Original Article

Etiological spectrum of hypokalemic paralysis: A retrospective analysis of 29 patients

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Abstract

Background: Hypokalemic paralysis is characterized by episodes of acute muscle weakness associated with hypokalemia. In this study, we evaluated the possible etiological factors in patients of hypokalemic paralysis. **Materials and Methods:** We reviewed the records of 29 patients who were admitted with a diagnosis of hypokalemic paralysis. Modified Guillain-Barre´ Syndrome disability scale was used to grade the disability. **Results:** In this study, 15 (51.7%) patients had secondary causes of hypokalemic paralysis and 14 patients (42.3%) had idiopathic hypokalemic paralysis. Thyrotoxicosis was present in six patients (20.6%), dengue infection in four patients (13.7%), distal renal tubular acidosis in three patients (10.3%), Gitelman syndrome in one patient (3.4%), and Conn's syndrome in one patient (3.4%). Preceding history of fever and rapid recovery was seen in dengue infection-induced hypokalemic paralysis. Approximately 62% patients had elevated serum creatinine phosphokinase. All patients had recovered completely following potassium supplementation. Patients with secondary causes were older in age, had significantly more disability, lower serum potassium levels, and took longer time to recover. **Conclusion:** In conclusion, more than half of patients had secondary causes responsible for hypokalemic paralysis. Dengue virus infection was the second leading cause of hypokalemic paralysis, after thyrotoxicosis. Presence of severe disability, severe hypokalemia, and a late disease onset suggested secondary hypokalemic paralysis.

Key Words

Acute flaccid paralysis, dengue virus, hypokalemia, hypokalemic paralysis

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Introduction

Hypokalemic paralysis is characterized by acute flaccid paralysis associated with hypokalemia. Hypokalemic paralysis is caused either by an enhanced shift of potassium ion into the cells or following a significant renal or gastrointestinal loss of potassium. Life-threatening complications such as cardiac arrhythmia and respiratory involvement may infrequently be associated with hypokalemic paralysis.[1-3]

Familial hypokalemic periodic paralysis is inherited in an autosomal dominant manner. Most individuals with familial hypokalemic periodic paralysis have an affected parent.[2] Thyrotoxicosis is the most frequent secondary cause of hypokalemic paralysis. Thyrotoxic hypokalemic paralysis is common in the Asian population whereas familial periodic paralysis is common in Caucasians.[1,4] Secondary hypokalemic paralysis often result from profound gastrointestinal and renal potassium loss. Viral infections, such as dengue virus and chikungunya virus, have also been reported to precipitate hypokalemic paralysis.[5-7] Patients with hypokalemic paralysis need a diligent search for the possible etiology and potassium replacement therapy. Majority of hypokalemic paralysis patients usually recover completely without any residual disability.[1-3]

There is a paucity of literature regarding the clinical and etiological spectrum of hypokalemic paralysis from tropical countries. In this retrospective study, we analyzed the clinical features and underlying causes of hypokalemic paralysis in 29 patients who were admitted in a tertiary care hospital.

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Materials and Methods

In this study, we analyzed the records of all hypokalemic paralysis patients who were admitted in a tertiary care facility from January 2009 to August 2011. The study protocol was approved by the Institutional Ethics Committee. The institutional review board issued a waiver of informed consent as the data used in the study was collected as part of the participants' routine care.

Definitions

Hypokalemic paralysis was defined as an acute-onset flaccid paralysis associated with low plasma potassium (<3.5 mEq/L). Hypokalemic periodic paralysis was characterized with recurrent attacks of weakness in association with hypokalemia. Thyrotoxic hypokalemic periodic paralysis was defined as occurrence attacks of acute flaccid paralysis and low serum K+ concentration in the presence of hyperthyroidism confirmed by thyroid function tests.[1,3,8] Patients of hypokalemic paralysis where no cause was identified were classified as having idiopathic hypokalemic paralysis. Familial hypokalemic paralysis was included in the idiopathic hypokalemic paralysis group. Patients with demonstrably known causes were classified as secondary hypokalemic paralysis.[9] Patients of thyrotoxic periodic paralysis were included in the secondary hypokalemic paralysis group.

