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Original Article

Primary transcatheter closure of post-myocardial infarction ventricular septal rupture using amplatzer atrial septal occlusion device: A study from tertiary care in South India

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ABSTRACT

Objective: The study investigated effectiveness of transcatheter closure of post-myocardial infarction (MI) ventricular septal rupture (VSR) using atrial septal device (ASD) occluder in a cohort of patients admitted at our institute.

Method: This was a retrospective, observational and single center study, which included patients who were treated with transcatheter closure for post-MI VSR at our tertiary care center between May 2000 and August 2014 depending upon inclusion and exclusion criteria. Primary outcome was all-cause mortality at 30-days follow-up. The MELD-XI (Model for End Stage Liver Disease) score was used as a predictor for poor outcome in these patients.

Results: A total of 21 patients (mean age 66.4 ± 5.9 years) were included in the study. Study cohort predominantly included male patients (n = 15; 71.4%) and patients with single vessel disease (n = 15; 71.4%). Revascularization of the culprit lesion, before VSR closure, was attempted in 6 patients. Except one patient (treated with Cera® occluder), all patients were treated with Amplatzer® ASD occluders. Average diameter of VSR was 20.8 ± 6.9 mm. Diameter of the device used in the study ranged from 10 mm to 30 mm. Residual defect was detected in 13 patients (62%). All-cause mortality at 30-day follow-up was observed in 9 (42.9%) patients. Time to VSR closure, diameter of VSR, and serum creatinine levels were significantly related to the 30-day mortality. MELD-XI score was found to be strongly associated with increased risk of mortality.

Conclusion: Primary transcatheter VSR closure using ASD occluders is a feasible approach which can provide reasonable survival outcomes along with equitable mortality rates.

1. Introduction

Ventricular septal rupture (VSR) is a rare but lethal mechanical complication of acute myocardial infarction (MI). Although introduction of reperfusion therapy reduced the incidence of this deadly complication (from 1 to 3% to 0.2–0.5%), the prognosis is still poor.[1] When conservative treatment is applied, mortality approaches to 90–95% within two months of diagnosis without intervention.[2,3] Extremely poor outcomes can be explained by advanced age of the patient, existence of multiple co-morbidities, severity of coronary artery disease and hemodynamic instability.[4–7]

Surgery is the mainstay for the treatment of post-MI VSR. Evidences documented that surgery in the early stage is favorable for survival. Evidence based guidelines suggest immediate repair and closure of VSR to shorten duration of left-to-right shunting and duration of systemic hypoperfusion which ultimately lessen the chances of multiple organ failure, coma, and death.[8,9] However, in order to allow scarring of tissues surrounding the rupture and to achieve better anchoring of sutures, the surgeons usually delay the surgical repair for 3–4 weeks.[10] This delay further reduces the chances of survival in these patients. Recent series revealed high mortality rate, ranges from 20 to 87% in acute stages.[4–7] Hence, transcatheter closure of VSR has been introduced as a less invasive procedure providing a definitive single treatment for VSR, a bridge to subsequent surgical correction or in selected cases a procedure for residual defects closure after surgical repair.[2] As a result of lower incidence of the complication, there is dearth of clinical

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experience on transcatheter closure of post-MI VSR, particularly in Indian hospital setting. Hence, we designed this retrospective study to investigate effectiveness of transcatheter closure of post-MI VSR using atrial septal defect (ASD) occluder in a cohort of patients admitted at our institute over a period of 14 years.

2. Methodology

2.1. Study design and patient population

This was a retrospective, observational and single center study. The study included all the patients who were treated with transcatheter closure for post-MI VSR at our tertiary care center between May 2000 and August 2014 depending upon inclusion and exclusion criteria of the study. Inclusion criteria of the study were 1) Patients who were admitted or diagnosed with VSR as a result of preceding AMI; 2) patients who were treated with transcatheter closure of post-MI VSR during study period. However, the patients with congenital heart disease or patients who developed VSR as a result of trauma were excluded from the study.

Medical records, electronic charts, procedure reports and discharge summaries were reviewed for demographics and clinical characteristics of the patients including age, gender, type of myocardial infarction, culprit vessel(s), presence of co-morbidities, pre-procedural clinical characteristics, device related information and post-procedural outcomes. Though treatment was decided on an individual basis (in agreement with cardiac surgeon and interventional cardiologists), primary transcatheter closure was opted for unstable patients with significant risk factors (age, right

