

CASE REPORT

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Extreme elevation of lactate with nebulized albuterol in a complex patient with respiratory failure

Morgan K Kemerling, Zhaoyang Wen, Thomas J Nuckton

ABSTRACT

A 67-year-old man with a history of severe chronic obstructive pulmonary disease (COPD) and multiple medical problems presented to the emergency department (ED) with worsening shortness of breath for one to two days. He was initially treated with bilevel positive airway pressure (BIPAP) but required intubation approximately 4 hours after presentation. Multiple doses of nebulized albuterol (total of 21 mg) were given both before and after intubation. The patient's initial lactate level was 2.3 mmol/L, with rapid elevation to a maximum of 11.3 mmol/L. In combination with the markedly elevated lactate, the patient had abdominal distention and nonspecific abdominal pain in the setting of chronic CO₂ retention and acute hypercapnic respiratory failure, transient hypotension, an elevated peripheral white blood cell (WBC) count with neutrophil predominance, and an increased anion gap. The clinical presentation, elevated lactate level, and other laboratory abnormalities prompted a concern for severe sepsis and possible bowel ischemia. The patient was treated with intravenous fluids and broad-spectrum antibiotics, but at the same time, albuterol was held. Lactate decreased rapidly after albuterol was discontinued. A computed tomography (CT) scan of the abdomen did not show signs of ischemia, and abdominal distention improved with an orogastric tube. With mechanical ventilation and steroids, the patient's respiratory status improved, and he was extubated on hospital day six. Final blood cultures were negative, and in retrospect, the patient did not have severe sepsis. The

timeline of lactate increase and decrease suggests that the markedly elevated lactate levels in this patient were due to a Type B lactic acidosis related to albuterol.

Keywords: Albuterol, Lactate, Lactic acidosis, Metabolism, Sepsis

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INTRODUCTION

Lactic acidosis has been well described, with classification into Type A lactic acidosis and Type B lactic acidosis. Type A lactic acidosis is due to tissue hypoxia and/or hypoperfusion, whereas Type B lactic acidosis occurs with adequate oxygenation and can be due to a variety of causes including liver and kidney disease, diabetes, malignancy, and medications including metformin, human immunodeficiency virus (HIV) reverse transcriptase inhibitors, linezolid, beta agonists, and others [1]. Several cases and studies have reported elevations in lactate associated with nebulized albuterol, as a Type B lactic acidosis [2–6]. When described in a clinical setting, elevations typically have been in the range of 2–8 mmol/L [3–6]. We report a case of a patient with chronic obstructive pulmonary disease (COPD) and respiratory failure who received nebulized albuterol and subsequently had an extreme elevation in lactate level to 11.3 mmol/L. The lactate elevation added confusion to the patient's diagnosis and management.

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CASE REPORT

A 67-year-old man with a history of severe COPD (50 pack-year smoking history, home O₂ requirement of 3 L/min, GOLD D/mMRC>2), persistent sinus tachycardia, osteoporosis, gastroesophageal reflux disease (GERD), benign prostatic hyperplasia (BPH), hyperlipidemia, and chronic constipation, presented to the emergency department (ED) with worsening shortness of breath for one to two days. On arrival in the ED, he was noted to have a respiratory rate greater than 30 breaths per minute and difficulty speaking in complete sentences. His initial blood pressure was 144/73 mmHg (MAP 97 mmHg) with a heart rate of 124 bpm (sinus tachycardia). He also reported pleuritic chest pain and nonspecific abdominal pain. Bilevel positive airway pressure (BIPAP) was initiated in the ED shortly after arrival. Physical exam was remarkable for decreased breath sounds in all lung fields and moderate abdominal distention, without abdominal tenderness or guarding. Initial arterial blood gas (ABG) showed a pH of 7.36, PCO₂ of 99 mmHg, PO₂ of 159 mmHg, and a calculated HCO₃ of 55.9 mmol/L (with a serum total CO₂ of 43 mmol/L), while the patient was receiving BIPAP with a fraction of inspired oxygen (FIO₂) of 1.0. The patient's initial respiratory acidosis worsened while on BIPAP (PCO₂ increased to 118 mmHg and pH decreased to 7.27) and he was intubated 4 hours after presentation. Following intubation, his BP decreased to 89/60 mmHg (MAP 69 mmHg) and heart rate decreased to 106 bpm (sinus tachycardia). Approximately 2 hours after intubation, peripheral WBC count was 13.2 K/uL with 98% neutrophils (from a WBC count of 9.9 K/uL with 93% neutrophils initially), total CO₂ was 32 mmol/L (from 43 mmol/L initially) with an anion gap of 16 mmol/L (from 8 mmol/L initially); other laboratory values were unremarkable.

