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Case Report

Hyponatremia-Induced Rhabdomyolysis in a Patient with Psychogenic Polydipsia: A Case Report

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ABSTRACT

Hyponatremia is a common electrolyte abnormality that can lead to various complications, including rhabdomyolysis. Observational studies have identified a correlation between hyponatremia and the occurrence of rhabdomyolysis in hospitalized patients. We report a 45-year-old male with a medical history of hypertension, hyperlipidemia, and a congenital neurological defect featuring an 8 cm right frontal porencephalic cyst communicating with the right lateral ventricle, colpocephaly, congenital left hemiparesis, paranoid schizophrenia, anxiety, and depression. He presented with rhabdomyolysis caused by hyponatremia and aggravated by psychogenic polydipsia. A hyponatremic state caused by psychogenic polydipsia may induce rhabdomyolysis in patients with a genetic predisposition. Hence, monitoring muscle markers in these patients is crucial, with further evidence needed to establish hyponatremia as the primary cause of rhabdomyolysis in the absence of other confounders.

1. Introduction

Hyponatremia frequently occurs as an electrolyte disorder and causes several complications, including rhabdomyolysis [1] Rhabdomyolysis is a clinical and biochemical condition that occurs when injured muscle tissue releases its proteins and electrolytes into the bloodstream, causing heart and kidney injury and permanent disability or even mortality [2]. Observational studies [3, 4, 5] have established hyponatremia as a risk factor for rhabdomyolysis in hospitalized patients, particularly in settings of rapid sodium correction, extreme physical exertion, or comorbid metabolic disturbances. However, existing literature mostly focuses on hospitalized patients with apparent risk factors, leaving gaps in understanding the condition's presentation and management in non-hospitalized individuals. Although hyponatremia-induced rhabdomyolysis is uncommon, it represents a serious complication with significant renal morbidity. Our study presents a case report of hyponatremia-induced rhabdomyolysis with atypical presentation in a non-hospitalized patient with mild hyponatremia and no traditional triggers.

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2. Case presentation

A 45-year-old male with a history of hypertension, hyperlipidemia, and an 8 cm right frontal porencephalic cyst communicating with the right lateral ventricle, colpocephaly, congenital left hemiparesis, paranoid schizophrenia, anxiety, depression, and psychogenic polydipsia experienced an episode of loss of consciousness. Witnesses reported that the patient often visited his favorite fast-food restaurants and requested large water refills to prolong his stay. Emergency medical services found him unresponsive on the floor of a fast-food restaurant for approximately two minutes.

At the Emergency Department (ED), his blood pressure measured 141/73 mmHg, his heart rate was 82 beats per minute, his temperature was 97.5°F, his respiratory rate was 18 breaths per minute, and his oxygen saturation was 95% on room air. Upon examination, the patient appeared lethargic but was awake, cooperative, and oriented to person, place, and time. He was normocephalic and atraumatic on physical exam. Trace bilateral lower extremity pitting edema was observed, but no signs of volume overload were present. Although he could follow commands, his level of alertness prevented a full neurological assessment.

Laboratory findings revealed hyponatremia (111 mmol/L), hypochloremia (77 mmol/L), hypomagnesemia (1.5 mg/dL), hypocalcemia (8.2 mg/dL), and normal serum glucose. Additional findings included mild leukocytosis ($11.57 \times 10^3/\mu L$) and an elevated venous lactate level (3.2 mmol/L). Serum creatine kinase (CK) levels were markedly elevated at 3571 U/L **(Tables 1 and 2)**.

