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Dural Arteriovenous Fistula Associated With Meningioma: Spontaneous Disappearance After Tumor Removal

—Case Report—

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Abstract

A 45-year-old woman presented with a non-dominant transverse-sigmoid sinus dural arteriovenous fistula (AVF) associated with convexity meningioma on the same side. The dural AVF disappeared spontaneously after surgical removal of the meningioma, even though there was no manipulation of the dural AVF. Dural AVFs are usually acquired lesions, and may develop after trauma, surgery, and dural sinus thrombosis. Dural AVFs of the acquired origins are rarely associated with brain tumor. Dural AVFs associated with a tumor may develop even in the absence of sinus occlusion.

Key words: dural arteriovenous fistula, sinus thrombosis, meningioma, pathophysiology

Introduction

Dural arteriovenous fistulas (AVFs) are defined as arteriovenous shunts of blood confined within dural leaflets. The cause and pathogenesis of dural AVFs remain unclear. Most dural AVFs are considered to be acquired,[5,10,19] although some dural AVFs in infancy may be congenital.[14] Trauma, surgery, sinus thrombosis, and other factors often initiate the formation of dural AVFs.[2] Dural AVFs are associated with various types of flow compromise in the transverse/sigmoid sinus, such as thrombosis, trauma (cranial fracture, craniotomy), infection, previous tumor resection in the area, a hypercoagulable state, pregnancy, hormonal disease, the rupture of an aneurysm, and arterial dysplasia.[2,4,5,7,9,10,13,16,19] Tumors causing occlusion of the major sinuses are rarely associated with dural AVFs, suggesting that the induced sinus occlusion may be the cause of the dural AVF.[1,6,20–22] The most common tumor is meningioma.[1,6,18,19,22] Dural AVFs without sinus occlusion are rarely reported.[3]

We recently encountered a case of transverse/sigmoid sinus dural AVF associated with meningioma that did not compromise the dural venous sinus, but which disappeared spontaneously after removal of the tumor.

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Case Report

A 45-year-old woman presented with a 4-month history of dull headache in the left side. Neurological examination at admission found no abnormalities. She had no history of head injury or cranial surgery. Preoperative magnetic resonance imaging with contrast medium revealed an enhanced extra-axial mass along the left parietal convexity (Fig. 1). Left external carotid angiography showed a tumor blush that was fed by the anterior branch of the left middle meningeal artery, and a coexistent dural AVF which was fed by many branches of the ascending pharyngeal, occipital, and middle meningeal arteries and drained by the transverse sinus (Fig. 2A). The venous phase of the left internal carotid angiography revealed good patency of the sagittal and transverse sinuses except for the transverse-sigmoid sinus junction. The preliminary diagnosis was partial occlusion of the left sigmoid sinus. However, there was no evidence of venous congestion or retrograde venous drainage (Fig. 2B, C).

The anterior branch of the left middle meningeal

artery was selectively catheterized and embolized with polyvinylalcohol particles (150–250 μm) (Fig. 3A). Postembolization angiography showed no evidence of tumor staining at the left parietal area, but the dural AVF was still present near the transverse sinus (Fig. 3B). Under the impression of meningioma, the convexity tumor was resected via a posterior parietal craniotomy with no significant intraoperative hemorrhage. The histological diagnosis was transitional type meningioma. The patient was free from headache for one year postoperatively. Follow-up angiography revealed complete disappearance of the dural AVF (Fig. 4).

Fig. 1 Coronal T_1-weighted magnetic resonance image after intravenous injection of contrast medium revealing an enhanced extra-axial mass along the left parietal convexity with no relationship with the involvement of the sagittal sinus.

Discussion

Dural sinus obstruction and/or thrombosis induced by the tumor may cause the development of abnormal dural arteriovenous shunts. Involvement of the dominant sinus is an important factor in transverse/sigmoid sinus dural AVFs.[1)] Dural AVF associated with meningioma may have resulted from infiltration of the meningioma into the sinus wall causing accelerated involvement of the sinus and subsequent sigmoid sinus thrombosis, in addition to the direct compression by the meningioma.[22)] The downstream sinus obstruction may act as a trigger, changing the local hemodynamics and producing flow turbulence and/or venous hypertension.[20)] These hemodynamic changes may contribute to the development of dural AVFs.

Most cases of dural sinus obstruction or invasion by the tumor are not associated with dural AVF, so dural sinus obstruction or thrombosis cannot be the only reason for the presence of acquired dural AVFs. In our case, the dural sinus was intact and there was no definite proof of sinus infiltration of the tumor. Therefore, the coexistence with a meningioma may have been incidental. This concept is convincing when the two lesions are located at different sites as in our case. However, the meningioma without direct sinus obstruction may have

