

Outcome Analysis After Surgical Management of Ventricular Septal Defect Complicating Acute Myocardial Infarction

Kallol Dasbaksi¹, Suranjan Haldar², Mohammad Zahid Hossain³, Plaban Mukherjee⁴

ABSTRACT

Introduction: Surgical approaches to closure of post myocardial infarction ventricular septal defect (PIVSD) are associated with high morbidity and mortality. Timing of intervention for its closure remains controversial. Several studies advocate early operative intervention, after diagnosis of PIVSD but these are associated with high mortality. However, the strategy of delayed closure around 14 to 20 days or higher has been advocated in certain subsets of patients who can be stabilized from cardiogenic shock (CS) with pharmacological means with or without temporary mechanical circulatory support (tMCS). This helps to allow tissue fibrosis around PIVSD which increases the chance of operative success. Results of 5 such patients in whom surgery was moderately delayed, and 2 in whom early operation were performed are reported in this paper.

Material and methods: Between May 2012 to April 2016, 7 consecutive patients of PIVSD had operative closure under cardio pulmonary bypass in our hospital. 5 patients had diuretic and inotrope responsive CS and had delayed closure of PIVSD within 12 to 20 days, while 2 with severe CS, who were supported preoperatively with tMCS like intra aortic balloon pump (IABP), had early closure within 72 hours. Patient data of these seven subjects were retrospectively collected, and the current status of the survivors was ascertained by out patient follow up.

Results: 4 of 5 patients from delayed surgery group with small PIVSD survived while 1 patient had early mortality due to severe right ventricular dysfunction post operatively resulting in LCOS. 1 patient out of the 2 early surgery group with a large PIVSD survived while the other patient with a small PIVSD and an extensive MI had early post operative mortality. 1 had from late surgery group had delayed mortality after 3 years. 4 patients are living at present.

Conclusion: We advocate delayed elective repair of PIVSD, in patients with CS who responded to aggressive conservative management maintaining hemodynamic stability, to allow inflammatory state to subside. In those patients with severe CS, additional rescue therapy with temporary mechanical circulatory support is needed to prevent further deterioration of systemic perfusion. If the severe CS is due to high left to right shunt rather than infarct size, prognosis after repair of PIVSD is better than in patients with CS due to extensive myocardial damage.

Keywords: Post Infarct Ventricular Septal Defect, Cardiogenic Shock

myocardial contractility due to acute myocardial infarction (AMI) and in turn further reduces stroke volume. An impeded cardiac output with increased end diastolic pressure increase wall tension and consequently increases tissue hypoperfusion and worsens myocardial ischaemia, resulting in a vicious cycle resulting in cardiogenic shock (CS).¹ Early surgical intervention for all such cases, to prevent further loss of functioning cardiac myocytes has been advocated in the past in an attempt to break this vicious cycle. As per American College of Cardiology/American Heart Association (ACC/AHA) 2013 guidelines of ST-elevation myocardial infarction, emergency surgical repair of PIVSD was advocated every patients of PIVSD. It was advocated even in stable patients to avoid sudden expansion of the PIVSD size resulting in sudden hemodynamic collapse in previously stable patients.² Such early surgery were associated with mixed results of success, with mortality ranging from 20% to 87% in reported series.³ It has also been reported that mortality was significantly reduced to 18.4% if repair was delayed for a week.^{2,3} Temporary mechanical circulatory support (tMCS) as Intra aortic balloon pump (IABP) was applied frequently in PIVSD with cardiogenic shock (CS) with an aim to stabilize the patient before operation. On the other hand, meta-analysis failed to demonstrate a reduced mortality in unselected patients with CS treated with tMCS used on a routine basis.⁴ High mortality in these studies may have been due to intervention in subsets of patients who developed irreversible multi organ dysfunction syndrome (MODS) apart from the hazards of open heart surgery.⁵ But in some with CS, response with tMCS was positive with improved hemodynamic parameters and end-organ function which allowed settling down of the acute inflammatory state for an effective delayed surgical closure.^{6,7} Therefore, the triage of patients with CS for proper tMCS may play a crucial role. Devices with limited improvement rates, like IABP may be chosen more liberally in the early stage of CS when cardiac

