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Original Research Article

Aetiological, Clinical and Metabolic Profile of Hypokalemic Periodic Paralysis in Adults

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Conflict of interest: Nil

Abstract:

This prospective observational study focuses on patients diagnosed with hypokalemic periodic paralysis, with the objective of analysing their etiological, clinical, and metabolic profiles. The study population comprised patients admitted to the medical wards and the medical intensive care unit of a tertiary care center in Mumbai. The research was conducted over a duration of 18 months. Hypokalemic paralysis is a significant, treatable, and reversible cause of acute flaccid paralysis. However, limited Indian studies are available to guide the investigation and treatment within the Indian context hence the study was planned. This condition is also referred to as "sporadic paralysis" in some literature and, due to its recurrent nature, is classified as a type of "periodic paralysis." Most cases are sporadic, while non-sporadic instances are exceedingly rare. Management strategies vary based on individual case scenarios Early diagnosis, prompt treatment, and necessary lifestyle modifications can lead to remarkable improvements in patient outcomes. These steps play a crucial role in the effective management of hypokalemic paralysis, reducing its recurrence and ensuring better quality of life for affected individuals.

Keywords: hypokalemia, paralysis, TTKG.

Introduction

Potassium is a chemical element with the symbol K (Latin: kalium). Acute Hypokalemic paralysis, a clinical syndrome characterized by acute systemic weakness and low serum potassium, is a rare but treatable cause of acute weakness. It is best known form of periodic paralysis. First case was reported by Hartwig in 1874 [1] Major study was done by Talbott's who reviewed 152 cases prior to 1941 [2]. Hypokalemic paralysis represents heterogenous group of disorders with a final common pathway, presenting as acute weakness and hypokalemia [2]

Ingrid Gamstorp campaigned vigorously for the rejection of the terms "hypo" and "hyper" kalemic, hypopotassemia, and hyperpotassemia. She stated that..."It is likely that the severity of symptoms are better related to the quotient between the intra and extracellular potassium than to the extracellular level alone. "[3]

Therefore, it is imperative for physicians, particularly those working in acute care settings, to be knowledgeable about the causes of hypokalemic paralysis. At this time, it is also necessary to rule out other causes of acute systemic weakness which may be neurologic, metabolic or infectious. Physicians immediate action in emergency set up can prevent further deterioration in patient condition.

Most cases are due to familial hypokalemic paralysis; however sporadic cases are associated with diverse underlying etiologies including thyrotoxic periodic paralysis, barium poisoning, renal tubular acidosis, primary hyperaldosteronism, liquorice ingestion and gastrointestinal potassium losses [4]. It is important to note at this point that treatment varies according to etiology of hyopkalemia, so simple test are important in emergency room to make a working diagnosis to prevent mismanagement [5]

The approach to the patient with hypokalemic paralysis includes a vigorous search for the underlying etiology and potassium replacement therapy. The mortality and morbidity associated with this condition is mainly related to complications secondary to hypokalemia which mainly includes cardiac arrhythmias and respiratory failure.

Aims and Objectives

It is a prospective observational study of patients with hypokalemic periodic paralysis, to study their aetiological, clinical and metabolic profile. The patients included were admitted in medical wards and medical intensive care unit, in a tertiary care centre in Mumbai. The study was done over a period of 18 months.

1. To study various etiological and precipitating factors.
2. To study clinical profile of the same.
3. To study various metabolic parameters in these patients.

Materials and Methods

This study was conducted in a tertiary care centre in Mumbai over period of 18 months. Patients presenting to EMS with paralysis of acute onset and documented to have hypokalemia were enrolled in this study.

Study included 52 patients' purpose of the study, procedures, investigative course of study was explained to the patient and legally acceptable representatives relative. If satisfying the criteria, written informed consent was obtained after explaining the study protocol in detail and the patient were included in the study.

Detailed history including age, sex, duration of illness, the present episode of weakness, precipitating factors like high carbohydrate diet. heavy exercise, starvation, vomiting, diarrhea, drugs, number of previous episodes and the occupation of the patient, family history were noted.

