Primary PCI in Patients Admitted with Cardiogenic Shock and STEMI: Outcome and Predictors of In-hospital Mortality

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Abstract: *Background*: It is a well-known phenomenon that cardiogenic shock (CS) is a serious complication of acute myocardial infarction. The mortality rate is approximately 50% even with rapid revascularization, optimal medical care, and use of mechanical support. *Aim of the Work*: To investigate the outcome of primary percutaneous coronary intervention (PCI) in patients admitted with cardiogenic shock and ST-segment elevation myocardial infarction (STEMI) and the predictors of in-hospital mortality. *Patients and Methods*: This prospective, observational study was conducted in the national heart Institute, Alazhar University, and Military hospitals in the period from 6/2019 to 9/2021 on fifty six consecutive patients presenting to Alazhar University hospitals, National Heart Institute (NHI), Military hospitals. *Results*: The prevalence of dyslipidemia and diabetes mellitus were significantly higher among died patients than those who survived. The degree of LV impairment was significantly higher among patients who died than those who survived. As regard to PCI procedure characteristics, TIMI flow post PCI (<Grade III), multi-vessel disease, and procedural failure were significantly associated with high in-hospital mortality rate. Patients who received treatment with IABP were significantly older. *Conclusion*: Multi-vessel coronary artery disease, TIMI flow after PCI (grade III), and ↑ CK-MB (72-hour serial measurement), were all found to be significant predictors of in-hospital mortality. The onset from chest pain to ED arrival and the door-to-balloon time were higher than that reported in the previous studies. The use of IABP was not found to have a significant predictor effect on the different outcome among our patients with STEMI.

Keywords: PPCI, Cardiogenic Shock, ST-elevation, Myocardial Infarction, In-hospital Mortality

1. Introduction

Atherosclerosis is a chronic disease with severe cardiovascular symptoms. A local artery occlusion with a thrombus covering a pre-existing atherosclerotic plaque is the most common cause of acute athero-sclerosis symptoms. Despite significant breakthroughs in the prevention and treatment of cardiovascular disease, it remains the major cause of morbidity and mortality worldwide, accounting for 35% of all deaths in the US and 30% of all deaths worldwide in 2005 [1].

Acute myocardial infarction is a common cause of cardiogenic shock, which is defined by inadequate tissue perfusion due to cardiac failure [2]. The incidence of cardiogenic shock (CS) in patients with acute myocardial infarction (AMI) varies depending on the definition of CS, although it is estimated to be between 5 and 15%, with a recent decrease [3]. There are a number of clinical consequences linked to the development of AMI, but none are more dangerous or have a poorer prognosis than CS [4].

Mortality of patients with AMI was reduced from 30% to

5% for non-CS patients during the last decades but in the subgroup of patients with CS, improvements were much less extensive. Despite advances in treatment during the last two decades leading to a steady reduction in mortality rates, CS remains to be the leading cause of death with hospital mortality rates still approaching 50% [5].

Primary percutaneous coronary interventions (PCI) are the preferred treatment for ST-segment elevation myocardial infarction (STEMI) and are effective in opening the infarct-related artery [6]. The exact relationship between primary percutaneous coronary interventions (PCI) volume and mortality remains unclear. No data are available on how this relationship could be affected by time-to-presentation [7].

Retrospective study suggested that early PCI may improve outcome in patients with cardiogenic shock. The randomized SHOCK trial showed that strategy of early revascularization with surgery or PCI increase one-year survival from 34 to 47% (p=0.025) compared to initial aggressive medical therapy in patients with shock complicating acute myocardial infarction [8].

This study aims at investigating the outcome of primary percutaneous coronary intervention (PCI) in patients admitted with cardiogenic shock and ST-segment elevation myocardial infarction (STEMI) and the predictors of in-hospital mortality.

2. Patients and Methods

2.1. Type of the Study

This sudy was carried out as a prospective descriptive study on fifty six consecutive patients presenting to alazhar univeristy hospitals, National Heart Institute (NHI), Military hospitals, and other hospitals in the period from 6/2019 to 6/2021 with acute ST segment elevation myocardial infarction (STEMI) complicated with cardiogenic shock and were managed by primary PCI with or without IABP support.

2.2. Inclusion Criteria

All patients with the diagnosis of STEMI and cardiogenic shock had primary PCI within the first 12 hours after the onset of symptoms (within the first 18 hours after the onset of symptoms for patients with hemodynamic instability and persistent chest pain).