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INTRODUCTION

Post infarction ventricular septal defect (PIVSD) associated with acute left to right shunt impairs already depressed

index (CI) reduces below 2.2 Liter per minute per square meter (L/min/m²) and can maintain upto around 1.2 L/min/m². Advanced tMCS devices like extra corporeal membrane oxygenation (ECMO) with higher flow rates may be required for more severe refractory CS with CI below 1.2L /min/m².⁴ ACC /AHA guidelines for STEMI(2013) advocated class IIa (level of evidence: B) recommendation for use of IABP in patients with AMI complicated by CS as a temporary stabilizing measure in those not able to quickly achieve hemodynamic stability with pharmacologic treatment. However, alternative mechanical assist devices like ECMO as class IIb (level of evidence C) has been advocated in those patient group with refractory or severe CS.²

In this paper we attempted to analyze our experience in 7 patients over a period of 6 years. Delayed closure of PIVSD was done in 5 patients after management of CS and early closure in 2 patients who presented with refractory CS, with one early death, within 6 days, in each group.

MATERIALS AND METHODS:

From May 2012 to April 2016, 7 consecutive patients, who were referred from Department of Cardiology, underwent surgical repair of PIVSD were enrolled for the study. Patient data of these seven subjects were retrospectively collected, and the current status of the survivors was ascertained by out patient follow up. Table-1 shows demographic profiles, transthoracic echocardiography and coronary angiography (CAG) profiles, preoperative, operative, and postoperative variables were collected retrospectively. The parameters also included co-morbidities, history of thrombolysis, requirements of intra-aortic balloon pump (IABP), operative parameters such as aortic cross clamp time (CC), type of VSD closure, concomitant CABG, cardiopulmonary bypass (CPB) time, post operative length of stay in intensive therapy unit (ITU). Preoperative, and peri operative patient characteristics according to results of operation, that is in those survived or those died, are summarized in Table No 2. Analysis of Perioperative variables in 7 patients with PIVSD according to timing, in Late surgery and Early surgery groups, size and locations of VSDs, and results of surgery are included in Table no. 3. Table No. 4 shows analysis of post operative outcome variables, mortality, survival and recurrence in 7 patients with PIVSD according to Late surgery and Early surgery groups.

Low cardiac output syndromes were stabilized by pharmacotherapy in 5 patients and additional IABP was employed in 2 patients of refractory shock who failed to respond adequately to pharmacotherapy. All patients had coronary angiography performed. Surgical closure of PIVSD was moderately delayed in 5 patients (between 12th and 20th day, mean 13.14±7.72 days) and managed on pharmacotherapy to optimize hemodynamic conditions and had single mortality. Early surgical closure was performed in the two patients with IABP within 72 hours of referral to our department. This Early Surgery group had 1 in hospital mortality.

Operation

Under cardiopulmonary bypass distal anastomosis of vein

grafts were done in beating heart and after that aorta was cross clamped and St Thomas 2 cardioplegia, both antegrade and retrograde, were taken in. PIVSD, (Figure-1) closure was done next. Through the left ventriculotomy, the VSD was closed with effectively with Gluteraldehyde preserved bovine pericardium with pledgeted interrupted # 3-0 polypropylene mattress sutures with pledgets placed on the right ventricular side. An additional larger patch of same material was placed around the 1st one was using continuous 3-0 polypropylene suture on the LV side covering the previous patch (vide Figure No. 2). The patch was next exteriorized and included within the Ventriculotomy closure with interrupted and next layer of continuous # 1 polypropylene mattress sutures buttressed with bovine pericardium. In one case of anterior PIVSD (case # 6, vide Table No 1), the left ventricular aneurysm in the anterolateral wall was reconstructed with bovine pericardium. The details of the technique are shown in Fig. 2. The proximal aortic vein graft anastomoses were done after release of cross clamp. IABP used in cases preoperatively were continued in the post operative period. One patient from the Delayed Group (SI No. 4 vide Table No. 1) also needed IABP support while coming off bypass but succumbed after 3 days.

All the datas were processed in Microsoft XL tabulation done, and statistical averages, standard deviations and relevant proportion were calculated. No further statistical tests could be done due to small num,bers of these rare cases.

