

Closer Insight through Ventriculo-Arterial Coupling Perspective of Late-recognized Peripartum Cardiomyopathy in The Presence of a Predictor of Non-Recovery: Case Report

Mochamad Rizky Hendiperdana¹

Abstract

Background: Peripartum Cardiomyopathy (PPCM) is ventricular systolic dysfunction that develops in the last months of pregnancy to several months postpartum. Emerging evidence suggests that PPCM may develop up to 1 year after delivery. This condition is associated with several predictors of non-recovery.

Case Illustration: A 39-year-old woman was admitted with heart failure syndrome. The patient had late-recognized PPCM after an 18-month postpartum period. Echocardiography showed Left Ventricular (LV) dilation and severely reduced Ejection Fraction (EF). The predictor of non-recovery is also present in this case. However, after 5 months of administered Guideline-Directed Medical Treatment (GDMT), the patient developed structural and complete functional reverse remodeling. During the follow-up period, we observed significant improvement in Left Ventricular Ejection Fraction (LVEF) from 23% to 57%, Global Longitudinal Strain (GLS) from -5.2% to -17.5%, Left Atrial Strain (LAS)-reservoir from 8% to 31%, and global work index (GWI) from 516 mmHg% to 1702 mmHg% from myocardial work index analysis.

Conclusions: Several factors have been identified as predictors of non-recovery in PPCM in previous studies, including LVEF <30%, LV dilation, and severe valvular regurgitation. The current scoring system for PPCM recovery, developed by ESC EORP, also predicts 6-month recovery. There was significant improvement in surrogate markers for myocardial systolic function despite of the presence of late-recognized predictors of non-recovery in this case. Hemodynamic phenotype, rather than a single marker measurement, is emerging as a key factor in PPCM prognostication.

¹Division of Cardiovascular Medicine, Pandan Arang General Hospital, Boyolali, Indonesia.

Correspondence:

Mochamad Rizky Hendiperdana,

(Indonesian J Cardiol, 2026;47;206-213)

Division of Cardiovascular Medicine, Pandan Arang General Hospital, Boyolali, Indonesia.

Email: mhendiperdana@gmail.com

Keywords: Peripartum cardiomyopathy, Hemodynamic phenotype, Ventriculo-arterial coupling, Prognostication, Case report

Introduction

Peripartum Cardiomyopathy (PPCM) is ventricular systolic dysfunction that develops in the last months of pregnancy to several months postpartum. Emerging evidence suggests that PPCM may develop up to 1 year after delivery. This condition is associated with several predictors of non-recovery. When PPCM is inadequately managed and persists beyond 6 months postpartum, it reduces the potential for recovery and reversibility.¹⁻⁴ Although Left Ventricular (LV) function recovery was observed in more than half of the PPCM population,⁵ previous studies also describe several factors as poor predictors of LV function recovery in PPCM. These predictors of non-recovery are often linked to baseline severe Left Ventricular Systolic Dysfunction (LVSD) (Left Ventricular Ejection Fraction [LVEF] < 30%), Left Ventricular End-Diastolic Dimension (LVEDD) > 56 mm, Right Ventricular (RV) dysfunction, duration of symptom, national Human Development Index (HDI), QRS duration, and the presence of pre-eclampsia.⁴⁻⁹

Case Illustration

A 39-year-old woman presented with progressive shortness of breath, orthopnea, abdominal enlargement, and bilateral pretibial edema (December 2024) (Figure 1 for visual summary). On admission, vital signs were unremarkable. Physical examination was also unremarkable except for the ascites sign. A 12-lead electrocardiography showed sinus rhythm with a QRS duration of 90 ms and no specific abnormality. Chest x-ray revealed cardiac silhouette enlargement with minimal pulmonary congestion sign (Figure 2).

The patient's previous medical record revealed a history of a cesarean section procedure 18 months prior to the patient's recent admission (June 2023), and the patient started to develop clinical symptoms such as dyspnea on effort in September 2023. The patient then started to seek medical attention 2 months after the first clinical symptom (December 2023), but unfortunately, the patient described that the symptom was not well-managed. Then later, the patient developed ascites and was suspected of having liver disease. The patient then lost follow-up for a long period until 2 months prior to recent admission, when the patient developed progressive shortness of breath and abdominal enlargement. Patient denied any fever-like symptoms before the onset of dyspnea and had no family history of cardiac disease.

