Therapeutic Management Changes and Mortality Rates over 30 Years in Ventricular Septal Rupture Complicating Acute Myocardial Infarction

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Recent studies have shown that the decrease in ventricular septal rupture (VSR) incidence after acute myocardial infarction is related to the improvement of reperfusion strategies. Our main objective was to explore the influence of therapeutic management changes on post-infarct VSR patient outcomes in a single reference center over a period of 30 years. We analyzed therapeutic management strategies and mortality rates in 228 patients with VSR after acute myocardial infarction admitted from 1981 to 2010. Patients were classified in 3 successive decades. There were no significant differences in clinical characteristics of patients with VSR at admission among those decades. Overall, surgery was performed in 159 patients (71.9%), primary transcatheter VSR closure was attempted in 5 patients (2.2%), and 64 patients (27.6%) were managed medically. Independent predictors of in-hospital mortality were VSR surgical repair (odds ratio [OR] 0.22, 95% confidence interval [CI] 0.1 to 0.7, p = 0.008), cardiogenic shock (OR 6.06, 95% CI 2.8 to 13.1, p <0.0001), and Killip class on admission (OR 1.75, 95% CI 1.1 to 9.9, p = 0.02). We found a significant 1-year mortality reduction between the first and second decades (hazard ratio 0.48, 95% CI 0.28 to 0.80; p = 0.005), with no significant change in the last decade (p = 0.2). This change was related to a systematic referral to surgical repair and shorter delays to VSR surgery (5.2 ± 6.3 vs 1.9 ± 3.2 days from first to second decade; p = 0.012). In conclusion, surgical repair remains the only significant efficient therapy to reduce mortality in patients with VSR ($p < 10^{-3}$). In-hospital prognosis remains disappointing. This contrasts with the favorable long-term outcome of patients who survive the perioperative period and are discharged from hospital. **(Am J Cardiol 2013;112:1273−1278)**

Ventricular septal rupture (VSR) is a severe and rare complication of acute myocardial infarction (AMI). Reperfusion therapies have significantly reduced the incidence of VSR. However, mortality rates have remained unchanged.[1] In patients without surgical treatment, this disease is associated with a 90% in-hospital mortality.[2] Current guidelines recommend that patients with post-AMI VSR should be considered for urgent surgical repair, unless further support is futile.[3,4] The timing of surgery and perioperative therapeutic management of patients with VSR remain controversial.[5−9] Therapeutic management strategies of patients with post-AMI VSR and effects of changes in these strategies in the previous 3 decades remain poorly explored.[1,10] This report assesses therapeutic management strategies as well as trends and predictors in mortality rates of patients with post-AMI VSR over 3 decades in a single tertiary referral center.

Methods

Data for this study were obtained from our institutional database, containing discharge records of all patients admitted with a diagnosis of AMI. This retrospective study was approved by our Institutional Review Board. The study cohort was constituted by patients admitted to our institution, a tertiary referral university hospital, with a diagnosis of acute ST-segment elevation myocardial infarction (STEMI) complicated with VSR from 1981 to 2010. Patients with VSR complicating STEMI during this period were identified by the diagnosis code for acquired VSR (International Classification of Disease code 429.71; n = 171) or a procedure code indicating VSR surgical repair within 30 days after initial AMI admission (International Classification of Disease codes 35.53, 35.62, or 35.72; n = 57).

Patients surviving the initial hospital period were contacted by telephone in 2010 to inquire about cardiovascular

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Table 1
Clinical and infarction characteristics in patients with ventricular septal rupture (VSR) according to the study decade

