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Case Report

Subclinical hyperthyroidism presenting with hypokalemic periodic paralysis in the emergency department

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Abstract

Subclinical hyperthyroidism (SH) is a condition in which blood levels of Thyroxine (T4) and Triiodothyronine (T3) are normal in the presence of low levels of thyroid-stimulating hormone (TSH). Patients with SH have either no symptoms or mild nonspecific symptoms. Hypokalemic periodic paralysis (HPP) is one of the rare presentations of overt hyperthyroidism; however, only a few cases are reported to date to occur with SH. This case report presents a case of a young Asian male who was admitted to the emergency department (ED) with paralysis of the lower extremities that progressed to the upper extremities and neck muscles in 2 days. Clinically, he was euthyroid; however, his thyroid profile revealed normal levels of T3 and T4 with undetectable TSH levels. This case report aims to add to the limited literature on SH that presents with HPP in the ED.

INTRODUCTION

Hypokalemic periodic paralysis (HPP) is classified into two forms: familial and sporadic. Familial HPP is a rare autosomal dominant channelopathy that causes low potassium levels in the blood and results in episodic transient attacks of muscular paralysis [1]. However, sporadic HPP has several causes, such as hyperthyroidism, renal tubular acidosis, hyperaldosteronism, Gitelman syndrome, cocaine, diuretics, steroids and alcohol ingestion. Although a wide spectrum of thyroid disorders has been reported to be present with HPP, hyperthyroidism is the most implicated, with Graves' disease being the most common cause [2]. HPP secondary to subclinical hyperthyroidism (SH) is a rare but potentially lethal clinical entity. The diagnosis could be delayed because the rarity of such presentation and might lead to unnecessary investigations and consultations in the ED. Here we present a case of a young man who had quadriparesis for 2 days. Despite his unclear clinical picture, lab testing revealed hypokalemia and SH. This case report aims to present that HPP, though rare, might be associated with SH.

CASE REPORT

A man in his 20s, without previous illness, was diagnosed with quadriparesis for 2 days. He had a progressive disability involving his thigh muscles which eventually progressed to the upper limbs and neck muscles in two days. Initially, he noticed difficulty walking, which progressed to buttoning his shirt and holding his neck. The possible trigger was physical exercise in the gym which he started 3–4 days before developing the symptoms. He denied a history of fever, sore throat, coryza, diarrhea, and urinary or fecal incontinence. He denied using steroids, diuretics, supplements, alcohol or illicit drugs. He had no history of spinal cord injury, trauma or tick bite. He had no history of stress or anxiety and did not have any other underlying medical conditions. The family history was unremarkable.

On physical examination, he had a blood pressure of 140/83 mm Hg, a heart rate of 89 beats/min, a respiratory rate of 22 breaths/min, oxygen saturation of 99% on room air and an axillary temperature of 37.6°C. There were no visible or palpable neck swelling, tremors, palmar sweating or other clinical signs of hyperthyroidism. The cardiovascular and respiratory examination was unremarkable. Neurological examination revealed the power of 3/5 in the hip flexors and 4/5 in the flexors and extensors of the legs bilaterally, whereas the power in the upper limbs was 3/5 in all the muscle groups bilaterally. The biceps, triceps and supinator reflexes of the upper limbs as well as the knee and ankle jerk reflexes of the lower limbs were normal bilaterally. The plantar reflexes were down-going bilaterally. Additionally, neither any sensory deficit nor any evidence of dysautonomia was observed.

