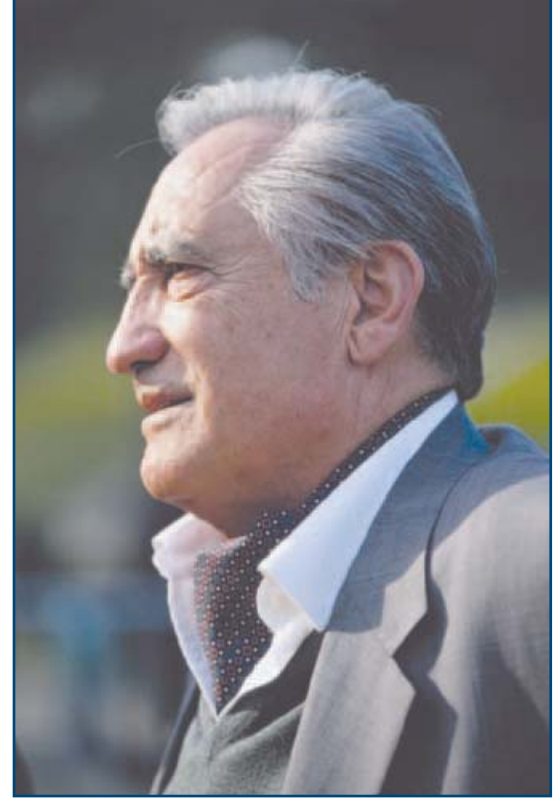


CardioCase

of the Month

A Case of Hyperthyroid Cardiomyopathy



By Miroslaw Rajda, MD, FRCPC; Peter Hooley; and Jonathan Howlett, MD, FRCPC

CardioCase Presentation

A 53-year-old single man is brought by ambulance to the emergency department. He presents with severe shortness of breath, chest tightness, and palpitations. According to his neighbour, he had flu-like symptoms for four to five weeks, with productive cough and shortness of breath. He complained he has “not been himself” for the last six months.

Vital signs:

- Temperature: 38.5 C
- Heart rate: around 160 to 180 beats per minute
- Blood pressure: 204/90 mmHg
- Respiratory rate: 40
- Oxygen saturation on room air: 70%

He was subsequently intubated and ventilated.

About the authors ...

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What's Your CardioCase Diagnosis?

CardioCase Discussion

What did the exams show?

A chest examination revealed decreased breath sounds, expiratory wheezes, and inspiratory crackles, particularly at the left base. The patient's pulse was bounding with increased volume and the jugular venous pressure was distended. S_1 was variable, S_2 normal, and S_3 present at the apex. There was a pansystolic murmur at the apex grade II/VI radiating to the axilla. The apex was displaced to the left and enlarged. Mild peripheral edema was present. A neck examination revealed a prominent thyroid gland. The abdomen was soft and non-tender.

The ECG showed rapid atrial fibrillation (AF) with a ventricular rate of 160 to 180 beats per minute (bpm) (Figure 1).

A chest X-ray showed cardiomegaly, changes of pulmonary edema, and possible left lower lobe pneumonia.

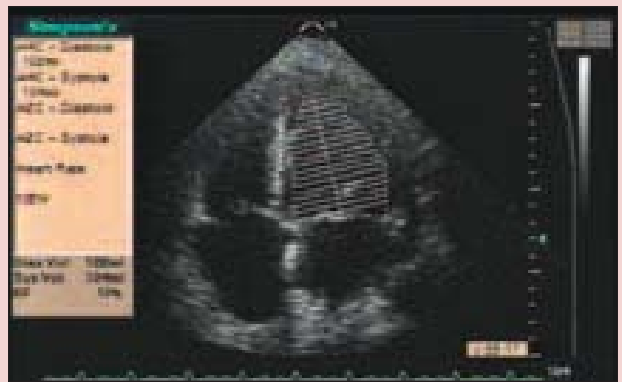


Figure 2.

A transthoracic echocardiogram revealed severe systolic left ventricular (LV) dysfunction with an ejection fraction of approximately 25% (Figure 2).

The left ventricle was dilated with a 3+ mitral regurgitation (MR). The right ventricle was dilated and hypokinetic with a 2+ tricuspid regurgitation. The right ventricle systolic pressure was calculated at 25 mmHg.

