International Journal of Clinical Obstetrics and Gynaecology

ISSN (P): 2522-6614
ISSN (E): 2522-6622

www.gynaecologyjournal.com
2019; 3(4): 102-106
Received: 24-05-2019
Accepted: 26-06-2019

Ban Asad Hussein
Ministry of Health-Baghdad Al-Russafa Health Directorate, Al-Wasiti Teaching hospital for Reconstructive Surgery, Baghdad, Iraq

Shahla Muthana Khlil-Fahmi
Ministry of Health - Medical City-Ghazi al-Hariri Hospital for Specialized Surgery, Baghdad, Iraq

Correspondence
Ban Asad Hussein
Ministry of Health-Baghdad Al-Russafa Health Directorate, Al-Wasiti Teaching hospital for Reconstructive Surgery, Baghdad, Iraq

Updates in the preoperative anesthetic management of cardiomyopathy in pregnant patients

Ban Asad Hussein and Shahla Muthana Khlil-Fahmi

DOI: https://doi.org/10.33545/gynae.2019.v3.i4b.299

Abstract
Introduction: Tremendous advance in mechanization and fastness of travel have been accompanied by steep increase in number and severity of fractures and those of tibial plateau are no exception. Knee being one of the major weight bearing joints of the body, fractures around it will be of paramount importance.
Materials and Methods: A 4 year prospective study was conducted, to know the functional outcome in operatively treated tibial plateau fractures in adults were included from BLDE University, Vijayapura. The total number 60 patients.
Results: Out of 60 cases treated with surgical procedure, 39 cases gave excellent result, 12 cases came out with good result, fair in 3 case and 3 case had poor result, mainly due to the severity of the injury and infections. It was found that high velocity injuries (type IV - VI) have poor outcome than low velocity injuries (type I-III) 4. All fractures united within expected time. Not a single case of nonunion was noted in our series. Average time for union was 14 weeks (range 10-22 weeks). The three cases had wound infection two had stiffness of the knee joint and one case had malunion.
Conclusion: We conclude that the functional outcome is good in operatively treated tibial plateau fractures in adults. The surgical management of tibial plateau fractures is challenging and gives excellent anatomical reduction & rigid fixation to restore articular congruity, fascilitate early knee motion by reducing post-traumatic osteoarthritis and thus achieving optimal knee function.

Keywords: Key words-proximal tibia fracture, MIPPO, knee stiffness, wound dehiscence

Introduction

Peripartum cardiomyopathy (PPCM) is a potentially life-threatening pregnancy-associated disease that typically arises in the peripartum period. While the disease is relatively uncommon, its incidence is rising. It is a form of idiopathic dilated cardiomyopathy, defined as pregnancy-related left ventricular dysfunction, diagnosed either towards the end of pregnancy or in the months following delivery, in women without any other identifiable cause.

The clinical presentation, diagnostic assessment, and treatment usually mirror that of other forms of cardiomyopathy. Timing of delivery and management require a multidisciplinary approach and individualization. Subsequent pregnancies generally carry risk, but individualization is required depending on the pre-pregnancy left ventricular function. Recovery occurs in most women on standard medical therapy for heart failure with reduced ejection fraction, more frequently than in other forms of nonischemic cardiomyopathy [1].

The purpose of this review is to summarize the current state of knowledge with regard to diagnosis, treatment, and management, with a focus on long term implications.

Cardiomyopathy is a disease of the heart muscle that can be inherited or acquired and can affect people of all ages. Cardiomyopathy affects the shape, function, and electrical system of the heart. In the UK, the estimated incidence of cardiomyopathy is 1 in 500. Although it is not a curable condition, the signs and symptoms can usually be managed successfully and patients can have a good life expectancy [2].

