

## Case Report

# Serial Magnetic Resonance Images of a Right Middle Cerebral Artery Infarction : Persistent Hyperintensity on Diffusion-Weighted MRI Over 8 Months

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A lesion that is hyperintense on diffusion-weighted imaging (DWI) and hypointense on the apparent diffusion coefficient (ADC) map is a characteristic magnetic resonance imaging (MRI) finding in acute ischemic infarction. In some cases, however, these findings can persist for a few months after infarct onset. It is thought that these findings reflect the different evolution speeds of the infarcted tissue. We report a patient with a right middle cerebral artery territory infarction with persistent hyperintensity on DWI and hypointensity on the ADC map for over 8 months. To our knowledge, this is the most persistent case of hyperintensity lesion on DWI and the serial MRI images of this patient provide important information on the evolution of infarcted tissue.

**Key Words :** Apparent diffusion coefficient · Cerebral infarction · Diffusion-weighted imaging · Perfusion-weighted MRI.

## INTRODUCTION

Diffusion-weighted imaging (DWI) is very useful for detecting acute ischemic infarction. In acute stroke, the evolution patterns of DWI, apparent diffusion coefficient (ADC), and T<sub>2</sub>-weighted images are well known.<sup>4,9)</sup> The appearance of the infarction on DWI is influenced by the ADC and T<sub>2</sub> relaxation time. In the acute stage, the decrement in the ADC value is the main cause of hyperintensity on DWI. By about 2 weeks, the ADC gradually increases to or above the normal level. Subsequently, the hyperintensity seen on DWI is related to the increment in the T<sub>2</sub> relaxation time (the T<sub>2</sub> shine-through effect).<sup>4,9)</sup>

Persistent cases of DWI hyperintensity combined with hypointensity on the ADC map during the subacute to chronic stage of infarction have been demonstrated in some studies,<sup>2,3,6-8)</sup> mainly in cases of minor stroke that involved the cerebral white matter. We present a patient with complete right middle cerebral artery (MCA) infarction showing persistent hyperintensity on DWI and hypointensity on the ADC map for over 8 months. To our knowledge, this is the first report on the persistent hyperinten-

sity on DWI with hypointensity on the ADC map in the patient of total territorial infarction as well as the most persistent case of these characteristic MRI findings of acute ischemic infarction.

## CASE REPORT

A 70-year-old woman with a history of untreated atrial fibrillation developed left-side weakness on awakening. About 5 years earlier, she had developed dyspnea and was diagnosed with atrial fibrillation, but did not take any medication regularly. About 5 months earlier, she had visited our hospital because of severe whirling-type vertigo. However, brain magnetic resonance images (MRI) at that time showed no evidence of acute infarction (Fig. 1A, 2A). She was discharged on anticoagulant medication, but she did not return to the out-patient department for follow-up.

On neurologic examination, the patient was alert, fully oriented, and had severe dysarthria. She had a central facial palsy of her left face and left-side motor weakness, with 1/5 strength in the upper extremity and 3/5 strength in the lower extremity. Babinski's sign was also observed on her left side. Her NIHSS score was 10 points.

Multiple acute infarctions were observed in her right frontal and temporo-parietal lobes (MCA territory) on MRI (Fig. 1B). On magnetic resonance angiography (MRA), complete occlusion of an entire segment of the right internal carotid artery (ICA) was

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observed (Fig. 2B). On the third day of admission, her motor weakness progressed and she could not move her left arm and leg at all. Her NIHSS score was worse at 18 points. Computed tomography showed progression of the previous infarction to the entire territory of the right MCA. She was transferred to a local hospital 17 days after admission, but her neurologic deficits had not recovered on discharge.

About 2 months later, she was re-admitted to our hospital with decreased consciousness. A work-up revealed that a urinary tract infection was the cause of her altered mentality. DWI taken the day of admission showed high signal intensity in the previously infarcted right MCA territory, especially the deep white matter (Fig. 1C). It was seen as a hypointensity on the ADC map, and the complete occlusion of the right ICA persisted on MRA (Fig. 1C, 2C). She was discharged after receiving antibiotics therapy.

