Beer Potomania:

A Case Study

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Introduction

The following case study will discuss the nutrition implications of beer potomania, as well as the patient's medical history, treatment, and progress.

Patient Profile and Social History

MT was a 63 year old female with history of tobacco use, and alcohol abuse. MT had been drinking a 12-pack of beer daily until a few days prior to admit when she reportedly decreased her consumption to forty ounces daily.

Medical History

MT was admitted to the Intensive Care Unit at McKay Dee Hospital on November 8, 2012 with hyponatremia, hypokalemia, hypophosphatemia, hypomagnesemia, and acute metabolic encephalopathy after experiencing nausea, vomiting, decreased appetite, and being found confused and unstable with her gait and with a serum sodium level of 101. MT presented with a medical history significant for hypertension, major depressive behavior, and alcohol abuse. MT had no previous significant surgical history.

Beer Potomania

Beer potomania is a condition describing an individual who is experiencing a combination of low serum sodium concentration (<134mEq/L), low daily solute consumption and excretion, and excessive alcohol ingestion (1,2,3). Reports of Low serum sodium, or hyponatremia, in alcoholics were first reported in the early 1970s (1). Research has shown that nearly 17% of all chronic drinkers experience hyponatremia, but only a portion of that percentage experience extreme hyponatremia (<120mEq/L) with clinical presentation requiring hospitalization(1). Common clinical indicators of severe hyponatremia include confusion, seizures, loss of consciousness, aspiration, falls, fractures, and loss of gait (1,2).

Hyponatremia occurs in chronic excessive alcohol drinkers when their fluid intake exceeds their body's excretory capacity (1). A body's ability to excrete free-water depends on the amount of solute excreted and the body's ability to dilute its urine (1). For an individual with a normal healthful diet, typical osmole excretion ranges from 600-900mOsm daily (1). One's osmolar load, and excretion, is determined by urea generated from protein, sodium (Na), and potassium (K) intake. In short, a diet composed of very little sodium and protein(which are urea precursors) inhibits the body's ability to excrete urinary solutes. When solute excretion is limited, one's ability to excrete free water is limited which leads to an increase in the dilution of sodium in the body ultimately leading to hyponatremia (1,3).

Another contributing factor to hyponatremia in beer potomania is the over secretion of anti-diuretic hormone (ADH) (3). Both hypovolaemia and hypo-osmolality (common in alcoholics) stimulate ADH secretion which results in water retention that further contributes to hyponatremia (3).

The human brain responds to hyponatremia and attempts to limit brain swelling by shedding organic osmolytes to allow osmotic equilibrium between extracellular and intracellular fluids without an increase of cell water (2). This adaptation puts the brain at risk for injury and damage upon sodium and electrolyte repletion (2).

Both when serum sodium reaches extreme low levels, and when or if it is restored too quickly in a chronic hyponatremic individual, that individual may experience osmotic demyelination syndrome (ODS) (2). Osmotic demyelination syndrome results when the brain gets dehydrated leading to the dehydration of oligodendrocytes and cell death (4). Individuals experiencing osmotic demyelination syndrome may experience dysphagia, oculomotor alterations, quadriplegia, apathy, emotional lability, delusions, memory loss, or decreased attention span (5).

It is critical that ODS symptoms be identified and treated promptly because it is possible for the demyelination not to appear on an MRI for days to weeks after the ODS incidence (5). In the event that

osmotic demyelination occurs, rapid re-lowering of serum sodium has been associated with a decrease in brain damage and possible restoration of neurological deficits (1,2,4).

Generally, dangerously low serum sodium levels are considered to be levels less than 120mEq/L (1,2). Many studies have concluded that safest repletion rates are approximately 4-6mEq/L daily. It is recommended that repletion does not exceed 10mEq/L within a 24 hour time period, and 18mEq/L within 48 hours (1,2). However, some individuals have experienced osmotic demyelination syndrome even when their repletion rates are kept within the recommended ranges (2). Commonly, individuals that present with hyponatremia experience excessive diuresis upon admit related to elimination of vasopressin secretions. This can lead to a rapid, and unsafe elevation of serum sodium levels (2). In order to keep up with unpredictable excessive fluid losses, either D5W or Desmopressin is administered to aid in serum sodium control. In cases where serum sodium has been increased too quickly, D5W or Desmopressin may also be administered to restore sodium levels to a lower concentration (1,2).

