Ruptured Anterior Spinal Artery Aneurysm: Repair via a Posterior Approach

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A case of aneurysm at the junction of the vertebrospinal and anterior spinal arteries at the level of C-1 is presented. There was severe intracranial subarachnoid hemorrhage with subsequent right hemiplegia and 6th nerve palsies. Angiograms showed an anomalous arrangement of the arteries, but no arteriovenous malformation was seen. The lesion was repaired via a posterior approach. Anatomical and technical considerations of this approach are discussed. A review of the pertinent literature is presented. (Neurosurgery 10:626–630, 1982)

Key words: Angiography, Anterior spinal artery, Posterior approach, Saccular aneurysm, Subarachnoid hemorrhage

Subarachnoid hemorrhage, with the sudden onset of severe headache with or without altered sensorium or focal neurological deficit, is a common presentation for intracranial saccular aneurysms. Rarely does subarachnoid hemorrhage from lesions of the spinal cord present with primarily intracranial manifestations (6, 12). A case of ruptured aneurysm at the junction of the vertebrospinal and the anterior spinal arteries at the level of C-1 presenting in such a manner was recently encountered. The aneurysm was successfully obliterated via a posterior approach.

CASE REPORT

This 30-year-old, right-handed woman developed generalized headache without associated symptoms 3 days before hospitalization on January 20, 1980. On the evening of admission while riding in an automobile, she had the abrupt onset of severe headache, neck pain, nausea, and vomiting. She became confused and was brought to the emergency room of the Munson Medical Center in Traverse City, Michigan.

There was no history of trauma, previous illness, or operation. Her family history included diabetes mellitus.

Her blood pressure was 195/100 mm Hg, her pulse was 65/minute, and her respirations were 16/minute and regular. She was confused and lethargic. When stimulated, she would follow simple commands and mutter a few words. There was marked nuchal rigidity. No cranial or cervical bruit was heard. Her pupils were 3 mm, equal, and reactive to direct and consensual light. The optic discs were flat. Spontaneous venous pulsations were absent. There were no retinal hemorrhages. Her extraocular movements were full, with nystagmus on lateral gaze bilaterally. There was a right arm drift, increased tone in both lower extremities, right-sided hyper-reflexia, and bilateral Babinski signs.

A complete blood count, serum electrolyte determinations, a clotting profile, and determinations of blood urea nitrogen, creatinine, and glucose were all normal. Computed tomography of the head without contrast enhancement (Fig. 1) showed a radiopacity consistent with clotted blood in the basilar cisterns, sylvian fissures, 4th ventricle, 3rd ventricle, and anterior horns of the lateral ventricles.

The diagnosis was subarachnoid hemorrhage, Clinical Grade III (9). Intensive medical therapy was initiated, including anticonvulsant, steroid, and antifibrinolytic medication; fluid restriction; and observation in an intensive care unit. Because of hypertension in the range of 200/120 mm Hg, a sodium

nitroprusside infusion was administered to maintain her blood pressure in the range of 150-180/90-100.

Three hours after admission, the patient had a generalized convulsion accompanied by 45 seconds of apnea. Intubation was performed for respiratory support. The coma deepened until she responded only semipurposefully to noxious stimuli.

Over the ensuing days, gradual improvement allowed extubation and movement became spontaneously purposeful. On the 9th day of hospitalization, right facial weakness and a right hemiparesis were noted. By the 10th day, dysarthric speech, bilateral 6th nerve palsies, and a dense right hemiplegia were noted. Her serum glucose value was 600 mg/ml and her serum sodium level was 172 meq/litre. Her blood pressure was unchanged. Hydration and potassium and insulin therapy were initiated. With correction of these metabolic parameters, slow neurological improvement was seen over 2 weeks. By the 30th day after hemorrhage, the patient had only a mild right hemiparesis and slightly decreased abduction of the left eye (Clinical Grade IA (9)).

Angiography at this time showed a normal anterior circulation. Injection of the vertebral arteries showed a dominant left vertebral system and a small saccular collection of contrast material filling from ramifications of the anterior spinal artery at the level of C-1 (Figs. 2 and 3). The right vertebral artery terminated in the right posterior inferior cerebellar artery (PICA) without contributing to the basilar artery. There was a small branch on the right to the anterior spinal cord that did not fill the aneurysmal sac.

Because of the unusual location of this aneurysm, the patient was transferred to The Ohio State University Hospital for additional neurosurgical consultation on March 1, 1980. At that time there was no headache, nuchal rigidity, or oculomotor, speech, or intellectual deficit. The only finding was a resolving right hemiparesis with 4/5 strength in all muscle groups and right Babinski and Hoffmann signs. There were no sensory deficits. Operation was carried out on March 11, 1980, Posthemorrhage Day 50.

