Magna Neurologica Vol. 3, No. 2, July 2025

https://journal.uns.ac.id/magna-neurologica
DOI: 10.20961/magnaneurologica.v3i2.1683
e-ISSN 2985-3729 p-ISSN 2963-6027

CASE REPORT **OPEN ACCESS**

A CASE REPORT OF 29-YEARS-OLD MAN WITH HYPOKALEMIC PERIODIC PARALYSIS

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Article History:
Received: July 22, 2024
Accepted: October 12, 2024
Published: July 1, 2025

Cite this as:
Mulyono RD, Hasyim. A Case Report of 29-Years-Old Man with Hypokalemic Periodic Paralysis. Magna Neurologica. 3(2) July 2025: 123-127. 10.20961/magnaneurologica.v3i2.1683

ABSTRACT

Background: Primary hypokalemic periodic paralysis is a rare neuromuscular disorder of episodic muscle weakness. The estimated prevalence is 1:100.000. Hypokalemia is a decrease in serum potassium level <3.5 mmol/L. Hypokalemia can cause periodic paralysis.
Case: A 29-year-old man complained of sudden weakness in four limbs, flaccid, proximal-dominant. Weakness in both legs on the first day, followed by weakness in both hands the next day. The patient ate a lot of rice 3 days before the attack. History of four limbs weakness 5 years ago. Upper limb motor strength was 4/3/3 | 3/3/4, and lower limb was 4/3/2 | 2/3/4. Potassium level was 2.59 mmol/L. Potassium correction therapy was performed with KCl 25 mEq in NaCl 0.9% 500cc at a rate of 20 TPM and KCl tablets 600mg/8 hours. The potassium level after the correction was 4.91 mmol/L. Motor strength returned to normal on the third day.
Discussion: Our patient was diagnosed with hypokalemic periodic paralysis (HypoPP). The patient had moderate hypokalemia and returned to normal with a single correction. The leading cause of hypokalemia was suspected to be the underconsumption of potassium and overconsumption of carbohydrates. The patient's condition improved as potassium levels normalized.
Conclusion: HypoPP is a rare neuromuscular disorder associated with hypokalemia, and the primary therapy is potassium correction. The cause of hypokalemia in this case is due to underconsumption of potassium and overconsumption of carbohydrates, but other causes of hypokalemia still need to be considered.

Keywords: acute paralysis, hypokalemia, periodic paralysis

Introduction

Primary periodic paralysis is a rare neuromuscular disorder characterized by episodic muscle weakness.[1,2,3] Primary periodic paralysis results from changes in serum potassium levels.[1,2] The paralysis is caused by depolarization of the striated muscle sarcolemma, leading to inactivity of sodium channels and reduced excitability of muscle fibers.[1,4,5] Primary periodic paralysis includes hypokalemic (HypoPP), hyperkalemic periodic paralysis (HyperPP), and Andersen-Tawil syndrome.[1] HypoPP is more common than the other types.[1] The prevalence of HypoPP is 1 per 100,000.[1,3] HypoPP is classified as primary (inherited) and secondary (acquired).[2,7] Primary HypoPP is caused by canalopathy due to mutations in electrolyte channels in skeletal muscle, resulting in muscle weakness when serum potassium levels decrease.[2,6,7] Secondary HypoPP is caused by other diseases such as thyrotoxicosis, endocrine disorders, renal disorders, or gastrointestinal disorders [3,4]

Hypokalemia is a decrease in serum potassium levels below 3.5 mmol/L.[4,8] Hypokalemia is classified as mild (3.0-3.4 mmol/L), moderate (2.5-3.0 mmol/L), and severe (less than 2.5 mmol/L).[8] Potassium loss due to gastrointestinal disorders such as vomiting, diarrhea, and food malabsorption is the most common cause of hypokalemia.[8] Hypokalemia can cause life-threatening arrhythmias, limb paralysis, and respiratory muscle paralysis that require immediate treatment.[8,9]

This paper presents a detailed account of a case involving a young male patient who exhibited acute and progressive paralysis affecting all four limbs, accompanied by moderate hypokalemia. Remarkably, the paralysis showed significant improvement following the administration of potassium correction. This observation highlights the critical role of electrolyte balance in the manifestation and resolution of neurological symptoms.

