CASE REPORT

Thyroid storm in a patient with Wolff-Parkinson-White syndrome

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SUMMARY

A 44-year-old woman with no medical history presented to the emergency department with a 2 h history of sudden onset chest pressure, palpitations, diaphoresis and shortness of breath. She reported a 90-pound unintentional weight loss, increased appetite, irritability, night sweats and palpitations for 2 months. Physical examination revealed a heart rate (HR) of 269 bpm and a blood pressure of 116/94 mm Hg. Her ECG revealed a wide-complex tachycardia with right bundle branch morphology and an HR of 265 bpm. Intravenous adenosine was administered with resolution of the arrhythmia and symptoms. Her subsequent ECG revealed sinus tachycardia with δ waves, which was consistent with Wolff-Parkinson-White (WPW) syndrome. Laboratory findings confirmed thyroid storm and treatment began with intravenous hydrocortisone, methimazole, metoprolol, amiodarone and iodine drops. Graves' disease was confirmed based on the presence of serum thyroid-stimulating hormone receptor antibody. The patient underwent successful WPW accessory tract ablation 6 weeks after initial presentation.

BACKGROUND

This is the first documented case of thyroid storm-induced wide-complex tachycardia in a patient with an accessory pathway (AP). Wolff-Parkinson-White (WPW) syndrome was first recognised in the 1930s, where it had been associated with sudden cardiac death (SCD).¹ The majority of individuals with WPW syndrome are asymptomatic, but those who have symptoms can experience palpitations, recurrent supraventricular tachycardia or SCD. Individuals with hyperthyroidism are at increased risk of cardiac arrhythmias, especially atrial fibrillation (AF).² The occurrence of AF in a patient with an AP can be detrimental, leading to electrical impulses bypassing the protective atrioventricular node and instead travelling down the AP and causing ventricular stimulation, which induces ventricular fibrillation (VF).

CASE PRESENTATION

A 44-year-old white woman with no medical history presented to the emergency department, with a 2 h history of sudden onset chest pressure, palpitations, diaphoresis and shortness of breath. She reported a 2-month history of a 90-pound unintentional weight loss, increased appetite, irritability, intermittent night sweats and palpitations. Physical examination revealed a heart rate (HR) of 269 bpm and a blood pressure of 116/94 mm Hg. Cardiopulmonary examination was unremarkable.

Her ECG revealed a wide-complex tachycardia with right bundle branch morphology and an HR of 265 bpm (figure 1A). Intravenous adenosine was administered at 6 mg followed by 12 mg, with resolution of the wide-complex tachycardia. Given the response to adenosine, the differential diagnosis was anti-dromic tachycardia, atrioventricular (AV) node re-entry tachycardia (AVNRT) with bystander AP and adenosine-sensitive atrial tachycardia with a bystander AP.

The patient's subsequent ECG revealed sinus tachycardia with an HR of 120 bpm and δ waves—consistent with WPW syndrome (figure 1B). Physical examination revealed pressured speech, a non-tender diffusely enlarged goitre with a bruit (figure 2A), sweaty palms and a systolic flow murmur. Her laboratory tests were notable for normal troponin and serum levels of thyroid-stimulating hormone (TSH) of <0.01 mU/L (0.27–4.20 mU/L), total T4 of 24.8 μ g/dL (4.5–12.0 μ g/dL) and free T3 of 29.6 pg/mL (2.0–4.4 pg/mL), respectively. She met diagnostic criteria for thyroid storm.³ Thyroid ultrasound revealed a diffusely enlarged gland with increased vascularity without discrete nodules. She was admitted to the intensive care unit, for continuous haemodynamic monitoring. She was treated with intravenous hydrocortisone 100 mg every 8 h, methimazole 20 mg every 6 h, metoprolol 25 mg every 6 h, amiodarone 400 mg every 8 h and potassium iodine 5 drops in water every 8 h. Her thyroid hormone levels slowly reduced over time and her thyroid gland decreased in size. A diagnosis of Graves' disease was confirmed based on her elevated serum TSH-receptor antibody concentration of 24.22 IU/L (normal ≤ 1.75). A transthoracic echocardiogram showed mild pulmonary hypertension with normal ventricular function. Electrophysiology (EP) study and ablation was considered, but deferred secondary to uncontrolled hyperthyroidism. The patient was discharged home on a prednisone taper, metoprolol, amiodarone and methimazole. Amiodarone was chosen because of a national shortage of procainamide. It is an effective drug to inhibit the AV node and to slow the AP. Because of the prompt effectiveness of methimazole and prednisone, the side effect of amiodarone on the thyroid gland is negligible.

