Terminal QRS Distortion as a predictor of Inhospital Outcome In patients with Acute ST Elevation Myocardial Infarction

Thesis

Submitted for the partial fulfillment of the master degree

in Cardiology

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2011

إنحراف المركب الطرفى **QRS** كمؤشر على النتائج داخل المستشفى للمرضى المصابين باحتشاء عضلة القلب الحاد الناتج عن ارتفاع الفلقة **ST**

رسالة

توطئة للحصول علي درجة الماجيستير

في **أمراض القلب**

مقدمة من

الطبيبة/ مـرمـر فخـرى إبراهيـم

بكالوريوس الطب والجراحة

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List of Abbreviations

AMI	: Acute myocardial infarction
CABG	: Coronary artery bypass grafting
CHD	: Coronary heart disease
CHF	: Congestive heart failure
CK	: Creatine Kinase
CK-MB	: Creatine Kinase MB
CSA	: Chronic stable angina
IHD	: Ischemic heart disease
IS	: Infarct size
LBBB	: Left bundle branch block
LVEF	: Left ventricular ejection fraction
NSTEMI	: Non ST- segment elevation myocardial infarction
PCI	: Percutaneous coronary intervention
PDFCF	: pressure derived fractional collateral flow
SPECT	: Single Photon Emission Computed Tomography
WMSI	: Wall motion score index

Introduction

Electrocardiography (ECG) is a noninvasive method that can be recorded easily at bedside. Its diagnostic role in acute myocardial infarction (AMI) is well known. ECG is useful to clinicians for deciding whether thrombolytic therapy should be given to patients with suspected AMI on admission [1]. In addition, rapid ST segment resolution is a reliable ECG indicator of reperfusion [2].

A major determinant of short- and long-term prognosis in acute myocardial infarction (MI) is the size of the myocardial area at risk for necrosis. During the past two decades, It has become increasingly well acknowledged that a large, transmural acute myocardial infarction (AMI) may result in complex alterations in the architecture and function of the left ventricle (LV), involving both the infracted and non–infracted zone. These alterations, usually referred as "LV remodeling", can profoundly affect the patient's prognosis [3].

Several studies were carried out to estimate the ischemic area at risk or predict final infarct size or assess prognosis with the admission ECG, mainly by using the number of leads with ST deviation (elevation and/or depression) or the absolute amplitude of ST deviation [3-6].

All these studies were based on the assumption that each lead represents the same amount of myocardium and that a

similar size of ischemic area in different segments of the LV will result in similar magnitude of ST deviation in the same number of leads. However, not all myocardial regions are equally represented by the 12-lead ECG. Furthermore, involvement of opposed regions may cancel or augment ST deviation [7].

Recently, Birnbaum et al described an alternative simple method for predicting both prognosis and final infarct size from the initial pattern of the admission ECG [7,8]. In this method, patients with ST elevation and positive T waves in the leads with ST elevation are divided into two groups, patients with terminal QRS distortion in 2 or more adjacent leads (no S waves in leads with a usual rS morphology [V1 through V3]., and/or a J/R wave ratio of ≥ 50% in leads with qR morphology) have higher mortality, larger final infarct size, less salvage by thrombolytic therapy, and a more rapid progression of necrosis over time. The mechanisms causing these differences between patients with and without terminal QRS distortion are not clear, but may be related to collateral circulation or ischemic preconditioning [9]. Most studies which studied the terminal QRS distortion were applied only in patients with anterior myocardial infarction [1,10]. Only one study included both anterior and inferior myocardial infarction but it didn't discriminate between both groups in the analysis [11].

Aim of the Work

The aim of the study is to assess the value of the terminal QRS distortion in admission ECG as a simple predictor of inhospital outcome in patients with acute Anterior or Inferior ST elevation myocardial infarction.

Acute Myocardial Infarction

Acute myocardial infarction (AMI), commonly known as a heart attack, is the interruption of blood supply to part of the heart, causing heart cells to die. This is most commonly due to occlusion of a coronary artery following the rupture of a vulnerable atherosclerotic plaque, which is an unstable collection of lipids (fatty acids) and white blood cells (especially macrophages) in the wall of an artery. The resulting ischemia and oxygen shortage, if left untreated for a sufficient period of time, can cause damage or death of the myocardium[16].

Myocardial infarction is now considered part of a spectrum referred to as acute coronary syndrome, which refers to a range of acute myocardial ischaemia that also includes unstable angina and non-ST segment elevation myocardial infarction (NSTEMI). The new criteria for diagnosing myocardial infarction are detection of rise and/or fall of cardiac biomarkers (preferably troponin) with at least one value above the 99th percentile of the upper reference limit, together with evidence of myocardial ischaemia with at least one of the following:

- Symptoms of ischaemia
- Electrocardiogram (ECG) changes indicative of new ischaemia (new ST-T changes or new left bundle branch block (LBBB))

- Development of pathological Q wave changes in the ECG
- Imaging evidence of new loss of viable myocardium or new regional wall motion abnormality [17].

