Selective transvenous liquid embolization of a Type 1 dural arteriovenous fistula at the junction of the transverse and sigmoid sinuses

Case report

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The authors describe the case of a 51-year-old man with a Type 1 dural arteriovenous fistula (AVF) located at the junction of the transverse and sigmoid sinuses. The dural AVF developed after the patient underwent an intracranial hematoma. The patient experienced pulsatile tinnitus 3 months after surgery. After several attempts at transarterial embolization (TAE), the venous channel located close to the skull fracture was accessed via a transfemoral–transvenous approach and was embolized by administering a liquid nonadhesive agent. Successful embolization of the dural AVF was achieved both clinically and radiologically without causing considerable hemodynamic alterations. This procedure, either alone or combined with TAE, would seem to be an alternative treatment for dural AVFs in this location, without causing compromise of flow within the affected sinuses, when selective venous access is available.

Key Words • dural arteriovenous fistula • head injury • embolization • transverse sinus • sigmoid sinus

The transverse and sigmoid sinuses are the most common locations for symptomatic intracranial dural AVFs. Debate continues regarding the etiopathogenesis of these lesions. Although several congenital dural AVFs have been reported, many now agree on the acquired nature of these fistulas. The prevailing belief is that dural AVFs develop after compromise of the venous sinus, such as that caused by sinus thrombosis or tumor invasion. Some dural AVFs in this location, however, have been reported to occur in patients with a history of trauma. Dural AVFs have been managed in a variety of ways including clinical observation, manual carotid compression, endovascular therapy, radiosurgery, and/or direct surgery. Endovascular techniques have been widely used as a first treatment, via either a transarterial or transvenous route.

We describe the case of a Type 1 dural AVF located at the junction of the transverse and sigmoid sinuses, which developed in a patient who underwent a craniotomy for a traumatic intracranial hematoma. In this case, after several sessions of TAE, the single venous channel was accessed via a transfemoral retrograde approach and was embolized by administering a liquid nonadhesive agent. Successful fistula embolization was documented both clinically and radiologically without undesirable hemodynamic alterations. To our knowledge, this selective transvenous embolization performed using a liquid nonadhesive agent has not been previously described in the literature.

Case Report

History. This 51-year-old man fell from the second floor of a construction site and was struck on the right parietal region. The patient was immediately referred to our institution with the diagnosis of an acute extradural hematoma (Fig. 1 left). The hematoma was evacuated via a right temporoparietooccipital craniotomy. The patient experienced an uneventful postoperative course and seemed fully recovered except for hyposmia. On postoperative T2-weighted MR imaging, the patient’s right temporal lobe displayed a high intensity (Fig. 1 center). Three-dimen-
sional CT scanning clearly revealed a linear fracture crossing the right corner of the transverse and sigmoid sinuses (Fig. 1 right). Three months after surgery, the patient suffered right pulsatile tinnitus with subsequent worsening, leading to sleeplessness, and was readmitted to our institution.

**Examination.** Neurological examination revealed hyposmia and a pulse-synchronous bruit in the right retroauricular region. Initial cerebral angiography demonstrated a dural AVF fed by numerous small pedicles from the ipsilateral ECA, ICA, and VA (Fig. 2). The dural AVF drained antegrade into the junction of the right transverse and sigmoid sinuses (Fig. 2). This drainage pattern was consistent with that of a Type 1 dural AVF according to the classification of Djindjian and Merland,10 as modified by Cognard, et al.6 Single-photon emission CT scanning revealed a diminution of cerebral blood flow in the patient’s right posterior temporal region.

**Attempts at TAE.** Embolization of numerous branches from the right ECA was performed using polyvinyl alcohol particles, platinum microcoils, and/or liquid embolic materials. We cannulated as many feeding branches arising from the right OA, middle meningeal artery, superficial temporal artery, and PAA as possible by using guide-wire-directed microcatheters and then embolized them. Several embolization sessions resulted in a significant decrease in the patient’s bruit and the arteriovenous shunt, but complete obliteration of the dural AVF was not accomplished because of recanalization and recollateralization of the feeding arteries. After careful consideration of available treatment options, a selective TVE was conducted 8 months after the initial diagnosis.

