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# Percutaneous Closure of Post-infarction and Iatrogenic Ventricular Septal Ruptures Using Amplatzer Occluder®: A Systematic Review

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**Abstract** Ventricular septal rupture (VSR) is a rare complication of myocardial infarction (MI), open heart surgery, and cardiac-based procedures, such as septal myectomy and valve replacement. VSR is associated with high mortality rates and the reported 30-day survival rate is less than 10% without any interventional therapy. Hence, prompt diagnosis and aggressive medical treatment with appropriate surgical intervention are necessary to improve survival. Immediate surgical intervention which is the standard treatment of VSR has a mortality rate of 19-60%. Due to persistent high mortality rate and challenging management of VSR, alternatives to surgical repair has been proposed; transcatheter approach as a new alternative method has been used for the closure of post-surgery residual defects or as a bridge to surgery and in some cases as a definitive therapy instead of surgical repair. Amplatzer Occluder® (AO), a type of transcatheter closure devices, is an approved method of repairing congenital atrial septal defects and it is being used as an alternative method of treatment in VSR. In this systemic review, we assessed the cases of VSR who underwent septal repair by using AO. The study shows that the total mortality rate of percutaneous VSR repair with AO is 20% which is comparable to 19-60% rate of death in patients who undergo surgery. While early intervention is necessary to prevent biventricular dysfunction, immediate surgical intervention on soft and friable tissue surrounding the infarction increases the risk of residual shunt and reoperation. However, this study reveals that the mortality rate of primary percutaneous VSR closure within 7 days of VSR detection was 37 % which is significantly lower than 60% in surgical repair in the same period. In conclusion, given that the prevalence of residual leak in both interventions are similar and close to 20%, percutaneous VSR closure with AO device could be superior to the surgical repair as a primary intervention in unstable or high-risk surgical patients.

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## 1. Introduction

Ventricular septal rupture (VSR) is a rare but well-known complication of myocardial infarction (MI); however, it has been also reported as a complication of open heart surgery and cardiac-based procedures, such as septal myectomy and valve replacement [1,2,3]. Studies indicate that VSR initially complicated MI at a rate of 1-2%, however, advancing the reperfusion therapy has significantly reduced the incidence to 0.2-0.3% in this cohort [4,5]. Despite this achievement, VSR is still associated with high mortality rates, and the reported 30-day survival rate is less than 10% without any interventional therapy [5,6]. Hence, prompt diagnosis and aggressive medical treatment with appropriate surgical intervention are necessary to improve survival [4].

The current American College of Cardiology and American Heart Association recommendation is immediate surgical repair for patients with VSR, regardless of the hemodynamic status [7]. However, the surgical repair outcome of VSR is still disappointing with the mortality rate of 19-60%, mainly due to the hemodynamic instability condition before the surgery following the ventricular dysfunction and left-to right shunt [5,8]. In

addition, early intervention on soft and friable myocardial tissue surrounding the infarction increases the risk of residual shunt and reoperation [5]. Due to persistent high mortality rate and challenging management of post-MI VSR, alternatives to surgical repair have been proposed; transcatheter approach as a new alternative method has been used for the closure of post-surgery residual defects or as a bridge to surgery and in some cases as a definitive therapy instead of surgical repair [8].

Amplatzer Occluder® (AO), a type of transcatheter closure device, is an approved method of sealing congenital atrial septal defects [9]. The AO is a self-expandable double-disk device consisting of a polyester material (Nickel-Titanium) that develops occlusion and tissue growth. After locating the device on the ruptured wall, the first and second discs are deployed on the outer and inner aspects of the ruptured septum, respectively [10]. The AO sizes are available in a wide range from 3-38 mm corresponding to the septal defects. Even though thrombotic events following AO placement have been rarely reported, the complication could be serious [11]. Hence, starting proper antiplatelet therapy and regular echocardiographic evaluation are necessary, and if any thrombosis is detected anticoagulation therapy with close follow-up is required [12]. In this study, we assessed the literature describing the use of AO in repairing VSR following myocardial infarction and cardiac-based procedure to have a better understanding of the benefits, risks, and outcomes.

