



Severe Symptomatic Hyponatremia Secondary to Excessive Consumption of Hard Seltzer: Mehandru/Vachharajani Syndrome

Sushil K. Mehandru^{1*}
Supreet Kaur^{1*}
Avais Masud¹
Islam Elkherpitawy¹
Arif Asif¹
Tushar J. Vachharajani²

¹Department of Medicine, Division of Nephrology & Hypertension, Jersey Shore University Medical Center, Hackensack Meridian School of Medicine, Neptune, New Jersey, USA

²Division of Nephrology and Hypertension, Cleveland Clinic, Cleveland Ohio, USA

Abstract

Alcoholic carbonated drinks have taken up a large share of the beer industry, particularly among the young adults. The consumption of hard seltzer has grown exponentially over the past few years. When consumed in large quantities, hard seltzer may result in severe symptomatic hyponatremia. This is a first study reported to the best of our knowledge including three cases with severe hyponatremia caused by the consumption of large amounts of hard seltzer. The patients reported here, presented to the emergency department (ED) with seizures and serum sodium (PNa) of 96-112 mEq/L. The patients consumed on average two or more 6-packs of hard seltzer per day and more over the weekends. The alcohol content was noted at 5% with minimal solute in these beverages. More cases can be expected to emerge in the future with severe electrolyte imbalance and dilutional hyponatremia, if people are not educated about appropriate consumption of hard seltzer drinks. The availability of these drinks in the market in different fruity flavors and low calorie count makes it even more enticing for the young, leading to excessive consumption.

KeyWords: Hyponatremia; Beer potomania; Electrolyte disturbance; Urinary dilution; Serum sodium concentration; Urine sodium; Hard seltzer

Introduction

Hyponatremia is one of the most common diagnoses in the hospital or the office practice for the nephrologist. When defined as plasma sodium concentration <135 mEq/L, the prevalence of hyponatremia in hospitalized patients may be as high as 15 to 30% [1]. This can be encountered in dilutional hyponatremia when poor dietary consumption is noted along with increased consumption of low solute drinks. It is commonly seen in patients with alcohol abuse. Hyponatremia patients present with multitude of sign and symptoms, ranging from asymptomatic to severe, with sometimes life threatening conditions. The factors that lead to this occurrence can range from medication induced, excessive water consumption, or underconsumption of sodium containing foods. Being one of the most common electrolytes disturbances, especially in alcoholic patients, the underlying process and subsequent correction is crucial in patients with hyponatremia. Individuals consuming alcoholic beverages especially those with low sodium content are most affected by this. Severity of hyponatremia along with duration helps govern the pace of correction while considering prevention of osmotic demyelination syndrome, that is caused by rapid correction. Hard seltzer sales are increasing exponentially adding to the concern of increase in hyponatremia cases. The category is keeping its monster-growth pace, leading to several brands launching new spiked seltzers at a rapid pace.

Causes of hyponatremia

Excessive water intake can cause hyponatremia by overwhelming normal water excretory capacity (primary hyponatremia) Table 1 [2]. Most common causes of severe hyponatremia in adults are thiazides, use of Selective Serotonin Reuptake Inhibitors (SSRI), syndrome of inappropriate antidiuretic hormone (SIADH), polydipsia in psychiatric patients, and gastrointestinal fluid losses. Water retention and hyponatremia usually occurs with impaired renal function that was not noted in our patients. The renal functions of all our patients were normal. The hyponatremic state, hypotonic hyponatremia related to excess intake of free water; this was solely a result of over consumption of hard seltzer; a low solute beverage. The Edelman equation established that hypotonic (or dilutional) hyponatremia represents an excess of water relative to the sodium potassium store (Figure 1) [3].

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*Corresponding author:

