Hearts Under Pressure: Challenging The Myocardium — A Critical Care Perspective

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Abstract
Cardiac complications arising in the peripartum period represent a rare but life-threatening constellation of disorders that challenge multidisciplinary teams in critical care settings. We present three cases of acute cardiomyopathy in young obstetric patients: (1) Peripartum Cardiomyopathy
(PPCM) with Posterior Reversible Encephalopathy Syndrome (PRES) following eclampsia; (2) Reverse Takotsubo Cardiomyopathy (TTS) precipitated by the stress of emergency abdominal surgery in the postpartum period; and (3) a case of Dilated Cardiomyopathy (DCM) diagnosed antenatally at 30 weeks of gestation. All three cases highlight the critical importance of timely echocardiographic evaluation, guideline-directed medical therapy, and a coordinated multidisciplinary approach.
Prompt diagnosis and vigilant haemodynamic monitoring led to either complete recovery or
significant clinical improvement in all patients. These cases underscore the need for clinicians to maintain a high index of suspicion for reversible and non-reversible cardiomyopathies in the peripartum setting.

Keywords: Peripartum Cardiomyopathy, Takotsubo Syndrome, Reverse Takotsubo, Dilated Cardiomyopathy, Eclampsia, PRES, Obstetric Critical Care, Echocardiography, ESC Guidelines

Introduction

Peripartum cardiomyopathy (PPCM) is a potentially fatal form of dilated cardiomyopathy that occurs in the last month of pregnancy or within five months following delivery, in the absence of pre- existing cardiac disease or identifiable causes of heart failure.[1] Its global incidence ranges from
1:1,000 to 1:4,000 live births in Western countries, with notably higher rates in sub-Saharan Africa (1:100 in Nigeria and 1:300 in Haiti).[2] Despite advances in medical care, PPCM remains a major contributor to maternal cardiac mortality and morbidity worldwide.[3]
Takotsubo Syndrome (TTS), also known as stress cardiomyopathy or apical ballooning syndrome, is an acute, reversible cardiomyopathy characterised by transient left ventricular wall motion abnormalities, predominantly triggered by emotional or physical stressors.[4] The classic variant exhibits apical ballooning with preserved basal function, whereas the reverse (basal) variant — more frequently observed in younger women — demonstrates basal and mid-ventricular akinesia with a hyperdynamic apex.[5] Both forms are triggered by catecholamine surges that mimic acute coronary syndrome (ACS), and both are diagnoses of exclusion requiring coronary angiography or CT coronary angiography to rule out obstructive coronary artery disease.[6]
Dilated cardiomyopathy (DCM) presenting during pregnancy imposes additional physiological burden on an already haemodynamically stressed cardiovascular system. The physiological increases in plasma volume and cardiac output during pregnancy may unmask or exacerbate pre-existing DCM.[7]
Management requires a delicate balance between maternal cardiac optimisation and fetal wellbeing, avoiding teratogenic medications while maintaining adequate haemodynamic support.[8]
This case series aims to: (1) illustrate the clinical heterogeneity and diagnostic challenges of acute cardiomyopathies in the peripartum period; (2) describe evidence-based multidisciplinary management strategies; and (3) reflect on pharmacological safety profiles in pregnancy and lactation, counselling for future pregnancies, and ethical considerations in complex obstetric cardiac cases.[9]

CASE REPORTS

Case 1: Peripartum Cardiomyopathy with Posterior Reversible Encephalopathy Syndrome (PRES) Following Eclampsia

A 28-year-old primigravida at 33 weeks of gestation was brought to the emergency department with eclamptic seizures and severe hypertension. She was commenced on magnesium sulphate for seizure prophylaxis and high-risk obstetric consent was obtained. Given the acute clinical
deterioration, an emergency preterm lower segment caesarean section (LSCS) was performed on 17/10/2024.
Intraoperatively and in the immediate postoperative period, the patient exhibited persistently elevated blood pressure with progressive respiratory compromise, developing frank pulmonary oedema manifesting as pink frothy sputum — a hallmark of acute decompensated heart failure.[10] A bedside echocardiogram performed on postoperative day 0 (POD-0) revealed dilated cardiomyopathy with severe left ventricular (LV) systolic dysfunction: left ventricular ejection fraction (LVEF) of 20%, Grade II left ventricular diastolic dysfunction (LVDD), and a markedly elevated B-type

natriuretic peptide (BNP) of 7,200 pg/mL. Troponin levels were within normal limits, effectively excluding an ischaemic aetiology. The patient was promptly transferred to the intensive care unit (ICU), where she was initiated on intravenous diuretics, glyceryl trinitrate (GTN) infusion, followed by carvedilol and vasodilators once haemodynamically stable.[11] Serial echocardiograms on POD-1 and POD-2 demonstrated progressive improvement: LVEF rose to 38–40%, with global LV hypokinesia showing an apical predominance pattern consistent with PPCM.

