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Review Article

Peripartum Cardiomyopathy: A review

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Introduction

Peripartum cardiomyopathy (PPCM) is an infrequent but critical disorder in which a destabilized heart is diagnosed within the last months of pregnancy or early puerperium and often complicating obstetrics as well as anesthetic management. Setting aside several historical expressions[1-5] "peripartum cardiomyopathy" is now the term widely used to describe this clinical situation[1-3, 6,7.] The strict time limit used in their diagnostic criteria was intended to exclude congenital and acquired causes of heart failure that usually manifest by the second trimester[1]. Even so, the occurrence of PPCM has been overestimated[6]. As a result, the PPCM workshop committee recommended the inclusion of echocardiographic features of the left ventricular dysfunction to redefine PPCM which are included in the two tables to follow.

Incidence

The incidence of PPCM was one case per 1374 live births in an Indian study [8]. PPCM occurs in 1 in 3,000 to 1 in 4,000 pregnancies in the United States[9]. In South Africa, the reported incidence is higher (1: 1,000 live births)[10]. A much higher incidence of 1:300 live births has been reported from Haiti[11] and an extremely high rate of 1% has been described in Nigeria[12]. Higher rates in developing countries may be due to variations in local cultural as well as puerperal practices, ecological factors, environmental influence, diagnostic criteria and reporting pattern used [13]. Diagnosis based only on clinical features has also overestimated the incidence [1].

Etiology

Cardiomyopathy occurs when there is damage to the heart. As a result, the heart muscle becomes weak and cannot pump blood efficiently. Decreased heart function affects the lungs, liver, and other body systems. The etiology of this disease remains uncertain, current evidence suggests myocarditis of viral, autoimmune, or idiopathic origin to be important as underlying factors [15]. Though the etiology remains unclear myocarditis was present in 78% of those with this condition [16].

Major risk factors for the development of PPCM were multiparity and advanced maternal age[8]. It may occur in childbearing women of any age, but it is most common after the age of 30. Risk factors also include obesity, having a history of cardiac disorders such as myocarditis, use of certain drugs, smoking, alcoholism, multiple pregnancies, being African American, and being lean. Twin pregnancy appears to cause a higher risk of developing PPCM [1].

Typical etiological nature points towards hypertensive heart failure caused by fluid overload rather than a true variety of PPCM. Preeclampsia and hypertension have been associated with a

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significant number of PPCM cases[1]. In contrast with the past, recent reports showing an association of hypertension and preeclampsia with PPCM are less frequent [11].

Recent advances in understanding the pathophysiology of PPCM assign a key role to unbalanced oxidative stress and the generation of a cardiotoxic prolactin subfragment. Recent studies have indicated that increased proteolytic cathepsin D activity in cardiomyocytes results in16kDa prolactin fragments with anti-angiogenic and apoptotic properties, which may contribute to the development of this disease [17]. In this regard, pharmacological blockade of prolactin holds the promise of novel, more disease specific therapy options[18]. Also, recent evidence supports inflammation, viral infection and autoimmunity as the leading causative hypotheses[19] as described in the preceding paragraphs.

The actual etiology of PPCM is unknown. Several hypotheses like myocarditis, viral infection, autoimmune factors, inflammatory cytokines and abnormal hemodynamic response to physiological changes in pregnancy, prolonged tocolysis and selenium deficiency have been postulated. Cenac et al[20] found significantly low selenium concentrations in PPCM patients which might be a mere incidental association rather than a cause.

Pathological features

Heart specimens appear pale, soft, dilated and heavier in PPCM[21]. Mural thrombi are invariably seen in one or more cardiac chambers in patients with persistent ventricular dysfunction. Gray-white patches of endocardial thickening are often seen at the sites of mural thrombi [21]. Cardiac valves and coronary vessels appear normal with the occasional presence of pericardial effusion[21]. Histological evidence of hypertrophy, degeneration, fibrosis, interstitial edema, fatty and mononuclear cell infiltration is seen in the myocardium with a sparse to abundant collection of eosinophils[22]. Electron microscopy has revealed varying degrees of enlargement, destruction or fragmentation of myofibrils, an increase in size and number of mitochondria, glycogen and some abnormal pertinacious material deposits[22]. Histochemical pictures of myocardial cells indicate occasional sarcoplasmic fat vacuoles containing triglycerides without any increase of lipofuscin or amyloid[13]. Significantly low levels of plasma albumin, prealbumin, selenium and zinc have also been reported [23].

