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Clinical differences between transient epileptic amnesia (TEA) and recurrent transient global amnesia (r-TGA)

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ABSTRACT

Both transient epileptic amnesia (TEA) and transient global amnesia (TGA) are clinically characterized by temporary amnesic symptoms. TEA involves recurring amnesic episodes, while TGA typically manifests as a singular occurrence. TGA rarely occurs repeatedly, known as recurrent TGA (r-TGA), which complicates differentiation, and raises the possibility of overlap between two disorders. Given the effectiveness of suitable antiseizure medications (ASMs) in treating TEA, accurate diagnosis of the initial event is crucial. In this study, We retrospectively analyzed cases of sudden-onset amnesia at Juntendo University Hospital over the past 14 years. Our investigation identified a total of 17 patients with TEA who met the criteria established by Zeman, along with a total of 9 patients with r-TGA meeting Hodges' criteria.

Compared to r-TGA, TEA exhibited a higher recurrent rate, shorter symptoms, more additional symptoms, and increased structural abnormalities on head magnetic resonance imaging (MRI), and more abnormalities electroencephalography (EEG).

Moreover, individual amnestic episode duration varied in both TEA and r-TGA. Our study reveals that TGA retains key features in recurrent cases and demonstrates distinctions from TEA. Nevertheless, distinguishing between the two conditions based solely on initial episodes remains challenging.

Introduction

Transient global amnesia (TGA) is a condition characterized by a brief episode of amnesia, a symptom also observed in transient epileptic amnesia (TEA). TGA typically occurs in individuals aged 50 to 70 and is marked by sudden onset anterograde amnesia usually lasting 4–6 h, resolving within 24 h at longest [1,2,3]. During amnesic episodes, anterograde amnesia occurs, in which new information is not retained for more than a few seconds. Retrograde amnesia may also occur, going back hours, days, or longer. The patient repeats the same question many times at irregular intervals of about 30 s. How did I get here?", "Where am I?", "What happened?", "What time is it?", etc [1,3]. Temporary high diffusion-weighted imaging signals in the hippocampus can be seen in head MRI scans [1,3,4]. Cognitive function prognosis is favorable, though relapses occur in 3–24 % of cases [1]. The pathomechanism, linked to arterial ischemia, impaired venous return, epilepsy, migraine, and stress, remains under exploration [1,4]. In 1990, Hodges et al. [5] proposed the diagnosis criteria TGA as follows: (a) Attacks must be witnessed and information available from a capable observer who was present for most of the attack; (b) Three must be clear-cut anterograde amnesia during the attack; (c) Clouding of consciousness and loss of personal identity must be absent, and the cognitive impairment limited to amnesia (that is, no aphasia, apraxia, etc); (d) There should be no accompanying focal neurological symptoms during the attack and no significant neurological signs afterward; (e) Epileptic features must be absent; (f) Attacks must resolve within 24 h; (g) Patients with recent head injury or active epilepsy (that is, remaining on medication or one seizure in the past two years) were excluded. TEA is characterized by transient amnesic attacks as an epileptic symptom. In 1998, Zeman et al. [6] defined the diagnostic criteria for TEA as follows: (1) a history of recurrent witnessed episodes of transient amnesia; (2) cognitive functions other than memory judged to be intact during typical episodes by a reliable witness; (3) evidence for a diagnosis of epilepsy on the basis of one or more defined characteristics; (a) Epileptiform abnormalities on electroencephalography (b) The concurrent onset of other clinical features of epilepsy (e.g., lip-smacking, olfactory hallucinations) (c) A clear-cut response to anticonvulsant therapy. Attacks occur several times a week at most and less than once a year, often upon awakening,