Each patient underwent a detailed clinical examination and other causes for weakness were rule out. Low serum potassium was documented. Each patient underwent ECG and ABG (arterial blood gases). Patient's spot urine sample was collected to calculate urine potassium, Urine K/C ratio and TTKG Serum bicarbonate levels of patients having normal or low blood pressure were checked and categorized accordingly for further investigations, as described in the given flow chart.

Definition of Patient

Inclusion Criteria

1. All patients of paralysis detected to have hypokalemia i.e. ($K^* < 3.5$meql/L).
2. Age 12 yrs.

Exclusion Criteria

1. Patient with weakness with documented potassium >3.5mmol/L.
2. Patients diagnosed to have condition with weakness with neurologic, infectious and other metabolic etiologies.
3. Patients who did not give valid consent.

Data Analysis

Data analysis was done by using statistical methods with application of Student 't' test wherever indicated.

Ethical Considerations

Ethical clearance to proceed was obtained from the Ethical Review Board of our teaching institute before commencement of the study.

Results

Table 1: Demographic data

Parameters	
No of cases	52
Age in years Range SD Mean	 17-60 yrs 08.97 28.81
BMI (Kg/Sqm) Range SD Mean	 15.5-28.0 02.39 22.51
Sex(%) Male Female	 41(82.0) 11(22.0)

In this study, age of the cases ranged from 17-60 years with average age **was 28.81 years.** Mean BMI **was 22.51 kg/sqm and 82.0% of total cases were male.**

Table 2: Hypokalemic periodic paralysis (HPP) divided into HPP and non-HPP group.

HYPOKALEMIC PERIODIC PARALYSIS (HPP)	**43**
NON-HYPOKALEMIC PERIODIC PARALYSIS (HPP)	
Diarrhoea	**2**
Hypomagnesemia	**1**
Distal renal tubular acidosis	**2**
Sjogren's Syndrome	**1**
Toluene exposure	**1**
Bartter's Syndrome	**1**
Gitelman's Syndrome	**1**

Table 3: Profile of Motor Weakness:

Motor Weakness	Total No of Cases (N=52)	Percentage
Paraparesis	36	69.2
Quadriparesis	10	19.2
Quadriparesis + Truncal involvement	04	07.7
Quadriparesis + Truncal weakness + Respiratory Involvement	02	03.9

69.2% of the total cases had paraparesis motor weakness followed by 19.2% quadriparesis, 7.7% quadriparesis + truncal involvement, 3.9% quadriparesis + truncal weakness + respiratory involvement

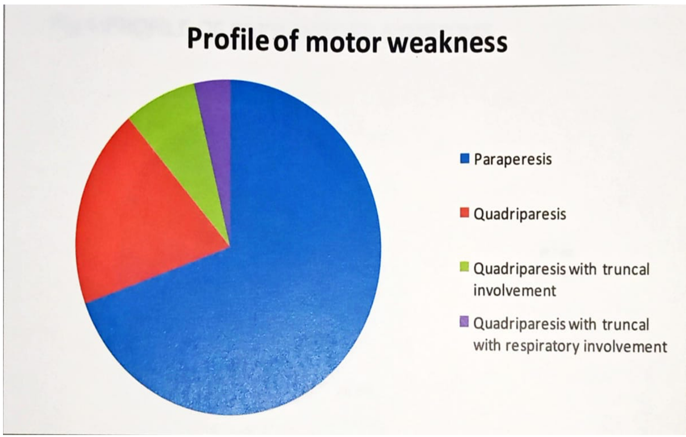

Figure 1: Graph showing distribution of cases according to presentation

Profile of Premonitory Symptoms.

Total No. of Cases (N = 52),78.8% cases had premonitory symptoms and 21.2% had no premonitory symptoms. The cases had an average duration of 13 hrs of premonitory symptoms.