Diagnosis

PPCM is a form of dilated cardiomyopathy in which no other cause of heart dysfunction (weakened heart) can be identified. This diagnosis should be limited to previously healthy women who present with congestive heart failure (CHF) and decreased left ventricular systolic function in the last month of pregnancy or within six months after delivery [24].

Common symptoms include fatigue, palpitations, nocturia, orthopnea and pedal edema. However, the classical symptoms of heart failure can be masked - especially in obese women[25]. Patients with PPCM present with the typical signs and symptoms of left ventricular failure. A physical examination will reveal tachycardia with or without abnormal heart sounds. There will be features of lung congestion. The liver may be enlarged and neck veins may be swollen. Blood pressure may be low or may drop when the patient stands up. Heart enlargement, congestion of the lungs or the veins in the lungs, decreased cardiac output, decreased movement or functioning of the heart or heart failure may show on chest x-ray, chest CT scan, coronary angiography, echocardiogram or nuclear heart scan.

The bulk of cases occur after delivery and the immediate postpartum period. However, when the disease develops during the last month of pregnancy the diagnosis of cardiac failure is tricky to make, by signs and symptoms alone since some of those symptoms, such as fatigue, orthopnea, and pedal edema, are common among normal parturients during late pregnancy. Further testing is required to establish the presence of cardiac failure.

It is important that a high index of suspicion be maintained to identify the rare case of PPCM. This is because general examination showing symptoms of heart failure with pulmonary edema can occur in many other situations as well. PPCM remains a diagnosis of exclusion. No additional specific criteria have been identified to allow distinction between a peripartum patient with new

onset heart failure and left ventricular systolic dysfunction as PPCM and another form of dilated cardiomyopathy. Therefore, all other causes of dilated cardiomyopathy with heart failure must be thoroughly excluded before accepting the designation of PPCM [9].

Recent observations from Haiti suggest that a latent form of PPCM without clinical symptoms might exist. The investigators identified four clinically normal postpartum women with asymptomatic systolic dysfunction on echocardiography, who subsequently either developed clinically detectable dilated cardiomyopathy or improved and completely recovered heart function[25].

A chest x-ray consistently demonstrates cardiomegaly and pulmonary edema. Echocardiography confirms ventricular failure with increased left ventricular end-diastolic dimensions and decreased ejection fraction (LVEF). The diagnosis of PPCM rests on the echocardiographic identification of new left ventricular systolic dysfunction during a limited period surrounding parturition [25]. Once cardiac failure is identified, PPCM must be differentiated from other disease processes that lead to heart failure, such as valvular heart disease.

A heart biopsy may be helpful in determining an underlying cause of cardiomyopathy. Many cases of PPCM seem to be related to myocarditis, which can be confirmed by a heart biopsy. However there is a paucity of good quality literature on this aspect of diagnostic modality.

Specific diagnostic criteria have been shown in Table 1 [26], and their addition has resulted in easier differentiation between PPCM and other causes of cardiac failure [27]. Table 2 shows echocardiographic criteria for diagnosis of PPCM

Table 1. Diagnostic Criteria For PPCM

1. Development of heart failure within last month of pregnancy or six months post-partum
2. Absence of any identifiable cause of heart failure
3. Absence of any recognizable heart disease before last month of pregnancy

Table 2. Additional Echocardiographic Diagnostic Criteria

Demonstrable echocardiographic criteria of left ventricular dysfunction:
1. Ejection fraction < 45%
2. Left ventricular fractional shortening <30%
3. Left ventricular end-diastolic dimension > 2.7cm/m^2 body surface area

Differences between PPCM and IDCM

The most common and confusing differential diagnosis of PPCM is Idiopathic Dilated Cardiomyopathy (IDCM). Though PPCM is identical to IDCM in several ways [1-3], most researchers now accept PPCM as a distinct entity for the following reasons [28,29].

