

# IJMA



## INTERNATIONAL JOURNAL OF MEDICAL ARTS

*Volume 3, Issue 2 [Spring (April-June) 2021]*

10<sup>th</sup>  
المؤتمر السنوي العاشر  
COVID-19 - Lessons Learned  
كلية الطب - جامعة الأزهر بدمياط  
Virtual conference | 25 March 2021

<http://ijma.journals.ekb.eg/>

Print ISSN: 2636-4174

Online ISSN: 2682-3780

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- ✓ International Journal of Medical Arts is the Official Journal of the Damietta Faculty of Medicine, Al-Azhar University, Egypt
- ✓ It is an International, Open Access, Double-blind, Peer-reviewed Journal
- ✓ Published four times a year
- ✓ The First Issue was published in July 2019
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## Original article

# Primary Percutaneous Coronary Intervention for Acute Myocardial Infarction with ST Segment-Elevation: Effect on the Severity of Ischemic Mitral Regurgitation

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Submission date: November 02, 2020; Revision date: March 26, 2021; Acceptance date: March 27, 2021

DOI: [10.21608/ijma.2021.50324.1210](https://doi.org/10.21608/ijma.2021.50324.1210)



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## ABSTRACT

**Background:** Myocardial infarction is a daily challenging emergency. Proper diagnosis and intervention in a timely manner is crucial.

**The aim of the work:** We aimed to detect the impact of primary percutaneous coronary intervention [PPCI] on the development and severity of mitral valve regurgitation in patients presenting with acute myocardial infarction with ST-segment elevation [STEMI].

**Patients and Methods:** This was a cross-sectional comparative study that was conducted from January 2018 to November 2019 at the Department of Cardiology, Al-Azhar University Hospital, Damietta, Egypt. Patients were divided into four groups; Group I: STEMI patients with PPCI and single-vessel disease; Group II: STEMI patients with PPCI of culprit vessel and staged PCI with in-hospital admission; Group III: STEMI patients with streptokinase and pharmaco-invasive PCI of the single vessel; Group IV: STEMI patients with streptokinase and multi-vessel PCI at the same session.

**Results:** One-hundred and five patients were included in this study. Patients in group I showed a significant improvement after six months of follow-up in terms of ejection fraction [ $p < 0.001$ ]. Regarding the coaptation height, it showed a significant elevation in group I [ $p < 0.001$ ], and a significant reduction in group II [ $p < 0.001$ ] and III [ $p = 0.013$ ]. Similarly, PISA reduced significantly after six months in group I [ $p < 0.001$ ], II [ $p = 0.002$ ], and IV [ $p = 0.007$ ]. EPSS was reduced significantly in group I [ $p = 0.0004$ ]. Likewise, the distance of intrapapillary showed a significant reduction in group I [ $p < 0.001$ ]. Regarding the regurgitant area, it was significantly [ $p < 0.05$ ] reduced in all groups.

**Conclusion:** Our results showed that PPCI could decrease the incidence and the severity of IMR and improve IMR parameters, especially in patients with acute STEMI. Further well-designed longitudinal studies with a larger sample size are required to evaluate these findings.

**Keywords:** Primary PCI; STEMI; Acute Myocardial Infarction; Ischemic Mitral Regurgitation.

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**Citation:** Hammad MA, Mustafa MM, Attia MA, Reihan MS, Said EF. Primary Percutaneous Coronary Intervention for Acute Myocardial Infarction with ST Segment-Elevation: Effect on the Severity of Ischemic Mitral Regurgitation. IJMA 2021; 3 [2] April-June: 1437-1446. [DOI: [10.21608/ijma.2021.50324.1210](https://doi.org/10.21608/ijma.2021.50324.1210)].

\* Main subject and any subcategories have been classified according to the research topic.

## INTRODUCTION

The ST-segment elevation myocardial infarction [STEMI] mortality is affected by many factors, including age, Killip class, treatment delays, the way it has been treated, a history of previous myocardial infarction, diabetes mellitus, ejection fraction, renal insufficiency, and the number of affected arteries [1].