Blood test examination was unremarkable. Patient has euthyroid profile. Transthoracic echocardiography revealed Right Atrial (RA) dilation with RA area of 26.2 cm² (normal < 12 cm²), LV dilation with LVEDD 58 mm and Left Ventricular End-Diastolic Volume (LVEDV) 157 ml, reduced LVEF of 23%, with LV Global Longitudinal Strain (GLS) value of [-5.2%], reduced RV systolic function (Tricuspid Annular Plane Systolic Excursion [TAPSE] 13 mm) and severe functional tricuspid regurgitation (TR) (Figure 3) (Table 1). Left atrial strain (LAS) assessment also revealed impaired Left Atrial (LA) function (Left Atrial Strain [LAS]-reservoir 8%, LAS-conduit -5%, LAS-contraction -3%) (Figure 4A)(Video 1).

The pressure-strain derived Myocardial Work Indices (MWI) analysis showed severely reduced myocardial systolic performance as follows: Global Work Index (GWI) 516 mmHg%, Global Constructive Work (GCW) 692 mmHg%, Global

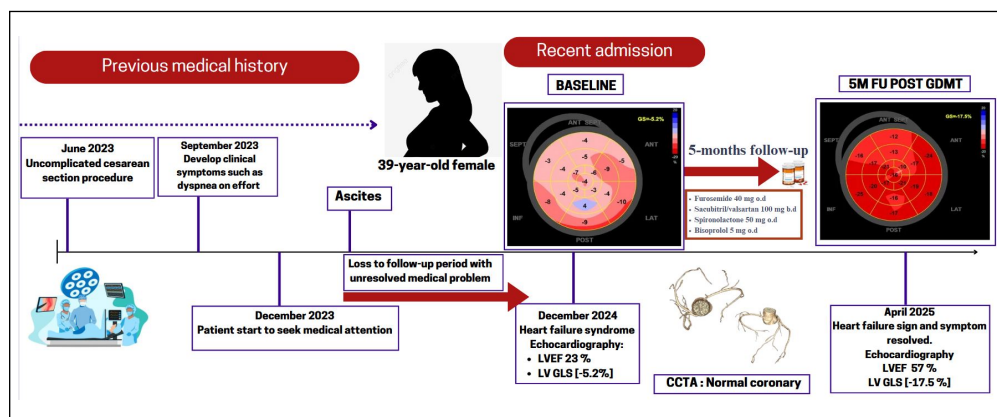


Figure 1. Visual summary of the case. Chronological events and imaging milestone.

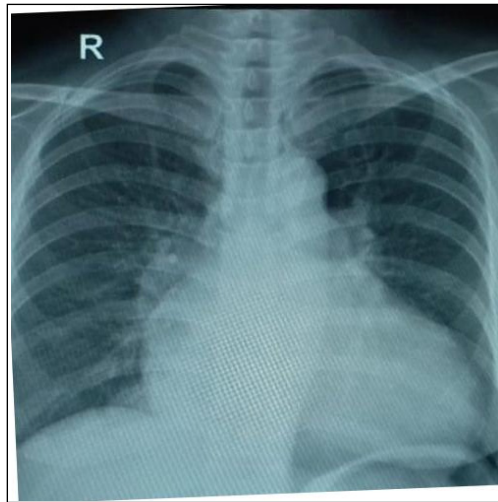


Figure 2. Chest x-ray revealed cardiac silhouette enlargement with minimal pulmonary congestion sign.

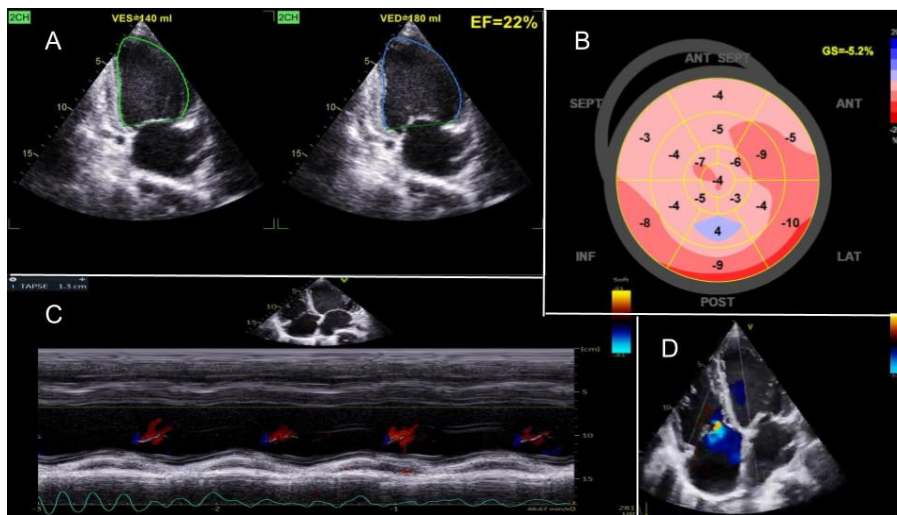


Figure 3. Baseline transthoracic echocardiography revealed: (A) Reduced LV ejection fraction (LVEF) of 23%; (B) Reduced LV GLS value of [-5.2%]; (C) Reduced RV systolic function (TAPSE 13 mm); (D) severe functional TR. LV: left ventricular; LVEF: left ventricular ejection fraction; GLS: global longitudinal strain; RV: right ventricular; TAPSE: tricuspid annular plane systolic excursion; TR: tricuspid regurgitation.

