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Original Research Article

Acute-Onset Weakness with Documented Hypokalemia in Adults: Perspectives and the Role of Collaborative Care Team in its Management

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Conflict of interest: Nil

Abstract

Background: Acute-onset weakness due to hypokalemia is a critical clinical condition that can lead to significant morbidity if not promptly diagnosed and managed. The etiology of hypokalemia varies widely, and a structured, multidisciplinary approach is essential for effective management and prevention of recurrence.

Objective: This study aims to analyze the clinical characteristics, etiologies, and outcomes of adult patients presenting with acute hypokalemic weakness, while assessing the impact of a collaborative care team in optimizing management strategies.

Methods: A prospective observational study was conducted on adult patients admitted with acute-onset weakness and documented hypokalemia (<3.5 mmol/L) at a tertiary care hospital over a period of 13 months. Data on demographic characteristics, clinical presentation, biochemical parameters, etiological factors, treatment approaches, and patient outcomes were collected and analysed. The role of a multidisciplinary team—including emergency physicians, intensivists, nephrologists, and physiotherapists—was evaluated in terms of time to diagnosis, treatment efficacy, and patient recovery.

Results: A total of 49 patients were included in the study. The most common etiologies identified were gastrointestinal losses, renal potassium wasting, endocrinopathies and various syndromes. The mean serum potassium level at presentation was below 3.0 mEq/L mmol/L. The primary dangers of HPP arise from complications related to hypokalemia, particularly cardiac arrhythmias and respiratory insufficiency, which contribute to morbidity and mortality. A multidisciplinary approach—involving neurologists, nephrologists, and dietitians is essential for optimizing long-term outcomes, minimizing complications, and ensuring comprehensive care for individuals with hypokalemic periodic paralysis.

Conclusion: Acute hypokalemic weakness in adults requires prompt recognition and a systematic approach to management. A multidisciplinary team-based strategy improves diagnostic accuracy, treatment efficiency, and long-term outcomes. Standardized protocols and increased awareness can further enhance patient care and prevent recurrence.

Keywords: Hypokalemia, Potassium, Collaborative Care Team, Paralysis.

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Introduction

Background

Acute-onset weakness is a distressing clinical presentation with a broad differential diagnosis, among which hypokalemia is a significant yet often underrecognized cause. Hypokalemia, defined as a serum potassium level <3.5 mmol/L, can impair neuromuscular transmission, leading to muscle weakness, paralysis, and, in severe cases, respiratory failure [1]. The prevalence of hypokalemic paralysis varies globally, with a higher incidence reported in

Asian populations, particularly in cases of thyrotoxic periodic paralysis (TPP) [2] .

Hypokalemia can result from multiple mechanisms:

1. Excessive potassium loss: This includes gastrointestinal losses (vomiting, diarrhea, laxative abuse) and renal potassium wasting (diuretics, renal tubular disorders, hyperaldosteronism) [3].
2. Intracellular potassium shift: Seen in insulin overuse, TPP, and metabolic alkalosis, where potassium moves into cells rather than being lost from the body [4].
3. Inadequate dietary intake: Though uncommon, severe malnutrition and prolonged fasting can contribute to hypokalemia, particularly in critically ill patients [5].

Severe hypokalemia (<2.5 mmol/L) has been associated with cardiac arrhythmias, rhabdomyolysis, and respiratory compromise, making early recognition and intervention crucial [6]. Despite its importance, hypokalemic paralysis is often misdiagnosed as Guillain-Barré syndrome, myopathy, or psychiatric illness, leading to inappropriate treatments and delays in management [7] .

Rationale for the Study

While several studies have documented the clinical spectrum of hypokalemic paralysis, there is limited research on the impact of a multidisciplinary care model in its management. Given the variability in presentation, timely electrolyte correction alone may not be sufficient—identifying and treating the underlying cause, preventing recurrence, and ensuring appropriate rehabilitation are equally critical [8]. A collaborative care team, including emergency physicians, intensivists, nephrologists, endocrinologists, and physiotherapists, can provide comprehensive and timely intervention, reducing morbidity and hospital stay duration.

