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CASE REPORT

Postpartum Thyroiditis Complicated by Peripartum Cardiomyopathy and Right Ventricular Thrombi: A Case Report from Rural Uganda

Mughanda Mugheni Olive[1], Kusemererwa Byaruhanga[1], Nsimire Mulanga Roseline[1], Thomas Kwesiga[1], Ernest Badako Mogonza[1], Bigabwa Baharanyi Dominique[1], Venance Emmanuel Mswelo[1], Hildah Kirabo Nlumansi[1], Charles Malisaba Posite[2,3]

[1]Department of Internal Medicine, Kampala International University, Kampala, Uganda; [2]Department of Pathology, Kampala International University, Kampala, Uganda; [3]Faculty of Medicine, Université Catholique du Graben, Butembo, Democratic Republic of the Congo

Correspondence: Mughanda Mugheni Olive, Department of Internal Medicine, Kampala International University, Kampala, Uganda, Email olivemughanda50@gmail.com

Background: Postpartum thyroiditis (PPT) and peripartum cardiomyopathy (PPCM) are important postpartum conditions, but their coexistence is rare. Thyrotoxicosis can increase myocardial stress and precipitate heart failure, particularly in resource-limited settings where diagnosis may be delayed.

Case Presentation: A 30-year-old woman presented two months postpartum with thyrotoxic symptoms and was diagnosed with postpartum thyroiditis. Poor adherence to therapy led to progressive dyspnea and edema. Six months postpartum, she re-presented with decompensated heart failure. Chest X-ray showed cardiomegaly. Echocardiography revealed a dilated left ventricle (LVEDD 6.7 cm, LVESD 5.9 cm) with severely reduced systolic function (LVEF ~23%). The right ventricle was dilated with preserved function (TAPSE 1.7 cm). Mild pulmonary hypertension (RVSP 36 mmHg) and multiple right ventricular apical thrombi (n=3) were identified. She was diagnosed with peripartum cardiomyopathy and treated with guideline-directed heart failure therapy, anticoagulation, bromocriptine, and antithyroid medication, with significant clinical improvement.

Conclusion: This case highlights the importance of early echocardiography, integrated endocrine–cardiac evaluation, and close postpartum follow-up in women presenting with unexplained dyspnea.

Keywords: hyperthyroidism, heart failure, cardiomyopathy, ventricular thrombi, postpartum, rural Uganda

Background

Postpartum thyroiditis (PPT) is an autoimmune thyroid disorder that typically presents within the first year after delivery, often with transient hyperthyroid and/or hypothyroid phases. Its global prevalence is estimated at 5–10% but ranges from 1% to over 11% depending on iodine status, thyroid antibody prevalence, and access to screening.[1,2] In high-income countries, PPT is usually recognised early through routine follow-up. In contrast, in low- and middle-income countries (LMICs), underdiagnosis is common due to limited laboratory capacity and persistent iodine insufficiency during pregnancy.[3,4] In Uganda, specific prevalence data are lacking.

Peripartum cardiomyopathy (PPCM) is an idiopathic form of systolic heart failure occurring in late pregnancy or the postpartum period. Its incidence is relatively low in high-income countries (1 in 1000–4000 live births) but significantly higher in sub-Saharan Africa, reaching up to 1 in 100 live births in some reports, with poorer outcomes due to delayed presentation and limited access to diagnostic and therapeutic resources.[5–8] In Uganda, PPCM accounts for a substantial proportion of acute heart failure in women, with low rates of ventricular recovery.[9]

Thyrotoxicosis exerts significant cardiovascular effects, including increased heart rate, myocardial contractility, and oxygen consumption, which may lead to arrhythmias, high-output heart failure, and, in severe cases, dilated

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cardiomyopathy. Persistent excess thyroid hormone can also impair ventricular function and exacerbate underlying myocardial disease.[10–12]

The coexistence of postpartum thyroiditis (PPT) and peripartum cardiomyopathy (PPCM) is clinically important because thyrotoxicosis may act as a precipitating or aggravating factor for myocardial dysfunction in susceptible individuals. This interaction may accelerate the progression of heart failure and increase the risk of complications such as intracardiac thrombus formation. Despite this, the overlap between these two conditions remains poorly described in the literature, particularly in resource-limited settings, where delayed diagnosis is common.[11,12]

This dual burden highlights the need for early recognition, multidisciplinary care, and improved postpartum surveillance in Uganda and other resource-limited settings.

