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Optimal Time Repair of Ventricular Septal Rupture Post Myocardial Infarction

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Abstract

Ventricular septal rupture (VSR) is an uncommon complication of myocardial infarction (MI). The mortality rate of VSR is high. The management of VSR is including the stabilization of the hemodynamic status and surgical closure of the rupture. In spite of the agreement of experts on the necessity of surgical repair, the timing of VSR repair management remains unclear. In this review article, we evaluate the optimal time repair of VSR. To collect the data, Pubmed, EMBASE, and Cochrane Central Registry databases were searched for potentially suitable studies. Search terms were including “Ventricular Septal Rupture”, “Myocardial Infarction”, “Timing”, and “MI”. According to the result of the studies, it seems that the time between VSR detection and its repair is a determining factor in the survival or mortality of patients in post-myocardial infarction VSR. Studies showed that earlier surgical repair in VSR increases the risk of mortality, because in the early phase after MI, infarcted myocardium is very fragile, and it is very difficult surgical repair and increases the risk of recurrent septal defects. The longer time is needed for the heart and different body systems to adapt to the hemodynamic results of the abrupt left to right shunt. It seems that the best time for the operation is after the maturation of VSR with scarring at the edges of the defect. Moreover, in a large number of patients, it is not possible to delay the operation since they are at risk of severe heart failure and organ dysfunction. In these cases operation immediately after diagnosis of VSR is recommended to prevent further hemodynamic deterioration. In hemodynamically compromised patients, it may be considered to use a ventricular assist device, requiring an intra-aortic balloon pump (IABP), or extracorporeal membrane oxygenation (ECMO) preoperative to postpone the operation which leads to higher survival in post-MI-VSD.

Keywords: Ventricular Septal Rupture, Myocardial Infarction, Optimal Time Repair

1. Introduction

Myocardial infarction (MI) is one of the primary reasons for morbidity and mortality in the world. MI that can occur as a result of ischemic heart disease or coronary artery disease in conjunction, and when an atherosclerotic plaque ruptures and a developing thrombus obstructs the coronary artery completely or partly, limiting blood flow to the heart it will be manifest [1, 2]. In this case, the primary percutaneous

coronary intervention (PCI) of the coronary artery is generally provided by inserting a stent and when stents are inadequate, coronary bypass surgery is done by the left internal mammary artery or saphenous vein for revascularization [3]. The disease is diagnosed and evaluated by clinical assessment, the electrocardiogram (ECG), color Doppler Echo and left Heart Catheterization [4].

The infarcted heart shows typical cell death cascades demonstrated by a loss of cells and fibrotic scarring in the myocardium. Cardiac hypertrophy and fibrosis mainly lead to ventricular wall

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thickening and stiffening, which defining an adverse cardiac remodeling which eventually causes impaired cardiac function and consequently hearts failure [5].

1.1. Ventricular Septal Rupture

About 1% of patients with acute myocardial infarction developed with ventricular septal rupture (VSR) [6]. This mechanical complication generally happens between 10 to 14 days after MI when necrotic tissue is most frequent and the collateral coronary circulation is not well improved [7]. VSR has a mortality rate of 87% within 2 months in patients who treated with medications [8]. Therefore, VSR is fatal unless when it is corrected by surgery. The presentation of septal rupture complicating anterior myocardial infarctions is somewhat more frequent (60%) than that associated with an inferior location of myocardial infarction (40%). Ruptures associated with anterior MI are characteristically localized in the apical part of the septum and are simple. While the ruptures associated with inferior MI is characteristically localized in the basal septum and these are usually complex with wide intramural rupture diffusion in diverse directions, with an important injury of the right ventricle and mitral regurgitation as a result of papillary muscle dysfunction. Also, inferior myocardial infarct showed more preoperative occurrence of cardiogenic shock anterior myocardial infarction [6, 9].

1.2. Pathophysiology of VSR

The time interval between an MI till the appearance of VSR has a higher incidence in the first 24 h after the beginning of infarction and on day 3 to 4. It progresses infrequently at more than 2 weeks after an MI [10].

