

## Acute Management of Cardiac Complications in Pregnancy

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### Abstract

Cardiac disease complicates more than 1% of pregnancies in the US and causes 20% of non-obstetrical deaths. The increase of heart disease in pregnancy is attributed to increased rates of obesity, hypertension and diabetes, in addition to the survival of women with congenital heart disease to maternal age. Furthermore, women are increasingly postponing pregnancy until the fourth decade of life. These factors increase the incidence of cardiac disease complicating pregnancy. In developed countries, cardiomyopathies, structural heart diseases, pulmonary hypertension, acute myocardial infarction and conduction abnormalities are the leading cardiac causes of maternal mortality.

The physiologic changes that occur during pregnancy lead to increases in preload, cardiac output, blood volume and oxygen consumption. Such changes can unmask, worsen or induce cardiac dysfunction. The diagnosis of cardiac decompensation is often difficult to make because complaints of shortness of breath, peripheral edema and chest pain can be attributed to normal pregnancy. The times of greatest risk for cardiac disease during pregnancy are the third trimester, peripartum and immediate postpartum periods.

**Keywords:** Pregnancy; Cardiac disease; Myocardial infarction; Heart failure; Cardiomyopathy; Hypertension; Dysrhythmia

### Heart Disease in Pregnancy

Cardiac disease complicates more than 1% of pregnancies in the US and causes 20% of non-obstetrical deaths [1,2]. The increase of heart disease in pregnancy is attributed to increased rates of obesity, hypertension and diabetes, in addition to the survival of women with congenital heart disease to maternal age. Furthermore, women are increasingly postponing pregnancy until the fourth decade of life. These factors increase the incidence of cardiac disease complicating pregnancy. In developed countries, cardiomyopathies, structural heart diseases, pulmonary hypertension, Acute Myocardial Infarction (AMI) and conduction abnormalities are the leading cardiac causes of maternal mortality [3].

Cardiovascular changes in pregnancy include a 45 percent increase in circulating blood volume, a 43 percent increase in cardiac output and a 17 percent increase in the resting heart rate [4]. Furthermore, systemic vascular resistance is lowered and blood pressure decreases. Anatomically, the heart is displaced up and to the left along with the elevation of the diaphragm. These physiologic changes lead to increases in preload and oxygen consumption. Such changes can unmask, worsen or induce cardiac dysfunction. The diagnosis of cardiac decompensation is often difficult to make because complaints of shortness of breath, peripheral edema and chest pain can be attributed to normal pregnancy. The times of greatest risk for cardiac disease during pregnancy are the third trimester, peripartum and immediate postpartum periods [5].

### Acute Myocardial Infarction in Pregnancy

Acute coronary syndromes are rare events in women of child bearing age. The incidence of AMI is estimated to be 6.2 per 100,000 pregnancies with a case fatality rate of 5.1% [6]. However, this represents a 3 to 4 fold increase in comparison to non-pregnant women. The incidence of AMI increases with maternal age, and is 30 fold higher for women older than 40 years of age [6]. Risk factors associated with AMI are consistent with traditional risk factors, including chronic hypertension, diabetes mellitus and smoking. Additionally, the following have been shown to be independent risk factors: age greater than 30, multigravidas, thrombophilia, preeclampsia and eclampsia, transfusions and postpartum infections [6]. AMI can occur at any stage of pregnancy but is most common in the third trimester and within the

first 6 weeks of the postpartum period [7].

In a review of 103 obstetric patients diagnosed with AMI from 1995-2005, coronary angiography and post-mortem evaluation demonstrated that 40% had stenosis from atherosclerosis, 27% coronary dissection, and the remaining 33% showed thrombus without stenosis, vasospasm or normal angiography [7]. Coronary dissection is a rare cause of AMI in non-pregnant women, however it represents the majority of cases of MI in the peripartum period. It is postulated that the excess level of progesterone during pregnancy leads to structural vessel wall changes. This, in combination with the increased cardiac output and blood volume of pregnancy, may lead to greater shear forces causing coronary dissection [7].

