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A Case of Thyrotoxic Periodic Paralysis (TPP) with Severe Hypokalemia: Diagnostic Challenges and Management

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ABSTRACT

Background and Objectives: Thyrotoxic periodic paralysis (TPP) is a life threatening complication of hyperthyroidism, characterized by sudden onset muscle weakness and profound hypokalemia. Its dignosis is delayed, mainly in non-Asian populations or in the absence of overt thyrotoxic symptoms. This case report aims to found the diagnostic and therapeutic challenges of TPP and fortify the importance of early recognition. Case Presentation: A 37 year old Asian male presented with acute flaccid quadriparesis and severe hypokalemia (K 1.14 mEq,L). Initial ECG show sinus tachycardia and QTc prolongation. He had no known thyroid disease, but the presentation raised suspicion for TPP. Hypomagnesemia and thyrotoxicosis detected in laboratory findings. The patient was treated with IV and oral potassium, magnesium sulfate, and propranolol, which result in rapid clinical improvement. He was discharged with plans of long-term management of thyrotoxicosis. This case indicate the pathophysiology of TPP of thyroid hormone induced Na,K ATPase hyperactivity leading to intracellular potassium shifts. Conclusion: TPP should be considered in all patients presenting with acute muscle weakness and hypokalemia, regardless of ethnicity or overt thyroid symptoms. To prevent morbidity and recurrence, early diagnosis, cautious potassium repletion, beta-blocker therapy, and definitive treatment of thyrotoxicosis are essential.

Keywords: Thyrotoxic periodic paralysis, hypokalemia, hyperthyroidism, muscle weakness, potassium

1. INTRODUCTION

Thyrotoxic periodic paralysis (TPP) is generally described as an acquired form of hypokalemic periodic paralysis in which episodes of acute muscle weakness occur in the setting of thyrotoxicosis and are accompanied by low serum potassium (Barahona et al., 2009; Basnet et al., 2023). Epidemiologic observations from several reports indicate that TPP is strongly influenced by ethnicity and sex. Basnet et al., (2023) note that TPP is common in East Asian populations, with an incidence of about 2% among patients with thyrotoxicosis, and Bilha et al., (2020) state that the highest incidence is seen in Asian populations. At the same time, Elston et al., (2007) describe thyrotoxic hypokalaemic periodic paralysis as a cause of muscle weakness

affecting only a small minority of hyperthyroid patients, but highlight Polynesians as an ethnic group at particular risk.

Several authors stress the marked male predominance: Atrash et al., (2024) report that, although hyperthyroidism is more frequent in women, TPP occurs more often in men and typically between 20 and 40 years of age, while Bilha et al., (2020) remark that the condition was classically confined to young Asian men, even though it can occur regardless of age, sex or race. Cases described in Caucasian and other non-Asian patients, including those by Barahona et al., (2009) Hannon et al., (2009) Abbasi et al., (2018) and Florescu et al., (2024) underline that TPP should also be considered outside the traditional high-risk groups. From a pathophysiologic perspective, Basnet et al., (2023) describe TPP as a form of hypokalemic periodic paralysis associated with hyperthyroidism, in which patients develop acute proximal weakness with hypokalemia that can progress to involve all four limbs and even the respiratory muscles.

Abbasi et al., (2018) explain that the pathogenesis involves increased β-adrenergic responsiveness, leading to up-regulation of the Na^+,K^+-ATPase pump and a shift of potassium into cells rather than true depletion of total body potassium. Barahona et al., (2009) also comment that the high incidence in Asians and the association with HLA antigens support a genetic predisposition, although the precise mechanism remains incompletely defined. In line with this concept of transcellular potassium shift, Alziadat and Ismail (2020) describe TPP precipitated by urethral dilatation and note that situations associated with catecholamine or insulin surges can trigger attacks.

Barahona et al., (2009) report attacks progressing from leg weakness to quadriparesis and note that bulbar and respiratory muscles are rarely affected, yet their involvement may occur. Hannon et al., (2009) emphasize that, although the paralysis is temporary, unrecognized episodes of severe hypokalemia may lead to respiratory muscle paralysis or cardiac arrest. Florescu et al., (2024) highlight that severe hypokalemia can result in paralysis or dangerous cardiac arrhythmias, and Abbasi et al., (2018) present a case complicated by thyrotoxic cardiomyopathy, illustrating the cardiovascular impact of the syndrome.

Several authors underline the diagnostic difficulty of TPP. Barahona et al., (2009) state that TPP is often not recognized at the first attack because of its very low prevalence in Caucasians and the usually mild symptoms of hyperthyroidism. Bilha et al., (2020) explicitly present TPP as a misleading challenge in the emergency setting, noting that it can be confused with renal, gastrointestinal or other neuromuscular disorders.

