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Challenges in the diagnostics and management of hydrochlorothiazide-induced severe hyponatremia in a habitual beer drinker

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Introduction. Beer potomania is a syndrome of severe hyponatremia caused by excessive beer consumption. The risk of hyponatremia increases in the case of a combination of beer potomania and the use of thiazide diuretics.

Case report. A 55-year-old male patient with the anamnesis of a long-lasting alcohol use disorder was presented to the emergency department after seizures accompanied by an impaired mental status. He had been drinking beer regularly for ten years. On physical examination, the patient was tachypneic, tachycardic, disorientated, restless, the Glasgow Coma Scale score of 9, observed tremor, and the smell of alcohol from the mouth. Laboratory results showed plasma sodium 105 mmol/L, blood urea nitrogen 1.8 mmol/L, the alcohol concentration in the blood 0.06 g/l, and calculated serum osmolality 219 mOsm/kg H_2O. After a detailed initial evaluation of the patient and labs for hyponatremia, a diagnosis of beer potomania was established. On the third day of hospitalization, the patient's anamnesis was filled with information about the use of Valsartan/hydrochlorothiazide, together with reduced salt intake in the diet for three months for arterial hypertension treatment. It was decided that the combination of heavy beer drinking with the use of diuretic and reduced consumption of salt provoked hyponatremia. The patient was treated with infusion therapy of sodium chloride; plasma sodium level reached 136 on the third day of treatment.

Conclusions. This case represents an unusual syndrome of beer potomania in conjunction with hydrochlorothiazide usage and reduced salt consumption, which can result in severe hyponatremia that may provoke severe neurologic damage.

Keywords: beer potomania, hyponatremia, thiazide diuretics

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INTRODUCTION

'Beer potomania', an uncommon cause of hyponatremia provoked by excessive beer drinking (usually five or more drinks per day), was first mentioned by Demanet et al in 1971 (1–3). This syndrome is associated with patients who develop hyponatremia due to inadequate daily solute intake and excessive beer consumption (2, 4). Although hyponatremia is quite common among chronic alcohol abusers, it is vital to diagnose beer potomania quickly. Time is essential since hyponatremia usually presents with other serious conditions such as malnutrition, cirrhosis, or congestive heart failure (3, 5). Heavy drinkers, especially in older age, usually use diuretics or antipsychotic drugs (3). For this reason, it is necessary to ascertain an anamnesis of the use of these medications due to the possibility that hyponatremia is caused by them (6).

Chronic hyponatremia frequently results in cognitive or coordination dysfunction, as well as mood disorders (7). On the other hand, severe acute hyponatremia can be life-threatening by causing brain oedema, seizures, and, in rare cases, death (8). Considering that there could be numerous underlying reasons, it is crucial to identify pathophysiology of hyponatremia because it determines its appropriate management. In heavy drinkers' case, beer, which contains little or no salt at all, along with carbohydrate and alcohol causes suppression of protein breakdown and reduces the solute load to the kidney (9). This mechanism provokes defective water clearance and induces dilutional hyponatremia (2).

Recognition of beer potomania is a difficult challenge for a physician, particularly in those cases when a patient has other risk factors for developing hyponatremia. Arterial hypertension is a widespread disease usually treated with antihypertensive drugs such as diuretics, which are used in 70.2% cases (10). Moreover, recent studies show that alcohol consumption is also common: in a survey conducted in 2018, 26.5% of people reported they had engaged in binge drinking in the past month. In combination, these two conditions may increase the risk that adjusting the treatment of one disease will not take into account the appearance of another dangerous condition such as hyponatremia.

This article presents a 55-year-old alcoholic male patient, who presented with neurological dysfunction due to hyponatremia caused by beer potomania together with the use of diuretics.

CASE REPORT

A 55-year-old male patient was admitted to the emergency department due to seizures that were provoked by four-day-long drinking. The patient had a history of heavy drinking of ten years, a head trauma, and diabetes mellitus. According to the patient's relatives, he had a single episode of epileptic seizures as a consequence of a long alcohol drinking period 15 years earlier. The patient was treated in the department of psychiatry several times for his problems with alcohol. However, the treatment did not result in improvement. From the physical examination: the patient was asthenic (probably because of his habit to skip meals), tachypneic with a respiratory rate of 28, tachycardic with the heart rate of 100, disorientated, restless, with Glasgow Coma Scale score of 9, observed tremor, the smell of the alcohol from the mouth, and a dry tongue. The patient's relatives denied any periods of vomiting or diarrhea or any other health problems.