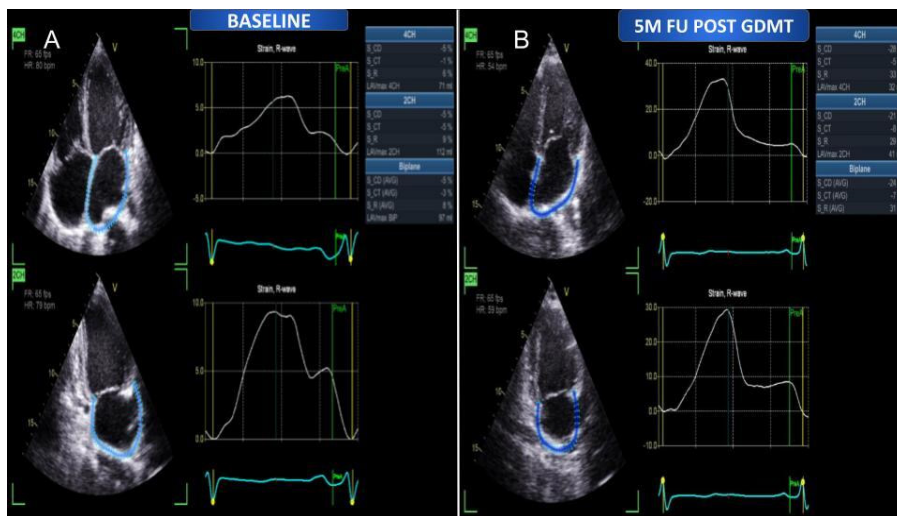


Figure 4. Transthoracic Echocardiography LAS analysis. Left panel: Baseline LAS measurement; Right panel: Post-treatment LAS evaluation showed significant improvement. LAS: left atrial strain.

Table 1. Patient's summary on echocardiographic finding and clinical finding during initial presentation, 3-month post-treatment and 5-months post-treatment.

| Parameters | Initial Presentation (December 2024) | 3-Month Evaluation (March 2025) | 5- Months Evaluation (May 2025) |
|--|---|------------------------------------|------------------------------------|
| Echocardiographic Findings (GE Vivid E95) TM | | | |
| LVEDV (mL) | 157 | 128 | 129 |
| LVESV (mL) | 121 | 59 | 56 |
| LVEDD (mm) | 58 | 54 | 52 |
| LVEF (%) | 23 | 54 | 57 |
| TAPSE (mm) | 13 | 18 | 20 |
| RV S' velocity (cm/s) | 7 | 8 | 11 |
| Mitral peak E velocity (m/s) | 0.56 | 0.71 | 0.7 |
| E/e' average | 11.47 | 8.53 | 9.08 |
| LV averaged GLS (%) | -5.2 | -15.8 | -17.5 |
| RA area (cm ²) (Normal < 18 cm ²) | 26.2 | 9.9 | 9.9 |
| Functional TR severity | Severe | Mild | Mild |
| Clinical Finding | | | |
| ECG QRS duration (ms) | 90 | 80 | 80 |
| Blood pressure (mm/Hg) | 137/98 | 95/60 | 120/90 |
| Heart rate (beat per minute) | 90 | 60 | 58 |

LVEDV: Left Ventricular End-Diastolic Volume; LVESV: Left Ventricular End-Systolic Volume; LVEDD: Left Ventricular End-Diastolic Dimension; LVEF: Left Ventricular Ejection Fraction; TAPSE: Tricuspid Annular Plane Systolic Excursion; RV: Right Ventricle; GLS: Global Longitudinal Strain; LA: Left Atrial; RA: Right Atrial; TR: Tricuspid Regurgitation; ECG: Electrocardiography.

Wasted Work (GWW) 183 mmHg%, Global Work Efficiency (GWE) 77% (Figure 5A) (Table 1). The non-invasive echocardiography-based hemodynamic assessment of Ventriculo-Arterial (VA) coupling showed Elastance arterial (Ea) of 3.51 and Elastance end-systolic (Ees) 1.01 with Ea:Ees ratio 3.47 (Table 2). A Coronary Computed Tomography Angiography (CCTA) showed normal coronary anatomy without stenosis.

