A Rare Case of Diabetes Insipidus Following Outpatient Constipation Treatment

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Introduction

Central diabetes insipidus is a common sequelae after cerebral trauma, primary tumors, infiltrative disease, and neurosurgery. Data on central diabetes insipidus after iatrogenic hyponatremia is limited. Regardless of the causative agent, current consensus indicates the same emergent correction rate and goals for the treatment of severe acute hyponatremia. However, it is likely that the causative agent for the hyponatremia may have an impact in the progression of the disease and its response to emergent correction.

Case Study

In this case, we present the case of an otherwise healthy 37-year-old male that develops severe hyponatremia after the consumption of magnesium citrate as a laxative for the treatment of constipation. Patient had occasional episodic constipation that he had treated with stimulant senna based laxatives in the past. Due to patient's lifestyle and time constraints, patient went to the pharmacy seeking treatment with shorter onset of action. Patient was recommended the osmotic laxative Magnesium Citrate as a shorter acting alternative. Patient reported taking the laxative and continuing to hydrate with "several glasses of water" while experiencing bowel movements that turned into diarrhea. 4 hours after the ingestion of the laxative, patient experienced sudden onset of generalized weakness and dizziness and presented to the ED for further evaluation. He reported injesting one bottle, 10 ounces, of magnesium citrate early on the day of presentation and denied taking any other medications. On examination, the patient was lethargic, grunting, and experienced a tonic-clonic seizure. He was noted to have dry oral mucosa, pale sclera, and poor skin turgor. Based on the patient's history and examination, 1 L bolus of intravenous normal saline was initiated.

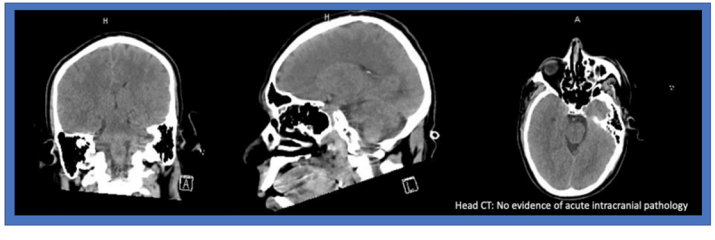
Discussion

Severe hyponatremia secondary to consumption of over the counter laxatives a rare complication. Additionally, diagnosis of central DI can be based on plasma hyperosmolality (>300 mOsm/L), urine hypoosmolality (<300 mOsm/L or urine/plasma osmolality ratio <1), and polyuria exceeding three liters per day [3].

Time	Event
On admission:	Serum sodium concentration of 114mmol/ with witnessed episode of generalized seizure. Prompted immediate treatment with 100 mL of 3% sodium chloride and subsequently endotracheal intubation.
2 hours post treatment	Sodium concentration of 120mmol/L. Further treatment with normal saline was discontinued as the patient was within the 4-6 mmol/L goal in a 24 hour period.
26th hour post treatment	Serum sodium was noted to 134 mmol/L and urine production increased to a net fluid output of 6,849 ml. Treatment with 4 mcg of DDAVP and D5W infusion was started with serial monitoring of sodium levels which revealed persistent overcorrection of serum Na to 134 mmol/L.
3rd Day	Patient was given an additional dose of 4 mcg of DDAVP and D5W infusion for 5 hours which resulted in correction to 131mmol/L and 128 mmol/L at 5 and 17 hrs respectively post treatment with the second dose of DDAVP.
4th Day	Patient was successfully extubated
5th Day	Discharged home with a serum sodium concentration of 138 mmol/L

Results

	11/7/2019 1536	11/7/2019 2005	11/7/2019 2208	11/8/2019 0118	11/8/2019 0503	11/8/2019 0523	11/8/2019 1541	11/8/2019 1813	11/8/2019 2044	11/8/2019 2135	11/8/2019 2211	11/9/2019 0220	11/9/2019 0226
CHEMISTRY-ROUTINE													
Glucose	148	160	103	121	104	106	109	104	107		137		131
BUN	5	4	4	6	8	8	5	6	5		5		5
Creatinine	0.59	0.63	0.56	0.63	0.68	0.67	0.58	7.60	0.65		0.69		0.80
eGFR NonAfrican Am	154.57 *	143.30 *	164.17 *	143.30 *	131.21 *	133.48 *	157.65 *	8.10 *	138.23 *		129.02 *		108.77 *
eGFR African Am	187.03 *	173.40 *	198.64 *	173.40 *	158.77 *	161.51 *	190.76 *	9.80 *	167.25 *		156.12 *		131.62 *
Sodium	114	120	121	120	119	118	121	122	134		134 *		134
Potassium	2.9	3.1	3.4	3.0	3.3	3.2	3.1	3.1	3.5		3.5		3.2
Chloride	82	92	93	93	92	92	92	92	102		102		101
Calcium	7.7	6.7	6.8	6.9	6.9	7.0	7.5	7.6	8.1		8.1		8.0
Carbon Dioxide	22	22	21	19	19	21	22	20	25		26		27
Phosphorus					1.2								
Magnesium	1.7				1.8								



As with this patient, initial labs were suggestive of hyponatremia secondary to cerebral hypersecretion of ADH, indicated by hyponatremia with elevated urine sodium and urine osmolality. However, in a matter of 24 hours, urine osmolality and urine sodium had decreased significantly, highlighting the compensatory presence of diabetes insipidus.

Central suppression of ADH was hypothesized as the compensatory drive of the subsequent volume depletion. This was believed to be due to the laxative agent, magnesium citrate, causing volume depletion triggering a compensatory increase of ADH secretion. This hypersecretion of ADH combined with the laxative triggered electrolyte imbalances with ingestion of free water resulted in severe hyponatremia. Rapid increase in intravascular volume after the 1L bolus of 0.9% NS and the emergent infusion of 100 mL of hypertonic saline, resulted in suppression of ADH secretion and presented as clinical central DI with polyuria and aquaresis. This sequelae of compensatory ADH suppression was likely the primary cause for the spontaneous overcorrection of serum sodium in the 24 hrs following initial treatment with hypertonic saline. This is further supported by suppression of the patient's polyuria and cessation of further overcorrection after the administration of DDAVP. While hyponatremia caused by polydipsia and central DI are well-known conditions, it is rare to have both occur sequentially as a result of the consumption of the OTC laxative, magnesium citrate [5]. This phenomenon of spontaneous central DI after hypovolemic hyponatremia is an unique clinical presentation that should be considered and anticipated during the management of acute hypovolemic hyponatremia and instructions for adequate hydration with electrolyte supplemented beverages during treatement of constipation with osmotic laxatives .

References:

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