Sys Rev Pharm 2020;11(12):1093-1101
A multifaceted review journal in the field of pharmacy

Updates in the Perioperative Anesthetic Management of Cardiomyopathy in Pregnant Patients

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ABSTRACT

This study aimed to identify the management of anesthesia in patients with PPCM include: Avoiding drug-induced myocardial depression, sudden alterations in heart rate, sudden decreases in afterload and systemic vascular resistance with maintenance of normal sinus rhythm and preload. And aimed to identify 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 Results: Peripartum cardiomyopathy (PPCM) is a potentially life-threatening pregnancy-associated disease that typically arises in the peripartum period the disease is relatively uncommon, its incidence is rising. It is a form of idiopathic dilated cardiomyopathy, defined as pregnancyrelated left ventricular dysfunction 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

Keywords: cardioimyopathy-LBV-CSF-CAD-AMI

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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 pregnancyrelated 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

Aim of work

- To summarize the current state of knowledge with regard to diagnosis, treatment, and management, with a focus on long term implications.
- To identify the management of anesthesia in patients with PPCM
- Discuss the Avoiding drug-induced myocardial depression, sudden alterations in heart rate, sudden decreases in afterload
- To identify Types of To identify Types of ystemic vascular resistance with maintenance of normal sinus rhythm and preload

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 Peripartum cardiomyopathy (PPCM) was first reported in the year 1849. Till the middle of 20 th century, it was known as postpartum cardiomyopathy ,as most of the cases reported had symptomatic onset in the postpartum period only. Demakis et al. 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 by Demakis et al. They described the data about 27 patients who presented in the late pregnancy or early puerperium with heart failure. Included ~ 103 ~ 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.

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.

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% 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 based on the work by Demakis et al.

CORE OF RESEARCH

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/m2 of body surface area.

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.

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.

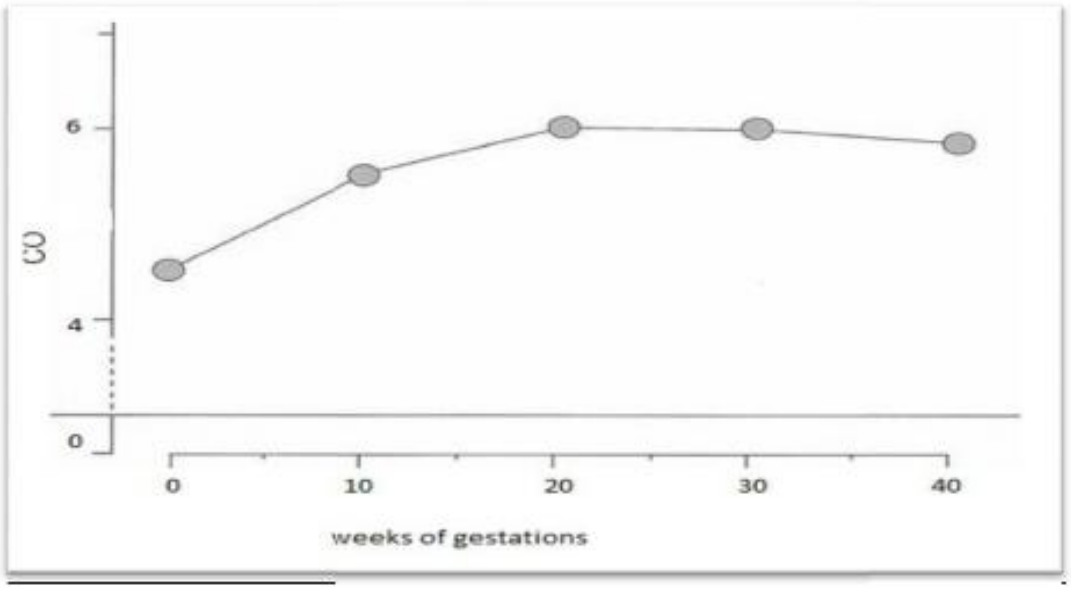

Figure 1-1; Cardiac output increases during the first trimester of pregnancy, remaining elevated throughout gestation

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.

