PREVENTIVE PCI VERSUS CULPRIT ARTERY PCI WITH SECOND GENERATION DRUG ELUTING STENTS IN SETTING OF ACUTE ST-SEGMENT ELEVATION MYOCARDIAL INFARCTION

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Abstract

Objectives: The purpose of this study was to compare the clinical outcomes of the preventive percutaneous coronary intervention (PCI) versus culprit artery PCI with 2nd generation drug eluting stents (DES) in setting of acute ST-segment elevation myocardial infarction (STEMI).

Background: Timely reperfusion of the culprit vessel improves survival. However, the management and revascularization strategy for stenosis in non-culprit artery is still debated.

Methods: Acute STEMI patients with multi-vessel disease (MVD) undergiong primary PCI between December 2014 and October 2015 were divided into 1-Preventive PCI (Non culprit artery PCI during the index primary procedure) and 2- Culprit artery PCI. Mortality rates and clinical outcomes were compared between the two groups in hospital and during 12 months after discharge.

Results: One hundered patients had STEMI and multivessel-disease, 49 (49%) patients were assigned to preventive PCI and 51 (51%) were assigned to culprit artery PCI. There was no difference in clinical characteristics between the two groups. Although preventive PCI took significantly more, stents (mean 2.82±0.858 versus 1.25±0.523, P<0.001), contrast amount (203.4±29.5 ml versus 162.5 ±23.9 ml, P<0.001) and total procedural time (57.16±6.9 min versus 47.8±4.1 min, P<0.001), compared to culprit artery only PCI, no significant difference in periprocedural safety outcomes of stroke, major bleeding and CIN rates (8.1% vs 5.9%,P=0.658).There was no benefit on mortality or recurrent MI at 12 months follow up after discharge (8.1% vs 11.8%,P= 0.553). Repeated revascularization and refractory angina were significantly reduced in the preventive PCI compared to culprit artery PCI (38.8%vs 60.8%,P= 0.0278)

Conclusion: The preventive PCI with 2nd generation DES appears to be safe as culprit artery PCI with effective reduction of refractory angina and repeated revascularization in selected patients but no benefit on mortality or recurrent MI.

Keywords: Preventive PCI, Culprit artery PCI, Myocardial infarction, Multi-vessel disease, Drug Eluting Stents

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1. Introduction

About 50% of patients with ST- segment elevation myocardial infarction (STEMI) have multi-vessel coronary artery disease. [1] The higher morbidity and mortality seen in STEMI patients with MV-CAD are likely multifactorial and include the presence of diffuse atherosclerosis as a harbinger of plaque instability, total ischemic burden, and impaired contractility of non-infarct zones in the presence of multiple obstructive stenosis. [2] In patients with acute myocardial infarction the multi-vessel PCI may offer advantages over a strategy of culprit lesion–only PCI because plaque instability may not be limited to the infarct-related artery but may involve other territories in the coronary vasculature.[2] Moreover, complete revascularization has been associated with improved long-term clinical outcome in patients with stable coronary artery disease (CAD).[3-5] Finally, patients and clinicians are often more comfortable with complete revascularization rather than medical therapy for angiographically significant residual coronary stenoses, especially if they are associated with a large territory of myocardial jeopardy.

Conversely, assessment of bystander disease severity, both visually and functionally, may be difficult in the acute setting [6], and thus treating these stenoses may not be wholly justified at the time of index intervention. Furthermore, multivessel revascularization is associated with coronary microembolization, iatrogenic myocardial infarction (MI), coronary reserve reduction,[7] and increased risk of contrast nephropathy.[8] Despite the technical improvements in the coronary intervention field, the introduction of noble drug-eluting stents, and the use of newer anti-platelet agents[9], the optimal management of patients with multivessel disease in setting of acute ST elevation myocardial infarction remains still unclear.

2. Aim of the study

To compare the clinical outcomes of the preventive PCI versus culprit artery PCI with 2nd generation DES in setting of acute ST-segment elevation myocardial infarction.

3. Methods

3.1. Study population

This prospective observational study enrolled one hundred eligible STEMI patients presenting to ER

in Al Hussien University Hospital and National Heart Institute, Egypt from December 2014 to October 2015. Patients with acute STEMI within 12 hours from symptoms onset with stenosis of ≥ 70 % in one or more coronary arteries other than infarct artery, ≥ 2.5 mm in diameter and technically amenable to stenting were included. Both infarct artery only PCI and preventive PCI were acceptable treatment options. Patients with cardiogenic shock at admission, previous history of PCI, CABG , contraindication to the administration of aspirin, heparin or clopidogrel and serum creatinine level >1.4 mg/dl were excluded. Unsuccessful infarct related artery PCI, left main disease, chronic total occlusion, two possible infarct arteries, single-vessel disease and PTCA only to either culprit or non culprit artery were excluded as well. Patients were able to verbally confirm understanding of risks, benefits of treatment strategies by PCI with 2nd generation DES, and legally authorized to provide a written informed consent prior to any study related procedure.

