Case report

An unusual cause of autobiographical memory loss

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Ir J Psych Med 2011; 28(2): 97-99

Abstract

We describe an unusual cause of autobiographical memory loss in a 55 year old man who presented with prominent memory loss for significant events in his life over a period of five years with evidence of patchy memory loss for events prior to this. It was associated with emotional lability and was complicated by a number of tragic events in his life in the previous four years. In addition there were a number of brief episodes (< 30 mins) where he would transiently lose his memory for events including for hours, days or months prior to the event. Neuropsychological assessment confirmed prominent autobiographical memory loss with minimal deficits in other domains. An electroencephalogram (EEG) revealed a simple partial seizure arising from the right temporal lobe, pointing to a diagnosis of Transient Epileptic Amnesia. He was commenced on anti-epileptic medication and responded both subjectively and objectively. There are approximately 94 cases of TEA described in the literature and the diagnostic criteria and postulated aetiology of this illness is discussed here. Clinicians need to have high index of suspicion of epilepsy when assessing a patient with prominent autobiographical memory impairments.

Key words: Autobiographical memory loss; Amnesia; Seizure; Transient epileptic amnesia (TEA).

Case description

A 55-year-old, married, self-employed business man was referred to a specialist memory clinic by his community psychiatric service in 2006, because of his complaint "my memory is going". He had become concerned about his memory problem and the effect it was having on his business and his GP had referred him to his local psychiatric service, querying a depressive illness. His memory loss was for personal events going back particularly over the previous five years but also over his life. He described having no memory of meeting clients or carrying out transactions, only being aware if meetings were documented, subsequently discussed, or if he had written cheques. He described one event where he had forgotten he had accepted a cheque from a client and he found it in his wallet five weeks later. This episode embarrassed him as he had approached this client and requested the money again before the cheque was found. He became reliant on a notebook for all business dealings. When he looked at graduation photographs of his children, he accepted that he must have been there but had no recollection of the occasions. He denied problems with prospective memory, planning or organisational skills. He was driving and reported no problems.

His wife first noticed his memory problem two years prior to presentation in 2004, following an epidural for a left total hip replacement. She acknowledged that the tragic death of his brother, with whom he was very close to, and the death of his daughter's boyfriend, had a devastating effect on him. He developed prominent emotional lability when talking about his brother's death and would become sad and tearful when watching upsetting events on television. There were no biological symptoms of depression or inappropriate jocularity.

He was on oral hypoglycaemics for non-insulin dependent diabetes mellitus diagnosed three years earlier and hypertension, which was controlled on antihypertensives. He drank three units of alcohol per week and was a non-smoker. He had six children, all alive and well and aged between 26 and 16 years. His mother had late-onset Alzheimer's disease and died aged 78. His premorbid personality was of an outgoing individual.

Mental state examination revealed him to be a middle-aged, right-handed man, who was appropriate and forthcoming during the interview. His speech was logical and coherent, with no dysphasia or word-finding difficulties. His mood was subjectively and objectively euthymic, although there was prominent emotional lability when speaking about his brother's death. There were no other features of unresolved grief. He was insightful into his problem and appropriately concerned. Cognitively, there were minor problems in backward tracking on WORLD backwards. He was temporally and spatially orientated and recall was 2/3 on Mini Mental State Exam. Clock drawing test was intact. Performance on Delayed Word Recall (DWR test) showed some minor problems on free recall, which was nonetheless in the normal range with DWR free recall = 5/10, with intact DWR recognition memory.

Diagnosis and differentials were considered. Mild cognitive impairment (MCI) was considered although the memory impairment was atypical for pre-Alzheimer's disease. A vascular aetiology was considered given his vascular risk factors, and he was referred for an MRI brain. His emotional lability, particularly surrounding his brother's death, raised the

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SUBMITTED: FEBRUARY 1, 2010. ACCEPTED: NOVEMBER 16, 2010.

