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Case Report

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A Case With Bilateral Hippocampal Infarction Resembling Transient Global Amnesia

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Abstract

Transient global amnesia (TGA) is a benign and transient condition with a sudden short-term amnesia. One of the conditions resembling TGA is hippocampal infarction, which requires relapse prevention treatments. In this report, we present a case with bilateral hippocampal infarction in whom distinguishing these two conditions was difficult for up to 1 week from the onset. A 60-year-old female visited our hospital with sudden onset retrograde and anterograde amnesia. Thin-slice magnetic resonance imaging (MRI) with 2-mm thickness revealed hyperintense signals on diffusion-weighted imaging (DWI) with signal loss on apparent diffusion coefficient (ADC) on both sides of the hippocampus. MRI with 5-mm thickness on day 7 revealed persistent restricted diffusion on both sides, one of which was still with decreased ADC values. Based on this finding, the diagnosis of bilateral hippocampal infarction was reached, and the relapse-preventive antiplatelet was continued. This case implied the potential difficulty of distinguishing cases with TGA and those with hippocampal infarction based on MRI findings within the first several days after onset. Thin-slice brain MRI, careful search of potential cardiovascular risks, and follow-up MRI ≥ 7 days after onset will be helpful to reach a correct diagnosis in cases with sudden amnesia.

Keywords: Apparent diffusion coefficient; Diffusion-weighted imaging; Hippocampal infarction; MRI; Transient global amnesia

Introduction

Transient global amnesia (TGA) is a benign transient amnesia with sudden onset, typically involving middle-aged individuals and usually requiring no relapse prevention [1-3]. Conditions that should be discriminated from this benign condition include hippocampal infarction, which may require antiplatelets to prevent future relapses [4]. Currently, the diagnosis of TGA largely depends on clinical findings, such as the diagnostic criteria by Hodges et al [2]. The exact diagnostic criteria to discriminate TGA from hippocampal infarction based on magnetic resonance imaging (MRI) findings remains uncertain [5, 6]. In ischemic neurological conditions with cellular damages based on compromised blood flow, including hippocampal infarction, signal loss on apparent diffusion coefficient (ADC) is an important MRI finding in addition to the diffusion-weighted imaging (DWI)-high signals to estimate perfusion status [7]. However, signal loss on ADC can also be seen in cases of TGA [8-10]. A promising MRI finding to discriminate hippocampal infarction from TGA is the involvement of posterior cerebral artery (PCA) territories other than hippocampal lateral portion corresponding to the CA1 region [11]. Meanwhile, when such extra-hippocampal lesions are absent, discriminating these two conditions are often difficult. Moreover, the detection of hippocampal lesions on MRI with 5-mm-thick slices is often difficult, as the size of hippocampal lesions are often smaller than 5 mm [12]. Consequently, some cases with sudden amnesia must be followed up for more than 1 week to reach a correct diagnosis. In this report, we present a patient initially suspected of TGA based on brain MRI findings on hospital admission, but later diagnosed with bilateral hippocampal infarction based on MRI findings 1 week later.

Case Report

Investigations

A 60-year-old female with the past medical histories of hypertension and dyslipidemia, treated with pitavastatin, visited our hospital at 6:00 pm with sudden onset amnesia started from 1:00 pm on the same day, which was noticed by her colleague at the workplace. Her colleagues reported that she was normally working before the lunch break, but she suddenly started to repeat that "what am I doing here?" and "how did I get to this place today?" at approximately 1:00 pm. She was also reported to have forgotten whether she had finished lunch. She had no previous history of alcohol or cocaine use disorder. The day was in the midsummer season and the highest air temperature of the day before the onset was higher than 35 °C in the area,

