

# Thyroid Disease and the Heart: A Common Connection

Cardiac symptoms are often the first and most prominent signs of thyroid disease. In this review, Dr. Voth discusses the connection between the heart, thyrotoxicosis and hypothyroidism.

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## Christina's Chest Pain

Christina, 42, is a previously healthy woman who presents with a four-month history of progressive:

- shortness of breath on exertion,
- weakness,
- heat intolerance and
- symptoms of bronchitis.

Several courses of antibiotics prove to be ineffective. She has developed chest pain, which has become worse over the past three weeks and is referred to Cardiology.

On admission, Christina is found to have:

- severe pulmonary edema due to chronic heart failure (CHF),
- an abnormal wall motion index on EKG and an akinetic anterior wall,
- mitral regurgitation,
- pulmonary hypertension (HT) and
- severe diastolic dysfunction on echocardiogram.

Christina's thyroid hormone, free thyroxin (FT4) is > 155 (N = 9 pmol/L to 23 pmol/L). A coronary angiogram reveals marked coronary vessel spasm, but no occlusion. She is treated with the usual coronary care, plus huge doses of propylthiouracil and  $\beta$ -blockers. Christina eventually recovers completely over many months.

**For another case, go to page 31.**

## Thyroid cardiac physiology

Christina's case illustrates everything that can happen to the heart in severe thyroid disease except, fortunately, arrhythmias.

Excess thyroid hormone assaults the heart by multiple pathways. Triiodothyronine (T3) increases cardiac output by increasing tissue thermogenesis, decreasing systemic vascular resistance and directly increasing cardiac inotropy and chronotropy. Together, these effects reinforce each other. It is therefore no surprise that cardiac symptoms are often the first and most prominent signs of thyroid disease.

## Thyrotoxicosis and the heart

Respectively, the most common presentations of thyrotoxicosis are:

- tachycardia and/or atrial fibrillation (AF),
- decreased exercise tolerance and
- presence of a goiter.

While < 1% of new onset AF is caused by thyrotoxicosis, 13% of *unexplained* new onset AF is caused by thyrotoxicosis.

The most common cardiac complication of thyrotoxicosis is AF. Other cardiac complications are relatively rare. More serious complications, such as Christina's case, most often indicate underlying heart disease (HD). Even AF,

more often than not, occurs in patients with pre-existing HD. Thyrotoxicosis often unmasks unsuspected HD; therefore the appearance of *any* cardiac complication in the thyrotoxic patient should always lead to a full cardiac assessment.

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### Treatment

$\beta$ -blockers are the cornerstone of early treatment for any thyrotoxic patient and are doubly important when there is co-existing HD.  $\beta$ -blockers should be given as soon as the diagnosis is made, in doses sufficient to control tachyarrhythmias of any kind. Usually, this will require the equivalent of 80 mg q.d. to 300 mg q.d. of propranolol in divided doses.

In mild-to-moderate HD, this would be followed by radioiodine ablation or antithyroid drugs and much less often, by thyroid surgery.

In severe HD, the treatment would include huge doses of antithyroid drugs and steroids. Radioiodine ablation would be contraindicated. Rarely, surgery might be required for rapid control of the disease.

### Hypothyroidism and the heart

Cardiac involvement in hypothyroidism is common, but mild. Bradycardia and mild diastolic hypertension are almost universal. Creatine kinase and lactate dehydrogenase (LDH) are often increased, at times dramatically and are easily misinterpreted as suggestive of a MI. (Troponin I, however, is NOT increased by hypothyroidism.) Cardiac output is almost universally decreased, but overt chronic heart failure is rare. The QT interval in an electrocardiogram is prolonged and the rate is slowed, resulting in the perfect setting for arrhythmias and indeed, they are more common, ranging from ventricular ectopy to *torsades de points*; but globally, they are still unusual.

While significant clinical changes in cardiac function are unusual, subclinical changes at the research level are common. Even the mildly hypothyroid patient has accelerated coronary artery disease and decreased myocardial efficiency and contractility; all effectively reversed by thyroxine therapy. Despite this, the treatment of the so-called subclinically hypothyroid patient remains controversial.

#### About the author...



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## Trisha's Treatment

Trisha, a 55-year-old woman with a background history of Type 2 diabetes mellitus, HT and obesity presents with symptoms of:

- tiredness,
- even more weight gain,
- fluid retention and
- mild cold intolerance.

She has recently been diagnosed with:

- mild coronary artery disease,
- mild angina and
- mild CHF for which conservative therapy was recommended.

Her thyroid stimulating hormone level is 8.0 (N = < 4.0 mU/L) and her FT4 is normal.

**Should you treat her with thyroxine?**

## Take-home message


- Think of the thyroid when there is unexplained tachycardia or atrial fibrillation
- Administer  $\beta$ -blockers early and in adequate doses to treat thyrotoxic heart disease
- Refer a patient early rather than late so as to prevent outcomes like that described in Christina's case
- Mild-to-moderate hypothyroidism, in the presence of heart disease, should be treated cautiously
- For severe hypothyroidism, begin treatment and refer. Little reliable data exists on the best treatment approach

*Thyroxine therapy should be started cautiously at 25 mcg q.d., certainly no more than 50 mcg q.d., and increased as needed to normalize the thyroid stimulating hormone.*

### *Treating Trisha*

I believe Trisha should be treated with thyroxine because this therapy will:

- improve her cardiac output levels,
- improve her mobilization of fluid,
- improve her diabetic control,
- help her to control her weight problem,
- improve her lipid profile,
- improve her control of her hypertension, especially the diastolic component and
- probably improve her angina, if given cautiously.

Thyroxine therapy should be started cautiously at 25 mcg q.d., certainly no more than 50 mcg q.d. and increased as needed to normalize the thyroid stimulating hormone no sooner than every six weeks to eight weeks. 

### Resources

1. Klein I, Ojamaa K: Thyroid hormone and the cardiovascular system. *N Engl J Med* 2001; 344(7):501-9.
2. American Association of Clinical Endocrinologists: AACE medical guidelines for clinical practice for the evaluation and treatment of hyperthyroidism and hypothyroidism. *Endocr Pract* 2002; 8(6):457-69.