There is a catecholamineinduced 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 postpartum. 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

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. 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 . 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, 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

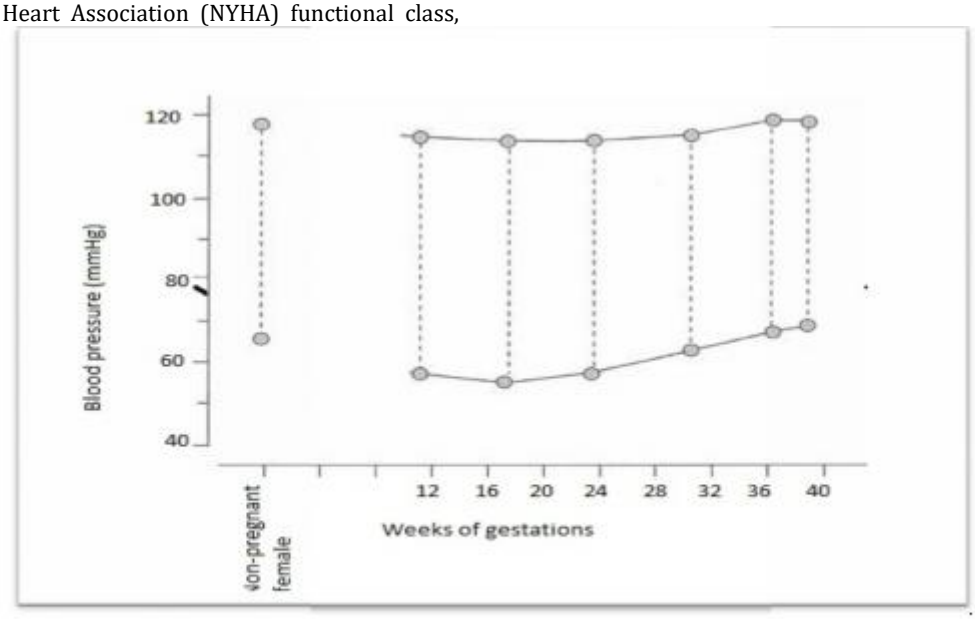

Figure 1-2; Blood pressure changes throughout pregnancy, falling during the first trimester, un l approximately 20 weeks gesta on, increasing towards or slightly abov normal levels at 40 weeks gesta on.

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 .

Poor functional status has been previously identified to be associated with maternal or fetal complications 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

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 preeclampsia to control blood pressure

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

They, however, should be used in all symptomatic patients in the postpartum period and are safe for the breastfed infant. Betablockers 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.

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 may further increase the risk of development of this complication

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-molecularweight 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. 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. Bridging with non-invasive

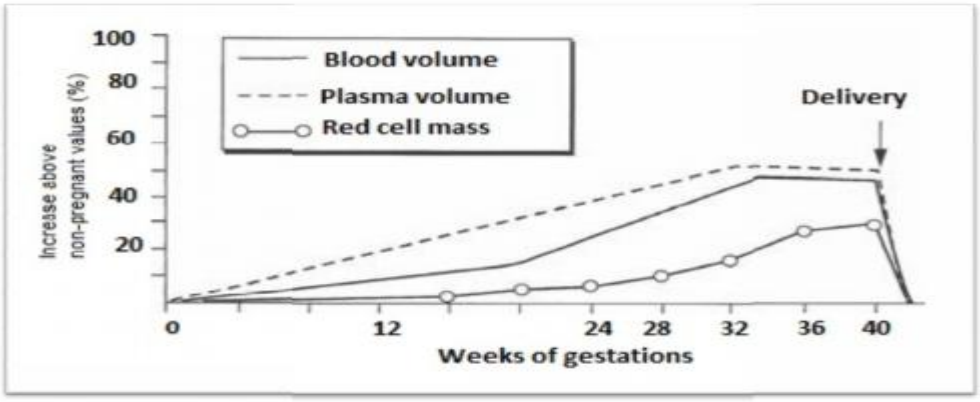

Figure 1-3; Increase in volume of constituents of blood during pregnancy, indicating that the largest component is expansion of plasma volume.

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 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

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)

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)

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

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. Sliwa et al. looked at the incidence of PPCM in 2010.

