Section: Cardiovascular and Thoracic Surgery

Systematic Review

OUTCOMES OF EARLY VENTRICULAR SEPTAL SURGICAL REPAIR IN PATIENTS WITH POST-MYOCARDIAL INFARCTION: A SYSTEMATIC REVIEW

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Abstract

Background: Post-myocardial infarction ventricular septal rupture (VSR) poses a significant clinical challenge, with high mortality rates. This systematic review and meta-analysis aimed to comprehensively evaluate surgical outcomes, risk factors, and associated factors in patients with post-MI VSR. **Material and Methods:** A systematic literature search was conducted in the PubMed, Scopus, Cochrane, and Google Scholar databases for studies published between 2015 and 2024. Fourteen studies comprising 1,380 patients were included in the analysis. Data on demographics, risk factors, anatomical considerations, and clinical outcomes were extracted and analysed. **Results:** The mean age of the cohort was 69.5 years, with a male predominance. Anterior and apical regions were predominantly affected, and cardiogenic shock, older age, smoking, and preoperative IABP usage were identified as significant risk factors for mortality. Early intervention following diagnosis is associated with improved survival rates. The mortality rate within 30 days of VSR closure ranged from 42.9% to 70%, with delayed closure, VSR diameter, and elevated serum creatinine levels contributing to adverse outcomes. **Conclusion:** Post-MI VSR remains a formidable challenge with high mortality rates, despite advancements in surgical techniques and perioperative care. Timely intervention and careful patient selection are crucial for optimising the outcomes in this patient population. Early intervention following diagnosis is associated with improved survival rates, emphasising the importance of timely surgical management.

INTRODUCTION

Ventricular septal defect (VSD) or ventricular septal rupture (VSR) is a significant complication of acute myocardial infarction (MI).[1] VSD manifests within the initial insult period, ranging from 3 days to two weeks post-MI, during ventricular remodelling. Localization of VSD or MI may transpire in the anterior or posterior wall or septum.[1,2] Concurrent risk factors for coronary artery disease, including smoking, dyslipidaemia, hypertension, and diabetes mellitus, have been identified to correlate with post-MI VSD, complicating the pathological condition.[3-6] However, the comprehensive impact of these risk factors on clinical outcomes of post-MI VSD remains unclear. Post-MI VSD is a fatal condition owing to its unfavourable prognosis.[1-4] Surgical intervention is considered the gold standard treatment for post-MI VSD.[7] Clinically, diverse techniques such as patch closure, infarct exclusion technique, and modified infarct exclusion technique are employed to correct post-MI VSD.[8-10] Disease recognition, maintenance of haemodynamic stability, and timely surgical intervention are pivotal determinants for the effective management of the condition.[11] Current guidelines advocate early and urgent repair of the defect to address haemodynamically unstable post-MI VSD patients.[12]

The timing from MI to VSD and VSD repair remains highly contentious. Delays in surgical repair are linked to adverse outcomes in unstable patients, resulting in elevated mortality rates for those awaiting surgical intervention in this subgroup. Conversely, the outcomes improve if surgery is performed after an acute episode.[13] In recent

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decades, the advent of various therapeutic approaches for post-MI VSD correction has substantially reduced the incidence of post-MI VSD from 3% to 0.2% in recent decades.[1,2,14] Despite this progress, the surgical management of post-MI VSD remains exceptionally challenging and is associated with exceedingly poor outcomes, including low postoperative cardiac output syndrome, low 30-day survival rates after surgery, and a high mortality rate. Treatment of post-MI VSD without surgical intervention may result in a mortality rate exceeding 90%.[15] Presently, limited knowledge exists regarding the overall effects of risk factors, VSD and MI localisation, surgical intervention with or without coronary artery bypass graft (CABG), and preoperative intra-aortic balloon pump (IABP) on the clinical outcomes of patients with post-MI VSD. Against this backdrop, this meta-analysis systematically reviewed the clinical and surgical experiences to elucidate the potential outcomes associated with post-MI VSD.

