Percutaneous Closure of Post-Infarction Ventricular Septal Rupture: A Review of Patient Selection, Techniques and Outcomes

.

ABSTRACT

|  |
| --- |
| Post-infarction ventricular septal rupture (PIVSR) represents one of the most lethal complications of acute myocardial infarction. This narrative review examines current evidence supporting percutaneous closure as an alternative to surgical repair.  Optimal patient selection depends on defect characteristics, hemodynamic stability, and surgical risk. Simple, apical defects smaller than 24 mm with adequate tissue rims favor percutaneous approaches, while large (>35 mm), complex, or multiple defects typically require surgery. Timing critically influences outcomes, delaying intervention 2-4 weeks post rupture when tissue has stabilized consistently improves survival compared to early closure.  Contemporary series report 30-day mortality rates of 15-30% for percutaneous closure in selected patients versus 40-60% for emergency surgery. Device selection must match septal anatomy, with dedicated post-infarction VSD occluders preferred over adapted congenital devices. Technical success rates reach 70-100%, though residual shunting remains common.  Current evidence supports percutaneous closure as a viable option that reduces procedural morbidity and enables staged management in high-risk patients. Success requires multidisciplinary planning, comprehensive imaging assessment, and surgical backup availability. While mortality remains high due to underlying myocardial damage, percutaneous approaches offer meaningful benefits for carefully selected patients when optimal timing and techniques are employed. |

*Keywords: Ventricular Septal Rupture, Percutaneous Closure, Myocardial infarction complication, Device Selection, Outcomes*

1. INTRODUCTION

Post-infarction ventricular septal rupture (PIVSR) represents one of the most devastating mechanical complications following acute myocardial infarction (AMI) (Hussein et al., 2025). Characterized by a tear in the interventricular septum secondary to myocardial necrosis, PIVSR leads to an acute left-to-right shunt, precipitating hemodynamic collapse, cardiogenic shock (CS), and multi-organ failure (Jones et al., 2014). The advent of rapid reperfusion strategies, particularly primary percutaneous coronary intervention (PCI), has dramatically reduced the incidence of PIVSR from 1-3% in the pre-thrombolytic era to approximately 0.17-0.34% in contemporary practice (David, 2022). Optimal and timely reperfusion salvages myocardium, limits infarct expansion, and reduces the likelihood of transmural necrosis required for septal rupture.Conversely, delayed or absent reperfusion remains a significant risk factor. Despite this decreased incidence, the mortality associated with PIVSR remains high, often exceeding 40%, even when definitive closure is attempted, and this figure has shown little improvement over the past few decades. This persistent high mortality, despite the condition becoming rarer, underscores that while primary prevention through effective reperfusion is impactful, the management of PIVSR once it occurs remains a formidable clinical challenge, suggesting that the inherent lethality of the rupture and the difficulties in achieving timely and effective repair are now the primary drivers of poor outcomes (Wilson & Horlick, 2016).

Historically, the prognosis for PIVSR patients managed solely with medical therapy was dismal, with mortality rates exceeding 90% within weeks to months (Jones et al., 2014). Surgical repair emerged as the definitive treat ment and standard of care.Early surgical techniques involved approaches through the right ventricle, but these offered limited exposure. Subsequently, trans-infarct left ventriculotomy approaches became favored, allowing better visualization and repair, often involving infarctectomy or infarct exclusion techniques (e.g., Daggett, David repairs) using patch materials (David, 2022). Despite technical refinements, surgical mortality, particularly when performed early after VSR diagnosis, remains substantial, frequently reported between 30% and 50% or higher, and has not demonstrably improved over the last two decades (Hussein et al., 2025).

The limitations and high risks associated with surgery, especially in acutely ill patients with friable myocardial tissue, spurred the development of less invasive alternatives. Percutaneous, transcatheter device closure of PIVSR was first reported in the late 1980s and has since evolved as a viable option. Initially, percutaneous closure was primarily reserved for managing residual shunts after surgical repair or for treating chronic, stable defects (Wilson & Horlick, 2016). However, with accumulating experience and technological advancements in devices and delivery systems, its application has expanded. It is now increasingly considered as a primary therapy for acute PIVSR, particularly in patients deemed to be at high or prohibitive risk for surgery, or as a bridge to stabilize patients before potential surgical intervention (Hussein et al., 2025).

Despite these advancements, the management of PIVSR remains challenging. The complexity of the patient population, often presenting with cardiogenic shock and significant comorbidities, contributes to the persistently high mortality rates. Furthermore, significant controversy persists regarding the optimal timing of intervention, balancing the risks of early repair on friable tissue against the dangers of delaying closure in unstable patients. A critical limitation in guiding clinical practice is the lack of high-level evidence, particularly the absence of randomized controlled trials comparing surgical and percutaneous approaches or different timing strategies (Hussein et al., 2025). This review aims to synthesize the current evidence on percutaneous PIVSR closure, focusing on patient selection, procedural techniques, device considerations, outcomes, and future directions in this challenging field

**1.1 Literature Search Strategy**

A comprehensive literature search was conducted using PubMed/MEDLINE, Embase, and Google Scholar databases from January 1990 to December 2024. Search terms included combinations of: "post-infarction ventricular septal rupture," "PIVSR," "percutaneous closure," "transcatheter repair," "Amplatzer occluder," and "device closure" using Boolean operators (AND, OR). We included peer-reviewed articles in English reporting on percutaneous PIVSR closure techniques, patient selection criteria, procedural outcomes, and device considerations. Case reports with fewer than 5 patients, pediatric congenital septal defects, and non-human studies were excluded. Reference lists of identified articles were manually reviewed for additional relevant sources. Priority was given to recent systematic reviews, multicenter registries, and comparative studies when available.

2. Pathophysiology and Natural History of PIVSR

**2.1 Mechanism of Rupture**

Ventricular septal rupture following AMI is fundamentally a consequence of myocardial necrosis.It almost invariably occurs in the setting of a transmural infarction, typically affecting patients experiencing their first MI, often with complete occlusion of a single coronary artery and poor collateral circulation (David, 2022). Risk factors include advanced age, female gender, hypertension, chronic kidney disease, and delayed or absent reperfusion therapy (Cadogan et al., 2023). Furthermore, a study focusing on risk factors identified active smoking as having a significant correlation with the development of PIVSR (Ali et al., 2022)

The rupture typically occurs at the border zone between infarcted and non-infarcted myocardium. The location is dictated by the territory of the infarct-related artery: anterior MIs usually involving the left anterior descending artery tend to cause apical septal ruptures, while inferior MIs right coronary or circumflex artery typically result in basal infero-posterior septal defects (Matteucci et al., 2021). Apical PIVSRs are often described as simple, direct communications, whereas basal or posterior PIVSRs are in one case described as more complex, exhibiting serpiginous tracts, irregular shapes, multiple fenestrations, and potential involvement of the right ventricular free wall or papillary muscles (Morton et al., 2023).

Becker and colleagues established a classification system for free wall rupture based on three distinct morphological types (Figure 1). Type 1 is an abrupt, slit-like tear in the myocardium, typically occurring in the acute phase of a myocardial infarction, within 24 hours. Type 2 involves a more slowly progressing tear, characterized by an area of myocardial erosion. Type 3, which occurs late in the course of an MI (after 7 days), presents as a perforation within a markedly thinned, aneurysmal section of the myocardium. This pathological classification is also applicable to ventricular septal rupture.

A diagram of different types of rupture

AI-generated content may be incorrect.

**Figure 1. Morphological classification of myocardial rupture**

Classification system demonstrating three distinct types of cardiac rupture morphology. Type 1: Abrupt slit-like tear occurring within 24 hours. Type 2: Progressive erosive tear. Type 3: Late perforation within aneurysmal tissue (>7 days). This classification applies to both free wall and septal ruptures (Modified from Reynolds & Hochman, 2010).

The timing of rupture often follows a bimodal pattern. Early rupture, within the first 24 hours post-MI, may result from dissection through tissue planes by intramural hematoma or hemorrhage, potentially accelerated by thrombolysis.The more common presentation occurs 3-5 days post-MI, coinciding with the phase of coagulation necrosis, marked inflammation, and enzymatic degradation of the myocardial extracellular matrix (Morton et al., 2023).This degradation process is mediated significantly by matrix metalloproteinases (MMPs), particularly the gelatinases (MMP-2, MMP-9) and collagenases (MMP-1, MMP-8, MMP-13), which are upregulated by inflammatory cells infiltrating the infarct zone (DeLeon-Pennell et al., 2017). MMP-9 activity peaks early around days 1-4, coinciding with neutrophil infiltration, while MMP-2 activity rises later, peaking around day 7 alongside macrophage infiltration (Tao et al., 2004).This enzymatic breakdown of structural proteins, primarily type I collagen, weakens the necrotic septal tissue, rendering it friable and unable to withstand the interventricular pressure gradient, ultimately leading to rupture.The timing of peak MMP activity and collagen degradation directly correlates with the period of maximum tissue fragility, explaining the difficulty and high risk associated with early repair attempts.Over subsequent weeks, the inflammatory phase subsides, MMP activity decreases, and reparative processes involving myofibroblasts lead to collagen deposition and scar formation, gradually increasing tissue strength (DeLeon-Pennell et al., 2017).

