Original Article

Perioperative Challenges and Outcome After Surgical Correction of Post-myocardial Infarction Ventricular Septal Rupture: A Retrospective Single Center Study

Kedar Bangal

Narayana Institute of Cardiac Sciences, Narayana Health City, Bengaluru, Karnataka, India

ABSTRACT

Background: Ventricular septal rupture (VSR) is a rare but grave complication of acute myocardial infarction (AMI). It is a mechanical complication of myocardial infarction where patients may present either in a compensated state or in cardiogenic shock. The aim of the study is to determine the in-hospital mortality. The study also aims to identify the predictors of outcomes (in-hospital mortality, vasoactive inotrope score (VIS), duration of ICU stay and mechanical ventilation in the postoperative period) and compare the clinical and surgical parameters between survivors and non-survivors.

Methods: This is a retrospective study. The data of 90 patients was collected from the medical records and the data comprising of 13 patients who underwent VSR closure by single patch technique, or septal occluder, and those who expired before receiving the treatment, was excluded. The data of 77 patients diagnosed with post-AMI VSR and who underwent surgical closure of VSR by double patch technique was included in this study. Clinical findings and echocardiography parameters were recorded from the perioperative period. The statistical software used was SPSS version 27. The primary outcome was determining the in-hospital mortality. The secondary outcome was identifying the clinical parameters that are significantly more in the non-survivors, and the factors predicting the in-hopsital mortality and morbidity (increased duration of ICU stay, and of mechanical ventilation, postoperative requirement of high doses of vasopressors and inotropes). Subgroup analysis was done to identify the relation of various clinical parameters with the postoperative complications. The factors predicting the in-hospital mortality were illustrated by a forest plot.

Results: The mean age of the patients was 60.35 (±9.9) years, 56 (72.7%) were males, and 21 (27.3%) were females. Requirement of mechanical ventilation preoperatively (OR 3.92 [CI 2.91-6.96]), cardiogenic shock at presentation (OR 4 [CI 2.33 – 6.85]), requirement of IABP (OR 2.05 [CI 1.38-3.94]), were predictors of mortality. The apical location of VSR had been favorable for survival. The EUROScore II at presentation correlated with the postoperative VIS (level of significance [LS] 0.0011, R 0.36. The in-hospital mortality in this study was 33.76%.

Conclusion: The in-hospital mortality of VSR is 33.76%. Cardiogenic shock at presentation, non-apical site of VSR, preoperative requirement of mechanical ventilation, high VIS preoperatively, perioperative utilization of IABP, prolonged CPB time, postoperative duration of mechanical ventilation, and high postoperative VIS were the factors associated with increased odds of in-hospital mortality.

Keywords: Cardiogenic, coronary artery disease, echocardiography, Myocardial infarction, shock, vascular resistance, ventricular septal rupture

Address for correspondence: Dr. Kedar Bangal, Department of Cardiac Anesthesiology, Narayana Institute of Cardiac Sciences, Bengaluru - 560 099, Karnataka, India.
E-mail: kedar4xcell@gmail.com

Submitted: 14-May-2023 **Revised:** 13-Jul-2023 **Accepted:** 24-Jul-2023 **Published:** 12-Jan-2024

Access this article online	
Quick Response Code:	Website: https://journals.lww.com/aoca
	DOI: 10.4103/aca.aca_75_23

How to cite this article: Bangal K. Perioperative challenges and outcome after surgical correction of post-myocardial infarction ventricular septal rupture: A retrospective single center study. Ann Card Anaesth 2024;27:17-23.

