



Anaesthesia considerations: Active Prosthetic Valve Endocarditis with cardiogenic shock

Dr Rajvee K Gala¹, Dr Manojkumar Chauhan², Dr Rajkumar Vishwakarma³, Dr Mayur Kumar¹,
Dr Kunal Khera¹, Dr Saylee Dukhande¹

¹Cardiac Anesthesia resident, Asian Heart Institute and Research Centre, BKC

²Senior Cardiac Anesthesia Consultant, Asian Heart Institute and Research Centre, BKC

³Cardiac Anesthesia consultant, Asian Heart Institute and Research Centre, BKC

Corresponding Author

Dr Rajvee K Gala,
Cardiac Anesthesia resident, Asian
Heart Institute and Research Centre,
BKC

Source of support: Nil.

Conflict of interest: None

Received: 11-01-2025

Accepted: 13-02-2025

Available online: 23-02-2025



This work is licensed under the Creative
Commons Attribution 4.0 License.

Published by TRJMS

Abstract

Prosthetic valve endocarditis (PVE) is an infection of the endocardial surface that occurs on parts of the prosthetic valve due to microbial infestation. It is the most severe form of IE with an incidence of 1 – 6% of patients with valve prosthesis (Delgado V et al, 2023). According to 2023 European Society of Cardiologists (ESC) guidelines for management of endocarditis early PVE (within 6 months of valve surgery) is class I indication of surgery with new valve replacement and complete debridement (Delgado V et al, 2023). Surgery is also an indication in patients presenting with cardiogenic shock and heart failure secondary to PVE. Despite early use of surgical approaches, the nature of redo procedures increase the rate of morbidity and mortality with an inhospital mortality rate of about 20 – 40% (Delgado V et al, 2023). According to the 2023 ESC statistics, even though surgery is the best treatment option for PVE, it is performed in only 70% of the indicated cases due to the complexities and extensive postoperative care associated with the same. This article presents management of a patient presenting with cardiogenic shock and heart failure secondary to prosthetic valve endocarditis.

KEYWORDS: Prosthetic valve endocarditis (PVE), Aortic valve replacement (AVR) with aortoplasty, Intra aortic balloon pump (IABP), embolectomy

INTRODUCTION

Prosthetic valve endocarditis (PVE) is an infection of the endocardial surface that occurs on parts of the prosthetic valve due to microbial infestation. It is one of the most dreaded and lethal complication of valve replacement. It occurs in 1-6% of patients with valve prosthesis, with an incidence of 0.3 – 1.2% per patient per year (Delgado V et al, 2023) and accounts for 20% of all infective endocarditis (Glaser N et al, 2017). A higher risk of PVE is reported in patients with biological compared with mechanical valves (Glaser N et al, 2017). Patients with PVE present with symptoms similar to other types of infectious endocarditis (IE), including fever and chills, shortness of breath, pleuritic chest pains, anorexia, and weight loss (Khalil H et al, 2022). The medical management involves maximum non-toxic dose of empirical higher antibiotic therapy for atleast 6 weeks. However, a surgical intervention is indicated in case patient develops heart failure, severe prosthetic valve dysfunction, intracardiac fistula, presence of bacteremia even after appropriate antibiotic regime, development of abscess or fungal/highly resistant bacterial organism causing PVE. Despite the appropriate diagnosis and use of surgical treatment in PVE, morbidity and mortality remain high (Khalil H et al, 2022). Redo surgeries in addition to PVE with cardiogenic shock greatly increase the perioperative morbidity and mortality. Age, CPB time, IABP use, postoperative cerebrovascular accident and MREDO are identified as independent risk factors for 30-day mortality post redo cardiac surgeries (Norton EL et al, 2023).

PVE carries the highest mortality rates compared to other types of IE (Khalil H et al, 2022). Hence, multidisciplinary action with interdepartmental coordination is required to successfully manage such a case.

This case report describes successful anaesthetic, surgical and intensive care management of prosthetic aortic valve endocarditis requiring redo aortic valve replacement with a preoperative EuroscoreII of 76.73%.

