INTRODUCTION

Myocardial infarction (MI) can be recognized by clinical features, including electrocardiographic (ECG) findings, elevated values of biomarkers of myocardial necrosis, and by imaging, or may be defined by pathology ***(Thygesen et al., 2012).*** The ECG is an integral part of the diagnosis and prognosis of patients with suspected MI and should be acquired and interpreted promptly after clinical presentation ***(Thygesen et al., 2012).***

The current guidelines for the treatment of ST-segment elevation myocardial infarctions (STEMI) emphasize the importance of shortening the time interval between the occlusion of the infarct-related artery and reperfusion to salvage myocardium and minimize infarct size ***(O'Gara et al., 2013).***

Inversion of the T waves (T-) in the leads with ST-segment elevation early after initiation of reperfusion therapy has been described as a marker of reperfusion and a good prognostic sign ***(Atar et al., 2006)***. However, the significance of T wave invertion on presentation before the initiation of reperfusion therapy and patency of the infracted-related artery (Thrombolysis in Myocardial Infarction {TIMI} Flow Grades) is unclear.

AIM OF THE WORK

The aim of the present study is to evaluate whether T wave inversion in the presenting ECG in patients with anterior STEMI predicts spontaneous reperfusion.

ACUTE MYOCARDIAL INFARCTION

Definition of myocardial infarction

Last updated definition of AMI is the "Universal Definition of Myocardial Infarction" conducted by ***Thygesen et al. (2012)*** on behalf of the Joint ESC/ACC/AHA Task Force for the Redefinition of Myocardial Infarction.

Criteria for Acute Myocardial Infarction:

The term myocardial infarction should be used when there is evidence of myocardial necrosis (myocardial cell death) in a clinical setting consistent with myocardial ischemia. Under these conditions any one of the following criteria meets the diagnosis for myocardial infarction:

- ***Detection of rise and/or fall of cardiac biomarkers*** (preferably troponins) with at least one value above the 99th percentile of the upper reference limit (URL) together with evidence of myocardial ischemia with at least one of the following:
  - Symptoms of ischemia
  - ECG changes indicative of new ischemia (new ST-T changes or new left bundle branch block (LBBB)
  - Development of pathological Q waves in the ECG

- Imaging evidence of new loss of viable myocardium or new regional wall motion abnormality.
- Identification of an intra coronary thrombus by angiography or autopsy.

- ***Sudden, unexpected cardiac death, involving cardiac arrest*** often with symptoms suggestive of myocardial ischemia and accompanied by presumably new ST elevation or new LBBB, and/or evidence of fresh thrombus by coronary angiography and/or autopsy, but death before blood samples are obtained, or before the appearance of cardiac biomarkers in the blood.

- ***For percutaneous coronary interventions (PCI)*** in patients with normal baseline troponin values, elevation of cardiac biomarkers above the 99thpercentile URL are indicative of peri-procedural myocardial necrosis. By convention, increase of biomarkers greater than 3 x 99th percentile URL has been designated as defining PCI-related myocardial infarction.

- ***Stent thrombosis*** documented by coronary angiography or autopsy and in addition, meeting the criteria for spontaneous myocardial infarction.

- ***For coronary artery bypass grafting (CABG)*** in patients with normal baseline troponin values, elevation of cardiac biomarkers above the 99thpercentile URL are indicative of peri-procedural myocardial necrosis. By convention, increase of biomarkers greater than 5 x 99th percentile URL

plus either new pathological Q waves or new LBBB, or angiographically documented new graft or native coronary artery occlusion, or imaging evidence of new loss of viable myocardium have been designated as defining CABG-related myocardial infarction.

Criteria for Prior Myocardial Infarction:

- Any one of the following criteria meets the diagnosis for prior myocardial infarction:

  1) Development of new pathological Q waves with or without symptoms.

  2) Imaging evidence of a region of loss of viable myocardium that is thinned and fails to contract, in the absence of a non-ischemic cause.

  3) Pathological findings of a healed or healing myocardial infarction ***(Thygesen et al., 2007).***

Diagnosis and Management of STEMI

- ***Symptoms:***
  - Chest pain is the most common symptom of acute myocardial infarction and is often described as a sensation of tightness, pressure, or squeezing. It radiates most often to the left arm, but may also radiate to the lower jaw, neck, right arm, back and epigastrium, where it may mimic heartburn. Shortness of breath

(dyspnea) occurs when the damage to the heart limits the output of the left ventricle, causing left ventricular failure and consequent pulmonary edema. Other symptoms include diaphoresis, weakness, light headedness, nausea, vomiting and palpitations**.** Loss of consciousness and even sudden death can occur in myocardial infarction ***(McSweeney et al., 2003)***.