Acute ST segment elevation myocardial infarction was detected by rise and /or fall in cardiac biomarkers (preferred troponin) with at least one value above 99th percentile of the upper reference limit with at least one of the following [9]: symptoms of ischemia, ECG changes of new ischemia (ST elevation or LBBB), development of pathological Q waves and imaging evidence of new loss of viable myocardium.

2.3. Exclusion Criteria

Patients with STEMI and received thrombolysis before PCI, patients with non-STEMI, unstable or stable angina. Patients with STEMI with no cardiogenic shock, patients with cardiogenic shock due to (congestive heart failure due to other causes rather than acute MI, Cardiomyopathy, dysrhythmias, cardiac tamponade, Severe valvular dysfunction, acute pulmonary embolism, tension pneumothorax, Papillary muscle rupture and Ventricular septal rupture as mechanical complications to acute MI, aortic dissection, myocarditis, endocarditis, drug overdose, Cardiac or chest trauma).

2.4. Study Protocol

All patients were subjected for the following:

Full history taking with emphasis on: personal data (age and sex), history of cardiovascular disease, risk factors (smoking, dyslipidaemia, diabetes mellitus, hypertension, and +ve family history), previous history of (CAD, PCI or CABG) and onset of symptoms of STEMI and cardiogenic shock.

Complete clinical examination with emphasis on: General examination as regard vital signs (arterial blood pressure, pulse, temperature, and respiratory rate). Local cardiac examination (S3 gallop, bilateral basal crepitations, elevated Jugular venous pressure, Hemodynamic instability and others).

Type of STEMI was diagnosed from the electrocardiogram upon his admission as follows: ST segment elevation and the leads affected. Rhythm presentation (sinus, Atrial fibrillation, Ventricular arrhythmias or Asystole). Conduction disturbances: LBBB whether old, new or unknown, RBBB whether old, new or unknown and first, second or third degree A-V block.

Laboratory work up: Daily blood sample results (cardiac enzymes=CK-MB and troponin) of all the patients on admission and thereafter were recorded. Blood glucose level (random samples) at admission. Kidney function tests (serum creatinine level) at admission and then daily for the first 72 hours. Lipid profile including (Total cholesterol, low density lipoprotein (LDL), high density lipoprotein (HDL), and triglycerides).

In hospital management: All patients received aspirin (300 mg loading then 150 mg daily), Un-fractionated heparin (UFH) (70 IU/ kg) & clopidogrel (600mg as a loading dose and 75mg once daily as a maintenance dose) in addition to conventional treatment (Beta- blocker, ACEI, and statin). V asopressors were used to set a systolic blood pressure>90mmHg and were mentioned.

Coronary angiography: Informed written consent was obtained for all patients.

Pre-treatment: All patients were given an oral loading dose of Clopidogrel 600 mg and 300 mg chewable aspirin are also given before the intervention.

Anti-coagulation with UFH was routinely given (80-100IU/kg).

Glycoprotein IIb/IIIa receptor antagonists may be given at the operator's discretion.

Sterilization & local infiltration of anaesthesia of the right groin was routinely undertaken.

Right femoral artery puncture using Seldinger's technique was done.

Selective left and right coronary angiographies in multiple views starting with the non-infarct related artery.

PTCA was optionally done using a suitable balloon (usually under-sized).

A thrombus aspiration catheter was utilized as indicated in case of: The presence of a heavy thrombus burden or Absence of flow after the passage of the guiding wire.

A stent suitable in diameter and length was inserted according to the angiographic findings in each case.

And the angiographic findings as TIMI flow grade after PCI, left main disease, number of diseased coronary vessels were analysed. The door-to-balloon time and pain-to-ED were obtained from the hospital records.

Patients were then admitted to CCU.

The sheath removal was undertaken after normalization of the ACT or APTT.

TIMI flow before and after PCI was evaluated as follows: Grade 0: (no perfusion): There is no ante-grade flow beyond the point of occlusion. Grade 1: (penetration without perfusion): The contrast material passes beyond the area of obstruction but "hangs up" and fails to opacify the entire coronary bed distal to the obstruction for the duration of the cineangiographic filming sequence.

Grade 2: (partial perfusion): The contrast material passes across the obstruction and opacifies the coronary bed distal to the obstruction. However, the rate of entry of contrast material into the vessel distal to the obstruction or its rate of clearance from the distal bed (or both) is perceptibly slower than its entry into or clearance from comparable areas not perfused by the previously occluded vessel.

Grade 3: (complete perfusion): Ante-grade flow into the bed distal to the obstruction occurs as promptly as ante-grade flow into the bed from the involved bed and is as rapid as clearance from an uninvolved bed in the same vessel or the opposite artery [10].

The Door-to-Balloon time:

was defined as the time lapse between hospital visit and post-balloon angioplasty coronary reperfusion.

Mechanical support with Intra-Aortic Balloon Pump (IABP): was used in indicated patients if needed and available according to the clinical status of the patient.

Transthorthic echocardiography was done before and after PCI (within 30 days and after 3 months) stressing on:

Left ventricular ejection fraction (Normal, fair, mild, moderate or severe) and Wall motion score index (WMSI).

Using the commercially available equipment, the digital ultrasound system with a 2- to 3 MHz transducer.

M-mode, two dimensional & Doppler echocardiographic assessment was performed for all patients.

Examinations were done with the patient in left semi-lateral position; utilizing left parasternal long, short axis views apical 4 chambers, apical 2 chambers and apical 5 champers.

The left ventricular dimensions (end-systolic and end-diastolic) were determined from parasternal M-mode acquisitions. The LVEF% was calculated from the conventional apical 2- and 4-chamber images) using the following formula:

$$EF\% = (EDV - ESV) / EDV \times 100 = SV / EDV \times 100$$

Where SV is stroke volume.

LV systolic dysfunction was defined as LVEF% less than 55% [11].

Regional wall motion was assessed by 2D-echocardiography, and assessed in terms of wall motion scoring index based on 17 segments approach recommended by the American-Society of Echocardiography [12].

Clinical in hospital follow up of MACE within 30 days and after 3 months for the following:

Cardiovascular mortality: defined as unexpected sudden death or death related to acute MI, heart failure, or arrhythmia.

Morbidity:

Hospital re-admission as for (major arrhythmias, heart failure or others).

Re-infarction after PCI: The term 're-infarction' is used for an acute MI that occurs within 28 days of an incident or recurrent MI. The ECG diagnosis of suspected reinfarction following the initial MI may be confounded by the initial evolutionary ECG changes [13].

Re-infarction should be considered by the following:

ST elevation ≥ 0.1 mv or new pathognomonic Q waves appeared, in at least two contiguous leads, particularly when associated with ischaemic symptoms for 20 min or longer. An immediate measurement of cTn is recommended. A second sample should be obtained 3–6 h later with ≥ 20% increase of the cTn value in the second sample.

CK-MB (or CK, if MB is not available) > 3 times the upper limit of normal and ≥ 50% greater than the previous value [14].

TVR (target vessel revascularization):

Defined as repeated PCI or CABG due to stenosis or occlusion in the IRA.

Bleeding according to TIMI scale of bleeding:

Major bleeding leading to haemodynamic compromise requiring intervention (e.g. blood or fluid replacement, inotropic support, ventricular assist device, surgical repair) or life-threatening or fatal bleeds e.g. (Intracranial bleeding, Gastrointestinal bleeding, Genitourinary bleeding, or Decrease of haemoglobin concentration by more than 5 gm/dl).

Moderate bleeding requiring transfusion of blood but which did not lead to haemodynamic compromise requiring intervention.

Minor bleeding neither requiring blood transfusion nor leading to haemodynamic compromise e.g. (Vascular puncture site or Decrease of haemoglobin concentration by 3-5 gm/dl) [15].

Contrast induced nephropathy: Acute renal impairment after PCI (caused, at least in part, by radiographic contrast material) occurs in up to 2% of patients. Contrast-induced nephropathy (CIN) is defined as the impairment of renal function and is measured as either a 25% increase in serum creatinine (SCr) from baseline or 0.5 mg/dL (44 μmol/L)

increase in absolute value, within 48-72 hours of intravenous contrast administration [16].

3. Statistical Analysis

Data were analysed using the SPSS programme and the statistical package for social science. Descriptive statistics (for quantitative data) were used to describe the demographical and pathophysiological correlated data (risk factors for mortality). Data were grouped as discrete and continuous variables:

Discrete variables: were presented in the form of frequency and percentage tables. Inferences were done using the Chi-square test of significance to evaluate the differences between categorical variables.

Probability value (P-value) or the calculated probability is the estimated probability of rejecting the null hypothesis of a study question when that hypothesis is true. The choice of significance level at which the null hypothesis is rejected is arbitrary. P-value<0.05 was considered significant,<0.01 was considered as highly significant and>0.05 was considered an insignificant [17].

Continuous variables: were presented in the form of mean and standard deviation. The Paired student t-test was used to compare numerical values between the groups. ANOVA tables were used to determine the level of significance between multiple variables. Spearman's correlation coefficient was used to estimate different correlations between variables.

