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Hypokalemic Paralysis Due to Thyrotoxicosis

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Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

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Case Study

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ABSTRACT

Thyrotoxicosis hypokalemic paralysis is a relatively uncommon, life-threatening complication of hyperthyroidism that is most frequently seen in young Asian males but can occur sporadically in other races. The present case report discusses a 33-year-old non-diabetic non-alcoholic male admitted to the emergency room with complaints of lower limb weakness, generalized body aches, and palpitations. Laboratory investigations showed extremely low levels of potassium. An ECG showed ST depression, T-wave inversion, a prolonged Q—T interval, and the presence of a U-wave. T4 and thyroid antibody levels were found to be abnormally raised, indicating thyrotoxicosis. On a thyroid ultrasound image, enlargement of both thyroid lobes with homogenous texture and marked vascularity was noted. This hidden thyrotoxicosis led to hypokalemia which caused paralysis, palpitations, and associated symptoms. The patient was treated with intravenous potassium and other medications accordingly. The above-mentioned case was a unique presentation, one that was not commonly found in the hospital with such evident indications resulting in an absolute diagnosis. This case study will explore how the patient was diagnosed and managed with intravenous potassium to relieve hypokalemic paralysis caused by thyrotoxicosis.

Keywords: Hypokalemia; hypokalemic paralysis; hyperthyroid periodic paralysis; thyrotoxicosis hypokalemic paralysis.

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1. INTRODUCTION

Thyrotoxic periodic paralysis (TPP) is also known as thyrotoxicosis hypokalemic paralysis. TPP is a rare but potentially fatal form of hyperthyroidism that affects primarily young Asian males between the ages of 20 and 40 years [1]. It is characterized by abrupt paralytic episodes and hypokalemia in combination with hyperthyroidism [2]. The majority of TPP cases are inherited due to the disease's autosomal dominant nature. However, certain people acquire TPP as a result of thyrotoxicosis are also reported [3]. Numerous studies have reported cases of TPP in European, Indian, Hispanic, children, Malaysians, Saudis, Vietnamese, Turkish and Polynesian population [4,5]. According to studies, thyrotoxic males had a significantly higher incidence of TPP than females, despite females' higher incidence of thyrotoxicosis [4-6]. Similarly, a higher incidence of 4.3 percent was observed in thyrotoxic Japanese males and 0.04 percent in thyrotoxic female Japanese patients [7]. TPP patients typically present in the early morning hours or after a period of rest following strenuous exercise to which they are unaccustomed or a meal high in carbohydrates. The symptoms can last for hours to days and are temporary. Symptoms usually disappear rapidly if hypokalemia is corrected. As reported, TPP resolves when the patient achieves euthyroid status; at that point, either thyroidectomy or radioactive iodine is used as the definitive treatment [8]. Other treatment options for TPP include a low sodium and carbohydrate diet, the use of beta-blockers, and oral KCL [9]. The following case report describes a patient who presented with paralysis but was later diagnosed with thyrotoxicosis hypokalemic paralysis caused by his thyroid disease.

2. CASE STUDY

A thirty-three year-old non-diabetic non-alcoholic male was admitted to the emergency room due to complaints of lower limb weakness, palpitations, bilateral hand tremors, and generalized body aches. The patient worked as a shower operator and had a two-month history of anxiety. There were no associated complaints of nausea or vomiting, coughing, shortness of breath, fever, or joint pain. His vision was perfectly fine; there was no evidence of dryness of mouth or eyes. Additionally, there was no associated history of traveling or gastrointestinal/genitourinary infection. The patient also denied any intake of drugs.

2.1 Examination

The patient was examined thoroughly. On examination, his vital signs, showed a blood pressure of 130/74 mmHg, a pulse rate of 108/minute, a respiratory rate of 22/minute, and an oxygen saturation (room air) of 99%. Cardiovascular and chest examination findings were unremarkable. However, there was evidence of a palpable non-tender right lobe to his thyroid gland. Bilateral hand tremors were also observed while the patient was asked to perform fine motor tasks. Moreover, there was evidence of bilateral upper limb proximal myopathy.

On lower limb examination, hyporeflexia was found in both knees and ankles while the plantar response was equivocal bilaterally. Power was normal in both upper limbs but 2/5 in both lower limbs. Additionally, there was no sensory deficit or cranial nerve abnormalities.

2.2 Investigations

As shown in the table, laboratory findings, including hemoglobin levels, WBC, sodium, chloride, urea, eGFR Hct, MCV, PT, and INR, were all within normal limits. Potassium levels were found to be extremely low, indicating a state of hypokalemia (Table 1).

Furthermore, T4 and TPO antibody levels were extremely high, indicating a thyroid abnormality. On the other hand, TSH levels were within the normal limits. PCR testing was also performed to rule out COVID-19, and gave a negative result.

2.3 ECG Findings

An ECG demonstrated some typical features of hypokalemia, which can be seen below in the electrocardiogram.

