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Thyrotoxicosis-Induced Cardiomyopathy Complicated by Refractory Cardiogenic Shock Rescued by Extracorporeal Membrane Oxygenation

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Patient: Male 53-year-old
Final Diagnosis: Thyrotoxicosis-induced cardiomyopathy
Symptoms: Dyspnea
Medication: —
Clinical Procedure: —
Specialty: Cardiology • Critical Care Medicine

Objective: Unusual clinical course

Background: Thyrotoxicosis-induced cardiomyopathy is a rare but potentially life-threatening condition that occurs in less than 1% of thyrotoxic individuals. Severely impaired left ventricular systolic function can lead to an overt cardiogenic shock requiring mechanical circulatory support. Abnormal cardiac structure and function are potentially reversible after achievement of euthyroid state.

Case Report: We present a case of a 53-year-old patient with a diagnosis of thyrotoxicosis-induced acute heart failure. Transthoracic echocardiography revealed a mildly dilated left ventricle and severely reduced systolic function with ejection fraction of 20%. Subsequently, the patient developed refractory cardiogenic shock, which was treated with the use of extracorporeal membrane oxygenation (ECMO). After early intensive treatments to achieve euthyroid state, the clinical status significantly improved. Echocardiography prior to discharge showed improvement of left ventricular ejection fraction to 40%. The anti-TSH receptor was positive and Grave's disease was diagnosed. The patient eventually returned to baseline functional status and could return to basic activities of daily living without limitations.

Conclusions: Early diagnosis of cardiac involvement in patients with thyrotoxicosis is critical. Promptly delivered intensive treatment with rapid achievement of euthyroid state can reverse cardiac dysfunction and improve patient outcomes. The use of ECMO can be considered as a "bridge" to recovery of cardiac function after restoration of euthyroid state.

Keywords: Cardiomyopathies • Extracorporeal Membrane Oxygenation • Heart Failure • Thyroid Crisis

Full-text PDF: https://www.amjcaserep.com/abstract/index/idArt/935029

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Background

Heart failure (HF) is the most profound manifestation of thyrotoxicosis. Presentations may include palpitations, exertional dyspnea, and edema. Approximately 1% of thyrotoxic patients develop thyrotoxicosis-induced cardiomyopathy, a rare but life-threatening type of dilated cardiomyopathy causing severely impaired left ventricular function and resulting in overt cardiogenic shock [1,2]. Early diagnosis is crucial since the patient is critically ill and urgently needs support measures in the intensive care unit. The use of mechanical circulatory support may be required in patients with refractory shock to prevent organ failure and reduce short-term mortality [2]. However, the abnormal cardiac structure and function are potentially reversible after achievement of euthyroid state [1]. In this report, we discuss the case of a 53-year-old patient with progressive dyspnea for 4 months who presented with unstable atrial fibrillation and cardiogenic shock. The diagnosis of thyrotoxicosis-induced acute HF was made. The patient was successfully treated by extracorporeal membrane oxygenation (ECMO). The cardiovascular effect of thyrotoxicosis and the role of ECMO in patients with thyrotoxicosis were reviewed.

Case Report

A 53-year-old previously healthy patient without underlying conditions presented with a 4-month history of progressive dyspnea with orthopnea, paroxysmal nocturnal dyspnea, and lower-extremities edema. The patient reported hand tremors, palpitations, irritability, and a 13-kg weight loss (from 63 kg to 50 kg) over the last 12 months. On examination, the body temperature was 37.1°C. The heart rate was 180 beats per minute with blood pressure of 100/62 mmHg. Examination revealed totally irregular heart rhythm, an apical impulse at the 6th intercostal space and anterior axillary line, fine crepitations at both lower lungs, and pitting edema in both legs. The thyroid gland was unremarkable.