1. PPCM occurs at a younger age and is generally associated with better prognosis.
2. The incidence of PPCM is higher than IDCM.
3. PPCM occurs mostly postpartum (78 - 93%), whereas IDCM usually manifests by the second trimester.
4. PPCM exclusively affects pregnant women and recurrent PPCM is seen to manifest again in the peripartum period.
5. Varying types of hemodynamic patterns are seen in PPCM compared to IDCM.
6. Unique sets of antigen and antibodies against myocardium are seen in PPCM compared to IDCM patients.
7. The incidence of myocarditis is higher in PPCM than in IDCM.
8. Heart size returns to normal after delivery in a greater percentage of PPCM patients compared to IDCM.
9. Contrary to IDCM, PPCM may lead to rapid worsening of clinical condition.

Prognosis

There are several possible outcomes in PPCM. Some women remain stable for long periods, while others get worse slowly. Others get worse very quickly and may be candidates for a heart transplant. The death rate may be as high as 25 - 50%. The prognosis is

poor in patients with persistent cardiomyopathy. Subsequent pregnancies are often associated with recurrence of left ventricular systolic dysfunction[30].

Interestingly recently Cardiac Magnetic resonance Imaging (CMRI) has been used for prognostication in PPCM. Although CMRI is largely used for diagnosis and prognosis assessment in cardiomyopathies, its importance in PPCM is unknown. Two cases of patients with PPCM who underwent CMRI have been reported. One patient had no CMRI abnormality, while the second patient had several areas of myocardial delayed enhancement. During follow up, the patient with normal images was asymptomatic and had full recovery of cardiac function, whereas the patient with myocardial delayed enhancement was still symptomatic with persistence of a left ventricular dysfunction. CMRI could have prognosis value in PPCM as demonstrated in other cardiomyopathies[31].

The outlook is good for women whose hearts return to normal size after the baby is born. If the heart remains enlarged, future pregnancies may result in heart failure. It is not known how to predict who will recover and who will develop severe heart failure. Long-term follow-up results of patients with PPCM and assessment of the echocardiographic findings relating to prognosis at time of diagnosis was undertaken in a study[32]. It was found that 24% patients recovered completely, 30% died, 6% underwent heart transplants, and 39% were left with persistent left ventricular dysfunction. Cut-off values for initial left ventricular end-systolic diameter (=5.5 cm) and LVEF (>27%) were obtained from the patients who had completely recovered.

Women who develop PPCM are at high risk of developing the same problem with future pregnancies and should discuss contraception with their physician.

Treatment

Early diagnosis and initiation of treatment are essential to optimize pregnancy outcome. For most women, treatment focuses simply on relieving the symptoms. Some symptoms resolve without treatment. The woman may need to stay in the hospital until acute symptoms subside. Because the heart dysfunction is usually reversible, and the women are usually young, everything possible is done to ensure survival. This may include taking extreme measures such as use of a balloon heart pump (aortic counter-pulsation balloon), immunosuppressive therapy and heart transplant. Patients with systolic dysfunction during pregnancy are treated similar to patients who are not pregnant. Pharmacological Management.

The mainstays of medical therapy are digoxin, loop diuretics, sodium restriction and after-load reducing agents (hydralazine and nitrates). Due to a high risk for venous and arterial thrombosis, anticoagulation with subcutaneous heparin should be instituted. Angiotensin converting enzyme inhibitors and angiotensin receptor blockers should be avoided during pregnancy because of severe adverse neonatal effects [32].

Digoxin

Digoxin is beneficial for its ionotropic and rate reducing effect, and provides symptomatic relief without reducing the mortality rate [32].

Diuretics

Diuretics are relatively safe in pregnancy and lactation. These are indicated for preload reduction and symptomatic relief when salt restriction fails. However, precaution must be taken against iatrogenic dehydration causing uterine hypoperfusion resulting in fetal distress [33,34].

