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Original Research Article

Clinical and Etiological Profile of Hypokalemic Paralysis

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Abstract:

Introduction: Hypokalemia is one of the most commonly encountered fluid and electrolyte abnormalities in clinical medicine. Muscle weakness secondary to low serum potassium levels is characteristically seen in Hypokalemic paralysis (HP). This heterogenous syndrome can be due to skeletal muscle channelopathies, thyrotoxicosis or various conditions causing excessive loss of potassium (renal or extra renal causes). With appropriate therapy, most cases respond rapidly and often show a complete recovery.

Materials and Methods: This was a prospective observational study, where patients with hypokalemia and acute weakness presenting to a tertiary care center were included in the study. A total of 43 patients were incorporated and their clinico etiological profile was assessed.

Results: The mean (± standard deviation) age of cases (n=43) was observed to be 29.18(± 10.23) years, with a range of 11 to 63 years. The study group was observed to comprise of 26 (60.4%) males and 17 (39.5%) females. The mean (± standard deviation) serum potassium level of patients at baseline was 2.19(±0.54) with a range between 1.3 mEq /L and 3.1 mEq/L. None of the patients died during hospital stay. The etiological profile revealed causes as Acute gastroenteritis in 15(34.8%), Renal tubular acidosis in 3 (6.9%), diuretic use in 3 (6.9%), Bartter syndrome in 2 (4.65%), Gitelman syndrome in 2 (4.65%), hypothyroid in 2(4.65%) , Thyrotoxic Periodic Paralysis in 4 (9.3%), Cushing's syndrome in 1 (2.3%), Diabetic Ketoacidosis in 3(6.9%) and Primary hypokalemic periodic paralysis in 3(6.9%).

Conclusion: It is imperative for an astute clinician to have an understanding of this extensive syndrome, for acute management and to enable complete unraveling of the underlying pathophysiology and diagnosis with a goal of preventing further attacks and improve quality of life in most patients.

Keywords: Hypokalemia, Paralysis, Potassium

Introduction

Hypokalemia is conventionally defined as a serum potassium ion concentration (K+) less than 3.5 mEq/L. It can manifest with neuromuscular weakness, renal abnormalities, glucose intolerance or life-threatening cardiovascular abnormalities. An almost 10-fold increase in mortality is observed in admitted patients with low potassium levels. Hypokalemia is commonly a result of excess loss, via renal or non-renal routes. Additionally a shift of the K+ from extracellular fluid (ECF) to intracellular fluid (ICF) can cause low serum levels of potassium, also called 'redistributive hypokalemia [1, 2].

Hypokalemic paralysis is characterized by hypokalemia associated with systemic weakness [3]. This clinical syndrome consists of a heterogenous group of disorders which are broadly classified as either Hypokalemic Periodic Paralysis (HypoPP) when the hypokalemia is due to redistribution, or as Hypokalemic non-periodic paralysis (non-HypoPP) where the hypokalemia is secondary to excessive loss of K+ [4].

Primary or hereditary HypoPP are due to genetic channelopathies in skeletal muscle membranes, causing episodic muscle weakness. They are inherited as autosomal dominant in most cases, but are a result of de novo mutations in about a third of cases. Secondary or acquired causes of HyoPP include thyrotoxic periodic paralysis (TPP), which is common in Asian population compared to the former, inherited periodic paralysis, seen more commonly in Caucasians. [3,5] Redistributive hypokalemia resulting in paralysis can also be caused by excess β2 agonist use or long term insulin use. Hypokalemic paralysis can be seen in wide range of conditions like barium poisoning, endocrinopathies (Cushing syndrome, hyperaldosteronism, Liddell syndrome), renal disorders (Bartter syndrome, Gitelman syndrome, Renal tubular acidosis), persistent vomiting with/without diarrhea, diuretic use and also viral infections (dengue and chikungunya virus) [6-9].

