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Authors

Alexander, Matthew David
Halbach, Van
Nicholson, Andrew
et al.

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Republished: Transvenous ethanol sclerotherapy of feeding arteries for treatment of a dural arteriovenous fistula

Matthew David Alexander, **Van Halbach**, **Andrew Nicholson**, **Fabio Settecase**, **Robert J Darflinger**, **Matthew R Amans**

Department of Radiology and Biomedical Imaging, University of California San Francisco, San Francisco, California, USA

Abstract

Dural arteriovenous fistulas (DAVFs) can be complex lesions that require a well trained eye for proper characterization and management decisions. With numerous possible arteries to supply and veins to drain them, DAVFs are often complex lesions. To best treat these complex lesions, the neurointerventionalist should be adept at treating them with multiple techniques. This report describes a unique case in which a DAVF was treated with arterial sclerotherapy using ethanol from a transvenous approach.

BACKGROUND

Dural arteriovenous fistulas (DAVFs) are among the most challenging lesions treated by neurointerventional radiologists, and all treatment options should be at the disposal of a well equipped physician. Ethanol is a potent therapeutic that can lead to outstanding treatment results, but it is not used by many practitioners. This report describes a unique opportunity for the treatment of a DAVF with ethanol that has not previously been described.

CASE PRESENTATION

A 64-year-old man presented with 9 years of right pulsatile tinnitus (PT). He suffered traumatic brain injury in a motor vehicle collision 1 year prior to the onset of PT. Over subsequent years he developed progressive cognitive decline and headaches. MRI demonstrated extensive leptomeningeal and pachymeningeal enhancement that was initially interpreted at an outside facility as meningitis versus leptomeningeal carcinomatosis (figure 1). Upon transfer of care to our institution, further inquiry into his situation led to greater suspicion of venous congestion, most likely from a DAVF. Diagnostic cerebral angiography was pursued for further evaluation.

Correspondence to: Dr M D Alexander, Department of Radiology and Biomedical Imaging, University of California San Francisco, 505 Parnassus Ave, San Francisco, CA 94143, USA; matthew. alexander@ucsf.edu.

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TREATMENT

Angiography demonstrated an extensive high risk DAVF involving the right transverse and sigmoid sinuses and downstream occlusion of the left sigmoid sinus. Extensive cortical venous drainage was noted, as well as extensive redirection of flow to the cavernous sinus, where stagnation was noted (figure 2). Treatment was performed with ethylene vinyl alcohol copolymer (EVOH) embolization through the anterior division of the right middle meningeal artery in two stages. This accomplished a marked reduction in shunt flow with no further stagnation in the cavernous sinus or cortical drainage, consistent with conversion from a high risk to a low risk fistula (figure 3).

Following treatment, he reported dramatic improvement in symptoms, but right PT recurred 13 months later. Diagnostic angiography demonstrated residual low risk DAVF. The shunt was localized to the right sigmoid sinus, and drainage was noted in a parallel channel that was accessed with a microcatheter. The arterial supply was noted from the jugular division of the neuromeningeal trunk of the ascending pharyngeal artery, the meningohypophyseal trunk, and the transmastoid perforators of the right occipital artery (figure 4). Concern for inducing cranial neuropathy, particularly in CNN IX-II, with transarterial embolization led to the decision to perform transvenous treatment. Upon injection of the microcatheter guided to the parallel channel via the transfemoral transvenous route, it was noted to be adjacent to the fistula site, and the very distal arterial feeders were opacified with contrast (figure 4). Careful ethanol infusion was performed from this site to sclerose the feeding arteries as they enter the fistula site. Ethanol was titrated to minimal penetration of the most distal afferents to preserve vasonervosa. Post-treatment selective injection of the venous parallel channel demonstrated no residual arterial filling across the fistula (figure 4). Transvenous coiling of the parallel channel was then performed. Post-treatment angio-gram demonstrated no residual DAVF (figure 5).

OUTCOME AND FOLLOW-UP

PT was no longer present after the procedure, and the patient has remained symptom free for nearly a year after the curative treatment.