Case Report

A 29-year-old man presented to the emergency department with complaints of weakness in all four limbs, slowness, and dominant weakness in the proximal extremities. The weakness initially began manifesting in both legs on the first day, gradually worsening. This was followed by the onset of weakness in both hands the next day. Throughout this period, the patient reported no complaints of numbness or tingling sensations, suggesting that sensory deficits or neurological issues did not accompany the weakness. The patient had eaten a lot of rice and no vegetables or fruits in the previous 3 days. The patient worked as a construction worker. A history of weakness in all four limbs occurred 5 years ago and improved with potassium correction. There was no history of diarrhea, nausea, vomiting, or trauma. There was no history of drug use. There was no history of other diseases. There was no family history of the disease.

On examination, blood pressure was 136/68 mmHg, pulse 90x/min regular, respiration 18x/min, temperature 36 °C. Motor strength of upper extremities 4/3/3 | 3/3/4 and lower extremities 4/3/2 | 2/3/4. Physiological reflexes were reduced in all four extremities. No sensory abnormalities were noted. No facial nerve abnormalities were noted. The potassium laboratory was 2.59 mmol/L. Electrocardiography was within normal limits. Potassium correction therapy was performed with KCl 25 mEq in NaCl 0.9% 500cc at a rate of 60 cc/hour and KCl tablets 600mg/8 hours. The potassium laboratory result after correction was 4.91 mmol/L, and motor strength improved in all four extremities (Table 1). On the third day of treatment, the patient demonstrated remarkable progress, marked by the restoration of motor strength to normal levels. This significant improvement proved that the patient's condition had stabilized, enabling a safe discharge from the hospital. With this positive development, the patient was considered well enough to return home, where they could gradually resume their daily activities, responsibilities, and overall routine under proper guidance and care.

Table 1. Laboratory results

Laboratory tests	Results	Unit	References value
`Haemoglobin	16.1	g/dL	13.2-17
Leukocytes	9870	/uL	3800-10600
Thrombocyte	304	thousand/uL	150-400
Erythrocyte	6.07	10^6/uL	4.4-5.9
Potassium (K)	2.59	mmol/L	3.6-5.5
Sodium (Na)	143.5	mmol/L	131-145
Cloride (Cl)	107.6	mmol/L	92-108
Blood Glucose	81	mg/dL	70-140
Ureum	44.6	mg/dL	10-50
Creatinine	1.1	mg/dL	0.6-1.4
Thyroid-stimulating hormones (TSHs)	1.210	IU/mL	0.610-5.200
Free Thyroxine (FT4)	1.60	mg/dL	1.10-1.70

Discussion

Hypokalemic periodic paralysis causes periodic paralysis of proximal muscles, lasting hours to days, with hypokalemia and possible myopathy.[1,2] The first paralytic attack often occurs between the ages of 15 and 35.[10] Paralytic attacks may be triggered by a high-carbohydrate diet, alcohol, or strenuous activity.[1,9] In HypoPP, symptoms improve with potassium correction.[11] Starland et al. formulated the diagnostic criteria for HypoPP as follows:

Table 2. Supportive diagnostic criteria for HypoPP[1,12]

Supportive Diagnostic Criteria
1. Two or more attacks of muscle weakness with documented potassium serum <3.5 mEq/L
2. One attack of muscle weakness in the proband and one in a relative, both with potassium serum <3.5 mEq/L.
3. Three of 6 clinical or laboratory features:
a. Onset in the first or second decade
b. Attack duration (muscle weakness involving one or more limbs) > 2 hoursPositive triggers (high carbohydrate-rich meal, rest after exercise, stress)
c. Improvement with potassium intake
d. Positive family history or genetically confirmed skeletal calcium or sodium channel mutation
e. Positive McManis prolonged exercise test
4. Exclusion of other causes of hypokalemia (renal, adrenal, thyroid dysfunction; renal tubular acidosis; diuretic and laxative abuse)
5. Absence of myotonia (clinically or latent detected by needle electromyography (EMG)), except eyelids