**Selective TVE Using a Liquid Nonadhesive Agent.** After local anesthesia had been induced in the patient, a No. 6 French long vascular sheath was placed in his right common femoral vein. The patient was intravenously administered heparin: initially 3000 U followed by 800 U/hour. For purposes of roadmapping the venous channel, a No. 5 French catheter was inserted into the right ICA. For venous access, a No. 6 French guiding catheter was advanced to the level of the right jugular bulb. A microcatheter (FasTracker 18MX; Target Therapeutics, Fremont, CA) was inserted in a coaxial fashion through the guiding catheter and, using a roadmapping injection of contrast agent from the arterial side, the microcatheter was advanced over a 0.014-in guidewire (Transcend; Meditech, Watertown, MA) through the sigmoid sinus and wedged into the venous channel. At that time, selective injection of contrast agent into the venous channel demonstrated only retrograde filling of the venous channel and the adjacent dural veins (Fig. 3). For this reason, the venous channel was embolized using 0.05 ml of a 15% concentration of a nonadhesive liquid embolic material (Eudragit; Rohm Chemische Fabrik, Darmstadt, Germany). On completion of the embolization, both right CCA and VA injections of contrast material demonstrated no residual filling of the dural AVF and preservation of flow within the affected sinus. The patient’s bruit disappeared immediately. No complications occurred during or after the procedure.

**Postembolization Course.** Follow-up angiograms obtained 3 months after TVE demonstrated complete obliteration of the dural AVF with preservation of flow within the affected sinuses (Fig. 4). The patient showed no clinical evidence of recurrent dural AVF during a 19-month follow-up period.

**Discussion**

**Etiopathogenesis of the Dural AVF**

In the present case, the arterial supply to the dural AVF came from numerous small pedicles from the scalp, meningeal, and cortical arteries, whereas venous egress was only provided through a single distinct channel within the wall of the transverse–sigmoid sinus junction.4 Additionally, the venous channel was located close to the skull fracture. The presence of a pial arterial supply to traumatic dural AVFs in this location is extremely rare.2,15,30 The patient’s previous head injury and subsequent surgical
procedure likely played a considerable role in the development and progression of this dural AVF.

Skull fracture can cause damage both to meningeal vessels and to the wall of the venous sinus, leading to an abnormal communication between meningeal arteries and the sinus. Cerebral contusion can cause a fibroglial scar between the damaged brain and the overlying dura, which leads to pial arterial inputs to the dural AVF due to angiogenesis and/or reopening of arterial “retia mirabilia” (transdural anastomoses). In addition, following a craniotomy some dural A VFs arise adjacent to or remote from the fistula site. Conceivably, the surgical procedure performed in the extradural space might cause damage to the meningeal vessels or subclinical infection. Repositioning of vessels of the scalp or muscles might also facilitate angiogenesis. Previous surgery might act as a stimulus for arterial recruitment to the fistula. In contrast, in the present case the patient had shown no evidence of venous sinus thrombosis. Once dural AVFs have occurred, resultant venous hypertension may foster the growth of microscopic arteriovenous shunts within the vasa vasorum of the normal pachymeninges and may also stimulate the release of angiogenic factors.

The pathogenesis of the present dural AVF was likely related to multiple predisposing factors: head trauma, subsequent craniotomy, subclinical infection, and venous hypertension. The numerous arterial supplies to this AVF might reflect these factors.

