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# FACTORS AFFECTING OUTCOME AND SURVIVAL AFTER SURGICAL REPAIR OF POST-INFARCTION VENTRICULAR SEPTAL DEFECT

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## ABSTRACT

**Objective:** To review the experience of surgical repair of post-infarction ventricular septal rupture (VSR) and analyze the associated outcomes and prognostic factors.

**Methodology:** A retrospective review was performed on 38 consecutive patients who had undergone surgical repair of post- infarction VSD between 2002 and 2012. Continuous variables were expressed as either mean ± standard deviation or median with 25th and 75th percentiles. These were compared using two-tailed t-test or Mann–Whitney U test respectively. A two-tailed p-value $< 0.05$ was used to indicate statistical significance.

**Results:** Mean age was 46.5 ± 5.9 years, all patients were males. The VSD was anterior in 19 (82.6%) and posterior in 4 patients. Median interval from myocardial infarction to VSR was 1 day (1-4). Pre-operative intra-aortic balloon pump was inserted in 37 patients (97.8%). Thirty-six patients (94.7%) underwent coronary angiography. All patients underwent patch repair. Mean aortic cross clamp time was 68 ± 34minutes and mean cardiopulmonary bypass time was 132 ± 44 minutes. Coronary artery bypass grafting (CABG) was performed in 15 patients (65%), with a mean of 1.5 ± 0.7 distal anastomoses. Operative mortality within 30 days was 43.5%. Univariate analysis identified emergency surgery, New York Heart Association (NYHA) class, inotropic support, right ventricular dysfunction, EuroSCORE II, intra-operative red cell transfusion, post-operative renal failure and renal replacement therapy (RRT) as predictors of operative mortality.

**Conclusions:** Surgical repair of post-infarction VSD carries a high operative mortality. NYHA class at presentation and post-operative RRT are predictors of early mortality. Concomitant CABG does not improve survival.

**Key Words:** Ventricular septal defect, Myocardial infarction, Surgical repair

## INTRODUCTION

The reported incidence of acute ventricular septal defect (VSD) following acute myocardial infarction (AMI) is 1% to 3% in the era prior to widespread reperfusion therapy.[1] The incidence has since declined to around 0.3% following the advent of thrombolysis.[2]

Despite advances in critical care and prompt surgical intervention, the incidence of post-infarction VSD has not changed during the past 2 decades i.e in the post thromboysis era. The mortality rate of post-infarction VSD remains high and relatively constant. [3] Reported 30-day mortality rates range from 19 to 54%.[3-11] This study aims to investigate the survival outcome and prognostic factors associated with surgical repair of post-infarction VSD at a tertiary referral center over a 10 year period.

Percutaneous closure has been increasingly used in patients with post infarction VSDs, initially in patients with recurrent ventricular septal defects (VSDs) after primary surgical repair, but more recently as primary therapy in patients with acute VSR and high surgical risk, or as a temporizing bridge to surgery.[12]

Ventricular septal occluder devices were initially developed for percutaneous closure of Ventricular septal occluder devices were initially developed for percutaneous closure of congenital VSDs. Percutaneous deployment of an occluding device may be precluded by the geometry and location of the VSD, or interference from valvular apparatus, especially in posterior VSDs. The highest technical success has been reported with the Amplatzer device (AGA Medical Corp, Plymouth, MN), with procedural success rates ranging from 86% to 89%.[13,14] Lee et al. [15] recently described the use of a hybrid approach with the advantage of allowing direct manipulation of the Amplatzer device. This approach prevents interference of the valve apparatus, avoiding the need to perform ventriculotomy in an already infarcted ventricle.

Large (≥15 mm) VSDs should undergo immediate surgery as these are prone0to device embolization or residual VSD. Amplatzer closure can be used as definitive therapy for small or medium VSRs. It can also be used to stabilize patients and allow myocardial fibrosis, thus facilitating delayed surgical correction. [13] Despite a less invasive technique, procedural mortality and morbidity are high, especially in patients with cardiogenic shock (mortality in cardiogenic shock 88% vs. 38% non-shock $P<0.001$).[12] Overall 30-day mortality rates for percutaneous closure range from 28% to 65%.[12-15] Other procedure-related major complications include residual shunting, left ventricular rupture and device embolization. The incidence of residual shunts ranges from 8.3% to 13.8%, much lower than 73% reported in an earlier series.[12-15]

Ventricular assist devices (VADs) are useful adjuncts for the treatment of VSDs in the setting of univentricular or biventricular failure, either preoperatively as a bridge to surgery or postoperatively following VSD repair. Mechanical circulatory support with VADs allows restoration of peripheral organ perfusion and provides an opportunity for recovery and maturation of the infarcted myocardium before secondary VSD repair.[16] Applying this concept, a staged approach of initial left ventricular or biventricular mechanical support with an implantable left ventricular assist de-vice (LVAD) or biventricular assist device (BiVAD) followed by secondary VSD repair has been described.[17,18] Previously, BiVAD support without VSD repair has been de-scribed as a bridge-to-transplantation approach.[19]

## METHODOLOGY

A retrospective case-note and database review was performed on consecutive patients who had undergone surgical repair of post-infarction VSR between January 2002 to September 2012 at our tertiary referral center.