The differential diagnosis of the patient's subjective and objective findings can be described as coronary artery disease, myocarditis, autoimmune disorders, thyroid dysfunction, or genetic cardiomyopathies. While coronary stenosis was not found from CCTA, the Coronary Artery Disease (CAD) etiology was unlikely. An euthyroid profile also made thyroid heart disease unlikely. Lastly, the absence of fever and an inflammatory-like syndrome, and the absence of a family history of any cardiac disease, made myocarditis, autoimmune, and genetic cardiomyopathies at least possible. The limitation of Cardiac Magnetic Resonance (CMR) imaging in our case was a lack of a myocardial tissue characterization profile. Therefore, the patient was diagnosed with Heart Failure with reduced Ejection

Fraction (HFrEF) due to late-recognized PPCM. Based on these data, the patient was classified as cluster 2 of the hemodynamic PPCM cohort according to Meledin et al.'s study.⁴

Based on these findings, the European Society of Cardiology (ESC) EURObservational Research Programme (EORP) PPCM Recovery Score classified this patient with a recovery score of 4, with a predicted 6-month recovery of 35%. The ESC EORP PPCM Recovery Score elaborated several validated predictors for 6-month recovery, which include baseline LVEF at presentation, either of $\leq 35\%$ or $> 35\%$, LVEDD, national HDI, duration of symptoms, QRS duration, and the presence of pre-eclampsia.⁹

The patient was treated with intravenous loop diuretic for decongestion management along Guideline-Directed Medical Therapy (GDMT) with uptitrated dose to sacubitril/valsartan 100 mg b.i.d., bisoprolol 5 mg o.d., and spironolactone 50 mg o.d. The patient was discharged from hospital admission uneventfully. The patient was then evaluated monthly during outpatient visits.

In five months of outpatient follow-up evaluation (April 2025), the symptoms improved without

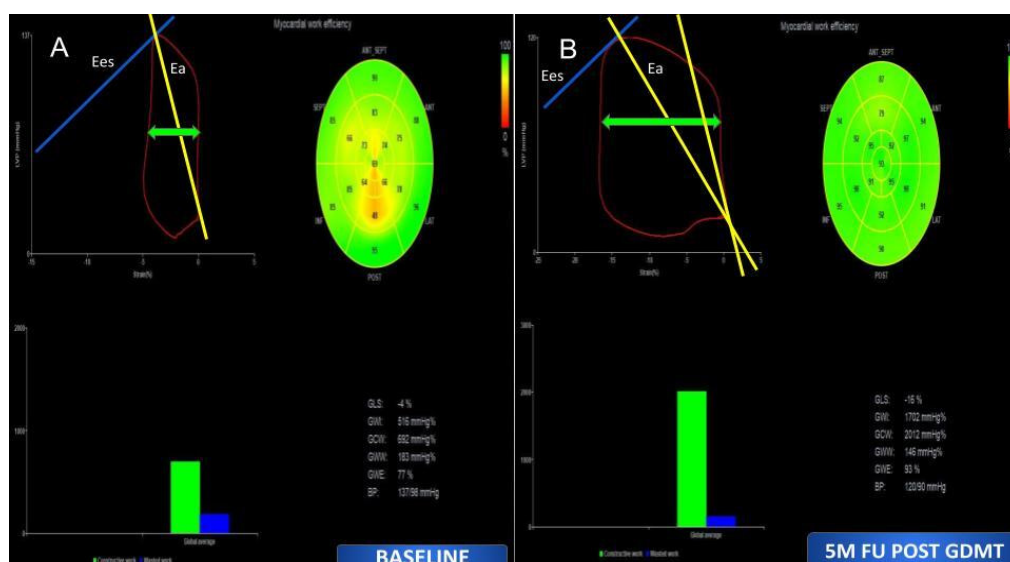


Figure 5. (A) Baseline pressure-strain derived MWI analysis showed severely reduced myocardial systolic performance, which was demonstrated in reduced GWI and GCW. (B) 5-month post-treatment evaluation of MWI showed significant improvement in GWI and GCW. Note that the baseline Ees slope in the PS loop has no difference compared to the post-treatment PS loop. Post-treatment follow-up PS loop resulting in reduced Ea slope angle, which depicts afterload reduction. Green arrow showed increased in stroke work in follow-up compared to the baseline. MWI: myocardial work indices; GWI: global work index; GCW: global constructive work; Ees: end-systolic elastance; Ea: arterial elastance; PS: pressure-strain.