Figure 1a: Normal Resting EEG in the waking state

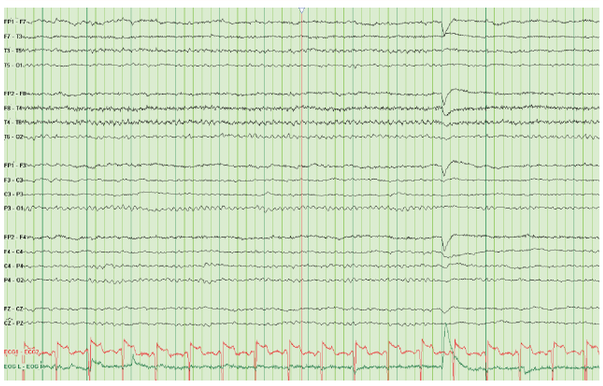
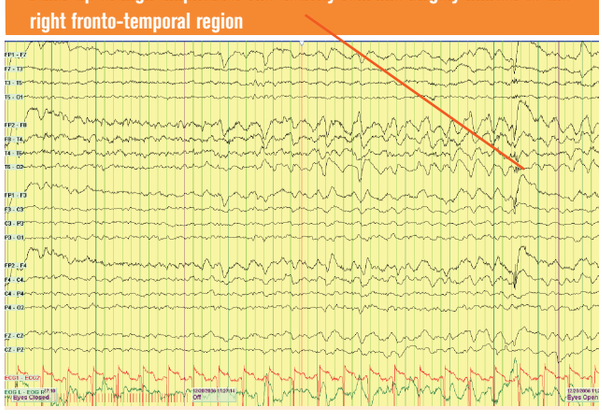


Figure 1b: Seizure begins in the right temporal leads with rhythmic build-up of high amplitude. The activity remains largely limited to the right fronto-temporal region



question of a depressive disorder or unresolved grief. Transient Global Amnesia could also have been considered given the prominent autobiographical memory loss and the elevated emotional state. Given the prominence of autobiographical memory loss, he was referred for a neuropsychological assessment and an EEG.

Baseline neuropsychological testing revealed MMSE of 27/30, CAMCOG 94/107 and tests of new learning/delayed recall were relatively intact. His pre-morbid IQ was predicted to be within the average range based on the National Adult Reading Test (NART2).Wechsler Memory Scale (WMS-III) Logical Memory (story recall) was normal for immediate recall (50th percentile) and delayed recall (37th percentile), with 25th percentile savings (normal range retention). There were false positive responses on Logical Recognition Memory suggesting poor discrimination. Verbal fluency was poor for semantic categories (< 11th percentile) and weak for letter fluency (FAS test 20th percentile). Trail Making Part B was borderline at 12th percentile. Verbal abstraction was weak (CAMCOG Similarities 3/8). Otherwise language and visuo-construction were intact. Autobiographical Memory Interview (AMI)[1] revealed highly significant deficits in recall of personal memories especially prominent over the past five years with deficits extending back to the 1980s, with no significant deficits from childhood and early adulthood.

An MRI showed minimal white matter changes, with normal medial temporal configurations on both sides. The EEG recorded a simple partial seizure from the right fronto-temporal region, lasting 30 seconds in duration *(see Figure 1a and Figure 1b)*. During the EEG he did not appear to lose consciousness nor appear confused. He was referred to a consultant neurologist.

At neurology review, he described two episodes of 'confusion' in the past year, each lasting 15-30 minutes, where he asked repeated questions, yet forgetting that they had happened afterwards. Apart from asking questions repeatedly he was deemed by his wife to be performing normally during the events. One of these episodes occurred after returning from a business trip abroad and on probing he reported that he had no recollection of what occurred on the trip.

He admitted to having one to two episodes of visual disturbance per week over the previous two years, where objects had a reddish glow and sometimes appeared bigger and people and objects became distorted and looked like animals. He denied any change in sense of smell or taste. These episodes would last approximately one minute and there was no obvious change in his level of consciousness, however his wife noticed a vacant look on his face during these episodes. He also reported experiencing sensory abnormalities "a prickly sensation of warmth and pins and needles" at the same time as the visual distortions.

