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Original Research Paper

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CASE SERIES OF PREGNANCY-ASSOCIATED MYOCARDIAL INFARCTION: AN EPIDEMIC IN WAITING

Dr. Shree Bharathi* MBBS, MS, DNB (OG) Senior Resident, Department Of Obstetrics And Gynaecology, Jawaharlal Institute Of Postgraduate Medical Education And Research Puducherry-06, India. *Corresponding Author

Dr. Sasirekha Rengaraj MBBS, MS, DNB, Fellow In Obstetric Medicine Additional Professor, Department Of Obstetrics And Gynaecology, Jawaharlal Institute Of Postgraduate Medical Education And Research Puducherry-06, India.

ABSTRACT Acute myocardial infarction during pregnancy or postpartum period is a rare but life-threatening condition associated with poor maternal and fetal outcome.

Although atherosclerotic coronary artery disease is the most common etiology in general population, the causation is more diverse in pregnancy and this may have therapeutic implications.

Early diagnosis and timely management with collaboration among Maternal-fetal medicine specialist, interventional cardiologist, cardiac anaesthetist, intensivist and neonatologist is essential to prevent maternal cardiac deaths.

We present a case series of two patients with postpartum and antepartum acute MI, respectively and their diagnosis, management and outcomes in a tertiary care center.

KEYWORDS : Anti-platelets, Coronary angiogram, Pregnancy associated myocardial infarction, Takotsubo cardiomyopathy

BACKGROUND

Ischaemic heart disease(IHD) in pregnancy is a major contributor to maternal mortality and poor perinatal outcomes and is the most common cardiac cause of death in developed countries.(Wuntakal et al., 2013) Pregnancy increases risk of myocardial infarction (pregnancy-associated MI or PAMI) four-fold and this risk is 30times higher in elderly gravidas >40years. Overall incidence of PAMI is low, occurring in 2 and 4.2 per 1,00,000 pregnancies antepartum and postpartum respectively, with half of them being immediate postpartum.(Turitz & Friedman, 2014)

The most important cause for IHD is atherosclerosis and its risk factors in pregnancy include preeclampsia and other comorbidities like chronic hypertension, smoking, obesity etc. A steep rise in incidence is noted over past 2decades. In high-risk patients, multiple cardiovascular adaptations of pregnancy and labour, overload the compromised coronary blood supply and lead to ischaemia and MI. Stress-induced coronary vasospasm can also cause ischaemia and MI. Therefore, high index of suspicion is essential and antenatal and peripartum monitoring is necessary in patients with risk factors to prevent/reduce risk of cardiac deaths.

We present two cases of acute MI in pregnancy with different presentations and deliberate on their management in our tertiary care centre.

CASE REPORTS

Case 1:

A 36-year-old elderly primigravida presented to us at 32+5 weeks with threatened preterm labour, with monochorionic diamniotic twin gestation. She was noted to be of short stature(135cm) and was anemic(Hb-8.8g/dl). On obstetric evaluation, there was no discordancy between the fetuses and growth was appropriate with reassuring fetal heart rate patterns. She received antenatal corticosteroids and tocolysis with nifedipine, following which her contractions subsided.

A week later, she developed preterm premature rupture of membranes(PPROM) and spontaneously went into labour at 33+6 weeks of gestation. In the intrapartum period, she had one episode of atypical left sided, non-radiating chest pain which subsided after some time. It was not accompanied by electrocardiogram(ECG) and Echocardiogram(ECHO) abnormalities. Although fetal heart rate patterns were reassuring, due to cephalopelvic disproportion(CPD), she was taken up for emergency caesarean section(CS) under spinal anaesthesia. Two healthy male babies of 2.01kg and 1.80kg respectively, were delivered. There was uterine atonicity which was managed medically with slowly infused intravenous Oxytocin, intramuscular methylergometrine, intra-myometrial carboprost and sublingual misoprostol. She sustained a blood loss of 600ml during the surgery. End-operatively she developed hypertensive crisis with high bp recordings in the range of 190/110mmHg which was controlled by intravenous Labetalol 20mg.

Six hours into the post-operative period, the patient started to desaturate and bilateral crepitations were noted on lung auscultation. With the diagnosis of pulmonary edema, she was administered intravenous diuretic(furosemide) and morphine. Cardiac evaluation was normal and the flash pulmonary edema was attributed to hypertensive crisis. However, over the next 8hours, she worsened, with desaturation, hypotension, tachycardia, hypoxemia and metabolic acidosis. She was resuscitated with inotropes and started on mechanical ventilation.

