International Journal of Medical Anesthesiology

E-ISSN: 2664-3774
P-ISSN: 2664-3766
www.anesthesiologypaper.com
IJMA 2021; 4(3): 194-197
Received: 20-06-2021
Accepted: 23-07-2021

Dr. Nilesh Juvekar
Consultant Anaesthesiologist
Deenanath Mangeshkar Hospital and Research Centre, Pune, Maharashtra, India

Dr. Swapna Naik
Consultant Anaesthesiologist
Deenanath Mangeshkar Hospital and Research Centre, Pune, Maharashtra, India

Dr. Prachi Nisar
Consultant Anaesthesiologist
Deenanath Mangeshkar Hospital and Research Centre, Pune, Maharashtra, India

Dr. Nishant Rajadhyaksha
a) Senior Resident, Tata Memorial hospital and Research Centre, Mumbai, Maharashtra, India
b) Former, DNB resident at Deenanath Mangeshkar Hospital and Research Centre, Pune, Maharashtra, India

Corresponding Author:
Dr. Nilesh Juvekar
Consultant Anaesthesiologist
Deenanath Mangeshkar Hospital and Research Centre, Pune, Maharashtra, India

A case series discussing perioperative management of parturients with severe aortic stenosis for elective caesarean section

Dr. Nilesh Juvekar, Dr. Swapna Naik, Dr. Prachi Nisar and Dr. Nishant Rajadhyaksha

DOI: https://doi.org/10.33545/26643766.2021.v4.i3c.311

Abstract
Cardiovascular diseases are one of the leading causes of maternal morbidity and mortality. With the advancement in medicine and surgery, more and more women are reaching child-bearing age [1] Valvular heart disease (VHD) of rheumatic etiology remains a prevalent cause of maternal morbidity and mortality [2]. The severity of aortic stenosis (AS) decides the peripartum complications in parturients and the fetal outcomes. Mild to moderate AS is well tolerated during pregnancy but severe AS is associated with pulmonary edema, dysrhythmias and even death. Given the complexity of VHD in pregnancy, patient should be managed with multidisciplinary approach throughout pregnancy. We report two cases of AS with different patient profile needing adjustment.

Keywords: Aortic stenosis, peripartum complications, general anaesthesia, obstetric anaesthesia, maternal mortality and morbidity

Introduction
Cardiovascular disease has emerged as one of the leading causes of maternal morbidity and mortality. With advancements in medicine and surgery more women are reaching child bearing age and desiring pregnancy [1]. Valvular heart disease (VHD of rheumatic etiology remains a prevalent cause of maternal morbidity and mortality [2]. Mitral valve involvement of rheumatic etiology is the commonest cause while congenital bicuspid aortic valve (BAV) is the commonest etiology when aortic valve involvement is present in women of child bearing age group. Significant cardiovascular changes occur during normal pregnancy. The presence of a valvular lesion predisposes parturient to peripartum complications. Severity of AS decides the peripartum complications in parturient and the fetal outcomes. Mild to moderate AS is well tolerated but severe AS is associated with pulmonary edema, dysrhythmias and even death. Given the complexity of VHD in pregnancy, patients should be managed with multidisciplinary approach before and throughout pregnancy [3].

Case series
Case report – 1
A 34-year-old multigravida at 38 weeks gestation was referred to our pre-anaesthesia clinic for an elective lower segment cesarean section (LSCS). She had previously undergone LSCS for cephalo-pelvic disproportion 11 years ago during which she experienced breathlessness due to moderate AS with bicuspid aortic valves. Patient had no cardio-respiratory symptoms and hence, did not comply with any medications or routine follow-up at the cardiology clinic. Her present pregnancy was uneventful and fetal growth was normal. On examination, her pulse rate was 105/min regular, low volume and blood pressure (BP) was 116/68 mmHg in right brachial artery in supine position. An ejection systolic murmur (grade 4) was noted in the aortic area conducting to the carotids. Other systemic examination was normal. A two-dimensional echocardiography (2D echo) revealed bicuspid aortic valve with severe calcific AS. The aortic valve area was 0.6 cm^2, peak gradient of 153 mmHg, mean gradient of 105 mmHg and mild aortic regurgitation and moderate pulmonary hypertension. Taking cognizance of these findings, we categorised this patient as a high-risk obstetric case. A cardiologist, intensivist and cardiac anesthesiologist were involved and a peri-operative plan was carved out.

