

A pathophysiology driven case based approach to managing pregnancy complicated by cardiac disease: Redefining the obstetrician's role

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Abstract

Background: Cardiovascular disease (CVD) is a leading and potentially preventable cause of maternal morbidity and mortality. Management is complicated by the profound physiological adaptations of pregnancy, including increases in plasma volume, heart rate, stroke volume and cardiac output. Understanding the interaction between these physiological changes and the underlying cardiac condition enables obstetricians to anticipate clinical deterioration, individualize management and facilitate multidisciplinary care.

Case presentation: This approach is illustrated by the case of a 27-year-old woman with Eisenmenger syndrome (modified World Health Organization class IV) who became pregnant despite medical advice and declined termination. Cardiovascular optimization, thromboprophylaxis, infection prevention, fetal surveillance, management of pre-eclampsia, management of fetal growth restriction, preterm caesarean delivery, intensive postpartum care and definitive contraception are the cornerstones of management.

Conclusion: Management of CVD in pregnancy should be based on the pathophysiology of the underlying cardiac lesion and the cardiovascular demands imposed by pregnancy, labour and the puerperium. The obstetrician has a central role in integrating specialist recommendations into a coordinated plan that ensures maternal and fetal wellbeing.

Keywords: cardiovascular disease in pregnancy, Eisenmenger syndrome, high-risk pregnancy, multidisciplinary care, cardiovascular physiology

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Background

Cardiovascular disease (CVD) is a leading cause of maternal mortality worldwide, with up to 68% of maternal deaths attributable to CVD being considered preventable through early recognition and appropriate management^{1,2}. From 2006 to 2018, the maternal mortality ratio attributed to CVD in Sri Lanka was 7.24 per 100,000 live births. Rheumatic heart disease is the most common cardiac condition complicating pregnancy, followed by cardiomyopathies and congenital heart disease^{3,4}.

CVD in pregnancy comprises a broad spectrum of conditions that vary in anatomical characteristics, pathophysiological consequences and severity. Therefore, management should be essentially individualised and guided by a multidisciplinary team at a high-risk obstetric centre. Understanding the cardiovascular adaptations in pregnancy is central to this approach.

This article highlights a pathophysiology-based approach from the obstetrician's perspective, through the

management of a pregnant woman with Eisenmenger syndrome complicated by pre-eclampsia, fetal growth restriction, and very preterm delivery.

Cardiovascular changes in pregnancy

Pregnancy is a state of generalised vasodilation. Oestrogen and relaxin promote nitric oxide-mediated vasodilation. Progesterone reduces sensitivity to angiotensin II, a potent vasoconstrictor. The developing placenta creates a large, low-resistance vascular bed. Consequently, systemic vascular resistance (SVR) and afterload decrease, resulting in reductions in systolic, diastolic and mean arterial blood pressure⁵.

Plasma volume increases by approximately 40–50% by 32 weeks of gestation due to hormone-mediated sodium and water retention. Maternal heart rate (HR) increases during early pregnancy and reaches its peak at approximately 28–30 weeks of gestation. It may increase by up to 25% above the non-pregnant baseline and generally returns to pre-pregnancy levels within six weeks after delivery⁵.