Management across these reports focuses on rapid but careful potassium replacement, combined with non-selective β-blockade and definitive control of the thyrotoxic state. Basnet et al., (2023) state that the acute phase is corrected by aggressive potassium repletion together with a non-specific β-adrenergic blocker such as propranolol, while Abbasi et al., (2018) highlight the risk of rebound hyperkalemia if potassium is replaced too rapidly and emphasize the role of propranolol and antithyroid therapy. Atrash et al., (2024), Alziadat and Ismail (2020), and others similarly stress that long-term prevention of recurrent paralysis requires achieving a euthyroid state. Within this context, our case of TPP with severe hypokalemia and marked electrocardiographic abnormalities illustrates the diagnostic challenges and management considerations described in the current literature. Table 1 and 2 present case reports from previous studies.

Table 1. Reported cases of thyrotoxic periodic paralysis (TPP) in the literature

Citation	Country, ethnicity	Age, sex	Underlying thyroid disease	Triggers, context	Lowest documented K^+ (mmol, L)	Neuromuscular & major complications
Lulsegged et al., 2011	UK; Chinese and Caucasian patients	47-year-old Chinese man; 28-year-old Caucasian man	Thyrotoxicosis; Graves' disease confirmed in the second case (TSH-receptor antibodies positive)	Recurrent acute lower-limb weakness, paralysis; onset on waking or at night; specific precipitating factors not clearly stated	3.1 (Chinese case); 2.6 (Caucasian case)	Flaccid lower-limb or quadriparesis with intact sensation; no respiratory failure or malignant arrhythmias; full recovery after potassium, β-blocker and antithyroid therapy, plus thyroidectomy or radioiodine
Jung et al., 2014	South Korea; Korean	16-year-old male adolescent	Graves' disease (diffuse goiter, hypervascularity,	Two early-morning episodes of	2.7 during first paralytic episode	Acute, reversible flaccid weakness confined to lower extremities; no

			positive antibodies)	lower-leg paralysis; second episode after breakfast; no family history of neuromuscular disease		respiratory or cardiac instability reported; symptom-free for one year on β-blocker and antithyroid therapy once hyperthyroidism controlled
Barahona et al., 2009	Spain; Caucasian	37-year-old man	Graves' thyrotoxicosis (diffuse homogeneous radioiodine uptake)	Recurrent attacks over 4 months, starting during sleep or post-exercise rest; mild hyperthyroid symptoms (weight loss, tremor, heat intolerance)	2.3 (with mild hypomagnesemia 1.53 mg,dL)	Attacks ranged from mild proximal leg weakness to flaccid quadriparesis; normal sensation, normal ECG during documented episode; complete remission of paralysis after potassium replacement and subsequent euthyroidism with propranolol and methimazole
Basnet et al., 2023	USA; Asian ethnicity	27-year-old male	Newly diagnosed Graves' disease (TSH <0.01, high free T4,T3, positive thyroid-stimulating immunoglobulin and TPO antibodies)	Recurrent episodes precipitated by drinking beer and physical exertion; current attack on awakening with inability to move all four limbs and dyspnea	1.3	Acute symmetric flaccid quadriparesis (0,5 power) with preserved sensation; associated hypomagnesemia and hypophosphatemia; marked QTc prolongation (503 ms) on ECG; rapid improvement after aggressive potassium,magnesium replacement, IV propranolol and methimazole
Bilha et al., 2020	Romania; Caucasian	Young adult male (36 years) and two Caucasian women (elderly and middle-aged)	Graves' hyperthyroidism in all three cases (positive TRAb and,or typical ultrasound)	Case 1: acute malaise and rash after fast food and hydrocortisone; cases 2–3: hypokalemic paralysis without obvious precipitant reported	1.2 (case 1); 2.7 (cases 2 and 3)	Sudden flaccid paralysis with severe or refractory hypokalemia; case 1 complicated by complete AV block and malignant ventricular arrhythmias (VT,VF) requiring defibrillation; neurological and cardiac recovery after potassium repletion, antithyroid drugs, propranolol and, in one case, thyroidectomy
Soneji et al., 2021	USA; Vietnamese (Asian)	48-year-old man	Profound hyperthyroidism (TSH <0.01; very	Out-of-hospital ventricular fibrillation arrest	1.9	Initial presentation with VF cardiac arrest; later recurrent ventricular