Owing to the great advances in the management of acute STEMI using the primary percutaneous coronary intervention [PPCI], reperfusion therapy, and secondary prevention treatments, several recent studies have highlighted a significant reduction in acute and long-term mortality [2-4].

Compared to in-hospital fibrinolytic therapy, randomized clinical trials showed that timely PPCI is superior in acute STEMI patients [1,5,6].

Ischemic mitral regurgitation [IMR] is a frequent coronary artery disease [CAD] complication that may be developed in the acute or chronic phase, especially due to the left ventricular [LV] remodeling [7,8]. Therefore, chronic MR is not a valve disease but rather a disease of the LV. Ameliorating heart failure symptoms and improving LV remodeling and ejection fraction are the main medical and/or surgical therapy objectives in patients with IMR [9,10].

It was reported that IMR occurs in about one-half of patients with congestive heart failure and one-fifth of those with acute myocardial infarction [AMI] [11,12].

It is very important to distinguish between functional and organic IMR. In patients with severe functional MR, the intensity of cardiac murmur is low due to the low output, but in organic MR, the cardiac murmur is significantly correlated with regurgitant volume [13,14].

Regarding the therapeutic modes of IMR, many studies showed significant benefits from medical treatment, cardiac resynchronization therapy, thrombolysis, and surgical intervention [15-17].

Moreover, the PPCI application in IMR patients demonstrated a significant reduction in the incidence and severity of IMR following acute STEMI [17]. However, data from the Middle East regarding its efficacy are scanty. The study aimed to detect the

impact of PPCI on the incidence and severity of IMR in patients presenting with acute STEMI.

## THE AIM OF THE WORK

This current work aimed to detect the impact of primary percutaneous coronary intervention [PPCI] on the development and severity of mitral valve regurgitation in patients presenting with acute myocardial infarction with ST-segment elevation [STEMI]

## PATIENTS AND METHODS

### Study Design:

This cross-sectional comparative study, which was performed at the Department of Cardiology, Al-Azhar University Hospital, Damietta, Egypt, from January 2018 to November 2019. Our patients were selected from the patients who attend our hospital with STEMI and IMR and requiring PCI. Patients were divided into four groups; Group I: patients with STEMI with PPCI and single-vessel disease [40 patients]; Group II: patients with STEMI with PPCI of culprit vessel and staged PCI with in-hospital admission [31 patients]; Group III: patients with STEMI with streptokinase and pharmaco-invasive PCI of the single vessel [18 patients]; Group IV: patients with STEMI with streptokinase and multi-vessel PCI at the same session [16 patients]. Ethical approval was obtained from the Institutional Research Board [IRB] of Al-Azhar University. Informed consent was obtained from all included patients.

### Inclusion and exclusion criteria:

We included adult patients with STEMI and IMR. On the other hand, we excluded patients with previous coronary intervention history, either PCI or CABG, patients presenting with aortic dissection, patients with valvular heart disease, and chronic systemic illness [chronic kidney disease, connective tissue disease, etc.].

### Sample size calculation:

We calculated our sample using the OpenEpi software. To fulfill a 95% confidence level and 80% power, the total required sample size was 105 patients diagnosed with STEMI and requiring PCI.

**Data collection and clinical investigations:**

All patients were subjected to complete history taking, including personal history, history of the present illness includes symptoms of IHD, past medical history, full clinical examination, general examination, and local examination. Complete blood count, serum creatinine level, qualitative troponin, INR, and random blood glucose level were collected. A 12-lead ECG for detection of site and extent of ischemia was performed.

**Echocardiography:**

Trans-thoracic Echo-Doppler study was performed according to the American Society of Echocardiography guidelines. The left ventricle was examined using 2D to assess ejection fraction [Simbison method], mechanical complication of MI, LV end diastolic and LV end systolic dimensions, and regional wall motion abnormality, LV geometry, and LV thrombus. In terms of the mitral valve [MV], it was assessed for any organic lesion of MV leaflets, MV leaflets excursion, coaptation height, tenting area [18], E-point septal separation [EPSS] [19], MV prolapse, flail leaflets [20], MV chordae, MV annulus, and the displacement of the papillary muscle.

