

Hypokalemic paralysis complicated by Takotsubo cardiomyopathy in an euthyroid elderly male with Graves' disease during methimazole dose reduction

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Thyrototoxic hypokalemic periodic paralysis (THPP) is a sporadic complication of thyrotoxicosis, predominantly occurring in young Asian males with active Graves' disease (GD), its onset in euthyroid patients with GD is rare.^[1] Most previous studies suggest that elevated circulating thyroid hormones are an indispensable prerequisite for THPP attacks, yet isolated clinical case reports have demonstrated that paralysis can still be triggered in euthyroid individuals upon exposure to multiple precipitating factors.^[2,3] Herein, we describe an elderly Graves' male on long-term methimazole (MMI) with stable euthyroidism, who suffered acute THPP during MMI tapering complicated by ventricular fibrillation and Takotsubo cardiomyopathy.

An 83-year-old male underwent thyroid function tests (TFTs) in primary care two years prior, which showed suppressed thyroid-stimulating hormone (TSH < 0.005 mIU/L, normal range: 0.35–5.50 mIU/L) and free thyroxine (FT4 of 40.5 pmol/L, normal range: 10.4–24.3 pmol/L). Thyrotropin receptor antibody was subsequently found to be elevated (2.91 IU/L, normal range: < 1.75 IU/L), confirming the diagnosis of GD. Although the patient initially reported no symptoms, he was later found to have heat intolerance and loose stools. His biochemical thyroid function returned to normal after 6 months of treatment with MMI. Subsequently, MMI was gradually tapered from the initial dose of 10 mg/day to the current 2.5 mg/day over 12 months. TFT results one week prior to admission were as follows: TSH of 2.89 mIU/L, FT4 of 16.8 pmol/L, FT3 of 4.68 pmol/L (normal range: 2.7–6.3 pmol/L), and serum potassium of 4.07 mmol/L (normal range: 3.5–5.5 mmol/L). His significant past medical history included hypertension and dyslipidemia, with no diagnosis of other autoimmune diseases.

On the day of admission, after breakfast, the patient had a verbal altercation in the hospital outpatient department. A few minutes later, while conversing in a seated position, he collapsed forward onto the table in front of him and was subsequently transported to the emergency department. On physical examination, his body temperature was 36.5 °C, while pulse, blood pressure, and pulse oxygen saturation were unmeasurable. Emergency electrocardiography (ECG) indicated ventricular fibrillation; following successful cardiopulmonary resuscitation (including electrical defibrillation, mechanical ventilation, and administration of adrenergic agonists), the patient was transferred to the intensive care unit. Initial blood biochemical tests revealed a serum potassium level of 2.88 mmol/L, blood glucose of 12.8 mmol/L, and normal acid-base, calcium, and magnesium levels; transaminases were slightly elevated, with both aspartate aminotransferase and alanine aminotransferase at 64 U/L (normal range: < 40 U/L). Other laboratory examinations, including the routine blood test, inflammatory markers, muscle enzymes, and renal function, were all normal. Emergency computed tomography of the chest and abdomen demonstrated a large amount of gastric contents. Echocardiography showed a left ventricular ejection fraction of 56% (normal range: 50%–65%), reduced wall motion of the left ventricular apex (Figure 1), and no signs of increased right ventricular load. Subsequent TFTs results revealed TSH of 0.256 mIU/L, FT4 of 17.80 pmol/L, FT3 of 3.68 pmol/L, and anti-thyroglobulin antibody of 243 IU/mL (normal range: < 115 IU/mL). Thyroid ultrasonography suggested a slightly enlarged right thyroid lobe (23 mm × 30 mm × 45 mm) with abundant blood flow (Figure 2). Post-resuscitation ECG showed sinus tachycardia without ischemic ST-T changes (Fig-

