VOLUME - 14, ISSUE - 07, JULY - 2025 • PRINT ISSN No. 2277 - 8160 • DOI : 10.36106/gjra

Original Research Paper

Radio-Diagnosis

CASE SERIES: BILATERAL HIPPOCAMPAL BRIGHT SPOTS IN TRANSIENT GLOBAL AMNESIA

Dr. Sangeetha. K*	Junior Resident In Radio Diagnosis, Topiwala National Medical College And B.Y.L Nair Hospital, Mumbai *Corresponding Author
Dr. Sunita Tibrewala	Professor And Head In Radio Diagnosis, Topiwala National Medical College And B.Y.L Nair Hospital, Mumbai
Dr. Srishti Rai	Junior Resident In Radio Diagnosis, Topiwala National Medical College And B.Y.L Nair Hospital, Mumbai

ABSTRACT Transient Global Amnesia (TGA) is a sudden, temporary episode of memory loss not attributable to common neurological conditions such as epilepsy or stroke. It primarily affects short-term memory lasting for 1–24 hours and is often associated with stressful events or physical exertion. The patient did not exhibit any focal neurological deficits. Cognition and alertness remain preserved. Repetitive questioning is typical, even after being answered. It is a self-limiting disorder, and no specific treatment is indicated for a typical episode.

KEYWORDS : Transient Global Amnesia (TGA), MRI, punctate diffusion hyperintensities, hippocampus.

INTRODUCTION

Transient Global Amnesia (TGA) is characterized by abrupt onset of anterograde amnesia, sometimes accompanied by retrograde impairment, in the absence of other neurologic deficits. It resolves within 24 hours. First described in the 1950s [1], TGA predominantly affects middle-aged to elderly individuals, with an incidence of ~5–10 per 100,000 annually and a slight female predominance [2].

Clinically, patients present with an inability to form new memories, temporal disorientation, preserved personal identity, repetitive questioning, and no motor/sensory deficits or altered consciousness. Resolution usually occurs within 4–6 hours and always within 24 hours, leaving no residual deficits.

The pathophysiology remains unclear; proposed mechanisms include transient hippocampal dysfunction due to metabolic stress, ischemia or venous congestion (e.g., Valsalva-like maneuvers), migrainous phenomena, or transient epileptic activity (though EEG is typically normal) [3]. The CA1 sector of the hippocampus is particularly vulnerable to such stress.

High-resolution MRI with diffusion-weighted imaging (DWI) has revealed small, punctate hyperintense foci in the hippocampal CA1 region, corresponding to restricted diffusion on ADC maps [4,5]. These bright spots are often unilateral but may be bilateral, especially when imaging is done 24–72 hours after onset [2]. They resolve within days without structural damage. Recognizing these findings aids differentiation from stroke, limbic encephalitis, or seizures, avoiding unnecessary interventions.

MATERIALS AND METHODS

Study Design

This retrospective, observational, and descriptive study was conducted on 14 patients (aged 25–80 years) presenting with sudden-onset memory loss at Topiwala National Medical College over 12 months (Jan 2023–Feb 2024). MRI brain was performed using 1.5T Philips MRI with sequences including T1, T2, STIR, SWI, FLAIR, IR, and DWI.

Inclusion And Exclusion Criteria

Included: Patients referred for brain MRI for evaluation of sudden memory loss.

Excluded: Patients with metallic implants or claustrophobia.

Case Study

Among 14 patients, punctate diffusion hyperintensities were observed with Unilateral hippocampus in 3 patients and Bilateral hippocampi in 1 patient. Corresponding ADC maps showed low signal in all positive cases.

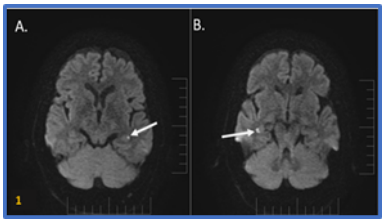

Figure 1: MRI showed punctuate diffusion hyperintensities in bilateral Hippocampi (FIG 1A-B). Corresponding ADC maps showed low signal.

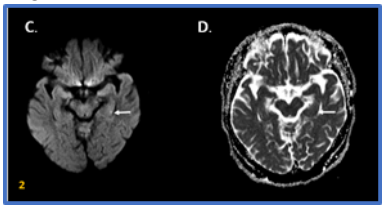

Figure 2: Punctuate hyperintensity seen in left hippocampus with subtle hypo intensity in the corresponding ADC map (image C,D)

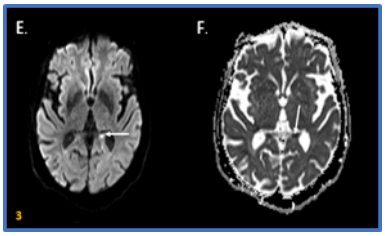

Figure 3: Images E and F , showing small focal diffusion restriction involving the tail of left hippocampus

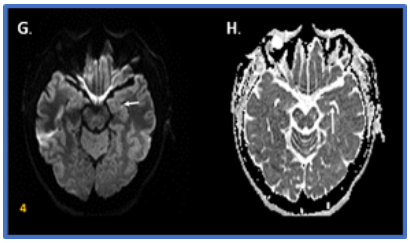

Figure 4: Punctuate diffusion hyperintensity (image G) with corresponding low signal on ADC images (image H)

DISCUSSION

Our case series demonstrates bilateral punctate areas of restricted diffusion in the CA1 hippocampal region—a finding considered specific for TGA in the correct clinical context [2]. While unilateral lesions are more commonly reported, bilateral involvement may indicate symmetric transient metabolic dysfunction [4].

Timing Of Imaging Is Crucial: DWI abnormalities peak at 24–72 hours and may be missed if imaging is performed too early (<12 hours) or too late (>7 days) [2,5]. Hence, thin-slice hippocampal DWI is essential when TGA is suspected.

These non-ischemic lesions likely reflect transient disruption in cerebral blood flow, venous congestion, or excitotoxicity affecting the CA1 subfield [3]. They are distinguishable from stroke (territorial), seizures (diffuse), and limbic encephalitis (T2/FLAIR changes and enhancement).

CONCLUSIONS

TGA is primarily a clinical diagnosis, but characteristic hippocampal diffusion restriction on MRI—particularly bilateral CA1 lesions—provides compelling supportive evidence. Our case series emphasizes the Importance of imaging within 24–72 hours and utility of high-resolution DWI focused on hippocampi. Radiologists should remain alert to this radiologic signature to avoid misdiagnosing stroke or encephalitis and guide appropriate, conservative management.

REFERENCES:

1. [Abe O, Aoki S, Hayakawa K, et al. Diffusion-weighted MRI of the brain in patients with transient global amnesia. AJNR Am J Neuroradiol. 1998;19(2): 239–243.
2. Sedlaczek O, Hirsch JG, Grips E, et al. Detection of delayed focal MR changes in the hippocampus in transient global amnesia. J Neurol Neurosurg Psychiatry. 2004;75(11):1540–1544.
3. Bartsch T, Deuschl G. Transient global amnesia: functional anatomy and clinical implications. Lancet Neurol. 2010;9(2):205–214.
4. Bartsch T, Alfke K, Stingele R, et al. High-resolution magnetic resonance imaging reveals hippocampal lesions in transient global amnesia. Ann Neurol. 2006;59(6):829–836.
5. Shinnar S, Berg AT, Kang HC, et al. Transient global amnesia: the role of hippocampal imaging. Neurology. 2007;69(23):2119–2125.