Prolactin metabolism may also contribute to the disturbance of cardiomyocyte angiogenesis leading to cardiac failure

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

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)

It is important to recognize the increased thromboembolic incidence in patients with an ejection fraction of

5 Case in Baghdad HPS[

a Case 1 A 29-year-old gravida (G) 4, para (P) 3, live (L) 3, at 36 weeks of gestation was referred with shortness of breath (SOB). At the time of admission, the patient was acutely dyspneic and hypoxic. She was afebrile and had a blood pressure (BP) of 156/88 mm Hg, a pulse rate (PR) of 154 beats/min, a respiratory rate (RR) of 45 breaths/min, and an oxygen saturation of 75% on oxygen 5 L with facemask. On auscultation, she had bilateral coarse crepitations. She was admitted to the intensive care unit (ICU), with a diagnosis of acute pulmonary edema. Her medical history revealed history of PPCM in the previous pregnancy on second postoperative day (POD) after lower-segment cesarean section (LSCS). On admission a transthoracic two-dimensional (2D) echo was done, which revealed left atrial and LV dilatation, global LV hypokinesia, and EF of 25 to 30%. Electrocardiogram (ECG) showed sinus tachycardia. In view of acute pulmonary edema with respiratory failure, the patient was intubated with suxamethonium and connected to ventilator with positive end-expiratory pressure (PEEP) of 8 cm H2O. Intubation response was minimized with fentanyl 100 μg, midazolam 2 mg, Xylocard 80 mg, and careful titration of propofol. Fetal monitoring was done with cardiotocography (CTG).

Termination of pregnancy by LSCS after initial maternal stabilization was decided. Preoperative optimization was done with repeated boluses of frusemide, head-up position, 100% oxygen, and PEEP. Heart rate was controlled with guarded doses of intravenous (IV) metoprolol. Two hours after stabilization, the patient was taken up for emergency LSCS under general anesthesia. Intraoperative monitoring was standard for cardiac diseases of pregnancy and included pulse oximetry, continuous ECG, invasive BP monitoring, and right internal jugular vein (IJV) cannulation. Urine output was monitored with bladder catheterization.

The intraoperative management as per the management of cardiac diseases of pregnancy included restricted IV fluids, with noradrenaline and nitroglycerine (NTG) backup, exaggerated left uterine displacement (LUD), opioid fentanyl 100 μg IV after baby delivery, and frusemide 40 mg IV bolus after baby delivery. Oxytocics included oxytocin as infusions at 5 U/h and misoprostol 1,000 mg (prostaglandin E1 [PGE1]) rectal suppository. Prophylactic B-lynch compression sutures were applied to the uterus. Intraoperative vitals were maintained, and the patient was shifted to ICU for elective ventilation and extubated after 24 hours. Postoperative pain relief was given by IV infusion of fentanyl at 10 μg/h, with IV paracetamol every 6 hours. Postoperative care included head-up position, frusemide 40 mg IV 8th hourly, angiotensin-converting enzyme inhibitor (ACE-I) enalapril 2.5 mg once a day, oral Metoprolol 12.5 mg 12th hourly, and fluid restriction to 75 mL/h of crystalloid. Postoperative pulmonary complications (POPC) were prevented by nebulization, deep breathing exercises, chest physiotherapy, and deep vein thrombosis (DVT) prophylaxis with low-molecular-weight heparin (LMWH) enoxaparin 40 mg once in 24 hours. The patient had an uneventful recovery, and she and her baby were discharged in hemodynamically stable condition for further cardiologist's review and follow-up.

Case 2 A G2P1L1 at 37 weeks of gestation with previous LSCS was referred with acute onset of dyspnea in active labor. Her previous pregnancy was uneventful. The patient was a known asthmatic. At the time of presentation, she was tachypneic with an RR of 28

breaths/min. Oxygen saturation was 92% with 6 L of oxygen on facemask. Her noninvasive blood pressure (NIBP) was 120/80 and heart rate was 114 beats/min. On auscultation bilateral wheeze and basal crepitations were present. Two-dimensional echo showed global hypokinesia and 35% left ventricular ejection fraction (LVEF). ECG showed sinus tachycardia. Preoperative optimization was started with frusemide, ivabradine 5 mg for rate control, levo-salbutamol, and budesonide nebulizations for wheeze. As the patient was in active labor, after initial stabilization, emergency LSCS was done under epidural anesthesia with monitored incremental doses of local anesthetic with opioid fentanyl. Instead of contemplating a rapid-sequence induction in a pregnant asthmatic with wheeze, we preferred incremental epidural anesthesia, taking care not to cause hemodynamic compromise.

Intraoperative monitoring, fluid input, and oxytocic dosing were done as per our standard management of cardiac diseases of pregnancy. The patient was shifted to ICU in hemodynamically stable condition. Six hours after the surgery, she became dyspneic and started desaturating. Initially noninvasive ventilation (NIV) was tried, but the patient had to be intubated and noradrenaline support was started due to fall in the BP and she was referred to cardiology department for further management. The patient succumbed on third POD.