## 2. Materials and Methods

A comprehensive computer-based literature search of English language studies was performed, using PubMed, Google Scholar, CINAHL, Cochrane CENTRAL, and Web of Science databases, to identify the relevant literature. Our search keywords included cardiac septal rupture, ventricular septal rupture, ventricular septal defect, myocardial infarction, cardiac-based procedure, septal ablation, Amplatzer Occluder®, transcatheter ventricular septal rupture closure, and percutaneous closure were used to determine cases of VSR who underwent percutaneous closure procedure. This extensive scoping study includes articles from May 1998 until September 2019. Relevant cases were selected by reviewing the reference list of each article. All non-English and non-human studies were excluded. Furthermore, all meta-analysis, review articles, and abstracts were excluded from this study. Data regarding demographic information, cause of free wall rupture, location of the perforation, size of Amplatzer, and complication of the percutaneous closure and outcome were reviewed and analyzed.

## 3. Results

Applying the keywords in database searching, we identified 1360 related studies. However, only 774 of them were qualified given the eligibility criteria and title. After reviewing the eligible article abstracts, 25 studies were selected. Subsequently, 12 case reports were selected for collecting the data and analysis by reviewing the full-text of the literature.

A total of 25 patients with the mean ± standard deviation (SD) age of 70.08 ± 12.04 years and male gender dominancy (52%) were identified (Table 1). VSR following acute myocardial infarction was related to 76% (19/25) of cases in this study. The other mechanisms involved in VSR were septal myomectomy and cardiac valve replacement (Table 2). The average size of VSR was 11.38 mm (range 5-32mm). In 20/25 (80%) of the cases AO was used as the primary device for management. The mean ± SD duration between the VSR development and AO placement for primary treatment was 25.73 ± 13.76 days. In the remaining 5/25 (20%) cases underwent surgical VSR repair. All 5/25 cases required percutaneous AO placement later to fix hemodynamic instability following persistent residual shunt. The mean ± SD duration between surgical repair and AO placement was 59 ± 39.47 days. Procedural success without procedure related complications and without significant residual leak was achieved in 68% (17/25) of the cases. In the remaining 32% (8/25) there were complications during the procedure or were diagnosed with persistent significant residual shunt. Among these 50% (4/8) of them survived with continued post-procedural medical management

The total number of deaths in this cohort was 20% (5/25). 80% (4/5) of them were related to peri-procedural worsening of leak and hemodynamic instability due to unsuccessful AO placement. In one case (1/5) the death was secondary to progressive renal failure even though the successful AO placement, proved by the autopsy, provided stable hemodynamic condition. Furthermore, 32% (8/25) of cases underwent AO placement within the first 7 days of diagnosis with a mortality rate of 37% (3/8). Therefore, the procedural success rate of VSR repair through percutaneous AO placement was 84% (21/25) with the total mortality rate of 20% (5/25) (Table 4). The average post-procedure follow-up in this cohort was about 15 months.

**Table 1. Demographic data and outcome for the studies reviewed**

| Information | Number |
|---|---|
| Total cases | 25 |
| Age | Mean 70.08 ± 12.04<br>Median 72.5 ± 12.04 |
| Sex | Females 12 (48%)<br>Males 13 (52%) |
| Primary and secondary percutaneous closure | 20 (80%) and 5 (20%) |
| Cases of successful closure<br>(primary and secondary device use) | 21 (84%) |
| Subsequent death | 5 (20%) |

**Table 2. Etiology of ventricular septal rupture**

| Mechanism | Number (%) |
|---|---|
| Post-myocardial infarction | 19 (76) |
| Non-surgical septal reduction | 1 (4) |
| Septal myectomy | 2 (8) |
| Traditional aortic valve replacement | 2 (4) |
| Mitral valve replacement | 1 (4) |

