Peripartum cardiomyopathy (PPCM) is a rare, potentially lethal complication of pregnancy. It is defined as a cardiomyopathy of unknown cause that occurs in the last month of pregnancy through the fifth month after birth in women without preexisting heart disease. Diagnostic criteria include clinical evidence of left ventricular dysfunction confirmed by echocardiography examination. This serious complication of pregnancy and postpartum affects women and their families at a pivotal point in their lives. The course of the disease and its resolution are variable and uncertain, with potential impact on future childbearing and maternal life expectancy. Management of this complex disorder requires the coordinated effort of an interdisciplinary team. The purpose of this article is to review the pathophysiology, diagnosis, management, prognosis, and nursing care implications of caring for the woman with PPCM.

Peripartum cardiomyopathy is a rare and potentially lethal cardiac complication of pregnancy occurring in the final month of pregnancy through the first 5 months after birth. It is characterized by the development of congestive heart failure and left ventricular systolic dysfunction, in previously healthy women with no other identifiable cause for heart failure. The etiology of peripartum cardiomyopathy is not well understood. Potential causal mechanisms include infection, autoimmune disease, and abnormal response to the hemodynamic stresses of pregnancy. There is significant risk of recurrence in subsequent pregnancies. The purpose of this article is to review the pathophysiology, diagnosis, management, prognosis, and nursing implications of peripartum cardiomyopathy. **Key words:** congestive heart failure, maternal complications, peripartum cardiomyopathy, postpartum complications, pulmonary edema

**DIAGNOSTIC CRITERIA, INCIDENCE, AND RISK FACTORS**

The diagnosis of PPCM is based on the presence of 4 clinical criteria, including presentation between the last month of pregnancy and the 5th month postpartum, and clinical evidence of left ventricular function confirmed by echocardiographic examination. The specific criteria are outlined in Table 1. Strict adherence to these criteria is helpful in differentiating cases of PPCM from other types of cardiomyopathy. PPCM is a diagnosis of exclusion, as heart failure occurring earlier in pregnancy may reflect previously undiagnosed cardiac disease that is revealed by the normal hemodynamic and hormonal stresses of pregnancy. The reported incidence of PPCM varies in the literature. The reports are primarily from tertiary centers, which may reflect a referral bias. The most currently accepted incidence of PPCM is 1 per 3000 to 1 per 4000 live births, or between 1000 and 1300 women affected annually in the United States.

While PPCM is a relatively rare condition, mortality associated with it is considerable. The true mortality rate of PPCM is difficult to determine. Factors that contribute to an inability to accurately estimate mortality include the rare occurrence of PPCM, inconsistent diagnostic criteria, referral bias, and inclusion of other cardiomyopathies with specific underlying causes.
Table 1. Diagnostic criteria for peripartum cardiomyopathy

<table>
<thead>
<tr>
<th>All 4 of the following:</th>
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<tbody>
<tr>
<td>1. Heart failure within the last month of pregnancy through the fifth month after birth</td>
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<tr>
<td>2. Absence of prior heart disease</td>
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<tr>
<td>3. No determinable cause for heart failure</td>
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<td>4. Strict echocardiographic indication of left ventricular dysfunction:</td>
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<tr>
<td>- Ejection fraction of &lt;45%</td>
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<tr>
<td>- Fractional shortening &lt;30%</td>
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<tr>
<td>- End-diastolic dimension &gt;2.7 cm/m²</td>
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*Adapted from Pearson et al¹ and Hibbard et al.²

Published estimates range from 18% to 56%, gathered primarily from case series studied at tertiary referral centers.¹,³,⁵ Death usually results from congestive heart failure, arrhythmia, or thromboembolism.³

In a review of pregnancy-related mortality secondary to cardiomyopathy, Whitehead and colleagues attempted to identify contributing factors to the observed increase in reporting pregnancy-related deaths due to cardiomyopathy from 1979 (3.0%) through 1997 (7.7%).⁵ Reviewers compared pregnancy-related mortality rates for cardiomyopathy, which was further segmented as PPCM and cardiomyopathy due to other causes. Data from the Centers for Disease Control and Prevention’s Pregnancy Mortality Surveillance System from 1991 to 1997 were reviewed. Of the 245 deaths from cardiomyopathy during this time frame, 70% were attributed to PPCM, with 2% of deaths occurring prior to delivery, 48% within 42 days of delivery, and 50% between 43 days and 1 year postpartum.¹ The authors additionally noted that with the potentially long disease duration of PPCM, it could be reasonably concluded that significant numbers of deaths were not reported as pregnancy-related deaths. Based on the available data, this study concluded that PPCM accounted for 8% of all reported pregnancy-related deaths from 1991 to 1997.

ETIOLOGY

While the exact etiology of PPCM remains elusive, a number of clinical conditions have been implicated in the development of PPCM. These include myocarditis, abnormal immune response to the adaptations to pregnancy, viral infection, and maladaptive response to the hemodynamic stresses of pregnancy.¹,⁶–⁸ In addition, there are case reports associating PPCM with selenium deficiency, maternal cocaine use, and long-term oral tocolytic therapy.¹,⁷–¹⁰ A higher prevalence of PPCM has also been noted in women of advanced maternal age, multiparity, African American race, and in pregnancies complicated by multiple gestation, hypertension, preeclampsia, and/or prolonged tocolytic therapy.¹,²,⁵–¹¹ Whether race is an independent risk factor for PPCM or whether the interaction of race with hypertension contributes to the incidence of PPCM is currently unclear. Unfortunately, no screening tools to identify high-risk populations are currently available.¹

MYOCARDITIS AND VIRAL INFECTION

While the underlying mechanism of PPCM is unknown, there is a well-documented association between myocarditis and PPCM.¹,⁷,⁸,¹² However, the reported incidence of myocarditis in the setting of PPCM varies from 29% to 78%.¹,⁸ A diagnosis of myocarditis requires an endomyocardial biopsy. Variations in biopsy timing, histological criteria used in the processing of specimens, and the interval between disease presentation and biopsy performance have all contributed to difficulty drawing firm conclusions regarding the relationship between myocarditis and PPCM.

Felker and colleagues studied a population of 42 women with PPCM, all of whom had undergone an extensive cardiac evaluation (including echocardiography, endomyocardial biopsy, and right heart catheterization) over a median follow-up interval of 8.6 years.¹³ The premise of the study was to identify the prevalence of myocarditis and clinical variables associated with endpoint outcomes of death or cardiac transplantation. In this study, 62% of the study subjects had clinical histologic evidence of myocarditis or borderline myocarditis. In addition, 3 women died (7%) and 3 underwent cardiac transplantation (7%). The presence of myocarditis was not significant for any demonstrated difference in clinical outcomes of transplantation or morbidity.

In a recent study, Bultmann and colleagues performed molecular pathologic investigation of endomyocardial biopsy specimens from 26 patients diagnosed with PPCM.¹² Eight patient specimens (30.7%) demonstrated viral genomes from a wide variety of pathogens, including parvovirus B19, human herpes simplex virus 6, Epstein Barr virus, and human cytomegalovirus. However, the prevalence of these viruses was virtually identical in 33 women control subjects (30.3%) without evidence of myocardial tissue inflammation who had endomyocardial biopsy for the exclusion of myocarditis or medication-associated cardiac disease. The authors postulated that the depression of immune
function during pregnancy might contribute to triggering the reactivation of a latent virus that could then attack myocardial tissue and result in PPCM.\textsuperscript{12} The mild immunosuppression of normal pregnancy may permit viral replication and increase the risk of myocarditis in women with viral infection through an autoimmune response targeting cardiac tissue.\textsuperscript{1,7,8,12,15} Further investigation of this hypothesis may offer new insight regarding the role of immunosuppressive and/or antiviral treatment in PPCM.\textsuperscript{12,15}

