

Extremely Severe Hyponatremia Caused by Wrong Belief in a Patient with Cervical Cancer

Myeong Su Park, Hyuk Jin Park, Hong Sang Choi, Chang Seong Kim, Eun Hui Bae, Seong Kwon Ma, Soo Wan Kim, Minah Kim

Department of Internal Medicine, Chonnam National University Hospital, Chonnam National University Medical School, Gwangju, Korea

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Corresponding Author: Minah Kim, MD, PhD
Department of Internal Medicine, Chonnam National University Hospital, Chonnam National University Medical School, 42 Jebongro, Gwangju 61469, Korea

Tel: +82-62-220-6296

Fax: +82-62-225-8578

E-mail: karbiya@naver.com

A 56-year old female patient who was undergoing follow-up for cervical cancer in our oncology center was presented to the emergency center with anxiety and excessive thirst. The initial serum sodium level of the patient exceeded 200 mEq/L, rising up to 238 mEq/L during hospitalization. The extremely severe hyponatremia was caused by patient's wrong belief that bay salt would cure the cancer. The patient was treated with hypotonic solution and finally with appropriate hydration, she was fully recovered without any neurological complications.

Key Words: Dehydration, Hyponatremia, Malignancy

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Introduction

Hyponatremia, defined as a serum sodium concentration exceeding 145 mEq/L, is a frequently observed electrolyte abnormality. Especially, it is more vulnerable in infants, the elderly, patients with underlying neurologic disease or critical illness^{1,2)}. The hyperosmolar state associated with hyponatremia adversely affects various cellular functions, leading to neurologic, metabolic, and cardiac dysfunctions³⁻⁶⁾ and ultimately high morbidity and mortality^{1,7,8)}. Thus, proper treatment of hyponatremia is important for the prognosis of hyponatremic patients.

Here, we report a case of a patient with the highest serum sodium concentration reported in the literature. This case describes our successful experience in management of an extremely severe hyponatremic patient combined with water loss caused by excessive salt intake.

Case Report

A 57-year-old female was presented to our emergency

center with severe anxiety and general weakness. She was undergoing regular follow-up after concomitant chemoradiation therapy (CCRT) for cervical cancer 3 months ago. Also she was taking an amlodipine as an antihypertension drug. The patient usually consumed bay salt with her wrong belief that eating salt would be helpful to cure cancer. Two days before the hospital visit, her family member got to know that the patient had been taking 4 teaspoons of bay salt a day. In addition, the patient's oral intake had been markedly decreased, and had intermittent vomiting several days before the visit.

On arrival on our hospital, the patient showed dehydrated features such as dry tongue and reduced skin turgor. Blood pressure was 210/100 mmHg and heart rate was 74 beats/min in the supine position. Initial laboratory tests showed the following values: serum sodium 203 mEq/L [135-145], potassium 2.9 mEq/L [3.5-5.0], chloride 167 mEq/L [96-108], bicarbonate 26.7 mEq/L [21-28], inorganic phosphorus 1.1 mg/dL [2.5-5.5], blood urea nitrogen (BUN) 42.5 mg/dL [8-23], creatinine 2.21 mg/dL [0.5-1.3], serum osmolality 415 mOsm/kg [280-295], urine osmolality 369 mOsm/kg [300-900], urine sodium 158 mEq/L [40-220]. Owing to

the patient's inappropriately high BP and peripheral cyanosis, we checked transthoracic 2D-echocardiography. Echocardiography revealed markedly decreased the left ventricular (LV) chamber size and resultant concentric remodeling, which are caused by depletion of circulating volume. Kidney ultrasonography showed enlarged kidney (both kidney size about 12 cm) with increased corticomedullary differentiation, which was compatible findings with acute kidney injury. Brain CT showed no significant abnormal findings such as cerebral infarction, hemorrhage, hydrocephalus and infection.

She was transferred to the medical intensive care unit. For the proper hydration, we calculated free water deficit: estimated free water deficit [free water deficit = total body water \times ((serum sodium/140) - 1)] was calculated as 12 liters. Hydration was initiated with hypotonic fluid (5% dextrose and 0.45% sodium chloride). During initial aggressive hydration serum sodium level was even more elevated to 238 mEq/L 2 hours later, therefore we increased the replacement rate of water deficit. After that, however, serum sodium was unintendedly corrected at rates above 1 mEq/L per hour (203 mEq/L to 177 mEq/L for 24 hours) and after 72 hours, the serum sodium concentration was nearly corrected to the normal level (less than 155 mEq/L) (Fig. 1). Serum creatinine level also decreased to normal (0.8 mg/dL) at the fourth day of hospitalization. Despite the high correction rate of hyponatremia, but the patient was completely recovered without any significant neurologic complications, and was discharged to home. In an outpatient

clinic conducted after discharge, the patient's serum sodium level was well maintained around 140 mEq/L. And she did not show any neurological signs including headache in at least six months of outpatient care.