Concurrently, the patient developed episodic severe headaches with hypertensive spikes. MRI Brain demonstrated features consistent with Posterior Reversible Encephalopathy Syndrome (PRES), a condition well recognised in the setting of eclampsia and hypertensive emergency.[12]

Electroencephalogram (EEG) was normal. Levetiracetam was commenced for seizure control and DVT prophylaxis was initiated. Antihypertensive therapy was optimised with Nicardia-R, Isolazine, and Labetalol.

At pre-discharge echocardiography (04/11/2024), the patient demonstrated complete recovery of LV systolic function: LVEF 62%, mild mitral regurgitation (MR) and tricuspid regurgitation (TR), with moderate pulmonary arterial hypertension (PAH). Temporary contraception was initiated and cardiac medications were continued for two additional weeks per cardiology advice. At the 3-month follow- up, the patient remained asymptomatic with complete radiological resolution of PRES and normal cardiac function.

Case 2: Reverse Takotsubo Cardiomyopathy in the Postpartum Perioperative Setting

A 28-year-old woman (G2P1L1) with a history of previous preterm LSCS at 32 weeks presented with acute abdominal pain, bilious vomiting, and abdominal distension. Clinical and radiological assessment established a diagnosis of adhesive intestinal obstruction. She underwent emergency laparotomy with adhesiolysis and ileal resection.

During extubation in the immediate postoperative period, she developed acute haemodynamic compromise: heart rate of 38 beats per minute and blood pressure of 60/40 mmHg. Twelve-lead ECG revealed transient ST-segment depression. Peak troponin-I was elevated at 224 ng/L, raising concern for an acute ischaemic event.[13]

An urgent echocardiogram demonstrated basal and mid-ventricular hypokinesia with preserved apical contractility and an LVEF of 33%. This pattern — notably the inverse of the classic Takotsubo apical ballooning — was strongly suggestive of Reverse Takotsubo Cardiomyopathy.[5] CT Coronary Angiography excluded obstructive coronary artery disease, thereby confirming the diagnosis of stress-induced (reverse) Takotsubo Syndrome precipitated by the physiological and emotional stress of emergency surgery.[6]

Haemodynamic stabilisation was achieved with cautious intravenous fluid resuscitation and norepinephrine infusion, which was successfully weaned within six hours. Oral metoprolol and ivabradine were initiated.[14] Serial echocardiograms demonstrated rapid recovery: LVEF improved to 52% by Day 2 and to 63% by Day 6. The patient was discharged haemodynamically stable and asymptomatic on oral beta-blockers with temporary contraception. At follow-up, there was no recurrence of symptoms and complete functional cardiac recovery was documented.

Case 3: Dilated Cardiomyopathy Presenting at 30 Weeks of Gestation

A 21-year-old woman (G2P1L1) with a prior LSCS presented to the outpatient department at 30 weeks of gestation with an outside echocardiogram report suggestive of dilated cardiomyopathy. Repeat echocardiography at our institution revealed global LV hypokinesia (without regional wall motion abnormality), LVEF of 45%, mild mitral regurgitation, dilated left atrium, and normal right ventricular function with no evidence of pulmonary arterial hypertension.[7]

Cardiology evaluation confirmed the diagnosis of DCM and advised early termination of pregnancy given the risk of progressive haemodynamic decompensation. A preterm emergency LSCS with concomitant tubal sterilisation was performed. The patient was transferred to the ICU
postoperatively.

Postnatal cardiac assessments on POD-0 and POD-6 demonstrated no significant change in echocardiographic parameters. Initial medical management comprised Tablet Hydralazine 25 mg (half tablet twice daily) and Tablet Carvedilol 3.125 mg twice daily, consistent with pharmacological safety profiles in the lactating patient.[8] On POD-6, the patient reported intermittent headaches; neurological evaluation including MRI Brain was normal.

