

# T3 Thyrotoxicosis Induced Dilated Cardiomyopathy

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Received June 14, 2019; Revised July 20, 2019; Accepted July 26, 2019

**Abstract** The most recognizable features of hyperthyroidism are those that result from the effects of triiodothyronine (T3) on the heart and cardiovascular system: decreased systemic vascular resistance and increased resting heart rate, left ventricular contractility, blood volume, and cardiac output. Although these measures of cardiac performance are enhanced in hyperthyroidism, the finding of clinical cardiac failure can be somewhat paradoxical. About 6% of thyrotoxic individuals develop symptoms of heart failure, but less than 1% develop dilated -cardiomyopathy with impaired left ventricular systolic function. Heart failure resulting from thyrotoxicosis is due to a tachycardia-mediated mechanism leading to an increased level of cytosolic calcium during diastole with reduced ventricular contractility and diastolic dysfunction, often with tricuspid regurgitation. Pulmonary artery hypertension in thyrotoxicosis is gaining awareness as a cause of isolated right-sided heart failure. In both cases, older individuals are more likely to be affected. Treatment needs to be directed at management of the acute cardiovascular complications, control of the heart rate, and thyroid-specific therapy to restore a euthyroid state that will lead to resolution of the signs and symptoms of heart failure.

**Keywords:** T3 thyrotoxicosis, heart failure, AFib

**Cite This Article:** David Bassilly, Michael Figart, Krithika Suresh, Mohamed Saad Eldin, and Samuel Massoud, "T3 Thyrotoxicosis Induced Dilated Cardiomyopathy." *American Journal of Medical Case Reports*, vol. 7, no. 10 (2019): 228-229. doi: 10.12691/ajmcr-7-10-2.

## 1. Introduction

The thyroid gland serves as an important regulator of metabolism, and its hormones affect many organs, including the cardiovascular system. Thyroid hormones have a significant role in regulating cardiac chronotropism and inotropism. Thyrotoxicosis can cause a high output cardiac state characterized by increases in heart rate, contractility, and cardiac output and reductions in peripheral systemic vascular resistance, which then can lead to high output heart failure. Although unusual, hyperthyroidism also causes low-output heart failure in 6 to 15% patients. Even in those patients, dilated cardiomyopathy with impaired systolic function is rare

## 2. Case Presentation

A 67-year-old white female with PMH of Hypothyroidism and Hypertension presented to the ED with palpitations and Shortness of breath for the past 3 months. She previously had a para-thyroid nodule that was removed 9 months prior. At that time, she was found to have hypothyroidism and was started on Levothyroxine and Liothyronine. In the ED, EKG showed A Fib with RVR at a rate of 168 bpm (Figure 1). BNP was elevated at 484.

Troponin I was elevated at 0.19, TSH was <0.01. Free T4 was normal at 1.40, T3 was markedly elevated at 10.24. She was started on Cardizem drip to achieve rate control and was also started on Heparin drip. Cardiology was consulted. Levothyroxine was held and she was transferred to the floor. Cardizem was discontinued and she was started on Carvedilol. As her troponin remained elevated, she underwent cardiac catheterization which showed angiographically normal coronary arteries and EF 20% (Figure 2). Echo done also showed EF 20-25% (Figure 3). She was placed on LifeVest and prescribed Entresto, Aldactone and Lasix on discharge. Levothyroxine dose was decreased and Liothyronine was discontinued. Free T3 decreased to 1.56.

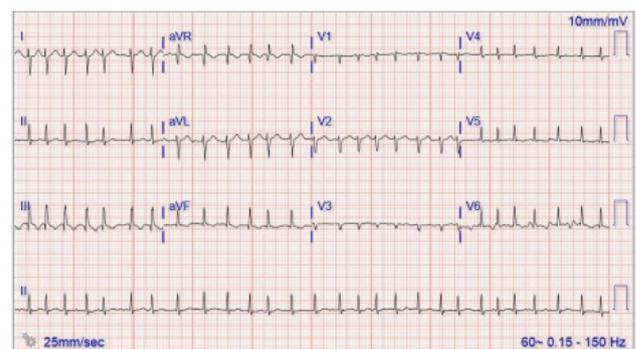


Figure 1. EKG showing AFib with RVR

**Cath Report:**

- The ejection fraction is estimated to be 20%.

Right dominant coronary system.  
 Angiographically normal coronary arteries.  
 Mildly dilated left ventricle, ejection fraction 20%, severe global hypokinesis.  
 Moderate to severe elevation of the left ventricular end-diastolic pressure measured at 22 to 24 mmHg.

Right radial access was used, patient tolerated procedure with no acute complications.

Impression: Rate related cardiomyopathy probably related to iatric hyperthyroidism.

**Figure 2.** showing Cath report

**Echo Report**

Left Ventricle: Normal in size. Ejection fraction estimated to be 20-25%, with decreased systolic function. Atrial fibrillation pattern observed.

**Figure 3.** Showing Echo report

The cause of hyperthyroidism should be determined and corrected. Non-selective beta blockers are used to help control heart rate and contractility as a temporary measure until further testing and correction of the cause of hyperthyroidism can be completed.

### 3. Discussion

- An increase in thyroid hormone can lead to increase in myocardial contractility and heart rate. Elevated thyroid hormone can lead to CHF, Atrial fibrillation or even acute coronary syndrome. Triiodothyronine, T3, binds to nuclear receptors on cardiac myocytes which alters gene expression & causes a beta-adrenergic effect on myocytes. This causes an increase in LVEF, HR, diastolic relaxation and cardiac output. Over the course of time, LV function can decrease allowing for CHF to set in which pre-disposes the patient to arrhythmias.

### 4. Conclusion

- In patients with congestive heart failure, atrial fibrillation or pulmonary hypertension that is caused by thyrotoxicosis, the underlying problem should be treated.

### Acknowledgements

The author(s) received no financial support for the research, authorship, and or publications for this article.

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