



Urgent aortic valve replacement in severe aortic stenosis with severe left ventricular dysfunction and severe pulmonary hypertension: a perioperative multidisciplinary management approach

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ABSTRACT

Severe aortic stenosis (AS) with reduced left ventricular systolic function and pulmonary artery hypertension (PH) is associated with poor outcome if remained untreated. We report a case report of a 62 years old male patient weighing 69 kg had progressive dyspnea for 5 years and was diagnosed cardiac patient, and was scheduled for an urgent aortic valve replacement. He had severely reduced left ventricular (LV) function and severe pulmonary hypertension. The patient was put on bypass with special emphasis on myocardial protection. Tissue valve was placed and patient was successfully put off cardiopulmonary bypass on high inotrope score, which was tapered after some time. The patient was shifted to CICU after chest closure and was extubated on fast track mode. The patient was followed up for three months showing improvement in symptoms and LV function.

The objective of reporting the case is to highlight the role of multidisciplinary integrated approach in the perioperative period for best patient outcome.

Key words: Severe Aortic stenosis; Aortic valve replacement; Pulmonary Hypertension

Citation: Akhtar MI, Butt S, Shahabuddin S, Hamid M. Urgent aortic valve replacement in severe aortic stenosis with severe left ventricular dysfunction and severe pulmonary hypertension: a perioperative multidisciplinary management approach. *Anaesth Pain & Intensive Care* 2017;21(3):366-369

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Received : 22 Aug 2017

Reviewed : 24 Aug 2017

Corrected : 28 Aug 2017

Accepted – 15 Sep 2017

INTRODUCTION

Severe aortic stenosis (AS) with reduced left ventricular systolic function and pulmonary artery hypertension (PH) is associated with poor outcome if remain untreated.¹ In severe but symptomatic AS, risk of sudden death is 1%. Eventually, symptoms of angina, exertional syncope, or heart failure occur and the prognosis becomes poor with average survival being 2 years, a 50% incidence of sudden death and a monthly mortality of 2%.²

Aortic valve replacement is required to modify the natural history; however, in the presence of severe

pulmonary hypertension it is associated with an increased mortality.^{3,4} Perioperative management of these patients is always a great challenge for the anesthesiologist especially at the time of induction of anesthesia. We report a case in which the aortic valve area was severely reduced to 0.3 cm² with severely reduced LV function and severe pulmonary hypertension to highlight the role of multidisciplinary approach in the perioperative period for the best patient outcome.

CASE REPORT

A 62 years old male patient, 69 kg and height 172

cm, had progressive dyspnea for the last five years. Initially dyspnea came on moderate exertion but for the last few months, it appeared even on mild exertion (NYHA–III) and was associated with diaphoresis and palpitations. Occasionally he had had orthopnea too. He was admitted in a peripheral hospital and was diagnosed as a case of severe AS and referred to our hospital. He was scheduled for an urgent aortic valve replacement.

The patient underwent a thorough preoperative assessment and optimization. ECG showed normal sinus rhythm with RBBB. He had deranged LFTS with an increased SGPT (668 units/L). He also required upper and lower GI endoscopies for anemia. Chest x-rays were suggestive of bilateral effusion that was drained.

Echo report showed aortic valve area of 0.3 cm², LV EF 25%, severe HT and mild RV dysfunction. Aortic valve area by PISA was 0.36 cm², peak pressure gradient of 110 mmHg and mean pressure gradient of 75 mmHg. Estimated pulmonary artery systolic pressure was 60 mmHg.

Considering his advanced age and need for the aortic valve replacement, surgical disease in the coronary arteries was ruled out by cardiac catheterization prior to surgery.

Pre-induction invasive monitoring lines were inserted under local anesthesia after counseling the patient with USG guidance in addition to the routine pre-induction radial arterial line. Defibrillating combo pads were placed proactively to manage lethal ventricular arrhythmias, as CPR is usually ineffective in severe AS in cases of cardiac arrest.

Cardiac surgeon and the perfusionist in their capacity were vigilant at induction to manage hemodynamic compromise at induction. The patient was induced and intubated with fentanyl 250 µg, etomidate 12 mg and atracurium 40 mg with vigilant hemodynamic monitoring by all team members.

TOE probe was inserted after induction to monitor heart function continuously. BIS monitoring was done to monitor depth of anesthesia and to prevent awareness. BIS was kept at 40-60. Patient's systemic pressures remained stable despite high PA pressures.

Pulmonary artery catheter (PAC) was inserted after pericardial opening to avoid any lethal arrhythmias. Baseline PA pressures were 81/38 with systemic pressures of 99/65 and CVP was 17.

Chest was opened and the patient was put on bypass. MAP was kept around 70 mmHg during bypass with maintenance of adequate urine output and

other necessary metabolic parameters. Temperature was dropped down to 32° C. Cardioplegic solution was delivered initially through aortic root to arrest the heart and then after opening the aortic root, it was delivered through right and left coronary ostia. Aortic root opened and on inspection the valve was heavily calcified with bicuspid morphology. Tissue valve was inserted after opening the aortic root. Total bypass time and cross clamp time were 130 min and 100 min respectively.

At rewarming, the patient was given 16 meq of magnesium sulphate and loaded with 150 mg of amiodarone to prophylactically address reperfusion ventricular arrhythmias. Ventilation was started after endobronchial suctioning. After optimizing the metabolic and oxygenation /ventilation status and activation of AV sequential pacing at 90 with DOO mode due to sinus bradycardia, the patient was started to be off the bypass with infusions of epinephrine 0.1 µ/kg/min, nor-epinephrine 0.05 µ/kg/min and dobutamine 5 µ/kg/min. The systemic pressures remained stable with systolic pressures above 100 and PAs initially 2/3rd systemic, but eventually the PAs came down to less than half of the systemic pressure. Immediately post-valve replacement reversibility of PH showed that it was secondary to tightly stenosed aortic valve. TOE showed no intra-cardiac air (left atrium and left ventricle), an appropriate aortic valve placement and function with no leak.

