Approach to Metabolic Alkalosis

Jennifer T. Soifer, MD*, Hyung T. Kim, MD

KEYWORDS

- Metabolic alkalosis • Chloride depletion • Mineralocorticoid excess syndrome
- Apparent mineralocorticoid excess syndrome

KEY POINTS

- Metabolic alkalosis is a common disorder amongst patients presenting to the emergency department.
- Patients often present without any symptoms but can develop neurologic and respiratory symptoms as their alkalosis worsens.
- The mainstay of treatment is supportive care; however, once a specific cause is identified, it should be addressed to correct the alkalosis and any electrolyte abnormalities.

INTRODUCTION

Metabolic alkalosis is defined as increased arterial pH greater than 7.42 or an increase in serum bicarbonate to greater than 30 mmol/L. It is the result of an increase in bicarbonate production, a decrease in bicarbonate excretion, or a loss of hydrogen ions. In a person with normal renal function, the regulatory response of the kidney leads to a decrease in bicarbonate by excreting the excess alkali. Metabolic alkalosis can be sustained only when renal regulation is disrupted.[1,2]

Metabolic alkalosis is common, accounting for half of all acid-base disorders in hospitalized patients.[3] Although most of the patients can tolerate mild metabolic alkalosis, severe alkalosis can have significant adverse effects on cellular function and can lead to increased mortality.[4] Patients with mild to moderate metabolic alkalosis, with serum bicarbonate levels less than 40 mmol/L, are typically asymptomatic. However, mortality approaches 45% when patients develop arterial pH of 7.55% and 80% when the pH is greater than 7.65.[5]

CLINICAL PRESENTATION

The workup of any patient in the emergency department should always begin by obtaining a history. Any history of excessive vomiting or diarrhea, recently added or

Department of Emergency Medicine, Keck School of Medicine, University of Southern California, 1200 North State Street, Los Angeles, CA 90033, USA
* Corresponding author.
E-mail address: jtsoifer@yahoo.com

Emerg Med Clin N Am 32 (2014) 453–463
http://dx.doi.org/10.1016/j.emc.2014.01.005 **emed.theclinics.com**
0733-8627/14/$ – see front matter

increased dosages of diuretics, recent history of surgery that required nasogastric tube insertion, or a history of family members with excessive thirst and urination as children or fatigue and muscle wasting as adults may lead the clinician to a diagnosis of metabolic alkalosis.

Patients with mild to moderate metabolic alkalosis, with serum bicarbonate levels less than 40 mmol/L, are typically asymptomatic. When symptoms do occur, they are usually a consequence of an electrolyte abnormality rather than the alkalosis itself. For instance, in patients with ischemic heart disease, hypokalemia increases the risks of developing cardiac arrhythmias. Other symptoms include paresthesias, muscular cramping, and tetany, which are again likely caused by electrolyte abnormalities associated with the alkalosis. As bicarbonate levels increase to 45 mmol/L, the physiologic compensation is to correct the alkalosis by hypoventilation, leading to hypoxemia, especially in patients with chronic obstructive pulmonary disease. Once bicarbonate levels increase higher than 50 mmol/L, patients may develop seizures, altered mental status, and coma. Therefore, identifying the cause of this acid-base disorder and initiating specific treatment is important.

DIAGNOSIS

The diagnosis of metabolic alkalosis is sometimes a clinical one, but it is often found incidentally of laboratory work. On a routine serum chemistry panel, a bicarbonate level higher than 30 mmol/L in association with hypokalemia is pathognomonic for metabolic alkalosis. Once the diagnosis of metabolic alkalosis has been established, it is important to fully characterize the disorder by obtaining an arterial or venous blood gas to obtain a pH and $Paco_2$ measurement (partial pressure of carbon dioxide, arterial), especially if the alkalosis is severe with bicarbonate levels greater than 40 mmol/L. As the serum bicarbonate level increases, there is an increase in $Paco_2$ which is caused by compensatory hypoventilation.

Another useful tool in the diagnosis of metabolic alkalosis is the measurement of the urine chloride concentration. Urine chloride concentration of less than 10 mmol/L is usually observed in chloride-responsive metabolic alkalosis, whereas a concentration greater than 30 mmol/L is usually seen in non–chloride-responsive metabolic disorders such as mineralocorticoid excess or apparent excess syndromes. Patients with metabolic alkalosis associated with severe hypokalemia, volume depletion caused by diuretic use, Bartter and Gitelman syndromes, or alkali ingestion can have a urine chloride concentration in an indeterminate range between 10 and 30 mmol/L. There is little usefulness of urine chloride concentration alone as a diagnostic tool if the result is in this range.[1,6]

CAUSE AND MANAGEMENT

There are several possible causes of metabolic alkalosis in patients. The most common causes are listed in **Box 1**. The major decision point in making the diagnosis is based on volume status and blood pressure. An algorithmic approach to the workup and management of metabolic alkalosis is detailed in **Fig. 1**. Patients with evidence of volume depletion who are either normotensive or hypotensive are more likely to have metabolic alkalosis caused by chloride depletion. If the cause is clear, such as a history of vomiting, nasogastric suction, or diuretic use, the appropriate management is to treat the underlying disorder. If the cause is unclear, a trial of chloride repletion often helps elucidate the cause. Metabolic alkalosis that is easily corrected is usually caused by a simple chloride depletion disorder. If it is not easily corrected, then a hereditary chloride wasting disorder such as Gitelman syndrome or Bartter syndrome