Hyperacute hyponatremia mimicking acute ischemic stroke: A case report.

Alanna Balbi

Jennifer A Sadowski

Daniel Torrens

Jeanne L Jacoby

Hussam A Yacoub

See next page for additional authors

Authors

Alanna Balbi, Jennifer A Sadowski, Daniel Torrens, Jeanne L Jacoby, Hussam A Yacoub, and Jessica K Eygnor

American Journal of Emergency Medicine xxx (2021) xxx

Contents lists available at ScienceDirect

American Journal of Emergency Medicine

journal homepage: www.elsevier.com/locate/ajem

Hyperacute hyponatremia mimicking acute ischemic stroke: A case report

Alanna Balbi, MD [a], Jennifer A. Sadowski, DO [a], Daniel Torrens, DO [a], Jeanne L. Jacoby, DO [a], Hussam A. Yacoub, DO, MS [b], Jessica K. Eygnor, DO [a,*]

[a] Lehigh Valley Health Network, Department of Emergency and Hospital Medicine/USF Morsani College of Medicine, Cedar Crest Boulevard & I-78, Allentown, PA 18103, USA
[b] Lehigh Valley Health Network, Department of Neurology, Cedar Crest Boulevard & I-18, Allentown, PA 18103, USA

ARTICLE INFO

Article history:
Received 22 February 2021
Received in revised form 9 June 2021
Accepted 5 July 2021
Available online xxxx

Keywords:
Hyperacute hyponatremia
Stroke

ABSTRACT

We present a case of hyperacute hyponatremia with stroke like symptoms on presentation. Symptoms included confusion, left-sided facial droop, right-sided hemiparesis, dysarthria and aphasia, with an NIH stroke score of 5. Sodium level at the time of presentation was 119 mmol/L which dropped acutely from 138 mmol/L seven hours prior. Symptoms improved after treatment with 3% saline and no evidence of stroke, intracranial hemorrhage or space-occupying lesion was seen on imaging. The most likely cause of the hyponatremia was increased free water consumption and ADH surge. The patient remained symptom free after discharge with resolution of hyponatremia. Acute hyponatremia can cause focal neurological complaints and deficits, mimicking acute ischemic stroke. We advise clinicians to be aware of this entity when considering interventions for possible acute ischemic stroke and evaluating a patient with focal neurological deficits.

1. Case report

A 67-year-old woman presented to the emergency department with altered mental status, facial asymmetry, and hemiparesis. Medical history was significant for hyperlipidemia. The morning prior, routine serum and urine laboratory testing was obtained for difficulty urinating. The patient was instructed to increase fluid intake to at least 1.5 l of water per day. She subsequently developed nausea and vomiting, followed by confusion, left-sided facial droop, and right-sided hemiparesis. Initial evaluation in the emergency department revealed a blood pressure of 110/51 mmHg, pulse of 69 beats per minute, temperature of 98 °F (36.7 °C), respiratory rate of 18 breaths per minute, and room SpO2 of 97%. She was noted to be dysarthric and aphasic, with left-sided facial droop and right-sided upper and lower extremity drift. On further evaluation, she was found to be euvolemic. Initial GCS was 14 (E4, V4, M6) for confusion, and the NIH Stroke Scale was 5 for impaired response to level of consciousness questions, right-sided weakness, left-sided facial palsy, and mild aphasia.

Initial morning outpatient laboratory workup revealed sodium of 138 mmol/L, calcium of 8.4 mg/dL, and glucose of 121 mg/dL. Seven hours later, studies in the emergency department showed serum sodium of 119 mmol/L, glucose of 128 mg/dL, potassium of 3.6 mmol/L, and osmolality of 261 mOsm/kg. Urinalysis revealed glucose of 100 mg/dL, potassium of 10 mg/dL, sodium of 37 mg/dL, chloride of 39 mg/dL, and a specific gravity of 1.005. Urine osmolality was 258 mOsm/kg and 112 mOsm/kg six and nine hours after arrival to the emergency department, respectively. Antidiuretic hormone (ADH) twelve hours after presentation was normal at <0.5 pg/mL. The patient's lab results over a 24 hour period are reported. (See Table 1 .) Computed tomography of the head did not reveal any acute intracranial hemorrhage, and vessel imaging of the head and neck was unremarkable. Magnetic resonance imaging of the brain did not show an acute ischemic stroke or any space-occupying lesion. She was diagnosed with hyperacute hyponatremia mimicking symptoms of an acute stroke.

The patient was given a bolus of 100 ml of 3% saline, and after correction of serum sodium to 136 mmol/L and 140 mmol/L five and twelve hours after treatment intervention, respectively, was given D5W since her urine remained diluted. As a result, her mental status and focal neurological deficits significantly improved. Three days after hospital discharge, serum sodium level was stable at 139 mmol/L, with no recurrence of symptoms.

