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Volume 1 • Issue 3 • 2017

Mini Review

Therapeutic Advances in Cardiology

ISSN: 2575-5161

Pregnancy-Associated Myocardial Infarction- Unusual but Potentially Lethal

Monika Thakur[1], Akhil Gautam[2], Kunal Mahajan[3]* and Suman Lata[4]

[1]Senior Resident, Department of Obstetrics and Gynaecology, Indira Gandhi Medical College (IGMC), Shimla, Himachal Pradesh, India

[2]Consultant, Department of Cardiology, Fortis Hospital, Kangra, Himachal Pradesh, India

[3]Consultant, Department of Cardiology, Holy Heart Advanced Cardiac Care and Research Centre, Rohtak, Haryana, India

[4]Senior Resident, Obstetrics and Gynaecology, Apollo Hospital, Bangalore, Karnataka

***Corresponding Author:** Kunal Mahajan, Consultant, Department of Cardiology, Holy Heart Advanced Cardiac Care and Research Centre, Rohtak, Haryana, India.

Received: November 09, 2017; **Published:** November 30, 2017

Abstract

The incidence of ischemic heart disease in women of child bearing age is rising due to changing demographics associated with advancing maternal age and increased incidence of risk factors, including diabetes mellitus, hypertension, and preeclampsia. The diagnosis of myocardial infarction in the pregnant population can be challenging and inappropriate management may lead to the risk to the mother as well as to the fetus. Epidemiology, diagnosis, medical and surgical treatment, and prognosis of ischemic heart disease in pregnancy are the subject of the present review.

Keywords: *Pregnancy; Myocardial Infarction; Spontaneous coronary artery dissection; Management*

Volume 1 Issue 3 November 2017

Introduction

Acute myocardial infarction (AMI) is a rare, but possibly lethal event during preg¬nancy, delivery, or puerperium. The frequency of AMI is increasing because of continuing trend of childbirth in older women and the use of reproductive technologies that allow older and postmenopausal women to conceive [1-3]. Pregnancy increases the risk of AMI ≈3-fold when compared with nonpregnant women of similar age [1]. Approximately 20% of cases occur during labour, with the remainder nearly equally divided between the antepartum and the postpartum periods. Maternal mortality ranges from 5% to 8% and appears to have decreased in recent years [1]. Mortality is approximately 20% when the MI occurs in the peripartum period. This is roughly twice the rate during the antepartum period. Prematurity is reported to be 43% in patients with an antenatal MI. Fetal outcome is ultimately related to maternal status and outcome.

Risk Factors

Peripartum myocardial ischemia is a dif¬ferent entity because atherosclerosis which is the most common cause of AMI in the non-pregnant population is responsible for only a third of MI in pregnancy. The majority of pregnant patients develop AMI by other mechanisms [4]. Factors such as increasing maternal age (> 35 years), increasing rates of maternal obesity, smoking, diabetes mellitus, hyperlipidemia and family history are possibly contributing to the rise in frequency of pregnancy-associat¬ed AMI [5]. Obstetric risk factors include preeclampsia, multifetal gestation, thrombophilia, transfusion, postpartum infection, use of oral contraceptives and multiparity [2,3]. Acute myocardial infarction has been related to pregnancy-induced hypertension and preeclampsia. Possible explanation includes enhanced vascular reactivity to angiotensin II and norepinephrine and endothelial dysfunction. All these together promote coronary constriction in patients of pregnancy induced hypertension. [2]

Etiopathogenesis

Acute MI in pregnant females has diverse etiology, where as atherosclerosis dominates in the antepartum phase [1], coronary dissection is the leading cause in peripartum phase. Hemodynamic stress during pregnancy, anemia, pain, anxiety, blood loss during delivery all lead to strain to the coronary vasculature and increase the chances of coronary dissection which most commonly involves LAD. Proposed mechanisms for coronary dissection related to pregnancy have been hormonally-mediated arterial structural changes, with a loss of normal corrugation of elastic fibres and a decrease in acid mucopolysaccharide ground substance, which may lead to cystic medial necrosis and lack of structural support of the vasa-vasorum in the media-adventitia border. This lack of support may lead to rupture and intramural hematoma. [1]

Coronary spasm due to endothelial dysfunction and increased vascular reactivity to angiotensin-II and nor-adrenaline is another possible etiology [1]. Coronary thrombosis without atherosclerosis due to hypercoagulable state is also observed in a small subset of patients [1]. Other causes include aortic valve stenosis, sickle cell anemia, and pheochromocytoma.

A recent report of 132 women studying coronary anatomy showed coronary dissection in 43%, atherosclerosis in 27%, clot without evidence of atherosclerosis in 17%, normal anatomy in 9%, spasm in 2%, and Takotsubo cardiomyopathy in 2% as possible causes of MI during pregnancy [6].

Clinical Features

Criteria for the diagnosis of MI do not change during pregnancy. The diagnosis remains a challenge, in part because the index of suspicion is often low. Physiologic changes of pregnancy may mimic the symptoms of MI and delay the diagnosis. The diagnosis is mainly on the basis of symptoms, electrocardiography (ECG) changes, and biomarkers. Normal changes during pregnancy as well as fetal safety influence some of the diagnostic criteria and approaches.During normal pregnancy, most women experience some increase in exercise intolerance, dyspnea, fatigue, weight gain, and pedal edema. Chest pain due to reflux is also common. ECG changes may not be as reliable and may mimic ischemia even in normal patients. Nevertheless, it is important to remember that even during pregnancy, ECG is diagnostic of acute MI. ST Elevation on ECG clinches the diagnosis and treatment should be started immediately once changes are seen without waiting for other test reports.