Early recognition and management of Hypokalemic paralysis is essential, as the recovery of weakness is usually complete with appropriate therapy in the majority of cases, while a delay in diagnosis or treatment could be fatal. HPP should be managed with small doses of potassium chloride as the hypokalemia does not reflect true K depletion in the body, and aggressive excess replacement can result in iatrogenic hyperkalemia [3]. On the contrary, patients with non-HPP require large doses of KCl to correct the hypokalemia and weakness [10]. Thus, a thorough awareness of the various causes of this heterogeneous syndrome will enable the clinician to initiate therapy at the earliest, prevent morbidity, mortality and also facilitate a diligent workup for the underlying pathophysiological abnormality to help prognosticate the patient and initiate prophylactic therapy as warranted. The correction of hypokalemia can be simple, but if performed inappropriately can lead to worsening symptoms, and even death [11].

Materials and Methods

The present study included patients admitted with a diagnosis of acute paralysis due to hypokalemia (serum potassium level less than 3.5 mEq/L) and weakness involving two or more limbs. Patients with CNS causes of quadriparesis (GBS, Acute transverse myelitis, stroke)were excluded, and thus the clinico etiological profile was studied for 43 patients. Complete clinical examination including assessment of muscle power and GBS disability scoring was performed in patients included in the study. The following investigations were also sent: ECG, Serum electrolytes, ABG, Spot urine potassium creatinine ratio, 24hr urine potassium ratio, Urine osmolality, serum osmolality, 24hr urine chloride levels, Urine calcium/creatinine ratio, Serum Aldosterone, Renin, Cortisol, magnesium, and thyroid profile was assessed Patients with mild to moderate hypokalemia were managed with oral potassium supplementation; patients with severe hypokalemia (serum potassium <2 meq/L) or patients unable to take oral supplementation or patients with potentially severe disease were treated with intravenous therapy. Based on severity of hypokalemia patients received either oralor intravenous potassium replacement. Patients of thyrotoxicosis were additionally treated with standard of care treatment.

Results

The mean (±standard deviation) age of cases (n=43) was observed to be 29.18(±10.23) years. The age of patients ranged from a minimum of 11 years to a maximum of 63 years. The study group was observed to comprise of 26 (60.4%) males and 17 (39.5%) females. Three patients had a positive family history of similar episodes. Past history of attacks of weakness was present in 15 (34.3%) patients, and 30 (69.7%) patients had some precipitating factors prior to the present attack of acute hypokalemic paralysis. The precipitating factors identified in patients were diarrhoea in 15 patients, profuse sweating in 5 patients, and intake of heavy carbohydrate meal in 4 patients. A history of strenuous exercise was present in 4 patients, while 2 patients had discontinued insulin recently. The etiologic profile of the patient population is shown in Table 1

All 43 patients presented with quadriparesis, and 36 (83.7%) of the patients had grade ≥3 weakness as measured on the GBS disability scale. Deep tendon reflexes were absent in 24% of patients, while they were demonstrable but decreased in 38% of the patients. None of the patients were found to have sensory abnormalities. The mean (± standard deviation) serum potassium level of patients at baseline was 2.19(±0.54) and serum potassium level ranged between 1.3 meq /L and 3.1meql/L. All the patients improved after potassium supplementation. The mean recovery time (from initiation of potassium supplementation to complete recovery) was 32.2(±9.33) hours and the mean duration of hospital stay was 3.2 (±1.10) days. There was no mortality in our study population.

Table 1: Etiologic profile of the patient population

ETIOLOGY	NUMBER (%)
Diarrhea	15(34.8)
Sweating	5(11.6%)
Renal Tubular Acidosis	3(6.9%)
Diuretic	3(6.9)
Bartter Syndrome	2(4.65)
Gitelman Syndrome	2(4.65)
Hypothyroid	2(4.65)
Thyrotoxic Periodic Paralysis	4(9.3%)
Cushing's syndrome	1((2.3)
Diabetic Ketoacidosis	3(6.9)
Primary Hypokalemic periodic Paralysis	3(6.9)
TOTAL	43

