Indonesian Journal of Cardiology
Indonesian J Cardiol • Vol. 47 • 2026
pISSN: 2830-3105 / eISSN: 2964-7304
doi: 10.30701/ijc.2056

Case Report

Intraoperative Cardiac Arrest as the First Manifestation of Undiagnosed Peripartum Cardiomyopathy in a Secondary Rural Hospital: A Case Report

Miftah Habi Farid[1], Rikho Ade Putera[2]

Abstract

Background: Peripartum Cardiomyopathy (PPCM) is an uncommon cause of acute heart failure occurring in late pregnancy or early postpartum. In resource-constrained hospitals without advanced cardiac support or biomarker testing, delayed recognition may lead to catastrophic complications.

Case Illustration: A 28-year-old primigravida underwent elective cesarean delivery and developed sudden Pulseless Electrical Activity (PEA) five minutes postpartum. Return of spontaneous circulation was achieved after two cycles of cardiopulmonary resuscitation. Post-arrest evaluation revealed severe global Left Ventricular (LV) systolic dysfunction (Left Ventricular Ejection Fraction [LVEF] 16%) with LV dilation (Left Ventricular Internal Diameter at end-diastole [LVIDd] 5.4 cm) and preserved Right Ventricular (RV) systolic function (Tricuspid Annular Plane Systolic Excursion [TAPSE] 2.2 cm). Emergency echocardiography was performed in the Intensive Care Unit (ICU); therefore, comprehensive hemodynamic data were limited. Inferior vena cava size was within normal limits. Hemoglobin decreased modestly (12.2 to 10.6 g/dL), and no evidence of massive hemorrhage or distributive shock was observed. In the absence of biomarker availability, diagnosis was established based on clinical echocardiographic correlation. The patient required short-term norepinephrine (0.1 mcg/kg/min for 12 hours) and dobutamine (5 mcg/kg/min for 48 hours) with gradual down-titration. Guideline-directed medical therapy was initiated after stabilization, including sacubitril–valsartan, spironolactone, bisoprolol, dapagliflozin, diuretics, and bromocriptine (5 mg twice daily for one week). LVEF improved to 34% at day 10 and recovered to 64% at three months.

Conclusions: PPCM may present as abrupt intraoperative cardiac arrest even in previously healthy women. Early echocardiography and timely heart failure therapy are critical, particularly in secondary hospitals lacking advanced diagnostic tools.

[1]General Practitioner, Sungai Dareh General Hospital, Dharmasraya, Indonesia.
[2]Department of Cardiology, Sungai Dareh General Hospital, Dharmasraya, Indonesia.

Correspondence:
Miftah Habi Farid,

General Practitioner, Sungai Dareh General Hospital, Dharmasraya, Indonesia.

Email: mhabifarid@gmail.com

Keywords: Postpartum Cardiomyopathy, Cardiac Arrest, Pulseless Electrical Activity, Bromocriptine, Rural Hospital

Introduction

Peripartum Cardiomyopathy (PPCM) is defined as new-onset systolic heart failure occurring toward the end of pregnancy or within five months postpartum, with Left Ventricular Ejection Fraction (LVEF) ≤45% in the absence of other identifiable causes.[1] The incidence varies widely worldwide, ranging from 1 in 300 to 1 in 15,000 live births.[2]

In Southeast Asia, precise epidemiological data remain limited; however, Indonesian observational data suggest PPCM contributes significantly to maternal cardiac morbidity, with delayed diagnosis frequently observed due to symptom overlap with physiological pregnancy.[3] In Southeast Asia, epidemiological data remain limited. However, an Indonesian cohort from Dr. Hasan Sadikin General Hospital, Bandung, from January 2014 to December 2021 (n=138) reported a 6-month mortality of 6.7%, while 63.3% of patients recovered. These findings indicate that PPCM remains an important contributor to maternal cardiovascular morbidity and mortality in Indonesia.[4]

The pathophysiology involves oxidative stress-mediated cleavage of prolactin into a 16-kDa antiangiogenic fragment, endothelial dysfunction, inflammatory activation, and genetic susceptibility.[5-6] Severe presentations may include cardiogenic shock, malignant arrhythmias, thromboembolism, and rarely sudden cardiac arrest.[7]

We report a case of intraoperative Pulseless Electrical Activity (PEA) as the first manifestation of PPCM in a secondary rural hospital without access to advanced cardiac biomarkers or mechanical circulatory support.

