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Etiological, Clinical and Metabolic Profile of Non-familial Hypokalemic Paralysis – A Study in a Tertiary Care Centre.

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ABSTRACT

Background: Hypokalemic Periodic Paralysis, a skeletal muscle disorder, is characterized by muscle weakness often due to lowering of potassium levels in the body. The cause could be due defect in voltage gated calcium or sodium channels caused by genetic defect, thyroid dysfunction, and other secondary reasons. High carbohydrate diet, exercise, alcoholism is known to trigger the symptoms. **Methods:** 35 patients with non-familial periodic paralysis occurring during summer months in Hyderabad and the neighboring areas were considered in the study. The precipitating factors, onset of symptoms, the type of motor weakness were all taken into account. Biochemical tests like Potassium levels and creatinine phosphokinase were also done. **Results:** Female preponderance was observes with the prominent age group being 20 -30 years. Most of the patients had sudden onset of symptoms (in less than 6 hours) with all patients having the involvement of the axial, proximal and distal muscles. Hypotonia was seen in 71% of the patients. More than 90% of the patients had lowered potassium levels while majority of them had normal CPK levels. Carbohydrate meal, rest after exercise and alcohol were found to be the most common predisposing factors. **Conclusion:** As a significant number of people have non-familial and sporadic and potentially reversible causes of hypokalemic paralysis, a proper and detailed work up must be done for other causes of HPP other than familial. Carbohydrate meal, exercise and alcoholism were observed to be the most common causes. Administration and elevation of potassium levels normally reverses the situation.

Keywords: Carbohydrate meal, Hypokalaemic periodic paralysis, Hypotonia, Low potassium levels, Muscle weakness.

INTRODUCTION

The Hypokalemic Periodic Paralysis (HPP) is primarily the disorder of skeletal muscles characterized by transient attacks of muscle weakness and often associated with alteration. Other organs including cardiac and smooth muscle are not affected by this disease.

HPP is a relatively uncommon but potentially life threatening clinical syndrome. Most of the times, it is a very rare autosomal disease leading to paralysis of skeletal muscles and muscle weakness often associated with lowering of potassium levels in the blood. This may be primarily due to defect in voltage gated calcium or sodium channels. The symptoms of this mutation very often are observed in adolescence where they may be triggered by physical exercise followed by rest, meals high in carbohydrates and sodium, changes in temperature, excitement, noise etc. Weakness may be mild to total body paralysis. The attacks may last for a few hours to several days.

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Some of the attacks may lead to a chronic muscle weakness later in life.[1,2] Hypokalemic paralysis may also occur because of thyrotoxicosis (TPP). Biochemical hyperthyroidism with normal urinary potassium excretion and ECG changes are characteristic of TPP. Thyrotoxic periodic paralysis (TPP) is more commonly reported in Asians.[3] It has also been reported from the western countries as a result of migration of different ethnic populations.[4]

There are some sporadic cases which are clinically indistinguishable from the familial HPP. These are non-familial hypokalemic paralysis. The trigger for this is also high carbohydrate diet, rest and exercise. The typical attack come in during rest after exercise and a diet rich in carbohydrates. The distribution of the paralysis varies, with limbs affected earlier and often more severely than trunk muscles with proximal more susceptible than the distal ones. Attack of paralysis tend to occur every few weeks and lessen in frequency with advancing age.[3]

Unlike other forms of periodic paralysis, persons with congenital hypokalemic periodic paralysis have normal thyroid function. But they have a very low blood level of potassium during episodes of weakness.[5]

Failure to differentiate between HPP and other causes of severe hypokalemia with weakness in the emergency room may lead to errors in management. When serum potassium levels are normalized, the patient usually recovers.

This study was conducted to study the causes of non-familial, sporadic hypokalemic paralysis, its clinical presentation and the biochemical levels in the patients.

MATERIALS AND METHODS

We had done a retrospective analysis of 35 patients who had muscle tone weakness and were suspected of hypokalemic periodic paralysis. The study was performed at Malla Reddy institute of Medical Sciences, from July 2012 to June 2015. Records of all the hypokalemic paralytic patients were analyzed. Routine tests were done on all the patients which included complete blood counts, hemoglobin, ESR, complete urine analysis, blood sugar, Blood urea, serum creatinine, serum potassium, sodium, and ECG. Motor conduction studies were done on median, ulnar, peroneal and tibial nerves on both sides. Sensory conduction studies were performed on median and ulnar nerves on both sides. Minimal latencies of 'F' response were studied in the median and ulnar nerves using conventional procedures. Electromyography was done with concentric needle.
Depending on the condition of the patients, electrophysiological exam was done in all patients within 48 hours of admission.

RESULTS

Most of the 35 cases occurred in the months of May and June. Females were slightly more affected than males [Figure 1], while the majority of the age group affected was between 20 – 30 years [Figure 2].
Sudden onset of symptoms had a preponderance over gradual or at night time occurrence [Figure 3].
The presenting symptoms were weakness of all the four limbs, both proximal and distal and also the weakness of the axial muscles without any cranial nerve involvement. There was no respiratory insufficiency [Table 1].

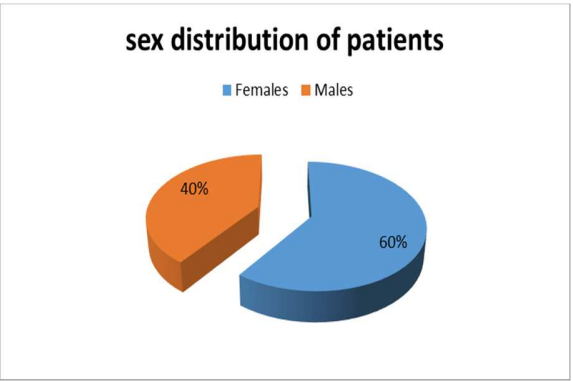

Figure 1: Sex wise distribution of patients

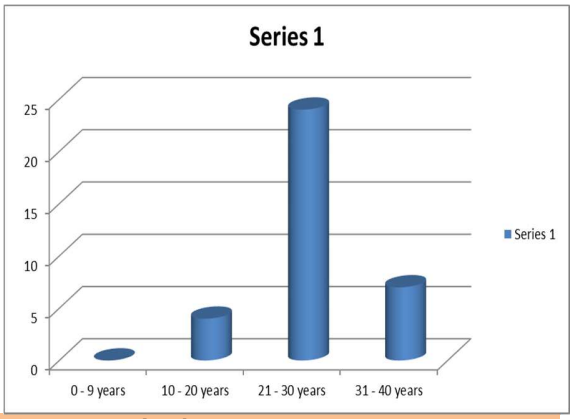

Figure 2: Age distribution.

