Acute Reversible Tetraplegia Induced by Hyperkalemia in a Patient with Paravertebral Mass due to Lymphoma

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Abstract

Acute onset of tetraplegia is a medical emergency. Hyperkalemia has been described as a very uncommon cause of tetraplegia. A 79 year old male presented with an acute onset of tetraplegia. His past medical history was significant for stage III follicular lymphoma diagnosed 4 years ago that 1 month prior to the admission progressed with biopsy-proven transformation to a diffuse large B-cell lymphoma. Recent CT and PET scans revealed multiple active lesions including a large paravertebral mass measuring 29 cm x 12 cm x 7 cm starting in the axial level of the diaphragm involving left psoas, the left renal vein and encasing the aorta. Multiple skeletal lesions, as well as numerous lymph nodes were found. The liver and spleen were also compromised. His surgical history was remarkable for radical prostatectomy for localized prostate cancer. The patient did not have previous history of renal failure and his blood urea nitrogen and creatinine were within normal range (15 mg/dL and 0.8 mg/dL, respectively).

Case Presentation

Two days prior to presentation, the patient began experiencing malaise, weakness and myalgia in his lower extremities, which progressed to paraparesis several hours later, and he was unable to ambulate without assistance. Thereafter he developed weakness in both upper extremities. On the day of admission he suddenly became completely tetraplegic. He denied seizures, chorea, rigidity, spasms, recent trauma, or any recent febrile or viral illness. He did admit to have experienced recent poor oral intake related to his generalized malaise, with no vomiting or diarrhoea and he denied recent use of non-steroidal anti-inflammatory drugs or any other new medications, including steroids).

On admission his vital signs were all within normal limits; the blood pressure was 136/76 mmHg, heart rate was 68 per minute, respiratory rate was 18 per minute, and temperature was 36.6 degrees Celsius. He was alert and oriented, in no distress with flaccid tetraplegia, areflexia, preserved cranial nerve function, normal rectal sphincter tone and absent plantar responses. The electrocardiogram showed wide QRS complexes, a shortened QT interval and tall peaked T waves. Laboratory testing revealed a serum potassium of 9.3 mEq/L, sodium 120 mEq/L, urea nitrogen 81 mg/dL, creatinine 9.9 mg/dL, calcium 8.5 mg/dL, phosphorus 4.1 mEq/L, creatine kinase 126 units/L, uric acid 9.6 mg/dL, Lactate Dehydrogenase (LDH) 1118 mg/dl (LDH was 1114, two weeks earlier), aspartate transaminase 28 IU/L, alanine transaminase 23 IU/L, and alkaline phosphatase 65 IU/L. Imaging studies were negative for hydronephrosis and there was no evidence of spinal cord involvement on chest, abdominal and pelvic CTs performed only two weeks before the admission.

While spinal cord compression was considered in the differential diagnosis, however hyperkalemia induced flaccid paralysis with severe acute renal failure were more likely causes and the decision was to initiate treatment of hyperkalemia and renal failure before proceeding with dedicated studies to rule out spinal cord compression. Spontaneous tumor lysis syndrome was also less likely since besides hyperkalemia, there was no change in blood levels of uric acid, calcium, phosphorus and LDH. The patient was immediately treated for hyperkalemia with intravenous calcium gluconate, intravenous hydration with normal saline, intravenous insulin and dextrose, inhaled albuterol, and oral polystyrene sulfonate resin. Within 60 minutes of initiating therapy for his hyperkalemia, the strength in his upper extremities was restored, further suggesting absence of cord compression. Thereafter the strength in his lower extremities recovered, regaining the mobility in his body after 4 hours of treatment. During the hospital course the patient did not experienced signs of autonomic dysfunction. Due to the rapid resolution of the symptoms and the clinical presentation EMG- NCV were not obtained. Search for potential causes of acute renal failure in this patient revealed presence of only decreased fluid intake and dehydration. Within 24 hours, his hyperkalemia and acute renal failure completely resolved and then the patient was started on chemotherapy and successfully discharged home.

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Secondary hyperkalemic paralysis is extremely rare and potentially life threatening condition. Although the precise mechanism of secondary hyperkalemic paralysis remains unclear, the persistent state of depolarization in the neurons is believed to be responsible for the reversible neuromuscular paralysis. It manifests clinically with an ascending pattern, from the lower extremities followed by a reversion of the initial changes.

References


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