Variable	Time Period			All Patients (n = 228)
	1981−1990 (n = 92)	1991−2000 (n = 83)	2001−2010 (n = 53)	
Age (yrs)	70 ± 8	72 ± 9	78 ± 8	71 ± 8
Women	40 (43.5)	41 (49.4)	35 (66.0)	116 (50.9)
Diabetes mellitus	15 (23.4)	19 (22.9)	8 (15.7)	42 (21.2)
Smoker	28 (30.4)	18 (21.7)	17 (32.1)	63 (27.6)
Hypertension	40 (43.5)	33 (39.8)	25 (47.2)	98 (43.0)
Preinfarct angina pectoris	18 (19.6)	16 (19.3)	3 (5.7)	37 (16.2)
Location of infarct				
Anterior wall	45 (48.9)	43 (52.4)	36 (67.9)	124 (54.6)
Inferior/lateral wall	47 (51.1)	39 (47.6)	17 (32.1)	104 (45.4)
Right ventricular dysfunction	39 (50.0)	24 (35.8)	20 (38.5)	83 (42.1)
Killip class at admission				
I	23 (41.8)	41 (55.4)	31 (60.8)	95 (52.8)
II	8 (14.5)	23 (27.7)	11 (31.1)	42 (23.3)
III	13 (23.6)	3 (4)	3 (5.9)	19 (10.6)
IV	11 (20.0)	7 (9.9)	6 (11.8)	24 (13.3)
Cardiogenic shock during hospitalization	60 (65.2)	42 (50.6)	33 (62.3)	135 (59.2)
Time interval between MI and VSR (days)	3.8 ± 4.4	5.9 ± 10.1	3.7 ± 5.0	4.5 ± 7.1
GRACE score	NA	232.8 ± 34.1	227.4 ± 36.5	228.7 ± 35.7

All values expressed as absolute number and percentage or mean ± SD.
GRACE = Global Registry of Acute Coronary Events; MI = myocardial infarction; NA = not applicable.

diagnoses and death. Next-of-kin interviews for out-of-hospital cardiovascular deaths were obtained. To verify self-reported diagnoses, copies of all death certificates and medical records were requested for all outpatient cardiovascular diagnoses. We were successful in getting medical records in 99% of patients. Follow-up telephonic interviews were completed in 95% of living patients.

Demographic, medical history, clinical and hemodynamic data at admission and at the time of surgery, echocardiographic data, and therapeutic management procedures were recorded as covariates. Cardiogenic shock was defined as a systolic blood pressure of <90 mm Hg for 30 minutes, not responsive to fluid administration alone, and associated with clinical signs of hypoperfusion (urine output of <30 ml/h and cold extremities).

During the whole study period, a senior cardiac surgeon, a senior anesthesiologist, and a senior cardiologist in the intensive cardiac care unit were on call. Based on our internal institution protocol, each VSR case was collegially discussed for global therapeutic management and the indication and timing of VSR surgical repair. Depending on the period, various protocols within our institution were applied. From 1981 to 1990, if the patient presented with a stable hemodynamic status, VSR surgical repair was not considered in emergency and was performed from 1 to 3 weeks after index AMI hospitalization. Patients were therefore initially managed with medical treatment with or without intra-aortic balloon pump (IABP) support and close monitoring within our intensive care unit. Urgent VSR surgical repair was performed only in case of significant alterations of the hemodynamic clinical status. A systematic change in therapeutic management strategy occurred after 1991 when an institutional consensus decision was made to attempt VSR surgical repair for all patients as early as possible after diagnosis or hospital admission. At the same time, systematic support with IABP for all patients with VSR was applied while waiting for surgery.

Various surgical repair techniques were used, depending on the location and pathologic characteristics of VSR and at the surgeon's discretion. All open-chest surgeries were performed with cardiopulmonary bypass, crystalloid cardioplegia, and moderate systemic hypothermia. Concomitant coronary artery bypass was performed at the surgeon's discretion.

From 2001 to 2010, with the advent of systematic primary percutaneous coronary intervention for patients with STEMI referred to our institution, the rates of efficient reperfusion significantly improved. Therapeutic management strategy of patients with VSR remained the same as the previous decade. However, from 2006 to 2010, a percutaneous interventional strategy was attempted. This strategy was based on immediate primary transcatheter closure of VSR in a selected group of patients presenting with acute anteroseptal myocardial infarction. Transcatheter closure was attempted according to VSR location and its anatomical characteristics. Also, in this period, extracorporeal membrane oxygenation was proposed and applied in case of severe right ventricular dysfunction after VSR surgical repair.

We compared demographic, clinical and cardiac procedures data over the 3 successive decades of our study period. Chi-square tests were performed for categorical variables and Student t test or Wilcoxon rank sum test for continuous variables as appropriate to compare clinical and demographic characteristics.

