

Complete Recovery of Thyrotoxicosis Cardiomyopathy After Antithyroid and Corticosteroid Therapy: A Case Report

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ABSTRACT

Thyroid storm (TS) is an extreme hypermetabolic state due to thyrotoxicosis. TS may manifest as cardiovascular symptoms, including atrial fibrillation (AF) (9 – 23%) and heart failure (HF). Dilated cardiomyopathy (DCM) and HF develop at a later stage in approximately 1% of thyrotoxicosis patients. A 46-year-old man presented to the emergency department with a chief complaint of severe dyspnea and rapid AF. Blood test examination showed overt thyrotoxicosis. The Burch-Wartofsky point scale is 60 (highly suggestive of TS). Echocardiography showed severely reduced left ventricular (LV) systolic function. The patient was diagnosed with TS, rapid AF with DCM, probably due to thyrotoxic cardiomyopathy. After 3 months of antithyroid therapy, corticosteroids, and optimal HF management, we observed complete recovery of his ventricular function, along with spontaneous conversion to sinus rhythm. LV systolic dysfunction is described in a small percentage of thyrotoxicosis patients. In later stages of thyrotoxicosis cardiomyopathy (TCM), HF phenotype of DCM will develop, characterized by progressive reduction of myocardial systolic function that marks advanced stages of TCM. This report highlighted the importance of thyroid investigation as an integral part of HF etiological work-up due to its reversibility.

Keywords: thyrotoxicosis, heart failure, dilated cardiomyopathy, antithyroid, case report.

INTRODUCTION

Thyroid storm (TS) is a state of extreme hypermetabolic condition that increases the sensitivity of β adrenergic receptor and high catecholamine response due to thyrotoxicosis. Without adequate treatment, TS has a mortality rate of as high as 100%, while proper treatment lowers the rate to 10-50%.¹ Medication non-compliance and untreated chronic hyperthyroidism are known to precipitate TS. Thyroid storm may manifest in various

organ systems, including but not limited to the cardiovascular system, presenting as atrial fibrillation (AF) (9-23%) or heart failure (HF).² Other organs affected by TS are the nervous, gastrointestinal, and integumentary systems.¹ Dilated cardiomyopathy (DCM) and HF develop in approximately 1% of thyrotoxicosis patients.^{2,3} The progression of thyrotoxicosis cardiomyopathy (TCM) is divided into three progressive stages: the first is the “hyperkinetic” phase characterized by high-output HF with

preserved left ventricular (LV) systolic function, followed by the “normokinetic” phase marked by compensated LV hypertrophy, and lastly the “hypokinetic” phase which is a decompensated stage with reversible or irreversible dilated cardiac chamber and reduced LV systolic function.² We reported a case of TS with overt cardiovascular manifestation who experienced complete recovery after management with antithyroid and corticosteroid therapy.

CASE ILLUSTRATION

A 46-year-old man presented to the emergency department with a chief complaint of severe dyspnea and “pounding” heart sensations. The symptoms commenced earlier that day and worsened progressively. The patient also complained of chest pain, nausea, and frequent vomiting. There is no history of similar symptoms. On admission: patient fully alert, blood pressure 140/85 mmHg, heart rate 155 beats per minute irregular, respiratory rate 26, SpO₂ 99 %, temperature 38.2 °C.

Electrocardiography revealed rapid AF with a QRS rate of 155-165x/minute (**Figure 1**). Blood test examination showed TSH <0.05 mIU/L (normal value 0.5-4.15 mIU/L) and FT₄ > 7.77 ng/dL (normal value 0.7-1.55 ng/dL), normal renal functions, and other results were unremarkable. Chest x-ray demonstrated cardiac silhouette enlargement with increased pulmonary vascular markings and infiltrate

with right hemithorax predominance (**Figure 2**). Transthoracic echocardiography showed right atrial (RA) and left atrial (LA) dilation (RA and LA areas were 24.3 and 34.5 cm², respectively) (Table 1), left ventricular dilation with severely reduced LV ejection fraction [LVEF 19 % (Simpson) without segmental wall motion abnormality, averaged LV global longitudinal strain (GLS) -5.8 %]. Right ventricular systolic function was preserved [TAPSE 2.1 cm], with mild functional mitral regurgitation with E/e' 11.9 (**Figure 3**). The Burch-Wartofsky point scale is 60. This scale is used to evaluate the likelihood of TS based on temperature, heart rate, presence of AF, heart failure, gastrointestinal, and central nervous system disturbance. The scale > 45 is highly suggestive of TS.

