Introduction

Acute myocardial infarction (AMI) remains a leading cause of morbidity and mortality worldwide. It occurs when irreversible myocardial cell damage or death occurs ***(Bolooki and Askar, 2010).***

ST segment elevation myocardial infarction (STEMI) is the most serious presentation of atherosclerotic coronary artery disease (CAD) carrying the most hazardous consequences ***(Tosteson et al., 1996).***

STEMI is caused by occlusion of a major coronary artery and primary percutaneous coronary intervention (PCI) is the preferred reperfusion strategy especially when performed by an experienced team within the shortest possible time from first medical contact (FMC) ***(Van de Werf et al., 2008).***

AMI results in left ventricular (LV) remodeling which manifests as disproportionate thinning and dilation of the myocardium. Post infarction remodeling may occur early within 72 hours or late after 72 hours. Early phase involves expansion of the infarct zone while late phase involves the LV globally with dilatation, cavity shape distortion and hypertrophy of non-infarcted segments ***(Sutton et al., 2000).***

LV remodeling is an important factor in the pathophysiology of advancing heart failure (HF) and several studies support the role of measures of LV remodeling in the clinical investigation of novel HF treatments ***(Kramer et al., 2011).***

Aim of the Work

To evaluate the clinical, coronary angiographic and echocardiographic predictors of LV remodeling 6 months after AMI in patients managed by primary PCI.

Primary PCI for STEMI and Determinants of Success

AMI remains a leading cause of morbidity and mortality worldwide. It occurs when irreversible myocardial cell damage or death occurs ***(Bolooki and Askar, 2010).***

The term AMI should be used when there is an 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 AMI:

Detection of a rise and/or fall of cardiac biomarker values preferably cardiac troponin with at least one value above the 99th percentile upper reference limit (URL) and with at least one of the following:

Symptoms of ischaemia, new or presumed new significant ST-segment or T wave changes or new left bundle branch block (LBBB) , development of pathological Q waves in the electrocardiography (ECG) , imaging evidence of new loss of viable myocardium or new regional wall motion abnormality or identification of an intracoronary thrombus by angiography or autopsy ***(Thygesen et al., 2012).***

Any one of the following criteria meets the diagnosis for prior myocardial infarction (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 or pathological findings of a prior MI ***(Thygesen et al., 2012).***

Universal classification of MI:

Type 1: Spontaneous MI.

Type 2: MI secondary to ischaemic imbalance.

Type 3: MI resulting in death when biomarker values are unavailable.

Type 4a: MI related to PCI Type 4b: MI related to stent thrombosis.

Type 5: MI related to coronary artery bypass grafting (CABG) ***(Thygesen et al., 2012).***

STEMI is the most serious presentation of atherosclerotic CAD carrying the most hazardous consequences ***(Tosteson et al., 1996).***

STEMI is caused by occlusion of a major coronary artery and primary PCI is the preferred reperfusion strategy

especially when performed by an experienced team within the shortest possible time from FMC ***(Van de Werf et al., 2008).***

ST- segment elevation in AMI, measured at the J point, should be found in at least two contiguous leads and be ≥0.25 millivolts (mV) in men below the age of 40 years, ≥0.2 mV in men over the age of 40 years, or ≥0.15 mV in women in leads V2–V3 and/or ≥0.1 mV in other leads in the absence of left ventricular hypertrophy (LVH) or LBBB ***(Thygesen et al., 2012).***

Myocardial necrosis caused by complete coronary artery occlusion begins to develop after 15–30 minutes of severe ischaemia and progresses from the sub-endocardium to the sub-epicardium in a time dependent fashion. Reperfusion, including recruitment of collaterals may save myocardium at risk from undergoing necrosis, and sub-critical but persistent forward flow may extend the time window for achieving myocardial salvage ***(Van de Werf et al., 2008).***

Reperfusion therapy:

Reperfusion therapy is indicated in all patients with symptoms of <12 hours duration and persistent ST-segment elevation or presumed new LBBB ***(Boersma, 2006),*** class I level of evidence A ***(Windecker et al., 2014).***