Serial ECG showed ST-segment elevation in chest leads V3 to V6 and leads I and aVL. ECHO showed new-onset hypokinesia of antero-septal, anterior and antero-lateral walls along with dilated left atrium and dilated ventricle, and serum levels of Troponin-T were grossly elevated. The patient was diagnosed with ST-elevation myocardial infarction(STEMI) of anterior and anterolateral wall belonging to Killip's class IV. She was immediately administered loading dose of Aspirin(300mg), Clopidogrel(300mg), Atorvastatin(80mg) and intravenous Heparin(5000U) followed by maintenance dose of the same drugs as per Cardiologist's opinion. After stabilization, coronary angiogram(CAG) was performed under high-risk and revealed normal coronary-vessel anatomy. Thus, no intervention was required. STEMI was concluded to be most likely due to stress induced cardiomyopathy or Takotsubo Cardiomyopathy.

Over the next 3 days, inotropic support was slowly tapered and she was extubated. She recovered well and was continued on the above medications. After 2weeks, repeat ECG showed persistent ST segment elevation in anterior chest leads V2 to V6 and reciprocal depression in V1 and aVR.

ECHO showed concentric left ventricular hypertrophy with ejection fraction of 55%. She was counselled regarding need for regular cardiology follow-up and contraception and was discharged with antiplatelets, statins and beta-blockers.

Case 2:

A 29year-old multigravida with obstetric index G3P1L1A1, with previous CS, was referred to us at 37weeks of gestation with history of substernal chest pain 2weeks back which was diagnosed at her primary hospital to be Acute coronary syndrome/ non-ST elevation MI at 34+5weeks, where she underwent heparinisation for 8days and then was thrombolysed with streptokinase in view of an apical intraventricular thrombus.

She had past history of a spontaneous first trimester abortion and a term CS with an uneventful antenatal period.

At admission, she was asymptomatic, hemodynamically stable and her obstetric evaluation was satisfactory. Her ECG showed ST elevation in chest leads V2-V4, QS pattern with normal axis. ECHO showed anterior wall hypokinesia, more apical than basal, with an ejection fraction of 55%. Hence, diagnosis was revised to STEMI with ventricular aneurysm. Cardiologist was consulted and dual antiplatelets, furosemide and metoprolol were continued.

At 38+5weeks, 4weeks after MI, she underwent elective CS with concurrent sterilisation. Though, she had no obstetric contraindication for vaginal delivery, in view of recent MI, she was taken up for CS under spinal anaesthesia with moderate cardiac risk. Perioperative period was uneventful and mother and baby were stable. She received post-operative thromboprophylaxis and ivabradine(anti-failure therapy) for 5days. She was discharged with cardiac medications as per cardiologist advice and was planned for CAG and thrombophilia workup after 6weeks at follow-up.

We compare the cardiac and maternal characteristics of both the patients in table 1.

Table 1: Summary of maternal obstetric and cardiac characteristics

Sl. no	Age	Obstetric Index	Gestational age at presentation	Occurrence of MI	ECG and ECHO findings	Complications	Intervention	Outcome	Indication for CS
1	36	Primi-gravida	32+5 Weeks	Postpartum day 1	ST^ in V3 to V6, I, aVL; New onset hypokinesia of AS, AW, AL, dilated LA, LV	Cardiac failure with cardiogenic shock* Metabolic acidosis; Mechanical Ventilation; ICU stay for 4days	CAG Normal anatomy PCI deferred	Recovery after intensive care	Cephalopelvic disproportion
2	29	G3P1L1A1	37weeks	34+5 weeks	V2-V4, QS pattern; AW hypokinesia, EF 55%.	Apical Intraventricular clot	Thrombolysis, Heparinisation	Recovery after thrombolysis	Recent MI (4weeks)

Note: ST^- ST segment elevation; AS- antero-septal, AW- anterior wall, AL- antero-lateral wall;
LV- Left ventricle, LA- Left atrium; EF- left ventricular ejection fraction
*categorized as class IV in Killip Classification of Acute MI (Killip & Kimball, 1967)

DISCUSSION

PAMI is a rare but life-threatening event and causes prolonged hospital stay, possible need for coronary interventions and prolonged ventilation, and may lead to sudden cardiac deaths in 5-50%cases and fetal demise in 12-34%.(Kealey, 2010; Wuntakal et al., 2013) Its incidence is 0.6-1 per 10000live births.(Regitz-Zagrosek, 2017; Turitz & Friedman, 2014)

The most important cause is atherosclerosis and thus risk is much higher with advancing maternal age; other causes include coronary artery spasm, spontaneous coronary dissection and thrombosis, Kawasaki disease, pheochromocytoma etc.(Kealey, 2010; Turitz & Friedman, 2014) Pregnancy induced changes in vessels, especially root of the aorta may predispose to dissection which is the closest differential diagnosis for PAMI. Both MI and dissection together constitute common cardiac causes of mortality.

