EsSalud
Archivos Peruanos de Cardiología y Cirugía Cardiovascular
Arch Peru Cardiol Cir Cardiovasc. 2026;7(3). doi: 10.47487/apcyccv.v7i3.581
Instituto Nacional Cardiovascular

Case report

Peripartum cardiomyopathy and antiphospholipid syndrome: a rare association revealed by a right ventricular thrombus. A case report

Alexis Vallejos-Barrientos[1,a], **Diego Davila-Flores**[1,b], **Ofelia Araoz-Tarco**[1,b]

Received: December 1, 2025
Accepted: July 26, 2026
Online: August 5, 2026

Authors' affiliation
[1] Instituto Nacional Cardiovascular, INCOR-EsSalud, Lima, Peru.
[a] Cardiology resident.
[b] Cardiologist.

Correspondence
Alexis Vallejos-Barrientos.
Av. Coronel Zegarra 417.
Jesús María, Lima, Peru.

Email
cardiologiaconalexis@gmail.com

Source of funding
Self-funded.

Conflicts of interest
None.

Cite as
Vallejos-Barrientos A, Davila-Flores D, Araoz-Tarco O. Peripartum cardiomyopathy and antiphospholipid syndrome: a rare association revealed by a right ventricular thrombus. A case report. Arch Peru Cardiol Cir Cardiovasc. 2026;7(3). doi: 10.47487/apcyccv.v7i3.581.

ABSTRACT

Peripartum cardiomyopathy (PPCM) is uncommon, and its association with antiphospholipid syndrome (APS) and right ventricular (RV) thrombus is exceptional. We report a 33-year-old woman who, three months after cesarean delivery for severe preeclampsia, developed acute heart failure with severe biventricular dysfunction requiring mechanical ventilation. An RV thrombus was identified; two computed tomographic pulmonary angiograms, one week apart, were negative for pulmonary embolism. Thrombophilia work-up disclosed persistently positive lupus anticoagulant on repeat testing 12 weeks apart, supporting APS. Therapeutic anticoagulation led to complete thrombus resolution after 10 days, and optimized heart failure therapy was followed by hemodynamic improvement and successful extubation. Coronary angiography ruled out ischemic heart disease, and endomyocardial biopsy showed no histologic evidence of myocarditis, supporting PPCM. Myocardial recovery was partial, requiring follow-up within an advanced heart failure program. This case highlights the need to evaluate secondary prothrombotic conditions, particularly APS, and to institute early anticoagulation in PPCM with intracardiac thrombosis.

Keywords: Peripartum Cardiomyopathy; Thrombosis; Antiphospholipid Syndrome (Source: MeSH-NLM).

RESUMEN

Miocardiopatía periparto y síndrome antifosfolipídico: una infrecuente asociación revelada por un trombo en ventrículo derecho. Reporte de caso

La miocardiopatía periparto (PPCM) es infrecuente y su asociación con el síndrome antifosfolipídico (SAF) y trombo en el ventrículo derecho (VD) es excepcional. Presentamos el caso de una mujer de 33 años quien, tres meses después de una cesárea indicada por preeclampsia severa, presentó insuficiencia cardíaca aguda con disfunción biventricular grave, que requirió ventilación mecánica invasiva. Se identificó un trombo en el VD; para descartar tromboembolismo pulmonar, se realizaron dos angiotomografías pulmonares con un intervalo de una semana, ambas negativas. Se realizó un estudio de trombofilias, diagnosticándose SAF. Se instauró anticoagulación terapéutica, con resolución del trombo a los 10 días. La coronariografía descartó cardiopatía isquémica y la biopsia endomiocárdica no encontró evidencia de miocarditis; el diagnóstico fue PPCM. La recuperación miocárdica fue parcial, requiriendo seguimiento en el programa de falla cardíaca avanzada. Este caso subraya la importancia de la evaluación sistemática de SAF y la pronta anticoagulación en PPCM con trombosis intracardiaca.

Palabras clave: Cardiopatía Periparto; Trombosis; Síndrome Antifosfolípido (Fuente: DeCS-BIREME).