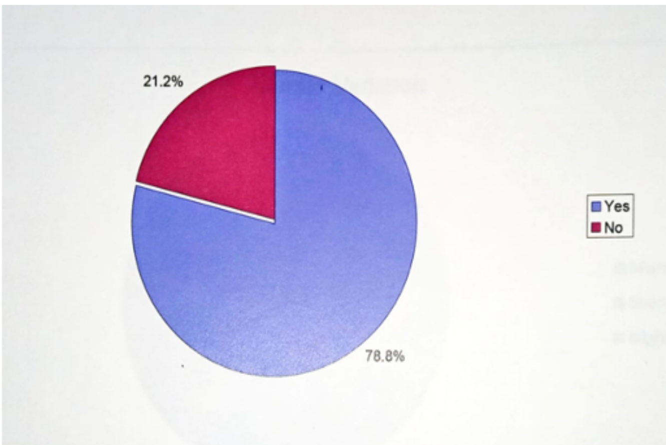

Figure 2: Profile of Premonitory Symptoms

Profile of diurnal variation in occurrence of attacks of weakness.

Out of all the patients, 31 patients had hypokalemic weakness in morning, 12 had in evening,9 had in night.

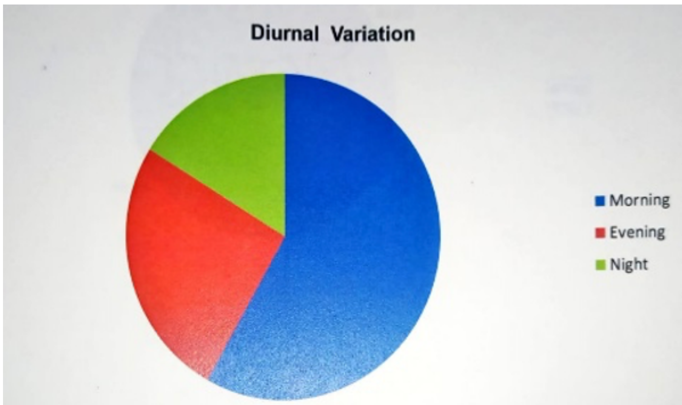

Figure 3: Profile of duration of premonitory symptoms

Profile of High Carbohydrate Intake History

The data states that 69.2% of the total cases did not have history of high carbohydrate intake followed by 30.8% had history of high carbohydrate intake.

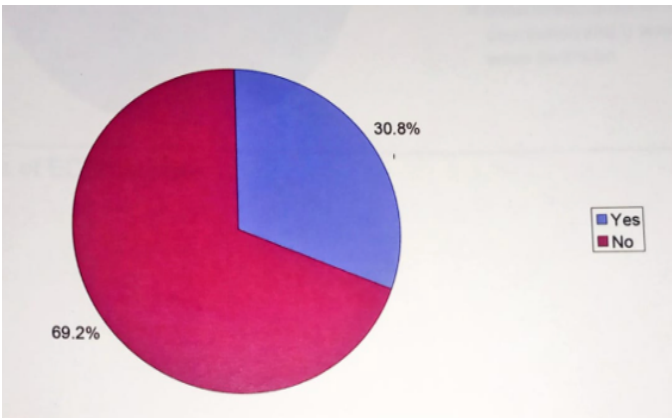

Figure 4: Profile of high carbohydrate intake history

Table 4: Profile of ECG changes

ECG Changes	No of Patients
Sinus bradycardia	20
U waves	28
Sinus bradycardia with ST depression, U waves and T wave inversion	4

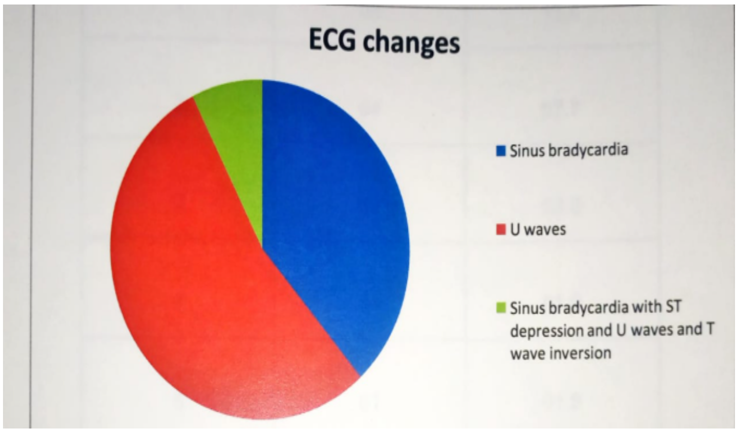

Figure 5: ECG Changes

Table 5: Number of Previous Attacks with Hypokalemic Paralysis

No of attacks	Total No of Cases (N=52)	Percentage
0	36	69.2
1	08	15.4
2	04	07.7
3	02	03.9
4	01	01.9
5	01	01.9

