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Ibnu Sina: Jurnal Kedokteran dan Kesehatan-Fakultas Kedokteran Universitas Islam Sumatera Utara

ISSN 1411-9986 (Print) | ISSN 2614-2996 (Online)

Laporan Kasus

MEMUTUS SIKLUS: BAGAIMANA TROMBOSITOSIS MEMPERBESAR RISIKO PADA KARDIOMIOPATI PERIPARTUM DENGAN DISFUNGSI KATUP BERAT

BREAKING THE CYCLE: HOW THROMBOCYTOSIS AMPLIFIES RISKS IN PERIPARTUM CARDIOMYOPATHY WITH SEVERE VALVE DYSFUNCTION

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Histori Artikel

Diterima:
19 November 2025

Revisi:
17 Desember 2025

Terbit:
1 Januari 2026

Kata Kunci

Peripartum cardiomyopathy, trombositosis, regurgitasi mitral berat, keadaan hiperkoagulabel

Keywords

Peripartum cardiomyopathy, thrombocytosis, severe mitral regurgitation, hypercoagulable state

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A B S T R A K

Peripartum cardiomyopathy (PPCM) merupakan kondisi yang jarang namun serius, ditandai oleh gagal jantung dengan penurunan fraksi ejeksi yang muncul pada akhir kehamilan atau dalam lima bulan setelah persalinan. Trombositosis dapat meningkatkan risiko tromboemboli, sementara regurgitasi mitral (MR) fungsional berat akibat dilatasi ventrikel kiri semakin memperburuk kondisi klinis. Seorang perempuan berusia 38 tahun, tiga bulan postpartum, datang dengan dispnea yang memburuk, ortopnea, paroksismal nokturnal dispnea, dan edema tungkai. Ia memiliki riwayat hipertensi dan dislipidemia. Pemeriksaan fisik menunjukkan overload cairan dan peningkatan tekanan vena jugularis. EKG menunjukkan takikardia sinus dengan perubahan ST-T, dan foto toraks menggambarkan kardiomegali dengan kongesti paru. Pemeriksaan laboratorium menemukan trombositosis (627.000/µL). Ekokardiografi menunjukkan dilatasi ventrikel kiri berat (LVEDD 63 mm), fraksi ejeksi yang sangat menurun (21%), disfungsi diastolik derajat 3, MR fungsional berat, serta spontaneous echo contrast. Kasus ini menggambarkan interaksi antara PPCM, trombositosis reaktif, dan MR berat, serta menekankan pentingnya evaluasi kardiologis–hematologis yang menyeluruh dan penatalaksanaan multidisiplin untuk mengoptimalkan luaran klinis.

A B S T R A C T

Peripartum cardiomyopathy (PPCM) is a rare but serious condition involving heart failure with reduced ejection fraction developing late in pregnancy or within five months postpartum. Thrombocytosis may increase thromboembolic risk, and severe functional mitral regurgitation (MR) from left-ventricular dilation further worsens clinical status. A 38-year-old woman, three months postpartum, presented with worsening dyspnea, orthopnea, paroxysmal nocturnal dyspnea, and leg edema. She had hypertension and dyslipidemia. Examination revealed fluid overload and elevated jugular venous pressure. ECG showed sinus tachycardia with ST-T changes, and chest X-ray demonstrated cardiomegaly with pulmonary congestion. Laboratory testing identified thrombocytosis (platelets 627,000/µL). Echocardiography revealed severe LV dilation (LVEDD 63 mm), markedly reduced ejection fraction (21%), grade 3 diastolic dysfunction, severe functional MR, and spontaneous echo contrast. This case illustrates the interaction of PPCM, reactive thrombocytosis, and severe MR, emphasizing the need for thorough cardiac–hematologic evaluation and multidisciplinary management to optimize outcomes.