Introduction

Peripartum cardiomyopathy (PPCM) is a pregnancy-associated cardiomyopathy characterized by new-onset heart failure near term or within 5 months postpartum, the absence of an alternative cause, and left ventricular (LV) systolic dysfunction, with an LV ejection fraction (LVEF) <45%, with or without LV dilation. Although thrombotic complications are recognized, isolated right ventricular (RV) thrombus is rarely reported. [1,2]

PPCM occurs across all ethnic groups, with a mean presentation age of 31 years and a mean parity of three. The global incidence is estimated at 1 in 2000–4000 live births, with higher rates in Haiti and Nigeria. [3] The incidence appears to be increasing, partly driven by advanced maternal age and more multifetal gestations related to assisted reproductive technologies. [3,4]

Risk factors include African ancestry, preeclampsia, cocaine use, and prolonged β-agonist tocolysis. Up to 15% of cases harbor pathogenic variants, most commonly truncating titin mutations. [5,6] PPCM carries substantial morbidity, including pulmonary edema, thromboembolism, and cardiogenic shock, with a mortality rate of 7–20%. Approximately 44% of patients recover LV function within 6 months, and nearly recover 59% within 12 months. [2,7] Pregnancy-related hypercoagulability, severe ventricular dysfunction, and antiphospholipid syndrome (APS) may synergistically promote intracardiac thrombosis in PPCM. [3,8]

We report a woman 3 months postpartum with severe PPCM complicated by an isolated RV thrombus and concomitant APS, an association that is rarely described.

Case report

A 33-year-old woman with class I obesity, two previous uncomplicated term pregnancies, and no history of pregnancy loss or familial cardiomyopathy underwent emergency cesarean delivery at 33 weeks and 5 days for severe preeclampsia. She was discharged on antihypertensive therapy. Three months postpartum, she developed progressive exertional dyspnea, orthopnea, and peripheral edema, without fever, respiratory prodrome, or gastroenteritis. She initially received diuretics at another institution, without echocardiographic evaluation. She subsequently deteriorated and presented with New York Heart Association (NYHA) functional class IV dyspnea and acute cardiogenic pulmonary edema requiring mechanical ventilation.

On transfer, she had congestion with hypoperfusion, prompting intravenous furosemide escalation and dobutamine initiation. Laboratory abnormalities included hemoglobin 8.8 g/dL, neutrophilic leukocytosis, and an N-terminal pro-B-type natriuretic peptide (NT-proBNP) concentration of 3715 pg/mL. Chest radiography showed pulmonary congestion, and electrocardiography demonstrated LV hypertrophy **(Figure 1)**. Transthoracic echocardiography (TTE) revealed severe global biventricular systolic dysfunction **(Figure 2)**.

Because of persistent hemodynamic instability, right-heart catheterization was performed 48 hours after transfer. Hemodynamic measurements—obtained during inotropic infusion, sinus tachycardia, moderate anemia, and evolving community-acquired pneumonia–related sepsis—were as follows: right atrial pressure 13 mmHg; pulmonary artery

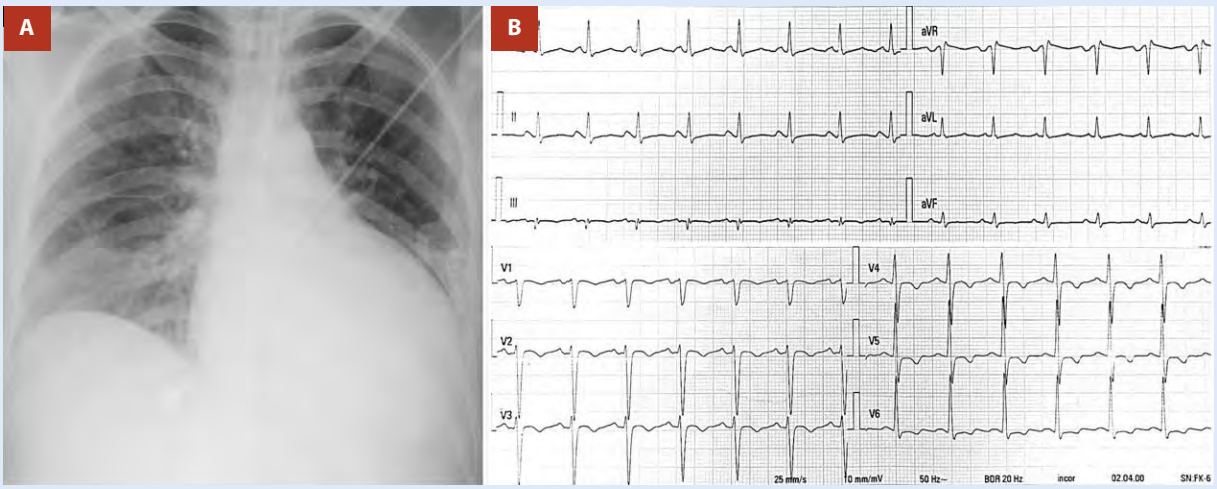

Figure 1. Baseline chest radiograph and 12-lead electrocardiogram. **A.** Portable anteroposterior chest radiograph demonstrating cardiomegaly with a globular cardiac silhouette and diffuse pulmonary vascular congestion, with bilateral perihilar interstitial opacities consistent with cardiogenic pulmonary edema. **B.** Twelve-lead electrocardiogram showing sinus tachycardia, electrocardiographic criteria for left-atrial enlargement (broad, notched P waves in limb leads), and voltage criteria for left-ventricular hypertrophy with secondary ST–T repolarization abnormalities in the lateral precordial leads. Precordial leads V1–V3 display relatively low-amplitude R waves with prominent S waves, yielding an overall pattern compatible with biventricular pressure overload in the setting of advanced cardiomyopathy.

