

An unusual presentation of thyrotoxicosis

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ABSTRACT

Cardiac dysfunction in thyrotoxicosis is believed to arise from changes in preload, afterload, heart rate, and contractility. Cardiomyopathy is a less frequent cardiac complication linked to thyrotoxicosis. Here, we present a rare case of thyrotoxicosis induced cardiomyopathy in a 30-year-old male, where dilated cardiomyopathy manifested as the initial sign of thyrotoxicosis. The patient was treated with antithyroid drugs, diuretics and beta blockers. General surgery opinion obtained and planned to do thyroidectomy after euthyroid status. Patient was followed up and after 6 weeks was symptomatically better and was found to be euthyroid and repeat 2D ECHO showed an improvement in systolic function with EF of 55% and the patient was planned for thyroidectomy. In the management of thyrotoxicosis induced cardiomyopathy, pharmacological interventions involve the use of specific thyroid therapies such as thionamides (methimazole, carbimazole, propylthiouracil) and beta-blockers, coupled with heart failure management.

Keywords: thyrotoxicosis, dilated cardiomyopathy, carbimazole, hyperthyroid, heart failure

INTRODUCTION

Thyrotoxicosis is a clinical syndrome characterized by a hypermetabolic state, stemming from elevated levels of thyroid hormones in the bloodstream, particularly free thyroxine (T4) and/or triiodothyronine (T3) [1,2]. Increased activity, irritability, dysphoria, heat intolerance, excessive sweating, palpitations, fatigue, weight loss accompanied increased appetite, diarrhoea, polyuria, oligomenorrhoea, and reduced libido are common indicators of thyrotoxicosis [3,4].

Altered preload, afterload, heart rate, and contractility are considered as contributing factors to cardiac dysfunction in thyrotoxicosis [2]. Cardiomyopathy, a primary disorder of the heart muscle leading to abnormal myocardial performance, is a less frequent cardiac complication associated with thyrotoxicosis [3]. Thus, we report a case of a 30-year-old male with cardiomyopathy presenting as the initial manifestation of thyrotoxicosis.

CASE DETAILS

A 30-year-old male, with no known comorbidities, presented with complaints of dry cough, swelling of

the face and bilateral lower limbs. He reported a history of dyspnea (grade 4) with a progressive course associated with orthopnea, and palpitations over the past month. Additionally, he noted symptoms of heat intolerance, excessive sweating, and tremors.

During the examination, the patient showed a body temperature within the normal range, a respiratory rate of 28 per minute, and a blood pressure of 130/70 mmHg. Additionally, a steady pulse of 125 beats per minute was observed and he had bilateral pitting pedal edema. Graves's ophthalmopathy was evident with a moderate exophthalmus and lid retraction, although no lid lag was observed. The hands were warm and moist and tremors were observed. Neck examination revealed an enlarged thyroid gland. Cardiovascular examination revealed an elevated jugular venous pressure, a displaced apex beat downwards and laterally, the presence of LV S3 at apex, and a loud P2 in pulmonary area on auscultation. Respiratory examination revealed bilateral crackles.

The chest radiograph revealed cardiomegaly (Figure 1). The electrocardiogram displayed sinus tachycardia and nonspecific T wave changes. A plain CT thorax indicated mild pleural effusion on the right

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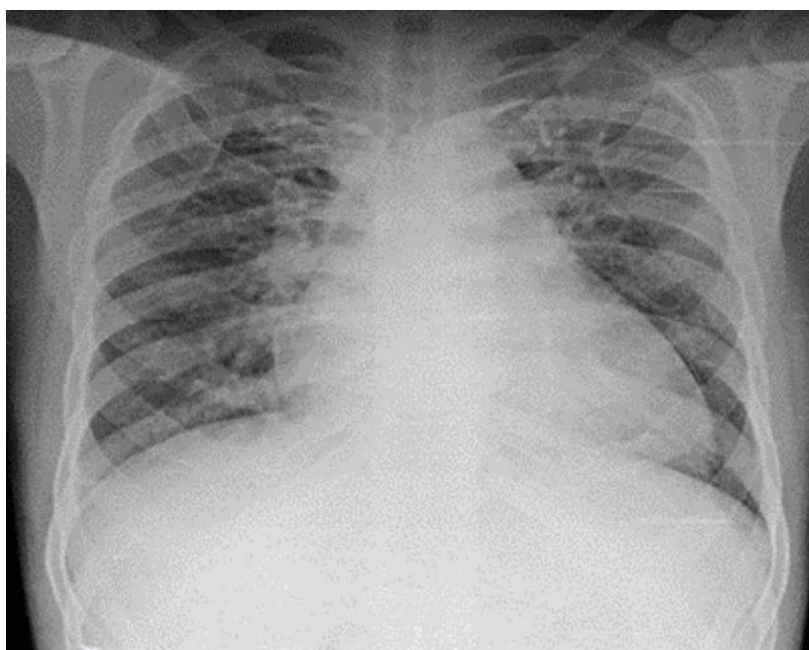