INTRODUCTION

Ventricular septal rupture (VSR) is a rare but grave complication of acute myocardial infarction (AMI). In the post-thrombolysis (reperfusion) era this condition had an incidence of 0.2% and had a mortality rate of 45% to 90%.[1-3] The urgent surgical closure of VSR is a class 1 indication of the American College of Cardiology Foundation/American Heart Association (ACCF/AHA) guidelines.[4] VSR is a condition that results in left to right shunt and depends on the relative resistances of systemic and pulmonary circulation. There is resultant pulmonary over-circulation, reduced cardiac output, systemic hypotension, and organ hypoperfusion. Total occlusion of the infarct-related artery leads to VSR.[5,6] It is critical to understand the factors that predict in-hospital mortality and morbidity (extended ICU stay and prolonged ventilatory support, necessity of high doses of vasopressors and inotropes postoperatively), as well as the factors that are more prevalent in nonsurvivors.

METHODS

The study was approved by the institutional review board (IRB), and the data was accessed from the medical records. Informed consent was waived by the IRB. The study design is illustrated in a flowchart in Figure 1. Arterial cannulation and central venous cannulation were utilized for hemodynamic monitoring. Pulmonary artery catheter (PAC) was not utilized for all the cases. According to clinician's discretion, PAC was inserted in the intra-operative or postoperative period, to manage hemodynamics. Hence, PAC derived data was not included in this study. Patient demography, clinical presentation, anesthesia management, surgical technique, postoperative course, and in-hospital mortality were reviewed. All patients received balanced general anesthesia (intravenous[IV] Fentanyl, IV Etomidate, IV Vecuronium/Pancuronium/Atracurium) with endotracheal intubation, and maintenance with Sevoflurane/Isoflurane. FiO2 was set at 60% and increased if necessary to maintain arterial oxygen saturation >90%, EtCO2 levels were maintained between 35 to 45 mmHg, and mean arterial pressure (MAP) was targeted > 65 mmHg. The vasoactive inotrope score (VIS) was calculated as: Inj. Dopamine dose (μg/kg/min) + Inj. Dobutamine dose (μg/kg/min) +100 × Inj. Epinephrine dose (μg/kg/min) +10 × Inj. Milrinone dose (μg/kg/min) +10 000 × Inj. Vasopressin dose (unit/kg/min) +100 × Inj. Norepinephrine dose (μg/kg/min). This study has been approved by the institutional ethics committee named, 'Narayana Health Academic Ethics Committee (NHAEC)', on the 30th November 2020. The NHAEC is registered under DCGI with EC registration number, 'ECR/772/Inst/KA/2016/RR-19' under rules 122DD of the Indian Drugs and Cosmetics Rules 1945. The number allocated to my study is NHH/AEC-CL-2020-583.

Unfractionated Heparin 400 units/kg IV was administered to target an activated clotting time (ACT) of 480 seconds. Cardiopulmonary bypass (CPB) was performed with bi-caval cannulation of the superior and inferior vena cava, and arterial return cannula was inserted in the aorta. Systemic hypothermia was instituted to maintain the core temperature between 30°C and 32°C. During normothermia, the flows of 2.4 L/min/m^2 were maintained and the flows of 2 – 2.4 L/min/m2 were maintained during mild to moderate hypothermia. The cardioplegia used was St Thomas. During CPB period, alpha-stat pH management was employed. De-airing was done by venting the aortic root cardioplegia cannula connected to cardiotomy suction, with gentle bag ventilation. The hematocrit was maintained above 25% while coming off CPB.

Postoperatively, all the patients were electively ventilated and received vasoactive and inotropic infusions in the intensive care unit (ICU). Patients were extubated when adequate recovery was achieved.

Statistical analysis was performed by SPSS version 27. Categorical variables were expressed as percentages and continuous variables as mean±SD. Chi-square test was used to compare categorical variables, and Student's *t*-test was used for continuous variables. The clinical parameters that are significantly more among non-survivors were identified. Univariate and multivariate analysis was performed to identify independent predictors of mortality, postoperative duration of mechanical ventilation, and ICU stay, postoperative VIS. Odds ratios with confidence intervals were calculated to identify the relationship with in-hospital mortality. Subgroup analysis was performed by the Cohen's d test to analyze the effect size of the statistically significant parameters among survivors and non-survivors by calculating the number of standard deviations between two means. The Cohen's d value above 0.8 is very significant, 0.5 to 0.8 is moderately significant, and from 0.2 to 0.5 is mildly significant. Subgroup analysis was performed for identifying the relation between single, double, or triple vessel disease with in-hospital mortality, and to identify the relation of IABP utilization with in-hospital mortality.

