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Treatment Outcome of Acute Onset Hypokalemia: A Prospective Study at Tertiary Care Center

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Abstract
Background:*Hypokalemia, one of the most common electrolyte disturbances, is diagnosed if plasma potassium concentration is <3.5 mmol/L. The present study was carried out with an objective to know the varied presentation of hypokalemia, its contributing factors, and clinical outcome after potassium replacement therapy.* ***Method:*** *Total 50 patientswho presented with hypokalemia (serum potassium level <3.5 mmol/L) were enrolled during a study period of April 2019 to June 2019.A detailed history was taken. All relevant investigations including complete blood count, urea, creatinine, sodium, potassium, thyroid function test, serum magnesium, ultrasound and ECG were done.* ***Results:****Among 50 patients, 45 (90%) were recovered completely with potassium correction and 5 (10%) died because of respiratory failure. On investigation, serum K+ was low in 5 (10%) patients of which 4 recovered and all these patients had ST-T flattening, U waves and prolonged QT interval. ST-T flattening was present in a majority of 64% patients and all were recovered. 14 (28%) patients had associated hyponatremia (<130 mmol/l) of which 2 patients expired whereas 33 (66%) patients had normal Na+ levels of which 3 patients expired. 88% of patients turned to have chronic kidney disease evidenced by elevated urea, creatinine and small kidney sizes with loss of CMD in ultrasonography. 30 patients were alcoholics and which confirms that alcohol causes kaliuresis, promotes increased K+ rentry into the cells. Concomitant magnesium deficiency aggravates hypokalemia which was noted in 7 patients.****Conclusion:****A careful history, physical, and systematic approach can identify the aetiology of most hypokalemia. A thorough search for aetiology should be undertaken to prevent repeated attacks of this potentially life-threatening dyselectrolytemia.*

Keywords:*Hypokalemia, Potassium, Ultrasonography, Hyponatremia, Kaliuresis, Aetiology, Dyselectrolytemia*

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I. Introduction

Hypokalemia is a common electrolyte abnormality encounteredin clinical practice. It has been found in up to 21% ofhospitalized patients according to reports from studies done mainly among Caucasians [1]. Hypokalemia, defined as a plasma K+ concentration of <3.5 mmol/l, is associated with a10-fold increase in in-hospital mortality, due to adverse effects on cardiac rhythm, blood pressure, and cardiovascular morbidity [2]. Mechanisticallyhypokalemia can be caused by redistribution of K+ between tissues andthe ECF or by renal and nonrenal loss of K+.Theeffective treatment of hypokalemia requires the identificationof its cause [3].The ever expanding armamentarium of drugs with a potential to affect serum potassium (k+) has both complicated clinical analysis and provided new insight.The evolving molecular understanding of rare disorders affecting serum k+ has also uncovered novel pathways of regulation [4].

Patients with hypokalemia often have no symptoms, particularly when the disorder is mild (serum potassium, 3.0 to 3.5 mmol per liter). With more severe hypokalemia, nonspecific symptoms, such as generalized weakness, lassitude, and constipation, are more common. When serum potassium decreases to less than 2.5 mmol per liter, muscle necrosis can occur, and at serum concentrations of less than 2.0 mmol per liter, an ascending paralysis can develop, with eventual impairment of respiratory function. The likelihood of symptoms appears to correlate with the rapidity of the decrease in serum potassium. In patients without underlying heart disease, abnormalities in cardiac conduction are extremely unusual, even when the serum potassium concentration is below 3.0 mmol per liter. In patients with cardiac ischemia, heart failure, or left ventricular hypertrophy, however, even mild-to-moderate hypokalemia increases the likelihood of cardiac arrhythmias [5].

The present study was undertaken to know the varied presentation of hypokalemia, its contributing factors, and its clinical outcome after potassium replacement therapy of this easily treatable metabolic disorder.

II. Materials and Methods

The present study was a prospective observational single centre study in patients with hypokalemia, (defined as serum potassium level<3.5 mmol/L) conducted in the Department of Medicine at ShriVasantraoNaik Government Medical College, Yavatmal over a period of 3 months from April 2019 to June 2019. The study was approved by the Institute Ethics Committee and informed consent was obtained from all the patients.The patients receiving insulin, beta agonists, antacids, magnesium supplements, digoxin, steroids, and GI fistula were excluded from the study.

