

Anaesthesia for Coarctation of Aorta and Aortic Valve Replacement in Pregnancy - A Challenge

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Abstract: Cardiac disease can be encountered during pregnancy with the frequency being 1% to 2% of all pregnant women. Because of the high foetal and relative maternal mortality during surgery, medical management is the first line of treatment. Nevertheless, when medical treatment fails, cardiac surgery becomes necessary. Since many of these deaths occur during or immediately following parturition, heart disease is of special importance to the anesthesiologist. This importance arises from the fact that drugs used for preventing or relieving pain during labor and delivery exert a major influence – for better or for worse – on the prognosis of the mother and newborn. Properly administered anesthesia and analgesia can contribute to the reduction of maternal and neonatal mortality and morbidity. We present the anaesthetic management of a case of Coarctation of aorta with Aortic valve disease in a pregnant woman undergoing surgery. Where patient underwent Coarctation of aorta repair during 20th week of gestation in the first sitting, Aortic valve replacement surgery in the second sitting 2 weeks after first surgery, followed by Elective cesarian section at term during 36 weeks of gestation.

Keywords: Open heart surgery, Pregnancy, Fetus, Repeat sternotomy, Carctation of aorta, aortic valve replacement, elective cesarian surgery

1. Introduction

In developing countries with a higher prevalence of rheumatic fever, cardiac disease may complicate as many as 5.9% of pregnancies [5] with a high incidence of maternal death. Since many of these deaths occur during or immediately following parturition, heart disease is of special importance to the anesthesiologist. This importance arises from the fact that drugs used for preventing or relieving pain during labor and delivery exert a major influence – for better or for worse – on the prognosis of the mother and newborn. Properly administered anesthesia and analgesia can contribute to the reduction of maternal and neonatal mortality and morbidity.

Cardiac pathology represents a wide spectrum of conditions, including congenital or acquired, functional or structural, cyanotic or acyanotic, and endocardial, myocardial, or pericardial defects. These cardiac lesions may be associated with few symptoms in the non pregnant state, but they become apparent for the first time in mid - to - late pregnancy as a result of the physiologic hemodynamic stresses that develop. Various structural cardiac lesions may be uncorrected, fully corrected, or partially corrected (palliated) when the pregnant patient is encountered by the anesthetist. Although general and regional anesthesia both affect the hemodynamic changes occurring during labor and delivery, the choice of technique is often immaterial if appropriate hemodynamic goals are considered and invasive monitoring is used to attain them.

Overall maternal and fetal morbidity and mortality from cardiac disease are directly related to the severity of cardiac disease. Major concern for a pregnant woman with a cardiac disease is cardiac decompensation because of the inability to meet the additional demands imposed by the physiologic changes of pregnancy and parturition. If present, infection, hemorrhage, and thrombo embolism compound the risk. It is essential to understand the impact of the physiologic changes of pregnancy upon the specific heart lesion to properly counsel and manage these patients. Pregnant women with heart disease should be managed by a team of representatives from obstetrics and perinatology, anesthesiology,

neonatology, cardiology, cardio - thoracic surgery, intensive care, nursing, and social work. [9]

Signs and Symptoms of Heart Disease in Pregnancy

Severe or progressive dyspnea, progressive orthopnea, paroxysmal nocturnal dyspnea, hemoptysis, exertional syncope, chest pain related to effort or emotion, and progressive or generalized edema indicate the presence of heart disease. Physical findings strongly suggestive of heart disease include cyanosis, clubbing, persistent neck vein distension, positive hepatojugular reflux, palpable thrill, diastolic murmurs, paradoxical splitting of cardiac sounds, true cardiomegaly, documented sustained dysrhythmias, and pulmonary hypertension.

Aims and Objectives

- Maintain mean blood pressure in normal limits.
- Maintain placental perfusion.
- Maintain normothermia.
- Monitor fetal heart rate.
- Use of tocolytic drugs.
- Multidisciplinary approach.

