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Case Report

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Epidural anaesthesia a safe option for caesarean section in parturient with severe pulmonary hypertension - a case report

Abstract

Rheumatic heart disease is the commonest cardiac disease complicating pregnancy in developing countries. Heart disease accounts for 15% pregnancy related mortality. In the presence of maternal heart disease, the circulatory changes of pregnancy may result in exacerbation of the haemodynamic perturbations due to complex cardiac valvular lesions leading to decomposition or death of mother or foetus. Determining the ideal anesthetic technique for Caesarean section in presence of complex cardiac conditions remains a much debated topic. General anesthesia is associated with a further increase in pulmonary pressure in response to laryngoscope and intubation along with myocardial depression by anesthetic agents. Neuraxial blockade may lead to decrease in SVR and Cardiac output. We report the successful anesthetic management of a parturient suffering from Rheumatic heart disease with multi valvular lesions resulting in severe pulmonary hypertension under epidural anesthesia with good maternal and neonatal outcome. Successful management requires vigilant perioperative monitoring and thorough knowledge of the haemodynamic of complex cardiac valvular disease.

Keywords: rheumatic heart disease, multiple valvular lesions, severe pulmonary hypertension, caesarean section, epidural anaesthesia

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Introduction

Prevalence of heart disease in pregnancy varies from 0.3-3.5%. In the presence of maternal heart disease, the circulatory changes of pregnancy may result in decomposition or death of mother or foetus. Severe pulmonary hypertension is poorly tolerated in pregnancy. We shall discuss the anaesthetic management for elective Caesarean section of a young parturient suffering from Rheumatic heart disease with multi valvular involvement and severe pulmonary hypertension in a tertiary care teaching hospital.

Case history

A 20 yr old primigravida with history of rheumatic heart disease with multi valvular lesions and severe pulmonary hypertension was posted for elective Caesarean section at 36 wk of gestation. Preoperative examination revealed history of exceptional breathlessness and chest discomfort since 12 weeks of gestation which gradually occurred on less than ordinary activities with progression of pregnancy. There were no symptoms suggestive of paroxysmal nocturnal dyspnoea, syncope or congestive heart failure. Echocardiography revealed severe mitral regurgitation, moderate mitral stenosis (MVA 1.2sqcm, Pressure gradient 20mmHg), moderate aortic regurgitation and mild tricuspid regurgitation with severe pulmonary hypertension (PASP 75mm Hg, LVEF 52% with mild LV systolic dysfunction). ECG showed sinus rhythm with bifid P waves (Figure 1). Haematological and biochemical parameters were within normal limit. She was prescribed Tab Metoprolol 25mg twice daily, Tab Frusemide 20mg twice daily, PENTIDS 400mg twice daily, salt restriction, bed rest and was considered to be at a high cardiac risk for pregnancy.

In the operating room, standard ASA monitors were applied. Clinical examination revealed bilateral basal crepitations, tachypnoea, NIBP 113/60mmHg, MAP 73mmHg, HR 96/min, regular in rhythm, SPO2 100% in room air. Central venous cannulation was done through the Right Internal Jugular Vein for perioperative CVP monitoring. Baseline CVP was 7cm of H2O.

A graded epidural anaesthesia was planned to maintain haemodynamic stability and to maintain optimum SBP, DBP, HR, CVP and prevent further rise in pulmonary vascular resistance (PVR). Epidural catheter was inserted through L1-L2 inter space with patient seated and was placed 4 cm into the epidural space. Position was confirmed following administration of a test dose of 3 ml of 2% lignocaine with adrenaline. Patient was placed in supine position with a left tilt of 15 degrees to prevent aortocaval compression and a sensory block to T6 dermatome was achieved by 12ml of 0.5% Bupivacaine in fractionated doses of 3ml over a period of 20 minutes with 50 microgram Fentanyl. Oxygen was administered by a face mask at 6litre/min throughout the intra operative period. IV fluid infusion was guided by continuous monitoring of CVP. A male baby of 2.2 kg with Apgar score 9 and 10 at 1min and 5 min respectively was delivered. Following delivery of the baby 5 units of Oxytocin was administered intramuscularly followed by 5 units in 500 ml of lactated Ringer solution (RL) infused over one hour. Hypotension (BP 116/43mmHg, MAP 70mmHg) occurred after oxytocin infusion which was corrected by intermittent bolus of 50 microgram of Phenylephrine to a total of 300mcg (Figure 2). Total one litre of RL was infused maintaining a CVP of 5-7cm of H2O (Figure 3).

Duration of surgery was 60 minutes. Following surgery BP 112/58mmHg, MAP 73mmHg, PR 110/min, SpO2 100% in room air, CVP 5cm H2O was recorded. There was slight improvement of the symptoms with mild decrease in the basal crepitations. Patient was transferred to the critical care unit for observation for 48 hours. Analgesia was maintained with 0.125% Bupivavaine at 5ml/hr infusion and Paracetamol infusion (1gm in 100ml) 6 hourly. Patient was shifted to the ward after 48 hours. Post operative period was uneventful. Prescribed cardiac drugs were continued throughout the perioperative period. Patient was discharged from the hospital on

7th post operative day after obtaining cardiological consultation for further management of underlying cardiac disease.