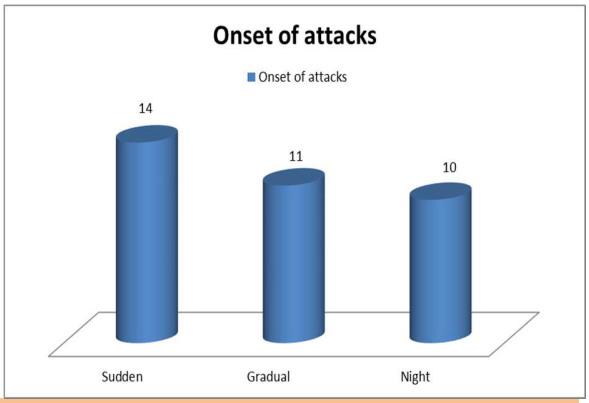

Figure 3: Onset of attacks

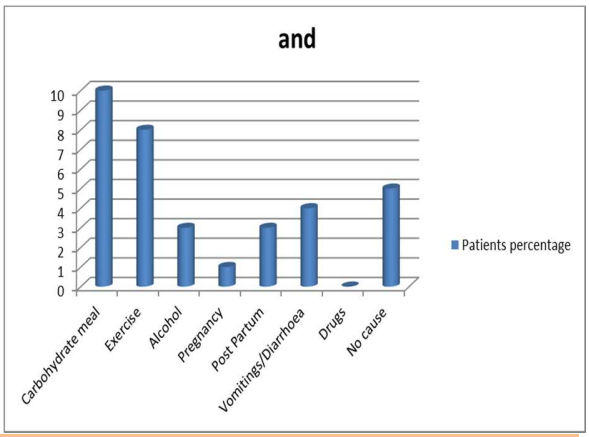

Figure 4: Precipitating factors

Most of the patients showed symptoms of weakness within 6 hrs. Neck drop was seen on 17% of the cases while all the patients showed trunk, upper and lower limb weakness. 71% of the patients had hypotonia. Reflexes were sluggish in 60% of the cases while 20% of them had brisk reactions. 20% were normal [Table 2].

The precipitating factors for paralysis was high carbohydrate meal, alcohol, vomiting, and diarrhea. In women, pregnancy was one of the disposing factor. None of the patients had a history of taking drugs like diuretics, laxatives etc. Secondary causes of hypokalemia were ruled out as the renal parameters for all the patients was normal [Figure 4].

Almost 90% of the cases had low potassium levels while in 8% of the cases, the potassium level was normal. Normal CPK levels were seen among most of the patients [Table 3].

Table 1: Motor weakness

Muscles Involved	No of Patients	Percentage
Neck Muscles	6	17.1%
Axial	35	100%
Proximal	35	100%
Distal	35	100%
Respiratory	0	0

Table 2: Motor System

Features		No of Patients	Percentage
Bulk	Normal	35	100
Tone	Normal	10	29
	Hypotonia	25	71
Power	Upper limbs (2-3/5)	35	100
	Lower Limbs(0-2/5)	35	100
Reflexes	Normal	7	20
	Sluggish	21	60
	Brisk	7	20

Table: 3 Serum potassium and CPK levels

Serum Potassium level	2.5-3.0 mcg/L	18	51.4%
(Normal – 3.6-5.5mcg/L	3.1--3.5 mcg/L	14	40%
	3.6--4.0 mcg/L	3	8.6%
CPK (Normal 24 – 195 U/L	>200 U/L/L	11	31.4%
	<200 U	24	68.6%

DISCUSSION

It was in 1727, that the first case of Hypokalemic Periodic Paralysis was diagnosed by Musgrave.[7] Subsequently, one of the largest studies was conducted in Taiwan, where 97 cases were reported over a period of 10 years.[8] In India, Agarwal et al., reported 40 cases of HPP over a period of 23 years[9] while in South India, 31 patients were reported over a period of 30 years.[10] In a more recent study by Maurya et al. 30 patients over a period of 3 years were reported[11], while 56 patients over a period of 24 months were diagnosed with HPP by Ashok K Kayal et al.[6]

Of the 35 cases in our study, we noticed a predominance of summer months especially during May and June for the prevalence of hypokalemic paralysis. This could be due to the atmospheric conditions like high temperatures, low humidity associated with electrolyte imbalance making the patients more susceptible to attacks of paralysis. The mean age of the patients was 24.6 years with a slight preponderance of females to males. Majority of the patients were in the age group of 20 to 30 years. But, in a similar study conducted by Kayal AK et al., though prevalence of HPP in summer season was observed, similar to our study, there was a predominance of male patients to female patients.[6] This phenomenon was observed by Pal et al., also.[12]

The main precipitating factors for HPP in our study were found to be high carbohydrate diet, exercise and alcohol. The presenting symptoms were weakness of all the limbs, and axial muscles without the involvement of the cranial nerves. Triggers such as rest after exercise and carbohydrate-rich meals, was reported as one of the important cause of HPP.[13] PK Pal et al reported 10 hours to 7 days for onset of symptoms which slowly progressed towards quadri-paresis with some of the patients having multiple attacks of paralysis.[12] In some of the other studies, dengue was found to be the cause of HPP[13,14] , while in some other studies, renal tubular acidosis was found to be common.[10,11] Thyrotoxic periodic paralysis was also not uncommon especially in the Asian countries.[8].

