Chapter 11 / Capítulo 11

Electrolyte and Internal Environment Imbalances in Emergencies (English Edition)
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Respiratory and Metabolic Alkalosis

Alcalosis respiratoria y metabólica

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Objectives:

- Describe the characteristics of respiratory alkalosis
- Identify the factors responsible for metabolic alkalosis
- Understand the mechanisms by which metabolic alkalosis can be corrected

RESPIRATORY ALKALOSIS

- Caused by alveolar hyperventilation (decreased pCO_2).
- In the acute phase, plasma bicarbonate decreases by ~2 mEq/L for every 10 mmHg of reduction in pCO_2; in the chronic phase, it decreases by ~4 mEq/L.
- At the renal level, there is less bicarbonate reabsorption and reduced ammonium excretion.
- Common conditions: anxiety, pneumonia, sepsis, liver failure.

METABOLIC ALKALOSIS

This condition is characterized by a primary increase in plasma HCO_3^- and extracellular pH. The clinician should ask a series of questions to facilitate or guide the diagnosis for future treatment: What caused the increase in HCO_3^-? What prevents renal excretion of HCO_3^-?

Table 11.1 summarizes the main causes of metabolic alkalosis.

Table 11.1. Causes of metabolic alkalosis

I. Hydrogen loss
- A. Gastrointestinal loss
  1. Elimination of gastric secretions, either by vomiting or nasogastric suction.
  2. Antacids in advanced renal failure.
- B. Urinary loss
  1. Loop or thiazide diuretics
  2. Primary mineralocorticoid excess (hyperaldosteronism)
  3. Post-hypercapnic alkalosis
  4. Hypercalcemia and milk-alkali syndrome
- C. Entry of H^+ into cells
  1. Hypokalemia

II. Administration of bicarbonate or an organic ion that can be metabolized into bicarbonate, such as citrate in blood transfusions

III. Contraction alkalosis
- A. Loop or thiazide diuretics in edematous patients
- B. Vomiting or nasogastric suction in achlorhydria
- C. Loss through sweat in cystic fibrosis

Source: Own elaboration

Perpetuating mechanisms

Several mechanisms perpetuate this situation. Volume and chloride depletion prevents bicarbonate excretion. Hypovolemia stimulates aldosterone, which promotes sodium reabsorption and H^+ and K^+ secretion. Hypochloremia, in turn, prevents HCO_3^- secretion by type B intercalated cells of the collecting duct.

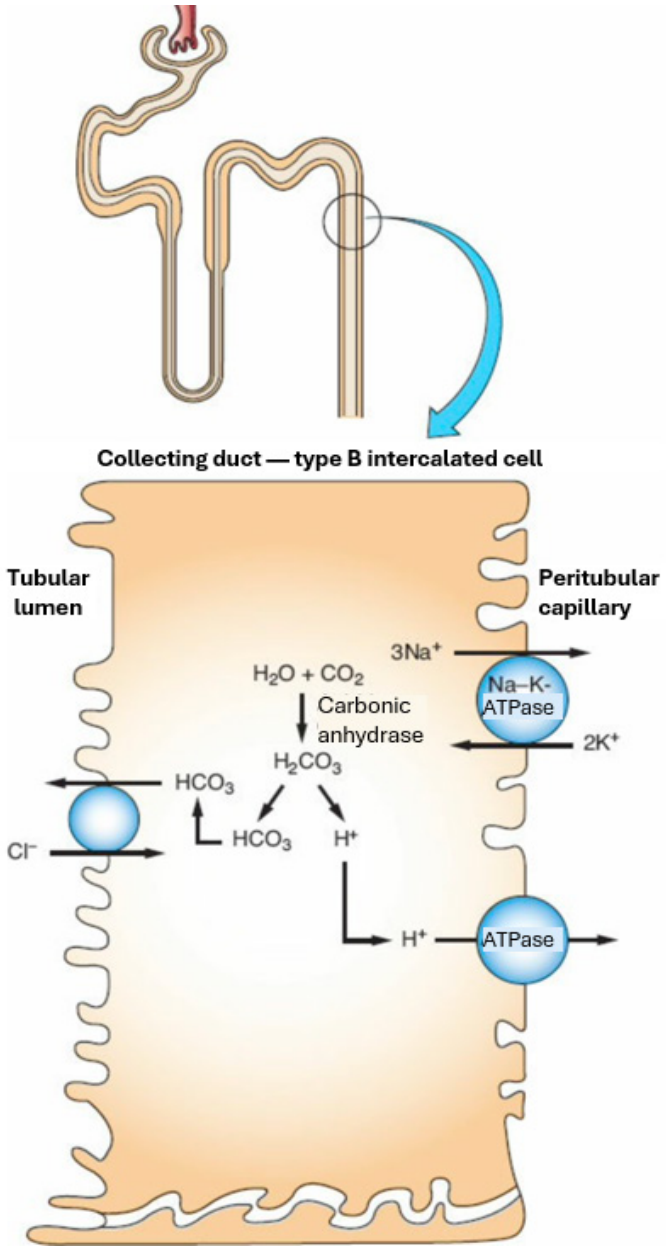

Figure 11.1. Bicarbonate secretion in the collecting tubule

Primary hyperaldosteronism and hypokalemia

- Excess aldosterone leads to:
  - Sodium retention.

- Increased secretion of H^+ and K^+ → metabolic alkalosis and hypokalemia.

- Hypokalemia worsens alkalosis through intracellular acidosis, reinforcing HCO_3^- reabsorption.
- Correction with KCl reverses these effects.

Diagnosis with urinary chloride

- Useful when the clinical history is unclear
- Urinary chloride <25 mEq/L → suggests volume depletion (vomiting, diuretics)
- Urinary chloride >40 mEq/L → indicates mineralocorticoid excess or normovolemia

Urine electrochemistry

- In the presence of bicarbonaturia:
  - HCO_3^- is excreted accompanied by Na^+ or K^+ to maintain electroneutrality.
  - This leads to loss of Na^+, K^+ and a decrease in urinary Cl^-.
- Paradoxical aciduria (urinary pH <5,5 in alkalosis) is observed when all HCO_3^- is reabsorbed due to hypovolemia and chloropenia

TREATMENT

The goal of treatment is to correct the volume, potassium, and chloride deficits. Replacement with NaCl or KCl decreases HCO_3^- reabsorption, allows its excretion, and increases tubular Cl^-, which favors HCO_3^- secretion by type B intercalated cells.

In edematous states (CHF, cirrhosis), treatment with sodium chloride is contraindicated. As alternatives, the use of IV hydrochloric acid (which can be risky) or the administration of acetazolamide, a carbonic anhydrase inhibitor that promotes $NaHCO_3$ excretion, are considered.