VSR improves after full-thickness infarction of the ventricular septum and can happen at any anatomic location. Ventricular septal rupture appears to happen with a comparable incidence in anterior and inferior or lateral infarctions. Anterior infarctions are more probable to cause apical flaws and inferior or lateral infarctions are more probable to cause basal flaws at the connection of the septum and the posterior wall [11]. The conventional mechanism of septal rupture is coagulation necrosis of ischemic tissue with neutrophilic infiltration, finally leading to thinning and weakening of the septal myocardium [10].

The first days (3 to 5) after the incidence of a VSR is characterized by the development of the myocardium coagulation necrosis develop along

Abbreviations

VSR	Ventricular septal rupture
MI	myocardial infarction
ECG	electrocardiogram
IABP	Intra-aortic balloon pump
ECMO	extracorporeal membrane oxygenation
PCI	Percutaneous coronary intervention
ACCF/	American College of Cardiology Foundation and
AHA	and American Heart Association

with the infiltration of neutrophilic. The dissection of an intramural hemorrhage into ischemic myocardial tissue is probably leads to the occurrence of VSR in the first 24 h after the onset of MI. The surrounding tissue may be retracted subsequently and enlarge defect in the following weeks [12].

In the initial phases of rupture development, the typical clinical manifestation of VSR is the abrupt progress of cardiogenic shock. The median time interval from MI to VSR reports 16 h by shock trial registry [13].

The shunting of blood from the left ventricle to the right ventricle is caused by VSR. The right ventricle is regularly evolved in the infarct and suffers the extra burden of an increased volume load. Obviously, the rupture size will affect the shunt size, however, physiological factors affect hemodynamic results. The systemic vascular resistance increases will increase and the pulmonary vascular resistance increases will decrease the shunt [14].

1.3. Types of Ventricular Septal Rupture

There are two types of ventricular rupture: simple and complex. Simple ruptures have a straight connection between the left and right ventricles, happening at the same level in both chambers, without gross hemorrhage or tear. The simple SVR is more prevalent after anterior myocardial infarction. A complex rupture is an interventricular communication with a tortuous route, with a tract that might spread into areas far from the acute myocardial infarction site, and with hemorrhage and disruption of myocardial tissue and it is more prevalent after inferior myocardial infarction [15].

1.4. The Diagnosis of VSR

The VSR is diagnosed when a patient's condition worsens after myocardial infarction, and the symptoms such as dyspnea, cardiogenic shock, and chest pain increases [16]. Mitral valve regurgitation

is the most important differential diagnosis that is resulted from papillary muscle rupture. The acute papillary muscle rupture has the loudest murmur at the apex and has a diastolic component. The ECG will show an anterior or inferior infarction [17]. Chest x-ray probably shows a mixture of findings such as cardiomegaly, increased pulmonary vascular marking, pleural effusions, and interstitial fluid, however, they are not specific for VSR. Two-dimensional echocardiographic imaging with color flow Doppler is the main method of diagnosis for VSR. It determines the septal rupture from papillary muscle rupture, as well as the size and site of the rupture, and amount of injury in the right and left ventricular, also the size of the shunt size [18].

1.5. Treatment of VSR

If systolic blood pressure is above 90 mmHg, vasodilator therapy involving nitroglycerine or nitroprusside must be started at the earliest time. To support sufficient cardiac output, Inotropes may be required. These interventions are important to stabilize the patient's condition in preparation for further repair. If pharmacological therapy is not effective, Intra-aortic balloon pump (IABP) should be used [19, 20].

Surgical techniques are comprised Excision of Infarct and reconstruction of the ventricular septum and free walls of the heart by Dacron patches [9]. Others techniques exclude infarct of the left ventricle using an endocardial patch, without injury to the right ventricle [21]. The classic surgical opening to the septum has been a ventriculotomy via the infarcted part. Due to poor access and the technical complications of restoring the fragile septum, surgeons hardly approach this injury via the right atrium (19).

The American College of Cardiology Foundation and American Heart Association (ACCF/AHA) guidelines suggested urgent surgery to repair VSR after acute MI regardless of hemodynamic stability at the time of diagnosis [22].

According to the result of the studies, it seems that the time between VSR detection and its repair is a determining factor in the survival or mortality of patients in post-myocardial infarction VSR. In spite of the agreement of experts on the necessity of surgical repair, the timing of VSR repair management remains unclear. Therefore, the aim of this review is to evaluate the optimal time repair for VSR after MI.

