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Acute Myocardial Infarction in Pregnancy

An Update

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ABSTRACT

Despite current trends that indicate increasing incidence, acute myocardial infarction remains an uncommon event in pregnant women, yet an important contributor to perinatal morbidity and mortality. Diagnosis and treatments represent a complex challenge during pregnancy, and timeliness and coordination of both are critical. This article reviews the comprehensive, collaborative approach necessary for management of acute myocardial infarction during pregnancy to optimize outcomes for the woman, neonate, and family.

Key Words: acute myocardial infarction, cardiac disease, high-risk obstetrics, pregnancy

Acute myocardial infarction (AMI) remains a relatively rare occurrence, estimated to be 6.6 per 100 000 women during pregnancy, yet carries significant risk of perinatal morbidity and mortality.[1] The maternal mortality rate with AMI during pregnancy or the puerperium is reported to be as high as 37%.[2] Concomitant neonatal mortality is most closely linked to maternal mortality and noted to be from 13% to 17%.[3,4] Pregnancy has also been demonstrated to increase the risk of AMI as many as three to four times as compared with nonpregnant women of childbearing age.[1,3,4] As more women delay childbirth until later in life, and preexisting cardiovascular risks such as obesity, diabetes, and chronic hypertension become more prevalent, the incidence of AMI in pregnancy may increase. The purpose of this article is to review care principles for women experiencing AMI in pregnancy.

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RISK FACTORS

Risk factors for AMI in pregnant women are the same as the general population and include age greater than 35 years, hypertension, diabetes mellitus, obesity, smoking, and dyslipidemia.[5] The cardiac effects of smoking may be exacerbated during pregnancy because of the increased vascular reactivity effects of estrogen and progesterone.[1] Differences in presentation of AMI between younger and older (>35 years) women are related to differences in pathogenesis. Nonatherosclerotic mechanisms, such as coronary spasm and thrombosis, are most commonly reported in younger women, while atherosclerotic disease is more common in older women.[6] The presence of other risk factors such as diabetes, hypertension, hyperlipidemia, and oral contraceptive use is also more prevalent in older women. Women older than 35 years are at greatest risk for AMI in pregnancy or postpartum with cases occurring in women ranging from 16 to 45 years of age.[6–8]

Other risk factors for AMI in pregnancy are thrombophilias, including a history of thrombosis and antiphospholipid syndrome, pregnancy complications such as blood transfusion and postpartum infections, migraines, preeclampsia, collagen diseases, and cocaine use.[1,5,7] Systemic vasospasm and endothelial damage from preeclampsia can contribute to coronary artery ischemia.[1] Increased cardiac work resulting from postpartum hemorrhage may predispose a woman to AMI because of increased myocardial oxygen demand or coronary artery thrombosis secondary to compensatory hypercoagulability. Vascular inflammation associated with infection has been associated with increased risk for AMI, but this higher risk is more closely linked with

chronic conditions as opposed to acute infection.[1] Cocaine may precipitate an ischemic event by causing systemic and/or coronary artery vasoconstriction.[4]

Whether pregnancy alone is a risk factor for AMI is unclear.[9] Increased plasma volume and cardiac output in pregnancy may contribute to coronary ischemia and AMI. The hypercoagulability of pregnancy may predispose some women to thrombotic events. The hemodynamic and physiologic changes during pregnancy are most pronounced in the third trimester with most AMIs occurring during this time.[8]

ETIOLOGIES

The most common cardiac etiologies of maternal death are aortic dissection and AMI.[10] One cause of AMI in pregnancy is atherosclerosis, with or without intracoronary thrombus, but less often than the general population. Nonatherosclerotic causes include coronary artery spasm, coronary artery dissection, thrombus/embolism in a normal coronary artery, arteritis, and in situ thrombosis from thrombophilias.[4,8]

PHYSIOLOGIC CHANGES

Pregnancy, labor, and birth

There are substantial physiologic changes in the cardiovascular system during pregnancy, labor, birth, and into the postpartum period to meet the metabolic demands of both the pregnant woman and fetus. Knowledge and understanding of these changes are necessary to understand the pathophysiology and management principles of AMI in the pregnant woman. Increases in heart rate, blood volume, and stroke volume with resultant increased cardiac output intensify maternal myocardial demand for oxygen. Expected dilutional anemia and decreased diastolic blood pressure during pregnancy may affect myocardial oxygen delivery. During labor and birth, pain, anxiety, and uterine contractions increase oxygen consumption. Dramatic fluid shifts in maternal circulation after birth from relief of aortic-venal compression and loss of the placenta contribute to increased hemodynamic load. Pregnancy is a hypercoagulable state. Factors V, VII, VIII, IX, X, XII; prothrombin; and fibrinogen are increased, whereas the fibrinolytic system is depressed. This alteration in coagulation has implications for management with thrombolytic therapy.[11]