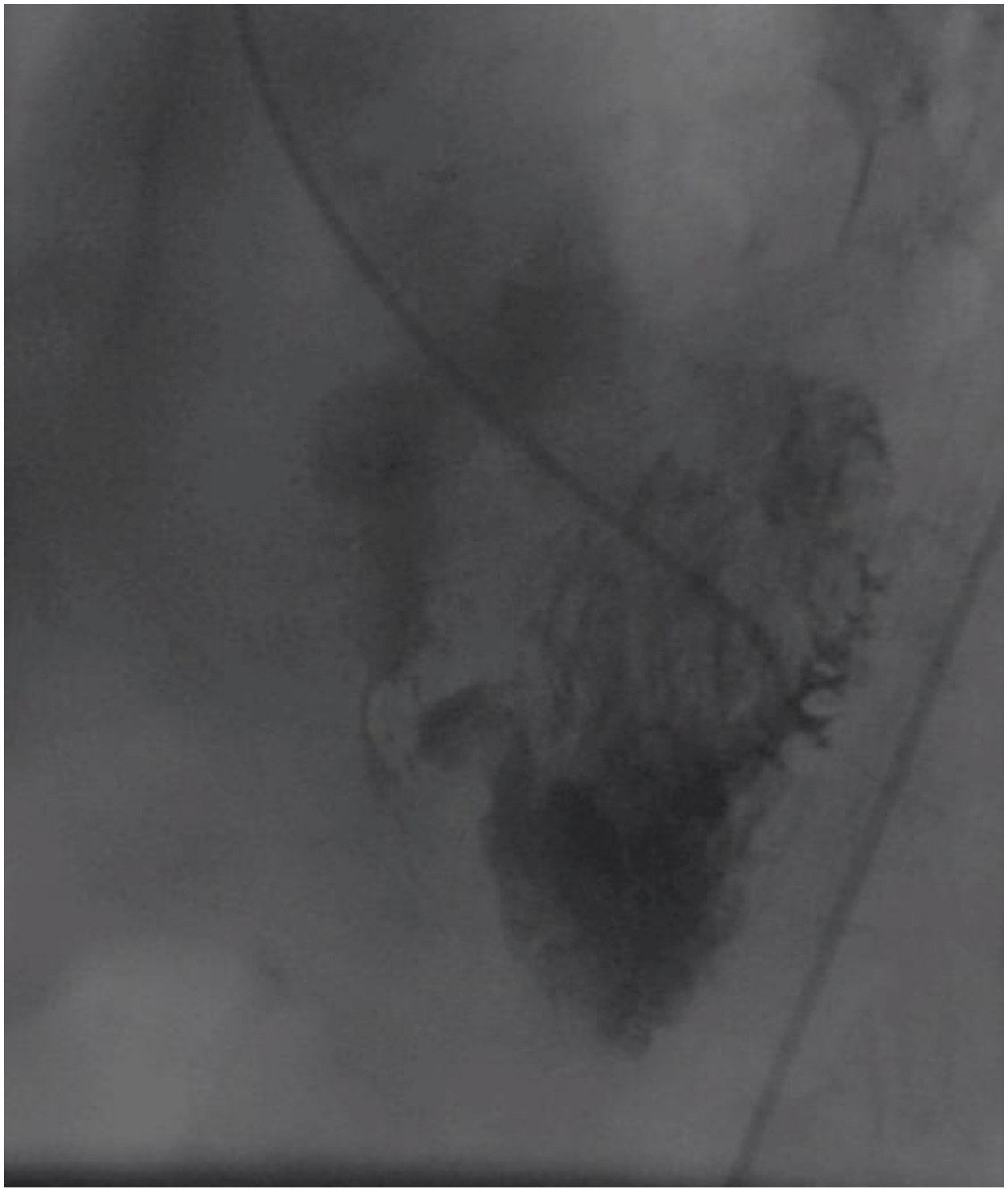

Fig. 1. LV angiogram of a post MI patient showing a defect across the inter-ventricular septum.

ventricular failure, low ejection fraction, major co-morbidities and need of intra-aortic balloon pump (IABP).

2.2. Study device

Except one patient (who was treated with Cera® occluder (Lifetech)), all the patients were treated with Amplatzer® ASD occluders (AGA Medical Corporation, Plymouth, MN, USA). Amplatzer® ASD occluders consist of two self-expanding umbrellas joined together with 'waist' (single spring-loaded stainless steel arm). These self-expanding umbrellas are composed of nitinol mesh coated with polyester fabric which provide a foundation for tissue growth over the device after deployment. Size of the device, as measured from diameter of waist, vary from 4 mm to 38 mm. The only difference between Amplatzer® occluder and Cera® occluder was the coating of titanium nitrate on nitinol mesh on latter to reduce the dissolution of nickel ion and thus provide safe long-term biocompatibility. Diameter of Cera® occluder device ranges between 4 mm to 24 mm.

