Timing of Repair in Postinfarction Ventricular Septal Defect

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The optimal timing of postinfarction ventricular septal defect (PI-VSD) repair is subject to debate. Patients with ventricular septal defect (VSD) and ST-elevation myocardial infarction (STEMI) were queried using appropriate *International Classification of Diseases, Ninth and Tenth Revision Clinical Modification* codes from the National Inpatient Sample (2003 to 2018). VSD repair was identified using appropriate *International Classification of Diseases, Ninth and Tenth Revision Procedure Coding System* codes. Data were stepwise stratified by cardiogenic shock (CS) and time of repair from admission to create 6 clinically relevant groups: shock 1 (CS; 0 to 7 days), shock 2 (CS; 8 to 14 days), and shock 3 (CS; >14 days). Nonshock groups were classified similarly. The primary outcome was in-hospital mortality. Multilevel hierarchical logistic regression was used to adjust for confounders for each group. We identified 10,902 patients with PI-VSD. In shock 1 (n = 5,794), VSD repair was associated with lower mortality (OR 0.76; 95% CI 0.68 to 0.86, p <0.001) compared to no VSD repair. In shock 2 (n=1,009) mortality was numerically lower in those who received VSD repair, but not statistically different. In shock 3 (n=483), mortality was numerically higher in those who received VSD repair, but not statistically different. In nonshock 1 (n=5,108), VSD repair was associated with higher mortality (odds ratio [OR] 1.59; 95% confidence interval [CI] 1.33 to 1.90; p <0.001). In nonshock 2 (n = 1,265), mortality was numerically higher in patients with VSD repair, although not statistically different. In nonshock 3 (n = 472), mortality was numerically lower in patients with VSD repair, although not statistically different. Mechanical circulatory support use increased over the 16 years (relative change + 18%, p <0.001), with no significant change in mortality among patients with PI-VSD. In conclusion, in patients with CS, early PI-VSD repair was associated with lower mortality. However, in patients without CS, early PI-VSD repair was associated with higher mortality. **(Am J Cardiol 2022;175:44−51)**

Introduction

Postinfarction ventricular septal defect (PI-VSD) is a rare but devastating complication of ST-elevation myocardial infarction (STEMI).[1−3] Surgical management is the definitive treatment, but is associated with high mortality.[4] The optimal timing of PI-VSD repair is heavily debated.[1,5] Most recently, a 2012 study of timing of PI-VSD repair utilizing the Society of Thoracic Surgeons database noted lower mortality associated with patients who received intervention at later time periods compared to earlier time periods.[6] However, it was unclear whether the difference was due to an effect from the intervention or whether it was from the selection of patients with better substrate and hemodynamic profile (immortal time bias). To address these limitations, we created 6 clinically relevant groups from patients with PI-VSD using a 2-step stratification approach on the basis of the presence of cardiogenic shock (CS) and timing from admission. We compared patients undergoing ventricular septal defect (VSD) repair versus no VSD repair in each of these 6 groups to minimize selection and survival bias.

Methods

Data were extracted from the Nationwide Inpatient Sample (NIS) from the years 2003 to 2018. NIS contains data from a 20% stratified sample from discharges in hospitals across the United States and has been utilized across multiple studies.[7,8] This study included patients who presented with STEMI and VSD as any primary or secondary diagnosis using appropriate and standard definitions of *International Classification of Disease, Ninth and Tenth Revision, Clinical Modification* (ICD-9 CM and ICD-10 CM) codes. CS was defined by appropriate and standard ICD-9/10 CM codes in any primary or secondary diagnosis. Patients with missing information on age, gender, mortality, or were younger than 18 years were removed. Patients with missing information on timing of repair were also excluded (Figure 1, Supplementary Table 1). Institutional review

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See page 51 for disclosure information.