DISCUSSION

DAVFs can be treated in multiple ways, and treatment is best tailored to the territories and vessels involved.[1–4] Similarly, the risk profile of the considered treatment should be tailored to the risk of the underlying disease. When treating a low risk lesion, the treatment should be low risk. Treatment can be carried out from both transarterial and transvenous approaches using coils, EVOH, n-butyl cyanoacrylate, ethanol, polyvinyl alcohol particles, or a combination of these agents.[1–4]

Ethanol has an extensive history of treatment for vascular lesions, including intracranial lesions with arteriovenous shunting.[24–6] Intravascular administration causes protein denaturation in endothelium that leads to subsequent vessel wall denuding and vessel thrombosis.[7] The effectiveness of ethanol derives from its potent activity, and it must be used with caution because of possible side effects and complications. A primary

consideration is the painful response to treatment.[6] General endotracheal anesthesia is requisite, with deep sedation and paralysis to prevent patient motion during catheterization of small delicate vessels. Additionally, anesthesia colleagues should maintain a high index of suspicion for bronchospasm and cardiovascular collapse, the most common and potentially dangerous complications in neurointerventional procedures.[8,9] Other adverse reactions to ethanol, such as hemolysis, alterations in anesthetic agent activity, and skin necrosis, are more common with higher doses used in treatments outside the CNS.[8,10–12] Additionally, specific concerns should be considered depending on end organs perfused by vessels treated, such as diabetes insipidus occurring following ethanol sclerotherapy of the meningohypophyseal trunk.[4,13]

In the treatment of certain DAVFs, ethanol can be the most effective treatment modality. Most commonly, this is true when the arterial supply is a very extensive network of small caliber vessels fed from a larger single pedicle, such as in transverse–sigmoid lesions fed by transmastoid perforator branches of the occipital artery. In our practice, we dilute dehydrated ethanol (American Regent Inc, Shirley, New York, USA) in iodinated contrast to 60% strength. Injection is performed with a 1 mL syringe under blank roadmap technique. Injection is performed with the aim of maximizing contact of the agent with endothelial cells by injecting sufficiently to maintain the appearance of a static column of the agent in the target without reflux. An advantage of ethanol is that it dilutes to an inconsequential dose when entering vessels other than the small target, either due to outflow from the lesion or from reflux. This is a distinct advantage over Onyx, with which non-target embolization cannot be reversed. Injection is performed until the static column appearance can no longer be achieved, which typically requires 1–2 mL for a pedicle suitable to this treatment. After a 15 min delay, angiography is performed to evaluate progress. Sluggish flow will often progress to cure, but more robust flow is treated further with the same techniques. This is a distinct advantage over n-butyl cyanoacrylate, which can be injected only one time in a single pedicle.

The patient described above had involvement of small vessels amenable to ethanol treatment. However, treatment is traditionally performed with the microcatheter position in the larger parent artery that divides into the targeted network of arteries. Due to very superselective positioning of the microcatheter in the recipient venous parallel channel at the location of the fistula site, the feeding arteries could be reached with ethanol from this position by refluxing across the fistula. This ensured targeting of the distal most segments of the arteries adjacent to the fistula, which maximized the effects of the sclerotherapy. Visualization of the embolic material by mixing with contrast is imperative to allow for careful titration of injection pressure. Over injecting the ethanol may result in penetration of the vasonervosa and could result in permanent cranial nerve palsy. Test injections with contrast diluted by saline can help to tune the interventionalist’s hand to the pressure required to transgress the fistula site and minimize penetration into the more proximal arteries. Treatment with ethanol also requires a ‘tincture of time’ to demonstrate the maximal effects of the ethanol on the endothelium. Our practice routinely involves administration of small doses of ethanol followed by angiography after 15–20 min to see the initial effects of the sclerotherapy. With further treatment, it is important to again titrate the

pressure of the treatment injections with the changes in the hemodynamics induced by sclerotherapy.

While efficacious, ethanol treatment is typically best suited in conjunction with other treatment modalities in additional vessels involved in these complex lesions.[124] Such a strategy was employed in the above described treatment, with coil embolization of the parallel venous channel performed following treatment with ethanol. Thrombosis from coil embolization was promoted by the reduced flow into the lesion following sclerotherapy. While scenarios suitable for such transvenous ethanol sclerotherapy of arterial structures will likely be rare, we believe such treatment can be useful in the proper setting and should be considered by neurointerventionalists treating DAVFs.

