Transient epileptic amnesia
By C P Panayiotopoulos MD PhD
(Dr. Panayiotopoulos of St. Thomas’ Hospital has no relevant financial relationships to disclose.)
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Introduction

This article includes discussion of transient epileptic amnesia, epileptic amnesic attacks, pure amnestic seizures, epileptic global amnesia, epileptic amnesia, and epileptic transient amnesia. The foregoing terms may include synonyms, similar disorders, variations in usage, and abbreviations.

Overview

Transient epileptic amnesia has been considered a syndrome of mesial temporal lobe epilepsy characterized by (1) recurrent episodes of isolated memory impairment of epileptic cause (ictal or postictal) while other cognitive functions remain intact; (2) interictal memory disturbances of accelerated long-term forgetting and autobiographical and topographical amnesia; and (3) late age of onset with a mean of 57 years. The duration of episodes of amnesia is usually less than an hour with usual recurrence of around 20 times each year in untreated patients. In addition, brief seizures typical of mesial temporal lobe epilepsy are detected in two thirds of patients. Interictal EEG, particularly when recorded in sleep, shows temporal lobe spikes whereas EEG during attacks of amnesia demonstrates either ictal discharges or postictal features. In most cases of transient epileptic amnesia, no clear cause for the epilepsy is identified though MRI may show hippocampal atrophy or focal structural lesions in the temporal lobes. Transient epileptic amnesia is considered rare though it is frequently underdiagnosed or misdiagnosed as transient global or psychogenic amnesia. Patients with transient epileptic amnesia usually have an excellent prognosis; seizures respond extremely well to monotherapy with small doses of lamotrigine or levetiracetam though interictal memory disturbances may persist.

Key points

• Transient epileptic amnesia has been considered a syndrome of mesial temporal lobe epilepsy with recurrent episodes of amnesia (ictal or postictal).
• The duration of episodes of amnesia is usually less than an hour (median duration 30 to 60 minutes).
• The attacks are frequent, usually around 20 times each year in untreated patients.
• Two third of patients also present with brief seizures of mesial temporal lobe epilepsy.
• Documentation of the epileptic nature of transient epileptic amnesia requires meticulous clinical assessment and EEG.
• Transient epileptic amnesia usually affects middle-aged or elderly subjects, men (60%) more than women, and the attacks often occurring on waking.
• Intercital memory manifestations consist of accelerated long-term forgetting as well as autobiographical and topographical amnesia.
• Monotherapy with lamotrigine or levetiracetam is effective in controlling the seizures and the amnestic attacks in the majority of patients, but interictal memory disturbances may persist.

Historical note and terminology

The earliest description of epilepsy-related transient amnesia is attributed to the case of Dr. Z detailed by Hughlings Jackson in 2 reports (Jackson 1888; Jackson and Colman 1898; Butler and Zeman 2008b). Dr. Z had from the age of 21 years attacks typical of temporal lobe epilepsy with secondarily GTCS and severe postictal amnesia caused by a single, circumscribed lesion in the left uncus discovered at autopsy after his death from chloral overdose:
In the remainder of this article I shall speak only of Z’s slight seizures. I witnessed but two of them... In one he stopped talking to me, remained standing, and made slight, very slight, just audible (*vide supra*) smacking movements of his lips. ... On another occasion he was sitting in a room consulting me; he stopped talking--I have no remembrance of any smacking movements of his mouth on this occasion--his head was bent forward, but in a second or two, the paroxysm being then, I suppose, over, he looked up, and next (postparoxysmal stage of actions) he leaned over one arm of his chair and felt about on the floor as if searching for something. Next he did the like on the other side......

Soon, perhaps a minute, afterwards, his actions, or I should say the irrelevant-seeming actions, ceased; he replied correctly to simple questions and told me that it was not necessary for me to go home with him. He, however, looked confused and seemed strange. When we got to his house a few yards away, I thought he was fully recovered, and, as I was thinking of making another room on the ground floor of my house, I took the opportunity of speaking to him about a third room on the ground floor of his house. Among other things he said he used to breakfast there. I was surprised when he afterwards, next day, told me that he remembered nothing from the time of being in my room consulting me (before the fit) to a little time after I left him at his own house (*Jackson and Colman 1898*).

