Differences in Diffusion-Weighted and T₂-Weighted Magnetic Resonance Imaging Findings in the Acute and Chronic Stages of Ischemic Cerebrovascular Disease

—Two Case Reports—

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Abstract

A 71-year-old man presented with sudden onset of vertigo and a 77-year-old man suffered consciousness disturbance. Diffusion-weighted magnetic resonance (MR) imaging on admission showed hyperintense areas in the left cerebellar hemisphere in the first patient and in the brainstem in the second patient. Both patients were treated with argatroban and edaravone, and the neurological deficits markedly improved one month after admission. T₂-weighted MR imaging one month after the onset showed much smaller hyperintense areas compared with the findings on admission in both patients. These results indicate that findings of hyperintense areas by diffusion-weighted MR imaging in the acute stage of ischemic cerebrovascular disease indicate not only the ischemic core but also parts of the reversible incomplete ischemic lesion and suggest that intensive treatment in the acute stage might reverse ischemic brain damage in some patients.

Key words: cerebrovascular disease, diffusion-weighted magnetic resonance imaging, ischemia, T₂-weighted magnetic resonance imaging

Introduction

Magnetic resonance (MR) imaging is commonly used in neurosurgical daily clinical practice to monitor the development of ischemic brain injury and to indicate suitable treatment in patients with ischemic cerebrovascular disease.^{4,8,11,16)} Diffusion-weighted MR imaging can detect changes in ischemic brain tissue in the acute stage, which cannot be detected by T₂-weighted MR imaging, as a hyperintense area generally considered to show the ischemic core.⁵⁾ The size of the hyperintense area is believed to correlate with that demonstrated by T₂-weighted MR imaging in the chronic stage.^{10,12,15)} However, the hyperintense area in the acute phase may disappear or become smaller in the chronic follow-up period.^{3,4,6,12)}

We treated two patients with ischemic cerebrovascular disease, in whom the hyperintense area shown by diffusion-weighted MR imaging in the acute stage was significantly smaller than that by T₂-weighted

MR imaging in the chronic stage, and was associated with good recovery from neurological deficits. Furthermore, findings of T₂-weighted MR imaging in the acute and chronic stages were different.

Case Reports

Case 1: A 71-year-old man suffered sudden onset of vertigo and vomiting, and was admitted to our hospital on the next day. On admission, the patient was awake and alert, but complained of vertigo. Neurological examination revealed left dysmetria and ataxic gait.

Computed tomography (CT) did not show any abnormality. Diffusion-weighted MR imaging showed a hyperintense area in the left cerebellar hemisphere (Fig. 1A). T₂-weighted MR imaging showed similar findings (Fig. 1B). Under a diagnosis of left cerebellar infarction, the patient was treated using argatroban, an anti-thrombin agent,⁷⁾ (60 mg/day for 2 days, and 20 mg/day for following 5 days) and edaravone, a free radical scavenger,¹⁴⁾ (60 mg/day for 7 days). One month after the onset, the

Fig. 1 Case 1. A: Axial diffusion-weighted magnetic resonance (MR) image (repetition time [TR] 10000 msec, echo time [TE] 101 msec, field of view [FOV] 22×22 cm, b value 1000 sec/mm², slice thickness 5 mm, 1.5 mm gap) on admission showing a hyperintense area in the left cerebellum (arrowheads) indicating left cerebellar infarction. B: Axial T₂-weighted MR image (fast spin-echo sequence; TR 3300 msec, TE 104 msec, FOV 22×16 cm, slice thickness 5 mm, 1.5 mm gap) in the acute stage showing the same findings (arrowheads). C: Axial T₂-weighted MR image 3 months after admission showing that the hyperintense area has significantly decreased in size (arrowheads).

Table 1 Comparison of ischemic lesion volumes*

Case No.	Location	Initial diffusion-weighted MR imaging	Initial T ₂ -weighted MR imaging	Follow-up T ₂ -weighted MR imaging
1	lt cerebellum	8.41	9.49	1.66
2	brainstem	2.36	4.49	0.67

*Ischemic lesion volumes (cm³) were calculated as the sum of each slice area multiplied by the slice thickness. MR: magnetic resonance.

patient's neurological findings had markedly improved except for slight difficulty in walking.

Follow-up T₂-weighted MR imaging 3 months after the onset revealed a much smaller hyperintense area in the left medial cerebellar hemisphere (Fig. 1C), and calculation of the volumes showed a significant decrease compared with the diffusion-weighted and T₂-weighted MR imaging in the acute stage (Table 1).

Case 2: A 77-year-old man suffered sudden onset of consciousness disturbance, and was transferred to our hospital on the same day. The patient had undergone a right superficial temporal artery-middle cerebral artery anastomosis 15 years before the admission, but the details were unknown. On admission, his consciousness level was E1V1M5 on the Glasgow Coma Scale. The patient was almost tetraplegic, but all brainstem responses were detected.

