

Anaesthetic management of a pregnant patient for Aortic valve replacement

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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. We present the anaesthetic management of a case of Aortic valve disease in a pregnant woman undergoing surgery.

Case Report

A 25-year-old woman gravida III para II had history of breathlessness since 3-4 years. She noticed shortness of breath and dyspnea on exertion at 20 weeks gestation. She also complained of dizziness and fainting one month back. On her antenatal checkup she was found to have a grade 3 systolic murmur. Echocardiography showed calcified aortic valve with ventricular movement, and mild symptomatic hypertrophied normal sized left ventricle. She was referred to the cardiothoracic surgeon who decided for aortic valve replacement. Grade III dyspnea was found on pre operative evaluation. Obstetrical examination revealed positive foetal viability. Poor foetal outcome was feared due to non pulsatile flow during cardio pulmonary bypass. Patient was prepared for the surgery. Aspiration prophylaxis along with antibiotics were given prior to the procedure.

During the surgery, routine monitors were applied, and a 14G cannula inserted in the right hand under local anaesthesia. Invasive arterial line using Seldinger's technique was also placed. Rapid sequence induction was done with injection fentanyl 7µg/kg body weight, thiopental sodium 3mg/kg body weight and rocuronium 0.9mg/kg body weight. Air way was secured by size 7.5mm endotracheal tube (ETT) which was confirmed by end tidal carbon dioxide and chest auscultation. Swan ganz catheter from right internal jugular vein for haemodynamics and fluid management was placed after induction along with extra 14G cannula in right hand.

Aortic valve replacement was done using cardio pulmonary bypass with non pulsatile blood flow using membrane oxygenator and blood cardioplegia. The patient was cooled to 30° centigrade during the procedure. Anaesthesia was maintained with propofol infusion at the

rate of 2mg/kg/hr. Mean arterial pressure was maintained between 50-60 mmHg during cardio pulmonary bypass with a pump flow of 2.5 liters/min. After completion of surgery patient was weaned off the cardio-pulmonary bypass. Patient remained haemodynamically stable. After surgery the obstetrician performed the foetal examination for viability, there was obvious heart sound which was confirmed by cardio tocho graph (CTG). The patient was shifted to cardiac intensive care unit. Extubation was done on the next day of surgery. Patient was shifted to the ward and discharged, on the 5th postoperative day.

At 37th week of pregnancy she was admitted again for elective Caesarean section, under general anaesthesia. A healthy baby boy weighing 2.9 kg with Apgar score of 8 and 9 was delivered.

Discussion

The incidence of maternal heart disease during pregnancy has been estimated to be 1.5%¹. Most of the pregnant women with heart ailments seen at referral centers are those with congenital heart disease. The next largest group includes women with rheumatic heart disease². 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.

There are now many reports of foetal survival to term after corrective surgery performed in the second or third trimesters⁴. The main goal in the management of these patients 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 foetus. When cardiac surgery is performed during pregnancy, foetal 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 foetus. During the first trimester, any injury to the foetus, 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 foetus, 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 foetus 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 labour⁵.

There is always a concern on the effects of anaesthetic agents on foetal 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 patient⁶. 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}

Conclusion

Cardiac surgery during pregnancy can be performed safely by using cardiopulmonary bypass. Maternal risks are related to the specific procedure performed. A high risk of foetal morbidity and mortality is associated with CPB during pregnancy. Foetal bradycardia and increased uterine contractions need immediate attention and management. With the optimal care provided by the modern neonatal care units and considering caesarian section as an option, the deleterious effects of CPB can be avoided when the gestational age is above 28 weeks.

References

1. Szekely P, Snaith L. Heart disease and pregnancy. Edinburgh: Churchill Livingstone 1974. pp 137.
2. Siu SC, Colman JM. Heart disease and pregnancy. Heart 2001. 85:710-15.
3. Abbas AE, Lester SJ, Connolly H. Pregnancy and the cardiovascular system; Int J Cardiol 2005; 98:179-89.
4. Bernal JM, Miralles PJ. Cardiac surgery with cardiopulmonary bypass during pregnancy. Obstet Gynecol Surg 1986. 41:1-6.
5. Agarwal RC, Bhattacharya PK, Bhattacharya L, Jain RK. Pregnancy and cardiopulmonary bypass. Indian J. Anaesth; 2004. 48:259-63
6. Duncan PG, Pope WD, Cohen MM, Greer N. Fetal risk of anaesthesia and surgery during pregnancy. Anesthesiology 1986; 64: 790-94.
7. Hammon JW Jr, Edmunds LH Jr. Extracorporeal circulation: Organ damage. Cohon LH, Edmunds LH Jr. Editors Cardiac surgery in the adult. McGraw-Hill, New York 2003; pp 361-388.
8. Levinson G, Shnider SM, DeLorimier AA, Steffenson JL. Effects of maternal hyperventilation on uterine blood flow and fetal oxygenation and acid base status. Anesthesiology 1974; 40: 340-47.
9. Koh KS, Friesen RM, Livingstone RA, Peddle LJ. Fetal monitoring during maternal cardiac surgery with cardiopulmonary bypass. Can Med Assoc J 1975; 112: 1102-04.
10. Parry AJ, Westaby S. Cardiopulmonary bypass during pregnancy. Ann Thorac Surg 1996; 61: 1865-59.
11. Kahler R. In : Medical complications during pregnancy, in: Burrow G and Ferris T, Eds. Cardiac Diseases Philadelphia Saunders, 1975; pp 105.