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Clinico-Etiological Evaluation of Hypokalemic Flaccid Paralysis in a Tertiary Care Hospital in Odisha

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Abstract

Introduction: Hypokalemic periodic paralysis also known as familial hypokalemic flaccid paralysis (HFP) is a rare and autosomal dominant channelopathy characterized by muscle weakness or paralysis when there is a fall in potassium levels in the blood.

Aim: To study the etiological profile of patients presenting with Hypokalemic flaccid paralysis.

Methodology: The study was conducted from August 2019 to July 2020 at Medicine Department and Neurology Department, SCB Medical College Hospital, Cuttack. In medical history of patient was observed weakness, thyroid disease, drug intake, diarrhoea, vomiting, hypertension, and renal disease. Nerve conduction study was done in all the patients of hypokalemic paralysis and the data was documented. All patients underwent: 12 lead Electrocardiogram, serum electrolytes, random blood sugar, Spot urine potassium, Arterial blood gas analysis and Early morning urinary pH. Patients with normal acid base status underwent thyroid function tests.

Results : In our study, most of our cases were seen in third and fourth decades of life with male preponderance. Most cases were detected in coastal area of our state where the land is fertile for agriculture with the principal food being carbohydrate in nature. Clinically majority of the patients presented with quadriparesis. Carbohydrate meal and exercise were important precipitating factors. The secondary etiological factors were more responsible for hypokalemic flaccid paralysis with thyrotoxicosis being the first cause. Less frequent variants like Gitelman syndrome, renal tubular acidosis, acute gastroenteritis, excess sweating and hypothyroidism were also detected. Dengue as a type of secondary etiological factor was found in our study. Severe form of hypokalemia was seen in most of the cases with muscle power grade zero to three. Serum hypomagnesemia was observed in Gitelman syndrome. ECG changes had no significant correlation with the clinical presentation of our cases. All our patients recovered within 24 hours without any casuality.

Conclusion: Hypokalemic flaccid paralysis is a treatable disease. Targeting the main aetiology stops upcoming difficulties. Also it is economical in the long run.

Index Terms— : Hypokalemic Flaccid Paralysis; Clinico-Etiological Evaluation; Muscle power**;** Tertiary Care Hospital in Odisha

1. INTRODUCTION

Today in medical science Hypokalemia is a common electrolyte disorder which is generally showing in hospitalized patient in medical. Various type of reason is responsible for this disorder; endocrine is one of the causes which are responsible for Hypokalaemia. The patient can need medical care when a patent with intracellular shift, increased potassium excretion and reduced intake. However many clinical statement and guideline refer to this disease, they do so, generally in the perspective of other clinical body.

The plasma potassium level is normally maintained within narrow limits (typically, 3.5 to 5.0 mEq per litre) by multiple mechanisms that collectively make up potassium homeostasis. Such strict regulation is essential for a broad array of vital physiologic processes, including the resting cellular membrane potential and the propagation of action potentials in neuronal, muscular, and cardiac tissue, along with hormone secretion and action, vascular tone, systemic blood-pressure control, gastrointestinal motility, acid–base homeostasis, glucose and insulin metabolism, mineralocorticoid action, renal concentrating ability, and fluid and electrolyte balance. [1]

In a healthy human body, potassium (K+) excretion is 90% by kidney and 10% by skin and intestine. In clinical observed that more than 98% K+ is intercellular. Generally total body K+ is function by kidney and keep potassium in hypokalemic conditions and evacuate potassium in hyperkalemic state.[2]

Hypokalemic Flaccid Paralysis (HFP) represents a group of disorders presenting with acute flaccid paralysis, with a documented hypokalemia (K+< 3.5mmol/L) during the episode and recovery following treatment. If it will not treated, this will transformed to secondary hypokalemia such as respiratory paralysis or cardiac arrhythmia which can be cause of morbidity and mortality. It can also be easily misdiagnosed as Guillain-Barre Syndrome, which leads to an incorrect management, which can lead to potentially life-threatening sequelae.