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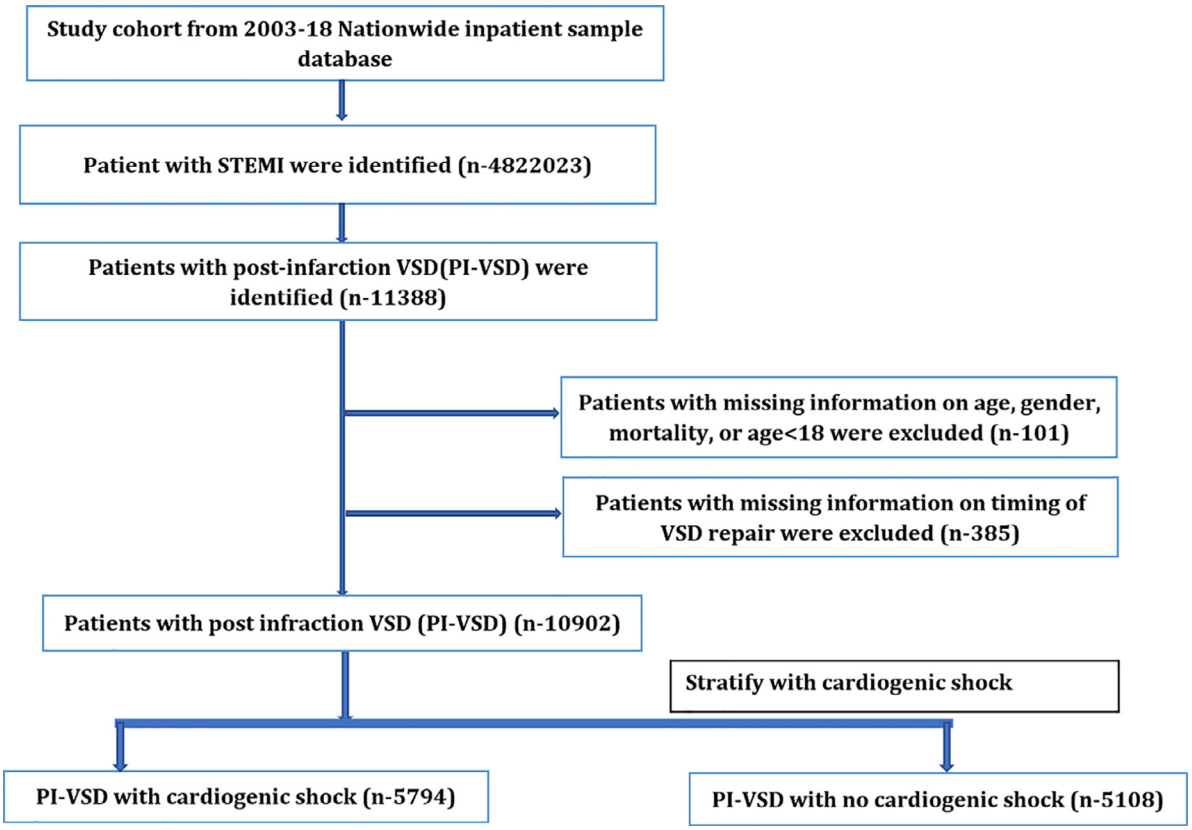

Figure 1. Patient Selection and Study Design. This figure illustrates patient selection and study design

board approval was not required due to use of deidentified data.

This study used variables provided in NIS by the Healthcare Cost and Utilization Project (HCUP) to identify baseline characteristics, including age and gender; hospital characteristics, such as bed size and teaching status; and other patient-specific aspects, including median household income category for patient's ZIP Code, primary payer, admission type, and admission day of the week.[9,10] Mechanical circulatory support (MCS) was defined by the use of intra-aortic balloon pump, percutaneous ventricular assist device (PVAD), or extracorporeal membrane oxygenation (ECMO). IABP, PVAD, and ECMO were identified using ICD-9/10 Procedure Coding System codes in either primary or secondary procedural fields (Supplementary Table 2).[9,10] We used the ICD-9/10-CM codes provided by the Elixhauser co-morbidity index calculator given by the HCUP to identify obesity, hypertension, diabetes, chronic obstructive pulmonary disease, peripheral vascular disease, and anemia (Supplementary Table 2).[11,12] Other co-morbidities, such as previous coronary artery disease (CAD), family history of CAD, heart failure, chronic kidney disease stage 3 or more, previous coronary artery bypass grafting, previous percutaneous coronary intervention, hyperlipidemia, previous stroke/transient ischemic attack, and tobacco use were identified using appropriate ICD-9/10-CM codes (Supplementary Table 2).

