Peripartum Cardiomyopathy

Anant Agrawal *[1], Sanket Saraiya [2].

*[1]Assistant Professor, Department of Cardiology, Parul Sevashram Hospital, Vadodara, Gujarat, India.
[2]Assistant Professor, Department of Cardiology, Parul Sevashram Hospital, Vadodara

Corresponding Author: Dr. Anant Agrawal, [DM Cardiology]. Assistant Professor, Department of Cardiology, Parul Sevashram Hospital, Vadodara, Gujarat, India. E-Mail: dragrawal.anant@gmail.com

Abstract
Peripartum Cardiomyopathy [PPCM] is rare form of cardiomyopathy of unknown etiology occurring in late pregnancy or postpartum period. PPCM is associated with significant morbidity and mortality in peripartum period. As ACE inhibitors are contraindicated during pregnancy, diuretics and betablockers are main stay of treatment. In postpartum period, PPCM is treated as per guideline directed therapy for heart failure. In many patients with PPCM, LVEF improves after postpartum period but in those, whose LVEF remains depressed has high tendency for recurrence in subsequent pregnancies with increasing mortality. Most common arrhythmias occurring with PPCM are tachyarrythmias and main stay of treatment are beta blockers. Many newer therapies including bromocriptine, pentoxifylline and immunoglobulin are being tried to treat PPCM. Device therapy is seldom required in PPCM and even if recommended, should be waited for at least 3 to 6 month after diagnosis.
Keywords: Peripartum, Cardiomyopathy, Pregnancy, Heart Failure.

Background:

Peripartum cardiomyopathy, also known as Toxic postpartum heart failure, Meadow's Syndrome, Zaria Syndrome, Postpartum Myocardiosis is a disease state of unknown etiology. It was first described as separate clinical entity in 1930 [1].

Definition: Peripartum Cardiomyopathy is defined as condition comprising of3 clinical criteria Development of Heart Failure in last month of pregnancy or within 5 months of delivery, absence of another identifiable cause for HF and LV systolic dysfunction [LVEF <45% or decreased fractional shortening [2].

Women who develop cardiomyopathy earlier in pregnancy though do not meet the criteria for the definition of peripartum cardiomyopathy, the disease process is considered to be the same based upon the presentation, recovery and maternal outcomes [3] and the term 'Pregnancy Associated Cardiomyopathy' has been used to describe this condition

Epidemiology: PPCM is a uncommon disorder with reported incidence varying in different geographical regions with incidence as low as 1 per 2500 to 4000 live births in USA to as high as 1 in 300 and 1 in 100 live births in Haiti and Nigeria respectively [2,4]. The precise incidence in India is not known, an incidence of one case per 1374 live births has been reported from a tertiary care hospital from South India [5].

Etiology: The underlying cause of PPCM has not been clearly defined. Heart biopsies performed in women with PPCM have shown inflammation in 10% to 75% of cases. This may be attributable to a prior viral illness or abnormal immune response; although there is no evidence that antiviral or immunosuppression medications improve outcomes. Other potential causes of PPCM

include nutritional deficiencies, coronary artery spasm, small-vessel disease, and defective antioxidant defenses. Genetics may also play a role in the tendency to develop PPCM. Role of prolactin metabolites in Pathogenesis of PPCM has recently been proposed

Risk Factors: Several risk factors are associated with increased risk of PPCM.[Table 1].Although multiparity has been traditionally considered a risk factor for PPCM, studies have shown that the majority of patients who develop PPCM do so during the first or second pregnancy [3,6]. Some studies have suggested association between selenium deficiency and peripartum cardiomyopathy [7,8].

Clinical Presentation: Most patients [80%] present within 3 months of delivery, with the minority presenting in the last month of pregnancy [10%] or 4 to 5 months postpartum [10%][9]
Symptomatology and clinical presentation of PPCM is same as that of Heart Failure from any cause and most common presentation is Dyspnea on exertion. Other symptoms include cough, orthopnea, paroxysmal nocturnal dyspnea, fatigue, palpitations, weight gain, hemoptysis, chest pain, and unexplained abdominal pain.

