EPIDEMIOLOGY

The actual incidence of PPCM is not known. PPCM constitutes less than one per cent of all cardiovascular events related to pregnancy. Incidences of PPCM in the United States are 0.03-0.06% of pregnancy. However, they are more common in Africa, 1:300 pregnancies. This may be due to the consumption of kanwa, a tradition, for 40 post-partum days. Kanwa is a dry salt and causes hypervolemia and hypertension. Ninety percent of PPCM occurs within two months of delivery.

RISK FACTORS

The following risk factors are associated with increased risk of PPCM:
Age over 30 years, pregnancy with multiple fetus, African descent, maternal cocaine abuse, long term tocolytic therapy and familial. Selenium deficiency leading to PPCM is controversial.

ETIOLOGY

The exact etiology of PPCM is still not known, but the following hypothesis had been proposed:
(i) Familial; Familial clustering of PPCM is well known, it could be due to genetic or environmental factors.
(ii) Myocarditis; Melvin proposed myocarditis as a cause for PPCM. Myocarditis could be viral or autoimmune, as with pregnancy there is increase susceptibility to both.
study endomyocardial biopsies in five patients showed features of myocarditis.

(iii) Abnormal immune response; the fetal cell enters maternal circulation and remains in circulation without rejection due to weak immunogenic paternal halotype of chorionic cell. If these cells lodge into the cardiac tissue it triggers an immune response. Raised titers of immunoglobulins and other autoantibodies in patients with PPCM are suggestive of abnormal immune response. However, contrast to it, Cenac et al. found no significant difference in levels of immunoglobulins and other autoantibodies in PPCM and control group of patients (12).

(iv) Maladaptation to stress of pregnancy; hyperdynamic circulation during pregnancy causes remodeling and transient hypertrophy of left ventricle, the exaggerated reduction in left ventricular systolic function with stress of gestational hypertension may contribute to heart failure in PPCM patients. Raised titers of immunoglobulins and other autoantibodies in patients with PPCM are suggestive of abnormal immune response. However, contrast to it, Cenac et al. found no significant difference in levels of immunoglobulins and other autoantibodies in PPCM and control group of patients (12).

DIFFERENTIAL DIAGNOSIS

PPCM should be differentiated from other forms of cardiomyopathy, heart failure, pulmonary thromboembolism, severe eclampsia and pneumonia. From history, physical examination and investigations one must exclude, myocardial infarction, idiopathic dilated cardiomyopathy and valvular heart disease.

MEDICAL MANAGEMENT

Management of PPCM is similar to other types of heart failure, apart from concerning the adverse effect of treatment on fetus or breast-feeding infant. The aim of therapy in PPCM is to reduce preload, after load and increase the cardiac contractility. Heart failure during pregnancy can be acute or acute on chronic. Pregnant patients with known cardiovascular disease can present in stable condition in early stages of pregnancy; their management is mainly adjustment of their cardiac medications and regular monitoring for cardiac decomposition, an initial detailed cardiovascular and careful physical examination should be done, New York heart association functional status should be documented, ECG and echocardiogram should be performed. Patients presenting in decompensate cardiac status during pregnancy or in the peripartum period, may be known to have cardiac disease or it may be acquired during the pregnancy such as peripartum cardiomyopathy. Management of these patients includes detail history and physical examination, evaluation of severity of decomposition. An ECG may reveal deteriorating left ventricular functions, arrhythmia, LVH or arterial abnormality. The aims of therapy in these patients include optimizing hemodynamics, reducting after load, optimizing preload and cardiac contractility. These can be achieved by treatment of pulmonary congestion, control of hyper/hypotension, treatment of cardiac arrhythmia and prevention of thromboembolic events. Digoxin is safe to use in pregnancy. Diuretics can be used if salt restriction is not sufficient. Beta-blocker improves left ventricular functions in patients of PPCM, but ACE inhibitors are the drug of choice in postpartum PPCM.

Ventricular arrhythmias should be treated aggressively in cases of PPCM. Class III antiarrhythmic medication is the best option.
Intravenous medications are needed in PPCM patients admitted to the intensive care therapy unit. Therapy with ionotropes such as dobutamine, adrenaline and milrinone, should be directed by invasive cardiac monitoring. While interpreting the invasive hemodynamic monitoring, one should take into account of normal changes that occurs during pregnancy. Before delivery, unfraction or low molecular weight heparin is the choice, while in postpartum period warfarin is used. Cardiothoracic medication, effects on fetus and their secretion in the breast milk are shown in detail in Table 1. Occasionally, when medical therapy fails in patients with PPCM, need for mechanical cardiovascular support (intra-aortic balloon pump, ventricular assisting device) or even cardiac transplant has been reported in the literature.

