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*Original Article*

# *Clinical Profile in Hypokalemic Periodic Paralysis Cases*

M Rajesh Kumar[1], RV Bharath[1], P Rammohan[2], Amit Agrawal[1]

***ABSTRACT***

*The present article was aimed to study demographic and clinical pattern, periodicity and precipitating events for hypokalemic paralysis and to assess the response to treatment both during acute attacks and as prophylaxis in comparison with available literature. Forty patients with hypokalemic paralysis were admitted in Narayana Medical College and Hospital during the last two years, in all the medical units and neurology wards. Patients were assessed clinically, with symptomatology and precipitating factors were evaluated. There were total 40 patients in the present study. Younger more including male gender. Mean age was 30.95 years. Common precipitating factor were diarrhea, fever, strenuous activity, following dextrose administration, in patients with diabetic ketoacidosis, however in majority of the cases we could not identify the precipitating factors. Most common electrocardiographic change on ECG was U' wave (36 cases) followed by flat 'T' wave (14 patients) and ECG was normal in 4 patients. Hypokalemic paralysis was predominantly seen in younger males. Vomiting, diarrhea were important precipitating factors. The response to oral potassium chloride supplementation was good as only few patients requiring intravenous potassium chloride. The approach to hypokalemic paralysis patient includes a vigorous search for the underlying etiology and potassium replacement therapy.*

***Key words:*** *Hypokalaemia, hypokalaemic periodic paralysis, periodic paralysis, paralysis*

## *Hipokalemik Periyodik Paraliz Hastalarında Klinik Görünüm*

***ÖZET***

*Bu makale hipokalemik paralizide demografi, klinik tipler, yineleme sıklığı ve presipite eden faktörlerin değerlendirilmesi ve hem akut ataklar sırasındaki tedavinin ve hem de profilaksinin etkinliğinin mevcut bilgilerle kıyaslanması amacını gütmektedir. Son iki yıl içerisinde Narayana Tıp Koleji'ne ve Hastanesine; tüm tıbbi birimler ve nöroloji servisine toplam 40 hipokalemik paralizili hasta başvurmuştur. Hastalar semptomatoloji ve presipite eden faktörler açısından klnik olarak değerlendirilmişlerdir. Erkekler ve genç yaştakiler daha fazladır. Ortalama yaş 30.95 dir. Sık presipite eden faktörler ishal, ateş, ağır egzersiz ve diyabetik ketoasidozda dekstroz infüzyonudur, bununla birlikte çoğu olguda böyle bir faktör tespit edilememiştir. En sık EKG bulgusu U dalgasıdır (36 olgu), sonra düz T dalgasıdır (14 olgu), 4 hastada ise EKG normaldir. Hipokalemik paralizi başlıca genç bayanlarda görülmüştür. Kusma ve diyare önemli presipite eden faktörlerdir. Oral potasyum klorüre cevap iyidir sadece az sayıda hastada intravenöz potasyum klorür gerekmiştir. Hipokalemik paraliziye yaklaşım altta yatan etyoloji açısından detaylı araştırma ve potasyum replasmanıdır.*

***Anahtar kelimeler:*** *Hipokalemi, hipokalemik periyodik paralizi, periyodik paralizi, paralizi*

[1]*Department of Medicine, Narayana Medical College Hospital, Chintharedypalem, Nellore, Andhra Pradesh, India.* [2]*Department of Pharmacology, Narayana Medical College Hospital, ChintharedyPalem, Nellore, Andhra Pradesh, India.*

***Correspondence:*** *Dr M Rajesh Kumar (MD)*
*Associate Professor of Medicine, Department of Medicine Narayna Medical College Hospital Chintharedypalem Nellore PIN-524003 AP, India*
*Email- rajeshkumarmeriga@gmail.com Mobile- +91-9440760435*

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## INTRODUCTION

Weakness is a common, albeit non-specific, presentation the differential diagnosis for the complaint of weakness is extensive. Acute hypokalaemic paralysis is a rare but treatable cause of acute weakness, characterized by acute attacks of muscle weakness of variable duration and severity and low serum potassium . Characteristically serum potassium content decreases during an attack was reported by Biemond and Dan¬iels in 1934. These disorders are amenable to treatment and progressive weakness can be prevented or even reversed (1-9). The present article was aimed to study demographic and clinical pattern, periodicity and precipitating events for hypokalemic paralysis and to assess the response to treatment both during acute attacks and as prophylaxis in comparison with available literature.