RESULTS

The intra- and postoperative outcome details are presented in Table No 2 and 3. Average Aortic cross(ACC) clamp time was 129 ± 10.10 minutes and Cardiopulmonary bypass time (CPB) was 178 ± 37.41 minutes. Both the values were longer in late surgery (130±13.21,180±40.04 respectively) than in early surgery (128.50±3.53 and 169.5±48.79 respectively) groups. ACC and CPB times were 127±12.05, 157±6.29 minutes in those patients who survived and 135.40±4.50, 232 ±39.59 minutes respectively in those who died. Concomitant CABG was performed in 6 (85.7%) patients. Table No. 4 shows mortality and survival among both the groups. Median postoperative stay in the intensive care unit was 6.5±2.22 days (upto 9 days, leaving aside the patients who died). Early mortality was 2 out of 7 (28.5%). Both the patients expired by 3 rd day. Mortality was 1 in each delayed group 1/5 (20%) and early surgical group 1/2 (50%). Mortality was similar for anterior and postero inferior PIVSD vide table No.2. There was a trend towards lower mortality in patients undergoing concomitant CABG. All the 5 patients in the delayed surgery group, had higher preoperative left ventricular ejection fractions (LVEF 38.8±5.45%) compared to the early surgery group (LVEF 29.5±5.55) (vide Table No. 2). But while the LVEF was 24% in the deceased patient in early surgery group had an extensive MI with refractory shock with a smaller VSD, the LVEF was 35% in the late surgery group and the cause of death was due to right ventricular failure leading to LCOS. For those 5 patients discharged alive after surgery, survival at 1 and 3 years was 100% in both the groups, and

Serial No E L or E	Age & sex	Interval in days between AMI and PIVSD and & pre operative IABP	LV EF And Diabetes “+”	CAG and ves- sels involved LAD, Cx, Diagonal, RCA, PDA	History of MI	Location and size of VSD in mm & history of Thrombolysis (T)	Interval Between PIVSD and Operation (days)	Operation Closure of VSD “C”± CABG, ACC, CPB times	Survival or death “S” or “D”, ITU stay in days
1L	F 62	4	42%,+ +	Mid LAD 100%, Cx 80%, Diagonal, RCA, PDA	-	A, 10	20	C+CABG ACC 112 CPB 160	S, ITU9 days
2L	M 61	3	42% +	Mid LAD 100%, PD 70%	-	A, 9(T)	18	C+CABG ACC 121 CPB 157	S, ITU 8 days
3E	M 63	2 IABP	24%, +	Prox LAD 100%, Cx 90%, PDA 70%	+	A, 8 (T)	3	C+CABG ACC 131 CPB 204	D, ITU 3 days
4L	M 58	5	35%,+ +	Prox RCA 100%, L 70%	+	P,8	12	C+CABG ACC 140 CPB 260	D, ITU4 days
5L	M 50	4	40%, -	Distal RCA 100%, LAD 80%	-	P,9(T)	19	C+CABG ACC 135 CPB 160	S, ITU 7 days
6L	M 56	5	42%, +	Mid LAD 100% only,	+	A,12, Ant lateral aneurysm*	20	C ACC 143 CPB 163	S, ITU 8 days, (died after 3 years)
7E	M 68	4 IABP	28%, +	Distal RCA 100%, Cx 90%, Mid LAD 70%	+	A, 15	3	C+CABG ACC 126 CPB 145	S, ITU 7 days

SI No= Serial number, E=Early surgery group within 3 days, L=Late surgery Group, M=male, F=female, LVEF=left ventricular ejection fraction, ACC=Aortic cross clamp time in minutes, CPB= cardiopulmonary bypass time in minutes, CAG= coronary angiography, LAD=left anterior descending artery, PD=posterior descending coronary artery, RCA= right coronary artery, A=anterior location of post infarct ventricular septal defect (PIVSD) in midpart of interventricular septum (IVS), P= Postero inferior location of PIVSD in IVS, S= survived operation, D= death after operation within 7 days. * SI No. 6, additionally, anterolateral wall aneurysm was present. I= IABP= Intra aortic balloon pump, MI = Myocardial infarction, ITU= Intensive care unit, T= History of thrombolysis,

Table-1: Demographic and clinical, angiographic, timing and nature of surgery, surgical pathology, and results of surgery in 7 patients.