With the patient under endotracheal anesthesia in the forward-oblique position, left side up, a suboccipital craniectomy and upper cervical laminectomy was performed. Under the microscope the dura mater was opened, showing yellowish, discolored, thickened arachnoid and organized clot in the subarachnoid space. Using intraoperative fluoroscopy with image amplification, the precise level of the aneurysm was identified. A tantalum clip was applied to the 1st denticulate ligament on the left, which then was transected. A film was

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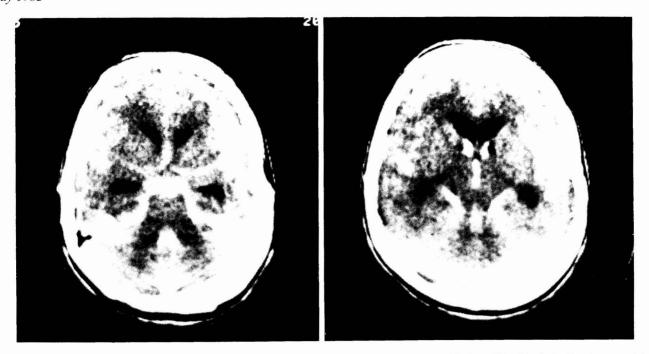


Fig. 1. Computed tomography of the head without contrast enhancement: radiopacity consistent with clotted blood in the basilar cisterns, sylvian fissures, 4th ventricle, 3rd ventricle, and anterior horns of the lateral ventricles.

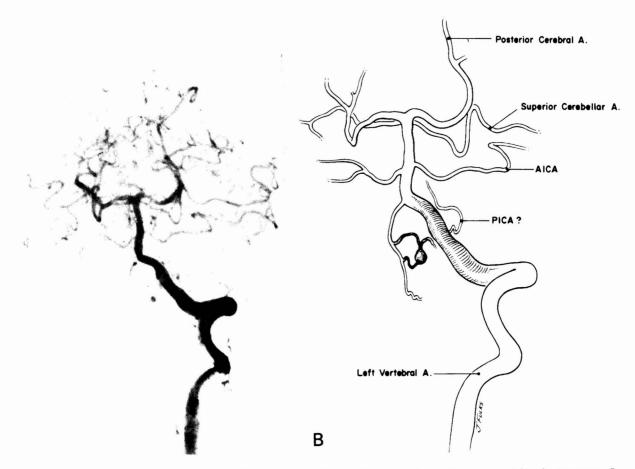


Fig. 2. A, left vertebral angiogram in the arterial phase: filling of the anterior spinal artery with ramifications supplying the aneurysm. B, schematic diagram of A. AICA, anterior inferior cerebellar artery; PICA, posterior inferior cerebellar artery; A., artery.

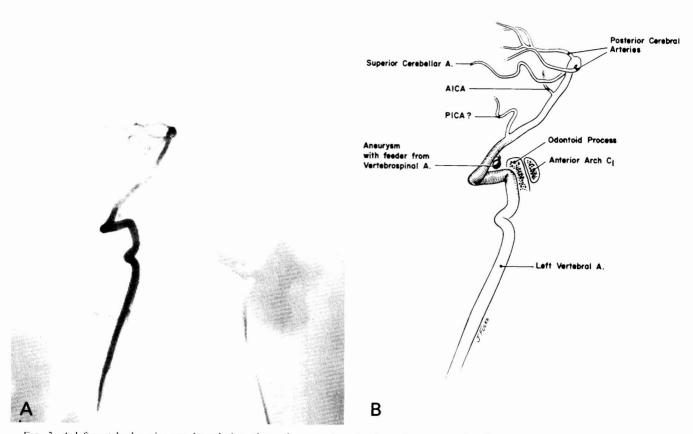


Fig. 3. A, left vertebral angiogram, lateral view, shows the aneurysm. B, schematic representation shows the relation of the aneurysm to the odontoid process and the anterior arch of C-1. (For abbreviations, see the legend to Fig. 2.)

taken. By comparing the location of the tantalum clip to bony landmarks and then to the site of the aneurysm on preoperative angiograms, the level of the aneurysm could be identified. The anterior rootlets of C-2 and the spinal accessory nerve were identified. One accessory nerve rootlet was transected and used for traction.

Dissecting on the anterolateral surface of the spinal cord near the known location of the aneurysm, the surgeon removed dense arachnoid and organizing clot. Ultimately, a bit of the aneurysm was seen. Its dome was adherent to the ventral dura mater, limiting permissible rotation of the spinal cord. A variangled clip was then arranged, again using fluoroscopic localization, to pass anterior to the spinal cord, one blade above and one blade below the barely identifiable aneurysmal neck. The clip was then allowed to close, its blades in contact with the spinal cord. Finally, dissection of the dome was completed and it was collapsed with a needle puncture.

The postoperative course was without incident; there were no neurological sequelae of the procedure and the mild right hemiparesis remained unchanged from the preoperative state. Left vertebral angiography on the 9th postoperative day (Posthemorrhage Day 59) showed good placement of the clip with no filling of the aneurysm (Fig. 4).