## MATERIALS AND METHODS

Forty patients with hypokalemic paralysis were admitted in Narayana Medical College and Hospital from January 2008 to December 2009. Demographic details including age, gender, occupation of all the patients were recorded. Patients were assessed clinically, with symptomatology and precipitating factors were evaluated. Precipitating factors and recurrence of attacks of paralysis in the same individual, and frequency of attacks were taken into consideration for all the patients. The clinical diagnosis of hypokalemic paralysis was suspected and the following investigations were done to confirm the diagnosis. The blood and urine analysis was done with special emphasis on serum electrolytes and urine potassium. Arterial blood gas analysis was done. In all cases electrocardiogram was taken, and the changes were studied. The response of therapy with oral potassium chloride is studied in all the above forty cases and also the time taken to regain normal muscle power after starting oral/intravenous (IV) potassium chloride (KCl) therapy was studied. The other details regarding history and clinical examination of the patients are recorded as per the proforma. All the patients were followed up to one year.

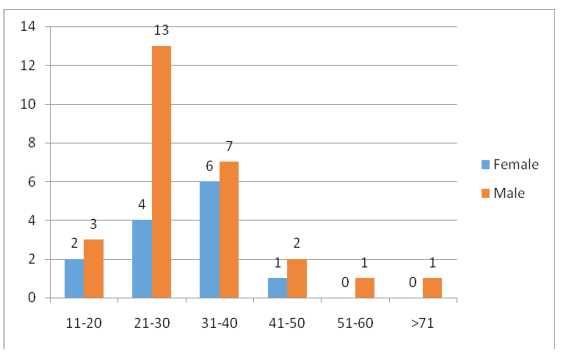


***Figure 1**. Age and sex distribution*

## RESULTS

There were total 40 patients in the present study. Younger more including male gender (Figure 1). Mean age was 30.95 years (minimum 14 maximum 7 range 59 years SD±10.51). All the patients had weakness of all four limbs without cranial nerve involvement. Sixteen patients had muscle pain and five patients had muscle cramps at the time of presentation. Common precipitating factor were diarrhea, fever, strenuous activity, following dextrose administration, in patients with diabetic ketoacidosis, however in majority of the cases we could not identify the precipitating factors (Table 1). Most of the patients seek medical attention for first episode of paralysis. Thirteen patients had recurrent episodes of paralysis. In some cases there were secondary causes for hypokalemic paralysis (renal tubular acidosis- 5 (12.5%) cases, diabetes-1 (2.5%) case, lymphoma-1 (2.5%) case and primary hyperaldosteronism in 1 (2.5%) case (2.5%). Most of the patients responded well to oral

***Table 1**. Showing clinical details*

| Precipitating factor | Male | Female |
|---|---|---|
| Diarrhea | 3(7.5%) | 2(10%) |
| Fever | 1(2.5%) | 2(10%) |
| Following strenuous activity | 2(5%) | 0 |
| Following iv 5% dextrose | 1(2.5%) | 0 |
| Diabetic ketoacidosis | 1(2.5%) | 0 |
| No precipitating factor | 18(45%) | 9(22.5%) |
| **Number of attacks** | | |
| Single | 21(52.5%) | 6(15%) |
| 2 to 3 | 4(10%) | 5(12.5%) |
| > 3 | 2(5%) | 2(5%) |
| **Secondary causes** | | |
| Renal tubular acidosis | 1(2.5%) | 4(10%) |
| Diabetes | 1(2.5%) | 0 |
| Lymphoma | 1(2.5%) | 0 |
| Primary hyperaldosteronism | 0 | 1(2.5%) |
| **Response to potassium** | | |
| Oral KCl | 21(%) | 11(%) |
| IV KCl | 6(%) | 2(5%) |