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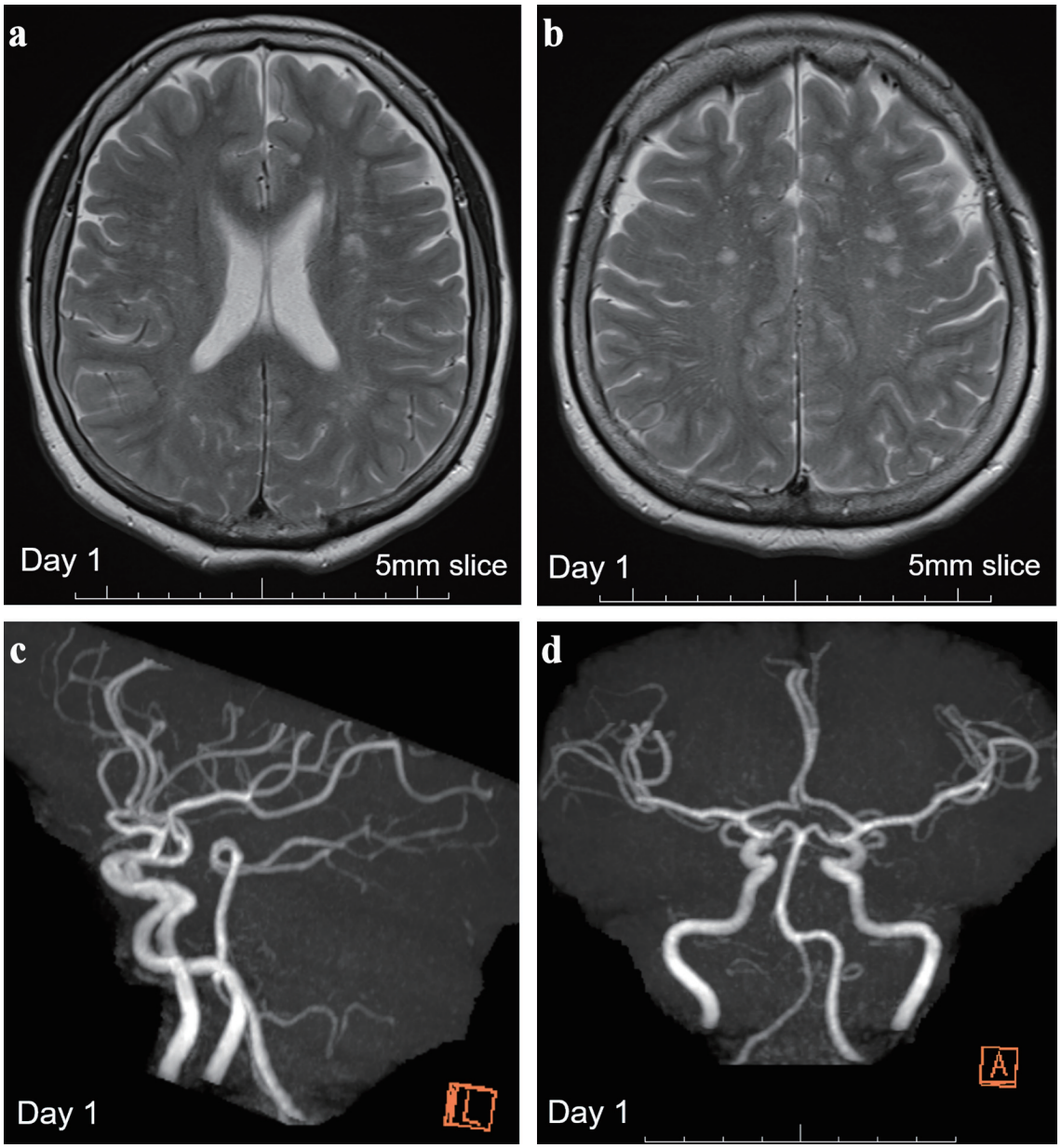

Figure 1. Brain MRI showing multiple old ischemic changes and MR angiography on day 1. (a, b) T2-weighted images with 5-mm-thick brain MRI on day 1 revealed multiple old ischemic changes in both hemispheres. (c, d) Normal MR angiography findings with a patent posterior circulation system. MRI: magnetic resonance imaging; MR: magnetic resonance.

