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ANAESTHETIC MANAGEMENT OF A PATIENT WITH ISOLATED PULMONARY STENOSIS POSTED FOR CAESAREAN SECTION

Dr.karishma P	Junior Resident
Dr.atul B Vyas*	Professor*Corresponding Author
Dr.krupa Patel	Junior Resident
Dr.yogesh Patel	Junior Resident

ABSTRACT Cardio circulatory changes associated with pregnancy result in a significant haemodynamic burden and lead to morbidity and even mortality in women with cardiac disease. We report a rare case of severe pulmonary stenosis who underwent elective caesarean section under general anaesthesia with satisfactory maternal and neonatal outcome.

KEYWORDS : Caesarean section, general anaesthesia, pulmonary stenosis

INTRODUCTION

Pulmonary stenosis refers to a dynamic or fixed anatomic obstruction to flow from the right ventricle to the pulmonary arterial vasculature.[1] Although most commonly diagnosed and treated in the paediatric population, individuals with complex congenital heart disease and more severe forms of isolated pulmonary stenosis are surviving into adulthood and require ongoing assessment and cardiovascular care.[2,3] Considering the rarity, the optimal anaesthetic management is either controversial or inconclusive.

CASE REPORT

A30 year-old, 65-kg female, 2rd gravida with 38 weeks 3 days of pregnancy was admitted for safe confinement. She had history of breathlessness on mild exertion, otherwise well adjusted to normal course of life. There was no evidence of right heart failure.

On pre-anaesthetic examination, she was found to be comfortable lying on the bed with a heart rate of 83/min, baseline pressure 140/90 mmHg, respiratory rate 14/min and New York Heart Association class 1. On inspection no specific findings on auscultation, chest was clear with normal S1 and a widely split S2, with a soft and delayed P2 and a systolic crescendo–decrescendo ejection murmur in the left upper sternal border.

Her investigations revealed Hb of 12.2 g% and serum creatinine 0.67 mg. The electrocardiogram (ECG) showed sinus rhythm with right axis deviation, a dominant R wave in aVR and T wave inversion in II,III,aVF. An echocardiography demonstrated severe valvular pulmonary stenosis gradient of 64 mmHg, right atrium and right ventricle dilated, trivial MR,AR,TR, no Patent Ductus Arteriosus and no coarctation of aorta. Plain chest radiograph showed prominence of pulmonary artery, with right atrial and ventricular dilatation.

She received prophylactic antibiotics, ranitidine, ondansetron and metoclopramide intravenously (IV) in the pre-induction room. In the operating room, routine monitors (ECG, pulse oximeter) were attached. A 15° wedge was given for left uterine displacement. She was premedicated with glycopyrrolate 0.004 mg/kg body weight IV and pre-oxygenated with 100% O2 for 3 min. Lignocaine 2% 1.5 mg/kg body weight IV was administered.

Rapid sequence induction was performed with etomidate 0.2mg/kg body weight IV and succinylcholine 1.5 mg/kg body weight IV. The patient was intubated with a 7.0mm-sized oral cuffed endotracheal tube and ventilated on volume control mode with a tidal volume of 8 mL/kg. Anaesthesia was maintained with oxygen/air mixture at 50:50, and vecuronium 0.08mg/kg body weight IV loading dose given, after that TIVA started ; inj propofol 2 mg / ml infusion at 40-60 ml / hr i.v . After delivery of the baby, syntocinon 20 units in infusion was started . Inj Methergine given iv 0.2 mg . Fentanyl 1 µg/kg body weight IV. She was reversed with glycopyrrolate 0.01 mg/kg body weight IV and neostigmine 0.05 mg/kg body weight IV and extubated after thorough suctioning. She was shifted to the post-anesthesia care unit and observed for 3 h. Intra-operatively, a total of 1.5 L of crystalloids was infused. Urine output at the end of surgery was 300 mL. The estimated blood loss was 500 mL. Pulse rate ranged between 80 and 100/min and blood pressure between 120/80 and 140/90 mmHg.

A male baby weighing 2.6 kg was delivered with APGAR score of 6 and 10 at 1 and 5 min. Post-operatively, she was continued on supplemental oxygen at 4 L/min through a venti-mask. Dextrose Normal Saline was started at a rate of 70 mL/h.Her further course in the hospital was uneventful.