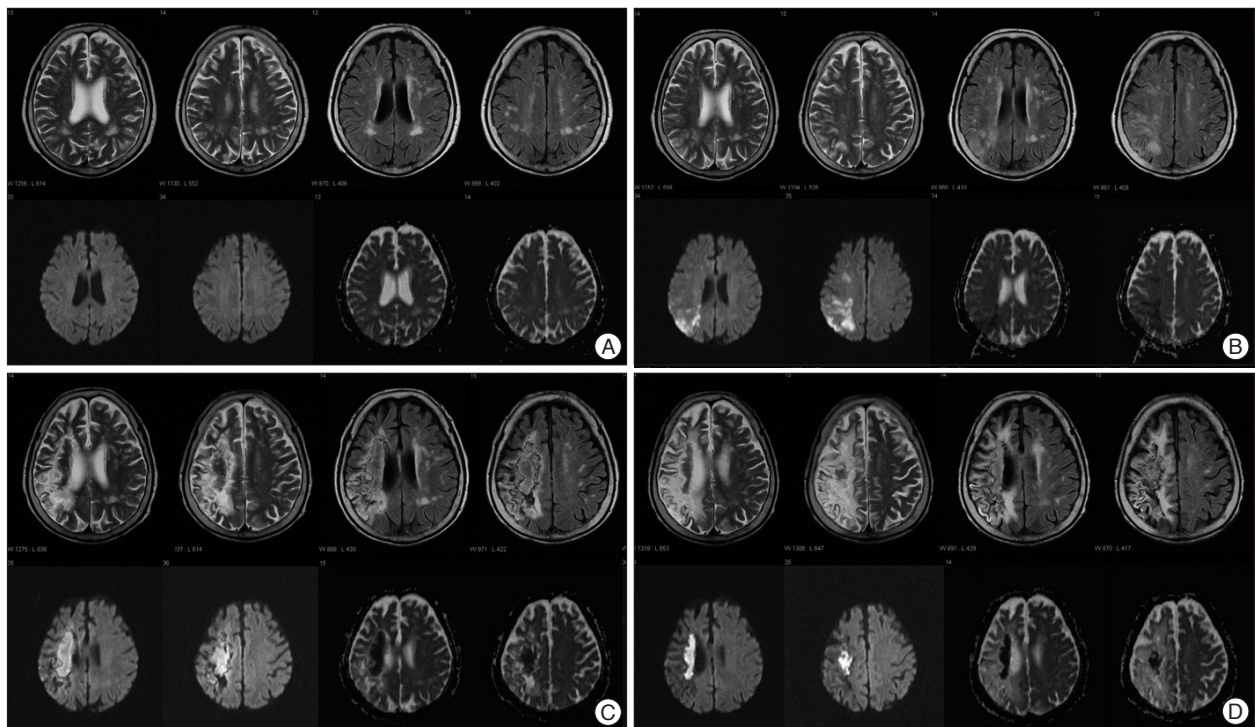
About 8 months after the first discharge, she presented to our hospital again with decreased responses to external stimuli. Again, her altered mentality was caused by a urinary tract infection. On DWI taken the day of this admission, the hyperintensity previously seen in the deep white matter of the right MCA territory was still observed, with hypointensity on the ADC map (Fig. 1D). The previously occluded right ICA had not recanalized on MRA (Fig. 2D). One week later, follow-up MRI was performed; the hyperintensity in the deep white matter of the right MCA

territory persisted, and decreased cerebral blood flow and cerebral blood volume and delayed mean transit time and time to peak were observed in the same area on the perfusion MRI (Fig. 3).

## DISCUSSION

Two points are of particular interest in this case. First, to our knowledge, this is the most persistent case of well-known characteristic MRI findings of acute ischemic infarction, hyperintensity on DWI with hypointensity on the ADC map. Second, this is the first report on these persistency occurred in the patient of total territorial infarction. In the previously reported cases, all of the persistent hyperintensity on DWI with hypointensity on the ADC map were in cases of minor stroke that involved the cerebral white matter and watershed infarction.