**Table 3. Cases of percutaneous closure of ventricular septal rupture included in this study**

| | Year, Author | Etiology of the perforation | Location of the perforation | Post perforation signs and symptoms | Initial management once VSR was detected |
|---|---|---|---|---|---|
| 1 | 2008, Giombolini [18] | Myocardial infarction | Sub-aortic valve VSD | Hemodynamic deterioration | IABP for 20 days |
| 2 | 2007, Ahmed [19] | Myocardial infarction | Infero-apical VSD | Hypotension and dyspnea | IABP, CABG, and emergency surgical repair of the VSR |
| 3 | 2007, Ahmed [19] | Myocardial infarction | Infero-basal VSD | Dyspnea and new systolic murmur | IABP and emergency surgical repair of the VSR |
| 4 | 2007, Ahmed [19] | Myocardial infarction | Infero-basal VSD | New systolic murmur | Observation for 50 days |
| 5 | 2007, Ahmed [19] | Myocardial infarction | Infero-septal VSD | New systolic murmur | CABG and observation for 4 weeks |
| 6 | 2007, Ahmed [19] | Myocardial infarction | Infero-basal VSD | Hypotension and shock | IABP and stent placement |
| 7 | 2001, Mullasari [20] | Myocardial infarction | Distal VSD | Dyspnea | Observation for 2 weeks |
| 8 | 2008, Szkutnik [21] | Myocardial infarction | Apical VSD | Dyspnea and low cardiac output | IABP and inotropic drugs |
| 9 | 1998, Lee [22] | Myocardial infarction | Mid-septal VSD | Hypotension, dyspnea, and new murmur | IABP, inotropic drugs, CABG, and urgent surgical VSR repair |
| 10 | 2005, Wacinski [2] | Myocardial infarction | Infero-septal VSD | New murmur | Prompted percutaneous VSR closure |
| 11 | 2004, Aroney [1] | Non-surgical septal reduction | VSD | Dyspnea, elevated JVD, and new murmur | Prompted percutaneous VSR closure |
| 12 | 2008, Martinez [3] | Myocardial infarction | Infero-basal VSD | - | - |
| 13 | 2008, Martinez [3] | Myocardial infarction | Inferior VSD | - | - |
| 14 | 2008, Martinez [3] | Myocardial infarction | Infero-basal VSD | - | - |
| 15 | 2008, Martinez [3] | Myocardial infarction | Apical VSD | - | - |
| 16 | 2008, Martinez [3] | Myocardial infarction | Apical VSD | - | CABG and urgent surgical VSR repair |
| 17 | 2008, Martinez [3] | Septal myectomy | Mid-basal VSD | Dyspnea and low cardiac output | - |
| 18 | 2008, Martinez [3] | Septal myectomy | Mid-inferior VSD | - | - |
| 19 | 2008, Martinez [3] | AVVR | Membranous VSD | - | - |
| 20 | 2008, Martinez [3] | MVR | Sub-aortic valve VSD | - | - |
| 21 | 2008, Martinez [3] | AVR | Sub-aortic valve VSD | - | - |
| 22 | 2015, Shabestari [23] | Myocardial infarction | Apical VSD | Progressive dyspnea | IABP and CABG |
| 23 | 2008, Tejedor [24] | Myocardial infarction | Anterior VSD | Dyspnea and low cardiac output | - |
| 24 | 2019, Wang [25] | Myocardial infarction | Apical VSD | Dyspnea and palpitation | - |
| 25 | 2019, Ishiyama [26] | Myocardial infsrction | Anterior VSD | Hypotension, JVD, and new murmur | IABP and urgent surgical VSR repair |

VSR = Ventricular septal rupture, VSD = Ventricular septal defect, IABP = Intra-aortic balloon pump, CABG = Coronary artery bypass graft, JVD = Jugular venous distention, AVVR = Atrioventricular valve replacement, MVR = Mitral valve replacement, AVR = Aortic valve replacement.

**Table 4. Outcomes of percutaneous closure of ventricular septal rupture in this study**

| | Year, author | Size of Amplatzer Occluder (mm) | Significant residual shunt or complications related to Amplatzer Occluder placement | Proper intervention after Amplatzer placement complication | Length of stay | Follow-up |
|---|---|---|---|---|---|---|
| 1 | 2008, Giombolini [18] | 10 | None | None | 20 days | Death, 3 days later due to progressive renal failure despite adequate hemodynamic conditions. Autopsy showed good position of the device |
| 2 | 2007, Ahmed [19] | 18 | None | None | 13 days | Stable, 287 days later |
| 3 | 2007, Ahmed [19] | 12 | Moderate residual leak | None | 15 days | Death, 5 days later due to multi-organ failure |
| 4 | 2007, Ahmed [19] | 20 | Hemolytic anemia due to very small residual leak. Large groin hematoma | Blood transfusion | - | Stable, 540 days later |

