

Case Report

Pseudo Metabolic Acidosis in the Setting of Hypertriglyceridemia: A Case Report

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ABSTRACT

Low serum bicarbonate, with or without an elevated anion gap, can indicate metabolic acidosis. High anion gap metabolic acidosis is due to the unmeasured anions. Hypertriglyceridemia can also be associated with spuriously low serum bicarbonate levels due to lab interference. We present a 44-year-old woman who presented with anion gap metabolic acidosis in the presence of severe hypertriglyceridemia.

Keywords

Hypertriglyceridemia; Acid-base disorders; Low serum bicarbonate; Metabolic acidosis.

INTRODUCTION

Low serum bicarbonate levels are seen in multiple acid-base disorders, most commonly seen in conditions such as diabetic ketoacidosis (ketones), severe sepsis (lactic acid), and renal failure (organic acid anions). We report a patient with high anion gap metabolic acidosis with low bicarbonate levels and hypertriglyceridemia due to interference created by the high-levels of circulating lipids in the estimation of serum bicarbonate concentration.

CASE PRESENTATION

A 44-year-old woman presented to the emergency department with syncope at home. Past medical history was notable for chronic obstructive pulmonary disease (COPD), ongoing cigarette smoking and alcohol use. Upon examination, the patient was awake and alert, vital signs were unremarkable, with a normal respiratory pattern. Laboratory evaluation revealed mild hyponatremia with profound high anion gap metabolic acidosis, bicarbonate levels < 8 mEq/L and anion gap >22. Arterial blood gas (ABG) was obtained which showed normal pH and close to normal bicarbonate levels. Thyroid stimulating hormone (TSH) was normal, lipid panel revealed total cholesterol of 460 mg/dL, triglyceride levels of 3,231 mg/dL. Work up for syncope including orthostatic vi-

tal signs, electrocardiogram (EKG) and echocardiogram was unremarkable. Continuous telemetry monitoring did not reveal any arrhythmias. Given clinical stability of the patient and near-normal bicarbonate levels on blood gas, the low bicarbonate levels were felt to be factitious in the setting of hypertriglyceridemia. Table 1 shows all laboratory results. The patient was started on atorvastatin

Table 1. Laboratory Data

pH	7.41
pCO ₂ mmHg	30.3
pO ₂	61
Arterial HCO ₃ mEq/L	19
Na mEq/L	134
K mEq/L	3.4
Cl mEq/L	105
Serum HCO ₃ mEq/L	<8
Anion gap	22
Glucose mg/dL	133
Total cholesterol mg/dL	460
Triglycerides mg/dL	3,231
TSH microunit/mL	1.19

and discharged home with a plan for an outpatient endocrinology assessment for her lipid disorder.

DISCUSSION

The presence of anion gap metabolic acidosis during the evaluation of patients must prompt further work up to identify the etiology. Serum bicarbonate levels are measured as total carbon dioxide whereas the bicarbonate levels resulted in an arterial blood gas is calculated using Henderson-Hasselbalch equation. Discrepancy between these two values should be investigated further to determine the etiology for such an occurrence. The most common reasons for such inconsistencies are significant hyperproteinemia as seen in certain hematological disorders causing paraproteinemia, and significant hyperlipidemia often associated with genetic or familial lipid disorders. The interference caused by lipids in the enzymatic autoanalyzer is recognized as a possibility for the low serum bicarbonate levels.¹ Excessive amounts of circulating triglycerides can increase the turbidity of the sample, hence leading to erroneous lab values.² Circulating lipids can also lead to volume displacement, thus reducing the aqueous phase of the sample which can affect the levels of solutes such as sodium, potassium and chloride leading to factitiously low values.³ In the our case, the patient's clinical stability was reassuring, leading to the suspicion that her profound metabolic acidosis was a spurious finding, which was subsequently confirmed on an arterial blood gas analysis. Subsequently, we were able to identify the interfering agent to explain this discrepant finding. The discovery of severe hypertriglyceridemia was not only beneficial to the patient to avoid inappropriate treatment based on a spurious lab finding, but also to identify a previously undiagnosed lipid disorder which may have led to future morbidity to our patient.

CONCLUSION

In conclusion, when encountering lab findings which do not ap-

pear to correlate with the clinical status of the patient being assessed, an interfering agent should be suspected to explain the likely spurious finding. Caution must be exercised to confirm the accuracy of such lab abnormalities prior to initiating inappropriate and potentially harmful treatments. Once a discrepancy is established by alternative means of testing, additional investigations should be undertaken to identify the etiology of interference in order to treat the underlying disorder.

CONSENT

The patient described herein had given consent to the use of de-identified patient data for use in research and education.

CONFLICT OF INTEREST

The author(s) declared no potential conflicts of interest with respect to the research, authorship and/or publication of this article.

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