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Case Report

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Beyond the Usual: A Case Series on Acute Hypokalemic Paralysis with a Rare Neurological Twist

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ABSTRACT

Hypokalemic paralysis is characterised by acute muscular weakness due to low levels of potassium in the blood. We report three cases (ages 23, 27, and 25) presented with acute weakness in upper and lower limbs, leading to quadriparesis with no sensory, cranial nerves, bowel, bladder, or cerebellar deficits. On examination, all three patients were conscious, alert, and vitally stable, Neurological examinations revealed flaccid paralysis with exaggerated reflexes and extensor plantars. The rest of the examination was normal. Blood potassium levels were critically low (1.6mEq/L, 2.1mEq/L, 1.3mEq/L), and ECG changes suggestive of hypokalemia. All three showed prompt response to Potassium replacement with complete recovery.

Keywords: Hypokalemic periodic paralysis, potassium and deep tendon reflexes

INTRODUCTION

Hypokalemic periodic paralysis also known as familial hypokalemic periodic paralysis is a very rare genetic disorder characterised by transient, recurrent episodes of acute flaccid paralysis associated with low levels of potassium in the blood (1). These episodes can vary from few hours to many days or weeks and are usually triggered by various factors like high carbohydrate meals, physical exertion or strenuous exercise, emotional stress, pregnancy, alcohol consumption, anaesthesia, surgery or certain drugs including Diuretics, Insulin, Methylxanthines, Amphotericin-B or Steroids (2, 3). Hypokalemic periodic paralysis is inherited in an autosomal dominant fashion with mutations in the CACNA1S or SCN4A genes that are associated with muscle function. Diagnosis is made based on clinical signs and symptoms, family history, and genetic evaluation. Management involves immediate reversal of muscle paralysis by correcting hypokalemia using potassium supplements and prevention of future attacks by avoiding triggers, and making dietary changes. In this report, we present a series detailing three cases of hypokalemic paralysis who presented with atypical signs.

CASE REPORT

Case 1: A 23-year-old male presented to a medical emergency department with sudden onset weakness in both hands and legs on waking up in the morning around 7 am. He denied any recent

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illness, exercise, significant changes in diet, or any previous history of similar illness. On examination, the patient was conscious and alert. His vital signs were stable, and cardiovascular examination was unremarkable. Neurological examination revealed flaccid paralysis of both lower and upper limbs with exaggerated deep tendon reflexes. Sensory examination and cranial nerves were intact. There were no signs of respiratory distress or bulbar involvement. No feature of any psychiatric illness or anxiety was noted. Laboratory investigations revealed severe hypokalemia with a serum potassium level of 1.6 mEq/L (normal range: 3.5-5.0 mEq/L). Electrocardiogram (Figure 1) showed prolonged PR interval, ST segment depression and U waves, consistent with hypokalemia. Thyroid function tests, Renal function tests, serum magnesium levels, vitamin B12/folic acid levels, basal cortisol, urinary potassium and MRI brain and spine were normal.

Case 2: A 27-year-old male presented with medical emergency with complaint of rapidly progressive weakness of bilateral upper and lower limbs evolving over 2 hours. He felt as if his limbs were heavy. There was no history of high carbohydrate diet intake or exercise, no recent illness history, no history of similar illness in past and no family history. On examination, he was conscious and well oriented to time place and person. His vitals were stable, and cardiovascular examination was unremarkable. Neurological examination revealed quadriparesis with power of 2/5 in lower limbs and 3/5 in the upper limbs and symmetrical hyperactive reflexes and extensor plantars. There was no spinal tenderness or deformity of spine. There were no signs of respiratory distress or bulbar involvement. Sensory examination and cranial nerves were intact. No feature of any psychiatry illness or anxiety noted. Laboratory investigations revealed severe hypokalemia with a serum potassium level of 2.1 mEq/L (normal range: 3.5-5.0 mEq/L). Electrocardiogram (Figure-2) showed flattening of T waves and U waves, consistent with hypokalemia. Thyroid function tests, renal function tests, serum magnesium levels, vitamin b12/folic acid levels, basal cortisol and MRI brain and spine were normal.

Case 3: A 25-year female, 16 weeks of POG, presented with complaint of bilateral lower limb cramps followed by symmetrical ascending flaccid quadriparesis. There was no history of high carbohydrate diet intake or exercise, no recent illness history, no history of similar illness in past and family history. During examination the patient was conscious and alert. His vital signs were stable, and cardiovascular examination was unremarkable. Neurological examination revealed flaccid paralysis of both lower and upper limbs with brisk deep tendon reflexes and extensor plantars. There was no spinal tenderness or deformity of spine. Sensory examination and cranial nerves were intact. There were no signs of respiratory distress or bulbar involvement. No feature of any psychiatry illness or anxiety noted. Laboratory investigations revealed severe hypokalemia with a serum potassium level of 1.3 mEq/L (normal range: 3.5-5.0 mEq/L). ABG was suggestive of Normal Anion gap metabolic acidosis (NAGMA), serum magnesium was borderline low, and urinary studies showed features suggestive of Renal Tubular Acidosis. The electrocardiogram showed U waves, consistent with hypokalemia. Thyroid function tests, renal function tests, vitamin B12/folic acid levels, basal cortisol, serum ANA panel and MRI brain and spine were normal.

