

Peripartum cardiomyopathy



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Peripartum cardiomyopathy is increasingly recognised and diagnosed in clinical practice. Over the past two decades, a substantial amount of new knowledge on this condition has been accrued, including a better understanding of the pathophysiology, genetic predisposition for a proportion of patients, diagnostic tools, management with a disease-specific therapy, and predictors of outcome. Peripartum cardiomyopathy occurs globally in all ethnic groups and should be suspected in any women who are peripartum presenting with symptoms and signs indicative of heart failure towards the end of pregnancy or in the months following delivery. Verification of left ventricular systolic dysfunction (ejection fraction <45%) is crucial for the diagnosis of peripartum cardiomyopathy and the exclusion of other causes of heart failure, such as pre-existing cardiomyopathy, valvular heart disease, or congenital heart disease. Peripartum cardiomyopathy is a disease with considerable maternal and neonatal morbidity and mortality, with only half of women experiencing complete myocardial recovery within 6 months of the onset of symptoms. This Seminar summarises current knowledge of peripartum cardiomyopathy genetics, pathophysiology, diagnostic approaches, medical management, and outcome. Furthermore, we provide guidance on both risk stratification by use of a novel score to predict recovery and on the outcomes of a subsequent pregnancy.

Introduction

Peripartum cardiomyopathy is a life-threatening cardiomyopathy characterised by acute or slowly progressing left ventricular dysfunction. It can occur in women with no previous cardiac history or apparent cardiac disease, such as congenital or valvular heart disease shortly before or after delivery, or in the first postpartum months, including pregnancies ending with pregnancy loss or preterm birth.^{1,2} Peripartum cardiomyopathy is now the leading cause of maternal death in many parts of the world.^{3,4} Cardiac function recovers in more than half of affected patients within 6 months of diagnosis, with a good chance for further improvement later. However, morbidity and mortality remain high, with some patients requiring left ventricular-assist devices or cardiac transplantation.^{5,6}

Epidemiology

The incidence of peripartum cardiomyopathy differs widely between countries and ethnic groups (figure 1).⁷ The incidence is highest in countries that have a tropical climate and a wet and dry season, and ranges from 1 per 102 livebirths in Nigeria⁸ to 1 per 299 livebirths in Haiti,⁹ with other countries such as Pakistan, the Philippines, China, Sudan, and South Africa reporting about 1 case of peripartum cardiomyopathy for every 1000 livebirths.⁷ The USA reported incidences ranging between 1 per 1000 livebirths and 1 per 4000 livebirths, most frequently occurring in women with African-American ancestry.^{10,11} Populations with ancestry outside of the tropical belt (eg, Caucasian women) seem to be less affected, with countries reporting peripartum cardiomyopathy incidences of 1 per 1500 livebirths (Germany), 1 per 4717 livebirths (Scotland), 1 per 5719 livebirths (Sweden), and 1 per 10 000 livebirths (Denmark).^{12–15} The lowest incidence has been reported in Japan (1 per 20 000 livebirths).¹⁶ More subtle forms of the disease probably often remain undiagnosed and unreported;¹⁷ however, with increased awareness and

better diagnosis of the condition, the incidence of peripartum cardiomyopathy is expected to increase.¹¹

Pathogenesis

Peripartum cardiomyopathy is a distinct cardiomyopathy resulting from multiple pathological mechanisms that initiate and drive the disease,^{1,2,18} and is reflected by the variable cardiac presentation and risk factor profiles observed in patients. Risk factors and pathological mechanisms linked to peripartum cardiomyopathy are summarised in figure 2. Genetic predisposition, physiological changes during pregnancy, and other risk factors might act cumulatively to induce peripartum cardiomyopathy in otherwise healthy women. Multiple mechanisms—ie, mutation in sarcomeric and DNA repair genes, hypertension, and increased β -adrenergic activation—promote the generation of enhanced oxidative stress and subsequent cleavage of the nursing hormone prolactin into an N-terminal 16 kDa metabolite. This metabolite affects the vasculature by inducing endothelial dysfunction and killing endothelial cells. Subsequently, decreased oxygen and nutrition supply and reduced metabolism in cardiomyocytes induce peripartum cardiomyopathy. Key factors include oxidative stress, angiogenic imbalance, and inflammation, with elevated concentrations of various circulating cytokines as potential factors, inducing and driving peripartum

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Search strategy and selection criteria

We searched the Cochrane Library, MEDLINE (PubMed), and Embase from Jan 1, 2000, to Jan 1, 2025, using the search term “peripartum cardiomyopathy”, for articles published in English. We mostly selected articles published in the past 5 years but did not exclude commonly referenced and highly regarded older publications. Review articles were cited to provide readers with more details and references than this Seminar can accommodate.

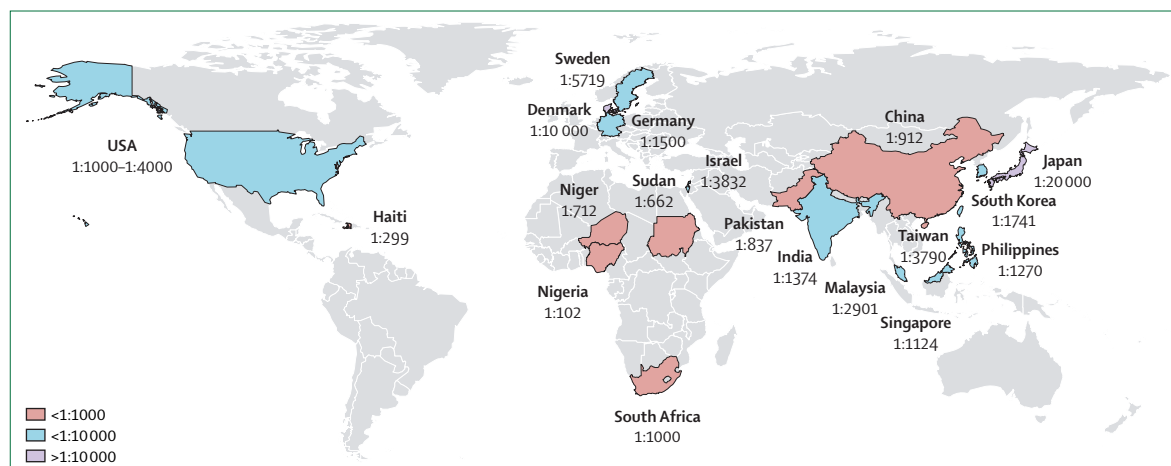


Figure 1: Global incidence

Adapted from Viljoen et al,⁷ by permission of Wolters Kluwer Health.

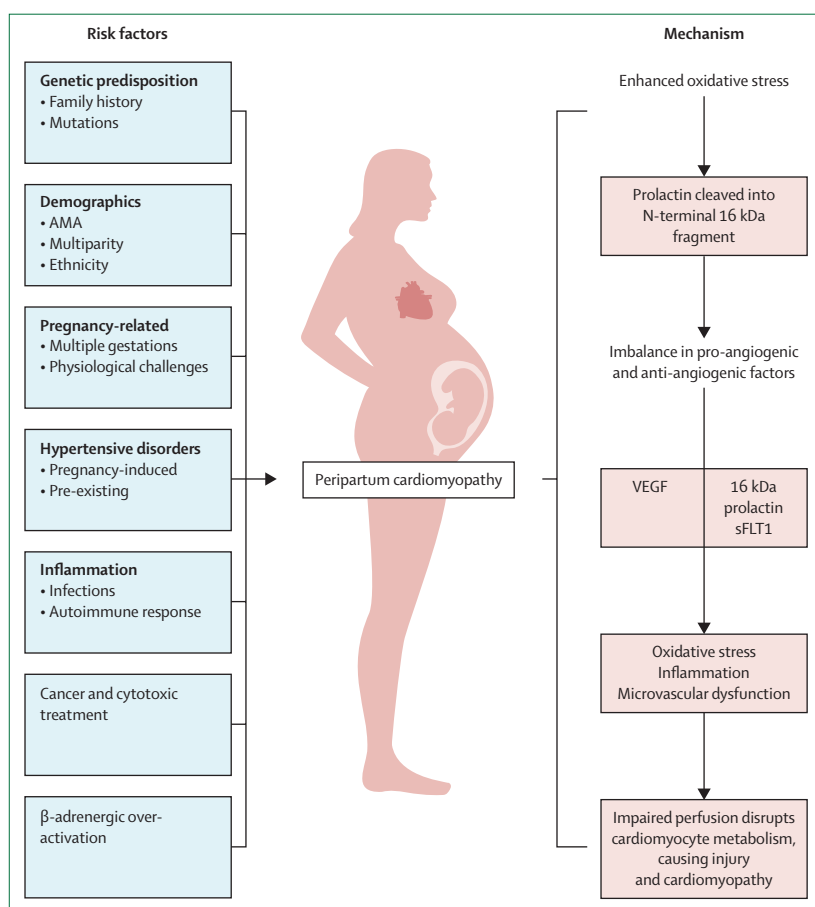


Figure 2: Multiple hit model

AMA=advanced maternal age. sFLT1=soluble fms-like tyrosine kinase-1. VEGF=vascular endothelial growth factor.

cardiomyopathy.¹⁹ As in other forms of heart failure, IL-6, TNF, C-reactive protein, and interferon-γ are elevated and correlate with the severity of cardiac failure.^{20–22} Anti-inflammatory strategies with pentoxifylline, a

xanthine-derived agent known to reduce the release of TNF and IL-6, improved the cardiac function and outcome of patients with peripartum cardiomyopathy in an African cohort.²⁰ The origin of enhanced inflammation in patients with peripartum cardiomyopathy is unclear and might be linked to autoimmune processes that are evolutionarily essential to protect pregnant women living in the tropical belt.²³

People with tropical-zone ancestry have long been known to possess genetic variations that confer resistance to malaria, a mosquito-borne protozoan parasitic infection. Such genetic variants include sickle cell disease, alleles causing α-thalassaemia and β-thalassaemia at the haemoglobin loci, and others.²⁴ Evolutionary adaptation of inflammatory immune responses in humans needs more research. However, emerging evidence suggests that pro-inflammatory alleles in several genes involved in inflammation are more prevalent in populations originating from a tropical region than in people from temperate regions.²⁵

Several pathological mechanisms in peripartum cardiomyopathy converge on a common pathway that involves unbalanced oxidative stress and the generation of the antiangiogenic 16 kDa prolactin.²⁶ Experimental and clinical observations suggest that specific activation of major signalling pathways, including the signal transducer and activator of transcription 3 (STAT 3), the peroxisome proliferator-activated receptor γ coactivator 1α (PRGC1), phosphoinositid-3-kinase (PI3K), and protein kinase B (PKB), plays a central role in protecting the maternal heart from oxidative stress, as summarised by Hilfiker-Kleiner and colleagues²⁶ and Ricke-Hoch and colleagues.²⁷ The important roles of these salient biological themes related to immune response proteins, inflammation, fibrosis, angiogenesis, and apoptosis were verified in 2023 by proteomic profiling of plasma samples from patients with peripartum cardiomyopathy in the European Society of Cardiology (ESC) EURObservational Research

Programme (EORP) and Intervention in Myocarditis and Acute Cardiomyopathy cohorts.²⁸

Dysregulation of any of these pathways is associated with increased oxidative stress, which promotes cleavage of prolactin released from the pituitary gland and elevated during pregnancy and, to a lesser extent, during lactation into the shorter N-terminal 16 kDa prolactin fragment. Myocardial endothelial dysfunction and damage are induced by the 16 kDa prolactin, leading to heart failure. Based on clinical trials by Sliwa and colleagues in a cohort from South Africa,²⁹ a larger study done in Germany,³⁰ and recent validation in a global peripartum cardiomyopathy study cohort,³¹ blocking prolactin with the dopamine D2 receptor agonist, bromocriptine, has emerged as a disease-specific therapy for peripartum cardiomyopathy, and has been added to the ESC guidelines on cardiac disease in pregnancy as a therapeutic option that should be considered.³² Bromocriptine is now used in many countries globally, but is under investigation in the USA.³¹

In approximately 15% of patients with peripartum cardiomyopathy, heterogeneous gene variations with likely pathogenic potential in one of several genes known to be associated with non-ischaemic cardiomyopathies have been identified.³³ A 2021 study³⁴ including 469 patients worldwide with peripartum cardiomyopathy showed that peripartum cardiomyopathy and dilated cardiomyopathy have a considerable genetic overlap. The study verified an approximately 10% prevalence of pathogenic truncating variants in women with peripartum cardiomyopathy, and around a 20-fold higher prevalence in these women than in control cohorts.

How these genetic mutations predispose women to peripartum cardiomyopathy is unclear. One possible explanation posits a multiple hit model, in which accumulation of risk factors (genetic and environmental) increases the likelihood of disease (figure 2). Consistent with this idea of strong genetic or environment interaction, 95% of people with truncating mutations in *TTN* show no evidence of cardiac disease—ie, the penetrance of truncating mutations in *TTN* is low—indicating that cardiac function is preserved in the absence of additional cardiac stressors.³⁵ The haemodynamic, vasculo-hormonal stressors of pregnancy or labour and ethnicity likely represent such stressors. In 2019, an increased prevalence of cancer was observed in patients with peripartum cardiomyopathy in Germany and Sweden, which was linked to cancer predisposition syndromes due to mutations in DNA damage response pathway genes.³⁶ However, whether peripartum cardiomyopathy after cancer resulted as a form of late cardiotoxicity triggered by the pregnancy, or whether DNA damage response mutations, per se, pose a risk for peripartum cardiomyopathy, is unclear. This observation, together with ethnic bias and the increasing prevalence of cardiomyopathy-associated mutations, suggests that in health-care systems offering it, genetic testing for

prognosis and to facilitate family screening can be considered in peripartum cardiomyopathy cases, especially those with familial aggregation of cardiomyopathies.

Presentation, diagnosis, and risk stratification

Women commonly present to medical professionals of various disciplines with non-specific symptoms of heart failure late in pregnancy, during delivery, or in the postpartum months. Most women present 4–8 weeks postpartum and commonly make an appointment with a general medical practitioner, with symptoms such as fatigue, swollen ankles, and mild shortness of breath, which are often attributed to being postpartum and feeling tired due to having a newborn and breastfeeding. Peripartum cardiomyopathy can also develop after a pregnancy complicated by abortion or stillbirth. The ESC EORP peripartum cardiomyopathy study showed that most women with peripartum cardiomyopathy present postpartum in Africa (75%), Europe (69%), the Middle East (66%), and the Asia-Pacific region (57%).⁵ Knowledge of peripartum cardiomyopathy and a low threshold for suspicion of heart failure are crucial for diagnosis in women with suggestive, but subtle, clinical features. Although most women with peripartum cardiomyopathy have moderate-to-severe symptoms (New York Heart Association Class III or IV), a substantial proportion have few physical symptoms, despite quite marked cardiac dysfunction.⁵ Peripartum cardiomyopathy is generally viewed as a diagnosis of exclusion, and a detailed medical history is essential to eliminate the possibility of a pulmonary embolism or other conditions, such as those listed (table), that can cause cardiomyopathy.

Hypertensive disorders complicate about 10% of pregnancies worldwide, with pre-eclampsia observed in approximately 3% of cases.^{37,38} The prevalence of hypertensive disorders in women with peripartum cardiomyopathy was substantially higher in global studies on peripartum cardiomyopathy conducted by the ESC, with more than 25% of cases developing pre-eclampsia during the index pregnancy.^{5,39} Pre-eclampsia was most common in women with peripartum cardiomyopathy from Asia, followed by those from Europe, Africa, and the Middle East. Diagnostic assessment is summarised in figure 3A, and prompt referral to a specialist is recommended in any woman who is peripartum with signs or symptoms of heart failure.

Electrocardiography is a widely available and powerful diagnostic tool for any cardiac condition. In a prospective study of women with peripartum cardiomyopathy in South Africa, more than 90% had at least one electrocardiographic irregularity, and almost half had a substantial electrocardiographic aberration (eg, extended QT interval, T-wave inversion, ST segment depression, bundle-branch block, and frequent ectopy).⁴⁰ The peripartum cardiomyopathy EORP study verified these observations.⁴¹

	History	Onset	Biomarkers	Echocardiography or CMRI	Differentiation from peripartum cardiomyopathy
Peripartum cardiomyopathy	No known cardiac disease; no heart failure signs or symptoms before pregnancy	Towards the end of pregnancy and in the months following delivery	Elevated natriuretic peptides	Reduced systolic left ventricular function; LVEF <45%	NA
Myocarditis	Previous viral infection (eg, respiratory)	Acute or subacute onset after viral infection	Elevated troponin; elevated C-reactive protein	Normal or reduced systolic left ventricular function; typical myocardial LGE pattern; pericardial effusion	CMRI (LGE pattern); myocardial biopsy
Pre-existing idiopathic or familial DCM or acquired cardiomyopathy	Heart failure signs or symptoms or known heart disease before pregnancy	During the second trimester of pregnancy	Elevated natriuretic peptides	Reduced systolic left ventricular function; right ventricular dysfunction possible; typical myocardial late enhancement pattern (DCM)	History; echocardiography; CMRI (LGE pattern)
Takotsubo syndrome	Chest pain; stressful delivery or emergency due to fetal complications	Acute onset during delivery or immediately after	Elevated natriuretic peptides	Regional wall motion irregularities with typical anatomical patterns	History; echocardiography
Pregnancy-associated myocardial infarction	Chest pain; epigastric pain	Acute onset during pregnancy or after delivery	Elevated troponin	Regional wall motion irregularities; ischaemic myocardial scar	History; echocardiography; coronary angiography; CMRI (LGE pattern)
Pulmonary embolism	Chest pain; unilateral leg swelling; acute dyspnoea	Acute onset during pregnancy or after delivery	Elevated natriuretic peptides or troponin; elevated D-dimer	Right ventricular dysfunction; right ventricular dilation; left ventricular function usually normal	CT; ventilation or perfusion scan
Amniotic fluid embolism	Chest pain during or immediately after delivery; acute dyspnoea	Acute onset during delivery or immediately after	Elevated natriuretic peptides possible	Reduced systolic right ventricular function; right ventricular dilatation	History; echocardiography
Hypertensive heart disease or severe pre-eclampsia	Pre-existing or new-onset hypertension; proteinuria	During the second trimester of pregnancy	Elevated natriuretic peptides	Left ventricular hypertrophy; diastolic dysfunction; transient left ventricular dysfunction	History; echocardiography
Hypertrophic cardiomyopathy	Familial predisposition	During the second trimester of pregnancy	Elevated natriuretic peptides	Left ventricular hypertrophy; typical myocardial LGE pattern; LVOTO (HOCM)	History; echocardiography; CMRI (LGE pattern)
HIV/AIDS cardiomyopathy	HIV infection; AIDS	During the second trimester of pregnancy	Elevated natriuretic peptides	Reduced systolic left ventricular function; left ventricle or right ventricle often not dilated	HIV serology; test
Pre-existing (unknown) congenital heart disease	Heart failure signs or symptoms before pregnancy; known heart disease; previous cardiac surgery	During the second trimester of pregnancy	Elevated natriuretic peptides	(Corrected) Congenital heart defects; cardiac shunts	History; echocardiography
Pre-existing valvular heart disease	Heart failure signs or symptoms before pregnancy; known heart disease	During the second trimester of pregnancy	Elevated natriuretic peptides	Valvular stenosis or regurgitation; prosthetic heart valves	History; echocardiography