Case Illustration

A 28-year-old primigravida at 38 weeks' gestation underwent elective cesarean delivery under regional anesthesia for cephalopelvic disproportion. Antenatal history was unremarkable. Preoperative hemoglobin was 12.2 g/dL and leukocyte count 8,190/μL. No hypertension or preeclampsia was documented. Five minutes after delivery, the patient suddenly became unresponsive with apnea and absent pulse. The cardiac monitor showed organized sinus tachycardia consistent with PEA. Immediate high-quality cardiopulmonary resuscitation was initiated. Return of spontaneous circulation was achieved after approximately two cycles. Electrocardiography performed before and after return of spontaneous circulation showed sinus rhythm without S1Q3T3 pattern, new right bundle branch block, or other signs suggestive of acute Right Ventricular (RV) strain.

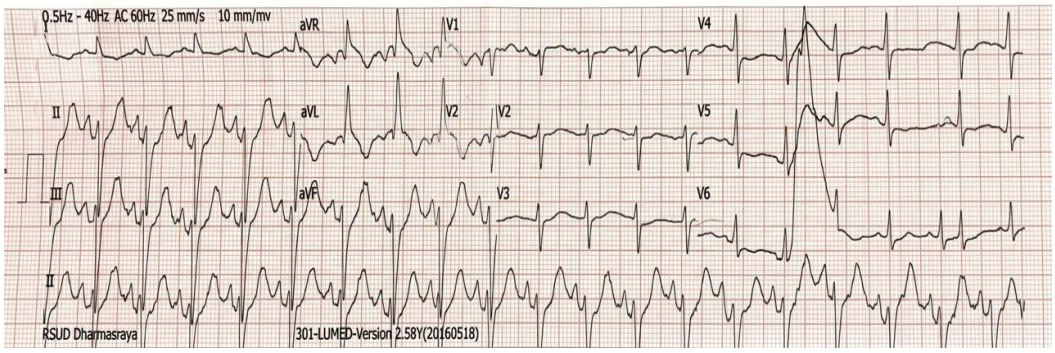


Figure 1. Pre-arrest electrocardiography showing sinus tachycardia during pulseless electrical activity.

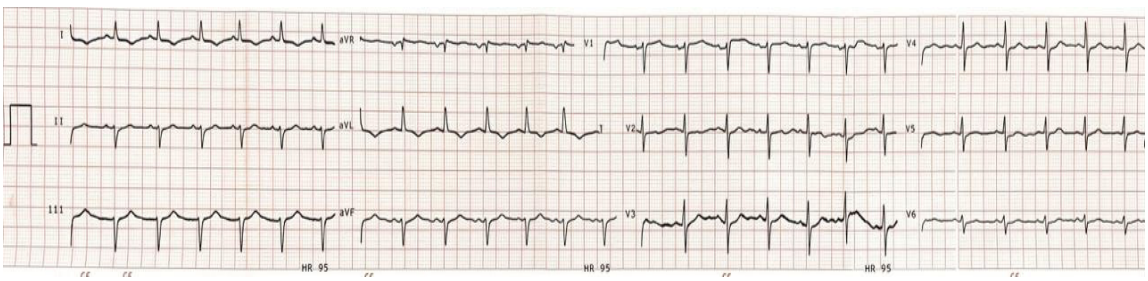


Figure 2. Post–cardiac arrest electrocardiography demonstrating sinus rhythm following return of spontaneous circulation.

