กรณีศึกษาอิทธิพลของฮอร์โมนไทรอยด์ที่มีผลต่อกล้ามเนื้อ: ผู้ป่วยชายไทยอายุน้อยที่มาด้วยอาการอ่อนแรง และมีหัวใจห้องบนเต้นเร็วร่วมกับภาวะสัญญาณไฟฟ้าถูกขัดขวางแบบสมบูรณ์

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บทคัดย่อ

ผู้ป่วยชายอายุ 24 ปี มีอาการกล้ามเนื้ออ่อนแรงเฉียบพลันโดยเฉพาะกล้ามเนื้อส่วนต้น ซึ่งสาเหตุที่พบบ่อยของกล้ามเนื้อส่วนต้นอ่อนแรง ได้แก่ ภาวะโพแทสเซียมในกระแสเลือดต่ำ, กล้ามเนื้ออักเสบทั้งจากการติดเชื้อและสาเหตุที่ไม่ได้เกิดจากการติดเชื้อเช่น ความผิดปกติของฮอร์โมน (เช่น ภาวะพร่องไทรอยด์ฮอร์โมน และภาวะไทรอยด์เป็นพิษ) ยาต่างๆ (สเตียรอยด์และแอลกอฮอล์) ผู้ป่วยรายนี้ได้รับการวินิจฉัยว่าเป็นอัมพาตเป็นระยะจากต่อมไทรอยด์เป็นพิษ (thyrotoxic periodic paralysis) และผลการตรวจคลื่นไฟฟ้าหัวใจ(electrocardiogram) แสดงให้เห็นว่ามี atrial tachycardia with 2:1 atrioventricular (AV) conduction สลับกับ complete AV block.

คำสำคัญ

Thyrotoxicosis, hypokalemia, Atrioventricular block, Atrial tachycardia

Case report

Forceful hormone under your neck affects your legs: A young adult man sudden weakness and atrial tachycardia with transient complete AV block.

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Abstract

We are demonstrating a case of a 24-year-old man presented with acute symmetrical proximal muscle weakness in both upper and lower extremities. Common causes of proximal muscle weakness include hypokalemic periodic paralysis, acquired myopathy, both from infection and non-infection, endocrinopathy (e.g., hypothyroidism and hyperthyroidism, drugs (steroid and alcohol). He was diagnosed with thyrotoxic periodic paralysis. Moreover, his electrocardiography showed atrial tachycardia with 2:1 atrioventricular (AV) conduction and transient complete AV block.

Keyword

Thyrotoxicosis, hypokalemia, atrioventricular block, atrial tachycardia

Introduction

Thyrotoxic periodic paralysis (TPP) is an alarming and potentially lethal complication of hyperthyroidism characterized by muscle paralysis and hypokalemia due to a massive intracellular shift of potassium. This condition mainly affects male Asian patients. Many affected patients do not have obvious symptoms and signs of hyperthyroidism. Typical electrocardiography (ECG) of TPP was rapid heart rate, high QRS voltage, and first-degree atrioventricular (AV) block.1 Immediate therapy with potassium supplementation can prevent serious cardiopulmonary complications and may hasten recovery of the periodic paralysis. Effective control of hyperthyroidism is indicated to prevent the recurrence of paralysis.2 Here, we described a case of TPP who presented with atrial tachycardia and transient complete AV bock, which is an atypical ECG of TPP.

case presentation

A 24-year-old Thai man presented with acute symmetrical proximal muscle weakness in both upper and lower extremities. One day before the incidence, he reported strenuous exercise without adequate rehydration followed by a large meal of high carbohydrate dinner. In the morning after he woke up, he complained of difficulties in getting up and standing up, without a sign of breathing or speech difficulties. He denied any history of fever, nausea, vomiting nor diarrhea. Apart from feeling palpitant from time to time and unintentional weight loss of 5 kilograms over 3 months, he was otherwise previously healthy. He denied using alcohol and illicit drugs. He denied neither prescribed drug nor herbal use.