when she worked outside all day to guide visitors from other areas (day -1). Upon the initial neurological examination on arrival at hospital, her consciousness level was almost clear, and she showed no neurological deficits. Her memorization ability appeared to have already returned to normal on admission at 6:00 pm, but the memory from approximately 8:00 am of the same day to 5:00 pm was kept lost, suggesting the presence of both retrograde and anterograde amnesia. She also temporarily noticed tingling sensations in the periphery of four limbs before arriving at the hospital. Vital signs on arrival were normal. The blood test suggested the presence of mild dehydration that may predispose the risk of ischemic stroke, with the blood urea nitrogen (BUN) level of 10.8 mg/dL and the serum creatinine level of 0.50 mg/dL, yielding a BUN/creatinine ratio greater than 20 [13, 14]. Serum N-terminal pro-brain natriuretic peptide (NT-proBNP) level was slightly elevated with 157 pg/mL.

Diagnosis and treatment

In the day of the onset, multiple old ischemic changes were confirmed in both hemispheres, but no DWI-high signals were confirmed with 5-mm-thick MRI imaging (Fig. 1), obtained using a Siemens MAGNETOM Symphony 1.5 Tesla MRI

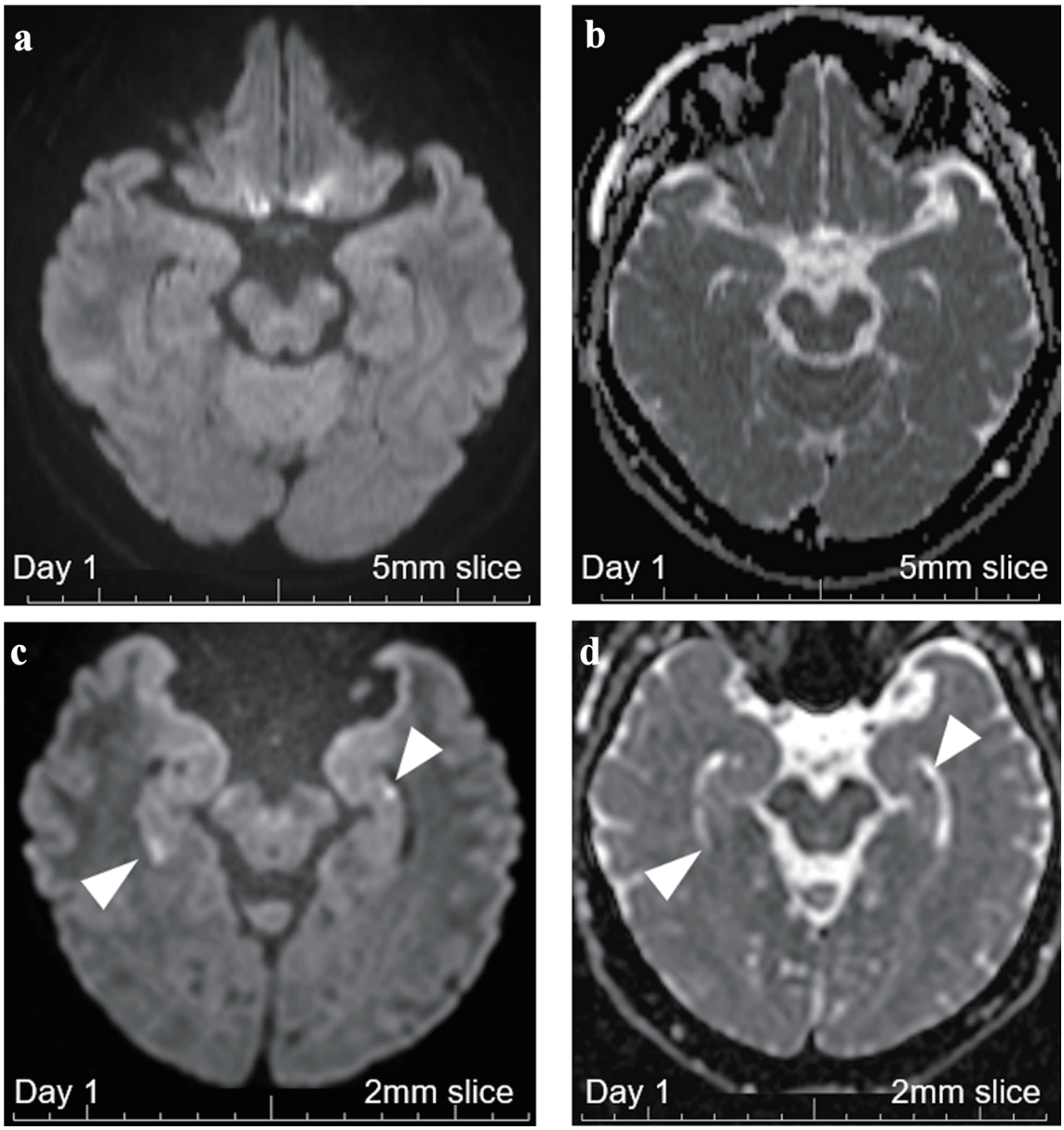

Figure 2. Brain MRI with 5-mm thickness and 2-mm thin-slice on day 1. (a) DWI with 5-mm-thick brain MRI on day 1 revealed no abnormalities. (b) ADC with 5-mm-thick brain MRI on day 1 revealed no abnormalities. (c) DWI with 2-mm thin-slice brain MRI on day 1 revealed two hippocampal signal high