Diagnosis of Vertebral Artery Dissection with Basiparallel Anatomical Scanning Magnetic Resonance Imaging

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Abstract

Background and Purpose: There is no consensus regarding the optimal method for diagnosing the dissection of intracranial arteries. We have developed a rapid and accurate examination method to diagnose vertebral artery dissection in the acute stage of cerebral infarction.

Methods: Twenty-two patients with severe headache and neck pain and/or symptoms of brain stem or cerebellar ischemia underwent magnetic resonance imaging (MRI) with a 1.5-T scanner. Our protocol generated 3 contrast-weighted scans (T2-weighted, diffusion-weighted, and basi-parallel anatomical scanning [BPAS]-MRI) and conventional angiographs within 3 hours of the onset of symptoms. Then, we retrospectively analyzed the findings to identify the most reliable imaging method for diagnosing vertebral artery dissection in the acute stage of cerebral infarction.

Results: Based on the symptoms and the findings of T2-weighted imaging and conventional angiography, the initial diagnosis was dissection in 17 patients, lacunar infarction in 3 patients, and atherothrombosis in 2 patients. After follow-up studies the diagnosis was changed in 7 patients. The diagnosis based on symptoms and the findings of T2-weighted MRI and BPAS-MRI was dissection in 13 patients, atherothrombosis in 6 patients, and lacunar infarction in 3 patients. In 3 patients the diagnosis was changed during the follow-up phase.

Conclusions: The diagnostic accuracy rate was higher with T2-weighted MRI and BPAS-MRI than with T2-weighted MRI and conventional angiography. We suggest that when intracranial vascular dissection is suspected, both the inner and outer contours of vessels must be inspected and that BPAS-MRI should be performed instead of conventional angiography to establish the definite diagnosis.

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Key words: dissection, diagnosis, magnetic resonance imaging, angiography, vertebral artery

Introduction

Patients in the hyper-acute stage of cerebral

infarction usually receive thrombolytic therapy. Because some patients with cerebral infarction also have intracranial vertebral artery dissection, their treatment remains controversial. Because dissection

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of the intracranial vertebral artery may lead to subarachnoid hemorrhage during anticoagulation therapy for cerebral infarction¹, a rapid and accurate diagnosis is important for appropriate management. However, there is no consensus regarding the optimal method for diagnosing intracranial dissection. Conventional angiography has been considered the standard means of diagnosis²³, but carries a risk of complications and does not provide information on the condition of the arterial wall. Therefore, rapid, noninvasive diagnostic techniques, e.g., magnetic resonance imaging (MRI) and MR angiography (MRA), have been developed.

The MRI findings suggestive of arterial dissection⁴⁻⁸ include arterial intramural hematoma on T1-weighted images, intimal flap on T2-weighted images, and double lumen and enhancement of the wall and septum on contrast-enhanced 3-dimensional (3-D) spoiled gradient-recalled acquisition images⁴. According to Nagahata et al.⁹, basiparallel anatomical (BPAS-MRI) MRI provides scanning useful information on vertebrobasilar vascular lesions, especially in patients with arterial dissection.

Diffusion-weighted imaging (DWI) is sensitive to the diffusion of water molecules and is commonly used as the initial imaging examination for detecting acute cerebral infarction. In the present study, patients in the acute stage of vertebrobasilar vascular disease underwent DWI MRI, T2-weighted MRI, BPAS-MRI, and conventional angiography to identify the fastest and most accurate method for diagnosing vertebral artery dissection.

Materials and Methods

Our study population comprised 22 patients (17 men, 5 women; age range 36 to 76 years; mean age 56.4 years) who presented with severe headache and neck pain and/or symptoms of a disturbance of the posterior circulation, such as dysphagia, ataxia, diplopia, and dizziness. Within 3 hours after the onset of symptoms, all patients underwent conventional angiography and MRI examinations that generated 3 contrast-weighted images (T2, diffusion, BPAS). Because some reports have described the MRA as being insufficient for

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screening for intracranial vertebrobasilar artery dissection⁴, we did not perform MRA.

All MRI examinations were performed with a 1.5-T scanner (SIGNA Infinity EXCITE XL 1.5T, GE Medical Systems, Milwaukee, WI, USA) using axial turbo spin-echo T2-weighted imaging and axial gradient-echo imaging. The imaging parameters were 4,800/100 (TR/TE) for T2 and 700/23 for gradient-echo imaging, a 200- to 220-mm field of view, a 256×256 matrix, and 3-mm section thickness. BPAS-MRI was performed in a 20-mm-thick coronal section parallel to the clivus using a fast spin-echo sequence. The imaging parameters were 10,000/400 (TR/TE), a $220- \times 220$ -mm FOV, and a 512×256 matrix. With this pulse sequence, the total acquisition time was 4 minutes 28 seconds.