This case with postictal amnesia is unlikely to be considered part of the syndrome of transient epileptic amnesia because of the early age at onset. In subsequent reports, it has been well documented that transient amnesia:

(A) occurs as an ictal or postictal symptom in association with temporal lobe epilepsy and (B) is sometimes the only manifestation or the only seizure type of temporal lobe seizures in some patients (*Zeman et al 1998; Zeman and Butler 2010*).

It has also been reported that seizures causing prominent amnesia are easily mistaken for episodes of transient global amnesia or of psychogenic amnesia, and various terms have been used to describe these attacks, including pure amnestic seizures, ictal amnesia, epileptic amnesia, epileptic amnesic attacks, epileptic transient amnesia, and transient epileptic amnesia (*Zeman et al 1998; Zeman and Butler 2010; Butler and Zeman 2011*).

The term “transient epileptic amnesia” was introduced by Narinder Kapur, who highlighted that amnesic attacks caused by epilepsy are similar to the syndrome of transient global amnesia, but they may have certain distinguishing features, including brevity and recurrence, which appeared to stand out as supportive of a diagnosis of transient epileptic amnesia and as rather atypical for transient global amnesia (*Kapur 1993*).

A significant advance in our current understanding of transient epileptic amnesia has been made thanks to the UK-wide The Impairment of Memory in Epilepsy (TIME) Project (*Kapur 1993; Butler et al 2007; Butler et al 2009; Butler et al 2013; Milton et al 2010; Muhlert et al 2010; Zeman et al 2012*).

**Clinical manifestations**

**Presentation and course**

Transient epileptic amnesia has been considered a syndrome of mesial temporal lobe epilepsy characterized by recurrent episodes of isolated memory impairment of epileptic cause (ictal or postictal) while other cognitive functions remain intact (*Kapur 1993; Zeman et al 1998; Zeman et al 2012; Butler et al 2007; Butler and Zeman 2008b; Butler and Zeman 2011; Bilo et al 2009; Zeman and Butler 2010; Ioannidis et al 2011; Asadi-Pooya 2014; Lapenta et al 2014; Mosbah et al 2014; Felician et al 2015*).

Transient epileptic amnesia usually affects middle-aged or elderly subjects (older than 40 years; mean age of 57 years), men (60%) more than women, and the attacks often occur on waking. The amnesia itself has anterograde (inability to lay down memories during the episode) and retrograde (difficulty in retrieving memory from the past days or years) components. Anterograde amnesia may be partial in 44% of the patients.

Transient amnesia is the sole manifestation of the attacks in 34% of patients. Additionally, complex focal seizures occur in the other two thirds of patients whereas secondarily generalized tonic-clonic seizures are rare. In the report of
Butler and colleagues, 46 of 50 patients reported at least one attack in which amnesia was the sole feature (Butler et al 2007). Other features that may occur during the attacks are in order of frequency: olfactory hallucinations (42%), orofacial automatisms, or a brief period of unresponsiveness. However, it should be noted that patients with transient epileptic amnesia and prolonged attacks of amnesia may have additional seizures that are so mild and brief they are clinically undetected (Nicastro et al 2014). Furthermore, it should be remembered that hippocampal seizures are often followed by prolonged postictal disturbances and that olfactory hallucinations are relatively less common than other seizure hallucinations; they are attributed to amygdala with tumors and hippocampal sclerosis as their main causes (Panayiotopoulos 2010). In 1 study of 55 patients with transient epileptic amnesia, it was found that impairments in odor identification are common in patients and exceed changes that occur in normal aging (Savage et al 2017). Olfactory hallucinations occur in approximately half of patients but do not always coincide with reduced sense of smell. Olfactory impairment and interictal memory problems both occur frequently but are not closely associated.

It should be remembered that in hippocampal seizures olfactory hallucinations are relatively less common, and they are attributed to amygdala with tumors and hippocampal sclerosis as their main causes (Panayiotopoulos 2010).

