Thyrotoxic periodic paralysis presenting as a broad complex tachycardia

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Introduction

Thyrotoxic periodic paralysis is a rare but potentially fatal manifestation of hyperthyroidism, characterised by ascending symmetrical muscle weakness and hypokalaemia (McFadzean and Yeung, 1967). This case report describes a patient who developed broad complex tachycardia secondary to thyrotoxic periodic paralysis.

Case report

A 35-year-old man was brought to the heart attack centre as an emergency primary arrhythmia call. His partner called the ambulance when he described a rapidly progressing weakness that initially affected his feet and then spread to his thighs and upper limbs. The ambulance crew performed a resting 12-lead electrocardiogram, which demonstrated a broad complex tachyarrhythmia and prompted the patient's admission to the heart attack centre. On arrival he reported tight central chest pain. His heart rate was 146 beats per minute, systolic blood pressure was 110 mmHg and Glasgow Coma Score was reduced at 10/15.

His medical history included hypertension and Graves' disease, diagnosed 4 years prior with positive thyroid-stimulating hormone receptor antibodies 9.6 U/litre (normal range <0.9 U/litre). However, the patient reported poor adherence to his treatment, carbimazole, for the previous 2 months.

The initial 12-lead electrocardiogram confirmed a broad complex regular tachyarrhythmia (Figure 1). Arterial blood gas showed a potassium level of 1.2 mmol/litre (normal range 3.5–5.3 mmol/litre). Intravenous potassium replacement therapy was commenced at 20 mmol/hr via peripheral infusion. Within moments of commencing the infusion, his heart rate slowed and he reverted to sinus rhythm (Figure 2). Soon after, his chest pain and paralysis resolved.

Formal biochemistry confirmed a potassium level of 1.9 mmol/litre, which increased to 5.0 mmol/llitre following 40 mmol of intravenous potassium supplementation. Thyroid function tests demonstrated thyrotoxicosis with a thyroid stimulating hormone level of <0.01 mU/litre (normal range 0.3–4.2 mU/litre), free triiodothyronine level of 19.7 pmol/litre (normal range 2.5–5.7 pmol/litre) and a free thyroxine level of 33.9 pmol/litre (normal range 9–23 pmol/litre). High-sensitivity troponin-I was also elevated at 1832 ng/litre (normal range 0–15 ng/litre). A repeat high-sensitivity troponin-I the following day was 781 ng/litre. All other blood tests were within normal ranges.

An echocardiogram, computed tomography coronary angiogram and cardiac magnetic resonance imaging were conducted. He had normal biventricular function and no valvular abnormalities. The computed tomography coronary angiogram demonstrated unobstructed coronary arteries, and there was no inflammation seen on the cardiac magnetic resonance image.

Carbimazole treatment of 40 mg once a day was re-initiated, alongside 40 mg propranolol three times a day. He was reviewed by the endocrine team who initiated a short course of prednisolone 40 mg once a day to reduce the peripheral conversion of free thyroxine to free triiodothyronine, and organised an outpatient follow up.

Discussion

The pathogenesis of thyrotoxic periodic paralysis is related to the thyrotoxic state resulting in hyperactivation of the sodium-potassium adenosine triphosphatase pump within the cell membranes, which is responsible for maintaining electrolyte homeostasis (Lin, 2005). Thyrotoxicosis potentiates catecholamine activity and overstimulation of the

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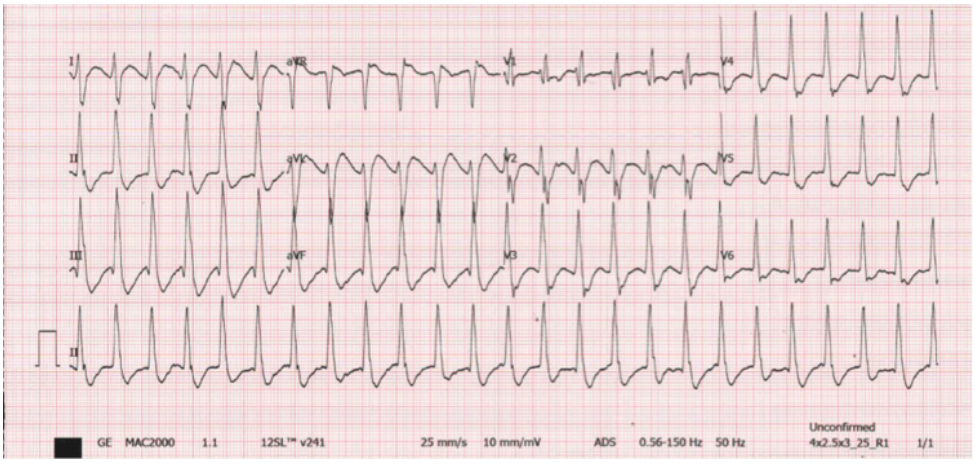

Figure 1. Resting 12-lead electrocardiogram performed on arrival to the heart attack centre.