Variables	Total (n=7)	Survived(S) (n=5)	Died(D) (n=2)
Age, years	59.71±5.73	59.40±6.05	60.50±2.5
Male gender	6/7(85.7%)	4/7(57.14)	2/7(28.57%)
Female	1/7(14.28%)	1/5(20%)	
Hypertension	5/7(71.4%)	3 (42.85%)	2(28.57%)
DM (%)	6/7(85.7%)	4(57.14%)	2 28.57%)
Thrombolysis done	3/7(42.8%)	2 /7(28.57%)	1/7(14.28%)
P/H of MI	5/7(71.4%)	3 (42.85%)	2 (28.57%)
Echocardiography			
LVEF	36.14± 6.98	38.8±5.45	29.5±5.55
CAG			
Proximal LAD	1/7(14.2%)	0	1/7(14.2%)
Osteal RCA	1/7(14.2%)	0	1/7(14.7%)
Mid LAD	4/7(57.1%)	4/7(57.1)	0
Mid RCA	1/7(14.2%)	1/7(14.2%)	0
Preoperative delay:			
More than 10 days	5/7(11.4%)	1/7(14.2%)	1/7(14.2%)
Average ±mean(days)	13.14±7.72	16±6.54	7.50± 6.36
Less than 3 days	2/7(28.40%)	1/7(14.2%)	1/7(14.2%)
Preoperative IABP	2/7(28.5%)	1/7(14.2%)	1/7(14.2%)
Size of PIVSD			
Less than 10mm	5/7(71.4%)	4/7(57.1%)	1/7(14.2%)
More than 10mm	2/7(28.5%)	1/7(14.2%)	1/7(14.2%)
Operation type			
PIVSD closure with CABG	6/7(85.71%)	4/7(57.14%)	2/7(28.5%)
Only PIVSD closure	1/7(14.28%)	1/7(14.28%)	0
Aortic CC time(minutes)	129± 10.10	127± 12.05	135.50± 4.50
CPB Time (minutes)	178.42± 40.41	157.00± 7.03	232± 39.59
Intensive care unit stay (days)	6.5±2.22	7.80±0.74	3.5±0.70
Postoperative LCOS leading to death	2 (28.5%)	1/5(20%)	1/2(50%)

PIVSD= post infarction ventricular septal defect, IABP= intra aortic balloon pump, Anterior PIVSD= anterior location of post infarction ventricular septal defect (PIVSD) in midpart of interventricular septum (IVS) Posterior PIVSD= PIVSD in proximal and inferior part of interventricular septum (IVS), Values are presented as mean ± standard deviation, number (%) or median (range). MI= Myocardial infarction, CAG= Coronary angiogram, LAD= Left anterior descending coronary artery, RCA= Right coronary artery, LVEF=left ventricular ejection fraction, CABG=coronary artery bypass grafting, Aortic CC=Aortic cross clamp time, CPB= cardiopulmonary bypass time, LCOS= Low cardiac output state

Table-2: Analysis of baseline characteristics and perioperative variables in 7 patients with PIVSD according to survivability after operation whether survived (S) or died (D)

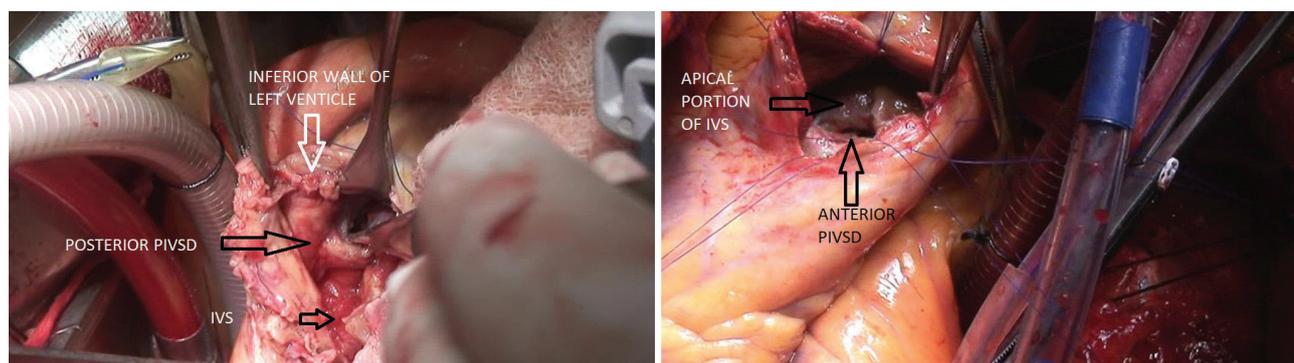


Figure-1: Photograph showing both the anterior (in lower frame) and posterior (in upper frame) post infarction ventricular septal defect (PIVSD). IVS= inter ventricular septum. Note the friable LV margin in the upper frame with the heart held up exposing the inferior wall.

80% for delayed surgery group at 3 to 6 years from 2012 to 2016 and followed till mid 2019.

On follow up, all the surviving patients had improved LVEF and 4 of them are still surviving after 5 years One patient (# No 6, vide Table No. 1) from the late surgery group had late

mortality after 3 years. He had single vessel 100% proximal LAD lesion and did not require CABG. He presented in OPD later with dilation of LV (LVEDD 64 and ESD 56) on 3rd year and succumbed despite medical management. Overall 3 year survival was 100% among the 5 survivors and 80% after 3

Variables	No of patients (n=7)	Late surgery group (more than 10 days) (n=5)	Early surgery group (within 3 days) (n=2)
Age (years) average ± means	7 (59.71±5.73)	5 (59.16±5.55)	2 (60.5±2.5)
Male sex	6(85.7%)	4 (57.1)	2 (100 %)
Thrombolysis	3(42.8%)	2 (28.5%)	1 (50%)
Anterior PIVSD	5(71.4%)	4 (57.1)	1 (50%)
Posterior PIVSD	2(28.5%)	1 (14.2%)	1 (50%)
IABP (preoperative) +inotropes	2/7(28.5%)	0 /5(0%)	2/2(100%)
Refractory Cardiogenic shock	2 (28.5%)	0/5 (0%)	2/2 (100%)
Time (days) from PIVSD to operation (days)	13.57±7.14	17.8± 2.99	3± 0
Site of block on CAG (n=7)			
LAD(Proximal 100% occlusion)	1	0	1 (D)
LAD (Mid 100% occlusion)	4	4(S)	0
RCA (proximal)*	1	1(D)	0
RCA (distal)	1	0	1(S)
Site of PI-VSD (n=7)			
Anterior	5	4	1(D)
Posterior	2	1	1(S)
Size < 10 mm	5	4(S), 1(D)*	1(D) **
Size > 10 mm	2	1(S)	1 (S)
Concomitant CABG	6 (85.7%)	4(57.1%)	2(100%)
Aortic CC time (min)	129 ± 10.10	130.2±13.21	128.5±3.53
CPB time (min)	178 ± 37.41	180± 40.04	169.5±48.79
Intensive care unit stay (days)	6.5±2.22	7.20±1.92	5±2.80
Postoperative LCOS			
Leading to death(n=3)	2/7 (28.5%)	1/5(20%)	1/2(50%)

PIVSD= post infarction ventricular septal defect, IABP= intra aortic balloon pump, Anterior PIVSD= anterior location of post infarct ventricular septal defect (PIVSD) in midpart of interventricular septum (IVS), Posterior PIVSD= PIVSD in inferior part of inter-ventricular septum (IVS), Values are presented as mean ± standard deviation, number (%) or median (range). CABG= coronary artery bypass grafting, LAD=Left anterior descending artery, RCA=Right coronary artery, Osteal= at the origin of the RCA or LAD, *=RCA proximal 100% lesion with a small <10 mm PIVSD postero inferior PIVSD, **= LAD proximal 100% lesion with a small <10 mm PIVSD , D=died, S=survived

Table-3: Analysis of Perioperative variables in 7 patients with PIVSD according to timing, Late surgery and Early surgery groups

Variables	No of patients N=7	Delayed surgery Group n=5	Early surgery Group n=2
30 day hospital mortality n=7	2/7 (28.5%)	1/7(14.28%)	1/7(14.28%)
Survivors after 2 years, n=5	5/7 (71.4%)	5/5 (100%)	1/1(100%)
Survivors after 5 years n=4	4/7 (57.1%)	3/5 (60%)	1 /1(100%)
Residual or recurrent shunt	0	0	0

Values are in number of patients = n, (%)

Table-4: Analysis of post operative survival, mortality and recurrence in 7 patients with PIVSD according to Late surgery and early surgery groups.

