Analysis of Clinical and Metabolic Profile of Acute Neuromuscular Weakness Related to Hypokalemia

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Abstract-

Objective: Acute neuromuscular weakness related to hypokalemia is a readily treatable disorder associated with diverse aetiologies. In this study we aim to report clinical pattern and biochemical features to identify the different aetiologies of the hypokalemic neuromuscular weakness.

Methods: Retrospective reviews of the medical record were analysed. Evaluation included demography, clinical features, investigations performed to ascertain the aetiologies. All the patients were categorised in to 3 groups; Idiopathic hypokalemic paralysis (IHP), dengue associated hypokalemic paralysis (DHP) and secondary group (SG) which included renal tubular acidosis (RTA- 1 and 2), thyrotoxic periodic paralysis (TPP) and Gitelman's syndrome (GS).

Results: Forty patients were analysed and the mean age was 31.78 (range, 14-60) years and 35 (87.5%) were male.The underlying aetiologies comprised of IHP in 20, DHP in 12, RTA-2 in 4, RTA-1 in 2, TPP, GS in one each. Weakness on Medical Research Council (MRC) grade was 2.6±1.19 (range 0-4). Comparison of various clinical and laboratory parameters revealed that more patient in IHP and SG had recurrent attack (p=0.001). DHP group had low platelet (p=0.001), high creatine phosphokinase (CPK) (p=0.01) and serum glutamic oxaloacetic transaminase (SGOT) (p=0.008). SG had significantly lower serum potassium (p=0.04) and more time to improve (p=0.02). Recovery time correlated negatively with serum potassium (r=-0.44, p=0.004) and grade of weakness (r=-0.42, p=0.007).

Conclusion: In half of the patients, secondary causes were identified. After IHP, the DHP emerged as second common cause in post monsoon season. SG had significantly lower serum potassium, recurrent attack and more time to improve.

Key words: Hypokalemia, paralysis, neuromuscular weakness, dengue, outcome

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INTRODUCTION

Acute neuromuscular weakness due to hypokalemia may range from mild weakness to severe paralysis, is a medical emergency and sometime leads to cardiac arrhythmia, respiratory paralysis which may require

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ventilation support [1]. Hypokalemia may be idiopathic or associated with various causative factors like thyrotoxicosis, renal tubular acidosis, Gitelman's syndrome, barium poisoning and gastrointestinal loss [2]. Appropriate treatment and prophylaxis to prevent recurrence relies on underlying cause. Recently the dengue fever has been recognised as an important cause of acute neuromuscular weakness related to hypokalemia. The dengue associated hypokalaemic paralysis; a short lasting condition has been reported in the literature[3-6]. Early recognition of these etiological factors and associated complications is crucial for timely and appropriate therapy. Familial hypokalemic periodic paralysis has been reported as the commonest cause of hypokalemic paralysis in the Caucasians and thyrotoxic periodic paralysis in Asian[7,8]. The aim of the present study was to analyse the clinical pattern and biochemical features to identify the different aetiologies of the hypokalemic neuromuscular weakness.

MATERIAL AND METHODS

Study design: This is a retrospective hospital based observational study in a tertiary care institution. The study was approved by institutional ethics committee. The medical record was retrieved form our computerised hospital information system.

Inclusion criteria: The patients with demonstrable acute flaccid weakness of all four extremities with hypokalemia (serum K+ < 3.5 meq/l) from February 2011 to January 2016 were included in our study cohort.

Exclusion criteria: The patients with Guillain-Barre syndrome, acute transverse myelitis, polio and nonpolio enteroviral infections, acute myelopathy and encephalopathywere excluded from the study.

EVALUATION

A detailed review of medical record of patients was performed regarding history and neurological examination. The demographic data collected included age, sex, locality and season of hospitalization. History of similar illness in the family, episodic weakness in the past, thyroid disease, drug intake, diarrhoea, vomiting, hypertension, kidney disease, fever and any precipitating events were recorded. The examination included blood pressure, pulse, pallor, icterus and temperature. Presence of rash, muscle and joint pain were recorded. The grade of muscle power on Medical Research Council (MRC) scale 0-5, tone, deep tendon reflexes and plantars were noted. Sensory examination included sensation for pin prick, touch and joint positions. During hospital stay, progression of weakness, respiratory failure, need of mechanical ventilation and time to recovery were included in the study.