Systemic and Pulmonary Emboli are more common in Peripartum Cardiomyopathy owing to the Hypercoagulable state induced by pregnancy. Patients with PPCM and left ventricular ejection fraction [LVEF]<35% are at risk for developing LV thrombus. In one series, LV thrombus was identified by echocardiography in 16 of 100 patients with PPCM [with mean LVEF of 26 percent][10].

Diagnosis: Diagnosing PPCM requires high level of suspicion as most of its symptoms can be present during normal pregnancy. Also there is no definite criteria to differentiate patients of PPCM from those of Heart failure from other causes who present first time during peripartum period, so other causes of Heart failure should be ruled out before making diagnosis of PPCM.Diagnosis of PPCM is based upon the clinical criteria mentioned above in the definition [2].

ECG: ECG findings include Sinus tachycardia, nonspecific ST-T changes and voltage abnormalities [low voltage or criteria for left ventricular hypertrophy]. Tachyarrythmias including AF and VT are more common than bradyarrythmias

Echocardiography: The echocardiogram generally reveals a global reduction in LV systolic function with LVEF nearly always <45% [2]. The left ventricle is frequently but not always dilated.Other possible echocardiographic findings includeregional heterogeneities of systolic wall thickening,left atrial enlargement,mitral and tricuspid regurgitation and small pericardial effusion.

BNP: BNP levels in healthy women increase approximately twofold during pregnancy but are lower than the levels observed in patients with heart failure [11] and cutoff value of BNP and NT-pro BNP for diagnosing Heart failure are same in pregnancy as that of non-pregnant patients

Cardiac magnetic resonance imaging: Cardiac magnetic resonance imaging [CMR] in PPCM can be helpful to assess LV systolic function and LV volumes, particularly if echocardiography is technically suboptimal. Case reports and small series have noted variable presence of late gadolinium enhancement [LGE] in patients with PPCM. This variability likely reflects the diverse processes that lead to PPCM. The presence and persistence of LGE may be associated with poor recovery of cardiac function ; Improving LGE may be associated with cardiac recovery, while lack of LGE may be associated with presence or absence of cardiac recovery [12]. However, the prognostic value of CMR in PPCM has not been established.

Cardiac catheterization: Right heart catheterization is rarely needed because assessment of cardiac pressures can usually be made with physical examination and Doppler echocardiography. Left heart catheterization with coronary angiography is only indicated in selected patients in whom it is deemed necessary to evaluate coronary artery disease as a potential cause for the cardiomyopathy.

Endomyocardial biopsy: Endomyocardial biopsy [EMB] is generally not required in patients with suspected PPCM. There are no pathognomonic findings in PPCM and a variable proportion of patients have evidence of myocarditis

Differential Diagnosis: Peripartum cardiomyopathy [PPCM] is a diagnosis of exclusion. As noted in the 2010 European Society of Cardiology working group statement on PPCM [2], the conditions that should be considered in the differential diagnosis are preexisting cardiomyopathy, preexisting acquired or congenital valvular heart disease unmasked by pregnancy. In both these conditions, heart failure is more likely to manifest antepartum in contrast to PPCM. Other Differential diagnosis includes preexisting undetected congenital heart disease, diastolic heart failure due to hypertensive heart disease, myocardial infarction mainly due to coronary artery dissection or spasm and pulmonary thromboembolism.

Management

Treatment of Heart failure

Antepartum: Women with HF during pregnancy should be treated similarly to other patients with HF. Angiotensin converting enzyme inhibitors[ACEI] andangiotensin II receptor blockers[ARBs] are main stay of treatment in Heart failure. The teratogenicity with ACEI and ARBs occur particularly in the second and third trimester, characterized by fetal hypotension, pulmonary hypoplasia, oligohydramnios, anuria, and renal tubular dysplasia [13,14]. A recent study suggested risk of malformations after first trimester exposure to ACE inhibitors [15]. Aldosterone Antagonists, spironolactone has been associated with feminization of the male fetus due its antiandrogenic effects in animal studies. There are neither data nor clinical experience to support the safety of these agents during pregnancy.

