INVITED ARTICLE

Critical Care Management of the Parturient with Cardiac Disease

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Abstract

Parturient with heart disease forms a challenging group of patients and requires specialized critical care support in the peripartum period. Maternal heart disease may remain undiagnosed till the second trimester of pregnancy, presenting frequently after 20 weeks of gestation, due to increased demands imposed on the cardiovascular system and pose a serious risk to the life of mother and fetus. Management of critically ill parturient with heart disease must be tailored according to individual assessment of the patient and requires a strategic, multidisciplinary, and protocol-based approach. A dedicated obstetric intensive care unit (ICU) and team effort are the need of the hour.

Keywords: Arrhythmias, Cardiac failure, Cardiac risk assessment, Cardiomyopathies, Congenital cardiac lesions, Critical care, Ischemic heart disease, Parturient, Pericarditis, Pulmonary hypertension, Valvular heart disease.

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Introduction

Cardiac disease in a parturient is one of the most important causes of maternal morbidity and mortality. It can result in both planned and unplanned intensive care unit (ICU) admissions, which can be either pre- or postdelivery or even after obstetric-related surgeries.[1] Cardiac disease complicates 1–4% of all pregnancies and accounts for 10–15% of maternal mortality in the western world.[2] Peripartum heart disease has a wide variation, ranging from pregnancy-induced hypertension (PIH), valvular heart disease (VHD), congenital heart disease (CHD), myocardial infarction (MI), cardiomyopathies, aortic dissection, pulmonary hypertension (PH), cardiac failure, etc. Especially in developing countries, with a lack of universal antenatal care (ANC), these patients may present late, as the demand on the cardiovascular system increases with the progression of pregnancy or in the peripartum period. The outcome in such cases depends on the cardiac pathology, ventricular function, functional status of the mother, degree of cyanosis, the status of the pulmonary vasculature, concurrent maternal diseases, prior cardiac surgical interventions, and prosthesis.

There are four indicators for risk assessment: (a) functional status, (b) left heart dysfunction, (c) prior cardiac events, and (d) left heart obstruction. As more and more women are postponing their pregnancies into the later parts of their reproductive age, the incidence of acquired heart diseases (e.g., hypertension, cardiomyopathy, and ischemic heart disease) is also increasing.[3] The earlier school of thought that pregnancy is contraindicated in the presence of severe cardiac disease is no longer applicable. For better feto-maternal outcomes, dedicated cardiac obstetric critical care units must be envisaged and developed with multidisciplinary support. The members of the core care team should include an experienced obstetric anesthesiologist, cardiologist, cardiothoracic–vascular (CTVS) surgeon, obstetrician, neonatologist, and intensivist. Preconception counseling should be considered in all cases.

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Cardiac Physiology in Pregnancy and Puerperium

The physiological changes in pregnancy are seen in parturient (Table 1), and these are exaggerated in patients with cardiac disease, resulting in decompensations and complications leading to ICU admissions.[3–6] The parturients with cardiac diseases do not tolerate fluid shifts or compensatory physiological changes of pregnancy like an increase in blood volume.

The major concerns related to physiologic changes in a parturient responsible for causing problems in cardiac disease are[4] as follows:

- Increase in intravascular volume: It is poorly tolerated in those with valvular heart disease, coronary artery disease, or cardiomyopathies, leading to congestive cardiac failure, myocardial ischemia, or aortic dissection due to volume overload. Marked fluctuations in cardiac output and autotransfusion

Table 1: Physiological changes of concern in a parturient with cardiac disease

Sl. No.	*Maternal cardiac parameter*	*Physiologic change*
1	Uterine blood flow	7–10% increase
2	Red blood cell mass	20–30% increase
3	Plasma volume	45–55% increase
4	Systemic vascular resistance	35% decrease in second trimester 20% less than baseline at term
5	Cardiac output	30% increase in second trimester 60% increase during labor Cardiac output increases immediately following delivery by 60–80%, due to uterine contraction and release of the aortocaval compression by the gravid uterus, rapidly declining to prelabor values 1 hour postpartum.
6	Heart rate	17% increase
7	Pulmonary vascular resistance	34% decrease
8	Colloid oncotic pressure (COP)	14% decrease
9	COP—pulmonary capillary wedge pressure (PCWP)	28% decrease
10	Mean arterial pressure (MAP), central venous pressure (CVP), Left ventricular stroke work index	No statistically significant change

during delivery are poorly tolerated in patients with pulmonary hypertension or fixed-output lesions.

- Decrease in systemic vascular resistance: This aggravates right-to-left shunts, worsening the cyanosis, which can be deleterious in patients with valvular and congenital heart disease.
- Hypercoagulable state: Increase in coagulation factors in pregnancy elevates the risk of arterial thrombosis, necessitating anticoagulant therapy, which can have adverse fetal effects (coumarin derivatives) or increase the chances of maternal hemorrhage.
- Systemic changes: Tachycardia, dilated neck veins, dynamic precordium, bounding pulses, third heart sound, systolic murmur, and peripheral edema.