Electrocardiogram revealed atrial fibrillation without significant ST segment elevation or depression (**Figure 1**). Chest radiography showed cardiomegaly and cephalization of both lungs (**Figure 2A**). Transthoracic echocardiography revealed a mildly dilated left ventricle with severely reduced systolic function with ejection fraction of 20% (**Figure 3**). High-sensitivity serum troponin-I was 36.9 ng/l (normal range: <15.6 ng/l) without dynamic change at 1 h after initial test. The thyroid function test revealed a suppressed thyroid-stimulating hormone (TSH) of <0.008 uIU/ml (normal range 0.35 to 4.94 uIU/ml), a normal free triiodothyronine (Free T3) of 2.04 pg/ml (normal range 1.6 to 4.0 pg/ml), and an elevated thyroxine (Free T4) of 1.65 ng/dl (normal range 0.7 to 1.48 ng/dl).

The patient was treated for an unstable acute atrial fibrillation with a 100J synchronized cardioversion, which did not successfully convert the rhythm to sinus rhythm. Subsequently, the patient developed hypotension, alteration of consciousness and respiratory distress. The patient was intubated for positive ventilation. The patient was diagnosed with thyroid storm complicated by acute HF causing cardiogenic shock and respiratory failure. The Burch-Wartofsky point scale (BWPS) score was 75. The patient was then admitted to the coronary care unit (CCU).

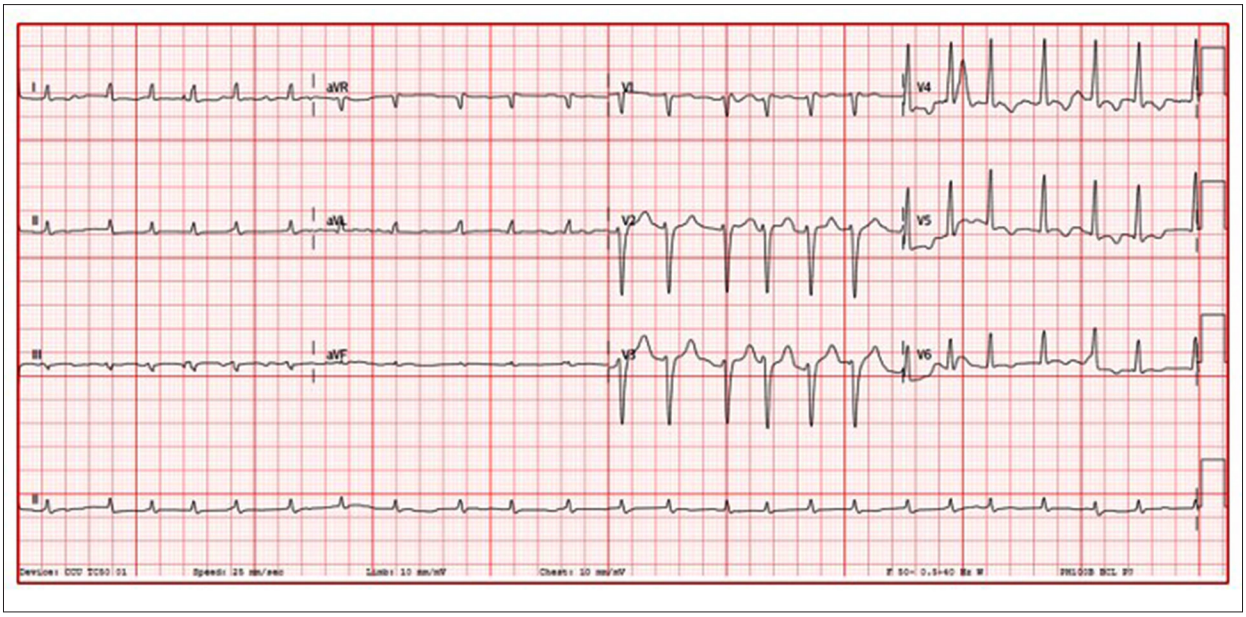

Figure 1. Electrocardiogram showing atrial fibrillation with rapid ventricular response, poor R-wave progression, and low voltage in the limb leads, with no specific ST and T wave abnormalities.