In the absence of coronary artery disease(CAD), i.e. a normal coronary vessel anatomy on CAG, stress-induced or Takotsubo cardiomyopathy, is another rare but significant etiology of MI, as was the case in our first patient. (Oindi et al., 2019)

Risk factors for IHD in pregnancy include preeclampsia and other comorbidities like chronic hypertension, smoking, obesity and dyslipidemia, cocaine abuse, diabetes mellitus, etc.(Roth & Elkayam, 2008) Most patients with these risk factors will be asymptomatic before pregnancy, till the cardiac adaptations like increased intravascular volume, stroke volume and cardiac output increase the demand on the compromised coronary blood supply and lead to ischaemia and MI.

Thus, the highest fraction of PAMI is seen in intrapartum and immediate postpartum, as maternal cardiac adaptations are most pronounced in this time.(Kealey, 2010; Regitz-Zagrosek, 2017; Turitz & Friedman, 2014) Therefore, high risk patients need screening and periodic reassessment for possible cardiac event. Neither of our patients had any identifiable high-risk factors in antenatal period.

Uterotonics like methyl ergometrine and misoprostol may cause coronary vasospasm and precipitate MI in patients with high-risk factors like anaemia or advanced age. (Spencer & Lowe, 2019) This could have been an inciting factor in our first patient.

Diagnosis of PAMI is achieved by serial ECG showing ST segment elevations in specific chest leads with axis deviation, and elevated cardiac enzyme levels specifically Troponins, similar to non-pregnant state and can be supplemented by ECHO findings of wall motion abnormalities. Anterior wall is the most commonly involved part.(Roth & Elkayam, 2008) Both our patients had typical ECG and ECHO features of anterior wall STEMI.

However, certain ECG changes are seen in pregnancy without associated ECHO abnormalities, like mild cardiac axis deviation, small q wave in lead III, inversions of T wave and left ventricular stress patterns. Also Myoglobin and Creatine kinase-MB can be elevated with a contracting uterus.(Kealey, 2010) Troponin-I may be elevated in preeclamptic patients who may present with atypical chest pain.(Regitz-Zagrosek, 2017)

Our first patient had atypical chest pain during labour but ECG ECHO showed no changes. However, anaesthesia and

drugs administered intraoperatively might have caused a precipitous fall in cardiac blood supply and postoperatively led to MI, cardiac failure and pulmonary edema.

The treatment of PAMI involves resuscitation with inotropes, loading doses of anticoagulant(heparin), anti-platelets (aspirin), analgesics(morphine). The usage of statins and angiotensin-pathway drugs is contraindicated in pregnancy. (Regitz-Zagrosek, 2017; Roth & Elkayam, 2008) However, the cornerstone of management involves diagnostic angiogram and primary PCI. It is of utmost importance to note that 'Time is myocardium' as well as that mother and baby both need to be salvaged. Coronary angiogram may help to diagnose Coronary dissection as well. Metal expandable stents are deployed in both diagnoses. Thrombolysis is relatively contraindicated in pregnancy and its use is mainly in venous thromboembolism and thrombosed prosthetic heart valves.(Kealey, 2010)

In our first patient, once MI was diagnosed, we commenced medical therapy immediately with inotropic support, and then she underwent CAG which showed a normal anatomy without thrombosis or dissection. Our second patient received thrombolysis for intraventricular clot followed by maintenance therapy.

There is no consensus regarding mode of delivery in patients with MI. CS is usually reserved for obstetric indications unless it is a recent MI. Vaginal delivery with adequate epidural analgesia is generally well-tolerated and minimises stress. (Kealey, 2010). CS under controlled epidural anaesthesia or low dose spinal anaesthesia with high-risk consent has the advantage of being a planned and elective approach. In our first patient, we opted for CS due to obstetric indication of CPD and second patient underwent CS in view of recent MI.

CONCLUSION

Pregnancy-associated MI outcomes have improved in the last few decades due to advances in medicine, anaesthesia and cardiology; both our patients had a good outcome due to multidisciplinary care at our centre. Regular screening of cardiac status of elderly gravidas with risk factors should be protocolised. Early diagnosis and appropriate medical and interventional management have an important role to play in short term and long-term survival of these patients.

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