Above table shows that 69.2% patients of the total cases did not have any previous attacks followed by 15.4% one, 7.7% two, 3.9% three and 1.9% four and five attacks

Table 6: Comparison of Changes in Mean Potassium Required Between The Groups

Groups	Mean Potassium (Mean± SD)
HPP (N = 43)	2.25± 0.43
Non HPP(n=9)	1.72±0.53

By Student 't' Test *P <0.05 Significant

Mean potassium was 2.25 meql/l among HPP group which was significantly more as compared to 1.72meql/L in non HPP Group

Table 7: Comparison of Changes in Mean Serum Ph Between the Groups

Groups	Mean Serum pH
SPP (N = 43)	7.39 plus/minus 0.07
Non SPP (N = 9)	7.2 plus/minus 0.2

By Student t' Test * P <0.05 Significant

Mean serum pH was 7.39 among HPP group which was more as compared to 7.20 in Non HPP group and difference was statistically significant.

Table 8: Comparison of Changes in Mean Urine Potassium Between the Groups

Groups	Mean Urine Potassium (Mean ± SD)
HPP (N=43)	14.59 ±6.48
Non HPP (N = 9)	32.62 ±23.95

By Student't' Test * P < 0.05 Significant

Mean Urine potassium was 14.59 mmol/L among HPP group which was significantly low as compared to 32.62 mmol/L in Non HPP group.

Table 9: Comparison of change in Mean Bicarbonate between the two groups

Groups	Mean Bicarbonate (Mean ± SD)
HPP (N = 43)	24.91 plus/minus 2.73
Non HPP (N = 9)	17.2 plus/minus 13.53

Student't' Test P >0.05 Not Significant

According to this table mean bicarbonate was 24.91 meq/L among HPP group which was more as compared to 17.20 meq/L in Non HPP group but difference was not statistically significant.

Table 10: Comparison of Changes in Mean Chloride Between the Groups

Groups	Mean Chloride (Mean ± SD)
HPP (N = 43)	*101.86±05.90
Non HPP (N = 9)	112.11 +07.65

By Student't' Test *P < 0.05 Significant

This analysis states that mean chloride was 101.86 meql/L among HPP group which was significantly less as compared to 112.11 meql/L in Non HPP group.

Table 11: Comparison of Changes in Mean Urine Potassium Between the Groups

Groups	Mean Urine Potassium (Mean plus/minus S * D)(mmol/L)
HPP (N = 43)	* 14.59 plus/minus 6.48
Non HPP (N = 9)	32.62 plus/minus 23.95

By Student't' Test * P < 0.05 Significant

Mean Urine potassium was 14.59 mmol/L among HPP group which was significantly low as compared to 32.62 mmol/L in Non HPP group.

Table 12: Comparison of Changes in Mean TTKG Between the Groups

Groups	Mean TTKG (Mean ±SD)
HPP (N=43)	2.57 +0.31
Non HPP (N=9)	8.43±9.67

Student't' Test P>0.05 Not Significant

Above table shows that mean TTKG was 2.57 among HPP group which was low as compared to 8.20 in Non HPP group but difference was not significant

Table 13: Comparison of Changes in Mean Urine K/C Ratio Between the Groups

Groups	Mean Urine K/C(Mean plus/minus S * D)
HPP (N = 43)	* 2.13 plus/minus 0.27
Non HPP (N = 9)	3.23 plus/minus 1.15

By Student't' Test *P < 0.05 Significant

According to this table mean Urine K/C was 2.13 among HPP group which was significantly less as compared to 3.23 in Non HPP group.

