Clinical and Neuroradiological Features of Intracranial Vertebrobasilar Artery Dissection

Takaaki Hosoya, MD; Michito Adachi, MD; Koichi Yamaguchi, MD; Tamami Haku, MD; Takamasa Kayama, MD; Takeo Kato, MD

Background and Purpose—We sought to determine the clinical and neuroradiological features of intracranial vertebrobasilar artery dissection.

Methods—The clinical features and MR findings of 31 patients (20 men and 11 women) with intracranial vertebrobasilar artery dissections confirmed by vertebral angiography were analyzed retrospectively. The vertebral angiography revealed the double lumen sign in 11 patients (13 arteries) and the pearl and string sign in 20 patients (28 arteries).

Results—The patients ranged in age from 25 to 82 years (mean, 54.8 years). Clinical symptoms due to ischemic cerebellar and/or brain stem lesions were common, but in 3 cases the dissections were discovered incidentally while an unrelated disorder was investigated. Headache, which has been emphasized as the only specific clinical sign of vertebrobasilar artery dissection, was found in 55% of the patients. Intramural hematoma on T1-weighted images has been emphasized as a specific MR finding. The positive rate of intramural hematoma was 32%. Double lumen on 3-dimensional (3-D) spoiled gradient-recalled acquisition (SPGR) images after the injection of contrast medium was identified in 87% of the patients. The 3-D SPGR imaging method is considered useful for the screening of vertebrobasilar artery dissection.

Conclusions—Intracranial vertebrobasilar artery dissection is probably much more frequent than previously considered.

Key Words: angiography ■ dissection ■ magnetic resonance imaging ■ vertebrobasilar circulation
tained in all examinations. We used spin-echo sequences with 400/182 (repetition time/echo time/excitations) for the T1-weighted images, fast spin-echo sequences for the T2- and proton-density–weighted images, and fast inversion recovery sequences for the FLAIR images. The slice thickness of the sections was 3 to 5 mm, with 0.5- to 2.5-mm spacing between adjacent sections. To avoid flow artifacts on the T1-weighted images, a presaturation pulse was used in the lower portion of the area of scanning. Three-dimensional (3-D) imaging with spoiled gradient-recalled acquisition in the steady state (SPGR) sequences was also performed after an injection of 0.1, 0.15 mmol/kg (≤ 20 mL) of gadopentetate dimeglumine in all patients, but not in all examinations. The 3-D SPGR parameters were 26/4.5/1, with a 35° flip angle, 16- to 18-cm field of view, 1.0-mm section thickness, and 256/192/128 (matrices) until June 1996, and these parameters were 29/4.3/1, with a 35° flip angle, 21 × 15-cm field of view, 0.8-mm section thickness, and 512/256/100 (matrices) from July 1996 to December 1997.

Some MR findings suggesting arterial dissection have been reported, including an arterial intramural hematoma10–12 on T1-weighted images, intimal flap13 on T2-weighted images, and double lumen13 and enhancement of wall and septum13 on contrast-enhanced 3-D SPGR. Of these MR findings, we evaluated intramural hematoma on T1-weighted images using the spin-echo sequence and the finding of double lumen on contrast-enhanced 3-D SPGR. To avoid flow artifacts in the evaluation, a high-signal lesion seen in the lowest slice was not considered an abnormal finding in this review, in addition to the presaturation pulse in the lower portion of the area of scanning. We also considered linear high-signal lesions along the arterial wall as normal, which have been frequently observed in curved and/or dilated arteries. Intimal flaps were not evaluated, because they have often been seen in normal internal carotid arteries on T2-weighted images, which indicates that they might be caused by flow artifacts. Enhancement of the wall and septum on contrast-enhanced 3-D SPGR could not be evaluated either because this finding has been shown in occluded arteries, which could not be confirmed as arterial dissection according to these angiographic criteria. Finally, we evaluated the extension of the arterial dissection in each patient on the basis of both MR and angiographic findings.

