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A Case Report on Hypokalemic Quadriparesis

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ABSTRACT

Hypokalemic quadriparesis is a rare phenomenon with potentially life-threatening clinical syndrome. It represents a heterogeneous group of disorders characterized by hypokalemia and acute systemic weakness which are clinically modifiable. It results from alteration in transcellular distribution of potassium or transcellular shift of potassium and actual potassium depletion from renal and extra-renal losses. Hypokalemic quadriparesis management depends on the etiology of hypokalaemia, duration of disease, underlying diseases, family history, medication history and disease state. The cause of hypokalemia induced quadriparesis is relatively low which gets resolved over time; with most of the symptoms remain undetected. Patients with normal thyroid function, no renal loss of potassium and normal acid balance can be pharmacologically managed with oral potassium and intravenous potassium.

Introduction

Hypokalemic quadriparesis is a rare clinical syndrome characterized by acute extreme muscle weakness with sudden onset of generalized or focal flaccid paralysis associated with low serum potassium level (<3.5mmol/L). Severe hypokalemia is when the plasma potassium concentration is less than 2.5mmol/L.

Severe hypokalemia was found to be associated with flaccid paralysis that can range from mild muscle weakness to paralysis of cardiac muscles and respiratory muscles paralysis. Quadriparesis is termed from the Latin word quadra which means "four" with Greek word plegia which means "paralysis".

Most of hypokalemic quadriparesis are due to familial hypokalemic periodic paralysis and primary hypokalemic period paralysis. Sporadic cases are associated with other conditions including barium poisoning, hyperthyroidism, renal disorders, endocrinopathies and gastrointestinal potassium losses (vomiting, diarrhea) [1]. Several studies reported an incidence between 10% and 57% of sporadic hypokalemic paralysis [2,3].

Case Report

A 22 years old female patient with history of vomiting 4 episodes (non-projectile) 5 days back and LSCS 3 months back presented to ER with weakness of both upper and lower limbs since three days. She complained of sudden onset of heaviness of both lower limbs which progressed to weakness of both upper limbs. Patient was unable to maintain head posture; she had difficulty in getting up from chair (proximal muscle weakness).

On admission her vitals were as follows – Blood Pressure: 130/70 mmHg, Pulse rate: 76 bpm, GCS: $E_4M_1V_5$ (10/15). On examination of the motor system, the power of both upper and lower limbs was found to be decreased. There was decreased tone in both upper and lower limbs. Her lab reports on the day of admission showed decreased hemoglobin (11.4 g/dl) content, low PCV (32.2 %), low serum potassium level (2.4 meq) and low serum magnesium level (1.2 mg/dl).

Her chest X-ray, thyroid profile, renal function test, liver function test, MRI scan, USG abdomen, ECG were found to be normal.

The patient was treated with Injection - KCl 2 amp in 500 ml NS, Injection - Pantoprazole 40 mg, Syrup - Potklor 15 ml on the first day of admission. From the second day the patient was managed with Injection - KCl 20 meq in 500 ml NS, Tablet - Zincovit and Tablet - Ultra Mag. 200 mg. After three days of treatment the patient serum potassium was found to be normal and there were no complaints of weakness of both upper and lower limbs and she was discharged the following day.

Table 1: Vitals observed in the patient

Vitals	Values Observed
Blood Pressure	130/70 mmHg
Respiratory Rate	24 bpm
Pulse Rate	76 bpm
Temperature	98.4^{0}F

Physical Examination

Glasgow Coma Scale**:** E_4 V_1 M_5 (10/15)

Motor System

Table 2: Power

Power	Right	Left
Upper limb	1/5	1/5
Lower limb	1/5	1/5

Table 3: Tone

Tone	Right	Left
Upper limb	↓	↓
Lower limb	↓	↓

Table 4: Laboratory Investigations

Complete Blood Picture (CBP)			
Test	**Result (Day 1)**	**Result (Day 3)**	**Normal values**
HB	11.4 gm/dl	10.8 gm/dl	12.0 - 15.0
PCV	34.2 %	35.8 %	36 - 46
Serum Electrolytes			
Test	**Result (Day 1)**	**Result (Day 3)**	**Normal values**
Serum Sodium	142 meq/L	138 meq/L	135 – 155
Serum Potassium	2.4 meq/L	3.7 meq/L	3.5 – 5.5
Serum Calcium	11.8 mg/dl	10.4 mg/dl	8.4 – 11.5
Serum Magnesium	1.2 mg/dl	1.8 mg/dl	1.6 - 3

Discussion

Hypokalemic quadriparesis can be precipitated by hereditary disorders (familial hypokalemic periodic paralysis, primary hypokalemic period paralysis), sporadic case (hypomagnesaemia, anesthetics, diuretics, penicillin's, aldosterone, iatrogenic factors, abdominal lacerations, gastrointestinal loss, renal wasting like distal renal tubular acidosis, barium poisoning), thyrotoxicosis, dengue viral fever, Gitelmans Syndrome and Conn's syndrome [4].

Hypokalemic quadriparesis is a rare event which is found in association with extra-renal potassium loss (hyper-emesis) and anesthesia in surgical interventions [5]. The mechanism for hypokalemic quadriparesis due to hyper-emesis and anesthesia is yet to be fully understood. In hyper-emesis there is severe potassium electrolyte loss causing hypokalemia, low potassium ion repolarises muscle resting potential rapidly even when there is calcium conductance that makes the muscles to relax. Axonal demyelization due to inflammation can lead to motor deficit following regional as well as general anaesthesia [6].

The primary goal of treatment is focused on normalizing potassium level by administering oral potassium chloride which is readily absorbed to alleviate the symptoms of muscle weakness. Patient needs to be monitored for 24 hours by ECG along with muscle strength examination to minimize the adverse effects of post treatment rise in serum potassium level [7,8]. In swallowing difficulties IV potassium is administered which requires continuous ECG monitoring.

Administered potassium ions acts on Na^+-K^+ Pump across the cell membrane and cause influx of sodium ions and efflux of potassium ions that generates endplate potential (depolarization) and triggers action potential that travels along the sarcolemma and enters into the muscle fiber via transverse tubules which stimulates the opening of voltage gated calcium channels that are coupled to calcium release channels in sarcoplasmic reticulum (SR). The calcium ions thus diffuse out of SR to the myofibrils and stimulate contraction of muscles. Prophylaxis against hypokalemic quadriparesis can be achieved with supplement potassium and low carbohydrate diet.

Conclusion

The cause of hypokalemia induced quadriparesis is relatively low which gets resolved over time; with most of the symptoms remain undetected. Patients with normal thyroid function, no renal loss of potassium and normal acid balance can be pharmacologically managed with oral potassium and intravenous potassium.

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Abbreviation used

LSCS: Lower Segment Caesarean Section
ER: Emergency Room
ECG: Electrocardiography
HCl: Hydrochloride
NS: Normal saline
KCl: Potassium chloride

Conflict of Interest

Nil

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