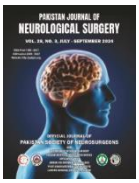

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Original Research

Etiological Spectrum of Patients with Hypokalemic Paralysis

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ABSTRACT

Objective: To determine the etiological spectrum of patients with Hypokalemic paralysis.

Materials & Methods: A descriptive Cross-Sectional study was conducted in the Department of Neurology, Mayo Hospital Lahore. The duration of the study was 12 months. After history and examination, biochemical tests (serum levels of sodium, potassium, magnesium, bicarbonate, chloride, blood pH, urine pH, and urine calcium), along with serum TSH and free T4 levels were measured. Dengue serology was performed for fever-related cases. The following were considered as the outcome variables: thyrotoxic periodic paralysis (TPP), hypokalemic periodic paralysis (HPP), renal tubular acidosis (RTA), dengue fever, Gitelman syndrome, and gastroenteritis.

Results: A total of 125 patients participated in the study. The mean age was 42.62 years. There were 65 males and 60 females. The mean potassium level was 2.61 ± 0.40 mmol/L (range: 2-3.40 mmol/L). Etiological factors included HPP (55.2%), TPP (25.6%), RTA (32.8%), and Gitelman syndrome (24.8%).

Conclusion: Over half of the patients (55.2%) were diagnosed with HPP, with other causes being TPP (25.6%), RTA (32.8%), and Gitelman syndrome (24.8%). It is crucial to quickly identify these conditions in patients with hypokalemic paralysis to improve treatment prognosis.

Keywords: Hypokalemic Paralysis (HP), Hypokalemic Periodic Paralysis (HPP), Thyrotoxic Periodic Paralysis (TPP), Gitelman Syndrome, Renal Tubular Acidosis (RTA).

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INTRODUCTION

Hypokalemic paralysis, a metabolic myopathy, encompasses a wide range of disorders marked by hypokalemia, acute muscle weakness, and life-threatening cardiac and respiratory complications.[1] Hypokalemic periodic paralysis can arise from either a temporary movement of potassium into cells or a more significant deficiency of potassium due to substantial loss through the kidneys or gastrointestinal tract.[2] It

usually starts in the first two decades of life and can be triggered by stress, carbohydrate-rich meals, infections, exercise, and other factors. It typically spares the bulbar, ocular, and respiratory muscles.[3,4]

Individuals affected by PP usually encounter intermittent episodes of muscle weakness, either focal or widespread, frequently triggered by certain stimuli.[5] During an episode, the neurologic examination often shows weakness predominantly in proximal muscles rather than distal ones, with the legs usually more affected than the arms. Hyporeflexia or areflexia is commonly observed.[6] A study by S Holm-Yildiz et al, showed that Hypokalemic periodic paralysis may evolve into a progressive myopathy in patients, regardless of whether they experience episodes of paralysis.[7]

Hypokalemia may be idiopathic or may present in association with diverse factors including thyrotoxicosis, barium poisoning, Gitelman syndrome, renal tubular acidosis, and gastrointestinal potassium loss.[8] Recently, dengue fever has emerged as a significant contributor to acute neuromuscular weakness associated with hypokalemia. This condition, known as dengue-associated hypokalemic paralysis, has been documented in the literature as a transient ailment.[9,10] Thyrotoxic periodic paralysis involves a Kir2.6 potassium channel mutation, leading to reduced potassium efflux and muscle weakness. Renal tubular acidosis (RTA) causes severe hypokalemia and is linked to kidney conditions. Gastrointestinal potassium losses are also common due to significant fluid loss.[11,12]

A study by Kayal et al. showed that out of 56 patients, 24 experienced hypokalemic paralysis due to secondary causes: 4 with distal RTA, 3 with TPP, 4 with Gitelman syndrome, and 1 with dengue fever. All patients recovered after potassium replacement, but the secondary group took significantly longer to recover than those with primary hypokalemic paralysis.[13,14]

The rationale of this study was to investigate the clinical characteristics and outcomes of patients presenting with hypokalemic paralysis, particularly comparing those with primary and secondary causes. Understanding the differences in recovery time and treatment response between primary and secondary hypokalemic paralysis could provide valuable insights into the management and prognosis of these patients. Additionally, identifying the specific secondary causes contributing to hypokalemic paralysis could aid in early diagnosis and appropriate management strategies for affected individuals. Given the lack of data on the causes of hypokalemia in developing nations, especially in hospitalized patients, and no local studies conducted thus far, our research aims to unveil the diverse causes of hypokalemic paralysis among patients seeking care at a tertiary hospital.

