Case Report

Irreversible Dilated Cardiomyopathy Due to Thyrotoxicosis

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Abstract

Irreversible dilated cardiomyopathy due to thyrotoxicosis is an unusual clinical entity. We report this case of a young female who presented with congestive cardiac failure and was diagnosed as dilated cardiomyopathy due to thyrotoxicosis. Restoration of euthyroid levels did not revert the cardiomyopathy. ©

INTRODUCTION

Cardiac involvement is common with thyroid disorders. However, irreversible dilated cardiomyopathy due to thyrotoxicosis is an unusual clinical entity. We report this case who had dilated cardiomyopathy as an initial symptom of thyrotoxicosis that did not resolve inspite of the restoration of the euthyroid levels.

CASE REPORT

A 36 years female, housewife, presented with chief complaints of progressive dyspnoea on exertion, palpitations, oligomenorrhoea for 2 months, puffiness of face and oedema feet for 15 days. There was no history of orthopnoea, paroxysmal nocturnal dyspnoea, chest pain, oliguria and hematuria. There was no history of any cardiac disease in the past.

On examination, she was afebrile, pulse was 124/minute, regular and bounding in nature. Blood pressure was 130/84 mm Hg. She had pallor, oedema feet, exophthalmos and lid lag. There was no thyroid swelling. Fine tremors were present in both the hands. The apex impulse was in the 5th intercostal space, hyperdynamic in nature, just outside the mid-clavicular line. First heart sound was loud, S3 gallop was present. There was grade 3 systolic flow murmur in the mitral area. There were no rales in the chest.

The skiagram of the chest showed cardiomegaly. The ECG showed sinus tachycardia with a left bundle branch block. 2D-echo was suggestive of dilated cardiomyopathy with left ventricular ejection fraction (LVEF) of 24%. The thyroid hormones were elevated: T3 – 260 ng/dl (80-200), T4 – 17.6 ug/dl (6-11.8), TSH – 0.03 uiu/ml (0.4-3.1) [The numbers in the bracket indicate the normal range]. Ultrasonography of the thyroid was normal. The thyroid scan showed diffuse radiotracer uptake suggestive of thyrotoxicosis. Thus, a diagnosis of dilated cardiomyopathy due to thyrotoxicosis was made.

The patient was treated with Diuretics, ACE inhibitors, Digoxin, Neomercazole and Propranolol with a regular follow up. 2D-echo was repeated after 3 months, 6 months and 1 year which did not show any improvement in the ejection fraction. The thyroid profile normalized after 1 month of treatment and remained normal on subsequent follow up at 3, 6 and 12 months of treatment.

DISCUSSION

Cardiac dysfunction in thyrotoxicosis is believed to be caused by altered preload, afterload, heart rate and contractility. It can cause circulatory alterations, including increase in total blood volume, decrease in total systemic vascular resistance and shortened circulation time. The increase in cardiac work can lead to cardiac hypertrophy, which results in reduction in the contractile reserve that is reflected in the failure of exercise to create an increase in the ejection fraction, a pattern that can be reversed after thyroid suppression treatment.1 Thyrotoxicosis may precipitate myocardial infarction or congestive cardiac failure if underlying heart disease is present.2 Whether hyperthyroidism can cause a dilated cardiomyopathy is controversial. Reversible cardiomyopathy due to thyrotoxicosis is rare, with very few cases reported.2,3,5,6 However, Ebisawa et al reported that cardiomyopathy in patients with thyrotoxicosis may be irreversible even 15 years after successful treatment of their thyrotoxicosis. Four patients with this condition had increased left ventricular end-diastolic volumes and decreased ejection fraction, even after 13 to 15 years following treatment of their

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hyperthyroidism. Myocardial biopsies, performed in two patients, showed no specific light microscopic abnormalities. The exact cause of this cardiomyopathy remains unknown.

Besides its effect on the peripheral circulation, a direct action of thyroid hormone on the heart leading to disproportional structural and functional changes of myocytes appears to be responsible for clinical features of dilated cardiomyopathy seen in thyrotoxicosis. This case adds further evidence of the association between thyrotoxicosis and dilated cardiomyopathy in adults. It is important to appreciate that dilated cardiomyopathy may be the initial manifestation of thyrotoxicosis because it can be reversed with an appropriate therapy. Although there have been a few case reports where cardiomyopathy due to thyrotoxicosis was reversed with treatment, there was no improvement in our patient at the end of 12 months of regular treatment.

REFERENCES