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Case Report,

Isolated Right Heart Failure and Tricuspid Regurgitation in a Patient with Untreated Hyperthyroidism: A Case Report

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Abstract:

Introduction: Hyperthyroidism has been known to cause a variety of cardiovascular manifestations. Isolated right heart failure (RHF) occurs occasionally, is usually due to pulmonary hypertension or tricuspid valve abnormalities. In rare cases, hyperthyroidism could be the underlying disease.
Case Presentation: A 72-year-old woman with suspected but untreated hyperthyroidism presented with progressive dyspnea and lower extremity swelling in the last ten days. Physical examination showed an irregular and high heart rate, increase in JVP, enlargement of the thyroid gland, systolic murmur, and clear lungs. The laboratory findings showed an elevated level of free T4 (2.51 ng/dL) and a low level of TSH (<0.003 uIU/mL). Electrocardiogram revealed atrial fibrillation with a rapid ventricular response. Echocardiography showed right atrial and right ventricular dilatation with moderate tricuspid regurgitation. Left ventricular size and systolic function were normal. Chest x-ray showed a cardiothoracic ratio of 53% with organized left pleural effusion. Symptoms resolved as her thyroid hormone levels normalized with adequate treatment.
Discussion: The most common changes that result from hyperthyroidism to the cardiovascular system are increased cardiac preload, decreased peripheral vascular resistance, direct injury, increased heart rate and contractility, which together produce a hyper dynamic circulatory state that leads to increased blood volume and venous return resulting in the increased risk of RHF.
Conclusion: Hyperthyroidism is a potentially reversible cause of heart failure and should be ruled out in every heart failure patient, especially in those with isolated right heart failure, tricuspid regurgitation, and pulmonary hypertension. These conditions can potentially be well managed with adequate treatment.

Keywords: Hyperthyroidism, Right Heart Failure, Tricuspid Regurgitation, Pulmonary Hypertension

Introduction:

Hyperthyroidism is a condition of thyrotoxicosis (clinical manifestations due to an increase in the amount of thyroid hormone in circulation) caused by hyper function of the thyroid gland.[1]Hyperthyroidism has been known to cause a variety of cardiovascular manifestations of which palpitations and tachycardia are the most common manifestations.[2] However, heart failure is a rare manifestation of hyperthyroid in previously healthy patients.[2,3]Several theories have been proposed regarding the mechanism causing cardiovascular disorders in hyperthyroidism, such as producing changes in cardiac contractility, myocardial oxygen consumption, cardiac output, blood pressure, and systemic vascular resistance (SVR).[4,5]

Heart failure is one of the most common causes of morbidity and mortality worldwide. Several studies have shown that hyperthyroidism is a potentially reversible cause of heart failure and should be ruled out in every heart failure patient,

especially in the absence of common causes of heart failure and structural heart disease.[7] Right heart failure (RHF) is often accompanied by severe left heart failure. Isolated RHF occurs occasionally, and the common causes are pulmonary hypertension (PH), tricuspid stenos is or regurgitation, and constrictive pericarditis. In rare cases, hyperthyroidism may present as isolated RHF.[7] Here we report a case of RHF with moderate tricuspid regurgitation (TR) which occurred in a patient with untreated hyperthyroidism.

Case Presentation:

A 72-year-old Balinese woman visited our hospital due to a limp a few days ago. She also complained of swelling of both legs in the last ten days ago. She started complaining of shortness of breath which has gotten worse since two years ago and currently has to sleep using two pillows. She had a weight loss of 10 kg over the recent several months. Two years ago, she was suspected of having hyperthyroidism and was advised to check thyroid hormone levels because there was an enlargement of the neck. However, the patient refused due to cost reasons. She reported no fever, cough, hemoptysis, chest pain, did not smoke or drink alcohol, and had not travelled in the recent past.

At presentation, her pulse rate was 160 beats/min irregular, body temperature was 37.2^0C, blood pressure was 120/80mmHg, and peripheral oxygen saturation was 96% in room air. On physical examination, there was an increase in jugular veins pressure, diffuse enlargement of the thyroid gland with no palpable nodules. Cardiac auscultation showed a systolic murmur at the left lower sternal border, and pulmonary auscultation revealed normal results. The patient had bilateral lower limb edema.