DISCUSSION

Pulmonary stenosis is a common form of congenital heart disease that occasionally is diagnosed for the first time in adulthood. Isolated valvular pulmonary stenosis comprises approximately 10% of all congenital heart diseases.[4] Many of these patients remain asymptomatic to adult life.[5,6] Except for critical stenosis in neonates, survival is the rule in congenital pulmonary stenosis. A slight female predominance exists.[1]

Anaesthetic management of patients with severe pulmonary stenosis requires understanding of its physiological adaptations and also the events and drugs that can alter the magnitude of right ventricular outflow.

Pulmonary stenosis increases right ventricular work and dramatically impairs left ventricular output. It is important to maintain right ventricular filling pressure to optimize myocardial contractility. Also, excessive fluid can precipitate acute right heart failure and atrial arrhythmias.[7] During pregnancy, preload may decrease due to aortocaval compression, also by reduced forward flow, which can be further precipitated by neuraxial sympathetic blockade. Hence we avoided the use of neuraxial anesthesia in this patient.

The goals of haemodynamic management are maintenance of right ventricular preload, left ventricular afterload and right ventricular contractility[7] and also avoiding further increase in pulmonary vascular resistance. The goals of anaesthetic management are to reduce pulmonary vascular resistance and systemic vascular resistance. In general, hypothermia, hypercarbia, acidosis, hypoxia and high ventilator pressures should be avoided.[7] Our parturient did not have any signs of right heart failure and no other associated medical illness. The goals of anaesthetic management were met by giving intravenous induction drugs in titrated doses and slowly. Invasive monitoring could be avoided.

The cardiovascular changes associated with labour and vaginal delivery, including a further increase in cardiac output and oxygen consumption and Valsalva manoeuvres, were thought to be too hazardous to attempt induction of labour. Considering these factors, general anaesthesia was chosen. Both the inhalational and the narcotic-based induction were avoided in view of slow induction, neonatal depression and maternal myocardial depression. Nitrous oxide may increase pulmonary vascular resistance, hence it was avoided. Inhalational anaesthetics was also not used. Anaesthesia was maintained with TIVA. Response to induction was decreased by giving xylocard 2% 1.5 mg/kg body weight IV. Systemic vascular

resistance was kept low by propofol infusion.

CONCLUSION

Pulmonary stenosis can worsen during labour and delivery, resulting in high maternal mortality. Spinal anaesthesia may be associated with an uncontrolled reduction in right ventricular preload and should therefore be avoided in severe cases. Epidural anaesthesia again can result in episodes of hypotension, which is not acceptable. General anaesthesia offers a safe approach for fluid management. Although no conclusions can be drawn from this single case report, it is demonstrated that in selected cases, caesarean section can be performed safely under general anaesthesia.

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REFERENCES

1. Xiushui Ren (Mike), Cannistra Lauralyn B. Clinical Cardiology Fellow, Division of Cardiology, Kanbar Cardiac Center, California Pacific Medical Center, eMedicine specialities. Cardiology, Valvular Heart Disease. 2009 Jul 15;
2. Elkayam U, Gleicher N. Hemodynamics and cardiac function during normal pregnancy and the puerperium. In: Elkayam U, Gleicher N, editors. Cardiac problems in pregnancy. 3rd ed. New York: Wiley-Liss; 1998. pp. 3–20.
3. Siu SC, Sermer M, Colman JM, Alvarez AN, Mercier LA, Morton BC, et al. Prospective multicenter study of pregnancy outcomes in women with heart disease. Circulation. 2001;104:515–21.
4. Brickner ME, Hillis LD, Lange RA. Congenital heart disease in adults.First of two parts. N Engl J Med. 2000;342:256–63.
5. Hayes CJ, Gersony WM, Driscoll DJ, Keane JF, Kidd L, O'Fallon WM, et al. Second natural history study of congenital heart defects.Results of treatment of patients with pulmonary valvar stenosis. Circulation. 1993;87(2 Suppl):I28–37.
6. Perloff JK. Congenital heart disease and pregnancy. Clin Cardiol. 1994;17:579–87.
7. Joubert IA, Dyer RA. Anaesthesia for the pregnant patient with acquired valvular heart disease. Anaesthesia. 2005;19:1–2.