Timing of procedure in days was identified from the day of admission using the PRDAYn variable.[9] VSD repair was identified using appropriate ICD-9/10 Procedure Coding System codes in either primary or secondary diagnosis fields (Supplementary Table 1). The primary outcome of the study was in-hospital all-cause mortality, which was provided in the NIS database.

In *shock 1,* the inclusion criteria were: (1) clinical presentation of CS and (2) alive on Day 0. The intervention of interests was comparing VSD repair during 0 to 7 days versus no VSD repair (Figure 2). If VSD repair took place any time after Day 7 of admission, it did not count as an intervention for this group. In *shock 2*, the inclusion criteria were: (1) clinical presentation of CS, (2) alive on Day 8, (3) not discharged before Day 8, and (4) did not receive VSD repair before Day 8 (Figure 2). The intervention of interest was comparing VSD repair during 8 to 14 days to no VSD repair in this timeframe. If VSD repair took place after 14 days, it was not counted as an intervention in this group. In *shock 3*, the inclusion criteria were: (1) clinical presentation of CS, (2) alive on Day 15, (3) not discharged before Day 15, and (4) did not receive VSD repair before Day 15 (Figure 2). The intervention of interest was VSD repair after Day 14 compared with those who had not undergone intervention until that time point. Nonshock groups 1, 2, and 3 were assigned in the same way as shock groups 1, 2, and 3, except their clinical presentation did not include CS (Figure 2, Supplementary Table 3).[9,10]

SAS 9.4 (SAS Institute Inc, Cary, North Carolina) was used for statistical analysis. Categoric variables were compared using the chi-square test. Multilevel hierarchical logistic regression models with hospital ID as a random effect were used to adjust for demographic, co-morbidities,

