Hypokalemic Periodic Paralysis Secondary to Dexamethasone Injection

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CE Earn Up to 7.5 Hours. See page 109.

Patients often present to the emergency department for evaluation and treatment of muscle weakness and paralysis. Hypokalemic periodic paralysis (HPP) is a rare skeletal muscle disorder characterized by abrupt onset of painless muscle weakness or paralysis that may last for a few hours or up to several days.[1,2] HPP is caused or exacerbated by a number of different underlying etiologies, primarily genetic, endocrine, gastrointestinal, renal, neurologic,[2] and viral[3] conditions. However, various medications,[2] strenuous exercise, stress, and carbohydrate-rich food[1] also may exacerbate this condition. In this article I present a patient who experienced muscle weakness or paralysis on multiple occasions subsequent to receiving a dexamethasone injection.

Case Report

A 27-year-old white man presented to the emergency department via private vehicle with a report of paralysis below the neck that began approximately 12 hours prior to arrival. The patient stated that he woke up the previous night and was able to move his fingers and toes but had paralysis in the remainder of his extremities; sensation remained intact. He described 2 previous instances of extremity weakness and paralysis that resolved spontaneously within several hours. He never sought medical care for these episodes because they resolved spontaneously, and he did not want to take time off from work to see a health care provider. He also thought the episodes were not serious because they only lasted a short time and resolved without intervention. He decided to come to the emergency department this time after his paralysis extended to his hands and feet and persisted for several hours. Further history taking revealed that the patient had received a dexamethasone injection for acute asthma exacerbations and/or acute musculoskeletal strain prior to each episode of extremity weakness and/or paralysis.

Upon arrival at the emergency department, although the patient reported lack of motor function below his neck, he stated that his sensation was intact. A review of systems was negative for chest pain, shortness of breath, headache, fever, chills, visual changes, syncope, loss of consciousness, mental status changes, paresthesia, vomiting, diarrhea, and back pain. His medical history was positive for hypertension (treated with amlodipine/olmesartan 5/40 mg) and hyperlipidemia (treated with rosuvastatin, 10 mg, and triple-strength fish oil, 3,000 mg) for the past year, along with asthma (treated with an albuterol inhaler). He denied previous hospitalizations, surgeries, tobacco or illicit drug use, and use of oral supplements. He reported occasional alcohol consumption of 1 to 2 glasses of wine 2 to 3 nights a week. His family medical history was negative for neurologic, rheumatologic, and endocrine disorders; however, he has a strong family history of premature coronary artery disease.

Physical examination revealed that the patient was alert and oriented to person, place, and time. Vital signs were as follows: oral temperature, 97.8°F; heart rate, 115 beats per minute; respirations, 20; and blood pressure, 144/92. A stat electrocardiogram revealed sinus tachycardia with flattened T waves in leads I, II, III, and V_{1-6}. The patient appeared well nourished and in no acute distress. His heart rhythm was regular and tachycardic without a murmur, rub, or gallop. His peripheral pulses were +2 in the bilateral upper and lower extremities. Lungs were clear throughout. Muscle strength was 0/5 in the bilateral upper and lower extremities with absent deep tendon reflexes. Sensation remained intact throughout. Cranial nerves II to XII remained grossly intact. Initial differential diagnoses included transverse myelitis, spinal cord compression, Guillain-Barré syndrome, electrolyte abnormality, acute stroke, multiple sclerosis, and neuropathy.

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Initial diagnostic study results obtained in the emergency department included normal findings of magnetic resonance imaging of the cervical, thoracic, and lumbar spine, a complete blood cell count, international normalized ratio, cardiac enzymes, and a complete metabolic panel with the exception of potassium, 1.5 mEq/L (normal range, 3.5-5.0 mEq/L) and magnesium, 1.6 mg/dL (normal range, 1.8-2.9 mg/dL). Additionally, nerve conduction velocity in the emergency department revealed diminished amplitude of all motor nerves.

The patient was treated with an immediate intravenous infusion and oral potassium repletion and was held in the emergency department for a neurology consultation. During the next 4 hours, the patient slowly began to regain motor function in his extremities and was admitted to an inpatient unit for continued potassium repletion. His serum potassium level 8 hours after beginning potassium repletion was 5.8 mEq/L, and potassium repletion was discontinued. The patient was able to walk without assistance 14 hours after admission to the emergency department. All diagnostic studies returned to normal limits within 18 hours of admission to the emergency department, and the patient was discharged home with follow-up appointments with internal medicine and neurology in 2 weeks; he was also advised to avoid use of corticosteroids.

Discussion

Muscle weakness and paralysis are not uncommon presentations in any emergency department, and patients present with these signs and symptoms as a result of a variety of etiologies. Episodic weakness is often seen as a result of renal, gastrointestinal, or other underlying systemic disease. Frequent causes of abrupt-onset paralysis include Guillain-Barré syndrome, transverse myelitis, medication overdose, and electrolyte imbalance. Thyrotoxic periodic paralysis closely mimics HPP and must be differentiated by evaluating thyroid function studies.

HPP is a rare genetic disorder classified as a channelopathy with weakness or paralysis and hypokalemia, which is usually normal between exacerbations.[4] The prevalence of HPP is 1:100,000, and although sporadic mutations have been reported, it is primarily autosomal dominant.[1] Mutations of genes *CACNA1S* and *SCN4A* are frequently associated with HPP,[5] and it can be described phenotypically as myopathic and paralytic.[4] The paralytic form is reversible, and it is interesting to note that skeletal muscles lose the ability to contract, whereas cardiac and respiratory muscles are not affected and sensation remains intact.[4,5] These episodes can last hours to days and can be triggered by infection, stress, high carbohydrate intake, or medications (eg, β-agonist, insulin, diuretics, and corticosteroids).[4,5] The myopathic type accounts for 25% of cases and is often characterized by exercise intolerance with slow, fixed, progressive muscle weakness of the lower extremities.[4]

Rapid assessment and diagnosis of HPP and its underlying cause is essential in the ED setting to prevent morbidity and mortality. Intravenous potassium repletion should be instituted to quickly reverse hypokalemia but should also be closely monitored to minimize rebound hyperkalemia during the recovery phase. Total daily potassium replacement should not exceed 200 mEq within the first 24 hours to avoid hyperkalemia and the associated sequelae.[5]

Frequent, recurrent episodes of HPP can be prophylactically managed with carbonic anhydrase inhibitors such as acetazolmide.[4] If limited response to carbonic anhydrase inhibitors is observed, potassium-sparing diuretics such as triamterene and spironolactone can be considered. Genetic counseling is important for patients with HPP. Nonpharmacologic interventions include a low-carbohydrate diet and avoidance of vigorous exercise.[1] Milder exacerbations may be eliminated with low-level exercise.[1]

Conclusion

Although rare, HPP can be seen in patients presenting to the emergency department with acute-onset muscle weakness or paralysis. This case illustrates the challenges of determining an effective diagnostic and treatment strategy with the plethora of primary and secondary causes for acute paralysis. Early identification and treatment can mitigate adverse cardiac, renal, and neurologic complications. Management of risk factors is essential in preventing or minimizing recurrence. It is essential to assess for secondary causes of HPP, such as dexamethasone, as seen in this case.

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