CT did not show any obvious abnormal findings. Diffusion-weighted MR imaging demonstrated a hyperintense area in the pons (Fig. 2A). T₂-weighted MR imaging also showed similar findings, although

the hyperintense area was not diffuse (Fig. 2B). MR angiography demonstrated significantly decreased blood flow in the basilar artery (Fig. 3A).

Under a diagnosis of brainstem infarction, the patient was treated with the same regimen as in Case 1. One month after the onset, follow-up T₂-weighted MR imaging revealed a much smaller hyperintense area in the ventral pons (Fig. 2C), and measurement of the volumes showed a significant decrease compared with the diffusion-weighted and T₂-weighted MR imaging in the acute stage (Table 1). MR angiography showed recanalization of the basilar artery (Fig. 3B). The patient's neurological deficits gradually improved and he became ambulatory with the use of a cane 3 months after the onset.

Discussion

Hyperintense areas observed by diffusion-weighted MR imaging are considered to reflect the reduction of free water diffusion in the ischemic brain tissue,¹⁶⁾ and presumably correlate with the restricted extracellular space due to intracellular water accumulation (cytotoxic edema).¹¹⁾ Therefore, such a hyperintense area in the acute stage of ischemic cerebrovascular disease is thought to represent the ischemic core. On the other hand, the hyperintense area observed by T₂-weighted MR imaging reflects several factors, including complete necrosis due to ischemia or hemorrhage, edematous change, inflammation, demyelination, and so on.^{1,2,9,13)} Therefore, the hyperintense area by T₂-weighted MR imaging might reflect both complete necrosis and any edematous change due to vasogenic edema in

Fig. 2 Case 2. A: Axial diffusion-weighted magnetic resonance (MR) image (repetition time [TR] 10000 msec, echo time [TE] 99 msec, field of view [FOV] 24×24 cm, b value 1000 sec/mm², slice thickness 5 mm, 1.5 mm gap) on the day following onset showing a hyperintense area in the pons (arrows) indicating brainstem infarction. B: Axial T₂-weighted MR image (fast spin-echo sequence; TR 2800 msec, TE 103 msec, FOV 24×118 cm, slice thickness 5 mm, 1.5 mm gap) on the same day showing a less homogeneous hyperintense area in the pons (arrows). C: Axial T₂-weighted MR image one month after the onset showing marked decrease of the hyperintense area (arrows).

Fig. 3 Case 2. A: Magnetic resonance (MR) angiogram (three-dimensional time-of-flight sequence; repetition time 40 msec, echo time 6.9 msec, field of view 16×16 cm) on the day after onset showing decreased blood flow in the basilar artery (arrows). B: MR angiogram one month after the onset showing recanalization of the basilar artery (arrows).

the ischemic cerebrovascular lesion.

Neurological deficits caused by ischemic brain injury, demonstrated by diffusion-weighted MR imaging in the acute stage, have gradually improved in a few cases,³⁾ indicating the reversal of the ischemia. The decreased apparent diffusion coefficient in the lesions also returned to normal in five patients without persistent neurological symptoms or abnormality by T₂-weighted MR imaging.³⁾ The estimated probability of reversal is 0.2% to 0.4%.³⁾ The lesions were located in the supratentorial white matter (3), deep gray nuclei (thalamus) (1), and cortical gray matter in the parietal lobe (2). The five patients presented with sinus thrombosis and seizure (3),

hemiplegic migraine (1), and acute cerebrovascular ischemia followed by intravenous thrombolysis (1). These presentations are different to those seen in our patients. The probability of normalization of the hyperintense area in patients with transient ischemic attack, including one with cerebellar ischemia, was suggested to be 25%.⁵⁾ From our experience, the probability of reversal was 2.7%, as seen in two of 133 patients with ischemic cerebrovascular disease treated from January 2001 to January 2003. However, such cases are still rare, so any link with location cannot be identified.

Both our patients had complete ischemic stroke, and the size of the hyperintense area by T₂-weighted MR imaging in the chronic stage was significantly smaller compared with that by diffusion-weighted and T₂-weighted MR imaging in the acute stage. The volume of the ischemic lesion remarkably decreased in the chronic stage compared with the acute stage. The reduction in size of the hyperintense area in the chronic stage was associated with great improvement in the neurological deficits of the patients.

The difference in the sizes of the hyperintense areas in the acute stage and chronic stage in patients with ischemic cerebrovascular disease reflect the fact that the hyperintense area demonstrated by diffusion-weighted MR imaging consists of both ischemic core and reversible ischemic lesion. Intensive treatment and restoration of cerebral blood flow can reverse the brain damage caused by ischemia, so the hyperintense area became significantly smaller by T₂-weighted imaging in the chronic stage, reflecting the presence of necrosis due to infarction, edematous change, inflammation, and so on. Therefore, the hyperintense area by T₂-weighted MR

imaging in the acute phase might have been caused mostly by edematous change due to ischemia, but not necrosis.

The neurological recovery in our patients suggest that intensive treatment to restore cerebral blood circulation and protect neural tissues insulted by ischemia should be performed even in patients with hyperintense areas demonstrated by diffusion-weighted MR imaging in the acute stage.

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