Case Presentation

A 30-year-old woman (gravida 3, para 3, abortus 0), with no known history of chronic illness, presented in March 2025, two months following an uncomplicated delivery, with a four-week history of progressively worsening exertional dyspnea and easy fatigability. She also reported paroxysmal nocturnal dyspnea, bilateral lower limb edema, abdominal distension, anorexia, recurrent non-bilious, non-bloody vomiting, diarrhea, and an unintentional weight loss of approximately 10 kg over one month. Additional symptoms included headache, palpitations, and a productive cough with whitish sputum.

On examination, the patient appeared cachectic and was tachycardic, with a pulse rate of 136 beats per minute. There were no signs of adrenergic overactivity such as tremor, excessive sweating, heat intolerance, or proximal muscle weakness. Mild, non-prominent exophthalmos was noted.

The thyroid gland was diffusely enlarged, smooth, soft in consistency, non-tender, and mobile on swallowing, with no associated bruit, retrosternal extension, or cervical lymphadenopathy (Figure 1).

Cardiovascular examination revealed a laterally displaced apex beat located at the sixth intercostal space along the anterior axillary line.

Initial investigations included a thyroid ultrasound, which demonstrated bilateral echo-complex masses with cystic areas. Thyroid function tests revealed suppressed thyroid-stimulating hormone (TSH 0.00 μIU/mL) with elevated free triiodothyronine (T_3 8.6 pmol/L) and thyroxine (T_4 21.1 pmol/L), consistent with thyrotoxicosis. Electrocardiography showed sinus tachycardia (Figure 2). Other laboratory investigations, including full blood count, renal function tests, and serum electrolytes, were within normal limits.

A diagnosis of postpartum thyroiditis was made. She was initiated on oral propranolol 40 mg twice daily for symptomatic control, carbimazole 15 mg twice daily for thyrotoxicosis, furosemide 40 mg once daily for fluid

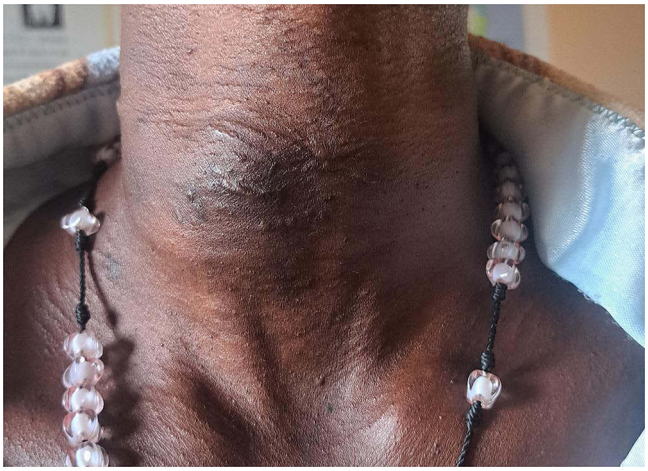

Figure 1 Diffuse enlargement of the thyroid gland observed on clinical examination, consistent with postpartum thyroiditis.

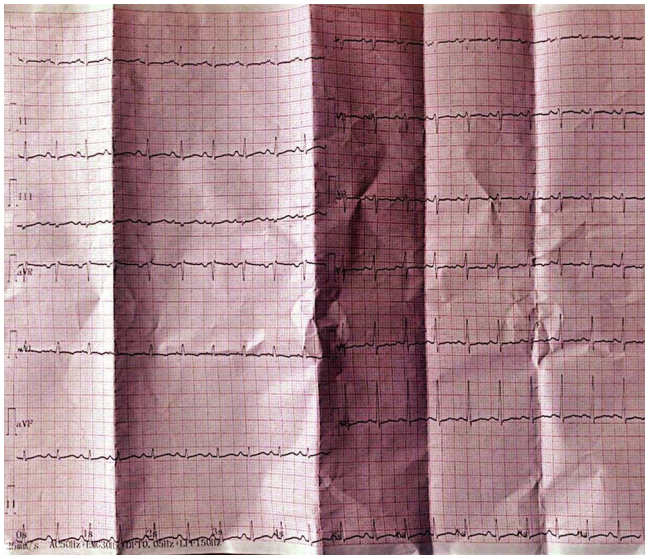

Figure 2 Electrocardiogram film showing sinus tachycardia.

management, and paracetamol 1 g three times daily as needed. Although partial clinical improvement was achieved, adherence to treatment was poor.

