

Global amnesia: Frequency of silent brain infarction

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ABSTRACT

To assess the frequency and pattern of acute DWI lesions outside the hippocampus in patients with Transient Global Amnesia (TGA). Patients who presented with TGA and were hospitalized to our hospital between January 2010 and January 2017 were retrospectively assessed. TGA diagnostic criteria were met by all of the patients. We looked at the imaging and clinical data of all patients who had a high-resolution diffusion-weighted

MRI within 72 hours after onset of symptoms. The research comprised a total of 126 cases. One or more acute lesions in the hippocampus CA1-area were present in 53% (n=71/126) of the patients. In 11% of the cases (n=14/126), additional acute DWI lesions were discovered in other cortical areas. All of the patients who had DWI lesions outside of the hippocampus had TGA-like neurological symptoms. MRI demonstrates acute ischemic cerebral lesions in a significant proportion of clinical TGA patients. As a result, in individuals with TGA, a cerebral MRI should be conducted to rule out cardiac involvement and detect stroke chameleons.

Key Words: *Transient global amnesia; High-resolution*

INTRODUCTION

Transient Global Amnesia (TGA) is a neurological disease characterized by the onset of anterograde amnesia that lasts for less than 24 hours. TGA's pathophysiological causes are yet unknown, but it is not thought to be caused by an ischemic stroke. Ischemic stroke, on the other hand, can induce acute transitory amnesia, and distinguishing it from TGA based on clinical presentation alone might be challenging. In the diagnostic workflow, Magnetic Resonance Imaging (MRI) with high-resolution Diffusion-Weighted Imaging (DWI) can provide useful information. As a result, the goal of our investigation was to see how common ischemia lesions outside of CA1 were on high-resolution DWI in individuals with TGA.

Methods

Study populations- Patients who presented with TGA and were hospitalized at our hospital between January 2010 and January 2017 were retrospectively assessed. All patients were found by searching our hospital's digital patient records (SAP Clinical Workstation, SAP, Germany) for in-patients with the ICD-10 diagnosis TGA. We screened for TGA as a suspected diagnosis in the emergency department rather than as a final diagnosis to prevent selection bias. After admission, an MRI was usually done the next morning. As a result, in patients with multiple brain infarcts, the presumed diagnosis was altered from TGA to stroke as the final diagnosis. In the emergency room, each patient was assessed by a neurologist. TGA was defined by established diagnostic criteria: witnessed attack, anterograde amnesia, no loss of consciousness or personal identity, cognitive impairment limited to amnesia; no other focal neurological symptoms during or after the attack; no epileptic features or active epilepsy; no recent head injury; and symptom resolution within 24 hours [1]. TGA was diagnosed in patients who had clinical TGA with a single or bilateral isolated punctuate DWI lesion in the hippocampus. Ischemic stroke was detected in patients with clinical TGA and additional acute ischemic lesions on DWI. Age, sex, the timing of symptom onset, and cardiovascular risk factors were among the demographic and clinical data collected from patients.

Acquisition and analysis of images- A 3 T MR scanner was used for all MRI exams (Magnetom Trio; Siemens AG, Germany, 32-channel head coil). High-resolution DWI, T2*-weighted imaging, MR-angiography, and fluid-attenuated inversion recovery were all part of the conventional MRI stroke protocol (FLAIR). Slice thickness 2.5 mm, repetition time TR 8900 ms, echo duration TE 93 ms, slice gap 0, b values 0, and 1000 mm²/s were the sequence parameters for high-resolution DWI. DWI was evaluated for probable lesions outside the hippocampus level, in addition to the predicted abnormalities in the hippocampal CA1-area. Statistical methods- The percentage of patients with additional ischemia lesions on DWI in all

clinically diagnosed TGA patients throughout the study [2]. We employed the Mann-Whitney U test for continuous variables and the 2 test for categorical variables to compare groups of patients with and without additional ischemic lesions. A P value of 0.05 (two-tailed) was utilized as a statistical significance criterion in all analyses. We utilized the Statistical Package for Social Sciences to analyze the data (SPSS Version 23, IBM, USA).