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Key Messages

- Multiple options exist for treatment of dural arteriovenous fistulas (DAVFs), and treatment should be tailored to each individual lesion.
- Ethanol has achieved excellent results for the endovascular treatment of arteriovenous shunting lesions, including DAVFs.
- While effective, a healthy respect for potential side effects of ethanol treatment is required.
- Ethanol treatment of DAVFs requires very selective positioning, which can be achieved from a transvenous approach.

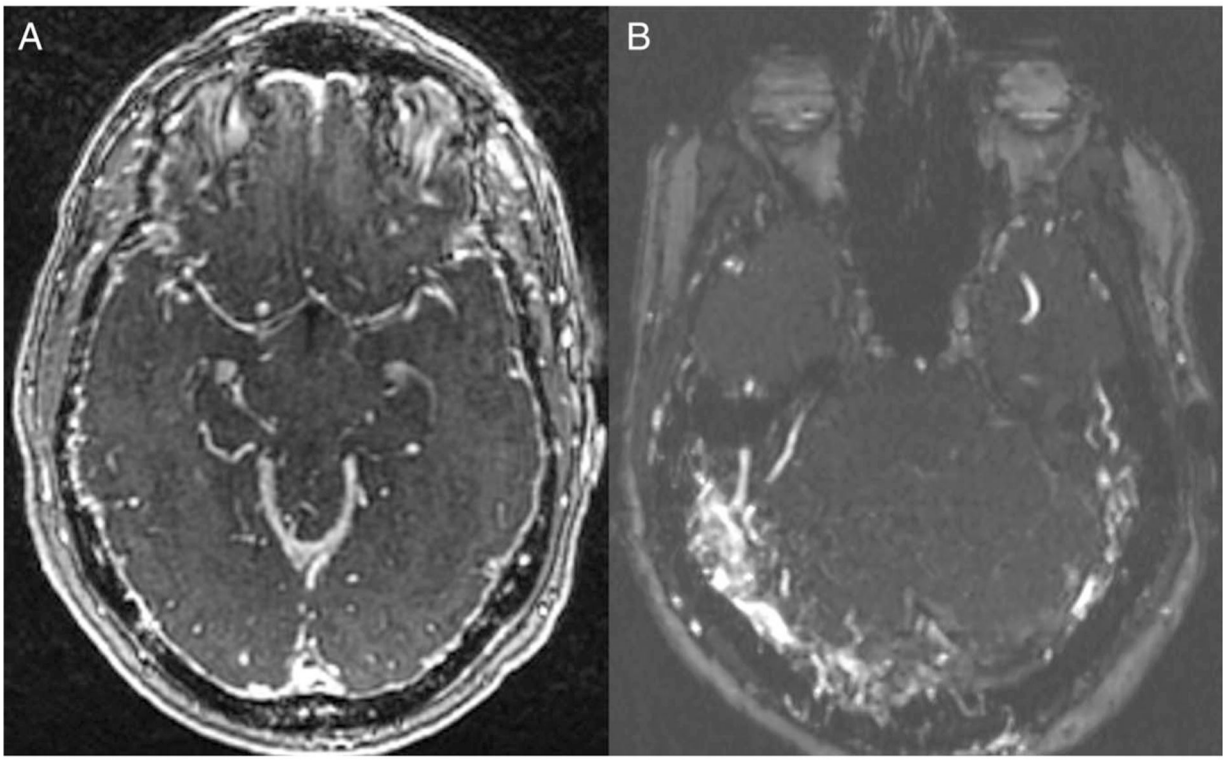

Figure 1.

(A, B) Contrast enhanced T1 weighted MRI images demonstrate extensive leptomeningeal and pachymeningeal thickening and enhancement. There is abnormal prominently asymmetric enhancement of the right transverse–sigmoid sinus with numerous adjacent serpiginous vessels (B). Additionally, there is no flow in the medial left transverse sinus suggestive of occlusion.