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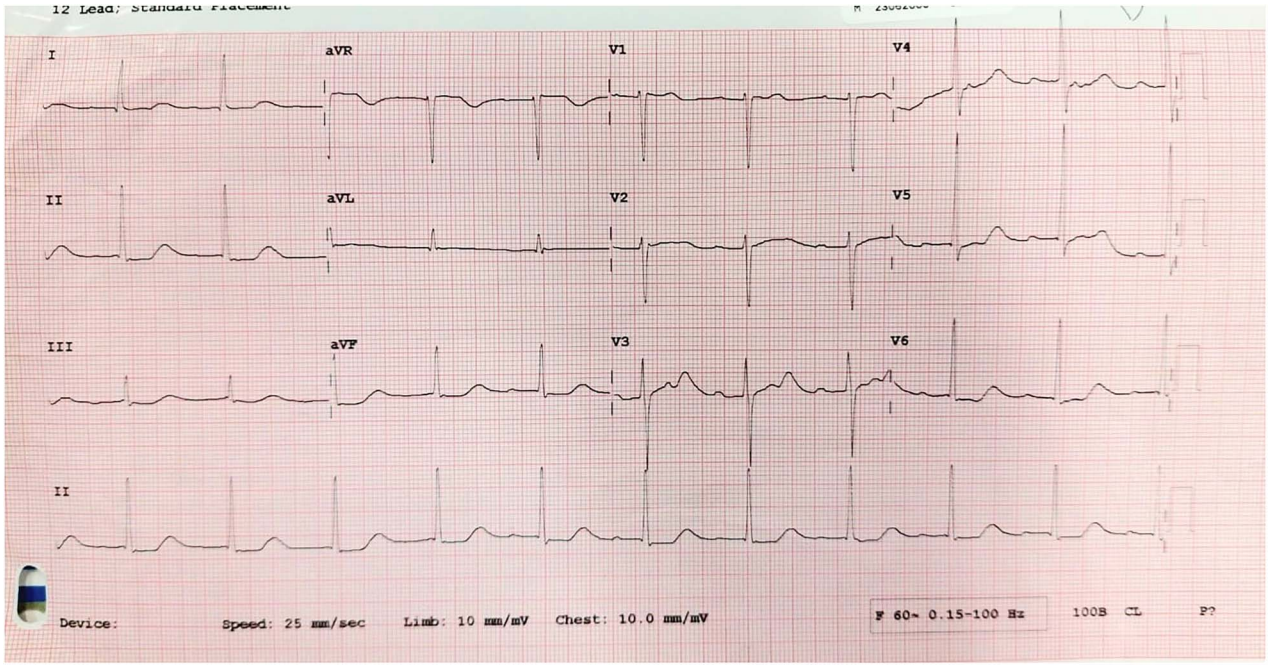

Figure 1. ECG showing a normal sinus rhythm with a prolonged PR interval, a depression of the ST segment and a prominent U wave.

An electrocardiogram (ECG) showed normal sinus rhythm with prolonged PR interval, ST segment depression and prominent U waves (Fig. 1). The laboratory data are summarized in Table 1.

His blood workup showed severe hypokalemia (1 meq/L) and metabolic acidosis. Further study revealed a low spot urinary potassium to creatinine ratio (0.14), suggesting a low urinary potassium excretion rate. Considering the ambiguous clinical picture, a thyroid profile was sent which showed normal T3 levels of 3.90 pg/ml (normal range 2.1–4.4 pg/ml) and slightly raised T4 levels of 2.33 ng/dl (normal range 0.89–1.76 ng/dl) with undetectable thyroid-stimulating hormone (TSH) levels of < 0.01 IU/ml (normal range 0.4–4.2 IU/ml).

The provisional diagnosis of HPP was made in the context of SH and persistently low serum potassium. He was treated with intravenous and oral potassium replacement, nonselective beta-blockers and anti-thyroid medication until optimal potassium levels (>3.5 meq/L) were achieved. His functional ability improved within 24 h of treatment with the regaining of muscle power back to normal. The potassium levels remained within normal limits (up to 3.6 meq/L) consistently on follow-up lab workup. The causes of transcellular potassium shifting were evaluated. He was not taking beta-agonists, insulin, aminophylline or theophylline. Ketoacidosis was ruled out based on a urine detailed report, blood gas analysis and normal blood sugars. Guillain Barre Syndrome was ruled out clinically as the patient didn't have areflexia or dysautonomia and the weakness resolved with potassium correction. However, thyroid antibodies checked on follow-up showed normal Serum Anti Thyroglobulin levels of < 20 IU/ml (normal < 40 IU/ml) with increased Anti-thyroid Peroxidase levels of 66.3 IU/ml (normal < 35 IU/ml). Radionuclide thyroid scintigraphy was performed, which showed evidence of enhanced tracer uptake by functioning parenchyma of both thyroid lobes and Gamma camera-based thyroid uptake was 4% (normal 0.5–3.5%), suggesting diffuse toxic goiter. Based on positive thyroid antibodies and radionucleotide thyroid scintigraphy findings, a diagnosis of Graves' disease was made.

