CASE REPORT

A case of recurrent transient global amnesia: don’t forget the hippocampal punctuate diffusion restriction

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Abstract

Transient global amnesia (TGA) is a syndrome characterized by reversible impairment of short-term memory. TGA itself is a benign disease, however, it is reasonable to investigate and exclude sinister causes of global amnesia; such as stroke or a seizure activity. A case of TGA is presented here with special emphasis on the typical TGA lesion that was detected on the patient’s magnetic resonance imaging (MRI) of Brain. In patients with TGA, the typical MRI Brain finding is a tiny focus of diffusion restriction in the mesiotemporal lobe. This finding can potentially aid in early diagnosis and management of TGA. Although in overwhelming majority of cases, the TGA episode occurs only once, yet a second TGA episode can occur rarely. This patient, two years later, had a recurrence of TGA episode, albeit with a normal MRI brain, and without residual changes from prior episode.

INTRODUCTION

Transient global amnesia (TGA) is a relatively uncommon neurological condition that is characterized by reversible disruption of short-term memory. In these patients, MRI of the brain often times reveal punctuate focal diffusion restriction lesion in mesiotemporal region that disappear without long term sequelae. The recurrence rate of TGA is considered low.

We report a case of recurrent TGA, with typical MRI lesion detected during the first episode.

CASE DESCRIPTION

A 51-year-old lady with a past medical history of migraine, presented to Emergency Department with transient loss of memory. The patient was well prior to this episode of memory loss. Earlier, she had played badminton and went home and slept, however, she woke up confused and was noted to be repeatedly asking her husband how she got home. The loss of memory was global and persisted for 3–4 h. The patient’s family, who witnessed this episode, did not notice any unusual behaviour or automatism during this period. She came to the Emergency Department, where her vital Signs were stable. Her neurological examination was normal. Her Investigations revealed a normal blood glucose, full blood count, liver and kidney function. Non-contrasted computed tomography of the brain was unremarkable. Her electroencephalography (EEG) was normal as well.

Her ultrasound dopplers of carotids did not reveal any hemodynamically significant stenosis. Her brain MRI performed at 36 h after onset of symptoms revealed a right medial temporal lobe, tiny focus of high signal intensity on diffusion weighted imaging (DWI), with corresponding apparent diffusion coefficient (ADC) signal loss as shown in Fig. 1a and b.
Time of flight magnetic resonance angiography did not reveal any stenosis in anterior or posterior circulation. The patient was diagnosed to have TGA and was discharged well. At the follow-up visit, she was in good health.

It was noted that there was no family history of TGA or seizure disorders.

Two years later, she self-referred to outpatient neurology clinic a few days after a repeat episode of amnesia. There were no antecedent symptoms or aura. This time, reportedly she was at the gym, met a new gym instructor but subsequently had no recollection of that event. The patient’s son received multiple calls from her asking if she was supposed to pick him up, who also noted that the patient seemed slightly disoriented on the road and could not keep to the lane very well. She had both anterograde and retrograde amnesia. This episode also lasted few hours.

Again her neurological exam was normal. An MRI of the brain performed a week later this second episode did not reveal any signal changes. Her EEG was unremarkable.

Notably, the prior mesiotemporal lobe signal changes from a couple of years ago did not leave any residual signal changes as evidenced by a normal T1, T2, DWI and FLAIR (Fluid Attenuation Inversion Recovery) sequences. This episode also lasted few hours.

DISCUSSION

TGA is a neurological syndrome characterized by abrupt onset of transient and reversible disruption of short-term memory. The incidence of TGA is estimated to be 5–32 per 100,000 people every year. This case satisfied the diagnostic criteria for TGA as suggested by Hodges and Warlow [1] during both episodes, i.e. (i) Attacks must be witnessed. (ii) There must be anterograde amnesia during the attack. (iii) There should be no clouding of consciousness or loss of personal identity and the cognitive impairment is limited to amnesia. (iv) There should be no associated focal neurological deficits. (v) Epileptic features must be absent. (vi) Attack must resolve within 24 h. (vii) A patient with head injury or active epilepsy is excluded.

Thus, collateral history from the witnesses can be helpful in making the diagnosis, however, in case of diagnostic uncertainty, it is important to investigate and exclude sinister causes of Global amnesia such as posterior cerebral artery transient ischaemic attack (TIA), transient epileptic amnesia (TEA) and hypoglycaemia.

Personal history of migraine is associated with TGA and common precipitating events include stress, whether physical or emotional, exposure to cold water and sexual intercourse [1]. Our patient had a history of episodic migraine and on both occasions she had physical exertion antecedent to the TGA event.

In patients with TGA, MRI of the brain can reveal a 2–3 mm punctuate focal diffusion restriction lesion in mesiotemporal region. This ‘lesion’ is well recognized in literature and aids in the diagnosis of TGA. It has a reported incidence range from 11.5 to 84% [2].

The timing of MRI from the onset of symptoms may have an impact on the detection rates for these tiny lesions, with the highest incidence between 12 and 72 h of symptoms onset [3].

Higher detection rates for this tiny lesion can be accomplished with repeated scanning at later time intervals, higher field strengths, dedicated protocols such as a DWI resolution of B = 2000, and a thin DWI slice thickness of 2–3 mm [2, 4].

These lesions show complete resolution on follow-up imaging performed one to six months after the index event [5] a fact demonstrated in this patient’s case as well.

TGA is a benign neurological disorder and cannot be equated to and treated as stroke. The occurrence of these tiny infarct-like signal changes in TGA has produced a debate over the possibility of a cerebrovascular aetiology. However, there has yet to be vigorous evidence supportive of an ischaemic aetiology. Thus far, interventions such as anti-platelet and statin therapy are not warranted in TGA [6–11]. Once the diagnosis of TGA is established, all the patient needs is reassurance.

Figure 1: (a) Axial MRI brain, DWI sequence shows punctuate high signal in medial temporal lobe, as pointed out by white arrow. (b) Corresponding ADC mapping shows signal loss as pointed out by black arrow
Although the exact pathobiology remains elusive, it is possible, that this TGA lesion represents a tiny focus of diffusion restriction from ischaemia with subsequent cytotoxic oedema, rather than an acute infarction, as suggested by prior studies that performed follow-up MRI of brain [5].

Unlike TEA or TIA, the estimated recurrence rate of TGA is low [1]. In a review of 142 case reports, the estimated annual rate of TGA recurrence was 5.8%. Intervals between the two attacks varied from 1 month to 1 year. In 35 group studies (n = 1259) and 52 case reports (n = 94 patients) recurrences were noted for 138 patients (10.19%) [12].

Some have reported that familial cases of TGA may be associated with higher recurrence of TGA [13]. Other studies have raised the possibility of an association between recurrent TGA and primary progressive aphasia [14].

Predictors of TGA recurrence are yet to be ascertained.

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CONFLICT OF INTEREST STATEMENT
The authors declare no competing interest.

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CONSENT
Written informed consent was obtained.

GUARANTOR
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