DISCUSSION

Saccular aneurysms of the spinal cord are exceedingly rare (5, 13). The literature includes only one case of isolated spinal

cord aneurysm without associated angiomatous malformation, a 51-year-old man with the onset of headache, neck pain, and confusion who died without diagnosis (6). Autopsy showed a $1.2\text{-}\times0.7\text{-}\mathrm{cm}$ aneurysm of the posterior spinal artery lying at the medullospinal junction. Hopkins et al. reported the case of a 27-year-old man with the acute onset of headache, nuchal rigidity, and loss of consciousness, preceded by 2 months of progressive right-sided weakness (8). At operation, a $1\text{-}\times1\text{-}\times0.5\text{-}\mathrm{cm}$ aneurysm on the right radicular artery at C-4 was clipped. A small venous angioma was found to be in close proximity. These cases demonstrate that subarachnoid hemorrhage from high cervical lesions can present with intracranial subarachnoid blood.

More commonly, aneurysms of the spinal cord are associated with angiomatous malformation (1, 4, 10, 12). Such patients typically present with back pain, with or without a radicular component, subarachnoid hemorrhage, or progressive paraparesis. Herdt et al. stated that only 3 of their 50 cases of spinal cord vascular anomalies presented with subarachnoid hemorrhage (7). These 3 patients with hemorrhage were all documented to have aneurysms using strict angiographic criteria (an aneurysmal sac connected to an arterial channel that fills in the early arterial phases).

H. W. Pia in a review of aneurysms of the posterior intracranial circulation described aneurysms of the vertebral artery as occurring primarily at the junction of the vertebral artery (VA) and the PICA (60%). They also occur where medullary branches or the anterior spinal artery leave the VA. Drake.



Fig. 4. Postoperative left lateral vertebral angiogram shows nonfilling of the aneurysm, the location of the variangled clip, and the presence of several tantalum clips in the posterior fossa.

in his operative approach to VA aneurysms, advocates the transsoral, transclival approach to those aneurysms that lie over the lower one-third of the clivus and are within 1.0 cm of the midline (2, 3). Because of the relative immobility of the brain stem, only the more laterally situated aneurysms have been considered accessible via the posterior approach.

In our case, consideration was given to the anterior approach, but we performed the posterior approach for several reasons. First, it provides an easier exposure of the brain stem and spinal cord. Second, postoperative morbidity, especially the chance of infection, cerebrospinal fluid leakage, or wound dehiscence, is less. Third, the tracheostomy required for the anterior approach is avoided by a posterior approach.

We recognized preoperatively the difficulty in exposing the aneurysmal neck from behind. However, we thought that the aneurysm was lateral enough that its neck might be visualized by rotation of the medullospinal junction. If, as actually proved to be the case, the neck could not be seen, we hoped that fluoroscopic localization (as described) would allow blind clipping of the aneurysm. The feasibility of the posterior approach and the higher morbidity of the anterior approach were important factors in our decision.

CONCLUSION

A case of aneurysm at the junction of the vertebrospinal and anterior spinal arteries at the level of C-1 is presented. The symptoms were those of severe intracranial subarachnoid hemorrhage with subsequent right hemiplegia and bilateral 6th nerve palsies. There was an anomalous arrangement of the arteries, but no arteriovenous malformation. A posterior approach with a partially blind, fluoroscopically assisted clipping of the aneurysm was successful.

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COMMENT

Moore et al. present an unusual case of aneurysmal dilation originating from the vasculature of the ventral aspect of the cervical-medullary junction. In contrast with previously reported aneurysms in this region, this aneurysm did not arise from the vertebral or posterior inferior cerebellar arteries, but from a smaller vessel. The fusiform dilation of this more distal vessel suggests a mycotic origin but, unfortunately, the surgical exposure did not allow close examination of the lesion or biopsy. The authors, however, are to be commended for an inventive approach to a lesion on the ventral surface at the cervical-medullary junction.

Although the surgical approach used in this case did give enough exposure to place a clip about the aneurysmal sac, the anterior transcervical approach to the upper cervical region described by Stevenson et al. should have been considered (1). Although the transcervical dissection is tedious, it provides good exposure of the ventral superior spinal cord and the caudal medullary region, the region of concern in this case.

It should also be mentioned that the early CT scan did demonstrate acute hydrocephalus. Although ventriculostomy placement is not recommended routinely for the moderate hydrocephalus that frequently accompanies subarachnoid hemorrhage, the neurological status of this patient might have improved more rapidly after hemorrhage and the interval to surgery might have been shortened if her acute hydrocephalus had been treated. I recently cared for two patients with ruptured

PICA aneurysms who presented in extremis and who had scans similar to that of the present patient. The CT scans showed blood in the anterior horns of the lateral ventricles and filling the 3rd and 4th ventricles as well as showing signs of acute hydrocephalus. Placement of bilateral ventriculostomies with care not to reduce intracranial pressure too rapidly resulted in rapid neurological improvement in both cases.

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