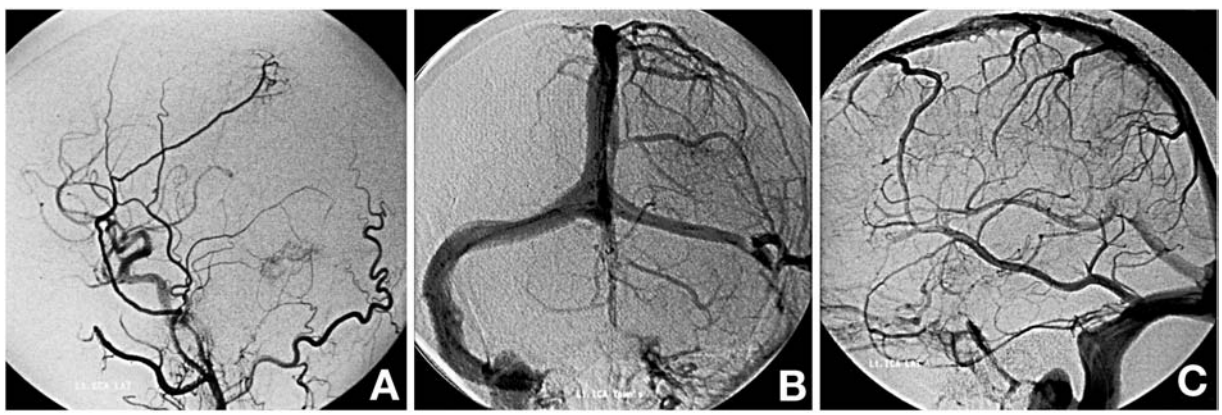

Fig. 2 (A) Left external carotid angiogram showing a tumor blush fed by the anterior branch of the left middle meningeal artery, and a dural arteriovenous fistula fed by many branches of the left ascending pharyngeal, occipital, and middle meningeal arteries and drained by the left transverse sinus. (B, C) Left internal carotid angiograms in the venous phase revealing good patency of the sagittal and transverse sinuses except for the transverse-sigmoid sinus junction, and partial occlusion of the left sigmoid sinus, but no evidence of venous congestion or retrograde venous drainage.

Fig. 3 (A) Selective angiogram of the tumor feeding branch of the left middle meningeal artery showing embolization with polyvinylalcohol particles (150–250 μm). (B) Left common carotid angiogram after the tumor embolization revealing no tumor staining, but persistent staining of the nidus of the dural arteriovenous fistula.

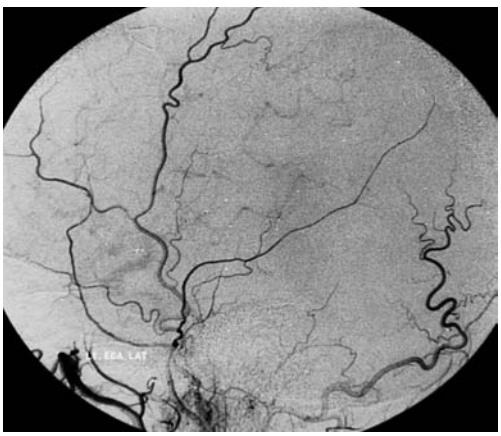

Fig. 4 Follow-up left external carotid angiogram at 12 months after removal of the tumor demonstrating completely spontaneous disappearance of the dural arteriovenous fistula.

been involved in the development of the dural AVF because the associated dural AVF in our case disappeared after tumor resection. The meningioma may have induced the dural AVF, or the meningioma may have occurred in the dural AVF. A meningioma-induced vascular malformation might be explained as an exceptional consequence of tumor-related angiogenesis, which is a complex process including diverse angiogenic factors such as growth factors or cytokines.[11,15] Expression of angiogenic stimulators and angiogenic activity are induced by tissue hypoxia and vascular endothelial growth factor is an example of such a stimulator.[18]

In addition to the controversy about the pathogenesis of dural AVFs, there are numerous additional questions related to lesion maturation and progression. Progressive recruitment of additional arterial feeders does not always occur in a predictable fashion or at a predictable rate. Many dural AVFs maintain a stable size and profile of arterial feeders during years of prospective follow up.[2] No factors predisposing to the spontaneous resolution of dural AVFs are known.[4,8] Spontaneous thrombosis may be involved in some cases, and thrombosis may occasionally extend into the adjacent dural sinus. Recanalization of the sinus may coincide with the spontaneous closure of the fistula.[12] Dural AVFs involving the cavernous sinus region are more likely to undergo spontaneous resolution, whereas spontaneous closure is less common in dural AVFs of other locations.[17] It is not known whether local hemodynamic or pathophysiologic phenomena peculiar to that location predispose such dural AVFs to spontaneous involution.[8] In our case, thrombosis of the dural AVF may have been triggered by the subtle hemodynamic change at the time of preoperative embolization of the feeding branch or at the time of surgical removal of the tumor. However, the anterior branches of the left middle meningeal artery were selectively catheterized and embolized. Drift of some particles originally delivered into the anterior branch of the middle meningeal artery into the posterior branch of the middle meningeal artery is less likely.

The present case of dural AVF associated with meningioma without sinus encroachment at a remote site showed spontaneously resolution after removal of the meningioma, even though there was no manipulation of the dural AVF. Our experience supports the hypothesis that dural AVFs are acquired and induced. However, this case might be a chance association because there was no sinus thrombosis or occlusion induced by the tumor.

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