The upregulation of nitric oxide (NO) synthesis by estradiol occurs in early gestation and leads to arterial vasodilation and reduced systemic vascular resistance (SVR) and PVR. Postpartum, hemodynamic changes may resolve by 6 weeks and cardiovascular changes by 12 weeks.

Common Cardiac Conditions in Parturient Requiring ICU Care

Various cardiac conditions in the parturient may be preexisting, before pregnancy, or maybe first detected during the pregnancy itself (Table 2).[7–10]

Cardiac failure is a dreaded complication of heart disease in pregnancy. As per the Registry on Pregnancy and Cardiac Disease (ROPAC) study, the following are the predictors for the development of heart failure during pregnancy leading to ICU admissions:[11]

- New York Heart Association (NYHA) class III or greater
- World Health Organization (WHO) risk classification of 3 or greater
- Prepregnancy heart failure
- Pulmonary hypertension
- Cardiomyopathy with reduced subaortic ventricular systolic function (EF <40%)

Various underlying pathologies need to be identified for the need of ICU admission (Table 3).[12] Acute heart failure during pregnancy is more common in the late second or third trimester or the immediate postpartum period. It can be precipitated or exaggerated by PIH or eclampsia; severe anemia; development of tachyarrhythmias, especially atrial fibrillation; sudden discontinuation of cardiac medications during pregnancy; and obstetric complications. The risk factors for the development of right heart failure during pregnancy include those with repaired right ventricular outflow obstruction, like Tetralogy of Fallot or pulmonary atresia.

Identification and Assessment of Critical Illness in the Parturient with Cardiac Disease

The early identification of the cardiac conditions and/or their deterioration is paramount for an optimal outcome in the parturient with cardiac diseases. Various clinical parameters may be used for the identification of decompensation (Table 4).[13]

Various risk assessment tools have been described for the parturient with heart diseases[14,15] (Table 5).

Other used tools for assessment includes Shock Index, Preeclampsia Integrated Estimate of RiSk (miniPIERS) or full PIERS model, obstetrically modified quick-sequential organ failure assessment score (omqSOFA), Maternal Early Warning Scoring system (MEWS), NYHA classification, and European Society of Cardiology (ESC) guidelines.

Principles of Critical Care Management in Parturients with Cardiac Disease

Critical care management of the cardiac parturient must be individualized and tailor-made to suit the requirements of the patient as per her clinical presentation and cardiac status. Alterations in feto-placental circulation and oxygenation due to maternal cardiovascular disturbances like shock can adversely

Table 2: Common cardiac conditions in parturients

Preexisting cardiac disease	*Exacerbation of preexisting cardiac disease*	*New-onset*
Rheumatic and nonrheumatic VHD	Arrhythmias, hemodynamic instability, cardiac failure, infective endocarditis, sudden death	Severe preeclampsia/eclampsia Peripartum cardiomyopathy Cardiac failure
Congenital heart disease (CHD)	Failure of oxygenation, arrhythmias, Eisenmenger syndrome, cardiac failure	
Pulmonary hypertension	Respiratory failure, cardiac failure	
Aortic dissection	Acute hemodynamic deterioration, death	
Cardiomyopathy	Cardiac failure, arrhythmias, hemodynamic compromise, death	
Coronary artery disease and vasospastic angina	Worsening of symptoms, arrhythmias, cardiac failure, sudden death	
Arrhythmias	Hemodynamic compromise, cardiac failure, sudden death	

Table 3: Reasons for ICU admission in obstetric patients

Pregnancy-related	*Aggravation of medical conditions*	*Conditions unrelated to pregnancy*
Eclampsia/severe pregnancy-induced hypertension	Rheumatic/nonrheumatic valvular heart disease	Trauma
Obstetric hemorrhage	Congenital heart disease	Asthma
Amniotic fluid embolism	Pulmonary hypertension	Diabetes
Peripartum cardiomyopathy	Anemia	Autoimmune disorders
Acute fatty liver	Renal failure	
Aspiration syndromes		
Infections		
Ovarian hyperstimulation syndrome (OHSS)		

Table 4: Parameters for identification of critical illness in the parturient with cardiac disease

Symptoms	Palpitations at rest, edema, dyspnea (progressive, paroxysmal nocturnal dyspnea, rest), chest pain (exertional or rest), syncope (exertional)	
Signs (Examination)	Bradycardia (heart rate <50/minute), tachycardia, raised jugular venous pressure (JVP), cardiomegaly, right ventricular heave, loud P2, gallop rhythm, loud systolic murmur (intensity >3), diastolic murmur, cyanosis, crepitations, persistent pedal edema (unresponsive to limb elevation)	
Investigations	Electrocardiogram (ECG), Holter, event monitor	Detect arrhythmias
	Doppler echocardiography	Assess VHD, pulmonary artery systolic pressures, cardiac function
	Transesophageal echocardiography (TEE)	Detect atrial thrombi, atrial septal defect, endocarditis, aortic dissection
	Stress testing	Assess severity of valve disease, detect ischemia, provoke arrhythmia
	Cardiac catheterization/angiography	Diagnose and treat coronary artery disease
	Cardiac magnetic resonance imaging (MRI)	Assess whole aorta, congenital heart disease, myocardial disease
Monitoring	Noninvasive	ECG, Holter, natriuretic peptide tests [blood levels of B-type natriuretic peptide, *BNP;* N-terminal (NT)-prohormone *BNP,* NT-proBNP], transthoracic echocardiography Doppler echo
	Invasive	Invasive blood pressure (IBP), central venous pressure (CVP), pulmonary artery catheter (PAC) derived values, TEE, esophageal Doppler

affect both the mother and the fetus (fetal distress, fetal demise, emergency operative deliveries, and preterm births). There are four major goals in the management of the critically ill parturient:[16]