Reperfusion therapy (preferably primary PCI) is indicated if there is evidence of ongoing ischaemia, even if symptoms may have started >12 hours beforehand or if pain and ECG changes have been stuttering ***(Gierlotka et al., 2011),*** class I level of evidence C ***(Windecker et al., 2014).***

Reperfusion therapy with primary PCI may be considered in stable patients presenting 12–24 hours after symptom onset ***(Ndrepepa et al., 2009),*** class IIb level of evidence B ***(Steg et al., 2012).***

Primary PCI:

Primary PCI is the recommended reperfusion therapy over fibrinolysis if performed by an experienced team within 120 minutes of FMC **(*Cucherat et al., 2003),*** class I level of evidence A ***(Steg et al., 2012).***

It is recommended that primary PCI-capable centers deliver a 24- hour/7-day service and ensure for primary PCI to be performed as fast as possible and at the latest within 60 minutes of hospital arrival, class I level of evidence B ***(Windecker et al., 2014).***

Primary PCI is indicated for patients with severe acute HF or cardiogenic shock due to STEMI independent from time delay of symptom onset, class I level of evidence B ***(Windecker et al., 2014).***

In patients with time from symptom onset >12 hours, primary PCI is indicated in the presence of continuing ischaemia, life threatening arrhythmias or if pain and ECG changes have been stuttering , class I level of evidence C ***(Windecker et al., 2014).***

Routine PCI of a totally occluded artery >24 hours after symptom onset in stable patients without signs of ischaemia (regardless of whether fibrinolysis was given or not) is not recommended ***(Menon et al., 2009)***, class III level of evidence A ***(Windecker et al., 2014).***

Stenting is recommended (over balloon angioplasty alone) for primary PCI ***(Nordmann et al., 2004),*** class I level of evidence A ***(Windecker et al., 2014).***

New-generation drug eluting stents (DES) are recommended over bare metal stents (BMS) in primary PCI ***(Windecker et al., 2014).***

In patients undergoing primary PCI dual antiplatelet therapy with Aspirin, class I level of evidence A, and an adenosine diphosphate receptor blocker is recommended with Prasugrel in Clopidogrel-naive patients, if no history of prior stroke/transient ischaemic attack and age <75, class I level of evidence B or Ticagrelor if not contraindicated, class I level of evidence B or Clopidogrel, if only Prasugrel or Ticagrelor are not available or contraindicated, class I level of evidence B ***(Windecker et al., 2014).***

Anticoagulation is recommended for all patients in addition to antiplatelet therapy during PCI, class I level of evidence A ***(Windecker et al., 2014).*** Bivalirudin is preferred over Heparin and glycoprotein (GP) IIb/IIIa inhibitor. Enoxaparin may be preferred over unfractionated Heparin. Unfractionated Heparin must be used in patients not receiving either Bivalirudin or Enoxaparin ***(Steg et al., 2012).***

Fibrinolytic therapy:

Fibrinolytic therapy is recommended within 12 hours of symptom onset in patients without contraindications if primary PCI cannot be performed by an experienced team within 120 minutes of FMC, class I level of evidence A ***(Steg et al., 2012).***

In patients presenting early (<2 hours after symptom onset) with a large infarct and low bleeding risk, fibrinolysis should be considered if time from FMC to balloon inflation is >90 minutes ***(Pinto et al., 2006),*** class IIa level of evidence B ***(Steg et al., 2012).***

Transfer to a PCI-capable center following fibrinolysis is indicated in all patients after fibrinolysis, class I level of evidence A ***(Steg et al., 2012).*** Coronary angiography with the intent to revascularize the infarct related artery (IRA) is indicated after successful fibrinolysis within 24 hours, class I level of evidence A and optimal

timing of angiography for stable patients after successful fibrinolysis: 3-24 hours, class IIa level of evidence A ***(Windecker et al., 2014)***.