2. Discussion

Hyponatremia is defined as a disorder of total body water and sodium imbalance [1]. It is the most common electrolyte disturbance with a higher incidence in older patients and can be caused by either solute loss or water retention with an excess of water in relation to

* Corresponding author at: LVH-M-5th floor EM Residency S, 2545 Schoenersville Road, Bethlehem, PA 18017, USA.
E-mail address: Jessica_K.Eygnor@lvhn.org (J.K. Eygnor).

https://doi.org/10.1016/j.ajem.2021.07.017
0735-6757/

Table 1
Patient's lab results over a 24 h period.

Time	Serum Osmo	Na	K	Cl	Ca	Glucose	Urine Specific Gravity	Urine Osmo	Urine Na	Urine K	Urine Cl
10:11		138	5.2	105		121					
17:00		119	3.5	89		128					
19:13	261						1.005	258	37	10	39
23:00		132	3.5	100	8.1	107		122	24	2.4	17
2:20		136	3.7	104	8.4	139					
6:43		141	4.0	108	8.4	117					
10:45		139	4.5	109	8.6	136					

solute [2,3]. Hyponatremia refers to a serum sodium level of less than 136 mmol/L and can be grouped into three different categories; euvolemic hyponatremia, hypervolemic hyponatremia, and hypovolemic hyponatremia. Hypovolemic hyponatremia is a decrease in total body water with greater decrease in total body sodium as is seen with vomiting, diarrhea and sweating. Euvolemic hyponatremia is a decrease in total body sodium with normal total body water. This category can be further divided based on urine sodium concentration. Dilute urine sodium concentration can be seen in adrenal insufficiency, hypothyroidism, excess free water intake, and beer potomania. Concentrated urine is exhibited in syndrome of inappropriate antidiuretic hormone (SIADH). Hypervolemic hyponatremia is an increase in total body sodium with a greater increase in total body water as is shown in heart failure, liver failure and renal failure. Hyponatremia that develops within 48 h is defined as acute [13].

A physiological response to increased free water intake is a decrease in the release of ADH leading to excretion of excess water. Patients with primary polydipsia may overwhelm the free water excretory capacity of the kidneys, defined as 10 to 20 l of water per day, leading to hyponatremia [4]. Water retention may also occur with normal water intake and renal impairment. Most patients with hyponatremia have an excess amount of ADH either due to (SIADH) or relative volume depletion. ADH acts on the distal tubules and collecting ducts of the kidney inducing expression of water transport proteins leading to increased water reabsorption [2]. This can lead to a urine osmolality greater that 100 mOsmol/kg and an elevated urine sodium concentration above 40 meq/L [1]. Renal water excretion is impaired through effective circulating volume depletion as a result of diuretics use, hypoaldosteronism, vomiting, diarrhea, or edematous states such as heart failure.

In this case, acute hyponatremia was likely caused by the patient's free water intake following initial testing. This patient additionally had severe nausea and vomiting, a trigger of ADH release, which could have potentially led to an even more significant acute drop in serum sodium. Similar changes may occur with the recreational drug ecstasy (3,4-methylenedioxymethamphetamine [MDMA]). Ecstasy use is often associated with an increase in free water intake, and MDMA metabolites are known to stimulate ADH secretion [10,11].

Vomiting secondary to cerebral edema can lead to excessive loss of sodium and increased ADH secretion as well. Brain imaging in our patient did not reveal any evidence of edema. With a normal extracellular fluid volume, appropriately concentrated urine at 258 mOsm/kg, and normal urine sodium concentration of 37 mg/dL, etiologies such as hypothyroidism, diuretic use, and secondary adrenal insufficiency were also excluded as potential etiologies. Renal impairment also did not contribute to our patient's hyponatremia as she had no prior renal disease with normal BUN and creatinine. Although our patient did meet some of the criteria of SIADH, it is presumed she had increased free water intake which would potentially disqualify her from that diagnosis [4,6].

