

Dural Arteriovenous Fistula Involving the Superior Sagittal Sinus Following Sinus Thrombosis

—Case Report—

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Abstract

A 57-year-old woman presented with a dural arteriovenous fistula (AVF) involving the superior sagittal sinus (SSS) based upon serial radiological examinations. Her chief complaints were headache and vomiting. Cerebral angiography and magnetic resonance (MR) venography revealed the sinus thrombosis involving the SSS, the bilateral transverse sinuses (TSs), and the right sigmoid sinus. Her symptoms disappeared after anticoagulant therapy. Follow-up MR venography revealed almost complete recanalization of the occluded sinuses, followed by restenosis of the SSS and the left TS and occlusion of the right TS without symptoms. She developed transient right hemiparesis 13 months after the initial onset. Cerebral angiography revealed a dural AVF involving the SSS with cortical reflux into the left frontoparietal region. The dural AVF was occluded by transarterial and transvenous embolization. Her symptom disappeared during the follow-up period.

Key words: dural arteriovenous fistula, sinus thrombosis, superior sagittal sinus

Introduction

Intracranial dural arteriovenous fistulas (AVFs) account for 10–15% of all intracranial arteriovenous lesions in which meningeal and extracranial arteries shunt blood directly into the dural sinuses or, less commonly, into the meningeal or pial veins.⁹⁾ Dural AVFs usually involve the transverse-sigmoid sinus or the cavernous sinus, but may occur in the tentorial incisura, the superior sagittal sinus (SSS), the torcular Herophili, the anterior cranial fossa, the superior petrosal sinus, and the foramen magnum. Dural AVFs of the SSS account for 8–11% of intracranial dural fistulas.^{2,4)} The etiology of dural AVFs is still unclear. The present case radiologically documents the development of dural AVF in a patient with recurrent sinus thrombosis.

Case Report

A 57-year-old woman suffered sudden onset of severe headache and vomiting in December 1998. There was no history of trauma, previous surgery, or medical treatment. Protein-S and protein-C levels were within the normal ranges. Neurological examination revealed no deficits. Computed tomography on admission revealed no subarachnoid blood. Cerebral angiography and magnetic resonance (MR) venography revealed occlusion of the SSS and the bilateral transverse sinuses (TSs), and stenosis of the left sigmoid sinus (Figs. 1 and 2A). She was treated with anticoagulants for sinus thrombosis. Headache and vomiting subsided within 48 hours. MR venography 5 days after the initial onset revealed partial recanalization of the sinuses (Fig. 2B). MR venography in February 1999 revealed nearly complete recanalization of the occluded sinuses (Fig. 2C). Follow-up MR venography in August 1999 revealed restenosis of the SSS and the left TS, and occlusion of the right TS (Fig. 2D). However, she was asymptomatic. She had developed transient mo-

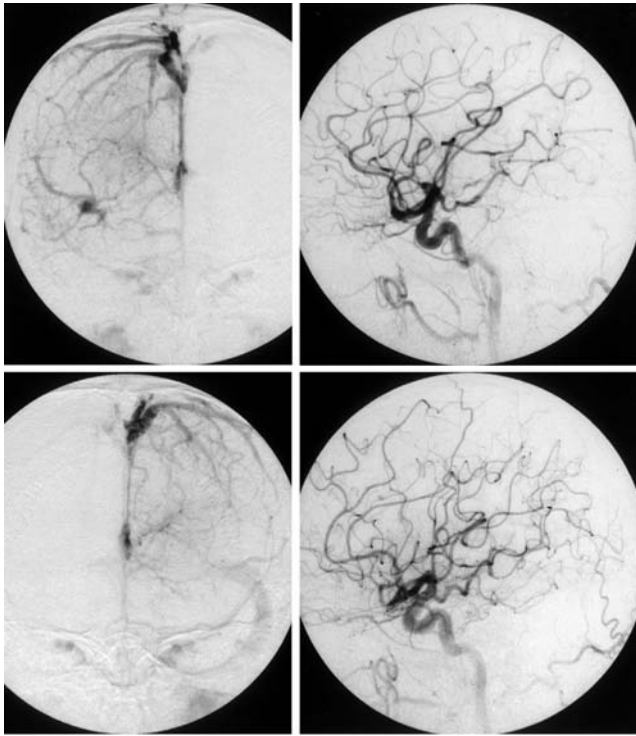


Fig. 1 Right (upper row) and left (lower row) common carotid angiograms, anteroposterior view (left column) and lateral view (right column), on first admission showing sub-total occlusion of the superior sagittal sinus and occlusion of the bilateral transverse sinuses without arteriovenous shunting.