DOI: http://doi.org/10.30743/ibnusina.v25i1.1068

INTRODUCTION

Peripartum cardiomyopathy (PPCM) is a rare but life-threatening form of heart failure characterized by new-onset left ventricular systolic dysfunction occurring toward the end of pregnancy or within five months postpartum. The condition is traditionally attributed to multifactorial mechanisms involving hemodynamic stress, hormonal changes, oxidative injury, and inflammatory pathways.[1] In recent years, however, growing evidence has highlighted the role of hematologic disturbances including thrombocytosis in exacerbating the already elevated thromboembolic risk inherent to PPCM. This association is clinically relevant because pregnancy induces a natural hypercoagulable state, and left ventricular dilation with reduced ejection fraction further promotes intracardiac stasis, increasing the likelihood of thrombus formation.[2]

In addition to thrombotic complications, structural cardiac abnormalities may arise as secondary consequences of ventricular remodeling. Progressive left ventricular dilation in PPCM frequently leads to functional mitral regurgitation (MR) due to annular dilation and impaired leaflet coaptation.[3] Importantly, the prothrombotic milieu associated with pregnancy and PPCM, characterized by endothelial dysfunction, systemic inflammation, and blood stasis resulting from severe left ventricular systolic impairment, may further exacerbate myocardial and valvular dysfunction.[4] Hypercoagulability and microthrombus formation within the myocardial microcirculation can impair regional perfusion, promote ischemia, and accelerate adverse ventricular remodeling, thereby indirectly worsening functional MR.[5]

Consequently, severe MR increases preload, worsens pulmonary congestion, and contributes to persistent or refractory heart failure symptoms.[3] Echocardiography, therefore, plays an essential role, not only in establishing the diagnosis of PPCM but also in quantifying ventricular function, monitoring valvular involvement, and assessing thrombotic risk markers such as spontaneous echo contrast.[6,7] The interaction between PPCM, thrombocytosis, and severe MR presents a unique clinical challenge requiring comprehensive diagnostic assessment and multidisciplinary management. The case presented below illustrates how these overlapping conditions can intensify disease severity and underscores the importance of integrated cardiovascular and hematologic evaluation to improve maternal outcomes.

CASE REPORT

The patient presented with complaints of progressively worsening shortness of breath over the past 4 months, with the most severe episode occurring 3 days before admission. This complaint has recurred three times, each requiring hospitalization. Symptoms began three months after the delivery of her third child. Before delivery, the patient reported no symptoms. Detailed information regarding antenatal care, mode of delivery, and immediate postpartum course was not available in the medical records and could not be retrospectively obtained, representing a limitation in fully characterizing obstetric risk factors associated

with the development of peripartum cardiomyopathy in this case.

Dyspnea on exertion (DOE), orthopnea (OP), and paroxysmal nocturnal dyspnea (PND) appeared in the fourth month postpartum. Currently, the patient frequently experiences lower limb edema and abdominal bloating. She was attempting to restrict fluid intake, though adherence has not been optimal. She had risk factors for cardiovascular disease, such as ex-smoker, hypertension, and dyslipidemia. She had hypertension before pregnancy. She has consumed routine medications such as furosemide 40 mg, spironolactone 25 mg, bisoprolol 2.5 mg, sacubitril/valsartan 50 mg, and acetylsalicylic acid 80 mg.

Upon referral, the patient was conscious with a blood pressure of 114/65 mmHg, pulse rate of 105 beats per minute, respiratory rate of 30 breaths per minute, temperature of 36.8°C, and peripheral oxygen saturation of 99% on nasal canula 5 Liters/minutes. Physical examination revealed elevated jugular venous pressure (5 cm+3 cmH20), minimal crackles in both lung fields, normal heart sounds, there is a systolic murmur heard at the apex, no gallop, also she has pitting edema on the lower limb.

The 12-lead electrocardiography (ECG) results showed sinus rhythm, heart rate of 109 bpm, normoaxis, normal P wave with a PR interval of 0.20 seconds, QRS duration of 0.08 seconds, and hyperacute T waves in leads V2-V4 (dynamic ST-T changes). The chest X-ray revealed pulmonary congestion and cardiomegaly.

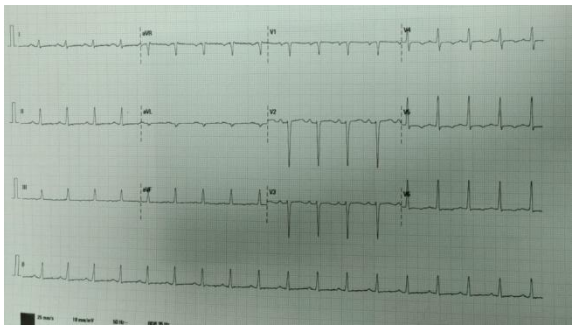

Figure 1. The 12-lead electrocardiography From the Patient.

