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A dural arteriovenous fistula in cavernous sinus developed from viral meningitis

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Abstract

Although hormonal influences, inflammation, trauma, sinus thrombosis, venous hypertension, and congenital origin have been proposed as sources of dural arteriovenous fistulas (DAVFs) in cavernous and sigmoid sinuses, the etiology of these lesions remains controversial. We present a case with a cavernous sinus DAVF developed from viral meningitis which has not been previously described. A 24-year-old male was admitted to our institute because of periorbital pain, decreased vision, pulsatile tinnitus, chemosis, and exophthalmos on the right side after he had suffered viral meningitis four months before. Cerebral angiography demonstrated a cavernous sinus DAVF, which was successfully obliterated with several platinum coils using a transvenous approach. The viral meningitis most likely caused the inflammation, that may be responsible for the occurrence of the cavernous sinus DAVF. Prompt treatment for inflammation may help to prevent the development of DAVFs.

Key words: Dural arteriovenous fistula; cavernous sinus; virus; inflammation; etiology.

Introduction

Intracranial dural arteriovenous fistulas (DAVFs) account for 15% of all cerebrovascular malformations and are thought to be acquired shunts between cerebral arteries and venous sinuses or epidural veins (1, 2). The most frequent locations of intracranial DAVFs are the cavernous and the sigmoid sinus (3, 4). Although hormonal influence, inflammation, trauma, sinus thrombosis, venous hypertension, and congenital origin have been proposed as causes of these lesions (1-7), the precise etiology of DAVFs remains controversial. We report a patient with a cavernous sinus DAVF developed from viral meningitis.

Case report

A 24-year-old man with fever, headache and vomiting was referred to local hospital on March 5, 2009. On admission, physical examination revealed neck stiffness. Cerebrospinal fluid (CSF) test obtained with lumbar puncture had an increased number of nucleated cells and normal levels of protein and glucose. Blood tests showed positive in Herpes Simplex Virus IgG and Cytomegalovirus IgG antibodies. Both CT and MRI scans did not demonstrate any abnormalities in the cavernous sinus region (Fig. 1). At this stage, the patient was diagnosed as the viral meningitis and received antivirus and steroid therapy. He improved gradually and recovered fully after more than one month.

Four months after the viral meningitis, the patient complained of diplopia which lasted for about 20 days. After that, he had developed right periorbital pain, decreased vision, pulsatile tinnitus, chemosis, and exophthalmos and admitted to our Neurosurgery Department on October 30, 2009. To confirm the pathology, cerebral digital subtraction angiography (DSA) was performed. An arteriovenous shunt from bilateral internal carotid arteries (ICA) and external carotid arteries (ECA) to the cavernous sinus, reflux to the right ophthalmic vein, inferior petrosal sinus and the other side of the cavernous sinus was demonstrated (Fig. 2A). We diagnosed therefore cavernous sinus DAVF.

Transvenous cavernous coiling through the right inferior petrosal vein was decided. The patient was systemically heparinized during the endovascular procedure, and we maintained an activated clotting time between 200 and 300 seconds. The fistula was occluded by several platinum coils. Postembolization angiograms showed that the DAVF disappeared

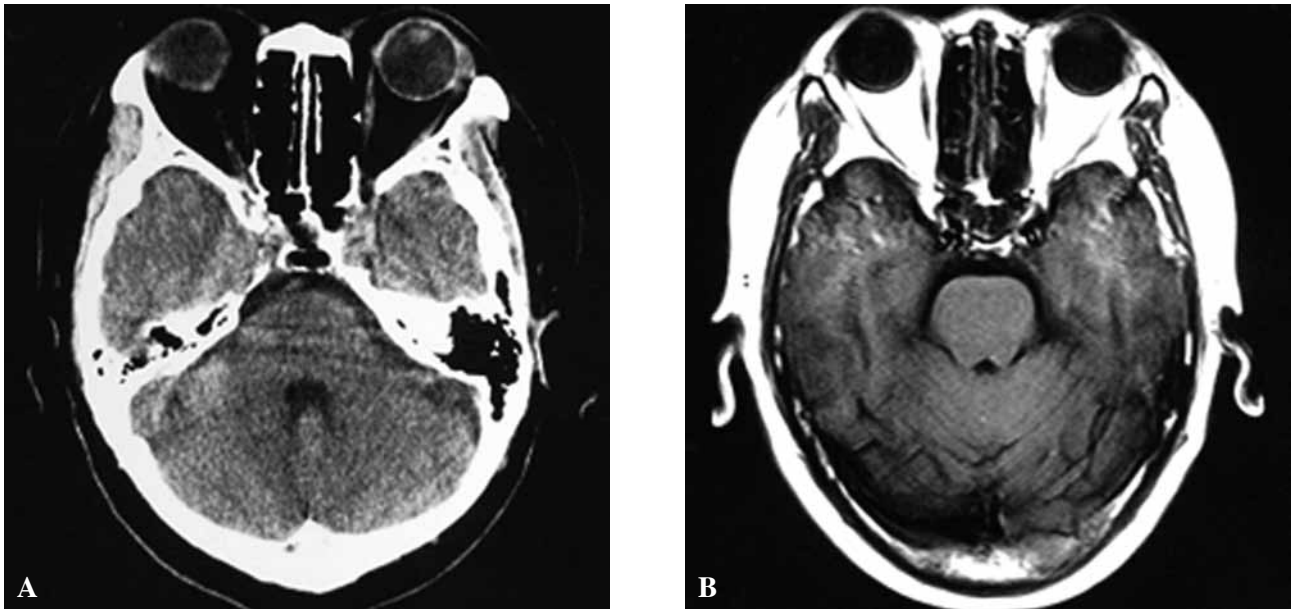

FIG. 1. — Axial CT (A) and MRI (B) scans showed no abnormalities around the cavernous sinus area

