**Case report:**

**A parturient with acute myocardial infarction during labour for management of labour analgesia and obstetric anesthesia during emergency caesarean delivery**

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**Abstract:**

Acute myocardial infarction (AMI) during pregnancy is rare emergency, having potential risk for adverse fetomaternal outcomes. AMI accounts for 5.6% of the people under 40 years and females being only 0.7%. During pregnancy incidence is 1:10,000 to 1:30,000. During entire pregnancy, third trimester is the period where AMI is more common. Till 2005 around 230-250 AMI during pregnancy are reported in literature since 1920, but none is reported of AMI during labour. High index of suspicion and early diagnosis is a keystone for favourable fetomaternal outcome along with multidisciplinary team approach during management of parturient with AMI. We report a case of 28 year old parturient (G3P2L2 with 37weeks of gestation) admitted in active labour with breathlessness, chest pain diagnosed with AMI. She was posted for labour analgesia and converted into emergency caesarean section because of foetal distress.

**Keywords**: Anaesthesia management, labour analgesia, Acute Myocardial Infarction, Emergency Caesarean Section

**Introduction:**

Coronary artery disease is uncommon in individuals below 30 years of age. Acute myocardial infarction (AMI) accounts for 5.6 % of the people under 40 years and female cases being only 0.7%1. AMI is an infrequent event during pregnancy and labour having an incidence ranging from 1:10,000 to 1:30,000 of the pregnancies1-3.Management of AMI and anaesthesia during labour is rare in literature. We report a case of multigravida who suffered with AMI at the onset of labour.

**Case Report:**

Antenatally unregistered; 28 years old parturient (G3P2L2 and 37 weeks of gestation) admitted with breathlessness [NYHA grade3], chest pain, palpitations and active labour pain in emergency room[ER]. Previous two pregnancies were uncomplicated with normal vaginal deliveries. Past history revealed similar episodes of chest pain, palpitations and diagnosed of hypercholesterolemia 18 months back, but not on any treatment. Cardio-respiratory auscultation was normal. Electrocardiogram (ECG) revealed T wave inversion in leads I-III, V1-V6 (Global T wave inversion). Blood pressure (BP) was 154/96 mmHg and pulse rate 112/minute and respiration rate 20/minute. As she had chest pain (30 min duration, fixed severity), palpitations, breathlessness, ECG changes and past history of hypercholesterolemia, she was diagnosed as stable angina by physician and referred for labour analgesia. Blood sample was sent for CBC, coagulation profile, serum electrolytes, lipid profile, thyroid functions and cardiac enzymes. Patient was managed by sublingual Nitro-glycerine sorbitrate and tab Aspirin 125mg and planned for labour analgesia. High risk written informed consent was confirmed. Emergency cardiac drugs and defibrillator were kept ready. Intravenous anti-aspiration prophylaxis rantidine50mg and metoclopramide 10mg was given. Monitors were attached -12 lead ECG, pulse-oximeter, NIBP, temperature monitor. Intravenous fluid Ringer lactate was started. Invasive blood pressure and central venous pressure monitoring was kept ready aside. Foetal heart rate monitoring was done with cardiotocogram. With all aseptic precautions, lumbar epidural catheter was inserted at L3-L4 level. Test dose Lignocaine2% plain, 3cc was given. During labour analgesia process foetal distress occurred in half hour, so obstetricians decided for emergency caesarean delivery. Spinal anaesthesia was given with 27G Quincke’s spinal needle using 0.5% heavy Bupivacaine 2cc and level of block T6 was confirmed. Judicious fluid therapy was given. Intra-operatively inj.NTG (0.5ug/kg/min) infusion started & oxygen supplementation by ventimask. Baby weighing 3100gm was delivered with APGAR score 9 and 10 at 1 and 5min. Infusion of Oxytocin 20 units was given very slowly. Surgery duration was 30 min with 200ml blood loss. Considering the need of postpartum cardiac angiography, epidural catheter was removed after giving analgesic dose (Bupivacaine 0.0625% 4cc, tramadol 50 mg). Post-operatively patient was managed in intensive care unit.

Postop Day 1: Vitals stable. Diagnosis of AMI was confirmed with raised CPK MB, Trop I. Lipid profile deranged. Inj. NTG 0.5ug/kg/min IV and Oxygen supplementation continued. Cardiologist added Tab.Metoprolol 25mg and Tab.Atorvastatin 40mg OD. Postop day 2: Vitals stable, 2Decho showed EF 60%, septal wall hypokinesia and normal systolic function, Inj. NTG was stopped. Patient was shifted to postnatal ward on day 3 and discharged home on day 10 with cardiology follow-up. Treatment on discharge was Tab.Metoprolol 25mg, Tab.Atorvaststin 40mg, and Tab.Aspirin75mg.

