The immediate angiographic result at the end of the endovascular treatment was classified as being either complete (ie, lack of opacification of the cavernous sinus throughout the arterial phase) or partial (ie, shunt flow was decreased but still persistent).

Clinical outcome was defined as the patient's clinical status on the last follow-up visit. Clinical outcome results were stratified into cured (when a patient was asymptomatic), improved (when symptom intensity had decreased),

Table 1 Clinical and angiographic baseline characteristics and follow-up of 27 patients with dural carotid-cavernous fistula

Age/sex/ lesion side	Delay in diagnosis	Clinical signs	Venous drainage	Thrombosed sinus or vein	Endovascular access	Results of the treatment	Clinical symptoms at last follow-up
32/M/L	7 days	Bilateral orbital and neuro-ophthalmic signs, tinnitus	SOV Coronary sinus Ipsilateral IPS	SPS	SOV	CO	Relapse at 1 month: ipsilateral IPS
73/M/R	12 months	Ipsilateral orbital and neuro-ophthalmic signs	SOV	SPS and IPS	SOV	CO	2 years: normal Improved (11 months)
50/M/R	3 months	Ipsilateral orbital signs, tinnitus	SOV	SPS and IPS	Ipsilateral IPS	CO	Normal (4 months)
74/M/R	2 months	Ipsilateral orbital and neuro-ophthalmic signs	SOV	SPS and IPS	None	Spontaneous cure	Unchanged (5 months)
59/M/R	1.5 months	Ipsilateral tinnitus, headaches	Ipsilateral IPS and SPS	SOV	None	Spontaneous cure	Improved (5 months)
68/F/L	0	Ipsilateral tinnitus, headaches	Ipsilateral SPS	IPS and SOV	Ipsilateral SPS	CO	Normal (6 months)
67/M/L	4 months	Bilateral orbital and neuro-ophthalmic signs, headaches	SOV Coronary sinus	SPS and IPS	Ipsilateral IPS	CO	Normal (17 months)
77/F/R	10 months	Ipsilateral orbital and neuro-ophthalmic signs, headaches	SOV	SPS and IPS	Ipsilateral IPS	CO	Improved (1.5 months)
61/M/R	2.5 months	Ipsilateral tinnitus, orbital and neuro-ophthalmic signs, headaches	SOV Leptomeningeal drainage		Ipsilateral IPS	PO	Normal (2 months)
79/F/L	0	Ipsilateral orbital signs, ocular palsy, loss of visual acuity	SOV	SPS and IPS	Ipsilateral IPS	CO	Normal (11 months)
71/M/R	0	Ipsilateral orbital and neuro-ophthalmic signs	SOV Leptomeningeal drainage	IPS	Ipsilateral IPS	CO	Normal (27 months)
80/F/L	22 months	Ipsilateral orbital and neuro-ophthalmic signs	SOV Leptomeningeal drainage	SPS	Ipsilateral IPS	CO	Improved (17 months)
72/F/L	0	Orbital and neuro- ophthalmic signs contralateral to the fistula	Coronary sinus	SPS and IPS	Transarterial (failure of transvenous routes)	PO	Lost to follow-up
74/F/R	0	Ipsilateral orbital and neuro-ophthalmic signs, tinnitus	SOV Leptomeningeal drainage	SPS and IPS	Ipsilateral IPS	CO	Improved (4 years)
74/F/R	1.5 months	Ipsilateral orbital and neuro-ophthalmic signs	SOV	SPS and IPS	Transarterial	PO	Improved (11 months)
62/F/L	10 months	Ipsilateral orbital and neuro-ophthalmic signs	SOV	SPS and IPS	Transarterial	PO	Unchanged (6 months)
60/F/L	5 months	Ipsilateral orbital and neuro-ophthalmic signs	SOV	SPS and IPS	Ipsilateral IPS	PO	Normal (5 years)
76/F/R	2 months	Bilateral orbital and neuro-ophthalmic signs, focal neurological signs, headaches	Coronary sinus SOV Leptomeningeal drainage	SPS and IPS	Contralateral IPS	CO Cerebral haemorrhage	Normal (12 months)
52/F/L	3.5 months	Ipsilateral orbital signs	Ipsilateral IPS and SPS	SOV	None	Spontaneous cure	Normal (2 months)
54/F/R	8.6 months	Ipsilateral orbital and neuro-ophthalmic signs, headaches	SOV	SPS and IPS	None	Spontaneous cure	Normal (9 months)
58/F/L	5 months	Ipsilateral orbital and neuro-ophthalmic signs, headaches	SOV Coronary sinus drainage	SPS	Ipsilateral and contralateral IPS	CO	Normal (9 months)
36/F/R	23 months	Ipsilateral orbital and neuro-ophthalmic signs	SOV	SPS	SOV	CO	Improved (5 days)
75/M/L	7.5 months	Ipsilateral orbital signs	Ipsilateral IPS	SPS and SOV	None	–	Normal (14 days)
69/F/L	6.2 months	Orbital and neuro- ophthalmic signs contralateral to the fistula, headaches	Coronary sinus drainage		None	–	Improved (9 months)
73/F/R	4 months	Ipsilateral orbital and neuro-ophthalmic signs	SOV	SPS and IPS	Ipsilateral IPS	CO	Normal (5 months)
68/F/L	1.5 months	Ipsilateral orbital signs, headaches	SOV	SPS and IPS	None	–	Normal (3 months)
49/F/L	12.5 years	Ipsilateral orbital and neuro-ophthalmic signs	SOV	SPS and IPS	Transarterial	CO	Improved (7 days)

