Dangers of Endovascular Treatment of an Unusual Carotid Cavernous Fistula

We read with interest two recent articles (1, 2) describing arteriovenous fistula between the posterior communicating artery and the cavernous sinus, caused by head trauma. It is rare for the intradural cerebral arteries to have a communication with the cavernous sinus (3–5). We have also reported an arteriovenous fistula between the intradural internal carotid artery and the cavernous sinus; it had been first misdiagnosed as a direct carotid cavernous fistula (6). It was treated with transarterial balloon occlusion, which resulted in rupture of the pseudoaneurysm formed at the internal carotid artery.

We would like to point out that this type of fistula, either at the internal carotid artery or at the proximal portion of the posterior communicating artery, is formed by a pseudoaneurysm without the normal arterial wall structure. Although Kinugasa et al elegantly occluded the fistula, endovascular treatment to occlude the fistula, either with balloons or coils, can cause rupture of the pseudoaneurysm. Thus, the treatment of choice for this type of traumatic direct arteriovenous fistula is direct surgery to exclude the pseudoaneurysm and occlude the fistula (3–5). However, if this is not feasible, endovascular treatment might be the alternative. Even if endovascular occlusion of the fistula without exclusion of the pseudoaneurysm is carried out, there remains a chance that the pseudoaneurysm may bleed or enlarge to behave as a mass lesion.

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Reply

We thank Drs Komiyama and Yasui for their comments on our article. As they point out, we also thought about the possibility that this type of fistula might be formed by a pseudoaneurysm without the normal arterial wall structure. However, we could not find such a pseudoaneurysm on MR imaging before intravascular surgery. Thus, we only performed occlusion of the fistula by coils, as shown in our report. We think that the orifice of the fistula of this case existed on the lateral wall of the cavernous sinus.

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Reply

The central theme of our article was of early recognition of a traumatic pseudoaneurysm of the posterior communicating artery to cavernous sinus fistula. However, a considerable amount of debate existed regarding appropriate therapy. It was both our opinion and that of our neurosurgeons that surgical intervention with clipping of the posterior communicating artery anterior and posterior to the aneurysm was the most appropriate therapy. A major concern was the uncertainty of the structural integrity of the aneurysm wall, coupled with the potential additional stress of hemodynamic changes during the endovascular procedure. This case was also presented at the Interventional Neuroradiology Conference in Wyoming. There it was also the informal consensus of opinion to proceed surgically. The subsequent surgery was not easy and a clip could only be placed occluding the posterior communicating artery anterior to the aneurysm.

A second surgery was planned to clip the posterior aspect of the posterior communicating artery. The patient, who had some decrease in her massive exopthalmos, declined further intervention. Consideration was also given to closing the fistula from an endovascular venous approach with coiling of the cavernous sinus. Because after surgery there is now only one very small pathway through the aneurysm, it was expected that with cavernous sinus coiling the aneurysm would also soon thrombose. The patient has also declined this procedure, but has only minimal exophthalmos 17 months after surgery.

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Comment

I have read with great fascination the letter from Drs Komiyama and Yasui warning of the dangers of treating a carotid cavernous fistula where the connection occurs from the supraclinoid carotid or posterior communicating artery and the cavernous sinus. Their own experience with an acute connection involving the supraclinoid carotid artery that had massive bleeding after endovascular treatment is compelling evidence of the dangers involved.

In our own experience with the treatment of more than 300 direct carotid cavernous fistulas we have not yet encountered such an unusual case. Therefore, our comments are based on extrapolation of experiences gained with pseudoaneurysms in other locations. We would certainly agree that if an embolic device is delivered into an acute pseudoaneurysm, especially one involved in a fistula, that there is a great risk of enlargement and rupture of the pseudoaneurysm. If a pseudoaneurysm associated with a supraclinoid connection is discovered in the acute phase, surgical therapy may be indicated. Psuedoaneurysms involving the cavernous sinus with a typical carotid cavernous connection can be and have been treated with detachable balloons with great success, in our experience. Because of the risk of fatal subarachnoid hemorrhage, pseudoaneurysms should be occluded as soon as they are discovered (1).

If a period of time has passed since the initial injury and the discovery of the pseudoaneurysm, usually at least 4 to 6 weeks, then the wall of the pseudoaneurysm may have matured enough to permit the safe delivery of an embolic device without the fear of rupture. It is therefore not surprising that Kinugasa and his associates were successful at transvenous coil embolization of a fistula between the posterior communicating artery and the cavernous sinus that was discovered 1 month after the injury. Tytle and his colleagues reported a similar fistula in the same location presumed to be from an injury that occurred 31 years earlier and caused clinical symptoms for at least 13 years. Surgical treatment, which Dr Komiyama and his colleague recommend in their letter for the treatment for this condition, failed to obliterate the connection. I suspect that transvenous coil occlusion would have been successful in this unusual fistula. We share Drs Komiyama and Yasui's concern about endovascular treatment for acute pseudoaneurysms in this unusual location, and their opinion regarding the need for prompt closure with surgical intervention. If the false aneurysm escapes detection and a sufficient time has passed, then endovascular occlusion is a viable alternative.

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MR Methods of Measuring Changes in Brain and Cerebrospinal Fluid Volume with Age and Menstrual Cycle

We read with interest the paper by Blatter et al, "Quantitative Volumetric Analysis of Brain MR: Normative Database Spanning 5 Decades of Life" (1). This appears to corroborate our own work to quantify change of brain and cerebrospinal fluid volume with age (2) which, though based on MR, used a very different nonplanimetric approach (3). We also used intracranial cavity volume as a normalizing factor for brain size. We found mean brain loss per decade to be 1.6% in men and 0.5% in women. These figures are comparable to those of Blatter et al, 0.92% and 0.8%, respectively, for the range of 25 to 55 years.

We would raise one word of caution about any comparison of individual female volumetric measurements with the normative data Blatter et al provide. A study we performed (4) showed that total intracranial cerebrospinal fluid volume in premenopausal female subjects increased before menstruation by 11.5 mL over a mean midcycle value of 101.3 mL ($n = 20$, $P < .0001$). Bearing in mind that skull cavity volume does not change during this period, the implication based on the modified Monro-Kellie doctrine (5) is that the volume of the brain must decrease in a commensurate fashion to compensate.

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Reply

We appreciate Drs Condon and Hadley's pointing out the agreement between our recently reported normative database for brain volume and their previous work. We are also aware of another recent MR-based study (1) showing an age-regression slope for total brain volume nearly identical to what we reported. Having multiple MR-based volumetric studies that are not only internally consistent but also show a high degree of correlation with large autopsy