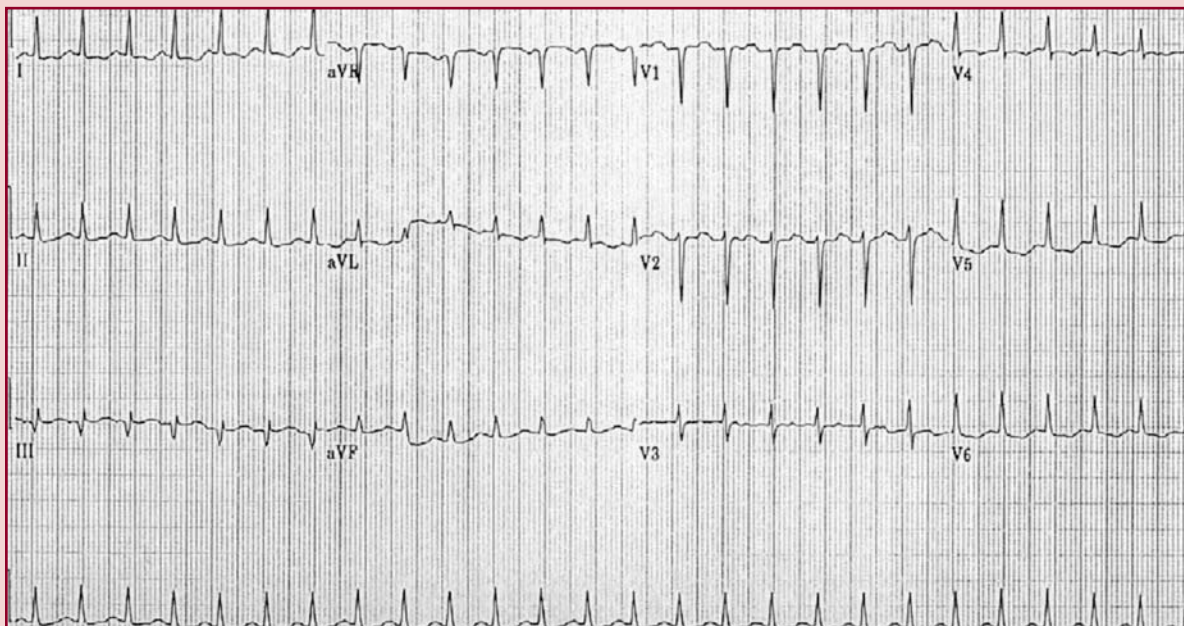


Figure 1.

CardioCase Discussion

The thyroid-stimulating hormone (TSH) was 0.01 mIU/L (N 0.17-4.2) and the free T4 was measured at 60.3 pmol/L (N 13-23). A diagnosis of thyrotoxic cardiomyopathy due to Graves' disease was made. Aggressive anti-failure therapy was initiated with an angiotensin converting enzyme (ACE) inhibitor, a beta blocker, diuretics, and nitrates. Hyperthyroidism was treated with Lugol's iodine and propylthiouracil, with a dramatic improvement over the next several days.

The patient was discharged from the hospital in stable condition in functional class II. Repeated ECG two weeks later showed almost normal LV function, an MR of 2+, and a TR of 1-2+.

roidism results in a decrease in systemic vascular resistance (SVR) by an average of 50% to 60%. The fall in SVR plays a central role in the alteration of hemodynamics due to high thyroid hormone levels. The lack of a rise in renal blood flow causes a decrease in renal perfusion and an activation of the renin-angiotensin system leading to increased sodium reabsorption and blood volume.³

Table 1

Alterations in cardiovascular hemodynamics in hyperthyroidism

Increases in:

- heart rate
- cardiac contractility
- myocardial oxygen consumption
- blood volume
- cardiac output
- systolic blood pressure

Reduction in:

- systemic vascular resistance
- diastolic pressure

Modified from *Heart Disease: A Textbook of Cardiovascular Medicine*. Sixth Edition. W.B. Saunders Company, U.S. 2001.

What are the clinical manifestations?

Most patients with hyperthyroidism, including subclinical forms of it, experience cardiovascular complications. The most common clinical manifestations of hyperthyroidism related

to the cardiovascular system are listed in Table 2.

What are the cardiovascular effects of hyperthyroidism?

