A case of severe Thyrotoxic Cardiomyopathy

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History and examination

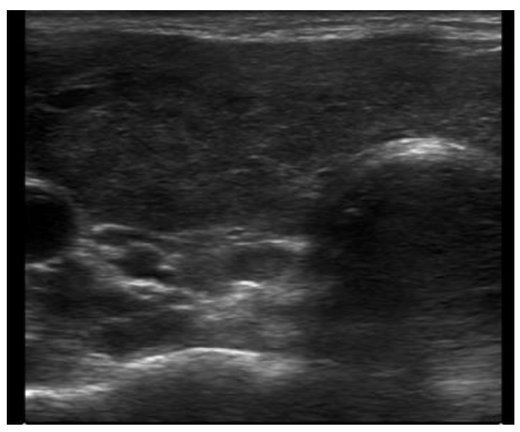
A female, age 47, usually fit and well, presented to the GP with a 4 week history of intermittent palpitations, weight loss, and feeling generally unwell. Over the next few days she developed shortness of breath at rest, heat intolerance, and a sensation of mass in the throat.

On admission she was anxious and had a raised JVP, generalised oedema, tremor, hypertension (BP 194/88mmHg), sinus tachycardia (HR 116 bpm), and a soft, smooth goitre with bruits.

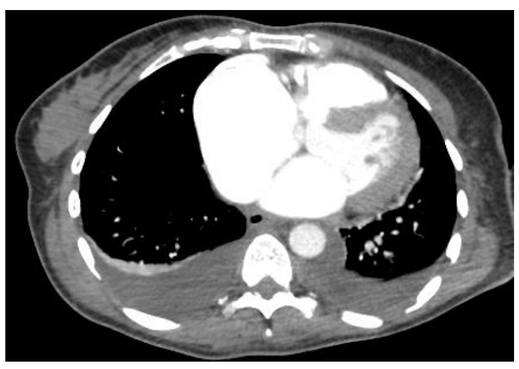
Initial investigations

TSH	< 0.01 mU/l
Free T4	84.1 pmol/l
Free T3	Not done
Alk phos	196 IU/L
TPO antibodies	405

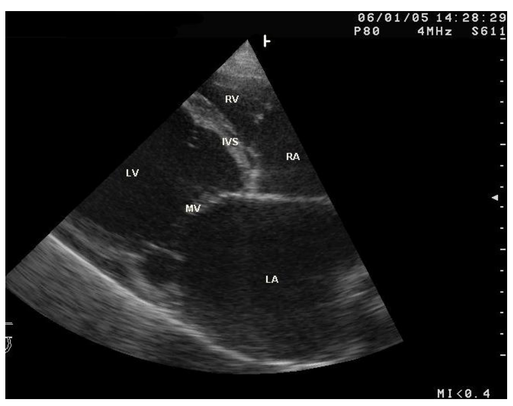
US neck:
The thyroid gland is diffusely enlarged, hypervascular, and has a lobulated contour.
No discrete nodules are detected.



CT chest:
There is cardiomegaly, bibasal atelectasis, bilateral pleural effusions and mild enlargement of the thyroid gland.



Echocardiogram:
Biventricular dilatation with severely impaired LV function, impaired RV function, with pulmonary Hypertension.
Severe TR, moderate MR, mild AR and trace PR.
Features consistent with dilated cardiomyopathy



Diagnosis and Management

A diagnosis of Grave's thyrotoxicosis leading to thyrotoxic cardiomyopathy(TCM) was made.

She was started on Carbimazole (60mg/day), I.V. Furosemide (80mg/day), VTE prophylaxis and Beta-blockers.

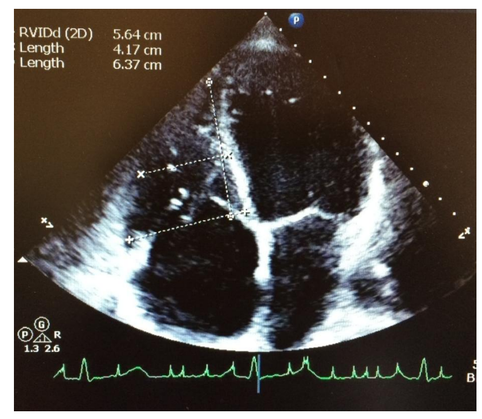
Further Progress

She was reviewed by cardiology to rule out other causes of LVF.

She responded to treatment with rapid improvement of symptoms and repeat TFT 7 days later showed normalisation of FT4.

	Initial	2 weeks	4 weeks	Current
TSH	<0.01mU/l	< 0.01mU/l	< 0.01mU/l	1.61mU/L
Free T4	84.1pmol/l	23.8pmol/l	17pmol/l	12.9pmol/l
Free T3	Not done	4.2pmol/l		
Alk phos	196	146	90	
Carbimazole dose	60mg	20mg	20mg	10mg

Follow up Echo at 3 months:
Both ventricles normal size, good LV function, moderate MR, trivial TR. No evidence of pulmonary hypertension



She is currently clinically euthyroid, with no evidence of LVF. She has been referred for Radio-iodine therapy.

Discussion

Thyrotoxicosis effects both the central and peripheral circulation . It causes increased circulatory volume, reduced pre-load, and reduced systemic vascular resistance but increased pulmonary resistance leading to pulmonary hypertension.

It also has direct cardiac effects including arrhythmias, alteration of myocardial contractility, left ventricular hypertrophy and cardio-myopathy.

Cardiac failure may be rate related, secondary to volume overload or left ventricular impairment. It may also be mediated through the direct toxic effect on duration of action potential and repolarisation in the cardiac myocyte. Prevalence varies from 12%-68%. Although usually associated with underlying heart disease, it can occur in young people even in the absence of heart disease.

TCM is a potentially life threatening complication of thyrotoxicosis [1]. Dilated cardiomyopathy is reported in 1%[2] .The mechanisms are poorly understood. It can be irreversible in up to 1/3 of patients. Risk factors include older age, male sex, pre-existing cardiovascular disease and duration of thyrotoxicosis.

Initial management with beta-blockers for rate control, diuretics and anti-thyroid medication should be followed by definitive management of thyrotoxicosis.

TCM is a serious but potentially reversible cause of cardiomyopathy and hyperthyroidism should be excluded in all patients. [3]

References

1. Cappola A. et al. (2006). Thyroid Status, Cardiovascular Risk, and Mortality in Older Adults., *JAMA. 2006. Vol. 295. □ .1033-1041*
2. Roffi M. et al. (2003). *Cleveland Clinic Journal Of Medicine. 2003. Vol. 70.P. 57-63*
3. Lutton S. et al. (2001) *Cardiomyopathy and myocardial failure*