The onset of the attack was sudden (within 6 hours), gradual or occurred during the night time. The most common symptom was neck drop in 16% of the patients, 71% had hypotonia. All the patients had muscle weakness in upper and lower limbs and most of them had sluggish reflexes.

Slow reflexes were observed in 27% of the patients in a study by PK Maurya et al.[11]

None of our patients have any history of taking drugs like diuretics or laxatives. Renal parameters were normal in all the cases as a result, no secondary causes of HPP were observed. None of them had any family members suffering from the same disease.

Lowered potassium levels were observed in more than 90% of the cases while CPK levels were in the normal range in 69% of the cases. Similar results were observed in other studies like Pal et al, Maurya et al, Garg et al.[11-13]

All patients received oral potassium chloride (10ml) three times a day and 10 patients received oral acetazolamide (DIAMOX) tablets 250mg twice a day. They all recovered and were able to walk within 4 days of hospitalization. There were no deaths neither due to acute cardiac dilatation nor respiratory paralysis.

CONCLUSION

A significant number of people have non familial with recurrent attacks, and potentially reversible causes of hypokalemic periodic paralysis. So a proper and detailed work up must be done for other causes of HPP other than familial. Carbohydrate meal, exercise and alcoholism were observed to be the most common causes. The patients respond to oral potassium without any residual weakness.

REFERENCES

1. Saperstein DS. Muscle channelopathies. Seminars in Neurology 2008; 28 (2): 260-9.
2. Jacob OL. Practical aspects in the management of hypokalemic periodic paralysis. Journal of Translational Medicine 2008;6:18.
3. Okinaka S, Shizume K, Iino S. The association of periodic paralysis and hyperthyroidism in Japan. J Clin Endocrinol Metabol 1957;17(12):1454–9.
4. Ober KP. Thyrotoxic periodic paralysis in the United States. Report of 7 cases and review of the literature. Medicine 1992;71(3):109–20.
5. Mount DB, Zandi-Nejad K. Disorders of potassium balance. In Taal MW, Chertow GM, Marsden PA, et al., eds.Brenner and Rector's The Kidney, 1998, pp 245-56.
6. Ashok K, Kayal G, Das M, Jain R. Clinical and biochemical spectrum of hypokalemic paralysis in North: East India. Ann Indian Acad Neurol.2013;16(2):211–7.
7. Musgrave W. A periodic palsy. The philosophical transactions and collections to the end of the year. Biol Sci. 1700;2:33–4.
8. Lin SH, Lin YF, Halperin ML. Hypokalemia and paralysis. QJM. 2001;94(3):133-9.
9. Agarwal AK, Singla S, Singla S, Kumar A. Hypokalemic Paralysis: Emergency Room Management. In: Agarwal AK, editor. Medicine Update. Vol 19(Part I) Noida: Association of Physicians of India; 2009. pp. 42–9.
10. Rao N, John M, Thomas N, Rajaratnam S, Seshadri MS. Etiological, clinical and metabolic profile of hypokalemic periodic paralysis in adults: a single-centre experience. Natl Med J India. 2006;19(5):246-9.
11. Maurya PK, Kalita J, Misra UK Spectrum of hypokalemic periodic paralysis in a tertiary care center in India. Postgrad Med J. 2010;86(1022):692-5.
12. Pal PK, Vengamma B. Atypical neurological manifestations of hypokalemia. Ann Indian Acad Neurol. 2004;7:375–80
13. Garg RK, Malhotra HS, Verma R, Sharma P, Singh MK. Etiological spectrum of hypokalemic paralysis: A retrospective analysis of 29 patients Ann Indian Acad Neurol. 2013;16(3):365–70.
14. Jha S, Ansari MK Dengue infection causing acute hypokalemic quadriparesis. Neurol India. 2010;58(4):592-4.

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