Hyperthyroidism-induced dilated cardiomyopathy

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Abstract

Hyperthyroidism is a common endocrine disorder with a prevalence of 1.3% in the general population, affecting more women than men. Prolonged hyperthyroidism without appropriate management may lead to high output cardiac failure characterized by increases in heart rate, cardiac contractility, and cardiac output and by reductions in peripheral systemic vascular resistance. Dilated cardiomyopathy with impaired systolic function is rare and occurs in less than 1% of patients with thyrotoxicosis. The exact mechanism of hyperthyroidism-induced dilated cardiomyopathy is not well established. The combination of direct toxic effects of excess thyroid hormone along with prolonged tachycardia, arrhythmia, and a hyperdynamic state could be contributing factors. We present a case of a young woman with prolonged sinus tachycardia due to a long history of medication non-compliance who developed dilated cardiomyopathy with low output heart failure. Early detection and management of hyperthyroidism are crucial to restore cardiac function.

Keywords: hyperthyroidism, dilated cardiomyopathy, impaired ventricular systolic function

Introduction

The thyroid gland serves as an important regulator of metabolism, and its hormones affect many organs, including the cardiovascular system. Thyroid hormones have a significant role in regulating cardiac chronotropism and inotropism. Studies suggest that thyroid hormone has a direct effect on cardiac chronotropy due to early enhancement of beta-adrenoreceptors, followed by a late modification of the electrophysiological properties of the myocardium.[1] In another study done with mouse models, thyroid hormone-treated myocardium modulated peak calcium influx and increased inotropy as compared to a hypothyroid myocardium.[2] Thyrotoxicosis can cause a high-output cardiac state characterized by increases in heart rate, cardiac contractility, and cardiac output and reductions in peripheral systemic vascular resistance, which then can lead to high-output heart failure. Although unusual, hyperthyroidism also causes low-output heart failure in 6% to 15% patients.[3] Even in those patients, dilated cardiomyopathy with impaired systolic function is rare. We report a case of hyperthyroidism induced dilated cardiomyopathy.

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Case

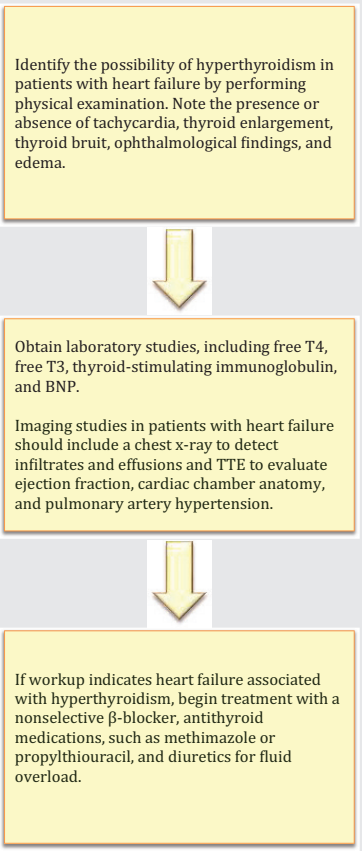
A 26-year-old woman with history of hyperthyroidism presented to our hospital with increasing dyspnea, orthopnea, and leg edema. She had been diagnosed with hyperthyroidism three years prior to presentation but was noncompliant with treatment. Physical examination revealed sinus tachycardia, proptosis with intact ocular movements, diffuse mobile goiter, crepitation of bilateral lungs, right upper quadrant abdominal tenderness, and peripheral edema. Laboratory results showed markedly elevated free T3 and free T4 with suppressed TSH, elevated thyroid-stimulating immunoglobulin, mild hyperbilirubinemia, and elevated

B-type natriuretic peptide. Chest x-ray revealed bilateral pleural effusions. Transthoracic echocardiography showed an ejection fraction of 35-39% with moderate global hypokinesis, dilatation of all cardiac chambers, and moderate pulmonary artery hypertension. The patient was diagnosed with congestive heart failure associated with her hyperthyroidism. Treatment with antithyroid drugs and propranolol was started, leading to significant improvement. However, a week after hospital discharge, she again presented with decompensated heart failure due to noncompliance secondary to financial constraints. Treatment with antithyroid and heart failure medications was resumed, and total thyroidectomy was recommended.

Discussion

Uncontrolled hyperthyroidism can lead to ventricular dilation, persistent tachycardia, and eventual chronic heart failure.[3] Thyroid hormone centrally stimulates activity in the sympathetic nervous system by positively regulating β1-adrenergic receptors. Additionally, the hormone upregulates sarcoplasmic reticulum Ca^{2+} ATPase, involved in the process of excitation-contraction coupling and calcium-induced calcium release.[4] Calcium release from the ryanodine receptor in the sarcoplasmic reticulum activates the cardiac myofilament and results in positive inotropy observed in hyperthyroid states.[5] Free T3 and free T4 increase the expression of the more rapid contractile isoforms of the alpha-myosin heavy chain, which further contribute to enhanced systolic function. Independently, free T3 increases the rates of depolarization and repolarization of the SA node and thus increases the heart rate.[4] The consequence of these changes is a combination of inotropic and chronotropic effects that along with increased adrenergic sensitivity lead to increased heart rate and contractility. Thyroid hormone also stimulates erythropoietin secretion contributing to increased blood volume.[6] Altogether, these alterations in the hemodynamic status with increases in cardiac output and blood volume may cause high output congestive heart failure.

Less than 1% of patients with thyrotoxicosis develop dilated cardiomyopathy with impaired left ventricular systolic function.[7] The exact mechanism of hyperthyroidism-induced dilated cardiomyopathy is not well established. The combination of direct effects of excess thyroid hormone (manifesting as altered myocyte energy production, change in intracellular metabolism, and myofibril contractile dysfunction) along with prolonged tachycardia and arrhythmia could be factors contributing to development of cardiomyopathy.[4,7,8] Advanced age and preexisting comorbidities, such as hypertension, valvular heart disease, and ischemic heart disease, increase the risk of heart failure with reduced systolic function. Our patient did not have any documented episodes of atrial fibrillation but did have prolonged sinus tachycardia due to long history of noncompliance. This is a potential explanation for heart failure as the cardiac contractility was unable to compensate with the increased heart rate. We cannot



This figure outlines an approach to the identification and management of patients with cardiomyopathy secondary to hyperthyroidism.

completely exclude the possibility of episodes of silent paroxysmal atrial fibrillation. Our patient was young and no prior existing heart conditions, suggesting that she was otherwise low-risk for developing heart failure. This case shows that even in the absence of other risk factors, hyperthyroidism in young people can precipitate cardiomyopathy. There are reports that most cases of hyperthyroidism induced dilated cardiomyopathy are reversible. However, in a minority of patients it can be irreversible.[9]

It is important to suspect hyperthyroidism in patients presenting with unexplained heart failure. Heart failure patients with abnormal thyroid function have a 60% higher risk of mortality compared to euthyroid patients with heart failure.[10] However, there are no long-term studies available showing that intervening in thyroid dysfunction changes the incidence or prognosis in individuals with heart failure.[11] Treatment with β-adrenergic blockage for heart rate reduction and diuretics for fluid reduction is crucial for symptomatic management. Timely detection and appropriate treatment may help in cases such as this one.

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