In September 2025, she re-presented with worsening dyspnea, orthopnea, paroxysmal nocturnal dyspnea, and generalised oedema. On examination, she was tachypneic (respiratory rate 28 breaths per minute) and tachycardic (pulse rate 130 beats per minute); the blood pressure was 104/78 millimeter of mercury and the oxygen saturation at 96% on room air, with a displaced apex beat and parasternal heave suggestive of cardiac enlargement.

The investigations showed cardiomegaly on chest X-ray (cardiothoracic ratio 0.65) with increased pulmonary vascular markings and a small left-sided pleural effusion (Figures 3 and 4). Echocardiography demonstrated a markedly dilated left ventricle with a left ventricular end-diastolic diameter (LVEDD) of 6.7 cm and end-systolic diameter (LVESD) of 5.9 cm. Left ventricular systolic function was severely reduced, with an estimated left ventricular ejection fraction (LVEF) of approximately 23% and fractional shortening of 11%. The left atrium was mildly enlarged (4.0 cm; left atrial volume 49 cm^3). The right ventricle was dilated (RV diameter 5.0 cm) but maintained preserved

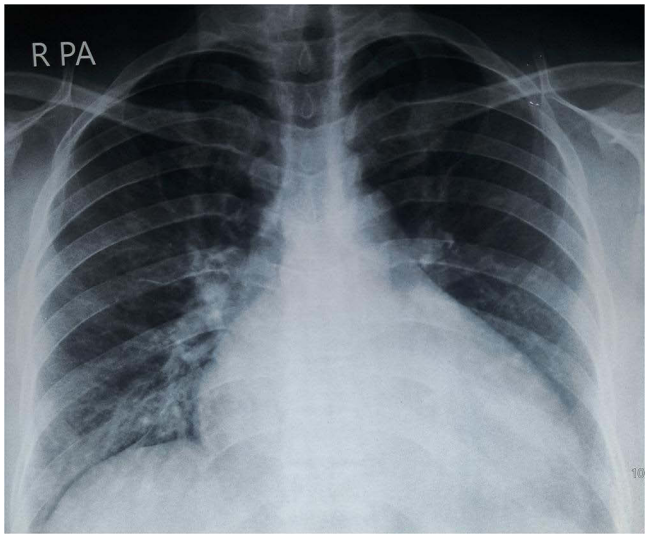

Figure 3 Chest Xray PA (initial presentation) demonstrating cardiomegaly with increased pulmonary vascular markings with left-sided pleural effusion (with R = right side and PA = postero-anterior view).

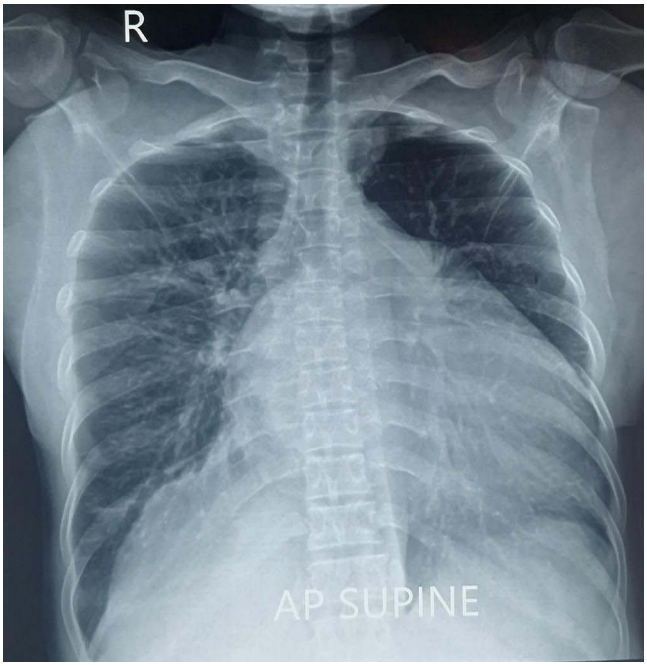

Figure 4 Chest X-ray on the 3rd visit. Chest X-ray (follow-up) showing persistent cardiomegaly (with R = right side and AP = anteroposterior view).