Conventional angiography was performed with digital subtraction 1 to 2 hours after the MRI scans. The diagnosis of dissection, fusiform aneurysm, or atherosclerotic change at the site of vertebrobasilar vascular lesions was based on the clinical history and the findings of conventional angiography and MRI. We suspected dissection when there was evidence of intra-arterial lesions, such as mural hematoma, intimal flap, double lumen, and plaque on T2-weighted MRI; intimal flap, double lumen, the pearl sign, the string sign, stenosis, dilatation, and tapered occlusion on angiographs; and dilatation or irregularity on BPAS images. We studied these patients on admission and diagnosed dissection if MRI findings changed during the follow-up phase; for example, if there was a change in the intensity of intra-arterial lesions on T1- or T2-weighted MRI or a change on BPAS-MRI scans. However, we suspected atherothrombosis if the initial BPAS image showed no abnormal findings, even when other examination demonstrated abnormal findings.

Results

The final diagnosis was intracranial vertebrobasilar artery dissection in 14 patients, atherothrombotic infarction in 4 patients, and lacunar infarction in 3 patients; 1 patient had a true aneurysm (**Table 1, 2**).

The initial symptoms were headache alone in 4

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Case	Age/Sex	Symptoms	High intensity area of DWI	Site of abnormal vessel	T2WI (intensity)	BPAS	Angiography
1	36/F	HA, Dysphagia	Lateral medullary	VA	NP	Dilatation	Pearl&String
2	63/M	HA, Gait disturbance	none	VA	Plaque or Mural Hx (iso)	Dilatation	Dilatation
3	62/M	HA	none	VA	Plaque or Mural Hx (iso)	Dilatation	Intimal flap
4	68/M	HA, Gait disturbance	Lateral medullary	VA	Plaque or Mural Hx (iso)	NP	String
5	41/F	Dysphagia	Medial medullary	NP	NP	NP	NP
6	71/M	Tetraplegia	none	BA	Plaque or Mural Hx (high)	Dilatation	Dilatation
7	55/M	HA, Dysphagia	Lateral medullary	VA	Plaque or Mural Hx (low)	Irregularity	Occlusion
8	67/M	Hoarseness	none	VA	NP	Dilatation	Dilatation
9	48/M	HA, Gait disturbance	Cerebellar hemisphere	VA	NP	NP	NP
10	55/M	HA	none	VA	Plaque or Mural Hx (iso)	Dilatation	Dilatation
11	48/M	HA, Dizziness	Lateral medullary	VA	Double lumen	Dilatation	Intimal flap
12	69/M	HA, Double vision	none	VA	Plaque or Mural Hx (mix)	Dilatation	Occlusion
13	54/M	HA, Dysphagia	Pons	BA	Intimal flap	Dilatation	Stenosis
14	71/M	HA	none	VA	NP	Dilatation	Dilatation
15	76/M	Double vision	Lateral medullary	NP	NP	NP	NP
16	53/M	Gait disturbance	Cerebellar hemisphere	VA	Plaque or Mural Hx (low)	NP	Occlusion
17	72/F	HA, Double vision	Cerebellar hemisphere	VA	Plaque or Mural Hx (high)	NP	NP
18	62/M	Gait disturbance	Cerebellar hemisphere	BA	Plaque or Mural Hx (iso)	NP	String
19	45/F	НА	none	VA	Intimal flap	Dilatation	Double lumen
20	63/M	Gait diturbance	Cerebellar hemisphere	VA	Plaque or Mural Hx (iso)	NP	Stenosis
21	46/F	HA, Dysarthria	Cerebellar vermis	VA	Plaque or Mural Hx (high)	Irregularity	Dilatation
22	53/M	Gait disturbance	Cerebellar	NP	NP	NP	NP

Table 1 Summary of clinical and initial neuroradiological findings in 22 patients

Abbreviations: DWI, diffusion-weighted image; T2WI, T2-weighted image; BPAS, basiparallel anatomical scanning; F, female; M, male; HA, headache; VA, vertebral artery; BA, basilar artery; Mural Hx, mural hematoma; NP, no particular finding.

