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TWO PATIENTS WITH TRANSIENT GLOBAL AMNESIA IN WHICH REPEATED DIFFUSION-WEIGHTED IMAGING REVEALED HIPPOCAMPAL HYPERINTENSITY: CASE REPORTS

TEKRARLANAN DİFÜZYON AĞIRLIKLI GÖRÜNTÜLEMEDE HİPOKAMPAL HİPERİNTENSİTE SAPTANAN İKİ GEÇİCİ GLOBAL AMNEZİ OLGUSU: OLGU SUNUMLARI

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ABSTRACT

An episode of temporary memory loss which is recovered within 24 hours is called transient global amnesia. The etiology and triggering factors are unclear but hippocampal lesions may contribute to this condition. Diffusion-weighted magnetic resonance imaging is used to detect these lesions. In this case report, two cases with transient global amnesia who had hippocampal lesions are presented.

Keywords: Transient global amnesia, hippocampus, diffusion weighted magnetic resonance image

ÖZET

Geçici global amnezi, 24 saat içinde iyileşen geçici hafıza kaybı ataklarıdır. Etyolojisi ve tetikleyici faktörleri aydınlanmış değildir ancak hipokampal lezyonlar geçici global amneziye katkıda bulunabilir. Bu lezyonları saptamak için difüzyon ağırlıklı manyetik rezonans görüntüleme kullanılır. Bu olgu sunumunda hipokampal lezyonu olan geçici global amnezili iki olgu sunuldu.

Anahtar Kelimeler: Geçici global amnezi, hipokampus, difüzyon ağırlıklı manyetik rezonans görüntüleme

INTRODUCTION

Transient global amnesia (TGA) is a condition that is characterized by sudden onset anterograde amnesia including partially retrograde features and episodes of memory loss that resolve within 24 hours. It is not accompanied by other neurological deficits such as weakness or numbness. Cranial imaging is used to support the diagnosis of TGA. Diffusion-weighted magnetic resonance imaging (DW-MRI) can usually help to detect the lesions in the brain. In early stages of TGA, a DW-MRI may not generate any findings so the MRI may need to be repeated (1). We present two patients in which DW-MRI revealed hippocampal lesions because the detection of these lesions changes the treatment strategies.

CASE PRESENTATIONS

CASE 1

A 50-year-old female patient was admitted complaining of the inability to remember the last 3 hours. The patient was cooperative. She followed all instructions given by examiner, but she was disorientated. She could not remember how or when she came to the hospital. She was constantly asking where she was. Aside from these issues, neurological examination was normal. There

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was no limb weakness or numbness**.** She was on medical treatment for asthma and hypothyroidism and had a history of one abortion. There was no pathology on the brain computerized tomography (CT) and a DW-MRI was performed within 3 hours after symptom onset. The patient was hospitalized with TGA as preliminary diagnosis. She completely recovered 7 hours after symptom onset. Electroencephalogram (EEG) was normal. A second DW-MRI repeated 48 hours after symptom onset revealed diffusion restriction in both hippocampi. Hypointensities were detected on the apparent diffusion coefficient (ADC) map consistent with areas of the DW-MRI hyperintensities (Figure 1). The appearance of suspected signal increase in the right mesencephalon on the DW-MRI was interpreted in favor of artifact because although the second DW-MRI in Figure 1 was performed at 48 hours after the symptom onset, when cytotoxic edema is greatest during this time period in ischemic brain tissue, there was no marked hypointensity on the ADC map. The patient also had no symptoms associated with brainstem involvement. Acetylsalicylic acid (ASA) and low molecular weight heparin were added to treatment. Electrocardiogram (ECG) and echocardiogram (ECHO) for the etiology of ischemic stroke was normal. No abnormality was detected in 24-hour Holter monitoring. No significant stenosis was detected on head and neck CT angiography. In the blood test, the low-density lipoprotein cholesterol (LDL-C) level was 84.5 mg/dl. Atorvastatin was added to treatment. For detailed investigation of a young patient with cerebrovascular disease, anti-beta 2 glycoprotein 1 (anti-B2GPI) and anti-cardiolipin (ACA) IgMs that had been tested twice with an interval of 12 weeks were detected as high titration. Anti-B2GPI was positive with the values of 63.37 and 30.75 U/ml (normal laboratory value: <20 U/ml) and ACA IgM was positive with the values of 10.24 and 19 U/ml, in the first and second tests, respectively. No abnormality was found in other hemogram and biochemistry parameters. No mutation was detected in thrombophilia genetic panel (TGP). We continued with antiaggregant therapy until the autoimmune tests and TGP were completed. We planned treatment with warfarin for secondary prophylaxis against risk of embolism recurrence after positive antibodies were detected but the patient did not come to the 3rd month follow up. There was no new neurological symptom within the 3 months.