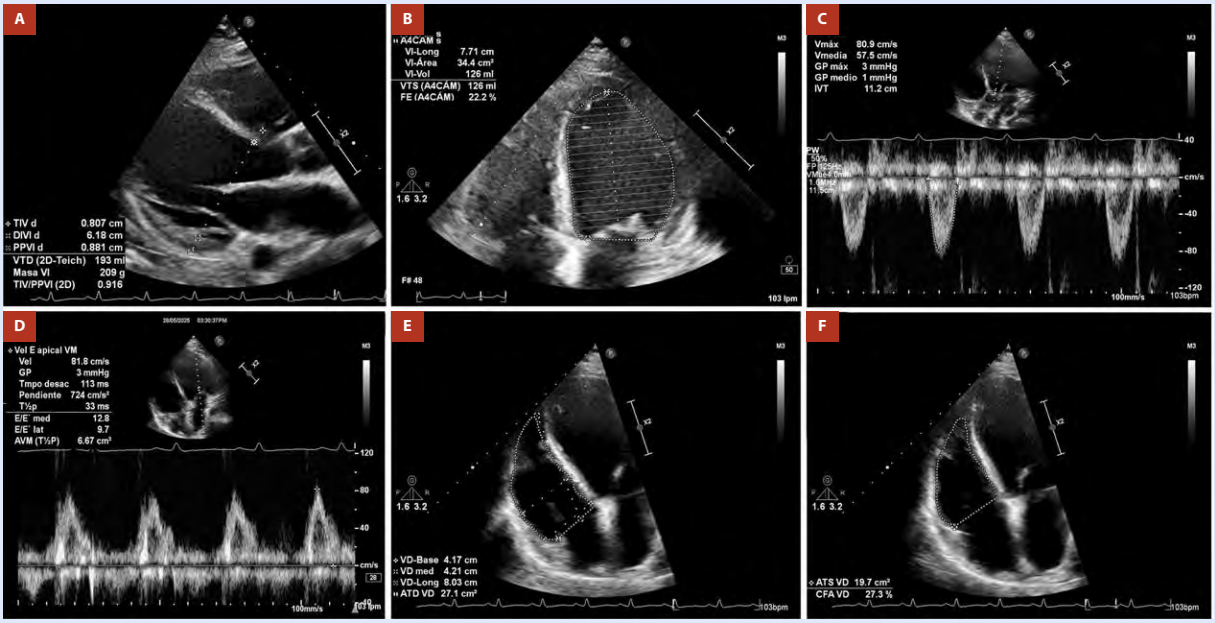

Figure 2. Transthoracic echocardiography demonstrates severe biventricular systolic dysfunction. **A.** Parasternal long-axis view showing a markedly dilated LV (end-diastolic diameter 61 mm). **B, C.** Apical four-chamber view demonstrating severe global LV systolic dysfunction; biplane Simpson LVEF was 22%. **D.** Pulsed-wave Doppler showing a reduced LVOT VTI: 11 cm, consistent with low forward stroke volume in the context of advanced HF. **E.** RV-focused apical four-chamber view showing RV dilatation. **F.** RV FAC reduced to 27%, confirming significant RV systolic dysfunction.

HF: heart failure; **LV:** left ventricle; **LVEF:** left ventricular ejection fraction; **LVOT VTI:** left ventricular outflow tract velocity–time integral; **RV:** right ventricle; **RV FAC:** right ventricular fractional area change.

pressure 38/23 mmHg (mean 29 mmHg); pulmonary capillary wedge pressure 22 mmHg; mean arterial pressure 70 mmHg; heart rate 130 beats per minute; cardiac output 7.0 L/min (cardiac index 4.0 L/min/m^2); pulmonary vascular resistance 80 dyn·s·cm^{-5}; and systemic vascular resistance 650 dyn·s·cm^{-5}. This pattern was consistent with post-capillary pulmonary hypertension and markedly elevated biventricular filling pressures in the setting of sepsis and inotrope-driven low systemic vascular resistance, rather than her baseline hemodynamic state. After inotropic withdrawal, diuretic and vasodilator therapy were carefully titrated.