A diagnosis of complex partial seizures with transient epileptic amnesia was made and antiepileptic medication (AED), sodium valproate, was commenced.

Repeat neuropsychological testing six months later revealed a CAMCOG 98/107 – four points higher than baseline. There was no change in category fluency (10th percentile), Trail Making Part B (12th percentile), letter fluency was slightly better (improved from 20th percentile to 30th percentile). Verbal abstraction remained weak (CAMCOG Similarities 4/8). WMS–III Logical Memory (story recall) was much improved with immediate recall at 95th percentile, delayed recall at 91st percentile, with 50th percentile savings. His Logical Recognition Memory was on this occasion entirely normal (29/30). There was no objective change in performance on autobiographical memory. Neuropsychological testing at 12 months revealed no change in episodic memory, language, visuo-construction and executive function. CAMCOG = 96/107. AMI revealed a persistent dense autobiographical memory gap for events at least from 1980s up to six months prior to the assessment, however indicated that autobiographical memory had improved significantly in past six months. Subjectively, he noted a better autobiographical memory recall for the previous six months (probably related to seizure control) but no improvement in the gap prior to that time. From a neurological perspective he continued to have some breakthrough simple and complex partial seizures with no definite episodes of TEA. He was switched to the AED, levetiracetam and more recently, oxcarbazepine. Since then on annual review in the memory clinic, the test findings are similar to previous assessments and the autobiographical memory improvements remain. At his most recent review in 2010, it was noted that there was excellent recall on the WMS–III Logical Memory (story recall) during the test session, but no recall six weeks later, despite prompting and priming.

Discussion

Transient epileptic amnesia (TEA) is a relatively recently described diagnosis in neurology literature,[2] where temporal

https://doi.org/10.1017/S0790966700011526 Published online by Cambridge University Press

lobe epilepsy manifests as episodes of amnesia, often without other cognitive or ictal phenomena.[2-4] Patients are able, during seizures, to engage in whatever activity appears appropriate, implying that, except for the inability to incorporate the ongoing activity and perceptions into long-term memory, their cognitive functioning during the seizure is intact as judged by people around them. Patients also typically complain of prominent interictal memory difficulties.[5] However performance on standard tests of memory is usual normal. The diagnostic criteria of TEA have been drawn up[4] *(see Table 1)*.

According to the diagnostic criteria, there needs to be a history of recurrent witnessed episodes of transient amnesia. In our case report, a classically described amnestic event occurred on at least two occasions in the year prior to presentation, where the patient asked questions continuously for a 15 to 30-minute period and had no recollection of them afterwards. The phenomenon where the subject is constantly asking questions is good evidence that he was amnestic during the seizures.

Recent work has revealed two relatively novel forms of memory impairment in TEA, which are not detected by standard tests,[6,7] accelerated long-term forgetting and remote memory impairment. In accelerated long term forgetting (ALF) individuals learn and initially retain information normally but forget it at an unusually rapid rate over the following days or weeks. ALF was evident in our case report, when the patient displayed excellent recall on the WMS–III Logical Memory (story recall) during the test session, but no recall six weeks later. In remote memory impairment (RMI) there is a patchy loss of autobiographical memories extending back over many years. These forms of memory impairments may explain our patent's significant memory deficits despite having brief amnestic episodes.

The second diagnostic criteria states that cognitive functions other than memory should be judged intact during typical episodes by a reliable witness. His wife did not report any concerns regarding impairment in other cognitive domains during the episodes.

The third criteria states that there should be definite evidence for a diagnosis of epilepsy to be made and in our case report the EEG recorded a simple partial seizure from the right fronto-temporal region and there was a clear-cut response in memory on AEDs. Complete cessation of TEA episodes was achieved with anticonvulsant therapy in 88.5% of cases published[8] and the reminder showed a substantial decrease in attack frequency. In our case, despite continued occasional breakthrough seizures, the AEDs significantly improved autobiographical memory but not until after six months of taking it. The improvements continued into the future but the dense autobiographical memory gap remained for events prior to AED commencement.

