Anaesthetic Management of Pulmonary Stenosis Already Treated with Pulmonary Balloon Valvuloplasty

SUSHMA K.S.¹, SAFIYA SHAIKH²

ABSTRACT

Anaesthesia Section

The presence of congenital valvular heart disease can pose a challenge to anaesthetists in paediatric patients planning for incidental surgeries. Most of the available case reports from various journals describe the anaesthetic management in adults, whereas, pulmonary stenosis can present anytime during childhood and adolescence. The present case-report highlights anaesthetic management in a 9-year-old child, who was diagnosed with severe pulmonary stenosis that was already treated with pulmonary balloon valvuloplasty, yet had an eventful perioperative period due to residual valvular dysfunction.

Keywords: Pulmonary stenosis, Pulmonary balloon valvuloplasty, Congenital valvular heart desease

CASE REPORT

A nine-year-old patient weighing 20 kgs was posted for closed reduction of fracture tibia with K-wire fixation and secondary suturing of wound in right arm. Her records revealed that she had undergone pulmonary balloon valvuloplasty two weeks before she met with an accident causing present injuries.

Her diagnosis of pulmonary stenosis was incidental during the health-checkup camp held at her school. Even though patient's mother gave history of easy fatiguability, she was not evaluated previously. She was referred to a cardiac center, where she was investigated and diagnosed with 'Isolated congenital pulmonary stenosis'. Her echo findings were found stenotic, dooming annulus (16 mm) with severe valvular pulmonary stenosis with pulmonary pressure gradient of 80 mmhg. She underwent percutaneous pulmonary balloon valvuloplasty and was put on oral beta-blocker tab propranalol 2.5 mg twice daily.

On admission to our hospital, two weeks had passed since pulmonary balloon valvuloplasty. Our pre-anaesthetic evaluation revealed a New York Heart Association (NYHA) class 2 patient with pulse rate of 100bpm, blood pressure of 120/70mm hg and respiratory rate of 20pm. On inspection, there was a precordial bulge and auscultation revealed delayed pulmonic component of second heart sound and long systolic murmur in the neo aortic area, radiating to all other areas including back. Her electrocardiogram revealed right axis deviation and right ventricular hypertrophy. Her echocardiogram showed mild pulmonary stenosis with pulmonary pressure gradient of 38 mm hg, with normal biventricular function. Jugular venous pressure was not raised and haematological profile was normal.

On the day of surgery, morning dose of tab propranalol was continued. She received Ranitidine 25 mg and Ondansetron 2mg intravenously (i.v) in the pre-induction room. In the operating room, routine monitors like electrocardiogram, non invasive blood pressure and pulse- oximeter were connected. Inj. Glycopyrrolate 0.1mg, inj. Midazolam 1mg and inj. Fentanyl 50mcg were given intravenously. Pre-oxygenated with 100% oxygen for 3 min, inj. Lignocaine 40mg i.v. given 90 seconds before intubation to counteract pressor response. Induced with inj. Propofol 40mg i.v., given in slow titrated doses. Intubated with 4.5 uncuffed endotracheal tube after three minutes of inj. Vecuronium i.v. and maintained with nitrous oxide and oxygen in ratio of 50:50.

As there was tachycardia post-intubation, Fentanyl 25 mcg was

repeated and Propofol infusion started at the rate of 3mg/min (150mcg/kg). Closed reduction could not be achieved, open reduction and K-wire fixation was done and procedure lasted 90 minutes. Vital signs were maintained throughout the procedure. Total fluid transfused intraoperatively was 400ml of ringer's lactate. At the end of procedure, Paracetamol suppository 500mg was placed. She was reversed with inj. Neostigmine 1 mg and Glycopyrrolate 0.2 mg i.v. and extubated after patient gained adequate respiratory efforts. The patient was conscious & comfortable with vital signs stable. The patient was shifted to post-operative ward.

About 10 hours after the procedure, patient developed tachycardia (120bpm), tachypnoea (40pm) and desaturation (87-88 %) on room air. On auscaltation bilateral basal crepitations were revealed. She was shifted to paediatric Intensive Care Unit (ICU). She was treated with inj. Digoxin 0.25mg slow i.v., inj Frusemide 20mg i.v. and Dobutamine infusion started at the rate of 100mcg/min. Oxygen supplementation was continued via facemask. Inj.digoxin 0.125mg and inj. Frusemide 20mg were repeated at 6 hours intervals. Patient improved after 10 hours of treatment with respiratory rate of 20/ min and SpO2 of 98-99% with fio 2 of 0.4. Echocardiography after recovery, showed pulmonary pressure gradient of 33mm of hg with normal biventricular function. Tab Digoxin 0.25mg ½ bd, Syp Potassium chloride 15ml bd and Tab Enalapril 5mg 1/4 bd were started and continued for two weeks. The patient was discharged after two weeks with strict instructions to have regular follow-up with cardiologist.