**Discussion:**

AMI is an infrequent event during pregnancy and labour having an incidence ranging from 10,000 to 1:30,000 of the pregnancies1-3. Since 1920-2005 only 251 cases of MI have been reported in parturient. During entire pregnancy, third trimester is the period where AMI is more commonly seen.4.Coronary angiography reveals no pathological findings in more than half of the cases. The overall mortality rate of women with AMI during pregnancy has been estimated at 37%, increasing to 50% if delivery occurred within two weeks of acute infarction. AMI leads to irreversible myocardial injury resulting in necrosis of myocardium and high mortality, hence high index of suspicion and early diagnosis is a cornerstone for favourable feto-maternal outcome 2, 3.

**Pathophysiology of AMI in pregnancy**: IHD (Ischaemic Heart Disease) is classified into angina pectoris (transient myocardial ischaemia) and MI (fixed myocardial ishaemia/necrosis). Myocardial infarction presents with central chest pain, severe in nature, radiating to the left arm and jaw, may be associated with fainting, profuse sweating, anxiety, nausea/vomiting. Angina pectoris should be vigilantly managed as it has potential risk to convert into AMI in parturient. Non-obstructed coronary vasospasm is more common cause than the obstructed coronary artery disease (arteriosclerosis, embolism) for AMI in pregnancy. Reproductive age is protective for AMI but pregnancy increases risk 3-4 times due to hypercoagulability and abnormal fibrinolytic system. Factors such as multiparity, maternal obesity, pre-eclampsia, increased maternal age, thrombophilia, diabetes mellitus, tobacco, blood transfusion, postpartum infection, family history are associated with increased risk of pregnancy related AMI5. Multiple cases of AMI are seen in young patients (25–89%), have been associated with hypercholesterolaemia6.Hong etal 7 reported hypercholesterolemia as important risk factor for acute coronary syndromes in young patients than in elder patients. Our patient had hypercholesterolemia which is an independent risk factor with advance maternal age and multiparty.

Cardiovascular changes in pregnancy prime the patient with unrecognised cardiovascular disease which progresses to development of symptoms of myocardial infarction .These changes are raised plasma volume by 50%, raised stroke volume by 30-50% raised cardiac output by 40-60% which is further increased up to 80% during delivery due to autotransfusion. Thus there is sharing of common clinical features in parturient with AMI making diagnosis possible only if vigilant.

Anaesthetic considerations are focused on 1) evaluation of disease and risk stratification 2)maintaining hemodynamic stability 3) adequate analgesia 4)modify anaesthesia technique for labour and delivery 4)vigilant monitoring and multidisciplinary team approach5) preparedness for resuscitation in emergencies like pulmonary oedema, heart failure or arrhythmias.

Oxygen supplementation, bed rest in left lateral, judicious fluids, analgesia, anxiolysis and sublingual Nitro-glycerine should be started immediately. Preparedness for specific surgical or medical measures for AMI should be consulted with cardiologists. Two weeks interval if possible between vaginal deliveries and AMI suggested to reduce maternal mortality by 50%. Vaginal deliveries with cut-short of second stage of labour are acceptable and caesarean deliveries should be reserved for obstetric reasons. Caesarean delivery increases risk for blood loss, surgical stress, infection, re-infarction, cardiopulmonary complications. The anaesthesiologist must primarily consider the prevention of increases in pulmonary vascular resistance from under ventilation, anesthetic drugs, pulmonary hyperinflation, and stress. Epidural analgesia is useful due to excellent reduction in pain, stress, anxiety, reduced afterload and benefit of epidural anasthesia for emergency caesarean section, as time to effective surgical anaesthesia may be shortened9. Further, general anaesthesia was avoided because of risk of aspiration, difficult intubation, pressor response, high prevalence of cardiovascular depression, maternal awareness during light plane of anaesthesia, increased intra-operative blood loss and delayed postoperative recovery.

We preferred regional anaesthesia in due to its benefits like excellent perioperative analgesia, no pressor response, awake mother for breast feeding and less intaoperative hemorrhage.

After delivery, the parturient should undergo cardiovascular evaluation to provide diagnostic and prognostic information, thereby aiding further management. In our case, parturient with AMI, labour analgesia was successfully converted to obstetric anaesthesia for emergency caesarean section and accomplished with favourable feto-maternal outcome

**Conclusion**:

The peripartum management of parturient with AMI should include multidisciplinary teams. A comprehensive understanding of physiology of pregnancy and pathophysiology of underlying cardiac disease is of primary importance in provision of labour analgesia and obstetric anaesthesia.

Epidural analgesia was given for labour pains and emergency caesarean section was managed under spinal anaesthesia successfully in this parturient who had AMI during labour.

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