Rhabdomyolysis in Acute Severe Asthma: A Case Report and Literature Review

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Abstract

An elevated serum CPK and the presence of myoglobin in the urine characterize rhabdomyolysis. Rhabdomyolysis has been described in various traumatic and non-traumatic conditions; there are few reports of its association with acute severe asthma. In this paper, we report a case of acute severe asthma complicated by respiratory failure and rhabdomyolysis. We discuss the relationship between rhabdomyolysis and acute severe asthma and review the relevant literature.

CASE REPORT

A 26 year-old man with asthma was seen in the emergency department (ED) with shortness of breath, chest tightness, wheeze of 2 day's duration. There had been a productive cough, sore throat and rhinorrhea for one week. The patient's only medication was albuterol that he had been using with increasing frequency by metered dose inhaler (MDI) without a spacer for 2 days prior to ED arrival. On the day of presentation he was using 2 puffs every 15 minutes without relief. In the ED severe dyspnea and a deteriorating mental status lead to emergent intubation. The pre-intubation arterial blood gas was pH of 6.99, pCO2 90 mmHg, pO2 450 mmHg on 100 % oxygen by facemask. Peri-intubation medications were epinephrine 0.3 mg subcutaneously, Ketamine 100 mg intravenously (IV), succinylcholine 150 mg IV, pancuronium 7 mg IV, methylprednisolone 125 mg IV and magnesium sulfate 2 g IV. Albuterol and ipratropium bromide were delivered continuously by nebulization during initial mechanical ventilation.

The patient had had asthma since the age of 14. He had had 1 to 2 exacerbations each year, but he had never been hospitalized or treated in an ED. There was no history of allergy. There was no sensitivity to aspirin. There was no history of tobacco, alcohol or illicit drug use.

In the medical intensive care unit, the patient had temperature of 37.1° centigrade (98.8° F). His heart rate was 126 beats/minute and blood pressure was 175/95 mmHg. There were diffuse inspiratory and expiratory wheezes with expiratory phase prolongation. The cardiac examination was normal except for tachycardia. There was no pallor, cyanosis, or clubbing. The skin and extremities were normal.

The white blood cell count, electrolytes, minerals and liver and renal function tests were normal. Blood lactate was 7.9 mmol/L (normal: 0.2 to 2.5). Creatine phosphokinase (CPK) was 6,868 U/L (normal: 0-125U/L). The first CPK level was performed in the ED as soon as the patient arrived, prior to administration of systemic steroids, paralytics and epinephrine. The initial CPK was performed to rule out myocardial infarction. The CPK MB was not elevated. Urine analysis revealed 50 red blood cells on microscopic urine examination and myoglobin. Urine toxicology screen was negative. The chest radiograph was normal.

The patient received lorazepam and morphine, but no additional paralytics. Mechanical ventilation was continued with a strategy of permissive hypercapnia. An arterial blood gas shortly after intubation was pH 7.20, pCO2 54 mmHg, pO2 490 mmHg on 100 % FiO_2.

Significant improvement in lung mechanics and blood gases allowed for successful extubation after 14 hours of mechanical ventilation. In the peri-extubation period, albuterol and ipratropium were continued by nebulization every four hours; methylprednisolone 125 mg IV was given twice in the first 8 hours; prednisone 60 mg daily was continued until discharge on hospital day 6.

CPK 18 hours after admission increased to 16,263 U/L with a normal serum creatinine, potassium, and phosphate. Hydration and sodium bicarbonate were initiated for

management of rhabdomyolysis. The peak CPK was 21,953 U/L at 36 hours, it dropped steadily thereafter to 414 U/L by the 6th hospital day. (see figure)

DISCUSSION

An elevated serum CPK and the presence of myoglobin in the urine ([1]) characterize rhabdomyolysis. It may result from crush injury, tissue ischemia, prolonged immobilization, major trauma, illicit drugs and use of paralytic agents like succinylcholine. Myoglobinemia after severe exercise has also been reported ([2]). In acute severe asthma, vigorous and repeated contractions of the diaphragm and accessory muscles of respiration are analogous to strenuous exercise (2). Repeated coughing with vigorous muscle contraction may cause additional muscular injury leading to myoglobinuria ([3]). Hypoxemia due to acute respiratory failure may lead to muscle ischemia and muscle injury in some cases ([4]).

Other factors like prolonged mechanical ventilation ([5]), extended use of high dose corticosteroids ([6]) and use of muscle relaxants may contribute to rhabdomyolysis in asthma ([7]). Prolong use of neuromuscular blocking agents have been implicated as a cause of rhabdomyolysis ([5,8]). There are reports of single dose succinylcholine causing rhabdomyolysis, but this is rare ([9]). agonist are known to cause mild elevation in CPK in at therapeutic dosage ([10]). Excessive use of beta sympathomimetic agents may cause agitation and tremors leading to rhabdomyolysis ([11]). Acute oral theophylline intoxication has also been reported to cause rhabdomyolysis (11). Finally cocaine use has been associated with both acute asthma and rhabdomyolysis.

Acute myopathy complicating severe asthma has been described ([3,12,13]). Lovis et al reported elevation of CPK and plasma myoglobin with normal cardio-specific troponin T in five out of fifteen patients with acute severe asthma, suggesting that elevation of CPK was non-cardiac origin ([14]).

In our case, we suspect that vigorous muscle contraction and high dose beta agonist therapy resulted in rhabdomyolysis, although the precise mechanism of CPK elevation is not known. The absence of fever and muscle rigidity was not supportive of a diagnosis of malignant hyperthermia, and the absence of muscle wasting or any evidence of chronic neuromuscular weakness was not suggestive of McArdle disease ([15,16,17]). We cannot exclude some contribution from use of succinylcholine and corticosteroids but in this case the CPK was elevated before these drugs were administered.

In our case there were no obvious consequences of CPK elevation such as overt neuromuscular weakness prolonging the need for assisted ventilation or acute renal failure. The latter complication has been reported (3) and may have been averted in our case by early diagnosis of CPK elevation and vigorous hydration. The level of CPK elevation in this case, was similar to the cases reported in the literature. This report adds to the literature confirming an association between acute severe asthma and the development of rhabdomyolisis.

We recommend CPK determinations in all severely ill asthmatics and treatment of rhabdomyolisis using accepted measures such as vigorous hydration to avoid complications of acute renal failure.

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