**Mitral regurgitation:**

Vena Contracta Width [VCW], which is the narrowest proximal jet width estimated at or near the MR orifice at the tip of the leaflet, was used to determine the MR and its severity. Based on existing guidelines, the severity of MR was categorized as mild [ $<0.3$  cm], moderate [ $0.3$  to  $0.69$  cm], or severe [ $\geq 0.7$  cm] [21]. Moreover, the Proximal isovelocity surface area [PISA], a non-invasive technique for quantifying the severity of MR, was applied. Our patients were assigned to a two- or three-dimensional image with a superimposed color doppler. In terms of regurgitant area, it was measured by tracing the regurgitant colored area and is divided by the left atrium area mild [ $<20$  cm<sup>2</sup>], moderate [ $<40$  cm<sup>2</sup>], and severe [ $\geq 40$  cm<sup>2</sup>].

**Coronary intervention and follow-up:**

After obtaining written informed consent, all patients were subjected to PPCI according to the recommendations of the American Society of interventional cardiology [22]. Assessment of MR and

its severity was done pre-hospital discharge and six months later.

**Statistical analysis:**

Data were collected and coded using Microsoft Excel software. Data analysis was performed using the Statistical Package for the Social Sciences [SPSS version 22.0]. Qualitative data were represented as number and percentage, while continuous quantitative data were represented by mean  $\pm$  standard deviation [SD]. We evaluated the difference and association of qualitative variables by the Chi-square test. Differences between quantitative independent multiple groups were assessed by ANOVA test. P-value was set at  $<0.05$  for significant results.

**RESULTS**

**Demographic characteristics:** This study included 105 patients, with no significant difference between the four groups in terms of age [ $p=0.91$ ] or gender [ $p=0.65$ ]. In terms of the weight and body mass index [BMI], patients in Group I are associated with a higher mean of weight [ $p<0.001$ ] and BMI [ $p=0.002$ ] compared to other groups [Table 1].

**Baseline Clinical characteristics, vital signs, and laboratory investigations:** Patients in group I were associated with a higher prevalence of smoking [40%] and diabetes [30%]; however, the difference was not significant [ $p=0.25$  and  $p=0.14$ ], respectively. Group I, III, and IV, had the same prevalence of hypertension [50%]. Regarding the family history of IHD, it was prevalent in group IV [12.5%], compared to other groups [ $p=0.87$ ]. Heart rate and systolic blood pressure [SBP] were comparable in the study groups [ $p=0.73$  and  $p=0.12$ ], respectively. On the other hand, the respiratory rate was higher in group I compared to group III and group IV [ $p=0.034$ ]. Moreover, the diastolic blood pressure [DBP] was elevated in group I, compared to the other groups [ $p=0.005$ ]. Our findings showed a non-significant difference between all groups in terms of serum creatinine [ $p=0.11$ ], hemoglobin [ $p=0.45$ ], and random blood glucose [ $p=0.052$ ] [Table 2].

**Distribution of Affected vessels, color doppler, and direction of regurgitant jet:** Our analysis demonstrated that there was a significant difference

between the studied groups regarding affected vessels except for the posterior lateral artery [PL]. The left anterior descending artery [LAD] was the most frequently affected vessel among 37.5% of group I, 41.6% of group II, 50% of group III, and 81.3% of group IV [ $p=0.021$ ]. On the other hand, there was no significant difference between the studied groups regarding the colored doppler [ $p=0.47$ ] and the direction of the regurgitant jet [ $p=0.57$ ]. Group IV showed that 62.5% had moderate affection in colored doppler and 62.5% have central direction [Table 3].

**MV anatomy and echocardiography distribution:** There was no significant difference between studied groups regarding MV anatomy measures except for papillary muscle displacement that was significant in group I the group II than group III. Regrading echo parameters, Table [4] showed that there was no significant difference between studied groups regarding their echo parameters.

**Vital signs and Echo findings after six months follow up:** The highest mean LVEDD during follow-up was found among group II [ $p=0.003$ ], and EPSS during follow up was higher among group IV [ $p=0.016$ ]. There was no significant difference between the studied

groups in terms of other vital signs and echo findings [Table 5].

