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Acute correction of hyponatremia secondary to psychogenic polydipsia

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Summary

Background:

Psychogenic polydipsia is prevalent amongst psychiatric patients, but less common in the general population. Generally, hyponatremia ensues with complications of cerebral edema resulting in confusion, seizures, coma, and death. Rapid correction of serum sodium levels can lead to further complications of osmotic demyelination of neurons, e.g. central pontine myelinolysis.

Case Report:

We present a case of a 32-year-old male who presented with seizures while being treated at a drug rehabilitation facility. He was discovered to be hyponatremic secondary to suspected psychogenic polydipsia. The patient impressively responded to treatment of fluid restriction and desmopressin and symptoms improved.

Conclusions:

Amongst the causes of hyponatremia, psychogenic polydipsia may be more difficult to diagnose especially if an apparent psychiatric condition is not present. Current literature supports cautious correction of hyponatremia to prevent complications. However, rapid corrections may be driven by the physiology of the patient and may not be avoidable. Fortunately, our case illustrates rapid, positive outcomes for the patient.

key words:

hyponatremia • psychogenic polydipsia • seizure • correction

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BACKGROUND

Acute hyponatremia has several causes. Most commonly, dilutional (hypotonic) hyponatremia is seen when water intake exceeds the excretional capacity of the kidneys, usually in the setting of renal impairment [1,2]. Rarely, hyponatremia is seen as a result of extreme water consumption.

We present a case of acute hyponatremia precipitated by psychogenic polydipsia in a male being treated for drug dependence. This case highlights the physiologic effects of acute hyponatremia.

CASE REPORT

The patient is a 32-year-old male being treated at a rehabilitation facility for long-term history of alcohol, amphetamine, opiate, benzodiazepine, and marijuana dependency. After three days of treatment, the patient was seen at the local emergency department (ED) for suspected delirium tremens, aggression, and agitation related to alcohol and benzodiazepine withdrawal. The ED evaluation disavowed delirium tremens, but agreed with alcohol/drug withdrawal. No abnormalities were noted in behavior, on physical exam, or laboratory studies and the patient was discharged back to the rehab facility with a 3 day course of chlorthalidone (Librium), for the management of alcohol withdrawal.

Subsequently, the patient's behavior improved throughout the week. Ten days later, the patient was noted to have altered mental status including hallucinations and was observed by staff to be drinking water directly from the faucet. Later that day, the patient developed acute seizure activity, exhibiting tonic-clonic movements, loss of consciousness, and urinary incontinence. He was treated with lorazepam (Ativan) and was transported to the ED.

In the ED, the patient had a GCS of 11 (E4, V3, M4). Due to large clear liquid emesis, the patient was intubated. Following intubation, the patient had another seizure and was treated with midazolam (Versed) and propofol. The patient's initial serum sodium in the ED was 113 mmol/L, markedly decreased from 142 mmol/L 10 days earlier. The patient was treated with infusion of two liters of normal saline, which raised his sodium to 119 mmol/L. An orogastric tube was placed and returned large amounts of clear liquid. He was started on piperacillin/tazobactam (Zosyn) for possible aspiration and received fosphenytoin load for the seizures. A Foley catheter was placed and head CT was obtained, which showed generalized cerebral edema. The diagnosis was hyponatremia presumably secondary to psychogenic polydipsia.

After transfer to the intensive care unit (ICU), efforts of correcting the hyponatremia ensued with a goal of sodium correction of no more than 10–12 mmol/24 hr while closely monitoring sodium levels. The patient was maintained on a fluid restriction for a 24-hour period and was administered 2 mcg of desmopressin to prevent overcorrection. The patient was also treated for low magnesium, potassium, and calcium per standard protocol. Initial labs in ICU consisted of urine osmolality of 204 mmol/kg with urine sodium of 37 mmol/L and serum osmolality of 240 mmol/kg. Thyroid function and cortisol levels were normal.

Despite desmopressin, the serum sodium level rapidly increased from 113 mmol/L to 136 mmol/L in 9.5 hours (a difference of 23 mmol/L). In addition, the patient exhibited urine output of 8 liters in 30 hours. Additional testing included MRI, which showed no abnormalities, and EEG that showed periodic diffuse theta slowing, consistent with mild diffuse encephalopathy, but no epileptiform or seizure activity.

