

ACUTE FLACCID PARALYSIS SECONDARY TO DRUG INDUCED HYPERKALEMIA

Neurology

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ABSTRACT

Hyperkalemia-induced acute flaccid paralysis secondary to familial hyperkalemic periodic paralysis is well described. However, secondary hyperkalemic paralysis in the absence of an underlying genetic disorder has only been described in a handful of case reports. Herein, we report a case of a fifty year gentleman presenting with drug induced secondary hyperkalemic paralysis, where the patient recovered without any deficits, after correction of hyperkalemia.

KEYWORDS

Hyperkalemia, Periodic Paralysis, Hypotonia

CASE REPORT

A fifty year gentleman, who is a hypertensive, diabetic and CAD patient, presented with one day history of weakness of all four limbs. It started suddenly in the left upper limb first, when he woke up from the sleep. Few hours later, the weakness also involved right upper limb and both lower limbs. He also had neck weakness and dyspnea. He also had generalized muscle ache. There was no sensory disturbances, cranial nerve or bladder and bowel involvement. Examination showed hypotonia and symmetrical weakness of all limbs involving proximal more than distal regions. Deep tendon reflexes were normal. There were no sensory signs or cranial nerve involvement. Investigations showed Urea- 97mg/dl, creatinine- 2.7mg/dl and potassium – 7.4 mEq/L. ECG showed tall T waves, ectopic atrial rhythm followed by junctional rhythm (Fig-1). Anti hyperkalemic measures (Calcium gluconate, insulin, Salbutamol nebulisation) were initiated and his symptoms started improving within six hours and he completely recovered by next day. There was sensory motor axonal neuropathy involving all four limbs on nerve conduction study. The cause of hyperkalemia was found to be drug induced- spironolactone and enalapril (which he was consuming for CAD), and acute on CKD.

DISCUSSION

Secondary hyperkalemic paralysis is characterized by vague muscle pain with subsequent muscle weakness, generally occurring in an ascending pattern, with facial and respiratory muscles affected last. Patients usually experience areflexia, flaccid motor paralysis, with intact sphincter tone and sensory function. Onset commonly occurs over a few days but may be more sudden in onset. These nonspecific symptoms make the diagnosis difficult; however, elevated serum potassium and possible ECG changes help to immediately distinguish the diagnosis. In this case, the sudden onset and progression were most likely related to the rapid rise in his serum potassium causing neuromuscular conduction alterations to produce his symptoms. The mechanism of muscle weakness and paralysis is likely due to abnormal nerve membrane depolarization secondary to changes in potassium gradient and resting membrane potentials.

CONCLUSION

This case of secondary hyperkalemic paralysis emphasizes the importance of including this in the differential in a patient presenting with acute flaccid paralysis. A diagnosis of secondary hyperkalemic paralysis can be challenging given the wide variety of causes of flaccid paralysis, and early ECG changes and elevated serum potassium can assist in making the diagnosis. Timely reversal of paralysis not only relieves symptoms but also prevents the development of potentially life-threatening dysrhythmias.

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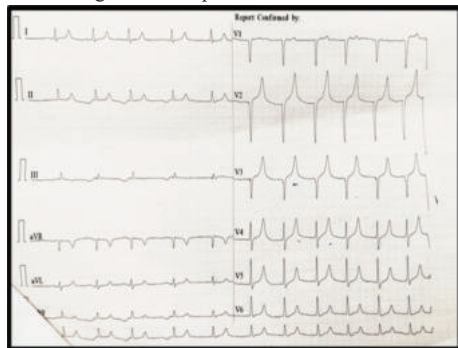


Figure-1 Showing ECG Changes