Management Issues During Postinfarction Ventricular Septal Defect and

Abstract

The development of a myocardial infarction ventricular septal rupture is a rare fatal complication, and the surgical repair is the treatment of choice. In most of the scenarios, the operation will be done as an emergency procedure that carries high mortality. Prognosis of these patients depends on prompt echocardiographic diagnosis and the proactive medical and surgical therapy. More recently, various options have been put forward including the timing for surgery, percutaneous closure devices, and the improved outcome with initial stabilization with medical treatment including mechanical support. In this retrospective case series, we are presenting the management of these patients who presented us in different clinical scenarios and trying to identify the risks for the poor outcome and to formulate a strategy to improve the outcome.

Keywords: Cardiogenic shock, coronary artery bypass graft, mechanical circulatory support, postmyocardial infarction ventricular septal rupture, ventricular septal defect

Introduction

The development of myocardial infarction (MI) ventricular septal rupture (MIVSR) is a lethal complication that can be saved by early referral, swift echocardiography evaluation, cardiac catheterization, and operation at an appropriate time. The preoperative stabilization by inotropes and mechanical support has shown to improve the outcome in these patients.^[1] Although the recommendation for an ideal timing may differ, the studies are in favor for the surgical repair as the definitive treatment.^[2] This retrospective case series details the key points in the perioperative management of MIVSR that are presented to us in different clinical scenarios.

Case Series

Informed consent has been obtained from all patients for publication.

Patient A

A 58-year-old male presented with increasing dyspnea following MI. He had thready pulse and blood pressure of 86/34 mmHg on dobutamine and adrenaline infusion at 10 and $0.1/\mu g/kg/$

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min, respectively. His peripheral oxygen saturation was 88% on high flow oxygen with widespread bilateral lung opacities on chest X-ray. Blood investigations showed hemoglobin 13.5 g/dl, blood urea 142 mg/ dl, serum creatinine 2.3 mg/dl, random blood sugar 170 mg/dl, serum sodium 128 meq/1, and serum potassium 5.1 meq/1. Urgent coronary angiography revealed blockade of left anterior descending (LAD) 90%, circumflex 50% and the total occlusion of right coronary artery (RCA). The RCA occlusion was stented successfully during the procedure. The transthoracic echocardiography showed left ventricular (LV) ejection fraction (EF) 20% and a large posterior-inferior ventricular septal defect (VSD) with a shunt gradient of 48 mmHg. Mechanical circulatory support was initiated with intra-aortic balloon pump (IABP) and as he was deteriorating rapidly, the decision was taken to perform an urgent operation with extracorporeal membrane oxygenation (ECMO) as a backup support.

General anaesthesia was induced with fentanyl/propofol in slow titrated dosage, and endotracheal intubation was facilitated with cisatracurium. Intraoperative transesophageal echocardiography (TEE) was consistent with preoperative TTE

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finding. Anaesthesia was maintained on propofol and cisatracurium infusion with supplemental midazolam, fentanyl boluses, and sevoflurane. The patient required O₂:Air (80:20) to maintain oxygenation with increasing requirement for inotropic and vasoconstrictor support. We decided to go for immediate central cannulation and subsequent cardiopulmonary bypass following anaesthetic induction. Our main aim was to decrease the shunt, prevent hypoxia and to provide an early organ support. The VSD was approximately 3.5 cm in diameter repaired by a bovine pericardial patch, and the friable ventriculotomy was closed using interrupted pledgeted stitches. The coronary artery bypass graft was performed to artery LAD using left internal thoracic artery and venous graft to its diagonal branch. Postoperative TEE showed no residual VSD, and as expected, he was unable to wean from bypass machine. We decided to continue him on central ECMO support to decrease the tension on VSD pericardial patch during immediate postbypass period.

Postoperatively, he required venovenous hemofiltration and the central ECMO switched over to peripheral with femoral cannulation after 72 h. He was eventually weaned from ECMO and IABP support on day 6 and required 43 days stay in cardiac rehabilitation facility and continued to do well following discharge and subsequent follow-up.