Early or operative mortality was defined as death within 30 days of surgery, either in hospital or after hospital discharge. Renal failure was defined by serum creatinine levels higher than 1.36 mg/dl or the need for renal re- placement therapy (RRT). Inotropic support was defined as infusions of dopamine over 5 mcg/kg/min or any use of adrenaline, noradrenaline or vasopressin. Emergency surgery was defined as surgery within 24 hours of diagnosis of VSD. Post-operative RRT was in the form of Continuous Veno-Venous Hemofiltration (CVVH).

Statistical analysis was performed for the surgically treated group, using SPSS version 17 statistical software. Continuous variables were expressed as either mean ±standard deviation or median with 25th and 75th percentiles, depending on the normality of distribution. These were compared using two-tailed t-test or Mann–Whitney U test respectively. Categorical variables, expressed as percentages, were analyzed with chi-square or Fisher's exact test. To identify risk factors predictive of operative mortality, univariate analysis of perioperative variables was per- formed. Significant univariate risk factors were examined using backward logistic regression analysis. Survival function was presented using Kaplan-Meier survival curves and comparisons performed with log-rank test. A two- tailed p-value less than 0.05 was used to indicate statistical significance.

## RESULTS:

During the period January 2002 to September 2012, 23 patients underwent surgical repair of VSD. No patients were treated with percutaneous closure devices.Data was

retrospectively collected from patient case notes and electronic records. Patient demographics and comorbidities are shown in Table 1.

Male patients accounted for 100%. Mean age was 46.5 ± 5.9 years (range 38 – 77). In all patients, a new car- diac murmur was present in the setting of an ST-segment elevation myocardial infarction (STEMI). Transthoracic echocardiography (TTE) was performed for all patients to confirm the diagnosis of VSD and exclude the differential diagnosis of acute mitral regurgitation from papillary muscle rupture. Evidence of right ventricular (RV) dysfunction such as RV dilatation or wall motion abnormality was also diagnosed with pre-operative echocardiography. RV dysfunction was present in 7 patients (30.4%), of which 6 had presented with inferior MI and 1 with anterior MI. RV dysfunction was present in all patients with posterior VSDs and 6 of 19 (31.6%) with anterior VSDs, ($P<0.0005$). The VSD location was anterior in 19 patients (82.6%) and posterior in 4 patients (17.3%). All patients had VSD at presentation. 01 patient developed the VSD 3 days after admission in CCU with acute anterior myocardial infarction.

Six patients (26%) had undergone thrombolysis with either streptokinase or recombinant tissue plasminogen activator. Coronary angiography was performed in all patients, which showed single vessel disease (SVD) in 4 (17.4%), double vessel disease (DVD) in 4 (17.46%) and triple vessel disease (TVD) in 15 (65.2%) of patients. Within the SVD group, 3 of 4 patients (75%) had total occlusion of the infarcted artery.

All twenty three operations were performed via median sternotomy. Moderately hypothermic cardiopulmonary bypass was instituted in all cases. Aortic cross clamping and cold cardioplegic myocardial protection was applied in all patients. Mean cardiopulmonary bypass time was 130 ± 44 minutes and mean aortic cross clamp time was 68 ± 39 minutes.

Single patch closure was performed in 18 cases (78.2%), using either a Dacron or bovine pericardial patch, adopting an infarct exclusion technique similar to that of David et al.[8] Five cases were repaired by direct suturing without a patch. In 1 case, the VSD was too extensive and not amen- able for repair by any method, resulting in an immediate post-operative death. The ventriculotomy was closed by direct suture buttressed on felt strips in all patients. Coronary

**Table 1: Patient Demographics and Comorbidities**

| Variable | Survivors n = 13 (%) | Non-survivors n = 10 (%) | P-value |
|---|---|---|---|
| Age (years) | 45.3 ± 8.2 | 46.3 ± 9.9 | 0.356 |
| Gender (Male) | 13 | 10 | 0.134 |
| BMI(kg/m2) | 28.1 ± 4.3 | 29.7 ± 2.0 | 0.186 |
| LVEF (%) | 45.0 ± 12.0 | 35.0 ± 9.4 | 0.746 |
| **Comorbidities** | | | |
| Smoking | 8 (61.5) | 8 (80.0) | 0.258 |
| Diabetes mellitus | 9 (69.2) | 7 (70.0) | 0.879 |
| Renal failure | 9 (69.2) | 6 (60.0) | 0.132 |
| Hypertension | 11 (84.6) | 9 (90.0) | 0.631 |
| Hyperlipidemia | 9 (69.2) | 5 (50.0) | 0.166 |
| Previous stroke | 0 (0) | 0 (0) | 0.239 |

