In-hospital outcome of patients with post-MI VSD: a single-center study

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Abstract

Introduction: Ventricular septal defect (VSD) is a rare but life-threatening complication of acute myocardial infarction (AMI) [1, 2]. Early reperfusion therapy including thrombolysis and primary percutaneous coronary intervention (PPCI) has reduced the incidence of this complication from 1–2% to 0.2% [2–4]. However, the mortality rate of VSD associated with conservative or surgical management is still high [5]. There is a paucity of data regarding the natural history of this devastating complication of myocardial infarction in the Middle East region with restricted financial resources and unsolved major health problems. For example, about


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10–15% of the Iranian population have no health insurance coverage at all [6]. Not surprisingly, health expenditure per capita is about US$ 350 in Iran, far less than European and North American countries [7]. Also, there are limited data regarding outcome and valuation of cardiac surgical procedures in Iran as one study reported in-hospital mortality of 0.47% for isolated coronary artery bypass grafting (CABG) surgery [8] but the presentation and management of post-MI VSD remain largely unknown.

**Table I. Baseline characteristics of patients**

| Parameter                        | Value      |
|----------------------------------|------------|
| Age, mean ± SD [years]           | 71.62 ±9.38|
| Gender, n (%)                    |            |
| Male                             | 27 (42.2)  |
| Female                           | 37 (57.8)  |
| Diabetes mellitus, n (%)         |            |
| Hyperlipidemia, n (%)            |            |
| Hypertension, n (%)              |            |
| Renal failure, n (%)             |            |
| Previous myocardial infarction or angina, n (%) | 16 (25) |
| Systolic blood pressure [mm Hg]  | 100.98 ±21.69|
| Diastolic blood pressure [mm Hg] | 65.28 ±13.64|
| Heart rate [bpm]                 | 94.04 ±20.97|
| VSD location, n (%)              |            |
| Anterior                         | 53 (82.81)|
| Non-anterior                     | 11 (17.19)|
| Number of diseased vessels, n (%)|            |
| 1                                | 19 (29)    |
| 2                                | 27 (42)    |
| 3                                | 18 (29)    |
| Culprit vessel                   |            |
| LAD                              | 53 (82.81)|
| LCX                              | 3 (4.68)   |
| RCA                              | 8 (12.5)   |
| Reperfusion, n (%)               | 22 (34.4)  |
| Type of reperfusion, n (%)       |            |
| Thrombolysis                     | 20 (31.3)  |
| Primary PCI                      | 2 (3.1)    |

VSD – ventricular septal defect, PCI – percutaneous coronary intervention, LAD – left anterior descending artery, LCX – left circumflex artery, RCA – right coronary artery.

0.65%. The present study was approved by the ethics committee of Tabriz University of Medical Sciences (Study No. 94/1-7/4). Written informed consent was obtained from all patients or the patient’s relatives. The diagnosis of AMI was based on prolonged anginal chest pain, electrocardiographic signs of infarction, and a documented rise of cardiac enzymes. The VSD was diagnosed by transthoracic echocardiogram as disrupted ventricular septum with evidence of left-to-right shunt by color Doppler in all cases. Left ventricular systolic function was calculated using Simpson’s method.

Demographic data, coronary risk factors, Killip class, hemodynamic status on hospital admission, reperfusion therapy, and angiographic data including culprit vessel, echocardiographic findings, and intra-aortic balloon pump usage were recorded. Long-term follow-up was done by phone contact and regular visits among patients who were discharged home alive. All patients underwent coronary angiography. Coronary artery stenosis more than 70% was considered significant and multivessel coronary artery disease was defined as stenosis of > 70% in two or more major coronary arteries. Right heart catheterization was performed in 34 patients. Left-to-right shunt estimation was performed by catheterization data or echocardiographic finding. Cardiogenic shock was defined as systolic blood pressure less than 90 mm Hg associated with signs of hypoperfusion including cold extremities, altered mental status, and reduced urine output [9]. Renal failure was defined as serum creatinine more than 1.5 mg/dl or need for renal replacement therapy. The primary outcome was in-hospital mortality. Also, several demographic, hemodynamic, angiographic, and echocardiographic variables were compared between in-hospital survivors and the non-survivor group and between the surgical treatment group and the medical treatment group.

