



Hyponatremia Mimicking Transient Global Amnesia *Geçici Global Amneziyi Taklit Eden Hiponatremi*

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Dear Editor,

Transient global amnesia (TGA) is a clinical syndrome characterized by temporary loss of anterograde memory, followed by retrograde amnesia, with preserved consciousness, attention and awareness. It usually lasts 1-8 hours and recovers without need for treatment. TGA usually occurs as a single attack and does not recur. The annual recurrence rate is reported to be 6-10%. Time and place orientation may be impaired and patients may repeat the same questions, leading to anxiety and agitation. However, consciousness is not affected and higher cortical activities such as driving and talking are preserved. TGA is often seen in patients aged over 50 years and is equally common in both sexes.

Although TGA was first described in 1956, no definite pathogenesis has been revealed. Ischemic, epileptic, metabolic, migrainous, and psychological factors are thought to be responsible. The affected brain regions are memory-related structures such as the thalamus, hippocampus (especially CA1 domain), splenium of the corpus callosum, fornix, and cingulate gyrus (1,2). In contrast, there are usually no etiologic factors in patients. Ischemia (vasospasm, venous congestion) or changes in neurotransmitter concentrations are usually listed among the causes of TGA. To the best of our knowledge, TGA accompanied by hyponatremia has never been identified in the literature.

A man aged 56 years was admitted to our neurology clinic with memory loss lasting 5-6 hours. He did not remember where he lived and how he had been brought to the hospital during this time. He was asking the same questions again and again. The patient was fully conscious, he could recognize his relatives and could fulfill instructions given by the physician. It was observed that he could solve mathematical problems and count backwards from 100 by 7 (93, 86...) in serial 7 examination. It was learned that there had had no such previous attacks, and that he only had arterial hypertension in his medical history; he was not on any antihypertensive drugs. The patient's wife said that the patient was on salt restriction and he drank about 6 liters of water that day. His blood pressure was normal and the neurologic examination was normal except for the amnesic situation. The other blood tests were within normal limits except that the serum sodium level, which was 120 mEq/L. No lesions were detected in computed tomography and diffusion-weighted magnetic resonance imaging (MRI) scans. Electroencephalography (EEG) of the patient was reported as normal. After sodium replacement, the patient returned to normal, recalling events that occurred before the attack, but he could not remember the 8-hour symptomatic period. The patient was diagnosed as having sodium electrolyte imbalance mimicking TGA.

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The etiology and pathogenesis of TGA and affected brain structures are still unclear. Based on the disappearance of symptoms after correction of hyponatremia in our case, we hypothesized that hyponatremia mimicked TGA symptoms. There was no other reason on EEG and MRI to explain the symptoms. Findings of ischemia in the temporal region can often be seen on MRI within 24 hours after a TGA attack. Nowadays, vascular mechanisms are suggested more in the pathogenesis of TGA. Ischemia in the fornix, cingulate gyrus, and hippocampus CA1 area, and reduced cerebral perfusion in the left precuneus, left inferior temporal and superior parietal areas have been shown in TGA (2). It was observed that patients with TGA experienced more depression and anxiety symptoms, gave a stronger stress response to cortisol suppression test, and that no changes were seen in neuropsychological tests; all these support hippocampal involvement (3). The presence of physical activity or Valsalva maneuver prior to amnesia in some patients with TGA has suggested the role of venous congestion in the pathogenesis, and evidence demonstrating inadequate jugular drainage supports this. In a patient with hypertensive TGA, MRI findings suggesting posterior reversible encephalopathy syndrome were detected. In our case, there was a history of hypertension, but the blood pressure was normal and there was no ischemic lesion in diffusion-weighted MRI.

Metabolic disorders such as hyperhomocysteinemia have also been associated with TGA. Animal studies have shown that hyponatremia causes hippocampal neurotransmitter changes, and thus cortically spreading depression and epilepsy (4,5). To our knowledge, our case is the first to be associated with hyponatremia. It is a known phenomenon that hyponatremia can cause brain edema, thus causing neurologic and psychiatric manifestations, and even death. Edema can lead to impaired function in cells by reducing the extracellular space and activating NMDA receptors and calcium channels (5). In our case, we think that hyponatremia due to polydipsia led to transient hippocampal dysfunction and that the symptoms improved with the correction of sodium levels.

Acute confusional state, complex partial seizures, transient epileptic amnesia, psychogenic amnesia, and transient ischemic attack are amongst the differential diagnosis of TGA. Vascular risk factors are often encountered in patients with transient ischemic attacks. Symptoms usually show lateralization and can recur. The differential diagnosis of acute confusional state, which can be

caused by metabolic conditions such as hypoglycemia, thiamine deficiency and intoxication, is made clinically. Unlike TGA, attention deficit is present in acute confusional state. Frequent repetition of questions is more frequently seen in TGA. There was no automatism or epileptiform discharge in our patient to suggest a complex partial seizure. Migraine was not considered, as there was no history of headache.

In conclusion, in order to accurately diagnose and rapidly treat TGA, it should not be forgotten that hyponatremia could mimic TGA. Correction of low sodium levels provides rapid improvement in symptoms.

Ethics

Informed Consent: Consent form was filled out by all participants.

Peer-review: Internally peer-reviewed.

Authorship Contributions

Medical Practice: Y.D., B.G., Concept: S.K., B.G., Design: S.K., B.G., Data Collection or Processing: Y.D., S.K., B.G., Analysis or Interpretation: S.K., B.G., Literature Search: S.K., B.G., Writing: S.K., B.G.

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