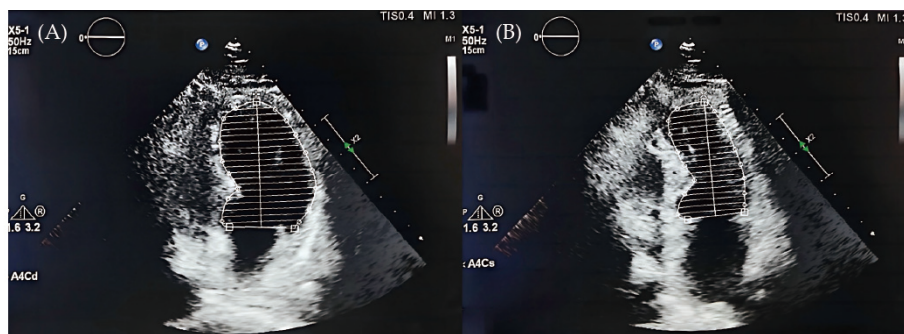


Figure 1 Emergency echocardiography showing reduced wall motion of the left ventricular apex in the diastolic phase (A) and the systolic phase (B).

ure 3). There was no evidence of diarrhea or increased urinary potassium excretion (24-hour urinary potassium of 68 mmol, normal range: 25–125 mmol/24 h). Prior to admission, the patient was taking MMI, rosuvastatin, and anti-osteoporotic drugs.

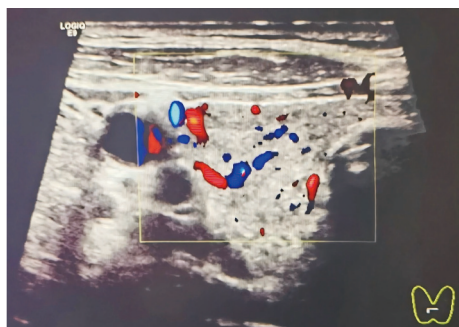


Figure 2 Thyroid ultrasonography showing a slightly enlarged right thyroid lobe (23 mm × 30 mm × 45 mm) with abundant blood flow.

The patient was diagnosed with THPP complicated by ventricular fibrillation. After emergency potassium supplementation and organ support therapy, the patient's neuromuscular system and systemic symptoms improved rapidly, and

the endotracheal tube was removed the next day. On the following day, troponin I was elevated (10.56 µg/L, normal range: < 0.045 µg/L), and N-terminal pro-brain natriuretic peptide was also elevated (7614 pg/mL, normal range: < 450 pg/mL). Forty-eight hours later, ECG showed ST-segment elevation in precordial leads accompanied by prolonged QT interval (QTc: 562 ms) and T-wave inversion (Figure 4). Ten days later, echocardiography suggested resolution of the reduced left ventricular apical wall motion. Takotsubo syndrome was diagnosed after excluding obstructive coronary heart disease (Figure 5). The patient was given 5 mg of MMI daily to control hyperthyroidism. During six months of follow-up, the patient had no recurrence of hypokalemia and muscle paralysis, and the electrocardiogram returned to normal.

This article reports a special case: an elderly patient with GD and biochemically normal thyroid function developed THPP complicated by Takotsubo syndrome. THPP is more common in GD patients with acute thyrotoxicosis, while reports of THPP in GD patients with normal thyroid function are very rare.^[1,2] Classically, this disease occurs in young Asian males and is extremely rare in the elderly population, which may be

