Role of Angiographic Perfusion Score in Prediction of Perfusion Success and Risk Stratification Following Primary PCI versus Pharmaco-Invasive PCI for Anterior STEMI

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Role of Angiographic Perfusion Score in Prediction of Perfusion Success and Risk Stratification Following Primary PCI versus Pharmaco-Invasive PCI for Anterior STEMI

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Abstract

Background: When a susceptible coronary atherosclerotic plaque is damaged, myocardial infarction in the ST segment occurs. The angiographic perfusion score (APS) compares the primary Percutaneous Coronary Intervention (PCI) clinical outcome for a STEMI patient to the pharmaco-invasive PCI.

Aim: to assess the short-term clinical outcomes between primary PCI and pharmaco-invasive PCI in a STEMI patient.

Methods: Eighty individuals who presented within the first day of onset of symptoms and were diagnosed with anterior STEMI underwent this prospective observational research. The patients were separated into two groups of equivalent size: Group II was treated with streptokinase, a fibrinolytic agent, at a hospital that was unable to perform PCI before being moved to a PCI-capable center that performed PCI within 3 to 24 hours. Group I got primary PCI.

Results: There were no statistically significant differences between the groups receiving fibrinolytic therapies and primary PCI as regards ejection fraction (EF%) and SWMI pre and post-treatment, with a substantial increase in APS-raised thrombolysis in myocardial infarction (TIMI) in Group II, and minimized tissue myocardial perfusion grade (TMPG) than group I. The univariate analysis illustrated that history of diabetes (COR=3.13), decrease in EF, pre-treatment (COR=0.694), increase in SWMI pre (COR=533.89), and reduction in APS (COR=0.413) were Major adverse cardiac events (MACE) estimations that are statistically noteworthy. Multivariate analysis illustrated that EF and APS are statistically significant predictors affecting MACE among studied cases (AOR=10.15 & 0.662).

Conclusions: APS is a more accurate indicator of perfusion success and a good predictor of MACE in anterior STEMI patients after primary PCI or pharmaco-invasive treatment.

Keywords: Angiographic Perfusion Score; Primary PCI; Pharmaco-Invasive PCI; STEMI

1. Introduction

Beforehand restoration of coronary blood flow to the imperiled myocardium is the primary goal of medical treatment for acute myocardial infarction (AMI). Primary percutaneous coronary intervention (PCI), when performed swiftly, is an efficient therapy for anterior ST-Segment elevation myocardial infarction (STEMI). Although intravenous thrombolysis is effective and widely accessible, A substantial incidence of re-occlusion and a high percentage of failed reperfusion seriously jeopardize this therapy’s utility. As a result, many STEMI patients arrive at hospitals with on-site PCI capability, making it possible for them to undergo PCI within the guidelines’ proposed time frames. Instead, the initial sort of reperfusion therapy that patients get is fibrinolytic.

Angiographic results, procedural events, and clinical outcomes are the three connected factors that most accurately describe the effectiveness of a reperfusion operation.
In ACCF/AHA/SCAI 2011, A minimum diameter stenosis of 10% [with an ideal target of as close to 0% as practical] was used to determine angiographic success. When grade 3 final thrombolysis in myocardial infarction (TIMI) flow is achieved in the infarct–related coronary artery without the obstruction of a large side branch, flow-limiting dissection, distal embolization, or angiographic thrombus; reperfusion therapy is regarded as angiographically successful. Reduced mortality following coronary revascularization has been linked to improving TIMI flow grades (TFG) for epicardial flow. Even when a good TIMI flow is produced, some patients still have less than perfect tissue-level reperfusion, and myocardial reperfusion is only sometimes effective in patients with a good TIMI flow of the epicardial coronary artery. 3

A single perfusion grade is determined using the Angiographic Perfusion Score (APS), a simple angiographic measurement considering indications of myocardial and epicardial perfusion before and during PCI. Grades ranging from 0 to 12 are possible since the APS is calculated by including the TFG (0-3) and TIMI Myocardial Perfusion Grade (TMPG) (0-3) before and following PCI. 4

The attainment of angiographic success with no serious in-hospital clinical consequences (passing away, myocardial infarction (MI), stroke, or urgent coronary artery bypass grafting (CABG)) was considered a procedural success for reperfusion. The clinically effective PCI involves morphological and procedural success in alleviating myocardial ischemia signs and symptoms. 5 Our study aimed to assess the short-term clinical outcomes between primary PCI and pharmaco-invasive PCI in a STEMI patient.