The ethics committee of each participating hospital approved the study. Routine transthoracic echocardiography was performed during hospitalization, and every 3 months after discharge till one year to assess LVEF% using Simpson's method.

3.2.Percutaneous coronary intervention procedures

All primary PCIs were performed according to the international guidelines on myocardial revascularizations. Before PCI all patients were received a bolus of 70 IU/kg of unfractionated heparin , 325 mg of asprin, 600 mg of clopidogrel and additional heparin was administered to patients to maintain activated clotting time at 250 to 300 s. Glycoprotein (Gp) IIb/IIIa receptor inhibitors was administered in large thrombotic burden on angiography, while the use of thrombus aspiration systems was at the interventional cardiologist's discretion. Four types of permanent polymer 2nd generation drug-eluting stents during the index procedure were used at discretion of each cath lab resources preferable as possible the everolimus-eluting stents because they are proven safe and efficient; Endeavor®(Medtronic Vascular, Inc., Santa Rosa, CA, USA), Resolute®(Medtronic Vascular, Inc.), Xience V®(Abbott Cardiovascular Systems, Inc., Santa Clara, CA, USA), PROMUS Element®(Boston Scientific Scimed, Inc. Corporation). After the completion of PCI in culprit artery, eligible patients were assigned to undergo immediate preventive PCI in non-infarct arteries with ≥ 70 % stenosis (**preventive PCI**) or no further PCI procedures (**Culprit artery PCI**). Successful PCI was documented by self-reporting of operator in each center and traditionally accepted when defined to achieve angiographic success without associated in-hospital major clinical outcomes such as death, MI, cerebrovascular event and emergency CABG. [9]

3.3. Clinical outcomes and End points

3.3.1.Follow up

In-hospital and 12 months clinical follow up after hospital discharge. Patients were seen at 3,6,9 and 12 months at study centers outpatient clinics for adverse clinical events. The primary endpoint was the occurrence of cardiac death. The secondary endpoints included the individual components of the MACE contained; death, myocardial infarction, stent thrombosis, or any repeated revascularization, as well as major bleeding, contrast-induced nephropathy (CIN), refractory angina and stroke.

3.3.2.Repeated revascularization

Repeated revascularization was defined as either PCI or CABG for any reason.[10] Subsequent PCI or CABG for angina was recommended only in case of refractory angina supported by objective evidence of ischemia with abnormal results on exercise electrocardiography, stress echocardiography or stress nuclear perfusion scan.

3.4. Statistical analysis

Continuous variables were presented as means ± standard deviation and compared using the Student's t test, while categorical variables, expressed as relative frequencies, were compared with the chi-square test or Fisher's exact test. A p value of <0.05 was considered statistically significant.

4. Results

4.1. Demographic characteristics

Out of 100 patients with STEMI and MVD, 49 (49%) received preventive PCI and 51 (51%) underwent culprit artery PCI. The enrolled patients had a mean age of 54.96±10 years which did not differ between the two groups. A lower proportion of patients in both groups were female. Smoking and diabetes were the most common cardiovascular risk. Concerning other risk comorbidities such as obesity (BMI >30kg/m^2), hypertension, dyslipidemia, history of MI, cerebrovascular disease, peripheral arterial disease and family history of CAD , are presented in table (1).

4.2.Cilnical status

There were no statistically significant differences between the studied groups in terms of ECG presentation, with a similar distribution of anterior and non-anterior MI. No significant differences between the groups in mean systolic-diastolic blood pressure, heart rate and heart failure at admission. Prevalence of impaired left ventricular function where higher in both groups 46.3±3.6 vs. 46.6±4.9 p=0.709.

Table (1) Demographic characteristics of study population

Characteristics	Total no 100	Culprit artery PCI no= 51 (51%)	Preventive PCI no=49 (49%)	p value
Age (years)	54.96±10	53.75±9.6	56.22±10.34	0.219
Female	17(17%)	9 (17.6%)	8 (16.3%)	0.86
Risk factors and comorbidities				
BMI >30kg/m2	10 (10%)	6 (11.8%)	4 (8.2%)	0.548
Current smoker	51 (51%)	26 (51%)	25 (51%)	0.997
Hypertensive	38 (38%)	21 (41.2%)	17 (34.7%)	0.504
Diabetic	45(45%)	23 (45.1%)	22 (44.9%)	0.984
Dyslipidemic	37 (37%)	18 (35.3%)	19 (38.8%)	0.718
Previous MI	13 (13%)	6 (11.8%)	7 (14.3%)	0.708
PAD	2 (2%)	1 (2%)	1 (2%)	0.977
Previous stroke	5 (5%)	2 (3.9%)	3 (6.1%)	0.614
Family history of CAD	14 (14%)	6 (11.8%)	8 (16.3%)	0.511

