Case Report

Hyperkalemia: A rare cause of acute flaccid quadriparesis

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Abstract

Acute flaccid quadriparesis secondary to hyperkalemia is a very rare and serious but reversible medical emergency. We present a case of a 73-year-old female who was admitted with 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.

Keywords: Acute flaccid paralysis, hyperkalemia, 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 AFQ in about 10 h. She woke up with bilateral lower limb weakness with gradual inability to move upper limbs and had developed difficulty in breathing by the time she presented to the hospital. She was on glimipride, metformin, thyroxine, atenolol, ramipril and spironolactone. There was no other significant history.

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 4/5. 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 mixed primary metabolic and the 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

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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.

Quadriparesis induced by hyperkalemia has been described only in a few case reports.[1-6] AFQ with no sensory deficit may mimic Guillain-Barré-syndrome (GBS).[7]

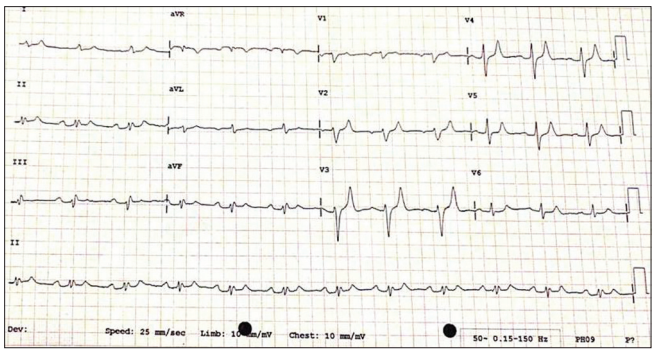

Figure 1: Electrocardiogram on day 1 showing tall T wave in anterior and lateral leads

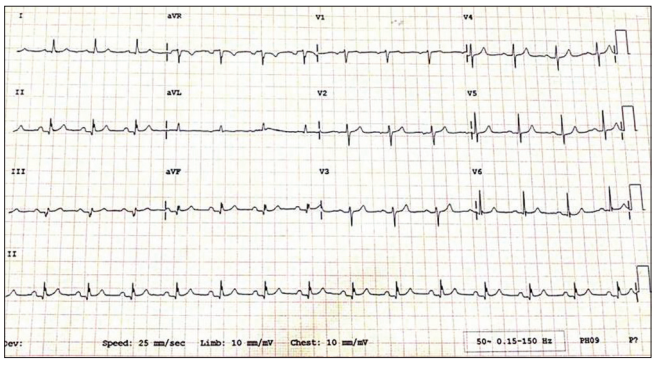

Figure 2: Electrocardiogram on day 2 showing resolution of tall T wave in anterior and lateral leads

Autonomic disturbance, a remarkable feature of GBS is not a recognized feature of hyperkalemic ascending quadriparesis, 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 quadriparesis is usually ascending, with areflexia, normal sensory function and normal cranial nerves. In all case reports, quadriparesis 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 quadriparesis, 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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