In conclusion, beer potomania is a serious condition defined by hyponatremia that when severe enough, is accompanied by clinical indications such as loss of consciousness, altered mental status, seizures, loss of gait, and even death. If serum sodium is not replaced cautiously, osmotic demyelination may occur causing severe brain damage. It is crucial that the entire medical team is aware of the signs and symptoms of osmotic demyelination syndrome, so that it can be caught and corrected before permanent brain damage is done.

Treatment and Progress

Upon admission, MT did not require intubation, and was tolerating two liters of nasal cannula oxygenation. MT's serum sodium(Na) levels were checked every two hours as D5W dextrose solution and normal saline were administered at a very low rate to control a slow repletion

	11/8	11/9	11/10	11/11	11/12	11/13	
Test							**Normal**
Gluc	111 H	123 H	145 H	158 H	141 H	134 H	65-99
Albumin	4.4	3.3					3.3-4.8
Hct	52 H	49 H	47 H	50 H		39.3	36-46
Hgb	16.2 H	15.6				14.3	12-16
Na	101	101	110	113	117	119	136-147
K	2.5 L	4.0	3.8	3.2 L	4.2	3.6	3.5-5.0
Mg	1.3 L	2.7 H	2.7 H	1.9	1.9	1.9	1.6-2.3
Cl	50 L	66 L	71 L	74 L	79 L	85 L	98-109
WBC	15 H	15.6 H	14.3 H	13.1 H		12.1 H	3.6-10.6

MT's blood glucose levels were likely elevated due to stress. Albumin remained normal which was likely not an adequate marker for protein stores given the pt's diet history. MT's hematocrit and hemoglobin were elevated upon admission possibly due to dehydration. Sodium, potassium, magnesium, and chloride were all low upon admission due to alcoholism. Sodium remained low but increased at an appropriate rate according to hyponatremia repletion guidelines. MT's white blood cells were elevated during admission due to the critical state of being.

On November 9, 2012 MT was still confused, but able to communicate with medical staff. MT progressed to room air by November 11, 2012, and was able to sit up in a chair, and walk around by November 13, 2012. At this point MT's mental status was improving.

Normal saline and D5W dextrose solution were administered intravenously to provide slow and controlled serum sodium repletion and also maintenance fluids. A nicotine patch was administered to

minimize withdrawal symptoms and cigarette cravings. Novolog was also prescribed as needed if medicinal blood glucose control was needed.

MT's admit weight was 59.9 kilograms, and admit height was 157.5 centimeters equating to a body mass index (BMI) of 24.8 which is within the appropriate healthy BMI range for age. Clinically, MT appeared to have muscle wasting. MT's limbs were thin but her abdomen was a bit thicker, which is common among alcohol abusers. In addition, MT's hair looked frail and dark circles were present around her eyes likely due to sub-optimal nutrition previous to admittance. MT was admitted at a moderate nutritional risk level due to her poor intake prior to admit, her report of nausea and vomiting prior to admit, and diet order of nothing by mouth (nothing per os, NPO) related to severe hyponatremia. Although MT was admitted with an appropriate BMI of 24.8, pt was at risk for malnutrition due to history of alcoholism, and poor intake prior to admit.

According to McKay Dee protocol, for a patient that has a healthy BMI between 18.5 and 24.9, actual body weight is used to calculate dietary needs. The actual body weight in kilograms (kgs) is multiplied by 25 and 30 kilo-calories (kcals) to estimate an appropriate calorie range. Because MT had no wounds, infection, or other indications for increased protein needs, protein needs were calculated by multiplying actual body weight in kgs by 0.8 and 1.0 according to McKay Dee protocol. Based on these guidelines, MT's needs were 1550-1850 kilo-calories (kcals), 49-62grams (g) of protein, and 7.8-9.9g of nitrogen daily. Based on MT's actual body weight, 1550-1850 milliliters (ml) of fluids were recommended to provide one ml of fluid for every kcal given.