**The difference in Echo findings from baseline to six months:** We observed a significant improvement after six months in terms of EF in group I [ $p<0.001$ ], II [ $p<0.001$ ], and III [ $p<0.001$ ]. Regarding the coaptation height, it showed a significant elevation in group I [ $p<0.001$ ], and a significant reduction in group II [ $p<0.001$ ] and III [ $p=0.013$ ]. Similarly, PISA reduced significantly after six months in group I [ $p<0.001$ ], II [ $p=0.002$ ], and IV [ $p=0.007$ ]. EPSS was reduced significantly in group I [ $p=0.0004$ ]. Likewise, the distance of intrapapillary showed a significant reduction in group I [ $p<0.001$ ]. Regarding the regurgitant area, it was significantly [ $p<0.05$ ] reduced in all groups [Table 6].

**The association between affected vessels and other parameters:** Our findings indicated a significant lower ejection fraction among cases with LAD affected, compared to other vessels [ $p=0.004$ ]. On the other hand, there was no significant relation between vessels affected and auscultation findings or mortality results [Table 7].

Table [1]: Demographic characteristics of included patients

	Group I	Group II	Group III	Group IV	P-value
Age, years	59.4±10.3	60.16±9.8	61.33±10.07	60.31±8.57	0.91
Gender [Male]	24 [60%]	21 [67.7%]	12 [66.7%]	8 [50%]	0.65
Weight [Kg]	93.45±12.9 <sup>abc</sup>	79.22±9.4 <sup>a</sup>	84.9±13.18 <sup>b</sup>	82.81±15.81 <sup>c</sup>	<0.001*
Height [cm]	171.65±9.26 <sup>a</sup>	173.23±6.17 <sup>a</sup>	172.78±7.32 <sup>a</sup>	175.8±6.67 <sup>a</sup>	0.345
BMI [kg/m <sup>2</sup> ]	32.32±7.25 <sup>ab</sup>	26.57±4.31 <sup>a</sup>	28.88±6.85 <sup>c</sup>	27.17±7.43 <sup>b</sup>	0.002*

\* Statistically significant BMI; Body mass index; Similar superscripted letters denote significant differences between groups with the Post Hoc Tukey test.

Table [2]: Clinical characteristics, vital signs, and laboratory investigations

Parameters	Group I	Group II	Group III	Group IV	P-value	
<b>Clinical Characteristics</b>	Family history of IHD	3[7.5]	2[6.5]	2[11.1]	2[12.5]	0.87
	Diabetes	12[30.0]	8[25.8]	1[5.6]	2[12.5]	0.14
	Hypertension	20[50.0]	13[41.9]	9[50.0]	8[50.0]	0.90
	Smoker	16[40.0]	11[35.5]	6[33.3]	6[37.5]	0.25
<b>Vital signs</b>	Heart Rate [beat/min]	88.7±18.31	85.58±16.45	85.39±13.36	83.81±15.75	0.73
	Respiratory rate	20.83±3.61 <sup>ab</sup>	19.77±3.01	18.39±3.96 <sup>a</sup>	18.31±3.77 <sup>b</sup>	0.03*
	SBP [mmHg]	79.75±14.04 <sup>abc</sup>	72.58±9.65 <sup>a</sup>	72.78±7.52 <sup>b</sup>	70.0±3.65 <sup>c</sup>	0.005*
	DBP [mmHg]	125.5±20.62	125.16±20.96	123.89±18.5	112.5±9.31	0.122
<b>Laboratory investigations</b>	Serum creatinine	1.10±0.243	1.058±0.21	1.02±0.23	0.94±0.17	0.11
	Hemoglobin	11.85±1.05	12.19±1.22	12.28±1.49	11.88±0.96	0.45
	Random blood glucose	137.25±22.98	133.87±18.38	125.0±18.23	123.75±15.86	0.052

\* Statistically significant SBP; systolic blood pressure, DBP; diastolic blood pressure; Similar superscripted letters denote significant differences between groups with the Post Hoc Tukey test.

