Giant posterior communicating artery aneurysm presenting as third ventricle mass with obstructive hydrocephalus

Case report

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The case of a giant posterior communicating artery (PCoA) aneurysm is reported in which the initial presentation was coma secondary to obstructive hydrocephalus. The primary radiological diagnosis was a probable craniopharyngioma. A cerebral angiogram revealed a partially thrombosed giant PCoA aneurysm on the right side. The patient underwent pterional craniotomy with aneurysm clipping and thrombectomy to relieve mass effect, and has made a good recovery. Review of the literature documents that giant PCoA aneurysms are rare. This is believed to be the first reported case of a PCoA aneurysm presenting as a third ventricle mass with obstructive hydrocephalus. The magnetic resonance imaging characteristics of those third ventricle masses that mandate vascular workup are discussed.

KEY WORDS • giant aneurysm • magnetic resonance imaging • obstructive hydrocephalus • differential diagnosis

Giant posterior communicating artery (PCoA) aneurysms are rare. We describe what we believe is the first reported case of a PCoA aneurysm presenting as a third ventricle mass with obstructive hydrocephalus.

Case Report

This 60-year-old woman complained of worsening headaches for 3 weeks and of nausea and repeated emesis for 1 week prior to her initial presentation. She was taken to the emergency room of a local hospital when she became very lethargic and confused. A basic computed tomography (CT) scan of the brain showed severe obstructive hydrocephalus and a mixed-density mass in the third ventricle. By the time she had been transferred to a local facility with neurosurgical capabilities, she had progressed to coma with decerebrate posturing and had bilaterally fixed and dilated pupils. Emergency bifrontal ventriculostomies were performed and she recovered consciousness thereafter. The ventriculostomies were converted to bilateral ventriculoperitoneal shunts 2 days later. The patient continued to improve and became alert but was confused and had a moderate residual right third nerve palsy and persistent bilateral Babinski signs.

Magnetic resonance (MR) imaging with gadolinium enhancement was performed to further evaluate the third ventricle mass (Fig. 1). The lesion measured 4 × 3 × 3 cm and filled the entire third ventricle. The mass was heterogeneous with high- and low-signal areas, and had a peripheral hypointense rim; there was minimal peripheral gadolinium enhancement. The lesion appeared to be entirely suprasellar and retrochiasmatic. The radiologist from the referring institution reported a differential diagnosis of third ventricle tumors including craniopharyngioma, hypothalamic glioma, and atypical colloid cyst with blood products. Aneurysm was not mentioned in the differential diagnosis.

Craniotomy for tumor resection was recommended by the neurosurgeon who initially treated the patient, to be undertaken after adequate time had been allowed for neurological recovery, and she was discharged home in the care of her family 1 week after the shunts were placed. She continued to improve and had nearly complete resolution of neurological deficits. The mild
residual third nerve palsy persisted, but she was fully ambulatory without assistance, although she easily became fatigued. She was fully oriented to her surroundings and had no speech impairment. Detailed neuropsychiatric studies were not performed; however, according to her family and findings from routine examination, she was mildly impaired with respect to memory and cognition. Laboratory workup of pituitary function was entirely normal. She was deemed sufficiently stable to tolerate craniotomy and was referred to our institution for extirpation of the lesion.

**Examination.** Review of the patient’s MR images revealed high signal intensity peripherally within the lesion and in direct proximity of the mass to the flow void areas of the basilar artery and the right internal carotid artery (ICA) (Fig. 1). A basilar apex or carotid artery bifurcation aneurysm was thus considered as a potential diagnosis. Cerebral angiography was scheduled in order to rule out a giant aneurysm. A complete four-vessel cut film and a digital subtraction cerebral angiogram revealed an aneurysm arising from the proximal right supraclinoid ICA (Fig. 2). With oblique subtraction views, the aneurysm was thought to arise from the origin of the PCoA, although the artery itself could not be visualized. It was apparent that the majority of the aneurysm was thrombosed because none of the mass in the third ventricle region filled with contrast medium. An aneurysmal neck appropriate for surgical application of an aneurysm clip was apparent on angiography (Fig. 2 right).

**Operation.** The patient was taken to surgery and placed under general endotracheal anesthesia. Moderate electroencephalographic burst suppression with pentobarbital was used for cerebral protection. A standard right pterional craniotomy was performed, with moderate dissection of the sylvian fissure being necessary for exposure of the carotid bifurcation and the aneurysmal neck (Fig. 3 left). The aneurysm was found to arise from the site where the PCoA arises from the lateral proximal ICA. The PCoA itself was obliterated by the giant aneurysm. The ICA was diseased and moderately dilated in appearance. The anterior choroidal, middle cerebral, and anterior cerebral arteries were identified in their normal locations. The aneurysmal neck was approximately 12 mm in diameter and was successfully clipped with two sequentially placed 15-mm Sugita aneurysm clips (Fig. 3 right). The neck began by pointing laterally from the ICA, but then made a sharp medial turn and the bulk of the giant thrombosed mass was directed medial and superior to the ICA, then expanded retrochiasmatically between the optic tracts and filled the third ventricle. After clip application, the aneurysm was punctured with a needle and drained of blood. Complete occlusion had been achieved. A thromboendarterectomy was then performed to debulk the mass with ultrasonic aspiration and forceps.