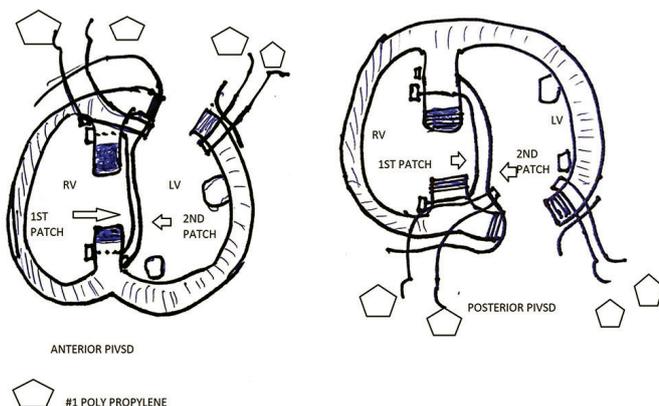


Figure-2: The diagram shows the method of repair of the post infarct ventricular septal defect (PIVSD) both anterior and posterior type. The 1st patch of gluteraldehyde preserved bovine pericardium is stitched to the inter ventricular septum (IVS) with interrupted # 3-0 polypropylene with pledgets on the RV (right ventricle) side through the VSD. A 2nd patch is stitched to the healthy portion of the IVS with same continuous suture with a few interrupted ones. In the cut margin of the LV, the patch is exteriorized with full thickness interrupted # 1 polypropylene and taking bites through the adjacent IVS. On the opposite end of the cut margin, similar interrupted #1 polypropylene sutures are also taken with the patch on the exterior side . Another layer of through and through #1 polypropylene suture used to approximate both sides with additional sutures if required.

year follow up (Table-4).

DISCUSSION

Pathophysiology of PIVSD

The conventional mechanism of septal rupture involves coagulation necrosis of ischaemic tissue with neutrophilic infiltration, eventually resulting in thinning and weakening of the IVS. This sub-acute process resulting in PIVSD requires 3–5 days, likely accounting for the traditional timing of rupture reported in the early surgical literature.³ PIVSD occurring within 24-h of presentation has been reported to be more common in a recent American study.² Early PIVSD has been described to be more likely due to dissection of an intramural haematoma or haemorrhage into ischaemic myocardium. This has been explained to occur due to physical shear stressors at the border of an infarct zone, combined with a hypercontractile, remote myocardial segment³ and highlights the value of beta blockers and afterload reduction after AMI. Extension of infarction occurs due to breakdown of inter-myocyte collagen struts by matrix metalloproteinases (MMP) which begins within three hours of infarction. MMP activity reaches peak around 7th post-infarction days. Deposition of collagen begins by days 2-4. Fibrosis is completed by the end of 4th week.⁷ This provides a pathophysiological basis for delaying VSD closure if possible.

Cardiogenic shock (CS)

Despite advances in the management of AMI like early reperfusion, which reduced the incidence of PIVSD from 1-2%, to 0.17 and 0.31% respectively,^{8,9} current mortality among patients with PIVSD remains high 41–80% and appears mostly unchanged over the last few decades.^{3,10} In-depth studies have shown that post surgical survival has been better in patients who presented with low to moderate hypotension and stabilized with pharmacological management with or without MCS and higher in patients who present with refractory CS and required tMCS.^{11,12} However, unwitnessed or prolonged cardiac arrest, disseminated malignancy, severe cerebral dysfunction, and severe aortic regurgitation are contra indication to tMCS.⁷ We had mixed result as far as refractory CS was concerned and one patient with less myocardial damage survived the operation and is discussed later.

Incidence and types of CS

Around 5 to 7% of patients of AMI present with CS. It seems to occur with a higher frequency (7.5%) amongst patients with ST-segment elevation myocardial infarction (STEMI) and in 2.5% of patients with non-ST-segment elevation myocardial infarction (NSTEMI). The underlying mechanism may be the rapid cell necrosis that takes place in STEMI in comparison to a slower cell loss in NSTEMI.^{4,13} Presentation of CS may be as milder forms of euvolemic or “cold and dry” type of CS are present in patients with pulmonary congestion and typically describes a diuretic, fluid and inotrope-responsive patient with a subacute decompensation.¹² It is known that approximately 50–60%