Table [3]: Affected vessels, Color Doppler, and direction of the regurgitant jet

Parameters		Group 1	Group 2	Group 3	Group 4	p-value
Affected vessels	LAD	15 [37.5%]	13 [41.9%]	9 [50.0%]	13 [81.3%]	0.021*
	D	5 [12.5%]	1 [3.2%]	1 [5.6%]	2 [12.5%]	0.486
	LCX	7 [17.5%]	5 [16.1%]	4 [22.2%]	8 [50.0%]	0.04*
	OM	1 [2.5%]	1 [3.2%]	1 [5.6%]	2 [12.5%]	0.432
	RCA	11 [27.5%]	8 [25.8%]	3 [16.7%]	14 [87.5%]	<0.001*
	PDA	0 [0.0]	0 [0.0]	0 [0.0]	2 [12.5%]	0.01*
	PL	1 [2.5%]	2 [6.5%]	1 [5.6%]	0 [0.0]	0.668
Color Doppler	Mild	5 [12.5%]	0 [0.0]	3 [16.7%]	2 [12.5%]	0.474
	Moderate	23 [57.5%]	23 [74.2%]	10 [55.6%]	10 [62.5%]	
	Severe	12 [30.0%]	8 [25.8%]	5 [27.8%]	4 [25.0%]	
Direction of jet	Eccentric	19 [47.5%]	16 [51.6%]	11 [61.1%]	6 [37.5%]	0.570
	Central	21 [52.5%]	15 [48.4%]	7 [38.9%]	10 [62.5%]	

\* Statistically significant; LAD: Left anterior descending, D: Diagonal, LCX: Left circumflex, OM: Obtuse marginal, RCA: Right coronary artery, PDA: Posterior descending artery, PL: Posterior lateral artery.

Table [4]: Mitral valve anatomy and Echo parameters distribution

Parameters		Group 1	Group 2	Group 3	Group 4	p-value	
Mitral valve anatomy	MVP	Normal	39 [97.5%]	31 [100%]	18 [100%]	16 [100.0%]	0.65
		Severe	1 [2.5%]	0 [0.0%]	0 [0.0]	0 [0.0]	
	Flial MV	Normal	39 [97.5%]	31[100%]	18 [100%]	16 [100%]	0.65
		Present	1 [2.5%]	0[0.0]	0 [0.0]	0 [0.0]	
	Papillary muscle displacement	No	18 [45%]	19[61.3%]	14 [77.8%]	12 [75%]	0.054
		Yes	22 [55%]	12[38.7%]	4 [22.2%]	4 [25%]	
	MV chorda	Normal	39 [97.5%]	31[100%]	18[100.0%]	16 [100%]	0.65
		Rupture	1 [2.5%]	0[0.0]	0 [0.0]	0 [0.0]	
MV Annulus	Dilated	35 [87.5%]	26[83.9%]	17 [94.4%]	15 [93.8%]	0.62	
	Normal	5 [12.5%]	5[16.1%]	1 [5.6%]	1 [6.2%]		
Echo findings	EF	43.53±6.52	42.41±5.75	46.17±6.55	42.81±5.15	0.211	
	Coaptation high	8.11±1.0	8.13±0.99	8.16±0.97	8.46±0.92	0.671	
	Tenting area	3.48±0.49	3.57±0.51	3.67±0.51	3.42±0.47	0.425	
	PISA	0.40±0.47	0.32±0.07	0.28±0.12	0.31±0.09	0.472	
	EPSS	0.975±0.23	1.03±0.21	1.04±0.21	1.13±0.13	0.122	
	MV-VC	0.53±0.18	0.52±0.14	0.51±0.15	0.50±0.15	0.957	
	Interpapillary distance	2.82±0.62	2.94±0.51	2.71±0.61	2.68±0.62	0.415	
	Area of regurgitation	32.27±10.05	33.06±6.93	34.50±11.6	32.19±10.28	0.856	

\* Statistically significant; MVP; mitral valve prolapses, MV; mitral valve, EF; ejection fraction, EPSS; E-point septal separation, PISA; Proximal isovelocity surface area

