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DEMOGRAPHIC AND CLINICAL PROFILE OF HYPOKALEMIC PARALYSIS IN PATIENTS OF TERTIARY CARE HOSPITAL IN SOUTH INDIA

Internal Medicine

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ABSTRACT

Introduction: Hypokalemic paralysis is an important reversible cause of acute flaccid paralysis. Severe hypokalemia if not managed appropriately can lead to cardiac arrhythmia, respiratory failure and cardiac arrest. Therefore, this study was conducted with the objective of finding the causes of hypokalemic paralysis in the study population. **Materials and Methods:** A cross sectional study was done from January 2019 to January 2021 in the patients admitted in the Department of Nephrology, Thanjavur Medical College, Tamil Nadu. Systematic random sampling method was followed and every fifth patient admitted with documented serum potassium levels of <3.5mEq/L and acute onset of flaccid weakness was included in the study till a minimum sample size of 50 was reached. **Results:** Most of the study participants, 16 (32%) had dRTA (distal renal tubular acidosis) followed by SPP (sporadic periodic paralysi) in 14 (28%). Gitelman syndrome (GS) was found in 9 (18%), Bartter syndrome (BS) in 4 (8%), thyrotoxic periodic paralysis (TPP) in 4 (8%) and proximal renal tubular acidosis (pRTA) in 3 (6%) participants. **Conclusion:** Renal loss of potassium (distal renal tubular acidosis) was the most common cause for hypokalemic paralysis. Early recognition as well as prompt treatment of hypokalemic paralysis will reduce the morbidity and mortality.

KEYWORDS

Hypokalemic paralysis, renal loss, potassium, renal tubular acidosis, South India

INTRODUCTION:

Hypokalemic paralysis is an emergency condition, with features of acute flaccid paralysis and the documentation of hypokalemia. Patients will recover without any neurological sequelae if it is diagnosed and treated at the earliest. Cardiac arrhythmia, respiratory failure and cardiac arrest can occur if it is left untreated [1].

Hypokalemic paralysis is classified into periodic and non-periodic paralysis. HPP (Hypokalemic periodic paralysis) is characterized by transcellular shift of potassium with normal acid-base balance. The causes of HPP includes sporadic, thyrotoxic and familial. Hypokalemic non-periodic paralysis results from renal and extra renal loss of potassium with acid-base disturbance [2].

The initial treatment of hypokalemic paralysis patients includes replacement of potassium and finding the underlying etiology. Further treatment depends on the cause of hypokalemia, severity and duration of symptoms and illness[3].

Therefore, this study was conducted with the objective of finding the causes of hypokalemic paralysis in our region. The metabolic and clinical profile of the study participants were also analyzed that will help in the diagnosis, management and outcome in hypokalemic paralysis patients.

MATERIALS AND METHODS:

Study Design:
A cross sectional study was conducted from January 2019 to January 2021 in the patients admitted in the Department of Nephrology, Thanjavur Medical College, Tamil Nadu. Systematic random sampling method was followed and every 5th patient was included in the study till a minimum sample size of 50 was reached.

Study Participants:
Inclusion Criteria:
Patients admitted with acute onset of flaccid weakness and documented serum potassium levels of <3.5mEq/L

Exclusion Criteria:
1. Patients with renal failure (serum creatinine >1.5mg/dl)
2. Patients who were on diuretic drugs

Data Collection:
It included demographic profile, history of evaluation of symptoms, precipitating factors like high carbohydrate intake and heavy exercise in the preceding 24hrs, similar episodes in other family members, history of thyroid disease, hypertension, vomiting, diarrhea, dry eyes, dry mouth and kidney disease were recorded. History of drugs intake like diuretics, β2 agonists and insulin were also noted. The physical examination included pulse, blood pressure, thyroid and complete neurological examination. Laboratory investigations include urine analysis, complete blood count, blood sugar, blood urea, serum creatinine, serum electrolytes, arterial blood gas(ABG), electrocardiogram and thyroid function tests.

Data Analysis:
Data was entered in the excel spread sheet and variables were coded accordingly. The statistical analyses were performed using Graph pad Prism version 5 software. Fisher's exact test was used to compare the frequencies between the groups. Unpaired t test was used to compare the mean between two groups and one way analysis of variance (ANOVA) was used in case of more than two groups. P value of less than 0.05 was considered to be statistically significant.

