CEN Case Reports
https://doi.org/10.1007/s13730-022-00733-y

CASE REPORT

Severe hyponatremia due to surreptitious water intoxication in a hospitalized patient

Sophie E. Claudel[1] · **Sushrut S. Waikar**[2] · **Ashish Verma**[2]

Received: 5 July 2022 / Accepted: 31 August 2022

Abstract
Hyponatremia is a common electrolyte abnormality among hospitalized patients and often present as first sign of other underlying medical conditions. Severe hyponatremia can be life threatening and requires prompt diagnosis and treatment. We present a case of refractory hyponatremia that was a diagnostic challenge requiring a prolonged hospitalization. Diagnosis of primary polydipsia was ultimately suspected due to improbable 24-h urine studies and confirmed through complete removal of free water access in the form of disconnecting the bathroom faucet in the patient's hospital room. Diagnosis and management of primary polydipsia is further discussed.

Keywords Hyponatremia · Primary polydipsia · Water restriction

Introduction

Hyponatremia is a common electrolyte among hospitalized patients that poses diagnostic and therapeutic challenges. A recent retrospective study associated persistent hyponatremia with increased length of stay, readmissions, and mortality [1]. Accurate diagnosis of the etiology of hyponatremia is dependent on an understanding of water homeostasis [2]. The diagnosis is established by first assessing serum osmolality to confirm or refute hypotonicity and then using urine osmolality and urine sodium to differentiate antidiuretic hormone (ADH)-dependent versus ADH-independent hyponatremia [3]. Non-hypotonic hyponatremia can also be caused by pseudohyponatremia, a laboratory artifact due to elevated concentration of triglycerides, protein, and cholesterol [4]. In hospitalized patients, hyponatremia is often multifactorial with multiple potential causes—disentangling them becomes a challenge. Here we present a case of hyponatremia secondary to primary polydipsia in the absence of known psychiatric comorbidity. Informed consent was obtained from the individual described in the case below.

✉ Sophie E. Claudel
Sophie.claudel@bmc.org

[1] Department of Internal Medicine, Boston Medical Center, 72 E. Concord St, Office Evans 112, Boston, MA 02118, USA

[2] Section of Nephrology, Department of Internal Medicine, Boston Medical Center, Boston, MA, USA

Case report

A 38-year-old man with a reported (unconfirmed) history of Gitelman's syndrome, hypertension, and diabetes presented to Urgent Care with leg pain, dizziness, and headache and was found to have COVID-19 and a serum sodium concentration ([SNa]) of 114 mmol/L. He was transferred to the Emergency Department, where repeat labs 4 h later demonstrated [SNa] 121 mmol/L, serum osmolality of 248mOsm/kg H_2O, urine osmolality of 61mOsm/kg H_2O, and urine sodium < 20 mmol/l (Tables 1, 2). He received 1L of normal saline for a blood pressure of 93/63 mmHg and the [SNa] rapidly increased to 127 mmol/l thereafter. This prompted infusion of dextrose-containing water upon hospital admission, totaling 2.5 L (Fig. 1). Nephrology was consulted for further management.

The patient reported that he had not been taking his home potassium or magnesium supplementation and endorsed nausea and lower extremity pain. On examination, temperature was 99 ˚F, blood pressure 100/58 mmHg, pulse 97, and oxygen saturation 94% on ambient air. The examination was normal with no findings of volume depletion or overload.

Treatment included reinitiation of amiloride, fluid restriction of 1.2L/24 h, and discontinuation of tramadol. Thyroid studies and cortisol were within normal limits. Despite three days of reported adherence to water restriction and salt tablets (2 g three times daily), the [SNa] remained between 123 and 128 mmol/L.

