Acid Base Online Tutorial

University of Connecticut

Metabolic Alkalosis

Primary metabolic alkalosis is characterized by an elevation in the arterial pH, an increase in the plasma HCO3- concentration, and a compensatory hypoventilation, resulting in a rise in the pCO2. It is often accompanied by **hypochloremia** and **hypokalemia**.

Pathogenesis

Metabolic Alkalosis can be induced by **a loss of hydrogen ions, transcellular H+ shift, exogenous alkali administration or by contraction alkalosis**. These factors are known as **initiator factors** because they are said to initiate the alkalosis. Under normal circumstances, alkalosis should never develop because the kidney is excellent at excreting excess bicarbonate. However in conditions where kidney function might be impaired, excretion of bicarbonate may become compromised. Metabolic alkalosis is always associated with an initiating factor and and an impairment in kidney function referred to as the **maintenance factor,** that is thought to maintain the alkalosis . See table below.

The most common maintenance factor is a **reduction in ECV** that leads to a reduction in GFR and an increase in Na and HCO3- reabsorption. Another factor that maintains alkalosis is **Hypokalemia**. Alkalosis can be both a cause and a result of hypokalemia, as will be discussed. **Mineralocorticoid excess** is another factor that initiates metabolic alkalosis. In those cases, the alkalosis is maintained by the development of hypokalemia as will be discussed.

Metabolic alkalosis that is associated with a reduction in volume responds very well to treatment with normal saline and is said to be **saline responsive**. Mineralocorticoid or hypokalemia induced alkalosis does not respond to volume administration and is said to be **saline unresponsive.** This will be discussed.

Initiating Factors	Maintenance Factors	
1. Loss of hydrogen ions from GI **2. Exogenous addition of alkali** **3. Transcellular H+ shift**	**1. Reduced ECV – decreased GFR and increased absorption of HCO3-** **2. Hypokalemia**	Saline Responsive

4. Contraction alkalosis		
1. Mineralocorticoid excess **2. Severe Hypokalemia**	**Hypokalemia**	Saline Unresponsive

Causes of metabolic Alkalosis

1) Loss of hydrogen
- **A. Gastrointestinal loss**
  - **1. Removal of gastric secretions: Vomiting or nasogastric suction**
  - **2. Chloride-losing diarrhea**
  - **3. Gastrocolic fistula**
  - **4. Villous adenoma**
  - **5. Antacid therapy, particularly if combined with cation exchange resin**
- **B. Renal loss**
  - **1. loop or thiazide diuretics**
  - **2. Mineralocorticoid excess (Primary Aldo, Cushings, steroids, licorice)**
  - **3. Post chronic hypercapnia**
  - **4. Hypercalcemia, including the milk of alkali syndrome**
- **C. H+ movement into cells**
  - **1. Hypokalemia**

2) Exogenous Alkali
- **A. Administration of $NaHCO_3$, sodium citrate, gluconate, acetate, antacids**
- **B. Massive blood transfusion**
- **C. Antacids - Milk alkali syndrome**

3) Contraction alkalosis
- **A. Loop or thiazide-type diuretics**
- **B. Sweat losses in cystic fibrosis**
- **C. Gastric losses in achlorhydria**

4) Miscellaneous
- **A. Bartter's syndrome**
- **B. Gitelman's syndrome**

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