

# “Postpartum Cardiogenic Storm” – The Importance of Investigating and Understanding Peripartum Cardiac Decompensation

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## Introduction

Cardiac decompensation in pregnancy and the postpartum period is complex, can be multifactorial, and often life-threatening. We describe a post-partum case of rare combination of Graves’ disease-induced thyrotoxic cardiomyopathy, superimposed on severe pre-eclampsia and COVID-19 infection.

## Case Study

### Ante-natal Course

A 22-year-old Nigerian woman (P0), BMI-25, booked for antenatal care at 24 weeks, and she was a known  $\beta$ -thalassemia carrier with borderline low haemoglobin.

She was diagnosed with polyhydramnios and developed **pre-eclampsia** at 32+4 weeks (uPCR 170), for which she was started on labetalol.

At 33 weeks, she had PPROM and went into spontaneous labour within 24 hours.

### Delivery & Immediate Postpartum Collapse

She underwent an emergency caesarean section (EMCS) under general anaesthesia for pathological CTG following a single dose of terbutaline, blood loss 700mls.

Forty minutes post-delivery, she developed profound bradycardia (HR 20 bpm), followed by cardiac arrest. Return of spontaneous circulation (ROSC) was achieved after one cycle of CPR. Post-resuscitation, myoclonic jerks noted, possibly due to seizure or neuromuscular blockade recovery.

**The cause of collapse thought to be hyperkalaemia ( $K^+$  7.3 mmol/L) related.**

She was managed with intravenous magnesium sulphate for 24 hours and continued on labetalol.

Remained tachycardic and mildly hypertensive in the few days following delivery.

### COVID-19 Infection

Day 1 postpartum, she developed pyrexia (37.7°C)  
Positive for COVID-19 (Omicron strain), with CRP of 30.

By **Day 5**, she had worsening shortness of breath, requiring high-flow oxygen.

**CXR:** consistent with COVID-19 pneumonia.

**CTPA:** ruled out pulmonary embolism.

### Echocardiography:

Normal LV function, high right ventricular systolic pressure (RVSP 59 mmHg).

**Cardiomyopathy related to COVID-19 and or severe pre-eclampsia were considered differentials.**

### Diagnosis of Graves’ Disease

For persistent tachycardia (HR 115–125 bpm) prompted further tests.

By **Day 6** thyrotoxicosis diagnosed

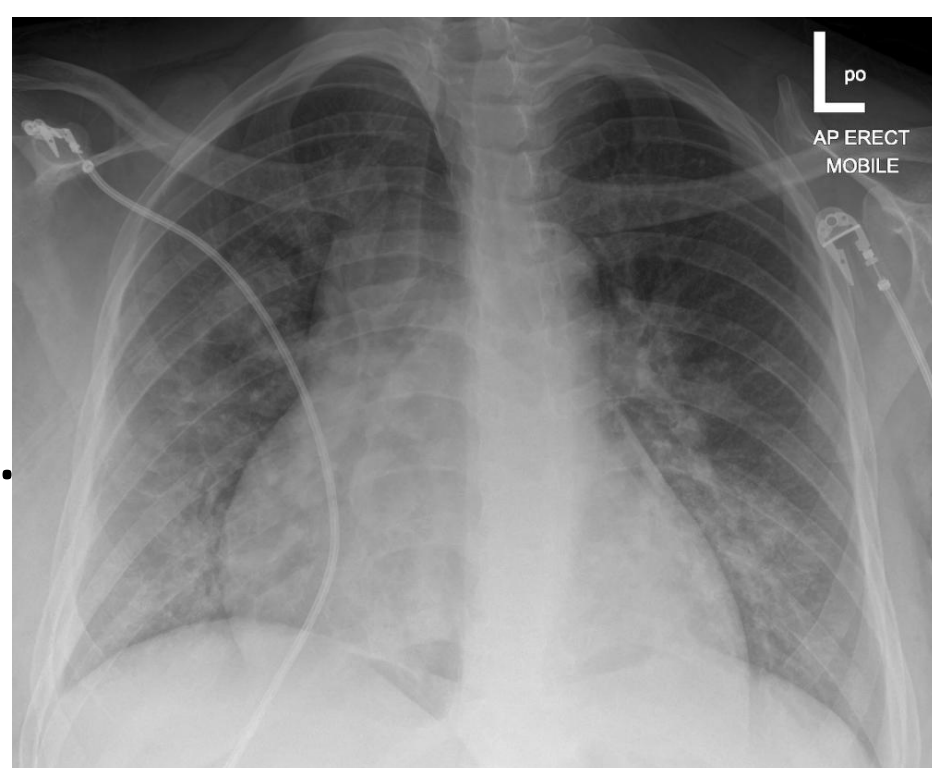
**TFT:** T4 77 pmol/L, TSH <0.01

**Ultrasound thyroid:** Coarse echotexture and increased vascularity, consistent with thyroiditis.

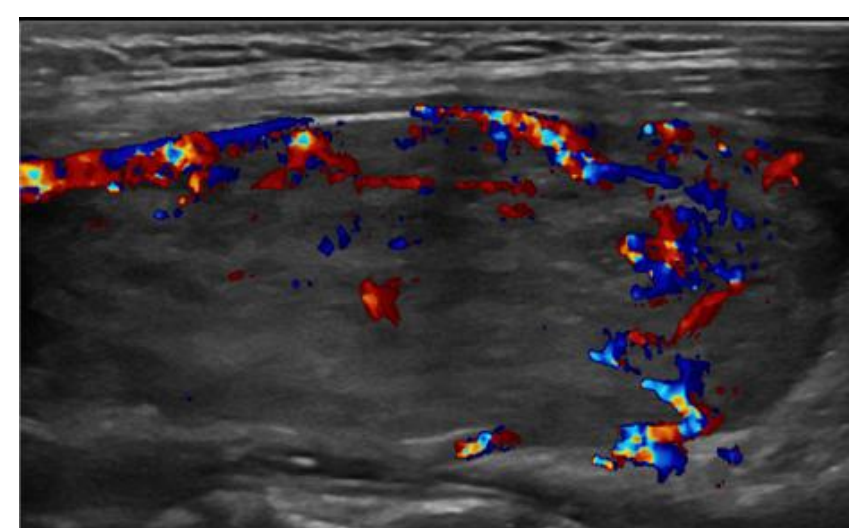
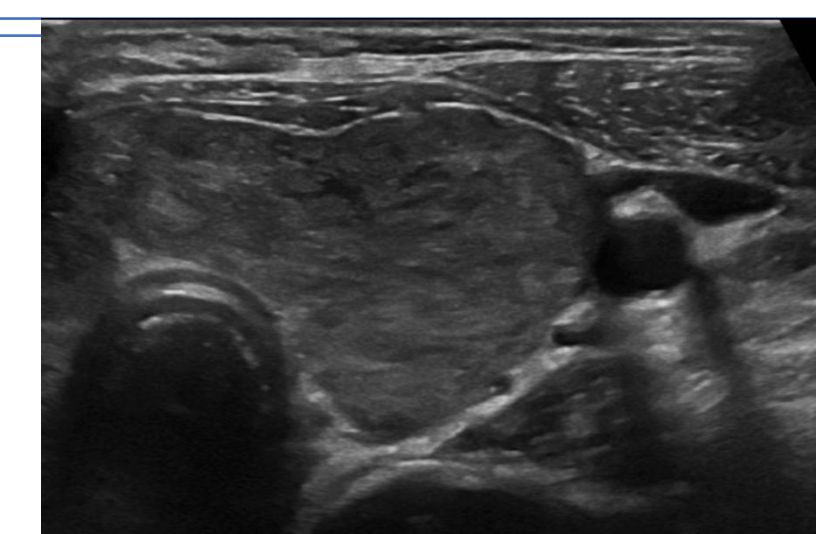
She was started on carbimazole, with clinical improvement.

Later confirmed high TSH receptor antibodies 3.33 suggestive of Graves’ D

She was discharged on Day 10 with follow-up arranged at 4 weeks.



Bilateral, multifocal, peripheral infiltrates



### Persistent Thyrotoxicosis & poor cardiac function

At her **Day 52** outpatient review, she remained thyrotoxicosis, normotensive.

**TFT:** (T4 72.4 pmol/L, TSH <0.01) after discontinuing carbimazole 2 weeks after discharge.

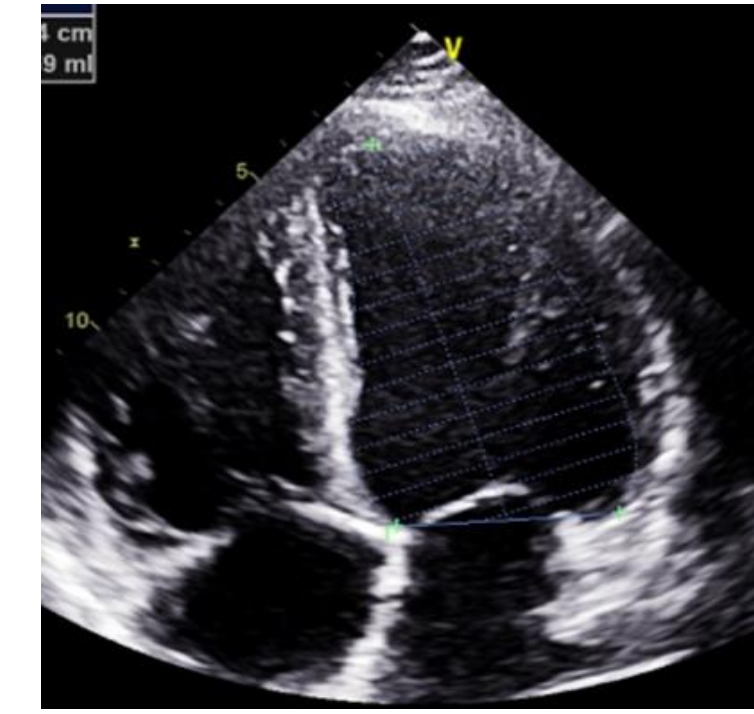
**Nuclear medicine thyroid scan:** She was restarted on carbimazole and beta-blocker therapy.

**Echocardiography** showed dilated LV & RV, with LV hypokinesia & LVEF 37%, PASP 51 mmHg



#### NM Thyroid

Symmetrical, homogenous increased uptake in both lobes.



#### Echocardiography

Dilated LV size (LVIDd 6.2cm), LVEF 37%, LV hypokinesia  
Dilated RV, mild TR, mild-to-mod PR,  
PASP 51 mmHg  
Mild-to-mod MR, biatrial dilatation

### Thyroid Storm & Biventricular Heart Failure

**Day 64** postpartum, the patient collapsed at home. In A&E she was delirious, hypotensive, with unrecordable oxygen saturation but maintained a central pulse. Resuscitation revealed high cardiac output (10 L/min), improved lactate with warm extremity.

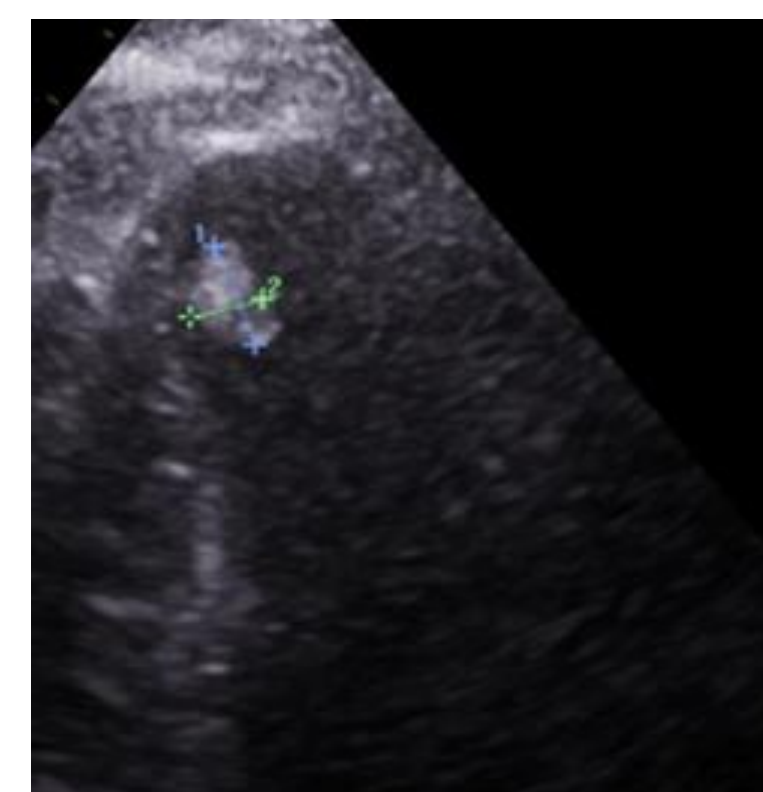
**CXR:** No pulmonary edema

**TFTs** confirmed thyroid storm (fT4 > 100 pmol/L, TSH < 0.01)

**TEE and stress cardiac MRI:** Biventricular Heart failure evidenced by LV dysfunction (EF-19%) & thrombus. Fully anticoagulated for cardiac thrombus.

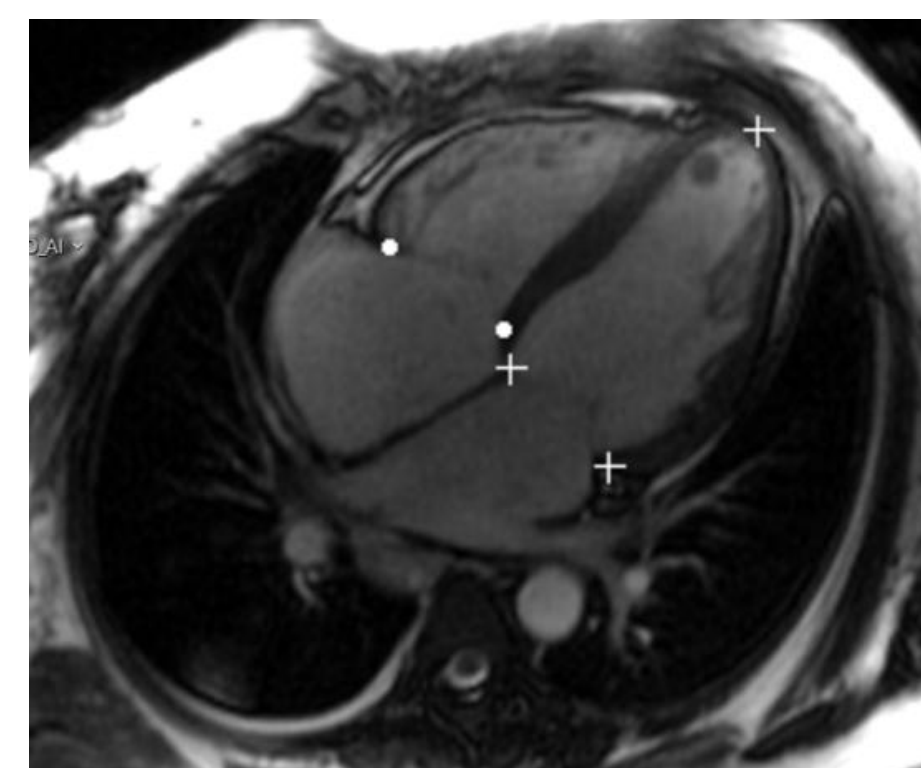
**Differentials at this time included**

1. Peripartum cardiomyopathy
2. Preeclampsia-related HF
3. Thyrotoxic cardiomyopathy



#### Echocardiography

14x12 mm LV apical thrombus.



#### Cardiac MR

Severe chamber dilatation with hypertrophy, no oedema or late enhancement.  
Confirmed LV thrombus

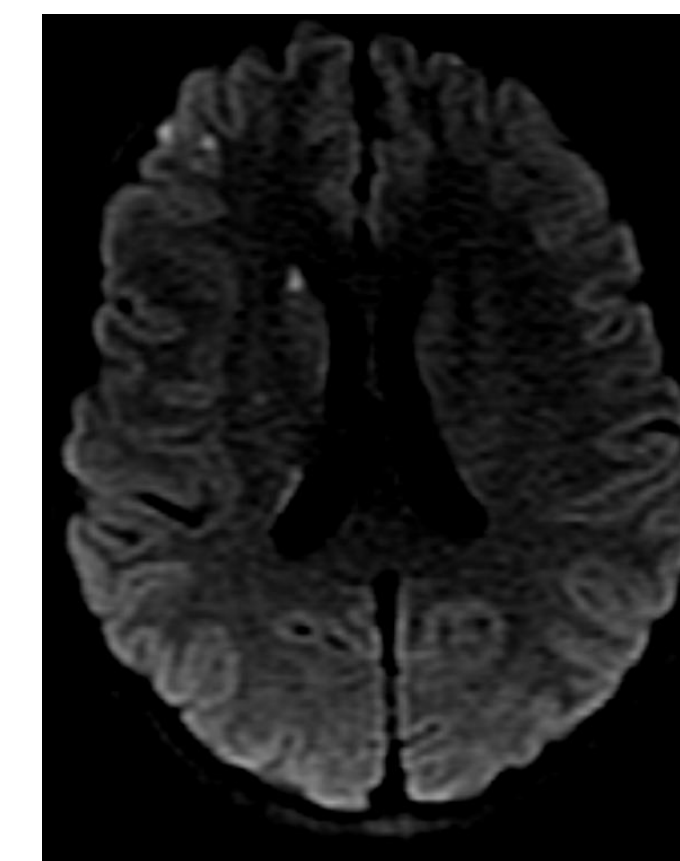
### Management of thyroid storm in the background of heart failure

- ✓ Propylthiouracil (act quickest, block production of T3, T4 and peripheral conversion of T4 to T3)
- ✓ Cholestyramine to reduce GI absorption of thyroid hormones
- ✓ Hydrocortisone infusion (decrease peripheral conversion of T4 to T3, prevents relative adrenal insufficiency)
- ✓ Esmolol as beta-blockade (cautious use as negative chronotropic and inotropic)

### Cardio-embolic Stroke (TIA)

On **Day 70**, during anticoagulant bridging therapy while introducing warfarin, even with good compliance, she developed brief left-sided weakness, facial droop, and dysphasia (<60 mins).

She recovered without deficit.



#### MRI Head

Multiple right-sided infarcts

### Outcome

Discharged on carbimazole, bisoprolol, with a progesterone implant and cardiology follow-up

At her 3-month post-discharge review with TEE, ventricular function had normalized.

## Discussion

*This case illustrates multifactorial decompensated heart failure from Graves’ thyrotoxicosis, preeclampsia, COVID-19, and immediate postpartum changes.*

Regarding Graves’ Disease, it typically improves mid-pregnancy but may worsen postpartum due to immune reactivation. Signifying the importance of close observation when underlying undiagnosed maternal condition can worsen.

**"Thyroid storm"** — a rare, life-threatening crisis — was considered the major contributing factor to high-output cardiac failure in the second instance, occurring two months after delivery.

**Management** of heart failure in the context of thyroid storm necessitates simultaneous treatment of both the cardiac and endocrine abnormalities.

#### Pathophysiology of HF in untreated thyrotoxicosis

Uncontrolled hyperthyroidism leads to systemic hypermetabolism and excessive sympathetic nervous system activity.

#### Vasodilation and Hemodynamic Changes

T3 decreases peripheral vascular leading to reduced mean arterial pressure and compensatory increase in cardiac output.

#### Cardiac Output and Volume Overload

Increased preload and heart rate, along with enhanced myocardial contractility, contribute to elevated cardiac output.

#### Myocardial Toxicity and Energetic Imbalance

Excess thyroid hormone disrupt cardiomyocyte energy metabolism by altering oxidative phosphorylation and glycolysis, impair intracellular protein synthesis, and compromise myofibril contractile function.

#### Structural and Electrical Remodelling

Prolonged exposure to high thyroid hormone levels can induce left ventricular hypertrophy and promote arrhythmias.

HF often begins as high-output heart failure and may progress to low-output failure with systolic dysfunction if hyperthyroidism remains untreated.

## Conclusion

Graves’-induced cardiomyopathy must be considered in postpartum heart failure.

This case highlights the need for close monitoring, treatment adherence, and multidisciplinary care.

Pre-conception counselling is vital in patients with known thyroid disorders to prevent

## Acknowledgements & Reference

Patient consent taken for presentation.

<https://doi.org/10.17925/EE.2023.19.1.78>

DOI: 10.15761/IMM.1000350

<https://doi.org/10.1186/s40842-018-0054-7>

<https://doi.org/10.12998/wjcc.v7.i19.2953>

