Development of Sigmoid Dural Arteriovenous Fistulas after Transvenous Embolization of Cavernous Dural Arteriovenous Fistulas

Kohsuke Yamashita, Waro Taki, Ichiro Nakahara, Shogo Nishi, Akiyo Sadato, and Haruhiko Kikuchi

Summary: We present a case in which a sigmoid sinus dural arteriovenous fistula developed after successful transvenous embolization of a cavernous dural arteriovenous fistula and discuss the pathogenesis of the second dural arteriovenous fistula.

Index terms: Fistula, arteriovenous; Arteriovenous malformations, intracranial; Arteriovenous malformations, embolization; Interventional neuroradiology, complications of

Dural arteriovenous fistulas (DAVFs) comprise 10% to 15% of intracranial arteriovenous malformations (1). They most commonly involve the transverse, the sigmoid, and the cavernous sinuses (2).

Case Report

A 54-year-old woman presented with left proptosis, chemosis, ophthalmoplegia, and tinnitus in March 1989. Angiography demonstrated a left cavernous DAVF. The feeding arteries from the left external carotid artery were embolized. Her symptoms were relieved for a while but soon recurred.

On May 2, she was referred to us for treatment. Angiography revealed that all the branches of the left external carotid artery were recanalized and fed the DAVF (Fig 1A). In addition, the DAVF was also fed by the branches of the right external carotid artery and by the dural branches of the left internal carotid artery. A 5-F catheter was placed in the left external carotid artery and a Tracker-18 catheter (Target Therapeutics Inc, San Jose, Calif) was advanced coaxially within it. The middle meningeal artery and the internal maxillary artery were occluded with coils (Hilal embolization microcoils, Cook, 489 Bloomington, Ind) (Fig 1B). Her ocular symptoms were greatly alleviated, but tinnitus remained. The DAVF was still supported by the left internal carotid angiogram. One month later, the remnant fistula was occluded with coils introduced through the left inferior petrosal sinus from a jugular vein access. The DAVF disappeared angiographically, and the patient's clinical symptoms disappeared as well (Fig 1C).

In May 1990, the patient again presented with left tinnitus. There were no ocular signs. Angiography revealed a new DAVF involving the left sigmoid sinus, which was fed by the occipital artery, the retroauricular artery, and the ascending pharyngeal artery of the left external carotid artery (Fig 1D), and the clival branch of the meningohypophyseal trunk of the left internal carotid artery (Fig 1E). Because there were no signs of increased intracranial pressure and no cortical venous reflux, only some of the feeding arteries were occluded with coils. Her tinnitus was lessened.

Discussion

The present case is interesting because the sigmoid sinus DAVF appeared for the first time after the successful transvenous embolization of the cavernous DAVF.

Multiple DAVFs seem to be a rare clinical entity (3–11). The incidence of multiple DAVFs has been reported to be 7% (12), but excluding duplicate reports (9, 11, 12), we know of only 17 cases described in the literature. Excluding a case of multiple spinal DAVFs (12) and two ill-defined cases, 14 intracranial cases are well described. The involved sinuses have been the transverse and sigmoid sinus in 10 cases, the superior sagittal sinus in eight, the cavernous sinus in six, the occipital sinus in one, the inferior petrosal sinus in one, the sphenoparietal sinus in one, and the ethmoid groove vein in one. It may be interesting that the superior sagittal sinus has been involved with surprisingly high incidence. Coexistence of multiple DAVFs has been observed in 13 cases, although it is not clear that they coexisted from the beginning. A case reported by Kuwayama et al (10) is interesting because a lateral sinus DAVF appeared after the spontaneous resolution of a

Received March 17, 1992; revision requested August 11, received and accepted September 11.

All authors: Department of Neurosurgery, Faculty of Medicine, Kyoto University, 54 Shogoinkawaharacho, Sakyo-ku, Kyoto, 606 Japan. Address reprint requests to Kohsuke Yamashita, MD.

