

Cardiac Critical Care of the Cardio-Obstetric Patient

Journal of Intensive Care Medicine
2026, Vol. 41(3) 186-199
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DOI: 10.1177/08850666241308207
journals.sagepub.com/home/jic
Mary Ann Liebert
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Amrin Kharawala, MBBS¹, Sanjana Nagraj, MBBS^{2,3},
Gayatri Setia, MD^{4,5} , Deborah Reynolds, MD^{4,5},
and Rosy Thachil, MD^{4,5} 

Abstract

Cardiovascular disease (CVD) is the leading cause of pregnancy-related mortality in the United States, with an incidence that has increased from 7.2 to 32.9 fatalities per 100,000 live births in the last 3 decades. This trend underscores the potential for an increase in the volume of admissions to cardiac intensive care units (CICUs) in the peripartum period. While congestive heart failure remains at the forefront of maternal morbidity, other life-threatening conditions include myocardial infarction (MI), hypertensive emergencies, fatal arrhythmias such as ventricular fibrillation, venous thromboembolism, aortopathies, valvular dysfunction, cardiac arrest, and cardiogenic shock. The lack of standardized guidelines to facilitate management of these conditions highlights the significant gap in medical knowledge while caring for acutely ill pregnant women. Through this comprehensive review, we highlight the most common cardiac pathologies encountered in the obstetric population and their diagnosis and contemporary management in the cardiac intensive care unit.

Keywords

cardio obsetrics, CICU in pregnancy, congestive heart disease, myocardial infarction, cardiac arrest, arrhythmia, valvular heart disease, pulmonary embolism, cardiogenic shock, hypertensive emergency

Introduction

There has been an increase in the incidence of pregnancy-related mortality in the United States from 7.2 to 32.9 deaths per 100 000 live births from 1987 to 2021 as per the data derived from the Centers for Disease Control and Prevention (CDC).^{1,2} Maternal mortality is significantly higher in Black women and the rates tend to worsen with increasing maternal age.¹ Among these, cardiovascular disease (CVD) stands as the foremost contributor to pregnancy-related mortality, accounting for almost 43% of such fatalities including cardiomyopathies (12.1%) and mortality associated with hypertensive disorders of pregnancy (6.3%).¹ CVD can complicate nearly 4% of pregnancies in women without risk factors and up to 16% of pregnancies in women with pre-existing cardiac risk factors.³ This indicates the potential for an increased number of planned and unplanned cardiac intensive care unit (CICU) admissions during or after pregnancy. A recent Nationwide Inpatient Sample (NIS) based study of 13.79 million delivery-based hospitalizations, found 11 152 hospitalizations to be complicated by severe cardiovascular maternal morbidity leading to 495 maternal deaths.⁴ Of these, congestive heart failure (CHF) was reported as the most common cause of maternal morbidity, while other significant conditions included myocardial infarction (MI), ventricular fibrillation, and cardiogenic shock (CS). Even the rate of pregnancy associated myocardial infarction (PAMI) has increased over the last two decades with an estimated incidence of 2.8-8.1 per 100 000 deliveries, which is 4 times higher than the rate of MI in non-pregnant women in

the reproductive age group.^{5,6} While caring for the pregnant patient, it is important to note that nearly 50% of maternal deaths occur within 24 h of childbirth, and 66% of deaths occur within the first week after delivery.⁵ Hence, meticulous care and consideration should be exercised through the first 12-weeks postpartum.⁵ As per current literature, almost 80% of pregnancy-related deaths in the US attributable to cardiovascular conditions are preventable.² In recent times, an increasing maternal age, and presence of cardiovascular risk factors like hypertension, diabetes, and obesity in pregnant women can contribute to maternal morbidity and make management challenging.⁶ Besides the above-mentioned pathologies, pregnancy complicated by hypertensive emergencies and aortic pathology, thrombotic diseases,

¹University of Nebraska Medical Center, Omaha, NE, USA

²Montefiore Medical Center, Bronx, NY, USA

³Albert Einstein College of Medicine, Bronx, NY, USA

⁴Elmhurst Hospital, New York City Health & Hospitals Corporation, Queens, NY, USA

⁵Mount Sinai Hospital, New York, NY, USA

Received September 24, 2023. Received revised November 8, 2024.

Accepted December 4, 2024.

Corresponding Author:

Rosy Thachil, Director, Cardiac Intensive Care Unit (CICU), Division of Cardiology, Elmhurst Hospital Center, Mount Sinai School of Medicine/NYC H&H, New York, NY, USA.
Email: thachilr@nychhc.org

Table 1. Physiological Changes on Cardiovascular Parameters During pregnancy.⁷⁻⁹

Parameter	First trimester	Second trimester	Third trimester	Labor
1 Blood Pressure	↓ in DBP > SBP	↓ Nadir is reached with a 5-10 mmHg drop below baseline	BP normalizes or even ↑	Can increase due to pain
2 Heart Rate	↑ by 15%–30%	↑↑	↑↑↑ (peaks at end)	↑↑↑↑
3 Systemic Vascular Resistance ^a	↓ to 30%–40% of baseline	↓↓ Reaches nadir	↓↓ 20% less than baseline at term	↓
4 Stroke Volume	↑ by 25%	Constant	Constant or ↓	
5 Cardiac Output	↑	↑↑ to 45% of normal up to 24 weeks	↑↑	60% ↑↑↑↑ during labor.

^aPregnancy does not affect PVR, even though changes in SVR are noted.

Abbreviations: BP: Blood pressure; DBP: Diastolic blood pressure, PVR: Pulmonary vascular resistance, SBP: Systolic blood pressure, SVR: Systemic vascular resistance.

arrhythmias, and cardiac arrest form some of the most important conditions warranting cardiac intensive care.

Despite an increasing prevalence of cardiovascular morbidity and mortality in the pregnant population, there is a lack of robust management literature of these high-risk cardiovascular conditions in the CICU. Through this review, we highlight the most common cardiac pathologies encountered in the obstetric population and their management strategies in the cardiac intensive care unit.

Cardiac Physiology

Pregnancy leads to multiple physiologic changes in the body, an understanding of which is essential to understand the cardiovascular pathologies that inflict the pregnant population. The change in the cardiovascular physiology mainly affects the blood pressure, heart rate, stroke volume, cardiac output, and systemic vascular resistance. Due to a relatively larger increase in the plasma volume compared to the increase in red blood cell mass, pregnancy causes a state of physiological anemia (normal hemoglobin cut off is reduced to 10.5-11 g/dL depending on the trimester).⁷ Increases in plasma volume, mediated by the activation of the renin-angiotensin system, peak by the end of pregnancy.⁵ As estrogen production increases, the substrate for renin (angiotensinogen) also increases, leading to higher levels of angiotensin.⁷ Additionally, relaxin mediated secretion of vasopressin and increased thirst lead to water retention. However, progesterone acts as a potent aldosterone antagonist, preventing excessive sodium retention and hypokalemia. These hormonal changes along with an increase in maternal vasodilators like prostaglandins and nitric oxide contribute to an increased plasma volume with a reduction in systemic vascular resistance.⁷

The increase in cardiac output is secondary to an increase in the stroke volume in the first part of the gestation, and later secondary to an increase in the heart rate. This is secondary to a constant or reduced stroke volume after the second trimester.⁷ With respect to structural cardiac changes, left ventricular wall mass increases, along with enlargement of all 4 chambers and an increased distensibility of the aorta.⁷ Functional changes in the cardiac hemodynamics include up to a 34% reduction in pulmonary vascular resistance (PVR) and pulmonary capillary wedge pressure (PCWP). However, no significant changes are

noted in the central venous pressure (CVP), mean arterial pressure (MAP) and left ventricle stroke work index.