Beta Blockers are generally safe and effective during pregnancy, although there may be an increased rate of fetal growth restriction when they are administered [16,17]. In general, agents that are beta1 selective [e.g., metoprolol] are preferable, since these agents are less likely to interfere withbeta2mediated uterine relaxation and peripheral vasodilation. However, atenolol should not be used during pregnancy since its use is associated with fetal growth restriction [18]. Digoxin, loop diuretics, sodium restriction and drugs that reduce afterload such as hydralazine and nitrates have been proven to be safe and are the mainstays of medical therapy of heart failure during pregnancy.

Postpartum: Treatment is identical to that for non-pregnant women with dilated Cardiomyopathy. ACE inhibitors and ARBs are useful. Levels in breast milk of ACE inhibitors are low and not expected to cause adverse effects in breastfed infants[19].An ACE inhibitor that has been studied in breast milk and/or breastfed infants[such as enalapril, captopril, quinapril, or benazepril] is preferred. There are no data on ARB safety during breastfeeding. Limited data suggest that spironolactone use is acceptable during breastfeeding since maternal diuretic effect is unlikely potent enough to effect lactation and the dose to the breastfeeding infant is low[19]. Data are lacking on use of eplerenone during breast feeding. The excretion of beta blockers into breast milk appears to be higher for drugs with low protein binding and thus Metaprolol and

carvedilol are preferred over Bisoprolol in Nursing woman [19,20].

Antithrombotic therapy: Although Patients with PPCM are at high risk for thrombus formation and thromboembolism due to both the hypercoagulable state of pregnancy and stasis of blood due to severe LV dysfunction, data are inconclusive on the utility of antithrombotic therapy [antiplatelet therapy or anticoagulation] to reduce thromboembolic events or mortality in patients with systolic HF who are in sinus rhythm. Neither the 2011 European Society of Cardiology [ESC] guidelines on management of cardiovascular disease during pregnancy nor the 2010 ESC position statement on peripartum cardiomyopathy recommend routine anticoagulation for this indication [2,21]. Anticoagulation with subcutaneous heparin or Vitamin K antagonist should be instituted in those with low LVEF, presence of atrial fibrillation, mural thrombi orthose with history of thromboembolism [22]

Mechanical circulatory support and cardiac transplantation: Mechanical circulatory support and heart transplantation are potential treatment options when HF is refractory to conventional therapy. Mechanical circulatory support with a ventricular assist device can be used as a bridge to transplantation or recovery when other therapies are not adequate to sustain life as the patient awaits the arrival of a new heart, particularly if the patient is dependent on inotropes despite expert HF therapy. Outcomes were reported for 1258women, which included 99 with PPCM, who had received durable mechanical circulatory support. Women with PPCM who received durable mechanical circulatory support had better survival than women without PPCM, with two year survival of 83 percent for the PPCM cohort [24].

Cardiac transplantation should be reserved for patients where mechanical circulatory support is not possible or not desirable for individual reasons or for patients who do not recover after 6–12 months on mechanical circulatory support. In addition to the potential maternal and fetal risks related to pregnancy after heart transplantation for any reason women who have been transplanted for PPCM are at increased risk for graft failure[23].

Arrhythmia: Atrial Fibrillation occurs occasionally in PPCM. DC cardio version is recommended for hemodynamically unstable AF. For hemodynamically stable AF Quinidine and Procainamide can be tried for their better safety profile during pregnancy but due to negative inotropic effect of Procainamide, it should be used cautiously in PPCM. Guidelines for anticoagulation during pregnancy for AF are same that of non-pregnant patients

Ventricular Tachycardia is common in PPCM. For acute treatment of VT with hemodynamic instability, immediate cardio version, which seems safe in all phases of pregnancy, is recommended. Timely restoration of sinus rhythm is desirable even if VT is well tolerated, and can be achieved with cardioversion, anti-arrhythmic medication, or, in selected cases, overdrives pacing. I.v. sotalol acutely can be considered to terminate the tachycardia. I.v. amiodarone should be considered for patients with sustained monomorphic VT that is hemodynamically unstable, refractory to conversion with countershock, or recurrent despite other agents. I.v. amiodarone is not ideal for early conversion of stable monomorphic VT[22]. Amiodarone has been associated with congenital goiter/hypothyroidism & hyperthyroidism. For prevention of VT optimization of Beta Blocker therapy should be done

Device Therapy: Up to 20 to 60 percent of women with PPCM have complete recovery of left ventricular ejection fraction [LVEF] to normal by six months to five years[3,25-27],.ICD placement should generally be deferred at least three months and possibly

even six months following presentation, with the patient receiving optimum medical therapy to determine whether criteria for placement are present. Specific indications for use of ICD therapy have not been established for PPCM. Role of Wearable defibrillator has not been established during pregnancy As for ICD specific indications of cardiac resynchronization therapy has not been established but should be deferred for at least 3-6 months.

