Dissecting hematoma of the cervical vertebral artery

Case report

STEVEN J. GOLDSTEIN, M.D.

Department of Diagnostic Radiology, University of Kentucky Medical Center, Lexington, Kentucky

A 31-year-old woman developed a sudden onset of vertebrobasilar ischemia while exercising. Cerebral arteriography revealed a dissecting hematoma of the cervical portion of the dominant vertebral artery. Both the neurological deficits and the intramural hematoma of vertebral artery resolved with conservative therapy alone.

KEY WORDS • vertebral artery • dissecting hematoma • vertebrobasilar ischemia • medial necrosis • dissecting aneurysm

REPORTS of carotid dissection appear quite regularly in the current medical literature; however, cases of dissecting hematoma of the vertebral artery are rarely encountered. A review of the world literature reveals only eight angiographically or pathologically documented cases of vertebral artery dissecting hematoma. In the majority of these cases the involved segment was the distal intradural portion of the vertebral artery.

This report describes a patient who presented with posterior fossa ischemia secondary to a long-segment cervical vertebral artery dissection. With conservative therapy alone, the neurological deficits and pathological appearance of the vertebral resolved without permanent sequelae. To our knowledge, the angiographic findings in this case are unique and have not previously been described in cervical vertebral artery dissection.

Case Report

This previously healthy 31-year-old woman was referred to the University of Kentucky Medical Center because of the sudden onset of left-sided headache, dizziness, slurring of speech, and left-sided weakness while playing softball. Upon admission her vital signs and general physical examination were unremarkable. Neurological examination, however, revealed right facial weakness, ptosis of the right eye, left upper and lower extremity weakness, and dysmetria on finger to nose testing with the left hand. Cranial computerized tomographic scan and cerebrospinal fluid examination performed on the day of admission were both normal.

Cerebral angiography the following day revealed no abnormalities of either the cervical carotid arteries or the supratentorial intracranial circulation. A right vertebral injection was then performed, and this demonstrated a small artery which terminated in the posterior inferior cerebellar artery without opacification of the basilar artery or its branches. In order to more fully evaluate the status of the posterior fossa circulation, the left vertebral artery was then selectively catheterized. A test injection under fluoroscopic control revealed an irregularly narrowed vessel beginning approximately 2 cm beyond its origin. Because of this finding the catheter was retracted into the left subclavian artery, and a hand injection was made with filming of the cervical portion of the left vertebral artery. The angiogram confirmed the fluoroscopic observation of irregular narrowing of the left vertebral artery beginning 2 cm distal to its origin. This string of beads, or wave-like appearance of the lumen was thought to be diagnostic of vertebral artery dissecting hematoma (Fig. 1 left). The pathological segment of dissection extended to the level of the C-2 vertebral body, beyond which point the vertebral artery resumed a normal caliber and configuration. Even though the angiogram failed to reveal any evidence of intraluminal thrombus, the contrast flow in the vessel was quite sluggish. The study was terminated.
FIG. 1. Left: Left subclavian angiogram showing long-segment narrowing of the left vertebral artery beginning just beyond to its origin (arrow). The vertebral artery distal to C-2 is normal (double arrow). This angiographic appearance is characteristic of dissecting intramural hematoma with compromise of the residual vascular lumen. Right: Left subclavian angiography, 3 months later. The appearance of the left vertebral artery is now almost normal. There is slight residual narrowing of the intracanicular segment (arrows) as compared to the uninvolved distal portion of the vertebral artery (arrowheads).

without any further attempt to demonstrate the posterior fossa vascular anatomy via a selective injection.

As the patient's left vertebral artery provided the dominant flow to the posterior fossa circulation, consideration was given to possible bypass surgery. Since the patient was neurologically stable and, considering that the vessel was not occluded, it was elected to treat her with anticoagulation therapy and a firm cervical collar.

An exhaustive medical and laboratory evaluation of the patient during her admission was entirely unrevealing as to the possible etiology of the vertebral artery dissection. All of the neurological deficits, however, fully resolved by the time of her discharge 3 weeks later. She was treated with anticoagulation drugs for the next 3 months and then readmitted for follow-up angiography. Her neurological examination upon readmission remained normal. A repeat subclavian angiogram demonstrated almost total resolution of the left vertebral artery dissection (Fig. 1 right). Since the posterior fossa circulation had not been adequately demonstrated previously, an attempt was made to selectively catheterize the left vertebral artery. This procedure was immediately abandoned, however, when the intima of the vessel was noted to be extremely friable and easily disrupted by passage of a soft No. 5 French polyethylene catheter. The patient was discharged the day after angiography and continued on warfarin therapy for 3 months. She has remained asymptomatic without neurological sequelae during 6 months of clinical follow-up review.

Discussion

Vascular dissection results when a pathological transintimal communication is established between the lumen of the vessel and its wall. This permits blood to dissect the internal elastic membrane from the media or to dissect through the media or adventitia proper. Even though a pseudoaneurysm may be demonstrated at the site of the original intimal defect, the extraluminal hematoma itself, and not the aneurysm, is the pathological agent which dissects through layers of the vessel wall. For this reason, this entity is more properly termed a "dissecting hematoma," rather than the more commonly used "dissecting aneurysm."