Table 14: Comparison of Mean Potassium Required for Treatment Between the Two Groups

Groups	Mean Potassium Required (Mean plus/minus S * D) (meql/L)
HPP (N = 43)	* 58.72 plus/minus 20.15
Non HPP (N = 9)	95 plus/minus 49.75

Student't' Test therefore *P < 0.05 Significant

Above table shows that mean potassium required was 58.72 meql/L among HPP group which was significantly low as compared to 95.00meql/L in Non HPP group.

Table 15: Comparison of Changes in Mean Potassium Required with Motor Weakness

Motor Weakness	Mean Potassium Required (Mean plus/minus S * D) (meql/L)
Paraparesis (N = 36) (P)	58.47 plus/minus 20.31
Quadriparesis (N = 10) (Q)	62.5 plus/minus 32.01
Quadriperesis + truncal Involvement (N = 4) (Q + T)	77.5 plus/minus 29.86
Quadriparesis + Truncal +respiratory involvement (N = 2) (Q + T + R)	135 plus/minus 0

Table 16: Comparison of Changes in Mean Recovery Between the Groups

Groups	Mean Recovery (Mean ± SD) (in hours)
HPP (N = 43)	*26.77 +27.56
Non HPP (N = 9)	76.29 +30.73

Student 't' Test *P <0.05 Significant

In this study group mean recovery time was 26.77 among HPP group which was less as compared to 76.29 in Non HPP group and difference was statistically significant.

Discussion

In this prospective study patients with hypokalemia with weakness were screened for inclusion in the study Patients with other causes for weakness were excluded from study. Hypokalemia often has no symptoms, particularly when the disorder is mild (serum potassium, 3 to 3.5 meq/L). With more severe hypokalemia, nonspecific symptoms, such as generalized weakness, lassitude and constipation are more common.

When serum potassium decreases to less than 2.5 meql / L, muscle necrosis can occur, serum potassium less than 2.0 meql / L an ascending paralysis can develop, with eventual impairment of respiratory function.

The likelihood of symptoms appears to correlate with the rapidity of the decrease in serum potassium, underlying heart disease, and other physical co morbidities. [6]

For purpose of discussion and to make it simpler patient were divided into two groups, Hypokalemic Periodic Paralysis (HPP) in whom no cause for hypokalemia was found and Non-Hypokalemic Periodic Paralysis (Non-HPP) in whom a cause was found for hypokalemia that was in form of mainly gastrointestinal or renal loss of potassium.

In our study majority cases were males 82%(41) comparable with other studies [5,6] Male to female ratio is 4:1 is comparable to similar studies [1,5]

In study done by Lin Shih Hua male female ratio was 38:5. In study done by Rao et al male female ratio was 3:1.

Attacks usually occur in late childhood or late adolescence, rarely seen after age 25. Mean age in our study was 28. Age of patients range from 17-60 yrs. Mean BMI of our patients was 22.51 kg/sqm. Out of 52 patients 43 patients were diagnosed as case of HPP. Of 9 patients with Non-HPP 2 patients diagnosed as distal renal tubular acidosis, 2 patients with diarrhea and hypokalemia, 1 as toluene exposure with secondary distal renal tubular acidosis, 1 as Sjogren's with distal renal tubular acidosis, 1 as Bartter's syndrome, 1 as Gitelman syndrome and 1 patients as hypomagnesemia,

The etiology of HPP varies in different series In the study done by Narsing Rao et.al,31 patients presented with periodic paralysis, 42% diagnosed with renal tubular acidosis, 42 % with hyperaldosteronism, 2 with thyrotoxic paralysis 2 with sporadic periodic paralysis, 1 with Gitelman syndrome .

In similar study by Lin Shih-Hua; in series of 30 patients 13 had secondary causes of hypokalemia [7].

In Taiwan study in series of 97 patients 40% had thyrotoxic periodic paralysis,21% had sporadic periodic paralysis .

In other study by Arya SN in series of 22 patients 12 had other causes of hypokalemia mainly gastroenteritis, thyrotoxicosis and diuretic use.

In our study most common cause was sporadic with no cause been found for hypokalemia. In similar studies commonest cause for HPP was familial or sporadic [5,6] The most common cause also depends on the ethnicity, as thyrotoxic periodic paralysis been the most common cause in China.