Repeat TTE 7 days after admission identified an RV thrombus, subsequently confirmed by cardiac computed tomography (CCT). Anticoagulation with unfractionated heparin was initiated. Serial computed tomography pulmonary angiography (CTPA) studies ruled out pulmonary embolism **(Figure 3)**. Lower-limb Doppler ultrasonography excluded deep vein thrombosis, supporting an isolated intracardiac thrombotic process rather than an embolus in transit from the peripheral venous system. A broader etiologic work-up excluded concomitant systemic autoimmune disease, malignancy, and infectious causes. The antiphospholipid antibody profile showed isolated lupus anticoagulant positivity, persistently positive on repeat testing 12 weeks apart. Together with the objectively documented RV thrombus, these findings fulfilled the revised Sydney classification criteria and supported the clinical diagnosis of thrombotic APS. In addition, preterm delivery before 34 weeks' gestation because of severe preeclampsia was consistent with the contemporary American College of Rheumatology/European Alliance of Associations for Rheumatology (ACR/EULAR) obstetric APS classification framework. The diagnosis was validated by the institutional hematology service, and long-term warfarin anticoagulation was instituted, targeting an international normalized ratio (INR) of 2.0–3.0.

After 10 days of therapeutic anticoagulation, TTE confirmed complete RV thrombus resolution, and the patient was successfully extubated while guideline-directed medical therapy was optimized. Coronary angiography demonstrated angiographically normal epicardial coronary arteries **(Figure 4)**. Repeat right-heart catheterization showed normal right-sided filling pressures and pulmonary hemodynamics, with a right atrial pressure of 5 mmHg, pulmonary artery pressure of 21/10 mmHg (mean 15 mmHg), pulmonary capillary wedge pressure of 9 mmHg, mean arterial pressure of 90 mmHg, cardiac index of 2.2 L/min/m^2, and pulmonary vascular resistance of 144 dyn·s·cm^{-5}. Endomyocardial biopsy (EMB) excluded myocarditis and infiltrative cardiomyopathies **(Figure 5)**. The patient was discharged after 2 weeks; TTE showed modest LVEF improvement to 27% with RV systolic recovery. During advanced heart failure follow-up, she remained symptomatic despite progressive optimization of standard heart failure therapy. Intermittent levosimendan was used as an individualized advanced heart failure strategy and was associated with clinical stabilization, allowing further optimization of guideline-directed medical therapy, including a beta-blocker, sacubitril/valsartan,