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and lasting 30–60 min [7]. TEA may be a subtype of temporal lobe epilepsy (TLE), featuring associated symptoms like behavioral arrest, automatic movements, gastrointestinal issues, and olfactory hallucinations [7]. While standard 21-chnannel scalp EEG abnormalities are present in only 37 % of cases, TEA responds well to ASMs [7,8]. Ordinarily, differentiation between TEA and TGA is uncomplicated owing to clear-cut clinical traits, recurrence, symptoms, and EEG profiles. However, the emergence of Recurrent TGA (r-TGA), documented in 3–24 % of cases, complicates the differentiation, leading to misdiagnosis, with some TEA cases being mistaken for r-TGA, and vice versa. Also, it may suggest the potential overlap of the two disorders. Some cases with r-TGA may exhibit epileptic features, a characteristic that is associated with TEA. Although various studies have compared TGA and TEA, none have contrasted TEA with r-TGA. This study aims to investigate the distinctiveness of TEA and r-TGA are distinct disorders by delineating both commonalities and distinctions in their clinical, radiological, and electrophysiological features.

Methods

A retrospective investigation was conducted on patients visiting Juntendo University outpatient clinics for sudden memory loss between October 31, 2010, and April 30, 2024, utilizing electronic medical records. All patients underwent neurological examination, brain MRI scan, and standard 16-channel scalp EEG. Zeman's criteria [6] were used for the diagnosis of TEA. Hodges' criteria [5] were used for the diagnosis of TGA, and cases with recurrence were selected. Exclusion criteria encompassed psychiatric disorders, dementia history, use of sleep-inducing medications, or secondary causes such as hypoglycemia or head trauma. All cases were meticulously screened for relevance to TEA or r-TGA, focusing on (1) age, (2) gender, (3) recurrence rate, (4) amnesia duration, (5) onset time of amnesia, (6) presence or absence of non-amnesia associated symptoms, (7) head MRI abnormalities (DWI or structural), and (8) EEG findings including spike, sharp, sharp and wave complexes, or focal theta waves with spike-like morphology. To address discrepancies in amnesia duration reporting, mean duration was calculated and employed for analysis. Moreover, the duration of initial attack and recurrent memory loss was investigated for each case. All statistical analyses were performed using statistical software (EZR; Saitama Medical Centre, Jichi Medical University, Saitama, Japan), a graphical user interface for R (v2.13.0; The R Foundation for Statistical Computing, Vienna, Austria). The data are presented as mean ± standard deviation. Continuous variables were analyzed with an unpaired *t*-test, and proportions were analyzed with Fisher's exact test for count data. Receiver operating characteristic (ROC) analysis was conducted to assess the predictive value for the duration of amnesia. Statistical significance was defined as $p < 0.05$.

Result

We retrospectively investigated 137 patients from our university, excluding 27 due to missing data. The remaining 110 patients underwent neurological examination by specialists, general blood tests, head magnetic resonance imaging (MRI), and electroencephalography (EEG). Based on Zeman's criteria [6], 11 patients were diagnosed with TEA, and 99 as TGA, of which 9 were identified as r-TGA using Hodges' criteria [5] (Fig. 1). Their features were summarized in the Table 1 and Table 2. Our cases showed a significant difference in the onset of amnesia ($p < 0.01$). The results of the analysis are summarized in Table 3. Seventeen patients (age 60.64 ± 11.14 years old, male/female: 9:8) were diagnosed with TEA and 9 patients (age 63.55 ± 8.21 years old, male/female: 3:6) with r-TGA. No significant age and sex differences were observed ($p = 0.498$, $p = 0.234$). Recurrence of amnesia was a total of 78times (mean = 4.5, SD = ±8.89, range = 2–39 times) in the