As patients with AS do not tolerate sudden reduction in afterload caused with central neuraxial blockade, general anaesthesia (GA) with invasive cardiovascular monitoring was planned. Procedure was scheduled in cardiac theatre and cardiac surgeon with his team were kept standby in case emergency aortic valve replacement was warranted. Patient and her family were counselled in detail about the risks involved (risk to life, need for emergency cardiac procedure, type of anaesthesia and its effect on newborn). Patient was kept fasting overnight, 18 G cannula was secured on right hand and Ringer lactate solution was started to prevent hypovolemia. Intravenous pantoprazole and metoclopramide was administered for aspiration prophylaxis. Standard monitors were attached in the operating room and IV Cefuroxime 1.5 gm was administered to achieve surgical prophylaxis. Her baseline pulse rate and BP were 110/min and 118/70 mmHg respectively. Left radial artery was cannulated and transduced after performing negative Allen's test under local anaesthesia. A 7F triple lumen catheter was inserted in right internal jugular vein (IJV) under local anaesthesia and transduced for central venous pressure monitoring. Nor-adrenaline infusion was connected to the central line anticipating its need following induction of GA. After taking the patient into confidence, surgical painting and draping was done prior to induction. Patient was induced with inj. Etomidate 6 mg slowly and intubated with 7.5 cuffed oral endotracheal tube (ETT) aided by Succinylcholine 100 mg following the principles of rapid sequence induction (RSI). Baby was delivered within two minutes of securing of ETT, after which inj. Atracurium 25 mg was given to provide muscle relaxation, fentanyl 125mcg was preferred to deepen the anaesthesia. Sevoflurane at a MAC of 0.7 was administered from the beginning of induction to ensure amnesia. Noradrenaline infusion was adjusted to maintain mean arterial pressure of 75 mmHg. Uterine retraction was achieved by a bolus of 5 units of IV oxytocin and an intravenous drip of Oxytocin with 15 units in 500ml of 5% dextrose over 2 hours. Episodes of abrupt fall in BP were corrected with 100 mcg bolus of phenylephrine.

Surgery was completed within 30 minutes and good hemostasis was achieved. Inj. Paracetamol 1 gm, Diclofenac sodium 75 mg IV was administered in addition to 20 ml of 0.75% Ropivacaine at surgical site. Muscle relaxation was reversed with neostigmine 2.5 mg and glycopyrolate 0.5 mg. The patient was extubated awake after ensuring adequacy of respiration. The patient was observed in cardiac postoperative ICU where noradrenaline infusion was gradually tapered off. Oxygen supplementation at 4 lit/min was given by nasal prongs. Patient was shifted out of ICU the next day and discharged on postoperative day (POD) 4. Both mother and fetus had uneventful outcomes.

Case report 2

A 32-year-old, full term, primi gravida was posted for elective LSCS. She had undergone aortic valve replacement (21 mm bioprosthetic) and tricuspid valvotomy 7 years ago. Echocardiography showed valve degeneration with severe AS, peak gradient of 130mmHg and mean gradient of 87 mmHg, aortic valve area of 0.5 sq cm, moderate tricuspid stenosis, severe tricuspid regurgitation and moderate pulmonary hypertension. Patient was euthyroid with thyronorm supplements and had history of mild covid 19 infection 5 months ago. Overall management principles followed in this case were similar to the first case. Infective endocarditis prophylaxis was given with Inj Ceftriaxone within an hour before surgery. After attaching routine monitors, 20 G arterial cannula inserted and transduced in right radial artery and 7 r triple lumen catheter secured in right IJV. Additional 16 G single lumen cannulae were inserted in right femoral artery and vein due to possible emergency institution of cardiopulmonary bypass (CPB). Patient was induced with etomidate 12 mg with inhalation sevoflurane to achieve mac of 0.7. Patient was intubated with 7.5 cuffed ETT aided by succinylcholine 100 mg iv following principles of RSI. After delivery of baby fentanyl 100 mcg was given to deepen anesthesia and Atracurium 20 mg was given to ensure muscle relaxation. Inj. Pitocin 5 units bolus and 15 mg infusion was given to assist uterine retraction. The patient remained stable throughout the procedure. Muscle relaxation was reversed with neostigmine 2.5 mg along with glycopyrolate 0.3 mg and patient was extubated awake. Patient was shifted to cardiac surgical intensive care unit for postoperative observation. After half hour patient developed atonic postpartum haemorrhage for which additional 30 units Pitocin, 600mg sublingual Misoprostil, 250 mcg intramuscular Carboprost and 1 gm Tranexamic acid was given. During this episode patient needed volume resuscitation and 2 units of packed cells. Once the uterus retracted, patient remained hemodynamically stable for the rest of the ICU stay. Invasive lines were removed after 48 hours before shifting patient out of the ICU. Patient and baby were discharged on POD 5.