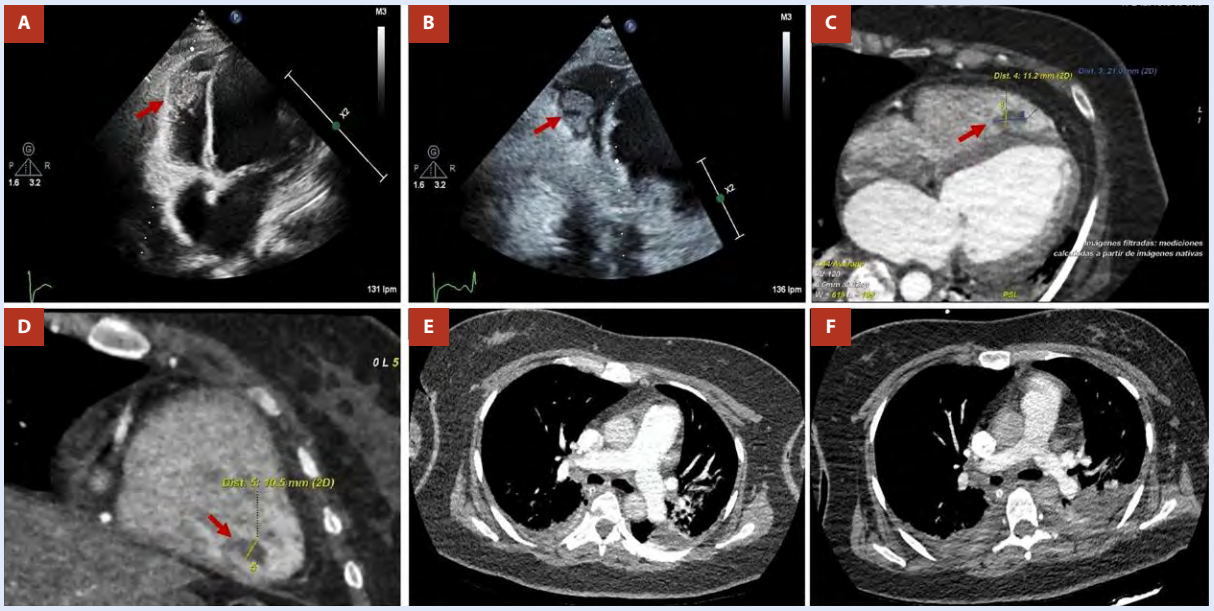

Figure 3. Multimodality imaging demonstrates an isolated right ventricular thrombus and excludes pulmonary embolism. **A–B.** Transthoracic echocardiography, parasternal long-axis (A) and parasternal short-axis mid-ventricular views (B), demonstrating a mobile, echodense, RV intracavitary mass (red arrows) consistent with thrombus, anchored to the RV free-wall trabeculations. **C–D.** Cardiac computed tomography confirming a well-defined intraluminal thrombus within the RV cavity (red arrows), measuring 11 × 21 mm in orthogonal planes. The mass exhibits homogeneously low attenuation, sharply demarcated from the endocardial border, supporting the diagnosis of thrombus rather than tumor. **E.** Baseline CTPA, performed the same day as the echocardiographic identification of the RV thrombus, demonstrating patent pulmonary arteries without filling defects. **F.** Follow-up CTPA performed one week later, again showing no pulmonary artery thromboembolism.

CTPA: computed tomographic pulmonary angiography; **RV:** right ventricle.

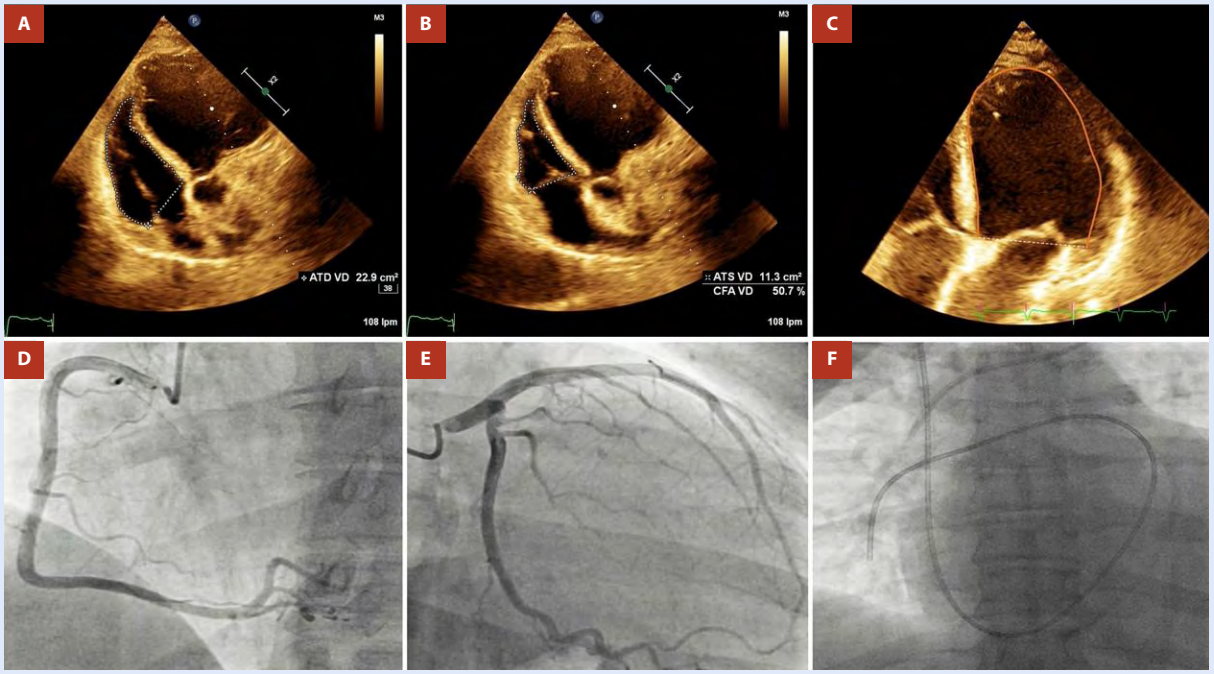

Figure 4. Follow-up multimodality imaging after anticoagulation and hemodynamic stabilization. **A.** Follow-up transthoracic echocardiography (apical four-chamber view) performed 10 days after initiation of full anticoagulation demonstrates complete resolution of the previously visualized RV thrombus. **B.** Apical four-chamber view showing significant improvement in RV FAC of 50.7%, compared with severely depressed function at baseline. **C.** Left ventricular-focused apical four-chamber view used to calculate left ventricular ejection fraction, which was 27%. **D–E.** Invasive coronary angiography demonstrating angiographically normal coronary arteries in both right and left coronary systems, effectively excluding obstructive epicardial coronary disease as a contributor to the ventricular dysfunction. **F.** Posteroanterior fluoroscopic projection demonstrating a Swan–Ganz catheter in appropriate position for right-heart hemodynamic assessment.

