CLINICAL PROFILE OF HYPOKALEMIC ACUTE PARALYSIS

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ABSTRACT: Background: Acute flaccid motor paralysis is a common neurological emergency with diverse causes and variable outcome. There is paucity of reports documenting the clinical profile of hypokalemia paralysis in neurological practice. **AIMS:** To study the clinical features, etiology and correlation of hypokalemia with ECG changes and outcome of the subjects with hypokalemic paralysis in a tertiary care teaching hospital in central India. **MATERIALS AND METHODS:** This study encompassed consecutive subjects with acute flaccid paralysis with hypokalemia from 2009 to 2011. Subjects with Gullain Barre syndrome (GBS), porphyria were excluded. Detailed clinical examination, urine analysis, renal function tests and ECG were carried out. Patients received intravenous or oral potassium supplementation and the underlying causes were treated. **STATISTICAL ANALYSIS:** The data were analyzed by Fisher exact test, correlation and coefficient test, using EPI Info software. **RESULTS:** Thirty patients aged 19 to 55 years, including 21 males 09 females were included. Secondary causes such as diarrhea were preset in 23 patients and cause could not be found in 6 patients. All the patients had quadriparesis and (MRC) grade (0 to IV). Deep tendon reflexes were reduced in all patients. Respiratory paralysis occurred in one patient. All patients had potassium levels between patients with severe 0.9 to 3.4mg/L. All patients had ECG change of hypokalemia. More sever hypokalemia needed longer time to recover. One patient died because of respiratory failure. **CONCLUSION:** Diagnosis of hypokalemic acute paralysis should be considered in any patient presenting with sudden onset, aflexic, pure motor weakness involving one or more limbs without alteration in level of consciousness or sphincter disturbances. It is imperative for physicians, particularly those working in acute care setting, to be aware of this condition.
KEY WORDS: - Hypokalemia, paralysis, diarrhea, Electrocardiogram.

INTRODUCTION: Acute muscle weakness with different etiologies of neurologic, metabolic and renal origin is commonly seen in emergency room, especially where there is large Asian population.[1] Among the metabolic causes, hypokalemic paralysis (HP) is a potentially reversible electrolyte and muscle disorder that is characterized by acute muscle weakness and sever degree of hypokalemia (serum k^+ < 3.4 mEq/L). Hypokalaemia is a common problem and may be due to gastrointestinal losses (infectious diarrhea, vomiting), laxative abuse [2.3] or renal losses (diuretic abuse, renal tubular acidosis). Hypokalemic paralysis is a dramatic clinical entity. The sudden onset of flaccid paralysis could be life threatening but if diagnosed in time and properly treated the response and recovery can be equally dramatic and gratifying and patient recovers without any sequelae. But there are very few studies in India in adults.

MATERIAL AND METHOD: Study design -This prospective, observational, analytical study was carried out in medicine department in an 800 bedded tertiary care hospital in central India, from 2009 to 2011. Study was initiated after permission from institutional ethical committee. In this study patient with acute paralysis with serum potassium level < 3.4mEq/L were included. Acute

paralysis with normal serum potassium level, G.B. syndrome, and porphyria were excluded. The serum potassium level was correlated with the ECG changes and severity of motor paralysis. All subjects were informed about the objectives of the study, and informed consent was obtained.

RESULTS: A total of 30 patients presented with acute paralysis and hypokalemia. 21 (70%) were male and 9 (30%) were female. They were in the age group of 20-50 years (Table 1), 23 (76.6%) patients had history of diarrhea, 1 (3.3%) patient was on diuretics and in 6 (20%) patients there was no history of any precipitating factor contributing to hypokalemia or weakness. None of the patients had any associated systemic illness (Table 2).

Out of 30 patients all had weakness in all 4 limbs, one (3.3%) patient had respiratory muscle paralysis and 15 had associated sensory symptoms. On examination of nervous systems all had hypotonia. The power in the muscle ranged from grade 0 in five patients, grade I - II in 15 patients, grade III in 9 patients and grade IV in 1 patient (Table 3). Deep tendon reflexes were absent in all while planter responses were absent in 25 (83.4 %.) patients. In this study all patients with grade zero power had a serum potassium level < 1 mEq/l. Majority of patients (n=12) with grade II power had a serum potassium between 2.4 – 1.5 mEq/L, while 3 patients with this grade power had serum potassium less than 1.4 mEq/L. 5 patients with grade III power had serum potassium between 3.4 – 2.5 mEq/L and 4 patients with grade III power had serum potassium in range of 2.4 – 2.5. One died within 20 min of admission because of K^+ 2.4 to 1.5 mEq/L. One patient of grade IV power had serum potassium between 3.4 – 2.5 mEq/L. when serum K^+ is compared with motor weakness, serum K^+ < 2.5 and motor weakness is less (Gr. II), it is found statistically significant $P < 0.0003$ (Fisher exact test). It is also seen by using correlation and coefficient = 0.8772, it shows serum K^+ level ↓ motor weakness ↑. Though patient gave history of sensory symptoms but there was no sensory deficit in any of the patients.

On investigation the serum potassium was low and ranged from 3.4 to 0.9 mEq/L. ECG changes were present in all patients. All patients showed U wave and pseudo QT prolongation. The correlation between ser K^+ and QTC is given in (Table 4). In management 28 patients were given IV potassium in a dose ranging from 300-500 mEq over a period of 48 hours. Oral potassium was given to 2 patients over a 3 days period. Three cases recovered within 24 hours. One died within 20 min of admission because of respiratory paralysis, serum potassium was 0.9mEq/L in this patient. While the rest recovered within 4 days.

