Thyrotoxic periodic paralysis complicated by near-fatal ventricular arrhythmias

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ABSTRACT

A 35-year-old Chinese man presented with acute thyrotoxic periodic paralysis complicated by near-fatal cardiac arrhythmias due to persistent hypokalaemia, despite maximum potassium supplementation. He was eventually resuscitated with external cadioversion. In this unusual case of severe refractory hypokalaemia leading to ventricular fibrillation in a patient with underlying thyrotoxicosis, the potential dangers concerning the use of dextrose infusion and beta-adrenergic agent for resuscitation are highlighted.

Keywords: hypokalaemia, thyrotoxicosis, thyrotoxic periodic paralysis, ventricular fibrillation

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INTRODUCTION

Thyrotoxic periodic paralysis is a fairly common accompaniment of hyperthyroidism in Asian populations[1,2], and is accompanied by hypokalaemia that is usually transient. Precipitating factors are a high carbohydrate intake, periods of inactivity, trauma, exposure to cold, infection, menses and emotional stress. Ventricular arrhythmias during the paralytic attacks are rare and confined to sporadic case reports[2,3]. We report a case of thyrotoxic periodic paralysis who had life-threatening cardiac arrhythmias and persistent hypokalaemia.

CASE REPORT

A 35-year-old Chinese male polytechnic lecturer was admitted in January 2002 for acute quadriparesis at 3 am while getting up to void. He had had a buffet dinner the preceding evening. He had thyrotoxicosis since 1998, for which he took carbimazole only for six months. There was no family history of similar illness. Baseline investigations showed normal serum sodium levels, potassium 2.0 mmol/L (normal range 3.5-5 mmol/L), free T4 of 47 pmol/L (normal range 10-20 pmol/l), thyroid stimulating hormone (TSH) <0.005 mIU/L (normal range 0.4-3.98 mIU/L), and TSH receptor antibody 20.6 U/L (normal <1.5 U/L). The electrocardiogram (ECG) showed features of hypokalaemia.

A diagnosis of thyrotoxic periodic paralysis was made. He was treated with 7.45% potassium chloride in concurrent 5% dextrose infusions and propylthiouracil 200 mg 8-hourly. Three hours after admission, he had two episodes of ventricular fibrillation with loss of consciousness (Fig. 1). Fortunately, he was successfully resuscitated with 30 J, followed by 100 J cardioversion for the first episode and 200 J, 360 J and 360 J, respectively, for the second episode, along with two 1 mg bolus doses of 1:10,000 (10 mL) adrenaline during each episode.

His serum potassium dropped to a nadir of 1.8 mmol/L, in spite of intravenous replacement. Serial ECGs remained normal, but cardiac enzymes showed an elevation of creatinine kinase to 902 U/L (normal range 40 – 210 U/L) on the second day. Troponin I was 1.23 μg/L (normal <0.80 μg/L). An endocrine consult was called, following which the 5% dextrose infusion was stopped and propranolol 40 mg bd commenced. Two-dimensional echocardiography showed mild systolic wall motion abnormalities with abnormal left ventricular diastolic relaxation, and with a dilated left ventricle; ejection fraction was 54%. Serum potassium returned to normal and he was asymptomatic at the time of discharge. A repeat echocardiogram performed one month later (when patient was in euthyroid state) showed complete resolution of the subtle abnormalities detected earlier. Subsequently, he underwent I^{131} thyroid ablative therapy as definitive treatment for thyrotoxic Graves' disease. He is currently well and is on levo-thyroxine replacement because of post-ablative hypothyroidism.

DISCUSSION

Our patient showed many typical features of thyrotoxic periodic paralysis, namely: male gender, Mongoloid ancestry, onset of illness in the 3rd to 5th decade, and absence of obvious stigmata of hyperthyroidism on initial examination. Most subjects have spontaneous recovery as the transcellular

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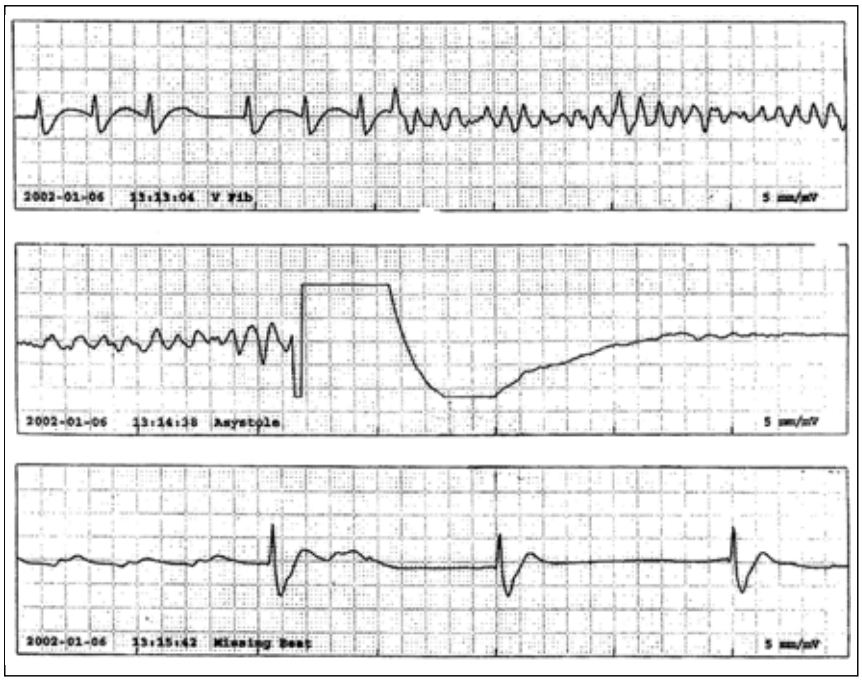

Fig. 1 Electrocardiogram rhythm strip shows onset of ventricular fibrillation. The patient was promptly defibrillated with return to a junctional escape rhythm.

potassium shifts in thyrotoxic periodic paralysis are transient, and usually oral potassium supplements would suffice. Although premature ventricular contractions have been described during the paralytic attack, ventricular arrythmias are extremely rare and confined to sporadic case reports[2,3]. In our patient, we believe the dextrose infusion and adrenaline used for resuscitation aggravated the hypokalaemia and further provoked ventricular arrythmias.

Thyrotoxic patients with periodic paralysis demonstrate an exaggerated insulin response to a carbohydrate load compared to those without periodic paralysis[4]. Just as a high carbohydrate load in patients with thyrotoxic periodic paralysis can provoke a paralytic attack, the liberal use of dextrose infusion would likewise lead to insulin-mediated transcellular potassium shift because of hyperinsulinaemia. In an unrecognised case of thyrotoxic periodic paralysis, worsening of hypokalaemia and paralysis with subsequent death had been reported with the administration of a 10% glucose infusion[5].

While patients with thyrotoxicosis demonstrate an increase in Na^+-K^+ ATPase pump activity, those with thyrotoxic periodic paralysis show an even greater augmentation in the Na^+-K^+ ATPase pump activity[6]. Patients with thyrotoxicosis also manifest increased β-adrenergic stimulation because of an increase in both β-adrenergic receptor number and its sensitivity[7]. Hence, it is conceivable the use of adrenaline in such situations may produce critical hypokalaemia from the marked intracellular potassium shift. In conclusion, this case highlights the uncommon but plausible occurrence of serious cardiac arrhythmias in patients with thyrotoxic periodic paralysis. Caution should be exercised upon the use of dextrose infusion and/or β-adrenergic agent in patients with cardiac arrhythmias associated with hypokalaemia.

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