MATERIALS AND METHODS

Search Tool

The guidelines outlined in the Cochrane Handbook and Meta-analysis of Observational Studies in Epidemiology,[16] adhering to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) recommendations, were employed for this meta-analysis. A comprehensive literature search was conducted using PubMed, Scopus, Cochrane, and Google Scholar databases. The search utilised various combinations of keywords including "ventricular septal defect", "VSD", "VSR", "post-myocardial infarction", "post-MI VSD", "surgical outcomes", "post-MI VSR", and "ventricular septal rupture". Studies published in English between 2015 and 2024 were included in this meta-analysis. Exclusion criteria were case reports, expert opinions, literature reviews, editorials, and conference abstracts.

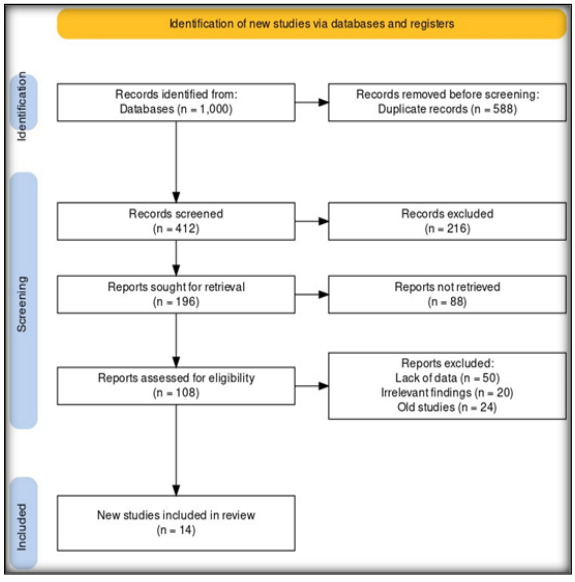

Figure 1: Consort Flow Diagram

Data Extraction

The following parameters were extracted: age, sex, preprocedural interventions such as PCI, Qp: Qs ratio, use of inotropes, ejection fraction, surgical procedures undertaken, duration from MI to VSD occurrence and from VSD detection to repair in days, presence of residual shunt, and identification of the affected coronary artery. The parameters analysed for the meta-analysis included the incidence of cardiogenic shock in post-MI VSD patients, anatomical localisation of MI, site of VSR, preoperative IABP usage, concomitant CABG, risk factors (prior MI, diabetes mellitus, smoking, and dyslipidaemia), 30-day survival, overall mortality, and mortality associated with concomitant CABG, location of MI, and site of VSR.

RESULTS

The review encompassed 14 studies conducted between 2000 and 2022, comprising a cohort of 1,380 patients who underwent VSR repair. Across these investigations, the mean age of patients was determined to be 69.5 years. Notably, males exhibited a predominant prevalence of VSR repair cases, constituting a cumulative incidence of 55.6%. The average time for VSD repair was 11 days based on all 14 studies. [Table 1]

Location of MI and VSR

The anterior and apical regions were affected in 235 cases (53.9%), with 149 instances (58.7%) occurring in the former and 86 cases (47.3%) in the latter (p = 0.02). Posterior involvement was observed in 201 cases (46.1%), with 105 occurrences (41.3%) attributed to this region and 96 cases (52.7%) to posterior localization.[17] In addition, a case series of eight patients reported that 62.5% of them presented with anterior wall myocardial infarction. All 9 patients presented with anterior MI in the case series by Shi et al,[[19] Among 126 patients included in the study by Heckel et al., 60 had anterior MI and 32 had posterior MI.[20] Wiemers et al. reported that posterior defects were more prevalent in patients (60%) than in anterior defects (40%).[21]