- Optimization of maternal cardiac condition
- Early detection of cardiovascular deterioration
- Management of any complications
- Maintenance of fetal well-being

The routine obstetric principles of left uterine displacement to prevent supine hypotension syndrome and aspiration prophylaxis (with oral sodium citrate or injectable antacids) to prevent

Table 5: Risk assessment tools in the parturient with heart disease

Assessment tool	*Advantages*	*Limitations*
CARPREG	A prospective study including patients with known heart disease, primarily CHD Identified several independent predictors for the occurrence of an adverse maternal cardiac event during pregnancy	Overrepresentation of complex CHD, thus very high rate of serious complications
ZAHARA	Weighted scoring system for women with CHD, incorporated several other variables	High-risk lesions were underrepresented leading to inaccurate pregnancy risk prediction for sicker patients
Modified WHO cardiac risk assessment	Based on underlying heart disease, presence, and severity of ventricular and valvular dysfunction Pregnancy-related risks are additive, a patient with low-risk cardiac disease may become high risk because of other cardiac/noncardiac risk factors	

Mendelson syndrome must be routinely employed. In addition, the following general principles of critical care management can be considered in a cardiac parturient: maintenance of normoxia, normocarbia, normotension, and normothermia; deep vein thrombosis prophylaxis; cardiac and fetal monitoring; maintenance of acid–base and electrolyte balance; complete asepsis and antibiotic prophylaxis.

The initial assessment of a sick parturient is the same as nonpregnant patients. Airway, Breathing, and Circulation (ABC) are assessed, and attempts must be made to stabilize the patient.[17]

- Airway management: Oxygen supplementation, noninvasive ventilation, or invasive mechanical ventilation with endotracheal intubation should be instituted as per the patient's requirement. Lung-protective ventilation strategies should be followed with lower tidal volumes (3–6 mL/kg), higher respiratory rates, plateau pressures <28 cm H_2O, and judicious use of PEEP. Target partial pressure of carbon dioxide ($PaCO_2$) should be lower than nonpregnant patients and permissive hypercapnia is not advisable.
- Hemodynamic monitoring is to be instituted as soon as possible with invasive arterial blood pressure (IBP) and central venous catheter (CVC) to guide fluid administration and vasopressor/inotropic management since maintenance of uteroplacental circulation is crucial.
- Nutrition: Ketoacidosis may set in if prolonged starvation is allowed, therefore enteral feeding should be instituted as soon as possible.
- Sedation: Most of the commonly used agents cross the placenta and the neonatology team should be made aware of the drugs used in case the patient is going in for delivery/cesarean section.
- Thromboprophylaxis: Parturients are at four times higher risk of developing deep vein thrombosis compared to other critically ill patients. Unfractionated (UFH) or low molecular weight heparins (LMWH) can be instituted.

Ventilation Strategies in the Critically Ill Parturient with Heart Disease

Positive pressure ventilation (PPV) may be divided into noninvasive or invasive. NIPPV may be beneficial in a small fraction of patients with obstructive airway disease with good respiratory drive and fewer secretions.[18] However, the threshold for intubation should be kept low as parturients are at a high risk of aspiration. Airway edema can create a potentially difficult intubation scenario and readiness should be there with a difficult airway cart. Lung-protective ventilation strategies along with the use of sedation are recommended. Oxygen requirement is increased by 30–50 mL/minute in parturients, and a higher PaO_2 is required to maintain fetal oxygenation. Thus, partial pressure of oxygen (PaO_2) of 70 mm Hg and saturation of 95% are desirable in them; the higher fraction of inspired oxygen (FiO_2) and use of positive end-expiratory pressure (PEEP) may be essential.[19] $PaCO_2$ should be maintained between 40–45 mm Hg and pH 7.25–7.35 so that both fetal acidosis due to hypercarbia and uterine hypoperfusion due to respiratory alkalosis are avoided. Upward displacement of the diaphragm by the gravid uterus reduces chest wall compliance so slightly higher plateau pressures may be targeted to achieve oxygenation.[12]