Upon arrival, his body temperature was 36.5 $^{\circ}$C, respiratory rate was 16 times per minute, pulse rate was 80 beats per minute, and blood pressure was 130/60 mmHg. His general appearance was alert, mildly anxious, and well cooperative. His conjunctivae were pink without exophthalmos. Diffused thyroid gland enlargement was noted with bruit heard upon auscultation. He had full and regular pulse. No murmur was heard. His trachea was in the midline, without chest retraction and abnormal breathing. His skin was dry, with no pretibial myxedema, no tremors, no thyroid acropathy nor onycholysis. He was alert. His cranial nerves were all

intact. He had proximal muscle weakness in both upper and lower extremities without tenderness, hypotonia was noted without muscle atrophy. His deep tendon reflexes were 1+ all. His sensation to pain, temperature, and light touch were all intact.

Investigation

His complete blood count, Blood urea nitrogen (BUN), serum creatinine and liver enzyme were within normal limit. His serum electrolytes showed sodium 140 mEq/L (136-145 mEq/L), potassium 1.8 mEq/L (3.5-5 mEq/L), bicarbonate 20.5 mEq/L (23-30 mEq/L), chloride 104 mEq/L (98-106 mEq/L) and magnesium 2.0 mg/dL (1.6-2.6 mg/dL). His thyroid function tests showed thyrotoxicosis, free triiodothyronine (T3) level 25.7 pg/ml (2-4.4 pg/ml), free thyroxine (T4) level > 7.77 ng/dL (0.93-1.7 ng/dL) and thyroid-stimulating hormone (TSH) < 0.005 mIU/L (0.2-4.2 mIU/L). His Point-of-care testing (POCT) glucose was 128 mg/dL. His chest x-ray was unremarkable. He was diagnosed with TPP as the laboratory test above. His initial electrocardiography (ECG) showed atrial tachycardia with QT prolongation with 2:1 atrioventricular (AV) conduction as shown in **figure 1**. He occasionally had complete AV block which shown in **figure 2**.

Treatment

He was given intravenous potassium 10 milliequivalent (mEq) in 1 hour and oral potassium 30 mEq initially at the resuscitation room immediately after the ECG was done, followed by intravenous potassium 6 mEq/hour and methimazole 15 mg per oral was given at the intermediate care unit.

Outcome

After 10 mEq of intravenous potassium and 30 mEq of oral potassium, ECG converted to sinus tachycardia with QT prolongation as shown in **figure 3**. 10 hours after potassium infusion, serum potassium rose to 6.4 mEq/L. The potassium infusion was immediately stopped, and potassium binder (Kalimate) was given to lower the serum potassium.

He was admitted for 2 days. Methimazole 15 mg twice daily was prescribed for his thyrotoxicosis. His muscle weakness was fully recovery. He was arranged for radioactive iodine ablation in the next 3 weeks.

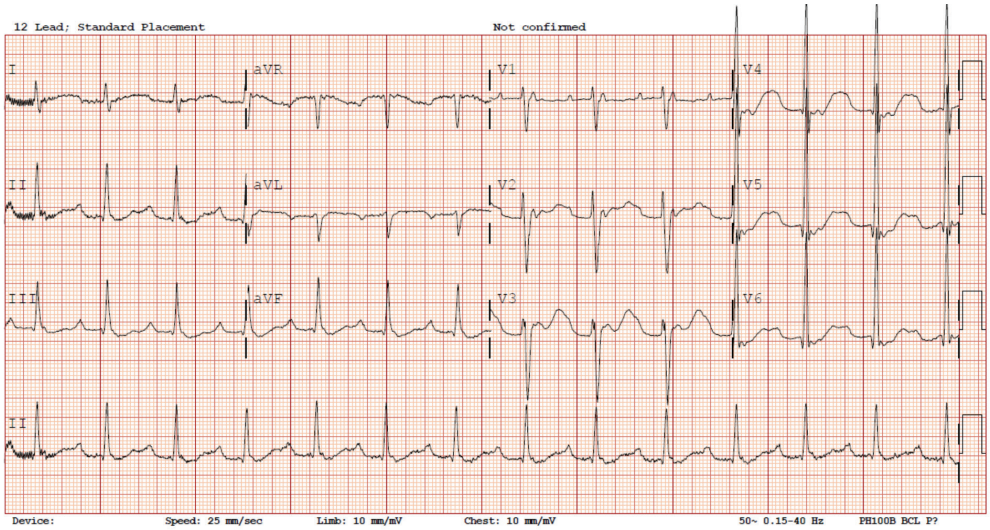

Figure 1 Initial 12 leads ECG Atrial tachycardia with 2:1 AV conduction (Abnormal P wave best seen in V1 with atrial rate about 150 bpm). Flat T wave and large U wave best seen in V2, V3.