Peripartum cardiomyopathy (PPCM) was first reported in the year 1849. Till the middle of 20 th century, it was known as postpartum cardiomyopathy [3] as most of the cases reported had symptomatic onset in the postpartum period only. Demakis *et al.* [4] were probably the first to realize this as more of a peripartum disease rather than a postpartum one, hence the term peripartum cardiomyopathy was considered more acceptable. The first case series of patients with PPCM was published in the year 1971 [5] by Demakis *et al.* They described the data about 27 patients who presented in the late pregnancy or early puerperium with heart failure. Included

development of cardiac failure in the last month of pregnancy or within 5 months of delivery, absence of a determinable etiology for the cardiac failure and absence of demonstrable heart disease before the last month of pregnancy. [6]

Over the years, the diagnostic criteria have remained almost the same with the addition of echocardiography findings as another parameter. As more and more research is being conducted, a lot more is now known about the pathophysiology, epidemiology, diagnosis and clinical outcome of the disease. At the same time, the unique anesthetic challenge that these patients pose for management of their pregnancy was also recognized. This review will undertake a comprehensive look on the above and give a detailed account of the treatment modalities available for this disease in present times. [7]

Background

Definition and Diagnostic Criteria

One of the most recent definitions of PPCM has been provided by the Heart Failure Association of the European Society of Cardiology Working Group on PPCM which describes it as "*an idiopathic cardiomyopathy presenting with heart failure secondary to left ventricular systolic dysfunction towards the end of pregnancy or in the months following delivery, where no other cause of heart failure is found. It is a diagnosis of exclusion. The left ventricle may not be dilated but the ejection fraction is nearly always reduced below 45%* [8].

Earlier, in the year 1997, the National Heart, Lung and Blood Institute and the Office of Rare Diseases of the National Institutes of Health had convened a Workshop on Peripartum Cardiomyopathy to foster a multidisciplinary review and defined the diagnostic criteria [9] based on the work by Demakis *et al*. [Table 1]. This definition was being followed comprehensively till now, but the European Society definition claims to be more simplified and capable of preventing underdiagnosis of the disease.

Table 1: Criteria for diagnosis of peripartum carclionlyopathy't

• Development of heart failure in the last month of pregnancy or within 5 months postpartum.
• Absence of an identifiable cause of heart failure. • Absence of recognizable heart disease before the last month of pregnancy. • Left ventricular systolic dysfunction demonstrated by left ventricle ejection fraction of less than 45%, fractional shortening of less than 30%, or both, with or without a left ventricle end-diastolic dimension less than 2.7 cm/m^2 of body surface area.

*As defined by Workshop convened by NH LBI and NIH Office of Rare Diseases [151]

General Considerations

Hemodynamic changes during pregnancy. Dramatic changes occur to the cardiovascular system during pregnancy. Initially, marked increases in circulating blood volume are met with an increase in stroke volume and a 15% to 20% increase in heart rate. The net effect is a 30% to 50% increase in cardiac output by the end of the first trimester, an effect that peaks between the second and third trimesters [10].

Another important consideration is the maturation of a placental circulation, which provides a substantial reduction in systemic vascular resistance. During the third trimester, preload reduction might occur due to compression of the inferior vena cava (IVC) by the gravid uterus, thus reducing cardiac output. Increases in cardiac output and intravascular volume allow 1 cardiac pump to feed both maternal and fetal tissues. It is indisputable that blood volume increases in pregnancy, but studies differ on when volume expansion levels off, if at all. Increases in blood volume enhance left ventricular end-diastolic volume, which peaks during the third trimester. This increased preload is thought to be due, in part, to an estrogenic effect, which creates higher circulating renin levels and greater sodium and water retention. Alternatively, hormones such as prolactin, human placental lactogen, prostaglandins, and growth hormone have also been implicated.

Hemodynamic changes during labor and delivery. The cardiovascular system of women with heart disease is limited in its ability to accommodate the demands of pregnancy. These limitations become more evident during labor and delivery, where several changes in the circulatory system could result in hemodynamic decompensation [11]. There is a catecholamine-induced increase in heart rate and stroke volume due to pain and anxiety. During the peripartum period, there can be an increase of cardiac output of up to 31% and approximately 50% in the second stage of labor.

