Case Report

Hyponatremia and Acute Kidney Injury as a Consequence of Malnutrition: A Case Report

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Keywords

Hyponatremia · Acute kidney injury · Malnutrition · Nephrology

Abstract

This case presents how malnutrition due to underlying psychiatric disease can cause severe, chronic hyponatremia and acute kidney injury. A 31-year-old man was admitted due to fatigue. Blood tests displayed hyponatremia of 101 mmol/L and acute kidney injury. The patient had restricted himself to a uniform diet mainly consisting of rice boiled without salt. Isotone and hypertonic sodium chloride were used to secure a controlled rise in the sodium level. Despite fluid therapy, a delayed response in improvement in renal function was seen. After discharge, the patient started a balanced diet and the sodium level was almost normalized. Renal function eventually recovered. Long-term malnutrition may affect the tubular function of the kidney. Severe hyponatremia, other electrolyte disturbances, and protein and vitamin deficiency can be factors that interact in this pathogenesis. Resuming a normal diet may allow the kidney's function to return to normal despite malnutrition during months.

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Background

Malnutrition is known to affect the function of several organs and can lead to increased morbidity and mortality [1]. This case presents how extreme dietary restriction can cause severe somatic manifestations, more specifically hyponatremia and acute kidney injury (AKI). Hyponatremia is the most common electrolyte derangement in hospitalized patients and is associated with an increased risk of mortality [2, 3]. Rapid development of severe hyponatremia within 48 h may cause cerebral edema as the brain cells do not have time to adapt to the hypotonic extracellular environment, resulting in symptoms ranging from nausea, confusion, and seizures to coma [4]. In gradually developed hyponatremia, the symptoms are less dramatic as the neurons can adapt to the extracellular environment. These symptoms include nausea, vomiting, fatigue, and headache as well as hallucinations and confusion. In patients with chronic hyponatremia, a controlled rise in the sodium level is vital to prevent osmotic demyelination [5]. Hyponatremia has several underlying causes, with syndrome of inappropriate antidiuretic hormone secretion (SIADH), use of diuretics, polydipsia, hypovolemia, heart failure, and liver cirrhosis as the most common ones [3].

Case Presentation

A 31-year-old man was admitted due to fatigue, constipation, and vomiting for 4 days. The patient had previously been admitted to the hospital and seen in an outpatient clinic because of gastrointestinal symptoms, more specifically nausea, regurgitation, abdominal pain, and diarrhea. Despite a thorough examination, no somatic cause was ever found. During the 24 h prior to admission, the patient had not taken any fluid or food and was anuric. He could not provide any information on previous weight loss. The patient appeared malnourished, had a body mass index of 18, and was clinically suspected to be dehydrated, presenting dry mucosa of the mouth, dry, scaled skin, and reduced skin turgor. Examination of the abdomen showed diffuse tenderness, but auscultation of the heart and lungs was normal. Blood pressure was 108/81 mm Hg and the heart rate was 65 bpm. The patient received no medication or dietary supplements.

The blood tests at the time of admission displayed severe hyponatremia, low plasma albumin, and AKI accompanied by metabolic acidosis and hyperkalemia (Table 1). Furthermore, they showed severe vitamin D deficiency, low levels of plasma zinc, and high levels of vitamin B12. The urine showed no proteinuria, low urine osmolality, and low sodium excretion (<20 mmol/L). The low sodium level in the urine and low urine osmolality minimized the probability that the hyponatremia was caused by SIADH and increased the probability of it being hypovolemic.

An ultrasound examination of the kidneys and lower urinary tracts was made to rule out postrenal AKI and was found to be normal. Acute adrenal insufficiency was considered due to the diffuse symptoms with nausea, abdominal pain, and biochemical results with hyponatremia and hyperkalemia. The patient had, however, not received any glucocorticoid

treatment before admission and blood pressure was close to normal; thus, this differential diagnosis was rejected.

The patient claimed to be allergic to numerous types of foods, but according to the medical chart, no food allergy was confirmed in previous medical examinations. Due to the alleged food allergies, the patient had gradually restricted himself to an extremely uniform diet which for the previous 6 months had consisted solely of rice boiled in unsalted water and every 5 days a small amount of meat.

The initial treatment consisted of a slow inlet of intravenous sodium chloride (NaCl) 50 mL/h to secure a controlled rise in the sodium level, as the hyponatremia was interpreted as chronic due to the duration of symptoms and eating disorder. The patient was monitored for development of refeeding syndrome, but blood tests did not indicate this, as the patient had hyperphosphatemia and -kalemia, hypocalcemia and -natremia, and a normal level of magnesium.

During admittance, the patient seemed to realize the severe consequences of a uniform diet and gradually agreed to a more varied diet, increased calorie intake, prescription of a vitamin D supplement due to the severe vitamin D deficiency, and sodium bicarbonate due to metabolic acidosis.

Inlet of intravenous NaCl was gradually enhanced to 150 mL/h under close monitoring of the elevation of the sodium level. The high inlet of NaCl resulted in overhydration with a weight gain of 6 kg in two days, consequently a fluid restriction of 1,200 mL per day were prescribed (Table 1). Sodium levels had elevated from 101 mmol/L at admittance to 130 mmol/L at discharge 10 days later.