Total 50 patients were recruited for the study.A detailed medical history including any previous episodes of weakness, thyroid disease, dehydration, alcoholism, high carbohydrate diet, any drug intake, and family history was taken.The investigations including complete blood count, urea, creatinine, sodium, potassium, thyroid function test, serum magnesium, ultrasoundand ECG were done in all the cases and ECG changes were documented.All patients were treated with potassium supplementation.Descriptive statistics was done for getting the number and percentage etc. of the data set.

III. Observations and Results

Among the 50 patients included in the study 45 patients were recovered completely with potassium correction and 5 patients died because of respiratory failure.Males (82%) were preponderantly affected than females (18%).Among the 41 males affected 36 (87.8 %) were recovered and 5 (12.2%) died.All the females were recovered. Among that 1 female was 8 months primiwho presented with hypokalemia and recovered completely with potassium correction.The maximum numbers of cases were in 30-40 years age group. 76% (38) of cases presented within 12-24 hours duration in which 3 (7.9%) patients expired. Among the 50 patients presented with hypokalemic paralysis 10 (20%) patients had 0/5 power of which 4 patients' expired. 6 patients presented with 5/5 power in which all had recovered with potassium replenishment, (Table 1).

Table 1: Socio-demographic features of patients of acute hypokalemia according to its outcome

Socio Demographic Factors		Recovered (n=45)	Death (n=5)	Total (n=50)
Age (Years)	≤21 – 30	1 (100%)	0 (0%)	1 (100%)
	31 – 40	22 (100%)	0 (0%)	22 (100%)
	41 – 50	14 (87.5%)	2 (12.5%)	16 (100%)
	51 – 60	7 (77.77%)	2 (22.22%)	9 (100%)
	≥ 61	1 (50%)	1 (50%)	2 (100%)
Sex	Male	36 (87.8%)	5 (12.19%)	41 (100%)
	Female	9 (100%)	0 (0%)	9 (100%)
Duration of Weakness	< 3 Days	35 (92.10%)	3 (7.89%)	38 (100%)
	3 – 7	6 (100%)	0 (0%)	6 (100%)
	>7	4 (66.66%)	2 (33.33%)	6 (100%)
Power In All 4 Limbs	0/5	6 (60%)	4 (40%)	10 (100%)
	2/5	7 (87.5%)	1 (12.5%)	8 (100%)
	3/5	16 (100%)	0 (0)	16 (100%)
	4/5	10 (100%)	0 (0%)	10 (100%)
	5/5	6 (100%)	0 (0%)	6 (100%)
Duration of Hospital Stay	3	4 (57.1%)	3 (42.9%)	7 (100%)
	4	14 (93.3%)	1 (6.7%)	15 (100%)
	5	6 (100%)	0 (0%)	6 (100%)
	6	3 (100%)	0 (0%)	3 (100%)
	> 7	18 (94.7%)	1 (5.3%)	19 (100%)
Intubation Required		9 (90%)	1 (10%)	10 (100%)

On investigation, serum K+ was low in 5 (10%) patients of which 4 recovered with potassium correction and all these patients had ST-T flattening, U waves and prolonged QT interval. ST-T flattening was present in a majority of 64% patients presented with hypokalemia in which all recovered with k+ correction. U wave was present in 11(22%) patients of which 4 patients expired. 1 patient had ventricular tachycardia who couldnot be revived. 14 (28%) patients had associated hyponatremia (<130 mmol/l) of which 2 patients expired. 2 (4%) patients had hypernatremia (>150 mmol/l).Remaining 33 (66%) patients had normal Na+ levels of which 3 patients expired. 7 (14%) patients had hypomagnesemia. Most of the patients had deranged kidney function

test. 44 (88%) patients presented with creatinine level >1.4. 17 (34%) patients had eGFR between 15- 29 in which all the patients recovered with potassium correction. 7(14%) patients had eGFR< 15 of which 5 expired.2 (4%) patients had hyperthyroidism, 1 of which was newly diagnosed and other was on Thyroxine 50mcg OD since 5 years.Both of them recovered with potassium correction and antithyroid drug initiation, (Table 2).

