Subdural hemorrhage in the posterior fossa caused by a ruptured cavernous carotid artery aneurysm after a balloon occlusion test

Case report

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Given the relatively benign natural history of cavernous carotid artery aneurysms and based on anecdotal reports in the literature of subarachnoid hemorrhage (SAH) or subdural hemorrhage (SDH) from these aneurysms, observation is warranted and typically recommended. In this case report, the authors describe a woman who harbored a partially thrombosed, giant cavernous aneurysm that ruptured after she underwent a balloon occlusion test (BOT) and predominately led to an SDH. The authors believe that this occurrence is the first such report in the English literature. They discuss possible mechanisms for this event and the literature related to SAH or SDH from cavernous aneurysms, including why cavernous aneurysms cause such hemorrhages. The authors also recommend that attention be paid to such lesions regarding the possibility of aneurysmal rupture following a BOT.

KEY WORDS • aneurysm • cavernous carotid artery • subdural hemorrhage

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Case Report

History and Examination. This 33-year-old woman was examined at another hospital because she had sudden-onset right-sided palpebral ptosis, diplopia, and right temporal and retroocular pain of moderate intensity. A CT scan revealed a heterogeneous, approximately 3-cm ellipsoid mass in the right middle cranial fossa that was compatible with a giant, partially thrombosed cavernous CA aneurysm (Fig. 1). On admission to our institution, the patient had right-sided palpebral ptosis and a mydriatic, hyporeactive 6-mm right pupil. Her extraocular movements were severely af-

Abbreviations used in this paper: BOT = balloon occlusion test; CA = carotid artery; CT = computed tomography; HMPAO = hexamethylpropyleneamine oxime; ICA = internal CA; ICP = intracranial pressure; SAH = subarachnoid hemorrhage; SDH = subdural hemorrhage; SPECT = single-photon emission computed tomography.
fected in all directions, suggesting dysfunction of the third, fourth, and sixth cranial nerves. Her right corneal reflex was diminished, her visual acuity was 20/20, and her facial sensation was decreased on the right side in all three trigeminal divisions (V1–V3). No proptosis, chemosis, or bruit was identified. The patient reported having three episodes of transient double vision within the 2 months preceding hospital admission.

A selective cerebral angiogram revealed a 26 × 15 × 14-mm aneurysm of the right ICA, which arose near the petrocavernous junction. The small size of the lesion, compared with its 3-cm diameter on CT scans obtained at that time confirmed a partially thrombosed component (Fig. 2). No additional aneurysm or vascular abnormality was identified. Of clinical importance for eventual treatment were the small posterior communicating arteries and a hypoplastic right A1 segment of the anterior cerebral artery.

Operations. A BOT of the right ICA (including full anticoagulation, which left an activated clotting time > 300 seconds) with a hypotensive challenge was performed to assess the patient’s collateral circulation and clinical tolerance to a possible therapeutic CA occlusion, in which the ICA segment harboring the aneurysm could be trapped endovascularly. A radioisotope (99Tc HMPAO) was injected before the hypotensive challenge to allow evaluation by SPECT. The patient clinically tolerated these procedures. A left ICA injection seen on angiography during balloon occlusion demonstrated cross-filling from the left anterior cerebral artery into the smaller right A1 segment with incomplete opacification of the middle cerebral artery and poor development of the venous phase. After 32 minutes of occlusion, the nondetachable silicone balloon was deflated and the patient was returned to the neurosurgical intensive care unit.

Three hours later, the patient experienced a sudden-onset headache and became unresponsive. Emergency intubation and mechanical ventilation were required and the patient was resuscitated. An emergency CT scan of the woman’s head revealed a subdural hematoma in the right tentorium, retroclival region, and upper cervical spine as well as a minimal SAH in the interpeduncular cistern (Fig. 3). The right ICA was occluded with coils on an emergency basis to trap the arterial segment containing the aneurysm. On angiography, the configuration of the aneurysm had changed and now demonstrated a rounded base with enlargement of the bleb (Fig. 4), which had been oriented dorsomedially on the original angiogram.