- ***Electrocardiographic Diagnosis:***
  - The ECG is the primary diagnostic tool used to identify STEMI and is of central importance because it identifies those patients who are candidates for emergent reperfusion therapy. The definition of ST elevation indicative of myocardial ischemia, as proposed by the Joint Committee of the European Society of Cardiology (ESC) and American College of Cardiology (ACC) for the redefinition of MI, is a new, or presumed new, ST segment elevation in 2 or more contiguous leads of at least 2mm at the J point in leads V1-V3 or 1mm in other leads ***(Alpert et al., 2000)***.
  - Decision-making is more complicated when a BBB confounds ECG interpretation. Right bundle branch block (RBBB) generally confounds the ECG interpretation of an acute infarct less than does an LBBB; identification of myocardial ischemia in the presence of LBBB is quite difficult. Numerous criteria exist for the diagnosis of AMI in this setting, of which

the most widely accepted are those developed by Sgarbossa (Table 1). The ACC and AHA recommend reperfusion therapy for patients with new or presumably new left bundle branch block (LBBB) ***(Antman et al., 2006)***.

Table (1): Sgarbossa Criteria for Identifying Acute Myocardial Infarction in Left Bundle Branch Block ***(Sgarbossa et al., 1996).***

- ST elevation ≥ 1mm in leads with dominant R wave (concordant with QRS complex) (5 points).
- ST elevation ≥ 5mm in leads with dominant S waves (discordant with QRS complex) (2 points).
- ST depression ≥ 1mm in V1, V2 or V3 (3 points).

A total score of 3 points yields ≥ 90% specificity and more than 88% positive predictive value.

Cardiac Biomarkers:

- Serum cardiac biomarkers (creatinine kinase [CK], CKMB, cardiac-specific troponins and myoglobin) are useful for confirming the diagnosis of MI and estimating infarct size. Serum cardiac biomarkers also provide valuable prognostic information. For patients with ST-segment elevation, the diagnosis of STEMI is secure; initiation of reperfusion therapy should not be delayed while awaiting the results of a cardiac biomarker assay ***(Luepker et al., 2003).***

Management of STEMI

I) Reperfusion

- **Options for Reperfusion Therapy:**
  - Reperfusion of the infarct-related artery (IRA) is the cornerstone of therapy for STEMI. Fibrinolysis and percutaneous coronary intervention (PCI) are both well established as effective options, but PCI has generally come to be regarded as the treatment of choice. A recent meta-analysis of 23 randomized controlled trials (RCTs) comparing PCI to fibrinolysis revealed that PCI reduced short term mortality, non-fatal re-infarction and stroke when compared to fibrinolysis ***(Keeley et al., 2003).***
  - Analysis from the National Registry for Myocardial Infarction (**NRMI** registry), which included hospitals of various sizes with a wide geographic distribution in the US, revealed that while PCI provided a greater mortality benefit than fibrinolysis in centers with an intermediate or high volume of procedures per year, there was no mortality benefit in low volume centers (fewer than 17 procedures per year) ***(Magid et al., 2000).***
- **Pharmacologic Reperfusion:**
  - The development of newer, fibrin-specific fibrinolytics, such as t-PA, reteplase, and tenecteplase, represents a small but significant improvement over the first-

generation drugs (i.e., streptokinase and urokinase). The newer agents have the advantage of activating plasminogen to form the clot-lysing enzyme plasmin when they are bound to fibrin in a thrombus, thereby promoting targeted fibrinolysis rather than systemic anticoagulation, and theoretically improving clot lysis while lowering the risk of bleeding ***(The GUSTO investigators, 1993)***.

- **Prehospital Fibrinolysis:**
  - The benefit of fibrinolysis is greatest when administered early following the onset of symptoms, and declines rapidly after the first several hours ***(Boersma et al., 1996)***. In select settings, prehospital fibrinolysis appears to offer a mortality advantage over in-hospital administration. A meta-analysis of 6 trials with 6,434 patients found a reduction in all-cause hospital mortality (odds ratio 0.83, 95% CI 0.70-0.98) with prehospital fibrinolysis ***(Morrison et al., 2000)***.
  - **Rescue PCI** should be considered when there is evidence of failed fibrinolysis based on clinical signs and insufficient ST-segment resolution (<50%), if there is clinical or ECG evidence of a large infarct, and if the procedure can be performed within a reasonable time delay (up to 12hrs after onset of symptoms) ***(Van de Werf et al., 2008).***

- A more disputed regimen is so-called **facilitated PCI**, which includes full or partial dose fibrinolytics, alone, or in combination with glycoprotein IIb/IIIa (GP IIb/IIIa) inhibitors, started prior to early, planned PCI. Recent trials showed no evidence of a significant clinical benefit with facilitated PCI over primary PCI as shown in ***(Keeley et al., 2006)*** study, **ASSENT-4**, **FINESSE** and **ON-TIME 2** trials ***(The ASSENT-4 Investigators 2006; Ellis et al., 2008 and Van't Hof et al., 2008)***.