The patient was diagnosed with TS, rapid AF, and DCM, with a suspicion of TCM, with a differential diagnosis of coronary artery disease (CAD) etiology. He administered 150 mg i.v. Amiodarone for emergency acute rate control and 40 mg i.v. Furosemide for congestive HF symptoms. Contrast-enhanced computed tomography coronary angiography revealed a normal coronary artery without stenosis (**Figure 4**), thus ruling out ischemic origins of LV systolic dysfunction. Hence, the patient was diagnosed with DCM due to TCM. During admission, the patient was given enoxaparin 60 mg subcutaneously b.i.d. and warfarin 2 mg o.d. for thromboembolism prophylaxis, methimazole

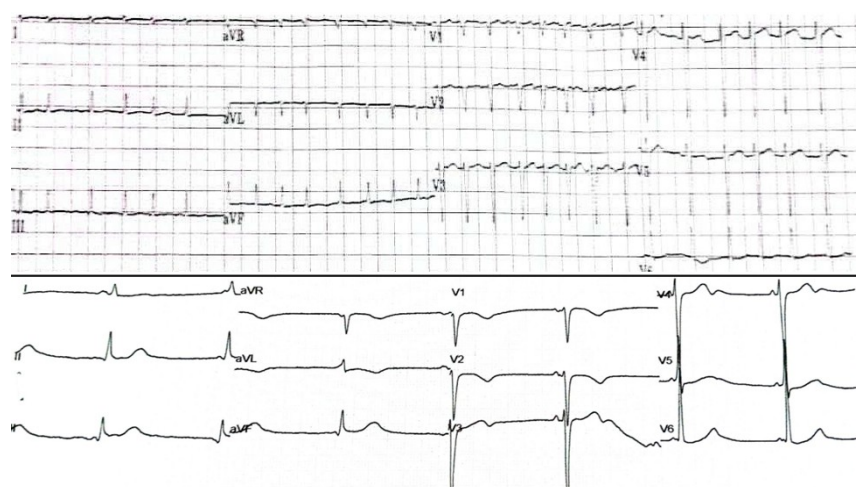


Figure 1. 12-lead electrocardiography. Upper panel: initial presentation ECG showed rapid atrial fibrillation; Lower panel: post-treatment follow-up ECG showed sinus rhythm, non-specific T wave inversion in precordial lead.



Figure 2. Patient chest x-ray on initial clinical presentation demonstrated cardiac silhouette enlargement with increased pulmonary vascular marking and infiltrate with right hemithorax predominance.

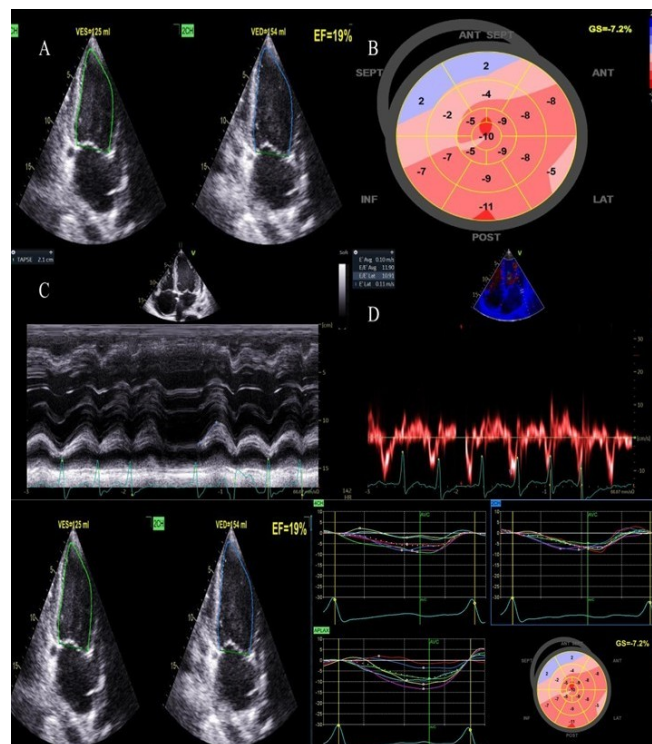


Figure 3. Baseline echocardiographic findings: (A) Simpson's method measurement, which showed LVEF of 19%; (B) LV GLS showed global reduction in LV systolic strain; (C) RV systolic function measurement showed TAPSE 2.1 cm; (D) Mitral annulus TDI showed E/e' 11.9. [LVEF: Left ventricular ejection fraction; GLS: Global longitudinal strain; TAPSE: Tricuspid annular plane systolic excursion; TDI: Tissue doppler imaging].

10 mg b.i.d. to suppress thyroid hormone synthesis, and hydrocortisone i.v. 100 mg, as well as propranolol 40 mg b.i.d. for rate control.

The standard guideline-directed medical therapy (GDMT) for HFrEF (loop diuretic, ACE-i, MRA) was also prescribed.^{4,5}

On the 5th admission day, vital signs were stable: HR 98 b.p.m irregular, BP 130/70 mmHg, RR 20 with significant improvement in heart failure symptoms. The patient was discharged uneventfully with methimazole, hydrocortisone, propranolol, warfarin, and HF GDMT. We observed spontaneous sinus rhythm conversion

at the 1st ambulatory outpatient visit (**Figure 1**). After 3 months of evaluation, euthyroid state was achieved. We also noted a complete recovery of LV function along with structural improvement [LVEF 54 % (Simpson) with LV GLS -17.1 %], along with a reduction of RA and LA size (15.9 and 14.4 cm², respectively) (Figure 5) (Table 1).

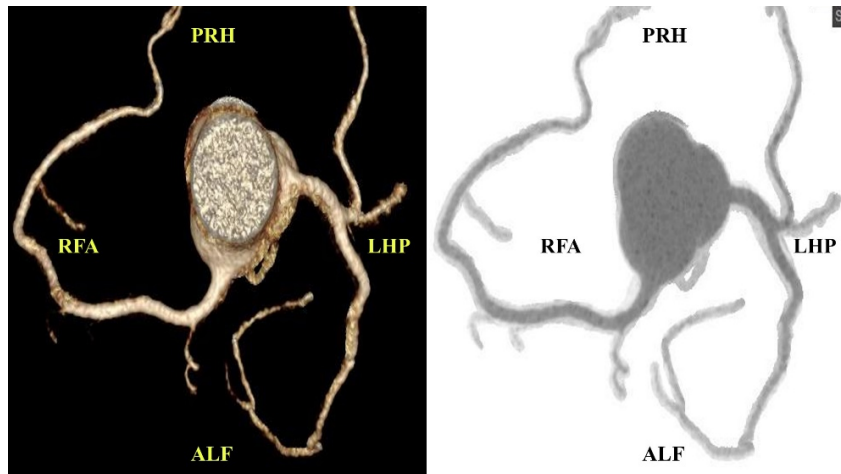


Figure 4. Coronary computed tomography scan with contrast showed normal coronary anatomy without stenosis.