FIGURE 1. Chest X-ray shows cardiomegaly



FIGURE 2. CT thorax shows right mild pleural effusion, minimal ascitis, cardiomegaly with dilated IVC, minimal pericardial effusion

side, minimal ascites, cardiomegaly with a dilated inferior vena cava, suggesting a need for correlation with echocardiography. Additionally, minimal pericardial effusion was noted, and the thyroid gland appeared bulky and hypodense, prompting the recommendation for thyroid function tests and ultrasound correlation.

Echocardiography was suggestive of dilated cardiomyopathy showing global hypokinesia of the left ventricle (LV), moderate LV systolic dysfunction (EF: 40%), Grade II LV diastolic dysfunction, dilation of all cardiac chambers, mild mitral regurgitation, mild tricuspid regurgitation, and underestimated tricuspid regurgitation pressure gradient due to right ventricular dysfunction. Severe pulmonary artery hypertension was observed, along with right ventricular dysfunction (TAPSE-1.1 cm), dilated inferior vena cava (3.0 cm, non-collapsing), trace pericardial effusion, and no clot (Figure 3).

The final diagnosis of thyrotoxicosis induced cardiomyopathy was made. The patient was treated with thionamides – T. Carbimazole 10 mg twice a day and escalated to 10 mg thrice a day, anti failure medication including diuretics and beta blockers like Propranolol 20 mg twice a day. Patient was planned for thyroidectomy after attaining euthyroid status at a later date.

TABLE 1. Laboratory investigations

Lab test	Result	Normal range
Hb	13 g/dl	13-17 g/dl
Total counts	7651 cells/ cu mm	4000-10000 cells/ cu mm
Platelets	2.3 lakhs/cu mm	1.5-4.5 lakhs/ cu mm
Urea	19mg/dl	17-43mg/dl
Creatinine	0.9mg/dl	0.7-1.4mg/dl
Sodium	140 mEq/L	135-145 mEq/L
Potassium	4.4 mEq/L	3.5-5 mEq/L
Albumin	4g/dl	3.5-5 g/dl
Free T3	>22.8 pg/ml	2.77-5.27 pg/ml
Free T4	>6.99 ng/dl	0.78-2.19 ng/dl
TSH	<0.015 mIU/ml	0.46-4.68 mIU/ml

