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Thyrotoxic Periodic Paralysis with Sensory Deficits in Young African American Male: A Case Report and Literature Review

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Abstract

Thyrotoxic periodic paralysis is a sporadic entity characterized by hypokalemia and paralysis in the setting of hyperthyroidism. TPP is most commonly described in young Asian males. Studies have shown an association with mutations affecting inward rectifying potassium channels. The pathophysiology involves Na^+-K^+- ATPase channel causing an increased intracellular shift of potassium ions in the hyperthyroid state and in the presence of another precipitating condition. Most cases of thyrotoxic periodic paralysis are defined in young Asian males of 20–40 years of age, here we present an interesting case of thyrotoxic periodic paralysis in 32-year-old African American male, who presented with sudden onset weakness in the bilateral lower extremity and left upper extremity. Interestingly, the patient also has sensory deficits, a feature not known to be associated with thyrotoxic periodic paralysis.

Keywords

thyrotoxic periodic paralysis; hypokalemia; african american

1. Introduction

Thyrotoxic periodic paralysis is a sporadic entity that only occurs in association with thyrotoxicosis. It is different from hypokalemic periodic paralysis that has an autosomal dominant pattern of inheritance (one of the sub-types of familial periodic paralysis). The pathophysiology always involves increased ionic transport via Na K ATPase channel activity [1,2]. Certain ion channels defects are found to be associated with TPP. In one study, 10 out of 30 Caucasians or Brazilian patients with TPP had a mutation in gene encoding KCNJ18 (kir2.6), an inward rectifying potassium channel expressed in skeletal muscle [2,3]. Similar mutations have been found in another study in the patient population from Singapore with TPP [3]. Another novel gene is identified in population from Taiwan negatively regulating KCNJ2 (Kir2.1) expression [4]. All these channelopathies along with increased Na K ATPase activity have been shown to cause a positive feed-forward cycle of hypokalemia in the presence of hyperthyroid state and an additional precipitant accentuating the ion

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transport [1,2,5,6,7,8,9]. While most cases of TPP are described in young Asian males [1,2,5,8], in this report we present a very interesting case of TPP in a young 32-year-old African American male.

2. Case Presentation

A 32-Year-old male with no significant past medical history presented to the emergency department with complaints of sudden onset bilateral lower extremity weakness as well as weakness in the left upper extremity. Along with the motor weakness patient also had sensory deficits in right upper extremity with dysarthria and aphasia. On presentation, the patient was afebrile, tachycardic to 110 BPM, blood pressure was in 120/80 mmHg and respiratory rate was 14 Breaths/minute. Stroke code was initiated, and the patient was evaluated by neurology. Physical exam was unremarkable except for neurological exam which was significant for 1/5 strength in bilateral lower extremities, 2/5 strength in left upper extremity and sensory deficits in right upper extremity with complete loss of fine sensations. Further history taking revealed that the patient was drinking fifteen beers the night before. On review of systems, the patient reported having lost weight in the last year with periodic palpitations, episodes of diarrhea, sweating, insomnia and appetite changes. Patient denies any history of similar symptoms in the past. No recent history of infections, tick bites, vaccines, sick contact or travel.

Initial labs were significant for potassium of 2.5 mmol/L (3.5–5.0) on the comprehensive metabolic panel and venous blood gas shock panel with a potassium of 2.1 mmol/L and a pH of 7.326. His thyroid stimulating hormone (TSH) was <0.005 mIU/L (0.27–4.20) with a free Thyroxine of 3.94 ng/dl (0.7–1.9). Rest of the laboratory workup was found to be unremarkable. Endocrinology was consulted, and his constellation of symptoms was attributed to thyrotoxic periodic paralysis likely precipitated by heavy drinking the day before. Thyroid stimulating immunoglobulin and thyroid peroxidase antibody were sent which were subsequently found to be 18.20 IU/L (<1.3) and 1935 IU/ml (<35) respectively. CT scan head and CTA head and neck showed no evidence of acute pathology. The patient was given 40 mEq IV potassium along with 40 mEq oral potassium and his symptoms resolved. The patient was started on methimazole 20 mg twice a day and propranolol 20 mg three times a day. Serial basic metabolic panels were done to monitor rebound hyperkalemia in the setting of beta-blockade from propranolol. The patient was admitted to telemetry monitoring and potassium was monitored over the next 24 hours. The patient was discharged the next day with follow up appointments to the endocrine clinic. The patient was also counseled extensively on alcohol abstinence.

