INTRODUCTION

Cardiac emergencies during pregnancy are uncommon but have significant morbidity and mortality when they arise. Management of these high-risk patients involves close coordination of care between emergency medicine providers, obstetrics, and cardiovascular medicine. This article discusses management of chest pain syndromes in pregnancy, including acute myocardial infarction (AMI) and spontaneous coronary artery dissection (SCAD). An overview of the medical management of hypertensive crisis, mitral stenosis (MS), and acute heart failure (HF) are discussed.

CHEST PAIN SYNDROMES IN PREGNANCY

Chest pain syndromes in a pregnant woman may be the result of conditions that can range from benign to life-threatening, including AMI, SCAD, aortic dissection, pulmonary embolism, and hypertensive crisis. AMI and SCAD are highlighted.

Acute Myocardial Infarction in Pregnancy

AMI in pregnancy is an uncommon event with significant maternal, fetal, and neonatal morbidity and mortality. The risk of AMI is 2 to 3 times higher in pregnant women relative to nonpregnant women of reproductive age. As pregnancy rates in women older than 40 years have increased in recent years, the incidence of AMI in pregnancy has also increased. Epidemiologic data gathered from a report of births from a 10-year period between 1991 and 2000 in California revealed an incidence rate of 1 in 35,700 deliveries, and the incidence rate increased over the study period. In that study, compared with women who did not have an AMI in pregnancy, those women with an AMI were more likely to be older (30% were older than age 35 years...
compared with 10% in the non-AMI group), multiparous (78% compared with 61%), non-Hispanic white (40% compared with 35%), or African Americans (15% compared with 7%). In another study of 125 documented cases of AMI in pregnancy, the highest incidence was seen to occur in the third trimester and in multigravidas older than 33 years of age. AMI was most commonly located in the anterior wall. The maternal death rate was 21% and death tended to occur most often at the time of AMI or within 2 weeks of the infarction, and was usually related to labor and delivery. Coronary atherosclerosis with or without intracoronary thrombus was found in 43% of patients, whereas coronary thrombus without atherosclerotic disease was found in 21% of patients. Coronary dissection was seen in 16%, and 29% of patients had normal coronary arteries. Another review of 103 cases from 1995 to 2005 showed that coronary atherosclerosis was present in only 40% of cases, with the remaining cases consisting of coronary artery dissection (27%), thrombus in a normal coronary artery (8%), coronary artery spasm (2%), emboli (2%), and normal coronary arteries (13%). These studies suggest that atherosclerosis is less often the direct cause of AMI than in the general population, although some risk factors seem to overlap. Additionally, there seems to be a relatively high incidence of coronary artery dissection in this population.

Risk factors

Risk factors for AMI in pregnant women are similar to those in the population in general, which includes age older than 35 years, hypertension, diabetes mellitus, obesity, smoking, and lipid disorders (Box 1). The Nationwide Inpatient Sample for the years 2000 to 2002 evaluated all pregnancy-related discharges and a total of 859 discharges included a diagnosis of AMI with a rate of 6.2 per 100,000 deliveries. The odds of AMI in pregnant women were 30-fold higher for women older than 40 years of age than for women younger than 20 years of age. Demographic features associated with myocardial infarction (MI) included hypertension, with an odds ratio (OR) of 21.7, thrombophilia with an OR of 25.6, diabetes mellitus with an OR of 3.6, smoking with an OR of 8.4, transfusion with an OR of 5.1, and postpartum infection with an OR of 3.2. In that study, there were 44 deaths due to AMI (case fatality rate of 5.1%) and a mortality rate of 0.35 per 100,000 deliveries.

Clinical presentation

Pregnant women with AMI present with signs and symptoms similar to the general population, with complaints of anterior chest pain or epigastric discomfort. Shortness of breath and nausea may also be present. There is some evidence that women more often present with atypical features such as dyspnea or nausea without chest discomfort, syncope, palpitations, or cardiac arrest, and it is unclear whether pregnancy affects the clinical presentation of MI. In a 2014 study of 150 subjects with pregnancy-associated MI, 75% of women presented with an ST-segment-elevation MI and 25% with a non-ST-segment elevation MI. The MI involved the anterior wall in 69% of cases. For both types of MI, 50% of cases occurred after delivery and, for the remainder, the frequency increased with each trimester.