A nondetachable silicone balloon was advanced into the petrous portion of the ICA. Under balloon inflation, coils were placed distal and proximal to the aneurysm ostium, as well as in the cervicopetrosal junction and petrous portions of the ICA. The contrast agent, which had puddled within the aneurysm, diffused into a more dorsal location. This location was compatible with that of thrombus, perhaps related to clotted portions of the aneurysm and/or blood released at aneurysmal rupture, and the tentorial subdural location.

After coil occlusion, a right CA angiogram did not demonstrate flow in the external CA, ICA opacification, or retrograde flow in the ophthalmic artery. A left CA angiogram demonstrated cross-filling through the anterior communicating artery from the left into the right ICA distribution and occlusion of the right posterior parietal cortical branch. A 2- to 3-mm displacement of the internal cerebral...
vein over the convexity of the right hemisphere was suggestive of an SDH. Injection of the left vertebral artery revealed opacification of the right suprachoinoid ICA via a right posterior communicating artery (1 mm) with a hemodiluted flow into the distribution of the right middle cerebral artery. A right posterior parietal cortical branch filling defect was again identified. Postprocedure, a CT scan revealed stable-sized tentorial, perimedullary, and upper cervical subdural hematomas with a right-to-left shift of 4 mm at the level of the septum pellucidum and a slight asymmetric effacement of the right lateral ventricle. A 2-cm area of hypodensity in the right anterotateral parietal region and a loss of gray-to-white matter differentiation were also identified.

Postoperative Course. While intubation and sedation were maintained in the patient, a fiberoptic parenchymal ICP monitor inserted into the right frontal lobe revealed an opening pressure of 32 mm Hg. Aggressive ICP management was instituted, along with hypotensive-hypovolemic-hemodilution therapy and anticoagulation. Although a CT scan of the head obtained 24 hours after CA occlusion showed no significant change in the size of the subdural hematoma and its mass effect, we noted increased definition of an area of hypodensity measuring 1 to 2 cm suggesting infarction in the anterior parietal region. A $^{99}$Tc HMPAO SPECT scan revealed findings at the cranial base that were compatible with the therapeutic right ICA occlusion, a focal right parietal area with absence of radionuclide uptake compatible with the known infarction, and no other ischemic area. The cerebellar uptake of the tracer was slightly asymmetrical; a relatively decreased uptake on the left side indicated left crossed cerebellar diaschisis.

The patient’s treatment remained unchanged for the next 48 hours. Attempts at weaning her from sedatives resulted in a significant elevation in ICP. Cerebral perfusion pressure was maintained between 75 to 100 mm Hg primarily by administering pressor agents. Serial CT scans of the head did not demonstrate any significant change or new lesion. Four days after coil occlusion, a follow-up SPECT scan revealed no change in the size of the right parietal infarction, no new area of ischemia, and resolution of the crossed cerebellar diaschisis. All sedatives, pressor agents, and mechanical ventilation as well as ICP monitoring were successfully discontinued within the next 48 hours. When the patient awakened, a left pronator drift was noted. The right palpebral ptosis and paresis of the oculomotor muscles remained unchanged from the time of admission. No new visual deficit was noticed in the right eye and the right pupil remained mydriatic with a weak photomotor response. The patient’s
Based on our review

Nevertheless, these cases were reported before intracavernous aneurysms were recognized to lead to SAH. Linskey and colleagues, in a similar dilemma posed by the diagnostic accuracy of extension of cavernous aneurysms, reported a 7% incidence of SAH after rupture of an intracavernous CA aneurysm. However, these authors stated that the occurrence of an SAH or SDH depends on extension of at least part of the sac or neck of the aneurysm through the dura mater of the cavernous sinus. Only one of several intracavernous aneurysms reported to lead to SAH proved to be purely intracavernous. Most “totally intracavernous” aneurysms presented with subarachnoid extension. Nevertheless, these cases were reported before Al-Rodhan and coworkers recognized that these particular aneurysms (that is, transitional aneurysms) have an intradural component and, therefore, bear a risk of SAH equal to that of other intradural aneurysms.