Results

Clinical Features

Among 31 patients with intracranial vertebrobasilar artery dissection, 28 showed 1 or more clinical symptoms. In 3 patients, the vertebrobasilar artery dissection was discovered incidentally while an unrelated disorder was investigated: 1 patient with left hemiparesis due to a right cerebral infarction who had cerebral angiography, 1 with dysarthria who underwent MR angiography, and another healthy individual who requested a medical examination for brain disease, including MR angiography. The initial symptoms of the 28 patients are listed in Table 1. Brain stem and/or cerebellar signs were common, and some patients had only mild symptoms, such as headache, vertigo, or tinnitus. Associated disease was encountered in 16 patients (some patients had ≥ 2 diseases): 7 patients presented with hypertension, 6 with diabetes mellitus, 4 with cerebral infarction, 2 with angina pectoris, 1 with hyperlipidemia, 1 with cerebral aneurysm, and 1 with abdominal aortic aneurysm.

Headache and/or posterior neck pain as the only specific clinical sign of vertebrobasilar artery dissection was found in 17 patients (55%). Severe pain occurred in 11 patients, including 3 with subarachnoid hemorrhage. The locations of the pain varied: 4 patients had a whole-head headache, 1 had a frontal headache, and 12 had posterior neck and/or occipital pain (only 5 had unilateral pain). In 5 patients, there was a mild headache or neck dullness preceding the onset of symptoms.

Of the 28 patients with clinical symptoms due to intracranial vertebrobasilar artery dissection, 25 had a sudden or acute onset and 3 had an insidious onset and gradual worsening of symptoms. Seventeen patients had an acute onset with a monophasic clinical course; their symptoms gradually improved. The symptoms of 5 patients worsened for a while, and 3 patients had ≥ 2 attacks within 1 month to 12 years. Precipitating activities of 20 patients were noted. Possible dissection-related trauma was found in 9 patients: during awakening in the morning in 4, during driving in 2, during skiing in 1, after falling while carrying a child on the shoulders in 1, and while putting away the bedding in 1. Of the remaining 11 patients, 8 had symptoms during sitting, 2 during sleeping, and 1 during teaching.

As shown in Table 2, various clinical diagnoses were made by physicians. Subarachnoid hemorrhage was found in 3 patients (9.7%). Among the 3 patients with subarachnoid hemorrhage, only 1 patient showed a high-density clot in the subarachnoid space on CT. The other 2 patients were diagnosed by lumbar puncture. MR showed brain stem infarction, including lateral medullary infarction, in 12 patients. Seventeen of the 31 patients had clinical brain stem ischemia.

### TABLE 1. Initial Symptoms Due to Intracranial Vertebrobasilar Artery Dissection in 28 Patients

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Number</th>
</tr>
</thead>
<tbody>
<tr>
<td>Severe headache</td>
<td>11</td>
</tr>
<tr>
<td>Vertigo</td>
<td>11</td>
</tr>
<tr>
<td>Nausea, vomiting</td>
<td>9</td>
</tr>
<tr>
<td>Dysarthria</td>
<td>4</td>
</tr>
<tr>
<td>Tinnitus</td>
<td>4</td>
</tr>
<tr>
<td>Double vision</td>
<td>4</td>
</tr>
<tr>
<td>Dysesthesia</td>
<td>3</td>
</tr>
<tr>
<td>Consciousness disturbs</td>
<td>2</td>
</tr>
<tr>
<td>Hemiparesis</td>
<td>1</td>
</tr>
<tr>
<td>Dysphasia</td>
<td>1</td>
</tr>
</tbody>
</table>

### TABLE 2. Clinical Diagnoses of 31 Patients With Intracranial Vertebrobasilar Artery Dissection

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Number</th>
</tr>
</thead>
<tbody>
<tr>
<td>Subarachnoid hemorrhage</td>
<td>3</td>
</tr>
<tr>
<td>Infarction</td>
<td>15</td>
</tr>
<tr>
<td>Wallenberg syndrome</td>
<td>7</td>
</tr>
<tr>
<td>Other brain stem infarction</td>
<td>5</td>
</tr>
<tr>
<td>Cerebellar infarction</td>
<td>1</td>
</tr>
<tr>
<td>Cerebral infarction*</td>
<td>2</td>
</tr>
<tr>
<td>Others</td>
<td>13</td>
</tr>
<tr>
<td>Aneurysm</td>
<td>4</td>
</tr>
<tr>
<td>Vertebrobasilar insufficiency</td>
<td>4</td>
</tr>
<tr>
<td>Sudden deafness</td>
<td>1</td>
</tr>
<tr>
<td>Headache</td>
<td>1</td>
</tr>
<tr>
<td>No abnormality</td>
<td>3</td>
</tr>
</tbody>
</table>

Numbers indicate the number of patients.