Significance

By understanding the diverse etiological spectrum and optimizing team-based management strategies, this research will contribute to improving patient outcomes. Findings from this study may aid in the development of standardized protocols for early diagnosis, risk stratification, and comprehensive treatment of hypokalemic paralysis. If shown in the study a multidisciplinary approach can help minimize complications, reduce hospital burden, and enhance the quality of care for affected patients. This can help in improved patient outcomes

Objective

This study aims to:

1. Analyze the clinical characteristics of adult patients presenting with acute-onset weakness due to documented hypokalemia.
2. Identify the underlying etiologies contributing to hypokalemic paralysis in a hospital-based setting.
3. To understand Can Collaboration with interprofessional healthcare teams improve the clinical outcome for such patients or can multidisciplinary approach be effective in improving diagnosis, treatment outcomes, and recurrence prevention.

Materials and Methods

This prospective observational study was conducted in a tertiary health care centre over period of 13 months. This study recruited patients who presented to the emergency department with acute-onset paralysis and documented evidence of hypokalemia."

The study included 49 patients. The objectives, protocol, and investigative processes were thoroughly explained to ensure clear understanding and informed participation to both the patients or their legally acceptable representatives. After meeting the eligibility criteria, written informed consent was obtained following a detailed explanation of the study protocol, and the patients were enrolled in the study.

A thorough history was obtained, covering demographic factors such as age, sex, illness duration, current episode of weakness, and other potential precipitating factors like high carbohydrate intake, vigorous exercise, long starvation, severe diarrhea or vomiting, and other medication use. The number of previous episodes, the patient's occupation, and family history were also documented.

Each patient underwent a detailed clinical examination, and other possible causes of muscular weakness were ruled out. Low serum potassium levels were confirmed. All patients had a baseline ECG along with blood gases. A spot urine sample was collected to evaluate urine potassium levels, calculate the urine potassium-to-creatinine (K/C) ratio. Serum bicarbonate levels were measured in patients with normal or low blood pressure, and further investigations were performed based on these findings as necessary.

A subgroup of patients with low serum bicarbonate levels, urine potassium levels greater than 15 mmol/L, hyperchloremic metabolic acidosis with a normal anion gap, and no signs of gastrointestinal losses, along with a fasting urine pH greater than 5.5, underwent additional tests to rule out other causes as renal tubular acidosis.

Inclusion Criteria

1. All Adult patients presenting with paralysis and diagnosed with hypokalemia (i.e., serum potassium levels < 3.5 mEq/L).
2. Patients willing to give informed consent

Exclusion Criteria

1. Patients with weakness but serum potassium levels > 3.5 mmol/L.
2. Patients diagnosed with conditions leading to muscular weakness due to drug induced, neurologic, infectious, or any other metabolic causes.

Statistical Analysis

Data analysis was done by using appropriate statistical methods.

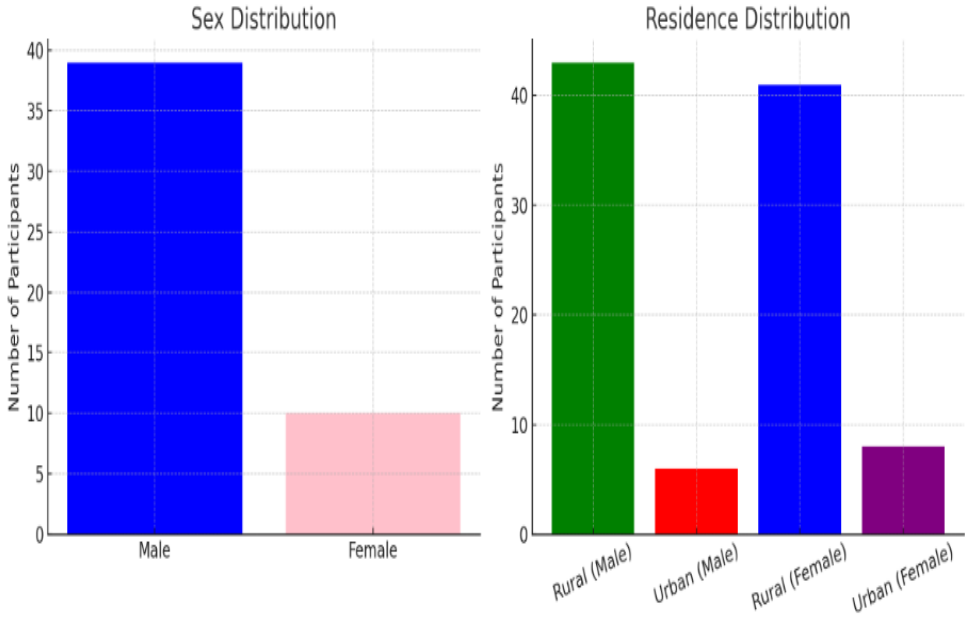
Ethical Clearance:

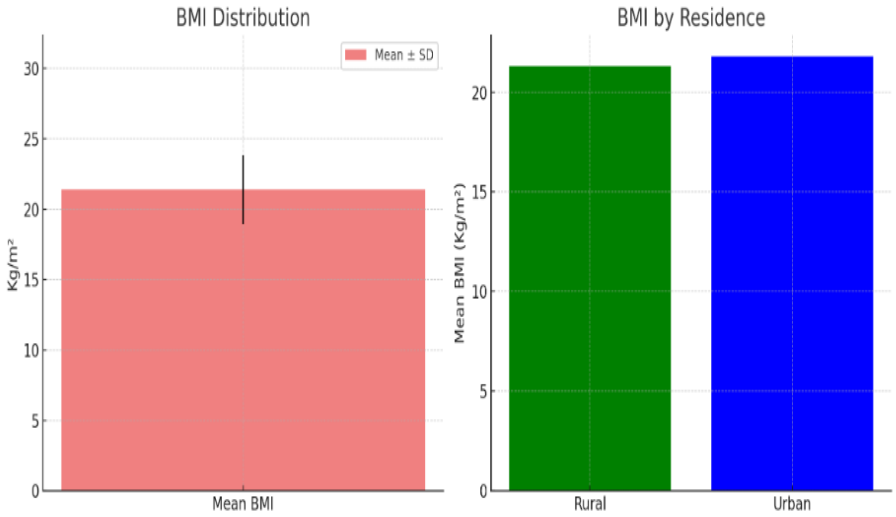
Ethical clearance to proceed was obtained from the IEC of the teaching institute before commencement of the study. Patients who gave valid informed consent were recruited in the study.

Results:

Table 1: Demographic Characteristics of Study Participants

Parameters	Values
Number of Cases	49
Age (years)	
- Range	18 - 62
- Mean ± SD	26.43 ± 7.17
BMI (Kg/m²)	
- Range	15.4 - 27.4
- Mean ± SD	21.41 ± 2.46
Sex (%)	
- Male	39 (79.5%)
- Female	10 (20.5%)
Residence	
- Rural (Males)	43 (89%)
- Urban (Males)	6 (11%)
- Rural (Females)	41 (86.1%)
- Urban (Females)	8 (13.9%)