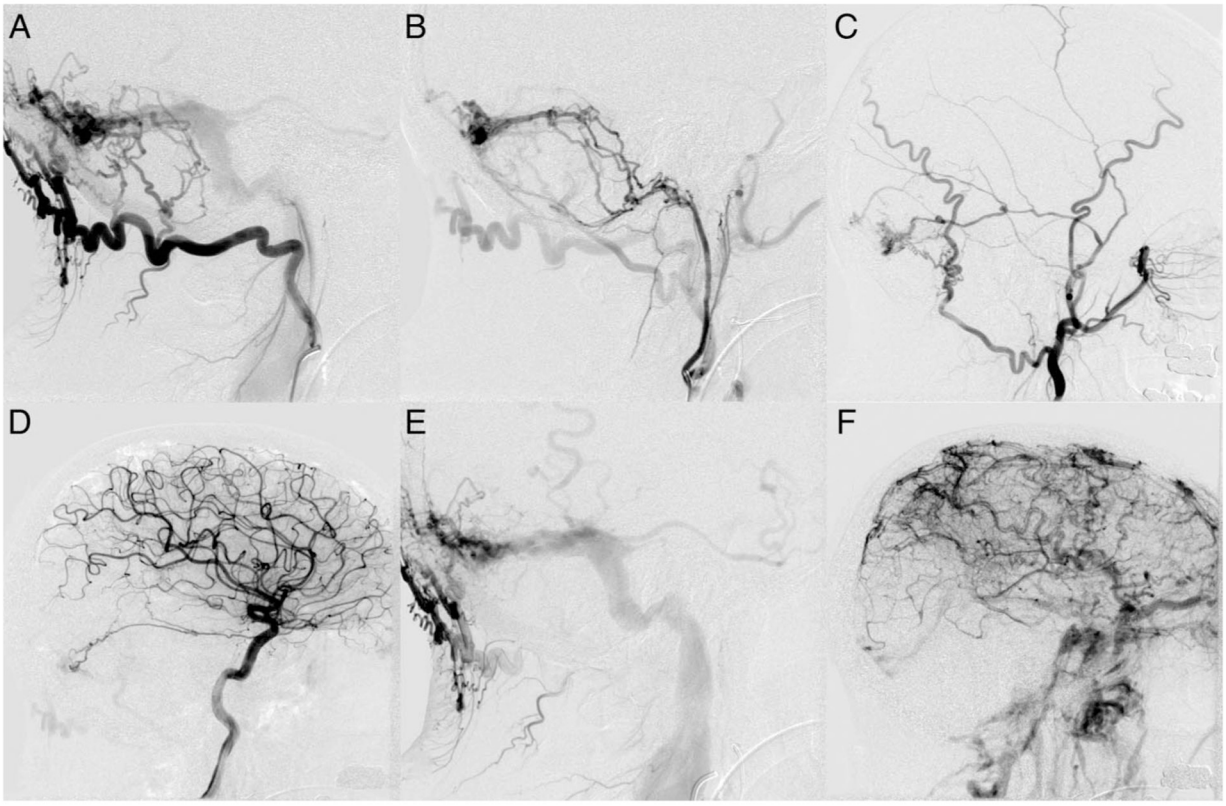

Figure 2.

Digital subtraction angiography demonstrates a dural arteriovenous fistula with multiple feeding arteries, including transmastoid perforator branches during injection of the right occipital artery (A), branches of the neuromeningeal trunk during injection of the ascending pharyngeal artery (B), and branches of the posterior division of the middle meningeal artery and posterior auricular artery during injection of the proximal internal maxillary artery (C). Selective injection of the right internal carotid artery (D) demonstrates supply to the fistula from the meningohypophyseal trunk. Early venous phase imaging during injection of the right occipital artery (E) demonstrates arteriovenous shunting with early venous drainage into the right transverse–sigmoid sinus with reflux into multiple cortical veins and the cavernous sinus. Stagnation was noted in the cavernous sinus (not pictured). Late venous phase imaging following selective injection of the right internal carotid artery (F) demonstrated no outflow into the transverse and sigmoid sinuses, indicating that brain parenchyma is not using these structures for venous drainage. There is pronounced sluggish outflow in the right cavernous sinus and prominent inferior petrosal and pterygoid sinuses draining into irregular appearing veins. There is markedly delayed outflow in markedly enlarged and tortuous phlebectatic cortical veins.

