Hippocampal Dot Sign in Transient Global Amnesia; A Neurologist's Experience

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Abstract

Transient global amnesia, coined by Fisher and Adams, is characterized by a sudden, complete inability to retain new information, lasting several hours, in middle-aged or older persons, with preservation of alertness and all other cognitive functions. It affects 3 to 10 persons per 100,000 people. It has a benign prognosis with low rates of recurrence. MRI brain taken during the episode is often normal and often shows dot like diffusion restriction in hippocampus when MRI brain is taken 24 to 72 hours after the ictus. Presence of hippocampal dot sign is helpful in confirming the diagnosis of TGA and ruling out the usual differential diagnosis.

Key words: Transient global amnesia, Hippocampus, CA 1 region, Hippocampal dot sign, MRI

Introduction

Transient global amnesia (TGA) is a clinical syndrome characterized by anterograde amnesia, mild retrograde amnesia, and confusion lasting up to 24 hours. Clinically, patients have disorientation in time and often ask questions repeatedly regarding the day's events. Common differential diagnosis are transient ischemic attack (TIA), transient epileptic amnesia (TEA), psychogenic amnesia and post traumatic amnesia. MRI brain taken during the episode is often normal. Diffusion weighted images taken 24 to 72 hours after the ictus often shows dot like area of restricted diffusion either unilaterally or bilaterally in cornu ammonis region 1 (CA1) of hippocampus, the hippocampal dot sign (HDS). Detection rates of HDS is improved by high resolution MRI (3T), thin cuts with higher b value and by delaying the MRI to 24 to 72 hours after the ictus. It has a benign prognosis with low chances of recurrence.

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Case report

A 51-year-old neurologist from India along with a fellow neurologist and few friends went for a trip to Pattaya. On second day they went for undersea walking at a depth of 3 meters. During the descent he (Neurologist) had ear block and he had to do Valsalva manoeuvre several times. His symptoms started around 4 hours after the undersea walking. The last thing he remembers is that he had snacks from a restaurant. After that he was asking them when did we come here? What is this place? When are we going back? He was repeatedly asking the same questions. He was complaining of mild headache during this period. When they reminded him that he was asking the same questions repeatedly, he commented that he may be having transient global amnesia. They took him to Bangkok hospital, Pattaya where he was seen by a neurologist and underwent MRI Brain which was normal. Neurologist documented recent memory impairment and retrograde amnesia. Language functions were normal. His blood pressure was 160/94 mm of Hg at hospital. He was on azilsartan 40 mg daily for the last 1 year for hypertension and blood pressure was under control. He has a history of very rare episodes of migraine without aura since college days. There was no history of any alcohol or illicit drug usage. Basic metabolic panel showed

normal blood sugar. HbA1C was 6.4%. Since MRI brain was normal, his friends took him back to hotel after dinner and he had a peaceful sleep. Next day when he got up, his friends described him all the events happened during the previous evening, but he does not remember the travel to hospital, hospital itself or the experience of MRI or even the doctor who attended him. It was transient as it lasted few hours only. It was truly global as he did not have any memory of the visual, verbal or emotional experience he had. MRI brain was repeated 48 hours after the incident which showed bilateral hippocampal dot like hyperintensity in CA1 region in diffusion weighted images with corresponding hypointensity in apparent diffusion coefficient images (Hippocampal Dot Sign). Figure [1], Figure [2].

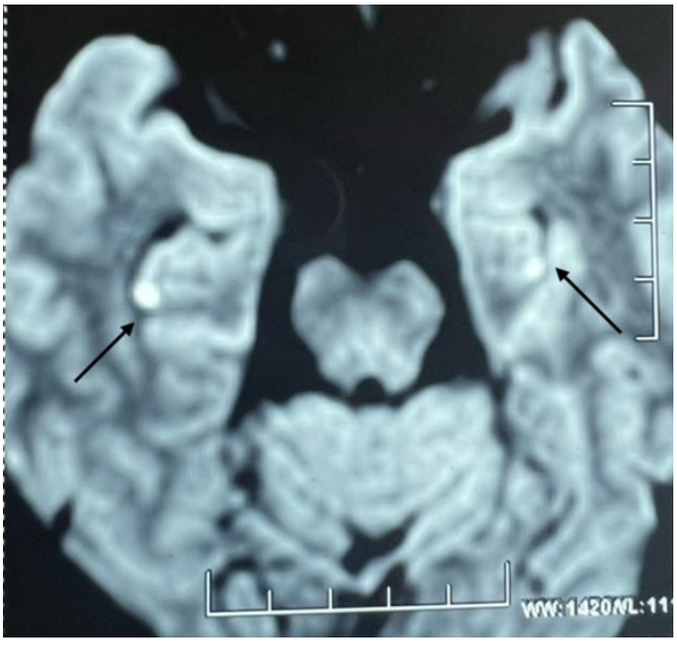

Figure 1 Axial MRI brain diffusion weighted image showing dot like hyperintensity in CA1 region of both hippocampi