At late follow-up (POD-88), repeat echocardiography showed similar findings with no recovery of LVEF. Therapy was escalated: Tablet Vymada (sacubitril/valsartan) 50 mg twice daily, Tablet Lasilactone 20/50 (half tablet once daily), and Tablet Dapagliflozin 10 mg once daily were commenced, while Hydralazine was discontinued.[15] The patient was subsequently readmitted for further cardiac optimisation and is currently under close follow-up with ongoing improvement.

Discussion

Pathophysiology

The pathogenesis of PPCM is best explained by the "Two-Hit Hypothesis," which integrates vascular, hormonal, and oxidative stress mechanisms.[1,3] Under normal physiological conditions, STAT3
activation in cardiomyocytes during pregnancy confers cytoprotection. In susceptible women, reduced STAT3 expression leads to excessive accumulation of reactive oxygen species (ROS), which in turn activates Cathepsin D — a lysosomal protease — promoting cleavage of full-length prolactin (23- kDa) into its pathological 16-kDa fragment.[3]

The 16-kDa prolactin fragment exerts potent anti-angiogenic and pro-apoptotic effects: it induces endothelial dysfunction, promotes capillary rarefaction, impairs myocardial perfusion, and upregulates microRNA-146a — a non-coding RNA that blocks cardioprotective signalling cascades, ultimately culminating in cardiac myocyte death.[1,3] Simultaneously, placental secretion of soluble FMS-like tyrosine kinase-1 (sFlt-1), which normally declines after delivery, persists in PPCM patients. sFlt-1 inhibits vascular endothelial growth factor (VEGF) and placental growth factor (PlGF), thereby maintaining an anti-angiogenic state and perpetuating systemic endothelial injury.[3]

This shared endothelial injury mechanism provides a compelling biological explanation for the strong clinical

association between PPCM and pre-eclampsia (present in approximately 22% of PPCM cases), as seen in Case 1.[2] The therapeutic rationale for bromocriptine — a dopamine agonist that
suppresses prolactin secretion — derives directly from this pathophysiological model: by preventing prolactin cleavage, bromocriptine limits 16-kDa fragment formation and attenuates myocardial injury.[1,16]
In TTS, the pathophysiology centres on an exaggerated catecholamine surge — whether from
emotional distress, physical illness, or surgical stress — causing direct myocardial toxicity through adrenergic receptor-mediated mechanisms.[4,6] This leads to transient myocardial stunning,
characterised by the distinctive wall motion pattern seen on echocardiography. In the classic form, catecholamine-induced microvascular spasm preferentially affects the apical myocardium (which has a higher density of sympathetic innervation), causing apical ballooning.[5] The reverse variant, as
observed in Case 2, reflects basal predominance of adrenergic stimulation — a phenomenon more frequently observed in younger women and in the perioperative setting.[6,13]

Epidemiology and Risk Factors

PPCM has a global incidence of 1:1,000–1:4,000 pregnancies in Western countries.[2] Notably higher rates are documented in sub-Saharan Africa: 1:100 in Nigeria and 1:300 in Haiti, reflecting the influence of genetic, socioeconomic, and nutritional risk factors.[2] Established risk factors include Afro-Caribbean/Black ethnicity, advanced maternal age (>30 years), multiparity, multiple gestation, obesity, chronic hypertension, and pre-eclampsia.[1,2,3] The condition disproportionately affects younger women of childbearing age, creating significant long-term implications for family planning and subsequent pregnancies.
TTS accounts for 1–3% of all patients presenting with suspected ACS and has a strong female predominance (>90% of cases), with the majority occurring in post-menopausal women.[4] However, perioperative and peripartum TTS in younger women, as illustrated in Case 2, is increasingly
recognised as the anaesthetic and surgical community's awareness of this entity grows.[6,13] Reverse TTS specifically occurs in approximately 20–25% of all TTS cases and shares the same catecholamine- mediated mechanism with the classic form.[5]