Table 1: Risk Factors for Peripartum Cardiomyopathy.

Older maternal age [>30 yrs]
Multiparity [1 or more prior pregnancies]
Multifetal pregnancy [eg, twins]
African descent
History of preeclampsia, eclampsia , or postpartum hypertension in earlier or same pregnancy -
Prior toxin exposure [e.g., cocaine]
Long term [>4 weeks] oral tocolytic therapy with beta Adrenergic agonists.

Table 2: Poor prognostic factors in PPCM.

Worse New York Heart Association functional class [10]
Left ventricular ejection fraction [LVEF] ≤25 percent [32]
Black race [33]
Multiparity [33]
Age greater than 30 to 35 years [34]

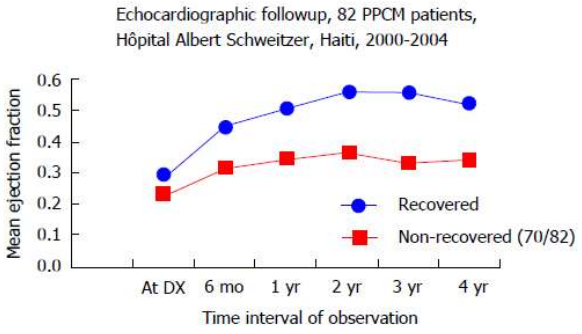

Figure 1 [38]

Newer therapies

Bromocriptine: This treatment strategy is based upon an experimental observation of prevention of PPCM in mice via prolactin blockade with bromocriptine[28]. Of note, the drug stops the production of breast milk making breastfeeding impossible. In a randomized open label study performed in South Africa, 20 women with newly diagnosed PPCM were randomly assigned to receive either standard care plus bromocriptine [2.5 mg twice daily for two weeks followed by 2.5 mg daily for six weeks] or standard care alone. The 10 women receiving bromocriptine demonstrated significantly greater improvement in LVEF as compared to the 10 women receiving standard care only [27 to 58 percent versus 27 to 36 percent]. One patient in the bromocriptine group died as compared to four in the standard care group. Fewer patients in the bromocriptine group reached the composite end point of death, New York Heart Association functional class III or IV HF , or LVEF <35 percent at six months, as compared to patients in the standard care group [one versus eight][28]. The generalizability of these results is unclear given the small sample size, the higher than expected mortality rate in the standard care group, and differences in characteristics of PPCM in patients in Africa as compared to those elsewhere. This drug appears promising; however, available data are insufficient to recommend routine use of bromocriptine treatment for PPCM.

Pentoxifylline: Pentoxifylline, as an inhibitor of the proinflammatory cytokine, Tumor Necrosis Factor-α, appeared earlier in South Africa to be helpful to improve left ventricular function. However, in recent trials in Haiti, pentoxifylline failed to show any evidence for improved survival or improved clinical or echocardiographic left ventricular function[29].

Intravenous immune globulin: Intravenous immune globulin [IVIG] has been tried in

patients with myocarditis or recent onset dilated cardiomyopathy with no clear evidence of clinical benefit.

Delivery: Vaginal delivery is always preferable if the patient is hemodynamically stable and there are no obstetric indications for caesarean delivery. Close hemodynamic monitoring is required. Epidural analgesia is preferred. Pre-term delivery has been reported in 17% of patients with no marked negative effects on the child[30]. Urgent delivery irrespective of gestation duration should be considered in women with advanced heart failure and hemodynamic instability despite treatment. Caesarean section is recommended with combined spinal and epidural anesthesia [31]

Contraception: Since women with PPCM with persistent left ventricular [LV] dysfunction or LV ejection fraction [LVEF] ≤25percent at diagnosis are at high risk of recurrent PPCM, future pregnancy should be avoided in such patients[22]. Estrogen progestin contraceptives should be avoided, particularly early after diagnosis and in women with persistent LV dysfunction because of their potential to increase the risk of thromboembolism and water retention. Suggested Contraception in patients of PPCM are sterilization procedure, highly effective non estrogenmethod of contraception, such as the etonogestrel implant, a copper intrauterine device [IUD], or levonorgestrel releasingIUD.

