The Anxious Alcoholic

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Barbara's presentation

- Barbara, 51, is triaged with "anxiety."
- She is hyperventilating and anxious, had several vomiting episodes during the night and feels dehydrated.
- She has no other medical complaints and denies any history of diabetes.
- She appears very dehydrated and her breath has a strong fruity odour.
- Her abdomen is diffusely tender to palpation, especially in the epigastrium and right upper quadrant.
- Barbara admits that she is a recovering alcoholic, but began drinking again two weeks ago (consuming about four litres of wine a day until yesterday).
- She has had persistent vomiting since yesterday and is unable to keep anything down.

Barbara's vital signs

- Temperature: 36.7 C
- Respiratory rate: 28 breath
- Blood pressure:
- Pulse:

- L to 97.0 FL)
- g/L to 140 g/L)
- nsferase: 191 units/L (1 units/L to 41 units/L)
- ate aminotransferase: 318 units/L (10 units/L to 42 units/L)
- Gamma glutamyltransferase: 1,452 units/L (0 units/L to 40 units/L)
- Alkaline phosphatase: 147 units/L (32 units/L to 92 units/L)
- Total bilirubin: 42 umol/L (0 umol/L to 16 umol/L)
- Albumin: 43 g/L (38 g/L to 50 g/L)
- INR: 1.54 (0.8 to 1.2)
- Anion gap: 25 mEq/L (8 mEq/l) to 16 mEq/l)
- Specific analysis of serum for alcohol, methanol, formate, ethylene glycol, salicylate and acetaminophen were all negative.
- Glucose: 8 mmol/L (3.9 mmol/L to 6.1 mmol/L))
- Arterial blood gas:
  - pH 7.26 (7.35 to 7.45)
  - PCO_2 (partial pressure of carbon dioxide): 18 mmHg (35 to 45)
  - PO_2 (partial pressure of oxygen): 118mmHg (75 to 100)
  - Bicarbonate: 8 mEq/L (18 mEq/L to 23 mEq/L)
  - Lactate: 1.4 mg/dL (0.5 mmol/L to 2.2 mmol/L)

Questions & Answers

1. What's going on?

Barbara is suffering from alcoholic keto acidosis (AKA). This syndrome results from a complex interplay between the effects of alcohol metabolism, withdrawal and starvation on endogenous insulin and glucagon secretion and on stimulation of increased free fatty acid release and ketogenesis. The physiologic state is one of an ion gap metabolic acidosis and pletion, with norm se and y are nge- ical

ted in any patient who, ge or prolonged period of alcohol use, suddenly stops ingesting alcohol, most commonly because of the vomiting that accompanies alcoholic gastritis or pancreatitis. The cessation of drinking usually occurs 24 to 48 hours before presentation, so emergency department (ED) blood ethanol levels are usually normal. An easy way to remember the syndrome is that it has a picture typical of diabetic keto-acidosis (DKA), but with a normal blood sugar level. Patients tend to be tachypneic, tachycardic and anxious. In severe cases, patients may be obtunded at presentation. Persistent vomiting and dehydration is invariably present, as is abdominal pain. Peritoneal signs may be apparent and may lead to unnecessary laparotomy if the diagnosis is not considered.

Back to Barbara

Barbara was admitted to the hospital and responded well to the above regimen. A gastroscopy during her stay revealed severe gastritis, which was treated by a proton pump inhibitor. She was counseled by a social worker and was offered support to maintain sobriety after her discharge.

Table 1

Causes of anion gap metabolic acidosis
using the "MUDPILES" mnemonic

- Methanol
- Uremia
- Diabetic keto-acidosis (or alcoholic keto acidosis)
- Paraldehyde
- INH, iron
- Lactic acidosis
- Ethylene glycol, ethanol
- Salicylates, solvents, starvation

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***This department covers selected points to avoid pitfalls and improve patient care by family physicians in the ED. Submissions and feedback can be sent to* diagnosis@sta.ca.**

3. *How should AKA be managed?*

After the obligatory attention to the "ABCs" of resuscitation, the first priority is aggressive fluid administration. A preferable option is 10% dextrose in normal saline. While the volume inhibits the release of counter regulatory hormones and facilitates urinary ketone excretion, the glucose interrupts ketogenesis by stimulation of insulin secretion and repletion of glycogen stores.

Potassium should be replaced if low or normal, as correction of the acidosis may result in a rapid fall in potassium levels as extracellular potassium is driven into the cells.

Thiamine (to prevent Wernicke's encephalopathy and restore pyruvate dehydrogenase) and magnesium sulphate (to restore calcium and phosphate homeostasis) are also indicated.

Insulin is not indicated, unless the blood glucose is elevated (in which case you may be dealing with DKA). The acid-base status should normalize with the above treatment and bicarbonate administration is rarely needed.

4. *Are there any other issues?*

Firstly, other causes of anion gap metabolic acidosis must be considered (Table 1). As in DKA management, a careful search for precipitating or other coincidental pathologies must be made, including a chest and abdominal X-ray to look for pneumonia, bowel obstruction or perforated viscus, complete skin examination, a urinalysis and, if necessary, a lumbar puncture to look for evidence of infection. Most patients will respond well to treatment, but the condition may recur, a fact that should prompt extra efforts to assist the patient to stop drinking. Dx