Evaluation

Records of patients, diagnosed as hypokalemic paralysis, were evaluated. All patients were subjected to a detailed clinical evaluation. Demographic and clinical data that were recorded included age, sex, duration of illness, number of prior episodes if any, any precipitating event such as exertion or heavy carbohydrate meal, time of onset of symptoms, whether weakness was first noticed in the morning on awakening or any other time of day, history of hypertension, history of renal stones, bone pain, dry mouth, drug intake, and a family history of hypokalemic paralysis. Neurological evaluation included assessment of cranial nerves, motor system, sensory system, and respiratory function. Power of muscles was assessed using the Medical Research Council scale. Modified Guillain-Barre´ syndrome (GBS) disability scale was used.[10]

A battery of laboratory tests, which included complete hemogram, serum sodium, potassium, bicarbonate, chloride, magnesium, serum creatinine, fasting serum calcium and phosphate, serum albumin, globulin, alkaline phosphatase, creatine phosphokinase (CPK; laboratory reference value = 24-195 IU/L), arterial blood gas analysis, and renal and liver function tests, was performed. Fasting urinary pH, 24 h urinary calcium, phosphate, and creatinine were also performed. Electrocardiogram and cardiac evaluation records were reviewed.[9] Patients were subjected to thyroid function tests. Immunoglobulin Mantibody-capture enzyme-linked immunosorbent assay or dengue viral antigen non-structural protein-1 detection was used to detect dengue virus infection. Nerve conduction studies were performed in all the patients after they recovered. Needle electromyography was performed after full recovery in 11 patients.

Thyrotoxicosis was defined as suppressed thyroid stimulating hormone level with high free T_4 and/or free T_3 levels. Distal renal tubular acidosis (RTA) was characterized by hypokalemia, hyperchloraemic metabolic acidosis, and inability to lower the urine pH below 5.5. The diagnosis of proximal RTA is based on the demonstration of a chronic hyperchloraemic metabolic acidosis and an acid urine pH <5.5. Patients with proximal RTA had a plasma bicarbonate concentration more than 15 mEq/L. The diagnosis of Gitelman syndrome was made in the presence of metabolic alkalosis, hypokalemia, hypomagnesaemia, and hypocalciuria.[11] Primary hyperaldosteronism or Conn's syndrome was diagnosed in the presence of hypertension with hypokalemia and alkalosis, with supportive evidence of high serum aldosterone level and a characteristic computed tomography of the adrenal glands.

Statistical analysis

Data analysis was carried out with SPSS software (version 16.0). Statistical analysis of categorical variables was carried out using Fisher's exact test or the Chi-square test. Independent *t*-test or the Mann-Whitney U test was used for continuous variables. A value of $P \leq 0.05$ was considered significant.

Results

During the study period, 33 patients with a diagnosis of hypokalemic paralysis were admitted in our department. In two patients who were taking loop diuretics and in two other cases adequate etiological work-up were not available, and hence were excluded. A total of 29 patients were included for final analysis.

The mean age, at presentation, was 27.6 years (range 16-47 years). Majority of patients were male (26 male; 89.5%). Seven patients had a history of similar episodes in the past. The mean duration of illness in patients having a past history of similar episodes was 12.7 months (median 9 months; range 4-30 months) and they had a median of 6 episodes (range 3-15). One of them was a previously diagnosed case of recurrent hypokalemic paralysis and was taking potassium supplementation along with acetazolamide; he was later diagnosed as a case of thyrotoxicosis [Tables 1 and 3].

Ten patients had myalgia and eight patients had a history of fever prior to development of weakness. Two patients had a family history of similar illness. One patient with idiopathic and two patients with secondary hypokalemic paralysis had respiratory involvement; none of them required ventilatory support. All patients had presented with quadriparesis and 24 patients had a grade ≥3 measured on the GBS disability scale. The mean time from onset to maximum weakness was 7.3 h ± 2.5 h. Deep tendon reflexes were absent in 5 patients and 11 patients had hypoactive but demonstrable deep tendon reflexes. Sensations were normal in all the patients. Nerve conduction studies were normal in all the patients. They were performed only after full recovery [Table 3].

Serum potassium level at the time of presentation ranged between 1.2 mmol/L and 3.2 mmol/L (mean 2.30 mmol/L; median 2.1 mmol/L). Thyrotoxicosis was present in six patients and dengue virus infection in four patients. Three patients had acidosis, and alkalosis was present in two patients. Hypomagnesaemia was present in one patient and hypocalcaemia in six patients. Nephrocalcinosis was present in two patients and electrocardiographic changes (ST changes and prominent U waves) of hypokalemia were noted in 15 patients. Serum CPK (more than 174 units/L) was elevated in 18 patients. Hypertension was present in one patient, who was later diagnosed as having Conn's syndrome [Tables 1-3].

