Albuterol-induced Lactic Acidosis Misdiagnosed As Sepsis – A Case Report

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Introduction

- Lactic acidosis is often attributed to sepsis, however it can result from other mechanisms.
- Type B lactic acidosis is understated in the hospital. Elevated lactate level should be interpreted together with appropriate clinical picture.
- Albuterol is commonly used in the emergency department (ED) for acute management of obstructive lung diseases.
- We present a patient with lactic acidosis resulting from excessive use of an inhaled beta-2-agonist.

Background

- Lactic acid is the end product of anaerobic metabolism of glucose.
- In general, causes of lactic acidosis can be divided into two groups. Type A lactic acidosis is due to marked tissue hypoperfusion and hypoxia, with resulting anaerobic glycolysis. Examples of type-A lactic acidosis include sepsis, circulatory insufficiency (shock, cardiac failure), hypovolemia, severe anemia, regional ischemia (limb, mesenteric), or cardiopulmonary arrest.
- In type B lactic acidosis, systemic impairment in oxygenation does not exist or is not readily apparent. Common causes of type-B lactic acidosis are malignancies, hepatic failure, medications (beta-adrenergic agonist, metformin, epinephrine), HIV, trauma, inborn errors of metabolism and ethanol intoxication.

Case presentation

- Our patient is a 59-year-old African-American female with history of reactive airway disease who presented to the ED with a 2-week history of shortness of breath associated with a non-productive cough and chest tightness.
- Prior to presentation, she used her albuterol-ipratropium nebulizer 3-4 times daily for two days. Vitals signs were within normal limits and physical examination was only remarkable for diffuse wheezing and decreased air entry on auscultation of both lungs.
- Initial lab work, including complete blood count, metabolic panel and venous blood gas were unremarkable. While initial lactate was elevated at 3.4 mMol/L (n < 2.2), repeat level obtained 5 hours later increased to 12.9 mMol/L.
- Of note, the patient received a total of approximately 18-20 mg of albuterol in the interim. Despite the low suspicion for infection, the patient received one dose of broad spectrum antibiotics.
- Throughout her hospitalization, the patient remained afebrile, white cell count remained normal, respiratory viral panel was negative and all blood and urine cultures were negative for infection. Repeat lactate level at 10, and 17 hours later were decreased to 8.6, and 2.2 respectively.
- In the setting of a negative infectious workup and continued hemodynamic stability, lactic acidosis was postulated to be secondary to excessive inhaled β-agonist therapy. Patient was discharged home the next day after her lactic acidosis resolved.

Case presentation

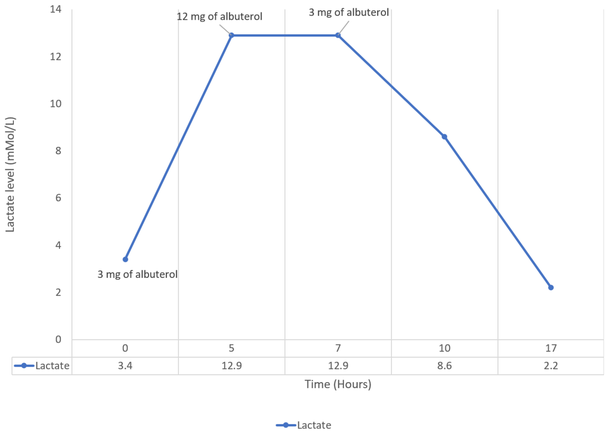

Figure 1.Lactate level over time

Discussion

- Lactic acidosis raises concern for sepsis, especially in the setting of exacerbation of reactive airway disease, as presented in this case.
- However, alternative etiologies should be sought in the presence of continued hemodynamic stability and negative infectious workup to avoid excessive and possibly harmful medical interventions.
- Albuterol, an inhaled beta-2-agonist, is an uncommon culprit in type-B lactic acidosis. The mechanism behind this phenomenon is not fully understood. Proposed mechanism is shown in figure 2.
- In this case, we believe that excessive albuterol therapy contributed to her elevated lactate level. She has no evidence of hypoxemia or tissue hypoperfusion. Her liver function tests were within normal limits.

Discussion

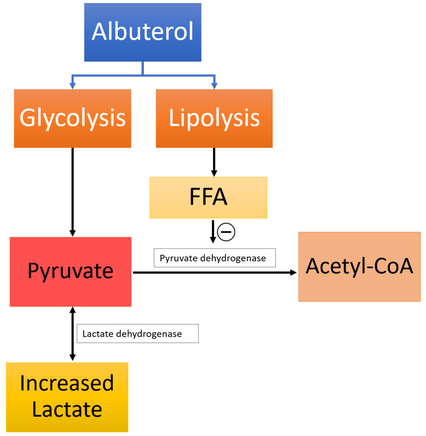

Figure 2 . Proposed mechanism of albuterol induced lactic acidosis

- Her lactic acidosis developed after excessive albuterol treatment and resolved without any further doses of β-agonist therapy.
- In summary, beta-agonist therapy is an under recognized causes of lactic acidosis that can complicate clinical evaluation and management of patients with exacerbations of reactive airway disease.

References

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