AJNR 14:1106–1108, Sep/Oct 1993 0195-6108/93/1405–1106

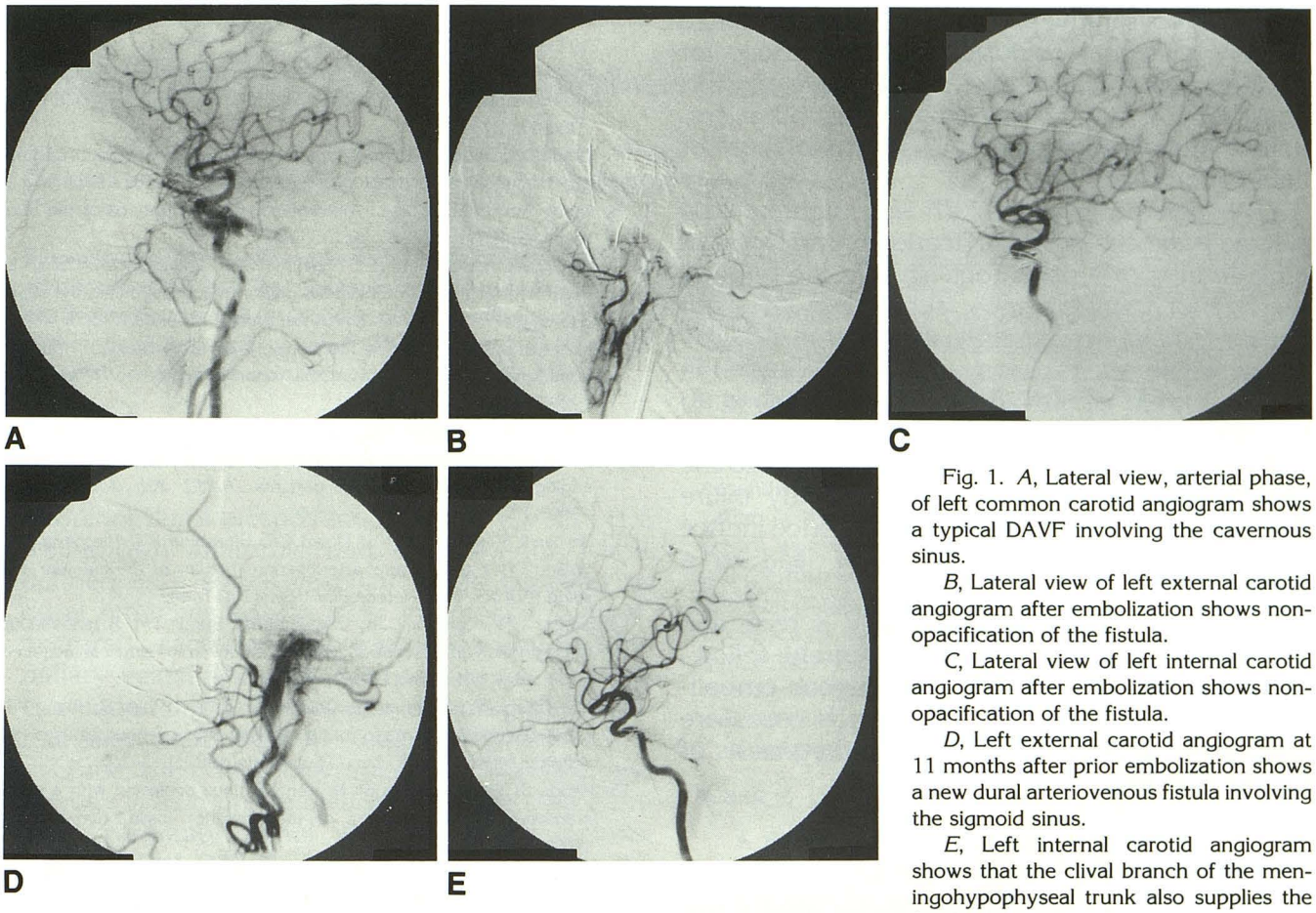

Fig. 1. *A*, Lateral view, arterial phase, of left common carotid angiogram shows a typical DAVF involving the cavernous sinus.

B, Lateral view of left external carotid angiogram after embolization shows nonopacification of the fistula.

C, Lateral view of left internal carotid angiogram after embolization shows nonopacification of the fistula.

D, Left external carotid angiogram at 11 months after prior embolization shows a new dural arteriovenous fistula involving the sigmoid sinus.

E, Left internal carotid angiogram shows that the clival branch of the meningohypophyseal trunk also supplies the fistula.

cavernous DAVF. Among six cases with a cavernous DAVF, the location of the other DAVF has been the ethmoidal groove, the sphenoparietal sinus (12), the inferior petrosal sinus (11, 12), the lateral sinus, the transverse sinus (10), and the superior sagittal sinus (9, 12), respectively. With the exception of the last case, the second DAVF was found on the same side as and involved the efferent veins or sinuses from the cavernous sinus DAVF. This feature is found in the present case.

Formation of a second DAVF can be explained by the well established venous pathology theory (13, 14). When a cavernous DAVF is present, the shunted flow might cause turbulence and reversal of venous flow, which could lead to stagnation and thrombosis within the efferent venous structures (10, 12, 15). Through the recanalization process, another DAVF may develop. However, thrombosis of the sigmoid sinus was not demonstrated in the present case.