Diagnosis and management

As with cases in the nonpregnant general population, diagnosis is made by ischemic symptoms, such as angina, electrocardiographic abnormalities, and elevation in cardiac biomarkers. Management of AMI in pregnant women is similar to that in the general population with the notable exception to avoid teratogenic agents, such as angiotensin inhibitors and statins. Standard recommended drug therapy in AMI for nonpregnant patients includes morphine, beta-blockers, nitroglycerin, heparin; and antiplatelet therapy with aspirin, clopidogrel, ticagrelor, or prasugrel, among others. These guideline-recommended drug therapies are beneficial for the gravid mother with AMI. However, only limited information is available on fetal safety for some of these drugs. A review of 150 patients with pregnancy-associated MI evaluated the use of medical therapy and coronary intervention. Coronary angiography was performed in 129 patients, and 5 patients developed acute coronary dissection during the procedure as a result of intracoronary

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Box 1
Risk factors for acute myocardial infarction in pregnancy

Older age (>35 years)
Multiparity
African American race
Third trimester
Hypertension
Diabetes mellitus
Obesity
History of tobacco use
History of lipid disorder

Data from Refs.1–5
contrast injection. Four patients had urgent bypass surgery and 2 patients required left ventricular assist device placement. In that study, there was a high incidence of iatrogenic coronary dissection related to either intracoronary angiography or coronary stenting, resulting in the need for multiple stents, urgent coronary bypass surgery, and death. Percutaneous coronary intervention with the goal of early reperfusion is still strongly recommended in both nonpregnant and pregnant patients. However, the data reported by Elkayam and colleagues suggest that a noninvasive approach to the management of stable, low-risk women with pregnancy-associated AMI should be considered, and that if coronary angiography is performed, catheter entry into the coronary ostia should be done carefully, followed by low-pressure intracoronary contrast injections to reduce risk of iatrogenic coronary dissection.

**Spontaneous Coronary Artery Dissection**

SCAD is gaining recognition as an important cause of MI, especially in young women. The first report of SCAD was of a 42-year-old woman who died unexpectedly following repetitive vomiting that elicited rupture of the coronary artery from dissection of an atheromatous aneurysm. SCAD is defined as the spontaneous separation of the coronary artery wall, not related to trauma, iatrogenesis, or atherosclerosis (nonatherosclerotic SCAD). It is characterized by the presence of blood or thrombus in a false lumen, usually occurring in the outer third of the vessel media. The false lumen or dissection flap may then cause compression of the coronary artery lumen, resulting in obstruction or restriction of flow within the true lumen, and myocardial ischemia or infarction. It is thought that the process of dissection is initiated in the media and adventitia. Bleeding within the wall of the vessel alters the tolerance of the endothelium to shear stress, resulting in an intimal tear. Patients can present with coronary hematoma alone, with no evidence of an intimal flap.

Pregnancy-related SCAD is a rare and potentially lethal complication of pregnancy that can occur in any trimester during pregnancy and even months postpartum. In early retrospective studies, approximately 1 in 16,000 pregnancies in the United States is complicated by AMI and up to 25% of these are due to SCAD. In more modern series, the percentages were much less with pregnancy-related SCAD accounting for less than 5% of SCAD cases. SCAD occurs most commonly in the left anterior descending artery. Although SCAD in pregnancy is not typically associated with traditional risk factors for coronary artery disease (eg, smoking, hypertension, hyperlipidemia, diabetes, and family history), it dramatically increases the risk of AMI during pregnancy, especially in patients without risk factors. It has been suggested that coronary arterial dissection during or early after pregnancy is related to structural changes in the intima and media of the arterial wall that are produced by hemodynamic, as well as hormonal, changes (estrogen can increase release of matrix metalloproteinase and progesterone can lead to loss of normal corrugation of elastic fibers). There may also be an association with increased shear stress, such as with exercise, sneezing, or with cocaine abuse. SCAD has also been described in association with heritable connective tissue disorders such as Marfan syndrome and Ehlers-Danlos syndrome. Urgent coronary angiography is indicated to establish the diagnosis and to determine the appropriate therapeutic approach.