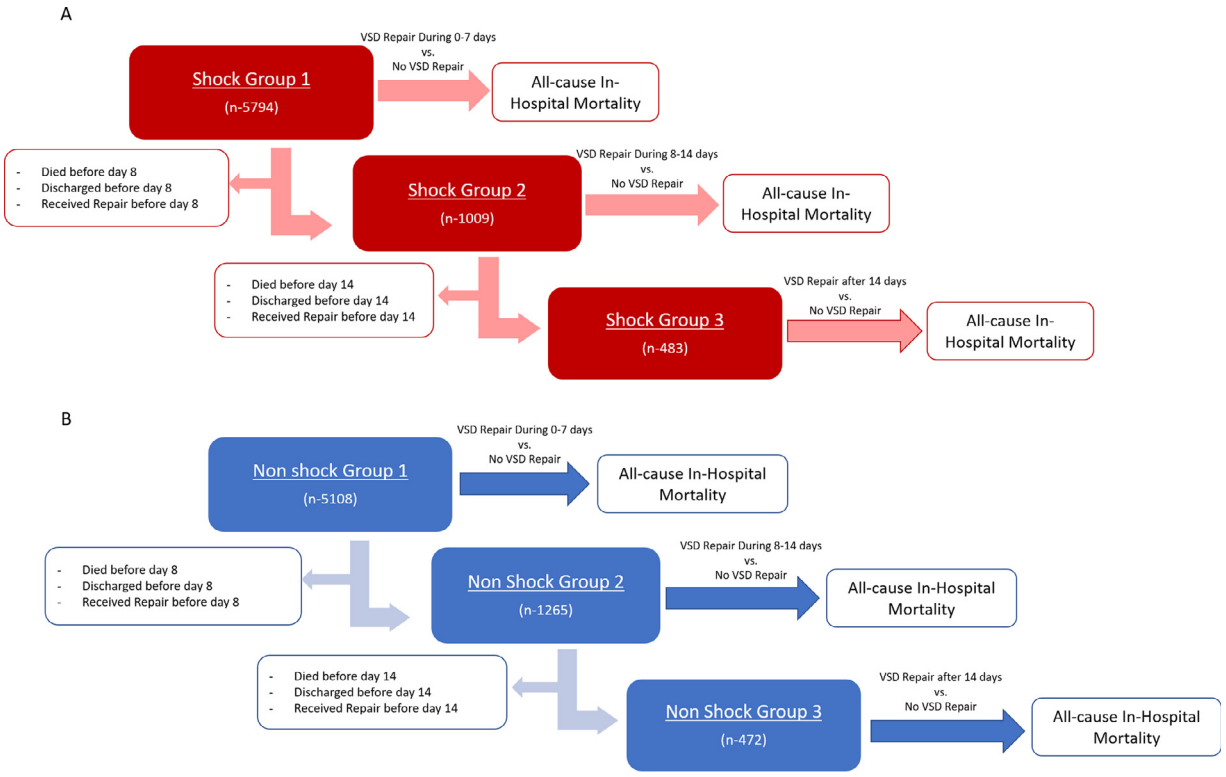

Figure 2. Shock and Nonshock Group Inclusion Criteria. This figure illustrates the inclusion criteria in (a) shock and (b) non-shock groups.

and socioeconomic factors. We created separate multivariate models for all 6 groups mentioned previously. Exploratory analysis was also conducted to assess the trends of MCS utilization, rate of VSD repair, and mortality in patients with PI-VSD over the years 2003 to 2018. Sensitivity analysis was performed using 1 : 1 propensity score matching of VSD repair and no VSD repair groups using the demographic, co-morbid, and socioeconomic factors described previously. P value for trends of categoric variables was assessed using Cochrane Armitage test. A 2-tailed p value of 0.05 was designated as statistically significant. We adhered to the method standards of the HCUP.[13]

Results

Our study identified a total of 10,902 patients with STEMI and VSD. Of these patients, 3,798 patients underwent PI-VSD repair (35%). Of the overall cohort, 5,794 patients (53%) presented with CS. Male gender and patients aged 65 to 79 were the most frequent demographic groups represented in both shock and nonshock groups (Table 1). Previous CAD, heart failure, hypertension, hyperlipidemia, and anemia were the most common co-morbidities across patients in both shock and nonshock groups. The majority of patients presented to large hospitals and academic centers (Table 1).

In *shock 1*, (n = 5,794), VSD repair was associated with lower mortality (50.8% vs 60.6%; OR: 0.76; 95% CI: 0.68 to 0.86; p <0.001) than no VSD repair. In *shock 2* (n = 1,009), there was lower numerical mortality with VSD repair (38.4% vs 44.7%; OR: 0.86; 95 CI: 0.57 to 1.27, p = 0.440) than no VSD repair that did not reach statistical significance. In *shock 3* (n = 483), there was higher numerical mortality with VSD repair (40.4% vs 33.8%, OR: 0.90; 0.48 to 1.69, p = 0.753) than VSD repair but did not reach statistical significance. (Figure 3, Table 2).

In nonshock 1 group (n = 5,108), VSD repair was associated with higher mortality (36% vs 23.8%; OR: 1.59; 95% CI: 1.33 to 1.90, p <0.001) than no VSD repair. In nonshock 2 group (n=1,265), there was numerically higher mortality with VSD repair (22.5% vs 19.6%; OR: 0.77; 95% CI: 0.44 to 1.34, p = 0.351) than no VSD repair, but this difference did not reach statistical significance. In nonshock 3, there was numerically lower mortality with VSD repair (24.6% v 27%; OR: 2.19; 95% CI: 0.76 to 6.36, p = 0.299) than no VSD repair, but this difference did not reach statistical significance (Figure 3, Table 2).

Exploratory analysis demonstrated an increase in the use of MCS overall (relative change = +18%, p <0.001) (Figure 4, Supplementary Table 4). Further analysis of subtype of MCS use demonstrated a decrease in the use of IABP (relative change: −12.8%; p <0.001) and increase in use of PVAD (relative change = +830%, p <0.001) and ECMO (relative change = +990%, p <0.001 devices) (Figure 4, Supplementary Table 4). There was a modest decrease in the rate of VSD repair over time (relative change: −6%, p = 0.007) (Figure 4, Supplementary Table 4). There was no significant change in mortality over the years analyzed (mean percent: 42%, relative change: −2%, p = 0.54) (Figure 4, Supplementary Table 4). Sensitivity analysis using propensity score matching demonstrated similar results to those described previously (Supplementary Table 5).