As shown in Table 1, increases in plasma volume, and therefore, the cardiac output can exacerbate pre-existing heart conditions like cardiomyopathies, potentially increasing the risk of pulmonary edema, MI and aortic dissection.¹⁰ An increased physiological state of hypercoagulability, leads to an increased risk of pulmonary embolism which has the highest incidence in the post-partum period.¹¹ A review of the different cardiovascular pathologies, including their diagnosis and management is present in the subsequent sections.

Risk Assessment and Stratification of Cardiac Pathology

Early identification and risk stratification of cardiac conditions in pregnant women is crucial for both maternal and fetal morbidity and mortality. The modified World Health Organization (mWHO) classification is one such screening tool as described in 2011 ESC guidelines which integrates all maternal cardiovascular risk factors and can be used for both congenital and acquired heart diseases.¹² It classifies patients into classes I to IV with increasing risk of cardiac complications, where pregnancy is contraindicated in patients falling under mWHO class IV.¹² Since then, other scores like CARPREG (Cardiac Disease in Pregnancy Study) that uses clinical and echocardiogram data and ZAHARA (Zwangerschap bij Aangeboren HARTafwijking [Pregnancy in Women With Congenital Heart Disease]) risk scores have also been validated.^{13,14} With increasing knowledge and understanding of maternal risk factors, the 4-predictor CARPREG score was modified to the CARPREG II risk index, which takes 10 parameters into consideration and assigns more points to predictors with higher odds ratio like history of prior cardiac events.¹⁵ The use of risk assessment scores to determine the severity of maternal cardiac risk can also guide the need for CICU admission for a patient. For instance, the California Quality Improvement Toolkit developed by the California Maternal Quality Care Collaborative (CMQCC) and California Department of Public Health provides an algorithm on the diagnosis and management of maternal cardiac conditions.^{16,17} Ideally, a dedicated multidisciplinary team with an obstetrician, cardiologist, neonatologist, anesthesiologist, intensivist, maternal fetal medicine (MFM) specialist,

cardiothoracic surgeon, nursing, pharmacist and social worker should be involved to provide optimal care to these patients.^{9,18} The following sections describes individual cardiac pathologies which may require CICU level of care and highlight their guideline-specific diagnosis and management.

Discussion: Diagnosis and Management of Major Cardiovascular Conditions

Hypertensive Emergencies

Hypertensive disorders are estimated to affect 1 in 10 pregnant women and are one of the leading causes of preventable maternal morbidity and mortality.¹⁹ Gestational hypertension (HTN) is the presence of persistent new-onset hypertension (BP ≥ 140 mmHg systolic or ≥ 90 mmHg diastolic) at ≥ 20 weeks gestation.¹⁹ Preeclampsia is defined as new-onset hypertension (BP ≥ 140 mmHg systolic or ≥ 90 mmHg diastolic) at ≥ 20 weeks accompanied by one or more features of end-organ damage: proteinuria, acute kidney injury (serum creatinine ≥ 1.1 mg/dl), hepatitis with elevated liver enzymes twice the upper limit of lab normal, pulmonary edema, neurologic involvement (altered mental status, blindness, stroke, clonus, headache, and visual field defects), hematologic complications (thrombocytopenia $<150\,000/\mu\text{l}$, disseminated intravascular coagulation, hemolysis), or uteroplacental dysfunction (fetal growth restriction, stillbirth, or abnormal umbilical artery doppler waveform).¹⁹ Along the spectrum of severe hypertensive disorders of pregnancy, as per the American College of Obstetricians and Gynecologists, hypertensive emergency or hypertensive crisis is defined as BP $\geq 160/110$ mmHg taken on two separate occasions 15 to 60 min apart.²⁰ Early recognition and prompt management begins with acknowledging that the BP cutoff of hypertensive emergency in pregnant individuals is considerably lower than that for non-pregnant adults. It indicates that pregnant individuals are at a high risk of stroke, convulsions, and death at a lower BP threshold.^{21,22}

Management. For Chronic hypertension in pregnancy, the cut off for the initiation of antihypertensive therapy has now been changed from 160/110 mm Hg to 140/90 mm Hg by the American College of Obstetricians and Gynecologists (ACOG) based on the results of the Chronic Hypertension and Pregnancy (CHAP) trial, as it results in better outcomes during pregnancy without negative effect on fetal growth or placental perfusion.^{23,24} In the CICU, management of hypertensive emergency entails ensuring antihypertensive therapy is administered within 1 h of establishing persistently elevated BP in the severe range.²⁵ Either IV Labetalol with a starting dose of 20 mg (cumulative dose should not exceed 220 mg in 24 h) or IV Hydralazine 5-10 mg (cumulative dose should not exceed 25 mg in 24 h) or oral nifedipine 10 mg. Current data show a significant quality lapse in the institution of timely pharmacologic therapy, with medication being administered within 1 h in $<50\%$ of hypertensive episodes.²⁶ Seizure prophylaxis should be provided with 10% MgSO₄ (Magnesium Sulfate) with a loading dose of 4-6 g administered over 20 min followed by a maintenance dose of 1-2 grams/hour.²⁰ While the management of gestational

hypertension, which should not be considered benign as it portends an increased long-term risk of cardiovascular disease, involves BP reduction to 110-140/85 mmHg, the management of preeclampsia is as follows: at first-time diagnosis, women with preeclampsia should be assessed in a hospital setting, until the stability of their condition is established and reliable outpatient follow-up set up. Severe hypertension (BP $\geq 160/110$ mmHg) requires urgent management in a monitored setting with either an oral or intravenous (IV) antihypertensive agent: oral nifedipine or IV labetalol or hydralazine. Preeclampsia with severe hypertension and proteinuria or neurological signs/symptoms should be managed with MgSO₄ prophylaxis to prevent eclampsia^{20,26} (Figure 1).

As pregnancy, and specifically, the placenta underpins the pathophysiology of preeclampsia, timing of delivery is an integral part of management.²⁷ Delivery is indicated in the following scenarios: (1) development of preeclampsia after 37 weeks; (2) if 37 weeks of gestation has been reached and course has been complicated by severe hypertension despite concurrent use of 3 different classes of antihypertensives, increasing liver and/or renal function tests, progressive thrombocytopenia, intractable headache, seizures, visual field defects, or non-reassuring fetal condition. The severity of proteinuria doesn't dictate the timing of delivery; (3) irrespective of gestational age, delivery is necessitated if there is failure to control BP despite using ≥ 3 classes of antihypertensives at optimal doses, maternal oxygen saturation by pulse oximetry $<90\%$, eclampsia, abruptio placentae, progressive decline in liver, renal, hematological, and/or neurological function, reversed end-diastolic flow in the umbilical artery doppler, or establishment of intrauterine fetal demise.²⁷ An expectant approach is used in women with onset of preeclampsia <37 weeks of gestation, wherein management in a center with maternal and fetal medicine expertise is important to safely carry the pregnancy till 37 weeks.