Published online: 15 September 2022

Table 1 Serum laboratory values

	1.5 Months prior to admission	Urgent care	Emergency department	Reference range
Sodium (mmol/L)	131	114	121	135–145
Potassium (mmol/L)	4.2	3.5	2.4	3.1–5.3
Chloride (mmol/L)	94	77	82	98–110
Carbon dioxide (mmol/L)	24	28	26	19–28
Urea nitrogen (mg/dl)	12	4	5	7–25
Creatinine (mg/dl)	0.95	0.6	0.76	0.7–1.3
Calcium (mg/dl)	10.5		8.9	8–1.5
Magnesium (mg/dl)	1.7		2.1	1.6–2.6
Osmolality (msom/kg H20)			248	275–295

To convert the values for urea nitrogen to millimoles per liter, multiply by 0.357. To convert the values for creatinine to micromoles per liter, multiply by 88.4

Reference values are affected by many variables, including the patient population and the laboratory methods used

Table 2 Urine laboratory values

	Day of admission	Hospital day 6	Hospital day 9	Hospital day 15	Hospital day 17	Reference values
Serum sodium, range	121–132	124–128	126	126–129	123–126	
Spot urine						
Osmolality (mOsm/kg H_2O)	61	65			92	
Sodium (mmol/L)	< 20	< 20	24	23	< 20	
Potassium (mmol/L)		7.8	15.4	8.3	12.3	
Magnesium (mg/dL)			3.2	< 1.8	3.1	
Calcium (mg/dL)			4.5			
Urea nitrogen (mg/dL)			79	61		
Creatinine (mg/L)			93	93		
Uric acid (mg/dL)			6.1	< 5.0		
24-h Urine						
Sodium (mmol/24 h)			75	82		40–220
Chloride (mmol/24 h)				100		110–250
Potassium (mmol/24 h)			48	30	36	25–125
Magnesium (mg/24 h)			100		91	7–125
Calcium (mg/24 h)			141	93		100–250
Urea nitrogen (g/24 h)			2	2	4	12–20
Creatinine (mg/24 h)			291	193	244	1,000–2,000
Uric acid (mg/24 h)			191			250–750
Volume (ml)			3128	3569	2938	

Blanks indicate value not calculated by laboratory

We obtained a 24-h urine study to assess adherence to fluid restriction and assess urinary electrolytes (Table 2). The urine sodium was 75 mmol, lower than the 155 mmol sodium the patient was receiving via salt tablets alone, not including additional dietary intake. Additionally, urine creatinine was less than 20% expected, urea nitrogen was 16% of the lower limit of normal, and uric acid was below assay. On two repeated measurements, urine creatinine excretion was less than 20% of expected. Creatinine clearance was 19 ml/min, suggesting advanced kidney failure, which the patient did not have.

Based on the patient's low excretion rates of urea, creatinine, and sodium, we suspected incomplete urine collection. With 3L of urine output, his likely fluid intake was potentially > 6L daily, despite repeated confirmation from the patient and nursing staff about strictly adhering to the fluid restriction. To test the hypothesis of incomplete urine collection and surreptitious polydipsia during bathroom visits, the water connection to the patient's bathroom faucet was

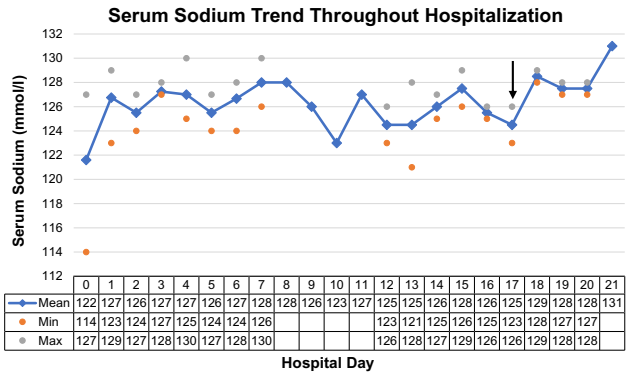

Fig. 1 Serum sodium trend throughout hospitalization. Blue line indicates mean serum sodium per hospital day. Orange dots indicate the lowest serum sodium value and gray dots indicate the highest serum sodium value in each hospital day. Arrow indicates hospital day 17, when bathroom faucet was turned off

disconnected for a 24-h period. The patient was informed of this and continued to have access to water from nursing staff and meal delivery. After this, the [SNa] improved to 131 mmol/l despite liberalizing the fluid restriction. The patient declined inpatient psychiatric evaluation, which was recommended as an outpatient.

