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Al-Azhar University
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Impact of Timing of Primary PCI in Patients with Acute Anterior STEMI on Left Ventricle Function and Perfusion

Thesis

Submitted for partial fulfillment of MD degree in Cardiology

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Contents

Abbreviation

ACC/AHA:	American Colleague of Cardiology/ American Heart Association.
AO:	Aortic diameter.
ASC:	American Society of Cardiology.
DM:	Diabetes mellitus.
EF:	Ejection fraction.
ESC:	European society of cardiology.
FH:	Family history.
HTN:	Hypertension.
LAD:	Left atrium diameter
LAD:	Left anterior descending coronary artery.
LCX:	Left circumflex coronary artery.
LM:	Left main coronary artery.
LV EDV:	Left ventricular end diastolic volume.
LV ESV:	Left ventricular end systolic volume.
LVEDD:	Left ventricular end diastolic diameter.
LVESD:	Left ventricular end systolic diameter.
MACE:	Major adverse cardiac events.
MBG:	Myocardial blush grade.
NSTEMI:	Non ST elevation myocardial infarction.
RCA:	Right coronary artery.
RWMS:	Regional wall motion score.
RWMSI:	Regional wall motion score index.
SPSS	Statistical Package for Social Sciences
STEMI:	ST elevation myocardial infarction.
TIMI flow:	Thrombolysis in myocardial infarction flow.
TVAC	Thrombus vacuum aspiration catheter.

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INTRODUCTION

Primary percutaneous coronary intervention (PPCI) is the standard treatment in patients with ST-segment elevation myocardial infarction (STEMI) achieving a Thrombolysis In Myocardial Infarction (TIMI) flow grade 3 in 90% of patients. However, despite a "brisk" epicardial coronary flow in the infarct-related artery, microvascular damage frequently limits the efficacy of PPCI ***(Svilaas et al., 2008).***

However, favorable outcomes with PPCI may be attenuated by intrahospital and interhospital transport delays from first medical contact to balloon inflation or reperfusion in the catheterization laboratory ***(Kiernan et al., 2007).***

Most critical point for PPCI is time; the golden period for myocardial salvage is the first 2 hours after onset of symptoms (***Gersh et al., 2005).***

Time to reperfusion for patients with ST-segment elevation myocardial infarction (STEMI) consistently predicts mortality for fibrinolytic therapy **(De *Luca et al., 2003).***

In contrast, studies have found conflicting results regarding the relationship between mortality and time to reperfusion with primary percutaneous coronary intervention (PCI). Some investigators have found lower mortality for shorter symptom onset to reperfusion time for all patients or just certain subgroups such as high risk patients or those presenting within 2 h of symptom onset ***(Antoniucci et al., 2002).***

Other studies found no lower mortality for shorter symptom onset to balloon time but did find lower mortality for shorter door to balloon time **(*Cannon et al., 2003*).** Some studies failed to find an association between either symptom onset to balloon time or door to balloon time and mortality ***(Zijlstra et al., 2002).***

Although the American College of Cardiology/American Heart Association (ACC/AHA) guidelines for management of patients with STEMI recommend door to balloon times of 90 min or less ***(Antman et al., 2008),*** a minority of patients are currently treated within this time period. The perception that time to reperfusion is less important in PCI may contribute to the current inertia in performance (***McNamara et al., 2006).***

Nowadays, the most advanced therapeutic regimen in AMI includes the execution of direct percutaneous coronary intervention (PCI) ***(Patrick, 2013*).** In this setting, the enzymatic measurements of infarct size are less reliable, particularly in the presence of reperfusion (***Gibbons et al., 2004*)** Similarly, the electrocardiographic criteria used in the patients submitted to thrombolysis are probably inadequate in the setting of primary PCI, there are no data about the incidence of aborted infarction, as defined by the usual enzymatic and electrocardiographic criteria, after primary PCI**.**

Primary percutaneous coronary intervention (PCI) is effective in opening the infarct-related artery in patients with myocardial infarction with ST-segment elevation. However, the embolization of atherothrombotic debris induces microvascular obstruction and diminishes myocardial reperfusion. (***Svilaas et al., 2008)***

The benefits of primary percutaneous coronary intervention in patients with STEMI have been ascribed to early restoration of thrombolysis in myocardial infarction grade 3 flow in the infarct-related artery that results in a limitation of infarct size and decreased mortality compared with thrombolytic treatment **(*Keeley et al., 2003).***

Although a clear relationship between mortality and the delay from symptom onset to treatment has been demonstrated in patients with ST-segment-elevation myocardial infarction (STEMI) treated by thrombolysis **(*Antoniucci et al., 2002*),** the impact of the time delay on prognosis in patients undergoing primary angioplasty has yet to be clarified***(Zijlstra et al., 2002*).**

Time-to-treatment was defined as the time from symptom onset to the first balloon inflation. Myocardial blush grade (MBG) assessed after primary angioplasty, Grade 0, no myocardial blush; Grade 1, minimal myocardial blush or contrast density; Grade 2, moderate myocardial blush or contrast density but less than that obtained during angiography of a contralateral or ipsilateral non infarct related coronary artery; Grade 3, normal myocardial blush or contrast density, comparable to that obtained during angiography of a contralateral or ipsilateral non infarct-related coronary artery **(*Tsvetkov et al., 2008*).**

AIM OF THE WORK

The aim of the present study is to evaluate the impact of timing of primary PCI (within 6 hours and after 6 up to 24 hours) of presentation on left ventricle function and perfusion.

