Critical Care Nutrition Consultancy
CriticalPoint

Metabolic Acidosis

Acidaemia
pH <7.35

- **Serum bicarbonate** → **Low <22 mmol/l**
- **Base deficit** → **High >2mEq/l**
- **pCO_2** → **High >6kPa**

Typifies respiratory, not metabolic, acidosis
Investigate respiratory causes of low pH
- Respiratory centre suppression (e.g. opioids, sedation)
- Airway obstruction
- Respiratory muscle disease/dysfunction
- Respiratory muscle innervation disorder
- Chest wall mechanics disorder
- Primary lung disease (acute or chronic)

Calculate anion gap

- **High >12 mmol/l** → **High anion gap acidosis** → **Indicates net addition of an acid**
- **Normal 8-12 mmol/l** → **Non-anion gap metabolic acidosis (hyperchloraemic acidosis)** → **Indicates net loss of bicarbonate, with exchange for chloride**; **Characterised by delta ratio <0.4**
- **Low**

Usually due to hypoalbuminaemia masking a higher anion gap
Calculate adjusted anion gap and interpret accordingly.
Adjusted anion gap = observed anion gap + 0.25 x ([normal albumin g/l]-[observed albumin g/l])

Investigate/calculate the delta ratio:
Δ (increase) in anion gap / Δ (reduction) in serum bicarbonate

- **0.4-0.8** mixed high and normal anion gap acidosis
- **0.8-2.0** pure high anion gap acidosis
- **>2.0** high anion gap acidosis with pre-existing elevated bicarbonate level

Toxic ingestion (methanol, glycols)
Paracetamol overdose
Lactic acidosis
D-lactic acidosis (may present with high anion gap)
Drugs (salicylates, INH, iron, valproate)
Uraemic acidosis (chronic renal failure)
Ketoacidosis (diabetes, starvation, alcohol)
Rhabdomyolysis

Cause can be distinguished by osmolar gap

Cause of high anion gap acidosis PLUS concurrent pre-existing metabolic alkalosis such as
- Vomiting/gastric drainage (H^+ loss)
- Volume depletion/contraction
- Cushing's/Conn's

or a compensated respiratory acidosis

Cause of high anion gap acidosis PLUS additional process associated with lowered bicarbonate levels, such as
- e.g. shocked patient with high lactate and pancreatic fistula
- Commonly occurs in renal failure, but mixed causes also possible

Determine plasma K^+ levels
NOTE: K^+ can be variable depending on when in the course of the clinical presentation of acidosis it is measured. Some hyperchloraemic acid-base disorders can fit in both categories.

- **Normal/high**

Administration of a form of HCl (substances metabolised to HCl)
- Parenteral nutrition solutions
- Ammonium chloride

Renal tubular acidosis (hyperkalaemic)
- Distal aldosterone deficiency/resistance (including Addison's disease)

Chronic renal disease
- Impaired H^+ excretion

High volume saline infusion
Drugs (incl NSAIDS, ARBs, CEIs, trimethoprim, spironolactone, amiloride, heparin)

- **low**

Renal tubular acidosis
- Proximal bicarbonate losses OR
- Distal H^+ ion excretion failure (retention)

High volume saline infusion
Toluene intoxication
Ureter diversion surgery (ileal conduit etc)
Intestinal fistula/stoma esp pancreatic/biliary (bicarbonate loss)
Diarrhoea (bicarbonate loss)
Ketoacidosis (esp in resolving phase)
Arginine/lysine supplement overdose
Drugs (acetazolamide, lithium, amphotericin B)
D-lactic acidosis (may present with normal anion gap)

To distinguish gastrointestinal cause from renal tubular acidosis calculate urine anion gap:
Urine anion gap = Urine Na^+ + Urine K^+ – Urine Cl^-
Positive value suggests renal cause, such as renal tubular acidosis.
Negative values suggests a non-renal cause, such as GIT bicarb-rich fluid losses.

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