Toxicologic Acid-Base Disorders

Sage W. Wiener, MD^{a,b,c,*}

KEYWORDS

• Acid-base • Acidemia • Alkalemia • Anion gap • Delta gap • Osmol gap • Toxicity

KEY POINTS

- Draw a blood gas with lactate and a chemistry panel in patients presenting after poisoning of unknown etiology.
- The toxicologic differential diagnosis of respiratory alkalosis, respiratory acidosis, metabolic alkalosis, and nonanion gap metabolic acidosis is fairly narrow.
- When approaching the patient with an anion gap metabolic acidosis, check for lactate first and ketones second, as these account for the vast majority of patients with anion gap metabolic acidosis.
- Patients with alcoholic ketoacidosis rapidly improve with fluids, dextrose, and thiamine.
- The osmol gap can sometimes be an important clue, but a normal osmol gap never excludes toxic alcohol poisoning.

INTRODUCTION

Interpretation of a patient's acid-base status can be critical to the evaluation of the poisoned patient. Many toxins will lead to a characteristic acid-base changes, and a blood gas analysis helps to establish a differential diagnosis that can be instrumental in narrowing down the potential cause of toxicity.

BASIC ACID-BASE PHYSIOLOGY

Human respiration is regulated by the brainstem to attempt to maintain a constant partial pressure of carbon dioxide (Pco_2) of approximately 40 mm Hg. Carbonic anhydrase converts carbon dioxide and water to carbonic acid, with a concentration of carbonic acid linearly related to the Pco_2 . Carbonic acid can dissociate to

E-mail address: sagewiener-em@yahoo.com

Emerg Med Clin N Am 32 (2014) 149–165 http://dx.doi.org/10.1016/j.emc.2013.09.011 0733-8627/14/\$ – see front matter © 2014 Elsevier Inc. All rights reserved.

emed.theclinics.com

Disclosures: Nothing to disclose.

^a Department of Emergency Medicine, SUNY Downstate Medical Center, 450 Clarkson Avenue, Box 1228, Brooklyn, NY 11203, USA; ^b Department of Emergency Medicine, Kings County Hospital Center, 451 Clarkson Avenue, Brooklyn, NY 11203, USA; ^c New York City Department of Health and Mental Hygiene, New York City Poison Control Center, 455 First Avenue, New York, NY 10016, USA

^{*} Department of Emergency Medicine, SUNY Downstate Medical Center, 450 Clarkson Avenue, Box 1228, Brooklyn, NY 11203.

bicarbonate and hydrogen ion (a proton). This equilibrium is the primary buffering system of the body, and it is governed by the Henderson-Hasselbalch equation (Fig. 1). Acidemia (pH <7.35) results from either excess carbon dioxide (acid) or a paucity of bicarbonate (base). Alkalemia (pH >7.45) results from either a low Pco_2 or an excess of bicarbonate.

INTERPRETATION OF THE ARTERIAL BLOOD GAS Identifying the Primary Acid Base Disorder

Interpretation of the arterial blood gas (ABG) starts with an assessment of the pH for acidemia or alkalemia. Most typically, a blood gas will have been drawn because of a suspicion of metabolic acidosis due to a low bicarbonate on a serum chemistry analysis. When this occurs, the pH can be used to distinguish metabolic acidosis from respiratory alkalosis as the primary process. If the serum bicarbonate is low and the pH is low, the primary process is a metabolic acidosis. If the pH is high, then the primary process must be a respiratory alkalosis. When the serum bicarbonate is high, a low pH indicates a respiratory acidosis, whereas a high pH indicates a metabolic alkalosis.

Determining Whether a Respiratory Disorder is Chronic or Acute

If there is a respiratory acidosis or alkalosis, the next step is to determine whether the process is acute or chronic. In an acute respiratory acidosis or alkalosis, for every 10 mm Hg change from 40 mm Hg in the Pco_2 , the pH should change from 7.40 by 0.08. If the process is chronic, the pH should change by approximately 0.03 for every 10 mm Hg change in the Pco_2 .

Determining Whether Compensation is Appropriate

If the primary process is a metabolic acidosis or alkalosis, it must next be determined whether respiratory compensation is appropriate. For a metabolic acidosis, this can be calculated using Winter's formula. The expected Pco_2 when compensation is appropriate should be approximately 1.5 times the serum bicarbonate concentration plus 8.

Predicted Pco_2 = (1.5 × [observed HCO_3^{-}]) + 8 ± 2

$$CO_{2} + H_{2}O \xleftarrow{carbonic anhydrase} H_{2}CO_{3}$$
$$H_{2}CO_{3} \longleftrightarrow HCO_{3}^{-} + H^{+}$$
$$[H_{2}CO_{3}] = P_{CO2} \times 0.03$$
$$pH = \log 6.1 \times \frac{[HCO_{3}^{-}]}{[H_{2}CO_{3}]}$$

Fig. 1. Relationship among carbon dioxide, carbonic acid, and bicarbonate. Carbon dioxide and water are converted to carbonic acid by carbonic anhydrase. Carbonic acid is in equilibrium with bicarbonate, a relationship governed by the Henderson-Hasselbalch equation. The concentration of carbonic acid is a linear function of the partial pressure of carbon dioxide.

Download English Version:

https://daneshyari.com/en/article/3236903

Download Persian Version:

https://daneshyari.com/article/3236903

Daneshyari.com