Myocardial Infarction

Impact. Pregnancy associated myocardial infarction (PAMI) is among the most common causes of cardiogenic shock in pregnant individuals, with peripartum cardiomyopathy being the most common cause.^{28,29} Acute coronary syndromes occur in approximately 3-10 out of every 100 000 pregnancies, at a rate that is 3 to 4 times higher than in similarly aged women who are not pregnant.²⁹ Not only does pregnancy appear to increase the incidence of ACS in women of childbearing age, PAMI is also associated with higher mortality than acute MI in the absence of pregnancy, with higher incidences of LV dysfunction and cardiogenic shock.²⁹

Risk Factors and Subtypes. As the mechanisms for PAMI vary widely from the traditional mechanisms in non-pregnant individuals, as do the risk factors for ACS in pregnancy. Maternal age greater than 30 years old at time of pregnancy is one of the strongest risk factors, regardless of mechanism. Pregnancy specific risk factors leading to PAMI include history of pre-eclampsia or gestational diabetes. Traditional cardiovascular risk factors, such as diabetes, hypertension, hyperlipidemia, and smoking do impose the expected risk of ACS, however this is more represented in the

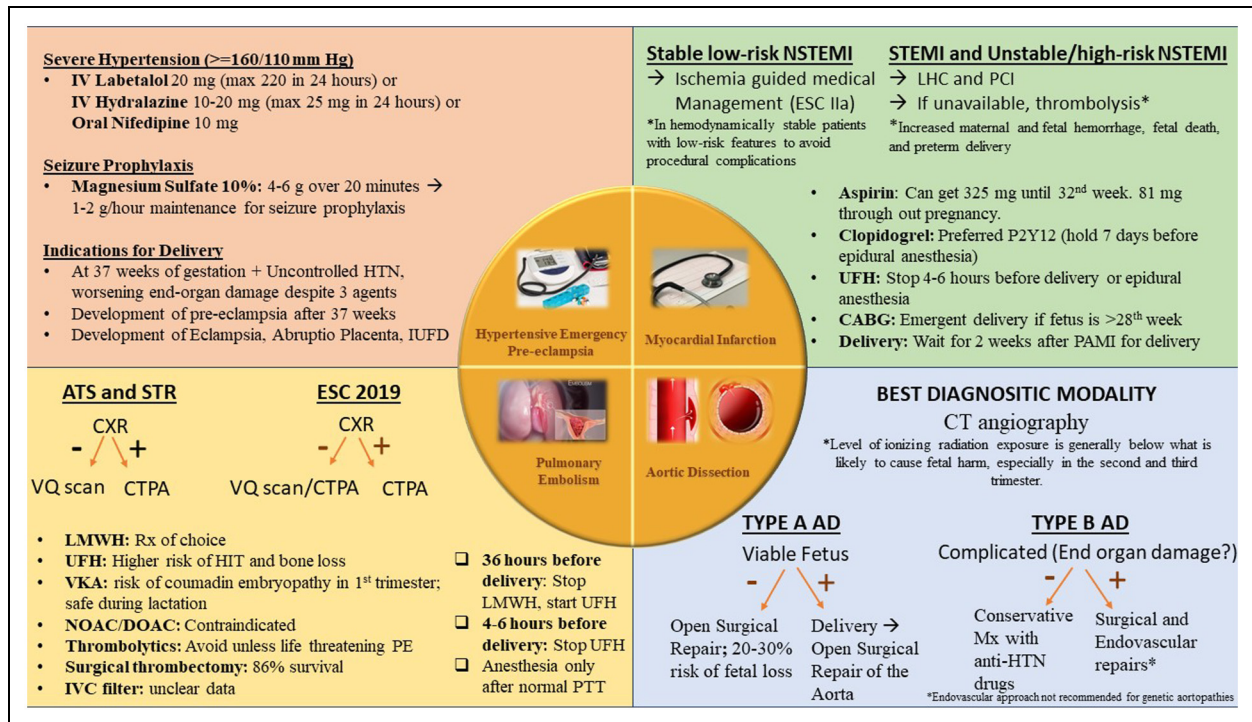


Figure 1. Considerations for management of myocardial infarction, hypertensive emergencies, aortic dissection and pulmonary embolism during pregnancy.

subset of patients who present with PAMI from obstructive atherosclerotic disease, which is increasing as the overall age at first pregnancy increases.^{29,30} More commonly, however, ACS in pregnancy occurs in the setting of nonobstructive coronary artery disease or normal coronaries.

The most common cause of ACS in pregnancy is spontaneous coronary artery dissection (SCAD), which is responsible for 14% to 43% of pregnancy-related MI.^{29,31} While there are multiple proposed contributors to SCAD, such as underlying arteriopathies, connective tissue disorders, hormonal shifts, catecholamine surges, genetics, and hypertension, pregnancy represents an important trigger for SCAD. The vast majority of pregnancy-related SCAD occurs postpartum ($>70\%$), with the first week having the highest risk. This can possibly be explained by the combination of hormonal changes and catecholamine surges that can last up to 6 months postpartum, not to mention significant valsalva-induced vascular wall stress during labor and delivery.^{29,31}

Pregnancy-related SCAD appears to have a distinct phenotype from non-pregnancy-related SCAD, both in terms of severity and location of the dissections. Pregnancy-related SCAD favors left main or multivessel coronary involvement, with subsequently worse left ventricular function and higher likelihood of cardiogenic shock and ventricular arrhythmias. SCAD outside of pregnancy, however, is more likely to occur in mid-to-distal portions of a single vessel, with the left anterior descending artery being the most common.³¹

Other mechanisms of PAMI include coronary thrombosis (17%–21%), coronary vasospasm (2%–5%) and coronary embolic events

(3%–4%). Although pregnancy is a hypercoagulable state, in the event of PAMI due to coronary thrombosis, secondary hypercoagulability workup is indicated. Embolic events are more likely in patients with pre-existing atrial fibrillation, underlying cardiomyopathy, or valvular disease. The mechanism of pregnancy-related coronary vasospasm is unclear, however elevated renin and angiotensin levels, underlying endothelial dysfunction, profound hypovolemia secondary to maternal hemorrhage, smoking, and ergot-derived medications have been suggested as predisposing factors.^{29,32}

The mechanism of ACS in pregnancy likely plays a role in when during the pregnancy it occurs. The most common period for ACS in pregnancy is in the late third trimester and early postpartum period. Post-partum incidence of AMI was noted to be more than double than that of antepartum MI in one retrospective study of 4471 PAMI patients (53% vs 20.6% respectively).³³ This timing is in line with the most common cause of PAMI being SCAD, which, as previously stated, shows a predilection for the postpartum period. More traditional, atherosclerotic obstructive CAD is implicated in approximately 27%–43% of cases, and is more likely to manifest antepartum.²⁹

Management. As in non-pregnancy-related ACS, invasive coronary angiography and percutaneous intervention remains the gold standard for PAMI in STEMI or hemodynamically unstable NSTEMI³⁴ (Figure 1). Medical, ischemia-guided management can be considered in stable NSTEMI patients without evidence of LV dysfunction or ongoing symptoms.³⁴ It is important to note that, while iodinated contrast crosses the placenta, as of yet there is no evidence of teratogenicity, and it is acceptable to use

in appropriate circumstances in pregnancy. Radiation exposure for angiography can be minimized by maternal and fetal shielding and short fluoroscopy times. The highest risk for fetal injury from radiation is prior to 20 weeks' gestation, and the required dose of radiation to cause fetal injury is generally well above the exposure from routine diagnostic and interventional catheterization procedures.²⁹

Thrombolysis is not routinely recommended in PAMI, as it is associated with increased maternal and fetal hemorrhage, fetal death, and preterm delivery. As in non-pregnant patients, thrombolysis should only be considered in setting of STEMI or hemodynamically unstable NSTEMI without the ability to perform coronary catheterization.²⁹

In terms of medical therapy in ACS, pregnant patients can receive full-dose aspirin (325 mg) up until about 32 weeks' gestation, at which point the risk of premature closure of the ductus arteriosus increases, and aspirin 81 mg can be administered throughout the entirety of pregnancy.^{29,35,36} As heparin does not cross the placenta, this remains the anticoagulant of choice in PAMI, however it does need to be discontinued prior to delivery given risk of maternal hemorrhage. In patients who undergo percutaneous coronary intervention (PCI), clopidogrel is the preferred P2Y₁₂ inhibitor in pregnant patients. Patients who receive P2Y₁₂ inhibition, however, cannot receive neuraxial blockade for labor and delivery, and the medication would need to be discontinued for at least 7 days prior to anesthesia. Due to this, bare metal stents used to be preferred for PCIs in pregnancy, but the newer generation drug-eluting stents have been studied with shorter periods of dual antiplatelet therapy and may now be more feasible.²⁹

In PAMI that results in hemodynamic instability, multivessel involvement, LV dysfunction, or shock, patients may require emergent PCI or coronary artery bypass grafting, despite the high morbidity and mortality of cardiac surgery on both mother and fetus (up to 3% and 20%, respectively). Emergent delivery should be considered prior to coronary artery bypass graft (CABG) in patients at >28 weeks' gestational age.²⁹

Outside of such an indication for an urgent or emergent delivery, waiting until at least 2 weeks after PAMI to deliver can help prevent maternal complications and is the current European Society of Cardiology (ESC) guideline recommendation. Vaginal delivery is also recommended outside of maternal instability or fetal distress.³⁴

Aortic Dissection

Aortic dissection (AD) tends to occur more in older populations, between 65 and 75 years of age. However, of those that occur in women under the age of 40, about half are related to pregnancy.^{37,38} As in non-pregnant populations, aortic dissections are rare but often fatal events. Maternal mortality from all types of AD has been estimated at 12% and fetal mortality even higher at 28%.³⁸ Maternal history of hypertension, connective tissue disorders such as Marfan's, or family history of aneurysm/dissections, have all been implicated as risk factors for pregnancy-associated AD.³⁷ Patients with known predisposing

factors should undergo preconception counseling and regular monitoring of aortic size during pregnancy, but dissection can happen in the absence of these risk factors. Pregnancy-induced changes in cardiac output and blood volume can increase wall stress on the aorta, and, coupled with hormonal changes affecting the media of the aorta, could lead to development of aortic aneurysms and dissection.^{34,38} Moreover, increased resistance on the arterial tree due to gravid uterus can also increase the risk for AD.³⁹ It follows, then, that the highest risk time to develop aneurysm/dissection in pregnancy is in third trimester and postpartum period, though dissections have been noted earlier in pregnancy as well.³⁸

Presentation of AD in pregnancy can be similarly nonspecific as in nonpregnant patients, with chest pain being the most common first symptom. As the mortality rate increases significantly with delays in care, providers should maintain a high index of suspicion for possible dissection in pregnant patients who complain of abrupt onset severe chest, back, or abdominal pain, especially if they describe the pain as ripping, stabbing, or tearing in nature.³⁷ CT angiography remains the diagnostic test of choice, even in pregnancy, as the level of ionizing radiation exposure from CT is generally below what is likely to cause fetal harm, especially in the second and third trimesters.³⁸

Type A aortic dissections, those involving the ascending aorta and arch, are surgical emergencies (Figure 1). If the fetus is viable, ESC guidelines recommend delivering the fetus by cesarean section first, immediately followed by open surgical repair of the aorta.³⁴ The recommendation remains emergent surgical repair of the aorta even if the fetus is nonviable, however patients should be counseled on the risk of fetal loss, which can be up to 20%–30%.³⁴ Uncomplicated Type B aortic dissections without evidence of end-organ malperfusion can be managed conservatively, as in the nonpregnant patient, with strict blood pressure control as using medications recommended during pregnancy.³⁴ Type B dissections complicated by malperfusion remain emergencies, and the management approach depends on both degree and location of the compromised organ. This could include a combination of surgical and endovascular repairs, peripheral surgical bypasses for limb ischemia, or thoracic endovascular repairs alone, however the endovascular approach in pregnancy has only been described in a few cases and is not the recommended approach for genetic aortopathies.^{34,38}

Valvular Heart Disease

Valvular heart disease affects approximately 0.18% of deliveries and is associated with increased incidences of obstetric complications such as hypertensive disorders of pregnancy, intrapartum and postpartum hemorrhage, PAMI, PPCM, pulmonary edema, and arrhythmias.⁴⁰ Patients with valvular heart disease are also at higher risk of preterm delivery.⁴¹ Stenotic lesions are more likely to cause complications and symptoms than regurgitant lesions, as are left-sided lesions when compared to right-sided lesions.^{42,43} While there is limited literature discussing specifically critical care of valvular disease in pregnancy, extrapolations can

be made based on the known changes in physiology of women during pregnancy and management of valvular lesions in the non-pregnant population.

As is the case in any known cardiovascular condition in a woman of childbearing age, prevention is always preferred to management of acute decompensation. Preconception counseling or intervention of a valvular lesion prior to pregnancy should be discussed. Previously asymptomatic women with valve disease can develop pulmonary edema or heart failure during pregnancy or immediately post-partum due to pregnancy-induced intravascular volume expansion and higher heart rate.⁴² This is particularly true with mitral and aortic stenosis, which are both sensitive to tachycardic states and more likely to result in pulmonary edema, arrhythmias, heart failure, and resultant increased maternal and fetal mortality.^{42,43}

Medical management of symptomatic mitral or aortic stenosis both involve activity restriction and heart rate control with a β 1-selective antagonist, with diuretic therapy if needed.⁴² While there is a concern of intra-uterine growth restriction (IUGR) with using beta blockers, β 1-selective antagonists such as metoprolol or bisoprolol are associated with lower rates of IUGR and are generally considered safe to use in patients with appropriate indications.⁴⁴ The exception to this is atenolol, which has been seen to result in higher rates of IUGR and should be avoided.⁴⁴