Case 3 A G2P1L1 with term gestation was referred in view of previous history of PPCM detected on second POD of previous pregnancy. At the time of presentation, the patient was dyspneic with an RR of 20 breaths/min, BP of 140/90 mm Hg, and SpO2 of 94% on room air. On auscultation, there were decreased breath sounds bilaterally. Two-dimensional echo showed global hypokinesia and 35 to 40% LVEF. Trivial mitral regurgitation (MR) and trivial aortic regurgitation (AR) were present. ECG showed sinus tachycardia. Preoperative optimization was done with frusemide and ivabradine 5 mg once in 24 hours. After optimization and stabilization of the condition, the patient was posted for elective LSCS under incremental epidural anesthesia. Intraoperative cardiac monitoring was done. Both intraand postoperative periods were uneventful.

Case 4 A G2P1L1 at 37 weeks of gestation with breech was referred in view of PPCM. PPCM was detected when the patient was evaluated for dyspnea and easy fatigability. Two-dimensional echo showed global akinesia, 40% LVEF, and mild MR. ECG showed sinus tachycardia. The patient was started on ivabradine 5 mg for rate control and spironolactone. Elective LSCS was done under incremental epidural anesthesia. Both intra- and postoperative periods were uneventful.

Case 5 A G3P2L2D1 with term gestation with previous history of PPCM with severe preeclampsia, hemolysis, elevated liver enzymes low platelet (HEELP) syndrome, acute kidney injury (AKI), severe LV dysfunction, and PPCM was referred for institutional delivery.

At the time of presentation, the patient was physically and hemodynamically stable with no symptoms of PPCM or HF. Her laboratory parameters in the present pregnancy were normal. However, 2D echo showed global hypokinesia and LVEF less than 45%. LSCS was done under incremental epidural anesthesia uneventfully. Postoperatively the patient was monitored in ICU and had an uneventful recovery. Observation All the five patients presented in the third trimester. The chief presenting complaint was SOB (►Table 1). Preoperative investigations included complete blood count, liver function tests, renal function tests, complete urine examination, thyroid profile, and transthoracic echocardiograph. The baseline blood investigations were normal in all the five cases. The risk stratification was done taking into consideration the American Society of anesthesiologists (ASA) grading,5 New York Heart Association (NYHA) class,6 Cardiac Disease in Pregnancy (CARPREG) score, and World Health Organization (WHO) classification of cardiac disease in pregnancy.7 All the five cases were high risk with ASA grade III to IV E and NYHA grade III to IV. WHO and CARPREG scores too were suggestive of increased risk of major adverse cardiac events (MACE) (►Table 2).

Anesthetic management was done by incremental epidural dosage in four out of the five cases, whereas one required mechanical ventilation for acute pulmonary edema and was continued as general anesthesia for cesarean delivery. Post-delivery, the patients were started on medications for HF titrated to the individual needs. All the patients were started on ACE-I enalapril 2.5 to 5 mg, sustained-release metoprolol or carvedilol or ivabradine 5 mg for rate control, and a diuretic spironolactone 25 mg and/or frusemide. POPCs were prevented by head-up position, nebulization, deep breathing exercises, chest physiotherapy, and DVT prophylaxis with LMWH enoxaparin 40 mg once in 24 hours. Early liquid diet was resumed in the last three cases

Table 1 Condition at the time of admission

Case number	Pregnancy status	Previous PPCM	Presenting complaint	PR (beats/min)	BP (mm Hg)	RR (breaths/min)	Lungs	Present diagnosis
1	G4P3L3 36 wk	Previous LSCS Postpartum PPCM	SOB	155–165	140/90	38–40	Bilateral coarse crepitations	APO PPCM
2	G2P1L1 37 wk	Previous LSCS	SOB	94	120/80	28	Bilateral diffuse wheeze	PPCM
3	G2P1L1 38 wk	Previous LSCS Postpartum PPCM	SOB, easy fatigability	110	140/90	28	Decreased air entry in the bases	PPCM with early decompensation
4	G2P1L1 38 wk breech	PPCM now Previous LSCS	SOB, easy fatigability	124	110/80	30	BAEE	PPCM
5	G3P2L2D1 38 wk	Postpartum PPCM Previous LSCS	Institutional delivery	86	140/90	18	BAEE	PPCM