The ECG revealed an ST-segment depression, a T-wave inversion, a prolonged Q-T interval, and the presence of a U-wave.

2.4 Thyroid Ultrasound

On thyroid ultrasound imaging, the right lobe of the thyroid gland (5.1 X 2.1 X 2.5cm, volume 15 mL) appeared enlarged with notable inhomogeneous decreased parenchymal texture and increased vascularity. However, there was no evidence of significant nodules in the right

lobe. Similarly, the left lobe (measured 4.7 X 2.4 X 2.5 cm, volume (15mL)) was enlarged with an inhomogeneous decreased parenchymal texture and increased vascularity. No evidence of significant underlying nodules was observed on the left lobe. The carotid sheath was clear, and there was no sign of the enlargement of lymph nodes, but the isthmus of the thyroid gland seemed enlarged (measures about 0.6cm in AP view).

Table 1. The following lab investigations were done to confirm the diagnosis

Event	Result	Ref. Range
K Ven POC	1.7 mmol/L	(3.4 - 5.1)
Na Ven POC	142 mmol/L	(136 - 145)
Gluc Ven POC	8.6 mmol/L	(3.9 - 6.0)
Lac Ven POC	1.5 mmol/L	(0.5 - 2.2)
Sodium Lvl	* 138.0 mmol/L	(135.0 - 145.0)
Potassium Lvl	* 2.0 mmol/L	(3.6 - 5.1)
Chloride Lvl	* 107.0 mmol/L	(98.0 - 107.0)
CO2	* 21.6 mmol/L	(22.0 - 29.0)
Creatinine	* 46.0 micromol/L	(62.0 - 106.0)
Urea Lvl	* 4.30 mmol/L	(0.00 - 8.30)
eGFR (CKD-EPI)	* 140 mL/min/1.73m2	(>=60 -)
WBC	4.96 x10^9/L	(4.00 - 11.00)
RBC	5.34 x10^12/L	(4.50 - 6.50)
Hgb	147.0 g/L	(130.0 - 180.0)
Hct	0.431 L/L	(0.400 - 0.540)
MCV	80.7 fL	(76.0 - 96.0)
Potassium Lvl	* 5.0 mmol/L	(3.6 - 5.1)
Chloride Lvl	* 107.0 mmol/L	(98.0 - 107.0)
CO2	* 21.4 mmol/L	(22.0 - 29.0)
Creatinine	* 48.0 micromol/L	(62.0 - 106.0)
Urea Lvl	* 4.20 mmol/L	(0.00 - 8.30)
TSH	<0.005 milli IU/L	(0.270 - 4.200)
PT	11.2 sec(s)	(11.0 - 14.5)
INR	0.99	(0.90 - 1.20)
APTT	20.8 sec(s)	(28.0 - 40.0)
Anticoagulant?	None	
Anticoagulant?	None	
T4 Free	>100.000 pmol/L	(12.000 - 22.00(
Thyroglob Ab	31.2 kIU/L	(- <=115.0)
TPO Ab	>600.0 kIU/L	(- <=34.0)

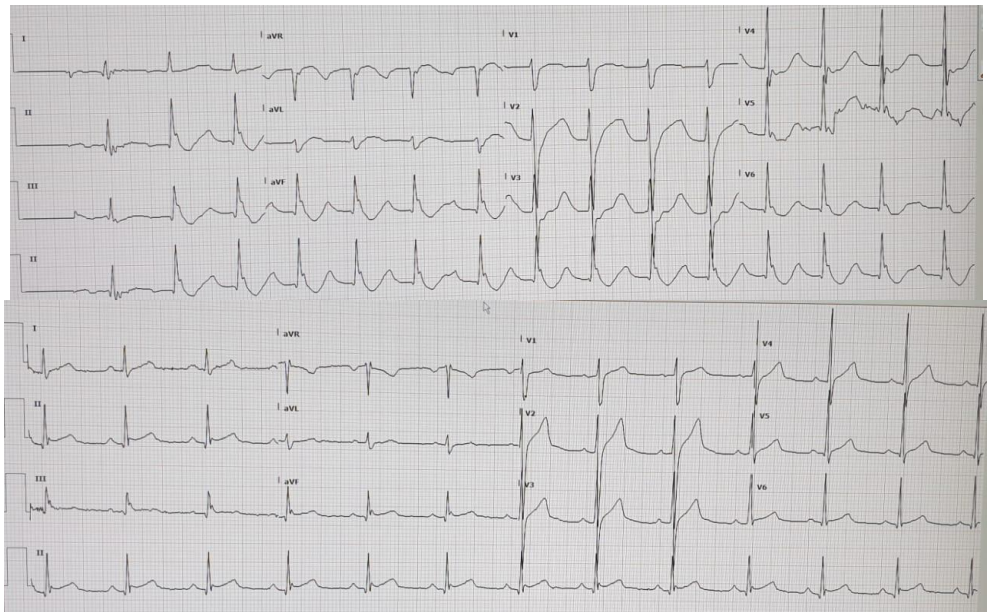

Fig. 1. ECG after correction of hypokalemia

2.5 Diagnosis and Management

From the above-given reports, it was clear that the patient was suffering from thyrotoxicosis hypokalemic paralysis as the thyroid Ab antibodies and T4 levels were very elevated. Similarly, potassium levels were extremely low, indicating a state of hypokalemia. The patient had also developed paralysis, and there were associated symptoms of palpitations, body ache, and tremors which can be caused by low potassium levels because of thyrotoxicosis. This verified the ultimate diagnosis as thyrotoxicosis hypokalemic paralysis caused by thyrotoxicosis-induced hypokalemia.

