Peri-operative challenges in post myocardial infarction ventricular septal rupture: A case series and review of literature

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ABSTRACT

Ventricular septal rupture (VSR) is a life threatening complication of myocardial infarction (MI). The incidence of post-MIVSR varied from 1% to 3% in the pre-thrombolytic era. There is almost a 10-fold decrease in the reported incidences (0.2-0.3%) of MIVSR today. The mortality in such an event is as high as 50-90%. Prognosis of post-MIVSR depends on prompt echo diagnosis and proactive surgical therapy. The peri-operative challenges during management of such a case can be enormous.

INTRODUCTION

Post-infarction ventricular septal rupture (VSR) is an uncommon but potentially fatal mechanical complication of myocardial infarction (MI). Latham first described this condition at autopsy in 1847.[1] In the year 1923, Brunn made first ante-mortem diagnosis of post-MIVSR.[2] Sager in 1934 established specific clinical criteria for diagnosis, stressing the association of post-MIVSR with coronary artery disease.[3] In an article in 1957, Cooley *et al*. performed first surgical repair of post-MIVSR.[4] It is known to complicate 1-3% of acute MI's in the pre-thrombolytic era and 0.2-0.34% in the post-thrombolytic era.[5,6] In this case series, we are presenting the current status of peri-operative management of post-MIVSR in the setting of congestive cardiac failure with compromised renal function.

CASE REPORTS

Case 1

The present case report is about a 55-year-old female patient who was referred with diagnosis of post-MI-VSR. Her presenting complaints were severe dyspnea on exertion with chest pain for 3 days. Physical examination showed that patient was conscious and oriented. Her pulse rate (PR) was 110 beats/min and blood pressure (BP) was 90/60 mmHg. Fine crepitations were present in both lung bases and pansystolic murmur detected over left fourth intercostal space. Laboratory findings showed hemoglobin 11.6 g%, blood urea 67 mg%, serum creatinine 1.5 mg%. Chest X-ray (posterior-anterior view) showed pulmonary congestion and cardiothoracic ratio of 0.6. Electrocardiogram (ECG) showed normal sinus rhythm, Q waves with ST elevation in V_1-V_4, T inversion in V_5-V_6 suggestive of antero-lateral wall MI.

Transthoracic echocardiography (TTE) with Doppler showed akinetic area in apex, ejection fraction of 40% and an apical VSR of 7 mm with left to right shunt. The pressure gradient across the defect was 50 mmHg. Tricuspid regurgitation was mild with moderate pulmonary

arterial hypertension. Coronary arteriogram showed left dominance with 100% occlusion in left anterior descending (LAD) artery in mid segment. Left circumflex artery showed 80% block in distal segment.

After taking informed consent for coronary artery bypass grafting (CABG) and VSR closure, patient was shifted to the operation theater (OT). General anesthesia was induced with fentanyl 4 mcg/kg, etomidate 0.2 mg/kg in slow titrated dosage and endo-tracheal intubation was facilitated with 50 mg of rocuronium bromide. Intra-operative transesophageal echocardiography (TEE) was consistent with pre-operative TTE findings [Figure 1]. General anesthesia was maintained with fentanyl, midazolam and vecuronium. Patient was ventilated with O_2: Air (50:50) to maintain normocarbia and to avoid hyperoxia.

After median sternotomy and systemic heparinization, cardio-pulmonary bypass (CPB) commenced and patient was cooled to 32°C. LAD and obtuse marginal artery were grafted with reversed saphenous vein. The ruptured septum was closed through trans-ventricular approach with a Gore-Tex patch. During CPB, mean perfusion pressure was maintained between 70 and 90 mmHg. After the repair, patient was weaned off CPB gradually with the support of dobutamine at 5 mcg/kg/min and nitroglycerin (NTG) at 0.5 mcg/kg/min infusions. TEE confirmed the successful repair of VSR. The CPB time and aortic cross clamp time was 177 and 117 min respectively. Intra-operative urine output was 1000 ml and 1000 ml of hemo-filtrate was removed. Patient was transported to intensive care unit (ICU) with invasive monitoring and 'handed off' to the critical care team.

In the ICU, patient developed hypotension for which adrenaline at 0.1 mcg/kg/min and dopamine at 10 mcg/kg/min were started and dobutamine stepped up to

Figure 1: The 7 mm ventricular septal defect (post-myocardial infarction) in mid-esophageal four chamber view with color flow Doppler for comparison

10 mcg/kg/min. Subsequently, her urine output decreased and lasix infusion was started. Patient was tracheostomized as she needed prolong ventilatory support. Her general condition gradually improved and she was transferred to the ward after 15 days.