In patients with severe mitral stenosis with persistent symptoms or heart failure on optimal medical therapy, percutaneous balloon mitral valvuloplasty (BMV) can be safely performed in pregnancy, preferably after 20 weeks' gestation.^{42,45} Transcatheter or open surgical aortic valve intervention should only be pursued if the patient is clinically deteriorating despite medical therapy and activity restriction.⁴⁶ Transcatheter aortic valve replacement in pregnancy has not been studied, with only isolated case reports in the literature, and surgical valve replacement does carry significant maternal (9%) and fetal (30%–40%) risk, as in other cases where cardiopulmonary bypass is required.⁴⁶ Regurgitant lesions, again, are generally much better tolerated in pregnancy than stenotic lesions, but in the rare case that a patient develops NYHA Class IV symptoms despite medical therapy, valve surgery is reasonable to pursue.⁴⁶

In patients who have previously undergone mechanical valve replacement, complications can still arise, particularly in the setting of the hypercoagulable state of pregnancy and the difficulties of anticoagulation in pregnancy. Mortality is increased in both mother and fetus, as well as risk of maternal and fetal hemorrhage. Maternal risks also include valve thrombosis and associated dysfunction, heart failure, and stroke. Fetal teratogenicity of warfarin is also an important consideration, as warfarin is the only oral anticoagulant recommended for mechanical valves.⁴¹ Teratogenic risk is lower at low doses, however, such that the 2020 ACC/AHA guidelines and 2018 ESC guidelines both state that continuation of warfarin through all 3 trimesters is reasonable if the patient requires ≤ 5 mg of warfarin to remain therapeutic, with transition to low molecular weight heparin (LMWH) or unfractionated heparin (UFH) at least 1 week prior to planned delivery.^{34,46} If higher doses are required, LMWH with routine anti-Xa level monitoring is the recommendation for thromboprophylaxis, with transition to unfractionated heparin at least 36 h prior to planned

delivery.⁴⁶ It is important to note that vaginal delivery is contraindicated in women on therapeutic warfarin due to risk of fetal intracranial hemorrhage, and maternal hemorrhage is a risk across all modes of anticoagulation.³⁴ The use of epidural anesthesia is also contraindicated in the anticoagulated patient.⁴² UFH should be held at least 6 h prior to planned delivery.⁴⁶ If labor begins or there is an indication for urgent delivery while patient is on full-dose warfarin, reversal agents should be given followed by cesarean delivery.⁴⁶

Arrhythmias

Sinus tachycardia and premature ventricular or ectopic atrial beats can be seen in 50%–60% pregnant women.⁴⁷ However, the overall prevalence of serious arrhythmias leading to hospitalizations is estimated to be around 68–166 per 100 000 pregnancy-related admissions.⁴⁸ An increase in pregnancy-related hospitalizations by almost 58% (from 2000 to 2012) has been attributed to atrial fibrillation (AF) and ventricular tachycardia (VT).⁴⁹ This rise of arrhythmias has been attributed to an increase in maternal age, obesity, HTN, DM, and congenital heart diseases.⁴⁷ Arrhythmias during pregnancy tend to be more prevalent in Black women as compared with White women (116 vs 73 per 100 000; $P < .001$).⁴⁹

There is a paucity of randomized clinical trials assessing the safety of anti-arrhythmic drugs (AADs) in pregnancy.⁵⁰ With majority of AADs being Food and Drug Administration (FDA) category C (risk to the fetus cannot be ruled out), it is recommended to use the lowest effective dose for treatment.⁵⁰ Table 2 highlights the major life-threatening arrhythmias in pregnancy, treatment options and their safety according to the 2023 HRS consensus.⁴⁷

Since there is no real data regarding the safety of direct oral anticoagulants (DOACs) in pregnancy, low-dose warfarin can be used for anticoagulation up to 36 weeks.⁴⁷ Thereafter, LMWH is used until 36 h pre-delivery, followed by unfractionated heparin, which is stopped 6 h before delivery.⁴⁷ Despite established teratogenicity of warfarin in pregnancy, a recent meta-analysis in pregnant women with mechanical heart valves showed that use of low dose warfarin < 5 mg/day had a significantly lower risk for fetal embryopathy than higher-dose warfarin (8.25% vs 0.45%, $P < .001$).⁵¹ Pregnant patients with long QT syndrome (LQTS), should continue therapy with a beta-blocker, particularly nadolol or propranolol, especially during the postpartum period, which represents a high-risk period for cardiac events.⁴⁷

Congestive Heart Failure and Cardiomyopathies

The incidence of new-onset heart failure (HF) in pregnancy has increased over the last decade, and is the cause of in-hospital deaths in $> 9\%$ of pregnancy-related hospitalizations.⁵² HF in pregnancy can be secondary to pre-existing or new cardiomyopathy, pulmonary hypertension, valvular heart disease, congenital heart disease, or ischemic heart disease.⁵³ Based on data from the Registry On Pregnancy And Cardiac disease (ROPAC), left sided HF is more common among pregnant women compared to right sided HF (68% vs 45%).⁵⁴ Cardiomyopathy (4.6%; $P < .001$),

Table 2. Treatment and Drug Safety for major Arrhythmias in pregnancy.^{47,50}

Arrhythmia	Treatment	Adverse Drug Reaction	Lactation safety
Hemodynamically stable acute SVT	1. Vagal maneuvers (I C) 2. iv Adenosine (I C) 3. *Beta blockers (propranolol or metoprolol) (IIa C) 4. CCB (Verapamil or Diltiazem) (IIb C) 5. iv procainamide (IIb C)	2. dyspnea, bradycardia 3. bradycardia, growth retardation, apnea 4. Maternal hypotension, fetal bradycardia 5. Drug induced lupus, torsade de pointes	2. unknown 3. Safe 4. Safe 5. Compatible for short term use
Symptomatic SVT (without pre-excitation)	1. Metoprolol, propranolol, and/or digoxin (first line; I C) 2. Verapamil (second line) for the chronic oral prophylaxis of SVT. (I C)	Digoxin: low birth weight	Compatible
Recurrent or refractory SVT	1. Flecainide, propafenone, or sotalol (IIa C) 2. Catheter ablation (IIa C) with techniques to minimize radiation exposure 3. Amiodarone (IIc C) if refractory to pharmacological and interventional therapies	• Flecainide and Propafenone - Well tolerated in structurally normal heart • Sotalol: β -blocker effects, torsade de pointes Teratogenic; Fetal hypothyroidism, growth retardation, prematurity	• Flecainide: compatible • Propafenone: unknown • Sotalol: compatible but with caution Avoid
Hemodynamically stable AF/AfI with RVR	1. iv beta blockers (I C) 2. Digoxin or CCB (second line; I C)		
Hemodynamically unstable AF/AfI or refractory or persistent AF/AfI	1. DC synchronized Cardioversion (energy same as in non-pregnant woman) (I C) along with anticoagulation. 2. Catheter ablation with minimal radiation exposure if the above don't work (IIa C) -Postpone to second trimester if possible ¹² 3. Flecainide (if no SHD) or sotalol (if no severe LV dysfunction) are reasonable for rhythm control		
Wolf Parkinson White syndrome	Oral flecainide or propafenone (I C)		
Hemodynamically stable VT	1. iv Beta-blocker or Adenosine for outflow tract VT (I C) I. iv Verapamil for fascicular VT (I C) 2. Iv procainamide for acute therapy (I C)		
Recurrent VT or VT refractory to or with C/I to beta blockers	1. Flecainide, sotalol, or mexiletine drug based on the underlying cardiac substrate.	Mexiletine: Bradycardia, CNS effects, low APGAR score	Compatible
Unstable VT or sustained VT refractory to pharmacotherapy or C/I to pharmacotherapy	1. DC synchronized Cardioversion (energy same as in non-pregnant woman) (I C)		
Indications for SCD or VT with high-risk features	1. ICD placement (minimize radiation exposure) (IIb C)		
Mobitz type 2 or CHB or severe sinus dysfunction	1. Permanent Pacemaker (I C) *Temporary ventricular pacing if risk of hemodynamic instability and or syncope in peripartum period		