CASE REPORT

A 51year old, male (height: 185cms, weight: 100kgs), known case of rheumatic heart disease and bicuspid aortic valve underwent bioprosthetic aortic valve replacement with aortic root replacement 2 years back, presented with multiple episodes of highgrade fever in past 2 months, which were insidious in onset and relieved with antibiotics prescribed by family physician. About 3 weeks back he developed breathlessness of NYHA grade III, associated with orthopnea and feeling of chest tightness for which he was taken to nearby hospital. He was diagnosed with heart failure associated with prosthetic valve endocarditis and acute kidney injury and was medically managed for the same. However, the patient condition deteriorated and he was transferred via cardiac ambulance to our institute for further evaluation and management. On examination his heart rate was 80/min and regular, blood pressure was 80/60 mm Hg, saturation of 100% on room air, bilateral pedal edema and crepitations were present in bilateral lung fields. He also gave history of undergoing dental procedure 6 months back without an adequate antibiotic cover which was thought to be the cause for PVE.

The inotrope and vasopressor support was escalated and intraaortic balloon pump (IABP) was inserted percutaneously via right femoral artery to maintain hemodynamics. Bedside 2D Echocardiography showed an echogenic structure of 18mm X 20mm moving to and fro and was attached to aortic valve, suggestive of aortic valve vegetation; severe Aortic stenosis with max/mean gradient of 82/46 mm Hg; Aortic annulus of 0.5cm²; severe Left Ventricle systolic dysfunction with ejection fraction of 20% and global LV hypokinesia. Based on the given scenario it was decided to post the patient for Redo Prosthetic Aortic Valve replacement with aortoplasty after preoperative optimization.

The left radial artery and right internal jugular central venous access was secured in ICU. Foleys catheter was inserted and hourly urine output was noted. Preoperative blood investigations stated hemoglobin 10.8gm%, serum creatinine 3.1mg/dL, blood urea nitrogen 44mg/dL, INR 1.7 and serum potassium 3.9mEq/L. Four units of fresh frozen plasma was transfused a day prior to surgery in view of deranged INR.

On the day of surgery, patient was shifted from ICU to operation theatre with hemodynamic monitoring and IABP support with arterial blood pressure of 90/56 mm Hg at 1:1 IABP support and augmentation of 106 along with infusion Inj. Noradrenaline @ 0.13 mcg/kg/min and Inj. Vasopressin 0.08 units/min. The standard ASA monitors including ECG, saturation probe were applied. Left radial artery cannula was transduced to determine invasive arterial pressure. He was induced with Inj. Etomidate and Inj. Rocuronium. Pulmonary artery catheter with CCO-SvO₂ monitoring was inserted via a 8.5Fr sheath through right internal jugular venous access. Pre procedure baseline cardiac index was 1.2ml/min/m², cardiac output 2.9L/min and opening Pulmonary artery pressure was 65/42 mm Hg. Transesophageal echocardiography was also used intraoperatively and findings were confirmed (fig 1 and 2).

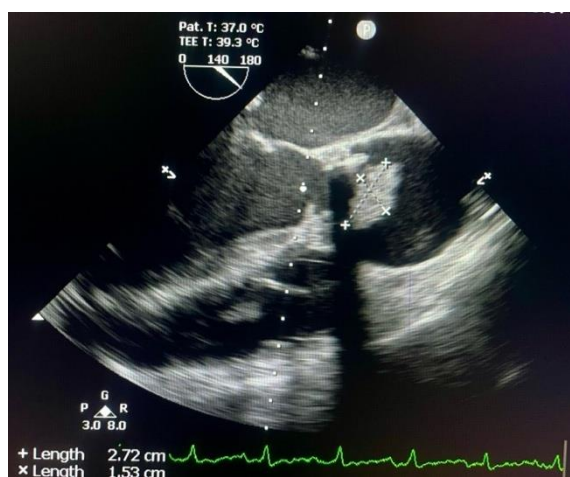


Fig 1 – TEE mid esophageal Aortic valve in long axis view showing echogenic structure attached to aortic valve leaflet s/o vegetation.

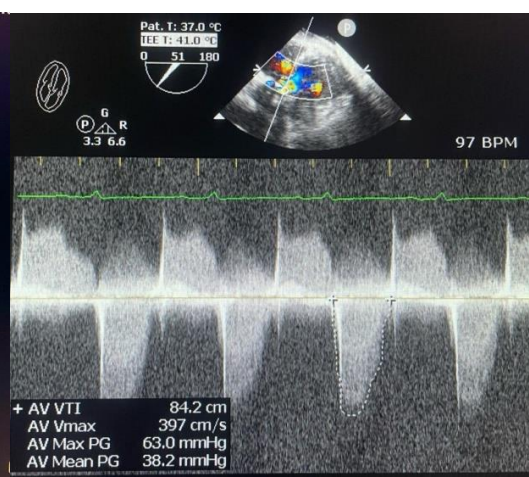


Fig 2 – TEE deep transgastric view pulse wave doppler showing gradient across aortic valve s/o Severe Aortic stenosis.