Discussion

Hypokalemia is one of the most commonly encountered fluid and electrolyte abnormalities. The clinical presentation of hypokalemia can be varied, ranging from mild weakness to severe respiratory weakness or cardiac arrhythmias. Furthermore, it could be an incidental laboratory finding in an asymptomatic patient on routine screening of electrolytes. In the present study, mean age was 29.18(±10.23) years, with a range of 11 years to 63 years. Lin SH et al. [1] reported similar baseline characteristics with a mean patient age of 29+/-1.1 yrs. In the study Kayal AK et al. [12] which included 56 patients with hypokalemic paralysis; the mean age was 36.76 ± 13.72 years (range 15-92) years, the male: female ratio being 2.73:1. In the present study 26 (60.4%) were males and 17(39.5%) were females. In the study by Lin SH et al. [1] the male:female ratio was 77:20. The study by Alkaabi JM et al. [5] included 17 patients, all were males and mostly Asians. Male preponderance was observed in the present study, which has also been documented in Indian and other Asian literature [1,12]. Hypokalemic periodic paralysis is inherited in autosomal dominant fashion and it is manifested less likely in females[13]. Among 30 (69.7%) patients; precipitating factors were present prior to the present attack of acute hypokalemic paralysis and in the remaining 13 patients precipitating factors were absent. In the study by Kayal AK et al five patients had a family history of a similar illness. [12] Thirty-one patients (55.35%) presented with a history of recurrent attacks in the past which is more than that observed in the present study, probably due to the presence of patients with primary periodic paralysis.All patients in our study presented with quadriparesis and on clinical examination, with a majority (83.7%) having grade ≥3 as measured on the GBS disability scale. This is similar to findings noted previously where none of the patients had sensory, ocular problems or cranial nerve abnormalities on examination [14-16]. In the present study the mean serum potassium level of patients at baseline was 2.19 (±0.54) and serum potassium level ranged between 1.3 mEq /L and 3.1 mEq/L. Similar findings are reported in other studies and it has been observed that the severity of hypokalemia is higher in secondary hypokalemic paralysis against in primary hypokalemic paralysis. [1,2]

In the present study 40 patients had secondary acute hypokalemic paralysis and 3 patients had primary HPP. Acute gastroenteritis was the most common cause of hypokalemic paralysis seen in 15 patients. The cause of secondary hypokalemic paralysis include gastrointestinal or renal disorders as also observed in the present study . Other studies have reported varying findings. In a large study by Lin SH et al. from Taiwan on 97 patients, 68% patients had a secondary cause. TPP was the most common cause .[1] Patients with HPP, required a lesser amount of therapy with potassium. Moreover, even though administered a much lower amount of K, subsequently one in three cases developed a high level of serum K+ (>5 mEq/L). In the study by Alkaabi JM et al the majority of patients were admitted during the summer months.[5] In a study by Kayal AK et al. [12] reported 32 patients with Primary HypoPP (52.7%), of which 5 patients had a significant family history (FHPP) and the remaining 27 cases were sporadic. Secondary hypokalemic paralysis in 24 (42.9%) patients was reported with underlying Giltelman syndrome in 4 (7.14%) cases, Liddle's syndrome in 2 (3.57%) patients, TPP in 3 (3.57 %) cases, hypothyroidism in 2 (3.57%) cases and primary hyperaldosteronism in 1 patient (1.78%). Hypokalemic paralysis was preceded by gastroenteritis in 2 (3.57%) cases and alcohol intake in 3 (5.35%) cases, while 1 (5.35%) patient developed weakness after dengue fever.In the present study, the mean recovery time (from initiation of potassium supplementation to complete recovery) was 32.2(±9.33) hours and mean duration of hospital stay was 3.2(±1.10) days. This is similar to the finding reported in other studies.

Conclusion

Hypokalemic paralysis should always be kept in mind when making a differential diagnosis for acute flaccid paralysis. The present study adds to the body of evidence on aetiological spectrum, clinical and biochemical parameters and treatment outcome of acute hypokalemic paralysis in India. Recovery with potassium replacement therapy was seen in all cases and there was no mortality during the period of study. Hypokalemic paralysis responds rapidly to potassium replacement. Recognizing and therefore avoiding trigger events cannot be overemphasized in its management. A limitation of the current study was that the period of observation was limited to the duration of hospital stay, and long term follow up could not be ascertained.

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