

Muscle spasms and creatine phosphokinase elevation following salbutamol administration

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The drug of choice for the treatment of occasional acute symptoms of asthma is an inhaled beta adrenergic agonist, such as salbutamol. The most frequent adverse reactions to salbutamol include nervousness, headache, tremor, tiredness and light-headedness [1]. The following report describes a patient with asthma from childhood, who developed creatine phosphokinase elevation associated with severe muscle spasms following oral and inhaled salbutamol administration.

Case Report

The patient, a 25 yr old woman, had a history of asthma since the age of 2-3 yrs and had been in her usual state of health with occasional flair-ups of her asthma that were easily managed with inhaled salbutamol. Her medical history included asthma, Raynaud's disease and asymptomatic premature ventricular contractions that were not treated. Smoking history was negative. On April 17, 1987, she had an exacerbation of her asthma, which did not abate with admitted overcompliance of her salbutamol metered-dose inhaler.

On April 18, she visited family members and was exposed to dog dander, one of her known allergens. Prior to the visit, anticipating the allergen exposure, she pre-treated with 2 mg of salbutamol tablets and an anti-histamine. Her symptoms worsened following the visit, so she continued with oral salbutamol treatment taking 4 mg every 6 h. By early morning on April 19, her breathing difficulty became severe enough to necessitate a visit to the emergency department. Here, she received two metoprolterenol treatments *via* a nebulizer over a 6-8 h period. Post discharge she continued on 4 mg of oral salbutamol every 6 h for the next 2-3 days.

On April 21, she developed severe muscle spasms in the right thoracic area of the chest. She described a wave-like progression of these spasms without any resolution. The pain associated with the spasms was so intense that it incapacitated her. She had a history of developing

muscle spasms, usually during the winter months. However, they had not equalled the intensity or duration of this current event. The pain had not subsided by the next morning, and on April 22 blood chemistries were drawn. Laboratory data, including the serum potassium were within normal limits, except for the creatine phosphokinase (CPK) which was 209 IU·l⁻¹ (normal range=25-145 IU·l⁻¹). Shortly afterwards the dose of salbutamol (oral and inhalation) was tapered, since respiratory symptoms had subsided. As the dose was decreased, the muscle spasms ceased. A T4 to rule out thyroid disease as a cause of the elevated CPK, and a repeat CPK were ordered: these were within normal limits. The patient had no further muscle spasms. She had not experienced any conditions such as an acute myocardial infarction (AMI) or an intramuscular injection that could account for the CPK elevation.

Investigation of the literature revealed one study in which CPK elevations were noted following salbutamol administration. DYSON and MACKAY [2] in a double-blind, cross-over study found an increase in CPK in 19 out of 93 patients with chronic obstructive pulmonary disease (COPD) treated with either salbutamol orally 4 mg four times daily or pirbuterol orally 10 mg daily. Six patients showed an abnormal CPK value on pirbuterol 10 mg a day orally, six on salbutamol 4 mg four times a day orally, and seven showed abnormal values during both treatments. In addition, one of the three patients on salbuterol was withdrawn from the study because of muscle spasms.

References

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