Hypokalemic paralysis was categorized as idiopathic in 14 (48.3%) patients and secondary in 15 (51.7%) patients.

Table 1: Demographic and clinical profile of 29 patients of hypokalemic paralysis

Age (years)	Sex	Family history	Previous attacks	Precipitating factors	Progression time (h)	Initial GBS disability scale (grade)	Recovery time (h)	Past recurrence (number of episodes)
35	M	A	P	Heavy meal	6	4	56	0
45	M	A	A	Exertion	8	4	45	3
47	M	A	P	None	7	5	60	1
31	M	A	A	Heavy meal	12	4	65	0
29	M	A	A	Exertion	9	4	55	1
32	M	A	A	Diarrhoea	3	2	48	0
22	M	A	A	None	5	4	58	0
21	M	A	A	None	7	4	24	0
29	M	A	A	None	4	4	28	0
39	F	A	A	None	8	4	16	0
23	F	A	P	Exertion	9	3	24	0
28	M	A	A	None	12	4	37	1
24	M	A	A	None	10	4	42	0
30	M	A	A	None	7	5	61	0
42	M	A	A	None	5	4	43	0
29	M	P	A	Heavy meal	9	3	21	0
20	M	A	A	Exertion	8	5	28	0
17	M	P	P	None	4	2	39	0
29	M	A	P	Diarrhoea	6	4	46	0
21	M	A	A	Diarrhoea	7	3	30	0
22	M	A	A	Heavy meal	5	4	12	0
16	M	A	P	None	7	3	16	1
20	M	A	A	Exertion	6	2	40	0
22	M	A	A	None	5	3	21	0
26	F	A	A	Heavy meal	11	2	16	0
30	M	A	P	None	12	4	18	0
32	M	A	A	None	6	4	26	0
22	M	A	A	None	4	3	14	0
17	M	A	A	Exertion	9	2	18	0

M=Male, F=Female, P=Present, A=Absent, GBS=Guillain-barre syndrome

The idiopathic category also included two probable familial cases of hypokalemic paralysis. In the secondary hypokalemic paralysis group, thyrotoxicosis was present in six patients (20.6%), dengue virus infection in four patients (13.7%), distal RTA in three patients (10.3%), Gitelman syndrome in one patient (3.4%), and Conn's syndrome in one patient (3.4%).

Patients with mild to moderate hypokalemia were managed with oral potassium supplementation; patients with severe hypokalemia (serum potassium <2 meq/L) or who were not able to take anything orally or patients with potentially life-threatening disease (cardiac arrhythmia, respiratory compromise) were treated with intravenous therapy. The dose for oral supplementation was 20-25 meq/6 hourly and for intravenous treatment it was 40 meq/4 hourly given in 20% mannitol as slow infusion.[12] Patients of thyrotoxicosis were additionally treated with carbimazole and propranolol. Patients with idiopathic hypokalemic paralysis having recurrent attacks were also given acetazolamide (250 mg thrice daily).

All the patients improved completely (Grade 0 on GBS disability scale) after potassium supplementation. The mean recovery time (start of potassium supplementation to complete recovery) was 34.7 h.

Comparison between idiopathic and secondary hypokalemic paralysis

Patients with idiopathic hypokalemic paralysis were significantly younger compared to those with secondary hypokalemic paralysis. Patients with secondary hypokalemic paralysis had significantly more disability (P = 0.025) and significantly lower serum potassium levels (P = 0.001). Patients with secondary hypokalemic paralysis took more time to recover completely (44.13 + 15.5 vs. 24.6 + 10.7 h; P = 0.001) and the duration of hospital stay was significantly prolonged (2.71 + 0.72 vs. 3.26 + 0.59 days; P = 0.033) [Table 3].

Follow-up

None of the patients died during follow-up (median 12 months, ranging from 2 months to 28 months). Five patients had recurrence during the follow-up period. Three of them had thyrotoxicosis, one patient had distal RTA, and one had idiopathic hypokalemic paralysis. None of the patients with dengue infection had recurrence during follow-up (median 7 months, ranging from 4 months to 9 months).

