

Thyrotoxic Hypokalemic Periodic Paralysis

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Abstract

Hypokalemia is a serious and life-threatening clinical condition. We present a case of a 45-year-old male, with known hyperthyroidism presenting with profound tremor, irritability, quadriparesis, and labored breathing since morning, on the day of admission. Arterial blood gas analysis showed severe hypokalemia. Patient's vital was stabilized and patient's oxygen saturation was maintained on oxygen inhalation. Intravenous potassium chloride infusion was administered with regular monitoring of vitals and electrolytes. Patient's symptoms improved. Thyroid function testing showed high free T3 (tri-iodothyronine) and free T4 (thyroxine) with low thyroid-stimulating hormone concentration in the serum, indicating thyrotoxic hypokalemic periodic paralysis. Treatment with antithyroid drug carbimazole resulted in an improvement during the follow-up visit. Hypokalemia is believed to be a consequence of a massive shift due to increased sodium-potassium-adenosine triphosphatase (Na⁺K⁺ATPase) pump activity in the presence of elevated thyroid hormones.

Keywords: Hyperthyroidism, hypokalemia, quadriparesis

INTRODUCTION

Hypokalaemic periodic paralysis is a very rare complication of hyperthyroidism due to Grave's disease.

We report a case of a 45-year-old male patient with thyrotoxicosis complicated with quadriparesis. This case is important because this information will help the physician to look for the cause of hypokalemia rather than implement symptomatic treatment of hypokalemia.

CASE REPORT

A 45-year-old male patient, with known hyperthyroidism since 2012 (which was revealed later), noncompliant to antithyroid drug, presented in medicine emergency ward with complaints of acute onset weakness in all four limbs since early morning. He was having easy fatigability for past 3 days. He was unable to walk or stand unassisted. On physical examination, the patient was conscious oriented, afebrile but was having profound tremor, irritability, and breathlessness. His pulse rate was 96/min regular and blood pressure was 126/78 mmHg. There was no evidence of goiter or any eye and nail bed changes. Neurologic examination revealed power of 2/5 in the upper limbs and 1/5 in both lower limbs with diminished deep tendon reflexes and flexor plantars. Higher mental function including speech, cranial nerves, sensory

system, and autonomic nervous system examination revealed no abnormality. Arterial blood gas (ABG) showed pH of 7.39, HCO₃⁻ – 18.1 mEq/L, pCO₂ – 30.6 mmHg, pO₂ – 63.1 mmHg, potassium (K⁺) – 1.58 mEq/L, chloride (Cl⁻) 95 mEq/L, and anion gap 8 mEq/L, suggestive of compensated respiratory alkalosis; however, electrocardiogram was normal. Laboratory investigation showed hemoglobin 14.9 g/L, total leukocyte count 9800 cells/cumm, polymorphs 76%, lymphocytes 23%, and platelet 1.76 lakhs cells/cumm. Random blood sugar was 109 mg/dl, sodium (Na⁺) 135 mmol/L, potassium (K⁺) 2.0 mmol/L, blood urea 40 mg/dl, and serum creatinine was 1.23 mg/dl. Liver function test showed total serum bilirubin 0.59 mg/dl and all liver enzymes were within normal limit. Serum protein was 6.7 mg/dL and serum albumin was 3.0 mg/dL. Serum ionic calcium 1.06 mmol/L (reference range: 1.1–1.35 mmol/L), total calcium 7.7 mg/dL, corrected calcium 8.5 mg/dL (reference range 8.5–10.2 mg/dL), serum magnesium 1.38 mg/dL (reference range 1.7–2.2 mg/dL), urinary Na = 48 mEq/L (normal value below 20 mEq/L), and urinary K = 26 mEq/L (normal value below 20 mEq/L). In

