Case report

Severe tricuspid regurgitation and isolated right heart failure due to thyrotoxicosis

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ABSTRACT

We describe the case of a patient presented with isolated right heart failure with atrial fibrillation and severe tricuspid regurgitation due to hyperthyroidism. Treatment of the thyroid disease resulted in the disappearance of signs of right heart failure and resolution of the valve incompetence and normalization of the heart rhythm. Although thyrotoxicosis may be associated with congestive heart failure, isolated right heart failure with marked tricuspid regurgitation is rarely seen.

1. Introduction

Cardiovascular manifestations of hyperthyroidism are diverse and frequent. Usually a hyperdynamic circulatory state hallmarks the disease with low peripheral resistance, increased circulatory volume and increased cardiac output. Symptoms of congestive heart failure (CHF) with normal right heart cavities represent the initial clinical presentation in approximately 6% of patients with overt hyperthyroidism and symptoms most often subside following treatment of the thyroid disease.

Overt hyperthyroidism as a cause of reversible pulmonary hypertension, which is usually mild or moderate, is also reported in the literature. These findings might be explained by the fact that thyroid hormone lowers peripheral resistance thereby increasing cardiac output and pulmonary flow.

Few case reports however, describe isolated right heart failure, severe right ventricle overload and tricuspid regurgitation (TR) as a consequence of hyperthyroidism.

We present the case of a young woman with thyrotoxicosis, right heart failure, marked TR, mild pulmonary hypertension and no signs of left ventricular dysfunction, which resolved slowly after adequate antithyroid therapy.

2. Case presentation

A 34-year-old woman was admitted to our hospital with a 3-months history of progressive shortness of breath with palpitations and diaphoresis. Clinical evaluation revealed jugular venous distention, hepatomegaly, ascites, bilateral ankle edema, a marked right ventricular heave and...
a holosystolic murmur at the left sternal border accentuated with inspiration.

Her blood pressure was 90/50 mmHg while oxygen saturation was decreased, 89% on room air. A chest X-ray showed cardiomegaly and resting ECG atrial fibrillation with rapid ventricular response (average 140 beats/min).

On transthoracic echocardiography the most striking finding was a dilated hyperdynamic right ventricle with incomplete systolic coaptation of the tricuspid valve leaflets resulting in severe TR (Fig. 1A). The right atrium was also dilated but left ventricular size and systolic function were normal. The estimated pulmonary artery systolic pressure was mildly elevated at 45 mmHg.

Blood tests revealed depressed TSH and elevated levels of free T3, free T4 consistent with severe hyperthyroidism. D-dimers were within normal limits and a spiral computed tomography excluded pulmonary embolism. Carcinoid syndrome and tricuspid endocarditis were also excluded. A transesophageal echocardiographic study was negative for the existence of an atrial septal defect.

The only concurrent illness identified was thyrotoxicosis. The patient was treated with unimazole, propranolol, furosemide and spironolactone. Within the first month the patient became euthyroid and rate control was achieved. However, after 5 months the clinical signs of right-sided heart failure and atrial fibrillation persisted and the patient still complained of shortness of breath and fatigue. A new echocardiography did not show much of improvement, severe TR and dilated right heart chambers persisted, only pulmonary artery systolic pressure had been normalized.

On subsequent follow-up 10 months after the initial evaluation the patient remained euthyroid, the cardiac rhythm had converted spontaneously from atrial fibrillation to sinus rhythm and her symptoms were finally resolved. Repeat cardiac ultrasound demonstrated normal right cavities and trivial TR (Fig. 1B).

3. Discussion

Hyperthyroidism may present with cardiovascular signs and symptoms such as palpitations, exercise intolerance, exertional dyspnea, systolic hypertension, hyperdynamic precordium, anginal chest pain, atrial fibrillation, peripheral edema and CHF.1

In a recent study2 patients with symptoms of CHF at presentation due to thyrotoxicosis had no evidence of significant valvular lesion, pericardial disease or hypertrophic or infiltrative cardiomyopathy and only 47% had an ejection fraction ≤50%. Approximately one-third of patients with left ventricular systolic dysfunction developed persistent dilated cardiomyopathy with partial recovery despite successful rate or rhythm control of tachyarrhythmias and atrial fibrillation and hyperthyroidism treatment. This suggests that a variety of mechanisms may contribute to the development of heart failure in this entity.

Thus, CHF without structural right heart disease may be related with congestive circulation secondary to sodium and fluid retention, diastolic dysfunction and/or tachyarrhythmia induced cardiomyopathy.1

In the present case, thyrotoxicosis was associated with severe TR and right ventricular heart failure, which was resolved a long period after the patient became euthyroid.

Primary TR can be due to several causes such as tricuspid valve prolapse, carcinoid syndrome, chest wall trauma and tricuspid valve endocarditis. Hyperthyroidism can be a rare cause of isolated tricuspid regurgitation and right-sided heart failure.

One of the possible postulations is that increased heart rate and increased right ventricular load, because of high cardiac output and rapid venous return dilate the thin-walled right ventricle, which is susceptible to volume overload.3,4 TR can occur as a result of the dilation of the right ventricle and the tricuspid annulus. Whether right heart failure is secondary to the severe TR or the other way round remains unclear.

Pulmonary hypertension in hyperthyroidism is probably caused by high cardiac output, endothelial damage/dysfunction, or increased metabolism of intrinsic pulmonary vasodilating substances.5

Despite that, right ventricular failure and TR might be directly related to thyrotoxicosis independently from the existence of significant pulmonary hypertension, as in our patient.

In conclusion, severe TR and isolated right heart failure can be associated with thyrotoxicosis, thus thyroid disease should be considered in the differential diagnosis. To the best of our knowledge, there are only a few cases reported in the

Fig. 1 – A. Transthoracic apical four chamber view demonstrating dilated right heart cavities with severe tricuspid regurgitation. B. Transthoracic apical four chamber view demonstrating normal right heart cavities with trivial tricuspid regurgitation.
literature, with severe TR, isolated right heart failure and mild pulmonary hypertension due to hyperthyroidism.

**Conflicts of interest**

All authors have none to declare.

**REFERENCES**