Pathophysiology of AMI

In an acute myocardial event, the damage to the heart muscle depends upon a number of factors: (1) the number of vessels and branches involved, (2) disease within the vessels, (3) the area(s) of myocardium supplied by the affected vessel(s), (4) the amount of collateral circulation to provide alternative perfusion to the affected region, and (5) the oxygen demands on the heart during and after the infarction.[11] Myocardial oxygenation is maintained through physiologic mechanisms to ensure appropriate coronary perfusion pressure, adequate volumes of oxygenated blood, and unobstructed coronary flow. When one or all of these mechanisms are challenged, myocardial ischemia may occur, yet a substantial deficit must exist before there is presentation of symptoms. A major coronary vessel has typically lost 70% in diameter before angina occurs.[12] As such, the presence of angina is an indicator of significant myocardial ischemia, tissue damage, and cellular death.

Oxygen consumption is determined by the metabolic rate; however, vascular diseases compromise the ability to deliver an adequate amount of oxygen for cellular metabolism. Oxygen delivery is not only dependent upon cardiac output (heart rate multiplied by stroke volume), but also by the oxygen-carrying capacity of the blood and the ability of the lungs to exchange respiratory gases. Tissues increase the rate of oxygen extraction in the presence of reduced oxygen delivery. Once limits of extraction are reached, tissues rely on anaerobic glycolysis for energy and release lactate as a metabolic by-product. Episodes of prolonged oxygen deprivation produce metabolic acidosis, followed by permanent tissue damage.

Myocardial tissue is perfused by the coronary arteries during ventricular diastole, which are dependent upon vascular distensibility and left ventricular function to maintain perfusion pressure. Atherosclerotic plaques from cholesterol deposits and platelet aggregation, hypertension, dissection, or coronary artery vasospasm decrease vascular distensibility. Reduced vascular distensibility contributes to increased energy expenditure in the left ventricle. In the presence of a chronic condition, ventricular hypertrophy may result. Ventricular hypertrophy and associated increased muscle mass increase ventricular stroke work, heart rate, and myocardial oxygen demand. Complications arising from AMI are presented in Table 1.

DIAGNOSIS

Diagnosis of AMI can be made on the basis of clinical presentation of ischemic symptoms supported by serial electrocardiograms (ECG) and evaluation of cardiac troponins. Diagnosis of AMI during pregnancy may be confounded with the low index of suspicion in childbearing women.

Table 1. **Potential complications of acute myocardial infarction**[a]

Complication	Description
Electrical–cardiac dysrhythmias	
Premature ventricular beats	Associated with acute myocardial ischemia; treat with lidocaine if symptomatic
Ventricular tachycardia	Associated with acute myocardial ischemia; treat with lidocaine
Bradycardia	More common in inferior and posterior AMI; intense vaginal stimulation with SA and AV node ischemia
Mechanical	
Pericarditis	Symptoms of chest pain unresponsive to antianginal agents and do not radiate; acute/early onset of delayed Dressler syndrome
Cardiac tamponade	Rare complication due to build-up hemorrhagic pericardial fluid in anticoagulated patients
Left ventricular failure	Clinical symptoms usually present with 20% left ventricular damage; consider when sinus tachycardia persists > 48 h
Cardiogenic shock	Shock usually from pump failure; rapid myocardial reperfusion is essential; increase filling pressures to optimize cardiac output
Papillary muscle dysfunction or rupture	Usually occurs 2-10 days following posterior or inferior AMI; rapid onset of shock, heart failure, or pulmonary edema
Ventriculoseptal rupture	Occurs in 1% of all AMIs; predisposing factors are anterior septal wall damage, hypertension, advanced age, and first AMI
Anterior or lateral wall rupture	Symptoms of recurrent chest pain without ECG changes; high mortality rate; more common 3-6 days following AMI
Thromboembolism	Associated with large infarctions in anterior and apical areas

Abbreviations: AMI, acute myocardial infarction; AV, arterioventricular; ECG, electrocardiogram; LV, left ventricular; PVC, premature ventricular contraction; SA, sinoatrial.
[a]Adapted from Baird and Kennedy.[11]

Clinical presentation

Diagnosis of AMI in pregnancy is the same for nonpregnant individuals, yet the presentation can be confusing as typical symptoms such as radiating substernal chest pain radiating to jaw, neck or arms, diaphoresis, nausea, and exertional dyspnea are also common discomforts of pregnancy. Such typical symptoms, combined with a low index of suspicion of AMI in women of childbearing age, contribute obstacles to diagnosis. Women are more likely to present with atypical symptoms such as dyspnea, nausea without chest discomfort, sharp chest pain, palpitations, syncope, indigestion, epigastric pain, or cardiac arrest.[7]

Electrocardiography

Electrocardiographic readings are a critical tool for AMI assessment and diagnosis. Electrocardiogram leads provide a graphic representation of electrical activity in the heart and reflect changes according to the site of infarction. Note that physiologic changes of pregnancy affect ECG readings, and understanding these changes is essential for correct diagnosis. The gravid uterus displaces the diaphragm upward during the third trimester, causing a left or right axis deviation. Other normal ECG findings in pregnancy include Q waves in lead III, T wave inversion, or an increased R/S ratio in leads V_1 and V_2.[13] ECG findings of ST elevation or depression and inverted Q waves are indicative of ischemic changes.