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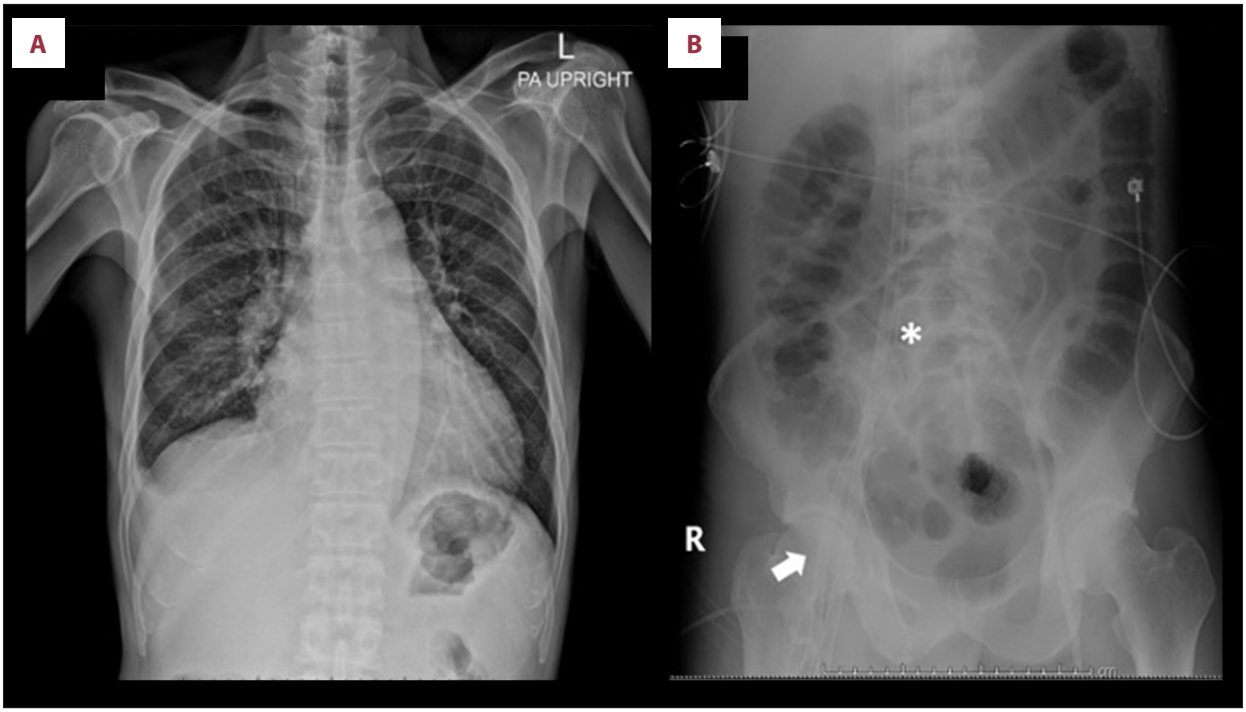

Figure 2. (**A**) Chest radiograph prior to ECMO showing cephalization (**B**) Abdominal X-ray showing inflow (asterixis) and outflow cannular (arrow) of the ECMO circuit in the inferior vena cava and the femoral artery, respectively.

