

Traumatic Carotid Cavernous Sinus Fistula: Serial Angiographic Studies from the Day of Trauma

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BACKGROUND AND PURPOSE: The purpose of this study was to ascertain the early angiographic features characteristic of traumatic carotid cavernous sinus fistulas (CCFs).

METHODS: Eight patients with severe craniofacial injuries underwent emergency diagnostic and therapeutic angiography for intractable oronasal bleeding, starting on an average of 6.7 hours after trauma. Carotid angiograms and the clinical manifestation of traumatic CCFs were then reviewed retrospectively to determine characteristic angiographic features.

RESULTS: In four of the eight patients, no arteriovenous fistulas were found in the cavernous sinuses and symptomatic CCF did not occur during the follow-up period. In the remaining four patients, dural CCFs (Barrow type B) were observed, unilaterally in three patients and bilaterally in one. One of these four patients subsequently became symptomatic and required transarterial coil embolization.

CONCLUSION: Traumatic dural CCFs are frequently observed in the early stage of severe craniofacial trauma, if investigated. Although their spontaneous disappearance is known, some of these do become symptomatic and need treatment.

Traumatic carotid cavernous sinus fistula (CCF) involves either direct communication between the internal carotid artery and the cavernous sinus (Barrow type A) or between dural branches of the internal or external carotid artery and the cavernous sinus (Barrow types B-D) (1, 2). Angiographic demonstration of a traumatic CCF, either the direct variety or the dural type, from the day of trauma is rare, because symptoms of traumatic CCF usually do not appear immediately after trauma (3) and angiography is rarely used in the diagnosis of acute head trauma. We had the occasion to perform diagnostic and therapeutic angiography on the day of trauma in eight patients with intractable oronasal bleeding. These angiograms enabled us to observe traumatic CCFs in the acute stage. We reviewed these angiograms retrospectively to determine the early characteristic angiographic features of traumatic CCFs.

Methods

Eight patients who suffered severe craniofacial trauma resulting in profuse, intractable, oronasal bleeding required diagnostic and therapeutic angiography on the day of trauma. All patients were males with an age range from 19 to 62 years (mean age, 27.5 years). Glasgow Coma Scale score on admission ranged from 3 to 14 (mean score, 6.6). CT was performed to determine the presence of intracranial lesions or skull-base fractures in all patients.

When tight nasal and oral packing failed to stop the oronasal bleeding, all eight patients underwent therapeutic superselective embolization with polyvinyl alcohol particles and/or platinum coils. Angiography was performed from 1.5 to 17 hours after trauma (mean time to angiography, 6.7 hours). Control of the oronasal bleeding was successful in all patients. In a retrospective review of the patients' carotid angiograms, special attention was paid to arteriovenous fistulas in the cavernous sinuses. The angiographic data were then compared with clinical manifestations of traumatic CCFs.

Representative Case Reports

Case 1

A 20-year-old man with severe craniofacial trauma following a traffic accident underwent angiography 6 hours after sustaining the trauma. The patient's lacerated left sphenopalatine artery was superselectively embolized. Internal carotid angiograms showed dural CCFs bilaterally (Barrow type B) (Fig 1). Shunt flow was greater on the left than on the right. Drainage was toward the left superior and inferior petrosal sinuses. Right external carotid angiograms showed a right middle meningeal artery to meningeal vein fistula draining mainly to the pterygoid plexus and superior sagittal sinus, and partially to the right cavernous sinus. This arteriovenous fistula was occluded with