**2.2 Hemodynamic Consequences and The natural history of PIVSR without definitive closure**

The rupture creates an abrupt communication between the high-pressure left ventricle (LV) and the low-pressure right ventricle (RV), resulting in a significant left-to-right shunt. The magnitude of this shunt, often quantified by the pulmonary-to-systemic flow ratio (Qp/Qs), depends primarily on the size of the defect and the pressure gradient across the septum, which is influenced by the relative resistances of the pulmonary and systemic vascular circuits (Gong et al., 2021).

The acute left-to-right shunt creates severe biventricular volume overload. Right ventricular dilation and dysfunction develop alongside increased pulmonary pressures and edema, while enhanced pulmonary venous return further compromises already impaired left ventricular function.(Tripathi et al., 2023). Systemic cardiac output is reduced due to the shunting of blood into the pulmonary circulation. The clinical presentation ranges from a new loud holosystolic murmur heard best at the lower left sternal border often accompanied by a thrill in relatively stable patients, to rapid hemodynamic deterioration with biventricular failure and profound cardiogenic shock. In severe shock, the murmur may become faint or inaudible (Asai et al., 2023). The presence and severity of RV dysfunction, particularly common with inferior MI and posterior VSR, is a critical determinant of hemodynamic status and overall prognosis (Matteucci et al., 2021).

The natural history of PIVSR without definitive closure either surgical or percutaneous is poor (Cubeddu et al., 2024). Medical management alone is considered futile for long-term survival. A retrospective observational trial starkly highlighted this, reporting that mortality for patients managed with medical therapy alone was 100% (Zhang et al., 2021). Mortality rates climb rapidly after diagnosis, approximately 24% die within 72 hours, 46-50% within the first week, 67-82% within two months, and over 90% fail to survive long-term (Wilson & Horlick, 2016). While rare cases of survival up to one year without intervention have been reported, likely involving very small defects with minimal hemodynamic impact, these are exceptions (Birnbaum et al., 2002).

3. Patient Selection for Percutaneous Management

The decision to chose percutaneous closure of PIVSR as definitive management is complex, requiring careful consideration of patient and defect characteristics, often within the framework of a multidisciplinary Heart Team discussion. Percutaneous closure is most frequently considered for patients deemed to be at high or prohibitive risk for conventional surgical repair, or as a salvage strategy for residual leaks following surgery (Hussein et al., 2025).

**3.1 Clinical Presentation And Hemodynamic Stability**

The timing of PIVSR presentation significantly influences management strategy and prognosis. Patients presenting acutely (<2 weeks post-MI) are often hemodynamically unstable, frequently in cardiogenic shock (Hussein et al., 2025). While percutaneous closure can be attempted in these critically ill patients, often as a bridge to recovery or surgery, it is associated with very high procedural risks and mortality rates. The presence of cardiogenic shock itself is a major predictor of poor outcome, irrespective of the closure method (Wilson & Horlick, 2016).

Patients presenting in the subacute phase (typically >14 days or >2-3 weeks post-MI) may have achieved some degree of hemodynamic stability, either spontaneously or with medical/mechanical support.In this phase, the myocardial tissue surrounding the defect tends to be less friable and more organized, potentially allowing for more secure device anchoring and improved procedural outcomes (Morton et al., 2023). Chronic PIVSR presentations at >4 weeks or months post-MI typically involve patients with residual shunts after prior surgical repair or those with smaller, hemodynamically tolerated defects who survived the acute phase without intervention. Percutaneous closure is often highly successful in this subset (Wilson & Horlick, 2016).

The use of mechanical circulatory support (MCS), such as intra-aortic balloon pumps (IABP), veno-arterial extracorporeal membrane oxygenation (VA-ECMO), or percutaneous ventricular assist devices (Impella, TandemHeart), may have an important role in stabilizing patients, particularly those in CS.MCS can reduce LV afterload, decrease the left-to-right shunt, improve systemic perfusion, preserve end-organ function, and potentially allow for delayed, safer intervention.Upfront IABP insertion, even in the absence of shock, has shown a trend towards improved survival in some series (Hussein et al., 2025).

**3.2 PIVSR Size, Location, Morphology Myocardial and Tissue Characteristics**

Anatomical characteristics of the PIVSR are important in determining suitability for percutaneous closure. Echocardiography (TTE and TEE) is the primary tool for initial diagnosis and sizing PIVSR. However, 2D echocardiography may underestimate the true defect size, especially in complex ruptures, as the beam may not be coaxial with the defect. 3D TEE and increasingly cardiac computed tomography (CT) offer more comprehensive anatomical assessment, including defect shape, dimensions, rim adequacy, and relationship to surrounding structures (Cadogan et al., 2023). Generally, defects smaller than 15 mm are considered more favorable for percutaneous closure. Larger defects greater than 15 mm are associated with increased risk of device embolization, residual shunt, and mortality (L. Wang et al., 2021). In one review (Cadogan et al., 2023) the factor that favours percutaneous PIVSR closure over surgery are are single or simple defect, defect size <24 mm, sufficient rim margins and Adequate distance from valve apparatuses (table 1)

|  |  |
| --- | --- |
| Favours Percutaneous Closure | Favours Surgical Closure |
| Single/simple defect | Complex/multiple defects |
| Defect size <24 mm | Defect size >35 mm |
| Sufficient rim margins | Concomitant surgical revascularisation |
| Adequate distance from valve apparatuses | Concomitant valvular heart surgery |
| Previous unsuccessful surgical attempt | Previous unsuccessful percutaneous attempt |

**Table 1. Factors Influencing Closure Decisions on PIVSR**

Apical PIVSRs, typically resulting from anterior MIs, are often anatomically simpler and further from valvular structures, making them generally more amenable to percutaneous closure with better reported outcomes (Cadogan et al., 2023). Basal or posterior PIVSRs, associated with inferior MIs, pose greater challenges. These defects are often located near the mitral and tricuspid valves, increasing the risk of device interference with valve function. Furthermore, they frequently lack adequate septal rims, particularly inferiorly and posteriorly, compromising secure device anchoring (Anbalakan et al., 2023).

The morphology of PIVSR is rarely a simple. Many defects are complex, irregular, and serpiginous, with separate entrance and exit points on the LV and RV sides of the septum. This complex anatomy makes complete sealing with a device difficult, often resulting in residual shunts (Anbalakan et al., 2023). Key anatomic patterns that favour percutaneous closure are single, well-circumscribed defect surrounded by reasonably firm, fibrotic tissue allows the retention discs of an occluder to anchor securely and adequate rim thickness (about 5–7 mm) and an eccentricity index (largest to smallest diameter) <1.5 provide a more circular profile that matches currently available devices and minimises residual gaps (Cadogan et al., 2023).

The quality of the myocardial tissue surrounding the VSR is a critical factor, particularly when considering the timing and feasibility of closure, especially percutaneous closure which relies on tissue anchoring. In the acute phase first 1-2 weeks, the infarct border zone is characterized by necrotic, edematous, and friable tissue due to inflammation and enzymatic degradation by MMPs. This friability makes secure suture placement challenging for surgeons and reliable device anchoring difficult for percutaneous approaches.9 Attempts at closure on such tissue carry a high risk of suture dehiscence, patch leaks, device malposition, embolization, or further tearing and enlargement of the defect (Wilson & Horlick, 2016). Consequently, acutely friable, necrotic tissue is often considered a relative or absolute contraindication to early percutaneous closure, favoring delayed intervention if hemodynamically feasible (Ahmed et al., 2008).

TTE and TEE plays a central role in assessing tissue characteristics, visualizing the defect margins, identifying associated aneurysms, and evaluating the thickness and integrity of the surrounding septum (Morton et al., 2023). While standard echocardiography provides valuable information, advanced imaging may offer more detailed tissue characterization. Cardiac CT can delineate septal thickness and morphology with high spatial resolution, aiding in the assessment of rim adequacy (Cadogan et al., 2023). Cardiac MRI, particularly with T1 and T2 mapping sequences, holds promise for quantifying myocardial edema, necrosis, and fibrosis, potentially offering a non-invasive way to assess tissue viability and maturation (Kim et al., 2017). However, the clinical application of these advanced MRI techniques specifically for PIVSR closure planning is not yet widely established, and their use is often limited in acutely unstable patients. The presence of adequate, firmer tissue rims around the defect is essential for the successful and stable implantation of percutaneous closure devices (Cadogan et al., 2023).

**3.3 Surgical Risk Assessment And Comorbidities**

Percutaneous closure is often the preferred strategy for patients considered high-risk for conventional surgery (Hussein et al., 2025). Surgical risk is assessed using clinical judgment, evaluation of comorbidities, and established risk stratification tools like the EuroSCORE II and the Society of Thoracic Surgeons (STS) Predicted Risk of Mortality score with Higher scores are generally associated with increased mortality (Faccini & Butera, 2019). Significant comorbidities heavily influence both surgical risk and overall prognosis. Advanced age, pre-existing renal failure especially requiring dialysis, severe lung disease, prior stroke, diabetes, poor LV function, and significant RV dysfunction are all associated with worse outcomes following PIVSR, regardless of the repair strategy. Acute kidney injury developing post-VSR is a particularly strong predictor of 30-day mortality. (Hussein et al., 2025). This is supported by surgical series, where cardiogenic shock on admission was found to be an independent predictor of mortality following surgical repair (Isik et al., 2021).