*Two patients presented with infarction in the middle cerebral artery territory.
The prognosis of vertebrobasilar artery dissection was relatively good. Twenty-five patients were followed up with only medication such as drug therapy for hypertension or anticoagulant therapy, 3 patients had balloon and/or coil embolization, and 3 patients had trapping or clipping operations. There were 8 patients with a complete remission of symptoms, 15 with good recovery, and 5 without remarkable change. The remaining 3 patients had no symptom of vertebrobasilar artery dissection, which was detected incidentally. There were no deaths in this patient series.

**Angiographic Findings**
All 31 patients showed positive angiographic findings (double lumen sign and/or pearl and string sign) in 41 arteries. Four patients had both the double lumen sign and pearl and string sign, 7 had the double lumen sign only, and 20 had the pearl and string sign only. Of the 41 dissected arteries, 18 were left vertebral arteries, 11 were right vertebral arteries, and 12 were basilar arteries. The right vertebral artery dissection was extended to an extracranial portion in only 1 case.

The initial arteriograms had been made from 6 hours to 8 months after the onset of symptoms in 28 patients, and diagnoses of vertebrobasilar artery dissection were obtained in 26 patients; 2 patients were diagnosed by follow-up angiography. The initial angiography was performed in 9 patients within 7 days after onset, 6 within a month, 9 within 3 months, and 5 after 3 months. The times of onset of dissection in 3 patients were not determined because their vertebrobasilar artery dissections were detected incidentally during investigation of other disorders.

Seven patients had follow-up angiography. There were 2 patients with progressive angiographic findings (Figure 1), 3 with regressive angiographic findings (Figure 2), and 2 with unchanged findings. In the 2 patients with progressive findings, evidence of vertebral or basilar artery dissection was not obtained by initial angiography. In 1 patient, although the initial angiography performed 3 days after onset showed localized stenosis of the intracranial segment of the right vertebral artery, the pearl and string sign was visible on follow-up angiography on day 96 (Figure 1). In another patient, the initial angiography performed on the day of onset showed mild wall irregularity of the basilar artery and occlusion of the right posterior cerebral artery. We suspected that these findings were the result of a basilar artery dissection, but these findings were not considered diagnostic. A follow-up angiography on day 8 revealed the pearl and string sign of the left vertebral artery to basilar artery. In 3 patients with regressive findings, initial angiography was performed on days 2, 7, and 20 after the onset of the symptoms. A follow-up angiography performed 36 days after onset in the first patient showed that the pearl and string sign of the left vertebral artery became regressive but was still evident. Reliable angiographic findings were not obtained by follow-up angiography of the remaining 2 of the 3 patients performed at 27 days and 5 months after onset, respectively.

**MR Findings**
An intramural hematoma on T1-weighted images emphasized as a specific MR finding was detected in 10 of the 31 patients (32%) and in 11 of the 41 arteries (27%). The initial MR images of these 10 patients had been made from 7 to 61 days after the onset of symptoms, except for 1 patient whose MR examination was done 6 months after onset. None of the 7
patients who had MR examinations within a week after onset showed an intramural hematoma. Three of the 4 patients examined in the second week showed an intramural hematoma, as did 0 of 2 in the third week, 1 of 1 in the fourth week, 5 of 14 in the second month, and 0 of 5 in the third month. In 1 patient, T1-weighted images obtained 7 days after onset did not show an intramural hematoma, which was identified on examination on day 21. In another patient, T1-weighted images obtained 45 days after onset did not show the intramural hematoma, which was confirmed by the examination on day 162 (Figure 1). Nine of the 10 patients with an intramural hematoma underwent follow-up MR examinations. The intramural hematoma completely disappeared in 4 patients (Figure 2) whose follow-up MR examinations were done from 75 days to 8 months after onset. In 2 patients who underwent MR examinations 9 and 11 months after onset, the size of the intramural hematoma was reduced. In another 2 patients, the intramural hematoma disappeared, but follow-up T1-weighted images performed 3 or 4 months later showed a new intramural hematoma at a different site. In 1 patient, the intramural hematoma did not change for 4 months. A patient with an intramural hematoma discovered on day 162 underwent surgery on day 169. Four arteries showed an intramural hematoma on T1-weighted images but no positive angiographic finding.

