A Clinical Study of Hypokalemic Periodic Paralysis

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ABSTRACT

Introduction: Hypokalemic paralysis is life threatening emergency which requires urgent treatment along with a rapid evaluation to determine the underlying cause. Current study aimed to observe the clinical presentation, ECG changes and response to treatment in patients with hypokalemic periodic paralysis

Material and methods: All the patients in the age group 18-65 years who were diagnosed with HPP (K< 3.5mEq/L) were included. In this study 34 patients had hypokalemia and 7 patients had normal potassium level. All cases of the episodic periodic paralysis admitted in the medical wards form the material of the study. All cases were subjected to detailed history, clinical examination and laboratory examination. E.C.G. and serum electrolytes were carried out on 1st day, 2nd day and 3rd day.

Results: Incidence of hypokalemic periodic paralysis is 0.40% of medical admissions and 0.16% of total admissions. Incidence is high in females i.e., 24 (71%) patients out of 34 are suffering from hypokalemic periodic paralysis, 21-30 yrs age group is most common age affected in present study. Most of the patients in study are effected for the first episode of hypokalemic periodic paralysis i.e., 79.4%. Most of the case are predisposed after exercise. Duration of illness is more than 48 hrs in most of the cases. There were 25 cases with serum potassium level between 2.6 to 3 meq/L, out of which 18 cases had quadriplegia and 7 had paraplegia.

Conclusion: Early recognition and prompt management of this condition will give good result

Keywords: Hypokalemic Periodic Paralysis

INTRODUCTION

The underlying etiologies of hypokalemia can be classified in two major categories: 1) acute conditions that cause the intracellular shifting of potassium without total body potassium depletion; and 2) total body potassium loss via excessive renal potassium excretion or extra-renal potassium loss due to vomiting or diarrhea. It is a known cause of hypokalemic paralysis due to the transcellular shift of potassium, whereas distal renal tubular acidosis (dRTA) is an important cause of hypokalemic paralysis due to excessive renal potassium loss. As the clinical presentation of hypokalemic paralysis secondary to dRTA can be similar to HPP, the evaluation of the acid-base status and the urine anion gap is pivotal in the differentiation of these two diseases. The failure to differentiate dRTA from hypokalemic periodic paralysis may result in improper management, which can lead to the development of potentially life threatening conditions.¹

In this institution, MGM Hospital, Warangal catering to the medical needs of rural people of four Telangana districts, it is a common clinical finding to see patients presenting with para and quadriplegia and responding to oral administration of potassium chloride, thus fitting into the clinical study of periodic paralysis. With the application of modern molecular biological techniques, major advances have taken place in the diagnosis of muscle diseases. Newer discoveries led to the definition of the new categories making hypokalemic periodic paralysis in the category of channelopathies.²

Even though latest investigation is far from needs a pure clinical study of this particular clinical problem is undertaken in this institution. So here we study the patterns of clinical presentation, electrocardiographic changes and response to treatment in patients with hypokalemic periodic paralysis.

MATERIAL AND METHODS

Current study was done in in M.G.M hospital, Warangal. All the patients in the age group 18-65 years who were diagnosed with HPP (K< 3.5mEq/L) were included in the study. 22, 626 cases were admitted as inpatients in MGM Hospital, Warangal from June 2011 to July 2016. Out of which 8467 were admitted to six medical units. During the same period 41 cases of periodic paralysis were admitted. Out of 41 cases only 34 patients had hypokalemia and 7 patients had normal potassium level.

All cases of the episodic periodic paralysis admitted in the medical wards form the material of the study. All cases were subjected to detailed history, clinical examination and laboratory examination. E.C.G. and serum electrolytes were carried out on 1st day, 2nd day and 3rd day. The treatment and prognosis were analyzed. All the patients who were admitted with sudden onset of weakness of extremities in this institution were taken for clinical study. Immediately after admission, ECG was taken for all the patients and serum electrolytes were estimated. E.C.G and serum electrolytes were carried out on 1st day, 2nd day and 3rd day and at the time of discharge. Serum electrolytes were assessed on 2nd day, 3rd day and at the time of discharge and assessed the prognosis of patients.

The cases which were not treated prior to the admission by general practitioners were only taken in this study and

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detailed examination was done as per the proforma.

**RESULTS**

In our M.G.Mhospital, Warangal, 41 cases of periodic paralysis were admitted, out of which 34 cases were hypokalemic periodic paralysis and 7 cases were normokalemic periodic paralysis. The incidence of hypokalemic periodic paralysis was 0.40% of medical admissions and 0.16% of total admissions (table-1).

Incidence is high in females ie 24 (71%) patients out of 34 are suffering from hypokalemic periodic paralysis (figure-1). 21-30 yrs age group is most common age affected in the present study (figure-2). Most of the patients in this study presented as the first episode of hypokalemic periodic paralysis {27 (79.4%)} (table-2). Exercise is the most common predisposing factor in most of the cases. Duration of illness is more in 24 to 48 hrs (table-3). There were 25 cases with serum potassium level between 2.6 to 3 meq/L, out of which 18 cases had quadriplegia and 7 had paraplegia (table-4).

There was no mortality in patients having hypokalemic periodic paralysis. All the patients have recovered after initiation of appropriate therapy to the underlying causes in addition to potassium supplementation.