The investigations to note included blood counts, arterial blood gas analysis, hemogram, hematocrit, blood urea nitrogen, serum creatinine, sodium, potassium, bicarbonate, chloride, calcium, anion gap, phosphate, albumin,alkaline phosphatase, creatine phosphokinase and transaminases. Fasting urinary pH and 24 hour urinary calcium, phosphorus, and creatinine if performed were recorded. Twelve lead electrocardiogram of patient were reviewed. Patients with positive dengue serology for either nonstructural protein 1(NS1), Immunoglobulin M (IgM) or both during acute phase were recorded in the study. Nerve conduction study was performed in patients whom weakness persisted beyond 24 hour. All the patients were classified into three groups, idiopathic hypokalemic paralysis (IHP), dengue associated hypokalemic paralysis (DHP) and secondary group (SG) which included renal tubular acidosis (RTA), thyrotoxic periodic paralysis (TPP) and Giteman's syndrome (GS).

The cases were grouped under following headings for analysis based on following observations

Idiopathic hypokalemic paralysis (IHP): This group was characterized by recurrent attacks of weakness in association with hypokalemia, normal acid base status and absence of any identifiable secondary cause.

Thyrotoxic periodic paralysis [TPP]: Patients in this group were designated by recurrent attacks of acute flaccid paralysis and low serum K+ concentration, normal pH and presence of hyperthyroidism.

Renal tubular acidosis (RTA): This subset was defined by hypokalemia, hyperchloraemic metabolic acidosis, normal anion gap and inability to lower the urine pH below 5.5 during systemic acidosis. It was further classified as proximal and distal in presence of nephrolithiasis on abdominal ultrasound or radiography.

Dengue associated hypokalemic paralysis (DHP): The patients admitted with acute febrile illness with motor

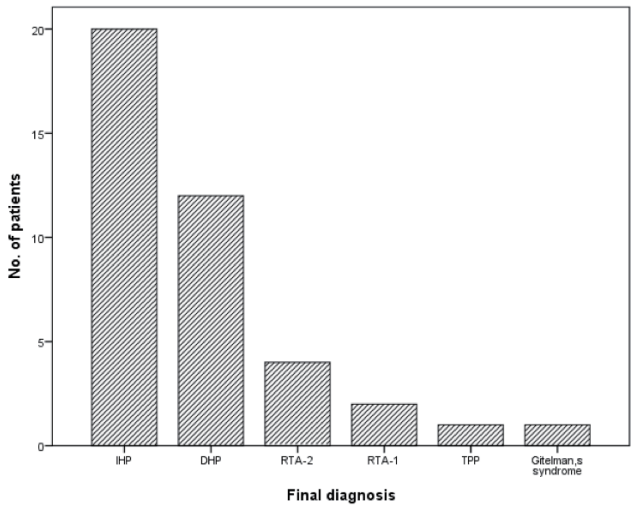

Figure 1. Aetiologies spectrum of acute neuromuscular weakness related to hypokalemia.
IHP, Idiopathic hypokalemic paralysis; DHP, dengue associated hypokalemic paralysis; RTA, renal tubular acidosis; TPP, thyrotoxic periodic paralysis.

weakness, hypokalemia, rapid resolution of symptoms with potassium correction and positive dengue serology for either nonstructural protein 1(NS1), Immunoglobulin M (IgM) or both during acute phase if illness were categorized as DHP.

Gitelman's syndrome (GS): Presence of metabolic alkalosis blood pH >7.45, serum bicarbonate >29 meq/l with hypokalemia (S. K+ <3.0 meq/l), hypomagnesaemia (<1.8 mg/dl), hypocalciuria (urinary calcium <100 mg/24 hour) were classified as Gitelman's syndrome.