BMI; body mass index. CAD; coronary artery disease. PAD; peripheral artery disease. Plus–minus values are means ±SD. p < 0.05 statistical significant

Table (2) Clinical status of the studied patients at presentation

Characteristics	**Total** no 100	**Culprit artery PCI** no= 51 (51%)	**Preventive PCI** no=49 (49%)	**p value**
HR (bpm)	78.26 ±15.4	79.82±17.89	76.63±12.3	0.304
SBP	132.4±13.4	131.8±12.7	133±14.1	0.631
DBP	87.5±11.9	85.9±11.9	89.2±11.8	0.168
ECG findings				
Anterior MI	45 (45%)	25 (49%)	20 (40.8%)	0.763
Inferior MI	40 (40%)	20 (39.2%)	20 (40.8%)	
Lateral MI	8 (8.0%)	4 (7.8%)	4 (8.2%)	
Posterior MI	3 (3.0%)	1 (2.0%)	2 (4.1%)	
LBBB	4 (4.0%)	1 (2.0%)	3 (6.1%)	
Killip class				
I	80 (80%)	41 (80.4%)	39 (79.6%)	0.818
II	17 (17%)	8 (15.7%)	9 (18.4%)	
III	3 (3%)	2 (3.9%)	1 (2.00%)	
Initial LVEF %	46.5±4.2	46.6±4.9	46.3±3.6	0.709
CK-MB (mg/dl)	50.1±23.6	49.9±20.8	50.3±26.5	0.939
Positive troponin	89 (89%)	48 (94.1%)	41 (83.7%)	0.095
S.creat.(mg/dl)	0.927±0.1	0.93±0.2	0.92±0.1	0.779

HR; heart rate. LVEF; left ventricular ejection fraction. SBP; systolic blood pressure. DBP; diastolic blood pressure. LBBB; left bundle branch block. $p < 0.05$ statistical significant

4.3.Coronary angiography and coronary artery disease

Assigned patients to radial vs femoral access sites were 29% vs 71%. The left anterior descending artery was the culprit artery in 49% of patients and the non- culprit artery in 40%. The complexity of coronary artery disease was similar in the both groups, table (3).

Table (3) Coronary angiography and coronary artery disease

Characteristics	**Total**=100	**Culprit artery PCI**=51(51%)	**Preventive PCI**=49(49%)	**P value**
Access site				
Femoral	71 (71%)	36 (70.6%)	35 (71.4%)	0.927
Radial	29 (29%)	15 (29.4%)	14 (28.6%)	
Culprit artery				
LAD	49 (49%)	26 (51%)	23 (46.9%)	0.952
CX	12 (12%)	6 (11.8%)	6 (12.2%)	
RCA	34 (34%)	17 (33.3%)	17 (34.7%)	
others	5 (5%)	2 (3.9%)	3 (6.1%)	
Proximal LAD	28(28%)	12 (23.5%)	15(30.6%)	0.43
Culprit artery preTIMI flow				
0	65 (65%)	33 (64.7%)	32 (65.3%)	0.71
1	12 (12%)	5 (9.8%)	7 (14.3%)	
2	16 (16%)	9 (17.6%)	7 (14.3%)	
3	7 (7%)	4 (7.8%)	3 (6.1%)	
Disease extent				
2VD	76 (76.0%)	40 (78.4%)	36 (73.5%)	0.565
3VD	24 (24.0%)	11 (21.6%)	13 (26.5%)	
LAD non-culprit artery	40 (40%)	20 (39.2%)	20 (40.8%)	0.871
Proximal LAD non-culprit artery	14 (14%)	6 (11.8%)	8 (16.3%)	0.515

LAD: left anterior descending artery. CX: circumflex artery. RCA: right coronary artery. VD: vessel disease. $p < 0.05$ statistical significant

4.4. PCI Strategy

Angiographic success was accepted when the minimum diameter stenosis of < 10% (with an optimal goal of as close to 0% as possible) visually assessed by angiography and there should be final TIMI flow grade 3 which was achieved in 100% of patients. The type of 2nd generation stent used was significantly different between groups (P=0.016), especially everolimus eluting (P=0.034). Although preventive group took significantly more, stents, contrast amount and total procedural time, compared to artery group (P<0.001), the procedural success did not differ significantly between the two groups (0.288) , table 4.