			high free T4 and T3; diffuse goiter on imaging)	following low-speed single-vehicle collision; received amiodarone and iodinated contrast before recognition of thyrotoxicosis		arrhythmias (SVT, nonsustained VT, PVCs) in setting of severe hypokalemia; potassium repletion and β-blockade plus antithyroid therapy led to resolution of arrhythmias and neuromuscular symptoms
Shields, 2015	USA; African American	43-year-old woman	Graves' disease; non-adherence to antithyroid and β-blocker therapy	Recurrent bilateral lower-extremity weakness and dyspnea; prior admissions for similar complaints	2.8 (with phosphorus 2.4 mg, dL)	Symmetric lower-extremity weakness progressing to acute respiratory arrest despite initially normal oxygenation and ABG; telemetry showed sinus tachycardia and short runs of nonsustained VT; required intubation and mechanical ventilation; recovery after potassium, propranolol and methimazole
Seshadri et al., 2004	USA; half Native American, half African American	53-year-old man	Untreated hyperthyroidism (24-h radioiodine uptake 60%; thyroid tests consistent with thyrotoxicosis)	Bilateral lower-extremity weakness in context of known but untreated thyrotoxicosis; no family history of periodic paralysis	Hypokalemia (exact value not specified in the text used)	Acute flaccid leg weakness due to hypokalemia from intracellular K^+ shift; rapid resolution after potassium, non-selective β-blocker and propylthiouracil; authors highlight rarity of TPP in Native American,African American populations
Martini et al., 2024	USA; Latin American	30-year-old man	Graves' disease with moderate diffuse goiter and exophthalmos	Recurrent episodes of weakness over weeks; prior severe hypokalemic paralysis thought to be worsened by alcohol; current event after inconsistent methimazole,propranolol use	Described as severe hypokalemia (exact value not specified in the text used)	Progressive lower-limb and girdle weakness leading to inability to walk; rapid improvement within hours after potassium and magnesium replacement plus re-initiation of methimazole and propranolol; authors stress risk of thyroid storm and importance of adherence

Table 2. Diagnostic features, acute management and key lessons from reported TPP cases

Citation	Diagnostic clues and challenges	Acute management	Long term management and main outcome
Lulsegged et al., 2011	Episodes of profound reversible limb weakness with hypokalemia and suppressed TSH; Chinese and Caucasian patients, showing TPP is not confined to Asians; subtle or absent thyrotoxic signs in one case	Potassium replacement, non selective β blocker (propranolol), high dose carbimazole; in first case, potassium iodide and lithium to achieve rapid euthyroidism before thyroidectomy	Definitive treatment with thyroidectomy (case 1) or radioiodine (case 2) prevented further attacks; authors emphasize high suspicion in acute flaccid paralysis with hypokalemia and possible KCNJ18 mutation
Jung et al., 2014	Adolescent with recurrent morning leg paralysis, normal neuro exam between attacks, low K^+ and suppressed TSH with elevated free T4,T3; TPP as first and sole manifestation of Graves' disease	Initial potassium correction during episodes; subsequent β adrenergic blockade and antithyroid drugs once hyperthyroidism documented	Good control of Graves' disease with antithyroid therapy led to complete disappearance of paralytic attacks; authors stress considering TPP even in children,adolescents with acute paralysis and hypokalemia
Barahona et al., 2009	Four month history of nocturnal or post exercise flaccid weakness, predominantly in lower limbs, with hypokalemia and mild hyperthyroid symptoms; diagnosis delayed due to low prevalence in Caucasians	IV potassium chloride during documented attack; ECG and electrolyte monitoring	Methimazole and propranolol to treat Graves' disease; after euthyroidism, no further TPP episodes; thyroid function should be checked in acute paralysis with hypokalemia regardless of ethnicity
Basnet et al., 2023	Young Asian male with acute quadriparesis, dyspnea, severe hypokalemia, hypomagnesemia, hypophosphatemia and QTc prolongation, with suppressed TSH and high free T4,T3; history of brief episodes after beer and exertion	ICU admission; aggressive potassium and magnesium repletion; IV propranolol; initiation of methimazole after confirmation of Graves' disease	After treatment, power normalized within hours and no recurrent paralysis was reported; article highlights TPP as key differential in young Asian males with acute paralysis and severe hypokalemia to avoid fatal arrhythmias
Bilha et al., 2020	Three Caucasian patients with sudden flaccid paralysis and severe or refractory hypokalemia as first presentation of hyperthyroidism; minimal thyrotoxic signs in some cases	IV potassium chloride, continuous ECG and electrolyte monitoring; early antithyroid drugs and non selective β blockers; resuscitation and defibrillation in the patient with malignant ventricular arrhythmias	Restoration of euthyroidism (medical therapy or thyroidectomy) prevented recurrence; thyroid testing is mandatory in hypokalemic paralysis and combined K^+ repletion plus propranolol and antithyroid therapy is needed
Soneji et al., 2021	Out of hospital VF arrest with no coronary disease, later found to have severe hypokalemia and marked hyperthyroidism with diffuse goiter; raised suspicion for TPP in an Asian man	Aggressive potassium repletion; β blocker and antithyroid medication; intensive care management of recurrent ventricular arrhythmias	Hypokalemia, ventricular arrhythmias and muscle paralysis were reversible once hyperthyroidism was treated; authors highlight TPP as a reversible cause of life threatening ventricular arrhythmias
Shields, 2015	African American woman with uncontrolled Graves' disease, recurrent leg weakness, hypokalemia and eventual acute respiratory failure; underrecognized TPP outside classic Asian male profile	Potassium replacement, non selective β blocker (propranolol), methimazole; intubation and mechanical ventilation; telemetry guided management of nonsustained VT	After control of thyrotoxicosis and electrolyte correction, she was weaned from ventilation and discharged; report underscores that TPP can cause respiratory failure and should be suspected in any hyperthyroid patient with weakness and hypokalemia
Seshadri et al., 2004	Middle aged man of mixed Native American,African American ancestry with untreated hyperthyroidism and acute flaccid lower limb paralysis due to hypokalemia; rarity of TPP in	Potassium supplementation, non selective β adrenergic blockade and propylthiouracil; correction of hypokalemia led to rapid improvement	Once the thyrotoxic state was treated, paralysis did not recur; authors conclude that TPP, although rare in these populations, should be considered in hypokalemic flaccid paralysis

