

# Heart Failure in Thyrotoxic Cardiomyopathy: Extracorporeal Membrane Oxygenation Treatment for Graves' Disease

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**Abstract:** Thyrotoxicosis-induced cardiomyopathy and consequent heart failure is one of the most grave complications of uncontrolled hyperthyroidism. In such patients, early recognition of thyrotoxicosis, and directed antithyroid therapy can lead to rapid normalization of left ventricular function. Herein, we present a case of a 29-year-old male with Graves' disease who developed heart failure with severe deterioration of left ventricular function

and eventually, circulatory collapse. Height and weight of the patient were 1.8 m and 84 kg, respectively. The patient was placed on venoarterial extracorporeal membrane oxygenation for immediate circulatory support, with restoration of cardiac function after 6 days. **Keywords:** extracorporeal membrane oxygenation, heart failure, hyperthyroidism, thyrotoxicosis, cardiomyopathy. *JECT. 2015;47:231–232*

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One of the most morbid complications of thyrotoxicosis is heart failure associated with hypotension, with a mortality rate approaching 30% (1). There is scant published information on the outcomes of venoarterial mode extracorporeal membrane oxygenation (VA ECMO) in treating circulatory collapse in patients with thyrotoxicosis-induced cardiomyopathy.

## DESCRIPTION

A 29-year-old African American male with Graves' disease on methimazole, bipolar disorder and depression presented with 3 days of dyspnea, palpitations, and epigastric abdominal pain. He had not been taking his methimazole for the past 45 days. Examination revealed an anxious, tachypneic young man with no jugular venous distension, no cardiac murmurs, lungs clear to auscultation, and no peripheral edema. Electrocardiogram (EKG) showed 2:1 atrial flutter with heart rate of 160. Laboratory evaluation showed suppressed thyroid stimulating hormone and markedly elevated free thyroxine (T4) and triiodothyro-

nine (T3). A diagnosis of thyrotoxicosis was made, and propylthiouracil and propranolol were started. Despite therapy, the patient's dyspnea worsened and a transthoracic echo (TTE) revealed left ventricular ejection fraction (LVEF) <20%, mildly dilated left ventricle (LV), severely enlarged left atrium, and moderate mitral regurgitation.

Despite being maintained on this antithyroid regimen, 2 days later, the patient developed hypoxia with bradycardia, degrading to pulseless electrical activity cardiac arrest. The advanced cardiac life support protocol was initiated, and he converted to a pulseless wide QRS complex tachycardia. He was defibrillated and subsequently regained spontaneous circulation; however, high doses of epinephrine, norepinephrine, and vasopressin drips were inadequate to maintain his blood pressure. An EKG-triggered intra-aortic balloon pump (IABP) was placed after the code for circulatory support, but due to unsatisfactory improvement in hemodynamics he was placed on mechanical circulatory support with VA ECMO a few hours later, and the IABP was removed. The patient was emergently taken for cardiac catheterization, which revealed no evidence of coronary artery disease. Propranolol was continued at this time. Chest x-ray at this time showed cardiomegaly with marked pulmonary edema. He remained hemodynamically stable on ECMO and was progressively weaned from inotropic support and decannulated on the 7th day after initiation of ECMO. TTE on the 6th day of ECMO showed LVEF improved to 35–39% as well as decreased LV dilatation and mitral regurgitation. TTE

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1 day after weaning ECMO showed a further increase in LV systolic function with EF 45–49%. The patient was extubated 2 days after decannulation from ECMO, and his clinical and functional status continued to improve during his hospital course. At discharge, he returned to baseline cognitive status and functional capacity.

## COMMENT

The hallmark of thyrotoxicosis-induced heart failure is a precipitous decrease in systemic venous resistance, mediated by the peripheral vasodilator adrenomedullin, leading to a decrease in blood pressure (2,3). The mechanism for heart failure is mediated by tachycardia, which leads to increased cytosolic levels of calcium in diastole, coupled with reduced ventricular contractility and diastolic dysfunction (4). Thyroid hormone action on the myocytes leads to an increase in messenger RNA coding for contractile elements and sarcoplasmic reticulum  $\text{Ca}^{2+}$  adenylypyrophosphatase with an accompanying increase in left ventricular mass (5). A direct toxic effect of thyroid hormone on cardiac myocytes has been implicated in systolic and diastolic dysfunction (6). Furthermore, notable causes of isolated right-sided heart failure in this condition are tricuspid regurgitation and pulmonary artery hypertension (4).

The most effective modality of treatment for heart failure in this setting remains antithyroid therapy, such as propylthiouracil and methimazole, which correct the primary hemodynamic disturbance responsible for the increase in stroke volume (7). A useful adjuvant to antithyroid therapy is nonselective  $\beta$ -receptor blockade through an agent such as propranolol, which markedly reduces the degree of tachycardia and vasodilation as both  $\beta_1$  and  $\beta_2$  receptors are targeted (8).

Electrophysiological derangements such as atrial premature depolarizations, paroxysmal atrial tachycardia, and atrial flutter are frequently seen in patients with hyperthyroidism. Most significantly, 10–21% of patients develop

atrial fibrillation (9), which is the final trigger for decompensation in 5–15% of patients (10).

This case demonstrates that prompt recognition of heart failure in the context of thyrotoxicosis is vital to hastening a return to normal cardiac function and prevention of adverse events. Even in young patients, thyrotoxicosis can lead to a reversible yet severe state of left ventricular dysfunction. Early treatment with antithyroid agents and  $\beta$ -receptor blockade in combination with close monitoring of hemodynamic function may lead to rapid improvement in clinical status as well as normalization of the LVEF. In the case of circulatory collapse, mechanical circulatory support through ECMO may be considered, when available, to stabilize patients and allow the myocardial function to recover.

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