

## Case Report

# Reversible Dilated Cardiomyopathy and Hyperthyroidism in a Patient with Ischemic Heart Disease

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### ABSTRACT

This article gives a clue about hyperthyroidism that may present as cardiomyopathy; how to diagnose and manage such cases.

KEY WORDS: myocardial dysfunction, paroxysmal nocturnal dyspnea, thyroid function test

### INTRODUCTION

Thyrotoxicosis is a well known cause of high cardiac output failure and gives rise to arrhythmias. In addition, it is a recognized, but rare, contributor to dilated cardiomyopathy with severe impairment of left ventricular function. A full recovery of myocardial dysfunction is usually observed once an euthyroid state is achieved<sup>[1]</sup>. A 46-year-old female with severe impairment of myocardial function is presented. She had a co-existing hyperthyroid state and coronary vessel disease. However, following a spontaneous remission of her hyperthyroidism, which was possibly due to transient thyroiditis, her left ventricular function improved dramatically. This article highlights thyrotoxicosis as a potentially treatable and reversible cause of heart failure. The mechanisms by which the thyroid hormones affect the cardiovascular system are elaborated<sup>[2-6]</sup>. Other causes of reversible cardiomyopathy are also outlined<sup>[7-12]</sup>. Literature on case reports demonstrating the association between thyrotoxicosis and low cardiac output failure are reviewed<sup>[13-19]</sup>. Finally, a brief outline on the treatment approach to such patients with left ventricular dysfunction and hyperthyroidism is given<sup>[6]</sup>.

### CASE REPORT

A 46-year-old female was admitted to Amiri Hospital Coronary Care Unit on 14/3/1997 through the emergency room with acute pulmonary edema. She was suffering for the last month prior to admission from exertional shortness of breath and attacks of paroxysmal nocturnal dyspnea. She was a diagnosed case of long-standing insulin dependent diabetes mellitus and hypertension. She was a heavy smoker. Serial

ECG's recorded persistent left bundle branch block. Chest X-ray showed cardiomegaly with left ventricular failure that cleared later. Serial cardiac enzymes were normal. For the next few days, she continued to complain of chest pain and shortness of breath.

She was kept on tridil and heparin infusions plus enalapril, baby aspirin and diuretics. Gated blood pool study done on 17/3/1997 showed the same picture of dilated left ventricle with a left ventricle ejection fraction of 30%.

In view of her underlying risk factors and unstable condition, she was shifted to the chest hospital for early coronary angiogram on 24/3/1997. The coronary angiogram reported a dilated left ventricle with global akinesia and a left ventricle ejection fraction of 35%. The left main stem artery was normal, left anterior descending showed a 50% distal stenosis. Ramus intermedius was large with 50% stenosis in two of its divisions, left circumflex had a mid segment stenosis and the right coronary artery was dominant with 99% mid segmental stenosis. Percutaneous trans thoracic coronary angioplasty of the right coronary artery was attempted but failed and was postponed as patient was quite tachycardic. Thyroid function report came back on 7/4/1997 with a picture of frank thyrotoxicosis with F-T4 of 63.21 pmol/l (N.R.12-24 pmol/l) and thyroid stimulating hormone (TSH) of 0.063 MIU/ml (N.R 0.23 - 5 MIU/ml). ESR was 60/hr. As she was clinically stable, she was discharged on baby aspirin, isordil, enalapril, and furosemide and referred to thyroid clinic for radioactive iodine treatment.

However, the lady decided not to visit the thyroid clinic and traveled to her native country for four months leave without receiving any specific treatment. She reappeared in my medical

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outpatient on 30/8/1997. She was symptom-free. Clinically she was euthyroid and had a palpable soft goiter. She apparently did not receive any specific anti thyroid treatment. No thyroid scan was done during her hyperthyroid state, as she failed to seek medical advice.

I decided to reevaluate her. A repeated ECG was completely normal. Chest X-ray was within normal. ESR was 20/hr. Repeated thyroid function were entirely normal (FT4 14 Pmol/l and TSH: 0.33 MIU/ml). A thyroid scan reported a diffuse goiter with normal uptake and thyroid antibodies were negative. A 24-hour ECG record was normal with no record of bundle branch block. A repeated echo on 4/10/1997 showed a normal global left ventricle systolic function with left ventricle ejection fraction of 60%. Activated gated blood pool study done at the same time showed a normal left ventricle with left ventricle ejection fraction of 68%.

She was continued on her antianginal and antihypertensive medication (B. aspirin, Diltiazem 60 mg x 3, Enalapril 20 mg x 1) and was doing quite well.

I re-referred the patient to the chest hospital for re-evaluation of her coronary vessels and need for any further intervention. Regular checkup of thyroid function will be needed.

## DISCUSSION

This lady evidently had a transient form of thyroiditis associated with hyperthyroidism which presented as frank low cardiac output failure confirmed by Echocardiogram, gated blood pool study and coronary angiogram. As she became euthyroid, she was clinically free apart from effort stable angina explained by her underlying coronary vessel disease. However, she definitely retained her normal systolic cardiac function (as documented by Echocardiogram and gated blood pool study) without receiving any definite therapy for her underlying coronary vessel disease.