CO, complete occlusion; DCCF, dural carotid-cavernous fistula; F, female; IPS, inferior petrosal sinus; L, left, M, men; PO, partial occlusion; R, right; SOV, ipsilateral superior ophthalmic vein; SPS, superior petrosal sinus.

Figure 2 Transvenous embolisation with recanalisation of the inferior petrosal sinus. Angiography of the right internal carotid artery in lateral projection (LP) showing the dural arteries of the carotid siphon feeding the right cavernous sinus shunts. (B) The lack of opacification of the inferior petrosal sinus. Angiography of the right external carotid artery, showing the small feeding arteries to the fistula. (C) Angiography of the right internal carotid artery in anteroposterior projection (APP), showing the drainage in the right cavernous sinus and the left superior ophthalmic vein (arrow). (D) Angiography of the left internal carotid artery in APP showing the small dural arteries (arrow) arising from the left siphon to the right carotid cavernous fistula. (E) Plain film in APP showing the 0.035-inch guidewire creating the channel through the inferior petrosal sinus. (F) Plain film in LP of the guidewire. (G) Plain film showing the cast of coils at the end of the embolisation. (H) Control angiography of the right common carotid artery at the end of embolisation, showing the cured fistula.

unchanged or worsened (in patients where symptoms had worsened or new symptoms had appeared).

RESULTS

Correlation between venous drainage and clinical signs

All patients with superior ophthalmic drainage of the DCCF presented with orbital or neuro-ophthalmological symptoms. All but one of the six patients with coronary sinus drainage had orbital or neuro-ophthalmological symptoms that were bilateral in three and contralateral to the fistula in two patients. One of the five patients with leptomeningeal drainage presented with focal neurological symptoms (right hemiplegia and aphasia), whereas no such symptoms existed among the 22 patients whose baseline angiography showed no leptomeningeal drainage.

Treatment

Table 2 summarises the angiographic treatment results.

Of the seven patients who received no endovascular treatment, three had spontaneous DCCF occlusion with symptom regression after the baseline angiography and the remaining four had only mild clinical signs, low-flow fistulas and absence of leptomeningeal venous drainage.