2. Case Presentation

A 23 year old primi - granida with 20 weeks of gestational age, with coarctation of aorta and aortic valve regurgitation presented with breathlessness and pedal edema. On physical examination was found to have unexpected hypertension during her routine ANC follow ups due to which cardiologist opinion was sought and after complete evaluation and 2D - Echo reports, she was diagnosed with coarctation of aorta along with aortic valve regurgitation. Thereby she was on regular follow up with her cardiologist.

In pre anesthetic evaluation, she was found to have NYHA class 3 dyspnoea. On auscultation medium pitched systolic blowing murmur was heard with clear and equal air entry both side. Patients vitals showed a pulse rate of 112/m, B. P of 162/86 mm of Hg in right upper limb, left lower limb 100/64 mm Hg, Respiratory Rate of 20/min.

Her routine Blood investigations were within normal limits. Her ECG showed sinus Rhythm with left axis deviation. 2D - Echo showed LVEF=60%, concentric LVH, bicuspid Aortic valve, coarctation of aorta distal to left, severe AR, mild AS, mild AR. CXR showed left ventricular enlargement. Patient was posted for Coarctation of aorta repair under ASA grade 3 High Risk in the first sitting and Aortic valve replacement surgery in the second sitting.

Patient was posted for Coarctation of Aorta repair during 20th week of gestation. In the operating room, ECG and Pulse Oximeter were connected. Radial Artery and femoral artery were cannulated for invasive Blood Pressure monitoring. Patient was pre oxygenated with 100% oxygen for 3 min and premedicated with injection Midazolam 0.25 mg i. v. Rapid sequence induction was performed with injection Fentanyl 100 mcg i. v, injection etomidate 6mg and injection Succinylcholine 100 mg i. v. Patient was intubated with a 7.0 mm size oral cuffed endotracheal tube and ventilated. Ventilator settings were adjusted to maintain normocarbida. Anaesthesia was maintained with oxygen/air mixture (50: 50), Isoflurane (0.8 - 1.0%) and Atracurium 30 mg i. v.

For fetal heart rate monitoring post CPB a stethoscope was attached on mothers abdomen. Temperature probe was inserted in nasopharynx to maintain normothermia. Tocolytic drugs were given at induction to prevent induction of early labour. Multidisciplinary approach was used were Gynaecologist, Paediatrician were kept stand by for emergency. Placental perfusion was maintained throughout the surgery by keeping mean blood pressure above 60mmHg. Minimum cardioplegia time and minimum CPB time was maintained.

2 weeks after Coarctation of aorta repair patient was posted for Aortic valve replacement surgery. Here along with anesthetic considerations in pregnancy repeat sternotomy factors were taken into consideration. Same protocols were followed. Blood (LD - PRC) and blood products (FFP and Platelets) were given after patient was off pump. Anti coagulants were started after valve replacement.

At term patient was posted for elective Caesarian section. Anti coagulation were managed with LMWH from 36 weeks of gestation. All the above protocols and considerations for pregnancy with heart disease were followed. Labour analgesia was given at term before induction of labour to avoid exacerbation of heart rate and blood pressure.

After delivery of baby, injection oxytocin 20 U i. v infusion was started and injection Carboprost 0.25 mg i. m was administered to achieve uterine contraction. Intraoperatively hemodynamics were monitored and BP remained stable throughout supplementing. Estimated blood loss was 350 ml and patient received 650 ml of crystalloid. Injection Furosemide 10 mg i. v was given intra operatively.

At the end of surgery, TAP block was given with Inj Bupivacanie 0.25%. She was extubated and shifted to ICU for hemodynamic monitoring. The new - born APGAR SCORE was of 8 and 10 at 1 and 5 min respectively. Patient was hemodynamically stable in the ICU and was shifted on the next day and the baby was breastfed.

Further course in the hospital was uneventful and the patient was discharged 5 days later and advised to follow up with the cardiologist.

