Review Article

Taking alcohol with a (large) pinch of salt: Understanding the osmoles in "beer potomania" and "starvation potomania"

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ABSTRACT

Alcoholism is a major problem globally and beer drinking is on the rise. Many of the alcoholics sustain only on beer for days and do not get adequate solutes in the form of food. Similar situation can arise in cases of decreased food intake due to severe deliberate restriction or other factors like decreased appetite from cocaine. In these cases, salt free liquid intake may still be adequate. Such situations are conducive to the development of hyponatremia. Some of the solutes in diet, such as salt and protein, act as osmoles in urine. In these conditions, understanding the role played by solutes in diet and osmoles in urine is of vital importance for appropriate treatment of hyponatremia.

Key words: Beer potomania, hyponatremia, osmoles, starvation potomania

Alcohol and Alcoholism

Alcoholism is a major issue globally. Alcohol consumption in USA is rising and in between 22% and 26% of US community hospital admissions are alcohol related.[1] Continued drinking despite adverse consequences is considered alcohol use disorder[2] and 9% of US adults meet these criteria.[3] In USA, according to National Institute on Alcohol Abuse and Alcoholism (NIAAA), 3 in 10 adults drink at levels that put them at risk for alcoholism, liver disease and other problems.[4] A standard drink in USA is considered to contain about 14 g of absolute alcohol. This amounts to one 12-ounce bottle or can of either beer or wine cooler, one 5-ounce glass of wine, or 1.5 ounces of 80-proof distilled spirits. Heavy or at risk drinking is considered when either daily or weekly limits are exceeded. According to NIAA those limits are 4 drinks/day or 14 drinks/week in men. In women it is either 3 drinks/day or 7 drinks/week. The risks of alcoholism exist on a continuum spectrum. Nearly 4% of at-risk drinkers have alcohol dependence, which is a condition of craving and loss of control over drinking despite harm, with daily or near daily drinking.

Besides many negative personal, social and health consequences,[5] electrolyte disorders are a common finding in alcohol related hospital admissions, the most commonly encountered being hyponatremia.

Beer Intake

Various other mechanisms of hyponatremia in alcoholics have been described. These include hypovolemia, pseudohyponatremia from hypertriglyceridemia and cerebral salt wasting syndrome from alcohol induced cerebral atrophy. Another scenario for development of hyponatremia involves excess water intake relative to solutes. Excessive chronic consumption of beer without adequate food intake can cause severe hyponatremia. It is called "beer potomania", if laboratory values are mostly consistent with water intoxication and no other cause of hyponatremia is found. Demanet *et al*.[6] first coined this entity in 1971 and since then it has been reported in the literature approximately 23 times.[7]

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Beer has a very low content of sodium and protein [Table 1] and no other major source of solutes or osmoles. Some chronic severe alcoholics subsist solely on beer. Many can drink more than 24 cans a day. Because of this they have extremely little daily intake of solutes.

Water Intake

Intake of water or solute free liquid, in excess, will not necessarily induce hyponatemia in an otherwise healthy patient with properly functioning kidney with normal diluting and concentrating capacity (for example, the absence of hydrochlorothiazide and arginine vasopressin [AVP] respectively). Ingestion of adequate food containing solutes, some of which end up as urinary osmoles, allows excretion of ingested water in excess of 20 L/d. However, such a person may develop water intoxication leading to hyponatremia if the excretion of water is limited due to the lack of simultaneous ingestion of solutes, as explained below.

Urinary Osmoles

Urinary osmolar excretion is determined by dietary intake of solutes, predominantly sodium chloride (NaCl) and urea. Urinary urea comes from mostly ingested protein nitrogen in nitrogen balanced patients. An average 24-h American adult osmolar excretion is about 900 mOsm. Of that, approximately 300 mOsm is contributed by urea, 300 mOsm by NaCl and rest by potassium, magnesium, calcium, phosphates, sulfates, bicarbonate, ammonia, uric acids, amino acids, and creatinine.[8]

Salt and Protein Osmolar Intake

One level teaspoon of table salt contains approximately 6200 mg NaCl. The ratio of Na is 40% in NaCl and ratio of Cl is 60% (molecular weight [MW] Na 23 and MW Cl 35.5). Hence, there is approximately 2400 mg Na in a teaspoon of table salt. To convert Na and Cl in mg to mEq it is divided by the respective MW. Hence, 2400 mg Na (1 teaspoon) will give 104 mEq of Na. Using the same method of calculation, 1 teaspoon of salt will provide about 104 mEq Cl and hence a total of 208 mEq or mOsm of NaCl.

