Case Report

Anaesthetic management of a child for surgical ligation of patent ductusarteriosus with severe valvular aortic stenosis with ventricular septal defect

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Abstract

Patent ductusarteriosus is one of the most common congenital heart disease[1]and is often associated with other cardiac defects e.g. ventricular septaldefect, Tetrology of Fallot, Transposition of Great Vessels. An isolated PDA is usually corrected by either surgical interruption or coil embolisation in cath lab. PDA with severe valvular stenosis and VSD is very rare. A review of literature revealed very few articles stating information on anaesthetic management of a large PDA with severe aortic stenosis with VSD.

Kewords: Patent Ductus Arteriosus, Ventricular Septal Defect, Aortic Stenosis.

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CASE REPORT

The patient, 2 yr old female child weighing 5.3 kg presented for surgical ligation of PDA. The heart disease was detected at the age of 3 months of age. Patient had symptoms of recurrent upper respiratory tract infection and failure to thrive. No history of infective endocarditis or hospitalization. Developmental milestones were delayed. Patient could stand with support and speak few words. Hearing and vision was normal. On general examination, patient was small for age. Cardiovascular examination revealed a continuous murmur heard at left side and ejection systolic murmur grade 4/6 in aortic area radiating to carotid. Investigations were within normal range. Chest X- ray had cardiomegaly more suggestive of volume overload of VSD and not typical of Left ventricular hypertrophy as of severe aortic stenosis.

ECHO REPORT

Congenital heart disease with large PDA of8 mm with left to right shunt of gradient 10mm of Hg. Bicuspid aortic valve with severe valvular aortic stenosis with gradient of 82/44 mm of Hg. Aortic valve annulus 12 mm. Small restrictive perimembranous VSD with left to right shunt with gradient of 60 mm of Hg. Dilated left atrium and left ventricle. Good biventricular function. Severe pulmonary hypertension. On the day of surgery, patient was premedicated with oral atropine 0.4 mg and Syruppedichloryl 2 ml. Anaesthesia was induced with Inj.ketamine 2mg/kg IV,Inj.fentanyl 4mic /kg IV, and Inj.vecuronium0.1mg/kg IV to facilitate endotracheal intubation with plain 4 no. portex endotracheal tube. During this period patient was ventilated with oxygen and sevoflurane. Further Anesthesia was maintained with sevoflurane,Inj.fentanyl0.5-0.1mic/kg and Inj.atracurium 0.25mg/kg .Nitrous oxide was not used. After induction and intubation, femoral artery and femoral vein were cannulated to monitor arterial blood pressure and central venous pressurerespectively. In addition, patient was monitored with American Standard of Anesthesianon invasive monitoring as ECG, Pulseoxymetry, capnography. Temperature monitoring, urine output, airway pressure and tidal volume were also monitored. Haemodyanamic parameters of patient after induction were- heart rate -112/min, BP- 94/38 mm of Hg,CVP- 9. After ensuring air entry on both sides of the chest in the right lateral position, the PDA was approached via a leftposterolateral thoracotomy incision and the pleural cavity was entered through the fourth intercostal space. Retraction of the lung was cautiously minimized. As

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patient had severe aortic stenosis with VSD with Left ventricular dilatation, pressure did not reduce to 50-60 mm of Hg. Pressure was maintained at systolic 80 mm of Hg. Test clamp was applied and rise in systolic and diastolic pressure noted. Initially saturation dropped for seconds but picked up gradually.PDA ligated with same systemic pressure of approximately 80 mm of Hg successfully. Haemostasis was achieved and thoracotomy closure done. 0.1%sensorcaine 5mg in intercostal block given .Multimodal analgesia in the form of Inj.paracetamol 7mg/kg IV, Inj.ketorolac 2mg /kg BD IV, Diclofenac suppository 2mg/kg.local infiltration of sensorcaine 0.1%2.5 ml at skin suture and Inj.fentanyl 0.5mic/kg/hr infusion post extubation postop for 24 hrs. Adequate analgesia was ensured to avoid tachycardia and pulmonary hypertension crisis. Extubation done at end of procedure after patient fulfilled extubation criteria with reversal of nondepolarising muscle relaxant with Inj.neostigmine 0.25 mg and Inj.glycopyrollate 0.05mg.

DISCUSSION

Patent ductusarteriosus is a persistent fetal communication from main pulmonary artery to the descending aorta . The incidence of this defect is 1 in 2500 live full term birth and 10 % of all congenital heart defects[1]. The shunt between the aorta and pulmonary artery can be restrictive or non restrictive .The magnitude and direction of flow are determined by the pressure in the aorta and resistance to the flow in two vascular beds – pulmonary and systemic. With non restrictive PDA, the normal low pulmonary vascular resistance leads to an increased pulmonary blood flow and rise in pulmonary vascular pressure while systemic blood flow is frequently reduced[1,2].Addition of severe aortic stenosis will compromise systemic flow more. Our case for PDA ligation is very rare with combination of PDA and severe aortic stenosis with VSD[3,4] Such combinations have been mentionedfor noncardiac surgery in literature[5,6] Catheter closure of PDA and subsequent valvotomy can be possible routinely[7] But in our case, in view of severe pulmonary hypertension, secondary to large PDA and VSD, cardiologist advised surgical closure of PDA first. Because of hardware unavailability, cost issues and nonsuitability for devise surgical closure was done. Surgery can have complications in such sick patient undergoing general anesthesia because they are prone to have cardiac failure secondary to severe pulmonary hypertension and severe aortic stenosis as well. Conventionally in PDA ligation, hypotensive anaesthesia should be given at the step of ligation to avoid tear of PDA tissue and torrential bleeding after tear[8]. In our case, as patient had severe aortic stenosis; induced hypotension can deteriorate myocardial perfusion leading to myocardial dysfunction. Because in aortic stenosis, left ventricular mass is hypertrophied and have highoxygen demand and require high perfusion pressure[3,9] Secondly as aortic stenosis is fixed cardiac output condition forward systemic flow also can deteriorate at low blood pressure. So we did not lower systolic pressure less than 80 mm Hg without any complications. Major implications caused by severe pulmonary artery hypertension and aortic stenosis in our case made us to keep heart rate and BP within normal limit for age and weight. Optimum fluid management in the form of IV Ringer Lactate and 25% dextrose and adequate analgesia given. Analgesia is very important for respiratory mechanics and avoid irritability and tachycardia in all paediatric patients and special population like aortic stenosis in our case. Post operatively patient remained stable. Calculated fluid management ,electrolyte management ,urine output, BP and CVP monitoring done. Next day orally feed started, patient delined, drain removed and mobilized. On third post operative day patient was shifted to ward uneventfully.

Post op ECHO: No residual PDA. Moderate aortic stenosis with gradient of 40 mm of Hg, good biventricular function. Moderate pulmonary hypertension. Patient was discharged on 5thpost operativeday. Advised follow up after 1 ½ months for further management.

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