Post Infarction Ventricular Septal Rupture: Transcatheter intervention or Surgical repair?

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September 25, 2021

Abstract

ABSTRACT Post infarction ventricular septal rupture (PIVSR) is an infrequent but potentially fatal complication of acute myocardial infarction. * The 30-day mortality rate with the transcatheter approach when performed in the acute phase (less than two weeks) was 25.3% compared to 50% when surgery is performed in the acute phase (within three weeks). * There is no correlation between defect size and mortality. * NYHA class IV and time to VSD closure are risk predictors for transcatheter closure for a 30-day mortality rate of 31.5%.

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Funding: There was no source of funding.

Disclosure: The authors state that there was no relationship with any industry and there was no conflict of interest.

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Acknowledgements : The authors have no acknowledgments to mention

ABSTRACT

Post infarction ventricular septal rupture (PIVSR) is an infrequent but potentially fatal complication of acute myocardial infarction.

- The 30-day mortality rate with the transcatheter approach when performed in the acute phase (less than two weeks) was 25.3% compared to 50% when surgery is performed in the acute phase (within three weeks).
- There is no correlation between defect size and mortality.
- NYHA class IV and time to VSD closure are risk predictors for transcatheter closure for a 30-day mortality rate of 31.5%.
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Post infarction ventricular septal rupture (PIVSR) is an infrequent but potentially fatal complication of acute myocardial infarction. The natural history study shows a bimodal peak with the first peak at 16–24 hours and the second at 3-5 days following acute myocardial infarction. Improved reperfusion strategies have decreased the incidence of PIVSR to less than 0.21% (1) however, irrespective of the frequency if untreated the mortality rate is as high as 94% (2). The complication occurs in the setting of complete occlusion of the coronary artery leading to myocardial necrosis and rupture of the ventricular septal wall. The resulting myocardial necrosis and the sudden onset of a cardiac shunt exposes the pulmonary vascular bed to increased pulmonary blood flow and near systemic pressures. Unlike the congenital shunts this change in hemodynamics is poorly tolerated and causes additional stress to the already infarcted heart and exacerbation of the cardiogenic shock. With medical management alone the one-month survival with medical therapy is around 6%. Several risk factors have been associated with the development of PIVSR including advanced age, systemic hypertension and late arrival to hospital following acute myocardial infarction (3).

Yang and colleagues through meta-analysis analyzed 27 trials which included a total of 462 patients to evaluate the outcome of transcatheter closure of PIVSR (4). Cardiogenic shock was present in 47.2% of the patients, 35.8% of the patients were on an intra-aortic balloon pump and 49.4% were on inotropes. There was immediate shunt reduction in 80.9% following transcatheter closure of defect which is consistent with the previously published data (5). NYHA class IV (OR=6.491, 95% CI: 1.444-29.188) and time to VSD closure were risk predictors for transcatheter closure for a 30-day mortality rate of 31.5%. They also summarized that there was no correlation between defect size (OR=2.592, 95% CI: 0.380-17.661) and mortality. An important finding in this meta-analysis is that the 30-day mortality rate with the transcatheter approach when performed in the acute phase (less than two weeks) was 25.3% compared to 50% when surgery is performed in the acute phase (within three weeks). The study suggests that transcatheter closure has a lower mortality rate than surgical repair. The article also confirmed previous reports (6) that although the technical success rate (defined as successful device placement) is high, it did not always correlate with improved long-term outcomes, as the copresence of cardiogenic shock plays an important role in the prognosis of these patients. Multiple devices were used for transcatheter closure with the Amplatzer PI Muscular VSD device in 36.9%, Amplatzer muscular VSD device in 33.2%, Amplatzer septal occluder in 33% and a host of other devices making it difficult to compares devices or draw conclusions about device selection and outcomes.

PIVSR should be suspected when a sudden change in hemodynamics occurs following acute myocardial infarction. Early closure of the PIVSR is recommended based on natural history data which suggest a 94% mortality rate within 2 months if untreated irrespective of the hemodynamic status. Surgical intervention following PIVSR can be challenging and carries a high risk for mortality. A recent meta-analysis found a high operative mortality of 38.2% with surgical repair (7). Analysis from the society of thoracic surgeons national database has demonstrated high mortality rate when operated within the first week compared to when repair is performed after seven days (54.1% vs 18.4%, respectively) (8). The high mortality rate associated with surgical repair can be attributed to operating on a highly fragile cardiac tissue under cardiopulmonary bypass on a patient with cardiogenic shock. There is also a 10-40% incidence of residual ventricular septal defects following surgical repair of the defect which can be related to patch dehiscence and ongoing necrosis

of the myocardium (9). There are no randomized controlled trials, however, the outcome of transcatheter intervention is similar or superior in some studies when compared to the available data for surgical repair outcomes. A review of available literature comparing the outcomes in the three arms of management (medical treatment, surgical repair, and transcatheter intervention) have demonstrated that mortality associated with transcatheter closure is significantly lower when compared to medical or surgical management (10). Published data that shows similar mortality between the surgical and transcatheter approach can be explained by the fact that patients selected for transcatheter closure were at high risk for surgical repair and therefore, the pre-intervention mortality was already high.

Patient selection for transcatheter device closure of the defects should include patient risk factors. The defect is serpiginous where the left ventricular entry and the right ventricular exit of the defect can be distant and posteriorly located (11). The defect is often complex and advanced imaging is necessary to outline the anatomic details of the defect and its proximity to adjacent structures to help with device selection. Patients in whom transcatheter closure is thought to be technically feasible are usually patients with defects less than 15mm in size (12). Transcatheter approach for the majority of patients results in an immediate shunt reduction thereby stabilizing the hemodynamics. Balloon sizing of the defect prior to device placement is not recommended because balloon manipulation can worsen tissue dehiscence or lead to rupture extension of the free (ventricular) wall in the presence of myocardial necrosis. Following transcatheter closure the defect there is a risk of shunt recurrence because of tissue necrosis and the patients should be evaluated with an echocardiogram at close intervals. It is not uncommon to require additional device implantation either at the time of initial closure or within 3 to 6 days of the initial procedure. In select patients with a large ventricular septal defect where the risk of device dislodgement and technical challenges to device placement are significant, a hybrid approach with early surgery and potential transcatheter closure of residual or recurrent defects is an alternative.

Yang and colleagues in their meta-analysis have shown that transcatheter closure is safe and feasible. Given the high surgical mortality rate to repair this lesion transcatheter closure of the defect is a viable option.

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