2. Patients and methods

In this prospective observational study, 80 patients with anterior STEMI who underwent primary PCI between 12 and 24 hours after the onset of symptoms or within 12 hours of the onset of symptoms with evidence of ongoing ischemia (typical ischemic chest pain lasting longer than 30 minutes or other symptoms suggestive of ischemia with ST-segment elevation >1mm in at least two contiguous leads, likely new Left Bundle Branch Block (LB) on Electrocardiogram (ECG) combined with increase in ST. The patients were randomly divided into two equal groups: Group I underwent primary PCI at a PCI-capable center. Group II: received fibrinolytic therapy in the form of streptokinase in a non-PCI capable center, then transferred to a PCI capable center for PCI within 3–24 h (Pharmaco-invasive strategy).

The study was carried out after receiving clearance from the Al Azhar University Hospitals’ Ethical Committee. The patient gave written, fully informed consent.

Exclusion standards: Patients with unstable angina, non-ST-segment elevation myocardial infarction, anterior "STEMI" patients presented 24 hours from symptoms onset, more than 24 hours afterward, to a PCI-capable center for PCI, failed PCI in anterior STEMI patients, patients refused to be enrolled in the study, patients with Poor echocardiographic windows.

All patients had a history-taking procedure, a clinical examination (including vital signs), an admission check of their heart rate and systolic blood pressure, usual laboratory tests, and a duration of chest pain recording. Killip classes II-IV: A delay of more than 4 hours has been reported in cases with anterior myocardial infarction MI or LBBB presentation6. TIMI stands for thrombolysis in myocardial infarction. TRS computations and the Global Registry of Acute Coronary Events (GRACE) were mentioned. 6

PCI: The transfemoral standard method used a 6 Fr sheath in all patients. As soon as the artery sheath was in place, 10,000 units of heparin were given if the procedure took more than an hour; an additional 5,000 units were administered after an hour.

Coronary angiography was done to determine the culprit lesion’s location, degree of occlusion, and TIMI flow grade. To address the issues of foreshortening and superimposition of the vessels, many projections were made to help visualize the coronary arteries. By visual inspection, the luminal diameter reduction relative to a proximal section of the unaffected conduit was used to evaluate the degree of stenosis.

Various guide wires are also used, including floppy, intermediate, and hydrophilic wires. The most used guiding catheter size is 6 Fr. Although vascular anatomy, lesion morphology, and the devices to be used all impact the choice of the guiding catheter and guide wire, the subsequent balloon angioplasty, if necessary, and stent implantation were performed with appropriately sized devices. Either DES or BMS was selected based on the circumstances.

Coronary flow assessment concerning APS, which is the total grade that may be assigned, ranging from 0 to 12, and is made up of the TMPG (0-3) added to the TIMI flow grade (TFG; 0-3), both before and after PCI. Following PCI, the coronary blood flow patterns were carefully evaluated using the TIMI flow grades of 0, 1, 2, and 3. 7

When performing coronary angiography, the quality of the coronary flow is assessed using the TIMI blood flow grades (The TIMI trial, 1985). As a measure of the myocardium’s filling and elimination of contrast, the TIMI perfusion grade “myocardial blush” has been proposed. 8

Criteria for assessing Success: ACCF/AHA/SCAI
2011 Angiographic, procedural, and clinical Success.

Care for the access site and monitoring for myocardial ischemia were part of the post-PCI treatment. An ECG with 12 leads is taken 60 to 90 minutes after PCI. The patient is observed in a CCU with continuous ECG monitoring and routine post-PCI care and is given medications (lifelong Aspirin (75-100 mg/d), clopidogrel 75 mg/d for at least six months after taking 150 mg/d for seven days, beta-blockers, ACEIs, or ARBs, and spironolactone as necessary for low-density lipoprotein (LDL) c level of 70 mg/dL, Tirofiban was used as a last resort therapy in patients with substantial thrombus burden and no-reflow when G IIb/IIa inhibitors were not an option. Patients were then monitored to look for any in-hospital Major Adverse Cardiac Events (MACEs) or other hemodynamic problems.