Diagnosis and treatment of AMI in pregnant women is similar to non-pregnant patients. Beyond the history and physical exam, paying particular attention to the above listed risk factors, electrocardiography and cardiac biomarker assessments should be performed. As with non-pregnant patients, troponins are preferred to CK-MB and myoglobin [8]. Both aspirin and heparin are safe in pregnancy. Intravenous heparin infusion is preferred because of the relative ease of anticoagulation reversal in the case of pregnancy complications associated with bleeding. Nitroglycerine can be used for the treatment of anginal pain and has tocolytic properties. However, maternal hypotension should be avoided and careful titration is necessary [9]. In the setting of ST elevation MI, Percutaneous Coronary Intervention (PCI) is recommended over thrombolytic therapy [10]. PCI is considered safe for maternal and fetal survival and is preferred to thrombolytics because of the decreased risk of bleeding in pregnancy and the increased incidence of coronary dissection as an etiology for AMI [9]. The fetus should be monitored and a plan for urgent delivery of a viable fetus should be established in case of maternal clinical deterioration.

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## Heart Failure

Causes of heart failure in pregnancy include underlying structural heart disease, cardiomyopathy, hypertensive disorders, dysrhythmias, and acute coronary syndromes. In the developed world, congenital heart disease has surpassed rheumatic fever as a major cause of maternal heart disease in pregnancy [5]. Predictors of cardiac complications during pregnancy include prior cardiac events, prior symptomatic sustained dysrhythmia, New York Heart Association class  $>2$ , aortic or mitral valve obstruction and a Left Ventricular Ejection Fraction (LVEF) less than 40% [11].

Pregnant women present with new onset heart failure as a result of the increasing demands on the heart during pregnancy or as an exacerbation of primary heart disease. Typically, patients exhibit dyspnea on exertion, orthopnea and lower extremity edema. History, physical exam, electrocardiogram and echocardiography are used to confirm the diagnosis. Brain Natriuretic Peptide (BNP) is helpful in ruling out the diagnosis of decompensated heart failure. A value less than 100 pg/ml is considered normal. Although pregnant patients demonstrate elevated BNP levels due to increased blood volumes, the absolute value is expected to remain less than 100 pg/ml in non-pathologic cases [12]. There is no significant physiologic change in BNP levels throughout pregnancy as well as the postpartum period [12].

## Pulmonary Hypertension

Pulmonary hypertension of any cause is considered a contraindication to pregnancy and poses a significant risk of maternal death [3]. Some women are diagnosed during pregnancy or refuse to adhere to obstetrical advice and may present with an exacerbation during pregnancy. The normal decrease in peripheral vascular resistance during pregnancy is restricted by the presence of pulmonary vascular disease. This leads to a progressive increase in lung plasma volume, which causes increased right ventricular load, and ultimately heart failure. Patients present with hypoxia, edema, chest pain, and syncope. They are at risk of sudden death from severe right heart failure and dysrhythmias leading to a 52% maternal mortality rate [13]. Medical therapy is focused on avoiding increased pulmonary vascular resistance and maintaining right ventricular preload.

## Cardiomyopathy

Peripartum Cardiomyopathy (PPCM) is defined as the development of heart failure from the end of the third trimester until five months postpartum in the absence of underlying cardiac disease or other identifiable cause. PPCM occurs in 1 of 1500 to 3000 pregnancies [4]. The etiology is unclear but potential causes include myocarditis, immunological dysfunction, genetic predisposition, and increased myocyte apoptosis [14]. Multiparity, advanced maternal age, multiple gestations, and preeclampsia are predisposing factors. These patients present with symptoms of left and right heart failure including marked dyspnea, orthopnea, and peripheral edema. The risk of intracardiac thrombus and potential for embolism increases with ejection fractions less than 35% and anticoagulation may be required in these cases [15]. Approximately half of the cases of PPCM will recover systolic function within six months [14]. Unfortunately, 20% will deteriorate to the degree of requiring heart transplantation or death.

In general the treatment of patients with heart failure is the same as that of non-pregnant patients. Severe failure may require either noninvasive or invasive ventilation. The medication strategies are similar with the notable exception of angiotensin-converting enzyme inhibitors which are teratogenic. There is extensive experience with

digoxin and it is considered a safe drug to use in pregnancy [16]. Nitrates, hydralazine, and furosemide have all been safely used in pregnancy.

