

Hypothyroidism with Recurrent Hypokalemic Paralysis

Pruthviraj Chauhan¹, Ajay Naxane²

¹Junior resident, Department of General Medicine, SBKS MIRC, Pipariya, Waghodia, Vadodara

²Assistant prof, Department of Nephrology, SBKS MISC, Pipariya, Waghodia, Vadodara

Abstract: Hypokalemic paralysis represents a medical emergency requiring both rapid diagnosis and treatment. The cause of this paralytic disorder may be genetic or acquired. Sudden onset of weakness is triggered by stress such as viral illness, fatigue, fight, emotional disturbance and medications like insulin, beta-2 agonists and steroids. This transient and episodic neurological condition is commonly associated with Thyrotoxicosis. Hypothyroidism presenting as recurrent hypokalemic paralysis is rare in the literature. We report a case of 38 year old female with sudden onset of weakness in lower extremities, no sensory deficits and normal higher mental function. There was no enlargement of thyroid gland. Laboratory evaluation revealed low serum potassium. Patient was diagnosed having chronic thyroiditis with high thyroid stimulating hormone and thyroid related antibodies. Follow up shows satisfactory result with thyroxine replacement. It is an extremely rare and unusual presentation of hypothyroidism with hypokalemic paralysis.

Keywords: Hypothyroidism, Recurrent Hypokalemic Paralysis

1. Introduction

Hypokalemic periodic paralysis, the most common periodic paralysis with a prevalence of 1 in 100,000.^{1,2,3} is also a rare channelopathy. It can result from reduced potassium intake, increased translocation from extracellular spaces into the cells (as a transient condition) or, most commonly, from increased gastrointestinal or urinary losses. Increased potassium secretion in the distal nephron may account for such losses, for example, with the intake of diuretics or because of mineralocorticoid excess.⁴ Hypokalemic paralysis may be caused by a short-term shift of potassium into cells, seen in hypokalemic periodic paralysis (caused by familial periodic paralysis or thyrotoxic periodic paralysis); Thyrotoxicosis is the most common cause of secondary hypokalemic periodic paralysis. Recurrent hypokalemic paralysis is an extremely unusual presentation of hypothyroidism.

2. Case Report

A 38 year old female presented with acute onset of B/L lower extremity weakness, inability to walk, and profound fatigue since two days. Patient had been having intermittent leg cramps on and off since past 2 years, also had a history

of flaccid paralysis 6-8 month back, and was treated with intravenous potassium supplement after which she had a complete recovery.

There was no history of fever, diarrhea, vomiting, rash or abdominal pain, no history of polyuria, polydipsia, thirst, history of altered sensorium, convulsion, visual or respiratory weakness, no history of drug intake or alcohol abuse.

On physical examination, she appeared weak and was able to stand with support. Her vital signs were: Temperature 98.7°F, heart rate 50/min, respiratory rate 20/min, BP 118/50 mmHg, and O2 saturation 99% on room air. Lungs were clear, heart sounds were normal, abdomen was soft, non tender with normal bowel sound. On central nervous system examination, higher mental functions were normal, no cranial nerve involvement, motor strength in distal upper extremities 4/5 grade, in proximal upper extremities 3/5 grade, in distal lower extremities 4/5 grade, and in proximal lower extremities 1/5 grade. There were diminished deep tendon reflexes, bilateral flexor plantar, and no sensory involvement.

Laboratory investigation as mention in Table 1.