| | Year, author | Size of Amplatzer Occluder (mm) | Significant residual shunt or complications related to Amplatzer Occluder placement | Proper intervention after Amplatzer placement complication | Length of stay | Follow-up |
|---|---|---|---|---|---|---|
| 5 | 2007, Ahmed [19] | 20 | Atrial flutter. Femoral arterial damage and large groin hematoma | Cardiac conversion. Femoral artery surgical repair | 6 days | Stable, 270 days later |
| 6 | 2007, Ahmed [19] | - | Device could not be placed successfully due to inability to cross the defect, likely related to tortuosity of the defect | The procedure was abandoned due to hemodynamic and rhythm instability | 3 days | Death, within a day after the procedure |
| 7 | 2001, Mullasari [20] | 10 | None | None | 2 weeks | Stable, 30 days later |
| 8 | 2008, Szkutnik [21] | 35 | None | None | 30 days | Stable, 540 days later |
| 9 | 1998, Lee [22] | 10 | None | None | 1 week | Stable, 49 days later |
| 10 | 2005, Wacinski [2] | 20 | None | Adjuvant prophylactic antibiotic therapy for 6 months | 7 days | Stable, 360 days later |
| 11 | 2004, Aroney [1] | 20 | Hemolytic anemia due to small high velocity shunt | Blood transfusion | 10 days | Stable, 180 days later |
| 12 | 2008, Martinez [3] | 30 | Moderate residual shunt | None | - | Yes, due to renal and CNS complications despite improved hemodynamics. |
| 13 | 2008, Martinez [3] | 20, 10 | Moderate residual leak after first device placement | Second device placement 4 months later with small residual leak at f/u | - | Death, due to renal and CNS complications despite improved hemodynamics. |
| 14 | 2008, Martinez [3] | 12 | None | None | - | Stable, 540 days later |
| 15 | 2008, Martinez [3] | 16 | None | None | - | Stable, 1800 days later |
| 16 | 2008, Martinez [3] | 18 | None | None | - | Stable, 540 days later |
| 17 | 2008, Martinez [3] | 18 | None | None | | Stable, 360 days later |
| 18 | 2008, Martinez [3] | - | None | None | - | - |
| 19 | 2008, Martinez [3] | 14 | None | None | - | Stable, 180 days later |
| 20 | 2008, Martinez [3] | 10-8 | None | None | - | Stable, 180 days later |
| 21 | 2008, Martinez [3] | 6 | None | None | - | - |
| 22 | 2015, Shabestari [23] | 20 | None | None | - | - |
| 23 | 2008, Tejedor [24] | 35 | Moderate residual leak and biventricular dysfunction | Continue IABP and inotropic therapy | | Stable, 2160 days later |
| 24 | 2019, Wang [25] | - | None | None | 10 days | Death, 2 days later due to severe tricuspid regurgitation and biventricular dysfunction |
| 25 | 2019, Ishiyama [26] | 12 | None | None | 50 days | Stable, 360 days later |

CNS = Central nervous system, IABP = Intra-aortic blood pump f/u: follow-up.

## 4. Discussion

Myocardial rupture is a rare but life-threatening complication of acute myocardial infarction, cardiac-based procedure, chest trauma, and open-heart surgery [1,2,9,13]. VSR, a type of myocardial rupture, is commonly related to MI and complicates 0.2-0.3% of the cases [4,5]. However, before introducing the reperfusion treatment, the incidence rate of post-infarction VSR was 10-fold higher compared to now [13]. Despite advancing the reperfusion procedure, VSR is still associated with a poor prognosis and high number of the deaths, exceeding 90% of patients who go untreated [5,6]. This high mortality rate might be due to prolonged left-to-right shunt following VSR which causes systemic hypoperfusion and ultimately multiple organ failure [14]. Hence, rapid diagnosis and appropriate surgical intervention is crucial for improving patient outcomes [4].