	this ethnic background		
Martini et al., 2024	Latin American man with Graves' disease presenting with recurrent acute muscle weakness, moderate goiter, exophthalmos and severe hypokalemia; high Burch Wartofsky score indicating risk of thyroid storm	ED management with potassium and magnesium repletion and re initiation of methimazole and propranolol; close monitoring for thyroid storm and ECG abnormalities	Rapid symptomatic improvement within hours; case highlights increasing recognition of TPP in non Asian populations and the importance of adherence to definitive hyperthyroidism treatment to prevent recurrence

2. CASE PRESENTATION

Patient History & Clinical Finding

A 37-year-old male presented to the emergency department with muscle weakness that started suddenly four hours prior to arrival, limiting his ability to walk or stand. He had no significant medical history, no known thyroid disorders, hypertension, or neuromuscular diseases. He did not have any trauma, hard exercise, or recent illness before his symptoms started, but he did have a similar episode a few months earlier for which he got medications but couldn't remember their names. No family history of periodic paralysis or thyroid disease. On examination the patient high blood pressure (166,77 mmHg) and sinus tachycardia (120 minute). The ECG showed sinus tachycardia with first degree AV block, criteria for left ventricular hypertrophy, and a nonspecific ST T abnormality (Fig. 1, 2, and 3). Neurological examination show a generalized weakness in all four limbs, with muscle strength 2.5 in both the upper and lower extremities. Deep tendon reflexes were reduced. The assessment of the cranial nerves was normal, with no ptosis or bulbar weakness. The patient awake and aware (GCS 15.15). The genitourinary examination was normal. These results, in addition to clinical findings, make Guillain-Barré syndrome, and myasthenia gravis less probable. When we considered the sudden onset of weakness with high blood pressure and a fast heart rate, TPP became a diagnostic possibility.

Diagnostic Assessment & Management

The first tests in the lab showed very low potassium levels, with a venous blood gas (VBG) potassium level of 1.14 mEq,L on admission. This level went up to 1.6 mEq,L on a repeat test within the first hour. Later tests showed that it had gotten better to 1.9 mEq,L. There was also concurrent hypomagnesemia (1.4 mg,dL), which made the risk of cardiac arrhythmias even higher. Clinical suspicion of TPP led to the ordering of thyroid function tests, as noted in the first assessment. The repeat ECG showed sinus tachycardia (118 bpm), QTc 559 ms, voltage criteria for LVH, and T-wave inversions (Fig. 1, 2, and 3). These findings are more likely due to chronic thyroid hormone excess than to a primary heart problem. Because the patient had severe hypokalemia and unstable blood flow, he was put in the ICU for continuous heart monitoring. The diagnosis was TPP; the lack of a family history of paralysis excluded familial periodic paralysis, and support the TPP diagnosis.

