Acute Myocardial Infarction entailing ST segment elevation in aVL: Electrocardiographic differentiation among occlusion of Left Anterior Descending, First Diagonal and First Obtuse Marginal arteries

Thesis
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By

Mohammad Abdull-Latif Amir

Supervised by

Pr. Dr. Ihab M. Attia
Professor of Cardiology, Ain Shams University

Dr. Mohammad D. Dardeer
Consultant of Cardiology, National Heart Institute

Dr. Azza A. El Fiky
Lecturer of Cardiology, Ain Shams University

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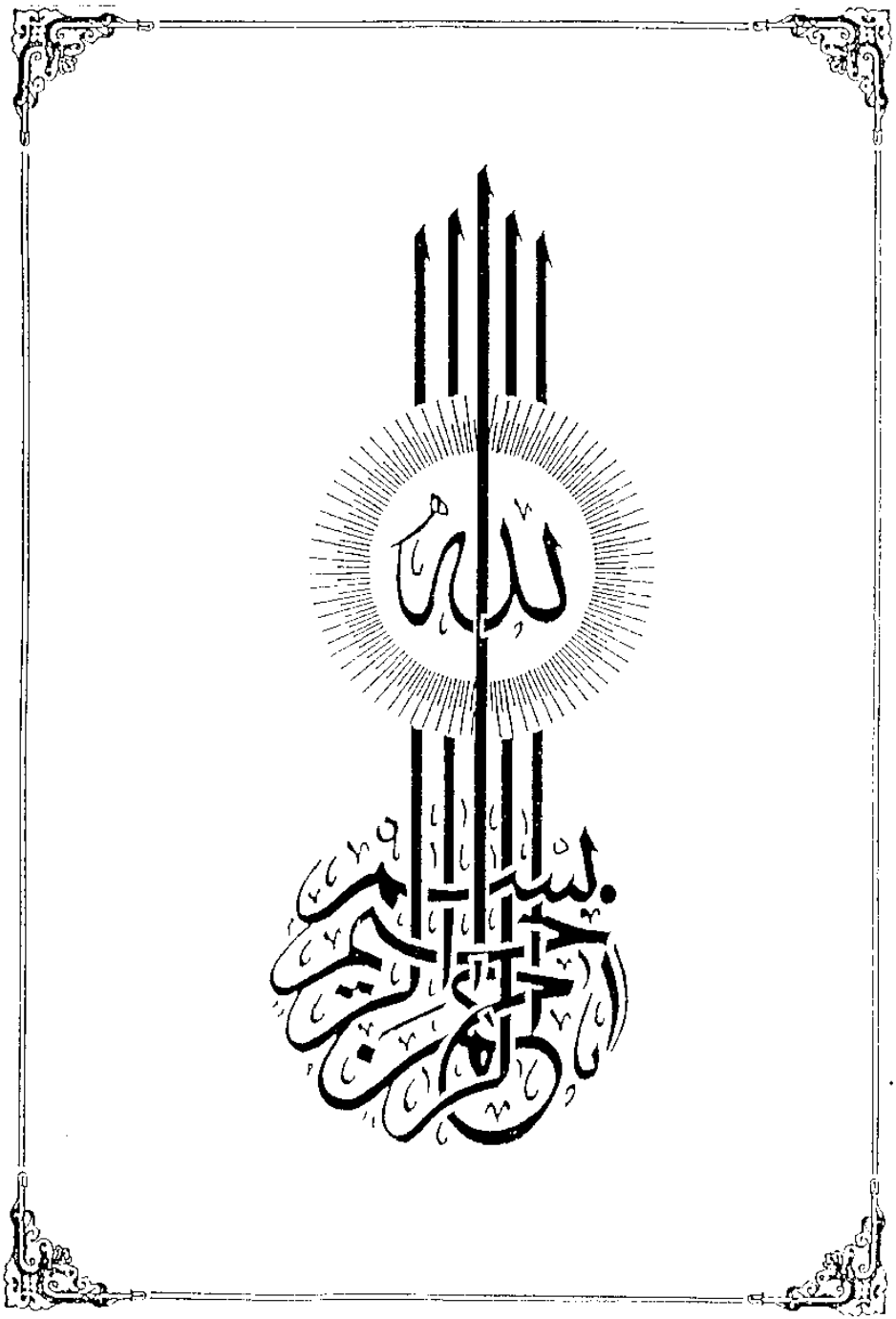

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INTRODUCTION

It is frequently difficult to determine the infarct related artery during coronary angiography, because of the presence of more than one lesion and also because of the large individual variation in the number, size and location of the branches originating from the three main coronary arteries. So, it would be helpful in the clinical setting if the initial electrocardiogram (ECG) with acute myocardial infarction (AMI) can specify the infarct-related artery (IRA) by a characteristic ECG picture.

Lead aVL was studied in multiple studies trying to localize the coronary artery responsible for its changes (1-4). Lead aVL was found to represent myocardium perfused by the diagonal branch of the left anterior descending (LAD) artery (1,2). However, ST segment elevation in lead aVL may be found in some AMIs caused by the left circumflex (LCx) artery occlusion (5,6). The ECG pattern accompanying ST segment elevation in lead aVL from LCx. occlusion is still not well characterized.

This study postulates that, by characterizing a unique ECG pattern associated with occlusion of a particular coronary artery, it may be possible to predict the site of the culprit lesion in the clinical setting.

Aim of the Study

This study assesses patients with AMI with ST segment elevation in lead aVL. The patterns of ST segment deviation in the various ECG leads will be analyzed to characterize a unique ECG pattern related to occlusion of each coronary vessel.

REVIEW OF LITERATURE

Chapter I

The electrocardiogram of acute myocardial infarction

DEFINITION OF EVOLVING MYOCARDIAL INFARCTION

An evolving myocardial infarction is said to be present when a patient develops chest discomfort, usually, but not always, lasting longer than 20 minutes, believed to be due to myocardial ischemia. When the chest discomfort due to myocardial ischemia lasts longer than 4 to 6 hours, the infarction should be considered completed rather than evolving (7).

BASIC ELECTROCARDIOGRAPHIC MANIFESTATIONS OF MYOCARDIAL INFARCTION:

The evolution of AMI, in an electrocardiographic sense, can be divided into 3 principles:

1-The early hyperacute phase.

2-The fully evolved acute phase.

3-The chronic stabilized phase.

It must, however, be emphasized that, although the classic features of every phase are easily recognized, each blends almost imperceptibly with the other, so that it is difficult to establish clear-cut separation (8).

1-Early hyperacute injury phase:

This phase is characterized by:

A-Acute injury block.

B-Slope elevation of the ST segment.

C-An increase in the T wave magnitude.

A- **Acute injury block:** (Figure1 diagram C)

Acute injury is associated with a delay in conduction and the process of depolarization through the injured zone, and has the following characteristics:

1- Relatively slow inscription of the R wave resulting in a delay in the intrinsicoid deflection to 0.45 seconds or longer, an increased ventricular activation time.

2-There may be an increase in the duration of the QRS which may be 0.12 seconds.

3- There is frequently an increase in the amplitude of the QRS. This is because the delayed activation front is no longer balanced or neutralized by the earlier activation of distant healthy regions of the ventricular myocardium.

4-The manifestations appear early in the evolution of AMI, during the hyperacute phase, i.e.:

- Before development of pathological abnormal Q waves.
- During the phase of hyperacute injury as reflected by marked slope elevation of the ST segment.
- Before inversion of the T waves.

5-The manifestations are usually of short duration and disappear when the manifestations of necrosis (pathological Q waves) and ischemia (deeply inverted T waves) appear.

Injury block is a constant feature during the hyperacute injury phase of AMI. It may be missed because it occurs transiently.

B- **Slope elevation of the ST segment:**

ST segment is that portion of ECG, which extends from the end of QRS complex (J point) to the beginning of the T wave. This portion is normally isoelectric, but it may vary from -0.55 to +2.0 mm below or above the baseline. This baseline can be the TP or the PR segments (9) (Figure 2). The first ECG sign of AMI is frequently a straightening of the normal upward concavity of the ST segment, which normally merges imperceptibly with the proximal limb of the T wave, so that the two cannot be separated. (Diagram A Figure 1) The earliest evidence of AMI may be loss of this upward concavity with straightening or ironing of the ST segment in leads oriented to the epicardial surface. (Diagram B Figure 1). This change results in an indirect concomitant widening of the T wave, which becomes absorbed into the straightened ST segment. This is the earliest sign of ST elevation (8).

Further evolution of the MI results in manifest elevation of ST segment (Diagram C Figure 1). The T wave remains upright and thus appears to be more wide and may also increase in magnitude. During this stage of evolution, the ST segment usually has an upward shape, since the proximal part is less than the distal part, which merges with the T wave. This will be termed slope elevation of the ST segment.