Transthoracic echocardiography with Doppler imaging is a sensitive and specific study in investigating VSR and

estimating size of the defects [8,15]. The size and morphology of VSR are variable but generally it can be characterized as simple or complex. Simple defects are defined as a distinct similar level connection across the ventricular septum, and complex defects includes multiple small serpiginous tracts between the LV and RV [15]. Given the fact that new left to right shunt decreases the cardiac output and leads hemodynamically instability, stabilizing the patient to preserve the cardiac output is necessary to precede the intervention. While vasopressors are commonly used in patients with different types of shock, they can increase afterload which could worsen the left to right shunt. Hence IABP devices are used in unstable patients (Table 3) [16].

Surgery has been the gold standard treatment of VSR. Evidence-based guidelines recommend surgical intervention in the early stages of VSR development to shorten the duration of shunt and systemic hypoperfusion that ultimately prevents multiple organ failure and death [14]. However, the surgeons usually have to postpone the intervention for 3-4 weeks since early surgical repair on soft and friable tissue surrounding the infarction increases the chance of residual shunt and ultimately reoperation [5,14]. It explains why the mortality rate of VSR surgical repair varies but remains persistently high between 19 to 60% [8]. In addition, the studies show that the mortality rate could go up to 100% and surgery is ineffective in some of the cases with severe right ventricular dysfunction and very elderly patients [8]. *Arnaoutakis et al* recommends percutaneous transcatheter repair as an alternative treatment method in high-risk surgical patients with multiple risk factors, or unstable patients with signs of cardiogenic shock, multiple organ failure and in those with mechanical circulatory support needs [17].

The AO device is a self-expandable double-disk device and a connecting waist of 10 mm in length [13]. The AO consists of polyester material (nitinol) that develops in situ thrombosis of the waist to occlude the VSR. Even though the devices are retrievable for repositioning if the initial result is unsatisfactory, once the procedure is completed they are not retrievable [11,13].

In these systematic reviews, (8/25) 32% of AO placement procedures were complicated with significant residual leakage, hemolytic anemia, or groin hematoma. Although, 20% of cases developed significant residual shunt, only one of them needed a new device placement four months later. The incidence of significant residual shunt is similar to the surgical repair residual leak which has been reported 20% [16]. This study showed that the total mortality rate of percutaneous VSR repair with AO is 20% which is comparable to 19-60% rate of death in patients who undergo surgery [8]. Mubarak et al reported that late surgical intervention (>7 days) has a better prognosis compared to emergent surgery (<7 days), and decreases the mortality rate from 60% to 18.4%. However, this study revealed that the mortality rate of primary percutaneous VSR closure within 7 days of VSR detection was 37 % which significantly lower than 60% in surgical repair [16]. Hence, while the prevalence of residual leak in both interventions are similar, percutaneous VSR closure with AO device might be superior to the surgical repair as a primary intervention in unstable or high-risk surgical patients. Furthermore, the long-term prognosis was reported well in our patients who survived and were discharged from the hospital. Average long-term follow-up for patients who survived to hospital discharge was about 15 months (Table 4). However, more investigations are required to extrapolate the data using AO device to repair VSR

## 5. Limitations

Devices for the VSR repair, it might be subject to selection bias. In addition, there are a limited number of cases that employed AO devices in order to repair VSR. All of the referenced studies were either case reports or case series without any control group to enhance the accuracy of the comparison. Importantly, some of the potential side effects of the AO device placement, such as device induced wall necrosis, and septic or thrombotic emboli formation might be disregarded given the limited studies on available AO devices.

## 6. Conclusion

VSR is an uncommon but fatal complication of MI and cardiac procedures. The mainstay treatment of VSR is surgical repair. While the ACC/AHA guideline recommends emergent surgical repair, most of the cases undergo delayed surgery to have a better outcome given the necrotic tissue surrounding the rupture is soft and friable, and it increases the risk of residual shunt and reoperation. It explains why the post-operation mortality rate is persistently high, especially in emergent surgical repair, less than 7 days. The current systematic review demonstrates that while the incidence rate of significant residual leak in VSR repair using AO device might be the same as surgical repair, the mortality rate of primary percutaneous VSR closure within 7 days of VSR detection is 37 % which is significantly lower than 60% in surgical repair. Therefore, employing AO in the VSR repair could be a potentially life-saving alternative, especially in patients who are unstable or very high risk to undergo surgery. However, further studies are required to evaluate the outcome and mortality rate of using AO in the VSR repair to provide us with more consistent and accuratedata.

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