OUTCOMES OF SURGICAL REPAIR OF POST-INFARCTION VENTRICULAR SEPTAL RUPTURE – A RETROSPECTIVE STUDY

Dr. Hema Krishna Sai Kadiyala

MCh CARDIOVASCULAR & THORACIC SURGERY

THESIS YEAR:2021-2023

SREE CHITRA TIRUNAL INSTITUTE FOR MEDICAL SCIENCES AND TECHNOLOGY, TRIVANDRUM

An Institution of National Importance established by an Act of the Indian Parliament (Act No.52 of 1980)

Dept. of Science and Technology,
Govt. of India

www.sctimst.ac.in

OUTCOMES OF SURGICAL REPAIR OF POST-INFARCTION VENTRICULAR SEPTAL RUPTURE – A RETROSPECTIVE STUDY

A THESIS SUBMITTED BY

Dr. Hema Krishna Sai Kadiyala

TO

SREE CHITRA TIRUNAL INSTITUTE FOR MEDICAL SCIENCES

AND TECHNOLOGY, TRIVANDRUM.

IN PARTIAL FULFILMENT OF THE

REQUIREMENTS FOR THE AWARD OF

MCh CARDIOVASCULAR & THORACIC SURGERY

YEAR: 2021-2023

श्री चित्रा तिरुनाल आयुर्विज्ञान और प्रौद्योगिकी संस्थान, त्रिवेन्द्रम
तिरुवनन्तपुरम - ६९५०११, केरल, इंडिया
SREE CHITRA TIRUNAL INSTITUTE FOR MEDICAL SCIENCES AND TECHNOLOGY, TRIVANDRUM
Thiruvananthapuram - 695 011, Kerala, India
(An Institute of National Importance under Govt. of India)
Grams : Chitramet, Phone : +91-471-2443152, Fax : +91-471-2550728 / 2446433, E-mail : sct@sctimst.ac.in, Website : www.sctimst.ac.in

DECLARATION BY THE STUDENT

CERTIFICATE

I, Dr. Hema Krishna Sai Kadiyala, hereby certify that I had personally carriedout the work depicted in the thesis titled,

"Outcomes of Surgical Repair of Post-Infarction Ventricular Septal Rupture – A Retrospective Study",

No part of this thesis has been submitted for the award of any other degree or diploma prior to this date.

Signature

Dr. Hema Krishna Sai Kadiyala

Date: 13.11.2023

श्री चित्रा तिरुनाल आयुर्विज्ञान और प्रौद्योगिकी संस्थान, त्रिवेन्द्रम
तिरुवनन्तपुरम - ६९५०११, केरल, इंडिया
SREE CHITRA TIRUNAL INSTITUTE FOR MEDICAL SCIENCES AND TECHNOLOGY, TRIVANDRUM
Thiruvananthapuram - 695 011, Kerala, India
(An Institute of National Importance under Govt. of India)
Grams : Chitramet, Phone : +91-471-2443152, Fax : +91-471-2550728 / 2446433, E-mail : sct@sctimst.ac.in, Website : www.sctimst.ac.in

CERTIFICATE BY THE RESEARCH GUIDE

Name of the Guide: Dr. Bineesh K R

Department: Department of Cardiovascular and thoracic surgery.

This is to certify that, Dr.Hema Krishna Sai Kadiyala, senior resident in the department of Cardiovascular and thoracic surgery, of this institute has fulfilled the requirements prescribed for the MCh degree of the Sree Chitra Tirunal Institute for Medical Sciences and Technology, Trivandrum.

The thesis entitled, "Outcomes of Surgical Repair of Post-Infarction Ventricular Septal Rupture – A Retrospective Study" was carried out under my direct supervision. No part of the thesis was submitted for the award of any degree or diploma prior to this date.

Clearance was obtained from the Institutional Ethics Committee for carrying out the study.

Signature

Dr. Bineesh K R
Additional Professor
Department of Cardiovascular and thoracic surgery
SCTIMST

Date: 13. 11. 2023

ACKNOWLEDGEMENT

Writing this thesis has been fascinating and extremely rewarding. I would like to thank a number of people who have contributed to the final result in many different ways.

An ostentatious use of words will not be sufficient to express my heartiest thanks to my guide **Dr. Bineesh K R** Additional Professor, Department of Cardiovascular and Thoracic Surgery, SCTIMST, Thiruvananthapuram. Their valuable and critical suggestions, constant inspiration and encouragement, instinctive corrective measures, timeless efforts, unstinted cooperation and mental support throughout have made this marathon task a smooth journey.

I will always remain obliged and grateful to my senior professor **Dr. Baiju S Dharan** and Professor & HOD **Dr. Vivek Pillai** for his inspiration and support. I have to mention thanks to my Consultants **Dr. Vargese T Panikar, Dr. Sabarinath Menon, Dr. Sudip Datta Baruah, Dr. Sowmya Remanan, and Dr. Shivanesan Pitchai.** who have been instrumental and constantly supported me during the course.

I would like to express my gratitude to my colleagues **Dr. Kaushik, Dr. Pravin , Dr. Anshuman V, Dr. Deepak, Dr. Pruthvi, Dr. Zarin, Dr. Sagar, Dr. Manish C and Dr. Jyothi** for their support throughout.

Sincere thanks to all other staff of the Department of Cardiovascular and Thoracic Surgery for their understanding, inspiration, and affectionate encouragement right from the budding stage of this work.

I take this opportunity to thank my seniors, and my friends for their continuous help and support which gave strength to my thoughts and mapped my way to find methodology for myself.

Sometimes it is not easy to express in words especially when you have to say thanks to your parents for their constant undemanding love, dedication, sacrifice, inspiring guidance, affectionate encouragement and never- ending enthusiasm, without which this study would not have seen the light of the day.

I would like to especially thank my entire Family for their constant support and care, encouragement and for being a pillar of strength throughout the duration of this study, without whom I would never been able to complete this endeavor.

My sincere gratitude to all patients who are the part of my study. Last, but not the least, I thank the **almighty God** for being the silent force behind everything.

Dr. Hema Krishna Sai Kadiyala

Senior Resident,
Department of Cardiovascular and Thoracic Surgery.
SCTIMST

Table of Contents

LIST OF FIGURES

LIST OF TABLES

LIST OF ABBREVIATIONS

SR NO	ABBREVIATION	FULL FORM
1)	**ACC**	Aortic Cross Clamp
2)	**ACCF/AHA**	American College of Cardiology Foundation/American Heart Association
3)	**AF**	Atrial Fibrillation
4)	**AKI**	Acute Kidney Injury
5)	**AMI**	Acute Myocardial Infarction
6)	**APEX AMI**	Assessment of Pexelizumab in Acute Myocardial Infarction
7)	**AWMI**	Anterior wall myocardial infarction
8)	**BUN**	Blood Urea Nitrogen
9)	**CABG**	Coronary Artery Bypass Grafting
10)	**CAD**	Coronary Artery Disease
11)	**CPB**	Cardiopulmonary Bypass
12)	**ECMO**	Extracorporeal Membrane Oxygenation
13)	**EHA**	European Heart Association

14)	**GUSTO I**	Global Utilisation of Streptokinase and TPA for Occluded Coronary Arteries trial
15)	**IABP**	Intra-Aortic Balloon Pump
16)	**ICU**	Intensive Care Unit
17)	**IWMI**	Inferior wall myocardial infarction
18)	**LCOS**	Low Cardiac Output Syndrome
19)	**LV**	Left Ventricle
20)	**LVEF**	Left Ventricular Ejection Fraction
21)	**MI**	Myocardial Infarction
22)	**MCS**	Mechanical Circulatory Support
23)	**NYHA**	New York Heart Association
24)	**PA**	Pulmonary Artery
25)	**PCI**	Percutaneous Coronary Intervention
26)	**PIVSR**	Postinfarction ventricular septal rupture
27)	**RV**	Right Ventricle
28)	**TPA**	Tissue Plasminogen Activator

29)	**VSD**	Ventricular Septal Defect
30)	**VSR**	Ventricular Septal Rupture
31)	**VT**	Ventricular Tachycardia

SYNOPSIS

Background:

Ventricular septal rupture (VSR) is a significant risk to patients following acute myocardial infarction, with high mortality rates. Despite the challenges and high risk of complications, surgical repair remains the preferred treatment option. VSR patients are particularly vulnerable due to right ventricular involvement, friable tissue, and complex rupture. Postoperative management aims to reduce left-to-right shunt and improve coronary blood flow.

Aim:

To study the early outcomes and survival rate after surgical repair of post-infraction ventricular septal rupture.

Methods:

This retrospective, observational study was conducted in the Cardiovascular and Thoracic Surgery Department between 2011-2021 on 23 patients who underwent Post Infarction Ventricular Septal Rupture. After obtaining permission from the IEC, Preoperative, intra-operative and post-operative data was collected, and the principal investigator and co-investigators performed retrospective data analysis. After the procedure, the principal investigator kept the data and patient details confidential. Data was obtained from the institutional electronic medical records. Data collection was done, and details of the patient information such as age, gender, preoperative parameters, co-morbidities, intra-operative parameters and postoperative parameters, along with follow-up, were compiled. Statistical analysis using IBM Statistical Package for Social Science (Statistics for Windows, version 21.0, Armonk, NY: IBM Corp.) was done, and the data was compared by applying statistical tests to find the statistical significance of the results.

Results:

Cardiogenic shock at the time of VSR repair, preoperative requirment of inotropic supports significantly influenced survival rates. Type of MI, preoperative ventricular function and multi-vessel coronary artery disease did not show any impact on survival. Even though it is an ongoing debate , we have noted that delayed VSR repair had better outcomes than early repair and coronary revascularisation(CABG) during VSR repair, increased survival rates . Cardiopulmonary bypass (CPB) and aortic cross-clamp (ACC) time did not differ significantly between survival and non-survival groups. However, using an IABP to wean off CPB was more common in the non-survival group. Survivors have shorter ventilation time compared to non-survivors , longer mechanical ventilation was associated with higher mortality. Patients with postoperative severe ventricular dysfunction were noted to have higher mortality risk. Arrhythmias, such as atrial fibrillation and ventricular tachycardia, did not strongly differentiate between survivors and non-survivors . On the other hand we have noted that patients needing renal replacement therapy postoperatively had bad prognosis but acute kidney injury alone had no significance value in determining mortality. Residual VSR after repair also was not statistically significant.

We also noted that out of the 13 survivors who were discharged after VSR repair , 3 patients lost follow up. The remaining 10 patients survived at the end of 1 year post discharge.

Conclusion:

The study found that pre-operative cardiogenic shock and inotropic support were critical factors influencing outcomes in patients undergoing VSR repair. Early surgery was associated with increased mortality, while delayed surgery offered better outcomes. It is also important to note that coronary revascularisation along with VSR repair gave better outcomes. Post-operative ventilation time, ventricular function and renal replacement therapy significantly impacted survival rates. The study emphasizes the importance of considering pre-operative, intra-operative, and post-operative factors in assessing patient outcomes. Further multi-centre studies with larger study population are required to provide further understanding.

Keywords:

Ventricular septal rupture, Post-infraction, Ventricular septal rupture, Survival rates, Cardiogenic shock, Atrial fibrillation, Ventricular tachycardias

SREE CHITRA TIRUNAL INSTITUTE FOR MEDICAL SCIENCES

AND TECHNOLOGY, TRIVANDRUM

An Institution of National Importance established by an Act of the Indian

Parliament (Act No.52 of 1980)

Dept. of Science and Technology,
Govt. of India

www.sctimst.ac.in

1. INTRODUCTION

1.1 General Introduction

Ventricular septal rupture (VSR) is infrequent, yet it poses a significant risk to the patient's life following an episode of acute myocardial infarction. Despite the inherent difficulties and elevated mortality rates associated with surgical correction, it continues to be regarded as the preferred treatment option (Matteucci et al., 2021). The mortality rate among patients afflicted with this complication continues to be elevated. Without surgical intervention, the mortality rate during the initial two-week period varies between 60% and 70%, with a subsequent increase to approximately 90% at the end of three months. Conversely, when surgical intervention is employed, mortality rates range from 19% to 66% (Naik et al., 2022). Despite the decline in VSR cases due to the introduction acute reperfusion strategies, this mechanical complication following acute myocardial infarction still indicates a grave prognosis (Elbadawi et al., 2019). Patients with VSR are a particularly vulnerable subset of those with an AMI. Clinical and anatomical challenges include right ventricular involvement (functional or anatomical), friable tissue around the infarct area, the complex nature of the rupture and its expansion over time, and associated hemodynamic instability that often leads to cardiogenic shock. Rupture of the ventricular septum can affect any septum region and typically follows a transmural infarction. In the hours or days following VSR, most patients develop unpredictable hemodynamics, and reports of long-term survival without corrective interventions are extremely rare. Medically managed VSR patients had a mortality rate of 94% in the multicenter Global Utilisation of Streptokinase and TPA for Occluded Coronary Arteries trial (Crenshaw et al., 2000) and a mortality rate of 96% in the SHOCK registry (Menon et al., 2000), both of which are thought to be close to the disease's natural course. Therefore, surgical repair is the recommended method of treatment. Despite these advances, surgery is still a complex procedure with a high risk of complications. This systematic review summarises the existing literature on the nature and results of VSR repair.

The best time to perform surgery is a topic of debate. It has been found that patients who wait longer before undergoing repair have a higher chance of survival (Arnaoutakis et al., 2012). VSR repair may be easier after an infarct has healed and scar tissue has formed, which may explain why postponing surgery improves outcomes. Nonetheless, many patients present with hemodynamic instability, necessitating and often receiving prompt intervention. Acutely, infarcted myocardium is fragile and prone to tearing and postoperative residual shunt because it does not hold sutures well. Patient status must be stabilised before surgery. Reducing left-to-right shunt in compromised subjects should be a primary goal of management, with afterload reducing agents and IABP insertion employed to that end. In addition to lowering ventricular wall stress and oxygen demand, IABP boosts coronary blood flow. The current meta-analysis found an indirect but significant association between the use of IABP before and during surgery and the risk for operative mortality (Matteucci et al., 2021). The severity of the patient's condition when inserting an IABP could be a contributing factor. The study could not delve deeper into this matter due to a lack of information in the IABP working group (Matteucci et al., 2021). Critical patients with persistent hemodynamic instability due to VSR have seen the emergence of other forms of short-term temporary mechanical circulatory support (MCS) in the past two decades, in addition to IABP. In several published studies, extracorporeal membrane oxygenation has been used to stabilise patients with VSR before surgery (Ariza-Solé et al., 2020). As a bridge to surgery or a transplant, a percutaneous left ventricular assist device like the Impella LP 5.0 (Abiomed, Danvers, MA) can be implanted (La Torre et al., 2011). Preoperative metabolic and hemodynamic stabilisation may improve the patient's condition enough to permit postponing the surgical correction by a few days.

However, the high rate of complications comes at the cost of this advancement (Cheng et al., 2014).VSR surgery has progressed over time. Two of the most popular approaches to repairing VSR were proposed by Daggett et al., 1977 and David & coworkers (David et al., 1995). Daggett and coworkers developed a method to repair VSRs that involves sewing a patch over the hole and attaching it to the ventricles on either side. On the other hand, David and colleagues' method is an infarct exclusion technique in which all sutures are placed in the left ventricle. Despite David and coworkers' method's widespread acclaim, the question of which approach is preferable has yet to be settled. Although studies did not reach statistical significance, we did notice a trend towards lower operative mortality in the infarct exclusion group. Another essential consideration is location of the VSR . The heart needs to be raised for proper exposure during a posterior VSR, and the posterior descending artery and posteromedial papillary muscle are near, all of which present technical challenges (Matteucci et al., 2021). Right ventricular dysfunction, complex ruptures, and challenging repair have all been linked to poor surgical outcomes in cases of posteriorly located VSR (Jeppsson et al., 2005). It is debatable whether CABG performed simultaneously as VSR repair improves patient outcomes (Pang et al., 2013).

Research Question: What are the early outcomes and survival rate after surgical repair of post-infraction ventricular septal rupture?

Hypothesis: Are there any differences in the common attributing factors that affect the early outcome and survival status after post-infraction VSR repair? (H)

Null Hypothesis: Yes, there is a difference in the common attributing factors that affect the early outcome and survival status after post-infraction VSR repair. (H_0)

Alternative Hypothesis: No difference in the common attributing factors that affect the early outcome and survival status after post-infraction VSR repair. (H_a)

1.2 Aim and Objectives-

Aim: To study the early outcomes and survival rate after surgical repair of post-infraction ventricular septal rupture.

Objectives:

1) To review all the patients who underwent surgical repair of post-infraction ventricular septal rupture and study the outcomes and prognostic factors.
2) To analyse the data and enumerate factors contributing to mortality and

morbidity.

1.3 Scope of the Study-

In recent literature, the incidence of ventricular septal rupture (VSR) due to acute myocardial infarction (AMI) was reported to be between 0.17 and 0.21 per cent (French et al., 2010). Although overall mortality for patients with AMI has decreased significantly over the past two decades, the prognosis for subjects who develop VSR remains dismal (Elbadawi et al., 2019). Due to the lacklustre response to medical management, VSR is best treated via surgical intervention. However, due to hemodynamic instability and tissue fragility, surgical repair results are often suboptimal and associated with high mortality. Single-centre experiences with small sample sizes and limited information regarding predictors on management and outcome are typical of published series on surgical outcomes for VSR due to the rarity of this condition. We are conducting this study to provide an up-to-date perspective and early postoperative results of the surgical management of VSR.

2. LITERATURE REVIEW

Introduction:

In 1847, Latham (1845) first documented the clinical presentation of ventricular septal rupture (VSR). The interventricular septum is responsible for partitioning the ventricular chamber into distinct right and left ventricles.

The interventricular septum comprises two distinct parts: the muscular and the membranous components. The muscular portion, which constitutes the bulk of the septum, is located inferiorly to the membranous part and exhibits substantial thickness. It originates from the bulboventricular flange. Conversely, the membranous part constitutes a smaller segment of the septum, positioned superiorly to the muscular counterpart, and displays a thinner structure. Its development arises from neural crest cells.

Ventricular septal rupture (VSR) represents a rare yet life-threatening complication that can emerge following acute myocardial infarction. In contemporary medical practice, the incidence of this condition has diminished owing to a proactive approach centred on early reperfusion therapy. Nevertheless, the associated mortality rate remains high (Cooley et al., 1957; Payne et al., 1963). VSR can manifest at any location along the interventricular septum, and the size of the rupture significantly influences the prognosis. Favourable outcomes are typically associated with small ruptures and hemodynamically stable patients.

VSR occurs predominantly within the first week following acute myocardial infarction (Daggett, 1982). In most instances, this is accompanied by an immediate deterioration in hemodynamic parameters, often leading to the development of cardiogenic shock. Notably, VSR constitutes a surgical emergency necessitating prompt intervention in symptomatic patients. The requisite procedure involves the closure of the VSR in conjunction with coronary artery bypass grafting. Surgery, in almost all cases, follows a transinfarct approach. Using prosthetic materials is customary to affect the closure of the septum and the ventricular wall, thereby averting undue tension. Over time, advancements in surgical techniques and enhanced support from pharmacological and mechanical means have contributed to achieving favourable clinical outcomes.

Postinfarction ventricular septal rupture (PIVSR) is a rare but life-threatening complication that typically occurs 1–14 days after acute myocardial infarction (AMI), with two distinct peak periods at 24 hours and 3–5 days after AMI (Wilson and Horlick, 2016). With the advent of reperfusion therapy, the incidence of PIVSR has decreased from 1-3% to the current range of 0.2-0.5% (Moreyra et al., 2010). Despite various treatment options, such as cardiac assist devices, surgical procedures, and interventional closures, the mortality rate of PIVSR remains high. Studies have indicated that the survival rate of PIVSR without surgery after one month is less than 10%, emphasizing the significant improvement in prognosis associated with surgical intervention (Crenshaw et al., 2000).

As for the timing of surgical treatment, the American College of Cardiology Foundation/American Heart Association (ACCF/AHA) guidelines suggest immediate surgical intervention for PIVSR patients, regardless of their hemodynamic status (Levine et al., 2016). However, this approach carries risks, including fragile myocardial tissue bleeding and residual shunting. Therefore, some scholars propose delayed surgery when the patient's condition allows it. The 2017 European Heart Association (EHA) guidelines for managing acute myocardial infarction also recommend considering delayed surgical treatment for PIVSR patients with stable hemodynamics after active treatment (Ibanez et al., 2018).

The current study must identify the common factors affecting the early outcome and survival status after post-infraction VSR repair.

Historical Note:

The inaugural successful surgical correction of a post-infarction ventricular septal rupture occurred in 1956 when Cooley (1957) performed the procedure on a patient diagnosed with septal rupture nine weeks earlier. The surgical approach was a right ventriculotomy, similar to those used for patients with congenital ventricular septal ruptures. Subsequently, due to the elevated surgical mortality rates, the prevailing strategy was to restrict the operation to patients who survived more than one month following acute septal perforation. This delay in surgery also offered the benefit of allowing the septal tissue to heal, facilitating a more secure closure of the septal rupture.

Heimbecker (1968) introduced an alternative method in which septal rupture repair was achieved through a left ventriculotomy within the infarcted area, encompassing infarctectomy and aneurysmectomy. This approach provided improved exposure of the apical and inferior septum compared to the right ventriculotomy. Furthermore, it avoided unnecessary damage to the vital right ventricle and allowed for surgical modification of the infarcted section of the left ventricle.

In 1977, Daggett and colleagues (1970) reported a substantial series of 43 patients who demonstrated enhanced surgical outcomes through a combination of infarctectomy and prosthetic patch material. These improved results were seen in a general sense and in the successful treatment of inferoposterior septal ruptures that had previously posed challenges.

In 1995, David (1995) documented 44 patients treated with infarct-excluding patches, resulting in an excellent mortality rate of 19%. Remarkably, this approach also displayed an unusually low incidence of right ventricular dysfunction in just two out of the 44 patients.

Presently, percutaneous devices designed for the closure of septal ruptures are undergoing continuous development and are becoming increasingly relevant for specific patient populations (Calvert et al., 2014). In addition, ventricular replacement therapy is emerging as a viable option for patients with substantial ventricular damage and an inability to sustain adequate end-organ perfusion within the context of septal ruptures (Samuels et al., 2003).

Morphology

Postinfarction ventricular septal rupture (VSR) is typically found in the anterior or apical region of the ventricular septum, accounting for approximately 60% of cases. This primarily results from a transmural anterior myocardial infarction (MI). About 20% to 40% of patients exhibit a VSR in the posterior part of the ventricular septum, which often occurs following an inferior MI. VSRs generally develop as a complication of the first acute MI (Mann and Roberts, 1987; Moore et al., 1986; Skehan et al., 1989)

Notably, well-established collateral coronary circulation is rare in hearts with postinfarction VSR. These ruptures are commonly associated with the complete occlusion of a coronary artery rather than severe stenosis, most frequently involving the left anterior descending coronary artery. In many cases, significant stenoses can also be present in the right coronary artery system. It's important to recognize that VSRs may occur singly or, more unusually, manifest separately over several days (Mann and Roberts, 1987; Moore et al., 1986; Skehan et al., 1989).

The significance of concomitant right ventricular (RV) infarction in postinfarction VSR patients is increasingly apparent. For some time, RV dysfunction was believed to be a mere consequence of the RV adapting poorly to the sudden rise in pulmonary blood flow due to the VSR. However, evidence suggests that actual infarction of the inferior RV wall, or at the very least severe ischemia in that region, is responsible for this dysfunction (Mann and Roberts, 1987; Moore et al., 1986; Skehan et al., 1989).

In cases of posterior VSR, mitral valve regurgitation may accompany the condition, primarily due to papillary muscle infarction or ischemia. Approximately 40% of patients who survive the initial phase after ventricular septal rupture may experience the formation of an aneurysm in the remaining infarcted septal and adjacent ventricular wall (Mann and Roberts, 1987; Moore et al., 1986; Skehan et al., 1989).

Coronary arteries anatomy :

The coronary arteries are two main distributions (right and left), supplied by three vessels; right coronary artery (RCA), left anterior descending (LAD), and circumflex artery (Cx).

Left main stem (LMS):

The LMS arises from the ostium of the left sinus of Valsalva, travels between the pulmonary trunk anteriorly and the left atrial appendage to the left AV groove, dividing after 1–2cm into LAD, Cx, and occasionally a third artery called ramus.

Left anterior descending (LAD):

The LAD runs down the anterior interventricular groove to the apex of the heart, usually extending round the apex to the posterior interventricular groove and the territory of the PDA. A variable number of diagonals are given off over the anterior surface of the LV, small branches supply the anterior surface of the RV, and superior septals are given off perpendicu- larly to supply the anterior 2/3 of the interventricular septum. The first septal is the largest. Some of the RV branches anastomose with infundibular branches of the proximal RCA

Left Circumflex (LCX):

The LCX originates perpendicular to the LMS and runs medially to the LA appendage for 2–3cm, continuing in the posterior left AV groove to the crux of the heart. In left dominant hearts (5–10%) the LCX turns perpendicularly into the posterior interventricular groove to form the posterior descending artery (PDA). In 85–90% of hearts the PDA arises from the RCA (right domi- nant). About 5% of hearts are co-dominant. A variable number of obtuse marginals (OM) arise from the LCX to supply the posterior LV. They are frequently intramuscular. The first branch of the LCX is the AV nodal artery in 45% which courses round the LA near the AV groove.

Right coronary artery (RCA):

The RCA arises from an ostium in the right sinus of Valsalva, gives off an infundibular branch and then a branch to the SA node early, and runs immediately into the deep right AV groove where it gives off RV branches to the anterior RV wall. The acute marginal is a large branch which crosses the acute margin of the heart to travel to the apex. In right dominant hearts the RCA reaches the crux of the hearts where it turns perpendicular to form the PDA, which runs towards the apex in the posterior interventricular groove. Inferior septals which supply the inferior 1/3 of the interventricular septum arise from the PDA. The AV node artery is given off by the RCA in 55% at the crux. One or more right posterolateral branches (RPLB) supply the LV.

Papillary muscles blood supply:

The anterolateral papillary muscle is vascularized by multiple branches from the anterior descending coronary artery and either the diagonal or a marginal branch of the circumflex vessels. The posteromedial papillary muscle is vascularized by a small supply from the circumflex artery or the right coronary artery. These differences between the vascularization of the papillary muscles explain why the posteromedial papillary muscle is more prone to necrosis and dysfunction than the anterolateral papillary muscle.

Pathophysiology

Postinfarction ventricular septal rupture typically arises from transmural infarction of the interventricular septum, leading to the dissection of ventricular blood into the septal myocardium, forming a ventricular septal rupture (Hutchins, 1979).

Patients with postinfarction septal ruptures often exhibit larger infarcts that affect approximately 26% of the left ventricular wall, in contrast to only 15% in other infarctions (Selzer et al., 1969). Most patients with postinfarction septal ruptures also have single-vessel disease (Daggett et al., 1970). These patients with acute postinfarction septal ruptures tend to experience greater right ventricular infarction than those with other acute infarctions (Selzer et al., 1969).

The interventricular septal rupture may manifest as a large single rupture, a common occurrence with anterior infarctions. Alternatively, there may be multiple ruptures in the septum with a serpiginous course, which is more typical with inferior infarctions leading to ventricular septal ruptures (Selzer et al., 1969).

Given the extensive myocardial infarction in patients with postinfarction ventricular septal ruptures, approximately half of the survivors of the acute phase develop ventricular aneurysms, in contrast to only 12% of acute infarction patients without ventricular septal ruptures developing such aneurysms (Pfeffer and Braunwald, 1990).

Approximately one-third of patients with postinfarction ventricular septal ruptures will experience some degree of mitral regurgitation due to left ventricular dilation and alterations in papillary and annular geometry (Miller et al., 1978).

The blood supply to the septum primarily originates from branches of the left anterior descending coronary artery and the posterior descending coronary artery. In exceptional cases, it may also receive blood from the circumflex artery. The resulting infarction is typically extensive and transmural. Approximately two-thirds of ventricular septal ruptures (VSR) occur in the anterior septal wall, with the remaining third in the inferior or posterior wall. When the latter is involved, it frequently leads to mitral valve insufficiency due to papillary muscle dysfunction or rupture. In autopsy examinations, the responsible coronary artery is often nearly completely occluded, and collateral circulation is generally absent. In rare instances, multiple septal perforations may be observed.

The predominant pathological feature of an infarcted septum is coagulation necrosis, characterized by the dry denaturation of proteins caused by oxygen deprivation due to a loss of blood supply. This process progresses to the thinning and weakening of the septum, typically occurring three to five days following an acute myocardial infarction. However, in some cases, ventricular septal rupture can develop within the first 24 hours of a myocardial infarction, often resulting from the dissection of an intramural hematoma or haemorrhage into the diseased myocardium.

The primary underlying cause of ventricular septum rupture is physical shear stress, particularly at the junction of the infarcted region and the adjacent healthy myocardium. Consequently, ventricular aneurysm, free wall rupture, or papillary muscle rupture are frequently associated with ventricular septum rupture.

Following establishing a new connection between the right and left ventricles due to ventricular septum rupture, oxygenated blood diverts from the high-pressure left ventricle to the low-pressure right ventricle.

The natural progression of VSR following a myocardial infarction is brief, and the prognosis is notably grim, with over 90% of individuals succumbing within the first year. The adverse outlook primarily results from the sudden increase in volume load on both ventricles, which are already compromised by a substantial myocardial infarction. Furthermore, patients may experience concurrent ventricular pseudoaneurysm and mitral valve insufficiency, further impairing ventricular function.

Once the ventricular septal rupture forms, blood shunts from the left ventricle to the right ventricle, resulting in the subsequent development of left or right ventricular heart failure (Hutchins, 1979). This shunting and impaired right or left ventricular contractility can result in low cardiac output, cardiogenic shock, and end-organ malperfusion, progressing to fatality within days to weeks (Hutchins, 1979).

In patients with postinfarction ventricular septal rupture, the loss of left ventricular muscle is substantial, to the extent that one-third or more of these patients may not have a survivable amount of remaining myocardium without ventricular replacement therapy (Berger et al., 1993). Ultimately, heart failure and shock are the primary causes of death, with right ventricular failure more common in cases of inferior infarction due to extensive right ventricular infarction and left-to-right shunting (Pfeffer and Braunwald, 1990).

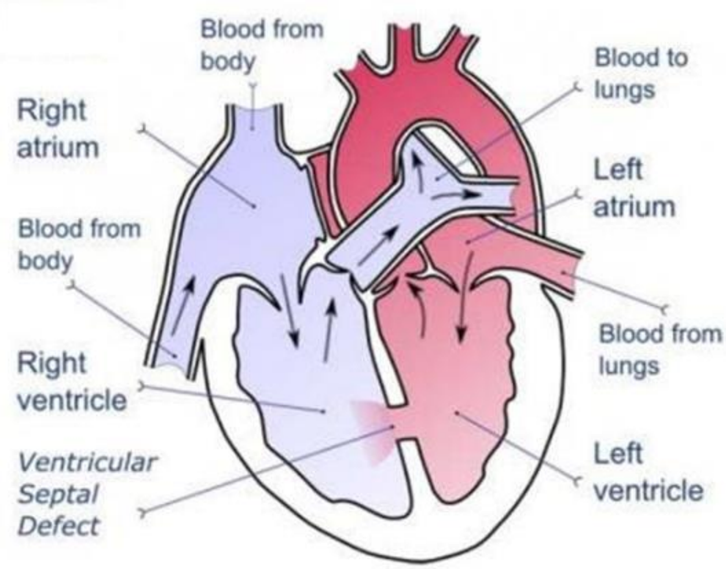


Figure 1: Depiction of postinfarction ventricular septal rupture

Clinical features

The initial sign of ventricular septal rupture (VSR) in a patient who has recently experienced a myocardial infarction (MI) is the development of a pansystolic murmur, typically at the left lower sternal border, with or without radiation to the axilla, and of varying intensity (Bishop et al., 1981). If this murmur is overlooked or its significance is ignored, most patients with VSR remain undiagnosed and face poor outcomes. Importantly, it's worth noting that a systolic murmur can arise from acute mitral regurgitation secondary to MI and postinfarction VSR, and these two conditions can coexist.

Most of these patients initially appear to be in the convalescent phase in the first few days following the MI. However, they may subsequently exhibit a new systolic murmur, experience recurrent chest pain, and see a deterioration in hemodynamics marked by the onset of dyspnea, tachycardia, hypotension, and oliguria. Flash pulmonary oedema may manifest in the patient within 1 to 3 days after a stable myocardial infarction. Cardiogenic shock is observed in severe instances (Mubarik and Iqbal, 2023).

Diagnosis

During clinical examination, approximately 90% of patients with postinfarction ventricular septal rupture (VSR) will exhibit a new and harsh pansystolic murmur at the left lower sternal border, often accompanied by a palpable thrill.

Chest X-rays typically reveal progressive pulmonary oedema and an enlarged heart. The electrocardiogram (ECG) displays signs of transmural myocardial infarction in the relevant region, with occasional episodes of transient partial atrioventricular conduction block occurring around VSR development. However, no specific ECG findings are highly predictive or diagnostic of VSR.

Once a left-to-right shunt is confirmed and initial management steps are taken, coronary angiography is recommended for hemodynamically stable patients. If echocardiographic studies effectively identify the VSR and the presence or absence of mitral regurgitation and left ventricular wall motion abnormalities, intracardiac pressure measurements and left ventriculography are unnecessary.

Two-dimensional echocardiography with Doppler is employed to diagnose ventricular septal rupture (VSR), visualising blood flow across the ventricular septum (Boettler et al., 2005). The echocardiogram highlights right ventricular dilatation and pulmonary hypertension due to increased right-sided blood flow. Colour Doppler echocardiography plays a crucial role in assessing the anatomical size of the rupture.

Echocardiography is currently the definitive diagnostic tool for identifying postinfarction VSR and distinguishing it from other complications, such as acute mitral regurgitation. Echocardiography can also provide valuable information regarding the location and size of the septal rupture and any associated ventricular wall motion abnormalities. Additionally, it allows for quantification of mitral or tricuspid regurgitation and assessment of left ventricular and right ventricular size and contractility.

