Spontaneous dissecting aneurysms of the basilar artery presenting with a subarachnoid hemorrhage

Report of two cases

KOHKICHI HOSODA, M.D., PH.D., SHIGEKIYO FUJITA, M.D., PH.D., TETSURO KAWAGUCHI, M.D., PH.D., YOSHITERU SHOSE, M.D., KAZUKI YONEZAWA, M.D., TAKAYUKI SHIRAKUNI, M.D., PH.D., AND MASATAKE HAMASAKI, M.D., PH.D.

Department of Neurosurgery, Hyogo Brain and Heart Center, Himeji and Shinsumu Hospital, Kobe, Japan

A spontaneous dissecting aneurysm of the basilar artery is a rare disorder, usually presenting with ischemia rather than a subarachnoid hemorrhage (SAH). Two cases are described of a dissecting aneurysm of the basilar artery presenting with an SAH. Vertebral angiography revealed a double lumen to the basilar artery. Magnetic resonance (MR) imaging detected the intramural hematoma. One patient was treated conservatively, and the other underwent operative intervention with wrapping of the aneurysm. The usefulness of MR imaging in the diagnosis and the treatment options are discussed.

KEY WORDS • dissecting aneurysm • basilar artery • subarachnoid hemorrhage • magnetic resonance imaging

Spontaneous dissecting aneurysm of the basilar artery has been considered a rare cause of brainstem infarction;6,6 recently, however, six cases have been reported in which dissecting aneurysms of the basilar artery were believed to be the cause of subarachnoid hemorrhage (SAH).1,3,9,10 Although the clinical and radiological features of dissecting aneurysms of the basilar artery have been described in the literature,1-10,13,14,16-19,22,24,25 the value of magnetic resonance (MR) imaging in such cases and the treatment of choice have yet to be determined.

We report two new cases presenting with SAH, and give a brief review of their clinical, radiological, and pathological presentations. The usefulness of MR imaging and the treatment options also are discussed.

Case Reports

Case 1

This 53-year-old man presented with a 2-week history of worsening headaches without focal neurological complaints. On the day of presentation he suddenly experienced severe headache and nausea. A diagnosis of cardiomyopathy had been made 10 years before, but he had received no medication.

Examination. On admission, the patient was alert but had a severely stiff neck. His pulse rate was 64 bt/min and his blood pressure 134/88 mm Hg. The neurological examination revealed no abnormality. Computerized tomography (CT) showed a mild SAH. Right vertebral angiography demonstrated basilar ectasia (Fig. 1). Close examination revealed a double lumen of the basilar artery. The true lumen (showing greater contrast) was present on the left side, and the false lumen (showing less contrast) was located on the right, which strongly suggested a dissecting aneurysm of the basilar artery. Bilateral carotid angiograms were normal.

Course. Repeat cerebral angiography on the 20th hospital day demonstrated no further progression of the dissection. A T₁-weighted spin-echo MR sequence (TR 500 msec, TE 20 msec) on the 21st day after admission revealed an area of high signal intensity in the wall of the basilar artery; this was consistent with the abnormality identified by vertebral angiography (Fig. 2). The basilar lumen that showed an absence of signal was narrowed by the intramural hematoma (Fig. 2a). The patient remained neurologically normal and was discharged on the 51st hospital day without additional treatment. He has since remained neurologically normal for 20 months.
Dissecting aneurysms of the basilar artery

Fig. 1. Right vertebral angiogram in Case 1. The basilar artery is dilated and appears to have a double lumen. The band of the true lumen showing greater contrast is visible on the left side, and the band of the false dissecting lumen showing less contrast is on the right (arrowheads).

Case 2

This 64-year-old woman experienced occipital headache. Seven days later, she suffered a severe occipital headache and became unresponsive. She had undergone an operation for breast cancer 6 years previously. A diagnosis of hypertension had been made 1 year before admission, but she had been normotensive without medication for the past 6 months.

Examination. On admission, she was semicomatose and had a moderately stiff neck. Her pulse rate was 85 bt/min and her blood pressure 174/110 mm Hg. Her pupils were slightly anisocoric (greater on the left than the right) and fixed in mid-position without a light reflex. Her limbs were flaccid with normal deep-tendon reflexes and flexor plantar response. After 20 minutes in the hospital she opened her eyes in response to commands, and her pupils became responsive to light; however, shortly thereafter, she complained of severe headache and showed rapid deterioration of consciousness progressing to semicoma.