Management included intravenous potassium chloride (40 mEq over two hours), oral potassium supplementation (50 mEq), and magnesium sulfate (2 g IV) to stop arrhythmias. Propranolol (20 mg) was given to stop potassium shifts caused by catecholamines and control tachycardia. This was a very important step in TPP because it can block the effects of thyroid hormones at the cellular level. An endocrinology consultation was requested for the management of thyrotoxicosis.

Transthoracic echocardiography revealed a left ventricle of normal size, with normal wall thickness and maintained systolic function. Transmitral Doppler flow exhibited a pattern indicative of compromised left-ventricular relaxation. The size and systolic function of the right ventricle were normal, as were the sizes of the left and right atria. All of the heart's valves were in good shape, and there was only a small amount of tricuspid regurgitation. There was no sign of an atrial septal defect, a pericardial effusion, or a pleural effusion.

Outcome & Follow-up

The patient showed quick clinical improvement within hours of treatment. Muscle strength went from 4,5 to 4.8 mEq,L, potassium levels stabilized at 4.8 mEq,L, and magnesium levels went from 1.8 mg,dL to 1.8 mg,dL. The last ECG showed that tachycardia (HR 95 bpm) and QT prolongation (QTc 395 ms) resolved, but the voltage criteria for LVH still present. The ECG showed that there were no structural problems. The patient was sent home with plans for follow up with an endocrinologist.

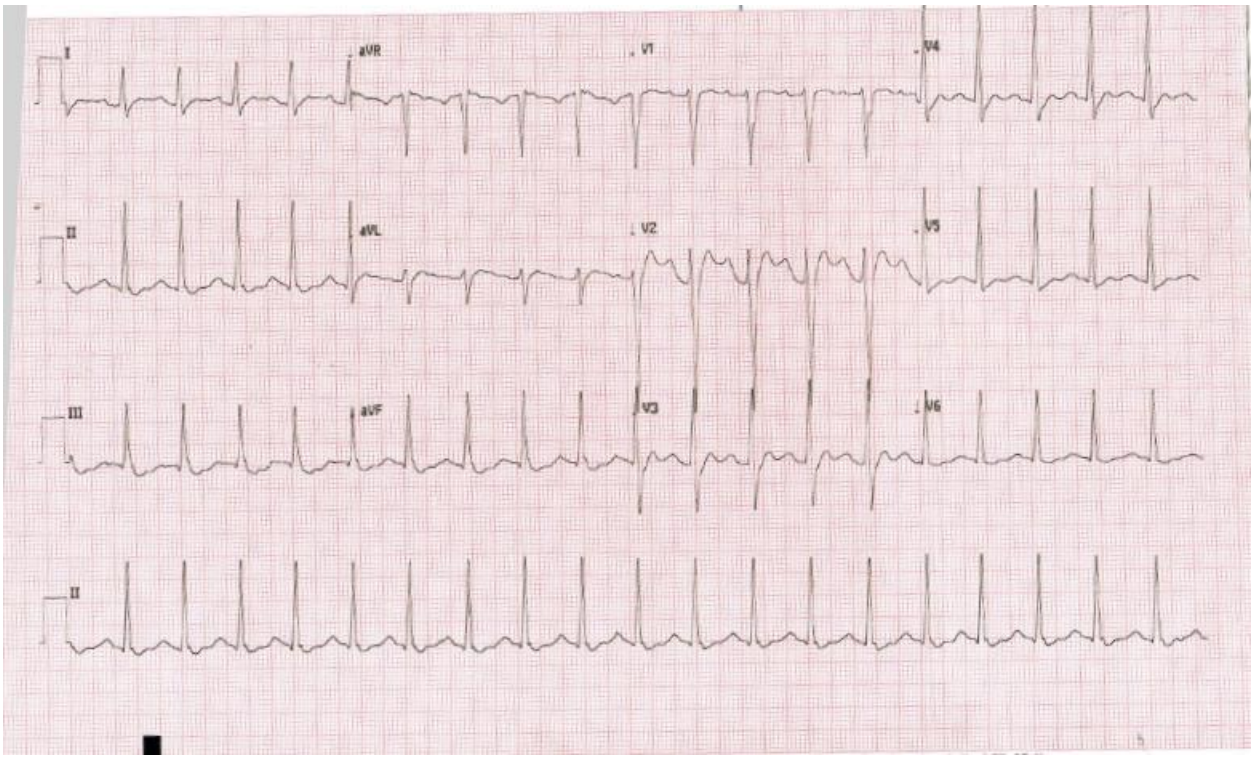

Figure 1: Electrocardiogram demonstrating sinus tachycardia with first-degree atrioventricular block. Additional findings include possible right atrial enlargement (0.25 mV P-wave), possible left ventricular hypertrophy, and nonspecific ST and T wave abnormalities.

