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Case Report / Olgu Sunumu

"If It Looks Like a Duck, then It Probably is a Duck!" No, Rarely It is not

"Eğer Ördek Gibi Görünüyorsa…Muhtemelen Bir Ördektir!" Ama Bazen de Değil

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Abstract

Hyponatremia is one of the major conditions that mimic stroke. Hyponatremia is known to cause focal neurological deficits; however, reported herein is a rare neurologic manifestation of hyponatremia, which mimics a proximal posterior cerebral artery occlusion. A 70-year-old man was admitted to our emergency unit with an acute history of vomiting, confusion, and impaired vision. Neurologic examination showed impaired orientation, left homonymous hemianopia, and left-sided hemispatial neglect. Hyponatremia was detected, and magnetic resonance imaging showed no abnormalities. The patient recovered completely after the correction of the serum sodium levels. This case illustrates that the clinical presentation of acute hemispatial neglect and homonymous hemianopia, which commonly implies a stroke, may result from hyponatremia. Hyponatremia should be considered in patients presenting with focal neurologic deficits, for which imaging evidence of a stroke cannot be shown.

Keywords: Homonymous hemianopia, hemispatial neglect, hyponatremia, stroke-mimics, case report

Öz

Hiponatremi inme taklit eden önemli nedenlerden biridir. Hiponatreminin fokal nörolojik defisitlere neden olduğu iyi bilinen bir durum olsa da burada hiponatreminin proksimal posterior serebral arter oklüzyonunu taklit eden nadir bir nörolojik tezahürünü sunuyoruz. Yetmiş yaşındaki erkek hasta akut başlançlı kusma, konfüzyon ve görme bozukluğu nedeniyle acil servisimize başvurdu. Nörolojik muayenesinde oryantasyonda bozulma, sol homonim hemianopsi ve sol yarı mekan ihmali saptandı. Acil değerlendirmesinde hiponatremi saptandı ve manyetik rezonans görüntülemede anormallik izlenmedi. Serum sodyum seviyesinin düzeltilmesinden sonra hasta tamamen düzeldi. Bu olgu yaygın olarak inme anlamına gelen akut yarı mekan ihmali ve homonim hemianopsinin hiponatremiden kaynaklanabileceğini göstermektedir. Fokal nörolojik defisit ile başvuran ve görüntülemesinde inme kanıtı gösterilemeyen hastalarda hiponatremi düşünülmelidir.

Anahtar Kelimeler: Homonim hemianopsi, yarı mekan ihmali, hiponatremi, inme taklitçileri, olgu raporu

Introduction

Stroke mimic (SM) is an important challenge in emergency departments, especially for the decision of thrombolysis treatment. Suspected patients with stroke that account for 5-30% are later diagnosed with SM (1). Seizures, toxic/metabolic causes, migraine, space-occupying lesions, and psychogenic disorders are common conditions that mimic stroke. Metabolic disorders, one-third of which is hyponatremia, comprise approximately 30% of all SMs. One large study, which included 113 SMs (13.4% of the total suspected stroke referrals), showed that the clinical presentations of SMs include (in the descending order) aphasia, posterior circulation symptoms, paresis, pure hypoesthesia, and others (2).

Severe hyponatremia (SH) (serum Na levels below 120 mmol/l) may cause hyponatremic encephalopathy (HE) that presents with lethargy, confusion, seizures, and coma, and may also cause focal neurologic deficits in 18% of HE (3,4).

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Herein, presented is a rare manifestation of hyponatremia likely due to the use of tacrolimus.

Case Report

A 70-year-old, right-handed male patient was brought to the emergency unit with a 24-hour history of nausea and vomiting, confusion, and impaired vision. His son reported that the patient has a medical history of liver transplant, hypertension, chronic renal failure, and glaucoma. He used tacrolimus at 1 mg/day, mycophenolate mofetil at 500 mg/day, prednisolone at 5 mg/day, ursodeoxycholic acid, and insulin aspart.

His blood pressure was within normal limits.

He was confused with a severely impaired temporal and spatial orientation. He had left homonymous hemianopia detected with confrontation visual field testing and left-sided somatosensory extinction, implying hemispatial neglect. Funduscopic examination was normal. Eye movements were normal with oculocephalic reflex testing. Sizes of pupils were normal with no ptosis. Muscle strength and gait were normal. He had a score of 6 on the National Institute of Health Stroke Scale.

Laboratory investigations included hemogram, biochemistry, thyroid function tests, chest X-ray, and electrocardiography. Serum sodium was 122 mmol/l, chloride was 90 mmol/l, blood urea nitrogen was 66.7 mg/dl, and creatinine was 2.07 mg/dl.

Stroke due to a right posterior cerebral artery (PCA) occlusion was considered the most likely diagnosis.

Computerized tomography (CT) of the brain was normal at the time of admission. Diffusion-weighted magnetic resonance imaging (MRI) showed no abnormal restricted diffusion. The only finding was the white matter hyperintensities that indicate small vessel disease with a Fazekas II severity (Figure 1).

A cerebral CT angiography was normal.

