Dilated Outer Diameter of the Dissected Artery: Acute Bilateral Anterior Cerebral Artery Dissection Evaluated by Repeat Magnetic Resonance Cisternography —Case Report—

Morio NAGAHATA, Hiroko SEINO, Shinya KAKEHATA, Kohei MORIMOTO, Takahiro NAKANO*, Kenichiro ASANO*, Norihito SHIMAMURA*, and Hiroki OHKUMA*

Departments of Radiology and Radiation Oncology, and *Neurosurgery, Hirosaki University Graduate School of Medicine, Hirosaki, Aomori

Abstract

A 42-year-old male patient presented with an anterior cerebral artery (ACA) dissection manifesting as sudden onset of severe headache. Initial computed tomography revealed faint subarachnoid hemorrhage in the frontal region. Initial angiography showed tapering stenosis at the A2 segment of right ACA. The patient was admitted to our hospital and treated conservatively. Magnetic resonance (MR) imaging and angiography did not detect intramural hematoma, intimal flap, or double lumen at the stenotic right A2 segment. The ACA dissection was difficult to confirm based on the findings on day 0. ACA dissection was confirmed by improvement of the right ACA stenosis on follow-up angiography on day 14. On the other hand, MR cisternography revealed a fusiform dilatation of the vascular outer contour at the right A2 on day 0, which had resolved on day 14. Fusiform dilation of the vascular outer contour at the affected segment on MR cisternography may be indicative of arterial dissection in the acute phase.

Key words: anterior cerebral artery, dissection, magnetic resonance imaging, angiography, magnetic resonance cisternography

Introduction

Confirmation of cerebral arterial dissection by neuroimaging techniques such as cerebral angiography or magnetic resonance (MR) imaging or angiography is not always easy in the acute phase. Detection of double lumen or intimal flap at the affected artery are definitive but uncommon findings of arterial dissection. Intramural hematoma on T1-weighted MR imaging is helpful to identify dissection in the early phase, but does not always appear in the acute phase. Morphological changes of the dissected artery on serial angiography are reliable indicators of dissection, but acute dissection cannot be established based only on the initial angiography. More suggestive findings for dissection in the acute phase would be very helpful to manage patients with suspected dissection.

We report a case of anterior cerebral artery (ACA) dissection appearing as transient dilatation of the vascular outer diameter at the affected segment in the acute phase on MR cisternography.

Case Report

A 42-year-old male patient complained of sudden onset of severe headache. He had no history of head trauma. Initial computed tomography revealed faint subarachnoid hemorrhage in the frontal region. Initial angiography revealed only a tapering stenosis at the A2 segment of the right ACA. The patient was admitted to our hospital and underwent cerebral angiography on the day of onset. Rupture of saccular aneurysm, arteriovenous malformation, and dural arteriovenous fistula were excluded. Right internal carotid angiography revealed only a tapering stenosis at the A2 segment of the right ACA. The subarachnoid clot was very faint, so he was treated conservatively under suspicion of right ACA dissection.

Serial MR imaging and angiography were performed using a 1.5-tesla MR machine. Diffusion-weighted images using echo planar imaging, T1-weighted images using spin-
Fig. 1 Initial computed tomography scan showing faint subarachnoid hemorrhage in the left frontal region (arrows).

Fig. 2 Initial right internal carotid arteriogram, right anterior oblique view (A), and left internal carotid arteriogram, left anterior oblique view (B), revealing tapering stenosis at the right A2 segment (arrow), but no abnormality at the left A2 segment.

Fig. 3 Serial axial T1-weighted magnetic resonance images (A to C) showing no intramural hematoma (hyperintensity) at the right A2 segment.

Fig. 4 Serial sagittal reformatted heavily T2-weighted magnetic resonance cisternograms (right [A] to left [F]) showing a fusiform dilatation of the right A2 segment (arrows) and the left A2 of normal diameter (small arrows). Please note that the vessel shadows do not indicate “flow void.”

Echo sequence, T2-weighted images using fast spin-echo sequence, and fluid-attenuated inversion recovery (FLAIR) images using fast inversion recovery sequence were obtained routinely in the axial plane. The slice thickness of the sections was 5 mm with 1-mm spacing between adjacent sections. Coronal or sagittal FLAIR and T1- and T2-weighted images were not obtained. In addition to three-dimensional (3-D) time-of-flight MR angiography, MR cisternography was also performed using sagittal 3-D heavily T2-weighted imaging with fast spin-echo sequence. The parameters of MR cisternography were 4000 msec/193 msec/1 (repetition time/echo time/excitations), 20-cm field of view, 1.0-mm section thickness, and 256/256 matrices. Follow-up right internal carotid arteriography was performed on day 14.