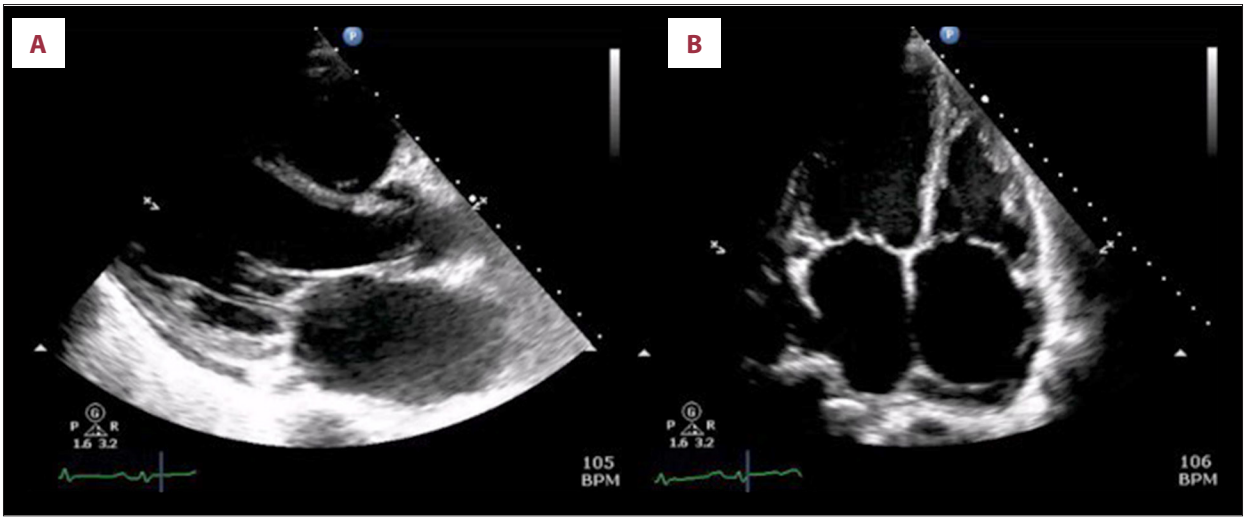

Figure 3. (**A**) Parasternal long-axis view of transthoracic echocardiography. (**B**) Four-chamber apical view of transthoracic echocardiography. Both show a dilated left ventricle with severely reduced systolic function and bilateral atrial enlargement.

A regimen of propylthiouracil, dexamethasone, and supersaturated potassium iodide was promptly initiated. Therapeutic plasmapheresis was performed at 13 h after presentation. Hemodynamics were supported with inotrope and vasopressor without improvement in clinical status. Due to severe tissue hypoperfusion and worsening serum lactate level despite maximum doses of epinephrine and dobutamine, peripheral veno-arterial ECMO was initiated (**Figure 2B**).

After initiation of ECMO, the clinical status improved and the lactate level returned to normal. The dobutamine and epinephrine infusions were subsequently weaned off after 3 days. During hospitalization, the patient developed acute kidney injury which required hemodialysis without the need for long-term renal replacement therapy. The electrical cardioversion was performed again to stabilize atrial fibrillation. The patient was extubated 6 days after admission and successfully decannulated from ECMO after 9 days of support. Levosimendan

