Hyperkalemia: A rare cause of acute flaccid quadriparesis

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Introduction
Primary hyperkalemic paralysis occurs from genetic defects in sodium channels and secondary hyperkalemic paralysis from diverse causes including renal dysfunction, potassium retaining drugs, Addison’s disease etc.[1] Acute flaccid quadriparesis (AFQ) induced by hyperkalemia has been described in only a few case reports. Here, we report a case of hyperkalemia-induced AFQ due to its rarity.

Case Report
A 73-year-old diabetic, hypertensive and hypothyroid female presented with the complaints of rapidly progressive ascending paraparesis, progressing to quadriparesis in about 10 h due to hyperkalemia. Patient was treated with antihyperkalemic measures. Her power improved dramatically as potassium levels normalized and she had an uneventful recovery.

On admission, she was conscious and oriented. Her pulse rate, blood pressure, respiratory rate and temperature were 72/min, 124/60 mmHg, 20/min and 98°F, respectively. Her oxygen saturation was 93% on room air. Neurological evaluation revealed normal pupils, symmetrical normal bulk and hypotonia in all limbs. Upper limbs had proximal and distal power of 2/5. In lower limbs, proximal and distal power was 2/5 with no sensory deficit. Deep tendon reflexes were absent. Bilaterally the plantar reflexes were mute. There were no lateralizing signs and the cranial nerves were normal. Other systemic examination was unremarkable. A clinical diagnosis of acute inflammatory demyelinating polyneuropathy was made. Arterial blood gas showed primary metabolic and respiratory acidosis and initial serum potassium level was 9.1 mmol/L. Other laboratory tests were normal. Electrocardiograph [Figure 1] showed tall T wave in anterior and lateral leads.

Raised potassium levels raised the possibility of hyperkalemia induced AFQ. Urgent antihyperkalemic measures in the form of calcium gluconate, insulin dextrose solution, furosemide, beta-2 agonist nebulization and calcium polystyrene sulfonate (K-bind) with sorbitol, were instituted. Patient required non-invasive ventilation (NIV) support for type-2 respiratory failure. Hemodialysis was not required as she had a good urine
output and serum potassium levels showed steady decline. Serial declining level of K: 9.1, 8.6, 7.1, 6.1, 4.4 was accompanied by improving power and resulted in weaning of ventilatory support within 48 h.

A nerve conduction velocity (NCV) study on day 1 was suggestive of demyelination. Subsequent NCV on day 4 was normal. She was shifted to the ward on day 4 by which time her biochemical parameters were normal. Electrocardiography on day 2 [Figure 2] showed normal T wave in anterior and lateral leads.

Discussion

Hyperkalemia is defined as measured serum potassium levels of >5.5 mEq/L. Clinical manifestations of hyperkalemia usually result from disordered membrane polarization. Cardiac manifestations are the most serious. Other symptoms include neuromuscular dysfunction, respiratory compromise and gastrointestinal signs. Our case was unique because she presented with AFQ and respiratory compromise requiring NIV support, which could be managed without hemodialysis.

Quadriplex induced by hyperkalemia has been described only in a few case reports. AFQ with no sensory deficit may mimic Guillain-Barré-syndrome (GBS).[7]

Autonomic disturbance, a remarkable feature of GBS is not a recognized feature of hyperkalemic ascending quadriplexes, although it has been described in other situations associated with hyperkalemia.[8]

The exact mechanism of secondary hyperkalemic paralysis is not clear. It is thought to be due to direct action of potassium on the muscle fiber and cell membrane,[9,10] whereas some believe that a functional peripheral neuropathy induced by high serum potassium level is responsible.[9,11]

The most frequently reported causes of hyperkalemia-induced AFQ are renal insufficiency,[2,3,5,6] use of potassium sparing drugs[12] or a combination of both.[3] One case was due to tumor lysis syndrome.[3] Consistent with these reports, our patient was receiving an angiotensin converting enzyme inhibitor (Ramipril), Beta-blocker (Atenolol) and a potassium sparing diuretic (Aldactone). In these case reports, potassium levels ranged from 8 mEq/L to 9.69 mEq/L[3,5,6] comparable to ours (9.1 mEq/L).

The quadriplex is usually ascending, with areflexia, normal sensory function and normal cranial nerves. In all case reports, quadriplex reversed regardless of how the hyperkalemia was treated. As in our patient, reversal started almost immediately after treatment, with full strength returning over the next few hours.

To conclude, as hyperkalemia may be a potentially life-threatening but rapidly reversible cause of quadriplex, a strong index of suspicion should be the key to early diagnosis in any patient presenting to the emergency room with features of AFQ especially in those who are on drugs with potential to cause hyperkalemia.

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