Hyperlactatemia during Acute Asthma Attack

Patcharee Tedcha1
Theerasak Kawamatawong2
Pongdhep Theerawi2
Yuda Sutherasan3
Satariya Trakulsrichai3

1Department of Medicine, Faculty of Medicine Ramathibodi Hospital, Mahidol University, Bangkok 10400, Thailand, 2Division of Pulmonary and Critical Care Medicine, Department of Medicine, Faculty of Medicine Ramathibodi Hospital, Mahidol University, Bangkok 10400, Thailand, 3Department of Emergency Medicine, Faculty of Medicine, Ramathibodi Hospital, Mahidol University, Bangkok 10400, Thailand

Background: Beta2-agonists have been used as bronchospasm relief for the acute asthmatic attack. In the meantime, lactic acidosis or hyperlactatemia is reported in acute asthmatic attack patients. Possible causes of hyperlactatemia in asthma include hypoxemia, increase of breathing, and beta-2 agonist therapy.

Objective: This study aimed to evaluate arterial lactate levels during treatment of acute asthma and effects of bronchodilator response.

Methods: We performed a prospective study at Emergency Department (ED) of Ramathibodi hospital in adults with acute asthmatic attack who were treated with either salbutamol or ipratropium bromide and fenoterol nebulizer solutions at 30-minute intervals for 4 times. All patients received standardized treatment of acute asthma during the first 2 hours, including nebulized bronchodilator and dexamethasone. Arterial lactate concentrations and physiologic parameters were measured at ED arrival and 2 hours after treatment.

Results: A total of 29 subjects were enrolled. The mean age was 55.55±20.30 years. Baseline systolic pressure (SP), diastolic pressure (DP), and SpO2 (%) were 145.55±26.14 mmHg, 80.41±13.93 mmHg, and 95.24±3.09, respectively. The initial lactate was 2.27±1.67 mmol/L. At 2 hours after treatment with 4 doses of bronchodilator, there was a significant decrease of SP from 145.55±26.14 to 133.48±15.24 mmHg (P=0.003), DP from 80.41±13.93 to 74.86±9.75 mmHg (P=0.03), and RR from 24.41±5.36 to 21.55±1.76 breaths (P=0.004). Whilst, an increase of SpO2 (%) was noted (95.24±3.09 vs 97.38±2.12; P<0.001). Besides, lactate slightly increased from baseline, but no statistical significance (2.27±1.67 vs 2.56±1.67 mmol/L; P=0.115). However, an increasing of lactate was observed in sixteen subjects (55.2%). Meanwhile, the decreasing of lactate was shown in ten subjects (34.5%). Subgroup analysis of lactate increase yielded a significant rising of lactate from baseline after bronchodilator therapy (1.83±1.63 vs 2.72±1.76; P<0.001). The Pearson correlation showed only a significant association of lactate with SpO2 (r=0.552; P=0.027), but not with SP and DP. In addition, the changing of lactate was not related to hospital admission or discharge.

Conclusion: During acute asthmatic attack, arterial hyperlactatemia is frequently present on ED admission. Nevertheless, plasma lactate level shows no significant difference between ED admission and 2 hours after treatment.

Keywords: Hyperlactatemia, Acute asthma attack