CMRI=cardiac magnetic resonance imaging. DCM=dilative cardiomyopathy. HOCM=hypertrophic obstructive cardiomyopathy. LGE=late gadolinium enhancement. LVEF=left ventricular ejection fraction. LVOTO=left ventricular outflow tract obstruction. NA=not applicable.

Table: Differential diagnosis of peripartum cardiomyopathy.

Chest x-rays can identify alternative causes of breathlessness or hypoxia, such as infection or pleural effusion, and, while commonly showing cardiomegaly and features of pulmonary congestion, chest x-rays can be normal in peripartum cardiomyopathy. However, even if a chest x-ray is read as a possible infection, a high suspicion for fluid and heart failure should be maintained, as women with peripartum cardiomyopathy might be misdiagnosed with pneumonia.

Echocardiography is the main diagnostic modality used to verify the presence of cardiac dysfunction and to quantify the degree of severity in peripartum cardiomyopathy. Alternative causes of heart failure, such as congenital heart disease and primary valvular disease, can be excluded. Left ventricular end-diastolic and end-systolic volumes are usually pathologically increased but

can remain within normal limits.⁵ The mean left ventricular ejection fraction (LVEF) at the time of diagnosis in a global study on peripartum cardiomyopathy was around 30% in all ethnic groups.⁵ Common coexisting findings include functional mitral regurgitation and right ventricular dysfunction. Routine echocardiography should comprehensively assess the right heart, as a reduced baseline right ventricular function is an independent predictor of worse outcome.⁴²

Further echocardiography is also important for the identification of heart failure sequelae, such as intra-cardiac thrombi, which should prompt therapeutic anticoagulation.

If a full echocardiogram is not available or affordable in resource-constrained settings, a point-of-care cardiac ultrasound identifying reduced LVEF should be sufficient

to make the correct diagnosis, allowing appropriate therapy to be started.

Cardiac MRI might be helpful in patients with suspected peripartum cardiomyopathy. In addition to detailed identification of right ventricular and left ventricular function and dimensions, myocardial structure can be assessed. Although no pathognomonic cardiac MRI pattern exists for the diagnosis of peripartum cardiomyopathy, cardiac MRI can be useful to differentiate it from other conditions, such as peripartum myocardial infarction, myocarditis, or infiltrative diseases. Unfortunately, access to cardiac MRI is often hindered by cost and geographical location.

Regional wall motion disturbances are found on cardiac MRI in many patients with peripartum cardiomyopathy. Myocardial oedema is present in a third of patients; a focal non-ischaemic intramyocardial or subepicardial late gadolinium enhancement, preferentially anteroapical in the basal to mid-ventricular wall, is much more common. Non-specific pericardial effusion can be observed, and occasionally, cardiac MRI detects left ventricular thrombi. Left ventricular dilatation often occurs with (mostly reversible) hypertrabeculation, which can also be present in patients with dilated cardiomyopathy. Diagnosis of (rare) acute myocarditis requires evidence of a myocardial oedema and typical non-ischaemic late gadolinium enhancement or pathological T1-mapping.

Routine haematological and biochemical testing will provide information on potentially reversible contributory factors such as anaemia and renal impairment. Natriuretic peptide concentrations (when available) should be measured in women with suspected peripartum cardiomyopathy.² A normal natriuretic peptide concentration is uncommon in patients newly diagnosed with peripartum cardiomyopathy and presenting with heart failure. Genetic testing could also be considered, especially in cases of familial aggregation of cardiomyopathies, as part of the tests. The identification of disease-causing genetic mutations will be of clinical relevance to the patient and her family, including her children, who would be at risk in the case of genetic cardiomyopathy.¹⁷

Several factors are important for risk stratification of women with peripartum cardiomyopathy. Specifically, sociodemographic factors associated with maternal and neonatal outcomes were investigated in women enrolled in the ESC peripartum cardiomyopathy global registry.⁴³ Both country-level factors (Gini index coefficient, health expenditure, and human development index [HDI]) and individual-level factors were described in the study. Low HDI was associated with increased left ventricular dilatation at time of diagnosis. The analysis showed that the baseline LVEF did not differ according to sociodemographic factors. Countries with low health expenditure prescribed guideline-directed heart failure therapy less frequently than those with high health