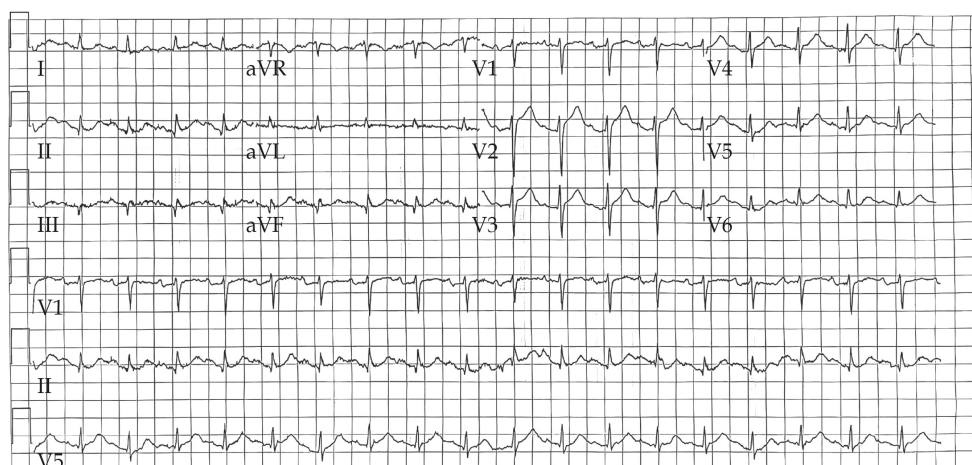


Figure 3 Initial electrocardiography showing sinus tachycardia with a heart rate of 112 beats/min and QTc of 447 ms.

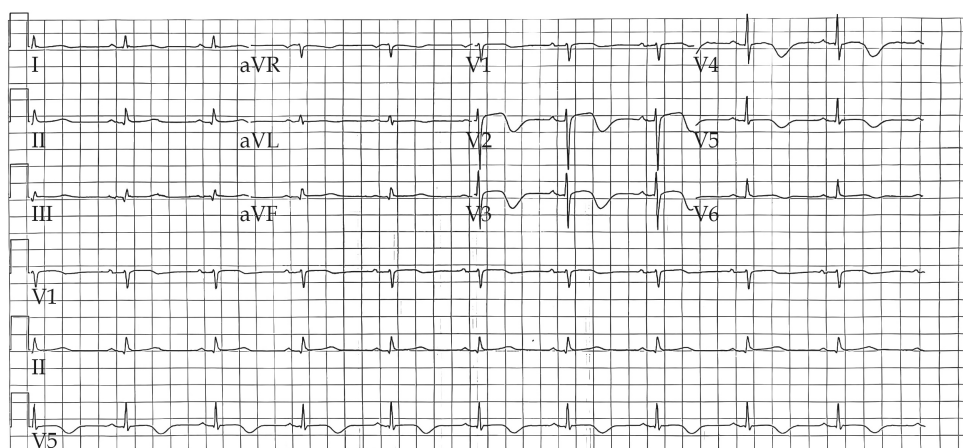


Figure 4 Electrocardiography 48 h later showing ST-segment elevation in precordial leads accompanied by prolonged QT interval (QTc: 562 ms) and T-wave inversion.

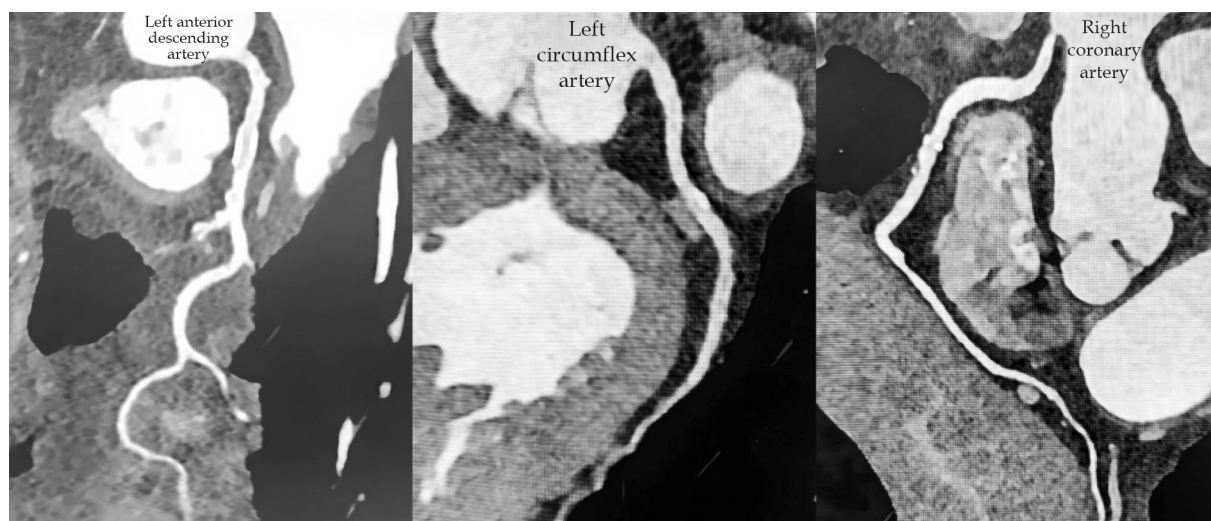


Figure 5 Coronary computed tomography angiography showed no significant vascular stenosis.