Transthoracic echocardiography performed emergently demonstrated:

- LVEF 16% (biplane Simpson)
- Global hypokinesia
- Left Ventricular Internal Diameter at end-diastole (LVIDd) 5.4 cm
- Left Ventricular Internal Diameter at end-systole (LVIDs) 5.0 cm
- Interventricular septum 1.0 cm
- No Left Ventricular (LV) thrombus
- Tricuspid Annular Plane Systolic Excursion (TAPSE) 2.2 cm (preserved RV systolic function)
- Inferior vena cava diameter was within normal limits
- No evidence of severe pulmonary hypertension
- Moderate tricuspid regurgitation without structural leaflet abnormality

Preserved TAPSE and absence of RV dilation reduced the likelihood of acute massive pulmonary embolism.[7] Absence of profound hemorrhage or uterine atony argued against hypovolemic arrest. Clinical stability before collapse and lack of bronchospasm or urticaria made anaphylaxis unlikely. Given the peripartum timing, severe global LV dysfunction, and exclusion of alternative causes, a diagnosis of PPCM complicated by cardiogenic shock and PEA arrest was established.

The patient required vasoactive support with norepinephrine 0.1 mcg/kg/min for approximately 12 hours and dobutamine 5 mcg/kg/min for 48 hours, both gradually down-titrated according to hemodynamic response. Intravenous furosemide 20 mg twice daily was administered for volume management. Spironolactone was initiated early during hospitalization. Sacubitril–valsartan (1 × 2.5 mg) was started after initial stabilization. Bisoprolol 2.5 mg once daily was introduced on day 2 when the dobutamine dose had been significantly reduced. Bromocriptine 5 mg twice daily was administered during the first week of hospitalization due to financial constraints. At discharge, the patient was prescribed sacubitril–valsartan (1 × 2.5 mg daily), dapagliflozin (10 mg daily), spironolactone (2.5 mg daily), bisoprolol (2.5 mg daily), and oral furosemide (40 mg daily).

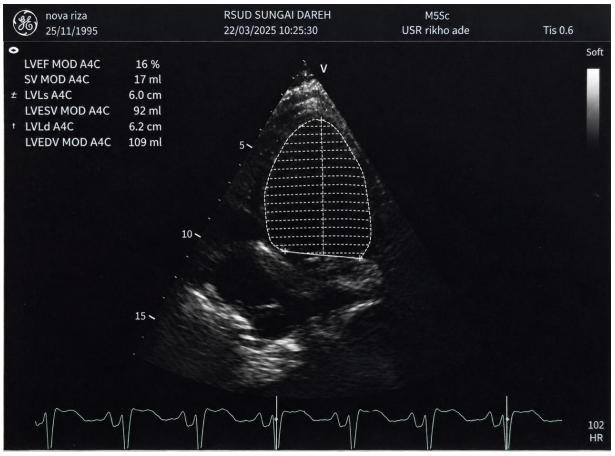


Figure 3. Transthoracic echocardiography showing LVEF 16%.

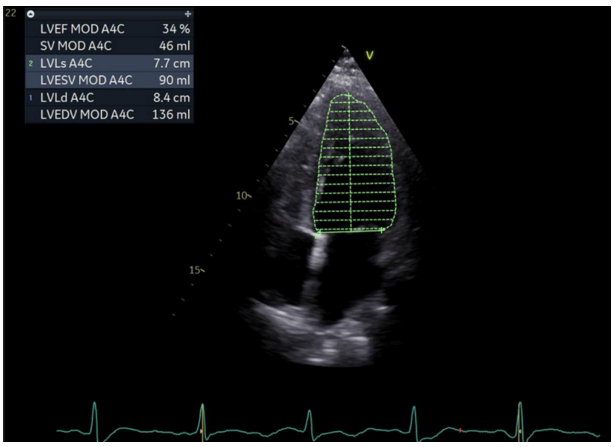


Figure 4. Follow-up echocardiography showing improvement of LVEF to 34%.