Abbreviations: AF/AfI: Atrial fibrillation/Atrial flutter, CCB: Calcium channel blockers, CHB: Complete Heart Block, C/I: contraindication, DC: CNS: Central nervous system, Direct current, FDA: Food and drug administration, ICD: Implantable cardioverter-defibrillator, LV: Left ventricle, RVR: Rapid ventricular rate, SCD: Sudden cardiac death, SVT: Supraventricular tachycardia, VT: Ventricular tachycardia.

*Beta blockers: Atenolol is FDA D; causes low birth weight and is not compatible with lactation; Pindolol is FDA B, compatible with lactation.

New York Heart Association (NYHA) class $\geq$ III (2.3%; $P = .01$), World Health Organization (WHO) class $\geq$ 3 (2.3%; $P < .001$), HF prior to pregnancy (9.6%; $P < .001$), and pulmonary hypertension (1.8%; $P = .04$) are the major risk factors for developing HF during pregnancy.⁵⁴ A review of some of the most prevalent causes of HF in pregnancy is presented below.

Peripartum Cardiomyopathy. Peripartum cardiomyopathy (PPCM) is idiopathic cardiomyopathy that presents as LV systolic dysfunction with an left ventricular ejection fraction (LVEF) of <45%⁵⁵ and occurs in 1 in 1000 pregnant woman worldwide.⁵⁶ It can occur at any time during pregnancy (13%), with the highest incidence during the last trimester. However, the incidence is even higher at delivery (27%), 6 weeks postpartum (60%), and has now also been described as late as 5-12 months post-delivery.^{52,53} In fact, late onset of PPCM has been associated with a 2-3-fold increase in maternal mortality and major adverse cardiac events (MACE). It is possible that some of these patients have takotsubo cardiomyopathy which has overlapping features with PPCM. The incidence of PPCM is nearly four-fold higher in Black women compared to White women.⁵⁶ On the contrary, Hispanic women have the lowest incidence of PPCM, a pattern that correlates with the overall prevalence of maternal heart failure among different races and ethnicities.⁵³ Pregnant women with HF have more than seven-fold higher likelihood of death.¹⁸ Neonates of affected mothers are prone to lower birth weights, small for gestational age, lower Apgar scores, and premature birth as compared to neonates of healthy controls.¹⁸ Recent studies have found that women with PPCM tend to have a loss of function variants in many genes including titin (TTN) which is the most common,⁵⁷ and others like FLNC, DSP and BAG3.^{58,59} Interestingly, some of these variants are also seen in patients with dilated cardiomyopathy, indicating that these conditions may share a similar genetic basis.⁵⁸ Whether genetic testing should be pursued even PPCM is currently debatable.

Hypertrophic Cardiomyopathy. An increased use of echocardiographic familial screening programs, HCM is being found in more women in their reproductive age.⁶⁰ Increased LV contractility and heart rate during pregnancy can worsen the obstructive symptoms of HCM, potentially resulting in a higher incidence of adverse maternal cardiac events as shown in the CARPREG II study.¹⁵ While metoprolol can be continued safely, verapamil and diltiazem should be avoided due to possibility of fetal bradycardia, heart block, hypotension and antitocolytic effect.⁶⁰

Diagnosis and Management (Figure 2). The considerable overlap between normal signs of pregnancy and initial clinical features of HF like peripheral edema, systolic murmur, and a third heart sound can delay the diagnosis of HF.¹⁸ Due to a higher than normal BNP level in pregnancy, a BNP level of ≤ 100 pg/mL is found to be acceptable to rule out CHF as it has 100% negative predictive value for identifying cardiac events during pregnancy.⁶¹ Diuresis with furosemide is safe in pregnant women with decompensated HF and has shown to improve hemodynamics and placental perfusion.^{34,35,62,63} While both loop diuretics and thiazide diuretics have not been found to be teratogenic, aldosterone antagonists should be avoided. Due to the association of 16-kDa form of prolactin with cardiac microvascular damage and worsening PPCM,⁶⁴ the 2018 ESC guidelines recommend bromocriptine (Class IIb, Level Of Evidence (LOE) B) 2.5 mg once daily for 1

week for uncomplicated cases of PPCM and 2.5 mg twice daily for 2 weeks followed by 2.5 mg once daily for 6 weeks in patients with LVEF <25% or cardiogenic shock (CS).³⁴ However, larger clinical trials to assess the efficacy of bromocriptine are still underway. Prophylactic anticoagulation should be used with bromocriptine due to its thrombogenic potential. The use of anticoagulation is also recommended in patients with PPCM and severe LV dysfunction (LVEF <30% as per AHA and <35% as per ESC guidelines) during and after pregnancy (until 8 weeks postpartum).^{34,62} The diagnostic features and management guidelines have been outlined in Figure 2.^{16,34,53,62,65-67}

Cardiogenic Shock

Cardiogenic shock (CS) is defined as a reduction in cardiac output (CO) secondary to decreased cardiac function, leading to systemic hypoperfusion and end-organ damage.⁶⁸ According to a NIS based study conducted from 2002-2013, CS most commonly occurs in the postpartum period (58.5%), followed by during labor (23.5%) and antepartum period (17.7%).²⁸ PPCM, amniotic fluid embolism, pulmonary thromboembolism and acidosis were the strongest risk factors associated with CS.²⁸ Sepsis and renal failure have been associated with worse mortality in pregnant women with CS.²⁸ Use of non-beta agonist drugs like milrinone and levosimendan is preferred over dobutamine for inotropic support in PPCM patients with CS.⁶⁷ Mortality from CS can be up to 18.8% in pregnant women, and can be potentially avoided with early use of mechanical circulatory support (MCS).^{28,67}