The patient's laboratory results are presented in the Table. Liver enzymes and the AST/ALT ratio were typical for alcoholic liver injury. Brain oedema was observed in the computed tomography scan of the head. Considering the patient's anamnesis, laboratory, and diagnostic test results, he was admitted to the toxicology department.

On the next day, the patient developed acute respiratory failure, was intubated and transferred to the intensive care unit. In the chest X-ray, nothing relevant was found. On the following day, an abdomen ultrasound was performed and liver steatosis and uncomplicated gallstones were detected. On the third day of hospitalization, anamnesis was supplemented with the information that the patient used 160 mg Valsartan and 12.5 mg hydrochlorothiazide for three months for arterial hypertension treatment. In addition, his general practitioner recommended reduction of salt intake. Based on this information, it was suspected that long-term use of diuretics along with the reduced salt in the diet and beer binge drinking triggered such severe hyponatremia. On the same day, the patient was extubated, his state of consciousness was good, the respiratory function stabilized, and he was returned to the ward.

Table. Patient's laboratory test results

Blood test	Result (on admission)	Result (after 48 hours)	Result (on discharge day)	Normal value
WBC ($\times 10^9$/L)	7.2	13.1	–	4.0–9.0
Hb (g/L)	129	124	–	130–180
Plt ($\times 10^9$/L)	185	115	–	150–350
Na (mmol/L)	105	129	137	136–150
K (mmol/L)	4.19	4.05	4.9	3.5–5.1
Cl (mmol/L)	66.9	94.8	103	98–107
Blood urea nitrogen (mmol/L)	1.8	3.6	3,0	2.1–7.1
Creatinine (μmol/L)	57	–	62	62–115
Glucose (mmol/L)	7.3	6.6	5.0	3.9–5.8
AST (UI/L)	305	–	153	10–37
ALT (UI/L)	154	–	148	13–40
Total bilirubin (μmol/L)	20.1	–	9.0	5.1–20.5
Alcohol concentration (g/L)	0.06	–	–	0
Serum osmolality (mOsm/kg)	219	268	282	285–295

During the hospitalization, the patient was treated symptomatically with infusion therapy of NaCl 0.9%, along with a small amount of NaCl 10% infusions to treat electrolyte imbalance. Infusion of osmotic diuretics with glucocorticoids was prescribed to treat brain oedema. Treatment included benzodiazepines to prevent the emergence of alcohol-related seizures and other alcohol withdrawal symptoms, and neuroleptics to reduce agitation and disorientation. Thiamine therapy was given to improve the mental status. After three days of treatment, the patient's sodium level rose to 136 mmol/L (Figure). Ten days later, he was released from the hospital with instructions to visit his general practitioner after a week and to apply for addiction rehabilitation.

DISCUSSION

The patient's hyponatremia was classified as hypotonic hyponatremia since serum osmolality was 219 mOsm/kg H_2O. It was considered severe due to sodium levels <120 mmol/L (10). Due to seizures and impaired consciousness that occurred

