Neuroradiology (2002) 44: 235–238
DOI 10.1007/s002340100699

M. Matsui
T. Imamura
S. Sakamoto
K. Ishii
H. Kazui
E. Mori

Transient global amnesia: increased signal intensity in the right hippocampus on diffusion-weighted magnetic resonance imaging

Received: 15 April 2001
Accepted: 18 June 2001
Published online: 7 November 2001

M. Matsui (✉) · S. Sakamoto · K. Ishii
Division of Neuroimaging Research,
Hyogo Institute for Aging Brain and
Cognitive Disorders, 520 Saisho-Ko,
Himeji, Hyogo 670-0981, Japan
E-mail: matsui@hiabcd.go.jp
Tel.: +81-792-955511
Fax: +81-792-958199

T. Imamura · H. Kazui · E. Mori
Division of Clinical Neurosciences,
Hyogo Institute for Aging Brain
and Cognitive Disorders,
Hyogo, Japan

Abstract We report on a patient with pure transient global amnesia (TGA) whose magnetic resonance imaging (MRI) demonstrated a small region of increased signal intensity in the right hippocampus on diffusion-weighted imaging (DWI). DWI was sensitive and useful for evaluating the early stage of TGA and might help to explain the pathophysiology of TGA.

Keywords Transient global amnesia · Magnetic resonance imaging · Diffusion-weighted imaging

Introduction

Transient global amnesia (TGA) is a syndrome which is characterized by a sudden-onset and transient memory disturbance with preserved alertness, attention and personal identity [1, 2, 3]. Although the pathogenesis and etiology of TGA still remain uncertain, three recent MRI studies [4, 5, 6] reported that diffusion-weighted imaging (DWI) might detect early parenchymal tissue changes, especially in the medial temporal regions. In this paper we present a patient with a transient amnestic attack which fulfilled the established diagnostic criteria of TGA [1, 2, 3], in whom DWI showed a small region of increased signal intensity in the right hippocampus 44 h after the onset. Our current findings and a review of previous reports suggest that DWI might help us to understand the pathogenesis of TGA and might be useful for clinical diagnosis and management.

Case report

A 63-year-old, right-handed retired office worker without a history of migraine, epilepsy or head injury was referred to us with an episode of amnesia. His wife found him well 1 h before the onset, and then he suddenly began to ask repetitive questions about subjects that he should have known about. For example, on seeing a package containing a computer printer that he had ordered 2 days before, he repeatedly asked his wife, "What is in this package?" and "Who ordered this?" He found a current volume of his favorite magazine that he had bought 1 week before and repeatedly asked, "Why is this here?" He understood his wife's explanations but immediately forgot them. He could identify himself and his family members, but he could not remember that he had gone to church earlier in the day. The patient continued to ask repetitive questions of a general physician at the presentation 4 h after the onset. The physician described him as alert, with preserved identity, but amnestic. A brain CT was normal. His amnesia and repetitive questioning gradually resolved. After he had stayed overnight at the hospital, his symptoms subsided except for the loss of the memory during the attack. At the initial presentation to us, 42 h after the onset, he was alert and well oriented. General physical and

neurological examinations were normal. He scored 30/30 on the Mini Mental State Examination (MMSE). The scores on the Wechsler Memory Scale Revised (WMS-R) were as follows: verbal memory index, 110; visual memory index, 106; general memory index, 108; attention index, 90; and delayed recall index, 104. A neuropsychological interview showed that his amnestic gap was from 1 h before to 5 h after the onset of the attack. An electroencephalogram (EEG) was normal. He underwent an MRI examination 44 h after the onset. Images were obtained on a 1.5-T unit capable of echo-planar imaging (GE Signa Advantage version 5). DWI revealed a small region of increased signal intensity in the right hippocampus (Fig. 1D). There was also a corresponding region of high signal intensity on the conventional T2-weighted image (T2WI) (Fig. 1C). He showed no recurrence of amnestic attack. There were no signal abnormalities in any of the sequences of the follow-up MRI examinations 2 weeks and 3 months after the onset (Fig. 2).

Discussion

Though this patient did not undergo a detailed neuropsychological assessment during the attack, the diagnosis was definite TGA based on the clinical diagnostic criteria [1, 2, 3], which consisted of the presence of a witness and absence of the following: clouded consciousness, loss of personal identity, cognitive impairment other than amnesia, and focal neurological symptoms or history of head injury or epilepsy. An important differential diagnosis is epilepsy, but in most patients with epilepsy that resembles TGA, the amnesia lasts less than 1 h [3].

