7.8: Prevention

There are 2 aspects of prevention for a metabolic alkalosis:

- Prevention of the primary or initiating process, **and/or**
- Prevention of the factors that are involved in maintaining the alkalosis.

Patients with nasogastric drainage and pyloric obstruction should receive adequate fluid replacement using a chloride containing fluid. Patients receiving thiazide diuretics likewise need to have adequate chloride intake.

Proton pump inhibitors can be used to greatly decrease gastric acid loss $^{1,2,3}$ despite continuing nasogastric drainage.

References


All Medline abstracts: PubMed HubMed
Important Points - Chapter 7: Metabolic Alkalosis

• Metabolic alkalosis is an abnormal primary process causing a decrease in fixed acids in the blood. Buffering results in an increase in plasma bicarbonate level.

• An acute metabolic alkalosis will NOT persist long as the normal kidney rapidly increases bicarbonate excretion from the body.

• A metabolic alkalosis requires BOTH an initiating process and a maintaining process. Without an abnormal process maintaining it, the alkalosis will rapidly correct as the kidney pours out HCO₃⁻ in the urine.

• The maintaining process causing persistence of the elevated plasma bicarbonate level works by impairing renal bicarbonate excretion. The four factors which are involved in maintaining the disorder are:
  ◦ chloride depletion
  ◦ reduced GFR
  ◦ potassium depletion
  ◦ ECF volume depletion

• The initiating cause in most cases is loss of gastric acid (eg vomiting) or diuretic use. Chloride depletion is the abnormality that impairs renal bicarbonate excretion.

• All these patients (>90% of clinical cases) require chloride replacement (usually as saline solution) before they can be corrected.

• Rare causes include various adrenocortical excess syndromes.

• Hypokalaemia is the most common associated electrolyte abnormality and can be life-threatening itself.

• Metabolic alkalosis is classified into 2 major groups:
  ◦ those causes associated with chloride depletion (urinary chloride > 10 mmol/l), and
  ◦ those causes not associated with chloride depletion (urinary chloride > 20mmol/l)

• Urinary chloride levels are particularly useful in differentiating the cause in those cases where vomiting or thiazide diuretic use are uncertain.

• The compensatory response is hypoventilation but there is variation in the degree of this. Oxygen therapy should be used in most hospital patients.

• Remember: Correction usually requires replacement of chloride usually in association with fluid and potassium. In rare severe cases, hydrochloric acid infusion or use of acetazolamide may be used but there are risks.