3. Initial Labs

Table 1.

Complete Blood Count

White blood cells (/nl)	6.61 (4.5–10.9)
Red Blood cells (/pl)	5.71 (4.20–6.10)
Hemoglobin (g/dl)	15.7 (14.0–18.0)

Hematocrit %	46.9 (42.0–52.0)
Platelets (/nl)	281 (130–400)

Table 2.

Basic Metabolic panel

Sodium (mmol/l)	142 (136–146)
Potassium (mmol/l)	2.5 (3.5–5.0)
Chloride (mmol/l)	104 (98–106)
Bicarbonate (mmol/l)	22 (24–31)
Blood urea nitrogen (mg/dl)	14 (6–20)
Creatinine (mg/dl)	0.59 (0.70–1.20)
Blood glucose (mg/dl)	134 (70–99)
Calcium (mg/dl)	9.38 (8.6–10.0)
Total protein (g/dl)	6.6 (6.0–8.5)
Albumin (g/dl)	4.0 (3.3–6.1)
AST (U/L)	29 (10–50)
ALT (U/L)	19 (0–41)
ALP (U/L)	144 (35–145)
Bilirubin (mg/dl)	0.35(0.0–1.2)

4. Imaging

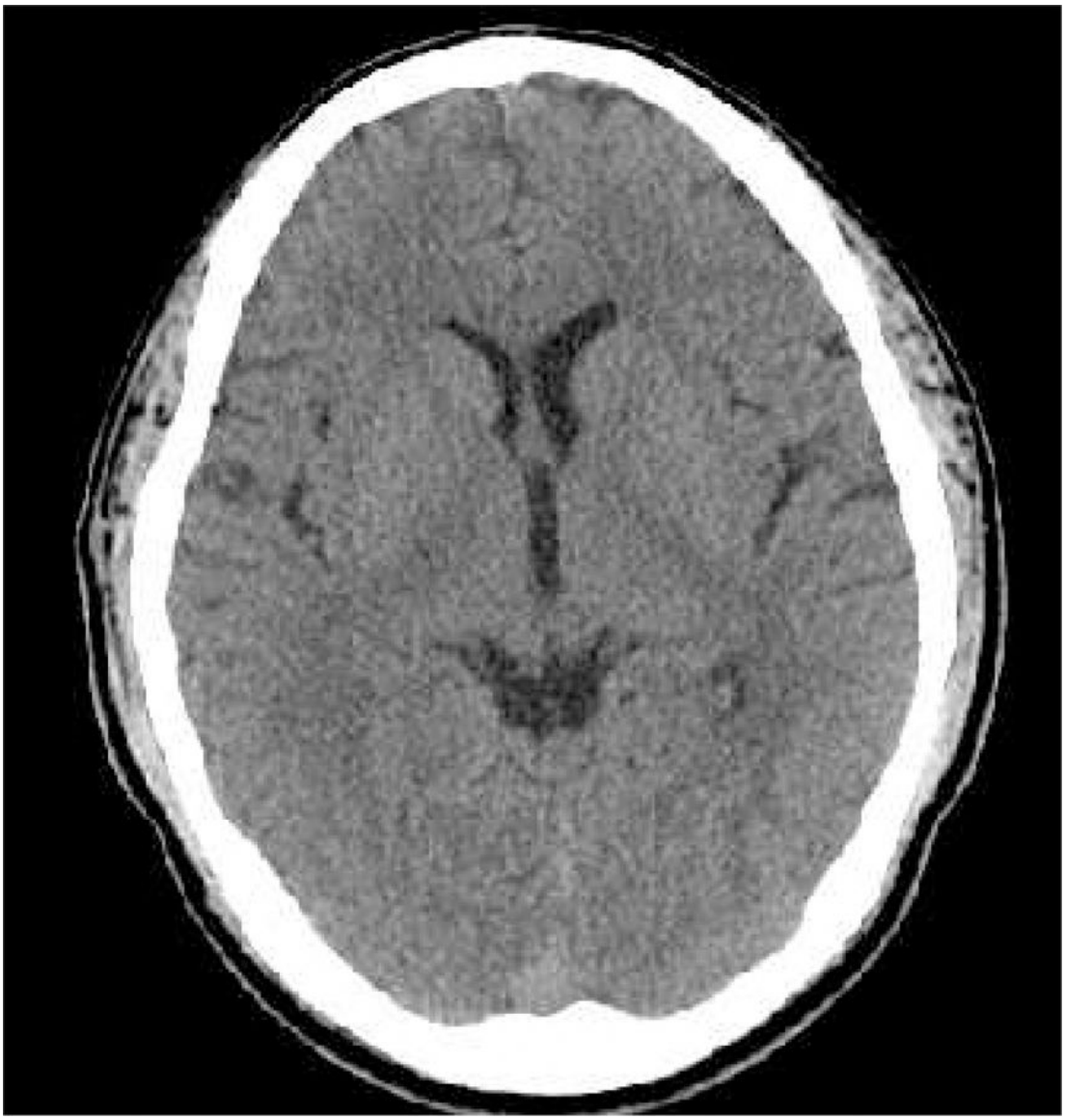

Figure 1. Head CT scan:
CT scan of the head showed no acute infarction, masses or hemorrhage. No acute intracranial abnormality is identified

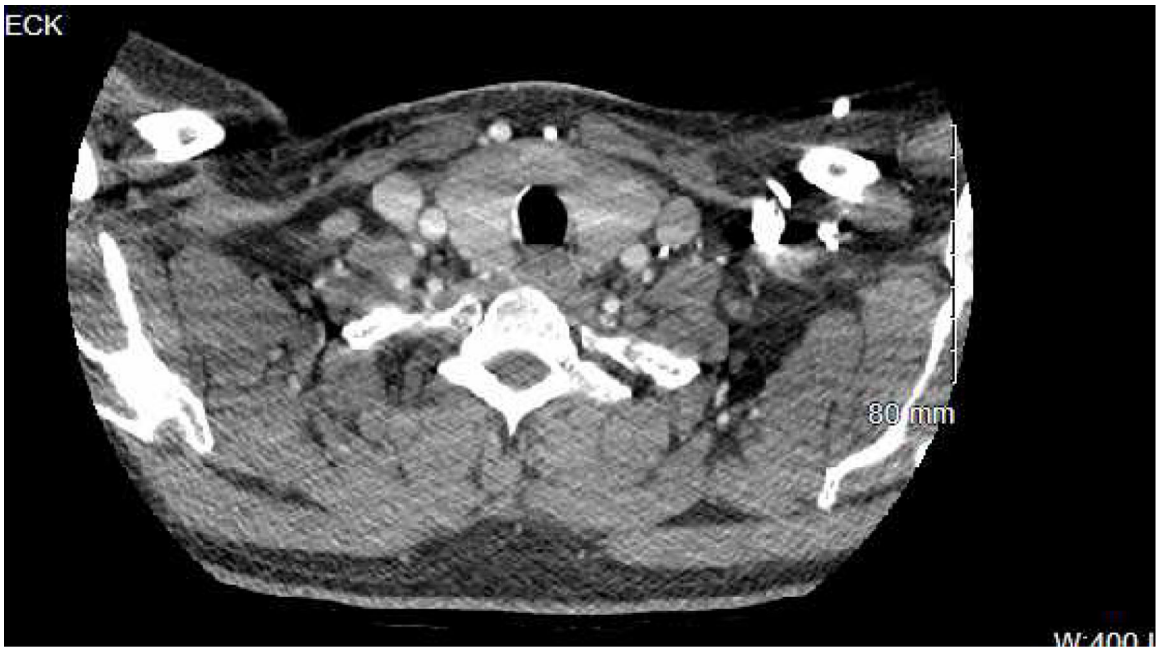

Figure 2. CTA Head and Neck:

CTA HEAD: No large vessel occlusion, central high-grade stenosis, aneurysm, or vascular malformation. 2. There are dental caries including in the left first maxillary premolar with a periapical dentigerous cyst. CTA neck:1. No significant atherosclerotic disease of the carotid bifurcation/proximal ICA. No hemodynamically significant stenosis, according to NASCET criteria. 2. No evidence of dissection, pseudoaneurysm, or vascular malformation. Carotid Stenosis Reference Using NASCET Criteria Mild: <50% stenosis Moderate: 50–69% stenosis Severe: 70–94% stenosis Near occlusion: 95–99% stenosis Occluded: 100% stenosis

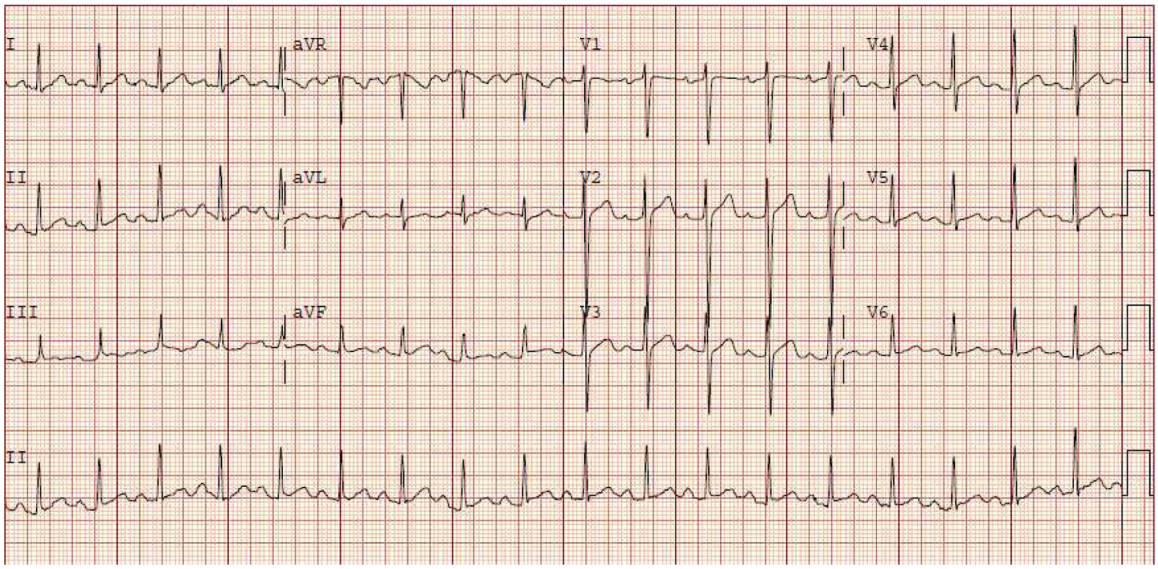

Figure 3.
EKG showed sinus tachycardia with HR of 109 BPM and QTc of 431ms with no appreciable T wave changes

5. Case Discussion and Literature Review

TPP is a rare presenting feature of the thyrotoxic state. The pathophysiology involves up-regulation of Na-K ATPase leading to an increased intracellular shift of potassium ions in hyperthyroid state and most often in the presence of another precipitant [1,2,5,6,7,8,9]. At the same time, efflux of potassium ions cannot take place because of decreased outward Kir current as explained by certain mutations [2,3,4,9,13,14]. Hyperthyroidism also leads to increased adrenergic drive leading to up-regulation of Na-K ATPase [1,9]. Any precipitant that leads to increased catecholamine surge or hyperinsulinemia ultimately cause increase Na-K ATPase pump activity expressed on skeletal muscles, liver, and kidneys due to increasing B2 adrenergic receptor activity [1,10,11,12]. Increased B2 activity causes an increase in cyclic adenosine monophosphate that further leads to increase protein kinase A. Increase protein kinase A is responsible for phosphorylation thus increasing pump affinity for intracellular Na. At the same time, insulin and catecholamine inhibit inward rectifier potassium channels in skeletal muscles which are normally responsible for efflux of potassium [13,14]. Many mutations responsible for Periodic Paralysis are described in association with inward rectifier channels which explains why an additional mechanism to prevent efflux of potassium is also needed to produce the effect [2,3,4,9,13,14]. The activity of Na-K ATPase alone cannot define the pathophysiology.

