Improving Hemodynamics – Prolonged Intra-aortic Balloon Pump Usage in Posterior Ventricular Septal Rupture with Right Ventricle Dysfunction

Abstract
The associated mortality and morbidity of posterior ventricular septal rupture (VSR) is quite high increasing to almost 80% due to severe right ventricle dysfunction and pulmonary artery hypertension. Herein, we present a case of posterior VSR due to inferior wall myocardial infarction (MI) who underwent surgery. Premature removal of intra-aortic balloon pump (IABP) led to hemodynamic deterioration and he was salvaged with prolonged and prompt re-institution of IABP. This case also highlights the importance of IABP in right ventricle failure.

Keywords: Intra-aortic balloon pump, posterior, right ventricle, ventricular septal rupture

Introduction
Posterior ventricular septal rupture (VSR) is quite a rare phenomenon with an incidence of 0.1%–0.2% among all the mechanical complications of myocardial infarction (MI). This case is presented for rarity and the importance of prolonged intra-aortic balloon pump (IABP) usage in salvaging the patient from complete hemodynamic collapse to restoring good hemodynamics.

Case Report
A 48-year-old diabetic male was referred to us with a diagnosis of basal VSR, approximately 2 weeks before MI. He had a history of breathlessness of New York Heart Association class III with bilateral pedal edema and oliguria for the past 15 days. On admission, he was hemodynamically stable with a blood pressure of 120/80 mmHg and heart rate of 110/min. He had elevated cardiac markers (troponin I of 0.1 ng/mL) with electrocardiogram showing Q waves in leads II, III avf showing evolved inferior wall (IW) and right ventricle (RV) MI. A two-dimensional echocardiography showed hypokinetic basal septum and a huge VSR [Figure 1a] of 18 × 20 mm in size shunting left to right [Video 1] with severe pulmonary arterial hypertension (RV systolic pressure 75 mmHg) and biventricular dysfunction and mild mitral regurgitation (MR) with an RV wall motion index (WMI) of 0.8. An elective coronary angiogram showed proximal tight occlusions of both the right coronary artery (RCA) and the obtuse marginal (OM) branch of the left circumflex. The left anterior descending artery was normal. He was planned for surgical closure of the VSR with grafts to OM and RCA territories after a week’s time.

A Swan-Ganz catheter was inserted after induction showing a pulmonary capillary wedge pressure of 24 mmHg, a pulmonary artery systolic pressure (PASP) and diastolic pressure (DP) of 60 and 33 mmHg, respectively, and a diastolic pulmonary gradient of 9 mmHg. The central venous pressure (CVP) was 15 mmHg. Simultaneous right atrial and pulmonary artery saturations showed a step up of 30%. Cardiopulmonary bypass (CPB) was instituted with aorto bicaval cannulation and the heart was arrested by Del Nido cardioplegia (aortic root). After adequate retraction, a vertical left ventriculotomy was done parallel to the posterior descending artery (PDA) in the diaphragmatic surface of the heart. The VSR was visualized with difficulty by retracting the papillary muscle [Figure 2a] and its chordae tendinae. It was closed with bovine pericardium with interrupted 2-0 polypropylene sutures using David technique. The left ventriculotomy was closed [Figure 2b] followed by OM

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and PDA grafting as planned. After adequate period of CPB support with an aortic cross clamp time of 88 min and CPB time of 145 min, the patient was weaned off CPB. Transesophageal echo showed intact VSR patch with biventricular dysfunction. During closure, arterial pressures were 100/60 mmHg with a PASP of 45 and DP of 23 mmHg. The patient was shifted to the intensive care unit (ICU) with supports of inj. dobutamine 5 μg/kg/min, inj. adrenaline 0.08 μg/kg/min, and inj. vasopressin 2 IU/h. As the cardiac contractility was reasonably alright, IABP was not inserted in the operation theater with a low threshold of insertion in case of eventuality.

The patient was shifted to the ICU with continuous monitoring with cardiac output (CO) of 3.5 L/min and cardiac index (CI) of 1.9 L/min/m² and systemic vascular resistance (SVR) of 1900 dynes-s/cm⁵. After 6 h, there was a drop in CO and SVR elevation, and hence a 40-cc IABP was inserted percutaneously through the right common femoral artery (CFA). The patient’s hemodynamics improved and he was extubated on the second postoperative day (POD). After 6 h, there was once again a drop in CI and CO and echo showed severe RV dysfunction with a tricuspid annular plane systolic excursion (TAPSE) value of 8 mm. Inotropes were hiked with no further improvement. Twelve hours later, the patient became dyspneic, tachypneic with a mixed venous oxygen saturation (MVO₂) of 35%, and a pulmonary vascular resistance of 250 dynes-s/cm² and was electively reintubated in view of hypoxia due to low cardiac output. A sheathless 34-cc IABP was re-inserted percutaneously through the left CFA. The hemodynamics improved and MVO₂ improved to 35%, and a pulmonary vascular resistance of 250 dynes-s/cm² and was electively reintubated in view of hypoxia due to low cardiac output. A sheathless 34-cc IABP was re-inserted percutaneously through the left CFA. The hemodynamics improved and MVO₂ improved to 70%. He was extubated the next day and a decision was made to retain the IABP. The IABP was removed after 10 days (1:1 augmentation for 8 days and 1:2 for the next 48 h and 1:3 for the next 24 h) and inotropes were reduced. Repeat echo showed improvement in the RV function with a TAPSE improving to 12 mm. He was discharged in a stable condition on 18th POD. Discharge echo revealed intact VSR patch [Figure 1b] with moderate biventricular dysfunction and TAPSE value of 12 mm and moderate MR [Video 2]. He is planned for MR follow-up and surgical intervention at a later date.