Table 1: Blood work done at the ED and ICU

Lab (Reference Range)	Reference Range	Result
Hemoglobin	14–18 g/dL	11.9 g/dL
Hematocrit	42–52%	33.4%
White blood cells	4.8–10.8 × 10^3/mcL	11.57 × 10^3/mcL
Platelets	150–450 × 10^3/mcL	219 × 10^3/mcL
Blood glucose	74–110 mg/dL	109 mg/dL
Sodium	136–145 mmol/L	111 mmol/L
Potassium	3.5–5.1 mmol/L	3.4 mmol/L
Chloride	98–108 mmol/L	77 mmol/L
Magnesium	1.6–2.6 mg/dL	1.5 mg/dL
Calcium	8.6–10.3 mg/dL	8.2 mg/dL
Phosphorus	2.5–4.5 mg/dL	3.7 mg/dL
BUN	6–23 mg/dL	8 mg/dL
Creatinine	0.7–1.2 mg/dL	0.64 mg/dL
Venous pH	7.32–7.43	7.45
Venous PO_2	30–50 mmHg	65 mmHg
Venous PCO_2	41–54 mmHg	31 mmHg
Venous HCO_3	22–29 mmol/L	22 mmol/L
Venous lactate	0.6–1.4 mmol/L	3.2 mmol/L
Albumin	3.5–5.2 g/dL	4.4 g/dL
Total bilirubin	0–1.2 mg/dL	1.3 mg/dL
Direct bilirubin	0–0.3 mg/dL	0.2 mg/dL
Alkaline phosphatase	40–129 U/L	59 U/L
ALT	0–41 U/L	38 U/L
Serum ammonia	16–60 µmol/L	51 µmol/L
Creatinine kinase	20–190 U/L	3571 U/L
Valproic acid level	50–100 µg/mL	41.8 µg/mL
Prolactin	4.1–18.4 ng/mL	13 ng/mL
Serum AM cortisol	6–18.4 µg/dL	6.6 µg/dL
TSH	0.27–4.2 µIU/mL	1.15 µIU/mL
Serum osmolality	285–303 mOsm/kg	264 mOsm/kg

Table 2: Urine work-up

Lab (Reference Range)	Reference Range	Result
Specific gravity	1005–1030	≤1005
Osmolality	50–1200 mOsm/kg	106 mOsm/kg
Random urea	—	31 mg/dL
Random chloride	—	11 mmol/L
Random sodium	—	44 mmol/L
Random potassium	—	4 mmol/L

The ED administered 2 liters of intravenous normal saline. A CT scan of the head showed no acute intracranial abnormalities, and chest imaging was unremarkable.

The patient was admitted to the intensive care unit (ICU) for close monitoring and gradual correction of sodium levels. Clinicians discontinued intravenous fluids, implemented fluid restriction, and corrected other electrolyte disturbances. A two-hour electroencephalogram (EEG) showed no seizure activity, and serum prolactin levels at admission (13 ng/mL; reference: 4.1–18.4 ng/mL) indicated no evidence of seizures. Over 36 hours, sodium levels were corrected to 143 mmol/L, and CK levels decreased to 124 U/L. The patient's symptoms improved, and he remained seizure-free during his hospitalization. After stabilization, he was discharged with instructions for outpatient follow-up.

3. Discussion

Psychogenic polydipsia manifests as a clinical disorder characterized by polyuria and polydipsia, which commonly affects psychiatric populations, particularly individuals with schizophrenia [6]. Although the exact mechanism by which psychogenic polydipsia leads to hyponatremia remains unclear, researchers attribute it to an increase in total body water relative to total body sodium content, which dilutes blood sodium levels [7].

Health practitioners frequently encounter hyponatremia in clinical practice but often fail to recognize its association with rhabdomyolysis. Malfunctioning of the sodium/calcium pump appears to drive the development of rhabdomyolysis in hyponatremic states by activating proteases and lipases that cause cell lysis [8]. While evidence linking low serum sodium levels to muscle breakdown continues to grow, a study involving animal models refuted a direct causal association between hyponatremia and rhabdomyolysis. Instead, factors such as genetic predispositions and convulsive states are well-established contributors [9]. Trimarchi et al. reviewed a case in 1999 involving acute excessive water ingestion and severe muscle pain in a patient on chronic hydrochlorothiazide therapy. The case demonstrated hyponatremia-induced rhabdomyolysis, emphasizing the importance of maintaining a high level of suspicion in patients presenting with acute muscle pain and hyponatremia. They recommended serial monitoring of muscle enzymes in such cases [10].

Several reports have identified hyponatremia as a trigger for rhabdomyolysis in patients using certain medications. For instance, myopathy has been documented with indapamide, a drug linked to hypokalemia and hyponatremia [11]. Similarly, severe rhabdomyolysis and hyponatremia were reported in patients using bisacodyl and picosulphate for bowel preparation before colonoscopies [12]. Increased water intake during competitive sports often disrupts fluid and electrolyte balance, particularly sodium levels. Studies have shown that ultra-endurance athletes experience higher rates of rhabdomyolysis in hyponatremic states compared to non-hyponatremic counterparts. This was evident in an investigation of exercise-associated hyponatremia and rhabdomyolysis across seven races and disciplines [13].

4. Conclusion

Recurrent rhabdomyolysis has been observed in cases of psychogenic polydipsia-induced hyponatremia, underscoring the importance of monitoring muscle markers in patients with severe hyponatremia. Although our case does not confirm seizures as a cause of myopathy, further research is necessary to establish hyponatremia as the primary cause of rhabdomyolysis in the absence of other confounders.