systolic function, with a tricuspid annular plane systolic excursion (TAPSE) of 1.7 cm. Mild pulmonary hypertension was present, with an estimated right ventricular systolic pressure (RVSP) of 36 mmHg, calculated from a tricuspid regurgitation gradient of 26 mmHg and an estimated right atrial pressure of 10 mmHg.

Diastolic function assessment showed an E/A ratio of 1.5 and an elevated E/E′ ratio of 23, indicating increased left ventricular filling pressures. Additionally, moderate mitral regurgitation and mild tricuspid regurgitation were noted. Multiple right ventricular apical thrombi (n=3) were visualised, the largest measuring 3.17×2.18 cm (Figures 5 and 6).

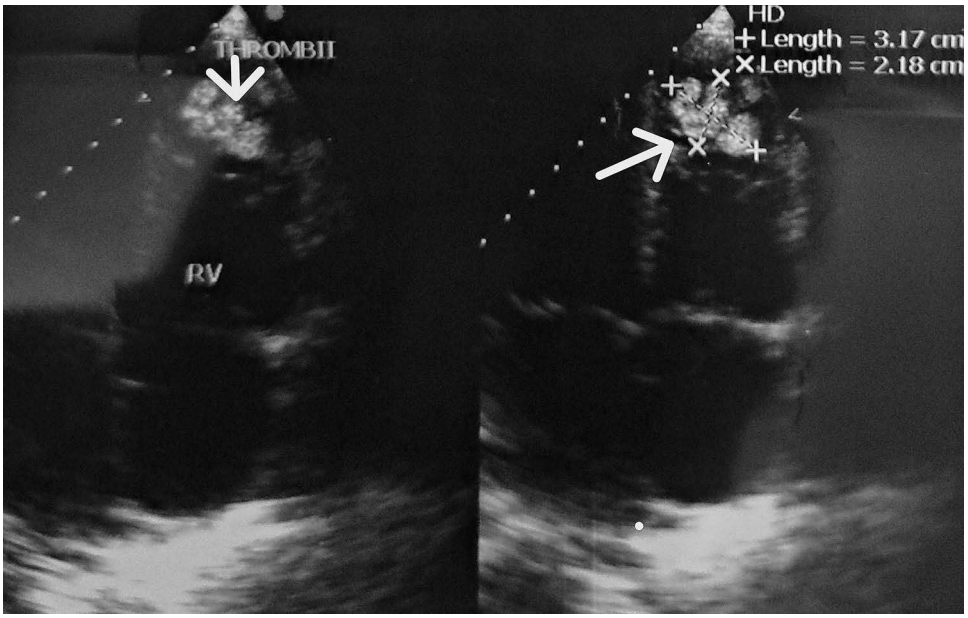

Figure 5 Transthoracic echocardiography showing right ventricular apical thrombi (largest measuring 3.17×2.18 cm) (arrow showing the biggest thrombi, RV = Right Ventricle).

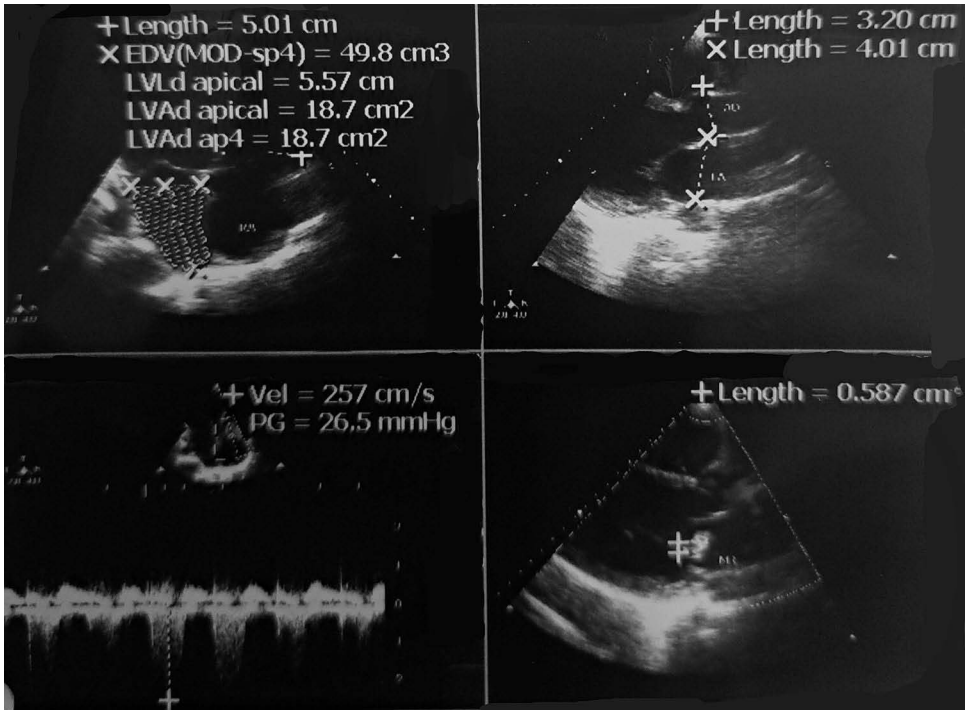

Figure 6 Transthoracic echocardiography demonstrating a dilated left ventricle with severely reduced systolic function.

A diagnosis of peripartum cardiomyopathy complicated by intracardiac thrombi was made. At presentation, the patient was classified as New York Heart Association (NYHA) functional class III. She was admitted and managed with intravenous furosemide 40 mg twice daily, oral enalapril 2.5 mg twice daily, carvedilol 3.125 mg twice daily, and dapagliflozin 10 mg once daily as part of guideline-directed heart failure therapy. Anticoagulation began with subcutaneous enoxaparin at a dosage of 1 mg/kg twice daily and was subsequently switched to oral apixaban at a dosage of 5 mg twice daily. Bromocriptine 2.5 mg twice daily was added as adjunct therapy, and carbimazole 15 mg twice daily was continued.

By day 4 of admission, her dyspnea and cough resolved, and her appetite improved, though mild fatigue and pedal oedema persisted. She was discharged with significant improvement and scheduled for follow-up.

Two weeks after discharge, she was reviewed with significant clinical improvement.

Discussion

It has been well documented that in developing countries, many diagnoses are established at late stages of the disease, often due to limited access to diagnostic tools, delayed health-seeking behaviour, and resource constraints.[3] In the present case poor compliance on treatment, explains the worsening condition.

Although both postpartum thyroiditis (PPT) and peripartum cardiomyopathy (PPCM) are recognised postpartum complications, their coexistence is exceedingly rare and clinically challenging. Thyroid hormones have profound effects on the cardiovascular system, and thyrotoxicosis can exacerbate or precipitate heart failure by increasing myocardial oxygen demand, heart rate, and contractility.[10,11] Case reports and small series describe instances where thyroid dysfunction coexisted with or unmasked PPCM, suggesting that hyperthyroidism may act as a trigger or aggravating factor for myocardial dysfunction in genetically or physiologically predisposed women.[11,12]

The initial clinical focus was on postpartum thyroiditis (PPT), supported by the patient's hyperthyroid symptoms and biochemical findings. This diagnosis was reasonable and aligns with endocrinology literature, which describes PPT as a destructive thyroiditis typically occurring in the first 1–4 months after delivery, often presenting with a transient thyrotoxic phase followed by hypothyroidism or recovery.[13] In most cases, anti-thyroid peroxidase (TPO) antibodies are

positive, confirming its autoimmune basis (Sara Naji Rad & Linda Deluxe, 2023).[14] Due to feasibility in our setting this test was not done.

However, the patient's more cardinal heart failure signs (progressive dyspnea, edema, PND) should have raised suspicion for peripartum cardiomyopathy (PPCM) earlier, especially given that thyroid dysfunction alone is unlikely to account for the degree of left ventricular dysfunction and intracardiac thrombi seen later. The updated review, titled "Diagnosis and management of peripartum cardiomyopathy" outlines the need for early echocardiography when unexplained heart failure arises in the peripartum period (ie, within months after delivery).[15]

PPCM is considered a diagnosis of exclusion, but prompt recognition is critical: guidelines recommend considering PPCM in any woman who develops heart failure with reduced ejection fraction (LVEF <45%) in late pregnancy or postpartum.[16] Moreover, the eMedicine Medscape guidance (2025) emphasizes the importance of early echocardiography and baseline cardiac assessment in postpartum women with symptoms beyond usual postpartum fatigue or dyspnea (which can be physiological) (Stephanie G Braunthal et al, 2025).[17]

In similar reported cases, delayed diagnosis is common, especially in resource-limited settings, and is a major contributor to adverse outcomes.[18] In the present case, the cardiomyopathy was recognised only once the disease had progressed to advanced heart failure and thrombus formation. Earlier integration of cardiac imaging in the assessment might have changed her trajectory.

In the present case, thyrotoxicosis likely contributed to cardiac stress and accelerated the onset of PPCM. This relationship has been echoed in the literature—patients with thyrotoxic cardiomyopathy can present with reversible heart failure, and persistent thyrotoxicosis may worsen recovery in PP.[12,19,20] Moreover, both disorders share immune and hormonal pathways, particularly involving autoimmune activation, oxidative stress, and prolactin metabolism, suggesting overlapping mechanisms in susceptible individuals.[14,21,22]

The development of intracardiac thrombi in PPCM is multifactorial and can be explained by Virchow's triad: reduced left ventricular systolic function leading to blood stasis, endothelial dysfunction, and the hypercoagulable state of the postpartum period. These mechanisms are well recognized contributors to thromboembolic complications in PPCM.[16,18]

Accordingly, anticoagulation plays a critical role in management. Current evidence supports the use of therapeutic anticoagulation in patients with severe left ventricular dysfunction or documented intracardiac thrombi to reduce the risk of systemic embolization and improve outcomes.[16,21] In this case, early initiation of weight-adjusted anticoagulation followed by oral therapy was associated with favorable clinical evolution, underscoring the importance of prompt recognition and intervention.

This case report has several limitations. First, serial echocardiographic assessments were not performed, limiting evaluation of ventricular recovery and thrombus resolution over time. Second, comprehensive cardiac biomarkers such as B-type natriuretic peptide (BNP or NT-proBNP) were not available, which could have provided additional objective assessment of heart failure severity. Third, pulmonary artery systolic pressure was estimated non-invasively without confirmatory haemodynamic measurements. Additionally, repeat thyroid function tests and thyroid autoantibody assays were not performed due to resource constraints, limiting further characterisation of the thyroid disorder. Finally, as a single case report, the findings cannot be generalised but provide important clinical insight into a rare and potentially severe postpartum condition in a low-resource setting.

Conclusion

This case illustrates the rare but clinically significant coexistence of postpartum thyroiditis and peripartum cardiomyopathy, conditions that can mutually worsen maternal outcomes when not promptly recognised. Thyrotoxicosis likely contributed to increased cardiac stress, accelerating heart failure progression. Delayed diagnosis and poor treatment adherence further complicated the clinical course. Early echocardiography, integrated endocrine–cardiac evaluation, and close postpartum follow-up are essential for timely detection and management of such overlapping conditions, especially in resource-limited settings. This case reinforces the need for heightened vigilance when postpartum women present with persistent dyspnea or fatigue beyond expected physiological recovery.

Ethics Approval and Consent to Participate

Institutional ethical approval was not required for this case report according to the guidelines of Kampala International University. Approval for publication of the case details was also not required under institutional policy. One written informed consent for publication was obtained from the patient.

Informed Consent

One written, informed consent was obtained from the patient for the publication of this case report and any accompanying images.

Author Contributions

All authors made a significant contribution to the work reported, whether that was in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

The authors declare that they have no conflicts of interest in this work.

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