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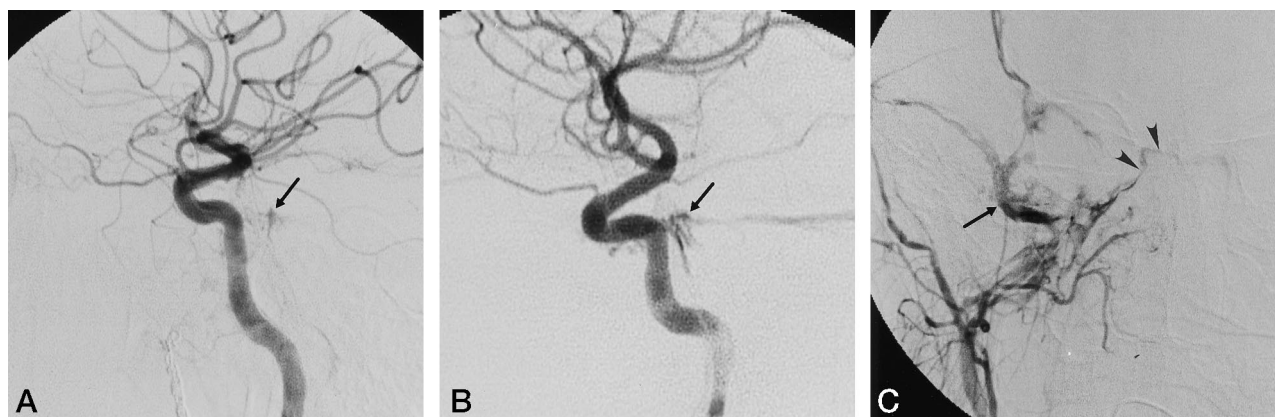


FIG 1. Case 1: angiograms obtained 6 hours after trauma.

- A, Right internal carotid angiogram, lateral view, shows a dural CCF (Barrow type B) (arrow), which disappeared by day 20.
 B, Left internal carotid angiogram, lateral view, shows a larger dural CCF (Barrow type B) on the left (arrow), which also disappeared by day 20.
 C, Right external carotid angiogram (anteroposterior view) shows a middle meningeal artery to meningeal vein fistula (arrow), mainly draining to the pterygoid plexus and superior sagittal sinus and partially to the cavernous sinus (arrowheads).

platinum coils. Follow-up angiograms on day 20 showed no CCF bilaterally. The patient remained asymptomatic during the follow-up period of 11 months.

Case 2

A 20-year-old man with severe craniofacial trauma caused by the impact of a falling object was in a state of cardiopulmonary arrest upon admission owing to massive oronasal bleeding. After successful cardiopulmonary resuscitation and blood transfusion, diagnostic and therapeutic angiography was started 80 minutes after trauma, although the patient remained comatose. Active bleeding from the bilateral facial and sphenopalatine arteries was controlled by superselective embolization.

Carotid angiograms showed a dural CCF (Barrow type B) in the left cavernous sinus but no CCF in the right cavernous sinus (Fig 2). Late-phase angiograms revealed venous flow in the left cavernous sinus but no venous flow in the right cavernous sinus, suggesting occlusion of the right cavernous sinus by thrombosis. Angiography on day 5 showed bilateral dural CCFs (Barrow type B) with greater shunt flow on the right than on the left. Since the whole face was markedly swollen and the patient was comatose, the symptoms of the dural CCFs could not be precisely evaluated during the first 2 weeks. A bruit, however, was audible over the right eye.

Follow-up angiography at 1 month showed obviously increased flow in the right CCF. At this time, chemosis and proptosis of the right eye were apparent. Treatment of the CCF was intentionally delayed because of a local infection (epidural abscess). Carotid angiograms obtained 2 months after ictus showed a right dural CCF (Barrow type B) and no CCF in the left cavernous sinus. The right dural CCF was finally occluded at 3 months by transarterial embolization with interlocking detachable coils (Target Therapeutics, Fremont, CA). The fistula was caused by a tear of the right meningohypophyseal trunk of the internal carotid artery.

Results

In four of the eight patients in our study group, no arteriovenous fistulas were found in the cavernous sinuses. Two, however, died of brain damage within 20 days. The remaining two did not develop a symptomatic CCF during respective follow-up periods of 3

months and 3 years. Only one of the four patients had a skull-base fracture.

