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Beer Potomania: A Literature Review of an Underrecognized Cause of Severe Hyponatremia

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ABSTRACT

Beer potomania is a rare but significant cause of severe hyponatremia, often misdiagnosed as syndrome of inappropriate antidiuretic hormone secretion (SIADH). It is characterized by excessive beer consumption and insufficient dietary solute intake, leading to impaired renal water excretion and dilutional hyponatremia. The pathophysiology involves reduced osmotic load, altered antidiuretic hormone (ADH) regulation, and compromised renal handling of free water. The clinical overlap with SIADH presents diagnostic challenges, necessitating a thorough dietary and biochemical evaluation. This review discusses the mechanisms, differential diagnosis, and management strategies for beer potomania, emphasizing the need for gradual sodium correction to prevent osmotic demyelination syndrome (ODS) and addressing underlying alcohol use disorder to reduce recurrence.

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Introduction

Hyponatremia, defined as a serum sodium concentration of <135 mEq/L, is one of the most common electrolyte abnormalities encountered in clinical practice. The etiology of hyponatremia is diverse, with common causes including SIADH, heart failure, cirrhosis, renal disease, and excessive water intake. Among these, beer potomania remains an underrecognized cause, particularly in chronic alcohol users. First described in 1971, beer potomania results from excessive consumption of beer, which is notably low in sodium and protein, coupled with poor overall nutritional intake. The lack of sufficient osmotic load impairs free water clearance by the kidneys, leading to severe dilutional hyponatremia. This condition is often confused with SIADH due to similar laboratory findings, yet the underlying pathophysiology is distinct, emphasizing the importance of accurate diagnosis and appropriate management strategies.

Methods

A comprehensive literature review was conducted using databases including PubMed, Google Scholar, and Medline. Search terms included 'beer potomania,' 'alcohol-associated hyponatremia,' and 'low solute syndrome.' Relevant studies published between 2000 and 2023 were reviewed, including case reports, clinical studies, and systematic reviews. Studies focusing on pathophysiology, diagnostic challenges, and management approaches were analyzed. Inclusion criteria consisted of peer-reviewed articles detailing patient presentations, biochemical markers, and treatment outcomes of beer potomania. Exclusion criteria included studies focusing on other causes of hyponatremia without specific discussion of beer potomania.

Discussion

Beer potomania is a distinct cause of severe hyponatremia resulting from a deficiency in solute intake, impairing the kidney's ability to excrete free water. Unlike SIADH, where inappropriate ADH secretion leads to water retention, beer potomania is driven by a low osmotic load due to inadequate dietary sodium and protein intake [1]. Pathophysiologically, the kidney requires a minimum solute load of 600–900 mOsm per day for effective water clearance. In beer potomania, this threshold is not met, leading to dilute urine production and progressive hyponatremia. Additionally, chronic alcohol use disrupts hepatic metabolism, reducing gluconeogenesis and protein availability, which further impairs urine concentration [2]. One of the major challenges in diagnosing beer potomania is its clinical resemblance to SIADH. However, key distinguishing features include low urine osmolality (<100 mOsm/kg), low urine sodium (<10 mEq/L), and an absence of euvolemic features typically seen in SIADH [3]. Unlike SIADH, which necessitates fluid restriction, beer potomania improves with solute administration, highlighting the importance of a thorough dietary history and biochemical assessment [4]. Management of beer potomania focuses on controlled sodium correction to prevent ODS, a severe neurological disorder that occurs if hyponatremia is corrected too rapidly. Guidelines recommend gradual correction at a rate of 6–8 mEq/L per 24 hours [5]. Nutritional repletion, including sodium and protein intake, is crucial to restore renal solute excretion capacity. Additionally, addressing underlying alcohol dependence through multidisciplinary care improves long-term outcomes [1,6].

Conclusion

Beer potomania is an often-overlooked cause of hyponatremia, frequently misdiagnosed as SIADH due to similar laboratory findings. However, a careful dietary and social history, along with urine studies, can differentiate these conditions. Appropriate management includes gradual sodium correction, nutritional repletion, and addressing alcohol dependence to prevent recurrence. Increased awareness and early recognition of beer potomania among clinicians will improve diagnostic accuracy and optimize patient outcomes.

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