Case Report

Bilateral Hippocampus Changes on MRI in Transient Global Amnesia

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Abstract

Transient Global Amnesia (TGA) is a dynamic condition. An undefined event causes anterograde amnesia (sometimes with retrograde amnesia) that lasts in a day and preserves of self-identity and of consciousness. Epilepsy, migraine and ischemic events are the possible causes for different hypothesis but still unproven. Sixty three years old woman admitted to emergency department because of amnesia. She felt ill herself and was repeatedly asking similar questions and making same sentences in that period. She was not remember doing things nearby, but she was fully conscious to time, places, herself and her relatives. Neurological examination was normal except the anterograde and partly retrograde amnesia. Within amnesic period, Isotropic Diffusion Weighted Imaging (DWI) and ADC (Apparent Diffusion Coefficient) maps revealed hyper and hypo intensities on MRI respectively at both hippocampus (slightly more prominent at left side) while T1, T2 weighted and Fluid Attenuated Inversion recovery (FLAIR) images were normal. T1W+C images revealed bilateral contrast enhancements at hippocampus, and more prominent at the left side. Bilateral hippocampus MRI signal changes in our patient within the amnesic period are the reflections of the abnormal cellular functions in a TGA patient.

Keywords: Transient global amnesia, MRI, hippocampus

Geçici Global Amnezi MRG'de Bilateral Hipokampus Değişikliği

Özet


Anahtar Kelimeler: Geçici global amnezi, MRG, Hipokampus
INTRODUCTION

Transient Global Amnesia (TGA) is a dynamic condition. An undefined event causes anterograde amnesia (sometimes with retrograde amnesia) that lasts in a day and preserves of self-identity and of consciousness. Event must be witnessed and all other causes must be ruled out by appropriate methods\(^5\). The pathogenesis is still unclear. Epilepsy, migraine and ischemic events (ischemia or venous insufficiency) are the possible causes for different hypothesis but still unproven.

CASE PRESENTATION

Sixty three years old woman admitted to emergency department because of amnesia. She felt ill herself and was repeatedly asking similar questions and making same sentences in that period. Her daughter noticed that she was not remember doing things nearby, but she was fully conscious to time, places, herself and her relatives. Neurological examination was normal except the anterograde and partly retrograde amnesia. Within amnesic period, Isotropic Diffusion Weighted Imaging (DWI) and ADC (Apparent Diffusion Coefficient) maps revealed hyper and hypo intensities on MRI respectively at both hippocampus (slightly more prominent at left side) while T1, T2 weighted and Fluid Attenuated Inversion recovery (FLAIR) images were normal. T1W+C images revealed bilateral contrast enhancements at hippocampus, and more prominent at the left side. Abnormal signal changes in DWI were considered as restricted diffusion. Another extraaxial contrast enhanced region were also observed besides the petrous bone near the cerebellum at the right, that compatible with the calcified menengioma. Routine biochemistry, tumor markers, INR and complete blood count examinations were within normal limits except the increased LDH (310 U/l) hematocrit (46,5 %), and erythrocyte numbers (5.66X10⁶/microliter). In another blood chemistry examination revealed slightly increased cholesterol (242mg/dl) and low density lipoprotein levels (155mg/dl) and minor atherosclerotic plaques at the right proximal internal carotid artery (below 30 %) at duplex ultrasound. Amnesia was ended at 16 hour and she was also normal on repeated examination one week later.

Figure 1: MRI examination were performed within the amnesic period. Isotropic Diffusion Weighted and ADC maps revealed restricted diffusion on MRI at both hippocampus (slightly more prominent at the left side) (A - B). T1, T2 weighted and Fluid Attenuated Inversion Recovery (FLAIR) images were appear normal (C - D). T1W+C images revealed bilateral contrast enhancements at hippocampus, and more prominent at the left side. Another contrast enhancements were also observed besides the petrous bone near the cerebellum at the right, that compatible with the calcified meningioma that was coincidentally observed (E - F).
DISCUSSION
We observed a patient that fully compatible with criteria of TGA. Interesting point is the MRI findings that not similar to classical findings. In TGA, one sided punctate hyperintense lesions (1-3 mm) were seen in DWI images. Rates of hyperintense DWI appearance were changed from 11% to 84% Design of study, (prospective or retrospective), timing of MRI examination, repeated MRI examination, MRI sensitivity, and also reader decision may have an effect on this diversity. Increased signal intensity on DWI imaging were seen in even a minute ischemia but not specific to on it. In addition to TGA; epilepsy, stroke, limbic encephalitis shows high signal intensity on DWI imaging. Similar appearances in different diseases should be share similar cellular processes at least the same end point. Forster et al. proposed these processes that both local and global changes related with the decreased oxygen and glucose uptake of the cell, first disrupt the respiratory chain and then with increased anaerobic glycolysis accumulate the cellular lactate level. Lactate, increase the osmolality and results with the water influx. Basically hyper intense DWI appearance was the reflections of the accumulated water in the cell and cytotoxic oedema. Increased lactate peak in hippocampus that reflecting an acute metabolic stress in CA1 neurons were also revealed by MR spectroscopy in TGA.

Different lesions, such as bilateral hippocampal increased signal intensity on FLAIR images and diffusion restriction on diffusion - weighted MR images were also defined in TGA. In this case bilateral hypo perfusion in the mesial temporal lobes were observed on ⁹⁹mTc- hexamethylpropyleneamine oxime SPECT images. Records were taken at 48 hours of episode while the episode had already finished. In contrast, our case's MRI examination was performed within amnesic episode and we observed diffusion restriction, ADC signal changes and also contrast enhancements on both hippocampus. Different from the Di Flippo and Cabelesi FLAIR images were normal. This may be related with the performing time of the examination. These findings were not related with infections or tumours. Contrast enhancements were not an artefact because of similar contrast enhancements were also seen at coincidently finding menengioma near the right petrous bone near the cerebellum.

In TGA, blood tests were found normal except the increased lactate dehydrogenase (LDH) and aspartate aminotransferase (AST) serum levels with them decreased L-alanine and glutamine serum levels. Some blood test abnormalities were also seen in our patients similar to the previous cases like as slightly increased LDH level. Hematocrit and erythrocyte numbers, cholesterol and low density lipoprotein levels were also above the upper limits of normal. We thought that these values were not important for this condition. She had also no TIA or stroke before and also had no stroke risk factors.

CONCLUSION
We believe that bilateral hippocampus MRI signal changes in our patient within the amnesic period are the reflections of the abnormal cellular functions in a TGA patient. Although results are clear, details of this process are obscure.

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