

## 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×12 cm×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.



**Figure 1:** CT scan revealing a large paravertebral mass involving left psoas, the left renal vein and encasing the aorta.

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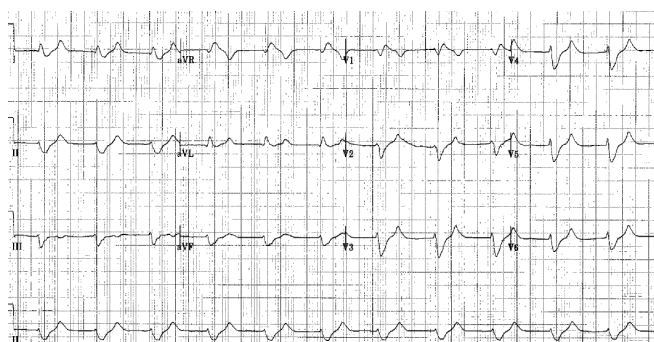
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**Figure 2:** CT scan revealing a large paravertebral mass involving left psoas.



**Figure 3:** Electrocardiogram on admission showing typical findings of acute hyperkalemia.



**Figure 4:** Electrocardiogram after the initial treatment for hyperkalemia showing reversion of the initial changes.

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 progression into the trunk and arms closely resembling the Guillain Barré syndrome [1,2]. In our patient, as well as most of other cases in the literature, there was no compromise of respiratory muscles, cranial nerves or sphincter tone [3]. Hyperkalemia induced flaccid paralysis has

been described mostly in the setting of end-stage renal disease, typically affecting lower extremities, and sometimes associated with ascending paralysis, but also focal neurological deficits have been described [4]. Shortening of the QT interval and peaked T waves (Figures 3 and 4), as well as other typical ECG changes are often observed in these patients [5-7]. If treatment for hyperkalemia is instituted promptly, complete recovery of the hyperkalemia-induced tetraplegia is achieved in 89% of patients [8]. However, the recovery of the symptoms is not related to potassium level or the concentration to which it is corrected [8,9].

Currently, about 70 cases of flaccid paralysis secondary to hyperkalemia were reported in the English literature. Our case is unique, since is the first one in the setting of lymphoma with no tumor lysis syndrome [8]. Moreover, our case had a large paravertebral mass (Figures 1 and 2) and the initial most likely diagnosis was paresis due to lymphoma involvement of the nervous system. Metabolic abnormalities need to be ruled out as potential causes of acute onset flaccid paralysis in oncological patients.

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