LVEF: left ventricular ejection fraction; **RV:** right ventricle; **RV FAC:** right ventricular fractional area change.

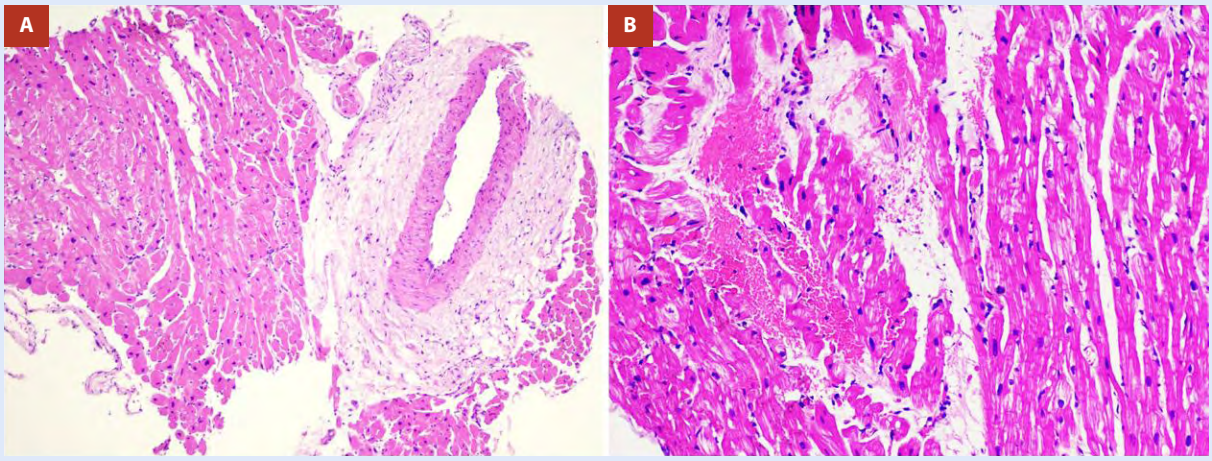

Figure 5. Histopathologic analysis of endomyocardial biopsy. **A.** Hematoxylin–eosin–stained section showing preserved myocardial architecture with normocellular interstitium, small intramyocardial arteries without vasculitis, fibrinoid necrosis, or luminal thrombosis, and only mild interstitial edema; scattered extravasated erythrocytes are interpreted as procedural artifact. **B.** Higher-power view confirming absence of myocyte necrosis, inflammatory infiltrates, granulomas, or storage material. Immunohistochemistry (not shown) was negative for inflammatory, viral, or infiltrative cardiomyopathy, and there was no evidence of amyloid deposition, supporting a non-inflammatory, non-infiltrative dilated cardiomyopathy consistent with peripartum cardiomyopathy.

spironolactone, and a sodium–glucose cotransporter-2 inhibitor. In confirmed APS, lifelong warfarin was maintained, and at 6 months post-discharge, she remained clinically stable in NYHA functional class II.

Discussion

PPCM complicated by an isolated RV thrombus in the setting of APS is exceptionally uncommon. This patient presented with severe biventricular heart failure and acute cardiogenic pulmonary edema, illustrating a high-risk and diagnostically challenging presentation of PPCM.

Adverse prognosis in PPCM is associated with reduced LVEF, late presentation, late gadolinium enhancement on cardiovascular magnetic resonance (CMR), LV dilatation, and RV dysfunction. [2,7] Although preeclampsia has been associated with a greater likelihood of LV recovery, it also confers higher cardiovascular morbidity. [9] This patient had three established risk factors—preeclampsia, multiparity, and age >30 years—and several adverse predictors, including reduced LVEF, delayed presentation, LV dilatation, and RV dysfunction. [2,10]

PPCM likely reflects angiogenic dysregulation, prolactin-mediated cardiomyocyte injury, pregnancy-related hemodynamic stress in genetically susceptible women, and immune-inflammatory activation. [10,11] Myocarditis has also been proposed as a contributor, but evidence remains limited and heterogeneous; in our patient, EMB showed no histologic evidence of myocarditis. [12]