The ECG findings reveal two groups of AMI: those with ST-segment elevation myocardial infarction (STEMI) and those with non-ST-segment elevation myocardial infarction (NSTEMI) (see Table 2). This distinction guides treatment strategies. Coronary artery dissection and coronary artery spasm are more uncommon causes of AMI in the general population, while AMI attributed to atherosclerotic causes is less common in pregnant women. Clinical presentation does not generally differ between the two, and the diagnostic approach is the same for all acute coronary syndromes.[14] Although uncommon, some women with documented AMI will have a normal ECG as evolution of the infarction may occur over the initial 48 hours.[7]

Chemical biomarkers

Uterine contractions can cause significant increases in cardiac markers such as myoglobin and serum creatinine kinase MB; therefore, troponins are recommended for diagnosis of myocardial injury in pregnant women.[15] Cardiac troponins I and T are sensitive and specific biochemical markers released from injured cardiac muscle. Elevation of troponins indicates myocardial tissue necrosis. Serum blood levels of troponins can be detected 4 to 6 hours after symptom onset from myocardial injury. However, levels may not be detectable soon after AMI; thus, diagnosis is not ruled out in the

Table 2. **Characteristics of STEMI and NSTEMI**

ST-segment elevation AMI (STEMI)	Non–ST-segment elevation AMI (NSTEMI)
ECG changes	
ST-segment elevation in ≥ 2 contiguous leads	ST-segment depression OR
A new or indeterminate left bundle block OR	T-wave inversions in ≥ 2 contiguous leads
ST depression in the right-sided precordial leads	
Vessel occlusion	
Usually indicates complete, persistent vessel occlusion	Usually indicates partial occlusion of the vessel
Pathophysiology	
Large epicardial coronary vessel involvement with significant resulting myocardial necrosis	Varying degrees of reduction coronary blood flow in combination with distal embolization of thrombotic material and coronary vasospasm resulting in myocardial necrosis
Management	
Early revascularization	Oxygenation
IV thrombolytics or PCI with stent placement	Pain medication
Prior to PCI medical management may follow NSTEMI principles	Treat tachycardia and hypertension
	Acute antiplatelet therapy
	Possible angiography and revascularization based on risk of medical treatment failure

Abbreviations: AMI, acute myocardial infarction; ECG, electrocardiogram; IV, intravenous; PCI, percutaneous coronary intervention.

presence of strong clinical suspicion.[7] Once elevated, troponin I levels will stay elevated for 1 to 2 weeks.[7] Unlike other laboratory values, troponin I is never increased above the upper normal limit in healthy pregnant women. The immediate postpartum period, anesthesia, and even surgical birth do not affect values and elevation is always considered abnormal.[7,15] Other conditions that might lead to elevated troponins include myocarditis, pericarditis, sepsis, and elevated pressures in the ventricles such as pulmonary embolism and hypertension. Reference ranges for troponin I may vary according to laboratory and method of measurement.

ACUTE COLLABORATIVE MANAGEMENT

Principles of AMI management do not generally differ in pregnancy and require a coordinated, collaborative approach to management. Obstetrics, cardiology, anesthesia, and nursing services are involved in planning and care delivery for the pregnant woman with AMI. Perinatal nursing plays a key role in maintaining team awareness of the physiologic changes in pregnancy that alter target hemodynamic and respiratory values and in planning for fetal assessment and intervention.[11]

Management, care setting, and transport decisions are individualized on the basis of maternal and fetal considerations, risks and benefits to both, and etiology of the infarction. Early therapy is focused on several goals: (1) relief of pain and anxiety, (2) reperfusion of affected vessels, (3) improving the balance between myocardial oxygen supply and demand, (4) initiation of antithrombotic therapy to prevent the formation of a secondary thrombus, (5) improving ventricular function, and (6) limiting the infarction size.[11] Timely recognition and treatment are the key to minimizing myocardial damage and optimizing maternal and fetal outcomes. Initial care measures include ensuring a patent airway, oxygen administration, peripheral intravenous access, laboratory analysis of electrolytes, hemoglobin and cardiac enzymes, Foley catheter placement, interventional therapy, and pharmacologic therapy to maintain hemodynamic and rhythm stability. STEMI and NSTEMI are managed differently because of the significance of vessel occlusion.

