Case Report

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A Rare Disorder: Hypokalemia Induced Paralysis

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Abstract

Hypokalemic (Induced) Periodic Paralysis is a rare group of disorders that can cause sudden onset of flaccid paralysis. Here we present a case of 41 year old female who presented to the ED complaining of severe flaccid paralysis in both lower & upper limbs since morning. Initially, she was treated as a stroke alert patient and had head scans which showed no acute pathologic changes. Laboratory evaluation revealed a markedly low potassium level. The patient's paralysis resolved after potassium correction and she was discharged with no neurologic deficits. Although rare, Periodic Paralysis must be differentiated from other causes of weakness and paralysis so that the proper treatment can be initiated quickly.

Keywords: Periodic paralysis, Hypokalemia, Hypokalemic periodic paralysis, Flaccid paralysis, Stroke, Muscle weakness.

Introduction

Sudden onset of muscle weakness is an alarming symptom in the emergency department (ED), particularly when it acutely presents in a young, healthy patient without predisposing factors for stroke, such as diabetes or hypertension.

Periodic paralysis is a rare condition that affects the muscle ion channels and may be genetic or acquired.[1-2] It can also be associated with one of four different diseases (hypokalemic/hyperkalemic periodic paralysis, thyrotoxic periodic paralysis and Anderson syndrome).[2-4] Potassium plays an important role in the physiologic functions of different tissues and membranes in the body like the heart, skeletal muscles, and nervous system. The clinical presentation in patients with hypokalemia ranges from mild fatigue to severe muscle weakness with necrosis and cardiac arrhythmias. The effect of hypokalemia symptoms depends on the severity of the change in the potassium level.[5]

Case Report

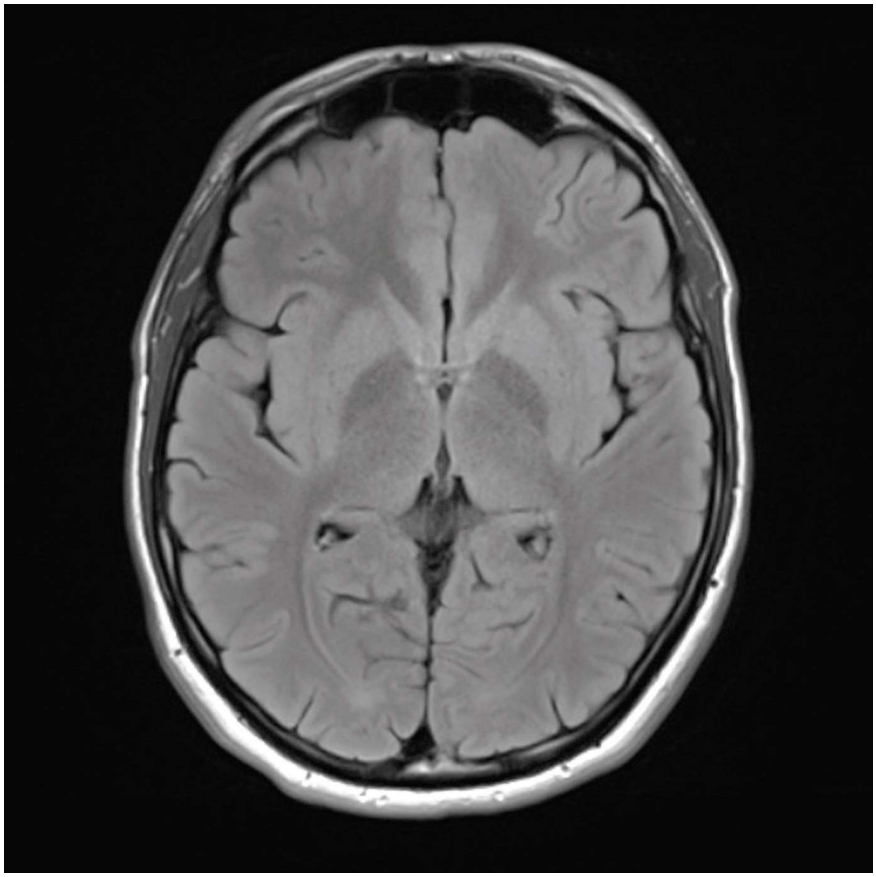
A 41-year-old female patient presented to the ED with complaints of sudden onset of weakness of all 4 limbs since morning, as per the patient, weakness started in lower limbs followed by upper limbs weakness (ascending paralysis). There is no pre-existing comorbidities.

In the emergency room (ER), she was awake, alert, and oriented and was in emotional distress due to her paralysis. Upon taking further history, the patient denied any history of recent fevers, vomiting, diarrhoea, syncope, chest pain, decreased urine output, shortness of breath, loss of consciousness, or bowel or bladder incontinence. Saddle area sensation was intact. The patient has no visual or hearing impairment.

