Anterior Spinal Artery Aneurysm Presenting as a Subarachnoid Hemorrhage

FREDERICK M. VINCENT, M.D.

SUMMARY A woman who presented with a subarachnoid hemorrhage secondary to a ruptured anterior spinal artery aneurysm of the spinal cord is reported. While this is the first report of a ruptured aneurysm of the anterior spinal artery with angiographic confirmation, it emphasizes that occasionally subarachnoid hemorrhage may be secondary to ruptured spinal artery aneurysms.

ANEURYSMS involving the arteries of the spinal cord are uncommon; 5 have been reported unassociated with arteriovenous malformations. In 2 the aneurysms arose from the anterior spinal artery in the thoracic cord.14 The clinical and radiographic features are reported of an anterior spinal artery aneurysm of the high cervical cord which was found in a normotensive woman with subarachnoid hemorrhage. The angiographic features of an aneurysm in this location have not been reported previously.

Three days prior to hospital admission a 30-year-old right-handed woman had developed a generalized headache without associated symptoms. The evening of admission she was riding in a car and suddenly developed severe head and neck pain associated with nausea, vomiting, confusion, and weakness in the legs. She was not taking medications and had not been previously ill. There was no trauma and her history was unremarkable. There was no family history of stroke or hypertension, but there was a strong family history of diabetes mellitus.

On physical examination her blood pressure was 95/100 mm Hg, pulse rate was 65, and respirations were 16/minute and regular. Her general examination was normal except for the presence of nuchal rigidity. On neurologic examination she was confused and lethargic, and would only mutter a few words. She could follow basic commands when stimulated. Pupillary diameter was 3 mm, and optic discs were flat; spontaneous venous pulsations were absent and there were no pre-retinal hemorrhages. Extraocular movements were full with nystagmus on lateral gaze bilaterally. A right pronator drift and external rotation of the right lower extremity were evident. She could not cooperate for formal motor and cerebellar tests. Tone was increased in both lower extremities, right greater than left, and tendon reflexes were increased on the right with bilateral Babinski signs. There were no cranial or cervical bruits.

Computerized tomography (CT) of the head performed without contrast enhancement revealed blood in the basilar cisterns and Sylvian fissures; the 4th ventricle was filled with blood, and a small amount of blood was present in the frontal horns of the lateral ventricles (fig. 1). Complete blood count, serum electrolytes, clotting profile, BUN, and glucose determinations were normal. She was transferred to the intensive care unit where her blood pressure was found to be 220/120 mm Hg. A sodium nitroprusside infusion was started, and her blood pressure was maintained in the 150-180/90-100 mm Hg range. Phenobarbital, dexamethasone, and epsilon amino-caproic acid (36 g/day) were administered, and she was placed on fluid restriction.

Three hours after admission to the hospital she had a generalized seizure followed by a 45-second period of apnea; endotracheal intubation was performed, but assisted ventilation was not required. The following morning she was less responsive, and diminished abduction of the left eye was evident. Within 48 hours her blood pressure became normal and nitroprusside was discontinued. On the third hospital day she was semicomatose, and would only respond to noxious stimulation. Later her mentation slowly improved, but on her 9th hospital day mild right-sided weakness was evident (including her face). The next day her speech was dysarthric, and bilateral 6th nerve palsies were evident, together with a dense right hemiplegia. Her blood glucose level rose to 600 mg/ml; sodium concentration was 172 meq/l. She was febrile and a urinary tract infection secondary to an enterococcus was discovered. She was treated for a non-ketotic hyperosmolar state with intravenous fluids and in-

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SPINAL ARTERY ANEURYSM AND SAH/Vincent

FIGURE 2. Left vertebral arteriogram in AP (A) and lateral (B) projections (subtraction technique) reveals a 4 mm aneurysm (arrow) arising from the proximal anterior spinal artery (arrowheads).

sulin. Her urinary tract infection was treated with ampicillin. Within 5 days her glucose and sodium levels were normal. Over the next 7 days she began to move her right lower extremity, and her speech became less dysarthric. Over the ensuing week her speech improved further, the 6th nerve palsy was less prominent, and she was able to move the right side fully, with slight distal weakness evident. A month after admission she had pancerebral angiography which revealed a 4 mm aneurysm of the anterior spinal artery located in the area of the tip of the odontoid process (fig. 2). Surgery was advised and the aneurysm was successfully clipped. Her postoperative course was without complications. Evaluated a month later, she had only a slight spastic right hemiparesis.

Discussion

Although aneurysms of the spinal arteries are similar histologically to those which occur intracranially, they are uncommon, and aneurysms involving the spinal arteries are rarely mentioned in the literature.

<table>
<thead>
<tr>
<th>Author</th>
<th>Age</th>
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<th>Neurologic findings</th>
<th>Site &amp; level of lesion</th>
<th>Ruptured</th>
<th>Alive</th>
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<tbody>
<tr>
<td>Echols et al1</td>
<td>30</td>
<td>F</td>
<td>paraparesis</td>
<td>T6-anterior spinal artery</td>
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<td>Yes</td>
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<tr>
<td>Leech et al2</td>
<td>25</td>
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<td>Hopkins et al3</td>
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<td>M</td>
<td>right spastic hemiparesis &amp; left sensory level</td>
<td>C4-right radicular artery</td>
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<tr>
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<td>51</td>
<td>M</td>
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<td>No</td>
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<tr>
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<td>C1,C2-anterior spinal artery</td>
<td>Yes</td>
<td>Yes</td>
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</table>

*(present report)*

TABLE Spinal Artery Aneurysms
In general, spinal artery aneurysms are more common in women, and back pain with radicular features at the level of the lesion are common. There have been only 5 aneurysms of the spinal arteries unassociated with arteriovenous malformations previously reported (table). None of these patients had preoperative arteriography, and there have been no prior angiographic descriptions of anterior spinal artery aneurysms and usually neuroradiologic textbooks do not comment on, nor illustrate, anterior spinal artery aneurysms. In a review of spontaneous spinal subarachnoid hemorrhage Henson and Croft commented that the diagnosis of spinal artery aneurysm is unlikely in a living patient. Our patient had a hemorrhage from her aneurysm, but recovered and was able to tolerate surgical clipping of the aneurysm. This patient is the first reported with a subarachnoid hemorrhage from a spinal artery aneurysm who did not expire.

The patient described had signs and symptoms suggestive of a subarachnoid hemorrhage, but when first seen it was difficult to attribute her symptoms to an anterior spinal artery aneurysm. She did complain of neck pain, but this complaint was not different from that reported by other patients with subarachnoid hemorrhage. Because of the large amount of blood in the basilar cisterns and the 4th ventricle, a posterior fossa location for a presumed aneurysm was considered. Her development of focal neurological signs was probably due to vasospasm. Patients with spinal artery aneurysms associated with spinal arteriovenous malformations have been reported, but in most of them the arteriovenous malformation had ruptured, and the aneurysm was an incidental finding.

References
Anterior spinal artery aneurysm presenting as a subarachnoid hemorrhage.

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