Discussion

Hyponatremia can develop by two pathologic mechanisms: net water loss or excessive salt intake. And the present case shows that extremely severe hyponatremia can develop when water loss and excessive salt intake occur simultaneously. In general, dehydration-induced hyponatremia caused by lack of water intake or vomiting accompanies decreased urine sodium concentration (less than 10 mEq/L)⁹. In the present case, however, the urine sodium concentration (158 mEq/L) was significantly increased even though the patient was dehydrated. This may be inferred from excessive salt intake based on her history and laboratory test results. In addition, the loss of urine concentrating capacity (urine osmolality 369 mOsm/kg) due to intrinsic acute kidney injury is thought to have contributed to the further exacerbation of hyponatremia¹⁰.

Before treating patients with hyponatremia, whether hyponatremia is acute or chronic should be assessed. In acute hyponatremia, which usually occurs within 48 hours, rapid correction (1-2 mEq/L per hour) of hyponatremia is warranted. The reason why it is possible is that the compensatory mechanism for replacing organic solute in brain cells has not begun yet¹¹. On the other hand, in chronic hyponatremia, the compensatory mechanism has already begun. In such circumstances, the rapid correction of hyponatremia could lead to brain edema, which can ultimately cause seizure, brain herniation and death. Therefore, in chronic hyponatremia, the principle of treatment is to correct less than 0.5 mEq/L per hour and less than 8-12 mEq/L per day¹². Also it is well known that the prognosis of patients with hyponatremia is poor if the correction time of hyponatremia is long or hyponatremia persists⁸.

We did not know how long the patient was exposed to hyponatremia, so we tried to correct the serum sodium concentration slowly, but it was quickly corrected at the rate of more than 1 mEq/L per hour. Nonetheless, the patient recovered without any neurological complications, and

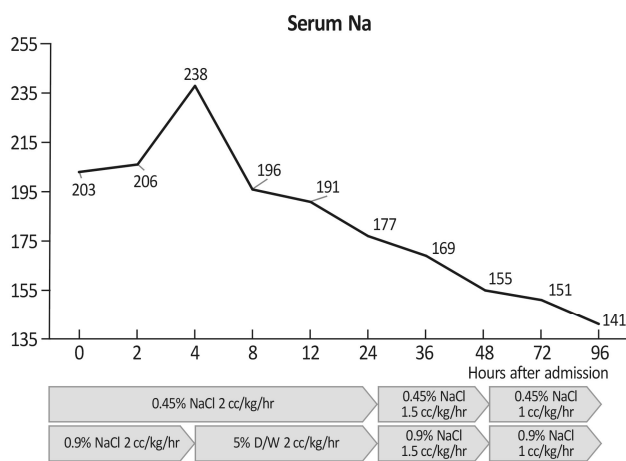


Fig. 1. Changes in the patient's serum sodium concentration.

we could assume two reasons for this outcome. First, given that patient's neurological symptoms (severe anxiety) suddenly occurred within hours of her visit to the hospital, it can be inferred that the patient developed acute hyponatremia, which can be a reason why the patient had no significant neurological complications despite rapid correction of serum sodium. Second, the generally accepted treatment principle of hyponatremia may not be applicable to adults. In a recent study of more than 100 adult patients, although hyponatremia was corrected at a rate of 0.5 mEq/L per hour or more (0.6-1.4 mEq/L per hour), no neurological complication such as seizure or brain edema occurred regardless of chronicity of the onset¹³. This study suggests that the reason why significant neurological complications do not occur even though hyponatremia is rapidly corrected in adult patients may be due to differences in brain capacity between adults and children. In general, it is known that the ratio of brain volume to cranial vault size increases most at 6 years of age, and brain volume gradually decrease after 45 years of age¹⁴. Therefore, it can be thought that the adaptability to the increase in brain volume is better in adult than in children.

In conclusion, a combination of water loss and excessive salt intake can cause very severe hyponatremia. Prompt diagnosis and appropriate management of hyponatremia can increase the rate of survival in extremely severe hyponatremia. To the best of our knowledge, this is the severest case of hyponatremia in adult reported in the literature. Cancer patients can behave on several wrong beliefs due to their desperation. So physicians should pay special attention to their changes.

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