Transoesophageal echocardiography is indicated for patients for whom obtaining a satisfactory view of the myocardium through transthoracic echocardiograms is challenging. This includes patients on mechanical ventilators or with a large body habitus.

Cardiac catheterization is reserved for stable patients and necessitates careful judgment. In cases of VSR, patients exhibit a step-up in oxygen saturation between the right atrium and the pulmonary artery.

Many patients with postinfarction VSR may have undergone left heart catheterization upon presentation with transmural infarction, and reperfusion of the infarct vessel might have already been initiated. Although left heart catheterization can identify or treat residual coronary stenoses, the mortality benefit from reperfusion of the infarct vessel diminishes rapidly after several hours into the infarction.

Right heart catheterization provides essential parameters like cardiac output, central venous pressure, and pulmonary artery pressure, which offer prognostic value and assist in optimizing medical management. The decision to perform preoperative cardiac catheterization may not be necessary when the diagnosis is firmly established through echocardiography (Smyllie et al., 1990). Furthermore, the relatively high incidence of single-vessel disease in postinfarction VSR patients, along with the risk of volume overload and nephrotoxicity from contrast dye, suggests that the decision to perform coronary angiography after the onset of septal rupture should be determined on a case-by-case basis.

Distinguishing postinfarction VSR from other conditions, particularly acute ischemic mitral regurgitation with or without papillary muscle rupture, is crucial in patients presenting with acute congestive heart failure and a new systolic murmur several days after a transmural myocardial infarction. While echocardiography is the definitive method for distinguishing between these conditions, certain clinical associations may raise initial suspicions. For instance, septal rupture is more common in anterior infarctions, whereas papillary muscle rupture is more frequently associated with inferoposterior infarctions.

Natural history

Before the advent of thrombolytic therapy and acute percutaneous coronary artery interventions, the incidence of postinfarction ventricular septal rupture (VSR) was approximately 1% to 3% among patients who had suffered a heart attack (Crenshaw et al., 2000; Massetti et al., 2000; Nakatani et al., 2003). However, with these therapeutic interventions' advent, the VSR occurrence frequency has significantly decreased to less than 0.5% of heart attack patients (Maltais et al., 2009; Mann and Roberts, 1987). Ventricular septal rupture typically manifests within the first week following an acute myocardial infarction (MI), with a particularly high incidence observed within the first day, as evidenced by the GUSTO-I trial (Crenshaw et al., 2000; Massetti et al., 2000). The median time for presentation of this condition, according to the SHOCK trial (Menon et al., 2000), was 16 hours. Early mortality is prevalent in cases without surgical intervention, with fewer than 30% of patients surviving for two weeks and merely 10% to 20% extending their survival past four weeks (Maltais et al., 2009; Mann and Roberts, 1987). The risk of death is most pronounced immediately after myocardial rupture, subsequently diminishing over time. It's worth noting that women and elderly individuals may exhibit a higher susceptibility to this condition (Maltais et al., 2009).

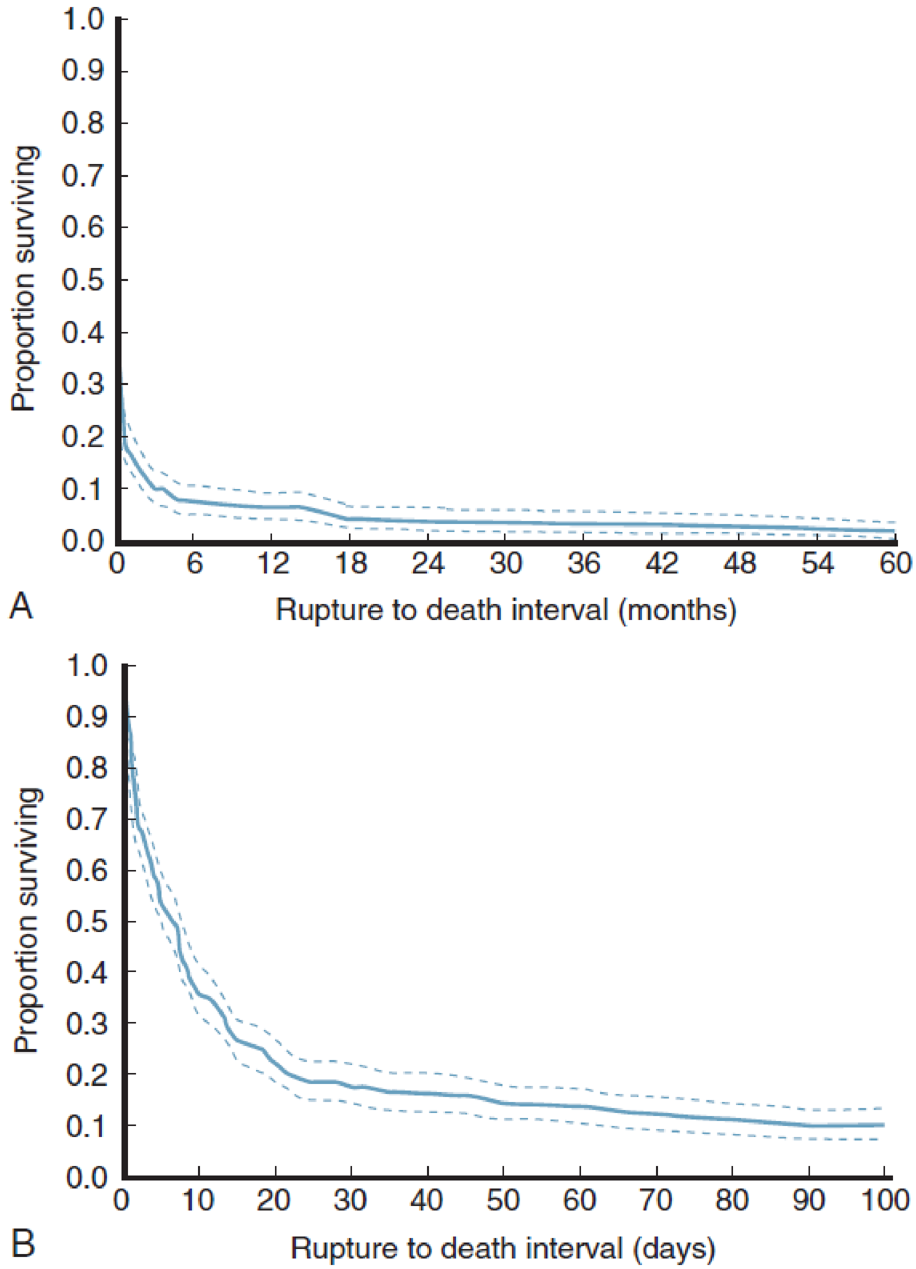


Figure 2: Survival without surgical treatment of patients with ventricular septal rupture after acute myocardial infarction, based on analysis of all proven cases (n = 139) reported in the literature until 1977. The solid line represents the proportion surviving, and the dashed lines enclose 70% confidence limits. A, Interval in months between rupture and death. B, Interval in days between rupture and death. Note that half the patients are dead within seven days of rupture. (From Berger TJ, Blackstone EH, Kirklin JW: Unpublished observations; 1978.)

Indication for surgery

Due to the high mortality associated with postinfarction ventricular septal rupture (VSR) and the progressive volume overload resulting from left-to-right shunting, it is advisable to consider correction of VSR in any patient demonstrating viability. Ideally, the correction of the septal rupture should be performed promptly to minimize subsequent end-organ damage arising from heart failure and hypoperfusion. In some cases, patients may have a limited likelihood of surviving surgery, making medical or percutaneous therapy more favourable. The timing of surgical intervention may range from emergent to urgent to delayed, contingent upon the patient's clinical condition. Delays in surgery may be warranted to achieve hemodynamic optimization, acquire necessary diagnostic data, and optimize the patient's volume status within a relatively short timeframe. However, after these objectives are met, prolonging the delay of surgery tends to result in a deterioration of the patient's condition. Exceptions could be considered for patients with relatively small shunts or other surgical contraindications, where the closure of the septal rupture might be deferred until acute conditions are adequately addressed. Historically, substantial delays in surgery were often selected for patients with a higher likelihood of long-term survival, while many potentially salvageable patients succumbed during the waiting period (Morillon-Lutun et al., 2013).

The optimal timing of definitive surgical repair remains elusive. Although the 2013 American College of Cardiology and American Heart Association guidelines recommended emergent surgical repair regardless of hemodynamic status, the timing of surgery in the setting of VSR remains controversial and should be individualized. Early corrective surgery should be considered in hemodynamically stable patients with preserved end-organ function and favourable anatomy because sudden and unpredictable hemodynamic compromise is often noted. Delayed surgery in hemodynamically stable patients may be considered when surgical anatomy is complex, and there is concern regarding tissue fragility and the ability to perform definitive repair. Although fraught with bias, the perceived benefit of delayed surgery does have a mechanistic basis. Following infarction, metalloproteinase activity and tissue breakdown peak by day 7, whereas new collagen deposition begins by days 2-4; necrotic myocytes are entirely replaced by collagen by 28 days.13 Therefore, delay might facilitate successful repair by allowing friable tissue to organize, strengthen, and become well-differentiated from surrounding healthy tissue. In this scenario, close follow-up in the intensive care unit may be considered to enable tissue healing and promote chances of definitive repair. Watchful waiting in this group of patients may also be appropriate for significant platelet inhibition from exposure to potent dual antiplatelet therapy. In recognition of the possible benefits of delayed repair, the 2017 European Society of Cardiology guidelines promote delayed elective repair in patients initially responding to aggressive conservative management.

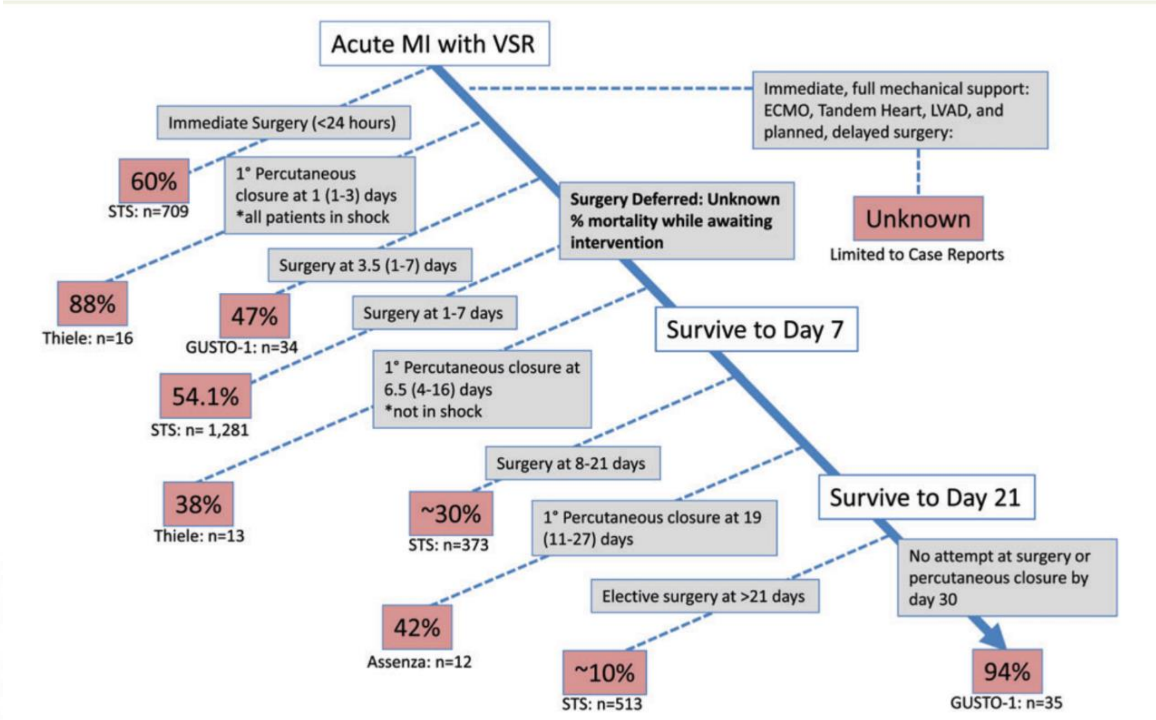


Diagram: Mortality and timing of repair.

Preparation for surgery

Given the critical, life-threatening nature of postinfarction ventricular septal ruptures, where approximately 50 to 60% of patients present with severe congestive heart failure or low cardiac output, these individuals should be managed in an intensive care environment (Agnihotri et al., 2012). Preoperative management for such cases serves three pivotal objectives: (1) sustaining cardiac output and arterial pressure to ensure sufficient perfusion of vital organs, (2) diminishing systemic vascular resistance to reduce left-to-right shunting, and (3) preserving an adequate coronary blood supply (Agnihotri et al., 2012). Immediate involvement of the surgical team upon diagnosis of postinfarction ventricular septal ruptures is essential. An early intervention includes placing an intra-aortic balloon pump, which helps enhance coronary blood flow and decrease the left-to-right shunt by lowering left ventricular outflow resistance (Gold et al., 1973).

Pharmacological therapy for patients with postinfarction ventricular septal ruptures encompasses using inotropic agents to ameliorate end-organ perfusion in impaired right and left ventricular function. Additionally, intravenous diuretics are often required early on to address pulmonary oedema. Although intravenous vasodilators such as sodium nitroprusside, nitroglycerin, or calcium channel blockers can be occasionally beneficial, most patients may not tolerate substantial doses of these agents due to significant hypotension. Since these patients typically have substantial ventricular dysfunction, which may not improve with surgery, some individuals might necessitate an evaluation for acute mechanical ventricular replacement therapy. This could involve the utilization of extracorporeal membrane oxygenation or right or left ventricular assist devices. Generally, patients receiving this type of support before surgery are those deemed poor candidates for immediate surgical closure of the septal rupture but in whom reversible end-organ dysfunction is feasible. Preoperative support over a defined period may enable some degree of infarct tissue healing, recovery of stunned myocardium, and restoration of other end organs. However, this comes with the inherent risks associated with cardiac assist devices (Gregoric et al., 2014).

Postinfarction ventricular septal rupture patients inherently risk right-to-left shunting from using a sole left ventricular assist device. Consequently, many patients might require biventricular support or, at the very least, partial left heart support (Gregoric et al., 2014).

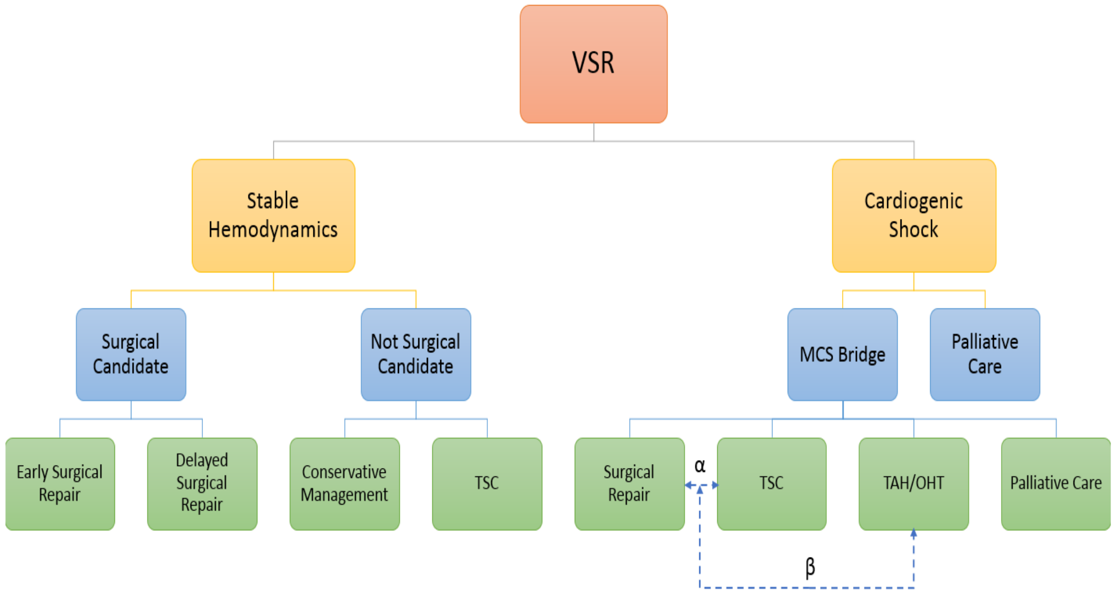


Diagram: VSR Management Algorithm (**α**) If deemed suitable for percutaneous repair, transcatheter septal closure (TSC) may be used as primary repair, bridge to surgery, in conjunction with surgery, or as salvage of residual rupture following surgical repair. (**β**) Candidacy for total artificial heart or cardiac transplantation should be considered for any unstable patient, whether as an alternative to, in addition to, or following failure of repair. (MCS = mechanical circulatory support; OHT = orthotopic heart transplant; TAH = total artificial heart).

Ref: Contemporary Management of Post-MI Ventricular Septal Rupture." American College of Cardiology. Published July 30, 2018.

Operative techniques

Overview

Surgical strategies for addressing a postinfarction ventricular septal defect can be categorized according to the site of the infarction and whether the approach involves infarct excision or exclusion. Infarctectomy, either with or without patch closure of the infarct, represents a viable technique for managing specific septal and apical infarctions. This approach aims to facilitate suturing to viable myocardial tissue capable of healing (Daggett et al., 1970).

The trans-infarct approach to managing ventricular septal defects involves several key steps:

Principles of Infarctectomy Repair of Postinfarction VSD

1) Precise trimming of the left ventricular edges of the infarct region to remove damaged tissue, ensuring that viable muscle remains intact to prevent future rupture of the closure.
2) Careful trimming of the right ventricular muscle as needed to provide a clear view of the defect margins.
3) Examine the left ventricular papillary muscles with mitral valve replacement only if these structures have obvious rupture.
4) Closure of the septal defect without creating tension, typically requiring the use of prosthetic material.
5) Closure of the infarcted area without tension, utilizing prosthetic material as needed, and securing the patch on the epicardial surface to avoid straining the delicate endocardial tissue.
6) Reinforcement of suture lines with pledgets or Teflon felt strips (or similar materials) to prevent sutures from damaging the fragile muscle tissue (Heitmiller et al., 1986).

Principles of Exclusion Repair of Postinfarction VSD (David et al., 1995)

1) Infarctectomy is only performed if necrotic muscle along the ventriculotomy demonstrates sloughing during closure.
2) Utilization of a bovine pericardial patch, in either an oval shape for anterior defects or a triangular shape for posterior defects, securely sutured with continuous Prolene around the defect to exclude it from the left ventricular (LV) cavity.
3) Full-thickness bites may be taken to the epicardial surface and anchored using strips of pericardium or Teflon as necessary (see text for details).
4) For an anterior patch, anchoring is carried out to the noninfarcted septum below the defect and then to the noninfarcted endocardium of the anterolateral ventricular wall. In cases where the infarct affects the base of the anterior muscle, full-thickness anchoring bites are employed.
5) A posterior patch is anchored to the mitral annulus, the noninfarcted septum, and through the infarcted posterior wall along a line corresponding to the medial margin of the posteromedial papillary muscle, using full-thickness anchoring.
6) The infarctectomy is closed with strips of pericardium or Teflon.
7) Whenever possible, the infarcted right ventricular free wall is left undisturbed during closure.

Intraoperative consideration

Patients with postinfarction ventricular septal rupture (VSR) often present in cardiogenic shock, displaying intolerance to vasodilation and requiring inotropic support during anaesthesia induction. To ensure stability, the placement of an intra-aortic balloon pump is recommended, particularly if not already present. It is crucial to avoid pulmonary and venous vasodilators to minimize left-to-right shunting. The insertion of a Swan-Ganz pulmonary artery catheter aids in postoperative inotropic management (Loisance et al., 1991).

The preferred incision for these cases is a median sternotomy. Cardiopulmonary bypass is established with bicaval venous drainage and bicaval isolation to prevent air entrainment into the venous line, given that the right heart will be open through the septal defect. Repairing postinfarction septal defects is most effective when the heart is arrested, with a strong focus on myocardial protection, considering the pre-existing impaired ventricular performance in these patients.

The decision to perform concurrent coronary bypass grafting depends on surgical philosophy and each patient's coronary anatomy. Some argue against it, citing potential delays, added risks, and limited benefits for already infarcted myocardium. However, others suggest that concurrent grafting might enhance myocardial protection and reduce late myocardial ischemia, even though the incidence of significant untreated coronary disease in patients receiving isolated repair has been low in certain series.

When weaning from cardiopulmonary bypass, it's advisable to utilise an intra-aortic balloon pump if possible. Multiple inotropic agents are required, with intravenous milrinone and inhaled nitric oxide holding particular value. Considering cases where standard weaning techniques fail in postinfarction VSR patients, special attention should be given to potential residual right-to-left shunting and resultant hypoxemia when using an isolated left ventricular assist device.

Postoperative hemostasis can pose challenges in these critically ill patients. The routine use of antifibrinolytic agents like ε-aminocaproic acid (Amicar) can be beneficial. Some recommendations include the application of a fibrin sealant to the ventricular septum surrounding the septal defect before formal repair, followed by applying biological glue to bleeding suture lines post-repair. Transfusion of appropriate blood products and isolated clotting factors may be necessary due to tissue fragility, intolerance of ongoing bleeding, or any degree of postoperative tamponade (Seguin et al., 1992).

Repair of Apical Septal Rupture

Daggett's apical amputation technique, introduced in 1970 (Daggett et al., 1970), involves resecting the necrotic apical myocardium, extending to the left ventricle, right ventricle, and septum (Figure 3). Following this resection, the residual apical defect is closed utilizing interrupted mattress sutures, employing 1-0 Tevdek, securing the left and right ventricular walls to the interventricular septum. Strips of Teflon felt are utilized outside the left ventricular wall, the right ventricular wall, and on both sides of the interventricular septum to reinforce the closure (Figure 4). Once all sutures are securely tied, a running suture is used to fortify the closure further, ensuring hemostasis (Agnihotri et al., 2012; Daggett et al., 1970).

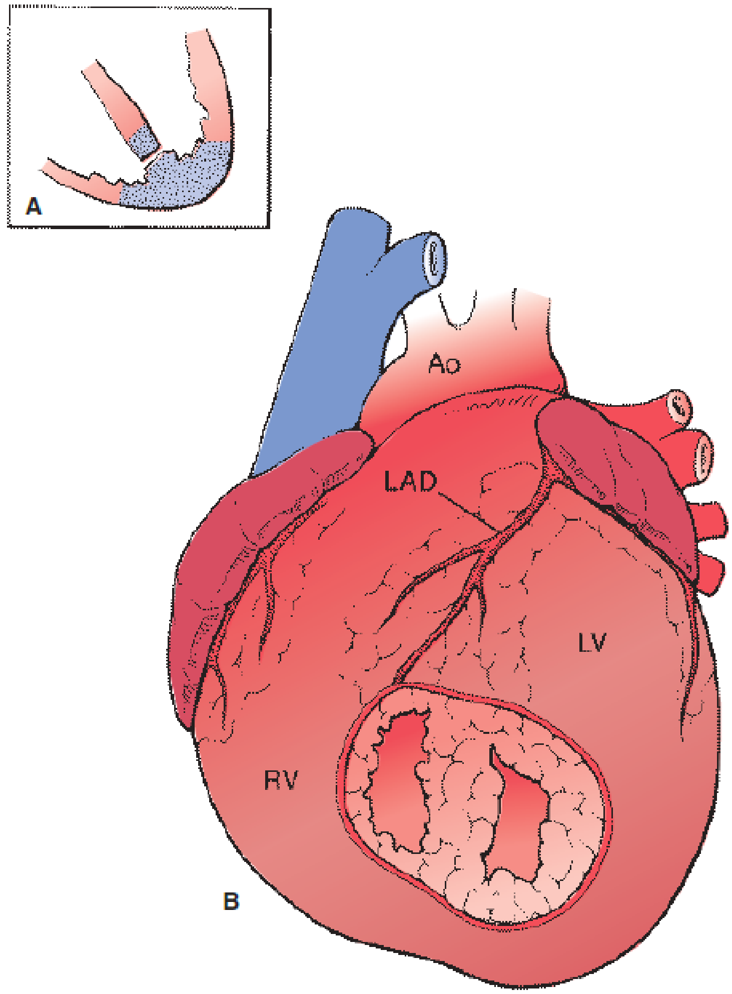
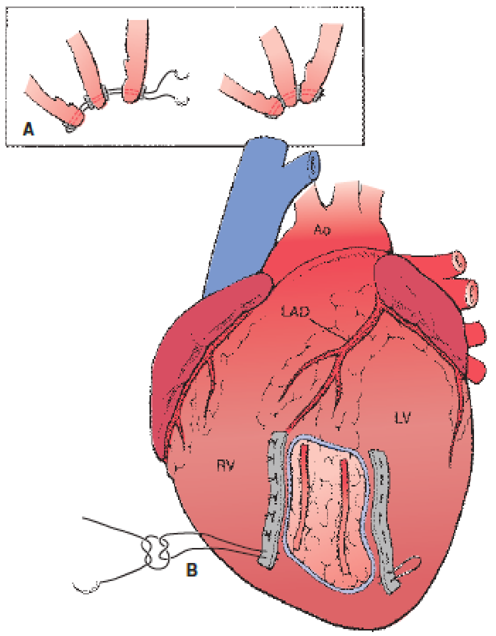


Figure 3: (A) Apical postinfarction ventricular septal defect (B) View of the ap cal septal rupture, which is exposed by amputating the apex of the left and right ventricles. Ao, aorta; LAD, left anterior descending coronary artery; LV, left ventricle; RV, right ventricle; stippled region, infarcted myocardium (Agnihotri et al., 2012)

Repair of anterior septal rupture with infarctectomy

Repairing anterior postinfarction septal defects involves a linear incision along the anterior infarct, parallel to the left anterior descending artery (Figure 4 & 5).



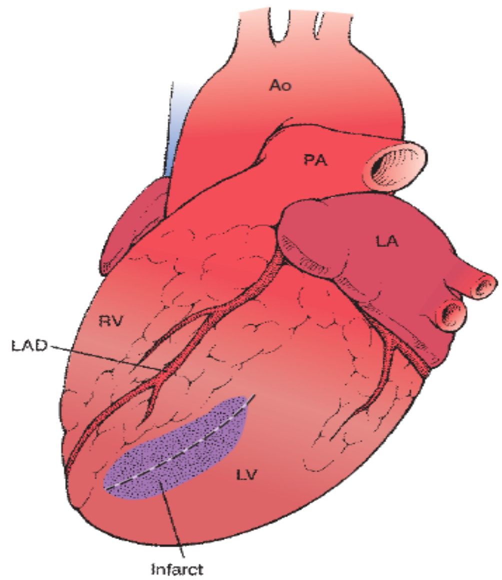


Figure 4: (A) The necrotic infarct and the apical septum have been debrided back to healthy muscle. Repair is made by approximating the left ventricle, apical septum, and right ventricle using interrupted mattress sutures of 1-0 Tevdek with buttressing strips of Teflon felt. Felt strips are used within the interior of the left and right ventricles as well as on the epicardial surface of each ventricle. (B) All sutures are placed before any are tied. A second running over-Andover suture (not shown) is used, as in left ventricular aneurysm repair, to ensure a

Figure 5: Transinfarct left ventricular incision to expose an anterior septal rupture. An incision (dashed line) is made parallel to the anterior descending branch of the left coronary artery (LAD) through the center of the infarct (stippled area) in the anterior left ventricle (LV). Ao, aorta; LA, left atrium; PA, pulmonary artery; RV, right ventricle

This approach includes infarctectomy or removal of nonviable tissue, following the technique described by (Daggett et al., 1970; Shumacker, 1972) introduced a method for closing small anterior septal defects, which involves suturing the free anterior edge of the debrided septum to the right ventricular free wall using 1-0 Tevdek mattress sutures along with strips of felt (Fig. 6). Afterward, a secondary row of mattress sutures, supported by Teflon felt strips, is used to close the transinfarct left ventriculotomy (see Figs. 6). Finally, an outer hemostatic running suture line is employed for complete ventriculotomy closure. This approach is suitable for anteriorly located small septal defects.

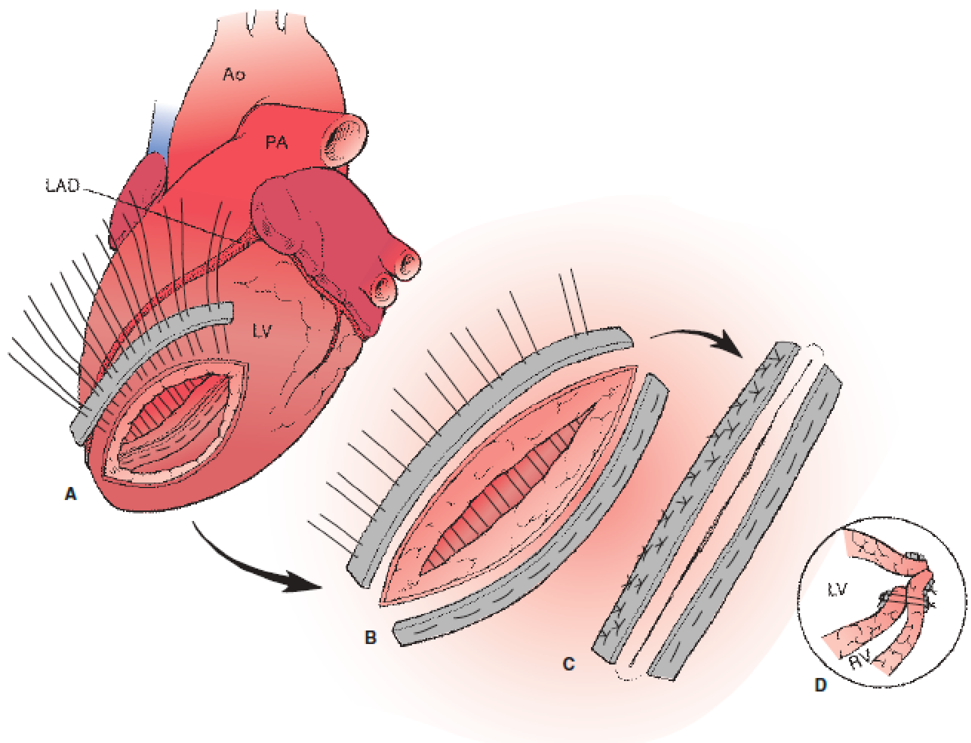


Figure 6: A) Repair of an anterior septal rupture by plicating the free anterior edge of the septum to the right ventricular free wall with interrupted 1-0 Tevdek mattress sutures buttressed with strips of Teflon felt. (B, C, and D) The left ventriculotomy is then closed as a separate suture line, again with interrupted mattress sutures of 1-0 Tevdek buttressed with felt strips. A second running suture (not shown) is used to ensure a secure left ventriculotomy closure. Ao, aorta; LAD, left anterior descending coronary artery; LV, left ventricle; PA, pulmonary artery; RV, right ventricle. (Adapted with permission from Cohn LH (ed): Modern Techniques in Surgery: Cardiac/Thoracic Surgery. Mt. Kisco, NY: Futura; 1983.)

A different technique is employed in cases where septal defects are not small and are situated in regions other than the anterior area. The debrided septal defect is closed using a prosthetic patch made of Dacron or bovine pericardium, as described by (Daggett et al., 1970; Shumacker, 1972) (see Fig. 7). After removing the necrotic septum and left ventricular muscle, a series of pledgeted interrupted mattress sutures are placed around the perimeter of the defect (see Fig. 7). Sutures along the posterior aspect of the defect are passed from the right to the left through the septum. Along the anterior edge of the defect, sutures traverse from the epicardial surface of the right ventricle to the endocardial surface. All sutures are positioned before inserting the patch, passing them through the edge of the synthetic patch on the septum's left side (Fig. 7). Each suture is then guided through an additional pledget, and all are securely tied. This approach is applied to close relatively larger non-anterior septal defects.

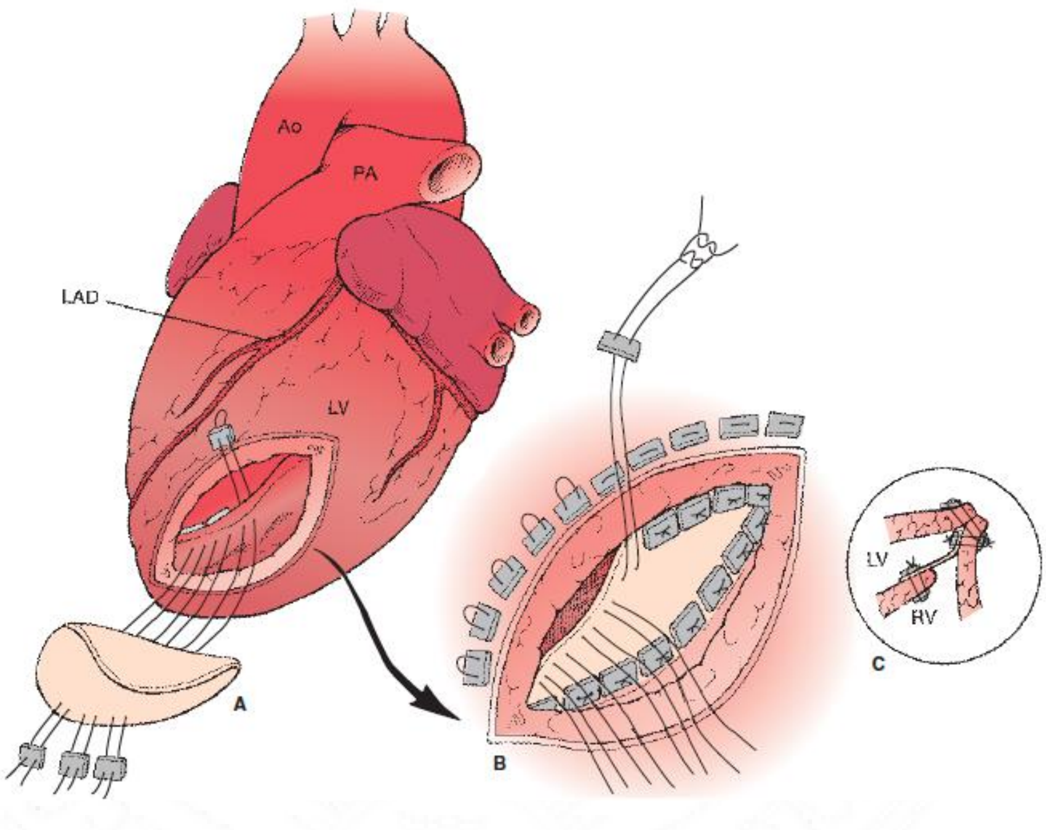


Figure 7: (A) Larger anterior septal defects require a Dacron patch, which is sewn to the left side of the ventricular septum with interrupted mattress sutures, each of which is bolstered with a pledget of Teflon felt on the right ventricular side of the septum and anteriorly on the epicardial surface of the right ventricular free wall. All sutures are placed before the patch is inserted (B and C). Additional pledgets are placed on the left ventricular side, overlying the patch to cushion each suture as it is tied down to prevent cutting through the friable muscle. Ao, aorta; LAD, left anterior descending coronary artery; LV, left ventricle; PA,

pulmonary artery; RV, right ventricle. (Adapted with permission from Cohn LH (ed): Modern Techniques in Surgery: Cardiac/Thoracic Surgery. Mt. Kisco, NY: Futura; 1983.

