

Hippocampal dot sign in transient global amnesia

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A 56-year-old man with rare migraine without aura since childhood was brought to the emergency department by his wife with complaints of memory impairment since morning. He was repeatedly asking the date and time despite telling him the date and time. He did not remember when he packed his bag with which he was supposed to go for a trip in the morning. Otherwise, his behavior was normal. There was no difficulty in comprehension, naming, identifying objects, or persons. There were no headache, vomiting, or visual symptoms. There was no history of hypertension, diabetes mellitus, or dyslipidemia. On examination, he had anterograde and retrograde memory impairment of events of the previous day evening. Computerized tomography head and electroencephalogram were normal. His metabolic parameters were normal. Magnetic resonance imaging (MRI) brain done 48 h after the resolution of symptoms showed bilateral dot-like diffusion-weighted image (DWI) hyperintensity in the lateral hippocampus (cornu ammonis I [CA1] region) with corresponding hypointensity in apparent diffusion coefficient images (Fig. 1). We did not take any follow-up MRI brain to look for resolution of signal changes.

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]. Long-term memory, language, visual-spatial orientation, and executive functions are typically preserved. This syndrome usually occurs between the fifth and seventh decade of life and is more common in males. The mean duration is 6 h with, most lasting 2–12 h. The reported incidence is 5–10/100,000 population. The common precipitating factors include emotional stress, physical exertion, pain, and the Valsalva maneuver. Various theories have been postulated, including venous congestion, focal ischemia, migraine as well as epilepsy. None of these, however, clearly explain all the condition's characteristics [2]. Classic DWI findings in TGA consist of unilateral or bilateral small punctuate hyper-intense lesions in the CA1 region of the hippocampal cornu ammonis (hippocampal dot sign). These changes generally appear after symptom resolution, and the highest rate of detection is approximately 2 days after symptom onset. Hippocampal

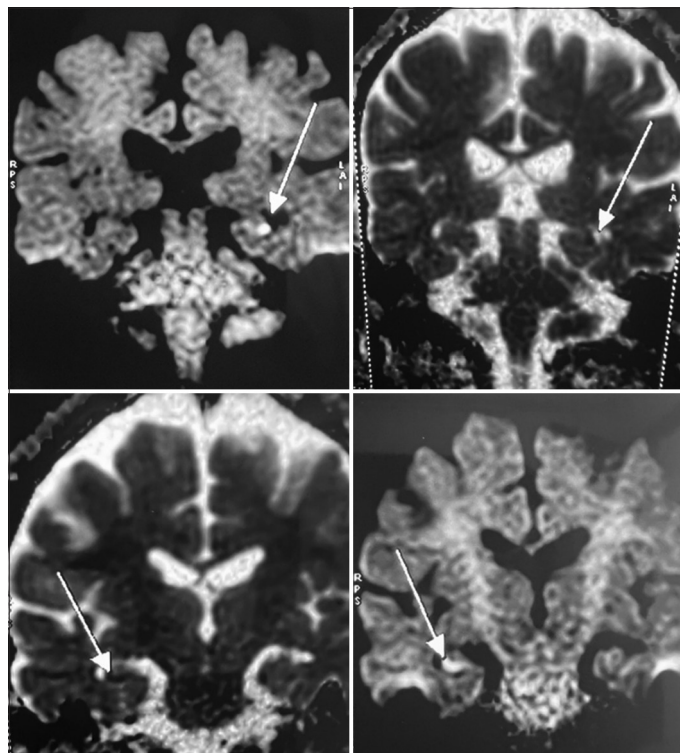



Figure 1: Magnetic resonance imaging brain showed bilateral dot-like diffusion-weighted image hyperintensity in the lateral hippocampus (cornu ammonis I region) with corresponding hypointensity in apparent diffusion coefficient images

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 [3]. Lesion detection is enhanced by high-resolution DWI, higher B-values, thin slice thickness (2–3 mm), and a delay of 48–72 h between symptom onset and scanning. These lesions can be bilateral and even multifocal. The highest lesion detection was achieved for DWI with $b = 2000/3$ mm or $b = 3000/3$ mm at 3 days post-onset. The lesion detection rate of the initial DWI was higher for $b = 2000$ or 3000 (54%) than for $b = 1000$ (38%) with equal section thicknesses (3 mm). When no lesion is detected by DWI within 24 h after onset, follow-up DWI is recommended several days later [4]. The condition rarely recurs in ~5% of patients and does not require any treatment.

Structurally, the cornu ammonis or hippocampus proper is divided into four distinct zones, based upon their sensitivity to

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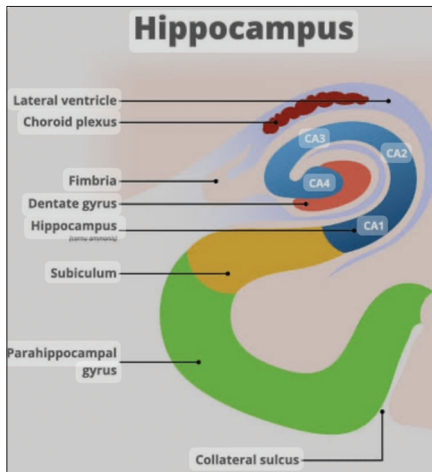


Figure 2: Structure of the hippocampus proper

Table 1: Adapted from Hodges and Warlow^[7]

Attack is witnessed
Clear anterograde amnesia
No clouding of consciousness, cognitive defect, or loss of personal identity
Attack resolves within 24 h
No focal neurologic signs during or after the attack
No epileptic features
No recent head injury or active epilepsy

hypoxia and histological differences. CA1 (Sommer's sector) is known as the vulnerable zone, CA2, and CA3 as the resistant sector, and CA4 as the medium vulnerability sector (Fig. 2). It is understood that neurons in this region of the hippocampus are

particularly susceptible to metabolic stress, suggesting that TGA may result from a physiological temporary inhibition of memory formation. The fact that DWI changes in TGA do not appear in the hyperacute phase, unlike the restricted diffusion seen in ischemia, further supports the theory that these imaging findings are due to an entirely different pathological process. These lesions have been noted to resolve on follow-up MRI performed after 6–12 months [5]. Various clinical criteria have been proposed for the diagnosis of TGA (Table 1) [6,7].

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