Prognosis

Maternal outcome: The mortality rate for PPCM has been reported as approximately 10 percent in two Years[3]. Death due to PPCM is usually caused by progressive pump failure, sudden death, or thromboembolic events. Various adverse prognostic factors have been identified in patients of PPCM [Table 2]

With Earlier detection of PPCM and at higher LVEF during diagnosis chances of poor maternal outcomes are less as compared to delayed diagnosis and lower EF during diagnosis[Figure 1]

Recovery of left ventricular function: Full recovery of heart function occurs more frequently in PPCM than with any other dilated cardiomyopathy. The first United States prospective study of PPCM, the IPAC study showed that full recovery [LVEF ≥ 0.50] at 6 month postpartum came to a remarkable over 65 % of patients[35].

Risk during subsequent pregnancies: For the matter of risk during subsequent pregnancy patients of PPCM can be divided into those whose whom LV function recovered completely [EF>50%] and those with persistent LV dysfunction.

In a series of 44 women who had peripartum cardiomyopathy and had a total of 60 subsequent pregnancies following results were noted[36]. Among the first subsequent pregnancies in the 44 women, 28 occurred in women in whom left ventricular function had returned to normal [group 1] and 16 occurred in women with persistent left ventricular dysfunction [group 2]. Both the groups were associated with fall in mean LVEF [from 56+/-7 percent to 49+/-10 percent in group 1, P=0.002; and from 36+/-9 percent to 32+/-11 percent in group 2, P=0.08].LVEF fell by >20% in 21 percent of the women in group 1 and 25 percent of those in group 2symptoms of heart failure occurred in 21 percent of the women in group 1 and 44 percent of those in group 2. The mortality rate was 0 percent in group 1 and 19 percent in group 2 [P=0.06].

There are no clear guidelines regarding subsequent pregnancies in patients of PPCM but woman with recovered LV functions should be explained about the risk in subsequent pregnancies and those with persistent LV dysfunction should be counseled against the subsequent pregnancy.

Peripartum cardiomyopathy patients with persistent left ventricular dysfunction should

be continued on standard heart failure treatment indefinitely. Those with full recovery of left ventricular function [ejection fraction [LVEF] >50 percent] left ventricular dysfunction can reoccur despite initial full recovery and this recurrence risk is not limited to occurring during subsequent pregnancies. There are no guidelines regarding withdrawal of drugs in these patients but a review suggests slow stepwise withdrawal of drugs over the period of 6-12 months with periodic screening of LV functions.

Conclusion

Peripartum Cardiomyopathy, though a rare condition, is an important diagnosis as both maternal and fetal outcomes depends on early diagnosis and prompt treatment. Due to the reversible nature of the disease it is important to have high index of suspicion of the disease during its defined period. As subsequent pregnancies in patients of PPCM is associated with substantial morbidity and mortality, meticulous counseling should be done and appropriate treatment regimen should be decided according to the current guidelines.

References

[1]. Hull E, Hafkesbring E. "Toxic" postpartal heart disease. New Orleans Med Surg J 1937; 89:550.

[2]. Sliwa K, HilfikerKleiner D, Petrie MC, et al. Current state of knowledge on aetiology, diagnosis, management, and therapy of peripartum cardiomyopathy: a position statement from the Heart Failure Association of the European Society of Cardiology Working Group on peripartum cardiomyopathy. Eur J Heart Fail 2010; 12:767.

[3]. Elkayam U, Akhter MW, Singh H, et al. Pregnancyassociated cardiomyopathy: clinical characteristics and a comparison between early and late presentation. Circulation 2005; 111:2050.

