

Lactic Acidosis and Asthma Exacerbation[☆]



Acidosis láctica y agudización del asma

To the Editor:

A relatively frequent observation during the treatment of asthma exacerbations is a transient rise in serum lactate (hyperlactatemia), unrelated with PaO₂ or PaCO₂, that may cause lactic acidosis.^{1–3} Little is known about the clinical impact of this metabolic phenomenon, despite the fact that lactic acidosis can produce symptoms that interfere in the assessment of asthma exacerbations.

Lactate or lactic acid is produced from pyruvate as an end product of glycolysis in anaerobic conditions. An increase in lactate can cause lactic acidosis, which exists in 2 forms: type A due to hypoxia and hypoperfusion, and type B, which is caused by other mechanisms, and is associated with multiple situations such as liver disease or malignancy, or the use of drugs such as metformin and adrenaline.⁴

We report the case of a 41-year-old woman, diagnosed with bronchial asthma and chronic urticaria, who presented lactic acidosis during treatment of an asthma exacerbation. The patient had made frequent visits to the emergency department, and had been hospitalized several times in the previous year. She received regular treatment with beta-adrenergic agonists, long-acting anticholinergics, and inhaled corticosteroids, and courses of oral corticosteroids, montelukast, antihistamines, and omalizumab. She presented to the emergency department with intense dyspnea and wheezing, and was treated with systemic corticosteroids and nebulized salbutamol (5 mg) and ipratropium bromide (0.5 mg), repeated on several occasions due to persistence of the dyspnea and severe tachypnea. Arterial blood gases after 2 nebulizations revealed pH 7.31, PaCO₂ 41 mmHg, PaO₂ 128 mmHg, bicarbonate 20.4 mmol/l, base excess –4.9 mmol/l, and lactate 5.7 mmol/l (normal: <2.2 mmol/l). Arterial blood gases performed in the following hours, with the same treatment but with the addition of benzodiazepines, showed a rapid normalization of pH and lactate, which fell to 2.2 and 0.6 mmol/l, respectively. The patient did not present hypoxemia, hypotension, or other alterations associated with hyperlactatemia.⁴

It has been postulated that hyperlactatemia in asthma may be associated with the endogenous production of catecholamines generated during exacerbations, or with respiratory muscle fatigue that occurs during these events, due to the increased demand for oxygen.³ However, evidence has shown that these are not the main mechanisms involved, since lactic acidosis also occurs in patients with muscle relaxation induced by beta-adrenergic drugs.⁵ The most recent data suggest that the increase in lactate is associated with the use of these beta-adrenergic agents,^{2,3} although a previous hyperadrenergic state may predispose to elevated lactate levels.¹ Data to support the direct role of beta-2 agonists in hyperlactatemia include evidence that nebulized salbutamol in healthy individuals also causes serum lactate levels to rise,⁶ and that intentional overdose with salmeterol has been associated with lactic acidosis.⁷ In a study of patients treated for severe asthma exacerbations, lactate

levels in blood were associated with plasma levels of salbutamol.⁸ However, hyperlactatemia does not appear to have prognostic significance, since it has not been associated with an increased risk of hospitalization or relapse.⁸

From a clinical perspective, it is interesting to note the paradoxical situation of patients who develop lactic acidosis, who, in spite of improving bronchospasm, present greater tachypnea and respiratory difficulty, as a compensatory mechanism for the acidosis. This situation may lead to the administration of higher doses of salbutamol, resulting in an effect contrary to expected.^{3,8} The intense tachypnea observed in our patient may be related with the attempt to achieve respiratory compensation for lactic acidosis which resolved rapidly, despite the continued administration of nebulized salbutamol. In these cases, it is particularly useful to confirm response to bronchodilators and to avoid confusing compensatory tachypnea with poor evolution of the asthma, with the use of peak expiratory flow measurements,^{3,8} a parameter that is often overlooked in emergency departments.^{9,10}

References

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