Abrupt changes in fluid balance result from a lack of IVC compression as well as the redistribution of blood from the lower limbs, particularly during uterine contractions. This rapid increase in preload can result in pulmonary congestion and clinical heart failure. Some of this intravascular volume is lost at delivery, where variable blood loss will occur— approximately 500 ml with a normal vaginal delivery, and 1,000 ml for a routine cesarean section. Further alterations in the hemodynamic status occur most commonly within the first 12 to 24 h post-partum. Within the first hour of delivery, cardiac output might continue to increase to as much as 80% above pre-labor values due to the relief of IVC compression and potentially rapid auto transfusion from the placenta (13). Moreover, further fluctuations in hemodynamic status can be due to the loss of the low resistance placenta and a relative increase in systemic vascular resistance as well as the mobilization of dependent edema and interstitial fluid. [12]

The use of anesthesia and analgesia can cause hypotension as a result of venous pooling and decreased systemic vascular resistance. Therefore, women with pre-existing cardiomyopathies might be at high risk for peripartum complications, due to the inability to accommodate increased cardiac output. Pre-conception risk assessment and counseling. Women with cardiac disease require a complete pre-conception evaluation and counseling to risk-stratify the maternal and fetal risks of pregnancy (Table 1) [13].

As such, appropriate evaluations can take place without putting the fetus at risk. A detailed history and physical examination, assessment of functional capacity and New York Heart Association (NYHA) functional class, and a 12-lead electrocardiogram are essential. Echocardiography is indicated in women with a history of valvular or congenital heart disease, significant dyspnea or any symptoms, any signs of heart failure, and systolic murmur gradeII, or any diastolic murmur. In addition, the etiology and degree of valvular regurgitation and/or stenosis, degree of pulmonary hypertension, [14] and—if present—aortic root dilation can be quantified.

Importantly, the left ventricular or systemic ventricular systolic function can also be determined. In certain congenital heart disease patients, assessment of the right heart size and function can be achieved most accurately with cardiac magnetic

resonance imaging. Exercise stress testing can be useful to quantify the functional capacity of a patient if the history of the patient is unclear. However, this should ideally be performed before pregnancy [15].

Poor functional status has been previously identified to be associated with maternal or fetal complications [16]. Functional capacity might be an important predictor of the ability to tolerate a pregnancy, regardless of the underlying lesion. In a recent study examining pregnancy outcomes in women with congenital heart disease, an abnormal chronotropic response correlated with adverse pregnancy outcomes and could be considered in refining risk stratification schemes [17].

Management

The goals of medical management in a patient diagnosed with PPCM should include measures to improve oxygenation and maintain cardiac output so as to improve both maternal and fetal outcome. Interventions are required to decrease both preload and afterload as well as to improve cardiac contractility.

Mild to moderate symptoms may be managed with rest, salt restriction and diuretic therapy. Oxygen may be instituted via face mask, or continuous positive airway pressure may be applied up to a level which does not further jeopardize the cardiac output. Salt restriction helps in preventing further water retention, while diuretics help in decreasing pulmonary congestion.

Fluid restriction may not be warranted in patients with mild to moderate heart failure. Hydralazine and nitrates decrease the afterload and are the mainstay of treatment in pregnant patients with heart failure. Calcium channel blockers, except amlodipine, have a negative inotropic effect and should be avoided. Amlodipine may be used if PPCM is associated with pre-eclampsia to control blood pressure [18].

ACE inhibitors, both direct acting or receptor blockers, although the first line of drug for patients in heart failure due to any cause, are, however, contraindicated in pregnant females due to the risk of fetal toxicity associated with them [19]. They, however, should be used in all symptomatic patients in the postpartum period and are safe for the breastfed infant. Beta-blockers such as metoprolol decrease the heart rate, improve left ventricular diastolic function and protect against arrhythmias but are only used as a second line of treatment as their prolonged usage in the prenatal period is associated with low-birth-weight of the baby. However, their use is considered safe during lactation. Digoxin may be indicated in certain patients for its inotropic effect [20].

Although it is a safe drug to be used during pregnancy and puerperium, its plasma level needs to be strictly maintained in the therapeutic range with close monitoring. Anticoagulation is recommended in patients with PPCM, especially if the ejection fraction is less than 35% and there are other associated risk factors such as severely dilated ventricles, atrial fibrillation, and presence of mural thrombus on echocardiography or history suggestive of previous thromboembolic episodes. The risk of venous thromboembolism is *per se* increased in pregnant patients and associated heart disease and bed rest (if advised for heart failure) may further increase the risk of development of this complication [21].