Posterior/Inferior repair with infarctectomy

Repairing postinfarction defects of the inferoposterior septum following inferolateral infarction has posed a significant challenge due to the complex geometry of the area. Daggett introduced a technique in 1974 combining infarctectomy and patch closure, which produced consistently favourable outcomes. However, many contemporary surgeons now advocate for an exclusion technique rather than an infarctectomy in the context of inferoposterior septal defects. With the heart arrested and the left ventricle vented, the heart is gently moved out of the pericardial space to expose the posterior descending coronary artery and the inferior infarction, which might extend into both ventricles (Fig. 8).

Following Daggett's approach, a trans infarct incision is made within the left ventricle, and the left ventricular part of the infarction is carefully removed (see Fig. 8), revealing the septal defect. The left ventricular papillary muscles are assessed during this procedure. If necessary, mitral valve replacement is performed through a separate, traditional left atrial incision to avoid damaging the fragile ventricular muscle. After the complete removal of the infarcted left ventricular muscle, less extensive debridement of the right ventricle is performed, aiming to excise only the muscle necessary for clear visualization of the defect(s). This approach has proven effective in preventing delayed rupture of the right ventricle (Daggett et al., 1970; Shumacker, 1972).

In cases where the posterior septum has experienced a crack or split from the adjoining ventricular free wall, and significant septal tissue loss has not occurred, the septal rim of the posterior defect can be brought together and sutured to the edge of the diaphragmatic right ventricular free wall. This repair is achieved using mattress sutures reinforced with Teflon felt strips or bovine pericardium (refer to Fig. 8 for visual representation) (Daggett et al., 1970; Shumacker, 1972). This technique ensures a secure closure of the posterior defect and promotes structural integrity within the heart.

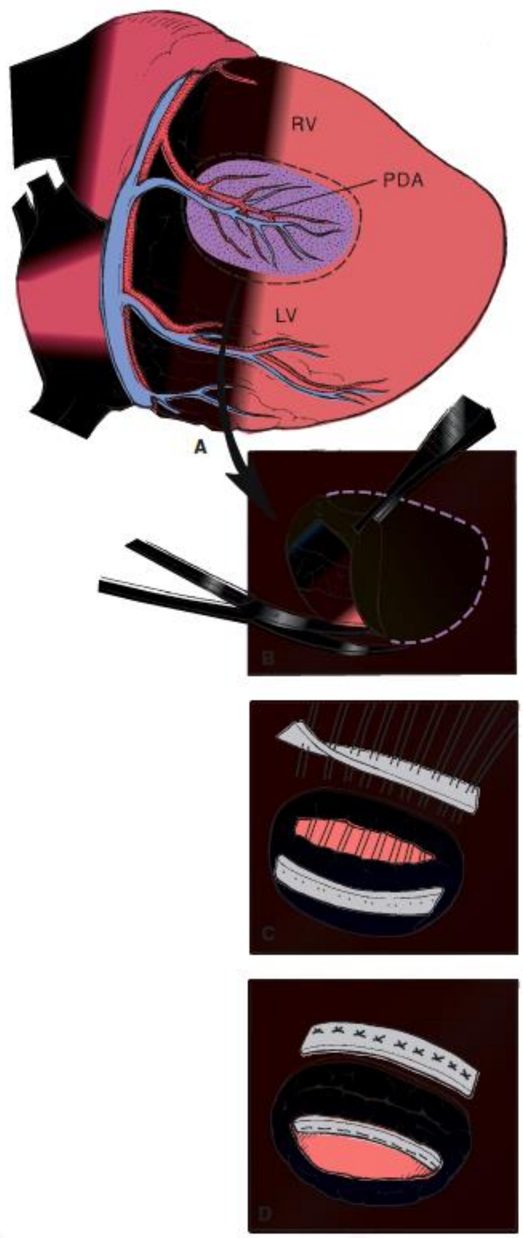


Figure 8: (A) Surgical view of an inferior infarct (shaded area) associated with a posterior septal rupture. To expose this area during surgery, the heart is dislocated up and out of the pericardial sac, followed by retraction toward the head, similar to the technique used in distal vein bypass and anastomosis to the posterior descending artery. (B) The inferoposterior infarct is excised to reveal the posterior septal defect. Complete removal of the left ventricular portion of the infarct is crucial to prevent delayed rupture of the ventriculotomy repair. The right ventricle's free edge is gradually trimmed to expose the defect's margins. (C and D) Repair of the posterior septal rupture is achieved by combining the edge of the posterior septum

and the free wall of the diaphragmatic right ventricle using mattress sutures reinforced with felt. This repair is suitable when the septum has cracked or separated from the posterior ventricular wall without significant septal muscle necrosis. The surgeon typically stands on the left side of the supine patient to perform this repair. The left ventriculotomy is closed as a separate suture line, employing interrupted mattress sutures of 1-0 Tevdek buttressed with felt strips. A second running suture is used to ensure secure closure of the left ventriculotomy (not shown). (LV: posterior left ventricle; PDA: posterior descending artery; RV: diaphragmatic surface of the right ventricle. Adapted with permission from Daggett WM: Surgical technique for early repair of posterior ventricular septum rupture, J Thorac Cardiovasc Surg. 1982; Aug; 84(2): 306-312.)

Anterior and inferoposterior repair by infarct exclusion

The concept of ventricular endoaneurysmorrhaphy, introduced by Dor and others, involves placing an intracavitary endocardial patch to exclude infarcted myocardium while maintaining proper ventricular geometry and preserving left ventricular function. The technique was applied to repair postinfarction ventricular septal defects (David et al., 1995) and various other surgeons. While some centres reported successful outcomes, others observed mixed results.

For inferoposterior septal defects, the endocardial patch technique described by David was particularly beneficial. In this approach, the interventricular septum is exposed via a left ventriculotomy made through the infarct, originating at the apex and extending proximally, parallel to the anterior descending artery but at a distance of 1 to 2 cm. Stay sutures assist in exposing the infarcted septum.

The septal defect is located, and the infarcted muscle margins are identified. A bovine pericardial patch is tailored to the shape of the left ventricular infarction, with a slight overhang, and sutured to a healthy endocardium around the infarct. The patch is also secured to the noninfarcted endocardium of the anterolateral ventricular wall. Proper suture placement and use of pledgets, like bovine pericardium or Teflon felt, help reinforce the repair (David et al., 1995).

Suppose the infarct extends to the base of the anterior papillary muscle. In that case, the suture is guided outside the heart and supported with a strip of the bovine pericardium, or Teflon felt on the epicardial surface of the left ventricle. This technique minimizes the risk of tearing muscle during suture tensioning. Once the patch is securely attached to the left ventricular endocardium, the ventriculotomy is closed in two layers using strips of bovine pericardium or Teflon felt and polypropylene sutures.

This approach effectively excludes the ventricular cavity from the infarcted myocardium, preserving ventricular geometry. The closure of the ventriculotomy is performed meticulously, as illustrated in Fig. 25-9C (David et al., 1995).

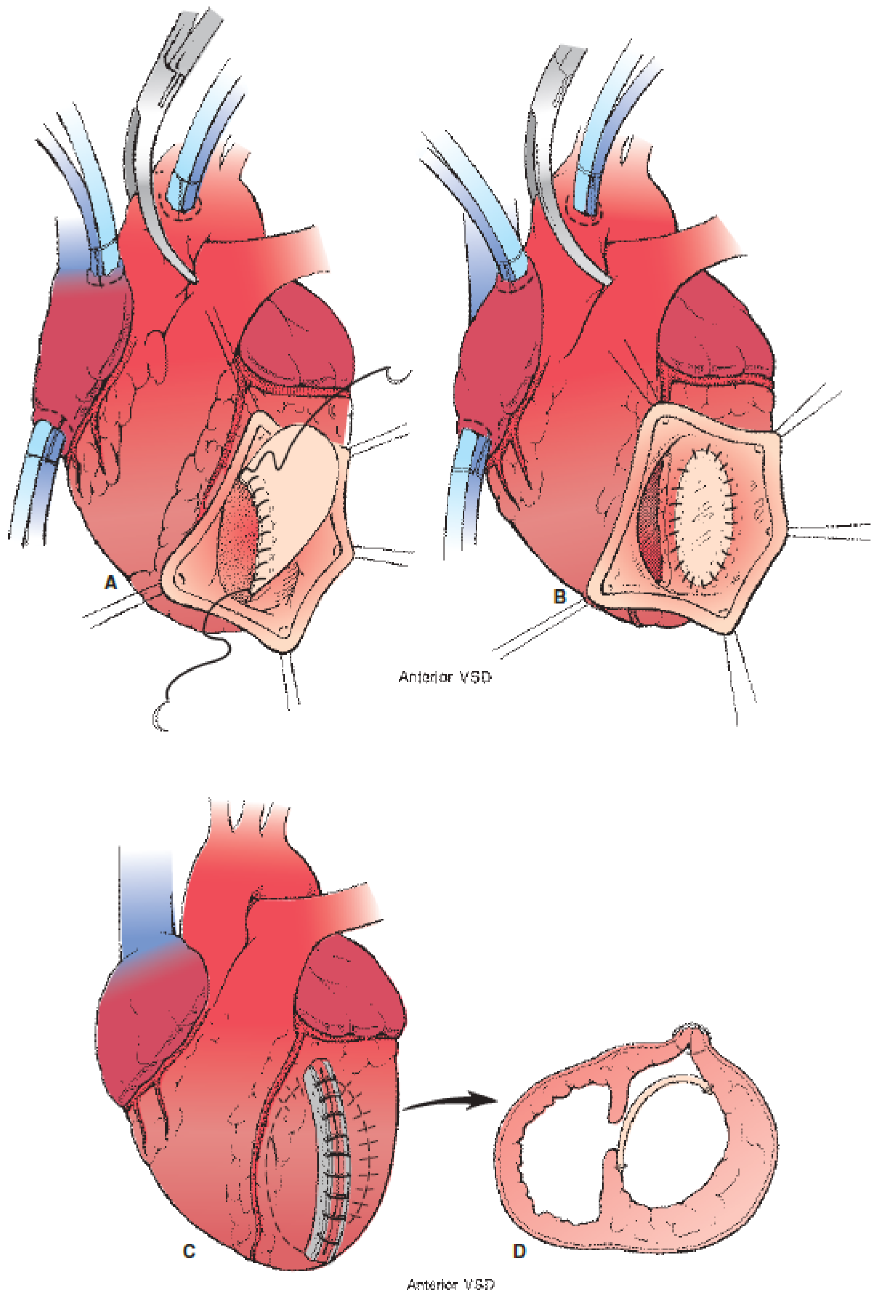


Figure 9: Repair of an anterior postinfarction ventricular septal rupture using the technique of infarct exclusion involves creating a ventriculotomy in the infarcted area of the left ventricular free wall. An internal patch made of materials like Dacron or pericardium replaces or covers damaged areas, including ventricular septal defect, septal infarction, or

free wall infarction. The internal patch is secured to the normal endocardium with sutures. This method does not involve significant myocardial resection, and the septal defect is not closed. The ventriculotomy is repaired with continuous sutures, and the endocardial patch is secured at multiple levels, ensuring a robust repair (David et al., 1995).

Infarctectomy is typically avoided unless the necrotic muscle along the ventriculotomy is deteriorating at the closure time. This approach minimizes infarcted muscle exposure to left ventricular pressures when the heart resumes functioning. Alternatively, sutures can be passed through the ventricular free wall and a customized external patch made of materials like Teflon or pericardium. (Figure 10)

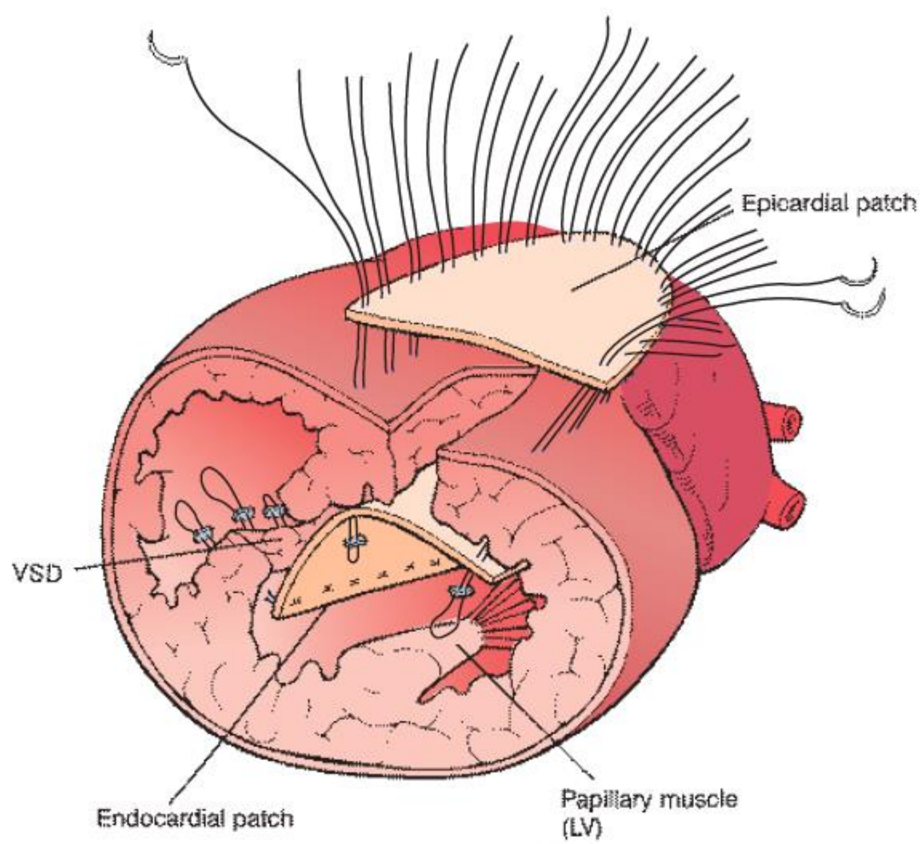


Figure 10: Repair an anterior postinfarction ventricular septal rupture using the infarct exclusion technique with external ventricular free-wall patching with tailored Teflon or pericardium. LV, left ventricle; VSR, ventricular septal defect.

In patients with inferoposterior septal defects, a specific incision is made in the inferior wall of the left ventricle, approximately 1 or 2 mm away from the posterior descending artery (Figure 11). This incision starts at the midportion of the inferior wall and extends proximally towards the mitral annulus and distally towards the apex of the ventricle. During this process, care is taken to avoid causing damage to the posterolateral papillary muscle. Stay sutures are passed through the fat pad at the ventricular apex and the margins of the ventriculotomy, which helps expose the ventricular cavity. Typically, the rupture is discovered in the proximal half of the posterior septum, often involving the posteromedial papillary muscle due to the infarction.

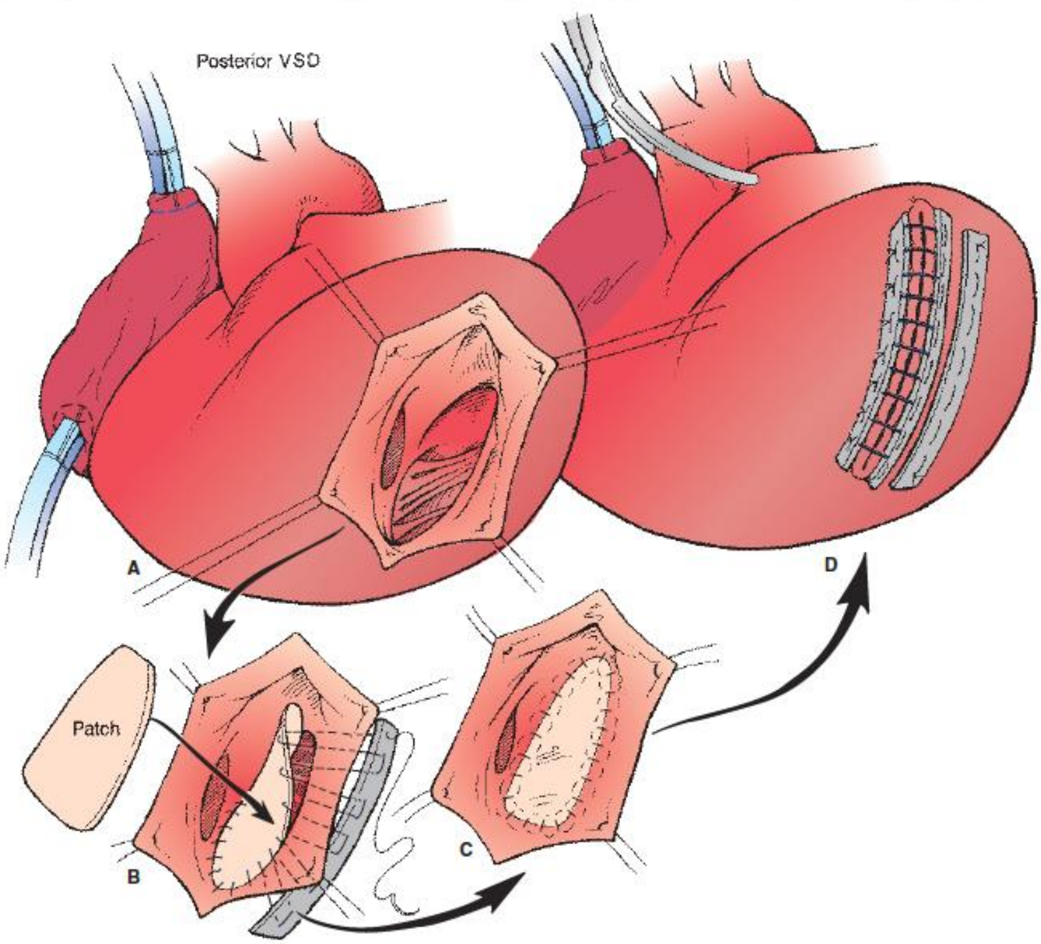


Figure 11: Endocardial repair of a posterior postinfarction ventricular septal rupture using the infarct exclusion technique. (A) An incision is made in the inferior wall of the left ventricle 1 or 2 mm from the posterior descending artery starting at the midportion of the inferior wall and extended proximally toward the mitral annulus and distally toward the apex of the ventricle. Care is taken to avoid damage to the posterolateral papillary muscle. (B) A bovine pericardial patch is tailored in a triangular shape. The base of the triangular-shaped patch is sutured to the fibrous annulus of the mitral valve with a continuous 3-0 polypropylene suture starting at a point corresponding to the level of the posteromedial papillary muscle and moving medially toward the septum until the noninfarcted endocardium is reached. (C) The medial margin of the triangular-shaped patch is sewn to a healthy septal endocardium with a continuous 3-0 or 4-0 polypropylene suture. The lateral side of the patch is sutured to the posterior wall of the left ventricle along a line corresponding to the medial margin of the base of the posteromedial papillary muscle. At this point, it is usually necessary to use full-thickness bites and anchor the sutures on a strip of the pericardium, or Teflon felt applied on the epicardial surface of the posterior wall of the left ventricle. (D) Once the patch is completely sutured to the mitral valve annulus, the endocardium of the interventricular septum, and the full thickness of the posterior wall, the ventriculotomy is closed in two layers of full-thickness sutures buttressed on strips of pericardium or Teflon felt. The infarcted right ventricular wall is undisturbed (David et al., 1995).

A triangular bovine pericardial patch, measuring approximately 4 x 7 cm in most patients, is tailored to fit. The base of this triangular-shaped patch is sutured to the fibrous annulus of the mitral valve using a continuous 3-0 polypropylene suture. This suture starts at a point corresponding to the level of the posteromedial papillary muscle and moves medially toward the septum, terminating once the noninfarcted endocardium is reached. Any excess patch material is trimmed at this stage. The medial margin of the triangular patch is sewn to the healthy septal endocardium with continuous 3-0 or 4-0 polypropylene sutures, with reinforcing pledgeted sutures being used as needed in this area of the septum.

The lateral side of the patch is sutured to the left ventricle's posterior wall along a line corresponding to the medial margin of the base of the posteromedial papillary muscle. Given that the posterior wall of the left ventricle is typically infarcted, full-thickness sutures are usually necessary, anchored to a strip of pericardium or Teflon felt applied to the epicardial surface of the posterior wall of the left ventricle at the level of the posteromedial papillary muscle insertion.

Once the patch is fully sutured to the mitral valve annulus, the endocardium of the interventricular septum, and the full thickness of the posterior wall, the ventriculotomy is closed in two layers, with sutures buttressed on strips of pericardium or Teflon felt. Importantly, the infarcted right ventricular wall is left untouched unless the posteromedial papillary muscle is ruptured, in which case mitral valve replacement becomes necessary.

Daggett and his colleagues believe this infarct exclusion technique offers several theoretical advantages. Firstly, it avoids the need for myocardial resection, as excessive resection can lead to decreased ventricular function, while insufficient resection increases the risk of septal rupture recurrence. Secondly, it helps maintain ventricular geometry, improving ventricular function. Lastly, it prevents tension in fragile muscles, thus reducing the likelihood of postoperative bleeding (Daggett, 1982).

Percutaneous closure

The percutaneous closure of postinfarction ventricular septal defects has been conducted using various devices originally designed for closing congenital atrial or ventricular septal defects (Calvert et al., 2014; Szkutnik et al., 2003; Thiele et al., 2009). These devices can be introduced through either the systemic venous circulation, accessing the septal defect through the right ventricle, or through the arterial system and the left ventricle to reach the septal defect, and sometimes through both routes. A study conducted by Calvert (2014) reported on 53 cases where percutaneous closure of postinfarction ventricular septal defects resulted in complete shunt reduction in 23% of cases, partial shunt reduction in 62% of cases, and an average post-procedure hospital stay of 5 days. The percutaneous approach presents the theoretical advantage of avoiding the need for cardiopulmonary bypass and the associated morbidity in critically ill patients. However, this approach faces technical challenges in delivering the device to the defect, which can be located in various parts of the interventricular septum, and in achieving a durable closure for large defects in a thick septum with poorly defined, necrotic edges. Complications have been reported, including recurrent septal defects due to ongoing septal tissue necrosis and device migration or embolization.

Generally, the results of percutaneous closure tend to be somewhat better when performed two weeks or more after the infarction. However, surgical outcomes also improve when patients can delay intervention (Szkutnik et al., 2003; Thiele et al., 2009). In some cases, hybrid approaches have been explored, where percutaneous devices are placed intraoperatively with open exposure on cardiopulmonary bypass, allowing avoidance of ventriculotomy (Lee et al., 2018). So far, the most suitable application for percutaneous catheter-based devices in postinfarction ventricular septal defects may be in patients with recurrent or residual defects after prior surgical treatment (Calvert et al., 2014; Maree et al., 2006).

Postoperative care

In patients with postinfarction ventricular septal defects, a common occurrence is the presence of impaired function in both ventricles, along with reduced cardiac output. This situation often necessitates a gradual reduction in the use of inotropic support and the intra-aortic balloon pump for several days. Some individuals may benefit from treatments involving intravenous milrinone and nitric oxide inhalation. Addressing hypoxia, hypercarbia, and acidosis is crucial to preserve proper right ventricular afterload. Additionally, it's noteworthy that these patients frequently experience acute renal insufficiency and pulmonary oedema. Effectively managing these conditions involves careful fluid control, early administration of diuretics following surgery, and the application of positive end-expiratory pressure during ventilation. Patients often exhibit ventricular and atrial arrhythmias, which can be challenging to endure. However, administering intravenous amiodarone can effectively control many of these arrhythmias.

Early results

Operative mortality, which is defined as death occurring before discharge or within 30 days following the operation, exhibited a range of 30% to 50% according to various studies (Agnihotri et al., 2012; Cooley et al., 1957; Jeppsson et al., 2005; Maree et al., 2006; Yam et al., 2013). In a study conducted at Massachusetts General Hospital involving 114 patients, Daggett reported an operative mortality rate of 37%, as illustrated in Figure 12. This study also revealed a rapid decrease in the risk of death after the initial year, as demonstrated in Figure 12 (Agnihotri et al., 2012). Several independent risk factors were identified for early mortality, including older age, elevated blood urea nitrogen (BUN) levels, emergency surgery, higher right atrial pressure, and preoperative use of catecholamines (Agnihotri et al., 2012). Notably, a septal rupture in the inferoposterior region was associated with an elevated operative mortality rate.

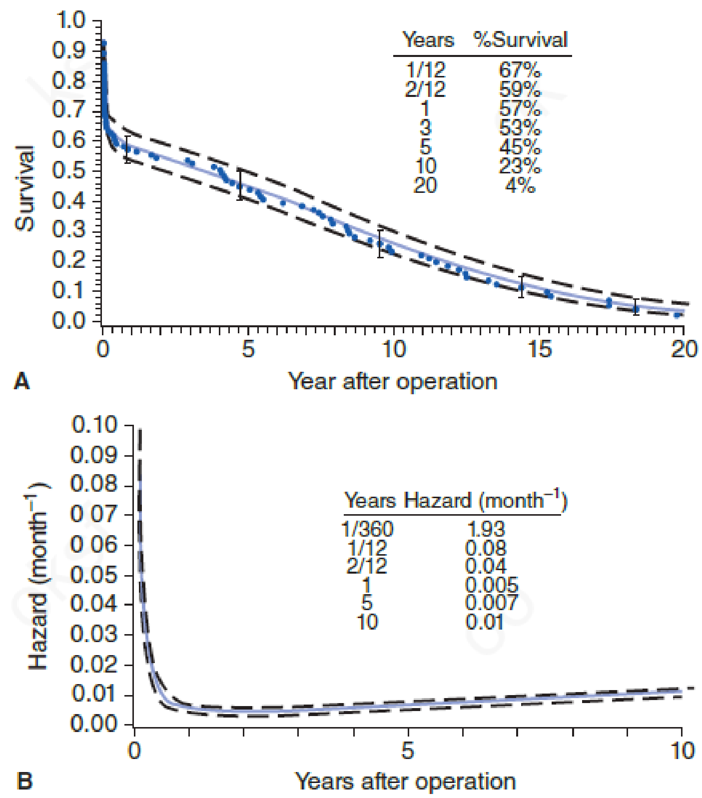


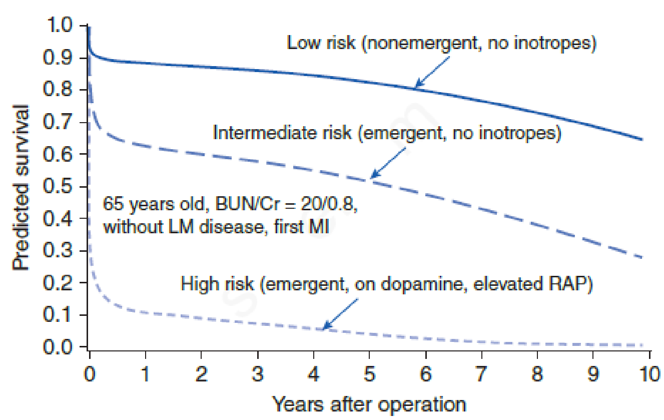
Figure 12: (A) Time-related survival after postinfarction ventricular septal defect repair at the Massachusetts General Hospital (n = 114). Note that the horizontal axis extends to 20 years. Circles represent each death, positioned on the horizontal axis at the interval from operation to death and actuarially (Kaplan-Meier method) along the vertical axis. The vertical bars represent 70% confidence limits (± 1 SD). The solid line represents the parametrically estimated freedom from death, and the dashed lines enclose that estimate's 70% confidence limits. The table shows the nonparametric estimates at specified intervals. (B) Hazard function for death after repair of postinfarction ventricular septal defect (n = 114). The horizontal axis is expanded for better visualization of early risk. The hazard function has two phases: an early, rapidly declining phase, which gives way to a slowly rising phase at about six months. The estimate is shown with 70% confidence limits.

The increased operative mortality in postinfarction ventricular septal defect (VSR) cases can be attributed to the heightened complexity of the repair procedure, a greater risk of concomitant mitral regurgitation, and associated right ventricular dysfunction. In a review of the Massachusetts General Hospital's experience, Daggett and colleagues (1982) found that patients presented a wide spectrum of risk factors, with hemodynamic instability (such as cases requiring emergency surgery and using inotropic support) playing a significant role in determining patient outcomes. It was noted that a small subset of high-risk patients substantially impacted the overall mortality rate. This observation may help account for the considerable variation in mortality rates among different healthcare institutions. This variation appears to be influenced by patient selection and referral patterns, irrespective of the surgical techniques employed.

According to the findings reported by Daggett and colleagues, the leading cause of mortality following the surgical repair of acute postinfarction ventricular septal defects was low cardiac output syndrome, accounting for 52% of cases. Technical issues, such as recurrent or residual septal defects and bleeding, contributed to 23% of deaths. Sepsis accounted for 17% of cases, followed by recurrent infarction (9%), cerebrovascular complications (4%), and intractable ventricular arrhythmias. These observations shed light on the in-hospital complications of this surgical procedure (Daggett, 1982).

Late results: Survival

Despite the notably high early mortality rate associated with the repair of postinfarction ventricular septal defects, the mortality rates stabilize at a much lower level after approximately one year. Actuarial data reveals survival rates at five years post-surgery varied between 40% and 60%. For patients who survived their hospital stay, the reported survival rates at 1, 5, and 10 years were 91%, 70%, and 37%, respectively. Various factors have been identified as predictors of late mortality, including older age, prior myocardial infarction, elevated creatinine levels, higher right atrial pressure, and the presence of left main coronary disease(Agnihotri et al., 2012; Cooley et al., 1957; Jeppsson et al., 2005; Maree et al., 2006; Yam et al., 2013). Figure: 13



3. MATERIAL AND METHODOLOGY

STUDY DESIGN: A retrospective, observational study was conducted to study the early outcomes and survival rate after surgical repair of post-infraction ventricular septal rupture.

STUDY AREA: The study was conducted in the department of Cardiovascular and Thoracic Surgery at Sree Chitra Tirunal Institute For Medical Sciences And Technology , Trivandrum.

ORGANISING THE STUDY:

i. Ethical consideration:

Before the study's conduction, relevant permission was taken from the Institutional Ethics Committee (IEC) of the Sree Chitra Tirunal Institute for Medical Sciences and Technology, Trivandrum (SCTIMST).

ii. Approval from authorities:

Permission was obtained for conducting a study in the cardiovascular and thoracic surgery department from the institutional ethics committee. The same were informed to the authority of the cardiovascular and thoracic surgery department in SCTIMST.

iii. Designing of Patient, consent form and data collection sheet:

The following forms were designed for study:

1) Consent form- not applicable

2) **Data collection sheet-** Data was obtained from the institutional electronic medical records. Data collection was done, and details of the patient information such as Age, Gender, Preoperative parameters, Co-morbidities, Intra-operative parameters and postoperative parameters, along with follow-up, was compiled. (Annexure-Other).

SAMPLE SIZE DETERMINATION:

We got a total 23 number of patients who underwent surgical repair for post-infarction ventricular septal rupture between 2011-2021, all of them were included in the study. Hence, data were analyzed with those numbers.

SAMPLE SIZE: All Post-Infarction Ventricular Septal Rupture cases operated between 2011 - 2021. A total of 23 patients were operated on in this period, all included in the study.

SAMPLING TECHNIQUE: Retrospective chart analysis from medical records.

ELIGIBILITY CRITERIA: Gender, class, caste, ethnicity, or race will NOT be used as Inclusion and Exclusion criteria**.**

Inclusion criteria:

1) Patients with post-infarction ventricular septal rupture who underwent surgical repair from 2011 to 2021.

Exclusion criteria:

1) Patients with post-infarction ventricular septal rupture who did not undergo surgical repair.

ARMAMENTARIUM USED FOR DATA COLLECTION:

Data from the computer records were obtained and compiled on a data collection form.

CLINICAL PROCEDURE AND EXAMINATION:

After obtaining permission from the IEC, Data was collected from electronic medical records. Preoperative, intra-operative and post-operative data was collected, and the principal investigator and co-investigators performed retrospective data analysis. After the procedure, the principal investigator kept the data and patient details confidential.

Interventions: This is a retro-prospective study, and no intervention is required.

Outcomes parameters: Evaluation of the study objectives was the outcome parameters.

Method: All patients who underwent surgical repair of post-infarction ventricular septal rupture from 2011- 2021 who meet the inclusion and exclusion criteria were included in the study.

The investigators collected the details from electronic medical records. The scanned copies of old files, discharge summary and OPD records were used to collect the required information.

STATISTICAL PROCEDURE:

The statistical procedures were carried out in two steps:

1. **Compilation and presentation of data:**

Compilation of data was done systematically. Using a Microsoft Excel worksheet (Microsoft, USA, version 8.1), A master table was made; accordingly, the data was subdivided and distributed, presented in the form of individual tables and graphs.

