HYPONATREMIA, 2 UNUSUAL CAUSES

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1. **Beer potomania.**

The original description of this condition goes back to 1972 about some VA patients who were admitted with significant hyponatremia, as low as 108 meq/L. The one thread in the history is inordinate consumption of beer associated with reduced solute intake[1]. The cardinal features of the condition entail reduced serum and urine osmolality as well as reduced random urine sodium[2]. We are accustomed to alcohol-induced hypomagnesemia[3], which is very common. However, beer potomania is not so common. Furthermore, such patients may have other underlying medical conditions that confound the picture. For example, the use of diuretics, the presence of cirrhosis, and congestive heart failure adds another layer of complexity.

Diuretics. The mechanism of hyponatremia is due to natriuresis. It's associated with low serum and urine osmolality.

Cirrhosis of the liver. The main pathology hinges on the increased secretion of ADH[4]. This is a commonly seen phenomenon.

Congestive heart failure. The presence of hyponatremia in this condition reeks of a multidimensional pathology. It is a combination of the use of diuretics, a higher level of ADH, activation of the renin-angiotensin axis, and another mechanism that includes the sympathetic system[5].

Symptoms. They are nonspecific, and there is no pathognomic presentation. Commonly observed symptoms include muscle weakness, fatigue, and dizziness.

Treatment. Like any case of chronic hyponatremia, rapid correction can lead to the so-called osmotic demyelination syndrome[6], a neurologic deficit that is slow to resolve and lasts up to and beyond a year. Strict guidelines are established to prevent this.

Recommendations by Sanghvi, et al. for correction of hyponatremia in beer potomania[7].

Management Recommendations for Correction of Hyponatremia in Beer Potomania
Nothing by mouth except medications for 24 hours
No intravenous fluids unless symptomatic
Prescribe intravenous fluids in finite amounts if needed
Intensive care status
Check serum sodium every two hours
Goals - Serum sodium increase < 10 mEq/L in first 24 hours - Serum sodium increase < 18 mEq/L in first 48 hours
Reduce serum sodium levels if necessary
Give any intravenous medications in sugar solutions (5% dextrose in water)
If caloric intake is needed, use intravenous sugar solution (5% dextrose in water)

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2. Losartan-induced hyponatremia.

Case presentation. A 77-year-old male with PMH of HTN on losartan 50mg daily and amlodipine 5mg daily presented to the ER for evaluation of a syncopal event while on a routine bike ride. He reports that about half a mile into the ride, his legs became "really like heavy," and he could not pedal anymore. Concurrently, he felt his neck was having trouble supporting his head. He managed to dismount and sit on a railing, where he drank some water before losing consciousness and falling onto his back. After regaining consciousness, he called a friend for assistance. He drinks 3-4 bottles of water a day and denies any use of a diuretic. He also has no history of any arrhythmia. BP 152/71, apical rate of 57 without any orthostatic changes. The cardiac and neuro exams were unremarkable. Labs: Na 118, Chloride 85, Creatinine 1.05 (GFR of 73) and otherwise negative CMP, serum osmolality 257 mOsm/kg, urine osmolality 275 mOsm/kg. The patient received 1000 cc of .9% NS in ER, and thereafter an infusion of it (100cc/hr) while his sodium was being monitored every 4 hrs. Levels ranged from 118 to 120, 121, 119, 120, and then 119. Nephrologist evaluated patient and ordered a stat random urine sodium. Once the result came back at 69mmol/L consistent with SIADH. He double-checked with another senior nephrologist, who suggested this could be a rare case of losartan-associated hyponatremia. To make matters a bit interesting, patient once upon a time was taking salt tablet for hyponatremia but stopped. The nephrologist recommended stopping the losartan and increasing the amlodipine. He also started a salt tablet. Patient was adamant about taking the losartan. It took quite a bit of convincing to get through to him that it might be contributing to the hyponatremia. Once he stopped taking it, the sodium began creeping steadily, and at the time of discharge a few days later, it was up to 127.

Losartan, the first marketed angiotensin receptor blocker (ARB), has been used since 1995. It has an impressive safety profile. A very rare complication is hyponatremia as monotherapy[1,2,3]. There isn't much published about it, but since it's used extensively, it may happen more often than we care if we look for it. Certainly, it wouldn't be unusual in the presence of a diuretic causing natriuresis[4]. The mechanism of hyponatremia in the presence of losartan monotherapy is not

altogether clear-cut. It's hypothesized that it occurs via the angiotensin axis to cause a decrease in aldosterone and may be more common in diabetics[1]. This patient had neither diabetes nor hyperkalemia.

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