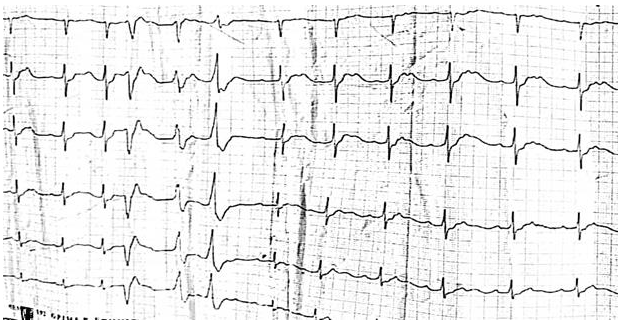

Figure 1: ECG of patient 1 showing- Prolonged PR interval, S-T segment depression and U waves.

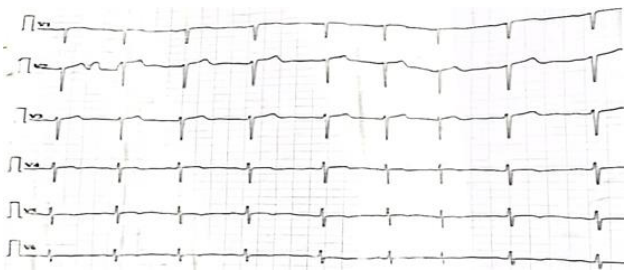

Figure 2: ECG of patient 2 showing- flattening of T waves and U waves.

Table 1: Showing Blood Investigation of case 1, case 2, and case 3.

Parameter	Case 1	Case 2	Case 3	Normal range
Hemoglobin	14.3	13.4	10.5g/dl	13-16g/dl
TLC	7800	6400	9700	4000-11000/cc
Platelet Count	2.82lac	2.12lac	1.16lac	1.5-4lac
ESR	14	9	26	
Total Bilirubin	0.4mg/dl	0.2mg/dl	0.6mg/dl	0.2-1.3mg/dl
Alanine Transaminase	28 U/L	34 U/L	31 U/L	<50 U/L
Alkaline Phosphatase	87 U/L	98 U/L	158 U/L	38-126 U/L
CPK Total	24 ug/L	32 ug/L	42 ug/L	10-120 ug/L
Aspartate Transaminase	34 U/L	41 U/L	38 U/L	17-59 U/L
Blood Urea	22 mg/dl	18 mg/dl	26 mg/dl	15-39 mg/dl
Serum Creatinine	0.6mg/dl	0.8 mg/dl	0.5mg/dl	0.66-1.25 mg/dl
Sodium	145 mmol/L	137mmol/L	138 mmol/L	135-145 mmol/L
Potassium	1.6mmol/L	2.1mmol.L	1.3 mmol/L	3.5-5 mmol/L
Magnesium	2.4	2.6	1.7	1.8-3 mg/dl
Phosphorus	3.2	3.5	3.3	3-4.5mg/dl
S. cortisol	14 ug/dl	16 ug/dl	18 ug/dl	10-20 ug/dl
Serum Iron	45	49	58	
B12 levels	569	465	990	
Folic Acid	13.6	12.8	16.3	
Ferritin	134 ng/ml	144 ng/ml	124ng/ml	6.24-137 ng/ml
Urine potassium	8mmol/L	9mmol/L	38mmol/L	Less than 10mmol/L
TSH	3.21mIU/L	3.67	3.12	0.465-4.68 mIU/L
fT3	7.89pmol/L	5.13	6.60	4.25-8.21 pmol/L
fT4	16.7pmol/L	11.8	14.5	10-28.2 pmol/L

Management and Outcome: All three patients were initially managed with intravenous potassium chloride supplementation under cardiac monitoring to prevent arrhythmias. Potassium replacement was administered under standard correction rate to avoid rapid correction and subsequent rebound hyperkalemia. Serial monitoring of blood potassium levels, ECG, and frequent clinical status was checked. Following potassium replacement therapy, the patients showed gradual improvement in muscle power. Deep tendon reflexes returned and were noted to be normal, likely reflecting transient hyperexcitability associated with severe hypokalemia. All three patients were discharged and advised for precautionary measure, dietary modifications, and regular follow up to prevent recurrence in future.