A CT scan showed a diffuse symmetrical SAH predominantly in the posterior fossa and intraventricular hematoma in the third and fourth ventricles. Vertebral angiography demonstrated moderate ectasia of the upper half of the basilar artery (Fig. 3). Close examination revealed a double lumen of the basilar artery. A central strip of true lumen (showing greater contrast) was present between two strips of false lumen (showing less contrast). Interestingly, the left superior cerebellar artery was opaque only through the false lumen. These findings strongly suggested a dissecting aneurysm of the basilar artery. Bilateral carotid angiograms were normal, except for severe vasospasm of the right anterior cerebral artery.

Operation. On the day after admission, a left temporal craniotomy was performed. The tentorium was divided to expose the basilar artery. The prepontine cistern was found to be packed with a very dense clot. There was a soft pulsatile reddish-purple swelling in the left outer wall of the distal basilar artery that was consistent with the dissecting lumen seen in the angiogram; this was thought to be the origin of the SAH. A small perforating artery from the dorsal surface of the mid-portion of the basilar artery was not involved in the dissection. The affected basilar artery segment was

Fig. 2. Magnetic resonance T1-weighted spin-echo images (TR 500 msec, TE 20 msec) in Case 1 (1.5-tesla system). a: Axial view showing a crescentic high signal-intensity mass (arrow) expanding the wall and reducing the lumen of the basilar artery. b: Coronal view showing high signal intensity (arrow) in the right wall of the middle part of the basilar artery and an absence of signal from the full length of the patent true basilar lumen. c: Sagittal view showing high signal intensity (arrow) along the basilar artery.
wrapped with Bemsheet and coated with cyanoacrylate. Careful attention was paid not to compromise the perforating artery with this wrapping.

Postoperative Course. After the operation, the patient was disoriented and showed left abducens nerve palsy, dysarthria, dysphagia, and monoparesis of the left lower limb. Postoperative CT revealed a small low-density area in the right frontoparietal region thought to be due to vasospasm of the right anterior cerebral artery. Repeat vertebral angiography revealed no further progression of the dissection. A T₁-weighted spin-echo MR sequence (TR 2000 msec, TE 100 msec) 2 months after the operation demonstrated a relatively high signal in the region of the left wall of the upper part of the basilar artery (Fig. 4b); this was consistent with the abnormality identified by the vertebral angiography (Fig. 3). The basilar lumen was narrowed by the intramural hematoma (Fig. 4a). Five months later, the only remaining neurological abnormality was monoparesis of the left lower limb.

Discussion

Spontaneous dissecting aneurysm has been considered an uncommon cause of arterial stenosis or occlusion that results in cerebral ischemia rather than SAH.⁴⁶ Recently, however, attention has been focused on dissecting aneurysms as a cause of SAH, particularly in the posterior circulation. A review of the reported dissecting aneurysms which have caused an SAH shows that approximately 86% have involved the posterior circulation.²³ Farrell, et al.,⁷ stated that all intracranial dissecting hematomas originate within the vessel lumen and extend through the intima and elastic lamina into the media for variable distances. The preponderance of posterior circulation dissections among the dissections that cause SAH strongly suggests that local anatomical factors have a role in determining the transmural extent of the dissection.

Incidence

Dissecting aneurysms account for 28% of reported vertebral artery aneurysms,¹⁶ whereas isolated dissecting aneurysms of the basilar artery are very rare. In reviewing the literature, we found only 25 reported cases of basilar dissection, including ours. One to 13,¹⁴,¹⁷,¹⁹,²²,²₄,²₅ The ages of the patients ranged from 15 to 64 years (mean 36.2 years). Yamaura, et al.,²⁵ reported that vertebral dissection tended to affect older individuals whose ages ranged from 37 to 69 years (mean 49.7 years). Unlike the 67% male predominance seen for vertebral dissec-
Dissecting aneurysms of the basilar artery