3. Discussion

The incidence of maternal heart disease during pregnancy has been estimated to be 1.5% to 1% (1). Maximum pregnant women with heart ailments seen at referral centers are those with congenital heart disease. In India largest group includes women with rheumatic heart disease (2). Aortic stenosis is infrequently encountered during pregnancy and is usually due to a congenitally abnormal valve. Patients with mild aortic stenosis usually tolerate pregnancy well. Patients with moderate or severe aortic stenosis are very sensitive to preload changes and hypotension. They are unable to augment cardiac output (Co), with increase in LV systolic and filling pressures leading to heart failure and ischemia. Cardiac surgery during pregnancy has played a limited but defined role alongside the accepted medical management in the overall care of the patient. Maternal mortality associated with cardiac surgery varies from 1.5% to 4.2%, compared with the foetal mortality rate of 9.5% to 33% (4).

Case reports of fetal survival to term after corrective surgery performed in the second or third trimesters (4). The main goal in the management is to prevent further derangement of cardiac function during surgery and labour in a heart which is already stressed by the "physiological" changes of pregnancy. This can be accomplished by effective anxiolysis, analgesia and anaesthesia. Ultimately, the aim of any anaesthetic intervention is to ensure the well being of both the mother and the fetus. When cardiac surgery is performed during pregnancy, fetal mortality is 20 - 35%. It is the additional cardiac burden associated with pregnancy that often causes heart disease to show itself at this time.

When surgery is needed, timing is of key importance for the welfare of the fetus. During the first trimester, any injury to the fetus, whether due to drugs, hypoxia or changes in blood flow, has a high probability of causing congenital defects and spontaneous abortion. The third trimester is a time of low risk for the fetus, especially beyond 28 weeks, because if labour is precipitated by surgery, there is a good chance that the baby will survive in a neonatal unit, and the fetus is more resilient during cardiopulmonary bypass at this stage. However, by the third trimester, a gravid woman requiring surgery for a cardiac defect, will have a higher risk because of the extra cardiac output. In the second trimester the risk is similar to a non - pregnant woman, along with a lower risk for premature labour (5).

There is always a concern on the effects of anaesthetic agents on fetal development and teratogenicity, especially during the first trimester. It has been evidenced that most anaesthetic agents, intravenous, inhalatory, and paralyzing agents are devoid of teratogenic effects and can be safely employed in a pregnant patient (6). Drugs that are known to be safe, or do not cross the placenta, should be used. Vasoconstrictors are avoided due to the effect on the uterine spiral arteries.

Hypocarbica as a result of mechanical hyperventilation decreases the uterine blood flow by 25%, although the blood

pressure remains unchanged during hyperventilation. The adverse effect on uterine blood flow is attributed to a decrease in venous return and cardiac output (7, 8).

The dangers of CPB include changes in coagulation, alteration in the function of cellular and protein components of the blood, release of vasoactive substances from leukocytes complement activation, particulate and air embolism, non pulsatile flow, hypothermia and hypotension (7). All these factors can compromise the delicate biological equilibrium between the foetus and the placenta.

There are only few studies regarding the effects of maternal CPB on the foetus. Since the first report of the use of foetal heart recording during bypass by Koh and Co - workers⁹ in 1975, it has been known that foetal bradycardia occurs almost invariably at the onset of maternal CPB. What causes bradycardia at the beginning of the bypass is unknown, but it may be related to decreased foetal oxygenation secondary to placental hypotension or to acid base changes. The changes in foetoplacental perfusion during cardiopulmonary bypass are poorly understood, despite new methods for monitoring flow in the uterine artery, ductus venosus and foetal aorta. It is known that hypothermia can cause foetal hypoxia and that rewarming can likewise cause hypoxia by inducing uterine contractions. There are only a few reports of surgery with circulatory arrest and deep hypothermia, and in all of these the foetus died postoperatively. During cardiopulmonary bypass, haemodilution, lack of pulsatile flow, uterine arterial spasm and particulate microemboli may all alter placental perfusion and contribute to foetal hypoxia. With pulsatile perfusion during cardiopulmonary bypass, the hazard of vasoconstriction in placental vessels, including spiral arteries, is believed to be lessened by release of nitric oxide.

Thus; in pregnancy cardiopulmonary bypass is best conducted with mild hypothermia, pulsatile perfusion, high flow rates and minimal haemodilution. (10, 11)

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