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Case Study

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A CASE STUDY ON HYPOKALEMIC PERIODIC PARALYSIS

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ABSTRACT

Hypokalemic periodic paralysis is an uncommon neuromuscular disorder which occurs 1:100,000especially in firstand second decade of life. It results from mutations in sodium, calcium and potassium channels in the skeletal muscles, affected individuals usually regain their muscle strength between attacks, repeated episodes can lead to persistent muscle weakness later in life. Here the case report of a 43year old male patient is being presented who got admitted to emergency with the complaints of weakness of both upper and lower limb, since morning and the patient felt difficult in walking. Patient had similar illness7 years before in the past and took treatment for 3 years with syrup potklor (potassium chloride)-2 tsp. Now 7 years later patient is being experienced with similar attack and got treated up by giving syrup potassium chloride 10 ml (diluted with 100 ml water).

KEYWORDS: Hypokalemia, Neuromuscular disorder, potassium chloride.

INTRODUCTION

Hypokalaemia periodic paralysis may be a rare autosomal dominant channelopathy which is characterized by muscle weakness or paralysis when there's a fall in K+ levels within the blood. Mutation commonly occurs on awakening or after sleep or rest following strenuous exercise, high carbohydrate meals with high sodium content, sudden changes in temperature and even excitement, noise, flashing lights, and cold temperature. Weakness may be mild and limited to certain muscle groups or full-body paralysis. The mutation occurs in following genes CACNA1S (Voltage-gated calcium channel Cav1.1found in transverse tubules of skeletal muscle cells), SCN4A (A Voltage gated sodium channel Nav1.4 found at neuro muscular junction) or KCNJ2 (An inward rectifier potassium channel KIr2.1) Mutations of the CACNA1S gene is responsible for approximately 70% cases and SCN4Agene is responsible for rest of the cases of HPP.[1]

The mutation alters the structure and function of these channels which can disrupt the regulation of muscle contraction leading to severe muscle weakness or paralysis episodes. Inpatient with mutations in SCN4A/ CACNA1S), the channel has reduced excitability, and signals from CNS are unable to depolarize the muscle. As a result, the muscle cannot contract efficiently resulting in paralysis. The condition is hypokalemic because a low extracellular k+ ion concentration will cause the muscle to repolarise to the resting potential. Quickly, it becomes more difficult to reach the calcium threshold at which the muscle can contract.

Hypokalaemic paralysis results from either alteration in the transcellular distribution of potassium or depletion of potassium renally or extra renal. Most cases are due to the transcellular shift of potassium and the other differentialdiagnosis includes familial periodic paralysis, thyrotoxic periodic paralysis, or barium poisoning.[2]

CASE REPORT

A 43year old male patient of 43 kg weight came to hospital with the complaints of difficulty in walking, difficulty in lifting his head and weakness of upper and lower limb since morning. History of similar episodes before seven years and received treatment for 1 month and now evaluated as hypokalemic periodic paralysis. Patient was a chronic smoker for 30 years and alcoholic for 2 years. General examination revealed that patient was conscious oriented, his CNS functions were normal. On examination of motor system tone was found to be hypertonic, power 2/5, reflex was observed to be brisk and plantar reflex shows flexor on both right and left of upper and lower limbs. Blood pressure was observed to be 130/90 mm Hg and pulse rate was 80 bpm. Laboratory investigation include hematology WBC 17.9 x 10^9

/L (normal range 4-10 x 10^9 /L), platelets was raised to 345 x 10^9 /L (normal range 100-300 x 10^9 /L), serum electrolytes shows potassium 1.2 mEq/L (decreased) (normal range 3.5 -5 mEq/L), Calcium 1.06 mEq/L and

sodium 143 mmol/L on first day. With this background of clinical observation and laboratory assessment hypokalemic periodic paralysis was confirmed. ECG and thyroid profile had no significant changes .The primary treatment given to the patient was syrup Kcl 10 ml diluted with 100 ml of water given thrice a day along with Intravenous fluid like ringer lactate 500 ml and normal saline 500 ml, Injection pantoprazole 40 mg IV twice a day and Inj emeset 8 mg IV (SOS) was given. Syrup KCL 10 ml has been shown to reduce the symptoms of hypokalemic periodic paralysis. On the second day potassium level was found to be 2 mEq/L, On the third day patient attained 3.5 mEq/L and the patient attained 4mEq/L of potassium on fourth day.