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Authors &amp; Year</th>
<th>Age (yrs)</th>
<th>Sex</th>
<th>Symptoms &amp; Signs</th>
<th>CT Findings</th>
<th>Angiography</th>
<th>Surgery</th>
<th>Outcome</th>
<th>Pathological Findings†</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Adams et al., 1982</td>
<td>F</td>
<td></td>
<td>headache, stiff neck, bilateral 6th nerve paresis, hemiataxia, coma</td>
<td>obliteration of 4th ventricle, blood in 3rd ventricle</td>
<td>narrow BA with small lateral protrusion</td>
<td>not done</td>
<td>dead</td>
<td>atherosclerotic plaque of BA, IEL/M/A dissection</td>
</tr>
<tr>
<td>2</td>
<td>Friedman &amp; Drake, 1984</td>
<td>F</td>
<td></td>
<td>headache, nausea, rt hemiparesis, dysphasia</td>
<td>not done</td>
<td>narrow BA with aneurysm in mid-basilar region</td>
<td>soft pulsatile reddish-purple swelling in BA, wrapping</td>
<td>not done</td>
<td>dead</td>
</tr>
<tr>
<td>3</td>
<td>Berger &amp; Wilson, 1984</td>
<td>39, F</td>
<td></td>
<td>headache, loss of consciousness, locked-in syndrome</td>
<td>not done</td>
<td>diffuse narrowing of BA distal to AICA origin</td>
<td>not done</td>
<td>GR</td>
<td>not done</td>
</tr>
<tr>
<td>4</td>
<td>Berger &amp; Wilson, 1984</td>
<td>57, M</td>
<td></td>
<td>headache, diplopia, bilateral 6th nerve paresis, dysarthria, rt hemiparesis</td>
<td>negative</td>
<td>fusiform dilatation at tip of BA</td>
<td>atherosclerotic dilatation of distal BA, wrapping</td>
<td>not done</td>
<td>IEL/M/A dissection</td>
</tr>
<tr>
<td>5</td>
<td>Farrell et al., 1985</td>
<td>46, F</td>
<td></td>
<td>headache, rt hemiparesis, dysarthria</td>
<td>not done</td>
<td>BA trunk narrowing with midsegment aneurysm</td>
<td>fusiform reddish-purple swelling along BA (AICA to SCA), wrapping</td>
<td>not done</td>
<td>dead</td>
</tr>
<tr>
<td>6</td>
<td>38, F</td>
<td>coma</td>
<td></td>
<td>midbrain &amp; pontine infarction</td>
<td>not done</td>
<td>not done</td>
<td>not done</td>
<td>IEL/M/A dissection</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>Hosoda et al., 1991</td>
<td>53, M</td>
<td></td>
<td>headache</td>
<td>SAH predominant in posterior fossa</td>
<td>basal artery ectasia with double lumen</td>
<td>not done</td>
<td>GR</td>
<td>not done</td>
</tr>
<tr>
<td>8</td>
<td>64, F</td>
<td>headache, semicoma</td>
<td></td>
<td>diffuse SAH &amp; intraventricular hemorrhage</td>
<td>basal artery ectasia with double lumen</td>
<td>reddish-purple swelling of BA, wrapping</td>
<td>not done</td>
<td>MD</td>
<td>not done</td>
</tr>
</tbody>
</table>

*SAH = subarachnoid hemorrhage; CT = computed tomography; BA = basilar artery; AICA = anterior inferior cerebellar artery; SD = severely disabled; GR = good recovery; SCA = superior cerebellar artery; MD = moderately disabled.
† Dissection: IEL = internal elastic lamina; M = media; A = adventitia; I = intima.

TABLE 1

Summary of reported dissecting aneurysms of the basilar artery presenting with SAH

...basilar dissection showed no sexual preference (13 males and 12 females). Eight patients (32%) with basilar dissection presented with SAH (Table 1) and 17 with ischemia. The incidence of SAH was 86% for vertebral dissection; however, among 14 patients with basilar dissection that have been reported since 1980, eight patients (57%) including our two cases presented with SAH. Most patients, including those who presented with ischemia, suffered severe headache. Clinical manifestations consisted of various combinations of hemiparesis, quadriplegia, cranial nerve dysfunction, cerebellar dysfunction, semicoma, and coma.

Diagnostic Studies

Basilar dissection is suspected when angiographic features such as the "string sign," "rosette sign," "pearl reaction," and luminal narrowing are present. The only definite angiographic diagnostic sign, however, is the demonstration of a "double lumen." This characteristic finding demonstrated in our patients (Figs. 1 and 3) has been reported only once before.

The use of MR imaging for vascular dissection has been reported recently, but our survey of the literature did not reveal any previous report of MR imaging in dissecting aneurysms of the basilar artery. A magnetic resonance imaging (MRI) scan of the cervical region will demonstrate fusiform reddish-purple swelling along BA (AICA to SCA), wrapping.