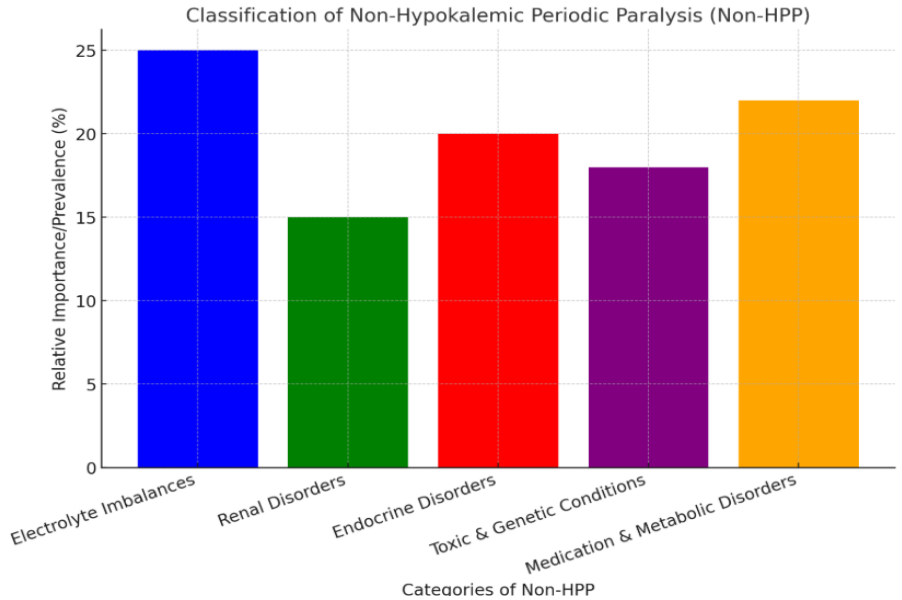




The study population had a mean age of **26.43 years** (range: 18-62 years) and a mean **BMI of 21.41 kg/m²**. Males constituted **79.5%** of the study group, while **20.5% were females**. The majority of participants resided in rural areas (**86-89%**).

NON-HYPOKALEMIC PERIODIC PARALYSIS (NON-HPP) AND ITS VARIOUS OTHER CAUSES [9,10,11,12]

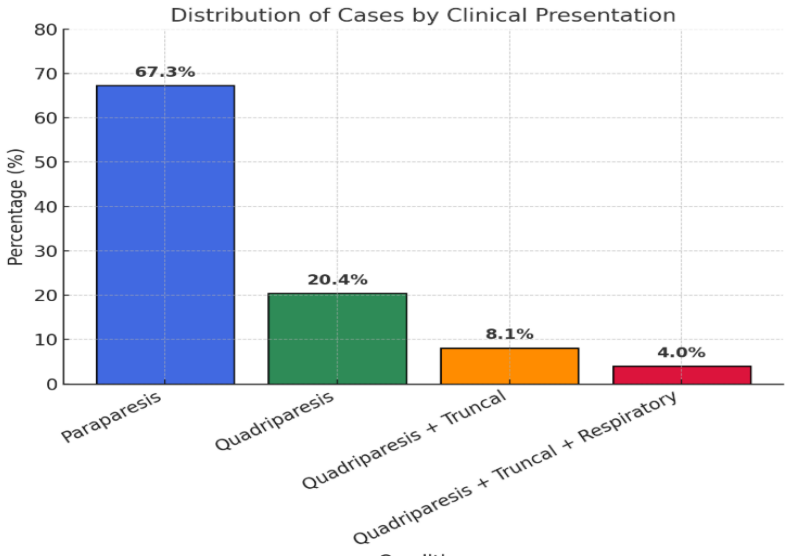
- Diarrhea – Causes excessive potassium and magnesium loss.
- Hypomagnesemia – Can lead to secondary potassium depletion and muscle weakness.
- Distal Renal Tubular Acidosis (dRTA, Type 1) – Leads to metabolic acidosis and potassium wasting.
- Sjogren's Syndrome – Autoimmune disorder causing renal tubular dysfunction and hypokalemia.
- Toluene Exposure – Inhalation of solvents disrupts renal potassium handling.
- Bartter's Syndrome – Genetic kidney disorder causing hypokalemia, metabolic alkalosis, and periodic paralysis.
- Gitelman's Syndrome – Similar to Bartter's but with additional hypomagnesemia and hypocalciuria.
- Hyperthyroidism (Thyrotoxic Periodic Paralysis, TPP) – Excess thyroid hormone increases Na^+/K^+ ATPase activity, causing potassium shifts.
- Cushing's Syndrome – Excess cortisol leads to potassium depletion and weakness.
- Familial Hypokalemic Periodic Paralysis – Genetic channelopathy affecting muscle sodium and calcium channels.