The duration of episodes of amnesia is usually less than an hour, most often lasting about 20 to 30 minutes (median duration is 30 to 60 minutes), though attacks lasting hours may occur (Butler and Zeman 2008b; Zeman et al 2012).

The attacks are frequent, usually around 20 times each year in untreated patients, with a wide range between individuals (Butler and Zeman 2008b).

**Interictal memory manifestation.** Patients with transient epileptic amnesia frequently report 3 varieties of persistent, interictal memory disturbance that are invisible to standard neuropsychological tests:

1. The **accelerated long-term forgetting**, over days or weeks, of newly acquired information happens in half of the patients (Zeman et al 1998; Muhlert et al 2010; Hoeijeijzers et al 2015); this persistent interictal memory deficit of an “evaporation” of memories for recent events is typically more troublesome for patients with transient epileptic amnesia than are the occasional, brief amnesic episodes caused by seizures.

2. Autobiographical amnesia, a patchy but dense loss of memories for salient autobiographical events in the more remote past, happens in two-thirds of patients (Manes et al 2001; Manes et al 2005).

3. Topographical amnesia occurs in one third of patients (Zeman and Butler 2010).

One study has also documented that patients with transient epileptic amnesia suffer from early picture recognition deficit, which could reflect either the early stages of the process that leads to accelerated long-term forgetting or a separable deficit of anterograde memory in transient epileptic amnesia (Dewar et al 2015). This study also found that that at least some patients with transient epileptic amnesia are prone to falsely recognizing new everyday visual information that they have not, in fact, seen previously (Dewar et al 2015).

**Working diagnostic consensus criteria for transient epileptic amnesia.** Criteria include: (1) a history of recurrent witnessed episodes of transient amnesia; (2) confirmation by a reliable witness that cognitive functions other than memory are intact during typical episodes; and (3) evidence for a diagnosis of epilepsy (Zeman et al 1998). This evidence is provided by either (a) wake or sleep EEG, (b) the co-occurrence of other seizure types, (c) a clear-cut response to antiseizure therapy, or (d) a combination of these factors.

**Prognosis and complications**

Transient epileptic amnesia is typically an epilepsy syndrome in which seizures are easily controlled with even small doses of antiepileptic drug monotherapy, though interictal memory disturbances might persist. The few cases that have been followed for a number of years indicate that once seizures are controlled, cognitive function does not progressively deteriorate (Butler and Zeman 2008a; Zeman et al 2012), or patients may become entirely normal (Razavi et al 2010).

Savage and colleagues described a cohort of 10 patients with transient epileptic amnesia, which was first reported in 1998 and followed up at two-time intervals, each 10 years apart (Savage et al 2016). Information regarding clinical outcomes and subjective reports of memory functioning was gained via their records and clinical interview. Where
Clinical vignette

The patient, a 54-year-old male, was first seen in the clinic in September 1991. He arrived saying "I'm having fugues, Doc," having been given a diagnosis of psychogenic amnesia elsewhere (Kopelman et al 1994). After a "collapse" in February 1990, for which a good description was not available, his wife witnessed a second attack in May 1990. Shortly before they were going to bed, he bent down to pick up a cup of coffee and started to clench and unclench his hands transiently. He did not seem to understand what his wife said, and he moved into the kitchen where he looked around the room and "through" his wife for one or 2 minutes, seeming perplexed and confused, and then asked repetitive questions: "What day is it? ... What have I been doing? ... Why am I not at work? ... Where do I work?" His wife asked if he knew who or where he was, and he did. He "came round" 1 hour later, after which he went to bed and was fine in the morning. The next month, there was another attack when he was at the helm of a boat, sailing round the Isle of Man: suddenly, he demanded his fellow yachtsman tell him where they were, and he seemed very agitated, asking repetitive questions, until he came round an hour later. On this occasion, and during 2 further attacks at home lasting half an hour, there was no hand clenching nor any other evidence suggestive of epilepsy. As well as these symptoms, the patient complained of "gaps" in his memory, such as for a period when his wife was in hospital for a breast abscess in 1987; he also mentioned forgetfulness for appointments, everyday tasks, what he had read in the newspaper, and even for the location of familiar shops. On the other hand, it was noticeable that he remembered previous medical consultations with pernickety detail, and he never failed to attend medical appointments. There was a family history of possible epilepsy in a niece and of dementia in the patient's mother. A Cambridge graduate, he had worked as a partner in a small business for 20 years, but had lost his job in 1989 because of personality differences (not because of any memory problems). He had been seen previously by a neurologist and a psychiatrist in his local hospital where, after 2 routine EEGs, 2 CT scans, and cognitive testing, a tentative diagnosis of psychogenic amnesia had been made, and he had been treated with an antidepressant and a benzodiazepine.