RESULTS

The data of 77 patients who underwent VSR closure by

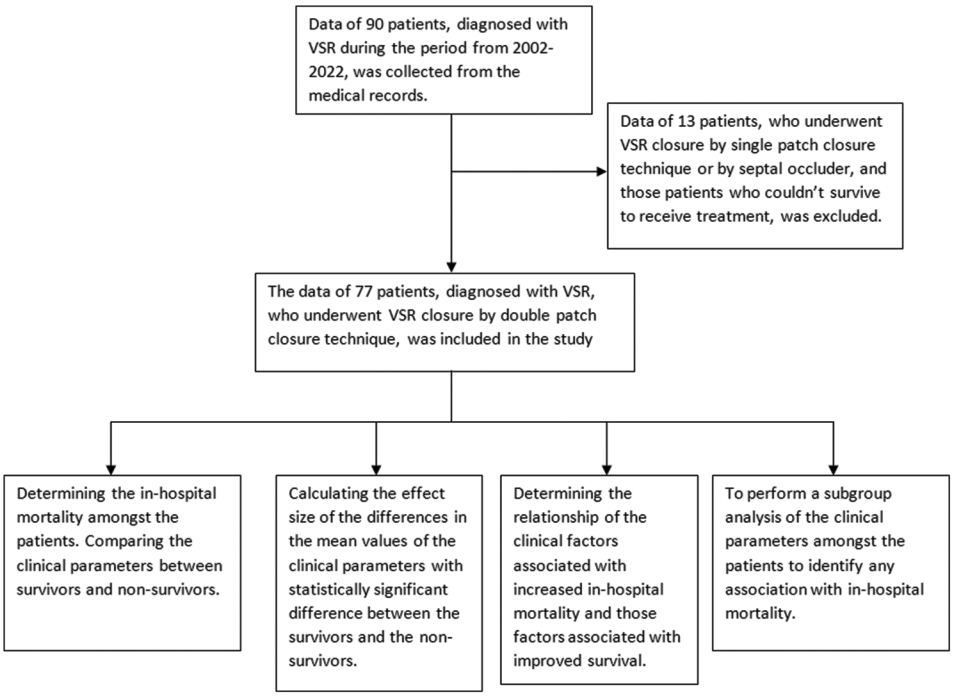

Figure 1: Flowchart illustrating the study design

double patch closure technique was analyzed retrospectively. Some patients had required concomitant coronary artery bypass graft (CABG), and Dor procedure (repair of left ventricular [LV] aneurysm by circular sutures and Teflon patch). The mean age of the patients was 60.35 (±9.9) years, 56 (72.7%) were males, and 21 (27.3%) were females. The patient demography and clinical parameters are presented in Table 1. The in-hospital mortality was 33.76%.

The difference in the period between the diagnosis of VSR and surgery was statistically not significant between survivors and non-survivors (p value 0.056). The number of patients requiring high VIS (p value 0.04) and mechanical ventilation preoperatively (p value 0.02) were more among non-survivors. There was no difference in the size of VSR among survivors and non-survivors (p value 0.49). The duration of CPB was significantly more among non-survivors (p value 0.009, effect size [ES] 0.68). In the postoperative period, the VIS was significantly more among non-survivors compared to survivors. The duration (days) of postoperative mechanical ventilation was significantly more among non-survivors (p value 0.02, ES 0.62) compared to survivors. Performing CABG had improved survival (0.0072 Chi-square test, OR (CI): 1.42 [1.05-2.1]).