Overview of Muscle Weakness:

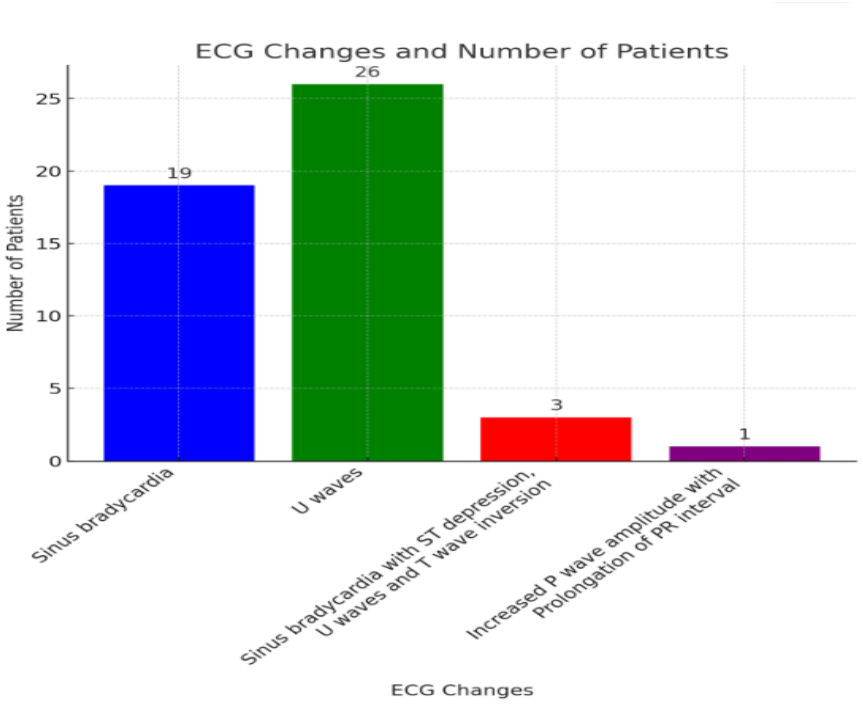
Motor Weakness	Total No of Cases (N=49)	Percentage
Paraparesis	33	67.34
Quadriparesis	10	20.4
Quadriparesis + Truncal involvement	04	08.1
Quadriparesis + Truncal weakness + Respiratory Involvement	02	04.0

Key Findings:



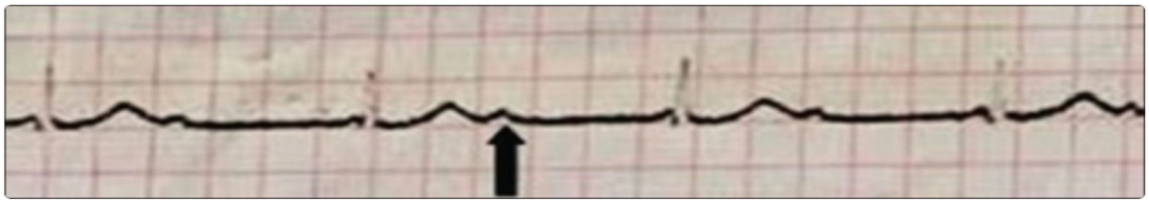
- 67.3% of cases had paraparesis, the most common presentation.
- 20.4% had quadriparesis, while 8.1% exhibited quadriparesis with truncal involvement.
- The most severe form, quadriparesis with truncal and respiratory involvement, was observed in 4.0% of cases.

ECG changes



ECG Changes	No of Patients
Sinus bradycardia	19
U waves	26
Sinus bradycardia with ST depression, U waves and T wave inversion	3
Increased P wave amplitude with Prolongation of PR interval	1

Key Finding: A **high prevalence of U waves (53%)** and **sinus bradycardia (38.8%)** was noted among participants.



ECG showing bradycardia and U wave (arrow) in a patient with hypothyroidism who had recurrent flaccid quadriplegia with serum potassium of 1.96 mmol/l

Number of Earlier Attacks with Hypokalemic Paralysis

No of attacks	Total No of Cases (N=49)	Percentage
0	31	63.26
1	12	24.49
2	03	06.12
3	01	02.0
4	01	02.0
5	01	02.0

Findings:

- **63.3% of patients had no prior attacks**, while **24.5% reported at least one previous episode**.
- Multiple previous attacks (≥3) were rare (**6.1% of cases**).