Several neuroradiological studies of TGA patients have been conducted, and recently, the usefulness of DWI in MRI has been discussed. Strupp et al. [4], using DWI, found high signal intensity in the left or bilateral medial temporal regions in patients during or immediately after a TGA attack. The authors found these observations to be consistent with the symptoms of TGA because medial temporal lesions may cause disturbances of episodic memory. They also noted the absence of signal abnormalities in follow-up MRI using T2WI and DWI, and reported that the pathogenesis of TGA is not of an ischemic nature. They attributed the high signal intensity to extracellular edema and interstitial narrowing between the cells caused by physiological neural dysfunction due to spreading depression [7].

On the other hand, Woolfenden et al. [5] described a patient who suddenly developed TGA-like amnesia after cerebral angiography. In the MRI of this patient, DWI and T2WI showed high signal intensities in several areas including the right hippocampus and bilateral occipital lobes. The signal abnormality completely disappeared 2 weeks after the attack. The authors reported that the alteration in the signal intensity was transient because

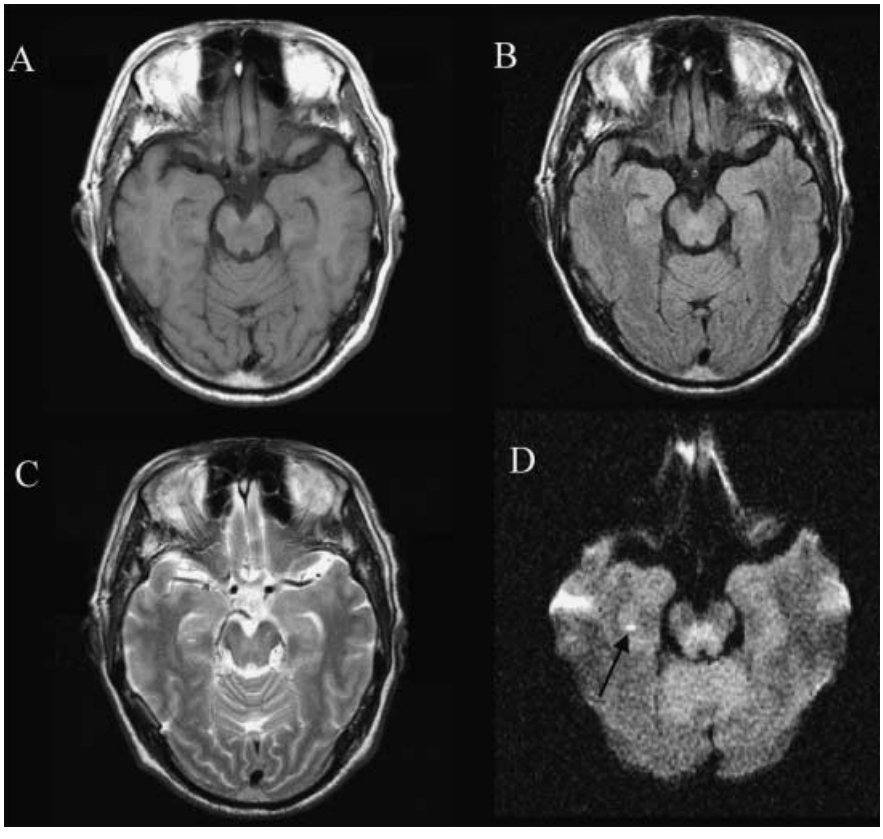

Fig. 1A–D. Initial MRIs acquired 44 h after the onset, showing the same level at a 5-mm slice thickness with 2.5-mm gap covering medial temporal lobe structures. **A** Conventional T1-weighted image (T1WI) (TR/TE/NEX = 500/13/2, FOV: 20×20 cm, matrix: 256×256). **B** FLAIR image (TR/TE/NEX = 9,000/147/1, FOV: 20×20 cm, matrix: 256×256). **C** Conventional T2WI (TR/TE/NEX = 3,000/105/2, FOV: 20×20 cm, matrix: 256×256). **D** DWI (TR/TE/NEX = 2,000/118/1, FOV: 24×24 cm, matrix: 256×256, b-value: 1, 000 s/mm^2). The conventional T1WI (**A**) and FLAIR image (**B**) are almost normal, but the conventional T2WI (**C**) and DWI (**D**) demonstrate the same small regions of increased signal intensity in the right hippocampus