Table 2: Clinical and laboratory findings of patients of acute hypokalemia

Findings of Hypokalemia		Recovered	Death	Total
K^+ Levels	Low Value	4 (80%)	1 (20%)	5 (100%)
	1 – 1.5	5 (62.5%)	3 (37.5%)	8 (100%)
	1.6– 2	14 (80%)	1 (20%)	15 (100%)
	2.1 – 2.5	16 (100%)	0 (0%)	16 (100%)
	2.6 – 3	6 (100%)	0 (0%)	6 (100%)
ECG Findings	St-T Flattening	32 (100%)	0 (0%)	32 (100%)
	U – Wave	7 (63.6%)	4 (36.4%)	11 (100%)
	Qt Prolongation	5 (100%)	0 (0%)	5 (100%)
	Atrial Bigemini	1 (100%)	0 (0%)	1 (100%)
	Ventricular Tachycardia	0 (0%)	1 (100%)	1 (100%)
Na^+ Levels	≤ 111 – 120	6 (100%)	0 (0%)	6 (100%)
	121 – 130	6 (75%)	2 (25%)	8 (100%)
	131 – 140	15 (83.3%)	3 (16.7%)	18 (100%)
	141 – 150	15 (100%)	0 (0%)	15 (100%)
	≥ 151	2 (100%)	0 (0%)	2 (100%)
Magnesium Levels	Normal (1.5 – 2.5 Ng/Dl)	39 (90.7%)	4 (9.3%)	43 (100%)
	< 1.5 Ng/Dl	6 (85.7%)	1 (14.3%)	7 (100%)
Creatinine Levels	0.6 – 1.4	6 (100%)	0 (0%)	6 (100%)
	> 1.4	39 (88.6%)	5 (11.4%)	44 (100%)
eGFR	< 15	2 (28.6%)	5 (71.4%)	7 (100%)
	15 – 29	17 (100%)	0 (0%)	17 (100%)
	30 – 40	14 (100%)	0 (0%)	14 (100%)
	60 – 89	4 (100%)	0 (0%)	4 (100%)
	≥ 90	3 (100%)	0 (0%)	3 (100%)
Thyroid Disorder	Hyperthyroidism	2 (100%)	0 (0%)	2 (100%)
	Hypothyroidism	1 (100%)	0 (0%)	1 (100%)

23 (46%) patients had history of heat exposure in which 3 patients expired. 30 (60%) patients were alcoholics among which 5 expired.45 (90%) patients had history of high carbohydrate intake. 7 (14%) patients had history of similar episode in the past in which 2 patients couldnot be revived, (Table 3).

Table 3: Role of contributing factors in the outcome of acute hypokalemia

Contributing Factor		Recovered	Expired	Total
Heat Exposure	Present	20 (87%)	3 (13%)	23 (100%)
	Absent	25 (92.6%)	2 (7.4%)	27 (100%)
Alcoholism	Alcoholics	25 (83.3%)	5 (16.7%)	30 (100%)
	Non Alcoholics	20 (100%)	0 (0%)	20 (100%)
H/O High Carbohydrate Diet	Present	45 (100%)	0 (0%)	45 (100%)
	Absent	5 (100%)	0 (0%)	5 (100%)
H/O Drugs Intake	Antipsychotics	1 (100%)	0 (0%)	1 (100%)
	Antiretroviral Therapy	2 (100%)	0 (0%)	2 (100%)
	Thyroxine	1 (100%)	0 (0%)	1 (100%)
Past History of Similar Episode	Present	5 (71.4%)	2 (28.6%)	7 (100%)
	Absent	43 (100%)	0 (0%)	43 (100%)
Hyperthyroidism	Present	2 (100%)	0 (0%)	2 (100%)
	Absent	48 (100%)	0 (0%)	48 (100%)
Hypomagnesemia	Present	6 (85.7%)	1 (14.3%)	7 (100%)
	Absent	43 (100%)	0 (0%)	43 (100%)

IV. Discussion

In the present study, total 50 cases of hypokalemia were detected during a study period of 3 months and which havesignificantly higher number of cases over a short duration of time compared to previous studies from India [6, 7].All these cases were reported in the month of April to June when the temperature in this regionranges from 22 to 43^0C which is comparable with the study done by Dungdung et al[8] and Kayal et al [9].But in the study of Kayal et al [9] patients didn't have signs of dehydration during admission, in contrary

patients in current study had signs of dehydration during hospital admission.Maharashtra has a typical monsoon climate with hot, rainy, cold weather seasons and extremely dry summers.This poses to be one of the factors for major prevalence of hypokalemia during summer.The majority of patients were in the age group of 30-40 years, as similar to the other studies [10, 11]. Males were preponderantly affected as they are more exposed to physically demanding jobs outside home with more sweating during the summer and resultant loss of potassium [10].