In our study, 69.2% of the total cases on presentation has paraparesis, 19.2% had quadriparesis, 7.7% had quadriparesis plus truncal involvement, 3.9% had quadriparesis plus truncal weakness plus respiratory involvement.

In hypokalemic periodic paralysis the limb muscles are more commonly involved mainly the lower limb muscles, with proximal muscles involved earlier than distal and rarely involving truncal, bulbar or respiratory muscles [1] Deep tendon reflex may be diminished or absent Sensory system and level of consciousness are not affected. Of all these patients, 41 had premonitory symptoms of sensations of heaviness in the legs or aching of back. Other prodromal symptoms that have been reported in past are excessive hunger or thirst dry mouth, palpitation, sweating, diarrhea, nervousness sense of weariness or fatigue [1,2] Majority of patients in HPP group had premonitory symptoms. Similar complaints were also noted by Lin Sh et.al [7], The mean duration was roughly 13 hours after which patient presented with clinical weakness.

Majority of patients (n=31) presented with attacks early in morning The weakness started in lower limbs first and then involve upper limbs over mean duration of 13 hours. None of these patients had family history of similar Illness.

In our study history of high carbohydrate intake was present in only 38% of patients (n=16). All of these patients are in HPP group. High carbohydrate diet is known to precipitate attacks of hypokalemic weakness. These findings are not noted in similar study.

In our study, ECG (electrocardiogram) of 20 patients had sinus bradycardia, 28 had U waves and 4 had sinus bradycardia, ST depression, U waves and T wave inversion as there ECG findings.

However, in this study we found that none of the patients had cardiac arrhythmias in spite of serum potassium being as low as approximately 2 mmol/L.

Johnsen's [8] study of 106 Danish patients with HPP revealed two patients who developed transient diastolic murmurs during paralysis and another who developed a transient, partial A-V block. He also describes patients who developed bradycardia and unspecified arrhythmias during episodes.

Of all these patients, 41 had premonitory symptoms of sensations of heaviness in the legs or aching of back. Other prodromal symptoms that have been reported in past are excessive hunger or thirst dry mouth, palpitation, sweating, diarrhea, nervousness sense of weariness or fatigue(1.2) Majority of patients in HPP group had premonitory symptoms.

Similar complaints were also noted by Lin Sh et.al [2], The mean duration was roughly 13 hours after which patient presented with clinical weakness.

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No other study correlating degree of hypokalemia and ECG changes have been done. There is no other study were ECG changes of hypokalemia have been considered in diagnosing hypokalemia even before serum potassium lab reports are awaited. No correlation could be found out between ECG changes and degree of weakness present.

The commonest finding in our study was presence of U waves. Whether ECG findings can help in management of patients with HPP is matter of research.

Of all patients 16 patients had repeated attacks of which 8 patients Had 2 repeated attacks, 4 had 2 attacks, 2 had 3 attacks, 1 patient had 4 and 5 attacks each respectively.

In our study 36 primary hypokalemic periodic paralysis cases with no apparent cause for hypokalemia been found out despite extensive Investigations. No antecedent disease was found out in above cases. These cases showed complete recovery without any neurological deficit on potassium replacement.

In our study 2 patients with hypokalemic periodic paralysis hypokalemia was secondary to diarrhoea. These patients presented with multiple episodes of diarrhea. Investigations revealed hypokalemia with hyperchloremic metabolic acidosis with extra renal potassium loss with negative urinary anion gap suggesting of preserved function of urine acidification differentiating it from renal cause of acidosis. Anything that increases stool volume like infectious diarrhea to cancer chemotherapy can result in clinically significant potassium depletion and hypokalemia. In other study 60 patients who presented with quadriparesis 58% had gastroenteritis as cause of hypokalemia [9].