**ABNORMAL IMMUNE RESPONSE TO PREGNANCY**

Another possible etiology for the development of PPCM is a maternal autoimmune reaction to exposure to fetal cells. Fetal cells can cross the placenta, entering maternal circulation during pregnancy, resulting in a genetic mutation (Chimerism).\textsuperscript{1,7,8,14} It is hypothesized that in the normal immunosuppressive state of pregnancy, these chimeric cells migrate into maternal cardiac tissue where they remain dormant. With the restoration of maternal immune competence during peripartum, these cells are recognized and targeted as a foreign agent and may initiate a pathologic immune response.\textsuperscript{1,7,8,14,15} Previous exposure to paternal histocompatibility complex antigens from sperm or previous pregnancies may be one factor in the production of an inflammatory response to the chimeric cells, with antigens to the chimeric cells now targeting healthy cardiac tissue. This could result in the release of cytokines, causing nonspecific myocyte toxicity and myocarditis. The elevated titers of autoantibodies against select cardiac tissue proteins in women with PPCM support the hypothesis of abnormal immunologic response as a possible cause of PPCM.\textsuperscript{15,16}

Other proposed etiologies for PPCM that have been recommended for further study include abnormalities of relaxin, an ovarian hormone that may be involved in the excessive relaxation of the connective tissue skeleton of the heart,\textsuperscript{1,7,14} and selenium deficiency, which increases the susceptibility of myocardial tissue to injury from viral infection, hypertension, or hypocalcemia.\textsuperscript{1,14} Tocolytic therapy has been associated with PPCM, but the nature of this association is unclear and may be related to the increased incidence of preterm labor in this population.\textsuperscript{8,9}

**DIAGNOSIS AND MANAGEMENT OF PPCM**

The diagnosis of PPCM is challenging because many of the symptoms are consistent with normal physiologic changes of pregnancy and postpartum. Initially, women usually present with symptoms of exertional dyspnea, cough, fatigue, and pedal edema, all of which are classic symptoms of congestive heart failure and are also seen frequently in uncomplicated pregnancies. As a result, it is not uncommon for PPCM to go unrecognized until disease is advanced. Advanced presentation includes signs and symptoms consistent with pulmonary edema, including tachycardia, paroxysmal nocturnal dyspnea, chest pain, neck vein distension, new murmur, pulmonary crackles, and occasionally hemoptysis.\textsuperscript{1,3,8} Evaluation for these patients may also include ruling out pulmonary embolism, previously undiagnosed rheumatic or congenital heart disease, myocardial infarction, complications of preeclampsia, tocolytic use, and sepsis.\textsuperscript{1,3,7,8,17,18}

The diagnosis of PPCM involves ruling out other forms of cardiomyopathy and the conditions noted above. Electrocardiogram evaluation may be normal or show nonspecific ST-T changes, left ventricular hypertrophy, and atrial or ventricular arrhythmias. Chest X-ray findings commonly reflect cardiomegaly with left ventricular hypertrophy, pulmonary edema, and pleural effusions.\textsuperscript{7} Evaluation of left ventricular function by echocardiography (Table 1) is critical to accurate diagnosis of PPCM. In women presenting with evidence of cardiogenic shock, assessment of cardiac function and anatomy by invasive cardiac catheterization can help guide pharmacological therapy.\textsuperscript{1,8} Endomyocardial biopsy remains controversial and is not standard practice at this time.\textsuperscript{8}

Therapy for PPCM is individualized on the basis of the severity and timing of clinical presentation. The treatment of PPCM is modeled on other forms of congestive heart failure, but patients benefit from a multidisciplinary team approach, including the obstetrician, maternal-fetal medicine specialist, neonatalogist, cardiologist, and experienced perinatal and cardiac nurses and clinical nurse specialists.\textsuperscript{14,19} Women with PPCM may require admission to an adult critical care unit. It is essential that the care team remain aware of normal physiologic changes of pregnancy and postpartum (such as increased baseline cardiac output) that affect the treatment goals in this population. Collaborative medical and nursing management by a team of perinatal and intensive care providers, with integration of these highly specialized disciplines, results in the best outcomes for these patients.