Participation of the physiologic arteriovenous shunts of the dura mater (16, 17) also should be considered. When a cavernous DAVF is present, the dural arterial pressure around the cavernous sinus may be lowered by a "steal" effect of the feeding arteries and the venous pressure may be elevated by the shunted venous flow, resulting in decreased perfusion pressure of the dura mater. This may make the physiologic arteriovenous shunts around the cavernous sinus dilate to maintain the dural circulation. In this state, the decreased arterial and perfusion pressure may act to keep the dural arterio-venous shunts dormant or nondemonstrable. However, by closing the cavernous DAVF, the local arterial pressure may be elevated and transmitted to the dural arteries adjacent to the cavernous sinus through the arterial communications while the venous pressure may be lowered. This elevated perfusion pressure may work to establish the physiologic shunts as fistula. This hypothesis is applicable to the present case and is interesting because the predilective site of the physiologic shunt and the DAVF coincided with each other at the superior sagittal sinus (13, 14). The feeding arteries were embo-

lized with coils in the present case, which resulted in only proximal occlusion and left room for development of collaterals. In order to lessen this possibility, it may be advisable to use other embolic materials such as polyvinyl alcohol foam or liquid adhesives.

Development of new DAVF also might be considered as a possible complication of transvenous embolization or a cavernous sinus lesion through the inferior petrosal sinus, although it does not appear to have been previously reported (18, 19). The possible role of transvenous embolization in the formation of DAVF might be ascribed to venous pathology (13, 14). Manipulation of a catheter and guide wire to negotiate a path to the inferior petrosal sinus may inadvertently injure the intima of the sigmoid sinus, and hence induce thrombosis, which may eventually lead to a DAVF.

Occurrence of the second DAVF is rare, and the pathogenesis is still uncertain. Careful follow-up of patients treated with transvenous embolization and the accumulation of such cases are needed for understanding of the pathogenesis of multiple DAVFs.

References

1. Newton TH, Cronqvist S. Involvement of the dural arteries in intracranial arteriovenous malformations. *Radiology* 1969;93:1071–1078
2. Lasjaunias P, Chiu M, Brugge KT, Tolia A, Hurth M, Bernstein M. Neurological manifestations of intracranial dural arteriovenous malformations. *J. Neurosurg* 1986;64:724–730
3. Newton TH, Weidner W, Greits T. Dural arteriovenous malformation in the posterior fossa. *Radiology* 1968;90:27–35
4. Houser OW, Baker HL, Rhoton AL, Okazaki H. Intracranial dural arteriovenous malformations. *Radiology* 1972;105:55–64
5. Aminoff MJ. Vascular anomalies in the intracranial dura mater. *Brain* 1973;96:601–612
6. Kataoka K, Taneda M. Angiographic disappearance of multiple dural arteriovenous malformations, case report. *J Neurosurg* 1984;60:1275–1278
7. Graeb DA, Dolman CL. Radiological and pathological aspects of dural arteriovenous fistulas, case report. *J Neurosurg* 1986;64:962–967
8. Grady MS, Pobereskin L. Arteriovenous malformations of the dura mater. *Surg Neurol* 1987;28:135–140
9. Halbach VV, Higashida RT, Hieshima GB, Rosenblum M, Cahan L. Treatment of dural arteriovenous malformations involving the superior sagittal sinus. *AJNR: Am J Neuroradiol* 1988;9:337–343
10. Kuwayama N, Takaku A, Nishijima M, Endo S, Hirao M. Multiple dural arteriovenous malformations, report of two cases. *J Neurosurg* 1989;71:932–934
11. Barnwell ST, Halbach VV, Dowd CF, Higashida RT, Hieshima GB. Dural arteriovenous fistulas involving the inferior petrosal sinus: angiographic findings in six patients. *AJNR: Am J Neuroradiol* 1990;11:511–516
12. Barnwell ST, Halbach VV, Dowd CF, Higashida RT, Hieshima GB, Wilson CB. Multiple dural arteriovenous fistulas of the cranium and spine. *AJNR: Am J Neuroradiol* 1991;12:441–445
13. Houser OW, Campbell JK, Campbell RJ, Sundt TM. Arteriovenous malformation affecting the transverse dural venous sinus: an acquired lesion. *Mayo Clin Proc* 1979;54:651–661
14. Chaudhary MY, Sachdev VP, Cho SH, Weitzner I, Puljic S, Huang YP. Dural arteriovenous malformation of the major venous sinuses: an acquired lesion. *AJNR: Am J Neuroradiol* 1982;3:13–19
15. Handa J, Yoneda S, Handa H. Venous sinus occlusion with a dural arteriovenous malformation of the posterior fossa. *Surg Neurol* 1975;4:433–437
16. Rowbotham GF, Little E. Circulations of the cerebral hemispheres. *Br J Surg* 1965;52:8–20
17. Kerber CW, Newton TH. The macro and microvasculature of the dura mater. *Neuroradiology* 1973;6:175–179
18. Halbach V, Higashida R, Hieshima G, Hardin C, Pribram H. Transvenous embolization of dural fistulas involving the cavernous sinus. *AJNR: Am J Neuroradiol* 1989;10:377–383
19. Takahashi A, Yoshimoto T, Kawakami K, Sugawara T, Suzuki J. Transvenous copper wire insertion for dural arteriovenous malformations of cavernous sinus. *J Neurosurg* 1989;70:751–754