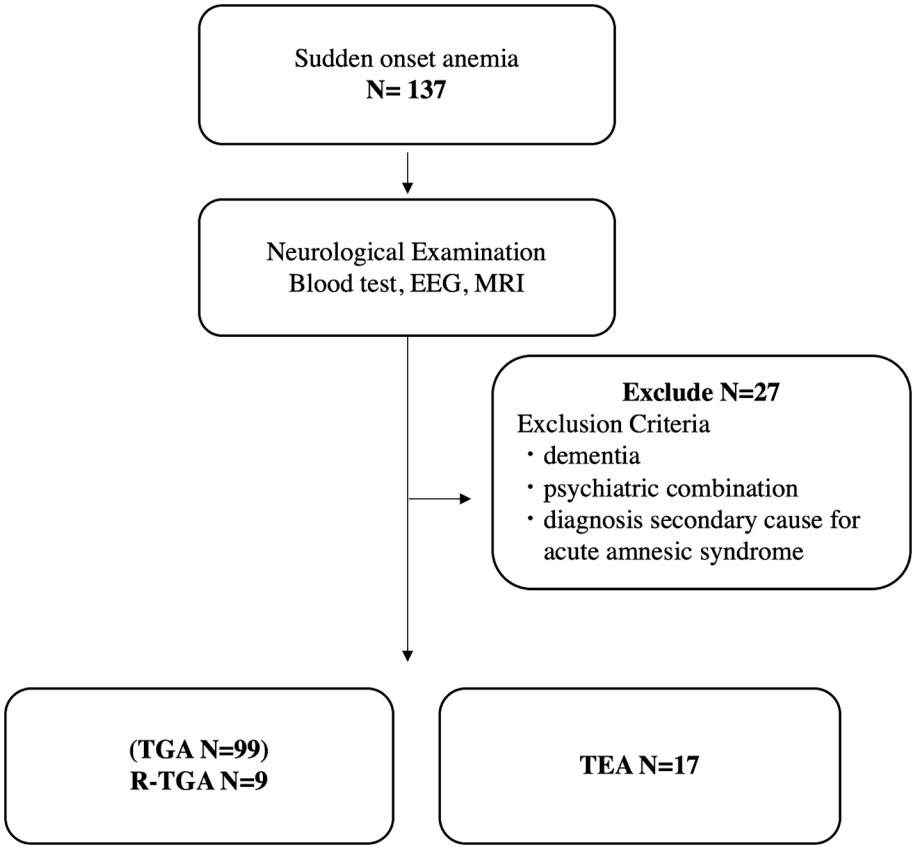

Fig.1. Flow chart of the study design. MRI; magnetic resonance imaging, EEG;16-channel standard scalp EEG recording.

TEA and a total of 18 times (mean = 2, SD = ±0, range = all 2 times) in TGA, with TEA has recurrence frequency was higher (p = 0.39). Since the follow-up period varied from case to case and it was difficult to average the number of recurrences over a given period, the count was based on the sum of the number of recurrences over the follow-up period. The mean amnesia duration was 110.62 ± 90.64 min (range = 8–360 min) in TEA and 363.55 ± 295.32 min (range = 2-1440 min) in r-TGA, with TEA having significantly shorter duration time (P < 0.05). Upon occurrence, 17.6 % (3/17) of TEA patients experienced amnesia upon waking, while none of the r-TGA patients did (p = 0.52). A cutoff time of 180 min was determined by the ROC curve (Fig. 2). Seizures lasting less than 3 h were found in all patients (17/17) of TEA and 37 % (1/8) of r-TGA cases. The amnesia occurred upon waking in 17.6 % (3/17) of TEA, while it occurred in none of the r-TGA patients. Most amnesia occurred while the patients were awake. 16 out of 17 patients with TEA experienced episodes while awake (7 in the afternoon, and 6 at unspecified times), whereas 8 out of 9 patients with r-TGA experienced episodes while awake (1 in the morning, 4 in the afternoon, and 3 at unspecified times). Additional symptoms were observed in 58.82 % (10/17) of TEA cases, while none of r-TGA cases. The details of symptoms are described in Table 1, Motor automatism, confusion and apraxia were the most common (3/17, 27.27 %). Multiple symptoms were occasionally observed within a single attack (Table 1). Brain MRI showed abnormalities in 5/17 (19.41 %) in TEA and 2/9 (22.22 %) in r-TGA (p = 1). Among, transient dot-like hyperintensities on DWI (diffusion-weighted imaging) or fluid-attenuated inversion recovery (FLAIR) sequences were observed in 11.76 % (2/17) of TEA and 22.22 % (2/9) of r-TGA cases (p = 0.591). Structural brain abnormalities were observed in 17.64 % (3/17) of TEA, while none were reported in r-TGA. (p = 0.529). All of the head MRI abnormalities were located in the hippocampus. Details are shown in Table 1 and Table 2. Abnormal Electroencephalography (EEG) findings were noted in 76.47 % (13/17) of TEA cases. EEG was deemed abnormal if focal or diffuse interictal epileptiform transients (spike, sharp wave, or spike-and-wave discharges) or focal theta waves with spiky morphology were detected. All EEG abnormalities involved the temporal region. Details are given in Table 1. In summary, the patients of r-TGA and TEA were predominantly middle-aged and older adults. TEA tends to have a higher recurrence rate, shorter amnesia duration, more onset upon waking, and additional non-amnestic symptoms compared to r-TGA. Additionally, TEA was less commonly associated with abnormal head MRI DWI signals, but more frequently linked with structural brain abnormalities compared to r-TGA. Approximately 80 % of TEA cases displayed EEG abnormalities. Due to TGA exclusion criteria, no comparison of the prevalence of Plus symptoms and EEG abnormalities between TEA and r-TGA was performed.