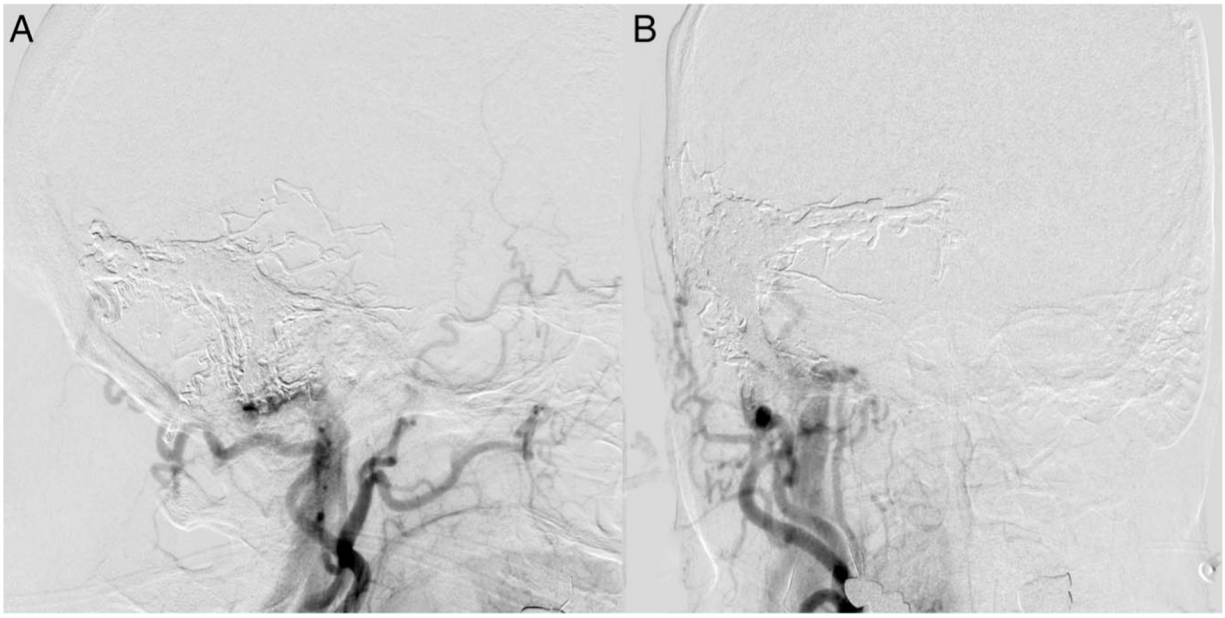

Figure 3.

Lateral (A) and Townes (B) projections during injection of the right external carotid artery following two stage ethylene vinyl alcohol copolymer embolization of two different branches of the right middle meningeal artery. Persistent venous drainage is demonstrated in a parallel venous channel of the right sigmoid sinus with the arterial supply from the jugular division of the neuromeningeal trunk and transmastoid perforator branches of the right occipital artery. No residual cortical venous reflux is seen, consistent with conversion to a low risk dural arteriovenous fistula.

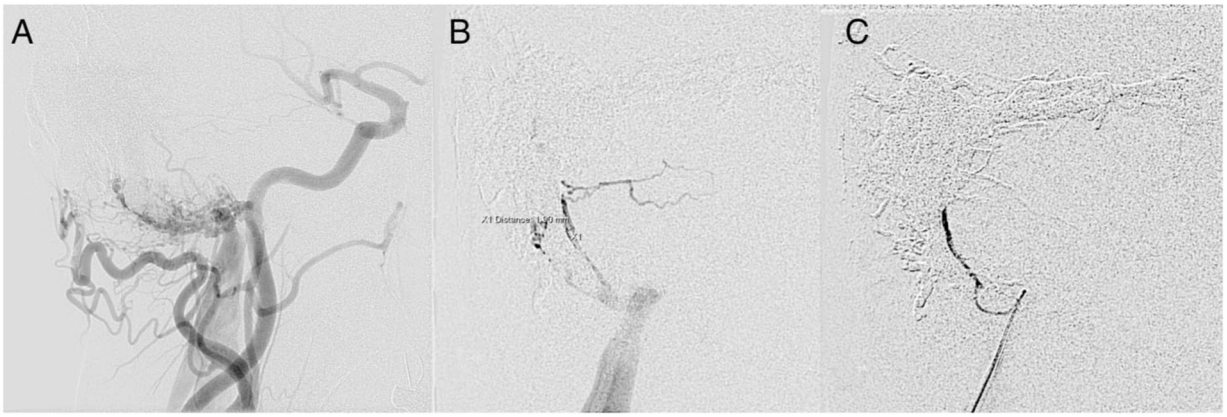

Figure 4.

