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A Mironov

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Selective Transvenous Embolization of Dural Fistulas without Occlusion of the Dural Sinus

Angel Mironov

Summary: Two patients with dural arteriovenous fistulas (DAVFs) and unsuccessful transarterial embolizations were treated with a technique for selective transvenous embolization. A 5F catheter was advanced from a femoral vein access into the internal jugular bulb and a catheter was navigated through the sinus lumen into the involved cortical veins or the parasinusal venous draining channels of the DAVFs. The venous recipients at the nidal level of the DAVFs were occluded by fibered platinum coils. Complete angiographic cure was effected in both patients, with occlusion of the venous recipients and the nidus, although the sinus segments next to the nidus of the DAVFs remained patent. Placement of coils in a transsinusal route into the venous channel of a DAVF yet outside the sinus lumen can result in complete obliteration of the fistula without damage to the physiological function of the dural sinuses.

Transarterial attempts to place embolic materials within the nidus of dural arteriovenous fistulas (DAVFs) tend to be largely ineffective. Nevertheless, transvenous placement of embolic agents using a transfemoral approach within the diseased segment of a dural sinus associated with a fistula can result in complete obliteration of the dural fistula (1). The most significant limitation of transvenous embolization is created by the unique hemodynamic pattern of the venous dural sinuses themselves. In fact, placement of embolic agents into a fistulous segment of sinus that is being used as a conduit for venous drainage of the remaining brain carries a high risk of venous infarction or hemorrhage. We describe a technique for selective transvenous embolization of dural fistulas in which there is no occlusion of the sinus lumen.

Case Reports

Case 1

A 72-year-old man presented with spontaneous parenchymal hemorrhage in the right parietal lobe. Angiography revealed a DAVF located on the right side close to the middle of the superior sagittal sinus, with venous drainage only through a cortical vein parallel to the superior sagittal sinus with access into the confluens (Fig 1A). Both the right and left middle meningeal arteries and the left anterior meningeal artery (via the left ophthalmic artery) supplied the fistula. Transarterial embolization of the right middle meningeal artery with a mixture of Ethibloc (Ethicon, Hamburg, Germany) and iodized oil in a ratio of 1:2 achieved complete angiographic obliteration of

the nidus. At the 6-month follow-up, angiography revealed recanalization of the fistula through the right transosseous scalp branches of both the temporal superficial (Fig 1B) and the occipital arteries, indicating that safe and effective transarterial therapy was now not feasible. Therefore, a 5F catheter was advanced from a femoral vein access into the internal jugular bulb, and a Tracker 18 catheter (Target Therapeutics, Calif) was navigated into the involved cortical vein (Fig 1C).

Transcatheter pressure measurements in the venous conduit were recorded before and after the transvenous embolizations through the microcatheter coupled to a transducer and a digital pressure monitor (Hewlett Packard, Waltham, Mass). For each procedure, the measurements were repeated several times during a 2-minute period. The venous pressure of the middle superior sagittal sinus was 10/9 mm Hg, that in the venous recipient before the transvenous embolization was 38/37 mm Hg, and the simultaneous systemic (brachial) blood pressure was 165/95 mm Hg. The lumens of the venous recipients were occluded with complex-shaped fibered platinum coils (Target Therapeutics). After the occlusion, the pressure in the venous recipient dropped to 12/11 mm Hg. Complete angiographic closure of the DAVF occurred at the time of embolization, and the same result was documented in a follow-up angiogram 1 month after the procedure (Fig 1D).

Case 2

A 45-year-old man had a 2-year history of a right ear pulse-synchronous bruit and headache. Angiography revealed a DAVF located at the right transverse-sigmoid junction (Fig 2A). The right external artery supplied the fistula from the middle meningeal artery, the occipital artery, and the posterior auricular artery. Venous drainage was through a short venous channel into the sigmoid sinus. The venous conduit of the fistula corresponded anatomically to the inner part of a mastoid emissary vein. Transarterial embolization of both the mastoid branch of the occipital artery and the posterior branch of the middle meningeal artery with a mixture of Ethibloc and iodized oil in a ratio of 1:3 resulted in complete obliteration of these pedicles. However, anatomic cure was not achieved, because the remaining supply of the DAVF by the posterior auricular artery and both the petrous and temporal convexity branches of the meningeal arteries was inaccessible for safe transarterial embolization. Therefore, a Tracker 18 catheter was placed from the transvenous femoral and transsinusal route into the venous conduit of the fistula itself (Fig 2B). The transcatheter venous pressure in the venous recipient increased to 42/38 mm Hg; the pressure at the sigmoid sinus below the drainage of the venous recipient was 18/14 mm Hg, and the systemic (brachial) blood pressure was 145/90 mm Hg. There