Patient **B**

A 53-year-old man transferred 2 days following MI was awake, comfortable on room air, and hemodynamically stable without inotropes. His coronary angiogram revealed subtotal occlusion of LAD, and transthoracic echo showed a VSD 1.3 cm located on the anterior septum with EF 50%. He was scheduled for urgent closure of septal defect and a bypass grafting for LAD after the IABP insertion. General anesthesia was induced with remifentanil and propofol in titrated doses and maintained on midazolam, remifentanil, and propofol infusion with cisatracurium for muscle paralysis. The LAD grafting was performed, and the closure of defect was performed using Dacron patch and the pledgeted sutures to close ventriculotomy. He could be weaned off bypass without inotropes and was extubated on 1st postoperative day. The patient was discharged from intensive care in 48 h, and his remaining postoperative course was free of major complications.

Patient C

A 56-year-old male admitted 4 days following a 2.2 cm postinfarction VSD on TTE. His BP was 102/66 mmHg on dobutamine 6 μ g/kg/min. Coronary angiography revealed total occlusion of LAD and 70% posterior descending artery stenosis. The intraoperative TEE revealed a VSD found at middle of the very friable, necrotic anterior septum with poor ventricular function. VSD was closed with Dacron patch, and grafting for two diseased vessels was performed. The patient could be weaned off

cardiopulmonary bypass (CPB) with moderate inotropes. However, unfortunately, postbypass TEE showed 0.3 cm residual leak that was left behind considering risk of second bypass and its consequences. He was carefully monitored, remained hemodynamically stable postoperatively, and required percutaneous closure to seal off the residual VSD in catheter room 4 weeks following surgery.

Patient D

A 64-year-old man with acute MI underwent primary angioplasty to the RCA. However, his symptoms got worse over the next 12 h and developed sudden florid pulmonary edema although his hemodynamics was stable requiring low dose inotropes. Echocardiography revealed moderate left ventricular function with EF 43% and a large VSD measuring 2.9 cms located in the posterior part of the ventricular septum. He was eventually required mechanical ventilation, and mechanical circulatory support was initiated using IABP. In the following 24 h, his renal function deteriorated with progressively increasing requirement of inotropic support. The surgery was deferred to optimize his respiratory and renal function using diuretics, nitrates, positive pressure ventilation, and inotropes. Over the next 3 days, his oxygen requirement gradually decreased with improvement in renal and hemodynamic parameters. The patient was taken to the operating room with minimal mechanical ventilation support 6 days following the onset of acute MI. Anesthesia continued on midazolam, fentanyl, propofol, sevoflurane and cisatracurium for muscle paralysis. Surgical repair of the defect was performed, and the operation was completed with associated saphenous grafting to the circumflex territory. The patient could be weaned off with milrinone and noradrenaline, and the IABP could be continued in postoperative period. Patients required hemofiltration for 2 days postoperatively with gradual weaning of inotropes and discharged to the ward on day 5. The patient stayed for 21 days in cardiac rehabilitation and currently doing well 4 months following the surgery.

Discussion

Ventricular septal ruptures complicate 1%–2% of MI in prethrombolytic era^[3] and account for 5% of all peri-infarction deaths. There is a 10-fold decrease today due to implementation of early thrombolysis and myocardial salvage.^[4] It usually occurs between 2 and 6 days but can be anytime within first 2 weeks following an acute infarction.^[5] The median time from the onset of infarction to septal rupture was 1 day in the GUSTO-I trial^[6,7] and 16 h in the SHOCK trial.^[8] The onset and severity of symptoms depend on the size of defect, and without reperfusion, the coagulation necrosis develops with disintegration of adjacent necrotic myocardium around the defect.^[9,10] This complication is usually associated with total occlusion of a coronary artery leading to infarction of the interventricular septum. The operative mortality depends on status of preoperative hemodynamics, which in turn related to the degree of shunting and extent of the acute infarct.[11] In surgically treated patients, the 30-day and the 1-year mortality rate was 47% and 54%, respectively, whereas in medically treated, this was 94% and 97%.^[12] Hence, surgical repair therefore is the treatment of choice. Other risk factors are old age, location of MI, urgent surgery, female gender, EF <40%, previous cardiac surgery and/or MI, lack of improvement in hemodynamic status in spite of inotropic support, shorter period from infarction to surgery, total occlusion of the infarcted artery, right ventricular dysfunction, and pathologically complex type. All our patients apart from the patient B in the current case series belong to high-risk category with multiple comorbidities. This case series has shown that implementation of preoperative optimization prior to surgery, appropriate selection and timing for an operative procedure or any interventions including ECMO may possibly contribute to better outcome in high risk patients.