**Medical management**

Anecdotal evidence suggests that, in patients without ongoing ischemia and with preserved thrombolysis in myocardial infarction (TIMI) 3 flow (normal flow, which fills the distal coronary bed completely on coronary angiogram), these dissections could be managed without intervention. In a systematic review of the literature, 16 women were identified with pregnancy-related SCAD and in 31% complete resolution of the dissection was achieved with only medical therapy, including aspirin, clopidogrel, and beta-blockers.

**Percutaneous intervention**

There are several challenges to percutaneous intervention in SCAD. The injection of contrast may increase pressure in the false lumen and cause more compression (and resulting obstruction) of the true lumen. During an intervention, the guidewire could enter the false lumen and extend the dissection. Long segments of stent may be required to completely exclude the false lumen and restore the true lumen to its normal size. A strategy of using optical coherence tomography or intravascular ultrasound to ensure accurate guidewire placement and to target the entry point of the hematoma has been suggested to limit necessity of long areas of stenting.

**Surgical management**

Coronary artery bypass has been performed in patients presenting with SCAD causing acute ischemia or cardiogenic shock. A surgical strategy can be considered if there is proximal dissection of multiple vessels with good distal target vessels or if a percutaneous strategy cannot be
pursued, such as in a total occlusion that has rendered it difficult to enter the true lumen with a guidewire.9

AMI in pregnancy is an uncommon complication with significant maternal and fetal morbidity and is now being increasingly recognized, particularly in the case of SCAD.

HYPERTENSIVE EMERGENCIES IN PREGNANCY

Hypertensive emergency in adults is characterized by significantly elevated blood pressure, defined as systolic blood pressure (SBP) greater than or equal to 180 mm Hg or a diastolic blood pressure (DBP) of greater than or equal to 120 mm Hg with signs or symptoms of acute end-organ dysfunction. Clinical signs and symptoms may include neurologic symptoms, such as headache, stroke, and flame hemorrhages, as well as nausea and vomiting, which may indicate increased intracranial pressure. Patients may complain of chest discomfort and dyspnea due to pulmonary edema. Laboratory data may reveal abnormal renal indices and elevated cardiac biomarkers. Prompt but cautious reduction of blood pressure is essential to patient outcomes. Hypertensive emergency in pregnancy generally belongs in 1 of 2 categories: (1) gestational hypertension and preeclampsia, or (2) exacerbation of preexistent chronic hypertension. Because acute management of hypertensive emergency in pregnancy is identical regardless of etiologic factors, the following discussion focuses on the former.

Gestational Hypertension and Preeclampsia: Definitions

Gestational hypertension and preeclampsia or eclampsia are hypertensive disorders induced by pregnancy and both disease spectrums typically resolve postpartum. Gestational hypertension is the most common cause of hypertension in pregnant women. It is defined by new-onset hypertension detected after 20 weeks of gestation in the absence of proteinuria or other diagnostic features of preeclampsia. Preeclampsia is the syndrome of new onset of hypertension, proteinuria, or end-organ dysfunction after 20 weeks in a previously normotensive woman. Definitive treatment of preeclampsia is delivery. Without delivery, the mother is at increased risk of complications such as seizures, placental abruption, thrombocytopenia, cerebral hemorrhage, pulmonary edema, liver hemorrhage, and acute kidney injury. However, the risks of preeclampsia to the mother must be weighed against the risk of premature delivery of the fetus. Detailed guidelines regarding gestational age and degree of severity of preeclampsia in the mother and recommendations for or against delivery are reviewed in the Report of the American College of Obstetricians and Gynecologists Task Force on Hypertension in Pregnancy.21

Management

In the setting of mild hypertension (defined as <150/100 mm Hg) in the context of preeclampsia, antihypertensive therapy may not necessarily be required based on data from a Cochrane Database review.22 Included in the review were 4723 women who showed a 50% reduction of the risk of developing severe hypertension associated with the use of antihypertensive drugs. However, there was little evidence of a difference in the risk of preeclampsia and no clear effect on risk of fetal death, preterm birth, or babies who were small for their gestational age.22 This study concluded that it is unclear if antihypertensive drug therapy for mild to moderate hypertension during pregnancy is necessary. Moreover, the benefit of reduction of the risk of severe hypertension may not be enough to warrant exposing the fetus to the potential adverse effects of drugs.