Sepsis in the Parturient with Heart Disease

Sepsis in parturients leads to mortality in 15% of cases worldwide.[20] They develop sepsis consequent to mainly four conditions: chorioamnionitis, pyelonephritis, endometritis, and pneumonia.[21] A certain degree of immunosuppression may cause fungal or viral pneumonia also. Clinically, sepsis in pregnancy manifests as multi-organ failure and coagulopathy, which may be compounded by the presence of heart disease leading to a rapid downhill course. Early recognition of the initial signs of sepsis is very important, and this is not difficult as the patient may already be admitted to a ward or ICU setting. Modified early obstetric warning system (MEOWS)[22] and sepsis in obstetrics score (SOS) scoring system[23] are some methods to recognize the development of sepsis early. Rapid rise or fall in blood counts, deranged coagulation parameters (INR), and clinical signs of shock in a previously stable patient should raise the suspicion of developing sepsis. Immediate management according to the surviving sepsis guidelines[24] should be started in the meantime, while the source of infection should be identified and managed definitively, if possible. Displacement of the gravid uterus by placing a wedge under the right hip, invasive monitoring of vitals, CVP-guided fluid, fetal monitoring, and inotropic/vasopressor therapy should be initiated. Choice and combination of vasopressors can be tricky in heart disease with sepsis. Noradrenaline is the vasopressor of choice in sepsis; however, in the case of heart disease

with poor ventricular function or pulmonary arterial hypertension (PAH), it may prove detrimental. Combination with inodilator agents such as dobutamine/milrinone or levosimendan can help maintain cardiac contractility and prevent undue elevation of peripheral or pulmonary vascular resistance. Epinephrine may also be used as a vasopressor but due to its β receptor stimulant action, it can cause uterine relaxation and delay in the progression of labor. Corticosteroids, intravenous immunoglobulin is not recommended for use in sepsis in pregnant patients.[25]

Management of Specific Cardiac Conditions of Parturient in ICU

Each cardiac disease has specific management and therefore every parturient admitted to ICU with complications of the underlying heart disease requires a goal-directed, multidisciplinary approach.

Medications of pregnant women with chronic heart failure should be optimized before delivery.[26] Feto-toxic medications like angiotensin-converting enzyme (ACE) inhibitors, angiotensin receptor blockers, and aldosterone antagonists must be avoided and replaced with hydralazine and oral isosorbide dinitrate (afterload reducers), and beta-blockers can be continued. Those with pulmonary edema or right heart failure require judicious use of loop diuretics (furosemide), with fetal monitoring as it can reduce placental flow as well as maternal intravascular volume. Digoxin can be safely administered in pregnancy. Vasodilators (nitroprusside and nesiritide) are also avoided. Patients who do not respond to conventional medications may be given low-dose infusions of inotropes (dopamine or dobutamine). Oxytocin[27] given in the delivery period in cardiac disease parturient should only be administered by infusion, with avoidance of bolus dose (due to association with peripheral vasodilatation, tachycardia, and fluid retention). Ergometrine and carboprost should be avoided in these patients, as both have the propensity for causing or exacerbating pulmonary edema.

Pregnancy-induced Hypertension/ Preeclampsia

Pregnancy-induced hypertension (PIH) is defined as a hypertensive disorder of pregnancy along with proteinuria developing after 20 weeks of gestation and resolving within 6–12 weeks of delivery. It is classified as mild and severe, with severe form characterized by systolic blood pressure (SBP) >160 mm Hg, diastolic blood pressure (DBP) >110 mm Hg, proteinuria >5 g/dL, oliguria <400 mL/day, cerebral irritation, epigastric pain, and pulmonary edema. Eclampsia is a severe complication of PIH, characterized by the occurrence of seizures without other neurological disorders, progressing to hepatic failure, hemorrhage, and infarction. Hemolysis, elevated liver enzymes, and low platelets (HELLP) syndrome may occur around 37 weeks of gestation and overlap with preeclampsia. Severe PIH, eclampsia, and HELLP syndrome often need ICU admission due to pulmonary edema leading to respiratory distress.[21]

Oxygen therapy, ventilatory support, invasive ABP and CVP monitoring, goal-directed fluid and inotropic support (if required), and specific therapy for PIH/eclampsia (antihypertensive agents, magnesium sulfate infusion) were followed as advised by supervising obstetrician. Antibiotic therapy, nutrition, and thromboprophylaxis were suggested as per ICU standards. Arterial blood gas (ABG) and magnesium levels should be closely monitored.

Valvular Heart Disease, Infective Endocarditis, Mechanical Heart Valves

The parturient may have various underlying valvular heart diseases[28] and may have undergone previous cardiac surgeries for the same (Table 6).

In patients with mechanical heart valves, transesophageal echocardiography (TEE) is the perfect way to assess valve function and thrombus burden.[29] Pregnant women with mechanical heart valves require early counseling, close monitoring of peak anti-Xa levels, anticoagulant therapy surveillance, and watching out for thromboembolic complications. The patient on therapeutic warfarin ≤5 mg/day should be continued on warfarin throughout pregnancy (until before delivery). If warfarin requirement is >5 mg/day, then use LMWH (with strict monitoring) or give continuous intravenous UFH in the first trimester and switch over to warfarin in the second trimester. Low-dose aspirin (75–100 mg/day) should be given to all pregnant women with mechanical valves. These patients may require ICU admission for hemorrhagic complications or thromboembolism pre- and/or postdelivery. Valve thrombosis in a pregnant woman requires multidisciplinary consultations and a team approach in a tertiary care center with the involvement of the cardiac surgeon as well.