Overall in-hospital and 1-year mortalities were the 2 studied outcomes. Overall in-hospital mortality was defined as mortality occurring at the hospital or within the month after admission, and 1-year mortality included all deaths from any cause that occurred within the first year of follow-up after admission.

For both outcomes, Kaplan-Meier survival curves were estimated per decade. The 3 survival curves were compared using log-rank test. We used univariate logistic regression

Table 2
Therapeutic management and cardiac procedures

Cardiac Procedure	Time Period			All Patients
	1981−1990 (n = 92)	1991−2000 (n = 83)	2001−2010 (n = 53)	
Diagnostic catheterization	18 (19.6)	56 (68.3)	37 (69.8)	111 (48.9)
Coronary bypass	1 (1.8)	14 (19.7)	9 (20.5)	24 (14.0)
IABP	11 (12.0)	50 (60.2)	41 (77.4)	102 (44.7)
Inotropic agents	75 (81.5)	53 (63.9)	33 (62.3)	161 (70.6)
VSR repair surgery	58 (63.0)	69 (83.1)	37 (69.8)	164 (71.9)
VSR diagnostic to VSR surgery time delay (days)	5.2 ± 6.3	1.9 ± 3.2	2.2 ± 4.0	3.2 ± 4.9
Percutaneous transcatheter VSR closure	—	—	5 (9.4)	5 (2.1)

All values expressed as absolute number and percentage or mean ± SD.
MI = myocardial infarction; NA = not applicable.

Table 3
Risk factors for in-hospital mortality by unadjusted (systematic adjustment for period and surgical treatment) and adjusted logistic regression

Risk Factors in Logistic Models	Unadjusted Regression			Adjusted Regression		
	OR	95% Confidence Interval	p	OR	95% Confidence Interval	p
Admission decade						
1981−1990	Reference			Reference		
1991−2000	0.56	0.28−1.12	0.10	0.53	0.25−1.16	0.114
2001−2010	0.50	0.22−1.10	0.09	0.37	0.15−0.89	0.027
Surgical repair	0.12	0.05−0.33	<0.0001	0.22	0.08−0.62	0.004
Age (yrs)	1.04	1.00−1.08	0.04	1.06	1.02−1.10	0.008
Time interval between MI and VSR (days)	0.96	0.91−1.00	0.07			
Gender (female)	1.12	0.61−2.06	0.714			
Cardiogenic shock	5.95	3.08−11.47	<0.0001	6.95	3.48−13.88	<0.0001
Oliguria at admission	8.94	1.71−46.86	0.01			
Diabetes	2.99	1.17−7.65	0.02			
Infarct location						
Anterior wall	Reference					
Inferior/lateral wall	1.46	0.79−2.70	0.23			
Right ventricle dysfunction	0.99	0.52−1.92	0.99			
Diagnostic catheterization	1.16	0.58−2.33	0.68			
Killip class at admission						
I	Reference					
II	1.09	0.52−2.28	0.82			
III	3.95	1.08−14.48	0.04			
IV	—	—	—			
CABG	0.99	0.39−2.49	0.98			
IABP	2.26	1.05−4.84	0.04			

Oliguria was defined by a urine output of <180 ml in the first 6 hours after admission.
CABG = coronary artery bypass graft; MI = myocardial infarction; OR = odd ratio.

models to analyze potential determinants of in-hospital mortality with systematic adjustment for time decade and surgical intervention. Variables that were tested as in-hospital mortality determinants were time decade, surgical intervention, age, gender, diabetes, Killip class, infarct location, coronary artery bypass graft surgery, IABP implantation, right ventricle dilation, coronary angiography, time delay between infarction and VSR diagnosis, cardiogenic shock, and urine output in the first 24 hours after VSR diagnosis. All these variables were then included in a full multivariate model. To study overall 1-year survival, nonadjusted and adjusted Cox proportional hazard models were built including relevant demographic and clinical variables aforementioned. A 2-tailed p value <0.05 defined statistical significance. Analyses were performed using R, version 2.15-2 (The R Foundation for Statistical Computing, Vienna, Austria).

Results

Two hundred twenty-eight consecutive patients with post-AMI VSR were hospitalized at our institution from 1981 to 2010. Sixty-nine patients (30%) were transferred from other centers. As listed in Table 1, 92 patients were hospitalized in 1981 to 1990, 83 in 1991 to 2000, and 53 in 2001 to 2010. Mean age was 71 ± 8 years, with 51% women. Infarct location was anterior in 55%, and mean time interval between onset of infarction and VSR diagnosis was 4.5 ± 7 days. Cardiogenic shock was present at presentation

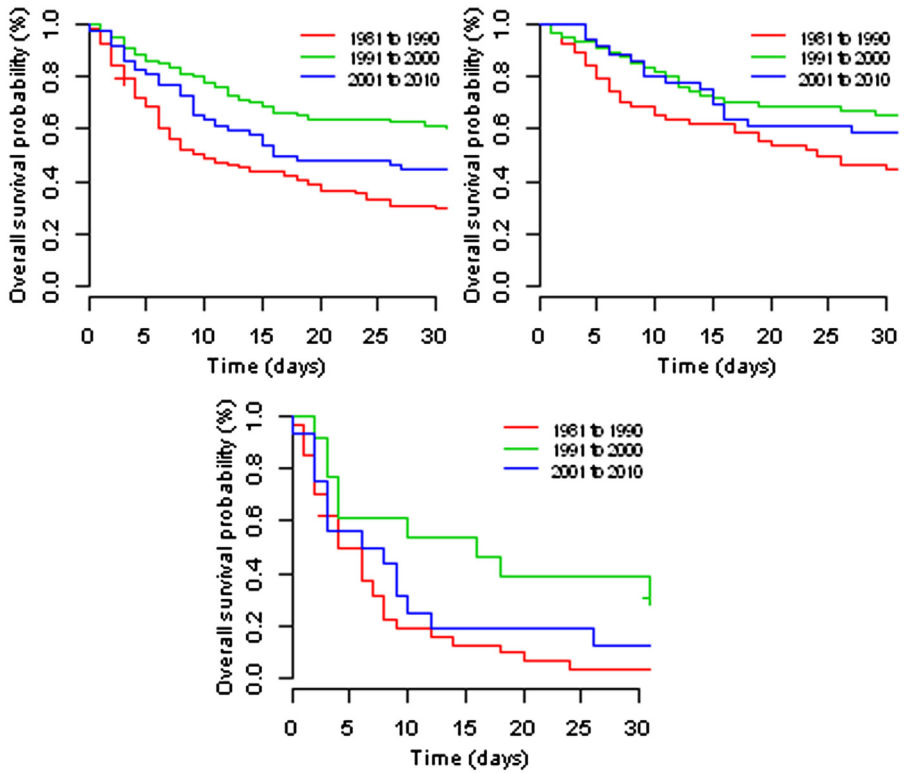

Figure 1. Kaplan-Meier 30-day overall survival curve for all patients *(top-left panel)*, operated patients *(top-right panel)*, and nonoperated patients *(bottom panel)*.

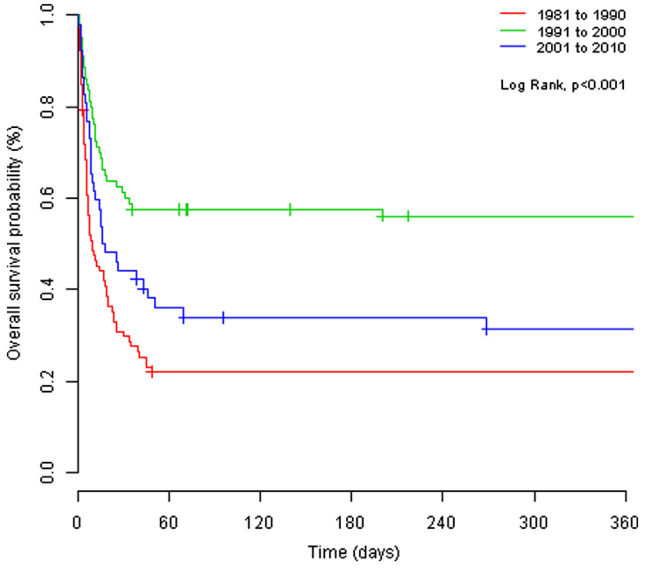

Figure 2. Kaplan-Meier 1-year overall survival curves.

in 24 patients (13.3%). Cardiac catheterization was performed in 111 patients (49%). Cardiac procedures by study decade are listed in Table 2. Overall, 159 patients (71.9%) were treated surgically. The proportion of patients who received VSR repair significantly increased from 63% in 1981 to 1990 to 83% in 1991 to 2000 ($p = 0.003$). It decreased to 70% in 2001 to 2010.

Surgical approach to repair VSR was through the left atrium in 156 patients and right atrium in 3 patients. In addition to VSR repair, 24 patients (14%) received combined coronary artery bypass graft surgery. One patient was treated with a heart transplant.

Mean time from VSR diagnosis to VSR repair was 3.2 ± 4.9 days. Mean delays between AMI and surgery significantly decreased from 9.2 ± 8.3 days in the first decade to

Table 4
Hazard ratios from adjusted Cox proportional model including the period, the surgical treatment, shock, age, intra-aortic balloon pump (IABP) for all patients; and time between VSR and surgery for operated patients

Variable	HR	95% CI	p
All patients			
Admission decade			
1981–1990	Reference		
1991–2000	0.47	0.28–0.80	0.005
2001–2010	0.68	0.38–1.22	0.199
Surgical repair	0.31	0.21–0.46	<0.0001
Cardiogenic shock	2.40	1.56–3.70	<0.0001
Age	0.10	0.97–1.02	0.761
IABP	1.14	0.70–1.86	0.584
Operated patients			
Admission decade			
1981–1990	Reference		
1991–2000	0.29	0.13–0.66	0.003
2001–2010	0.29	0.12–0.73	0.009
VSR repair surgery time delay	0.82	0.74–0.92	<0.0001
Cardiogenic shock	2.09	1.25–3.50	0.005
Age	1.02	0.99–1.05	0.111
IABP	1.29	0.60–2.80	0.512

CI = confidence interval; HR = hazard ratio.

6.2 ± 7.6 in the last decade ($p = 0.001$). This was because of a sharp decrease in the VSR surgery delay from 5.2 ± 6.3 days in the first decade to 2.2 ± 4.0 days in the second decade ($p < 0.0001$).

From 2001 to 2010, 5 patients (9.4%) were selected for percutaneous transcatheter VSR closure. Device implantation was successful in 3 patients. Left-to-right ventricle shunt persisted in all cases. All 3 patients underwent subsequent surgical VSR repair and died during the postoperative period. In the other 2 patients, device implantation failed.

Overall, 64 patients (27.9%) were treated medically. All but 5 died within 2 months after hospitalization. In the first decade, 13 patients (14%) died while waiting for surgery and 21 (22%) were denied surgical treatment. In the second decade, 2 patients (2.4%) died while waiting for surgery and 11 (13%) were denied surgical treatment. During the last decade, no patient died while waiting for surgery and 13 patients (24%) were denied surgical treatment.

In-hospital survival rate increased significantly from 23% in the first decade to 40% in the second and 38% in the last decade. The test for trend was significant over the 3 periods ($p = 0.03$). Patients with cardiogenic shock had significantly greater in-hospital mortality than patients without (84% vs 43%, respectively, $p < 0.05$). There was a nonsignificant trend toward a better in-hospital survival after surgery from 34% in 1981 to 1990 to 45% in 1991 to 2000 and 49% in 2001 to 2010 ($p = 0.31$). Determinants of in-hospital mortality analyses are listed in Table 3. The only independent predictors of in-hospital mortality were VSR surgical repair, presence or evolution toward cardiogenic shock, and Killip class on admission (Table 3).

Kaplan-Meier in-hospital survival curves by decade are reported in Figure 1. There was a significant increase of 1-year survival in the last 2 decade groups as illustrated in Figure 2. Table 4 presents adjusted hazard ratios for all patients and operated patients. One-year mortality decreased significantly with admission decade, especially for operated patients. Considering all patients, surgical repair significantly divided mortality hazard by 3, whereas cardiogenic shock multiplied it by nearly 2.4. For operated patients, cardiogenic shock remained one significant factor of poor prognosis after adjustment ($p = 0.005$).

Discussion

Our single-center observational study of all patients with post-AMI VSR complicating STEMI over a period of 30 years shows a significant reduction in mortality related to the systematic referral for surgical VSR repair between the first and second decades. Despite several changes in therapeutic management strategies in the last decade, mortality was unchanged thereafter. The presence of cardiogenic shock at admission or in the following days was an independent predictor of mortality.

Our VSR patient cohort is one of the largest case series reported in a single referral center setting. All management decisions were taken according to the clinical status of patients with VSR during the first hours after admission or VSR diagnosis.

The decision to operate all patients with VSR as soon as possible was taken in 1990 after a consensus agreement between all surgeons, anesthetists, and cardiologists of our institution. Our results show that this strategy significantly improved the overall mortality rate, although postoperative outcome was not significantly changed. Those results are different from previous reports in which operative mortality varied from 10% to 53%. The main explanation for this discrepancy might be related to the differences in study populations.[8]

The delay between infarction and surgery is a major determinant of survival.[2,5,6,11] Reports on postoperative mortality after delayed surgical repair inevitably select patients with a better prognosis because of the high mortality rates in the preoperative period. In many studies, the delay between myocardial infarction and surgery was >1 week, suggesting that many patients with VSR were excluded from these studies. Our results with a significant reduction of overall mortality between first and second decades show that longer delays between onset of MI and surgery resulted in higher rates of preoperative mortality. A systematic early surgical intervention strategy during the following decades resulted in a significant reduction of overall mortality.

The long-term outcome of these patients is uncomplicated, and this is in accordance with previous reports.[6,11–13] Most of these patients also had an excellent functional status when alive at follow-up.[5,6,8,11,12,14–16]

Previous reports in other multicenter or single-center series[1,10,17] have shown a significant decrease in the rates of VSR since the introduction of reperfusion strategies for patients with AMI. The significant decrease in the incidence of VSR between the first and last decades that we found confirms the previous report by Figueras et al[17] in which the incidence of heart rupture (including VSR) significantly decreased from 6.4% in the 1977 to 1982 period to 4.8% in the 2001 to 2006 period. Conversely, in their study from the Myocardial Infarction Data Acquisition System database, Moreyra et al show a significant decrease from 1990 to 2007. This might be explained by differences in diagnostic methods and the multicenter setting. As reported by López-Sendón et al,[10] there are significant differences in the mechanical complications rates after AMI between different institutions.

In the same way, the rates of in-hospital death in our study population were high but remain in the range of previous reports in which in-hospital mortality rates of VSR complicating STEMI vary from 40% to 75%.[18,19]

Medical management in the first decade may have been suboptimal. Only 44% of these patients had an IABP inserted. However, if IABP is now currently used for short-term hemodynamic support,[2] it does not have any significant effect on long-term outcome.[12] Compared with the timing of surgery, initial medical management alone has probably a small impact on the final outcome.

Although percutaneous interventional VSR closure has been presented as a promising alternative to surgery with high procedural success in small case series,[20,21] our results with this strategy during the last decade were disappointing. This might be in part related to our inexperience. However, even in the optimal conditions, this technique has high rates of procedure-related complications.

As an observational retrospective study, our study is subject to certain inherent limitations and potential biases, including collection of nonrandomized data, missing or incomplete

information, and potential confounding by unmeasured covariates that must be considered when interpreting our results. Nevertheless, all our study patients have been managed, at least in the first hours after admission in our intensive care unit, before they were eventually transferred to the operating room and subsequently to the postsurgical critical care unit of our institution. In this environment, patients benefit from the most recent diagnostic and therapeutic improvements, and data are collected in a timely fashion.

Because of the change in technologies and medical management over the 3 decades, precise information on certain clinical and paraclinical variables such as cardiac output or mitral regurgitation status was incomplete. Diagnostic catheterization has not become standard of care in patients with AMI at our institution before beginning/mid-90s. This explains why we only have coronary angiography data in 49% of our patients. Patients presenting in cardiogenic shock were significantly less likely than patients without cardiogenic shock to get to surgery. This might explain in part why cardiogenic shock was an independent predictive marker of in-hospital mortality.

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Disclosures

The authors have no conflicts of interest to disclose.

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