Discussion

Pure or predominant Mitral stenosis occurs in approximately 40% of patients with rheumatic heart disease. The increased blood volume and cardiac output by 30-40% during pregnancy and a further increase in the cardiac output to 80%-100% of pre lab our values following delivery are poorly tolerated by parturient with valvular heart disease like mitral and aortic stenosis. A severe decompensation in myocardial function can develop during third trimester, labor, and immediately after delivery. Cardiac output, heart rate, and stroke volume decrease to pre-labour values by 24 to 72 hours postpartum and return to non-pregnant levels within 6 to 8 weeks after delivery.[1]

Severe pulmonary hypertension (PASP>75mmHg), MS with NYHA class II-IV, MR and AR with NYHA class III-IV predispose to a high maternal and fetal morbidity and mortality.[2]

Cardiac disease was diagnosed incidentally in this patient at a time when therapeutic termination was not feasible. Caesarean delivery was planned at 36 weeks gestation to maximize fetal lung maturation and to avoid deterioration in maternal cardiac status. Anticoagulants were not prescribed as there was no evidence of thrombi in the LA and ECG showed sinus rhythm.

The choice of anaesthetic technique in this case was a challenge as it involved a rare combination of multiple valvular lesions along with severe pulmonary hypertension. Few cases of complex valvular heart disease in the obstetric patient population have been reported. Besides, the haemodynamic perturbations due to complex lesions were further exacerbated during pregnancy. Anesthetic management was individualized keeping in view the haemodynamic goals relevant to the underlying complex pathophysiology.

Echocardiography findings suggested a normal sized LV with mild global hypokinesia and mild systolic dysfunction (LVEF 52%) and a dilated LA (LAID 4.7 cm) with MVA 1.2 sq cm along with a bifid P wave on ECG. This indicates that MS was the predominant lesion with coexisting MR and AR which had led to an enlarged LA with Pulmonary hypertension. Coexistent MR and AR causes severe volume overload of LV. MS and AR generate opposite loading conditions hence hyperdynamic contractility and LV enlargement may be less evident. Mitral stenosis restricts left ventricular filling and thus diminishing the impact of the aortic regurgitation on left ventricular volume.[3]

In this patient, symptoms were predominantly due to longstanding increase in LA volume and pulmonary hypertension along with a mild decrease in LV systolic function. Neuraxial blockade in form of graded epidural anesthesia that allows a gradual onset of block with avoidance of hypotension by intermittent fluid bolus and judicious use of vasopressors has been used successfully in the past.[4–8]

Subarachnoid block was avoided to prevent the sudden haemodynamic perturbations. Some authors have described the use of general anesthesia with good maternal outcome[9] whereas; others have reported increased pulmonary arterial pressure during laryngoscopy and tracheal intubation. Adverse effects of positive-pressure ventilation on venous return may lead to cardiac failure. Avoidance of tachycardia is of utmost importance which may be achieved by the use of opioids like Fentanyl before or during the induction of general anesthesia with neonatal respiratory depression as an adverse outcome.

Weeks & Smith[10] had concluded that although epidural anesthesia has been used with success, but in preexisting RHF large decrease in SVR may cause further decrease in CO. On the other hand GA may cause increase PVR in response to laryngoscopy, intubation and negative isotropic effects of anaesthetic agents. According to Gomar C and coworker,[11] with the exception of Tetralogy of Fallot, primary pulmonary hypertension, idiopathic hypertrophic subaortic stenosis, and anticoagulation, neuraxial techniques with low segmental blockade of dermatomes offer an alternative to general anesthesia in parturient with cardiac disease during Caesarean section. Adequate cardiovascular invasive monitoring is essential and should be administered and maintained in the postpartum period with the same criteria that reduce morbidity and mortality in cardiac patients undergoing general surgery.

Keeping the underlying pathophysiology in view, we aimed to maintain haemodynamic stability by maintaining an optimum SVR, preload, heart rate, sinus rhythm and avoiding myocardial depression and increase in pulmonary vascular resistance. This was achieved by epidural anesthesia in a graded manner using small fractionated doses of local anaesthetic to ensure a gradual onset of block and minimize haemodynamic changes resulting from sympathetic autonomic blockade. We chose to avoid general anesthesia in our patient in order to prevent the rise in pulmonary vascular resistance and worsening of pulmonary hypertension resulting from sympathetic stimulation during laryngoscopy, intubation and nitrous oxide inhalation and to prevent myocardial depression in response to anaesthetics. We administered oxytocin by intramuscular route and by slow intravenous infusion to avoid tachycardia and diastolic hypotension.[12] Agents that increase in PVR like methylergometrine, prostaglandin F2 alpha, hypothermia, hypoxia and inadequate analgesia were strictly avoided.[13]

SVR and CO increase after delivery which is a critical period for patients with Pulmonary HT and thereby require vigilant postoperative monitoring. Keeping this is view we had monitored the patient closely for 48 hours in the intensive care unit. Epidural anesthesia provides a safer alternative to general anaesthesia in parturient with complex valvulvar lesions. However, it is easier said than done. Successful management necessitates strict vigilance and an extremely cautious approach to maintain the haemodynamic stability throughout the peripartum period.

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Conflicts of interest

The authors declare there is no conflict of interests.

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