Clinical assessment, investigations, and progress. An initial diagnosis of transient global amnesia was made based on the clinical history. Various investigations including EEG studies were organized, and a brief admission was arranged, which, for administrative reasons, did not take place for 4 months, during which the patient had a further 4 attacks that were identical to the previous ones. In the last of these attacks (although not in the others) clenching of the right hand was noted by his wife. Various psychological tests were carried out at the initial assessment. His verbal and full scale IQ were consistent with his educational accomplishments, although there did seem to be a relative decrement in performance IQ. Scores on the frontal lobe tests did not indicate any abnormality. The scores on the anterograde memory tests, however, were not as high as expected. After using published norms to convert these scores into "memory quotient" equivalents (with a population mean of 100 and SD of 15), an overall anterograde memory quotient of 98 to 0 was obtained, consistent with the population mean but 33 points below the patient's full scale IQ. A standard blood screen was normal, as were a chest radiograph, ECG, and MRI and a fluorodeoxyglucose PET (performed 6 months after cessation of the attacks). A standard EEG was normal, but 2 sleep EEGs showed frequent midtemporal sharp and slow wave complexes, arising independently in the right and left hemispheres. After a trial of carbamazepine, which produced adverse effects, he was started on phenytoin in February 1992, rising to a dose of 300 mg daily. No further attacks were recorded in the next 15 months, and there has been only 1 equivocal attack in the 21 months since this drug was commenced. Further neuropsychological testing was conducted after 6 months of treatment. Performance on the IQ, frontal, and anterograde memory tests was virtually identical to the (pretreatment) scores obtained a year earlier, although the verbal-performance IQ discrepancy had narrowed to 14 points. The anterograde memory quotient was unchanged; and the General memory quotient from the WMS-R scale gave a decrement of 23 points, relative to full scale IQ. On a retrograde memory test, requiring recall and recognition of pictures of famous news events, the patient performed at a superior level; his memory for facts about his past on a semistructured interview was also intact. He was hesitant, however, in recalling incidents from his childhood or early
adult life, and his scores were at a "borderline" level according to published norms (Kopelman et al 1994).

**Biological basis**

The pathophysiological basis of transient epileptic amnesia remains largely unknown though emotional and/or dysimmune factors may have a potential influence (Felician et al 2015). Clinical, EEG, and brain imaging findings indicate that transient epileptic amnesia is a focal mesial temporal lobe epilepsy syndrome of middle-aged and elderly patients with additional pathology in connected brain regions. The unusual interictal memory deficits of transient epileptic amnesia remain unexplained by structural pathology and may reflect physiological disruption of memory networks by subclinical epileptiform activity (Butler et al 2013). Neurometabolic data support a dysfunction of a hippocampal-neocortical network sustaining episodic memory (Mosbah et al 2014).

The attacks of transient epileptic amnesia are either ictal or postictal manifestations. It has been postulated that transient epileptic amnesia results from recurrent focal seizures that produce a physiological Todd paralysis of the mesial temporal lobe structures with resultant impaired memory function caused by an enduring postictal state (Soper et al 2011; Walsh et al 2011).

In most cases of transient epileptic amnesia, no clear cause for the epilepsy is identified though focal brain structural lesions, always in the temporal region, have been reported in a small minority of transient epileptic amnesia cases (Butler and Zeman 2011; Ioannidis et al 2011; Zeman et al 2012; Lapenta et al 2014). There is no evidence of an increased prevalence of recognized risk factors for epilepsy, such as birth injury, febrile seizures, head injury, intracranial infection, or family history of epilepsy. Frequent occurrence of depression and association with autoimmune disorders raises interesting questions regarding putative pathophysiologic mechanisms (Mosbah et al 2014).