Cardiomyopathies

Various types of cardiomyopathies may be seen in parturient (Table 7).

Peripartum cardiomyopathy/Takotsubo cardiomyopathy: It is diagnosed based on three criteria—idiopathic, life-threatening cardiomyopathy, in patients without preexisting heart disease, manifesting 1 month before delivery to 5 months postpartum. It is characterized by the classical echocardiographic findings of global hypokinesia with LVEF <45% and left ventricular end-diastolic dimension >2.7 cm^2, leading to hemodynamic compromise and the need for ventilatory support. ECG may show ischemic changes but coronary angiography is normal.[30] It is seen in 0.1% of parturients, risk factors including multiparity, advanced maternal age, PIH, HELLP, smoking, and severe anemia.[31] Oxidative stress, autoimmune mechanism, inflammatory process, and myocarditis are some of the proposed etiologies. Most patients are present with symptoms of heart failure with pulmonary edema and low cardiac output leading to tissue hypoperfusion. Besides routine investigations, disease-specific biomarkers like prolactin, factors in the prolactin cleavage pathway, markers of heart failure (BNP, NT-pro-BNP) should be sent. In addition to the above-described changes, echo may also show moderate-to-severe mitral and tricuspid regurgitation. End-stage heart failure is seen in 10–23% of cases, while recovery to LVEF >50% occurs in 35–50% cases.[32]

Oxygen supplementation, initiation of treatment for heart failure (β blockers, diuretics, ACE inhibitors), inotropic support in severely low cardiac output, and persistent pulmonary edema, with adrenaline, dobutamine, milrinone, are the mainstays of ICU management. Patients with persistent cardiogenic shock may require hemodynamic support with intra-aortic balloon pump (IABP), extracorporeal membrane oxygenator (ECMO), and left ventricular assist device (LVAD) as the bridge to recovery or heart transplant. They should be weaned off inotropic support as soon as hemodynamic stability and improvement in EF are observed.[33]

Table 6: Valvular heart diseases (VHD) and infective endocarditis (IE) in obstetric ICU

Sl. No.	*Condition*	*Challenges*	*Management*
1	Mitral stenosis (MS)	Mostly rheumatic Low, fixed output Tachycardia detrimental, atrial fibrillation often present, large volume infusion not tolerated, sudden rise in pulmonary vascular resistance (PVR), fall in Systemic vascular resistance (*SVR*) poorly tolerated, PAH may be present in long-standing cases May present during pregnancy with hemodynamic compromise and CCF	Initiate oxygen therapy/ventilator support early CVP-guided fluid administration Avoid tachycardia, vasodilation, hypoxia, hypercarbia, acidosis Vasoconstrictors to be used with great caution Antibiotic prophylaxis
2	Mitral regurgitation (MR)	May be rheumatic or Mitral valve prolapse (MVP) Eccentric cardiac hypertrophy with poor contractility in long-standing cases, PAH leading to RV dysfunction	Initiate oxygen therapy/ventilator support early CVP-guided fluid administration Avoid tachycardia, vasodilation, hypoxia, hypercarbia, acidosis Vasoconstrictors to be used with great caution Pulmonary vasodilators like phosphodiesterase III inhibitors to manage PAH Antibiotic prophylaxis
3	Aortic stenosis (AS)	Rheumatic or congenital bicuspid aortic valve disease (BAVD) Fixed output state LV hypertrophy may lead to ischemia Bradycardia and junctional rhythm poorly tolerated Sudden vasodilation or myocardial depression may lead to cardiovascular collapse very difficult to resuscitate	Close monitoring of ECG with ST-T changes Control of blood pressure and protection against ischemia with nitrates or NTG infusion Antibiotic prophylaxis They may come to ICU once cardiovascular collapse has occurred and require resuscitation/defibrillation
4	Aortic regurgitation (AR)	Poor myocardial contractility Prone to ventricular arrhythmias, low EF, poor peripheral perfusion	Initiate oxygen/ventilatory management early CVP-guided fluid Inodilators like dobutamine in combination with adrenaline for hemodynamic support May need IABP also
5	Infective endocarditis (IE)	In the acute phase, infective emboli may lodge in various end-arteries of the body leading to infarction in vital organs (lung, kidney, brain, retina) May rapidly decompensate into severe sepsis	Antibiotic prophylaxis/therapeutic as advised by a cardiologist Ventilatory support, fluid management, inotropic support according to sepsis guidelines
6	Cardiac prosthesis	May be mechanical/bioprosthetic valves, other prosthetic material Patients on anticoagulants which may undergo dose alteration during pregnancy Pregnancy being a hypercoagulable state may cause "stuck valve" especially in the mitral position, formation of atrial thrombi and embolism	Urgent review of coagulation status and referral to cardiologist/cardiothoracic surgery department for definitive management

Table 7: Various types of cardiomyopathies in the parturient

Condition	*Challenges*	*Management*
Dilated cardiomyopathy	Poor contractility due to dilated chambers after long-standing ischemic heart disease, low ejection fraction, increase in afterload poorly tolerated	CVP-guided fluid administration, inodilator combined with adrenaline to maintain hemodynamics
Hypertrophic cardiomyopathy	Dynamic obstruction of the left ventricular outflow tract (LVOT), vasodilation, hypovolemia lead to low cardiac output and hemodynamic instability	Maintain adequate preload Inotropes are detrimental as they increase LVOT gradient
Restrictive cardiomyopathy	Cardiac output is preload and HR dependant Hypovolemia, atrial arrhythmias may cause hemodynamic instability	Maintain HR and normal rhythm, avoid hypovolemia, arrhythmogenic drugs
Arrhythmogenic RV cardiomyopathy	Extremely high risk of sudden death, ventricular arrhythmias, require automatic implantable cardioverter-defibrillator (AICD) implantation for long-term management	Preferably transfer to critical care unit under the expert care of cardiologist after initial assessment and stabilization

Other cardiomyopathies:[34] These conditions are preexisting and the parturient may already be undergoing medical therapy or may have automated intracardiac defibrillator (AICD) *in situ*.

Congenital Heart Disease in Pregnancy

Parturient may have preexistent various congenital heart disease (Table 8).[35,36]

Pulmonary Hypertension (Primary and Secondary)

Parturients with PAH are considered at extremely high risk of maternal mortality and have various underlying etiologies (Table 9). Pregnancy is usually contraindicated or terminated early. Medical therapy for PAH is continued throughout (calcium channel blockers, phosphodiesterase III receptor antagonists, prostacyclin) but endothelin receptor antagonists should be discontinued. These patients are present with heart disease in late pregnancy or early postpartum period.[37]

Pulmonary hypertension (mean pulmonary artery pressure ≥20 mm Hg at rest) is not well tolerated during pregnancy.[38] PAH is common in both valvular and congenital heart disease patients, where it occurs due to chronic left-to-right shunting. The goal is to preserve right ventricular function and reduce pulmonary arterial resistance. Massive fluid shifts, volume overload, excessive blood loss, and oxytocic drugs should be avoided.

Pregnant women with Eisenmenger syndrome require ICU admission (if the oxygen saturation falls below 85%) and may benefit from pulmonary vasodilator drugs like phosphodiesterase-5 inhibitors (nebulized and intravenous iloprost). Multidisciplinary management with an experienced intensivist, a cardiologist with expertise in pulmonary hypertension management, and an obstetrician with experience in high-risk pregnancies should be roped in the team, starting from the counseling stage. Pregnancy is generally contraindicated in these women and, if termination is declined after conception, the risks, as well as plan of management, must be clearly explained to the patient and family. Termination of pregnancy is also a high-risk procedure in these patients, requiring a team approach and ICU care.

Ischemic Heart Disease and Acute Coronary Syndromes

Ischemic heart disease accounts for 8 per 100,000 hospitalizations in pregnant women, with a higher rate of maternal death than the nonpregnant population. Its spectrum includes stable angina, unstable angina, and myocardial infarction. The normal physiological changes in pregnancy can accelerate or unmask the development of underlying coronary artery disease. The risk factors for the development of myocardial infarction during pregnancy include:

- Obesity (elevated BMI)
- Older age
- High parity
- Smoking
- Diabetes mellitus
- Preexisting hypertension
- Strong family history or history of coronary artery disease
- Hyperlipidemia
- Kawasaki's disease
- Peripartum infection

Pregnant women having acute coronary syndrome may present with either typical (chest pain, dyspnea) or atypical symptoms (vomiting, acidity, diaphoresis) or with hemodynamic compromise (due to arrhythmias or shock).[39] The diagnostic workup of these patients in the ICU involves a 12-lead ECG (ST-elevation or depression), cardiac enzymes (elevated troponin levels), coronary angiography by a trained cardiologist, and baseline echocardiography to assess the presence of regional wall motion abnormalities with left ventricular function.

Appropriate management includes timely cardiologist opinion and coronary intervention as per requirement. In addition, supplemental oxygen, oral aspirin, and clopidogrel (300 mg) should be given. Some authors have suggested that a *"Pregnancy Heart Team"* may be formed for better management of such high-risk parturients. Emergency coronary procedures should not be withheld in the parturient, as salvaging the mother is vital for fetal survival. Thrombolysis may be considered by the cardiology team on an individual case basis, weighing the risk–benefit ratio, as it carries the possibility of significant maternal and fetal hemorrhage. The use of antiplatelet agents may preclude the

Table 8: Congenital heart disease (CHD) in parturient

Condition	*Acyanotic*	*Cyanotic*
Examples	• Atrial septal defects (ASD), • Patent foramen ovale (PFO), • Ventricular septal defects (VSD), • Patent ductus arteriosus (PDA), • Bicuspid aortic valve (BAV), • Coarctation of the aorta (CoA), • Congenital coronary anomalies	• Tetralogy of Fallot (ToF), • Congenitally corrected transposition of great arteries (ccTGA), • Ebstein's anomaly
Challenges	Pulmonary hypercirculation, long-standing cases may have severe PAH, present with HF or pulmonary edema leading to respiratory failure Severe PAH may lead to shunt reversal (Eisenmenger syndrome) poor prognosis	Inadequate oxygenation, PAH, right ventricular dysfunction, may present with cardiac or respiratory failure
Management	CVP-guided fluid management, inotropic support, early mechanical ventilation	Avoid increase in PVR (hypoxia, hypercarbia, acidosis), mechanical ventilation, infundibular stenosis may worsen with hypovolemia and tachycardia, maintain preload and avoid arrhythmias

