Graves' Disease Presenting as Right Heart Failure

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The differential diagnosis of right heart failure is wide and usually does not include thyrotoxicosis. The most common cardiovascular manifestations of thyrotoxicosis are sinus tachycardia, supraventricular arrhythmia, left heart failure and angina pectoris. We describe two patients in whom right heart failure was the presenting sign of thyrotoxicosis.

Patient Descriptions

Patient 1

A 46 year old woman was admitted to the hospital because of heart palpitations and leg edema that appeared 3 days before her admission. Her past history was unremarkable. Physical examination revealed an anxious and agitated woman with a diffusely enlarged thyroid gland, and a jugular venous distension of 10 cm when the head of the bed was elevated to about 30° from horizontal. Heart sounds were irregular with a systolic murmur at the apex and at the tricuspidal point. Examination of the lungs and abdomen was normal without signs of hepatomegaly. Moderate (+2) pitting edema was detected on the legs with no signs of deep vein thrombosis. Blood pressure was 109/69 mmHg. Pulse was 132/minutes and irregular. Oxygen saturation was 98% while the patient was breathing ambient air.

The laboratory results revealed normocytic normochromic anemia with a hemoglobin level of 11.4 g/dl. Serum alkaline phosphatase was elevated to 278 U/L (normal 30–120 U/L). Thyroid function tests confirmed a state of thyrotoxicosis with thyroid-stimulating hormone 0.005 mU/L (normal 0.23–4.0 mU/L), free thyroxine > 77.2 pml/L (normal 5–12 pml/L) and total triiodothyronine > 9.24 nml/L (normal 1.1–2.9 nml/L). The rest of the laboratory results were within normal range.

Electrocardiogram showed rapid atrial fibrillation at a ventricular rate of 132 beats per minute. Echocardiography revealed a normal sized and functioning left ventricle, moderate to severe tricuspid regurgitation without deformation of the leaflets, and pulmonary pressure reaching 35 mmHg. There were no signs of right ventricular enlargement or reduced function. Ultrasound Doppler of the legs did not demonstrate deep vein thrombosis. The lung function tests were interpreted as normal. Lung ventilation-perfusion scan and computerized tomography of the chest with intravenous contrast did not detect pulmonary emboli.

A Tc-99 thyroid scan confirmed a diffused enhanced uptake, which is compatible with Graves' disease. Propyltiouracil treatment was initiated at a dose of 150 mg 3 times/day as well as lugol solution, corticosteroids and beta-blockers. Several months later radioactive iodine treatment was administered, resulting in complete recovery from thyrotoxicosis. No signs of right heart failure were found during a 1 year follow-up and the patient remained in sinus rhythm without anti-arrhythmic therapy.

Patient 2

A 44 year old woman was admitted because of palpitations and leg edema that appeared 3 days before her admission, associated with fatigue and weakness on mild exertion during the preceding month. On physical examination she appeared well with anxiety symptoms. She had an increased maximal jugular venous pressure and exophthalmus. Her non-sensitive thyroid gland was diffusely enlarged. There was dullness at the base of the right lung and decreased breath sounds. Heart sounds were irregular without any murmur. There were no signs of hepatomegaly. Pitting edema (+2) was found on her legs with no signs of deep vein thrombosis. An ECG showed a rapid atrial fibrillation at a ventricular rate of 120 beats/minute. The laboratory results revealed a normochromic normocytic anemia with hemoglobin 11 g/dl. The level of alkaline phosphatase was 202 IU/L. The rest of the laboratory results were within normal limits.

The chest X-rays showed bilateral pleural effusion that was greater on the right. Diagnostic pleurocentesis demonstrated transudate. Echocardiography revealed normal ejection fraction, mild mitral and tricuspid regurgitations, and elevated pulmonary pressure reaching 55–60 mmHg. There was no evidence on lung perfusion-ventilation scan test or angio-CT for pulmonary emboli.

Thyroid gland function test demonstrated hyperthyroidism with TSH 0.003 mUI/L and FT4 49.5 pml/L. High titer of antithyroid-stimulating antibodies confirmed the diagnosis of Graves' disease. Propylthiouracil treatment was initiated at a dosage of 150 mg 3 times/day in combination with corticosteroids and beta-blockers.

During the 8 months of follow-up the thyroid tests returned to normal and the signs of right heart failure significantly diminished. Repeated echocardiography showed normal pulmonary artery pressure (30 mmHg) with normal left and right

TSH = thyroid-stimulating hormone
FT4 = free thyroxine

ventricular size and function. There was no evidence of pleural effusion on repeated chest X-rays.

Comment

Thyrotoxicosis has been associated with several cardiac complications, including atrial fibrillation, functional cardiomyopathy and congestive heart failure. Right heart failure is a rare manifestation of thyrotoxicosis. The literature contains only a few case reports depicting right heart failure and hyperthyroidism [1].

Studies in rats referred to isolated right ventricular hypertrophy associated with experimental hyperthyroidism [2]. In addition, several clinical studies showed a significant increase in right ventricular pressure and elevated pulmonary pressure in patients with hyperthyroidism. These findings were explained by increased cardiac output and venous return that are present in hyperthyroidism [3].

We report two cases of Graves' disease clinically associated with right heart failure. The first patient presented with hyperthyroidism and right heart failure due to moderate to severe tricuspid regurgitation. Valvular involvement in patients with Graves'-induced cardiomyopathy is extremely uncommon. The incidence of mitral regurgitation, tricuspid regurgitation, and a combination of the two, was significantly higher in the patients with Graves' disease than in an age-matched control group of patients without the disease. In hyperthyroid patients with signs of congestive heart failure, a significantly higher incidence of severe tricuspid regurgitation was reported [4]. The second patient presented with hyperthyroidism complicated by elevated pulmonary pressure. The possible mechanisms of pulmonary hypertension induced by hyperthyroidism entail pulmonary vascular endothelial dysfunction or damage due to the autoimmune process, the high cardiac output state, or the increased metabolism of intrinsic pulmonary vasodilators [5]. In both patients the signs of right heart failure diminished significantly after resolution of the hyperthyroid state.

This report provides further support to the potential link between hyperthyroidism and right heart failure. It is recommended that all patients with a diagnosis of idiopathic heart failure be examined for thyroid function in order to identify hyperthyroid subjects with a reversible myocardial dysfunction.

References

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