Coronary artery spasm due to thyrotoxicosis

Krystina Common, Krys Milburn, Tom Cawood, Ian Crozier

Abstract

A 50-year-old lady presented with chest pain, tremor, weight loss and intermittent ST elevation in her ECG associated with elevated markers of myocardial injury. She was diagnosed with Graves’ disease and coronary angiogram showed normal coronary arteries. It was felt coronary artery spasm secondary to thyrotoxicosis was the cause for her ECG changes and pain. She was successfully treated with diltiazem, metoprolol and carbimazole. This case highlights the importance of considering hyperthyroidism in the diagnosis of chest pain with normal coronary arteries.

Cardiac signs and symptoms are commonly associated with the presentation of hyperthyroidism, especially tachycardia. Other cardiac presentations include heart failure caused by hyperthyroid cardiomyopathy, systolic hypertension and anginal chest pain due to increased cardiac contractility and cardiac output.1

The earliest case report on MEDLINE of coronary artery spasm associated with thyrotoxicosis was in 1979, however this remains a rare presentation with no published series describing the underlying pathophysiology.4

Case reports suggest females have a higher incidence of coronary artery spasm associated with hyperthyroidism, however this is likely due to the increased incidence of both coronary spasm and thyroid disorders in the female population.1,6

Case report

A previously well 50-year-old lady was admitted with a 3-day history of palpitation and chest tightness. She had also noticed a 5 kg weight loss and fine tremor in her hands over the past 3 months.

On examination she had a sinus tachycardia at 104 beats per minute, a blood pressure of 129/62 mmHg and an enlarged thyroid gland which was easily palpable. Investigations demonstrated a thyroid stimulating hormone (TSH) of <0.01 mIU/L (0.04–4.0), a free T4 of 50 pmol/L (10–24), a free T3 of 42.6 pmol/L (2.5–6.0 pmol/L) and a troponin I (TNI) of 0.17 ug/L (0.01–0.03). A 99mTc-Pertechnetate isotope thyroid scan demonstrated a diffusely enlarged thyroid gland with intense uniform isotope uptake throughout both lobes typical of Graves’ disease.

Telemetry monitoring showed intermittent periods of ST elevation of 2–3 mm in leads II, III and AVF that were associated with episodes of chest pain (Figure 1). A transthoracic echo demonstrated normal left ventricular size and systolic function with an ejection fraction of 69%.
After careful consideration it was decided the patient should undergo a coronary
angiogram despite the risks of contrast containing iodine in thyrotoxicosis. The
concern was in relation to the risks of precipitating a thyroid storm secondary to the
iodine containing contrast used in coronary angiography. The coronary angiogram
was performed using a total of 20ml of iodixanol (visipaque 320) which delivered
5.4g of iodine and confirmed normal coronary arteries (Figure 2). This supported the
diagnosis of coronary artery spasm causing the ECG changes and pain.

She was commenced on diltiazem for the coronary artery spasm, carbimazole for
Graves’ disease and metoprolol for the tachycardia from the hyperthyroidism.
Thyroid function tests and clinical symptoms were monitored closely in the following
weeks in case of delayed thyrotoxicosis related to the iodine load, these steadily
returned towards normal over 6 weeks (Table 1).

Table 1. Table showing serial thyroid function tests

<table>
<thead>
<tr>
<th>Date</th>
<th>7/2/2012</th>
<th>9/2/2012</th>
<th>14/2/2012</th>
<th>20/2/2012</th>
<th>29/2/2012</th>
<th>14/03/2012</th>
</tr>
</thead>
<tbody>
<tr>
<td>Free T4</td>
<td>50</td>
<td>44</td>
<td>33</td>
<td>30</td>
<td>25</td>
<td>21</td>
</tr>
<tr>
<td>(10–14 pmol/L)</td>
<td></td>
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<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Free T3</td>
<td>14</td>
<td>10.7</td>
<td>9.2</td>
<td>6.4</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(2.5–6.0 pmol/L)</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>TSH</td>
<td>&lt;0.01</td>
<td>&lt;0.01</td>
<td>&lt;0.01</td>
<td>0.01</td>
<td>&lt;0.01</td>
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</tr>
<tr>
<td>(0.4–4.0 mIU/L)</td>
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</tbody>
</table>
Discussion

This case illustrates an atypical presentation for hyperthyroidism. Whilst cardiovascular symptoms are common in hyperthyroidism, coronary artery spasm has been infrequently reported.\(^8\)

Iodine based compounds can induce thyrotoxicosis in patients with underlying thyroid autonomy.\(^7\) Iodixanol (visipaque 320) contains 270 mg/ml of iodine and product literature indicated that thyrotoxicosis was an absolute contraindication to usage.

The literature in relation to the incidence or severity of thyrotoxicosis with iodixanol is scarce, but recent reports suggest an increased odds ratio of 2.5 for incident overt hyperthyroidism with iodinated contrast media but absolute risk being relatively low, with the number needed to harm being 23.\(^9\) These numbers are based on euthyroid patients, therefore the risk of causing severe hyperthyroidism in someone who is already significantly thyrotoxic remains unknown.

Due to the concern of ongoing chest pain, dynamic ECG changes and elevated troponin raising the possibility of obstructive unstable coronary disease, we felt the possible risk of exacerbation thyrotoxicosis with the coronary angiogram using the minimum possible iodixanol was justified. We were mindful of the need for careful thyroid status monitoring and follow-up.

This case highlights that coronary spasm can occur in thyrotoxicosis, and that thyrotoxicosis should be considered in patients with cardiac chest pain and normal coronary arteries.

Competing interests: Nil

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References: