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A Study of Post Myocardial Infarction Ventricular Septal Rupture (MI VSR) and its Outcomes - A Single Centre Experience

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Abstract

Objective: Post myocardial infarction (MI) the ventricular septal rupture (VSR), a fatal complication is associated with higher incidences of morality. The main objective of this study is to explore the correlation between the timing of intervention and clinical outcome (mortality), and to identify the risk factors affecting the outcome in MI VSR patients.

Methods: In this retrospective study, 48 patients diagnosed to have post MI VSR admitted to the Departments of Cardiology and Cardiovascular thoracic surgery at KLES Dr. Prabhakar Kore Hospital, Belagavi were analysed. The primary outcome was mortality and the effect of different treatment modalities, management of MI VSR and influence of intervention time on mortality was explored..

Results: Among 48 patients, mortality was 41.2% in 17 patients who received patch closure and 63% in 27 patients who received patch with additional CABG; however, the difference was not statistically different. Further, mortality rate was 54% despite the surgical interventions and between the non-survivor and survivor group mortality was significantly higher in patients with older age (64.77 ± 8.04 years vs. 54.64 ± 14.575, $p<0.01$) and with shorter duration of 'time from AMI to VSR' (2.95±3.51 days vs. 5±3.51 days, $p<0.05$). Post-operative complications such as renal failure ($\chi 2=4.273$, $p<0.05$) and sepsis ($\chi 2=10.688$, $p<0.01$) was significantly different between the two groups with higher occurrence in non-survivor group. The management of MI VSR using thrombolytic therapy, preoperative IABP and ECMO had no significant effect on the outcome.

Conclusion: The shorter duration of time from AMI to VSR predicted mortality, and shorter interval of CABG time and ACCT was effective in the survival of patients. Further, this study indicates that risk factors predisposing patients to mortality also includes post-operative renal failure and sepsis.

Keywords: CABG • Myocardial infarction • Thrombolysis • Ventricular septal rupture • Mortality

Abbreviations: CABG: Coronary Artery Bypass Graft • MI: Myocardial Infarction • PCI: Percutaneous Coronary İntervention • DAPT: Dual Antiplatelet Therapy • VSR: Ventricular Septal Rupture

Introduction

Myocardial infarction (MI) is a common form of coronary artery disease (CAD) and ventricular septal rupture (VSR) is a common fatal complication post first myocardial infarction [1]. It can happen within a few hours, days or weeks after MI and occurs only in 0.2-0.5% cases of MI, but have a very high mortality rate of 50% at 30-day outcome [2,3]. In India at least 11-16% of the young population of age 45 years suffers from MI and is one of the common causes of mortality [4]. Multiple risk factors such as poor socio-economic status, hypertension, smoking, low physical activity, poor diet, increasing age and co-morbidities like diabetes mellitus, hypertension, and obesity are associated with acute MI VSR [4]. Further, surgical closure/intervention is another trend and has been an immediate requirement in case of early myocardial infarction [3]. Over a period of time, surgical repair techniques of VSR have been modified and use of patch repair technique like double patch repair [5,6] and CABG has yielded varying outcome such as the hospitalisation length which can vary from few days to more than 2 weeks [7] and mortality rate varying from 20%-50% depending on the risk. Despite improvement in medical science and surgical techniques, the mortality associated with MI-VSR has not drastically reduced.

The predictors of mortality are multifactorial and include age, cardiogenic shock, heart failure, VSR location, technique of repair, etc [8]. In general the data on treatment modalities and post MI-VSR outcomes are limited. The main objective of the current study is to explore the clinical profile and outcomes in patients with MI VSR who underwent different treatment modalities such as 'only patch', CABG and patch closure or device implantation and to find a correlation between the timing of intervention and clinical outcome. Additionally, the identification of risk factors in affecting the outcome was explored.

Research Methodology

The present retrospective study was done between July 2010 and July

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2020. In total, 48 patients with post MI VSR admitted in the Departments of Cardiology and Cardiovascular thoracic surgery at KLES Dr. Prabhakar Kore Hospital, Belagavi were included. The exclusion criteria included patients from OPD and those associated with any congenital heart disease. Informed consent was mandatory to include the patients. Prior to the study, ethical clearance was obtained from the institution. For each patient the following data was collected: age, sex, NYHA class, BMI, DM, HTN, past H/O CVA. Clinical and angiographic data included time from AMI to VSR, time from AMI to VSR repair, SBP, DBP, heart rate, AMI location, VSR location, cardiogenic shock, LV repair, number of vessel disease and ejection fraction. The treatment modalities included technique of repair, patch closure, device closure, CABG and patch closure. Management of MI VSR included data on thrombolysis, presence of IABP and ECMO support. The postoperative data included length of ICU stay, renal failure and post-operative sepsis. The primary outcome was mortality.

All the quantitative variables were presented as frequencies indicating the number and percentage. All statistical association between variables were made using Chi-square test. The SPSS software v 24.0 was used to perform statistical calculations and a p value of less than 0.05 was considered statistically significant.

Results

Post infraction VSR requires surgical repair involving patch repair, device closure and patch closure with CABG to stabilise MI VSR patients. In this study, based on treatment modalities received by patients, the clinical profile was categorised into four categories, in particular, the patients receiving only patch closure (n=17), patch and CABG (n=27), device implantation (n=2) and none suggesting not receiving any treatment (n=2) (Table 1). Gender-specific difference with regard to treatment modalities was not observed, however, a larger number of male and overweight patients received patch closure and CABG but this difference was not significant (p>0.05). Further, the association of treatment modalities with co-morbidities like diabetes mellitus (χ2=4.566, p>0.05), hypertension (χ2=1.874, p>0.05) and history of CVA (χ2=0.794, p>0.05) was not significant. Similarly, cardiogenic shock (χ2=3.804, p>0.05) post-operative renal failure (χ2=1.399, p>0.05), and thrombolysis (χ2=0.115, p>0.05) had no significant association with treatment modalities.

Further the comparison of clinical and angiographic data between the treatment modalities is provided in Table 2. Treatment modalities were not significantly associated with 'time from AMI to VSR' (F=0.251, p>0.05) and 'time from AMI to VSR repair' (F=1.215, p>0.05). Similarly, other variables like SBP (F=1.185, p>0.05), DBP (F=0.601, p>0.05), heart rate (F=0.18, p>0.05) and PAP (F=0.52, p>0.05) also did not indicate a significant association with treatment modalities.

Treatment modalities and mortality

With respect to mortality, the clinical data were analysed between survivors and non-survivors. The association between treatment modalities and mortality was found to be insignificant (χ2=2.025, p>0.05) (Table 3). The 'only patch' closure performed using Sauvage, Dacron, bovine or double patch on 17 patients resulted in 41.2% mortality, whereas CABG and patch closure (n=27) resulted in higher mortality of about 63%, indicating lack of benefit of CABG in patient's survival post MI VSR. In the remaining four patients, two patients used device implantation and other two patients did not undergo surgery, and the reported mortality was 50% in each case.

Clinical profile of survivor and non-survivor

Table 4 revealed a significant association of mortality with age of patients (χ^2=9.712, p<0.01) with an average age of 64.77 ± 8.04 years in non-survivor and 54.64 ± 14.575 years in survivors. The association of mortality with other variables like NYHA (χ^2=0.201, p>0.05), gender (χ^2=2.859, p>0.05), BMI (χ^2=0.581, p>0.05), DM (χ^2=0.336, p>0.05), hypertension (χ^2=0.815, p>0.05) and history of CVA (χ^2=0.864, p>0.05) was insignificant. Likewise, LV repair (χ2=1.343, p>0.05), PAP (χ^2=3.544, p>0.05), ejection fraction (χ^2=2.973,

Table 1. Clinical profile of the groups for different treatments.