The various clinical and metabolic parameters of these groups were analysed to identify the different aetiologies of hypokalemic neuromuscular weakness.

STATISTICAL ANALYSIS

The statistical analysis was performed using IBM SPSS 20 version software. Mean ± SD was used to express the continuous variable and number for categorical values. The comparison between various clinical and biochemical parameters in three groups (IHP, DHP, secondary) were performed with one way analysis of variance (ANOVA) for continuous variable and chi-square test for categorical variable. A bivariate relationship between muscle power and time to recovery in weakness were correlated with various laboratory parameters by Pearson correlation test. The probability value of P <0.05 on 2 tailed test was taken to indicate statistical significance.

RESULTS

A total of 40 patients were included in our study cohort. The mean age was 31.78 (range, 14-60) years and there were 35 (87.5%) male and 28 (70.0%) belongs to urban dwelling. The etiological spectrum comprised of IHP in 20 (50.0%), DHP in 12 (30.0%), RTA-2 in 4 (10.0%), RTA-1 in 2 (5.0%) , TPP, GS in one each [Figure 1]. All the patients were admitted with rapidly progressive areflexic to hyporeflexic pure motor quadriparesis with predominant proximal involvement of extremities. Patients with IHP and Secondary group were admitted throughout the year while all DHP patients in the post monsoon (September to November) season which is more conducive environment for breeding of vector Aedesmosquito.Recurrent weakness was present in 14 patients which ranged from 1-10 episodes per year. Myalgia was noted in 13, preceding fever in 19, body rash 3, vomiting in 2 and paraesthesia in 2 patient. Maximum weakness on MRC grade ranged from 0-4 (0 in 2, 1 in 5, 2 in 12, 3 in 9, 4 in 12) and respiratory failure in 5 however only 2 patient needed mechanical ventilation. Areflexia/ hyporeflexia documented in 35 while 5 patients had normal deep tendon reflexes. Sensory examination was unremarkable. Mean serum K+ was 2.42±0.54 (range 1.4 to 3.4). Electrocardiograph was abnormal in 10 (tachycardia in 4, bradycardia in 3, U wave in 2, QT prolongation, ventricular bigeminy in one each) patients. Electrocardiograph of one patient with dengue associated hypokalemic paralysis revealed u wave [Figure 2]. One patient with TTP had recurrent attack of quadriparesis and his thyroid function tests were suggestive of thyrotoxicosis (TSH 0.01 uIU/ml, N=0.4-4; T3 272 ng/dl, N=81-178; T4 13.5 μg/dl, N=4.5-12.5). One patient with RTA 1 had history of recurrent weakness with dry eye, mouth and joint pain and found to have Sjögren'ssyndrome. Schirmer's test was performed and it showed 4 millimetre moisture on test strip at 5 minute. Her serum antinuclear antibody was 3 + positive, anti Ro antibody (>200 RU/ml, Normal <15) anti La antibody (>200 RU/ml, Normal

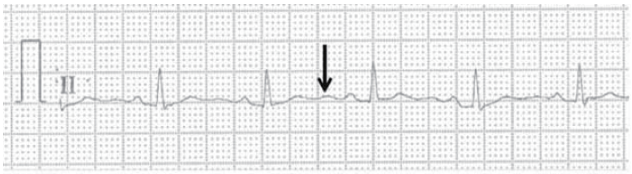

Figure 2. ECG in a patient with dengue associated hypokalemic flaccid quadriparesis (power 2/5 on medical research council scale) showing U wave (arrow) with serum potassium of 2.22 mmol/l.

<15) were significantly elevated suggestive of Sjögren's syndrome. Another female patient with recurrent quadriparesis S. K+ 2.4 meq/l, arterial blood pH 7.47, serum bicarbonate 29 meq/l , serum magnesium 1.6 mg /dl and 24 hour urinary calcium was 90 mg in 24 hour which suggested the diagnosis of Gitelman's syndrome.

