

PROTOCOL 18

Peripartum Cardiomyopathy

Sarah Rae Easter¹ and Carolyn M. Zelop²

¹Departments of Obstetrics and Gynecology, and Anesthesiology, Perioperative and Pain Medicine, Brigham and Women's Hospital, Harvard Medical School, Boston, MA, USA

²Department of Obstetrics and Gynecology, NYU School of Medicine, New York, USA

Peripartum cardiomyopathy and the obstetric care provider

Peripartum cardiomyopathy (PPCM) is a clinical diagnosis of exclusion defined as heart failure with a reduced ejection fraction towards the end of pregnancy or in the immediate postpartum period. This seemingly simple definition is challenged by the complexities of caring for patients in the third trimester of pregnancy and postpartum. Physiological dyspnea coupled with the other hallmark cardiovascular adaptations of pregnancy can confound the clinical presentation, and the overlap of PPCM with other obstetric and cardiovascular diagnoses further complicate the diagnostic dilemma. Despite these challenges, the increasing contribution of both cardiovascular disease and cardiomyopathy to maternal mortality highlights the importance of a framework for diagnosis and management of the disease.

The goals of the obstetric care provider responsible for the care of a patient with PPCM are threefold. First, the provider must entertain this condition in the patient with the appropriate clinical presentation and pursue appropriate work-up to establish the diagnosis. The provider is then challenged to stabilize the patient while awaiting the input of consulting colleagues with expertise in the management. Finally, the obstetric care provider must be familiar enough with the short- and long-term management to provide input about treatment options while balancing the goals of care for the patient and her fetus or neonate.

Clinical presentation and diagnosis

Clinical symptoms that may suggest PPCM include dyspnea, orthopnea, edema, chest pain, palpitations, dizziness, or fatigue. The obvious overlap between symptoms of this life-threatening condition and a normal

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uncomplicated pregnancy challenges even the most astute historian. Reassuringly, physical exam can help distinguish normal pregnancy physiology from pathological conditions warranting further investigation.

Patients with cardiomyopathy typically demonstrate vital sign abnormalities including hypoxia, tachypnea, hypo- or hypertension, and tachycardia. Other more specific exam findings such as crackles or rales, systolic or diastolic murmur, or extra heart sounds such as an S3 or S4 heighten concerns for PPCM. Evaluation includes a chest x-ray to evaluate for pulmonary edema and an electrocardiogram (ECG) to evaluate for ischemia or arrhythmias to suggest a cardiovascular etiology of the clinical presentation. Abnormalities in these studies clearly warrant follow-up, but a normal chest x-ray or ECG in the face of an atypical history or physical exam may also warrant further investigation (Figure 18.1).

The most recent guidelines from the European Society of Cardiology outline a diagnosis of PPCM based on three features.

- Heart failure secondary to left ventricular systolic dysfunction with left ventricular ejection fraction (LVEF) <45%.
- Occurrence towards the end of pregnancy or in the months following delivery (mostly in the month following delivery).
- No other identifiable cause of the heart failure.

Echocardiography is essential for the diagnosis and can be helpful to narrow the differential diagnosis for other disease states such as preeclampsia or noncardiogenic pulmonary edema. The presence of an elevated N-terminal pro-B-type natriuretic peptide (NT-proBNP) >300 can aid in distinguishing cardiovascular pathology from other diagnoses. Ultimately, PPCM is a diagnosis of exclusion that warrants an understanding of other causes of heart failure with reduced ejection fraction (HFrEF).

Differential diagnosis

As with many other diseases, a useful framework for the differential diagnosis of the pregnant or newly postpartum patients with HFrEF is to consider conditions exacerbated by the physiological changes of pregnancy, diseases unique to pregnancy, and diseases unrelated to the pregnant state (Figure 18.2). The diagnosis is made from clinical features with transthoracic echocardiogram (TTE) and ECG.

An ECG is mandatory to evaluate for HFrEF in the setting of pregnancy-associated myocardial infarction or heart failure as a downstream consequence of arrhythmias. For patients with co-morbid arrhythmias, one must consider whether this is a consequence of the disease or related to its pathophysiology as in cases of arrhythmogenic cardiomyopathies such as arrhythmogenic right ventricular cardiomyopathy (ARVC). Once concern for HFrEF is raised, a targeted personal and family history and labs (chemistry, thyroid stimulating hormone [TSH] and assessment of urine protein) guide the provider towards the etiology and appropriate follow-up testing.

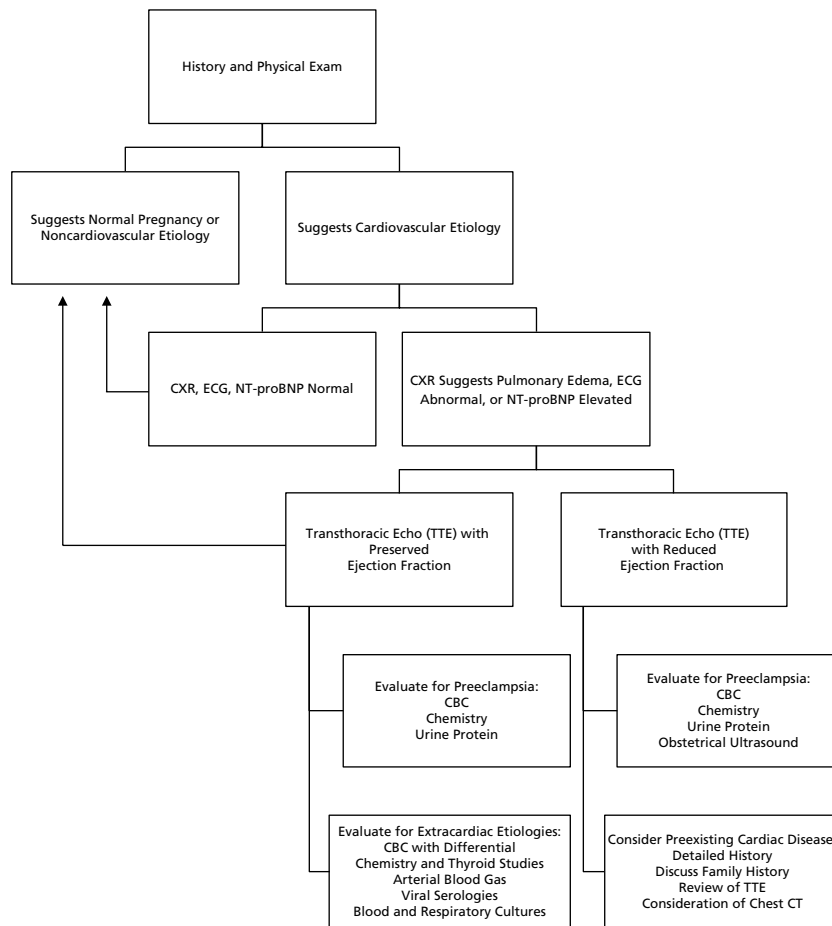


Figure 18.1 Diagnostic approach to the patient presenting with cardiopulmonary complaints. CBC, complete blood count; CXR, chest x-ray; ECG, electrocardiogram; NT-proBNP, N-terminal pro-brain natriuretic peptide.

Echocardiography can demonstrate the presence of known or newly diagnosed medical co-morbidities exacerbated by pregnancy such as congenital heart disease or valvular heart disease. The echocardiographic features in cases with reduced ejection fraction can further narrow the differential. In some cases, transesophageal echocardiography (TEE) or cardiac magnetic resonance imaging (MRI) may be useful adjuncts for better visualization of valvular structures (via TEE) or characterization of the myocardium (via MRI).

Cardiomyopathy is classically subdivided into dilated, restrictive, and hypertrophic types. Additional unclassified variants of note include stress cardiomyopathy, ARVC, and left ventricular noncompaction. Patient demographics and clinical presentation can suggest clues to the underlying diagnosis, but echocardiographic features are central to diagnostic criteria (Table 18.1).

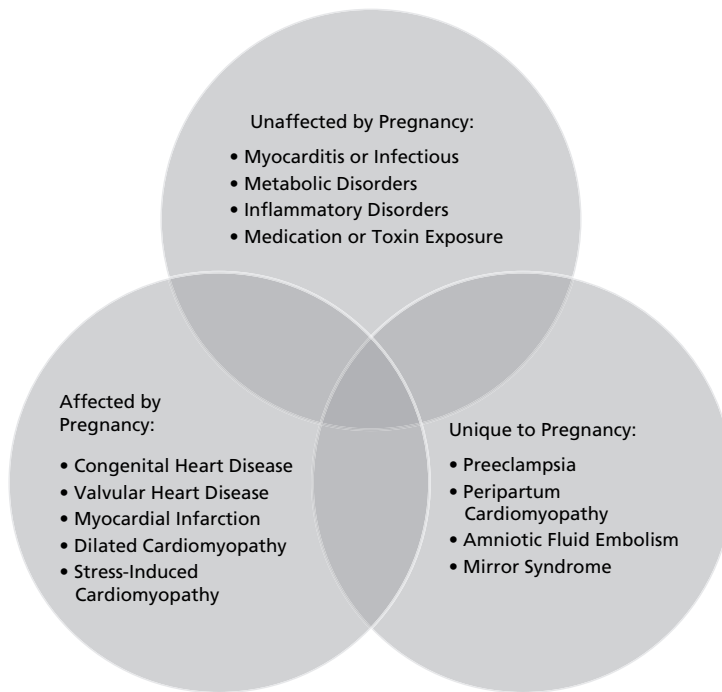


Figure 18.2 Differential diagnosis for heart failure with reduced ejection fraction in pregnancy.

Etiopathogenesis of peripartum and other cardiomyopathies

Dilated cardiomyopathy

Dilated cardiomyopathy, of which peripartum cardiomyopathy is a subtype, is the most frequently encountered type of cardiomyopathy. The echocardiographic features include dilation of the left ventricle with reduced systolic function most commonly expressed as reduced ejection fraction. Therefore, the clinical presentation of PPCM is most often that of decompensated left-sided heart failure though right-sided dysfunction can be present. Distinguishing PPCM from other forms of dilated cardiomyopathy is a diagnostic dilemma but a targeted history looking for infectious etiologies (such as viral, HIV, Lyme disease), toxic etiologies (alcohol, cocaine, medications like anthracycline derivatives), or chronic medical conditions (renal disease, sarcoidosis, lupus) can be instructive. Without historical or laboratory clues to suggest another cause of dilated cardiomyopathy, a diagnosis of PPCM is often assumed.

Understanding PPCM is complex due to the multifactorial pathogenesis and overlap with other clinical syndromes. PPCM has often been conceptualized as

Table 18.1 Clinical and echocardiographic features of variants of cardiomyopathy

Cardiomyopathy	Clinical and physiological features	Echocardiographic features	Etiologies
Dilated cardiomyopathy	Dilation and impaired contraction of one or both ventricles Heart failure Arrhythmias	LV spherical dilation Normal or reduced WT Reduced systolic indices ^a Four-chamber enlargement \pm Reduced TAPSE ^b \pm RWM abnormalities ^c	Infectious Medications Toxic Inflammatory Genetic mutations Peripartum Genetic mutations in sarcomere genes
Hypertrophic cardiomyopathy	LVOT obstruction (often dynamic) Diastolic dysfunction Myocardial ischemia Mitral regurgitation Systolic dysfunction (end stage)	Hypertrophied LV Nondilated LV Asymmetrical septal hypertrophy Septal WT \geq 15 mm \pm LVOT Obstruction \pm SAM of MV	
Restrictive cardiomyopathy	Pulmonary and systemic congestion History of co-morbid diagnosis	Biatrial enlargement No RV/LV hypertrophy No RV/LV dilation	Familial Infiltrative Storage diseases Endomyocardial fibrosis Genetic Desmosomal gene mutations
Arrhythmogenic right ventricular cardiomyopathy (ARVC)	Ventricular arrhythmias Sudden cardiac death	Abnormal RV function Regional RV akinesis	

(continued)

Table 18.1 (Continued)

Cardiomyopathy	Clinical and physiological features	Echocardiographic features	Etiologies
Stress cardiomyopathy	Acute substernal chest pain History of emotional or physical stress LVOT obstruction Mimics acute coronary syndrome Heart failure Thromboembolism Ventricular arrhythmias	LV systolic dysfunction Apical LV ballooning Hyperkinesis of basal walls ± LVOT obstruction Prominent LV trabeculae Continuity between LV cavity and deep intertrabecular recesses	Associated with excess catecholamines Must exclude evidence of coronary artery disease, typically with angiography
Left ventricular noncompaction			Due to intrauterine arrest of compaction of loose interwoven meshwork

^a Systolic indices include LV fractional shortening, fractional area change, and ejection fraction.

^b Reduced tricuspid annular plane systolic excursion (TAPSE) is a marker of right ventricular dysfunction. Reduced TAPSE not necessary to make the diagnosis but TAPSE <14 mm associated with poor prognostic outcome in cases of dilated cardiomyopathy.

^c Regional wall motion abnormalities should raise concern for ischemic cardiomyopathy or stress cardiomyopathy in cases of apical ballooning without coronary artery disease.

LV, left ventricle; LVOT, left ventricular outflow tract; MV, mitral valve; RWM, regional wall motion; RV, right ventricle; SAM, systolic anterior motion; WT, wall thickness.

a failure of the stress test of pregnancy and its associated increase in plasma volume, heart rate, and cardiac output. The contemporary understanding coupled with the timing of presentation seems to refute this notion. Women with PPCM tend to present towards the end of pregnancy or in the postpartum period which is well beyond the physiological increase in hemodynamic burden accompanying pregnancy. Evidence further supporting the distinct pathophysiology of PPCM comes from both basic science and translational research.

Commonly encountered risk factors for PPCM include maternal age >30 years, African descent, multiple gestation, and a history of hypertension or preeclampsia. More rare but notable risk factors include long-term tocolytic therapy with beta-adrenergic agonists such as terbutaline, maternal cocaine abuse, and nutritional deficiencies (such as selenium). These risk factors have a synergistic as opposed to simply additive effect on the odds of developing PPCM. In an epidemiological study, the presence of one risk factor conferred a twofold increase in the odds of PPCM whereas the presence of two risk factors conferred an 11-fold increase.

Development of PPCM may be mediated through a variety of pathways related to a genetic predisposition, antiangiogenic factors from the placenta, endothelial damage, and the excretion of prolactin in conjunction with oxidative stress (Figure 18.3). The presence of a genetic predisposition is suggested by both pedigrees and the wide variation in incidence globally, ranging from a reported 1 in 100 patients in Zaria, Nigeria, to 1 in 20000 live births in Japan. In a landmark prospective study, researchers sequenced 43 genes associated with dilated cardiomyopathy and identified variants in 15% of patients with PPCM. The rate of genetic abnormalities in this cohort

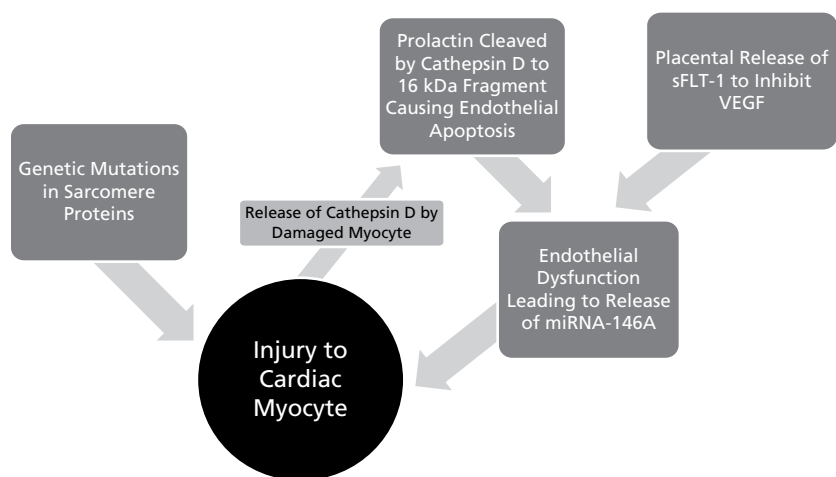


Figure 18.3 Contemporary understanding of the pathophysiology of peripartum cardiomyopathy.

parallels the 17% prevalence of variants in patients with idiopathic dilated cardiomyopathy, suggesting a common genetic basis for this condition. Moreover, specific genetic variants such as truncating mutations in *TTN* – the gene encoding the sarcomeric protein titin – were associated with lower odds of recovery of cardiac function.

An underlying genetic predisposition may explain part of the pathology but additional mediators of endothelial damage and oxidative stress leading to damage of cardiac myocytes play an important role in the pathogenesis of the disease. Basic science studies have revealed a role for both prolactin and the placenta in the mechanism of the disease. In times of increased oxidative stress, the enzyme cathepsin D cleaves prolactin released from the anterior pituitary to a 16 kDa fragment that causes endothelial apoptosis. The expression of soluble fms-like tyrosine kinase receptor 1 (sFlt-1) is an antiangiogenic protein that sequesters circulating vascular endothelial growth factor leading to endothelial dysfunction in a similar manner to preeclampsia. Endothelial dysfunction leads to increased expression of microRNA MiR-146a which blocks several important pathways leading to cardiac myocyte death. Apoptosis of myocytes leads to additional stress and release of cathepsin D, providing a positive feedback loop for cardiac damage. These mechanisms of injury underscore the challenges of distinguishing PPCM from other diseases with an overlapping clinical presentation, including dilated cardiomyopathy and preeclampsia.

Hypertrophic cardiomyopathy

In contrast to PPCM, other cardiomyopathies typically present with preserved ejection fraction except for those with end-stage disease. For example, hypertrophic cardiomyopathy is characterized by a hypertrophied left ventricle without dilation. Although chronic diseases like hypertension or valvular heart disease in the setting of aortic stenosis can lead to hypertrophy of the left ventricle, hypertrophic cardiomyopathy classically refers to asymmetrical septal hypertrophy due to mutations in the genes encoding the sarcomere proteins in the contractile apparatus. This asymmetrical septal hypertrophy can lead to a dynamic left ventricular outflow tract (LVOT) obstruction characterized by systolic anterior motion (SAM) of the mitral valve. This finding reflects movement of the mitral valve to the hypertrophied intraventricular septum leading to obstruction of the LVOT and reduced stroke volume. The dynamic nature of this obstruction is a hallmark of the disease and is worsened with decreased LV filling, which can be exacerbated by decreases in the systemic vascular resistance or tachycardia. The thickened hypertrophied ventricle can also predispose the patient to ischemia, ventricular arrhythmias, and cardiac death. Understanding this hallmark physiology helps anticipate the adaptation to pregnancy and inform the treatment needs for pregnant women with this co-morbidity.

Restrictive cardiomyopathy

Like hypertrophic cardiomyopathy, restrictive cardiomyopathy typically presents with diastolic dysfunction and preserved LV systolic function. The clinical presentation often includes pulmonary congestion manifest as pulmonary edema as well as systemic congestion with associated hepatosplenomegaly and ascites. Atrial arrhythmias can be common due to the increased burden of work with atrial contraction to force blood into a stiff and noncompliant ventricle. Echocardiographic features include a nondilated ventricle with normal wall thickness with impaired relaxation and diastolic filling from stiff noncompliant ventricles. Biatrial enlargement is often seen and excluding evidence of constructive pericarditis is essential. Restrictive cardiomyopathies are rare and can be caused by familial syndromes, infiltrative disorders (amyloidosis, sarcoidosis), glycogen storage diseases, or endomyocardial fibrosis. The clinical history can give insight into possible etiologies of the disease, but MRI can be necessary to make the diagnosis.

Stress cardiomyopathy

Though other rare cardiomyopathy variants exist, a relevant unclassified disorder that should be on the differential diagnosis for a pregnant or postpartum patient presenting with evidence of HF_rEF is stress cardiomyopathy. Stress cardiomyopathy is a catecholamine-associated phenomenon that can present with evidence of acute coronary syndrome (ACS) including substernal chest pain, ST segment elevations, and elevated troponins. The hallmark echocardiographic finding is ballooning of the apex of the left ventricle which informed the initial name of the disease – takotsubo cardiomyopathy – taken from the Japanese name for octopus trap with a similar appearance of the left ventricle. Interrogation of the coronary arteries to exclude coronary artery disease may be required in cases with clinical presentation overlapping with ACS. Distinguishing this entity from PPCM is clinically relevant as the management of stress cardiomyopathy and dilated cardiomyopathy, particularly in cases of decompensated heart failure, is vastly different. As with the other cardiomyopathies, an understanding of the echocardiographic features and pathophysiology is of critical importance, both to guide acute treatment and provide anticipatory guidance about prognosis and recurrence risk.

Stabilization and treatment

Immediate management of PPCM mirrors that of acute heart failure: optimization of preload typically with diuretics, afterload reduction, and supportive care. Frequent hemodynamic monitoring including cardiac

telemetry, continuous fetal monitoring, and close urine output monitoring with the placement of a urethral catheter are warranted. In addition to assessment of heart rate, blood pressure, and respiratory status, other components of the clinical exam should be considered, including evidence of tissue hypoperfusion such as oliguria, cold skin either centrally or peripherally, an elevated lactate, or a depressed mixed or central venous oxygen saturation for those rare patients in whom invasive hemodynamic monitoring is warranted. The obstetric care provider should understand and explain maternal-fetal physiology to the multidisciplinary team, and counsel the patient and her family about goals of care.

Stabilization of acute heart failure

Supplemental oxygen may be necessary with a target of an $\text{SpO}_2 >95\%$ for pregnant patients and $\text{SpO}_2 >90\%$ for postpartum patients. Supplemental oxygen in the absence of hypoxemia is not warranted and may reduce cardiac output via vasoconstriction. For patients with refractory hypoxia, noninvasive mechanical ventilation (NIV) reduces the need for intubation in those with cardiogenic pulmonary edema likely due to the hemodynamic impact of positive pressure ventilation in addition to the respiratory support. The use of continuous positive airway pressure (CPAP) or bilevel positive airway pressure (BiPAP) provides additional hemodynamic support through the increase in intrathoracic pressure and reduction of preload and LV afterload.

Optimization of preload is an essential stabilizing measure for patients presenting with heart failure. For most patients with PPCM and other HF_{rEF}, this typically means diuresis though volume overload should not be assumed. Patient positioning in left lateral tilt 30–90° may help to mitigate the effect of aortocaval compression upon cardiac output when uterine size is greater than or equal to 20 weeks' gestation. Clinical exam such as assessment of the jugular venous pulsation (JVP) or lower extremities for evidence of systemic congestion is essential. Additional maneuvers such as the passive leg raise or point-of-care TTE to assess left ventricular filling and for evidence of collapsibility of the inferior vena cava can aid in this assessment. For patients presenting with evidence of volume overload, the use of furosemide or other loop diuretics should be initiated at a low dose and quickly escalated until a clinical response is achieved. For diuretic-naïve individuals, beginning with 10 mg IV furosemide and doubling this dose in 30-minute intervals would be a favored approach.

Afterload reduction is the other critical component of hemodynamic optimization for patients with PPCM and is classically achieved with the use of nitrates to target a systolic blood pressure of <110 mmHg. Infusion of vasodilators may not be immediately attainable on the labor and delivery unit, so hydralazine is a reasonable alternative to achieve this goal. While beta-blockers are contraindicated in acute decompensated heart

failure, their use may be more appropriate adding rate control during the stabilized compensated phase of heart failure management. Oral antihypertensives are avoided due to their longer half-life and challenges with titration compared to intravenous medications.

Despite these measures, some women may require additional hemodynamic support to maintain a perfusing cardiac output through inotropes or mechanical circulatory support devices such as intraaortic balloon pump (IABP), percutaneous ventricular assist devices (e.g., Impella®, Protek Duo®) or venoarterial extracorporeal membrane oxygenation (VA-ECMO). Vasopressors often used first line for undifferentiated or cardiogenic shock, such as norepinephrine and epinephrine, are often avoided in favor of inotropic agents such as milrinone due to the catecholaminergic effects of the beta-agonists.

Cardiomyopathies characterized by LVOT obstruction such as hypertrophic cardiomyopathy or stress cardiomyopathy should not receive inotropes and nitrates but may paradoxically benefit from pure alpha-agonists like phenylephrine to increase systemic vascular resistance and preload, and decrease LVOT obstruction. As opposed to PPCM or other dilated cardiomyopathy, beta-blockers should be considered in the acute setting for hypertrophic, stress, and restrictive cardiomyopathy to control heart rate. Rate control is a mainstay of therapy for patients with heart failure associated with LVOT obstruction to allow for adequate ventricular filling and to reduce SAM pathophysiology as a cause of decreased cardiac output. Low doses of IV metoprolol such as 2.5–5 mg escalated in 2.5 mg intervals to maintain a target heart rate can be considered though some patients may benefit from continuous infusions such as esmolol due to its short half-life and easy titration. Patients requiring IV infusions of medications or inotropes may benefit from invasive hemodynamic monitoring with arterial lines, central venous catheters, or pulmonary artery catheters.

Management of women with heart failure is best achieved through a collaborative multidisciplinary approach involving obstetricians or maternal-fetal medicine specialists, cardiologists with familiarity with pregnancy, anesthesiologists with obstetric and possibly cardiac experience, pharmacists, neonatologists, and nurses. The availability of cardiac surgeons, intensivists, cardiac catheterization specialists, and heart failure specialists with expertise in mechanical circulatory support is ideal. Consideration of transfer to an intensive care unit or perinatal referral center with experience in the management of cardiomyopathy in pregnancy may be warranted.

Location and timing of delivery

An essential topic for discussion amongst the multidisciplinary team is the timing, location, and mode of delivery for patients presenting in pregnancy (Figure 18.4). In general, delivery is recommended for unstable PPCM but timing of delivery depends on gestational age of the fetus and the underlying

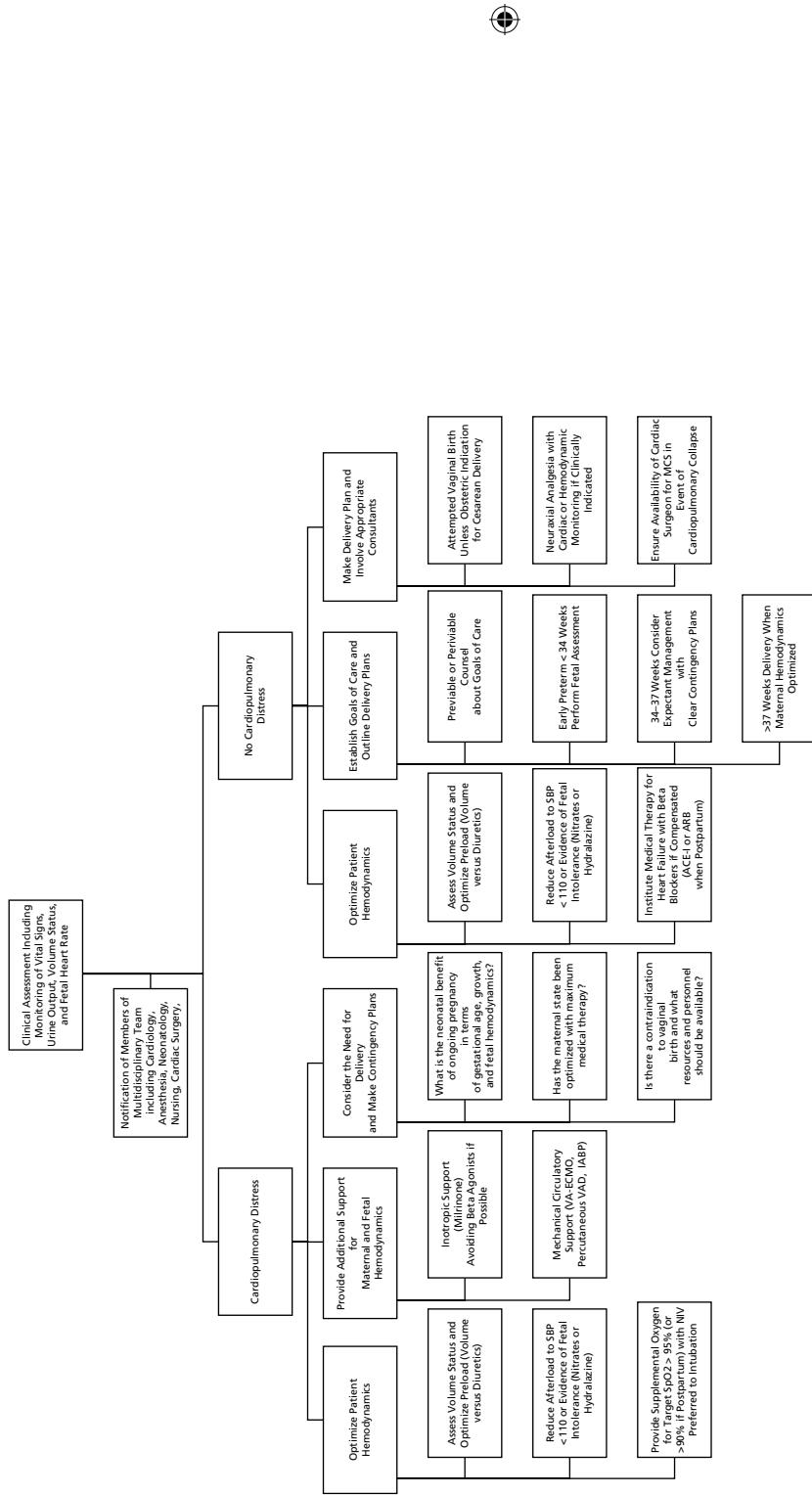


Figure 18.4 Framework and clinical pearls for stabilization and ongoing management of the pregnant patient with periparturient cardiomyopathy from the standpoint of the obstetric care provider. ACE-I, angiotensin converting enzyme inhibitor; ARB, angiotensin receptor blocker; ECMO, venoarterial extracorporeal membrane oxygenation; IABP, intraaortic balloon pump; MCS, mechanical circulatory support; NIV, noninvasive ventilation; SBP, systolic blood pressure; SpO₂, oxygen saturation; VA-VAD, ventricular assist device.

pathophysiology of the patient, and requires shared decision making among the patient, her family, and her extended care team. Patients at an early preterm gestation who stabilize with medical management can continue with expectant management. The obstetrician can be helpful in educating colleagues about anticipated neonatal outcomes at a given gestational age category and to help weigh the maternal risks of ongoing pregnancy against potential fetal benefit. Delaying delivery to allow for optimization of maternal hemodynamics is warranted independent of gestational age.

