Acute Myocardial Infarction in Pregnancy

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ABSTRACT

Acute myocardial infarction is a rare event in pregnancy and can be fatal. We describe a case of a thirty years old woman who presented in the eighth month of pregnancy with ante-partum haemorrhage and shock. An emergency caesarean section under general anaesthesia was performed to extract a still born foetus. The shock which was out of proportion to the blood loss was eventually diagnosed as Acute MI .The patient was managed conservatively and recovered well.

Key Words :

Acute myocardial infarction, APH, cardiovascular complications, caesarean section, haemorrhagic shock, risk factors, pregnancy.

INTRODUCTION

Acute myocardial infarction (AMI) is an important cause of mortality and morbidity worldwide. Its occurrence in pregnant women is uncommon. It has been determined that AMI occurs in 1 in 35,000 pregnancies and has serious consequences on the continuation of the pregnancy as well as the well being of the developing foetus. [1] AMI occurs mostly in the third trimester and in puerperium and has a high mortality rate (5 to 11 %). [1,2] The case reported merits attention because of the rarity of its occurrence in women of reproductive age

Case Report

A 30 year old woman in the eight month of pregnancy was admitted on account Ante-partum haemorrhage [APH] with shock in the emergency department of our hospital. The patient was gravida 5 with a history of 4 full-term normal deliveries [3 female children & 1 male child]. The last delivery was about a year and half back and the patient was fully immunized. The patient presented with a history of bleeding per vaginum and pain abdomen of six hours duration. On examination, the patient was pale in countenance, conscious, clammy with regular but high pulse rate of 128/min. blood pressure was 70 mm Hg systolic [on Dopamin Herne infusion], the respiratory rate was 26/min, and urinary output was 400 cc after catheterization.

The patient's past history, medical history and family history was unremarkable. Routine ultrasonography [USG] done six days earlier had revealed a single live foetus of 32.6 weeks size in breech presentation and an anterior placenta with its margin lying on internal os with grade I maturity . The foetal weight was estimated to be 2079 grams and the expected date of delivery was three weeks away.

On per-abdominal examination, the patient's uterine height corresponded to 32 weeks size foetus and it was not acting, breech presentation. The foetal heart sounds were normal. On local examination, her clothes were soaked with dried blood but there was no visible fresh bleeding. A provisional diagnosis of APH with haemorrhagic shock was made. Intravenous colloids and crystalloids were started. Investigations revealed that the haemoglobin was 5.5 gm%, total leucocyte count was 11,000 mm / 100 ml and the platelet count was 2.44 x 10^5/100ml. Tests for renal function, liver function and chest x-ray were within normal limits. The patient's oxygen saturation was 98%. One unit of packed RBC was infused because of anaemia. The patient was immediately shifted to operation theatre.

On arrival in operation theatre, the patient was drowsy, her pulse 160/min regular, while the systolic blood pressure on ionotropic support varied between 70 - 120 mm of Hg. While preparations for surgery was going on the patient developed ventricular tachycardia and was cardioverted to sinus rhythm. An emergency Caesarean Section was performed and a dead female child weighing 2.2 kilograms was extracted. The patient re-developed Ventricular Tachycardia soon after delivery of the baby, and was cardioverted again. The placenta was covering the internal os. A hysterectomy was performed as the patient developed atonic post-partum haemorrhage and as her general condition was poor. On completion of surgery the patient was shifted to the intensive care unit and electively mechanically ventilated. At this time the electrocardiogram (ECG) showed changes of acute anterior myocardial infarction (AMI) with ST elevation in leads I, aVL, V1 to V6. Elevated levels of cardiac enzymes, Trop. T and CPK MB, were found.

The patient was managed for acute MI with low molecular weight heparin, oral Clopidogrel and Aspirin [75 mg each] and Atorvastatin. She was not thrombolysed because of her recent surgery. The patient responded well to treatment and the inotropic support was withdrawn on the third day. Echocardiography revealed hypokinesia in left anterior descending (LAD) artery territory with left ventricular ejection fraction of 0.40. The patient was discharged on the seventh post-operative day in a stable condition. A coronary angiography done later showed single vessel disease with 70% lesion in mid LAD.

Discussion

AMI in pregnancy is quite uncommon but when it occurs, it is associated with poor maternal and foetal outcomes. Myocardial infarction during pregnancy was associated with a 35% mortality rate a few decades ago but with improved diagnosis and treatment, the current mortality is reported to be 11%, and it mostly occurs at the time of infarction or within 2 weeks after it. [3] The mortality is twice as high when AMI occurs during the per-partum period as compared to that occurring in the ante-partum and post-partum periods. A foetal mortality of 9% has been reported and it is mainly associated with maternal mortality. [3] Gravidity too has a bearing on the incidence of AMI and AMI is more common in multigravida, especially during the third trimester, than in primigravida women. [2]

Prompt diagnosis of AMI during pregnancy is often difficult due to the rarity of the condition and also because treating physicians may associate features of AMI with normal pregnancy manifestations. In this case too, an initial diagnosis of haemorrhagic shock was made because of the APH caused by placenta praevia. The post-operative electrocardiogram [ECG] was however suggestive of AMI and the diagnosis was confirmed by elevated cardiac

enzymes. Although rare it may be, AMI should always be kept as a differential diagnoses when confronted with APH and hemorrhagic shock and an ECG must be recorded promptly lest this rare and often fatal condition be missed. Failure to promptly diagnose and manage AMI during pregnancy leads to devastating long-term loss of cardiac function associated with delayed intervention in case the patient survives the episode.

