Alcoholic Ketoacidosis: Mind The Gap, Give Them What They Need

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Introduction

- Patients with alcohol use disorder commonly present to the ED critically ill due to a myriad of underlying pathologies.
- Alcoholic ketoacidosis (AKA) should be considered in anyone with prolonged and/or binge consumption of alcohol.
- Diagnosis and proper treatment results in rapid correction of underlying metabolic derangements often followed by rapid clinical improvement.
- Failure to make the diagnosis can result in shock, hypokalemia, hypoglycemia, and acidosis.

Case Description

History: A 32-year-old male presented to the ED with 24 hours of consistent non-bloody non-bilious emesis after a 10-day drinking binge. He had nausea, chills, sweats, and inability to tolerate oral intake.

Past Medical History: Alcohol use disorder, Alcoholic cardiomyopathy with an ejection fraction of 20%, renal artery embolism, alcohol induced pancreatitis, hepatic steatosis

Meds: digoxin, entresto, eplerenone, metoprolol, pantoprazole, and warfarin; however he reports medication noncompliance for 11 days prior to presentation

Social History: Denies any other substance use, married, two children, typically drinks in binges

ROS: + chest pain, 2 minutes substernal, non-radiating, resolved

Physical Exam

BP 113/72, P 112 bpm, T 36.7°C, 98% O2 on RA, RR 20, BMI 22

Gen: Toxic appearing in moderate distress, tremulous, dry heaving unable to lie still on gurney in hallway

HEENT: Dry mucous membranes, tongue fasciculations
CV: Tachycardic
Lungs: Clear to auscultation
Abd: Generalized epigastric tenderness to palpation, soft

+ Bruins Sign

Differential Diagnosis

Pancreatitis, Alcohol induced gastritis, Alcohol withdrawal, Alcohol induced hepatitis, Acute Kidney Injury, Sepsis, Metabolic abnormality (Alcoholic ketoacidosis), Acute coronary syndrome, Pulmonary embolism

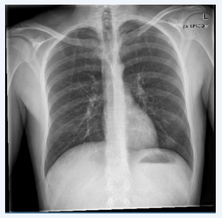
Clinical Data

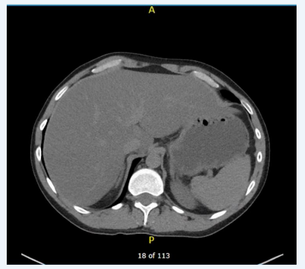
130	83	27	167
5	11	1.9	

	16.6	
17.2		241
	49.3	

90% PMNs
Neutrophils 15.6x10^3/µL
Lipase: 13 U/L
AST: 32 U/L
ALT: 22 U/L
Alk Phos: 83 U/L
Alb: 6 g/dL
Protein: 9.8 g/dL
T-Bili: 1 mg/dL

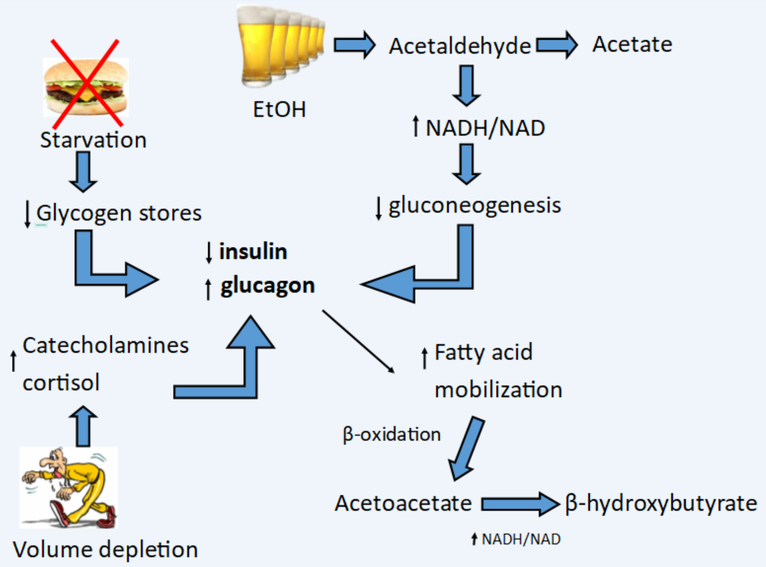
Anion Gap 36
Lactate 1.9
Salicylate, ethylene glycol, methanol not detected
Digoxin: 0.3 ng/mL
PT/INR: >120/>11
VBG: pH 7.34, pCO2 25
BNP: 66 pc/mL
Trop: 0.1 ng/mL
EKG: sinus tach





Urinalysis
Protein 2+
Ketones 3+
Urobilinogen +
RBCs 5/hpf
Hyaline casts 21 /hpf
UTox: caffeine

Pathophysiology



Adaptation of Figure 2 from Wrenn et al. The Syndrome of Alcoholic Ketoacidosis[1]

Case Discussion

Diagnostic Criteria for Alcoholic Ketoacidosis[2,3]
Binge drinking ending in nausea, vomiting, and decreased intake
Wide anion gap metabolic acidosis without alternate explanation
Positive serum/urine ketones
Low, normal, or slightly elevated serum glucose

Core Emergency Medicine Principles

- Treatment for AKA requires glucose administration, thiamine supplementation, and volume repletion.
  - D5 NS IV until rehydrated, D5 1/2NS for maintenance.
  - Thiamine 100 mg IV before glucose.
  - Supplement electrolytes PRN.
  - Continue treatment until anion gap closes, oral intake tolerated.
  - Consider other causes of anion gap if gap does not close with treatment
  - Consider sodium bicarbonate if despite treatment pH < 7.0.
- Volume repletion alone does not correct AKA as quickly as co-administration with glucose.[4,5,6]

Why This Case

- The patient's critically elevated INR could be due to several reasons: warfarin ingestion, fulminant liver failure, Vitamin K deficiency (cholestatic liver disease, chronic malnutrition).[7,8]

Vitamin K Deficiency

- Vitamin K deficiency can cause an elevation of INR due to poor function of vitamin K dependent coagulation factors.[9]
- One study found that patients with chronic alcohol use had abnormal carboxylation of prothrombin, resulting in abnormal prothrombin function. Subsequent vitamin K supplementation decreased levels of abnormal prothrombin in those same patients.[10]
- Taken together, we hypothesize our patient's INR may be elevated due to vitamin K deficiency as a result of chronic alcohol intake, causing altered prothrombin function, resulting in a supratherapeutic INR.
- Further, vitamin K administration in our patient resulted in normalization of his INR.

References

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