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the above patient due to traumatic catheterization, there was plenty of microscopic red blood cells (RBCs) in urine which could have lead to erroneously raised urinary Na^+ and K^+ due to hemolysis of RBCs. Patient may have also likely to have received diuretics by local practitioners for breathlessness. He was supplemented with intravenous (IV) potassium chloride, magnesium sulphate with cautious monitoring, and supportive management. Patient's quadriplegia improved after K^+ value returned to normal. Thyroid function test (enzyme-linked immunosorbent assay method) revealed free T3 (free tri-iodothyronine) 3.89 pg/ml (reference range: 1.71–3.71 pg/ml), free T4 (thyroxine) 10.0 ng/dl (reference range: 0.70–1.48 ng/dl), and thyroid-stimulating hormone (TSH) 0.0 $\mu\text{IU/ml}$ (reference range: 0.35–4.94 $\mu\text{IU/ml}$). There was high free T3 and T4 with low TSH in serum, indicating hyperthyroidism. Antithyroid peroxidase (TPO) antibody was 749.53 IU/ml. 99m Technetium thyroid uptake and scan shows well-visualized thyroid gland with increased trapping function favoring hyperthyroid status-Graves' disease [Figure 1]. Finally, after thorough clinical examination and laboratory workup, a diagnosis of thyrotoxic hypokalemic periodic paralysis (THPP) due to Grave's disease was made.

DISCUSSION

Our patient presented with acute onset lower motor neuron type quadriplegia without bladder bowel involvement. The differential diagnosis of such flaccid quadriplegia is Guillain-Barre Syndrome, transverse myelitis, myasthenia gravis, tick paralysis, and Botulism. Clinically transverse myelitis was ruled out due to no sensory deficit or bladder/bowel involvement. For myasthenia gravis, there was no diurnal variation, facial weakness, or drooping of eyelid. There was no history of fever or food poisoning to be suggestive of botulism. Initially, patient was suspected to be a case of Guillain-Barre' syndrome but ABG revealed hypokalemia with normal pH. Family history or previous such episode was denied by patient. In the presence of normal pH and low



Figure 1: Thyroid scan with $^{99\text{Tc}}$ pertechnetate showed a mildly enlarged thyroid gland with diffuse homogeneous radiotracer uptake, suggestive of Grave's disease

urinary K^+/Cr ratio, the differentials are familial periodic paralysis, spontaneous periodic paralysis, and thyrotoxicosis periodic paralysis. Elevated free level of T3 and T4 and a very low value of TSH confirmed the diagnosis of thyrotoxicosis periodic paralysis. Thyrotoxic periodic paralysis (THPP) is a very rare complication of hyperthyroidism with prevalence of 1 in 100,000.^[1,2] Although hyperthyroidism is most common in females, THPP has male preponderance, ratio ranging from 17:1 to 70:1.^[3] In THPP, hypokalemia is because of massive shift of potassium into the muscle cells and not due to net loss from body.^[4] This hypokalemia is triggered by biologically active thyroid hormone T3, which enter mitochondria freely and generate ATP that fuels the $\text{Na}^+\text{K}^+\text{ATPase}$ pump located in cell membranes. Increased sodium–potassium–adenosine triphosphatase ($\text{Na}^+\text{K}^+\text{ATPase}$) pump activity causes influx of K^+ .^[5] This is further aggravated by increased sympathetic activity due to thyrotoxicosis, which also stimulates $\text{Na}^+\text{K}^+\text{ATPase}$ pump and hence further worsening hypokalemia. In our patient cautiously and judiciously, treatment of IV infusion of KCL was given as there could be chances of rebound hyperkalemia due to redistribution of K^+ .^[6] Nonspecific-adrenergic blocker, propranolol in high dose (3 mg/kg), corrects hypokalemia without the risk of rebound hyperkalemia.^[7,8] Patient's power improved with correction of K^+ . Patient also was started with antithyroid drug carbimazole and discharged. On further evaluation of hyperthyroidism in this patient, there was raised anti-TPO antibody and thyroid scan was suggestive of Graves' disease.^[9] On follow-up, patient was asymptomatic and thyroid function normalized.

Consent

Patient/guardian consent was obtained.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given his consent for his images and other clinical information to be reported in the journal. The patient understand that name and initial will not be published and due efforts will be made to conceal identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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