ROC (Receiver Operator Characteristic) curve for the best cutoff for mortality.

Prospective multivariate stepwise Cox regression analysis was used to investigate the independent predictors of in-hospital mortality.

Ethical considerations.

All the steps of the study were explained to the participants with its possible complications stressing on the importance of data they were going to offer. Written informed consent was taken from patients shared in the study; this meant that the participants in the prospective study were fully informed about the procedures and risks involved in the study. All data and results of the study of the participants were confidential and were not being made available to anyone who was not directly involved in the study. The patients had the right to refuse participation without affecting the medical care expected to be offered to the patients.

4. Results

The total number of patients included in the study was 56 patients, they were 37 males (66.1%) and 19 females (33.9%). Their age ranged between 39-75 years with a mean age of 59.46±8.41.

Table (1) shows that 36 patients (64.3%) were smokers, 39 patients (69.6%) had dyslipidemia, 37 patients (66.1%) had DM, 42 patients (75%) had hypertension, 30 patients (53.6%) had +ve family history of CAD, 55 patients (98.2%) had prior CAD, one patient (1.8%) had previous PCI and 4 patients (7.1%) had prior CABG.

Table 1. Distribution of patients according to risk factors (N=56).

	Frequency	%
Smoking	36	64.3%
Dyslipidemia	39	69.6%
DM	37	66.1%
HTN	42	75.0%
+ve Family History of CAD	30	53.6%
Prior CAD	55	98.2%
Prior PCI	1	1.8%
Prior CABG	4	7.1%

All patients included in the study (n=56 patients) had chest pain, dyspnea, fatigue, tachycardia (except for patients who had inferior MI), Killip class IV, increased jugular venous pressure and clinical signs of hypoperfusion (altered mental status, pallor, cold extremities and faint peripheral pulsation) with haemodynamic instability.

On admission 19 patients (33.9%) had anterior wall MI, 6 patients (10.7%) had lateral MI, 16 patients (28.6%) had extensive anterior MI, 2 patients (3.6%) had infero-lateral MI, 4 patients (7.1%) had antero-inferior MI, 2 patients (3.6%) had posterior wall MI, and 7 patients (12.5%) had inferior wall MI,. 42 patients (75%) had moderate impairment of LV function (mean±SD=36.38±3.18), while 14 patients (25%) had severe impairment (mean±SD=27.71±1.59). Mean±SD WMSI was 1.75±0.24 with range 1.29-2.29 as shown in (Table 2).

Table 2. Evaluation of cardiac impairment on-admission (N=56).

		Frequency	%
	Anterior MI	19	33.9%
	Lateral MI	6	10.7%
	Extensive anterior MI	16	28.6%
Type of STEMI	Infero-lateral MI	2	3.6%
	Antero-inferior MI	4	7.1%
	Posterior MI	2	3.6%
	Inferior MI	7	12.5%
LVEF% on admission Range (mean±SD)	Moderate LVEF% impairment 30-40% (36.38±3.18)	42	75%
	Severe LVEF% impairment 25-29% (27.71±1.59)	14	25%
WMSI on admission– Range (mean±SD)		1.29-2.29 (1.75±0.24)	

There was no significant difference between patients who died at hospital (n=25) and those who survived (n=31) regarding prevalence of smoking, hypertension, +ve family history for CAD, prior CAD, prior PCI and prior CABG (P>0.05). The prevalence of dyslipidemia was significantly higher among died patients (n=21, 84%) than those who survived (n=18, 58.1%) (P=0.036). Moreover, the prevalence of DM was significantly higher among died patients (n=23, 92%) than those who survived (n=14, 42.2%) (P=<0.001) as shown in (Table 3).

Table 3. *Relation between in-hospital mortality & cardiovascular risk factors.*

	In-hospital mortality (*n*=25)		No In-hospital mortality (*n*=31)		p-value
	No.	%	No.	%	
Smoking	16	64%	20	64.5%	0.968
Dyslipidemia	21	84%	18	58.1%	0.036
DM	23	92%	14	42.2%	<0.001
HTN	20	80%	22	71%	0.438
+ve Family History for CAD	14	56%	16	51.6%	0.743
Prior CAD	25	100%	30	96.8%	0.365
Prior PCI	1	4%	0	0.0%	0.261
Prior CABG	2	8%	2	6.5%	0.823

Table 4 shows that the patients who received treatment with IABP were significantly older (mean age±SD for those who had IABP use 61.2±11.1 vs 54.3±9.4 for those who did not have IABP use) (P=0.012). The prevalence of male sex was not significant among those two groups (P>0.05). as regard to Admission clinical characteristics, cardiovascular risk factors and laboratory findings; there was no significant difference between patients with IABP placement and those without (P>0.05) expect for patients with prior CABG; as there was a significant difference among those two groups (p=0.031).