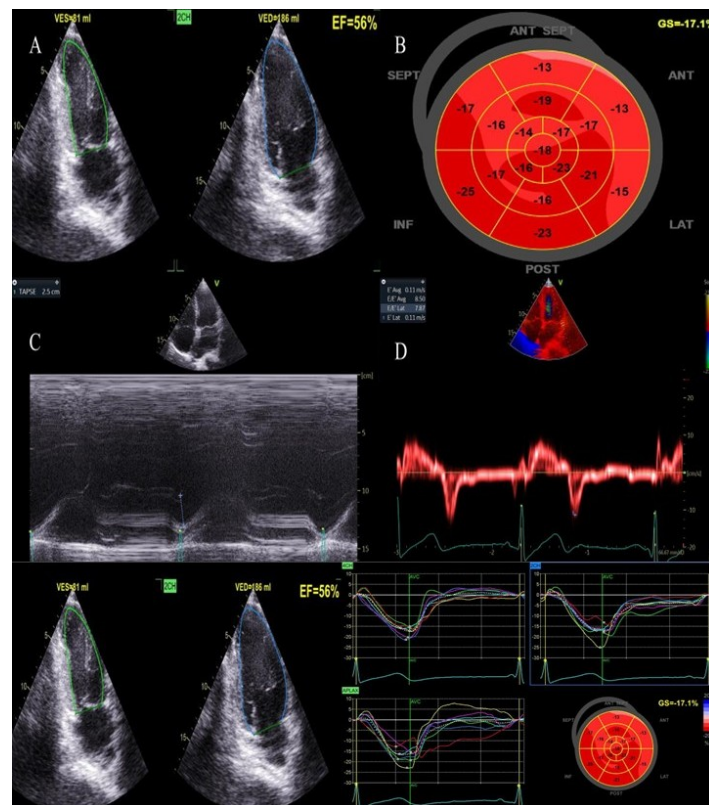


Figure 5. 3-month evaluation echocardiographic finding: (A) Simpson's method measurement showed LVEF improvement to 56%; (B) LV GLS showed LV reverse remodeling and global improvement in LV systolic strain; (C) RV systolic function measurement showed TAPSE 2.5 cm; (D) Mitral annulus TDI showed E/e' 8.5. [LVEF: Left ventricular ejection fraction; GLS: Global longitudinal strain; TAPSE: Tricuspid annular plane systolic excursion; TDI: Tissue doppler imaging].

Table 1. Patient's summary on trajectory of thyroid hormone profile, echocardiographic finding and medication during initial presentation, 1 month post-treatment, and 3 months post-treatment.

Parameters	Initial presentation (October 2024)	1-month evaluation (November 2024)	3-months evaluation (January 2025)
Thyroid hormone profile			
TSH (normal 0.5 – 5.0 mIU/L)	<0.05	<0.05	1.2
FT4 (normal 0.8 – 1.9 ng/dL)	>7.77	1.6	1.18
Echocardiographic findings			
LVEDV (mL)	165	167	170
LVESV (mL)	132	83	78
LVEF (%)	20	50	54
TAPSE (mm)	21	22	25
Mitral peak E velocity (m/s)	1.18	1.09	0.9
E/e' average	11.9	10.7	8.5
LV averaged GLS (%)	-7.2	-15.2	-17.1
LA area (cm ²)	34.5	N/A	14.4
RA area (cm ²) (normal <18 cm ²)	24.3	N/A	15.9
Drug regimen			
Ramipril p.o.	10 mg o.d.	10 mg b.i.d.	10 mg o.d.
Propranolol p.o.	40 mg b.i.d.	40 mg b.i.d.	40 mg b.i.d.
Spironolactone p.o.	25 mg o.d.	25 mg o.d.	25 mg b.i.d.
Warfarin p.o.	2 mg o.d.	2 mg o.d.	-
Thiamazole p.o.	10 mg b.i.d.	5 mg o.d.	5 mg b.i.d.
Hydrocortisone p.o.	-	20 mg o.d.	20 mg o.d.

TSH: thyroid-stimulating hormone; FT4: free T4; LVEDV: left ventricular end-diastolic volume; LVESV: left ventricular end-systolic volume; LVEF: left ventricular ejection fraction; TAPSE: tricuspid annular plane systolic excursion; GLS: global longitudinal strain; LA: left atrial; RA: right atrial; PO: per oral; OD: once daily; BID: twice daily.

DISCUSSION

We present a case of TS with HF_rEF. The patient showed acute decompensated HF syndrome that was precipitated by rapid AF. Echocardiographic investigation revealed significant LV systolic dysfunction, which was proven by EF and strain measurement.

Although thyrotoxicosis had been observed to result in high-output HF syndrome, characterized by preserved LVEF, in our patient, the reduction of LV systolic function has become evident, raising a concern of the involvement of cardiac myocytes in long-term thyrotoxicosis.^{2,3} In our patient, coronary CT angiography revealed no stenosis, while other conventional HF etiologies were absent. Hence, all evidence pointed toward hyperthyroidism as the single causal factor for his symptoms, also raising a suspicion of overt TCM. Since TCM is a diagnosis of exclusion, all other possible etiologies had to be ruled out.²

Thyrotoxicosis is a rare presentation of hyperthyroidism, resulting from excessive endogenous thyroid hormone production.