## Hypertension

Hypertension in pregnancy is an important cause of maternal morbidity and mortality. Chronic hypertension, gestational hypertension, and preeclampsia complicate 12-22% of pregnancies [4]. Pregnancy-induced hypertension is distinguished from preeclampsia by a lack of proteinuria and often returns to normal within 3 weeks after delivery. Gestational hypertension complicates 6-7% of pregnancies leading to preterm delivery, preeclampsia, placental abruption, and intrauterine growth retardation [17]. Diabetes, cardiac disease, renal disease, advanced maternal age, obesity, and multiple gestations are risk factors for the development of gestational hypertension [17]. The etiology of gestational hypertension is not known, but is likely similar to that of essential hypertension, as affected women frequently develop hypertension and cardiovascular disease later in life [18].

The management of pregnant patients with incidental hypertension varies depending on the trimester of pregnancy. Patients presenting beyond the second trimester (and up to 6 weeks postpartum) with hypertension should have testing for preeclampsia including complete blood count, comprehensive metabolic panel, urinalysis, Lactate Dehydrogenase (LDH) and uric acid levels. These women should be transferred to an obstetrical unit for fetal monitoring. Patients with mild hypertension and no evidence of end-organ damage or lab abnormalities may not require any medications. Women with blood pressures over 160/110 mm Hg would benefit from antihypertensive therapy [19]. Labetalol is a first-line medication for treatment of maternal hypertension with no known adverse maternal or fetal outcomes. Calcium channel blockers such as nifedipine have also shown to be safe in pregnancy. Angiotensin-converting enzyme inhibitors, thiazide diuretics, and the beta blocker atenolol should not be prescribed in pregnancy. Patients with end organ damage or evidence of preeclampsia will need aggressive management including magnesium therapy, intravenous antihypertensive medications (labetalol and hydralazine) and ultimately delivery.

## Dysrhythmias

Cardiac conduction disorders can be exacerbated in pregnant women with established dysrhythmias and structural heart disease or can occur de novo. The etiology for the increase in dysrhythmias during pregnancy is unknown. It is postulated to be related to the hormonal and physiologic cardiac changes which occur during pregnancy such as atrial and ventricular stretch due to the increased intravascular volume and heart rate.

The complaint of palpitations is common in pregnant patients. In the majority of cases, there is no concomitant cardiac dysrhythmia suggesting that the patients' perception of palpitations may be related to physiologic changes such as an increased heart rate and cardiac output. One study examining the incidence of dysrhythmia in pregnant women without pre-existing cardiac disease demonstrated a higher incidence of atrial and ventricular ectopic beats in patients presenting with symptoms of palpitations [20]. Twenty percent were found to have greater than 10 premature ventricular contractions per hour. Six percent had greater than 100 premature atrial contractions per 24 hours. The incidence of ectopic activity was significantly reduced during postpartum cardiac monitoring. The study further compared symptomatic patients with asymptomatic patients and found a higher incidence of ventricular ectopic beats in the symptomatic patients.

In patients with structurally normal hearts and mild symptoms, no treatment other than reassurance is required [21]. Furthermore, patients should be advised to discontinue potential precipitants including stimulants, caffeine, alcohol, and smoking.

Research addressing the relationship between pregnancy and Paroxysmal Supraventricular Tachycardia (PSVT) has demonstrated that first onset PSVT during pregnancy is rare [22]. However, symptoms of PSVT are exacerbated during pregnancy in 22% of patients with the underlying condition. Overall, attacks of PSVT were not associated with any significant maternal or fetal hazard. The management of PSVT is unchanged in the setting of pregnancy. Beyond vagal maneuvers, intravenous adenosine has been safely used in pregnant women [21]. Beta blockers can be used but the American Heart Association recommends avoiding their use in the first trimester. Atenolol is the only beta blocker which is absolutely contraindicated in pregnancy and is designated as unsafe by the Food and Drug Administration. Catheter ablation is safe and recommended for treatment of poorly tolerated, refractory SVT [21].

Atrial fibrillation and flutter are less common than PSVT but are more likely to occur in women with structural heart disease and cardiomyopathy. Furthermore, thyroid dysfunction is more common in pregnancy and should be investigated if these dysrhythmias develop. Rate control is recommended with beta blockers or digoxin. Amiodorone should be avoided given the potential for fetal harm. Patients should have an echocardiogram to assess for presence of structural heart disease. Anticoagulation should be deferred to obstetrical and cardiology consultants. Electrical cardioversion can be performed at all stages of pregnancy and should be used in cases of hemodynamic compromise, or medication refractory tachydysrhythmias [16].