Table 9: Clinical classification of pulmonary hypertension (World Symposium of Pulmonary Hypertension)

Sl. No.	*Entity of pulmonary hypertension (PHT)*	*Management*
1.1	Idiopathic/Primary	• Symptomatic treatment to prevent right ventricular failure and hypoxemia • Avoid conception if possible • Hospital admission in second trimester • ICU care for decompensation and in third trimester. • Early cardiologist care during antenatal visits, labor, delivery, and postpartum period
1.2	Heritable	• Includes familial and simplex PAH • Autosomal dominant inheritance • Mean survival is 2.8 years after diagnosis • Significant stress of pregnancy in HPAH with high maternal mortality rates • Early cardiologist intervention, monitoring of right ventricular function, genetic studies • Use of suitable contraceptive measures
1.3	Drugs and Toxin induced	• Exposure to appetite suppressants like aminorex, fenfluramine derivatives, benfluorex (withdrawn from the market) • Amphetamines, phentermine, mazindol, Dasatinib, and interferons associated with PAH • Identifying and stopping the implicating agent are vital for survival and reversibility • Counseling regarding contraception and psychological support
1.4	Associated with	• Treatment of connective tissue disease with disease-modifying agents and antiretroviral therapy will need modification in pregnancy to prevent fetotoxicity
1.4.1	Connective tissue diseases	• Concurrent pulmonary hypertension in these patients heralds a poor feto-maternal prognosis during pregnancy and labor.
1.4.2	HIV infection	• A multidisciplinary team approach with early involvement of cardiologist, rheumatologist, physician, and obstetrician
2	PAH associated with heart disease (left ventricular dysfunction, congenital heart disease, valvular heart disease, cardiomyopathy)	• Optimization of the primary cardiac disease • ICU admission for cardiac failure, arrhythmias, hypoxia, Eisenmenger syndrome • Infective endocarditis prophylaxis • Pulmonary vasodilators under the direct supervision of a cardiologist
3	PAH associated with lung disease or hypoxia	• Detected on chest examination, pulmonary function testing, and high-resolution computed tomographic lung imaging • The unfavorable feto-maternal outcome in these cases with pregnancy due to poor cardiorespiratory reserve and pregnancy-induced stress • Patients to continue home oxygen therapy along with medications for their lung disease • Early involvement of cardiologist, pulmonologist, and intensivist (especially if ventilatory support is required)
4	PAH due to pulmonary embolism or diseases of large pulmonary vessels	• Screening is done by lung perfusion scanning • Minimize radiation exposure for investigations during pregnancy • In chronic thromboembolic pulmonary hypertension (CTEPH), surgical thrombo-endarterectomy may be considered before conception • Early involvement of cardiologist and cardiothoracic vascular surgeon in care • Consider DVT prophylaxis as per institutional protocol.

use of labor epidural or central neuraxial blockade, especially during emergencies. They may require ICU admissions also for complications of acute coronary syndrome, in the form of serious arrhythmias, heart failure, cardiogenic shock, stent thrombosis, recurrent myocardial infarction, or maternal cardiac arrest.

Arrhythmias and Pacemaker

Physiological changes in pregnancy can lead to physiological ECG changes[40] in the form of the following:

- Shortening of PR, QRS, and QT intervals (increase in HR)
- Left axis deviation (small q wave): Rotation of heart
- Inverted T wave in lead 3: Diaphragmatic elevation

The following rhythm disturbances are common during pregnancy:

- Ectopic beats
- Supraventricular tachycardia
- Ventricular arrhythmias
- Paroxysmal atrial fibrillation/flutter
- Heart blocks

The risk factors for the development of arrhythmias in the parturient include the presence of congenital or valvular heart disease, history of the previous arrhythmia, episodes of catecholamine surge with enhanced adrenergic receptor excitability, sudden extra volume load, electrolytes imbalances, hyperemesis, and medication-induced.

The management of arrhythmias in a pregnant woman is on the same lines as in a nonpregnant woman. Those with hemodynamic instability require ICU admission and early cardiologist opinion. Tachyarrythmias with hemodynamic compromise can develop in the pregnant woman with Eisenmenger syndrome, single-ventricle

physiology after Fontan operation, those on digoxin therapy, or antiarrhythmic drugs.

Management depends on hemodynamic status and types of arrhythmias. Hemodynamically unstable arrhythmias require direct current cardioversion, which is considered safe in pregnancy. If hemodynamically stable, intravenous adenosine (does not cross the placenta) or beta-blockers (except atenolol) can be used for acute exacerbation of SVT. The most important step in the treatment of atrial flutter or atrial tachycardia is rhythm control. Intravenous metoprolol and oral sotalol (both selective beta-blockers) are recommended and considered safe (category B) pregnancy. Calcium channel blockers like diltiazem and verapamil can also be used, as they are relatively safe in pregnancy (category C). Amiodarone should preferably be avoided due to fetal toxicity. Thromboembolism prophylaxis should be considered in a high-risk woman with heparin or antiplatelet therapy.