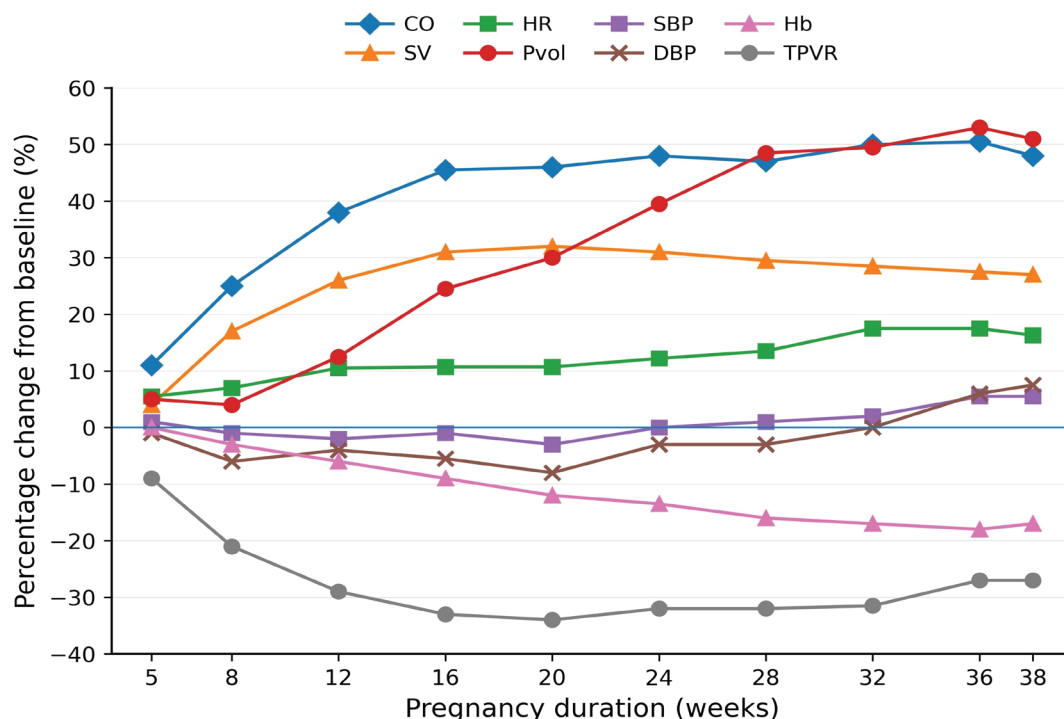


Figure 1. Cardiovascular changes in pregnancy. Adapted from Karamermer and Roos-Hesselink¹⁰.

CO: Cardiac output; DBP: Diastolic blood pressure; Hb: Hemoglobin concentration; HR: Heart rate; Pvol: Plasma volume; SV: Stroke volume; SBP: Systolic blood pressure; TPVR: Total peripheral vascular resistance.

Cardiac output (CO) is determined by stroke volume (SV) and HR. SV depends on myocardial contractility, preload and afterload. Neurohormonal stimulation, hypoxia, acidosis, pain, anxiety and sympathetic activity influence myocardial contractility. Venous return, ventricular compliance and diastolic filling time are important determinants of preload. Afterload is primarily determined by SVR⁶. The initial increase in CO is predominantly due to the rising SV, followed by the rise of HR later in pregnancy. Eventually, CO may increase by up to 80% above the pre-pregnancy level during labour and delivery⁵.

Labour imposes additional cardiovascular demands, including pain, anxiety and a catecholamine surge, which increase HR and myocardial contractility. Each uterine contraction transfers approximately 400–500 mL of blood into the maternal circulation. CO increases by approximately 15% above third-trimester values during the first stage of labour and by up to 45% during the second stage. Immediately after delivery, relief of caval compression and autotransfusion from the contracting uterus cause a further abrupt increase in CO⁷. Therefore, labour and early postpartum period are particularly critical for women with poor cardiac reserve.

Case presentation

A 27-year-old woman with a previous first-trimester miscarriage was transferred to a tertiary obstetric care centre at eight weeks of gestation for further

management. Her pregnancy was complicated by a large ventricular septal defect (VSD) with right-to-left shunting and severe pulmonary hypertension (PH), consistent with Eisenmenger syndrome. She was not on effective contraception and conceived despite medical advice. She was asymptomatic on admission. Physical examination revealed plethora, digital clubbing and a loud P2. Her pulse rate was 88 beats per minute, and her blood pressure was 100/70 mmHg. Arterial oxygen saturation was 72% on room air. Ultrasonography confirmed a viable singleton pregnancy consistent with the gestational age.

Defining the maternal risk

The first step focuses on identifying the underlying anatomical cardiac lesion and its pathophysiological sequelae. The basic assessment includes a detailed history, physical examination, electrocardiography and two-dimensional echocardiography. Depending on the underlying condition, additional investigations, including cardiac catheterisation, magnetic resonance imaging, coronary angiography and haematological investigations should be performed.

The second step is risk stratification using the modified World Health Organization (mWHO) 2.0 classification of maternal cardiovascular risk. It links the underlying cardiac condition to the anticipated maternal risk and guides counselling, monitoring and place of care.

