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Published In/Presented At

Cohen, R. Weaver, K. (2017, April 18). *A Unique Cause of Anion Gap Metabolic Acidosis: Euglycemia DKA*.
Poster Presented at: ACOEPS SPRING SEMINAR, Bonita Springs, FL.

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A Unique Cause of Anion Gap Metabolic Acidosis: Euglycemia DKA

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INTRODUCTION

SGLT-2 inhibitors are a new class of oral diabetes medication that have been associated with a rare complication- euglycemic DKA. This case illustrates the importance of recognizing this clinical entity that can lead to a gapped metabolic acidosis in the setting of a normal glucose level.

HISTORY

CHIEF COMPLAINT: Vomiting, weakness, palpitations

HPI: A 34 year old female with a past medical history significant for AML s/p multiple past chemo treatments, DM Type 2, HTN, anxiety and depression presented to the ED with head to toe generalized body aches x 4 days. Although her complaints ranged from chest pain to abdominal pain, she specifically came to the ED for evaluation of her fatigue after a night of vomiting >8 (non bloody) episodes and decreased po intake.

PAST MEDICAL & SURGICAL HISTORY: Dx w AML in 2011, anxiety, depression, DM II, Hysterectomy

SOCIAL HISTORY: No smoking, alcohol or illicit drug use, no history of SI/HI, poorly compliant with glucose monitoring diabetes

MEDICATIONS:

- Wellbutrin
- Aspirin
- Invokana
- Klonopin
- Levemir
- Victoza

ALLERGIES: Ambien

PHYSICAL EXAM

VITAL SIGNS: BP - 145/85 HR - 165 RR - 18 T - 99 F O₂ - 99% RA

GEN: Anxious, moaning in room

HEENT: PERRLA, EOMI, moderate DMM

PULM: CTABL.

CARDS: Sinus tachy, no M/R/G. Good distal pulses.

ABD: Soft, nondistended, nontender. +BS.

SKIN: Warm, dry, no rashes.

NEURO: AAO x 3, CN II-XII intact.

PSYCH: agitated, anxious, tearful.

LABORATORY STUDIES



AST: 87	UA: + ketones	TROP: <0.02	LACTATE: 2
ALT: 47	UPreg: Negative	TSH: 1.39	AG: 16

DIFFERENTIAL DIAGNOSIS

Having an anion gap metabolic acidosis of 16 with a bicarbonate of 8, the etiology was not readily apparent with the patient's normal lactate, absence of fever and only mildly elevated serum glucose 220.

- Infectious
- Structural (PE)
- Toxicologic

Anion Gap Metabolic Acidosis
Methanol
Uremia
Diabetic ketoacidosis
Paraldehyde
Iron, isoniazid
Lactic acidosis
Ethylene glycol
Salicylates

EXPANDED LABS

VBG: 6.91/34/99/7/95	UDS: + opiates	ETOH: Neg	BC x 2: Neg
ACETONE: 47	ASA: 4	TOX alcohols: Neg	D-dimer: 0.68
BHB: 7.16	APAP: <2	RVP: Neg	CXR: Nad
			EKG: Sinus145

HOSPITAL COURSE OF CARE

Her expanded labs ultimately revealed a worsening acidosis with positive blood and urine ketones, leading to the diagnosis of euglycemic DKA. She was admitted to the ICU on both an insulin and bicarbonate drip with dextrose containing fluids and frequent glucose checks. After her gap closed, her home medications revealed the culprit of this unique cause of DKA, her SGLT2 inhibitor which was immediately discontinued.

DISCUSSION

EUGLYCEMIC DKA: Although DKA is classically taught to include acidosis, hyperglycemia and increased plasma ketones, euglycemic DKA is basically DKA without the hyperglycemia. It is a more rare diagnosis; however, ED providers should have it on their differential of AGMA, specifically in diabetic patients on SGLT-2 inhibitors.

MECHANISM:

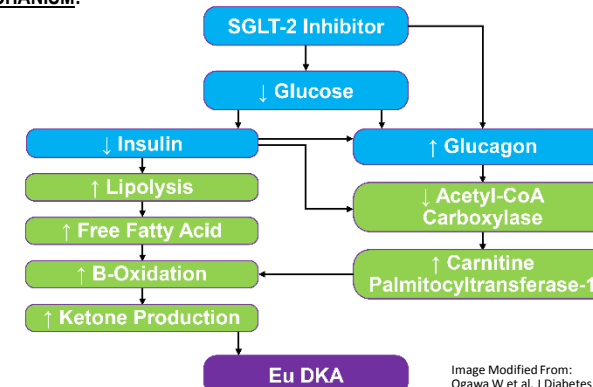


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Ogawa W et al. J Diabetes Investig 2016

SGLT-2 inhibitors block sodium glucose co transporters in the proximal renal tubules which promotes glucose excretion. Just as in normal diabetic ketotic states, when the body cannot use blood glucose for energy, ketones and acidosis will ensue; however, since the overall glucose levels in the body are lower, the pt will be euglycemic.

TREATMENT: The treatment of euDKA is practically identical to the management of DKA with a few added caveats.

- IV fluids with dextrose
- IV Insulin drip
- Frequent Glucose monitoring
- Discontinue all SGLT-2 inhibitors

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