2. Materials and methods

To collect the data, Pubmed, EMBASE, and Cochrane Central Registry databases were searched for potentially suitable studies. Articles published between the earliest possible times until the year 2019 were searched and 60 articles were selected. Search terms were including Ventricular Septal Rupture, Myocardial Infarction, Timing, and MI. The abstracts were reviewed first and the full text of those who met the criteria was reviewed to be used for the study.

3. Results

In Tang et al. study in 2015, the optimal timing and technique of transcatheter closure as a less invasive method for VSR has been evaluated. The average time between VSR diagnosis and transcatheter closure was eighteen days. In 10 out of 11 individuals the device closure was deployed effectively. Eight patients survived during the follow-up but 3 patients died within 7 days after the procedure. A follow-up displayed no remaining shunt in 3 patients and a small residual shunt in 5 patients. Therefore, they demonstrated that transcatheter closure of after VSR is possible and effective and it can be deferred to the late phase, more than 2 to 3 weeks after the diagnosis of VSR if the clinical conditions allow [23].

In a study by Di Summa et al. the average time from AMI to VSR was 5.24 days. The average time from VSR to the operation was 10 ± 17.7 days. The hospital mortality rate was 23.5%. Patients operated on very early (1 ± 1.41 days) had significantly poor results with a mortality rate of 87.5% while those operated within 1 week, had a mortality rate of 44.4%, and those operated later showed 0% mortality. They suggested immediate cardiac support such as Intra-aortic balloon pump (IABP) or ventricular assist device to stabilize the hemodynamic status before surgery [24].

Papalexopoulou et al. (2013) evaluated 6 studies including 3238 patients undergoing surgery for post-infarct ventricular septal rupture (PIVSR). According to their result early surgery, from >3 days to within 4 weeks post-myocardial infarction, showed in-hospital mortality of 52.4% and delayed surgery, from 1 week to after 4 weeks after MI, showed in-hospital mortality of 7.56%. The exact timing of surgery is related to patients' hemodynamic status. If the diameter of infarction in more than 15 mm with a significant shunt and subsequent hemodynamic deterioration, patients with VSR should undergo early surgical repair. If life expectancy is

strictly limited by another underlying pathology, surgery should be inhibited. If the patient is in cardiogenic shock immediate surgery should follow resuscitation measures and cardiac support. If the patient has a stable hemodynamic situation, surgery could be done after 3-4 weeks. If there is clinical deterioration, immediate surgery is recommended [25].

Dalrymple-Hay et al. study demonstrated an operative mortality rate of 31% in early repaired VSR patients (73% on the day of rupture and 16% in 2 days) and 0% in patients repaired after 4 weeks. They believe that surgery should be offered to almost all patients with VSR, however, hemodynamically stable patients can have delayed surgery, which results in higher survival [26].

Dawson et al. found that the insertion of an Amplatzer occluder device in patients with a post-infarction ventricular septal rupture is not amenable to surgical repair. Patients with cardiogenic shock commonly have an unfavorable result and closure should be used with caution. Patients had a better result if the intervention is deferred by 2 weeks or maybe as a result of the maturation of the VSD and recovery of myocardial function. In some conditions, device closure may be complicated by device dislocation or embolization, remaining shunting, etc. In these cases, surgical repair is the only choice. In patients who underwent surgical repair directly without percutaneous closure attempt, the mortality is 43%, and similar to percutaneous closure, there is a relation between those operated within 7 days of the VSR happening and those more than this time. An increased risk of death was seen in patients who presented cardiogenic shock and there was a significant decrease in operative mortality if the timing of myocardial infarction to VSR closure could be deferred by three weeks. Percutaneous closure may evade the need for surgical closure in VSR. Although, in some cases, it provides time to allow the VSR to mature and the patient to be stabilized and be prepared for the surgery to offer the finest probable result for the patient [27].

