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Case Report

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Extreme Hypernatremia due to Dehydration

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Abstract

Hypernatremia is defined as having a serum sodium concentration exceeding 145 mmol/L (normal range is 136 - 145 mmol/L). When the serum sodium level surpasses 160 mmol/L and 190 mmol/L, it is classified as severe and extreme hypernatremia, respectively. Extreme hypernatremia is an uncommon occurrence and is associated with significant risks of mortality and morbidity. The most frequently reported causes of extreme hypernatremia are accidental or intentional salt ingestion and diabetes insipidus, while dehydration due to poor water intake as a trigger for extreme hypernatremia is rarely documented. Here, we present a case study of an elderly patient who experienced extreme hypernatremia with a serum sodium level of 191 mmol/L due to dehydration and had altered mental status and ventriculomegaly as a complication.

Keywords: Hypernatremia; Ventriculomegaly; Extreme hypernatremia; Mortality; Ventriculoperitoneal shunt; Electrolyte abnormality; Dehydration; Correction

Introduction

Hypernatremia is a common electrolyte abnormality in critically ill patients, but extreme hypernatremia is a rare phenomenon and is defined as serum sodium concentration greater than 190 mmol/L [1]. We describe a case of extreme hypernatremia secondary to dehydration in a 70-year-old male who presented with altered mental status, jaw clenching, and a serum sodium concentration of 191 mmol/L. Case reports with extreme hypernatremia reported in the literature were mostly secondary to increased sodium intake or hypertonic solutions, despite the most common cause of hypernatremia being excess water loss.

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Severe and extreme hypernatremia is associated with high mortality and morbidity, and mortality increases with higher initial serum sodium on admission [2]. There are few reported cases of extreme hypernatremia with serum sodium of more than 190 mmol/L on admission especially secondary to dehydration. We are presenting a case of extreme hypernatremia secondary to dehydration, who developed ventriculomegaly/hydrocephalus, regardless of guideline-based therapy that did not have a successful outcome, posing a therapeutic challenge to treat patients with sodium levels of more than 190 mmol/L.

Case Report

Investigations

A 70-year-old male resident of a nursing home, with a past medical history of Parkinson's dementia, deep vein thrombosis on chronic anticoagulation, right ventriculoperitoneal shunt (VPS) for ventriculomegaly due to aqueduct stenosis, presented to the hospital with altered mental status. At baseline, the patient was bedbound and wheelchair-dependent but was alert, oriented, and able to hold a normal conversation, prior to this episode. One week prior to the hospital admission he had received a course of azithromycin for cough. Upon arrival at the hospital, the patient was afebrile with a temperature of 36.4 °C, tachycardic with a heart rate of 101 beats/min, hypotensive with a blood pressure of 78/51 mm Hg, respiratory rate of 15/min, and oxygen saturation of 97% at room air. On physical examination, he appeared cachectic with a scaphoid abdomen and mottled lower extremities. He was unresponsive to noxious stimuli and had coarse crackles throughout all the lung fields. He was intubated upon arrival at the hospital for airway protection, because of a low Glasgow comma scale (GCS) score of 3.

Diagnosis

The initial laboratory workup showed serum sodium 191 mmol/L (136 - 145 mmol/L), chloride > 150 mmol/L (98 - 107 mmol/L), potassium 5.1 mmol/L (3.5 - 5.1 mmol/L), bicarbonate 18 mmol/L (22 - 30 mmol/L), anion gap 23 mmol/L (< 18 mmol/L), glucose 162 mg/dL (70 - 99 mg/dL), blood urea nitrogen (BUN) 119 mg/dL (8 - 26 mg/dL), creatinine (Cr) 3.17 mg/dL (0.7 - 1.3 mg/dL). Blood gas analysis showed a pH of 7.36 (7.35 - 7.45), PCO_2 27.6 (35 - 45 mm Hg). Other lab work included creatinine phosphokinase (CPK) 598 U/L (30 - 200 U/L), magnesium 3.5 mg/dL (1.6 - 2.3 mg/dL), thyroid-stimu-

Table 1. Basic Metabolic Profile on Admission, 24, 48, 72, and 96 h

	Normal range	On admission	24 h	48 h	72 h	96 h
Sodium (mmol/L)	136 - 145	191	182	171	164	157
Potassium (mmol/L)	3.5 - 5.1	5.1	3.9	3.5	3.1	3.3
Chloride (mmol/L)	98 - 107	> 150	> 150	146	137	128
CO_2, venous, mmol/L	22 - 30	18	17	15	21	19
Anion gap (mmol/L)	< 18	< 23	< 17	10	6	10
Glucose (mg/dL)	70 - 99	162	236	204	155	97
BUN (mg/dL)	8 - 26	119	31	31	44	27
Creatinine (mg/dL)	0.7 - 1.3	3.17	2.9	2.27	1.43	1.13
Albumin (g/dL)	3.5 - 5	3.5	2.7	2.6	2.2	2.3

BUN: blood urea nitrogen.

