

Case Report

Hypernatremia secondary to post-stroke hypodipsia: just add water!

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Abstract

Disorders in water metabolism may occur in stroke patients. When hypernatremia arises in this setting, it is usually secondary to the development of central diabetes insipidus or it is the result of neurologic lesions that prevent patients from having free access to water. Much rarer are the cases of post-stroke hypernatremia caused by hypodipsia secondary to lesions of the thirst center. We report the case of a patient with severe hypernatremia, probably secondary to post-hemorrhagic stroke hypodipsia. The hypernatremia seen in this case was corrected by scheduling the patient's water intake.

Keywords: hypernatremia; hypodipsia; stroke; water

Introduction

Water disturbance may occur in hemorrhagic and ischemic stroke patients because of the major role that the central nervous system plays in water homeostasis [1–3]. Post-stroke hypernatremia is generally the consequence of central diabetes insipidus, but it can also take place if the patients do not have free water access, usually as the result of neurological sequelae which prevents them from drinking water or even asking for it from their relatives and caretakers. Hypodipsia secondary to lesions of the thirst center, not accompanied by other serious neurological damage, is a rare cause of post-stroke hypernatremia, but it may cause significant morbidity if not properly diagnosed and managed.

Case report

A 56-year-old Caucasian man was admitted to the cardiology ward of our hospital due to lethargy and muscle weakness, attributed to the presence of bradycardia. Routine admission laboratory tests revealed high plasma sodium levels (Na^+ : 157 mEq/L), which motivated, on the following day, a request for nephrology evaluation. At nephrology consultation, the patient was found to be in apparent good health. He was conscious, without obvious motor deficits. On physical examination, his blood pressure was

140/70 mmHg and his heart rate was 54 b.p.m. There were no clinical signs of decreased extracellular volume. The patient was not taking any kind of medication. His wife reported that he had been losing weight in the last years. His past medical history revealed that 9 years previously he had had a hemorrhagic stroke and needed brain surgery to clamp a ruptured aneurysm of the anterior communicating artery and to drain a cerebral hemorrhage. A representative figure of patient's cranial computed tomography (CT) done at that occasion is presented in Figure 1. The stroke resulted in some impairment of the capacity of space location, moderate reduction of the recent and fixation memory and a certain aversion to water. There was no history of polyuria. New laboratory tests were ordered and confirmed hypernatremia: plasma sodium of 155 mEq/L and plasma chloride of 116 mEq/L. Urinalysis: urine-specific gravity of 1026, no blood or protein and a normal sediment. Plasma potassium, creatinine and urea were, respectively, 4 mEq/L, 1.07 mg/dL and 41 mg/dL. In the 24 h that followed nephrology consultation, the patient passed only 400 mL of urine. The diagnosis of possible hypernatremia secondary to post-stroke hypodipsia was then made and supervised water intake of ~2 L a day was initiated. After this simple measure, there was an increase in diuresis and plasma sodium was reduced to 150 mEq/L on the third day and to 144 mEq/L on the fourth day after admission. Two weeks after hospital discharge, he had gained 3 kg and referred marked improvement in lethargy and muscle weakness. His plasma sodium level was normal. The evolution of the patient's laboratory tests, including the ones collected 2 weeks after hospital discharge, are presented in Table 1.

Discussion

Hypernatremia is relatively frequent in clinical practice, being common in the elderly and in critically ill patients [4]. In patients with free access to water, hypernatremia is exceptional since intact thirst mechanism is a powerful defense against hyperosmolality. When plasma sodium (the main determinant of plasma osmolality) increases, two defense mechanisms are triggered: stimulation of the thirst center and secretion of antidiuretic hormone (ADH).

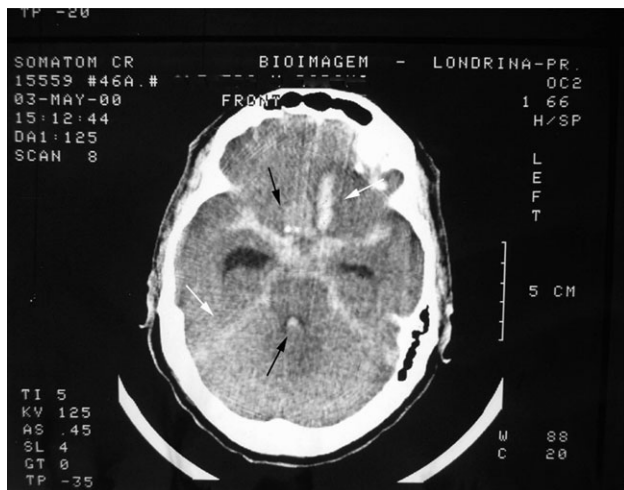


Fig. 1. Cranial CT scan showing diffuse subarachnoid hemorrhage (inferior white arrow), frontal left intraparenchymatous hematoma (superior white arrow), intraventricular hemorrhage (inferior black arrow) and nodular slightly hyperdense image with peripheral calcifications on anterior communicating artery topography, suggestive of giant aneurysm (superior black arrow).

Table 1. Evolution of plasma sodium and other laboratory tests after patient’s hospital admission

	Admission (D0)	D1	D2	D3	D17
Sodium (mEq/L)	157	155	150	144	139
Urine-specific gravity		1.026			1.011
Chloride (mEq/L)		116			102
Potassium (mEq/L)		4.0			4.2
Creatinine (mg/dL)		1.07			0.7
Urea (mg/dL)		41			21

This combination results in increased water intake and reduction in urinary water loss, leading, thus, to normalization of plasma osmolality. This mechanism is highly efficient, keeping plasma osmolality in a narrow range, despite the daily variation of water and sodium intake [5].

In the reported case, the patient’s mechanism of ADH liberation and the urinary concentration were preserved: when faced with significant hypernatremia, the patient’s 24-h urine volume was only 400 mL, and the urine had high specific gravity (1.026); normalization of plasma sodium concentration, urine dilution and weight gain occurred when supervised water intake was warranted. This case illustrates the fact that even when the mechanisms of ADH liberation and urine concentration are intact, and if the thirst mechanism is damaged, the body is not capable of maintaining normonatremia [3, 6, 7].

Post-stroke hypodipsia may be the result of damage to the hypothalamic thirst center, believed to be present in the

lamina terminalis of the third ventricle [8]—in the presented case, there was extensive intraventricular hemorrhage—but this may occur even in the absence of CT evidence of hypothalamic damage, possibly due to a cortical central nervous system lesion that interferes with cortical perception of thirst [9].

The clinical symptoms that motivated the patient’s hospital admission and that were at the first attributed to bradycardia were apparently caused by hypernatremia. In fact, this patient had been presenting muscle weakness and lethargy very frequently in his daily life and his wife reported that these symptoms had begun after the stroke. Indeed, the review of his past laboratory test values revealed that in the course of medical evaluations done in the past, high values of plasma sodium were documented (170 mEq/L in September 2007 and 151 mEq/L in February 2009) but were misinterpreted as laboratory artifacts since the patient presented no physical sequels of the hemorrhagic stroke and, as hypernatremia was chronic, significant neurological symptoms were absent. Sadly, a second measurement of plasma sodium was not ordered on neither of those two occasions. The patient’s weight loss was secondary to true dehydration since it was quickly corrected after appropriate water intake. No medical reason was found for the patient’s bradycardia, but when discharged from hospital, his heart rate was 72 b.p.m.

Conflict of interest statement. None declared.

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