Dissection of the great vessels in the neck may be caused by blunt or penetrating trauma, severe hyperextension or rotational injury, atherosclerosis, syphilis, systemic or local arterial degenerative disease such as Marfan's syndrome, fibromuscular dysplasia, and medial necrosis. Not infrequently,
Dissecting hematoma of the cervical vertebral artery

### Summary of nine cases of vertebral artery dissecting hematoma*

<table>
<thead>
<tr>
<th>Author, Year</th>
<th>Sex, Age (yrs)</th>
<th>Site of Vertebral Artery Dissection</th>
<th>Signs &amp; Symptoms</th>
<th>Pathological Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bostrom &amp; Liliequist, 1967</td>
<td>F, 56</td>
<td>C-2 to C-4 bilaterally intracranial &amp; basilar artery</td>
<td>none</td>
<td>medial degeneration</td>
</tr>
<tr>
<td>Escourroule, et al., 1973</td>
<td>M, 32</td>
<td>intracranial &amp; basilar artery</td>
<td>SAH &amp; seizures</td>
<td>medial degeneration</td>
</tr>
<tr>
<td>Ringel, et al., 1977</td>
<td>M, 49</td>
<td>C-2 right, C-6 left</td>
<td>global ischemia</td>
<td>fibromuscular dysplasia</td>
</tr>
<tr>
<td>Yonas, et al., 1977</td>
<td>F, 44</td>
<td>intracranial</td>
<td>SAH</td>
<td>—</td>
</tr>
<tr>
<td>Waga, et al., 1978</td>
<td>M, 53</td>
<td>intracranial</td>
<td>SAH</td>
<td>—</td>
</tr>
<tr>
<td>Pasquier, et al., 1979</td>
<td>F, 43</td>
<td>intracranial</td>
<td>SAH</td>
<td>intimal fibroplasia</td>
</tr>
<tr>
<td>Takita, et al., 1979</td>
<td>M, 33</td>
<td>intracranial</td>
<td>SAH</td>
<td>atherosclerosis?</td>
</tr>
<tr>
<td>Sherman, et al., 1981</td>
<td>F, 39</td>
<td>C1-2</td>
<td>PF ischemia</td>
<td>—</td>
</tr>
<tr>
<td>Goldstein, 1982</td>
<td>F, 31</td>
<td>T-2 to C-2</td>
<td>PF ischemia</td>
<td>—</td>
</tr>
</tbody>
</table>

* Abbreviations: SAH = subarachnoid hemorrhage; PF = posterior fossa.

Dissecting hematoma of the cervical vertebral artery occurs spontaneously without a demonstrable underlying pathological abnormality. Various radiographic patterns have been described as diagnostic of intramural arterial dissecting hematoma. These include the “string sign,” “string of beads” configuration, and “double lumen” sign. All of these result when the true lumen is compressed or deformed by the presence of the intramural hematoma lying within the vessel wall. The double lumen sign, which is rarely demonstrated radiographically, is seen when the dissection plane reestablishes direct communication with the true lumen distally, allowing blood and contrast material to flow in both the true and false passage simultaneously.

Vertebral artery dissection has been reported only in young adults in the fourth through sixth decades of life. Males and females appear to be equally affected. In the majority of cases the dissection involves the intradural portion of the vertebral artery exclusively, and will occasionally extend distally to the level of the basilar artery. Most of the patients with intradural dissection present with signs and symptoms of subarachnoid hemorrhage (Table 1).

A review of the literature reveals only three previous cases of vertebral artery dissection involving the extracranial portion of the vertebral artery. Although Ouchi, et al. ostensibly reported a case of vertebral artery hematoma, their patient is not included in this series because the radiographs reveal no associated luminal narrowing. It seems their patient probably had a false aneurysm at the origin of the right vertebral artery rather than a vertebral artery dissection as the title of this paper implies.

With the exception of the patient reported by Bostrom and Liliequist, who had no neurological findings prior to death, all of the cases of vertebral artery dissection, including our own, presented with signs and symptoms of vertebrobasilar ischemia. In each of the previous cases, the angiography or pathology demonstrated only short segmental dissections, usually involving the vertebral artery at the level of the C-2 to C-4 vertebral bodies. The angiographic findings in our patient are unique in that almost the entire extracranial vertebral artery was involved. Although surgical therapy was considered, the patient was treated conservatively with a good result.

The etiology of vertebral artery dissection in our case remains unclear. The patient gave no history of significant head or neck trauma prior to the onset of neurological symptoms. Her laboratory evaluation, which was quite extensive, was likewise unrevealing. Since she was young, normotensive, and had no abnormalities of the carotid arteries or right vertebral artery at angiography, it is unlikely that fibromuscular dysplasia or premature atherosclerosis are responsible for the vascular dissection. The only possible clue to the etiology of the dissection may lie in the fact that the left vertebral artery was noted to be extremely friable during the angiographic examination. Bostrom and Liliequist also noted “striking” friability of the vascular wall of their patient with vertebral artery dissection. The pathological examination in their case demonstrated a “loosening” of the media with a necrosis of the smooth muscles and disruption of the elastic membranes. Their final diagnosis was medial necrosis with resulting arterial dissection. Since our patient is now asymptomatic and we have obtained no pathological material, we can only speculate that the underlying cause of her dissection may also be medial degeneration of the vertebral artery.

### Acknowledgment

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Address reprint requests to: Steven J. Goldstein, M.D., Department of Diagnostic Radiology, University of Kentucky Medical Center, 800 Rose Street, Lexington, Kentucky 40536.