lating hormone (TSH) 1.057 mIU/L (0.3 - 5 mIU/L), albumin 3.5 g/dL (3.5 - 5 g/dL), calcium 9.5 mg/dL (8.7 - 10.5 mg/dL), total bilirubin 0.7 mg/dL (0.2 - 1.2 mg/dL) and normal liver function tests. White blood cell count was elevated to 18,700/mm^3 (4 - 12.00 × $10^3/mm^3$), hemoglobin 18.4 g/dL (13 - 16.5 g/dL), platelet 269,000/mm^3 (140 - 400 × $10^3/mm^3$), lactic acid 3.1 mmol/L (0.7 - 2 mmol/L), troponin 39 ng/L (≤ 35 mg/L), brain natriuretic peptide (BNP) 33 pg/mL (< 100 pg/mL). Coronavirus disease 2019 (COVID-19), influenza A/B, and respiratory syncytial virus by polymerase chain reaction (PCR) and urine analysis were negative. Chest X-ray showed infiltrate versus atelectasis on the left lower lobe. Computed tomography (CT) of the head showed marked ventriculomegaly with a VPS tip positioned in the left lateral ventricle. There was an initial concern about shunt malfunction, so an X-ray shuntogram was performed which did not show any disconnections. Externalization of the left VPS and placement of a right VPS was performed but follow-up imaging showed no significant change in the ventricular size. In the absence of shunt malfunction, ventriculomegaly was thought to be secondary to fluid shift in brain parenchymal cells due to acute hypernatremia.

Treatment

Given the extreme hypernatremia, encephalopathy, hypotension, and a clinical exam consistent with severe dehydration, he had received initial fluid resuscitation with 3 L of normal saline followed by dextrose 5% water at 75 mL/h. His electrolytes on admission, 24, 48, 72, and 96 h later are summarized in Table 1.

Electroencephalogram (EEG) demonstrated bitemporal epileptiform discharges concerning for high seizure risk and levetiracetam was initiated for seizure prevention. He was also treated with vancomycin and piperacillin and tazobactam at 4.5 g every 8 h for pneumonia. Other infectious disease workups including blood cultures remained negative.

Follow-up and outcomes

Despite electrolyte correction and cerebrospinal fluid diversion, the patient had minimal neurological improvement. His sodium was gradually corrected over a period of 6 - 7 days to 144 mmol/L. He developed ventilator-associated pneumonia and could not be weaned off the ventilator. His healthcare power of attorney chose comfort-focused care for him, and he eventually passed away on the 11th day of hospitalization.

Discussion

Hypernatremia is defined as a serum sodium level of more than 145 mmol/L, severe hypernatremia is serum sodium level above 160 mg/dL while extreme hypernatremia is serum sodium level above 190 mmol/L [3]. Hypernatremia is a very frequent electrolyte abnormality seen in critically ill patients with an incidence of 9% and prevalence accounting for 27% among patients during their intensive care unit (ICU) stay [4, 5]. Severe hypernatremia is associated with a 40% and 28% increase in mortality and length of stay, respectively [6]. Normal serum osmolality of 280 - 295 mOsm/kg is maintained by vasopressin, thirst, and renal response to vasopressin; hypernatremia is caused by dysfunction of these mechanisms [7].

The most commonly reported cause of extreme hypernatremia is accidental, intentional ingestion, or iatrogenic administration of hypertonic fluids (Table 2) [8-21]. Cases of extreme hypernatremia due to dehydration caused by inadequate oral intake are very rare in the realm of clinical practice. Our literature search revealed only one case of extreme hypernatremia with serum sodium levels of 187 mmol/L due to poor oral intake and resultant dehydration in an 85-year-old male patient with dementia [19]. In neurologically intact patients, as the plasma osmolality rises, anti-diuretic hormone (ADH) is released which stimulates the thirst center. Thus, minimizing further water loss and preventing the worsening of hypernatremia [22]. Our patient's immobility status, age, and comorbidities likely made him vulnerable to extreme hypernatremia given his inability to seek water when needed.

Hypernatremia is defined as acute when elevated serum sodium levels are present for 48 h or less. Initial symptoms