Transathoracic echocardiography (TTE):

Performed at the time of hospitalization at the CCU and within 4 to 6 weeks after discharge to ascertain the following:

- Calculating the left ventricular ejection fraction (LVEF) using fractional shortening (FS) and ejection fraction (EF) is as follows: [SV/EDV] x 100 LVEF. The LVEF, as evaluated by two-dimensional (2D) echocardiography, falls between the following typical limits, according to the American Society of Echocardiography and the European Association of Cardiovascular Imaging: The assessment methods used by echocardiography include linear (one-dimensional), area- or volume-based approaches. Ionizing radiation is not present, which makes echocardiography easier. The most effective 2D method for evaluating LVEF is recommended as the modified Simpson’s rule (biplane technique of discs). This modality necessitates area tracings of the LV cavity. The American Society of Echocardiography recommends this method, which includes measuring LVEF by tracing the endocardial boundary in both the apical four-chamber and two-chamber views at both end-systole and end-diastole. These tracings finally separate the LV cavity into a certain number of discs, usually 20. Based on the study’s tracings, disc volumes were created.

- Segments, as recommended by the American Society of the Wall Motion Score Index (WMSI) for the left ventricle:

  A common transthoracic echocardiography technique assigns a score of 1 to 4 to each myocardial segment. The 16-segment model of myocardial segmentation is suggested because the 17-segment model's contractile apical cap makes it less appropriate for perfusion imaging. The left ventricle was divided by line. Mid segments (six segments similar to the base), basal segments (six segments, each encompassing 60 degrees of the left ventricular short axis, basal anterior, basal anterolateral, basal inferior, and basal inferoseptal), and apical segments (four segments at 90-degree intervals, apical anterior, apical lateral, basal inferior, and apical septum) are all present.

  Then, each segment is assessed using the following criteria: Normal kinesia is defined as hypokinesia (2 points), decreased wall thickening, decreased endocardial excursion, akinesia (3 points), absence of either wall thickening or endocardial excursion and dyskinesia (4 points). The WMSI is then calculated by dividing the sum of the segmental values by the (16) myocardial segment count. It is thought that 1.0 WMSI (16/16) is norm kinetic. In contrast, a WMSI of 3.0 is seen as akinetic. Mild hypokinesia, hypokinesia, and severe hypokinesia are the labels given to WMSIs of 1.5, 2.0, and 2.5, respectively. The quantitative evaluation created a scoring system using the following point system: regular = 1, hypokinesia = 2, akinesia = 3, dyskinesia = 4, and aneurysm = 5. The cardiologist calculated the score, as mentioned before. The wall motion was classified as usual, hypokinetic, akinetic, or dyskinetic, depending on the systolic wall thickening. Hypokinesis is a systolic wall thickening of less than 30%, and akininesis is a systolic wall thickening of less than 10%. Dyskinesis is a myocardial segment moving outward during systole, usually associated with systolic wall thinning.

  Detection of acute MI mechanical consequences comprises actual aneurysm, fake aneurysm, rupture of the papillary muscle, rupture of the left ventricular free wall, and rupture of the ventricular septum.

  They were monitoring for major adverse cardiac events (MACEs) during inpatient follow-up. Cardiogenic shock, newly formed advanced heart failure (HF), pulmonary emphysema (as acute myocardial infarction and heart failure syndrome develop. (as MI is characterized by passive pulmonary congestion, which leads to hypoxemia, and this hypoxemia indicates the functional disturbance of the lung, which may lead to pulmonary emphysema), complete atrioventricular block (AVB) requiring a temporary pacemaker, severe ventricular arrhythmia, and in-hospital mortality during the post-PCI follow-up period were all categorized as MACEs. Only when a patient passed away in a hospital and an MI, a cardiac arrest, or another cardiac-related event was the cause of death was the death categorized as a MACE. Cardiogenic shock was defined as an SBP of less than 80 mmHg with symptoms of hypoperfusion brought on by left ventricular failure, right ventricular infarction, or mechanical issues with the heart. Severe ventricular arrhythmias (ventricular tachycardia, ventricular...
fibrillation, or systole) and newly established advanced heart failure were regarded as MACEs if they occurred within 48 hours after the start of the condition.