Increased levels of thyroid hormones exert significant effects on the heart and cardiovascular system. It has been shown that clinical manifestations of hyperthyroidism are due to alterations in cardiovascular hemodynamics (Table 1). Hyperthyroidism can unmask pre-existing cardiac diseases, such as silent coronary artery disease and compensated heart failure, or can cause cardiac complications in patients with a structurally normal heart.^{1,2}

Peripheral vasodilatation in hyperthy-

Table 2

Cardiovascular manifestation of hyperthyroidism

Symptoms	Prevalence
1. palpitations	85%
a. sinus tachycardia	90%
b. atrial fibrillation	5%-15%
2. exercise intolerance	65%
3. dyspnea	50%
4. fatigue	50%
5. pedal edema	30%

Modified from *Heart Disease: A Textbook of Cardiovascular Medicine*. Sixth Edition. W.B. Saunders Company, U.S. 2001.

CardioCase Discussion

Table 3

Cardiovascular signs in hyperthyroidism

Signs	Prevalence
1. tachycardia	90%
2. bouncing pulses	75%
3. wide pulse pressure	75%
4. hyperactive precordium	75%
5. systolic murmur	50%
6. systolic hypertension	30%

Modified from *Heart Disease: A Textbook of Cardiovascular Medicine*. Sixth Edition. W.B. Saunders Company, U.S. 2001.

Are there rhythm disturbances?

The most common rhythm problem in hyperthyroidism is sinus tachycardia. Elevated heart rate of > 90 bpm occurs at rest and during sleep, and an exaggerated cardiac response is witnessed during exercise. A more frequent and clinically problematic situation occurs in patients with hyperthyroidism and AF with rapid ventricular response, which can lead to rate-related cardiomyopathy. A TSH level should be obtained in patients with new onset of AF, although only < 1% of these patients will have subclinical or clinical forms of hyperthyroidism. Most patients with hyperthyroidism and AF convert to sinus rhythm within eight to 10 weeks after initiation of treatment.

Other forms of arrhythmias are uncommon. Only 15% of patients or less manifest a delay in intraventricular conduction with right bundle branch morphology. Advanced atrioventricular blocks may occur, but are not very common.^{4,5}

Can heart failure occur?

Most patients with hyperthyroidism are in a high cardiac output state with the absence of symptomatic heart failure. In patients with pre-existing clinically active or silent heart failure, hyperthyroidism can cause acute exacerbation. However, in a small group of patients who have a structurally normal heart, hyperthyroidism can cause clinically evident systolic heart failure. This is called hyperthyroid cardiomyopathy.⁶ The underlying pathophysiology of hyperthyroid cardiomyopathy remains unclear, although, in most cases, it is associated with persistent sinus tachycardia or AF with uncontrolled ventricular response leading to rate-related heart failure.⁷

Congestive heart failure (CHF) is a clinical diagnosis and patients with hyperthyroidism who develop heart failure manifest typical symptoms and signs of CHF, such as shortness of breath, fatigue, paroxysmal nocturnal disease, and orthopnea. However, there are signs which should alert a clinician of hyperthyroidism as a possible primary cause for CHF (Table 3).

Patients with hyperthyroidism who develop heart failure manifest typical symptoms and signs for CHF.


A high index of suspicion is required to diagnose hyperthyroidism in any new onset of CHF, especially in elderly patients who may present with atypical features.⁸

What is the treatment?

Prompt recognition and effective treatment of cardiac manifestations in patients with hyperthyroidism are extremely important, as cardiovascular complications account for most deaths after treatment of hyperthyroidism.⁹ In patients with hyper-

CardioCase Discussion

thyroidism and AF, initial therapy should focus on ventricular rate control using beta blockers (propranolol, atenolol), but conversion to sinus rhythm frequently occurs spontaneously with the treatment of hyperthyroidism. Electric or pharmacologic cardioversion should be attempted only in euthyroid patients who are hemodynamically unstable. If AF persists, consideration should be given to anticoagulation, particularly in patients who are at high risk for embolic events.

In patients with hyperthyroidism and CHF, diuretics should be used to reverse fluid overload, but initial treatment should include beta blockers. Beta blockers in hyperthyroidism-related heart failure should be used early and in high doses, in contrast to typical CHF therapy, where careful optimisation of other therapies should precede beta blockers. The goal of therapy with beta blockers is to lower heart rate to a nearly normal level and to subsequently improve the tachycardia-mediated component of LV dysfunction. Routine therapy for heart failure, including ACE inhibitors, should be used in patients with known pre-existing LV dysfunction or in patients in whom heart failure does not improve when heart rate is normalised.¹⁰ 

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Take-home message

1. Hyperthyroidism should be considered in patients with a new onset heart failure associated with persistent sinus tachycardia or AF.
2. Beta blockers are a first-line treatment for AF and heart failure in patients with hyperthyroidism.
3. TSH should be routinely checked in all patients with new onset AF.