2.3. Primary transcatheter procedure of VSR closure

Transesophageal echocardiography (TEE) was performed to determine size of VSR before commencing the transcatheter

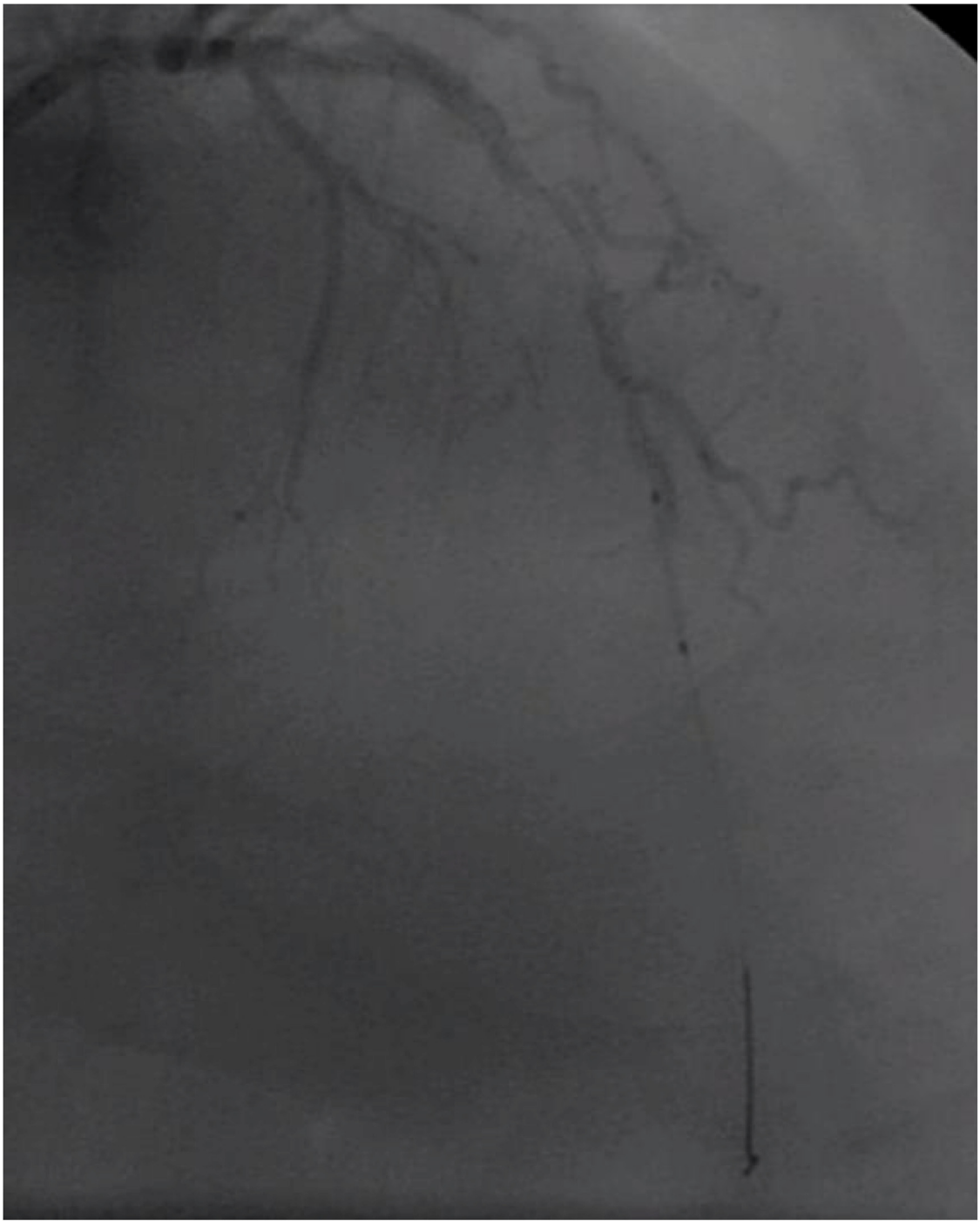

Fig. 2. Coronary angiogram of the same patient in PA cranial view showing a mid-LAD 90% lesion.

closure. The procedure was performed under fluoroscopic and echocardiographic guidance by experienced interventional cardiologists. All patients were administered prophylaxis antibiotic cefazolin (2 g), aspirin (500 mg) and non-fractioned heparin (60U/ kg bodyweight) intravenously. The standard technique of transcatheter closure was used (Fig. 1, Fig. 2). In brief, the right femoral artery and right internal jugular vein were accessed followed by establishment of an arterialvenous guidewire circuit (Fig. 3). After expansion of the discs, correct placement of the device was assured with echocardiography and left ventricular angiography (Fig. 4). Finally, the device was released by unscrewing the delivery cable (Fig. 5).