The primary outcome was the effectiveness of PCI versus pharmaco-invasive PCI. The secondary outcomes were the in-hospital mortality rate for anterior STEMI, the performance of the Angiographic Perfusion Score, and major cardiovascular events.

Statistical analysis

Versions of the Statistical Programme for Social Science (SPSS) and MedCalc were used to analyze the data. Standard deviation (SD) was used to convey quantitative data. Tests that were used to describe qualitative data as frequency and percentage included: Whitney Mann Non-normally distributed data may be compared between two means using the U test, two means can be compared using the independent-samples t-test of significance, and proportions between two qualitative characteristics can be compared using the Chi-square (X2) test of significance. Fisher The exact test, a test of significance, is used in 2 tables rather than the chi-square test, especially when there are few samples. ROC curve analysis, or receiver operating characteristic analysis, was used to identify the appropriate cut-off settings. Two-tailed P values < 0.05 were used to determine significant results.

3. Results

At the hospital, 80 patients were divided randomly into two equal groups for the study, and forty cases from each group were then examined. Figure 1

Table 1. Examining the sociodemographic differences and risk factors between the groups under study

<table>
<thead>
<tr>
<th>Group I (N=40)</th>
<th>Group II (N=40)</th>
<th>Test of Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>AGE 59.23±4.29</td>
<td>59.50±4.30</td>
<td>t=0.338 p=0.736</td>
</tr>
<tr>
<td>SEX Male 30(75.0 %)</td>
<td>25(62.5%)</td>
<td>χ²=1.46 p=0.228</td>
</tr>
<tr>
<td>Female 10(25.0%)</td>
<td>15(37.5%)</td>
<td></td>
</tr>
<tr>
<td>HYPERTENSION 14(35%)</td>
<td>13(32.5%)</td>
<td>χ²=0.056 p=0.813</td>
</tr>
<tr>
<td>DYSLIPIDEMIA 6(15%)</td>
<td>7(17.5%)</td>
<td>χ²=0.621 p=0.431</td>
</tr>
<tr>
<td>DM 11(27.5%)</td>
<td>8(20%)</td>
<td>χ²=0.238 p=0.808</td>
</tr>
<tr>
<td>SMOKING 13(32.5%)</td>
<td>11(27.5%)</td>
<td>χ²=0.556 p=0.456</td>
</tr>
<tr>
<td>FAMILY HISTORY 3(7.5%)</td>
<td>5(12.5%)</td>
<td></td>
</tr>
</tbody>
</table>

Data are shown as means with standard deviations (SD) or frequency (%), t: Student t test, and 2: Chi-Square test.

There is no statistically significant difference between studied groups as regard body mass index, systolic blood pressure, pulse, hemoglobin level, serum creatinine, ALT, total cholesterol, TGS, LDL, HDL, and troponin positive cases (p>0.05). A statistically significant higher mean AST is detected for group 1 as compared to group 2 (19.85 & 18.05 IU/ml, respectively).