ST-segment elevation myocardial infarction

Women with complete vessel occlusion (STEMI) must undergo early revascularization by either intravenous thrombolytics or percutaneous coronary intervention (PCI) with stent placement. With PCI, imaging of coronary arteries is completed using contrast media, with placement of stents in affected vessels as indicated. There are two types of stents: bare metal and drug-eluting. Bare metal stents are endothelialized earlier, preventing early thrombosis. Drug-eluting stents release antiproliferative medications that decrease risk of restenosis, but delay endothelialization, requiring additional and prolonged antiplatelet therapy. In pregnancy, bare metal stents are generally preferred.[16] Complications of PCI with stent placement include arterial embolization, contrast toxicity, arrhythmia, and vascular injury. In addition, there may be concerns about timing/mode of birth, anesthesia options, and increased risk of hemorrhage due to risks of anticoagulation after stent placement. These risks may be managed with

platelet transfusion and use of narcotics or general anesthesia for pain relief if birth is unavoidable. There is an increasing trend toward stent placement as opposed to coronary artery bypass graft surgery for AMI in active labor due to the minimally invasive approach.[16]

Percutaneous coronary intervention is the preferred treatment, but if the institution is not capable of performing the procedure, the woman may be treated with recombinant tissue plasminogen activator (r-TPA). Systemic thrombolysis may be used during pregnancy in the absence of absolute contraindications including history of intracranial hemorrhage, ischemic stroke in the last 3 months, head trauma in the last 3 months, brain arterioventricular malformations or neoplasms, suspected aortic dissection, active bleeding or known bleeding disorder, major trauma, bleeding, or surgery in the last 3 weeks, severe uncontrolled hypertension, and neurosurgery in the last 3 months.[7] Relative contraindications include current use of anticoagulants and cardiopulmonary resuscitation for more than 10 minutes. Women who have received thrombolytics should not undergo any operative procedures for 10 days after administration. Thus, obstetric management decision may be complicated if the fetus is viable at or near the time of thrombolytic therapy. The use of thrombolytics also creates concern about allergic reactions, fibrinolytic effects on placental implantation, activation of circulating plasminogen as a trigger of preterm labor, and the development of reperfusion arrhythmias.[4,17] Prior to stent placement, women with STEMI are managed following similar principles that guide NSTEMI care.

Non–ST-segment elevation myocardial infarction

Treatment of NSTEMI is guided by risk stratification from assessment findings at presentation. Women with NSTEMI who present with cardiogenic shock, overt heart failure, persistent and recurrent rest angina despite medical interventions, hemodynamic instability, or with unstable ventricular arrhythmias are considered very high risk and are usually referred for coronary angiography without delay for further risk stratification. Risk assessment using reliable and established scoring tools such as the Thrombolysis in Myocardial Infarction (TIMI) or Global Registry of Acute Coronary Events (GRACE) allows for identification of women who are at risk for failure of medical treatment. Risk scores are calculated from items such as age, vital signs, history, risk factors, current and recent symptoms, laboratory values, and ECG findings. Women with lower risk benefit from intensive medical treatments, while women with higher risk status benefit from early (24–48 hours of admission) coronary angiography and potential revascularization.[7]

GENERAL TREATMENT PRINCIPLES

Initial care measures for pregnant women with AMI include oxygen administration and pain management. Oxygen is usually initiated at 2–3 L/min per nasal cannula and titrated to optimize oxygenation status. Pain management is essential to reduce oxygen utilization and optimize oxygen delivery to the myocardium. Morphine sulfate, given for pain relief and to reduce anxiety, is considered safe and effective but may cause neonatal respiratory depression if administered shortly before birth.

In addition to pain management and oxygen administration, an understanding of safe medication management to improve maternal outcomes while limiting fetal effects is necessary. Nitrates are frequently administered to relieve ischemic chest pain and decrease myocardial oxygen consumption, as they act to reduce coronary vasospasm and ventricular preload. Nitrates may limit the area of damaged tissue when administered during AMI, but are titrated to avoid maternal hypotension, placental hypoperfusion, and precipitation of nonreassuring fetal status.[4] In women with AMI, β-Adrenergic and β 1-selective blocking agents are utilized for their antihypertensive, anti-ischemic, and antiarrhythmic properties. They act to reduce chest pain, myocardial wall stress, and infarction size. Other antiarrhythmics such as digoxin, quinidine, and adenosine may be used if AMI is complicated by supraventricular and/or ventricular arrhythmias. However, amiodarone HCl is used only in the case of drug-refractory or potentially lethal arrhythmias because of the potential for fetal bradycardia, congenital goiter, and hypothyroidism.[16]

Although commonly used for treatment of AMI in the general population, angiotensin-converting enzyme inhibitors are contraindicated in pregnancy because of potential for major congenital malformations in the first trimester and neonatal renal dysfunction in the second and third trimesters.[7] Therefore, alternative medications for vasodilation and blood pressure management, such as nitrates, are recommended. Statins are also used for AMI in the general population for their anti-inflammatory and antioxidant properties, but there is insufficient data to support use in pregnancy.[7]

Anticoagulation is a hallmark of AMI treatment to prevent systemic or venous thromboembolism, with two platelet aggregation blockers, aspirin and clopidogrel, most commonly used in the general population and pregnant women.[7] The combination decreases the number of cardiovascular events by 20%.[7] Antiplatelet therapy is used alongside antithrombotic therapy. Unfractionated heparin (UFH) and low-molecular-weight heparin (LMWH) are the most commonly used agents. Unfractionated heparin and LMWH do not cross to the placenta, and the effects are easily reversed with

protamine sulfate.[1] In women at high risk for development of recurrent symptoms, LMWH appears to be more effective. However, in women with a high risk for development of recurrent symptoms and potential early intervention with PCI, UFH is equally effective with fewer bleeding complications.[7] A summary of common pharmacologic treatments for AMI is presented in Table 3.

OBSTETRIC CONSIDERATIONS

Most maternal deaths occur at the time of or within 2 weeks of AMI. In addition, some data suggest that birth within 2 weeks of AMI is associated with increased risk for recurrent AMI.[8] Therefore, if a woman is beyond the acute phase of an MI postponing birth for 2–3 weeks to allow for cardiac healing is reasonable. However, there are not data regarding optimal gestational age of birth after an AMI. If the woman is determined to be in preterm labor and tocolysis is indicated, magnesium sulfate is preferred and has a neuroprotective fetal effect if given prior to 32 weeks' gestation. Sympathomimetics are not administered because of associated increases in heart rate and myocardial oxygen consumption, and the potential risk of ischemia, even in otherwise healthy women.[13]

Timing of birth is carefully considered on the basis of maternal and fetal status, gestational age, and planned management of anticoagulant therapy. Induction of labor and vaginal birth hold many advantages over operative birth, including elimination of surgical risks, dramatic hemodynamic fluctuations, and decreased blood loss. Prior to birth, interprofessional preparation and development of a plan of care assist providers with communication, staffing, medication, and equipment needs for monitoring. Administration of oxytocin during a scheduled induction of labor is safe; however, recommendations include that prepared oxytocin mixtures be diluted to prevent coronary artery spasm and theoretic antidiuretic hormone activity.[2,4,6] Goals of management for the laboring woman anticipating a vaginal birth include reducing cardiac workload and oxygen demands and normalizing the family birth experience. Lateral recumbent positioning is preferred for labor and birth to optimize cardiac output, while enhancing placental perfusion.[4] Shortening the second stage of labor with the laboring-down technique or instrumental assisted birth are recommended to decrease oxygen consumption, fatigue, and the maternal tendency to Valsalva during pushing.[18] Limited, open glottis pushing may be safe if the woman's ejection fraction is at least 40%.[12]

Recommended surveillance during labor, birth, and the first 24 hours postpartum includes continuous assessment of blood pressure, pulse, oxygen saturation, ECG, and fetal heart rate.[12] Supplemental oxygen administration and adequate pain management during labor and birth are essential to minimize myocardial oxygen consumption and increases in cardiac output demand with sudden catecholamine release. Neuraxial anesthesia for pain relief is considered preferable as general anesthesia may be associated with significant increases in blood pressure during intubation.[13] Tachycardia and abnormal blood pressure parameters can have adverse effects on the woman's condition by decreasing cardiac output and should therefore be prevented or corrected if they occur.[4] The use of ergonovines in the postpartum period is avoided because of the risk for coronary artery spasm.[4] An example of interprofessional plan of care is outlined in Table 4.[11]

OBSTETRIC ADVANCED CARDIAC LIFE SUPPORT

In the event that cardiopulmonary resuscitation is required following AMI, recommended algorithms and medications are the same as for the nonpregnant patient population. Chest compressions and artificial respirations may be less effective in the pregnant woman owing to the displacement of abdominal contents and elevation of the diaphragm in the second and third trimesters.[2] Modifications to improve cardiac output and manage the woman's airway and emergent perimortem cesarean birth are summarized in Table 5.[19–23]

Initiation of a perimortem cesarean birth has been advocated within 4 minutes if maternal resuscitation efforts have failed to return a pulse.[19] By relieving aorta and venacaval compression, venous return and subsequent cardiac output may improve, increasing the potential for maternal and fetal survival. Emptying the uterus also improves the effectiveness of chest compressions and autotransfusion increases intravascular volume after birth, improving cardiac output.[22,23]

Neonatal survival is directly proportional to the timing between maternal cardiopulmonary arrest and birth.[20] Delivery of the fetus within 5 minutes significantly improves the neurologically intact survival rate of the viable fetus.[20] Cessation of maternal cerebral blood flow occurs at 6 minutes, and fetal survivability decreases with each minute after maternal cardiac arrest. However, there are case reports of maternal cardiac arrest outside of the acute hospital setting with perimortem cesarean section performed as late as 26–35 minutes after arrest yielding a neonatal survival rate of 25%.[20,21] To reflect the benefits of early cesarean birth, a mindset and terminology change from perimortem cesarean section to resuscitative hysterotomy has been recommended.[22]