[4]. Pearson GD, Veille JC, Rahimtoola S, Hsia J, Oakley CM, Hosenpud JD, Ansari A, Baughman KL. Peripartum cardiomyopathy: National Heart, Lung, and Blood Institute and Office of Rare Diseases [National Institutes of Health] workshop recommendations and review. JAMA. 2000;283:1183–1188.

[5]. Pandit V, Shetty S, Kumar A et al; Incidence and out come of peripartum Cardiomyopathy from a tertiary hospitalin South India. Trop Doct2009;39:168-9.

[6]. Amos AM, Jaber WA, Russell SD. Improved outcomes in peripartum cardiomyopathy with contemporary. Am Heart J 2006; 152:509.

[7]. Kothari SS. Aetiopathogenesis of peripartum cardiomyopathy: prolactinseleniumInteraction Int J Cardiol 1997; 60:111

[8]. Fett JD, Ansari AA, Sundstrom JB, Combs GF. Peripartum cardiomyopathy: a selenium disconnection and an autoimmune connection. Int J Cardiol 2002; 86:311.

[9]. Peripartum Cardiomyopathy Michael M. Givertz, MD Circulation.2013; 127: e622-e626doi: 10.1161/CIRCULATIONAHA.113.001851

[10]. Sliwa K, Förster O, Libhaber E, et al. Peripartum cardiomyopathy: inflammatory markers as predictors of outcome in 100 prospectively studied patients. Eur Heart J 2006; 27:441.

[11]. Hameed AB, Chan K, Ghamsary M, Elkayam U. Longitudinal changes in the Btype natriuretic peptide levels in normal pregnancy and postpartum. ClinCardiol 2009; 32:E60.

[12]. Baruteau AE, Leurent G, Martins RP, et al. Peripartum cardiomyopathy in the era of cardiac magnetic resonance imaging: first results and perspectives. Int J Cardiol 2010; 144:143.

[13]. Alwan S, Polifka JE, Friedman JM. Angiotensin II receptor antagonist treatment during pregnancy. Birth Defects Res AClinMolTeratol 2005; 73:123.

[14]. Lavoratti G, Seracini D, Fiorini P, et al. Neonatal anuria by ACE inhibitors during pregnancy. Nephron 1997; 76:235.

[15]. Angiotensin-converting enzyme inhibitors and angiotensin receptor blockers in women of childbearing age: risks versus benefits. Pucci M[1], Sarween N, Knox E, Lipkin G, Martin U.

[16]. Easterling TR, Carr DB, Brateng D, et al. Treatment of hypertension in pregnancy: effect of atenolol on maternal disease, preterm delivery, and fetal growth. ObstetGynecol 2001; 98:427.

[17]. Bayliss H, Churchill D, Beevers M, Beevers DG. Antihypertensive drugs in pregnancy and fetal growth: evidence for "pharmacological programming" in the first trimester? Hypertens Pregnancy 2002; 21:161.

[18]. Lydakis C, Lip GY, Beevers M, Beevers DG. Atenolol and fetal growth in pregnancies complicated by hypertension. Am J Hypertens1999; 12:541.

