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Short communication

Reversible diffusion-weighted lesion in a TIA patient without arterial recanalization: A case report

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Abstract

A 70-year-old man with right hemiparesis (NIHSS score 15) was admitted to our hospital 1 h after onset. Diffusion-weighted imaging (DWI) revealed a hyperintense lesion in the left corona radiata and magnetic resonance angiography (MRA) revealed occlusion of the left middle cerebral artery (MCA). At 2.5 h after onset, his neurological deficits dramatically improved (with NIHSS score change from 15 to 2). Immediately thereafter, follow-up MRI revealed that the hyperintense lesion on DWI had disappeared, though the left MCA occlusion remained. By the end of follow-up MRI examination, his neurological deficits had completely disappeared. We report here the patient with transient ischemic attack with a reversible ischemic lesion on DWI without early arterial recanalization. © 2008 Elsevier B.V. All rights reserved.

Keywords: Transient ischemic attack; Diffusion-weighted; Spectacular shrinking deficit

1. Introduction

Diffusion-weighted magnetic resonance imaging (DWI) abnormalities have been found in the acute phase in several patients with transient ischemic attack (TIA) [1-4]. In TIA patients, early recanalization should result in disappearance of neurological deficits and abnormalities on DWI. We report here a TIA patient with a reversible ischemic lesion on DWI without early arterial recanalization.

2. Case

A 70-year-old right-handed man was admitted to our hospital after 1 h of weakness in the right arm and leg. His past medical history was hypertension for 10 years and hyperlipidemia not treated with medication. On physical examination, his height was 168 cm and body weight 65.5 kg. Body temperature was 36.6 $^{\circ}$ C, blood pressure 170/ 104 mmHg, and heart rate 68 bpm with regular sinus rhythm. No neck or orbital bruits were present, and peripheral arterial pulsations were palpable bilaterally. The heart sounds were clear without audible murmurs.

On neurological examination, he was alert. He could not speak spontaneously, but could respond normally to all commands. The pupils were round and equal, and light reflexes were prompt bilaterally. Both eyes were in the midline, and ocular movements were normal in all directions. A right central facial palsy was present. Motions of the tongue, jaw, and palate were normal. The right arm exhibited flaccid paralysis. Slight spontaneous movement remained in the right lower extremity. Sensation was slightly impaired on the right side. No extensor plantar response was present in the right foot. The National Institutes of Health stroke scale (NIHSS) score was 15 on admission.

Laboratory examinations on admission revealed a red blood cell count of 388×10^4 /mm³, hemoglobin 13.4 g/dl, hematocrit 38.8%, white blood cell count 5400/mm³, platelets 17.7×10^4 /mm³, D-dimer level < 0.5 (normal < 0.5 µg/ml) and

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Fig. 1. On admission, DWI revealed a hyperintense lesion in the left corona radiata (A) and MRA revealed left middle cerebral artery (MCA) occlusion (B). At 2.5 h after onset, the hyperintense lesion in the left corona radiata on initial DWI had completely disappeared (C). Follow-up MRA revealed persistent MCA occlusion (D). At 24 h after onset, DWI revealed multiple hyperintense lesions in the left MCA area (E) and the left MCA had completely recanalized (F).

level of thrombin-antithrombin III complex (TAT) 1.5 ng/ml (normal < 3.0 ng/ml). No liver or renal dysfunction was noted.

Seventy minutes after onset, he was examined using a 1.5-tesla magnetic resonance imaging (MRI) unit (Signa Echo Speed Horizon; GE Medical Systems, Milwaukee, WI). DWI revealed a hyperintense lesion in the left corona radiata (Fig. 1A). When we placed one small region of interest (ROI) on the affected area and contralateral region (mirror image) on initial DWI, the apparent diffusion coefficient (ADC) was lower in the left corona radiata than on the control side $(0.52 \times 10^{-3} \text{ mm}^2/\text{s})$ on the left side and $0.70 \times 10^{-3} \text{ mm}^2/\text{s}$ on the right). Compared with the right side, the decrement in ADC (ADC ratio) on the left side was 0.74. MR angiography (MRA) revealed an occlusion of the left middle cerebral artery (MCA) (Fig. 2A).

We considered administration of intravenous thrombolysis with rt-PA. However, his neurological symptoms dramatically improved from an NIHSS score of 15 to 2 within 40 min of arrival at the hospital, and he therefore did not undergo thrombolysis. When we found the clinical improvement of this patient, blood pressure was 166/ 102 mm Hg. We performed follow-up MRI 2.5 h from onset. The hyperintense lesion in the left corona radiata on initial DWI had completely disappeared (Fig. 1C), but the occlusion of the left MCA remained (Fig. 1D). By the end of follow-up MRI examination, his neurological deficits had recovered completely. We therefore diagnosed transient ischemic attack (TIA).

At 24 h after onset, MRA demonstrated complete recanalization of the left MCA. DWI revealed multiple hyperintense lesions in the left MCA area, but he had no symptoms. Holter 24-hour electrocardiography revealed

sinus rhythm without atrial fibrillation. Transesophageal echocardiography revealed the presence of a patent foramen ovale and atrial septal aneurysm. Carotid Doppler ultrasonography detected no arterial lesions. Although deep venous thrombosis was absent in his lower extremities, it appeared that TIA had contributed to paradoxical brain embolism in association with a right-to-left shunt. Finally, we considered him as clinical TIA because he had only asymptomatic lesions on DWI.

3. Discussion

We have reported here a patient with TIA with a reversible ischemic lesion on DWI without early arterial recanalization.

Dramatic neurological improvement immediately after acute hemispheric stroke has been described as spectacular shrinking deficit (SSD) following the rapid migration of an embolus [5]. However, follow-up MRA revealed persistent MCA occlusion despite dramatic neurological recovery. If we had performed follow-up MRI only at 24 h after onset, we would have suspected that the neurological improvement was due in part to the arterial early recanalization, as in a previous report [6].

In our case, the hyperintense lesion on initial DWI disappeared on follow-up DWI study. At 24 h after onset, DWI revealed multiple hyper-intense lesions in the affected arterial territory consistent with MCA recanalization. Several case series have reported that DWI hyperintense lesions disappeared in the sub-acute phase after onset [3,4]. While 20 to 70% of TIA patients had abnormal DWI lesions on admission [1–4], half of these lesions appear to have resolved by the time of follow-up study [4]. Early recanalization might

be thought to result in disappearance of abnormalities on DWI. However, in our case, the occluded artery did not reopen when the hyper-intense lesion on DWI disappeared. We believe that collateral flow like a leptomeningeal anastomosis developed immediately after arterial occlusion.

Low ADC ratio in the lesion is reported to be associated with irreversible ischemia, with a cut-off value of 0.82 [7]. In our patient, however, the ADC ratio was 0.74, inconsistent with this previous report [7]. Our findings are thus also inconsistent with the conclusion that low ADC value is associated with permanent ischemia.

In conclusion, we have reported a patient with TIA without early recanalization of the occluded artery.

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