Table 3. Common pharmacologic treatments for AMI

Description	Medication	Dose	Nursing implications
Platelet aggregation inhibitor	Aspirin	162-325 mg PO loading dose, then 81-100 mg PO daily	Started in combination with another platelet aggregate inhibitor
	Clopidogrel (Plavix)	300 mg PO bolus loading dose, then 75 mg PO daily	Use concurrently with aspirin after NSTEMI, STEMI, and/or PCI
	Prasugrel (Effient)	60 mg PO loading dose, then 10 mg PO daily	Use concurrently with aspirin after STEMI and/or PCI
	Ticagrelor (Brilinta)	180 mg PO loading dose, then 90 mg PO BID	Use concurrently with aspirin after STEMI and/or PCI
Vasodilator	Nitroglycerin (Nitrostat)	*Sublingual*: 0.4 mg every 5 min for a total of 3 doses prn	Give with symptoms of angina
		Intravenous: start 10 μg/min and titrate by increases of 5 μg/min every 5 min prn	Monitor BP trends—may cause hypotension
			Frequently causes headache
			May increase HR—monitor HR trends
			Decreases myocardial oxygen consumption
			Deceases CVP
Narcotic analgesic	Morphine sulfate	2-4 mg IV prn pain	Decreases pain, anxiety, and oxygen consumption
Antithrombotic	Enoxaparin (Lovenox)	1 mg/kg subcutaneous BID for a total of 8 d or earlier if successful percutaneous coronary intervention is achieved	Superior to unfractionated heparin
			No placental transfer
			Breastfeeding is safe
		STEMI—start with 30 mg IV bolus	Risk of epidural or spinal hematoma
	Unfractionated heparin	60 U/kg bolus followed by a continuous infusion at 12 U/kg/h to achieve an activated partial thromboplastin time (aPTT) between 50 and 70 seconds. Infuse for 48 h or earlier is successful, PCI is achieved.	May cause thrombocytopenia (5%-10% incidence)
			Does not cross placenta
			Breastfeeding is safe
			Monitor aPTT trends
			Risk of epidural or spinal hematoma
			Counteract effects with protamine sulfate
β-Blocker to control heart rate and/or blood pressure	Metoprolol (Lopressor)	2.5-5 mg IV every 5 min to a total of 15 mg and then PO 50 mg BID	Avoid in women with pulmonary disease
			May cause hypotension; monitor BP trends; hold if SBP < 120 mmHg
			May cause bradycardia; monitor HR trends; hold if HR < 60 bpm
			Rapidly enters fetal circulation; fetal serum levels equal to maternal levels; may cause persistent β-blockade in newborn
Calcium ion influx inhibitor to control heart rate and/or blood pressure; dilates coronary vessels	Diltiazem (Cardizem)	*Oral*: 30-90 mg every 6 h to maximum dose of 360 mg/d	May cause bradycardia—monitor HR trends; hold if HR < 60 bpm
		Intravenous: 0.25 mg/kg bolus followed by infusion at 5-15 mg/h	May cause dizziness and fatigue; implement fall precautions
			Excreted in breast milk and may approximate serum levels; pump and discard

Abbreviations: bid, twice a day; BP, blood pressure; CVP, central venous pressure; HR, heart rate; bpm, beats per minute; NSTEMI, non–ST-segment elevation myocardial infarction; PCI, percutaneous coronary intervention; PO, by mouth; prn, as needed; SBP, systolic blood pressure; STEMI, ST-segment elevation myocardial infarction.

Table 4. **Interprofessional plan of care**[a]

Goals
- Maternal hemodynamic stability
- Maximize myocardial oxygen delivery
- Minimize myocardial oxygen consumption
- Prevent or treat complications
- Pain and anxiety management
- Reperfusion of affected vessels and limitation of infarction size
- Maximize uteroplacental perfusion to optimize fetal status

	Action	Comments
Triage/Admission	Priority triage status based on: Initial screening Recognition of risk factors Recognition of signs and symptoms Maternal history	After initial screening upon arrival to hospital, "emergent" status acuity assigned for immediate assessment by a qualified medical provider per EMTALA regulations
	Request additional nurses to assist with care needs	As indicated
	Notify provider(s) to come to bedside for assessment	Evaluate need for rapid response team activation
	Place in left or right lateral position	If tolerated; if not tolerated, place in high Fowler's position
	Determine prior cardiac history	
	Determine current medications and dosage	
	Head-to-toe physical assessment	Report abnormal parameters Note cardiac sounds, rhythm, peripheral pulse quality, presence of edema, breath sounds, skin color, and temperature
	Frequent vital sign assessment	Report abnormal parameters
	Continuous ECG monitoring	
	Continuous SpO_2 monitoring	
	Administer oxygen 2L NC	Increase amount and administration technique as indicated by the woman's symptoms and response of SpO_2
	Continuous electronic fetal monitoring (EFM) if > 23 weeks' gestational age	Per hospital policy
	Review obstetric and prenatal history	
	Obtain IV access and maintain via infusion pump	Hourly assessment of intake
	Place Foley catheter	Hourly assessment of output
	Testing ■ ECG ■ Echocardiogram ■ Chest x-ray ■ Labs ○ Troponin I ○ CBC with differential ○ Electrolytes	All testing should be ordered as STAT Anticipate other testing needs for differential diagnosis such as computerized tomography (CT) scan
	Pain management with morphine sulfate	Keep woman comfortable to decrease oxygen consumption
	Anticipate other medication needs ■ Nitroglycerin ■ Aspirin ■ Clopidogrel ■ β-Blockers ■ Blood pressure management ■ Furosemide ■ Anti-arrhythmic agents	Coordinate needs with pharmacy