Table 2

<table>
<thead>
<tr>
<th>Group I (n=40)</th>
<th>Group II (n=40)</th>
<th>Test of Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Body mass index (kg/m²) 27.09±3.20</td>
<td>27.26±3.39</td>
<td>t=0.237 p=0.813</td>
</tr>
<tr>
<td>Systolic blood pressure (mm/Hg) 128.95±9.92</td>
<td>129±12.09</td>
<td>t=0.02 p=0.984</td>
</tr>
<tr>
<td>Diastolic blood pressure (mm/Hg) 72.68±8.85</td>
<td>74.33±10.26</td>
<td>t=0.770 p=0.443</td>
</tr>
<tr>
<td>Pulse 85.25±8.11</td>
<td>84.75±9.65</td>
<td>t=0.251 p=0.803</td>
</tr>
<tr>
<td>HB (gm/dl) 12.92±1.16</td>
<td>13.28±1.32</td>
<td>t=1.27 p=0.208</td>
</tr>
<tr>
<td>Serum creatinine(mg/dl) 0.98±0.184</td>
<td>0.93±0.219</td>
<td>t=1.04 p=0.303</td>
</tr>
<tr>
<td>AST(IU/ml) 19.85±4.15</td>
<td>18.05±3.79</td>
<td>t=2.03 p=0.046*</td>
</tr>
<tr>
<td>ALT(IU/ml) 23.55±8.07</td>
<td>21.25±6.34</td>
<td>t=1.42 p=0.160</td>
</tr>
<tr>
<td>Total cholesterol(mg/dl) 193.78±27.84</td>
<td>190.35±22.12</td>
<td>t=0.904 p=0.369</td>
</tr>
<tr>
<td>TGS (mg/dl) 140.20±22.38</td>
<td>136.08±18.22</td>
<td>t=2.03 p=0.046*</td>
</tr>
<tr>
<td>LDL (mg/dl) 117.13±8.36</td>
<td>116.55±9.61</td>
<td>t=0.285 p=0.776</td>
</tr>
<tr>
<td>HDL (mg/dl) 50.80±5.30</td>
<td>49.30±6.73</td>
<td>t=1.11 p=0.271</td>
</tr>
<tr>
<td>Troponin -ve 14(35.0%)</td>
<td>12(30.0%)</td>
<td>χ²=0.228 p=0.633</td>
</tr>
<tr>
<td>+ve 26(65.0%)</td>
<td>28(70.0%)</td>
<td></td>
</tr>
</tbody>
</table>

Table 2. Comparison of the studied groups regarding evaluations of the study groups' clinical examinations and laboratory results

Figure 1. Study design consort flow chart

Age, sex, medical history including hypertension, dyslipidemia, diabetes, smoking and family history, and other factors were statistically insignificantly different across the study groups. Table 1
The data is displayed as mean ± SD or frequency (%), t: Student t test, 2: Chi-Square test, and *statistically significant.

There was no statistically significant difference between studied groups as regard ejection fraction pre and post treatment. For group 1; a statistically significant increase of ejection fraction from 46.53 to 50.2 and for group 2; a statistically significant increase of ejection fraction from 44.6 to 47.53 between pre and post treatment. non statistically significant difference between studied groups as regard SWMI pre and post treatment. For group 1; a statistically significant decrease of SWMI from 1.71 to 1.59 between pre and post treatment. statistically significant higher decrease of SWMI from 1.62 to 1.46 post treatment. For group 1; a statistically significant increase of ejection fraction pre and post treatment. For group 2; a statistically significant increase of ejection fraction from 44.60 to 50.2 and for group 2; a statistically significant increase of ejection fraction pre and post treatment. Fischer exact test*statistically significant.

Table 4. TMPG TIMI and before and after PCI among analysed groups

<table>
<thead>
<tr>
<th>GROUP</th>
<th>N=40</th>
<th>GROUP</th>
<th>N=40</th>
<th>TEST OF SIGNIFICANCE</th>
</tr>
</thead>
<tbody>
<tr>
<td>(TMPG) BEFORE PCI</td>
<td>0±0</td>
<td>0.70±0.79</td>
<td>2(5%)</td>
<td>z=5.43</td>
</tr>
<tr>
<td>(TMPG) AFTER PCI</td>
<td>1.90±1.15</td>
<td>2.60±0.90</td>
<td>2(5%)</td>
<td>z=3.07</td>
</tr>
<tr>
<td>DELTA CHANGE</td>
<td>-1.9</td>
<td>-1.9</td>
<td>2(5%)</td>
<td>2(5%)</td>
</tr>
<tr>
<td>WILCOXON SIGNED RANK TEST</td>
<td>z=5.11</td>
<td>z=5.36</td>
<td>2(5%)</td>
<td>2(5%)</td>
</tr>
<tr>
<td>TEST (TIMI) BEFORE PCI</td>
<td>0.325±0.47</td>
<td>1.80±1.02</td>
<td>2(5%)</td>
<td>z=5.97</td>
</tr>
<tr>
<td>(TIMI) AFTER PCI</td>
<td>2.90±0.44</td>
<td>1.61±1.02</td>
<td>2(5%)</td>
<td>z=0.001*</td>
</tr>
<tr>
<td>DELTA CHANGE</td>
<td>-2.66</td>
<td>-0.61</td>
<td>2(5%)</td>
<td>2(5%)</td>
</tr>
<tr>
<td>WILCOXON SIGNED RANK TEST</td>
<td>z=5.73</td>
<td>z=4.21</td>
<td>2(5%)</td>
<td>2(5%)</td>
</tr>
</tbody>
</table>

