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Graves Disease Induced Dilated Cardiomyopathy

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Summary

Gravis' thyrotoxicosis is rarely complicated with heart failure. Here in, we present a case of 45 years old female without past medical history of cardiac disease who admitted to our hospital for altered mental status. Hospitalization course complicated with Atrial fibrillation with RVR and symptoms of acute heart failure. Subsequent echocardiogram revealed dilated cardiomyopathy. Labs were concerning about thyroid storm. Patient treated with Methimazole, steroids and Lugols iodine drops for thyroid storm and received appropriate management for heart failure.

Introduction

Graves' thyrotoxicosis has many cardiovascular complications; however, the most cardiac complication is atrial fibrillation but rarely causes heart failure. Less than 1% develops dilated cardiomyopathy with impaired left ventricular systolic function. In this case report we describe a case of Graves' hyperthyroidism-induced reversible cardiomyopathy.

Case Presentation

45-year-old female with a history of previously treated Graves' disease who presented to hospital for altered mental status and severe hypoglycemia. The hospital course complicated by Atrial fibrillation with RVR. Patient states that recently started feeling fatigue, orthopnea, decrease

exercise intolerance, lower extremities edema and distended abdomen. In emergency department, found to have hypotension tachycardia. She was given IV fluids but her blood pressure didn't respond. Patient was started on norepinephrine for hypotension. Physical examination demonstrated impressive proptosis, positive jugular venous distension, irregular irregularity of her plus and +2 lower extremities edema. Patient found to have Graves storm (TSH: 0.07 uIU/ml, Free T4: 1.89 ng/dL, T3: 36.6 ng/dL, cortisol level: 59.36 Ug/dL). She was started on Methimazole, steroids and Lugols iodine drops. Hypoglycemia that she had most likely was related to lack of glycogen stores and increased metabolic demand with graces. After the patient stabilized, echocardiogram obtained

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showed severe left ventricular dysfunction (LVEF 30%), bi-atrial dilatation, LV dilated, moderate MR and TR (Figure 1). So, patient was transferred to CCU for acute dilated cardiomyopathy secondary to graves storm. She was started on Lasix 40 mg IV then switched to 20 mg PO twice

a day, Metoprolol 25 mg twice a day and Digoxin 0.125 mg daily and Apixaban 5 mg twice daily. Ophthalmology consulted for proptosis who recommended artificial tears, ocular lubricant and decompression.



Figure 1: Chest X-ray showed cardiomegaly. Echocardiogram showed severe left ventricular dysfunction (LVEF 30%), bi-atrial dilatation, LV dilated, moderate MR and TR

Discussion

Thyrotoxicosis is a common disease. The prevalence is 2% in women and 0.2% in men. The incidence of thyrotoxicosis was 0.8:1000 in women and 0.6:1000 in men over the 20 years follow up ⁽¹⁾. However, only 6% of hyperthyroid patients have heart failure ⁽²⁾. About half of them have high output heart failure with normal ventricular ejection fractions and associated with coexisting atrial fibrillation ⁽²⁾.

manifestations The main of thyrotoxic cardiomyopathy are heart rhythm disturbances, usually atrial fibrillation⁽³⁾, left ventricular hypertrophy (4), pulmonary hypertension and diastolic dysfunction (5), dilation of the heart chambers and heart failure (6). Reports show 1% occurrence of dilated thyrotoxic cardiomyopathy in patients with thyrotoxicosis, and one thirds of cases is irreversible (6). Thyrotoxicosis accounts for 1% of dilated thyrotoxic cardiomyopathy (7). Dilated thyrotoxic cardiomyopathyis usually presented with an increase in heart rate, left ventricular stroke volume, ejection fraction, blood

volume. and cardiac output. Peripheral vasodilatation occurs as a result of increased metabolic end products, utilization of oxygen, and relaxation of arterial smooth muscle cell by thyroid hormones (8). This effect results in a systemic vascular resistance reduction by a 50-60%. A selective increase in blood flow to certain organs such as skeletal muscles and heart, and a decrease in diastolic blood pressure with widening of the pulse pressure ⁽⁹⁾. Lack of increase in renal blood flow cause renal perfusion pressure reduction and renin-angiotensin system activation, which cause increase inblood volume and sodium reabsorption (10).

The most common presentation of thyrotoxic cardiomyopathy are palpitations, irregular heartbeats, dyspnea, and chest pain. Tachycardia heart rhythm disturbances, systolic hypertension, heart enlargement, orthostatic hypotension, systolic murmur maximal over mitral valve and rales in case of heart failure are common findings in physical examination. Tachycardia during sleep and at rest,

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significant increase of heart rate with minimal exertion are typical for thyrotoxicosis. Patients with dilated thyroid cardiomyopathy reveals cardiomegaly and pulmonary congestion (11).

ECG changes include high, sharp P and T waves, atrial fibrillation, and extrasystole. ST segment changes might be present without angina pectoris, and due to metabolic changes. ECG changes are reversible upon restoration of a euthyroid state ⁽¹²⁾. The typical radiological signs are dilation of both ventricles and conus pulmonalis expansion in severe thyrotoxicosis. Echocardiography finds left ventricular hypertrophy, increased end-diastolic volume. *N* dilated thyrotoxic cardiomyopathy, dilation of all heart chambers and decrease in left ventricular systolic function of can be found.

The main predictors of heart failure in thyrotoxicosis are free T3 level before treatment, systolic blood pressure, and dilation of left ventricle and to a lesser extent of other heart chambers (13). Persistent euthyroid state is the most important management of thyrotoxic cardiomyopathy. However, long duration and older age of manifestation are associated with worse prognosis.The initial managementis volume overload reduction with loop diuretics. Bblockersare used for thyrotoxic symptoms alleviation (14) and amiodarone is used in atrial fibrillation treatment (15).

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