A delay in the diagnosis of her hyperthyroid state had probably led to premature referral of the patient for coronary angiogram and thus the failure of the balloon angioplasty. She definitely needed to treat her thyrotoxic state before proceeding with any definite invasive coronary intervention which could have been quite hazardous to the patient. A thyrotoxic crisis could have been precipitated by injecting contrast material containing iodine<sup>[6]</sup>. I do not deny that her underlying coronary vessel disease was significant and acute coronary ischemia could not be ruled out as another adding factor to her low cardiac output state. Unfortunately, a thyroid uptake scan was not done during the hyperthyroid state which made it difficult to establish the exact pathology of her

underlying transient hyperthyroid condition. The most probable diagnosis is a transient form of acute thyroiditis. She will definitely need regular follow up of her thyroid function.

The aim of this case review is to discuss the direct relationship of hyperthyroidism with reversible cardiomyopathy. Thyrotoxicosis has been associated commonly with high cardiac output failure but reports that caused dilated cardiomyopathy are rare.

Excess thyroid hormones can lead to direct cardiac disease through different potential mechanism, either at the cellular level through a nuclear-receptor mediated effect; at extranuclear sites, and affecting plasma membrane function. Other mechanisms include direct interaction with the sympathetic nervous system, a direct chronotropic effect independent of catecholamine's effect<sup>[1-2]</sup>. Different studies have also demonstrated that thyroid hormones have a direct effect on myocardial contractility and left ventricle diastolic function. Hyperthyroidism also has its potential effects on the peripheral circulation with documented increase in the blood volume, decrease in the peripheral resistance, increase in the mean blood pressure and proven increase in the atrial natriuretic factor<sup>[3-6]</sup>. Chronic tachycardia and arrhythmia, e.g., AF, have been reported as causes of cardiomyopathy<sup>[7-8]</sup>.

Reversible cardiomyopathy has been commonly reported in the literature. Endocrine dysfunction, apart from thyroid dysfunction, includes case reports of association of reversible cardiomyopathy with Addison's disease, Pheochromocytoma, congenital adrenal hyperplasia, growth hormone deficiency and hyperparathyroidism<sup>[9-12]</sup>.

It is essential to remember that heart failure can be the only manifestation of thyrotoxicosis and underlying heart disease may be absent. Clinical data support the existence of a reversible cardiomyopathy in hyperthyroid patients, however, reports are rare but they all show that most if not all, of the cardiac abnormalities return to normal once an euthyroid state has been achieved. The factors involved, as explained earlier, include decreased left ventricle contractile reserve, left ventricle hypertrophy with impaired left ventricle filling, existence of atrial fibrillation, decreased peripheral vascular resistance and increased blood volume (usually factors in high cardiac output failure) and finally increased myocardial O<sub>2</sub> demand.

I have come across few reports of documented clinical cases of thyrotoxicosis that presented as low cardiac output failure. These include around 11 patients who had documented hyperthyroid state and left ventricle systolic dysfunction proven by 2D-Echocardiogram. All were managed by

antifailure and antithyroid drugs. They improved rapidly and were followed up by echoes once euthyroid, done 6-12 months after, showed restoration of most if not all of the left ventricle normal function<sup>[13-17]</sup>.

There were autopsy reports of two clinical cases of thyrotoxic ladies, who were admitted with acute pulmonary edema and expired. The autopsy reported enlarged hearts, with normal coronaries, dilated ventricles with histopathology report of myocyte hypertrophy and myocardial edema<sup>[18]</sup>.

There was only one report of four cases of thyrotoxicosis associated with irreversible cardiomyopathy. They were diagnosed between the years 1978-1992. Their thyroid functions normalized but they remained in low cardiac output failure. They all had coronary angiography that showed normal coronaries with high left ventricle end diastolic volume and low left ventricle ejection fraction. A myocardial biopsy was reported normal<sup>[19]</sup>.

Once transient thyroiditis has been ruled out, patients with thyrotoxicosis can be offered radioactive iodine ablation, antithyroid drugs or surgical intervention. However, in the subgroup of thyrotoxic patients who present with low cardiac output failure, the recommended approach is to start them on antithyroid drugs for 4-8 weeks and then to stop for 3-5 days, followed by ablation with radioactive iodine. Ablation is indicated because of the increased risk of recurrent cardiac disease if thyrotoxicosis recurred. Heart failure is usually treated with diuretics and high dose of Digoxin-B-blockers should be introduced later once the patient is not in frank clinical failure<sup>[6]</sup>.

## CONCLUSION

Dilated cardiomyopathy is a rare clinical disorder of thyrotoxicosis. Direct action of the thyroid hormone on the heart leading to disproportional structural and functional changes may be responsible for dilated cardiomyopathy that is reversible with treatment.

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