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Case Report

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AN UNUSUAL PRESENTATION OF HYPOKALEMIC PARALYSIS IN FACIOMAXILLARY SURGERY

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ABSTRACT

Hypokalaemic periodic paralysis is a condition that causes episodes of extreme muscle weakness typically beginning in childhood or adolescence. Most often, these episodes involve a temporary inability to move muscles in the arms and legs. Attacks cause severe weakness or paralysis that usually lasts from hours to days. Some people may have episodes almost every day, while others experience them weekly, monthly, or only rarely. Attacks can occur without warning or can be triggered by factors such as rest after exercise, a viral illness, or certain medications. Hypokalaemia is a common electrolyte imbalance in surgical speciality which may be the cause of sudden onset of weakness. A case of a 20 year old female is presented here. The patient presented with sudden onset paralysis of her left side of face. Laboratory evaluation revealed a markedly low potassium level. The patient's paralysis resolved upon repletion of his low potassium and he was discharged with no neurologic deficits. Although rare, hypokalaemic Paralysis must differentiated from other causes of weakness and paralysis like facial nerve paresis or palsy so that the proper treatment can be initiated quickly.

KEYWORDS: Hypokalaemia, Paralysis, Electrolyte imbalance, Potassium level.

INTRODUCTION

Hypokalemic periodic paralysis (hypoKPP) is a rare, autosomal dominant channelopathy characterized by muscle weakness or paralysis with a matching fall in potassium levels in the blood (primarily due to defect in a voltage-gated calcium channel). In individuals with this mutation, attacks often begin in adolescence and most commonly occur on awakening or after sleep or rest following strenuous exercise (attacks during exercise are rare), high carbohydrate meals, meals with high sodium content, sudden changes in temperature, and even excitement, noise, flashing lights and induced by cold temperatures. Weakness may be mild and limited to certain muscle groups, or more severe full-body paralysis. During an attack reflexes may be decreased or absent. Attacks may last for a few hours or persist for several days. Recovery is usually sudden when it occurs, due to release of potassium from swollen muscles as they recover. Some patients may fall into an abortive attack or develop chronic muscle weakness later in life. [1]

A case of a 20 year old female is presented here. The patient presented with sudden onset paralysis of her left side of face. Laboratory evaluation revealed a markedly low potassium level. The patient's paralysis resolved upon repletion of his low potassium and he was discharged with no neurologic deficits.

CASE REPORT

A 20 year-old female with no significant past medical history presented to the emergency room with sudden onset of facial paralysis. The patient had first presented to primary health care centre and was referred to tertiary care hospital. Patient was unable to open her mouth and there was deviation of lower jaw to left side .She had no respiratory or swallowing difficulty and was able to move her neck and arms with mild pain. Prior to this episode, the patient had been healthy and denied any recent diarrhoea, chest pain, shortness of breath, or weight change. She did not take any medications and denied use of alcohol or drugs, or significant changes in diet or activity levels.

On clinical examination, the patient was haemodynamically stable with normal in overall appearance. There was difficulty in opening the mouth and tresmus was positive. Neurologic examination was unremarkable except slight weakness in left arm with intact sensations and muscle power of grade 4. Sensation and muscle power in right arm was normal. Cranial nerve function was found intact.

Routine chemistry, liver enzymes and complete blood count were normal except for a potassium level of 2.1 (3.5–5 mmol/L). A consulation was made to department of neurology and internal medicine and was consistent

with above mention findings. CT scan head and neck with facial bone was performed to rule out any lesion in brain and tempromandibular joint dislocation which was unremarkable.

Five hours after initiation of intravenous potassium replacement, the patient's neurologic symptoms had completely resolved. Follow up laboratory test were performed to determine the etiology of the patient's hypokalemia and were negative. The patient was diagnosed with Hypokalemic Paralysis. She was discharged home with an appointment to have MRI to rule out any organic cause or aneurysm. Later MRI was performed which was also negative for any findings.