BMI: Body Mass Index.
LVEF: Left Ventricular Ejection Fraction

**Table 2: Univariate Risk Analysis for Operative Mortality**

| Variable | Survivors n = 13 (%) | Non-survivors n = 10 (%) | P-value |
|---|---|---|---|
| Pre-operative | | | |
| NYHA Class (II-III / IV) | 3(23.0)/4(30.8) | 1 (10) / 9 (90) | 0.004 |
| Emergency Surgery | 13 (100) | 10 (100) | 0.031 |
| Cardiogenic Shock | 12 (92.3) | 9 (90) | 0.051 |
| Inotropic support | 5 (38.5) | 5 (50) | 0.028 |
| Type of MI (Anterior/Inferior) | 9(69.2)/4(30.8) | 8 (80) / 2 (20) | 0.259 |
| Right ventricular dysfunction | 3 (23.0) | 4 (40) | 0.021 |
| Multi-vessel coronary artery disease | 8 (61.5) | 7 (70) | 0.452 |
| Total occlusion of infarcted artery | 10 (76.9) | 7 (70) | 0.636 |
| Thrombolysis | 7 (53.8) | 5 (50) | 0.802 |
| Intra-aortic balloon pump | 13 (100) | 10 (100) | 0.413 |
| EuroSCORE II | 30.8 ± 9.4 | 36.3 ± 9.8 | 0.001 |
| Intra-operative | | | |
| VSD Location (Anterior/Posterior) | 12(92.3)/1(7.6) | 7(70.0)/3(30.0) | 0.122 |
| CPB time (minutes) | 132.0 ± 52.3 | 158.8 ± 53.0 | 0.540 |
| Red blood cell transfusion > 2 units | 9 (69.2) | 8 (80) | 0.012 |
| Concomitant CABG | 9 (69.2) | 7 (70) | 0.740 |
| Post-operative | | | |
| Renal failure | 4 (30.8) | 9 (90) | 0.020 |
| Renal replacement therapy | 2 (15.4) | 6 (60) | <0.0005 |
| Re-exploration for bleeding | 2 (15.4) | 0 (0) | 0.493 |
| DIVC | 0 (0) | 3 (30) | 0.025 |

CABG: Coronary artery Bypass grafting. CPB: Cardiopulmonary bypass.
DIVC: Disseminated intravascular coagulopathy.
EuroSCORE: European System for Cardiac Operative Risk Evaluation II.
MI: Myocardial infarction. NYHA: New York Heart Association.
VSD: Ventricular septal defect.

artery bypass grafting was performed in 15 patients (65.2%), with a mean of 1.5 ± 0.7 distal anastomoses.

Emergency surgery was performed for all patients. An intra-aortic balloon pump (IABP) was inserted in all patients prior to surgery. The median time interval between the diagnosis of VSD and surgery was 3hours (2, 26). All patients underwent VSD repair within 2 days of diagnosis.

Ten patients died within 30 days of operation, giving an operative mortality of 43.5%. There were 3 intra- operative mortalities. There was no significant difference in the duration of aortic cross clamping between survivors and non-survivors, (92 ± 16 minutes vs. 90 ± 20 minutes respectively, P=0.672).

Pre-existing medical conditions did not significantly affect

**Table 3: Logistic Regression for Operative Mortality**

| Variable | Odds ratio (OR) | 95% confidence | P-value |
|---|---|---|---|
| Post-operative RRT | 26.4 | 2.7 – 260.9 | 0.005 |
| NYHA Class IV | 20.3 | 0.3 – 320.0 | 0.033 |

NYHA: New York Heart Association. RRT: Renal replacement therapy.

early mortality. Significant predictors of operative mortality identified using univariate analysis included emergency operation, NYHA class, inotropic support, right ventricular dysfunction, EuroSCORE II, intra-operative red blood cell transfusion, post-operative renal failure and RRT. Logistic regression for multivariate analysis showed that the NYHA class at presentation and post-operative RRT were index-pendent risk factors for operative mortality, as shown in Table 3.

Post-operative echocardiography diagnosed a residual shunt of various degrees in 3 patients (13%) which were managed conservatively.

## DISCUSSIONS

The availability of early reperfusion therapy for myocardial infarction has led to the declining incidence of post-infarction VSDs. Despite this trend, this remains one of the most challenging conditions encountered by cardiac surgeons today.