Table 2. Patient’s summary on hemodynamic assessment of non-invasive ventriculo-arterial coupling profile at initial presentation and 5-months post-treatment.

| Parameters | Initial Presentation (December 2024) | 5-Months Evaluation (May 2025) |
|--|--------------------------------------|--------------------------------|
| Echocardiographic Findings (GE Vivid E95)™ | | |
| Systolic blood pressure (mmHg) | 137 | 120 |
| End-systolic pressure (mmHg) | 123 | 108 |
| LV stroke volume (mL) | 35 | 73 |
| LV end-systolic volume (mL) | 121 | 56 |
| Elastance arterial (Ea) | 3.51 | 1.47 |
| Elastance end-systolic (Ees) | 1.01 | 1.92 |
| Ea/Ees ratio | 3.47 | 0.76 |
| GWI (mmHg%) | 516 | 1702 |
| GCW (mmHg%) | 692 | 2012 |
| GWW (mmHg%) | 183 | 146 |
| GWE (%) | 77 | 93 |

LV: Left ventricular; GWI: Global work index; GCW: Global constructive work; GWW: Global wasted work; GWE: Global work efficiency.

recurrent congestion or ascites. We observed a reduction in RA area and LV volumes (26.2 to 9.9 cm² and LVEDV 157 to 128 mL, respectively) (Figure 6) and resolution of TR severity. We also found functional improvement of LV systolic function (LVEF of 57% and LV GLS of [-17.5%]) (Figure 7) (Table 1) with significant LA function improvement (LAS-reservoir 31%, LAS-conduit -24%, LAS-contraction -7%) (Figure 4B). There was a significant improvement in MWI, with GWI

1702 mmHg%, GCW 2012 mmHg%, GWW 146 mmHg%, and GWE 93% (Figure 5B). The non-invasive hemodynamic assessment of VA coupling also showed improvement of Ea:Ees ratio (Table 2) (Video 2).

After complete recovery from imaging criteria was achieved, the patient was advised to use contraception planning, especially non-hormonal contraception like an intrauterine device, to prevent subsequent pregnancy.

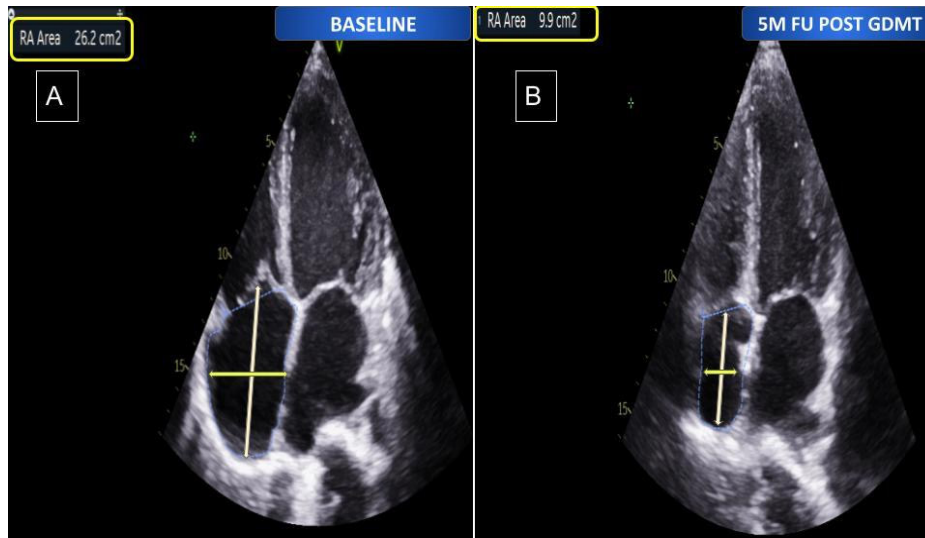


Figure 6. Transthoracic echocardiography. Left panel: Baseline echocardiography revealed RA dilation with RA area of 26.2 cm²; Right panel: Post-treatment echocardiography evaluation showed a reduction in RA area to 9.9 cm². RA: right atrial.