DISCUSSION

HPP is a rare manifestation of SH and is prevalent in the Asian population with a higher predilection for men than for women. The patients usually present with progressive muscular weakness; however, it is easily overlooked owing to the subtle symptoms of SH [2, 3]. Hypokalemia could be life-threatening if not immediately addressed, as it can lead to cardiac arrhythmias and paralysis of the respiratory muscles [4, 5]. Normal serum potassium levels are maintained by a constant shift between the intracellular and extracellular compartments [6]. The triggers that increase intracellular potassium shifting such as strenuous exercise, carbohydrate-rich meals or diuretic use are responsible for causing hypokalemia and resulting in paralysis [4]. Hyperthyroidism has been linked more closely to HPP, and Graves' disease is the most common cause [6]. Structured as catecholamines, thyroid hormones induce a hyperadrenergic state and stimulate beta-adrenergic receptors that increase the amount and activity of Na +/K + ATPase pumps, thereby decreasing extracellular potassium levels [7, 8].

Attacks begin with proximal muscle weakness gradually progressing to quadriparesis; however, the sensory system is never involved. Skeletal muscles are solely involved, and attacks are more common during the day [9]. The diagnosis is based on a good history, examination findings and their correlation with biochemical investigations. Immediate intravenous potassium replacement and beta-blockers use during acute attacks have shown marked improvement, but normal potassium levels can only be maintained once the patient is euthyroid [1, 2]. Hence, the main aim should be to control hyperthyroidism with antithyroid medications and avoidance of the trigger.

Although HPP is more common in hyperthyroidism, this case report adds that it can also occur in SH. To date, this is probably the first case of HPP secondary to SH with potassium of 1 meq/L. A high index of suspicion among emergency physicians is necessary to diagnose this condition because of covert signs and symptoms of SH.

Table 1. Summary of laboratory data

Laboratory test	Day 1	Day 2	Day 3	Reference range
Hemoglobin	15.1	12.5	13.4	12.3–16.6 g/dl
Hematocrit	44.8	37.0	40.0	38.4–50.7
WCC	21.3	12.0	9.3	4.8–11.3 × 10 E9/L
Platelets	248	179	171	154–433 × 10 E9/L
Potassium	1.0	3.9	3.6	3.5–5.1 mEq/L
Sodium	142	146	144	136–145 mEq/L
Creatinine	3.0	2.1	2.1	0.9–1.3 mg/dl
BUN	36	31	27	6–20 mg/dl
Magnesium	2.0			1.6–2.6 mg/dl
Bicarbonate	16.4	19.3	24.6	20–31 mmol/l
Chloride	108	113	108	98–107 mmol/l
Calcium	10.7	8.7		8.6–10.2 mg/dl
Troponin I	58			0–57 ng/l
TSH	<0.01			0.4–4.2 uIU/ml
FT4	2.33			0.89–1.76 ng/dl
FT3	3.90			2.1–4.4 pg/ml
Total bilirubin	0.4			0.1–1.2 mg/dl
Alkaline phosphatase	65			45–129 IU/l
Aspartate transaminase	14			<35 IU/l
Alanine transaminase	30			<45 IU/l
Prothrombin time	10.5			9.3–12.8 s
APTT	24.4			22.9–34.5 s
INR	1.0			0.9–1.2
Urinary potassium	4	10		Mmol/L
Urinary sodium	53	68		Mmol/L
Urinary creatinine	27	20		Mg/dl

Laboratory data showing elevated WCC, deranged creatinine, low bicarbonate, normal T3, T4, undetectable TSH and elevated spot urinary creatinine.

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None.

CONFLICT OF INTEREST STATEMENT

No conflicts of interest.

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ETHICAL APPROVAL

Standards concerning publication ethics were observed and the study was approved by the hospital's ethical review committee.

CONSENT

The patient gave consent to the publication.

GUARANTOR

I, Dr Nirdosh Kumar, accept official responsibility for the overall integrity of the manuscript and attest that all statements in the manuscript are true to his knowledge.

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