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ePHONATREMIA FROM A NON-Osmotic STIMULUS, Favored to be
ACCUMULATED WATER. The patient then also
PRESENTATION of hyponatremia range from asymptomatic to
A 42-year-old man with a history of PTSD and HTN presented to
Hypertension can be broadly categorized as primary or secondary.
Presentations of hyponatremia range from asymptomatic to
Intermediate left ventricular hypertrophy with normal wall thickness
Echocardiogram (TTE) revealed the development of moderate
pH 7.49, pCO₂ 42 mmHg, pO₂ 58 mmHg. Transthoracic
92 mEq/L, bicarbonate 33 mEq/L, glucose 105 mg/dL, BUN 10
Pounds of weight gain over the past 4-6 months. On presentation,
the hospital with bilateral edema and associated shortness of breath.
Anabolic steroid intake identified through careful examination of
and metabolic alkalosis strongly suggest possible mineralocorticoid
increased mineralocorticoid activity. The presence of hypokalemia
unusual which raises suspicion for proximal tubulopathy such as
Fanconi syndrome. Due to the progression of CLL, the
restriction. Electron microscopy showed no crystal formation in
glomerular and tubular light chain (LC) restriction. However,
urine study showed increased fractional excretion of phosphorus
relatively low uric acid, we suspected Fanconi syndrome. A 24 hour
proteinuria and glycosuria. Given hypokalemia, glycosuria and
Fatigue, but otherwise was asymptomatic with normal urine output.
Transitioned to subcutaneous insulin.

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MYSTERY OF UNRESPONSIVE SEVERE METABOLIC ACIDOSIS:
Ameet Kumar1, Sujit Kumar Raostray1, Swati Arora1. 1Allegheny General Hospital, Pittsburgh, PA, United States

Recently emerging evidence shows that Sodium glucose co
transporters (SGLT2) inhibitors result in significant decline in
progression of chronic kidney disease, mortality from renal and
cardiovascular causes in both diabetics and non-diabetics. We report
a case of severe metabolic acidosis to increase awareness of side
effects of SGLT2 inhibitors in routine practice.

A 56-year-old female with history of Diabetes Mellitus II on
Metformin, Insulin, Sitagliptin and Canagliflozin, recently had
debulking surgery for ovarian cancer 4 days prior, presented to ER
with persistent nausea, vomiting and constipation x 4 days. She had
stopped taking insulin. On exam, hemodynamically stable with dilated abdomen. Labs with HCO₃ 10, AG 24, lactate 1.0, glucose
122 mg/dL. UA with ketone 3+. CT abdomen showed SBO. Nasogastric tube was placed and started on bicarb drip. Next day, her
HCO₃ dropped to 7, AG increased to 25. Serum B-hydroxybutyrate was elevated, Osmolar gap of 14. Acetaminophen, salicylic acid level
were in normal range. She denied alcohol use; there was low possibility of ethylene glycol/methanol overdose. Less likely
starvation ketoacidosis with serum albumin of 3.9 mg/dL. She was
deemed to have euglycemic diabetic ketoacidosis secondary to SGLT-
2 inhibitor. Canagliflozin was stopped and she was treated with
insulin drip. Her acidosis resolved, and subsequently she was transitioned to subcutaneous insulin.

SGLT2 Inhibitors can cause euglycemic DKA by lowering the
-glucose levels, which results in decreased insulin and increased
glucagon production that promotes ketogenesis, especially, in
patients with low insulin reserve, and in stressful situations such as
infection or surgery. It also decreases acetoacetic secretion in
kidney by non-selective inhibition. We suggest keeping euglycemic DKA as
differential for metabolic acidosis in patients on SGLT2 inhibitors, as
delay in diagnosis could prove fatal, and consider discontinuing
SGLT2 inhibitors; continue with insulin in setting of acute illness.

We believe Nephrologist will be using SGLT2 inhibitors in future;
as data is convincing to slow down the CKD progression and mortality benefit. Recommend to consider euglycemic DKA as
differential for acidosis in patients on SGLT2 inhibitors, discontinue in dehydration and in acute setting e.g. infection, surgery.