Sushil K. Mehandru

Professor of Medicine

Hackensack Meridian School of Medicine

Chief, Division of Nephrology and Hypertension

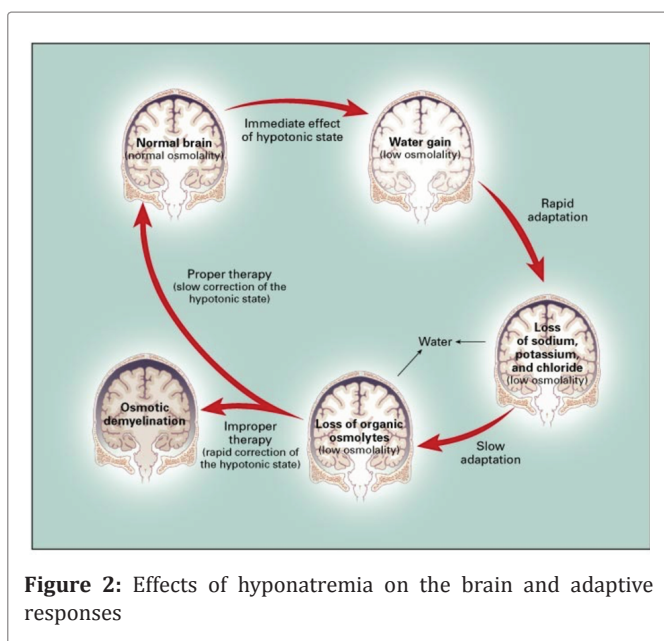
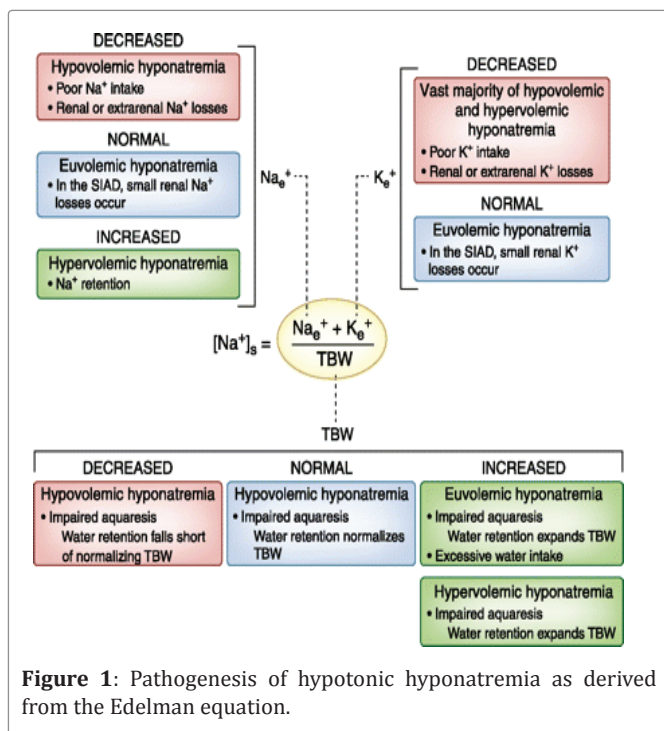
Jersey Shore University Medical Center

1945 State Route 33, Neptune NJ

E-mail: doc1732@aol.com

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Hypotonic hyponatremia represents an excess of water relative to the body's sodium and potassium stores. In that context, patients with hypotonic hyponatremia can feature decreased, normal, or increased Na_e^+ ; decreased or normal K_e^+ ; and decreased, normal, or increased TBW. Na_e^+ , exchangeable sodium; K_e^+ , exchangeable potassium; TBW, total body water [3].

Hypotonic hyponatremia causes entry of water into the brain, resulting in cerebral edema. This results in headaches, nausea, vomiting, restlessness, disorientation, depressed reflexes, seizures, coma, permanent brain damage, respiratory arrest, brain stem herniation, and death [2] (Figure 2).

Because the surrounding cranium limits expansion of the brain, intracranial hypertension develops, with an increased risk of brain injury. Fortunately, solutes leave the brain within hours, inducing a water loss and ameliorating brain swelling [4,5]. This process of adaptation by the brain accounts for the relative asymptomatic nature of severe hyponatremia if it develops slowly. Nevertheless,

brain adaptation is also a source of risk of osmotic demyelination syndrome [6-8]. As shown in Figure 2, hyponatremia effects brain cell dysfunction [2]. Although rare, osmotic demyelination is serious and can develop one to several days after aggressive treatment of hyponatremia by any method, including water retention alone [9-11]. Causes of hypo-osmolality syndrome in beer drinkers was first described by Gwinup et al in 1972 [12]. Beer potomania was described in 1986 by Joyce et al., resulting in severe hyponatremia in patients after consumption of large amounts of beer [13]. Hypokalemia has also been reported in many cases of hyponatremia with potassium levels as low as 1.3 mEq and PNa 105, these patients presented with weakness, seizures, and coma [14].