**Postoperative Course.** The patient had an uneventful postoperative course without complication. Cerebral angiography documented complete obliteration of the aneurysm (Fig. 4). The patient was transferred to a neurorehabilitation unit 1 week after surgery, and has since been discharged home with only mild cognitive and memory impairment that is unchanged from her preoperative status.
Giant PCoA aneurysm presenting as third ventricle mass

Fig. 3. *Left:* Intraoperative photograph taken through the operating microscope revealing the internal carotid artery (ICA) (1) and aneurysmal neck (2) at the site of the posterior communicating artery origin. The anterior choroidal artery (3) and the bifurcation of the ICA into the middle cerebral (4) and anterior cerebral (5) arteries are seen distal to the aneurysmal neck. The massive thrombosed aneurysmal dome (6) is seen extending medially below the optic chiasm (7). *Right:* Intraoperative photograph showing the ICA after aneurysm clip application. Note the preservation of the ICA lumen caliber and complete obliteration of the aneurysmal neck by the two sequential clips.

Fig. 4. Postoperative angiograms, anteroposterior (*left*) and lateral (*right*) projections, demonstrating no residual aneurysmal lumen. There is no spasm or other evidence of complication.

Discussion

Giant intracranial aneurysms are those with a diameter of at least 2.5 cm. They often present as large intracranial lesions causing signs and symptoms directly or indirectly attributable to the mass effect. The reported incidence of aneurysms presenting as mass lesions rather than with subarachnoid hemorrhage (SAH) varies in different studies from 23% to 64%. Aneurysms may also present with primarily intraventricular hemorrhage without SAH, but this is rare. Giant intracranial aneurysms have presented with cervicomедullary compressive symptoms similar to foramen magnum tumors, abducens nerve paralysis, homonymous hemianopsia, dysphagia, focal seizures, posterior fossa mass effects, and a frontal lobe syndrome. Giant aneurysms have also been noted to simulate arteriovenous malformations (AVM’s) and brain tumors on CT and MR imaging. Several cases have been reported in which third ventricle masses consisted of domes of giant aneurysms from the basilar and anterior communicating arteries. We could find no previous case in the literature of a giant aneurysm arising from the PCoA appearing as a third ventricle tumor and causing hydrocephalus.

Neuroimaging

Cross-sectional imaging modalities can usually be used to distinguish giant intracranial aneurysms from other mass lesions. The appearance demonstrated on CT is characteristic, consisting of a well-circumscribed round or oval mass. The presence of thrombus determines the central attenuation characteristics, which may range from iso- to hyperdense. A peripheral zone of increased attenuation is frequently seen due to mural thrombus. After administration of contrast material, CT scans show intense enhancement of the residual aneurysmal lumen, which rapidly declines after cessation of contrast medium injection. Despite this characteristic appearance, however, giant intracranial aneurysms may occasionally simulate brain neoplasms on CT. When the less common presentation of SAH occurs with giant aneurysms, CT is the modality of choice for the detection of extra-axial blood; it is also superior for the depiction of mural calcification associated with an aneurysm.

Magnetic resonance imaging provides superior characterization of thrombus, flow phenomena, and the relationship of the lesion to the parent vessel and other adjacent structures. In a patent lumen, a homogeneous signal-void area characterizes a region of rapid flow. Turbulence and slow-flowing blood give
rise to the heterogeneous signal which varies with pulse sequence. Thrombus within the aneurysm characteristically demonstrates a lamellated appearance with alternating zones of increased and decreased signal. The thrombus is generally deposited circumferentially within the aneurysm and will vary in signal characteristics depending on its stage of evolution. Although a giant intracranial aneurysm has been reported to simulate an AVM on MR imaging, the MR appearance is usually fairly specific. Magnetic resonance angiography increases the specificity of MR imaging, by providing information regarding flow within the residual lumen. Contrast angiography continues to be an integral component of the pre- and postoperative evaluation of patients with giant aneurysms. Although MR angiography has become an excellent screening modality, conventional angiography provides the most reliable delineation of the aneurysmal lumen and its relationship to the parent vessel and adjacent arteries.

Conclusions

Clearly, giant intracranial aneurysms may present symptomatically and radiographically as brain tumors. Occasionally, their true pathology is revealed upon intraoperative biopsy, the undertaking of which has had disastrous consequences. Had we approached our case through a transcrallosal route for a third ventricle tumor, it would have been impossible to achieve adequate control of the aneurysmal bleeding without destroying the floor of the third ventricle and hypothalamus. It is mandatory that aneurysm be considered in the differential diagnosis of third ventricle tumors. Vascular imaging studies should be obtained to exclude the presence of an aneurysm unless the possibility of aneurysm can confidently be excluded. The presence of signal flow-void areas on MR imaging in regions directly adjacent to a third ventricle mass, as was present in this case, makes further investigation imperative in order to rule out an aneurysm.

Acknowledgments

The authors thank Dana Evans, editor; Mar's Schornak, medical illustrator; Pamela A. Smith, medical photographer; and the editorial staff at the Barrow Neurological Institute for assistance in the preparation of this manuscript.

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Manuscript received July 13, 1993.
Accepted in final form November 23, 1993.
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