Table (4) Comparison of PCI strategic procedural aspect

Characteristics	Total=100	Culprit artery PCI=51 (51%)	Preventive PCI=49 (49%)	p value
Thromboectomy device use	15 (15%)	7 (13.7%)	8 (16.3%)	0.716
Type of 2nd generation DES				
EES (XIENCE V/PROMUS)	58 (58%)	26 (51%)	32 (65.3%)	0.034
Xience V	49 (49%)	19 (37.3%)	30 (61.2%)	0.016
Promus	9 (9%)	7 (13.7%)	2 (4.1%)	
Endeavor	5 (5%)	1 (2%)	4 (8.2%)	
Resolute	37 (37%)	24 (47.1%)	13 (26.5%)	
No of stents / culprit artery	1.3±0.5	1.2 ±0.5	1.33±0.5	0.338
Total stent number	2.02±1	1.25 ±0.5	2.8±0.85	<0.001
GpIIb/IIIa use	14 (14%)	6 (11.8%)	8 (16.3%)	0.511
Contrast amount (ml)	182.55±3	162.55±2	203.4 ±29.5	<0.001
Total procedural time (min)	52.4± 7.3	47.8± 4.1	57.16± 6.947	<0.001
CP2B duration (hrs)	7.91± 1.8	7.775 ±1.5	8.051± 2.1	0.446
Procedural success	96 (96%)	50 (98%)	46 (93.9%)	0.288

DES: drug eluting stent. EES: everolimus eluting stent. CP2B: chest pain to balloon. Plus–minus values are means ±SD. $p < 0.05$ statistical significant

4.5. In-hospital complications and mortality

No in hospital mortality in our studied population. Malignant arrhythmia (VT/VF) was the most common complication. Other hospital complications were variable, but non of them was statistically significant between the preventive PCI and culprit artery PCI. The prevalence of major bleeding (0% vs 3.9%), Stroke (2% vs 0%), CIN (4% vs 2%), cardiogenic shock (2% vs 5.9%), PCI-related MI (4.1% VS 2%), urgent PCI (4.1% vs 5.9%) and minor bleeding (6.1% vs 7.8%), were similar. There were no cases referred to urgent CABG in either groups, Fig (1).

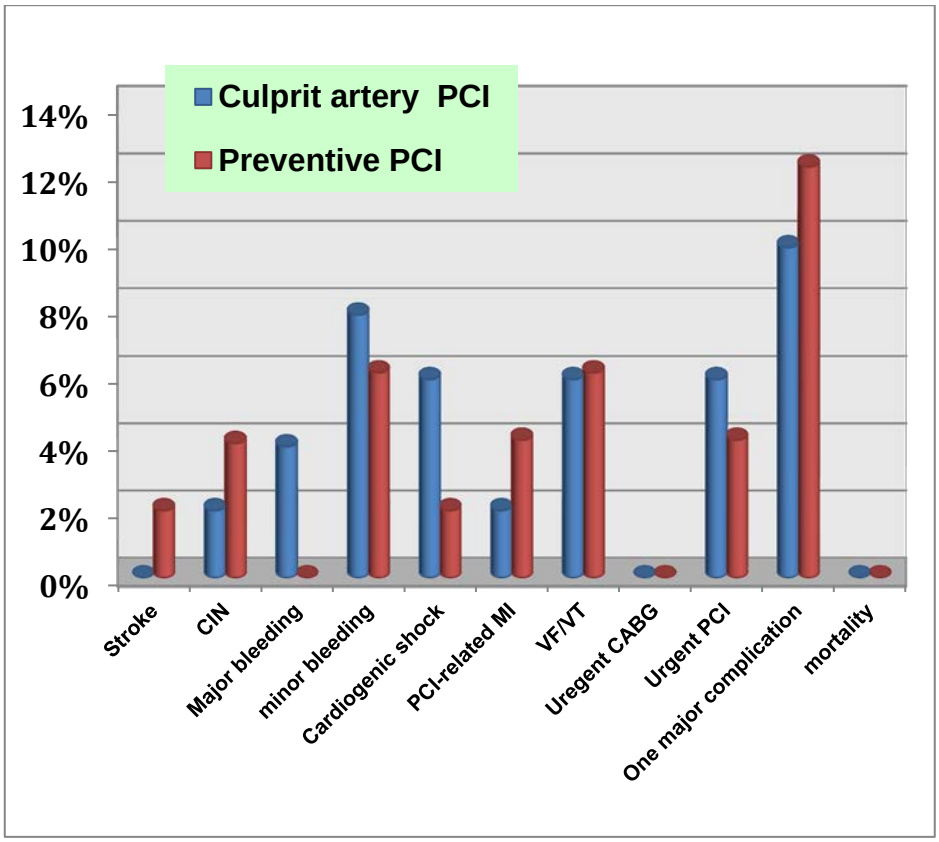

Figure (1) Comparison of in-hospital outcomes of the studied groups.

4.6. Discharge drug therapy

Culprit artery PCI group needed more cardiac medications, especially nitrates but this higher need for medications was not statistically significant, p=0.056, Fig (2).