Table 1.

mWHO 2.0 Class	Conditions	Maternal risk	Counselling	Obstetric and cardiac care during pregnancy
I	• Atrial or ventricular ectopic beats, (isolated) • Successfully repaired simple lesions (ASD, VSD, PDA) without significant residual complications • Small/mild PS, MVP	No detectable increased risk of maternal mortality and no/mild increased risk in morbidity	Yes (by regular healthcare professional)	Local hospital

(Continued)

mWHO 2.0 Class	Conditions	Maternal risk	Counselling	Obstetric and cardiac care during pregnancy
II	• Most supraventricular arrhythmias • Bradycardia requiring a pacemaker • Unoperated, uncomplicated ASD, VSD • Repaired TOF without complications	Small increased risk of maternal mortality or moderate increase in morbidity	Yes (by regular healthcare professional)	Local hospital
II-III	• Mild LVD: EF >45% • Repaired atrioventricular septal defect without significant residual lesions • Native, homograft or tissue valve disease not considered mWHO 2.0 I or IV: mild MS, moderate AS • Moderate valvular regurgitation. • Marfan syndrome without aortic dilatation • Repaired coarctation	Intermediate increased risk of maternal mortality or moderate to severe increase in morbidity	Yes: expert counselling Heart Team is required	Shared care with local hospital + Pregnancy Heart Team
III	• Moderate LVD: EF 30%–45% • Sustained ventricular tachycardia from any aetiology • Unrepaired cyanotic heart disease (not Eisenmenger) • Uncomplicated mechanical valve with stable well controlled INRs • Moderate MS • Severe asymptomatic AS • Severe left sided valvular regurgitation • Aortic dilatation: 40-45 mm • Prior ischaemic cardiac event (STEMI/NSTE ACS)	Significantly increased risk of maternal mortality or severe morbidity	Yes: expert counselling by Pregnancy Heart Team is required	Expert centre, care led by Pregnancy Heart Team

(Continued)

mWHO 2.0 Class	Conditions	Maternal risk	Counselling	Obstetric and cardiac care during pregnancy
IV	• Severe LVD: EF <30% or NYHA class III/IV • Eisenmenger syndrome • Pulmonary arterial hypertension • Severe MS • Severe symptomatic AS • Aortic dilatation: > 45 mm	Extremely high risk of maternal mortality or severe morbidity	Yes: expert counselling by Pregnancy Heart Team is required, with clear and thorough discussion of very high pregnancy risk and shared decision-making process for termination if pregnancy occurs	Care led by Pregnancy Heart Team

Table 1: Modified World Health Organization 2.0 classification of maternal cardiovascular risk. Adopted from 2025 ESC Guidelines for the management of cardiovascular disease and pregnancy. ASD: atrial septal defect; VSD: ventricular septal defect; PDA: patent ductus arteriosus; PS: pulmonary stenosis; MVP: mitral valve prolapse; TOF: tetralogy of Fallot; LVD: left ventricular dysfunction; EF: ejection fraction; MS: mitral stenosis; AS: aortic stenosis; INR: international normalised ratio; NYHA: New York Heart Association; STEMI: ST segment elevation myocardial infarction; NSTEMI: non-ST segment elevation acute coronary syndrome.

This patient was classified as mWHO 2.0 Class IV due to the presence of Eisenmenger syndrome and PH. Pregnancy is contraindicated because of the exceptionally high risk of maternal heart failure, arrhythmia, thrombosis, progressive hypoxaemia and death. If pregnancy occurs, early termination should be discussed. If the woman chooses to continue the pregnancy, she requires intensive multidisciplinary specialist management.

Decision about termination vs. Continuation of pregnancy

A multidisciplinary pregnancy heart team should assess the cardiac lesion, functional status, treatment options, anticipated maternal and fetal outcomes. The woman must receive clear, non-directive counselling about continuation and termination, including the possibility of deterioration despite apparently stable early pregnancy.

Gestational age provides useful physiological context. A woman presenting before 16 weeks is still experiencing the early rise in cardiac output. Failure to tolerate this change suggests that further increases may cause decompensation, strengthening the recommendation for termination. Between approximately 16 and 28 weeks, CO is relatively

stable. A woman who remains haemodynamically stable during this interval may be able to reach fetal viability, although continued stability cannot be guaranteed.

Maternal deterioration at any gestation requires reassessment of whether the pregnancy can safely continue. When termination is considered, valid informed consent is essential. The woman must understand the maternal and fetal risks and make the decision voluntarily with adequate mental capacity. Psychiatric assessment may be appropriate if capacity is uncertain, while cultural, religious and psychological influences should be approached sensitively.

This patient was counselled that her severe hypoxaemia and PH placed her at extremely high risk despite the absence of symptoms. Early termination was recommended, but she declined and elected to continue the pregnancy. Her informed decision was respected, and a comprehensive care plan was established.

