

## Original Research Article

# Reversible thyrotoxic cardiomyopathy: a case report

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### ABSTRACT

Hyperthyroidism significantly affects the hemodynamics of the cardiovascular system. It is associated with development of atrial fibrillation, high output cardiac failure, pulmonary hypertension and dilated cardiomyopathy (DCM). In this paper we report a case of thyrotoxicosis induced cardiomyopathy. She was a 54 year old woman who presented with cardiomegaly (dilatation of all four chambers of the heart) secondary to Graves' disease. She was treated with anti-thyroid drugs,  $\beta$  Blockers and diuretic and her euthyroid status was restored in 6 weeks. A repeat echocardiogram done at this point of time showed normal cardiac function with normalization of ejection fraction. So this was a case of reversible thyrotoxic cardiomyopathy.

**Keywords:** Thyrotoxicosis, Hyperthyroidism, Dilated cardiomyopathy

### INTRODUCTION

Thyrotoxic cardiomyopathy is defined as myocardial damage caused by excess circulating levels of thyroid hormones which leads to dilatation of all the chambers of the heart and subsequent heart failure. Hyperthyroidism is characterized by an elevated heart rate and a reduction in peripheral vascular resistance which in turn leads to an increased cardiac output.<sup>1</sup> This untreated high cardiac output state leads on to ventricular dilatation, persistent tachycardia and heart failure which is usually reversible.<sup>2</sup> We present one such case of hyperthyroidism (due to diffuse thyromegaly) with thyrotoxic cardiomyopathy.

### METHODS

We report this case of a 54 year old postmenopausal woman who presented with history of weight loss, palpitations and dyspnoea. She also complained of heat intolerance and tremulousness. There was no past history of hypertension/diabetes mellitus or coronary artery disease and she was non-smoker and non-alcoholic. On physical examination, the patient had a staring look due

to exophthalmos. She was a febrile with a pulse rate of 124/min. which was regular with a good volume. Her blood pressure was 138/86 mm Hg. Her outstretched hands demonstrated mild tremors. She had grade I diffuse goitre with no audible bruit over the gland. On cardiovascular examination, her jugular venous pressure was raised to 11 cms above the angle of Louis (manubrio sterna joint). Cardiac apex was felt in 5th intercostal space, just outside the midclavicular line and it was hyperdynamic in nature. On auscultation there were bilateral crepitations and on cardiac auscultation S3 was distinctly audible. Chest radiography showed moderate cardiomegaly. Patient was initially seen at Bharti hospital, Karnal and referred to us for tertiary care.

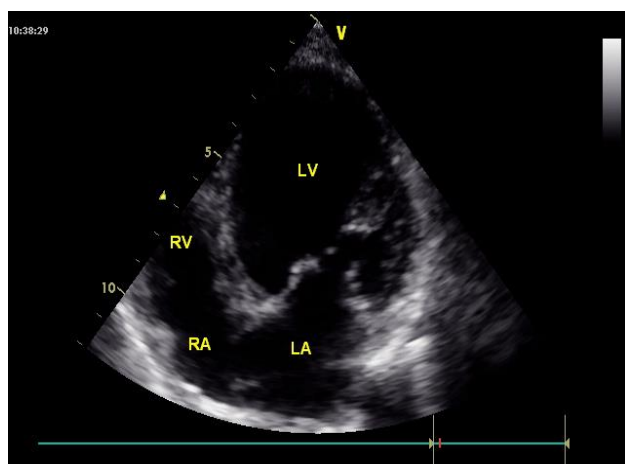
Electrocardiogram showed sinus Tachycardia with non-specific T wave changes. Her blood counts, liver function test, kidney function tests, electrolytes and cardiac enzymes were normal. Transthoracic Echocardiography showed dilatations of all four chambers of heart with global systolic dysfunction. At the time of initial presentation, patient had echocardiographic evidence of severe diastolic dysfunction with moderate RV systolic

dysfunction. Her ejection fraction was 30-35%. Her Technitium<sup>99</sup> scan showed diffuse toxic goiter consistent with Graves' disease. A diagnosis of thyrotoxic cardiomyopathy was made and patient was started with diuretics,  $\beta$  Blockers and antithyroid drugs. The antithyroid drug used to treat her was carbimazole at a dose of 40 mg daily in two divided doses. Propyl thio uracil (PTU) was not used to treat her because the drug is hepatotoxic and is approved only for treating pregnant women in first trimester of pregnancy.

After 6 weeks when she returned for follow-up, she was clinically and biochemically euthyroid. A repeat echocardiogram was performed which showed normal LV and RV systolic functions with an ejection fraction of 55-60%.

**Table 1: Thyroid function tests at different occasions during treatment.**

	Free T3 (2.6-6.8 pmol/l)	Free T4 (12-22 pmol/l)	TSH (0.27-4.2 iu/ml)	TPO ( $\leq$ 35 iu/ml)
<b>TFT at presentation</b>	>20	>100	<0.005	>600
<b>TFT after six weeks</b>	4.6	11.5	0.1	Not done



**Figure 1: Echocardiographic image showing dilatation of four chambers of the heart.**

## DISCUSSION

High circulating levels of thyroid hormone leads to a hyper metabolic state and which in turn leads to hemodynamic changes in the cardiovascular system. Hyperthyroidism is characterized by an increase in resting heart rate, stroke volume, myocardial contractility and cardiac output.

So, hyperthyroid patients manifest with a high output heart failure because these patients have increased cardiac

output and contractility due to thyroid hormone excess. This is in contrast with true heart failure which is characterized by decreased myocardial contractility.

Thyroid hormones have positive chronotropic and inotropic effects on the heart with the result that the cardiac manifestations of hyperthyroidism include arrhythmias, systolic hypertension, wide pulse pressure, pulmonary hypertension, high output cardiac failure and thyrotoxic cardiomyopathy.<sup>3</sup>

We present this case of thyrotoxic cardiomyopathy which can be defined as myocardial damage caused by excess circulating levels of thyroid hormones which leads to dilatation of all the chambers of the heart and subsequent heart failure. Dilated cardiomyopathy secondary to thyrotoxicosis is an unusual clinical presentation. Thyrotoxic cardiomyopathy is the initial clinical presentation in approximately 6% of patients with hyperthyroidism.<sup>4</sup> Goland et al reported two case of dilated cardiomyopathy in hyperthyroidism, and they had recovered without any sequel.<sup>5</sup>

$\beta$  adrenergic blockers play an important role in management of cardiac manifestations in hyperthyroid patients especially in treatment of tachyarrhythmias and cardiac failure.  $\beta$  adrenergic blockers reduces both resting as well as exercise heart rate, thereby improving ventricular filling pressure and eventually improving cardiac output.

Hyperthyroidism can aggravate pre-existing heart disease and can also lead to atrial fibrillation, worsening of angina pectoris, congestive cardiac failure and dilated cardiomyopathy.<sup>6</sup> Patients presenting with heart failure and dilated cardiomyopathy may have hyperthyroidism as the underlying cause. Early and meticulous restoration of euthyroid state in these patients will restore the left ventricular functions.

## CONCLUSION

Thyrotoxic cardiomyopathy is a known complication of hyperthyroidism. It can occur across all age groups but elderly people who are already suffering from other co-morbid conditions or structural heart disease are more susceptible.

Radiography of chest and echocardiography are two non-invasive modalities which can play a vital role in diagnosis and follow-up of these cases. Although thyrotoxic cardiomyopathy is usually reversible with early and prompt treatment with anti-thyroid medication,  $\beta$  Blockers, diuretics, but untreated and severe heart failure can be fatal.

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