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From the Department of Neuroradiology, Kantonsspital Aarau, CH-5001 Aarau, Switzerland. Address reprint requests to Angel Mironov, MD.

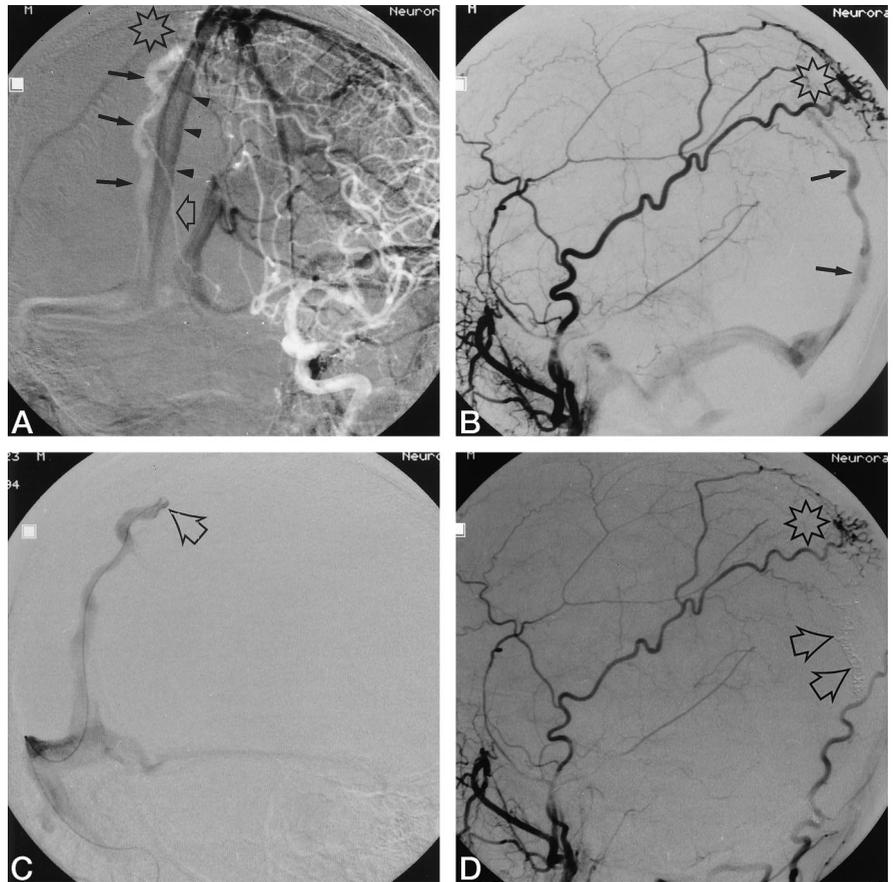
FIG 1. Case 1: 72-year-old man with parenchymal hemorrhage in the right parietal lobe.

A, Sequential subtraction of oblique left common carotid angiogram shows the supply to the fistula (*star*) located on the right side close to the middle of the superior sagittal sinus, via both the left ophthalmic (*open arrow*) and the middle meningeal arteries. The main supply was by the right middle meningeal artery. Note on the right the bright venous recipient (*arrows*) parallel to the dark superior sagittal sinus (*arrow-heads*), which is patent.

B, At 6-month follow-up, lateral right external carotid angiogram shows re-canalization of the fistula (*star*) through the transosseous branches of both the right temporal superficial and occipital arteries, with opacification of the venous recipient (*arrows*).

C, Oblique phlebogram of the involved cortical vein (same view as in A) shows the tip of the microcatheter at the level of the nidal area (*arrow*) in the lumen of the venous recipient outside the superior sagittal sinus. In this position, the lumen of the venous recipient was occluded with complex-shaped fibered platinum coils.

D, Lateral right external carotid angiogram shows obliteration of the fistula (*star*). Note the coils in the venous recipient, which is still occluded (*arrows*).



was a considerable narrowing of the sigmoid sinus at this level. Selective embolization of the venous recipient with two complex-shaped fibered platinum coils (2 mm in diameter and 10 mm, in length) resulted in occlusion of the DAVF. The primary narrowed sigmoid sinus was not occluded, nor was there damage to the vein of Labbé or the occipitotemporal veins (Fig 2C). In the sigmoid sinus, venous pressure after embolization fell to 9/8 mm Hg. In the transverse sinus, venous pressure above the drainage of the venous recipient of the fistula remained unchanged. The patient was fully recovered, with no remaining signs or symptoms, the day after embolization. Follow-up angiography 1 month later showed complete obliteration of the DAVF (Fig 2D).

Discussion

Classification of DAVFs has evolved with increasing knowledge of their pathophysiology and natural course. They have been classified on the basis of location, arterial supply, venous drainage, implications for treatment (2), and locational pathogenesis (3). Classification by type of venous drainage correlates with neurologic symptoms and hemorrhagic risk (4, 5); however, these are only temporary expressions of the venous pattern state, because spontaneous progression or regression due to secondary venous thrombosis can change the type and neurologic behavior of DAVFs (6). In terms of its likely pathogenesis, the lesion in case 1 may be classified as a DAVF of the parasinusal cortical veins (3), as there was no reliable imaging documentation of an associated phlebothrombosis, and the superior sagittal sinus appeared patent. Such fistulas of the cortical veins situated close to a patent dural sinus, particularly near the superior sagittal sinus, are thought to develop after phlebothrombosis in the region of the intrasinus venous orifice

of a cortical vein, assuming that the connection to the sinus lumen remains occluded (7). Therefore, they drain primarily into pial cortical veins and show an associated thrombosis on imaging studies only in those rare cases in which the thrombosis also involves the sinus lumen.

The DAVF in case 2 was located in the region of the transverse-sigmoid junction, but the venous drainage was only via one venous recipient into the sinus lumen. The venous conduit corresponded to the inner part of a mastoid emissary vein. In fact, this lesion was located in the dura around an emissary vein, and may be classified as a DAVF of the venous plexus at the base of the skull (3). In such cases, DAVFs develop after thrombosis in the emissary veins and drain into the sinus through the inside channel of the vein, assuming that the outside channel remains occluded (7).

From a pathophysiological point of view, the condition is a venous disease, and only after occlusion of the venous side will all arterial input (ie, venous arterialization) cease permanently. The therapeutic strategy is chosen to improve symptoms and/or to prevent catastrophic consequences of the natural history of the DAVFs (6). Transarterial embolization may significantly decrease flow through the fistula but is not likely to result in complete anatomic obliteration in cases in which there are several feeders, including numerous minute twigs of major arterial trunks. Cyanoacrylates (*N*-butyl cyanoacrylate, Histoacryl) are able to permeate very small vessels of the nidal area, and their polymerization can be prolonged by the addition of iophendylate (Pantopaque, iodized oil). However, in cases of tortuosity or very long feeding vessels (as in case 1), embolization with acrylic agents can result in proximal deposition of the embolic material, and their effectiveness will be substantially reduced. The nonaggressive Ethibloc, when mixed with iodized oil, has a very retarded precipitation (over 5 seconds) and is