Mechanism of hyponatremia in consumption of low solute drinks

In studies done in excessive beer intake resulting in hyponatremia, poor solute intake has been implicated in hyponatremia seen in beer potomania [15]. In these cases and in cases presented in our publication, pathophysiology of hyponatremia is based upon consumption of poor solute drinks such as hard seltzer, beer, etc., by the concept of solute free-water clearance in the kidney. Specifically, low solute intake reduces urinary excretion of osmoles, thereby capping a ceiling on the renal capacity of free-water excretion. A positive water balance follows an excess of water intake, causing dilutional hyponatremia [15]. A person with normal renal functions and normal dietary intake removes about 600-900 mOsm/day [16]. With maximum urinary dilution of 50 mOsm/L, a person can excrete about 20 liters of water without becoming hyponatremic, allowing for a broad range of water intake (reaching up to 20 L) [17-19]. As free-water clearance in a person with normal diluting capacity is dependent on osmole excretion, a decrease in daily dietary osmole intake can have a vast decrease in the excretory capacity of the kidney. Therefore, this decrease in daily dietary osmoles in even minute fluid excess can cause dilutional hyponatremia. Beer potomania patients have a long history of beer intake, as well as poor dietary patterns. Similar phenomena, we assume occurs in our cases presented here in. Hard seltzer drinks result in severe hyponatremia. There is a definite lack of publications regarding hard seltzer and its effects on electrolyte balance. Trace amounts of sodium in hard seltzer as in beer can be 14-20 mg/12 Fl Oz can and almost no protein content. In addition, beer has some calories that prevent muscular proteolysis resulting in dramatic decrease in urea generation. Thus, these patients have very low osmolar load as dietary protein breakdown is the main component of osmolar load, as well as small amounts from sodium and potassium.

In beer potomania patients as we assume in hard seltzer cases, the low ADH levels limit free-water reuptake in the collecting tubules of the kidney and explain why these patients have brisk diuresis when solute is presented [20].

Hyponatremia is among the common electrolyte abnormality observed in chronic alcoholic patients. In one study, hyponatremia was the third commonest electrolyte abnormality (17.3%) [21]. However, the prevalence of hyponatremia and its underlying pathophysiological mechanisms in these patients is not well delineated [22-24]. Effective volume depletion predisposes towards the development of hyponatremia by its effects on renal water excretion and thirst. In fact, hypovolemia is a potent stimulus to ADH secretion, resulting in water retention. Volume depletion can directly or indirectly stimulate thirst and consequently increase water intake [25]. Alcoholic patients are often malnourished, binge drinkers can present with profound hyponatremia, together with a minimal intake of adequate nourishment.

Case Studies

Case Study 1

A 23 year old Caucasian female with unremarkable primary health history, presented to the ED after a severe shaking episode

IMPAIRED CAPACITY OF RENAL WATER EXCRETION

Decreased volume of extracellular fluid

Renal sodium loss
 Diuretic agents
 "Osmotic diuresis urea (glucose, urea, mannitol)"
 Adrenal insufficiency
 Salt-wasting nephropathy
 Bicarbonaturia (renal tubular acidosis, disequilibrium stage of vomiting),
 Ketonuria
Extrarenal sodium loss
 Diarrhea
 Vomiting
 Blood loss
 Excessive sweating (e.g., in marathon runners)
 Fluid sequestration in "third space"
 Bowel obstruction
 Peritonitis
 Pancreatitis
 Muscle trauma
 Burns

Increased volume of extracellular fluid

Congestive heart failure
 Cirrhosis
 Nephrotic syndrome
 Renal failure (acute or chronic)
 Pregnancy

Essentially normal volume of extracellular fluid

Thiazide diuretics*
 Hypothyroidism
 Adrenal insufficiency
 Syndrome of inappropriate secretion of antidiuretic hormone
Cancer
 Pulmonary tumors
 Mediastinal tumors
 Extrathoracic tumors
Central nervous system disorders
 Acute psychosis
 Mass lesions
 Inflammatory and demyelinating diseases
 Stroke
 Hemorrhage
 Trauma
Drugs
 Desmopressin
 Oxytocin
 Prostaglandin-synthesis inhibitors
 Nicotine
 Phenothiazines
 Tricyclics
 Serotonin-reuptake inhibitors
 Opiate derivatives
 Chlorpropamide
 Clofibrate
 Carbamazepine
 Cyclophosphamide
 Vincristine
Pulmonary conditions
 Infections
 Acute respiratory failure
 Positive-pressure ventilation
Miscellaneous
 Post operative state
 pain
 Severe nausea
 Infection with the human immunodeficiency virus
Decreased intake of solutes
 Beer potomania
 Tea-and-toast diet

