

Case Report

Thyrotoxic Heart Failure

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Kuwait Medical Journal 2004, 36 (4):285-286

ABSTRACT

Most patients with congestive heart failure have systolic dysfunction with a low cardiac output and elevated systemic vascular resistance. A good percentage of patients have diastolic dysfunction, in which an increase in ventricular stiffness impairs filling during diastole. In

rare circumstances, the cardiac output is elevated and calculated systemic vascular resistance is very low. We report a case of thyrotoxicosis who presented with congestive heart failure, normal systolic function and cardiac output.

KEYWORDS: atrial fibrillation, heart failure, thyrotoxicosis

INTRODUCTION

Heart failure is a major cause of morbidity and mortality. In the United States, the annual mortality rate is high, ranging from 10-15% for patients with mild to moderate heart failure and 50% for patients with severe heart failure^[1]. Thyrotoxicosis can not only aggravate pre-existing heart disease but also lead to heart failure in a person with unknown existing heart disease^[2].

CASE REPORT

A 66-year-old man was admitted to our hospital with two weeks history of shortness of breath, wheezy chest, productive cough of whitish sputum, paroxysmal nocturnal dyspnea, and orthopnea. He had progressive weight loss for the last five years. He denied any history of palpitation, chest pain, heat intolerance, sweating, or change in bowel habit. He was not diabetic nor hypertensive, but a heavy smoker for more than 20 years.

On physical examination, he was dyspneic, anxious and tremulous. His heart rate was 140/minute in atrial fibrillation, BP 160/70 mmHg, temperature 37° celsius, and respiratory rate 28 breaths/minute. He had staring look, but no lid lag and no exophthalmos. He had no goiter. His jugular venous pressure was 6 cm above the sternal angle. His heart examination was normal apart from atrial fibrillation. His chest was wheezy, and he had basal crackles. He had tender hepatomegaly, but no lower limb edema. Electrocardiogram showed atrial fibrillation and a ventricular rate of 140 beats/minute, with left ventricular hypertrophy. There were no ischemic changes. Chest X-ray showed right sided pleural effusion and pulmonary congestion. His white cell count was $5.4 \times 10^9/L$, with a haemoglobin of 118 gram/L. Arterial oxygen tension was 10.24 Kpa, and arterial

carbon dioxide tension was 4.73 Kpa. Cardiac enzymes as well as liver and renal function were normal. Total cholesterol was 2.9 mmol, HDL 0.9 mmol, LDL 1.8 mmol, and triglycerides 0.68 mmol. Echocardiogram showed left ventricular hypertrophy with normal systolic function and an ejection fraction of 60%. Thyroxine was 315 nmol/L (normal 49 to 141), and thyroid stimulating hormone was <0.005 miu/L (normal 0.27 to 4.2). Thyroid scan and uptake showed diffuse toxic goiter with I-131 uptake of 57% (normal <30%).

He was diagnosed as thyrotoxic heart failure and was started on diuretics, heparin, and digoxin. On the next day, digoxin was replaced by metoprolol 50 mg twice daily. He tolerated the B-blocker very well and his heart rate dropped to 80 beats/minute, with disappearance of the heart failure. After the thyroid result was obtained, he was started on neomercazole and continued on warfarin. One month later he was clinically and chemically euthyroid, and out of heart failure. Neomercazole was stopped for seven days and radioactive iodine was given. Two months later his rhythm had returned to sinus. He was started on thyroxine and all other medications were stopped. Repeated echocardiogram after six months revealed resolution of left ventricular hypertrophy, with an ejection fraction of 60%.

DISCUSSION

High-output heart failure is characterized by an elevated cardiac index. Ineffective blood volume and pressure, chronic activation of the sympathetic nervous system and renin-angiotensin-aldosterone axis, increased serum vasopressin concentration and chronic volume overload gradually cause ventricular enlargement, remodeling and heart failure.

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Table 1
Cardiovascular hemodynamics in thyrotoxicosis

Parameter	Change	Comments
Systemic vascular resistance	Decreased	50-70% lower, similar to exercise
Cardiac output	Increased	200-300% increase
Blood pressure		Especially in elderly patients
Systolic	Increased	
Diastolic	Decreased	
Heart rate	Increased	Most often sinus tachycardia: 5% to 10% of patients have atrial fibrillation
Cardiac contractility	Increased	Systolic and diastolic function both increased
Cardiac mass	Increased	Hypertrophy from increased cardiac work
Blood volume	Increased	Increased serum erythropoietin and sodium reabsorption

The physiologic causes of high cardiac output include excitement, exercise, pregnancy, and fever. The pathologic causes of high output heart failure include systemic arteriovenous fistula, thyrotoxicosis, anemia, beriberi, dermatologic disorders such as psoriasis, renal disorders such as chronic renal failure, hepatic disease such as cirrhosis, skeletal disorders such as Paget's disease, and hyperkinetic heart syndrome.

Thyroid hormone has many effects on the heart and vascular system. These changes are the result of both regulation of cardiac-specific gene and changes in hemodynamic function induced by triiodothyronine (T_3)^[3,4]. Calcium release from and reuptake into sarcoplasmic reticulum of the cardiac muscle regulates the rate of ventricular systolic contractile function and diastolic relaxation^[4]. The gene for cardiac-specific-calcium ATPase that regulates the sequestration of calcium during diastole is activated by thyroid hormone. In addition, the expression of the protein phospholamban, which is a negative regulator of calcium uptake by the sarcoplasmic reticulum, is inhibited by thyroid hormones. Taken together, this may explain the increased rate of development of systolic tension and diastolic relaxation in the heart of patients with thyrotoxicosis.

The cardiovascular hemodynamics that accompany thyrotoxicosis are summarised in Table 1. There are decreases in systemic vascular resistance and increases in cardiac output, systolic blood pressure, left ventricular ejection fraction, cardiac contractility and mass, and blood volume^[2-4]. Increases in resting heart rate are characteristic of thyrotoxicosis. More than 90% of patients have resting tachycardia, and many have heart rates faster than 120 beats per minute. Sinus tachycardia is the commonest rhythm disturbance in thyrotoxicosis^[2-4]. However, its clinical importance is overshadowed by the challenges posed by atrial fibrillation, which occurs in 5-15% of patients with thyrotoxicosis^[2]. Thyrotoxicosis increases sinoatrial-node firing, decreases the electrical threshold for

atrial excitation, and shortens the refractory period of the conducting tissues^[2].

In a large series of patients with hyperthyroidism, congestive heart failure was noted in 6% of patients^[5,6]. Most patients are elderly and probably have underlying heart disease. In others, it is a complication of atrial fibrillation or prolonged marked sinus tachycardia, and resolves when the ventricular rate is slowed or sinus rhythm is restored. Congestive heart failure in the absence of cardiac disease or arrhythmia is thought to reflect hyperthyroid cardiomyopathy, which disappears when hyperthyroidism is treated. There is no clear histopathologic correlation with this cardiomyopathy, which disappears when thyrotoxicosis is treated^[2].

Our patient's heart failure was probably secondary to the presence of atrial fibrillation and left ventricular hypertrophy, both of which were induced by actions of excessive thyroid hormones. Although he did not undergo stress testing, we had no reason to suspect the presence of occult heart disease. His failure resolved with the correction of his rhythm and the left ventricular hypertrophy with the treatment of thyrotoxicosis.

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