**Statistical analysis**

Categorical variables were expressed as a percentage and continuous variables were presented as mean ± standard deviation (SD). The chi-square ($\chi^2$) or Fisher’s exact test was used to compare categorical variables and the Mann-Whitney U-test and Student’s t-test were used to compare continuous variables. Predictors of mortality in univariate analysis with a $p$-value less than 0.05 were included in the multivariate model. Multivariate logistic regression was used to estimate independent risk factors for the factors with a significant $p$-value (≤ 0.05) through a univariate analysis. $P$-values less than 0.05 were considered statistically significant. All data were analyzed using SPSS version 18 (SPSS Inc, Chicago, IL).

**Results**

The mean age of the patients was 71.62 ±9.38 years with 57.8% females. Demographic, hemodynamic, echocardiographic, and angiographic findings are shown in Table I. Most patients were above 60 years old (85%). The VSDs were anterior in 53 (82.81%) patients. The VSD size in most cases was > 10 mm. A total of 41 (64.1%) patients were in cardiogenic shock during initial presentation. Totally,
22 patients developed post-MI VSD after reperfusion therapy. Twenty patients received thrombolysis, of whom 15 were referred from another center after lytic therapy and development of VSD. Five patients received thrombolysis in our center and subsequently were found to have post-MI VSD. Mean door-to-needle time was 89 ± 13 min. Two patients developed VSD after being treated with primary angioplasty in our center. These 2 patients presented more than 12 h after initiation of chest pain and mean door-to-balloon time was 101 ± 4 min. Forty-two patients did not receive any type of reperfusion therapy, of whom 11 patients were referred from other centers and the remainder presented initially at our hospital. Because of the poor hemodynamic status of most patients, apart from ASA, statin, IV nitrate, furosemide and inotropes no other medication was used except clopidogrel in 2 patients who underwent coronary angioplasty. Multivessel coronary artery disease was found on coronary angiography in 70.3% of patients. Preoperative mean pulmonary artery pressure was 44.1 ± 7.9 mm Hg and mean left to right shunt was 3.5 ± 1.2.

An intra-aortic balloon pump (IABP) was used in 30 (46%) cases. Other types of mechanical circulatory support were not available in our center mainly due to limited financial resources. Surgical treatment was performed in 23 (35.9%) out of 64 cases including VSD repair in 8 (12.5%) patients and coronary artery bypass graft (CABG) (mean 1.4 grafts per patient) with VSD repair in 15 (23.4%) patients. All operations were performed via median sternotomy and hypothermic cardiopulmonary bypass was applied. Myocardial protection was provided with cold cardioplegia. Mean cardiopulmonary bypass time was 153 ± 42.1 min and mean aortic cross clamp time was 74 ± 37 min. All grafts were saphenous veins. The two main techniques used for surgical repair were: 1) infarct excision (Daggett procedure) [10] with single Dacron patch repair in 14 patients and 2) infarct exclusion (David procedure) [11, 12] with bovine pericardial patch repair in 9 patients. Sandwich repair was not performed in these series as our surgeons are not familiar with this technique [13, 14]. Daggett procedure and David approach had a mortality rate of 57.1% (8/14) and 55.5% (5/9) respectively. All of the surgical interventions were done during the index hospitalization and were not delayed. Thirteen (56.5%) patients of the surgically treated group died. The remaining patients (41/64) did not receive surgical treatment; all but one of them died (97.65%). The main reasons for not performing surgical treatment were as follows: Ten patients refused surgery. In the remaining patients, the treating surgical team considered surgery too high risk mainly due to unstable hemodynamics and extreme frailty and advised conservative management. None of our patients underwent device closure. In-hospital mortality was 82.8%. Mean duration of hospital stay was 6.96 ± 5.63 days. Long-term follow-up showed that all 11 patients who survived the hospital course were alive at the time of writing this report and had NYHA FC II–III symptoms. We also compared baseline characteristics between surgical and medical treatment groups and it revealed that patients who underwent surgery were younger, had more preserved left ventricular function and more received IABP than patients with conservative management. Also male gender and single vessel coronary involvement were more common in this group (Table II).