Comparison Of Changes In Mean Potassium Required Between The Groups

Groups	Mean Potassium (Mean± SD)
HPP (N = 43)	*2.16± 0.43
Non HPP(n=6)	1.43±0.55

By Student 't' Test *P < 0.05 Significant

Mean potassium was 2.16 meql/l among HPP group which was significantly more as compared to 1.43meql/L in non HPP Group

Potassium levels were significantly lower (p<0.05) in the Non-HPP group.

Comparison of Changes in Mean Serum PH Between the Groups

Groups	Mean Serum pH
SPP (N = 41)	7.18 plus/minus 0.07
Non SPP (N = 8)	6.91 plus/minus 0.2

By Student t' Test * P < 0.05 Significant

Mean serum pH was 7.18 among HPP group which was more as compared to 6.91 in Non

HPP group and difference was statistically significant.

Comparison of Changes in Mean Urine Potassium Between the Groups

Groups	Mean Urine Potassium (Mean ± SD)
HPP (N=41)	13.19 ±6.48
Non HPP (N = 8)	31.12 ±23.95

By Student't' Test * P < 0.05 Significant

Mean Urine potassium was 13.19 mmol/L among HPP group which was significantly low as compared to 31.12 mmol/L in Non HPP group.

Comparison of Changes in Mean Chloride Between the Groups

Groups	Mean Chloride (Mean ± SD)
HPP (N = 40)	*99.86±04.99
Non HPP (N = 9)	114.21 +08.65

By Student't' Test *P < 0.05 Significant

This analysis states that mean chloride was 99.86 meql/L among HPP group which was significantly less as compared to 112.11 meql/L in Non HPP group.

Table 16: Comparison of Changes in Mean Urine Potassium Between he Groups

Groups	Mean Urine Potassium (Mean plus/minus S * D)(mmol/L)
HPP (N = 40)	* 13.68 plus/minus 5.99
Non HPP (N = 9)	30.83 plus/minus 24.14

By Student't' Test * P < 0.05 Significant

Mean Urine potassium was 13.68 mmol/L among HPP group which was significantly low as compared to 30.83 mmol/L in Non HPP group.

Comparison of Mean Potassium Required for Treatment Between the Two Groups

Groups	Mean Potassium Required (Mean plus/minus S * D)(meql/L)
HPP (N = 40)	* 57.74 ± 19.32
Non HPP (N = 9)	94 ± 47.38

Student 't' Test *P < 0.05 Significant

Above table shows that mean potassium required was 57.74 meql/L among HPP group which was significantly low as compared to 95.00meql/L in Non HPP group.

Comparison of Changes in Mean Potassium Required with Motor Weakness

Motor Weakness	Mean Potassium Required (Mean plus/minus S * D) (meql/L)
Paraparesis (N = 33) (P)	57.34 ± 19.21
Quadriparesis (N = 10) (Q)	63.8 ± 32.76
Quadriparesis + truncal Involvement (N = 4) (Q + T)	76.5 ± 31.74
Quadriparesis + Truncal +respiratory involvement (N = 2) (Q + T + R)	135 ± 1.2

Comparison of Changes in Mean Recovery Between the Groups

Groups	Mean Recovery (Mean ± SD)(in hours)
HPP (N = 40)	*24.77 +27.56
Non HPP (N = 9)	76.29 +30.73

Student 't' Test *P <0. Student

In this study group mean recovery was 24.77 among HPP group which was less as compared to 76.29 in Non HPP group and difference was statistically significant.

Discussion

This prospective study evaluated patients with hypokalemia presenting with weakness, excluding individuals with alternative causes to specifically focus on manifestations attributable to hypokalemia.

Mild hypokalemia, defined as serum potassium levels between 3 and 3.5 mEq/L, is often asymptomatic. However, as potassium levels decrease further, nonspecific symptoms such as generalized weakness, fatigue, and constipation become more prevalent. In severe cases, hypokalemia can lead to critical complications:

- Serum potassium <2.5 mEq/L: Muscle necrosis may develop.
- Serum potassium <2.0 mEq/L: Ascending paralysis can occur, potentially leading to respiratory failure.

