

Tentorial Meningioma Encroaching the Transverse Sinuses and Sigmoid Sinus Junction Area Associated with Dural Arteriovenous Fistulous Malformation : A Case Report

A 62-year-old woman was evaluated for tinnitus and headache. Magnetic resonance imaging and angiography revealed the coexistence of a tentorial tumor encroaching the junction of the right transverse-sigmoid sinuses, and dural arteriovenous fistulous malformation (AVFM) of the right transverse sinus. AVFM was not manipulated at all during the surgery. The pathology was fibroblastic meningioma. Postoperatively, the dural AVFM completely disappeared on follow-up angiography. The fistulas were occluded also after surgery, even though there was no manipulation of the AVFM. It is suggested that the right dominant transverse-sigmoid sinuses are partially occluded by tentorial meningioma, developing the dural arteriovenous fistula of the right transverse sinus. An acquired origin of the dural AVFM was suggested in this case.

Key Words: Meningioma; Arteriovenous malformations

Yong Gu Chung, Ki Chan Lee, Hoon Kap Lee,
Nam Joon Lee*

Departments of Neurosurgery and Diagnostic
Radiology*, Korea University Hospital, College of
Medicine, Korea University, Seoul, Korea

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Address for correspondence

Yong-Gu Chung, M.D.
Department of Neurosurgery, Korea University
Hospital, 126, 5-ga, Anam-dong, Sungbuk-gu,
Seoul 136-705, Korea
Tel: +82.2-920-5696, Fax: +82.2-929-0629
E-mail: yongku@users.unitel.co.kr

INTRODUCTION

Dural arteriovenous malformations are vascular anomalies of the dura mater which are supplied exclusively by branches of the carotid or vertebral arteries before they penetrate the dura. Venous return may remain in the dura or enter the leptomeningeal and cortical veins. Dural arteriovenous fistulous malformations (AVFMs) are thought to be acquired artery-to-vein shunts without an intervening malformation or nidus (1, 2). These vascular malformations constitute 10-15% of all intracranial arteriovenous shunts (3). There is little information on the natural history of dural arteriovenous fistulas because they are uncommon and have been difficult to diagnose. These lesions most often afflict women over the age of 40 years, and predominantly involve the transverse sinuses, although any of the intracranial dural sinuses including the cavernous sinus may be affected. The majority of dural AVFM arises within or adjacent to a major venous sinus and is characterized by dural venous drainage (4). The cause and pathogenesis of dural AVFMs remain unclear. Tumors that occluded the transverse and sigmoid sinuses were rarely associated with dural AVFMs (5).

The symptoms of dural arteriovenous fistulas of the

transverse/sigmoid sinus range from benign pulsatile tinnitus to neurological deficits, visual disturbances, and fatal intracranial hemorrhage (6).

We present a case of dural AVFMs in the right transverse sinus with the tentorial meningioma encroaching the transverse-sigmoid sinus junction area.

CASE REPORT

A 62-year-old housewife was admitted with headache and right side tinnitus on June 10, 1998. There was no history of head trauma. A neurological examination at the time of admission revealed no abnormalities. Bruit was not audible around the right retromastoid region.

Examination

Preoperative magnetic resonance imaging (MRI) revealed an enhanced extraaxial tumor along the tentorium, encroaching the transverse dural sinus and transverse-sigmoid junction area (Fig. 1A). The lesion was isosignal intensity to gray matter on T2-weighted images (Fig. 1B), and characteristic dural reaction was revealed along the tentorium and sinus dural wall on Gd-DTPA