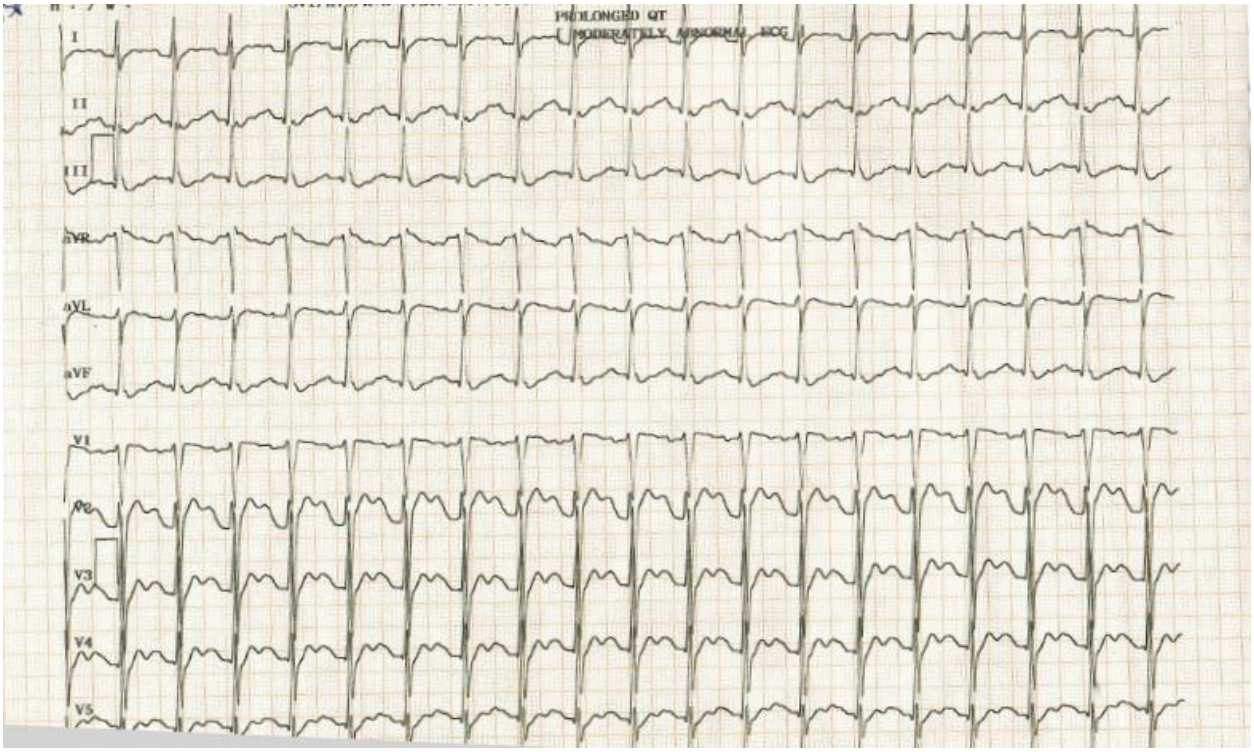

Figure 2: Electrocardiogram demonstrating sinus tachycardia (HR: 100-130 bpm), normal axis, left ventricular hypertrophy, T-wave inversions, possible myocardial ischemia (inferior), and prolonged QT interval.