All the patients were categorised in to 3 groups; IHP, DHP and Secondary group which included RTA (type 2, type1), TPP and GS patients. Nerve conduction sturdies (NCS) were performed in 13 patients (6 in DHP, 3 in IHP, 2 in RTA 1 and 2 each) who did not recover in 24 hour and it was normal in all DHP and 3 IHP patients. One patient with RTA1 with Sjögren's syndrome revealed bilateral common peroneal motor axonal neuropathy likely due to vasculitis and in other it was normal. Out of two patients with RTA 2 in whom NCS was performed revealed bilateral ulnar motor axonal neuropathy in acute phase.

COMPARISON BETWEEN IHP, DHP AND SECONDARY GROUP

On comparison of clinical and demographic variable revealed that there is significant seasonal variation in DHP as these patients were seen during post monsoon period exclusively while other group were admitted across all seasons (p=0.006). Male gender were wore commonly affected than female (p = 0.001) and most of the patients from urban dwelling (p= 0.01). Recurrent weakness were more in IHP and secondary group while none of DHP group had recurrent weakness (p= 0.0001) [Table 1]. On comparison of other clinical and laboratory parameters across the three groups revealed they were not significantly different except for number of attack (1.30 ± 2.05 vs 0.00 ± 0.00 vs 3.50 ± 2.77, p= 0.001), time to recover in days (1.27 ± 0.65 vs 1.75 ± 0.75 vs 2.56 ±1.91, p=0.02), platelet count (203250.0 ± 72491.0 vs 82250.0 ± 36935.0 vs 146166.0 ± 20461.0, p= 0.0001), serum creatinine (0.85 ± 0.17 vs 1.02 ± 0.19 vs 1.06 ± 0.25, p= 0.02), serum K+ (2.31 ± 0.44 vs 2.74 ± 0.48 vs 2.20 ± 0.71, p=0.04), CPK (345.50 ± 178.94 vs 1594.9 ± 887.47 vs 1046.0 ± 1260.0, p= 0.01) , and SGOT (66.00 ± 86.42 vs 193.83 ± 152.68 vs 34.30 ± 5.00, p= 0.01) in IHP, DHP and secondary group respectively [Table 2]. The mean total serum bilirubin level was 0.75 ± 0.79 mg/dl (normal <1.2 mg/dl).

Correlation. The muscle power correlated positively with serum potassium level (r= 0.81, p= 0.001) and negatively with platelet count (r= -0.35, p= 0.027) but not with CPK, SGOT, SGPT or serum creatinine. The recovery time correlated negatively with serum potassium (r=-0.44, p=0.004), grade of weakness (r=-0.42, p=0.007) and positively with serum creatinine (r=0.38, p=0.02), but not with other parameters [Table 3]. All the patients with DHP had preceding fever of 3-5 days duration with low

Table 1. Comparison of demographic and clinical parameters of patients with hypokalemic neuromuscular weakness.

Seasons		IHP (N=20)	DHP (N=12)	Secondary group (N=8) {RTA,TTP,GS}	p
Summer (March-May)		4	0	1	0.006
Monsoon (June-August)		7	0	3	
Autumn (September-November)		6	12	2	
Winter (December –February)		3	0	2	
Gender	Male	19	12	4	0.001
	Female	1	0	4	
Locality	Urban	10	12	6	0.01
	Rural	10	0	2	
Recurrent	Present	6	0	8	0.001
weakness	Absent	14	12	0	

IHP, Idiopathic hypokalemic paralysis; DHP, dengue associated hypokalemic paralysis, RTA; Renal tubular acidosis, TTP; Thyrotoxic periodic paralysis, GS; Gitelman's syndrome.

Table 2. Comparison of various clinical and laboratory parameters in three group of patients with hypokalemic neuromuscular weakness.