Case 2

The second case report is about a 55-year-old male patient who was referred from a private hospital with 72 h history of anteroseptal ST elevation MI. He was a known hypertensive and was on antihypertensive medication for 2 years. He had dyslipidemia and was a modified smoker (more than 20 cigarettes a day since 15 years of age) and he left the habit 2 years prior to current admission. He complained of dyspnea and angina on exertion for 2 years but his symptoms aggravated suddenly 3 days back.

On examination, he was conscious, oriented, afebrile and pale, had pitting type pedal edema and his jugular venous pressure was raised. His PR was 72/min, BP was 110/60 mmHg, respiratory rate was 28 breaths/min. Chest and cardiovascular examinations revealed bilateral fine basal crepitations and grade three holosystolic murmurs along the left parasternal border respectively. The initial investigation revealed Hb 10.1 g/dl, Na 131, K 3.88 mmol/l, urea 54 and creatinine 2.1 mg%. ABG revealed pH 7.39, pCO_2 23.1, pO_2 79 mmHg, bicarbonate 21.4 mmol/l and SaO_2 97% on room air. A Hudson oxygen mask (O_2 flow at 6 l/min) was provided to the patient. His ECG showed Q wave and ST segment elevation in lead V_1 to V_4. A TTE with color flow Doppler revealed a large mid-muscular VSR with left to right shunt and a gradient of 36 mmHg and left ventricular ejection fraction (LVEF) of 50%. Subsequent coronary angiography confirmed the presence of occlusive disease of LAD (mid segment) and a left ventriculogram confirmed the presence of the mid-muscular VSR.

After initial attempted hemodynamic stabilization in the ICU, an emergency CABG with VSR closure was planned, because of the deteriorating hemodynamics with evidence of poor systemic perfusion such as decreasing creatinine clearance and persistent cold extremities. After shifting patient to the OT and connecting standard monitoring, intra-aortic balloon pump (IABP) support was initiated before induction of anesthesia. Prior to the insertion, a written informed consent was obtained from the patient after explaining the benefits of IABP assistance. General anesthesia was induced with titrated doses of fentanyl and etomidate and tracheal intubation was facilitated with 1 mg/kg of injection rocuronium bromide. After a smooth and uneventful induction, a TEE probe was inserted for assessment of LV function and anatomy of the inter-ventricular septum. During CPB a mean perfusion pressure of 60-80 mmHg was maintained and the patient
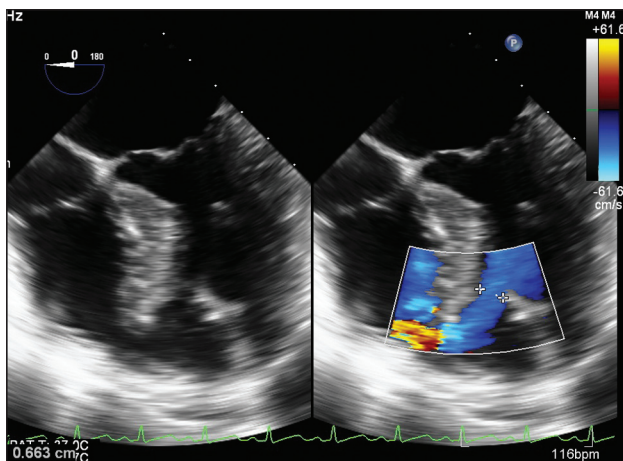
was cooled to 32°C. The aorta was clamped followed by cardioplegia administration for myocardial protection. The IABP was put on standby after the heart was arrested. The LAD was grafted with a reversed saphenous vein and the VSR was closed with a Gore-Tex patch. During the rewarming phase of CPB, infusions NTG at 0.5 and dobutamine at 5 mcg/kg/min were started. The IABP inflation was resumed before weaning the patient from the CPB. Post-operative TEE examination confirmed a good LV function with no residual ventricular septal defect (VSD). After the operation, patient was transferred to the ICU with IABP support. In the ICU, the patient was ventilated overnight with FiO_2 of 60%. The IABP and ionotropic supports were also continued. The patient was weaned from the ventilator and was extubated on post-operative day 1. On day 2, the IABP support could be withdrawn and the patient was shifted to the ward later. However,, on 4th post-operative day, patient became drowsy, dyspneic and hemodynamically unstable. A bedside TTE was performed, which revealed a new VSD for which he had to be re-operated and an extended patch closure of new defect was done. Post-operative TEE showed no residual VSD. Patient's hemodynamics were maintained with dobutamine and adrenaline infusion at 10 and 0.1 mcg/kg/min respectively. Unfortunately, patient's hemodynamic condition deteriorated the very next day despite increasing doses of vasoactive drugs and IABP support (systolic BP was 88 mmHg and diastolic augmentation was 70 mmHg). A state of low cardiac output prevailed with decreasing urine output and cold extremities. A bedside TTE in the ICU revealed a 6-8 mm VSD adjacent to the 'Gore-Tex' patch with a LVEF of 25%. Unfortunately, the patient succumbed on the 11th post-operative day.

DISCUSSION

VSR is a rare complication of MI with poor prognosis. The incidence of VSR after acute MI has declined in the thrombolytic era, most likely due to early reperfusion and myocardial salvage. The onset of symptoms depends on the size of the defect (mean onset time is 3 to 5 days). The defect may be small, multiple or may consist of a linear tear. Pathologically, the defect can have ragged edges with serpentine course or multiple small defects. The septum adjacent to the rupture is often thin and necrotic. Without reperfusion coagulation necrosis develops within the 1st 3-5 days after infarction, with neutrophils entering the necrotic zone. The neutrophils undergo apoptosis and release lytic enzyme, hastening disintegration of necrotic myocardium.[5] In our second patient, the infarcted area of the VSR was probably expanding when a patch closure was done in the apparently normal margin, thereby leading

to development of a new VSR while he was recuperating after the corrective surgery.

Septal rupture results in a left-to-right shunt, with right ventricular (RV) volume overload, increased pulmonary blood flow and secondary volume overload of the left atrium and LV. Patients usually present with biventricular failure, cardiogenic shock and new onset pansystolic murmur. As LV systolic function deteriorates and forward flow declines, compensatory vasoconstriction leads to increase in systemic vascular resistance (SVR), which in turn increases the magnitude of the left-to-right shunt. The degree of shunting is determined by the size of the septal rupture, the ratio of pulmonary vascular resistance (PVR) and SVR and RV and LV function. As the LV fails and the systolic pressure declines, there is a proportionate reduction in the left-to-right shunt fraction.

The current guidelines of the American College of Cardiology/American Heart Association recommend immediate surgical intervention to prevent further hemodynamic deterioration in patient with VSR.[7] Induction of general anesthesia for a patient with VSR and coronary artery disease is challenging. If SVR increases with sympathetic stimuli because of tracheal intubation, it also increases the shunt flow apart from increasing the oxygen demand. Therefore, induction of general anesthesia and tracheal intubation must be accomplished with minimal or no hemodynamic changes. In our patients, induction of anesthesia was performed with a combination of anesthetic drugs etomidate and fentanyl with appropriate titration.[8] The advantage of this regimen is that there is minimal myocardial depression with little alteration in SVR thus maintaining the forward flow and shunt fraction.

One of the most important concerns in anesthetic management of VSR after its closure is to reduce LV afterload while maintaining hemodynamics. Reduction of afterload is required to prevent tension in the suture line of newly repaired septum during the post-bypass period. To achieve these goals, nitroglycerin infusion was started. Dobutamine infusion was started to maintain systemic BP without increasing SVR.

Volatile anesthetics were administered to the patient as they have myocardial protective effects and afterload reduction properties which is beneficial in patient with VSR. Maintaining optimum PVR is equally important like SVR in patients with VSR. Anesthesiologists must avoid maneuvers that tend to decrease PVR/SVR ratio such as hypocapnia and hyperoxemia. Efforts should be made to maintain normocapnia and normoxia by controlled oxygen supplementation to these patients. However, it may be difficult to maintain low inspired oxygen concentration

(FiO_2) when the patient has pulmonary congestion due to left-to right shunt. In both cases, patients were ventilated with O_2-Air mixture (FiO_2 50%) to maintain normocarbia and to avoid hyperoxia as far as feasible.

In patients who received thrombolysis, the median time from the onset of symptoms of acute MI to rupture is generally 24 h or less. The median time from the onset of infarction to septal rupture was 1 day in the GUSTO-I trial and 16 h in the 'Should We Emergently Revascularize Occluded Coronaries in Cardiogenic Shock' (SHOCK) trial.[6-9] The early mortality rates for patients undergoing VSR repair are 32-54%, whereas the mortality rates for non-surgically treated patients are higher.[10-15] In surgically treated patients the 30-day mortality rate was 47% and the 1-year mortality rate was 54% whereas in medically treated patients the 30-day mortality rate was 94% and the 1-year mortality rate was 97%.[6,10-15] Surgical repair therefore, is the treatment of choice.

The risk factors for development of post-MIVSR are old age, anterior location of MI and female gender.[6] Poor prognostic factors for VSR are RV dysfunction, posterior or inferior location of infarction compared with anterior location, pathologically complex type, preoperative cardiogenic shock and lack of improvement in hemodynamic status in spite of inotropic support, shorter period from infarction to surgery, total occlusion of the infarcted artery, advanced age and female gender.[10-17]

CONCLUSION

In summary, we presented the essentials of managing a patient undergoing emergency VSR closure following acute MI. The main anesthetic concern is to maintain optimal perfusion pressure while lowering the afterload and also to maintain the SVR/PVR ratio so as to reduce the shunt fraction. Furthermore, intra-operative TEE plays an important role for assessing ventricular function and confirming the adequacy of repair. Post-operative course of these patients are usually protracted and challenging.

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