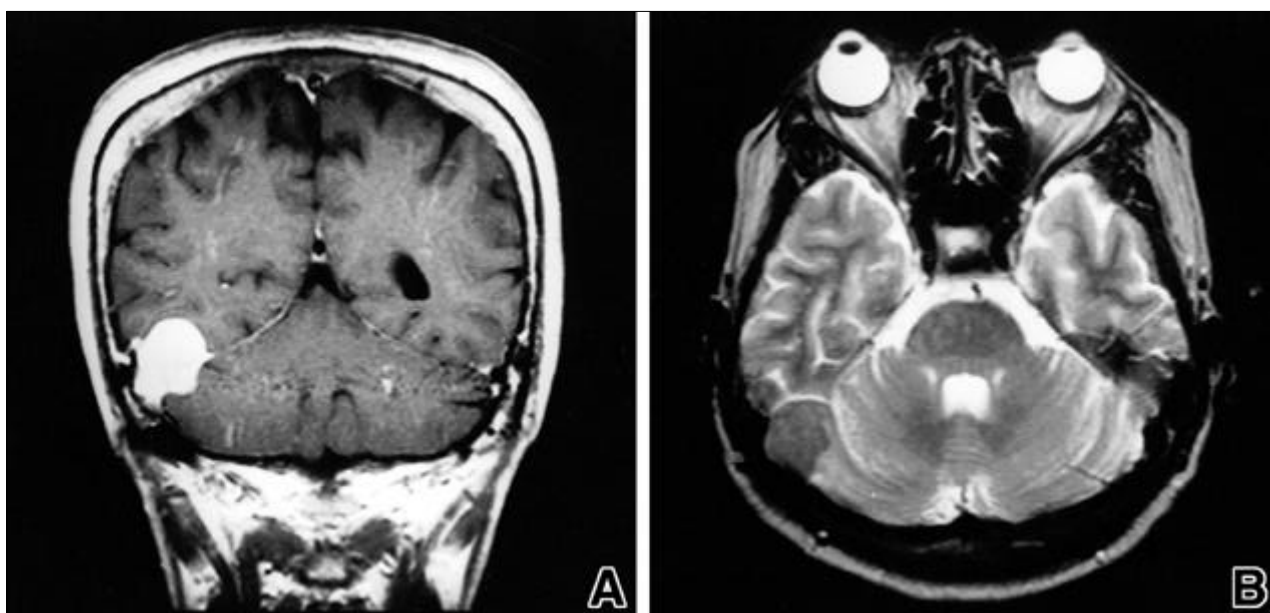


Fig. 1. **A**, Preoperative Gd-DTPA contrast enhanced T1-weighted coronal image shows homogeneously enhanced lobulated mass encroaching the transverse dural sinus with characteristic dural reaction along the tentorium and sinus dural wall. **B**, T2-weighted axial image shows well-marginated extraaxial mass which has iso-signal intensity to gray matter.

enhancement study. Right external carotid angiography revealed an early visualization of sigmoid sinus and jugular vein by transmeningeal arteriovenous fistulas supplied especially from occipital arterial branches of external carotid artery (Fig. 2A) and the right transverse sinus was larger than the left one. And right transverse and sigmoid sinuses were partially occluded on venous phase

angiogram (Fig. 2B).

Operation and postoperative courses

The tumor was shown to originate from the tentorium and involve the right transverse sinus and junction of the right transverse-sigmoid sinuses as well. The total resec-