The EUROScore II at presentation correlated with the postoperative VIS (level of significance [LS] 0.0011, R 0.36). The sites of VSR other than apical had a good association with in-hospital mortality (LS 0.021, R 0.32). On performing subgroup analysis, it was found that the requirement of vasopressors and inotropes preoperatively (p value 0.00014), and mechanical ventilation in the preoperative period (p value 0.0129) had an association with in-hospital mortality. The number of affected coronaries (single vessel [p value 0.92], double [p value 0.89], triple [*P* value 0.92]) had no correlation with in-hospital mortality. On performing univariate analysis, the perioperative IABP requirement was found to be a predictor of the in-hospital mortality [p value 0.041; R 0.23 Table 3]. The factors having association with the outcomes (in-hospital mortality, postoperative duration of mechanical ventilation and ICU stay, postoperative requirement of vasopressors and inotropes) are described in Table 3. The odds ratios and confidence intervals of the factors predicting the in-hospital mortality are illustrated in Figure 2 by means of a forest plot.

DISCUSSION

In this study, it was found that the patients presented with a wide spectrum of signs and symptoms, ranging from angina of variable intensity, tachycardia, hypotension, dyspnea (NYHA grade III/IV), atrial fibrillation, pulmonary edema, and cardiogenic shock. VSR was present in the apical, anteroseptal, and inferobasal regions of the interventricular septum. The patients requiring mechanical ventilation and high VIS in the preoperative

Table 1: Demography and clinical findings

Parameters	Number of patients [*n*%; (Mean±SD)]	
Age (years)	60.35±9.9	
Gender: Male/Female	56 (72.7%)/21 (27.3%)	
Weight (kg)	62.26±10.2	
Period between VSR diagnosis and surgery (days)	2.0±5.5	
Hypertension/diabetes	39 (50.6%)/45 (58.4%)	
CAD (old/recent/acute)	18 (23.4%)/32 (41.6%)/27 (35.1%)	
History of PTCA/thrombolysis	9 (11.7%)/17 (22.1%)	
Preoperative heart rate (beats/min)	99.97±20.3	
Preoperative SBP (mmHg)	111.25±24.8	
Preoperative DBP (mmHg)	70.25±14.8	
EUROScore II	13.0±25.3	
Killip class	**Total cases**	**Mortality**
Class 1 (*n*,%)	19 (24.7%)	3 (15.78%)
Class 2 (*n*,%)	23 (29.9%)	5 (21.73%)
Class 3 (*n*,%)	14 (18.2%)	6 (42.85%)
Class 4 (*n*,%)	21 (27.3%)	12 (57.14%)
Preoperative mechanical ventilation	17 (22.1%)	
Preoperative inotropes score	7.6±5.9	
Preoperative EF	41.97±6.7	
VSR size (mm)	8.5±7.0	
RWMA	45 (58.4%)	
Grades of MR: Mild/Moderate	46 (63.63%)/8 (10.38%)	
IABP: Preoperatively/Intraoperatively/Not utilized	44 (59.74%)/5 (6.5%)/28 (36.36%)	
Single/double/triple vessel coronary disease	28 (36.38%)/16 (20.77%)/33 (42.85%)	
PASP (mmHg)	42.74±18.9	
Cardiopulmonary bypass time (minutes)	153.48±70.5	
Aortic cross-clamp time (minutes).	90.0±40	
Postoperative SBP/DBP (mmHg)	103.3±18.7/56.25±14.7	
Postoperative heart rate/min	100.74±14.5	
Postoperative inotrope score	17.1±12.6	
Re-exploration/delayed chest closure/others	10 (12.98%)/6 (7.79%)/1 (1.29%)	
Postoperative RWMA	40 (51.95%)	
Presence of residual shunt (TEE)	21 (27.27%)	
Postoperative EF (TEE)	41.76±6.4	
ICU stay (days)	10.0±10.2	
Mechanical ventilation (days)	3.0±8.5	
Survivors/non-survivors	51 (66.24%)/26 (33.76%)	