FIG. 4. Magnetic resonance images in Case 2 (1.5-tesla system): a: Preoperative axial T2-weighted spin-echo image (TR 500 msec, TE 20 msec). A round mass with high signal intensity is seen expanding the wall and reducing the lumen of the basilar artery. b: Follow-up coronal T2-weighted spin-echo image (TR 2000 msec, TE 100 msec) showing relatively high signal intensity (arrowheads) in the region of the left wall of the upper part of the basilar artery and an absence of signal from the full length of the patent true basilar lumen.

J. Neurosurg. / Volume 75 / October, 1991 631
imaging in cases of basilar dissection. Formation of intramural hematoma often occurs rapidly in the dissection lumen. Subacute and chronic hematoma showed increased signal intensity both in T1-weighted (short TR and short TE) and T2-weighted (long TR and long TE) MR images.12-15 The flow-void sign is consistent with rapidly flowing blood in the residual patent lumen;16 therefore, MR imaging directly detects intramural hematoma in the false lumen of a dissecting aneurysm as a hyperintense area that contrasts strongly with the absence of signal caused by blood flowing in the true patent lumen, although the true lumen might be narrowed.14,20,21 Axial MR imaging demonstrated the anatomical relationship of the intramural hematoma to the full circumference of the artery wall and its lumen (Figs. 2a and 4a). The coronal view with the image plane parallel to the blood flow of the basilar artery clearly demonstrated the anatomical relationship of the intramural hematoma to the full length of the basilar artery (Figs. 2b and 4b). Quint and Spickler17 reported gradient refocused (“flow”) imaging in which rapidly flowing blood demonstrates an increased signal that confirms the luminal patency of the true lumen. Moreover, MR imaging can detect intramural hematoma even at the very chronic phase. In our Case 2 and in Case 3 of Pozzo, et al.,26 MR imaging clearly demonstrated the presence of intramural hematoma 2 months after its onset. Because of its noninvasiveness, we recommend that MR imaging be used as the first or at least the second diagnostic method after CT for this entity if clinically suspected, especially for a patient in the subacute or chronic phase of their disease. Further study is necessary to determine whether MR imaging can diagnose basilar dissection even in the acute phase.11

Prognosis

The prognosis for patients with basilar dissection is very poor; only seven (28%) of 25 patients have survived.3,10,11 The mortality rate was 80% in nonsurgical patients and 40% in the operative group, with an overall rate within 1 month after onset of approximately 50%.12,13,14,15,16,22,23,24,25 In contrast, the mortality rate for vertebral dissection was only 8% in the most recent series.26

Treatment Options

Proximal occlusion of the vertebral artery involved is the method of choice for vertebral dissection.23,26 Yamaura, et al.,26 reported no operative deaths for the 19 patients in their recent series. Vertebral dissection occasionally extends into the basilar artery, in which case proximal occlusion of the vertebral artery may prevent further dissection distally.3 For isolated basilar dissection, however, proximal occlusion of the basilar artery obviously is not suitable. Operations have been reported in only five patients with basilar dissection, all involving wrapping of the dissection.3,10 Postoperatively, two patients died and one suffered brain-stem and cerebellar disturbance; only one patient showed good operative results. Our patient suffered monoparesis of the lower left limb due to vasospasm. Patients’ deaths were related to hemorrhagic venous infarction of the temporal lobe, secondary to injury and thrombosis of the vein of Labbé.9,10

Because of the small number of patients reported with this lesion, the best treatment for basilar dissection has yet to be determined. The potential for this lesion to rupture, however, must be considered;13 therefore, anticoagulation therapy has not been widely advocated. Our policy is that dissecting aneurysms of the basilar artery presenting with SAH should be explored if the dissection is restricted to the upper portion of the basilar artery (as in our Case 2), which is amenable to wrapping. The operative procedure would be to reinforce (wrap) as much of the abnormal vessel as possible.3 Although this might seem unsatisfactory, wrapping in combination with application of an adhesive agent such as cyanoacrylate may be particularly beneficial for patients with SAH. When the dissecting segment is free of intact branches, application of a snugly fitting clip graft would be reasonable.10 If the dissection extends from the lower portion of the basilar artery (as in our Case 1), we recommend conservative treatment without anticoagulation therapy. More information on the natural history of this disorder is necessary before a decision can be made as to the best treatment.

References

Dissecting aneurysms of the basilar artery


Manuscript received September 28, 1990.
Accepted in final form February 21, 1991.
Address reprint requests to: Kohkichi Hosoda, M.D., Ph.D., Department of Psychiatry, Yale University School of Medicine, CMHC - Room 10, 34 Park Street, New Haven, Connecticut 06508.