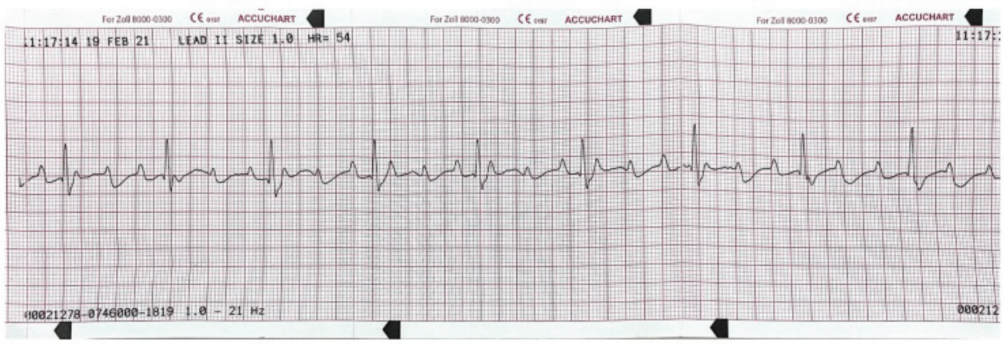

Figure 2 Lead II ECG

Atrial tachycardia with complete AV block with junctional escape rhythm rate 50 bpm

Discussion

We are demonstrating a case of a 24-year-old man presented with acute symmetrical proximal muscle weakness in both upper and lower extremities. He was finally diagnosed with TPP. This patient has many common clinical presentations: usually, young adult males 20-40 years of

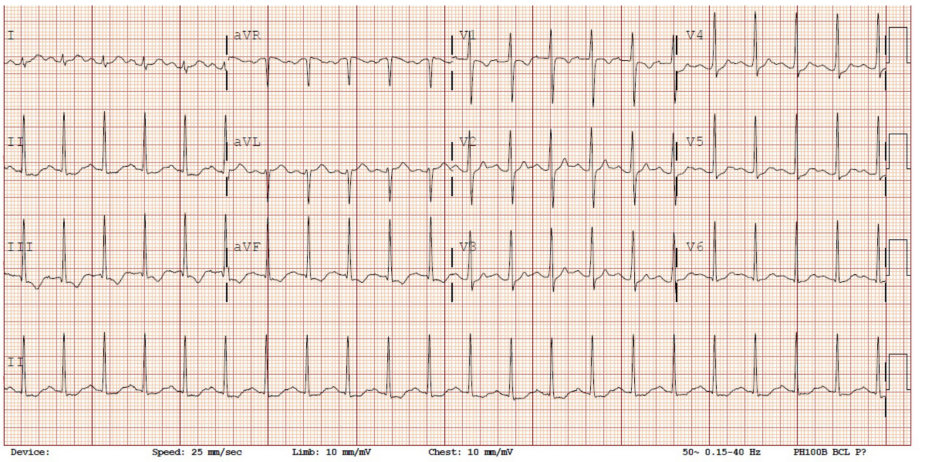

Figure 3 12 leads ECG after potassium infusion Sinus tachycardia rate 130 bpm, no QT prolongation, not seen U wave, Slightly ST depression at II, III, aVF, V4-6 suspected caused by tachycardia (Rate-related ST segment depression)

age, affected more on proximal muscle, normal sensation, no respiratory muscle involvement, the attack occurred a few hours after a heavy meal or early morning, precipitated by strenuous exercise.[2] However, a rare presentation found in our patient was AV block which was showed in **figure 1**. Wang HF[3] had reported a 41-year-old Chinese man with TPP. Interestingly, the patient described by Wang also had ECG of atrial tachycardia with abnormally tall P-wave with a P rate of 150 beats per minutes which could be found in hyperthyroidism.[4]

The pathogenesis of TPP remains unclear. Hypokalemia is the consequence of a rapid and massive shift of potassium from the extracellular into the intracellular compartment, mainly into the muscles. This is believed to be related to increased sodium-potassium-adenosine triphosphatase (Na/K-ATPase) pump activity **(figure 4)**.[2] When thyrotoxicosis was controlled, Na/K-ATPase activity returned to a level similar to that of healthy controls.