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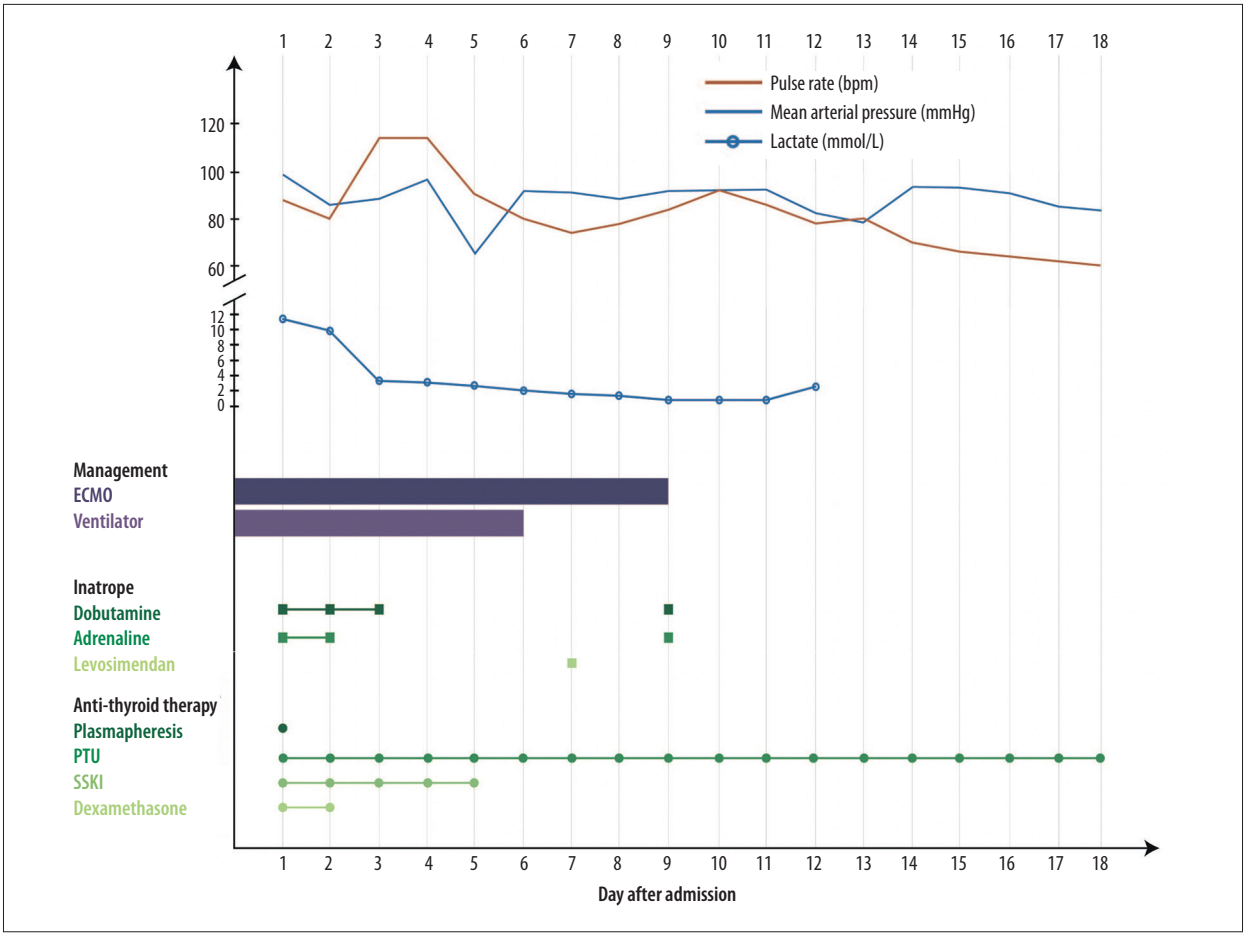

Figure 4. Graph showing the clinical course of the patient including mean arterial pressure, pulse rate, lactate level, and selected management before and after the initiation of extracorporeal membrane oxygenation (ECMO).

was given 2 days prior to ECMO weaning, then on the day of weaning. Dobutamine and epinephrine were given briefly in the operating room to support hemodynamics. The anti-TSH receptor was positive (22.8 IU/l; normal range ≤1.75 IU/l) and Grave's disease was diagnosed.

After 18 days of hospitalization, the patient was discharged on oral methimazole, carvedilol, amiodarone, and warfarin. Clinical status significantly improved. Echocardiography prior to discharge showed improvement of left ventricular ejection fraction to 40% (**Figure 4**). At 6-month follow-up, the patient eventually returned to baseline functional status and could do basic activities of daily living without limitations.

Discussion

Thyrotoxicosis-induced cardiomyopathy (TCM) is a rare but potentially life-threatening condition. Despite the fact that all measures of cardiac performance, including resting heart rate, left ventricular contractility, and cardiac output, are enhanced during thyrotoxicosis, clinical presentations of HF can still be found. Approximately 6% of thyrotoxic patients develop signs and symptoms of HF. This is a consequence of expanded plasma volume and a hyperdynamic circulatory state in the absence of left ventricular dysfunction. Less than 1% of these patients are found to develop dilated cardiomyopathy with evidence of severely impaired left ventricular systolic function consistent with TCM, as seen in echocardiographic findings of this patient [1,2].

