ISSN: 2634 - 1255

Journal of Gynecology Research Reviews & Reports

SCIENTIFIC Research and Community

Review Article

Open Access

Management of Peripartum Cardiomyopathy as Challenge Collaboration Between Cardiologists and Obstetricians

Sonila Bele

Department of Medical Imaging and Clinical Semiology Faculty of Medicine, University of Medicine, Tirana, Albania

ABSTRACT

Peripartum Cardiomyopathy (PPCM) is an idiopathic cardiomyopathy presented the last trimester during pregnancy or few months after the delivery and is associated with left ventricular systolic dysfunction. The incidence of PPCM is highly variable depending on the race and geographic regions, maternal age, multiple gestation pregnancies, hypertensive disorders of pregnancy. Diagnosis is often delayed, as symptoms misinterpreted those of normal pregnancy. During pregnancy cardiac images are limited in modalities that do not allow any sort of radiation but echocardiography is safe and should be performed in any suspected case of heart failure. The clinical outcome of PPCM is also diverse from complete recovery to death. In women with PPCM although the treatment and medical therapy are similar to those in patients with HF with reduced ejection fraction due to other causes requires special modifications for fetal safety. The mode of delivery in patients with PPCM during pregnancy should be discussed with cardiologists, obstetrics, anesthetists and neonatologist that should work together for the treatment of such patients and to minimize the risk for mother and the fetus. It is considered very risky for the patients to consider another pregnancy if the LV function is not recovered to normal and the patients have to be very aware for such a situation. Pre-conception cardiologic evaluation are important for every woman with a cardiomyopathy who is considering having a family

***Corresponding author**

Sonila Bele, Department of Medical Imaging and Clinical Semiology Faculty of Medicine, University of Medicine, Tirana, Albania.

Received: August 23, 2025; **Accepted:**August 29, 2025; **Published:** September 05, 2025

Keywords: Peripartum Cardiomyopathy, Left Ventricular Dysfunction, Pregnancy

Abbreviations:
PPCM: Peripartum Cardiomyopathy
HF: Heart Failure
ECG: Electrocardiogram
EF: Ejection Fraction
LV: Left Ventricle
LVEF: Left Ventricle Ejection Fraction
RV: Right Ventricle
LBBB: Left Bundle Branc Block
ACEI: Angiotensin-Converting Enzyme Inhibitor
ARB: Angiotensin Receptor Blocker
ARNI: Angiotensin Receptor Neprilyzin Inhibitor
MRA: Mineralo Receptor Antagonist
NT-Pro BNP: N Terminal Pro-Brain Natriuretic Peptide
IUD: Intrauterine Device

Introduction

During pregnancy, heart failure was first identified in 1849 by Ritchie, and it was commonly described as cardiomyopathy in the 1930s. But, lately the Peripartum Cardiomyopathy has been defined in a more explicit way. The pathophysiological mechanisms that explain the disease are quite clear now, due to knowledge gained and the increase in the number of tests conducted and the diagnostic criteria for peripartum cardiomyopathy that involve [1].

- The systolic dysfunction of left ventricular in echocardiography,
- Healthy women that are diagnosed with cardiac failure during the last month of pregnancy or within 6 months of delivery.
- Healthy women without cardiac disease before pregnancy and first four month of pregnancy.
- Healthy women with cardiac failure with unknown and/or undetermined etiology.
- The outcome of PPCM could be full recovery or regression to chronic heart failure to death.

According to a statement from the 2010 Peripartum Cardiomyopathy Working Group and the 2019 Heart Failure Association of the European Society of Cardiology Working Group defines PPCM as an idiopathic cardiomyopathy presenting with heart failure secondary to left ventricular systolic dysfunction towards the end of pregnancy or in the months following delivery, where no other cause is found. The definition of the PPCM has been elaborated because women who presented prior to the last month of pregnancy and up to 6 months postpartum were found to be clinically very similar from patients with classically defined with herat failure. For that reason, a thorough evaluation is necessary in order to eliminate other potential cardiac and non-cardiac explanations for the patient's clinical presentation.

Epidemiology and Risk Factors

Regardless the epidemiologic studies worldwide, it is difficult to define the incidence of Peripartum Cardiomyopathy. The true incidence is unknown. The incidence is highly variable depending on the race and geographic geographic regions. A population-based study showed the incidence of 10.3 patients per 10,000 live births. The disease is more prevalent in Africa and Asia and the incidence is higher among Nigerian women; nearly 1 out of 100 live births. The incidence of PPCM was 1 in 1741 deliveries in South Korea and 1 in 20,000 deliveries in Japan. In the United

States, reported incidence ranges from 1 in 1,000 to 1 in 4,000. The study of risk factors showed that the incidence increases with age with a maximum incidence of 40 to 54 years, teenage, smoking, diabetes, pre-eclampsia, hypertension of pregnancy, multiple and twin pregnancies due to fertility. Together with these factors the genetic variation might be cause for the various incidents. The PPCM treatment and other types of HF treatments are similar but, in the PPCM cases besides the management of arrhythmia, anticoagulation therapy, and mechanical support, the bromocriptine therapy is additional. It is considered very risky for the patients to consider another pregnancy if the LV function is not recovered to normal and the patients have to be very aware for such a situation [2-11].

Physiological Changes in Women During Pregnancy

During the pregnancy different physiological changes in the cardiovascular system happen. Blood volume increases by 40%, that means increase in cardiac output. Until the 32nd week of pregnancy the increase in cardiac output is realized by a rise in stroke volume and, later in pregnancy, an increase in the heart rate. The increases in plasma volume and cardiac output are stimulated by vasodilation, which occurs early in pregnancy owing to hormonal influences, especially relaxin. This hormone is produced by the corpus luteum in pregnancy, but also within the vasculature. Remodelling of placental vessels in the second trimester results in a further decrease in systemic vascular resistance accompanied by a lowering of the blood pressure. The above circulatory changes are matched by a 10–20% increase in the left ventricular mass. An important signalling pathway involved in this physiological hypertrophic response includes activation of calcineurin by a rise in calcium early in pregnancy. Progesterone and estrogen levels increase during pregnancy and influence the cardiovascular system. The hypercoagulable state occurs as a result of the rise in a number of blood coagulation factors and a reduction in fibrinolytic activity. The stroke volume increases during labour due to patient's anxiety and pain, and an increased venous return, as a consequence of the uterine contractions. Whereas after delivery, the increased cardiac output happens as a result of the blood loss during labour, auto-transfusion due to uterus contraction, and decompression of the inferior caval vein and absorption of edema [12-14].

Pathophysiological Changes in Women During Pregnancy

During pregnancy and post-partum, women with cardiomyopathies are exposed to a higher risk of cardiac function deterioration, also as a result of physiological changes of the cardiovascular system together, and also due with significant metabolic changes. Heart failure, arrhythmias, thrombo-embolic events may occur despite optimal medical treatment. The risk of cardiac complications appear higher with the progress of left ventricular dysfunction, poor functional class (NYHA class III or IV), or previous cardiac events. Asymptomatic patients are likely to tolerate the pregnancy and therefore there is not enough evidence to assess the cardiac complications. Until now the exact mechanisms of pathogenesis are not clear. But, inflammation, abnormal autoimmune responses, apoptosis, angiogenic imbalance, vascular damage and genetic factors are believed to play a role in pathogenesis. But, lately the application cascade of oxidative stress, the prolactin-cleaving protease cathepsin- D, and the hormone prolactin, suggested to the use specific therapy, for example, the use of bromocriptine for the blockage of prolactin. Angiogenic imbalance may explain why PPCM develops in late pregnancy, and why pre-eclampsia and multiple pregnancies are important risk factors [15-17].

Diagnosis of Peripartum Cardiomyopathy

According to 2018 European Society of Cardiology Guidelines states that PPCM presents with HF secondary to LV systolic dysfunction towards the end of pregnancy and in the months following delivery, with the majority diagnosed post-partum. Most women with PPCM develop symptoms within the first few months following delivery, rather than during pregnancy Diagnosis is often delayed, as symptoms are likely similar those of normal pregnancy. In all these patients, an accurate history and clinical examination are recommended. Presenting symptoms in PPCM patients are differently but may include fatigue, decreased exercise tolerance, dyspnea, orthopnea, tachypnea, palpitations, tachycardia, severe arrhythmias, chest pain, and abdominal discomfort, jugular venous distension, peripheral edema Signs of both right and left heart failure including rales, gallop rhythm, peripheral edema and ascites may be present. Blood pressure is usually normal or low due to low cardiac output. Although the hypercoagulable states is physiological changes in women during pregnancy and the postpartum period, patients may present with symptoms of pulmonary embolism or neurologic symptoms due to acute cerebrovascular events. In patients with signs, symptoms, or history of PPCM ECG, echocardiography, biomarkers and chest x-rays should be considered. The 12-lead ECG is a widely available, simple, and inexpensive tool that is able to help to assessing of cardiac risk. ECG may show nonspecific changes like nonspecific ST-T abnormalities, low voltage complex sinus tachycardia, sinus bradycardia, arrhythmias, interventricular delay and LBBB pattern. In some cases the electrocardiogram may show nonspecific abnormalities, but a normal electrocardiogram does not rule out PPCM. During pregnancy cardiac images are limited in modalities that do not allow any sort of radiation but echocardiography is safe and should be performed in any suspected case. Peripartum cardiomyopathy may be diagnosed for the first time during pregnancy by echocardiography only when the following criteria are met: left ventricular ejection fraction (LVEF) <0.45 or M-mode fractional shortening <30% (or both) and end-diastolic dimension >2.7 cm/m2. In addition to systolic dysfunction, the echocardiogram may demonstrate LV and right ventricular dilatation and/or dysfunction, functional mitral and/or tricuspid regurgitation, pulmonary hypertension, and left atrial or biatrial enlargement. Intracardiac thrombus may occur, and the LV apex should be clearly visualized particularly when the LVEF is severely reduced. Cardiac magnetic resonance imaging provides may used when the echocardiogram is inadequate, but gadolinium is avoided during pregnancy. Levels of brain natriuretic peptide (BNP) and N-terminal pro-BNP, which do not change significantly during normal pregnancy and may be mildly elevated in the setting of pre-eclampsia, are usually markedly elevated in PPCM. Chest x-rays show pulmonary venous congestion, pulmonary edema, bilateral pleural effusion, evidence of cardiomegaly or it may be normal [18-24].