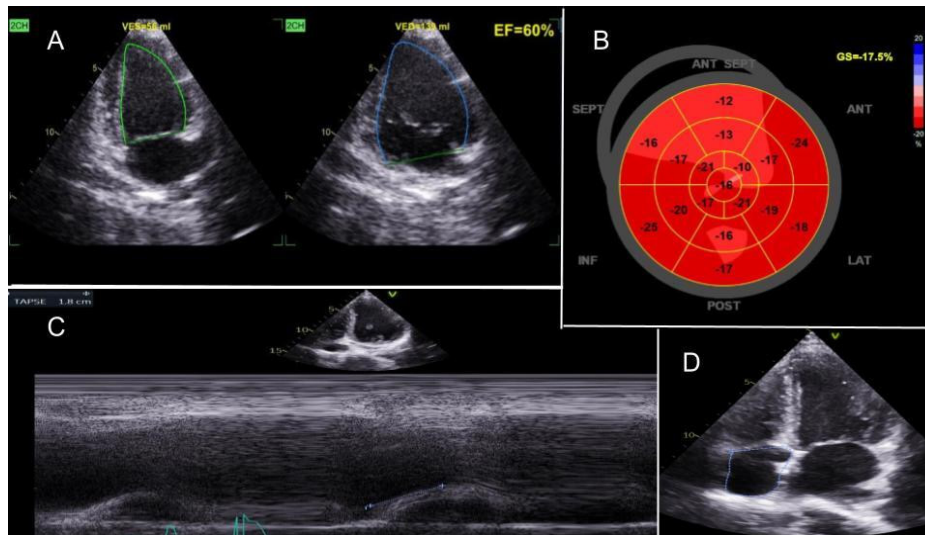


Figure 7. Post-treatment evaluation of transthoracic echocardiography revealed: (A) Improved LV ejection fraction (LVEF) to 57%; (B) Improved LV GLS value to [-17.5 %]; (C) Improved RV systolic function to TAPSE 18 mm; (D) Reduction in RA area. LV: left ventricular; LVEF: left ventricular ejection fraction; GLS: global longitudinal strain; RV: right ventricular; TAPSE: tricuspid annular plane systolic excursion; TR: tricuspid regurgitation. RA: right atrial.

Discussion

We presented a young female patient with late-recognized PPCM. Although symptom development commenced during the window period of typical PPCM window period (3 months postpartum), the patient was not recognized or treated for PPCM until 18 months postpartum.

Meledin et al. found that delayed PPCM diagnosis beyond 2 weeks was more common in the non-recovery group than in the recovery group (46.2% vs 20.6%; $p = 0.01$). Based on this finding,

the longer the interval from clinical onset to PPCM diagnosis, the poorer the prognosis for myocardial recovery. In multivariate analysis, baseline LVEF $> 35\%$ (HR 6.0; $p = 0.008$) and baseline LVEDD < 55 mm (HR 4.05; $p = 0.029$) were the only statistically significant factors associated with a favorable prognosis for recovery.⁴

In addition, ESC EORP PPCM Recovery Score also describes LVEF, LVEDD, and duration of symptoms, which become important prognostic factors.⁹ The ESC EORP registry describes LV

recovery that occurred in 46.5% studied population, with a mean change of LVEF of $\pm 13\%$. The recovered population has a smaller LA diameter, less frequent RV impairment, and less frequent significant Mitral Regurgitation (MR). Our case showed the presence of a predictor of non-recovery, with 35% prediction of 6-month recovery. Estimating the odds of LV recovery is important for individualizing management decisions and counselling plans.⁹ This underlines how exceptional the recovery finding of LVEF in our patient is compared to ESC EORP data. However, our case demonstrated successful Left Heart Reverse Remodeling (LHRR) after 5 months of GDMT, despite long-standing, untreated PPCM, with the presence of a predictor of non-recovery.¹⁻⁴ Unfortunately, in some patients, systolic function never completely recovers.⁴⁻⁵

More sensitive systolic performance parameters, such as GLS and MWI, did not differ between the recovery and non-recovery groups.⁴ This result was contradictory with Sugahara et al.'s finding, which revealed that a reduced GLS (cutoff of $[-10.6\%]$) at baseline was associated with death and persistent LV dysfunction.¹⁰ While our patient also poses poor predictor GLS profile $[-5.2\%]$ at initial presentation.

Structural reverse remodeling was observed from a significant reduction in RA size and LV volumes. The functional reverse remodeling is even more significant. The patient showed improvement in LA function, as reflected by increased of all LA strain values, particularly the LAS-reservoir. Moon et al found that in HFrEF patients, improvements of the LAS-reservoir to $> 12.5\%$ after GDMT indicate 'complete' LA reverse remodeling, and it is associated with favorable outcomes compared to those whose LAS-reservoir is still $< 12.5\%$.¹¹

The LV GLS improvement in our case also poses a favorable prognosis. As described by Moon et al, in HFrEF patients with LV GLS improvement to $> [-13\%]$ have a favorable prognosis.¹¹ As shown in our case, after 5 months of GDMT as recommended in the guidelines,^{5,12} the patient achieved significant improvement of LV GLS to $[-15.6\%]$. It appears that traditional predictors of PPCM recovery, such as LVEF, LVEDD, symptom duration, and pre-eclampsia, are insufficient, as our patient demonstrated significant LV recovery despite of low recovery prediction according to the ESC EORP PPCM Recovery score. Hence, emphasizing the need to explore other predictors of recovery.

Meledin et al. proposed a hemodynamic phenotype cluster to predict recovery in PPCM.

The study found that cluster 1 hemodynamic phenotype is marked by profound myocardial contractility dysfunction rather than increased afterload. Whereas cluster 2 is marked by relatively more preserved contractility reserve but extremely increased afterload, and cluster 3 is described as having mildly reduced contractility indexes, slightly increased afterload, and preserved cardiac output. Consequently, the recovery rates from clusters 1 to 3 are 12.5%, 78.6%, and 100%, respectively.⁴

We hypothesized that our case hemodynamic phenotype is more closely associated with cluster 2 hemodynamics, with a higher recovery probability, as demonstrated in Figure 5A, whereas the baseline end-systolic elastance (Ees) slope in the Pressure-Strain (PS) loop did not differ compared to the post-treatment PS loop (Figure 5B). Post-treatment follow-up PS loop results in a reduced Ea slope angle, indicating afterload reduction.