The risk of SAH from “true” totally intracavernous aneurysms should be less than reported. Even minimal insinuation of these aneurysms into the subarachnoid spaces disqualifies a classification as totally cavernous; thus they should be classified as “transitional” aneurysms. In our patient, an analysis of the change in aneurysm shape between the admission CT scan and angiogram led us to believe that the aneurysm had eroded through the skull base at the petroclival region and insinuated into the posterior fossa and sella turcica. Printed with permission from the Mayfield Clinic.

trigeminal pain disappeared. We documented hypesthesia on the V1 and V2 distribution, which was greater in V1, and a weakened right corneal reflex.

On admission to the rehabilitation service, the patient underwent physical therapy. Six months after her initial admission to the hospital, she had resumed normal activities and displayed no motor deficits. She had a residual mild horizontal diplopia at the extreme right lateral gaze, symmetrical pupils, palpebral elevation, decreased left corneal reflex, and decreased sensation in the right V1 distribution.

Discussion

Rupture of an intracavernous aneurysm into the subarachnoid or subdural spaces is rare. Based on our review of the English language literature, we found that two of three cavernous CA aneurysms reported to present as subdural hematomas were fatal. Linskey and colleagues reported a 7% incidence of SAH after rupture of an intracavernous CA aneurysm. However, these authors stated that the occurrence of an SAH or SDH depends on extension of at least part of the sac or neck of the aneurysm through the dura mater of the cavernous sinus. Only one of several intracavernous aneurysms reported to lead to SAH proved to be purely intracavernous. Most “totally intracavernous” aneurysms presented with subarachnoid extension. Nevertheless, these cases were reported before Al-Rodhan and coworkers recognized that these particular aneurysms (that is, transitional aneurysms) have an intradural component and, therefore, bear a risk of SAH equal to that of other intradural aneurysms.

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True totally intracavernous aneurysms have virtually no possible way of causing SAH or SDH. For such a hemorrhage to originate from an intracavernous aneurysm, extension of the lesion into the subarachnoid space is a sine qua non. In addition to transitional aneurysms that transgress the distal dural ring and enter the subarachnoid space, an aneurysm may erode bone and create an alternate route into the subarachnoid space (for example, the posterior cranial fossa). The real challenge remains to determine unequivocally the true totally intracavernous location of these aneurysms. Although bone erosion can be observed on CT scans, documentation of aneurysm penetration through the walls of the cavernous sinus is truly challenging using current diagnostic technology, as depicted in the case report published by Gottfried, et al. A similar dilemma is posed by the diagnostic accuracy of extension of cavernous aneurysms beyond the distal dural ring. Angiographic signs, such as “waisting,” can be used as an indirect indication of intradural extension of an aneurysm. Newer diagnostic methods, such as CT angiography, three-dimensional angiography, and the refinement of magnetic resonance imaging techniques may help improve the diagnosis of intracavernous aneurysms.

Conclusions

A complete understanding of the extension of aneurysms that involve the intracranial extradural ICA is mandatory for appropriate case management. The term “cavernous aneurysms” should be strictly applied to those lesions totally contained within the boundaries of the cavernous sinus. For all others that extend beyond those boundaries, even though they may be primarily intracavernous, the term “transitional aneurysm” should be used. We also propose extending the term “transitional” to all aneurysms that, regardless of their origin along the intracranial extradural ICA, have the possibility of an intradural component. Clarification of this terminology implies a higher risk of SAH or SDH, thus prompting a different therapeutic strategy. Finally, we recommend that special attention be paid to the possibility of aneurysmal rupture following BOT of such lesions.

References


Fig. 5. A: A CT angiogram demonstrating the flow of the circle of Willis above the level of the posterior communicating arteries, with excellent opacification of the branches of the right supraclinoid ICA. Note the enlarged aneurysm mass with artifact from the coil mass. B: A CT scan (bone window) showing bone erosion of the petrous and sphenoid bones. Note the expansile mass from the aneurysm filling the cavernous sinus region and insinuating into the posterior fossa and sella turcica. Printed with permission from the Mayfield Clinic.
Subdural hemorrhage after balloon test occlusion


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