Table 1

CBC	HB:13.9gm/dl, TC:7.9 * 10 ⁹ /L, PLATELETS:207 * 10 ⁹ /L,MCV: 86.3 fl, MCH 28.8 pg, MCHC:34.6 gm/dl
ESR	45ml/h
Urine routine	albumin +, sugar-absent, pus cell1-2-3wbc/hpf.
S. electrolytes	Na-138 mmol/L, K- 1.8 mmol/L, Cl-98 mmol/L
Renal function	Serum creatinine 0.7mg/dl, blood urea 27 mg/dl
liver function	SGPT-23 U/L; SGOT – 19 U/L; S.Bilirubin -0.4 mg/dl;direct -0.2 mg/dl;indirect -0.2 mg/dl.
total protein	6.90 gm/dl; albumin -4.4 gm/dl;globulin-2.50 gm/dl A/G -1.76
S.TSH	serum TSH >100mIU/L, serum antiTPO antibody titre was 287 IU/ml
Serology	HIV-negative; HBsAG - negative; HCV - negative
Lipid profile	Cholesterol – 97 mg/dl;Triglycerides – 154 mg/dl;HDL-C -38 mg/dl;LDL-C - 28.2 mg/dl;VLDL – 30.8 mg/dl.
Arterial blood gas	pH 7.36, Pco2 36 mmHg, Po2 97 mmHg, and bicarbonate 24 mEq/L
Others	serium calcium-10mg/dl, serum magnesium-2.2mg/dl

Her electrocardiogram showed normal sinus rhythm without features of hypokalemia (i.e. no flattening of T wave, inverted T wave, U wave, depression of ST segment, decreased QRS voltage or prolonged PR or QT interval), USG and chest x ray were normal. Further investigations: creatinine phosphokinase 241 U/L, 24 hr urinary potassium excretion was 16meq/l, urine pH 7.4, specific gravity 1.012; urinary calcium was within normal limit. The computed tomography scan abdomen was normal. Her plasma rennin was measured normal (2.4ng/ml/h).

Table 2

Selected laboratory daily investigation				
	Day 1	Day 2	Day 3	Day 12
Sodium	138	142	145	140
Potassium	1.8	2.6	3.8	4.2
Chloride	98	110	114	104
Creatinine	1.2	1.0	1.0	0.8

Hypokalemic paralysis was diagnosed based on clinical and biochemical parameters and was treated with infusion potassium chloride at 20ml/hr (40meq potassium chloride in 40 ml NS, 80 meq KCl to be replaced in 24/hr), oral and infusion supplementation of KCl for 3 days that lead to clinical and biochemical improvement. Patient was also started on tab levothyroxine 100mcg, and was discharged on oral syrup potassium chloride and tab levothyroxine and reviewed after 4 weeks. Patient serum potassium level and serum TSH level became normal. Patient potassium supplementation was stopped and levothyroxine was continued, there was no further complain of weakness, muscle cramp during the follow up of 1.5 years.

3. Discussion

Secondary hypokalemic periodic paralysis is less common. Clues indicating a secondary cause are the lack of family history and the time of onset of symptoms. Patients who have their first attack of weakness in adulthood should be screened carefully for a secondary cause. Secondary hypokalemic periodic paralysis is non-genetic in origin and results from causes such as thyrotoxicosis, barium poisoning, primary hyperaldosteronism, diuretic abuse, renal tubular acidosis, Bartter syndrome, and gastrointestinal diseases.³

In this case patient was admitted with the PARAPARESIS, laboratory studies showed hypokalemia, normal creatinine kinase; rules out possibility of a myopathy, absence of nausea, vomiting, constipating, hypochloremia, and hyponatremia rule out Bartter syndrome, normal serum magnesium and urinary calcium excretion rule out Gitelman's syndrome, there was no metabolic acid-base disturbances that rule out possibility of renal tubular acidosis, though urinary potassium was lower side, in CT scan abdomen adrenal were normal and no characteristic feature of hyperaldosteronism like hypertension and polyuria were present.

The levels of thyroid hormones and TSH values in this patient indicate severe deficiency of thyroxine. The presence of autoimmune thyroiditis is indicated by the high titer positivity of anti-TPO antibodies in serum.

Final diagnosis: Recurrent hypokalemic paralysis in hypothyroidism.

4. Conclusion

Hypokalemic periodic paralysis though common among Indian population varies greatly in disease spectrum and magnitude in our country due to the heterogeneous pattern of etiology. The association of periodic hypokalemic paralysis with hypothyroidism has not been established till now.

References

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