lesions each on both sides (white arrowheads). (d) ADC with 2-mm thin-slice brain MRI on day 1 revealed signal losses for the two DWI-high hippocampal lesions (white arrowheads). MRI: magnetic resonance imaging; ADC: apparent diffusion coefficient; DWI: diffusion-weighted imaging.

system. The magnetic resonance (MR) angiography revealed no abnormalities, such as reversible cerebral vasoconstriction syndrome, with patent PCAs on both sides [15, 16]. An additionally performed 2-mm thin-slice MRI (day 1) revealed bilateral hyperintense DWI signals with signal losses on ADC in the hippocampal lateral portions corresponding to CA1 areas on both side (Fig. 2), indicating the presence of cellular damages and the potential diagnosis of bilateral hippocampal infarction. At this point, both TGA and bilateral hippocampal infarction were possible, and intravenous argatroban for 7 days (60 mg/day for 2 days, followed by 20 mg/day for 5 days) was decided to be temporarily started.

On day 2, her lost memories of the previous day between 8:00 am and 5:00 pm were kept lost, but she fully remembered the episodes after arriving at the hospital. She had a reduction in appetite and could not eat solid food at breakfast. A 2-mm thin-slice MRI was performed again. This time, the signal loss on ADC became more conspicuous (Fig. 3). Additional extra-hippocampal lesions were still absent. Carotid ultrasonography and cardiovascular MRI, covering the aortic arch level, identified no apparent thromboembolic risks like unstable plaques. Even at this stage, both possibilities of TGA