Table III demonstrates baseline and clinical findings between survivor and non-survivor groups. Univariate predictors of in-hospital mortality were as follows: advanced age, female gender, lower systolic blood pressure at initial presentation, multivessel coronary stenosis, cardiogenic shock and no surgical treatment. The six above-mentioned factors were included in the multivariate analysis to predict independent factors associated with in-hospital mortality. Multivariate logistic regression analysis revealed cardiogenic shock as an independent predictor of in-hospital mortality and surgical treatment as a predictor of in-hospital survival (Table IV).

The mortality rate among patients with and without cardiogenic shock was 39/41 (95.1%) and 14/23 (60.9%), respectively ($p = 0.001$).

Discussion

The main findings of the present study are as follows: 1) post-MI ventricular septal defect had extremely high in-hospital mortality; 2) more than half of the patients in the present study were in cardiogenic shock in hospital admission; 3) cardiogenic shock was the only independent predictor of hospital death and surgical treatment as the main predictor of survival; 4) overall, fewer sick patients underwent surgery as evident by younger age, better left ventricular function, more common single vessel disease and male gender and IABP insertion was more common in this group.

Our study showed high in-hospital mortality among patients with a post-MI ventricular septal defect. This result is in line with some prior studies [15–19] while contradicting others [20–23]. The causes of high mortality rate in the present study may be related to multiple factors including older age, unstable hemodynamics, advanced coronary involvement, and underuse of support devices. Like previous studies [24, 25], our cohort mainly consisted of older patients and females, which are known risk factors for mortality in post-MI VSD. The other main cause of high mortality is compromised hemodynamic status during initial presentation. More than 60% of patients in the present study were in cardiogenic shock at hospital admission and, like previous reports, this independently predicted in-hospital mortality [26–29]. Cardiogenic shock and low blood pressure usually lead to more left ventricular dysfunction due to reduced coronary perfusion, which is followed by multi-organ failure and death.

Unlike some previous studies [2, 18, 19, 26, 30] but in agreement with others [15, 20, 23, 31, 32], multivessel coronary artery disease was more common in our cohort and was associated with in-hospital mortality in the univariate analysis. Advanced coronary artery stenosis may induce global left ventricular ischemia and necrosis and thus lead to the development of pump failure and cardiogenic shock.
One possible explanation for high in-hospital mortality in the present study may be the underuse of mechanical support devices including the IABP. Mechanical support devices may have a role in the management of mechanical complication of acute myocardial infarction as a bridge to surgery [33–35]. Less than half of our patients received an IABP while other types of support devices such as extracorporeal membrane oxygenation (ECMO) and ventricular assist devices (VAD) were not available during the study period in our hospital mainly due to limited financial resources. Thus, the high mortality rate of patients is not surprising. Another probable main reason for the complicated hospital course in the present study may be related to the more conservative treatment of the entire cohort. As most patients were critically unstable, medical treatment was of no value in them and failure of this approach led to high mortality. Based on Table II, other possible reasons for the lower survival rate in patients treated medically may be more advanced age and coronary artery disease in these patients as well as less cardiac reserve and less support with IABP. Results of the present study questioned the “late surgery approach” as many authors have recommend delayed surgery for healing of necrotic myocardium and stabilization of the patient [20, 22, 28, 36]. Based on the present study it seems unreasonable to delay surgery in patients with post-MI ventricular septal defect as these patients will never be really stabilized. So, early surgery planning after diagnosis of this devastating complication should be considered [37].