Variables	Only patch (n=17)	CABG+Patch (n=27)	Device (n=2)	None (n=2)	Chi-square
Sex					
Male	11 (35.5%)	19 (61.3%)	2 (11.8%)	1 (3.2%)	4.228 (NS)
Female	6 (35.3%)	8 (47.1%)	0 (0%)	1 (5.9%)	
BMI[a]					
Normal Weight	2 (33.3%)	3 (50%)	1 (16.7%)	0 (0%)	4.240 (NS)
Overweight	12 (35.3%)	20 (58.9%)	1 (2.9%)	1 (2.9%)	
Obese	3 (37.5%)	4 (50%)	0 (0%)	1 (12.5%)	
DM					
Yes	7 (29.2%)	13 (54.2%)	2 (8.3%)	2 (8.3%)	4.566 (NS)
No	10 (41.7%)	14 (58.3%)	0 (0%)	0 (0%)	
HTN					
Yes	5 (35.7%)	9 (64.3%)	0 (0%)	0 (0%)	1.874 (NS)
No	12 (35.3%)	18 (52.9%)	2 (5.9%)	2 (5.9%)	
Past H/O CVA					
Yes	0 (0%)	1 (100%)	0 (0%)	0 (0%)	0.794 (NS)
No	17 (36.2%)	26 (55.3%)	2 (4.3%)	2 (4.3%)	
Renal Failure					
Yes	1 (20%)	4 (80%)	0 (0%)	0 (0%)	1.399 (NS)
No	16 (37.2%)	23 (53.5%)	2 (4.7%)	2 (4.7%)	
Cardiogenic Shock					
Yes	11 (45.8%)	12 (50%)	1 (4.2%)	0 (0%)	3.804 (NS)
No	6 (25%)	15 (62.5%)	1 (4.2%)	2 (8.3%)	
Thrombolysis					
Yes	7 (33.3%)	12 (57.1%)	1 (4.8%)	1 (4.8%)	0.115 (NS)
No	10 (37%)	15 (55.6%)	1 (3.7%)	1 (3.7%)	

Note: BMI: Body Mass Index; DM: Diabetes Mellitus; HTN: Hypertension; CVA: Cardiovascular Accident [a]BMI Value Range: 18.5-24.9 (Normal Weight), 25.0-29.9 (Overweight), >30 (Obese) [9]

Table 2. Post MI VSR profile for different treatments.

Group	Only patch n=17	CABG+Patch n=27	Device n=2	None n=2	F	p-value
Time from AMI to VSR (days)	3.82 ± 2.58	3.93 ± 2.88	2.68 ± 3.29	5.2 ± 6.79	0.251	0.86
Time from AMI to VSR REPAIR (days)	6.53 ± 3.86	6.41 ± 4.6	0.38 ± 0.53	6.25 ± 8.13	1.215	0.315
SBP (n)	101.12 ± 24.22	101.19 ± 19.15	99 ± 15.56	130 ± 28.28	1.185	0.326
DBP (n)	65.29 ± 12.31	64 ± 10.35	63 ± 9.9	75 ± 21.21	0.601	0.618
Heart rate (n)	102 ± 13.47	104.07 ± 13.53	98 ± 5.66	104.5 ± 24.75	0.18	0.909
PAP (n)	38.24 ± 13.05	43.48 ± 18.66	36 ± 8.49	30.5 ± 4.95	0.52	0.671

Note: AMI: Acute Myocardial Infarction; VSR: Ventricular Septal Rupture; SBP: Systolic Blood Pressure; DBP: Diastolic Blood Pressure; PAP: Pulmonary Pressure

Table 3. Association of treatment modalities and mortality in post MI VSR patients.

Treatment modalities	Survivor (n=22)	Non-survivor (n=26)	Chi-Square
Only patch	10 (58.8%)	7 (41.2%)	2.025 (NS)
CABG+Patch	10 (37%)	17 (63%)	
Device	1 (50%)	1 (50%)	
None	1 (50%)	1 (50%)	

Table 4. Clinical profile of the survivor and non-survivor.