In addition to that, as shown in Table 2, non-invasive high Ea and normal Ees reflected very high LV afterload with relatively preserved LV contractility, resulting in VA uncoupling (high Ea:Ees ratio). This non-invasive hemodynamic measurement also showed improved of VA coupling after 5-month follow-up (Ea:Ees ratio 0.76). These data provided clear evidence regarding PPCM recovery in our case, although the presence of a predictor of non-recovery was noted. Hence, our case is consistent with Meledin et al.'s study, which found that hemodynamic phenotype is more predictive of prognosis. However, this inference is hypothesis-generating because invasive data were unavailable.

The usage of sacubitril/valsartan in PPCM, in our case, is extrapolated from GDMT for Dilated Cardiomyopathy (DCM) and HFrEF. The evidence of sacubitril/valsartan usage in PPCM case is remains limited but supportive.^{5,13-17} This report could be additional real-world clinical experience data of sacubitril/valsartan usage in PPCM patients. The administration of GDMT in our case was continued for 6 months after complete recovery was achieved. In non-recovery PPCM or persistent LV dysfunction, the GDMT is expected to continue indefinitely.¹ Bromocriptine is deferred in our case, considering the late-recognized origin in this case.

The optimal GDMT duration after LV recovery is still unknown, while evidence shows the latent risk of late deterioration of LV dysfunction without the presence of subsequent pregnancy. Therefore, continuing heart failure GDMT is rational in this case. However, if GDMT is intended to be

discontinued; the stepwise strategy should be kept in mind. GDMT weaning should be accompanied by serial echocardiographic monitoring (every 3 to 6 months). Evaluation of LV function is recommended after discontinuation of GDMT, followed by annual echocardiographic assessment.¹

It is recommended that the patient be advised concerning contraception. In our case, the multidisciplinary team considered avoiding estrogen-releasing contraceptives due to thromboembolism risk. In recovered PPCM, the subsequent pregnancy still carries the recurrence risk with a \pm 20% relapse rate. However, in a non-recovered woman with persistent LV dysfunction, the risk of a subsequent pregnancy outweighs any risk associated with contraception (50% relapse rate and increased mortality for subsequent pregnancy). Hence, women should be encouraged to select their preferred contraceptive method.¹

Conclusion

This case reported ‘complete’ LHRR in the setting of late-recognized PPCM with the presence of a predictor of non-recovery, with a 6-month LV recovery prediction of 35 % according to the ESC EORP PPCM Recovery Score. Highlighting the importance and efficacy of optimal GDMT for HFrEF in facilitating complete cardiac function recovery in the presence of a predictor of non-recovery PPCM. Finally, this case underscored the use of hemodynamic phenotyping and non-conventional echocardiographic parameters to evaluate a more sensitive surrogate marker of myocardial recovery for clinical decision-making.

List of Abbreviations

| | |
|------|--|
| CAD | Coronary Artery Disease |
| CCTA | Coronary Computed Tomography Angiography |
| CMR | Cardiac Magnetic Resonance |
| DCM | Dilated Cardiomyopathy |
| Ea | Elastance arterial |
| Ees | Elastance end-systolic |
| ESC | European Society of Cardiology |
| GCW | Global Constructive Work |
| GDMT | Guideline-Directed Medical Treatment |
| GLS | Global Longitudinal Strain |
| GWE | Global Work Efficiency |
| GWI | Global Work Index |
| GWW | Global Wasted Work |
| HDI | Human Development Index |

| | |
|-------|--|
| HFrEF | Heart Failure with Reduced Ejection Fraction |
| LAS | Left Atrial Strain |
| LV | Left Ventricular |
| LVEF | Left Ventricular Ejection Fraction |
| LVSD | Left Ventricular Systolic Dysfunction |
| LVEDD | Left Ventricular End-Diastolic Dimension |
| LVEDV | Left Ventricular End-Diastolic Volume |
| MWI | Myocardial Work Index |
| PPCM | Peripartum Cardiomyopathy |
| PS | Pressure-Strain |
| RA | Right Atrial |
| RV | Right Ventricular |
| TAPSE | Tricuspid Annular Plane Systolic Excursion |
| VA | Ventriculo-Arterial |

Ethical Clearance

Patient consent for publication and institutional approval as per journal policy were obtained.

Acknowledgments

None.

Conflict of Interest

The author stated no conflict of interest and nothing to disclose.

Availability of Data and Materials

Not applicable.

Funding

The author has not received funding or financial support.