Severe hypertension should be treated to prevent maternal morbidity and mortality from vascular complications such as acute coronary
syndromes, stroke, and HF. Optimal blood pressure thresholds are unclear, with some clinicians recommending initiation of therapy at SBP greater than or equal to 150 mm Hg or DBP greater than or equal to 100 mm Hg, and others recommending initiation at SBP greater than or equal to 160 mm Hg or DBP greater than or equal to 105 to 110 mm Hg. In an analysis of 24 women with stroke related to preeclampsia, 4% had SBP greater than 155 mm Hg and less than 160 mm Hg, supporting a lower threshold for initiating therapy.

Intravenous labetalol or hydralazine are first-line agents for acute therapy for severe hypertension in pregnancy. Labetalol is effective, has a rapid onset of action, and has a good safety profile. A prospective, randomized, single-blind study of nicardipine and labetalol was conducted and showed overall efficacy to be comparable, although the decrease in blood pressure was greater with nicardipine than with labetalol (P<.05). Nicardipine and labetalol are effective and safe in the initial treatment of severe hypertension of pregnancy. Table 1 highlights the different antihypertensive regimens, which are safe and effective in the treatment of acute severe hypertension in pregnancy.

For management of chronic hypertension treated with antihypertensive medication, it is suggested by the Report of the American College of Obstetricians and Gynecologists Task Force on Hypertension in Pregnancy that blood pressure levels be maintained between 120 mm Hg to 160 mm Hg systolic and 80 mm Hg to 105 mm Hg diastolic. Recommended pharmacologic agents for the initial treatment of pregnant women with chronic hypertension include labetalol, nifedipine, or methyldopa, above all other antihypertensive agents.

**MANAGEMENT OF MITRAL STENOSIS IN PREGNANCY**

MS is the most common valvular lesion complicating pregnancy and almost exclusively results from rheumatic heart disease. Specific physiologic stresses of pregnancy, including increased plasma volume and increased heart rate, can precipitate decompensation in patients with severe MS by exacerbating the diastolic gradient across the narrowed mitral valve, leading to increases in left atrial pressure. Although rarely encountered in developed nations due to reduced incidence of rheumatic heart disease, MS is associated with increased maternal and fetal morbidity and mortality.

**Clinical Presentation of Mitral Stenosis**

Symptoms of MS manifest in pregnancy as a result of flow-related increases in the gradient across a stenotic mitral valve. Elevated left atrial pressure results and leads to a multitude of symptoms, including dyspnea, decreased exercise capacity, orthopnea, paroxysmal nocturnal dyspnea, pulmonary edema, and atrial arrhythmias. In a case-control study, more than 70% of subjects with mild to severe MS experienced clinical deterioration in New York Heart Association functional class during pregnancy. Maternal morbidity is typically more significant in moderate and severe MS, compared with mild MS. Notably, however, maternal mortality is fairly low in MS, with no cases of maternal mortality reported in 2 North American case series totaling 124 pregnancies in MS subjects. Fetal outcomes are also affected in

<table>
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<tr>
<th>Drug</th>
<th>Starting Dose</th>
<th>Subsequent Dosing</th>
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| Labetalol  | 20 mg IV over 2 min or Continuous IV infusion of 1–2 mg/min | • 40 mg IV over 2 min if BP above goal at 10 min
• 80 mg IV over 2 min if BP above goal at 20 min
• Repeat 80 mg IV q 10 min if BP not at goal at 30 and 40 min
• Maximum cumulative dose: 300 mg |
| Hydralazine| 5mg IV over 1–2 min | • Measure BP q 20 min
• If BP above goal at 20 min, give 5–10 mg IV over 2 min
• If BP above goal at 40 min, give 10 mg IV over 2 min
• Maximum cumulative dose: 20 mg |
| Nifedipine ER | 30 mg oral | Can redose if target BP not achieved in 1–2 h |
| Nicardipine | Continuous IV infusion of 3–9 mg/h | Avoid rapid titration due to delayed onset of action and risk of overshooting dose. |

**Table 1**

Pharmacologic therapy for acute severe hypertension in pregnancy

Abbreviations: BP, blood pressure; IV, intravenous.