Variables	Survivor (n=22)	Non-survivor (n=26)	Chi-Square
Age (years)	54.64 ± 14.575	64.77 ± 8.04	9.712**
NYHA			
Yes	1 (33.3%)	2 (66.7%)	0.201 (NS)
No	21 (46.7%)	24 (53.3%)	
Sex			
Male	17 (54.8%)	14 (45.2%)	2.859 (NS)
Female	5 (29.4%)	12 (70.6%)	
BMI			
Normal Weight	2 (33.3%)	4 (66.7%)	.581 (NS)
Overweight	16 (50%)	16 (50%)	
Obese	4 (50%)	4 (50%)	
DM			
No	10 (41.7%)	14 (58.3%)	.336 (NS)
Yes	12 (50%)	12 (50%)	
HTN			
Yes	5 (35.7%)	9 (64.3%)	.815 (NS)
No	17 (50%)	17 (50%)	
Past H/O CVA			
Yes	0 (0%)	1 (100%)	.864 (NS)
No	22 (46.8%)	25 (53.2%)	
Cardiogenic Shock			
Yes	9 (37.5%)	15 (62.5%)	1.343 (NS)
No	13 (54.2%)	11 (45.8%)	
LV Repair			
Not done	13 (54.2%)	11 (45.8%)	1.343 (NS)
Done	9 (37.5%)	15 (62.5%)	
Mitral regurgitation			
0	4 (50%)	4 (50%)	5.547 (NS)
G1	10 (60%)	15 (40%)	
G2	3 (100%)	0 (0%)	
Trivial	5 (58.3%)	7 (41.7%)	
PAP[a]			
Normal PAP	9 (60.0%)	6 (40.0%)	3.544 (NS)
Mild PAP	7 (31.8%)	15 (68.2%)	
Moderate PAP	2 (66.7%)	1 (33.3%)	
Severe PAP	4 (50.0%)	4 (50.0%)	
EF[b]			
Severe LV Dysfunction	1 (4.5%)	3 (11.5%)	2.973 (NS)
Moderate LV Dysfunction	11 (50%)	17 (65.4%)	
Mild LV Dysfunction	10 (45.5%)	6 (23.1%)	
VSR location			
Apical VSR	17 (77.3%)	22 (84.6%)	0.646 (NS)
Mid muscular VSR	3 (13.6%)	3 (11.5%)	
Posterior VSR	2 (9.1%)	1 (3.8%)	

Note: ** p<0.01; NYHA: New York Heart Association; LV: Left ventricular; PAP: Pulmonary artery pressure; EF: Ejection Fraction; VSR: Ventricular Septal Rupture [a] Normal PAP range: <25 mmHg; mild: 25-40 mmHg; moderate: 41-55 mmHg; severe: >55 mmHg [10]. [b] EF range for LV dysfunction: 41-45% (mild), 36-40% (moderate) and ≤ 35% (severe) [11]

p>0.05), VSR location (χ^2=0.646, p>0.05) and mitral regurgitation (χ^2=5.547, p>0.05) had no statistically significant impact on mortality. Though insignificant, mortality was higher in patients with moderate LV dysfunction. Likewise, cardiogenic shock was not significantly associated with mortality (χ^2=1.343, p>0.05) but there was increased occurrence of death in patients (62.5%) who experienced cardiogenic shock.

Management of MI VSR

In the management of MI VSR, 21 patients received thrombolysis, 45 received IABP and 3 patients received ECMO (Table 5). The data revealed a non-significant association between thrombolysis and mortality (χ^2=0.048, p>0.05). The mortality rate with therapy was 52.4%, indicating that thrombolysis therapy did not aid in survival (47.6%). Likewise, the use of preoperative assist device - IABP and post MI VSR mortality was not significant (χ^2=0.559, p>0.05). Out of 45 patients who received preoperative IABP, mortality was higher in patients with IABP than rather no IABP (55.6%), however, about 44.4% survived even without IABP, indicating lack of advantage of IABP on survival. Further, there was no significant benefit of ECMO in the survival of MI VSR patients (χ^2=2.708, p>0.05), all three patients who received ECMO died.

Involvement of arteries In MI VSR

The involvement of arteries in MI-VSR is presented in Table 6. The mortality was 83.3% in patients with RCA involved with a significant Chi-square value of 5.116 (p<0.05). The other variables like number of diseased vessels (χ^2=1.681, p>0.05), left main (χ^2=1.766, p>0.05), left circumflex coronary artery (LCX) (χ^2=0.184, p>0.05) and LAD (χ^2=0.184, p>0.05) was not associated with mortality. Though insignificant, the mortality incidences were higher in patients with triple vessel disease, when left main was not involved.

Table 5. Management of MI-VSR.