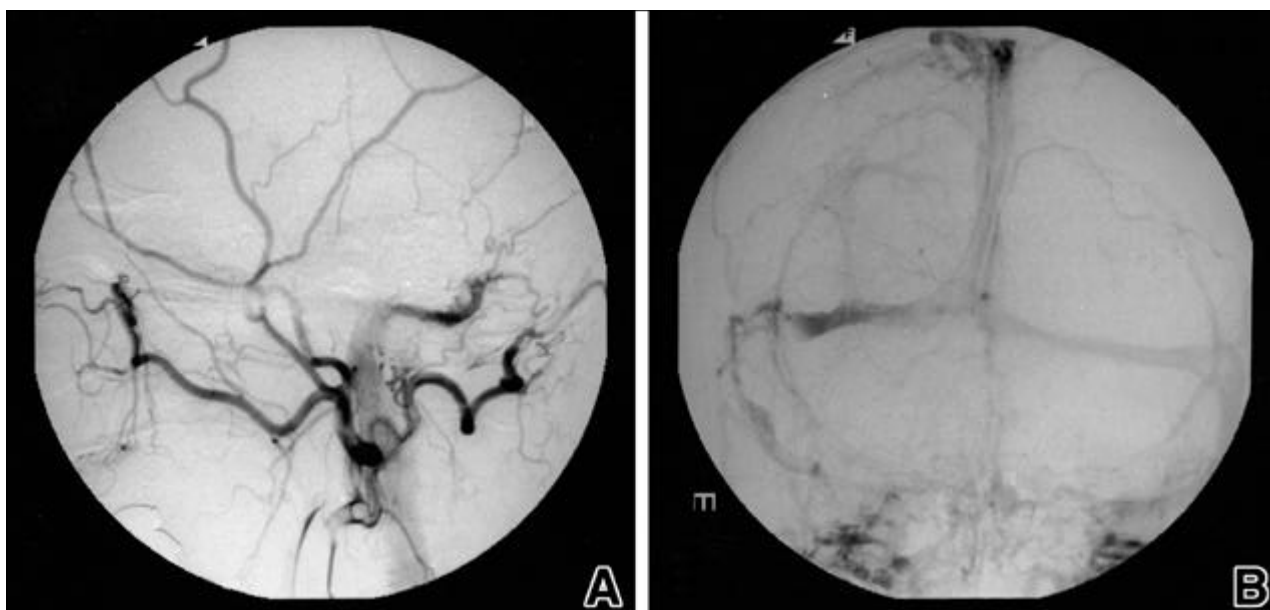


Fig. 2. Preoperative external carotid angiogram **A** shows abnormal early visualization of sigmoid sinus and jugular vein, by transmeningeal arteriovenous fistulas especially from right occipital arterial branches of external carotid artery. **B**, Internal carotid angiogram, venous phase shows poor opacification around transvers-sigmoid sinus junction area.

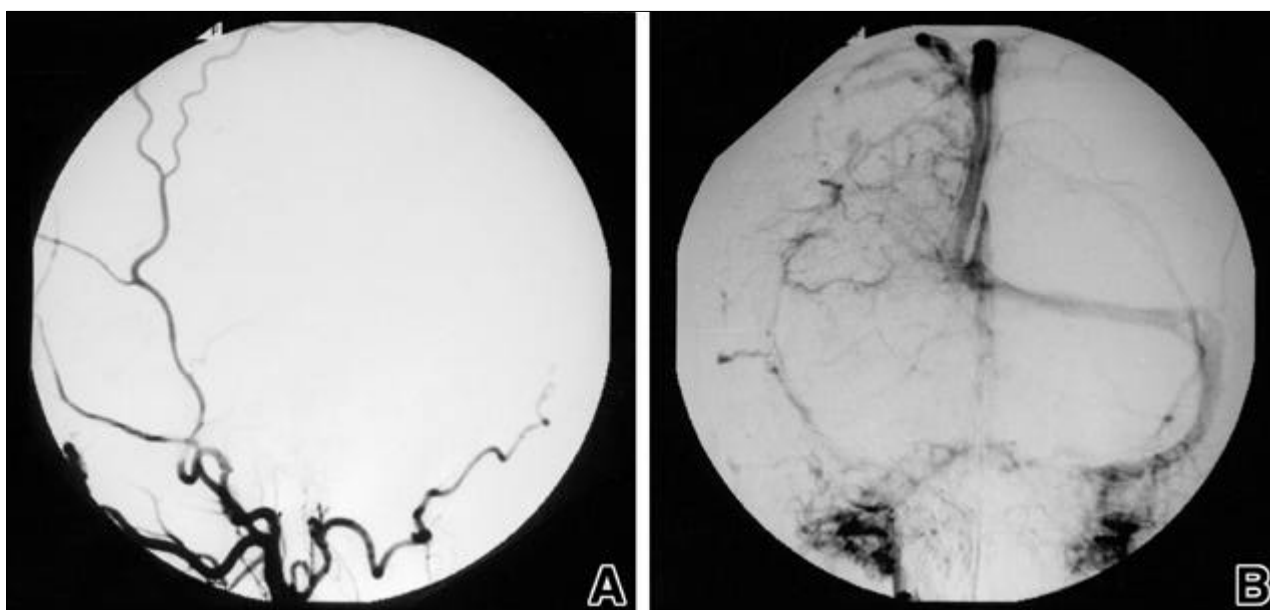


Fig. 3. Postoperative A external carotid angiogram, arterial phase shows disappeared arteriovenous fistulas. B, Internal carotid angiogram, venous phase shows poor opacification of right transverse and sigmoid sinus with multiple venous collaterals.

tion of the tumor was not performed due to the vein of Labbe, which was tightly adherent to the tumor at the junction of the transverse sinus. There was no management for AVFMs during the operation. Follow-up external carotid angiogram showed no dural arteriovenous fistulas and right transverse sinus, and sigmoid sinus was poorly opacified through multiple venous collaterals around the sinuses (Fig. 3). The patient was free from headache and tinnitus after surgery and exhibited no neurological deficits. A histological examination disclosed that the tumor consisted of a fibroblastic type of meningioma.