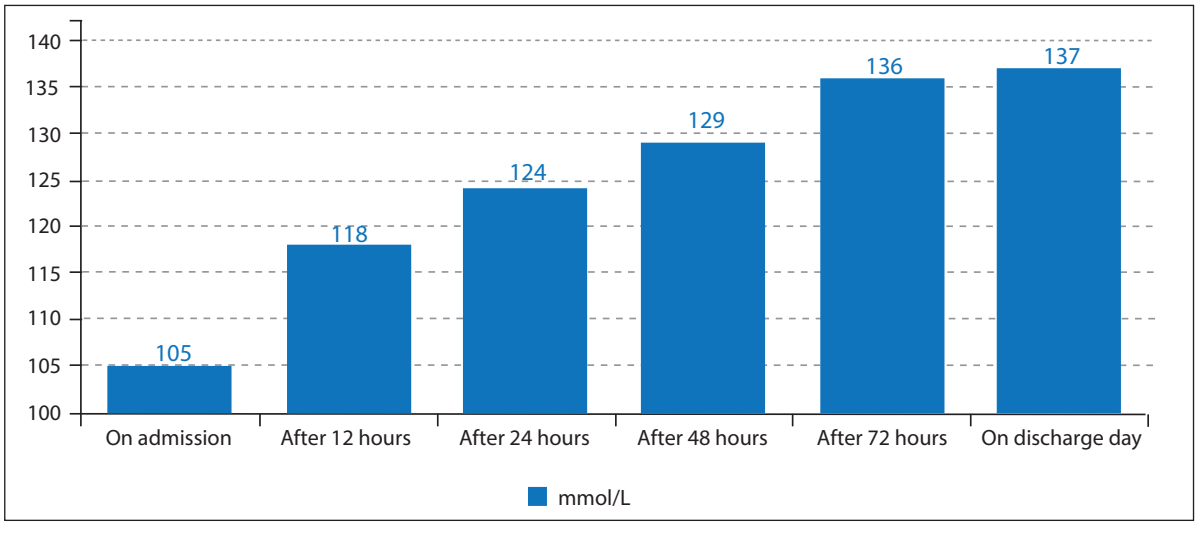

Figure. Changes in the sodium serum level during hospitalization

as the first symptoms, it was probably acute hyponatremia (<48 h) (12). It is unlikely that such severe hyponatremia was caused only by the use of hydrochlorothiazide because it usually occurs during the first two weeks of treatment in such cases (13). Meanwhile, the patient had been using these medications for three months without any symptoms. However, it is essential to mention that hyponatremia can develop any time during the use of thiazide diuretics in the presence of other triggering factors: old age, an increase in water intake, and poor salt intake (13). Hypertension has been treated with conventional medications without assessing the risks of habitual alcohol use. There is likely a group of patients who should be treated after the evaluation of both situations. Arterial hypertension and alcohol alone are not a significant risk for hyponatremia, but it should be emphasized that it is their combination that causes the problem. For this reason, it was decided that in this case hyponatremia was caused by beer potomania in conjunction with the use of hydrochlorothiazide and reduced salt consumption. Other reasons, such as congestive heart failure, hypothyroidism, psychogenic polydipsia, and malignancy were unlikely due to a lack of symptoms and relevant changes in laboratory results.

Beer potomania is described as a syndrome of euvolemic, hypotonic hyponatremia, which is associated with excessive beer drinking and low solute intake (14). The key to pathophysiology of this syndrome is that inadequate solute intake with meals leads to water retention, which results in dilutional hyponatremia (14–16). Healthy patients' normal osmole excretion is almost 600–900 mOsm/day (3). Urinary dilution capacity is approximately 50 mOsm/l, which means that a substantial amount of water must be drunk to exceed the urinary dilution capability (3, 14). Heavy beer drinking, together with low sodium intake with food, provokes a decrease in solute excretion, which triggers disturbed excretion of free water (2). It is also known that alcohol triggers inappropriate antidiuretic hormone secretion (3). Changes in the secretion of this hormone manifest in a decreased ability to reabsorb free-water from the collecting tubes and also affects water excretion (3, 4, 15).

As in our patient's case, beer potomania usually manifests in neurological symptoms, including an altered mental status, focal neurologic signs, or even seizures (8, 15). Symptoms also depend on the speed of developing hyponatremia: acute hyponatremia can cause brain oedema or intracranial hypertension, which can result in a coma, brain-stem herniation, respiratory arrest, or even death (7). Patients are usually misdiagnosed due to non-specific symptoms because the same symptoms can be provoked by alcohol intoxication or withdrawal (15). In the case of beer potomania, the serum sodium level usually ranges between 97 and 134 mmol/L; other common findings in laboratory results are hypokalaemia and decreased blood urea nitrogen (8). Similar patterns of laboratory diagnostics, except hypokalaemia, are also seen in the case of our patient.