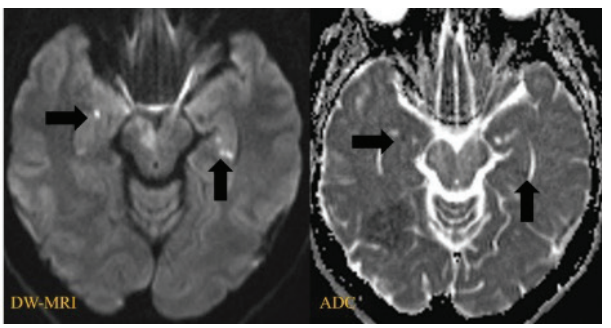

Figure 1: DW-MRI shows areas with restricted diffusion in both hippocampi (arrows). There are hypointensities in the corresponding areas on ADC (arrows)

CASE 2

A 56-year-old male patient was admitted with complaints of feeling unwell and not being able to remember the last few hours. When his relatives tried to tell him what had happened during the day, he could not remember. The patient was cooperative and had no lateralized motor impairment. Other neurological examination was normal. He did not have any history of chronic diseases. Brain CT and DW-MRI which were performed soon after symptom onset were normal. No abnormality was detected in EEG recording. In the left hippocampal gyrus, a DW-MRI repeated 12 hours after symptom onset showed millimetric area of restricted diffusion, with a corresponding low signal on ADC images (Figure 2). ASA was started. The patient completely recovered at 6 hours but did not recall this time period. In the evaluation of ECHO in terms of cardioembolism, mild mitral regurgitation was observed and no thrombus was detected. ECG was normal. No abnormality was detected in the 24-hour Holter monitoring. No stenosis was found on head and neck CT angiography. Atorvastatin was added to the treatment because the (LDL-C) level was 91 mg/dl. He had no new episodes at the 3rd month follow-up.

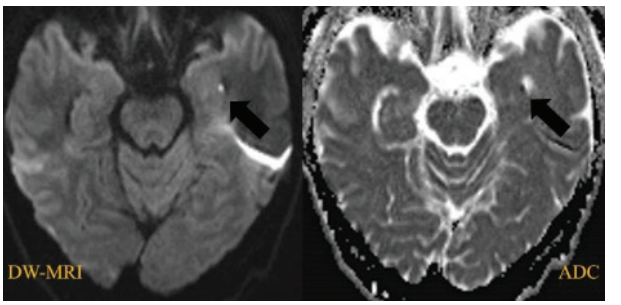

Figure 2: There is hippocampal restricted diffusion in the left hippocampus on DW-MRI and hypointensity is seen in the same area on the ADC map (arrows)

DISCUSSION

Transient global amnesia is a confusional clinical syndrome that resolves within 24 hours with anterograde amnesia and sometimes retrograde amnesia (2). Generally, patients tend to be disorientated and ask repetitive questions. The patient is awake during the attack and has no other neurological symptoms such as weakness or numbness. The patient can perform daily activities such as driving but is confused. Its incidence is higher in the 50-80 age range. Annual incidence of TGA is 3.4–