Warfarin is teratogenic in early pregnancy and can cause fetal warfarin syndrome, while intake in the second and third trimester may lead to fatal cerebral hemorrhage, microcephaly, blindness, deafness and growth return. Unfractionated heparin, on the other hand, has low bioavailability in pregnant patients and is associated with thrombocytopenia. Thus, low-molecular-weight heparins are preferred in pregnancy as they do not cross the placenta, have a lower risk of osteoporosis and thrombocytopenia and their bioavailability is more predictable.

In the postpartum period, thromboprophylaxis may be continued with warfarin if required as it appears in the breast milk in very insignificant quantities.

The choice of anesthetic technique is the anesthetist's prerogative and if the goals of hemodynamic management are adhered to, the outcome can be expected to be favorable of the anesthetic technique used. For any urgent or emergent lower segment cesarean section (LSCS), GA is preferred.

GA is also preferred in patients with borderline cardiac decompensation as an already dyspneic patient may not be amenable to the procedure of RA. In such a patient, even minor degrees of sympathetic blockade associated with RA may lead to fulminant cardiac failure. Another contraindication to RA is the anticoagulated patient. McCarroll *et al*. describe the cesarean section in a patient with PPCM under GA with use of remifentanil and propofol.

Remifentanil was chosen for its efficacy in controlling intraoperative stress response and rapid recovery independent of duration of infusion. They feel that the hemodynamic responses of the patient during a general anesthetic technique using appropriate agents are more predictable than those seen with RA. Similarly, Zangrillo *et al*. believe that potential benefits of cardiovascular effects of RA may not be greater than the risks of maternal hypotension and low cardiac output in such a patient.

Excessive reduction of preload may worsen cardiac output, while the decrease in afterload can actually jeopardize coronary perfusion in some patients. Opioid-based anesthesia provides good hemodynamic control and obtundation of response to endotracheal intubation but may require postoperative ventilatory support for both mother and neonate. Thus, it is obvious that favorable maternal and fetal outcome is not dependent on anesthetic technique, but strict hemodynamic control and meticulous cardiovascular monitoring with close coordination between various involved specialists. Use of other anesthetic drugs intraoperatively should be done with caution.

Ergometrine should preferably be avoided and oxytocin should be given as an infusion or slowly titrated to response. Autotransfusion after delivery can be countered by a small dose of furosemide just before delivery of the baby.

Anaesthetic Considerations

The anesthetic management of these patients can be challenging, given their reduced physiological reserve and potential detrimental effects on the baby. Any patient presenting with PPCM will almost certainly be reviewed by an anesthetist and may require emergency interventions. The anesthetic considerations can broadly be divided into pre-operative, operative and post-operative for cesarean sections. Even if the woman will undergo a vaginal (non-operative) delivery the same operative considerations are important [22].

Pre-operative considerations the signs and symptoms of PPCM can be subtle initially and be mistaken for normal changes in pregnancy such as leg swelling, shortness of breath, orthopnoea and paroxysmal nocturnal dyspnoea. These patients can have a first presentation of acute pulmonary edema and decompensated heart failure. A standard airway, breathing, and circulation (ABC) approach should be employed. If intubation is required, care must be taken as with any pregnant woman about awareness, maintenance of perfusion pressure to the placenta and risk of difficult airway [23].

Bridging with non-invasive positive pressure (continuous

positive pressure (CPAP) or rarely pressure support ventilation) is usually effective and obviates the need for intubation unless patients are in extremis. The management is primarily diuresis with oral and intravenous loop diuretics and vasodilation with nitrates to reduce pre and afterload. If these measures fail, inotropes and mechanical assist devices could be used. It is important when managing these patients in the acute setting to consider a wide differential diagnosis, as PPCM is essentially a diagnosis of exclusion [24].

Conditions such as an acute ischaemic event, aortic dissection, viral myocarditis and renal pathologies such as renal artery stenosis should be ruled out. Immediate investigations should include an ECG (to look for ischemia), blood tests including troponin and Brain Natriuretic Peptide (NT-BNP) and renal function, chest x-ray (to rule out chest infections) and echocardiogram [25]. Urgent further investigations would include cardiac catheterization to look for ischaemic heart disease and cardiac MRI to look for infiltrative disease and an accurate ejection fraction. Other investigations can include cardiac biopsies. An HIV test is important and should be repeated in any patient presenting with a cardiomyopathy in pregnancy (despite a negative test at booking) [26].