Conclusion

Our case report fulfilled the three criteria necessary to make the diagnosis, however the real strength of our case is that we have documented subjective and objective baseline deficits in autobiographical memory, a clear-cut EEG seizure and subsequent autobiographical memory improvements both subjectively and objectively following AEDs.

Cerebrovascular disease is the most commonly identified cause of epilepsy among older patients.[9] The cause of TEA has not been fully elucidated, although a significant cardiac history was common in some case series[4] but not in others.[6] Thus it has been postulated that cardiac-related hypoxic damage to the medial temporal lobe structures may cause the epilepsy in TEA. Our case had a history of two cardiac risk factors, type II diabetes mellitus and hypertension, which are likely to be of significance in the aetiology of his epilepsy.

Table 1: Diagnostic criteria for transient epileptic amnesia[4,8]

- A history of recurrent witnessed episodes of transient amnesia
- Cognitive functions other than memory judged to be intact during typical episodes by a reliable witness
- Evidence for a diagnosis of epilepsy based on one or more of the following:
  - Epileptiform abnormalities on electroencephalography
  - The concurrent onset of other clinical features of epilepsy (eg. lip smacking, olfactory hallucinations)
  - A clear-cut response to anticonvulsant therapy

Our case did have persisting weakness on a test of semantic category fluency (10th percentile range), an executive psychomotor task requiring mental flexibility (Trail Making Part B, 12th percentile), and a brief test of verbal abstraction. In pure cases of TEA interictal cognitive function is typically intact, though there has been some variability in findings, eg. reported mild impairment of naming or fear perception.[8] Our findings suggest the possibility of additional minimal vascular cognitive impairment in our case.

In a case series of 50 patients with TEA[6] a diagnosis of epilepsy was considered initially in only 12 patients, demonstrating that this manifestation of epilepsy is underrecognised. As patients with TEA may present first to psychiatrists, it is important to be aware of it as a diagnosis. It is important to diagnose it as there is an excellent response to AEDs. The concern is that patients may be misdiagnosed as psychogenic, either a depressive or dissociative-type disorder. Collateral history is of huge importance in assessing for discrete episodes of amnesia and, if autobiographical memory deficits are prominent, an EEG should be considered. With a higher index of suspicion the syndrome may not be as rare as is seen in the literature.

Declaration of Interest: None.

References
1. Kopelman MD, Wilson BA, Baddeley AD. The Autobiographical Memory Interview. Bury St. Edmunds (UK): Thames Valley Test Company; 1990.
2. Kapur N. Transient epileptic amnesia – a clinical update and a reformulation. J Neurol Neurosurg Psychiatry 1993; 56: 1184-1190.
3. Kopelman MD, Panayiotopoulos CP, Lewis P. Transient epileptic amnesia differentiated from psychogenic 'fugue': neuropsychological, EEG and PET findings. J Neurol Neurosurg Psychiatry 1994; 57: 1002-4.
4. Zeman AJ, Boniface SJ, Hodges JR. Transient epileptic amnesia: a description of the clinical and neurological features in 10 cases and a review of the literature. J Neurol Neurosurg Psychiatry 1998; 64: 435-443.
5. Gallassi R. Epileptic amnesic syndrome: an update and further considerations. Epilepsia 2006; 47(Suppl 2): 103-5.
6. Butler CR, Graham KS, Hodges KR, Kapur N, Wardlaw JM, Zeman AZ. The syndrome of transient epileptic amnesia. Ann Neurol 2007; 61: 587-98.
7. Manes F, Graham, KS, Zeman, de Lujan Calcagno M, Hodges JR. Autobiographical amnesia and accelerated forgetting in transient epileptic amnesia. J Neurol Neurosurg Psychiatry 2005; 76: 1387-91.
8. Butler CR, Zeman AZ. Recent insights into the impairments of memory in epilepsy: transient epileptic amnesia accelerated long-term forgetting and remote memory impairment. Brain 2008; 131: 2243-2263.
9. Stephen LJ, Brodie MJ. Epilepsy in elderly people. Lancet 2000; 355:1441-1446.