Techniques	Survivor (n=22)	Non-survivor (n=26)	Chi-Square
Thrombolysis			
Yes	10 (47.6%)	11 (52.4%)	0.048 (NS)
No	12 (44.4%)	15 (55.6%)	
IABP device			
Yes	20 (44.4%)	25 (55.6%)	0.559 (NS)
No	2 (66.7%)	1 (33.3%)	
ECMO			
Yes	0 (0.0%)	3 (100%)	2.708 (NS)
No	22 (48.9%)	23 (51.1%)	

Note: IABP: Intra-Aortic Balloon Pump; ECMO: Extracorporeal Membrane Oxygenation

Table 6. Involvement of arteries in MI VSR.

Variables	Survivor (n=22)	Non-survivor (n=26)	Chi-Square	p-value
Diseased Vessels				
SVD-Single Vessel Disease	10 (50%)	12 (48%)	1.681	0.641
DVD-Double Vessel Disease	5 (25%)	6 (24%)		
TVD-Triple Vessel Disease	1 (5%)	4 (16%)		
Normal	4 (20%)	3 (12%)		
Left Main				
Involved	22 (100%)	24 (92.3%)	1.766	0.184
Not Involved	0 (0%)	2 (7.7%)		
LAD				
Involved	15 (71.4%)	20 (76.9%)	0.184	0.668
Not Involved	6 (28.6%)	6 (23.1%)		
LCX				
Involved	6 (28.6%)	6 (23.1%)	0.184	0.668
Not Involved	15 (71.4%)	20 (76.9%)		
RCA				
Involved	2 (9.5%)	10 (38.5%)	5.116	0.024
Not Involved	19 (90.5%)	16 (61.5%)		

Note: LAD: Left Anterior Descending; LCX: Left Circumflex; RCA: Right Coronary Artery

Post-operative outcomes

The post-operative outcomes are presented in Table 7. Among patients who did not survive, a gradual decrease in length of ICU stay was observed (<10 days: 69.2%; 11-20 days: 26.9%; 21-30 days: 3.8%), however, the association was insignificant (χ^2=5.883, p>0.05). Further, the post-operative complication such as renal failure (χ^2=4.273, p<0.05) and sepsis (χ^2=10.688, p<0.01) was significantly different between the survivors and non-survivor. There was 100% mortality in case of renal failure and sepsis indicating that renal failure and sepsis are the crucial complications, which may need immediate attention after the surgery. In this study, there was no report of post-operative ventricular tachycardia and post-operative stroke.

Correlation between timing of intervention and mortality

The correlation analysis showed a significant relationship between 'time from AMI to VSR' and mortality (t=2.617, p<0.05), with lower time of 2.95 ± 3.51 days in case of non-survivors as compared to 5±3.51 days in survivors (Table 8). Mortality was not significantly correlated to 'time from AMI to VSR repair' (t=2.004, p>0.05), timing of IABP insert (t=1.216, p>0.05), CABG time (t=2-1.159, p>0.05) and aortic cross lamp time (t=-1.227, p>0.05). Nevertheless, in non-survivors the time from AMI to VSR repair was 5.04 ± 2.68 days, whereas in survivors it was 7.54 ± 5.65 days indicating that shorter the period from AMI to VSR repair, the chances of survival reduces. The timing of IABP insert was 3.73 ± 0.87 days in case of non-survivor group and 3.5 ± 1.14 days for survivors. The CABG time was 132.55 ± 49.30 min in case of survivors and 163.04 ± 108.13 min in case of non-survivors, suggesting that longer timing of coronary artery bypass grafting after AMI may have worse outcomes. The aortic cross clamp time (ACCT) was 70.13 ± 35.09 min and 83.42 ± 39.19 min in case of survivor and non-survivor, respectively, suggestive of longer ACCT affecting the survival.

Discussion

There have been high incidences of AMI complicated by VSR and mortality associated with it. In this study, the operative mortality was 54.0%, which is higher than the 30-days reported operative mortality of ranging from 19%-37%, but lower than reported 1-month or 2-month mortality of 94% or 90% in the absence of surgical procedures [12] suggesting that mortality incidences can be reduced by proper surgical interventions. Further, the risk of mortality was higher in patients of older age. Similar observation was reported by Menon et al. [13] wherein MI VSR non-survivors were older patients (>70 years) and non survivors were largely female of normal weight with co-morbidities like hypertension, past history of CVA and with cardiogenic shock. The mortality rate ranging from 35% - 87% among MI VSR patients with shock is observed in many studies [13] thereby emphasising the immediate treatment before the onset of septal rupture. In this study, though insignificant but high mortality (62.5%) was observed in patients presented with cardiogenic shock suggesting that faster intervention will help in survival of MI VSR patients. Further, reduced LVEF is known to increase the risk of cardiac death. In a study of 30-day

Table 7. Post-operative outcomes in MI VSR patients.

