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Multidisciplinary Approach to Cardiomyopathy in Pregnancy : From Diagnosis to Delivery Planning

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Abstract

Cardiomyopathy in pregnancy, particularly peripartum cardiomyopathy (PPCM), is a rare but potentially life-threatening condition that poses serious risks to both maternal and fetal health. Its clinical manifestations, such as dyspnea, fatigue, and edema often resemble normal pregnancy symptoms, contributing to delayed diagnosis and treatment. This narrative review aims to summarize the etiology, diagnostic approach, and multidisciplinary management strategies for cardiomyopathy during pregnancy, focusing on PPCM, dilated cardiomyopathy (DCM), and other less common subtypes. Literature was obtained through a non-systematic search of PubMed, ScienceDirect, and Google Scholar for studies published between 2010 and 2025. Diagnosis is based on clinical evaluation, echocardiography, NT-proBNP, troponins, and exclusion of other causes. Management includes symptom control, prevention of arrhythmias and thromboembolism, and careful adaptation of standard heart failure therapy to the physiological changes of pregnancy. Medications such as beta-blockers, diuretics, and low molecular weight heparin are considered safe, while ACE inhibitors are contraindicated during gestation. The use of bromocriptine shows potential benefit in improving cardiac function but remains controversial due to the risk of thrombosis, requiring concurrent anticoagulation. Device therapy and mechanical support may be indicated in severe or refractory cases. Prognosis depends on cardiomyopathy subtype, timing of diagnosis, and left ventricular function recovery. Early recognition, close monitoring, and individualized care are essential to improve maternal and fetal outcomes. Further research is needed to guide safe and effective management strategies in pregnant patients with cardiomyopathy.

Keywords: Cardiomyopathy, Peripartum Cardiomyopathy, Pregnancy, Heart Failure, Maternal Health, PPCM

1. Introduction

Cardiomyopathy during pregnancy is a rare but serious condition that affects maternal heart health, particularly in the form of peripartum cardiomyopathy (PPCM), which typically occurs toward the end of pregnancy or in the months following delivery. PPCM can lead to heart failure, posing significant risks to both mother and baby, although the exact cause remains incompletely understood. Epidemiologically, the prevalence of cardiomyopathies such as hypertrophic cardiomyopathy (HCM) and dilated cardiomyopathy (DCM) is estimated to be 0.2%–0.4%, while PPCM occurs in approximately 1 in 1,000 to 4,000 live births. Risk factors include maternal age over 30 years, hypertension, and multiple pregnancies.[1]

Cardiomyopathy during pregnancy presents unique challenges in both diagnosis and management. Its symptoms often resemble those of normal pregnancy, such as fatigue, leg swelling, and shortness of breath, which may delay recognition and diagnosis. Therefore, a thorough understanding of the various types of cardiomyopathy and associated risk factors is essential for timely and accurate diagnosis. Key risk factors include gestational hypertension, maternal age above 30, a history of multiple pregnancies, and the use of certain medications.[2]

The diagnosis of PPCM is based on three main criteria: the presence of heart

failure toward the end of pregnancy or within five months postpartum, left ventricular systolic dysfunction with a left ventricular ejection fraction (LVEF) below 45%, and the absence of other identifiable causes of heart failure. Supporting investigations include echocardiography, electrocardiography (ECG), cardiac biomarkers such as NT-proBNP and troponins, and, when necessary, cardiac MRI or catheterization. Other diagnostic tools, such as BNP testing and endomyocardial biopsy, are not routinely performed due to the lack of specific findings in PPCM.[3,4]

The management of PPCM is similar to that of general heart failure, with special consideration for maternal and fetal safety. Acute treatment focuses on reducing preload and afterload and enhancing myocardial contractility. Chronic management follows the European Society of Cardiology (ESC) and American College of Cardiology/American Heart Association (ACC/AHA) guidelines, incorporating antiarrhythmic therapy, anticoagulation, mechanical circulatory support, and even transplantation when necessary. The use of bromocriptine to promote recovery of ejection fraction remains controversial due to thromboembolic risk and must be carefully weighed.[2]

Cardiomyopathy in pregnancy can have long-term consequences for maternal health, especially if not promptly and adequately managed. Deterioration of cardiac function may result in severe complications, including congestive heart failure, arrhythmias, or even death. Therefore, it is crucial for healthcare professionals to closely monitor pregnant women with high-risk profiles and initiate timely evaluation and intervention.[2]

This narrative review aims to provide a comprehensive overview of cardiomyopathy in pregnancy, encompassing its definition, etiology, epidemiology, pathophysiology, and the classification of its various forms. The objective of this paper is to deepen the understanding of the underlying factors that contribute to the development of cardiomyopathy during pregnancy and to explore its clinical implications for both maternal and fetal health.

2. Methods

This article is a narrative review aimed at summarizing current knowledge on cardiomyopathy in pregnancy. Literature was collected through a non-systematic search of PubMed, ScienceDirect, and Google Scholar databases for articles published between 2010 and 2025. Keywords included “cardiomyopathy in pregnancy,” “peripartum cardiomyopathy,” “dilated cardiomyopathy,” and “heart failure in pregnancy.” Inclusion criteria were original research articles, clinical trials, and relevant reviews discussing diagnosis, management, and outcomes of cardiomyopathy during pregnancy. Articles not available in English or with limited access to full text were excluded. Due to the narrative nature of this review, no formal quality appraisal was conducted.

3. Cardiomyopathy

Definition

Cardiomyopathy is defined as a myocardial disorder in which the heart muscle is structurally and functionally abnormal, in the absence of coronary artery disease, hypertension, valvular disease, or congenital heart conditions sufficient to explain the observed dysfunction. It may be acquired or inherited and includes various types such as hypertrophic cardiomyopathy (HCM), arrhythmogenic right ventricular cardiomyopathy (ARVC), left ventricular non-compaction, restrictive

cardiomyopathy (RCM), and dilated cardiomyopathy (DCM). Peripartum cardiomyopathy (PPCM), the most common form during pregnancy, is often categorized under DCM. Although cardiomyopathy in pregnancy is rare and not well-documented, increasing case reports and small studies have emerged over the past decade.[1]

Etiology

The etiology of cardiomyopathy in pregnancy varies according to its subtype. Hypertrophic cardiomyopathy (HCM) and arrhythmogenic right ventricular cardiomyopathy (ARVC) are primarily genetic in origin, while dilated cardiomyopathy (DCM) and restrictive cardiomyopathy (RCM) often arise from a combination of genetic, idiopathic, infectious, or toxic factors. Peripartum cardiomyopathy (PPCM), a distinct subtype occurring in late pregnancy or postpartum, has a multifactorial etiology involving inflammation, autoimmunity, and vascular dysfunction. Understanding these etiological differences is essential for appropriate diagnosis and management during pregnancy.[1]

Epidemiology

Cardiomyopathy during pregnancy is relatively uncommon but carries significant maternal risk. Hypertrophic and dilated cardiomyopathies are each estimated to affect 0.2%–0.4% of the general adult population, while ARVC and RCM are much rarer. In a U.S. cohort, peripartum cardiomyopathy (PPCM) was the most frequently observed subtype among pregnant women with heart failure. Data from international registries also indicate that cardiomyopathy in pregnancy is associated with higher rates of arrhythmia, heart failure, and maternal mortality compared to other cardiac conditions.[1–3]

Pathophysiology

Normal pregnancy induces a 30%–50% increase in cardiac output through elevated stroke volume in the first two trimesters and increased heart rate (by 10–15 bpm) in the second trimester. Plasma volume rises more than red blood cell mass, causing physiological anemia. Systemic vascular resistance drops late in the second trimester but rises again near term. The heart undergoes concentric and/or mild eccentric remodeling and hypertrophy.[1]

During labor, cardiac output increases progressively and may peak at 80% above baseline. Blood loss can reach 500–1000 mL but is partially compensated by autotransfusion from uterine contractions and relief of vena cava compression. Hemodynamic changes normalize within six months postpartum. Pregnancy and the postpartum period remain hypercoagulable, adding cardiovascular strain.[1–3]

Classification

a. Dilated cardiomyopathy

DCM may be diagnosed before pregnancy or become symptomatic during gestation due to altered hemodynamics. Symptoms such as dyspnea, edema, and fatigue mimic normal pregnancy, often delaying diagnosis. BNP/NT-proBNP levels and echocardiography are helpful, though gadolinium-enhanced MRI should be avoided in pregnancy. Cardiac events are more common in women with higher NYHA class or moderate-to-severe left ventricular dysfunction.[5,6]

Studies by Grewal et al. show poor outcomes in DCM pregnancies: one by Grewal et al. found that only 28% of pregnant DCM patients were event-free at 16 months vs. 83% in non-pregnant DCM women.[5] Another study by Guo et al. with 35 pregnancies found 8 cases with peripartum complications; risk factors

included pre-pregnancy use of heart failure medication, elevated BNP, and diastolic dysfunction. While some women tolerated pregnancy with LVEF >30%, the risk of preterm delivery and peripartum events remained. Tachycardia-induced DCM may improve with treatment such as radiofrequency ablation. History of cardiac events strongly predicts future complications, but those with good NYHA class and minimal LV involvement may have favorable outcomes.[1]

b. Peripartum cardiomyopathy

Peripartum cardiomyopathy (PPCM) is a rare but potentially life-threatening form of heart failure caused by left ventricular systolic dysfunction (ejection fraction <45%) that occurs in the last month of pregnancy or within five months postpartum, without an identifiable cause. It typically presents with symptoms like dyspnea, orthopnea, edema, and palpitations, which may be mistaken for

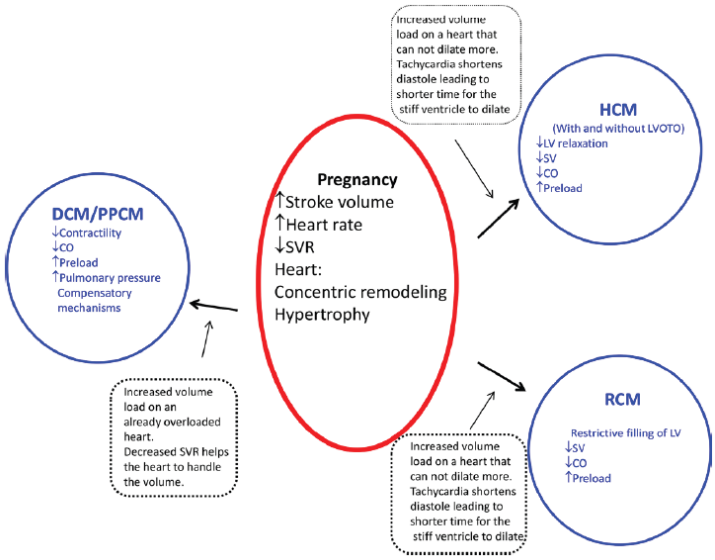


Figure 1. General description of hemodynamic changes during pregnancy and their effect on different types of cardiomyopathy[1]

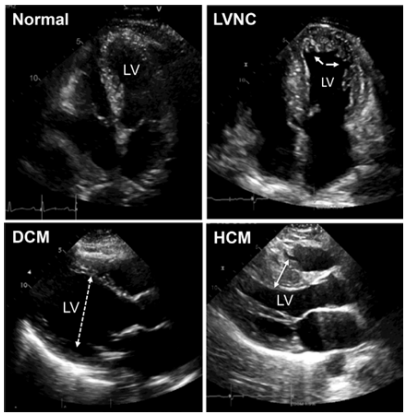


Figure 2. Echocardiography showing forms of cardiomyopathy. Left ventricular noncompaction cardiomyopathy (LVNC) is shown in the upper right (arrow indicates deep trabeculation in the left ventricle [LV]). Dilated cardiomyopathy (DCM) is defined by an enlarged LV diameter (dashed double arrow). Hypertrophic cardiomyopathy (HCM) is defined by thickening of the LV, including the septum (marked with double arrows)[5]

normal pregnancy changes, leading to delayed diagnosis. Risk factors include age over 25, African descent, multiparity, preeclampsia, multiple gestation, chronic hypertension, obesity, and prolonged tocolytic therapy. PPCM incidence varies globally, from 1 in 100 to 1 in 120,000 with the highest rates in Africa, possibly related to genetic and cultural factors. Its pathophysiology is multifactorial, involving inflammation, autoimmune mechanisms, genetic susceptibility (e.g., GNB3 polymorphism), prolactin dysregulation (16 kDa fragment), and angiogenic imbalance. Preeclampsia is both a major risk factor and a potential co-condition, associated with worse outcomes such as cardiogenic shock and prolonged hospitalization. Diagnosis relies on excluding other causes and confirming reduced ejection fraction via echocardiography, supported by biomarkers and imaging when needed.[1,7–11]

Peripartum cardiomyopathy (PPCM) usually presents within the first month postpartum and rarely before 36 weeks of pregnancy. Symptoms such as dyspnea, orthopnea, and pedal edema often resemble normal pregnancy changes, causing delayed diagnosis. In severe cases, arrhythmias or cardiac arrest may occur, and patients with LVEF <35% are at higher risk for thromboembolic events. Physical signs mimic systolic heart failure, including elevated jugular venous pressure, S3 heart sound, and mitral regurgitation murmur. Echocardiography has identified left ventricular thrombus in 16% of patients, underscoring the need for prompt and accurate diagnosis.[9,10,12,13]

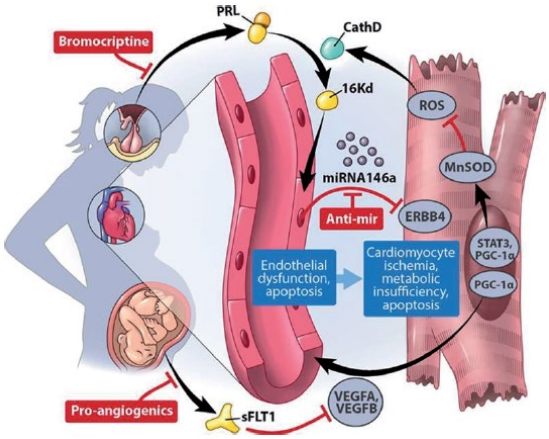


Figure 3. Pathophysiology of PPCM[2]

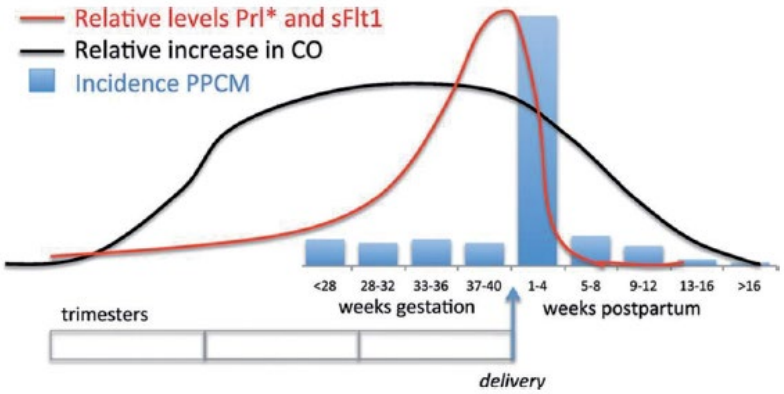


Figure 4. Pregnancy and incidence of PPCM[2]

c. Hypertrophic cardiomyopathy (HCM)

Hypertrophic cardiomyopathy (HCM) in pregnancy may involve localized or generalized thickening of the heart muscle, often causing left ventricular outflow tract (LVOT) obstruction. While increased plasma volume during pregnancy may reduce obstruction, arrhythmias and hypovolemia can worsen symptoms. Most women tolerate pregnancy well, though nearly half may experience dyspnea, angina, or syncope, especially those symptomatic beforehand. High-risk patients require close monitoring, with beta-blockers or verapamil for symptom control, and anticoagulation for arrhythmias. Vaginal delivery with regional anesthesia is preferred, while general anesthesia is avoided due to elevated risks.[1]

d. Non-compaction left ventricle (NCLV)

Non-compaction cardiomyopathy is partly congenital and marked by excessive trabeculations and deep recesses in the myocardium, posing a high risk for thromboembolic events. Diagnosing NCLV during pregnancy is challenging due to physiological increases in trabeculation seen in about 25% of healthy pregnancies. Women with NCLV may develop heart failure or arrhythmias during pregnancy, though no maternal deaths have been reported. Anticoagulation is recommended for patients with a history of thromboembolism, atrial fibrillation, intracardiac thrombi, or impaired left ventricular function. Careful cardiovascular monitoring is crucial throughout pregnancy due to the risk of deterioration.[1]

e. Restrictive cardiomyopathy (RCM)

Restrictive cardiomyopathy, either acquired or congenital, affects one or both ventricles and is characterized by increased myocardial stiffness and impaired relaxation, leading to reduced ventricular filling. During pregnancy, the increased plasma volume may precipitate left or right heart failure. Management is largely symptomatic, with beta-blockers and diuretics being the mainstay. Vaginal delivery can be successful, as reported in case studies, although careful volume management and anesthetic planning are essential due to the heart's limited ability to accommodate circulatory changes.[1,14]

f. Arrhythmogenic right ventricular cardiomyopathy (ARVC)

ARVC is a genetic cardiomyopathy involving fibrofatty replacement of right ventricular myocardium, which predisposes to arrhythmias and right heart failure. Pregnancy-related plasma volume expansion can worsen symptoms. Studies by Castrini and Gandjbakhch reported no pregnancy-related mortality but noted symptoms such as palpitations, breathlessness, ventricular tachycardia, and heart failure in some patients. Beta-blockers and antiarrhythmics like flecainide are used for management. Pre-pregnancy counseling is crucial, and symptomatic women are generally advised against conceiving due to potential risks.[1,15–17]

g. Takotsubo cardiomyopathy

Takotsubo cardiomyopathy, also known as stress-induced cardiomyopathy, most commonly occurs around the time of delivery, particularly during cesarean sections. Approximately one-third of cases are diagnosed intraoperatively. Despite its acute presentation, all affected patients in reported cases recovered within 4 days to 3 months postpartum, similar to outcomes

in non-pregnant populations. Early recognition and supportive care remain the mainstay of treatment.[1]

Diagnosis

a. History and physical examination

Cardiovascular symptoms such as dyspnea, fatigue, and edema often mimic normal pregnancy changes, leading to delayed heart failure diagnosis. However, warning signs like chest pain, exertional or resting dyspnea, orthopnea, persistent palpitations, new murmurs, tachycardia, cyanosis, and jugular venous distention warrant immediate cardiology referral. Further evaluation with imaging and lab tests is essential to distinguish pathological from physiological changes in pregnancy.[3,4,18–20]

b. Supporting investigation

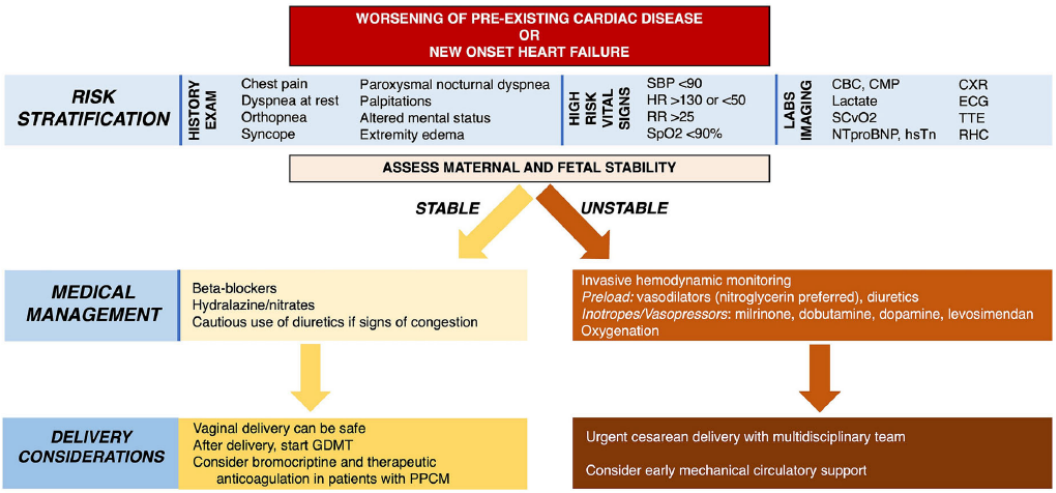
Echocardiography is the preferred and safest tool for diagnosing cardiovascular disease in pregnancy, especially peripartum cardiomyopathy (PPCM), which typically presents with global systolic dysfunction and LVEF <45%. ECG may show nonspecific changes such as sinus tachycardia or ST-T abnormalities, while chest X-ray and cardiac MRI are used selectively due to radiation or contrast concerns. CT or catheterization is reserved for critical cases. NT-proBNP and troponin are key biomarkers; low NT-proBNP (<128 pg/mL) suggests low cardiac risk, while high levels (>200 pg/mL) or elevated troponin indicate possible heart failure or myocardial injury. Combining both markers improves risk assessment, though further validation is needed in pregnancy.[2]

c. Criteria diagnosis of PPCM

PPCM is diagnosed based on three clinical criteria:[2]

1. Heart failure onset in the last month of pregnancy or within five months postpartum;
2. Left ventricular systolic dysfunction with LVEF <45%;
3. Exclusion of other identifiable causes such as accelerated hypertension, diastolic dysfunction, systemic infection, pulmonary embolism, preeclampsia, or amniotic fluid embolism.

These criteria help distinguish PPCM from other common cardiovascular complications of pregnancy and guide timely intervention.[2]



Figures 5. Assessment of worsening symptoms or acute heart failure during pregnancy[3]

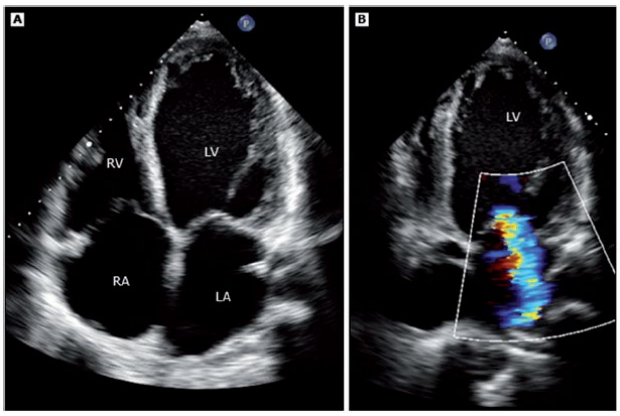


Figure 6. A: Four chamber view of PPCM. B: Mitral regurgitation on PPCM[2]

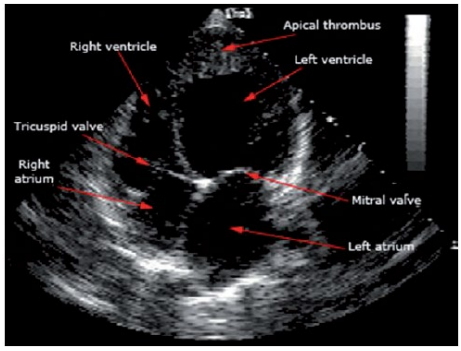


Figure 7. Four chamber view of PPCM showing the presence of thrombus[2]

General management

Management of dilated cardiomyopathy (DCM) and peripartum cardiomyopathy (PPCM) emphasizes early diagnosis, hemodynamic optimization, and individualized therapy based on pregnancy status. Treatment focuses on symptom control, arrhythmia management, thromboembolism prevention, and maternal-fetal safety. Evaluation includes history-taking, ECG, echocardiography, and NYHA classification monitoring. Safe drugs during pregnancy include beta-blockers, digoxin, and LMWH, while ACE inhibitors are reserved for postpartum. In severe cases, ICU support with inotropes or invasive monitoring may be needed. Bromocriptine may aid recovery but requires anticoagulation. ICD/CRT implantation is deferred for 3–6 months. Future pregnancies are high-risk if LVEF remains <50%, necessitating long-term follow-up, counseling, and contraception planning.[1,2,7,9]

The use of bromocriptine remains controversial. While several studies suggest its role in improving LVEF and reducing mortality in PPCM, others caution against its use due to increased thromboembolic risk, particularly without appropriate anticoagulation. Current guidelines recommend its administration only under specialist supervision and when combined with prophylactic anticoagulation.[7,8] Further randomized studies are needed to establish its efficacy and safety profile in diverse populations.

