

CASE REPORT: ANAESTHETIC MANAGEMENT OF ACUTE MYOCARDIAL INFARCTION IN PREGNANCY

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Abstract

Myocardial infarction is encountered rarely during pregnancy, but when it occurs the event is life threatening to both mother and fetus. Data on maternal and fetal outcome are limited, but overall maternal mortality approaches 35% to 40% of deaths occur during the third trimester. We present a case of myocardial infarction at 36 weeks of gestation, and discuss the anaesthetic management of the problems encountered during the delivery.

Keywords: Myocardial Infarction, Prgnancy, PPCM (Peripartam Cardiomyopathy), Cesarean Section.

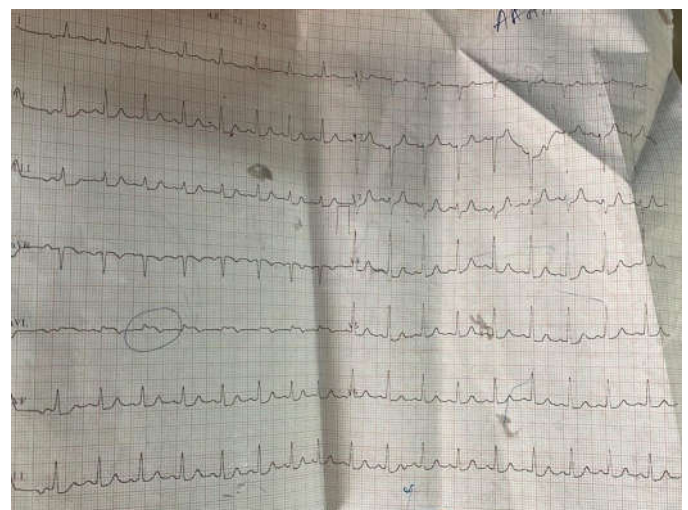
INTRODUCTION

Myocardial infarction is encountered rarely during pregnancy, but when it occurs the event is life threatening to both mother and fetus. Data on maternal and fetal outcome are limited, but overall maternal mortality approaches 35% to 40% of deaths occur during the third trimester. We present a case of myocardial infarction at 36 weeks of gestation, and discuss the anaesthetic management of the problems encountered during the delivery.

CASE REPORT

A 28 year old multigravida with 36 weeks period of gestation admitted in emergency with complaint of chest pain, single episode of syncope not associated with any abnormal body movement. Her previous obstetric history was unremarkable. On physical examination she was pale, distressed, initially her blood pressure was 110/60 mmHg, heart rate was 125 bpm with irregular pulse and saturation was 97% at room air. On auscultation chest was clear with normal heart sounds. Examination of abdomen was consistent 34 weeks of pregnancy, fetal heart rate was regular but low. Routine investigations were sent, ultrasonography was done which showed single live fetus with cephalic presentation with gestational age 36weeks±3 days. Placenta anterior wall upper segment, umbilical artery showed reduced diastolic flow which called for emergency lower segment caesarean section. Anaesthesia call was attended and on examination patient was conscious, cooperative well oriented to time place and person. Afebrile, blood pressure 90/60 mmHg, heart rate 116 bpm, patient was tachypneic with respiratory rate 20-25 breaths per minute and maintaining saturation of 95-96% at room air. No calf tenderness or swelling was present on examination. A 12 lead ECG was done and ECG showed ST elevation in lead I and aVL with contralateral depression in leads V4, V5, II, III and Avf likely high inferiolateral wall myocardial infarction. Physician call was done, and advised Trop T testing, 2D – ECHO, and cardiac consultation.

Trop T came positive meanwhile ABG done which was within normal limits with pH of 7.4mmHg, pO2 90 mmHg, pCO2 38mmHg, HCO3-22%. Hb was 9gm%, coagulation profile and rest of investigations were within normal limits.



Patient was taken inside operation theatre and an informed written high risk consent in view of possible major cardiac adverse event and post operative mechanical ventilation was taken. 2 I/V lines were secured with 18 G cannula, monitors attached (NIBP, ECG, SPO2), meanwhile Patient developed monomorphic ventricular tachycardia with heart rate of 210, blood pressure 90/40mmHg and loss of consciousness. Immediately 200 joules of DC shock was delivered, Inj. Amiodarone 150mg i/v given over 5mins, a normal sinus rhythm was achieved and infusion of inj. Amiodarone @60mg/hour started for first 6 hours followed by 30mg/hour for next 18 hours. Simultaneously LSCS was planned and Induction was done with inj midazolam 2mg i/v + inj etomidate 18 mg i/v, inj succinylcholine 50mg i/v stat and Rapid Sequence intubation was done orally with 7 mm cuffed endotracheal tube, confirmed by bilateral equal air entry. Anaesthesia was maintained with O2 + N2O + inj atracurium 25 mg i/v. Patient had another episode of pulseless ventricular tachycardia after delivery of baby immediately chest compressions were started and again 200 joules of DC shock