MS. In 1 study, 44% of women with severe MS experienced preterm labor and 33% experienced intrauterine growth retardation.

**Diagnosis**

Although rare, recognition of MS in the emergency department (ED) is critical to enable prompt and appropriate management. Clinical suspicion should be raised in a patient with a history of rheumatic fever presenting with unexplained HF symptoms such as dyspnea and pulmonary edema or new hemoptysis. Physical examination may reveal a diastolic rumble murmur on examination and a chest radiograph may demonstrate an enlarged left atrial silhouette and pulmonary venous congestion. Transthoracic echocardiogram is an effective, available, and efficient tool to definitively diagnose MS, which can demonstrate restricted mitral leaflet motion and elevated mitral valve inflow gradients (Fig. 2). Transesophageal echocardiography is

![Figure 2](image-url)

**Fig. 2.** Continuous wave Doppler across the mitral valve in a patient with severe MS before balloon valvuloplasty (A) and after valvuloplasty (B) that resulted in a reduction in mitral valve gradients. (Courtesy of University of Maryland Medical Center Echocardiography Laboratory, Baltimore, MD.)
useful for obtaining accurate mitral valve area measurements by planimetry (Fig. 3) and to assess if the valve is favorable for mitral balloon valvuloplasty.

Management

Initial therapy for severe MS during pregnancy centers on medical therapies, including beta-blockers to reduce heart rate and increase time in diastole for left atrial off-loading, diuretic therapy to reduce volume, and bed rest in certain cases to reduce heart rate and cardiac output. Metoprolol is the treatment of choice, preferred over atenolol, which has been associated with intrauterine growth retardation. Additionally, due to the frequency of developing atrial fibrillation with MS and the high potential for clot formation in the prothrombotic state of pregnancy, anticoagulation is often prescribed to pregnant patients, with subcutaneous heparin being the preferred agent. In severe MS that is refractory to medical therapy, percutaneous mitral balloon valvuloplasty may be indicated and, if required, is ideally performed after the second trimester to avoid radiation exposure during organogenesis (see Figs. 2 and 3). Based on higher-risk features, such as high gradients with preexisting symptoms, certain patients may be counseled to undergo valvuloplasty before conception.

Vaginal delivery is preferred in patients with MS with recommendations for delivery including shortening of the second stage of labor with the use of outlet forceps or vacuum extractor, epidural anesthesia for pain relief to reduce myocardial demand, and use of pulmonary artery catheters to guide fluid management and potential need for vasopressor therapy.

MANAGEMENT OF ACUTE HEART FAILURE IN PREGNANCY

The pregnant state is associated with substantial hemodynamic changes, including a 30% to 50% increase in cardiac output and blood volume. These added hemodynamic demands on a patient with a previous history of HF can lead to clinical decompensation. Management of the pregnant patient with acute HF involves a careful consideration of risks and benefits of each drug in the pharmacologic armamentarium on both mother and fetus. Pregnancy itself can be associated with increased risk of arrhythmias. Women with a history of structural heart disease, such as MS, can develop arrhythmias during pregnancy that precipitate HF.