Balanced anaesthesia was maintained with Isoflurane, Inj. Atracurium infusion and Inj. Midazolam + Buprenorphine infusion. Inotrope and vasopressor infusions of Inj. Noradrenaline, Vasopressin and Dopamine were titrated to maintain a mean arterial pressure of more than 65mmHg.

Right Innominate artery using a 10mm graft and right atrium was used for arterial and 29Fr three stage single venous cannulation respectively. LV vent was inserted in the right superior pulmonary vein and a retrograde cardioplegia cannula was inserted in coronary sinus. Cannulation was done after heparinization with 4mg/kg of intravenous Heparin and ACT of 511seconds. Del Neido cardioplegia solution was used to arrest the heart and start cardiopulmonary bypass and it was repeated every 45 minutes. The prosthetic aortic valve sutures were cut and it was removed along with the vegetation. The annular tissue was scooped and debrided. After a thorough wash the instrument set and trolley were changed. A new tissue bioprosthetic valve of 25 mm was inserted and ascending aortoplasty was done. On pump the ACT was maintained above 500 seconds. Hemofilter was used for hemoconcentration and ultrafiltration during CPB. Aortotomy was closed and after a hot shot of warm cardioplegia and adequate deairing the aortic cross clamp was removed. The total cross clamp time was 2 hours 30 minutes. After a reperfusion time of 45 minutes the patient was gradually weaned off bypass. The total cardiopulmonary bypass time was 4 hours 9 minutes. Decannulation was done and adequate hemostasis was achieved after an adequate dose of Inj. Protamine for heparin reversal and to achieve an ACT of 138 seconds post bypass. Post procedure Transeoesophageal study showed no paravalvular leak and a maximum/mean gradient of 6.6/3.3 mm Hg across the neo aortic valve (Fig 3). The CCO-SvO₂ monitor showed an improved cardiac output of 6.3L/min and cardiac index of 3.1 ml/min/m² (Fig 4) with high dose inotrope and intraaortic balloon pump. Massive blood transfusion including 3150ml of PRBC, 1600ml of fresh frozen plasma and 300ml of cryoprecipitate were transfused intraoperatively.

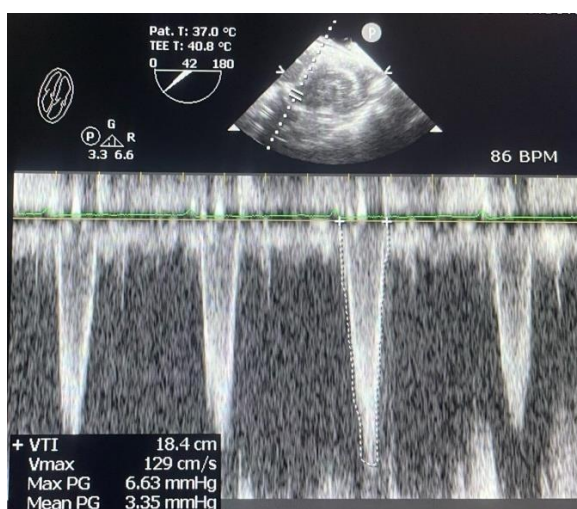


Fig 3 – TEE deep transgastric view pulse wave doppler showing reduced gradient across aortic valve post AVR

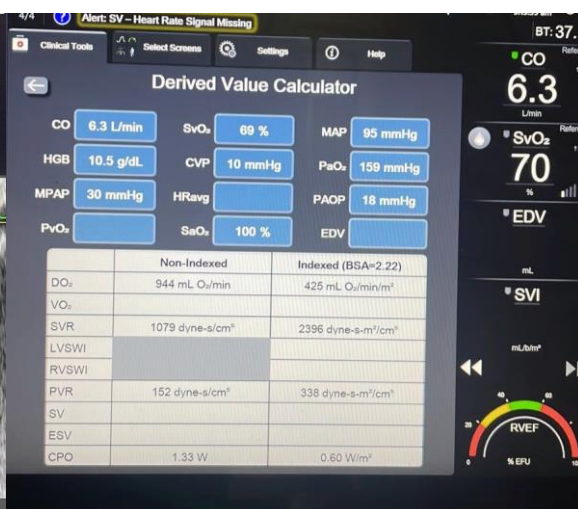


Fig 4 – Advanced CCO-SvO₂ monitoring setup monitor

A right ventricular epicardial pacing wire and two pericardial drains were inserted prior to sternum closure. Post procedure patient was shifted to surgical intensive care sedated, intubated and on maximum augmentation of intraaortic balloon pump, Inj Noradrenaline at 0.13mcg/kg/hr infusion, Inj. Dopamine at 0.06mcg/kg/min infusion, Inj. Vasopressin at 0.08 units/min and Inj. Adrenaline at 0.05mcg/kg/min infusion to maintain a mean arterial pressure of 65 to 80 mm Hg.

