IN THE NAME OF GOD

METHABOLIC ALKALOSIS

DR GH SARVARI

PEDIATRIC NEPHROLOGIST

INTRODUCTION

Metabolic alkalosis, a disorder that elevates the serum bicarbonate, can result from several mechanisms:

I. Intracellular shift of hydrogen ions
II. Gastrointestinal loss of hydrogen ions
III. Excessive renal hydrogen ion loss
IV. Administration and retention of bicarbonate ions
V. Volume contraction around a constant amount of extracellular bicarbonate (contraction alkalosis)

Patients with preserved renal function will most often rapidly excrete excess bicarbonate in the urine. Thus, metabolic alkalosis can only persist if the ability to excrete excess bicarbonate in the urine is impaired due to one of the following causes:

I. Hypovolemia; reduced effective arterial blood volume (due, for example, to heart failure or cirrhosis)
II. Chloride depletion
III. Hypokalemia
IV. Reduced glomerular filtration rate
V. Hyperaldosteronism
VI. Combinations of these factors

CLINICAL FEATURES

I. Clinical manifestations directly related to the metabolic alkalosis are uncommon

II. Asymptomatic

III. Symptoms that are primarily related to the alkalemia, to the underlying etiology of the metabolic alkalosis, or to accompanying electrolyte abnormalities. Symptoms can, for example, be due to volume depletion (which may produce lassitude, easy fatigability, muscle cramps, and postural dizziness) and hypokalemia (which may produce muscle weakness, cardiac arrhythmias, and, if persistent, polyuria and polydipsia due to impaired urinary concentrating ability and/or direct stimulation of thirst).

IV. Muscular spasms, tetany, and paresthesia can occur with severe metabolic alkalosis

V. Severe metabolic alkalosis can cause agitation, disorientation, seizures, and coma, especially when metabolic alkalosis develops in patients with chronic liver disease.

PHYSICAL EXAMINATION

- Abnormal findings on physical examination, when present, reflect the cause of the metabolic alkalosis.
- Hypovolemic patients (eg, vomiting, diuretic therapy for hypertension) may have signs of extracellular and intravascular volume depletion, such as reduced skin turgor, low-estimated jugular venous pressure, and postural hypotension.
- By contrast, patients with effective arterial blood volume depletion due to heart failure or cirrhosis who develop metabolic alkalosis (most often due to diuretic therapy) may have peripheral edema, ascites, and/or, in heart failure, pulmonary edema.

ARTERIAL BLOOD GASES

- Metabolic alkalosis is associated with a respiratory compensation that should raise the PCO2 by approximately 0.7 mmHg for every 1 mEq/L elevation in the plasma bicarbonate concentration, thereby minimizing the increase in arterial pH.
- The respiratory compensation is most effective acutely and then becomes less effective over time

EVALUATION

- The cause is usually apparent from the history
- The most common causes of metabolic alkalosis are external loss of gastric secretions due to vomiting or nasogastric suction and diuretic therapy. These and other causes of metabolic alkalosis are often apparent from the history

EVALUATION

When the cause is not apparent from the history

- Patients who are unwilling or unable to report vomiting or diuretic ingestion

- The history may not be helpful in patients with metabolic alkalosis due to primary mineralocorticoid excess syndromes(disorders that mimic mineralocorticoid excess including glycyrrhizic acid (licorice) ingestion, ectopic adrenocorticotropic hormone (ACTH) syndrome, the syndrome of apparent mineralocorticoid excess, and genetic

- Disorders involving renal tubular transport such as Bartter, Gitelman, or Liddle's syndrome)

- Certain conditions associated with diarrhea may produce a metabolic alkalosis:

•Laxative abuse, which is often denied by the patient.
•Villous adenoma
•Congenital chloridorrhea

DIFFERENTIAL DIAGNOSIS OF METABOLIC ALKALOSIS

Normal blood pressure or hypotension:

- Low spot urine [Cl] (<20 mEq/L)Generally chloride (saline) responsive
- Vomiting/nasogastric tube suction
- Congenital chloride wasting diarrhea (chloridorrhea)
- Villous adenoma
- Chronic laxative abuse
- Cystic fibrosis
- Status post reversal of chronic hypercapnia
- Loop or thiazide diuretics – remote treatment (effect has dissipated)

NORMAL BLOOD PRESSURE OR HYPOTENSION

- High spot urine [Cl] (>20 mEq/L)Generally chloride (saline) unresponsive
- Bartter syndrome
- Gitelman syndrome
- Loop or thiazide diuretics – recent treatment (effect persists

HYPERTENSION (ALL HAVE HIGH SPOT URINE [CL] [>20 MEQ/L])

- Primary hyperaldosteronism
- Renovascular and malignant hypertension
- Exogenous mineralocorticoids
- Apparent mineralocorticoid excess
- Use of substances made with licorice root
- Liddle's syndrome
- Cushing syndrome (usually ectopic ACTH)

DIAGNOSTIC APPROACH IN UNEXPLAINED METABOLIC ALKALOSIS

- When the etiology of metabolic alkalosis is not apparent from the history and physical examination, measurement of a spot urine chloride, urine sodium, and urine pH can be helpful.

THANKS FOR ATTENTION