A 12-year study conducted by Yam et al. demonstrated a high incidence of anterior defects among 35 patients, whereas posterior defects were only reported in four patients.[22] Tang et al,[1] Isoda et al,[23] Aggarwal et al,[24] and Arnaoutak et al,[25] also reported a higher prevalence of anterior defects. In contrast, Sabiniewicz et al. showed a higher incidence of posterior defects among 12 patients when compared to anterior defects in only 8 patients.[26]

Risk factors associated with VSD

One of the prevalent risk factors is cardiogenic shock which was reported by Ronco et al,[17] in a recent study. A total of 44.8% of the patients presented with cardiogenic shock as a risk factor, of which 61.5% died. A significant association (p

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<0.001) was also reported between cardiogenic shock as one of the independent risk factors for VSD. In the univariate analysis, several factors were found to be associated with early mortality, including older age, smoking, chronic kidney disease, preoperative cardiogenic shock or cardiac arrest, percutaneous revascularization, preoperative need for intra-aortic balloon pump (IABP) or extracorporeal membrane oxygenation (ECMO), time from acute myocardial infarction (AMI) to surgery of less than 7 days, and urgent/emergent surgery (Table 1). For instance, patients experiencing cardiogenic shock had a lower survival rate (survival, 95 [33.6%]; death, 118 [61.5%]; p < .001), as did those who underwent early surgery (time to surgery <7 days) (survival: 105 [57.4%]; death: 47 [35.1%]; p < .001). Additionally, a significantly higher mortality rate was observed among patients with posterior ventricular septal rupture (VSR), longer cardiopulmonary bypass (CPB) and cross-clamp times, and those requiring thoracotomy for bleeding or postoperative support with IABP or ECMO.[17]

A similar finding was also reported by Naik et al., where cardiogenic shock was reported in 62.5% of patients and 37.5% of patients had acute kidney injury. Other comorbidities such as diabetes mellitus and hypertension have also been reported as concomitant risk factors. Preoperative IABP was used in 37.5% of the patients.[18] Shi J et al. also reported that patients with VSD are more prone to cardiogenic shock and acute kidney injury. Preoperative IABP was reported in all patients included in the study (n = 9).[19] The prevalence of cardiogenic shock was also reported by Heckel et al.[20] Weimer et al. assessed VSR in 10 patients and reported a higher incidence of mortality in patients with comorbid conditions, such as hypertension, cardiogenic shock, and acute kidney injury. However, no significant assessment was performed. No association was found between the location of the defect or type of repair and operative mortality. Six patients exhibited residual or recurrent ventricular septal defects, but neither the location of the defect nor the type of repair predicted the presence of residual or recurrent defects.[21]

Other morbid risk factors were preoperative oliguria and postoperative sepsis which were also reported as independent predictors of mortality by Yam et al., among 40 patients who underwent VSR. The study also demonstrated event-free survival with risk factors such as angina (66%), myocardial infarction (43%), and congestive heart failure (25%).[22] A retrospective study conducted among 41 patients reported that hypertension (90.2%), AMI (68%), advanced age, and late arrival at the hospital were risk factors associated with higher mortality among patients with VSD.[6] Vondran M et al. reported that preoperative IABP was a significant risk factor (68%) for acute MI and surgery <7 days (OR 5.895, CI 1.615-21.515; p = .007).[23] Mortality among 537 patients with VSR was more prevalent in the initial 30 days after surgery, as reported by Arnaoutakis et al., with a consistent pattern of mortality, including preoperative IABP, acute MI, and smoking as risk factors.[25]