Statistics are reported as mean, standard deviation, and frequency (%). Statistically significant predictors of Major adverse outcome, EF, and APS among studied cases. Table 5

Table 5. Multivariate and univariate regression analysis for significant adverse outcome predictors

<table>
<thead>
<tr>
<th>UNIVARIATE ANALYSIS</th>
<th>MULTIVARIATE ANALYSIS</th>
</tr>
</thead>
<tbody>
<tr>
<td>COR (95%CI)</td>
<td>β</td>
</tr>
<tr>
<td>AGE/YEARS</td>
<td>0.656</td>
</tr>
<tr>
<td>SEX Male</td>
<td>0.949</td>
</tr>
<tr>
<td>HYPERTENSION</td>
<td>0.696</td>
</tr>
<tr>
<td>DM</td>
<td>0.036*</td>
</tr>
<tr>
<td>DYSLIPIDEMIA</td>
<td>0.617</td>
</tr>
<tr>
<td>SMOKING</td>
<td>0.677</td>
</tr>
<tr>
<td>FAMILY HISTORY</td>
<td>0.275</td>
</tr>
<tr>
<td>BODY MASS INDEX (KG/M2)</td>
<td>0.387</td>
</tr>
<tr>
<td>SYSTOLIC BLOOD PRESSURE(MM/HG)</td>
<td>0.766</td>
</tr>
<tr>
<td>DIASTOLIC BLOOD PRESSURE (MM/HG)</td>
<td>0.564</td>
</tr>
<tr>
<td>PULSE</td>
<td>0.071</td>
</tr>
<tr>
<td>HB</td>
<td>0.423</td>
</tr>
<tr>
<td>SERUM CREATININE</td>
<td>0.138</td>
</tr>
<tr>
<td>AST</td>
<td>0.057</td>
</tr>
</tbody>
</table>
ALT 0.632 (0.779 - 1.004)
TOTAL CHOLESTEROL 0.493 (0.975 - 1.01)
TGS 0.249 (0.962 - 1.01)
LDL 0.919 (0.946 - 1.05)
HDL 0.437 (0.952 - 1.12)
TROPONIN -VE (R), +VE 0.779 (0.322 - 2.34)
EF % PRE <0.001* 0.694 (0.579 - 0.832)
SWMI PRE <0.001* 533.89 (24.0 - 1187)
APS <0.001* 0.413 (0.278 - 0.612)
MECHANICAL COMPLICATIONS 0.453 2.17 (0.288 - 16.31)
GROUPS 1, 2(R) 0.633 1.250 (0.492 - 3.21)

OVERALL % PREDICTED =90%

AOR stands for adjusted odds ratio, COR stands for crude odds ratio, and both are used to show data.

Case no. (20) group (A)

Male patient, 58 years old, diabetic, not hypertensive, not smoker, and not known to be ischemic heart disease before. He is presented with retrosternal compressing chest pain 9 hours duration. Blood pressure was 120/60 mmHg, heart rate 95 bpm. 12-lead ECG: sinus rhythm with ST elevation in lead v1-v6 with pathological Q wave in v1-v3 Hb: 11.7, Serum creatinine: 1.2 mg/dl and Troponin: +ve. Echocardiography in the hospital: LVEF 49%. RSWMA inform of hypokinesia of basal, mid and apical septal wall with WMSI: 1.18. No mechanical complications.

Patient received Aspirin 300 mg PO, in form of chewable tablets and loading dose of 600 mg clopidogrel PO were given then admitted to Catheterization lab immediately.

Angiographic perfusion score (APS): TIMI flow grade before PCI: 1. TMPG before PCI: 0. TIMI flow grade after PCI: 3. TMPG after PCI: 3. APS: 7.