Table 2. Summary of Case Reports on Severe and Extreme Hypernatremia

Author/year	Patient's age (years)	Etiology	Na^+ level
Ofran et al, 2004 [8]	20	Exorcism (use of salt solution)	255 mmol/L
Metheny et al, 2020 [9]	34	Salt solution was used as anti-emetic	196 mmol/L
Turk et al, 2005 [10]	69	Salt solution given as anti-emetic	175 mmol/L
Furukawa et al, 2011 [11]	55	Intentional ingestion of depressed patient by drinking 700 mL of Japanese soy sauce (shoyu)	187 mmol/L
Yamazaki et al, 2000 [12]	60	Intentional ingestion of psychotic patient by drinking 1 L bottle of shoyu	Estimated at more than 177 mmol/L at the time of death
Carlberg et al, 2013 [13]	19	College prank, ingesting a quart of soy sauce	182 mmol/L
Xiao et al, 2019 [14]	58	Undiagnosed DM	191 mmol/L, corrected 202 mmol/L
Ju et al, 2013 [15]	20	Self-administered to lose weight and improve digestion	174 mmol/L
Moder et al, 1990 [16]	41	Ingestion of supersaturated salt water solution intended for gargling	209 mmol/L
Mohtadi et al, 2015 [17]	33	Emetic (self)	175 mmol/L
Machino et al, 2006 [18]	73	Suicide	188 mmol/L
Nur et al, 2014 [19]	85	Dehydration due to poor oral intake (dementia)	187 mmol/L
Choi et al, 2013 [20]	40	Paranoid adipsia	172 mmol/L
Bhosle et al, 2015 [21]	54	Iatrogenic from wrong bicarb concentration during HD	207 mmol/L

DM: diabetes mellitus; HD: hemodialysis.

with acute hypernatremia include lethargy, weakness, and irritability, and can progress to seizures and coma. Chronic hypernatremia is defined when elevated serum sodium levels were present for more than 48 h and are less likely to have severe symptoms given the cerebral adaptation. Severe symptoms with neurological manifestations occur when there is an acute elevation in serum sodium concentration above 160 mmol/L, and values above 180 mmol/L are associated with high mortality, especially in elderly individuals. In patients with extreme hypernatremia with neurological deficits, an initial CT head is recommended to detect possible intracranial hemorrhage due to vascular rupture from the shrinkage of brain cells [23]. Other rare neurological deficits have been reported, including transient ventriculomegaly, which was also seen in our patient. The brain is very vulnerable to fluid shifts associated with a hyperosmolar state from hypernatremia, and one case report showed a correlation between different sodium levels and the variation in ventricular size. Case reports have also described transient ventriculomegaly in patients with shunted hydrocephalus from hypernatremia which resolved after correction of osmolar imbalance. Shunted patients have altered brain elasticity as in other patients with tumors, hemorrhages predisposing them to a higher risk of ventriculomegaly with hypernatremia [24].

Age and levels of serum sodium are independent risk factors for mortality in hypernatremia [25]. Geriatric population, nursing home residents are mainly dependent on help to maintain water intake. Their urge to drink is usually impaired and they cannot seek water for themselves making them vulnerable to dehydration and resultant severe hypernatremia [26]. Studies showed that there is decreased thirst response in elderly patients compared to younger subjects after water deprivation for about 24 h [27]. Along with the impaired thirst mechanism, the renal concentrating ability to increase sodium excretion in response to sodium load is impaired with aging [28]. Another physiological change that occurs with aging is the decrease in total body water predisposing the elderly population to dehydration and subsequent hypernatremia with minimal loss of fluid [29].

A delay in diagnosis and treatment of hypernatremia can lead to increased mortality. Despite being a very common electrolyte abnormality, management of hypernatremia is challenging, as it needs an understanding of body fluid compartments and concepts of normal body water balance. Rapid and overcorrection can lead to fatal outcomes due to cerebral edema. As per current evidence in treating hypernatremia, a reduction rate of no more than 0.5 mmol/L per hour or > 12 mmol/L in 24 h is recommended. However, some studies identified that rapid correction of hypernatremia with more than 0.5 mmol/L per hour or > 12 mmol/L in 24 h was not associated with high mortality or cerebral edema and recommended correcting sodium in order to decrease the length of stay in this vulnerable patient population. The rapid correction which is increasing the rate of free water administration has been found to be beneficial, especially in patients developing hypernatremia secondary to dehydration. The goal of treatment for hypernatremia is the restoration of plasma tonicity, and rapid correction improves the prognosis by preventing the effects of cellular dehydration, especially when hypernatremia developed acutely [30]. Our patient's sodium was corrected gradually as per the current evidence-based guidelines and still did not have the desired outcome, and there was no

evidence of cerebral edema but ventriculomegaly which is a rare neurological complication.

Conclusions

This case illustrates the high risk of mortality and morbidity associated with extreme hypernatremia and the need for reinforcing the importance of hydration in the elderly population. Also, it highlights the need for more research and evidence on the efficacy and safety of rapid correction of serum sodium in hypernatremia to decrease mortality in these vulnerable patient populations.

Learning point

Extreme hypernatremia is associated with a high risk of mortality and morbidity; early recognition and appropriate treatment are vital to have desired outcomes. Severe dehydration, although a rare cause, can cause extreme hypernatremia making elderly patients vulnerable to severe electrolyte abnormalities.

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Conflict of Interest

All authors declare no conflict of interest.

Informed Consent

Informed consent was waived as this is a retrospective study. No patient identifier is included in the case report.

Author Contributions

Sravani Kamatam contributed to the literature search and wrote the manuscript. Ayesha Waqar contributed to case selection and proofreading. Tulika Chatterjee contributed to the literature search, manuscript editing, and proofreading.

Data Availability

Authors declare that the data supporting the findings in the case report are available within the article.

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