Diagnostic Approach

PPCM remains a diagnosis of exclusion.[1] The European Society of Cardiology (ESC) position statement defines PPCM as heart failure occurring in the last month of pregnancy or within five months
postpartum, in the absence of pre-existing structural heart disease, with echocardiographic evidence of LV systolic dysfunction (LVEF <45%).[1,9]
Key investigations include: ECG (non-specific but excludes ischaemia and pulmonary embolism), serum biomarkers (BNP/NT-proBNP, troponin, full blood count, CRP), chest X-ray, and
echocardiography — the cornerstone of diagnosis and follow-up.[9,11] Cardiac MRI may provide additional tissue characterisation in equivocal cases. Endomyocardial biopsy is reserved for refractory cases where inflammatory myocarditis is suspected.[1,7]
The diagnosis of TTS in Case 2 required systematic exclusion of ACS. Coronary CT Angiography was the diagnostic modality of choice given the postoperative setting, confirming the absence of obstructive coronary artery disease while simultaneously revealing the characteristic
echocardiographic wall motion pattern.[4,6] The Inter-TAK Diagnostic Criteria and Score — integrating clinical, ECG, biomarker, and echocardiographic data — provide a structured framework for TTS
diagnosis that may help clinicians in similar presentations.[4]
Differential diagnoses that must be systematically excluded include: benign dyspnoea of pregnancy, arrhythmias, atrial fibrillation, asthma, HIV-associated cardiomyopathy, pre-existing valvular heart disease unmasked by pregnancy, hypertensive heart disease, acute coronary syndromes (including spontaneous coronary artery dissection), and pulmonary embolism.[9,10]

Management Strategies

Management of PPCM and related cardiomyopathies in the peripartum setting mandates a multidisciplinary approach involving obstetricians, cardiologists, anaesthetists, neonatologists, and intensivists.[9,11]
Pharmacotherapy must navigate the competing demands of maternal haemodynamic optimisation and fetal/neonatal safety. Beta-blockers (with metoprolol preferred in pregnancy) are safe in both pregnancy and lactation and are essential components of guideline-directed medical therapy.[8,9] ACE inhibitors and ARBs, while central to heart failure management in the general population, are absolutely contraindicated during pregnancy due to teratogenicity (oligohydramnios, renal
dysgenesis, limb defects, and neonatal death); however, enalapril and captopril are considered relatively safe during lactation.[8,9]
The ESC EORP PPCM Registry and subsequent randomised controlled trials (the BOARD trial) have provided evidence supporting bromocriptine at a dose of 2.5 mg twice daily for two weeks, followed by 2.5 mg once daily for six weeks, as an adjunct therapy to suppress prolactin-mediated myocardial injury.[16] Given bromocriptine's prothrombotic effect (by suppressing prolactin), anticoagulation with low-molecular-weight heparin (LMWH) is recommended concurrently in all patients receiving bromocriptine and in those with LVEF ≤35%.[1,9]
For cardiogenic shock refractory to initial therapies, levosimendan (a calcium sensitiser and potassium channel opener) at 0.1 μg/kg/min for 24 hours is preferred over catecholamine inotropes as the first-line inotropic agent, given the theoretical concern that catecholamines may exacerbate TTS through adrenergic overstimulation.[9,17]
In Case 3, the escalation to sacubitril/valsartan (Vymada) — an angiotensin receptor-neprilysin inhibitor (ARNI) — reflects the 2023 ESC Guidelines for cardiomyopathies, which recommend ARNI

for HFrEF patients with LVEF <40% who remain symptomatic despite optimal ACEi/ARB therapy.[15] The addition of dapagliflozin — an SGLT2 inhibitor — is supported by robust trial data (DAPA-HF, EMPEROR-Reduced) demonstrating significant reductions in HF hospitalisations and cardiovascular mortality.[15]

Prognosis, Subsequent Pregnancies, and Contraception

The prognosis of PPCM is variable and depends critically on LVEF at diagnosis and speed of recovery. Approximately 50–75% of patients with PPCM recover LVEF to ≥55% within 6–12 months.[1,3] TTS carries a more favourable prognosis: 70–90% of patients normalise their LVEF within 4–8 weeks.[4,5]

Both cases in this series demonstrated rapid and complete recovery, highlighting the importance of early recognition and guideline-directed therapy in achieving favourable outcomes.

