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Isolated Transient Global Amnesia Following Hippocampal Stroke

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Dear Editor,

Transient global amnesia is a sudden, temporary condition marked by severe memory loss, affecting both anterograde and retrograde amnesia. It occurs without changes in consciousness or other cognitive issues, typically resolving within 24 hours with no lasting effects, aside from mild memory loss in some cases.

Isolated hippocampal infarction (HI) is relatively rare, which accounted for 0.03% of all strokes[1].We report a case of TGA with findings in the hippocampus as seen on DWI, in doing so, we hope to provide more data to the medical community regarding both incidence and areas of involvement in ischemic TGA to better our understanding of this condition.

The patient, a 64-year-old female, presented with transient memory loss that lasted for eight hours. On the day she was admitted, she remembered waking up around 7:00 AM and getting dressed to watch a GAA match at 4 PM. She remembered going to the match but couldn't recall any details of it. Her family reported that she appeared confused at the end of the game and didn't recall anterograde or retrograde events. She couldn't recall who won the game and later recognized gaps in her memory, with her last clear memory being before the match.

In the emergency department, a comprehensive neurological examination found no focal deficits. The patient was awake and alert upon arrival, but she was confused. She was unable to recall the date or the current president. Her Glasgow Coma Scale score was 14. Both the National Institutes of Health Stroke Scale (NIHSS) and the Cincinnati Prehospital Stroke Scale scores were normal. An urgent CT brain showed no intracranial hemorrhage, mass, or acute ischemia.

A T1-weighted, T2-weighted, and diffusion-weighted MRI was done within 48 hours which showed Two adjacent sub cm foci of restricted diffusion noted in the deep medial left temporal lobe/hippocampus on the medial aspect of the trigone of the left lateral ventricle.

Her memory improved later in the stay, and all of her amnestic symptoms were gone the next morning. She was then started on dual antiplatelet and he was also put on a statin.

For neurologists, the sudden development of solitary amnesia presents an interesting issue because the absence of concomitant symptoms may lead to a false diagnosis. The annual incidence of TGA is predicted to be 5-32 per 100,000 people. This instance met the diagnostic requirements for TGA as given by Hodges and Warlow[2].

An MRI of the brain in patients with TGA can indicate a 2-3 mm punctuate focal diffusion restriction lesion in the temporal area. This 'lesion' is well documented in the literature and aids in the diagnosis of TGA. It has a reported incidence of 11.5 to 84%[3].

Unlike TIA, TGA has a low predicted rate of recurrence[2]. Based on a study of 142 case reports, the expected annual rate of TGA recurrence was 5.8%. The intervals between the two attacks ranged from one month to a year. In 35 group trials (n = 1259) and 52 case reports (n = 94 individuals), recurrences were observed in 138 patients (10.19%)[4]. It is evident that the frequency of acute ischemic lesions in individuals who present clinically as TGA is higher than previously believed. Therefore, to identify ischemic lesions that may be associated with cardiac dysfunction or myocardial damage, cerebral MRI should be carried out in individuals who have clinical TGA.

Declarations of Conflicts of Interest:

None declared.

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