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Myocardial Infarction in Pregnancy: What Should We Do?

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Abstract

Acute myocard infarct during pregnancy is a rare event with high mortality and women with cardiac comorbid such as hypercholesterolemia, hypertension, and history of myocardial infarction are one of risk that should be warning. However, the main etiology myocard infarct in pregnancy is spontaneous coronary artery dissection, especially in the third semester. Women with unusual chest pain, ST-elevation in electrocardiogram, and sudden cardiac arrest should treat as soon as possible. Patient with unstable condition revascularization is recommended. However, there are several things that should be monitor during revascularization such as consideration type of stent, how much contrast that will be use, and planning after stent implantation. Thrombolysis should be the last option because of bleeding risk. Therapy during and after pregnancy should be monitor continuously because of their side effect to mother and baby, and delivery in women with acute myocard infarct should be one of main concerns. With this review, we hope that we can raise our awareness in pregnant women with their comorbid and their future pregnancy.

Introduction

According to the Centers for Disease Control and Prevention, cardiovascular illnesses are the primary cause of pregnancy-related mortality in the United States, accounting for over 15% of these deaths in recent years. Because of the hypercoagulability and hypervolemia that accompany pregnancy, there is a well-known three-fold increased risk of acute myocardial infarction (AMI) [1]. This increases blood pressure, heart rate, cardiac output, and myocardial oxygen consumption [2]. Myocardial infarction in pregnancy is a rare event; the incidence ranges from 0.06 to 10/100,000 pregnant women globally, and as the leading cause of pregnancy-related death with high mortality ranged, two risk factors contributed to pregnancy-related myocardial infarction, first – modified factor, e.g., smoking is the most prevalent risk, followed by hyperlipidemia and second – non-modifiable factor, e.g., age >35 years is one of the important risk factors [1], [2]. Spontaneous coronary artery dissection (SCAD) is the primary etiology in pregnancy myocardial infarction with presentation of 15–40% and other etiology such as pre-eclampsia and multiple gestations. To this day, the understanding of myocardial infarction in pregnancy is limited due to its scarcity [2]. The review aims to provide an overview of the present state of knowledge, consensus statements, and key details.

Epidemiology

The incidence of myocardial infarction in pregnancy is increasing and the case fatality rate is estimated at 5%, higher than non-pregnant women at reproductive age [2]. The risk factor that contributes to pregnancy-related myocardial infarction is non-modified factor and modified factors, the non-modified risk factor occurs between 31 and 40 years, the median age of 35 years, and mostly occurs in the antepartum and postpartum period, from the national inpatient sample found 859 of myocardial infarct (MI) in pregnancy. In MI occurred antepartum, there's 73% of cases with commonly present at a range of 23–36 weeks, whereas 27% of MI occurred in postpartum.

Other important non-modified risk factors are multiple gestations, gestational diabetes, and hypertensive disorders of pregnancy (eclampsia/pre-eclampsia) [1], [3], [4]. 18.3% of gestating women with myocardial infarction have experienced eclampsia/

pre-eclampsia. The most common modified risk factor is smoking with 25% cases followed by hyperlipidemia (20%). Women with a previous history of cardiovascular disease and pregnancy complications should be consulted regards their higher risk of myocardial infarction during pregnancy. MI is caused by obstructive or non-obstructive lesions, in obstructive [1]. Patients known to have coronary artery disease (CAD) in the past time had a greater chance of a heart attack, approximately 27–43% of myocardial infarctions are caused by atherosclerotic CAD. According to Cauldwell *et al*., women with underlying atherosclerotic disease during pregnancy are significantly older and have a higher body mass index, other significant CAD risk factors whereas patients with normal coroner have an MI with a non-obstructive coronary artery [5]. Spontaneous coronary dissection (SCAD) is the most common non-obstructive myocardial infarction in pregnancy with an estimated 14–43% [5].

Pathogenesis

Some conditions can cause acute coronary syndrome (ACS) in pregnancy. It should be differentiated between obstructive and non-obstructive [4]. SCAD plays a major role, the pathophysiology of SCAD occurs when there is a separation between the tunica media and adventitia, and there are two hypotheses behind the pathophysiology [6]. The first hypothesis is an initial intimal tear within the coronary arterial wall that allows blood to enter intimal space and creates the false lumen leading to dissection and the second hypothesis is the accumulation of hematoma that comes from the vasa vasorum narrows the coronary artery lumen without the presence of a tear in the tunica intima. This can be attributed to hormonal changes associated with pregnancy [6], [7].