Variables	Type of acute neuromuscular weakness			p
	IHP (N=20)	DHP (N=12)	Secondary Group (N=8) {RTA,TPP,GS}	
Age (years)	28.10±7.45	35.25±9.05	35.75±15.75	0.08
No. of attacks/ year	1.30±2.05	0.00±0.00	3.50±2.77	0.001
Weakness (MRC)	2.35±1.26	3.08±0.90	2.51±1.30	0.24
Time to recover (days)	1.27±0.65	1.75±0.75	2.56±1.91	0.02
Systolic BP (mmHg)	112.8±9.20	123.3±9.8	120.40±18.51	0.80
Haemoglobin (g/l)	130.0±30.6	142.2±15.8	122.8±29.5	0.34
Platelet count ($x10^9$ /l)	203.25±72.49	82.25±36.93	146.16±20.46	0.001
Serum K+(mmol/l)	2.31±0.44	2.74±0.48	2.20±0.71	0.04
Serum creatinine (μmol/l)	75.14±15.02	90.16±16.79	93.7±22.1	0.02
CPK (U/l)s	345.50±178.94	1594.9±887.47	1046.0±1260.0	0.01
SGOT (U/l)	66.00±86.42	193.83±152.68	34.30±5.00	0.008
SGPT(U/l)	93.38±109.36	163.32±166.12	58.83±32.72	0.18

Normal values: Platelets count: 150-450 × 10^9/l, CPK <171 U/l, SGOT: 0-45 U/l, SGPT: 0-45 U/l, S.K+: 3.5-5.5 meq/l, BP: 120-140 mmHg.

CPK, creatine phosphokinase; Cr, creatinine; K+, potassium; SGOT, serum glutamic oxaloacetic transaminase; SGPT, serum glutamic pyruvic transaminase; BP, blood pressure.

Table 3. Correlation of various clinical characteristics with different laboratory parameters in patients with hypokalemic neuromuscular weakness.

Clinical characteristics	Variable	r	p
Muscle weakness (MRC)	Serum potassium	0.80	0.001*
	CPK	-0.22	0.32
	SGOT	0.17	0.34
	SGPT	0.12	0.50
	Serum creatinine	0.10	0.53
	Platelet count	-0.35	0.02*
Recovery time (in days)	Serum potassium	-0.44	0.004*
	CPK	0.26	0.24
	SGOT	-0.12	0.51
	SGPT	-0.15	0.39
	Serum creatinine	0.37	0.02*
	Platelet count	-0.10	0.53
	Muscle weakness (MRC)	-0.42	0.007*

r indicates Pearson correlation coefficient.

*Significant at p value <0.05.

CPK, creatine phosphokinase; SGOT, serum glutamic oxaloacetic transaminase; SGPT, serum glutamic pyruvic transaminase, MRC: medical research council.

platelet count, more elevated CPK value. Test for malaria were negative in all febrile patients and test for human immunodeficiency virus were negative in all cases.

DISCUSSION

Hypokalemia associated neuromuscular paralysis is an important cause of acute flaccid weakness and had varied aetiologies related to renal, endocrinal diseases (Gitelman's syndrome, Bartter's syndrome, Conn's syndrome RTA 1 or 2, thyrotoxicosis) and other causes like barium poisoning and gastrointestinal loss. In our study cohort 20(50%) patients were found to have underlying secondary cause. In the earlier series the secondary cause of hypokalemia