Hypokalemic periodic paralysis is associated with genetic abnormalities of the CACNA1S (CaV.1) or SCN4A genes on chromosomes 1q31-32 or 17q23-25 (Table 2).[1,12] Finding abnormalities in these genes and appropriate clinical and laboratory criteria can establish the diagnosis of HypoPP.[1,4] Genetic testing for the diagnosis of HypoPP is considered relatively expensive. Hence, some researchers mention that clinical and laboratory examinations are sufficient for diagnosing HypoPP.[11,13] Additional diagnostic options include provocative testing and electromyography (EMG). Oral glucose load can be performed as a provocation test. It can help in the diagnosis.[14,15] EMG may reveal a reduced amplitude of compound muscle action potential (CMAP) and electrical silence during episodes of muscle weakness, the extent of which depends on the severity of muscle weakness observed during the attack. EMG and exercise testing can be performed to identify abnormalities between attacks. In the exercise test, a focal muscle weakness attack is induced by vigorous exercise of a single muscle for 2 to 5 minutes; EMG is performed to measure pre-exercise and post-exercise CAMP in muscle fibers. This EMG technique can be performed using the abduction range of the little finger. A reduction of 40% or more in CAMP is considered abnormal and typical for periodic paralysis.[15,16]

Hypokalemia can be caused primarily by decreased potassium intake, increased intracellular uptake, and increased potassium losses. Decreased potassium intake is due to consuming foods low in potassium or eating disorders.[8] Increased intracellular uptake is due to hyperthyroidism, use of insulin injections, or beta-adrenergic inhalation.[8,18] Increased potassium loss is due to excessive sweating, diuretic use, renal abnormalities, diarrhea, and vomiting.[2,8] Gastrointestinal disorders are the most common causes of hypokalemia, including vomiting, diarrhea, food malabsorption, inadequate potassium intake, and poor diet.[8] A fall in serum potassium levels triggers an attack of muscle weakness in patients with HypoPP.[19] It is known that during a HypoPP attack, the muscle is depolarized and cannot be excited. The mechanisms are not entirely understood.[20]

The differential diagnosis of primary hypoPP includes hyperkalemic or normokalemic periodic paralysis, thyrotoxic periodic paralysis, Andersen-Tawil syndrome, secondary hypokalemia, myasthenia gravis, and paramyotonia congenita.[6,15] The first episode of quadriparesis can be mistaken for another paralytic attack, such as Guillain-Barré syndrome, acute myelopathy, myasthenia crisis, tick paralysis, and botulism.[1,15]

HypoPP has similarities and differences in clinical features with other differential diagnoses. In hypoPP and other differential diagnoses, periodic paralysis is a standard feature. The main difference between hypoPP and hyperPP or normocalcemic paralysis is the potassium level during the attack. This difference is easily seen by immediately checking potassium levels during the attack.[1,15] Thyrotoxic periodic paralysis has the same characteristics as hypoPP, except that in hypoPP, there is no hyperthyroidism. Unlike hypoPP, treatment of hyperthyroidism is the primary therapy in thyrotoxic periodic paralysis, which is usually followed by improvement of the paralysis as the hyperthyroidism improves.[15,17] Myasthenia is characterized by generalized muscle weakness that worsens with physical activity, accompanied by bulbar paralysis, facial weakness, and ptosis. Muscle weakness is unaffected by potassium levels but worsens after physical activity.[6,15] Muscle weakness in secondary hypokalemia is usually due to chronic hypokalemia. The primary clinical difference between secondary hypokalemic paralysis and HypoPP is the finding of hypokalemia in the absence of muscle weakness between paralysis attacks. Congenital paramyopathy is a congenital, periodic muscle weakness triggered by cold temperatures and strenuous physical activity. Muscle weakness in congenital paramyopathy is first reported in childhood. Congenital paramyopathy may have elevated or normal potassium levels.[6,15] Andersen-Tawil syndrome has a clinical picture similar to hypoPP with decreased or normal potassium levels at onset.[1, 15]