Malhotra et al., in a study on 55 patients with post MI-VSR, observed that 76% of patients died if the surgery was done <3 days from the incidence of VSR whereas only 26% died when surgery was done >3 days after incidence of VSR. They suggest that, if they could postpone the surgery for >3 days after the incidence of VSR, the mortality rate decreased by 50% and if they could postpone it for 4-6 weeks, it more reduced to nearly 0. Therefore, they believed that the time between VSR and Surgery seems to be the most important interval since it indicates the time when the heart and various body systems get

to adapt to the hemodynamic consequences of abrupt left to right shunt. A short time between MI and VSR (MI to VSR + VSR to Surgery) is indicative of severe ischemia and inaccessibility of collateral circulation. A long time between MI and surgery recommends the heart is well collateralized and systems have had enough time to adjust to changed hemodynamics. They mentioned that the institution of Intra-aortic balloon pump (IABP) before surgery can stabilize the patients by reducing the afterload of the left ventricle and recovers coronary circulation [28].

In Killen et al. study (1997), it was demonstrated that in the entire VSR patients (76 patients), there was a reduction in operative mortality associated with the delayed repair. In patients who experienced repair in 24 hours, the operative mortality amount was 51.4%, in those who underwent an operation in 1-5 days, the mortality rate was 47.1%, when the operation was performed within 6-20 days after VSR, it was 28.6%, and when patients underwent repair in 1 month, no deaths were observed. For all patients, the hospital mortality was 40.8%; survival was 41.5% and 25.6% at 5 years and 10 years, postoperatively [29].

Bouchart et al. evaluated 67 VSR patients. According to their results, the operative mortality rate was 25%. The chief reason for death was cardiac failure. Issues affecting early deaths were preoperative hemodynamic status including cardiogenic shock, the location of the MI (posterior more than), the interval between infarction and surgery (less than 1 week was 33% and more than 1 week was 6.2%). The survival rates at 1 year were 74.6% \pm 5.3% and at 5 years was 66.2% \pm 6.2% [30].

In a case study by Estrada-Quintero et al. the time of surgical repair for VSR postponed by 36 days intra-aortic balloon pump which helped to preoperative hemodynamic management and the patients were discharged after 45 days [31].

Mantovani et al. study showed that the median time between VSR and repair was two days. The mortality rate in patients who had surgery in the first three days from VSR was 52% and those who operated after that had 11% mortality rate. Mortality rate was associated with emergency operation. However, the authors support early surgery in all patients [32].

Cinq-Mars et al. evaluated 34 patients with post MI-VSR. The 30 days of operative mortality was 65%. The multivariate analysis defined older age and shorter time between MI and surgery as independent predictors of 30-days and long term mortality. The mean time interval between MI and VSR was 7.21 days vs. 1.79 days in survivors and non-

Table 1. The timing of repair and the Mortality Rate in post-MI VSR patients.

Study	Type of Repair	Timing of Repair	Mortality rate
Killen et al., 1997 [29]	Surgery	24 h	51.4%
Di Summa et al., 1997 [24]	Surgery	1-5 days	47.1%
		1 ± 1.41 days	87.5%
		within 1 week	44.4%
Bouchart et al., 1998 [32]	Surgery	later	0%
		Less than 1 week	33%
Dalrymple-Hay et al., 1998 [26]	Surgery	More than 1 week	6.2%
		First day	73%
Cerin et al., 2003 [36]	Surgery	After 2 days	16%
		After 4 weeks	0%
		Within the first week	75%
Mantovani et al., 2006 [32]	Surgery	After 3 weeks	16%
		Within 3 days	52%
Coskun et al., 2009 [37]	Surgery	After 3 days	11%
		Within 3 days	100%
Arnaoutakis et al., 2012 [34]	Surgery	After 36 days	0%
		≤7 days	54.1%
Papalexopoulou et al., 2013 [25]	Surgery	>7 days	18.4%
		from >3 days to within 4 weeks	52.4%
		from 1 week to after 4 weeks	7.56%
Trivedi et al., 2015 [35]	Percutaneous closure Surgery	<21 days	Significantly more for Percutaneous closure
		>21 days	
Cinq-Mars et al., 2016 [33]	Surgery	2.74 days	Not Survived
		4.44 days	Survived
Malhotra et al., 2017 [28]	Surgery	<3 days	76%
		>3 days	26%

survivors, respectively. The mean time interval between VSR and operation was 4.44 days vs. 2.74 days in survivors and non-survivors, respectively, and the mean time interval between MI and operation was 11.65 days vs. 4.4 days in survivors and non-survivors, respectively [33].

The Arnaoutakis et al. study in 2012 showed that the general operative mortality rate was 42.9%. They showed a non-linear time trend for operative mortality. If the repair was done ≤7 days from MI, the operative mortality rate was 54.1%. If the operation performed >7 days from MI, the rate was 18.4%. The highest rate was among patients who underwent VSD repair within 6 h from MI. The main causes of death were cardiac, followed by pulmonary, neurologic, infectious, and renal complications. The older age, female sex, and higher serum creatinine were related to 30-day mortality. Longer cardiopulmonary bypass time was related to higher mortality [34].

Trivedi et al. (2015) reported that patients who experienced early VSD closure (<21 days after MI) had greater amounts of residual shunt and mortality, regardless of closure strategy. They also showed that the mortality was higher after early percutaneous closure, and not after early surgery [35].

In Cerin et al. work, the diagnosis of VSR happened after an average time of 4 ± 3 days after

MI and the average time from MI to surgery was 14 ± 12 days. The operative mortality rate was 52% and it was related to a shorter time from MI to surgery, cardiogenic shock, high systolic pulmonary artery pressure, and the diameter of VSR diameter. In patients who had an operation in the first week after MI was 75%, while in patients operated on 3 weeks after MI it was 16%. They suggested a delay in surgery by stabilizing the status of the patients by IABP and medical therapy [36].

In Coskun et al. study the time between MI to VSR was 8.7 days, and between to VSR and rupture to surgery was 23.1 days. Hospital mortality rate was 32% and a shorter time interval of MI and operation was associated with survival. The mortality rate in those operated within 3 days was 100% and in those operated after 36 days, it was 0%. Therefore, the authors recommended stabilization and later surgery unless if the patient is in cardiogenic shock [37] (Table 1).

4. Discussion

According to the result of the studies, it seems that the time between VSR detection and its repair is a determining factor in the survival or mortality of patients in post-myocardial infarction VSR. Studies showed that earlier repair and operation in VSR

patients increases the risk of mortality. It could be clarified by the fact that unstable hemodynamics and ischemia-reperfusion damage to an infarcted myocardium increases the injury of the myocardium [28]. Time is needed to restore heart muscle tissues in the necrotic part. In the early phase after MI, infarcted myocardium is fragile, and it is hard for the surgeon to work around the septal rupture properly and increases the risk of laceration and recurrent septal defects [38]. A short interval between MI and VSR is indicative of severe ischemia and inaccessibility of collateral circulation [28].

The advantages of late operation are the improvement of the infarcted parts of the myocardium, facilitation of surgical repair, and reduction of the risk of recurrent or residual defects [39]. The longer time between MI and VSR indicates the time when the heart and different body systems need to adapt to the hemodynamic results of the abrupt left to right shunt. It suggests enough time for collateralization of heart and adaptation of systems to changed hemodynamics [28].

The American Heart Association suggests an immediate closure of the rupture in all patients [22]. The European Society of Cardiology guidelines mentions that there is no agreement among experts about the optimal time of the closure [40]. It seems that the finest time for the operation is after fibrotic healing of the necrotic muscle. Although, in a histologic study it was revealed that proliferation of connective tissue was not present until the third week after infarction [41]. Moreover, in a large number of patients, it is not possible to delay the operation since they are at risk of severe heart failure and organ dysfunction [42]. In these cases operation immediately after diagnosis of VSR is logical to prevent further hemodynamic deterioration. In hemodynamically compromised patients, it may be considered to use a ventricular assist device, IABP, or ECMO preoperative to postpone the operation which leads to higher survival in post-MI-VSD [42, 43]. The IABP is useful in diminishing the afterload of the left ventricle and develops coronary perfusion, but not in all patients [42].

Author contribution

Ibrahim Shafiei: Conception; Data collection and/or processing; Literature review; Writer; Critical review.

Fatemeh Jannati: Fundings; Materials; Data collection and/or processing; Analysis and/or interpretation.

Mansour Jannati: Conception; Design; Supervision; Critical review.

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