Rescue PCI is indicated immediately when fibrinolysis has failed (<50% ST-segment resolution at 60 minutes) ***(Gershlick et al., 2005),*** class I level of evidence A ***(Steg et al., 2012).***

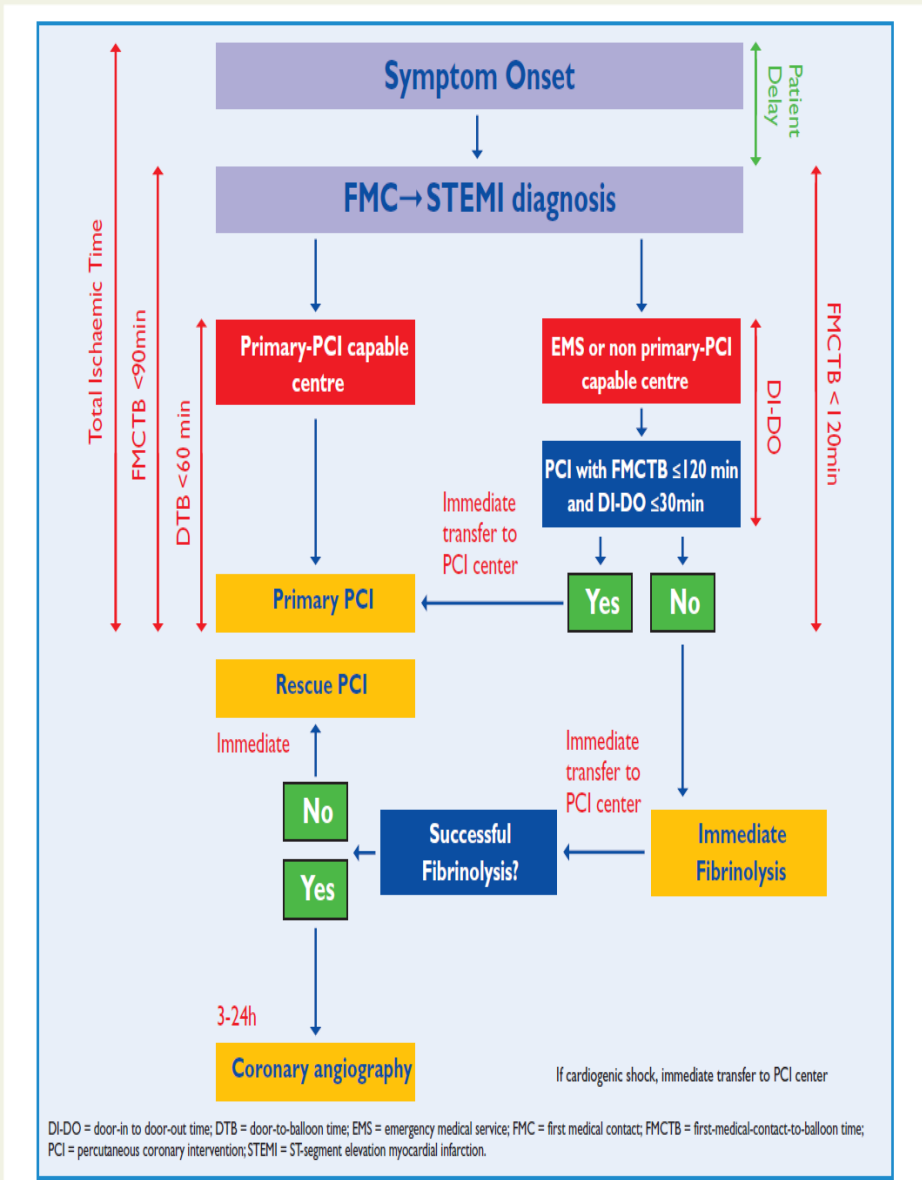

Figure (1): Reperfusion strategies ***(Windecker et al., 2014).***

When PCI is performed in a prompt and expert fashion, it is associated with a low incidence of death and other adverse events and enables early hospital discharge ***(Boersma, 2006).*** Its success relates to more frequent and

durable reperfusion than that obtained with pharmacological therapy alone ***(Brener et al., 2008).***

