Metabolic Alkalosis

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16.1 Definition

Metabolic alkalosis is a metabolic condition with an elevated pH beyond the normal range (7.35–7.45). This is the result of primarily increased bicarbonate plasma concentrations or decreased hydrogen ion concentration leading to a relative excess of plasma bicarbonate. Secondary or compensatory processes that cause an elevation in plasma bicarbonate should be separated from primary processes [1, 2].

16.2 Pathophysiology

Three main mechanisms lead to the development of a metabolic alkalosis:

1. Net loss of hydrogen ions from the extracellular fluid (ECF), either gastrointestinal or renal
2. Net addition of bicarbonate or bicarbonate precursors to the ECF
3. External loss of fluids containing high chloride but low bicarbonate concentrations (leading to the so-called contraction alkalosis)

The initiating processes for metabolic alkalosis may be a gain of alkali in the ECF (extracellular fluid) from an exogenous or an endogenous source or the loss of

Conflict of Interest
The authors declare that there is no conflict of interest in relation to this article.

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protons from ECF via kidneys or via the stomach. Beside hepatic metabolism of citrate, lactate or acetate to bicarbonate can cause a brief metabolic alkalosis. Chloride depletion reduced glomerular filtration rate (GFR), potassium depletion and ECF volume depletion can lead to persisting metabolic alkalosis [3, 4].

In some disorders (e.g. vomiting), volume depletion and potassium depletion may coexist. Severe potassium depletion alone can cause a metabolic alkalosis but typically of a mild to moderate degree. The mechanism is related to an intracellular shift of H+ in exchange for K+. While alkalosis is generated predominantly due to non-renal mechanisms, renal mechanisms are frequently involved in potassium depletion [5].

Volume depletion is implicated in maintenance of alkalosis since hypovolaemia is associated with increased fluid and sodium reabsorption in the proximal renal tubule. Bicarbonate is reabsorbed in preference to chloride, leading to maintenance of alkalosis [6]. Nevertheless, the coexisting chloride depletion is the most important factor for the persistence of alkalosis.

Diuretics can cause excess renal loss of nonvolatile (= fixed) acid anions and result in alkalosis. Their use can lead to depletion of chloride, water, and potassium. These factors together maintain the alkalosis.

Renal compensation for metabolic alkalosis consists of increased excretion of bicarbonate, as the filtered load of bicarbonate exceeds the ability of the renal tubule to reabsorb it, which is only efficiently excreted if concentration exceeds 24 mmol/l. Impairment of renal bicarbonate excretion can in turn cause persistence of the metabolic alkalosis. Since renal compensation for metabolic alkalosis is much more effective than respiratory, metabolic alkalosis rarely occurs in healthy renal status. However, in pathological renal status, compensatory mechanisms for metabolic alkalosis are mainly respiratory. Hypoventilation leads to the retention of carbon dioxide (CO_2), which is then transformed to carbonic acid, thus decreasing pH. In turn, PCO_2 is increased inhibiting hypoventilation by stimulating central chemoreceptors sensitive to the partial pressure of CO_2 increasing respiration again [7–9].

In urological care metabolic alkalosis may also be caused by excess intake of bicarbonate applied for correction of metabolic acidosis related to continent urinary diversion.

16.3 Diagnostic and Differential Diagnosis

While dehydration is the most prominent clinical symptom of metabolic alkalosis, symptoms often may not be noticeable [10]. Laboratory tests show blood pH >7.45. Levels of potassium, sodium, and chloride fall below normal ranges. Bicarbonate levels in the blood will usually exceed 29 mEq/l. Urine pH may rise to 7.0.

