Case report

Takotsubo cardiomyopathy in association with endogenous and exogenous thyrotoxicosis

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Case report

Case 1

A 79-year-old woman from Sri Lanka, with a history of hyperthyroidism, presented with chest pain and breathlessness following an episode of extreme anxiety after missing her connecting flight. She had been unable to take carbimazole during the preceding 3 months due to local shortages. Examination revealed signs of acute heart failure, with an electrocardiogram (ECG) showing anterior T-wave inversion, a 12-h cardiac Troponin I of 4.1 ng/ml and thyroid function tests demonstrating thyrotoxicosis (free thyroxine [fT4] 40.4 pmol/l, thyroid-stimulating hormone [TSH] 0.04 mU/l). Coronary angiography showed only minor disease in the left anterior descending and circumflex arteries. Ventriculography revealed classical apical ballooning and left ventricular dysfunction. Cardiovascular magnetic resonance (CMR) imaging on Day 3 confirmed this finding on cine imaging and without evidence of delayed gadolinium hyperenhancement (Figure 1). Her symptoms resolved within 4 days, with complete resolution of left ventricular function on echocardiography 5 days later.

Case 2

A 55-year-old woman presented with shortness of breath and central chest pain. She had been self-medicating with porcine thyroxine obtained over the Internet for several years. On examination, she was in cardiogenic shock with pulmonary oedema. Investigations include an ECG showing anterolateral T-wave inversion, a cardiac Troponin I of 6.1 ng/ml, TSH <0.01 mU/l and fT4 14.7 pmol/l. Coronary angiography demonstrated mild atheroma. Ventriculography showed akinesis of the distal two-thirds of her left ventricle with hyperdynamic contraction of the basal segment (Figure 2A and B). Following intra-aortic balloon pump insertion, diuretics and subsequent β-blockade she made a rapid recovery. An CMR 4 months later showed complete recovery of her ventricular function (Figure 2C and D).

Discussion

Takotsubo cardiomyopathy (TCM) is an acute impairment of cardiac function in the context of chest pain, electrocardiographic changes such as ST-segment elevation and/or T-wave inversion, and a modest Troponin rise, in the absence of obstructive coronary artery disease, myocarditis or phaeochromocytoma.1 It is commonly preceded by acute mental or physical stress. Its hallmark feature is transient hypokinesis, akinesis or dyskinesis of the left ventricular apical and mid segments in a distribution beyond that of a single coronary artery.
artery. A number of theories exist regarding the pathophysiology of Takotsubo cardiomyopathy (TCM), including coronary vasospasm, dysfunction of the microvasculature, shifts in cardiac metabolism from fatty acids towards carbohydrates and left ventricular outflow tract obstruction.\(^2,3\) Currently, the most popular theory centres on potential toxic effects of endogenous catecholamines on the myocardium, resulting in myocardial stunning.\(^4\) This is supported in rat models, where takostubo has been prevented by adrenergic blockade, sampling of cardiac catecholamine levels in TCM\(^5\) and reported cases in association with phaeochromocytoma and use of exogenous sympathomimetics.

The thyroid and adrenergic axes are closely interrelated, and pathologically high levels of thyroid hormone cause exaggerated chronotropic and contractile responses to catecholamines. This is mediated through a change in balance between sympathetic and vagal innervation\(^6,7\) and increased cAMP responses from \(\beta\)-adrenergic receptors in cardiac myocytes, alongside possible enhancement of its downstream signalling pathway.\(^8\)

Our cases link both endogenous hyperthyroidism (associated with cessation of carbimazole) and exogenous hyperthyroidism (self-medication with exogenous thyroxine) with TCM.

Alongside other published cases of TCM and hyperthyroidism,\(^9,10,11\) these two cases add further weight to the possibility that hyperthyroidism predisposes to TCM by acting synergistically with adrenergic hormones in states of catecholamine excess. This may be mediated via increased myocardial ischaemia (due to increased chronotropy, contractility and hence oxygen demand) or through enhanced sympathetic drive. Further research is needed to investigate such an association.

Conflict of interest: None declared.

References