Management goals typically include preload reduction, afterload reduction, and maintenance of cardiac contractility, as well as psychosocial support for the family in crisis. Table 2 presents a focused review of the major classes of medications used for the treatment
Table 2. Medications used in peripartum cardiomyopathy

<table>
<thead>
<tr>
<th>Class of medication</th>
<th>Treatment goal</th>
<th>Generic name (trade name)</th>
<th>Nursing implications</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diuretics</td>
<td>Preload reduction</td>
<td>Furosemide (Lasix)</td>
<td>Monitor electrolyte levels</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Monitor renal function</td>
</tr>
<tr>
<td>Vasodilators</td>
<td>Afterload reduction</td>
<td>Hydralazine (Apresoline)</td>
<td>Frequent small doses to avoid maternal hypotension</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Nitroglycerine</td>
<td>Avoid prolonged use; potential for fetal cyanide toxicity</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Nitroprusside</td>
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</tr>
<tr>
<td></td>
<td></td>
<td>Neseritide</td>
<td>Observe for hypotension and tachycardia</td>
</tr>
<tr>
<td>Angiotensin-converting enzyme inhibitors</td>
<td>Afterload reduction</td>
<td>Captopril</td>
<td>Contraindicated in pregnancy because of teratogenicity</td>
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<tr>
<td></td>
<td></td>
<td>Enalapril</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>Lisinopril</td>
<td></td>
</tr>
<tr>
<td>Beta blockers</td>
<td>Afterload reduction</td>
<td>Carvedilol</td>
<td>Monitor for cough, angioedema, hyperkalemia, and decreased renal function</td>
</tr>
<tr>
<td>Inotropic agents</td>
<td>Improve cardiac contractility</td>
<td>Digoxin (Lanoxin)</td>
<td>Monitor for hypotension, heart rate</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Dopamine</td>
<td>Monitor for worsening heart failure</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Dobutamine (Dobutrex)</td>
<td>Increased doses required in pregnancy because of increased renal clearance. In postpartum, monitor pulse to avoid toxicity.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Milrinone</td>
<td></td>
</tr>
</tbody>
</table>

*Data Adapted from Ardehali et al,7 Murali and Baldisseri,8 and Arafeh and Baird.19

of PPCM, including the therapeutic goal and nursing implications.

Diuretics such as lasix are used to reduce preload and provide relief from pulmonary congestion. Diuretics work to reduce the preload in both the ventricles and decrease cardiac wall tension, improving both cardiac and pulmonary function.7 In patients who continue to have congestion in spite of diuretic therapy, the addition of vasodilators, such as nitroglycerin, nitroprusside, or neseritide, may be indicated.8 Afterload reduction is achieved with the use of vasodilator agents such as hydralazine and/or nitrates during pregnancy and angiotensin-converting enzyme inhibitors (ACE inhibitors) in the postpartum period. ACE inhibitors (captopril, enalapril, or lisinopril) are contraindicated during pregnancy because of teratogenicity but are recommended treatment for the postpartum period.1,3,7,8 These medications work by preventing progressive ventricular remodeling, resulting in improvements in exercise tolerance, ejection fraction, and quality of life.8 Multiple studies of the use of ACE inhibitors in patients with heart failure (ranging from asymptomatic to advanced heart failure with severe left ventricular dysfunction) have demonstrated significant reductions in mortality.7,8 Their safety for use during lactation remains unclear.7

Inotropic support in patients with low perfusion improves myometrial contractility and can assist in rate control when needed. Digoxin is the first line of treatment. Higher doses are required to achieve therapeutic levels in pregnancy because of increased maternal intravascular volume and accelerated renal clearance. Dobutamine, dopamine, and milrinone are reserved for the treatment of women in cardiogenic shock (low cardiac output, elevated pulmonary capillary wedge pressure).8,19 In patients who remain in acute shock after aggressive medical therapy, intraaortic balloon pump support is helpful in the short term. Long-term balloon pump therapy (defined as more than 3 days) places the patient at risk of line sepsis and limb ischemia.8