Discussion

Secondary hypokalemic paralysis is usually associated with renal and gastrointestinal disorders.[13] In our study, 15 (52%)

Table 2: Biochemical parameters of 29 patients of hypokalemic paralysis

K^+ meq/L	Na^+ meq/L	HCO_3 meq/L	Cl^- meq/L	Mg^{2+} meq/L	Blood pH	Urine pH	CPK (IU/L)	Diagnosis
1.6	136	22	104	0.8	7.39	6.7	2100	TP
1.9	140	23	101	0.9	7.36	7	750	TP
2.1	138	21	100	1	7.4	6	400	TP
1.4	137	24	98	0.8	7.38	6.5	100	TP
2.4	142	23	102	0.9	7.39	6.6	2340	TP
1.2	144	22	101	0.8	7.42	6.8	900	TP
2.1	140	24	100	0.9	7.43	6.7	600	Dengue
1.6	138	24	99	0.8	7.44	6.3	100	Dengue
2	139	23	98	0.9	7.43	6.7	450	Dengue
2.1	137	25	99	1	7.43	6.6	45	Dengue
3.1	137	17	110	1	7.2	6.9	75	RTA1
2.1	140	16	112	1	7.1	6.2	563	RTA1
2.2	136	17	110	0.8	7.15	6	100	RTA1
1.8	142	30	101	0.25	7.5	8	508	Gitelman
1.9	150	30	105	0.9	7.47	6.8	900	Conn's
3	140	24	106	1	7.35	7	1300	IHP
2.9	136	22	101	1	7.38	6.8	100	IHP
3.1	134	23	100	0.9	7.39	6	1000	IHP
2.2	145	26	103	1	7.46	7	98	IHP
2.9	134	24	102	1	7.4	6.6	45	IHP
1.6	145	23	105	0.9	7.42	6.8	1145	IHP
3	140	22	101	0.8	7.42	6.5	775	IHP
2.9	136	22	100	0.9	7.4	6.8	90	IHP
3.2	138	24	102	0.9	7.42	6	1100	IHP
2.8	136	22	100	1	7.4	6.3	38	IHP
1.9	139	23	101	0.8	7.38	6.4	2145	IHP
1.9	140	24	100	1	7.37	6.7	458	IHP
2.9	141	22	99	0.9	7.39	6.4	87	IHP
3	136	26	102	0.8	7.35	6	1542	IHP

K^+=Serum potassium, Na^+=Serum sodium, Cl^-=Serum chloride, HCO_3=Serum bicarbonate, Mg^{2+}=Serum magnesium, CPK=Creatine phosphokinase, IU/L=International unit/liter; TP=Thyrotoxic paralysis, IHP=Idiopathic hypokalemic paralysis, RTA=Distal renal tubular acidosis

patients had secondary cause of hypokalemic paralysis, which included thyrotoxicosis, dengue viral infection, distal RTA, Gitelman syndrome, and Conn's syndrome. A study from the southern part of India had almost similar observations. Ninety-four percent of patients had secondary hypokalemic paralysis. Hyperaldosteronism was present in 42% patients, RTA in 42%, thyrotoxicosis in 6.4%, Gitelman syndrome in 3.2%, and sporadic paralysis in 6.4% of patients in that series.[9] In a large study from Taiwan, which included 97 patients, 68% patients had demonstrable secondary causes. Thyrotoxicosis was the most frequent (40.2%) cause.[1]

Hypokalemic paralysis is known to be associated with dengue virus infection.[5,6] In our series, dengue virus infection was an important cause for hypokalemic paralysis. Four of our patients had hypokalemic paralysis due to dengue infection. Myalgias are frequently associated with dengue virus infection. The main mechanism postulated for hypokalemia in dengue infection is the redistribution of potassium in cells. It has been suggested that the stress of infection leading to catecholamine release or hyperinsulinemia may result in intracellular shift of potassium, which in turn that may cause hypokalemia.[5,14] None of these patients had recurrence during follow-up. However, a long follow-up study will be required to understand whether dengue hypokalemic paralysis patients had a tendency for recurrent episodic weakness such as thyrotoxicosis hypokalemic paralysis. A genetic defect also needs to be explored.

Five patients (17.5%) had generalized areflexia in our study and 11 patients had hypoactive deep tendon reflexes. In the presence of acute onset flaccid paralysis and generalized areflexia, one can easily misdiagnose these patients with GBS and consider these patients for administering intravenous immunoglobulin therapy. Several features may help in distinguishing hypokalemic paralysis from GBS, especially when electrophysiological studies are normal. In hypokalemic paralysis, the progression of weakness is often very rapid compared to that of GBS. In our study, the mean time from onset to maximum weakness was 7.3 h + 2.5 h for hypokalemic paralysis, whereas in GBS, according to a published literature, 34% of patients develop maximum weakness within 7 days, 70% within 14 days, and 84% within 21 days after onset of neuropathy.[15,16] Patients of hypokalemic paralysis recover rapidly following potassium supplementation. In the present study, the mean recovery time was 34.7 h + 16.5 h, whereas in GBS, recovery usually takes several months and one-third patients with GBS may have some disability at 1 year and approximately 5% may die during the period of acute illness.[17]

