Hyponatremia is not uncommon among patients who present to emergency departments or are hospitalized. Under ordinary circumstances, blood osmolality is controlled by a combination of vasopressin (antidiuretic hormone (ADH)) release from the posterior pituitary and thirst-driven water ingestion. When blood osmolality is even slightly below normal, vasopressin release is suppressed [1]. Without vasopressin signaling, the renal collecting duct becomes impermeable to water reabsorption, and the urine quickly becomes very dilute (<100 mOsm/kg H₂O). Unfortunately, the story is a little more complex, because volume depletion is a secondary stimulus for vasopressin release. Hence, most individuals with an actually (e.g., hemorrhage) or effectively (e.g., congestive heart failure) reduced circulatory volume are at least mildly hyponatremic. The plot thickens further, because other nonphysiological stimuli can also stimulate vasopressin release, leading to the syndrome of inappropriate ADH secretion (SIADH). Common causes include nausea, pain, and hypoxia. Thus, hospitalized patients often have multiple reasons for ADH-mediated hyponatremia. In all of these cases, the hyponatremia is not due to excessive water intake. This case describes a more unusual cause of hyponatremia, so-called beer potomania [2], in which free-water (out of proportion to osmoles) ingestion is the culprit. Even if the urine is very dilute, one can still excrete only approximately 1 L of water for each 100 mOsm excreted. Ingesting the majority of one’s calories from beer generates very few osmoles because it is low in electrolytes and is accompanied by large amounts of free water and because ethanol metabolizes to CO₂ and water. In addition, nonosmotic stimuli for ADH release (nausea, volume depletion) are often present, so that the urine is not maximally dilute. Although such cases are rare, it is important to recognize them. Administration of even modest amounts of intravenous saline provides abundant osmoles (approximately 300 mOsm/L), can correct hypovolemia (and suppress vasopressin release), and thus overpromptly correct the hyponatremia. Thus, this patient group is at particularly high risk for the serious and often deadly sequelae of central pontine myelinolysis.

Author Contributions: All authors confirmed they have contributed to the intellectual content of this paper and have met the following 3 requirements: (a) significant contributions to the conception and design, acquisition of data, or analysis and interpretation of data; (b) drafting or revising the article for intellectual content; and (c) final approval of the published article.

References


Commentary

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This case of a 63-year-old patient with severe hyponatremia demonstrates the complications of alcohol abuse that lead to severe electrolyte imbalance. When seen initially, the patient already had a serum sodium concentration of 113 mmol/L. This is a potentially life-threatening condition unless it develops slowly enough to allow physiological compensating mechanisms to minimize complications. Severe hyponatremia should always be treated in a hospital setting, so it is somewhat puzzling why the patient was not advised to report to the closest emergency room when his serum sodium concentration of 113 mmol/L was discovered. It took