The predictors and impact of successful angiographic reperfusion, at both the epicardial and myocardial levels, have not been described in patients undergoing primary PCI with contemporary techniques and adjunctive therapy ***(Brener et al., 2008).***

The predictors are classified into:

A. Patient related predictors:

1. Age:

Biological age is a strong determinant of prognosis in patients with AMI ***(Guagliumi et al., 2004).***

Despite contemporary mechanical reperfusion strategies, mortality, major bleeding, and stroke rates remain high in elderly patients undergoing primary PCI, outcomes that are not affected by stents or GP IIb/IIIa inhibitors ***(Guagliumi et al., 2004).***

The overall 30-day mortality and cumulative 6 month mortality together with major adverse cardiac events (MACE) is lower in young population as compared to older groups ***(Chen et al., 2009).***

2. ***Gender:***

Women make up approximately one third of patients undergoing primary PCI for STEMI. Female sex is associated with an apparent hazard of increased mortality among patients undergoing primary PCI for STEMI, but this difference is likely explained by older age and worse baseline comorbidities among women ***(Jackson et al., 2011).***

The efficacy of primary PCI in patients with AMI appears to be gender-dependent. Myocardial salvage achieved by primary PCI is greater in women than in men ***(Mehilli et al., 2005).***

Women have longer ischaemic times compared with men ***(Sjauw, et al., 2010).***

3. ***Diabetes mellitus (DM):***

DM is an independent predictor of worse outcomes after primary PCI for AMI ***(Mathew et al., 2004).***

Despite similar high rates of thrombolysis in myocardial infarction (TIMI) flow grade 3 after primary PCI in patients with and without DM , patients with DM are more likely to have diminished microvascular perfusion ***(Prasad et al., 2005).***

Patients with DM are at high risk for recurrent cardiovascular events after acute coronary syndromes, in part because of increased platelet reactivity. Thus, subjects with DM tend to have a greater reduction in ischaemic events without an observed increase in TIMI major bleeding when treated with Prasugrel compared with Clopidogrel ***(Wiviott et al., 2008)***.

Acute hyperglycemia, but not DM, was a predictor for in-hospital mortality after AMI in the PCI era. No reflow occurred more frequently during PCI in patients with acute hyperglycemia, suggesting that microvascular dysfunction might have contributed to adverse outcomes of these patients ***(Ishihara et al., 2005).***

4. Hypertension:

Several studies reported that a history of hypertension was associated with an increased rate of adverse outcomes after AMI such as stroke, HF and cardiovascular death ***(Richards et al., 2002; Thune et al., 2008).***

In the GISSI-2 study, in-hospital and 6-month mortality in hypertensive AMI patients was significantly higher compared to normotensive patients as was the rate of LV failure, recurrent angina, and recurrent MI ***(Fresco et al., 1996)***.

On the other hand, GUSTO-1 study showed that elevated blood pressure was not an independent prognostic factor for 30-day mortality, but in these trial patients with very high values of blood pressure were excluded due to the thrombolytic treatment. A later sub analysis of the GUSTO-1 trial found a higher risk of early death in patients with elevated systolic blood pressure (SBP) at admission ***(Aylward et al., 1996).***

5. ***Cigarette smoking:***

Current smokers developed STEMI more than 10 years earlier than non-smokers with similar age and sex-adjusted risk of death at 1 year ***(Rakowski et al., 2012).***

Prior studies have found that smokers with AMI have lower mortality rates and a more favorable response to fibrinolytic therapy than non-smokers. This "smoker's paradox" extends to patients undergoing primary PCI for AMI, with increased survival seen in current smokers, an effect entirely explained by differences in baseline risk and not smoking status per se as in those studies smokers were younger, more often men, and less frequently had DM, hypertension, prior AMI, and triple-vessel CAD ***(Weisz et al., 2005).***