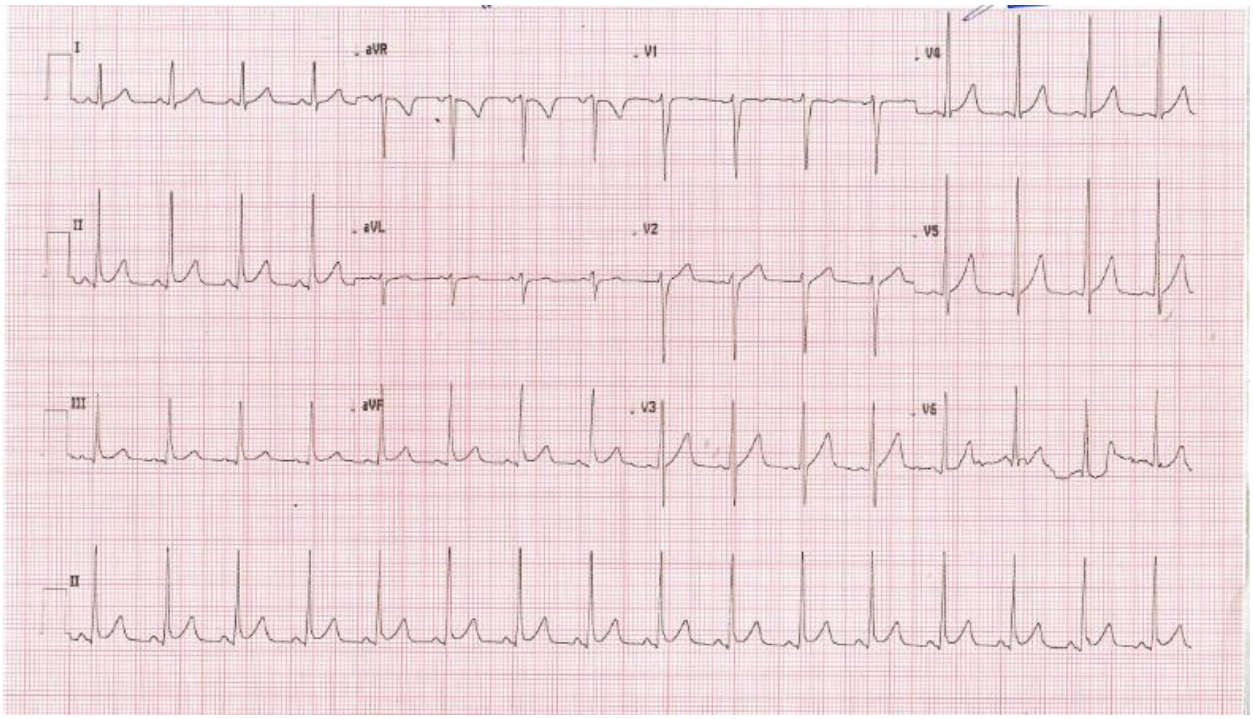

Figure 3: Electrocardiogram demonstrating sinus rhythm with short PR interval and criteria for left ventricular hypertrophy.

3. DISCUSSION

TPP is a neurological emergency presented as acute hypokalemia and muscle weakness in patients with hyperthyroidism (Martini et al., 2024). This case of a 37-year-old Asian male with potassium levels of 1.14 mEq,L and flaccid quadriparesis show the critical importance of recognizing TPP amidst more common neuromuscular mimics.

Our patient hypomagnesemia (1.4 mg,dL) align with the severe depletion (1.6 mg,dL) reported by Alziadat & Ismail (2020) and Basnet et al., (2023), whereas Barahona et al., (2009) found only borderline depletion (1.53 mg,dL), which indicate magnesium role in modulating paralysis severity. The initial QTc prolongation (559 ms) were similar to Basnet et al.'s (2023) findings (503 ms) and Alziadat & Ismail's (2020) reported QT abnormalities, while Barahona et al., (2009) observed a normal ECG, indicate the spectrum of cardiac manifestations despite similar biochemical derangements. All these cases, including our case, shared Na,K ATPase hyperactivity as the central mechanism.

Yoon & Raza's (2022) potassium rebound (1.7 to 5.7 mEq,L post-treatment) indicate the risks of overcorrection. The Graves' disease etiology (confirmed in Alziadat & Ismail (2020), Basnet et al., (2023), and Barahona et al., (2009)) and rapid response to beta-blockers (seen within hours in our patient, Basnet et al.(2023), and Yoon & Raza (2022)) support TPP thyrotoxic basis, while Barahona et al.'s (2009) long term methimazole success (Barahona et al., 2009) emphasizes definitive thyroid control as the cornerstone of prevention.

TPP is difficult to be diagnosed to its mimicry of other neuromuscular emergencies, need careful differentiation from conditions like Guillain-Barré syndrome and myasthenia gravis. Our presentation initially evaluation for these disorders, indicate the diagnostic dilemma described in Bilha et al., (2020), who show that TPP often goes unrecognized instead of its life threatening potential, mainly in non Asian populations, including Caucasian males and elderly women (Bilha et al., 2020). In our patient, the absence of sensory deficits and bulbar involvement argued against Guillain-Barré syndrome. Sinha and Raghupathy (2020) show that TPP spares these neurological functions, not like Guillain-Barré syndrome which commonly affects them. The rapid potassium correction with clinical improvement in our case provided further confirmation, similar to with Bilha et al., (2020), where recovery occurred after potassium repletion and beta-blocker therapy, though some cases show persistent hypokalemia for up to 5 days. Sinha and Raghupathy (2020) indicate the role of genetic mutations and catecholamine driven Na,K ATPase hyperactivity in TPP pathogenesis, which support the need for thyroid testing even without overt thyrotoxicosis.