These patients may need optimization with inotropic agents such as dobutamine or milrinone. These should ideally be started with cardiac output monitoring. Patients should be managed in a high dependency or intensive care setting, with continuous ECG, saturation and invasive blood pressure monitoring. As with any complicated obstetric patient, a multidisciplinary approach with involvement from consultant midwives, obstetricians, anesthetists, critical care doctors, and cardiologists is vital. Daily review by specialist cardiology teams is important to provide advice to the obstetricians as to the urgency of delivery (which may reduce cardiovascular strain) [27].

Results and Discussions

Peripartum cardiomyopathy (PPCM) is a form of dilated cardiomyopathy which presents with the signs and symptoms of heart failure late in the third trimester of pregnancy, up to 5 months postpartum [28]. For a diagnosis of PPCM, there should be no other cause for cardiac failure such as viral illness or ischaemic heart disease. The echocardiographic criteria include an Ejection Fraction (EF) 2.7cm/m2. Although PPCM is a rare disease, affecting 0.1% of all pregnancies, it has a morbidity and mortality rate of up to one third [29]. Sliwa *et al*. looked at the incidence of PPCM in 2010. They reported a relatively equal distribution between the Caucasian and black populations. This is in contrast to previous studies which indicated a greater incidence in black patients, especially from Africa [30].

The main associations with PPCM were multiparity (>3 children), twin pregnancies and preeclampsia (and to a lesser extent other hypertensive diseases). Maternal age (>30 years old) was also a significant risk factor [31]. Infection with Human Immunodeficiency Virus (HIV) seems to have a strong association with PPCM, especially in patients outside of the European Union. An exact etiology has not been found for this heterogeneous condition. This is likely to be due to its multifactorial nature, with a definition that is confined more to a specific time period for diagnosis than a specific pathophysiology. The underlying mechanism seems to be oxidative stress with decreased angiogenesis [32].

Oxidative stress may be perpetuated by hypertension, abnormal hemodynamic stress response, potential viral etiology, and nutritional deficiency. There may be an immunologically mediated mechanism with sensitization of maternal antibodies to fetal cells. These may sensitize maternal antibodies to myocardial epitopes, which would explain the presence of antibodies to cardiac myosin heavy chains in some PPCM patients. Prolactin metabolism may also contribute to the disturbance of cardiomyocyte angiogenesis leading to cardiac failure [33].

Given the rare nature of this disorder and the complicating factor of pregnancy, there are no clinical trials to inform the optimum management of these patients. The treatment is that of acute severe decompensated cardiac failure. This involves protection of the airway and maintenance of oxygenation with positive pressure ventilation. Loop diuretics are safe in the peripartum period and used to diverse patients [34]. Both the preload and afterload are reduced with continuous infusions of nitrates such as glyceryl trinitrate (GTN) or sodium nitroprusside (which needs to be used carefully due to the production of cyanide molecules). Some patients may require inotropic support which can either be pharmacological (such as Milrinone or dobutamine) or mechanical (left ventricular assist devices, aortic balloon pumps or veno-arterial extracorporeal membrane oxygenation) [35].

It is important to recognize the increased thromboembolic incidence in patients with an ejection fraction of <35%. They can form left ventricular thrombi, and so should be on low molecular weight heparin whilst pregnant and warfarin following delivery [36]. The hemodynamic strain placed on the mother during pregnancy is well recognized. With the increased intravascular volume, compression of pelvic veins, increased placental vascular bed and blood requirements of the gravid uterus, cardiac failure can be exacerbated. There is an argument for early delivery of the baby. [37] This is somewhat less problematic for the fetus since this condition manifests in the last month of pregnancy so the fetus should be viable. [38] In patients with refractory PPCM, there is some evidence for women to stop breastfeeding their children and starting them on Bromocriptine to reduce serum prolactin levels. Stopping breastfeeding and treatment with bromocriptine for even 1 week improves morbidity and mortality [39].