tor weakness in the limbs on the right in January 2000, but recovered completely within few minutes. She was re-admitted for further evaluation in May 2000. Neurological examination revealed no deficits. Cerebral angiography revealed a dural AVF involving the SSS fed by the bilateral superficial temporal arteries, the frontal branches of the middle meningeal arteries, the bilateral parietal branches of the middle meningeal arteries, and the occipital arteries (Fig. 3). The dural AVF drained into the cortical veins of the frontoparietal region on the left through shunt points located in the anterior third of the SSS and in the posterior third of the SSS.

Transarterial embolization with N-butyl cyanoacrylate was performed from the bilateral middle meningeal arteries followed by transvenous embolization of the involved SSS with interlocking detachable coils (Target Therapeutics, Fremont, Calif., U.S.A.). Nearly complete occlusion of the fistula was achieved (Fig. 4). At one-year follow up, she had no neurological problem or further episode of transient motor weakness and follow-up an-

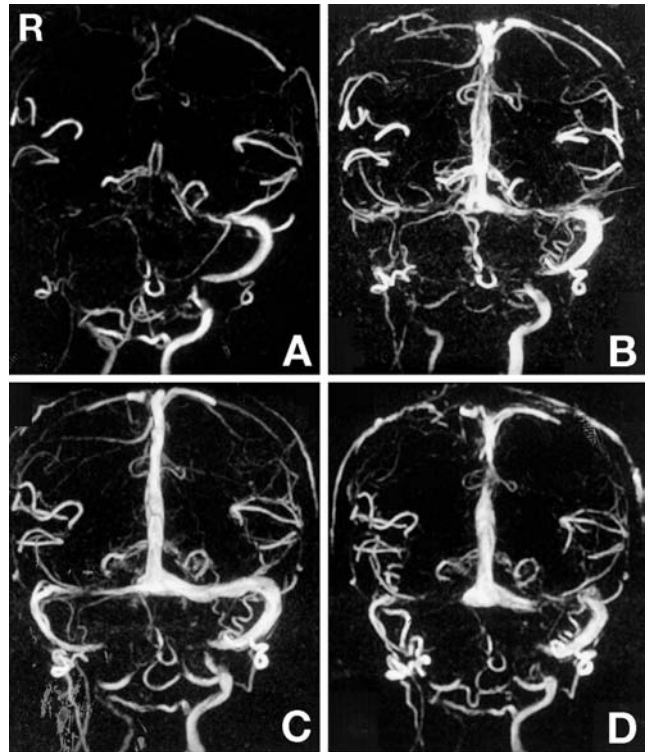


Fig. 2 Magnetic resonance (MR) venograms on admission (A) showing occlusion of the superior sagittal sinus (SSS) and the bilateral transverse sinuses (TSs), and stenosis of the left sigmoid sinus, and 5 days later (B) showing partial recanalization of the sinuses. Follow-up MR venograms 2 months after admission (C) showing almost complete recanalization of the occluded sinuses, and 8 months after admission (D) showing restenosis of the SSS and left TS, and occlusion of the right TS.

giography showed no remarkable change.