Thyrotoxic hypokalemic paralysis is a most of common disease in Asian population. Generally hypokalemic paralysis is divided into two types i.e. hypokalemic flaccid paralysis and paralysis which can be create due to lack of K+ or several additional aetiologies. [3] Acute attacks are often precipitated by rest after exercise, stress, or a high carbohydrate meal. The treatment of acute attacks is aimed

at restoring serum potassium levels into the normal range. This is best achieved by the oral administration of potassium. In addition, administration of a nonselective beta blocker is sometimes given in either familial or thyrotoxic periodic paralysis. [4]

There is only one report regarding clinical and aetiological spectrum of Hypokalemic flaccid paralysis in Western Odisha published J Assoc Physicians India. [5] According to previous case studies in literature, some patients may go on to develop permanent proximal muscle weakness after years with periodic paralysis.

In this study, we report etiological profile of patients presenting with Hypokalemic flaccid paralysis admitted at Medicine Department and Neurology Department, SCB Medical College Hospital, Cuttack. We also identify the differences in clinical parameters in relation to certain causes of the hypokalemic paralysis to guide a selection of appropriate treatment strategies.

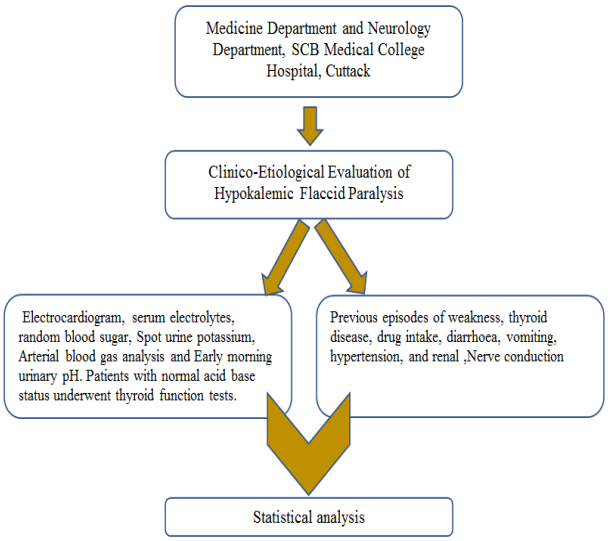

Figure.1. schematically presented clinico-etiological evaluation of Hypokalemic Flaccid paralysis in a Tertiary Care Hospital in Odisha

2. Materials And Methods

2.1. Patients and methods

The present study was done over a period of 12 months from August 2019 to July 2020 who was admitted to Medicine Department and Neurology Department, SCB Medical College Hospital, Cuttack. The patient suffering from acute flaccid weakness of two or more limbs without any sensory loss along with hypokalaemia i.e. serum potassium level less than 3.5 wmol/L was included in the study. History of episodes of similar illness in the past along with feature of thyroid disease, hypertension, renal disease, gastrointestinal disorder and associated treatment history was noted. Family history of muscle weakness were recorded, clinical examination of nervous system including muscle tone reflexes power were evaluated. The muscle power of scale 0 to 5 using medical research council was noted. Nerve condition study was done and observations were noted. Nerve conduction study was done in all the patients of hypokalemic paralysis and the data were documented. All patients underwent: 12 lead Electrocardiogram, serum electrolytes, random blood sugar, Spot urine potassium, Arterial blood gas analysis and early morning urinary pH.

Serum electrolyte was measured by ion selective electrode process and blood gas analyser was adapted to estimate arterial blood gas. Ion selective electrode method was adopted to estimate spot urinary potassium. The pH of urine was evaluated by pH meter. The patient with normal acid base status were subject to thyroid function test, thyroid stimulating hormone (THS) free triiodothyronine (FT3)and free thyroxine (FT4)were estimated by chemical urines screen.

Patients with urinary K+ loss and metabolic alkalosis without hypertension underwent 24 hrs urinary calcium which was done via Colorimetry. Patients with urinary K+ loss and metabolic alkalosis with hypertension underwent Ultrasound of abdomen and pelvis. Patients with urinary K+ loss and hyperchloraemic metabolic acidosis with normal anion gap underwent fasting Urinary pH as well as Ultrasound of abdomen and pelvis. Dengue NS1 was done in selected cases.