The 30-day operative mortality in this series was 43.5%, similar to the rates of 34% to 37% reported in other series.[4-7] Lower operative mortality rates ranging from 19% to 23% have been reported.[8-11] Univariate analysis identified emergency operation as a predictor of early mortality in this series, which was similar to the findings of other authors.[4,6,9,10] The same authors also identified the dur- ation of aortic cross clamp as a predictor of early mortality but this association was not demonstrated in this study. In-creased cardiopulmonary bypass time has also been reported as a predictor of operative mortality but this asso- ciation was not evident in this series.[11,19-21]

The optimal timing of surgical repair for VSDs is critical. A longer interval before surgery has been reported to be associated with improved survival.[22] Despite this, none of our 23 patients remained sufficiently stable in NYHA class II to permit delayed surgery.

We adopted the single patch technique in all cases. The incidence of post-operative residual shunt was 13%, comparable to the incidence of 24 to 26% described in other series in which a variety of techniques were used [6,23]. An alternative double patch technique has been reported to decrease the incidence of residual shunt from 11% to 0%.[24]

We routinely perform coronary angiography for all patients diagnosed with VSD at our center. In this series, coronary angiography was performed in SVD was significantly more common in anterior VSDs compared to posterior VSDs in this series, similar to the findings of Davies et al.[25] in the group with SVD, 75% had total occlusion of the artery to the infarcted area. Reported figures range from 57% to 82%.[2,26]

Lundblad et al.[27] found that concomitant CABG during VSD repair reduces both early and late mortality when compared with patients with unbypassed coronary artery disease. Although there was no significant difference in early mortality within other series, mid to long- term survival benefit has been reported .[23,27-29]

In a review of recent literature, Perotta et al.[30] reported an improvement of mortality rates from 26.3% in those without CABG to 21.2% in those who had undergone CABG. These results applied to patients with multi-vessel disease where complete myocardial revascularization was achieved by bypassing all stenotic coronary arteries supplying non-infarcted areas. Actuarial survival at five years from this series ranged from 29% to 72%. In this study, there was no statistically significant early or long-term survival advantage for patients who had undergone concomitant CABG. Other authors have reported similar findings.[4,6,7,10,11,22,24,31] Despite the lack of significant survival benefit associated with concomitant CABG in their studies, some authors advocate concomitant CABG during VSD repair as long as it can be performed safely.[4,5,22,24] The aim is to reduce further ischemic risk associated with multi-vessel coronary artery disease by improving collateral flow to the myocardium.

Similar to the findings of Moore et al. [32], our data shows that RV dysfunction has a negative impact on early and late survival. As RV failure ensues, the left-sided cardiac chambers are unable to fill, leading to biventricular failure. This exacerbates the low cardiac output state, contributing towards a higher mortality rate.

Percutaneous closure has been increasingly used in patients with post infarction VSDs, initially in patients with recurrent ventricular septal defects (VSDs) after primary surgical repair, but more recently as primary therapy in patients with acute VSR and high surgical risk, or as a temporizing bridge to surgery.[12]

La Torre et al.[33] reported a similar bridge to surgery strategy in their series of patients with posterior VSRs using the Impella Recover (ABIOMED, Inc., Danvers, Mass), a percutaneous left ventricular assist device (LVAD). Utilizing an intravascular micro axial blood pump inserted in the femoral artery, successful reduction in left-to-right shunting and increase in cardiac output was achieved while awaiting VSD repair about 3 weeks later. Despite successful surgical repair of VSD, some patients with extensive infarction and persistent ventricular failure remain in cardiogenic shock and require VAD support to maintain physiologic hemodynamics. Implantable LVADs have been used successfully to bridge these patients to cardiac transplantation.[34]

This is a retrospective descriptive study with inherent biases in data collection. Due to the relatively rare occurrence of post-infarction VSD, the small sample size underpowered the statistical analysis and could have limited the number of statistically significant variables. A prospective multi-center study incorporating a larger sample size would be useful to assess the prognostic value of the risk factors identified.

## CONCLUSION

Post-infarction VSD remains a serious and challenging complication of acute myocardial infarction in the modern surgical era. Surgical repair is associated with an operative mortality of 43.5%. The NYHA class at presentation and the need for post-operative RRT are independent predictors of early mortality. Concomitant CABG during VSD repair does not confer a significant early survival advantage.

### Abbreviations

AMI: Acute myocardial infarction; BiVAD: Biventricular assist device; CABG: Coronary artery bypass grafting; CVVH: Continuous veno-venous hemofiltration; CVVHDF: Continuous veno-venous haemodiafiltration; DVD: Double vessel disease; IABP: Intra-aortic balloon pump; LVAD: Left ventricular assist device; NYHA: New York Heart Association; RRT: Renal replacement therapy; RV: Right ventricle; STEMI: ST-elevation myocardial infarction; SVD: Single vessel disease; TTE: Transthoracic echocardiography; TVD: Triple vessel disease; VSD: Ventricular septal defect; VAD: Ventricular assist device.

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