Assessment of urinary chloride can be useful and define two subgroups: Firstly, urinary chloride levels lower than 10 mmol/l usually originate from previous thiazide diuretic therapy, from vomiting, or in fewer cases from volume depletion with

Table 16.1 Differential diagnoses of metabolic alkalosis

Addition of base to ECF
Milk-alkali syndrome
Excessive $NaHCO_3$ intake
Recovery phase from organic acidosis (excess regeneration of HCO_3)
Massive blood transfusion (due metabolism of citrate)
Chloride depletion
Loss of acidic gastric juice
Diuretics
Post-hypercapnia
Excess faecal loss (e.g. villous adenoma)
Potassium depletion
Primary hyperaldosteronism
Cushing's syndrome
Secondary hyperaldosteronism
Some drugs (e.g. carbenoxolone)
Kaliuretic diuretics
Excessive liquorice intake (glycyrrhizic acid)
Bartter's syndrome
Severe potassium depletion
Other disorders
Laxative abuse
Severe hypoalbuminaemia

increased proximal tubular reabsorption of bicarbonate or from saline infusion. Secondly, urinary chloride levels higher than 20 mmol/l originate from severe hypokalaemia, current diuretic therapy, or Bartter's syndrome and are often associated with volume expansion and resistance to therapy with saline infusion. The urinary chloride/creatinine ratio may be elevated in extra-renal alkalosis [6]. Table 16.1 shows differential diagnoses for metabolic alkalosis.

16.4 Treatment

While the underlying cause of metabolic alkalosis must be corrected, symptomatic treatment focuses on correcting the aforementioned imbalances. Intravenous correction of hypovolaemia, chloride, or potassium is indicated in more severe cases. Drugs to regulate blood pressure or heart rate or to control nausea and vomiting might be given. Vital signs like pulse, respiration, blood pressure, and body temperature will be monitored [10]. Figure 16.1 demonstrates the typical pathway of metabolic alkalosis.

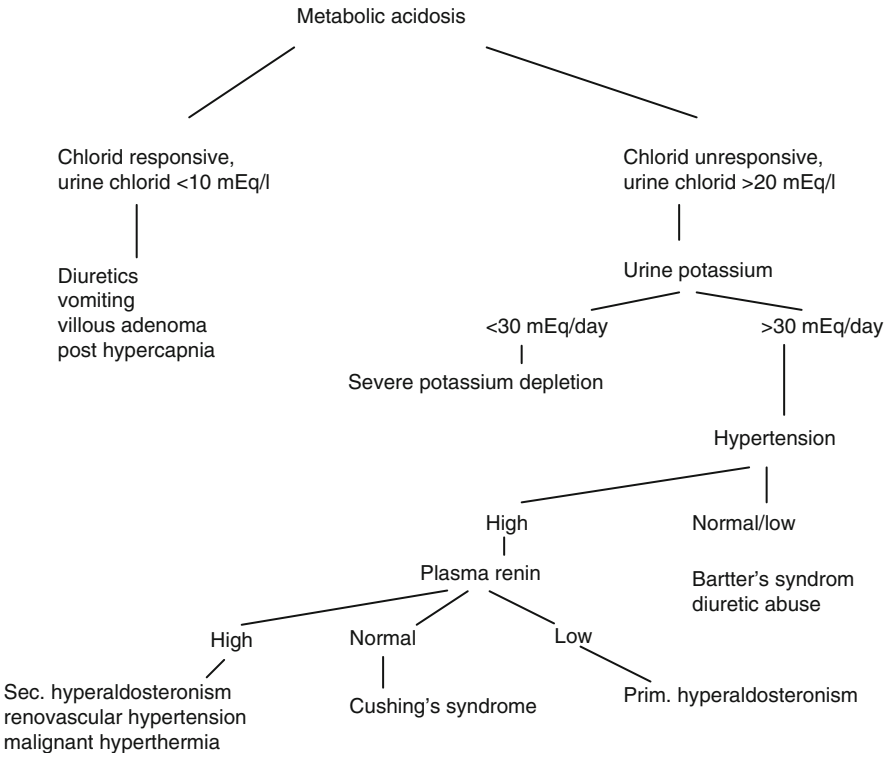

Fig. 16.1 Clinical pathway of metabolic alkalosis [6, 7, 11]

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