Table [5]: Vital signs and Echo findings after 6 months follow up

Parameters		Group 1	Group 2	Group 3	Group 4	p-value
Vital signs	Heart rate	73.42±7.0	71.67±6.83	71.11±8.7	76.25±8.4	0.168
	Respiratory rate	14.25±2.3	15.19±2.13	15.19±2.57	14.94±2.57	0.202
	SBP	120.85±16.7	117.1±14.8	118.33±20.6	123.7±17.46	0.595
	DBP	74.28±6.54	72.58±7.73	72.77±10.7	75.62±9.63	0.612
Echo findings	EF	49.05±7.54	47.41±8.64	50.0±10.28	44.68±8.26	0.260
	LVEDD	57.08±3.27 <sup>a</sup>	60.29±3.95 <sup>ab</sup>	55.82±7.62 <sup>bc</sup>	59.62±4.03 <sup>c</sup>	0.003*
	LVEDS	39.54±3.82	41.22±3.39	38.11±10.43	42.68±4.39	0.073
	Coaptation high	8.06±0.98	7.87±1.76	8.04±1.02	8.28±0.99	0.783
	Tenting area	2.64±0.79	2.83±0.73	3.08±0.58	3.14±0.66	0.069
	PISA	0.21±0.05	0.25±0.08	0.23±0.07	0.24±0.08	0.570
	EPSS	0.85±0.23 <sup>b</sup>	1.0±0.2	0.91±0.29 <sup>a</sup>	1.07±0.25 <sup>ab</sup>	0.016*
	MV-VC	0.35±0.09	0.41±0.12	0.41±0.13	0.42±0.13	0.612
	Interpapillary distance	2.56±0.53	2.77±0.51	2.66±0.66	2.75±0.67	0.472
Area of regurgitation	22.22±7.11	27.12±9.01	27.38±0.91	26.87±8.22	0.221	

\* Statistically significant; EF; ejection fraction, LVEDD; left ventricular end diastolic dilation, LVEDS; left ventricular end systolic dilation, EPSS; E-point septal separation, PISA; Proximal isovelocity surface area; Similar letters denote significant difference between groups within same row by Post Hoc Tukey test

Table [6]: Comparison of echo findings basal and after 6 months follow up among studied groups

Parameters		Group 1	Group 2	Group 3	Group 4
EF	Basal	44.89±5.60	42.41±5.75	46.17±6.55	42.81±5.15
	after 6 months	49.06±7.54	47.41±8.64	50.0±10.28	44.69±8.26
	P-value	p<0.001*	p<0.001*	p=0.03*	p=0.188
LVEDD	Basal	58.16±9.56	60.74±2.77	59.61±3.01	61.43±6.49
	after 6 months	58.29±3.28	60.29±3.95	55.22±7.62	59.63±4.03
	P-value	p=0.94	p=0.458	p=0.052	p=0.355
LVESDD	Basal	39.81±6.62	42.35±2.7	42.11±3.84	43.5±2.85
	after 6 months	39.54±3.82	41.23±3.39	38.12±10.43	42.69±4.39
	P-value	p=0.84	p=0.223	p=0.13	p=0.176
Coaptation high	Basal	7.82±1.67	8.13±0.99	8.16±0.97	8.46±0.92
	after 6 months	8.07±0.98	7.884±1.76	8.04±1.02	8.28±.99
	P-value	p<0.001*	p<0.001*	p=0.013*	p=0.085
Tenting area	Basal	3.50±0.51	3.57±0.51	3.67±0.51	3.42±0.47
	after 6 months	2.64±0.79	2.84±.73	3.08±.58	3.14±.66
	P-value	p=0.31	p=0.053	p=0.244	p=0.081
PISA	Basal	0.389±0.12	0.32±0.07	0.28±0.12	0.31±0.09
	after 6 months	0.214±0.05	0.25±.08	0.231±0.12	0.246±0.08
	P-value	p<0.001*	p=0.002*	p=0.132	p=0.007*
Epss	Basal	0.931±0.24	1.03±0.21	1.04±0.21	1.13±0.13
	after 6 months	0.854±0.23	1.00±.20	0.912±0.29	1.075±0.25
	P-value	p=0.0004*	p=0.50	p=0.915	p=0.341
MV_VC	Basal	0.531±0.136	0.52±0.14	0.51±0.15	0.50±0.15
	after 6 months	0.351±0.094	0.419±0.12	0.416±0.14	0.425±0.13
	P-value	p<0.001*	p=0.002*	p=0.04*	p=0.07
Inter papillary distance	Basal	2.82±0.62	2.94±0.51	2.71±0.61	2.68±0.62
	after 6 months	2.56±0.53	2.77±0.52	2.66±0.66	2.76±0.67
	P-value	p<0.001*	p=0.063	p=0.643	p=0.397
Regurgitant area	Basal	32.40±10.36	33.06±6.93	34.50±11.6	32.19±10.28
	after 6 months	22.20±7.11	27.13±9.01	27.39±9.11	26.80±8.22
	P-value	p=0.01*	p=0.007*	p=0.028*	p=0.009*