EXCESSIVE WATER INTAKE

Primary Polydipsia†
 Dilute infant formula
 Sodium-free irrigant solutions (used in hysteroscopy, laparoscopy, or transurethral resection of the prostate)‡
 Accidental intake of large amounts of water (e.g., during swimming lessons)
 Multiple tap water enemas

*Sodium depletion, potassium depletion, stimulation of thirst, and impaired urinary dilution are implicated.

†Often a mild reduction in the capacity for water excretion is also present.

‡Hyponatremia is not always hypotonic.

Table 1: Causes of hypotonic hyponatremia

followed by confusion. Patient started to have abdominal pain two days prior to presentation. Pain was mild and associated with nausea. Two days prior to admission, basic metabolic panel (BMP) was done and showed serum sodium of 129 mEq/L. Patient felt more tired and then shortly before presentation developed a tonic clonic seizure that lasted for one minute. There was no past history of seizure disorder. Patient was confused after a few minutes. She was brought to the emergency department and was completely oriented on presentation.

Patient reported mild fatigue and nausea for a few days prior

to ED visit. She did not report any vomiting, diarrhea, constipation, decreased water intake, severe dehydration, palpitation, dizziness, excessive exercise, or exposure to hot weather recently.

Patient denied any excessive water intake or beer consumption. However, Patient reported drinking six 12 oz cans of hard seltzer beverage on nightly basis and doubled her consumption on weekends.

No past medical or surgical history was reported. No use of thiazide diuretics or SSRIs was reported.

Patient denies smoking or drinking beer, however, she reported frequent consumption of hard seltzer beverages.

Patient does not have any allergies and does not take any medications.

Vitals: heart rate 101 per minute, blood pressure 120/82 mm Hg, respiratory rate 16 per minute, pulse oximetry 96% on Room air.

Physical Exam: Patient's physical exam was unremarkable. Patient was alert, awake, oriented x3, normocephalic/atraumatic, lungs were clear to auscultation, heart sounds normal s1 and s2 without murmur, abdomen was noted soft, non-tender, not distended. Neurological exam revealed no cranial nerve deficit, no focal sensory or motor deficits.

Laboratory and radiological data: Patient laboratory data included hemoglobin 15 g/dl, hematocrit 39.7 %, white blood cell count (WBC) 9.8 uL, platelets 287 uL, sodium 107 mEq/L, chloride 68 mEq/L, potassium 3.1mEq/L, blood urea nitrogen (BUN) 7 mg/dl, creatinine 1.08 mg/dl, magnesium 2.4 mg/dl, Phosphorus 3.3 mg/dl, creatinine kinase (CK) 224 iU/L, HCG qualitative negative, thyroid stimulating hormone (TSH) 0.988 IU/ml, albumin 4.5 g/dl, serum osmolality 230 mOsm/kg, urine osmolality 128 mOsm/kg, and urine sodium < 10 mmol/l.

Computed tomography (CT) scan of the head without contrast was negative for acute changes.

Treatment: Patient was diagnosed with severe hyponatremia resulting in a seizure episode. Based on the history, serum osmolality, low potassium and chloride, hyponatremia was assumed to be secondary to excessive consumption of hard seltzer beverages. Patient was admitted to the intensive care unit and was started on fluid restriction. She was also given potassium chloride for hypokalemia. Serum sodium improved to 111 mEq/L two hours post admission. Patient was not started on 3% saline to prevent over correction. Patient was continued on fluid restriction, the sodium improved to 132 mEq/L before discharge in 4 days. Patient did not have any neurological sequelae on follow up.

Second admission, May 2020: Patient presented two months post initial admission for abdominal pain, nausea, and vomiting with low serum sodium, 119 mEq/L. Urine osmolality was 136 mOsm/kg, urine sodium on this admission was < 10 mmol/L, and serum potassium was 3.2 mEq/L. Patient had no neurological event during this admission. Despite extensive counseling during the initial admission, patient continued consumption of 6 pack hard seltzer daily. Patient was placed on strict fluid restriction. She was subsequently discharged in three days with plasma sodium at 137 mEq/L and resolution of all presenting symptoms.