2.4. Study outcomes and definitions

Primary outcome was a composite of all-cause mortality at 30-days follow-up. Cardiogenic shock was defined as 1) persistent systolic blood pressure less than 90 mmHg or requirement of ionotropes to maintain blood pressure more than 90 mmHg; 2) requirement of IABP to maintain adequate circulation; 3) evidence of poor end-organ perfusion. Time to VSR closure was defined as the duration between symptom onset and VSR closure. MELD XI (Model for End Stage Liver Disease) score was used as a predictor for poor outcome in these patients. The score was calculated using creatinine and total bilirubin at the time of VSR closure according

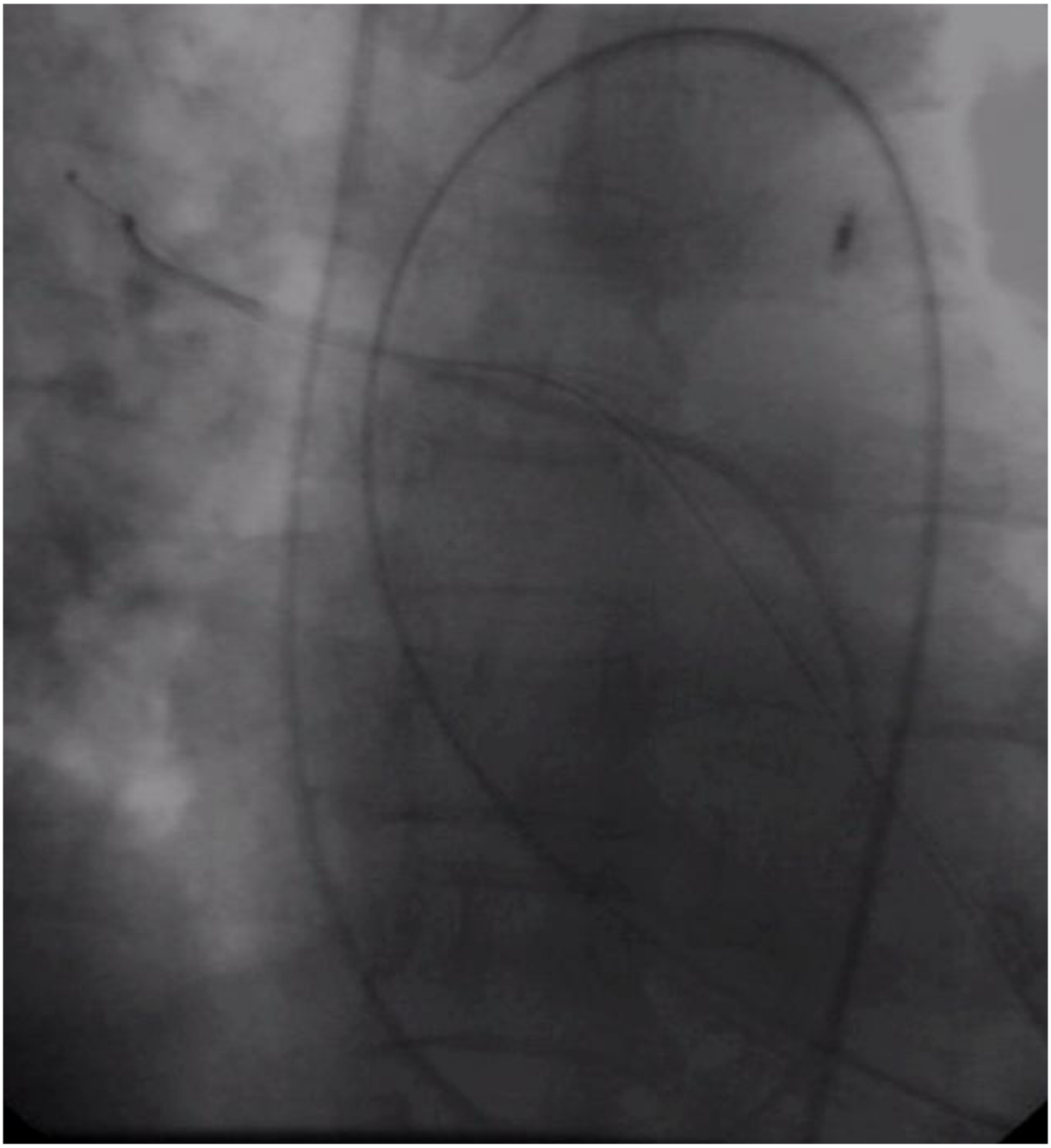

Fig. 3. Snaring process: After taking both the venous access through the internal jugular and the arterial through the femoral, two wires are passed from the femoral access through the defect and from the venous access into the pulmonary artery and snared.