On admission, T1-weighted imaging detected no intramural hematoma (Fig. 3). Axial FLAIR imaging did not reveal flow abnormalities of the ACAs. Axial T2-weighted imaging did not show dilatation of the A2 segment. Internal carotid angiography and MR angiography did not detect intimal flap or double lumen at the right A2 segment. MR cisternography showed a fusiform dilatation of the vascular outer contour at the proximal segment of the right A2 (Fig. 4). Right ACA dissection was difficult to confirm based on the neuroimaging findings on day 0. Follow-up angiography confirmed the dissection based on the improvement of right ACA stenosis on day 14 (Fig. 5). MR cisternography showed that the fusiform dilatation of the proximal A2 segment had resolved on day 14 (Fig. 6). Internal carotid angiography showed a newly developed left A2 stenosis, in addition to the morphological change of right ACA, on day 14 (Fig. 4). MR cisternography also revealed a fusiform dilatation of the left A2 on day 14. The left ACA abnormality was asymptomatic, so left ACA dissection was also suspected. Table 1 summarizes the findings of repeat MR imaging, MR angiography, and conventional angiography.

Conservative management was continued expecting spontaneous resolution of the left ACA dissection similar to the right ACA dissection. The patient was discharged from our hospital with no neurological symptom or complaint 3 weeks after onset. MR cisternography showed the
Fig. 5 Right internal carotid angiogram, posteroanterior view, with left carotid compression on day 14 indicating resolution of the tapering stenosis of the right A2 (arrows) and a newly formed severe stenosis of the left A2 (small arrows).

Fig. 6 Serial sagittal reformatted magnetic resonance cisternograms (A to F) revealing normalized diameter of the right A2 (arrows) and a newly developed fusiform dilatation of the left A2 (small arrows).

Fusiform dilatation of the left A2 had resolved 9 months after the ictus.

Discussion

In the present case, follow-up angiography confirmed the presence of the right A2 dissection. The initial angiography showed only tapering stenosis (string sign). The presence of string sign without intramural hematoma on T₁-weighted imaging is only suggestive of dissection, and not a definitive finding.

Fusiform dilatation of the vascular outer diameter may be suggestive of vertebral artery dissection. Basiparallel anatomical scanning (BPAS)-MR imaging is a simple and useful method to evaluate the vertebrobasilar arterial outer contour. We hope to apply evaluation of the vascular outer appearance to the diagnosis of cerebral artery dissection in other regions. BPAS-MR imaging is heavily T₂-weighted imaging using thick sections. Unfortunately, thick-section imaging is not suitable for visualizing the outer contour of the intracranial anterior circulation arteries. We think that thin-section 3-D MR cisternography is better for this purpose.

In our case, dilatation of the vascular outer diameter at the A2 segment and subsequent resolution excluded vasospasm or atherosclerotic change. Such changes of the vascular outer contour may reflect the condition of the intramural hematoma or the presence of pseudolumen. We think that dilatation of the vascular outer diameter at the affected vessel on MR imaging should be suggestive of arterial dissection even in the acute phase. We also think that resolution of the fusiform outer contour on repeat MR cisternography should be a definitive finding of acute arterial dissection and may be a sign of resolution. According to this point of view, serial MR cisternography also suggested asymptomatic contralateral ACA (A2) dissection and spontaneous healing in the present case. This dissection may correspond with the “non-occlusive subclinical dissection” described in 1985. Reports of the bilateral ACA dissections are very rare. In our case, a diagnosis of vasospasm of the left ACA could have been established based on the carotid angiography only. MR cisternography was very useful to correctly identify this interesting dissection.

Table 1 Summary of imaging findings

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<th>Day 0</th>
<th>Day 14</th>
<th>9 Months</th>
<th>22 Months</th>
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<td><strong>Right ACA</strong></td>
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<tr>
<td>CAG</td>
<td>string</td>
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<td>intramural hematoma on T₁WI</td>
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<td>MR cisternography</td>
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<td><strong>Left ACA</strong></td>
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<td>CT/FLAIR</td>
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<td>Ischemic lesion</td>
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Fusiform dilatation of the vascular outer contour at the affected segment on MR cisternography is very helpful to identify arterial dissection in the acute phase.

References


Address reprint requests to: Morio Nagahata, M.D., Department of Neurosurgery, Yamagata City Hospital SAISEIKAN, 1–3–26 Nanoka–mach, Yamagata, Yamagata 990–8533, Japan. e-mail: naght@saiseikan.jp