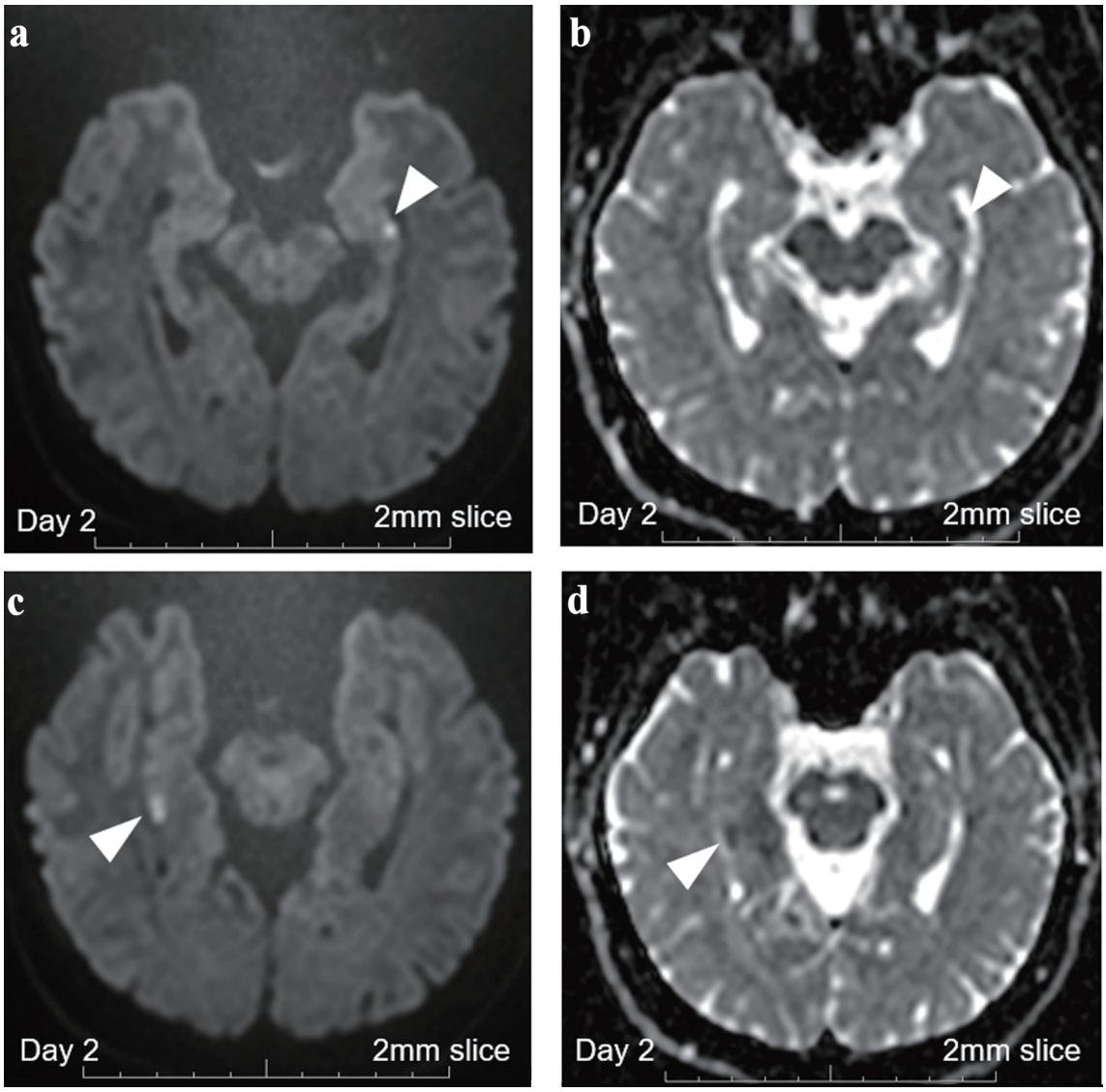

Figure 3. Brain MRI with 2-mm thin-slice on day 2. (a) DWI with 2-mm thin-slice brain MRI on day 2 revealed persistent punctate hyperintense DWI signals in the left hippocampus (white arrowhead). (b) ADC with 2-mm thin-slice brain MRI on day 2 revealed persistent punctate signal loss in the left hippocampus (white arrowhead). (c) DWI with 2-mm thin-slice brain MRI on day 2 revealed persistent high signals in the right hippocampus (white arrowhead). (d) ADC with 2-mm thin-slice brain MRI on day 2 revealed persistent signal loss in the right hippocampus (white arrowhead). MRI: magnetic resonance imaging; ADC: apparent diffusion coefficient; DWI: diffusion-weighted imaging.

and bilateral hippocampal infarction still remained.

On day 7, a 5-mm-thick MRI revealed persistent hyperintense DWI signals on both hippocampi, one of which was still with a signal loss on ADC (Fig. 4). These findings of persistent MRI abnormalities even after 3 - 6 days after the onset indicated the diagnosis of bilateral hippocampal infarction. Based on these findings, antiplatelet therapy was decided to be continued, and dual antiplatelet therapy (DAPT) with aspirin 100 mg/day and clopidogrel 75 mg/day was started. Other additional diagnostic examinations including the 24-h Holter monitoring and echocardiogram revealed no suspectable thrombotic risks. The Wechsler Memory Scale-Revised test on day 12 revealed an impaired verbal short-term memory (Table 1). The patient did not present with nausea or vomiting during the hospitalization. She left the hospital on day 15, and DAPT was switched to single antiplatelet therapy by aspirin 100 mg/day. Her lost memory between 8:00 am and 5:00 pm on the day of onset was not recovered at last. She has had no relapses with a single oral antiplatelet as of 6 months from the amnestic event.