[19]. http://toxnet.nlm.nih.gov/cgibin/ sis/htmlgen?LACTMED[Accessed on July 01, 2014].
[20]. Beardmore KS, Morris JM, Gallery ED. Excretion of antihypertensive medication into human breast milk: a systematic review. Hypertens Pregnancy 2002; 21:85.
[21]. European Society of Gynecology [ESG], Association for European Paediatric Cardiology [AEPC], German Society for Gender Medicine [DGesGM], et al. ESC Guidelines on the management of cardiovascular diseases during pregnancy: the Task Force on the Management of Cardiovascular Diseases during Pregnancy of the European Society of Cardiology [ESC]. Eur Heart J 2011; 32:3147.
[22]. ESC Guidelines on the management of cardiovascular diseases during pregnancy The Task Force on the Management of Cardiovascular Diseases during Pregnancy of the European Society of Cardiology [ESC] European Heart Journal [2011] 32, 3147–3197 doi:10.1093/eurheartj/ehr218
[23]. Rasmusson K, Brunisholz K, Budge D, et al. Peripartum cardiomyopathy: posttransplant outcomes from the United Network for Organ Sharing Database. J Heart Lung Transplant 2012; 31:180.
[24]. LoyagaRendon RY, Pamboukian SV, Tallaj JA, et al. Outcomes of patients with peripartum cardiomyopathy who received mechanical circulatory support. Data from the Interagency Registry for Mechanically Assisted Circulatory Support. Circ Heart Fail 2014; 7:300.
[25]. Pillarisetti J, Kondur A, Alani A, et al. Peripartum cardiomyopathy: predictors of recovery and current state of implantable cardioverterdefibrillator use. J Am CollCardiol 2014; 63:2831.
[26]. Sliwa K, Skudicky D, Bergemann A, et al. Peripartum cardiomyopathy: analysis of clinical outcome, left ventricular function, plasma levels of cytokines and Fas/APO1. J Am CollCardiol 2000; 35:701.
[27]. Cooper LT, Mather PJ, Alexis JD, et al. Myocardial recovery in peripartum cardiomyopathy: prospective comparison with recent onset cardiomyopathy in men and nonperipartum women. J Card Fail 2012; 18:28.
[28]. Sliwa K, Blauwet L, Tibazarwa K, et al. Evaluation of bromocriptine in the treatment of acute severe peripartum cardiomyopathy: a proofofconcept pilot study. Circulation 2010; 121:1465.
[29]. Fett JD, Sanon H, Carraway RD, Markham DW, Ernst S. Abstract 189: Pentoxifylline treatment for peripartum cardiomyopathy? J Cardiac Fail 2013; 19: S65-S66 [DOI: 10.1016/ j.cardfail.2013.06.213]
[30]. de Beus E, van Mook WN, Ramsay G, Stappers JL, van der Putten HW. Peripartum cardiomyopathy: a condition intensivists should be aware of. Intensive Care Med 2003;29:167–174.
[31]. Tomlinson M., Cardiac Diseases. In: James DK, Steer PJ, Weiner CP et al., eds. High Risk Pregnancy. Management Options. 3rd edn. Philadelphia: Elsevier Saunders; 2006. p798–827.
[32]. Haghikia A, Podewski E, Libhaber E, et al. Phenotyping and outcome on contemporary management in a German cohort of patients with peripartum cardiomyopathy. Basic Res Cardiol 2013; 108:366.
[33]. Murali S, Baldisseri MR. Peripartum cardiomyopathy. Crit Care Med 2005; 33:S340.
[34]. Kao DP, Hsich E, Lindenfeld J. Characteristics, adverse events, and racial differences among delivering mothers with peripartum cardiomyopathy. JACC Heart Fail 2013; 1:409
[35]. McNamara D, Damp J, Elkayam U, Hsich E, Ewald G, Cooper L, Modi K, Ramani G, Alexis J, Semigran M, Drazner M, Haythe J, Pisarcik J, Marek J, Gorcsan J, Fett J. Abstract 12898: Myocardial recovery at six months in peripartum cardiomyopathy: Results of the NHLBI multicenter IPAC study [Circulation 2013, Supplement]. Issue 22, Nov 26
[36]. Fett JD, Sannon H, Thélisma E, et al. Recovery from severe heart failure following peripartum cardiomyopathy. Int J GynaecolObstet 2009; 104:125.
[37]. HilfikerKleiner D, Haghikia A, Nonhoff J, Bauersachs J. Peripartum cardiomyopathy: current management and future perspectives. Eur Heart J 2015; 36:1090
[38]. Peripartum cardiomyopathy: A puzzle closer to solution James D Fett, Investigations of Pregnancy Associated Cardiomyopathy [IPAC], Peripartum Cardiomyopathy Network of North America [PCN], Cardiovascular Institute, University of Pittsburgh Medical Center, Pittsburgh, PA 15213, United States James D Fett, Department of Adult Medicine, Hospital Albert Schweitzer, Deschapelles, Haiti *World J Cardiol*2014 March 26; 6[3]: 87-99.

Article information

Manuscript Submitted: 27-07-2022 **Manuscript Revised:** 20-08-2022
Manuscript Accepted: 13-10-2022 **Manuscript published:** 22-11-2022

Scan here to access this article online