Cardiovascular optimization

Management aims to improve oxygen delivery, reduce unnecessary cardiac workload and oxygen

consumption. Physical activity should be limited according to functional status. Pain, anxiety, and psychological stress should be minimized. Anaemia should be corrected as reduced oxygen-carrying capacity increases cardiac demand. Supplemental oxygen may improve oxygen delivery in selected patients with hypoxaemia.

Infection may precipitate decompensation through fever, tachycardia and increased metabolic demand. Patients require active surveillance for infection, prompt treatment and attention to dental health. Cardiac medications should be individualised by the cardiologist, considering both maternal benefit and fetal safety.

This patient was managed with strict activity restriction, intermittent oxygen during the day and continuous overnight oxygen. Sildenafil was prescribed for PH, and propranolol was prescribed under cardiology supervision. She underwent dental assessment and received appropriate care. Despite infection-prevention measures, she developed two episodes of urinary tract infection, both of which were treated promptly with intravenous antibiotics.

Cardiac interventions required to preserve maternal life should not be withheld because of pregnancy. When a non-emergency procedure is necessary, the second trimester may offer the best balance between the risks of teratogenicity and preterm birth. Elective interventions that can safely be postponed are generally deferred until after delivery.

Thromboprophylaxis

Pregnancy is a pro-coagulant state. Cyanotic heart disease, PH and venous stasis further increase the thromboembolic risk. Prolonged bed rest intended to reduce cardiac workload may create an additional hazard. Prophylactic measures include graduated compression stockings, intermittent pneumatic compression and low-molecular-weight heparin (LMWH). The risks of thrombosis and bleeding must be reviewed repeatedly, particularly when delivery or an invasive procedure is anticipated. This patient received LMWH and graduated compression stockings.

Anticipating maternal and fetal complications

Maternal complications include heart failure, arrhythmia, thromboembolism, infection, pre-eclampsia,

eclampsia and death. Pregnancy-related expansion of plasma volume and cardiac output may overwhelm limited cardiac reserve. Labour, anaesthesia, haemorrhage, infection and postpartum autotransfusion may trigger sudden deterioration.

Fetal risk is primarily determined by maternal hypoxaemia, impaired uteroplacental perfusion, medication exposure and the need for iatrogenic preterm delivery. The risks of miscarriage, fetal growth restriction (FGR), prematurity and perinatal death are increased. Maternal congenital heart disease also increases the likelihood of congenital cardiac disease in the fetus. Therefore, detailed fetal echocardiography should be offered at approximately 20–23 weeks.

Low-dose aspirin should be considered where indicated to reduce the risk of pre-eclampsia⁸. Blood pressure and symptoms must be monitored. Diuretics may be required to control congestion, although intravascular volume must be managed carefully in preload-dependent lesions.

Fetal assessment includes an early dating scan, aneuploidy screening, detailed anomaly assessment, fetal echocardiography and serial evaluation of growth and amniotic fluid. Uterine and umbilical artery Doppler studies may help identify placental insufficiency. Antenatal corticosteroids should be administered when preterm delivery is anticipated, and magnesium sulphate should be used for fetal neuroprotection at appropriate gestations. Delivery should occur where neonatal intensive care is available.

The patient developed proteinuric pre-eclampsia and FGR. She underwent biochemical surveillance, serial ultrasonography and Doppler assessment. A detailed anomaly scan and fetal echocardiogram did not identify a fetal cardiac defect. Growth was assessed at two-week intervals.

Monitoring

Maternal monitoring should combine clinical findings with targeted investigations. Serial assessment should include symptomatology, NYHA functional class, pulse, blood pressure, oxygen saturation, signs of congestion and evidence of thromboembolism. Electrocardiography, echocardiography and biomarkers such as B-type natriuretic peptide may be used according to the lesion and clinical course.

Monitoring frequency and place of care depend on risk. Women with mWHO 2.0 Class IV disease often require prolonged hospitalisation or very frequent specialist reviews. A documented escalation plan should define warning symptoms, thresholds for intensive care, arrangements for emergency delivery and the responsibilities of each team member.

Fetal surveillance should be individualized according to gestation, growth, placental function and maternal condition. Cardiotocography is used when gestationally appropriate, while ultrasound and Doppler studies guide decisions when FGR or placental insufficiency is present.

Timing of delivery

Timing is determined by the balance between maternal CVD stability and the benefits of advancing fetal maturity. Most women with stable cardiac disease can continue towards term. However, in severe disease, delivery may be necessary once further continuation poses a significant maternal risk.