(*continues*)

Table 4. **Interprofessional plan of care[a] (*Continued*)**

	Action	Comments
	Anticipate need for invasive hemodynamic monitoring	Central line Arterial line
	Maintain calm, reassuring approach to care	
	Communicate plan of care with woman and family	
	Notify appropriate ancillary services/departments	Coronary catheterization laboratory Radiology Respiratory therapy Pharmacy
	Anticipate need for percutaneous coronary intervention	
Antepartum—intensive care unit until stable	ICU level of care after cardiac catheterization and intervention until hemodynamically stable	
	Collaborative medical and nursing management	
	Noninvasive hemodynamic monitoring of cardiac and respiratory status	At least every hour
	Continuous ECG monitoring	Documentation and strip verification per hospital policy
	Continuous EFM as indicated	Determined by gestational age and maternal hemodynamic stability
	Monitor fetal growth	β-Blockers may cause small-for-gestational age
	Evaluate for labor symptoms	
	Intake and output measurement	Continue infusion of all IV infusions via pump Continue Foley catheter; discontinue after maternal hemodynamic stability
	Monitor laboratory trends	Avoid maternal hyperglycemia due to stress response; monitor blood glucose values per ICU policy
	Implement fall precautions	Per hospital policy and procedure Some medications may cause dizziness
	Anesthesiology consult	Plan for pain management during birth Coordinate with obstetric provider regarding discontinuation of anticoagulants
	Interprofessional bedside rounds	Coordinate with all disciplines Collaborate to determine daily plan of care Communicate with patient and family and answer questions
	Venous thromboembolism prophylaxis	Sequential compression device Low-molecular-weight heparin
	Interprofessional care conference	Determine current patient care needs and services not routinely used in obstetrics Communicate will all team members regarding plan of care Determine plan for labor and birth Determine plan for anticoagulants as indicated
	Attempt to delay birth for 2 to 3 wks	
	Anticipate hospitalization until birth	
	Optimize hemoglobin levels for oxygen transport	
Intrapartum—Tertiary Care/Level IV Facility	Maintain 1:1 staffing	Anticipate need for additional nursing support during birth and immediate postpartum
	Collaborative medical management (maternal-fetal medicine and cardiology)	Notify provider if $< 96\%$
	Continuous SpO_2 monitoring	
	Continuous fetal and uterine monitoring with assessments her high risk guidelines	Every 15 min during active labor Every 5 minutes during 2nd stage of labor
	Continuous ECG monitoring	Documentation and strip verification per hospital policy

(*continues*)

Table 4. Interprofessional plan of care[a] (*Continued*)

	Action	Comments
	Oxygen 2 L NC	Increase amount and administration technique as determined by EFM interpretation
	Hourly intake and output measurement	Infusion pump for fluid administration
		Foley catheter placement during labor
	Arterial line placement for continuous BP assessment	
	Consider central line placement and central venous pressure (CVP) measurement	Monitor trends in CVP
	Optimize cardiac output	Provide adequate volume load during labor and prior to neuraxial anesthesia
	■ Avoid hypovolemia	Treat hypotension with intravenous boluses
	■ Avoid tachycardia (HR > 110)	Labor on left or right side as tolerated; sitting position for second stage of labor and immediate postpartum
	■ Avoid hypotension/hypertension	Investigate causes of tachycardia
		Anticipate need for β-blocker
	Physiologic dosing of oxytocin for induction or augmentation of labor	Start at 1–2 mu/min and increase every 40 min as needed on the basis of uterine contraction pattern
	Pain management	Neuraxial anesthesia preferred; provide adequate preload; dose and medication choice to prevent hypotension
	Bacterial endocarditis prophylaxis	Per AHA recommendations
	Second stage management	Position to optimize fetal descent and maternal hemodynamics
	■ Labor down	
	■ Avoid breath holding and Val Salva maneuver during pushing	
	■ Avoid lithotomy positioning	
	Cesarean birth for obstetrical indications	
	Anticipate blood loss at birth	Quantification of blood loss
	Anticipate autotransfusion after placental expulsion	Assess for rise in CVP and BP
	Anticipate medication needs and coordinate with pharmacy	Medications not routinely used in Labor and Delivery
	Consultations as indicated	
Postpartum	Preload reduction and furosemide administration as indicated immediate postpartum	Autotransfusion at birth may cause cardiogenic pulmonary edema
	Avoid methergine and prostaglandins as a uterotonic agent in third stage of labor	May cause coronary vasoconstriction
	Continue SpO_2, ECG, and hemodynamic monitoring for 24 h or as indicated by maternal status	
	Promote family-infant bonding	
	VTE prophylaxis	
	Treat fluid and electrolyte imbalances as indicated	
	Promote relaxation and rest	
	Minimize anxiety and fear with education and communication regarding plan of care	
	Stool softeners/laxatives—avoid constipation and Val Salva maneuver	
	Provide adequate pain management	
	Breastfeeding encouraged; determine if medication requires pumping and discarding milk	
	Follow-up with cardiology and maternal-fetal medicine services	