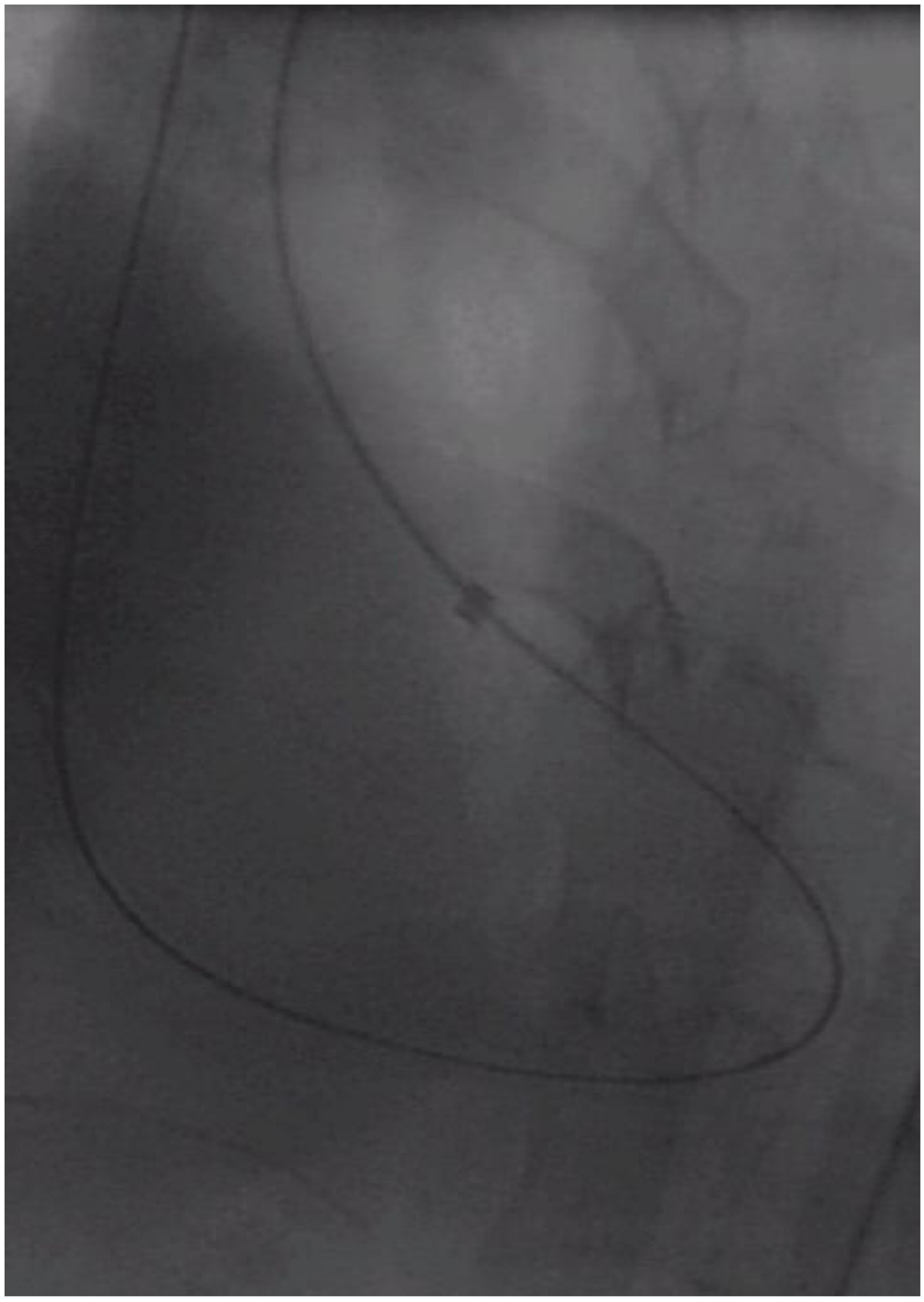

Fig. 4. Device Placement: The device is placed across the defect in the inter-ventricular septum.

to the following formula: 5.11 × ln (bilirubin mg/dL) + 11.76 × ln (creatinine mg/dL) + 9.44.[11]

2.5. Statistical analysis

Descriptive statistics was used and variables are expressed as either mean ± standard deviation (SD) or as numbers (percentage). We compared continuous variables by applying unpaired Student's *t*-test (normally distributed) or Mann-Whitney test (non-normally distributed). Univariate logistic regression was used to predict the primary outcome. The predictors used in the analysis were: time to VSR closure, diameter of VSR, and serum bilirubin and creatinine levels at the time of closure. Odds ratios (OR) are presented with 95% confidence intervals. A p value $\leq .05$ was considered statistically significant. Statistical analysis was performed using Statistical Package for Social Science (SPSS; Chicago, IL, USA) program, version 17.

3. Results

3.1. Baseline clinical characteristics of the study population

A total of 21 patients were included in the study. Mean age of the patients was 66.4 ± 5.9 years and 71.4% patients were male (n = 15). All the patients developed VSD after anterior wall MI. Coronary angiogram revealed single vessel disease in 71.4% patients (n = 15). Eighteen (85.7%) patients were thrombolyzed for MI. Revascularization of the culprit lesion, before VSR closure,