Treatment of Dural AVFs

Classifications of dural AVFs involving the transverse and sigmoid sinuses have been based on patterns of venous drainage. Dural AVFs with leptomeningeal retrograde venous drainage have been recognized as the lesions with the greatest propensity to manifest aggressive clinical symptoms, thus mandating prompt treatment. In contrast, Type 1 dural AVFs are generally considered to display benign behavior. Consequently, the aggressiveness of the treatment needs to be tailored to the importance of the symptoms and to the angioarchitecture. It was decided to proceed with treatment for the present dural AVF, because the patient wished to be relieved of his intolerable tinnitus and because he harbored a considerably high-flow dural AVF fed by numerous pedicles from the scalp, meningeal, and cortical arteries. Effective and less invasive treatment is demanded for such a low-risk dural AVF.

Endovascular techniques are now frequently used as the first treatment for most dural AVFs. Transarterial embolization can decrease the magnitude of the arteriovenous shunt and alleviate symptoms; however, it is generally recognized as an incomplete treatment. Despite initial
transfemoral approaches have been well described.\textsuperscript{9,11,15,17} Selective TVE by decreasing the flow of the shunt and, thereby, promoting thrombosis in the venous channel. Furthermore, selective angiography of each feeding artery allows us to clarify the vascular architecture, particularly the location and contour of venous egress.

Mullan\textsuperscript{28} has argued that the principle of treatment for dural AVFs should be interruption of venous egress of the fistula. Recently, many have advocated a primary transfemoral approach to deal with dural AVFs of the transverse–sigmoid sinus, by using coil packing or balloon occlusion of the affected sinus. Both surgical and percutaneous transfemoral approaches have been well described.\textsuperscript{9,11,15,17} The use of the balloon occlusion test has been recommended when contemplating endovascular occlusion of an involved sinus that is freely communicating with normal venous structures.\textsuperscript{33,39} In contradistinction to the cavernous sinus, which can be occluded with little adverse effect, the occlusion of the transverse or sigmoid sinus carries a potential risk of venous infarction and labyrinthine dysfunction due to hydrops of the endolymphatic sac.\textsuperscript{33,39} There is also a risk of changing the form of venous drainage of the dural AVF from a benign one to a more hazardous one with leptomeningeal vein involvement. The best candidates for venous sinus embolization are cases in which the involved sinus is already compromised and no longer contributes to the drainage of normal tissue.\textsuperscript{33,39} In the present case, the affected sinuses were on the dominant side for venous return and flow was not compromised.

Recently, Mironov\textsuperscript{26} reported on a selective TVE performed using platinum fiber coils for a Type 1 dural AVF located at the junction of the transverse and sigmoid sinuses. That procedure resulted in complete obliteration of the fistula without causing damage to the sinus. Transvenous coil embolization of Type 4 dural AVFs at the tentorial\textsuperscript{22} and superior sagittal sinus\textsuperscript{3} has also been performed with preservation of flow within the sinuses. After careful analysis of the vascular structure in the present case, the venous channel could be selectively accessed via a transfemoral–transvenous approach. We used a cationic polymer, Eudragit E mixture,\textsuperscript{41} as the embolic agent for selective TVE, achieving a satisfactory result. Eudragit E, developed by Yamashita, et al.\textsuperscript{41} is a mixture of methyl and butyl methacrylate, together with dimethylaminomethyl methacrylate copolymer, in a solvent consisting of ethanol and iopamidol. On contact with aqueous substances, it precipitates rapidly and forms a soft elastic sponge within 3 seconds as the ethanol diffuses. In blood, the positively charged Eudragit E aggregates the negatively charged blood elements. Its viscosity is the lowest of the precipitating materials.

The successful obliteration of the dural AVF obtained in this case might also have been related to the narrow lumen of the venous egress, which allowed a liquid nonadhesive agent to thrombose the venous channel without reflux into the sinus. When contemplating occlusion of a dilated venous channel, the use of coils combined with a liquid embolic agent may be advised.

Using modern microcatheters and guidewires, as well as careful angiographic studies with road mapping, this procedure can provide safe obliteration of the venous conduit of dural AVFs without causing undesirable hemodynamic alterations in the affected venous systems.

\textbf{References}

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