All the operations in our institution were carried out under CPB with moderate hypothermia. Preoperatively, nitroglycerine infusion was initiated depending on hemodynamic stability with a view to reduce afterload and left to right shunt systemic blood flow and to improve coronary perfusion. Induction of general anesthesia is particularly challenging as any increase in systemic vascular resistance (SVR) can increase the shunt flow and the myocardial oxygen demand. Another concern is to maintain optimal perfusion pressure while lowering the afterload and also to maintain the SVR/pulmonary vascular resistance (PVR) ratio so as to reduce the shunt fraction.

The postoperative management also should be focused on to prevent tension on VSD repair sutures. We prefer central cannulation as in patient A to keep the heart decompressed and plan to switch over to femoral at a later date to prevent tension on suture line of newly repaired septum. Our aim is to reduce LV afterload and at the same time to maintain hemodynamics during the postbypass period. We ventilate these patients at an early stage to achieve hypocarbic alkalosis and good oxygenation that reduces PVR and improves intrapulmonary shunt. Other considerations are avoiding hypoxia or hyperoxia and myocardial depression.

The ideal timing of VSD repair is a point for debate.^[13] Most of the guidelines recommend immediate surgical intervention to prevent further hemodynamic deterioration whereas few other studies support our current strategy of delaying surgery if feasible for optimization with medical management before surgery. Pang *et al.*^[14] found only 2 of 38 patients remained fit for delayed surgery and concluded that it can be applied to only for selected group of patients as the friable VSD enlarges during the first 10 days, and waiting for myocardial maturation is not entirely feasible option. An earlier study showed better survival rate with early surgical repair,^[15] and this

in agreement with SHOCK trial that reported an increased mortality with medical management. However, subjecting patients for delayed surgery for perioperative optimization and mechanical support may allow myocardial scar tissue formation and facilitate the technical aspects of VSD repair or an early percutaneous treatment may be an alternative option. Delaying surgery may present these patients with improved and better organ function and made to undergo a relatively lower risk procedure at a later stage after preload reduction and preoperative optimization as in patient D that could be the possible reason for his shortened postoperative intensive care stay. However, studies also have shown that the prolonged medical management at times is risky and even futile^[3] in few situations.

Ventricular assist devices (VADs) have been shown to be beneficial as a bridge to surgery or are placed postoperatively to allow for restoration of peripheral organ perfusion and provide recovery and maturation of the infarcted myocardium. By decreasing afterload and preload, VADs can help to provide the rest and increase coronary perfusion to the shocked myocardium. Blanche et al.[16] found that postoperative use of an IABP in acute MI reduces immediate postoperative mortality but does not improve long-term survival. acute MI reduces immediate postoperative mortality but does not improve long-term survival. The management of our patient A is supported by previous case reports.^[17] The authors used IABP and venoarterial ECMO to stabilize their patient and subsequently repaired the ruptured ventricular septum. The percutaneous ECMO also provides numerous benefits compared to conventional VADs, as they are economically efficient, prevent sternotomy, provide oxygenation support, and are easily reversible.

Recently, the percutaneous closure devices have permitted less invasive management of patients with post-MIVSR.^[18] A questionnaire search using thirty-one best evidence papers showed that the insertion of an occluder device in patients with a postinfarction VSD not amenable to surgical repair offers benefit only in selected patients. The authors concluded that device closure technique might avoid the surgical closure. In few selected patients, they may provide the time for VSD to mature and optimize the patient acting as a bridge prior to surgery to offer the best possible outcome in this group of patients. The current evidence suggests that in patients with defects <1.5 cm, subacute stage and for patients who are poor surgical candidate, the percutaneous intervention is a more attractive option than conventional surgery.^[19] More recently, in a series of 29 patients with percutaneous option, 41% experienced procedure-related complications, and the overall 30-day survival rate was 35% with higher mortality in patients with cardiogenic shock.^[20] Although it appears to be an attractive option, they are more suitable for congenital VSDs that are muscular, smaller with well-defined and healthy edges. . Not only are the postinfarction VSDs larger but also their