**DISCUSSION**

In this Northern Telangana region, it is not an uncommon finding to see patients with hypokalemic periodic paralysis. There are few reports of both familial and sporadic hypokalemic periodic paralysis in Indian journals. Though
sporadic cases of periodic paralysis are described, the disease is hereditary in 81% of the cases and males are affected three times more than females. In this study all the 34 cases of hypokalemic periodic paralysis are sporadic. None of these cases revealed the occurrence of similar neurological problem in other members of the family.9

In the present series of 34 cases there are 10 male patients and 24 female patients (Figure-1). There is high incidence of sporadic hypokalemic periodic paralysis among females. The attacks of hypokalemic periodic paralysis first begin in the second decade and are more frequent between the age of 20-35 yrs (Brain-Mc-Ardle text book of skeletal muscle disorders - Sir John - Walton). In this series the highest incidence of hypokalemic periodic paralysis is observed during third decade i.e., between the age group of 21-30 yrs and constitute about 70% (Figure-2).

In both male and females, the highest incidence is found in the third decade. Table – 2 given below depicts the number of attacks the patients had prior to the admission. 27 patients were presented with first attack (79.4%), 6 patients with second attacks (17.1%) and only one case with repeated bouts (Table-2).3

The most important predisposing factors of hypokalemic periodic paralysis are rest after exercise, heavy meal a few hours before, anxiety, emotion and cold. There are 20(58.8%) cases in this study who were laborers developed paralysis during early hours after a few hours of sleep and following severe exercise a day before. Four cases had gastroenteritis followed by paralysis. Two cases developed hypokalemic periodic paralysis after consuming toddy. No predisposing factor was found in the remaining 5 cases (Table-3).

The duration of paralysis in moderate to severe cases of hypokalemic periodic paralysis was reported as 24-36 hours and in few cases the paralysis was persisted for 2-3 days. In this study, none of the 34 cases were recovered within 24 hours. Among 26 cases the paralysis lasted for more than 24 hours and recovered within 48 hours (Table-3). Only 8 cases took more than 48 hours for recovery.6

In this series of 34 cases, 24 cases were presented with quadriplegia of sudden onset and was started with tingling and numbness in the feet and then developed weakness of all four limbs. Out of 34 cases, 10 cases presented with paraplegia and 24 presented with quadriplegia. The weakness was marked in proximal group of muscles. None of these cases had cranial nerve involvement, intellectual disturbances, bowel and bladder disturbances.

The serum potassium level in all the cases of hypokalemic periodic paralysis during the attack was low and ranged from 1.6 to 3.0 meq/L. Most of the cases had serum potassium levels between 2.6 to 3.0 meq/L. The serum potassium level returned to normal in most of the cases within 48 hours. One case was admitted for paraplegia and had very low serum potassium level (1.6 meq/L) whereas the other cases who had quadriplegia with zero power in all four limbs, the serum potassium level was 3.1 meq/L (Table-4).

There were 25 cases with serum potassium level between 2.6 to 3 meq/L. Out of which 18 cases had quadriplegia and 7 had paraplegia. These findings in hypokalemic periodic paralysis does not correlate with the serum potassium levels. The ECG changes in hypokalemic periodic paralysis show prominent U waves. Flattening of the T waves, prolongation of the PR interval, QRS and QT intervals and depression of the ST segment. Bradycardia is a common feature.

All the cases showed ST depression and prominent U waves. In only one cases the PR interval was prolonged to more than 0.20 seconds. There was no increase in the QRS interval and no arrhythmias were found. Bradycardia was found in 75% of the cases which reverted back with recovery. Brain MC ardle reported bradycardia as a common finding in hypokalemic periodic paralysis.7

All the cases studied were sporadic cases and none of these cases showed any evidence of thyroid disease, renal disease and hepatic abnormalities. Blood sugar levels were within normal limits in all the cases. In only one case, there was history of diarrhea which might have precipitated the attack. None of these cases showed any evidence of muscular dystrophy or cardiomyopathy.

Many other studies are done earlier from Kashmir which reported 21 cases of hypokalemic paralysis secondary to distal renal tubular acidosis (RTA) over a period of 8 years. 16 cases in the south Indian study, RTA was the cause in 13 out of 31 (42%) cases of HPP.9 In another study, RTA was the cause of HPP in 4 out of 30 (13.3%) cases; 3 cases were of distal RTA and only 1 case had proximal RTA.10

Hypokalemic periodic paralysis though common among Indian population varies greatly in disease spectrum and magnitude in our country due to the heterogeneous pattern of etiology behind it. Two case series that studied hypokalemic periodic paralysis in tertiary care centres of India have observed that around 45% of all those patients had a secondary cause for their condition and this secondary group had more severe hypokalemia that needed longer time to recover.9,10 Thyrotoxicosis, renal tubular acidosis, Gitelman's syndrome, and primary hyperaldosteronism were among the prime conditions leading to hypokalemic periodic paralysis but no case of hypothyroidism was found to be the etiology behind it.

CONCLUSION

All the cases of hypokalemic periodic paralysis are sporadic. There is highest incidence of hypokalemic paralysis among females compared to males (5:2). Decreased potassium levels were observed in all the cases (1.5-3.5 meq/L). ST-segment depression and prominent U waves were observed in all the cases. Bradycardia was present in 75% of the cases. No correlation was found between the serum potassium level and the severity of the paralysis. Thyroid, renal, hepatic and other systemic disease were not found to be associated with hypokalemic periodic paralysis. All the cases improved with oral potassium therapy. Early recognition and prompt management of this condition will give gratifying results.
REFERENCES


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