If the mother cannot tolerate the increasing haemodynamic burden, delivery may be required soon after fetal viability. When maternal and fetal conditions permit, prolongation from 28 to 32 or 34 weeks substantially improves neonatal survival and reduces long-term morbidity⁹. Corticosteroids and magnesium sulphate should be provided when preterm delivery is anticipated.

The patient progressed to severe pre-eclampsia, necessitating emergency delivery at 30 weeks' gestation to prevent fetomaternal morbidity and mortality.

Mode of delivery and intrapartum care

Vaginal delivery is generally preferred for women with stable cardiac disease because it is associated with lower risks of haemorrhage, infection and thromboembolism than caesarean delivery. The decision should nevertheless consider the specific cardiac lesion, maternal functional status, fetal condition, cervical favourability and conventional obstetric indications.

Pain, anxiety, contractions, Valsalva manoeuvres and autotransfusion increase CO. Effective regional analgesia, careful fluid management and avoidance of

prolonged active pushing can reduce haemodynamic stress. Assisted vaginal birth may be used to shorten the second stage when maternal expulsive effort is weak.

Caesarean delivery is indicated for obstetric reasons, severe maternal compromise or when labour is considered unsafe. Anaesthesia must be individualized. A single-shot spinal may cause rapid vasodilation and hypotension and can be hazardous in PH. Carefully titrated epidural or combined spinal-epidural techniques may offer greater haemodynamic control in selected patients. General anaesthesia may be required but also carries major risks. Invasive arterial pressure monitoring and advanced cardiovascular support should be available when indicated.

Blood loss, hypoxia, hypotension, acidosis, infection and hypothermia must be minimized because each can worsen pulmonary vascular resistance or cardiac performance. This patient underwent emergency caesarean delivery for severe pre-eclampsia. A 910 g infant was delivered and received neonatal intensive care for six weeks.

Postpartum care

The early postpartum period is among the most dangerous periods because uterine autotransfusion, relief of caval compression and mobilization of extracellular fluid abruptly increase venous return. Pain, haemorrhage, infection and thromboembolism may further destabilize cardiac function.

Women with severe disease require intensive monitoring after delivery. Fluid balance should be carefully controlled. Diuretics should be used where clinically indicated. Oxygenation, blood pressure, cardiac rhythm and signs of right or left heart failure should be monitored. Anticoagulation should be restarted at an appropriate time once haemostasis is secure. This patient was managed in the obstetric intensive care unit for one week with meticulous monitoring and supportive care.

Breastfeeding is possible in many women with stable cardiac disease, provided their medication is compatible and the additional physical demand is tolerated. In decompensated heart failure, severe hypoxaemia or haemodynamic instability, breastfeeding may increase fatigue and compromise recovery. Temporary or permanent suppression

of lactation may be appropriate following multidisciplinary review.

Future complications and contraception

Postpartum counselling should address prognosis, future treatment options and reproductive intentions. In women with Eisenmenger syndrome, further pregnancy is strongly contraindicated because pregnancy-related haemodynamic changes may cause fatal deterioration even when the woman is initially asymptomatic.

Effective contraception must be established before discharge. The choice should follow medical eligibility criteria and consider thrombosis, bleeding, infection, drug interactions and the consequences of contraceptive failure. Long-acting reversible methods, including contraceptive implants and appropriately selected intrauterine devices, are generally preferred when permanent contraception is not chosen. Oestrogen containing methods are usually unsuitable when thrombotic risk is high.

Sterilization may be appropriate when pregnancy is permanently contraindicated, but counselling must emphasize its irreversible nature and ensure that consent is voluntary and obtained without coercion. Following counselling, this patient underwent sterilization at the time of caesarean delivery.

Conclusion

Management of heart disease in pregnancy should extend beyond the application of isolated protocols. The cardiac lesion must be understood in relation to the physiological changes of pregnancy, labour and the puerperium. This allows the team to anticipate when increases in plasma volume, cardiac output, venous return, oxygen consumption or pulmonary vascular resistance may precipitate deterioration.

The obstetrician has a central coordinating role: defining maternal and fetal risk, facilitating informed decision-making, optimizing cardiovascular status, preventing avoidable complications, planning delivery and ensuring intensive postpartum and contraceptive care. A pathophysiology-driven, individualized and multidisciplinary approach provides the clear framework for improving maternal and perinatal outcomes.

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