There is no indication to terminate pregnancy as a form of therapy. If concurrent pre-eclampsia, maternal hemodynamic are inadequate to support fetus or therapy to support mother puts fetus at risk, early delivery of near term fetus is strongly recommended.

**COMPLICATIONS**

The most common complication is thromboembolism. A premature delivery rate of 25% has been reported in cases with PPCM. PPCM cases had increased incidence of cesarean section up to 40%.

**PROGNOSIS**

Maternal outcome; the mortality in mother who develops PPCM is 15-50%, various factors such as, black women, multiparity, LVEF less than 30% are indicators of worst maternal outcome. Neonatal outcome; preterm birth occurs in up to 25% of patients, a few intra-uterine fetal deaths and 40% of patients with PPCM under go cesarean section for obstetric reasons.

Predictors of persistent left ventricular dysfunction are, LVEF less than or equal to 30%, fractional shortening less than or equal to 20% and left ventricular end diastolic dimension greater than or equal to six cm.

**PREVENTION**

In patients who have recovered from left ventricular failure due to PPCM there is a high risk of PPCM in subsequent pregnancy and so the best way to avoid PPCM is avoid subsequent pregnancy.

<table>
<thead>
<tr>
<th>Medication</th>
<th>Potential side-effects</th>
<th>Safety in pregnancy</th>
<th>Safety during breast feeding</th>
</tr>
</thead>
<tbody>
<tr>
<td>ACE inhibitors</td>
<td>Embroyopathy</td>
<td>Not</td>
<td>Yes</td>
</tr>
<tr>
<td>AR blocker</td>
<td>Embroyopathy</td>
<td>Not</td>
<td>Yes</td>
</tr>
<tr>
<td>B-blockers</td>
<td>Fetal bradycardial/IUGR</td>
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<td>Yes</td>
</tr>
<tr>
<td>Hydralizin</td>
<td>None</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Digoxin</td>
<td>Low birth weight</td>
<td>Yes</td>
<td>Yes</td>
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<tr>
<td>Diuretics</td>
<td>Reduction in uteroplacental perfusion</td>
<td>Unclear</td>
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<tr>
<td>Nitrates</td>
<td>Fetal distress with maternal hypotension</td>
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<td>No data</td>
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<tr>
<td>Lidocain</td>
<td>Fetal CNS depression</td>
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<tr>
<td>Procainamide</td>
<td>Maternal osteoporosis</td>
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<tr>
<td>LMWH</td>
<td>Hemorrhage</td>
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<td>Yes</td>
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<tr>
<td>Heparin</td>
<td>Hemorrhage/ maternal osteoporosis/thrombocytopenia</td>
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<tr>
<td>Warfarin</td>
<td>Warfarin embryopathy</td>
<td>Yes after 12 weeks</td>
<td>Yes</td>
</tr>
<tr>
<td>Adenosine</td>
<td>Non reported</td>
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<td>No data</td>
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</tbody>
</table>


**CONCLUSION**

Peripartum cardiomyopathy (PPCM) is a rare pregnancy-induced dilated cardiomyopathy, more common in the postpartum period. The exact etiology of PPCM is not known. Various hypothesis have been proposed. The incidence of PPCM varies with geographical variation. Risk factors for PPCM are elder pregnancy, multiparity, African descent, pregnancy with multiple fetus, maternal cocaine abuse and longer oral tocolytic therapy.

Clinical presentations are dyspnea, palpitations, rarely present as thromboembolic phenomenon. Echocardiography is a must for the diagnosis of PPCM as it will show reduction in ejection fraction and rule out other cardiac lesions.

Management of PPCM includes optimizing hemodynamics, reduction in after load, optimizing preload and cardiac contractility. These can be achieved by treatment of pulmonary congestion, control of hyper/hypotension, treatment of cardiac arrytheamia and prevention of thromboembolic events. The thromboembolic phenomenon is the most feared complication of PPCM. The reported mortality of PPCM is 15-50%. The best prevention of PPCM is to avoid subsequent pregnancies.

**REFERENCES**


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