2. **Statistical Analysis:**

Statistical analysis using IBM Statistical Package for Social Science (Statistics for Windows, version 21.0, Armonk, NY: IBM Corp.) was done, and the data was compared by applying statistical tests to find the statistical significance of the results.

Statistical tests employed for the obtained data:

Qualitative data will be expressed in terms of percentages and proportions.

- Quantitative data was expressed in terms of mean and standard deviation.
- Descriptive statistics of each variable were presented in terms of Mean, standard deviation, and standard error of the mean.
- The chi-square test was applied to analyze the statistical significance of the data.
- A p-value of <0.05 was considered statistically significant, whereas a p-value of

  <0.001 was considered as highly significant.

3. RESULTS

A retrospective study investigated the early outcomes and survival rates following the surgical repair of post-infarction ventricular septal rupture. The study included a total of 23 patients.

Table 1: Distribution of study patients according to their age.

Sr. No.	Age Group (Years)	No. of Patients n (%)	Mean Age	Standard Deviation
1.	41-50	1 (4.3)	**64.96**	**±8.315**
2.	51-60	5 (21.9)		
3.	61-70	13 (56.6)		
4.	71-80	2 (8.6)		
5.	81-90	2 (8.6)		
Total		**23 (100)**		

Distribution of study patients according to age: Out of the total 23 study patients, the largest proportion, 56.6% (n=13), fell within the age range of 61 to 70 years. This was followed by 21.9% for the 51-60 age group and 8.6% for the 71-80 age group, respectively. Furthermore, there was only one patient who was 44 years old, representing a percentage of 4.3%. (**Table 1) (Figure 1).**

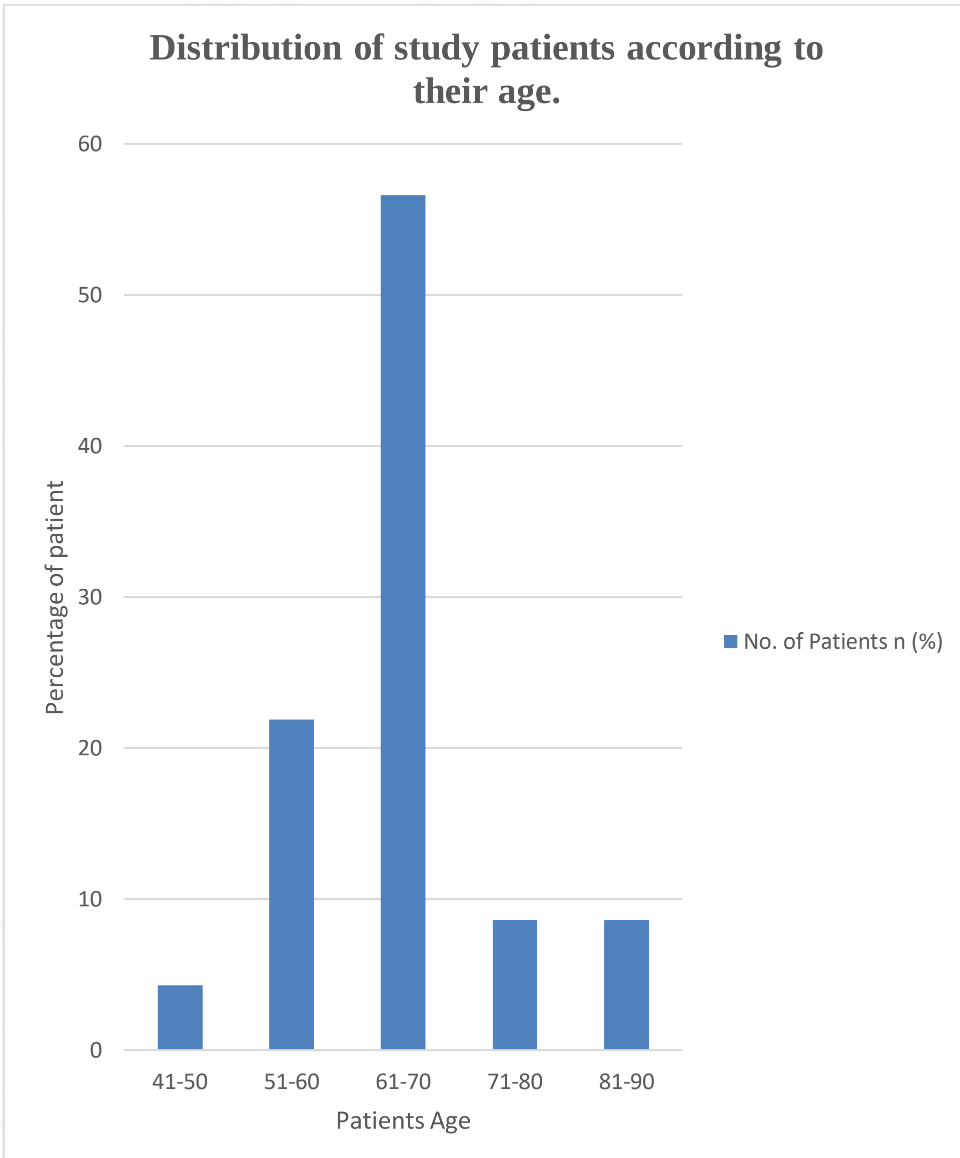


Figure 1. Distribution of study patients according to their age.

Table 2: Distribution of study patients according to their gender.

Sr. No.	Sex	No. of Patients (Percentage)
1	**Male**	13 (56.5)
2	**Female**	10 (43.5)
Total		23 (100)

Distribution of study patients according to gender: Out of 100% (n=23) patients, the recruitment of males and females was nearly balanced. Specifically, males accounted for 56.5% (n=13), slightly outnumbering females at 43.5% (n=10).

(Table 2) (Figure 2)

Figure 2: Distribution of study patients according to their gender.

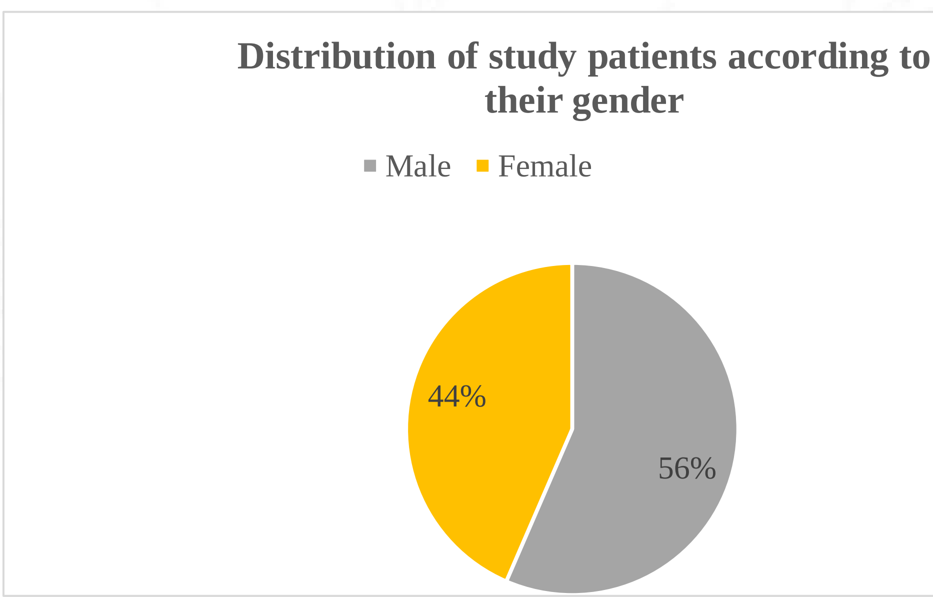


Table 3: Distribution of study patients according to their pre-operative medical complications.

Sr. No.	Pre-operative Medical Complications	Number of Patients (%)
1	**Hypertension**	17 (73.9)
2	**Diabetes Mellitus**	18 (78.3)
3	**Dyslipidemia**	15 (65.2)
4	**Smoking**	4 (17.4)
5	**Peripheral Arterial Disease**	1 (4.3)
6	**Chronic Kidney Disease**	9 (39.1)
7	**Cardiac Shock**	9 (39.1)
8	**Multi vessels Disease**	15 (65.2)

Distribution of study patients according to their pre-operative medical complications: Out of the total 100% (n=23) patients, the prevalence of medical conditions varied. Specifically, 78.3% (n=18) of patients had Diabetes Mellitus, followed by 73.9% (n=17) who were hypertensive. Additionally, 65.2% (n=15) of patients had both Dyslipidaemia and Multivessel disease. Chronic kidney disease and Cardiac Shock were observed in 39.1% (n=9) of patients each. Only a small proportion, 17.4% (n=4), had a smoking history. Lastly, one patient, representing 4.3% (n=1), had peripheral arterial disease. It's important to note that the records obtained from institutional computers lacked sufficient information regarding smoking history. **(Table 3) (Figure 3)**

Figure 3: Distribution of study patients according to their pre-operative medical complications.

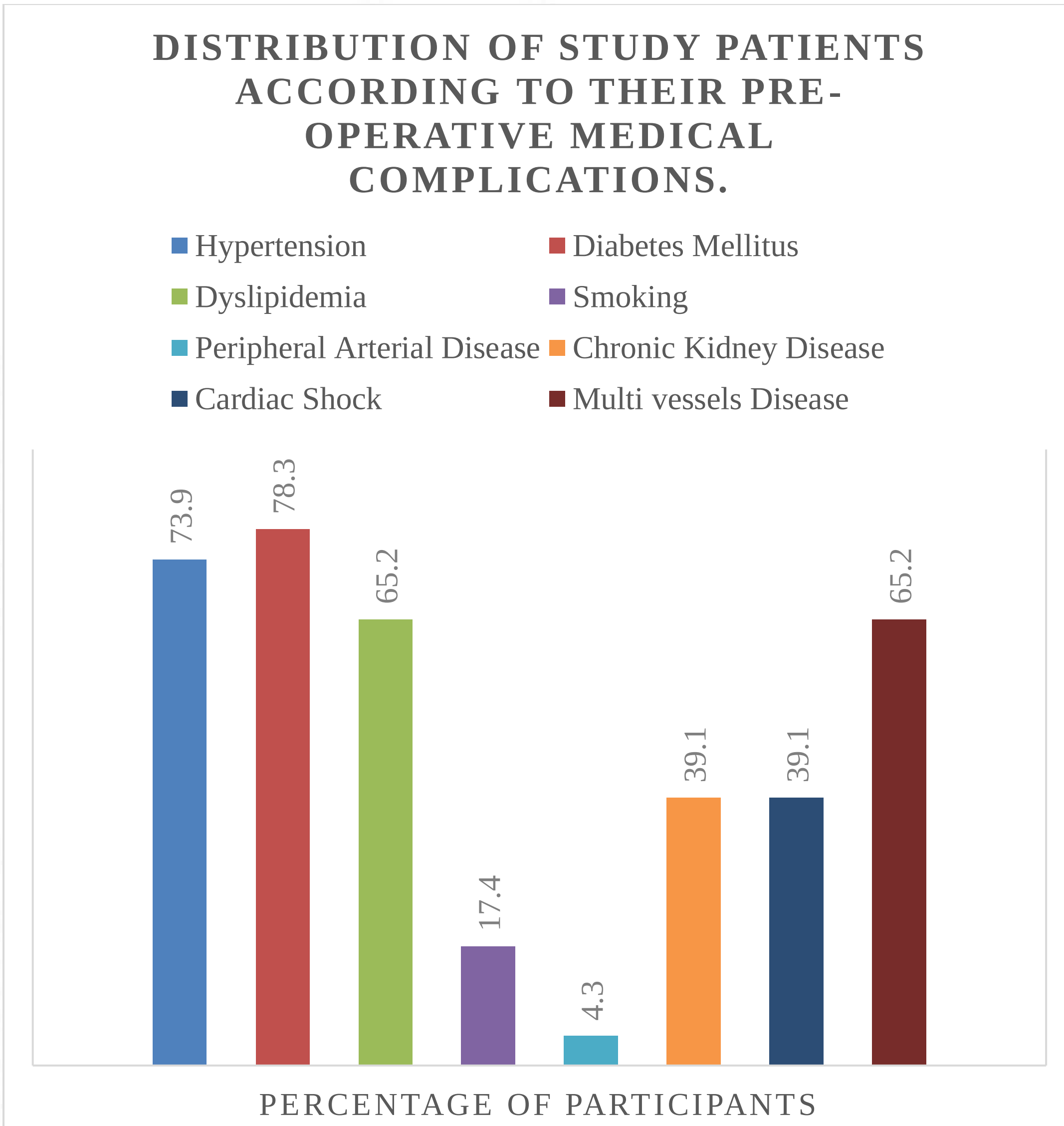


Table 4: Distribution of study patients according to their need for inotropic support before VSR repair.

Sr. No.	Pre-operative inotropic support	No. of patients n(%)
1	**Yes**	7 (30.4)
2	**No**	16(69.6)
Total		**23 (100)**

Distribution of study patients according to their need for inotropic support before VSR repair.

Of the total 100% (n=23) patients who underwent ventricular septal rupture repair, 30.4% (n=7) required ionotropic support pre-operatively. **(Table 4) (Figure 4)**

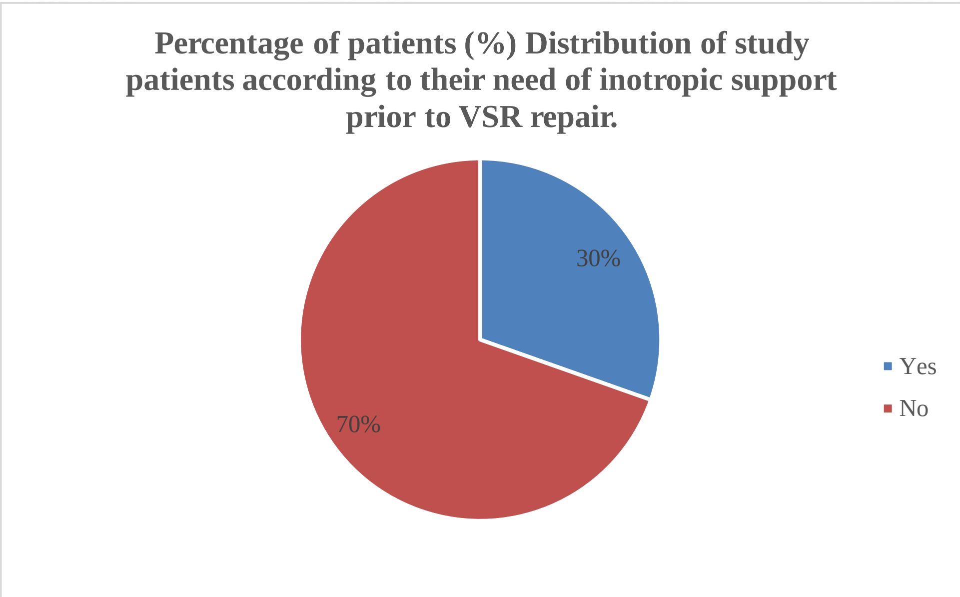


Figure- 4 Distribution of study patients according to their need for inotropic support before VSR repair.

Table 5. Distribution of study patients according to their type of myocardial infarct (MI).

Sr. No.	Pre-operative type of MI	No. of patients n(%)
1	Inferior Wall Myocardial Infarction	9 (39.1)
2	Anterior Wall Myocardial Infarction	14(60.9)
Total		23 (100)

Distribution of study patients according to their type of myocardial infarction (MI).

Out of the total 100% (n=23) patients, the majority, 60.9% (n=14), had pre-operative IWMI, while a smaller number, 39.1% (n=9), had AWMI type of Myocardial infarction were observed pre-operatively. **(Table 5) (Figure 5).**

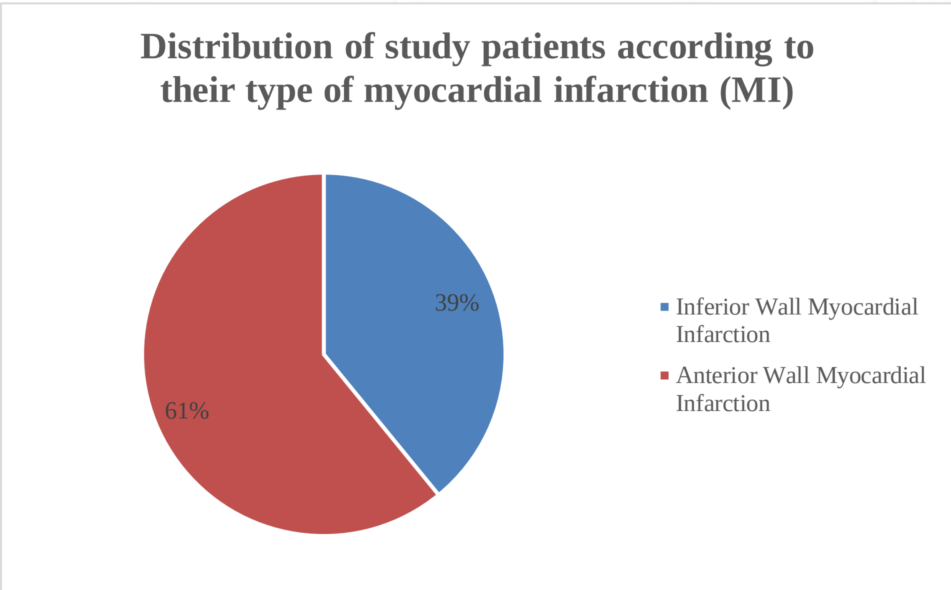


Figure-5 Distribution of study patients according to their type of myocardial infarction (MI).

Table 6. Distribution of study patients according to their ventricular functional status.

Sr No	Ventricular functional status	No of patients
1	**Normal biventricular function**	7 (30.1%)
2	**Only Left Ventricle dysfunction**	6 (25.8%)
3	**Only Right Ventricle dysfunction**	4 (17.2%)
4	**Bi Ventricle dysfunction**	6 (25.8%)
Total		**23 (100)**

Distribution of study patients according to their ventricular functional status. Out of the total, seven patients, which accounts for 30.1%, did not have Ventricular dysfunction. There were six patients (25.8%) with only Left Ventricle Dysfunction. Additionally, four patients (17.2%) had only Right Ventricle Dysfunction. A total of 6 patients (25.8% of the total) exhibited Bi-Ventricular Dysfunction. **(Table 6) (Figure 6)**

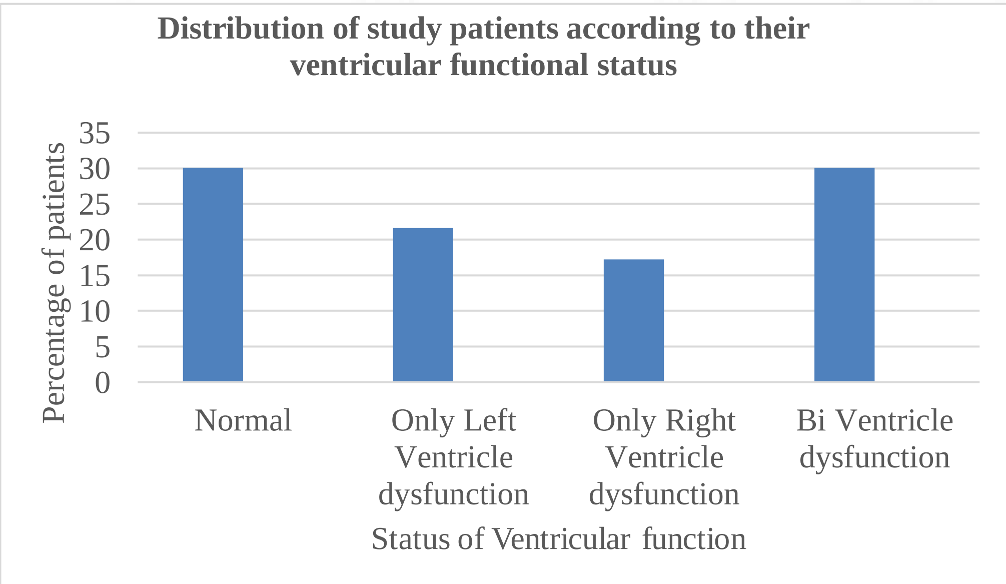


Figure 6. Distribution of study patients according to their left and right ventricular dysfunction.

Table 7. Distribution of study patients according to their previous procedure, such as PCI/CABG, before the surgery.

Sr. No.	Previous Surgery (PCI/CABG)	No. of patients n (%)
1	**No**	15 (65.4)
2	**PCI-LAD**	5 (21.7)
3	**PCI-RCA**	1 (4.3)
4	**PCI-LAD and RCA**	1 (4.3)
5	**CABGX2G**	1(4.3)
Total		**23 (100)**

Distribution of study patients according to their previous procedure, such as PSI/CABG, before the surgery.

The largest group, comprising 15 patients (65.4%), had no prior surgical interventions. Five patients (21.7%) had undergone previous PCI, specifically in the Left Anterior Descending (LAD) artery. A single patient (4.3% of the total) had a history of PCI in the Right Coronary Artery (RCA), while another individual (4.3% of the total) had received PCI in both LAD and RCA. Finally, one patient (4.3% of the total) had undergone a Coronary Artery Bypass Graft surgery with two grafts to LAD and RCA (CABGX2) 18 years ago.

(Table 7) (Figure 7)

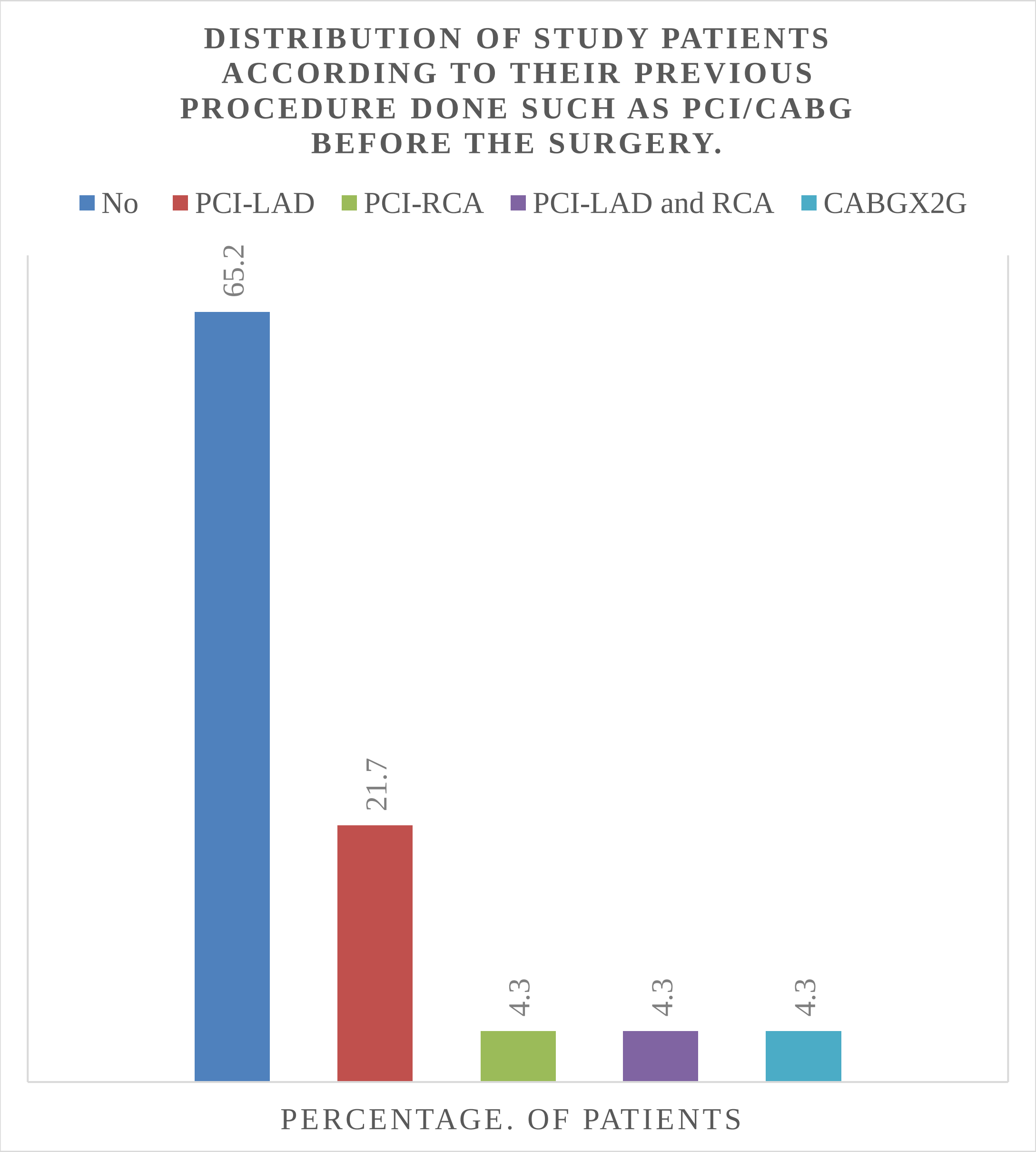


Figure 7: Distribution of study patients according to their previous procedure, such as PSI/CABG, before the surgery.

Table 8. Distribution of study patients according to their observed mitral regurgitation.

Sr. No.	Mitral Regurgitation	No. of patients n (%)
1	Mild	17 (73.9)
2	Moderate	6 (26.1)
Total		23 (100)

Distribution of study patients according to their observed mitral regurgitation. Among all 23 patients, 17 (73.9%) exhibited Mild Mitral Regurgitation, while six patients (26.1%) had Moderate Mitral Regurgitation.

(Table 8) (Figure 8)

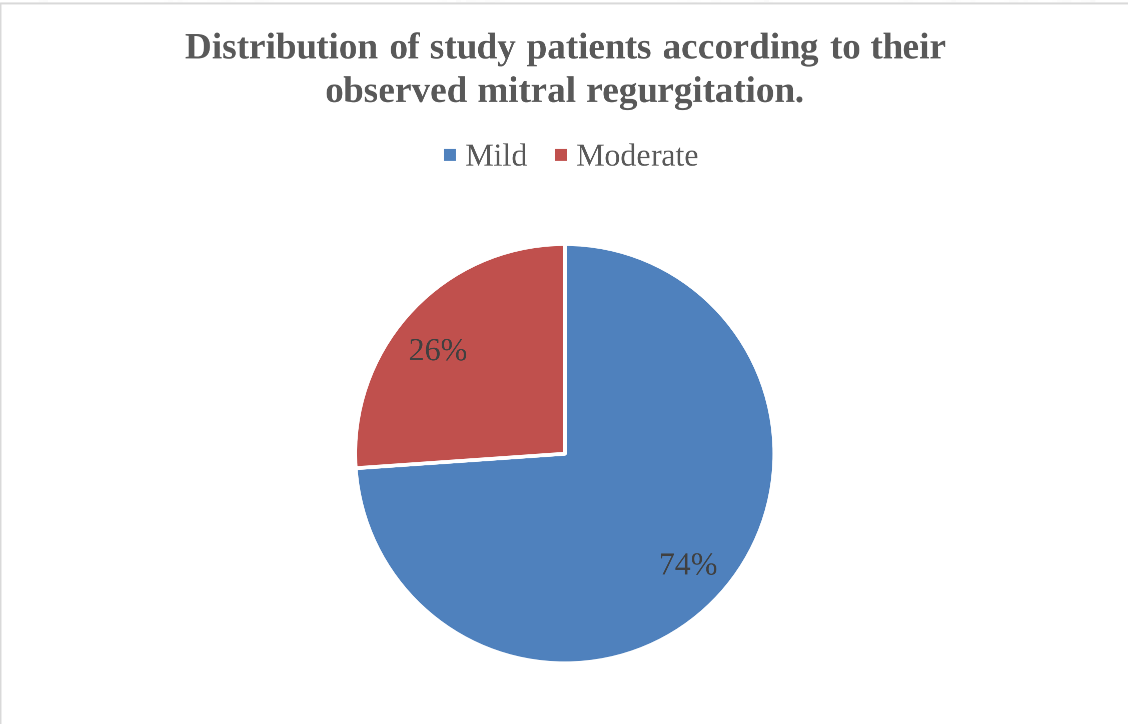


Figure 8: Distribution of study patients according to their observed mitral regurgitation.

Table 9. Distribution of study patients according to their left ventricular ejection fraction (LVEF) before the surgery.

Sr. No.	LVEF	No. of patients n(%)
1	**Less than 30%**	2(8.9)
2	**30-39 %**	1(4.3)
3	**40-49 %**	8(34.9)
4	**50% and above**	12(51.9)
Total		**23 (100)**

Distribution of study patients according to their left ventricular ejection fraction (LVEF) before the surgery.

Among the entire patient cohort, which comprised 23 individuals, 8.9% (n=2) exhibited a severe LVEF (<30%). Additionally, 4.3% (n=1) had a moderate LVEF (30-39%), 34.9% (n=8) displayed a mild LVEF (40-49%), and the majority, 51.9% (n=12), had a normal LVEF (>50%). **(Table 9) (Figure 9)**

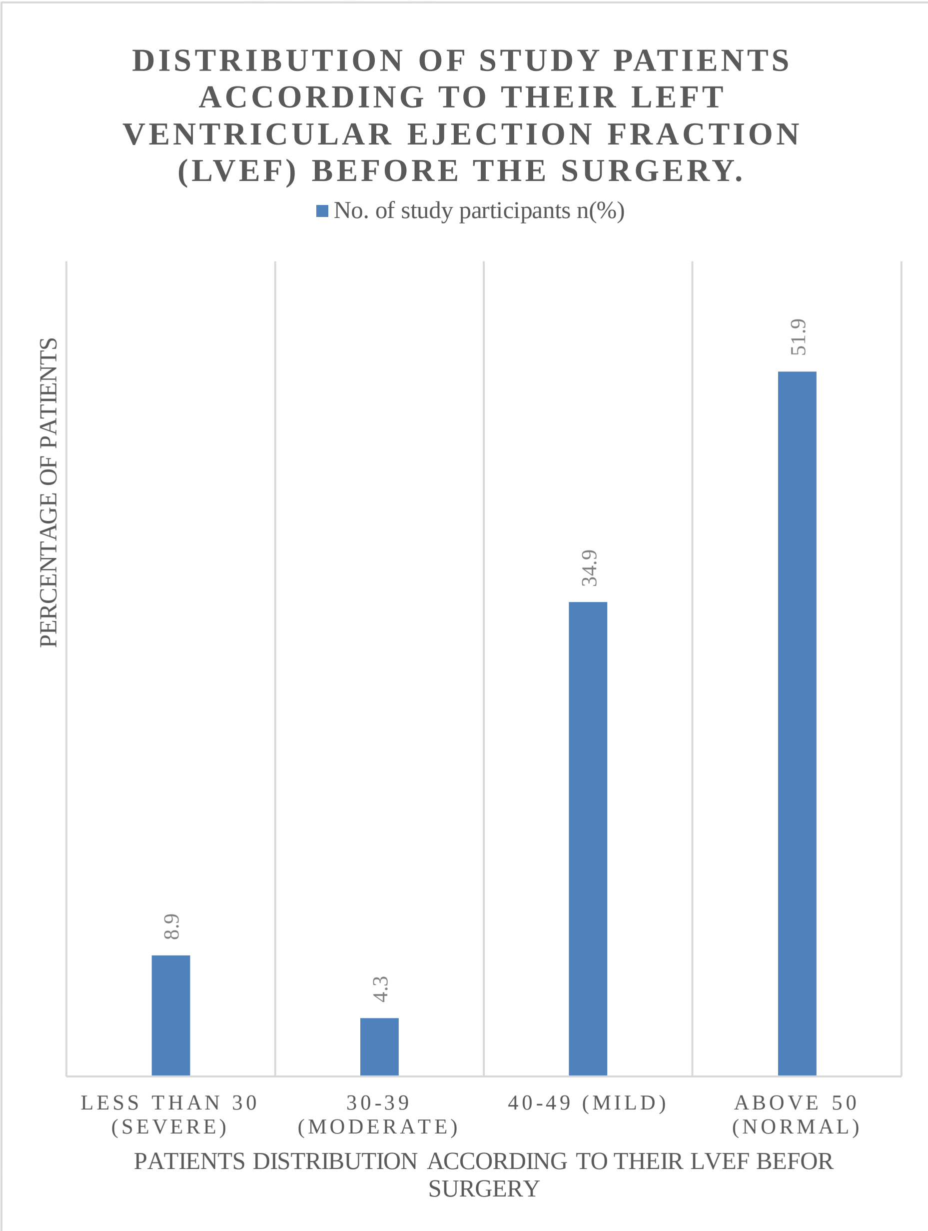


Figure 9. Distribution of study patients according to their left ventricular ejection fraction (LVEF) before the surgery.

Table 10. Distribution of study patients according to size of VSR defect.