Post-operative outcome	Survivor (n=22)	Non-survivor (n=26)	χ^2 (Sig)
ICU Stay			
<10 days	8 (36.4%)	18 (69.2%)	5.883 (NS)
11 - 20 days	10 (45.5%)	7 (26.9%)	
21 - 30 days	4 (18.2%)	1 (3.8%)	
Renal Failure			
Yes	0 (0%)	5 (100%)	4.723*
No	22 (51.2%)	21 (48.8%)	
Sepsis			
Yes	0 (0%)	10 (38.5%)	10.688**
No	22 (100%)	16 (61.5%)	

Note: * p<0.05; ** p<0.01; ICU: Intensive Care Unit

Table 8. Correlation between timing of intervention and mortality.

Variables	Survivor (n=22)	Non-survivor (n=26)	t value	Sig
Time from AMI to VSR (days)	5 ± 3.51	2.95 ± 3.51	2.617	0.012
Time from AMI to VSR repair (days)	7.545 ± 5.655	5.048 ± 2.685	2.004	0.051
Timing of IABP Insert (days)	3.5 ± 1.144	3.731 ± 0.874	1.216	0.23
CABG time (min)	132.55 ± 49.301	163.04 ± 108.133	-1.159	0.253
Aortic Cross Clamp Time (ACCT) (min)	70.136 ± 35.093	83.423 ± 39.193	-1.227	0.226

mortality EF <40 independently predicted mortality in MI VSR patients [14]. Likewise, the prognostic value of EF in predicting all-cause mortality in post myocardial infarction is known [15,16]. In the present study, in spite of high mortality of 75% in patients with LVEF <40, LVEF could not be regarded as an independent risk predictor of mortality likely due to the low specificity of EF in risk stratification in case of MI, which indicates that pre-existing cardiac conditions could not affect the outcome [17].

Treatment modalities

The current management of post MI VSR involves surgical technique like patch repair, CABG, etc. [18] and non-surgical interventions using septal occluder device [19]. In this study, patch repair along with CABG was the common treatment modalities among the overweight and male MI VSR patients. Balkanay et al. [5] advocated the usefulness of double-patch technique to repair inferoposterior infarction which resulted in no further reoperation in patients who had preoperative IABP. In this study, though insignificant but reduced mortality was observed in case of only patch repair and higher mortality in patients who were treated with both CABG and patch. The efficacy of single patch using Dacron/bovine [20] and double patch [21] in reduced incidence of residual shunt was observed. Clinical studies have advocated the benefit of CABG in reducing mortality post MI VSR [22]. However, the findings of this study were not in agreement. A lack of significant benefit of CABG along with patch on mortality was evident. The finding of this study was in concordance with Malhotra et al. [23], wherein the authors did not find any protective effect of CABG on mortality. Similarly, Khan et al. [24] found that concomitant CABG did not confer any early and long-term survival advantage in MI VSR patients. With respect to the device implantation, mortality within 30 days was 71% in MI VSR patients with occluder device (Amplatzer). In line with this, in the present study, VSD closure by Amplatzer resulted in 50% mortality suggesting that the outcome can vary depending on the risk factors associated with the case.