Excess of progesterone may lead to a weakened tunica media through a decrease in acid mucopolysaccharide ground substance and lead to coronary arterial wall dissection [3], whereas estrogen increases endothelial-mediated vasodilatation through a nitric oxide synthase-dependent mechanism that leads to reduced mitochondrial reactive oxygen species [1], [8]. These structural changes may result in vasa vasorum rupture or intimal tear [8]. Many cases occur in the postpartum period, mostly within the 1st week of delivery in the peripartum period of 3–90-day postpartum [3]. It might be related to cardiac stress due to rapid post-delivery uterine contraction, especially in patients with predisposing factors including fibromuscular dysplasia, while antepartum mainly presents in the third trimester, CAD affects in multiparous women with the average gravidity being 2.7 In pregnancy [1], coronary thrombosis may happen because of a hypercoagulability state in pregnancy due to alterations in the coagulation and fibrinolytic systems [9]. Patients with predisposing conditions should be a concern, including Kawasaki disease, systemic inflammatory conditions, autoimmune disorders, atrial fibrillation (AF), cardiomyopathy, and valvular heart disease [2].

In pregnancy low-density lipoprotein concentration increases by 40–50%, and triglyceride concentration can rise to fourfold start in the 14th week of gestation, however. Both cannot exceed 250 mg/dL in a normal pregnancy [10]. According to Cauldwell *et al.* 43, women had either a history of CAD or previous ACS. About 80% of women with atherosclerosis disease usually had at least more than one risk factor for CAD, unstable atherosclerotic CAD leads to rupture of the thin fibrous cap of a lipoprotein-rich plaque with exposure of the necrotic core and coronary thrombosis formation [2], [11]. Maternal outcomes are generally very good, with higher rates of pre-term delivery and lower birth weights. However, recent guidance from the European Society of Cardiology suggests that women with recent CAD and presenting with reduced ventricular function or with residual ischemic pregnancy should be discouraged [11].

Coronary vasospasm in pregnancy is estimated at 2–5% in pregnancy-associated MI [5]. Pre-eclampsia is one of the risk factors, resulting in a high-resistance uteroplacental circulation and will cause placental ischemia which impairs the function of maternal vascular endothelial growth factor resulting in systemic endothelial dysfunction. Moreover, in pregnancy, a low resistance uteroplacental circulation is created by the release of renin and angiotensin [3], [9].

Diagnosis

The principal diagnosis of myocardial infarction in pregnancy is the same with the non-pregnant patient. ACS in a woman usually presents with atypical symptoms, especially in pregnancy. All complaints of chest pain, but also in the neck, stomach, arms, and dyspnea on effort should be a warning and need more assessment [9]. Electrocardiogram (ECG) is one of the diagnostics supports that are available in most health facilities. In normal pregnancies, abnormal ECG including left axis deviation, T-wave inversions in leads V1-V2, and small Q waves together with T inverted T waves in lead III will be seen. However, ST-segment elevation is never normal [1]. The anterior wall is involved in 69–78% with the highest percentage of most common causes being from atherosclerotic or SCAD. The rest involved the inferior wall in about 27% of cases and the least is a lateral wall with 4% [3].

Biomarkers help diagnose myocardial injury, cardiac troponin is a specific biomarker, since creatine

kinase MB and myoglobin increase from uterine contraction during labor [1]. According to Cano-Castellote *et al*., cTnI significantly increases in an individual with gestational hypertension compared to normotensive individuals (median: 89 vs. 30 ng/L, $p < 0.001$) and increases in an individual with pre-eclampsia compared to gestational hypertension (median: 155 vs. 89 ng/L, $p = 0.03$). In SCAD, the values do not initially increase. The beginning troponin level was at 0.45 ng/mL but after some time raised to 50 ng/mL [8].

Coronary computed tomography angiography (CCTA) can be used if necessary. If CCTA is needed during the pregnancy, fetal and maternal radiation should be calculated. Iodinated contrast does cross the placenta. The estimated fetal radiation dose from CCTA is low, ranging from 1 to 3 mGy [1], [3].

Percutaneous Coronary Intervention (PCI)

PCI should be considered for patients with ST elevation and/or patients with high risk. An understanding of radiation exposure in a pregnant patient is needed to reduce radiation risk. In normal patients, radiation exposure is estimated to be <20 mGy, while fetal estimated share the exposure of about 0.074 mGy [12]. Risk of teratogenicity increase at radiation doses above 150 mGy, between 2 and 7 weeks gestasional age which organogenesis takes place in the period [1].

If PCI is undertaken consider the choice of the stent, whether to use bare metal stent (BMS) or drug-eluting stent (DES), especially for minimizing the duration of double anti-platelet (DAPT) to avoid bleeding in pregnancy [5]. BMS is recommended in women who have reached the third trimester because the short duration of DAPT only requires 4 weeks of treatment [3] whereas DES requires DAPT treatment more than >3 months after DES implantation. DES is preferred in the first 2 trimesters [2]. Nevertheless, DES is more favorable in non-pregnant adults because it reduces risks of MI and repeat revascularization compared with BMS [12].