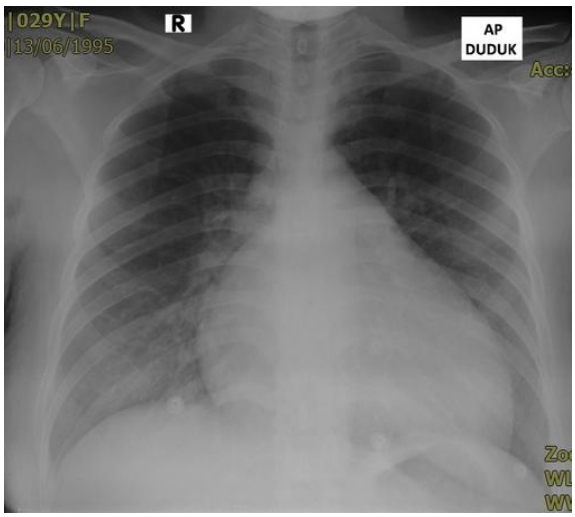

Figure 2. Chest X-Ray showed Cardiomegaly and Pulmonary Congestion.

Laboratory evaluation demonstrated hemoglobin of 11.6 g/dL and hematocrit of 36.6%, indicating no severe anemia. The leukocyte count was 11,250/µL. A markedly elevated platelet count of 627,000/µL was noted, consistent with thrombocytosis. Renal function was preserved, with urea of 20 mg/dL and creatinine of 0.75 mg/dL. Serum electrolytes were within acceptable ranges (sodium 138 mmol/L, potassium 3.5 mmol/L, chloride 106 mmol/L). Blood glucose was 115 mg/dL. Cardiac biomarker assessment showed a negative troponin T level, indicating no evidence of acute myocardial injury. Overall, laboratory findings did not reveal alternative metabolic,

renal, or ischemic precipitants of heart failure, highlighting thrombocytosis as the most prominent hematologic abnormality in this case.

The patient was subsequently diagnosed with acute decompensated heart failure with a wet and warm profile due to PPCM, mitral regurgitation, thrombocytosis, and controlled hypertension. The patient was then placed in a regular ward and given the following medications: sacubitril/valsartan 2x50 mg, spironolactone 1x25 mg, aspirin 1x80 mg, bisoprolol 1x2.5 mg, lactulosa syrup 1x1C, diazepam 1x3 mg, and furosemide 3x20 mg intravenously. Finally, the patient were performed echocardiography. The transthoracic echocardiogram demonstrated a dilated cardiomyopathy, characterized by a severely dilated left ventricle (LVEDD 63 mm) with eccentric left ventricular hypertrophy (LVMI 138 g/m²) and a dilated left atrium (41 mm). Left ventricular systolic function was severely impaired, with global hypokinesia and an ejection fraction of 21%, accompanied by evidence of Grade 3 diastolic dysfunction (E/A > 2, DT 116 ms). Significant valvular findings included severe mitral regurgitation secondary to annular dilatation and mild-to-moderate tricuspid regurgitation (TVG 37 mmHg). Notably, spontaneous echo contrast (SEC) was present in the left ventricle. Right ventricular systolic function was adequate (TAPSE 16 mm), and the patient's volume status was euvolemic, as suggested by an IVC collapse of over 40%.

Based on the echocardiography results, the patient was then given additional medication: dapagliflozin 1x10 mg, warfarin 1x2 mg, and bromocriptine 2x2.5 mg for seven days. Furosemide was then titrated down from 3x20 mg to 2x10 mg, and it was planned to stop it. Other heart failure medications were optimized. After 2 days of treatment, the patient's shortness of breath improved, jugular venous pressure appeared normal, there were no rales in the lungs, and there was no pitting edema. The patient was then discharged and scheduled for a follow-up appointment in 1 week. After 1 week, After 1 week, the patient returned for a follow-up and had no complaints. However, after being scheduled for the next follow-up, the patient did not return/was lost to follow-up.