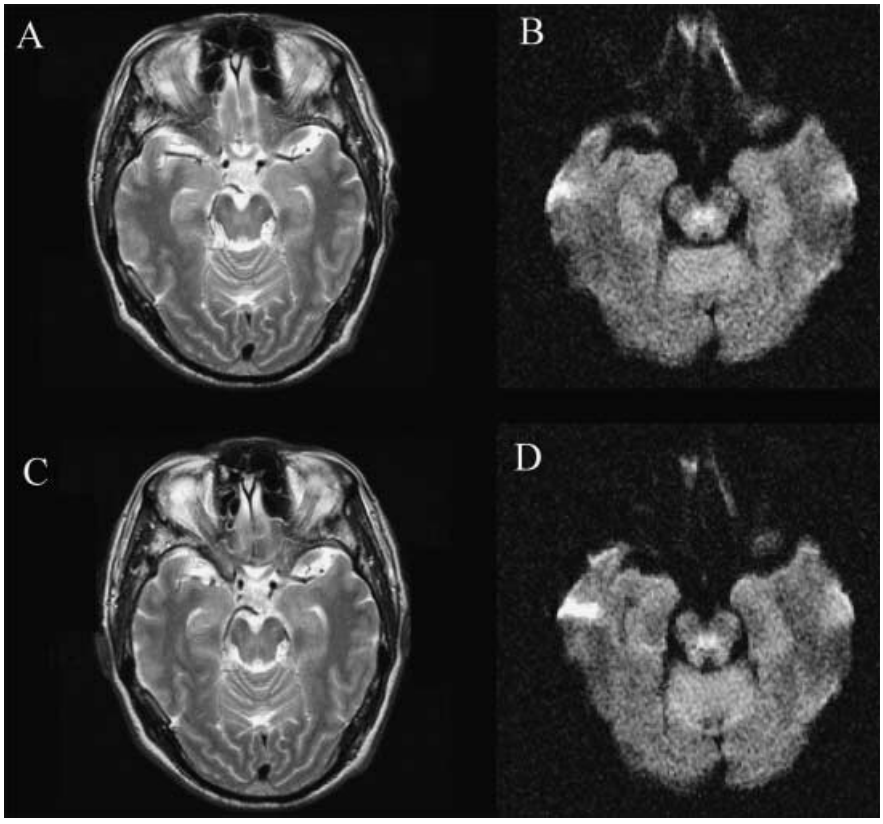

Fig. 2A–D. Follow-up MRIs. The top row (**A, B**) and the bottom row (**C, D**) show the images acquired 2 weeks, and 3 months, after the onset, respectively. None of the sequences shows any signal abnormalities

the infarct area caused by the ischemia was too small to be detected by follow-up MRI. The MRI findings in the current patient were similar to those of Woolfenden et al. [5] in that he had a very small and localized lesion that was observed as a high signal intensity on T2WI.

However, other studies failed to detect signal alterations in magnetic resonance DWI in TGA patients. Ay et al. [6] reported a TGA patient with normal DWI who had vascular risk factors, and Budson et al. [8], using both DWI and perfusion-weighted MRI, also reported a TGA patient with normal results. They suggested that the patients with a diagnosis of TGA might be heterogeneous. Two other studies that examined TGA patients [9, 10] attempted and failed to show abnormalities in DWI and apparent diffusion coefficients (ADC).

The findings of our study and previous reports suggest that DWI is useful for the clinical diagnosis of TGA patients. Our case appeared to support the pathophysiology of TGA described by Woolfenden et al. [5]. However, the pathogenesis of the signal alterations and the sensitivity and specificity for the diagnosis are still uncertain. Further clinical studies and advances in neuroimaging technology might establish the usefulness of DWI in the early diagnosis and documentation of the possible heterogeneity of TGA.

References

1. Caplan LR (1990) Transient global amnesia: characteristic features and overview. In: Markowitsch HJ (ed) Transient global amnesia and related disorders. Hogrefe & Huber, Toronto, pp 15–27
2. Hodges JR, Warlow CP (1990) The aetiology of transient global amnesia: a case control study of 114 cases with prospective follow-up. Brain 113:639–657
3. Hodges JR, Warlow CP (1990) Syndromes of transient amnesia: towards a classification. A study of 153 cases. J Neurol Neurosurg Psychiatry 53:834–843
4. Strupp M, Bruning R, Wu RH, et al. (1998) Diffusion-weighted MRI in transient global amnesia: elevated signal intensity in the left mesial temporal lobe in 7 of 10 patients. Ann Neurol 43:164–170
5. Woolfenden AR, O'Brien MW, Schwartzberg RE, et al. (1997) Diffusion-weighted MRI in transient global amnesia precipitated by cerebral angiography. Stroke 28:2311–2314
6. Ay H, Buonanno FS, Rordorf G, et al. (1999) Normal diffusion-weighted MRI during stroke-like deficits. Neurology 52:1784–1792

7. Olesen J, Jorgensen MB (1986) Leao's spreading depression in the hippocampus explains transient global amnesia. Acta Neurol Scand 73:219–220
8. Budson AE, Schlaug G, Briemberg HR (1999) Perfusion- and diffusion-weighted magnetic resonance imaging in transient global amnesia. Neurology 53:239–240
9. Ay H, Furie KL, Yamada K, Koroshetz WJ (1998) Diffusion-weighted MRI characterizes the ischemic lesion in transient global amnesia. Neurology 51:901–903
10. Gass A, Gaa J, Hirsch J, et al. (1999) Lack of evidence of acute ischemic tissue change in transient global amnesia on single-shot echo-planar diffusion-weighted MRI. Stroke 30:2070–2072