*VSR – ventricular septal rupture, CAD – coronary artery disease, PTCA – percutaneous transluminal coronary angioplasty, SBP – systolic blood pressure, DBP – diastolic blood pressure, EF – ejection fraction, RWMA – regional wall motion abnormality, MR – mitral regurgitation, IABP – intra-aortic balloon counter-pulsation, PASP – pulmonary artery systolic pressure, TEE – trans-esophageal echocardiography, ICU – intensive care unit

period were more among non-survivors. The apical site of VSR favored survival, whereas other sites had a significant correlation with the in-hospital mortality. The size of VSR had no relation with the in-hospital mortality, postoperative VIS, duration of ICU stay and postoperative mechanical ventilation. The factors like cardiogenic shock at presentation, requirement of mechanical ventilation preoperatively, requirement of IABP [Figure 2], longer CPB duration, higher postoperative VIS, increased duration of mechanical ventilation postoperatively [Table 2], a high VIS preoperatively, [Table 3] were significantly more among non-survivors. Size of VSR had no influence on the in-hospital mortality, duration of postoperative mechanical ventilation and ICU stay, and postoperative VIS. EUROScore II at presentation had a relation with postoperative VIS. The number of affected coronary arteries had no relation with the in-hospital mortality. The requirement of mechanical ventilation and vasopressor and inotropes preoperatively had a strong association with in-hospital mortality. In our study, the requirement of IABP was associated with an increased risk of in-hospital mortality, but there was no difference in the outcomes among the subgroup of patients requiring IABP preoperatively versus intraoperatively. Concomitant CABG was associated with improved survival in our study.

According to a study done by Skehan *et al.*,[6] VSR develops in 10 to 14 days after AMI, but Birnbaum *et al.*,[7] have stated in their study that VSR may also occur at 2 to 4 weeks after myocardial infarction. Early and late repair, both, have been practiced for the surgical closure of VSR. Delayed repair is associated with improved outcome but is suitable for hemodynamically stable patients with evolved infarcts, whereas early repairs are performed in hemodynamically unstable patients.[8] In our study, early or late repair of VSR was not a predictor of in-hospital mortality VSR

Table 2: Comparison of the clinical parameters between survivors and non-survivors

Parameters (Mean±SD)	Outcome (Mean±SD)		*P*
	Survivors (*n*=51)	Non-survivors (*n*=26)	
Period between VSR diagnosis and surgery (days)	2.0±7.0	1.0±1.5	0.056
Preoperatively mechanical ventilation	7 (9.09%)	10 (12.98%)	0.02*; ES 0.28
Preoperative VIS	5.0±6.1	10.0±12.6	0.04*; ES 0.86
Size of VSR (mm)	9.5±7	7.5±7.2	0.49
Mitral regurgitation			
Present	40 (51.94%)	14 (18.18%)	
Absent	16 (20.77)	7 (9.09)	
Number of coronaries involved:			
Single,	19 (24.67%)	9 (11.68%)	
Double,	10 (12.98%)	6 (7.79%)	
Triple.	21 (27.27%)	12 (15.58%)	
Time of cardiopulmonary bypass (minutes).	138.54±57.6	184.65±84.8	0.009*; ES 0.68
Time of aortic cross-clamp (minutes)	92.00±37.8	114.30±62.1	0.16
Postoperative VIS	14.96±6.4	28.28±13.3	0.001*; ES 1.412
Length of ICU stay (days)	10.0±9.2	8.5±15.2	0.42
Postoperative mechanical ventilation (days)	2.0±2.0	7.5±15	0.02*; ES 0.62
CABG performed	26 (33.76%)	11 (14.28%)	0.0072 Chi-square test, OR (CI): 1.42 (1.05-2.1)
CABG not performed	25 (32.46%)	15 (19.48%)	

*Statistically significant difference, VIS – vasoactive inotropic score; OR- odds ratio; CI - confidence interval; ES – effect size

Table 3: Association of the clinical factors with the outcomes. Subgroup analysis to determine the association with in-hospital mortality

