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Differential diagnosis of hypokalemic metabolic alkalosis

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Metabolic alkalosis is caused by retention of excess alkali, and is defined by a venous [total CO_2] >30 mmol/l or an arterial [HCO_3^-] >28 mmol/l. The increase in pH that results from the elevation in [HCO_3^-] induces hypoventilation, producing a secondary increase in arterial $PaCO_2$. Normally, the kidney responds to an increase in [HCO_3^-] by rapidly excreting excess alkali. Therefore, sustained metabolic alkalosis occurs only when some additional factor **impairs bicarbonate excretion**. Three factors play key roles: **chloride depletion**, **abnormal aldosterone secretion**, and **hypokalemia**.

The **causes** of metabolic alkalosis can be classified into three categories.
1. Secondary Stimulation of Collecting Duct Ion Transport
1) GI tract fluid loss (ex. vomiting, nasogastric suction)
2) Renal loss (ex. diuretics)
3) Genetic Impairment of Cl^--Linked Na^+ Transport (ex. Bartter syndrome, Gitelman syndrome)
2. Primary Stimulation of Collecting Duct Ion Transport
1) Primary aldosteronism (ex. adrenal adenoma, adrenal hyperplasia)
2) Apparent mineralocorticoid excess syndrome (ex. Liddle syndrome, glycyrrhizic acid)
3. Alkali Administration (ex. milk-alkali syndrome, bicarbonate administration with renal failure)

The **diagnostic process** of metabolic alkalosis can be summarized into three steps.
1. Discovery of metabolic alkalosis
pH>7.45, [HCO_3^-] >28 mmol/l
2. Evaluation of appropriate compensatory responses
PaCO2 (mmHg) = 40 + 0.7 × ([HCO_3^-] (mmol/l) - 24)
3. Identification of causes
1) History taking and physical examination: volume status, hypertension, diuretics administration
2) Check urine chloride: generally, urine chloride <10 mmol/l in chlorine deficiency (except diuretics abuse, immediately after vomiting in bulimia patient)
3) Check urine potassium (when urine chloride >20 mmol/l with volume deficiency)

(1) low urine potassium: severe potassium deficit, laxative abuse
(2) high urine potassium: diuretics abuse, magnesium deficiency, Bartter or Gitelman syndrome
4) Check renin activity and aldosterone level (hypertensive patients with sufficient body fluids)
(1) low renin with high aldosterone: primary aldosteronism
(2) low renin with low aldosterone: Liddle syndrome, glycyrrhizic acid
(3) high renin with high aldosterone: renovascular hypertension, renin secreting tumor, malignant hypertension

The **treatment** of metabolic alkalosis is to find and correct the causes, and try to correct the acid-base imbalance only in life-threatening, severe cases.
1. Chloride Depletion Metabolic Alkalosis
1) NG suction or vomiting: Administration of intravenous NaCl, KCl supplement to minimize K depletion, K-sparing diuretics (amiloride, triamterene, spironolactone or eplerenone)
2) diuretics-induced: complete repair of metabolic alkalosis is difficult.
3) Bartter, Gitelman syndrome: oral KCl (and Mg supplements in Gitelman syndrome), NSAIDs
2. Mineralocorticoid and Apparent Mineralocorticoid-Induced Metabolic Alkalosis
1) adrenal adenoma: surgical removal or medical treatment using spironolactone or eplerenone
2) other forms of primary aldosteronism: dietary NaCl restriction, K supplements, spironolactone or eplerenone
3) glucocorticoid-remediable aldosteronism: dexamethasone
4) Liddle syndrome: amiloride
5) 11β-hydroxysteroid dehydrogenase deficiency: eplerenone
3. Alkali Ingestion: identification and discontinuation