The pathogenesis of arrhythmia can be explained by the effect of thyroid hormones. Increasing transcription of Ca2+

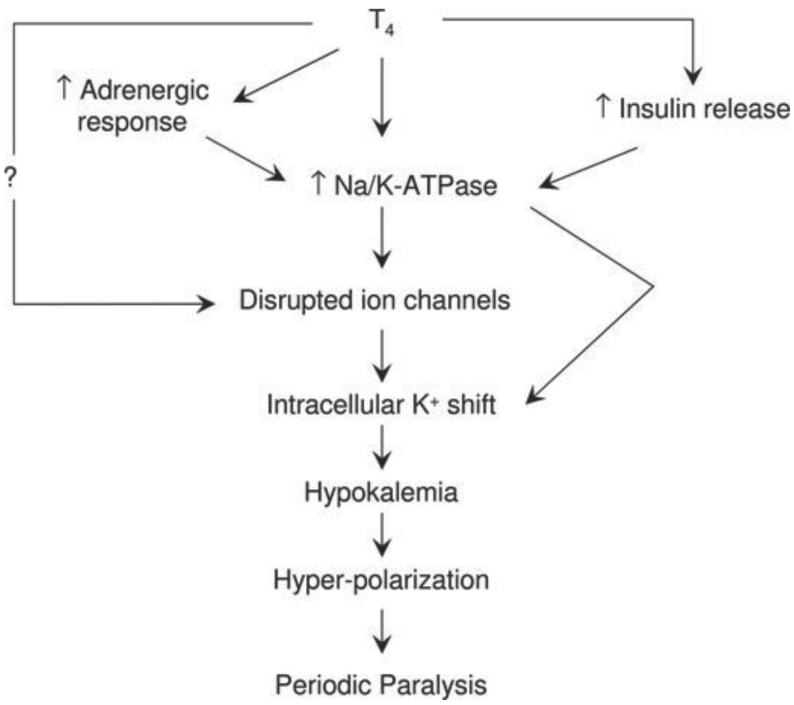

Figure 4 Mechanisms for acute muscle weakness in TPP[2]

ATPase in the myocardial sarcoplasmic reticulum[4,5] results in increased myocardial contractility but increasing of intracellular calcium in atrial cells causing atrial tachycardia by the mechanism of delayed-after depolarization. Excessive thyroid hormones also cause AV nodal tissue inflammation leading to transient AV nodal block.[5,6] AV block in hyperthyroidism is rare and reversible, most complete AV block patients with hyperthyroidism are reversible after achieving a euthyroid state and mostly not required a permanent pacemaker.

Normally, beta-blockers should be given in patients with hyperthyroidism for control symptoms but in this patient, beta-blockers are contraindicated due to impaired AV conduction.

During periodic paralysis and marked hypokalemia, immediate supplementation with potassium chloride (KCl) is warranted to prevent major cardiopulmonary com-

plications. KCl is given intravenously or orally or both. The dose of KCl required varies between 40 and 200 mmol. Excessive potassium replacement may result in rebound hyperkalemia during recovery of the paralysis when potassium is shifted back into the intravascular compartment which was also seen in our patient. It was reported that 40% of patients given iv KCl at a rate of 10 mmol/hour developed rebound hyperkalemia greater than 5.5 mmol/L[7]

Because TPP does not recur in euthyroid, adequate control of hyperthyroidism is the mainstay of therapy. The cause of hyperthyroidism should be identified. Definitive treatment with radioactive iodine or thyroidectomy should be given to patients with hyperthyroidism due to Graves' disease, multinodular goiter, or toxic adenoma.[2] In this patient, the cause of hyperthyroidism is Graves' disease. Therefore, radioactive iodine was arranged.

Learning point/Take home massage

- TPP is common in young adult Asian man 20–40 year of age.
- Complete AV block in hyperthyroidism is rare and reversible.
- During periodic paralysis and marked hypokalemia, immediate supplementation with KCl is warranted to prevent major cardiopulmonary complications.
- Excessive potassium replacement may result in rebound hyperkalemia during recovery of the paralysis when potassium is shifted back into the intravascular compartment.

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