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Thyrotoxic periodic paralysis with ventricular tachycardia

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ABSTRACT

A 47-year-old man presented to our emergency department (ED) with limbs weakness for 2 h. His heart rate was 127 beats per minute and blood pressure was 95/49 mm Hg. He found weakness of limbs after 4-h sleep. Physical examinations revealed that the muscle strength of upper limbs is 3/5, and lower limbs are 2/5.
Electrocardiogram (ECG) revealed wide QRS complex, monomorphic ventricular tachycardia (VT) with ST-segment depression and long QT interval. Serum potassium level was extremely low as 1.0 mEq/L. This led to periodic hypokalemic paralysis. Due to severe hypokalemia with possible atrioventricular block, the patient was admitted to the intensive care unit. During hospitalization, his potassium level returned to 5.1 mEq/L on the first day. He had a low level of thyroid stimulating hormone (TSH) of <0.03 micro-IU/mL (normal range: 0.25–4.00) and a high free thyroxine (T4) level of 2.43 ng/dL (normal range: 0.89–1.79 ng/dL). Therefore, hyperthyroidism was diagnosed, and 5 mg of methimazole was administered twice a day. The patient was discharged on the seventh day after admission. The final diagnosis is thyrotoxic periodic paralysis (TPP), also as known as nocturnal paralysis or night palsy.

Case report

A 47-year-old man presented to our emergency department (ED) due to limb weakness for 2 h. His vital signs upon arrival were a body temperature of 36.1 °C, heart rate of 127 beats per minute, respiratory rate of 14 times per minute, and blood pressure of 95/49 mm Hg. He felt weakness of the limbs after 4 h of sleep. He denied having blurred vision, slurred speech, difficulty in swallowing, or easy choking. Although he claimed to have mild nausea, he denied recently having abdominal fullness, poor appetite, vomiting, or diarrhea. The patient had no concurrent medication. Physical examinations revealed that the muscle strength of upper limbs is 3/5, and lower limbs are 2/5. His breathing sounds were clear. There was no obvious tenderness over the abdomen, percussion revealed dullness, and the bowel sound was normal to hypoactive.

An electrocardiogram (ECG) revealed broad complex tachycardia originating in the ventricles, monomorphic ventricular tachycardia (VT) with depression of ST segment (blue arrow), and a long QT interval, as shown in Fig. 1. The serum potassium level was extremely low at 1.0 mEq/L (normal range: 3.5–5.1 mEq/L), which possibly led to thyrotoxic hypokalemic paralysis. The serum magnesium was 1.7 mEq/L (normal range: 1.8–2.2 mEq/L).

Aggressive potassium supplementation was carried out with potassium chloride (20 mEq) added to 500 mL of normal saline. A central venous catheter was installed to supply the highly concentrated potassium fluid, and the flow rate was set at 100 mL per hour. Oral potassium gluconate (2.54 mEq/tablet) was also given. Due to severe hypokalemia with possible atrioventricular block (Fig. 2), the patient was admitted to the intensive care unit (ICU). During hospitalization, his potassium level returned to 5.1 mEq/L on the first day. He had a low level of thyroid stimulating hormone (TSH) of <0.03 micro-IU/mL (normal range: 0.25–4.00) and a high free thyroxine (T4) level of 2.43 ng/dL (normal range: 0.89–1.79 ng/dL). Therefore, hyperthyroidism was diagnosed, and 5 mg of methimazole was administered twice a day.

The patient was discharged on the seventh day after admission. His serum potassium level returned to the normal range after 12 h of aggressive supplementation. The followed-up ECG revealed sinus tachycardia (Fig. 3). During hospitalization at ordinary ward discharged from ICU, the results of Holter and ultrasound of heart were