Discussion

In this case report, a middle-aged woman with sudden amnesia was reported, for whom making the correct diagnosis based on clinical and MRI findings in the first several days was difficult.

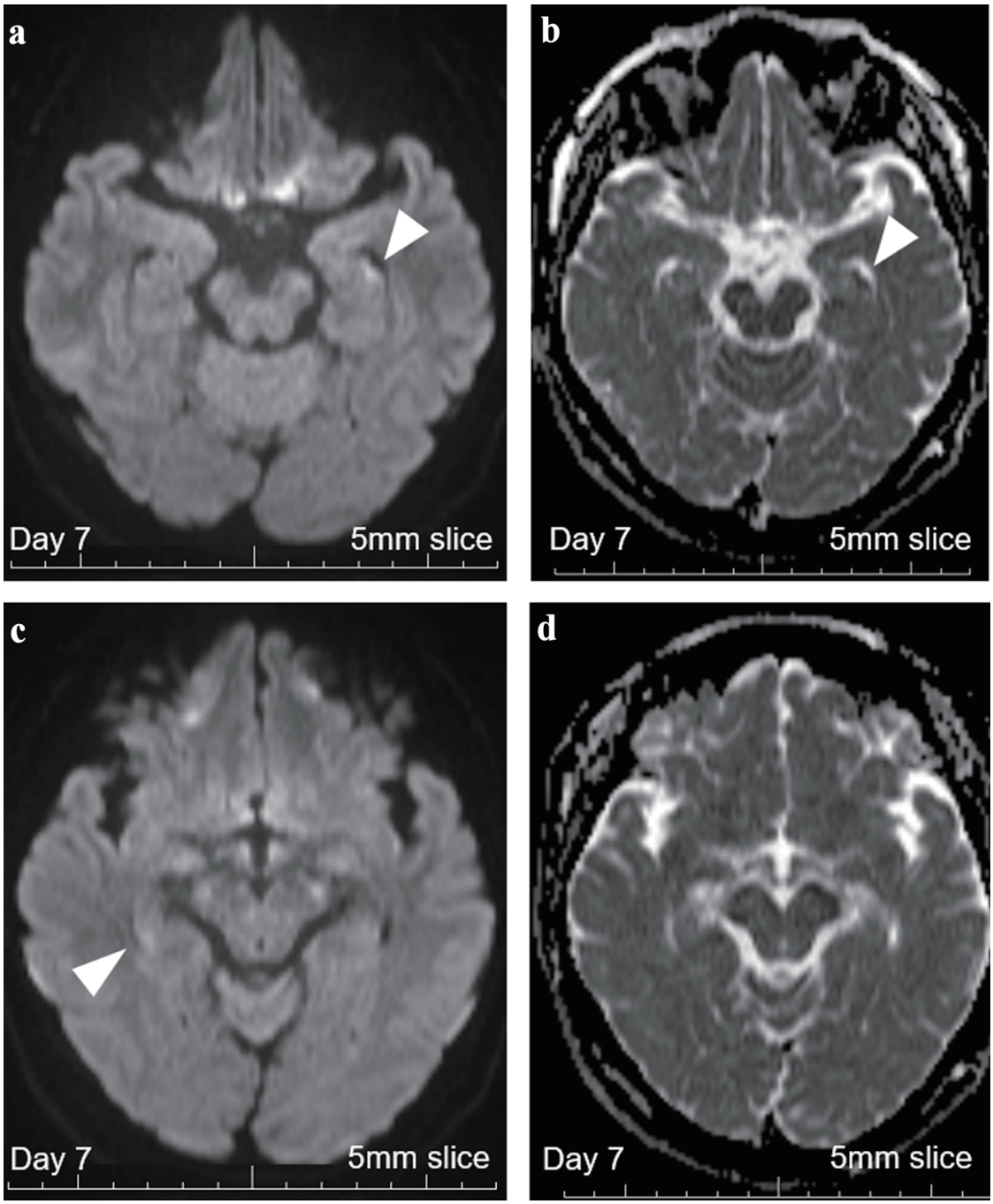

Figure 4. Brain MRI with 5-mm thickness on day 7. (a) DWI with 5-mm-thick brain MRI on day 7 revealed persistent high signals in the left hippocampus (white arrowhead), increasing the probability of hippocampal infarction. (b) ADC with 5-mm-thick brain MRI on day 7 revealed persistent signal loss in the left hippocampus (white arrowhead). (c) DWI with 5-mm-thick brain MRI on day 7 revealed persistent vague high signals in the right hippocampus (white arrowhead). (d) ADC with 5-mm-thick brain MRI on day 7 revealed no apparent signal loss, suggesting that cellular damages were milder on this side. MRI: magnetic resonance imaging; ADC: apparent diffusion coefficient; DWI: diffusion-weighted imaging.

Her clinical symptom and neuroimaging findings in the first week from the onset were compatible with those in TGA. Because the possible diagnosis of bilateral hippocampal infarction could not be ruled out, antiplatelet therapy was started. Based on the persistent diffusion restrictions with decreased ADC values on day 7, we made the final diagnosis of bilateral hippocampal infarction, and antiplatelet therapy was decided to be continued. Persistent deficits of verbal memory on day 12 further supported the diagnosis of hippocampal infarction rather than TGA [17-21]. This case report emphasizes the im-

Table 1. Results of the Wechsler Memory Scale-Revised Test (WMS-R) on Day 12

Subtests	I (immediate recall)	II (delayed recall)
Information and orientation	14/14	
Mental control	6/6	
Figure memory	8/10	
Logical memory (A)	14/25	14/25
Logical memory (B)	10/25	9/25
Visual paired associates	14/18	6/6
Verbal paired associates (related/easy)	12/12	4/4
Verbal paired associates (unrelated/difficult)	2/12	4/4
Visual reproduction	40/41	41/41
Digital span[a]	(Forward) 10/12	(Backward) 10/12
Visual memory span[a]	(Forward) 12/14	(Backward) 6/12
Subscale scores		
Verbal memory indicator	102	
Visual memory indicator	113	
General memory indicator	106	
Delayed recall indicator	128	
Attention/concentration indicator	120	

The obtained results with WMS-R revealed a slightly prolonged time in verbal memorization tasks, but they could be recalled once they are memorized successfully. The results indicated that the patient may experience a slight difficulty in remembering new tasks, which may not be a significant disturbance in the daily living and working. [a]Forward task scores in the second column and backward task scores in the third column.

