

## Case Report

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# Flaccid Motor Paralysis Induced by Hyperkalemia

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### Abstract

Neurological manifestations are not commonly reported with hyperkalemia. We report a case of flaccid muscle paralysis induced by profound hyperkalemia.

**Keywords:** Flaccid paralysis; Hyperkalemia; Acute renal failure

### Introduction

Hyperkalemia, which is a rare occurrence in normal subjects, is the commonest electrolyte manifestation in renal impairment, and symptoms and signs mainly limited to cardiac and neurological systems. However, neurological manifestations are not commonly encountered in clinical practice. According to the case review of 73 patients common presentations of secondary hyperkalemic paralysis were diminished reflexes, quadriparesis/paralysis, respiratory involvement and sensory loss. Complete recovery was achieved in 89% of cases and did not correlate either with the absolute potassium level or the degree to which it was corrected [1]. We report a case of hyperkalemic paralysis with pure motor involvement in a patient with acute on chronic renal failure.

### Case Report

An 80 year old, known patient with diabetes and hypertension for nearly 3 years, had been on metformin and nifedipine, presented with progressive ascending weakness of all four limbs and difficulty in breathing for 6 to 8 hours. Further into history revealed, loss of appetite and epigastric pain for 2 weeks, vomiting for 3 days and reduction of urine output for nearly 5 days.

He was conscious and rational, and had heart rate of 110 beats per minute and blood pressure of 90/50 mmHg. His respiratory rate was 35-40 breaths per minute and had poor cough. His breathing was rapid and shallow rather than kussmaul pattern, and was saturating 98% with 4 L of oxygen. Neurological examination revealed only flicker of movement (Medical Research Council scale/MRC for muscle power grade 1) in both upper and lower limbs, and absent reflexes with down going plantar. He had normal function of cranial nerves and normal sensation. His abdomen was soft.

Life threatening hyperkalemia was diagnosed on 12 lead Electrocardiography (ECG) (Figure 1) and treated with calcium gluconate, insulin dextrose infusion, salbutamol nebulisation and oral calcium resonium. Initial blood investigation showed serum potassium (K<sup>+</sup>) 10.1 mEq/L, sodium (Na<sup>+</sup>) 125 mEq/L, creatinine 11 mmol/L and random blood sugar (RBS) – 220 mg/dl. After 10 hours of treatment in the intensive care unit his weakness improved to the level where he was able to move all four limbs against gravity and against some resistant (MRC muscle power grade 4) and his rapid shallow breathing settled. K<sup>+</sup> came down to 8.7 and then to 5.8 mmol/L and ECG abnormalities disappeared. Rest of the laboratory test results showed Haemoglobin (Hb) 10.1 g/dl, white cell count (WCC) 11200 per Cumm, Platelets 420,000 per Cumm, aspartate aminotransferase (AST) 42 U/L, alanine



Figure 1: On admission ECG.

transaminase (ALT) 35 U/L, Troponin T- negative, Chest X ray -normal, Urine full report (UFR) - few pus cells.

Peritoneal dialysis was started and stopped after 150 cycles since his creatinine came down to 4.4 mmol/L and urine output improved to more than 1 litre per day. He had creatinine of 2.0 mmol/L on discharge, and renal function assessment done 2 weeks later showed creatinine clearance of 45 ml/min.

### Discussion

Hyperkalemia is defined as serum potassium concentration of greater than 5.5mEq/L. Renal impairment is the major cause of hyperkalemia, accounting for 75% of cases [2].

Hyperkalemia is often asymptomatic and diagnosed on routine

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laboratory test. When symptoms are present, they are non specific and predominantly cardiac and neurological. Nevertheless, neurological manifestations are sparsely reported in the literature.

Although, ECG is a useful tool to diagnose hyperkalemia prior to laboratory evidence, it is not a very sensitive method [2]. Various studies showed poor correlation between ECG changes and serum potassium level [3]. In a study by Acker and colleague nearly half the patients with serum potassium more than 6 mEq/L did not have ECG changes. Furthermore, some patients showed gradual progression in changes, others showed progression from benign to more fatal arrhythmias without warning [4]. However, hyperkalemia was clinically suspected in this case by ECG on admission. Chronicity of the process may also have an influence on symptom and signs. Pre treatment potassium level of this patient was 10.1 mEq/L, and he did not have lethal arrhythmias.

Most common neurological manifestation in hyperkalemia is ascending quadriparesis or quadriplegia with diminished or absent reflexes as in this case. Respiratory failure, sensory loss, bulbar weakness and myalgias could also occur [1]. The serum potassium concentration leading to hyperkalemic paralysis ranged from 7.0 to 11.2 mEq/L with a mean of 9.0 mEq/L [5]. However some cases have been reported with potassium level between 5 mmol/l and 7.0 mmol/l [1]. This highlights lack of consistent correlation between the serum potassium level and the severity of the weakness.

Chronic renal failure is the commonest underlying cause for hyperkalemia as in our patient. Other causes reported in the literature are exogenous potassium supplementation, medications, addison disease and rhabdomyolysis [1].

The mechanism of secondary hyperkalemic paralysis is unclear. The weakness has been attributed to abnormalities at various locations in the motor axis, including the muscle, nerve and neuromuscular junction. Nerve conduction studies showed decreased nerve conduction velocities and increased F wave latencies, but Electromyography (EMG) studies were normal. On the basis of nerve conduction studies, abnormal depolarization of the nerve membrane described as a possible cause of secondary hyperkalemic paralysis [5]; however, exact mechanism still unknown. Electrophysiological studies were not done in this case.

In summary, although hyperkalemic paralysis is not a common occurrence, clinicians should be aware of the potentially life threatening non cardiac complications of hyperkalemia.

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