Discussion

Posterior VSRs are quite large and very rare and associated with significant mortality and morbidity. In a study by Normand et al., the mortality rate was almost 80%. This was due to the following:
1. Greater extent of the infarcted area than expected
2. Associated MR due to papillary muscle/chordae tendinae involvement[2]
3. Difficult exposure.

The MR what we observed in our patient during discharge was probably attributed to the damage to the papillary muscles during VSR retraction.

Prognostic indicators in an inferior VSR are as follows:
(1) CI of 1.75 L/min/m² or less,
(2) Echo evidence of severe RV dysfunction shown by RV WMI of 1.0 or less
(3) CVP of 12 mmHg or more, and
(4) Early occurrence (i.e., <6 days) of VSR after MI.[3]

Our patient had all the above said factors.

A causal relationship between RV dysfunction and a diminished CI always exists in IW MI.[4] IABP improves RV function as in our case. The temporal relationship of trend of cardiac indices with the IABP removal and re-insertion in our patient is shown in Figure 3. The hemodynamics deteriorated on premature removal, and prompt and prolonged re-insertion of the IABP led to improvement in CI. The optimal timing and removal of IABP are debatable,[5] and we advocate keeping a sheathless IABP for a prolonged time (with good limb vascularity)
till the RV function improves significantly. There is also a case report wherein IABP was kept for 33 days for hemodynamic stability of a VSR patient.\[6\]

The efficacy of the IABP is due to the following:
1. Increase in coronary perfusion with rapid healing of ischemic myocardium around VSR
2. Salvage of peri‑infarct zone
3. Reduction in SVR favoring systemic flow
4. Decrease in RV afterload due to decreased LV filling pressure.\[5\]

The clinical status of the patient and the timing of surgery determine the outcome. There are two leading theories for delayed repair favoring good outcome. Delayed surgical repair (time from the onset of VSR to surgery >9 days) gives more chance for the friable infarcted tissues to mature. Clinically stable patients might be pushed into the delayed repair group rather than the urgent high risk and sicker substrate.\[7\] In the STSACSD study, 7 days was the cut‑off and earlier repair than this period was associated with a greater mortality (54.1% vs 18.4%, \(P < 0.01\)).\[8\] As our patient was clinically stable, we decided to operate in an elective basis with intensive monitoring.

To conclude, proper hemodynamic management with adequate IABP support can translate a dangerously mortal condition like posterior VSR into a smooth postoperative outcome.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Nil.

Conflicts of interest

There are no conflicts of interest.

References

1. Normand JP, Epois A, Melman S, Bergue A, Bourdarias JP, Mathivat A. La perforation du septum interventriculaire a la phase aigue de l’infarctus myocardique. Ann Med Intern (Paris) 1972;123:641‑7.
2. Buckley MJ, Mundth ED, Daggett WM, Gold HK, Leinbach RC, Austen WG. Surgical management of ventricular septal defects and mitral regurgitation complicating acute myocardial infarction. Ann Thorac Surg 1973;16:598‑609.
3. Crosby IK, Craver JM, Crampton RS, Schrank JP, Wellons HA. Resection of acute posterior ventricular aneurysm with repair of ventricular septal defect after acute myocardial infarction. J Thorac Cardiovasc Surg 1975;70:57‑62.
4. Gaediani VA, Miller DC, Stinson EB, Oyer PE, Reitz BA, Moreno‑Cabral RJ, et al. Postinfarction ventricular septal defect: An argument for early operation. Surgery 1981;89:48‑55.
5. Ashfaq A, Jaroszewski DE, Pajaro OE, Arabia FA. The role of the total artificial heart in the treatment of post‑myocardial infarction ventricular septal defect. J Thorac Cardiovasc Surg 2013;145:e25‑e6.
6. Estrada‑Quintero T, Uretsky BF, Murali S, Hardesty RL. Prolonged intraaortic balloon support for septal rupture after myocardial infarction. Ann Thorac Surg 1992;53:333‑7.
7. Jones BM, Kapadia SR, Smedira NG, Robich M, Tuzcu EM, Menon V, et al. Ventricular septal rupture complicating acute myocardial infarction: A contemporary review. Eur Heart J 2014;35:2060‑8.
8. Arnaoutakis GJ, Zhao Y, George TJ, Sciortino CM, McCarthy PM, Conte JV. Surgical repair of ventricular septal defect after myocardial infarction: Outcomes from the Society of Thoracic Surgeons National Database. Ann Thorac Surg 2012;94:436‑43; discussion 443‑434.