In the ED the patient received one nebulized albuterol (15 mg, continuous over 1 hour) treatment while on BIPAP and two nebulized combined albuterol/ipratropium (3 mg/0.5 mg) treatments while intubated, over the course of 8 hours. He also received 125 mg of intravenous (IV) methylprednisolone and was started on 80 mg of methylprednisolone every 8 hours. Initial lactate on presentation in the ED was 2.3 mmol/L. Lactate increased to 11.3 mmol/L approximately 7.5 hours after arrival to the ED and 7 hours after the first albuterol treatment. The timeline of lactate increase in relation to albuterol administration is summarized in Figure 1 (note that lactate decreased when albuterol was discontinued). Chest X-ray (CXR) showed hyperinflation, but no focal consolidations (Figure 2). Computed tomography (CT) scans of the chest with contrast were notable for centrilobular emphysema and lung hyperinflation, but no pulmonary embolism or airspace consolidation (Figures 3 and 4). A CT scan of the abdomen/pelvis was unremarkable for acute abdominal pathology but did show large amounts of stool in the colon, consistent with constipation, and air in

the stomach (Figure 5). The patient was admitted to the intensive care unit (ICU) where he received mechanical ventilation, IV fluids, and broad-spectrum antibiotics. His blood pressure improved with IV fluids, and he did not require vasopressors. Abdominal distention improved with intermittent suction via an orogastric tube. Blood cultures drawn at the time of admission were ultimately negative and antibiotics were discontinued on hospital day 5 (completing empiric coverage for community-acquired pneumonia). He was extubated on hospital day 6 and discharged to home on hospital day 11. Six months following discharge, he remains stable and is living independently with home nursing support.

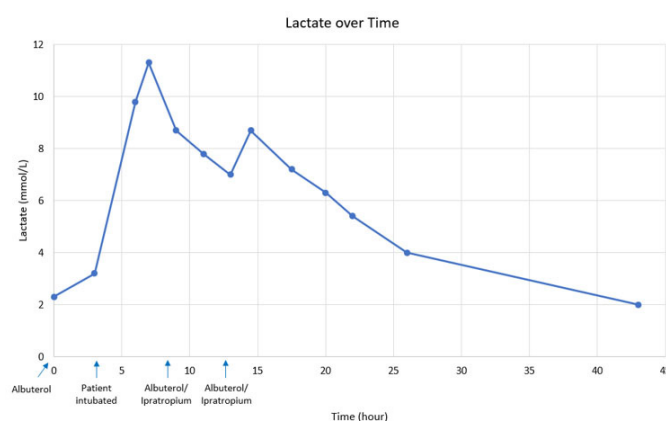


Figure 1: Illustration of blood lactate level (mmol/L) measured over time (hour). 15 mg of nebulized albuterol was administered at hour 0. The patient was intubated at hour 3. Nebulized combined albuterol/ipratropium (3 mg albuterol and 0.5 mg ipratropium) was administered twice, at hour 8 and at hour 12.5. No additional albuterol was given after hour 12.5.



Figure 2: CXR illustrating hyperinflation of the lungs bilaterally, with no focal consolidations.

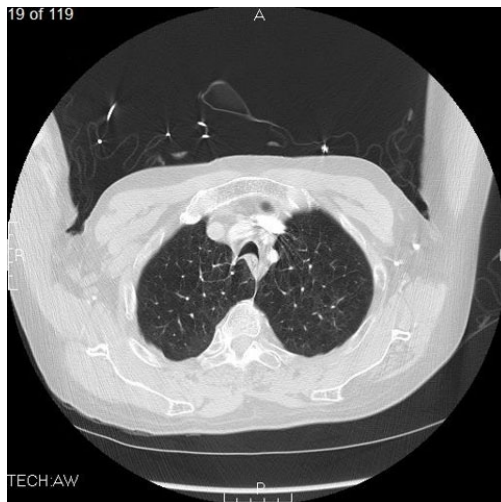


Figure 3: Axial cut of CT chest showing emphysematous changes.

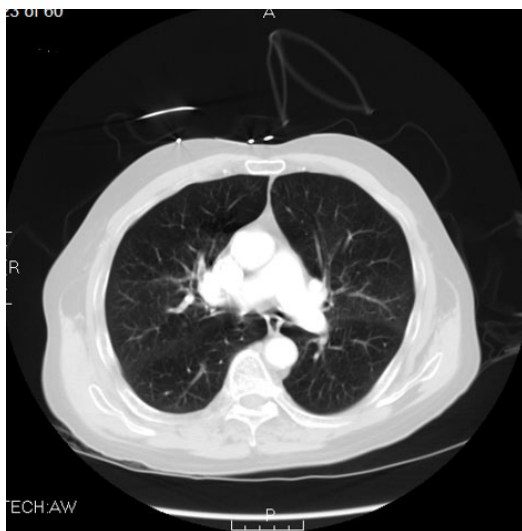


Figure 4: Axial cut of computed tomography angiogram (CTA) chest showing no pulmonary emboli in major pulmonary blood vessels.