In the four remaining patients, dural CCFs (Barrow type B) were observed: unilaterally in three patients and bilaterally in one. External carotid angiograms did not show arteriovenous fistula in the cavernous sinus in any patients, but a middle meningeal artery to meningeal vein fistula was evident in one patient (case 1). Owing to severe craniofacial trauma, it was difficult to determine whether these dural CCFs were symptomatic during the acute stage. In one patient (case 2), a symptomatic CCF developed in the follow-up period; another patient died on day 7 of associated brain damage. The remaining two patients did not incur a symptomatic CCF during respective follow-up periods of 4 months and 11 months. All four patients had a skull-base fracture.

Discussion

In most traumatic CCFs, the fistulas are of Barrow type A (2–5), but dural CCFs (Barrow types B–D), supplied by the inferolateral trunk or meningohypophyseal trunk of the internal carotid artery or by the middle meningeal artery, accessory meningeal artery, distal internal maxillary artery, or ascending pharyngeal artery, may occur (1, 6–8). Traumatic dural CCFs are rare, accounting for about 3% of dural CCFs (Barrow types B–D) (8, 9). Symptoms of traumatic dural CCFs are essentially the same as those of spontaneous CCFs.

Traumatic middle meningeal artery to meningeal vein fistulas draining to the cavernous sinus, as in our case 1, are not CCFs, but mimic type-C CCFs because of the similar angiographic features and clinical symptoms (8, 10–13). Traumatic type-C CCFs are usually fed by the middle meningeal artery or accessory meningeal artery (7, 8). Traumatic type-D CCFs have also been reported (6, 8). An iatrogenic, traumatic type-B CCF was reported to have occurred during emboliza-