Of the 20 patients who were treated by embolisation, 16 underwent transvenous embolisation during 22 procedures. Complete DCCF occlusion was achieved in 14 patients, leading to a transvenous success rate of 87% (14/16).

To achieve complete DCCF occlusion, several venous routes were chosen:

- The ipsilateral inferior petrosal sinus was used in 13 patients: 10 had a thrombosis of the sinus requiring mechanical recanalisation to access the cavernous sinus. This manoeuvre failed in two patients for whom a contralateral inferior petrosal sinus route and an ipsilateral superior petrosal sinus route were used.
- The contralateral inferior petrosal sinus was used in three patients who showed coronary sinus drainage on baseline angiography. Complete DCCF occlusion was achieved in one patient and partial occlusion in another. The access to the fistula failed in the third patient who finally had a successful embolisation via a facio-ophthalmic approach.
- The ipsilateral superior petrosal sinus approach was used in two patients, including one patient with failure of the inferior petrosal sinus access. One patient achieved complete

Figure 3 Transarterial embolisation. (A) Angiography of the right internal carotid artery, showing the dural arteries of the carotid siphon feeding the fistula. (B) Angiography of the right external carotid artery, showing the dural arteries supplying the fistula and especially the artery of the foramen rotundum (arrow). The small arterial diameters can be seen, which would not permit their superselective catheterisation. (C) Angiography of the right external carotid artery after embolisation with particles that were injected in free flow into the internal maxillary artery. Dural arteries of the carotid siphon were not embolised and the fistula was not cured; however, the symptoms improved after embolisation.

DCCF occlusion, but the embolisation failed in the other patient who was then successfully treated via a facio-ophthalmic approach.

- The facio-ophthalmic route was used in four patients and complete occlusion was achieved in three patients. This approach has been introduced during the past 3 years, which explains why few patients have been treated by this route in our series.

Finally, four patients underwent transarterial embolisation. All procedures were performed before 1997. Transarterial embolisation was chosen when transvenous embolisation was considered impossible. Complete occlusion of the fistulas was

Table 2 Angiographic results after 26 endovascular embolisations in 20 patients with dural carotid-cavernous fistula

	Procedures (n)	Final result		
		Complete occlusion	Partial occlusion	Failure of access to the fistulas
Venous routes	22	14	3	5
Ipsilateral patent IPS	3	2	1	0
Ipsilateral thrombosed IPS	10	7	1	2
Contralateral IPS	3	1	1	1
Facio-ophthalmic vein	4	3	0	1
Ipsilateral SPS	2	1	0	1
Arterial routes	4	1	3	0
Total	26	15	6	5

IPS, inferior petrosal sinus; SPS, superior petrosal sinus.

achieved in one patient and a partial occlusion of the fistulas in three without any complications.

Outcome

Overall, 12 of the 16 patients with transvenous embolisation and all 4 with transarterial embolisation showed early clinical improvement after the procedure. Three patients remained clinically unchanged, and one worsened because of cerebral haemorrhage. This patient had focal neurological symptoms, showing a DCCF with leptomeningeal drainage into the middle cerebral vein. Immediately after a transvenous embolisation, the patient had acute temporal haemorrhage. The venous guiding catheter in the inferior petrosal sinus might have reduced the outflow through this sinus, thereby leading to increased outflow through the middle cerebral vein and eventual rupture. Nonetheless, the patient recovered from the stroke, showing a normal neurological exam at 1-year follow-up.

All but two patients were followed up with a mean follow-up time of 11.5 (SD 13, range 15 days–64 months). At the time of the last follow-up, DCCF-related symptoms had completely disappeared in 10 patients and improved in eight. Most patients with a complete cure had been treated by a transvenous approach. By contrast, none of the patients treated by a transarterial approach became completely asymptomatic, although all had clinically improved. One patient had recurrent symptoms (proptosis contralateral to the fistula) 2 months after the first transvenous endovascular embolisation through the superior orbital vein with initial DCCF occlusion. After symptom recurrence, he underwent a second transvenous embolisation via the ipsilateral inferior petrosal sinus, leading to complete occlusion of the recurrent fistula and improvement of associated symptoms.