A double lumen on 3-D SPGR images after an injection of contrast medium was identified in 27 of the 31 patients (87%) and 27 of the 41 arteries (66%). This finding was observed in approximately half of the patients who underwent MR examinations more than a year after onset. Twelve patients with a double lumen underwent follow-up MR examinations. The double lumen disappeared in 3 patients, was present but limited in 1 (Figure 3), and did not change in 7. In 1 patient the double lumen became more clear (Figure 1). Eleven arteries showed a double lumen on 3-D SPGR but no positive angiographic finding.

MR findings of 2 of the 3 patients with subarachnoid hemorrhage are shown in Figures 1 and 2. In 1 patient whose CT revealed the spread of a high-density clot in the subarachnoid space, the intramural hematoma was not clear because of a high-intensity clot within the cistern, but MR angiography clearly showed the pearl and string sign. The double lumen was partly demonstrated at the junction between pearl and string.

**Extension of Vertebral Basilar Artery Dissection**

We evaluated the extension of the arterial dissection in each patient on the basis of both angiographic and MR findings. In 10 of 31 patients (32%), arterial dissection was limited to 1 vessel. Nine patients showed unilateral vertebral artery dissection, and only 1 patient showed basilar artery dissection. In the other 21 of 31 patients (68%), arterial dissection extended into 2 or 3 vessels. Bilateral vertebral arteries were involved in 7 patients, unilateral vertebral artery and basilar artery were involved in 7 patients, and bilateral vertebral arteries and basilar artery were involved in 7 patients.

**Discussion**

**Demography and Risk Factors**

Our series includes 31 patients with intracranial vertebrobasilar artery dissection documented by vertebral angiography and who had MR examinations. Although extracranial verte-
bral artery dissection has been reported to be more common than intracranial vertebrobasilar artery dissection,7,8,14,15,22 we found only 1 patient with extracranial vertebral artery dissection. Intracranial vertebrobasilar artery dissection is probably much more frequent than previously considered and is at least not a rare condition compared with extracranial vertebral artery dissection.

We adopted strict criteria for evaluating angiograms. Among abnormal angiographic findings suggesting vertebrobasilar artery dissection, the double lumen sign (the presence of a true and false lumen or an intimal flap)1,2,16,17 is considered the only pathognomonic finding of an arterial dissection, but this has rarely been confirmed. The pearl and string sign1,18 is not pathognomonic but is considered a reliable finding of an arterial dissection. Many authors described the pearl and string sign as a reliable finding of arterial dissection,1,2,4,5,16–18 and we concur. The string sign1,19 and tapered narrowing1,19 may also be reliable findings; however, an intracranial arterial stenosis without dilatation is often difficult to distinguish from arteriosclerotic changes or vasospasm due to subarachnoid hemorrhage. Total occlusion with proximal distension1 and retention of contrast medium1,3 may be important angiographic findings as results of an arterial dissection. We did not consider these findings to be arterial dissection in the present study.

Our patients with dissection are older than those in other reports. The mean age in other reports was nearly 45 years and ranged from 21 to 63 years.2–7,18,20,22 In our series the mean age was 54.8 years. Our series included 13 patients older than 60 years, whereas previous reports included only a few such patients. The peak age of onset in our patients was in the 40s, and the mean age of 54.8 years is younger than that observed for arteriosclerotic stroke. We emphasize that vertebrobasilar artery dissection occurs not only in young adults but also in older adults. In contrast to a female predominance or no gender dominance in extracranial vertebral artery dissection,8,20,22 intracranial vertebrobasilar artery dissection has shown a male predominance in many reports2–6,8 but a female predominance in 1 report.7 In the present study the male/female ratio was 20/11.