DISCUSSION

Intracranial dural AVFMs are estimated to occur only one-tenth as frequently as intraparenchymal brain arteriovenous malformations (AVMs) (3).

There is a debate regarding the correctness of the term “dural venous fistula” versus “dural arteriovenous malformation”. Some author prefer the term “malformation” because a single malformation can contain one or more fistulas (7). The term “dural arteriovenous fistulous malformation” (AVFM) would best describe all abnormalities whether they consist of one or more fistulas. It has been recognized that the venous drainage pattern determines the clinical presentation of cranial dural AVFMs (8-11).

Dural AVFMs are classified into two major categories, congenital and acquired. Acquired AVFMs have been

reported to develop secondarily to sinus thrombosis (12, 13), trauma (14-16) or craniotomy (17).

Several hypotheses as to the pathogenesis for AVFMs have been suggested. Awad et al. (8), in their comprehensive meta-analysis, outlined three possible stages in the natural history of dural AVFMs: 1) sinus thrombosis with engorged dural venous collaterals and the opening of embryonic arteriovenous communications; 2) arteriovenous shunting, which favors the recruitment of arterial feeders into the nidus with secondary venous hypertension; and 3) leptomeningeal retrograde venous drainage, with possible subsequent varicose and aneurysmal dilatation. Some degree of flow compromise in the transverse and sigmoid sinus by the either compression or infiltration of sinus wall by the tumor was a prominent feature of pathology in our case. It has been postulated that thrombosis or thrombophlebitis of the dural sinuses is the initial event in the development of dural AVFMs associated with tumors in the sinus (5). The multiple fistulous channels that are seen angiographically are thought to represent a pathological enlargement of normal dural AV shunts in response to thrombosis. Arnautovic (5) claimed that dural AVFMs developed after the tumor occluded the dominant sinus if the contralateral, patent, and nondominant venous sinuses were not able to manage the additional burden of venous drainage from the occluded side. The findings in our case also supported that the flow compromise in dominant transverse sinus by the tumor was responsible for the development of dural AVFMs.

The therapeutic strategy for dural AVFMs includes

transarterial embolization (18), transvenous embolization (19) and surgical resection of the involved sinus (13, 20).

Cranial dural AVFMs are classified into three types, based on anatomical similarities (7). Type I dural AVFMs drain directly into dural venous sinuses or meningeal veins and can be treated by transarterial embolization or surgical skeletonization if venous drainage needs to be preserved. Both of these techniques are less than optimal, being associated with moderate risk, and should be used rarely, especially since type I dural AVFMs usually have minor symptoms. If it is safe to sacrifice the venous drainage, the malformation can be treated by thrombosis of the sinus and coagulation or excision of the meningeal vein. Type II malformations drain into subarachnoid veins. These malformations are treated by ligation of arterialized subarachnoid draining veins and thrombosis or excision of sinus. Type III malformations drain directly into a subarachnoid vein without dural sinus or meningeal vein drainage. These malformations are treated by coagulation and/or excision and ligation of the draining subarachnoid vein. AVFM in our case belongs to the type I malformations which drain directly into sigmoid sinus. Many of these lesions resolve spontaneously, probably on thrombosis of the fistulous connections (21).

In conclusion, we report a case of dural AVFM associated with tentorial meningioma encroaching the transverse and transverse-sigmoid sinus junction area. The dural AVFM disappeared after subtotal resection of the tumor without any neurointervention for the AVFM. Our experience supports the hypothesis that dural AVFMs are acquired and induced. In addition, this lesion was resolved completely without surgical intervention for the AVFMs, indicating the spontaneous resolution of lesion. The possibility of associated AVFMs should be also considered in the diagnostic evaluation of tumors arising adjacent to the dominant transverse/sigmoid sinus.

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