DISCUSSION

Most of our patients were postmenopausal women, and most presented with orbital or neuro-ophthalmological symptoms of moderate intensity.^{9–10} All patients with an unusual drainage into the coronary sinus, had either bilateral or contralateral clinical symptoms. Focal brain symptoms were present in only one patient whose DCCF showed leptomeningeal drainage, confirming prior reports of a possible correlation between the venous drainage pattern and the presenting symptoms.^{6–11} Although leptomeningeal venous drainage is considered rare in patients with DCCF, it was seen in 5 of 27 (18.5%) of our patients, which is far higher than previously reported.^{6–12}

Spontaneous occlusion of the DCCF occurred in 7 of 27 (26%) patients in our cohort. Among them, both petrosal sinuses were

patent in three patients, whereas two had either complete thrombosis of the ipsilateral superior orbital vein or partial thrombosis of the ipsilateral cavernous sinus. These findings suggest that the persistence of the physiological venous flow from the cavernous sinus to the petrosal sinuses or a thrombosis upstream to the dural fistula may be factors favouring spontaneous occlusion.

The transvenous endovascular approach is currently considered the best therapeutic option for patients with symptomatic DCCF.^{13–14} It was first described by Halbach *et al.*,¹² who used steel coils and sclerosing liquid injections into the cavernous sinus via the superior ophthalmic vein. Complete DCCF obliteration can also be obtained by an inferior petrosal sinus route, which represents probably the easiest, shortest and safest approach even in patients with inferior petrosal sinus thrombosis, as in eight of our own patients.^{13–15} A superior petrosal sinus approach has been reported as an alternative to catheterisation of the inferior petrosal sinus or of the superior ophthalmic vein.¹⁶ However, the superior petrosal sinus must be patent, because mechanical recanalisation has proved hazardous because of the anatomical proximity to the vein of Labbé. As an alternative to inferior or superior petrosal sinus pathways, a facio-ophthalmic route may be used, as shown in four of our patients, three of whom had successful occlusion.¹⁷

In this retrospective series of patients with DCCF, more patients were completely asymptomatic at follow-up after transvenous embolisation than after transarterial treatment of the DCCF. On the basis of these data, transvenous embolisation constitutes our preferred treatment strategy. Only in patients where venous access is not possible, may arterial embolisation be considered.

We found a high frequency (85%) of thrombosis of both the ipsilateral inferior and superior petrosal sinuses associated with DCCF. As the inferior petrosal sinus can be bypassed via drainage through a basilar plexus, we assumed that thrombosis is the underlying mechanism in all cases where descending venous pathways from the cavernous sinus to the internal jugular vein were not visualised on an angiogram.¹⁸

Whether petrosal sinus thrombosis is a cause or a consequence of the DCCF is as yet unknown. Two pathophysiological hypotheses have been suggested in the past. The first hypothesis is that petrosal sinus thrombosis occurs before the development of dural shunts, thereby leading to increased pressure in the carotid-cavernous sinus and secondary recanalisation of embryonic arteriovenous communications.¹⁹ Prior reports on concomitant congenital or acquired coagulopathies in patients with dural arteriovenous fistulas further support this idea.²⁰ The second hypothesis is that petrosal sinus thrombosis occurs after the development of dural shunts due to the vascular wall stress induced by increased blood flow velocities.²¹ In addition, worsening of clinical symptoms as a result of either stenosis or thrombosis of the draining veins has been reported in dural arteriovenous fistulas.²² Further prospective studies, including a systematic investigation of haemostatic parameters and multimodal brain imaging, will be needed to better understand the natural history and the underlying physiopathology of DCCFs.

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