patients, symptoms of posterior circulation disturbance alone in 8 patients, and both in 10 patients. Among the 14 patients with dissection, 4 complained of headache and neck pain but had no symptoms of posterior circulation disturbance; only 1 patient had quadriplegia without headache. In 14 patients DWI confirmed cerebral infarction: at the cerebellar hemisphere in 6 patients; at the lateral medulla in 5 patients; and at the medial medulla, cerebellar vermis, and pons in 1 patient each. Three of the 5 patients with a diagnosis of dissection had a cerebral infarction at the lateral medulla. Fifteen patients showed anomalous intra-arterial findings raising the possibility of dissection on T2-weighted MRI: mural hematoma or plaque in 12 patients, intimal flap in 2 patients, and double lumen in 1

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Case	Initial diagnosis from T2WI and AG findings	Initial diagnosis from T2WI and BPAS findings	Final diagnosis from follow up examinations
1	Dissection	Dissection	Dissection
2	Dissection	Dissection	Dissection
3	Dissection	Dissection	Dissection
4	Dissection	Atherothrombosis	Atherothrombosis
5	Lacunar infarction	Lacunar infarction	Lacunar infarction
6	Dissection	Dissection	Dissection
7	Dissection	Dissection	Dissection
8	Dissection	Dissection	True aneurysm
9	Athreothrombosis	Atherothrombosis	Dissection
10	Dissection	Dissection	Dissection
11	Dissection	Dissection	Dissection
12	Dissection	Dissection	Dissection
13	Dissection	Dissection	Dissection
14	Dissection	Dissection	Dissection
15	Lacunar infarction	Lacunar infarction	Lacunar infarction
16	Dissection	Atherothrombosis	Atherothrombosis
17	Atherothrombosis	Atherothrombosis	Dissection
18	Dissection	Atherothrombosis	Atherothrombosis
19	Dissection	Dissection	Dissection
20	Dissection	Atherothrombosis	Atherothrombosis
21	Dissection	Dissection	Dissection
22	Lacunar infarction	Lacunar infarction	Lacunar infarction

Table 2 Difference between initial and final diagnoses in 22 patients

Abbreviations: T2WI, T2-weighted image; AG, angiography; BPAS, basiparallel anatomical scanning.

patient.

Abnormal angiographic findings were noted in 17 patients: vascular dilatation in 6 patients; tapering occlusion in 3 patients; intimal flap, the string sign, and stenosis in 2 patients each; and the pearl-and-string sign and double lumen in 1 patient each. The sensitivity and specificity of angiography were 86.7% and 42.9%, respectively. BPAS-MRI showed abnormalities in 13 patients: dilatation in 11 patients and irregularities in 2 patients. The sensitivity and specificity of BPAS-MRI were 84.6% and 77.8%, respectively.

On the basis of symptoms and the findings of T2weighted MRI and conventional angiography, dissection was initially diagnosed in 17 patients, lacunar infarction in 3 patients, and atherothrombosis in 2 patients. After the follow-up study, the diagnosis was changed in 7 patients: from dissection to atherothrombosis in 4 patients, from atherothrombosis to dissection in 2 and from dissection to true aneurysm in 1 patient. The sensitivity and specificity of the combination of T2weighted MRI and convetional angiography were 85.7% and 37.5%, respectively. On the other hand, on the basis of symptoms and the findings of T2weighted MRI, and BPAS-MRI, dissection was initially diagnosed in 13 patients (**Fig. 1**, case 3), atherothrombosis in 6 patients, and lacunar infarction in 3 patients. In the chronic stage, the diagnosis was changed in 3 patients: from atherothrombosis to dissection in 2 and from dissection to true aneurysm in 1 patient (**Fig. 2**, case 4; **Fig. 3**, case 17). The sensitivity and specificity of the combination of T2-weighted MRI and BPAS-MRI were 85.7% and 87.5%, respectively.

The diagnostic accuracy of the combination of T2weighted MRI and BPAS-MRI was greater than that of the combination of T2-weighted MRI and angiography.



Fig. 1 Case 3. A 62-year-old man complaining of headache. Both the initial diagnosis based on the findings of T2-weighted imaging and angiography and on the findings of T2-weighted imaging and basiparallel anatomical scanning (BPAS), and the final diagnosis was dissection. (A) No abnormal findings were detected on diffusion-weighted image. (B) T2-weighted imaging revealed a mural hematoma in the right vertebral artery. (C) BPAS showed dilatation of the right vertebral artery. (D) Right vertebral angiography (lateral view) demonstrating an intimal flap in a fusiform aneurysm.



