Acute Coronary Syndrome During Pregnancy: A Case Report and Literature Review

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SUMMARY

A 32-year-old multiparous woman who presented with chest pain at seven weeks gestation was admitted to our hospital 35 minutes after the onset of symptoms. Sudden cardiac arrest developed while the patient was waiting in the triage room. Cardiopulmonary resuscitation was performed, and the patient was immediately intubated. Electrocardiography revealed an inferior myocardial infarction. The patient underwent coronary angiography, which revealed slow coronary flow of the circumflex and left anterior descending coronary arteries. For treatment, the combination of aspirin with clopidogrel and unfractionated heparin was initiated. She had previously had three healthy children and hadn’t had any problems during her previous pregnancies. She had a history of family and smoking, but no history of other coronary risk factors such as diabetes mellitus, hypertension, or dyslipidemia. She was discharged home on day five after admission with clopidogrel, aspirin and a beta-blocker with close outpatient follow-up. Elective abortion was planned for two weeks after the myocardial infarction.

Key words: Coronary thrombosis; myocardial infarction; pregnancy.

ÖZET

Otuz iki yaşında yedi haftalık multipar gebe bir kadın, acil servisime 35 dakika önce başlayan göğüs ağrışi şikayeti ile başvurdu. Hasta bekleme odasında beklerken ani kardiyak arrest gelişti. Kardiopulmoner resüsitasyon yapılırken hasta hemen entübe edildi. Elektrokardiyografide inferior miyokart enfaktüsü saptandı. Hasta koroner anjiyografi yapıldı ve sirkumfleks arter ile sol ön inen arterde yavaş akım izlendi. Medikal tedavide asetilsalisilik asit ve klopidogrel kombinasyonu ile unfraksiyone heparin başlandı. Hastanın üç tane sağlıklı çocuğu sahibi ve önceki gebeliklerinde herhangi bir problem yaşamamıştı. Risk faktörlerinden aile öyküsü ve sigara içilmesi mevcut ancak diyabetes mellitus, hipertansiyon, dislipidemi yoktu. Çıkış tedavisi asetilsalisilik asit, klopidogrel ve beta bloker olarak düzenlendi ve yatışının beşinci gününde sıkı takip önerildi. Miyakart enfaktüsünden iki hafta sonra elektif abortus planlandı.

Anahtar sözcükler: Koroner trombüs; miyokart enfaktüsü; gebelik.

Introduction

Acute myocardial infarction (AMI) during pregnancy is rare but serious condition that it is a cause of maternal mortality and fetal loss. Pregnancy has been shown to increase the risk of AMI, which has been reported to occur in 3-10 cases per 100,000 deliveries.[1-3] With the rise in maternal age and the increasing number of high-risk women who become pregnant, the prevalence of pregnancy-related acute coronary syndrome (ACS) is expected to increase. Pregnancy leads to excessive hypercoagulability by increasing platelet adhesion and decreasing fibrinolysis; these hemostatic changes lead to an increased risk of thromboembolic events.[1-3] It is strongly related to the traditional risk factors of coronary heart disease, including diabetes mellitus, hypertension, dyslipidemia, family history of coronary artery disease and smoking. Additionally, other conditions that contribute to ACS are preeclampsia, eclampsia, thrombophilia, postpartum infections, severe postpartum hemorrhage, and spontaneous

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coronary artery dissections. Maternal mortality after ACS is estimated to be 5-10% and is highest during the peripartum period. Survival has improved with primary percutaneous coronary intervention (PCI).

This article accompanies pregnancy in patients with ST elevation myocardial infarction (STEMI), and we consider a general approach to treatment.

**Case Report**

A 32-year-old multiparous woman at seven weeks gestation presented to the Emergency Department (ED) with a sudden onset of chest pain within the previous 35 minutes. She had three healthy children and had no problems during previous pregnancies. She had a family history of coronary artery disease but no history of other coronary risk factors such as diabetes mellitus, hypertension, dyslipidemia or smoking. Sudden cardiac arrest developed while she was waiting in the triage room. Cardiopulmonary resuscitation (CPR) was performed and patient was immediately intubated. Ventricular fibrillation developed during CPR and defibrillation was performed. Clinical examination showed she had flexor response to painful stimuli and her pupillary light reflex was present. Her blood pressure was 90/55 mmHg with a pulse of 102 beats per minute, oxygen saturation of 96%, and normal heart sounds. There were ST elevations in leads DII, DIII, and aVF; conversely ST depression in leads V1-V4; and atrial fibrillation (AF) on electrocardiography (ECG) following CPR (Figure 1a, b). Portabilizer echocardiography was performed in the emergency department by cardiologists. The echocardiogram demonstrated that the wall motions were severely reduced in the inferior and lateral regions, consistent with AMI, and the estimated ejection fraction of the left ventricle was 40%. With the diagnosis of an inferior STEMI, aspirin (300 mg) and clopidogrel (600 mg) were given in the ED. The patient was then referred for primary angioplasty, which was performed within 45 minutes. Catheterization was performed after shielding the patient’s back and abdomen with lead aprons. Coronary angiography showed slow coronary flow of the circumflex (CX) and left anterior descending (LAD) coronary arteries. Right coronary artery was normal (Figure 2). The combination of aspirin (100 mg/day) with clopidogrel (75 mg/day for 2 weeks) and unfractionated heparin was initiated for medical treatment. Biochemistry tests [urea, creatinine, glucose, aspartate aminotransferase (AST), alanine aminotransferase (ALT)] were normal, except for mild leukocytosis. The serum levels of troponin peaked at 13 ng/ml.