The laboratory findings showed an elevated level of free T4 (2.51 ng/dL), a low level of TSH (<0.003uIU/mL), and mildly elevated serum creatinine results (1.38 mg/dL). SARS-CoV-2 rapid test antigen was negative. The electrocardiogram revealed atrial fibrillation (AF) with a rapid ventricular response (RVR) (**Figure.1**), and the chest x-ray showed a cardiothoracic ratio (CTR) of 53% with organized left pleural effusion (**Figure.2**). Echocardiography was also performed and revealed right atrial (RA) and right ventricular (RV) dilatation with moderate tricuspid regurgitation (TR) high probability of pulmonary hypertension (PH), **tricuspid annular plane systolic excursion (TAPSE)** 13 mm, cardiac output (CO) 3.3 L/min and ejection fraction (EF) 74%.Left ventricular (LV) size and systolic function were normal, and there were no abnormal left-sided valve findings. There is no evidence of an abnormal shunt.

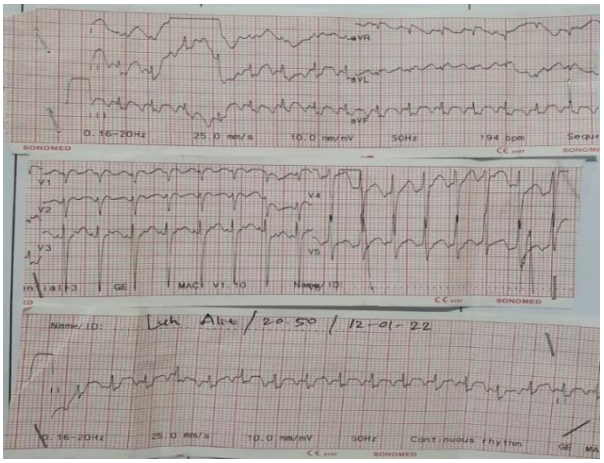

Figure 1: Electrocardiogram revealed atrial fibrillation (AF) with a rapid ventricular response (RVR)

Based on these findings the patient was diagnosed with RHF and moderate tricuspid regurgitation due to hyperthyroid accompanied with AF RVR. Treatment was begun with furosemide (20mg/twice/day), spironolactone (25mg/day), digoxin (0.25mg/day), propranolol (10mg/3times/day), thiamazole (10mg/3 times/day) and warfarin (2mg/day) because older age has a higher risk factor for embolic phenomena. The patient was referred to the endocrinology and/or cardiology department of Regional Hospital for further treatment and evaluation.

We have tried to follow up patient's condition during treatment at the referral hospital. Although the patient was hypotensive at the beginning of the treatment there, eight days after she was started on thiamazole, thyroid function tests revealed a normal free T4 level (1.18 ng/dL), and her condition gradually improved. Symptoms resolved with adequate treatment, and she was discharged after being hospitalized for 10 days.

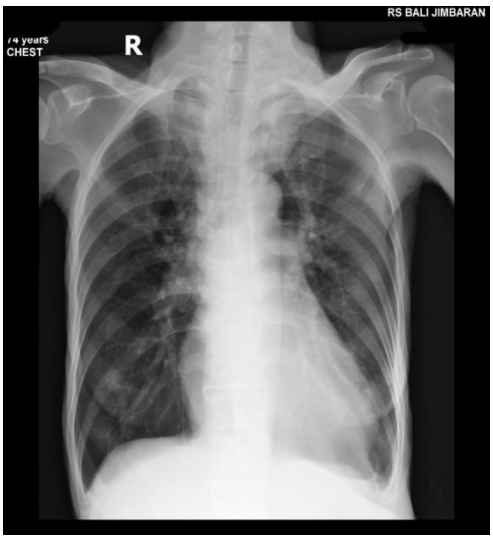

Figure 2. Chest X-Ray showed a cardiothoracic ratio (CTR) of 53% with organized left pleural effusion

Discussion:

Knowing how thyroid hormone induces heart failure. Is important for both endocrinologists and cardiologists. Patients with overt or subclinical hyperthyroidism are at increased risk of heart failure. The most common changes that result from hyperthyroidism to the cardiovascular system are an increase in cardiac preload, a decrease in peripheral vascular resistance, an increase in heart rate and heart contractility, which together produce a hyper dynamic circulatory state.[8,9] Cardiac preload is increased because of the increase in blood volume and the improvement in diastolic relaxation. The increase in diastolic relaxation in the presence of Triiodothyronine (T3); the biologically active thyroid hormone, is due to down regulation of phospholamban with increased phosphorylation and up regulation of important cardiac structural and functional proteins, namely, sarcoplasmic reticulum Ca^{2+}-ATP ase (SERCA2).[4,5,9] In addition, T3 promotes the relaxation of peripheral blood vessels. It decreases systemic vascular resistance indirectly by influencing tissue thermo genesis and directly by acting on vascular smooth muscle cells and endothelial nitric oxide production. The decrease in the vascular resistance of the system is responsible for the decrease in renal perfusion pressure and the activation of the renin-angiotensin-aldosterone system (RAS), with a consequent increase in sodium absorption and blood volume.[9] Therefore, the hyperthyroid enhances its performance by modulating the hemodynamic load.

The increased circulating dynamics in hyperthyroid patients leads to increased blood volume and more rapid venous return to the right atrium. As a result, the pulmonary artery and right ventricular pressures increase, and the right ventricle become dilated. This can result in functional TR.[10]However,the impact of hyperthyroid on cardiac valves through the combined of a direct effect on the connective tissue of tricuspid valves (myxomatous degeneration)leading to primary valvular insufficiency, and hemodynamic volume overload leading to cardiac chamber dilatation and further valve incompetence.[11]

Despite the direct effects of thyroid hormone, high output-induced endothelial injury and chronic tachycardia have been proposed as mechanisms of heart failure in patients with hyperthyroidism, the mechanism by which hyperthyroidism causes predominant or isolated RHF and TR has not been fully explained. Several studies reveal possible mechanisms of RHF in thyrotoxicosis are a combination of factors, namely, pressure overload of PH plus volume overload with secondary TR development and possibly direct toxic effect of excess circulating thyroid hormone causing a *stunned myocardium* form which mainly involves the right ventricle.[7,12]However, Ji Yeon Hong *et al*[13] showed that RV dysfunction in thyrotoxicosis is not related to secondary hemodynamic change by LV systolic dysfunction, which suggests there may be a direct toxic effect of thyroid hormone on the right ventricle. In addition, thyroid auto antibodies secreted in Graves' disease, the most common cause of hyperthyroidism, can injure the pulmonary endothelium; further contributing to pulmonary hypertension.[2] further study is needed to find out more about the path physiology of increased thyroid hormone on its effects, especially on the right heart chamber.

A few case series in the past have shown hyperthyroidism is manifested first by right heart failure with pulmonary artery hypertension and tricuspid regurgitation. Most of them show that adequate treatment of hyperthyroidism can reduce cardiac output and pulmonary hypertension and subsequently result in the improvement of heart failure. Sun Ho Hwang *et al*[2] showed completely resolved right heart failure and severe TR in 60 years-old man patients with adequate treatment of

hyperthyroidism. Joseph P. Frolkis *et al*[3]also described the case of 2 middle-aged women who had RHF with TR and PH that ended well after receiving adequate therapy for their hyperthyroidism accompanied with propranolol and furosemide. In our case, the patient experienced an improvement in her symptoms as her thyroid hormone levels normalized with adequate treatment. This suggests that hyperthyroidism is a potentially reversible cause of heart failure.

Conclusion:

Hyperthyroidism has many systemic effects on various organ systems. The most common changes that result from hyperthyroidism to the cardiovascular system are an increase in cardiac preload, a decrease in peripheral vascular resistance, an increase in heart rate and heart contractility resulting in heart failure. Therefore, clinicians should be aware of the possibility of hyperthyroidism as a cause of heart failure and thyroid function tests as a useful diagnostic tool in patients with heart failure, especially in those with right heart failure, tricuspid regurgitation, and pulmonary hypertension. The conditions are potentially reversible with adequate treatment.

Conflict of Interest:
None declared

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