Autobiographical amnesia maybe caused by repeated seizures in the temporal lobe resulting in the progressive "erasure" of memories. Alternatively, autobiographical memory loss may result from subtle changes in the temporal lobe, giving rise to temporal lobe epilepsy and memory problems. As with accelerated long-term forgetting, it is unlikely that antiepileptic drugs or problems with mood cause autobiographical memory loss in people with transient epileptic amnesia.

**Epidemiology**

Incidence and prevalence is unknown though it appears that transient epileptic amnesia is rare. There are around 150 cases reported in the literature. In 2003 to 2005, only 32 patients were recruited via the British Neurological Surveillance Unit (Butler et al 2007). However, transient epileptic amnesia is often misdiagnosed or underrecognized. Three cases (4%) of transient epileptic amnesia were found in a cohort of 76 consecutive patients with mild cognitive impairment (Del et al 2014).

**Differential diagnosis**

Transient epileptic amnesia should not be difficult to diagnose for patients who also have coexistent attacks typical of temporal lobe seizures. In the absence of such brief seizures or if their presence is not detectable, the diagnosis of transient epileptic amnesia may be confused with transient global amnesia and psychogenic attacks of amnesia (Kapur 1993; Butler and Zeman 2008a; Zeman et al 2012; Sugiyama et al 2015).

The main differential diagnostic criteria of transient global amnesia are:

- Attacks are usually rare (2 to 5 per life time) and of long duration (4 to 10 hours).
- During the attacks, there is profound anterograde amnesia and repetitive questioning as well as variable retrograde amnesia with intact nondeclarative memory.
- Ictal olfactory hallucinations are common at around have of the patients.
- Postictal and interictal memory is grossly intact, but subtle deficits might persist for several months.
- Seizures are not evident with normal EEG and brain imaging.

The main differential diagnostic criteria of psychogenic seizures are:
• Attacks are extremely variable in duration (from hours to months) and symptomatology.
• During the attacks, there may be other functional neurologic symptoms, such as hysterical hemiparesis or blindness.
• Postictal and interictal memory is variable, and the acquisition for new memories is preserved.
• Seizures are not evident with normal EEG and brain imaging.

Diagnostic workup

**EEG** is the most useful diagnostic procedure, particularly if the recording is obtained during the amnestic attack.

There are very few reported EEGs taken during these episodes (Bilo et al 2009; Zeman et al 2012), with some documenting that the transient epileptic amnesia is an epileptic seizure or nonconvulsive status epilepticus itself (Lee et al 1992; Meo et al 1995; Vuilleumier et al 1996) while other recordings are consistent with postictal events (Tassinari et al 1991; Gallassi et al 1992; Palmini et al 1992; Maheu et al 2004; Butler et al 2007).

**Illustrative cases of postictal amnesic attacks.** One patient had an amnesic episode while undergoing EEG. A brief (less than 1 minute) burst of left temporal spikes, during which the patient was unresponsive to speech, was followed by normalization of the EEG and a 10-minute period of amnesia characterized by repetitive questioning about recent events (Butler et al 2007). The video-EEG recording of an attack of another patient showed that the amnesic state was characterized by a normal EEG tracing followed by electroclinical complex focal seizures (Tassinari et al 1991). In another patient with medicinally-resistant transient epileptic amnesia, prolonged postictal slowing in the mesial temporal structures was evident on invasive EEG 5 hours after the occurrence of a brief focal seizure, supporting the theory of a Todd phenomenon as the underlying pathophysiological mechanism in transient epileptic amnesia (Soper et al 2011; Walsh et al 2011).

**Illustrative cases of ictal amnesic attacks.** In one woman, a prolonged amnestic state was caused by complex focal status epilepticus with bilateral mesiotemporal lobe involvement, confirmed by EEG with nasopharyngeal electrodes. An **MRI** scan obtained shortly after recovery from the amnesia showed reversible focal abnormalities consisting of increased signal intensity on **T2-weighted** scan in the mesiotemporal lobe (Lee et al 1992).

