Lactic acidosis secondary to albuterol inhaler use

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Background:
A lactate level elevation of more than 4 millimoles/liter is considered to be lactic acidosis (LA), more than 2 millimoles/liter is considered hyperlactatemia [1]. The adjusted odds ratio (OR) of lactate > 4 for mortality was 7.6 in few days [2].

Case:
We present a case of a 68-year-old male with a past medical history of hypertension, COPD (no home O2), PE who presented to the ED with concern for shortness of breath and wheezing for the past two days since running out of inhalers. Tachycardiac, tachypenic, hypertensive in ED. Received IV Solumedrol and 3 rounds of DuoNebs. Labs significant for WBC 13.7, lactate of 11.8 that improved to 10.1 after 30 cc/kg of normal saline, then 9.4 after an additional 500cc, pH 7.25 CO2 32 bicarb 14, anion gap 24, creatinine 1.6 (baseline 0.9), potassium 2.9, troponin 54–108. EKG with sinus tachycardia 103 bpm and new ST depressions in I, aVF, v4-6. CXR and CT chest/abdomen/pelvis were negative for infection. Denied chest pain. His lactate decreased, and his creatinine improved with 3 L IVF.

Discussion:
Elevated lactate was likely type B lactic acidosis due to overuse of albuterol inhaler and nebulizer treatments. His hypokalemia was also likely a result of the short-acting beta agonist.

LA is categorized into two forms based on pathophysiology: type A LA from hypoperfusion/hypoxia and type B LA from abnormal metabolism within cells. With the implementation of improved sepsis protocols/guidelines in emergency rooms and hospitals, the frequency of LA has lately increased [3]. Because of differences in management, it is critical to identify the specific type [4]. Metformin, alcoholism, cancer, antibiotics such as linezolid, antidepressants, and beta-agonists are among the most common causes of type B LA [5].

Conclusion:
Elevated LA is diagnosed as sepsis in emergency departments mostly. Other differentials of LA should be evaluated.

References: