BMC Cardiovascular Disorders

RESEARCH

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Surgical versus percutaneous closure of post-infarction ventricular septal rupture; review of literature and single-center experience

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Abstract

Background Post-infarction ventricular septal rupture (PIVSR) is a rare mechanical complication, characterized by a dismal prognosis. Despite the widespread timely reperfusion and recent advances in management, short-term mortality of PIVSR remains high. The complexity of the hemodynamic profile, confusing evidence for the optimal timing for intervention, and lack of head-to-head trials, all make the management of such a condition very challenging.

Methods The database of a tertiary cardiac center was retrospectively analyzed for PIVSR cases through the period from April 2015 to April 2023. Clinical, echocardiographic, and interventional data were explored. The primary outcome was 30-day mortality that was contrasted for surgical versus percutaneous repair.

Results A total of 32 patients with PIVSR were identified. The median age was 65 years, 50% were males, 56% had diabetes, and 50% had cardiogenic shock (CS) on presentation, with a median time of 3 days from acute myocardial infarction (AMI) to PIVSR diagnosis. The median left ventricular ejection fraction (LVEF) was 38%. Culprit vessel patency was acutely restored in 26 patients (81%), while intra-aortic balloon pump (IABP) was inserted in 25 (78%). Upfront insertion of IABP (in the absence of CS) showed a trend towards improved survival (43% vs. 9%). PIVSR was surgically repaired in 15 patients (47%), while 9 (28%) underwent percutaneous device closure, with no significant difference in outcomes and with a median time to intervention of 21 days for both groups. The overall 30-day mortality rate was 44%. Acute kidney injury (AKI) was a significant predictor for 30-day mortality (odds ratio 7.5, 95%CI: 1.3 – 43.7, $p = 0.028$).

Conclusion PIVSR still carries a grave prognosis. Early surgical or percutaneous intervention seems associated with higher mortality, while upfront insertion of IABP for a safe deferral of repair beyond the acute phase may lead to better outcomes. Larger randomized studies are required to dictate the best management.

Keywords Ventricular Septal Rupture VSR, Acute myocardial infarction AMI, Cardiogenic shock CS, Percutaneous device closure, Intra-aortic balloon pump IABP

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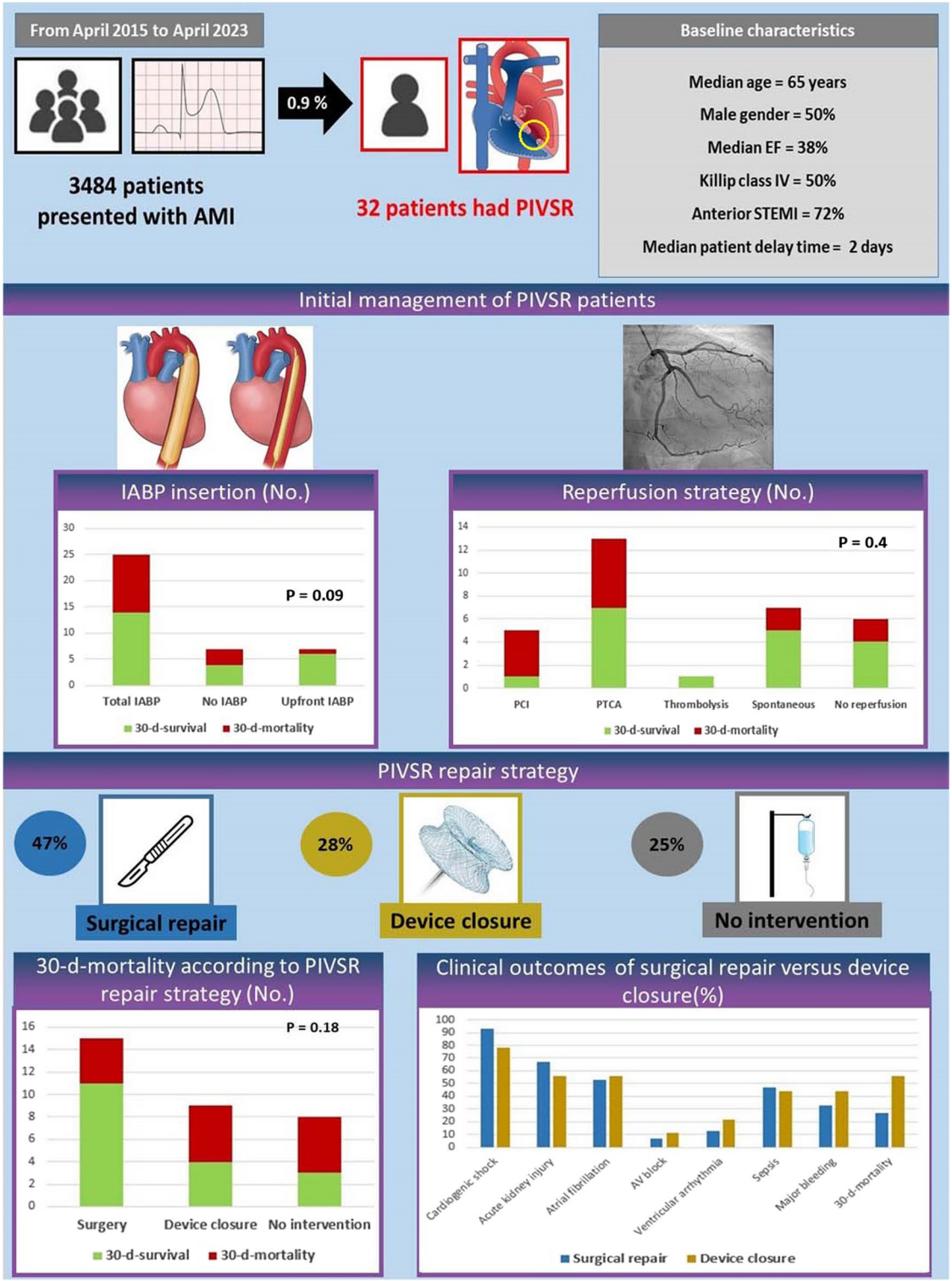
Graphical Abstract



Background

Despite the major advances in the management of acute myocardial infarction (AMI) [1], and the widespread implementation of timely reperfusion by percutaneous coronary intervention (PCI) [1], patients presenting with cardiogenic shock (CS) often have a dismal prognosis [2]. Among various mechanisms of CS complicating AMI, pump failure is the most common while mechanical complications remain the most deadly [3]. Mechanical complications after AMI, including papillary muscle rupture, free wall rupture, and ventricular septal rupture (VSR), have always been associated with challenging management and grave prognosis [4, 5].

VSR complicating AMI has a reported incidence of 0.17% - 0.31% [6], and a mortality rate of 41%–80% [7]. It causes an acute and massive increase in pulmonary flow, with enormous volume overload to the stunned ischaemic left ventricle (LV), and a significant drop in the effective cardiac output jeopardizing organs perfusion, with no exception to the coronary flow [4]. Hence, VSR often presents with acute heart failure (HF) and rapid hemodynamic deterioration with pulmonary edema and/or CS a few hours/days after AMI. Furthermore, in patients with VSR who present initially with stable hemodynamics, it is quite unpredictable how long they can tolerate the massive overload and when they would acutely worsen.

For the past decades, surgical repair for post-infarction VSR (PIVSR) was the only resort, with a huge dilemma considering the optimal timing for repair [8]. Despite the advantage of avoiding prolonged hemodynamic burdens, early surgical intervention on the infarcted friable myocardium carried a high risk for failure and/or recurrent rupture. Conversely, delaying the surgery for several days striving for stabilization (fibrosis) of the rupture edges may risk sudden hemodynamic crashing after exhaustion of all homeostatic reserves and subsequent multiple system-organ failure [5, 9].

Through the past few years, percutaneous PIVSR repair has been an appealing alternative to surgical repair. Nevertheless, appropriate case selection and procedural planning including timing and technicalities are still highly individualized.

In this observational report, we retrospectively reviewed a single-tertiary-center experience in the presentation, management, and outcomes of AMI patients complicated by PIVSR.

Methods

Patient inclusion

This registry represents the experience of a tertiary center that acts as a hub for a network of non-primary PCI-capable centers. The local AMI database through the period from April 2015 to April 2023 was searched for VSR cases. Medical records for identified cases were manually reviewed to confirm the diagnosis and analyze patients' characteristics and hospital courses.

After reviewing the patients' files and clinical data, profiling was made by defining the Killip class of heart failure (HF), admission electrocardiograms (ECG), baseline laboratory data, and in-hospital events. All relevant time intervals were retrieved and registered, including chest pain onset-to-presentation, chest pain onset-to-VSR diagnosis, and VSR diagnosis-to-VSR repair.

The thorough transthoracic echocardiography (TTE) that led to PIVSR diagnosis was reviewed. Often non-standard, modified views were required to assess the maximum diameter and the gradient across the VSR. The LV dimensions and ejection fraction (EF), regional wall motion abnormalities, grades of valve dysfunction, right ventricular (RV) dimensions and function, and estimated RV systolic pressure, were acquired and tabulated.

Coronary procedure

Coronary angiograms were reviewed from the local archiving system, revising all the data concerning the infarct-related artery (IRA), including the baseline Thrombolysis In Myocardial Infarction (TIMI) flow [10], the final TIMI flow (if reperfusion was performed) and whether other severe non-culprit lesions existed. Restoration of culprit vessel patency was registered as: (i) percutaneous transluminal coronary angioplasty (PTCA) when balloon only was used; (ii) percutaneous coronary intervention (PCI), when coronary stent(s) were implanted; (iii) successful thrombolysis, when a patient had received thrombolysis and demonstrated TIMI flow ≥ 2 on subsequent angiography; (iv) spontaneous, when the culprit vessel shows spontaneous recanalization; or (v) no reperfusion, when restoration of culprit lesion patency was deemed futile in late presentations (>48 hours) and evidence of completely scarred corresponding myocardial territory.

Whenever possible, the operators opted for balloon angioplasty rather than stenting the IRA, not to complicate the scenario if the patient was to proceed to surgery after the multi-disciplinary assessment. However, if the results were suboptimal after balloon angioplasty, stenting of the IRA was executed

IABP insertion

According to the institutional policy and in line with practice guidelines [1], AMI patients with CS due to a mechanical complication (like PIVSR) received an intra-aortic balloon pump (IABP). However, IABP was occasionally implanted upfront (prophylactically) for stable VSR patients, according to the operator's discretion, striving for a safe deferral of the repair for several days to weeks.

PIVSR repair strategy

PIVSR repair strategy (surgical versus percutaneous) was elected based on a case-by-case heart team discussion, considering the VSR characteristics, coronary anatomy, other indications for heart surgery (such as severe valve dysfunction or unsuitability for percutaneous revascularization), and the patient's estimated procedural risk. Generally, patients selected for percutaneous device closure had percutaneous revascularization, either in a separate- or same session, while those selected for surgical repair were evaluated for a concomitant coronary artery bypass grafting (CABG), aneurysmectomy, or valve intervention according to the pre-operative anatomical assessment.

All surgically treated patients underwent left ventriculotomy and septal defect closure by infarct exclusion with an endocardial patch as per the technique described by David et al. [11]. More details about surgical repair and revascularization (whenever performed) are added to the supplementary files.

All percutaneous device closure procedures were done under fluoroscopic plus either TTE or trans-esophageal echocardiography (TEE) guidance. Pre-procedural multimodality imaging including cardiac computed tomography (CCT) or cardiac magnetic resonance (CMR) was performed whenever deemed safe and feasible for detailed assessment of the defect characteristics, however, occasionally patient instability was a limitation. Assessment of the closure efficacy was routinely performed to identify residual or recurrent shunts. Echocardiographic grading of residual shunt severity relied on color Doppler assessment of the jet neck, where a width of < 2, 2-to-4, or > 4 mm defined mild, moderate, or severe shunts, respectively [12]. Technicalities of the PIVSR percutaneous closure procedures are detailed in the supplementary files.

The clinical course to discharge, then periodic clinical follow-up assessments through outpatient visits were reviewed and tabulated.

The 30-day mortality was defined as all-cause death within 30 days from hospital admission. Subsequently, the study cohort was subdivided according to this variable into 2 subgroups; 30-day survivors and 30-day mortality, where different patient characteristics and outcomes were contrasted to find relevant correlates.

Ethical committee approval

The registry protocol and methodology were thoroughly reviewed and approved by the Institutional Research Ethics Committee (AHC-REC). Due to the complete retrospective nature of the study, and assessment of completely anonymized data without any breach of patients' confidentiality, the need to have informed signed consent from participants was waived by AHC-REC (approval number: 20230728MYFAHC_VSRReg20230803).

Statistical analysis

Statistical analysis was conducted using IBM Statistical Package for Social Science software for Microsoft Windows, version 24 (IBM SPSS Statistics for Windows, Armonk, NY: IBM Corp). Categorical variables were represented as frequency and percentages. While continuous variables were subjected to normality testing and were expressed as median [inter-quartile range (IQR): 25^{th} – 75^{th} percentiles], or mean ± standard deviations (SD) accordingly. Chi-square or Fisher exact tests were used to compare categorical variables and the rank sum test was used for comparisons of continuous variables. Logistic regression analysis was performed to find predictors for the 30-day mortality. Survival analysis was performed using the Kaplan Meier method by Log-rank test. In case of early separation of curves with consistent higher risk in one group, Gehan-Breslow-Wilcoxon test was performed. Univariate Cox regression analysis was done to examine the effect of significant variables on 30-day survival over time. Hazard ratio (HR) and 95% confidence interval (CI) were calculated. A p-value of less than 0.05 was considered significant.

Results

Through the period between April 1^{st}, 2015 -to- April 30^{th}, 2023; 3484 AMI (both STEMI and NSTEMI) cases were received by our system. After a thorough review, there were 32 cases with confirmed diagnoses of PIVSR, representing an incidence of 0.9%. All PIVSR cases were post-STEMI.

Baseline clinical characteristics

The study group had a median [IQR] age of 65 years [60 - 73], with comparable gender distribution. Among the whole cohort, 18 patients (56%) had diabetes mellitus, 14 (44%) had systemic hypertension and 11 (34%) were current smokers. Only 1 patient had a prior history of AMI while the index AMI was the first for the 31 remainder cases. The basic demographic features of the whole cohort and the differences between the 30-day survivors (n=18) versus the 30-day mortality (n=14) subgroups are demonstrated in Table 1.

Upon presentation, 16 patients (50%) were classified as Killip IV (cardiogenic shock), with a rate in the 30-day survivors' that is less than half the rate in the 30-day mortality group, (33% vs. 71%, p = 0.07). The left anterior descending (LAD) artery was the most frequent IRA in both groups followed by the right coronary artery (RCA). A single case of PIVSR complicating lateral STEMI was encountered, with a large Ramus identified by angiography as the IRA. The median LVEF on admission was 38% [25% - 50%] with no difference between the 2

Table 1 Baseline characteristics of the study cohort and the two subgroups based on 30-day mortality

	Total VSR group ($n=32$)	30-day-survival group ($n=18$)	30-day-mortality group ($n=14$)	*p*-value
Age (years)	65 (60–73)	64 (55–71)	66 (60–74)	0.36
Female gender	16 (50%)	8 (44.4%)	8 (57.1%)	0.72
BMI (Kg/m^2)	27.7 (24.8–31.2)	27.5 (24.7–30.8)	27.9 (25.2–31.2)	0.72
Diabetes mellitus	18 (56%)	8 (44.4%)	10 (71.4%)	0.16
Systemic hypertension	14 (43.8%)	7 (38.9%)	7 (50%)	0.72
Dyslipidemia	9 (28.1%)	7 (38.9%)	2 (14.3%)	0.23
Smoking	11 (34.4%)	8 (44.4%)	3 (21.4%)	0.27
Family H/o premature CAD	1 (3.1%)	1 (5.6%)	0 (0%)	>0.99
Prior MI	1 (3.1%)	1 (5.6%)	0 (0%)	>0.99
Killip class IV on admission	16 (50%)	6 (33.3%)	10 (71.4%)	0.07
Admission Systolic BP	109 (93–120)	110 (99–116)	96 (88–122)	0.56
Admission heart rate	115 (103–130)	110 (89–121)	123 (110–143)	0.23
LVEF (%)	38 (25–50)	35 (25–45)	40 (35–50)	0.33
VSR gradient (mmHg)	46 (30–58)	46 (30–56)	50 (35–65)	0.59
VSR diameter (mm)	13 (9–15)	13.5 (8.5–16)	13 (11–14)	0.68
VSR site				0.17
Apical	22 (73.3%)	15 (83.3%)	7 (58.3%)	
Basal	8 (26.7%)	3 (16.7%)	5 (41.6%)	
Significant MR	5 (16.6%)	5 (27.8%)	0 (0%)	0.05
RV dysfunction	6 (20%)	4 (22.2%)	2 (16.7%)	>0.99
Peak Troponin (mg/dL)	8.5 (2.1–24.0)	4 (2–9.6)	14.8 (4.9–34.8)	0.16
Hemoglobin (gm/dL)	12.9 (11.3–14.1)	13 (11.5–13.4)	12.4 (11–15)	0.61
HbA1C (%)	7.7 (5.9–9.8)	7.1 (6–9.7)	8.5 (5.9–10.1)	0.68
AMI – FMC (days)	2 (0.7–5)	2 (0.8–5)	1.5 (0.5–5)	0.64
AMI – VSR (days)	3 (1–6)	3 (2–6)	4 (1–6)	0.84
Infarct related artery				0.15
LAD	23 (71.9%)	15 (83.3%)	8 (57.1%)	
RCA	8 (25%)	3 (16.7%)	5 (35.7%)	
Ramus	1 (3.1%)	0 (0%)	1 (7.1%)	
Single-vessel disease	15 (46.9%)	10 (55.6%)	5 (35.7%)	0.31
Multi-vessel disease	17 (53.1%)	8 (44.4%)	9 (64.3%)	
Acute reperfusion mode				0.41
Spontaneous	7 (21.9%)	5 (27.8%)	2 (14.3%)	
Thrombolytic	1 (3.1%)	1 (5.6%)	0 (0%)	
PTCA	13 (40.6%)	7 (38.9%)	6 (42.9%)	
PCI by DES	5 (15.6%)	1 (5.6%)	4 (28.6%)	
No reperfusion	6 (18.8%)	4 (22.2%)	2 (14.3%)	
Restored Culprit Patency	26 (81.3%)	14 (77.8%)	12 (85.7%)	0.67

Data expressed as median (25th-75th percentile) or frequency (percent) as appropriate

AMI Acute myocardial infarction, *BMI* Body mass index measured in kilograms/meter^2, *BP* Blood pressure, *CAD* Coronary artery disease, *FMC* First medical contact, *HbA1c* Glycated hemoglobin, *H/o* History of, *LAD* Left anterior descending, *LVEF* Left ventricular ejection fraction, *MI* Myocardial infarction, *MR* Mitral regurgitation, *PCI* Percutaneous coronary intervention, *PTCA* Percutaneous transluminal coronary angioplasty, *RCA* Right coronary artery, *RV* Right ventricle, *STEMI* ST-elevation myocardial infarction, *VSR* Ventricular septal rupture

subgroups. A significant mitral regurgitation (MR) (grade ≥3 by TTE) was found in 5 patients, all of whom were among the 30-day survivors ($p = 0.052$). Time delays for the whole PIVSR cohort were substantially longer than usual. In our records of 3484 AMI patients, pain-to-first medical contact time delay had a median of 3 (1–5) hours, with less than 2.6% presenting later than 24 hours in contrast to PIVSR patients who had a median delay of 2 days, yet with no significant difference between the 30-day survivors and the 30-day mortality subgroups.

Acute restoration of culprit vessel patency was achieved in 78% vs. 86% in the 30-day survival vs. the 30-day mortality group, respectively. Overall, 25 patients (78%) received IABP with a comparable overall rate in both groups, while the upfront IABP strategy was implemented in 6 patients (43%) of the 30-day survivors vs. only 1 patient (9%) of the 30-day mortality group, yet with a non-significant *p*-value ($p = 0.09$). Of the 25 patients who received IABP, 2 developed acute lower limb ischemia (8%) that necessitated IABP removal without the need for vascular intervention. One of those underwent surgical repair of PIVSR and survived to discharge, while the other died in-hospital before intervention.

VSR dedicated management

Based on a case-by-case heart team discussion, 15 patients (47%) underwent surgical repair, while 9 (28%) underwent percutaneous device closure. One patient among the percutaneous closure group had acute device embolization and was transferred for emergency surgery. Of note, 8 patients (25%) did not undergo repair intervention, where 5 patients showed rapid deterioration with irreversible shock and died before attempting repair, 1 patient developed sepsis precluding proceeding to the intervention (beyond 30 days), then finally died of septicemia, and 2 patients (fortunately with tiny PIVSR) refused any intervention and favored conservative management. The data concerning VSR repair strategies are detailed in Table 2.

In the group selected for percutaneous closure, 3 patients underwent PCI, all scheduled in a separate session 1–2 weeks before PIVSR intervention. The default closure device was the Amplatzer atrial septal defect (ASD) occluder (Abbott Vascular, Santa Clara, California), except for 2 cases that received a LifeTech Konar multifunctional occluder (MFO) (LifeTech Scientific, Shenzhen, China), and a single case who received an Amplatzer muscular ventricular septal defect (VSD) occluder.

The mean device diameter was 19 mm (minimum 14 mm and maximum 27 mm). Technical success was 89% (8/9), while procedural success was 78% (7/9). Actually, the 2 major peri-procedural complications occurred after the device release. A patient developed incessant ventricular tachycardia (VT) that remained refractory to electric cardioversion and antiarrhythmics, then eventually degenerated into ventricular fibrillation and death. Another case was complicated by acute device migration and was transferred to emergency surgery.

Sixteen patients underwent surgical repair (15 as a primary strategy and 1 after emergent transfer from the Cath Lab after device embolization). Among those 16, concomitant CABG was performed for 8 patients (50%), while the others were either revascularized by PCI or deemed unindicated (coronary occlusion is very distal or supplying a scar). All 8 patients who underwent VSR repair and CABG survived the 30-day post-admission, while 5 of the 8 patients (62.5%) who underwent VSR repair without CABG met the 30-day mortality, $p = 0.026$, of whom, only one patient died intra-operatively. Also, concomitant aneurysmectomy was performed in 6 patients (37%) of the surgical group with a non-significant difference between 30-day survival or mortality (5 vs. 1, $p = 0.588$). A small residual VSD was detected in 4 cases however, it didn't affect the 30-day mortality (Figs. 1, 2 and 3).

In-hospital course and outcomes

CS was the most common in-hospital complication affecting 28 patients (87%) of the whole cohort, followed by AKI (20 patients, 62%), atrial fibrillation (14 patients, 44%), sepsis (12 patients, 37%), and major bleeding (11 patients, 34%). Patients in the 30-day mortality group showed significantly higher rates of AKI compared to the 30-day survivors. In binary logistic regression, AKI was a significant predictor for 30-day mortality, with an odds ratio of 7.5 (95%CI: 1.3 – 43.7, $p = 0.028$). Various

Table 2 VSR dedicated management

	30-day-survival group ($n = 18$)	30-day-mortality group ($n = 14$)	*p*-value
Received IABP	14 (78%)	11 (79%)	> 0.99
Upfront IABP	6 (43%)	1 (9%)	0.09
IABP duration (days)	15 (9–24)	10 (4–14)	0.06
VSR Intervention			0.18
Surgical repair	11 (61%)	4 (29%)	
Percutaneous device closure	4 (22%)	5 (36%)	
No intervention	3 (17%)	5 (36%)	
Residual VSD	3 (21%)	1 (13%)	> 0.99
VSR diagnosis -to– Intervention (days)	31 (16–69)	14 (6–18)	**0.034**

Data expressed as median (25th-75th percentile) or frequency (percent) as appropriate

IABP Intra-aortic balloon pump, *VSD* Ventricular septal defect, *VSR* Ventricular septal rupture

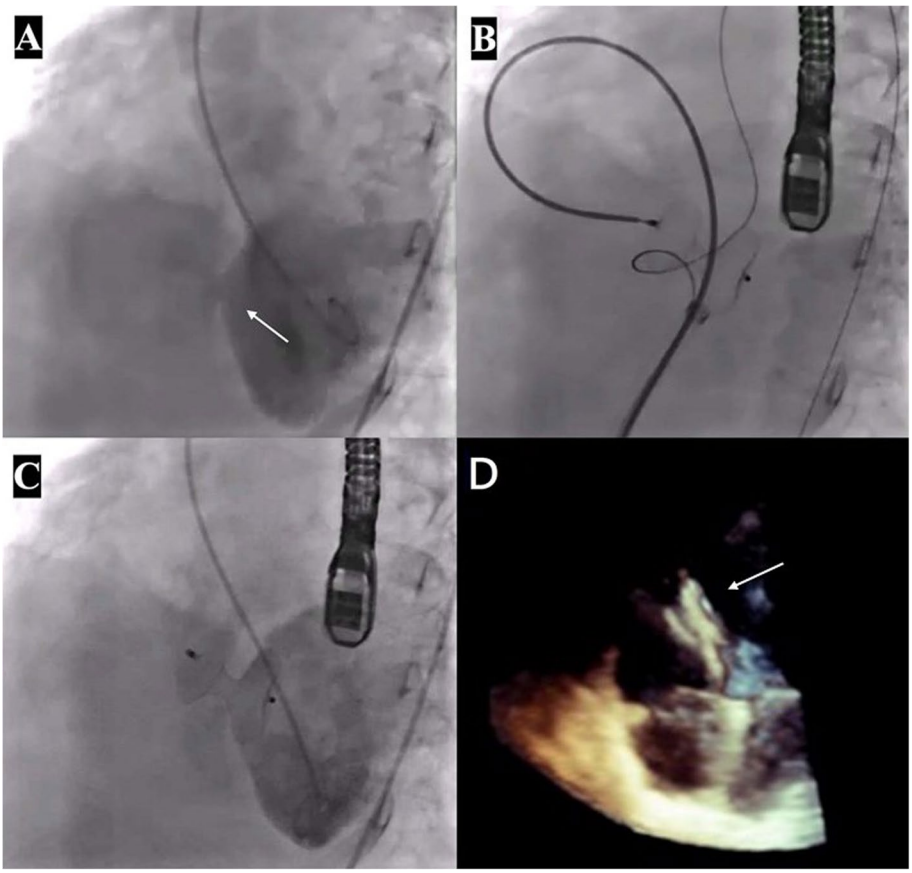


Fig. 1 **A** Left ventriculography showing left to right shunting through a PIVSR (white arrow). **B** TEE-guided release of an Amplatzer atrial septal occluder device. **C** Post-release ventriculography showing minimal residual shunt. **D** Follow-up three-dimensional transthoracic echocardiography after 2 years showing well-seated device on the inter-ventricular septum (white arrow). PIVSR, Post-infarction ventricular septal rupture; TEE, Trans-esophageal echocardiography

in-hospital complications are detailed in Table 3, while features' association with mortality are shown in Table 4

Contrasting features and outcomes of percutaneous device closure vs. surgical repair

Compared to patients elected for primary surgical repair (n=15), patients in the percutaneous device closure group (n=9) were characterized by a numerically predominant female gender, higher rates of systemic hypertension, and lower rates of smoking and dyslipidemia. There was a significantly longer duration from symptom onset to PIVSR diagnosis in the percutaneous closure group (7 days vs. 3 days in the surgical repair group, p = 0.047). Otherwise, baseline characteristics had no significant differences between the 2 groups. Analysis of the pre-procedural reports revealed that VSR site (assessed binarily as apical vs. basal), VSR diameter, or trans-defect gradient, showed no significant differences between those elected for percutaneous vs. surgical closure strategies. Comparisons between the percutaneous closure- vs. the surgical repair groups concerning patients' characteristics and outcomes are demonstrated in Tables 5 and 6, respectively. Nevertheless, improved survival was observed in patients with a longer median time from VSR diagnosis-to-VSR intervention (31 days vs. 14 days, p = 0.034) irrespective of the repair strategy.

Time-to-event analysis

Kaplan–Meier survival curves are illustrated in Fig. 4. Notably, patients presenting with CS showed a significant association with mortality over 30 days (HR: 4.0, 95% CI: 1.3 – 13.0, p=0.019). Similarly, patients who did not undergo any intervention for PIVSR had earlier mortality. On the other hand, there was no significant association between 30-day mortality and the delay time to presentation, nor IABP insertion.

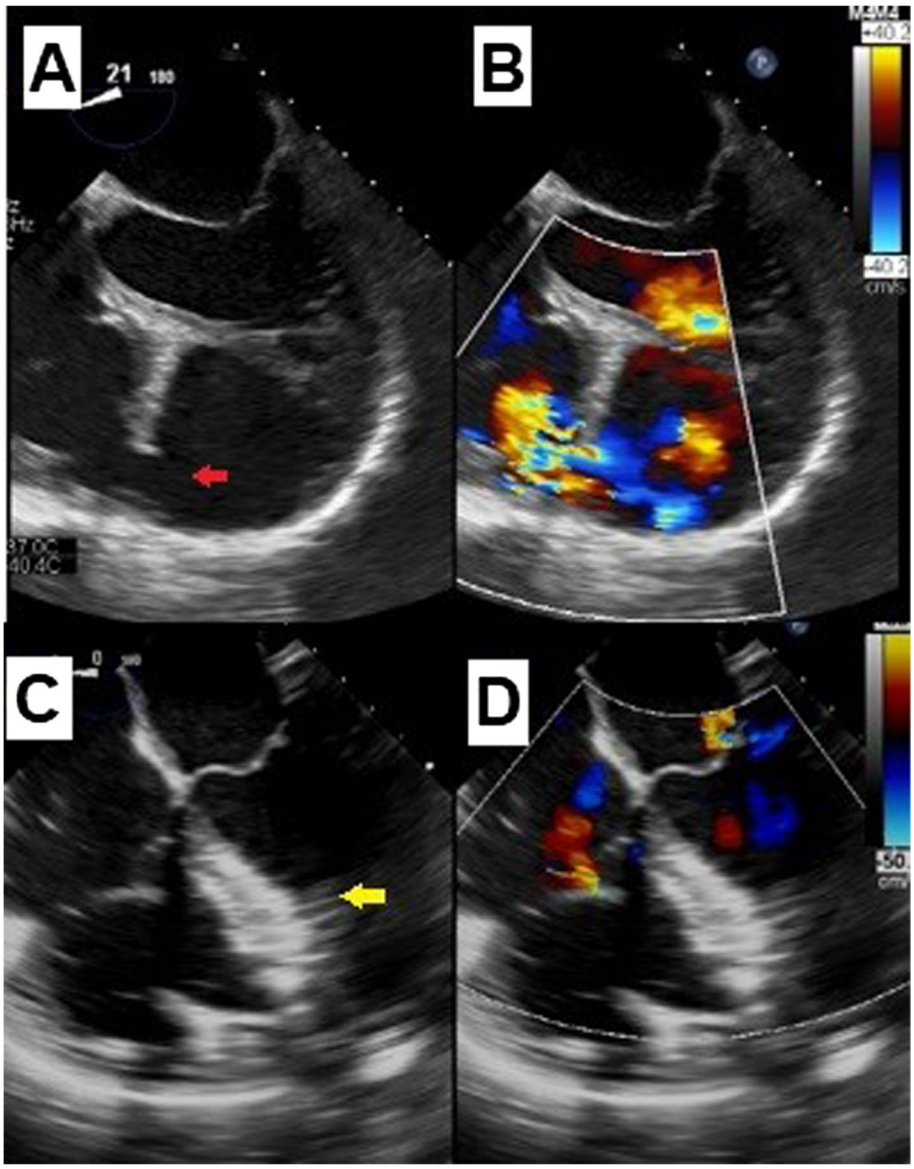


Fig. 2 Intra-operative TEE, demonstrating a color compare for 4-chamber view. Panels **A** and **B** show an apical PIVSR (red arrow) with left to right shunt. Panels **C** and **D** show the post-surgical outcome showing a septal patch (yellow arrow) with no residual shunt. PIVSR, Post-infarction ventricular septal rupture; TEE, Trans-esophageal echocardiography

Discussion

Incidence and outcomes of PIVSR

PIVSR is a rare but devastating mechanical complication that may ensue AMI [6, 7, 13]. It classically occurs in a bimodal pattern, where its incidence peaks in the first 24 hours and then again by the 3rd -to- 5th day after AMI [13]. A differential incidence after STEMI versus non-ST segment elevation myocardial infarction (NSTEMI) is controversial, but several registries report equivalent incidences [5, 14]. A large PIVSR often leads to immediate CS [5]. Conversely, a relatively small PIVSR may initially show stable hemodynamics, nevertheless, those patients frequently show sudden rapid deterioration after few days to weeks if left unintervened [5].

MCS is often offered to PIVSR patients presenting with pulmonary edema and/or CS. Current practice guidelines recommend considering an IABP for AMI patients with CS complicating a mechanical complication [1]. Other devices including Extracorporeal Membrane Oxygenator (ECMO), Impella, Tandem heart, or ECPELLA (combined ECMO and Impella) were attempted with

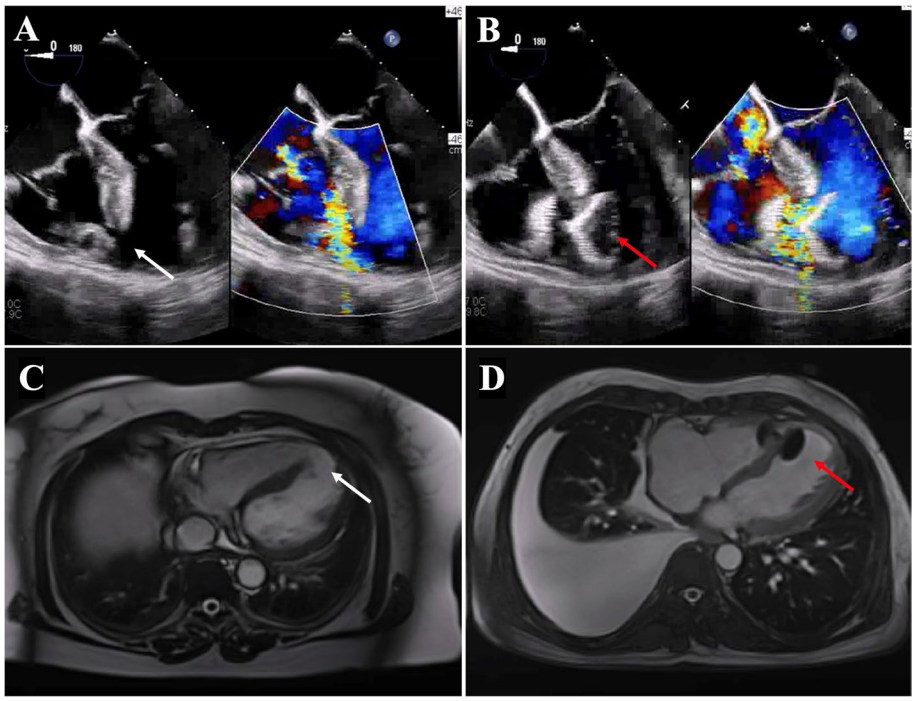


Fig. 3 Baseline and after percutaneous closure of a PIVSR imaging by color-compare TEE (**A** and **B)** and cardiac magnetic resonance imaging (**C** and **D**). The defect was located in the apical septum (white arrow). The occluder is well-seated with minimal residual shunt (red arrow). PIVSR, Post-infarction ventricular septal rupture; TEE, Trans-esophageal echocardiography

promising preliminary data [15–18], yet, further supportive evidence is needed.

In the present registry, 32 PIVSR patients were identified, representing 0.9% of the AMI registry of a tertiary cardiac center. Coronary artery disease (CAD) has consistently shown an earlier onset and accelerated course in the Middle East region, rendering the mean age of the first AMI 10–12 years earlier than in the West [19–21]. Although such a tendency to premature CAD could have promoted the development of coronary collaterals and provide a protective effect against myocardial rupture [5, 21], it seems that this is counterbalanced by other unclear factors rendering PIVSR incidence comparable in different regions. The female gender represented 32% of our AMI database, nevertheless, 50% in this PIVSR registry were females, supporting the universal higher female-to-male PIVSR incidence.

Table 3 In-hospital clinical outcomes

	30-day-survival group ($n=18$)	30-day-mortality group ($n=14$)	*p*-value
Cardiogenic shock	14 (78%)	14 (100%)	0.11
Atrial fibrillation	8 (44%)	6 (43%)	> 0.99
High-grade heart block	0 (0%)	3 (21%)	0.07
Acute kidney injury	8 (44%)	12 (86%)	**0.028**
Dialysis	6 (33%)	9 (64%)	0.15
Major bleeding events	4 (22%)	7 (50%)	0.14
Stroke	1 (6%)	0 (0%)	> 0.99
Sepsis	8 (44%)	4 (29%)	0.47
Pneumonia	3 (18%)	1 (7%)	0.61

Data expressed as median (25th-75th percentile) or frequency (percent) as appropriate

Characteristically, PIVSR patients in our registry had very delayed presentation with a median pain-to-FMC of 2 days compared to 3 hours in the general AMI cohort. Also, for all except a single patient, the index AMI was the first MI in life. It is also presumed that previous myocardial insults with the ensuing fibrosis are often protective against PIVSR [5].

It was demonstrated that presenting with CS was numerically associated with poorer outcomes and excess 30-day mortality, yet with a statistical trend. Although the solid guidelines' recommendations for MCS address PIVSR patients with CS, recent experiences report a general inclination for more liberal use of MCS in initially stable patients, striving for a safer deferral of VSR repair [22]. Operators frequently opt to insert IABP (or alternative MCS) in stable patients aiming to reduce the shunt volume and minimize chances for sudden hemodynamic deterioration, allowing to delay the reparative

Table 4 Features' association with 30-day mortality

	30-day-survival ($n=18$)	30-day-mortality ($n=14$)	*OR (95% CI) for 30-day-mortality*	*p-value*
Killip class IV on admission	6 (33%)	10 (71%)	5.0 (0.97 – 22)	0.07
Significant MR on admission	5 (28%)	0 (0%)	0.4 (0.02 – 11)	0.05
Restored culprit patency	14 (78%)	12 (86%)	1.7 (0.26 – 11)	0.67
Upfront IABP	6 (43%)	1 (9%)	0.1 (0.01 – 1.4)	0.09
Residual VSD post-repair	3 (21%)	1 (13%)	0.5 (0.04 – 6.1)	>0.99
In-hospital cardiogenic shock	14 (78%)	14 (100%)	9 (0.44 – 18.3)	0.11
Atrial fibrillation	8 (44%)	6 (43%)	0.9 (0.23 – 3.8)	>0.99
High grade heart block	0 (0%)	3 (21%)	11.3 (0.53 – 23.9)	0.07
Major bleeding events	4 (22%)	7 (50%)	3.5 (0.76 – 16.1)	0.14
Acute kidney injury	8 (44%)	12 (86%)	**7.5 (1.30 – 43.7)**	**0.028**
Dialysis	6 (33%)	9 (64%)	3.6 (0.63 – 15.6)	0.15
Pneumonia	3 (18%)	1 (7%)	0.4 (0.03 – 3.9)	0.61
Stroke	1 (6%)	0 (0%)	0.4 (0.01 – 10.6)	>0.99

Data expressed as frequency (percent) as appropriate

OR Odds ratio, *CI* Confidence interval, *MR* Mitral regurgitation, *IABP* Intra-aortic balloon pump, *VSD* Ventricular septal defect

intervention for several days. This strategy is not yet valued by the guidelines; however, it appears very promising in many recent reports utilizing IABP or other MCS modalities [22]. Our results support that a liberal strategy for upfront IABP implantation may be associated with better 30-day survival, though, the limited case number likely led to the non-significant *p*-value.

Surgical repair of PIVSR

Surgical closure remains the gold standard management for PIVSR [23–25]. However, it is still plagued by high peri-procedural mortality and a lack of determination on the ideal timing for intervention [8, 25, 26]. Daggett's technique [27] for PIVSR repair entails excision of the infarcted tissue and closing the defect by a patch sewn to the left and right ventricles [28]. On the other hand, David's procedure is an infarct exclusion technique, where all sutures are placed in the LV [11]. Studies have shown that infarct exclusion repair portended better results compared to Daggett's repair [25, 29]. Generally, repair of posterior VSR has been exceptionally challenging as compared to apical VSR. This is mainly attributed to its higher likelihood of having a complex serpiginous course besides that adequate exposure of the defect necessitates heart elevation, and risks injury to the posterior descending artery and posteromedial papillary muscle [25].

In other registries of PIVSR surgical repair, several patient characteristics were associated with increased 30-day mortality including older age, smoking, chronic kidney disease, posterior PIVSR, significant RV dysfunction, and preoperative CS or cardiac arrest. Also, short intervals from AMI to repair (particularly < 7 days), emergent surgery, longer CPB, re-operation for bleeding, and postoperative MCS dependency were identified as procedure-related factors associated with higher short-term mortality [25, 30].

The evidence about the benefit of concomitant CABG with PIVSR repair remains controversial [24, 31]. Although few reports implied a trend towards harm, likely due to the prolongation of CPB times, other studies emphasized its benefit, particularly in multi-vessel CAD [32]. A plausible confounder in this conflict may be the transition from the era of immediate surgical repair to PIVSR (when concomitant revascularization would confer the maximum benefit), to the contemporary inclination to defer repair beyond the acute phase where the harms of prolonged bypass time overweigh the minimal benefit from delayed revascularization.

In this registry, VSR repair and concomitant CABG had better outcomes than VSR repair only. However, it is critical to note that the group not receiving concomitant CABG represented either those who had undergone PCI before surgery or those with evidence of completely scarred myocardial territory. Hence, the poorer outcomes may be attributed to excess bleeding for the uninterrupted antiplatelet therapy or a poor LV function with extensive scarring.

We reported a post-operative mild residual VSD in 25% of the surgical group. In several other registries, a small residual shunt is reported as frequent as 21% yet, with no effect on short-term outcomes, even if re-intervention was performed [25].

Table 5 Patients and procedural characteristics dichotomized by the PIVSR primary repair strategy

	Device Closure ($n=9$)	Surgical Repair ($n=15$)	*p*-value
Age (years)	64 (55–74)	63 (60–70)	0.95
Female gender	7 (77.8%)	6 (40%)	0.10
BMI (Kg/m^2)	28.1 (26.7–34.2)	27.7 (24.9–30.5)	0.52
Diabetes mellitus	6 (66.6%)	8 (53.3%)	0.68
Systemic hypertension	7 (77.7%)	5 (33.3%)	0.09
Dyslipidemia	1 (11.1%)	8 (53.3%)	0.08
Smoking	1 (11.1%)	7 (46.6%)	0.18
Past history of MI	0 (0%)	1 (6.6%)	> 0.99
AMI – FMC (days)	3 (1–6)	2 (0.7–5)	0.71
AMI – VSR diagnosis (days)	7 (4–9)	3 (2–5)	**0.047**
Killip class IV on admission	2 (22.2%)	8 (53.3%)	0.21
LVEF (%)	40 (30–40)	40 (25–55)	0.86
Significant MR	3 (33.3%)	1 (6.7%)	0.13
EuroSCORE II	0.24 ± 0.12	0.27 ± 0.16	0.73
VSR site			0.47
Apical	7 (77.8%)	12 (80%)	
Basal	2 (22.2%)	3 (20%)	
VSR gradient (mmHg)	58 (45–75)	45 (30–50)	0.12
VSR size (mm)	12 (10–15)	14 (11.5–17.5)	0.38
RV dysfunction	2 (22.2%)	3 (20%)	> 0.99
Peak Troponin (mg/dL)	12.2 (2.4–31.8)	4.5 (1.9–9.9)	0.16
Hemoglobin (gm/dL)	12 (11–15)	13.2 (11.6–14.7)	0.52
HbA1C (%)	8.5 (6.3–9.7)	9 (6.5–10.6)	0.43
Infarct related artery			> 0.99
LAD	7 (77.7%)	12 (80%)	
RCA	2 (22.2%)	3 (20%)	
Restored Culprit Patency	8 (88.8%)	10 (66.6%)	0.35
Immediate PCI	3 (33%)	0 (0%)	0.13
IABP insertion	6 (66.6%)	13 (86.6%)	0.33
Upfront IABP	2 (33.3%)	3 (23.1%)	0.52
Total IABP days	11 (8–19)	14 (10–19)	> 0.99
VSR diagnosis to VSR intervention (days)	21.5 (16–31.5)	21 (8.5–64.5)	> 0.99

Data expressed as median (25th-75th percentile) or frequency (percent) as appropriate

AMI Acute myocardial infarction, *BMI* Body mass index measured in kilograms/meter^2, *FMC* First medical contact, *HbA1c* Glycated hemoglobin, *IABP* Intra-aortic balloon pump, *LAD* Left anterior descending, *LVEF* Left ventricular ejection fraction, *MI* Myocardial infarction, *MR* Mitral regurgitation, *PCI* Percutaneous coronary intervention, *RCA* Right coronary artery, *RV* Right ventricle, *VSR* Ventricular septal rupture

Percutaneous device closure of PIVSR

Although initially emerged as an off-label bail-out management for patients at prohibitive surgical risk, percutaneous repair has become an established alternative option for PIVSR in many proficient centers [33]. With increasing experience, percutaneous device closure for PIVSR showed widening of the indications, expansion of the accepted anatomies, and increasing success rates approaching 90% [34]. Nevertheless, patient- and device selection for percutaneous device closure of PIVSR seems highly variable among different registries, where anatomical suitability and procedural techniques remain heterogeneous without standardization.

Adequate planning and thorough defect analysis utilizing multi-modality imaging are fundamental for better outcomes. PIVSR defects are often irregular or ovoid, frequently with systole-to-diastole dynamicity in size and contour, and maybe (particularly in basal PIVSR) characterized by serpiginous intramuscular tracks. These factors make a single-phase measurement often misleading, emphasizing the value of CCT and CMR for a thorough understanding of the defect and appropriate pre-procedural planning [35]. Although some operators believe that balloon sizing can aid in the appropriate device selection for irregular defects, many others discourage it for the risk of tissue dehiscence particularly if early

Table 6 Percutaneous device closure vs. surgical repair, hospital course, and procedural outcomes

	Device Closure ($n=9$)	Surgical Repair ($n=15$)	*p*-value
Cardiogenic shock	7 (78%)	14 (93%)	0.53
Atrial fibrillation	5 (56%)	8 (53%)	> 0.99
Ventricular arrhythmia	2 (22%)	2 (13%)	0.61
High-grade heart block	1 (11%)	1 (7%)	> 0.99
Acute kidney injury	5 (56%)	10 (67%)	0.68
Dialysis	4 (44%)	8 (53%)	> 0.99
Major bleeding events	4 (44%)	5 (33%)	0.68
Stroke	0 (0%)	1 (7%)	> 0.99
Sepsis	4 (44%)	7 (47%)	> 0.99
Residual VSD	0 (0%)	4 (27%)	0.25
ICU stay (days)	25 (10–39)	24 (13–36)	> 0.99
Total hospital stay (days)	27 (10–39)	26 (24–40)	0.64
Procedural mortality	1 (11%)	0 (0%)	0.13
30-day-mortality	5 (56%)	4 (27%)	0.21
In-hospital mortality	5 (56%)	5 (33%)	0.40

Data expressed as median (25th-75th percentile) or frequency (percent) as appropriate

ICU Intensive care unit, *VSD* Ventricular septal defect

in the PIVSR course [33]. Even with adequate planning, a significant residual shunt may be encountered either immediately after device release or several days after the procedure, occasionally necessitating the implantation of an additional device [33, 34].

It seems that the Amplatzer ASD occluder device remains the most frequently used followed by the muscular VSD occluders or the dedicated post-infarction muscular VSD, however, the latter is unavailable in several sites [36]. Recently, there were some raised concerns about the rigid-made of the Amplatzer occluders (Nitinol alloy: 55% nickel and 45% titanium) with the possibility of myocardial erosion, or (less commonly) inducing hemolysis, particularly in anatomies where they got extensively deformed [33].

Similar to the majority of other registries, the most utilized device in the present study was the Amplatzer ASD occluder due to device availability, size versatility, and configuration suitability for PIVSR. However, the Konar MFO was found to be very appropriate for selected anatomies, with its ease and versatility of deployment. The Konar MFO offers the feasibility of deployment from either side (has two hubs) obviating the need for an arterio-venous loop and has demonstrated very good short- and mid-term outcomes in small defects (8–10 mm).

The reported procedural complications for percutaneous PIVSR closure varied from vascular complications, heart valve interaction/dysfunction, device embolization, stroke, arrhythmias, high-grade AV block, myocardial perforation, and cardiac tamponade. Several of these complications may be potentially fatal and often require emergency surgery. Device embolization was more likely reported in larger defects > 18–20 mm [33]. It is consistently observed that basal defects are generally associated with a lower success and a higher risk of complications [37]. In the current series, a case of device deformation was encountered, leaving a major residual defect, yet it was managed by implanting another small Amplatzer muscular VSD occluder anchored across the deformed device. Another case after successful device release developed incessant uncontainable VT/VF that led to death. Lastly, a case had acute device migration. This patient was immediately transferred to the surgical theatre and underwent device externalization and surgical repair, however, this particular patient showed a poor post-operative course and eventually passed away.

In-hospital outcomes

The in-hospital outcomes of PIVSR patients are far beyond defect repair. Despite the improving technical success of PIVSR closure, post-procedural mortality remains high and largely dictated by the poor patients' profiles, delayed presentations, usually after a massive AMI, and often frail with multiple comorbidities. The main causes for the persistently high post-procedural deaths seem not related to the closure technique but are mainly attributed to CS, sepsis, and/or multi-organ failure [29, 38].

In this series, the overall 30-day mortality was 44% (56% in the percutaneous closure and 27% in the surgical group), acknowledging the lack of randomization and potential bias in selecting patients at high-surgical risk for device closure. CS and AKI were the most commonly encountered complications, with AKI recognized as a significant predictor for 30-day mortality.

Intriguingly, a tendency of association for significant MR with improved 30-day survival was observed. To the best of our knowledge, there is no sufficient evidence explaining this association. Arguably, in patients with a large non-restrictive VSR, the pressure gradient between LV and RV may be in favor of systolic shunting across the VSR rather than regurgitating into the left atrium, hence diminishing the MR volume, in contrast to those with significant MR which probably suggest a more restrictive VSR. Another plausible explanation is that patients presenting with pulmonary edema or CS, who are known for worse prognosis, usually have markedly elevated left atrial pressures (LAP), which often diminishes MR volumes, particularly in the presence of another low-pressure vent for LV ejection. These suggested explanations await prospective hemodynamic confirmation before

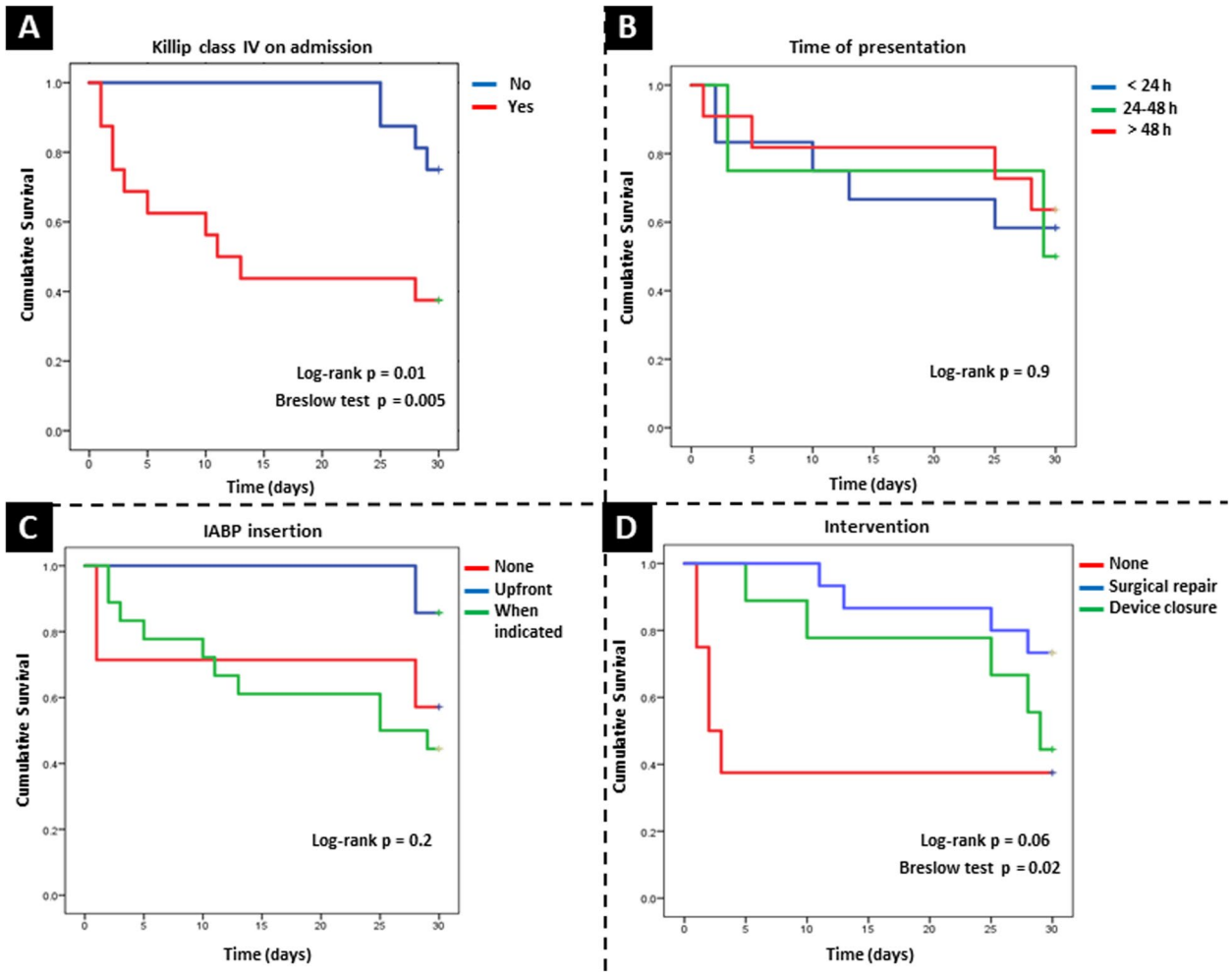


Fig. 4 Kaplan–Meier analysis curves for 30-day survival according to: **A** Killip class on admission, **B** time of presentation (< 24 h, 24–48 h, and > 48 h), **C** IABP insertion (none, upfront, or when indicated), and **(D)** intervention stratified by 3 groups (none, surgical repair, device closure). IABP, Intra-aortic balloon pump

presuming that severe MR (that indirectly refutes a massive trans-VSR shunt or a markedly elevated LAP) may be correlated with better outcomes.

Also, delayed VSR repair (independent from strategy) in this series was significantly associated with reduced mortality. Similarly, several other PIVSR registries have reported that post-procedural mortality ranges from 66% for those treated in the acute phase, to only 10% if performed in the chronic phase [9, 24, 39, 40]. However, being derived from observational non-randomized data, the perceived superiority of deferred intervention may be subjected to selection bias. It is very conceivable that patients with deteriorating hemodynamics are compelled for earlier intervention, compared to the feasibility of deferral in stable patients. Hence, the poorer outcomes encountered with early intervention may be confounded by the poorer patients' profiles [8].

Although the lack of randomized evidence makes it difficult to conclude solid recommendations concerning the appropriate time for intervention, the strategy of upfront elective MCS seems to be expanding, increasing the chances for safe deferral of intervention beyond the acute phase [17, 18, 22]. Nevertheless, MCS confers more frequent vascular complications, bleeding, and infections, necessitating appropriate selection of the candidate and the support modality, as well as tailoring the appropriate times for insertion and weaning [18].

Limitations

This registry represents the experience in the management of 32 PIVSR cases. Being single-center, outcomes might reflect the local operator's experience in both

surgical and percutaneous strategies and must be analyzed after consideration of other experiences. The retrospective, observational design and the limited case number pose limitations in deriving recommendations based on the outcomes from this series. Also, the lack of randomization in deciding the repair strategy might have allowed selection bias, which would affect the generalizability of derived observations and comparative analyses. Another drawback is having MCS experience only with IABP, where neither ECMO nor Impella were evaluated due to lack of availability. Hence, whether such superior MCS modalities would improve outcomes cannot be concluded from this series.

Conclusion

PIVSR is still associated with high short-term mortality even after successful surgical- or percutaneous device closure. A strategy of upfront MCS and deferral of PIVSR repair beyond the acute phase is likely to improve outcomes, irrespective of the repair modality. Solid evidence from multi-center randomized trials is critically needed to dictate the best management.

Abbreviations

AKI	Acute kidney injury
AMI	Acute myocardial infarction
ASD	Atrial septal defect
AV	Atrio-ventricular
BMI	Body mass index
BP	Blood pressure
CABG	Coronary artery bypass graft
CAD	Coronary artery disease
CCT	Cardiac computed tomography
CI	Confidence interval
CMR	Cardiac magnetic resonance imaging
CPB	Cardio-pulmonary bypass
CS	Cardiogenic shock
ECG	Electrocardiogram
ECMO	Extra-corporeal membrane oxygenation
EF	Ejection fraction
FMC	First medical contact
HbA1c	Glycated hemoglobin
HF	Heart failure
HR	Hazard ratio
IABP	Intra-aortic balloon bump
ICU	Intensive care unit
IQR	Inter-quartile range
IRA	Infarct-related artery
LAD	Left anterior descending
LAP	Left atrial pressure
LV	Left ventricle
LVEF	Left ventricular ejection fraction
MCS	Mechanical circulatory support
MI	Myocardial infarction
MR	Mitral regurgitation
NSTEMI	Non-ST elevation myocardial infarction
PCI	Percutaneous coronary intervention
PIVSR	Post-infarction ventricular septal rupture
PTCA	Percutaneous transluminal coronary angioplasty
RCA	Right coronary artery
RV	Right ventricle
SD	Standard deviation
STEMI	ST-elevation myocardial infarction
TEE	Trans-esophageal echocardiography
TIMI	Thrombolysis in myocardial infarction
TTE	Trans-thoracic echocardiography
VF	Ventricular fibrillation
VSD	Ventricular septal defect
VSR	Ventricular septal rupture
VT	Ventricular tachycardia

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12872-024-04370-4.

Supplementary Material 1

Acknowledgements
Not applicable.

Authors' contributions
H. Hussein, Data entry, tabulating results, designing figures, reviewing literature and drafting the manuscript. S. Eltayeb, Data entry and revising the manuscript. E. Mosaad, Reviewing literature and contributing to the manuscript writing. M. Shehata, Operator of percutaneous device closure procedures, reviewing literature and contributing to the manuscript writing. A. Elafifi, Operator of percutaneous device closure procedures and revising the manuscript. H. Hosny, Leader of multi-disciplinary team discussion for PIVSR cases, primary operator of surgical repair procedures and revising the manuscript. A. Samir, Statistical analysis, reviewing literature, writing the manuscript and revision.

Funding
This work didn't receive any specific grant from funding agencies.

Data availability
The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

Declarations

Ethics approval and consent to participate
The registry protocol and methodology were thoroughly reviewed and approved by Aswan Heart Centre Research Ethics Committee (AHC-REC). Due to the complete retrospective nature of the study, and assessment of completely anonymized data without any breach of patients' confidentiality, the need to have informed signed consent from participants was waived by AHC-REC (approval number: 20230728MYFAHC_VSRReg20230803, email: ahcrec@aswanheartcentre.com).

Consent for publication
Due to the complete retrospective nature of the study, and assessment of completely anonymized data without any breach of patients' confidentiality, the need to have informed signed consent from participants was waived by AHC-REC (approval number: 20230728MYFAHC_VSRReg20230803, email: ahcrec@aswanheartcentre.com).

Competing interests
The authors declare no competing interests.

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Received: 2 March 2024 Accepted: 22 November 2024
Published online: 12 March 2025

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