In our study one patient had hypokalemic periodic paralysis secondary to hypomagnesemia due to diarrhoea in HIV positive Human Immunodeficiency Virus) patients who presented with multiple episodes of diarrhoea with hyperchloremic metabolic acidosis with hypokalemia with negative urine anion gap. Persistent hypokalemia even after correction lead to further investigation which lead to diagnosis of hypomagnesemia. Magnesium depletion, induced either by dietary restriction or by abnormal loss, reduces intracellular potassium concentration and causes renal potassium wasting The depletion of intracellular potassium stores appears to be due to impairment of the activity of cell membrane Na*/K ATPase, but the mechanism by which magnesium depletion causes renal potassium loss in unclear. Magnesium depletion often coexists with potassium depletions as a result of drugs (e.g. and amphotericin B) or disease processes (eg.hyper aldosteronism and diarrhea) that cause loss of both ions, making it difficult to assess whether, the hypokalemia is caused by hypomagnesemia or is an independent effect. Regardless of the cause, ability to correct potassium deficiency is impaired when magnesium deficiency is present, particularly when the serum magnesium concentration is less than 0.5 mmol/L. Magnesium repletion improves the coexistent potassium deficit.

In our study 2 patients were diagnosed as case of dRTA. The presentation was hypokalemia, hyperchloremic metabolic acidosis normal anion gap, alkaline urine despite severe metabolic acidosis positive urine anion gap. USG (ultrasonography) abdomen was done to document nephrocalcinosis. One patient in our study was diagnosed as Sjogren's syndrome with hypokalemic periodic paralysis [10] .Patient on investigation was found to have dRTA. In view of her complaints of dry eyes, Schirmer's test was performed which was positive (wetting <5mm).

All patients in our study underwent thyroid function test but no patients was detected to have thyrotoxic periodic paralysis. We like to mention thyrotoxic periodic paralysis here as it is important cause of hypokalemic periodic paralysis in South-east Asian region.

Though no patient in our study was diagnosis as case of thyrotoxic periodic paralysis (TPP). TPP needs special mention due to its unique presentation and treatment

In our study it was also noted that severe potassium loss was associated with respiratory muscle involvement. In our study two patients required ventilator support in view of their respiratory failure.

Respiratory failure rarely occurs in patient with hypokalemic periodic paralysis (87.88) We found that respiratory weakness was associated with low mean serum potassium. These findings were not noted in similar study. Why respiratory muscles are rarely affected and whether there affection is related to serum potassium level is matter of research.

The mean serum potassium in patients in HPP group was 2.25 meql/L as compared 1.72 meql/L in non-HPP group which was statistically significant

In other study non-HPP group had serum potassium significant lower than HPP group

In our study mean serum pH was 7.39 among HPP group which was more as compared to 7.20 in Non HPP group and difference was statistically significant

Mean bicarbonate was 24.91 meql/L among HPP group which was more as compared to 17.20 meql/L in non-HPP

Mean chloride was 101.86 meql/L among HPP group which was significantly less as compared to 112.11 meql/L in Non HPP group.

In this study group mean Urine pH was 5.88 among HPP group which was low as compared to 6.39 in Non HPP group

Patients with secondary hypokalemic periodic paralysis thus have acid- base disturbances. Thus, patient with hypokalemic periodic paralysis should be investigated for secondary causes for periodic paralysis. It is important at this point to note that the further investigation differ depending on nature of acid-base disorder. Hyperchloremic metabolic acidosis form a major group of patients with non HPP which include (gastrointestinal loss, RTA). Hyperchloremia thus help in differentiation of this disorder from HPP.

In our study mean Urine potassium was 14.59 meql /L among HPP group which was significantly low as compared to 32.62meql/L in Non HPP group. Thus Renal and extra renal loss of potassium can safely be differentiated on basis of urinary potassium excretion. In non-HPP group the renal and extra-renal loss can be differentiated by urinary potassium excretion. Random measurement of the urinary potassium concentration is simple to perform but may be less accurate than a 24-h collection, since it is influenced by two independent factors, potassium secretion and water reabsorption in the medulla.

In our study mean TTKG was 2.57 among HPP group which was low as compared to 8.20 in Non HPP group but difference was not significant.

In study conducted by Joo KW it was found that TTKG is an important predictor to differentiate between renal and extra renal loss of potassium. It was found that higher TTKG correlated well renal potassium loss and high serum aldosterone levels.

In our study mean Urine K/C was 2.13 among HPP group which was significantly less as compared to 3.23 in Non HPP group. In non HPP group mean Urinary K/C ratio was 3.39 in patient with renal loss as compared to ratio of 2.11 in patients with Gastrointestinal loss.