Table 4. *Comparison between the use of IABP regarding the different demographic, clinical characteristics, cardiovascular risk factors and laboratory findings.*

Parameter	Without IABP (*n*=29)	With IABP (*n*=27)	P value
Demographic characteristics			
Age (mean±SD)	54.3±9.4	61.2±11.1	0.012
Male sex (n, %)	19 (65.5%)	18 (66.7%)	0.139
Admission clinical characteristics			
Basal HR (b/min.)	109.4±24.99	105.4±24.96	0.551
Systolic BP (mm Hg)	81±3.1	80.2±3.5	0.342
Diastolic BP (mm Hg)	52.4±4.7	50.9±4.6	0.240
Cardiovascular risk factors			
Smoking (n, %)	20 (69%)	16 (59.3%)	0.574
Dyslipidemia (n, %)	19 (65.5%)	20 (74.1%)	0.484
DM (n, %)	19 (65.5%)	18 (66.7%)	0.928
HTN (n, %)	20 (69%)	22 (81.5%)	0.280
+ve Family History for CAD (n, %)	17 (58.6%)	13 (48.1%)	0.432
Prior CAD (n, %)	28 (96.6%)	27 (100%)	0.330
Prior PCI (n, %)	1 (3.4%)	0	0.330
Prior CABG (n, %)	0	4 (14.8%)	0.031
Laboratory findings			
↑ CK-MB (72-hour serial measurement)	15 (51.7%)	15 (55.6%)	0.774
↑ S. Creatinine (72-hour serial measurement)	14 (48.3%)	19 (70.4%)	0.093

The degree of LV impairment was significantly higher among patients with IABP placement than those without (P<0.001). Furthermore, mean±SD LVEF% was significantly lower among patients with IABP use (31.85±4.19) than those without (36.97±4.7). In addition mean±SD WMSI was significantly high (1.86±0.25) among patients with IABP use than those without (1.66±0.18) (p=0.002) as shown in (Table 5).

Table 5. *Comparison between the use of IABP regarding the cardiac impairment on admission and PCI procedure characteristics.*

Parameter		Without IABP (*n*=29)	With IABP (*n*=27)	P value
cardiac impairment on admission				
LVEF% (mean±SD)		36.97±4.7	31.85±4.19	<0.001
WMSI (mean±SD)		1.66±0.18	1.86±0.25	0.002
PCI procedure characteristics				
Tirofiban use		15 (51.7%)	17 (63%)	0.396
LM disease		7 (24.1%)	8 (29.6%)	0.643
No. of diseased vessels (>one vessel)		2 (6.9%)	2 (7.4%)	0.969
	LAD	22 (75.9%)	17 (63%)	
Infarct-related artery (n, %)	LCX	2 (6.9%)	7 (25.9%)	0.146
	RCA	5 (17.2%)	3 (11.1%)	
Total revascularization		14 (48.3%)	10 (37%)	0.396

Parameter		Without IABP (*n*=29)	With IABP (*n*=27)	P value
Multi vessel PCI		12 (41.4%)	8 (29.6%)	0.359
TIMI flow before PCI (n, %)	Grade O	25 (86.2%)	20 (74.1%)	0.253
	Grade I	4 (13.8%)	7 (25.9%)	
TIMI flow after PCI (n, %)	Grade I	1 (3.4%)	4 (14.8%)	
	Grade II	12 (41.4%)	11 (40.7%)	0.309
	Grade III	16 (55.2%)	12 (44.4%)	
Procedure success (n, %)		28 (96.6%)	23 (85.2%)	0.163
PCI duration (mean±SD) (min.)		63.45±18.52	64.07±19.01	0.901
Contrast volume (mean±SD) (ml.)		260.35±57.29	255.56±59.38	0.760

Case:-no. 1

History:

A 68 years old man, carpenter who is known to be hypertensive, diabetic, smoker and dyslipidemic, he presented to the emergency room with an acute attack of retrosternal chest pain that started 12 hours before presentation with dyspnea at rest, nausea, vomiting and sweating.