Case Study 2

A 59 year old African American male with past medical history of hypertension and alcohol abuse, with frequent hospitalizations for hyponatremia related to hard seltzer binge drinking episodes. Patient presented to the ED for generalized weakness and seizure. There was no past history of seizure disorder. He also presented with abdominal pain and nausea. Patient exhibited intermittent confusion in the ED. He reported mild weakness and fatigue that was initially exertional but becoming functional. He did not report any vomiting, diarrhea, constipation, change in water intake, severe dehydration, palpitation, dizziness, or excessive exercise.

Patient denied any excessive water intake or beer consumption. He reported drinking hard seltzer daily with varying degrees from 6-8 cans a night to more than 14 cans.

Past medical history included hypertension and alcohol abuse.

No surgical history was reported.

Patient reports drinking beer in the past, however, now drinks hard seltzer consistently for the past few years.

Patient does not have any allergies and takes Lisinopril 5mg daily, no thiazide diuretics or SSRI use was reported.

Vitals: heart rate 67 per minute, blood pressure 129/76 mm Hg, respiratory rate 18 per minute, pulse oximetry 96% on Room air.

Physical exam: Patient's physical exam was unremarkable. Patient was alert and awake with intermittent periods of confusion, normocephalic/atraumatic, lungs were clear to auscultation, heart sounds normal s1 and s2 no murmur, abdomen was noted soft, non-tender, not distended. Neurological exam revealed no cranial nerve deficit, no focal sensory or motor deficits.

Laboratory and radiological data: Patient laboratory data included hemoglobin 13.1 g/dl, hematocrit 36.6 %, white blood cell count (WBC) 6.2 uL, platelets 198 uL, sodium 112 mEq/L, chloride 72 mEq/L, potassium 3.8 mEq/L, blood urea nitrogen (BUN) 11 mg/dl, creatinine 0.95 mg/dl, magnesium 2.5 mg/dl, Phosphorus 3.2 mg/dl, creatinine kinase (CK) 180 iU/L, HCG qualitative negative, thyroid stimulating hormone (TSH) 1.21 IU/ml, albumin 3.9 g/dl, serum osmolality 232 mOsm/kg, urine osmolality 116 mOsm/kg, and urine sodium < 10 mmol/l.

Computed tomography (CT) scan of the head without contrast was negative for acute changes.

Treatment: Patient was diagnosed with severe hyponatremia with the complication of seizure. Based on the history, serum osmolality, hyponatremia was assumed to be secondary to excessive consumption of hard seltzer. Patient was admitted to the hospital for management of hyponatremia. Patient was also placed on fluid restriction. Serum sodium improved to 118 mEq/l the day after admission; patient was not started on 3% saline to prevent over correction. Plasma sodium started to improve slowly with strict fluid restriction. Patient's plasma sodium improved to 138 mEq/L before discharge. Patient did not have any neurological sequelae on follow up.

Case Study 3

A 24 year old Caucasian male with past medical history of hypertension, diabetes mellitus, and alcohol abuse presented to ED for severe weakness, nausea, and seizures. He also complained of abdominal pain on admission. Patient reported weakness that started 2 days prior to ED visit. He did not report any vomiting, diarrhea, constipation, change in water intake, severe dehydration, palpitation, dizziness, excessive exercise, or exposure to extreme heat. No prior history of seizures was reported. Patient denied any excessive water intake or beer consumption. He reported drinking hard seltzer daily, 2 six packs and more on the weekends.

Past medical history included hypertension, diabetes mellitus, and alcohol abuse.

No surgical history was reported.

Patient reports drinking beer heavily in the past but now drinks hard seltzer.

Patient does not have any allergies and was not taking any medications. No use of thiazide diuretics or SSRIs was reported

Vitals: heart rate 72 beat per minute, blood pressure 110/74 mm Hg, respiratory rate 16 breaths per minute, pulse oximetry 98% on Room air.

Physical exam: Patient's physical exam was unremarkable. Patient was alert and awake with confusion, normocephalic atraumatic, lungs were clear to auscultation, heart sounds normal s1 and s2 no murmur, abdomen was noted soft, non-tender, not distended. Neurological exam revealed no cranial nerve deficit, no focal sensory or motor deficits, 2+ reflexes in all extremities and normal tone.

