College of Human Medicine
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When Creatinine Spikes and Sodium Drops: Managing Acute Kidney Injury with Severe Symptomatic Hyponatremia

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Introduction

The clinical management of patients with multiple comorbidities presenting in the setting of acute kidney injury (AKI) with severe symptomatic hyponatremia is complex. Management is often complicated by polypharmacy, impaired water excretion, and increased susceptibility to rapid shifts in serum sodium increasing the risk for adverse effects.[1]

Case Description (CD)

This case describes a 85-year-old male with history of stage 3 chronic kidney disease (CKD), heart failure with preserved ejection fraction, and hypertension on Hydrochlorothiazide and Lisinopril who presented to the emergency department (ED) with altered mental status complicated by suspected pneumonia on Azithromycin.

In the ED, he was hypotensive in the 90s/40s which improved with intravenous fluids. CMP showed severe hyponatremia at 112, low bicarbonate at 11, and AKI on CKD with a creatinine of 8.79 from baseline of 1.5 with BUN of 124. Serum osmolarity was 289, urine osmolarity was 284, and urine sodium was <20.

He was admitted to the intensive care unit and started on isotonic sodium bicarbonate at 75 ml/hr with Q2 BMPs with improvement in serum sodium to 118. Subsequent sodium was 116 and he was started on 3% saline infusion (HTS).

Urine output increased significantly and sodium increased faster than goal. As the urine output remained elevated, 1 mcg of Desmopressin was given and sodium continued to improve at goal.

Continued CD

He was transitioned to normal saline with serum sodium slowly returning to normal. Renal function and mental status improved daily with creatinine down to 1.7 from 8.79 by time of discharge with recommendations to discontinue HCTZ and obtain weekly labs for following 3-4 weeks.

Clinical Diagram

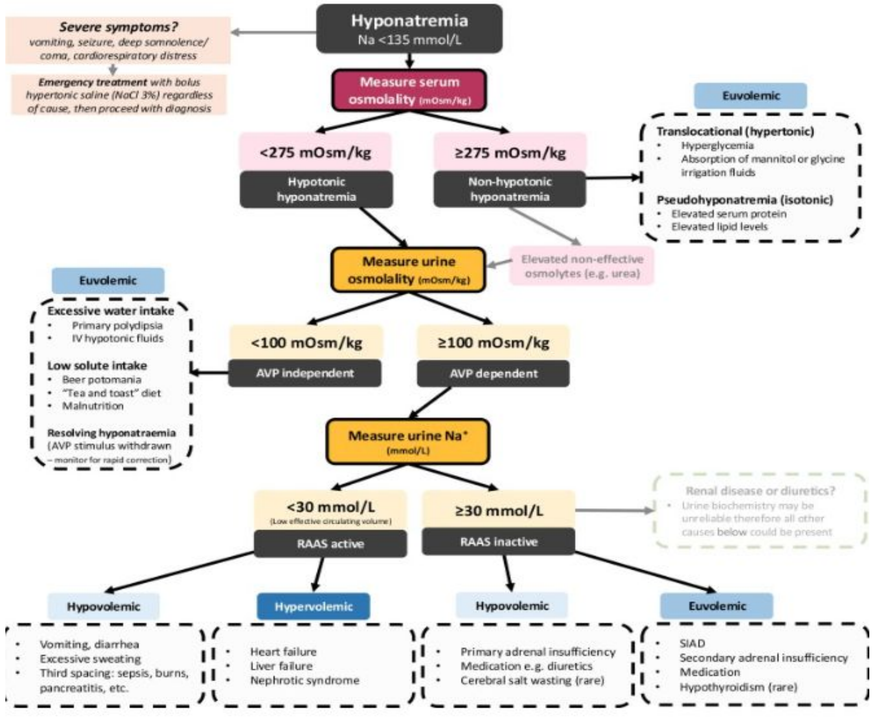


Figure 1:Hyponatremia Algorithm[2]

Discussion

This case is unique for the rare presentation of severe symptomatic hyponatremia with advanced AKI. Although the clinical presentation is likely multifactorial, a likely cause is thiazide-induced hyponatremia. Lisinopril and pneumonia can also precipitate volume depletion.

In the treatment of this patient, isotonic sodium bicarbonate was useful for correcting volume depletion and acidosis. HTS was subsequently given due to persistent hyponatremia with AMS. Desmopressin was given to halt overcorrection by limiting free water excretion as the patient started to have rapid water diuresis after volume repletion.[3]

In high risk patients with acute severe symptomatic hyponatremia, it is recommended to not increase serum sodium greater than 4-6 mmol/L in 24 hours with a strict limit of 8-10 mmol/L in 24 hours to avoid central pontine myelinolysis.[4] Thus, it is recommended to frequently monitor sodium concentration and strictly adhere to correction limits to prevent overcorrection.

References

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