Table 1
Baseline characteristics

	Shock				Non shock			
	No VSD repair	VSD repair	Overall	p-value*	No VSD repair	VSD repair	Overall	p-value†
Variable	3,279	2,515	5,794		3,825	1,283	5,108	
Age (Years)				<0.001				<0.001
18-49	3.3	3.3	3.3		11.6	3.4	9.6	
50-64	21.7	33.5	26.8		24.3	32.5	26.4	
65-79	41.4	49.9	45.1		33.2	50.6	37.6	
>=80	33.6	13.3	24.8		30.9	13.5	26.5	
Gender				<0.001				0.493
Male	49.4	60.5	54.2		53.7	54.8	54	
Female	50.6	39.5	45.8		46.3	45.2	46	
MCS	62.9	75.8	68.5	<0.001	17.7	52.4	26.4	<0.001
IABP	56	70.2	62.2	<0.001	16.2	51.2	25	<0.001
PVAD	8	8.7	8.3	0.354	1.04	0.8	1	0.421
ECMO	4.9	9.2	6.8	<0.001	0.9	2.4	1.3	<0.001
Comorbidities‡								
Obesity	7.5	13.8	11.9	<0.001	7.6	11.3	8.5	<0.001
Hypertension	53.7	50.6	52.4	0.018	56.7	52.3	55.6	0.006
Diabetes Mellitus	27.5	31	29	0.004	29.9	26.4	29	0.017
Hyperlipidemia	33	34.6	33.7	0.201	40.9	31.5	38.6	<0.001
Tobacco use	28.5	26.5	27.6	0.088	29.1	25.2	28.1	0.006
History of TIA or Stroke	5.6	2.7	4.3	<0.001	6.2	1.9	5.1	<0.001
COPD	13.2	13.4	13.3	0.809	15.1	14.7	15	0.766
Heart failure	48.2	60.5	53.6	<0.001	15.8	58.4	49	<0.001
CKD stage 3 or more	18.6	28.1	22.7	<0.001	17	16.4	16.9	0.632
Prior CABG	2.9	3.6	3.2	0.128	3.9	2.6	3.6	0.036
Prior PCI	5.6	7.9	6.8	0.002	9.3	11.8	9.9	0.009
Prior CAD	75	75.3	75.2	0.806	74.1	771	74.9	0.033
Family history of CAD	6	4.6	5.4	0.017	5.8	7.7	6.2	0.016
Peripheral vascular disease	7.9	9.4	8.5	0.043	8.9	7.3	8.4	0.088
Anemia	20.7	44.7	31.1	<0.001	17.8	35.3	22.2	<0.001
Median household income category for patient's zip code (percentile)§				0.048				0.036
1. 0-25th	25.3	23.7	24.6		24.7	247.4	25.4	
2. 26-50th	28.6	26.5	27.7		29.3	25.6	28.4	
3. 51-75th	24.9	26.3	25.5		26.2	25.9	26.1	
4. 76-100th	21.3	23.5	22.3		19.8	21.2	20.1	
Primary Payer				<0.001				<0.001
Federal insurance	74.1	66.6	70.8		68	63.9	67	
Private insurance	19.3	22.8	20.8		21.6	29.7	23.7	
Self-pay/others	6.7	10.6	8.4		10.3	6.5	9.4	
Hospital characteristics								
Hospital bed size¶				<0.001				<0.001
Small	7.5	3.8	5.9		10.5	5.7	9.3	
Medium	23.3	17.5	20.8		24.3	17.1	22.5	
Large	69.2	78.7	73.3		65.2	77.2	68.2	
Hospital teaching status**				<0.001				<0.001
Non-Teaching	36.7	24.9	31.6		44.1	26	39.5	
Teaching	63.26	75.1	68.4		55.9	74	60.5	
Admission type				0.034				0.003
Non elective	92.9	91.4	92.2		89.2	86.1	88.4	
Elective	7.1	8.6	7.7		10.8	13.9	11.6	
Admission day				0.015				0.165
Weekdays	73.9	76.7	75.1		77.7	79.5	78.1	
Weekend	26.1	23.3	24.9		22.4	20.5	21.9	
Disposition				<0.001				<0.001
Home	6	22.2	13		38.6	41.4	39.3	
Facility/others	31.4	28.3	30		37	24.6	33.9	
In hospital mortality	62.4	49.5	56.8	<0.001	23.8	34.1	26.4	<0.001