Decompensated HF can present during pregnancy or in the peripartum period, and can be the result of preexisting cardiac disease or disease acquired during the pregnancy. Peripartum cardiomyopathy is a unique disease entity that shares some genes common to idiopathic dilated cardiomyopathy and presents any time from the last month of pregnancy and up to the first 6 months.
Heart Failure Definitions

HF is a clinical condition that can occur in patients with a reduced left ventricular ejection fraction (LVEF) less than or equal to 40%, termed HF with reduced ejection fraction (HFrEF). It may also occur in patients with preserved LVEF, termed HF with preserved ejection fraction (HfPEF), with LVEF greater than or equal to 50%. HF with LVEF of 41% to 49% is considered borderline HfPEF.30

Presentation of Acute Heart Failure

Acute HF may present with persistent cough, progressively worsening dyspnea, and volume overload. Initial evaluation of a patient presenting with acute HF should include a focused history and physical examination with attention to lung and cardiac auscultation, assessment of jugular venous pressure, extremity examination to assess for edema, strength of peripheral pulses, and a mental status examination to assess for perfusion. Additional testing includes echocardiography, which may diagnose previously undetected structural heart disease (valvular abnormalities or worsening ventricular function). An electrocardiogram (ECG) can assess for arrhythmias that may predispose to development of acute HF (eg, atrial fibrillation) or may be a manifestation of acute HF (eg, sinus tachycardia). The ECG may also reveal left or right ventricular hypertrophy, atrial enlargement, or myocardial ischemia. Usefulness laboratory testing includes a complete metabolic panel, complete blood count, and measurement of cardiac troponin. Brain natriuretic peptide (BNP) levels may be helpful for prognostication and risk stratification. If in the normal range, HF can often be ruled out as a diagnosis (with the caveat that median BNP levels during normal pregnancy are approximately double those in nonpregnant controls).31

Counseling

Although continuity of care in an ED setting may not afford the opportunity for patient counseling, many preexisting HF patients may learn about their pregnancy in an ED setting, or a pregnant patient presenting with acute dyspnea may learn of a new diagnosis of HF. In either case, counseling of the HF patient regarding the expected prognosis, and potential risks of pregnancy should be pursued. The risk of clinical decompensation during pregnancy and the peripartum period depends on the severity of systolic dysfunction. Avoidance of pregnancy is advised in women with a dilated cardiomyopathy with LVEF less than 20% due to high risk of maternal mortality.32 For these high-risk women who become pregnant, termination of pregnancy should be discussed.32

Management in the Emergency Department

Management of acute HF in the pregnant patient in an ED setting is similar to the nonpregnant patient and should focus on relief of symptoms, treatment of precipitating factors (eg, thyroid disorders, infection, and rhythm disturbances), and improvement of hemodynamic status with diuretics and vasodilators to decrease venous pressure and afterload. Once a patient is stabilized with optimized hemodynamics, continuation or initiation of chronic medical therapies that improve mortality, such as beta-blockers, should be considered. Pharmacologic agents used to improve symptoms in general population HFrEF patients include a diuretic, beta-blocker, angiotensin-converting enzyme (ACE) inhibitor, angiotensin II receptor blocker (ARB), angiotensin receptor-neprilysin inhibitor (ARNI), and digoxin. However, due to their safety profiles, ACE-inhibitors, ARBs, and ARNIs are contraindicated for the treatment of HFrEF in pregnant patients (Fig. 4).33–36

Acute Heart Failure Therapy

The mainstay of acute HF therapy in the pregnant patient includes supplemental oxygen and pharmacologic therapy, with diuretics and vasodilators if blood pressure allows. Vasodilator therapy improves cardiac output in moderate to severe HF by inducing afterload reduction. For the pregnant patient with severe decompensated HF and stable or elevated blood pressure, intravenous vasodilator therapy with nitroglycerin or nitroprusside should be administered. Should pronounced afterload reduction be required, such as in hypertensive emergency with pulmonary edema, acute aortic regurgitation, or acute mitral regurgitation, nitroprusside should be considered over nitroglycerin. Intravenous afterload reduction should be cautiously undertaken in the pregnant state given risk of fetal heart rate deterioration with a rapid and profound drop in maternal blood pressure. Hemodynamic monitoring of the patient with an arterial line and continuous fetal monitoring should be used.
Angiotensin Inhibition