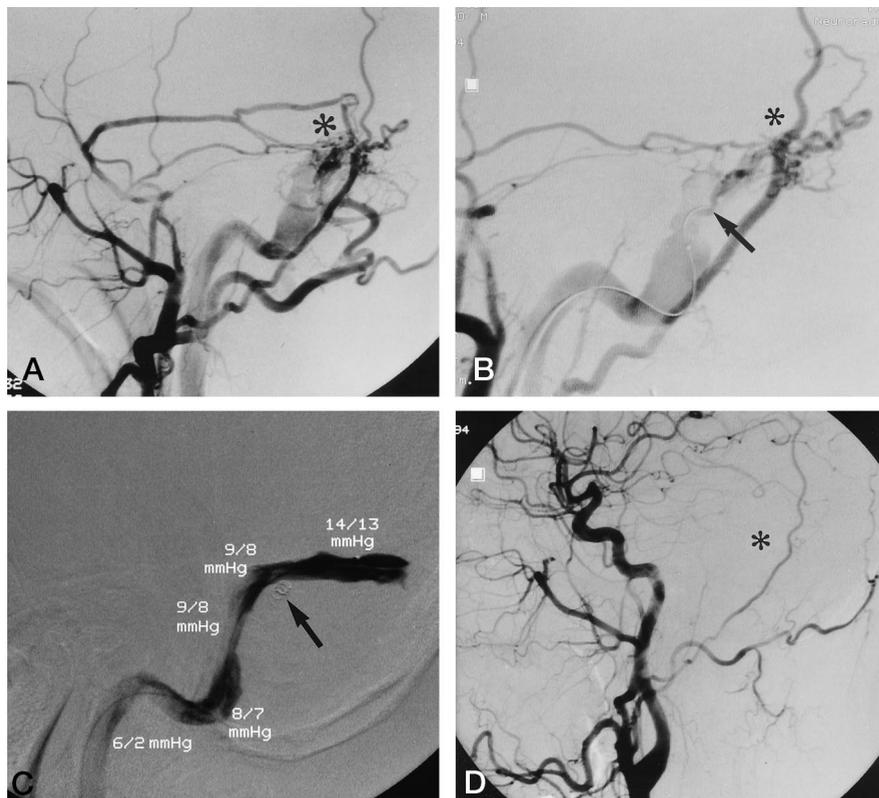


FIG 2. Case 2: 45-year-old man with a 2-year history of a right ear pulse-synchronous bruit and headache.

A, External carotid angiogram shows the DAVF at the right transverse-sigmoid junction (*asterisk*), supplied by the middle meningeal, auricular posterior, and occipital arteries.

B, Internal maxillary artery injection after embolization of both the mastoid occipital artery and the posterior branch of the occipital artery and the posterior branch of the middle meningeal artery shows the same DAVF (*asterisk*). The remaining supply is by the posterior auricular artery and both the petrous and the temporal convexity branches of the middle meningeal artery. Note the tip of the catheter, placed simultaneously and transvenously in the venous recipient (*arrow*).

C, Phlebogram of the sigmoid sinus after transvenous embolization shows two complex-shaped fibered platinum coils (2 mm in diameter 10 mm in length) positioned in the short recipient of the DAVF, outside the sinus (*arrow*). The initially narrowed sigmoid sinus was not occluded, and there was no damage to the occipitotemporal veins. The venous pressure measurement in the sigmoid sinus fell to 9/8 mm Hg.

D, Angiogram of the common carotid artery at 1-month follow-up shows complete obliteration of the DAVF (*asterisk*).

capable of penetrating far distally to access very long feeding vessels. However, in cases in which there are several feeders (as in case 2), this treatment is rarely curative, because not all arterial feeders are accessible for safe transarterial embolization.

The transvenous route, on the other hand, allows access to the dural venous channels adjacent to the nidus of a DAVF, initially by an intraoperative approach (8) and then by transfemoral venous catheterization (1, 9, 10). Obliteration with induced thrombosis within the venous circulation may effect sufficient anatomic cure in 55% of DAVFs so treated (10). However, the most significant limitation of transvenous embolization may be attributed to the unique hemodynamic pattern of the venous dural sinuses themselves. In rare cases, undesirable hemodynamic alterations have occurred after transvenous occlusion of a dural sinus, resulting in venous infarction or hemorrhage, or in diversion of arterialized blood to the cortical venous drainage (10). In fact, placement of embolic agents into a fistulous segment of sinus that is being used as a conduit for venous drainage of the remaining brain carries a high risk of venous infarction or hemorrhage.

Conclusions

Selective transvenous embolization in our two patients resulted in complete and permanent obliteration of the lesions without damage to the dural sinus. In the appropriate setting, following careful analysis of venous morphology and drainage in adjacent brain parenchyma, this technique can produce safe obliteration of the venous channel of DAVFs without undesirable hemodynamic alterations.

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