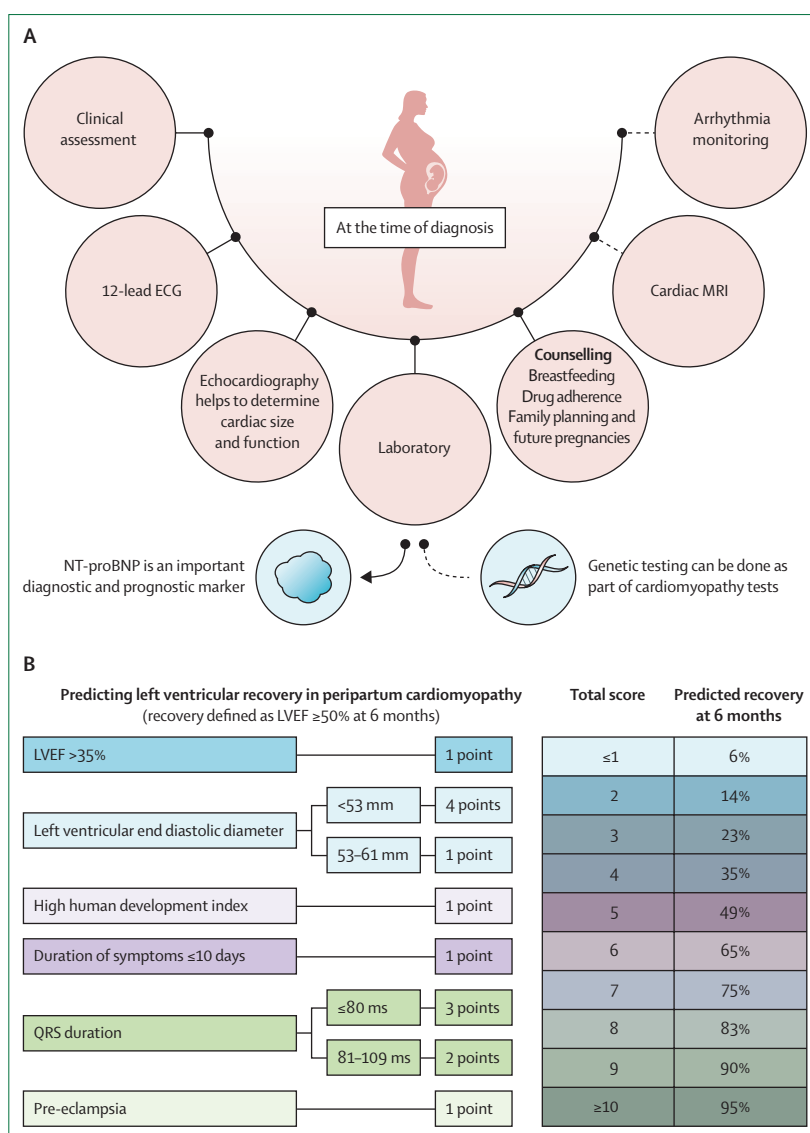


Figure 3: Testing and scoring

(A) Diagnosis testing for peripartum cardiomyopathy. Adapted from Sliwa et al.⁶⁰ (B) The Peripartum Cardiomyopathy Recovery Score. Adapted from Jackson et al.⁴⁴ ECG=electrocardiography. LVEF=left ventricular ejection fraction.

expenditure. Countries with low health expenditure had an increased 6-month mortality. Left ventricular recovery was reduced in countries with a low HDI, low health expenditure, and low levels of educational attainment. Maternal outcome (death, re-hospitalisation, or persistent left ventricular dysfunction) was independently associated with household income. Neonatal death was considerably more common in countries with low health expenditure and a low HDI, but was not influenced by maternal income or education attainment. Attempts should therefore be made to allocate adequate resources to health and education, so that maternal and fetal outcomes in peripartum cardiomyopathy can be improved.

In another peripartum cardiomyopathy registry analysis,⁴⁴ a prediction model with good discriminative ability (C statistic of 0.79) was derived by stepwise regression, and included the final variables of baseline LVEF, baseline left ventricular end diastolic diameter, HDI, duration of symptoms, QRS duration, and pre-eclampsia. From this model, the Peripartum Cardiomyopathy Recovery Score was generated (figure 3B) to allow calculation of the probability of left ventricular recovery at 6 months for an individual. Women with an LVEF less than 35%, a left ventricular end diastolic dimension of 62 mm or more, from a country with a low HDI, showing symptoms for more than 10 days before diagnosis, a QRS duration of 110 ms or more, and without pre-eclampsia had the lowest probability of recovery. This score includes readily available data and is therefore an accessible clinical tool. Better prediction of reduced probability of recovery can support decisions regarding

early referral to advanced heart failure services and more aggressive optimisation of guideline-directed medical therapy (GDMT). Therefore, risk stratification should be done immediately to identify the appropriate level of care. Peripartum cardiomyopathy is associated with substantial morbidity and mortality. Most life-threatening complications, including severe heart failure, cardiogenic shock, arrhythmias, thromboembolic complications, and death, can be prevented by early referral to a centre with expertise in managing acute heart failure and by appropriate use of medical and non-pharmacological therapy.

Management

The management of peripartum cardiomyopathy differs according to the clinical scenario of presentation—in particular, whether the patient is still pregnant or is post-delivery and whether the patient is stable or has acute decompensated heart failure. A dedicated pregnancy heart team for patients with acute heart failure should be created in all larger centres to ensure rapid and appropriate decision-making processes regarding the best supportive care for mother and child, including (urgent) delivery, inhibition of lactation, and potential mechanical circulatory support for patients in cardiogenic shock.⁴⁵

We have summarised the acute and chronic therapeutic options for the treatment of peripartum cardiomyopathy (figure 4). For the management of heart failure presenting during pregnancy, some components of GDMT are applicable, including β blockers, diuretic agents, vasodilators, and digoxin. Angiotensin-converting enzyme (ACE) inhibitors, angiotensin II antagonists, and angiotensin receptor neprilysin inhibitors (ARNIs) should be avoided during pregnancy due to the potential for malformation and, in later pregnancy, because of negative effects on the fetal renal system, resulting in renal failure and oligohydramnios.⁴⁶ β blockers are considered safe, although a variable degree of growth restriction depending on the agent used is to be expected. Loop diuretics, such as furosemide and bumetanide, are safe⁴⁷ but mineralocorticoid receptor antagonists, specifically spironolactone, are avoided due to the theoretical feminisation of the male fetus, although this theory has never been reported in humans; it was only observed after large doses were administered in animal studies (rabbit and rat).⁴⁸ In terms of vasodilators, hydralazine is safe and commonly used in the management of hypertension during pregnancy; however, the fetal heart should be closely monitored during intravenous administration.³² Nitrates have also been used during pregnancy for a range of indications, including induction of labour and management of growth restriction, and are safe but can cause nausea, vomiting, and headaches.⁴⁹ In terms of arrhythmias, if they are acutely, haemodynamically unstable, then direct-current cardioversion is advisable to rapidly obtain a