varies from 42.9 to 79.4% [9-11]. In a retrospective study, which included 97 cases of hypokalemic paralysis over 10 year period, the majority of cases of patients with hypokalaemic paralysis were due to secondary causes (68%); underlying aetiologies included thyrotoxic paralysis in 40.2%, sporadic in 29.8%, familial in 2.1%, primary aldosteronism in 6.2%, renal tubular alkalosis, Bartter and Gitelman's syndrome in 6.2%, diuretic use in 3.1%, and ingestion of toluene blue in 3.1% of patients [8]. In a study from endocrinology setting 31 patients were identified over 6 year period and the secondary cause was identified in 93.6% of patients, which were due to hyperaldosteronism in 42% of patients, RTA in 42%, thyrotoxicosis in 6.4%, Gitelman's syndrome in 3.2%, andsporadic periodic paralysis in 6.4% of patients [12]. The underlying aetiology of hypokalemic paralysis depends upon clinical setting, meticulous search for secondary cause and availability of investigations. In earlier reported series the dengue associated hypokalemic paralysis was found in 1.78 % to 13.7% of patients [9,10]. In a recent study of hypokalemic paralysis, dengue associated neuromuscular weakness was found in 18 (31.0%) out of 58 patients [5]. In a study 799 patients with dengue infection from Northwest India, neurological manifestations were present in 21 (2.63%), 19 of whom were men with a mean age of 33.7+13.9 years. The neurological diagnoses were hypokalemia with: quadriparesis (7, 33.3%); myositis (4); encephalopathy (4); Guillain Barre syndrome (2); acute disseminated encephalomyelitis (2); lumbosacral plexopathy and intracranial haemorrhage in one each [13]. In our study DHP was documented in 12 (30%) of the patients which is second most common cause of hypokalemic paralysis followed by renal tubular acidosis in 6 (15%). There are two possible mechanism operates in disease process which may lead to hypokalemia are either transcellular shift of potassium (hypokalaemic periodic paralysis, thyrotoxic periodic paralysis, barium poisoning, insulin excess, alkalosis) or renal loss (excessive mineralocorticoids, renal tubular diseases, diuretics, magnesium depletion) if extra renal loss (decrease intake or gastrointestinal loss) is excluded [2]. The exact pathogenic mechanism of hypokalemia in dengue is unclear but possible explanations may include either excess release of catecholamine in response to stress, secondary insulin release with resultant transcellular potassium shift or self limiting transient tubular dysfunction leading to potassium depletion [3]. Whether the association of hypokalemia in dengue is incidental or there is underlying genetically mediated channel disorder is a matter of debate [14,15]. In our patients with dengue associated hypokalemic weakness, the clinical features, routine urinalysis and arterial blood gas analysis were not suggestive of renal tubular dysfunction. Statistically significant recurrent attack of weakness, numbers of attacks per year were more common in idiopathic hypokalemic paralysis and secondary group and not in those with dengue infection as later being monophasic illness. One of our patients had familial hypokalemic paralysis. Two patients in secondary group (RTA-1) had respiratory failure and needed mechanical ventilation. One of these patients with RTA-1 was diagnosed as Sjögren's syndrome. Association of Sjögren's syndrome with RTA-1 is well known and may present as hypokalemic paralysis with respiratory failure [8,12,16]. Only one patient in our study cohort had recurrent attacks of quadriparesis and diagnosed as thyrotoxic periodic paralysis. TPP is more common in Asian while familial hypokalemic paralysis is more commonly reported in western countries [8]. In our study the male gender outnumber the female sex and this male preponderance in our study is similar to earlier reported series without any plausible explanation for this striking observation [10,12,17]. Precipitating factors were identified in 10 patients with IHP (preceding fever in 6, high carbohydrate in 3 and heavy exercise in 1) but none of DHP and secondary group had any identifiable trigger. Out of 20 patients with IHP group 19/40 (47.5%) were sporadic in nature and only 1 (2.5%) had familial hypokalemic paralysis. Patient with DHP group had significantly lower platelets count as compared to IHP and secondary group. Serum potassium level was significantly low in secondary group as compared to IHP and DHP patients and this is likely due to negative potassium balance. A similar trend was observed in one study while other studies did not support this finding [8,10]. We could not found any significant difference in weakness on MRC grade and systolic blood pressure between IHP, DHP and secondary group.