Table 3. Clinical presentation of HypoPP and Andersen-Tawil syndrome[1,15]

Feature	HypoPP	Andersen-Tawil syndrome
Ictal K+ level	Low	Normal/LOr
Age at onset (years old)	5 – 35	2 - 18
The mean duration of episodes	2 hours	1 – 36 hours
Maximum weakness	Severe	Moderate
Characteristic facies	Absent	Present
Arrhythmias	Absent	Present
Gene mutation	CACNA1S and SCN4A	KCNJ2

Management in hypoPP is divided into two types of management. Acute attack management and preventive management. Acute attacks require immediate potassium correction with oral or intravenous potassium supplements. Oral potassium chloride can be given gradually with an initial dose of 0.5-1 mEq/kg; if there is no clinical improvement, it can be repeated with a dose of 0.3 mEq/kg every 30 minutes and a maximum dose of no

more than 200 mEq in 24 hours. Intravenous potassium is usually given to patients with heart rhythm problems, difficulty swallowing, or respiratory muscle paralysis. The dose used is not more than 20 mEq/hour, and the maximum dose is not more than 200 mEq in 24 hours. During potassium administration, it is necessary to monitor ECG and muscle strength up to 24 hours after completion of therapy. Serum potassium levels may be rechecked after 24 hours of therapy (Table 3).[1,15]

Preventive therapies include pharmacologic and nonpharmacologic. Nonpharmacological therapies include educating patients to avoid triggers of hypoPP and modifying dietary patterns to reduce carbohydrate consumption. Pharmacologic therapy includes potassium supplementation and therapy with acetazolamide or dichlorphenamide. 250 mg of acetazolamide taken twice daily effectively reduces the incidence of attacks in HypoPP. Acetazolamide can be given as a single dose or in combination with potassium-sparing diuretics such as spironolactone at 100 mg daily. Dichlorphenamide may be given at a dose of 50mg twice daily. Dichlorphenamide is the drug of choice for patients who do not respond to treatment with acetazolamide.[11,15]

Holm-Yildiz et al. in 2023 reported that HypoPP could lead to permanent muscle weakness and increased fat replacement of muscle. This condition can cause myopathy in patients with hypoPP.[11,21] Regular exercise and medications such as acetazolamide or dichlorphenamide may reduce the frequency of attacks and incidence of myopathy.[11,18] Myopathy and respiratory insufficiency can occur due to paralysis of the respiratory muscles. But this complication is rare in HypoPP.[15]

We found clinical and laboratory conditions consistent with HypoPP in this patient, so we diagnosed the patient with HypoPP. The patient was found to have moderate hypokalemia, and the cause of the hypokalemia could not be determined. Inadequate potassium intake and a high carbohydrate diet were suspected to be the cause of hypokalemia and the trigger of hypoPP. However, further investigation is necessary to identify the underlying cause of hypokalemia in this patient accurately. The patient did not undergo further examination such as EMG, genetic examination, or examination to find other causes of hypokalemia due to inadequate equipment, high cost, and considering that the patient's condition had returned to normal after the potassium was corrected and there were no signs and symptoms of other diseases in the patient.

Conclusion

Hypokalemic periodic paralysis is a rare neuromuscular disorder with an ascending progression and predominance in the proximal muscles. HypoPP is associated with hypokalemia and genetic disorders, and the primary therapy is potassium correction. In this case, the leading cause of hypokalemia is thought to be a lack of potassium intake and excessive carbohydrate consumption, but other causes of hypokalemia should be considered. Patients with hypoPP should avoid hypoglycemia triggers such as excessive carbohydrate consumption, vigorous physical activity, and consumption of foods with low potassium. If paralysis occurs, patients should go to a health facility immediately. Patients should have regular medical check-ups to prevent recurrent paralysis attacks and to prevent complications that may occur. Patients must have health insurance due to the long-term treatment and possible attack periods requiring intensive care.

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