Sr. No.	Size of VSR defect	No. of patients n (%)
1	**Less than10 mm**	4 (17.7)
2	**10-20 mm**	18 (77.9)
3	**More than 20 mm**	1 (4.3)
***Only one patient had Multiple VSR**		
Total		**23 (100)**

Distribution of study patients according to size of VSR defect. Among all 23 patients, the majority, 77.9% (n=18), had a VSR size between 10 to 20 mm. VSR sizes less than 10 mm were observed in 17.7% (n=4) of the patients, and a single patient, accounting for 4.3%, had a VSR size exceeding 20 mm. **(Table 10) (Figure 10).**

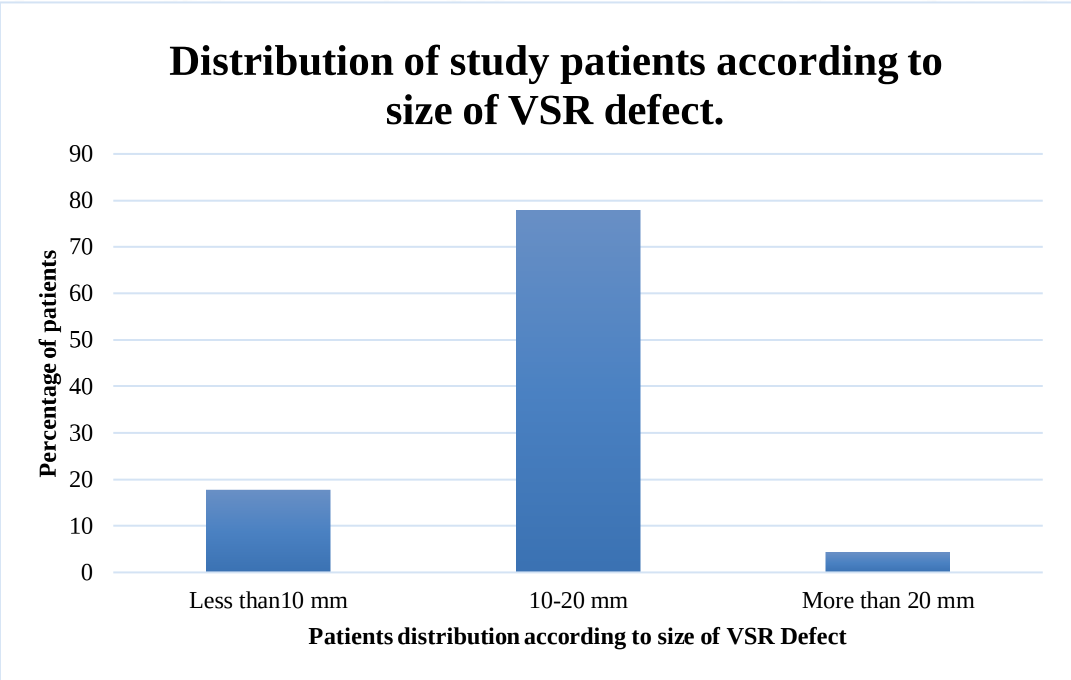


Figure 10. Distribution of study patients according to size of ventricular septal rupture.

Table 11. Distribution of study patients according to the time interval between MI and VSR repair.

	The time interval between MI and VSR repair	No. of study patients n (%)
1	**Less than seven days**	5 (21.7)
2	**8-15**	7 (30.4)
3	**16-30 days**	4 (17.5)
4	**More than 30**	7 (30.4)
Total		**23 (100)**

Distribution of study patients according to the time interval between MI and VSR repair. Among all 100% (n=23) patients, the time intervals between MI and VSR repair were distributed as follows: less than seven days for 21.7% (n=5) of patients, 8-15 days for 30.4% (n=7), 16-30 days for 17.5% (n=4), and more than 30 days for 30.4% (n=7). **(Table 11) (Figure 11).**

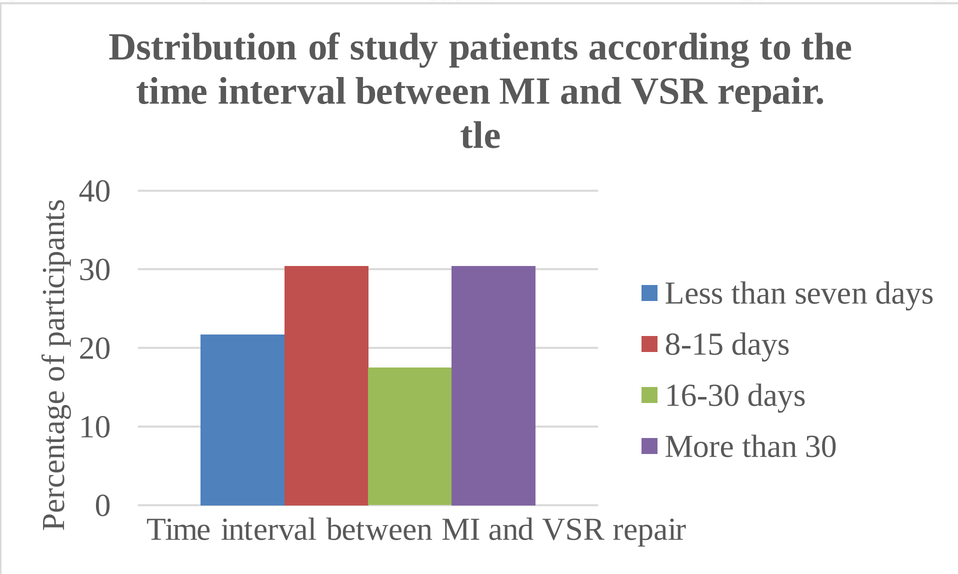


Figure 11. Distribution of study patients according to the time interval between MI and VSR repair.

Table 12. Distribution of study patients according to the location of ventricle septal rupture (VSR).

Sr. No.	Location of VSR	No. of patients n (%)
1	**Posterobasal**	10 (43.5)
2	**Apical**	9 (39.1)
3	**Mid Muscular**	4 (17.4)
Total		**23 (100)**

Distribution of study patients according to the location of ventricle septal rupture (VSR).: Out of all 100% (n=23) patients, 43.5% (n=10) had a posterobasal VSR location, followed by 39.1% (n=9) of patients with an Apical VSR location. VSR located at the mid-muscular region was observed in 17.4% (n=4) of patients.

(Table 12) (Figure 12).

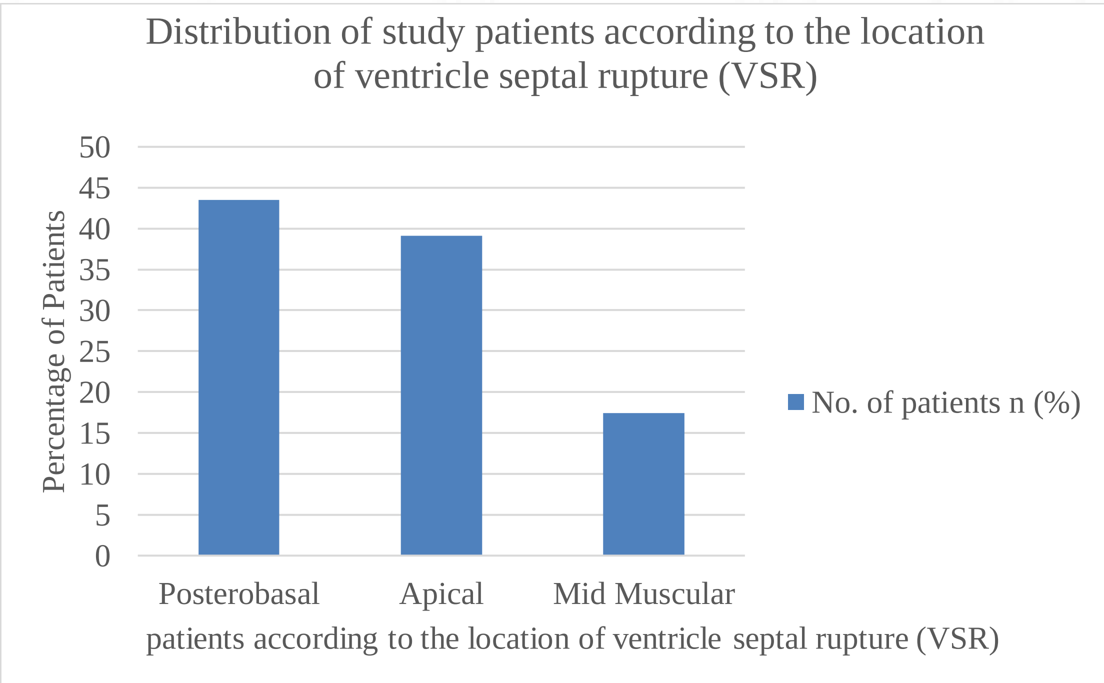


Figure 12. Distribution of study patients according to the location of ventricle septal rupture (VSR).

Table 13. Distribution of study patients according to the type of VSR repair.

Sr. No.	Type of VSR repair	No. of patients n(%)
1	**Double Patch**	2 (8.7)
2	**Patch Closure**	19 (82.6)
3	**Patch Closure + Apical exclusion**	2 (8.7)
Total		**23 (100)**

Distribution of study patients according to the type of VSR repair.

Out of all 100% (n=23) patients, the majority, 82.6% (n=19), underwent Patch closuer VSR repair. Double patch and Patch closuer with apical exclusion VSR repairs were observed in 17.4% (n=4) of the patients, with each method accounting for 8.7% (n=2) of cases. **(Table 13) (Figure 13).**

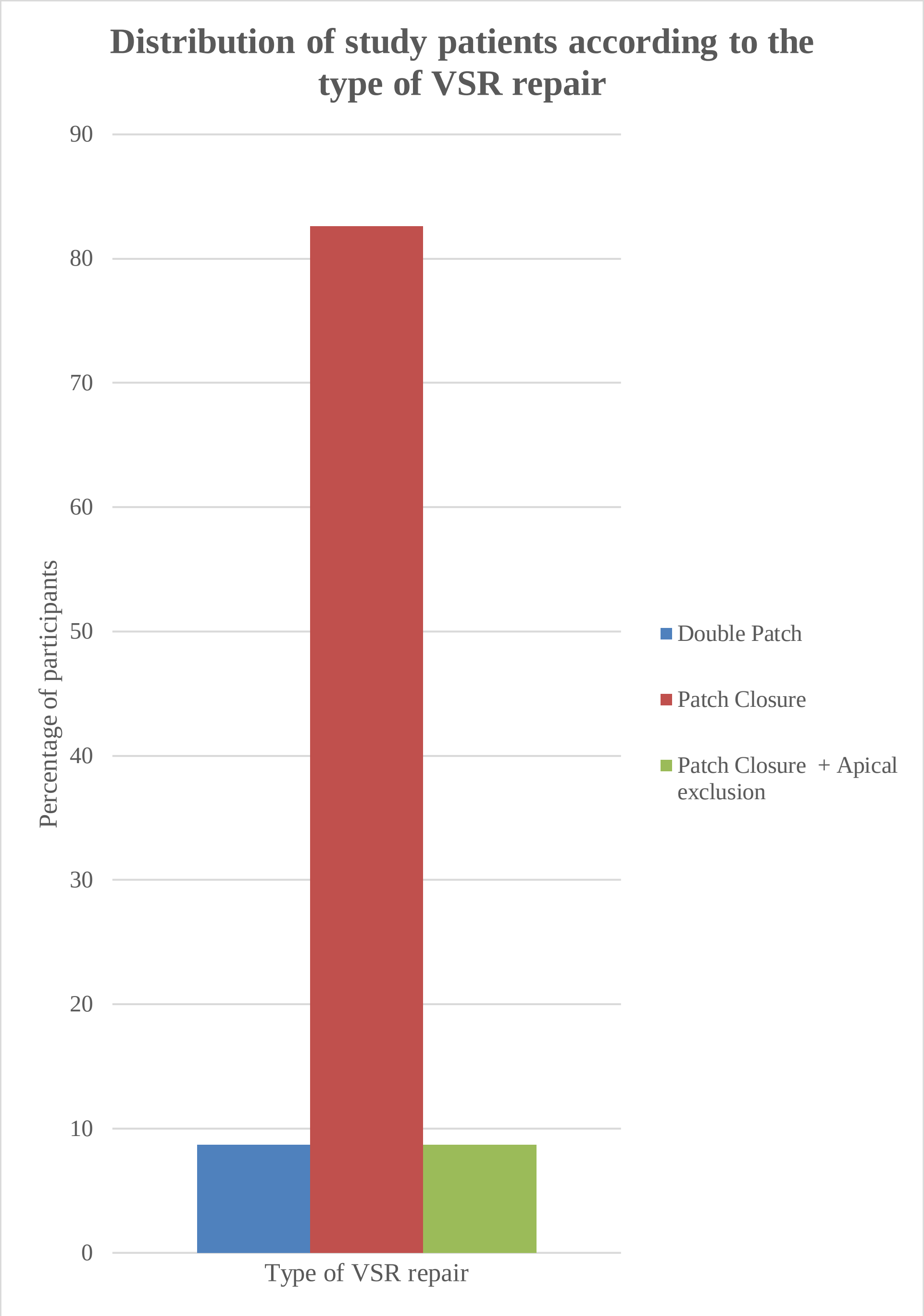


Figure 13. Distribution of study patients according to the type of VSR repair.

Table 14. Distribution of study patients according to their concomitant procedure.

Sr. No.	Type of repair	No. of patients n(%)
1	**Nil**	6 (26.1)
2	**CABG X 1G**	8 (34.9)
3	**CABG X 2G**	2 (8.7)
4	**CABG X 3G**	3 (13)
5	**CABG X 4G**	1 (4.3)
6	**CABG + LV Anuerysmoraphy**	2 (8.7)
7	**CABG x 3G + MV repair**	1 (4.3)
Total		**23 (100)**

Distribution of study patients according to their concomitant procedure.

Among all 100% (n=23) patients, 26.1% (n=6) underwent no concomitant procedure. A total of 34.9% (n=8) underwent CABG surgical procedures, with varying numbers of grafts: 8.7% (n=2) had one graft, 13% (n=3) had two grafts, and 4.3% (n=1) had four grafts. Additionally, 8.7% (n=2) of patients had undergone CABG with Aneurysmorrhaphy. Furthermore, only one patient, representing 4.3% (n=1), had undergone CABG with three grafts and MV repair.

(Table 14) (Figure 14).

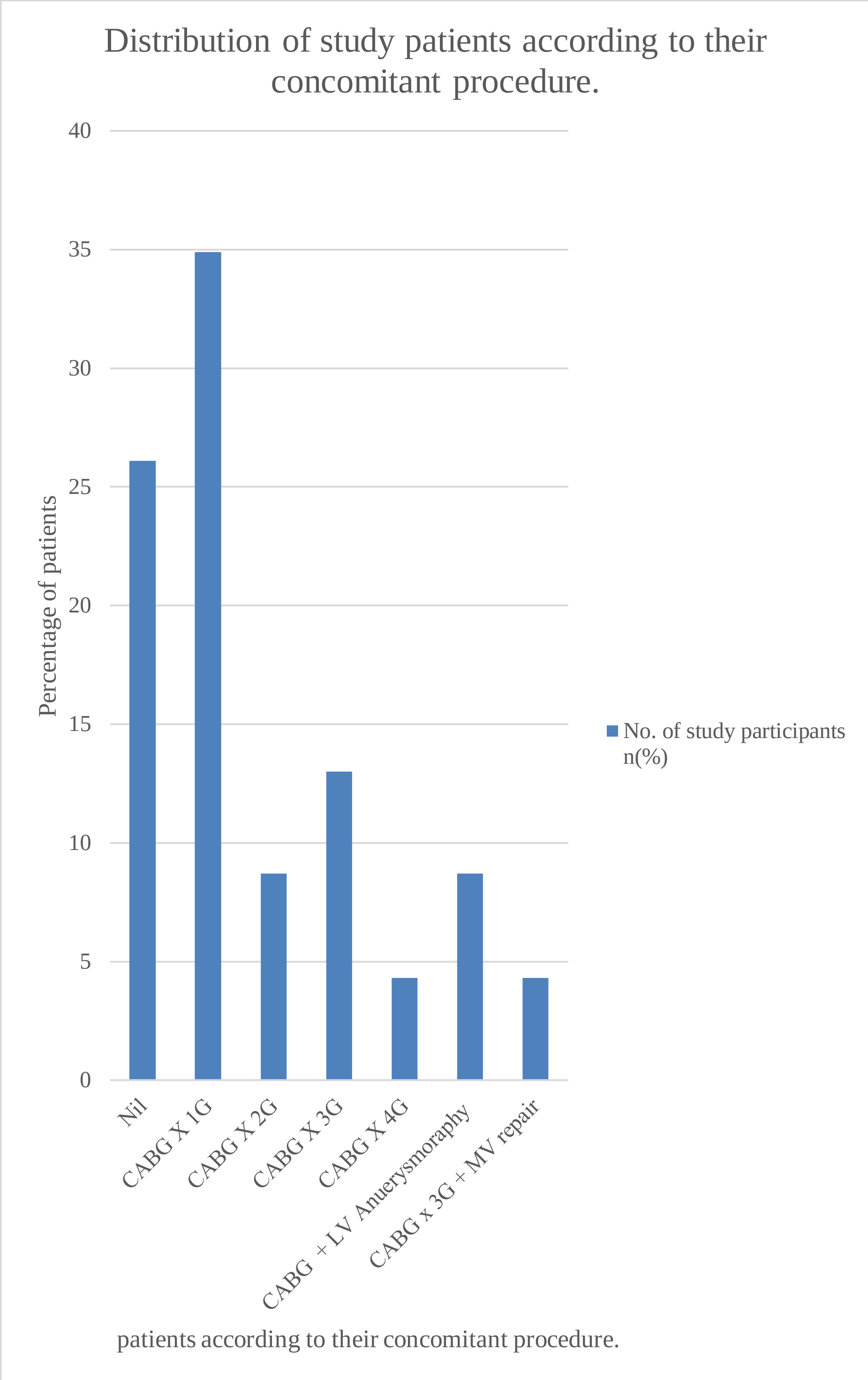


Figure 14. Distribution of study patients according to their concomitant procedure.

Table 15. Distribution of study patients according to their required CPB time.

Sr. No.	Time duration (minutes)	No. of patients n (%)
1	Less than 90 minutes	2 (8.7)
2	91-120 minutes	7 (30.5)
3	More than 120	14(60.5)
Total		23 (100)

Distribution of study patients according to size of ventricular septal rupture.

Out of 100 patients (n=23), the majority, 60.5% (n=14), required a CPB time exceeding 120 minutes. Following them, 30.5% (n=7) of patients required CPB time ranging from 90 to 120 minutes, while only 8.7% (n=2) needed CPB time less than 90 minutes. **(Table 15) (Figure 15)**

.

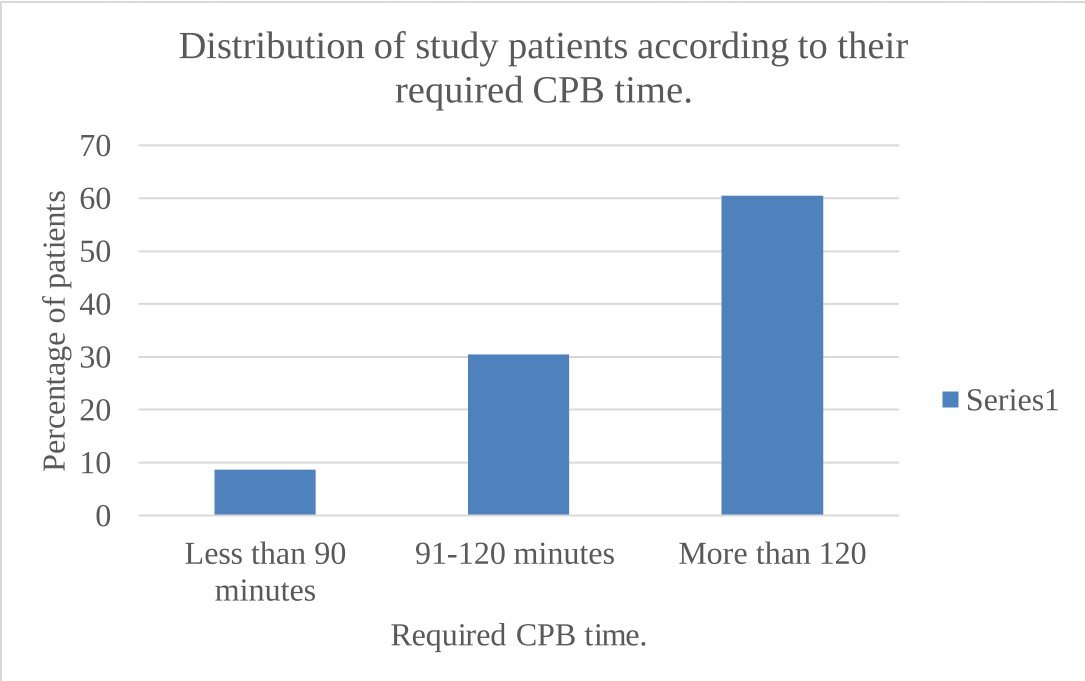


Figure- 15 Distribution of study patients according to size of ventricular septal rupture.

Table 16. Distribution of study patients according to their required ACC time.

Sr. No.	Time duration (minutes)	No. of patients n (%)
1	**Less than 60**	3 (13.60)
2	**61-90**	9 (40.90)
3	**91-120**	7 (31.80)
4	**More than 120**	3 (13.60)
Total		**22 (100)**
***Note: 1 patient was done under fib arrest; hence, n=22 only.**		

Distribution of study patients according to their required ACC time. Of 100% (n=22) patients, 13.60% (n=3) required less than 60 minutes of ACC time. Additionally, 40.90% (n=9) of patients needed ACC time in the 61-90 minutes range, while 31.80% (n=7) required ACC time within the 91-120 minutes range. Three patients, accounting for 13.60% (n=3), needed ACC time exceeding 120 minutes. Only one patient was under fib arrest, hence n=22. **(Table 16) (Figure 16).**

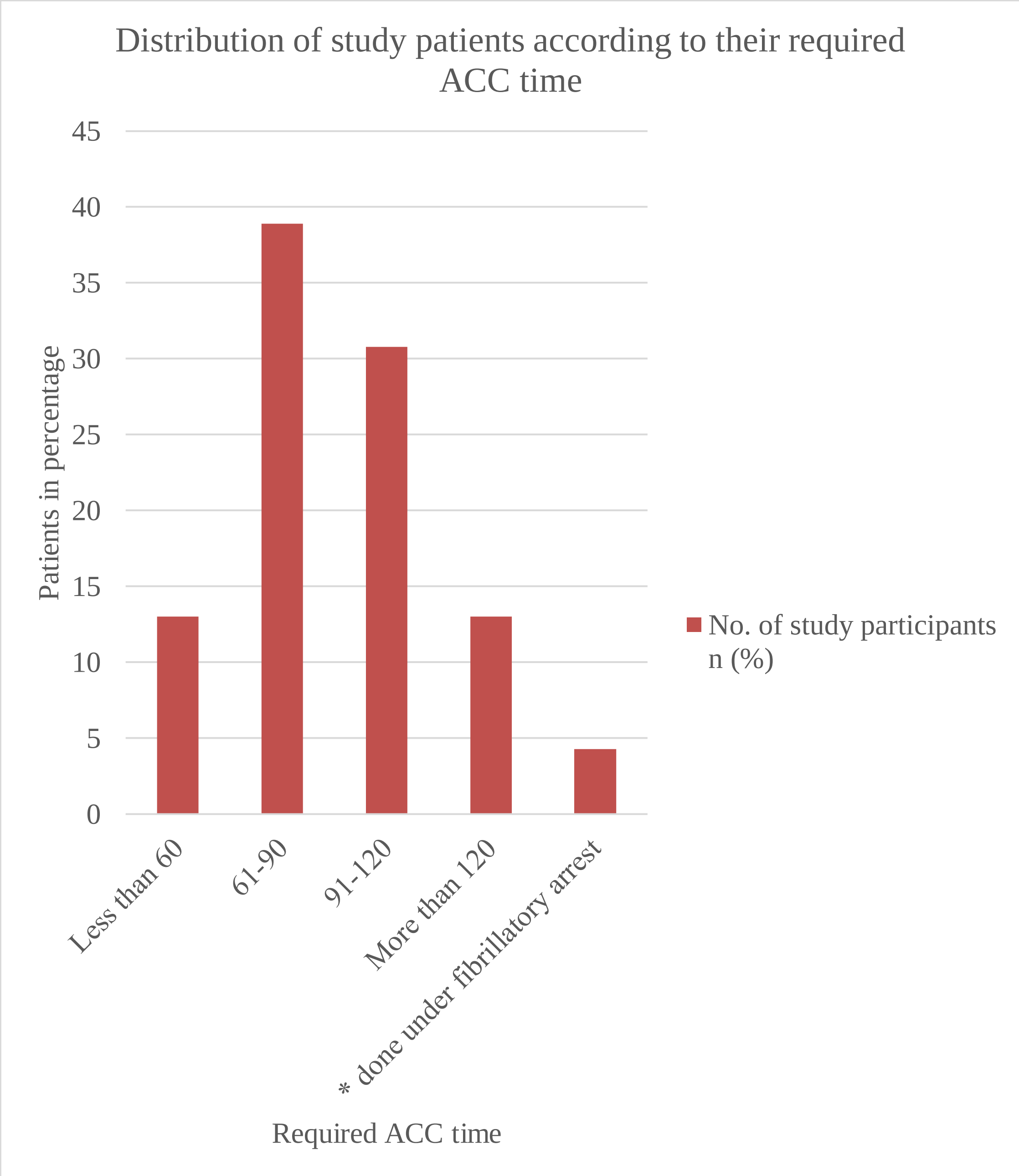


Figure 16. Distribution of study patients according to their required ACC time.

Table 17. Distribution of study patients according to their requirement of intra-aortic balloon pump (IABP) intra-operatively for weaning from CPB.

Sr. No.	Intra-operative IABP	No. of patients n (%)
1	**No**	18 (77.5)
2	**Yes**	5 (21.5)
Total		**23 (100)**

Distribution of study patients according to their requirement of intra-aortic balloon pump (IABP) intra-operatively. Among all 100% (n=23) patients, 77.5% (n=18) did not require intra-operative IABP, while the remaining 21.5% (n=5) needed intra-operative IABP support for weaning of from CPB.

(Table 17) (Figure 17).

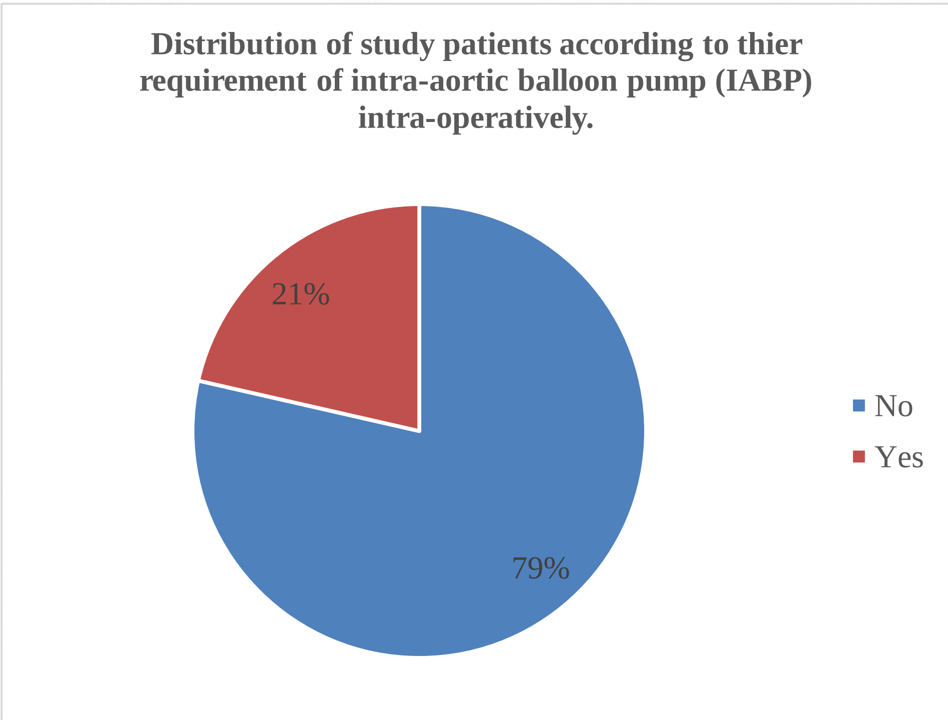


Figure 17. Distribution of study patients according to their requirement of intra-aortic balloon pump (IABP) intra-operatively.

Table 18. Distribution of study patients according to their required time duration in days for Ventilation. Support.

Sr. No.	Time duration of Ventilation Support (Days)	No of patients n(%)
1	**Less than one day**	10 (43.6)
2	**1-2 days**	5 (21.7)
3	**3-7 days**	3(13)
4	**More than seven days**	5 (21.7)
Total		**23 100)**

Distribution of study patients according to their required time duration in days for Ventilation support.

Of all 100% (n=23) patients, 43.6% (n=10) required ventilation for less than one day. The remaining patients needed ventilation for different durations: 21.7% (n=5) for 1.2 days, 13% (n=3) for 3-7 days, and 21.7% (n=5) for more than seven days.

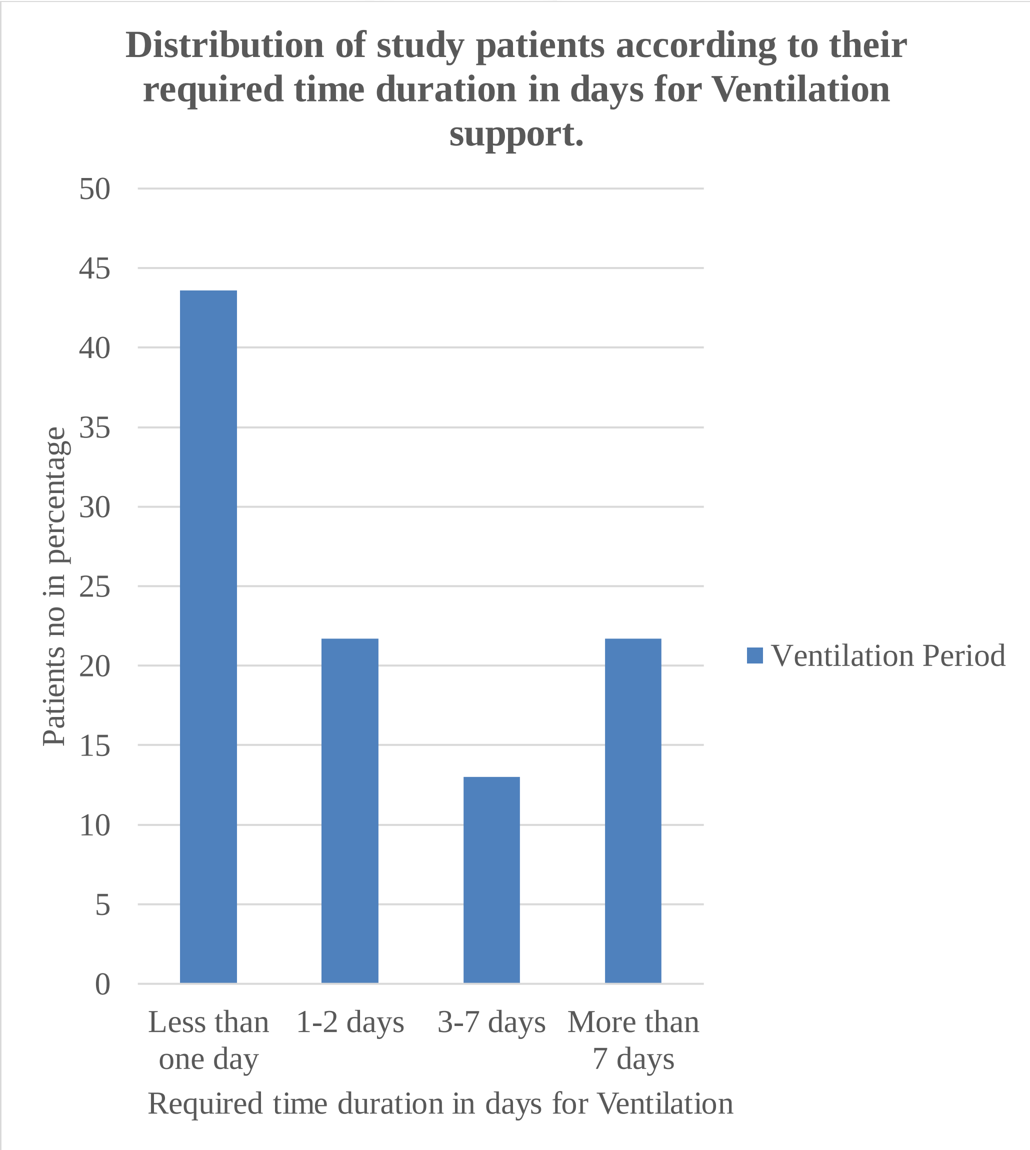


Figure 18. Distribution of study patients according to their required time duration in days for Ventilation support

Table 19. Distribution of study patients according to their required time duration in days for ICU stay.

Sr. No.	Time duration of ICU stay (Days)	No. of patients n (%)
1	**Less than 7**	15 (65.5)
2	**7-15 days**	1(4.3)
3	**More than 15 days**	7 (30.2)
Total		**23 (100)**

Distribution of study patients according to their ICU stay.

Out of all 100% (n=23) patients, the majority, 65.5% (n=15), had an ICU stay of less than seven days. Approximately 30.2% (n=7) of the patients had an ICU stay exceeding 15 days, and only one patient, accounting for 4.3%, stayed in the hospital for 7-15 days.