Management of beer potomania depends on the severity of the symptoms. If the patient displays no symptoms, then restriction of fluids, no oral intake except medications for 24 hours, and regular monitoring of the vital and biochemical parameters are safe (3, 14). On the other hand, if the patient is experiencing severe symptoms, number one treatment option is hypertonic saline in the intensive care unit (3, 14). In our patient's case, treatment included hypertonic along with isotonic sodium chloride infusion therapy, which was efficient enough according to the rapid improvement of the patient's condition.

It is essential to mention that if the concentration of plasma sodium is below 120 mmol/L, there is a high risk of developing osmotic demyelination (17). Patients admitted with beer potomania are subject to many risk factors for developing osmotic demyelination syndrome, such as chronic hyponatremia, alcohol consumption, and the probability of fast correction of hyponatremia because of the underlying pathophysiological pathway (4). In such cases, correction of sodium levels by more than 8 mmol/L per day should be avoided (17). In our patient's case, the sodium level was corrected in a shorter time, although we followed all recommendations. It means that even with adherence to the mentioned guidelines, it is very difficult to control the speed of correction of sodium levels.

An important message to general practitioners who prescribe diuretics is to clarify the patient's anamnesis of drinking alcohol before starting diuretics therapy. If the answer is positive, it is necessary to emphasize the importance of salt in daily nutrition to avoid such serious consequences.

CONCLUSIONS

The reported case represents beer potomania – an unusual cause of hyponatremia, which develops due to excessive beer consumption and inadequate diet with salt deficiency. Usually, identifying this cause of hyponatremia poses a challenge, because patients deny drinking excessive amounts of beer, forget to mention using diuretics or other essential anamnesis details as was the case of our patient. Early and proper management of hyponatremia is crucial in treating beer potomania. Educating heavy drinkers on appropriate dietary habits while using diuretics simultaneously with alcohol is essential. Moreover, treating alcohol dependence is very important in avoiding recurrence.

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ALUMI PIKTNAUDŽIAUJANČIO VYRO VARTOJAMO HIDROCHLOROTIAZIDO SUKELTOS SUNKIOS HIPONATREMIJOS DIAGNOSTIKA IR GYDYMAS

Santrauka

Įvadas. Alaus potomanija yra sunkios hiponatremijos sindromas, kurį sukelia per didelis alaus suvartojimas. Hiponatremijos išsivystymo rizika padidėja tais atvejais, kai esant alaus potomanijai kartu vartojami tiazidiniai diuretikai.

Klinikinis atvejis. Ilgai alkoholį vartojantis 55 metų vyras atvyko į priėmimo-skubios pagalbos skyrių sutrikusios sąmonės būklės po traukulių priepuolio. Jis nuolat vartojo alų 10 metų. Fizinės apžiūros metu pacientui buvo išsivysčiusi tachipnėja, tachikardija, dezorientacija, neramumas, sąmonės būklė įvertinta 9 balais pagal Glazgo komos skalę, stebimas tremoras, alkoholio kvapas iš burnos. Laboratoriniais tyrimais nustatyta plazmos natrio koncentracija 105 mmol/L, šlapalas 1,8 mmol/L, alkoholio koncenracija kraujyje 0,06 g/l ir apskaičiuotas serumo osmoliališkumas 219 mOsm/kg H_2O. Po detalaus pradinio įvertinimo ir laboratorinių tyrimų rezultatų, rodančių hiponatremiją, pacientui buvo diagnozuota alaus potomanija. Trečią hospitalizacijos dieną paciento duomenys buvo papildyti informacija apie tris mėnesius vartojamą valsartaną / hidrochlorotiazidą dėl diagnozuotos arterinės hipertenzijos ir rekomenduotą mažesnį druskos kiekį maiste, todėl buvo nuspręsta, jog gausus alaus vartojimas kartu su diuretikais ir mažesniu druskos kiekiu maiste išprovokavo hiponatremiją. Pacientas buvo gydytas natrio chlorido infuzine terapija, trečią gydymo dieną natrio koncentracija plazmoje pakilo iki 136 mmol/L.

Išvados. Nedažnas sindromas – alaus potomanija – kartu su hidrochlorotiazido vartojimu bei mažesniu druskos kiekiu maiste pasireiškia sunkia hiponatremija, galinčia išprovokuoti sunkius neurologinius pažeidimus.

Raktažodžiai: alaus potomanija, hiponatremija, tiazidiniai diuretikai