Early and late VSR repair

Survival rates were notably lower among patients experiencing cardiogenic shock (survival: 95 [33.6%]; death: 118 [61.5%]; p <0.001) and those undergoing early surgery (time to surgery <7 days, survival: 105 [57.4%]; death: 47 [35.1%]; p <0.001). In multivariate analysis, factors associated with increased mortality included older age (odds ratio [OR], 1.05; 95% CI, 1.02-1.08; p = 0.001), preoperative cardiac arrest (OR, 2.71; 95% CI, 1.18-6.27; p = 0.02), percutaneous revascularization (OR, 1.63; 95% CI, 1.003-2.65; p = 0.048), and postoperative requirement for an intra-aortic balloon pump (OR, 2.98; 95% CI, 1.46-6.09).[17] However, the study findings by Shi et al. reported that intermediate insertion of IABP and haemodynamic stabilisation with early surgical intervention of VSD repair and concomitant revascularization can improve clinical outcomes.[19] A study conducted by Heckel et al. revealed that the overall 30-day survival rate was 62.7% (79 patients). In the early repair group, only 36.2% of the patients were alive at 30 days compared to 85.3% in the delayed repair group. The difference was found to be statistically significant, with a p-value of less than 0.01. According to Heckel et al., the early transcatheter VSD repair group displayed a larger preprocedure Qp: Qs ratio and inferior 30-day survival rates when compared to the late repair group.[20] Additionally, the presence of residual ventricular septal defects did not correlate with an elevated risk of short-term mortality or decline in functional status. Notably, early mortality following the repair of post-infarction ventricular septal defects is noteworthy. However, discharged patients maintained favourable functional capacity and quality of life and demonstrated favourable mid-term survival outcomes.[21]

A significant correlation was identified between the time elapsed to ventricular septal rupture (VSR) closure and the 30-day mortality. Patients treated within a mean duration of 5.9 ± 3.3 days exhibited a higher mortality rate compared to those treated at a mean duration of 21.1 ± 14.3 days (p < .008). Furthermore, a heightened 30-day mortality rate was associated with elevated serum bilirubin levels (p < .02), serum creatinine levels (p < .002), and Model for End-Stage Liver Disease (MELD) scores (p < .006) at the time of VSR closure. In addition, patients required IABP.[24]

Mortality outcomes

The mortality rate based on the included 14 studies was reported to be 40.854%. Fourteen studies included a total of 1378 patients, of which 562 patients died.

In 36 patients (87%), treatment was initiated 24 h or more after the development of AMI symptoms. In 34 patients (83%), rupture occurred during the first

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episode of AMI, and in the majority of these patients (19 patients, 46.3%), preoperative coronary angiography demonstrated disease in only one coronary artery. During the first 10 days after the onset of AMI, five patients (12.2%) were treated surgically but did not survive the operation; 33 patients (80.5%) underwent operation 3-4 weeks after the onset of AMI and all survived.[6]

Mortality within 30 days of ventricular septal rupture (VSR) closure occurred in nine patients (42.9%). All fatalities were attributed to systemic hypoperfusion, followed by cardiogenic shock. Despite aggressive medical intervention, inotropic support, or intra-aortic balloon pump (IABP) use, these patients could not be resuscitated. Notably, the patient treated with an occluder survived without any major complications. Factors linked to 30-day mortality were time to VSR closure, VSR diameter, and serum creatinine levels, which were all significantly associated with this outcome. Moreover, the Model for End-Stage Liver Disease, including the INR (MELD-XI) score, demonstrated a strong correlation with increased risk of mortality.[24] In an investigation conducted by Sabiniewicz et al., four patients experienced mortality within 48 h following the procedure. The 30-day survival rate of the entire cohort was 70%. Univariate analysis revealed the influence of the technical success of the procedure (HR 0.13, CI 0.03-0.68, p = 0.016) and white blood cell count (HR 1.36 per unit increase, CI 1.1-1.69, p = 0.005) on 30-day mortality.[26]

Table 1:

Studies included	Data collection in years	Sample Size (n)	Mean Age (y)	Gender (M/F)	Preprocedures (PCI, Ionotropes)	Timing of VSD from MI (days)	Timing from VSD to repair (days)	Ejection fraction (%)	Follow-up (years)
Ronco D et al., 2021,[17]	2001-2019	475	68.5	290/185	-	-	7	<45% = 43.2%	18
Naik B R et al.2022,[18]	2017-2021	8	68	6/2	-	4	3.0	35.0	5
Shi J et al., 2023,[19]	2013-2020	9	58	8/1	1	1	1-9	-	7
Heckle et al., 2020,[20]	2001-2014	126	69.8	73/53	-	1.26	-	3.5	92.5
Wiemers et al., 2012,[21]	2000-2008	10	65.3	5/5	3	-	6	3.5	18.12
Yam et al., 2012,[22]	1995-2012	40	69	16/24	17	-	28	2	3
Serpytis et al., 2014,[6]	1991-2007	41	67.5	15/26	-	-	-	39	55
Vondran et al., 2020,[23]	1994-2016	53	68.9	30/23	-	-	-	11.9	41.4
Isoda et al., 2012,[14]	2001-2010	7	70.9	3/4	-	3.68	-	-	4.1
Aggarwal et al., 2018,[24]	2000-2014	21	66.4	15/6	4	-	17	-	-
Arnaoutakis et al., 2019,[25]	2008-2012	537	74	277/192	131	-	-	8.4	55
Sabiniewicz et al., 2017,[26]	2003-2016	20	70	11/9	16	1.5	-	182.6	38.5
Tang et al., 2015,[1]	2006-2013	11	67	4/7	6	3	-	18	44
Malhotra et al., 2017,[27]	2009-2014	40	61.65	26	14	-	-	-	3.2

DISCUSSION

Post-myocardial infarction ventricular septal rupture (VSR) presents a significant clinical challenge owing to its high mortality rates.[1-3] While numerous studies have investigated the surgical outcomes associated with this condition, knowledge regarding its risk factors and related factors remains scarce. Against this backdrop, the present systematic review and meta-analysis aimed to aggregate the collective impact of various risk factors and associated clinical outcomes in patients with post-MI VSR. This systematic review and meta-analysis aimed to comprehensively evaluate the surgical outcomes, risk factors, and associated factors in patients with post-MI VSR.

The methodological approach adhered to the established guidelines outlined in the Cochrane Handbook and Meta-analysis of Observational Studies in Epidemiology (MOOSE), ensuring rigorous methodology and transparency. A thorough literature search encompassing multiple databases yielded 14 studies, providing data on 1,380 patients who underwent VSR repair between 2000 and 2022. The mean age of the cohort was approximately 69.5 years, with males comprising the majority of cases (55.6%). The average time from VSR diagnosis to repair across all the studies was 11 days.

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Anatomical considerations, such as the location of myocardial infarction (MI) and VSR, have emerged as important variables. Although posterior involvement was notable, the anterior and apical regions were predominantly affected. Despite differences in the prevalence of anterior and posterior defects among various studies, no significant correlation was observed between defect location and operative mortality.

Several risk factors associated with post-MI VSR have been identified, including cardiogenic shock, older age, smoking, chronic kidney disease, and preoperative interventions such as percutaneous revascularization and intra-aortic balloon pump (IABP) usage. Cardiogenic shock has emerged as a significant predictor of early mortality, emphasising the importance of haemodynamic stabilisation in this patient population.

The incidence of this condition has significantly declined in the era of surgical interventions for post-MI VSR patients, the incidence of this condition has significantly declined.[3] The literature reports an early mortality rate of 19.66% following VSR closure in post-MI VSR patients.[15] The timing of VSR closure has also emerged as a crucial determinant of outcomes, with earlier closure associated with higher mortality rates. Elevated serum bilirubin and creatinine levels at the time of VSR closure, along with a higher Model for End-Stage Liver Disease Excluding INR (MELD-XI) score, were identified as additional predictors of mortality. Additionally, numerous guidelines recommend urgent and early surgical interventions for managing post-MI VSR.[17] Available data indicate that the average time from myocardial infarction (MI) to VSR diagnosis is approximately 4.5 ± 3.3 days, and the average time from VSR diagnosis to repair is around 21.5 ± 40.1 days. During this timeframe, early mortality accounts for 45-50%, mirroring the overall mortality rates. The mortality rate within 30 days of VSR closure was substantial, ranging from 42.9% to 70% across different studies. Factors contributing to mortality include systemic hypoperfusion, cardiogenic shock, and delayed VSR closure. However, the technical success of the procedure and lower white blood cell count were associated with improved survival outcomes.