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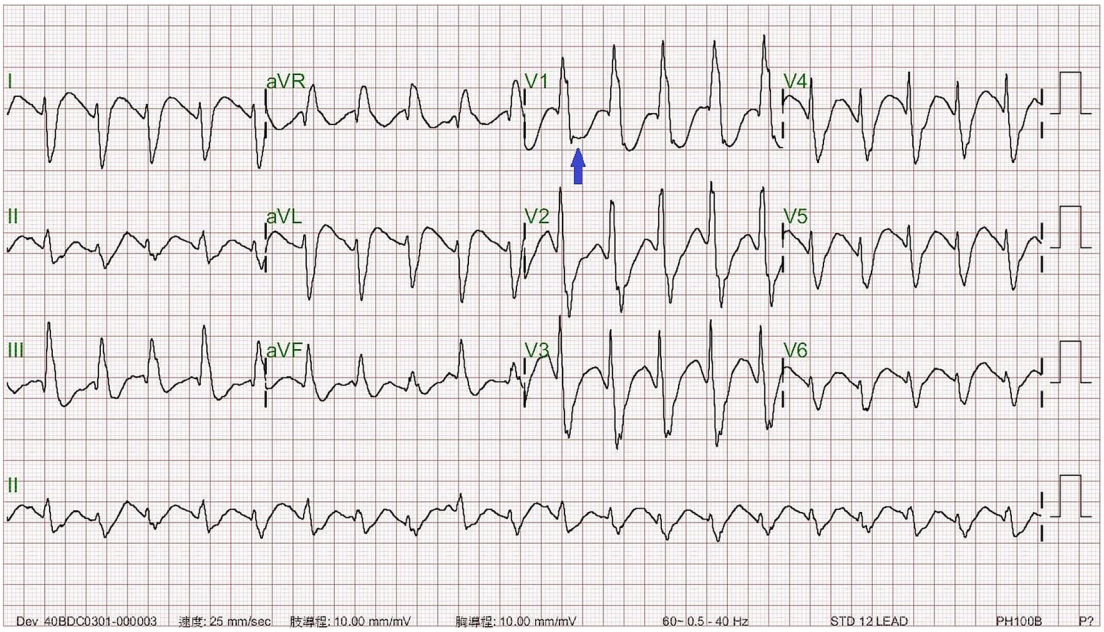

Fig. 1. Electrocardiogram (ECG) revealed wide QRS complex ventricular tachycardia (VT) with depression of ST segment (blue arrow), and long QT interval. U wave is covered in T wave. Patient's serum potassium is 1.0 mEq/L.

unremarkable. The final diagnosis was thyrotoxic periodic paralysis (TPP), which is also as known as nocturnal paralysis or night palsy. The patient follows up in the endocrine outpatient department (OPD) with medication control with methimazole and propranolol.

Discussion

Thyrotoxic periodic paralysis (TPP) is one kind of emergencies in endocrinology. The presentation is variable in muscle power from transient episodes of muscle weakness to complete flaccid paralysis [1]. Despite the fact that hyperthyroidism is more common in females, thyrotoxic periodic paralysis (TPP) is more commonly seen in male than the female with a ratio ranged from 17:1 to 70:1 [2]. The mechanism of TPP includes increased Na+–K+ ATPase activity stimulated by thyroid hormone and/or hyperadrenergic activity and hyperinsulinemia [2]. These made notably low serum hypokalemia in the results of blood tests. Unfortunately, life-threatening arrhythmia maybe happened in severe hypokalemic patients. Aggressive supplement of potassium can cause a dramatic improvement in muscle strength.

Patients usually suffer painless muscle weakness after a heavy workload or exercise, long-term fasting, an alcohol binge, or high carbohydrate intake [3]. Hypokalemia increased membrane excitability in the Purkinje fibers of the cardiac conducting system. Electrocardiogram in TPP may show widened QRS complexes, high QRS voltage, lower amplitude of T-wave, and U waves. Sinus tachycardia is commonly seen in