## Congenital Heart Disease

Congenital Heart Diseases (CHD) include a broad spectrum of cardiac defects leading to a range of morbidity from life threatening to asymptomatic. Advances in medicine have improved survival of women with CHD reaching reproductive age. Furthermore, one recent study has shown that women with CHD have similar motivations to conceive compared to women without CHD, and there was a tendency to underestimate the ultimate maternal and fetal risks associated with pregnancy [23]. These women require pre-conception counseling and skillful management during pregnancy, considering the woman's capacity to tolerate the hemodynamic changes of pregnancy. The most high risk CHDs in pregnancy include Eisenmenger syndrome, uncorrected coarctation of the aorta, Marfan syndrome with a dilated aortic root, severe aortic stenosis and single ventricle syndromes (with or without Fontan procedures) [24]. Acute heart failure, arrhythmias and premature labor are the most common complications [25]. Coarctation of the aorta and Marfan syndrome can predispose pregnant woman to aortic dissection [25]. CHD significantly increases the risk of thromboembolic events, with some studies suggesting an incidence of (1:50) [25]. As a result many of these patients may be on chronic anticoagulation and may present with life threatening hemorrhage. The unusual hemodynamics associated with Fontan (procedure directing systemic venous blood into the pulmonary artery) physiology mandate that the peripartum management be performed at a tertiary care center [23]. Children of women with CHD are at higher risk for fetal and perinatal mortality [25].

## Heart Valve Replacements

Biological heart valve replacement does not require anticoagulation

but may require replacement after 10-15 years [26]. Mechanical heart valves, on the other hand, do not require replacement but mandate long-term anticoagulation. Pregnancy physiology threatens the function of artificial heart valves. Pregnancy with mechanical heart valve poses the risks of heart failure, arrhythmia, infectious endocarditis, hemorrhage (related to anticoagulation therapy) and mechanical valve thrombosis [27]. The management of pregnant women with mechanical valves is a significant challenge, because there are no controlled clinical trials to provide guidelines for optimal antithrombotic therapy [27].

## Cardiac Arrest

Cardiac arrest in pregnancy is an uncommon catastrophic event and is estimated to occur in 1 out of 30,000 pregnancies in the United States [16]. The causes of cardiac arrest are varied but in general can be classified into three major categories: Hypovolemia, pump failure, and obstruction. Hypovolemia can be caused by hemorrhage, trauma, sepsis, or aortic dissection. Pump failure can be due to myocardial infarction, cardiomyopathy, or unstable dysrhythmias. Obstruction refers to pulmonary embolism, amniotic fluid embolism, or pericardial tamponade.

Several modifications to standard cardiopulmonary resuscitation are necessary in pregnant patients. The primary approach of addressing circulation with Chest compressions followed by Airway and then Breathing and Defibrillation (C-A-B-D) is unchanged. However, the physiologic changes of pregnancy present several unique differences in the cardiopulmonary resuscitation efforts. Compression of the inferior vena cava by a gravid uterus can result in decreased venous return and cardiac output. The traditional left lateral tilting of the patient with hypotension or fetal distress poses a challenge to the performance of effective chest compressions. Therefore, the recommended technique is to manually displace the uterus to the left allowing the patient to remain in the supine position [16].

Venous access should be established above the diaphragm. Medications delivered via the femoral vein and other lower extremity access sites may not reach the maternal heart [28,29]. Chest compression should be performed higher on the sternum in order to compensate for the displacement of the diaphragm by the gravid uterus [16]. There are no changes in the medications used in advance cardiac life support during pregnancy. Perimortem Cesarean Delivery (PCD) leads to decreased aorta and vena cava compression and increases maternal cardiac output by up to 25% [29]. Optimal maternal and fetal outcomes occur if the procedure is performed within the first 5 minutes after the arrest. PCD is not indicated in women with a gestational age less than 20 weeks [29]. Cardiopulmonary resuscitation should not be interrupted during PCD.

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