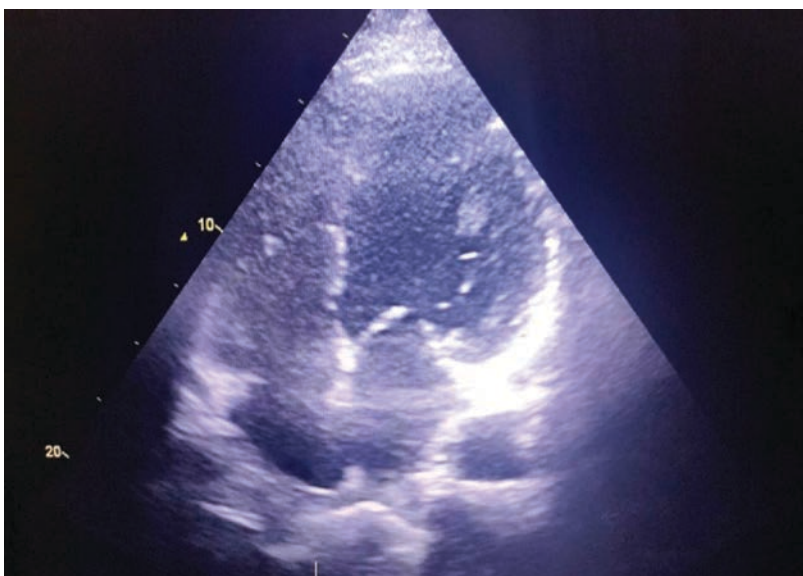


FIGURE 3. 2D echocardiography suggestive of dilated cardiomyopathy showing global hypokinesia of left ventricle with moderate LV systolic dysfunction with EF -40%

Clinical Outcome – The patient was followed up after 6 weeks, was symptomatically better, and was found to be euthyroid and repeat 2D ECHO showed an improvement in systolic function with EF of 55% and planned for thyroidectomy.

DISCUSSION

Elevations in resting heart rate, blood volume, stroke volume, myocardial contractility, ejection fraction, and diastolic relaxation are all signs of hyperthyroidism. These changes closely resemble a condition of heightened adrenergic activity [5]. In cases where underlying heart disease exists, thyrotoxicosis can potentially trigger myocardial infarction or congestive cardiac failure.

Dilated cardiomyopathy is defined as a progressive condition affecting the myocardium leading eventually to impaired ventricular contractility. Although, it is considered as an idiopathic condition, but it was found that factors like genetic, infections, pregnancy, alcohol consumption, and endocrine disorders in particular “thyroid disorders” are some of the causes in developing reversible DCM [6,7].

The term “thyrotoxic cardiomyopathy” denotes cardiac damage triggered by an excess of thyroid hormone, leading to alterations in intracellular metabolism, myofibril contractile activity, and myocyte energy production. Key symptoms include diastolic dysfunction, heart failure, pulmonary arterial hypertension (PAH), diastolic dysfunction, and left ventricular hypertrophy in primary atrial fibrillation [8]. Without intervention, if left unaddressed, an unchecked high-output state coupled with hyperthyroidism may progress to ventricular dilation, persistent tachycardia, and eventually lead to chronic heart failure, carrying the potential for a life-threatening outcome [9].

Pump function was restored in previously discussed cases when antithyroid medications such as propylthiouracil, methimazole or carbimazole were included to the patient's treatment. When thyrotoxic dilated cardiomyopathy is rapidly diagnosed and

treated, a distinguishing feature of its course is the relatively quick leveling of heart volumes [10-12].

In the management of thyrotoxic dilated cardiomyopathy, evidence-based pharmacological interventions applicable to cardiac arrest should consistently be complemented by use of thyroid therapies which includes thionamides. The prolonged onset of action of these thyroid therapies and their effectiveness can be enhanced by the intravenous administration of small doses of beta-blockers [13].

In the management of cardiac symptoms in hyperthyroid patients, β -adrenergic blockers are particularly useful for addressing tachyarrhythmias and cardiac failure. These blockers effectively decrease both resting and exercise heart rates, thereby enhancing ventricular filling pressure and ultimately improving cardiac output [14].

Thyrotoxic cardiomyopathy is a recognized complication of hyperthyroidism. Timely and careful restoration of a euthyroid state in these individuals contributes to the recovery of left ventricular functions as in our patient. The attainment of a lasting euthyroid status holds significance, as it correlates with a more favorable diagnosis and enhances vascular parameters, including heart rate, the occurrence of extrasystoles, and cardiac output [15].

CONCLUSION

Dilated cardiomyopathy, an early sign of thyrotoxicosis, shows promise for reversal with appropriate therapy. It is important to recognize because it involves ruling out other diagnoses, and identifying it quickly is crucial as a reversible cause of heart failure, achieving a euthyroid state with antithyroid drugs is crucial. Our case emphasizes a comprehensive approach with antithyroid drugs, β -blockers, and euthyroid restoration. The positive outcome reinforces timely intervention's potential to mitigate cardiac issues. In conclusion, thyrotoxic cardiomyopathy underscores the reversibility of certain heart failures, emphasizing the importance of swift diagnosis and targeted therapies for optimal outcomes.

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