Ethical Issues:
The Institutional Ethical Committee (IEC) of Thanjavur Medical College, Thanjavur gave approval for the study protocol. All the study participants gave informed written consent in their local language for taking part in the study.

RESULTS:

Age And Sex Distribution:
Among 50 study participants, 30 (60%) were males and 20 (40%) were females. The mean age of the study population was about 33.1 years with a standard deviation of 6.2 years (range 20-45 years).

Clinical Profile:
Among 50 study participants, 35 patients (70%) had1st episode of hypokalemic paralysis while 12 patients (24%) had two episodes and 3 patients (6%) had three episodes. Respiratory paralysis was observed in 3 (6%) individuals. When the ABG profiles were analyzed, 18 (36%) patients with normal acid base balance and urine spot potassium:creatinine ratio<13mEq/g were considered to have HPP.

Among remaining 32 (64%) patients with evidence of renal potassium loss (urine spot potassium:creatinine ratio >13mEq/g), 19 (38%) had metabolic acidosis and 13 (26%) had metabolic alkalosis, dRTA was the most common disorder observed in 16 (32%) followed by SPP in 14 (28%) individuals. Gitelman syndrome was found in 9 (18%), Bartter syndrome in 4 (8%), TPP in 4 (8%) and pRTA in 3 (6%) participants respectively. Males and females were in equal proportion in SPP. In GS, females are affected more than males whereas in all other groups (TPP, BS, dRTA, and pRTA) males are affected more than females.

The etiological groups of hypokalemic paralysis were compared with different serum parameters using one way ANOVA test. The results are given in the table 1.

Table 1: Comparison of various parameters between the different etiological groups of hypokalemic paralysis observed in the study (n =50)

Variable	Etiology of hypokalemic paralysis												Fvalue	Pvalue
	SPP (n=14)		TPP (n=4)		GS (n=9)		BS (n=4)		dRTA (n=16)		pRTA (n=3)			
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD		
Age in years	38.4	3.7	31.5	1.7	29.8	6.5	24	1.6	33.7	5.5	28	2	7.93	<0.001*
Number of episodes	1.3	0.6	1.7	0.9	1.3	0.5	1.3	0.5	1.4	0.6	1	0	0.662	0.654
Sr K+ (mmol/L)	2.4	0.3	2.5	0.1	2.3	0.3	2.3	0.3	2.3	0.3	2.4	1	0.317	0.901
Sr Na+ (mmol/L)	139	3	141	3	138	2.6	142	2.2	137	2.2	141	4.3	3.08	0.018*
Sr Ca2+ (mg/dL)	9.6	0.41	9.5	0.3	9.4	0.5	9.6	0.3	9.3	0.4	9.5	0.2	0.746	0.593
Sr Mg2+(mg/dL)	2.2	0.5	2.1	0.09	1.3	0.13	2.1	0.24	1.9	0.23	1.9	0	9.21	<0.001*
Sr PO4-(mg/dL)	3.6	0.28	2.2	0.21	3.5	0.18	3.5	0.27	3.6	0.22	2.2	0.2	31.4	<0.001*
Sr Cl-(mmol/L)	99	3.9	102.5	3.4	102.7	2.4	103	3.2	111	3.2	116	7.2	24.8	<0.001*
Sr RBS (mg/dl)	95.6	9.7	89	8.8	91.8	11.5	87.3	8.9	90.4	7.8	112.6	6.4	3.68	0.007*
Blood urea (mg/dl)	34.9	2.5	27.5	1.9	32	4.4	33	4.7	35.8	5.8	39	1	3.67	0.006*
Sr Creat (mg/dL)	0.9	0.2	1.1	0.1	0.9	0.16	0.9	0.08	0.89	0.13	0.96	0.15	1.37	0.251
pH	7.4	0.02	7.39	0.02	7.49	0.01	7.5	0.01	7.23	0.04	7.3	0.02	114.3	<0.001*
Sr HCO3-(mmol/L)	23.7	1.8	23.7	1.7	30.6	1.9	35.7	2.6	13.7	3.3	15	3	86.1	<0.001*
pCO2	40.8	2.7	39.2	2.5	45.3	3.9	49.7	1.7	26.4	5.3	30.3	3.8	42.7	<0.001*
Urine spot K+ (mEq/L)	9.7	2.2	9	0.8	19.5	2.7	23.3	0.9	26.9	5	35.3	4.2	56.6	<0.001*
Urine spot Creatinine (mg/dL)	7.4	1.8	7.1	0.1	5.7	0.8	7.2	0.6	6.4	0.6	8.4	0.8	4.21	0.003*
Urine spot K+/Cr (mEq/g)	11.3	0.07	11.2	0.05	13.4	0.39	13.2	0.4	14.3	0.76	14.1	0.21	69.4	<0.001*