Prognosis

Recovery from peripartum cardiomyopathy (PPCM) typically occurs within 3 to 6 months postpartum, although some cases have shown improvement up to 48 months after delivery. Favorable prognostic factors include smaller left ventricular end-diastolic diameter (<5.5 cm), higher left ventricular ejection fraction (LVEF >30–35%), fractional

shortening >20% at diagnosis, absence of elevated troponin levels, absence of left ventricular thrombus, and non-African American ethnicity. Conversely, poor prognosis is associated with prolonged QRS duration (>120 ms), delayed diagnosis, higher NYHA functional class, multiparity, and African ancestry. The risk of PPCM recurrence in subsequent pregnancies is significant, thus future pregnancies are generally discouraged and require close monitoring. Maternal complications may include thromboembolism, arrhythmias, progressive heart failure, and misdiagnosis as preeclampsia, while fetal complications may involve hypoxia-induced fetal distress.[21]

Table 1. Summary of cardiac medication use during and after pregnancy in dilated cardiomyopathy (DCM) and peripartum cardiomyopathy (PPCM)[2]

Drug	Pregnancy	Recommendation During Pregnancy	Recommendation During Breastfeeding	Postpartum Use
ACE-I / ARB	Teratogenic. 48%–87% of exposed fetuses experience complications	Contraindicated.	Captopril, benazepril, and enalapril are considered safe	Use according to guidelines
ARNI	Same as ACE-I/ARB	Contraindicated	Limited data	One case report shows benefit in PPCM
Beta-blockers	May cause hypoglycemia, bradycardia, and small-for-gestational-age infants	Metoprolol recommended; atenolol contraindicated	Metoprolol: acceptable; carvedilol: limited data	Use according to guidelines
MRA	Antiandrogenic effect with spironolactone; high-dose eplerenone causes miscarriage in rabbits	Contraindicated	Not recommended	Use according to guidelines
Diuretics	Risk of oligohydramnios and electrolyte disturbance; limited human data	Furosemide and bumetanide are considered safe	Furosemide: limited data, acceptable	Use according to guidelines
Inotropes	Levosimendan is recommended in PPCM, though human data are limited	Levosimendan may be an option	Unknown	Use according to guidelines
Vasodilators	High-dose hydralazine and long-acting nitrates recommended; hydralazine is teratogenic in animals	Hydralazine is considered safe; data on isosorbide dinitrate are unknown	Switch to ACE-I/ARB/ARNI postpartum	
Ivabradine	Teratogenic in animals	Not used	Unknown	Shown beneficial effect in PPCM

Anticoagulants	LMWH if needed; discontinue 4–6 hours before planned delivery	Considered safe	Considered safe	Continue for 4–6 weeks postpartum and follow guidelines
Digoxin	Crosses the placenta.	Safe	Considered safe; minimal exposure through milk	Use according to guidelines
Bromocriptine	–	2.5 mg once daily for 1 week in mild PPCM; higher doses if EF <25% or cardiogenic shock, with anticoagulants	–	–

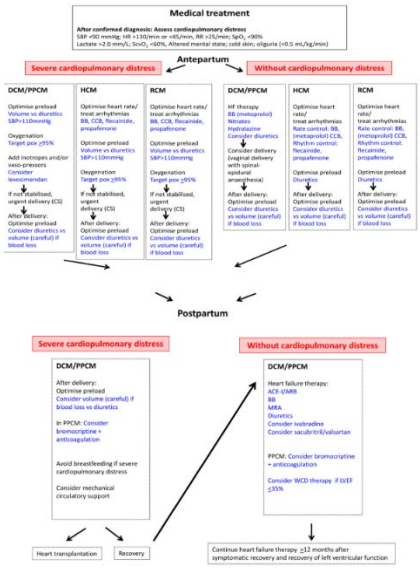


Figure 8. Treatment algorithm for DCM/PPCM, HCM, and RCM during pregnancy and after delivery[1]

Limitations and future directions

This review is limited by its narrative design and reliance on existing literature without formal quality appraisal. The lack of high-quality randomized controlled trials in pregnant populations with cardiomyopathy limits the strength of evidence for certain interventions. Future research should focus on prospective, multicenter studies to better define the safety and efficacy of emerging therapies such as bromocriptine, wearable defibrillators, and tailored heart failure regimens during pregnancy and postpartum. Additionally, more data are needed from low- and middle-income countries to improve global applicability of recommendations.

4. Conclusion

Cardiomyopathy is a rare condition that can lead to heart failure during or after pregnancy, with risk factors such as age over 30 and hypertension. Its symptoms often resemble normal pregnancy, leading to delayed diagnosis, but evaluation using echocardiography, ECG, and NT-proBNP biomarkers can aid detection. Management includes acute and chronic therapies focusing on reducing preload and afterload, along with antiarrhythmic and anticoagulant treatments; the use of bromocriptine to improve ejection fraction remains controversial.

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