Acute myocardial infarction (MI) is defined as death or necrosis of myocardial cells. It is a diagnosis at the end of the spectrum of myocardial ischemia or acute coronary syndromes. Myocardial infarction occurs when myocardial ischemia exceeds a critical threshold and overwhelms myocardial cellular repair mechanisms designed to maintain normal operating function and hemostasis. Ischemia at this critical threshold level for an extended period results in irreversible myocardial cell damage or death [18].

Critical myocardial ischemia may occur as a result of increased myocardial metabolic demand, decreased delivery of oxygen and nutrients to the myocardium via the coronary circulation, or both. An interruption in the supply of myocardial oxygen and nutrients occurs when a thrombus is superimposed on an ulcerated or unstable atherosclerotic plaque and results in coronary occlusion. A high-grade (more than 75%) fixed coronary artery stenosis caused by atherosclerosis or a dynamic stenosis associated with coronary vasospasm can also limit the supply of oxygen and nutrients and precipitate an MI [18].

Classification:

There are two basic types of acute myocardial infarction:

- **Transmural**: associated with atherosclerosis involving major coronary artery. It can be subclassified into anterior, posterior, or inferior. Transmural infarcts extend through the whole thickness of the heart muscle and are usually a result of complete occlusion of the area's blood supply [19].
- **Subendocardial**: involving a small area in the subendocardial wall of the left ventricle, ventricular septum, or papillary muscles. Subendocardial infarcts are thought to be a result of locally decreased blood supply, possibly from a narrowing of the coronary arteries. The subendocardial area is farthest from the heart's blood supply and is more susceptible to this type of pathology [19].

Clinically, a myocardial infarction can be further subclassified into a ST elevation MI (STEMI) versus a non-ST elevation MI (non-STEMI) based on ECG changes [20].

A 2007 consensus document classifies myocardial infarction into five main types:

- Type 1 - Spontaneous myocardial infarction related to ischaemia due to a primary coronary event such as plaque erosion and/or rupture, fissuring, or dissection

- Type 2 - Myocardial infarction secondary to ischaemia due to either increased oxygen demand or decreased supply, e.g. coronary artery spasm, coronary embolism, anaemia, arrhythmias, hypertension, or hypotension

- Type 3 - Sudden unexpected cardiac death, including cardiac arrest, often with symptoms suggestive of myocardial ischaemia, accompanied by presumably new ST elevation, or new LBBB, or evidence of fresh thrombus in a coronary artery by angiography and/or at autopsy, but death occurring before blood samples could be obtained, or at a time before the appearance of cardiac biomarkers in the blood

- Type 4 - Associated with coronary angioplasty or stents:
  - Type 4a - Myocardial infarction associated with PCI
  - Type 4b - Myocardial infarction associated with stent thrombosis as documented by angiography or at autopsy

- Type 5 - Myocardial infarction associated with CABG [21].

Myocardial infarction can be subcategorized on the basis of anatomic, morphologic, and diagnostic clinical information. From an anatomic or morphologic standpoint, the two types of MI are transmural and nontransmural. A transmural MI is characterized by ischemic necrosis of the full thickness of the affected muscle segments, extending from the endocardium

through the myocardium to the epicardium. A nontransmural MI is defined as an area of ischemic necrosis that does not extend through the full thickness of myocardial wall segments. In a nontransmural MI, the area of ischemic necrosis is limited to the endocardium or endocardium and myocardium. It is the endocardial and subendocardial zones of the myocardial wall segment that are the least perfused regions of the heart and the most vulnerable to conditions of ischemia. An older subclassification of MI, based on clinical diagnostic criteria, is determined by the presence or absence of Q waves on an electrocardiogram (ECG). However, the presence or absence of Q waves does not distinguish a transmural from a nontransmural MI, as determined by pathology [18].

A more common clinical diagnostic classification scheme is also based on electrocardiographic findings as a means of distinguishing between two types of MI, one that is marked by ST elevation (STEMI) and one that is not (NSTEMI). Management practice guidelines often distinguish between STEMI and non-STEMI, as do many of the studies on which recommendations are based. The distinction between an STEMI and NSTEMI also does not distinguish a transmural from a nontransmural MI. The presence of Q waves or ST-segment elevation is associated with higher early mortality and morbidity; however, the absence of these two findings does not confer better long-term mortality and morbidity [18].

Epidemiology [22].

- Coronary heart disease (CHD) is the most common cause of death in the UK. CHD is responsible for the deaths of approximately one in five men, and one in six women.
- The average incidence of myocardial infarction for those aged between 30 and 69 years is about 600 per 100,000 for men, and 200 per 100,000 for women.
- Mortality rates from CHD are higher for men than women, people living in deprived areas and in people of South Asian origin. There is evidence of earlier deaths for men than women after an acute myocardial infarction [23].
- Pre-menopausal women appear to be protected from atherosclerosis.
- Incidence increases with age and elderly people also tend to have higher rates of morbidity and mortality from their infarcts.
- Incidence rates of myocardial infarction are lower in the South of England compared with the North of England, Scotland and Northern Ireland [23].

Prevalence:

Myocardial infarction is the leading cause of death in the United States and in most industrialized nations throughout the world. Approximately 800,000 people in the United States are