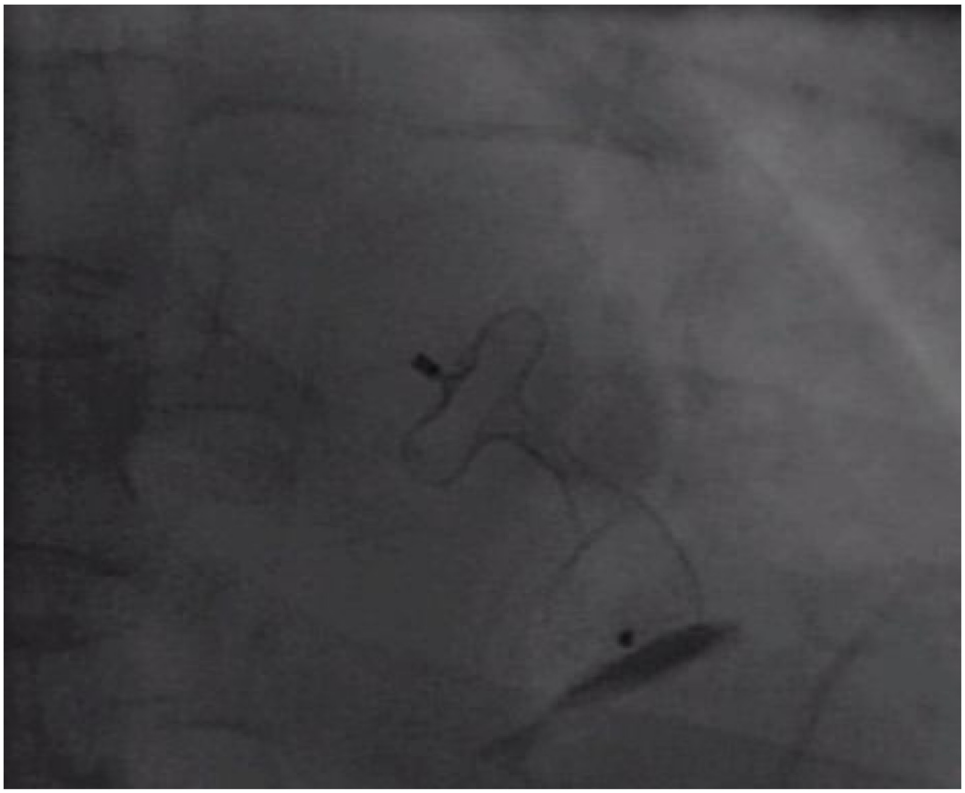

Fig. 5. Device deployment: The finally deployed septal occluder device across the inter-ventricular septum.

was attempted in 6 patients (n = 4 patients underwent percutaneous coronary intervention [PCI]; n = 2 patients underwent coronary artery bypass graft [CABG]). VSR was diagnosed within 24 h of onset of symptoms in 12 patients (57.1%). Table 1 summarized demographic details and pre-procedural clinical characteristics of the study cohort.

3.2. Transcatheter procedure of VSR closure and outcomes

Majority of the patients had smaller sized VSR ie. 10–15 mm (n = 9; 42.9%) and 5–10 mm (n = 7; 33.3%). Diameter of the devices used in this study ranged from 10 mm to 30 mm. In order to achieve maximum VSR closure, 30 mm device was used in 19% patients (n = 4) followed by 24 mm device (n = 3; 14.3%) and 16 mm device (n = 3; 14.3%). Details about VSR diameter and device characteristics are given in Table 2. Residual defect, as detected by echocardiography, was observed in 13 (62%) patients out of whom 5 patients failed to survive. Device embolization from the optimal position was seen in two patients. In 1 patient, a 14 mm device dislodged from the initial position which was then retrieved using a transcatheter approach. The patient was intensively treated with IABP and ionotrope therapy to prevent the complications of cardiogenic shock. The patient survived and was later treated with surgical correction of VSR. Another patient (who experienced

Table 1
Demographic detail and pre-procedural clinical characteristics of study population.

	Overall N = 21	Survivors n = 12	Non-survivors n = 9
Age, years (mean ± SD)	66.4 ± 5.9	66.8 ± 4.8	65.9 ± 7.3
Male gender, n (%)	15 (71.4)	10 (83.3)	5 (55.6)
Smokers, n (%)	7 (33.3)	3 (25)	4 (44.4)
Co-morbidities, n (%)			
Diabetes mellitus	13 (61.9)	8 (66.7)	5 (55.6)
Dyslipidemia	15 (71.4)	8 (66.7)	7 (77.8)
Previous history of coronary artery disease	4 (19.0)	2 (16.7)	2 (22.2)
Stroke	1 (4.8)	–	1 (11.1)
Time to ventricular septal rupture detection >24 h, n (%)	12(57.1)	7 (58.3)	5 (55.6)
Type of disease, n (%)			
Single vessel disease	4 (19.0)	3 (25.0)	1 (11.1)
Double vessel disease	15 (71.4)	8 (66.7)	7 (77.8)
Triple vessel disease	2 (9.5)	1 (8.3)	1 (11.1)
Revascularization of the culprit lesion, n (%)			
Percutaneous coronary intervention	4 (19.0)	1 (8.3)	3 (33.3)
Coronary artery bypass grafting	2 (9.5)	1 (8.3)	1 (11.1)
Thrombolysis before the procedure, n (%)	18 (85.7)	11 (91.7)	7 (77.8)
Left ventricular dysfunction at time of presentation, n (%)	16 (76.2)	9 (75)	7 (77.8)
Requirement of IABP[a] at time of presentation, n (%)	12 (57.1)	3 (25.0)	9 (75.0)
Requirement of ionotrope at time of presentation, n (%)	17 (81.0)	9 (75.0)	8 (88.9)
Pulmonary hypertension at time of presentation, n (%)	14 (66.7)	6 (50.0)	8 (88.9)

[a] Intra-aortic balloon pump.