In hospital follow up:

patient transferred to CCU under medical treatment and observation, no major acute cardiac events (MACE) and discharged after 2.5 days.

Follow up patient after 8 weeks:

patient was compliant in medical treatment has no chest pain with good function capacity. Follow up Echocardiography: LVEF 52 %. RSWMA inform of hypokinesia of basal, mid and septal wall with WMSI: 1.12.

No mechanical complications. Figure 2

Case no. (71) Group (B)

Demographic and clinical data: Female patient, 57 years old, diabetic, not hypertensive, not smoker, and not known to be ischemic heart disease before.

Clinical presentation: She is presented with retrosternal compressing chest pain starting from one hour. Blood pressure was 123/6 mmHg, heart rate 113 bpm. ECG: 12-lead ECG: sinus rhythm, with ST elevation in lead v1-v6 Laboratory findings: Hb: 13, Serum creatinine: .9 mg/dl and Troponin: +ve. Echocardiography in the hospital: LVEF 57%. RSWMA informs hypokinesia of basal, mid and apical septal wall with WMSI: 1.76. No mechanical complications.

Management:

patient received Aspirin 300 mg PO, in form of chewable tablets and a loading dose of 600 mg clopidogrel PO then received fibrinolytic therapy in the form of streptokinase and after finishing fibrinolytic therapy patient was vitally stable, off chest pain and referred to Catheterization lab within 6 hours.

Angiographic perfusion score (APS): TIMI flow grade before PCI: 2. TMPG before PCI: 0. TIMI flow grade after PCI: 3. TMPG after PCI: 3. APS: 8.

In hospital follow up:

patient transferred to CCU under medical treatment and observation, no major acute cardiac events (MACE) and discharged after 2 days.

Follow up patient after 8 weeks:

Figure 2. Case no. (20) of Group A (A) ECG, (B) Echocardiographic images during hospitalization, (C) Coronary angiography revealed proximal subtotal LAD occlusion, LCX: normal non dominant artery with normal dominant RCA, (D) Primary PCI to LAD with one DES with TIMI flow III and (E) Follow up Echocardiographic images after 8W.

Follow up patient after 8 weeks:
patient was compliant in medical treatment has no chest pain with good function capacity. Follow up Echocardiography was the same. LVEF 57%.

RSWMA informs hypokinesia of basal, mid and septal wall with WMSI: 1.32. Figure 3.

Figure 3. Case no. (71) Group (B). (A) ECG Group (B), (B) Echocardiographic images in hospital, (C) Coronary angiography after fibrinolytic therapy Revealed proximal 80% LAD lesion, LCX: normal dominant artery with normal RCA, (D) PCI to LAD with one DES with TIMI flow I and (E) Follow up Echocardiographic images (71) after 8W.

4. Discussion

Primary PCI and the group getting fibrinolytic therapy were not significantly distinct from one another in our research about Body mass index, systolic and diastolic blood pressure, pulse, hemoglobin level, serum creatinine, ALT, total cholesterol, TGS, LDL, HDL, and cases of troponin positive instances (p>0.05), among other factors, are used to determine a patient's medical history (including diabetes, smoking, hypertension, dyslipidemia, obesity, and positive family history). Among the primary PCI group, 35% were hypertensive, 15% were dyslipidemia, 27.5% had diabetes, 32.5% were smoking, and 7.5% had a family history, and for the fibrinolytic therapy group; 32.5% were hypertensive, 17.5% were dyslipidemia, 20% DM, 27.5% smokers, and 12.5% positive family history.

In agreement with this result, Mustafa et al. observed that there was no statistically significant difference between the standard conservative group and the pharmaco-invasive strategy group for smoking, hypertension, diabetes, family history of coronary artery disease, BMI, SBP, HR, peak troponin level, serum creatinine, cholesterol, triglyceride level, EF, and SWMI six weeks after MI. This finding agreed with Helal et al. He demonstrated no significant difference between the major PCI and fibrinolytic therapy groups in the risk factors for ischemia (smoking, diabetes mellitus, hypertension, dyslipidemia, obesity, and positive family history).