BOARD scheme		
Bromocriptine (dose according to severity of the disease)	Oral heart failure drugs (β blockers, ACE inhibitor or ARB, MRA)	Anticoagulation (at least in prophylactic dose)
		Relaxants (intravenous vasodilators if SBP >110 mm Hg)
		Diuretics (in case of fluid overload)
Drug	Heart failure and absence of complete left ventricular recovery	Complete and sustained recovery (LVEF >55% and NYHA functional class I)
β blocker	Essential for all patients in standard or maximally tolerated dosages	Continue all drugs for at least 6–12 months or lifelong after full recovery; individual approach or discuss with patient. Discontinue stepwise and monitor symptoms and left ventricular function: 1) MRA 2) SGLT2 inhibitor 3) ARNI, ACE inhibitor, or ARB 4) β blocker
ARNI	Essential for all patients in standard or maximally tolerated dosages	
ACE inhibitor	Recommended for all patients in standard or maximally tolerated dosages when ARNI is not available or contraindicated	
ARB	Recommended in patients who do not tolerate ACE inhibitor	
MRA	Recommended for all patients in standard or maximally tolerated dosages; preferably eplerenone due to less hormonal side-effects and less blood pressure reduction compared with spironolactone	
SGLT2 inhibitor	Essential for all patients in standard dosage (dapagliflozin 10 mg or empagliflozin 10 mg once daily)	Discontinue if heart rate <50 bpm or in case of complete recovery
Ivabradine	Recommended in patients in sinus rhythm with a persisting heart rate >70 bpm at rest despite maximal tolerated β blocker dose	
Diuretics	Recommended in patients with fluid overload	
		Taper dose or discontinue if no signs of fluid overload; maintain only if part of antihypertensive therapy

Figure 4: Drug treatment for patients with peripartum cardiomyopathy during the acute phase (upper panel) and during the subacute or chronic phase (lower panel)

ACE=angiotensin-converting enzyme. ARB=angiotensin II receptor blockers. ARNI=angiotensin receptor neprilysin inhibitors. bpm=beats per minute. LVEF=left ventricular ejection fraction. MRA=mineralocorticoid receptor antagonist. NYHA=New York Heart Association. SBP=systolic blood pressure. SGLT2=sodium-glucose co-transporter-2.

regular cardiac output and improve uterine perfusion. For atrial fibrillation, β blockers and digoxin are safe. For ventricular tachycardia in the acute setting, lidocaine, procainamide, and quinidine have no adverse fetal effects and β blockers are safe for longer-term management. Other agents such as flecainide, which is used to manage fetal arrhythmia, or mexiletine, appear safe but amiodarone should be avoided due to fetal hypothyroidism and growth restriction. Further advice can be found in the relevant guidelines.⁵⁰

Most patients with acute heart failure respond well to intravenous diuretic therapy. However, patients with cardiogenic shock should be stabilised as quickly as possible and transferred to a centre capable of, and experienced in, advanced acute heart failure therapies, including transient and continuous mechanical circulatory support (eg, microaxial pumps, extracorporeal membrane oxygenation, and ventricular-assist devices) and, potentially, heart transplantation.^{2,45}

Treatment with catecholamines such as dobutamine, epinephrine, and norepinephrine at high dosage, or for extended periods, appears to be associated with reduced left ventricular recovery in patients with peripartum cardiomyopathy with cardiogenic shock; early consideration of mechanical circulatory support might be preferable.^{45,51,52} Whether the calcium-sensitiser levosimendan—which is not approved in the USA—plays a role in the treatment of cardiogenic shock in peripartum cardiomyopathy is uncertain. A small study indicated that levosimendan can be used in combination with bromocriptine.⁵²

In the subgroup of patients with peripartum cardiomyopathy presenting in the third trimester of pregnancy, specific aspects should be taken into account. Once stabilised, depending on the gestational age, delivery may be considered. Early delivery appears to be indicated in cases of severe cardiomyopathy, major challenges in volume medication management, and other indicators of severe disease. Optimally, in terms of fetal maturity, delivery should be after 32 weeks, with antenatal steroid administration to mature the fetal lungs and magnesium infusion to reduce the risk of cerebral palsy, depending on the gestational age. Although caesarean section is an option in the case of decompensated heart failure that cannot be stabilised,³² induction and vaginal delivery should be considered, given the associated reduced risks of infection, haemorrhage, or thrombosis. Patient preference should also be taken into consideration. Whether delivery is by caesarean section or vaginally, particular care to maintain haemodynamic stability and avoid fluid overload is essential.

After delivery, patients with peripartum cardiomyopathy should be treated according to the guidelines for heart failure^{50,53} with all available GDMTs, including β blockers, renin-angiotensin system inhibitors, preferably ARNIs, mineralocorticoid receptor antagonists, and SGLT2 inhibitors (figure 4). Ivabradine and digoxin might also

be helpful in selected patients with sinus tachycardia or atrial fibrillation with a rapid ventricular rate. Clearly, contraindications for the application of medications in patients who are lactating need to be respected.^{2,32}

Patients with acute peripartum cardiomyopathy also have an elevated risk of sudden cardiac death.^{54,55} However, after surviving the acute phase of decompensation and cardiogenic shock, the probability for recovery, or at least improvement of LVEF, is high in patients with peripartum cardiomyopathy; thus, the decision to insert an implantable cardioverter-defibrillator or cardiac resynchronisation therapy device should not be made before 6–12 months of optimised GDMT.^{54,56} To reduce the potential risk of life-threatening ventricular arrhythmias in patients with peripartum cardiomyopathy with severely reduced LVEF (<35%), a wearable cardioverter-defibrillator can be considered in the first months during optimisation of GDMT.^{2,55} In a 2024 study, such an approach improved the LVEF of patients with other causes of newly diagnosed heart failure—insertion of an implantable cardioverter-defibrillator was avoided.⁵⁷

Management post-pregnancy and neonatal outcome

For women who are peripartum and presenting with heart failure, breastfeeding is controversial. The 2025 ESC guidelines for the management of cardiovascular diseases during pregnancy³² suggest that prevention of lactation should be considered in patients with severe heart failure to avoid the high metabolic demands of breastfeeding and to enable safe treatment with established heart failure drugs.