The six patients classified as non-HPP had the following diagnoses:

- Distal Renal Tubular Acidosis
- Diarrhea with hypokalemia
- Toluene exposure with secondary distal renal tubular acidosis
- Sjögren's syndrome with distal renal tubular acidosis
- Bartter's syndrome
- Hypomagnesemia

In this study, the most common cause of Hypokalemic Periodic Paralysis (HPP) was sporadic, with no identifiable cause for hypokalemia in these cases, consistent with previous studies that have identified familial or sporadic causes as the most prevalent [13,14].

Among our patients, 41 experienced warning symptoms such as sensations of heaviness in the legs or back pain. Other reported prodromal symptoms included excessive hunger or thirst, dry mouth, palpitations, sweating, diarrhea, nervousness, and fatigue. The majority of patients in the HPP group exhibited these warning symptoms, which were also noted in a similar study by Lin Sh et al. [15] . The mean duration of these symptoms was approximately 13 hours before clinical weakness onset.

Most patients (n=31) presented with attacks early in the morning. Weakness typically began in the lower limbs and spread to the upper limbs over a mean duration of 13 hours. Notably, none of these patients had a family history of similar illnesses.

ECG findings in our study revealed:

- 19 patients had sinus bradycardia.
- 26 exhibited U waves.
- 3 showed a combination of sinus bradycardia, ST depression, U waves, and T wave inversion.
- 1 patient had increased P wave amplitude with PR interval prolongation.

Despite serum potassium levels dropping to approximately 2 mmol/L, no significant cardiac arrhythmias were observed. Not many studies have correlated the degree of hypokalemia with ECG changes; however, a case report by Sagray E et al. documented cardiac arrhythmias in primary hypokalemic periodic paralysis. Additionally, ECG findings have not been widely used to diagnose hypokalemia before laboratory results become available. In this study, no correlation was established between ECG changes and the degree of weakness present [16] . The most common ECG finding was the presence of U waves. Some studies suggest that ECG findings may not correlate with serum potassium levels, and clinicians should not overly rely on ECG for diagnosing hypokalemia; rather, ECG should be used to identify cardiac rhythm abnormalities and aid in prognostication.

Among all patients, 16 experienced repeated attacks:

- 8 patients had two repeated attacks.
- 4 had three attacks.
- 2 had four attacks.
- 1 patient had five attacks.

We identified 36 cases of primary hypokalemic periodic paralysis with no apparent cause for hypokalemia despite extensive investigations. These patients fully recovered without neurological deficits upon potassium replacement.

Two patients with HPP had secondary hypokalemia due to diarrhea, a well-documented cause of significant potassium depletion, which can lead to quadriparesis. Additionally, two patients were diagnosed with distal renal tubular acidosis (dRTA), presenting with hypokalemia, hyperchloremic metabolic acidosis with a normal anion gap, and alkaline urine despite severe metabolic acidosis. Ultrasonography of the abdomen confirmed nephrocalcinosis.

Two patients had dRTA secondary to toluene exposure, commonly associated with paint thinner ingestion [17] . These patients presented with quadriparesis and respiratory failure. Lab findings showed severe hyperchloremia, hypokalemia, and metabolic acidosis. Further evaluation confirmed a renal potassium-wasting disorder and alkaline urine. Toluene poisoning can lead to a spectrum of electrolyte and acid-base disturbances, gastrointestinal symptoms, and neuropsychiatric manifestations.

All patients underwent thyroid function tests, but none were diagnosed with thyrotoxic periodic paralysis. However, thyrotoxic periodic paralysis is a significant cause of HPP in Southeast Asia, including China. A study involving 34 patients with

hypokalemic paralysis found no prior history of hyperthyroidism or family history of paralysis. All participants demonstrated low potassium excretion rates [18] .

Our study found that severe potassium loss was associated with respiratory muscle involvement. Two patients required ventilatory support due to respiratory failure. Although rare, respiratory muscle weakness correlated with significantly low mean serum potassium levels, a finding not observed in similar studies. Further research is needed on this rare presentation.