Hypertension is suggested as one of the risk factors for vertebrobasilar artery dissection.7,20 The incidence of previously documented hypertension varies between 25% and 50%.4,7 In our patients, hypertension was recorded in 7 of 31 patients (23%). We agree with Berger and Wilson,5 who stated that hypertension and atherosclerosis were rare in dissecting aneurysm of the vertebral artery.

Precipitants and Activities, Including Trauma
Minor or trivial trauma preceding dissection is found in many patients with extracranial vertebral artery dissection.22 Common events reported to cause such traumas are as follows: sports activities (eg, jogging), daily activities (eg, sneezing, head turning, sleeping with unusual head position), and potentially excessive torsion of the cervical spine (eg, during chiropractic manipulation, resuscitation, intubation, tonic-clonic seizure). In this study such events were reported by 9 of the 20 patients. Minor or trivial trauma is probably also related to intracranial vertebrobasilar artery dissection. Movement may directly injure the vertebral artery or often stretch it. Stretching of the extracranial vertebral artery may result in a tear at the boundary between the third (V3) and fourth (V4) segments, which are fixed around the dura matter. Vertebral artery dissection, which occurs at the craniovertebral junction, may extend both proximally and distally.
Clinical Symptoms and Signs
Unilateral occipital headache and/or posterior neck pain has been emphasized as one of the most important clinical symptoms of spontaneous vertebral artery dissection.\textsuperscript{1–7,20–22} The frequency of headache in the present series was 55%, which is relatively low compared with that in other studies.\textsuperscript{11} The true incidence of headache is probably higher, because this result was obtained from review of patients’ records. Some physicians may not have mentioned the presence of headache. Some patients did not complain of any symptom, including headache.

The clinical presentation of vertebral artery dissection is quite varied.\textsuperscript{7,8,20–22} Subarachnoid hemorrhage was shown in only 3 patients in the present study, and 2 of them were confirmed by lumbar puncture (Figures 1 and 2). This incidence of subarachnoid hemorrhage is lower than that in reports from neurosurgical departments.\textsuperscript{2–5} Brain stem stroke was common, in agreement with reports from neurological departments.\textsuperscript{7,20–22} More than 50% of patients with intracranial vertebral artery dissection demonstrate symptoms related to brain stem ischemia. Wallenberg syndrome is the most frequent clinical syndrome in patients with brain stem stroke due to intracranial vertebral artery dissection. The reported incidence of Wallenberg syndrome in patients with vertebral artery dissection ranges from 26% to 43%.\textsuperscript{3,7,20,22} In addition, reports have indicated a probable association between intracranial vertebral artery dissection and Wallenberg syndrome.\textsuperscript{13,23} In the present study, 7 patients showed Wallenberg syndrome (23%). This incidence is less than in other reports, but Wallenberg syndrome is the most frequent brain stem infarction and occurred more frequently than subarachnoid hemorrhage in our patients with intracranial vertebral artery dissection. The dissection decreases blood flow to branches that supply the lateral medulla.

Some patients had only mild symptoms such as headache, vertigo, or tinnitus, and in 3 patients vertebral artery dissection was detected incidentally. No symptom relating to vertebral artery dissection was observed in these 3 patients.

MR Imaging
Intramural hematoma\textsuperscript{9–12,14,15} on T1-weighted images, intimal flap\textsuperscript{11} on T2-weighted images, and double lumen and enhancement of the wall and septum on contrast-enhanced 3-D SPGR\textsuperscript{13} are MR findings that suggest arterial dissection. Of these MR findings, intimal flap on T2-weighted images was not evaluated in the present study. Enhancement of the wall and septum on contrast-enhanced 3-D SPGR was also not evaluated because this finding was shown only in occluded arteries and there was only 1 patient with this finding in this study. Therefore, we evaluated the findings of intramural hematoma on T1-weighted images using a spin-echo sequence and double lumen on contrast-enhanced 3-D SPGR. To avoid flow artifacts, a high-intensity signal lesion seen in the lowest slice was not considered an abnormal finding. We also rejected a finding of linear high-intensity signal lesion along the arterial wall, which could not be differentiated from flow-related enhancement.

