Reversible Cardiomyopathy

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Abstract

We report a case of reversible dilated cardiomyopathy, in a middle-aged male. The patient presented with severe left ventricular dysfunction and atrial fibrillation. Inspite of vigorous medical therapy there was only mild clinical improvement. Subsequently laboratory test results diagnosed it as hyperthyroidism and then specific thyrostacic treatment was added. There was a prompt clinical and hemodynamic improvement in the form of reversal of left ventricular dysfunction and achievement of sinus rhythm at the end of two weeks. ©

INTRODUCTION

Cardiomyopathies are diseases that involve the myocardium directly and not secondary to hypertension, congenital, valvular, coronary artery disease or pericardial abnormalities.1 Reversible cardiomyopathy may be found with alcohol use, pregnancy, selenium deficiency, hypophosphatemia, hypocalcemia, thyroid disease, cocaine use and chronic uncontrolled tachycardia. Thyrotoxicosis is one of the rare but important cause of reversible cardiomyopathy. Awareness of this possible presentation of hyperthyroidism is essential while evaluating patients of dilated cardiomyopathy.2 There are very few case reports, which have documented thyrotoxic dilated cardiomyopathy.

CASE REPORT

A 40 years non-diabetic, non-hypertensive male patient presented with history of exertional breathlessness, palpitations, swelling over both lower limbs of one month duration and paroxysmal nocturnal dyspnea fifteen days prior to admission.

On examination patient had low volume, irregularly irregular pulse, respiratory rate of 40/minute, blood pressure of 100/60 mm Hg and raised jugular venous pressure. Patient had mild pallor, pedal edema and diffuse enlargement of thyroid gland (Fig. 1) with thyrotoxic ophthalmic features and thyroid acropachy. On cardiovascular examination, heart sounds were soft with gallop rhythm and soft systolic murmur at apical area. Respiratory examination revealed reduced breath sounds with bilateral basal rales. Abdominal examination revealed tender hepatomegaly with evidence of free fluid.

On investigations, hemoglobin was 9.8 gm% (normocytic normochromic). Liver function tests were deranged (bilirubin total 3.6 mg%, direct 2.6 mg%, indirect 1.0 mg%, AST 55 IU and ALT 60 IU) while renal functions were within normal limits. Chest radiograph (Fig. 2a) revealed bilateral vascular congestion with pleural effusion and cardiomegaly while electrocardiogram showed evidence of atrial fibrillation. Two-dimensional echocardiography on second day of admission revealed global left ventricular systolic dysfunction with moderate mitral regurgitation and ejection fraction of 30%. Ultrasonography of abdomen showed mild hepatomegaly with normal echotexture, dilated inferior vena cava and free fluid. Thyroid functions showed raised T3, T4 and very low TSH (T3 8.53 ng/ml, T4 20.2 µg/dl, TSH <0.01 µU/ml).

Ultrasongraphy of neck showed bilaterally symmetrical, diffuse enlargement of thyroid gland without any focal lesion.

Patient responded partially to the standard treatment of congestive cardiac failure. After addition of tablet Carbimazole 10 milligram three times a day along with a beta-blocker patient showed dramatic improvement. At the end of two weeks patient improved clinically in the form of reduction of signs of congestive cardiac failure, radiologically with resolution of pleural effusion and cardiomegaly (Fig. 2b), electrocardiographically in the form of achievement of sinus rhythm and echocardiographically with improvement in left ventricular ejection fraction to 41% and near normalisation of thyroid function tests (T3 1.36 ng/ml, T4 7.2 µg/dl, TSH 0.4 µU/ml).

DISCUSSION

Thyroid hormone has profound effects on number of metabolic processes in virtually all organs and can produce dramatic cardiovascular effects. Its action on the heart can be grouped into three broad categories: direct cardiac effects, effects mediated by sympathetic...
nervous system and effects secondary to hemodynamic changes.¹ Direct thyroid effects can be nuclear or extranuclear which leads to changes in the proportion of myosin heavy chain protein from $\beta$ to $\alpha$ thus enhancing velocity of contraction and diastolic relaxation.³ Thyroid hormone also increases transcription of calcium-ATPase gene and ATP consumption which augments transsarcolemmal calcium influx. It has been proposed that many cardiovascular effects of hyperthyroidism, i.e., tachycardia, systolic hypertension, increased cardiac output and myocardial contractility are because of not only increased activity of sympato-adrenal system but also increased responsiveness of cardiac tissue to catecholamines by upregulating beta adrenergic receptors and post-receptor G protein milieu. Thus, while thyroid hormone itself has major direct effect on modifying protein synthesis, changes in cardiac workload may also contribute. The tachycardia observed in hyperthyroidism appears to be due to combination of increased rate of diastolic depolarization and decreased duration of action potential in the sinoatrial nodal cells. The propensity for the development of atrial fibrillation may be due to the shortened refractory period of atrial cells.

The mainstay of treatment is beta-adrenergic blocking agent and definitive treatment of the hyperthyroidism. Prompt treatment of hyperthyroidism can significantly reduce associated cardiovascular symptoms. The most useful agents are antithyroid drug like carbimazole which inhibit thyroid hormone synthesis.

There are similar isolated case reports found in patients of thyrotoxicosis.³⁻⁵ Thus we conclude that thyrotoxicosis though rare, is one of the potentially reversible cause of dilated cardiomyopathy.

REFERENCES