

Lithium-Induced Nephrogenic Diabetes Insipidus Self-Treated With Beer Potomania and Masquerading as Shock: A Case Report

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Introduction: Nephrogenic diabetes insipidus (NDI) is a known adverse effect from lithium use, commonly presenting as polyuria and polydipsia. Patients are often able to drink enough water to keep up with urinary losses. Here we discuss a patient accustomed to drinking 10 beers daily who developed rapid volume depletion, shock, and hypernatremia after his access to oral fluids was disrupted.

Case Description: A 66-year-old male with a past medical history of bipolar disorder and alcohol use disorder presented with altered mental status, ataxia, and severe weight loss. Admission labs were significant for leukocytosis, hyponatremia, lactic acidosis, and acute kidney injury. Despite empiric antibiotics and volume resuscitation for presumed septic shock, on day two, he required pressors for hemodynamic instability and intubation for mental status. Repeat labs revealed hypernatremia and an elevated lithium level. Diagnosis of NDI was confirmed by a high serum and low urine osmolality, without improvement after DDAVP administration. We stopped lithium and initiated hypotonic fluids, amiloride, hydrochlorothiazide, and indomethacin. Gradually the patient's sodium normalized, pressors were weaned off, and he was extubated.

Conclusion: In this case, we describe a delayed presentation of lithium-induced NDI, initially appearing as hypovolemic shock and hyponatremia, then manifesting as persistent hemodynamic instability and hypernatremia on day two. Ad lib fluids and possibly beer potomania enabled self-correction of sodium levels until our patient's oral intake was restricted. Clinicians should include NDI in the differential for patients taking lithium who develop hypotension, hypernatremia, or shock. Rapid identification and treatment may help avoid decompensation.