DISCUSSIONS

Peripartum cardiomyopathy (PPCM) is an uncommon but life-threatening form of heart failure manifesting as left ventricular (LV) systolic dysfunction toward the end of pregnancy or in the early postpartum period in previously healthy women.[8-12] PPCM is a diagnosis of exclusion based on clinical presentation and echocardiographic evidence of reduced left ventricular ejection fraction (LVEF <45%) with no other identifiable causes of heart failure, as emphasized by the ESC guidelines on cardiovascular diseases during pregnancy.[11,13] Patients with suspicion of PPCM should therefore undergo a comprehensive evaluation, beginning with detailed history taking and followed by appropriate diagnostic testing such as echocardiography. Early recognition and prompt initiation of guideline-directed management in severe PPCM have been shown to improve cardiac function and overall outcomes.[14] The exact etiology of PPCM remains incompletely understood but is

recognized as multifactorial, involving genetic predisposition, inflammatory myocarditis, oxidative stress, and hormonal factors such as prolactin cleavage. The core pathophysiological mechanism involves unbalanced oxidative stress leading to proteolytic cleavage of the nursing hormone prolactin from its benign 23-kDa form to a toxic 16-kDa fragment. The 16-kDa fragment has antiangiogenic and proapoptotic properties, contributing to myocardial injury.[9,11,12,15] The ESC guidelines also highlight the role of vascular factors and systemic inflammation.[9,11] The pathogenesis involves dual vascular insults.[12,16] First, cardiac-specific deletion of transcriptional regulators such as STAT3 or PGC-1α reduces expression of antioxidant genes, promoting 16-kDa prolactin formation.[16] Second, placental secretion of soluble Fms-like tyrosine kinase-1 (sFlt-1) during late gestation antagonizes VEGF, creating a profound anti-angiogenic environment that exacerbates microvascular rarefaction.[15]

Thrombocytosis is not a classical feature of PPCM but may occur as a reactive process due to systemic inflammation, endothelial dysfunction, and oxidative stress inherent to PPCM.[9,11] In the present case, the absence of prior hematologic disease and the acute clinical context favor reactive thrombocytosis rather than essential thrombocythemia, although definitive myeloproliferative work-up was not performed. Essential thrombocythemia (ET) during pregnancy creates a complex hypercoagulable state superimposed on pregnancy's inherent prothrombotic changes.[17,18]

The hypercoagulable state of pregnancy, combined with blood stasis from LV hypokinesis, fosters a prothrombotic environment.[9,11] Reactive thrombocytosis is characterized by an elevated platelet count secondary to underlying conditions such as inflammation or systemic disease rather than clonal myeloproliferation. It is generally a benign condition, although in rare cases with markedly elevated platelet counts or additional prothrombotic factors it may be associated with an increased risk of thrombotic events.[19]

Within the myocardial microcirculation, platelet-rich microthrombi may impair regional perfusion and exacerbate ischemia, which can contribute to inflammatory injury and potentially promote adverse ventricular remodeling.[20] This process can indirectly worsen functional mitral regurgitation through progressive left ventricular remodeling and displacement of subannular structures, including the papillary muscles, leading to impaired leaflet coaptation and annular dilatation. This maladaptive remodeling may contribute to a vicious cycle of ventricular dysfunction and valvular incompetence, with potential downstream implications for thrombotic risk in advanced disease.[21]

Large observational studies have shown that thrombocytosis is independently associated with an increased risk of major adverse cardiovascular events, with adjusted odds ratios ranging from approximately 1.6 to 1.9 depending on platelet count severity. Although PPCM-specific data are limited, the coexistence of thrombocytosis, severely reduced left ventricular ejection fraction, and spontaneous echo contrast in this patient likely represents a

synergistic prothrombotic state, supporting antithrombotic therapy in selected high-risk cases.[22]

In line with these mechanistic considerations, in our case, the presence of SEC in the LV, together with marked thrombocytosis and severe left ventricular systolic dysfunction, suggests a clinically prothrombotic milieu characterized by pregnancy-related hypercoagulability and intracardiac blood stasis due to ventricular hypokinesis. The reactive thrombocytosis thus potentially exacerbates the risk of thromboembolic complications, underscoring the need for vigilant anticoagulation management as recommended by comprehensive PPCM management protocols.[9,11]

Severe mitral regurgitation in PPCM generally arises as a functional complication due to LV dilation and remodeling.[9,11,23] The resultant mitral annular dilation and papillary muscle displacement impair leaflet coaptation, increasing regurgitant volume.[9,11,23] This further worsens volume overload and contributes to left atrial enlargement, pulmonary congestion, and atrial arrhythmias, thereby complicating the clinical picture and worsening heart failure symptoms.[9,11]