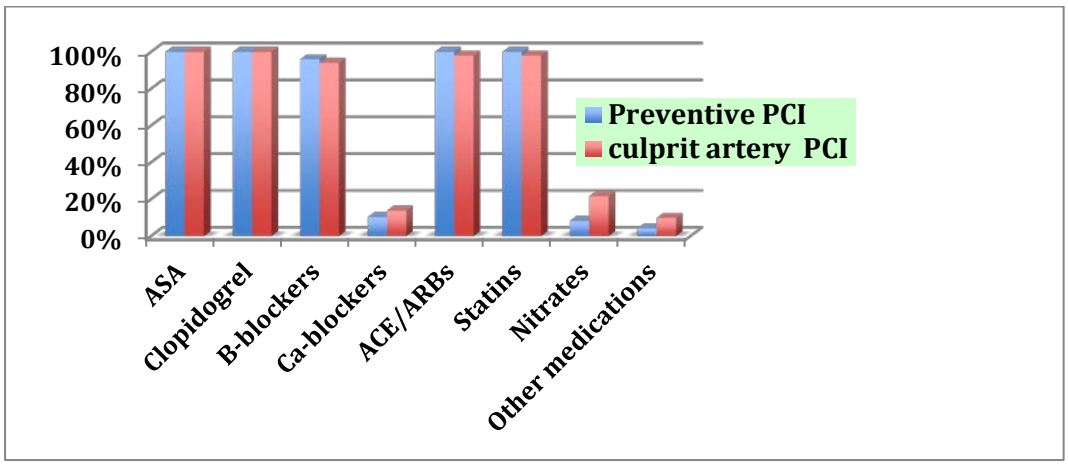

Figure (2) Comparison of the discharge medications of the studied groups.

4.7. One year outcomes

Clinical follow-up was performed for 12 months. Patients were seen at 3,6,9 and 12 months to document major adverse cardiac events (MACE) comprising all-cause mortality, recurrent MI, heart failure (HF), and ischemic-driven revascularization by PCI or CABG. while the one year cardiac mortality tended to be decreased in the preventive group but this decrease was non significant 2% vs 3.9%, p=0.586. There was no non-cardiac mortality in both groups Fig (3).

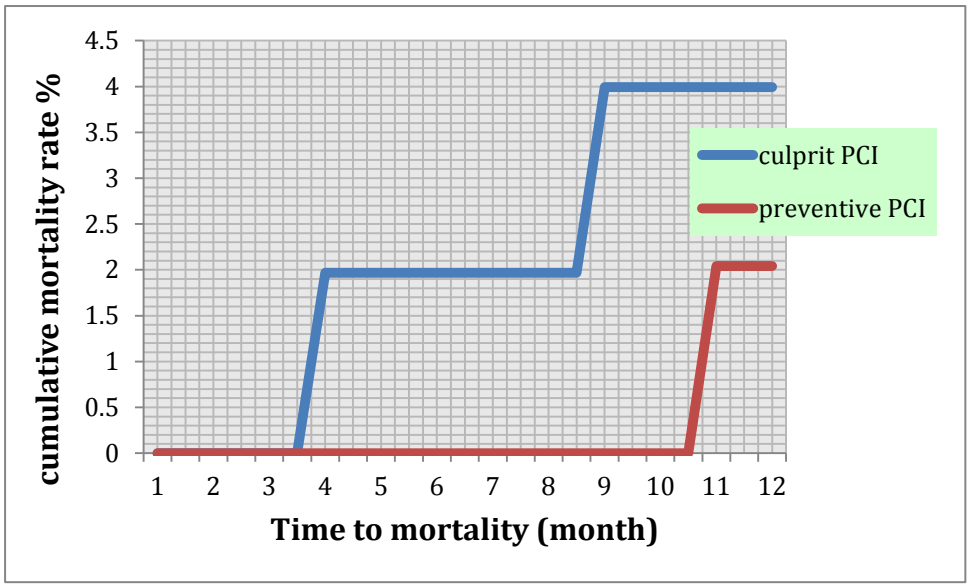

Figure (3) Kaplan Miere curve for mortality.

No definite stent thrombosis in both groups. There was no increase in recurrent MI , (STEMI; 2% vs 3.9% p=0.586, non-STEMI; 4% vs 3.9% p=0.967), HF 6.1%vs 9.8% p=0.502 and stroke 2% vs 0% p=0.31. The preventive group were significantly, less suffering from refractory angina 6.1% vs 29.4%p= 0.002 and less re-hospitalized for cardiac causes 18.4%vs 43.1% p= 0.007.