Table 2
Characteristics of ventricular septal rupture and implanted device.

	Frequency (n)	Percentage (%)
Diameter of VSR[a] (mm)		
5–10	7	33.3
10–15	9	42.9
15–20	2	9.5
15–30	1	4.8
>30	2	9.5
Diameter of ASD[b] occluder (mm)		
10	2	9.5
12	1	4.8
14	2	9.5
16	3	14.3
18	1	4.8
20	2	9.5
22	1	4.8
24	3	14.3
28	2	9.5
30	4	19.0
Patients experienced residual defect		
Survivors	8	38.1
Non-survivors	5	23.8
Patients experienced device embolization		
Survivors	1	4.8
Non-survivors	1	4.8

[a] Ventricular septal rupture.
[b] Atrial septal defect.

device dislodgement) was treated with 24 mm device for 20 mm VSR. The patient failed to survive following device embolization, despite of intensive treatment with IABP and ionotrope therapy. With the exception of the one patient with device embolization, none of the patient population was treated with a surgical repair to manage left-to-right shunt.

A significant association was observed between the time to VSR closure and 30-day mortality. Higher mortality rate was observed in the patients treated within 5.9 ± 3.3 days as compared to those treated at 21.1 ± 14.3 days ($p < .008$). Higher mortality rate at 30-day follow-up was also related to higher serum bilirubin ($p < .02$), serum creatinine levels ($p < .002$) and MELD-score ($p < .006$) at the time of VSR closure. Patients who required IABP to manage cardiogenic shock at the time of VSR closure had higher rate of death due to multiple organ failure ($p < .001$). Clinical and laboratory characteristics of the study population at the time of VSR closure are given in Table 3.

3.3. Predictors of 30-day mortality

Primary outcome of the study, mortality within 30 days of VSR closure, was observed in 9 patients (42.9%). All deaths were due systemic hypoperfusion followed by cardiogenic shock. The patients could not revive despite aggressive medical therapy, inotropic support or use of IABP. The patient who was treated with Cera® occluder survived without any major complication. Factors associated with 30-day mortality are presented in Table 4. Time to VSR closure, diameter of VSR, and serum creatinine levels were all significantly related to 30-day mortality. MELD-XI score was also strongly associated with increased risk of mortality.

4. Discussion

Post-MI VSR is a life-threatening condition, mainly because of the associated incidence of multiple organ failure. The present study retrospective analyzed patients who were treated with primary transcatheter closure using ASD occluder for post-MI VSR closure.

4.1. Thirty-day mortality and VSR occluder

High rates of 30-days mortality (42.9%) was observed in the present study. Previous studies also reported high mortality rates with primary transcatheter closure procedure. Reported 30-day mortality (using Amplatzer occluder) ranges between 28% and 42%.[10,12,13] High mortality rate with the device may be attributed to its semipermeable nature at the time of implantation. It takes a number of days for complete occlusion of the device by organized thrombus. High transventricular pressure may also lead to a persistent left-to-right shunt via VSR until complete thrombus mediated occlusion and endothelialization of the device occurs. Highly unstable patients with cardiogenic shock may not tolerate this persistent residual shunting even for few days and may die.[14] Other concern with the use of Amplatzer® device is its rigid structure which can increase the risk of myocardial rupture and thus increase the VSR size and leading to residual defect or device embolization. The present study used Amplatzer® ASD occluder as ventricular septal occluders (for treatment of post-MI VSR) were not available during the course of the study. Amplatzer® ASD occluders have suboptimal profile in treating VSR as the short waist of the device can lead to device deformation (cobra effect) after deployment.[10] This may also explain high mortality rate of our study. Assenza et al also found similar high mortality rate (42%) at 30-day follow-up with first generation Clamshell device, second generation CardioSEAL device, and third generation STARFlex device.[15] These findings warrants for further research to better understand the working of septal occluder devices in primary closure of post-MI VSR.

4.2. Cardiogenic shock at the time of VSR closure and outcomes

Expert consensus and previous research suggests that cardiogenic shock (observed as a need for IABP and ionotrope management), poor systemic perfusion (observed as increased serum creatinine and bilirubin), and hemodynamic burden due to left-to-right shunt (observed as increased pulmonary arterial hypertension (PAH)) at the time of VSR closure are important predictors of mortality in patients treated with primary transcatheter closure.[13–15]

Table 3
Clinical and laboratory features of the study population.