RESULTS

Between January 2010 and January 2017, we found 126 patients with clinically confirmed TGA and 3 T MRIs within 72 hours of symptom start, with a mean age of 66 (10) years with 66 (52%) of them being female. We identified and removed 78 patients with clinically confirmed TGA who did not have a 3 T MRI for various reasons in addition to the 126 patients. One or more acute punctuate DWI lesions were identified in hippocampal CA1-areas in 56% (71/126) of all patients [3]. Unilateral hippocampal lesions were seen in 41% of patients (52/126) and bilateral hippocampus lesions were found in 15% of patients (19/126). Patients with additional acute DWI lesions in brain locations other than the hippocampus were found in 11% of cases (14/126). Eight patients had cortical lesions in the anterior circulation, one patient had cortical lesions in the posterior circulation, and two patients had subcortical lesions in the posterior circulation. The anterior and posterior circulations of three patients had cortical DWI abnormalities. Individuals with extra DWI lesions had a considerably greater rate of coronary heart disease (25% vs. 6.8%, p=0.034), whereas arterial hypertension and female sex were not significantly more common in these patients. Age, past stroke frequency, and other cardiovascular risk factors such as atrial fibrillation were not different between the two groups [4].

DISCUSSION

In this 3 T MRI investigation, nearly 10% of patients with acute amnesia clinically considered to be TGA had acute lesions on high-resolution DWI outside the hippocampus level, in addition to the expected and verified punctuate hippocampal lesions in the CA1-area. A transitory disruption in the hippocampal functional memory network is thought to be the source of acute and transient amnesia in TGA [5]. As a result, a punctuate DWI lesion in the hippocampus area can be identified in many patients clinically presenting with TGA. Recent data has shown that ischemic amnesia, or transitory amnesia, can be caused by ischemia lesions affecting the hippocampus circuit. In one of these investigations, 1.2% of patients with acute amnesia were later found to have cerebral ischemia, but the exact rate of ischemic amnesia could not be calculated because only 25% of clinical TGA patients had an MRI and information on DWI image resolution was unavailable [6]. Second, because they met all clinical TGA criteria, only

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two patients with ischemic amnesia were considered certain TGAs before imaging. In our stroke center, we use 3-Tesla MR imaging to identify stroke events in patients who are clinically presenting with TGA. More crucially, we use a high-resolution DWI with a 2.5-mm slice thickness and no slice gap, which allows us to detect tiny lesions that would otherwise go undetected at lower image resolution. Additional acute lesions were mostly found in the anterior circulation or throughout numerous vascular areas, implying heart (or aortic arch) involvement [7].

The hippocampus is known as the “forgotten” border zone of brain ischemia, therefore this could be significant. Coronary heart disease and arterial hypertension were more common in patients with additional acute lesions, increasing the risk of acute cardiac dysfunction. Overall, high emotional or physical stress as a frequent triggering event combined with excessive sympathetic activation may cause cardiac diastolic and/or systolic output dysfunction, damaging vulnerable brain areas such as the hippocampus. Furthermore, these patients were considerably more likely to have a history of coronary heart disease [8]. In addition, two patients were found to have Takotsubo Syndrome (TTS). TTS, like TGA patients, is an acute but temporary left ventricular cardiac failure with a previous mental or physically stressful event.

Overall, high emotional or physical stress as a frequent triggering event combined with excessive sympathetic activation may cause cardiac diastolic and/or systolic output dysfunction, damaging vulnerable brain areas such as the hippocampus. This probable etiology of TGA and ischemia amnesia is supported by a newly published study that found that individuals with TGA had a twofold higher risk of cardiac injury than patients with the transient ischemic attack [9]. In another recent study, over 9% of TGA patients showed high sensitive troponin T levels, indicating myocardial damage. This could point to a pathophysiological link between TGA and TTS, bolstering the theory that TGA is linked to acute cardiac dysfunction, which leads to further brain injury. Thus, in individuals clinically presenting with TGA, a cerebral MRI with high-resolution DWI and a cardiovascular workup can help detect additional acute lesions that could be related to cardiac failure or myocardial injury. The retrospective nature of our investigation, as well as the single-center design, increases the possibility of selection bias. Because of contraindications to undergoing MRI or because they were released before 3 T MRI was available, not all patients with clinical TGA had 3 T MRI. In addition, cardiovascular and neuropsychological testing was not done systematically. The homogeneity of our cohort, with routinely acquired high-

resolution DWI in most clinically presenting patients with TGA over a long study period, is one of our strengths [10].

CONCLUSION

Acute ischemic lesions in patients who present with TGA are more common than previously considered. As a result, in patients with clinical TGA, a brain MRI should be conducted to look for ischemia lesions that could be associated with cardiac dysfunction or myocardial injury.

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