An attempted vaginal delivery is recommended for patients without an obstetric contraindication, with consideration of cesarean delivery for causes of acute decompensated heart failure. Neuraxial analgesia is the preferred mode of anesthesia to avoid the hemodynamic shifts with induction of a general anesthetic. These decisions should be individualized and should consider the hemodynamic status of the patient, obstetric comorbidities, and future fertility goals. Outlining a delivery plan for patients requires multidisciplinary input with an overarching goal of prioritizing maternal stability to improve both maternal and fetal outcomes. Contingency plans in the event of fetal distress or maternal decompensation should be discussed specifically with an emphasis on the role of delivery in cases of maternal cardiac arrest.

Chronic therapy and long-term follow-up

After initial stabilization and delivery, chronic heart failure therapy mirrors that of the general population and includes beta-blockers, angiotensin converting enzyme inhibitors (ACEi) or angiotensin receptor blockers (ARB), and diuretics. Prophylactic or even therapeutic anticoagulation may be recommended for those with a persistently depressed ejection fraction less than 30–35% to avoid thromboembolic complications. Patients with a depressed ejection fraction <35% may also be prescribed a wearable cardioverter for prevention of arrhythmia-associated sudden cardiac death while awaiting LV recovery. Women with a depressed ejection fraction after 3–6 months should generally be referred for consideration of implantable cardioverter-defibrillator. For those patients whose course has been complicated by arrhythmias, additional chronic therapy may be warranted. Considering the range of available options for the aforementioned medications, an understanding of the patient's desire for breastfeeding is essential to the multidisciplinary team to ensure the choice of medications within each class with the best safety profile for lactation.

The role of prolactin in the pathogenesis of PPCM has led some to question the safety of breastfeeding and even consider bromocriptine to suppress prolactin and lactation. Data informing the use of bromocriptine come from case series or uncontrolled studies and the benefits of breastfeeding for both mother and baby are well established. Advising against breastfeeding or the

uniform use of bromocriptine is controversial and not universally recommended. The current guidelines from the European Society of Cardiology support its use but the American Heart Association guidelines consider its use investigational. Breastfeeding may be a priority for many women but for others can be a daunting challenge in the setting of critical illness. The obstetric care provider can play an essential role on the multidisciplinary team by helping understand the patient's goals of care surrounding breastfeeding.

Ultimately, 50–80% of women with PPCM recover to an ejection fraction >50% at six months, though 2–7% may require ventricular assist device placement as a bridge to either transplant or recovery of ventricular function. LVEF >30% or LV end-diastolic diameter <6 cm at the time of presentation are the most important favorable prognostic features. Conversely, in a cohort of women with PPCM, no patient with LVEF <30% and LV end-diastolic diameter >6 cm recovered systolic function. Close clinical follow-up with the cardiology team, serial echocardiography, and referral to a cardiovascular geneticist are warranted.

For those who recover, some desire another pregnancy. Postpartum follow-up should include counseling about reliable contraception and the critical importance of preconception care prior to seeking another pregnancy. Input from the cardiologist and a cardiovascular disease assessment should be undertaken, but in general women with a LVEF <50% should be advised against pregnancy due to risk of relapse and mortality. Women with partial or full recovery of LVEF have a risk of relapse in the subsequent pregnancy but can consider this under the close care of a multidisciplinary pregnancy heart team.

Cardiomyopathy pearls for the obstetric care provider

Peripartum cardiomyopathy is a rare but serious disease that should be considered for any patient presenting with cardiopulmonary complaints accompanied by objective evidence. Echocardiographic features establish the diagnosis of a given type of cardiomyopathy but there is significant clinical and pathophysiological overlap between entities. An understanding of the pathophysiology of the disease state can help guide the treatment which should focus on maternal stabilization and contingency planning in conjunction with a multidisciplinary team. The obstetric care provider plays an essential role in coordinating a team approach that prioritizes both maternal and neonatal outcomes while keeping the patient's goals of care at the forefront of clinical decision making.

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