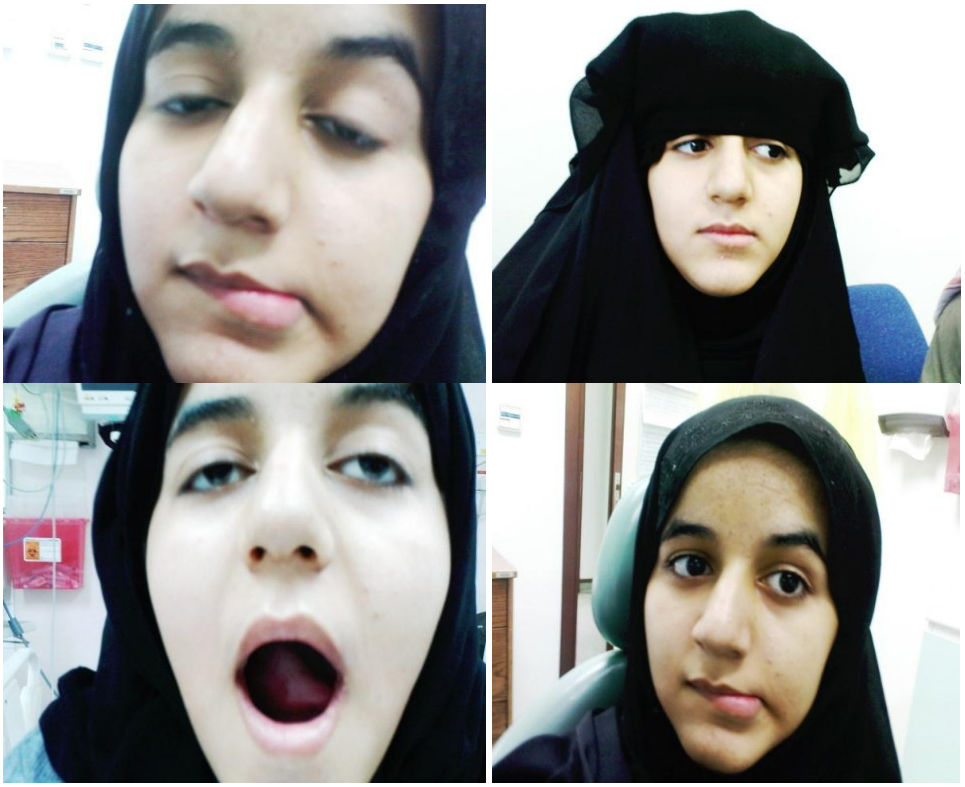

Figure: 1 Case report with patient suffering with hypokalaemic paralysis of lower jaw.

DISCUSSION

Facial weakness is not uncommon, presentation in both the emergency and outpatient clinics. Although the differential diagnosis is extensive, bells, palsy-strokes and tumours are important and common causes and these must be ruled-out in a patient presenting with facial weakness. Facial weakness due to Hypokalaemia is relatively rare but important cause and should be kept in mind.[2]

Hypokalaemic periodic paralysis is a clinical identity with a prevalence of 1 in 100,000.the clinical features of this syndrome vary from mild weakness to life threatening respiratory paralysis. Attacks may be provoked by stress such as a viral illness or stress, or certain medications such as beta-agonists, insulin or steroids. A perturbation of sodium and calcium ion channels results in low potassium levels and muscle dysfunction.[2] As this is primarily a problem with muscle contraction rather than nerve conduction, tendon reflexes may be decreased or absent but sensation is generally intact. Although the serum potassium level is often alarmingly low, other electrolytes are usually normal. Electrocardiographic changes are common particularly of severe hypokalaemia <2.0 meq. Electromyography reveals abnormalities in some patients but is often normal, especially between episodes when no clinically detectable weakness is present.