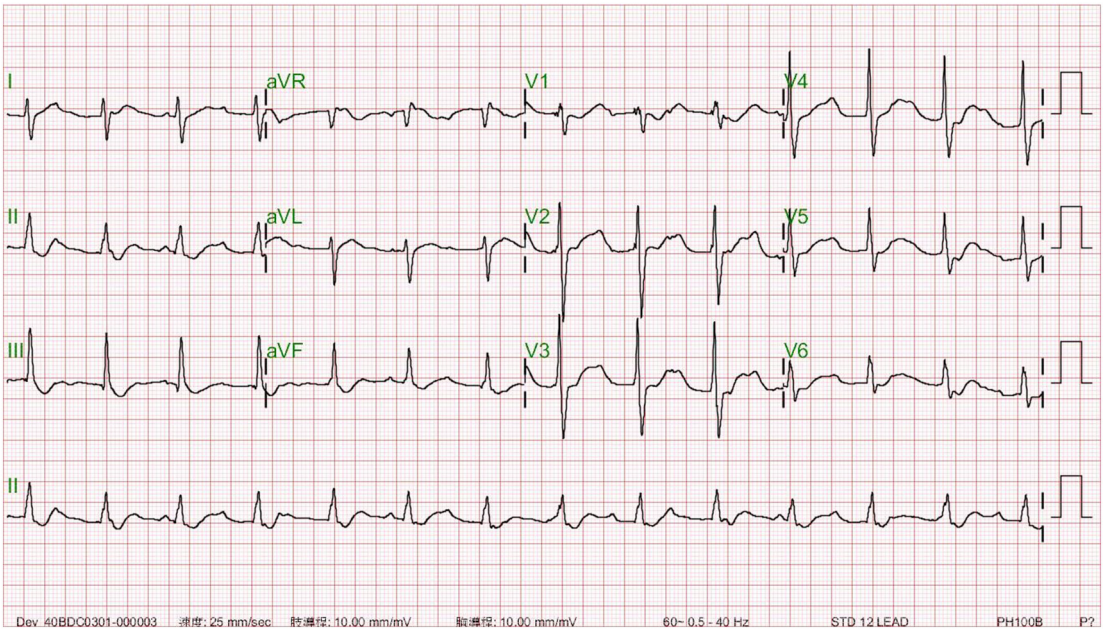

Fig. 2. ECG showed high degree atrioventricular block with QT prolongation.

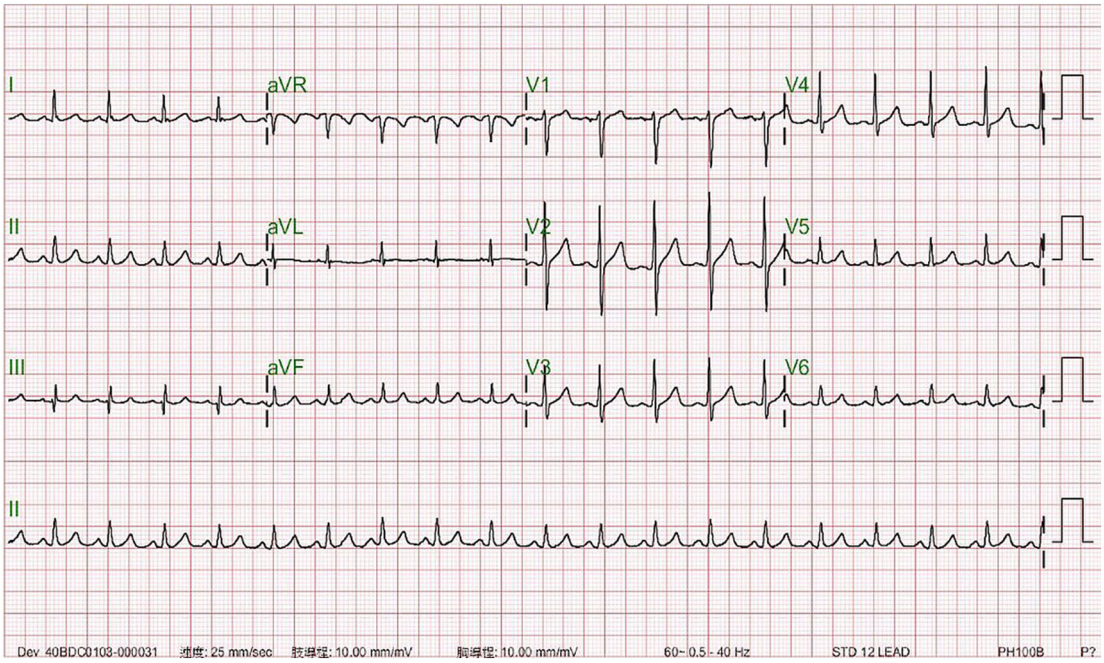

Fig. 3. The followed electrocardiogram showed regular P wave with narrow QRS tachycardia, an ECG of sinus tachycardia. Patient's serum potassium is 5.1 mEq/L.

patients with TPP. Other dangerous electrocardiographic abnormalities include an atrioventricular block (AVB), atrial fibrillation, ventricular fibrillation, and asystole [2,4]. In this case, monomorphic ventricular tachycardia is relatively rarely seen in TPP with severe hypokalemia and mild hypomagnesemia clinically. Although Torsades de pointes is one kind of polymorphic VT, in this case, it is monomorphic VT by the finding of the equal amplitude of QRS and broad complex tachycardia originating in the ventricles.

Severe hypokalemia can induce ventricular arrhythmias and delays ventricular repolarization by inhibiting potassium channel activity [5,6]. Complete recovery can be achieved with acute supplementation to fix any electrolyte imbalance, administration of a beta-blocker, and treatment of the thyrotoxicosis. The physicians in the emergency department should be aware of this endocrinological emergency when confronted with any case of muscle weakness.

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Conflict of interest statement

No.

Role in writing

Tsai IH wrote the manuscript. Su YJ revised and corresponded.

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