The selection of appropriate candidates for percutaneous PIVSR closure demands a holistic evaluation framework that incorporates comprehensive clinical assessment, hemodynamic parameters, perioperative risk stratification, and precise anatomical characterization of the septal defect, encompassing geometric dimensions, anatomical positioning, structural morphology, and perilesional tissue viability. Given the heterogeneity of patient presentations and defect characteristics, optimal treatment decisions require individualized risk-benefit analysis through collaborative multidisciplinary Heart Team deliberation.

4. Timing of Intervention

The optimal timing for PIVSR closure remains one of the most debated and challenging aspects of management. This challenges reflects the difficult balance between the immediate hemodynamic benefit of shunt closure and the increased procedural risks associated with closure on acutely inflamed and friable tissues (Hussein et al., 2025)

**4.1 Early vs. Delayed Closure**

The primary rationale for early intervention <1-2 weeks post PISVR is to promptly eliminate the detrimental left-to-right shunt, thereby improving systemic perfusion, reducing pulmonary overcirculation, and preventing irreversible end-organ damage and hemodynamic collapse. Proponents of immediate intervention argue that delaying closure for hemodynamically unstable, or even seemingly stable, patients can precipitate sudden deterioration and multi-organ failure, potentially making a subsequent repair futile (David, 2022).

The major argument for delayed closure > 2-4 weeks PIVSR comes from the nature of the infarcted myocardium. In the first 1-2 weeks, the septal tissue surrounding the rupture is necrotic, inflamed, and extremely friable.Attempting surgical patch suture or percutaneous device anchoring in this phase is technically demanding and associated with a high incidence of residual leaks, patch or device dehiscence, or further tissue tearing (Wilson & Horlick, 2016). Delaying intervention allows time for the inflammatory process to subside and for scar tissue to form around the defect margins. This fibrotic tissue provides a firmer substrate for surgical sutures or device anchors, theoretically leading to more durable repairs and lower rates of residual shunting (Morton et al., 2023).

Numerous observational studies and registries consistently report significantly lower mortality rates for patients undergoing delayed repair typically >7 days to >3-4 weeks post-MI or PIVSR diagnosis compared to those undergoing early repair (Hussein et al., 2025). However, this association is heavily confounded by survival bias; patients who survive long enough to undergo delayed repair inherently represent a lower-risk cohort who were stable enough to wait (Wilson & Horlick, 2016). A European survei study indicated a preference for delayed repair among participating centers (Ronco et al., 2023).

**4.2 Defining the Optimal Timing Window**

Identifying a definitive optimal timing window for percutaneous closure is difficult due to the lack of randomized data and the influence of patient stability and survival bias. However, analysis of observational data provides some insight presented in Table 2 (modified from Shafiei et al., 2020).

**Table 2. Analysis of observational data of the Optimal Timing Window (modified from Shafiei et al., 2020).**

|  |  |  |  |
| --- | --- | --- | --- |
| Study & Year | Intervention Type | Timing Category | Mortality Rate (%) |
| Killen et al. (1997) | Surgery | <24 hours | 51.4 |
| 1-5 days | 47.1 |
| 6-20 days | 28.6 |
| >1 month | 0 |
| Di Summa et al. (1997) | Surgery | Very Early (~1 day) | 87.5 |
| <1 week | 44.4 |
| Later | 0 |
| Bouchart et al. (1998) | Surgery | Less than 1 week | 33% |
| More than 1 week | 6.2% |
| Dalrymple-Hay et al. (1998) | Surgery | Day 1 | 73 |
| >2 days | 16 |
| >4 weeks | 0 |
| Cerin et al. (2003) | Surgery | Within the first week | 75% |
| After 3 weeks | 16% |
| Mantovani et al. (2006) | Surgery | <3 days post-VSR | 52 |
| >3 days post-VSR | 11 |
| Coskun et al, 2009 | Surgery | Within 3 days | 100 |
| After 36 days | 0 |
| Arnaoutakis et al. (2012) | Surgery | < 7 days post-MI | 54.1 |
| >7 days post-MI | 18.4 |
| Papalexopoulou et al. (2013) | Surgery | from >3 days to within 4 weeks | 52.4 |
| from 1 week to after 4 weeks | 7.56 |
| Trivedi et al. (2015) | Percutaneous closure Surgery | <21 days | Significantly more for Percutaneous closure |
| >21 days |
| Cinq-Mars et al. (2016) | Surgery | 2.74 days | Not Survived |
| 4.44 days | Survived |
| Malhotra et al. (2017) | Surgery | <3 days | 76 |
| >3 days | 26 |

The data consistently demonstrate a survival advantage with delayed closure, typically beyond the first week and ideally after 2-4 weeks, allowing for tissue maturation (Hussein et al., 2025).

For percutaneous closure, data from (Wilson & Horlick, 2016) showed that interventions performed 1 to 13 days after PISVR has highest in-hospital mortality rates ranging from 42% to 86% and closure done at 27 days to 15 weeks has lower in-hospital mortality from 18% to 28%. Therefore, if hemodynamically permissible often requiring MCS bridging, delaying percutaneous closure until the subacute phase beyond 2-3 weeks seems prudent to optimize outcomes (Hussein et al., 2025).

**5. Device Selection for Percutaneous PIVSR Closure**

The selection of an appropriate closure device is important, yet challenging, given the complex and variable morphology of these defects. PIVSR are typically irregular, may have serpiginous tracts, and often lack well-defined, firm rims. This section details the spectrum of devices used for percutaneous PIVSR closure, compares their characteristics, and outlines the methodologies employed for device sizing. The historical evolution from early devices to contemporary options reflects an ongoing search for an optimal solution, frequently necessitating the off-label use of devices designed for other intracardiac defects due to the lack of a universally ideal VSR-specific occluder (Risseeuw et al., 2014).

**5.1 Primary Occluder Devices**

**5.1.1 Amplatzer Post-Infarction Muscular VSD (PIMVSD) Occluder**

The Amplatzer PIMVSD Occluder (Abbott Vascular) was specifically designed for PIVSR, featuring a wider waist and larger retention discs than standard muscular VSD devices to better accommodate thickened, friable septal tissue. This design aims to span the often thickened or necrotic septal wall and provide a larger surface area for anchoring in fragile tissue. Its use has been documented in several registries and case series (Risseeuw et al., 2014). An early US registry reported successful deployment in 16 out of 18 patients, establishing feasibility, although accompanied by a significant 30-day mortality of 28%.A more recent post-approval multicenter study (2011-2021) involving 131 patients confirmed its continued role as a therapeutic alternative for patients considered poor candidates for surgical repair. However, this study also underscored the persistent high morbidity associated with the condition, reporting a technical success rate of 76.8% and 6-month survival rates between 37.2% and 46.4% across different patient cohorts analyzed (Holzer et al., 2004).

**5.1.2 Amplatzer Muscular VSD (mVSD) Occluder**

The standard Amplatzer mVSD occluders have also been used for PIVSR closure, particularly when the dedicated PIMVSR device is unavailable or if the defect morphology is deemed suitable.Case reports describe the successful implantation of 18 mm and 16 mm mVSD devices (Anbalakan et al., 2023).The selection depends critically on the specific defect anatomy, including size, shape, and the presence of adequate tissue rims for stable device anchoring.

**5.2 Alternative and Less Common Devices**

**5.2.1 Amplatzer Atrial Septal Occluder (ASO)**

The ASO devices, designed for atrial septal defect closure, have been utilized off-label for PIVSR closure (Risseeuw et al., 2014). This typically occurs when specialized ventricular septal rupture equipment is not available or in particular circumstances such as recurring PIVSR following surgical intervention. Its use can be attributed to its availability in a broad range of sizes, familiarity among interventional cardiologists due to its common application in congenital heart disease, and its relatively straightforward deployment mechanism. The ASO consists of two self-expanding nitinol mesh discs connected by a short, narrow waist. The polyester fabric within the discs promotes thrombosis and tissue ingrowth, aiming for complete closure of the defect over time (Aggarwal et al., 2018). In the context of VSR, operators often select an ASO device with a waist diameter significantly larger than the measured defect size to ensure adequate coverage and stability, given the often irregular and friable nature of the infarcted septal tissue. For example, one research study documented the use of ASO devices sized between 10 mm and 30 mm for ventricular septal ruptures that had mean diameters of 20.8 ± 6.9 mm, employing a sizing approach that selected devices 6-8 mm larger than the actual defect dimensions (Aggarwal et al., 2018) (Islam et al., 2025).

However, the use of ASO devices for VSR closure presents several inherent limitations. The shorter waist of the ASO, designed for the typically thinner atrial septum, may not adequately span the often thicker and more complex ventricular septum, particularly in the setting of acute infarction and tissue edema. This can lead to device deformation, improper apposition of the discs against the septal walls, and consequently, a higher incidence of residual shunts , . The fabric in ASO devices is also designed to withstand lower transatrial pressures, which might contribute to increased permeability and persistent shunting when subjected to the higher pressures of the ventricular system (Anbalakan et al., 2023). A retrospective study involving 21 patients undergoing VSR closure with ASO devices (predominantly Amplatzer ASO) reported a residual defect in 62% of patients, although the clinical significance of these residual shunts varied . Device embolization, though less common, has also been reported with ASO use in VSR closure, necessitating careful device selection and deployment techniques (Aggarwal et al., 2018). Despite these limitations, the ASO continues to serve as an important therapeutic option, especially in settings with limited resources or when specialized PIVSR devices are not accessible, providing a potentially life-saving treatment for patients who are at high surgical risk (Islam et al., 2025; Premchand et al., 2017).

**5.2.2 Konar Multifunctional Occluder (MFO)**

The Konar MFO, manufactured by Lifetech Scientific, is a relatively newer device that has shown promise for percutaneous closure of various septal defects, including PIVSR (Hussein et al., 2025; Islam et al., 2025). This device is characterized by its low profile, flexibility, and the unique feature of being deliverable from either the arterial or venous side, as it possesses two hubs (Hussein et al., 2025). The MFO is a self-expanding nitinol device with two discs connected by a waist, and it incorporates a polytetrafluoroethylene membrane to promote occlusion. Its design aims to minimize radial force on the surrounding tissue, which can be particularly advantageous when dealing with the friable and necrotic myocardium characteristic of PIVSR (Álvarez-Fuente et al., 2022).

Several case reports and small series have highlighted the utility of the Konar MFO in challenging VSR anatomies. For instance, it has been found to be particularly appropriate for VSRs with a serpiginous course or those located in complex positions where its flexibility and low profile offer an advantage over more rigid devices. In one series, the MFO was used in three cases, with successful closure in two and one device embolized, likely due to the serpiginous nature of the VSR track, but was successfully retrieved percutaneously. This case series suggested that the MFO's low radial force makes it well-suited for serpiginous VSRs, minimizing the risk of necrotic tissue damage (Islam et al., 2025). The ability to deploy the MFO from either side antegrade or retrograde can also simplify the procedure and obviate the need for an arteriovenous loop in certain situations, potentially reducing procedural time and complexity (Hussein et al., 2025).

**6. Device Sizing for Percutaneous VSR Closure**

**6.1 Principles of Device Sizing**

The principles guiding device sizing for percutaneous closure of PIVSR are critical for achieving successful and durable defect closure while minimizing complications. A fundamental aspect is the accurate assessment of the VSR's dimensions and morphology, which is often complex due to the irregular, serpiginous, or multi-channeled nature of these defects within necrotic tissue. The selected occluder device must be large enough to provide adequate coverage of the defect, ensuring that the discs can anchor securely on stable septal tissue on either side of the rupture (Chen et al., 2023; Hussein et al., 2025).

The sizing procedure must also account for the specific features of the selected occluder device. For example, devices featuring a defined waist, such as the Amplatzer Muscular VSD Occluder or the specialized PI Muscular VSD Occluder, are dimensioned according to the diameter of this waist component, which is designed to sit within the defect opening (Anbalakan et al., 2023). The diameter of the retention discs is also a crucial factor, as they must be sufficiently large to overlap the defect margins and provide stable apposition against the septal walls without encroaching on adjacent cardiac structures such as valve leaflets or chordae tendineae. The thickness of the ventricular septum at the margins of the VSR can also influence device selection and sizing, particularly the required length of the connecting waist. An undersized device risks embolization, residual shunt, or failure to adequately stabilize the ruptured septum. Conversely, an excessively oversized device can distort cardiac anatomy, impair valve function, or even cause mechanical trauma to the already compromised myocardial tissue, potentially leading to arrhythmias or erosion . Therefore, a balance must be struck between achieving secure closure and avoiding device-related complications (Cadogan et al., 2023).

The reasoning behind oversizing involves multiple factors. It compensates for the changing nature of the defect, which may expand due to continued tissue death or the mechanical stress from ventricular contractions, it helps establish secure anchoring in tissue that is often fragile and it seeks to minimize the risk of substantial residual blood flow across the defect (Chen et al., 2023). Furthermore, oversizing helps to compensate for potential inaccuracies in defect measurement and anticipates further enlargement of the VSR that might occur post-procedure due to ongoing tissue remodeling or stress. The goal is to achieve complete coverage of the defect margins and promote effective sealing (Islam et al., 2025).

The degree of oversizing varies depending on the type of device used, the specific anatomy of the VSR, and institutional or operator preference. For Amplatzer ASO used in VSR, an oversizing of 6-8 mm or even more relative to the largest defect diameter is common (Aggarwal et al., 2018). For instance, one study reported using ASO devices 8-14 mm larger than the VSR diameter with good results in reducing residual shunts (Chen et al., 2023). When using dedicated Amplatzer PIMVSD Occluders, the oversizing might be slightly less aggressive but still substantial, for example, 4 mm more than the defect size. or the Konar Multifunctional Occluder (MFO), an oversizing of 2 mm has been reported (Islam et al., 2025). While oversizing is beneficial for stability and closure, it must be balanced against the risk of complications such as device impingement on adjacent structures or interference with valve function. Excessive oversizing can also lead to device deformation or further tissue damage in the already compromised myocardium.

**6.2 Role of Pre-procedural Imaging in Sizing**

Pre-procedural imaging plays an indispensable role in accurate device sizing for percutaneous PIVSR closure. Transthoracic (TTE) and transesophageal (TEE) echocardiography are fundamental tools for initial diagnosis and assessment. TEE offers superior image quality and is indispensable for intraprocedural guidance, allowing detailed visualization of the defect's size, shape, rims, relationship to adjacent structures, and real-time monitoring of device deployment and residual shunt assessment. Three-dimensional TEE can provide even more accurate morphological detail regarding defect shape and size (Cadogan et al., 2023).

Cardiac computed tomography (CCT) can also be invaluable, particularly for complex VSRs, as it offers detailed anatomical information, including the extent of myocardial necrosis, the presence of serpiginous tracts, and the 3D spatial relationships of the defect (Hussein et al., 2025). Similar to CCT, cardiac Magnetic Resonance Imaging (MRI) can offer accurate assessment of VSR location, size, and anatomy, potentially guiding repair strategies. It can also provide valuable information about surrounding myocardial tissue characteristics. Its utility may be limited by patient instability and longer acquisition times. MRI has been shown feasible even in the presence of recently implanted coronary stents (Zhong et al., 2019).

**7. Percutaneous PIVSR Closure Technique**

**7.1 Patient Preparation and Anesthesia**

Given the complexity and duration of the procedure, as well as the need for optimal imaging and patient stability, percutaneous PIVSR closure is typically performed under general anesthesia (Anbalakan et al., 2023). This allows for controlled ventilation, patient immobility crucial for precise device manipulation, and facilitates TEE guidance. Many patients presenting with post-MI VSR are hemodynamically compromised, often in cardiogenic shock (Risseeuw et al., 2014). Therefore, pre-procedural and intraprocedural hemodynamic support is frequently necessary. This may involve inotropic or vasopressor infusions and the use of MCS devices such as IABP. In some centers, VA-ECMO may be initiated pre-procedure or kept on standby, particularly for patients in profound shock (Ishiyama et al., 2020).

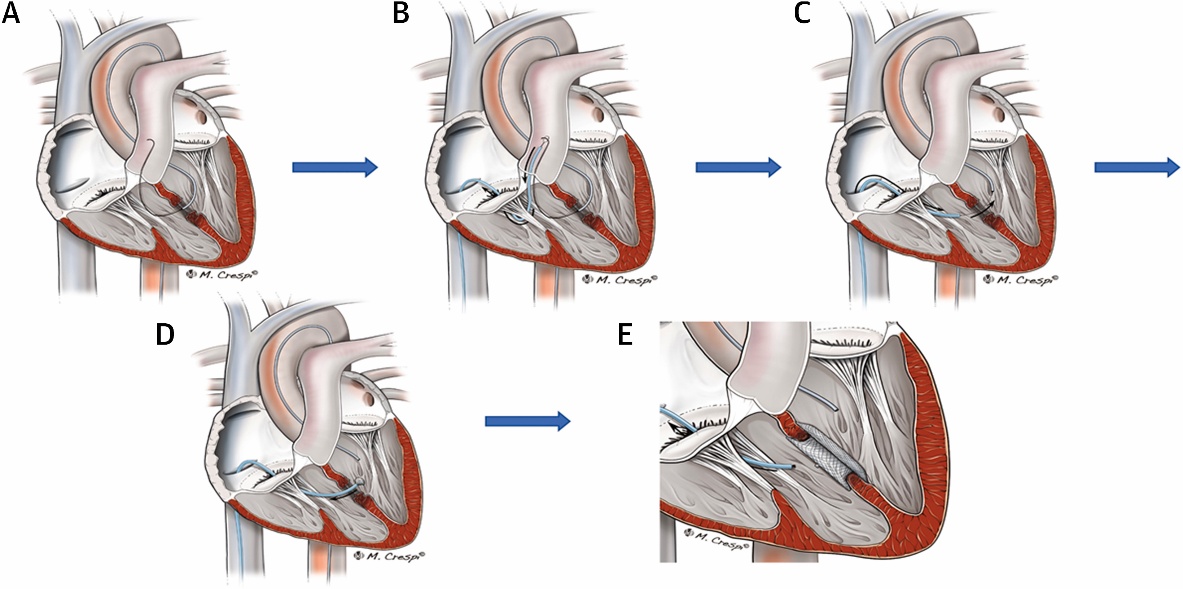
**7.1 Vascular Access Strategies and Crossing Approach**

Establishing secure vascular access is the first invasive step. The standard technique involves obtaining both arterial and venous access to facilitate the creation of an arteriovenous (AV) wire loop, which provides essential support for device delivery. Arterial access is typically gained via the femoral artery, Venous access can be achieved through the femoral vein or the internal jugular vein (IJV) (Oman et al., 2020). The specific choice of venous access site may be influenced by operator preference, patient anatomy, and the planned route for advancing the delivery system.

Different techniques have been reported in percutaneous PIVSR closure, including retrograde, antegrade, transseptal, and hybrid periventricular approaches.

**7.1.1 Retrograde Approach**

In the retrograde approach, transcatheter closure is carried out after establishing arterial access through the femoral artery with retrograde passage across the aortic valve. The VSR can be accessed using a 5- to 6-F Judkins right coronary catheter along with a hydrophilic wire to cross the defect into the right ventricle and main pulmonary artery. The wire is then captured and brought out through an opposite femoral or jugular venous access point, creating an arteriovenous loop that improves guide catheter stability and facilitates effective defect closure (Cubeddu et al., 2024).

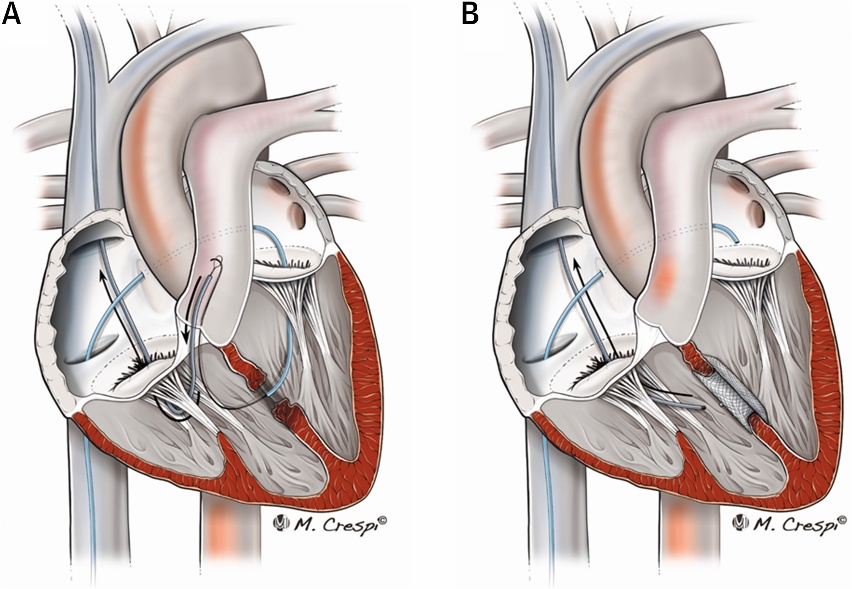


**Figure 2. Transcatheter Closure of PIVSR: Retrograde Approach**

(A) The catheter and wire are positioned retrograde through the aortic valve and ventricular septal rupture, with the wire subsequently advanced into the main pulmonary artery. (B) The catheter and snare device are positioned antegrade into the main pulmonary artery to capture the wire. (C) The wire is captured and externalized, forming an arteriovenous loop. (D) The Amplatzer delivery catheter (Abbott) is advanced antegrade along the wire and across the tricuspid valve through the defect. (E) The Amplatzer Occluder is positioned and released to seal the septal defect (adapted from Cubeddu et al., 2024).

**7.1.2 Antegrade Approach**

In the antegrade approach, the septal defect is accessed similarly following right femoral venous puncture, crossing the tricuspid valve and entering the left ventricle from the right ventricle. This method may be especially beneficial for patients with mechanical aortic valve prostheses (Cubeddu et al., 2024).



**Figure 3. Transcatheter Closure of PIVSR: Antegrade Approach**

(A) The catheter and wire are advanced in an antegrade manner from the femoral vein and across the interatrial septum, through the mitral valve into the ventricular septal rupture, with the wire then advanced into the main pulmonary artery. The subsequent steps mirror those of the retrograde technique shown in **Figure 2**. In brief, the wire is then captured in the main pulmonary artery and externalized to establish a venous-venous loop. This is accomplished using a second femoral venous access site or the right jugular vein, as demonstrated in this example. (B) The Amplatzer delivery catheter is advanced over the wire and across the tricuspid valve from the right jugular vein. The Amplatzer Occluder is positioned and released to seal the septal defect (adapted from Cubeddu et al., 2024).

**7.1.3 Hybrid Periventricular Approach**

Perventricular device closure (PVDC) is an innovative technique that has been effectively used for closing congenital apical muscular ventricular septal defects. It offers the benefit of direct access, which is particularly well-suited for apical muscular VSDs with complex anatomy. This approach can ensure optimal device positioning and reduce the risk of residual shunting. This hybrid procedure can be performed with cardiopulmonary bypass on standby to minimize the risk of hemodynamic compromise (Meng et al., 2013).

A diagram of the heart

AI-generated content may be incorrect.

**Figure 4. Transcatheter Closure of PIVSR: Hybrid Transcatheter-Periventricular Approach**

(A) Following percutaneous or surgical exposure (via small surgical window), a catheter is advanced through the right ventricular free wall and across the ventricular septal defect. (B) The Amplatzer Septal Occluder is deployed and released to close the septal defect (adapted from Cubeddu et al., 2024).

**8. Outcomes of Percutaneous PIVSR Closure**

**8.1 Technical And Procedural Success Rates**

Technical success in percutaneous VSR closure is generally defined as the successful deployment of the occluder device in the intended position across the VSR, leading to a significant reduction in shunt flow. Procedural success encompasses technical success plus the absence of major procedural complications. Reported technical and procedural success rates vary widely in the literature, influenced by patient selection, operator experience, VSR characteristics, and the type of devices used. Some studies report technical success rates ranging from 70% to 100% (Premchand et al., 2017). One series using Amplatzer ASD occluders reported successful device placement in five out of seven patients (71.4%) . Another study using various devices, including the Amplatzer PI VSD occluder and Konar MFO, reported successful device implantation in all attempted cases, although some required adjunctive procedures for residual shunts (Islam et al., 2025; Premchand et al., 2017). However, achieving complete and durable closure can be challenging. Residual shunts are common immediately post-procedure, reported in up to 62% of cases with ASO devices in one study, though many of these were small and clinically insignificant (Aggarwal et al., 2018).

**8.2 Short-Term and Long-Term Survival**

Short-term survival after percutaneous VSR closure is significantly influenced by the patient's pre-procedural hemodynamic status, the extent of myocardial infarction, and the presence of other comorbidities. In-hospital or 30-day mortality rates remain high, often reported between 30% and 60%, even with successful device implantation (Islam et al., 2025; Premchand et al., 2017). This reflects the critical nature of post-infarction VSR and the severity of underlying cardiac dysfunction. Patients who are in cardiogenic shock at the time of intervention have a particularly poor prognosis. For example, one study reported a 30-day mortality of 60% among successful ASO closures (Islam et al., 2025). Another study found that the median time from VSR diagnosis to intervention was significantly longer in the 30-day survivor group compared to the 30-day mortality group (31 days vs. 14 days, p=0.034), suggesting that patients who are stable enough to tolerate a delay in intervention may have better short-term outcomes, although this could also reflect selection bias (Hussein et al., 2025).

Data on long-term survival specifically after percutaneous closure are more limited and primarily derived from registries and smaller case series. An early US registry using the Amplatzer PIMVSD device reported that 11 of the initial 18 patients were alive at a median follow-up of 332 days. A subsequent multicenter study with the same device found 6-month survival rates of 37.2% and 46.4% in two analyzed cohorts (Holzer et al., 2004). A single-center report described long-term survival at 1 and 5 years follow-up in only 2 out of 7 patients who underwent the procedure (Premchand et al., 2017).

One systematic review suggested that long-term outcomes appear favorable for those patients who survive the initial hospitalization, regardless of whether the percutaneous closure was primary or for a post-surgical residual defect (Wilson & Horlick, 2016). However, this observation must be interpreted with caution due to the profound impact of survival bias. Patients who survive the acute phase likely represent a selected cohort with less severe initial myocardial damage, smaller or more favorably located defects, or better physiological reserve, leading to inherently better long-term prospects compared to the overall population presenting with VSR. Comparing these outcomes to surgical series, which report 5-year survival rates around 38% and 10-year survival rates between 33% and 44%, suggests that long-term survival remains a significant challenge irrespective of the initial repair strategy. Factors known to influence long-term survival after PIVSR repair include residual shunt, underlying coronary artery disease severity, right and left ventricular function, and functional status at presentation (Pang et al., 2013; S. Wang et al., 2024).

**8.3 Risks and Potential Complications of Percutaneous PIVSR Closure**

**8.3.1 Device Embolization or Malposition**

Dislodgement of the occluder device from the VSR site is a serious complication that can occur acutely or subacutely. Embolization typically occurs into the pulmonary artery or, less commonly, the left ventricular outflow tract or aorta. Contributing factors include inadequate tissue rims for anchoring, implantation in overly friable tissue, improper device sizing with undersizing or even excessive oversizing causing tissue stress, complex defect morphology, or device rigidity (Risseeuw et al., 2014). While percutaneous retrieval using snares may be feasible, embolization often necessitates emergency surgery and can be fatal (Oman et al., 2020).

**8.3.2 Residual Shunting and Hemolysis**

Incomplete closure with persistent left-to-right shunting is a common finding after percutaneous VSR closure, reported in up to 30% of cases.1 Shunting can occur directly through the interstices of the device especially with more permeable designs like ASD occluders before endothelialization or more commonly, paradevice leak due to incomplete apposition against the irregular septal defect or continued tissue retraction or necrosis (Aggarwal et al., 2018; Anbalakan et al., 2023). While trivial or small shunts may be hemodynamically tolerated, significant residual shunting contributes to ongoing volume overload, persistent heart failure, pulmonary hypertension, and increased mortality risk. It may necessitate further intervention (Holzer et al., 2004; Jorge et al., 2012). The high-velocity jet of blood that persists through or around the device can lead to ongoing heart failure symptoms and can also cause mechanical trauma to red blood cells, resulting in intravascular hemolysis. This can be severe enough to cause significant anemia and hemoglobinuria, sometimes requiring repeated blood transfusions (Chen et al., 2023).

**8.3.3 Arrhythmias and Conduction Disturbances**

The ischemic and irritable ventricular myocardium is highly susceptible to arrhythmias. Mechanical stimulation from guidewires, catheters, or the device itself can provoke life-threatening ventricular tachycardia or fibrillation. Furthermore, trauma to the cardiac conduction system, which runs through the interventricular septum, can result in high-grade atrioventricular block requiring pacemaker implantation (Schlotter et al., 2016).

**8.4 Factors Influencing Outcomes**

Numerous factors influence the outcomes of percutaneous VSR closure, encompassing patient-related, VSR-related, and procedure-related variables. Patient-related factors include age, baseline hemodynamic status the presence of cardiogenic shock, severity of underlying left ventricular dysfunction, and the extent of comorbid conditions (Hussein et al., 2025). Patients in profound shock or with extensive myocardial damage have a poorer prognosis. VSR-related factors are critical and include the size and location of the defect, its morphology simple vs. Complex or serpiginous, the quality of the surrounding tissue, the friability, extent of necrosis, and the presence of multiple defects (Chen et al., 2023). Larger defects, basal or apical locations, and complex morphologies are generally associated with higher procedural difficulty and worse outcomes. Procedure-related factors include the timing of intervention relative to the acute infarction, with delayed repair if hemodynamically feasible often associated with better outcomes due to tissue stabilization (Andersen & Zhao, 2018). The type of device used and the operator's experience also play significant roles. Dedicated VSR devices like the Amplatzer PI Muscular VSD Occluder may offer advantages over off-label use of ASD occluders in certain anatomies . Finally, the completeness of VSR closure and the absence of major procedural complications are strong determinants of both short-term and long-term outcomes. Successful revascularization of the infarct-related artery is also an important factor influencing long-term survival and ventricular function (Anbalakan et al., 2023).

9. FOLLOW UP AND SURVEILANCE IMAGING

Current practice typically involves routine clinical follow-up focused on assessing heart failure symptoms, functional capacity, and medication management. Periodic TTE is the mainstay of imaging surveillance, used to monitor device position, assess the magnitude of any residual shunt often quantified by color Doppler and Qp/Qs estimation, evaluate left and right ventricular size and function, and estimate pulmonary artery pressures (Holzer et al., 2004). The optimal frequency and duration of this surveillance are not defined and likely vary based on the initial procedural result, the size of any residual defect, and the patient's overall clinical status. Long-term follow-up extending to 1, 5, or even 10 years has been reported in some research cohorts (Premchand et al., 2017). Advanced imaging modalities like cardiac MRI may be considered in specific cases for more detailed assessment of device position, shunt quantification, or tissue characterization during follow-up (Zhong et al., 2019).

10. Comparison with Surgical Repair of PIVSR

**10.1 Comparative Mortality and Morbidity**

The management of PIVSR presents a significant challenge, with both percutaneous closure and surgical repair carrying substantial risks. A critical factor influencing outcomes for both approaches is the timing of intervention relative to the AMI and VSR diagnosis. For surgical repair, early intervention within 24 hours of presentation is associated with a mortality rate of approximately 60%. This high mortality reflects the instability of the freshly infarcted and ruptured tissue, making surgical repair technically demanding and prone to failure. As the interval from AMI to surgery increases, allowing for some degree of tissue healing and stabilization, mortality rates tend to decrease. For instance, surgical repair performed between 8 to 21 days post-presentation carries a mortality rate of around 30%, and if surgery can be delayed beyond 21 days, the mortality rate further drops to approximately 10% (Premchand et al., 2017). This highlights a widely accepted principle that delayed surgical repair, when hemodynamically feasible, is associated with better outcomes due to the maturation and strengthening of the necrotic septal tissue, which holds sutures better.

In contrast, the data for percutaneous closure, shows a different temporal pattern of mortality. Percutaneous closure attempted very early 1-3 days post-VSR was associated with an extremely high mortality rate of 88% (n=16). This likely reflects the selection bias towards attempting percutaneous closure in the most critically ill, hemodynamically unstable patients who are deemed too high-risk for immediate surgery. These patients often have extensive myocardial damage and are in profound cardiogenic shock. When percutaneous closure was performed slightly later 4-16 days post-VSR, the mortality rate was lower at 38% (n=13) . This suggests that even a short delay in percutaneous intervention, allowing for some stabilization, might improve outcomes. However, direct comparison of these percutaneous mortality rates with surgical rates is complicated by differences in patient selection, with percutaneous approaches often reserved for patients with prohibitive surgical risk. Conservative management, without any attempt at defect closure, carries a dismal prognosis, with a 30-day mortality rate exceeding 90%. The inherent selection bias must be recognized, patients who undergo early closure procedures, whether percutaneous or surgical, are frequently more critically ill, while those who can be treated conservatively or with delayed interventions are generally more stable. This factor complicates direct comparisons of mortality rates between these different treatment strategies (Premchand et al., 2017).

**10.2 Advantages and Disadvantages of Percutaneous Versus Surgical Approach**

From a treatment perspective, the percutaneous approach provides immediate, minimally invasive hemodynamic stabilization that can be conducted under conscious sedation, making it appealing for patients experiencing severe shock or who have excessive surgical risk. The capacity to repeat the procedure by deploying a second or larger device if the defect expands and to function as a bridge to delayed surgery once necrotic tissue stabilizes represent additional advantages (Ronco et al., 2023; Tripathi et al., 2023).

Surgical repair, while more invasive, provides definitive closure under direct vision and allows concomitant procedures such as coronary artery bypass grafting, left ventricular aneurysmectomy or placement of mechanical circulatory support (Dimagli et al., 2022; Firuzi et al., 2023). However, the benefit of concomitant CABG is not universally reported; a recent surgical series found no beneficial effect on survival for patients who underwent CABG at the time of VSR repair (Isik et al., 2021). Mature fibrotic tissue several weeks after infarction affords more reliable suture anchoring. accordingly, centres practising deferred surgery report markedly lower operative mortality compared with emergency repair within the first week (Sharma et al., 2013). Nonetheless, even optimal timing does not eliminate risk, and postoperative recovery is prolonged, with substantial need for prolonged ventilation, dialysis and inotropic support (Arnaoutakis et al., 2012; Ronco et al., 2021).

11. Future directions

The management of PIVSR continues to evolve across three critical domains that will define the next decade of clinical advancement.

Prospective registry development represents the most immediate need for evidence generation. Coordinated, multicenter registries capturing comprehensive patient characteristics, procedural details, and longitudinal outcomes will enable development of evidence-based selection algorithms.(Cadogan et al., 2023). Such registries should systematically evaluate the impact of mechanical circulatory support timing, optimal intervention windows, and comparative effectiveness of surgical versus percutaneous approaches across different patient subsets.(Hussein et al., 2025).

Advanced device innovation focused specifically on post-infarction anatomy holds significant promise. Purpose-designed occluders incorporating low-radial-force anchoring, adjustable waist dimensions for varying septal thickness, and bioabsorbable scaffolding components could address current limitations of adapted congenital devices. Complementary advances in steerable, low-profile delivery systems would improve procedural success rates, particularly for challenging posterior ruptures(Anbalakan et al., 2023; Susilo et al., 2025).

Integrated care Protocols combining standardized timing algorithms with mechanical support strategies represent the third priority area. Development of consensus-based decision frameworks that systematically incorporate tissue viability assessment, hemodynamic status, and defect characteristics will transform current "case-by-case" approaches into evidence-based clinical pathways (Anbalakan et al., 2023; Wilson & Horlick, 2016).

12. CONCLUSION

PIVSR remains one of the most challenging complications of acute myocardial infarction, with persistently high mortality rates despite therapeutic advances. While PIVSR incidence has declined significantly in the primary percutaneous coronary intervention era, effective management continues to require multidisciplinary expertise and individualized treatment approaches.

This review demonstrates that percutaneous closure has evolved from an experimental procedure to a viable therapeutic option for carefully selected patients. Technical success rates of 70% to 100% establish the feasibility of transcatheter approaches, particularly for high-risk surgical candidates. However, in-hospital mortality rates of 30% to 60% underscore the critical nature of this condition and the complexity of optimal patient selection.

Several key management principles emerge from current evidence. Intervention timing represents a critical determinant of outcomes, with delayed repair beyond two to three weeks consistently associated with improved survival when hemodynamically feasible. This advantage reflects tissue maturation from friable, necrotic myocardium to stable, fibrotic substrate capable of supporting device anchoring. Patient selection criteria continue to evolve, with defect characteristics including size, location, and morphology serving as primary determinants of procedural success. Apical defects from anterior infarctions generally offer more favorable anatomy than complex basal defects from inferior infarctions.

Current device limitations reflect the absence of purpose-designed occluders for post-infarction septal ruptures. The off-label use of devices developed for congenital defects necessitates careful consideration of device characteristics relative to defect anatomy. The Amplatzer Post-Infarction Muscular VSD Occluder represents the most appropriate available option, though continued device innovation remains necessary.

Outcomes analysis reveals that while percutaneous closure achieves technical success in selected patients, long-term survival remains limited by underlying myocardial damage and patient comorbidities. Successful defect closure, though necessary, proves insufficient to ensure favorable outcomes in patients with extensive infarction and associated complications.

Percutaneous and surgical approaches serve complementary rather than competing roles. Percutaneous closure offers reduced invasiveness and bridge-to-recovery capability, while surgical repair provides definitive closure with opportunity for concomitant procedures. Treatment selection should be individualized based on patient characteristics, defect anatomy, and institutional expertise.

Current field limitations reflect the condition's rarity and consequent reliance on small, retrospective series lacking statistical power for definitive guidance. The absence of randomized controlled trials limits evidence-based decision making. Prospective, multicenter registries with standardized protocols represent critical infrastructure needs for advancing clinical knowledge.

Future directions should prioritize device innovation targeting post-infarction septal rupture characteristics, advanced imaging for enhanced patient selection and procedural planning, and integration of mechanical circulatory support into standardized algorithms. Multi-institutional collaboration through coordinated registries and clinical trials will be essential for generating high-quality evidence to guide optimal management strategies.

Percutaneous closure has established itself as an important therapeutic option within interventional cardiology. While significant challenges persist regarding patient selection, timing optimization, and device technology, continued evolution of percutaneous approaches offers potential for improved outcomes. Success requires recognition that optimal results depend on comprehensive patient care encompassing appropriate timing, multidisciplinary collaboration, and individualized strategies tailored to each clinical presentation.

**Disclaimer (Artificial intelligence)**

Author(s) hereby declare that generative AI technologies such as Large Language Models, etc. have been used during the writing or editing of manuscripts. This explanation will include the name, version, model, and source of the generative AI technology and as well as all input prompts provided to the generative AI technology

Details of the AI usage are given below:

1. Model: Claude Sonnet 4, Source: Anthropic. Prompts: Suggest several potential titles for my review article

2. Model: Claude Sonnet 4, Source: Anthropic. Prompts: Please review the following text for grammatical errors, clarity, and scientific tone. Ensure the language is formal and suitable for a peer reviewed publication

References

Aggarwal, M., Natarajan, K., Vijayakumar, M., Chandrasekhar, R., Mathew, N., … Thachathodiyl, R. (2018). Primary transcatheter closure of post-myocardial infarction ventricular septal rupture using amplatzer atrial septal occlusion device: A study from tertiary care in South India. *Indian Heart Journal*, *70*(4), 519–527. https://doi.org/10.1016/j.ihj.2018.01.036

Ahmed, J., Ruygrok, P. N., Wilson, N. J., Webster, Mark. W. I., Greaves, S., & Gerber, I. (2008). Percutaneous Closure of Post-Myocardial Infarction Ventricular Septal Defects: A Single Centre Experience. *Heart, Lung and Circulation*, *17*(2), 119–123. https://doi.org/10.1016/j.hlc.2007.09.001

Ali, A., Mal, V., Ahmed, R., Memon, F., Lalchand, ., … Tariq, M. (2022). Frequency of Ventricular Septal Rupture in Patients of Acute Myocardial Infarction. *Journal of Pharmaceutical Research International*, 45–50. https://doi.org/10.9734/jpri/2022/v34i48B36420

Álvarez-Fuente, M., Ignacio Carrasco, J., Insa, B., Toledano, M., Peiró, E., … Del Cerro, M. J. (2022). Percutaneous closure of ventricular septal defect with the KONAR-MF device. *REC: Interventional Cardiology (English Edition)*. https://doi.org/10.24875/recice.m22000277

Anbalakan, K., Yap, J., Foo, J. S., Yan, L., Lee, P. T., … Tan, J. L. (2023). Technical Considerations for the Percutaneous Management of Post-MI Ventricular Septal Rupture. *Journal of Asian Pacific Society of Cardiology*, *2*, e39. https://doi.org/10.15420/japsc.2023.12

Andersen, M. M., & Zhao, D. X. M. (2018). Percutaneous Post-Myocardial Infarction Ventricular Septal Rupture Closure: A Review. *Structural Heart*, *2*(2), 121–126. https://doi.org/10.1080/24748706.2017.1421799

Arnaoutakis, G. J., Zhao, Y., George, T. J., Sciortino, C. M., McCarthy, P. M., & Conte, J. V. (2012). Surgical Repair of Ventricular Septal Defect After Myocardial Infarction: Outcomes From The Society of Thoracic Surgeons National Database. *The Annals of Thoracic Surgery*, *94*(2), 436–444. https://doi.org/10.1016/j.athoracsur.2012.04.020

Birnbaum, Y., Fishbein, M. C., Blanche, C., & Siegel, R. J. (2002). Ventricular Septal Rupture after Acute Myocardial Infarction. *New England Journal of Medicine*, *347*(18), 1426–1432. https://doi.org/10.1056/NEJMra020228

Cadogan, D., Daghem, M., Snosi, M., Williams, L. K., Weir-McCall, J., … Giblett, J. P. (2023). Percutaneous Transcatheter Closure of Post-infarction Ventricular Septal Defect: An Alternative to Surgical Intervention. *Interventional Cardiology: Reviews, Research, Resources*, *18*, e19. https://doi.org/10.15420/icr.2023.01

Chen, T., Liu, Y., Zhang, J., Sun, Z., Han, Y., & Gao, C. (2023). Percutaneous closure of ventricular septal rupture after myocardial infarction: A retrospective study of 81 cases. *Clinical Cardiology*, *46*(7), 737–744. https://doi.org/10.1002/clc.24027

Cubeddu, R. J., Lorusso, R., Ronco, D., Matteucci, M., Axline, M. S., & Moreno, P. R. (2024). Ventricular Septal Rupture After Myocardial Infarction. *Journal of the American College of Cardiology*, *83*(19), 1886–1901. https://doi.org/10.1016/j.jacc.2024.01.041

David, T. E. (2022). Post-infarction ventricular septal rupture. *Annals of Cardiothoracic Surgery*, *11*(3), 261–267. https://doi.org/10.21037/acs-2021-ami-111

DeLeon-Pennell, K. Y., Meschiari, C. A., Jung, M., & Lindsey, M. L. (2017). Matrix Metalloproteinases in Myocardial Infarction and Heart Failure. In *Progress in Molecular Biology and Translational Science* (Vol. 147, pp. 75–100). Elsevier. https://doi.org/10.1016/bs.pmbts.2017.02.001

Dimagli, A., Guida, G., Sinha, S., Dixon, L., Fudulu, D., … Angelini, G. D. (2022). Surgical outcomes of post‐infarct ventricular septal defect repair: Insights from the UK national adult cardiac surgery audit database. *Journal of Cardiac Surgery*, *37*(4), 843–852. https://doi.org/10.1111/jocs.16178

Faccini, A., & Butera, G. (2019). Techniques, Timing, and Prognosis of Transcatheter Post Myocardial Infarction Ventricular Septal Defect Repair. *Current Cardiology Reports*, *21*(7), 59. https://doi.org/10.1007/s11886-019-1142-8

Firuzi, A., Shekarchizadeh, M., Yadollahi, M., Mohamadifar, A., Ferasati, E., & ShekarchizadehEsfahani, M. (2023). Relationship between Complete Revascularization and Survival after Post-Infarction Ventricular Septal Rupture. *ARYA Atherosclerosis Journal*, *19*(Issue 3). https://doi.org/10.48305/arya.2022.11857.2539

Gong, F. F., Vaitenas, I., Malaisrie, S. C., & Maganti, K. (2021). Mechanical Complications of Acute Myocardial Infarction: A Review. *JAMA Cardiology*, *6*(3), 341. https://doi.org/10.1001/jamacardio.2020.3690