ECG:

He had an ECG in the emergency department that showed ST segment elevation in leads V1-V6. That confirmed the diagnosis of extensive anterior MI (Figure 1).
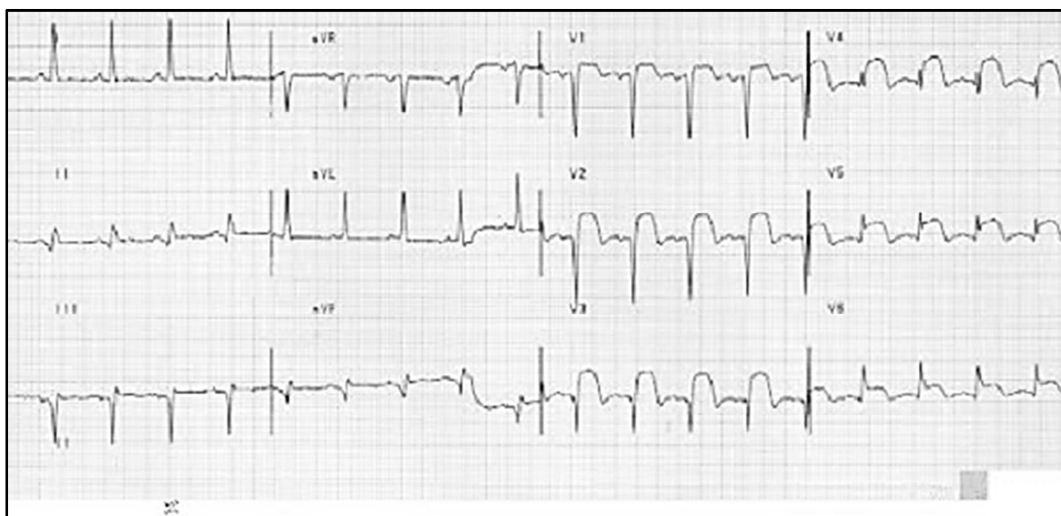

Figure 1. Emergency department ECG showing ST segment elevation in leads V1-V6.

The patient had criteria of cardiogenic shock and received inotropes to set systolic BP above 90 mmHg and received 300 mg aspirin, 600 mg clopidogrel then the patient was referred to the catheter lab for primary PCI (Door- to-Balloon was 120 min). And was ventilated before moving to the catheter lab.

Cardiac enzymes.

The troponin was positive.

CK-MB was 59 U/ml (normal level up to 25 U/ml) which increased after primary PCI.

Echocardiography.

Echocardiography had done at admission that revealed Left ventricular ejection fraction of 25%. Resting segmental wall motion abnormality in the form of mid, basal, apical inferior wall hypokinesia, mid lateral wall hypokinesia and akinetic apical cap with mild mitral regurgitation and WMSI of 1.94.

Primary PCI procedure.

The procedure was done through the right femoral access using Seldinger technique, left XP 3.5", 6 French guiding catheter. Coronary angiography showed that the Left main coronary artery was a long vessel that was totally occluded at its end A BMW 0.014 inch PCI guide wire was advanced till the distal LAD, and then a 2x20 sprinter balloon was inflated up to 8 ATM then a 3x20 mm DES was deployed at 14 ATM in the site of the total occlusion, with end result of TIMI I flow after giving of loading dose of Tirofiban and intracoronary adrenaline. The patient was admitted to the CCU where he received medical treatment including inotropes, maintenance dose of Tirofiban.
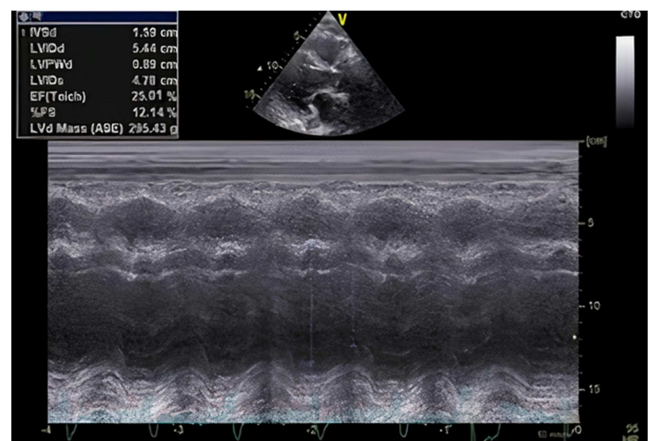

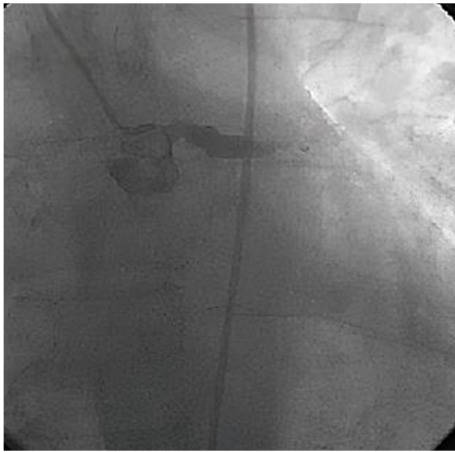*Figure 2. Parasternal long axis view, M-mode echo of the left ventricle showing severe LVEF% impairment.*