Table 3.

Demographics, laboratory values for TPP patients based on our literature review of 15 case reports:

Case Report	Age	Ethnicity	Potassium levels (mmol/l)	TSH levels (unit)	Free T4 (unit)
Tella et al. [1]	28	Hispanic	1.3 (3.5–5.1)	<0.05 (0.34–5.60) (IU/ml)	5.81 (0.6–1.60) (ng/ml)
Tella et al. [1]	26	Hispanic	1.2 (3.5–5.1)	0.05 (0.34–5.60)(IU/ml)	6.57 (0.6–1.60) (ng/ml)
Lulsegged et al. [17]	47	Chinese	3.1	<0.01 (0.35–5.50) (mU/L)	38.5 (9.4–22.7) (pmol/L)
Lulsegged et al. [17]	28	Caucasian	2.6	<0.01)0.35–5.50) (mU/L)	54 (12–22)(pmol/l)
Hagel et al. [8]	32	Turkish	1.2 (3.5–5.0)	<0.01 (0.27–4.20) (μIU/ml)	3.2 (0.9–1.7)pg/ml
Belayneh et al. [18]	26	Ethopian	2.7 (3.6–5.5)	0.0005 (0.27–4.2)(IU/ml)	5.33 (0.93–1.71) (ng/dl)
Naqi et al. [19]	20	Chinese	3.1	0.06 μIU/ml	2.6 ng/dl
Zumo et al. [20]	41	Hispanic	2.3	0.01	37.5
Bo Oh et al. [21]	25	Korean	2.42	0.00 (0.3–5.0)(μIU/ml)	2.38 (0.75–2.00) (ng/dl)
Thethi et al. [22]	25	Caucasian	1.7 (3.5–5.0)	<0.005 (0.4–4.5)(μU/ml)	4.2 (0.7–1.9)(ng/dl)
Barahona et al. [23]	37	Caucasian	2.3 (3.5–5.1)	<0.03 (0.25–5.0)μU/ml	3.14 (0.77–1.71) (ng/dl)
Lam et al. [24]	33	Hispanic	1.7 (3.6–5.0)	<0.02 (0.50–6.80) (mU/L)	4.56 (0.89–1.76) (ng/dl)
Meseeha et al. [25]	19	Caucasian	1.9 (3.5–5.1)	<0.02 (0.47–4.68) (mIU/L)	5.5 (0.8–2.2)(ng/dl)
Hegde et al. [26]	32	Asian	2.3 (3.5–5.5)	<0.005 (0.5–4.4)(mU/L)	12.8 (12–22) (pmol/L)
Hakami et al. [27]	28	Middle Eastern	2.0	<0.005 mIU/L	39.7 pmol/L
Sehmer et al. [28]	48	Filipino	2.3 (3.5–5.2)	< 0.01	64.4 (10–21)
Jung et al. [29]	16	Korean	2.7	<0.025 (0.35–4.94) (mU/L)	2.10 (0.70–1.48) (ng/dl)

In the above-mentioned case, increased alcohol intake likely led to increased catecholamine secretion causing an intracellular shift of potassium [1,2,15]. Also, excessive alcohol intake results in hyperinsulinemia which along with hyperthyroidism and hyperadrenergic state leads to low serum potassium [1,10,11,12,13,14,16,18]. Hypokalemia leads to hyperpolarization of skeletal muscles groups thus making them non-responsive to neuronal impulses leading to loss of contractility.

Almost all cases of TPP are associated with muscle weakness, with the proximal muscle groups being affected more than the distal muscle groups. In the above-mentioned case, the patient also presented with sensory deficits, not typically linked with TPP. The complete resolution of symptoms with potassium supplementation and anti-thyroid regimen supports the diagnosis of thyrotoxic periodic paralysis in our reported case.

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