Table 1: Pre-operative clinical presentation and post-operative status of studied patients											
Patient	Age	Pre-operative status					Post-operative status				
		Inotropes	Ventilation	IABP	ЕСМО	Surgical priority	Ventilation days	ICU days	ЕСМО	Percutaneous intervention	Survival at 60 days
Patient (A)	58	++++	Yes High FiO,	Yes	Yes	Е	6	17	Yes	No	Alive
Patient (B)	53	_	No	Yes	No	U	4	2	No	No	Alive
Patient (C)	56	+++	No	Yes	No	U	1	11	No	Yes	Alive
Patient(D)	64	+ (Weaning)	Yes. Minimal support FiO 04	Yes	No	$\mathbf{P}.\mathbf{S}^{\times}$	3	8	No	No	Alive

++++ high, +++ moderate, + minimal, - No inotropes, E: Emergency, U: Urgent, P.S[×]: Post stabilization with medicine/mechanical support

rim consists of unhealthy infarcted tissue that degenerates over time and increases the VSD size. This phenomenon also increases the likelihood of device dislodgment and malposition. This technique offer some advantage of being less invasive and an option of "bridge" for a definitive procedure. It may improve the pulmonary haemodynamics and allow the myocardium to heal and fibrose so as to facilitate better surgical repair and improve the outcome. However, significant advances need to be made in the device design, and further studies are needed to validate the durability of the percutaneous approach and to compare the results with surgical repair.

The echocardiography has been a very useful tool for diagnosing interventricular septal rupture easily in patients with acute MI. It is recommended that every new onset or progression of right or left heart failure following MI should lead to further diagnostic procedures including immediate echocardiography.^[21] Recently, the real-time 3-dimensional TEE plays a key role in guiding the device placement both during percutaneous and the surgical management. It also helps by providing reliable information about the size of the VSD and quantification of the initial and the residual left-to-right shunt through the defect.^[22]

Our case series is limited by the relatively short follow-up period and may not provide the overall information on the long-term outcome and the durability of surgical closure of post-MIVSR. A single-center study^[13] with 32 patients during their 22 years of experience showed an early mortality rate 20%, 26% had residual shunt, and five patients required reoperation. The overall 5- and 10-year survival was 55% and 44%, respectively. The recurrence rates of VSD after the initial surgical repair of postinfarct VSD have been reported to range from 10% to 44%.^[23] Furthermore, the posterior location of VSD has been found to be associated with poor prognosis in most of the previous studies.^[24] Furthermore, a retrospective multicenter study^[25] showed that patients who underwent early VSD closure had higher rates of residual shunt and mortality. In addition, the mortality rate was higher after early percutaneous closure. The all-cause mortality was 40%, and the authors recommended a vigorous pursuit of closure of post-MI VSD with a sequential surgical and/or percutaneous approach for improved outcomes. A long-term follow-up study^[26] involving 35 patients

showed 30-day mortality of 31%, and the actual survival was 66%, 62%, and 58% at 1 year, 3 years, and 9 years, respectively. To improve the surgical results, the double patch technique and glue have been recommended^[27] and shown to avoid the recurrence and reduction of the hospital mortality. We have employed this technique in all our patients. Generally, the NYHA class at presentation and postoperative continuous renal replacement therapy are predictors of early mortality, and the age and time between MI and operation are independent predictors of 30-day and long-term mortality.^[14]

The present case series depicts the various forms of clinical presentation of postinfarction VSD. The outcome can be improved in selected group of patients with sensible preoperative preparation and aggressive perioperative management as shown in videos 1-10 and the outcomes as shown in Table 1. However, we need a large prospective multicenter data to verify the results and long-term outcome of these patients. Furthermore, these patients that have multiple comorbidity with undergoing these complex procedures may need to be referred to experienced centers with mechanical support including ECMO with the caution that some patients may be too unstable to be transferred to referral units.

Conclusion

In this case series, we have presented four different ways of clinical presentation of postinfarction ventricular septal defect. The overall outcome gives an idea of how the timely and careful perioperative planning, intensive medical treatment, and support of organ can be effective in improving the outcome of these patients.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patients have given their consent for 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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Conflicts of interest

There are no conflicts of interest.

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