Holzer, R., Balzer, D., Amin, Z., Ruiz, C. E., Feinstein, J., … Hijazi, Z. M. (2004). Transcatheter closure of postinfarction ventricular septal defects using the new Amplatzer muscular VSD occluder: Results of a U.S. Registry. *Catheterization and Cardiovascular Interventions*, *61*(2), 196–201. https://doi.org/10.1002/ccd.10784

Hussein, H., Eltayeb, S., Mosaad, E., Shehata, M., Elafifi, A., … Samir, A. (2025). Surgical versus percutaneous closure of post-infarction ventricular septal rupture; review of literature and single-center experience. *BMC Cardiovascular Disorders*, *25*(1), 174. https://doi.org/10.1186/s12872-024-04370-4

Ishiyama, M., Kurita, T., Ishiura, J., Yamamoto, N., Sugiura, E., … Dohi, K. (2020). Successful percutaneous treatment of recurrent post-infarction ventricular septal rupture using an Amplatzer duct occluder. *Journal of Cardiology Cases*, *21*(1), 12–15. https://doi.org/10.1016/j.jccase.2019.09.003

Isik, M., Tanyeli, O., Dereli, Y., Gormus, N., & Yildirim, S. (2021). Surgical Repair of Post Myocardial Infarction Ventricular Septal Defects: Single Center Experience of Fifteen Years. *Selcuk Tip Dergisi*, *4*(37), 301–306. https://doi.org/10.30733/std.2021.01530

Islam, N., Saha, S., Parida, A. K., & Dutta, S. N. (2025). Percutaneous Closure of Postmyocardial Infarction Ventricular Septal Rupture – A Single Center Experience from the Eastern Part of India. *Journal of Indian College of Cardiology*, *15*(1), 22–26. https://doi.org/10.4103/jicc.jicc\_50\_24

Jones, B. M., Kapadia, S. R., Smedira, N. G., Robich, M., Tuzcu, E. M., … Krishnaswamy, A. (2014). Ventricular septal rupture complicating acute myocardial infarction: a contemporary review. *European Heart Journal*, *35*(31), 2060–2068. https://doi.org/10.1093/eurheartj/ehu248

Jorge, C., De Oliveira, E. I., Martins, S. R., Nobre, Â., Da Silva, P. C., & Diogo, A. N. (2012). Hybrid closure of postinfarction ventricular septal rupture enlargement after transcathether closure with Amplatzer occluder. *European Heart Journal: Acute Cardiovascular Care*, *1*(1), 57–59. https://doi.org/10.1177/2048872612441578

Kim, P. K., Hong, Y. J., Im, D. J., Suh, Y. J., Park, C. H., … Choi, B. W. (2017). Myocardial T1 and T2 Mapping: Techniques and Clinical Applications. *Korean Journal of Radiology*, *18*(1), 113. https://doi.org/10.3348/kjr.2017.18.1.113

Matteucci, M., Ronco, D., Corazzari, C., Fina, D., Jiritano, F., … Lorusso, R. (2021). Surgical Repair of Postinfarction Ventricular Septal Rupture: Systematic Review and Meta-Analysis. *The Annals of Thoracic Surgery*, *112*(1), 326–337. https://doi.org/10.1016/j.athoracsur.2020.08.050

Meng, W., Lin, K., & Zhang, E. (2013). Perventricular Closure on the Beating Heart: An Effective Hybrid Approach in Managing a Postinfarction Apical Ventricular Septal Defect: PERVENTRICULAR CLOSURE OF POSTINFARCTION VSD. *Journal of Cardiac Surgery*, *28*(1), 16–18. https://doi.org/10.1111/jocs.12031

Morton, K. F., Hasnie, U. A., Prime, D., Still, S. A., & McElwee, S. (2023). A Case of Post-Myocardial Infarction Ventricular Septal Rupture Complicated by Postoperative Septal Rupture. *JACC: Case Reports*, *22*, 101996. https://doi.org/10.1016/j.jaccas.2023.101996

Oman, Z., Kumar, S., Ghani, A., Sayed-Ahmad, Z., Horbal, P., … Helmy, T. (2020). Percutaneous repair of post-myocardial infarction ventricular septal rupture presenting with cardiogenic shock. *American Journal of Cardiovascular Disease*, *10*(4), 376–381.

Pang, P. Y., Sin, Y. K., Lim, C. H., Tan, T. E., Lim, S. L., … Chua, Y. L. (2013). Outcome and survival analysis of surgical repair of post-infarction ventricular septal rupture. *Journal of Cardiothoracic Surgery*, *8*(1), 44. https://doi.org/10.1186/1749-8090-8-44

Premchand, R. K., Garipalli, R., Padmanabhan, T., & Manik, G. (2017). Percutaneous closure of post-myocardial infarction ventricular septal rupture–A single centre experience. *Indian Heart Journal*, *69*, S24–S27.

Reynolds, H. R., & Hochman, J. S. (2010). Heartbreak. *European Heart Journal*, *31*(12), 1433–1435. https://doi.org/10.1093/eurheartj/ehq089

Risseeuw, F., Diebels, I., Vandendriessche, T., De Wolf, D., & Rodrigus, I. E. (2014). Percutaneous occlusion of post-myocardial infarction ventricular septum rupture. *Netherlands Heart Journal*, *22*(2), 47–51. https://doi.org/10.1007/s12471-013-0498-4

Ronco, D., Ariza-Solé, A., Kowalewski, M., Matteucci, M., Di Mauro, M., … Lorusso, R. (2023). The current clinical practice for management of post-infarction ventricular septal rupture: a European survey. *European Heart Journal Open*, *3*(5), oead091. https://doi.org/10.1093/ehjopen/oead091

Ronco, D., Matteucci, M., Kowalewski, M., De Bonis, M., Formica, F., … Lorusso, R. (2021). Surgical Treatment of Postinfarction Ventricular Septal Rupture. *JAMA Network Open*, *4*(10), e2128309. https://doi.org/10.1001/jamanetworkopen.2021.28309

Schlotter, F., De Waha, S., Eitel, I., Desch, S., Fuernau, G., & Thiele, H. (2016). Interventional post-myocardial infarction ventricular septal defect closure: a systematic review of current evidence. *EuroIntervention*, *12*(1), 94–102. https://doi.org/10.4244/EIJV12I1A17

Shafiei, I., Jannati, F., & Jannati, M. (2020). Optimal Time Repair of Ventricular Septal Rupture Post Myocardial Infarction. *Journal of the Saudi Heart Association*, *32*(2), 288–294. https://doi.org/10.37616/2212-5043.1120

Sharma, Y. P., Kamana, N. K., & Vadivelu, R. (2013). Precision in cardiology: should all cases of myocardial infarction with ventricular septal rupture require early repair? *Heart Asia*, *5*(1), 235–237. https://doi.org/10.1136/heartasia-2012-010219

Susilo, H., Prabowo, E., Kurniawan, R. B., Kartikasari, D. P., Maulana, A. S., & Oktaviono, Y. H. (2025). Transjugular approach percutaneous closure: a preferred solution for challenging surgical management of ventricular septal rupture. *The Egyptian Heart Journal*, *77*(1), 42. https://doi.org/10.1186/s43044-025-00638-y

Tao, Z.-Y., Cavasin, M. A., Yang, F., Liu, Y.-H., & Yang, X.-P. (2004). Temporal changes in matrix metalloproteinase expression and inflammatory response associated with cardiac rupture after myocardial infarction in mice. *Life Sciences*, *74*(12), 1561–1572. https://doi.org/10.1016/j.lfs.2003.09.042

Tripathi, A., Bisht, H., Arya, A., Konat, A., Patel, D., … Sharma, K. (2023). Ventricular Septal Rupture Management in Patients With Acute Myocardial Infarction: A Review. *Cureus*. https://doi.org/10.7759/cureus.40390

Wang, L., Xiao, L. L., Liu, C., Zhang, Y. Z., Zhao, X. Y., … Dong, J. Z. (2021). Clinical Characteristics and Contemporary Prognosis of Ventricular Septal Rupture Complicating Acute Myocardial Infarction: A Single-Center Experience. *Frontiers in Cardiovascular Medicine*, *8*. https://doi.org/10.3389/fcvm.2021.679148

Wang, S., Liu, H., Yang, P., Wang, Z., & Chen, S. (2024). Current Understanding of Timing of Surgical Repair for Ventricular Septal Rupture following Acute Myocardial Infarction. *Cardiology*, 1–14. https://doi.org/10.1159/000538967

Wilson, W. M., & Horlick, E. M. (2016). Management of post-myocardial infarction ventricular septal rupture. *EuroIntervention*, *12*(X), X18–X23. https://doi.org/10.4244/EIJV12SXA4

Zhang, X.-Y., Bian, L.-Z., & Tian, N.-L. (2021). The Clinical Outcomes of Ventricular Septal Rupture Secondary to Acute Myocardial Infarction: A Retrospective, Observational Trial. *Journal of Interventional Cardiology*, *2021*, 1–7. https://doi.org/10.1155/2021/3900269

Zhong, W., Liu, Z., Fan, W., Hameed, I., Salemi, A., … Zhong, Z. (2019). Cardiac MRI-guided interventional occlusion of ventricular septal rupture in a patient with cobalt alloy stent. *Annals of Translational Medicine*, *7*(16), 395–395. https://doi.org/10.21037/atm.2019.07.55