Standard heart failure drugs should be used with prudence during lactation. If a shared decision is made to continue breastfeeding (eg, in patients with mild-to-moderate heart failure), cautious use of β blockers (metoprolol), ACE inhibitors (enalapril, lisinopril, benazepril, or captopril), mineralocorticoid receptor antagonists (spironolactone), and sacubitril plus valsartan might be possible according to the US National Institutes of Health Drugs and Lactation Database, although data regarding the safety of the infant are scarce. SGLT2 inhibitors and eplerenone should be avoided.

The dopamine D2 receptor agonist, bromocriptine, reduces the concentrations of prolactin and its devastating derivatives in peripartum cardiomyopathy, and has emerged as a disease-specific therapy.^{29,30} It is prescribed in many countries globally,⁵⁸ but is under investigation in the USA in the REBIRTH trial (NCT05180773). Data from the EORP global peripartum cardiomyopathy registry³¹ indicated that bromocriptine treatment in addition to GDMT is associated with better left ventricular recovery in patients with acute peripartum cardiomyopathy. Bromocriptine is an affordable, safe medication that stops lactation, enabling rapid uptitration of GDMT. Bromocriptine, according to the ESC guidelines on cardiac disease in pregnancy,³² can be

considered as a therapeutic option (figure 4). The suggested dosing of bromocriptine is 2·5 mg once daily for 1 week in uncomplicated cases; long-term treatment (2·5 mg twice daily for 2 weeks and then 2·5 mg once daily for another 6 weeks) can be applied in patients with severe acute heart failure and cardiogenic shock². If bromocriptine is not available, cabergoline can be used (0·5 mg per week or 1 mg per week).⁵⁹

The peripartum cardiomyopathy EORP study did not show an increase of thromboembolic events in patients given bromocriptine.³¹ However, around the time of peripartum cardiomyopathy diagnosis (which is mostly around delivery or shortly thereafter) the rate of peripheral arterial and venous thromboembolic events is rather high (7% in the first 30 days after delivery in the peripartum cardiomyopathy worldwide registry, independent of bromocriptine treatment).^{5,31} Therefore, a high index of suspicion should be maintained and an appropriate diagnostic procedure done if venous thromboembolism is suspected. Prophylactic anticoagulation should be considered in all patients with peripartum cardiomyopathy, and therapeutic anticoagulation is essential in patients with intracardiac thrombus, evidence of systemic thromboembolism, or paroxysmal or persistent atrial fibrillation.

Data on neonatal outcomes are rarely reported. The ESC EORP cohort study reported neonatal death in 5% of the 739 patients included in the global study, with a marked regional variation (2% in Europe, 5% in Africa, 4% in the Asia-Pacific region, and 9% in the Middle East, $p=0\cdot012$).⁵

Outcome, subsequent pregnancy, and supporting women with peripartum cardiomyopathy

Outcomes in patients with cardiovascular disease are usually defined by re-hospitalisation and survival of 30 days, 6 months, or 12 months. However, in a young cohort of patients, as is the case in peripartum cardiomyopathy, long-term outcomes are also important (eg, >10 years) but have not been studied. The lack of information causes anxiety for patients and their families,⁶⁰ and many patients meet the criteria for mental disorders such as depression or post-traumatic stress disorders.⁶¹

Maternal mortality, no matter when and where it occurs, results in sequelae that extend beyond the loss of the life of a single woman. The death of a mother adversely affects the ability of her family to survive and thrive, especially if they are socioeconomically deprived. As death due to peripartum cardiomyopathy commonly occurs after 42 days postpartum, it is often not documented as a maternal death since few countries document late maternal death.^{62,63}

The mortality rates of patients with peripartum cardiomyopathy vary widely depending on geographical regions. The EORP global registry on peripartum

cardiomyopathy⁵ investigated the clinical presentation, management, and outcome of patients with peripartum cardiomyopathy. A total of 743 patients from more than 40 countries in Europe, Africa, the Asia-Pacific region, and the Middle East were enrolled over a 6-year period. Initial results showed rather low mortality rates 1 month after the diagnosis (2·4% overall, 3·4% in ESC countries, and 1·4% in non-ESC countries). However, the 6-month mortality was 6%, with 42% due to heart failure and 30% due to sudden death.⁵ Mortality rates in other studies were substantially higher: 15–30% in Turkey,⁶⁴ 18% of which were within 17 months, and a 6-month mortality of 8% (median death rates) in the Nigerian PEACE study,⁶⁵ which included 244 patients.

Recovery of left ventricular function can occur after 6 months of the onset of disease and even beyond 12 months.³³ A multicentre study by the Investigation of Pregnancy-Associated Cardiomyopathy group analysed 100 women with peripartum cardiomyopathy from North America.⁶⁶ Left ventricular recovery was observed in 72% of women after 12 months. Few prospective data report on the long-term follow-up of patients with peripartum cardiomyopathy beyond 5 years after initial diagnosis. A study done in Germany prospectively investigated the outcome of 67 patients, with a mean follow-up of 63 months (SD 11).⁵⁶ Left ventricular function improved from an ejection fraction of 26% at initial diagnosis to 50% after 12 months and 54% after 5 years. The number of patients who fully recovered was 48% after 6 months, further rising to 60% after 1 year and then to 72% after 5 years. Women with African ethnicity were widely believed to always have worse outcomes compared with women of other ethnicities;⁴⁵ however, data from the global ESC EORP studies^{5,43} showed a worse outcome in women from the Middle East than in those from Africa. These data also showed the profound impact of patient education and access to health care.