DISCUSSION

The most prominent clinical features of hypokalemia (potassium depletion) are neuromuscular, although other systems such as cardiovascular and gastrointestinal, are also involved. Patients generally complain about muscular weakness, especially in the lower extremities, and marked tremendous weakness of skeletal muscles is common with more severe potassium depletion. In addition to decreased motor power, other physical examinations such as decreased or absent tendon reflexes may also be demonstrated. A very severe hypokalaemia will result in complete paralysis of respiratory, bulbar, and cranial musculature, which is the most prominent cause of mortality risk due to respiratory depression and arrhythmia.[1]

The patient has experienced weakness, which is a common but an albeit non-specific, presentation in both the emergency and outpatient setting. Although the differential diagnosis for the complaint of weakness is extensive, the focus is considerably narrowed when a patient presents with a demonstrable decrease in muscle strength on physical exam3. Patients may experience abortive attacks-daily weakness which varies from hour to hour or day today. These attacks can last for months and maybe mistaken for permanent weakness. Abortive attacks become more common in patients who are in their 40's. Some patients may experience having paralysis when they are in their 40s, but it would be an abortive attack. Abortive attacks can be more troublesome than paralytic attacks because young patients usually feel strong between attacks of paralysis and patients with abortive attacks rarely feel strong.[4] In the case of hyperkalemia paralysis, muscle weakness is accompanied by hypertonia. An Andersen-Tawil syndrome characterized a triad of episodes which includes muscle weakness, cardiac abnormalities (ventricular arrhythmias, prolonged Q-T intervals, prominent U waves), and distinctive skeletal features. Features of thyrotoxicosis may also be present if it's a thyrotoxic periodic paralysis. None of these conditions were observed in our patient.[1]

Sweets or starchy foods trigger attacks because these foods cause potassium to move into the muscle cell, and lower the level of potassium in the blood, which is also troublesome in our patient. Potassium (by mouth or by an IV line) can reduce the incidence of this attack. Alcohol can also trigger HPP in predisposed patients as in the case of beer, can contain 13 grams of aldehyde, so high carbohydrate content can result in insulin surge producing a rapid intracellular shift of potassium through N-KATPase channels, thus producing hypokalemia.[5] Therefore, to prevent HPP avoidance of alcohol consumption is also important in this patient.

HPP also has a male preponderance. The age of onset of HPP is earlier in males than in females. Moreover, males are more prone to have a symmetrical weakness while asymmetrical is common in both genders. Primary hypokalaemia paralysis is more frequent than secondary.[6]

CONCLUSION

Periodic Paralysis is an important consideration while seeing a patient with sudden onset weakness or paralysis, especially those with no history or evidence of other diseases and no significant risk factors for stroke. Failure to properly diagnose and treat Periodic Paralysis can increase the mortality and morbidity but a rapid correction of potassium abnormalities can resolve the symptoms quickly and completely. When possible, the underlying cause must be adequately addressed to prevent the persistence or recurrence of paralysis.

Management of HPP involves the treatment of acute attacks and the prevention of further attacks. For the acute management of hypokalaemia, potassium chloride is administered as 30mmol orally every 30 minutes until serum potassium normalized. Simultaneous cardiac monitoring is also needed. Slower rates of administration (10mmol per hour), has also been recommended to minimize rebound hyperkalemia.

The carbonic anhydrase inhibitor (acetazolamide) is often a more prescribed choice for HypoKPP patients as this medication helps to keep the potassium from getting out of balance in the first place, by affecting the mechanism that moves potassium from the blood into the cell. Other carbonic anhydrase inhibitors called dichlorphenamide, and methazolamide is found to be more potent, and often works with patients who have become resistant to acetazolamide after prolonged use of the drug.[1] About 25% of patients do not respond to acetazolamide and must be put on other drugs. Patients who take carbonic anhydrase inhibitors must be alert to potentially dangerous drug interactions as they slow the excretion of other drugs from the system. Patients with carbonic anhydrase inhibitors generally need to take some potassium to replace what is lost due to the therapy itself. Most patients with HypoKPP use potassium to abort developing episodes. Patients differ in their reactions to different forms of potassium, but, as a rule, most people find that the effervescent potassium citrate or bicarbonate tablets that dissolve in water are the most

effective and easiest on the stomach. Potassium chloride tablets are slow to dissolve and are hard on most people's stomachs.

The risk of rebound hyperkalemia is lowest with the oral route, possibly due to the kaliuretic reflexes arising from putative potassium receptors in the gut.[7] Intravenous potassium that should be avoided whenever possible; however, it is indicated for arrhythmia due to hypokalaemia or airway compromise due to ictal dysphagia or accessory respiratory muscle paralysis. Milder attacks can be aborted by low-level exercise. For prevention, lifestyle measures and drug treatment are the available options. Low-carbohydrate diet and avoiding vigorous exercise may also help to prevent paralytic attacks.

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