Hypokalemic flaccid paralysis (HFP) cases were diagnosed if spot urinary potassium excretion is < 20 mmol/L, in presence of hypokalemia and flaccid weakness with normal acid base status without any other causes.

Cases with flaccid weakness and hypokalemia with spot urine potassium excretion <20 mmol/L with increase in FT3 and/or FT4 with decrease in TSH having normal acid-base status, were diagnosed as thyrotoxic hypokalemic paralysis.

Patients of hypokalemic paralysis with low urinary potassium excretion (<20 mmol/L) with hypochloremic metabolic alkalosis with vomiting and excessive sweating were diagnosed as cases of HFP due to prior/non renal loss of potassium.

Patients with hypokalemia and flaccid weakness having low renal urinary potassium loss (<20 mmol/L) and hyperchloremic metabolic acidosis with diarrhoea is regarded as another cause of non-renal loss of potassium causing HFP.

Distal renal tubular acidosis (dRTA) was diagnosed by evaluating urinary potassium loss more than 20MMOL/l, fasting urine pH more than 5.5 with hyper chronic metabolic acidosis with normal anion gap in the absence of gastrointestinal loss. Abdominal USG and radiographic evaluation for detection nephrolithiasis supported the distal renal tubular acidosis.

The Gitelman syndrome was diagnosed with increase in urinary potassium loss more than 20mmol/L, hyperkalaemia, hypomagnesaemia, hypocalciuria and metabolic alkalosis with serum bicarbonate level more than 29 mmol/L.

2.2. Statistical analysis

Data analysis was done with SPSS version 20.0. The data were expressed as mean ± SD. Differences in group means were compared using one-way analysis of variance (ANOVA). Differences in categorical variables were compared using Fisher's exact test. The difference was considered significant if p-value was < 0.05.

3. RESULTS

3.1. Number of cases studies district wise

We have analysed 47 patients in our centre with hypokalemic flaccid paralysis. The mean age was 36.74 ± 12.18 years (range 16-76 years). 36 (76.6%) cases were male and 11 cases (23.4%) were female. Maximum cases (32 cases;68.08 %) were in the third and fourth decades of life with male preponderance. Maximum number of cases (61.7%) were encountered in the month of summer from March to June along with equal number of cases (9 each) in rain and winter. Seventeen cases (36.17%) were seen in coastal districts of Odisha; whereas maximum numbers (45 cases i.e., 95.7%) were seen in coastal and adjacent coastal districts. Anugul and Kandhamal districts are located in interior hilly areas with one case each figure.2.

3.2. Clinical presentation

The commonest clinical presentation was quadriparesis, found in 44 (93.6%) of total patients. Three patients (6.4%) presented with paraparesis. Deep tendon reflexes were preserved in 3 cases (3.68%), diminished in 31 cases (65.96%) and absent in 13 (26.66%) patients. Other presentations were neck muscle weakness in one case (2.13%), trunk muscle weakness in one case (2.13%). No cases had on presentation respiratory paralysis or cranial nerve abnormalities or sensory deficit or bladder and bowel involvement. Precipitating factors included heavy exertion in 27 (57.4%) patients, heavy carbohydrate meal in 11 (23.4%) of cases. In 7(14.8%) of total patients, we could not identify any precipitating factors . History of previous attack was present in 15 (31.9%) of cases. Majority of patients (68%) presented first time with features of hypokalemic flaccid paralysis figure.3.

Out of total 47(100%) patients maximum cases were thyrotoxicosis 23 (48.93%) followed by sporadic 8 cases (17.02%) and Gitelman 4 cases (8.15%). Familial and Dengue hypokalemic paralysis are 3 cases each alongwith one case each of Hypothyroidism, proximal RTA, distal RTA, distal RTA with Sjogren, Acute gastroenteritis and excess sweating figure.4.

