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CASE REPORT

Rhabdomyolysis Related to Dyskinesia in Parkinson's Disease

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ABSTRACT

Rhabdomyolysis is a life threatening syndrome. It accounts for an estimated 8% to 15% of cases of acute renal failure and is associated with a mortality rate of 5%. In movement disorders, various causes of rhabdomyolysis have been reported including status dystonicus, myoclonus, generalized chorea and parkinsonism-hyperprexia syndrome in Parkinson's disease (PD). Levodopa-induced dyskinesia leading to rhabdomyolysis is a very rare phenomenon in PD. We report a case of 76 years old PD patient with dyskinesia and rhabdomyolysis.

Key Words Rhabdomyolysis; Parkinson's disease; Levodopa-induced dyskinesia.

Rhabdomyolysis is characteried by the breakdown of skeletal muscle resulting in the subsequent release of intracellular contents into the circulatory system. These cell contents include enzymes, such as creatine kinase (CK), glutamic oxalacetic transaminase, lactate dehydrogenase, and aldolase; the heme pigment myoglobin; electrolytes, such as potassium and phosphate; and purines.[1-3] Rhabdomyolysis ranges in severity from an asymptomatic elevation of CK levelin blood to a severe, life-threatening disorder associated with very high CK levelmyoglobinuria and acute renal failure.[4] Rhabdomyolysis accounts for an estimated 8% to 15% of cases of acute renal failure and is associated with a mortality rate of 5%.[5,6] We report a case of a Parkinson's disease (PD) patient with dyskinesia and rhabdomyolysis.

CASE

A 76-year-old woman with a 15-year history of PD who was being treated with levodopa presented to the emergency department with severe generalized dyskinesia of 3 days' duration. She had begun to show bradykinesia and rigidity at age 61 and levodopa treatment had improved her parkinsonian symptoms. For the past 2 years, she had been suffering from difficulties with walking and moving. She had felt that her symptoms were worsening with levodopa 500 mg/day; thus, her medication had been changed to levodopa/carbidopa/entacapone (400 mg/day, 150 mg/day, 800 mg/day, respectively) combination therapy. With the progression of the patient's symptoms, the dosages of levodopa/carbidopa/entacapone had been increased to 600 mg/day, 150 mg/day, and 800 mg/day, respectively. Her dosages had gradually been increased until she was taking levodopa/carbidopa/entacapone at up to 900 mg/day, 225 mg/day, and 1200 mg/day, respectively. However, she had begun to experience wearing off and was immobile during her off periods. Her levodopa/carbidopa/entacapone dosages had then been raised to 1500 mg/day, 375 mg/day, and 2000 mg/day, respectively. Three days before admission to the emergency department, she began to suffer from dyskinesia. The dyskinesia affected all of her limbs and her trunk, and she was not able to sit or walk without support. Her body temperature, blood pressure, heart rate and respiration rate were normal. Upon admission, blood tests showed a urea level of 178 mg/dL (10−48.5), a creatinine level of 3.7 mg/dL (0.5−1.2), a CPK level of 2253 U/L, a myoglobin level of 3000 ng/mL (0−58), and a CPK-MB level of 12.39 ng/mL (0−2.9). The patient was anuric; thus, acute renal failure due to rhabdomyolysis was diagnosed,

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and the patient was enrolled in hemodialysis. Levodopa dosage was lowered, and dyskinesia severity decreased. She was awake but showed immobility and severe rigidity in her arms, legs and neck. On follow-up, her blood urea, creatinine, CPK, myoglobin and CPK-MB levels had decreased to 100 mg/dL, 1.2 mg/dL, 119 U/L, 1342 ng/mL, and 12.39 ng/mL, respectively. We performed diagnostic work-ups for the common known causes of rhabdomyolysis and found no etiology. Levodopa-induced dyskinesia (LID) leading to rhabdomyolysis was diagnosed. On the seventday after hospital admission, the patient's body temperature increased to 38.2°C, and a chest X-ray revealed pneumonia. Antibiotic therapy was started. However, her oxygen saturation fell, and arterial blood gas analysis showed severe hypoxemia; she then required intubation and mechanical ventilation. Although she recovered renal function, the patient died due to sepsis.

DISCUSSION

Muscles of the body are prone to various forms of damage. Damage leads to the release of intracellular components into the systemic circulation that causes rhabdomyolysis.[7] The most common causes of rhabdomyolysis in adults are drugs and toxins, trauma, excessive muscular activity, temperature extremes, muscle ischemia, prolonged immobilization, infection, electrolyte and endocrine abnormalities, genetic disorders, connective tissue disorders and unknown conditions.[4] Other significant causes of rhabdomyolysis include excessive muscular activity, such as sporadic strenuous exercise (e.g., marathons), status epilepticus, status asthmaticus, severe dystonia, acute psychosis, and exercise experienced by military recruits in boot camp. The more strenuous or prolonged the exercise, the more damage is incurred. Excessive muscular activity results in a state in which ATP production cannot keep pace with demand, which subsequently exhausts cellular energy supplies and leads to a disruption of muscle cell membranes.[8] Rhabdomyolysis cases associated with low-intensity exercise have also been reported; in these cases, the mechanism remains unknown.[9]

Rhabdomyolysis is diagnosed clinically and with laboratory tests. Urine is dark, and while blood is present in the urine, there is a lack of urinary erythrocytes; myoglobinuria is present. Serum creatine kinase levels that are at least 5 times normal values, high serum levels of lactate dehydrogenase, aspartate aminotransferase, alanine aminotransferase, and potassium phosphate and a low serum calcium concentration are features of rhabdomyolysis.[1] Serum CK concentration, mainly the CK-MM subtype, is the most sensitive indicator of damage to muscles; serum CK levels begin to rise approximately 2 to 12 hours after the onset of muscle injury, peak within 24 to 72 hours, and then decline at the relatively constant rate of 39% of the previous day's value.[4] Complications of rhabdomyolysis include hypovolemia, compartment syndrome, arrhythmias and cardiac arrest, disseminated intravascular coagulation, hepatic dysfunction, acidosis and acute renal failure.[4] In movement disorders, several causes of rhabdomyolysis have been reported, including status dystonicus, myoclonus, generalized chorea and parkinsonism-hyperpyrexia syndrome in PD. In PD, LID leading to rhabdomyolysis is very rare complication of treatment. Two previously reported patients developed rhabdomyolysis following severe LID and recovered after reduction in the dosages of antiparkinsonian medications.[10] Although the number of known cases is limited, our case, together with the previously reported cases, suggests that the development of rhabdomyolysis and dyskinesia may be possible side effects of levodopa, especially following long-term use at high dosages. Further studies are needed to understand the mechanism of levodopa-induced dyskinesia leading to rhabdomyolysis.

Conflicts of Interest

The authors have no financial conflicts of interest.

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