The management of PPCM complicated by thrombocytosis and severe mitral regurgitation requires a multidisciplinary and severity-guided approach, integrating heart failure treatment, thrombosis risk mitigation, and valvular pathology management.[9,13,24,25] PPCM patients benefit from a combined team of cardiologists, obstetricians, intensivists, and neonatologists to optimize both maternal and fetal outcomes. Management should be individualized based on clinical presentation and echocardiographic findings.[9,13,24] According to ESC and up-to-date clinical reviews, PPCM can be stratified by severity: Mild PPCM: LVEF 30–45%, stable hemodynamics; managed outpatient or in a ward.[9,24] Moderate PPCM: LVEF 20–35%, with significant symptoms but stable; usually admitted to heart failure units.[9,24] Severe PPCM: LVEF <20%, with cardiogenic shock or respiratory failure; requires intensive care with advanced hemodynamic support.[9,24]

Standard heart failure therapies remain the cornerstone of management, including beta-blockers, ACE inhibitors or ARBs, diuretics, and vasodilators when indicated. In selected patients, bromocriptine may be used to inhibit prolactin secretion, mitigating oxidative myocardial damage; however, it requires concomitant anticoagulation due to increased thrombotic risk.[9,24,26] Patients with LVEF ≤35% should receive prophylactic anticoagulation, preferably low-molecular-weight heparin during pregnancy and early postpartum, transitioning to warfarin or direct oral anticoagulants postpartum if breastfeeding status and individual risks are low. Anticoagulation is especially critical if bromocriptine therapy is administered due to reported thrombotic events.[9] For the severe mitral regurgitation management in PPCM, the causes of MR may be due to LV dilation exacerbates volume overload, worsening heart failure.[9,11,24] Severe symptomatic MR refractory to medical therapy may require surgical repair or replacement, if the patient's hemodynamic condition permits.[9,11,24] Percutaneous edge-to-edge repair (e.g., MitraClip) may be considered

as a less invasive option in high-risk patients. For long-term follow-up and lifestyle, the patient should be informed that regular echocardiographic is needed every 3–6 months until LV function normalizes, contraception counseling is important; estrogen-containing contraceptives are generally avoided in the early postpartum due to thrombotic risk.[9,25]

Previously reported cases of peripartum cardiomyopathy illustrate the wide spectrum of clinical outcomes. A reported case of a multiparous woman with peripartum cardiomyopathy demonstrated that prognosis is closely related to recovery of left ventricular function, with approximately 30% of patients achieving normalization of ventricular function within six months and up to 50% showing clinical improvement. Recurrence in subsequent pregnancies was reported in 30–50%, emphasizing the importance of long-term counseling and appropriate contraception.[27]

In contrast, another case highlighted severe thromboembolic complications, in which a young woman with peripartum cardiomyopathy developed cardioembolic stroke, underscoring the critical role of anticoagulation in patients with severe ventricular dysfunction.[28] Compared with these reports, the present case highlights thrombocytosis and spontaneous echo contrast as additional factors that may further increase thromboembolic risk.

Despite short-term clinical improvement following optimized heart failure therapy, the long-term prognosis of peripartum cardiomyopathy is largely determined by recovery of left ventricular systolic function.[29] Severely reduced left ventricular ejection fraction at presentation, the presence of spontaneous echo contrast, and concomitant hematologic abnormalities such as thrombocytosis have been associated with an increased risk of thromboembolic events and adverse outcomes.[30] Consequently, the inability to evaluate long-term ventricular recovery and clinical outcomes in this patient due to loss to follow-up represents an important limitation of this case.

Although the therapeutic approach in this case largely followed current guideline-directed management for peripartum cardiomyopathy, the novelty of this report lies in the unique constellation of diagnostic findings and clinical reasoning. The coexistence of marked thrombocytosis, severely reduced left ventricular ejection fraction, and spontaneous echo contrast in the absence of overt intracardiac thrombus represents an uncommon presentation that underscores a heightened prothrombotic risk. This case highlights the importance of integrating echocardiographic markers, hematologic abnormalities, and hemodynamic impairment to identify high-risk PPCM patients who may benefit from early antithrombotic intervention despite the absence of documented thromboembolic events. As such, this report provides practical insights into risk stratification and management considerations that are not explicitly addressed in current guidelines.

CONCLUSION

Peripartum cardiomyopathy (PPCM) is a rare but potentially fatal cause of heart failure occurring late in pregnancy or early postpartum.