Figure.2. District wise distribution of cases in Odisha

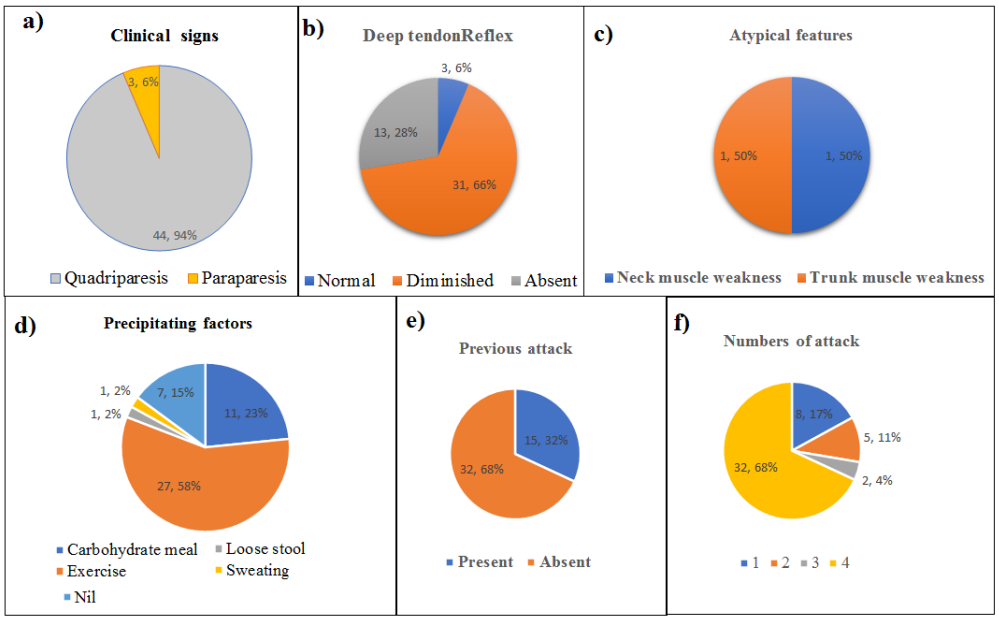

Figure.3. Clinical presentation of cases Hypokalemic flaccid paralysis

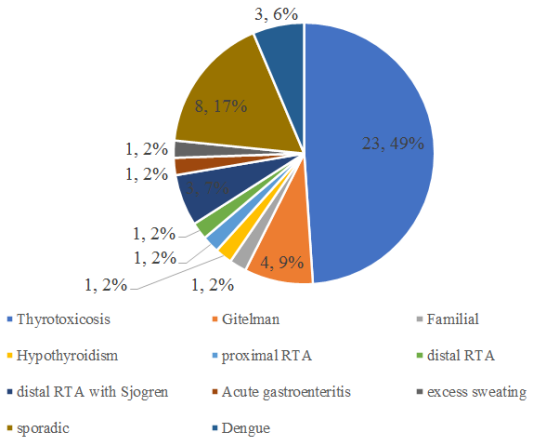

Figure.4. Distribution of patient's cases

3.3. Age-sex distribution of different cases

The diagnosis of hypokalemic flaccid paralysis (HFP) in 35 (74.47%) patients with trans cellular potassium shift. Sporadic periodic paralysis (SPP) were diagnosed in 8 (17.2%) cases and family periodic paralysis (FPP) were diagnosed in 3 (6.38%) patients. Twenty three patients (48.94%) were diagnosed as thyrotoxic periodic paralysis (TPP) and one case 2.13% of hypothyroidism was detect 7 patient (14.89%) presented with rend potassium loss whereas 5 patients presented with non-renal potassium loss (table-1 and table-5).

Table.1. Age-Sex distribution of different cases

Variables	Age (mean±SD (Range)	M:F	Total (%)
Patients with transcellular K^+ shift	38.31±13.84 (16-76)	27:8	35 (74.47)
Thyrotoxicosis	36.78±13.8 (16-76)	17:6	23 (48.94)
Hypothyroidism	65	1:0	1(2.13)
Sporadic	42.37±13.21 (23-64)	6:2	8 (17.02)
Familial	30.33±6.88 (20-36)	3:0	3(6.38)
Patients with renal potassium loss	34.85± 8.16 (22-55)	6:1	7(14.89)
Proximal RTA	40	1:0	1(2.13)
Distal RTA	55	1:0	1(2.13)
Distal RTA Sjogren	26	1:0	1(2.13)
Gitelman's	30.75± 5.75 (22-38)	3:1	4(8.51)
Patients with non-renal potassium loss	28.4± 3.12 (24-33)	3:2	5(10.64)
Acute gastroenteritis	24	1:0	1(2.13)
Excess sweating	30	0:1	1(2.13)
Dengue	29.33± 2.88 25-33	2:1	3(6.38)
Total	36 .74± 12. 18 (16-76)	36:11	47(100)