There is a familial **hypokalaemic paralysis syndrome**, a autosomal dominant condition caused by mutations *CACNA1S* and *SCN4A* genes more common in children.[3,4]

Thyrotoxic Periodic Paralysis (TPP) occurs in the setting of hyperthyroidism particularly grave's disease. It is the most common form of HPP and is seen primarily in Asian males occurring in 1.9% of Japanese hyperthyroid patients overall and up to 8% of hyperthyroid Japanese men.[5]

Rarely, HPP can result from substantial gastrointestinal or renal potassium losses. In these cases, total body potassium is depleted and requires aggressive replacement. Endocrine abnormalities such as hyperinsulinemia and primary hyperaldosteronism have been associated with HPP.[11] Surgical removal of the aldosterone producing tumor is the preferred treatment although symptoms can often be managed with spironolactone.

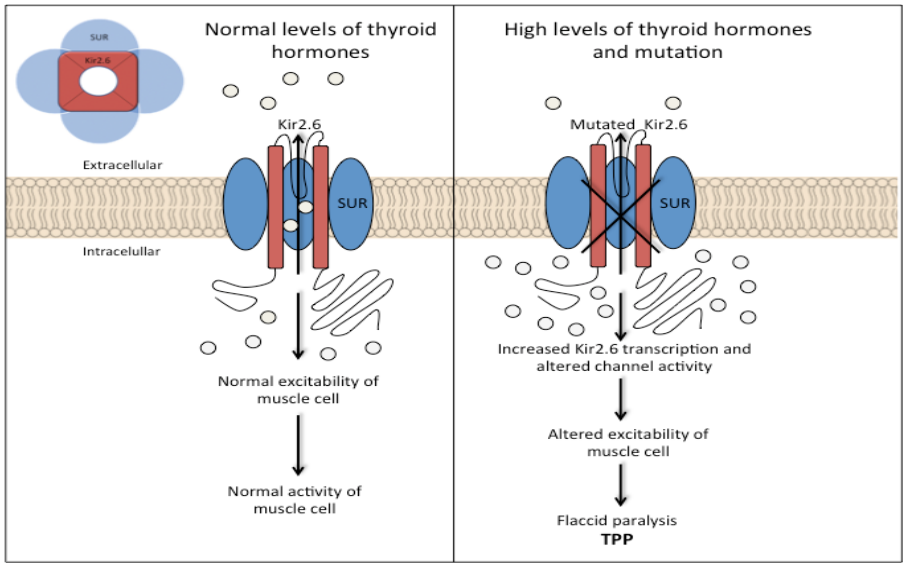

Figure: 2 Physiological regulation of Kir2.6 by thyroid hormone and when mutated.

Anderson Tawil syndrome is a rare, autosomal dominant disorder that is caused by mutation of the *KCNJ2* gene in 60% of cases.[12] Mutation of this gene alters the structure and function of potassium channels disrupting the flow of potassium ions in muscle cells leading to Periodic Paralysis and long QT syndrome.

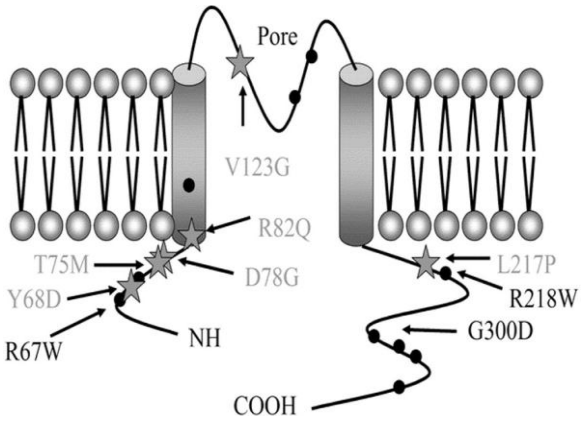

Figure 3: The mutations in KCNJ2 gene in Anderson Tawil Syndrome.

CONCLUSION

This patient presented with sudden onset paralysis of facial paralysis and markedly low potassium. This presentation can easily be confused with facial nerve palsy and temporomandibular joint dislocation. The paralysis resolved completely following potassium replacement. At the time of discharge, she had no neurologic findings /deficit.

Hypokalaemic Paralysis is important to consider when 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 Paralysis can be fatal, but prompt 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.

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