After hospitalization, the patient was seen in the nephrological outpatient clinic for follow-up. The patient started a balanced diet, the sodium level had almost normalized, and creatinine improved towards normalization. At discharge, the patient was instructed to keep the fluid restriction of 1,200 mL per day. Unfortunately, the overhydration worsened after discharge and resulted in severe peripheral edemas. The edemas were treated with diuretics and disappeared 10 days later, by which time renal function had recovered completely, as assessed by the creatinine level. A psychiatrist suspected that the strict diet was a result of anorexia nervosa (AN) induced by a crisis or stress reaction to a breakup 4 years earlier. The patient denied misuse of diuretics or laxatives. During admission, he agreed to be referred to the psychiatric department for follow-up but cancelled after the first appointment.

Discussion

A uniform diet due to psychiatric disease can lead to severe somatic disease, as shown in this case. At admission, the patient was malnourished and dehydrated due to the remarkably uniform diet with minimal salt intake. Severe dietary restrictions in conditions such as AN can lead to hyponatremia through several pathophysiological mechanisms, e.g., due to an SIADH response, hypervolemic hyponatremia as a result of polydipsia, and hypovolemic hyponatremia caused by malnutrition [6]. The anamnesis and initial clinical observations suggested extreme sodium deficiency and chronic hypovolemic hyponatremia due to

malnutrition and volume depletion. The biochemical urine tests suggested hypovolemic hyponatremia. Following treatment initially with isotonic (0.9%) and later also with hypertonic NaCl (3%), the hyponatremia was corrected.

Initially, the AKI was suspected to be entirely prerenal and caused by dehydration. However, the close-to-normal blood pressure, the delayed response in improvement in renal function despite fluid therapy, and the severe edemas present after admission may suggest an intrarenal problem as well. A uniform diet can cause prerenal AKI due to volume depletion, but acute or chronic tubular damage due to electrolyte deficiency, e.g., hypokalemic nephropathy, is also seen in patients with AN [7, 8]. Our patient had hyperkalemia, hyperphosphatemia, and magnesium within the normal range, which is consistent with renal failure but differs from what could be expected in a patient with complete AN [8]. Electrolyte disturbances such as hyponatremia and protein deficiency caused by restrictive dieting may have an effect on the renal tubules and thereby lead to an intrarenal impact on the kidney. Studies have shown that hyponatremia constitutes an independent risk factor for AKI, but it is unknown whether severe hyponatremia can affect the tubular system of the kidney independently [9, 10].

It is plausible that an interaction between the observed electrolyte derangements and vitamin, protein and nutrient insufficiency could have led to tubular damage and subsequent AKI. This pathogenesis may differ from what is seen in hypokalemic nephropathy. This is supported by the observation that renal function and the sodium level were normalized, which could indicate that a deficiency of nutritional components, including hyponatremia, may cause reversible tubular renal failure.

Conclusions

Malnutrition can lead to hyponatremia and prerenal AKI. However, long-term malnutrition may also affect the tubular function of the kidney. Severe hyponatremia, other electrolyte disturbances, and protein and vitamin deficiency can be factors that interact in this pathogenesis. Resuming a normal diet may allow kidney function to return to normal despite malnutrition during months.

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Statement of Ethics

The patient gave his consent for the publication of this case report.

Conflict of Interest Statement

The authors declare that they have no competing interests.

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Author Contributions

F.H. Mose was involved in the treatment of the patient. A.J. Nielsen drafted the manuscript and analyzed the available literature data. Both authors read and approved the final manuscript.

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Table 1. Development of selected biochemistry parameters and the patient's bodyweight

Day	1	2	4	6	8	10	13	15	24	34
P-K ⁺ (3.5–4.6 mmol/L)	5.6	5.2	4.0	3.9	3.9	4.0	4.3	4.0	3.7	3.5
P-Na ⁺ (135–145 mmol/L)	101	102	113	122	127	130	143	139	140	141
P-Ca ²⁺ (2.20–2.55 mmol/L)	2.15	2.03	1.91	1.86	1.90	1.93	2.13			2.20
P-albumin (36–48 g/L)	37	32	27	24	24	25	31	30		40
P-creatinine (60–105 μ mol/L)	174	200	166	137	129	173	105	84	85	84
P(vb)-CO ₂ -total (25–32 mmol/L)	18	16	16	15	15	14		21		
25-OH-vitamin D (50–160 nmol/L)	11						22			64
P-zinc (10–19 μ mol/L)		8								
P-vitamin B12 (200–600 pmol/L)	639						409			
P-folic acid (>6 nmol/L)							8			
P-ALT (10–70 U/L)	21						155		88	89
P-bilirubin (5–25 μ mol/L)	43						5			8
P-ALP (35–105 U/L)	39						58		74	
P-glucose (4.2–7.8 mmol/L)	5.8									
B-hemoglobin (8.3–10.5 mmol/L)	8.3	8.1					4.9	4.4	4.8	6.4
B-leukocytes (3.5–10.0 $\times 10^9$ /L)	11.7	9.5					5.4		8.8	6.3
Bodyweight, kg	61			69.1	75.2			84.1		

P, plasma; K⁺, potassium; Na⁺, sodium; Ca²⁺, calcium; Alb, albumin; vb, venous blood; ALT, alanine aminotransferase; ALP, alkaline phosphatase; B, blood.