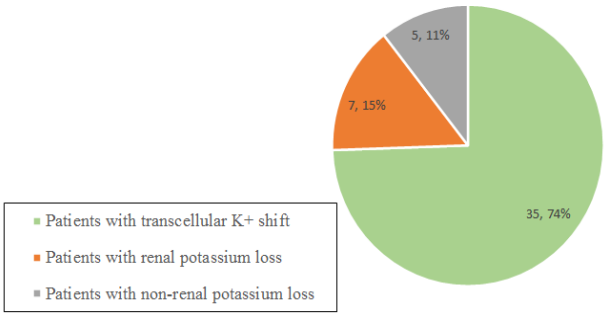

Figure.5. % of total distribution of different cases

3.4. Comparison between potassium shift and loss

There was a significant difference between serum potassium levels in the cases of hypokalemic paralysis due to causes with transcellular shift of potassium versus renal and non renal loss. Also, serum calcium, serum magnesium and urinary potassium showed significant difference. Serum CPK did not show any significant difference. Regarding muscle power as per MRC scale (0-5), there was significant difference between the two groups i.e. transcellular K+ shift (where blood pH is normal) and renal & non renal potassium loss. In our study serum CPK was raised in majority (61.7%) of cases. Serum potassium was lowest in Gitelman syndrome and Dengue. Serum magnesium is lowest in Gitelman syndrome and most in Dengue. Serum calcium was more in Gitelman syndrome than Dengue. Serum CPK was raised in almost all types of cases but highest in Dengue. Spot urinary potassium evaluation differentiated renal loss from non-renal loss table. 2.

Table. 2. Comparison between potassium shift and loss

Parameters	Transcellular K^+ shift (n=35) 74.5%	Renal and non renal K^+ loss (n=12) 25.5%	P value
Mean age(range)	38.31±13.84	32.16±6.69	P<
	(16-76)	(22 - 55)	0.1476
M:F	27:8	9:3	P<0.8797
Recovery time (Hrs)(mean±SD (Range)	11.6±4.25	13.33±3.66	P
	(4 -24)	(4 - 24)	<0.2152
Na^+(mmOI/L) (mean±SD (Range)	136.97 ± 3.45	136.66 ± 5.44	P<
	(125 - 144)	(124 - 150)	0.8191
K^+ (mmOI/L) (mean±SD (Range)	2.44 ± 0.51	2.1 ±0.43	P
	(1.5 - 3.4)	(1.2-3.2)	>0.0445
Cl^- (mmOI/L) (mean±SD (Range)	105.29±4.53	105.1±7.1	P<
	(97.2-118)	(97.2-125)	0.9147
pH (Blood) (mean±SD (Range)	7.39±0.01	7.38±0.09	P<
	(7.35-7.44)	7.15-7.48)	0.5130
Ca^{+2} (mg/dl) (mean±SD (Range)	9.08±0.26	9.61±0.26	P>0.0001
	(8.7-10.1)	(9.1-10.1)	
Mg^{+2} (mg/dl) (mean±SD (Range)	1.53±0.44	1.008±0.47	P
	0.6-2.1)	(0.2-2.2)	>0.0011
UK^+ (mmOI/L) (mean±SD (Range)	19.28±4.36	23.66±8.28	P >
	(11-28)	(10-38)	0.0234
Muscle power (MRC scale of 0 - 5)	3±0.57	3.08±0.61	P
			>0.0234
CPK(U/L)	578.48±439.98	601.33±366.16	P<
	(45-3133)	(109-2390)	0.8725

3.5. Muscle power versus hypokalemia

There was no correlation between the severity of hypokalemia and severity of weakness (p value=0.784) in figure.6 and table.3. Electrocardiograms showed U wave in 18 cases, followed by flattening of T waves in 10 cases, ST segment sagging observed in 8 patients, PR prolongation in 5 cases and one patient with ventricular ectopics.