Selective injection of the right external carotid artery (A) following recurrence of pulsatile tinnitus 13 months after treatment demonstrates persistent venous drainage into a parallel venous channel. Venography following superselective catheterization of the parallel channel (B) demonstrates reflux of contrast across the fistula into feeding arteries. Selective injection of the microcatheter in the parallel channel following ethanol sclerotherapy from this position (C) demonstrates no residual reflux across the fistula.

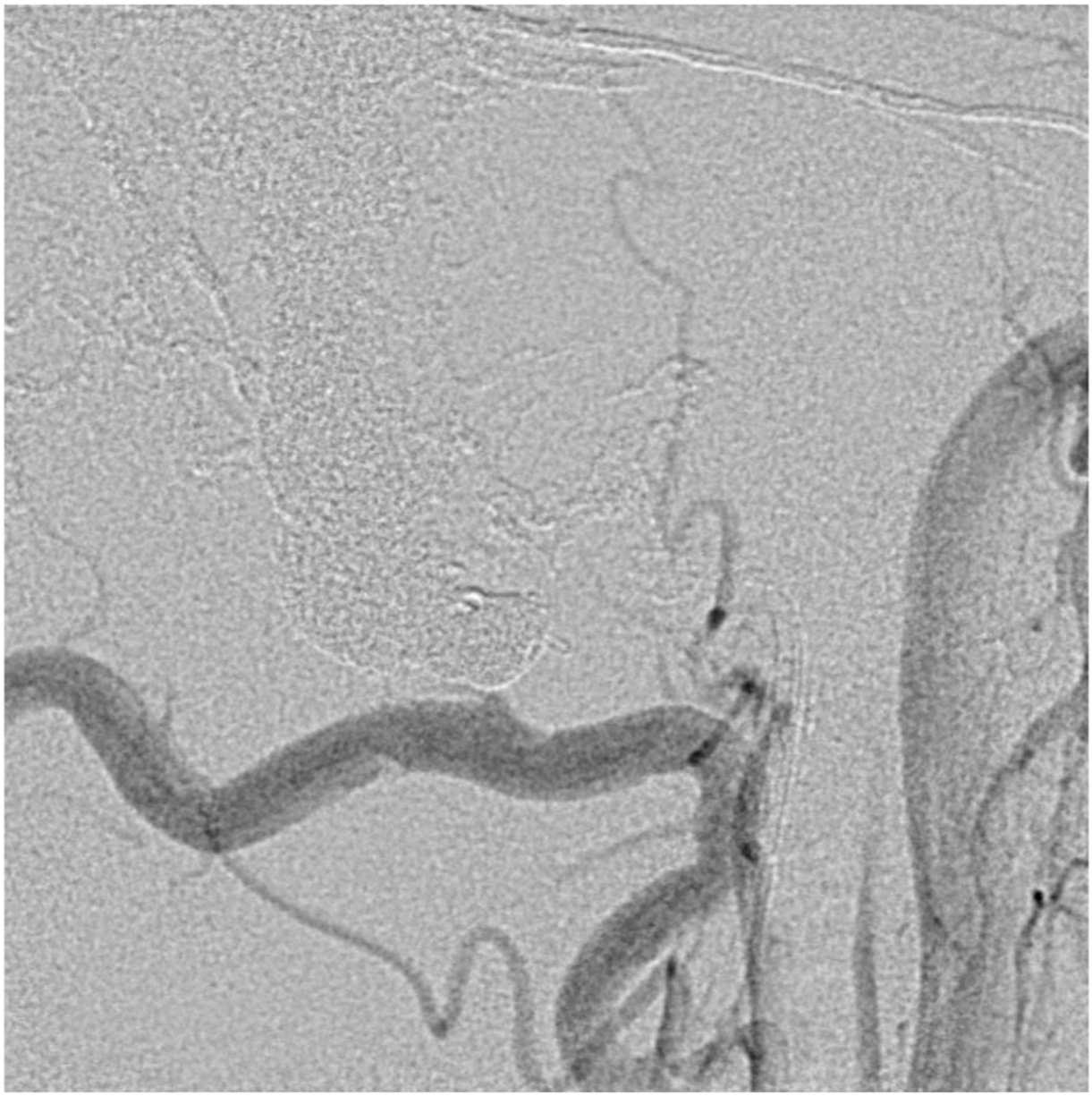

Figure 5.
Post-treatment selective injection of the right external carotid artery following transvenous coiling of the parallel channel demonstrates no residual arteriovenous shunting, consistent with cure of the dural arteriovenous fistula.