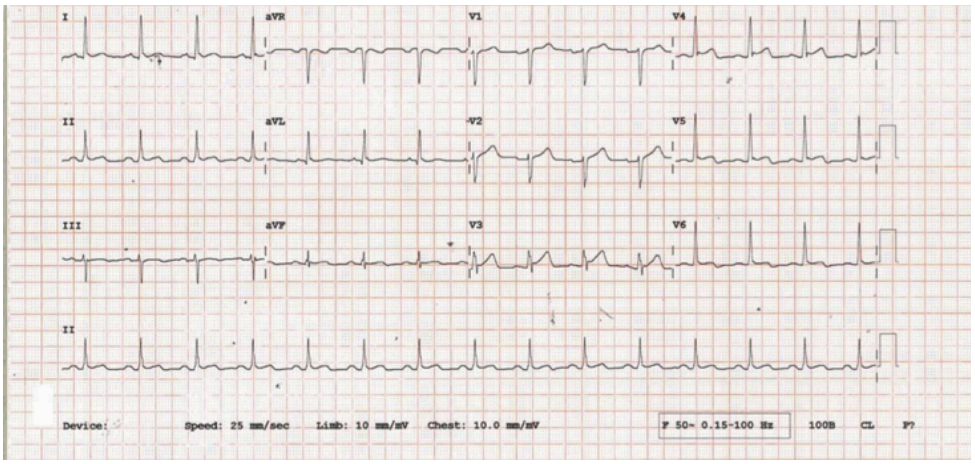

Figure 2. Resting 12-lead electrocardiogram performed when the patient reverted to sinus rhythm.

beta-2-adrenergic receptors on muscle cells. In turn, the activity of the sodium-potassium adenosine triphosphatase pump increases, resulting in the intracellular shift of potassium and a reduced level of serum potassium. The muscle cells are unable to depolarise, causing a transient state of muscle weakness (Chan et al, 1991; Wimmer et al, 2001). The activity of the sodium-potassium adenosine triphosphatase pump is affected by the level of thyroid hormones, adrenergic activity and insulin levels. Therefore, attacks can be precipitated by vigorous exercise, emotional stress, trauma or ingesting a high carbohydrate meal (Mellgren et al, 2002). Thyrotoxic periodic paralysis is predominantly found in those of Asian ethnicity, with an incidence that is ten times greater compared to the non-Asian population. Intriguingly, this man is of Afro-Caribbean ethnicity (Chan et al, 1991).

The hypokalaemia observed in thyrotoxic periodic paralysis is conducive to increased arrhythmogenicity because of a paradoxical increase in the excitability of cardiomyocytes through the hyperpolarisation of the resting membrane potential. Hypokalaemia increases the action potential duration and refractory period, which increases the likelihood of an ectopic beat formation during repolarisation. Thyroid hormones increase the calcium uptake into the sarcoplasmic reticulum, which can result in spontaneous uncontrolled release of calcium waves. This pro-arrhythmogenic state is augmented by increased adrenergic activity, and can result in life-threatening arrhythmias (Helfant, 1986; Stöckigt et al, 2012).

Management of thyrotoxic periodic paralysis involves treatment of the underlying thyrotoxicosis and correction of the hypokalaemia. Clinicians must be vigilant in managing the potential risk of rebound hyperkalaemia caused by the release of intracellular potassium during recovery, hence why, in this case, the levels of serum potassium rapidly normalised

Learning points

- Thyrotoxic periodic paralysis should be considered as a differential diagnosis in patients presenting with a broad complex tachyarrhythmia and paralysis.
- Thyrotoxicosis can also lead to severe hypokalaemia.
- Thyroid function tests should be requested in all patients presenting with arrythmias, even if they do not display the classic signs of hyperthyroidism.
- Treatment of thyrotoxic periodic paralysis should include cautious potassium supplementation, non-selective beta blockade and antithyroid drugs.

following only 40 mmol of potassium supplementation (Lu et al, 2004). A non-selective beta-blocker (such as propranolol) reduces the intracellular potassium shift by blunting the adrenergic stimulation of the sodium-potassium adenosine triphosphatase pump. Beta-blockade inhibits the release of spontaneous calcium waves from the sarcoplasmic reticulum and suppresses ectopic beats, which reduces the risk of ectopy occurring during cardiomyocyte repolarisation. Definitive management involves treating the hyperthyroidism with antithyroid medications, surgical thyroidectomy or radioiodine therapy (Yeung and Tse, 1974; Zhou et al, 2011).

Conclusions

Thyrotoxic periodic paralysis should be considered in any patient presenting with a broad complex tachyarrhythmia and paralysis. Thyroid hormone evaluation is mandatory, even in those not displaying the classical signs of hyperthyroidism. Initial treatment should include cautious potassium supplementation, non-selective beta-blockade and antithyroid drugs.

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