Thyroid storm, on the other hand, is an extreme manifestation of thyrotoxicosis marked by multi-organ manifestations.⁶ In response to thyroid-stimulating hormone (TSH), the thyroid gland secretes T3 (triiodothyronine) and T4 (thyroxine). The myocardium is mainly affected by T3, which at the cellular level has both genomic and nongenomic effects on the cardiac myocytes; the former involves binding to thyroid hormone receptor, while the latter includes direct modulation of membrane ion channels. Increased ventricular contraction function and cardiac output are also directly affected by T3 through MHC- α expression as well as upregulation of β -adrenergic and Na/K-ATPase receptors, increasing preload, reducing afterload, and enhancing inotropy and chronotropy by up to 300%.^{2,3,7} Furthermore, thyroid hormones can also cause changes in the membrane ion channels of extra-nuclear non-genomic bodies and produce vasodilatory effects through the induction of phosphatidylinositol 3-kinase/protein (PI3K/Akt) signalling pathway.⁸

In the long run, hyperthyroid impact substantial risk to AF development due to the abundance of β_1 adrenergic receptors in atrial myocytes, which leads to greater atrial myocyte sensitivity to thyroid hormone.² AF is the most commonly identified arrhythmia in thyrotoxicosis, with a prevalence rate of 13.8% as opposed to 2.3% in patients who have achieved euthyroid state.⁹ The typical phenotypes of HF due to thyrotoxicosis are marked by an increase in LVEF, stroke volume, and cardiac output.¹ However, if thyrotoxicosis persists, the HF phenotype of DCM will develop, characterized by the progressive reduction of myocardial systolic function that marks advanced stages of TCM.^{1,2,10} LV systolic dysfunction finding is described in only a small population of patients with thyrotoxicosis.² The development of DCM is due to the overexpression and release of excessive T3, stimulating the sympathetic nervous system by increasing β -adrenergic receptors.¹¹ The presence of DCM phenotype predominance with severely reduced LVEF found in our patient's echocardiographic results indicates a long period of thyrotoxicosis exposure.

This report highlighted the importance of thyroid etiology investigation as an integral part of HF etiological work-up due to its reversibility and favorable prognosis after antithyroid medication and GDMT, as seen in our case. The European Society of Cardiology guideline for management of HF recommended thyroid function assay as a basic diagnostic test in all patients with HF.⁵ In our case, we observed a brief period of complete reverse remodeling after 3 months of pharmacological management, which commonly takes 6 months after achieving the euthyroid state.² In addition to the improvement of ventricular function, we also observed the restoration of sinus rhythm. Similar cases of sinus rhythm conversion from AF and improvement of functional status after optimal antithyroid medication have been reported multiple times.¹²⁻¹⁶ Additionally, Rodriguez-Santiago *et al* reported successful management of acute HF during thyroid storm using only beta-blockers, where antithyroid therapy is not feasible.¹⁷

The reduction of RA and LA size was observed in our case (Table 1). However, we also noted a persistent atrial enlargement despite overall cardiac recovery. To explain this discrepancy, we hypothesized that the underlying mechanism of this phenomenon is related to greater sensitivity of atrial myocytes, compared to ventricular myocytes, to thyroid hormones. Hence, it is reasonable if atrial enlargement and dysfunction persist despite ventricular function recovery, since the adverse remodeling process on the atrium is more pronounced.²

The use of antithyroids to inhibit thyroid hormone hyperactivation is a mandatory main therapeutic approach.^{3,10} Similarly, the Japanese guideline for the management of thyroid storm focuses on suppressing excessive thyroid hormone release and the management of complications.¹⁸ Our patient was also prescribed hydrocortisone. In cases of thyrotoxicosis, the hypothalamic-pituitary-adrenal axis is compromised, resulting in a decrease in adrenal reserve. Steroids act as a prophylaxis to prevent insufficiency during thyroid storm.¹¹ A permanent euthyroid state is the paramount therapeutic goal that could lead to a better prognosis. Hence, the management principle in our case is comprised of β -blockers, antithyroids, hydrocortisone, and standard GDMT for heart failure.^{1,7,10}

CONCLUSION

Thyrotoxicosis cardiomyopathy is a rare but potentially lethal complication of hyperthyroidism. The presence of DCM and systolic dysfunction marked the advanced stages of cardiac thyrotoxicosis. Therefore, thyroid function assessment is of importance in the etiological investigation for HF patients, due to its reversibility and favorable prognosis if managed appropriately.

CONFLICT OF INTERESTS

The authors declare no conflicts of interest.

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ETHICAL APPROVAL AND INFORMED CONSENT

Patients provided informed written consent before data publication, in compliance with the COPE guideline.

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