ALCOHOLIC KETOACIDOSIS

A WIDE ANION GAP METABOLIC ACIDOSIS MOST OFTEN ASSOCIATED WITH ACUTE CESSATION OF ALCOHOL CONSUMPTION AFTER CHRONIC USE

PATHOPHYSIOLOGY:

- Occurs when NAD is depleted by ethanol metabolism, resulting in inhibition of aerobic metabolism in the Krebs cycle, depletion of glycogen stores, ketone formation and lipolysis stimulation
    - When glycogen stores are depleted in a patient stressed by concurrent illness or volume depletion, insulin secretion is also suppressed with stimulation of counter-regulatory hormones
    - This hormonal milieu inhibits aerobic metabolism, favours anaerobic metabolism and stimulates lipolysis, which are then metabolized to ketoacids
    - Lactate levels are elevated, but not as markedly as in shock or sepsis
- Ketone production can further be stimulated in malnourished, vomiting patients or in those who are hypophosphataemic (all commonly seen in alcoholic patients)

CLINICAL FEATURES:

- Typically occurs after an episode of heavy drinking followed by vomiting and an acute decrease in alcohol and food intake
- NON SPECIFIC SYMPTOMS PREDOMINATE (SEE BELOW)

Table 221-1 Common Symptoms and Signs in Alcoholic Ketoacidosis

Symptoms	%	Signs	%
Nausea	76	Tachycardia	56
Vomiting	73	Tachypnea	49
Abdominal pain	62	Abdominal tenderness	43
Shortness of breath	20	Heme-positive stool	18
Tremulousness	20	Hepatomegaly	18
Hematemesis	19	Altered mental status	15
Dizziness	19	Hypotension	12

DIAGNOSIS:

- Diagnosis made in the appropriate clinical setting
- FBC, EUC, CMP, LFT, lactate all important
    - ↓Na, ↓PO4, ↓Mg, ↓K all common
    - May also have ↑bilirubin/liver enzymes
    - Elevated urea → volume depletion
- Without evaluation of the anion gap in ALL alcoholic patients at risk, the diagnosis can easily missed → raised anion gap is due to KETONAEMIA
- Criteria for diagnosis below

Table 221-2 Diagnostic Criteria for Alcoholic Ketoacidosis*
Low, normal, or slightly elevated serum glucose
Binge drinking ending in nausea, vomiting, and decreased intake
Wide anion gap metabolic acidosis
Positive serum ketones*
Wide anion gap metabolic acidosis without alternate explanation

Table 221-3 Differential Diagnosis for Alcoholic Ketoacidosis	
Alternative diagnosis	Alternative or concomitant diagnosis
Lactic acidosis	Methanol ingestion
Uremia	Ethylene glycol ingestion
Secondary lactic acidosis	Isopropanol ingestion
Sepsis	Pancreatitis
Hypotension	Gastritis
Salicylic acid ingestion	Upper GI bleeding
Toxic alcohol ingestion	Seizures
Diabetic ketoacidosis	Ethanol withdrawal
Starvation ketosis	Pneumonia
	Sepsis
	Hepatitis

TREATMENT:

Table 221-4 Treatment of Alcoholic Ketoacidosis
Hydration with 5% dextrose in normal saline.
Carbohydrate and fluid replacement reverses alcoholic ketoacidosis by increasing insulin levels and suppressing release of glucagon. Fluids alone do not correct alcoholic ketoacidosis as quickly as fluids and carbohydrates together.
Thiamine supplementation should also be given as a prophylaxis against Wernicke encephalopathy.
Replace electrolytes, in particular, potassium and magnesium.
Bicarbonate therapy should only be used in severe life-threatening acidosis unresponsive to fluid and glucose administration.

- Both glucose administration and volume repletion → fluid of choice is 0.9% saline plus 5% dextrose
    - Glucose stimulates insulin production, which STOPS LIPOLYSIS AND HALTS FURTHER KETONE FORMATION
- These patients ARE NOT HYPEROSMOLAR AND THUS CEREBRAL OEDEMA IS NOT A CONCERN WITH FLUID RESUSCITATION
- INSULIN IS OF NO PROVEN BENEFIT AND CAN BE DANGEROUS → alcoholics have depleted glycogen stores and normal or low glucose levels
- Hypophosphataemia can retard resolution of acidosis, as phosphorus is needed for mitochondrial utilization of glucose and oxidization of NADH
- Consider thiamine administration before glucose → Wernicke's prophylaxis