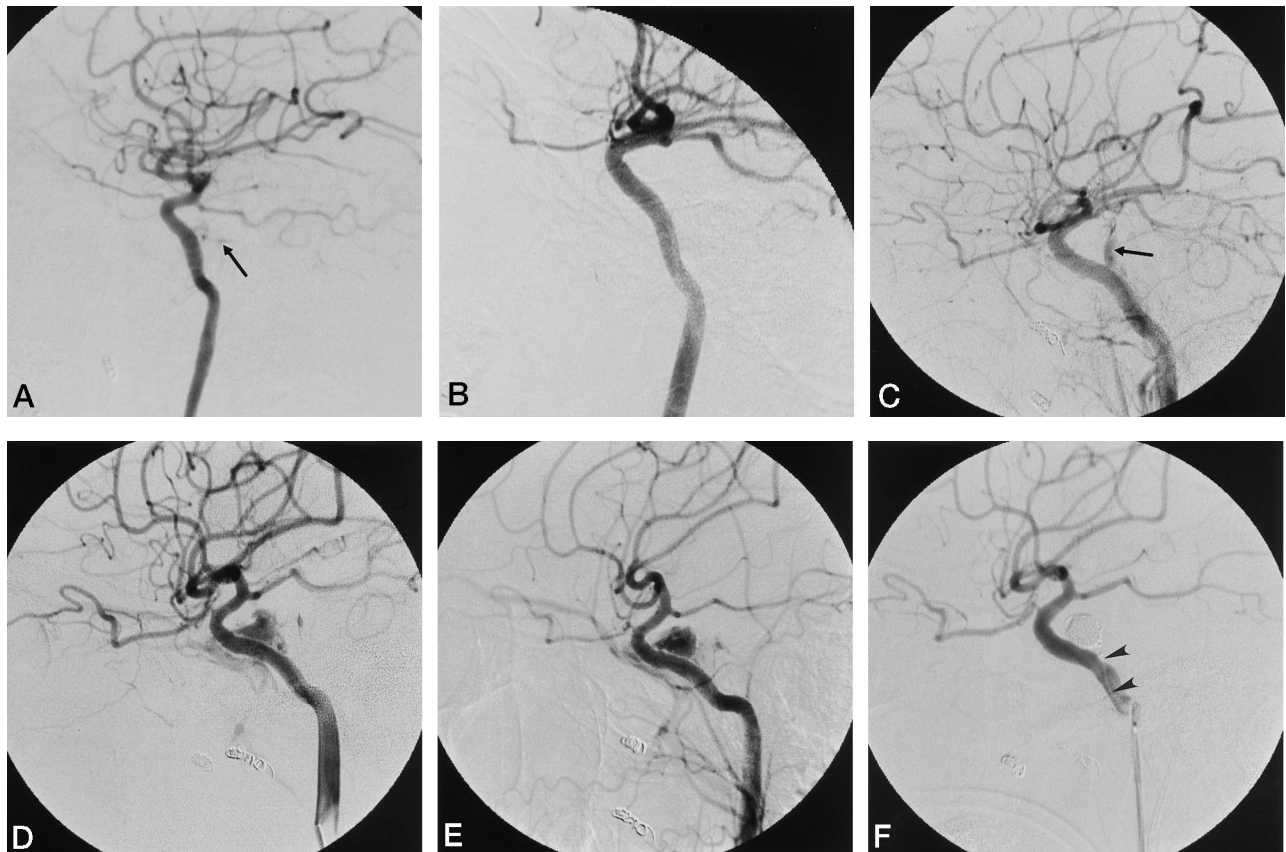


FIG 2. Patient 2: serial angiograms of a traumatic dural CCF from the day of trauma.

A, Left internal carotid angiogram, lateral view, taken 80 minutes after the trauma shows a dural CCF (Barrow type B, arrow), which was not seen 2 months after ictus.

B, Right internal carotid angiogram, lateral view, taken 80 minutes after trauma does not show any arteriovenous fistulas in the right cavernous sinus.

C, Right carotid angiogram, lateral view, taken on day 5, shows a dural CCF (Barrow type B, arrow) in the right cavernous sinus.

D, Right carotid angiogram, lateral view, 1 month after ictus shows a dural CCF (Barrow type B) on the right side.

E, Right carotid angiogram, lateral view, 2 months after ictus, shows increased arteriovenous shunt flow.

F, Right carotid angiogram, lateral view, obtained 3 months after ictus, during transarterial coil embolization of the dural CCF, shows the microcatheter (arrowheads) passing through the meningo-hypophyseal trunk of the right internal carotid artery.

tion of a meningioma through selective catheterization of the meningo-hypophyseal trunk (14). Although a traumatic, bilateral type-B CCF caused by minor head trauma was spontaneously obliterated (15), most symptomatic traumatic dural CCFs require treatment by either transarterial (6, 7, 14, 16) or transvenous embolization, or their combination (9). All four traumatic CCFs in our series were type B.

It seems that traumatic dural CCFs have a single feeding artery or smaller number of feeding arteries than do spontaneous dural CCF. It has been postulated that traumatic dural CCFs are caused by either resolution and fistulization of the thrombosis in the cavernous sinus or by direct tear of the dural branches connecting directly to the cavernous sinus (see comment by Mullan in [7]). We believe that the dural CCFs in this series were caused by the latter mechanism because of the immediate appearance of the CCFs after trauma. In case 2, no CCF was seen on the right side initially, but subsequently a type-B CCF was seen. This delayed appearance of a dural CCF might be attributed to acute thrombosis in the cavernous sinus and its resolution, to rupture of the pseudoan-

eurysm formed in the dural branches, or to resolution and late fistulization of the thrombosis in the cavernous sinus.

Conclusion

Owing to a paucity of literature regarding traumatic dural CCFs, their natural history is not understood. However, recognition of traumatic dural CCFs is clinically important, because it allows proper selection of treatment strategies (7). Asymptomatic traumatic dural CCFs or those with minor symptomatology can be treated conservatively, like those of spontaneous origin. From our limited experiences, it can be concluded that severe craniofacial trauma, especially when accompanied by skull-base fractures, may frequently cause dural CCFs in the acute stage, but not all such fistulas become symptomatic. Some of these disappear spontaneously while some symptomatic ones may require treatment similar to that given spontaneous dural CCFs.

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