Association of the clinical factors with the outcomes. Subgroup analysis to determine the association with in-hospital mortality	Regression analyses; Level of significance (correlation coefficient "R"); *P* (Chi-square test)
Size of VSR and in-hospital mortality	0.61 (R [-0.0029])
Size of VSR and postoperative VIS	0.45 (R [-0.58])
Size of VSR and duration of ICU (days)	0.59 (R [-0.02])
Size of VSR and duration of postoperative mechanical ventilation	0.89 (R [-0.011])
EUROScore II and postoperative VIS	0.0011 (R 0.36)
Single vessel disease and in-hospital mortality (subgroup analysis)	0.92 (R -0.021)
Double vessel disease and in-hospital mortality (subgroup analysis)	0.89 (R -0.04)
Triple vessel disease and in-hospital mortality (subgroup analysis)	0.92 (R 0.018)
Anterior and inferior wall MI and survival (multivariate)	0.39 (R [-0.11])
Preoperative versus intraoperative IABP use and in-hospital mortality (subgroup analysis)	*P*=0.79 (Chi-square test)
Requirement of vasopressors and inotropes preoperatively, and in-hospital mortality (subgroup analysis)	*P*=0.00014, (Chi-square test)
Requirement of mechanical ventilation preoperatively and in-hospital mortality (subgroup analysis)	*P*=0.0129 (Chi-square test)

*VIS – vasoactive inotrope score, IABP – intra-aortic balloon counter-pulsation

causes a sudden onset left to right shunt, concurrent mitral regurgitation causes volume loading of the LV, and the RV is subjected to systemic pressures. Thus, the LV is subjected to an excess preload.

2D echocardiography with Doppler is mandatory to be done in patients with a high suspicion of VSR to locate the site, size, left to right (L→R) shunt magnitude, mitral regurgitation (MR), and to estimate biventricular function.[9,10]

VSR management entails reducing the amplitude of the LR shunt by avoiding hypocapnia and prudent afterload reduction, which also reduces the MR fraction (in cases where there is concomitant MR). There is optimization of forward cardiac output (CO).[11] VSR needs urgent surgical correction.[4] David infarct exclusion technique is most widely used and is proven to be better than infarctectomy technique.[12] There are several techniques of VSR closure comprising of single, double, and triple patch closure, but the durability of repair is less with single patch closure technique.[13] Double patch closure technique with infarct exclusion has lesser probability of patch dehiscence, residual shunt, better durability, and maintains LV geometry.[14] Matteucci *et al.*,[15] performed a meta-analysis of 41 studies, which included 6361 patients and found that the patients requiring IABP perioperatively and posterior location of VSR had increased odds of in-hospital mortality. They have mentioned the probable reason behind this to be the preoperative poor clinical status of such patients. Contradictory to our study, they have found that there was no protective effect of concomitant CABG. Their operative mortality was 38.2%. Fifteen studies comprising of 2312 patients were evaluated for the temporal trend in the operative mortality. The