In another woman, EEG during a prolonged amnesic episode showed diffuse bursts of delta activity prominent in the frontal areas, subcontinuous polymorphic delta activity more evident in the temporal areas and more often on the left side, and paroxysms of spikes and spike and wave complexes in the left temporal or temporofrontal areas and, asynchronously and more rarely, in the right temporal areas. In the occipital areas, a quite stable alpha rhythm was generally preserved, superimposed on the slow activity. During the recording, an ictal discharge was observed as well. This was apparently subclinical because no further changes in behavior and consciousness were observed. The discharge began with diffuse, fast, low-amplitude activity followed by small spikes originating from the right temporo-central areas and successively by a recruiting rhythm in the right hemisphere spreading to the left side. The ictal discharge lasted about 50 seconds and was followed by a return to the preictal picture, without postictal flattening and with preserved occipital alpha (Meo et al 1995). The patient also had clinical evidence of overt complex focal seizures with automatisms. In another patient with transient epileptic amnesia, the ictal EEG during an amnesic episode documented nonconvulsive status epilepticus (Bilo et al 2009). The discharge beginning with diffuse, fast, low-amplitude activity was followed by small spikes in the occipital areas, progressively progressing to a recruiting rhythm on the right hemisphere spreading to the left side (Bilo et al 2009).

**Illustrative case of ictal EEG of clinically inconspicuous seizure.** A 79-year-old patient had an episode of anterograde amnesia that lasted 90 minutes. An EEG, performed after the episode, showed bilateral temporal electrographic seizures. A standard EEG performed 3 hours after the episode showed a right temporal electrographic seizure with a recruiting pattern of about 25-second duration, immediately followed by a left temporal electrographic seizure of shorter duration (about 15 seconds); the patient was not tested by the technician, but there were no obvious clinical symptoms (Nicastro et al 2014).

When ictal EEG recording is not possible, interictal EEG during sleep often reveals unilateral (32% left sided; 12% right sided) or bilateral (56%) spikes or sharp waves in the temporal regions (Butler and Zeman 2008b; Zeman et al 2012). Routine awake EEG is usually normal or shows...
minor nonspecific abnormalities. Brain MRI is usually normal (Mosbah et al 2014) or shows evidence of bilateral atrophic changes mainly involving the hippocampi (Butler et al 2009; Butler et al 2013). In the latter study, 40 patients with transient epileptic amnesia and 20 healthy controls were investigated using manual volumetry and automated multi-atlas-based segmentation of whole-brain MRI data. Both methods confirmed the presence of subtle, bilateral hippocampal atrophy. Additional atrophy was revealed in perirhinal and orbitofrontal cortices. The volumes of these regions correlated with memory performance. No structural correlates were found for accelerated long-term forgetting or autobiographical amnesia (Butler et al 2013). However, is a significant number of reports describe patients diagnosed to have transient epileptic amnesia and structural brain abnormalities (Della Marca et al 2010; Butler and Zeman 2011; Ioannidis et al 2011; Zeman et al 2012; Lapenta et al 2014).

In one patient, the fluid-attenuated inversion-recovery MRI scan revealed high signal in the left hippocampus. Dramatic and circumscribed hypermetabolism in the left medial temporal lobe was evident on the PET scan. After 1 month, during which the patient had no intervening acute episodes, the PET abnormalities had resolved (Butler and Zeman 2008a). This patient went on to develop left-sided hippocampal atrophy (Zeman et al 2012).

In a report, 18F-FDG-PET showed positive correlations between left mesial temporal metabolism levels and anterograde and retrograde memory scores (Mosbah et al 2014).

