Beer Potomania

Introduction

- Beer potomania is described as excessive alcohol intake coupled with poor dietary solute intake that results in reduced solute delivery to the kidneys
- This reduced solute delivery to the kidneys leads to a decrease in the excretory capacity of the kidneys and can cause dilutional hyponatremia, or beer potomania
- Beer potomania was first reported in 1972

Pathophysiology

- Free-water clearance is dependent on solute excretion and urinary diluting capability
- Based on a normal diet, typical osmole excretion is ~600-900 mOsm/day, and >20L of water must be ingested to overwhelm the capacity of urinary dilution
- In patients with a history of beer drinking, poor diet leads to very low osmole intake, and decreased or diminished urea and ketone generation
- The net effect of heavy beer drinking is an excess of solvent (basically free water) and a lack of solute for diuresis
- With a decrease in solute excretion, the ability to excrete free water becomes limited, and much less volumes of water intake are enough to overwhelm the capacity of urinary dilution, leading to water retention and hyponatremia
- ADH levels are also suppressed in these patients due to excess of water
- When solute is reintroduced to the patient, brisk diuresis can be expected (even after just a single liter of NS) due to the low levels of ADH and can produce large increases in serum sodium levels in a short period of time

Osmotic Demyelination Syndrome (ODS)

- A rare but potentially devastating complication of hyponatremia that can lead to permanent neurologic injury
- In patients with hyponatremia, cellular swelling occurs because the fluid shifts from the hypotonic extracellular space to the relative hypertonic intracellular space
- In the brain space, this can lead to cerebral herniation
- In response to decreased intracellular tonicity, compensatory mechanisms involving intracellular solute and water losses begin to develop to allow for cerebral adaptation
- With rapid correction of hyponatremia, the brain cells cannot recapture lost osmolytes as quickly, and water from within brain cells shifts extracellularly, causing dehydration and demyelination of white matter
- Symptoms include dyspnea, dysphagia, dysarthria, and ataxia

Treatment

- Patients who present with beer potomania are at greater risk of developing ODS given the degree and chronicity of hyponatremia, their alcohol use, and their underlying pathophysiological state
- If the patient is asymptomatic, fluid restriction and monitoring is a recommended approach in the literature
- If the patient is symptomatic, finite amounts of IVFs (i.e., 500 mL of NS) is a recommended approach in the literature
- Every 2 hour serum sodium levels are recommended
- Treatment goals: serum sodium increase <10 mmol/L in the first 24 hours and <18 mmol/L in the first 48 hours
- If desired goal is exceeded, D5W infusion can be started at a rate equal to urine output, and adjusted based on sodium levels every 2 hours

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Overview of Evidence

Author; year	Patient Case	Intervention	Outcome
Uzair Lodhi M, et al; 2017 **Case 1**	59 year-old male with alcohol use disorder for 25 years presents s/p seizure Serum NA = 118 mmol/L Urine osmolality = 72 mOsm/kg H2O Urine NA = 19 mmol/L	1 L of 0.9% sodium chloride	16 hours after presentation: -Brisk diuresis of 3 L -Serum NA = 129 mmol/L (increase of 11 mmol/L in 16 hours) -D5W initiated 24 hours after presentation: -Serum NA = 127 mmol/L (increase of 9 mmol/L in 24 hours) -D5W infusion adjusted to maintain serum NA between 131-133 mmol/L for the next few days
Uzair Lodhi M, et al; 2017 **Case 2**	60 year-old male with alcohol use disorder presents with weakness, dizziness, and lack of appetite Serum NA = 106 mmol/L Urine osmolality = 159 mOsm/kg Urine NA = 19 mmol/L	1 L of 0.9% sodium chloride and two big meals in the emergency room	16 hours after presentation: -Brisk diuresis of unmeasured amount -Serum NA = 119 mmol/L (increase of 13 mmol/L in 16 hours) -NS-based IV fluids were discontinued, however serum NA continued to increase to 128 mmol/L in the first 32 hours -D5W infusion initiated, and serum NA was 121 mmol/L after 12 hours
Rafei H, et al; 2016	66 year-old male with alcoholism presents with tremors of upper and lower extremities Serum NA = 122 mmol/L	Fluid restriction, adequate nutritional supplementation and withholding of diuretics IV fluids were withheld given lack of symptoms	3 days later: Gradual increase in serum NA Serum NA = 130 mmol/L
Kujubu D, et al; 2015	84 year-old male with chronic alcohol abuse presents with nausea, weakness, decreased appetite, and abdominal pain Serum NA = 116 mEq/L	1 L of 0.9% sodium chloride and encouraged to increase oral intake	8 hours after presentation: -Brisk diuresis of 1800 mL -Q2-3H serum NA levels 24 hours after administration: -Serum NA = 124 mEq/L (increase of 8 mEq/L) 48 hours after administration: -Serum NA = 130 mEq/L (increase of 14 mEq/L) Remainder of hospital stay: -Serum NA remained between 130-133 mEq/L and no change in neurologic status
Bhattarai N, et al; 2010	52 year-old male with chronic alcohol abuse presents with drowsiness, lethargy, and change in mental status for 3-4 days Serum NA = 107 mmol/L	1 L of 0.9% sodium chloride and 30 mL of 3% sodium chloride	24 hours after administration: -Brisk diuresis of 6 L -Serum NA = 128 mmol/L (increase of 21 mEq/L in 24 hours) -D5W initiated to match urine output

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Sanghvi S, et al; 2007 **Case 1**	39 year-old female with long-standing alcohol use presents with increasing confusion Serum NA = 100 mEq/L	1 L of 0.9% sodium chloride	5 hours after presentation: -Urine output of 500 mL/hour -Serum NA went from 100 mEq/L to 106 mEq/L -D5W initiated at a rate to match urine output 24 hours after presentation: -Urine output of 7600 mL -Serum NA = 115 mEq/L (increase of 15 mEq/L in first 24 hours) 48 hours after presentation: -Urine output of 4300 mL -Serum NA = 124 mEq/L (increase of 24 mEq/L in first 48 hours) Day 7: level of alertness started to decrease Day 9: MRI findings consistent with pontine and extrapontine myelinolysis
Sanghvi S, et al; 2007 **Case 2**	63-year-old male with alcoholism presents with weakness and dizziness Serum NA = 104 mEq/L	Fluid intake was restricted to 1 L and received a dose of moxifloxacin in 0.9% NS solution	4 hours after presentation: -Brisk diuresis of 3000 mL 9 hours after presentation: -Serum NA = 111 mEq/L (increase of 7 mEq/L) -D5W initiated at a rate to match urine output 24 hours after presentation: -Urine output of 6500 mL -Serum NA = 111 mEq/L (increase of 7 mEq/L) 48 hours after presentation: -Serum NA = 118 mEq/L (increase of 14 mEq/L) Day 6: discharged with no clinical evidence of neurologic sequelae

Conclusions

- Beer potomania in a severely hyponatremic alcoholic patient is important to recognize and treat accordingly to avoid overcorrection of sodium in these patients
- ODS is a permanent neurologic injury that can occur with rapid correction of sodium
- There is no clear-cut consensus of sodium replacement in beer potomania, but this should be done judiciously to prevent ODS
- While evidence is limited to case reports, brisk diuresis and rapid correction/over-correction of serum sodium levels has been seen after just a single liter of 0.9% NS, and D5W solution is often needed to prevent over-correction and ODS
- The principle of beer potomania should be kept in mind when replacing sodium in chronic alcoholics

References

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