Letters to Editor

De novo formation and rupture of an intracranial aneurysm

Sir,

De novo aneurysm is a newly formed aneurysm developing from a cerebral vessel which had appeared to be normal in a previous angiographic study. They have been usually discovered some time after successful treatment of aneurysms located elsewhere or after subarachnoid hemorrhage (SAH) of unknown origin.[1-5] This report concerns two such patients.

**Patient 1**

A 40-year-old male, a chronic heavy smoker with uncontrolled hypertension, was admitted after SAH in grade II according to Hunt and Hess (H and H) scale. CT scan showed SAH predominantly located in the right Sylvian fissure. Four-vessel angiography demonstrated right internal carotid artery aneurysm [Figure 1a and 1b], in the region of the posterior communicating artery and no other aneurysms. The aneurysm was successfully clipped. Inspection of the carotid artery revealed no other abnormalities. The patient recovered completely, but later suffered from arterial hypertension and was treated with antihypertensive medication.

The patient was readmitted five years later with new episode of SAH in H and H grade III. CT scan showed a SAH and an intracerebral hematoma in the left frontal lobe. Angiography revealed an aneurysm of the anterior communicating artery [Figure 2c], which projected superiorly and filled by the left carotid artery. The aneurysm was clipped. Postoperatively the patient had slight dysphasia and disorientation, which gradually resolved.

Our observations and similar reports from literature demonstrate that de novo aneurysms can develop over some periods of time after initial SAH. Although there are no guidelines at present, periodic follow-up to detect de novo aneurysms is recommended by most authors,[1,3-5] particularly in young patients with risk factors such as arterial hypertension,[2] smoking,[2,4] multiple and familial aneurysms,[1] and congenital connective tissue disorders.[3] MR angiography and 3-dimensional CT angiography can be used as a good screening tool for de novo aneurysms.

**Patient 2**

A 41-year-old man, non-smoker with a history of arterial hypertension, was admitted after SAH in H and H grade II. CT scan revealed a SAH into basal cisterns and the interhemispheric fissure. Four-vessel angiography did not show any abnormalities or signs of spasm [Figure 2a and 2b]. The patient refused control angiography and was lost to further follow-up.

Ten years later, he was readmitted with a new episode of SAH in H and H grade III. CT scan showed a SAH and an intracerebral hematoma in the left frontal lobe. Angiography revealed an aneurysm of the anterior communicating artery [Figure 2c], which projected superiorly and filled by the left carotid artery. The patient had slight dysphasia and disorientation, which gradually resolved.

Crucial problem concerning de novo aneurysms is that what we assumed to be newly developed aneurysm may have been present already, but unrecognized at the time of the first angiography. We believe that none of our two de novo aneurysms were present in initial angiograms. In our first patient not only the angiograms, but surgical exploration also confirmed the absence of any lesion at carotid artery bifurcation. In our second patient, bilateral carotid and vertebral angiography covering multiple directional views and very long time interval between first and second SAH make false negative results very unlikely.

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References


Accepted on 02-03-2006
Brain stem infarction: A complication of microvascular decompression for trigeminal neuralgia

Sir,

Microvascular decompression (MVD) of trigeminal nerve (TN) is the treatment of choice for typical trigeminal neuralgia in patients refractory to medical therapy. In experienced hands, this procedure is safe; however, the complication rate reported in different series is 1-4% with a mortality of 0.5%. The incidence of complications such as cerebellar and brainstem venous infarct, transverse sinus thrombosis following MVD is not known except for scanty case reports.

We encountered a rare complication of MVD for trigeminal neuralgia in a 54-year-old man. He presented with the right-sided facial pain in V2, V3 distribution, typical of trigeminal neuralgia, for a period of 6 years. He was refractory to medical treatment. MRI of brain was normal. Microvascular decompression of TN was done in semi-sitting position. Trigeminal nerve was compressed by superior cerebellar artery along the anteroinferior aspect. Superior petrosal vein got avulsed during retraction and hence was coagulated. A PTFE (polytetrafluroethylene) patch was placed between TN and the superior cerebellar artery.

Postoperatively, patient was conscious but response to command was delayed. On the first postoperative day, he became tachypnoeic, confused, dysarthric and ataxic with right-sided lower cranial nerve paresis and cerebellar signs. CT brain showed right cerebellar hemispheric hypodensity with hydrocephalus. Right VP shunt was done. CSF pressure was raised and CSF examination was normal. Postoperative MRI was suggestive of cerebellar and brain stem venous infarct. MR venogram revealed agenesis of right transverse sinus, hypoplastic right sigmoid sinus and internal jugular vein.

On the third postoperative day, the patient was intubated as he developed respiratory difficulty. Thereafter, he was managed conservatively as relatives did not consent for decompression of posterior fossa. Patient did not show improvement and died on the fifth postoperative day.

Superior petrosal vein is an important draining vein of the posterior fossa. It is a constant vein and drains a very large area, including the anterior parts of cerebellum, posterior part of medulla and mesencephalon, anterior part of the pons and medulla. The coagulation of this vein during surgery may result into venous infarct in the mentioned territory.

However, the collateral veins take over the function of the coagulated vein and this is possibly the reason that in many patients operated for CP angle mass, surgery is uneventful even if this vein is coagulated. It is difficult to assess the functional capacity of the collateral venous channels and the neurological outcome depends upon the extent and the speed at which they develop.

References


Accepted on 02-03-2006