This study has some limitations. First, the research population was relatively small, limiting its statistical power. Second, this was a single-center nonrandomized observational retrospective study, but due to the grave nature of this complication of myocardial infarction, conducting a randomized trial in this setting may be very difficult or even impossible. Third, a small number of patients underwent surgery. There was no significant difference regarding mortality between two common procedures used by our surgical team and newer techniques like the sandwich procedure were not performed in the period of the study, which may be responsible for higher mortality even in surgically treated patients. So, the impact of surgical approaches and techniques needs further large scale studies.

Table II. Baseline characteristics in surgical versus medical groups

| Parameter                                | Surgical group (n = 23) | Medical group (n = 41) | P-value |
|------------------------------------------|-------------------------|------------------------|---------|
| Age [years]                              | 67.43 ±9.33             | 73.97 ±8.67            | 0.006   |
| Gender                                   |                         |                        |         |
| Male                                     | 14 (60.9%)              | 13 (31.7%)             | 0.02    |
| Female                                   | 9 (39.1%)               | 28 (68.3%)             |         |
| Diabetes mellitus                        |                         |                        |         |
| Male                                     | 6 (26.1%)               | 6 (14.6%)              | 0.26    |
| Female                                   | 13 (56.5%)              | 26 (63.4%)             | 0.58    |
| Hyperlipidemia                           | 7 (30.4%)               | 10 (24.4%)             | 0.59    |
| Previous MI                              | 4 (17.4%)               | 12 (29.3%)             | 0.29    |
| Systolic blood pressure [mm Hg]          | 104.69 ±19.15           | 98.90 ±22.95           | 0.30    |
| Diastolic blood pressure [mm Hg]         | 67.73 ±13.33            | 63.90 ±13.67           | 0.28    |
| Heart rate [bpm]                         | 95.86 ±18.56            | 93.02 ±22.36           | 0.60    |
| MI                                        |                         |                        |         |
| Anterior                                 | 19 (82.6%)              | 34 (82.9%)             | 0.97    |
| Non-anterior                             | 4 (17.4%)               | 7 (17.1%)              |         |
| Killip class                             |                         |                        |         |
| I, II                                    | 5 (21.7%)               | 11 (26.8%)             | 0.65    |
| III, IV                                  | 18 (78.3%)              | 30 (73.2%)             |         |
| Number of diseased vessels               |                         |                        |         |
| Single vessel                            | 11 (47.8%)              | 8 (19.5%)              | 0.01    |
| Multivessel                              | 12 (52.2%)              | 33 (80.5%)             |         |
| Reperfusion                              |                         |                        |         |
| > 45%                                    | 6 (26.1%)               | 3 (7.3%)               | 0.03    |
| < 45%                                    | 17 (73.9%)              | 38 (92.7%)             |         |
| LVEF                                     |                         |                        |         |
| > 45%                                    | 4 (17.4%)               | 6 (14.6%)              | 0.77    |
| < 45%                                    | 19 (82.6%)              | 35 (85.4%)             |         |
| VSD size [mm]                            |                         |                        |         |
| 5–10                                     | 4 (17.4%)               | 6 (14.6%)              |         |
| > 10                                     | 19 (82.6%)              | 35 (85.4%)             |         |
| Time of onset of VSD [days]              |                         |                        |         |
| < 4                                      | 23 (100%)               | 37 (90.2%)             | 0.12    |
| ≥ 4                                      | 0                       | 4 (9.8%)               |         |
| IABP                                     | 16 (69.6%)              | 14 (34.1%)             | 0.006   |
| Cardiogenic shock                        | 14 (60.9%)              | 27 (65.9%)             | 0.69    |
| Renal failure                            | 5 (21.7%)               | 10 (24.3%)             | 1.0     |

MI – myocardial infarction, VSD – ventricular septal defect, LVEF – left ventricular ejection fraction, IABP – intra-aortic balloon pump.
Conclusions

Our study demonstrated an extremely high in-hospital mortality rate associated with VSD complicating myocardial infarction. Cardiogenic shock was the only independent predictor of in-hospital mortality and surgical treatment was a predictor of in-hospital survival.

Disclosure

The authors report no conflict of interest.

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