Mechanical Circulatory Support. Intra-aortic balloon pump (IABP), Impella, left ventricular assist devices (LVAD) and extra corporeal membrane oxygenation (ECMO) are frequently used forms of MCS. IABP and Impella can be used as a temporary MCS device or as a bridge to LVAD or transplantation in pregnant women.⁶⁸ While some centers may defer using anticoagulation while the IABP is on 1:1 augmentation, it is necessary to use anticoagulation for all other settings and for Impella as well.⁶⁸ Hemolysis due to mechanical shearing of red blood cells can occur within the first 24 h of Impella use in 5%–10% of patients, which may be post a risk of sensitization.⁶⁹ There is limited data on the use of right ventricular support devices like Impella RP, TandemHeart and PROTEK Duo (LivaNova PLC, London, UK) system in this cohort.⁶⁸ ECMO is another common form of MCS used in pregnancy. The overall survival after VA-ECMO use in the antepartum period was 75%, for pregnant women and 25% for their fetuses.^{67,70} ECMO leads to an increase in prolactin level which is associated with worse outcomes.⁶⁷ Hence, the ESC guidelines recommend use of up to 10 mg bromocriptine twice daily to suppress prolactin while on ECMO.⁷¹ Multiple studies have shown that VA-ECMO can also be a reasonable treatment modality in the third trimester or after delivery, and should be considered in patients with hemodynamic instability.^{72,73} The most common indications for ECMO include ARDS, heart failure or PPCM, pulmonary embolism and management after revived cardiac arrest.^{72,73} Bleeding is the most common maternal complication, and pre-term delivery is the most common fetal

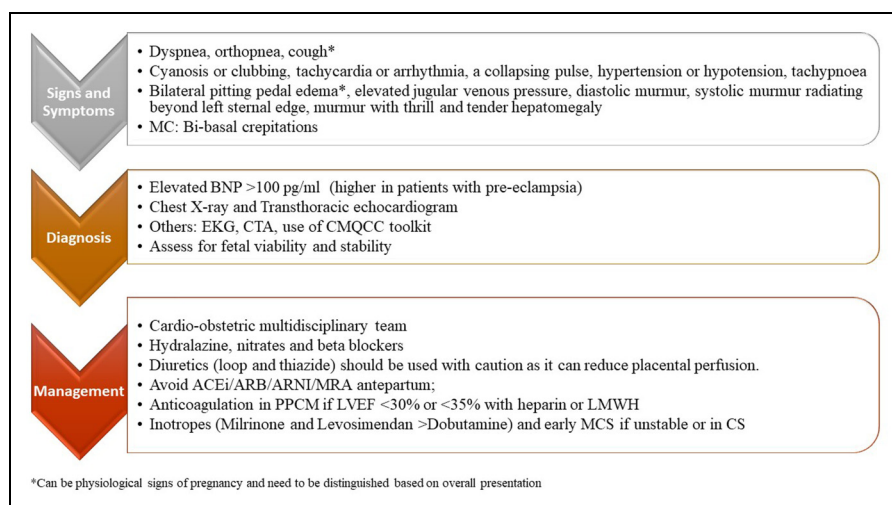


Figure 2. Diagnosis and management of CHF in pregnancy.

complication.⁷³ LVADs are primarily used as a bridge to heart transplant given the younger age and usually favorable profile of women with PPCM.⁷⁴ Placing LVAD under fluoroscopy is deemed relatively safe when the radiation dose is <50 mGy, although larger studies evaluating this are needed.⁷⁵ Orthotopic heart transplant is the definitive management in eligible patients with irreversible HF despite optimal therapy. The multicenter North America IPAC (Investigations of Pregnancy Associated Cardiomyopathy) study showed that although 72% women experienced a recovery of LVEF to >50%, 13% continued to have an LVEF <30% requiring LVAD or cardiac transplantation at the end of 1 year.⁷⁶ However, an analysis of 1258 women from the Interagency Registry for Mechanically Assisted Circulatory Support showed that 48% women with PPCM received heart transplant at around 36 months.⁷⁴ Care after heart transplant tends to be complicated by higher rates of allograft rejection and mortality in women whose indication for transplant was PPCM. This has been attributed to younger age, female sex, history of pregnancy and increased pre-transplant panel reactive antibodies (PRA).⁷⁶

Maternal Cardiac Arrest and Postmortem C-Section

Maternal cardiac arrest (MCA) is a rare but devastating complication of one or more of the several conditions discussed in this review. In a recent systematic review and meta-analysis of 63 859 400 maternities worldwide, the pooled global incidence of MCA was 5.83 per 100 000 maternities (95% CI: 3.07-8.59).⁷⁷ Of these, 42% of the women who suffered a cardiac arrest survived to hospital discharge, although data regarding the neurologic outcomes of these women were not known.⁷⁷ The most commonly reported etiologies of MCA in literature are maternal obstetric hemorrhage, cardiac etiology, amniotic fluid embolism, and VTE. Other causes include complications of anesthesia, preeclampsia and eclampsia, drugs, trauma, and a range of indirect etiologies, including sepsis.⁷⁸

Management. Pregnant women who are ill should be risk stratified routinely using validated obstetric early warning scores (Class I, Level C).⁷⁹ Severe early warning scores are available but are not specific to maternal cardiac arrest. Carle et al in their study of a new obstetric score and evaluation of preexisting early warning scores (eg: Swanton's Modified Early Obstetric Warning System) in a large British ICU patient population demonstrated excellent discrimination between survivors and non-survivors using the new obstetric score.⁸⁰ Validated early warning scores carry practical value and have shown to accurately identify women at high risk of mortality. Although not specific to MCA, these scores allow for appropriate disposition, pre-event planning, including timely preparation for MCA and neonatal resuscitation.⁷⁹ Mobilization of resources, including key personnel such as the intensivist, obstetrician, nursing staff, neonatologist should occur concurrently as timely multidisciplinary management (maternal cardiac interventions and obstetric interventions) is key.⁸¹

In non-pregnant individuals, cardiopulmonary resuscitation restores about 33% of the cardiac output.⁸² Due to compression of the great vessels by the gravid uterus, cardiopulmonary resuscitation restores only 10% of the maternal cardiac output in MCA.⁸² Therefore, high quality chest compressions are extremely important and left-lateral displacement of the uterus can help relieve compression of the aorta and inferior vena cava, facilitating circulation. Following advanced cardiovascular life support (ACLS) guidelines, as used in non-pregnant adults, including defibrillation is recommended. Intravenous access should be obtained above the level of the diaphragm.⁸¹

Fetal assessment should not be performed during resuscitation and fetal monitors should be removed immediately to avoid hindrance with perimortem cesarean section (PMCS). Perimortem cesarean section which involves delivery of the fetus via cesarean section during ongoing resuscitation offers several benefits in the unfortunate occurrence of MCA. By delivery, compression of the great vessels is relieved and the relative blood flow to other organs

increases, thus, restoring close to 25% of the cardiac output. Second, in the absence of a compressing uterus, functional residual capacity of the mother's lungs increases and oxygenation improves. Improved circulation and oxygenation after timely PMCS has been documented by Katz and Dijkman et al in their respective landmark studies.^{83,84} Therefore, resources for PMCS should be mobilized emergently with a goal cut-off time for performing PMCS at 4 min after MCA if return of spontaneous circulation is not achieved, and delivery by 5 min. In real-world conditions, only around one-third of those who suffer MCA undergo PMCS. Understandably, maternal and neonatal survival are contingent upon several factors including the etiology of arrest, location of MCA (out-of-hospital vs in-hospital and if in a monitored setting), available resources, and preparation and experience of the medical team attending to the patient.⁷⁸ Pregnant women who survive MCA but remain comatose should receive targeted temperature management according to the 2020 American Heart Association resuscitation guidelines.⁸⁵ Continuous monitoring of the fetus that may remain in-utero for bradycardia is recommended while targeted temperature management is administered.⁸⁵

