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Case Report

A CASE OF HYPOKALAEMIC PERIODIC PARALYSIS

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ABSTRACT
Hypokalaemic periodic paralysis is a medical emergency when patients present with acute onset paraparesis usually noticed in the mornings secondary to low serum potassium levels with a prevalence of 1 in100, 000. The symptoms resolve promptly with correction of potassium. The patient experiences motor symptoms while the sensation is preserved and can be differentiated from acute inflammatory demyelinating polyneuropathy with preserved ocular, bulbar or respiratory involvement.

Keywords: *Hypokalaemic Periodic Paralysis*

INTRODUCTION
Hypokalaemic periodic paralysis (HPP) presents clinically in similar ways as acute demyelinating polyneuropathy but the hall mark of HPP is low serum potassium levels. The symptoms revert completely with potassium replacement and patients can have recurrent presentations and cases have been reported where they have been discharged on long term potassium supplements, Acetazolamide or Spironolactone.
Patients usually present early hours in the morning or after rest following strenuous exercise or a high carbohydrate meal (Abbi *et al.,* 2011). Cases have been reported where the symptoms were triggered by trauma, infections, emotional stress, cold, alcohol and drugs like diuretics, steroids and insulin (Soule and Simone, 2008). The symptoms can last hours to days and are usually transient.

CASES
A 23yr old man presented with generalised weakness of few hours duration. He woke up in the morning to find that he was unable to move his arms and legs. There was no respiratory distress. He experienced diarrhoea about twice a day for 2days which was about 3 days prior to his presentation. Neurological examination revealed reduced power 2/5 in all the limbs with hypotonia, hyporeflexia in all the joints and down going plantars. There was no sensory deficit. The respiratory rate was 16/minute and Blood Pressure 136/75 mm Hg.
Bloods showed low serum potassium of 2.0 with normal sodium and other electrolytes. Blood gas showed an acidotic picture with a pH of 7.28 and HCO3 of 19. ECG showed sinus tachycardia of heart rate 105/minute. A spirometry could not be performed. The symptoms improved following intravenous administration of potassium chloride (KCL).

DISCUSSION
Hypokalaemic periodic paralysis can present as a medical emergency. Patients usually experience weakness in all four limbs associated with hypokalaemia. The mechanism is due to activation of Na/K/ATPase pump leading to influx of potassium into the cells thus causing reduced serum potassium levels. Androgens activate the pump too; hence the condition is seen more commonly in men than women (Guerra *et al.,* 1987).
HPP may be primary or secondary. The primary hypokalemic periodic paralysis is autosomal dominant in inheritance and involves mutations in CACNA1S and SCN4A genes affecting calcium and sodium channels (Venance *et al.,* 2006). Mutation of KCNJ2 gene has been noted to cause HPP and arrhythmias known as Anderson Tawil syndrome (Cannon, 2002). Primary HPP is exacerbated by various factors like cold, strenuous exercise, high glycaemic index foods, stress, diarrhoea and drugs.

Secondary hypokalaemic periodic paralysis has been reported in association with renal tubular acidosis, Barter syndrome, Gitelman's syndrome, villous adenoma of colon and hyperthyroidism (Bagga and Dutta, 1994). Rao *et al.,* (2006) have noted 3 cases of HPP that were associated with Sjogren's syndrome. Sharma *et al.,* (2014) reported 2 cases of hypokalaemic periodic paralysis in 2014 that underwent nerve conduction studies which reversed after recovery. They noted reduction in amplitudes of CMAPs (compound muscle action potential) and non recordable F wave latencies on motor conduction studies which reached normal levels with potassium replacement. Another study done previously on 10 patients revealed reduced sensory action potentials during the paralysis which normalised following correction of hypokalaemia. They have not found any motor nerve dysfunction (Inshasi *et al.,* 1999). The mechanism was attributed to inactivation of sodium-potassium pump due to low potassium which in turn caused neuronal inexcitability, mainly in the dorsal root ganglia owing to an incomplete blood nerve barrier. This is very interesting as nerve conduction studies are not routinely performed in HPP.

ECG changes in HPP include the changes usually seen in hypokalaemia which are prolonged PR interval, T wave flattening or inversion with ST depression, appearance of U waves and QT prolongation. All of these changes may not be typically present. It has been noted in patients with thyrotoxic periodic paralysis, tachycardia being a predominant ECG finding. Atrial fibrillation, atrioventricular blocks, ventricular fibrillation and asystole are the other features that have been reported (Boccalandro *et al.,* 2003; Miyashita *et al.,* 2006).

Treatment includes potassium supplementation intravenously not faster than 10mmol/hr to avoid rebound hyperkalaemia. Prompt treatment is mandatory as severe dyselectrolytaemia can cause asystole and cardiac arrest. Patients should be warned to avoid trigger factors and maintain adequate hydration.

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