Dural Arteriovenous Fistula of the Cavernous Sinus with Brainstem Edema

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Dural arteriovenous fistula of the cavernous sinus is usually a benign disease and rarely has brainstem edema as its complication. We present a patient with dural arteriovenous fistula of the cavernous sinus and brainstem edema. The brainstem edema resolved after embolization. Embolization should be considered first whenever brainstem edema occurs, progressive visual deterioration or increased intracranial pressure is encountered in a case of dural arteriovenous fistula of the cavernous sinus.

Key words: Arteriovenous malformation, Brainstem edema, Dural AVF, Dural arteriovenous malformation of the cavernous sinus, Embolization

Patients with dural arteriovenous fistulae of the cavernous sinus (DAVFC) have abnormal communications between arteries and vein (the cavernous sinus) at the wall of the cavernous sinus. The DAVFC may also be called dural arteriovenous malformation of the cavernous sinus. The arterial blood supply may come from dural branches of the internal carotid artery (Barrow's classification B), external carotid artery (Barrow's classification C), or both the internal and the external carotid arteries (Barrow’s classification D) [1]. The fistula may be present in unilateral or bilateral cavernous sinuses. In addition to self-heard noise and bruits, its clinical presentations are closely related to the pattern of venous drainage of DAVFC. Most patients have symptoms of proptosis, chemosis, and conjunctival congestion because of venous drainage to the superior or inferior ophthalmic vein. Some patients may have cranial nerve palsy of III, IV, VI nerves as well. Severe neurorological complications such as intracerebral hemorrhage, and subarachnoid hemorrhage [2], and brainstem edema occur rarely.

We present a patient with brainstem edema and acute aggravation of neurorological status. The brainstem edema was reversed after embolization.

CASE REPORT

A 76-year-old woman fell from a bicycle and sustained abrasion wounds over her extremities about 2-3 weeks prior to admission. Left eye redness and swelling was noted gradually after that. Dysarthria was noted about 10 days prior to admission. Left hemiplegia was noted about 5 days prior to admission.

Neurological examination on admission showed: clear consciousness, dysarthria and left
hemiplegia (muscle power 1/5). Sensory functions could not be checked.

MRI showed marked edema with an area of marginal enhancement indicating disruption of blood-brain barrier in her right brainstem at the level of the pons and midbrain (Fig. 1).

Angiogram showed a dural AVF (or AVM) of the right side cavernous sinus fed by bilateral external carotid arteries and posterolateral trunk of right side internal carotid artery (Fig. 2). It was compatible with a type D arteriovenous communication at the cavernous sinus by Barrow’s classification [3]. The bilateral superior and inferior petrosal sinuses were not patent. Because of brainstem edema, emergent embolization was performed. Because superselective catheterization of external carotid feeders failed, the first embolization was performed on June 18, 1998. We used coils and an NBCA mixture using direct puncture of the cavernous sinus through the orbit and the superior orbital fissure according to techniques described previously [4] (Fig. 3). The second stage embolization was performed 4 days later using superselective catheterization of the distal internal maxillary artery with injection of NBCA mixture inside (Fig. 4).

Follow-up angiogram on June 30, 1998 showed complete obliteration of the fistula (Fig. 5). The patient recovered from left hemiplegia about 1 month after embolization. MRI of brain performed 4 months after the operation (November 5, 1998) showed resolution of brainstem edema and swelling, leaving a small lacuna in the right side pons (Fig. 6).
Drainage must be considered [8]. These patients may present with rapidly progressive myelopathy and autonomic disorders. Slow venous drainage may be drained through dilated perimedullary cervical veins. Spinal dural AVF has also been reported to cause spinal cord edema [9].

The mechanisms of brain edema in the brainstem or spinal cord from dural AVF are similar due to venous hypertension. The venous hypertension is partly from AVF with arterial blood draining to the area of brain or spinal cord, and partly from impaired venous drainage of the fistula.

From our case report and most others in previous reports, the brainstem edema or spinal cord edema was reversible after suitable treatment such as embolization. Therefore in cases of DAVFC with brainstem edema, embolization should be performed as soon as possible to revert patient’s clinical symptoms.

The treatment of patients with DAVFC includes embolization, radiosurgery, and conventional radiation therapy [10-15]. Radiosurgery alone was effective for patients with DAVFC whose arterial supply was not accessible via the transarterial approach, although the time course for improvement was longer (median, 12 months) [10]. Hirai et al reported that conventional radiation therapy resulted in cure of DAVFC in 75% of patients [11]. The response of patients to conventional radiation therapy may take months to years [11-13]. Fast flowing DAVFC may not always be improved after radiation therapy, thus embolization should be considered [11]. If the patient has brain edema, progressive visual deterioration, or signs of intracranial hypertension, then embolization should be considered just as we have done in this case report.

Embolization of the DAVFC in the past could be transarterial or transvenous [12]. Direct puncture of the cavernous sinus has been reported before for embolization of direct type carotid cavernous fistula [15]. This is our first time to try this technique in the treatment of DAVFC because of the urgent condition of symptomatic brainstem edema and the difficulty in using other techniques.

In conclusion, brainstem edema may be a complication of dural AVF of the cavernous sinus. Embolization should be conducted as soon as possible to relieve the clinical symptoms.

Figure 4. Superselective angiogram of the distal branch of the right internal maxillary artery before second stage embolization. Abnormal early venous opacification is still noted in the posterior fossa (arrow).

Figure 5. The dural AVF is not seen in the lateral view of right common carotid angiogram after two stages of embolization.

Figure 6. T2-weighted MRI 3 months after embolization shows disappearance of previously shown high signal change in the midbrain.
REFERENCES


海綿狀竇的硬膜動靜脈瘻管合併腦幹水腫

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海綿狀竇的硬膜動靜脈瘻管通常是一個良性疾病。腦幹水腫是其一個很少見的併發症。我們報告一例合併腦幹水腫之海綿狀竇的硬膜動靜脈畸形。此一例經由栓塞術治療而使腦幹水腫緩解。常規之放射線治療或加馬刀定位治療後要等幾個月才能看出治療效果。而栓塞術可以立即達到阻斷動靜脈瘻管之目的。因此，當海綿狀竇之硬膜動靜脈瘻管有腦幹水腫、視力快速變差、或顱內壓升高之現象時，應優先用栓塞術治療。

關鍵詞：動靜脈畸形，腦幹水腫，硬膜動靜脈瘻管，海綿狀竇的動靜脈畸形，栓塞術