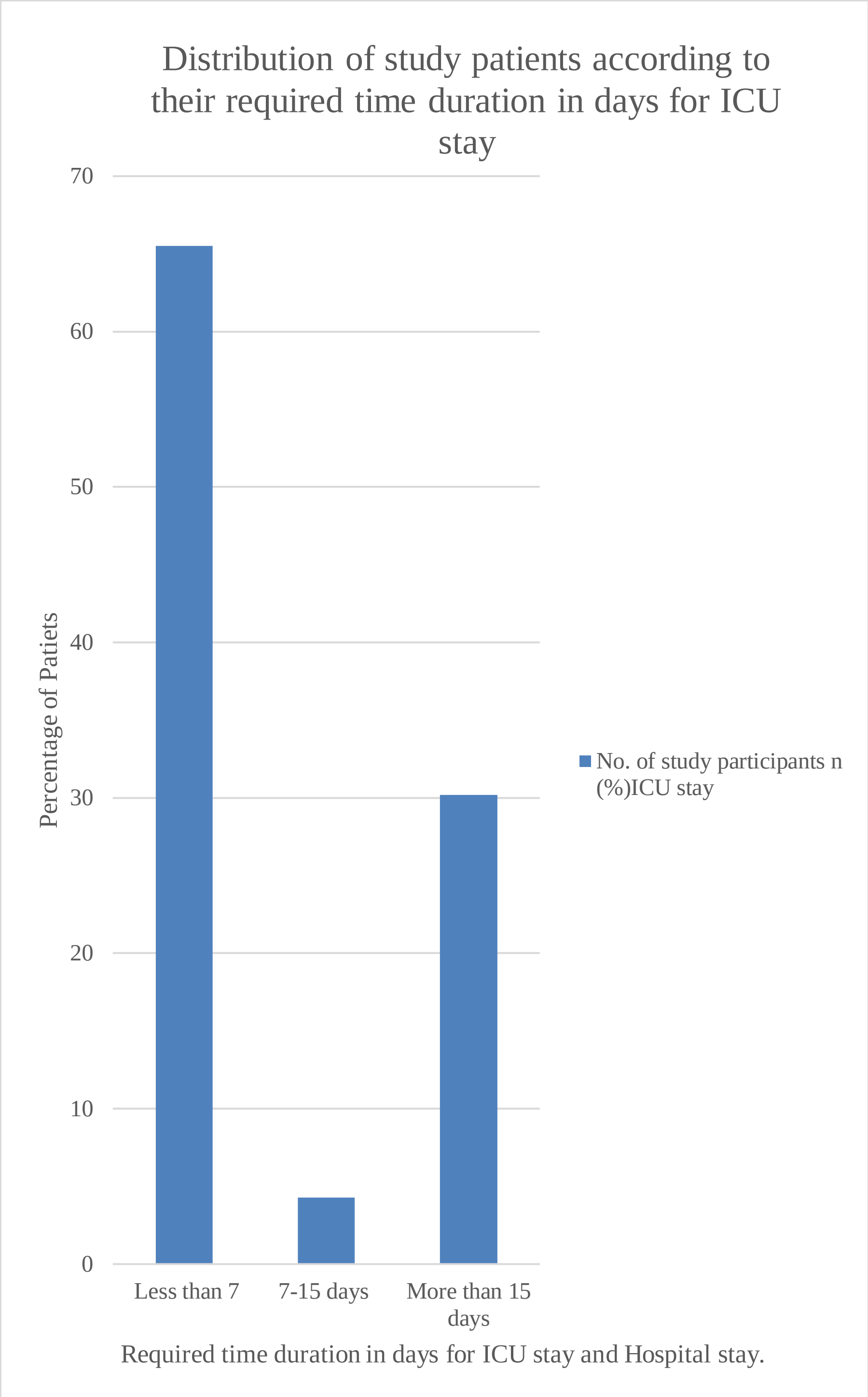


Figure 19. Distribution of study patients according to their required time duration in days for ICU stay.

Table 20. Distribution of study patients according to their required time duration in days for Hospital stay.

Sr. No.	Time duration of Hospital Stay (Days)	No. of patients n (%)
1	**Less than ten days**	4(17.4)
2	**10-20 days**	8 (34.4)
3	**More than 20 days**	11(47.2)
Total		**23**

Distribution of study patients according to their required time duration in days for Hospital stay.

Out of all 100% (n=23) patients, 47.2% (n=15) had a hospital stay exceeding seven days. Following them, 34.4% (n=8) stayed in the hospital for 10-20 days, and 17.4% (n=4) had a hospital stay of less than ten days.

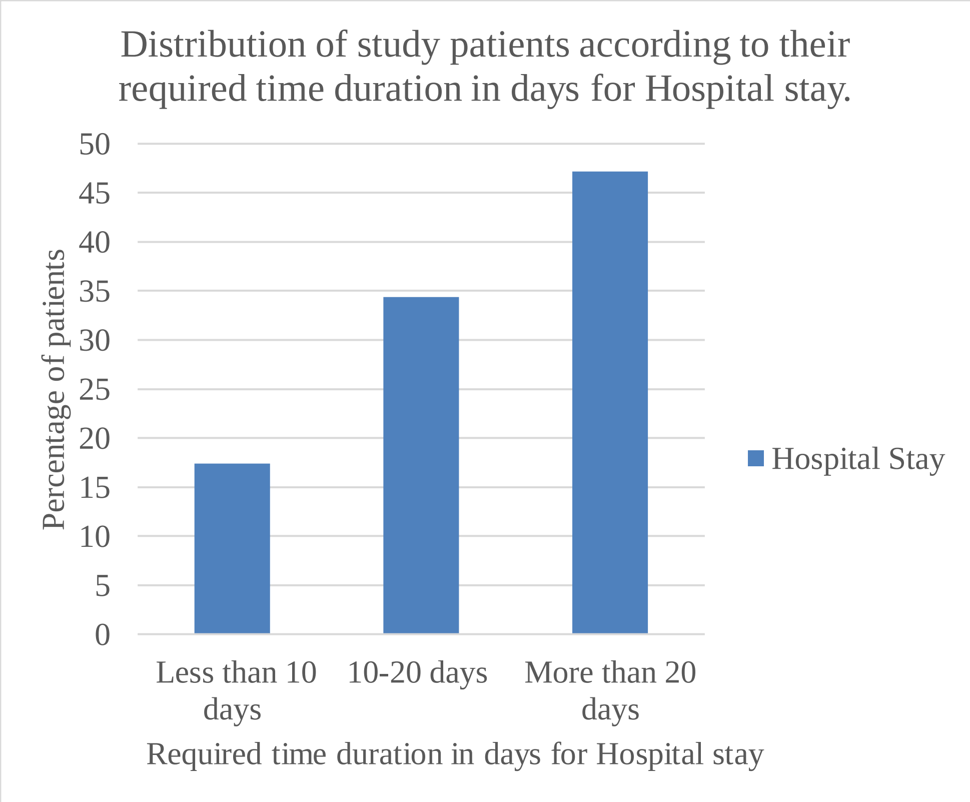


Figure 20. Distribution of study patients according to their required time duration in days for Hospital stay

Table 21. Distribution of study patients according to re-exploration after surgery.

Sr. No.	Re-exploration after surgery	No. of patients n (%)
1	**No**	22 (95.7)
2	**Yes**	1 (4.3)
Total		**23 (100)**

Distribution of study patients according to their Re-exploration immediately after surgery.

Among all 100% (n=23) patients, only one individual required re-exploration, accounting for 4.3%. The re-exploration of the patient was done on the same day for excessive bleeding. The remaining 95.7% (n=22) patients did not need re-exploration in this study. **(Table 21) (Figure 21).**

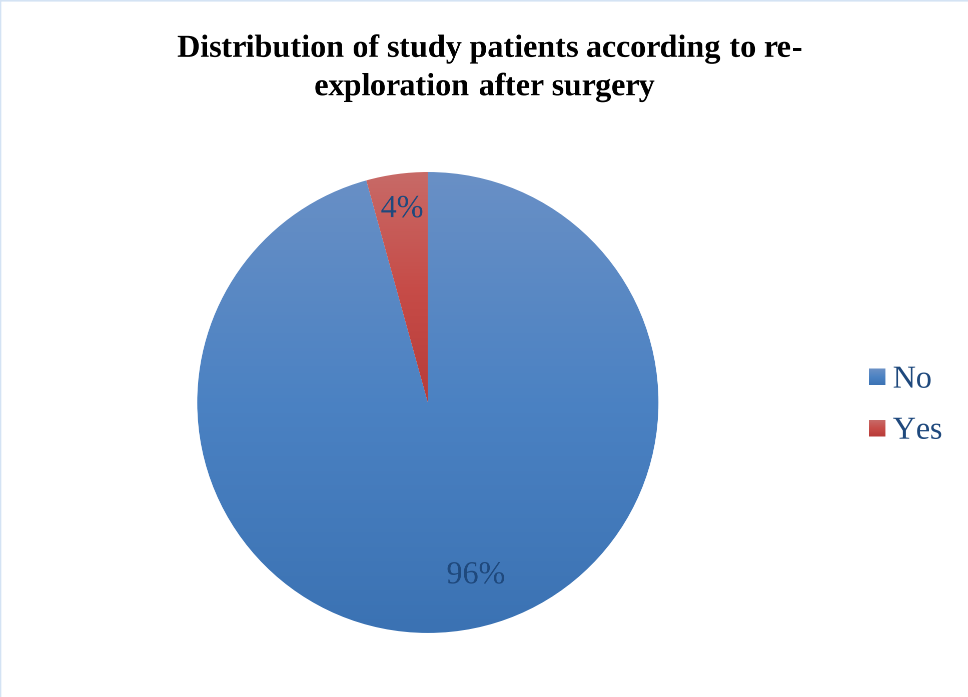


Figure 21. Distribution of study patients according to their Re-exploration immediately after surgery.

Table 22. Distribution of study patients according to their post-operative left Ventricular function.

Sr No	Left Ventricular Function	No. of patients n (%)
1	**Good**	5(21.7)
2	**Mild**	5(21.7)
3	**Moderate**	7(30.5)
4	**Severe**	6(26.1)
	Total	**23 (100)**

Distribution of study patients according to their post-operative left Ventricular function

Among all 100% (n=23) patients, 21.7% (n=5) had good left ventricle function. The distribution of LV dysfunction was as follows: mild dysfunction in 21.7% (n=5) of patients, moderate dysfunction in 30.5% (n=7) of patients and severe dysfunction in 26.1% (n=6)

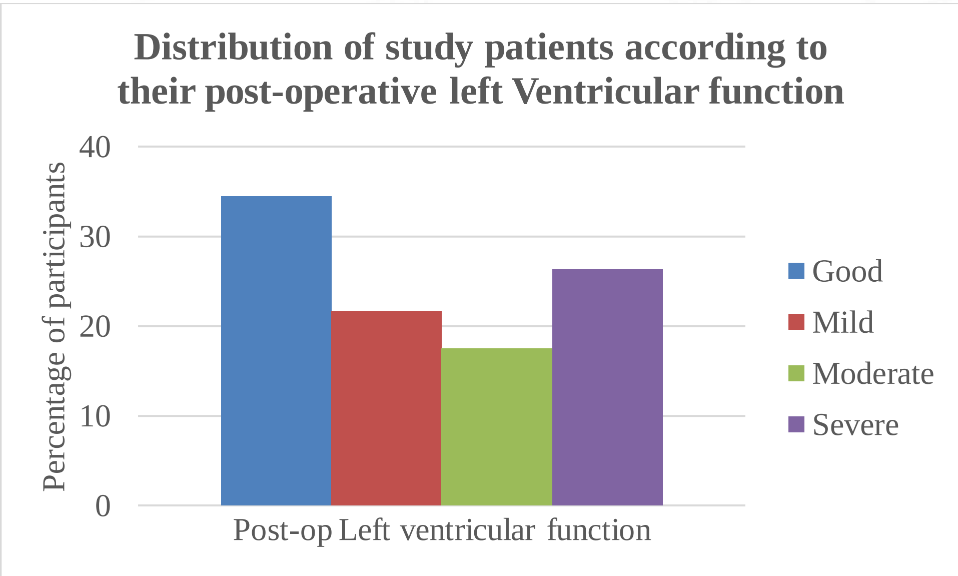


Figure 22. Distribution of study patients according to their post-operative left Ventricular function

Table 23. Distribution of study patients according to their post-operative right ventricular function

Sr. No.	Right Ventricular Function	No. of patients n (%)
1	Good	8(34.5)
2	Mild	4(17.5)
3	Moderate	5(21.7)
4	Severe	6(26.3)
Total		23 (100)

Distribution of study patients according to their post-operative right ventricular function

Among all 100% (n=23) patients, 34.5% (n=8) had good right ventricle function. The distribution of LV dysfunction was as follows: severe dysfunction in 26.3% (n=6) of patients, moderate dysfunction in 21.7% (n=5) and mild dysfunction in 17.5% (n=4) of patients.

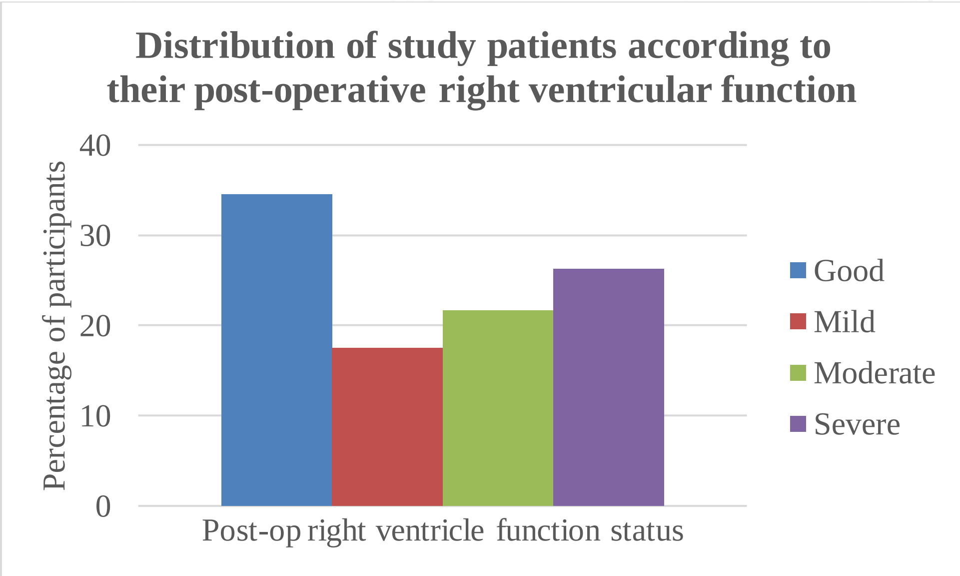


Figure 23. Distribution of study patients according to their Right ventricular function.

Table 24. Distribution of the study patient according to the biventricular function status.

Sr. No.	Biventricular Function status	No. of patients n (%)
1	Good Biventricular Function	4 (17.5)
2	Severe Biventricular Dysfunction	5 (21.5)

Distribution of the study patient according to the biventricular function status. Among all 100% (n=23) patients, 17.5% (n=4) had a good biventricular function, while the remaining 21.5% (n=5) had severe biventricular dysfunction. **(Table 24) (Figure 24).**

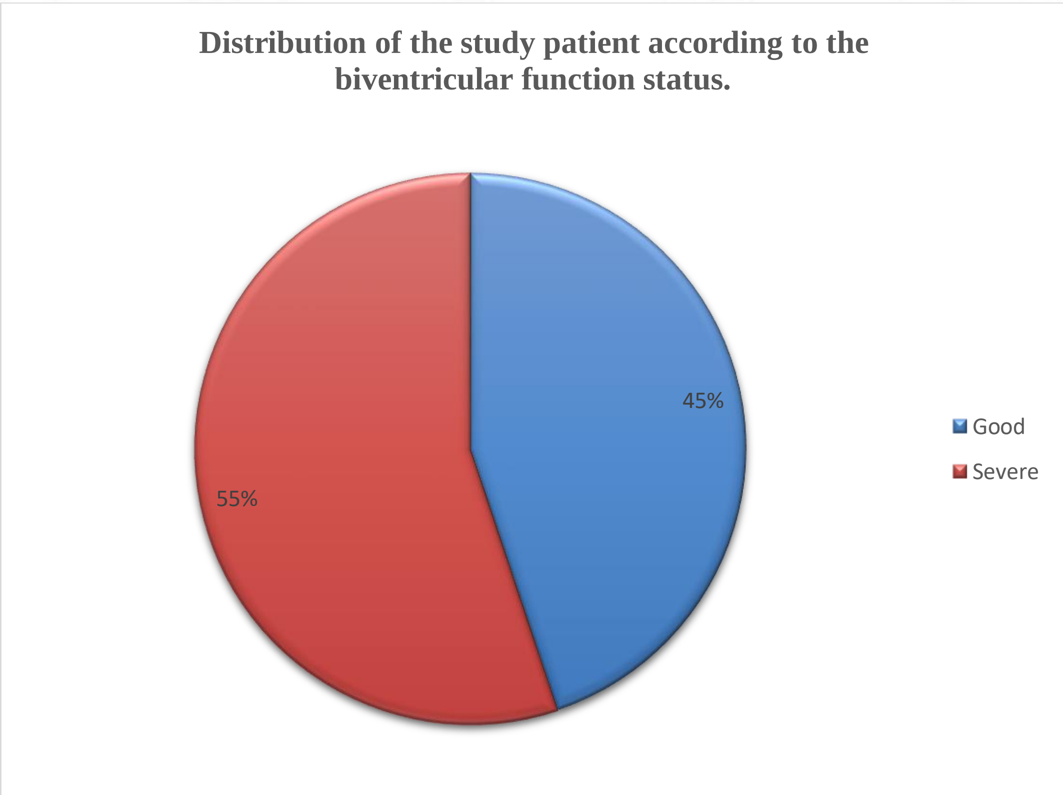


Figure 24. Distribution of the study patient according to the biventricular function status.

Table 25. Distribution of study patients according to residual ventricle septal rupture (VSR).

Sr. No.	Residual VSR	No. of patients n (%)
1	Yes	6 (26.1)
2	No	17 (73.9)
Total		23 (100)

Distribution of study patients according to residual ventricle septal rupture (VSR).

Among all 100% (n=23) patients, only 26.1% (n=6) had residual VSR, while the remaining 73.9% (n=17) did not have any residual VSR. **(Table 25) (Figure 25).**

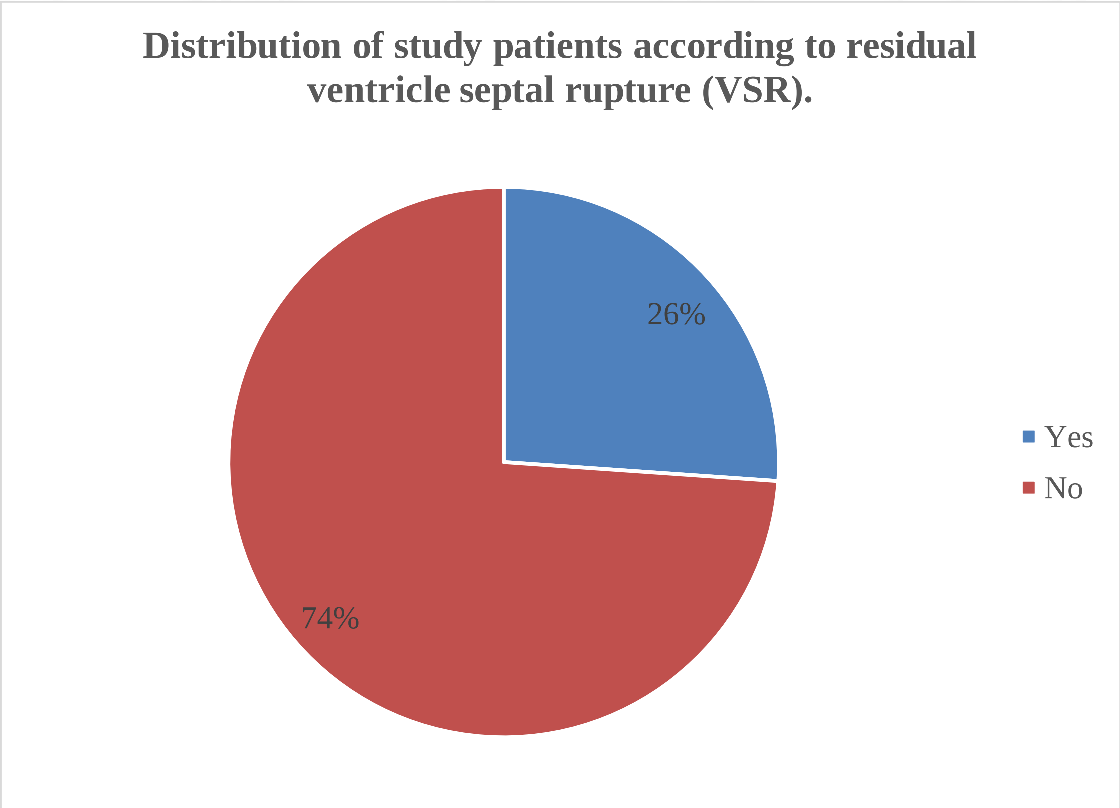


Figure 25. Distribution of study patients according to residual ventricle septal rupture (VSR).

Table 26. Distribution of study patients according to their post-operative complications.

Sr. No.	Post-operative Complications		No. of patients n(%)
1	Sepsis		7 (30.4)
2	Arrhythmia	AF	7 (30.4)
		VT	5 (21.7)
3	Acute kidney dysfunction		15 (65.2)
4	Adverse neurological outcome		2 (8.7)
5	Renal replacement therapy		7 (30.4)
6	Tracheostomy		6 (26.1)

Distribution of study patients according to size of ventricular septal rupture.

(Table 26) (Figure 26). Post-operative medical complications among all 23 patients were as follows: sepsis affected 30.4% (n=7) of patients, acute kidney dysfunction affected 65.2% (n=15), and adverse neurological outcomes were observed in 8.7% (n=2) of patients. Renal replacement was required by 30.4% (n=7) of the patients, and tracheostomy was indicated for 26.1% (n=6) of them. Among patients with arrhythmia, the type AF was observed in 30.4% (n=7), slightly higher than VT, which was observed in 21.7% (n=5) of the patients.

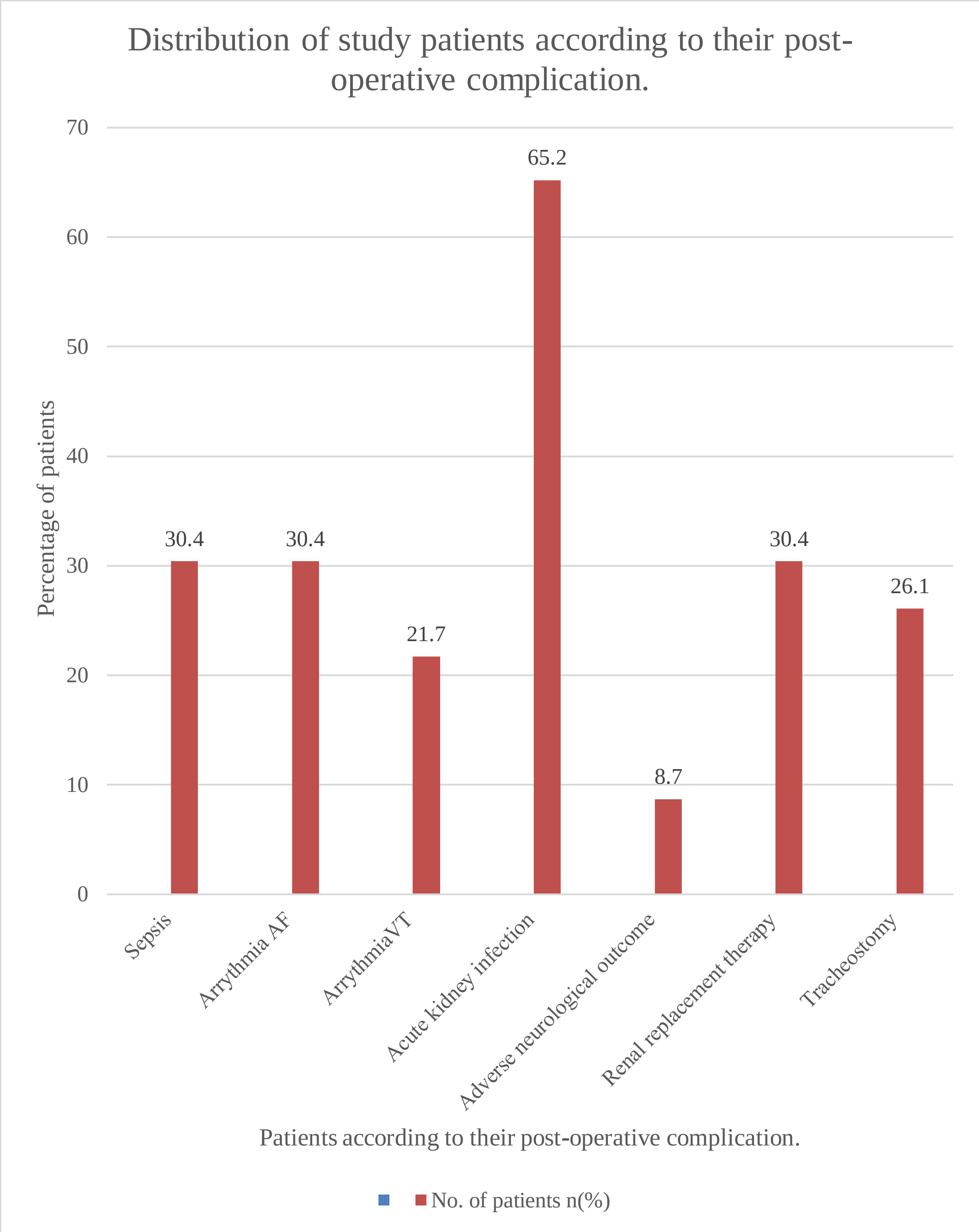


Figure 26. Distribution of study patients according to their post-operative complication.

Table 27. Distribution of study patients according to in-hospital mortality.

Sr. No.	In hospital mortality	No. of patients n (%)
1	**Yes**	10 (43.5)
2	**No**	13 (56.5)
Total		**23 (100)**

Distribution of study patients according to in-hospital mortality.

Among all 100% (n=23) study patients, 43.5% (n=10) were non-survivors at the time of discharge, while the remaining 56.5% (n=13) were survivors at discharge. **(Table 27) (Figure 27).**

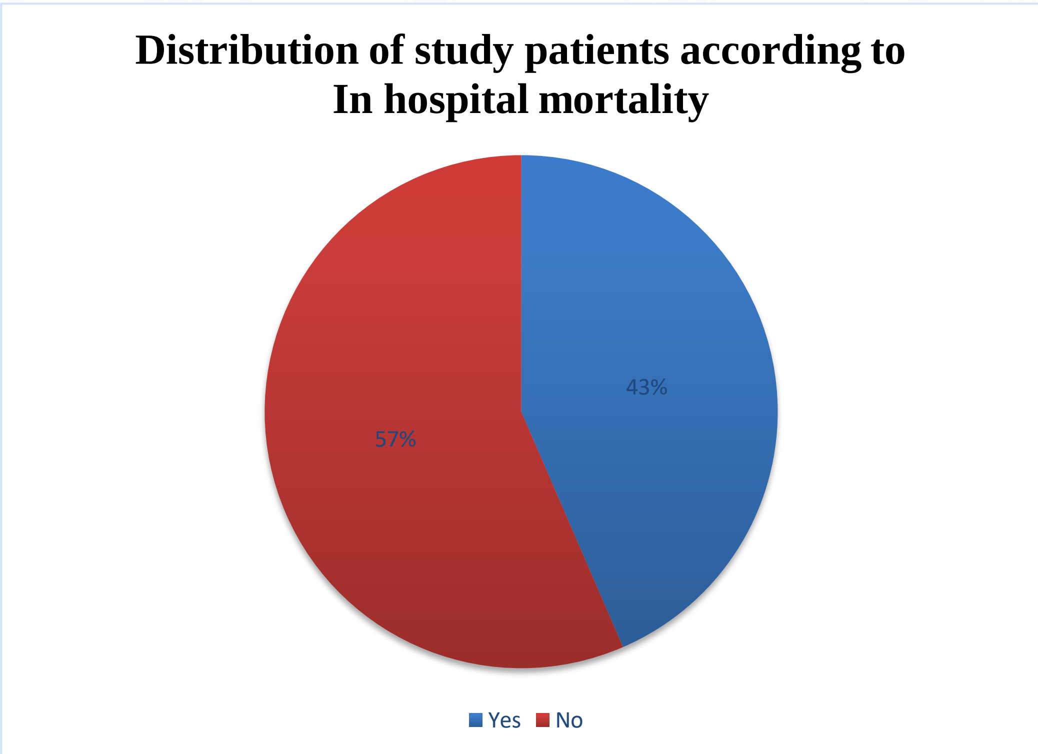


Figure 27. Distribution of study patients according to in-hospital mortality

Table 28. Distribution of study patients according to their follow-up examination of NYHA functional class at 3 months, 6 months and 1 year, respectively.

Sr. No.	NYHA Functional Class	No. of patients n (%)		
		At 3 Months	At 6 Months	At 1 Year
1	**Class I**	1 (10)	1 (10)	2 (20)
2	**Class II**	6 (60)	8 (80)	8 (80)
3	**Class III**	3 (30)	1 (10)	0
Total		**10 (100)**	**10 (100)**	**10 (100)**

Distribution of study patients according to their follow-up examination of NYHA functional class at 3 months, 6 months and 1 year, respectively. Of the 13 patients who survived and were discharged from the hospital, only ten came in for post-operative follow-up examinations. Unfortunately, three patients did not respond, and their post-operative data was unavailable.

Among the ten patients who responded, NYHA functional class I was observed in only one patient, accounting for 10% (n=1), at the 3rd and 6th-month follow-up examinations. By the 1-year follow-up, this number increased to 20% (n=2). NYHA class II functional class was observed in 60% (n=6) of the patients. At the 6th-month and 1-year follow-up, the number of patients in this category increased to 80% (n=8) for both follow-up periods. About 30% (n=3) of patients showed NYHA functional class III at the 3rd-month follow-up examination. This number decreased to 10% (n=1), with a single patient showing Class III FC at the 6th-month follow-up. Not a single patient exhibited Class III FC at the 1-year follow-up examination.

(Table 28) (Figure 28).

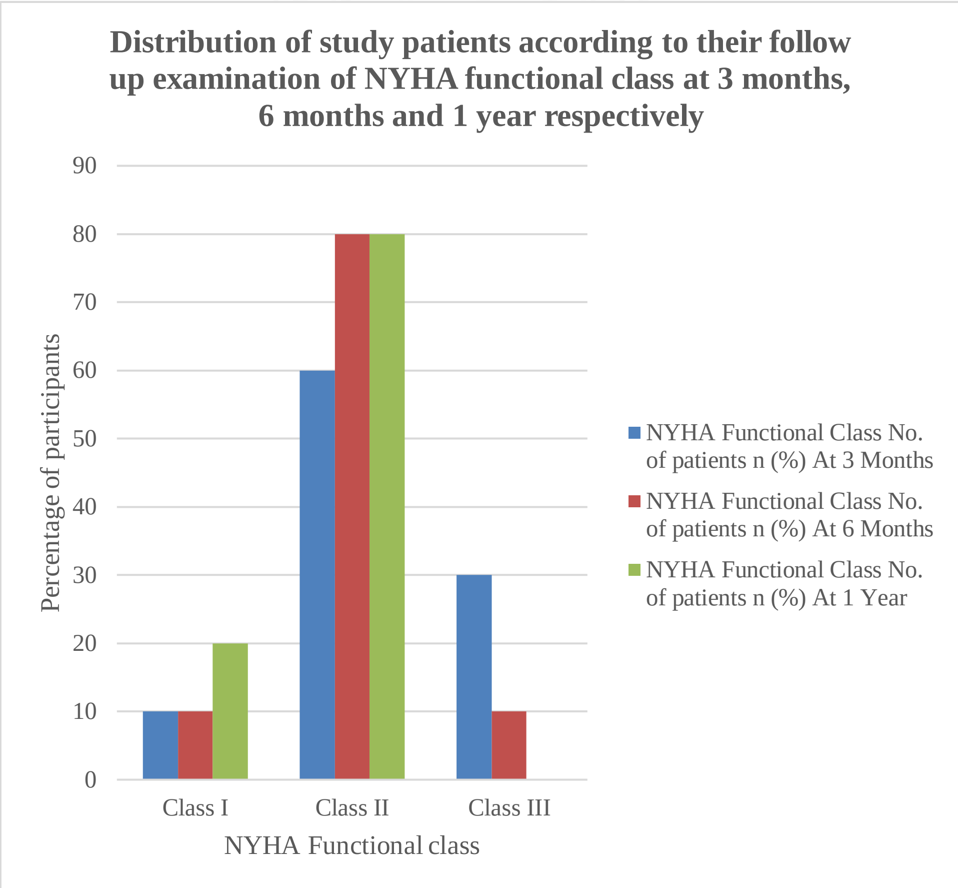


Figure 28. Distribution of study patients according to their follow-up examination of NYHA functional class at 3 months, 6 months and 1 year, respectively.

Table 29: Distribution of study patients according to myocardial infarction (MI) occurrence after VSR Repair.

Sr. No.	Post-op discharge MI	No. of patients n(%)
1	**No**	10 (100%)
2	**Missing Data**	3
Total		**10 (100)**
***3 Patient did not come to the hospital for review after discharge; hence, their data is missing and not included here.**		

Distribution of study patients according to myocardial infarction (MI) occurrence after VSR Repair.

Out of the 13 patients discharged from the hospital after VSR repair, 3 Patient did not come to the hospital for review after discharge; hence, their data is missing and not included here. In the remaining 10 patients none reported post-operative MI after hospital discharge, accounting for 100% (n=10). **(Table 29) (Graph 29).**

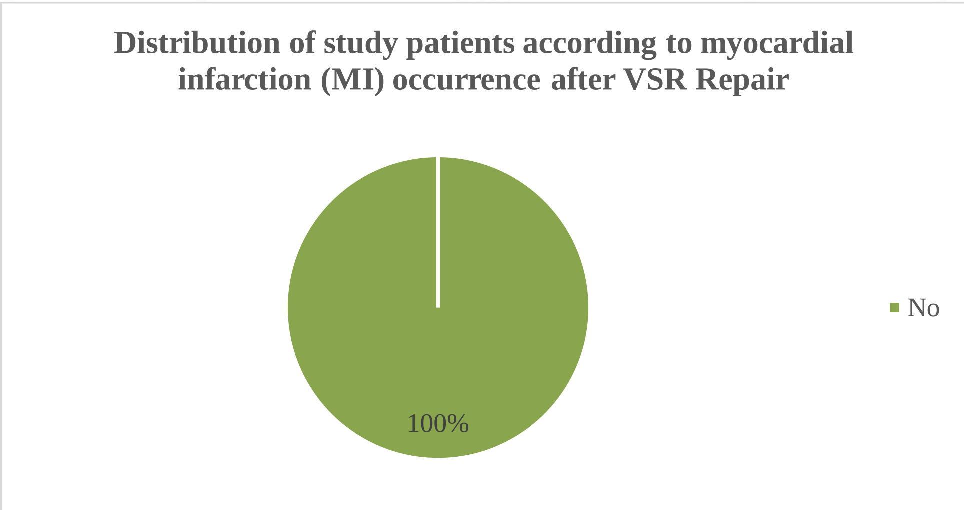


Figure 29. Distribution of study patients according to myocardial infarction (MI) occurrence after VSR Repair.

Table 30. Distribution of study patients according to their 1-year survival after VSR repair.

Sr. No.	1-year survival	No. of study patients n(%)
1	**Yes**	10 (100)
2	**Missing data**	
Total		**10 (100)**

***Note- All ten patients survived till 1 year. Three patients did not come for post-op follow-up; hence, data was missing for those 3.**

Distribution of study patients according to their 1-year survival after VSR repair. Of 13 patients, three did not attend the review following discharge. Hence, their follow-up data is considered missing in the current study. However, the remaining ten patients showed 100% survival at one year. **(Table 30)**

Table 31: Comparative distribution of various pre-operative factors attributing to the study patients' survival and non-survival status.

Sr No	Attributing pre-operative factors for survival and non-survival status		Survival at discharge N=13	Non-Survival N=10	Independent t-Test (p-value)
1	Hypertension		9 (69.2%)	8 (80%)	0.317
2	Diabetes Mellitus		9 (69.2%)	9 (90%)	0.180
3	Dyslipidemia		9 (69.2%)	6 (60%)	1.000
4	Peripheral artery disorder		1 (7.7%)	0	0.317
5	Chronic Kidney disease		5 (38.5%)	4 (40%)	1.000
6	Cardiac shock		2 (15.4%)	7 (70%)	0.025*
7	Inotropic Support		1 (7.7%)	6 (60%)	0.025*
8	Type of Myocardial Infarction	IWMI	6(46.2%)	3 (30%)	0.083
		AWMI	7(53.8%)	7 (70%)	0.180
9	Only LV dysfunction		5 (38.5%)	1 (10%)	0.500
10	Only RV dysfunction		2 (15.4%)	2 (20%)	0.414
11	Biventricular Dysfunction		2 (15.4%)	4 (40%)	0.480
12	No Ventricular Dysfunction		4 (30.8%)	3 (30%)	0.317
13	Multivessel disease		8 (61.5%)	7 (70%)	0.317
14	Previous PCI/CABG		4 (30.8%)	4 (40%)	0.680
15	MR	Mild	9 (69.2%)	8 (80%)	0.0564
		Moderate	4 (30.8%)	2 (20%)	0.564
16	LVEF		50.00 ±12.871%	48.50 ±14.136%	0.333
17	VSR Size		12.00 ± 6.014 mm	13.70 ± 5.438 mm	0.106
18	Time Interval between post-MI and VSR repair		53.46 ± 63.231 days	14.50 ± 16.250 days	0.025*

To identify common pre-operative factors that can be attributed to the patient's survival status after the post-MI VSR repair surgical procedure. A comparison between the two groups, survival and non-survival groups, was made. Patients who survived and had hospital discharge were included in a survival group, and they were 13. Patients in hospital mortality were enrolled in the non-survival group, and all were 10 in number.

The common factors reported in this study were given as follows-

1. **Hypertension:** Hypertension appears to be more common among patients who survived (69.2%) than those who did not (80%). However, the difference is not statistically significant (p = 0.317).

2. **Diabetes Mellitus:** Diabetes mellitus is more common among survivors (69.2%) than non-survivors (90%). The difference is not statistically significant (p = 0.180).

3. **Cardiac Shock and Inotropic Support:** Both cardiac shock and pre-operative requirement of inotropic support are significantly more common among non-survivors than survivors (p = 0.025* for both). These factors seem to be associated with a higher risk of mortality.

4. **Time Interval between post-MI and VSR repair:** The time interval between the myocardial infarction (MI) and VSR repair is significantly longer for survivors than non-survivors (p = 0.025*). This suggests that delayed surgical intervention is associated with better survival.

5. **Moderate MR (Mitral Regurgitation):** Patients with moderate MR have a lower survival rate (20%) compared to those with mild MR (30.8%). However, the difference is not statistically significant (p = 0.564).

Table 32: Comparative distribution of various intra-operative factors that attribute the study patients' survival and non-survival status.

Sr No	Affecting factor		Survival N=13	Non-Survival N=10	Independent t Test (p-value)
1	VSR location	Posterobasal	6 (46.2%)	4 (30%)	0.625
		Apical	6 (46.2%)	3 (30%)	0.317
		Mid muscular	1 (7.7%)	3 (40%)	0.317
2	Type of repair	Double patch	0	2 (20%)	0.157
		Patch closer	13 (100%)	6 (60%)	0.046*
		Patch closer with apical exclusion	0	2 (20%)	0.157
3	Concomitant procedure done		10 (76.9%)	7 (70%)	0.026*
4	CPB Time (Min)		143.31 ±50.506	131.50 ± 27.476	0.959
5	ACC Time (Min)		87.46 ±28.307	83.89± 25.615	0.953
6	IABP		0	5 (50%)	0.025*

For identification of common Intra-operative factors, those can attribute the patient's survival status after the surgical procedure of post-MI VSR repair. A comparison between the two groups, survival and non-survival groups, was made. Patients who survived and had hospital discharge were included in a survival group, and they were 13. Patients in hospital mortality were enrolled in the non-survival group, and all were 10 in number.

The common factors reported in this study were given as follows-

VSR Location: The location of the VSR appears to have some influence on survival.

Patients with apical and posterobasal VSR locations have similar survival rates, around

46.2%, while those with mid-muscular VSR have a lower survival rate (7.7%). However, the differences are not statistically significant.

Type of Repair: The type of repair performed is significantly associated with survival. Patients who received a "Patch closer" repair had a 100% survival rate, whereas those who received a "Double patch" repair had a 20% survival rate. This difference is statistically significant (p = 0.046*). The "Patch closer with apical exclusion" group had no survivors, but the sample size was small.

Concomitant Procedure: Patients who underwent concomitant procedures had a higher survival rate (76.9%) than those who did not (70%). The difference is statistically significant (p = 0.026*). This suggests that CABG during VSR repair is associated with improved survival.

CPB Time (Min) and ACC Time (Min): The time on cardiopulmonary bypass (CPB) and aortic cross-clamp (ACC) time did not show significant differences between the survival and non-survival groups. Both groups had similar mean times, and the p-values were not significant.

IABP (Intra-aortic Balloon Pump) weaning of CPB: The use of an IABP to wean the CPB is significantly more common in the non-survival group (50%) compared to the survival group (0%). This difference is statistically significant (p = 0.025*), suggesting that the patients who could be weaned of CPB without IABP support had better survival.

Table 33: Comparative distribution of various post-operative factors that attribute the study patients' survival and non-survival status.

Sr No	Affecting factor		Survival N=13	Non-Survival N=10	Independent t-Test (p-value)
1	Ventilation Time (days)		2.15 ± 4.220	7.80 ± 8.257	0.017*
2	Re-exploration		1 (7.7%)	0	1.000
3	ICU stay (days)		8.77 ± 9.98	13.20 ± 12.709	0.507
4	Hospital Stay (days)		21.08 ± 9.57	23.40 ± 17.658	0.313
5	Post-op LV function	Good	3 (23.1%)	2 (20%)	0.655
		Mild	3 (23.1%)	2 (20%)	0.655
		Moderate	7 (53.8%)	0	0.014*
		Severe	0	6 (60%)	0.031*
6	Post-op RV function	Good	7 (53.8%)	1 (10%)	0.025*
		Mild	3 (23.1%)	1(10%)	0.317
		Moderate	3 (23.1%)	2(10%)	1
		Severe	0	6 (60%)	0.031*
7	Biventricular	Good	3 (23.1%)	1 (10%)	0.317
		Severe	0	5 (50%)	0.025*
8	Residual VSR present		3 (23.1%)	3 (30%)	0.564
9	Sepsis		2 (15.4%)	5 (50%)	0.046*
10	Arrhythmia	AF	5 (38.5%)	2 (30%)	0.317
		VT	1 (7.7%)	4 (40%)	0.180
11	AKI		7 (53.8%)	8 (80%)	0.317
12	Adverse Neurological outcome		1 (7.7 %)	1 (10%)	1.00
13	Renal replacement Therapy		1 (7.7 %)	6 (60%)	0.014*
14	Tracheostomy		2 (15.4%)	4 (40%)	0.083

To identify common post-operative factors that can be attributed to the patient's survival status after the post-MI VSR repair surgical procedure. A comparison between the two groups, survival and non-survival groups, was made. Patients who survived and had hospital discharge were included in a survival group, and they were 13. Patients in hospital mortality were enrolled in the non-survival group, and all were 10 in number.

The common factors reported in this study were given as follows-

1. **Ventilation Time (days):** The duration of ventilation time is significantly shorter in survivors (2.15 days) compared to non-survivors (7.80 days). This difference is statistically significant (p = 0.017*), suggesting that a longer time on mechanical ventilation is associated with higher mortality.
2. **Postoperative LV Function:** The LV function post-surgery significantly differs between survivors and non-survivors. Patients with moderate or severe LV dysfunction have a significantly higher mortality risk (p = 0.014* and p = 0.031*, respectively). Conversely, patients with mild LV dysfunction did not showed any significant difference between survivors and non-survivors (p = 0.655).
3. **Postoperative RV Function:** Similar to LV function, postoperative good RV function is significantly associated with survival. Patients with severe RV dysfunction have a higher mortality risk (p = 0.031*).
4. **Post-operative severe biventricular dysfunction:** Post-op Higher mortality was observed in patients with severe biventricular dysfunction, and this result is significant with a p-value of 0.025*.

5. **Sepsis:** Sepsis is significantly more common among non-survivors (50%) than survivors (15.4%). This suggests that sepsis is associated with a higher risk of mortality (p = 0.046*).
6. **Renal Replacement Therapy:** The need for renal replacement therapy is significantly higher among non-survivors (60%) than survivors (7.7%). This difference is statistically significant (p = 0.014*), indicating that the requirement for renal replacement therapy is associated with higher mortality.
7. **Arrhythmia:** While there are differences in the occurrence of atrial fibrillation (AF) and ventricular tachycardia (VT), the p-values are not significant, indicating that these specific arrhythmias do not strongly differentiate between survivors and non-survivors.

5. DISCUSSION AND LIMITATIONS

VSR represents a grave complication arising from myocardial infarction and is characterized by a clinical presentation marked by deteriorating hemodynamic status and the development of cardiogenic shock (*Shimamoto et al., 2008; Damluji et al., 2021*). Surgical intervention is widely acknowledged as the foremost therapeutic approach for VSR. It is commonly held in medical consensus that posterior VSR confers a graver prognostic outlook compared to anterior VSR (*Durko et al., 2018*). This retrospective study assessed early outcomes and 1year survival rates after surgical intervention for post-infarction ventricular septal rupture. A cohort comprising 23 eligible individuals who met our specified criteria were enrolled in this study, all sourced from a single institutional setting.

Demographic details

Age distribution

All subjects in the study fell within the age range of 44 to 82 years, with an average age of 64.96 years and a standard deviation of ±8.315. In a study by Shi Tai and colleagues (Tai et al., 2020), a similar mean age of approximately 66±10 years was documented for their patient cohort.

Gender distribution

Regarding the gender distribution among the patients, 56.5% (n=13) were male, while 43.5% (n=10) were female. This distribution is consistent with the findings of Brancaccio and collaborators in 2021 (Brancaccio et al., 2021), where 61.1% (n=290) were male, and 38.9% (n=185) were female in their study.

- **PRE-OPERATIVE PARAMETERS**

- **Medical Complications**

The current study evaluated pre-operative parameters, including hypertension, diabetes mellitus, smoking, peripheral artery disease, chronic kidney disease, cardiogenic shock, and multi-vessel coronary artery disease in patients undergoing post-MI VSR repair.

- **Comorbidities comparison**

Among the patients with survival at discharge, 69.2% had hypertension, while 80% of non-survivors also had hypertension. The difference between the two groups was insignificant (p = 0.317). This suggests that hypertension did not impact this cohort's survival outcomes significantly. Both groups had a relatively high prevalence of diabetes mellitus, with 69.2% of survivors and 90% of non-survivors having this condition. However, the difference in diabetes prevalence between the two groups was not statistically significant (p = 0.180). This indicates that diabetes alone may not be a major determinant of survival in these patients. The presence of peripheral artery disease was rare in both groups, with only 7.7% of survivors and none of the non-survivors having this condition. The difference was not statistically significant (p = 0.317), likely due to the low prevalence in the dataset. Chronic kidney disease was present in 38.5% of survivors and 40% of non-survivors. The data shows no statistically significant difference between the two groups (p = 1.000). This suggests that chronic kidney disease did not independently impact the likelihood of survival in these patients. Our non-significant findings, as previously mentioned, align with the investigations of Takahashi H et al. and Huang et al., where no significant differences were reported between survival and non-survival patients regarding hypertension (p = 0.57), diabetes (p = 0.75), and smoking (p = 0.199) (Huang et al., 2015; Takahashi et al., 2015). The retrospective study (Wang L et al., 2021) among 127 patients also reported non-significant findings in terms of mortality and comorbidities such as hypertension (p = 0.807), diabetes (p = 0.453), and smoking (p = 0.260).

- **Cardiogenic shock**

Our findings indicate that patients who experienced cardiogenic shock preoperatively had poor survival following surgical repair. Notably, a noteworthy risk of mortality was associated with a history of pre-operative cardiogenic shock, in line with the study conducted by Moreyra and colleagues (p < 0.0001) (Moreyra et al., 2010).

We have observed that 70%(n=7) of patients who underwent surgical repair with preoperative cardiogenic shock did not survive (p = 0.025) at discharge. As per the study conducted by Wang L (Wang L et al., 2021), cardiogenic shock was demonstrated as an independent factor for mortality among 127 patients who were undergoing VSR (p < 0.0001). In addition, few studies have reported that cardiogenic shock, renal injury with dialysis, posterior infarct, and mitral regurgitation affect patient outcomes (Arnaoutakis G. et al., 2012; Serpytis P., 2015). In a study conducted by Bouchart and colleagues, they examined a group of 67 patients with VSR. Their findings indicated that the operative mortality rate was 25%. The primary cause of death among these patients was cardiac failure. The study revealed several factors influencing early mortality, including the preoperative hemodynamic condition, focusing on cardiogenic shock. One-year survival rates were reported at 74.6% with a margin of error of 5.3%, while the five-year survival rates were estimated at 66.2% with a margin of error of 6.2% (Bouchart F et al., 1998).

- **Requirement of Inotropic Support:**

Our investigation observed a significant association between the need for inotropic support before the repair of post-MI VSR and high in-hospital mortality. Specifically, the pre-operative requirement for inotropic support emerged as a substantial risk factor for in-hospital mortality ($p = 0.025$). In our study, we report the use of inotropic support in 7 patients, of which only one survived. This finding diverges from the non-significant relationship between pre-operative inotropic support and mortality reported in the studies conducted by Fukushima Satsuki et al. ($p = 0.28$) and Egidy Assenza et al. ($p = 0.9$) (Assenza et al., 2013; Fukushima et al., 2010).

- **Type of MI**

The analysis yielded several pertinent observations regarding these variables. Initially, the type of MI exhibited some potential influence on patient outcomes, as indicated by the survival rates of patients with inferior wall MI (IWMI) and anterior wall MI (AWMI). While the observed survival rate for IWMI was 46.2% and for AWMI was 53.8%, the statistical analysis did not attain conventional significance ($p = 0.083$). Nonetheless, this finding suggests that the type of MI may not contribute to the variability in VSR survival.

- **Mitral regurgitation**

Secondly, the analysis did not unveiled any notable contrast in survival based on the severity of mitral regurgitation (MR). Patients with mild MR displayed a higher survival rate (69.2%) than those with moderate MR (30.8%). Seventeen patients were reported with mild MR and six patients with moderate MR. While the p-value ($p = 0.0564$) approached the conventional significance threshold, it implies that the degree of MR severity may not significantly influence VSR survival. Nevertheless, this observation necessitates validation through a more comprehensive study involving a larger patient population.

- **Time interval between MI and VSR repair**

Furthermore, the time elapsed between MI and VSR repair emerged as a significant factor impacting survival. Patients subjected to delayed VSR repair (with an average interval of 53.46 days) exhibited superior survival outcomes compared to those who underwent prompt repair (with an average interval of 14.5 days). This difference was statistically significant ($p = 0.025$*), suggesting that delayed surgical intervention may enhance survival prospects. This finding carries crucial implications for clinical practice, emphasizing the potential benefits of a judiciously timed surgical approach. Papalexopoulou et al. (2013) conducted a comprehensive analysis encompassing six studies involving 3238 patients who underwent surgical interventions for post-infarct ventricular septal rupture (PIVSR). Their findings unveiled a substantial difference in in-hospital mortality rates based on the timing of surgical intervention. Patients who underwent early surgery between >3 days and within 4 weeks following myocardial infarction (MI) exhibited a significantly higher in-hospital mortality of 52.4%. In contrast, those who underwent delayed surgery, ranging from 1 week to 4 weeks after MI, experienced a substantially lower in-hospital mortality rate of 7.56%. The timing of the surgical procedure was intricately linked to the patient's hemodynamic status. An early surgical repair was deemed necessary for individuals with an infarct diameter exceeding 15 mm, accompanied by a substantial shunt and subsequent hemodynamic deterioration. A surgical procedure could be postponed for 3-4 weeks for patients with stable hemodynamic conditions. Nevertheless, prompt surgical intervention was strongly recommended, if there was clinical deterioration.

These findings underscore the critical importance of carefully evaluating the timing of surgery for patients with VSR, taking into account their specific clinical condition and hemodynamic status (Papalexopoulou N et al., 2013)

In contrast, other variables, including the presence of ventricular dysfunction, multivessel disease, previous history of percutaneous coronary intervention (PCI) or coronary artery bypass grafting (CABG), left ventricular ejection fraction (LVEF), and VSR size, did not manifest significant differences in survival outcomes among the examined groups.

- **INTRA-OPERATIVE PARAMETERS**

  - **Location of VSR**

The ventricular septal rupture was predominantly located at the posterior basal region in 43% (n=10) of cases, while 39.1% (n=9) of patients exhibited an apical location for the rupture. Only a minority, constituting 21.8% (n=5) of the patients, had the VSR at a milder muscular site. The study conducted by Malik J et al., 2021) found that four out of nine did not survive with basal rupture, whereas for those with an apical VSR, five out of eighteen patients did not survive. However, no significant difference was reported between basal or apical rupture.

The analysis considers the location of VSR, categorizing it into posterobasal, apical, and mid-muscular locations. While posterobasal and apical VSRs demonstrated relatively similar survival rates, mid-muscular VSRs displayed a notably lower survival rate. However, it's important to note that these differences did not reach statistical significance. This suggests that the location of the VSR might not be a significant independent factor in predicting patient survival.

Type of repair

Secondly, the type of repair was investigated, including double patch repair, patch closure, and patch closure with apical exclusion. Strikingly, patients who underwent patch closure showed a remarkable higher survival rate, while those who had double patch repair or patch closure with apical exclusion had considerably lower survival rates. The statistical analysis confirmed that the type of repair significantly influenced patient survival, emphasizing the importance of the surgical approach in VSR cases (p = 0.046*).

Concomitant procedures along with VSR repair

Moreover, the study assessed the impact of concomitant procedures during VSR surgery. Patients with concomitant procedures involving coronary revascularization displayed a higher survival rate than those without such additional interventions. This finding was statistically significant, indicating that coronary revascularization contributes to improved patient survival and should be carefully evaluated in the surgical decision-making process (p = 0.026*). A similar study finding was reported by a retrospective study (Lundblad et al., 2009) who reported that performing concomitant CABG reduces both early and late mortality in patients with VSR repair. In contrast, to our study findings Khan MY et al., 2018 reported no significant difference in mortality by performing concomitant CABG in patients with VSR

- **CBP and ACC time with VSR repair**

This study revealed no statistically significant difference between the two groups regarding CPB Time and ACC Time. Non-survivors had a mean CPB Time of 131.50 minutes, while survivors had a mean of 145.50 minutes, and their respective standard deviations were 27.476 and 50.95. Similarly, for ACC Time, non-survivors had a mean of 83.89 minutes with a standard deviation of 25.615, while survivors had a mean of 87.60 minutes with a standard deviation of 29.387. The p-values for CPB Time ($p = 0.386$) and ACC Time ($p = 0.859$) are above the conventional significance level of 0.05. However, prolonged surgery duration did not resulted in higher mortality among patients.

- **IABP utilization in VSR repair**

The utilization of an Intra-aortic Balloon Pump (IABP) for weaning off the Cardiopulmonary Bypass (CPB) is notably more prevalent among non-survivors, with 50% of them requiring IABP support. This finding implies a significant association between IABP use for weaning off CPB with a heightened risk of not surviving ($p = 0.025$).

This underscores the potential clinical importance of IABP usage as a prognostic indicator in this context. In cases of hemodynamically compromised patients, the consideration of employing a ventricular assist device, IABP, or Extracorporeal Membrane Oxygenation (ECMO) before surgery is an option worth exploring, as it can contribute to a higher likelihood of survival following post-myocardial infarction ventricular septal defect (post-MI-VSD) repair (Deja, M. A et al., 2000; Tsai, M. T et al., 2012). The IABP, in particular, is valuable in reducing the left ventricle's afterload and enhancing coronary perfusion. However, it's essential to note that the effectiveness of IABP support may vary among patients and may not provide the same benefits to all individuals (Deja MA et al., 2000). However, in our study, none of the patients were having IABP support before VSR repair. Our findings align with a retrospective study conducted among 2876 patients, where IABP was used pre-operatively and intra-operatively increased all-cause mortality ($p < 0.01$) (Arnaoutakis GJ et al., 2012).

❖ POST-OPERATIVE PARAMETERS

➢ Duration of ventilation

The duration of ventilation post-surgery was significantly different between survivors and non-survivors, with survivors requiring shorter ventilation time (2.15 ± 4.220 days) compared to non-survivors (7.80 ± 8.257 days). This observation is statistically significant ($p = 0.017$*) and suggests that a shorter duration of postoperative ventilation is associated with improved survival. In the present study, patients who required a shorter duration of ventilation, typically around 2 to 4 days, demonstrated a higher survival rate. In contrast, patients who needed ventilation for over a week faced increased mortality. A similar retrospective study demonstrated that ventilation support for a shorter period was reported with better clinical outcomes (Fu W et al., 2022; Arnaoutakis GJ et al., 2012).

➢ Post-operative ventricular function

Postoperative left ventricular (LV) function, the severity of dysfunction had a significant impact on survival. Non-survivors had a higher prevalence of severe LV dysfunction (60%) than survivors, where it was absent ($p = 0.031$). Any ventricular dysfunction which is less than severe was associated with a better survival This observation is statistically significant with a p-value of 0.014* for moderate LV dysfunction and p value of 0.025 for mild LV dysfunction. Similarly, for postoperative right ventricular (RV) function, severe dysfunction was significantly associated with non-survival, affecting 60% of non-survivors and only 1 (10%) survivor, with a p-value of 0.031*. This was similar to the study's Pang PY et al. finding, where RV and LV dysfunction has postulated with significant mortality at discharge and 1-year survival (Pang PY et al., 2013). Moore et al. also reported similar study findings where RV or LV dysfunction can lead to low cardiac output and high mortality (Moore et al., 1986). We have also noted that patients with post-ooperative severe biventricular dysfunction had higher mortality rate and a significant p value of 0.025.

- **Renal replacement therapy**

Regarding the need for renal replacement therapy, this was significantly more common in non-survivors, with 60% requiring it compared to only 7.7% of survivors. This difference is supported by a p-value of 0.014*, indicating the importance of this factor in predicting survival. A significant difference was also reported where RRT was needed in 12 patients, out of which only two survived, indicating a significant mortality ($p < 0.0005$) (Pang PY et al., 2013). In contrast to our study findings, a non-significant difference was seen among patients undergoing RRT ($p = 0.275$), where no mortality was reported (Fu W et al., 2022). Even though renal replacement therapy was identified as a significant indicator for mortality, post-operative acute kidney injury did not show any significant impact on the outcomes.

Sepsis in VSR

The occurrence of sepsis demonstrated a noteworthy difference, with sepsis affecting 50% of non-survivors and only 15.4% of survivors. This significant difference is supported by a p-value of 0.046*, indicating that sepsis is associated with a higher risk of non-survival. A similar study finding was reported where sepsis has proven to be a significant factor for mortality post-VSR repair (Arnaoutakis G. et al., 2012). However, the retrospective study (Malik J et al., 2021) revealed a non-significant difference in sepsis and infection compared to mortality. The study reports an incidence of infection in only two to three patients, of which 2 survived ($p = 1$).

➢ **Residual VSR after surgery**

Our study findings reports that residual VSR was not a significant factor for mortality with a higher p value of 0.564. A similar study finding was reported where residual VSR was not associated with higher mortality (Ronco D et al., 2021).

Other factors like re-exploration, ICU stay, hospital stay, arrhythmias, adverse neurological outcomes, and the need for tracheostomy did not show significant differences in survival between both the groups.

❖ **In-hospital mortality**

Of the 23 patients who underwent post-MI VSR repair, 10 patients did not survive (43.47%), 13 patients were survivors at discharge (56.53%). We have observed an in-hospital mortality of 43.47% However, despite prompt intervention, a higher in-hospital mortality rate was reported up to 65% (Cinq-Mars A et al., 2016).

➢ **NYHA Class at follow-up**

Of 13 patients discharged after VSR repair, three patients did not attend hospital follow-up, the remaining 10 (76.9%) patients were assessed at 3 month, 6-month, and 1-year. Based on the NYHA classification, we have reported good surgical outcomes after VSR repair in survivors.

➢ **MI after VSR repair and one-year survival**

Out of the 10 patients who were followed up after VSR repair, none of the patient's developed MI after VSR repair and all of them survived at one-year. One-year survival after discharge is 100%, as data of the three patients who did not attend is missing. The study conducted by Malik J et al., 2021 reported a one-year survival of 66.7% in patients with post-VSR repair.

Limitations of the study:

1. The study was conducted in a single institute, which may limit the generalizability of the results.

2. The study design was retrospective, which may introduce selection bias.

3. The sample size was relatively small (n=23), which may limit the study's statistical power.

4. The study did not include a control group, making it difficult to compare the outcomes.

5. The study only looked at early post-operative outcomes, and 1-year survival did not assess long-term outcomes.

.

6. SUMMARY AND CONCLUSION

In conclusion, VSR remains a severe complication stemming from myocardial infarction, often accompanied by deteriorating hemodynamic status and the onset of cardiogenic shock. Surgical intervention stands as the primary therapeutic approach for VSR. This retrospective study, including 23 eligible participants from a single institutional setting, aimed to evaluate the early outcomes and 1-year survival rates following surgical repair of post-infarction ventricular septal rupture. The study considered demographic factors, pre-operative parameters, intra-operative parameters, postoperative factors and follow-up data.

The assessment of pre-operative parameters indicated a significant association between pre-operative cardiogenic shock and increased mortality. The requirement for inotropic support before VSR repair was linked to a heightened risk of in-hospital mortality. These findings were consistent with previous research, suggesting that cardiogenic shock and the need for inotropic support were critical factors influencing outcomes. The study did not report any significance of pre-operative hypertension, diabetes, smoking on the outcomes of surgery. Pre-operative ventricular function, previous PCI/CABG and VSR size were not proven to be predictors of outcomes following surgical repair. Even though it is an ongoing debate our study shows that early surgical intervention was associated with increased mortality, while delayed surgery offered more favourable outcomes. However, this cannot be generalized as early surgeries might have been performed on unstable patients, the cohorts are not comparable.

Intra-operatively, the location of the VSR was observed, with no significant differences in outcomes. The study did not find statistically significant differences in Cardiopulmonary Bypass (CPB) and Aortic Cross-Clamp (ACC) times, though longer surgical durations were associated with lower mortality. Single patch closure of the VSR defect showed better outcomes compared with double patch and apical exclusion. Use of Intra-aortic Balloon Pump (IABP) for weaning from CPB was significantly more common among non-survivors. We have also noted that Coronary revascularisation at the time of VSR repair had better outcomes. However, there is an conflicting interest in terms of revascularization during VSR repair amongst many authors.

Post-operatively, the study emphasized the significant impact of ventricular function on survival. Patients with good left ventricular (LV) and right ventricular (RV) function exhibited higher survival rates. Biventricular function also significantly influenced survival, with good biventricular function associated with higher survival rates. Patients with LV/RV and biventricular dysfunction reported a higher mortality rate. Acute kidney injury, following VSR repair has not been proven as a significant factor in determining outcomes. However, the requirement of renal replacement therapy postoperatively is associated with bad outcomes. In our institute we reported an in-hospital mortality rate of 43.47% (n = 10). Thirteen patients (56.5%) (n = 13) were survivors at discharge, follow-up data of 3 patients is missing as they have not reported to hospital following discharge, rest of the 10 patients were followed at 3-months, 6-montsh, and at 1-year with no incidence of MI following discharge, a one-year survival was 100.0% which is commendable.

The study demonstrated the importance of considering pre-operative, intra-operative, and post-operative factors in assessing patient outcomes following VSR repair. These findings can inform clinical decision-making and emphasize the potential prognostic value of parameters such as cardiogenic shock at the time of surgery, preoperative use of inotropic support, the timing of surgery, coronary revascularisation along with VSR repair, The use of IABP to wean off CPB, prolonged ventilation, post-operative ventricular function, sepsis, and renal replacement therapy, in this patient population.

7. REFERENCES

1) Agnihotri AK, Madsen JC and Daggett Jr Willard M (2012) Chapter 28. Surgical Treatment of Complications of Acute Myocardial Infarction: Postinfarction Ventricular Septal Defect and Free Wall Rupture. In: Cohn LH (ed.) Cardiac Surgery in the Adult, 4e. New York, NY: The McGraw-Hill Companies. Available at: accesssurgery.mhmedical.com/content.aspx?aid=55918915 (accessed 21 October 2023).

2) Ariza-Solé A, Sánchez-Salado JC, Sbraga F, et al. (2020) The role of perioperative cardiorespiratory support in post infarction ventricular septal rupture-related cardiogenic shock. European Heart Journal. Acute Cardiovascular Care 9(2): 128–137.

3) Arnaoutakis, G. J., Zhao, Y., George, T. J., Sciortino, C. M., Mc Carthy, 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.

4) Assenza GE, McElhinney DB, Valente AM, et al. (2013) Transcatheter closure of post-myocardial infarction ventricular septal rupture. Circulation. Cardiovascular Interventions 6(1): 59–67.

5) Barker TA, Ramnarine IR, Woo EB, et al. (2003). Repair of post-infarct ventricular septal defect with or without coronary artery bypass grafting in the northwest of England: a 5-year multi-institutional experience. Eur J Cardiothorac Surg, 24(6), 940–946.

6) Becker AE and van Mantgem JP (1975) Cardiac tamponade. A study of 50 hearts. European Journal of Cardiology 3(4): 349–358.

7) Bishop HL, Gibson RS, Stamm RB, et al. (1981) Role of two-dimensional echocardiography in the evaluation of patients with ventricular septal rupture postmyocardial infarction. American Heart Journal 102(6 Pt 1): 965–971.

8) Boettler P, Claus P, Herbots L, et al. (2005) New aspects of the ventricular septum and its function: an echocardiographic study. Heart (British Cardiac Society) 91(10): 1343–1348.

9) Bouchart F, Bessou JP, Tabley A, Redonnet M, Mouton-Schleifer D, Haas-Hubscher C, et al. (1998). Urgent surgical repair of postinfarction ventricular septal rupture: early and late outcome. Journal of Cardiac Surgery, 13(2), 104–112.

10) Calvert PA, Cockburn J, Wynne D, et al. (2014) Percutaneous closure of postinfarction ventricular septal defect: in-hospital outcomes and long-term follow-up of UK experience. Circulation 129(23): 2395–2402.

11) Calvert, P. A., Cockburn, J., Wynne, D., et al. (2014). Percutaneous closure of postinfarction ventricular septal defect: in-hospital outcomes and long-term follow-up of UK experience. Circulation, 129, 2395-2402.

12) Cheng R, Hachamovitch R, Kittleson M, et al. (2014) Complications of extracorporeal membrane oxygenation for treatment of cardiogenic shock and cardiac arrest: a meta-analysis of 1,866 adult patients. The Annals of Thoracic Surgery 97(2): 610–616.

13) Cinq-Mars, A., Voisine, P., Dagenais, F., et al. (2016). Risk factors of mortality after surgical correction of ventricular septal defect following myocardial infarction: retrospective analysis and review of the literature. International Journal of Cardiology, 206, 27-36.

14) Cooley DA, Belmonte BA, Zeis LB, et al. (1957) Surgical repair of ruptured interventricular septum following acute myocardial infarction. Surg Journal. 41(6): 930–937.

15) Crenshaw BS, Granger CB, Birnbaum Y, et al. (2000) Risk factors, angiographic patterns, and outcomes in patients with ventricular septal defect complicating acute myocardial infarction. GUSTO-I (Global Utilization of Streptokinase and TPA for Occluded Coronary Arteries) Trial Investigators. Circulation 101(1): 27–32.

16) Daggett WM (1982) Surgical technique for early repair of posterior ventricular septal rupture. The Journal of Thoracic and Cardiovascular Surgery 84(2): 306–312.

17) Daggett WM, Burwell LR, Lawson DW, et al. (1970) Resection of acute ventricular aneurysm and ruptured interventricular septum after myocardial infarction. The New England Journal of Medicine 283(27): 1507–1508.

18) Damluji, A. A., van Diepen, S., Katz, J. N., et al. (2021). Mechanical Complications of Acute Myocardial Infarction: A Scientific Statement From the American Heart Association. Circulation, 144, e16-e35. doi:10.1161/CIR.0000000000000985.

19) David TE, Dale L and Sun Z (1995) Postinfarction ventricular septal rupture: repair by endocardial patch with infarct exclusion. The Journal of Thoracic and Cardiovascular Surgery 110(5): 1315–1322.