Secondary hypokalemic periodic paralysis cases often involve acid-base disturbances. Differentiating these conditions requires assessing hyperchloremia, which helps distinguish non-HPP causes (e.g., gastrointestinal loss, RTA). Urinary potassium excretion further differentiates renal from extra-renal potassium loss. In our study, mean urinary potassium was significantly lower in the HPP group (13.68 mmol/L) compared to the non-HPP group (30.83 mmol/L). Random urinary potassium measurements are simple but may be less accurate than 24-hour collections.

Our study noted that the mean potassium requirement for treatment in HPP patients was approximately 60 mEq/L, compared to 100 mEq/L in non-HPP patients, which was statistically significant. Clinicians should be cautious with potassium supplementation in HPP to prevent rebound hyperkalemia. There was no correlation between the amount of potassium required for treatment and the degree of neurological weakness present.

Attacks in HPP generally resolve within a few hours, though residual paralysis may take days to recover. Recovery time was longer in secondary HPP, possibly due to greater potassium loss or ongoing renal losses, as observed in a study by Maurya.

Six primary HPP patients with recurrent attacks were started on acetazolamide. Griggs et al. [15] found that acetazolamide not only prevented attacks but also improved inter-attack weakness in 8 of 10 patients. Similar findings were noted by Rao et al. and Sushil et al [19] . However, there is no standardized treatment regimen, and the optimal timing for initiating acetazolamide remains unclear. Further research is needed to determine the best treatment for reducing attack frequency, severity, and preventing permanent muscle weakness.

Acetazolamide is thought to induce mild acidosis, preventing attack precipitation. However, some patients worsen on acetazolamide. In such cases, alternatives like triamterene, spironolactone, or dichlorophenamide may be used. The recommended dose of acetazolamide is 250 mg thrice daily, while dichlorophenamide is given at 50-150 mg/day. Acetazolamide should be avoided in patients with RTA, as it may exacerbate periodic paralysis due to its kaliopenic effect, though this remains debated in literature. Spironolactone is an alternative, but its androgenic side effects limit its use.

A multidisciplinary healthcare team, including hospitalists, nurses, dietitians, pharmacists, and geneticists, plays a crucial role in managing HypoPP [20] . A collaborative approach enhances patient outcomes by identifying triggers, managing acute symptoms, preventing complications, and reducing attack frequency [21] . Nurses closely monitor patients to prevent life-threatening complications, dietitians tailor diets to minimize attacks, and pharmacists ensure optimal potassium dosing and medication reconciliation. Couples with a positive family history may benefit from prenatal genetic testing [22] . A comprehensive, cost-effective, multidisciplinary approach involving neurologists, nephrologists, and dietitians is critical for improving long-term outcomes in HypoPP [23] .

Conclusion

- **Clinical Spectrum and Differentiation:** This study highlights the diverse clinical spectrum of hypokalemia-induced weakness, emphasizing the importance of distinguishing primary Hypokalemic Periodic Paralysis (HPP) from secondary causes. While sporadic HPP remains the most common presentation, a significant number of patients were found to have underlying renal, metabolic, or toxic etiologies.
- **Prodromal Symptoms and Onset:** Patients with HPP frequently experienced prodromal symptoms, including limb heaviness, thirst, and palpitations, with a mean onset duration of 13 hours before clinical weakness.
- **ECG Findings and Respiratory Complications:** ECG abnormalities, particularly U waves and sinus bradycardia, were commonly observed. However, no significant correlation was found between ECG changes and the severity of weakness. Severe potassium depletion was associated with respiratory muscle involvement, underscoring the risk of life-threatening complications.
- **Management and Treatment Considerations:** HPP patients required significantly lower potassium replacement than non-HPP cases, highlighting the need for cautious supplementation to avoid rebound hyperkalemia. Acetazolamide showed potential in preventing recurrent attacks, but its effectiveness varied, particularly in patients with renal tubular acidosis.
- **Multidisciplinary Approach:** A multidisciplinary team, including

nephrologists, neurologists, dietitians, and pharmacists, is crucial for optimizing patient care, preventing recurrent episodes, and improving long-term outcomes.

- **Future Directions**

Further research should focus on:

- Refining treatment protocols for hypokalemia-related paralysis
- Exploring genetic predisposition in HPP
- Investigating alternative pharmacologic agents for managing recurrent episodes

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