Triiodothyroxine (T3) is an active form of the thyroid hormone that is responsible for cardiovascular manifestations in hyperthyroidism. T3 increases cardiac workload by increasing heart rate and cardiac contractility and decreasing systemic vascular resistance. Excess T3 can result in up to a 300% increase in cardiac output [1]. It also has direct genomic effects on multiple regulatory and structural genes of cardiac myocytes such as sarcoplasmic reticulum calcium-activated adenosine triphosphatase (SERCA2a) and α myosin heavy chain (α-MHC), resulting in abnormal intracellular calcium regulation and impairing systolic and principally diastolic function [3]. T3

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also has a non-genomic effect on both vascular smooth muscle cells and the endothelial nitric oxide pathway leading to a systemic vascular resistance reduction [1,3]. As seen in this case presentation, prolonged elevation of the thyroid hormone can result in atrial fibrillation, which occurs in about 10% to 25% of hyperthyroid patients due to enlargement of the left atrium from either an expanded plasma volume or impaired ventricular function [4].

Clinical signs and symptoms of acute HF in patients with thyrotoxicosis can be found during a thyroid storm, a life-threatening manifestation of multiorgan decompensation that can progress to cardiovascular collapse and shock. Thyroid storm is a clinical diagnosis. Patients commonly present with severe pyrexia, profuse sweating, and tachycardia. Symptoms can vary in severity and involve multiorgan systems, resulting in non-specific clinical manifestations and pending multiple-organ failure. The Burch-Wartofsky point scale (BWPS) is widely used, with a score of 45 points or greater being highly suggestive of a thyroid storm. However, if clinical manifestations are highly suspected, regardless of BWPS, treatments should be initiated as soon as possible to reduce mortality [5]. If the clinical status fails to improve despite appropriate medical treatment, therapeutic plasmapheresis (TPE) is considered to be the most effective management to decrease circulation of the thyroid hormone in patients with thyroid storm [2]. TPE effectively exchanges serum proteins, which is the binding agent for approximately 99% of thyroid hormones. According to research conducted in Japan, a high mortality rate of 37.5% was still observed in patients who received TPE. Although TPE initially alleviated severe thyrotoxicosis, most patients died from late-onset complications such as refractory circulatory derangement and multiple-organ failure, which may require the use of mechanical circulatory support such as ECMO to improve survival [6].

Veno-arterial extracorporeal membrane oxygenation (VA-ECMO) helps maintain sufficient end-organ perfusion and is an efficacious treatment for patients with severe cardiogenic shock from causes including post-cardiac surgery, acute myocardial infarction, myocarditis, and post-cardiac arrest [7-9]. The role of ECMO in circulatory collapse resulting from endocrine emergencies remains unclear [10]. White et al performed a systematic review of 13 case reports of patients with thyrotoxicosis-induced cardiomyopathy requiring support with ECMO; the survival-to-discharge rate was 78.5%, with nearly complete recovery of left ventricular ejection fraction in these patients [7]. Chai et al compared the clinical outcomes of patients receiving ECMO in endocrine emergencies and acute myocardial infarction, showing that cardiac function recovery was significantly faster in patients with endocrine emergencies compared to those with acute myocardial infarction. These results suggest that ECMO can be beneficial in endocrine emergencies [10].

Conclusions

Diagnosis of thyrotoxicosis requires a high level of vigilance and a low threshold for early diagnosis of cardiac involvement. Refractory circulatory collapse can be life-threatening, but early intensive treatments with rapid achievement of euthyroid state can reverse the left ventricular dysfunction and improve patient outcomes. The use of ECMO can be considered as a "bridge" to recovery of cardiac function after restoration of euthyroid state.

Declaration of Figures' Authenticity

All figures submitted have been created by the authors who confirm that the images are original with no duplication and have not been previously published in whole or in part.

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