Normally ECG may show sinus tachycardia, a leftward shift, ST-segment depression, flattened or inverted T-waves, and a Q wave in lead III in pregnancy. Measurement of serum levels of the cardiac-specific contractile protein, troponin-I, is accurate for the diagnosis with the exception of women with hypertension or preeclampsia [7]. CK-MB is not reliable for the diagnosis because levels are known to be elevated during uterine contractions. An echocardiogram showing abnormal wall motion in the ischemic region confirms the diagnosis. It also detects LV dysfunction and other mechanical complication of MI.

Coronary angiography provides both diagnostic and option of therapeutic PCI. The use of radiation during pregnancy should be minimized if possible. Cardiac catheterization and interventional procedures may result in the fetal exposure of < 1 rad. Termination of pregnancy is usually recommended only when the radiation exposure exceeds 10 rads [8].

When coronary angiography is performed, the procedure should be done cautiously with the use of a nonselective injection to assess for left main dissection, avoiding deep catheter intubation, and using the minimum number of low-pressure contrast injections to minimize the risk of dissection. The bleeding and radiation risks can be reduced by use of a radical approach, appropriate abdominal shielding, and decreasing fluoroscopy time [9].

Management

TIME IS MUSCLE in acute MI, and this holds true whether the patient is pregnant or not. Urgent reperfusion is the most important therapeutic intervention. In clinical trials this group of patients has often been excluded, however, recommendations are similar to non-pregnant patients and a collaborative effort between the cardiologist and the obstetrician is a must.

Percutaneous Coronary Intervention (PCI): Done in a timely fashion PCI has clear-cut benefit over thrombolytic therapy and is the preferred modality of reperfusion. Percutaneous angioplasty and stent placement during pregnancy have been successfully reported with favourable outcomes [10,11]. Bare metal stents are usually preferred over drug-eluting stents. The latter is generally avoided due to the necessary long-term need for dual antiplatelet therapy (DAPT) with aspirin and Clopidogrel. DAPT can lead to excessive bleeding during delivery and can complicate epidural anesthesia which is otherwise ideal for pain control during labour [12]. Though radiation exposure can lead to undeniable detrimental effect on the fetus, therapeutic benefits to mother should not be neglected for fetal wellbeing.

Thrombolytic agents: Generally the thrombolytic therapy has been considered as a relative contraindication in pregnancy in almost all the guidelines.Thrombolytic therapy has been used in pregnant women in early pregnancy [13]. Although effective, there may be a small but real incidence of associated maternal bleeding. Also, higher incidence of coronary artery dissection as the cause of pregnancy-associated MI urges caution with the use of thrombolytic therapy because the risk of bleeding outweighs anticipated benefits in such a situation and may even worsen the condition.

Coronary Artery Bypass Grafting (CABG): Emergency CABG should only be considered in cases of failed PCI, development of complications like dissection during PCI or in patients with coronary dissection where PCI is not amenable. Maternal mortality with CABG is 1.5%, as with the non-pregnant population [14].

Pharmacological Treatment: Unfractionated heparin and low molecular weight heparin (LMWH) are the anticoagulants of choice as they don't cross the placenta and have no teratogenic effects [15]. LMWHs are associated with a lower risk of bleeding as well as reduced incidence of heparin-induced thrombocytopenia. However, they should be withheld 24 hours before the delivery. Anti-platelet therapy is a must, as not only it maintains the benefits of PCI, but also prevents future coronary events. Aspirin use is largely considered safe during pregnancy. A large randomised trial of 9000 patients showed that low-dose aspirin (60 to 150 mg/d) administered in the second or third trimester is safe for both the mother and the child [16]. Aspirin is also secreted in small concentration in breast milk. Data remain scarce as far as the safety of thienopyridines and GP IIb/IIIa inhibitors, during pregnancy and breastfeeding are concerned. Patients should be monitored for evidence of arrhythmias or congestive heart failure. Most of the other drugs used in the medical management of acute MI are category C, except where otherwise noted. Acute management of acute MI includes administration of nitro-glycerine and morphine (category B), with close blood pressure monitoring [17]. Lidocaine (category B) is used to suppress malignant arrhythmias if present. Calcium channel blockers or β-blockers are given if indicated.

Labour and delivery: Elective delivery within 2 weeks of infarction should be avoided because it is associated with an increased risk of maternal death. Caesarean delivery is reserved for the usual obstetrical indications as well as those who have had a MI in close proximity

to labour or in unstable patients. External cardiac monitoring is necessary. Supplemental oxygen should be given, and epidural analgesia is ideal for pain control during labour. The second stage of labour is generally cut short.

Postnatal Care: Volume status is monitored in the postpartum period, and the patient should avoid exertion. A reliable plan for contraception should be made. Combined oral contraceptives are contraindicated. Continued use of progesterone-only methods should also be avoided because they may have adverse effects on the lipid profile. If permanent sterilization is not desired, then copper-containing IUD is a reasonable alternative.

Conclusion

In conclusion, clinicians should be aware of the classic risk factors for coronary artery dis¬ease and typical symptoms of myocardial ischemia, as pregnancy itself represents a risk factor. Particularly, older childbear-ing women should be followed up closely. Diagnosis is often delayed due to the rarity of the event, lack of awareness and failure to recognize the symptoms during pregnancy. MI in pregnancy is different from MI in nonpregnant patients and requires special consideration. Early diagnosis and appropriate treatment are necessary to reduce the morbidity and mortality in such patients.

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