This study found a 20% improvement in left ventricular systolic function after a week. It should, however, be noted that no placebo was used in this study, rather it was compared to another study where the drug was not given to the patients.

Reference

1. Hilfiker Kleiner D, Haghikia A, Nonhoff J, Bauersachs J Peripartum cardiomyopathy: current management and future perspectives. Eur Heart J. 2015; 36(18):1090-1097.
2. Tidswell M. Peripartum cardiomyopathy. Crit Care Clin. 2004; 20(2):777-788.
3. Lewis NL, Dob DP, Yentis SM. UK registry of high-risk obstetric anaesthesia arrhythmias, cardiomyopathy, aortic stenosis, transposition of the great arteries and Marfan's syndrome. Int J Obstet Anesth. 2003; 12:28-34
4. Pitton MA, Petolillo M, Munegato E, Ciccarese AA, Visentin S, Paternoster DM *et al* Hypertrophic obstructive cardiomyopathy and pregnancy anesthesiological observations and clinical series. Minerva Anestesiol. 2007; 73:313-8.
5. Maron BJ, Niimura H, Casey SA. Development of left ventricular hypertrophy in adults in hypertrophic cardiomyopathy caused by cardiac myosin-binding protein C gene mutations. J Am Coll Cardiol. 2001; 38:315-21.
6. Tidswell M Peripartum cardiomyopathy. Crit Care Clin. 2004; 20(2):777-788.
7. Tessler MJ, Hudson R, Naugler-Colville M, Biehl DR.