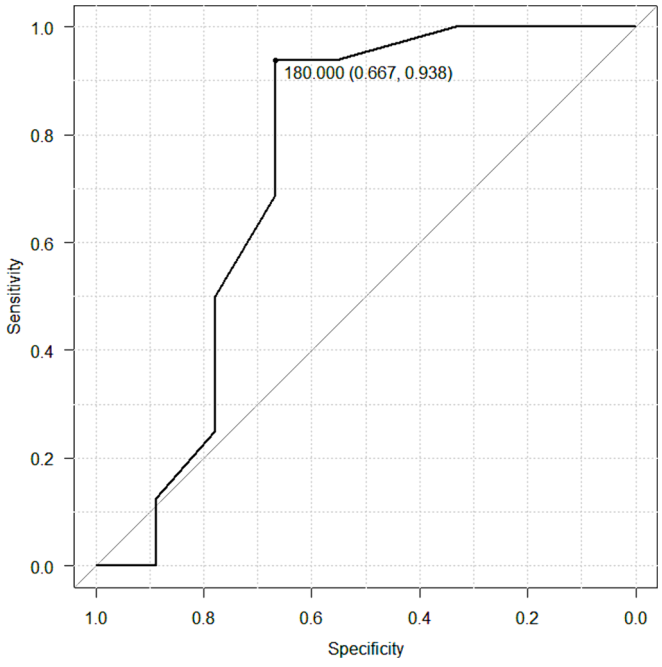

Fig.2. ROC curve depicting amnesia duration (in minutes) for both TEA and r-TGA. The ROC curve evaluates amnesia duration for the TEA and r-TGA, indicationgindicating a shared cutoff 180 min (sensitivity 0.938, specificity 0.667).