CABG = coronary artery bypass graft; CAD = coronary artery disease; CKD = chronic kidney disease; COPD = chronic obstructive pulmonary disease; ECMO = extracorporeal membrane oxygenation; IABP = intra-aortic balloon pump; PCI = percutaneous coronary intervention; PVAD = peripheral ventricular assist device; MCS = mechanical circulatory support; TIA = transient ischemic attack.

*p-value comparing VSD repair vs No VSD repair in patients with shock.

† p-value comparing VSD repair vs No VSD repair in patients without shock.

‡ ICD-10 codes were utilized to identify respective comorbidities.

§ Represents a quartile classification of the estimated median household income of residents within the patients' ZIP Code, https://www.hcup-us.ahrq.gov/db/vars/zipinc_qrtl/nrdnote.jsp.

¶ The bed size cutoff points divided into small, medium, and large have been done so that approximately one third of the hospitals in a given region, location, and teaching status combination would fall within each bed size category. https://www.hcup-us.ahrq.gov/db/vars/hosp_bedsize/nrdnote.jsp.

**A hospital is considered to be a teaching hospital if it has an American Medical Association-approved residency program. https://www.hcup-us.ahrq.gov/db/vars/hosp_ur_teach/nrdnote.jsp.

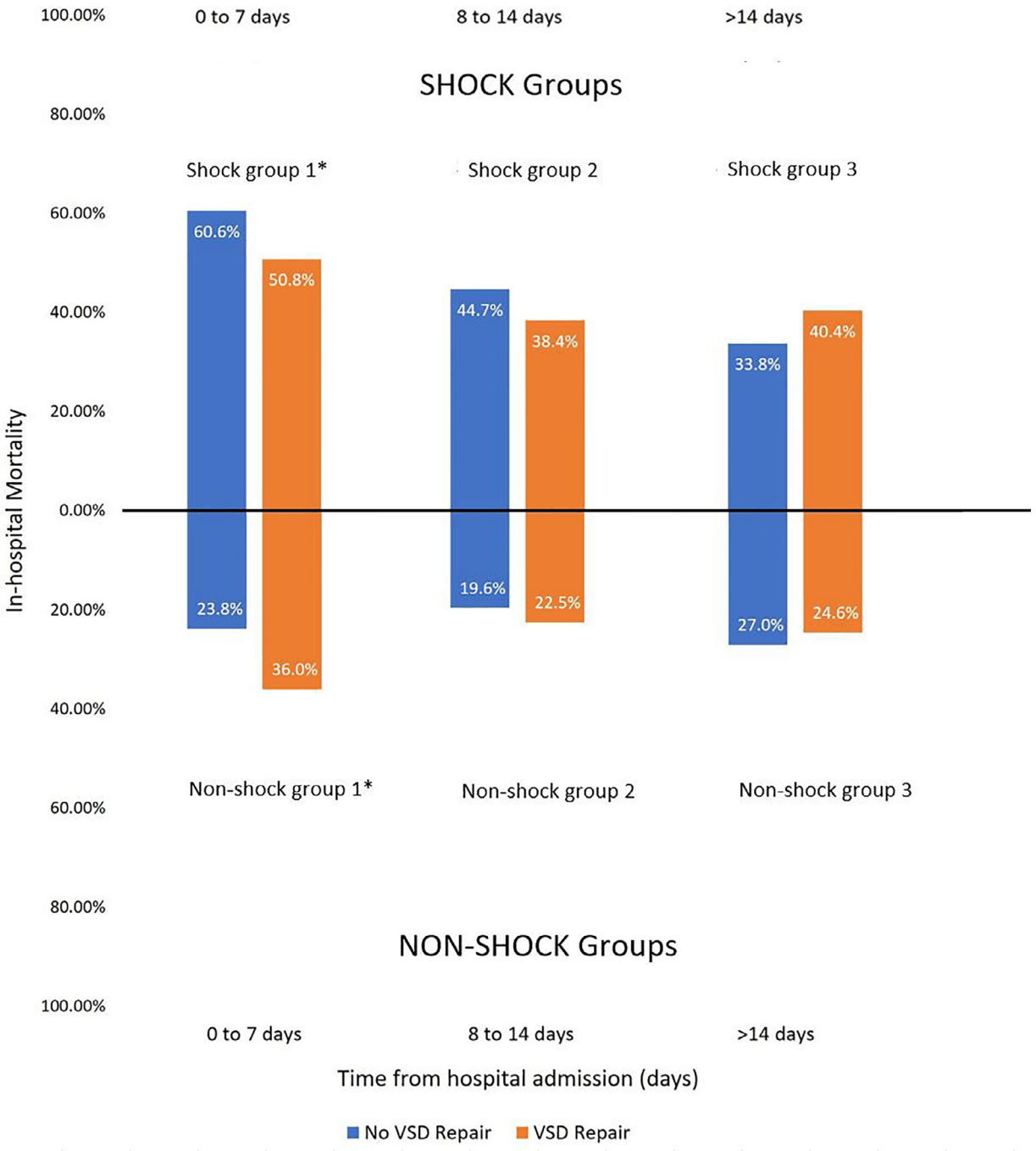

Figure 3. In-Hospital Mortality in PI-VSD by Time to VSD Repair and Shock. This figure illustrates in-hospital mortality in patients who underwent VSD repair (orange) compared no VSD repair (blue). Patients are stratified by time between hospital admission and VSD repair (0-7 days [left], 8-14 days [middle], and >14 days [right]) as well as presence of shock (top) or no shock (bottom). *Represents $p<0.05$.

Discussion