due to the higher incidence of GD in young people.^[3] However, in the post-COVID-19 era, the incidence of GD in the elderly population has also increased compared with before,^[4] and emergency and clinicians should be more vigilant about this.

THPP is a rare but potentially fatal complication of thyrotoxicosis, which is characterized by transient episodes of muscle weakness in the setting of hypokalemia and underlying hyperthyroidism. Acute hypokalemia is its main laboratory finding. THPP can be triggered or exacerbated by any factor that promotes hypokalemia. These triggering factors include physical exertion, high-carbohydrate or high-sodium diet, psychological or physiological stress, infections (such as upper respiratory tract or urinary tract infections), cold exposure, alcohol consumption, menstruation, and drugs (such as corticosteroids or β_2 -adrenergic bronchodilators).^[5] In THPP, these triggers enhance Na^+/K^+ -ATPase activity, causing rapid intracellular shift of potassium, leading to a significant decrease in serum potassium level in a short time and thus inducing muscle paralysis.^[6]

Studies have shown that GD is the most common cause of hypokalemic paralysis.^[7] A thyrotoxic state is generally considered a necessary condition for inducing THPP. However, several studies have reported that acute paralysis can occur even after the treatment of thyrotoxicosis or in the state of normal thyroid function.^[1,2] The case of GD reported by us also confirms this phenomenon again. During the tapering of MMI and with biochemically normal thyroid function, he suddenly developed THPP complicated by ventricular fibrillation after multiple triggers such as early morning full meal, cold exposure, and emotional agitation. These events highlight the importance of triggering factors in the pathogenesis of THPP. A possible mechanism for the occurrence of THPP with biochemically normal thyroid function is as follows: during the tapering of MMI, the thyroid function is still in a state of hyperthyroidism, which can lead to a significant decrease in serum potassium level in a short time and thus inducing muscle paralysis.^[6]

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emically normal thyroid function is that the bioavailability of thyroid hormones in specific organs of patients with GD is increased, which is not dependent on the level of serum thyroid hormones.^[8] In addition, the increased expression and sensitivity of tissue β -adrenergic receptors after the tapering of anti-thyroid drugs also play a certain role.^[9]

The special feature of this patient with GD is the concurrent occurrence of THPP and Takotsubo cardiomyopathy. Previously, thyrotoxicosis was rarely described as a precipitating factor for Takotsubo cardiomyopathy. In recent years, more and more evidence has shown that Takotsubo cardiomyopathy may occur in patients with hyperthyroidism, which is not correlated with thyrotoxic symptoms.^[10-12] Thyroid hormones can enhance the agonistic effect of catecholamines by increasing the density of β -adrenergic receptors in the heart. In addition, thyroid hormones can also increase the level of cyclic adenosine monophosphate after β -adrenergic receptor activation by enhancing the activity of adenylate cyclase. Even if the level of catecholamines does not increase in hyperthyroidism, their effects can be amplified, and this amplification can lead to a state of excessive response to minimal stress.^[11] Therefore, Takotsubo cardiomyopathy induced by thyrotoxicosis has a significant risk of complications, with 12% of patients developing cardiogenic shock.^[10] At the same time, the use of adrenergic agonists in this patient during cardiopulmonary resuscitation may also be the cause of Takotsubo cardiomyopathy.

THPP complicated by Takotsubo cardiomyopathy can occur in elderly GD patients with biochemically normal thyroid function during MMI dose reduction, reminding clinicians to maintain vigilance and optimize hyperthyroidism management.

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