β-Adrenergic blockers (carvedilol and metoprolol) are recommended to reduce symptoms, to improve ejection fraction, and to prevent progressive ventricular remodeling in stable patients with PPCM who are not receiving inotropes or mechanical support.7,8 Meta-analyses of studies of β-adrenergic blockers in patients with congestive heart failure demonstrated
significant reductions in both mortality (30%) and re-
hospitalization (40%).\textsuperscript{7} Approximately 15% of patients are intolerant of these medications, requiring careful monitoring of blood pressure for hypotension, heart rate, and symptoms of recurrent heart failure.\textsuperscript{7,8}

Women with PPCM who demonstrate depressed left ventricular function (ejection fraction < 35%) are at increased risk for thromboembolism because of the combined effects of stasis in the ventricles and the procoagulant effects of elevated cytokine levels. These women are candidates for anticoagulation therapy.\textsuperscript{1,7,8} Heparin is the anticoagulant of choice during pregnancy, and warfarin is the medication of choice in the postpartum period.

Patients with PPCM who fail to respond to aggressive medication therapy have been treated with defibrillator implantation to prevent sudden cardiac arrest, and with a ventricular assist device as a “bridge” to cardiac transplantation.\textsuperscript{20,21} Patients with PPCM who remain in persistent New York Heart Association class IV, which is unresponsive to all medical treatment, may be candidates for cardiac transplantation.\textsuperscript{1,7,8} Table 3 reviews the New York Heart Association classification system. Women with PPCM who undergo transplantation appear to have similar survival to patients with other forms of cardiomyopathy, with reported survival rates of 88% at 2 years and 78% at 5 years.\textsuperscript{22} However, PPCM patients require higher doses of cytolytic (antirejection medication) therapy to prevent early rejection.\textsuperscript{1,8}

Follow-up for patients with PPCM is consistent with patients with other forms of cardiac disease. Repeat assessment of cardiac function by echocardiography is recommended at 3- and 6-month intervals postdiagnosis to evaluate functional status. Subsequent serial evaluation is recommended at least annually or when symptoms present.\textsuperscript{8} Patients with ongoing compromise of left ventricular function must be encouraged to continue pharmacological therapy for management of chronic heart failure.\textsuperscript{8}

**PROGNOSIS**

Long-term prognosis for women with PPCM is dependent on recovery of left ventricular function and size in the immediate 6 months after birth. Historically, left ventricular ejection fraction returns to normal within the first 6 months after birth in more than 50% of patients with PPCM. However, in patients with ongoing left ventricular dysfunction mortality rates approach 85% over 5 years.\textsuperscript{1,3,8}

Data regarding association of type of cardiomyopathy, morbidity, and long-term survival for women experiencing PPCM are limited. Felker and colleagues examined the outcomes of 1230 patients with both PPCM and cardiomyopathy of all other types. Over a mean follow-up interval of 4.4 years, patients with PPCM had significantly improved survival rates as compared with patients with idiopathic cardiomyopathy.\textsuperscript{23} Patients with PPCM had a 94% survival rate at 5 years. Survival was significantly worse in patients who had cardiomyopathy secondary to infiltrative cardiac disease, HIV infection, doxorubicin therapy, and ischemic heart disease.

Elkayam and colleagues studied 123 women with PPCM. Four percent of the patients required cardiac transplantation, and there was a 9% reported mortality rate.\textsuperscript{24} This variation in reported rates of morbidity and mortality may reflect differences in medical management of cardiac failure and aggressive use of technology, such as implantable defibrillators for prevention of sudden cardiac death in this population.\textsuperscript{8,23}