While traditionally attributed to hemodynamic and hormonal factors, emerging evidence highlights the significant contribution of hematologic abnormalities such as thrombocytosis, which can exacerbate the already elevated risk of thromboembolism in PPCM patients. Additionally, left ventricular dilation and dysfunction in PPCM often lead to functional mitral regurgitation (MR), further worsening heart failure symptoms and complicating management.

This case emphasizes the critical role of comprehensive evaluation, including echocardiography, to assess ventricular function, valvular involvement, and thrombotic risk markers such as spontaneous echo contrast. Management requires a multidisciplinary approach tailored to disease severity, involving optimized heart failure therapy, careful anticoagulation, especially in the setting of thrombocytosis and bromocriptine use, and timely intervention for severe mitral regurgitation when indicated. Early diagnosis and integrated treatment strategies are essential to improve maternal outcomes, while regular follow-up with echocardiographic monitoring and lifestyle counseling remains crucial for long-term recovery and prevention of complications.

CONFLICT OF INTEREST

The authors declare that there was no conflict of interest regarding the publication of this paper.

ACKNOWLEDGMENTS

The authors would like to express their gratitude to all individuals involved in the care of the patient. No specific acknowledgments are applicable.

FUNDING SOURCES

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

REFERENCES

1. Rodriguez Ziccardi M, Siddique MS. Peripartum Cardiomyopathy. *StatPearls*. StatPearls Publishing

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2. Badge M, Nagashweta L, Rajgopalan K, Görlinger K, Kapoor P. Peripartum Cardiomyopathy and Thrombosis – A Match Made In Hell! *Journal of Cardiac Critical Care TSS*. 05/05 2025;9:1-4. doi:10.25259/JCCC_47_2024

3. Douedi S, Douedi H. Mitral Regurgitation. *StatPearls*. StatPearls Publishing

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4. Sigauke FR, Ntsinjana H, Tsabedze N. Peripartum cardiomyopathy: a comprehensive and contemporary review. *Heart Fail Rev*. Nov 2024;29(6):1261-1278. doi:10.1007/s10741-024-10435-5

5. Guagliumi G, Sonzogni A, Pescetelli I, Pellegrini D, Finn AV. Microthrombi and ST-Segment-Elevation Myocardial Infarction in COVID-19. *Circulation*. Aug 25 2020;142(8):804-809. doi:10.1161/circulationaha.120.049294

6. Greatorex B, Colebourn C, Ormerod O. Echocardiographic assessment and critical care management of peri-partum women with unexpected left ventricular failure. *J Intensive Care Soc*. May 2022;23(2):210-221. doi:10.1177/1751143720978862

7. Ricci F, De Innocentiis C, Verrengia E, et al. The Role of Multimodality Cardiovascular Imaging in Peripartum Cardiomyopathy. *Front Cardiovasc Med*. 2020;7:4. doi:10.3389/fcvm.2020.00004

8. Trivedi J, Goyal VK, Sohail, et al. Peripartum Cardiomyopathy (PPCM) – A Comprehensive Review. *The Journal of Hypertension*. 2025;11(1)doi:10.52338/tjoht.2025.4774
9. Farroni E, Taggiasco A, Santulli G. Peripartum cardiomyopathy: a clinical review. *Vessel Plus*. 2025;9:2. doi:10.20517/2574-1209.2025.15
10. Tukeni KN, Asefa ET, Woyimo TG, Gudina EK, Estner H, Haas NA. Peripartum cardiomyopathy: a review of prevalence and treatment trends from an African perspective. *Front Cardiovasc Med*. 2025;12:1568493. doi:10.3389/fcvm.2025.1568493
11. Davis MB, Arany Z, McNamara DM, Goland S, Elkayam U. Peripartum Cardiomyopathy: JACC State-of-the-Art Review. *J Am Coll Cardiol*. Jan 21 2020;75(2):207-221. doi:10.1016/j.jacc.2019.11.014
12. Laksono S, Nandifa VNP. Case Report: Diagnosis and Management of Peripartum Cardiomyopathy. *International Islamic Medical Journal*. 2023;5(1):36-44.
13. Iannaccone G, Graziani F, Kacar P, et al. Diagnosis and management of peripartum cardiomyopathy and recurrence risk. *Int J Cardiol Congenit Heart Dis*. Sep 2024;17:100530. doi:10.1016/j.ijcchd.2024.100530
14. Laksono S, Prawara AS, Halomoan R. Diagnosis and management of severe peripartum cardiomyopathy. *Indonesian Journal of Obstetrics and Gynecology*. 2021:215-218.
15. Arany Z, Elkayam U. Peripartum Cardiomyopathy. *Circulation*. 2016/04/05 2016;133(14):1397-1409. doi:10.1161/CIRCULATIONAHA.115.020491
16. Cowgill JA, Francis SA, Sawyer DB. Anthracycline and Peripartum Cardiomyopathies. *Circulation Research*. 2019/05/24 2019;124(11):1633-1646. doi:10.1161/CIRCRESAHA.119.313577
17. Nugraheni A, Wijayanti K, Adnyana I. Essential Thrombocythemia in Pregnancy: A Case Report. *European Journal of Medical and Health Sciences*. 12/29 2023;5:22-26. doi:10.24018/ejmed.2023.5.6.1978
18. İskender D, Yılmaz-Ergani S, Aksoy M, et al. High Rate of Obstetric Complications in Patients With Essential Thrombocythemia. *Cureus*. Dec 2021;13(12):e20449. doi:10.7759/cureus.20449
19. Rokkam VR, Killeen RB, Kotagiri R. Secondary Thrombocytosis. *StatPearls*. StatPearls Publishing