Counselling regarding subsequent pregnancies is a critical component of long-term management. Both the ESC and AHA recommend advising women against future pregnancy unless LVEF has fully normalised. Women with LVEF <25% at diagnosis carry particularly high relapse risk and should be strongly counselled against future conception.[1,9] Even with normalised LVEF, a residual 20% relapse risk persists in subsequent pregnancies.[1] Pre-pregnancy planning should include discontinuation of teratogenic agents (ACEi/ARB/MRA) with substitution of hydralazine–nitrate, early antenatal booking with consultant-led care, continuation of beta-blockers, serial echocardiography and BNP monitoring, and consideration of LMWH anticoagulation.[9] Regarding contraception, intrauterine devices (IUDs) are the preferred method given the absence of thromboembolic risk. Combined oral contraceptives (COCs) should be avoided due to their prothrombotic profile. Progesterone-only long-acting reversible contraception (LARC) methods — including injectables, implants, and subdermal implants — are safe alternatives. Permanent sterilisation (tubal ligation or vasectomy) may be offered to women who have completed their family, as was performed in Case 3, in keeping with MBRRACE-UK 2019 recommendations.[9,18]

Neurological Complications: PRES in Eclampsia and PPCM

PRES is a clinico-radiological syndrome characterised by headache, visual disturbance, seizures, and altered consciousness, with MRI demonstrating predominantly posterior white matter oedema.[12] It is strongly associated with hypertensive emergency, eclampsia, and immunosuppressive therapy. In Case 1, the co-occurrence of PRES with PPCM highlights the shared endothelial injury mechanism: eclampsia-driven endothelial dysfunction simultaneously predisposes to both conditions.[3,12] Management involves aggressive blood pressure control, seizure prophylaxis with anticonvulsants, and removal of the precipitating cause — goals that overlap substantially with the management of PPCM and eclampsia.

LEARNING OBJECTIVES AND ETHICAL CONSIDERATIONS

This case series illustrates several core learning objectives in the management of peripartum cardiomyopathies: (1) the critical importance of differentiating PPCM from physiological adaptations of pregnancy and other mimics; (2) the need for familiarity with pharmacological safety profiles and evidence-based therapies in the pregnant and lactating patient; (3) the value of multidisciplinary team management in complex obstetric cardiac cases; and (4) the importance of structured long- term counselling regarding prognosis, contraception, and future pregnancy planning. Ethical considerations are significant in this patient population: PPCM and DCM often present unexpectedly in young women early in their reproductive lives, with the disease profoundly altering family planning decisions. Limited data on long-term outcomes — particularly regarding subsequent pregnancy safety — complicate shared decision-making. Clinicians must provide balanced, evidence- based information while respecting patient autonomy and addressing psychosocial dimensions of care.[1,9]

Conclusion

These three cases collectively underscore the dynamic and potentially life-threatening cardiocerebral complications that may follow obstetric or abdominal emergencies in young women. Bedside echocardiography, vigilant haemodynamic monitoring, and adherence to current ESC guidelines enabled swift diagnosis and recovery in all cases.[1,9] Clinicians must maintain a high index of suspicion for reversible cardiomyopathies — particularly PPCM and TTS — and be prepared to institute guideline-directed therapy expeditiously. The complete or near-complete cardiac recovery observed in our patients, achieved through early diagnosis and targeted multidisciplinary therapy, emphasises that timely intervention is the decisive determinant of outcome in peripartum cardiac emergencies.[4,15]

APPENDIX: PHARMACOTHERAPY SAFETY PROFILE IN PERIPARTUM HEART FAILURE

Drug Class	Pregnancy	Lactation	If LV Function NOT Recovered	If LV Function Recovered
Beta-blockers	Safe (Metoprolol preferred)	Safe	Essential; titrate to max tolerated dose	Continue ≥12 months
ACE inhibitors	AVOID (teratogenic)	Relatively safe (Enalapril, Captopril)	Essential; titrate to max tolerated dose	Continue ≥12 months
ARBs	AVOID (teratogenic)	Best avoided (limited data)	Use if ACEi intolerant	Continue ≥12 months

MR Antagonists	Best avoided (no data)	Best avoided	Recommended if LVEF <40% (Eplerenone preferred)	Continue ≥6 months, then stop if sustained recovery
Diuretics	Use sparingly (↓ placental flow)	Thiazides preferred	Use only for congestion; taper early	Stop once congestion resolves
Vasodilators	Use with caution (Hydralazine, nitrates)	Safe	Symptom-based use	Stop when asymptomatic

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