In our study, for the primary PCI group, there was a statistically significant increase in TMPG after PCI as compared to before PCI from 0 to 2 and before PCI from 1 to 3, TIMI before PCI without Difference complying with PCI that is of statistical significance between the groups using fibrinolytic therapy and primary PCI. There was a statistically significant increase in median TIMI after PCI compared to before PCI, from 0 to 2 or 3 for the primary PCI group. For the fibrinolytic therapy group, there was a statistically significant increase in TIMI after PCI compared to before PCI from 2 to 3. There is no discernible variation between the groups obtaining fibrinolytic therapy after PCI and primary PCI. There was a statistically significant increase in median TIMI after PCI compared to before PCI, from 0 to 2 or 3 for the primary PCI group.

In agreement with this result, Pu et al. found that TMPG flow from 1 to 3 pre-PCI and TIMI flow pre-PCI from 0 to 2 or 3 were statistically significantly different between the PPCI and PhI groups.

Similarly, Sousa et al. revealed a statistically significant difference in terms of TIMI between the PIT group and the rescue PCI group. Additionally, for the PIT group, there was a statistically significant increase in median TIMI after PCI as compared to before PCI from 0 or 1 to 2 or 3. For the required rescue PCI group, there was a statistically significant increase in TIMI after PCI as compared to before PCI from 0 or 1 to 2 or 3.

In our study, univariate analysis illustrated that the following were statistically significant predictors of primary adverse outcomes among studied cases: history of diabetes (COR=3.13), decrease in EF % pretreatment (COR=0.694),
increase in SWMI pre (COR=533.89) and reduction in APS (COR=0.413).

In agreement with this result, Lee et al. Patients who got PCI with second-generation drug-eluting stents (DES) were analyzed for composite outcomes of death from any cause, MI, and any repeat revascularization in a multicenter observational analysis of 1,913 MACE patients. In terms of univariate analysis, they discovered that prior diabetes history (COR= 2.24) is considered a significant predictor of primary adverse outcomes among studied cases.

In contrast to our results, Bianco et al. conducted a prospective and observational study on 2,290 STEMI patients. They found that, regarding univariate analysis, the following were statistically significant predictors of major adverse outcomes among studied cases: dyslipidemia (COR=1.39) and chronic kidney disease (COR=0.53).

Our study's multivariate analysis illustrated that EF and APS are statistically significant predictors affecting MACE among studied cases (AOR=10.15 & 0.662) with the overall % predicted =90%. In agreement with this result, Ghazal et al. found that patients with post-TIMI 1, 2, and 3 had a significantly higher risk of MACE than patients with post-TIMI 3 (P <0.05) following initial PCI, so TIMI was a significant predictor affecting MACE.

In agreement with this result, Ghazal et al. conducted prospective observational research on 100 STEMI patients to assess how well the RISK-PCI score and the shock index (SI) predict major adverse cardiovascular events (MACE) and death following primary percutaneous coronary intervention (PCI). They found that patients with post-TIMI 1, 2, and 3 had a significantly higher risk of MACE than patients with post-TIMI 3 (P 0.05) following initial PCI. As a result, TIMI significantly predicted MACE. Similarly, Kiatchoosakun et al. Found that regarding multivariate analysis, TIMI final grade 0/1 (OR 20.55, 95% CI 3.49 to 120.94) and LVEF< 40% (OR 2.53, 95% CI 1.20 to 5.36). Were significant predictors of in-hospital mortality.

Gibson, et al.’s findings, corroborate our findings. They found that the APS integrates epicardial and tissue-level perfusion indexes before and after PCI or after diagnostic cardiac catheterization to arrive at a single grade related to the 30-day mortality rate, MI rate, or both, and infarct size.

Limitations: The sample size was small. The study was done at a single facility; more clinical outcome data must be acquired to support the perfusion advantage shown in this trial; differences in medication consumption patterns were not examined; and the investigation was limited to one location, which might undoubtedly impact the outcomes of reperfusion. Long patient transfers to hospitals, and a limited follow-up timeframe (confined to hospital stays) also contributed to the limitations.

5. Conclusion

APS serves as a superior mirror for hemodynamic efficiency as well as a favorable prediction for MACE among the patients with anterior STEMI following primary PCI or pharmaco-invasive.

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