In a cohort of 123 patients with PPCM, intracardiac thrombus was identified in 22 (17.9%), predominantly in the LV (86.4%); RV thrombus occurred in 4.5%, and biventricular thrombi occurred in 9.1%. Lower LVEF, anemia, and thrombocytosis were independent predictors of LV thrombus. [13] PPCM promotes thrombosis through endothelial injury, LV stasis, and pregnancy-related hypercoagulability. Thrombi may also occur in the atria, aorta, pulmonary artery, or vena cava, and embolic events are reported in 5–9%, mainly ischemic stroke and pulmonary embolism. [14,15] In this case, an isolated RV thrombus prompted systematic evaluation for prothrombotic conditions and led to an APS diagnosis. Coexisting APS and PPCM may further amplify endothelial dysfunction and hypercoagulability. [8,16]

TTE in PPCM typically shows marked LV systolic dysfunction with LV dilatation; left atrial enlargement and RV dilatation may also occur, whereas significant mitral or tricuspid regurgitation is less frequent. Although CCT is not first-line in PPCM, it is useful when pulmonary embolism, intracardiac thrombus, or coronary artery disease must be excluded. [17] Here, CCT confirmed the RV thrombus and, with serial CTPA studies, excluded pulmonary embolism; the thrombus resolved after 10 days of anticoagulation. Early recognition and therapeutic anticoagulation may have reduced the risk of pulmonary embolization. CMR is recommended when feasible, particularly when TTE is inconclusive or tissue characterization may influence management. [18]

Because PPCM is a diagnosis of exclusion, coronary angiography was performed to exclude an ischemic etiology. [11] EMB is not routinely indicated and is generally reserved for suspected fulminant myocarditis, refractory shock, malignant arrhythmias, or possible infiltrative or metabolic cardiomyopathies. [2,10] In our patient, EMB was justified because myocarditis remained clinically relevant, and persistent severe LV dysfunction suggested potential progression to advanced heart failure.

No disease-specific therapy has improved hard outcomes in adequately powered randomized trials. Bromocriptine

has shown potential benefit in small PPCM studies and is recognized in the 2025 European Society of Cardiology Guidelines as a Class IIb, Level B option that may be considered in addition to optimal heart failure therapy. [3] No guideline-defined postpartum cut-off exists, and available evidence derives mainly from acute PPCM treated early after diagnosis. Bromocriptine is not approved by the United States Food and Drug Administration and is not routinely recommended in American Heart Association/American College of Cardiology guidance. [9,10] Bromocriptine was not administered because the patient presented relatively late after delivery, after the phase in which most supporting studies initiated therapy, and because evidence remains limited. [3,11]

Therapeutic anticoagulation is mandatory with intracavitary thrombus, and many experts also consider it in PPCM with severely reduced LVEF or atrial fibrillation. [3,10] The 2025 European Society of Cardiology Guidelines further recommend counseling on recurrence risk in future pregnancies and contraception for all women with PPCM, even after LV recovery (Class I, Level C). [3] A wearable cardioverter-defibrillator may be considered for selected patients at high arrhythmic risk, whereas left ventricular assist device implantation or heart transplantation is ultimately required in 3%–10% of cases. [11,19]

Limitations include the absence of genetic testing and the inability to perform CMR because of clinical instability. In conclusion, severe PPCM with atypical intracardiac thrombosis should prompt evaluation for secondary prothrombotic conditions, particularly APS, and support early, serial surveillance for intracardiac thrombus in patients with severe biventricular dysfunction.

Ethical Considerations

This case report was approved by the institutional ethics committee. Written informed consent for publication was obtained from the patient.

Authors' contributions

AVB, DDF: conceptualization, investigation, writing – original draft and writing – review & editing. **OAT:** supervision, review, and editing.