Differencial Diagnosis

Differencial diagnosis is recommended because the symptoms could misinterpreted. The symptoms of PPCM may mimic normal physiological findings of pregnancy and the postpartum period. To avoid missdiagnosis, would need careful attention to differential between acute pulmonary edema from severe preeclampsia ,eclampsia or prolonged tocolysis, pulmonary embolism, amniotic fluid embolism syndrome, cardiomyopathy related to myocarditis, cardiomyopathy related to other acute conditions such as sepsis , treatment in intensive care unit,post-respiratory arrest, severe pneumonia,asthma, cardiomyopathy related to other systemic medical diseases such as hemochromatosis,systemic lupus erythematosus,antiphospholipid syndrome and chemotherapy-

related cardiomyopathy which is related to myocardial injury and also left ventricular dysfunction . But don't forget to make differencial diagnosis from previous history of cardiac disease, undiagnosed congenital or valvular disease,cardiac dysfunction in arrhythmogenic right ventricular cardiomyopathy secondary to arrhythmia, ischemia or stress cardiomyopathy, Takotsubo cardiomyopathy, a family history of cardiomyopathy or sudden cardiac death. Althought it is important to exclusion of a patient from diagnosis of PPCM should accentuating be making a diagnosis for patient.

Clinical Outcome in Peripartum Cardiomyopathy

The clinical outcome in PPCM is diverse from complete recovery to death. PPCM may be associated with mortality or severe and lasting morbidity, including brain injury, cardiopulmonary arrest, pulmonary edema, thromboembolic complications, mechanical circulatory support, cardiac transplantation, and death. Predictors of major adverse events associated with PPCM have not been fully selected. But review of cases for LVEF at the time of diagnosis is the most reliable predictor of adverse events or long-term recovery. The review of cases with LVEF <30% was associated with lower rates of recovery and increased risk of adverse events. Studies suggested that additional predictors of worse outcome include LV dilatation, LV thrombus, RV systolic dysfunction and obesity. Concomitant preeclampsia has been associated with lower 1-year survival, but higher rates of LV recovery in survivors. High resting heart rate is a predictor of adverse outcome in PPCM, and treatment with ivabradine may be useful if the patient is not pregnant or breastfeeding. Predictors of maternal mortality are NYHA class III/IV and EF <40%. Highly adverse risk factors include EF <20%, mitral regurgitation, RV failure, atrial fibrillation, and/or hypotension(38). Mortality estimates differ significantly based on racial groups, geographical region, and duration of follow-up. The estimated mortality of 1.36% to 2.05% (95% confidence interval 0.29% to 10.8%) is less than previously reported from most case series. (40) Prospective larger cohort studies have mainly focused on 6month outcomes, reporting a mortality ranging from 2.0% in Germany to 12.6% in a large cohort of 206 patients with PPCM from South Africa. A prospective study over 24 months from Turkey reported a 24% mortality. (3,41,42).The recovery frequently occurs within the first 3 to 6 months. Delayed recovery can also occur, even up to 2 years following diagnosis. According to retrospective study reported 43% recovered to LVEF of ≥50% by 12 months with a median time to recovery of 8 months. Fortunately PPCM has been associated with a higher rate of recovery than other forms of HF with reduced LVEF. Up to 20–25% of women with PPCM develop end-stage heart failure, while one-third to a half show recovery to normal left ventricular function. When reduced LV function persists, but also after full recovery of PPCM, there is an increased risk of mortality and of developing heart failure in subsequent pregnancies. On the other hand there are no similar data in other studies. German patients suffering from PPCM with concomitant hypertension had a good recovery rate (97% of hypertensive patients). In the Japanese cohort, hypertension-induced protection from bad outcome was milder, with the same death and hospitalization rate when compared to the non-hypertensive PPCM women. However, the investigators observed a shorter duration of hospitalization in PPCM patients with hypertension compared to the non-hypertensive PPCM women. The outcome was also different between the two populations, with a high in-hospital mortality in South Korea and 4% mortality in Japan Sudden cardiac death has been reported in PPCM patients with decreased LVEF in both the acute and chronic stages of this disease, as well as in those whose LVEF has completely normalized, indicating that the risk of sudden cardiac death may persist well into recovery in this patient population [23-48].