Upon admit, MT was written a nothing by mouth diet order. On November 10, 2012 MT was written a full liquid diet but shortly thereafter changed back to NPO on November 11, 2012.

MT's weight history was significant for slight weight fluctuations from two to six kgs during her stay at McKay Dee. The Pt weighed 61.5kgs upon admit with a BMI of 24.8 which is within the healthy BMI range but had a decreased weight recorded of 55.1kgs on day three and an increased weight

recorded of 59.5kgs two days later. These weight fluctuations could be due to fluid status, NPO diet order, or error of the bed scale used.

MT was admitted with low serum levels of Magnesium, Phosphorus, Potassium, and Sodium due to poor intake by mouth (PO) prior to admit, and alcoholism. Glucose and white blood cells were elevated due to MT's stressed state. MT's albumin was within normal limits (WNL) upon admit which is likely not a good indicator of nutrition status.

Patient diet history was unobtainable because the patient could not recall the last time food was eaten. The patient stated typically only dinner is eaten with sporadic snacking throughout the day.

Summary and Conclusions

MT's sodium levels were slowly brought back up to the 130s, at which point the diet was advanced as tolerated. MT progressed to a regular diet and was tolerating it with no problems upon follow up assessment. Prognosis was good in regards to a full recovery, but due to the history of alcohol abuse, there is a possibility of reoccurrence.

Nutrition Notes

11/13/2012

Pt at risk for malnutrition. Current diet order NPO appropriate. Regular diet once pt awakes and diet advances. If PO <50%, consider nutrition support. Admit alb WNL BGs 130s-160s. Stool x1 on 11/11. Skin is intact.

PES Statements

11/13

- Inadequate oral intake related to decreased ability to consume sufficient energy, nutrients as evidenced by NPO diet order, and hyponatremia.
- Impaired nutrient utilization related to alcohol addiction as evidence by hyponatremia, hypokalemia, hypomagnesemia, and hypophosphatemia.

Goals

11/13

- Safe, slow repletion of serum Na
- Diet advancement as appropriate

Interventions

11/13

- Thiamine and Folate supplementation

Nutritional Recommendations

11/13

- Once Pt awakes, if PO is <50%, consider nutrition support.

Caregiver notes:

11/9 ETOH abuse, N/V with decreased PO PTA. Admit with Na 101, protecting airway. No vent, obtunded, NPO, BG WNL, no stool yet, skin intact. Receiving thiamine and folate

11/10 Diet advance to fulls, slowly correcting Na.

11/12 Na still coming up slowly with D5, still somewhat confused, BG controlled. Pt has been NPO to avoid any extra Na sources. Last BM on 11/11.

11/13 Pt remains NPO, Na continues to rise slowly.

References:

1.Sanghvi SR, Kellerman PS, Nanovic L. *Beer Potomania: An Unusual Cause of Hyponatremia at High Risk of Complications From Rapid Correction.* American Journal of Kidney Diseases. 2007; 50:673-680.

2. Sterns RH, Hix JK, Silver S. *Treating Profound Hyponatremia: A Strategy for Controlled Correction.* American Journal of Kidney Diseases. 2010; 56: 774-779.

3. Liamis GL, Milionis HJ, Rizos EC, Siamopoulos KC, Elisaf MS. *Mechanisms of Hyponatremia in Alcohol Patients.* Alcohol & Alcoholism. 2000; 35: 612-616.

4. Hassan I, Shing C, Bajraszewski CE, Gleason A, Hayhow BD, Velakoulis D. *Osmotic demyelination syndrome: An under-recognised cause of delirium.* Australian & New Zealand Journal of Psychiatry. 2012; 00(0):1-2.

5. Yamada H, Takano K, Ayuzawa N, Seki G, Fujita T. *Relowering of Serum Na for Osmotic Demyelinating Syndrome.* Case Reports in Neurological Medicine. 2012; Article ID 704639:1-2.