The effort to predict indicators of recovery and potential for reoccurrence in future pregnancies has generated research to identify effective prognostic testing. While 30% to 50% of patients are reported to recover baseline ventricular function within 6 months of birth, the response of the ventricles to future hemodynamic stress is unpredictable. Lampert and colleagues compared key hemodynamic parameters between postpartum women with a history of PPCM and matched nonpregnant control subjects.\textsuperscript{25} Both groups demonstrated normal baseline heart rates, blood pressures, ventricular dimensions, and left ventricular function when undergoing a dobutamine challenge test. However, women with previous PPCM demonstrated decreased contractile reserve, which may explain the reoccurrence of clinical onset of heart failure in subsequent pregnancy. The study concluded that the ventricles of these women may respond with reoccurrence

| Table 3. New York Heart Association classification* |
|-----------------|-------------------------------------------------|
| Grade 1         | *No limitations of physical exercise; ordinary activity does not cause undue fatigue, palpitations, dyspnea, or angina* |
| Grade 2         | *Slight limitations of physical exercise; ordinary activity results in fatigue, palpitations, dyspnea, or angina* |
| Grade 3         | *Marked limitations of physical activity; less than ordinary activity causes symptoms* |
| Grade 4         | *Complete inability to carry on physical activity without symptoms* |

*Adapted from Arafeh and Baird.\textsuperscript{19}
of left ventricular dysfunction when undergoing the hemodynamic stresses of a new pregnancy and recommended serial noninvasive testing of cardiac function in women who did undertake future pregnancy.25

Dorbala and colleagues tested whether or not dobutamine stress echocardiography could predict left ventricular function during recovery and follow-up.26 Because of the high potential for morbidity in the first 3 months postpartum in these women, early risk stratification and assessment of prognosis are critical to identification of patients who may need cardiac transplantation. Identification of an effective prognostic test is the first step in efficient allocation of resources and targeted use of aggressive intervention. Six women who met the diagnostic criteria for PPCM underwent testing at initial presentation and follow-up, with a median interval of 133 days between testing. Left ventricular ejection fraction (LVEF) and maximal inotropic contractile reserve were compared at baseline and at follow-up. Four of these women were subsequently followed for a mean of 2.6 years. None of the women in long-term follow-up experienced further compromise of their cardiac function during the study period. Two of the women had a subsequent pregnancy without deterioration in their LVEF. Although the study size was limited, there was correlation between left ventricular function at maximal contractile reserve and follow-up LVEF. The authors noted that the exclusion of women who were too ill for enrollment may have resulted in selection bias that contributed to the positive outcomes. On the basis of the results of this initial study, they recommended a larger multicenter trial.

Chapa and colleagues studied 32 women to determine whether echocardiography findings at the time of diagnosis are predictive of persistent cardiac dysfunction.27 In this cohort of patients, 13 (41%) had recovery of left ventricular function, with 19 (59%) demonstrating persistent left ventricular dysfunction. In this study, the women with persistent cardiac disease were noted to have higher left ventricular end diastolic dimension and lower fractional shortening at diagnosis than those who recovered. A fractional shortening value less than 20% and a left ventricular end diastolic dimension of 6 cm or greater at the time of diagnosis conferred a 3-fold higher risk for persistent left ventricular dysfunction. No significant differences were noted in demographic characteristics between women who recovered and those who had ongoing cardiac dysfunction. Of the patients with ongoing disease, 2 patients (6.5%) received cardiac transplantation and 3 patients (9.6%) died from heart failure. The authors suggested that echocardiography in addition to being a valuable diagnostic tool could also be used to predict prognosis for recovery and for follow-up in persistent disease.

Predicting the maternal risks of subsequent pregnancy in patients with a history of PPCM remains difficult. The rarity of PPCM, small numbers of patients in reports, and confounding variables such as selection and referral bias all contribute to contradictory results. The National Heart, Lung, and Blood Institute and the Office of Rare Diseases workgroup were not able to come to consensus regarding recommendations for future pregnancy after PPCM.1

Chapa and colleagues examined the outcomes of subsequent pregnancy in women with a history PPCM in a previous pregnancy.27 Four of the patients with recovered cardiac function had 1 additional pregnancy. Two patients with persistent left ventricular dysfunction had 1 and 3 subsequent pregnancies, respectively. All 4 of the patients with recovered cardiac function had recurrent disease in the third trimester. After birth, 1 patient again demonstrated full recovery, while the other 3 women had persistent cardiac disease after their subsequent pregnancy. In the 2 women entering pregnancy with persistent cardiac disease, there was no recurrence or worsening of cardiac function during or after their pregnancies.