COURSE IN SICU

Patient was maintained on spontaneous intermittent mechanical ventilation overnight with all standard monitoring and invasive monitoring like arterial blood pressure, pulmonary artery pressure and CCO-SvO₂ Swan Ganz. An additional 450ml of PRBC, 300ml of cryoprecipitate and 300ml of single donor platelet concentrate were transfused overnight and inotropes were tapered to maintain a mean arterial pressure above 65mm Hg. On postoperative day 1 the patient was found to have absent distal pulses in right lower limb. An arterial doppler was done which suggested diffuse narrowing with linear flow likely chronic diathesis in right popliteal artery followed by very sluggish, non-pulsatile flow in anterior and posterior tibial arteries. The patient was immediately taken for an emergency embolectomy and IABP removal from right femoral artery which was done under monitored anaesthesia care using Inj Remifentanyl 2mcg/kg/hour infusion and inhalational anaesthetic isoflurane. Procedure resulted in profuse bloodloss requiring 900ml of PRBC transfusion intraoperatively. Patient was thoroughly monitored in the surgical ICU and extubated on post operative day 4. Post operatively the patient's serum creatinine showed a downward trend reaching 1.3mg/dL by post operative day 4. Rest of the hospital stay remained uneventful and the patient was discharged on post operative day 18.

DISCUSSION

Prosthetic valve endocarditis (PVE) is one of the most deleterious complications of valve replacement surgeries affecting the surgical outcome and survival rate of patients even after timely detection. It is the most severe form of IE with an incidence of 1 – 6% of patients with valve prosthesis with an incidence of 0.3 – 1.2% per patient-year (Delgado V et al,

2023). As depicted in our case, inadequate antibiotic cover after high risk procedures like dental treatment or scopes or other surgical procedures post surgery are the leading cause of PVE. The evolution and a wider availability of imaging tools leading to an early diagnosis and the general improvement in the treatment of sick patients with comorbidities have certainly enhanced the possibility of offering a successful surgical treatment in the case of complicated prosthetic aortic valve endocarditis (De Palo M et al, 2021). However, despite these advances, aortic PVE is still associated with substantial early morbidity and mortality (De Palo M et al, 2021). It is also essential to distinguish early and late PVE based on time of valve surgery, because of significant differences in microbiological profiles between these two groups (Delgado V et al, 2023). A major difference being that the vegetations of PVE are relatively larger than native valve endocarditis and require greater doses of antibiotics. A surgical strategy is recommended for PVE in high risk subgroups like PVE complicated with heart failure, severe prosthetic valve dysfunction, abscess or persistent fever (Delgado V et al, 2023). According to 2023 ESC guidelines for management of endocarditis early PVE (within 6 months of valve surgery) is class I indication of surgery with new valve replacement and complete debridement (Delgado V et al, 2023). The primary objectives of surgical treatment are radical debridement and the removal of infected tissues, the reconstruction of cardiac and aortic morphology, and the restoration of the aortic valve function (De Palo M et al, 2021). As mentioned our patient was also on IABP support preoperatively in view of hemodynamic instability due to cardiogenic shock. Cardiogenic shock is a state of low cardiac output associated with hypotension and evidence of end organ hypoperfusion (Telukuntla KS et al, 2020). There has been an increase in the use of mechanical circulatory support (MCS) devices to improve outcome in patients with cardiogenic shock. Various MCS devices are available with wide range of use, it is important to select an appropriate device based on patient demographics and underlying pathology. The SHOCK Trial Registry (Sanborn et al. 2000) found that patients with cardiogenic shock had decreased in-hospital mortality when treated with IABP therapy (Sice A, 304). The balloon works on the principle of counterpulsation. In our case the patient presented with cardiogenic shock and IABP counterpulsation aided in reducing the myocardial demand by reducing preload and afterload and in turn maintaining coronary and cerebral perfusion pressures which improved patient outcome. Despite these advantages IABP also has increases the morbidity and mortality due to its associated vascular complications. Complications associated with IABP can be either major like vascular injury and limb ischemia or minor like local hematoma or infection. The incidence of IABP-related vascular complications varies widely, from 0.94% to 31.1% (De Jong MM et al, 2018). The most frequent vascular complication associated with IABP insertion was limb ischemia which had an incidence ranging from 0.9% to 26.7% (De Jong MM et al, 2018). Mesenteric ischemia is rare (0.9% incidence). However it can be life threatening if progressed to bowel gangrene. Some cases of minor limb ischemia resolve after removal of IABP. However in our case it was major, embolectomy was performed to resume blood flow. IABP acts as a bridge, stabilizing and optimizing the patient for definitive management of the underlying cause. Hence, prompt management and reversal of the underlying disease is of utmost importance in these cases as duration of IABP support is also an independent risk factor for development of vascular complications.