An IV saline solution infusion was started to treat hyponatremia with a moderate correction rate (<6-8 mEq/l correction in 24 hours), considering the risk of osmotic demyelination syndrome. After 24 hours, the patient's symptoms completely regressed. He was awake and oriented to time and space. Examination showed no visual field defects and no hemispatial neglect. His serum sodium was 131 mmol/l.

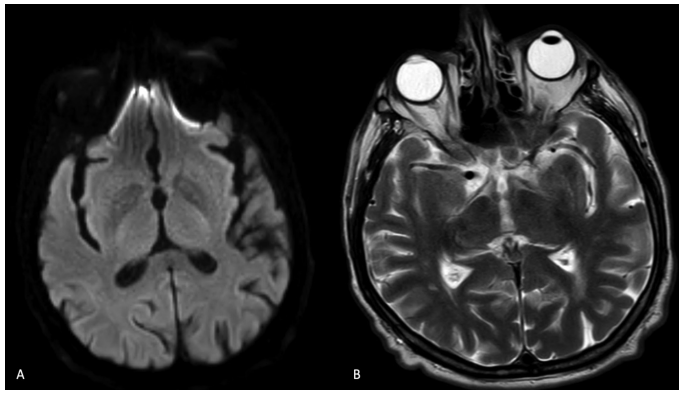

Figure 1. A) Diffusion-weighted magnetic resonance imaging of the patient showing no abnormal restricted diffusion. B) No abnormality has been seen that indicates posterior cerebral artery territory on T2 sequences

The rapid recovery of the patient hindered the objective documentation of neglect and visual field defect by planned examinations such as neuropsychological evaluation and perimetric visual field test.

The electroencephalogram recording to detect the evidence of seizure or encephalitis, which was performed on the second day, was normal. The confused state of the patient was already substantially recovered by then.

The patient was discharged on the second day with complete neurologic recovery.

He was seen in the outpatient clinic on day 4. He had no symptoms or signs. Serum sodium level was 135 mmol/l.

A final diagnosis of drug-induced HE was made.

Discussion

SH was defined as a serum sodium level of <121 mmol/l in a relatively large prospective study (n=92) by Daggett et al. (3) as early as 1982. This study has listed focal neurologic signs, including pyramidal, extrapyramidal, cerebellar, and ocular motor signs. As a specific cognitive syndrome, aphasia is the most common presentation of hyponatremic SM (37.6%) in Brunser et al.'s (2) study. Left homonymous hemianopia and hemispatial neglect may co-occur in proximal PCA occlusions due to the simultaneous infarctions in the right occipital lobe and the right posterior thalamus (pulvinar nucleus) (5).

Aulakh et al. (6) reported a patient who presented with left homonymous hemianopia with MRI findings of posterior reversible encephalopathy syndrome due to hyponatremia. Wareing et al. (1) reported a patient with HE who had right homonymous hemianopia accompanying aphasia and right faciobrachial weakness. Latifi et al. (7) reported a patient who presented with left-sided faciobrachial weakness and left-sided neglect due to SH. All these three cases imply a middle cerebral artery occlusion SM. To the best of our knowledge, a right proximal PCA occlusion SM due to SH was not previously reported. "Symptoms that mimic the posterior circulation" is listed as the second most frequent SM in Brunser et al.'s (2) study; however, they alluded to the signs and symptoms of brainstem dysfunction, as no symptom can be attributed to PCA were explicitly noted.

Hyponatremia causes neurologic dysfunction via intracerebral osmotic fluid shifts and brain edema. Acute changes in sodium cause neurologic dysfunction; thus, chronic hyponatremia may be asymptomatic due to the brain's adaptation (7).

Renal impairment, pneumonia, inappropriate antidiuretic hormone secretion, and fluid overload were listed as the most common causes of hyponatremia, followed by drug-induced cases by Daggett et al. (3). The medical history of our patient was not suggestive of any of the first four conditions. Tacrolimus is an immunosuppressive drug that is commonly used after organ transplants, which may cause hyponatremia (8). The patient completely recovered with sodium replacement, without discontinuation of the drug. However, this drug is still considered to induce hyponatremia.

In conclusion, this case exemplifies an SM syndrome due to hyponatremia with the clinical presentation of hemispatial neglect and homonymous hemianopia. Total recovery with the correction of serum sodium levels supports our diagnosis. As an important cause of SMs, hyponatremia should be considered in patients

with focal neurological deficits without imaging evidence of an acute stroke. The syndrome is entirely curable if care is taken not to hasten for correction and if the possibility of life-threatening osmotic demyelination syndrome is avoided. Colleagues in the emergency department should be aware of SMs as they have a very favorable prognosis.

Ethics

Informed Consent: Written consent was obtained from the patient.

Peer-review: Externally and internally peer-reviewed.

Authorship Contributions

Surgical and Medical Practices: Z.Y., E.D., Concept: Z.Y., Design: Z.Y., Data Collection or Processing: Z.Y., E.D., Analysis or Interpretation: Z.Y., E.D., N.K., Literature Search: Z.Y., E.D., Writing: Z.Y., E.D., N.K.

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