Discussion

Serial radiological studies demonstrating the evolution of dural AVF are rare. Previously, five case reports have included angiographic demonstration of sinus occlusion prior to the development of dural AVF.^{1,6,7} Initial angiography demonstrated occlusion of a dural sinus prior to subsequent angiographic demonstration of a typical dural AVF in the same or an immediately adjacent sinus in three cases.^{6,7} Angiography performed prior to and soon after head injury revealed no evidence of dural AVF whereas follow-up angiography demonstrated typical dural AVF in two cases.¹

Serial angiography in our case showed that sinus

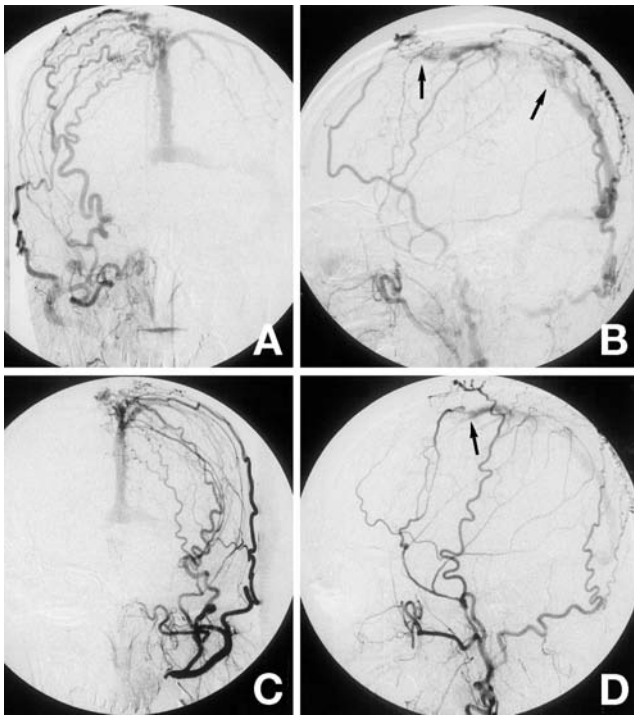


Fig. 3 Right external carotid angiograms, anteroposterior view (A) and lateral view (B), on second admission 17 months after the initial onset showing a dural arteriovenous fistula (AVF) involving the superior sagittal sinus (SSS) fed by the middle meningeal artery, the occipital artery, and the superficial temporal artery with cortical reflux into the frontoparietal region on the left, occlusion of the right transverse sinus (TS), and stenosis of the left TS, with two shunt points (arrows). Left external carotid angiograms, anteroposterior view (C) and lateral view (D), also showing the dural AVF involving the SSS fed by the middle meningeal artery and superficial temporal artery with the same shunt point (arrow) as seen in the right external carotid angiograms.

thrombosis preceded the development of dural AVF and follow-up MR venography a few days after the initial onset showed recanalization probably due to the increase of secondary fibrinolysis. Thereafter, sinus thrombosis occurred again possibly resulting in long-standing venous hypertension and then dural AVF development.

Possible etiologies of sinus thrombosis include infection, head trauma, Behçet's disease, oral contraceptives, lupus erythematosus, antithrombotic III deficiency, carcinoma, nephritic syndrome, and protein-S or protein-C deficiency, but our patient had no known cause of sinus thrombosis. The etiolo-



Fig. 4 Left common carotid angiogram after the embolization showing nearly complete occlusion of the fistula, but a minimal shunt persisting in the anterior portion of the superior sagittal sinus (SSS) fed by the falx artery draining into the SSS anteriorly without cortical reflux.

gy of dural AVF is unclear, although many cases have been associated with sinus occlusion and/or sinus stenosis. Sinus thrombosis may not always lead to the development of dural AVF, whereas venous hypertension may be an essential condition for the formation of dural AVF.^{5,10)}

Treatment of dural AVFs includes oral drug therapy, direct surgical resection or isolation of the involved sinus, and endovascular treatment.^{3,4)} The involved sinuses may be part of the normal drainage route, even if only marginal. In this situation, total resection or total occlusion of the sinus could result in venous infarction. Therefore, feeder embolization, feeder coagulation, or occlusion of shunt site should be performed if the sinus is patent.⁸⁾ If the involved sinus does not participate in normal venous drainage, total resection or total occlusion can be safely performed. Normal cortical venous flow may drain via the sinus wall rather than the sinus. In this situation, endovascular occlusion of the involved sinus should be performed safely with preservation of drainage in this sinus wall because total resection of the involved sinus including the sinus wall will cause venous infarction. The involved SSS was not part of the normal drainage route in our case, so we selected embolization of the involved SSS. This case indicates that patients with sinus thrombosis should be followed up to detect the development of dural AVF.

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