In-house psychiatric evaluation was sought due to suspicion of psychogenic polydipsia. He had a DSM IV Axis V global assessment of functioning of 60–70 (defined as mild depressive symptoms, difficulty in social/occupational settings, generally functioning well), but he was not believed to be psychotic. No additional seizures or psychotic behavior was observed during the course of his hospital stay. The patient was discharged to home after four days in hospital. He refused continued treatment at the rehab facility.

DISCUSSION

Hyponatremia is defined as serum sodium less than 136 mmol/L and indicates excess of water relative to sodium [1,3]. The effects of acute hyponatremia are a result of water movement down its concentration gradient into the cells causing swelling, which may induce headache, confusion, seizure, and/or herniation of the brain-stem [1,2]. Andrew indicates that cells in the brain, specifically neurons and glial cells, are very susceptible to acute changes in osmolality. The hypoosmolar state on the brain can physiologically provoke neuron excitability and result in seizures [4].

Management of acute hyponatremia is based upon total sodium correction per day and is limited to 8–12 mmol/24 hr to prevent rapid correction and demyelination [1,5]. The rate of initial correction can be drastic, such as 1–2 mmol/hr, depending on the severity of symptoms, but should still be limited to 8–12 mmol/24 hr [1]. Alternatively, Schrier suggests hypertonic saline in conjunction with a loop diuretic [5]. In addition, desmopressin has been shown to help prevent and reverse inadvertent overcorrection [6].

Acute corrections in solute concentrations can cause osmotic demyelination by a rapid movement of water out of the cell, which can result in persistent seizures, palsies, plegias, and death [1,2]. Demyelination is proposed to occur from injury to endothelial cells causing the release of toxic factors creating edema and subsequent separation of myelin from the axon [7]. Reported cases of demyelization occurred when correction rates exceeded 12 mmol/L/day and have been reported in as little as 9–10 mmol/L/day [1]. Myelinolysis can even occur without hyponatremia within several days after alcohol withdrawal [7]. Sterns reported a study of 62 patients with chronic severe hyponatremia and seven of these patients having neurologic complications after rapid correction (>13 mmol/L/24 hr) [8].

The differential diagnosis for hyponatremia can be vast and includes diabetes insipidus, syndrome of inappropriate antidiuretic hormone, hyperthyroidism, and hypercortisolism. Rarely, hyponatremia is seen with extreme water consumption. Causes of water intoxication commonly result from dilution of bottle formula in infants or psychogenic polydipsia in the setting of schizophrenia [9,10]. Cosgray notes that one possible etiology of water intoxication may

be preexisting alcoholism [11]. Ripley indicates there is a strong correlation between alcoholism and self-induced water intoxication, especially in a hospital setting as water is substituted for alcohol [12]. In addition, the overhydration may induce a pleasurable experience with confusion, delirium, etc [12].

Psychogenic polydipsia may be associated with several psychiatric conditions including psychotic depression, manic-depressive psychosis, and most commonly schizophrenia with up to 18% of patients in mental hospitals displaying polydipsic behavior [3,13,14]. The pathogenesis of the polydipsia may be hypersensitivity to vasopressin, an increase in dopamine activity, or a defect in osmoregulation [3,14–16]. This particular patient was not diagnosed as psychotic, but may have underlying psychotic features and alcohol withdrawal symptoms that led him to water intoxication. Jos reports similar behavior of a psychiatric patient that was witnessed drinking large amounts of water and suffering repeated episodes of seizures and emesis afterwards, but improved unremarkably after treatment [17].

Tonic clonic seizures are the most common presenting feature (up to 80%) of hyponatremia [18]. However, polydipsic patients with seizures rarely show epileptic EEG abnormalities [9]. Our patient exhibited seizures, which subsided with administration of anti-epileptics and correction of his serum sodium. The patient's ongoing treatment for polysubstance and alcohol abuse may have contributed to a lower seizure threshold. The initial CT showed cerebral edema, consistent with a hypotonic state. In addition, our patient had only a mildly abnormal EEG without clear seizure or epileptiform activity. Ligtenberg reports a case of advanced psychogenic polydipsia ending with lethal cerebral edema and herniation. In this case, the serum sodium was 114 mmol/L with a serum osmolality of 245 mOsm/kg, measurements very similar to our patient (serum sodium 113 mmol/L, serum osmolality 240 mmol/kg) [19].