Two thirds of the patients (60%) were alcoholics and which confirms that alcohol causes kaliuresis,promotes increased K+ rentry into the cells which was also seen in study done by Chandramohan et al [12] and Kardalaset al [13].However, hypomagnesemia often occurs with and worsen hypokalemia especially in presence of alcoholism.Because both promote the risk of occurrence of cardiac arrhythmias like torsades de pointes,hence all the patients were investigated for concomitant hypomagnesemia and 7 patients in present study who had low Mg+ levels were given magnesium supplementation as the presence of hypomagnesemia can make hypokalemia refractory to potassium supplement alone [14]and the 1 patient who expired had concomitanthypokalemia and hypomagnesemia which lead to the occurrence of ventricular tachycardia. The other major precipitating factor was found to be alcoholism and high carbohydrate diet which is also observed in the previous studies [8, 15, 16]. Hypokalemiaplays a pivotal role in causing ECG derangements and myopathy in alcoholics. Many such cases of proximal myopathy due to hypokalemia in alcoholics with muscle weakness have been documented[16-18]which emphasizing the need for early recognition of low potassium states in alcoholics with muscle weakness and its prompt correction.

The majority of patients (88%) turned to have chronic kidney disease evidenced by elevated urea, creatinine and small kidney sizes with loss of CMD in ultrasonography.This is because of the diminished capacity for excretion of K+ in CKD and the resultant hypokalemialeads to renal fibrosis and renal insufficiency via modulation of renal inflammation and local activation of RAS.The 5 patients who expired in the study were in ESRD.A meta- analysis conducted about the association of hypokalemia and all-cause mortality in CKD revealed that hypokalemia was associated with higher mortality in CKD patients,especially in those with K+ <3.5 mEq/L [19].

In the current study, 2 patients with the history of taking anti retro viral therapy had hypokalemia, both of them recovered with k+ replacement. In a study conducted byRamteke et al[20]showed that tenofovir-an NRTI widely used in combination with other antiretrovirals in the treatment of HIV proposed to cause caspase mediated proximal tubular cell injury through mitochondrial toxicity by inhibiting mitochondrial DNA gamma polymerase leading to acute and chronic renal failure,fanconisyndrome, diabetes insipidus[20].Despite the side effects withdrawal of tenofovir has not been reported except for a recent case report [21].

Among the 18% females affected,an 8 months ANC,Primiwas presented with acute onset paraparesis.ECG showed features of hypokalemia.She had no history of pica, steroid intake, past history, family history. On investigating, she turned out to be hyperthyroidic. Antithyroid medications started and normal thyroid functions achieved without further episodes of TPP and she was delivered normally at 38 weeks of gestation. A similar case was documented in the study of Donovan et al [21]. Many such cases of hypokalemic myopathy was documented previously [22, 23]which emphasis that with timely detection and prompt management of hypokalemia there is a scope for vaginal delivery without any fetal, maternal complications [22].

Among the 50 patients, 5 patients were died due to respiratory failure.All of them presented with acute onset quadriparesis with absent neck holding and got intubated on the day of admission.3 of them had low potassium values and 2 of them had potassium levels <1.5 mmol/l. All of them were alcoholics and had ECG changes of hypokalemia and 1 patient had ventricular tachycardia. All of them had end stage renal disease with history of heat exposure in 3 patients, concomitanthypomagnesemia in 1 patient.In a study conducted by Humnay et al also, death occurred due to respiratory paralysis[23].

V. Conclusion

In the present study most common preceding event of these hypokalemic paralytic episodes was severe dehydration in summers and this is reversible with appropriate potassium therapy.Alcoholism confirmed that the alcohol causes kaliuresis,promotes increased K+ rentry into the cells. Concomitant magnesium deficiency aggravates hypokalemia and renders it refractory to treatment by potassium. Maximum numbers of patients turned to have chronic kidney disease which was life threatening and was associated with sudden cardiac deaths. A thorough search for aetiology should be undertaken in patients with hypokalemia as appropriate treatment prevents repeated attacks of this potentially life-threatening dyselectrolytemia. Further, targeting the primary aetiology prevents future complications. A careful history, physical, and systematic approach can identify the aetiology of most hypokalemia, including obscure ones.

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