Table 2 Risk stratification

Case number	ASA	NYHA	WHO	CARPREG		EF (%)	Morbidity/Mortality
				Score	MACE (%)		
1	IV E	IV	IV	> 1	75	25–30	Pulmonary edema, mechanical ventilation, emergency LSCS, ICU stay
2	I–II E	III	IV	> 1	75	35	Wheeze, emergency LSCS, postpartum heart failure, mechanical ventilation, shifted to cardiology department; succumbed on 2nd POD
3	III	III	IV	> 1	75	35–40	ICU admission and optimization
4	III	II–III	II–III	1	27	40	ICU admission and optimization
5	III	II	IV	1	27	< 45	Regular antenatal care, cardiologist follow-up, anesthetic evaluation and plan

RESULTS

Peripartum cardiomyopathy may present as HF in pregnant women. Viral infections, selenium deficiency, autoimmunity, and genetic predisposition are some of the factors predisposing to PPCM.8 Advanced maternal age, multiple gestation, chronic hypertension, and assisted reproductive techniques may contribute to the increased incidence. Recently the role of Fms-like tyrosine kinase 1 (sFlt-1) and the hormone prolactin in PPCM is being evaluated.8 Awareness of the condition, early echocardiography, availability of obstetric anesthesia, and critical care services improved the outcome in this rare condition. Typically, patients become symptomatic after 36weeks of gestation or within the first month of delivery.9 Worldwide registry on PPCM reported that 21% of women with PPCM had cardiomyopathy in a previous pregnancy.3 The presenting symptom in most patients is dyspnea on exertion (NYHA-III or IV). Some may present with complex arrhythmias or peripheral or pulmonary embolic episodes or cardiac arrest. For the evaluation of PPCM, apart from ECG and echo, complete blood cell count, blood urea, creatinine, electrolytes, liver function test, and thyroid-stimulating hormone should be done. Echo findings consistent with PPCM include a decreased ejection fraction, global dilatation, and thinned-out cardiac walls.10 Brain natriuretic peptide (BNP) or N-terminal pro-brain natriuretic peptide (NTproBNP) levels have high sensitivity in HF. NTproBNP levels of 100 pg/mL or less have a high negative predictive value for adverse maternal events and can be helpful to screen potential PPCM. Cardiac magnetic resonance imaging can be a complementary tool in the diagnosis and further evaluation of PPCM women.

In this case series, four out of the five patients presented with dyspnea after 36 weeks of gestation. Four out of the five patients had a history of previous PPCM. All the five PPCM cases in this case series had an EF of less than 45%. Management of PPCM is the same as that of cardiac failure of other etiologies. Acute management is the management of airway, breathing, and circulation (ABCs). Acute decompensation presenting as acute pulmonary edema adds to the difficult airway in pregnant women. The decreased functional residual capacity (FRC) and the increased oxygen demand in pregnancy can lead to rapid desaturation, hypoxia, and maternal acidosis. Maternal desaturation can lead to fetal compromise, putting both lives at peril. Hence oxygen supplementation is of paramount importance. Although a relative contraindication in pregnancy, NIV, when used judiciously, can obviate the need for endotracheal intubation. Hypoxia, respiratory failure, and hypotension mandate endotracheal intubation and mechanical ventilator support. Ventricular off-loading can be done by IV loop diuretics such as frusemide and vasodilation by NTG infusion at 5 to 10 µg/min, titrated to the clinical condition and BP. Antenatal mothers with poor cardiac output and hypoperfusion will be benefited with exaggerated LUD to alleviate the aortocaval compression and may need an inotropic support. Patients with relative hypotension, who do not respond to vasodilators and diuretics, also benefit from the inotropic support. Inotropic agents improve cardiac contractility and cardiac output, increase renal perfusion and facilitate diuresis, preserve end-organ function, and promote clinical stability.

Urine output monitored every hour acts as a surrogate for cardiac output monitoring. Those in refractory shock may benefit from temporary circulatory support with either intra-aortic balloon counter-pulsation or extracorporeal membrane oxygenation.