Abbreviations: AHA, American Heart Association; BP, blood pressure; CBC, complete blood cell; ECG, electrocardiogram; HR, heart rate; ICU, intensive care unit; IV, intravenous; VTE, venous thromboembolism.
[a]Adapted from Vander Hoek et al[10] and Baird and Kennedy.[11]

Table 5. **Modifications of Cardiopulmonary Resuscitation in Pregnancy**[a]

Action	Rationale
Place the woman in a supine position	Compressions with the woman in a lateral position are less effective
Manually displace uterus if ≥ 20 wks' gestational age	Prevention of compression of aorta by gravid uterus
Early intubation by an experienced provider	Pregnancy changes in airway mucosa o Edema o Friability o Hypersecretion o Hyperemia During apnea, pregnant women desaturate at a more rapid rate Physiologic changes in pregnancy cause the woman to become acidotic at a more rapid rate
Apply cricoid pressure during intubation	Pressure applied to the cricoid cartilage pushing the trachea posteriorly, pressing the esophagus against the cervical vertebrae to reduce risk of aspiration
Hand placement for chest compressions should be slightly higher on the sternum	Adjusted because of elevated diaphragm and abdominal contents by the gravid uterus Deeper—chest wall compliance is decreased
Insert intravenous catheter above the diaphragm (usually a central line)	Rapid volume expansion
Remove external and internal fetal and uterine monitors	Allows for access to maternal abdomen for perimortem cesarean birth
Perform perimortem cesarean birth beginning at 4 min after cardiopulmonary arrest	Improve maternal resuscitation efforts Improve neonatal outcomes Potentially avoid permanent CNS injury in the newborn that may occur after 5 min after cessation of flow

Abbreviation: CNS, central nervous system.
[a]Adapted from Vander Hoek et al,[10] Baird and Kennedy,[11] and Lipman et al.[23]

The obstetric nurse is a key member of the resuscitation team and potentially fulfills several roles in the event of a maternal cardiac arrest. These may include the historian to provide pivotal patient information regarding pregnancy and maternal assessment prior to arrest, administration of emergency medications, intravenous insertion, documentation of code events, and providing chest compressions. Further recommendations include practice with simulated code scenarios for all team members on a routine basis.

PREGNANCY AFTER AMI

Pregnancy after AMI is associated with increased incidence of short- and long-term illness, rather than high maternal mortality.[4] The amount of residual cardiac function, anatomy, and cause of initial infarction determine maternal morbidity. Testing to determine evidence of coronary vascular disease and myocardial function is advised before future pregnancy. Women are typically advised to wait at least 1 year following an AMI before becoming pregnant again. Because episodic myocardial ischemia may be occurring without angina, treadmill exercise tolerance testing and myocardial perfusion imaging at rest and after peak exercise is recommended. If treadmill testing reveals abnormal response, angina, or a drop in blood pressure at a low level of exercise, the woman is at high risk for a serious and/or fatal myocardial ischemic event and is advised against becoming pregnant. Coronary arteriography may be also performed to determine degree of coronary stenoses and potential need for revascularization. Myocardial infarction can cause left ventricular damage, resulting in decreased cardiac contractility and cardiac output. Therefore, left ventricular function studies such as echocardiography or transesophageal echocardiography are performed prior to a future pregnancy. If severe damage, decreased left ventricular function, and heart failure are present, women are strongly advised against further childbearing.[18]

CONCLUSION

Acute myocardial infarction during pregnancy remains rare, but trends indicate that perinatal nurses may be faced with the challenges of caring for women with AMI in the future. To minimize morbidity and mortality associated with AMI, it is critical to remain vigilant

of maternal symptoms such that diagnostic testing and treatment may be rapidly initiated to preserve myocardial functioning. The perinatal nurse plays a key role in planning and caring for the woman experiencing AMI during pregnancy. Team and interprofessional communication is essential for providing safe, accurate, patient-centered care to achieve optimal maternal and fetal outcomes.

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