The patient returned to hospital 6 weeks after initial presentation for a staged WPW accessory tract ablation. Given the ventricular rate of 270 bpm, the effective refractory period (ERP) was calculated to be 222 ms (60 000/270), therefore making it a high-risk AP. Current guidelines recommend EP ablation if the patient is symptomatic or has an ERP of <250 ms. Indeed, the AP was

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Figure 1 (A) Regular wide-complex tachycardia with rate of 265 bpm. (B) Sinus tachycardia with δ wave pattern.

confirmed to be high risk with an ERP of <200 ms. During the EP study, the patient did not have any evidence of dual AV nodal physiology, therefore excluding AVNRT. The tachycardia was reproduced using pacing manoeuvres, confirming antidromic tachycardia along the AP. The AP was located at the posterolateral aspect of the mitral valve and successful radiofrequency ablation (RFA) was performed to that area. An ECG performed postoperatively no longer showed the δ wave pattern of WPW syndrome (figure 3). The patient was clinically in a euthyroid state and her thyroid gland had shrunk in size (figure 2B). Her serum TSH and free T4 were found to be <0.01 mU/L and 1.39 μ g/dL, respectively. She was continued on

methimazole 10 mg twice daily and aspirin 325 mg daily; both amiodarone and metoprolol were discontinued on discharge. The patient underwent successful elective thyroidectomy for definitive management of her Graves' disease. She is clinically euthyroid and is maintained on 100 μ g of levothyroxine once daily.

DISCUSSION

Thyroid storm is an endocrine emergency that has a mortality of 10–30%; therefore, prompt recognition is needed in order to initiate treatment.³ There are no specific laboratory criteria for diagnosing thyroid storm, however, Burch *et al* developed

Figure 2 (A) The patient's large goitre and (B) the goitre improving at 6 weeks.

Figure 3 Normal sinus rhythm after accessory pathway ablation without δ wave pattern.

scoring criteria based on clinical criteria (fever, elevated HR, central nervous system dysfunction, heart failure symptoms, AF, gastrointestinal dysfunction and a precipitant history). The treatment of thyroid storm includes therapy against new thyroid hormone production (methimazole or propylthiouracil), therapy against thyroid hormone release (Lugol's solution or potassium iodide drops) and blocking the peripheral effects of thyroid hormone (propranolol and hydrocortisone).⁴

The mortality in patients with pre-excited tachycardia due to thyroid storm is unknown. Patients with symptomatic WPW syndrome often need RFA for prevention of fatal arrhythmias. EP study may be useful in asymptomatic individuals to assess for 'high-risk' AP with characteristics that would make an individual at elevated risk for SCD.^{5,6} During EP study, patients who have short AP antegrade refractory period (<250 ms) are at high risk of VF and would benefit from RFA.⁵ The success rate of this procedure is 98.5% with modern complication rates of <2%.⁵

In patients with thyroid storm, EP study should be deferred until they are biochemically euthyroid. These patients must be started on antiarrhythmic agents, such as procainamide in the short term or amiodarone until ablation, in order to maintain sinus rhythm. In this case, amiodarone was chosen—instead of

Learning points

- ▶ Thyroid storm has a mortality of 10–30%, and prompt recognition is needed in order to initiate treatment.
- ▶ Patients with symptomatic Wolff-Parkinson-White (WPW) syndrome can develop fatal arrhythmias including anti-dromic tachycardia, atrioventricular node re-entry tachycardia with bystander accessory pathway (AP) and adenosine sensitive atrial tachycardia with a bystander AP.
- ▶ Patients with thyroid storm require intensive care unit monitoring, and treatment includes intravenous steroids, β -blockers, iodine drops and methimazole.
- ▶ Patients with symptomatic WPW syndrome who present with thyroid storm require loading with amiodarone to maintain sinus rhythm and to prevent the development of fatal atrial arrhythmias.
- ▶ The ultimate treatment of a patient in thyroid storm with symptomatic WPW syndrome is radiofrequency ablation of the AP, but this must only be carried out while the patient is in a euthyroid state.

β -blockers—and antithyroid medications were used to treat the symptoms and hyperthyroidism, respectively. Once ablation had been performed, the amiodarone and metoprolol were both safely stopped.

Contributors SYN wrote the case report. SGR and JIL edited the case report.

Competing interests None declared.

Patient consent Obtained.

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