Calcium channel blockers

Initially, the use of calcium channel blockers (CCB) was not acceptable in heart failure because of their negative contractile effect and the potential risk of uterine hypoperfusion. Amlodipine has now been shown to improve survival in non-ischemic cardiomyopathy patients [35].

Antiarrhythmic agents

Antiarrhythmic agents may sometimes be required to treat symptomatic patients. No antiarrhythmic agent is completely safe in pregnancy. Non-pharmacological means like assurance, carotid massage or the Valsalva maneuver may be tried initially. Quinidine and Procainamide should be tried first because of their higher safety profile[36]. Treatment should always start in a hospital setting because of the high incidence of torsades de

pointes associated with their use. Beta blockers can also be used. Digoxin can be considered for atrial arrhythmias [37].

Anticoagulant therapy

Patients with an LVEF <35% and bedridden patients with atrial fibrillation, mural thrombi, obesity and a history of thromboembolism benefit the most from anticoagulant therapy. The usual hypercoagulable state of pregnancy and stasis of blood due to ventricular dysfunction makes PPCM patients more prone to thrombus formation and subsequent complications. This situation may persist for as long as six weeks in the puerperium, necessitating the use of heparin in the antepartum and heparin or warfarin in the postpartum period. Warfarin is contraindicated in pregnancy for its teratogenic effect, but the use of both heparin and warfarin is safe in lactation [38].

Some new developments have been tried in the treatment of PPCM. Extracorporeal membrane oxygenation is an earlier option for the treatment and management of these patients as a bridge to recovery[39]. Recently bromocriptine has been reported to have given successful outcome along with treatment of heart failure in the management of PPCM[40]. Recent evidence suggests that the breakdown products from prolactin can induce cardiomyopathy. Prolactin secretion can be reduced with bromocriptine which had beneficial effects in a small study [41]. Cases of a patient with PPCM who received cabergoline, a strong and long lasting antagonist of prolactin secretion have also been reported. Following the treatment, prolactin levels dropped swiftly. N-terminal pro-BNP levels, which had remained high up to that point, dropped within one day. Echocardiographic left ventricular ejection fraction recovered from day one and postpartum significantly by day five after cabergoline treatment [41]. This may prove an important treatment modality in the management of PPCM in times to follow.

A low salt diet may be recommended. Fluid may be restricted in some cases. Activities, including nursing the baby, may be limited when symptoms develop. Daily weighing may be recommended. A weight gain of 3 or 4 pounds or more over one or two days may be a sign of fluid buildup. Women who smoke and drink alcohol will be advised to stop, since these habits may make the symptoms worse.

As regards the obstetric management, in addition to treatment of the cardiac failure, an obstetric plan of care must be developed when the disease occurs during pregnancy. Collaboration among the obstetrician, cardiologist, and anesthesiologist is essential to optimize care. If the parturient's cardiac status can be stabilized with medical therapy, induction of labor is usually recommended with cesarean section reserved for obstetric indications. However, in parturients who experience acute cardiac decompensation, cesarean delivery may be required because of an inability of the mother to tolerate the prolonged stress of labor.

Intensive Care Management

Patients with severe forms of heart failure may require more aggressive management in an ICU with monitoring of arterial blood pressure (ABP), central venous pressure (CVP), a pulmonary artery catheter (PAC) and echocardiography along with inotropes, vasodilator and ventilator therapy [42]. In extreme situations, multiorgan support like circulatory assist devices and continuous veno-venus hemodialysis may be instituted [43].

Anesthetic Management

Preoperative optimization

Labor analgesia

Single shot spinal anesthesia is currently not preferred because of severe consequences like cardiac arrest and pulmonary edema [44]. Controlled epidural analgesia (EA) under invasive monitoring is a safe and effective method [45].

Anesthesia for cesarean section

Both general anesthesia (GA) and regional anesthesia (RA) have been used.
Regional anesthesia (RA):

Though RA has the advantages of sympathetic blockade-induced preload and after load reduction [42,46], spinal anesthesia is rarely used because of complications, as mentioned above. Graded EA has been mostly used because of its better hemodynamic stability [42,46].

