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Ventricular Septal Rupture after Acute Myocardial Infarction

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Abstract: Ventricular Sepal Defect (VSD) remains an infrequent but a serious, devastating and life-threatening complication of Acute Myocardial Infarction (AMI); our aims were study on Post-MI VSD and identifying risk factors of mortality. forty-one patients, admitted with Post-MI VSD in Shahid Madani hospital in Tabriz from March 2000 until March 2006, assessed and correlation among mortality with age, sex, Hypertension (HTN), Hyperlipidemia (HLP), previous MI, Diabetes Mellitus (DM), renal dysfunction, respiratory disease and clinical and paraclinical findings such as vital sings and Ejection Fraction (EF) considered. Patients age was between 46- 83 year with average age of 67.6± 9.2 year. Female to male ratio was 1.05: 1 and hospital mortality was 58.5%. From 24 patients who admitted with Killip classification above two, 87.5% expired ($p<0.0001$). From 20 patients with cardiogenic shock, 90% expired ($p<0.0001$). 83.3% from 28 patients who did not operated, was dead ($p = 0.02$). From 21 patients with systolic blood pressure under 90 mmHg, 85.7% expired ($p<0.0001$). Other factors such as age, sex, previous MI, HTN, HLP, DM, renal dysfunction, respiratory disease, Congestive Heart Failure (CHF), EF, type of surgery, thrombolytic therapy, smoking, location of MI, diastolic blood pressure, size of VSD has no correlation with mortality ($p>0.05$). Killip Classification 3 and 4, cardiogenic shock and early surgery are the most important factors in patients' prognosis.

Key words: Killip classification, ventricular sepal defect, cardiogenic shock, prognosis

INTRODUCTION

Post-myocardial infarction Ventricular Septal Defect (VSD) complicates approximately 2% of myocardial infarctions (Rhydwen *et al*., 2002). VSD remains an infrequent but a serious, devastating and life-threatening complication of Acute Myocardial Infarction (AMI) (Crenshaw *et al*., 2000; Juraszyñski *et al*., 2006; Anantharaman *et al*., 2004; Tanaka *et al*., 2002). It is estimated that 8-17% of all fatal MIs are the result of myocardial rupture. Despite improvements in medical therapy and in percutaneous and surgical techniques, mortality from this complication remains extremely high. These patients usually die immediately, even before a diagnosis can be confirmed (Yip *et al*., 2003).

Cardiac ruptures after AMI are classified as ventricular Free Wall Ruptures (FWR), Ventricular Septal Ruptures (VSR) and Papillary Muscle Ruptures (PMR). A combination of any 2 types of rupture is called "Ventricular Double Rupture;" (VDR) (Yip *et al*., 2003; Tanaka *et al*., 2003). While LV free wall rupture and VSD are uncommon mechanical complications after AMI, they carry an extremely high mortality rate (Crenshaw *et al*., 2000; Slater *et al*., 2000). Post-MI VSDs occur usually within 2 weeks after infraction (Juraszyñski *et al*., 2006). They results from perforation of the ventricular septum secondary to ischemic injury following AMI (Yonga *et al*., 2005) and in 60% of the cases are the result of completely occluded coronary artery supplying anterior wall of left ventricle and subsequent transmural MI. Fifty percent of patients die within the first week after the rupture unless they are surgically treated (Juraszyñski *et al*., 2006).

When ventricular septal rupture complicates AMI, the mortality is high. Reperfusion therapy has reduced the incidence of septal rupture. However, rapid diagnosis, aggressive medical management and surgical intervention are required to optimize recovery and survival (Yochai *et al*., 2002).

The goals of the present study were to evaluation of Post-MI VSD and to identifying correlation among mortality with age, sex, Hypertension (HTN), Hyperlipidemia (HLP), previous MI, Diabetes Mellitus (DM), renal dysfunction, respiratory disease and clinical and paraclinical findings such as vital sings and Ejection Fraction (EF) considered.

MATERIALS AND METHODS

The cross-sectional descriptive-analytic study was performed on patients with post-MI VSD in Tabriz Shahid Madani Heart Referral Center. All hospital records from March 2000 until March 2006 were assessed and 45 patients with initial diagnosis of post-MI VSD were found, of which the diagnosis were confirmed in 41 cases.

The patient characteristics were recorded in a questionnaire. These characteristics were including: patient name, file number, age, sex, admission date, date of discharge or death, clinical findings (such as vital signs, heart rate, blood pressure), NYHA and/or Killip score and other findings such as cardiogenic shock (before or after admission), use of inotropic agents and IABP, duration of ICU stay and hospital stay.

New York Heart Association (NYHA) functional and therapeutic classification for prescription of physical activity for cardiac patients is considered as following:

Class 1: Patients with no limitation of activities

Class 2: Patients with slight, mild limitation of activity

Class 3: Patients with marked limitation of activity

Class 4: Patients who should be at complete rest confined to bed or chair.

Killip classification is considered as following:

Class 1: Without pulmonary edema

Class 2: S3 or fine crackles in <1/3 of lungs

Class 3: Pulmonary edema

Class 4: Cardiogenic shock

Gradient of shunt, EF and VSD size measured by echocardiography, were recorded. Angiographic results were including the number and name of narrowed arteries and/or completely obstructed arteries. Narrowing of more than 75% was considered significant and 100% obstruction (without distal flow or with distal flow by collateral arteries) was considered as completely obstructed.

Findings related to MI were as following:

- The duration between MI to diagnosis of VSD (the MI initiation was considered as the initiation of chest pain).
- Use of thrombolytic therapy (using Streptokinase) in admission (indications were ST elevation >1 mv in two adjacent limb leads or >2 mv in 2 adjacent precordial leads, typical chest pain for >20 min and new LBBB in ECG).
- Location of MI: Anterior, posterior, inferior, posterior-inferior (according to the ECG).
- Location of VSD: anterior, posterior, inferior, apical (according to the echocardiography and ventriculography).

Findings related to operation were as following:

- Type of surgery (VSD repair only or concomitant VSD repair in association with CABG).
- Type of VSD repair (pericardial patch, Dacron patch, or gortex patch).
- Number of grafts (one, two, or three).
- Repeat surgery (for surgery complications such as bleeding or residential VSD confirmed with post operative echocardiography).
- Emergent or urgent surgery (surgery soon after admission or after the patient became stable in the same admission).

Post operative results were recorded as: renal failure (raised creatinin or need for dialysis), need for mechanical ventilation or tracheostomy, postoperative shock, need for IABP more than 2 days after operation, need for inotropic agents after operation, multi organ failure, arrhythmias and pace maker insertion,

The collected data were analyzed with SPSS-12 statistical software and the variables were compared with Chi-Square and ANOVA and Fishers exact test. The p-value = 0.05 was considered significant.

RESULTS

We studied 41 patients with post-MI VSD in this survey. The patients had the age range of 46-83 years and average age of 67.6±9.2 years. The baseline clinical characteristics of the 41 Patients are showed in Table 1.

Of all patients, 20 (48.8%) suffered from cardiogenic shock before or during hospitalization. Thirty three patients (80.5%) received inotropic agent. IABP was inserted for 19 patients (46.3%) (Fig 1). Blood pressure and heart rate of patients are showed in Table 2.

Table 1: Baseline clinical characteristics of the 41 patients

Variable	Number	Percent
Age		
40-49	2	4.87
50-59	4	9.75
60-69	17	41.5
70-79	15	36.58
80-90	3	7.31
Male	20	48.80%
Female	21	51.20%
Killip classification		
I	3	7.31
II	14	34.14
III	6	14.6
IV	18	43.9
NYHA classification		
I	7	17.07
II	20	48.78
III	14	34.1
IV	0	0

The history of previous disease was reported as following: MI in 6 (14.6%) patients, chest pain in 8 (19.5%), hypertension in 24 (58.5%), hyperlipidemia in 12 (29.3%), diabetes mellitus in 17 (41.5%), renal failure in 14 (34.1%), pulmonary disease in 3 (7.3%), chronic heart failure in 17 (41.5%) and smoking in 5 cases (12.2%).

Diagnosis were made by echocardiography in 26 patients (63.4%), echocardiography and angiography in 7 patients (17.1%) and echocardiography and clinical findings in 8 cases (19.5%). Other diagnostic findings of patients are listed in Table 3.

VSD size had been measured in 36 cases which were varying from 5-25 mm with average of 14.2±5.7 mm. Of all 41 patients 13 (31.7%) dead before angiography and of remaining 28 cases underwent angiography, 3 (10.7%) had significant LAD narrowness, 3 (10.7%) had concomitant LAD and LCX narrowness, 6 (21.4%) had concomitant LAD and RCA narrowness and 9 (32.1%) had 2 vessel disease.

Sixteen patients (57.1%) had 3 vessel diseases and 10 patients (35.7%) had at least 1 completely obstructed vessel.

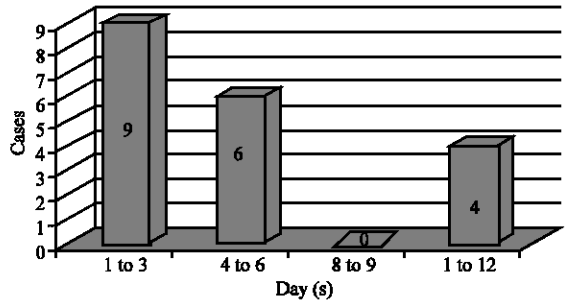

Fig. 1: Duration of IABP stay

Of all 41 patients, 17 (41.5%) had indication for thrombolytic therapy and received this therapy. Acute MI was anterior in 36 (87.8%), inferior in 3 (7.3%) and postero-inferior in 2 (4.9%). VSD was anterior in 3 (7.3%), posterior in 1 (2.4%), inferior in 4 (9.8%) and apical in 33 (80.5%) of cases.

Eleven cases (26.8%) were operated at the same admission, 8 (19.5%) planed for elective surgery of which only 2 cases had been presented later for surgery. Of 13 operated patients, 6 cases (46.2%), underwent only VSD repair, 7 (53.8%) underwent concomitant CABG and VSD repair. Two cases (15.4%) received single graft, 2 (15.4%) received double graft and 3 (23.1%) received 3 grafts. Pericardial patch was used in 3 cases (23.07%), gortex patch in 7 (53.8%) and Dacron patch in 2 cases (15.4%).

Of 13 operated patients, 2 (15.4%) underwent repeat surgery, one because of mediastinitis and one because of bleeding. Post operative echocardiography showed that 5 patients (38.5%) had remaining VSD whit QP/QS less than 2. These patients did not go under repeat operation and followed up.

Post operative complications were as following: renal failure in 4 (30.8%), need for mechanical ventilation in 9 (69.2%) for 2-9 days, need for tracheostomy in 1 (15.3%), shock in 9 (69.2%), multi organ failure in 4 (30.8%) and arrhythmia with subsequent death in 4 (30.8%).

Duration of hospital stay was 1-51 days and ICU stay was 1-29 days with average of 11.5±11.7 days and 10.1±7.5 days, respectively.

Of all 41 patients, 24 (58.5%) were dead. As showed in Table 4, the relation of death with Killip classification, cardiogenic shock and systolic blood pressure was significant, but the relation of death with other variables was not significant.

Table 2: Blood pressure and heart rate of patients

Heart rate		Systolic blood pressure		Diastolic blood pressure	
Range (beat/min)	Number (%)	Range (mmHg)	Number (%)	Range (mmHg)	Number (%)
60-69	2 (4.87%)	60-69	1 (2.44%)	20-29	1 (2.44%)
70-79	2 (4.87%)	70-79	4 (9.75%)	30-39	4 (9.75%)
80-89	6 (14.63%)	80-89	6 (14.63%)	40-49	1 (2.44%)
90-99	2 (4.87%)	90-99	10 (24.4%)	50-59	2 (4.87%)
>100	29 (70.7%)	100-109	8 (19.5%)	60-69	19 (46.3%)
		110-119	4 (9.75%)	>70	14 (34.14%)
		>120	8 (19.5%)		
Average	99.8±17.04	Average	99.7±22.8	Average	62.93±16.73

Table 3: Ejection fraction, shunt gradient and MI to VSD diagnosis time

Ejection fraction		Shunt gradient		MI to VSD diagnosis (day)	
Range	Number (%)	Range	Number (%)	Range	Number (%)
10-19%	2 (4.87%)	0-19	8 (19.5%)	0-2	28 (68.3%)
20-29%	12 (29.2%)	20-39	18 (51.4%)	3-5	6 (14.6%)
30-39%	18 (43.9%)	40-59	9 (21.9%)	6-8	4 (9.75%)
40-50%	9 (21.9%)	60-79	5 (12.19%)	9-11	2 (4.87%)
Average	32.3±8.07	80-100	1 (2.9%)	>12	1 (2.4%)

Table 4: The relation between death and some variables

Variable	Number (%)	Death rate	p value
Killip classification			
I or II	17 (%)	3 (%)	<0.0001
III or IV	24 (%)	21 (87.5%)	
Thrombolytic therapy			
Yes	17 (%)	13	
No	24 (%)	11 (45.8%)	0.062
Cardiogenic shock			
Yes	20 (48.8%)	18 (90%)	
No	21 (51.2%)	6 (28.6%)	<0.0001
Surgery			
Yes	13 (%)	4	
No	28 (%)	20 (71.4%)	0.02
Sex			
Male	20 (%)	12 (60%)	
Female	21 (%)	12 (57.1%)	1
Previous MI			
Yes	6 (%)	1 (16.7%)	
No	35 (%)	23 (65.7%)	0.066
History of chest pain			
Yes	8(%)	3 (37.5%)	
No	33(%)	21 (63.6)	0.241
Hypertension			
Yes	24 (%)	12 (50%)	
No	17 (%)	12 (70.6%)	0.217
Hyperlipidemia			
Yes	12 (%)	5 (41.7%)	
No	29 (%)	19 (65.5%)	0.184
Diabetes mellitus			
Yes	17 (%)	10 (58.8%)	
No	24 (%)	14 (58.3%)	1
Renal failure			
Yes	14 (%)	9 (64.3%)	
No	27 (%)	15 (55.6%)	0.742
Pulmonary disease			
Yes	3 (%)	2 (66.7%)	
No	38 (%)	22 (57.9%)	1
Smoking			
Yes	5 (%)	3 (60%)	
No	36 (%)	21 (58.3%)	1
Type of operation			
Only VSD repair	6 (%)	1 (16.7%)	
CABG and VSD repair	7 (%)	3 (42.9%)	0.559
Location of MI			
Anterior	25 (%)	15 (60%)	
Other locations	16 (%)	9 (56.3%)	1
Systolic blood pressure			
>90 mm Hg	20 (%)	6 (30%)	
<90 mm Hg	21 (%)	18 (85.7%)	0.0001
Diastolic blood pressure			
>75 mm Hg	8 (%)	3 (35.5%)	
<75 mm Hg	33 (%)	21 (63.6%)	0.241

Also, the relation between age and death (p = 0.347), EF and death (p=0.699), shunt gradient and death (p=0.08) and VSD size and death (p = 0.474) was not significant.

DISCUSSION

The interventricular septum receives its arterial blood supply from perforating branches of the left anterior descending and posterior descending arteries. Despite this dual blood supply, septal collateral flow is limited. Consequently, the septum remains vulnerable to ischemia and postnecrotic rupture (Calderon and Ott, 1991).

Advanced age (60-69 years) is a risk factor for septal rupture in the era before thrombolytic therapy (Yochai *et al*., 2002; Prêtre *et al*., 2000). In a study by Deja *et al*. (2000) on 110 consecutive patients underwent postinfarction VSD repair had the mean age of 65.5±7.8. Our patients had the age range of 46-83 years and average age of 67.6±9.2 years.

Advanced age has been found to be an increased risk factor for adverse outcomes after AMI (Yip *et al*., 2003). Ventricular septal rupture occurs more frequently in the elderly (Teo *et al*., 1990). In the present study, old age was an independent predictor of cardiac rupture following AMI. Morbidity and mortality after AMI have been reported to be higher in women than in men (Yip *et al*., 2003). Previous investigations have found female sex to be a risk factor for development of VSD following AMI (Crenshaw *et al*., 2000; Yip *et al*., 2003; Yochai *et al*., 2002; Prêtre *et al*., 2000; Barker *et al*., 2003). In Crenshaw *et al*. (2000) study, the female to male ratio was 1.33 to 1. Conversely, this ratio in Rhydwen *et al*. (2002) study was 1-2.6. However, in our series there was not any significant difference between two sexes and the female to male ratio was 1.05 to 1. Also, no correlation has been demonstrated between the risk of early death and age or sex (Yochai *et al*., 2002) and our study could not find significant relation between age or sex and death.

In our study, the heart rate of patients was 60-140 beat/min with average of 99.8±17.04 beat/min and majority had the heart rate of >100 beat/min. In the study by Crenshaw *et al*. (2000) average heart rate of AMI patients with and without VSD was 81 beats/min and 74 beats/min respectively. In Rhydwen *et al*. (2002) study, 62% of patients had the heart rate of >100 beat/min and they concluded that the heart rate of >100 beat/min is a risk factor of VSD development after AMI. Our study findings support this conclusion, although there is need for prospective studies to confirm this association.

In our study, the Systolic Blood Pressure (SBP) of patients was 60-160 mm Hg with average of 99.7±22.8 mmHg. Also, the Diastolic Blood Pressure (DBP) of patients was 25-100 mm Hg with average of 62.93±16.73 mm Hg. In the study by Crenshaw *et al*. (2000) average SBP and DBP of patients was 127 mm Hg and 80 mm Hg, respectively. In Rhydwen *et al*. (2002) study, 15.5% of patients had the SBP of >90 mm Hg and 19% had the SBP of <90 mmHg. Previous studies have reported a bidirectional association of SBP and DBP at enrollment with the incidence of VSD. The positive correlations (increase in the incidence of VSD as SBP increased to >130 mm Hg and the DBP to >75 mm Hg) reflect the association between hypertension and VSD. Extensive MI and right ventricular involvement, both known

risk factors for VSD, may cause hypotension and cardiogenic shock on admission. The negative correlations between enrollment SBP (=130 mm Hg) and DBP (=75 mm Hg) with the incidence of VSD probably reflect the incidence of hemodynamic compromise associated with extensive MI or right ventricular infarction (Crenshaw *et al*., 2000). In the GUSTO-I trial, there was a nonlinear relation between the SBP and DBP at enrollment and septal rupture, since hypertension (a blood pressure of more than 130/75 mm Hg) and extensive MI and right ventricular infarction (which are causes of hypotension) are also risk factors for septal rupture (Yochai *et al*., 2002). Nonsurvivors also were older and more likely to have reduced blood pressure at enrollment (Crenshaw *et al*., 2000). In our series, the mortality was decreased significantly with SBP >90 mm Hg in admission, but the relation between DBP on admission and mortality was not significant.

The average Ejection Fraction (EF) in our patients was 32.3±8.07 and majority of patients had the EF 30-39%; similarly, in Crenshaw *et al*. (2000) study the average EF was 40 and majority of patients had the EF 30-50%. We did not find any relation between mortality and EF or VSD size. The immediate preoperative hemodynamic status is a major determinant of the postoperative outcome, rather than the EF or the size of the intracardiac shunt (Yochai *et al*., 2002).

Killip classification of our patients in admission was as following: 1 (7.31%), 2 (34.14%), 3 (14.6%) and 4 (43.9%). In comparison, this classification in Crenshaw *et al*. (2000) study was as following: 1 (74%), 2 (16%), 3 (7%) and 4 (2%); these findings suggest that our patients present in later stages. In the GUSTO-I trial, all 8 patients with septal rupture who were in Killip class III or IV at presentation died, as compared with 53 of 74 patients (72%) who were in Killip class 1 or 2 at presentation (Yochai *et al*., 2002). In our study 17.6% of patients in Killip 1 and 2 and 87.5% of patients in Killip 3 and 4 were dead. All patients in Crenshaw *et al*. (2000) study with Killip class 3 or 4 at presentation died.

In our study, 14.6% had the previous MI and 19.5% had the history of chest pain. In Crenshaw *et al*. (2000) study, only 12% had the previous MI and in Rhydwen *et al*. (2002) study, only 7% had the history of chest pain. Ventricular septal rupture occurs more frequently in those presenting with the first MI (Calderon and Ott, 1991; Teo *et al*., 1990). Very few have a prior history of stable angina pectoris before the MI (Teo *et al*., 1990). The absence of a history of angina or MI is a risk factor for septal rupture. Angina or infarction may lead to myocardial preconditioning as well as to the development of coronary collaterals, both of which reduce the likelihood of septal rupture (Yochai *et al*., 2002; Barker *et al*., 2003).

In our series, the history of hypertension, hyperlipidemia and diabetes mellitus was 58.5, 41.5 and 18% respectively. These percents in Rhydwen *et al*. (2002) study were 41, 24 and 21%, respectively. In Crenshaw *et al*. (2000) study, 48% of patients had the history of hypertension and 18% had the history of hyperlipidemia. Others report that renal failure and diabetes mellitus are strong negative predictors of survival after surgery (Yochai *et al*., 2002). However, our findings did not show any significant relation between mortality and history of hypertension, hyperlipidemia and diabetes mellitus and renal failure. Also, as other studies (Rhydwen *et al*., 2002; Crenshaw *et al*., 2000; Yochai *et al*., 2002) we concluded that the absence of smoking is often associated with an increased risk of septal rupture.

Our study was consistent with other studies that suggest septal rupture occurs more frequently with anterior than other types of AMI (Yochai *et al*., 2002; Crenshaw *et al*., 2000; Birnbaum *et al*., 2000). Anterior location of infarction is one of the most important predictors of this complication. After VSD has developed, inferior location of infarction may be one of the most important prognostic factors in this patient population (Crenshaw *et al*., 2000). Patients with inferior infarcts and VSDs tended to have a worse outcome than those with anterior infarcts (Crenshaw *et al*., 2000; Yochai *et al*., 2002). However, our findings did not show significant relation between mortality and site of AMI.

VSD was apical, inferior, anterior and posterior in 80.5, 9.8, 7.3 and 2.4%; These ratios in Calderon *et al*. (1991) study (12) were as following: apical (39.2%), infero-posterior (42.8%) and anterior (17.8%). The apical type was more prevalent in our study.

Without reperfusion, septal rupture generally occurs within the first week after infarction (Yochai *et al*., 2002; Barker *et al*., 2003). There is a bimodal distribution of septal rupture, with a high incidence on the first day (Crenshaw *et al*., 2000) and on days 3 through 5 and rarely more than two weeks after infarction. The median time from the onset of symptoms of AMI to rupture is generally 24 h or less in patients who are receiving thrombolysis. The median time from the onset of infarction to septal rupture was 1 day in the GUSTO-I trial, 16 h in the Should We Emergently Revascularize Occluded Coronaries in Cardiogenic Shock (SHOCK) trial (Juraszyñski *et al*., 2006), 52.3 h (range, 4.8 h to 5 days) in Yip *et al*. (2003) study and 5.6+/-7.8 days (median 4) in Deja *et al*. (2000) study. Clinical trials also have demonstrated that although the frequency of cardiac rupture decreases, early thrombolytic therapy (i.e., within 6 h) paradoxically accelerates the timing of this complication and late thrombolytic therapy has been found to increase the risk of cardiac rupture. Several possible mechanisms, including

extension of myocardial hemorrhage, weakening and dissection of the necrotizing zone, diminishing of the myocardial collagen content and digestion of collagen by collagenases and plasmin, have been suggested (Rhydwen *et al*., 2002; Crenshaw *et al*., 2000; Yip *et al*., 2003; Yochai *et al*., 2002). In our patients, VSD was developed within the first week after AMI, although we did not compare this time in patients received thrombolytic therapy or not received it.

In our series, 57.1% had three vessel coronary artery diseases, 32.1% had 2 vessel diseases, 10.7% had one vessel diseases and 35.7% had at least one completely obstructed vessel. These values in Crenshaw *et al*. (2000) study were 4, 15, 19 and 25%, respectively. In Calderon and Ott (1991) study, almost 60% of the patients with postinfarction VSD have single-vessel disease. So, in our study, postinfarction VSD is more associated with three vessel diseases.

Patients with a postinfarction VSD usually present with sudden cardiogenic shock (Davies and Westaby, 1990) which is a poor prognostic factor (Barker *et al*., 2003; Sasseen *et al*., 2003). In our study, 48.8% of patients had cardiogenic shock. Ninety percent of patients with cardiogenic shock in comparison with 28.6% of patients without cardiogenic shock were dead. Calderon and Ott studied 28 patients underwent surgical repair for VSD after AMI. Cardiogenic shock was the most common predictor of a poor prognosis. Therefore, in order to avoid this complication, they recommend immediate surgical repair of postinfarction VSD (Calderon *et al*., 1991). In the SHOCK trial, the in-hospital mortality rate was significantly higher among patients in cardiogenic shock as a result of septal rupture than among patients with all other categories of shock (87.3%, as compared with 55.1-59.2%) (Yochai *et al*., 2002; Hochman *et al*., 2000). Cardiogenic shock prior to surgery adversely influenced early survival. Deterioration of haemodynamic status in between admission and surgery is stronger predictor of mortality than shock on admission. Preoperative cardiogenic shock affected late survival. Achieving haemodynamic stability prior to surgery may be beneficial but prolonged attempts to improve patients' cardiovascular state are hazardous (Deja *et al*., 2000).

In our study, 41.5% had CHF, 2% underwent pace maker insertion, 69.2% underwent mechanical ventilation and 46.3% underwent Intraaortic Balloon Pump (IABP) insertion. These values in Crenshaw *et al*. (2000) study were 59, 27, 49 and 57%, respectively. In a study by Deja *et al*. (2000) on 110 consecutive patients who underwent postinfarction VSD repair, 66 patients had IABP inserted and 15 were ventilated preoperatively. Intra-aortic balloon counterpulsation is especially useful (Teo *et al*., 1990). Yochai *et al*. (2002) found that preoperative use of an IABP reduced immediate postoperative mortality, but it was not associated with an improved long-term prognosis.

In our study, 30.8% of patients had post-operation arrhythmia which is similar to other studies (Barker *et al*., 2003). Reoperation rate in our study was similar to prior studies, although ICU stay and hospitalization were longer (Barker *et al*., 2003).

Repair of post infarction VSD is still a challenging procedure with a high risk of recurrence of the VSD and subsequent mortality. Labrousse *et al*. (2002) studied 85 patients operated post infarction VSD with Double patch technique. Concomitant CABG was not associated with higher hospital mortality and long-term survival rate was similar in patients with or without concomitant CABG. Concomitant coronary revascularization is recommended for patients in whom disease is present in another coronary territory (Calderon and Ott, 1991). In our study, 31.7% of patients were operated, of which 46.2% underwent only VSD repair and 53.8% underwent concomitant CABG and VSD repair. In Rhydwen *et al*. (2002) study, 32% underwent only VSD repair and 68% underwent concomitant CABG and VSD repair. These values in Barker *et al*. (2003) study were 36 and 64%. Our findings did not show significant relation between mortality and concomitant CABG.

In the prethrombolytic era, outcomes after septal rupture were extremely poor, with an in-hospital mortality rate of approximately 45% among surgically treated patients and 90% among those treated medically (Yochai *et al*., 2002; Barker *et al*., 2003). Mortality with this complication remains extremely high in the thrombolytic era, despite improvements in medical therapy and percutaneous and surgical techniques. The mortality rate was 74% in Crenshaw *et al*. (2000) study. In Rhydwen *et al*. (2002) study, within the surgical group survival with prior thrombolytic therapy was 25 and 69% without. They concluded that there is an earlier presentation of postinfarction VSD when thrombolytic therapy has been used. An early presentation can carry a worse prognosis (Rhydwen *et al*., 2002). Our finding is consistent with previous observations and the overall mortality rate was very higher in patients received thrombolytic therapy.

The cumulative survival rate (including perioperative deaths) was 78% at 1 year, 65% at 5 years and 40% at 10 years (Yochai *et al*., 2002). Ninety percent of ruptures occur within the 1st 10 days after the infarction. Twenty four percent die within the 1st 24 h, 60% within the 1st week and 85% within the 1st 2 months. Only 7% survive the 1st year (Calderon and Ott, 1991). In the present study, we found that the mortality rate caused by cardiac rupture was 58.5%. Our finding is consistent with

previous observations (Crenshaw *et al*., 2000; Yip *et al*., 2003; Yochai *et al*., 2002; Soriano *et al*., 2005). The overall mortality rate was very high (83.3%) in patients with cardiac rupture (Yip *et al*., 2003). Outcome remains extremely poor, with a mortality rate of approximately 50% in patients undergoing surgical repair and nearly 95% in those treated medically. More effective ways to predict, prevent and treat this devastating complication are needed (Crenshaw *et al*., 2000; Soriano *et al*., 2005).

In our study, mortality in operated group was less than patients treated medically (30.8 vs. 71.4%). In the GUSTO-I trial, the 34 patients who underwent surgical repair had a lower 30-day mortality rate than the 35 patients who were treated medically (47 vs. 94%) as well as a lower 1-year mortality rate (53 vs. 97%) (Crenshaw *et al*., 2000; Yochai *et al*., 2002). In Calderon and Ott (1991) study the overall operative mortality was 57% and the hospital survival rate was 43%. Deja *et al*. (2000) reported the 30 days mortality of 37%. In Teo *et al*. (1990) study, the operative survival was 42.9%.

Prompt diagnosis followed by surgical repair is essential for patients with VSR following MI (Sasseen *et al*., 2003). Early surgical repair of the VSD is important if overall prognosis of these patients is to be improved (Teo *et al*., 1990). Delayed hospital admission or undue in-hospital physical activity increases the risk of septal rupture in patients with AMI (Figueras *et al*., 1998).

There are several limitations to this study. First, the number of patients with cardiac rupture in this study was small, therefore, our results should be viewed as preliminary and need to await confirmation by larger clinical trials. Second, cardiac rupture, pseudoaneurysm, or incomplete rupture could easily be missed since premortem echocardiograms were often unavailable and cultural factors prevent postmortem examination. Therefore, the incidences of these complications could have been underestimated in our study.

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