Involvement of arteries

Observation of Malhotra et al. [23] on no impact of coronary artery vessels on the mortality outcomes was in agreement with our observation. Patients with single, double and triple vessel disease had higher mortality but the difference was not significant. Further, location of VSR also did not influence the outcome. A similar observation was observed by Malhotra et al. [23]. In line with our study, Huang et al. [25] also found apical VSR as the common location of VSR. In the present study, apical VSR was common among the patients (82%) and had larger number of non-survivors (84%); however, the difference was not significant. Among the culprit vessels, RCA was the only vessel significantly associated with the outcome; however presented with lower mortality when involved. Very limited and not so conclusive clinical data is available on the impact of coronary vessel on outcomes. Entezarjou et al. [25] showed that in case of ST-elevation myocardial infarction (STEMI) patients who underwent primary percutaneous coronary intervention (PCI) the 1-year mortality rate was high in cases with infarctions in LAD and LCX than the infarctions in RCA likely due to the heart failure. On the contrary, total occlusion of RCA and LAD was presented in some patients of AMI [27,28]. Nevertheless, in such cases where multi vessel occlusions are the risk factors of MI, the reperfusion therapy largely helps in survival of patients.

Management of MI VSR

One of the optimal management of MI VSR recommends preoperative IABP and ECMO to allow the stabilisation of hemodynamics [29,30]. In the present study presence of preoperative IABP and likewise the timing of IABP insert had no effect on mortality. Although the pre-operative IABP insertion in patients undergoing cardiac surgery reduces mortality [31] but the efficacy of optimal timing of the initiation of IABP in survival of patients with acute MI undergoing PCI has not been impactful [32] indicating that optimal timing of IABP is debatable and must be considered on a case-to-case basis. In the context of thrombolytic therapy the findings was in agreement with Srinivas et al. [33] wherein early or late thrombolytic therapy did not serve any advantage to the survival of MI VSR patients. ECMO normally has benefit on survival but in this study, ECMO did not show any advantage. Contrary to our results, Tsai et al. [34] found that preoperative ECMO can delay the surgery and increase the survival by restoring hemodynamic stability.

Post-operative outcome

Following first MI, deterioration of renal function is reported [35]. Additionally, post-operative renal dysfunction and failure in MI VSR patients have led to prolonged hospitalisation and increased mortality [36]. In this study, though there were lower incidences of post-operative renal failure and post-operative sepsis but none of the patients survived, suggestive of requirement of specialised care to mitigate renal failure and strategy to prevent multiple organ failure.

Timing of intervention

The period from MI to VSR followed by VSR repair influences the outcome. Higher mortality rate of about 90% without surgery or 75% at 30-day mortality is reported within 3-5 days [37] or 1-day [38] of time to VSR identification. In this study, we found that 'time from AMI to VSR diagnosis' is crucial for

the survival with 5 ± 3.51 days in survivors compared to 2.95 ± 3.51 days in non-survivor group, likely because the later occurrence of VSR after MI will provide sufficient time for the better achievement of hemodynamic stability and also delay the surgery process in these patients. A similar observation was reported by Malhotra et al. [23], wherein the time between AMI to VSR was 2.29 ± 1.4 days for non-survivor as compared to 4.16 ± 1.9 days for survivor. The events from MI to VSR repair with an average of 7.5 days improved survival, suggesting that this could be indicative of optimum waiting time for the intervention. A similar observation with reduced mortality by delaying VSR surgery or repair by more than 3 days [23] or 7 days [37] was observed. There is no consensus on optimal time for VSR repair but it can be inferred that delayed repair can reduce the mortality.

Further, the optimal timing of CABG in AMI has not been conclusive [39]; operative mortality of 39.5% was reported in MI VSR patients with a mean CABG time of 152 ± 52 minutes [20]. However, prolonged aortic cross-clamp time of >60 min and linear increase in ACCT time is an independent risk factor of mortality in patients with low and high cardiac risk [40]. In this study, though ACCT was insignificant between survivor and non-survivor group, but larger incidences of mortality coincided with prolonged ACCT time. Thus, it can be inferred that MI VSR patients with lower CABG time and lower ACCT has better chances of survival.

Limitations of the Study

This was a single centre retrospective study. Additionally, relatively small sample size and representation of data from one centre could limit the statistical significance and generalisation of findings.

Conclusion

It can be inferred that despite surgical interventions the mortality rate was 54% indicating requirement of further improvement in medical management of MI VSR patients. Among the time of interventions, the shorter time from AMI to VSR significantly reduces the survival and that mortality is higher in patients with post-operative complications such as renal failure and sepsis. Further, thrombolytic therapy and preoperative IABP insertion had no effect on the outcome. However, shorter interval of CABG time and ACCT was effective in the survival of patients.

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Conflict of Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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