of CS patients survive without any MCS¹⁴ and they fall within this group with less mortality after stabilization when compared to the severe CS which is next described. Severe CS or classic “cold and wet” type are characterized by a low cardiac index (CI), a high post capillary pulmonary wedge pressure (PCWP) with inadequate response to diuretics and inotropes. Such patients are more likely to have had a previous MI or chronic kidney disease and significantly higher PCWPs in comparison to euvolemic cold and dry CS. However prolonged and neglected euvolemic CS, may be complicated with multi organ failure [MOD] and systemic inflammatory response syndrome (SIS), which depresses myocardial pumping function further^{12,14} and present as severe or refractory CS. Cold and wet CS, may have less shunt in PIVSD due to lower pumping action, and is indicative of extensive MI.¹⁵ These are less responsive to pharmacological management with fluids and diuretics and inotropes as the main functioning mass of myocardium is severely damaged or depressed. Such cases, and also the previously described refractory euvolemic CS, often require tMCS to maintain perfusion and are associated with higher risk for pump failure after surgical repair of PIVSD and higher (around 87.3%) mortality.¹⁶ Other mechanisms of such severe cold and wet CS may be due to RV stunning as a result of sudden pressure and volume load by higher degree of shunt through a large VSD, or severe ischemic mitral regurgitation and pulmonary edema.^{3,4,15} As the previously preserved contractile function becomes severely depressed due to stunning, there is higher requirement of tMCS, the most common prototype of which is IABP. Eventually, both the severe cold and wet CS and refractory euvolemic cold and dry CS may need higher modes of tMCS. In such a setting, advanced tMCS like VA ECMO may be required to improve tissue perfusion for stabilization. But during this phase of stabilization, which is often of short duration, definitive surgical procedure must be done. Recently ECMO has been reported to allow delayed surgical closure with survival benefit in PIVSD patients with refractory CS complicated with MODS⁷ with intelligent assessment of markers of perfusion like decrement of blood lactates for final surgical closure.

5 of our patients were in the category of euvolemic fluid responsive CS who were stabilized without any tMCS and operated after a mean 17.8±2.99 days in much stable state (vide Table No.1,2, 3). The LVEF in all these 5 patients were between 35 to 42% and had 80% survival following closure of PIVSD. Delayed surgical closure in those who could be stabilized with conservative management has proved to have increased survivability.^{3,7}

2 of our patients with cold and wet type of CS, required IABP, and were subjected to early surgery. One had an extensive MI with a smaller PIVSD and had LVEF 24%, did not survive surgery (vide Table No. 1, 2,3). The other patient, who had a large PIVSD with large shunt with 28% LVEF, and survived the early corrective surgery as the extent of MI was limited. In this patient shock was due to stunning by a large shunt as described above and preoperatively inserted IABP could be discontinued 3 days after surgery as cardiac

function improved. In cardiogenic shock, due to higher shunt rather than infarct size early tMCS followed by surgery is advocated¹⁵ and is agreeable to our findings. There are reports of early reversal of prolonged shock stabilized with IABP, soon after closure of apical PIVSD.¹⁷

Timing of intervention for closure of PIVSD in CS

There is an ongoing debate as to what type of management should be provided to those patients who present with CS and maintained on tMCS, which is, high risk immediate surgical closure² or delayed^{3,5,7} closure? In most cases, patients with refractory CS are offered tMCS, like IABP initially, as a bridge to recovery of cardiac function followed by surgery or sometimes as a bridge to a more advanced devices with higher flow rates like ECMO¹⁴ or micro axial pump like Impella (Impella CP® / SmartAssist – ABIOMED, Danvers, Massachusetts 01923 US) or Tandem heart (CardiacAssist, Inc, Pittsburg, PA, USA). The goal is bridge-to-decision, whether it is stabilization for surgical closure of PIVSD in patients with preserved myocardial mass or palliation or a more durable MCS such as implantable ventricular assist device (VAD)s or heart transplantation in those with extensive myocardial dysfunction.¹⁸ However, because of the high costs involved, such facilities are available in a small number of cardiac centers in India. Late initiation of tMCS may also allow MODS to develop. Once developed, it is difficult to reverse CS by simply increasing cardiac output with a circulatory assist device, be it IABP or ECMO. So, shorter time to initiation of MCS to unload the failing LV followed by definitive surgical or endovascular procedure in stabilized patients has been advocated. Delay can lead to development of MODS and systemic inflammatory response syndrome (SIRS).^{10,14}

Serial blood lactate estimation

Reduction in the requirements for hemodynamic support and improvement of metabolic parameters are useful indicators of the response to and ongoing need for tMCS support. Persistently elevated blood lactate values at 24 and 48 hours of tMCS support is a strong predictor of mortality.⁷ Higher tendency to clearance of lactates sequentially, indicates satisfactory tissue perfusion and pave the way for better post surgical survivability.^{19,20} Serially measured decrement of arterial lactate level during tMCS support as a monitoring tool for tissue perfusion²¹ provides the basis for recovery and lead to successful delayed surgical repair.^{7,14,20} Arterial Blood lactates were not routinely done in our cases but we feel it is a necessity to assess recovery.