portance of careful medical history taking and diagnostic approaches for the individuals with sudden transient amnesia.

As the same in cases with hippocampal infarction, hyperintense DWI signals can be found in patients with TGA, especially when a thin-slice MRI is performed. Furthermore, utilizing a 7 Tesla MRI can realize a higher detection rate of DWI-high lesions in TGA, reportedly with the detection rate of 80-90% [22]. The detection rate of hyperintense DWI signals on MRI in TGA is thought to be the highest after 12 - 48 h from the onset [23]. Meanwhile, it is unestablished whether a signal loss on ADC is also an indicative finding of TGA. Many previous reports utilized the findings of signal loss on ADC in making the diagnosis of TGA [8, 10, 24, 25]. Therefore, signal loss on ADC cannot be a rationale to rule out TGA. Additional MRI abnormalities outside the hippocampal CA1 region in the PCA territories would indicate the diagnosis of hippocampal infarction.

However, in the absence of such additional extra-hippocampal lesions, like the present case, making the diagnosis based on the MRI findings is difficult. Because the CA1 region is susceptible to ischemia [12], ischemic lesions restricted to hippocampi may be possible upon dehydration and other hemodynamic changes with increased blood viscosity [26]. The number, size, or bilaterality of the hyperintense DWI signals in CA1 regions would not be determinant information for distinguishing TGA and hippocampal infarction. As the MRI abnormalities in TGA may persist up to 5 - 6 days from onset [23, 27], careful follow-up of the subsequent clinical course and MRI abnormalities for 1 week or longer are needed for making a correct diagnosis. If the MRI abnormalities persist even after 1 week from the onset, the possibility of hippocampal infarction becomes higher. If all MRI abnormalities (i.e., DWI, ADC, T2, and fluid attenuated inversion recovery (FLAIR)) vanish in the first week after the onset, the diagnosis of TGA will become more likely and the relapse-prevention antiplatelets may be unnecessary. Further studies are needed to establish MRI criteria to longitudinally distinguish the cases with punctate hippocampal infarctions and those with TGA presenting hippocampal diffusion restrictions.