MATERIALS & METHODS

Study Setting and Duration

The study was conducted in the Department of Neurology, Mayo Hospital Lahore after approval from the Ethical Review Board of King Edward Medical University Lahore. The duration of the study was 12 months after approval of the synopsis, from April 2023 to April 2024. This study used a non-probability consecutive sampling technique, including patients who presented with hypokalemic paralysis at Mayo Hospital.

Inclusion Criteria

Patients with acute paralysis were included in the research. This included both genders, aged 18-60 years, diagnosed with hypokalemic paralysis (serum potassium levels less than 3.5 mEq/L).

Exclusion Criteria

Patients with a history of acute neuropathies and myelopathies (e.g., polio), Guillain-Barré syndrome, or those on diuretic drugs were excluded.

Data Collection

Data collection regarding demographics, illness duration, previous episodes, family history of acute muscle weakness, and a thorough neurological examination. Biochemical parameters such as serum sodium, potassium, magnesium, bicarbonate, chloride levels, blood pH, urine pH, and urine calcium levels were assessed. Additionally, serum TSH levels and free T4 were measured in all patients, while those presenting with fever and acute muscle weakness underwent dengue serology. The study evaluated various underlying causes of hypokalemic paralysis, including hypokalemic periodic paralysis, Gitelman syndrome, thyrotoxic periodic paralysis, dengue fever, gastroenteritis, and RTA. All investigations were conducted at no cost to the participants. Patients were examined at the time of presentation and at the time of discharge and improvement was compared by using Hughes Scale.

Data Analysis

Data was analyzed using SPSS v23.0. Qualitative variables were expressed as frequencies and percentages, while quantitative variables were presented as means ± standard deviations. Stratification was performed for age, gender, and disease duration. As the data involved qualitative and quantitative variables, 2 × 2 contingency table was used, and a Chi-square test was applied with a significance level of $p \leq 0.05$.

RESULTS

Age and Gender Distribution

A total of 125 subjects participated in the study. The age ranged between 18 and 60 with a mean of 42.62±9.99. 60 subjects (48%) were female and 65 (52%) were male. The potassium levels ranged from 2.00 to 3.40 with a mean value of 2.61±0.40. Out of these subjects, 58 (46.40%) had a past recurrence, whereas, 67 subjects (53.60%) had no history of Hypokalemic paralysis.

Clinical Presentation

At the time of presentation, all the patients were categorized as grade 4 (bed or chair-bound) according to Hughes Scale. However, by the time of discharge, almost all the patients showed improvement in their weakness: 48 patients (38.4%) were considered normal as they completely regained their strength, 54 patients (43.2%) exhibited minor symptoms but were able to run, 18 patients (14.4%) could walk up to 5 meters without support, and only 5 patients (4.0%) required a walker or support to walk up to 5 meters.

The frequency based on etiology was highest for Primary Hypokalemic periodic paralysis, affecting 55.20% of cases. Thyrotoxic periodic paralysis was present in 25.60% of cases, Renal Tubular Acidosis in 32.80%, and Gitelman Syndrome in 24.80% (Table 1).

Table 1: Number and percentage of cases with respect to etiology.

Etiology	Yes	No
Primary Hypokalemic periodic paralysis	69 (55.20%)	56 (44.80%)
Thyrotoxic periodic paralysis	32 (25.60%)	93 (74.40%)
Renal Tubular Acidosis	41 (32.80%)	84 (67.20%)
Gitelman Syndrome	31 (24.80%)	94 (75.20%)

Comparison of Hypokalemic Paralysis with Respect to Age Groups

There was no significant effect of age on the prevalence of hypokalemic periodic paralysis (HPP) (p=0.354). Similarly, no difference was observed in terms of age in thyrotoxic periodic paralysis (TPP) and Gitelman syndrome (p=0.903 and 0.641, respectively). However, renal tubular acidosis (RTA) was more common in the 41-60 age group than in the 18-40 age group (p=0.024) (Tables 2-5).

Comparison of Hypokalemic Paralysis with Respect to Gender

The comparison of gender across different conditions shows no significant differences. For Hypokalemic Periodic Paralysis (p=0.300), Thyrotoxic Periodic Paralysis (p=0.577), Renal Tubular Acidosis (p=0.376), and Gitelman Syndrome (p=0.233), the prevalence is similar between males and females (Tables 6-9).