ELkayam and colleagues examined maternal and fetal outcomes of subsequent pregnancies in women with previous PPCM through a survey of members of the American College of Cardiology.28 In the 44 women identified to have PPCM, 28 subsequent pregnancies occurred in women with a return to normal left ventricular function (group 1) and 16 occurred in women with persistent left ventricular dysfunction (group 2). Symptoms of heart failure presented in 21% of patients from group 1 and in 44% of patients from group 2. The mortality rate was 0% in group 1 and 19% in group 2. Two women died suddenly (one 2 months after birth and one 2 years after birth), and 1 woman died of progressive heart failure 2 months after her pregnancy. Subsequent pregnancy in women with previous PPCM was associated with a significant decrease in left ventricular function and could result in deterioration of cardiac function and death.

In a small study of 6 subjects in South Africa, Silwa and colleagues found that subsequent pregnancy resulted in a more than 10% reduction in ejection fraction in 5 women at 1 month postpartum.29 Two women died within 3 months despite maximal medical therapy. All of these women were staged at New York Heart Association functional class 1 at the onset of pregnancy, were asymptomatic until birth, and had increased levels of an inflammatory cytokine in the postpartum period.
In summary, the clinical research on PPCM is hampered by the rarity of the condition. Even tertiary referral and teaching centers do not see significant numbers of women with PPCM to be able to develop concrete recommendations for treatment. An international registry to facilitate development of reliable data regarding prevalence, risk factors, prognostic variables, and a serum and tissue bank has been suggested to assist in the definitive identification of etiology of PPCM and to identify evidence-based treatment interventions.\textsuperscript{1}

Although Pearson and colleagues could not make definitive recommendations regarding future pregnancy in women with a history of PPCM, it seems clear that regardless of recovery of left ventricular function, there is risk of recurrence of PPCM in subsequent pregnancy.\textsuperscript{27–30} Women with recovered cardiac function and women with persistent cardiac disease may still experience deterioration of cardiac function in subsequent pregnancy. The risk of mortality is higher in women with persistent cardiac dysfunction. Current knowledge regarding PPCM suggests that strong consideration be given to avoidance of additional pregnancies.\textsuperscript{30} In women who wish to proceed with subsequent pregnancy, dobutamine stress echocardiography testing offers quantitative assessment of contractile reserve and may assist predicting a woman’s specific risk of deterioration of cardiac function with subsequent pregnancy.\textsuperscript{8,25} Ultimately, however, decisions regarding future pregnancy are made by the woman and her family. An extensive preconceptual consultation from both obstetric and cardiology specialists, with full discussion regarding the potential maternal and fetal risks, is critical to making an informed decision.\textsuperscript{19} Women who choose to proceed with subsequent pregnancy should be strongly encouraged to undergo preconceptual assessment and optimization of cardiac treatment and be closely followed by both obstetric and cardiology specialists during pregnancy.\textsuperscript{8,19}

**NURSING IMPLICATIONS**

Women with PPCM experience a life-threatening illness at a pivotal time in their lives. Birth of a child is a profound experience that is usually both a joyous and stressful time for all involved, as fundamental relationships are changed by the addition of a new baby. Attention to both physiologic and emotional needs will facilitate the successful birth of the new family.

Management of this complex disease requires the interaction of specialized, interdisciplinary teams, including medical and nursing personnel from obstetrics, cardiology, and critical care. During pregnancy and the immediate postpartum period, maternal cardiac physiology is altered significantly with increases in heart rate, stroke volume, cardiac output, and blood volume and decreases in peripheral vascular resistance, pulmonary vascular resistance, and colloid oncotic pressure.\textsuperscript{31} Hemodynamic parameters from invasive cardiac monitoring are altered in pregnancy and postpartum, with rapid shifts in volume in the first 24 to 48 hours.\textsuperscript{19,32} For an in-depth review of physiologic changes during labor and delivery and their impact on the cardiovascular system, the reader is directed to Arafeh and Baird’s recent review of cardiac disease in pregnancy.\textsuperscript{19} Women with acute PPCM are frequently managed in the adult critical care unit, and the adult critical care team may not be thoroughly familiar with these pregnancy-related changes. It is critical to ensure that physiologic targets for cardiac and respiratory function are appropriate to the pregnant and postpartum woman, as standard adult targets will not be adequate to meet physiologic needs. Although the woman with PPCM is critically ill, meeting her need to be with her family, other children, and infant will reduce anxiety and provide comfort. These needs can be met by facilitating visitation, providing photos, and assisting with preparation/continuance of lactation.\textsuperscript{19,31}

