Post Obstructive Diuresis Complications: A Rare Cause of Nephrogenic Diabetes Insipidus

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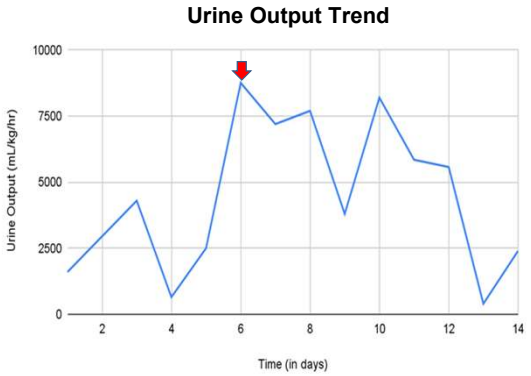
Introduction:

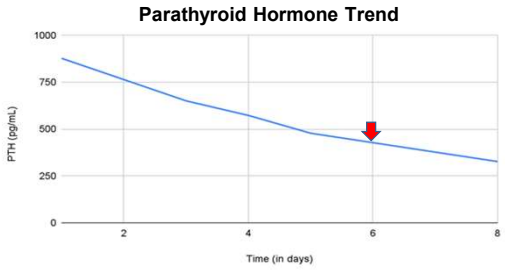
In the majority of patients, diuresis resolves once the kidneys reach homeostasis upon relief of urinary obstruction. In cases where diuresis does not cease, salt wasting and water loss continue, leading to Post Obstructive Diuresis (POD). This results in a high risk of severe dehydration and electrolyte imbalances. Some of the electrolyte imbalances associated with POD include hypomagnesemia and hypokalemia. Because these two electrolyte imbalances impair the urinary concentrating ability and decrease the collective tubule response to antidiuretic hormone (ADH), this can lead to nephrogenic diabetes insipidus. We present a case of hypomagnesemia and hypokalemia caused by POD, ultimately resulting in nephrogenic diabetes insipidus.

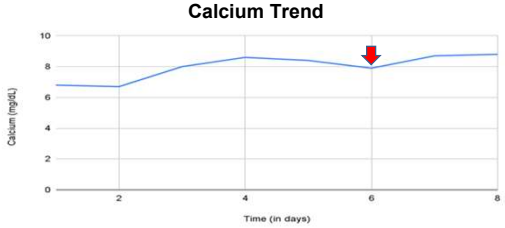
Case Description:

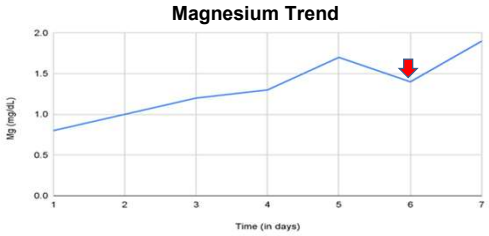
A 63-year-old male with no past medical history presented with fatigue and weakness for 2 weeks. Physical examination was significant for suprapubic tenderness. Labs were significant for BUN/Creatinine of 238 mg/dL / 7.1 mg/dL, hypertonic hyponatremia of 121 mEq/L, and a high anion gap of 29 with CO2 of 7 mmHg. The CT abdomen without contrast showed bilateral hydroureteronephrosis with enlarged prostate causing a mass effect in the urinary bladder. Foley was placed, and 850 mL of urine was relieved. He was admitted for acute renal failure from obstructive uropathy. The patient's BUN/Cr improved to 45 mg/dL / 2.6 mg/dL with Foley placement, and hyponatremia improved to 135 mEq/L with normal saline. During the patient's hospital course, his urinary output was noted to be elevated at 9L over 24 hours. Potassium was 3.0 mEq/L, magnesium 0.8 mg/dL, Uosm 250 mOsm/kg. Labs were consistent with post-obstructive diuresis leading to hypomagnesemia and hypokalemia, which caused nephrogenic diabetes insipidus. As a result of the hypomagnesemia, there was PTH resistance leading to hypocalcemia and secondary hyperparathyroidism. This was treated with calcium carbonate and magnesium repletion. Magnesium was repleted with IV magnesium and magnesium oxide tablets. The patient was initially given 200 mL/hr of normal saline for 3 days without resolution of polyuria. The patient was then given gentle hydration of normal saline at 42 mL/hr. Due to magnesium repletion, our patient's urinary output decreased to 2L upon discharge.

Trends of urine output, parathyroid hormone, calcium, and magnesium. Red arrows signify time point (Day 6) of calcium carbonate and magnesium repletion.









Discussion:

- POD is defined as >200cc of urine for at least 2 consecutive hours following obstruction relief.
- POD leads to electrolyte excretion, and our patient's hypomagnesemia and hypokalemia cause nephrogenic diabetes insipidus due to decreased ADH responsiveness in collecting tubules.
- Our case highlights the importance of monitoring electrolytes in those with post-obstructive diuresis.
- Treatment of nephrogenic diabetes insipidus caused by hypomagnesemia is by repletion of magnesium.
- Pitfall: Aggressive replacement of fluids prolonged patient's polyuria. Optimal therapy for post-obstructive diuresis consists of fluid infusion at a maintenance level.

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