Figure 3. Coronary angiography of the left coronary vessels, PA caudal projection was showing total occlusion of LM at its end.
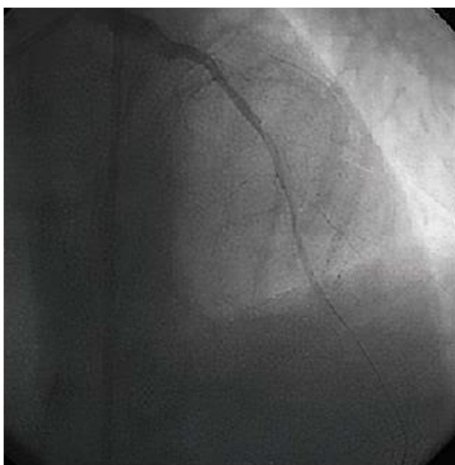

Figure 4. Final result after stent deployment with TIMI I flow.

Follow up:

The patient stayed in the CCU for one day then died.

Figure 5. Parasternal long axis view, M-mode echo of the left ventricle showing mild LVEF% impairment.

5. Discussion

It is a well-known phenomenon that coronary reperfusion can be established rapidly and efficiently through primary PCI in STEMI patients. Coronary complex lesions which require more complicate interventions are more common in patients with acute STEMI and cardiogenic shock. Hemodynamic instability in these patients is one of the main drawbacks for an effective coronary reperfusion after primary PCI in these patients. As a result, the success rate has been reported to be significantly lower in patients with cardiogenic shock who underwent primary PCI, compared to those without cardiogenic shock.

Tarantini et al., reported that the success rate of reperfusion was 53% in patients with cardiogenic shock who underwent primary PCI, while *Giri et al.* reported a success rate of 71%. In our study the success rate of reperfusion after primary PCI was 55.4%.

Our study is a clinical prospective trial that was conducted in the National Heart Institute, Alazhar University, military hospitals in order to investigate the efficacy of primary PCI with and without the use of IABP for treatment of patients with ST-elevation myocardial infarction and cardiogenic shock (CS).

We enrolled 56 patients, 37 males (66.1%) and 19 females (33.9%). Their age ranged between 39 – 75 years with a mean age of 59.46±8.41. 36 patients (64.3%) were smokers, 39 patients (69.6%) had dyslipidemia, 37 patients (66.1%) had DM, 42 patients (75%) had hypertension, 30 patients (53.6%) had +ve family history of CAD, 55 patients (98.2%) had prior CAD, one patient (1.8%) had previous PCI and 4 patients (7.1%) had prior CABG.

We are in discordance with the study of *John et al.,* in the SHOCK Trial. The independent correlate of mortality was increasing age (p<0.001). Conversely with the study of *Zeymer et al.,*. Who found older age was an independent predictor of in-hospital mortality in patients>75 years in the ALKK (Arbeitsgemeinschaft Leitende Kardiologische Krankenhausärzte) European PCI registry.

Unlike the previous studies of *John et al.,* [8], *Zeymer et al., Klein et al., Mehmet et al., Ayaz et al., Francesco et al.,* and *Etienne et al.,* that exhibited older age was an independent predictor of in-hospital mortality. Our results revealed that there was no significant difference between patients who died and those who survived regarding their mean age (P>0.05) and this may be Attributed to high prevalence of risk factors in young age population.

We did not find significant difference among patients who died and those who survived regarding their sex distribution. Moreover there was no significance difference regarding the prevalence of male sex among patients who received treatment with IABP and those who did not have IABP use (P>0.05). Also we found that gender difference was not significantly associated with high in-hospital mortality rate (P>0.05) when included in the univariate analysis of independent predictors of in-hospital mortality.

We did not find significant difference between patients who died and those who survived regarding the prevalence of smoking, hypertension, +ve family history of CAD, prior CAD, prior PCI and prior CABG (P>0.05). The prevalence

of dyslipidemia and diabetes mellitus were significantly higher among died patients [n=21 (84%) and 23 (92%) respectively] than those who survived [n=18, (58.1%) and 14 (42.2%) respectively] [P=0.036 and<0.001 respectively].