DISCUSSION: Potassium with other electrolytes is lost in diarrheal stool at all ages [4]. Disturbances of potassium equilibrium may produce a wide range of clinical disorder, including myopathy; marked muscle wasting, diminution of muscle tone, power and reflexes [5-7]. Hypokalemia hyperpolarizes the skeletal muscle cells impairing their ability to develop the depolarization, which is necessary for muscle contraction. It can also reduce blood flow to skeletal muscle. The combination of these effects may lead to muscle weakness. Severe potassium depletion can result in two major neuromuscular consequences, paralysis and rhabdomyolysis. Paralysis has a predilection for extremities; with legs more involved than arms. Trunk musculature can also be involved and may result in life threatening respiratory paralysis. In our study only one patient had respiratory paralysis. Sever paralytic complications usually occur with serum potassium values less than 2.6 meg/l, In most reported cases, the cause of hypokalemia has ranged from familial periodic paralysis and sporadic paralysis [8]

secondary to diuretic or laxative abuse, renal tubular acidosis. Though gastroenteritis is a well known cause of hypokalemia, not much information is available about muscle paralysis including respiratory paralysis, due to hypokalemia following gastroenteritis. Zaman et al[9] have reported that 307children, nearly 66.6% had evidence of muscular involvement in the form of muscle weakness following gastroenteritis. Subba Rao SD.[10] reported four out of five cases had acute gastroenteritis and dehydration as a cause of hypokalemia. But in our study we found 23 subjects (76.6%) having gastroenteritis as a cause for hypokalemia.

The effect of hypokalemia on the cell membrane is to increase the resting membrane potential and increase the duration of action potential and refractory period. This explains the arrhythmogenic effect of hypokalemia."[11] The typical changes include flattening of T wave, appearance of U wave and ST segment sagging. In our study ECG changes however did not correlate well with severity of disturbance in potassium metabolism. This result is in conformity with earlier report. The probable reason for this could be that the ECG might be linked to potassium at tissue level rather than serum potassium values [13].

A potential risk of cardiac arrhythmia and respiratory failure induced by hypokalemia calls for urgent treatment. There are four notes of cautions when treating patients with a severe degree of hypokalemia. First, if bowel sounds are absent the oral route for K^+ administration should not be used. Second, parental KCI might be administered in saline instead of glucose solution which may cause the redistribution of K^+ from extra cellular into intracellular space via enhanced insulin secretion.11 third; the aim of therapy is to get the patient out of danger, not to immediately correct the entire K^+ deficit if severe potassium depletion is evident. Fourth, it is not uncommon for there to be a lag period where K^+ excretion fails to rise once the plasma K^+ concentration returns to the normal range[12]. This danger should be anticipated and K^+ supplements should be discontinued after to 48 hours.

Potassium deficits must be replaced quickly if severe muscle weakness or paralysis is present, with careful watch on serum potassium and ECG monitoring to document the effects of changing potassium levels. Whenever possible potassium deficits should be replaced by oral rather than intravenous route, 3 mEq/kg/day plus maintenance needs being appropriate to initiate potassium repletion. If intravenous potassium is used, care must be taken to avoid abrupt rise in serum potassium. It can be given in a concentration of 20-30 mEq/L or saline infusion, concentrations above 40 mEq/L should be used only with ECG monitoring [3,10]. The normal rate of infusion should be 0.25 mEq/kg/h except in cases of quadriparesis with threatened respiratory insufficiency, when the initial rate can be as high as 1mEq/kg/h [12].

There were certain limitations in our study. As the sample size was small it is likely that we have overestimated actual prevalence. Due to financial constraint we could not evaluate the cause of gastroenteritis in all the patients, who were found to have abnormalities and we could not evaluate acid base disorder of the all patients.

CONCLUSION: A severe degree of hypokalemia with a paralysis is potentially life threatening medical emergency. In our study major cause for hypokalemia was acute gastroenteritis. This is reversible with appropriate potassium therapy. Patient presented with paralysis resembling GBS. Hence acute gastroenteritis with hypokalemia becomes a close differential diagnosis when GBS is diagnosed and hypokalemia is proven unnecessary financial burden of investigations and treatment

for GBS is avoided. In conclusion we feel that in all patients with paralysis look for hypokalemia, as this could be life saving.

Table 1 Distribution of cases as per age and gender

Age yrs	Male	%	Female	%
20-30	05	16.6	02	6.6
31-40	12	40	05	16.6
41-50	04	13.3	02	6.6
Total	21	70	09	30

Table 2 Distribution of cases as per precipitating factor for hypokalemia

PPT Factors	No. of Cases	%
Diarrhoea	23	76.6
Diuretics	01	3.3
No cause	06	20
Total	30	100

Table 3: Correlation between serum K^+ and motor weakness

Serum K^+ mEq/L	motor weakness grades of power			
	0	I/II	III	IV
< 3.4 – >2.5 (n=06)	-	-	05	01
< 2.4 - 1.5 (n=16)	-	12	04	-
< 1.4 (n=08)	-5	03	-	-

Fisher exact test p = 0.0003

Table 4 Correlation between ser. K^+ and QTC

Serum K^+ mEq/L	QTC (sec.)	No. of cases
3.4 – 2.5	.55 - .58	06(20%)
2.4 – 1.5	.50 - .60	16 (53.4%)
<1.4	.50 - .58	08(26.6%)

U wave present in ECG of all patients

Correlation value = 0.2855 (weak correlation)

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