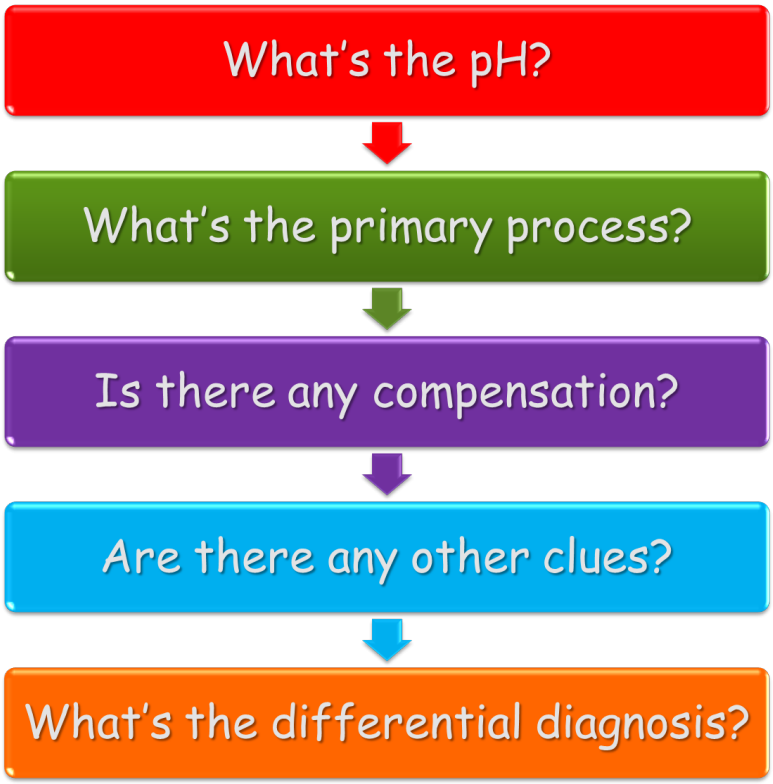
Blood Gas #1

History: A 43-year-old man is found unresponsive after absconding from the psychiatric ward. Below is his arterial blood gas following endotracheal intubation:

pH 7.03	Na 131 mmol/L
pCO2 44.3 mmHg	K 5.8 mmol/L
pO2 89.4 mmHg (FiO2 0.8)	Cl 108 mmol/L
HCO3 11 mmol/L	Glucose 21.3 mmol/L
Lactate 12.6 mmol/L	Urea 11.5 mmol/L
Osmolality 372 mOsm/L	Albumin 18 g/L

Show solution



What's the pH?

7.03 = severe acidaemia

What's the primary process?

HCO3 11 = Primary metabolic acidosis

Is there any compensation?

Expected pCO2 = 1.5 x HCO3 + 8

= 1.5 x 11 + 8

= 24.5 mmHg

Actual pCO2 is higher therefore there is inadequate compensation (ventilation) or respiratory acidosis.

Are there any other clues?

Anion gap = Na – (Cl + HCO3)

= 131 – (108 + 11)

= 12

however …

AG corrected for hypoalbuminaemia = AG + 2.5 x {(40-Albumin)/10}

= 12 + 2.5 x {(40 – 18)/10}

= 17.5

= HAGMA

Corrected sodium = Na + (glucose – 5)/3

= 131 + (21.3 – 5)/3

= 136.4

Delta gap = (AG – 12) ÷ (24 – HCO3)

= (17.5 – 12) ÷ (24 – 11)

= 0.42

This suggests a coexisting NAGMA.

Calculated osmolality = (2 x Na) + urea + glucose

= (2 x 131) + 11.5 + 21.3

= 294.8 mOsm/L

Osmolar gap = Measured osmolality – calculated osmolality

= 372 – 294.8

= 77.2

Therefore there is an elevated osmolar gap.

Electrolyte Clues

The lactate is markedly elevated. The urea, potassium and glucose are elevated. Chloride is at the upper limit of normal. Corrected sodium is normal. Albumin is markedly low.

Expected PAO_2 = (713 x FiO_2) – (pCO_2 x 1.25)

= (713 x 0.8) – (44.3 x 1.25)

= 515 mmHg

A-a gradient = PAO_2 – PaO_2

= 515 – 89.4

= 425.6

Normal A-a gradient = Age/4 + 4

= 43/4 + 4

= 14.75

Therefore there is a markedly elevated A-a gradient.

What's the differential diagnosis?

Description: This arterial blood gas of an intubated patient demonstrates severe acidaemia secondary to a primary metabolic acidosis (with both anion gap and non-anion gap components) with inadequate respiratory compensation and/or a co-existing primary respiratory acidosis. There is an elevated osmolar gap and a marked hyperlactataemia with elevated urea, potassium and glucose and hypoalbuminaemia. The corrected sodium is normal and chloride is at the upper limit of normal. There is a markedly elevated A-a gradient with high oxygen requirement.

Interpretation: In this clinical context the differential diagnosis is broad, however toxic alcohol ingestion should be considered high on the differential in view of the severe acidosis and elevated osmolar gap. Elevated lactate may

contribute to the elevated anion gap and the osmolar gap, and ketoacidosis remains a possibility. A serum ethanol should be measured to further assess the elevated osmolar gap – low albumin may suggest chronic liver toxicity from alcohol abuse. If the osmolar gap persists after correction for ethanol, a serum methanol level and urinary microscopy for crystals may give further information about the underlying cause – if doubt persists, blockade of alcohol dehydrogenase with ethanol or fomepizole (not available in Australia) and dialysis should be instituted (dialysis could be justified in view of acid-base status anyway, especially if there is an element of renal failure). An elevated A-a gradient with high oxygen requirement and inadequate ventilation may suggest underlying aspiration with respiratory failure and/or inadequate mechanical ventilation.

Additional information: The patient had a history of chronic alcohol abuse. Serum ethanol was 0.25% accounting for almost all the calculated osmolar gap. A chest radiograph demonstrated white-out of the right lung secondary to aspiration. A serum methanol level was negative and there were no urinary calcium oxalate crystals detected. The final diagnosis was aspiration pneumonia, sepsis and multiple organ dysfunction syndrome. He made a full recovery.