Amnestic symptoms can be caused by any cerebral lesions in the areas involved in memory functions, such as cingulate gyrus and mammillary bodies comprising the Papez circuit. Previous studies reported the development of amnesia in cases with lesions in cingulate gyrus, mammillary bodies, and fornix [28-31]. Careful evaluation of these cerebral regions associated with memory functions is recommended in amnestic patients, because a small punctate lesion in any of these cerebral components including the limbic system structures may result in amnestic symptoms resembling TGA [30].

As a limitation of this case report, electroencephalogram (EEG) was not performed for this case. As an epileptic condition resembling TGA, transient epileptic amnesia (TEA) is sometimes seen typically in middle-aged and older people [32]. TEA is considered to be a rare subtype of temporal lobe epilepsy with amnesic seizure. To diagnose the condition, detection of ictal EEG abnormalities is a key finding [33]. However, the present case has not relapsed after the initial amnesic episode, and the diagnosis of TEA seems to be less likely for this case. Another limitation of this case report was that the existence of patent

foramen ovale (PFO) has not been investigated. To detect the PFO, transthoracic echocardiography with bubble contrast is among the standard diagnostic approaches [34]. As the presence of PFO is one of the causes of paradoxical cerebral embolism, cases with unknown thrombotic origin are better to be investigated for the presence of PFO not to overlook thrombotic backgrounds including deep venous thrombosis [35].

Conclusions

A case with bilateral hippocampal infarction was presented, whose clinical symptom and MRI findings on admission were totally compatible with those with TGA. In some cases, with sudden amnesia, distinguishing these two conditions within the first several days is difficult, as DWI- and ADC-abnormalities can be found in both conditions within this time window. If the possibility of hippocampal infarction cannot be confidently ruled out in the first several days after onset, relapse-preventive antiplatelet therapy may be temporarily started unless contraindicated. A follow-up MRI after 7 days from onset will aid to determine the diagnosis and decide whether to continue the antiplatelet therapy. Persistent MRI abnormalities after this time period may indicate ischemic lesions, and relapse preventions may be required.

Learning points

Although TGA is a benign condition with no required relapse preventions, all cases with sudden onset amnesia are better to be evaluated with brain MRI on day 1, day 2, and after day 7. Only reassuring the patients that they are possibly with TGA, and no therapeutic intervention are needed, based only on the episode without follow-up MRI, may be insufficient. This is because the differential diagnosis of hippocampal infarction and other resembling conditions that require specific treatments cannot be confidently ruled out within the first several days after the onset in some cases.

In addition to the usual 5-mm- or 6-mm-thick brain MRI, a thin-slice hippocampal MRI with 2-mm or 3-mm thickness is better to be performed to correctly estimate the presence and extent of cellular damages.

If hippocampal infarction cannot be ruled out because of the presence of diffusion restriction with decreased ADC values, antiplatelet therapy may be temporarily started unless contraindicated. A follow-up MRI after day 7 after the onset will aid to determine the diagnosis and decide whether to continue the antiplatelet therapy or not.

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Conflict of Interest

The authors have no conflict of interest to disclose.

Informed Consent

Written informed consent was obtained from the patient.

Author Contributions

All authors participated in patient care. TA drafted the manuscript. MA, SS, TM, and MH contributed to reviewing and revising the manuscript.

Data Availability

All data were obtained during the patient's hospitalization. Any inquiries regarding the additional information should be directed to the corresponding author.

Abbreviations

ADC: apparent diffusion coefficient; DWI: diffusion-weighted imaging; PCA: posterior cerebral artery; TGA: transient global amnesia

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