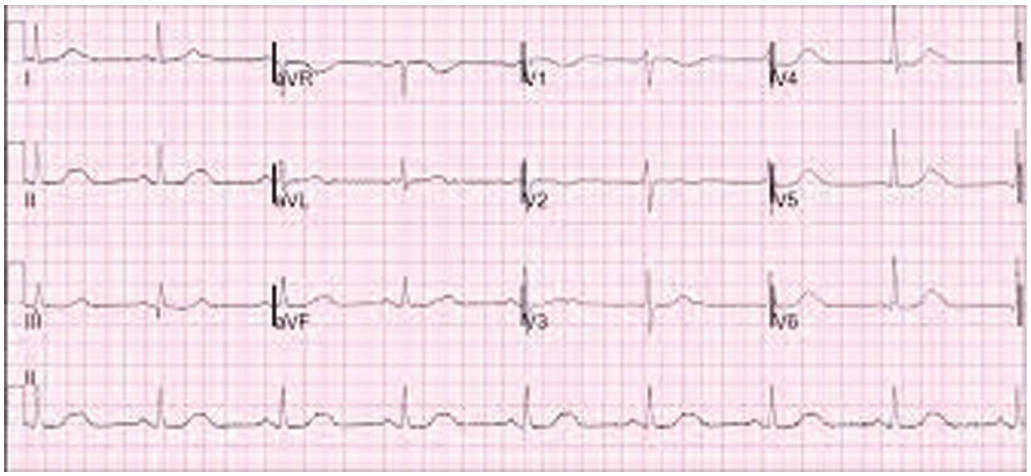
Physical examination revealed normal sets of initial vitals. Flaccid paralysis was noted in the bilateral lower limbs and upper limbs; Power in B/L LL:0/5, UL:1/5, B/L Planters were mute. The sensation was intact all over. The patient had no facial weakness or numbness and had normal extraocular muscle movements. Deep tendon reflexes were absent in all limbs. No focal spinal tenderness was identified. Other systems were within normal limits.

Initial labs showed low serum potassium at 1.3 mmol/L (normal range: 3.5 - 5 mmol/L), minimally elevated creatine phosphokinase level at 300 IU/L (normal range: 55 - 170 IU/L), and a low normal serum magnesium level at 1.6mg/dl (normal range: 1.8 - 2.4 mg/dl). Thyroid function tests were normal. Urine sodium and potassium, serum aldosterone and renin levels were measured to rule out adrenal involvement and were found to be normal. Electrocardiogram (EKG) showed sinus bradycardia with a heart rate at 54 beats per minute, a long QTc interval at 550 ms (Figure 2). MRI Brain showed no evidence of acute intracranial haemorrhage, mass, or acute infarction (Figure 1).

In the ED, the patient was started on intravenous (IV) potassium chloride (KCl) with oral Potassium supplement as well and then admitted to the intensive care unit (ICU). In the ICU, Potassium supplements were continued, in addition to IV magnesium sulphate ($MgSO_4$) was started. In the next 48 hours, the potassium level reached the



MRI Brain: No abnormality detected



EKG: sinus bradycardia with long qtc

normal level and the Potassium supplementation was stopped. The patient regained her muscle power completely and was able to walk without any assistance. Repeat EKG showed QTc interval was back to baseline. The patient then started on physical therapy and was discharged with an appointment to follow up with nephrology & neurology as an outpatient.

Discussion

There are different types of Periodic Paralysis associated with electrolyte and metabolic abnormalities. Of these, Hypokalaemia Periodic Paralysis (HPP) is the most common with a prevalence of 1 in 100,000.[1] Although the pathogenesis of Hypokalemic paralysis remains unclear, alterations in potassium regulation have been well documented. Hypokalaemic paralysis results from either alteration in transcellular distribution of potassium into muscle cells which causes the muscles to become electrically inexcitable or actual potassium depletion from renal or extrarenal losses or any abnormality in muscle membrane. Recent electrophysiologic studies have suggested that the fundamental defect in hypokalaemic periodic paralysis is possibly a calcium channel problem. Genetic linkage data have suggested that the defect in hypokalaemic periodic paralysis may be within a dihydropteridine-binding, voltage-sensitive, skeletal muscle calcium channel.[4]

The clinical features of the syndrome vary, depending on the underlying aetiology but the most striking feature is the sudden onset of weakness ranging from mild, transient weakness to severe disability resulting in respiratory failure. It has an autosomal dominant genetic background, but our patient denied that any of his family members had the same symptoms. In comparison to thyrotoxic periodic paralysis, she did not show hyperthyroidism symptoms and her thyroid function tests were normal. Furthermore, compared to Anderson syndrome, an autosomal dominant disease that has classic dysmorphic features, our patient did not carry those type of features and none of her family members had genetic or dysmorphic diseases. A 24-hour urinary potassium levels is required for a definitive diagnosis, but it is challenging in ER (acute care settings). In this situation, acute treatment and potassium replacement were preferred over urine collection for an accurate diagnosis. Also, urine collection will likely give false results, due to prompt potassium replacement by the ED physicians.

Conclusion

Periodic Paralysis is important to consider when a patient presented with sudden onset weakness or paralysis, especially those with no history or evidence of other comorbidities and no significant risk factors for stroke. Failure to diagnose properly and inappropriate treatment of Periodic Paralysis can be fatal, but rapid correction of abnormal potassium can resolve the symptoms quickly and completely. When possible, the underlying cause must be seen properly to prevent the persistence or recurrence of paralysis.

Conflict of Interest: None declared

The causes of acute weakness are listed below:

Neurological	Inflammatory	Infection	Metabolic
CVA	Poliomyositis	Polio	Alcohol
Post seizure paralysis	Dermatomyositis	Diphtheria	Opiated
Cataplexy	–	Botulism	Electrolyte imbalance
Myasthenia gravis	–	–	–
Multiple sclerosis	–	–	–

Causes of Hypokalemia.

Potassium depletion – renal	Potassium depletion – extra renal	Potassium shift into the cells
Increased aldosterone	Decreased intake	Increased insulin
Diuretics	Vomiting/diarrhea	Alkalosis
Hypomagnesemia	Zollinger-Ellison syndrome	Thyrotoxic periodic paralysis
(RTA (type I and II	Fistulas	Familial hypokalemic paralysis
Metabolic alkalosis	–	–

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