- Another reperfusion strategy is **early PCI after fibrinolytic therapy** in high risk patients where routine PCI is employed 3-24hrs after fibrinolysis, rather than as soon as possible, as is the case with facilitated PCI. Early PCI would reduce the need for expensive 24hour catheterization laboratories, and lessen the demand for resource consumption by the emergency department (ED), or catheterization laboratory transfer. This attractive strategy had met the recent evidence in **CARESS-IN-AMI** ***(Di Mario et al., 2008)*** and **TRANSFER- AMI** ***(Cantor et al., 2009)*** trials which showed better clinical outcome without significant increase in rate of major bleeding. So it is recommended by recent ACC/AHA guidelines update as (class IIa level of evidence B) that high-risk patients who received fibrinolytic therapy as primary reperfusion therapy at a non-PCI-capable facility to be transferred as soon as

possible to a PCI-capable facility where PCI can be performed when needed with better outcome ***(Kushner et al., 2009)***.

II) Adjunctive Therapies:

- Reperfusion therapy, while central to the treatment of STEMI, must be accompanied by appropriate adjunctive treatments.

A) Oxygen.

B) Analgesia.

C) Anti-platelet Therapy:

1) Aspirin:
2) Clopidogrel:
3) Glycoprotein IIb/IIIa inhibitors:

D) Anti-thrombin Agents:

E) Other Medications:

- Beta blockers
- ACE-inhibitors
- Nitrates
- Statins

III) Coronary Artery Bypass Surgery:

Emergency coronary artery bypass graft surgery (CABG) is usually undertaken to treat a mechanical complication, such as a ruptured papillary muscle or a ventricular septal defect, with ensuing cardiogenic shock. In uncomplicated MI, the mortality rate can be high when the surgery is performed immediately following the infarction. In patients developing cardiogenic shock after a myocardial infarction, both PCI and CABG are satisfactory treatment options, with similar survival rates ***(Hochman et al., 2006).***

ECG CHANGES IN ANTERIOR MYOCARDIAL INFARCTION

Clinical Relevance

- Anterior STEMI results from occlusion of the left anterior descending artery (LAD).
- Anterior myocardial infarction carries the worst prognosis of all infarct locations, mostly due to larger infarct size.
- In addition to anterior STEMI, other high-risk presentations of anterior ischaemia include left main coronary artery (LMCA) occlusion, Wellens' syndrome and De Winter's T waves.

How to Recognize Anterior STEMI

- ST segment elevation with Q wave formation in the precordial leads (V1-6) ± the high lateral leads (I and aVL).
- Reciprocal ST depression in the inferior leads (mainly III and aVF).

NB: The magnitude of the reciprocal change in the inferior leads is determined by the magnitude of the ST elevation in I and aVL (as these leads are electrically opposite to III and aVF), hence may be minimal or absent in anterior STEMIs that do not involve the high lateral leads.

Patterns of Anterior Infarction

- The nomenclature of anterior infarction can be confusing, with multiple different terms used for the various infarction patterns. The following is a simplified approach to naming the different types of anterior MI.
- The **precordial leads** can be classified as follows:
  1) Septal leads = V1-2
  2) Anterior leads = V3-4
  3) Lateral leads = V5-6
- The different **infarct patterns** are named according to the leads with maximal ST elevation:
  1) Septal = V1-2
  2) Anterior = V2-5
  3) Anteroseptal = V1-4
  4) Anterolateral = V3-6, I + aVL
  5) Extensive anterior / anterolateral = V1-6, I + aVL

(NB: While these definitions are intuitive, there is often a poor correlation between ECG features and precise infarct location as determined by imaging or autopsy. For an alternative approach to the naming of myocardial infarctions, take a look at this 2006 article from Circulation).

- Three other important ECG patterns to be aware of:

1) **Anterior-inferior STEMI due to occlusion of a "wraparound" LAD**: simultaneous ST elevation in the precordial and inferior leads due to occlusion of a variant ("type III") LAD that wraps around the cardiac apex to supply both the anterior and inferior walls of the left ventricle.

2) **Left main coronary artery occlusion**: widespread ST depression with ST elevation in aVR ≥ V1

3) **Wellens' syndrome**: deep precordial T wave inversions or biphasic T waves in V2-3, indicating critical proximal LAD stenosis (a warning sign of imminent anterior infarction).

4) **De Winter's T waves**: upsloping ST depression with symmetrically peaked T waves in the precordial leads; a "STEMI equivalent" indicating acute LAD occlusion.