

Lactic Acidosis in Status Asthmaticus : A Potential Side Effect of Albuterol Treatment

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Background

Nebulized albuterol is the standard bronchodilator therapy used to treat patients with status asthmaticus. However, excessive use of albuterol is associated with several side effects including tachycardia, hypokalemia, and tremors.

In rare cases albuterol can also cause lactic acidosis which leads to worsening clinical status. We report a pediatric case of status asthmaticus with worsening respiratory status due to metabolic acidosis from albuterol induced lactic acidosis.

Case

The patient is a previously healthy 14 year old female who presented to ED with one day history of coughing, wheezing, tachypnea and shortness of breath. Oxygen saturation on presentation was 92% on room air. Initial labs showed serum bicarbonate level of 22 meq/L and anion gap of 14.6. Patient was diagnosed with first episode of asthma exacerbation and received 3 consecutive albuterol nebulized treatments (total dose of 15mg) over a period of one hour with no significant clinical improvement.

She was admitted to intensive care unit. She was treated with continuous albuterol, O₂ by face mask and IV methylprednisone. Labs were repeated and showed metabolic acidosis with pH of 7.2, pCO₂ of 30 mm Hg, pO₂ of 50 mmHg, bicarbonate of 16 meq/L, and anion gap of 20. Serum lactate level was 9.7 mmol/L. Despite treatment, patient developed worsening tachypnea and increased work of breathing. Oxygen saturation remained above 95% throughout hospitalization. There were no signs or symptoms of hypovolemia, dehydration or poor tissue perfusion (normal blood pressure, normal capillary refill and adequate urine output).

Albuterol was suspected to be the cause of the lactic acidosis and the worsening of her respiratory status; so it was discontinued and ipratropium bromide was used as an alternative bronchodilator. The patient's respiratory rate then decreased and her clinical status was much improved with respiratory rate in the 20's and 99% saturations on room air. Serum lactate, pH, and bicarbonate normalized within 24 hours after discontinuing albuterol

Discussion

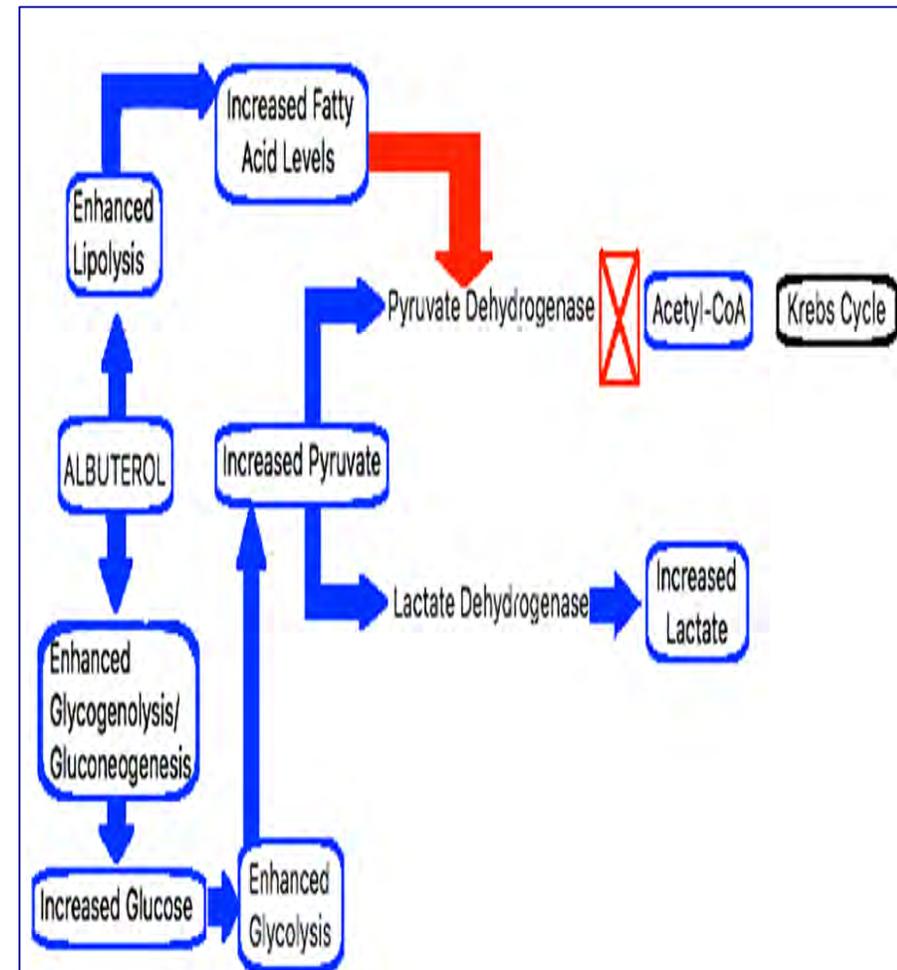
Lactic acidosis is a potential side effects of albuterol treatment in asthma exacerbation which can lead to worsening clinical status. The mechanism by which albuterol induces lactic acidosis is postulated as increased glycogenolysis which leads to overproduction of pyruvate (the precursor of lactic acid). Also, the increased pyruvate leads to increased lipolysis, which increases fatty acid levels. High fatty acid levels inhibit pyruvate dehydrogenase which diverts pyruvate from entering the Krebs cycle (Figure 1) and shunts it into anaerobic metabolism by lactate dehydrogenase. This results in an end product of lactate¹

In this case, the patient was diagnosed with severe asthma exacerbation upon presentation, but did not respond to the standard treatments. The subsequent clinical decompensation and the rising lactate levels could not be explained by the more common etiologies of lactic acidosis. For example, tissue oxygenation was adequate as indicated by oxygen saturations staying above 92%. There were no signs of decreased tissue perfusion such as hypotension, hypovolemia, or decreased capillary refill. She did not have any history of metabolic disorder and had not experienced any similar episodes before this presentation. Lack of prior exposure to albuterol and the high dose used during continuous nebulization are possible predisposing factors to development of lactic acidosis in this case.

Lactic acidosis quickly abated within 24 hours of stopping albuterol. Her lactate decreased from 9.7 mmol/L to within normal range in this time period. Subsequent testing of pulmonary function showed scooping of flow volume loops and an FEV₁/FVC ratio of 69% which supported asthma diagnosis. Ipratropium bromide was given as alternative bronchodilator to avoid any further reaction to albuterol. She was started on fluticasone 44mcg 2 puffs BID for controller therapy.

Lactic acidosis due to albuterol should be considered in any asthmatic with clinical worsening when exposed to high dose albuterol for the first time. Continuing to give albuterol exacerbates tachypnea and can lead to patient decompensation. Obtaining lactate levels in this case is necessary for early detection of the complication. Decreasing albuterol dose and/or use of alternative bronchodilators (i.e. ipratropium) with close follow up of serum lactate can help alleviate symptoms.

Figure 1



Conclusions

- Lactic acidosis is a rare but clinically significant side effect of albuterol use in asthma exacerbation
- Continued use of albuterol in this setting can result in worsening tachypnea and respiratory distress
- Early recognition of this adverse effect can help avoid and ameliorate the deterioration of symptoms.

References

1. Dodda, V.R., Spiro, P. Can Albuterol Be Blamed for Lactic Acidosis? *Respiratory Care* (2012) 57:12 2115-2118