Severity of muscle weakness on MRC grade correlated with the level of serum potassium in our study as patients with lower potassium had more severe weakness (r=0.80, p=0.0001). Serum creatinine levels

were within normal limits in all the three groups but slightly higher in secondary group likely as result of renal tubular dysfunction. Serum creatine phosphokinase was significantly elevated in DHP group as compared to IHP and secondary group (p= 0.013). The acute pure motor neuromuscular weakness in dengue patients may be due to myositis or rhabdomyolysis [18,19]. In a study of 39 patients with dengue associated transient muscle dysfunction, 31 had shown muscle involvement: 16 clinical (elevated CPK with weakness) and 15 subclinical (elevated CPK without weakness). Mild myopathic changes were documented on needle electromyography. The mean serum potassium level was 3.92 ± 0.70 meq/l and CPK was elevated in all patients. Complete recovery was reported in 2 weeks with improvement in haematological and biochemical parameters [20]. In the above study, there was a subgroup of 15 patients with subclinical muscle dysfunction (without weakness and elevated CPK suggests that elevated CPK might be due to myositis, not leading to weakness. In a description of 6 patients presenting with clinical features of pure motor flaccid weakness with markedly elevated CPK levels found to have potassium depletion myopathy. Histopathological examination showed phagocytosis of degenerating muscle fibres and regeneration. Reversals of clinical and morphologic features were found after potassium supplementation. It has also been suggested that hypokalemia leads to energy failure, vasoconstriction and subsequent muscle ischemia leading to elevation in CPK levels [21]. Elevated CPK levels in our patients are probably due to above phenomenon and significantly high CPK in DHP group may be responsible for myalgia (9/12) in these patients. The patients with rhabdomyolysis have clinical features of muscle weakness, but this entity is a rare but more severe form of muscle involvement and the characteristics features are myalgia, high CPK (at least 10 times of upper limit), and myoglobinuria which may lead to acute renal failure and increases mortality [22,23]. All patients in our study cohort improved with potassium supplementation followed by treatment of underlying cause. Recovery time in days was longer in secondary group (2.56±1.91) as compared to IHP (1.27±0.65) and DHP (1.75±0.75) patients (p=0.02). The recovery time correlated with severity of muscle weakness on MRC grade(r=-0.42, p=0.007) and serum potassium level (r=-0.44, p=0.004)).

Along with CPK elevation there were rise in SGPT (ALT) and SGOT (AST) were also found and increase in later was statistically significant in DHP group as compared to IHP and secondary group (p= 0.008). Increased serum levels of alanine transaminase ALT and AST indicate hepatocyte injury and necrosis. Although ALT is a cytosolic enzyme that is found in highest concentration in the liver, AST is located predominantly in the mitochondria (80%) as well as the cytosol (20%) of hepatocytes, and in the heart, skeletal muscle, kidney, brain, pancreas, lungs, leukocytes, and erythrocytes [24]. Significant elevation of SGOT may be due to skeletal muscle origin in our patients as total serum bilirubin levels were normal except one (S.bilirubin 2.8 mg/dl, N=< 1.2).

One of our patients had TTP. Though exact pathogenesis of TPP is unclear but there is some evidence to support platelet Na+, K+-ATPase activity and in vivo sodium pump activity are increased in thyrotoxicosis [25]. An underlying genetic mechanism is likely to play a significant role as it commonly reported in Orientals than Caucasians, male preponderance and rarely the familial nature[2].

Limitation of our study is retrospective study design, based form a tertiary care neurology setting. The higher number of DHP patients may not reflect true incidence of hypokalemic neuromuscular weakness in dengue patient due to referral bias. Like TTP and underlying genetic predisposition in DHP patients may be contributing to the pathogenesis of this clinical condition which can be elucidated by a more systematic and detailed workup. Furthermore electromyography was not performed in DHP patients due to invasive nature of test and rapid recovery on potassium supplementation.

CONCLUSION

Acute neuromuscular paralysis related to hypokalemia is an important clinical entity with diverse aetiology and it is crucial to differentiate form other causes of acute quadriparesis as treatment and prognosis differ. The features of idiopathic hypokalemic paralysis differ from that of DHP by history of recurrence, relatively younger age without any seasonal preponderance, normal platelet count and relatively normal CPK levels. Secondary causes of hypokalemic neuromuscular weakness are characterised

by higher chances of recurrent attacks, abnormal ABG, lower serum potassium and normal platelets. DHP has emerged as an important cause of hypokalemic neuromuscular weakness during post monsoon season in the endemic areas.

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