Among the different demographic characteristics, cardiac risk factors and laboratory findings analyzed; univariate analysis indicated that dyslipidemia (hazard ratio=0.304, 95% confidence interval 0.085-1.084, P=0.046), DM (hazard ratio=0.127, 95% confidence interval 0.028-0.580, P=0.008), hypertension (hazard ratio=0.289, 95% confidence interval 0.093-0896, P=0.032) were significantly associated with high in-hospital mortality rate. We performed a logistic regression to ascertain the predictors of in-hospital mortality. Dyslipidemia, DM and Hypertension were the independent predictors of in-hospital mortality. Furthermore, Survival analysis and Kaplan Meier survival curve showed that dyslipidemia and DM were associated with lower survival time expectancy.

The onset from chest pain to ED arrival and STEMI diagnosis in our study were higher than previous observational studies, this may be attributed to some factors like; late arrival of patients to the hospital due to crowded and unorganized traffic, delayed patients preparation and transportation to the catheter laboratory with delays in setup of procedure or equipment like IABP with its limited availability or lake of sufficient skillful physicians.

Similar to our findings: *Mehmet et al.,* [18] found that MVD was more frequent in non-survivors (p=0.004). In addition procedural success rate was substantially lower in non-survivors (39% vs. 84.4%; p<0.001). In multivariate regression analysis, unsuccessful procedure (OR 7.2, 95% CI 1.77-29.27; p=0.006) was the independent predictor of in-hospital mortality. *Arun et al.,* [19] showed that the mortality was 50% and TIMI flow 0/1 was one of the causes of mortality. *Silber et al.,* [20] found that primary PCI reduces the mortality rate in STEMI patients who have worsening condition due to cardiogenic shock. PCI is recommended for only infarct-related coronary artery in STEMI patients, whereas total revascularization is recommended in patients with cardiogenic shock and lower mortality rate for those patients is probably related to the higher success rate of the procedure. And *Marcin et al.,* [21] also found that successful PCI significantly reduced mortality in multivariate analysis of the independent predictors of in-hospital mortality (p<0.05).

Similar to our findings: meta-analysis done by *Krischan et al.,* [22] exhibited IABP therapy to be associated with an absolute increase in 30 day mortality of 6% (95% CI, 3–10%; P=0.0008). *NRMI-2 cardiogenic shock cohort study*; reported that IABP therapy was independently associated with a higher-30-day mortality after multivariate adjustment for age, several clinical risk factors, PCI, and CABG. So IABP therapy may have been preferentially given to patients in worse condition. *Mehmet et al.,* found that the incidence of intra-aortic balloon pump use was significantly higher in the patient group with procedural failure, compared to those who underwent a successful procedure as regard viability (58% vs. 85.3%; p=0.004). *Sergio et al.,* [23] also found no benefit from IABC use on reducing mortality in patients with STEMI with CS and undergoing revascularization.

The IABP-SHOCK II trial endorses the downgraded recommendations for IABP therapy in AMI complicated by CS. Most important, this successfully conducted large-scale trial should be an encouragement for further research, since mortality in CS is still unacceptably high. Currently, the ACC/AHA (2013) and ESC (2012) guidelines do not explicitly address the use of IABP therapy in high-risk STEMI patients. The pooled randomized data do not support IABP therapy in this setting. As many practitioners still use IABP therapy in high-risk STEMI patients, a guideline statement about IABP therapy according to the appropriate classification of recommendation and level of evidence should be considered for this indication. Cardiogenic shock, when not quickly reversed by pharmacologic therapy, is listed in the ACC/AHA (2013) guidelines as a class IIa recommendation for IABP use.

6. Conclusion

1) The in-hospital mortality rate was 44.6% (25 out of the 56 patients included in the study). This rate may be higher than other studies as it ranged between 24 – 40%.

2) Older age was not an independent predictor of in-hospital mortality, unlike other observational studies, and this may be attributed to high prevalence of risk factors in young age population in our Egyptian setting.

3) Risk factors (dyslipidemia, DM), multi-vessel disease, only culprit vessel revascularization, Post PCI TIMI flow (<grade III), procedure failure, and lower ejection fraction on admission were all associated with a lower survival time expectancy.

4) Dyslipidemia, DM, Hypertension, LVEF% on admission, Multi-vessel coronary artery disease, TIMI flow after PCI (<grade III), and ↑ CK-MB (72-hour serial measurement), were all found to be significant predictors of in-hospital mortality.

5) The onset from chest pain to ED arrival and the door-to-balloon time were higher than that reported in the previous studies.

6) The use of IABP was not found to have a significant predictor effect on the different outcome among our patients with STEMI.

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