20. Hałucha K, Rak-Pasikowska A, Bil-Lula I. Protective Role of Platelets in Myocardial Infarction and Ischemia/Reperfusion Injury. *Cardiology Research and Practice*. 05/25 2021;2021:1-14. doi:10.1155/2021/5545416
21. Van Laethem AJ, Figiel J, Mahnken AH, et al. Topography of the Papillary Muscles in the Mitral Valve Complex and Their Relevance for Mitral Valve Function. *J Cardiovasc Dev Dis*. Sep 11 2025;12(9)doi:10.3390/jcdd12090348
22. Wilcox T, Smilowitz NR, Berger JS. Association between preoperative platelet count and perioperative cardiovascular events after noncardiac surgery. *Journal of Thrombosis and Haemostasis*. 2025/07/01/ 2025;23(7):2314-2321. doi:https://doi.org/10.1016/j.jtha.2025.04.011
23. Padron E, Waldrop AR, Minor K, et al. Peripartum cardiomyopathy with severe mitral regurgitation: A novel application of an intra-aortic balloon pump as a bridge to recovery. *Open J Clin Med Case Rep*. 2022;8(15):1907.
24. Arbelo E, Protonotarios A, Gimeno JR, et al. 2023 ESC Guidelines for the management of cardiomyopathies. *Eur Heart J*. Oct 1 2023;44(37):3503-3626. doi:10.1093/eurheartj/ehad194
25. Abdelmajide R, Marouane B, Taheri H, Saadi H, Mimouni A. Peripartum cardiomyopathy: a case report and review of current diagnostic and therapeutic strategies. *International Journal of Reproduction, Contraception, Obstetrics*

and Gynecology. 07/29 2025;14:2776-2780. doi:10.18203/2320-1770.ijrcog20252360

26. Soesanto V, Jonathan B. Diagnosis dan Tata Laksana Kardiomiopati Peripartum: Tinjauan Pustaka. *Cermin Dunia Kedokteran*. 04/07 2025;52:236-240. doi:10.55175/cdk.v52i4.1244
27. Firman, Hanif A. Peripartum Cardiomyopathy: A Case Report. *Bioscientia Medicina : Journal of Biomedicine and Translational Research*. 12/20 2023;8:4123-4126. doi:10.37275/bsm.v8i3.940
28. Pradhan R, Yadav A, Yadav S, Gupta P. Case Report: Peripartum cardiomyopathy in a young female complicated by cardioembolic stroke [version 1; peer review: 1 approved with reservations, 1 not approved]. *F1000Research*. 2019;8(2135)doi:10.12688/f1000research.21712.1
29. Jha N, Jha AK. Peripartum cardiomyopathy. *Heart Failure Reviews*. 2021/07/01 2021;26(4):781-797. doi:10.1007/s10741-020-10060-y
30. Glavinovic T, Cheung DY, Cordova Perez FJ, Soni A, Memauri B, Jassal DS. Right ventricular free wall dyskinesis in the setting of left ventricular non compaction: a case report. *BMC Res Notes*. Nov 5 2014;7:787. doi:10.1186/1756-0500-7-787