While comparing the biochemical parameters between primary and secondary hypokalemic paralysis serum magnesium showed significant difference. There was no other significant difference in different parameters between the two groups.

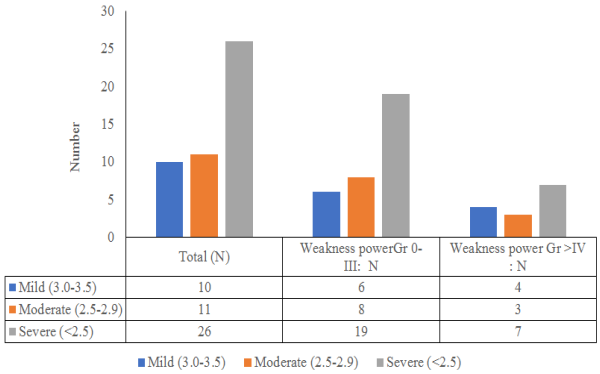

Figure.6. Muscle power versus Hypokalemia with Chi-Square: 0.633937 Degrees of Freedom:2 P<0.7284

Table.3. Muscle power versus Hypokalemia

Hypokalemia (mmol/L)	Total N(%)	Weakness powerGr 0-III N(%)	Weakness power Gr >IV N(%)
Mild (3.0-3.5)	10 (21.3%)	6	4
Moderate (2.5-2.9)	11 (23.4%)	8	3
Severe (<2.5)	26 (55.3%)	19	7
Total	47	33	14

3.6. Comparison of data between Primary and secondary Hypokalemic paralysis

Majority of patients (35) recovered within 6 to 18 hours. There was no mortality in any of our patients having hypokalemic flaccid paralysis. In this study, all the patients had recovered after initiation of appropriate therapy to the underlying causes in addition to potassium supplementation. [Table.4]

Table.4. Comparison of data between Primary and secondary Hypokalemic paralysis

Parameters	Primary N=11 (23.4%)	Secondary N=36 (76.6%)	P value
Mean age(range)	39.09±11.93	36.02±12.14	P <0.4650
	(20-64)	(16-76)	
M:F	9:2	27:9	P<0.6402
Recovery time(Hrs)	10.18±3.83	12.61±4.15	P <0.0908
(mean±SD (Range)	(4-18)	(4-24)	
Na^{+}(mmOI/L)	138.18±2.89	136.5±4.22	P <0.2249
(mean±SD (Range)	133-144)	(124-150)	
K^{+} (mmOI/L) (mean±SD	2.6±0.44	2.28±0.5	P< 0.0630
(Range)	(1.6-3.4)	(1.2-3.4)	
Cl^{-} (mmOI/L) (mean±SD	107.18±3.98	104.65±5.32	P <0.1531
(Range)	(99-118)	(97.2-125)	
pH (Blood) (mean±SD	7.39±0.02	7.39±0.04	P <1.0000
(Range)	(7.35-7.44)	(7.15-7.48)	
Ca^{+2} (mg/dl) (mean±SD	9.06±0.25	9.26±0.36	P< 0.0934
(Range)	(8.7-9.8)	(8.8-10.1)	
Mg^{+2} (mg/dl) (mean±SD	1.88±0.08	1.06±0.43	P >0.0001
(Range)	(1.7-2)	(0.2-2.2)	
UK^{+} (mmOI/L)	19.27±4.42	21.68±6.61	P< 0.2645
(mean±SD (Range)	11-28)	(10-38)	
Muscle power (MRC scale of 0 - 5)	3.18±0.59	2.97±0.59	P< 0.3071
CPK (U/L)	771.27±570.66	527.19±356.14	P<0.0936
	(45-2740)	(45-3133)	

4. Discussion

In a study from Western Odisha, Mohapatra et al found more cases in males and the patients were between the ages of 18 to 56 years. [5] In the present study, over a period of 12 months 47 cases of HFP were detected. Maximum cases (68.8%) where detected in the 3rd and 4th decade of life. Also, in our study male patients outnumbered female patients. A study from northern India also reported the name. Probably this male predominance is due to outward activities and excess exposure to heat and exertion.