The patient's chest was closed after appropriate hemostasis and shifted to cardiac intensive care unit (CICU). The patient was extubated after 6 h in the CICU with optimized hemodynamics, chest tube output, metabolic and extubation parameters. He remained stable post-extubation.

On third postoperative day, he was shifted to special care unit. His symptoms improved remarkably. He was discharged home on 6th postoperative day after an uneventful hospital course on beta-blockers, ACE inhibitor and diuretic.

He was followed up in the clinic within 2 weeks and found to be doing well. Indirect telephonic follow up at 3 months revealed improved functional status.

DISCUSSION

Calcific aortic stenosis (AS) has become one of the most frequent types of valvular heart disease (VHD) among elderly patients. Prevalence of aortic valve disease (AVD) increases with age and the incidence of calcific AS is on the rise as the general age of the population increases. Severe AS with reduced LV function has high operative mortality.^{5,6} Presence of pulmonary artery hypertension makes it further

urgent aortic valve replacement

challenging. According to a study severe PH in patients with severe AS is associated with increased rates of in-hospital adverse events and decreased 5-year survival after Surgical aortic valve replacement (AVR).⁷ SVR is the only effective corrective treatment, prolongs survival, and greatly improves symptoms. LV dysfunction is due to afterload mismatch plus diastolic dysfunction due to concentric hypertrophy as seen in severe AS. Aortic valve replacement results in improvement in symptoms and survival. In patients undergoing AVR with reduced LV function mortality is around 10-25%. Severe AS is defined as an aortic valve area (AVA) of 1 cm^2 and or indexed AVA $0.6 \text{ cm}^2 / \text{m}^2$ and a mean trans-valvular gradient (40 mm/Hg) based on Doppler echocardiography. In our patient the valve area was 0.3 cm^2 , that is less than critical stenosis, along with reduced LV function and severe PAH.

Providing safe effective anesthesia for these procedures is through understanding the pathophysiology of AS, knowing details of echocardiographic findings and of the processes and potential complications of these complex procedures in high-risk patients. In our reported case, the high operative risk was attributed to severe LV dysfunction with severe pulmonary hypertension in addition to severely reduced aortic valve area.

During intraoperative management, critical periods including induction of anesthesia, sternotomy, aortic cannulation, and institution and withdrawal of cardiopulmonary bypass should be proactively dealt with multi-disciplinary effort. Induction of anesthesia must be done with drugs having stable pharmacodynamics and with slow dose titration as the drugs have got prolong circulation time and onset of action due to fixed low cardiac output state. Maintenance of SVR is very crucial as blood pressures are maintained by normal to high afterload. Cardioplegia of a severely hypertrophied LV can be challenging particularly with aortic valve incompetence or coexisting coronary artery disease but can be aided by direct coronary ostial or retrograde coronary sinusoplegia. In the majority of patients with adequate LV function and after correction of afterload mismatch by valve replacement, weaning from bypass is uneventful. When intraoperative complications occur, they frequently relate to poor ventricular function, air embolism, and bleeding. Ventricular epicardial pacing wires reduce the risks of immediate and delayed complete heart block as done in our case.

The potential benefit of aortic valve replacement like relief of symptoms, improved quality of life and prolongation of survival outweighs extraordinary

risk in patients with LV dysfunction and pulmonary hypertension. Our patient presented with congestive heart failure, supposed to be worst symptom and associated with dismal outcome in terms of survival. Considering a very high mortality we took all the considerations in account and made our perioperative plan as a team of anesthetist, surgeon and perfusionist. The surgical team did its job by taking care of myocardial protection as in these patients due to left ventricular hypertrophy, adequate cardioplegia delivery is challenging. Similarly appropriate sizing of valve is important to have minimal gradient postoperatively. Perfusionist played their role as an important team member contributing equally to the best surgical outcome by ensuring the cardiac electrical quiescence and maintenance of optimal pump flows, perfusion pressures and metabolic parameters.

The three interventional options for severe symptomatic AS include surgical AVR, TAVI, or balloon valvuloplasty of the aortic valve. The decision to offer intervention is dependent upon risk-benefit ratio assessment. Surgical AVR remains the gold-standard intervention for severe AS. Despite a poor prognosis without intervention, at least one-third of patients with severe symptomatic AS are not surgically intervened because of high probability of perioperative risk.

The anesthetic management of this specific group of patients remains challenging as far as induction of anesthesia, pre and post CPB strategies are concerned. The presence of LV dysfunction and Pulmonary HTN requires extra vigilance and team effort to achieve desirable surgical outcome.

Aortic valve intervention improves survival and improves symptoms in patients with severe AS. History and echocardiography are indicated when intervention is mandatory. Surgical valve replacement can be undertaken with very low morbidity and mortality in the majority of patients. Perioperative management in patients with severe AS compounded by severe LV dysfunction and severe PH is extremely challenging, requiring thorough perioperative multidisciplinary preparation to address the associated complications like hemodynamic collapse at induction, lethal arrhythmias plus difficult weaning coming off the bypass.

Conflict of interest: None declared by the authors

Authors' Contribution:

MIAK + SB - Manuscript writing, literature search

SS - Manuscript editing

MH - Manuscript review

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