Postpartum women need additional counseling and support regarding balancing the demands of caring for an infant and their own health. Women with PPCM have all the normal adjustments and demands of the postpartum period, which are further complicated by an uncertain recovery from a medically complex pregnancy. The importance of avoiding stress, anxiety, and getting adequate rest are goals for all new mothers but are imperative for women with PPCM. Avoidance of extreme physical exertion and frequent rest periods are critical to recovery. Thorough assessment of each woman’s living situation can assist in the development of an individualized care plan to support these women to rest. Table 4 reviews a structured review of cardiac symptoms that can assist in developing this plan. Inclusion of the staff nurse, advanced practice nurse from all involved disciplines, social services, and family during this discussion offers the opportunity for questions, education, support, and interdisciplinary planning. Concrete suggestions such as requesting visitors to bring meals, assist with cleaning, laundry, care of other children, planning where to spend most of the day with the baby, and avoidance of stairs are all strategies that can facilitate successful transition to life at home. Social services referral for help with household activities and coordination of other services can assist with activities of daily living as needed.
Women need instruction regarding signs and symptoms of congestive heart failure, including unusual weight gain, peripheral/dependent edema, exertional dyspnea, cough, pallor, syncope, chest pain, and cardiac arrhythmias. Encourage prompt reporting of symptoms of cardiac dysfunction to the appropriate care provider. Good nutrition, a low-sodium diet to prevent fluid retention, and high-iron foods to prevent anemia are cornerstones of management of cardiac disease.

Women with documented history of systemic or pulmonary embolism or atrial fibrillation are at risk for thromboembolic disease secondary to decreased venous return and ventricular remodeling. These women will require ongoing anticoagulation therapy. Patient education regarding signs and symptoms of thrombosis, including unusual warmth or redness in extremities, increasing leg pain, sudden severe chest pain, increased dyspnea, tachypnea, and tachycardia, and the need to seek prompt medical attention are key. When the patient is receiving anticoagulant therapy, information regarding signs and symptoms of abnormal bleeding, such as hematuria, bleeding gums, excessive bruising, or petechiae, should also be reviewed.

Pharmacological therapy is a cornerstone of management for cardiac disease. Adherence to the medication regimen requires extensive education regarding specific medications, their mechanism of action, and their side effects, as well as exploration of any concerns regarding medication use, access, and cost. For women with residual disease, commitment to lifelong medication therapy for chronic heart failure is necessary. Because PPCM is a dynamic disease process with potential for recovery, frequent adjustments of medication regimens are to be expected. Ongoing follow-up with a cardiologist is necessary for optimal function and recovery. Women who do experience recovery of left ventricular function and maintain recovery for 1 year may have their medication gradually withdrawn with ongoing monitoring of cardiac function by echocardiography.

CONCLUSIONS

PPCM is a rare, potentially lethal complication of pregnancy that presents in the last month of pregnancy through the fifth month postpartum and is characterized by the onset of acute left ventricular heart failure. Usually, more than 50% of women will regain normal cardiac function, but in those who continue to experience cardiac compromise mortality rates approach 85% over 5 years. Women with PPCM represent a unique challenge for care, requiring the services of coordinated care of obstetric and cardiac professionals, in addition to the interdisciplinary team. Nursing care should focus on assessment of physical and emotional parameters, anticipation and detection of problems, focused interventions, and prevention of complications. Optimal outcomes are best achieved by a comprehensive approach from a multidisciplinary team that includes clear communication and an individualized care plan that meets the needs of the woman and her family.

REFERENCES