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Case Report

Clinical and neuropsychological changes after the disappearance of seizures in a case of transient epileptic amnesia

Masanori Sekimoto *, Reimi Muramatsu, Masaaki Kato, Teiichi Onuma

Musashino Kokubunji Clinic, Tokyo, Japan

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ABSTRACT

We encountered a female patient with late-onset temporal lobe epilepsy who presented with transient amnesia as the sole ictal manifestation, an accelerated rate of forgetting daily life events, and a retrograde memory deficit. We describe the memory function of the patient both before and after the administration of antiseizure medication. After the patient's seizures were controlled with antiseizure drugs, her neuropsychological memory performance scores showed improvement. We presumed that the disappearance of seizures was associated with a decrease in the accelerated rate of forgetting medication. However, her lost memories were not recovered after the seizures were controlled by antiseizure medication.

1. Introduction

Memory disturbance is common among people with temporal lobe epilepsy (TLE). Transient epileptic amnesia (TEA) [1] is a subtype of TLE and is characterized by brief, recurrent episodes of transient amnesia during which other cognitive functions are preserved [2,3]. The condition typically arises in middle and old ages. Most patients with TEA experience interictal memory difficulties such as an accelerated forgetting rate and isolated autobiographical memory deficit [2,3]. The former is characterized by the normal acquisition and retention of memories over short periods of up to 30 min but abnormally fast forgetting over periods of days or weeks after the event. The phenomenon has been termed long-term amnesia or accelerated long-term forgetting [3,4]. The latter is characterized by a patchy loss of memories of salient personal events, such as family events or holidays or weddings, from the remote past extending back over many years [3]. The performance of patients with these three types of memory deficit on standard neuropsychological memory tests that assess the retention of new memory after delays of 30–40 min is usually normal [2].

We encountered a woman with late-onset TLE who presented with transient amnesia as the sole ictal manifestation, an accelerated rate of forgetting of daily life events, and a retrograde memory deficit that specifically affected her autobiographical memory. We herein investigated her memory function both before and after the administration of antiseizure medication and discussed the findings about the relationships between her seizures during the study period and her memory function.

2. Case presentation

Ms. A was a 67-year-old right-handed woman who was referred to us due to transient amnesia and memory disturbance. She was the product of a normal pregnancy and delivery, and her development was normal. Her family history was unremarkable. She had no history of head injury, neurological illness, or drug abuse. After graduating high school, she was employed as an office worker; after getting married, she became a homemaker. She was living with her husband, daughter and two granddaughters. Six months prior to her first attendance at our clinic, her family observed several episodes of transient amnesia of approximately 15 min in duration. These episodes were characterized by a sudden onset of disorientation regarding her location or her purpose for being at the location. Although impaired consciousness was absent and responsiveness was maintained, she had no recall for events during these attacks. She was unaware of her seizures. One morning, soon after getting up in her house, she asked her daughter where the switch for the heater was. She did not know the location of the switch for the electric floor heater. She looked at her husband and could not recognize him. On another occasion, when she and her family went on a day trip by, she suddenly repeated, “Why am I here?” She could respond when spoken to, although her responses were slightly superficial. At the same time as these episodes occurred, she also began to experience a baseline memory disturbance. She described accelerated forgetting as follows. She started to worry about her inability to remember what she had done approximately one month previously. When she consulted our clinic for a third time two months later, she did not remember that she had undergone a psychological examination at our clinic one month previously. She also described a patchy loss of remote autographical memories. Patchy memory loss of family travel and ceremonial occasions had occurred over the past 3 years. Even if she saw commemorative

* Corresponding author at: Musashino Kokubunji Clinic, Kokubunji Honcho Crystal Building 3F, 4-1-9 Honcho, Kokubunji, Tokyo 185-0012, Japan.
E-mail address: msekimoto@circus.ocn.ne.jp (M. Sekimoto).

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photos of the visits, she was not able to remember having traveled with her family one or two years previously.

Her Mini-Mental State Examination and Hamilton Depression Rating Scale scores were 30 and 7, respectively. The results of the Wechsler Abbreviated Scale of intelligence (WAIS-III) [5] and the Wechsler Memory Scale—Revised (WMS-R) [6], are shown in Table 1. Her visual memory, general memory and delayed recall scores were in the lower ranges. Overall, her WMS-R score levels were lower in comparison to her WAIS-III score levels.

Interictal electroencephalography (EEG) revealed low-voltage spikes in the right temporal region on awake (Figs. 1 and 2) and during light sleep. Brain magnetic resonance imaging showed slight cerebral atrophy with no remarkable hippocampal atrophy. Interictal single-photon emission computed tomography revealed an area of slight hypoperfusion in the right frontal and parietal regions.

Treatment with carbamazepine (50 mg, twice daily with a serum concentration of 3.1–4.5 μg/ml) was started focal epilepsy after the completion of the above-mentioned tests. Her seizures disappeared one month after starting carbamazepine. An EEG recording at one year after disappearance of seizures showed no epileptiform. One year after the disappearance of seizures, she no longer worried about forgetting daily life personal events. On repeat WAIS-III [5] and WMS-R testing [6] one year after disappearance of seizures, although there was no difference in her WAIS-III scores before and after the administration of the antiseizure medication, her visual memory, general memory and delayed recall scores in the WMS-R at one year after the disappearance of seizures improved overall in comparison to those before treatment (Table 1). However, the lost memories about recent and remote personal events, such as family travel experiences, were not recovered.