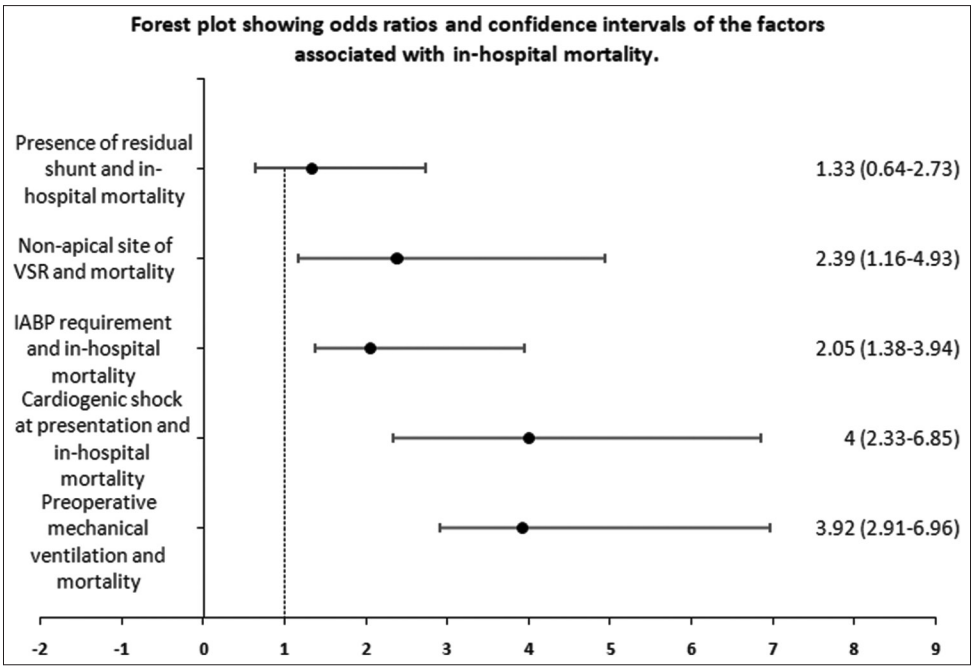

Figure 2: Forest plot showing odds ratios and confidence intervals of the factors associated with in-hospital mortality

authors found that in both the time frames (first time frame of 21 years and the second time frame of 18 years), the operative mortality was 34%. In our study, the in-hospital mortality was 33.76%. Our findings are consistent in terms of the outcomes of patients with nonapical VSR and perioperative IABP requirement.

Mortality in this study is similar to previous studies.[16-19] Similar to our study findings, Lundblad *et al.*,[20] and Perrota *et al.*,[21] have found that mortality is reduced by concomitant CABG.

Single vessel disease was the most prevalent coronary artery disease followed by double vessel disease and triple vessel disease in a study by Malhotra *et al.*,[22] and Labrousse *et al.*[23]

The exact time of occurrence of AMI could not be included in this study due to its retrospective nature. PAC derived data regarding mixed venous saturation could have helped identify magnitude of L→R shunt, systemic, and pulmonary vascular resistances in the operation theatre, which guide hemodynamic management, but PAC was not utilized for all cases. The long-term outcomes of the patients were not analyzed in this study.

CONCLUSION

The in-hospital mortality of VSR was 33.76%. Cardiogenic shock at presentation, non-apical site of VSR, preoperative requirement of mechanical ventilation, high VIS preoperatively, perioperative utilization of IABP, prolonged CPB time, postoperative duration of mechanical ventilation, and high postoperative VIS were the factors associated with increased odds of in-hospital mortality. More studies are required to compare the outcomes of interventional management of VSR with surgical management. Large sample size studies and meta-analyses are required to gain further knowledge about VSR.

Financial support and sponsorship
Nil.

Conflicts of interest
There are no conflicts of interest.

REFERENCES

1. Crenshaw BS, Granger CB, Birnbaum Y, Pieper KS, Morris DC, Kleiman NS, *et al.* Risk factors, angiographic patterns, and outcomes in patients with ventricular septal defect complicating acute myocardial infarction. GUSTO I (Global Utilization of Streptokinase and TPA for Occluded Coronary Arteries) trial investigators. Circulation 2000;101:27-32.
2. Gusto investigators. An international randomized trial comparing four thrombolytic strategies for acute myocardial infarction. N Engl J Med 1993;29:673-82.
3. GUSTO Angiographic investigators. The effects of tissue plasminogen activator, streptokinase or both on coronary artery patency, ventricular function, and survival after acute myocardial infarction. N Engl J Med 1993;329:1615-22.
4. O'Gara PT, Kushner FG, Ascheim DD, Casey DE Jr, Chung MK, de Lemos JA, *et al.* 2013 ACCF/AHA guideline for management of ST-elevation myocardial infarction: A report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. J Am Coll Cardiol 2013;61:e78-140.