Figure 5: Coronal cut of CT abdomen and pelvis illustrating a significant stool burden in the colon and a stomach distended with a large volume of air.

DISCUSSION

Lactic acidosis is often used as a marker for severity of metabolic derangement and is frequently seen in critically ill patients presenting to the ED. Lactate is a substrate normally produced by the human body, and an excess of lactate in the blood can be due to increased production or decreased breakdown. Under normal aerobic metabolism, glucose is broken down to create pyruvate which is then converted to acetyl-coenzyme A (CoA). Acetyl-CoA then undergoes oxidative phosphorylation in the mitochondria to produce adenosine triphosphate (ATP), a molecule that stores and releases energy. If the cell cannot perform oxidative phosphorylation due to insufficient oxygen, pyruvate is instead diverted to create lactate [1]. Previously, lactate was thought of purely as a byproduct of metabolism, created when tissue hypoxia occurs; however, more recent literature has demonstrated that lactate is continuously produced within the cell in normal aerobic respiration and may have additional functions [7]. Our case highlights that lactate interpretation in the clinical setting can be difficult due to the varied causes of lactate elevation.

When a patient arrives in the ED and requires immediate treatment, often prior to obtaining studies such as labs and imaging, the treatment provided may confound the clinical picture. In our case, the administration of albuterol resulted in an extreme elevation in lactate to 11.3 mmol/L—one of the highest levels reported in a patient with COPD receiving albuterol. The lactate level in our patient exceeded the albuterol-associated lactate levels in experimental studies in healthy volunteers [2], in studies in patients with asthma [3], and in case reports of patients with asthma and COPD [4–6]. A prior study in healthy adult volunteers showed that subjects who received albuterol (10 mg nebulized) had a mean increase in lactate of 0.77 mmol/L, with all 14 subjects who received albuterol experiencing an increase in lactate [2]. Another study in adult ED patients with severe asthma showed a mean increase in lactate level to 2.94 mmol/L from 1.1 mmol/L with high dose nebulized albuterol [3]. Others have reported increased lactate levels in the range of 3–8 mmol/L in single patients treated with albuterol for asthma or COPD [4–6].

In other selected case reports, even higher lactate levels have been described with albuterol use. Gami and colleagues reported a lactate increase to 9.1 mmol/L in a patient with severe asthma receiving albuterol via continuous nebulization [8], and Hockstein and Diercks reported an increase in lactate to 10.3 mmol/L, also in a patient with asthma who received a total of 25 mg of nebulized albuterol [9]. A prior report of a younger patient (41 years) with severe asthma had yet a higher lactate level—as high as 15 mmol/L; the patient in that report received nebulized salbutamol, which is the beta-agonist commonly used in Europe, and which is essentially identical to albuterol [10]. Thus, very high lactate levels with beta-agonists, while uncommon, are not unique to our patient.

A key feature in our case, however, is that a markedly elevated lactate level clouded the clinical diagnosis in a patient with multiple medical problems and a complex presentation. Our patient had abdominal distention and nonspecific abdominal pain in the setting of chronic CO₂ retention and acute hypercapnic respiratory failure, with a markedly elevated lactate, transient hypotension, an elevated peripheral WBC with neutrophil predominance, and an increased anion gap. These findings prompted a concern for severe sepsis, and particularly intra-abdominal pathology such as bowel ischemia. A CT scan of the abdomen and pelvis did not reveal acute pathology. Although a negative CT of the abdomen does not rule out bowel ischemia, the rapid decrease in lactate when albuterol was held (Figure 1) and the patient's overall clinical improvement were reassuring.

Many clinical features of this case became clear in retrospect. Abdominal distention was likely secondary to aerophagia related to BIPAP [11], which is supported by a large volume of air in a distended stomach that was seen on abdominal CT. The patient had a history of chronic constipation (with a large stool burden on abdominal CT), a plausible cause of nonspecific abdominal pain. A transient decrease in blood pressure is not uncommon with sedation after intubation and initiation of mechanical ventilation [12]. An increase in peripheral WBC with neutrophil predominance was likely due to administration of steroids or a generalized stress response [13–15]. Increased work of breathing and respiratory muscle production may have contributed to the increase in lactate in our patient, especially prior to intubation [16–20]. However, increases in lactate from a high respiratory muscle workload have been moderate in prior studies [17–19], and dramatic increases in lactate from respiratory muscles are less likely in patients with COPD and asthma in respiratory failure [20–22]. Others have proposed that increased lactate may indicate excessive beta-agonist treatment and should be examined as a biomarker for overtreatment in future studies [23]. In our patient, the rapid decline in lactate when albuterol was discontinued, combined with the other features of this case, strongly suggests that the marked increase in lactate was a Type B lactic acidosis caused by nebulized beta-agonist therapy.