Laboratory and radiological data: Patient laboratory data included hemoglobin 15.2 g/dl, hematocrit 43.6 %, white blood cell

Nutrition Facts	
8 servings per container	
Serving size	2/3 cup (55g)
Amount per 2/3 cup	
Calories	230
% DV*	
12%	Total Fat 8g
5%	Saturated Fat 1g
	Trans Fat 0g
0%	Cholesterol 0mg
7%	Sodium 160mg
12%	Total Carbs 37g
14%	Dietary Fiber 4g
	Sugars 1g
	Added Sugars 0g
	Protein 3g
10%	Vitamin D 2mcg
20%	Calcium 260mg
45%	Iron 8mg
5%	Potassium 235mg
* Footnote on Daily Values (DV) and calories reference to be inserted here.	

Figure 3A: Example of nutrition label of a beer

Nutrition Facts	
1 serving per container	
Serving size	12 FL.OZ. (355mL)
Amount per serving	
Calories	100
% Daily Value*	
Total Fat 0g	0%
Saturated Fat 0g	0%
Trans Fat 0g	0%
Cholesterol 0mg	0%
Sodium 30mg	1%
Total Carbohydrate 2g	<1%
Dietary Fiber 0g	0%
Total Sugars 1g	
Includes 1g Added sugars	<1%
Protein 0g	0%
Vitamin D 0mcg	0%
Calcium 0mg	0%
Iron 0mg	0%
Potassium 0mg	0%
*The % Daily Value (DV) tells you how much a nutrient in a serving of food contributes to a daily diet. 2,000 calories a day is used for general nutrition advice.	

INGREDIENTS: FILTERED CARBONATED WATER, ALCOHOL, NATURAL FLAVORS, CANE SUGAR, CITRIC ACID, AND SODIUM CITRATE

Figure 3B: Example of nutrition label of a hard seltzer

count (WBC) 7.2 uL, platelets 222 uL, sodium 96 mEq/L, chloride 52 mEq/L, potassium 4.0 mEq/L, blood urea nitrogen (BUN) 10 mg/dl, creatinine 0.98 mg/dl, magnesium 2.4 mg/dl, Phosphorus 3.2 mg/dl, creatinine kinase (CK) 75 iU/L, HCG qualitative negative, thyroid stimulating hormone (TSH) 1.0 IU/ml, albumin 4.2 g/dl, serum osmolality 205 mOsm/kg, urine osmolality 101 mOsm/kg, and urine sodium < 10 mmol/l.

Computed tomography (CT) scan of the head without contrast was negative for acute changes.

Treatment: Patient was diagnosed with severe hyponatremia with the complication of seizure. He was admitted to the hospital and started on 3% saline for management of hyponatremia. Based on the serum osmolality on presentation, hyponatremia was assumed to be secondary to excessive consumption of hard seltzer. Patient was also placed on fluid restriction.

Consistent and stable rise in PNa was noted for the first 20 hours to 101 mEq/L. At 24 hour mark, the PNa suddenly rose to 106 mEq/L, patient exhibited signs and symptoms (extreme confusion and agitation) of Central Pontine myelinolysis (ODS: Osmotic Demyelination Syndrome) which was confirmed with MRI. At that time 3% saline was discontinued and patient was given D5W. Patient sodium improved to 135 mEq/L one week post admission to the hospital. Patient was then discharged to rehabilitation center. Two months post discharge from the hospital, post intensive rehabilitation, patient was discharged to home and was able to resume his professional duties without any residual neurological sequelae.

Discussion

The market for hard seltzer has grown exponentially over the past few years. This rapid growth has reduced consumption of beer specifically among the young adults. Beer consumption now carries a stigma among younger generations. Almost all alcohol producing companies have started to produce and market hard seltzer to benefit from this growing trend. With the increase in degree popularity of this product, many more young adults are expected to consume

this beverage, with some in excess leading to severe symptomatic hyponatremia, as seen in cases presented here. This publication may be followed by many other reports about severe hyponatremia resulting in neurological sequelae. It is also seen in clinical practice and reported that hyponatremia may also cause death [26]. Patients with moderate hyponatremia have severe underlying disease than those with severe hyponatremia [27] and therefore higher mortality rate [26]. Thirty nine (73%) of the patients who died in the study conducted by Chawla et al on hyponatremia had PNa < 125 mEq/L at the time of admission to the hospital; 14 (27%) were normo-natremic or mildly hyponatremic at admission, but their serum sodium fell below 120 mEq/L during hospitalization [26].