Discussion

PPCM may present with a wide spectrum of severity, ranging from mild heart failure symptoms to cardiogenic shock and sudden cardiac arrest. In this case, intraoperative PEA occurred abruptly in a previously healthy woman without preceding symptoms, representing an extreme initial manifestation of PPCM. Compared with the reported 6-month mortality of 6.7% in a cohort from an Indonesian tertiary center, our patient achieved complete recovery, underscoring the importance of early recognition even in secondary hospitals. Although uncommon, sudden circulatory collapse has been reported in patients presenting with severely reduced LV systolic function, particularly when LVEF is profoundly depressed.[1,4]

The immediate postpartum period is characterized by abrupt hemodynamic shifts, including autotransfusion from uterine contraction and sudden alterations in preload and afterload. In the presence of profound LV systolic dysfunction (LVEF 16%), these physiological changes may precipitate acute pump failure. In our patient, the absence of preceding arrhythmic events and the presence of organized electrical activity during collapse suggest that PEA likely resulted from critically reduced forward stroke volume rather than a primary malignant arrhythmia.[6,8] This mechanism is consistent with severe cardiogenic shock physiology.

Massive pulmonary embolism and amniotic fluid embolism were important differential diagnoses in the immediate postpartum setting. However, preserved RV systolic function (TAPSE 2.2 cm), absence of RV dilation, and lack of echocardiographic signs of acute pressure overload significantly reduced the likelihood of acute massive pulmonary embolism.[9] Additionally, there was no clinical evidence of profound hypoxemia, disseminated intravascular coagulation, or excessive hemorrhage to support amniotic fluid embolism or hypovolemic shock. The modest decrease in hemoglobin (12.2 to 10.6 g/dL) was not consistent with massive blood loss. The global pattern of LV hypokinesia further supported a non-ischemic cardiomyopathic process consistent with PPCM.

After hemodynamic stabilization, guideline-directed medical therapy was initiated, including sacubitril–valsartan, a mineralocorticoid receptor antagonist, a beta-blocker, an SGLT2 inhibitor, and diuretics. Initial vasoactive support consisted of norepinephrine and dobutamine during the cardiogenic shock phase, followed by a gradual transition to oral heart failure therapy.

Bromocriptine 5 mg twice daily was administered during the first week following stabilization. Given the severe phenotype (cardiac arrest and LVEF 16%), short-term prolactin blockade was considered reasonable in accordance with the European Society of Cardiology (ESC) 2019 recommendations. Treatment duration was limited to one week due to financial constraints. Experimental and clinical data suggest that prolactin blockade may mitigate myocardial injury by preventing the formation of the 16-kDa prolactin fragment implicated in PPCM pathophysiology.[6] A randomized multicenter study demonstrated improved LV recovery with bromocriptine in severe PPCM cases, supporting its use as an adjunctive disease-modifying strategy.[10] Considering the life-threatening presentation, the decision to initiate bromocriptine was consistent with evidence favoring its use in severe phenotypes.

Although randomized data specifically addressing sodium–glucose cotransporter 2 inhibitors in PPCM are lacking, large contemporary trials in heart failure with reduced ejection fraction have demonstrated consistent reductions in mortality and heart failure hospitalization irrespective of underlying etiology.[11] Given the severely reduced LVEF and postpartum status, dapagliflozin was introduced as part of comprehensive Heart Failure with reduced Ejection Fraction (HFrEF) therapy, acknowledging that its use in PPCM is extrapolated from broader heart failure evidence rather than PPCM-specific trials.

This case also underscores the diagnostic challenges in a secondary rural hospital without access to N-Terminal pro-B-type Natriuretic Peptide (NT-proBNP), troponin testing, cardiac Magnetic Resonance Imaging (MRI), or mechanical circulatory support. In such settings, timely bedside echocardiography becomes the cornerstone of diagnosis and therapeutic decision-making. Despite limited resources, prompt recognition, structured hemodynamic stabilization, and evidence-aligned therapy resulted in complete myocardial recovery, highlighting that favorable outcomes are achievable even outside tertiary centers.

Conclusion

PPCM may manifest abruptly as intraoperative cardiac arrest without preceding symptoms. Severe LV dysfunction can lead to PEA during the immediate postpartum hemodynamic transition. In secondary hospitals lacking advanced diagnostics, early echocardiography and timely initiation of

guideline-directed therapy, including prolactin blockade in selected severe cases, may enable full myocardial recovery and a favorable maternal outcome.