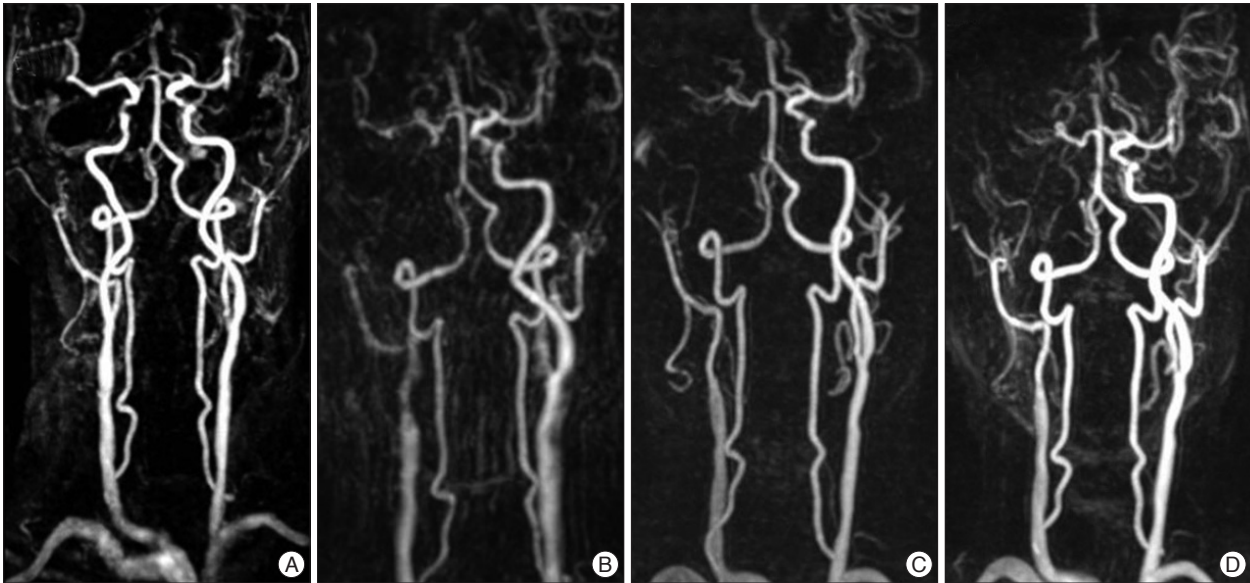
Lesions that are hyperintense on DWI and hypointense on the ADC map are characteristic findings of acute infarction on MRI. The DWI and ADC appearance reflect pathological changes in the infarcted brain tissue. The reduced ADC value in the acute stage is associated with cytotoxic cell swelling, and the subsequent increase in the ADC value reflects the evolution of vasogenic edema and cell necrosis.<sup>1)</sup> Finally, in the chronic stage, the increased ADC value indicates complete cell necrosis and permanent tissue loss.<sup>1)</sup>



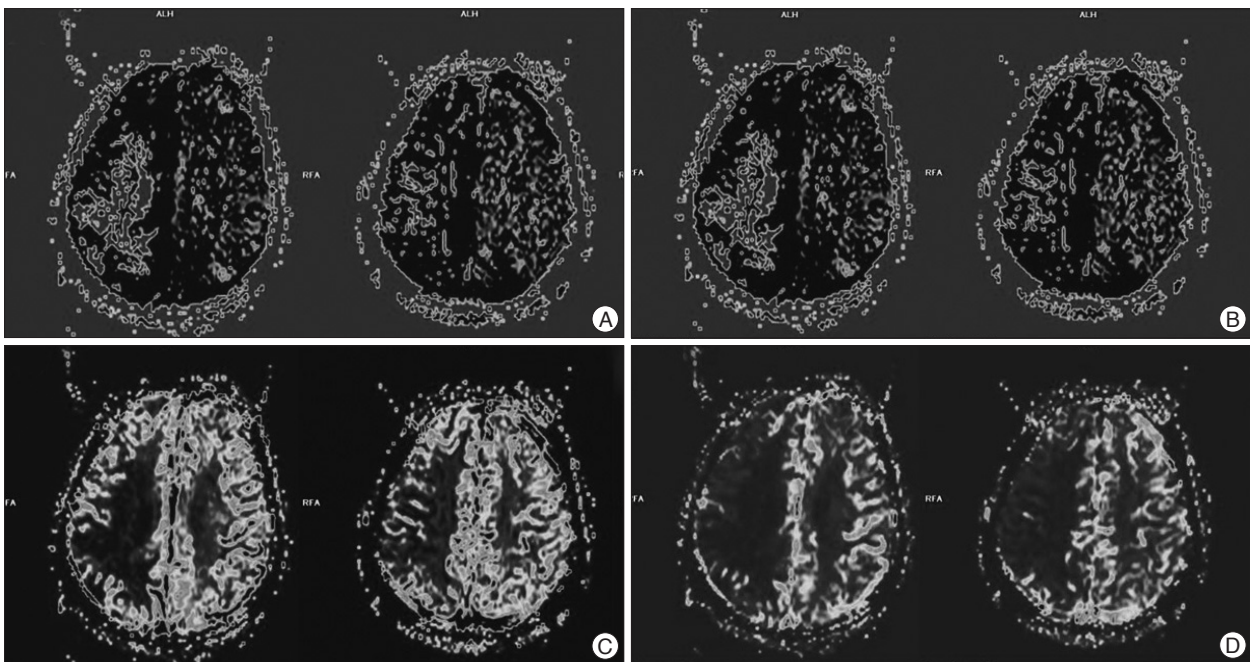
**Fig. 1.** Serial MRI. Each series includes axial T2WI, FLAIR, DWI, and ADC map : (A) obtained 141 days before the MCA infarction. T2WI and FLAIR image shows the multiple ischemic lesions on the bilateral deep white matter area but no evidence of acute infarction on DWI and ADC map; (B) obtained on the initial day of MCA infarction, 2 days before infarction progression. Note the newly appeared multiple acute infarctions on the right MCA territory; (C) obtained 72 days after complete infarction; Note the persistent hyperintense on DWI and hypointense on the ADC map in the deep white matter of previously infarcted right MCA territory; (D) obtained 227 days after complete infarction. The lesion of hyperintensity on DWI and hypointensity on the ADC map in the white matter is still persistent.