Discussion

The typical clinical features of TGA manifest in middle-aged and older adults, typically aged 50–70 years, presenting with sudden onset anterograde amnesia lasting 4–6 h, often without accompanying neurological abnormalities other than amnesia [1,2,3]. Head MRI may reveal transient high density in the hippocampus on DWI or FLAIR sequences [1,3,4], while EEG results are typically normal. Recurrence has been reported in 3–24 % of cases [1]. Our study found that recurrent TGA exhibited similar clinical features, with patients typically in their mid-60 s, experiencing sudden onset amnesia lasting approximately 6 h, often without additional clinical symptoms. Head MRI typically showed hyperintensities in DWI or FLAIR localized to the hippocampus, while EEG results were generally normal. These findings align with those of the previous study [9]. Some differences between TEA and r-TGA were found, especially significant differences in the duration of amnesia in our institutional cases. TEA exhibited a shorter duration of amnesia (110 min) compared to r-TGA (363 min), consistent with the previous reports where TEA episodes typically lasted 30–60 min, with a wide variation ranging from less than 1 min to a few day [6,8]. TGA episodes, on the other hand, typically lasted 4–6 h, resolving within 24 h [1,2,3]. The ROC analysis indicated a cutoff time of 3 h (Fig. 2), yet distinguishing between the two disorders based on the initial episode remained challenging due to the varying durations (Fig. 3). TEA is a known subtype of temporal lobe epilepsy, but it is known that the seizure duration of TEA is 30–60 min, whereas the seizure duration of typical TLE is a few minutes at most. Instances included amnesia persisting for 4800 min (3 days) [10] and 2890 min (2 days) [8], often accompanied by confusion, suggesting a possible postictal state. Prolonged seizures in TEA, lacking witnesses and featuring convulsions, may indicate a postictal decrease in consciousness linked to temporal lobe epilepsy (TLE) [7]. To establish this supposition, it may be necessary to perform a video-EEG and monitor the persistence of clinical symptoms and EEG abnormalities. In a prior study, as many as 94 % of TEA patients reported amnesia upon morning awakening, with 7 % exclusively experiencing symptoms after waking [7]. In contrast, TGA episodes are not restricted by time of day. Similarly, we noted a greater incidence of TEAs occurring upon waking compared to r-TGA. TEA is often accompanied by additional symptoms, consistent with a large cohort study [7]. This suggests that TEA is situated within the spectrum of temporal lobe epilepsy (TLE), emphasizing the diagnostic value of comorbid symptoms. In contrast to TEA, TGA often presents with one or more small focal restrictive lesions in the lateral hippocampus, visible on head MRI [11,12]. Additionally, transient dot-like hyperintensities in the hippocampus on DWI sequences, more prevalent in r-TGA than TEA, were observed in our study, aligning with prior research. High-resolution 3-tesla head MRI proves useful for diagnosis [13]. While TEA usually shows a normal MRI, occasional cases exhibit decreased hippocampal volume [14]. Notably, structural abnormalities, including hippocampal atrophy, were more common in TEA, suggesting a potential link to TLE. Standard EEG is essential for TEA diagnosis, but in 30–43 % of cases, it yields normal results [12,15]. Studies indicate that around 37 % show abnormalities, primarily in temporal electrodes, with over 50 % displaying bilateral involvement [7,15]. In our study, 76.47 % of TEA cases had EEG abnormalities, likely influenced by repeated assessments, with the majority observed in the temporal region. Earlier

Table 1
Clinical findings of 17 patients with TEA.

No	Age	Sex	Duration ofamnesia (min) 1st/2nd attack	Recurrence(times)	Onset	Plussymptoms	EEG	MRI	Therapy/Outcome
1	65	M	60/5	2	afternoon	no	Bil FT	normal	lost at F/U
2	70	F	no detail/180	2	afternoon	no	Bil T	normal	no/SF
3	73	F	120/120	2	afternoon	no	Bil FT	normal	no/SF
4	64	F	15/<15	3	awaking	no	L T	normal	GBP/SF
5	54	M	30/no detail	4	awaking	no	diffuse	normal	lost at F/U
6	56	F	60/no detail	2	sleeping	oral-facialautomatism	Rdiffuse	normal	LCM/SF
7	66	M	120/no detail	2	afternoon	motorautomatism	normal	Bil HIPPatrophy	lost at F/U
8	60	F	180/1	2	awaking	headache	Bil T	normal	no/SF
9	49	M	180/180	2	afternoon	motorautomatism	normal	R HIPPDWI high	lost at F/U
10	58	F	60/<60	2	awaking	language disturbance	L T	normal	LEV/SF
11	66	M	15/<60	39	Wake up	aphasia	R FT	normal	LCM/SF
12	27	M	8/60	2	awaking	apraxia	normal	R HIPPatrophy	lost at F/U
13	70	F	<30/<30	2	afternoon	no	Bil T	L HIPPDWI high	no/SF
14	53	F	few hour/few hour	3	wake up	confusion	normal	normal	no/recurrence
15	62	M	no detail/120	2	wake up	no	RT	Bil HIPPswelling	lost at F/U
16	73	M	<360/<360	3	afternoon	confusion apraxia	normal	normal	no/SF
17	65	M	60/180	4	awaking	confusion	normal	normal	no/recurrence

R; right, L; left, Bil; bilateral, T; temporal, FT; frontotemporal, HIPP; hippocampal, GBP; gabapentine, LCM; lacosamide, LEV; levetirasetum, SF; seizure free.

Table 2
Clinical findings of 9 patients with r-TGA.