List of Abbreviations

ESC	European Society of Cardiology
HFrEF	Heart Failure with reduced Ejection Fraction
ICU	Intensive Care Unit
LV	Left Ventricular
LVEF	Left Ventricular Ejection Fraction
LVIDd	Left Ventricular Internal Diameter at end-diastole
LVIDs	Left Ventricular Internal Diameter at end-systole
MRI	Magnetic Resonance Imaging
NT-proBNP	N-Terminal pro-B-type Natriuretic Peptide
PEA	Pulseless Electrical Activity
PPCM	Peripartum Cardiomyopathy
RV	Right Ventricular
TAPSE	Tricuspid Annular Plane Systolic Excursion

Ethical Clearance

Not applicable.

Publication Approval

All authors consent to the publication of this manuscript.

Authors' Contributions

MHF performed the conception and design of the case report, data collection, literature review, and preparation of the manuscript. RAP supervised the project and critically revised the manuscript for important intellectual content. Both authors have read and approved the final version of the manuscript.

Acknowledgments

None.

Conflict of Interest

None.

Availability of Data and Materials

Not applicable.

Funding

None.

Copyright/Permissions for Figures

Not applicable.

Generative AI and AI-Assisted Technologies in the Writing Process

The authors acknowledge that artificial intelligence (AI) tools were only used to assist in language editing and did not generate or alter the scientific content, analyses, or conclusions presented in this manuscript.

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 ESC Working Group on Peripartum Cardiomyopathy. Eur Heart J. 2019;40(38):3599-613.
2. Iorgoveanu C, Zaghloul A, Ashwath M. Peripartum cardiomyopathy: a review. Heart Fail Rev. 2021;26(6):1287-96.
3. Sliwa K, Mebazaa A, Hilfiker-Kleiner D, Petrie MC, Maggioni AP, Böhm M, et al. Clinical characteristics of patients from the worldwide registry on peripartum cardiomyopathy. Eur J Heart Fail. 2017;19(9):1131–41.
4. Prameswari HS, Dewi TI, Hasan M, Martanto E, Astuti A, Saboe A, Cool CJ. Clinical Presentation and 6-Month Outcomes of Patients with Peripartum Cardiomyopathy in Indonesia. Int J Gen Med. 2024 Mar 21;17:1073-83
5. Hilfiker-Kleiner D, Sliwa K. Pathophysiology and epidemiology of peripartum cardiomyopathy. Nat Rev Cardiol. 2014;11(6):364–70.
6. Sliwa K, Hilfiker-Kleiner D, Petrie MC, Mebazaa A, Pieske B, Buchmann E, et al. Current state of knowledge on aetiology, diagnosis, management, and therapy of peripartum cardiomyopathy. Eur J Heart Fail. 2010;12(8):767–78.

7. Bello N, Rendon ISH, Arany Z. The relationship between pre-eclampsia and peripartum cardiomyopathy: a systematic review and meta-analysis. J Am Coll Cardiol. 2013;62(18):1715–23.
8. Kjaergaard J, Akkan D, Iversen KK, Kober L, Torp-Pedersen C, Hassager C. Right ventricular dysfunction as an independent predictor of mortality in heart failure. Eur J Heart Fail. 2007;9(6-7):610–16.
9. Clark SL. Amniotic fluid embolism. Obstet Gynecol. 2014;123(2 Pt 1):337–48.
10. Hilfiker-Kleiner D, Haghikia A, Berliner D, Vogel-Claussen J, Schwab J, Franke A, et al. Bromocriptine for the treatment of peripartum cardiomyopathy: a multicentre randomized study. Eur Heart J. 2017;38(35):2671–79.
11. McMurray JJV, Solomon SD, Inzucchi SE, Køber L, Kosiborod MN, Martinez FA, et al. Dapagliflozin in patients with heart failure and reduced ejection fraction. N Engl J Med. 2019;381:1995–2008.