DISCUSSION

Right-sided valvular disease has received less attention from clinicians in part because of a protracted latent asymptomatic period [1].

The pulmonary stenosis is used to describe the lesions that collectively are associated with obstruction to the right ventricular outflow tract. A stenosis may be valvular, subvalvular, supravalvular or in pulmonary arterial tree.

Isolated pulmonary stenosis comprises approximately about 10% of all congenital heart diseases [2]. Pulmonary stenosis can be detected clinically at different stages of life. The more severe the obstruction, earlier the valvular abnormality is detected. Long term asymptomatic survival is common, with the exception of neonates with critical stenosis [3].

Most patients with mild to moderate pulmonary stenosis are

asymptomatic. Severe PS may cause exertional light-headedness or syncope and eventually right heart failure [1]. Grading of severity of the hemodynamic consequences of pulmonary stenosis, based on peak systolic pressure gradient, Trivial < 25 mm of hg, Mild 25-49 mm of hg, Moderate 50-79 mm of hg, Severe >80 mm of hg [4]. Echo and Doppler flow studies help to determine the site of obstruction and severity of stenosis. Relief of pulmonary stenosis is recommended for all patients with a peak gradient >/= 50 mm of hg and percutaneous pulmonary balloon valvuloplasty is the treatment of choice for severe or symptomatic pulmonary stenosis. The procedure carries a low risk of morbidity, mortality and restenosis. Those patients who have poor results with PBV, unfavourable valve morphology, fixed supravalvular or subvalvular stenosis, surgical valvotomy or valve replacement may be undertaken [1].

Long-term outcome of patients who have undergone balloon valvuloplasty is found to be excellent and the significant decline in the pressure gradient during first year of valvuloplasty is well recognised [5-8].

The present case-patient had severe pulmonary stenosis (peak gradient >80mm of hg) before PBV. Although, post valvuloplasty, before being taken up for orthopaedic surgery, her peak gradient was 30 mm hg which could categorize her into mild stenosis. Even though the pressure gradient reduces soon after valvuloplasty, exact duration required for optimal functioning is not clear. Pulmonary stenosis is often associated with some amount of subvalvular muscular obstruction secondary to hypertrophy of right ventricular myocardium.

Post-valvuloplasty, infundibular obstruction may worsen transiently but then regress over months [1,6]. The goals of hemodynamic management are maintenance of right ventricular preload, left ventricular afterload and right ventricular contractility. A low normal heart rate is preferable. Pulmonary stenosis increases the intraventricular pressure and work of the right ventricle [5].

Preload has to be maintained to optimize myocardial contractility; however excessive preload can precipitate right heart failure. In case of cardiac arrest, these patients are extremely difficult to resuscitate as cardiac compressions are not effective in forcing blood across the stenotic pulmonary valve [5]. Cardiac dysrhythmias, if develop, should be rapidly treated with Lidocaine, Propranalol or Esmolol and defibrillator to be readily available [5,7].

We did not attempt to insert a central venous catheter as surgery was planned to be a short procedure with no major fluid loss or shifts. Right-sided filling pressures would not have reflected leftsided filling pressures in these patients. General anaesthesia helped us in better control of airway, achieving hemodynamic stability & fluid management. In present case, main reason leading to heart failure post-operatively could have been infundibular obstruction because of hypertrophied myocardium as only two weeks had passed since balloon valvuloplasty.

CONCLUSION

As pulmonary stenosis can be a progressive condition and manifest any time till adulthood, such patients presenting for incidental surgeries can pose a challenge to an anaesthetist. They require careful preoperative, multi-disciplinary assessment and meticulous planning before surgery.

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PARTICULARS OF CONTRIBUTORS:

Assistant Professor, Department of Anaesthesiology, KIMS, Hubli, Karnataka, India.
Professor and HOD, Department of Anaesthesiology, KIMS, Hubli, Karnataka, India.
NAME, ADDRESS, E-MAIL ID OF THE CORRESPONDING AUTHOR:

Dr. Sushma K.S.

206, Department of Anaesthesiology, Karnataka Institute of Medical Sciences, Hubli, Dharwar, Karnataka-580021, India.

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Phone:. 9844310403, E-mail id:dr.sushsam@gmail.com
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