Management of Peripartum Cardiomyopathy

Both the treatment and medical therapy seemed to be similar in patients with HF with reduced ejection fraction due to different causes and those with PPCM. However, the management of HF during pregnancy need to be careful to ensure fetal safety by consulting together cardiologist and obstetrician. Every patient that complicated with acute severe heart failure should be transferred to the Intensive Care Unit for better treatment that should be determined by the clinical status.If a patient is in cardiogenic shock or haemodynamic instability and dependent on inotropes or vaso- pressors, she should be transferred early to a facility where mechanical circulatory support teams are available. It must be considered to avoiding foetotoxic agents (ACE inhibitors, ARB, ARNI, MRA, and atenolol). HF with pulmonary congestion is treated with loop diuretics and thiazides if required however, diuretics should be avoided in the absence of pulmonary congestion, due to the potential reduction in placental blood flow. Furosemid and hydrochlorthiazide are mostly used for these reason. In PPCM patients with very low LVEF, prophylactic anticoagulation should be considered. Standard indications for anticoagulation in PPCM apply during and after pregnancy. The choice of anticoagulant agent depends upon the stage of pregnancy and patient preference. Anticoagulation should be considered in the setting of severely decreased LVEF during late pregnancy and 6 to 8 weeks postpartum.Warfarin crosses the placenta and is avoided during pregnancy as low-molecular-weight heparin does not cross the placenta and can be used during pregnancy,The novel anticoagulants have not been studied during pregnancy or lactation and are generally avoided. Anticoagulation is suggested by the European Society of Cardiology when the LVEF is <35%,whereas the American Heart Association suggests using LVEF ≤30% as the threshold. Beta-blockers and non-vasoselective calcium-channel blockers may safely be used for rate control of tachyarrhythmias. Beta-blockers should be initiated cautiously and gradually uptitrated to the maximum tolerated dose.B-1 selektive like (metoprolol) should be preferred. However, lower fetal birth weight has been documented and should be monitored. Hydralazine and nitrates can be used instead of ACE inhibitors and ARB for reduction afterload. Hydralazine and nitrates appear safe in pregnancy, although with less evidence for benefit than ACE inhibitors, and should only be used in the presence of hypertension, severe LV dysfunction, and/or evidence of congestion in decompensated HF. Dopamine can be used if inotropic drugs are neededThe 2018 European Society of Cardiology guidelines include a recommendation (Class IIb, Level of Evidence: B) for the use of bromocriptine. Due to the association with thrombotic complications, therapeutic anticoagulation is recommended in conjunction with bromocriptine. Bromocriptine is a dopamine agonist and inhibits the release of prolactin.It was originally marketed for lactation suppression, but due to the association with myocardial infarction stroke and seizures ,it is no longer approved for this indication. The patients have to be very aware for the implications of not breastfeeding due to bromocriptine and they should receive standard heart failure therapy with prophylactic anticoagulation, diuretic and vosorelaxing medicament. Relapse of PPCM has been observed after rapid tapering of HF therapies, and therefore treatment should continue for at least 6 months after full recovery of LV function followed by gradual tapering. In severe LV dysfunction during the 6-12 months following first presentation despite optimal medical therapy, implantation of an ICD and cardiac resynchronization therapy for patients with left bundle branch block and QRS >130ms are recommended. Cardiac transplantation is reserved for patients where mechanical circulatory support is not possible or desirable, or for

patients who do not recover after 6-12 months. Patients with PPCM have higher rates of graft failure and death after heart transplantation Despite successful pregnancies post-cardiac transplantation, data are limited .All immuno- suppressive medications enter the foetal circulation, thus the management of immunosuppression in the pregnant post-transplant recipient is highly specialized. On the other hand, as all immunosuppressive agents are excreted into breast milk with unknown long-term effects, the International Society for Heart and Lung Transplantation currently recommends against breastfeeding [49-70].