	Over all (N = 21)	Survivors (n = 12)	Non-survivors (n = 9)	p-value
Time to VSR[a] closure (days), mean ± SD	14.6 ± 13.3	21.1 ± 4.3	5.9 ± 3.3	0.008
Diameter of VSR (mm), mean ± SD	20.8 ± 6.9	17.8 ± 6.7	24.7 ± 5.3	0.022
Creatinine at time of VSR closure (mg/dL), mean ± SD	1.97 ± 1.01	1.55 ± 0.29	2.53 ± 1.35	0.002
Bilirubin at time of VSR closure (mg/dL), mean ± SD	1.17 ± 0.44	1.01 ± 0.43	1.38 ± 0.37	0.020
INR[b] at time of VSR closure, mean ± SD	1.45 ± 0.32	1.33 ± 0.22	1.61 ± 0.38	0.075
MELD-XI[c] at time of VSR closure, mean ± SD	10.67 ± 4.09	9.42 ± 1.73	12.33 ± 5.68	0.006
Leukocytes at time of VSR closure (cells/μL), mean ± SD	12085 ± 6045.3	10908 ± 6583.4	13655.56 ± 5188.2	0.117

[a] Ventricular septal rupture.
[b] International normalized ratio.
[c] Model for End Stage Liver Disease-XI.

Table 4
Predictors of 30-day mortality.

Variable	Unadjusted Odds ratio (95% confidence interval)	p-value
Time to VSR[a] closure (hrs)	0.80 (0.63–1.01)	0.050
Diameter of VSR (mm)	1.20 (1.01–1.41)	0.036
Serum creatinine at the time of VSR closure (mg/dL)	1.06 (1.01–1.11)	0.020
Bilirubin at the time of VSR closure (mg/dL)	11.85 (0.82–171.09)	0.070
MELD-XI[b] score at the time of VSR closure	1.13 (1.03–1.23)	0.009

[a] Ventricular septal rupture.
[b] Model for End Stage Liver Disease.

In the present study, we also observed strong associations between the requirement of IABP for managing complications of shock with high mortality rate ($p < .001$) and increased serum creatinine level ($p < .002$) with higher mortality rate ($p < .001$). However, we were unable to establish any association between high mortality rates and requirement of ionotropic support, increased serum bilirubin levels, and PAH.

4.3. Time to VSR closure

Another study showed experience of transcatheter closure of post-MI VSR in 18 patients. The authors found statistically significant reduction in post-procedural mortality in patients who were treated after three weeks of symptom onset ($p < .001$).[12] A systematic review of 31 published studies for assessing effectiveness of transcatheter closure of post-MI VSR using Amplatzer® occluder concluded that if the VSR closure was delayed by 2 weeks, especially in the patients with any signs of cardiogenic shock, a statistically significant improvement in survival rates was observed ($p < .005$).[16] The present study also observed that duration to VSR closure as an important predictor of 30-day mortality. It was found that the patients treated with transcatheter VSR closure after 17–21 days of symptom onset were more likely to survive than the patients treated within 7–8 days of symptom onset (OR: 0.80 (0.63–1.01) Cl:95%, $p < .05$). It is noteworthy that patients intervened upon after 3 weeks do better because they are naturally selected subset of patients. According to the experts, delay in intervention not only allows myocardium to recover its function, aids in stabilizing the patient for hemodynamic and perfusion changes but also allows to identify VSR size once it matures properly and thus reduces the changes of device dislodgment or residual shunting.[16]

4.4. MELD-XI score as a predictor of 30-day mortality

MELD-XI score has been used by multiple researchers to determine the risk level associated with multiple organ failure in patients with cardiac diseases. Numerous studies on transcatheter or surgical closure of VSR in patients with MI or patients with congenital heart defect have found it to be strongly associated with 30-days mortality.[11] Assenza et al. studied 30 patients with post-MI VSR over the period of 20 years.[15] They observed that 62% of patients with MELD-XI score $\geq$20 could not survive for 30-days after transcatheter closure of post-MI VSR. Our study showed that 42.9% patients with MELD-XI scores between 12 and 17 died within 30-days of VSR closure (OR: 1.13 (1.03–1.23); $p < .009$). These findings suggest that MELD-XI score at the time of VSR closure can be used as an important predictor of 30-days mortality in the patients undergoing primary transcatheter VSR closure.

4.5. Limitations of the study

We acknowledge limitations of the study. Limitations are majorly those factors which are inherent in any retrospective study. The present study also skipped the proper diagnosis and assessment of post-procedural residual defect. In view of smaller patient population, multivariate analysis could not be performed. Hence, independent predictors of mortality which has adequate specificity and selectivity were not identified.

5. Conclusion

In conclusion, primary transcatheter closure using ASD occluder for post-MI VSR is a feasible approach which can provide reasonable survival outcomes along with equitable mortality rates. Moreover, MELD-XI score, duration to VSR closure, requirement of IABP, and increased serum creatinine levels are also associated with higher 30-days mortality in these patients.

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