Dural Arteriovenous Malformation Associated with Meningioma: Spontaneous Disappearance after Tumor Removal
- Case Report -

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Dural arteriovenous malformations may be congenital, but most dural arteriovenous malformations are acquired lesions. The acquired dural arteriovenous malformations are rarely associated with brain tumors. We describe a case of dural arteriovenous malformation at the non-dominant transverse-sigmoid sinus associated with a convexity meningioma on the same side. The lesion was spontaneously disappeared after removal of the meningioma, even though the dural arteriovenous malformation was not manipulated. The authors describe a possible pathophysiology of dural arteriovenous malformations associated with tumors at the remote area and spontaneous closure after tumor resection.

KEY WORDS : Dural arteriovenous malformation · Sinus thrombosis · Meningioma · Pathophysiology.

Introduction
Dural arteriovenous malformations (AVMs) consist of arteriovenous shunts of blood confined within the dural leaflets. The cause and pathogenesis of dural AVMs remain unclear. Many reports have noted that dural AVMs were associated with some degree of flow compromise in the transverse/sigmoid sinus, such as thrombosis, trauma (cranial fracture, craniotomy), infection, previous tumor resection in the area, hypercoagulable state, pregnancy, hormonal disease, rupture of an aneurysm, and arterial dysplasia2,4,5,7,9,10,12,14,17). In rare cases, tumors that occluded the major sinuses were associated with dural AVMs, suggesting that the sinus occlusion by the tumors may induce the development of dural AVM1,6,16,18,19). However, dural AVMs have also been reported without sinus occlusion3).

We recently encountered a case of transverse/sigmoid sinus dural AVM associated with meningioma that did not compromise of the dural venous sinus. That was disappeared spontaneously after removal of the tumor. We describe a plausible pathophysiological mechanism of dural AVM associated with a meningioma unrelated with sinus occlusion and its spontaneous closure.

Case Report
A 45-year-old woman visited with a 4-month history of dull headache in the left side. She was neurologically normal at the time of admission. Preoperative magnetic resonance imaging (MRI) revealed an enhancing, 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. In addition, a dural AVM was identified incidentally, which was fed by many branches of the left ascending pharyngeal, occipital, and middle meningeal arteries and drained into the left transverse sinus (Fig. 2A). Tumor feeding branches of the left middle meningeal artery were selectively catheterized and embolized with polyvinylalcohol (150 to 250μm in size). After the embolization, there was no tumor staining, but the staining of

Fig. 1. T1-weighted sagittal image of preoperative magnetic resonance imaging shows an enhancing, extra-axial mass along the left parietal convexity.
nidus of dural AVM was persistent (Fig. 2B). The convexity tumor unrelated with the major draining sinus was then resected. There was no severe dural bleeding at the dural opening. The histological diagnosis of the tumor was meningioma of transitional type. After the surgery, there is no tumor staining, but the staining of nidus of dural arteriovenous malformation was remained (Fig. 2B).

**Discussion**

Although some of the dural AVMs in infancy may be congenital lesions, the majority has been considered to be acquired. Trauma, surgery, sinus thrombosis, or other factors often initiate the formation of dural AVMs. The association between dural AVMs and intracranial tumors is rare. The most common tumor associated with dural AVMs is reported as a meningioma. A potential causative relationship between the dural sinus obstruction and/or thrombosis induced by the tumor and the development of abnormal dural arteriovenous shunts has been suggested in the previous reports. Arnaoutové et al. suggested that involvement of the dominant sinus was an important contributive factor to transverse/sigmoid sinuses dural AVMs. Yokota et al. reported a dural AVM associated with a meningioma and postulated that the infiltration of the meningioma into the sinus wall may accelerate the subsequent occurrence of sigmoid sinus thrombosis, in addition to the direct compression by the meningioma. Vilela et al. presumed that the downstream sinus obstruction may act as a trigger, changing the local hemodynamics and producing flow turbulence and/or venous hypertension. These hemodynamic changes may contribute to the development of dural AVMs. Most cases of dural sinus obstruction or invasion by the tumor are not associated with the dural AVM, however, dural sinus obstruction or thrombosis cannot alone explain the presence of acquired dural AVMs. Dural sinus was also intact and there was no definite proof of sinus infiltration of the tumor in our case. However, meningioma without sinus obstruction might be relevant to the development of dural AVMs because the associated dural AVM in our case was disappeared after tumor resection. We assume that meningioma might be related with role of aberrant angiogenesis in the pathogenesis of dural AVMs. Sawamura et al. presumed that 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.

In addition to the controversy about the pathogenesis of dural AVMs, there are still numerous questions related to the lesion maturation and progression. A progressive recruitment of additional arterial feeders does not always occur in a predictable fashion or at any predictable rates. Many dural AVMs maintain a stable size and profile of arterial feeders during years of prospective follow-up. Factors predisposing to spontaneous resolution of dural AVMs are not known. It is possible that spontaneous thrombosis may play a role in some cases, and thrombosis may occasionally extend into the adjacent dural sinus secondarily. The recanalization of the sinus coincided with the spontaneous closure of the malformation. Certain dural AVMs involving the cavernous sinus region are more likely to undergo spontaneous resolution, but spontaneous closure has been less frequently reported in dural AVMs of other locations. It is not known whether local hemodynamic or pathophysiologic phenomena peculiar to that location predispose such dural AVMs to spontaneous involution.

**Conclusions**

We experienced a dural AVM associated with a menin-
glioma without sinus encroachment at the remote site. The lesion was spontaneously disappeared after removal of the meningioma, even though the dural AVM was not manipulated. Our experience supports the hypothesis that dural AVMs are acquired and induced. However, this case might be fortuitous in association because there was no sinus thrombosis or occlusion induced by the tumor. We propose the possibility of a meningioma related aberrant angiogenesis, which is a complex process including diverse angiogenic factors.

References