**Management**

Once a definite diagnosis of transient epileptic amnesia has been established, the physician can reassure the patient with a good prognosis with appropriate antiepileptic drug monotherapy. Though the antiepileptic drug treatment is empirical, the expert view is to prescribe rather small doses either with lamotrigine or levetiracetam, also considering the age of the patient and possible comorbidities (Butler et al 2007; Striano et al 2010; Zeman et al 2012; Mosbah et al 2014). With antiepileptic drug therapy, seizure-freedom is achieved in 96% (Butler et al 2007) to 73% of patients (Mosbah et al 2014). However, despite control of the seizures, interictal memory problems remain, though without progression to deterioration (Butler et al 2007; Zeman and Butler 2010; Zeman et al 2012; Mosbah et al 2014). A patient with transient epileptic amnesia and a left temporal seizure focus developed isolated compulsive versifying, producing multiple rhyming poems, following seizure cessation induced by lamotrigine (Woollacott et al 2015).

Exceptionally, a few patients do not respond to antiepileptic drugs but see good results with neurosurgical intervention (Soper et al 2011; Walsh et al 2011).

**Outcomes**

Transient epileptic amnesia is largely a treatment-responsive syndrome with seizures amenable to antiepileptic drugs though interictal memory deficits might remain.

**Special considerations**

**Pregnancy**

Though complications are rare, see precautions listed in the article titled Pregnancy and epilepsy for care of pregnant women with transient epileptic amnesia.

**Elderly**

For elderly people, it is important to consider their safety and comorbidities.

**References cited**

Asadi-Pooya AA. Transient epileptic amnesia: a concise review. Epilepsy Behav 2014;31:243-5. PMID 24230990


atrophy in transient epileptic amnesia. Epilepsy Behav 2013;28:363-9. PMID 23832133


Butler CR, Zeman A. The causes and consequences of transient epileptic amnesia. Behav Neurol 2011;24:299-305. PMID 22063818

Butler CR, Zeman AZ. Recent insights into the impairment of memory in epilepsy: transient epileptic amnesia, accelerated long-term forgetting and remote memory impairment. Brain 2008b;131:2243-63. PMID 18669495


Kapur N. Transient epileptic amnesia--a clinical update and a reformulation. J Neurol Neurosurg Psychiatry 1993;56:1184-90. PMID 8229029

Kopelman MD, Panayiotopoulos CP, Lewis P. Transient epileptic amnesia differentiated from psychogenic “fugue”: neuropsychological, EEG, and PET findings. J Neurol Neurosurg Psychiatr 1994;57:1002-4. PMID 8057091


Razavi M, Barrash J, Paradiso S. A longitudinal study of transient epileptic amnesia. Cogn Behav Neurol 2010;23:142-5. PMID 20535066


Vuilleumier P, Despland PA, Regli F. Failure to recall (but not to remember): pure transient amnesia during nonconvulsive status epilepticus [see comments]. Neurology 1996;46:1036-9. PMID 8780086

Walsh RD, Wharen RE, Jr, Tatum WO. Complex transient epileptic amnesia. Epilepsy Behav 2011;20:410-3. PMID 21262589


Zeman A, Butler C. Transient epileptic amnesia. Curr Opin Neurol 2010;23:610-6. PMID 20885322


Zeman AZ, Boniface SJ, Hodges JR. Transient epileptic amnesia: a description of the clinical and neuropsychological
features in 10 cases and a review of the literature. J Neurol Neurosurg Psychiatry 1998;64:435-3. PMID 9576532

**References especially recommended by the author or editor for general reading.

ICD and OMIM codes

ICD codes

ICD-9: Localization-related (focal) (partial) epilepsy and epileptic syndromes with complex partial seizures: 345.4

ICD-10: Complex partial status epilepticus: G41.2

Profile

Age range of presentation

19-44 years
45-64 years
65+ years

Sex preponderance

Male > female

Family history

None

Heredity

None

Population groups selectively affected

None

Occupation groups selectively affected

None

Differential diagnosis list

Psychogenic amnesia
Transient global amnesia

Associated disorders

complex focal seizures
mesial temporal lobe epilepsy
secondarily generalized tonic-clonic seizures

Other topics to consider

Alzheimer disease
Drug induced memory disturbance
Dyscognitive focal status epilepticus
Hippocampal and parahippocampal seizures
Memory loss
Mesial temporal lobe epilepsy with hippocampal sclerosis