Comparison of Hypokalemic paralysis with respect to the Duration of Disease

No significant differences were observed when comparing disease durations. For Hypokalemic periodic paralysis (p=0.78), Thyrotoxic periodic paralysis (p=0.40), Renal Tubular Acidosis (p=0.80), and Gitelman syndrome (p=0.133), the prevalence of the conditions was similar regardless of whether the disease duration was less than one year or more than one year (Tables 10-13).

Table 2: Comparison of Hypokalemic periodic paralysis with respect to age groups.

Age Groups (Years)	Hypokalemic Periodic Paralysis		Total	Chi-square	p-value
	Yes	No			
18-40	29(60.4%)	19(39.6%)	48(100%)		
41-60	40(51.9%)	37(48.1%)	77(100%)	0.857	0.354
Total	69(55.2%)	56(44.8%)	125(100%)		

Table 3: Comparison of Thyrotoxic periodic paralysis with respect to age groups (years).

Age Groups (Years)	Thyrotoxic Periodic Paralysis		Total	Chi-square	p-value
	Yes	No			
18-40	12(25%)	36(75%)	48(100%)		
41-60	20(26%)	57(74%)	77(100%)	0.015	0.903
Total	32(25.6%)	93(74.4%)	125(100%)		

Table 4: Comparison of Renal Tubular Acidosis with respect to age groups (years).

Age Groups (Years)	Renal Tubular Acidosis		Total	Chi-square	p-value
	Yes	No			
18-40	10(20.8%)	38(79.2%)	48(100%)		
41-60	31(40.3%)	46(59.7%)	77(100%)	5.062	0.024
Total	41(32.8%)	84(67.2%)	125(100%)		

Table 5: Comparison of Gitelman syndrome with respect to age groups (years).

Age Groups (Years)	Gitelman Syndrome		Total	Chi-square	p-value
	Yes	No			
18-40	13(27.1%)	35(72.9%)	48(100%)		
41-60	18(23.4%)	59(76.6%)	77(100%)	0.218	0.641
Total	31(24.8%)	94(75.2%)	125(100%)		

Table 6: Comparison of Hypokalemic periodic paralysis with respect to gender.

Gender	Hypokalemic Periodic Paralysis		Total	Chi-square	p-value
	Yes	No			
Male	33(50.8%)	32(49.2%)	65(100%)		
Female	36(60%)	24(40%)	60(100%)	1.07	0.300
Total	69(55.2%)	56(44.8%)	125(100%)		

Table 7: Comparison of Thyrotoxic periodic paralysis with respect to gender.

Gender	Thyrotoxic periodic paralysis		Total	Chi-square	p-value
	Yes	No			
Male	18(27.7%)	47(72.3%)	65(100%)		
Female	14(23.3%)	46(76.7%)	60(100%)	0.311	0.577
Total	32(25.6%)	93(74.4%)	125(100%)		

When the data was stratified by age, gender, and duration of disease, the frequencies of Hypokalemic periodic paralysis, Gitelman Syndrome, and Thyrotoxic periodic paralysis were found to be statistically similar across all strata, with p-values greater than 0.05. However, the frequency of Renal Tubular Acidosis differed significantly among different age groups (p-value < 0.05) but remained statistically similar between genders and across different durations of the disease (p-value > 0.05) (Table 14).

DISCUSSION

Hypokalemia, defined as a potassium concentration below 3.5 mmol/L, is linked to a tenfold increase in in-hospital mortality, mainly due to cardiovascular complications. It can occur through the redistribution of potassium from extracellular to intracellular fluid, where total body potassium remains normal, or through the loss of total body potassium, either renal or non-renal.[5]

Table 8: Comparison of renal Tubular Acidosis with respect to gender.

Gender	Renal Tubular Acidosis		Total	Chi-square	p-value
	Yes	No			
Male	19(29.2%)	46(70.8%)	65(100%)		
Female	22(36.7%)	38(63.3%)	60(100%)	0.783	0.376
Total	41(32.8%)	84(67.2%)	125(100%)		

Table 9: Comparison of Gitelman syndrome with respect to gender.

Gender	Gitelman syndrome		Total	Chi-square	p-value
	Yes	No			
Male	19(29.2%)	46(70.8%)	65(100%)		
Female	12(20%)	48(80%)	60(100%)	1.425	0.233
Total	31(24.8%)	94(75.2%)	125(100%)		

Table 10: Comparison of Hypokalemic periodic paralysis with respect to duration of disease

Duration of Disease	Hypokalemic Periodic Paralysis		Total	Chi-square	p-value
	Yes	No			
< 1 year	34(54%)	29(46%)	63(100%)		
≥1 year	35(56.5%)	27(43.5%)	62(100%)	0.078	0.78
Total	69(55.2%)	56(44.8%)	125(100%)		

Table 11: Comparison of Thyrotoxic periodic paralysis with respect to duration of disease.