Discussion

Congenital BAV is the most common congenital heart anomaly with a prevalence of 0.5-2%, a male preponderance and autosomal dominant pattern of inheritance [4, 5]. BAV can present as functionally normal aortic regurgitation or severe aortic stenosis in women of child bearing age group. AS is most commonly due to congenital BAV which may be associated with aortopathy or coartation of aorta [6]. The already compromised cardiac function worsens further due to physiological changes in pregnancy. Cardiac output increases by 30-50% due to increased stroke volume and to a lesser extent, due to increased heart rate during late stages of pregnancy. Cardiac output rises early in pregnancy and plateaus between the second and third trimester. Additionally systemic vascular resistance decreases by the end of second trimester and then slowly begins to increase until term [7]. Pregnancy is accompanied by physiologic anemia due to greater expansion in plasma volume. Together these changes lead to increased flow and thus increased gradients across pre-existing valvular lesion [8]. Aortocaval compression in late pregnancy reduces venous return which further complicates the situation. In AS, pregnancy related volume expansion overloads the pressure loaded left ventricle (LV). In pregnancy though mild and moderate AS are well tolerated when the LV ejection fraction is normal, severe AS can be complicated by heart failure in 10% and arrhythmias in 3-25% in these patients [9]. Adverse maternal and fetal outcomes increases with the severity of AS. Risk of hemodynamic compromise and heart failure is highest during second and third trimester, labour and delivery extending up to 24-72 hours after delivery as cardiac output peaks [10/11]. When pregnant women with severe AS develops major symptoms, case reports suggest

that balloon aortic valvuloplasty can reduce the risks of gestation, labour and delivery [12]. As our patient presented late, interventional procedure was not possible. In view of severity of AS, normal vaginal delivery was ruled out by obstetrician. There are 3 risk scores: ZAHARA risk score [14] CARPREG and WHO classification [15]. As per these scores, the possibility of mortality in our case ranged from 17.5-27%.

Patients with AS have a low fixed cardiac output. In case of sudden decrease in contractility or cardiac arrest, chest compressions will not maintain sufficient cardiac output. Thus, induction should focus on stable hemodynamics while achieving adequate depth of anesthesia. In patients with severe AS, GA should be preferred. The goals of anesthetic management include maintenance of a normal heart rate, sinus rhythm and adequate systemic vascular resistance; to maintain intravascular volume and venous return; avoidance of myocardial depression during GA and avoiding aorto-caval compression. Both bradycardia and tachycardia should be avoided. Bradycardia is undesirable as cardiac output may become unacceptably low in presence of fixed outlet orifice. Tachycardia should be avoided as it can further jeopardize the oxygen demand supply mismatch in presence of significant LVH [16]. Due to diastolic dysfunction and impaired relaxation of LV, the atrial contribution of sinus rhythm which accounts for nearly 40% of the total cardiac output. Any possible arrhythmia should be avoided. Preload should be maintained adequately to fill the noncompliant LV. Afterload should be maintained or increased. We planned invasive monitoring which enable us to act on any hypotension quickly. Patients were hydrated before induction with a balanced salt solution so as to avoid hypovolemia and maintain preload. A cardiovascular unit was kept standby to take her up for emergency aortic valve replacement in the event that the patient has a cardiac arrest in the perioperative period as cardiopulmonary resuscitation is ineffective in these patients. In case of an emergency, institution of CPB would have become challenging, specially with a re-do thoracic surgery. Hence, in the second case, a femoral arterial and venous access was secured to facilitate early initiation of CPB. Obstetric team counselled the patient and painted and draped her abdomen so as to minimize incision delivery time.

Anaesthesia was induced with Etomidate, an induction agent with the most cardio stable profile. A noradrenaline infusion was started at minimal dose to prevent any hypotension. RSI technique was used to secure airway with endotracheal tube. Due to quick delivery, babies cried immediately after birth and had normal APGAR scores. We refrained from giving opiods or sedatives at induction so as to prevent neonatal depression and maintained depth of anesthesia with iv agents and sevoflurane. Oxytocin when given as bolus can cause hypotension due to vasodilation [17]. Hence it should be given as an infusion in a fixed cardiac output state [18]. We gave 5 units Pitocin as a bolus because we were concerned about the uterine relaxation brought about by sevoflurane. We adjusted our end tidal mac to 0.7 so that, amnesia was ensured with minimum relaxation of uterus. Giving bolus of pitocin brought about excellent uterine contraction and minimized the blood loss. Episodes of relative hypotension were treated by phenylephrine rather than increasing rate of noradrenaline as we preferred mild bradycardia of phenylephrine to tachycardia brought about by noradrenaline. Both patients were electively shifted to cardiac surgical postoperative ICU for observation and any further emergency management if needed.