Although rapid correction of both hypernatremia and hyponatremia in the hyperacute setting of less than 24 h has not been shown to be harmful or cause Osmotic Demyelination Syndrome (ODS) [5,9], D5W was eventually started since patient's sodium corrected to 132 mm/L at two hours and urine remained dilute. The initial goal was to increase the sodium concentration by 4–6 meq/L in the first six hours. Additionally, if the initial bolus had not accomplished our sodium goal, 2 further boluses of 3% saline could also have been given [5]. In the setting of hyponatremia occurring within 24–48 h after initial correction of 4–6 meq/L in the first 6 h one should avoid increasing the serum sodium concentration by more than 10 meq/L/day. If the hyponatremia is chronic or of duration longer than 48 h, the initial sodium can be increased by 4-6 meq/L in the first 24 h, but sodium should not be increased by more than 8 meq/L/day [5,6]. This can be further complicated by the water diuresis that can occur with hypertonic saline infusion, leading to even higher corrected sodium concentrations in some patients, as may have occurred in our patient [8]. In the setting of chronic hyponatremia, if overcorrection occurs, lowering the sodium should be considered, particularly in patients with a high risk for ODS due to liver disease, alcoholism, thiazide diuretic use, or hypoxia. ODS is related to cellular compensatory mechanisms than occur in the setting of chronic hyponatremia. The cells excrete osmolytes, such as inorganic ions and organic compounds in an effort to regulate cell volume. When sodium is corrected too rapidly, the cells cannot reabsorb the organic compounds or produce inorganic ions quickly enough leading to osmotic stress and apoptosis [7]. Evidence has also shown that methods (such as Adrogue-Medias formula) used to calculate predicted sodium concentration after hypertonic saline infusion often underestimate the increase in sodium concentration for various reasons and can lead to inadvertent overcorrection, therefore we advise clinicians to be cautious about additional water diuresis [8].

It is also important to be aware that patients with neurologic symptoms in the context of hyponatremia are at risk for pulmonary edema (Ayus-Arieff syndrome) secondary to cerebral edema. This occurs via one of two mechanisms: 1) centrally mediated increased pulmonary vascular protein permeability leading to interstitial edema, or 2) increased catecholamine release leading to increased capillary pressure and injury [12]. This often leads to hypoxia triggering a cycle of additional cerebral edema. Our patient fortunately did not exhibit any clinical or radiographic evidence of cerebral or pulmonary edema.

Declarations of interest

None.

The authors have no outside support information, conflicts or financial interest to disclose.

Author contributions

All authors provided substantial contributions to manuscript content. All authors gave final approval of the version of the article to be published.

Acknowledgements

Authors would like to acknowledge Dr. James Reichart for his critical review of this case report.

Authors would like to acknowledge Erin Shigo, BA for formatting and editing.

References

[1] Rose BD. Clinical physiology of acid-base and electrolyte disorders. 5th ed.. McGraw-Hill; 2001.
[2] Grant P, Ayuk J, Bouloux P-M, et al. The diagnosis and management of inpatient hyponatraemia and SIADH. Eur J Clin Invest. 2015;45(8):888–94. https://doi.org/10.1111/eci.12465.
[3] Ganguli A, Mascarenhas RC, Jamshed N, Tefera E, Veis JH. Hyponatremia: incidence, risk factors, and consequences in the elderly in a home-based primary care program. Clin Nephrol. 2015;84(2):75–85. https://doi.org/10.5414/CN108453.
[4] Hoorn EJ, Zietse R. Diagnosis and treatment of hyponatremia: compilation of the guidelines. J Am Soc Nephrol JASN. 2017;28(5):1340–9. https://doi.org/10.1681/ASN.2016101139.
[5] Spasovski G, Vanholder R, Allolio B, et al. Clinical practice guideline on diagnosis and treatment of hyponatraemia. Eur J Endocrinol. 2014;170(3):G1–47. https://doi.org/10.1530/EJE-13-1020.
[6] Sterns RH. Disorders of plasma sodium — causes, consequences, and correction. N Engl J Med. 2015;372(1):55–65. https://doi.org/10.1056/NEJMra1404489.
[7] Sterns RH. Brain volume regulation in response to hypo-osmolality and its correction. Am J Med. 2006 Jul:119.
[8] Mohmand HK. Hypertonic saline for hyponatremia: risk of inadvertent overcorrection. CJSAN. November 2007;2(6):1110–7.
[9] Carlberg DJ. Survival of acute hypernatremia due to massive soy sauce ingestion. JEM. August 2013:228–31.
[10] Fallon JK. Action of MDMA (ecstasy) and its metabolites on arginine vasopressin release. Ann N Y Acad Sci. 2002;965:399–409 [PubMed] [Google Scholar].
[11] Forsling ML. The effect of 3,4-methylenedioxymethamphetamine (MDMA, 'ecstasy') and its metabolites on neurohypophysial hormone release from the isolated rat hypothalamus. Br J Pharmacol. 2002;135:649–56.
[12] Smith WS. Evidence for a hydrostatic mechanism in human neurogenic pulmonary edema. Chest. 1997;111:1326–33.
[13] Berkowitz R, Rose G. Pancreas. In: Walls RM, Hockberger RS, Gausche-Hill M, et al, editors. Rosen's emergency medicine: Concepts and clinical practice. 9th ed.Elsevier; 2018 Chapter 117 Electrolyte Disorders.