There is significant debate as to the optimal timing of postinfarction VSD repair primarily owing to limited data availability. The expansion of intensive care unit care and mechanical support available in the management of patients has offered options to defer surgery and allow for recovery of patients before invasive cardiac surgery. Guidelines have remained divergent on this subject, with United States guidelines recommending the pursuit of early repair and European guidelines recommending repair only in patients unable to be stabilized. A modern and large retrospective study using data from the Society of Thoracic Surgeons database was performed in 2012 and had demonstrated a mortality reduction in patients who had delayed postinfarction VSD repair compared with early intervention.[6,14] Selection and immortal time bias complicate the interpretation of this approach in determinining the optimal timing of repair. Lower mortality associated with late VSD repair

Table 2
Multivariate analysis of mortality in postinfarction VSD stratified by time since initial hospital presentation.

Time since initial hospital presentation	Patients at risk (n)	No VSD Repair	VSD Repair	p-value
Cardiogenic Shock*				
0 to 7 days (shock 1 group)	5,790	60.6 %	50.8 %	<0.001
		OR (95% CI): 0.76 (0.68-0.86)‡		<0.001
8 to 14 days (shock 2 group)	1,009	44.7 %	38.4 %	0.132
		OR (95% CI): 0.86 (0.57-1.27)‡		0.440
>14 days (shock 3 group)	483	33.8 %	40.4 %	0.214
		OR (95% CI): 0.90 (0.48-1.69)‡		0.753
No Cardiogenic Shock†				
0 to 7 days (non-shock 1 group)	5,108	23.8 %	36 %	<0.001
		OR (95% CI): 1.59 (1.33-1.90)‡		<0.001
8 to 14 days (non-shock 2 group)	1,265	19.6 %	22.5 %	0.445
		OR (95% CI): 0.77 (0.44-1.34)‡		0.351
>14 days (non-shock 3 group)	472	27 %	24.6 %	0.658
		OR (95% CI): 2.19 (0.76-6.36)‡		0.299