Pulmonary oedema in two parturients with hypertrophic obstructive cardiomyopathy (HOCM). Can J Anaesth 1990; 37:469-73.
8. Pryn A, Bryden F, Reeve W, Young S, Patrick A, McGrady EM *et al* Cardiomyopathy in pregnancy and caesarean section: Four case reports. Int J Obstet Anesth. 2007; 16:68-73.
9. Geisterfer-Lowrance AA, Kass S, Tanigawa G. A molecular basis for familial hypertrophic cardiomyopathy: A beta cardiac myosin heavy chain gene missense mutation. Cell. 1990; 62:999-1006.
10. Thaman R, Varnava A, Hamid MS. Pregnancy related complications in women with hypertrophic cardiomyopathy. Heart. 2003; 89:752-6.
11. Niimura H, Bachinski LL, Sangwatanaroj S. Mutations in the gene for cardiac myosin-binding protein C and late-onset familial hypertrophic cardiomyopathy. N Engl J Med. 1998; 338:1248-57.
12. Tidswell M Peripartum cardiomyopathy. Crit Care Clin. 2004; 20(2):777-788.
13. Maron BJ, Niimura H, Casey SA. Development of left ventricular hypertrophy in adults in hypertrophic cardiomyopathy caused by cardiac myosin-binding protein C gene mutations. J Am Coll Cardiol. 2001; 38:315-21.
14. Maron BJ, Niimura H, Casey SA. Development of left ventricular hypertrophy in adults in hypertrophic cardiomyopathy caused by cardiac myosin-binding protein C gene mutations. J Am Coll Cardiol. 2001; 38:315-21.
15. Tidswell M. Peripartum cardiomyopathy. Crit Care Clin. 2004; 20(2):777-788.
16. Secher NJ, Thayssen P, Arnsbo P, Olsen J. Effect of prostaglandin E2 and F2alpha on the systemic and pulmonary circulation in pregnant anesthetized women. Acta Obstet Gynecol Scand. 1982; 61:213-8.
17. Alegria J, Nishimura RA. Hypertrophic cardiomyopathy and pregnancy. In Oakley C, Warnes CA, editors. Heart Disease in Pregnancy. Malden, MA: Blackwell Publishing, 2007, 173-85.
18. Tidswell M. Peripartum cardiomyopathy. Crit Care Clin. 2004; 20(2):777-788.
19. Gunderson EP, Croen LA, Chiang V, Yoshida CK, Walton D. Epidemiology of peripartum cardiomyopathy: Incidence, predictors, and outcomes. Obstet Gynecol. 2011; 118(3):583-591.
20. Tidswell M. Peripartum cardiomyopathy. Crit Care Clin. 2004; 20(2):777-788.
21. Gunderson EP, Croen LA, Chiang V, Yoshida CK, Walton D. Epidemiology of peripartum cardiomyopathy: incidence, predictors, and outcomes. Obstet Gynecol. 2011; 118(3):583-591.
22. Tidswell M. Peripartum cardiomyopathy. Crit Care Clin. 2004; 20(2):777-788.
23. Gunderson EP, Croen LA, Chiang V, Yoshida CK, Walton D. Epidemiology of peripartum cardiomyopathy: Incidence, predictors, and outcomes. Obstet Gynecol. 2011; 118(3):583-591.
24. Elkayam U, Akhter MW, Singh H, Khan S, Bitar F. Pregnancy-associated cardiomyopathy: Clinical characteristics and a comparison between early and late presentation. Circulation. 2005; 111(16):2050-2055.
25. Sliwa K, Mebazaa A, Hilfiker KD, Petrie MC, Maggioni AP. Clinical characteristics of patients from the worldwide registry on peripartum cardiomyopathy (PPCM): EUR Observational Research Programme in conjunction with the Heart Failure Association of the European Society of Cardiology Study Group on PPCM. Eur J Heart Fail. 2017; 19(9):1131-1141.
26. Tidswell M. Peripartum cardiomyopathy. Crit Care Clin. 2004; 20(2):777-788.
27. Coyle JL, Jensen L, Sobey A. American College of Cardiology Foundation; American Heart Association. Peripartum cardiomyopathy: review and practice guidelines. Am J Crit Care. 2012; 21(2):89-98.
28. Pryn A, Bryden F, Reeve W, Young S, Patrick A, McGrady EM *et al* Cardiomyopathy in pregnancy and caesarean section: Four case reports. Int J Obstet Anesth. 2007; 16:68-73.
29. Kaufman I, Bondy R, Benjamin A. Peripartum cardiomyopathy and thromboembolism; Anaesthetic management and clinical course of an obese, diabetic patient. Can J Anaesth. 2003; 50:161-5.
30. McCarroll CP, Paxton LD, Elliott P, Wilson DB. Use of remifentanil in a patient with peripartum cardiomyopathy requiring caesarean section. Br J Anaesth. 2001; 86:135-8
31. Patten IS, Rana S, Shahul S, Glenn C. Rowe, Cholsoon Jang *et al*. Cardiac angiogenic imb
32. Coyle JL, Jensen L, Sobey A. American College of Cardiology Foundation; American Heart Association. Peripartum cardiomyopathy: review and practice guidelines. Am J Crit Care. 2012; 21(2):89-98.
33. Gupta D, Wenger NK. Peripartum cardiomyopathy: Status 2018. Clin Cardiol. 2018; 41(2):217-219.
34. Lapaire O, Hosli I, Zanetti DR, Huang D, Jaeggi C. Impact of fetal-maternal microchimerism on women's health-a review. J Matern Fetal Neonatal Med. 2007; 20(1):1-5.
35. Patten IS, Rana S, Shahul S, Glenn C. Rowe, Cholsoon Jang *et al*. Cardiac angiogenic imb, 2012.
36. Lata I, Gupta R, Sahu S, Singh H. Emergency management of decompensated peripartum cardiomyopathy. J Emerg Trauma Shock. 2009; 2(2):124-128.
37. Safirstein JG, Ro AS, Grandhi S, Wang L, Fett JD. Predictors of left ventricular recovery in a cohort of peripartum cardiomyopathy patients recruited via the internet. Int J Cardiol. 2012; 154(1):27-31.
38. Biteker M, Ilhan E, Biteker G, Duman D, Bozkurt B. Delayed recovery in peripartum cardiomyopathy: An indication for long-term follow-up and sustained therapy. Eur J Heart Fail. 2012; 14(8):895-890.
39. Wang J, Han Z, Zou Y, Wang N. Anaesthetic management of a pregnant patient with acute pulmonary oedema and peripartum cardiomyopathy: A case report. Anaesth J E M. 2017; 24(2):155-158.
40. Kaufman I, Bondy R, Benjamin A. Peripartum cardiomyopathy and thromboembolism; Anesthetic management and clinical course of an obese, diabetic patient. Can J Anaesth. 2003; 50(2):161-165.