Table 1: One can of regular beer

Calories	153
Total fat	0 g
Sodium	14 mg
Potassium	96 mg
Carbohydrate	12.64 g
Protein	1.64 g

The contribution that protein intake make on urinary osmoles cannot be calculated accurately. This also depends on the nitrogen balance in a person. Approximately 45-50 mOsm of urinary urea are contributed by 10 g of protein intake.

Normal Renal Physiology

Normal human renal physiology is such that amount of urine excretion is dependent on solutes in urine that act as osmoles. In normal human urine osmolality can be as low as 50 mOsm/L; means that for every liter of urine excretion at least 50 mOsm would be required. Hence, the maximum volume of urine excretion/day can be calculated by dividing the total daily solute excretion by maximally diluted urine osmolality for that patient.[9] Using an average of 900 mOsm, daily excretion will be 900/50, or 18 L/d urine volume. In other words, this person can drink 18 L water with no problems.

Mechanism of Hyponatremia

Predominantly in heavy beer drinkers with no other food intake there will be very minimal solute intake. Beer does contain carbohydrates which prevent ketosis while drinking and hence generally ketones will not be acting as osmoles in these cases.[10] Without ketosis, 70 kg alcoholics will consume about 35 g of their own protein stores/day. That will provide about 170 mOsm from urea and other obligatory solutes from cellular catabolism will add about 75 mOsm to a total of about 245 mOsm.[10] Consuming a 24 pack of 12-ounce beer cans/day with each about 14 mg Na will give 336 mg, which is 14 mOsm Na (336/23). Similar 24 pack beer will give 2304 mg potassium which will provide 59 mOsm (2304/39). So only about 73 mOsm will be added by daily alcohol, leading to total daily solute excretion of 318 mOsm (245 + 73). However, the patient is still ingesting a volume of 8640 ml. Taking out about 1000 ml for insensible loss will still leave roughly 7640 ml in the body. The total 24 h urine volume will be about 6360 ml (318/50), leaving 1280 ml daily in extracellular space to cause water intoxication. This daily extra addition of water will lead to hyponatremia.

On top of this, some of these beer drinkers may also have some non-osmotic secretion of antidiuretic hormone as a result of volume depletion from chronic gastrointestinal losses/diarrhea or nausea/vomiting. This will prevent urine osmolality to go as down as low as 50 mOsm. In fact in such patients maximal urine dilution may just be no lower than 100 mOsm.[11] In above example the urine excretion will be cut in half to 3180 ml leaving about 3180 ml for water intoxication on daily basis. Also in these patients the measured serum osmolality from lab

may not be low due to contribution of ethanol levels in blood. Blood alcohol level in mg/dl has to be divided by 4.6 to convert to mOsm. Similarly urinary alcohol levels may mask urine hypo-osmolality, though urine Na will be low.[12] Contribution of alcohol to urine osmolality can be calculated by multiplying serum alcohol levels by 1.4.[13]

Though alcohol induces diuresis via antagonizing AVP, is well-known, but it is initiated by the increase in blood alcohol levels. However it is not maintained for long even if high steady levels of alcohol maintained.[14] Hence, likely this does not help counter development of hyponatremia in chronic alcoholics. Also in these cases the clinical state is such that although there is conducive situation for brisk diuresis due to suppressed AVP but no solutes that will maintain urine output. This causes the extra water to stay in body causing hyponatremia, oliguria and edema.

Osmolar Replenishment

However, these patients have brisk diuresis when solutes are introduced. Take an example of 1 L of 0.9% saline that has 308 mEq. With urine osmolality of 50 mOsm/L this 1 L will be excreted with 6 L of urine, or 5 extra L of free water. As an example if a 70 kg alcoholic man who has some extra body water, e.g. about 5 L, total body water of about 45 L then his serum Na will go up by about 13 points ($110 \times 45/40$) after 5 L of free water urine loss. This can cause more rapid than expected rise of serum Na. 18% of patients with beer potomania developed osmotic demyelination syndrome (ODS).[11] In order to minimize risks of ODS, Sanghvi *et al.*[11] have described a detailed approach to management of these hyponatremic patents, after admission. Rules of rate of rise of Na remain same as any other patients but these include intensive care units with frequent Na monitoring, cautious initiation of oral feeding with nothing per oral in the initial hours, intra venous fluids in limited amounts, use of D5W if needed to restrict rate of rise of Na.

Similar situation can also be observed in starvation and with deliberately restricted diets.[15] Also in severe malnutrition and severe loss of appetite (as in cocaine abuse), where there is significant salt free liquid ingestion, as in alcoholics, "starvation potomania" can arise. It is important to recognize such conditions because recovery depends highly on very cautious osmotic replenishment.

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