20) Deja, M. A., Szostek, J., Widenka, K., Szafron, B., Spyt, T. J., Hickey, M. S., et al. (2000). Post infarction ventricular septal defect - can we do better? European Journal of Cardio-Thoracic Surgery, 18(2), 194–201.

21) Dogra N, Puri GD, Thingnam SKS, et al. (2019) Early thrombolysis is associated with decreased operative mortality in postinfarction ventricular septal rupture. Indian Heart Journal 71(3): 224–228.

22) Durko, A. P., Budde, R. P. J., Geleijnse, M. L., et al. (2018). Recognition, assessment, and management of the mechanical complications of acute myocardial infarction. Heart, 104, 1216-1223. doi:10.1136/heartjnl-2017-311473.

23) Elbadawi A, Elgendy IY, Mahmoud K, et al. (2019) Temporal Trends and Outcomes of Mechanical Complications in Patients With Acute Myocardial Infarction. JACC.

Cardiovascular interventions 12(18): 1825–1836.

24) French JK, Hellkamp AS, Armstrong PW, et al. (2010) Mechanical complications after percutaneous coronary intervention in ST-elevation myocardial infarction (from APEX-AMI). The American Journal of Cardiology 105(1): 59–63.

25) Fu W, Wu L, Ma X, Zhao Y, Dong R, Mu J. Outcomes of surgical repair of anterior or posterior ventricular septal rupture after myocardial infarction. Cardiovasc Diagn Ther. 2022;12(2):177-187. doi:10.21037/cdt-21-577

26) Fukushima S, Tesar PJ, Jalali H, et al. (2010) Determinants of in-hospital and long-term surgical outcomes after repair of postinfarction ventricular septal rupture. The Journal of Thoracic and Cardiovascular Surgery 140(1): 59–65.

27) Gold HK, Leinbach RC, Sanders CA, et al. (1973) Intraaortic balloon pumping for ventricular septal defect or mitral regurgitation complicating acute myocardial infarction. Circulation 47(6): 1191–1196.

28) Gregoric ID, Kar B, Mesar T, et al. (2014) Perioperative use of TandemHeart percutaneous ventricular assist device in surgical repair of postinfarction ventricular septal defect. ASAIO journal (American Society for Artificial Internal Organs: 1992) 60(5): 529–532.

29) Heitmiller R, Jacobs ML and Daggett WM (1986) Surgical management of postinfarction ventricular septal rupture. The Annals of Thoracic Surgery 41(6): 683–691.

30) Hernández-Meneses S, Meza-López LR, Olvera-Lozano JT, et al. (2020) Tratamiento quirúrgico de la ruptura septal ventricular postinfarto. Cirujano general 42(4). Asociación Mexicana de Cirugía General AC: 281–287.

31) Huang S-M, Huang S-C, Wang C-H, et al. (2015) Risk factors and outcome analysis after surgical management of ventricular septal rupture complicating acute myocardial infarction: a retrospective analysis. Journal of Cardiothoracic Surgery 10: 66.

32) Hutchins GM (1979) Rupture of the interventricular septum complicating myocardial infarction: Pathological analysis of 10 patients with clinically diagnosed perforations. American Heart Journal 97(2): 165–173.

33) Ibanez B, James S, Agewall S, et al. (2018) 2017 ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation: The Task Force for the management of acute myocardial infarction in patients presenting with ST-segment elevation of the European Society of Cardiology (ESC). European Heart Journal 39(2): 119–177.

34) Jeppsson A, Liden H, Johnsson P, et al. (2005) Surgical repair of post-infarction ventricular septal defects: a national experience. European Journal of Cardio-Thoracic Surgery.27(2): 216–221.

35) Khan MY, Waqar T, Qaisrani PG, et al. (2018) Surgical Repair of post-infarction ventricular septal rupture: Determinants of operative mortality and survival outcome analysis. Pakistan Journal of Medical Sciences 34(1): 20–26.

36) La Torre MW, Centofanti P, Attisani M, et al. (2011) Posterior ventricular septal defect in presence of cardiogenic shock: early implantation of the impella recover LP 5.0 as a bridge to surgery. Texas Heart Institute Journal 38(1): 42–49.

37) Lee PH, Hong JA, Sun BJ, et al. (2018) Impact of Significant Mitral Regurgitation on Assessing the Severity of Aortic Stenosis. Journal of the American Society of Echocardiography. 31(1). 1: 26–33.

38) Levine GN, Bates ER, Blankenship JC, et al. (2016) 2015 ACC/AHA/SCAI Focused Update on Primary Percutaneous Coronary Intervention for Patients With ST-Elevation Myocardial Infarction: An Update of the 2011 ACCF/AHA/SCAI Guideline for Percutaneous Coronary Intervention and the 2013 ACCF/AHA Guideline for the Management of ST-Elevation Myocardial Infarction. Circulation 133(11). American Heart Association: 1135–1147.

39) Li H, Zhang S, Yu M, et al. (2019) Profile and Outcomes of Surgical Treatment for Ventricular Septal Rupture in Patients With Shock. The Annals of Thoracic Surgery 108(4): 1127–1132.

40) Loisance DY, Lordez JM, Deleuze PH, et al. (1991) Acute postinfarction septal rupture: long-term results. The Annals of Thoracic Surgery 52(3): 474–478.

41) Lundblad R and Abdelnoor M (2014) Surgery of postinfarction ventricular septal rupture: The effect of David infarct exclusion versus Daggett direct septal closure on early and late outcomes. The Journal of Thoracic and Cardiovascular Surgery 148(6). Elsevier: 2736–2742.

42) Ma D, Zhang Z, Zhang S, et al (2022) Treatment Strategies for Ventricular Septal Rupture After Myocardial Infarction: A Single-Center Experience. Front Cardiovasc Med.9:843625.

43) Malhotra A, Patel K, Sharma P, et al. (2017) Techniques, Timing & Prognosis of Post Infarct Ventricular Septal Repair: a Re-look at Old Dogmas. Brazilian Journal of Cardiovascular Surgery 32(3): 147–155.

44) Malik, J., Younus, F., Malik, A., Farooq, M. U., Kamal, A., Shoaib, M., Naeem, H., Rana, G., Rana, A. S., Usman, M., & Khalil, S. (2021). One-year outcome and survival analysis of deferred ventricular septal repair in cardiogenic shock supported with mechanical circulatory support. PloS one, 16(8), e0256377. https://doi.org/10.1371/journal.pone.0256377 (Retraction published PLoS One. 2022 Aug 11;17(8):e0273165)

45) Maltais S, Ibrahim R, Basmadjian A-J, et al. (2009) Postinfarction ventricular septal defects: towards a new treatment algorithm? The Annals of Thoracic Surgery 87(3): 687–692.

46) Mann JM and Roberts WC (1987) Cardiac morphologic observations after operative closure of acquired ventricular septal defect during acute myocardial infarction: analysis of 16 necropsy patients. The American Journal of Cardiology 60(13): 981–

987.

47) Maree AO, Jneid H and Palacios IF (2006) Percutaneous closure of a post-infarction ventricular septal defect that recurred after surgical repair. European Heart Journal 27(13): 1626.

48) Massetti M, Babatasi G, Le Page O, et al. (2000) Postinfarction ventricular septal rupture: early repair through the right atrial approach. The Journal of Thoracic and Cardiovascular Surgery 119(4 Pt 1): 784–789.

49) Matteucci M, Ronco D, Corazzari C, et al. (2021) Surgical Repair of Postinfarction Ventricular Septal Rupture: Systematic Review and Meta-Analysis. The Annals of Thoracic Surgery 112(1): 326–337.

50) Menon V, Webb JG, Hillis LD, et al. (2000) Outcome and profile of ventricular septal rupture with cardiogenic shock after myocardial infarction: a report from the SHOCK Trial Registry. Journal of the American College of Cardiology 36(3, Supplement 1): 1110–1116.

51) Miller SW, Dinsmore RE, Greene RE, et al. (1978) Coronary, ventricular, and pulmonary abnormalities associated with rupture of the interventricular septum complicating myocardial infarction. AJR. American Journal of roentgenology 131(4): 571–577.

52) Moore CA, Nygaard TW, Kaiser DL, et al. (1986) Postinfarction ventricular septal rupture: the importance of location of infarction and right ventricular function in determining survival. Circulation 74(1): 45–55.

53) Moreyra AE, Huang MS, Wilson AC, et al. (2010) Trends in incidence and mortality rates of ventricular septal ruptu re during acute myocardial infarction. The American Journal of Cardiology 106(8): 1095–1100.

54) Morillon-Lutun S, Maucort-Boulch D, Mewton N, et al. (2013) Therapeutic management changes and mortality rates over 30 years in ventricular septal rupture complicating acute myocardial infarction. The American Journal of Cardiology 112(9): 1273–1278.

55) Mubarik A and Iqbal AM (2023) Ventricular Septal Rupture. In: StatPearls. Treasure Island (FL): StatPearls Publishing. Available at: http://www.ncbi.nlm.nih.gov/books/NBK534857/ (accessed 20 October 2023).

56) Naik RB, Srivastava CP, Mathur A, et al. (2022) Early outcomes of surgical repair of post-infarct ventricular septal rupture: A single centre experience. IHJ Cardiovascular Case Reports (CVCR) 6(4): 172–174.

57) Nakatani D, Sato H, Kinjo K, et al. (2003) Effect of successful late reperfusion by primary coronary angioplasty on mechanical complications of acute myocardial infarction. The American Journal of Cardiology 92(7): 785–788.

58) Okamoto, Y., Yamamoto, K., Asami, F., Kimura, M., Mizumoto, M., Okubo, Y., & Yoshii, S. (2016). Early and midterm outcomes of triple patch technique for

postinfarction ventricular septal defects. The Journal of thoracic and cardiovascular surgery, 151(6), 1711–1716. https://doi.org/10.1016/j.jtcvs.2016.01.056

59) Pang PYK, Sin YK, Lim CH, et al. (2013) Outcome and survival analysis of surgical repair of post-infarction ventricular septal rupture. Journal of Cardiothoracic Surgery. 8:44 1749-8090-8-44.

60) Papalexopoulou, N., Young, C. P., & Attia, R. Q. (2013). What is the best timing of surgery in patients with post-infarct ventricular septal rupture? Interact Cardiovasc Thorac Surg, 16(2), 193–196.

61) Patel K, Malhotra A, Shah K, et al. (2019) Early and late mortality and morbidity after post-MI ventricular septal rupture repair: predictors, strategies, and results. Indian Journal of Thoracic and Cardiovascular Surgery 35(3): 437–444.

62) Payne WS, Hunt JC and Kirklin JW (1963) Surgical repair of ventricular septal defect due to myocardial infarction. Report of a case. JAMA 183: 603–605.

63) Pfeffer MA and Braunwald E (1990) Ventricular remodeling after myocardial infarction. Experimental observations and clinical implications. Circulation 81(4): 1161–1172.

64) Ronco D, Matteucci M, Kowalewski M, et al. (2021) Surgical Treatment of Postinfarction Ventricular Septal Rupture. JAMA Network Open 4(10): e2128309–e2128309.

65) Sakaguchi G, Miyata H, Motomura N, Ueki C, Fukuchi E, Yamamoto H, et al. (2019) Surgical Repair of Post-Infarction Ventricular Septal Defect - Findings From a Japanese National Database. Circ J, 83(11), 2229–2235. doi: 10.1253/circj.CJ-19-0593.

66) Samuels LE, Entwistle JC, Holmes EC, et al. (2003) Mechanical support of the unrepaired postinfarction ventricular septal defect with the Abiomed BVS 5000 ventricular assist device. The Journal of Thoracic and Cardiovascular Surgery 126(6): 2100–2101.

67) Sánchez Vega JD, Alonso Salinas GL, Viéitez Florez JM, et al. (2022) Optimal surgical timing after post-infarction ventricular septal rupture. Cardiology Journal 29(5): 773–781.

68) Seguin JR, Frapier JM, Colson P, et al. (1992) Fibrin sealant for early repair of acquired ventricular septal defect. The Journal of Thoracic and Cardiovascular Surgery 104(3): 748–751.

69) Selzer A, Gerbode F and Kerth WJ (1969) Clinical, hemodynamic, and surgical considerations of rupture of the ventricular septum after myocardial infarction. American Heart Journal 78(5): 598–607.

70) Serpytis, P., Karvelyte, N., Serpytis, R., et al. (2015). Post-infarction ventricular septal defect: risk factors and early outcomes. Hellenic Journal of Cardiology, 56(1), 66-71.

71) Shimamoto, T., Marui, A., Saji, Y., et al. (2008). Infarction-exclusion technique with the on-pump beating heart approach for ventricular septal perforation. Interact Cardiovasc Thorac Surg, 7, 1207-1209. doi:10.1510/icvts.2008.188359.

72) Shumacker HB (1972) Suggestions concerning operative management of postinfarction septal defects. The Journal of Thoracic and Cardiovascular Surgery 64(3): 452–459.

73) Skehan JD, Carey C, Norrell MS, et al. (1989) Patterns of coronary artery disease in post-infarction ventricular septal rupture. British Heart Journal 62(4): 268–272.

74) Smyllie JH, Sutherland GR, Geuskens R, et al. (1990) Doppler color flow mapping in the diagnosis of ventricular septal rupture and acute mitral regurgitation after myocardial infarction. Journal of the American College of Cardiology 15(6): 1449–1455.

75) Swithinbank JM (1959) Perforation of the interventricular septum in myocardial infarction. British Heart Journal 21(4): 562–566.

76) Szkutnik M, Bialkowski J, Kusa J, et al. (2003) Postinfarction ventricular septal defect closure with Amplatzer occluders. European Journal of Cardio-Thoracic Surgery: Official Journal of the European Association for Cardio-Thoracic Surgery 23(3): 323–327.

77) Tai S, Tang J, Yu B, et al. (2020) Association between Cardiovascular Burden and Requirement of Intensive Care among Patients with Mild COVID-19. Cardiovascular Therapeutics 2020: 9059562.

78) Takahashi H, Arif R, Almashhoor A, et al. (2015) Long-term results after surgical treatment of postinfarction ventricular septal rupture. European Journal of Cardio-Thoracic Surgery 47(4): 720–724.

79) Thiele H, Kaulfersch C, Daehnert I, et al. (2009) Immediate primary transcatheter closure of postinfarction ventricular septal defects. European Heart Journal 30(1): 81–88.

80) Topaz O and Taylor AL (1992) Interventricular septal rupture complicating acute myocardial infarction: from pathophysiologic features to the role of invasive and noninvasive diagnostic modalities in current management. The American Journal of Medicine 93(6): 683–688.

81) Tsai, M. T., Wu, H. Y., Chan, S. H., & Luo, C. Y. (2012). Extracorporeal membrane oxygenation as a bridge to definite surgery in recurrent postinfarction ventricular septal defect. ASAIO Journal, 58(1), 88–89.

82) Vajifdar B, Vora A, Narula D, et al. (1996) Percutaneous balloon occlusion of post-infarction ventricular septal defect. Indian Heart Journal 48(4): 407–408.

83) Wang, L., Xiao, L. L., Liu, C., Zhang, Y. Z., Zhao, X. Y., Li, L., Wang, X. F., & 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, 679148. https://doi.org/10.3389/fcvm.2021.679148

84) Wilson WM and Horlick EM (2016) Management of post-myocardial infarction ventricular septal rupture. EuroIntervention: Journal of EuroPCR in Collaboration with the Working Group on Interventional Cardiology of the European Society of Cardiology 12 Suppl X: X18–X23.

85) Yam N, Au TW-K and Cheng L-C (2013) Post-infarction ventricular septal defect: surgical outcomes in the last decade. Asian Cardiovascular & Thoracic Annals 21(5): 539–545.

ANNESURES
CV of PRINCIPAL INVESTIGATOR

CV of investigator

<table>
<tr><td>Hema Krishna</td><td>Sai</td><td colspan="2">Kadiyala</td></tr>
<tr><td colspan="2">Date of Birth (dd/mm/yy) 12/10/1990</td><td colspan="2">Sex: Male</td></tr>
<tr><td colspan="4">Study Site Affiliation (e.g. Principal Investigator, Co-Investigator, Coordinator)

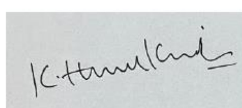
Co-investigator : principal investigator</td></tr>
<tr><td colspan="2">Professional
Mailing Address
(Include
Institution name)</td><td colspan="2">Study Site Address (Include
Institution name)</td></tr>
<tr><td colspan="2">Dr.Hema Krishna Sai
Kadiyala

Dept of
CVTS
SCTIMST

Trivandrum</td><td colspan="2">Dr.Hema Krishna Sai Kadiyala

Senior Resident ,

Dept of CVTS
SCTIMST
Trivandrum</td></tr>
<tr><td colspan="2">Telephone (Office): -</td><td colspan="2">Mobile Number: 8977455455</td></tr>
<tr><td colspan="2">Telephone (Residence):-</td><td colspan="2">Email: drsidkadiyala@gmail.com</td></tr>
<tr><td colspan="4">Travancore Cochin Medical Council Registration Number - 93737</td></tr>
<tr><td colspan="4">Academic Qualifications (Most recent qualification first)</td></tr>
<tr><td colspan="2">Degree/Certificate</td><td>Year</td><td>Institution, Country</td></tr>
<tr><td colspan="2">MS General. Surgery</td><td>2019</td><td>Sree Ramachandra institute of Higher Education and Research, Chennai</td></tr>
</table>

M B B S	2015	Dr.P.S.I of Medical Sciences & R.F, Chinaavutapalli, Andhra Pradesh.
Details of professional registration : (MCI/State Registration/Bar Council/DCI/etc includingRegistration Number and Year of Registration: APMC- 95152		
Current and previous positions (most recent position first)		
Month and Year	Title	Institution/Company, Country
Jan 2021	SENIOR RESIDENT	SCTIMST.
Brief summary of relevant research experience: -		
Current project/s at hand: -		
 Dr.Hema Krishna Sai .K		09-04-2023 TRIVANDRUM

CV of Guide

Format for CV of the Co-Investigator

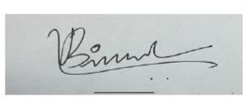
Last Name : R	First Name: Bineesh	Middle Name: K
Date of Birth (dd/mm/yy) :08/11/1976		Sex: Male
Study Site Affiliation (e.g. Principal Investigator, Co-Investigator, Coordinator) Co-investigator		

Professional Mailing Address (Include Institution name)	Study Site Address (Include Institution name)
Dr.Bineesh.K.R Additional Professor Dept of CVTS SCTIMST	Dr.Bineesh.K.R Additional Professor Dept of CVTS SCTIMST
Telephone (Office): 0471-2443152	Mobile Number: 9895417919
Telephone (Residence):0471-2443152	Email :bineesh@sctimst.ac.in

Academic Qualifications (Most recent qualification first)

Degree/Certificate	Year	Institution, Country
PDF- Adult Cardiac Surgery	2013	SCTIMST
MCh –CVTS	2012	SCTIMST
MS –General Surgery	2006	Medical College, Trivandrum
MBBS	1999	Medical College, Trivandrum

[redacted]

Details of professional registration : TCMC -29081		
Current and previous positions (most recent position first)		
Month and Year	Title	Institution/Company, Country
2021- till date	Additional Professor ,CVTS	SCTIMST
2018-2021	Associate Professor ,CVTS	SCTIMST
2014-2015	Associate Professor (ADHOC),CVTS	SCTIMST
01/01/2013 – 31/12/2013	PDF-Adult Cardiac Surgery ,CVTS	SCTIMST
Brief summary of relevant research experience: Participated in many studies as Principal investigator and co-investigator.		
Current project/s at hand: 1) Development of Biopresthetic Herat valve. 2) TTK Chitra – Titanium Heart Valve Pilot Study (Model TC2). 3) A Suction retractor device for aortic valve replacement in adult cardiac surgery.		
 Signature:Bineesh.K.R		09-04-2023 Trivandrum

APPENDIX – APPROVALS & PERMISSIONS

Institutional Ethics Committee (IEC) Approval Letter

श्री चित्रा तिरुनाल आयुर्विज्ञान और प्रौद्योगिकी संस्थान, त्रिवेन्द्रम
तिरुवनन्तपुरम - ६९५०११, केरल, इंडिया

SREE CHITRA TIRUNAL INSTITUTE FOR MEDICAL SCIENCES AND TECHNOLOGY, TRIVANDRUM
Thiruvananthapuram - 695 011, Kerala, India
(An Institute of National Importance under Govt. of India)

Grams : Chitramet, Phone : +91-471-2443152, Fax : +91-471-2550728 / 2446433, E-mail : sct@sctimst.ac.in, Website : www.sctimst.ac.in

Institutional Ethics Committee
CDSCO Registration No: ECR/189/Inst/KL/2013/RR-21
DHR Registration No:EC/NEW/INST/2022/2775

SCT/IEC/2126/ER/SEPTEMBER-2023 20.10.2023

Dr. Hema Krishna Sai Kadiyala
Senior Resident
Department of CVTS
SCTIMST, Thiruvananthapuram

Dear Dr. Hema Krishna Sai Kadiyal,

The Institutional Ethics Committee reviewed your project titled "**A RETROSPECTIVE STUDY THE OUTCOMES OF SURGICAL REPAIR OF POST-INFARCTION VENTRICULAR SEPTAL RUPTURE** (IEC/2126)" on 20th October, 2023.

Principal Investigator	Dr. Hema Krishna Sai Kadiyala , Senior Resident, Department of CVTS, SCTIMST
Co-Principal Investigator(s)	Dr. Bineesh K. R. Additional Professor, Department of CVTS, SCTIMST
Duration of the study	6 months from the commencement of study

The following documents were reviewed:

Original submission
1. Covering letter addressed to the Chairperson, IEC SCTIMST
2. IEC Application Form
3. Declaration form
4. Project Proposal
5. Proforma
6. Telephone script in Malayalam
7. CV of PI and Co-PI
8. Checklist Form

Revised submission
1. Covering letter addressed to the Chairperson, IEC SCTIMST
2. IEC Application Form
3. Declaration form
4. Project Proposal
5. Proforma
6. CV of PI and Co-PI
7. Checklist Form

IEC Decision

The IEC approved the conduct of the retrospective study titled "**A RETROSPECTIVE STUDY THE OUTCOMES OF SURGICAL REPAIR OF POST- INFARCTION VENTRICULAR SEPTAL RUPTURE**" in the present form.

IEC approval is issued on the basis of retrieval of secondary data from medical/laboratory records without involving human participants directly. The approval of this proposal is valid for a period of 6 months only. If, in any case the study is to be converted to a prospective nature, the approval given will be void and a fresh application has to be filed stating the reason for changing the design of the study.

Remarks:

The Institutional Ethics Committee expects to be provided a copy of the final report/publication.

There was no member of the study team who participated in voting / decision making process. The ethics committee is organized and operated according to the requirements of Good Clinical Practice and the requirements of the Indian Council of Medical Research (ICMR).

Sincerely,

Dr. G. Srinivas
Member Secretary, IEC

MEMBER SECRETARY
INSTITUTIONAL ETHICS COMMITTEE (IEC)
SCTIMST, THIRUVANANTHAPURAM

SREE CHITRA TIRUNAL INSTITUTE FOR MEDICAL SCIENCES & TECHNOLOGY
INSTITUTIONAL ETHICS COMMITTEE (IEC) SCTIMST THIRUVANANTHAPURAM

APPENDIX – SUPPLEMENTARY TABLES

MASTER CHART (DATA ENTRY)

S:NO	H:NO	AGE	GENDER	PREOPERATIVE PARAMETERS											
				Hypertension	Diabetes Mellitus	Dyslipedemia	Smoking	POAD	CKD	Cardiogenic Shock	Inootropic Support	TYPE of MI	LV Dysfunction	RV Dysfunction	Multi Vessel Disease
1	327508	66	M	Yes		Yes	No	No	Yes	No	No	IWMI	No	Yes	Yes
2	330924	77	M	No	Yes	No	No	No	Yes	Yes	Yes	IWMI	Yes	Yes	No
3	341794	69	F	Yes	Yes	No	No	No	No	Yes	Yes	AWMI	Yes	Yes	No
4	351263	82	M	Yes	No	No	No	No	Yes	No	No	AWMI	Yes	No	No
5	356026	44	F	No	No	No	No	No	No	Yes	Yes	AWMI	No	No	No
6	369435	65	M	Yes	Yes	Yes	No	No	Yes	Yes	No	IWMI	No	Yes	Yes
7	377526	74	M	No	No	No	No	No	No	No	No	AWMI	No	No	No
8	379097	67	F	No	Yes	Yes	No	No	No	No	No	AWMI	Yes	Yes	No
9	381081	65	F	No	Yes	No	No	No	No	No	No	AWMI	No	No	Yes
10	387171	63	M	Yes	Yes	Yes	Yes	No	No	No	No	IWMI	Yes	No	Yes
11	392590	61	M	Yes	Yes	Yes	Yes	Yes	Yes	No	No	IWMI	No	Yes	Yes
12	399642	65	F	Yes	Yes	Yes	No	No	No	Yes	Yes	AWMI	Yes	Yes	Yes
13	403106	59	F	No	Yes	No	No	No	Yes	Yes	No	AWMI	Yes	No	No
14	406100	58	F	Yes	No	No	No	No	No	Yes	Yes	AMWI	No	No	Yes
15	259440	81	F	Yes	Yes	Yes	No	No	No	Yes	Yes	AWMI	Yes	Yes	Yes
16	448549	58	M	Yes	Yes	Yes	No	No	Yes	No	No	IWMI	No	No	Yes
17	452413	63	M	Yes	No	Yes	No	No	No	No	No	AWMI	Yes	No	Yes
18	458036	68	M	Yes	Yes	Yes	Yes	No	No	No	No	IWMI	No	No	Yes
19	464229	66	F	Yes	Yes	Yes	No	No	Yes	Yes	Yes	AWMI	No	No	No
20	470479	56	M	Yes	Yes	Yes	Yes	No	Yes	No	No	IWMI	Yes	Yes	Yes
21	472774	63	F	Yes	Yes	Yes	No	No	No	No	No	AWMI	Yes	No	Yes
22	479359	59	M	Yes	Yes	Yes	No	No	No	No	No	AWMI	Yes	No	Yes
23	504858	65	M	Yes	Yes	Yes	No	No	No	No	No	IWMI	No	Yes	Yes

APPENDIX – SUPPLEMENTARY TABLES

MASTER CHART (DATA ENTRY)

							INRAOP
PreviousPCI / CABG	Mitral Regurgitati on	LVEF	VSR Size	Time Interval B/W MI & VSR Repair	Pulmonary / Systamtic Bllood Flow	VSR Location	Type of Repair
No	Mild	53	8 mm	10		Posterobasal	Double patch
No	Mild	50	19 mm	5		Posterobasal	Patch closure
No	Mild	40	18 mm	6		Apical	Patch closure + apical exlusion
No	Mild	41	7 mm	17		Apical	Patch closure
No	Mild	70	18 mm	8		Apical	Patch closure
No	Moderate	75	10 mm	4		Mid muscular	Patch closure
No	Mild	57	8 mm	11		Apical	Patch closure
No	Moderate	47	10 mm	32		Apical	Patch closure
No	Mild	41	15 mm	12	3.62	Mid muscular	Patch closure
No	Moderate	25	Multiple	180		Posterobasal	Patch closure
No	Mild	60	10 mm	6		Posterobasal	Patch closure
PCI-LAD	Mild	40	10 mm	12		Posterobasal	Patch closure
PCI-LAD	Mild	50	10 mm	39		Mid muscular	Patch closure
No	Mild	60	25 mm	8		Apical	Patch closure + apical exlusion
CABG X 2G	Moderate	25	10 mm	5		Posterobasal	Patch closure
NO	Mild	55	20 mm	120		Posterobasal	Patch closure
No	Moderate	47	10 mm	23		Apical	Patch closure
PCI-LAD & RCA	Mild	70	10 mm	180		Posterobasal	Patch closure
PCI-LAD	Mild	60	10 mm	26		Apical	Patch closure
PCI-RCA	Mild	35	13 mm	37		Posterobasal	Patch closure
PCI-LAD	Mild	41	12 mm	57		Mid muscular	Double patch
PCI-LAD	Mild	42	20 mm	15		Apical	Patch closure
No	Moderate	51	20 mm	27		Posterobasal	Patch closure

APPENDIX – SUPPLEMENTARY TABLES

MASTER CHART (DATA ENTRY)

ERATIVE PARAMETERS											POST OPERATIVE PARAMETEI	
Concomitanat Procedure	CPB Time	ACC Time	IABP	ECMO	Ventilation Time	RE-Exploration	ICU Stay	Hospital Stay	Post-op LEFT Ventricular Function	Post-Operative Right Ventricular Function	Residual VSR	Sespsis
Nil	101	77	No	No	19	No	34	44	Severe	Severe	No	Yes
Nil	142	111	Yes	No	2	No	2	2	Severe	Severe	No	No
Nil	113	64	Yes	No	6	No	19	21	Severe	Severe	Yes	Yes
CABG X 1G	127	94	No	No		No	4	23	Good	Good	No	No
CABG X 1G	106	74	No	No	2	No	4	13	Moderate	Good	No	No
CABG X 3G	132	69	No	No	6	No	22	48	Good	Moderate	Yes	Yes
CABG X 1G	95	68	No	No		No	4	20	Moderate	Moderate	Yes	No
CABG X 1G + LV aneurysmoraphy	92	46	No	No		No	4	30	Moderate	Good	No	No
CABG X 2G	125	72	No	No	4	No	4	13	Mild	Severe	No	No
CABG X 3G + MV repair	223	126	No	No	2	No	6	11	Moderate	Mild	No	No
CABG X 4G + LV aneurysmoraphy	204	122	No	No	1	No	5	17	Mild	Moderate	No	No
CABG X 2G	137	68	Yes	No		No		9	Severe	Severe	No	No
Nil	78	37	No	No		No	5	10	Moderate	Good	Yes	No
CABG X 1G	146	113	No	No	18	No	27	32	Good	Good	No	Yes
CABG X 1G	187		Yes	No		No	1	3	Severe	Severe	Yes	No
CABG X 3G	148	94	No	No		No	5	9	Moderate	Mild	No	No
CABG X 4G	212	118	No	No		No	4	26	Good	Good	No	No
Nil	109	71	No	No	12	No	28	32	Good	Good	No	Yes
CABG X 1G	88	55	Yes	No	2	No	2	17	Severe	Moderate	No	No
Nil	175	101	No	No	11	Yes	34	40	Moderate	Mild	No	Yes
CABG X 1G	144	126	No	No	21	No	21	45	Mild	Mild	No	Yes
CABG X 1G	110	76	No	No		No	4	16	Mild	Good	No	No
CABG X 3G	184	110	No	No		No	7	27	Mild	Moderate	Yes	No

APPENDIX – SUPPLEMENTARY TABLES

MASTER CHART (DATA ENTRY)

RS						FOLLOW UP				
Arrhythmia	AKI	Adverse Neurological Outcomes	Renal Replacment Therpay	Tracheostomy	In-Hospital Mortality	NYHA @ 3 Months	NYHA @ 6 Months	NYHA @ 1 Year	MI Post Discharge	1 Year Survival
AF	Yes	No	Yes	Yes	Yes					
VT	Yes	No	Yes	No	Yes					
VT	No	Yes	No	No	Yes					
No	Yes	No	No	No	No	FC 1	FC 1	FC 1	No	Yes
No	No	No	No	No	No	FC 2	FC 2	FC 2	No	Yes
No	Yes	No	No	Yes	Yes					
No	No	No	No	No	No					
AF	No	No	No	No	No	FC 2	FC 2	FC 2	No	Yes
VT	Yes	No	Yes	No	Yes					
AF	Yes	No	No	No	No	FC 3	FC 2	FC 2	No	Yes
VT	Yes	No	No	No	No					
No	No	No	No	No	Yes					
No	Yes	No	No	No	No	FC 3	FC 2	FC 1	No	Yes
No	Yes	No	Yes	Yes	Yes					
No	Yes	No	No	No	Yes					
No	Yes	No	No	No	No	FC 2	FC 2	FC 2	No	Yes
AF	No	Yes	No	No	No	FC 2	FC 2	FC 2	No	Yes
AF	Yes	No	No	Yes	No					
VT	Yes	No	Yes	No	Yes					
AF	Yes	No	Yes	Yes	No	FC 3	FC 3	FC 2	No	Yes
AF	Yes	No	Yes	Yes	Yes					
No	No	No	No	No	No	FC 2	FC 2	FC 2	No	Yes
No	No	No	No	No	No	FC 2	FC 2	FC 2	No	Yes

APPEDIX - Plagiarism Check Report

PATIENT INFORMATION PROFORMA

Case No:

- Age
- Gender

PRE-OPERATIVE PARAMETERS

- Hypertension
- Diabetes
- Dyslipidemia
- POAD
- Chronic kidney disease
- Chronic obstructive lung disease
- Cardiogenic shock
- Inotropic support
- Type of MI
- Left ventricular dysfunction
- Right ventricular dysfunction
- Multi vessel disease
- Previous PCI / CABG
- Mitral regurgitation
- Left ventricular ejection fraction
- VSR size
- Time interval between MI and VSR repair

INTRA-OPERATIVE PARAMETERS

- VSR Location
- Type of repair
- Concomitant Procedure
- CPB Time
- Aortic cross clamp time
- IABP
- ECMO

PATIENT INFORMATION PROFORMA

POST OPERATIVE PARAMETERS

- Ventilation time
- Reexploration
- ICU stay
- Hospital stay
- Post-op Ventricular function
- Residual VSR
- Sepsis
- Arrhythmia
- AKI
- Adverse Neurological outcome
- Renal replacement therapy
- Tracheostomy
- In-hospital Mortality

FOLLOW UP

- NYHA class at 3 months, 6 months & 1 year
- MI Post discharge
- 1 year survival

SREE CHITRA TIRUNAL INSTITUTE FOR MEDICAL SCIENCES ANDTECHNOLOGY, TRIVANDRUM

An Institution of National Importance established by an Act of the Indian Parliament (Act No.52 of 1980)

Dept. of Science and Technology,
Govt. of India