No	Age	Sex	Duration ofamnesia (min) 1st/2nd attack	Recurrence(times)	Onset	Plussymptoms	EEG	MRI
1	55	F	720/15	2	afternoon	no	normal	normal
2	65	F	120/120	2	morning	no	normal	normal
3	70	F	30/no detail	2	awaking	no	normal	R HIPPDWI high
4	58	F	360/360	2	no detail	no	normal	Normal
5	79	F	300/1440	2	awaking	no	normal	Normal
6	60	M	no detail/2	2	afternoon	no	normal	normal
7	53	M	no detail/240	2	afternoon	no	normal	normal
8	63	F	360/360	2	awaking	no	normal	L HIPP DWI high
9	69	M	Few four/720	2	afternoon	no	normal	normal

R; right, L; left, HIPP; hippocampal.

Table 3
Clinical characteristics of TEA and r-TGA in our study.

	TEA (n = 17)	r-TGA (n = 9)	p value
Age (yr, mean ± SD)	60.64 ± 11.14	63.55 ± 8.21	0.498
Sex (male: female)	9:8	3:6	0.234
Clinical findings			
Recurrence times	78	18	0.396
Duration (min, mean ± SD)	110 ± 90.00	363 ± 295.32	<0.05
Onset on awaking (n, %)	3(17.6)	0	0.529
Plus symptoms (n, %)	10(58.82)	0	–
Neuroradiological findings			
MRI abnormalities	5(19.41)	2(22.22)	1
DWI high (n, %)	2(11.76)	2(22.22)	0.591
Structural abnormalities (n, %)	3(17.64)	0	0.529
EEG findings			
EEG abnormalities (n, %)	13(76.47)	0	–

TEA;transient epileptic amnesia; r-TGA; recurrent global amnesia; MRI; magnetic resonance imaging; DWI; diffusion-weighted image; EEG, electroencephalography.

research has indicated that abnormal EEG patterns in TEA are also evident during sleep [16,17]. In a cohort of 30 TEA cases, 57 % exhibited interictal epileptiform discharges (IEDs) during wakefulness, increasing to 96 % during sleep [15]. Notably, 44 % of patients with recorded IEDs exclusively displayed them during sleep within the TEA cohort [12,17]. Activation of IEDs during sleep is a characteristic feature of TEA, highlighting the role of sleep in memory consolidation. TEA tends to show more abnormalities in sleep EEG than in wakefulness EEG, suggesting a potential correlation between the higher frequency of TEA onset upon awakening and sleep-related factors.

TEA typically responds well to ASMs. In our study, 4 of 17 TEA patients received ASMs and all had complete resolution of their seizures. Seven of 17 patients did not receive ASMs. Five of the 7 patients have remained in seizure free to date, but two have had recurrent seizures. Six of the 17 patients were difficult to follow up because they had lost their visits due to transfers to other doctors or interrupted visits. In contrast to TEA, the underlying mechanisms behind TGA remain elusive, despite various hypotheses [1,4]. A cortical spreading depression (CSD)-like pathophysiology has been suggested due to the frequent co-occurrence of TGA with migraine headaches [18]. However, there is a notable age of onset disparity between migraine and TGA. In our r-TGA cases, only 6 % were associated with concomitant migraine. Recent studies have also proposed the potential link between hippocampal congestion and insufficient perfusion of the internal jugular vein (IJV). Many TGA episodes coincide with Valsalva maneuvers, which can elevate venous perfusion to the superior vena cava (SVC) or raise intraosseous pressure, subsequently leading to heightened pressure in the IJV [19].