CI = confidence interval; OR = odds ratio; VSD = ventricular septal defect.
*Shock groups 1-3 as mentioned in figure 1 and in method section.
† Nonshock 1-3 groups as mentioned in figure 1 and in method section.
‡ Models were adjusted for age, gender, obesity, hypertension, diabetes mellitus, dyslipidemia, tobacco use, family history of coronary artery disease, personal history of coronary artery disease, CKD stage 3 or more, peripheral vascular disease, anemia, prior transient ischemic attack or stroke, prior coronary artery bypass grafting, prior percutaneous coronary intervention, heart failure, chronic obstructive pulmonary disease, median household income, hospital location, hospital teaching status, admission day.

likely represents the selection of patients who survived the early mortality associated with this condition.[6] This investigation sought to address this selection bias by comparing patient outcomes within the same time point to those who did not receive the intervention. When performing this comparison, patients in CS demonstrated potential benefit with early intervention, whereas those not in CS demonstrated potential harm with early intervention.

The findings of this study suggest that the decision of optimal timing for VSD repair requires a careful risk-benefit analysis. Those presenting with PI-VSD in CS represent patients with the most unfavorable prognosis even with intervention. Despite this dismal prognosis, patients demonstrated relatively lower rates of in-hospital mortality with VSD repair. Early intervention may help interrupt the spiral of hemodynamic deterioration that often drives increased early mortality. After significant irreversible end-organ damage has been established, later correction of hemodynamics may not significantly reduce mortality in this population.

In patients without CS, rates of in-hospital mortality were lower. Despite lower mortality, early intervention was associated with higher in-hospital mortality. In the setting of acute ischemia, inflamed and friable tissue directly adjacent to the defect may complicate surgical repair and lead to fatal postoperative complications. Delaying surgery may allow friable tissue to organize before surgical intervention, especially if the patient is not hemodynamically compromised.[1,15]

The advancement and incorporation of MCS in the care of patients in shock with PI-VSD has been a growing subject of study. Several studies have demonstrated improvement in hemodynamics in patients with PI-VSD treated with MCS devices, and guidelines have suggested these therapies as a method of hemodynamic stabilization.[16,17] Our study demonstrates consistently increased use of MCS over the last 15 years, predominantly in the form of PVAD and ECMO use. Despite this increase in utilization, our study has demonstrated that the pursuit of VSD repair and resultant mortality for patients presenting with PI-VSD remained relatively unchanged over the last 15 years, which has been demonstrated across multiple studies investigating outcomes in patients with PI-VSD.[3] On the basis of the results of our study, careful assessment and determination of optimal timing of VSD repair may offer an alternative strategy to help reduce mortality in this population.

The limitations include those related to the use of administrative databases. These errors include potential diagnostic coding errors leading to coding discrepancies. Furthermore, this database lacks information regarding symptom onset, laboratory data, echocardiographic data, and procedural data. Despite these limitations, this is the largest real-world analysis offering guidance about time of VSD repair in patients with PI-VSD, especially when a prospective randomized control trial would be logistically and ethically difficult to perform.

In conclusion, this study found high in-hospital mortality from PI-VSD in patients with CS. Early VSD repair in this cohort was associated with lower rates of in-hospital mortality. In contrast, in-hospital mortality was low in patients not in CS. Despite this, early VSD repair in this cohort was associated with a worse outcome. Our study suggests that delaying VSD repair in patients that demonstrate hemodynamic stability may be appropriate, but it may be counterproductive in patients with hemodynamic instability. Further study, in the form of a prospective trial, may be required to better understand this association and also understand the role of mechanical cardiac support in patients with PI-VSD and hemodynamic instability.

Figure 4. Trends of MCS and Mortality over time. This figure demonstrates the rates of (a) utilization mechanical circulatory support overall, (b) of each device category including intra-aortic balloon pump (blue), peripheral ventricular assist device (green), and extracorporeal membrane oxygenation (purple) along the left axis with mortality along the right axis over time, and (c) rate of VSD repair over time.

Disclosures

The authors have no conflict of interest to declare.

Supplementary materials

Supplementary material associated with this article can be found in the online version at https://doi.org/10.1016/j.amjcard.2022.04.017.

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