10.4/100,000 and in the over 50 population, this increases to 23.5/100.000 (4). Although clinical and imaging findings suggest hippocampal (especially cornu ammonis 1) dysfunction, the etiology of TGA is not clear. The diagnosis is made clinically according to the Hodges and Warlow criteria (3). It is suggested that a triggering situation including physical or psychological stress is usually detected before the disease onset, but the triggering factors are obscure. Migraine, focal ischemia, venous flow abnormalities and epileptic seizures have been suggested as factors that play a role in pathophysiology and differential diagnosis of TGA (5). A right hippocampal infarct was detected on DW-MRI in a case report with TGA (6) and a different study examining the retrospective data of 56 patients also suggested that TGA has a cerebrovascular background (7). It was stated in the same study that no relationship was found between migraine, epilepsy and TGA but another recent meta-analysis argued that there was a significant relationship between migraine and TGA (8). In our patients, epileptic amnesia was excluded because of the normal EEG recording and the absence of recurrent events in the follow-up. In our first patient with TGA, the hippocampal infarction was detected on the DW-MRI, and we detected APS in an investigation of etiologies of ischemic stroke. In 1998, the Sapporo criteria were established for APS (9). In 2006, the APS criteria were revised in the 11th International Antiphospholipid Symposium (10). Accordingly, while the clinical criteria remained the same, the laboratory criteria changed. Anti-B2GPI IgM and IgG were added to laboratory criteria. In our first patient, this diagnosis was considered due to the presence of a history of abortion, newly developed cerebrovascular ischemia, and positive test results for two different antiphospholipid antibodies (Anti-B2GPI and ACA IgMs). For secondary prophylaxis of APL, standard dose warfarin (INR: 2-3), low-dose ASA plus warfarin and high-intensity warfarin (INR: >3) have been recommended (11). We continued with antiaggregant therapy with ASA for the first patient until the autoimmune tests and TGP were concluded. We planned to start a standard dose of warfarin, but she did not come to her 3 months follow-up. We also investigated our second patient in terms of stroke etiology, but we could not find a cause and we continued his treatment with ASA. In one study, hippocampal hyperintensity was detected on the DW-MRI applied 24 and 36 hours later in 5 patients with TGA and it has been suggested that a DW-MRI performed at least 24 hours after symptom onset is important in supporting the diagnosis of TGA (1). In a meta-analysis, it was stated that the diagnostic yield of DW-MRI in TGA patients was 39% and a DWI performed between 24 and 96 hours after symptom onset showed higher diagnostic yield (12). In our patients, while the DW-MRI (3T MRI) within a short time (approximately 3 hours) after symptom onset was normal, a DW-MRI performed 48 hours later for the first patient and 12 hours later for the second patient showed hippocampal hyperintensity.

CONCLUSION

In this case report, two patients with TGA that had hippocampal hyperintensity on DW-MRI were presented. We think that repeating a DW-MRI in TGA cases increases the sensitivity for detecting lesions. Hippocampal lesions contribute to TGA. Detecting the lesion on the DW-MRI changes the treatment approach.

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REFERENCES

1. Abreu Junior L, Godoy LL, Vaz LPS, Torres AE, Wolosker AMB, Torres US, et al. Optimization of magnetic resonance imaging protocol for the diagnosis of transient global amnesia. Radiol Bras 2019;52(3):161-5. [CrossRef]
2. Sealy D, Tiller RJ, Johnson K. Transient Global Amnesia. Am Fam Physician 2022;105(1):50-4.
3. Arena JE, Rabinstein AA. Transient global amnesia. Mayo Clin Proc 2015;90:264-72. [CrossRef]
4. Hodges JR, Warlow CP. Syndromes of transient amnesia: towards a classification. A study of 153 cases. J Neurol Neurosurg Psychiatry 1990;53(10):834-43. [CrossRef]
5. Spiegel DR, Smith J, Wade RR, Cherukuru N, Ursani A, Dobruskina Y, et al. Transient global amnesia: current perspectives. Neuropsychiatr Dis Treat 2017;13:2691-703. [CrossRef]
6. Dölek T, Korkmaz M, Bek S, Kutlu G. Right Hippocampal Abnormality on diffusion-weighted MRI in transient global amnesia: case report. Turk J Neurol 2021;27:210-1. [CrossRef]
7. Waliszewska-Prosol M, Nowakowska-Kotas M, Bladowska J, Papier P, Budrewicz S, Pokryszko-Dragan A. Transient global amnesia - risk factors and putative background. Neurol India 2020;68(3):624-9. [CrossRef]
8. Liampas I, Siouras AS, Siokas V, Tsouris Z, Rikos D, Brotis A, et al. Migraine in transient global amnesia: a meta-analysis of observational studies. J Neurol 2022;269(1):184-96. [CrossRef]
9. Wilson WA, Gharavi AE, Koike T, Lockshin MD, Branch DW, Piette JC, et al. International consensus statement on preliminary classification criteria for definite antiphospholipid syndrome: report of an international workshop. Arthritis Rheum 1999;42(7):1309-11. [CrossRef]

10. Miyakis S, Lockshin MD, Atsumi T, Branch DW, Brey RL, Cervera R, et al. International consensus statement on an update of the classification criteria for definite antiphospholipid syndrome (APS). J Thromb Haemost 2006;4(2):295-306. [CrossRef]
11. Tektonidou MG, Andreoli L, Limper M, Amoura Z, Cervera R, Costedoat-Chalumeau N, et al. EULAR recommendations for the management of antiphospholipid syndrome in adults. Ann Rheum Dis 2019;78(10):1296-304. [CrossRef]
12. Lim SJ, Kim M, Suh CH, Kim SY, Shim WH, Kim SJ. Diagnostic yield of diffusion-weighted brain magnetic resonance imaging in patients with transient global amnesia: a systematic review and meta-analysis. Korean J Radiol 2021;22(10):1680-9. [CrossRef]