Proceedings of UCLA Healthcare
-VOLUME 16 (2012)-

CLINICAL VIGNETTE

Albuterol-Induced Lactic Acidosis: A Case Report

Stephanie B. Berman MD and Jessica S. Liao MD

Case Report

A 61-year-old Caucasian female with history of ulcerative colitis status post pancolectomy with ileostomy and history of mild-intermittent asthma presented to the Emergency Department with complaints of progressive shortness of breath, wheezing and concurrent chest tightness. Her symptoms began 2 hours prior while walking on the beach. One month prior to presentation she had undergone an ileostomy site repair and her post-operative course was complicated by symptoms due to asthma exacerbation. No additional work-up was completed at that time and the patient's symptoms resolved with routine interventions. She denied any recent fevers, chills, palpitations, leg swelling or cough.

The patient's vital signs were remarkable for respiratory rate of 40 and oxygen saturation of 98% on 3 liters of oxygen via nasal cannula while in the Emergency Department. On examination, the patient was in moderate respiratory distress, speaking three to four word sentences. She had good air movement without wheezes. Electrocardiogram demonstrated normal sinus rhythm with 1st degree AV block. Laboratory values on admission were unremarkable with anion gap of 10 and negative cardiac enzymes. A CT angiogram was performed and was negative for pulmonary emboli. Due to persistent tachypnea, the patient was started on continuous albuterol nebulizer treatments. Arterial blood gas showed pH 7.44, pCO2 34mmHg, pO2 186mmHg, and bicarbonate 23 mEq/L. Because the patient was symptomatically improving, she was maintained on continuous albuterol nebulizer treatments overnight.

A repeat arterial blood gas 12 hours after starting continuous albuterol nebulizers showed pH 7.28, pCO2 40mmHg, pO2 172mmHg, and bicarbonate 18mEq/L consistent with metabolic acidosis. Repeat laboratory values were remarkable for total CO2 of 17, anion gap of 21, and lactate of 84. The patient continued to have mild tachypnea but had good air movement, speaking in full sentences with no wheezes. Patient denied abdominal pain, had a benign abdominal exam, and had normal stool output per ostomy. Given the lack of wheezing and otherwise stable exam, albuterol therapy was discontinued and patient was closely observed given her abnormal laboratory values but otherwise benign exam.

Six hours after discontinuation of the continuous albuterol therapy the patient's anion gap returned to normal and lactate decreased to 15. She was then breathing comfortably on room air with resolution of dyspnea.

Discussion

Although the most frequent acid-base abnormality associated with an asthma exacerbation is respiratory alkalosis and subsequent respiratory acidosis from hyperventilation and then fatigue, lactic acidosis can also be a finding in patients with severe asthma exacerbations treated with β2 agonist therapy. In general, lactic acidosis can be classified into two distinct types. Type A lactic acidosis due to inadequate oxygen delivery to tissues leading to increased anaerobic glycolysis results in lactate accumulation; as seen in ischemia and septic shock where lactate levels are often used as a marker of disease severity. Type B lactic acidosis is generally thought to be more benign, resulting from increased lactate production or decreased clearance. Common etiologies include increased lactate production secondary to drugs (such as HIV medications, metformin, and salicylates), and decreased clearance secondary to liver or renal failure, and inborn errors of metabolism.[1,2].

The pathogenesis of lactic acidosis during an asthma exacerbation is not clearly understood, but the previously presumed etiology was fatiguing respiratory muscles. However, previous case reports of patients with asthma exacerbations who were intubated and mechanically ventilated, receiving β2 agonists still found lactic acidemia in the absence of respiratory muscle fatigue[3,4]. Therefore, the etiology of lactic acidosis during asthma exacerbations with β2 agonist exposure is now hypothesized to be related to metabolic changes in glycogenolysis and gluconeogenesis leading to increased glycolysis and

pyruvate production. With concurrent inhibition of pyruvate dehydrogenase enzyme, pyruvate is unable to enter the Krebs cycle and instead is reduced to lactate leading to lactic acidosis [5,6].

Because only some asthmatics develop elevated lactic acid with β2 agonists, it is crucial for healthcare providers treating asthmatics to determine whether the patient has type B lactic acidosis from the treatment of acute bronchospasm or the more serious type A lactic acidosis from sepsis or hypoxia. This can be determined with continuous clinical monitoring and re-assessment as well as monitoring with peak flow measurements. Although our patient did have initial increased work of breathing, in the absence of sepsis, hypoxia and hemodynamic instability, she likely had albuterol-induced type B lactic acidosis which resolved after discontinuation of β2 agonist treatments.

Clinical Pearls:

- There are two types of lactic acidosis: Type A caused by hypoxia and tissue hypoperfusion and Type B caused by increased lactate production or decreased clearance from liver or renal failure.
- Increased use of β2 agonists can cause lactic acidosis type B.
- In patients with asthma exacerbation, healthcare providers need to be able to determine whether lactic acidosis is secondary to increased β2 agonists use versus sepsis/hypoxia which can be accomplished by continuous clinical monitoring and peak flow measurements.

REFERENCES

1. **Liem EB, Mnookin SC, Mahla ME**. Albuterol-induced lactic acidosis. *Anesthesiology*. 2003 Aug;99(2):505-6. PubMed PMID: 12883427.
2. **Rodrigo GJ, Rodrigo C**. Elevated plasma lactate level associated with high dose inhaled albuterol therapy in acute severe asthma. *Emerg Med J*. 2005 Jun;22(6):404-8. PubMed PMID: 15911945; PubMed Central PMCID: PMC1726802.
3. **Manthous CA**. Lactic acidosis in status asthmaticus : three cases and review of the literature. *Chest*. 2001 May;119(5):1599-602. Review. PubMed PMID: 11348975.
4. **Braden GL, Johnston SS, Germain MJ, Fitzgibbons JP, Dawson JA**. Lactic acidosis associated with the therapy of acute bronchospasm. *N Engl J Med*. 1985 Oct 3;313(14):890-1. PubMed PMID: 4033719.
5. **Haffner CA, Kendall MJ**. Metabolic effects of beta 2-agonists. *J Clin Pharm Ther*. 1992 Jun;17(3):155-64. Review. PubMed PMID: 1353501.
6. **Kovacevic A, Schwahn B, Schuster A**. Hyperlactic acidosis as metabolic side-effect of albuterol and theophylline in acute severe asthma. *Klin Padiatr*. 2010 Jul;222(4):271-2. doi: 10.1055/s-0030-1248259. Epub 2010 Apr 30. PubMed PMID: 20437331.

Submitted on December 5, 2013