Table (5) One year outcomes of preventive PCI vs culprit artery PCI

Outcomes	**Total**=100	**Culprit artery PCI**=51 (51%)	**Preventive PCI**=49 (49%)	p value
Cardiac death	3 (3%)	2 (3.9%)	1 (2%)	0.586
STEMI	3 (3%)	2 (3.9%)	1 (2%)	0.586
Non-STEMI	4 (4%)	2 (3.9%)	2 (4%)	0.967
CHF	8 (8.0%)	5 (9.8%)	3 (6.1%)	0.502
Stroke	1 (1%)	0 (0%)	1 (2%)	0.31
Refractory angina	18 (18%)	15 (29.4%)	3 (6.1%)	0.002
Rehospitalization for cardiac causes	31(31.0%)	22 (43.1%)	9 (18.4%)	0.007
STEMI; ST segment elevation myocardial infarction. CHF; congestive heart failure. $p < 0.05$ statistical significant				

4.8. Subsequent revascularization

Thirteen percent of patients needed urgent PCI and 18% of patients underwent elective PCI or CABG for refractory angina. There were no significant differences between the groups in the need for urgent PCI 8.1% vs 17.64% p=0.161, CABG 2% vs 3.9% p= 0.586), target vessel revascularization(TVR) 10.2% vs 7.84% p=0.683, target lesion revascularization(TLR) 4% vs 3.9% p=0.967. The preventive group was significantly less received non urgent PCI 4% vs 25.49 %, p=0.0024)], non TVR 4% vs 43.1% p<0.0001 and total repeated revascularization events 18.4%vs 43.1% p=0.0013, Fig(4).

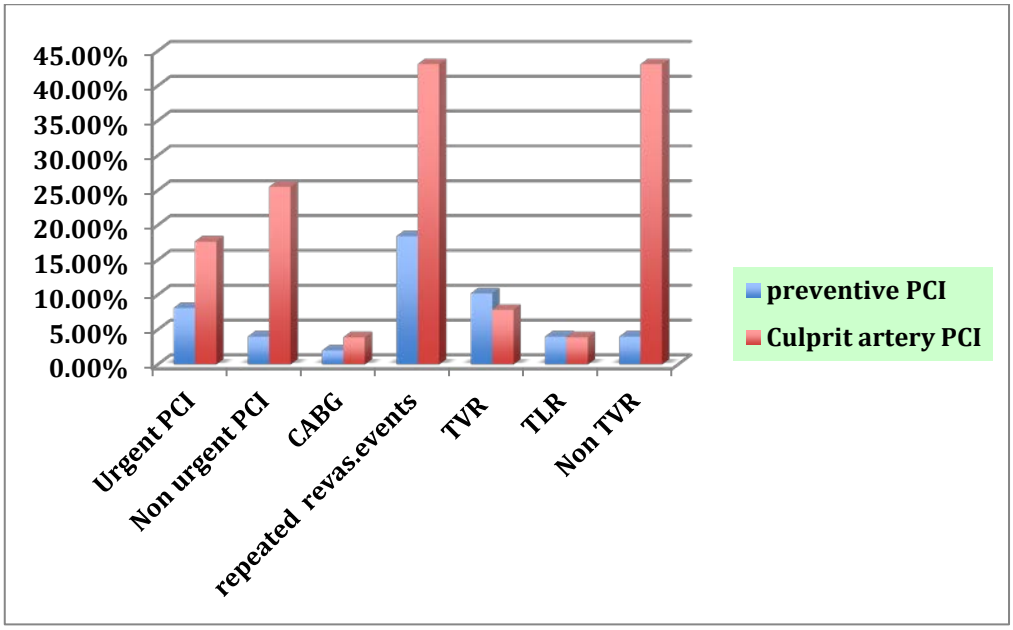

Figure (4) Comparison of repeated revascularization events between the studied groups.

4.9. Combined end points

The combined end points of death, recurrent MI, stroke, CIN, major bleeding, refractory angina and repeated revascularization were occurred in (2% vs 3.9%), (6.1%vs 7.84%), (4% vs 0%), (6.10% vs 1.9%), (6.1% vs 31.4%) and (18.4% vs 43.1%) in the preventive PCI vs culprit artery PCI respectively with total occurrence of 42.8% vs 92% p<000001.This significance drived by the significant reduction of refractory angina and repeated revascularization in the preventive PCI,Fig(5).

4.10. Study of left ventricular function

Although the LVEF% significantly improved after the procedure in both groups (p <0001), and patients with preventive policy did better, as shown on the Fig (6), yet group membership was not a significant factor (P=0.613) and a larger sample size is needed to put this finding into statistical significance.