Labor and Delivery

The mode of delivery in patients with PPCM during pregnancy should be discussed with cardiologists, obstetrics, anesthetists and neonatologist that should work together for the treatment of such patients and to minimize the risk for mother and the fetus. Urgent delivery irrespective of gestation duration should be considered in women with advanced HF and haemodynamic instability despite treatment. Caesarean section is recommended with cen-tral neuraxial anaesthesia. To prevent abrupt pressure or volume changes, epidural anaesthesia might be the method of choice but should be carefully titrated, guided by an expert anaesthetic team. Unstable patients may benefit from invasive hemodynamic optimization prior to delivery and monitoring during delivery and the early postpartum period. Stable patients can delivered vaginally if there are also in good obstetric condition with spinal or epidural analgesia. In stable congestive HF, vaginal delivery is preferred with spinal/ epidural analgesia. After delivery, the fluid overload risk by increase venous returned must be management very carefully. Peripartum management depend on the clinical status of the patient and certainly myocardial function. During that time, heart failure therapy include the use of diuretics to reduce preload, vasodilators to increase cardiac output and stroke volume and decrease vascular resistance as well as beta-blockers Neurohormonal blockade with angiotensin-converting enzyme inhibition, angiotensin receptor blockers, and mineralocorticoid receptor blockers are considered as first-line heart failure medication. [71].

Lactation

Fortunately, most heart failure medications are compatible with breastfeeding. Both warfarin and low-molecular-weight heparin are considered safe with lactation. The novel anticoagulants have not been studied during lactation and are generally avoided. There is clear evidence that breastfeeding confers multiple benefits for infants and mothers but in cases with severe HF, breastfeeding is discouraged because reduce the high metabolic demand and adapted optimal treatment for mother However, a small study of patients enrolled online in the United States indicated that women who breastfed actually had higher rates of recovery [72]

Counseling and Treatment During Subsequent Pregnancy

PPCM has been described in primiparous and multiparous women. Risks of a subsequent pregnancy differ based on the preconception recovery status. There is higher risk especially in women with persistent LV dysfunction after the first pregnancy and when the EF has not recovered to >50-55%, subsequent pregnancy should be discouraged. Even with normalized EF, counselling is required due to high risk of irreversible deterioration in ventricular function, maternal and fetal mortality. With expert interdisciplinary management and immediate bromocriptine treatment post-delivery, successful subsequent pregnancies, especially in patients with recovered EF, have been reported It is crucial to monitoring and treatment women with a history of PPCM during a subsequent pregnancy. The prognosis of women with a history of PPCM can be improved by discussed decision-making between cardiologist, obstetric-gynecologist, the patient, and the patient's relatives to minimize the risk for mother and the fetus. In women with recovered LV function who are taking HF medications, ACE inhibitors or angiotensin receptor blockers, and aldosterone receptor antagonists should be discontinued prior to conception, and it may be prudent to ensure stability of LV function after at least 3 months off of these medications prior to considering the LV recovered. Exercise stress echocardiography showing adequate contractile reserve may help to identify women at an even lower risk of relapse. Althought only few of data supported the prophylactic use of beta-blockers during subsequent pregnancies in women with recovered LVEF may be considered The contraceptive should be addressed in high risk women but strogen-containing contraceptives should be avoided for possibility the risk of thromboembolism. Progestin-only methods including the hormonal IUD, the subdermal implant and progestin-only pills are not associated with increased risk of thromboembolism and are considered safe in these patient groups [73,74].

Conclusion

The diagnosis of PPCM should be suspected in any pregnant or postpartum woman with symptoms of Heart failure. Echocardiography is safe and should be performed in any suspected case of PPCM especially in those with elevated BNP level. Despite the pathophysiological mechanisms that explain the disease are quite clear now, advances in management of PPCM due to knowledge gained and the increase in the number of tests conducted and the diagnostic criteria for peripartum cardiomyopathy questions remain about vascular and hormonal changes of pregnancy. Addition of bromocriptine to standard therapy for heart failure immediately after delivery was safe and seemed to be associated with a better outcome .It is necessary long-term monitoring and follow-up but the optimal duration of medications following recovery is not quite clear now. However, there is high risk during pregnancy many women with PPCM can be successfully managed through pregnancy, labor and delivery with conservative medical measures designed to optimize intravascular volume, systemic loading conditions, blood pressure, and rhythm.

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