References

1. Bauersachs J, König T, van der Meer P, Petrie MC, Hilfiker-Kleiner D, Mbakwem A, *et al.* Pathophysiology, diagnosis and management of peripartum cardiomyopathy: a position statement from the Heart Failure Association of the European Society of Cardiology Study Group on peripartum cardiomyopathy. Eur J Heart Fail. 2019;21(7):827-843. doi: 10.1002/ejhf.1493.
2. Sigauke FR, Ntsinjana H, Tsabedze N. Peripartum cardiomyopathy: a comprehensive and contemporary review. Heart Fail Rev. 2024;29(5):1261-1278. doi: 10.1007/s10741-024-10435-5.
3. De Backer J, Haugaa KH, Hasselberg NE, de Hosson M, Brida M, Castelletti S, *et al.* ESC Scientific Document Group. 2025 ESC Guidelines for the management of cardiovascular disease and pregnancy. Eur Heart J. 2025;46(43):4462-4568. doi: 10.1093/eurheartj/ehaf193.
4. Viljoen C, Hoevelmann J, Sliwa K. Peripartum cardiomyopathy: risk factors and predictors of outcome. Curr Opin Cardiol. 2023;38(3):223-232. doi: 10.1097/HCO.0000000000001037.
5. Sliwa K, Mebazaa A, Hilfiker-Kleiner D, Petrie MC, Maggioni AP, Laroche C, *et al.* Clinical characteristics of patients from the worldwide registry on peripartum cardiomyopathy (PPCM): EURObservational Research Programme in conjunction with the HFA of the ESC Study Group on PPCM. Eur J Heart Fail. 2017;19(9):1131-1141. doi: 10.1002/ejhf.780.
6. Spracklen TF, Chakafana G, Schwartz PJ, Kotta MC, Shaboodien G, Sliwa K, *et al.* Genetics of peripartum cardiomyopathy: current knowledge, future directions and clinical implications. Genes (Basel). 2021;12(1):103. doi: 10.3390/genes12010103.
7. Hoevelmann J, Engel ME, Muller E, Hohlfeld A, Böhm M, Sliwa K, *et al.* A global perspective on the management and outcomes of peripartum cardiomyopathy: a systematic review and meta-analysis. Eur J Heart Fail. 2022;24(9):1719-1736. doi: 10.1002/ejhf.2603.
8. Barbhaiya M, Zuily S, Naden R, Hendry A, Manneville F, Amigo MC, *et al.* The 2023 ACR/EULAR Antiphospholipid Syndrome Classification Criteria. Arthritis Rheumatol. 2023 Oct;75(10):1687-1702. doi: 10.1002/art.42624.
9. McNamara DM, Elkayam U, Alharethi R, Damp J, Hsich E, Ewald G, *et al.* Clinical outcomes for peripartum cardiomyopathy in North America: results of the IPAC study (Investigations of Pregnancy-Associated Cardiomyopathy). J Am Coll Cardiol. 2015;66(8):905-914. doi: 10.1016/j.jacc.2015.06.1309.
10. Davis MB, Arany Z, McNamara DM, Goland S, Elkayam U. Peripartum cardiomyopathy: JACC state-of-the-art review. J Am Coll Cardiol. 2020;75(2):207-221. doi: 10.1016/j.jacc.2019.11.014.
11. Arany Z. Peripartum cardiomyopathy. N Engl J Med. 2024;390(2):154-164. doi: 10.1056/NEJMra2306667.
12. Bültmann BD, Klingel K, Näbauer M, Wallwiener D, Kandolf R. High prevalence of viral genomes and inflammation in peripartum cardiomyopathy. Am J Obstet Gynecol. 2005;193(2):363-365. doi: 10.1016/j.ajog.2005.01.022.
13. Fu K, Zhang H, Chen N, Hu Y, Xiao J, Zhang X, *et al.* Risk factors for intracardiac thrombus in peripartum cardiomyopathy: a retrospective study in China. ESC Heart Fail. 2023;10(1):148-158. doi: 10.1002/ehf2.14219.
14. Radakrishnan A, Dokko J, Pastena P, Kalogeropoulos AP. Thromboembolism in peripartum cardiomyopathy: a systematic review. J Thorac Dis. 2024;16(1):645-660. doi: 10.21037/jtd-23-945.
15. Badge M, Nagashweta LS, Rajagopalan K, Görlinger K, Kapoor PM. Peripartum cardiomyopathy and thrombosis—a match made in hell! J Card Crit Care TSS. 2025;9(4):198-201. doi: 10.25259/JCCC_47_2024.
16. Bates SM, Middeldorp S, Rodger M, James AH, Greer IA. Guidance for the treatment and prevention of obstetric-associated venous thromboembolism. J Thromb Thrombolysis. 2016;41(1):92-128. doi: 10.1007/s11239-015-1309-0.
17. Ricci F, De Innocentiis C, Verrengia E, Di Stasio E, Di Carlo A, Santos AR, *et al.* The role of multimodality cardiovascular imaging in peripartum cardiomyopathy. Front Cardiovasc Med. 2020;7:4. doi: 10.3389/fcvm.2020.00004.
18. Prameswari HS, Kamarullah W, Pranata R, Putra ICS, Undarsa AC, Iqbal M, *et al.* Meta-analysis of cardiac magnetic resonance in prognosticating left ventricular function in peripartum cardiomyopathy. ESC Heart Fail. 2025;12(1):304-315. doi: 10.1002/ehf2.15024.
19. Rasmusson K, Brunisholz K, Budge D, Horne BD, Alharethi R, Folsom J, *et al.* Peripartum cardiomyopathy: post-transplant outcomes from the United Network for Organ Sharing database. J Heart Lung Transplant. 2012;31(2):180-186. doi: 10.1016/j.healun.2011.11.018.