The sodium correction in our patient was unexpectedly rapid, despite the administration of desmopressin with a change of serum sodium of 23 mmol/L in approximately 9.5 hours. The hyponatremia seemed to be acute in nature and even though correction was prompt, no demyelination was observed either symptomatically or on MR imaging.

CONCLUSION

Among the causes of hyponatremia, psychogenic polydipsia may be more difficult to diagnose especially if an apparent

psychiatric condition is not present. Current literature supports cautious correction of hyponatremia to prevent complications including demyelination syndromes. However, rapid corrections may be driven by the physiology of the patient and may not be avoidable. Fortunately, our case illustrates rapid, positive outcomes for the patient.

REFERENCES:

1. Adrogue HJ, Madias NE: Hyponatremia. *N Engl J Med*, 2000; 342(21): 1581–89
2. Nathan BR: Cerebral correlates of hyponatremia. *Neurocrit Care*, 2007; 6(1): 72–78
3. Siegel AJ: Hyponatremia in psychiatric patients: update on evaluation and management. *Harv Rev Psychiatry*, 2008; 16(1): 13–24
4. Andrew RD: Seizure and acute osmotic change: clinical and neurophysiological aspects. *J Neurol Sci*, 1991; 101(1): 7–18
5. Schrier RW, Bansal S: Diagnosis and management of hyponatremia in acute illness. *Curr Opin Crit Care*, 2008; 14(6): 627–34
6. Perianayagam A, Sterns RH, Silver SM et al: DDAVP is effective in preventing and reversing inadvertent overcorrection of hyponatremia. *Clin J Am Soc Nephrol*, 2008; 3: 331–36
7. Yoon B, Shim YS, Chung SW: Central pontine and extrapontine myelinolysis after alcohol withdrawal. *Alcohol Alcohol*, 2008; 43(6): 647–49
8. Sterns RH, Cappuccio JD, Silver SM, Cohen EP: Neurologic sequelae after treatment of severe hyponatremia: a multicenter perspective. *J Am Soc Nephrol*, 1994; 4(8): 1522–30
9. Delva NJ, Crammer JL: Polydipsia in chronic psychiatric patients body weight and plasma sodium. *Br J Psychiatry*, 1988; 152: 242–45
10. Medani CR: Seizures and hypothermia due to dietary water intoxication in infants. *South Med J*, 1987; 80(4): 421–25
11. Cosgray RE, Hanna V, Davidhizar RE, Smith J: The water-intoxicated patient. *Arch Psychiatr Nurs*, 1990; 4(5): 308–12
12. Ripley TL, Millson RC, Koczapski AB: Self-induced water intoxication and alcohol abuse. *Am J Psychiatry*, 1989; 146(1): 102–3
13. Boyd MA: Polydipsia in the chronically mentally ill: a review. *Arch Psychiatr Nurs*, 1990; 4(3): 166–75
14. Zafonte RD, Watanabe TK, Mann NR, Ko DH: Psychogenic polydipsia after traumatic brain injury A case report. *Am J Phys Med Rehabil*, 1997; 76(3): 246–48
15. Ali N, Imbriano LJ, Maesaka JK: The Case | A 66-year-old male with hyponatremia. Psychogenic polydipsia. *Kidney Int*, 2009; 76(2): 233–34
16. Döring WK, Herzenstiel MN, Krampe H et al: Persistent alterations of vasopressin and N-terminal proatrial natriuretic peptide plasma levels in long-term abstinent alcoholics. *Alcohol Clin Exp Res*, 2003; 27(5): 849–61
17. Jos CJ: Generalized seizures from self-induced water intoxication. *Psychosomatics*, 1984; 25(2): 153–57
18. Ferrier IN: Water intoxication in patients with psychiatric illness. *Br Med J (Clin Res Ed)*, 1985; 291(6509): 1594–96
19. Ligtenberg JJ, Wymenga AN, Tulleken JE et al: A lethal complication of psychogenic polydipsia: cerebral edema and herniation. *Intensive Care Med*, 1998; 24(6): 644–45