

Severe Muscle Weakness due to Hyperkalemia

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Abstract

Hyperkalemia is a commonly encountered electrolyte disturbance in patients with renal insufficiency. It develops very rapidly when potassium is supplemented while a patient is on a potassium-sparing diuretic. Most often it remains asymptomatic and manifests in the form of electrocardiographic changes. Muscle weakness and paralysis although described is seldom observed in clinical practice. We report one such case. ©

INTRODUCTION

Hyperkalemia, which is a rare occurrence in normal subjects, is a common electrolyte disturbance in patients with renal insufficiency. It develops rapidly when potassium is supplemented and/or used with a potassium-sparing diuretic. It is often asymptomatic and picked up on routine blood tests. The manifestations observed with hyperkalemia are limited to abnormal cardiac conduction and muscle weakness. In clinical practice however, muscle weakness as a manifestation is rarely seen. This may be because cardiac manifestations usually begin before muscle weakness and appropriate measures are taken before potassium concentration reaches a level at which weakness manifests. We report a case where a patient of renal insufficiency rapidly developed severe hyperkalemia following potassium supplement and use of potassium-sparing diuretic and developed severe muscle weakness.

CASE REPORT

A 50-year-old lady underwent a cadaveric renal transplant in October 2001. Her post-transplant course was complicated with ischemic acute tubular necrosis. She was supported on hemodialysis for the first 2 months post-transplant. She gradually came off dialysis and her serum creatinine stabilized at 3.0 mg/dl by January 2002. On her regular follow up in the clinic in March 2002 (5 months post-transplant); she had a rise in serum creatinine to 3.6mg/dl. An ultrasound of the transplant kidney revealed obstruction at the uretero-vesical junction. She was admitted for corrective surgery. Her intra-operative course was complicated by hypotension. She had a decreased urine output post-operatively, for which she was put on diuretics. There was a progressive azotemia but

potassium tended to be low (probably due to high dose of frusemide). Aldactone was added to maintain diuresis and achieve normal potassium. However, the potassium continued to be low (Table 1). She developed paralytic ileus, which was felt to be due to low potassium. At this stage, she was kept nil by mouth and started on intravenous fluids with potassium supplement (80 mEq/L day). The potassium normalized to 4.8 mEq/L on the next day. At this stage, potassium supplement was reduced to 40 mEq/day. The next day morning her s. potassium was 6.5 mEq/L. The potassium supplement was stopped and aldactone was held. In the afternoon at 2 p.m., she complained of weakness in both the lower limbs, which spread to the upper limbs, trunk and neck muscles by evening 8 p.m. On examination, she was normothermic and hemodynamically stable. She was alert and fully oriented. Her cranial nerves were intact. The power in the lower limbs was 0/5 and in the upper limbs was 1/5. The sensations were intact. She had areflexia and her plantar responses were flexors. A repeat blood sample for serum potassium was sent

Table 1: Trend of potassium after surgery

Dates	Potassium	Signs and symptoms	Treatment given
31/03/02	3.81		Frusenex
1/04/02	3.41	Oliguria	Aldactone
2/04/02	3.1		
3/04/02	3.2		
4/04/02	3.1		
5/04/02	3.0		
6/04/02	2.9	Paralytic ileus	Intravenous KCl 80 mEq/day
7/04/02	4.8		Intravenous KCl 40 mEq/day
8/04/02	6.5		K ⁺ supplement stopped Aldactone held
	9.3	Inability to move limbs, inability to lift the neck. ECG- Sine wave pattern	Hemodialysis

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which showed potassium to be 9.3 mEq/L. The electrocardiogram (ECG) showed a sine wave pattern. She was given 40 ml of 10% intravenous calcium gluconate and immediately taken up for hemodialysis. Thirty minutes after the dialysis was initiated, she could move her arms. Gradually she had a complete recovery.

DISCUSSION

Hyperkalemia is a common electrolyte disorder in patients with renal insufficiency. The occurrence of symptoms and signs of hyperkalemia depends on the chronicity of the disorder. The changes induced by hyperkalemia are essentially limited to abnormal cardiac conduction and muscle weakness, which can lead to potentially fatal arrhythmias and paralysis.¹ Although these cardiac and neurological complications have been described in the literature, in routine practice, cardiac manifestations are commonly encountered. An ECG can provide a rough idea about the severity of the hyperkalemia. The common findings are tall T waves, widened QRS, prolonged PR interval and loss of P wave. These changes can progress rapidly to fatal arrhythmias.² Fortunately, our patient did not progress to fatal arrhythmia.

Neurological manifestations have been less often reported.² The muscle weakness associated with hyperkalemia appears to result from changes in neuromuscular conduction. The increase in plasma K⁺ concentration reduces the ratio of intracellular K⁺ concentration to that in the ECF, resulting in a decrease in the magnitude of resting membrane potential. Although, this should increase membrane excitability (since less of a depolarizing stimulus is required to generate an action potential), the effect seen in patients is different. Persistent depolarization inactivates sodium channels in the cell membrane, thereby producing a net decrease in membrane excitability that may be manifested clinically by muscle weakness or paralysis.³

Recently a similar case was reported where a patient of renal insufficiency developed flaccid paralysis and was found to have potassium of 10 mmol/L. This patient also had renal insufficiency. An electromyogram done before hemodialysis showed profoundly decreased nerve conduction blocks,

which reversed completely post-hemodialysis. The authors concluded that the process causing hyperkalemic paralysis originates at the level of nerve rather than the muscle.⁴

Muscle weakness typically does not develop until the plasma K⁺ concentration exceeds 8 mEq/L. However, patients with periodic paralysis may become symptomatic at a plasma concentration around 5.5 mEq/L, probably because abnormal membrane function is the primary defect in this disorder.

Vague muscle pain is usually the first symptom of hyperkalemia. This is followed by muscle weakness. It is usually noticed in the legs, then in the trunk and in the arms. The facial and the respiratory muscles are affected last. In spite of muscle paralysis, the patient usually remains alert until any cardiac event due to hyperkalemia. These changes revert after bringing the potassium level to normal.⁵ This was exactly the sequence of events observed in our patient.

In summary, this case highlights that hyperkalemia should be considered a possible etiologic factor in all cases of paralysis. Potassium supplements along with potassium sparing diuretics can cause severe hyperkalemia particularly in the setting of renal insufficiency. A timely diagnosis and correction not only completely reverses the paralysis but avoids fatal cardiac arrhythmia.

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