As a result of this diagnosis, the patient's first care strategy included intravenous potassium, which led to the correction and complete resolution of fatigue, improved power in both lower limbs up to grade 5, and a normalization of his ECG within 36 hours. The patient was also referred to an endocrinologist, and a combination of carbimazole 45mg/day and 40 mg propranolol was prescribed to the patient. To improve potassium levels naturally, the patient was also recommended potassium rich diet such as leafy vegetables, potatoes, bananas, dry fruits, yogurt, and chicken. These diets are rich in potassium that help body meeting its natural needs. As expected, the outcomes were favorable, resulting in a significant improvement in the patient's symptoms and overall condition.

3. DISCUSSION

A 33 year-old male presented to ER with complaints of lower limb weakness, palpitations, and body aches. The patient was in a relatively stable condition. TPP diagnosis was made based on clinical manifestations and blood test results suggestive of hypokalemia and hyperthyroidism [10], including extremely low potassium levels, and abnormally high T4 and thyroid antibody levels. The severity of paralysis was connected to the degree of hypokalemia, not the thyroid hormone levels or hyperthyroidism's clinical signs/symptoms [11]. As paralysis can occur due to low potassium levels, and if low potassium levels are left untreated, cardiac arrhythmias and severe weakness of breathing muscles can follow, leading to the death of the patient [12]. However, previous studies have described low potassium levels and mildly increased serum thyroid hormones levels in TPP cases. According to Ko et al. [13], only 10% of individuals develop minor thyrotoxic symptoms. Therefore, TPP should be differentiated from other causes of acute paralysis, including Guillain-Barré syndrome, familial hypokalemic periodic paralysis, spinal cord compression, and myasthenic crises [12].

Similarly, an ECG showed ST depression, prolongation of Q-T interval, T-wave inversion, and the presence of a U-wave, typical of low potassium levels [14]. Hypokalemia occurs in TPP as a result of a huge, rapid movement of potassium from the extracellular to the intracellular compartments, mostly into the muscles. Thyroid hormone can increase Na+–K+ ATPase in skeletal muscle by increasing the transcription of the gene encoding the Na+–K+ ATPase and by increasing the membrane insertion of the pump and the pump's intrinsic activity [15]. The well-recognized activators of Na+–K+ ATPase include a high carbohydrate diet, strenuous exercise to which an individual is unaccustomed, high insulin levels, and high thyroid hormone levels. Other risk factors for TPP include increased salt intake, surgery, trauma, prolonged rest after an accident, alcohol, exposure to cold, emotional stress, and drug use such as laxatives, diuretics, amphotericin B, and estrogens [16,17]. Unfortunately, no such risk factor was found in this case, but there was a prominent elevation of thyroid hormone levels. Additionally, androgens increased the activity of the Na+ K+ ATPase pump. This resulted in hypokalemia and the following paralysis [18]. The main treatment of TPP includes immediate potassium replacement medication, either orally or intravenously, depending on the severity of the hypokalemia. However, if potassium is given between attacks, it cannot prevent acute paralysis [11]. TPP is mostly treated by controlling hyperthyroidism with antithyroid medications, beta-blockers, radioiodine ablation, or thyroidectomy [19]. It is necessary to avoid precipitating factors.

4. CONCLUSION

TPP is a potentially fatal complication of hyperthyroidism that mostly affects Asian men between the ages of 20 and 40. A patient who suffers from TPP presents with symmetrical muscle weakness, particularly of lower limbs, with diminished reflexes and palpitations. If it is left untreated, this condition can cause severe weakness in breathing muscles and cardiac arrhythmias, eventually leading to the death of the patient. Therefore, early diagnosis and management can help in the prevention of

severe cardiac complications. This condition can be caused by high thyroid levels or genetic mutations. However, people who have a diet high in carbohydrates and sodium, do intense exercise, and have higher levels of insulin are at more risk of developing this condition. The current mainstay of treatment of TPP is immediate potassium administration, use of beta-blockers, hyperthyroidism medications, and radioiodine ablations. There have been cases of disease recurrence, but because the condition is rare and the symptoms are frequently kept under control, the same treatment approach is often sufficient to control these flare-ups.

CONSENT

As per international standard or university standard, patients' written consent has been collected and preserved by the author(s).

ETHICAL APPROVAL

It is not applicable.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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