Ventricular tachyarrhythmias can be triggered if the parturient misses her dose of beta-blockers. If hemodynamically unstable, cardioversion should be attempted. Short-term treatment can be given with intravenous lidocaine, as it is considered safe during pregnancy. Fetal monitoring is recommended during cardioversion. Radiofrequency catheter ablation therapy is contraindicated in pregnancy due to the high risk of radiation exposure.

Heart blocks in pregnancy can be missed if not monitored properly and can result in fetal loss. Injection isoproterenol hydrochloride may be used for initial stabilization in an infusion dose of 0.1–1 μg/kg/minute (after bolus dose). Atrioventricular blocks can progress to complete heart block and may require a pacemaker insertion. Permanent pacemaker[41] insertion can be contemplated in those with symptomatic bradycardia and heart rate <50 beats/minute. Insertion of pacer may be planned after 8 weeks of gestation (in the second trimester) if possible, under echocardiographic guidance, to prevent radiation exposure in the fetus. In parturients with a preexisting pacemaker, the pacing is tolerated well, provided battery life and functioning are checked regularly. To account for the physiological increase in heart rate during early pregnancy, the lower limit of a pacemaker should be raised. The patient must be under the constant care of a cardiologist.

Pericardial Diseases in the Parturient

There is no specific predilection to pericardial pathology in parturients, except for those residing in endemic, tropical areas.[42] Pericarditis/pericardial effusion may be chiefly tubercular in origin and can be associated with pleural effusion as well. In case of severe anemia with hypoproteinemia, there may be generalized anasarca with ascites also. Conception should preferably be planned during remission. The commonest one is hydropericardium, especially in the third trimester of pregnancy. Most of these pericardial effusions are transient and benign, with the scope of spontaneous resolution. They can range from being asymptomatic to presenting with high fever, PUO (pyrexia of unknown origin), dyspnea, palpitations, or chest pain. Pericarditis requires treatment with cyclo-oxygenase inhibitors (nonselective) and aspirin, which are considered safe in the first and second trimesters of pregnancy. They should be discontinued before 32 weeks gestation due to adverse effects on ductus arteriosus and renal function. Low-dose steroids like prednisolone may also be required in some cases, which are considered safe in pregnancy. Colchicine is not recommended in pregnancy, except when required for Familial Mediterranean fever. Pericardiectomy may be indicated for recurrent pericarditis resistant to medication, especially in tuberculosis. Pericardial tamponade is a dangerous entity and may require (echo) ultrasound-and-ECG-guided pericardiocentesis with continuous drainage, under the expert care of a cardiologist and cardiothoracic surgeon (CTVS) in the ICU. In the case of cardiac tamponade, most likely due to traumatic cause, the patient may be in hypovolemic shock. After initial stabilization, transfer to CTVS for definitive management viz, drainage of cardiac tamponade.

Recent Advances and Future Prospects

With improvement in survival after cardiac transplantation, many of these patients survive into the childbearing age. Postcardiac transplant pregnant woman needs a multidisciplinary approach, including preconception and genetic counseling, the safety of immunosuppressive therapy, optimization of comorbid conditions, and the hemodynamic effects of pregnancy on the allograft. These patients may require critical care support due to graft rejection, infections, adverse effects of immunosuppressives, severe systemic hypertension, and thromboembolic complications. These patients require dedicated ICUs with a trained cardiologist, cardiothoracic surgeon, obstetrician, neonatologist, and obstetric intensivist for optimal management.

Maternal cardiac conditions such as severe PAH, peripartum cardiomyopathy, Eisenmenger syndrome, and cardiac failure due to any cause, not responding to optimal inotropic therapy, may be considered candidates for advanced techniques for hemodynamic support:[43]

- Intra-aortic balloon pump—It helps in maximum augmentation of coronary blood flow and minimizes the risk of embolization to cerebral vessels. A very useful method for additional hemodynamic support can be performed bedside preferably by the CTVS team.
- Ventricular assist devices (VADs)—LVAD/RVAD are employed in cases with long-term poor cardiac contractility, cardiomyopathies, and refractory cardiac failure to maintain hemodynamic function till definitive management like cardiac transplant becomes feasible (as a bridge to transplant), but are not definitive therapy themselves.
- Extracorporeal membrane oxygenation (ECMO)—This can be useful in both cardiac and respiratory failure cases. Benefits both hemodynamic support and gas exchange, many patients with cardiomyopathy may require ECMO support for a short period. Patients may still require inotropic support even on ECMO. Joint decision-making and prognosis regarding feto-maternal outcomes must be explained.

Conclusion

Parturients with cardiac disease present unique challenges and require an individualized, tailor-made approach by a dedicated, multidisciplinary team. Critical care management of these high-risk patients must be done in obstetric intensive care units for enhanced feto-maternal outcomes.

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