CASE REPORT

Hyperkalemic quadriparesis in a patient of ESRD

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Abstract

Hyperkalemia is an ominous complication of renal failure. It may present with cardiac or neuromuscular abnormalities. Rarely, it may present with flaccid, areflexic quadriparesis with no sensory involvement. We report a patient of ESRD, who presented with flaccid quadriparesis due to hyperkalemia and recovered dramatically following a session of HD.

Key Words: Hyperkalemia, quadriparesis, end stage renal disease.

Introduction

Hyperkalemia is associated with sinister significance in patients of renal failure. It may usually present with cardiac or neuromuscular abnormalities; occasionally it may be asymptomatic. Literature review revealed the occurrence of quadriparesis, ascending muscular weakness and hemiplegia in association with hyperkalemia. We report a patient of End Stage Renal Disease (ESRD) on irregular hemodialysis (HD), who presented with rapid onset flaccid quadriparesis due to hyperkalemia.

Case Report

Mr. G. R, 55 year old man, a patient of ESRD, hypertension and cardiac dysfunction is on irregular follow up. He had his last HD 3 weeks prior to this admission. He presented with sudden onset extreme weakness of all the 4 limbs, swelling of the whole body and breathlessness of grade IV severity. There was no history of taking potassium sparing diuretics, beta blockers, non steroidal anti-inflammatory drugs, ACE inhibitors or Angiotensin receptor antagonists. Evaluation revealed flaccid quadriparesis with no sensory abnormalities, severe azotemia (S. Creatinine: 14 mg/dl) and hyperkalemia (S. Potassium: 5.9 mEq/L). ECG showed typical features of hyperkalemia (Fig 1). Following HD support, the quadriparesis improved, the serum K+ came down to 5.3 mEq/L and the ECG abnormalities disappeared (Fig 2).

Discussion

Hyperkalemia is a life threatening complication occurring in patients with renal failure. Though muscle paralysis is an uncommon manifestation in hyperkalemia, it responds well to dialysis. The transmission of a nerve impulse from nerve endings to the motor end plate of muscle is due to the release of acetylcholine. Further conduction of the stimulus along the muscle fibre is related to electrical currents produced by potassium. Normally, the membrane of the muscle fibre is polarized due to differences in the concentration of potassium between inside and outside of the cell. In hyperkalemia, the increase of extracellular potassium decreases the transmembrane potassium gradient, there by causing lowering of resting membrane potential, which prevents the transmission of a stimulus along the muscle fibre. Sometimes, hyperkalemia also stimulate the pain receptors of peripheral nerves accounting for paraesthesias. Evers et al described progressive flaccid quadriparesis due to hyperkalemia attributed to spironolactone in a patient of renal failure and improvement with HD. Khullar et al reported hyperkalemic quadriparesis in a patient of CRF. Esposita et al reported neuromuscular abnormalities due to hyperkalemia in a patient of CRF and improvement with HD. Similar observation of association of flaccid quadriparesis with hyperkalemia was noted in our patient, which responded to a session of HD.

This is submitted to share the unusual manifestation of hyperkalemia and its dramatic improvement with dialysis and to invite feedback from the fraternity.
References


Figure 1 showing ECG abnormalities in hyperkalemia before HD (Tall peaked T wave with a narrow base, absent P wave and widened QRS interval)

Figure 2 showing disappearance of ECG abnormalities due to hyperkalemia following HD