Venous Thromboembolism

Venous thromboembolism (VTE) is nearly ten times more common in pregnant women compared to their nonpregnant counterpart, with an estimated incidence of 0.5-2 in 1000.^{86,87} Pregnancy is a hypercoagulable state due to the inevitable anatomic and physiologic changes that occur, such as increased venous stasis, decreased mobility, compression of the inferior vena cava and pelvic veins by the enlarging gravid uterus, and alterations in the levels of coagulation factors.⁸⁸ This risk can further increase by in-vitro fertilization, preeclampsia, cesarean section, postpartum hemorrhage, obesity, and prior VTE.⁸⁹ Deep vein thrombosis (DVT) and pulmonary embolism (PE) follow slightly different occurrence patterns, with around 60% of DVT occurring in the antepartum period compared to PE, which is most common in the immediate postpartum period.⁹⁰ Yet, DVTs account for most VTEs in pregnancy and overall, VTE account for 9.3% of maternal deaths.^{88,91}

Diagnosis and Management. Routine measurement and use of D-dimer levels to guide decision-making about VTE is not recommended by both ACOG and Royal College of Obstetricians and Gynaecologists (RCOG) as D-dimer levels increase during pregnancy, especially as gestational age increases.⁹² Similarly, falsely negative D-dimer values have been documented in the presence of VTE during pregnancy and postpartum period, further supporting the guideline advisory against their use.^{88,93,94} On the contrary, the 2019 ESC guidelines recommend obtaining a D-dimer in conjunction with clinical probability (some studies suggest using a higher D-dimer cutoff of 800 ng/mL), as an initial step considering the adverse effects of radiation from computed tomography pulmonary angiography (CTPA).^{95,96} As per ACOG, in the presence of new symptoms/signs of DVT, the recommended initial investigation is a duplex ultrasonography of the lower extremities. If

nonrevealing and there remains suspicion of iliac vein thrombosis, doppler ultrasonography of the iliac veins or magnetic resonance imaging should be pursued. Diagnosis of PE can be made with either ventilation-perfusion scanning (VQ scan) or CTPA. Contrary to common misconception, wherein the fetal exposure from VQ scan is low (approximately 0.32-0.64 mGy), mean fetal doses from helical CT are lower (0.0033-0.02 mGy for the first trimester, 0.0079-0.0767 mGy for the second trimester, and 0.0513-0.1308 mGy for the third trimester).^{93,97} As per the American Thoracic Society and the Society of Thoracic Radiology guidelines, investigation for PE in pregnancy should begin with a chest x-ray, followed by VQ scan if the x-ray is normal and CTPA if the x-ray is abnormal. According to the 2019 ESC guidelines, specific investigation of PE can begin with a chest x-ray, followed by either VQ scan or CTPA if the x-ray is normal, and CTPA if abnormal.⁸⁹

Management. Management should involve a multidisciplinary team with specific experience in managing PE in pregnancy (Figure 1). The ESC recommends written care pathways, jointly formulated by collaborating members, be made available.⁸⁹ Low Molecular Weight Heparin (LMWH) is the drug of choice for VTE in pregnancy as it does not cross the placenta. Dosing is similar to that in the nonpregnant population. Unfractionated heparin (UFH) has been associated with a relatively higher risk of heparin induced thrombocytopenia and bone loss during pregnancy. Coumadin and other Vitamin K antagonists (VKA) cross the placenta and carry a risk of fetal bleeding, neurodevelopment defects, and coumadin embryopathy in the first trimester.⁸⁹ However, VKA can be used in breastfeeding mothers. Direct oral anticoagulants too have been shown to cross the placenta and are contraindicated in pregnant women.

Delivery should be planned through multidisciplinary collaboration to avoid spontaneous labor on therapeutic anticoagulation. In high-risk cases, particularly if managing a recent PE, LMWH should be stopped, and unfractionated heparin should be started ≥ 36 h before delivery. 4-6 h before delivery, UFH should be stopped, and a normal activated partial thromboplastin time (aPTT) should be obtained before administering regional anesthesia.⁹⁸ Timing of anticoagulation initiation after delivery depends on an individual basis, including mode of delivery and complications of regional anesthesia. Under the realm of cardiac intensive care of the obstetric patient lies high-risk life-threatening PE. In a systematic review of 127 patients with high- and intermediate-risk PE who were either pregnant or <6 weeks postpartum, 23% of women suffered a cardiac arrest. Although survival following thrombolysis was 94%, major bleeding occurred in 18% of pregnant women and 86% of women during puerperium.⁹⁹ Among those who underwent surgical thrombectomy, 86% survived. Raising concerns for reporting bias, the ESC recommends against the use of thrombolytics in the peripartum period unless managing life-threatening PE. Data on the use of vena cava filters in pregnant women are limited, and procedural risk remains a valid concern.

Cardiac Tamponade

Presence of a small pericardial effusion may be a normal finding in pregnant women. However, cardiac tamponade is a life-threatening emergency. Presentation is largely similar to that in the general population and etiologies could either be those encountered in non-pregnant individuals, such as viral or idiopathic pericarditis, malignancy or secondary to conditions exclusive to pregnancy such as preeclampsia and HELLP (hemolysis, elevated liver enzymes and low platelets) syndrome. Management of cardiac tamponade is similar to that in the general population, and involves drainage of the effusion by percutaneous pericardiocentesis or surgically by creating a pericardial window.¹⁰⁰

Limitations

This review focuses on the critical and often life-threatening cardiovascular conditions that can complicate pregnancy, aiming to elucidate optimal approaches to management, within the confines of the CICU. We acknowledge the importance of adult congenital heart diseases, rheumatic heart disease, and the numerous etiologies of pulmonary hypertension that can complicate pregnancy. However, taking into consideration the breadth of pathologies that can complicate pregnancy and, the above conditions were beyond the scope of our review. Due to the primary focus being on cardiac critical care management, describing neonatal monitoring and breastfeeding is also beyond the scope of this review. Similarly, we have refrained from describing airway management and mechanical ventilation, due to the depth of the topic which would require more focused exploration in separate, targeted manuscripts or reviews.

Conclusions

Considering the dual-nature of lives involved, multidisciplinary management forms the basis of care in cardio-obstetric conditions. Use of validated checklists and risk-assessment scores allow for the early identification of at-risk pregnant women, decision on appropriate disposition, and pre-event planning. Use of system-wide management protocols and rigorous training of healthcare professionals involved in the cardio-obstetric care of pregnant women can help improve patient and neonatal outcomes. As stated previously, prevention takes precedence, and pregnant women with cardiovascular risk factors and cardiac diseases should be optimized at each visit to avoid decompensation and allow for a safe pregnancy and delivery.

Declaration of Conflicting Interests

The authors declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Ethics Approval Statement

Not applicable.

Funding

The authors received no financial support for the research, authorship, and/or publication of this article.

Patient Consent Statement

Not applicable.

Permission to Reproduce Material from Other Sources

Not applicable to this work as no copyrighted material has been used.

ORCID iDs

Gayatri Setia  <https://orcid.org/0000-0003-2369-4407>

Rosy Thachil  <https://orcid.org/0000-0003-3760-429X>

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