The positive rate of intramural hematoma was 32% in the present series, which was much lower than previously reported. According to Kitanaka et al,\textsuperscript{1,12} intramural hematoma of intracranial dissected vertebral artery was best revealed in the subacute to early chronic stage, but after 2 months, intramural hematoma could not be seen on MR images. On the basis of a review of patients with thoracic aortic dissections, Murray et al\textsuperscript{24} found a moderately strong correlation between the number of days after symptom onset and the signal intensity of the intramural hematoma, and they showed that T1-weighted images obtained within a week after onset rarely revealed high signal intensity. In our 7 patients who had MR examinations within a week after onset, we did not observe an intramural hematoma with a high signal. Our observations revealed a relatively high incidence of intramural hematoma in the subacute to early chronic stage. Among the 19 patients who had MR examinations after 1 week to 2 months, 9 patients with intramural hematoma were observed (47%). This low rate may be explained by the presence of a pseudolumen without thrombosis, in which case there is blood flow in the pseudolumen, as is also frequently seen in aortic dissections. We studied a patient whose intramural hematoma was depicted on day 162 but was unclear on day 45 (Figure 1). In this patient, we suggest that rerecrombosis of the pseudolumen occurred, which presented as an aneurysm on the follow-up angiogram obtained on day 95 because of thrombolysis of the thrombosed pseudolumen observed on the first angiogram. Several authors note the high sensitivity of intramural hematoma in arterial dissection,\textsuperscript{12,25} but they may have missed the diagnoses of arterial dissection in many patients without intramural hematoma.

Arterial dissection is probably a dynamic condition. Several authors described spontaneous improvement of the angiographic or MR angiographic findings.\textsuperscript{3,14,18,20,21} In addition, we observed progression of the angiographic findings in 2 patients, as previously observed.\textsuperscript{7,14,15} In these patients, the initial angiograms performed within a week after onset revealed a localized stenosis or a mild arterial wall irregularity. The occurrence of spontaneous thrombosis or spontaneous thrombolysis of the pseudolumen may not be rare. If spontaneous thrombolysis occurs, an intramural hematoma will not be detectable, because high signal intensity due to the thrombosed clot is observed in the subacute phase. In contrast, evidence of arterial dissection in the chronic phase may be obtained if the thrombosis occurs later. We emphasize that the duration between the onset and the examination is very important. It is possible that angiographic evidence of the arterial dissection or a finding of intramural hematoma on T1-weighted images cannot be obtained when the duration between the onset and the examination is within a week or >1 month. Accordingly, intracranial vertebral artery dissection should be considered a cause of vascular disease in the posterior fossa, even if T1-weighted images do not show an intramural hematoma.

Vascular Imaging
Contrast-enhanced 3-D SPGR is a useful method for screening of intracranial vertebral artery dissection; however, there have been few reports\textsuperscript{13} concerning contrast-enhanced
3-D SPGR. The positive rate of double lumen observed with this method was 87% in the present study, although the false-positive rate is unknown. We always observe the intracranial vertebral and basilar arteries using real-time reformation with planes perpendicular to the arteries. This has offered high-quality images on MR angiotomography with good spatial resolution. In normal subjects, some flow artifacts are observed. Central low signal intensity is frequently seen, and a semilunar hyposignal lesion is often visible with a bending artery, which is clearly found on a section along the artery. We defined a double lumen as the existence of a definite low-intensity boundary between a high-intensity lumen (true lumen) and a moderate-intensity lumen (pseudolumen), which may represent a thrombosed clot or slow blood flow. If dilatation of the arterial lumen is also noted (Figures 1 and 2), we make the diagnosis of arterial dissection by MR images only. In such cases, we performed angiography to demonstrate abnormal findings indicating double lumen on SPGR and made careful observations, a protocol that may increase the number of patients with a correct diagnosis of verteobasilar artery dissection. In our experience, approximately 50% of such patients had angiographic and arterial dissection was confirmed in one third of them by angiography. We hypothesize that we can obtain a correct diagnosis in only a small number of cases with intracranial verteobasilar artery dissection because the diagnosis of arterial dissection cannot be made if evidence of it is not obtained by neuroradiological examinations.