Although normally a vital medication in the treatment of HFrEF in the general population, angiotensin inhibition is contraindicated in pregnant patients. ACE inhibitor, ARB, and ARNI (a combination of ARB plus neprilysin inhibitor) are contraindicated during pregnancy due to their association with fetal renal failure, oligohydramnios, pulmonary hypoplasia, and other effects highlighted in Box 1.33–36

Aldosterone Antagonists

Aldosterone antagonists are also standard HFrEF medications that are contraindicated in pregnancy. Although the aldosterone antagonists spironolactone and eplerenone have been shown to prolong survival and reduce the risk of hospitalization in selected patients with HFrEF (ejection fraction 35%), these agents are not recommended in pregnancy due to lack of safety data in pregnancy and evidence from an animal study (rat model) that showed an antiandrogenic effect on the male fetus.38

Vasodilators

Hydralazine and nitrate therapy are vasodilators of choice during pregnancy given the contraindication of ACE-inhibitor and ARB.32 As a treatment of hypertension, hydralazine’s safety for mother and fetus has been demonstrated.39 The combination of hydralazine plus nitrate therapy should be used instead of ACE inhibitor, ARB, or ARNI in pregnant HF patients who are hypertensive or have evidence of congestion and decompensated HF.

Diuretics

Diuretics are a mainstay of acute HF therapy for symptomatic relief of pulmonary edema and peripheral edema. For HF, loop diuretics are preferred over thiazides. Potential maternal complications of loop diuretic use are similar to nonpregnant patients, including volume contraction, metabolic alkalosis, hypokalemia, hyponatremia, and pancreatitis. Risks to the fetus are based on concern for intravascular volume contraction and reduced perfusion to the placenta.

Digoxin

Digoxin, although not first-line therapy for acute decompensated HF nonpregnant patients due to lack of mortality benefit, can be a useful agent for HF in pregnancy. In contrast to more mainstay therapies, such as angiotensin blockade, digoxin is generally safe in pregnancy and may improve symptoms exacerbated by pregnancy, such as persistent fatigue, dyspnea, and exercise intolerance. Digoxin levels in breast milk are low and it is generally deemed safe to use during lactation.

Beta-Blockers

Management of beta-blocker therapy in the pregnant patient with acute decompensated HF is similar to that in the nonpregnant patient. It should not be initiated while the patient is acutely decompensated and should be started low dose on discharge once the patient is clinically improved. Agents that are beta-1 selective (eg, metoprolol) are preferable because they are less likely to interfere with beta-2 mediated uterine relaxation.32 Atenolol should be avoided in pregnancy due to its association with intrauterine

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**Fig. 4. Safety profile of various commonly used cardiovascular agents. (Data from Refs. 33–36,38)**
growth restriction. In most patients already on chronic beta-blocker therapy who present to the ED in acute decompensated HF, therapy should generally be continued. Important exceptions include patients with marked volume overload or low cardiac output, in which case beta-blockers may need to held or reduced to prevent development of cardiogenic shock.

Intravenous Inotropes

Pregnant patients with clinical signs of cardiogenic shock (eg, cool extremity examination, renal failure, transaminitis, and elevated lactic acid) may benefit from intravenous inotropic therapy. Although inotropes have never been shown to improve mortality and may increase risk for ventricular arrhythmias, inotropes can help preserve end-organ function while definitive treatment is pursued. Dobutamine (a beta-agonist) and milrinone (a phosphodiesterase inhibitor) have both inotropic and vasodilator properties that may be beneficial in a low cardiac output state with signs of shock. In the pregnant patient with acute or chronic HF that is refractory to treatment, mechanical circulatory support with a left ventricular assist device or venoarterial extracorporeal membrane oxygenation may be required. A successful pregnancy has been documented while being supported by a left ventricular assist device.

SUMMARY

Pregnancy poses challenges in patients presenting with cardiovascular emergencies such as AMI, acute heart failure (due to exacerbation of chronic heart failure, new cardiomyopathy, or symptomatic severe MS) and hypertensive emergencies. Prompt recognition, diagnosis, and knowledge of the medication safety profiles of cardiovascular drugs are of paramount importance for appropriate treatment in this special patient population to ensure the health of both mother and fetus.

REFERENCES