Duration	Thyrotoxic periodic paralysis		Total	Chi-square	p-value
	Yes	No			
< 1 years	19(30.2%)	44(69.8%)	63(100%)		
≥ 1 year	13(21%)	49(79%)	62(100%)	1.386	0.239
Total	32(25.6%)	93(74.4%)	125(100%)		

Table 12: Comparison of Renal Tubular Acidosis with respect to Duration of Disease.

Duration	Renal Tubular Acidosis		Total	Chi-square	p-value
	Yes	No			
< 1 year	20(31.7%)	43(68.3%)	63(100%)		
≥ 1 year	21(33.9%)	41(66.1%)	62(100%)	0.064	0.80
Total	41(32.8%)	84(67.2%)	125(100%)		

Table 13: Comparison of Gitelman syndrome with respect to the duration of the disease.

Duration	Gitelman Syndrome		Total	Chi-square	p-value
	Yes	No			
< 1 year	12(19%)	51(81%)	63(100%)		
≥ 1 year	19(30.6%)	43(69.4%)	62(100%)	2.25	0.133
Total	31(24.8%)	94(75.2%)	125(100%)		

Table 14: Overall comparison of periodic paralysis with respect to different age groups, gender, and duration of disease.

Etiology	Comparison with Respect to Age		Comparison with Respect to Gender		Comparison with Respect to Duration	
	18-40 Years	41-60 Years	Male	Female	<1 Year	≥1 Year
Primary Hypokalemic periodic paralysis	29/48 (60.4%)	40/77 (51.9%)	33/65 (50.8%)	36/60 (60%)	34/63 (54%)	35/62 (56.5%)
p-value	0.354	0.354	0.300	0.300	0.78	0.78
Thyrotoxic periodic paralysis	12/48 (25%)	20/77 (26%)	18/65 (27.7%)	14/60 (23.3%)	19/63 (30.2%)	13/62 (21%)
p-value	0.903	0.903	0.577	0.577	0.239	0.239
Renal tubular acidosis	10/48 (20.8%)	31/77 (40.3%)	19/65 (29.2%)	22/60 (36.7%)	20/63 (31.7%)	21/62 (33.9%)
p-value	0.024	0.024	0.376	0.376	0.80	0.80
Gitelman syndrome	13/48 (27.1%)	18/77 (23.4%)	19/65 (29.2%)	12/60 (20%)	12/63 (19%)	19/62 (30.6%)
p-value	0.641	0.641	0.233	0.233	0.133	0.133

In our study, the patients had a mean age of 42.62 ± 9.99 years, with ages ranging from 18 to 60 years. The study population comprised 65 males (52%) and 60 females (48%). The mean potassium level was 2.61 ± 0.40 mmol/L, with a range from 2 to 3.40 mmol/L. We found that 55.2% of cases were due to Hypokalemic periodic paralysis, 25.6% were due to Thyrotoxic periodic paralysis, 32.8% were related to renal tubular acidosis, and 24.8% were linked to Gitelman syndrome.

In a study by Murya et al, out of 30 patients, 56.7% (17 patients) had hypokalemic periodic paralysis, 16.6% (5 patients) had thyrotoxic periodic paralysis, and 13.3% (4 patients) had renal tubular acidosis (RTA), including 13.3% (4 patients) with Gitelman syndrome, which aligns with our findings. Another study by G Chandramohan et al. reported that the majority of cases (61%) were non-periodic hypokalemic paralysis. Among these patients, 39% had metabolic acidosis, 38% had normal acid-base status, and 23% had metabolic alkalosis. The predominant secondary cause was distal renal tubular acidosis (36%), followed by Gitelman syndrome (18%), Thyrotoxic paralysis (4%), Hyperaldosteronism (3%), and proximal RTA (4%).[10,17]