Discussion

Severe neurological and life-threatening complications can arise because of water intoxication and subsequent hyponatremia. Like most hospitalized patients, the patient in our case had multiple potential etiologies for his hyponatremia on presentation and the diagnosis of primary polydipsia was not initially high on the differential. The initial impression of etiology was a combination of low solute intake, episodic ADH release due to pain, nausea, and renin–angiotensin–aldosterone system activation from hypotension in the setting of his tubular electrolyte wasting disorder (i.e., combination of "tea and toast" with SIADH). However, his persistent hyponatremia, inconsistent urine studies, and limited response to management required reconsideration of the working differential diagnosis. Urine studies were invaluable in determining the diagnosis of primary polydipsia, as the 24-h results were incompatible with normal human physiology and raised suspicion of surreptitious water intake while hospitalized.

Hyponatremia due to primary polydipsia is most often described in patients with underlying psychiatric disorders due to compulsive water drinking, which may also be an anticholinergic side effect of antipsychotic medications. Hyponatremia from antipsychotic medications can result from drug-induced SIADH [5, 6]. As in the case we presented above, primary polydipsia may be the first presenting feature of a psychiatric comorbidity, as it is unlikely that a person would be able to voluntarily drink beyond maximal concentrating capacity. The patient described here denied any prior psychiatric history or symptoms. However, he exhibited extreme, compulsive water drinking and became visibly agitated when his free water access was definitively restricted by disconnecting his in-room faucet for a short period of time. We were unable to find prior documentation in the literature to suggest that this is a commonly implemented technique for restricting access to free water either on the general medical wards or in Psychiatric units.

Hyponatremia in primary polydipsia typically becomes a relapsing condition unless behavioral modifications are implemented. For example, in a small retrospective cohort study of patients admitted with primary polydipsia in the United Kingdom, approximately half of hospital readmissions were secondary to recurrence of hyponatremia [5].

Conclusion

Severe hyponatremia is an uncommon complication psychiatric disorders and is precipitated by excessive free water intake. 24-h urine studies are useful in differentiating low solute intake states from primary polydipsia in patients with hypoosmolar hyponatremia. Surreptitious water intake in hospitalized patients may lead to a confusing diagnostic odyssey; in such cases, maneuvers, such as disconnection of the water supply from the bathroom plumbing, may uncover the diagnosis of primary polydipsia.

Funding Research reported in this publication was supported by the National Heart, Lung, and Blood Institute of the National Institutes of Health under Award Number R38HL143584.

Declarations

Conflict of interest All the authors have declared no competing interest.

References

1. Lu H, Vollenweider P, Kissling S, Marques-Vidal P. Prevalence and description of hyponatremia in a Swiss tertiary care hospital: an observational retrospective study. Front Med. 2020;7:512.
2. Sterns R. Disorders of plasma sodium - causes, consequences, and correction. N Engl J Med. 2015;372:55–65.
3. Hoorn EJ, Zietse R. Diagnosis and treatment of hyponatremia: compilation of the guidelines. J Am Soc Nephrol. 2017;28(5):1340–9. https://doi.org/10.1681/ASN.2016101139.
4. Turchin A, Seifter JL, Seely EW. Mind the gap. N Engl J Med. 2003;349:1465–9. https://doi.org/10.1111/ors.12417.

5. Sailer CO, Winzeler B, Nigro N, et al. Characteristics and outcomes of patients with profound hyponatremia due to primary polydipsia. Clin Endocrinol (Oxf). 2017;87:492–9.
6. McDonald D, McDonell T, Crowley RK, Brosnan E. Extreme hyponatraemia due to primary polydipsia and quetiapine-induced SIAD. Endocrinol Diabetes Metab Case Rep. 2021;2021:21–28.