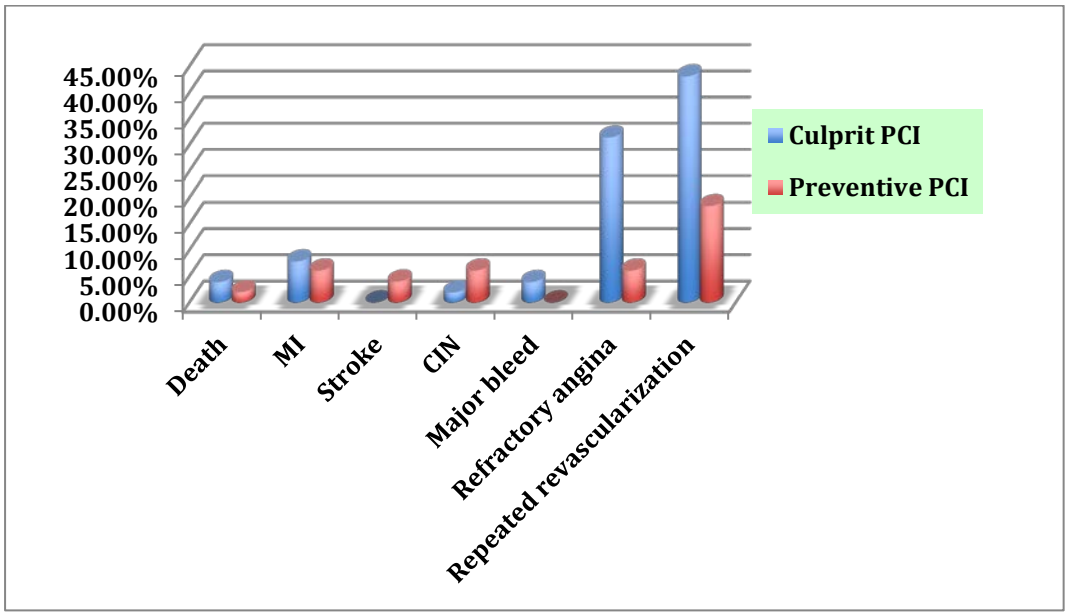

Figure (5) Comparison of the study end points between the two groups.

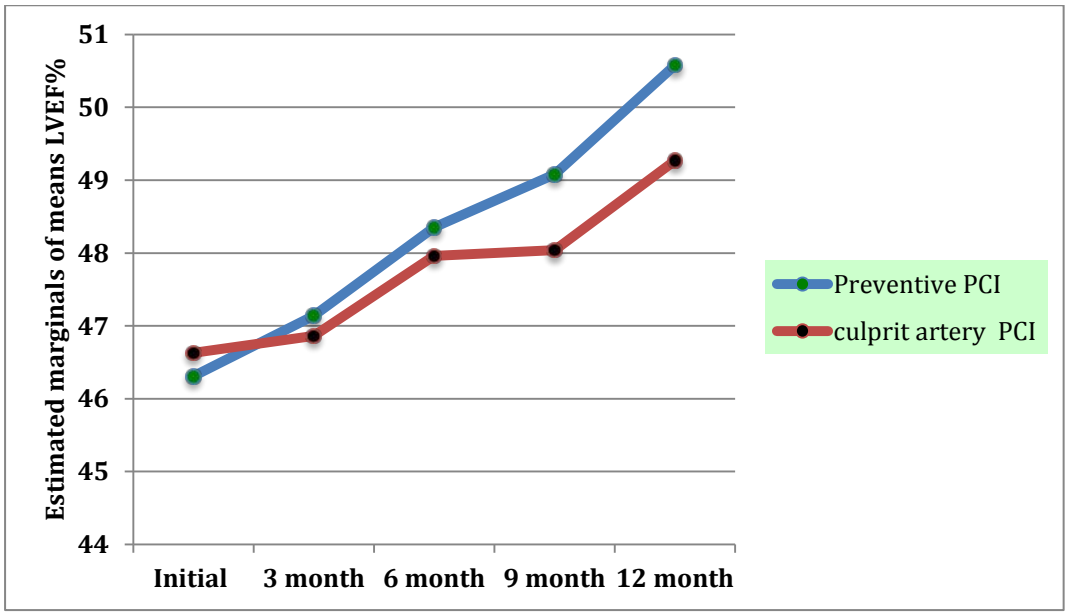

Figure (6) Study of the left ventricular ejection fraction up to 1 year post discharge.

5. Discussion

In this study, we demonstrated that preventive PCI with 2nd generation DES for STEMI and MVD , resulted in a significant reduction of repeated revascularization and refractory angina 38.8% vs 60.8% p= 0.0278 with no benefit on mortality or recurrent MI at 12 months follow up than when only the culprit artery was treated 8.1% vs 11.8% p= 0.553. Concerning periprocedural safety outcomes of stroke, major bleeding and CIN rates, we demonstrated no significant difference in patients undergoing preventive versus culprit artery PCI.(8.1% vs 5.9% p=0.658)

Previous studies examining the safety of non-IRA PCI at the time of the primary PCI procedure have shown mixed results and been heterogeneous, utilizing balloon angioplasty, bare metal stents as well as drug-eluting stents. Feng, Qarawani, and Khattab showed that multi-vessel revascularization at the time of primary PCI was not associated with increased 30-day to 1-year mortality in 225 patients.[12,13,14] In a retrospective analysis, Corpus et al.[15] showed that the 26 patients undergoing non-IRA PCI at the time of the primary PCI procedure had higher in-hospital mortality and higher MACE (repeat MI, target vessel revascularization, CABG, death) at 1-year . Four randomized trial [11,16-18] compared culprit artery to multi-vessel primary PCI.