A previous investigation examining the long-term risk of cerebrovascular events, seizures, and cognitive impairment in TGA patients found no significant disparities in occurrence rates for each event

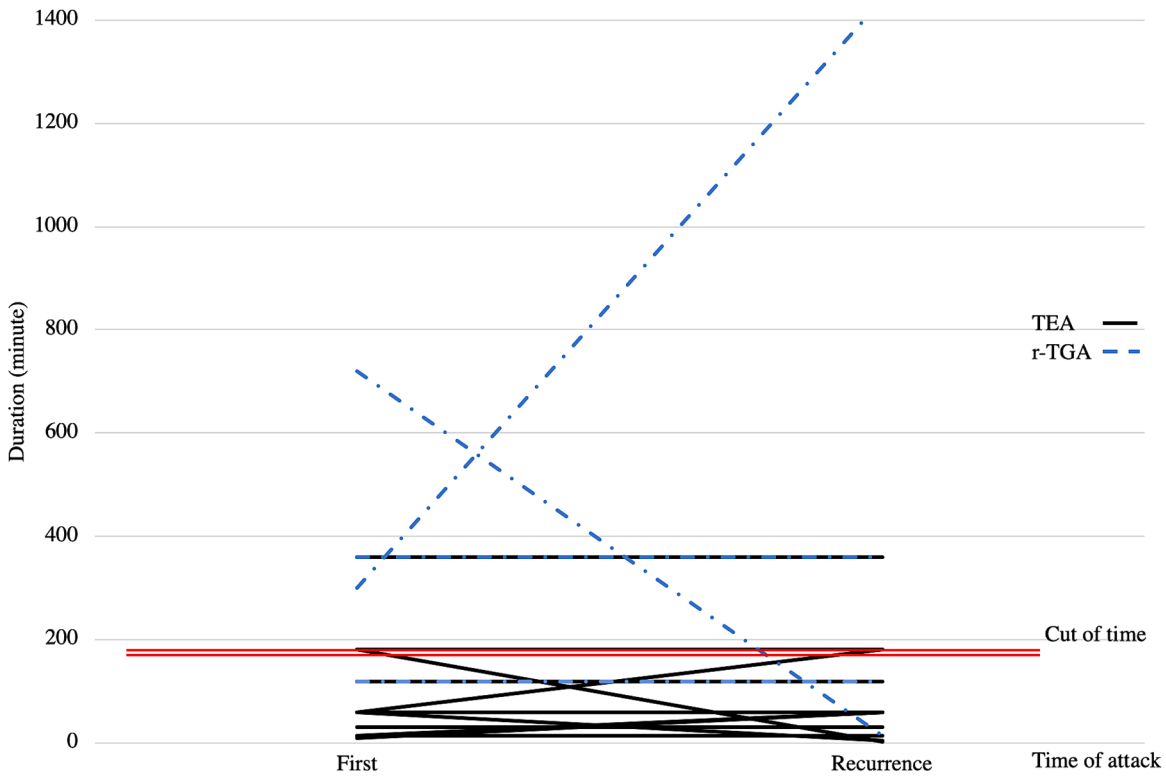

Fig.3. Duration of amnesia attack at first and recurrence. The graph illustrates the time of initial amnesic episode and subsequent attack durations for TEA and r-TGA, denoted by straight line and dot line, respectively.

between TGA cases and control subjects [20]. Consequently, treatment or prevention measures for TGA are not recommended.

Regarding the differentiation between TEA and r-TGA, our findings align with prior research, emphasizing the duration of amnesia as a distinguishing factor [16]. Notably, our study underscores the diverse range of amnesia durations in TEA, making diagnosis based solely on the initial episode challenging. Furthermore, r-TGA has no association with epilepsy. These results indicate that TGA, even in recurrent cases, maintains distinct features that set it apart from TEA. In conclusions, while many studies have compared single TGAs and TEA, no study have compared r-TGA and TEA. We found that r-TGAs retain the key features of single-episode TGA, placing it under a different spectrum from epilepsy. Additionally, although the duration of amnesia tends to be shorter in TEA, there are cases where the duration of amnesia is not consistent between the initial and recurrent episodes, indicating that it is impossible to differentiate two disorders solely based on the amnesia episode of the initial attack.

Ethical statement

The research has complied with all the relevant national regulations, followed the principles outlined in the Declaration of the Helsinki Declaration, and was approved by the ethics committee of Juntendo University. We confirm that we have read the journal's guidelines on ethical publication issues and affirm that this work is consistent with those guidelines.

CRediT authorship contribution statement

Mizuho Sugiyama: Writing – original draft. **Taiji Tsunemi:** Writing – review & editing. **Nobutaka Hattori:** Writing – review & editing.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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