We observed a seasonal variation in our patient with hypokalaemia flaccid paralysis HFP highest 58% number of cases during summer season (March to June) during which the external temperature range from 35 to 40^{0}C . Odisha located in humid subtropical climate expenenes with on average humidity of about 70% in summer which favours high prevalence of patients with hypokalaemia during summer season. Our observation was at par with a similar study published from western Odisha [5] where it was noted the high prevalence of HFP in summer season. Studies from outside India have not discussed about the impact of seasonal variation with respect of presentation of such paralysis of episodes [2].

Most of our cases (95.7%) were from coastal and adjacent coastal area. The coastal area, being a fertile land with rice as the principal carbohydrate diet for the people, who are busy in their agricultural work in the open field throughout the year, particularly in summer season for which they suffer more from this disease. These findings also correlated with the study from Western Odisha.[5] In a study from Lucknow by Maurya et al found more cases of quadriparesis. [6] The most common presentation (93.62% cases) in our study was also quadriparesis. Nerve condition study was normal in all our cases. Generalized areflexia was noted in 13 (26.66%) patients and hypoactive deep tendon reflexes were noted in 31 patients acute onset of flaccid paralysis and generalized areflexa one may misdiagnosis with Guillain barre syndrome (GBS). However, progression of muscle weakness in HFP is very rapid in comparison to GBS. In HFP supplementation of potassium the recovery is faster than GBS.

Trunk muscle weakness and early neck muscle weakness were some of the typical manifestations are may be due to failure of nerve impulse transmission in synaptic junctions due to hypokalaemia.[5] Bladder, bowel and cranial nerve involvement were not observed in our patient.

A study from West Bengal with 50 patients, Bhattacharya et al., [7] reported high carbohydrate diet as the common precipitating factor. High carbohydrate intake was also the most common precipitating factor in most of our cases having such paralytic attacks as rice was the staple food in Eastern India. In 23.4% of our cases had carbohydrate diet as precipitating factor. This is because carbohydrate rich diet stimulates more insulin secretion and in turn high serum insulin facilitates intercellular potassium shift which facilitate hypokalaemia flaccid paralysis.

We observed less numbers (31.9%) of cases with history of previous attacks. The study from Western Odisha [5] stated that only 6% cases had similar attacks in the past. Regarding number of attacks, majority of our patients (68%) had presented for the first time.

We have observed in eaten Odisha 47 cases within a span of 12 months as observed by another study in western Odisha [5]. However a study from Northern India by

Mayurya et.al [6] describes less number of cases of HFP during the specific time frame.

We had encountered 11 (23.4%) primary and 36(76.6%) secondary hypokalemic paralysis. Out of primary type familial hypokalemic periodic paralysis occurred in 6.38% of patients; whereas, sporadic hypokalemic periodic paralysis was of 17.02% of the cases. When we analyse secondary type of hypokalemic paralysis, thyrotoxic paralysis was the commonest cause in 48.93% cases which is the most common cause in our region. The earlier Indian studies from Northern India (16.7%) [6] and Western Odisha (22%) [5] have shown variable incidence of thyrotoxic paralysis. A study by Kamath et al. from Karnataka detected 4 cases of thyrotoxicosis out of 9 cases presented as secondary hypokalemic paralysis. [8] Studies outside India within the Asian subcontinent from South Korea by Wi et al. and from UAE by Alkaabi et al. with 34 and 17 patients respectively also reported thyrotoxicosis to be the predominant aetiology. [4,9]

Gitelman syndrome was diagnosed in 8.15 % of cases. In a large study with 200 patients from West Bengal, Patra et al. reported 56 (28%) patients with Gitelman Syndrome. [2]

In a study with 11 patients from Japan, Hiraga et al. reported 4 cases of acute gastroenteritis [10]. Other non-renal / renal causes of hypokalemic flaccid paralysis associated with acid-base disorders such as acute gastroenteritis and excessive sweating were present in our study. Both sub groups had shared one patient (2.13%) each. One case was detected as hypokalemic paralysis secondary to hypothyroidism. We have encountered 3 cases (6.38%) of Dengue as one of the secondary aetiology of hypokalemic flaccid paralysis. The study by Kamath et al. from South India [8] also presented 5 cases of dengue as one of the etiological factors of their secondary hypokalemic paralysis. In a study by Rao et al. from Vellore with 31 patients maximum cases presented with renal tubular acidosis. In our study we encountered 2 cases of distal RTA and 1 case of proximal RTA. Of the two cases of distal RTA, 1 case was diagnosed as Sjogren 's syndrome. Sjogren's syndrome is complicated by RTA in 40% of patients.[11] In a study from North India with 40 patients, Singh et al reported rise in levels of serum CPK. [12] However, the study from Western Odisha observed a significant rise of CPK level between the secondary and primary groups. This could be due to more awareness of disease among the primary cases in that region.