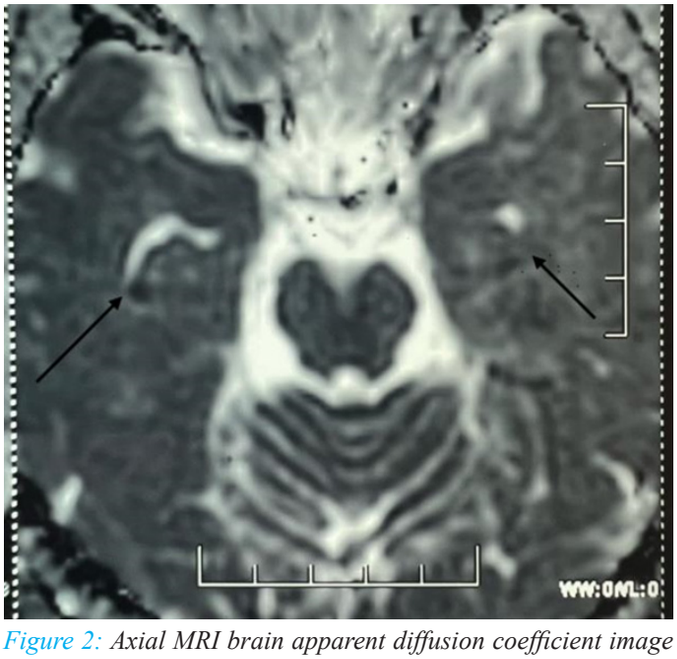

Figure 2: Axial MRI brain apparent diffusion coefficient image showing hypointensity in both hippocampi, CA1 region

Discussion

Transient global amnesia (TGA) is a reversible, benign, mostly non-recurrent clinical syndrome of anterograde amnesia lasting up to 24 h, manifesting as repetitive questioning and occasionally retrograde amnesia, without any gross neurological deficit.[1] In most patients (up to 85%), TGA is preceded by precipitating events. Events such as emotional stress, significant physical exertion, exposure to extreme temperatures, hot and cold baths or showers, high-altitude conditions, Valsalva manoeuvre, acute illness, sexual intercourse, severe pain or medical procedures (especially angiography) is often present. In a series of 277 cases with TGA, the mean age was 62 years, and slightly more men than women were affected. Mean duration of episodes were 6 hours, with most lasting 2 to 12 hours.[2] The lifetime recurrence rate is 2.9% to 23.8% with most patients experiencing 3 or fewer recurrences. Predisposing factors for recurrence include the presence of migraine, depression and sexual activity as a trigger factor. It is generally a clinical diagnosis and various criteria are proposed.[3,4]

Diagnostic criteria for TGA

Criteria by Caplan[3]	Criteria by Hodges and Warlow[4]
Attack is witnessed	Attack is witnessed
Dysfunction limited to repetitive queries and amnesia	Clear anterograde amnesia
No other major neurological symptoms or signs	No clouding of consciousness, cognitive deficit or loss of personal identity
Memory loss is transient, usually lasting hours to a day	Attack resolves in 24 hours
	No focal neurological signs during or after the attack
	No epileptic features
	No recent head injury or active epilepsy

The pathophysiology of TGA is not well understood but may be related to impaired venous drainage of the hippocampus. A single, definite etiology has not been determined, although epidemiologic and imaging data support several putative pathophysiologic processes, including vascular, migraine, epileptic, and psychogenic mechanisms. Cerebral ischemia as a cause of TGA is considered unlikely. There is no apparent increased risk of cerebrovascular events in patients who have had an episode of TGA. Paradoxical cerebral emboli, favoured by Valsalva manoeuvres triggering TGA has also been considered. In patients with a previous episode of TGA, controlled Valsalva manoeuvre could not elicit TGA in isolation and the interplay of other simultaneous factors is needed[5]. In another study ten out of 21 patients reported Valsalva-like activities preceding TGA. In these patients a retrograde flow in internal jugular vein was seen more frequently than in those who did not report preceding Valsalva-like activities. Internal jugular vein valve incompetence is postulated to provoke a transient mesial temporal ischaemia by venous congestion. Their results lend support to the hypothesis that TGA may be attributable to venous congestion,

and consequent venous ischaemia to bilateral diencephalic or hippocampal structures[6]. Atrial septal defects seem to be much more frequent in TGA patients (55%) as compared to the general population (25%). Duplex sonographic studies found incompetent internal jugular vein valves significantly more often in TGA patients than in control subjects (73% vs. 31%), which could favour venous congestion.[7] Recent evidence suggests an association between TGA and migraine headaches as well as takotsubo cardiomyopathy. MRI brain taken during the event is often normal. MRI Brain preferably 3T with thin (2mm or 3mm) cuts with higher b value (2000 or 3000 s/mm2) done 24 to 72 hours after the ictus is recommended for detection of hippocampal changes.[5] These findings are typically unilateral and tend to be small (1–3 mm), high-signal foci found in the CA1 field of the hippocampus. Hippocampal dot sign is present in up to 70% of patients with a mean size of 4 mm (range 1.7–8.6 mm) if MRI is done 12–48 h after symptom resolution. These lesions can be bilateral and even multifocal. In a retrospective series involving 390 patients with transient global amnesia, 70% had these lesions, which were most often apparent 12 to 48 hours after the episode and were often fleeting but sometimes persisted for up to several days. [8,9] These lesions can be found incidentally in patients who have cognitive or emotional aberrations without amnesia.[10]

Conclusion

TGA is a clinical diagnosis. Diagnosis is supported by hippocampal dot sign. Optimal method to detect HDS is by doing higher Tesla thin slice MRI brain with higher b values, done 24 to 72 hours after the ictus.

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