Mortality

There was a higher mortality rate of 50% in patients presenting with cardiogenic shock and low LVEF in our study group. One patient each had early mortality within 3 days in early surgery and delayed surgery groups and both had past history of MI (vide Table No. 1). In the early surgery group preoperative shock due to anterior wall STEMI with a small midbasal anterior VSD was responsible for 1 mortality with persistent low cardiac output state (LCOS) post operatively. Pre operative LVEF was 24% in this patient. There was a

small 8mm PIVSD with an extensive LV infarction. He also had CABG performed. Similarly the posterior VSD in the Delayed surgery group, who had presented with RV failure and responded well to inotropes and volume replacement, also had a small PIVSD but with moderate RV and postero inferior LV infarction. But the RV contractility was not adequate after release of cross clamp. He had difficulty in weaning and needed IABP to wean off CPB. However, he succumbed after 3 days due to LCOS. Both these patients had proximal acute total occlusion in the proximal LAD and proximal RCA arteries respectively and had CABG to other stenotic arteries. So, residual myocardial function is an absolute necessity to regain contractility even if ECMO or other tMCS^{5,15} are applied postoperatively. Hence case selection by exclusion of cases like persistently high (more than 7.5 mmol/L lactates can avoid mortality which can be an indication for bridge to decision or higher MCS, as observed by Park²¹ and Mc Laughlin.⁷

We did not find any correlation of mortality with higher age and female sex. The surviving female is a six year survivor till date. But ours is a very small group of this rare complication of PIVSD and difficult to comment over the risk factors.

Factors for Survival advantage

There was a trend towards better long-term survival with concomitant CABG and absence of cardiogenic shock. The 30-day operative mortality in this series was 28.5%. Since our series involved only a few patients, it can not be counted as statistically significant but comparable to rates of 34% to 37% reported in other series.^{5,22,23,24} The bulk of our patients were maintained in a much stable state which allowed delayed surgery and this might have influenced the results. CS and early operation were incriminated for early mortality in several series.^{22,24,25,26} and is agreeable to our findings of one mortality out of two patients. If the cardiogenic shock is due to pulmonary to systemic blood flow ratio shunt rather than infarct size, survival is better. This has also been confirmed by other authors.¹⁵ Duration of aortic cross clamp has also been regarded as a predictor of early mortality. Our average cross clamp time was 129 ± 10.10 minutes, and CPB time was 178 ± 40.41 minutes in both early and delayed series. After a few cases, the CC and CPB times were reduced with experience. It was longer than the average clamp times of 82±40 minutes and CPB time 152± 52 minutes reported by others.²⁷ Delayed closure after stabilization and concomitant CABG may have added to better survival. A reduction of mortality rates from 26.3% in those without CABG to 21.2% with CABG²⁸ in similar cases has been reported. The survivors from both the early and delayed groups in our report returned to NYHA class 2 after recovery and 1 survivor later on achieved class 1 status. One patient from the late surgery group had late mortality at third year as described before in result section. Overall 3 year survival was 100% among the 5 survivors and 80% after 3 year follow up (Table No 4). There is little data reporting long-term survival in the literature. In their report of 68 patients undergoing surgery for PIVSD between 1988 and

2007, Fukushima and colleagues from Brisbane, Australia provided some valuable insight and predictors of long-term outcomes.²⁹ In their report, 85% of patients underwent urgent surgery for PIVSD within 48 hours of diagnosis, 71% had concomitant CABG, and 30-day mortality was 35%. The mean follow-up was 9.2 years. The actuarial survival at 1 year was 67%, 63% at five years, 51% at 10 years, and 36% at 15 years. Overall predictors of survival were comparable to our small series but they had better short-time outcomes despite predominantly early repair.

Techniques of patch closure

We adopted the double patch technique in 6 cases (85.7%)^{30,31} (vide Figure No.2) and an extra patch in 1 case (concomitant repair of the LV aneurysm). The incidence of post-operative residual shunt was 0% comparable to the incidence of 24 to 26% described in other series in which a variety of techniques were used.¹⁵ Double patch technique has been reported to decrease the incidence of residual shunt from 11% to 0%.³⁰

CONCLUSION

Apart from the established risk factors, we observed that

A. Late surgical closure of PIVSD after optimum waiting period for scar maturation is associated with better survivability.

B. Failure of improvement of hemodynamic status and tissue perfusion, on resuscitative measures with IABP or advanced MCS are associated with poor prognosis after surgical intervention for PIVSD.

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