There is different treatment management in the etiology of AMI. Those with atherosclerotic disease are safer than patients with SCAD as etiology because of the higher risk of complications with only about one-third of PCI attempts being successful [12]. Catheter-induced dissection may happen due to the fragility of the arterial wall leading to further ischemic injury and acute heart failure which requires mechanical circulatory support devices and CABG [12], [13], so. Patients with stable hemodynamics should be avoided. Instead, adequate treatment is much needed. However, patients with a high risk of MI, ongoing chest pain, and ST-segment elevation urgent invasive strategies should be performed [2], [13].

Thrombolysis

The use of thrombolysis for revascularization strategy is not recommended and should be the last option, with a high risk of bleeding including maternal and fetal hemorrhage [12]. In patients with SCAD as the etiology, the risk of dissection flap propagation and expansion of the intramural hematoma should be a warning [5]. Major bleeding occurred in 18–58% of cases during pregnancy and in the postpartum period. A cohort study of 28 pregnant patients treated with fibrinolytic therapy for various indications (i.e., pregnancy MI, stroke, and pulmonary embolism) reported 2 deaths (7%) and three adverse events, including brain and abdominal hemorrhage (11%) [12].

Therapy

Treatment for myocardial infarction in pregnancy should be monitored because appropriate therapy will increase maternal outcomes also increase fetal survival. Full-dose aspirin can be used until 32 weeks of gestation, and 81 mg can be used anytime during gestation [2]. Clopidogrel is the preferred P2Y12 inhibitor and yet P2Y12 must be discontinued 7 days before neuraxial anesthesia [5], [14]. Both clopidogrel and prasugrel can be used in pregnancy, however. The use of clopidogrel needs to be monitored during pregnancy due to the risk of extension of dissection if the SCAD is the main etiology, bleeding risk, and due to insignificant evidence of benefit [14]. Ticagrelor is unsafe in lactation and associated with risk in pregnant animals such as higher bleeding risk, dual antiplatelet (DAPT) may be given in patients with intracoronary stenting planned while single-agent aspirin is given in patients with SCAD [3], [6].

The use of heparin in pregnancy is considered safe because it is early-onset, short half-life, and dose that is easy to adjust through monitoring of the activated clotting time.[16] Heparin does not cross the placenta and does not have teratogenic effects; however, heparin can induce thrombocytopenia in 3% of patients and it should be stopped 4–6 h before delivery [3], [12]. Low-molecular-weight heparin (LMWH) has advantages over heparin due to greater bioavailability, decreased affinity for heparin-binding proteins, and discontinuation of LMWH in 24 h before delivery is desirable. If indicated, treatment can be continued after delivery. Both heparin and LMWH are not excreted into breast milk and therefore safe for lactation [12], [15].

For the treatment of ACS, PCI combined with revascularization continues to be the gold standard. Obstetricians and interventional cardiologists who are worried about radiation danger to the growing fetus may

find it difficult to propose PCI [16]. According to evidence that has been published, doctors who frequently treat pregnant patients might not be aware of the full extent of radiation dangers associated with pregnancy. PCI decisions may be influenced by the pregnant patient's and the health-care provider's concerns about any radiation-related procedure. PCI can be performed during pregnancy and is a life-saving treatment that should be done when needed [17].

Standard revascularization guidelines should be followed while performing PCI if there is an atherosclerotic lesion [18]. DES should be used when coronary stent implantation is necessary to guarantee low restenosis rates and long-term coronary patency [18] and drug-eluting balloons [19] should be considered as optional therapy. This is especially crucial for patients who are pregnant and younger than the general revascularization patient population. With second and third-generation platforms, it is possible to administer dual antiplatelet medication for a brief period of time following the implantation of a DES, and the rates of ischemic events are similar to those of bare-metal stents. After stent insertion, delivery timing is critical since dual antiplatelet medication raises bleeding risks, especially when combined with neuraxial anesthesia and the possibility of epidural hematoma [14].

Patients should be treated in an intensive care unit that can offer rigorous maternal monitoring and obstetric care, and management should be decided by a multidisciplinary team made up of cardiologists, obstetricians, anesthesiologists, and neonatologists. The optimal time to carry out any PCI treatment is during the second trimester, following the 4th month, according to ESC guidelines [14]. This is mostly explained by the fact that fetal organogenesis has finished, the fetal thyroid is dormant, and the uterus is tiny at this point in the pregnancy, which permits a larger space between the fetus and the chest than in the later months.