Aside from all the complications of this case, a redo surgery poses additional risk. Redo aortic surgery can be performed with a 12% to 21% risk of mortality in a high-volume centre of expertise (Norton EL et al, 2023). For prior anticipation of difficult redo sternotomies computed tomography is an essential preoperative test as it traces the relation between cardiac structures and the posterior table of the sternum. This is also helpful in deciding the need for preparation of alternate cannulation sites (axillary or femoral) prior to sternotomy like in cases where aorta is densely adhered to sternum.

The adjunct of invasive monitoring also plays an important role in objectively managing such a case. The continuous cardiac output monitoring with a CCO – SvO₂ Swan Ganz catheter as used in our case not only measured cardiac output but had algorithms to indirectly calculate systemic and pulmonary vascular resistance. The ability to view simultaneous trends of mixed venous saturation and right ventricular performance parameters allows the operator to graphically see the impact of volume loading or inotropic therapy over time, as well as the influence of multiple factors, including right ventricular dysfunction, on mixed venous saturation (Cariou A et al, 1998).

CONCLUSION

This case depicts a successful outcome of a patient with cardiogenic shock associated with prosthetic aortic valve endocarditis. A well coordinated multidisciplinary team including an anaesthetist, surgeon and intensivist and an understanding of the underlying pathology and its implications at every stage of management plays a very important role in managing such a case.

REFERENCES

1. Delgado, V., Ajmone Marsan, N., de Waha, S., Bonaros, N., Brida, M., Burri, H., ... & Borger, M. A. (2023). 2023 ESC Guidelines for the management of endocarditis: Developed by the task force on the management of endocarditis of the European Society of Cardiology (ESC) Endorsed by the European Association for Cardio-Thoracic Surgery (EACTS) and the European Association of Nuclear Medicine (EANM). *European heart journal*, 44(39), 3948-4042.
2. Glaser, N., Jackson, V., Holzmann, M. J., Franco-Cereceda, A., & Sartipy, U. (2017). Prosthetic valve endocarditis after surgical aortic valve replacement. *Circulation*, 136(3), 329-331.
3. Khalil, H., & Soufi, S. (2021). Prosthetic valve endocarditis

4. Norton, E. L., Kalra, K., Leshnower, B. G., Wei, J. W., Binongo, J. N., & Chen, E. P. (2023). Redo aortic surgery: Does one versus multiple affect outcomes?. *JTCVS open*, 16, 158-166.
5. De Palo, M., Scicchitano, P., Malvindi, P. G., & Paparella, D. (2021). Endocarditis in patients with aortic valve prosthesis: Comparison between surgical and transcatheter prosthesis. *Antibiotics*, 10(1), 50.
6. Telukuntla, K. S., & Estep, J. D. (2020). Acute mechanical circulatory support for cardiogenic shock. *Methodist DeBakey Cardiovascular Journal*, 16(1), 27.
7. Sice, A. (2006). Intra-aortic balloon counterpulsation complicated by limb ischaemia: a reflective commentary. *Nursing in critical care*, 11(6), 297-304.
8. de Jong, M. M., Lorusso, R., Al Awami, F., Matteuci, F., Parise, O., Lozekoot, P., ... & Gelsomino, S. (2018). Vascular complications following intra-aortic balloon pump implantation: an updated review. *Perfusion*, 33(2), 96-104.
9. Cariou, A., Monchi, M., & Dhainaut, J. F. (1998). Continuous cardiac output and mixed venous oxygen saturation monitoring. *Journal of critical care*, 13(4), 198-213.