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Acute Myocardial Infarction

ST Segment Elevation Myocardial Infarction

Definition:

STEMI is a clinical syndrome defined by characteristic symptoms of myocardial ischemia in association with persistent electrocardiographic (ECG) ST elevation and subsequent release of biomarkers of myocardial necrosis. Diagnostic STelevation in the absence of left ventricular (LV) hypertrophyor left bundle-branch block (LBBB) is defined by the European Society of Cardiology/ACCF/AHA/World Heart Federation Task Force for the Universal Definition of Myocardial Infarction as new ST elevation at the J point in at least 2contiguous leads of 2 mm (0.2 mV) in men or 1.5 mm (0.15 mV) in women in leads V2–V3 and/or of 1 mm (0.1mV) in other contiguous chest leads or the limb leads **(*Thygesen et al., 2012*).**

The majority of patients will evolve ECG evidence of Q-wave infarction. New or presumably new LBBB has been considered a STEMI equivalent. Most cases of LBBB at time of presentation, however, are "not known to be old" because of prior electrocardiogram (ECG) is not available for comparison. New or presumably new LBBB at presentation occurs infrequently, may interfere with ST-elevation analysis, and should not be considered diagnostic of acute myocardial infarction (MI) in isolation **(*Jain et al., 2011*).**

Criteria for ECG diagnosis of acute STEMI in the setting of LBBB have been proposed Baseline ECG abnormalities other than LBBB (eg,

paced rhythm, LV hypertrophy, Brugada syndrome) may obscure interpretation. In addition, STdepression in _2 precordial leads (V1–V4) may indicate transmural posterior injury multilead ST depression with coexistent ST elevation in lead aVR has been described in patients with left main or proximal left anterior descending artery occlusion **(*Wang et al., 2003)*.**

Rarely hyperacute T-wave changes may be observed in the very early phase of STEMI, before the development of ST elevation. Transthoracic echocardiography may provide evidence of focal wall motion abnormalities and facilitate triage in patients with ECG findings that are difficult to interpret. If doubt persists, immediate referral for invasive angiography may be necessary to guide therapy in he appropriate clinical context **(*De Winter et al., 2008*).**

TABLE 1: Aspects of Diagnosis of Myocardial Infarction by Different Techniques

TECHNIQUE	FEATURES
Pathology	Myocardial cell death
Biochemistry	Markers of myocardial cell death recovered from blood samples
Electrocardiography	Evidence of myocardial ischemia (ST and T wave abnormalities); evidence of loss of electrically functioning cardiac tissue (Q waves)
Imaging	Reduction or loss of tissue perfusion; cardiac wall motion abnormalities

(Thygesen et al., 2012)

TABLE 2: : universal Definition of Myocardial Infarction:

Criteria for acute myocardial infarction: The term acute myocardial infarction (MI) should be used when there is evidence of myocardial necrosis in a clinical setting consistent with acute myocardial ischaemia. Under these conditions any one of the following criteria meets the diagnosis for MI:
1- Detection of a rise and/or fall of cardiac biomarker values [preferably cardiac troponin (cTn)] with at least one value above the 99th percentile upper reference limit (URL) and with at least one of the following:
a- Symptoms of ischaemia.
b- New or presumed new significant (ST–T) changes or new LBBB.
c- Development of pathological Q waves in the ECG.
d- Imaging evidence of new loss of viable myocardium or new RWMA.
e- Identification of an intracoronary thrombus by angiography or autopsy.
2- Cardiac death with symptoms suggestive of myocardial ischaemia and presumed new ischaemic ECG changes or new LBBB,but death occurred before cardiac biomarkers were obtained, or before cardiac biomarker values would be increased.
3- Percutaneous coronary intervention (PCI) related MI is arbitrarily defined by elevation of cTn values (>5 x 99th percentile URL) in patients with normal baseline values (≤99th percentile URL) or a rise of cTn values >20% if the baseline values are elevated and are stable or falling. In addition, either
a- Symptoms suggestive of myocardial ischaemia.
b- New ischaemic ECG changes.
c- Angiographic findings consistent with a procedural complication.
d- Imaging demonstration of new loss of viable myocardium or new RWMA.
4- Stent thrombosis associated with MI when detected by coronary angiography or autopsy in the setting of myocardial ischaemia and with a rise and/or fall of cardiac biomarker values with at least one value above the 99th percentile URL.
5- Coronary artery bypass grafting (CABG) related MI is arbitrarily defined by elevation of cardiac biomarker values (>10 x 99th percentile URL) in patients with normal baseline cTn values (≤99th percentile URL). In addition, either
a- New pathological Q waves or new LBBB.
b- Angiographic documented new graft or new native coronary artery occlusion.
c- Imaging evidence of new loss of viable myocardium or new RWMA.
Criteria for Healing or Healed Myocardial Infarction: Any one of the following criteria meets the diagnosis for prior MI:
Pathological Q waves with or without symptoms in the absence of non-ischaemic causes.
Imaging evidence of a region of loss of viable myocardium that is thinned and fails to contract, in the absence of a non-ischaemic cause.
Pathological findings of a prior MI.

(Thygesen et al., 2012).