CONCLUSION

In summary, our case highlights many important points. First, an elevation in lactate secondary to albuterol can make a clear diagnosis challenging. Clinicians should be aware of the association between beta-2 agonists and an increase in lactate and carefully consider the possible causes for an elevated lactate in individual patients. It is important to remember that lactate elevations can be due to more than hypoxia or hypoperfusion. Lastly, our case reinforces that elevation in lactate levels with albuterol

administration can be extreme. Although milder increases in lactate are more common with albuterol, our case illustrates that unexpected results can and do occur.

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Author Contributions

Morgan K Kemerling – Design of the work, Analysis of data, Interpretation of data, Drafting the work, Revising the work critically for important intellectual content, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

Zhaoyang Wen – Conception of the work, Acquisition of data, Analysis of data, Interpretation of data, Revising the work critically for important intellectual content, Final approval of the version to be published, Agree to be

accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

Thomas J Nuckton – Conception of the work, Design of the work, Acquisition of data, Analysis of data, Interpretation of data, Drafting the work, Revising the work critically for important intellectual content, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

Guarantor of Submission

The corresponding author is the guarantor of submission.

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Consent Statement

Written informed consent was obtained from the patient for publication of this article.

Conflict of Interest

Authors declare no conflict of interest.

Data Availability

All relevant data are within the paper and its Supporting Information files.

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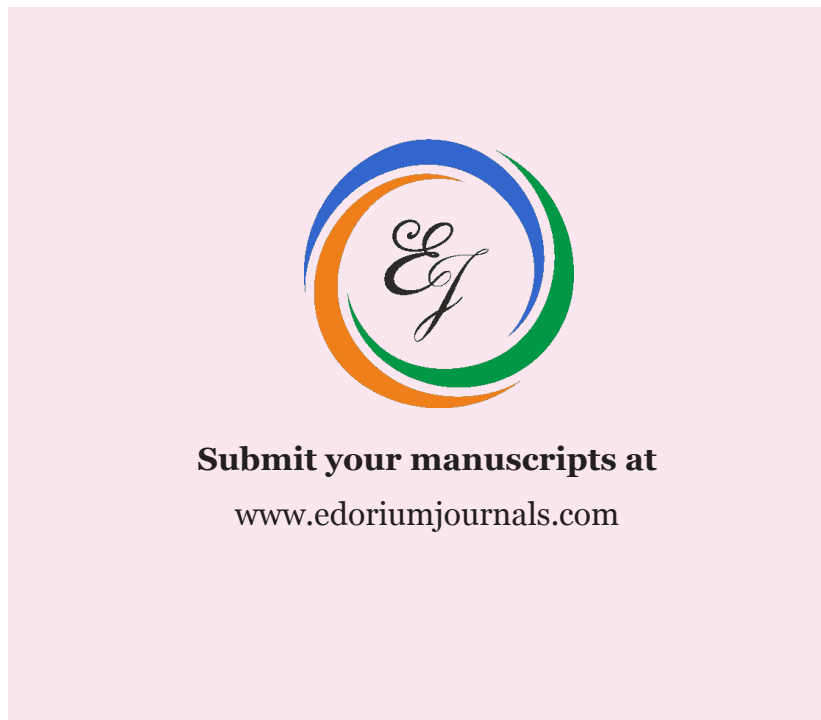
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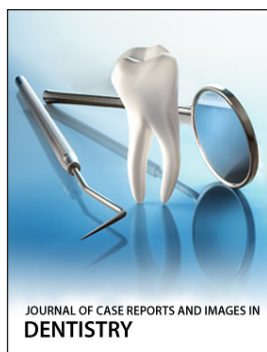
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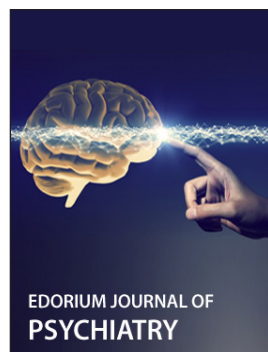
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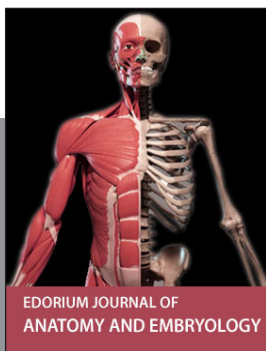
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