Fig. 2 Case 4. A 68-year-old man admitted for headache and gait disturbance. Although the initial diagnosis based on the findings of T2-weighted imaging and angiography, was dissection, both the initial diagnosis and final diagnosis, based on the findings of T2-weighted imaging and basiparallel anatomical scanning (BPAS) was atherothrombosis. (A) Acute cerebral infarction at the right lateral medulla was observed on the diffusionweighted imaging. (B) A mural hematoma was demonstrated in the right vertebral artery. (C) The shape of right vertebral artery was normal on BPAS. (D) Right vertebral artery demonstrating the string sign.



Fig. 3 Case 17. A 72-year-old man with headache and diplopia. Although the initial diagnosis, based both on the findings of T2-weighted imaging and angiography and findings on the findings of T2-weighted imaging and basiparallel anatomical scanning (BPAS), was atherothrombosis, the final diagnosis was dissection because a small mural hematoma was detected on follow-up magnetic resonance imaging. (A) Acute cerebral infarction at the right cerebellar hemisphere was observed on diffusion-weighted imaging. (B) (C) (D) There were no abnormal findings on T2-weighted imaging, BPAS or angiography.

Discussion

In the acute stage of cerebral infarction determining whether the infarct is due to vertebrobasilar artery dissection or atherothrombosis is difficult. Conventional angiography, which has been the standard diagnostic technique for diagnosing dissection⁴⁻⁸, has a risk of complications and does not provide information on the conditions of the arterial wall. Moreover, the angiographic appearance of artery occlusion due to dissection is nonspecific because other factors such as thromboembolism or atherosclerotic disease, can produce similar angiographic findings. MRI is an alternative noninvasive approach and the only reliable means for diagnosing occlusive dissection. We have previously reported on 7 patients in whom T2weighted MRI yielded both false-positive results (4 patients) and false-negative results (3 patients). We postulated that the false-negative results with T2weighted MRI were attributable to the fact that in the presence of arterial dissection, the signal intensity of intramural hematomas on T1- and T2weighted MRI depends on the stage of the hemorrhage. The isointense T1 signal and the increased T2 signal appear after a few hours of bleeding (oxyhemoglobin phase), the T2 signal hypointense in the becomes acute stage (deoxyhemoglobin phase), and T1 and T2 signals become hyperintense in the sub-acute stage (methemoglobin phase)¹⁰. Therefore, if the patient is examined during the acute stage (deoxyhemoglobin phase), the mural hematoma can appear hypointense, and an overlapping arterial flow void can be present on T2-weighted MRI.

An intimal flap, which is often seen on T2weighted MRI of normal internal carotid arteries, suggesting flow artifacts⁴. The false-positive T2weighted MRI findings in our earlier study showed isointensity- or hyperintensity. Vulnerable plaques such as lipid-rich plaques, and intra-plaque hemorrhages are often seen in patients with cerebral infarction due to artery-to-artery embolism¹¹. Lipid-rich plaques are reported hyperintense on T2-weighted MRI, and intra-plaque hemorrhage is isointense¹². Therefore, we postulate that in cases with false-positive results on T2weighted MRI, we were unable to distinguish between mural hematomas and vulnerable plaques.

Using BPAS-MRI we obtained a false-negative result in a patient who initially showed no abnormal findings on neurological imaging. However, because follow-up T1- and T2-weighted MRI revealed a small mural hematoma, we changed the diagnosis from cardiac embolization to artery-to-artery embolization due to dissection.

Although BPAS-MRI, which is essentially a modified form of MR cisternography, cannot detect small lesions, it does allow the unique outer contour of the vertebrobasilar artery within the cistern to be visualized. In this respect, BPAS-MRI differs from established vascular imaging methods, such as angiography and MRA, which reveal the inner vascular contour. In the present study, the final diagnosis in patients whose vessels demonstrated a normal outer appearance on BPAS-MRI scans was atherosclerotic change, despite vessel wall irregularities such as the string sign on conventional angiographs.

A diagnosis of vascular dissection is usually based on the combination of clinical symptoms and the findings of MRI and conventional angiography. We found that the specificity of BPAS-MRI or angiography was higher with T2-weighted MRI than without and that the diagnostic accuracy rate of T2weighted MRI was higher with BPAS-MRI than with angiography. However, even the diagnosis of dissection established with BPAS and T2-weighted MRI findings is not always accurate because the sensitivity and specificity of this combination in the present study was not 100%. On the basis of the present results, we recommend more careful studies to determine the presence of dissection.

Conclusion

Our results suggest that to accurately diagnose of dissection, both the inner and outer contours of the vessels must be considered and that BPAS-MRI might be used instead of conventional angiography to rule out dissection in patients with cerebral infarction.

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