Major adverse cardiac events, reinfarction, and repeat revascularization rates were lower with multivessel primary PCI. The HELP-AMI (Hepacoat for Culprit or Multivessel Stenting for Acute Myocardial Infarction) trial randomized 69 patients in a 3:1 ratio to MV or CVO primary PCI.[11] There was no excess in-hospital , 1-year MACE (defined as death, repeat MI, urgent PTCA, or CABG) and nonsignificant reduction of repeat revascularization 17% vs. 35% associated with multi-

vessel stenting. Politi et al.[17] randomized 214 patients to culprit artery primary PCI, multi-vessel primary PCI, or staged PCI. Again, repeat revascularization rates were lower with multi-vessel primary PCI, but there were no differences in death or reinfarction rates. The PRAMI (Preventive Angioplasty in Acute Myocardial Infarction) trial screened 1,922 patients and enrolled 465 patients at 5 sites over 5 years.[16] Recruitment was stopped prematurely by the data safety and monitoring board with a mean follow-up of 23 months due to significant differences between groups. The sample size was on the basis of an expected annual MACE rate of 20% for culprit artery primary PCI and a 30% risk reduction for multi-vessel primary PCI at 80% power. Thirteen patients did not receive assigned therapy, and 18 were lost to follow-up. The composite primary outcome of cardiac death, nonfatal reinfarction, or refractory angina occurred in 21 (9%) patients treated with multi-vessel primary PCI compared with 53 (22%) patients treated with culprit artery primary PCI (hazard ratio [HR]: 0.35; 95% CI: 0.21 to 0.58; $p < 0.001$). There were statistically significant reductions in the composite of death and myocardial infarction, and in refractory angina and repeat revascularization rates in favor of multi-vessel primary PCI.

The CvLPRIT (Complete Versus Culprit-Lesion Only Primary PCI) trial screened 850 patients and enrolled 296 patients at 7 sites over 2 years.[18] The sample size was calculated on the basis of an expected MACE rate of 37% for culprit artery primary PCI and 22% for multi-vessel PCI at 80% power. Eighteen patients crossed over, and 19 were lost to follow-up. Multi-vessel primary PCI was performed in 97 patients, and staged PCI was performed in 42 patients. The composite primary outcome of all-cause death, reinfarction, heart failure, and ischemia-driven revascularization at 12

months occurred in 15 (10%) patients with multi-vessel PCI compared with 31 (21%) patients with culprit vessel only primary PCI (HR: 0.45; 95% CI: 0.24 to 0.84; p 1⁄4 0.009). There were no statistically significant differences in death, reinfarction, heart failure, or repeat revascularization rates, although the trends favored multi-vessel PCI. It should be noted that 2 of the randomized trials that tested culprit vessel only versus multi-vessel primary PCI included patients with staged PCI. [17,18]

Although in the 2015 ACC/AHA/SCAI Focused Update on Primary Percutaneous Coronary Intervention for Patients With ST-Elevation Myocardial Infarction, multi-vessel primary PCI in hemodynamically stable patients with STEMI has been upgraded and modified to a Class IIb recommendation to include consideration of multi-vessel PCI, either at the time of primary PCI or as a planned, staged procedure. This change should not be interpreted as endorsing the routine performance of multi-vessel PCI in all patients with STEMI and multi-vessel disease. Rather, when considering the indications for and timing of multi-vessel PCI, physicians should integrate clinical data, lesion severity/complexity, and risk of contrast nephropathy to determine the optimal strategy.[19]

Future randomized clinical trials in progress evaluating the role of multi-vessel primary PCI versus culprit artery PCI in patient with STEMI and multi-vessel disease, COCUA (Complete Lesion Versus Culprit Lesion Revascularization; NCT01180218), ASSIST-MI (Revascularization Strategies for ST Elevation Myocardial Infarction Trial; NCT01818960) and CompareAcute (Comparison Between FFR Guided Revascularization Versus Conventional Strategy in Acute STEMI Patients With MVD; NCT01399736).

It is clear that further research in this area should be directed to the search criteria according to which it would be possible to choose the most effective and safe time for non culprit artery PCI in STEMI.

6. Study limitations

The present study was not a randomized controlled trial, and a selection bias may have existed. Small sample size which reduces the statistical validity of some of the differences between the groups. The criteria used by different operators to decide between culprit artery PCI and preventive PCI are not specified, which also weakens the validity of some of the results obtained. Nevertheless, safety profile and potential benefit of preventive PCI in STEMI shown in this report are worth further validation in larger multi-center randomized study.

7. Conclusion

The preventive PCI with 2nd generation DES appears to be safe as culprit artery PCI with effective reduction of refractory angina and repeated revascularization but no benefit on mortality or recurrent MI in selected hemodynamically stable patients.

Conflict of interests

No financial or nonfinancial conflicts of interests related to the subject matter or materials discussed in the manuscript.

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