DISCUSSION

Periodic paralysis refers to a group of neuromuscular disorders marked by sudden episodes of muscle weakness or paralysis, with sensory functions remaining unaffected. These conditions can be categorised as either familial (primary periodic paralysis) or acquired (secondary periodic paralysis) (4). Primary periodic paralysis is a type of channelopathy, resulting from the dysfunction of sodium, potassium, calcium, or chloride channels in the muscle cell membrane (5).

Hypokalemic periodic paralysis (HypoPP) is a type of familial periodic paralysis that demands prompt recognition and intervention due to its potential for causing severe complications, including respiratory compromise and cardiac arrhythmias. The condition is characterised by episodes of muscle weakness or paralysis, typically associated

with low levels of potassium in the blood. HypoPP is most commonly inherited in an autosomal dominant manner and is associated with mutations in the CACNA1S or SCN4A genes. These genes encode ion channels crucial for the proper functioning of muscle cells. Mutations in these channels lead to an impaired ability of muscle cells to maintain normal electrical gradients, resulting in the recurrent and transient episodes of muscle weakness or paralysis. Hypokalemic periodic paralysis may also be caused by other factor like thyrotoxicosis, potassium loss due to gastrointestinal and renal disease (6).

When diagnosing acute onset quadriparesis, it is essential to consider and exclude conditions such as Guillain-Barré syndrome, flaccid paralysis due to myelitis, and para-infectious causes. Periodic paralysis is frequently mistaken for Guillain-Barré syndrome because of their similar clinical presentations. However, the absence of a preceding febrile illness, the non-progressive nature of the paralysis, and normal cerebrospinal fluid (CSF) findings can help differentiate periodic paralysis from Guillain-Barré syndrome (7).

Classically, patients with HypoPP present with flaccid paralysis and reduced deep tendon reflexes during an attack. Episodes can be triggered by factors such as high carbohydrate meals, rest after exercise, stress, or certain medications. The duration of these episodes can vary from a few hours to several days. The frequency and severity of attacks may also vary widely among patient. While flaccid paralysis and reduced reflexes are typical, there are reports, including our own case, of paradoxical presentations such as brisk reflexes during severe hypokalemia. A similar study was presented by Dr PK Pal et al where a patient had atypical neurological presentation like brisk deep tendon reflexes or asymmetrical presentation etc. like in our case 8. This paradoxical response indicates a complex neuromuscular reaction to potassium imbalance that is not yet fully understood. Such atypical presentations are important to document as they can lead to misdiagnosis or delays in appropriate treatment. Hypokalemia paralysis can be precipitated in warmth weather with underlying renal tubular acidosis also shown in our one female case (9).

The diagnosis of HypoPP is primarily clinical, supported by family history and genetic testing. During an attack, serum potassium levels are typically low, and an electrocardiogram (ECG) may show changes indicative of hypokalemia, such as flattened T waves and U waves. Genetic testing can confirm mutations in the CACNA1S or SCN4A genes, providing a definitive diagnosis. Although few other studies also shows that CACNA1S or SCN4A genes are very rare in some patient which indicates that other genetic mutations in them are responsible.10 Management of HypoPP involves both acute treatment of attacks and preventive measures to reduce their frequency and severity. Acute management includes the administration of potassium supplements to correct hypokalemia and alleviate muscle weakness. Preventive measures include dietary modifications to avoid known triggers, such as high carbohydrate meals, and medications such as carbonic anhydrase inhibitors (e.g., acetazolamide) or potassium-sparing diuretics (e.g., spironolactone). Regular monitoring of serum potassium levels and cardiac function is also essential. The potential for atypical presentations, such as brisk reflexes in severe hypokalemia, underscores the need for clinicians to maintain a high index of suspicion for HypoPP, even in cases that do not fit the classic presentation. Early recognition and intervention are crucial to prevent severe complications. Increased awareness and reporting of atypical cases will contribute to a better understanding of the disorder and its varied manifestations, ultimately improving patient outcomes.

Conclusion

Hypokalemia periodic paralysis is a complex and potentially life-threatening condition that requires timely diagnosis and management. While the classic presentation includes flaccid paralysis and reduced deep tendon reflexes, atypical presentations such as brisk reflexes highlight the intricate neuromuscular response to potassium imbalance. Clinicians must be vigilant in recognising and managing this disorder, and ongoing research and case reporting are essential to enhance our understanding and treatment of HypoPP.

Author declaration

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Conceptualisation: SP; Data Collection: RIB, RR; Editing: AB, VKPP; Getting Approval – Final Manuscript: SK, SP.

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The authors declare that there is no financial or non-financial conflict of interest

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Written informed consent was obtained from the patient before they participated in the case, ensuring their understanding of the purpose, procedures, and potential implications involved.

Statement on data availability:
The data of the patient is available with the first author.

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