\*Statistically significant; EF; ejection fraction, LVEDD; left ventricular end diastolic dilation, LVESD; left ventricular end systolic dilatation, EPSS; E-point septal separation, PISA; Proximal isovelocity surface area

Table [7]: Relation between vessels affected and echo parameters, crepitation and mortality

		LAD	LCX	RCA	multiple	P-value
Echo findings	EF	40.484±5.64 <sup>abc</sup>	44.61±5.18 <sup>a</sup>	45.40±6.44 <sup>b</sup>	44.88±5.58 <sup>c</sup>	P=0.004*
	PISA	0.402±0.05	0.326±0.09	0.342±0.10	0.29±0.08	P=0.512
	EF Follow up	45.38±9.05	50.83±6.33	50.86±6.15	47.50±9.63	P=0.074
	PISA Follow up	0.252±0.07	0.225±0.07	0.209±0.06	0.238±0.07	P=0.573
Auscultation	Free	13 [86.7%]	6 [85.7%]	10 [90.9%]	13 [86.7%]	1.0
	Crepitation	2 [13.3%]	1 [14.3%]	1 [9.1%]	2 [13.3%]	1.0
Mortality	Survived	13 [86.7%]	5 [71.4%]	10 [90.9%]	13 [86.7%]	1.0
	Died	2 [13.3%]	2 [28.6%]	1 [9.1%]	2 [13.3%]	1.0

EF; ejection fraction, EPSS; E-point septal separation, PISA; Proximal isovelocity surface area; Similar letters denote significant difference between groups within same row by Post Hoc Tukey test

## DISCUSSION

The current treatments of IMR include conservative therapeutic management, open surgery, and percutaneous interventional therapies [23].

It has been shown that PPCI and coronary artery bypass surgery [CABG] decrease the incidence and enhance the prognosis of IMR following AMI [15,17].

In the current study, all patients had significant coronary affection of the culprit vessel. Moreover,

33.3% of them had a total coronary occlusion of the culprit vessel. LAD affection was the most frequent affection among the studied groups.

Gaber et al. [24] detected similar results by coronary angiography, where all patients had significant coronary lesions. Total coronary occlusion was found in 40%, where 40% presented with acute inferior STEMI, 30% had unstable angina, and the remaining had chronic ischemia. There were 12% with dominant LCX, 80% with dominant RCA, and 8% had co-dominant LCX and RCA. They concluded that the administration of PCI to totally occluded coronary arteries significantly improve the IMR [ $p < 0.05$ ].

Similar findings were reported by Catherine et al. [25] who reported that more severe MR is associate with more frequent multiple vessel coronary ischemia. Also, Ho [26] showed a significant improvement in patients with significant IMR and multivessel disease treated by PCI.

Pellizzon et al. [27] found that the presence of IMR is related to multiple vessel affection.

As regards to assessment of MV anatomy, papillary muscle displacement was common among the studied groups. In terms of the assessment of the severity of MV regurgitation, there was no significant difference among the studied groups as regard to capitation height, tenting aria, PISA, EPSS, MV-VC, inter papillary distance, and jet aria. Moreover, the EF was comparable in all groups.

Gaber et al. [24] reported similar findings. The mortality among the studied groups was comparable. Interestingly, in group I, five patients died; all of them had LM affection, and all of them were shocked with undetected blood pressure.

Uddin et al. [28] found that MR was a major long-term mortality predictor. In addition, it has been shown to be associated with lower EF, poor functional status, and a higher incidence of clinical shock, which could explain increased mortality with severe IMR.

In contrast to patients undergoing medical therapy alone, Trichon et al. found that improvement in IMR after revascularization was correlated with increased survival [29].

Gorman et al. have confirmed that revascularization with either PCI or CABG has been associated with enhanced survival compared to medical therapy alone in patients with IMR [30].

After six months of PPCI, our findings showed a significant improvement after six months in terms of EF in group I [ $p < 0.001$ ], II [ $p < 0.001$ ], and III [ $p < 0.001$ ]. it was also reported that reperfusion of totally occluded coronary artery with stents resulted in similar improvement in LVEF and less LV dilation. Regarding the coaptation height, it showed a significant elevation in group I [ $p < 0.001$ ], and a significant reduction in group II [ $p < 0.001$ ] and III [ $p = 0.013$ ]. Similarly, PISA and EPSS showed a significant improvement after six months of PPCI.

Our findings are in line with the study conducted by Chua et al. [31], who stated that PCI was associated with a reduction in the incidence of IMR compared to a therapeutically controlled group for the first acute STEMI. Early, rapid PCI coronary revascularization was closely correlated with a substantial reduction in risk for moderate or severe IMR.

Dzavik et al. concluded that a significant improvement in regional and global LV function, especially in patients with recent occlusions and depressed left ventricular function, is correlated with the restoration of coronary patentability of completely occluded coronary arteries [32].

Similarly, Booher et al. found that the incidence of IMR was substantially reduced in patients with good reperfusion with PCI [33].

El-Akabawy et al. reported a 31% improvement in the degree of IMR in patients who underwent PCI. However, 61% showed no improvement in the degree of IMR, and only 7% of patients showed deterioration of the degree of IMR [34].

The importance of the LV structure for normal mitral leaflet coaptation has been highlighted. The remodeling of the ischemic LV, and its expansion and assumption of a more spherical shape, results in an altered axis of contraction of the papillary muscle and apical tethering of the mitral leaflets. Likewise, the decreased force of leaflet closure in the setting of the disordered structure of the mitral apparatus was

observed in many patients with LV dysfunction [34].

Additionally, important predictors of improved IMR after PCI were LVEDD and EPSS follow-up, which support that the improvement of IMR occurs secondary to the LV function improvement [26].

Lastly, our results come in line with previous studies, as Paul et al. concluding that the PCI appears an acceptable first treatment option for patients with concomitant severe multivessel coronary artery disease and significant IMR, as they concluded that there was a significant improvement in IMR in all of the treated patients irrespective of the procedure performed either PCI or CABG and concluded that the PCI appears to be the first choice for patients with concomitant severe multivessel coronary artery disease and significant IMR [35].

Hickey et al. reported that PCI resulted in a significant reduction in the severity of IMR in 63 patients, and that was demonstrated in patients with successful reperfusion [36].

Chua et al. [31] demonstrated that the PCI is more effective than non-PCI in reducing the risk of moderate or severe IMR after MI.

The main limitations of our study are the cross-sectional sample type raises the incidence of confounders and the short follow-up period of the patients six months only. As with any prospective, single-center study, the current study may be limited by the selection bias, and the results could not be generalized. Therefore, it is recommended to carry a series of later on studies to confirm our results with a large sample size with a long follow-up period.

In conclusion, our results showed that PPCI could decrease the incidence and the severity of IMR and improve IMR parameters, especially in patients with acute STEMI. Further well-designed longitudinal studies with a larger sample size are required to evaluate these findings.

#### **Financial and Non-Financial Relationships and Activities of Interest**

None

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