Electrocardiographic diagnosis of AMI in pregnant women can be life saving. All obstetricians and care-giving medical/ paramedical staff in the ante-natal clinic as well as the labour room must be sensitised about this. The diagnostic criteria for MI in the case of pregnant women are identical to those for non-pregnant patients. The criteria include symptoms, elevated cardiac enzyme concentrations in blood and ECG changes such as development of Q waves or ST segment changes. [4] Diagnosing AMI requires a consummate understanding of the pregnancy associated changes that could be observed in a twelve-lead ECG.

Induction of anaesthesia for caesarean sections frequently results in misleading ST-segment depressions. Significant ST-segment changes have been reported in 42 % of the 26 patients undergoing elective caesarean sections and in 38.5% of these patients after the operation. [5] The physiological changes during pregnancy may also affect ECG presentations. In the third trimester, the upward displacement of thee diaphragm by the foetus, causes left axis deviation in the ECG. Other reported ECG changes include Q waves in lead III and augmented vector foot (aVF) and mildly inverted T waves in lead III. [6]

The risk factors for myocardial infarction [MI] include hypertension, smoking, family history of MI and hyperlipidaemia. In pregnancy, alteration in the coagulation system, increase in clotting factors (II, VII, VIII, IX and X), fibrinogen levels, platelet turnover and reduction in functional protein S levels decreasing fibrinolytic activity may contribute in precipitating MI. [7] Pre-eclampsia may play a role in ischaemic syndromes by causing an increase in workload and inducing the dissection of the coronaries. [2] Since Progesterone increases plasminogen activator inhibitor levels enhancing thrombus levels, there is an increased tendency of thrombo-embolism in pregnant women. The most common cause of MI during pregnancy is reportedly vasospasm along with a hypercoagulable state. [8] The unusual case of a woman who suffered a MI during caesarean section has been reported but the patient had angiographically normal coronaries, however, an intravascular ultrasound study demonstrated an atheroma with ruptured fibrous cap. [9] Significant risk factors for MI during pregnancy include advanced maternal age, black race, hypertension, thrombophilia, anaemia, diabetes mellitus, smoking and preeclampsia. [1, 10] In the case reported in his communication, lipid profile was normal, and there was no family history of MI. Anaemia followed by haemorrhage appears to be the most probable triggering factor for the MI in the case reported.

In conclusion, AMI during pregnancy has high maternal and foetal mortality rates. It is essential to maintain suspicion for the condition in case of pregnant patients presenting in shock. Timely recognition of its occurrence improves management and ensures maternal and foetal survival and well-being.

Conflict of Interest

The authors declare that they have no conflict of interest.

References

1. Ladner HE, Danielson B, Gilbert WM. Acute myocardial infarction in pregnancy and the puerperium: a population-based study. Obstet Gynecol 2005;105:480–4.

2. . Roth A, Elkayam U. Acute myocardial infarction associated with pregnancy. Ann Intern Med 1996;125:751-62.

3. Roth A, Elkayam U. Acute myocardial infarction associated with pregnancy. J Am Coll Cardiol 2008;52[3]:171-80.

4. Pierre-Louis B, Singh P, Frishman WH. Acute inferior wall myocardial infarction and percutaneous coronary intervention of the right coronary during active labor – a clinical report and review of the literature. Cardiol Rev 2008;16:260-8.

5. Moran C, Ni Bhuinnedin M, Geary M, Cunningham S, McKenna P, Gardiner J. Myocardial ischemia in normal patients undergoing elective cesarean section: a peripartum assessment. Anaesthesia 2001;56:1051-8.

6. Sheik AU, Harper MA. Myocardial infarction during pregnancy: management and outcome of two pregnancies. Am J Obstet Gynecol 1993;169:279-84.

7. Koh CL, Viegas OA, Yuen R, Chua SE, NG BL, Ratnam SS. Plasminogen activators and inhibitors in normal late pregnancy, postpartum and in the postnatal period. Int J Gynaecol Obstet 1992; 38: 9-18.

8. Badui E, Enciso R. Acute myocardial infarction during pregnancy and puerperium: a review. Angiology 1996; 47: 739-56

9. Kulka PJ, Scheu C, Tryba M, Grünewald R, Wiebalck A, Oberheiden R. Myocardial infarction during pregnancy. Anaesthesist 2001; 50: 280-4.

10. James AH, Jamison MG, Biswas MS et al. Acute myocardial infarction in pregnancy: a United States population based study. Circulation, March 2006; 113: 1564-71.