General anesthesia

GA may be needed in emergency situations or when RA is contraindicated, particularly in anticoagulated patients [47].

Possible Complications

The most common complications of PPCM include cardiac arrhythmias that can be fatal, congestive heart failure and pulmonary embolism. However, rarely has acute permanent unilateral blindness due to embolic retinal artery occlusion as a consequence of PPCM been reported [48]. There are reports of PPCM subjects presenting with acute abdomen due to unrecognized thromboemboli of the abdominal organs [49].

Thromboembolism

Thrombi often form in patients with LVEF <35%[50] and mortality rates of 30-50% have been reported to be due to thromboembolism[51]. Systemic embolism leading to transient ischemic attack (TIA), hemiplegia, pulmonary embolism, acute myocardial infarction (AMI), mesenteric artery occlusion presenting as acute abdomen, infarction of kidney resulting in pyelonephritis and splenic infarction have been reported[52,53]. Peripheral thromboembolism leading to limb ischemia and gangrene have also been reported [33].

Arrhythmias

Arrhythmias like sinus tachycardia, atrial and ventricular tachycardia, atrial flutter and fibrillation, ventricular premature beats, atrial and ventricular extra systoles and Wolfe-Parkinson-White Syndrome are reported in PPCM [54]. Ventricular tachycardia leading to cardiac arrest has also taken place[55]. Increasing use of automated implantable cardioverter defibrillators (AICD) in PPCM patients supports the high risk of life threatening arrhythmias[56].

Organ failure

Acute liver failure and hepatic coma arising from passive congestion of cardiac failure in a PPCM patient have been described. Fatal bacteremia and multiorgan failure including the heart, liver and kidney have also been reported [55,56].

Obstetric and perinatal complications

Increased incidence of premature delivery (11 - 50%), small for date and low birth weight babies, intrauterine growth retardation and fetal deaths are reported in PPCM. Congenital fetal anomalies are also described in a few cases (4 - 6%)[55]. Congestive cardiac failure is associated with higher infant mortality (10%).

Prevention

PPCM is difficult to prevent as its exact etiology is still unknown. However if pregnancy is avoided in subjects in whom the heart function has not recovered fully, recurrence can be prevented. Most reports describe recurrence of PPCM in subsequent pregnancies. It is not clear whether this is due to exacerbation of previous subclinical failures or reactivation of the same disease process[21]. The highest risk of recurrence remains in patients with persistent cardiac dysfunction and the lowest risk is in those whose cardiac functions have been normalized, as evidenced by dobutamine stress test[27,33].

Areas of future research

Areas for future research will be focusing on finding the etiology. This includes immune system dysfunction, the role of viruses, non-conventional treatments such as immunosuppression, immunoadsorption, apheresis, antiviral treatment, suppression of pro inflammatory cytokines, and strategies for control and prevention.

Conclusion

PPCM is an uncommon but potentially life threatening cardiac failure of undetermined etiology taking place in late pregnancy or early on in puerperium. Diagnosis of PPCM should essentially include echocardiographic substantiation of left ventricular dysfunction. The present diagnostic role of EMB in PPCM is uncertain but may be considered in defiant cases. Regular medical management should be commenced with digoxin, diuretics, vasodilators, β blockers and anticoagulants. In resistant cases, management with immunosuppressive drugs, immunoglobulin and pentoxifylline can be thought of. Severe cases may require exhaustive management, including mechanical circulatory support and heart transplant. Induction of labor should be done in intensive care surroundings. Cesarean section should be set aside for obstetric indications. Regional techniques are

less risky for labor analgesia as well as anesthesia. Prognosis is linked to recovery of ventricular dysfunction. Future pregnancy is better avoided in patients with unrelenting cardiac failure. If inevitable, subsequent pregnancy in patients with enhanced cardiac function should be managed in a multidisciplinary unit

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Note: These articles are not yet published at the time of writing this review. They have been referred to on the basis of their abstracts published as electronic publication (Epub ahead of print). Therefore their volume no. and page no. are not mentioned.