Although previous reports have described that arterial dissection in the posterior circulation frequently involved 2 or 3 vessels on the basis of pathological studies or angiographic or MR findings, its incidence is obscure. In the majority of reported cases, arterial dissections were limited to 1 vessel. Because of the limitation of each neuroradiological examination or field of view of the operation, the extension of arterial dissection might be an underestimation. In our present study we evaluated the extension of the arterial dissection on the basis of both MR and angiographic findings. An arterial dissection limited to a unilateral vertebral artery or basilar artery was rather rare (32%). However, there may also be many arterial dissections without positive neuroradiological findings. We consider that the vertebral artery dissection occurring at the third segment (V3) has a tendency to extend to the basilar artery and/or contralateral vertebral artery, since an aortic dissection often extends to its branches and/or the iliac arteries.

No case of subarachnoid hemorrhage due to intracranial vertebral artery dissection studied with MRI has been reported until now, to our knowledge. In the present study we described MR findings of 3 patients with subarachnoid hemorrhage. Intramural hematoma on T1-weighted images was clearly visible, except in 1 case with spread of a high-intensity clot within the cistern. A double lumen on 3-D SPGR was detected in all 3 cases. We did not find any specific MR finding or a specific angiographic finding for subarachnoid hemorrhage.

Some recent studies indicate the possibility of establishing the diagnosis of extracranial arterial dissection with the use of ultrasound, helical CT, and MR angiography. However, the ultrasound technique does not have diagnostic value for intracranial arteries, and helical CT is also less reliable for detecting abnormality in the posterior fossa because of many artifacts. MR angiography is not able to demonstrate pathognomonic signs of arterial dissection, and MRI is more useful than MR angiography for the diagnosis. In our department, MR angiography is not used for screening of the posterior circulation because we believe that contrast-enhanced 3-D SPGR is superior to MR angiography for this purpose; MR angiography does not demonstrate slow flow or thrombosed clot with low signal intensity within the pseudolumen. In addition, original images of MR angiography have a lower signal-to-noise ratio than those of contrast-enhanced 3-D SPGR. In our series, there were 3 patients in whom verteobasilar artery dissection was suggested by MR angiography performed in other hospitals. All findings obtained by MR angiography were pearl and string signs. MR angiography is probably useful in some patients with a pearl and string sign or for observations of the changes of the abnormality, which contributes to the correct diagnosis of arterial dissection. However, MR angiography might be insufficient for a screening of intracranial verteobasilar artery dissection.

Treatment and Outcome

We performed an embolization or operation in only 6 patients with subarachnoid hemorrhage, large aneurysm, or progressive stroke, although we do not have definite criteria for each treatment. Progressive brain stem infarction or fetal subarachnoid hemorrhage after surgical treatment has been reported; however, we had satisfactory results in this study.

Bassetti et al. reported that the recurrence of arterial dissection occurred rarely and the rate of recurrence was similar for the cervical carotid and vertebral arteries, whereas no recurrence of vertebral artery dissection was seen, as reported by several authors. In our present study, 3 patients showed ≥2 attacks with neurological deficit within 1 month to 12 years, and some patients complained of repeated symptoms, such as vertigo and headache. These attacks and repeated symptoms may have resulted from the aforementioned dynamic condition of verteobasilar artery dissection; however, it is possible that they were due to recurrent arterial dissection. Our series included only 1 patient with recurrent vertebral artery dissection confirmed by angiography. The recurrent dissection occurred at the contralateral vertebral artery as a dissecting aneurysm without apparent symptoms after embolization therapy.

In conclusion, intracranial verteobasilar artery dissection is probably much more frequent than previously considered. We emphasize that many patients with intracranial verteobasilar artery dissection are asymptomatic or have only minor symptoms. If adequate neuroradiological screening of the posterior fossa is used, the detection of intracranial verteobasilar artery dissection will increase. Contrast-enhanced 3-D SPGR with T1-weighted images is the most valuable method for detecting intracranial verteobasilar artery dissections. Angiography may be necessary for the definite diagnosis of intracranial verteobasilar artery dissection because the sensitivity of the finding of intramural hematoma is not satisfac-
tory and the reliability of the finding of double lumen on 3-D SPGR has not been determined.

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References

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