Counselling of patients who are considering a subsequent pregnancy is of utmost importance.^{30,67} The updated 2025 ESC guidelines on cardiac disease in pregnancy highlight that women with a previous peripartum cardiomyopathy diagnosis face a notably elevated risk of poor outcomes.³² A 2022 prospective observational study from two centres in Israel⁶⁸ reported that, of 59 full-term pregnancies (45 women), relapse was observed in 25% of women with a before subsequent pregnancy LVEF of less than 55% and only in one (3%) with a before subsequent pregnancy left ventricular recovery (LVEF $\geq 55\%$), which was not associated with mortality. Subsequent pregnancies, even in women with recovered LVEF, were associated with a small but not clinically significant reduction in LVEF, which did not drop to less than 50%. A 2023 retrospective study in the USA⁶⁷ provided insights into the risks associated with subsequent pregnancies in a different patient population, in which most were of African-American descent (80%) and from a low

socioeconomic background (75·6%). Upon comparing the outcomes in a non-recovered group (ejection fraction <50%) and a recovered group (ejection fraction >50%), high rates of adverse outcomes (53·3% vs 20%) and 5-year all-cause mortality (13·3% vs 3·3%) were reported. Although the study provides long-term follow-up, it is limited by the small patient numbers and the fact that the data were accrued over almost four decades, with considerable variability in management over that time.

A substudy of the peripartum cardiomyopathy EORP global study on 332 patients with peripartum cardiomyopathy undergoing 98 pregnancies provided new prospective data.⁶⁹ Maternal mortality was lower than anticipated (2%), and a baseline LVEF of less than 50% was not associated with an increased frequency of adverse maternal outcomes or a decline in LVEF. However, in the subsequent pregnancy cohort with an LVEF less than 50%, most patients had an LVEF of more than 40%, and only a few patients proceeded with a subsequent pregnancy with an LVEF less than 40%. In contrast to previous data, women with subsequent pregnancies and a baseline LVEF of 50% or more had a decline in LVEF, potentially attributable to reduced use of GDMT during pregnancy and in the postpartum period. Therapeutic termination was performed in approximately a fifth of cases but did not affect postpartum outcome. The data might also indicate that patients with peripartum cardiomyopathy with an LVEF between 40% and 50% can proceed with a subsequent pregnancy, emphasising the importance of careful monitoring by an experienced multidisciplinary team. The 2025 ESC guidelines also reflect this data, stating that more than mild left ventricular dysfunction (ie, <40% LVEF) before a new pregnancy increases the risk of LVEF deterioration.³²

Over the past decade the focus in medicine has shifted towards a more patient-centred approach and the tackling of complex medical conditions with a multidisciplinary team. A publication by the ESC Heart Failure Association Committee on peripartum cardiomyopathy summarises the overall support women with peripartum cardiomyopathy and their families should receive (figure 5) from health-care providers.⁶⁰

Conclusion and future research

Peripartum cardiomyopathy is a global disease in which the diagnosis is often delayed, leading to considerable morbidity and mortality. It is a substantial contributor to early (<42 days) and late (up to 1 year) postpartum maternal death. In the past two decades, remarkable advances in understanding the pathogenesis of the condition and patient management have been reached, including a disease-specific therapeutic option, bromocriptine, which should be considered in all cases of peripartum cardiomyopathy, in addition to GDMT for heart failure. In the USA, bromocriptine is not prescribed for peripartum cardiomyopathy—the ongoing REBIRTH

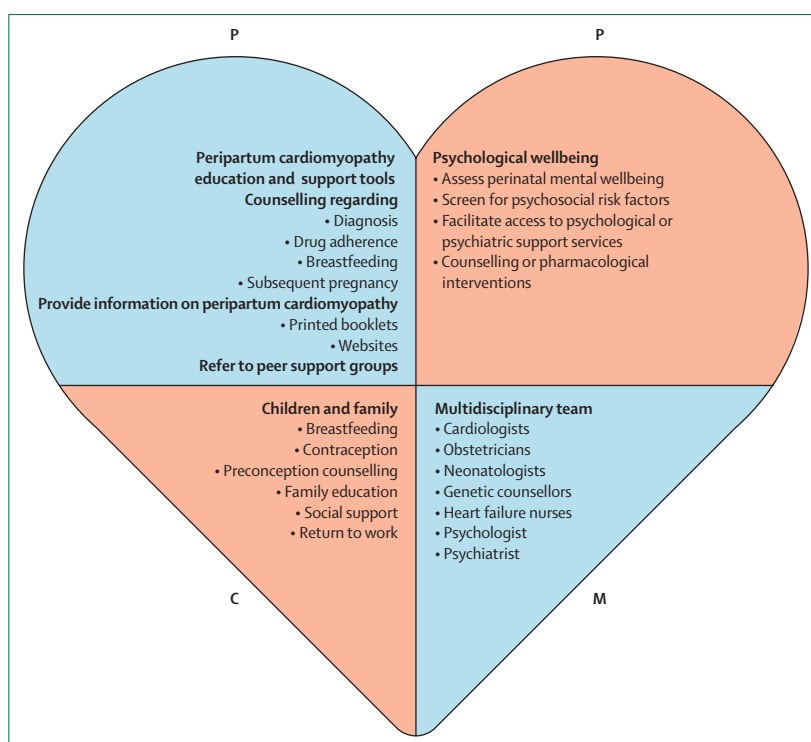


Figure 5: Supporting women to live with peripartum cardiomyopathy
Adapted from Sliwa et al.⁶⁰ PPCM=peripartum cardiomyopathy.

research study will provide randomised data regarding its value. Studies such as the P-CARE MRI in peripartum cardiomyopathy study and the observational or peer-to-peer network peripartum cardiomyopathy-R study might further expand the understanding of peripartum cardiomyopathy care, specifically for US patients. How to downtitrate or even stop heart failure GDMT in patients with peripartum cardiomyopathy will be investigated in a dedicated future study. Referral for genetic testing should be discussed, especially in patients with a family history of a cardiomyopathy or sudden death. With increasing awareness and better diagnostic tools, the disease has moved from rare to a frequent pregnancy complication, thereby raising research interest in this field. Despite ongoing research, numerous uncertainties persist regarding the incidence, pathophysiology, differences in mode and presentation, treatment, and prognosis of patients with peripartum cardiomyopathy, indicating the need for further investigation. Most importantly, global awareness of peripartum cardiomyopathy should be increased to improve detection rates and outcomes.

Contributors

KS and JB conceptualised and drafted the original manuscript. All other authors contributed to specific sections of the manuscript.

Declaration of interests

We declare no competing interests.

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For more on the **P-CARE MRI in peripartum cardiomyopathy study** see <https://pittplusme.org>

For more on the **peripartum cardiomyopathy-R study** see <https://www.ppcmr.org/>

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