The gestational age, alternative diagnostic methods, estimated fetal dose, and most importantly, the knowledge that the fetus's life depends on the mother's life should all be taken into account [18], [19]. In this instance, it was determined that the advantages of PCI outweighed the slight risk of radiation exposure to the mother and fetus, and care was taken to reduce radiation doses to both of them. Furthermore, after a patient-specific risk-benefit analysis is completed, drugs used in the cardiac catheterization laboratory (such as nitroglycerin, aspirin, clopidogrel, anticoagulants, calcium channel, or beta-blockers) may be administered in place of the previously used pregnancy risk categories [20].

Beta-blockers may reduce both myocardial oxygen consumption and shear stress in SCAD and it has been used in pregnancy for the management of hypertension, mitral stenosis, Marfan syndrome, and myocardial ischemia [1], [15]. Propranolol is considered safe in pregnancy, metoprolol is well tolerated and used for SVT and ventricular arrhythmias in pregnancy, and labetalol is used as first line for both acute and chronic hypertension in pregnancy, but dose adjustment is needed due to increasing clearance shorter half-life. However, neonates exposed to beta-blockers had significantly more bradycardia and hypoglycemia and were often associated with fetal growth restriction [14], [15], [21].

Nifedipine is part of calcium-channel blockers which commonly used in the treatment of hypertension, pre-eclampsia, and tocolysis, and verapamil is the second line for rate control in AF and treatment of idiopathic sustained VT in pregnant women [12], [15]. Those are excreted in human milk, therefore. It is not recommended for breastfeeding mothers to take these drugs [12]. Angiotensin-converting enzyme inhibitors and angiotensin receptor blockers have contraindicated fetotoxic effects [1], [14]. Nitrates may help to ease chest discomfort or to resolve coronary vasospasm but be aware of maternal hypotension and placental hypoperfusion, and nitroglycerin is recommended when pre-eclampsia is associated with pulmonary edema [5], [22].

Statin should be stopped in 1–2 months before pregnancy or immediately due to its teratogenic effects. However, in a recent systemic review, there was no clear relationship between the use of statin in pregnancy and congenital anomalies, due to its limited data, it would be very wise to discontinue before conception [10], [19].

Delivery

When it is possible to delay delivery for 2–3 weeks after AMI, it is recommended to avoid hemodynamic changes and increased cardiac stress, delivery within 2 weeks of AMI is related to increased risk of reinfarction and higher mortality [12]. Induction of labor is recommended at 40 weeks and induction with oxytocin is considered safe; it can be used for failure to progress in labor. Slow and low-dose administration of oxytocin are recommended to avoid tachycardia, hypotension, and myocardial ischemia [9]. Prostaglandin is also safe in these patients, whereas. Ergometrine should be avoided due to its powerful vasopressor activity that can cause coronary artery spasms [11].

Vaginal delivery considers to be the safest option in women with AMI due to less blood loss, infection risk, venous thrombosis, and embolism [23]. In cesarean section, the ROPAC investigator previously showed that planned cesarean section fails to give any benefit to mothers with heart disease [11]. However, cesarean delivery should be considered in patients with obstetric indications such as breech presentation, failure to progress in labor, elective cesarean delivery,

fetal heart rate abnormalities, poor cardiac function, and inability to tolerate the hemodynamic shift. Cesarean delivery should be achieved in the shortest time possible [22].

Epidural anesthesia during delivery for pain control has been reported to decrease cardiovascular events, including arrhythmias, however. Epidural analgesia should be titrated slowly to avoid hypotension and a major concern is the risk of spinal epidural hematoma formation in patients receiving DAPT. Intravenous opioid analgesics (fentanyl, meperidine, morphine) as the second option [2], [12], [24].

Outcomes

More than half of pregnancies in MI are followed by permanent left ventricular ejection fraction which leads to life-threatening complications and death. When women with history myocard infarct during pregnancy, severe LV dysfunction (LVEF <30%) is at high risk for developing complications [5]. With approximately 10% recurrences, the highest recurrences are in women with previous coronary dissection [12]. However, a case series from Roth and Elkayam reported a 19% incidence of heart failure and/or cardiogenic shock and mortality rates as high as 20%. Along the time, in their case series from 2008 mortality rates were 11% and dropped to 7% in 2014 and 4% in 2016 case series of AMI secondary to SCAD. Even though, there's an improvement in the management strategies over the years [25]. Women are still at higher risk of cardiac arrest, heart failure, and ventricular tachycardia which are the most common cardiac complications of myocardial infarction in pregnancy and yet the European Society of Cardiology recommends to delay conception for 12 months following MI [2].

Conclusion

Myocardial infarction in pregnancy is a rare event and yet. Cardiovascular disease in pregnancy is the leading of maternal and fetal death. Women who have comorbidities and risk factors should be assessed more to predict the future of pregnancy. If women with comorbidities and risk factors get pregnant, early diagnosis is important to predict the outcomes, a multidisciplinary approach including obstetricians, cardiologists, and anesthesiologists is essential to further treatment and plan, and a sufficient treatment will determine future prognostic.

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