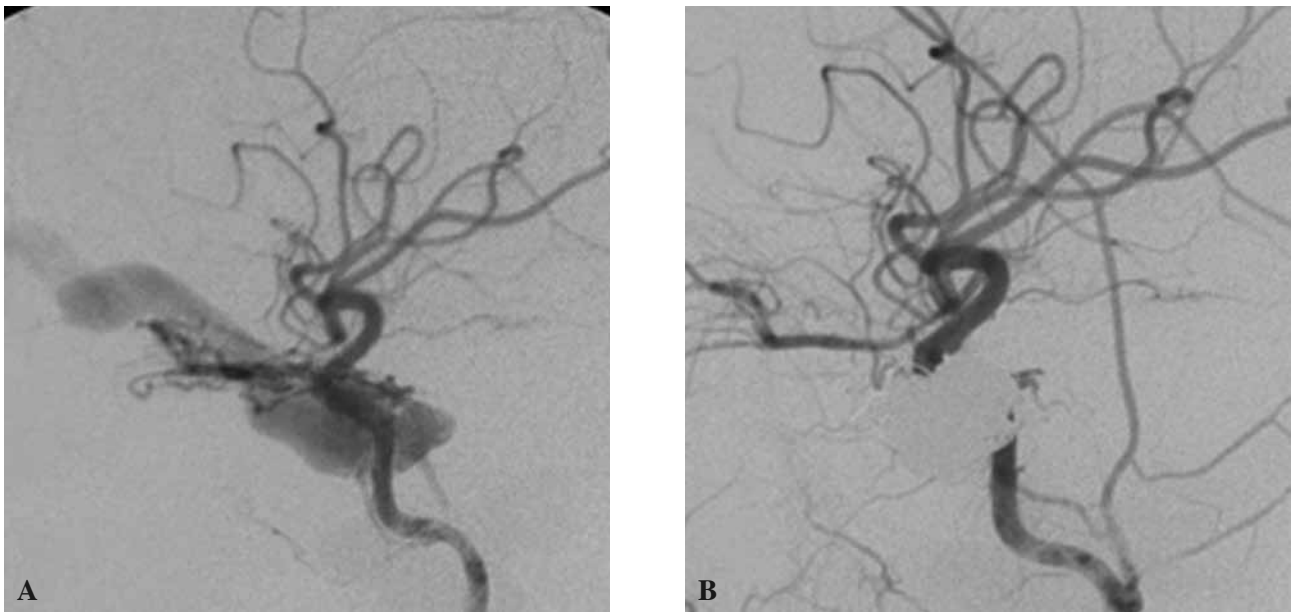

FIG. 2. — Cerebral angiography showed the cavernous sinus DAVF (A). After coiling, repeated angiograms revealed a completely occluded cavernous sinus DAVF (B).

(Fig. 2B) and the patient's symptoms improved immediately and completely.

Discussion

Intracranial DAVFs are generally considered to be acquired, progressive lesions and composed of innumerable tiny abnormal connections between dural arteries and venous sinuses (1). While the etiology of the DAVFs still remains unclear, several theories including hormonal influence, inflammation, trauma, sinus thrombosis and venous hypertension have been proposed (1-7).

In this case, the patient had a definite history of viral meningitis which confirmed by clinical symptoms, signs, CSF and blood tests. Four months after

the intracranial infection, the patient initially presented with diplopia, which may be related to the steal phenomenon of blood supply to the oculomotor nerve (8). Twenty days later, this ocular symptom resolved spontaneously. Typical symptoms and signs of a cavernous sinus DAVF developed subsequently. From the evidence of our case, we postulated that the initial step for developing the cavernous sinus DAVF might be the inflammation.

Several clinical and experimental studies have demonstrated that venous hypertension and sinus occlusion contributed to the development of intracranial DAVFs (1-3, 8, 9). Based on an animal DAVF model, Lawton et al suggested that sinus occlusion and venous hypertension may induce angiogenic activity by decreasing cerebral perfusion and increasing ischemia, and this neovascularization may increase intrasinus pressure (9). However, we did not detect any dural sinus thrombosis in this case during the procedure of cerebral angiography.

Another theory is that preexisting embryonic arteriovenous communications in normal dura mater can open to create abnormal fistulas between meningeal arteries and dural sinus under certain pathological conditions (7). Inflammatory angiogenesis can produce vascular connections between the dural mater and the potential portion of the sinus which are consistent with the presented case.

In addition, venous hypertension induced by arteriovenous shunting may lead to cerebral ischemia, followed by angiogenesis (10). Cerebral ischemia may be one of the key factors and could stimulate neovascularization. Aberrant angiogenic activity in the dural vessels then could exacerbate arteriovenous shunting, thereby creating a self-perpetuating cycle.

It is known that inflammation can stimulate angiogenesis. Experimental evidence has confirmed that several angiogenic growth factors such as vascular endothelial growth factor (VEGF) and basic fibroblast growth factor (bFGF) are involved in the inflammatory process and enhance angiogenesis (11). In our patient, the inflammatory response resulting from the viral meningitis may have led to the development of the cavernous sinus DAVF.

To the best of our knowledge, cavernous sinus DAVF developed from a viral meningitis has not been previously described. Our case suggests that the inflammatory response was one of the initial key factors leading to the occurrence of cavernous sinus DAVFs. Therefore, prompt treatment for inflammation may help to prevent the development of cavernous sinus DAVFs.

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