Copyright/Permissions for Figures

Not applicable.

Generative AI and AI-Assisted Technologies in the Writing Process

The author declares that no artificial intelligence (AI) tools were used in the writing, analysis, or preparation of this manuscript.

References

- Davis MB, Arany Z, McNamara DM, Golland S, Elkayam U. Peripartum cardiomyopathy: JACC state-of-the-art review. *J Am Coll Cardiol*. 2020; 75:207-21.
- Briasoulis A, Mocanu M, Marinescu K, Qaqi O, Palla M, Telila T, et al. Longitudinal systolic strain profiles and outcomes in peripartum cardiomyopathy. *Echocardiography* 2016; 33:1354-60.
- Biteker M, Ilhan E, Biteker G, Duman D, Bozkurt B. Delayed recovery in peripartum cardiomyopathy: An indication for long-term follow-up and sustained therapy. *Eur J Heart Fail* 2012; 14:895-901.
- Meledin V, Prameswari HS, Shimoni S, Ramon-Gonen R, Hasan M, George J, Golland S. Hemodynamic Patterns and Left Ventricular Function Recovery in Peripartum Cardiomyopathy: A Comprehensive Echocardiographic Analysis. *JACC Asia*. 2025 Apr;5(4):554-64. doi: 10.1016/j.jacasi.2024.11.019.
- Arany Z. Peripartum Cardiomyopathy. *N Engl J Med*. 2024 Jan 11;390(2):154-64. doi: 10.1056/NEJMra2306667
- Prameswari HS, Iswandi CP, Hasan M, Martanto E, Putra IC, Kamarullah W, et al. Predicting poor left ventricular function recovery in peripartum cardiomyopathy. *J Matern Fetal Neonatal Med*. 2023; 36:2279018.
- 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; 66:905-14.
- Ricci F, De Innocentiis C, Verrengia E, Ceriello L, Mantini C, Pietrangelo C, et al. The role of multimodality cardiovascular imaging in peripartum cardiomyopathy. *Front Cardiovasc Med* 2020; 7:4.
- Jackson AM, Golland S, Farhan HA, Yaseen IF, Prameswari HS, Böhm M, et al. A novel score to predict left ventricular recovery in peripartum cardiomyopathy derived from the ESC EORP Peripartum Cardiomyopathy Registry. *Eur Heart J*. 2024 Apr 21;45(16):1430-9. doi: 10.1093/eurheartj/ehad888.
- Sugahara M, Kagiya N, Hasselberg NE, et al. Global left ventricular strain at presentation is associated with subsequent recovery in patients with peripartum cardiomyopathy. *J Am Soc Echocardiogr*. 2019;32(12):1565-73.
- Moon MG, Hwang IC, Lee HJ, et al. Reverse Remodeling Assessed by Left Atrial and Ventricular Strain Reflects Treatment Response to Sacubitril/Valsartan. *JACC Cardiovasc Imaging*. 2022 Sep;15(9):1525-41. doi: 10.1016/j.jcmg.2022.03.019.
- McDonagh TA, Metra M, Adamo M, et al; ESC Scientific Document Group. 2021 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure. *Eur Heart J*. 2021 Sep 21;42(36):3599-3726. doi: 10.1093/eurheartj/ehab368.
- Iannaccone G, Graziani F, Kacar P, et al. Diagnosis and management of peripartum cardiomyopathy and recurrence risk. *Int J Cardiol Congenit Heart Dis*. 2024 Jul 19;17: 100530. doi: 10.1016/j.ijcchd.2024.100530.
- Arbelo E, Protonotarios A, Gimeno JR, Arbustini E, Barriales-Villa R, Basso C, et al; ESC Scientific Document Group. 2023 ESC Guidelines for the management of cardiomyopathies. *Eur Heart J*. 2023 Oct 1;44(37):3503-626. doi: 10.1093/eurheartj/ehad194.
- Beghini A, Sammartino AM, Papp Z, von Haehling S, Biegus J, Ponikowski P, et al. 2024 update in heart failure. *ESC Heart Fail*. 2025 Feb;12(1):8-42. doi: 10.1002/ehf2.14857.
- Asri N, Abdul-Razak S, Mokhtar MF, Ibrahim KS, Abdul Kadir RF. Delayed recognition of peripartum cardiomyopathy presenting with severe heart failure: a case report. *Eur Heart J Case Rep*. 2025 Jul 1;9(7): ytaf306. doi: 10.1093/ehjcr/ytaf306.
- Hendiperdana MR. Reverse Remodeling in Late Presenter Peripartum Cardiomyopathy: Strain and Myocardial Work Analysis. *J Cardiovasc Echogr*. 2025 Jul-Sep;35(3):280-284. doi: 10.4103/jcecho.jcecho_102_24.