Two cases in this publication had acute hyponatremia with PNa 107 mEq/L and 96 mEq/L, whereas the third patient had a history of hypotension, chronic alcoholism with recurrent hospitalizations for hyponatremia, resulting from binge drinking of hard seltzer. Case # 2 became symptomatic at PNa < 130. In all patients, serum sodium was corrected to over 132 mEq/L within 4-5 days. Importance of urine sodium in these and other hyponatremic patients for appropriate diagnosis has not been studied or published well. Urine sodium was found to be low in almost all the patients with large intake of hypoosmolar beverages, leading to severe hyponatremia. Urine sodium in all our patients was < 10 mmol/l on admission.

Hyponatremia in our cases was purely resulted from overconsumption of hard seltzer which is a low solute drink. Risk of osmotic demyelination syndrome was considered but couldn't be controlled in one patient. Correction of potassium is equally important while correcting low plasma sodium. Equal amount of sodium is retained with correction of potassium, they are directly correlated. 1 mEq of retained potassium affects serum sodium as much as 1 mEq of retained sodium, even partial correction of potassium depletion can cause excessive rise in serum sodium without sodium administration [3]. If sodium is being administered at the time, potassium should also be closely monitored. As correction of potassium may increase the risk of overcorrection of sodium and hence occurrence of ODS. It has been well documented and recommended that hyponatremia correction should not exceed 6-8 mEq/L in any 24 hour period;

this cutoff applies to both acute and chronic hyponatremic patients, regardless of clinical presentation and methods of treatment, including active management and spontaneous correction [3]. However, it is also recommended that the rate of sodium correction should be 6 to 12 mEq/L in the first 24 hours and 18mEq/L or less in 48 hours [27].

Electrolyte disturbances like hyponatremia, may cause further complications including organ damage. Hyponatremia may contribute to organ dysfunction and therefore indirectly contribute to mortality [24]. Emerging data may implicate hyponatremia in falls [28], osteoporosis [29], and fractures, suggesting effects on nervous system and bones [30-32]. A recent study identified hyponatremia as an independent predictor of myocardial infarction in community patients [33]. Hyponatremia can aggravate peripheral insulin resistance [34], impaired hepatic gluconeogenesis [35], and induce a negative isotropic response [36]. One of the studies focusing on hyponatremia reported, the overall mortality rate was 6.1% among all 45,693 hospitalized patients in this study with hyponatremia (PNa <135 mEq/L), compared with 2.3% among 165, 146 patients with PNa >135 mEq/L (Figure 3).

Hyponatremia has been well reported in beer potomania [9,10] but its prevalence with the consumption of hard seltzer has not been reported to the best of our knowledge. Recent addition of hard seltzer to the shelves of liquor stores has brought this product to many, mostly younger generation increasing the risk of hyponatremia and hyponatremia related complications by several folds. Additionally, low calorie and low carbohydrate count makes this product even more enticing compared to other alcoholic beverages available in the market. The trend is rapidly changing from beer consumption to consumption of hard seltzers due to its availability in several fruity flavors. The situation has even worsened due to the current COVID-19 pandemic, seltzers have racked up nearly \$1 billion in sales from March 7 to May 30, according to Nielsen. In comparison, spiked seltzer sales totaled \$1.5 billion in all of 2019 [37]. As most liquor companies, especially beer manufacturers have started producing and marketing hard seltzer, the demand is still hard to meet. An IWSR report predicts that consumption of hard seltzer brands is expected to triple by 2023 [38].

The low sodium content and poor dietary intake during binge drinking episodes adversely affect serum sodium levels. Since sodium is one of the main bio-elements, which is responsible for proper neuromuscular excitability maintenance and contributes in nerve impulse conduction, sodium balance disorders may be related to a risk to basic life functions [24]. In hard seltzer patients, hyponatremia is purely present due to dilutional causes. This is not the case in patients with general hyponatremia.

Conclusion

Hyponatremia in alcoholic patients is not a new concept, beer potomania has been part of literature and clinical practice for long. Newly discovered effects of excessive consumption of hard seltzer shows hyponatremia in alcoholic patients, sometimes severe leading to neurological sequelae. With increasing prevalence of hard seltzer consumption, an increase in the cases of hyponatremia can be expected.

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