*Significant p value

Urinary spot calcium levels were analyzed in Gitelman and Bartter syndrome and their mean (SD) levels were 12.8 (1.9) and 27.6 (1.3) respectively. The urinary calcium levels were significantly elevated in Barter's syndrome (P = 0.001). However, there was no statistically significant difference in urinary chloride levels. Urinary calcium creatine ratio was greater than 0.3 in Bartter syndrome and <0.2 in Gitelman syndrome.

DISCUSSION:

In this study about hypokalemic paralysis, distal RTA with 16 (32%) participants was the common cause followed by SPP (sporadic periodic paralysis) with 14 (28%) participants. Gitelman syndrome (GS) was observed in 9 (18%) individuals while BS (Bartter syndrome) and TPP (Thyrotoxic Periodic Paralysis) each were seen in 4 (8%) individuals. Proximal RTA was seen in 3 (6%) study participants. Respiratory failure was seen in 3(6%) participants. Similar results were obtained in a study conducted by Lin SH et al. in Taiwan in the year 2001 among 97 patients. In their study, TPP was found in 39 (40%), SPP in 29 (30%), Bartter's/Gitelman in 6 (6%), distal RTA in 6 (6%) and primary hyperaldosteronism (PH) in 6 (6%) participants [4]. In studies done in North India, most of them had secondary causes for hypokalemic paralysis[5,6].

In a study conducted by Rao et al in South India, 2 had SPP and TPP, 13 (42%) had dRTA, 1 (3%) had Gitelman syndrome and 13 (42%) had Primary Hyperaldosteronism [7]. Maurya et al in their study among 30 patients, found that 17 (56.7%) had primary idiopathic hypokalemic paralysis, 5 (16.7%) had TPP, 4 (13.3%) had RTA and 4 (13.3%) had Gitelman syndrome[8].

In most Asian studies, TPP was the major cause whereas in Caucasians, FPP is more common among hypokalemic paralysis patients. In our study, distal RTA was the commonest group and no FPP was reported. Rao et al. in their study found that HP due to Primary Hyperaldosteronism (PH) and d RTA are the most common causes. But in our study none of the study participants had PH[7].

In our study, 14 patients had sporadic periodic paralysis. This accounted for 38 percent of the total patients. But none of them had familial history of hypokalemic periodic paralysis. The male:female ratio was equal with 7 both in male and female respectively. The mean age at onset was on the higher side with 38 years when compared with the patients with other diagnosis.

Thyrotoxic paralysis was observed in 4 (8%) individuals. The mean age of onset was around 31 years with male to female ratio of 3:1 which coincided with the result of the various other studies where there was a male preponderance. Thyroid stimulating hormone (TSH) values were extremely low with spot urine potassium:creatinine ratio less than 13mEq/g. Similar results were also obtained in a study conducted in China by Ko et al[9].

Distal renal tubular acidosis turned out to be the major cause of hypokalemic paralysis in our study. About 16 patients had distal RTA and 3 patients had proximal RTA. The mean age of onset was 31 years and showed a predominant male preponderance. All these patients had

their urine potassium:creatinine ratio above 13mEq/g and ABG showed metabolic acidosis. Bagga et al in their study also obtained similar results[10].

CONCLUSION:

Renal loss of potassium (distal renal tubular acidosis) was the most common cause for hypokalemic paralysis. Early recognition as well as prompt treatment of hypokalemic paralysis will reduce the morbidity and mortality.

Declarations:
Funding: None

Conflict Of Interest: None

Ethical Approval:
The study protocol was given approval by the institutional ethical committee (IEC), Thanjavur Medical College, Thanjavur, Tamil Nadu.

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