Assadi (92) also noted that Urinary K/C ratio was effective in guiding in differentiating renal and extra renal loss of potassium thus helping in management of hypokalemia.

It was noted in our study that the amount of mean potassium required for treatment in HPP group was around 60 meql/L as compared to around 100 meql/L in non-HPP group which was statistically significant. It should be noted that less potassium is required for recovery in primary hypokalemic periodic paralysis.

The clinical implication is be cautious while using potassium in HPP as there are chances of rebound hyperkalaemia.

There was no relation found in amount of potassium required for treating weakness and the degree of neurological weakness that is present. In our study mean recovery was 26.77 hours among HPP group which was less as compared to 76.29 hours in Non HPP group and difference was statistically significant.

The attacks in Hypokalemic periodic paralysis usually recovery within few hours but some residual paralysis do remain which may take days to recover. The recovery time required for patients with secondary Hypokalemic periodic paralysis is larger than patients with primary hypokalemic periodic paralysis.

In similar study Maurya noted that recovery time was greater in patients with secondary hypokalemic periodic paralysis. Whether this increase recovery time is related to greater potassium loss(as compared to HPP), greater amount of potassium that is required for correction or ongoing renal loss of potassium is not known.

Two of our patients only had partial recovery on discharge but were of non HPP group. But partial recovery or residual weakness in postulated to occur in patient with primary hypokalemic periodic paralysis.

Six patients in our primary hypokalemic periodic paralysis group had more than two repeat attacks of motor weakness These patients were started on acetazolamide.

In similar study Griggs et al [13] found that in addition to eliminating attacks of weakness, acetazolamide also improved inter attack weakness in 8 of 10 affected patients. Similar findings were noted by Rao et alle, and Sushil et al [13]

Although the treatment of choice in periodic paralysis is generally considered to be acetazolamide, there is no standardised treatment regimen and no consensus as to when to start treatment. We do not know if acetazolamide treatment prevents any permanent weakness that may occur. We did not find other randomised or quasi-randomised studies, but only case reports and anecdotal articles using other drugs to reduce paralyses attacks .Further research is needed to determine the best treatment for reducing the frequency and severity of attacks and to treat or prevent permanent muscle weakness

The mechanism of action in HPP group is not known. It did not appear to affect serum potassium, total body potassium, total body sodium, blood pH, nor was the agent shown to affect muscle excitability directly. Acetazolamide is kaliuretic but usually prevents precipitation of attacks by induction of mild acidosis. Some patients have worsened on treatment with acetazolamide thus acetazolamide may not work in all patients in such patient's triamterene, spironolactone [1] both of which are potassium sparing diuretics. Dicholorophenamide is potent carbonic anhydrase inhibitor which can be used when acetazolamide does not bring improvement The exact dose of acetazolamide is not known but 250 mg thrice daily is optimal, the dose for dicholorophenamide is 50-150mg/day. Acetazolamide should not be given in patients with RTA, it may worsen RTA associated periodic paralysis.

Conclusion

1. Primary hypokalemic periodic paralysis is the most common cause of hypokalemic periodic paralysis. seen commonly in males. Commonest age of presentation been 18-25 years of age
2. Other patients were diagnosed as renal tubular acidosis, Gastrointestinal loss with hypokalemia, hypomagnesemia, Bartter's syndrome, Gitelman syndrome.
3. Paraperesis is commonest type of presenting weakness. Complete paralysis is rare in this patient. Respiratory weakness is rare.
4. Patients with hypokalemic paralysis usually have preceding symptoms of fatigue and myalgias. Hypokalemic paralysis can present without any premonitory complaints.
5. Weakness usually occurred early in the morning on getting up from sleep.
6. High carbohydrate diet is known to precipitate hypokalemic periodic paralysis, but the history may not be always present
7. ECG changes suggestive of hypokalemia are seen even with serum potassium levels of < 3.0 meq//L.
8. Hypokalemic periodic paralysis with acid base disturbances should always be investigated for secondary causes of hypokalemic periodic paralysis.
9. Genetic testing has no role in diagnosing patients with primary HPP

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