Table 3: Comparison of clinical and biochemical parameters (mean+SD) in idiopathic and secondary hypokalaemic paralysis

Parameter	Idiopathic hypokalemic paralysis (*n*=14)	Secondary hypokalemic paralysis (*n*=15)	*P* value
Age (years)	23.07+5.23	31.8+8.26	0.002
Sex (male)	13	13	1
Duration of symptoms (h)	29.78+8.6	25.8+11.68	0.308
Family history	2	0	
Carbohydrate rich meal	3	2	0.651
Heavy exertion	3	3	1
Fever	2	6	0.215
Diarrhoea	2	1	0.598
Myalgia	4	6	0.7
Respiratory compromise	1	2	1
Previous attack (number of patients)	4	3	0.682
GBS disability scale (grade)	3.9±0.7 (median=4)	3.1±0.9 (median=3)	0.025
DTRs (absent)	3	2	0.651
Serum potassium (meq/L)	2.66+0.52	1.96+0.44	0.001
Serum calcium (mmol/L)	2.02+0.06	1.96+0.16	0.216
CPK level (elevated)	8	10	0.710
Recovery time (h)	24.64+10.65	44.13+15.48	0.001
Hospital stay (number of days)	2.71+0.72	3.26+0.59	0.033

DTRs=Deep tendon reflexes, GBS=Guillain-barre syndrome, CPK=Creatine phosphokinase

In our study, all the patients had recovered completely.

In our study, thyrotoxicosis was present in 20.6% cases and was the most frequent identifiable cause. The Asian population has a high propensity to develop thyrotoxic periodic paralysis. The most common cause was Graves's disease, although it may occur in association with any of the causes of hyperthyroidism. Increased activity of the Na^+- K^+- ATP- ase pump by thyrotoxicosis induced the hyperadrenergic state, directly by thyroid hormone *per se* or by hyperinsulinemia , which lead to increased cellular potassium uptake and hypokalemia.[18]

Familial hypokalemic paralysis is an autosomal dominant condition. The symptoms usually begin before the age of 25 years and a male preponderance is observed. Attacks may be precipitated by a carbohydrate-rich meal (secondary to insulin secretion) or physical exertion.[8] In our study, five patients had a history of taking carbohydrate-rich meal and six patients had a history of heavy exertion prior to development of weakness.

Secondary hypokalemic paralysis was associated with significantly lower serum potassium levels, and possibly profound potassium loss led to a longer time to complete recovery, and a longer stay in the hospital. Several past studies had similar observations. These studies also noted that serum potassium levels were significantly lower and muscle strength was lesser in the secondary hypokalemic paralysis group compared to those with idiopathic hypokalemic paralysis.[19,20]

Identifying and avoiding triggers of attacks are important in the management of hypokalemic paralysis. Triggers have been reported to precipitate paralysis. The most important triggers are rest after exercise and carbohydrate-rich meals. Diarrhea can lead to potassium loss and subsequently hypokalemia. Hypokalemia is also common in patients who have fever. Fever-induced hypokalemia may be due to low caloric intake, excessive vomiting, excessive sweating, administration of chloroquine and other drugs, and adrenergic hyperactivity.[21] Other precipitating factors include cold, upper respiratory tract infections, lack of sleep, fatigue, intake of Chinese food, alcohol, dehydration, startle, drugs, menstrual cycle, change in humidity or barometric pressure, and change in daily activity patterns.[22] Some of these triggers were present in our patients as well.

In our study, approximately 62% patients with hypokalemic paralysis had elevated CPK. Elevated CPK has been reported in patients with acute episodes of hypokalemic paralysis as well in those recovering from the same. Increased CPK levels in acquired hypokalemic paralysis may indicate damage to muscle fibers. Profound hypokalemia can even lead to rhabdomyolysis. Hypokalemia-induced muscle ischemia leading to an alteration in the internal milieu of the muscle fiber has been proposed to be a mechanism responsible for muscle damage.[23-25]

Conclusion

More than half of our patients of hypokalemic paralysis had some secondary etiology. Dengue infection was an important cause of hypokalemic paralysis. Presence of severe disability and severe hypokalemia suggested a secondary cause of hypokalemic paralysis.

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