In many cases, delayed surgical intervention occurs as surgeons await tissue scar formation and improvement.[18-20] Early repair is imperative in cases of haemodynamic compromise, high inotropic support, and evidence of reduced tissue perfusion, such as escalating urea creatinine and liver enzyme levels. Otherwise, a short waiting period is required. Optimal operative outcomes are often achieved when surgery is performed after an acute episode and stable haemodynamics are maintained with minimal inotropic support. Thus, we propose that the average time from VSR diagnosis to repair should be regarded as a crucial factor that adversely affects survival rates. Current guidelines recommend immediate surgical VSR closure regardless of the patient's haemodynamic status. However, surgical intervention in older patients and those with poor right ventricular function is futile, with mortality rates approaching 100%.

Expert consensus and literature data have identified several operative mortality risk factors, including cardiogenic shock and preprocedure intra-aortic balloon pump (IABP) use in post-MI VSR patients.[23-28] Our study analysed data on the pooled proportions of cardiogenic shock and preprocedure IABP usage in patients, revealing incidences exceeding 50% and 66.99%, respectively. Arnaoutakis et al. also collected data from the Society of Thoracic Surgeons (STS) national database, reporting cardiogenic shock incidences of 51.7% upon admission or intervention, with a corresponding 30-day mortality of 42.9%.[25] Furthermore, the analysis of the included studies revealed insights into the impact of concomitant coronary artery bypass grafting (CABG) on mortality outcomes. Although CABG has been performed in a significant proportion of patients undergoing VSR repair, its association with mortality remains controversial, with some studies suggesting a potential increase in mortality risk.

The localisation of the MI or VSR may represent additional variables that influence the outcomes. Our data revealed a significantly higher number of patients with anterior disease localisation than those with posterior wall or septum defects, suggesting that the anterior wall or septum is more susceptible to both MI and VSR. Moreover, the risk difference between anterior and posterior MI or VSR localisation was statistically significant. However, the mortality risk associated with VSR localisation did not show a statistically significant difference between anterior and posterior defects. Notably, the postoperative course tends to be more challenging in posterior VSR cases because of the concurrent right ventricular dysfunction. Our findings suggest that defect location does not significantly affect patient survival rates, although this conclusion is based on only five studies involving 252 patients.[22-28]

Overall, this systematic review underscores the multifactorial nature of post-MI VSR and highlights the importance of timely intervention, haemodynamic support, and careful patient selection to optimise outcomes. Further research is warranted to validate these findings, explore additional prognostic factors, and identify novel strategies to improve the management of this challenging condition.

CONCLUSION

Post-myocardial infarction ventricular septal rupture (VSR) is a serious complication with a significant mortality rate. Through a systematic review and meta-analysis of 14 studies encompassing 1,380 patients, we delineated various risk factors and

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clinical outcomes associated with post-MI VSR. The mean age of the cohort was approximately 69.5 years, with males comprising the majority of cases. The average time from VSR diagnosis to repair across all the studies was 11 days. Although posterior involvement was notable, the anterior and apical regions were predominantly affected. Cardiogenic shock, older age, smoking, chronic kidney disease, and preoperative interventions such as percutaneous revascularization and intra-aortic balloon pump (IABP) usage were identified as significant risk factors for mortality. Early intervention following diagnosis is associated with improved survival rates, emphasising the importance of timely surgical management. The mortality rate within 30 days of VSR closure ranged from 42.9% to 70%, with factors such as delayed closure, VSR diameter, and elevated serum creatinine level contributing to adverse outcomes. Despite advancements in surgical techniques and perioperative care, post-MI VSR remains a formidable challenge, with high mortality rates.

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