Rao et al. reported on a study involving 31 patients with hypokalemic periodic paralysis (HPP). In this group, 42% (13 patients) were diagnosed with renal tubular acidosis (RTA), another 42% (13 patients) had primary Hyperaldosteronism, 2 patients had Thyrotoxic periodic paralysis (TPP), and 2 patients had sporadic periodic paralysis. All patients exhibited severe hypokalemia, hyperchloremic metabolic acidosis, and phosphaturia. In the study by Kayal et al, which included 56 patients, 24 had secondary hypokalemic paralysis, including 4 with distal RTA, 4 with Gitelman syndrome, 3 with TPP, and 1 with dengue fever. All patients recovered with potassium replacement therapy, but those in the secondary group required significantly more time to recover compared to those with primary hypokalemic paralysis. In another study of 56 hypokalemic paralysis patients (mean age 36.76), including 15 females, 24 had secondary causes like distal RTA (4 cases), Gitelman syndrome (4 cases), and TPP (3 cases). Other conditions were Hypothyroidism, Gastroenteritis, Primary Hyperaldosteronism, Liddle's syndrome, Dengue fever, and alcoholism (2 cases each). Two females were positive for Antinuclear antibody. Unusual presentations included neck muscle weakness (4 cases), bladder involvement (3 cases), and various single instances. Five had a positive family history of such conditions. All responded well to potassium supplementation. The study highlighted that 42.9% of cases were due to secondary causes, emphasizing the need to

address underlying conditions.[9,15] These findings differ from those reported in our current study.

In a study conducted by Jung Kook Wi et al, involving 34 patients with hypokalemic paralysis, the average age was 38.59 ± 16.92 years, and six of the patients were female. Among these patients, 20.6% (7 patients) were diagnosed with idiopathic hypokalemic periodic paralysis, which is classified as the sporadic form. The remaining 79.4% (27 patients) had secondary hypokalemic paralysis, with 47.1% (16 patients) of the secondary cases identified as thyrotoxic periodic paralysis (TPP). Other causes included diuretics (4 patients, 11.8%), licorice consumption (2 patients, 5.9%), Gitelman syndrome (2 patients, 5.9%), and distal renal tubular acidosis (dRTA) (2 patients, 5.9%) [16] which was inconsistent to our study as our study showed a much higher frequency of primary hypokalemic paralysis.

In a study done by RK Garg et al, hypokalemic paralysis was attributed to secondary causes in 15 patients (51.7%) and categorized as idiopathic in 14 patients (42.3%). The study reported that 20.6% of the patients (six individuals) had thyrotoxicosis, 13.7% (four individuals) were diagnosed with dengue infection, 10.3% (three individuals) had distal renal tubular acidosis, and both Gitelman syndrome and Conn's syndrome were each found in 3.4% (one individual each).[12] In contrast to our study, this study only focused on the secondary causes of hypokalemic paralysis.

In a study by BA Sial et al, a relationship between gender and age was examined regarding the frequency of hypokalemic periodic paralysis, demonstrating a significantly higher frequency among males compared to females. In contrast, our study found a more balanced male-to-female frequency.[18] A study by M Gupta et al, showed findings very similar to our study in which, primary hypokalemic periodic paralysis was observed in 52% of patients, while 48% had a secondary cause for their hypokalemic paralysis.[19]

Limitations

Although our study provided a thorough explanation of the etiology of hypokalemic paralysis, there were several limitations. Firstly, the study had a small sample size. Secondly, it was conducted at only one center. Additionally, genetic studies could not be performed in the patients due to cost constraints.

CONCLUSION

Distinguishing between HPP and secondary causes, such as TPP, RTA, or Gitelman syndrome, can significantly influence management decisions. Early diagnosis not only facilitates timely treatment but also reduces the risk of recurrent episodes and long-term complications associated with electrolyte disturbances. Therefore, clinicians should maintain a high index of suspicion for secondary causes of hypokalemic paralysis and promptly initiate comprehensive diagnostic evaluations to optimize therapeutic interventions and ensure favorable patient outcomes.

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Additional Information

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Human Subjects: Consent was obtained by all patients/participants in this study.
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AUTHORS CONTRIBUTION

Sr.#	Author's Full Name	Intellectual Contribution to Paper in Terms of:
1.	Khawaja Muhammad Ali & Muhammad Athar Javed	1. Study design and methodology.
2.	Muhammad Husnain & Khawaja Muhammad Ali	2. Paper writing.
3.	Khawaja Muhammad Ali & Muhammad Husnain	3. Data collection and calculations.
4.	Safia Bano & Khawaja Muhammad Ali	4. Analysis of data and interpretation of results.
5.	Safia Bano & Muhammad Husnain	5. Literature review and referencing.
6.	Ahsan Numan, Safia Bano & Muhammad Husnain	6. Editing and quality insurer.