Conflicts of Interest

The authors declare that they have no competing interests.

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Informed consent

Written consent for publication was obtained from the patient involved in this case report.

Large Language Model

None

Authors Contribution

HA contributed to case identification and drafting the main manuscript. TA was responsible for the case presentation. PM wrote the introduction section. OA and A contributed to the discussion. AH served as the corresponding author and performed the manuscript review.

Data Availability

All the data is available with the corresponding Author upon request

References

1. Rondon H, Badireddy M. Hyponatremia. StatPearls Publishing, Treasure Island (FL); 2025. [PMID: 29262111, PMCID: PMCID].
2. Stanley M, Chippa V, Adigun R. In: Rhabdomyolysis. Treasure Island (FL): StatPearls Publishing Copyright © 2025, StatPearls Publishing LLC.; 2025. [PMID: 28846335, PMCID: PMCID].
3. Lim AKH, Bhonsle A, Zhang K, Hong J, Huang KLC, Nim J. Observational Study of the Association between Hyponatremia and Rhabdomyolysis in Patients Presenting to Hospital. J Clin Med. 2022;11(11). [PMID: 35683602, PMCID: PMC9181719, https://doi.org/10.3390/jcm11113215].
4. Mathew DM, Chilakala A, Elfert K. Rhabdomyolysis in the Setting of Severe Hyponatremia: A Case Report. Cureus. 2023;15(6):e39993. [PMID: 37416016, PMCID: PMC10321562, https://doi.org/10.7759/cureus.39993].
5. Reakes E, Drak D, Gracey D. A suspected case of hyponatraemia induced rhabdomyolysis: a case report. BMC Nephrol. 2022;23(1):180. [PMID: 35546656, PMCID: PMC9092814, https://doi.org/10.1186/s12882-022-02787-7].
6. Bhatia MS, Goyal A, Saha R, Doval N. Psychogenic Polydipsia - Management Challenges. Shanghai Arch Psychiatry. 2017;29(3):180-3. [PMID: 28904515, PMCID: PMC5579464, https://doi.org/10.11919/j.issn.1002-0829.216106].
7. Dundas B, Harris M, Narasimhan M. Psychogenic polydipsia review: etiology, differential, and treatment. Curr Psychiatry Rep. 2007;9(3):236-41. [PMID: 17521521, https://doi.org/10.1007/s11920-007-0025-7].
8. Aguiar DT, Monteiro C, Coutinho P. Recurrent rhabdomyolysis secondary to hyponatremia in a patient with primary psychogenic polydipsia. Rev Bras Ter Intensiva. 2015;27(1):77-81. [PMID: 25909317, PMCID: PMC4396901, https://doi.org/10.5935/0103-507X.20150013].
9. Peled M, Dolkart O, Finn T, Amar E, Zeltser D. No association between hyponatremia and rhabdomyolysis in rats. J Emerg Med. 2014;47(4):472-8. [PMID: 24485700, https://doi.org/10.1016/j.jemermed.2013.08.086].
10. Trimarchi H, Gonzalez J, Olivero J. Hyponatremia-associated rhabdomyolysis. Nephron. 1999;82(3):274-7. [PMID: 10396001, https://doi.org/10.1159/000045413].
11. Yue QY, Caduff-Janosa P. Indapamide-Induced Rhabdomyolysis: An Evaluation of Case Reports in VigiBase Using the Bradford Hill Criteria. Drugs Real World Outcomes. 2022;9(2):189-98. [PMID: 35235186, PMCID: PMC9114225, https://doi.org/10.1007/s40801-022-00295-6].
12. Mas A, Chillaron JJ, Esteve E, Navalpotro I, Supervia A. Severe rhabdomyolysis and hyponatremia induced by picosulfate and bisacodyl during the preparation of colonoscopy. Rev Esp Enferm Dig. 2013;105(3):180-1. [PMID: 23735032, https://doi.org/10.4321/s1130-01082013000300016].
13. Chlibkova D, Knechtle B, Rosemann T, Tomaskova I, Novotny J, Zakovska A, et al. Rhabdomyolysis and exercise-associated hyponatremia in ultra-bikers and ultra-runners. J Int Soc Sports Nutr. 2015;12:29. [PMID: 26113805, PMCID: PMC4480906, https://doi.org/10.1186/s12970-015-0091-x].