We consulted an obstetrician regarding the safety of administering clopidogrel and the use of radiation after angiography, and both were consequently considered to be unsafe because organogenesis was not complete. She was discharged home on day five after admission with clopidogrel, aspirin and a beta-blocker with close outpatient follow-up. Elective abortion was planned for two weeks after the MI.

There was no prior history of connective tissue disease, vasculitis, impaired anticoagulant mechanism (protein C deficiency, protein S deficiency), or antiphospholipid antibody syndrome, which are associated with a thrombotic tendency. Birth control pills had never been used. Immunologic
tests performed prior to admission, which included antiphospholipid antibodies, were negative.

Discussion

There are few published statistics concerning acute myocardial infarction (AMI) in pregnant women. The incidence of AMI in pregnancy ranges from 3-6 cases per 100,000 deliveries.[1-4] A previous review demonstrated that pregnancy-associated AMI occurs at all stages of pregnancy. It is more common late in pregnancy, with a peak incidence during the peripartum or postpartum period. This may be due to the fact that, during this period, there is also a higher occurrence of hypertension and preeclampsia, which are the most important risk factors for AMI in these patients.[4] In our patient, however, the event was occurred relatively early, in the first trimester of pregnancy.

The physiopathology of ACS in pregnant women is quite different from the normal population. Pregnancy-associated AMI has been thought to be related to both an excess procoagulant state and increased stress on the cardiovascular system, leading to coronary thrombosis or spontaneous coronary dissection.[3-5] A study by Roth et al. reviewed coronary lesions in pregnant patients with ACS. The following results were revealed: 39% of arteries had atherosclerotic lesions, 19% were found to have an intracoronary thrombus without any other lesion, 15% had a coronary dissection, and 27% of the arteries were apparently normal.[4] As can be seen, contrary to the rest of the population, a much higher proportion of normal coronary arteries and intracoronary dissections are the cause of the coronary syndrome.[4] Paradoxical coronary embolism through a patent foramen ovale is an additional potential mechanism of myocardial infarction during pregnancy, although this case showed clear-cut coronary artery disease. Ladner et al. identified hypertension, diabetes, advanced maternal age, preeclampsia and eclampsia as independent risk factors for pregnancy-associated AMI, and James et al. also found that thrombophilia, transfusion, and postpartum infections were significant risk predictors for AMI.[2,3] Spontaneous coronary artery dissections are more prevalent among pregnant than non-pregnant women, and are mostly reported around delivery or in the early postpartum period.[4] They may be related to high progesterone levels with subsequent structural changes in the collagen of the vessel wall. Ergometrine given for bleeding postpartum may lead to coronary vasospasm and ischemia. Thrombi and dissections occur more frequently in the peripartum period than in delivery.[4] Although our patient was young and did not have any risk factors for pregnancy-associated AMI, percutaneous coronary angiogram demonstrated normal coronary arteries with slow flow in circumflex, the ECG was STEMI.

Maternal mortality after ACS is estimated at 5-10% and is highest during the peripartum period. Survival has improved with PCI.[2-4] Long-term maternal prognosis mainly depends on the severity of maternal heart disease and the cardiovascular risk profile. Before delivery, ACS may result in fetal mortality or prematurity.

The first step during pregnancy in ST-elevation ACS is a primary PCI, which is preferred to thrombolysis as it will also diagnose coronary artery dissection. Although recombinant tissue plasminogen activator does not cross the placenta, it may induce bleeding complications such as subplacental bleeding; therefore, thrombolytic therapy should be reserved for life-threatening ACS when there is no access to PCI.[6]

The risk of potential damage to the fetus should be kept in mind, especially in the first trimester. However, this can be overcome with the use of appropriate radiological protection. In our case, a decision was taken to use lead apron protection. The patient’s abdomen was covered and, to protect the fetus from excessive radiation, additional protection was also placed between the patient’s back and the table.

β-Blockers and low dose acetylsalicylic acid are considered to be relatively safe during both the second and third trimesters, while the safety profile is unknown for thienopyridines.[7] Heparin does not cross the placenta, and several reports have demonstrated that heparin is safe to use during pregnancy.[8]

In conclusion, AMI in pregnancy is a rare event with specific features that are related to both an excess procoagulant state and increased stress on the cardiovascular system, leading to coronary thrombosis or spontaneous coronary dissection.
on. Understanding the causes of pregnancy-related acute myocardial infarction and identification of women at risk are the first steps for prevention. Screening and preventive measures should focus on women with advanced maternal age, known coronary risk factors, thrombophilia, postpartum complications, and being a smoker. Family history and smoking are the most important risk factors, and these have a higher-than-normal rate for non-atherosclerotic coronary disease in these groups. Management of these patients should include the use of bare-metal stents, precaution with the use of antiplatelet therapy, and appropriate protection during radiological procedures.

Our patient had normal coronary artery with slow coronary flow and drug treatment was administered. Elective abortion was planned for two weeks after the MI.

This article suggests that acute coronary syndrome should be considered in pregnant women who have chest pain. In these patients, the ECG should be performed immediately.

Conflict of Interest

The authors declare that there is no potential conflicts of interest.

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