Conclusion

Pregnancy with severe AS is a challenge for anesthesiologist. One has to have a thorough knowledge of pathophysiology of AS associated with pregnancy. Multidisciplinary approach is mandatory with inputs from the cardiac surgical team. GA with invasive blood pressure monitoring and judicious vasopressor support will ensure an uneventful anaesthetic outcome. This case series demonstrates the importance of multidisciplinary preoperative assessment in such patients and careful anaesthetic planning to avoid the deterioration of perioperative cardiac performance in parturients with complex valvular heart disease.

References

1. Ntloudi D, Giannakoulus G, Parcharidou D, Panagiotidis T, Gatzoulis MA, Karvounis H. Adult congenital heart disease: a paradigm of epidemiological change. Int. J cardiol 2016;218:269-74.
2. Zuhlke L, Engel ME, Karthikeyan G *et al.* Characteristics, complications and gaps in evidence based interventions in rheumatic heart disease: the global rheumatic heart disease registry(the remedy study) eur heart J 2015;36:1115-229.
3. Nishimura RA, Otto CM, Bonow RO *et al.* Aha/ACC guidelines for the management of patients with valvular heart disease: executive summary: a report of the American college of cardiology/American heart association task force on practice guidelines. Circulation 2014;129:2440-92.
4. Fenoglio jr JJ, Mcallister HA, De castro CM. Congenital bicuspid aortic valve after age 20. American journal of cardiology 1977;39(2):164-169.
5. Ro Bonow BA, Carabello Chatterjee K *et al.* ACC/aha 2006 guidelines for management of patients of valvular heart disease; a report of the American college of cardiology/ American heart association task force on practice guidelines (writing comitte to revise the 1998 guidelines for the management of patients of valvular heart disease. Circulation 2006;114(5):e84-231.
6. Orwat S, Diller GP, Van Hagen IM *et al.* Risk of pregnancy in moderate and severe aortic stenosis: from the multinational ropac registry. J am coll cardiol 2016;68:1727-37.
7. Sanghavi M, Rutherford JD. Cardiovascular physiology of pregnancy. Circulation 2014;130:1003-8.
8. Samiei N, Amir Sardari M, Rezaei Y *et al.* Echocardiographic evaluation of hemohynamic changes in left sided heart valves in pregnant women with valvular heart disease. Am J cardiol 2016;118:1046-52.
9. Regitz – Zargosek V, Blomstrom Lundqvist C, Borghi C *et al.* ESC guidelines on management of cardiovascular diseases during pregnancy. Eur heart J 2011;32:3147-97.
10. Ouzounian JG, Elkayam U. Physiologic changes during normal pregnancy and delivery. cardiol clin 2012;30:317-29.
11. Robson SC, Dunlop W, Boys RJ, Hunter S. Cardiac output during labour. br med J (clin res ed) 1987;295:1169-72.

12. Vinotha *et al.* Baloon aortc valvuloplasty in pregnancy with severe aortic stenosis and infective endocarditis. Int J reprod contracept Obstet gynecol 2012;1(1):69-71.
13. Siu SC, Sermer M, Colman JM *et al.* Prospective multicentre study of pregnancy outcomes in women with heart disease. Circulation 2001;104:515-21.
14. Drenthen W, Boersma E, Balci A *et al.* Predictors of pregnancy complications in women with congenital heart disease. eur heart J 2010;31:2124-32.
15. European society of gynaecology (ESG), association of European paediatric cardiology (AEPC), German society for gender medicine *et al.* esc guidelines on management of cardiovascular diseases during pregnancy: the task force on management of cardiovascular diseases during pregnancy of the European society of cardiology (esc). eur heart J 2011;32:3147-97.
16. Christ M, Sharkova Y, Geldner G, Maisch B. Preoperative and perioperative care for patients with suspected or established aortic stenosis facing non-cardiac surgery. Chest 2005;128:2944-53.
17. Thomas JS, Koh SH, Cooper GM. Hemodynamic effects of oxytocin given as iv bolus or infusion on women undergoing caesarean section. British journal of anaesthesia 2007;98(1):116-119.
18. Leatherbarrow *et al.* Management of emergency caesarean section in a patient with decompensated critical aortic stenosis. J Obstet Anaesth crit care 2018;8:50-3.