5. Topaz O, Taylor AL. Interventricular septal rupture complicating acute myocardial infarction: From pathophysiologic features to the role of invasive and non-invasive diagnostic modalities in current management. Am J Med 1992;93:683-8.
6. Skehan JD, Carey C, Norrel MS, de Belder M, BalconR, Mills PG. Patterns of coronary artery disease in post-myocardial infarction ventricular septal rupture. Br Heart J 1989;62:268-72.
7. Birnbaum Y, Fishbein MC, Blanche C Siegel RJ. Ventricular septal rupture after acute myocardial infarction. N Engl J Med 2002;347:1426-32.
8. 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.
9. Smyllie JH, Sutherland GR, Geuskens R, Dawkins K, Conway N, Roelandt JR. Doppler color flow mapping in the diagnosis of ventricular septal rupture and acute mitral regurgitation after myocardial infarction. J Am Coll Cardiol 1990;15:1449-55.
10. Fortin DF, Sheikh KH, Kisslo J. The utility of echocardiography in the diagnostic strategy of post-infarction ventricular septal rupture: A comparison of two dimensional echocardiography versus Doppler color flow imaging. Am Heart J 1991;121:25-32.
11. Wang W, Cheung A. Mechanical complications of acute myocardial infarctions. In: Watson T, Ong P, Tcheng J, editors. Primary Angioplasty. Singapore: Springer; 2018. Print ISBN 978-981-13-1113-0. Online ISBN 978-981-131114-7.
12. Lundblad R, Abdelnoor M. Surgery of postinfarction ventricular septal rupture: The effect of David infarct exclusion versus Daggett direct septal closure on early and late outcomes. J Thorac Cardiovasc Surg 2014;48:2736-42.
13. Kim IS, Lee JH, Lee DS, Cho YH, Kim WS, Jeong DS, *et al*. Surgical outcomes of a modified infarct exclusion technique for post-infarction ventricular septal defects. Korean J Thorac Cardiovasc Surg 2015;48:381-6.
14. Parachuri VR, Tripathy AK, Gaikwad NM, Singh AP, Mahajan V, Niranjan S. Modified infarct exclusion technique for repair of postinfarction ventricular septal rupture. Ann Thorac Surg 2019;107:e219-21.
15. Matteucci M, Ronco D, Corazzari C, Fina D, Jiritano F, Meani P, *et al*. Surgical repair of postinfarction ventricular septal rupture: Systematic review and meta-analysis. Ann Thorac Surg 2021;112:326-37.
16. 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.
17. Hirata N, Sakai K, Ohtani M, Nakano S, Matsuda H. Assessment of perioperative predictive factors influencing survival in patients with posy-infarction ventricular septal perforation. Classified by the site of myocardial infarction. J Cardiovasc Surg (Torino) 2000;41:547-52.
18. Mantovani V, Mariscalco G, Leva C, Blanzola C, Sala A. Surgical repair of post-infarction ventricular septal defect: 19 years of experience. Int J Cardiol 2006;108:202-6.
19. Coskun KO, Coskun ST, Popov AF, Hinz J, Schmitto JD, Bockhorst K, *et al*. Experiences with surgical treatment of ventricular septal defect as a post infarction complication. J Cardiothorac Surg 2009;4:3.
20. Lundblad R, Abdelnoor M, Geiran OR, Svennevig JL. Surgical repair of post-infarction ventricular septal rupture risk factors of early and late death. J Thorac Cardiovasc Surg 2009:137:862-8.
21. Perrota S, Lentini S. In-patients undergoing surgical repair of post infarction ventricular septal defect, does concomitant revascularization improve prognosis? Interact Cardiovasc Thorac Surg 2009;9:879-87.
22. Malhotra A, Patel K, Sharma P, Wadhawa V, Madan T, Khandeparkar J, *et al*. Techniques, timing and prognosis of post infarct ventricular septal repair: A re-look at old dogmas. Braz J Cardiovasc Surg 2017;32:147-55.
23. Labrousse L, Choukroun E, Chevalier JM, Madonna F, Robertie F, Merlico F, *et al*. Surgery for post infarction ventricular septal defect (VSD): Risk factors for hospital death and long term results. Eur J Cardiothorac Surg 2002;21:725-31.