Implantation of a left ventricular assist device (LVAD) is also described in PPCM either as a bridge to recovery or transplantation. Fetal heart rate monitoring with CTG or ultrasound establishes the fetal well-being. Stabilization of the mother's condition is crucial than resorting to emergency cesarean delivery, which carries a higher maternal morbidity and mortality. Maternal stabilization can prevent or revert the fetal compromise. Pregnant women with PPCM and an EF of less than 35% are prone for LV thrombus and need thromboprophylaxis with LMWH. ACE-I can be given postpartum. β-Blockers are protective against tachyarrhythmias and can improve survival. Bromocriptine, 2.5 mg, 7 to 14 days showed promising results in PPCM.15 Cabergoline, a potent dopamine receptor antagonist, has long half-life of 14 to 21 days and may be given as a single dose. Approximately 14% of PPCM women may need cardiac transplantation. Anesthetic Considerations Anesthetic management of the PPCM patient includes preoperative optimization, intraoperative management, and postoperative stabilization. Both general and regional anesthesia can be given. Anesthetic technique depends on the urgency of delivery and the patient's hemodynamic stability. Vaginal delivery may be allowed with effective labor analgesia. Labor epidural analgesia decreases the sympathetic stimulation and decreases plasma catecholamine levels in the mother. The second stage of labor should be cut short with assisted vaginal delivery (forceps or vacuum), to reduce workload on the myocardium. The anesthetic goal is to optimize cardiac output by maintaining the preload and decreasing the afterload. Myocardial contractility should be maintained by avoiding drugs that can decrease the already compromised cardiac contractility. Subarachnoid block and rapid induction of general anesthesia can result in precipitous fall in systemic vascular resistance and should be avoided. Incremental epidural dosing has been used in patients posted for nonemergent cesarean section with relatively stable hemodynamic status.18 General anesthesia is preferred in category 1 cesarean sections, in conditions of severe cardiac decompensation where even minor sympathetic blockade may lead to fulminant cardiac failure, and in anticoagulated patients. The associated problems are the rapid fall in the systemic vascular resistance, intrinsic difficult airway associated with pregnancy, and laryngosympathetic response during intubation and extubation.19 Undue reduction in the preload may worsen the fall in the cardiac output, whereas the decrease in afterload can compromise the coronary perfusion. Opioid-based cardiac induction provides good hemodynamic control and suppresses the laryngosympathetic response. Opioid induction has to be used judiciously as it may not be ideal for rapid-sequence induction and cause neonatal respiratory depression. Remifentanil infusion can alleviate some of these problems.20 Regional anesthesia techniques can be a better alternative. Incremental epidural, combined spinal epidural (CSE)21 and continuous spinal

anesthesia (CSA) have been used in PPCM patients.22 In patients on anticoagulant therapy, insertion and removal of neuraxial catheter should be planned according to the timing of anticoagulation administration. The advantage of incremental epidural is that it gives freedom of titrating the local anesthetic dose to achieve the desired level of motor and sensory block without causing sudden hypotension and decompensation in these patients. Regional anesthesia can decrease the cardiac preload and afterload and lead to an improvement in cardiac function. There is decrease in POPCs with regional techniques. A working epidural gives good postoperative pain relief. Left uterine displacement is advocated to avoid aortocaval compression. Prophylactic infusion of phenylephrine can be used to maintain BPs without causing tachycardia. IV fluids should be titrated to maintain the hemodynamic stability without excessive preloading. Oxytocic drugs should be used with caution. Bolus or rapid infusion of oxytocin can cause a marked decrease in systemic vascular resistance, and in higher doses, it has an antidiuretic effect. Ergometrine should not be used as it causes significant increases in afterload and coronary vasospasm. Intramuscular injection of 15 methyl prostaglandin F2 alpha can be given but should be avoided in reactive airways. PGE1 can be used as rectal suppository or sublingual dose. It can cause increased temperature and shivering. Postpartum care should be in the ICU. The intraoperative cardiac monitoring should be continued in the postoperative period. Analgesia can be provided by epidural catheter or IV opioids or by transversus abdominis plane (TAP) block. Postoperative care includes spironolactone, ACE-I, β-blocker, fluid restriction, input-output chart, POPC protocol, and DVT prophylaxis with LMWH. Early referral of patients with previous PPCM; multidisciplinary approach with early involvement of the obstetrician, cardiologist, and anesthetist; and planning of anesthesia and preoperative care can decrease the maternal mortality and morbidity in PPCM. Careful selection of anesthesia technique, meticulous hemodynamic monitoring, and slow and cautious titration of anesthetic drugs can improve the maternal and fetal outcome in this rare cardiomyopathy.

CONCLUSIONS

- Peripartum cardiomyopathy (PPCM) is a potentially life-threatening pregnancy-associated disease that typically arises in the peripartum period
- the disease is relatively uncommon, its incidence is rising. It is a form of idiopathic dilated cardiomyopathy, defined as pregnancyrelated left ventricular dysfunction
- 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

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