3. Discussion

Our patient showed recurrent episodes of isolated TEA as the sole manifestation of seizures and did not have any other features of seizures in TLE such as brief loss of awareness, olfactory or gustatory hallucinations and autonomic symptoms. She also had the characteristics of amnesia on waking and repeated questioning.

Some reports have suggested that TEA is sometimes the sole ictal manifestation of TLE [1,2,7,8]. Ictal amnesia episodes are sometimes mistaken for episodes of transient global amnesia, transient ischemic attacks, or psychogenic amnesia [3]. The depression ratings of our patient were normal. She had no history of psychiatric disease and did not have any psychological problems.

Zeman et al. advocated the following diagnostic criteria for TEA [2]: 1) a history of recurrent witnessed episodes of transient amnesia; 2) cognitive functions other than memory were judged as being intact during typical episodes by a reliable witness; 3) sufficient evidence for a diagnosis of epilepsy (based on one or more of the following: a) epileptiform abnormalities on EEG, b) the concurrent onset of other clinical features of epilepsy, c) a clear-cut response to anticonvulsant medication).

Table 1
Neuropsychological results.

Test		Before medication	1 year after disappearance of seizures
WAIS-III	Verbal IQ	97	97
	Performance IQ	75	80
	Full scale IQ	86	89
WMS-R	Verbal memory	77	84
	Visual memory	58	95
	General memory	67	86
	Attention/concentration	98	98
	Delayed recall	69	83

Butler et al. described patients with "pure" attacks of TEA, in which amnesia was the only ictal symptom [3]. They excluded cases where witness accounts were unavailable, unreliable, or indicated more extensive cognitive impairment during all attacks.

Although the presence of ictal EEG discharges during a typical event confirms the diagnosis of ictal amnesia [8], we could not capture a typical event because of the infrequent occurrence of seizures in the present case. Our patient showed interictal low voltage spikes on her awake EEG. These spikes were different from benign epileptiform transients of sleep because of clear phase reversal in right temporal region and appearance on awake. Witness accounts of her family were available and reliable and she did not indicate more extensive cognitive impairment during all her attacks. She did not show the concurrent onset of other clinical features of epilepsy. From these facts, we concluded that her symptoms were broadly consistent with the diagnostic criteria for pure attacks of TEA.

Interictal epileptiform abnormalities on EEG in TEA were seen in about 40% and were localized over the temporal or fronto-temporal region [9]. Interictal EEG in our patient was similar to those reports. TEA is responsive to relatively low doses of antiseizure medication [3]. After commencing extremely low dose of sodium valproate monotherapy (daily dose = 100 mg), episodes ceased in patients with TEA [10]. Like these reports, treatment with low dose of antiseizure drug abolished the attacks in our patient.

When treating patients with epilepsy-associated memory problems, we need to consider the influence of antiseizure medication, because there is a possibility that the medication itself may be a confounding factor that affects the patient's memory function [11,12]. In the present study, the influence of antiseizure medication could be excluded because she was treatment-naïve.

Our patient not only had brief recurrent episodes of amnesia but also reported interictal accelerated forgetting and autobiographical amnesia. Her accelerated forgetting and the patchy impairment of episodic autographical memory were similar to the memory impairment that often occurs in TEA.

No previous reports have directly compared the pretreatment and posttreatment memory performance of patients in whom TEA was the sole manifestation of seizures. The findings regarding the relationship between seizure frequencies during the study period and memory function are controversial. Hendriks et al. noted that a high seizure frequency is particularly disruptive to the first encoding stage of the memory process [13]. Mameniskiene et al. reported that frequent seizures during the study period were related to poor long-term recall and that uncontrolled seizures can be a significant factor in the accelerated decay of memory [14]. In addition, O'Connor et al. found that forgetting increased in conjunction with more frequent seizures and that this trend was reversed by antiseizure medication [15]. In contrast, isolated seizures do not generally cause patients to forget material that they have recently learned, because there was no correlation between memory performance and seizure frequency [16,17]. We presumed that the disappearance of epileptic seizures and the temporal lobe epileptiform abnormalities in our patient was associated with subjective memory improvement and the improvement in her WMS-R scores.

In our patient, even after seizures were controlled with antiseizure medication, the memories that lost in TEA, long-term anterograde and retrograde amnesia were not recovered. It is suggested that an irreversible change in the brain occurred due to repeated clinical and subclinical activity. Structures in both the hippocampal complex and neocortex play an important role in the establishment and maintenance of long-term episodic memory representation [18,19]. It remains unclear whether epileptic activity interferes with the memory consolidation, storage and retrieval processes. The possibility that recurrent seizures are responsible for the impairment of long-term memory consolidation has been raised by several authors. Kapur suggested that repeated burst of clinical and subclinical epileptiform activity over months and years may disrupt neocortically-based neural networks that act as storage or