was delivered. Normal sinus rhythm was achieved & patient was revived after 1 min of CPR and DC shock. An ABG was sent in intraoperative period which was within normal limits and total surgical time was 45 mins. APGAR score of neonate was 10/10 after 1 minute. Analgesia was maintained with inj butrum 0.5mg i/v +inj fentanyl 50 mcg i/v after delivery of baby. Intravenous fluid were given judiciously, urine output was 500 ml at the end of surgery. Another ABG was done before shifting to ICU which shows PH - 7.46, pO₂-227, pCO₂- 36, HCO₃- 27 Patient was not extubated and shifted to gynae ICU for further monitoring and elective mechanical ventilation. Patient was sedated with infusion of inj midazolam 4mg/hour for 24hrs and inj butrum 1mg 6hrly. . In ICU Tablet Aspirin 325mg was given to patient via ryles tube. Cardiology call was attended, they advised APLA, ANA, coagulation profile, thyroid profile, bed side 2D ECHO(unavailable in our hospital) and Doppler after weaning of patient. She was put on inj enoxaparin 60 mg BD, tab clopidogrel 300 mg stat followed by 75 mg OD, tab bisoprolol 2.5 mg BD, tab atorvastatin 80 mg stat followed by 20 mg OD. As patient was haemodynamically stable so extubated smoothly on first post operative day. Meanwhile her investigations were received which were within normal limits. APLA IgG -3, IgM -8, PT-13sec, TSH 2.4, ANA 0.4, TROP I -0.06ng. ECHO was done which shows the findings of global hypokinesia, mild mitral regurgitation, EF 35-45% (suggestive of peripartum cardiomyopathy). Her revised treatment was tab Atorvastatin 20 mg OD, tab Metoprolol 25 mg BD. On day 5th of admission patient was shifted to cardiology unit for further management.

DISCUSSION

Ischaemic heart disease in pregnancy is not common, seen in an estimated 1 in 10 000 deliveries (Dwyer *et al.*, 2005). The highest incidence of acute myocardial infarction (AMI) has been reported in multigravidas older than 33 years during the third trimester (Roth and Elkayam, 1996). Pregnancy is a procoagulant state increasing stress on the cardiovascular system, particularly during delivery. If untreated, AMI in pregnancy has a high mortality up to 37–50% (Thorne, 2006). In the absence of fetal distress, delivery should be postponed at least 3 weeks post myocardial infarction (Duarte *et al.*, 2011). Peripartum cardiomyopathy (PPCM) is a disease affecting the parturient during late pregnancy or immediately after delivery (Ramachandran *et al.*, 2011). Symptoms of PPCM are fatigue, edema and dyspnoea are similar to the normal spectrum of peripartum state and pregnancy co morbidities such as pregnancy induced hypertension (Dutt *et al.*, 2013). The hallmark of the disease is onset of decreased cardiac ejection fraction either in the late pregnancy or early puerperium (Ramachandran *et al.*, 2011). For any urgent or emergent lower segment caesarean section (LSCS), GA(General Anaesthesia) is preferred. GA is also preferred in patients with borderline cardiac decompensation as an already dyspneic patient may not be amenable to the procedure of RA (Regional Anaesthesia). In such a patient, even minor degrees of sympathetic blockade associated with RA may lead to fulminant cardiac failure.

Another contraindication to RA is the anticoagulated patient (Ramachandran *et al.*, 2011). In general, RA has been used in patients undergoing non emergent caesarean section with relatively stable hemodynamics, while moderately symptomatic patients or parturients undergoing emergency surgery should receive general anaesthesia (GA) (Mellor and Bodenham, 1996). PPCM is a disease associated with high morbidity and mortality, it can lead to maternal and fetal loss. Early diagnosis, continued monitoring and prolonged therapy may be beneficial in many patients. Principles of therapy remain more or less same as that in heart failure. In PPCM cases anaesthetic considerations are to maintain a normal physiology in terms of acid-base balance, hypoxia and anaemia regardless of the anaesthetic technique, haemodynamic goals include avoidance of sudden variation in heart rate and blood pressure. Initial treatment with aspirin and heparin is probably safe (Sebastian *et al.*, 1998). A careful continuous assessment of intravascular volume is vital in the preoperative, intraoperative and postoperative period to ensure safe management. Integrated management by obstetricians, cardiologists and anaesthetists is essential. Invasive maternal monitoring during labour and delivery and continuous fetal heart rate recording should be considered (Ray *et al.*, 2004). To conclude, in developing nations not all parturients undergo regular antenatal checkups and in emergency conditions full evaluation of patients is not possible in our set up. So high level of clinical suspicion is very important for early diagnosis and anesthetic management of PPCM for better patient outcome.

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