*Table 2. Showing investigation details*

| **Serum potassium values** (meq/dL) | **Patients** |
|---|---|
| 3.1 - 3.5 | 7(17.5%) |
| 2.6 - 3.0 | 11(22.5%) |
| 2.1 - 2.5 | 13(32.5%) |
| < 2 | 9(22.5%) |
| **Urine Potassium Levels** (meq/L) | |
| < 20 | 34(85%) |
| > 20 | 6(15%) |
| **Serum Magnesium Levels** (mg/dL) | |
| > 1.6 | 38(95%) |
| < 1.6 | 2(5%) |
| **Electrocardiographic Changes** | |
| U' wave | 36(90%) |
| Flat 'T' wave | 14(35%) |
| ST segment depression | 12(30%) |
| Bradycardia | 8(20%) |
| Tachycardia | 3(7.5%) |
| First degree AV block | 4(10%) |
| Ventricular ectopics | 5(12.5%) |
| Ventricular tachycardia | 1(2.5%) |
| Normal | 4(10%) |

potassium supplementation (32, 80% patients) and in 8 (20%) patients' intravenous potassium supplementation was required (Table 1). Details of serum potassium values are shown in Table 2. Urine potassium was less than 20 meq/L in 34 (85%) cases and more than 20 in 6 (15%) cases. Serum Magnesium Levels were more than > 1.6 mg/dL in 38 patients and < 1.6 in mg/dL 2 patients. Most common electrocardiographic change on ECG was U' wave (36, 90% cases) followed by flat 'T' wave (14, 35% patients) and ECG was normal in 4 (10%) patients (Table-2).

## DISCUSSION

Familial hypokalemic paralysis is the most common cause of acute hypokalaemic paralysis, but other uncommon causes include heavy carbohydrate meal, followed by a period of rest, anxiety and exposure to cold, thyrotoxic periodic paralysis, barium poisoning, renal tubular acidosis, primary hyperaldosteronism, licorice ingestion, and gastrointestinal potassium losses (2,10-22). Acute hypokalemic periodic paralysis can be primary or familial periodic paralysis, and secondary periodic paralysis. Primary or familial periodic paralysis is a group of disorders due to single gene mutation resulting in abnormalities of calcium, sodium, potassium, and chloride channels on muscle cell - membrane (9,18). In acute hypokalemic paralysis, the muscular weakness can occur in association with hypokalaemia, normokalaemia or hyperkalaemia (2). The exact underlying pathophysiology behind the paralysis is not well understood (2,3,5,23). Hypokalaemic paralysis may result from alteration in transcellular distribu¬tion of potassium or may be due actual potassium depletion from renal or extrarenal losses (2,3). The acute hypokalemic paralysis is characterized by attacks of reversible flaccid paralysis with concomitant hypokalemia (24). Characteristically the onset is noted in adoles¬cence, the attacks may range from a mild weakness of a single muscle group to rare complete quadriplegia with respiratory paralysis and death with cranial nerves sparing (21,25-27). Low serum potas¬sium coupled with high muscle potassium levels produce hyperpolarisation of the muscle membrane, making it inexcitable (23). Tendon reflexes may be decreased or absent but sensation is generally intact and the cardinal laboratory manifestation is a serum potassium of less than 3.5 mmol/l during an attack, although it can be much lower (28). The acute hypokalemic paralysis needs to be differentiated from many other causes of acute systemic weakness in the emergency department including neurologic, metabolic, and infectious etiologies (1,2,5,22).

The constant association of a fall in concentra¬tion of serum potassium with attacks of muscular weakness, and the relief of the weakness by the administration of potassium salts suggests hypokalemic paralysis (29) During an acute attack, fibers in affected muscles are electrically inexcitable (30). The episodes of hypokalemic paralysis more commonly occur in males than females. The prominent ECG changes of hypokalemia include U waves, ST segment depression, bradycardia and first degree heart block (22,31). Early diagnosis and treatment with oral potassium chloride in hypokalemic paralytic patient, can help to regain normal muscle power. Potassium can be given orally for attack prophylaxis and should be taken with ample volumes of water (22). Intravenous potassium is indicated for arrhythmia due to hypokalemia or airway compromise due to ictal dysphagia or accessory respiratory muscle paralysis (22). Avoidance of precipitating factors (eating a high carbohydrate load and vigorous activity or at the start of an episode of weakness) can help to prevent the episodes of paralysis(19,22).

Hypokalemic paralysis was predominantly seen in younger males. Vomiting, diarrhea were important precipitating factors. The response to oral potassium chlo-

ride supplementation was good as only few patients requiring intravenous potassium chloride. The approach to hypokalemic paralysis patient includes a vigorous search for the underlying etiology and potassium replacement therapy.

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