The management of TPP include several therapeutic approaches with debates in treatment strategies. The combination of intravenous potassium and propranolol administered to our patient show current therapeutic paradigms. Jung et al., (2014) report a successful treatment of a 16 year old patient using propranolol with methimazole (5mg initially), with complete symptom resolution

and no recurrence at 1 year follow up. Lulsegged et al., (2011) described two cases where carbimazole plus propranolol prevented TPP recurrence, and identify potassium channel mutations as a contributing factor to TPP pathogenesis. Our patient's treatment was consistent with these approaches, though specific potassium correction rates weren't addressed in either study. The addition of magnesium sulfate in our case supported by Florescu et al., (2024), who found a successful resolution of paralysis (from 1.8 mEq,L to 4 mEq,L potassium within 12 hours) using combined magnesium sulfate (5mL of 25% solution) and potassium replacement in a Graves' disease patient with TPP. This effect can be attributed to magnesium's ability to stabilize KCNJ18-encoded potassium channels which is similar to Lulsegged et al.'s (2011) genetic findings, while inhibiting catecholamine driven Na,K ATPase hyperactivity, addressing both the channelopathy and thyrotoxic components of TPP pathophysiology.

Cardiac complications in TPP range from transient arrhythmias to life threatening events, which need vigilant electrocardiographic and metabolic monitoring. In our patient, the evolution from sinus tachycardia with prolonged QTc to normal sinus rhythm (95 bpm) with QTc normalization (395 ms) show the reversible cardiac effects of TPP. While our patient show transient repolarization abnormalities, Soneji et al., (2021) found a more severe presentation, ventricular fibrillation cardiac arrest with arrhythmias at potassium levels of 1.9 mmol,L, indicate TPP related cardiac instability.

TPP indicate important ethnic and demographic variations that influence diagnostic recognition. While it has been described in Asian males, guidelines suggest wider demographic distribution than previously recognized. Cases in Caucasian, African, and Middle Eastern populations, show identical pathophysiological mechanisms and clinical presentations (Cope et al., 2013; Elston et al., 2007; Shields 2015; Hannon et al., 2009; Seshadri et al., 2002; Aldasouqi et al., 2009; Atrash et al., 2024). This demographic expansion challenges traditional teaching about TPP epidemiology and indicate the importance of considering this diagnosis regardless of patient ethnicity. The condition's occurrence in diverse populations indicate that genetic susceptibility factors can be more widely distributed than initially believed, and environmental triggers play a greater role in non-Asian cases.

4. CONCLUSION

The association between thyroid dysfunction and neuromuscular manifestations in TPP play a significant diagnostic and therapeutic challenges for healthcare professionals. The pathophysiology of this condition, significant intracellular potassium fluctuations without depletion, results in severe transient hypokalemia that result in life threatening complications. The absence of common hyperthyroidism symptoms in many cases, with its occurrence in diverse demographic groups, obstructs diagnosis and intervention.

Limitations and strength

This case report provides a clinical description of TPP, including detailed laboratory results, ECG evolution, and neurological evaluations. The discussion give controversies regarding potassium repletion and the use of betablockers. The findings are limited by a single-case generalizability, mainly concerning non-Asian populations or atypical presentations, and the absence of advanced diagnostics, genetic testing or circulating microRNA profiling. The short follow up period makes it hard to draw conclusions about long-term recurrence risk and heart health outcomes. The aggressive potassium repletion approach worked in this case, but it goes against standard guidelines and may not work for everyone.

Implications

This case report highlights critical factors for clinicians to evaluate TPP. Any patient presented with acute, unexplained weakness requires immediate assessment of potassium levels and thyroid function, regardless of demographic characteristics or the presence of overt hyperthyroid symptoms. Management requires a multidisciplinary approach that includes emergency doctors, endocrinologists, and often cardiologists to deal with both acute electrolyte imbalances and thyroid dysfunction that has been going on for a long time. This case reminds us that TPP doesn't happen very often, but needs to be thought about in all cases of sudden paralysis to avoid bad outcomes. Future research should focus on elucidating the genetic and molecular mechanisms that underlie TPP to improve treatment and outcomes.

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Informed consent

The patient consented to the publication of photographs. Written & Oral informed consent was obtained from individual participants included in the study.

Ethical approval

Not applicable. This article does not contain any studies with human participants or animals performed by any of the authors.

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Conflict of interest

The authors declare that they have no conflicts of interests, competing financial interests or personal relationships that could have influenced the work reported in this paper.

Data and materials availability

All data associated with this study will be available based on reasonable request to the Corresponding Author.

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