The cause of the persistent hyperintensity on DWI with hypointensity on the ADC map is still unclear. It is thought that slower ischemic tissue repair is the best explanation.<sup>2,3,6-8</sup> In a serial MRI study of small cortical and lacunar ischemic lesions, two of 21 lesions showed high DWI signals with an ADC below normal 54-144 days after infarction onset.<sup>2</sup> This was thought due to completely absent blood flow to the infarcted tissue that delayed the travel of metabolites for exchange with normal neighboring tissue.<sup>2</sup> An MRI study of the subtypes of MCA-

territory infarction showed that deep watershed infarction had a longer duration of ADC decline than did territorial infarction.<sup>3</sup> It was postulated that slower metabolic and perfusion changes in watershed infarction were the cause of the longer ADC decline, more than 30 days after symptom onset.<sup>3</sup> A DWI study performed during the subacute stage of stroke (median time 21 days) identified five cases of persistent hyperintensity on DWI with hypointensity on ADC.<sup>7</sup> One patient had a watershed infarction, and the other four had severe leukoaraiosis.<sup>7</sup>



**Fig. 2.** Serial MRA : (A) 141 days before the occurrence of the MCA infarction; (B) on the initial day of the MCA infarction, 2 days before infarction progression, complete occlusion of the entire right ICA is observed; (C) 72 days after complete infarction; and (D) 227 days after complete infarction. The previously occluded right ICA is not recanalized.



**Fig. 3.** Perfusion MRI obtained 234 days after the complete infarction : (A) MTT, (B) TTP, (C) CBV, and (D) CBF. Note the delays MTT and TTP and decreases CBV and CBF in the deep white matter of the right MCA territory.

As leukoaraiosis may reflect chronic hypoperfusion, slower evolution due to hypoperfusion explains the finding.

The meaning of the persistent hyperintensity on DWI map is also still unclear. On a serial DWI/Perfusion MRI study, authors suggested the possibility of better functional outcome through the later intervention.<sup>6)</sup> In this case, the hyperintensity lesion on DWI was remained under the inadequate perfusion state on perfusion MRI even 8 months after infarct onset. And, the hyperintensity lesion on DWI and the hypoperfusion area on perfusion MRI were nearly identical, in the deep white matter of the right MCA territory. We think the persistent hyperintensity lesion on DWI may reflect more the status of infarcted tissue repair or infarct evolution than the persistent state of tissue-at-risk.

The preference for white matter involvement was explained by differences in the evolution of the ADC value in white matter compared with gray matter.<sup>5)</sup> In the case of infarction due to large artery occlusion, longstanding flow depletion is unlikely because of the high probability of collateral flow.<sup>2)</sup> In our case, the patient suffered total territorial infarction of the right MCA due to ICA occlusion. We think that the restriction of the hyperintense lesion to the deep white matter reflected a preference for white matter involvement as well as inadequate perfusion on perfusion MRI.

## CONCLUSION

Although this report contains limited information collected from single patient, we think that the serial MR images of this patient provide important information on the evolution of infarcted tissue.

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