In our study serum Creatinine Phospho Kinase (CPK) was evaluated in 61.7% of cases, which indicate damage to muscle membrane while comparing the primary group with secondary group of HFP there was no significant correlation in the value of serum CPK probably the hypokalaemia produces muscle ischemia resulting is rise of serum CPK.

A study by Garg et al from KGMU also reported rise in serum CPK among the patients in their study.[13]

In our study, 35 (74.5%) patients with hypokalemic paralysis had a cause of transcellular shift of potassium for their condition and rest 12 (25.5%) patients were having renal and non-renal loss of potassium. In the study from Northern India with 31 patients Maurya et al reported a significant difference in severity of weakness and severity of hypokalemia.[6] In a series of case reports from Taiwan, Lin SH et al reported no correlation between severity of weakness and hypokalemia.[8] In our study, we could not find any correlation between severity of hypokalemia and severity of weakness. This could be due to difference in negative potassium balance in different patients and variation in treatment protocol.

Interestingly, the prototype change of hypokalemia, i.e., appearance of "U" waves, was present only in 38.2% of the participants. This finding is similar to the observations of S. Patra et al. [2] Regarding serum magnesium value, we have observed a significant

difference between primary and secondary hypokalemic paralysis.

In our study the mean recovery time was 6 to 8 hours and all our patients recovered with treatment, however in 6BS recovery time make take several months and about 5% of patients may die during acute illness. In GBS potassium supplementation may not have any impact but in HFP complete recovery is possible with potassium therapy. Therefore differential of HFP from GBS is significant in a clinical setup.

While comparing the etiological types of hypokalemic flaccid paralysis the primary hypokalemic paralysis are less in number in comparison to the secondary type in our study which is at par with other authors.[2,5] However some authors observed a rise in primary hypokalemic paralysis than secondary type. [6,14] Out of secondary hypokalemic paralysis we observed highest number in the individual suffering from thyrotoxicosis which is at par with Vidyasagar et al. of Andhra Pradesh.[15] In our series 4 cases of Gitelman syndrome were detected. Similar observations were noted by Kayal et al. of Assam, B.N. Mohapatra et al. of Burla. However, more number of cases was documented by S. Patra et al. of

Medinapur. [2,5,6,14] It is documented that in primary HFP there is intracellular shift of potassium resulting in hypokalaemia where is in secondary HFP there is depletion of total body potassium. Therefore in primary HFP with smaller amount of potassium supplementation the recovery is early compared to the secondary group. In the present study, there was no mortality because timely diagnosis and adequate treatment resulted in complete recovery.

5. Limitation Of The Study:

This study is a hospital-based study. So actual data in the community regarding hypokalemia may not reflect the true picture. Also, in hospital-based studies there is always a chance of bias, that is the subjects may not ideally represent the population. Epidemiological studies are required in the community for an unbiased estimate. Sporadic cases need further genetic studies to determine molecular abnormalities, if any.

6. Conclusion

Hypokalaemia flaccid paralysis, a treatable disease, presenting with acute neuromuscular symptoms, should be dealt properly with Arterial blood gas analysis and spot urinary potassium assay along with other routine investigations without depending heavily on ECG and

nerve conduction studies. However thyroid function test should be done even if there are no clinical signs in favour of hyperthyroidism or hypothyroidism as all cases of hypokalemic flaccid paralysis are not periodic paralysis. Thus a timely approach to evaluate the hypokalaemia flaccid paralysis (HFP) patient adequate potassium supplementation and management of precipitating factors will prevent this life threating dyselelectrolylemia and complication.

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