Two concepts based on systolic and diastolic phenomena have been suggested to explain the ST- segment displacement. One postulates local reduction or loss of resting potential, resulting in a diastolic current of injury. The second concept assumes an unopposed current flowing from the injured area during the isoelectric ST segment, resulting in a systolic current of injury. These systolic and diastolic phenomena cannot be differentiated with the ordinary clinical alternating - current (AC) ECG but can be recorded experimentally with direct -current (DC) equipment. (Figure 3).

The concept of the diastolic current of injury proposes that localized injury is associated with a flow of current from the uninjured to the injured area. As a result, the T-Q segment is displaced downward but is automatically shifted to control level by the capacitor - coupled amplifier of the ECG. When the entire heart (including the injured area) is depolarized, the ST segment is elevated with respect to the depressed but rectified (isoelectric) diastolic T-Q segment (Figure 4).

The concept of the systolic current of injury proposes that during the ST segment, the normal heart is depolarized, but the injured area undergoes early repolarization. The result is a current flow from the more positive, injured area to a more negative, uninjured area. The result is true elevation of the ST segment. Similarly, if, rather than repolarizing early , the injured area fails to depolarize with the normal myocardium, a current of injury would exist and an elevated ST segment would be recorded (Figure 3) .(10).

Earlier experimental studies indicate that during injury both systolic and diastolic currents are present (11), and at times the systolic precedes the diastolic current of injury. Subsequent studies, however, indicate that the diastolic current predominates while the systolic current plays a lesser role and that the magnitude of the current is modified by the heart rate (12) (Figure 3). As indicated, the clinical ECG does not differentiate between systolic and diastolic currents of injury. Furthermore, unless the onset of injury is recorded, even a DC - coupled ECG would not identify the mechanism of the ST- segment shift.

An electrode facing subendocardial injury registers an elevated ST segment, while an epicardial electrode subtended by the normal myocardium registers ST segment depression. Similarly, an electrode facing epicardial injury registers elevation of the ST segment, while the endocardial electrode inscribes ST segment depression (10).

At times, the ST segment may be markedly elevated without the associated straightening, leading to an elevated ST with upward concavity.(Diagram D Figure1). (8). The magnitude of the ST segment elevation may in the early hyperacute injury be considerable reaching amplitudes of 10-15 mm especially in the mid precordial leads. It may, under this circumstance, dominate the entire ECG presentation, presenting as a single positive phase of deflection (monophasic curve). (8).

ST segment and estimation of infarct size

The ST segment deviation was used to estimate the infarct size (13-19). One of these trials (13) correlated between the sum of ST elevation in millimeters, number of leads with ST elevation, and the infarct size using the QRS Selvester scoring system (20) in anterior and inferior infarctions. They found that, in anterior infarction, the number of leads with ST segment elevation was a more important variable than the sum of ST elevation for prediction of AMI size. On the other hand, the sum of ST elevation was a more important factor than number of leads with ST segment elevation for prediction of inferior myocardial infarction size.

The relation between initial ST segment elevation and final infarct size was found to be influenced by the infarct -related artery (IRA) patency in a study by Hackworthy et al. (18) who found that, in patients with anterior infarction and subtotal occlusion of the IRA, infarct size was smaller than predicted from the extent of initial ST segment elevation, and in patients with a persistently occluded IRA without collaterals, the final infarct size correlated with the extent of initial peak ST elevation.

ST segment as an indication of reperfusion after thrombolytic therapy

In a study by Dardir et al (21) the electrocardiographic changes in 30 patients with acute myocardial infarction who received early (<6hours) streptokinase (SK), were evaluated. Percentage of reduction in ST segment height in the lead showing the greatest ST segment elevation, and summation of ST segment area (area above the iso-electric line from the J point to the

end of the T wave) in leads showing ST segment elevation were followed up in the ECGs done immediately after SK infusion, one hour and 3 hours later, ST segment measurements showed significant progressive reduction in follow up ECGs in comparison to the reference pre-treatment one.

Changes in ST segment were related to the coronary artery patency estimated by the TIMI grade obtained from coronary angiography done pre discharge for every patient in this study. According to the results of the coronary angiography, patients were divided into two groups, those with patent IRA and those with occluded IRA.

Patent IRA group had a significantly higher fractional change (percentage of reduction) in ST segment height and ST area than occluded IRA group. The study concluded that resting 12 lead ECG is a good method for predicting coronary artery patency after thrombolytic treatment. Simple measurement of ST segment height changes in a single lead showing maximum elevation, at 3 hours post treatment is a good predictor of coronary patency. If the information is needed earlier than 3 hours, then summation of ST segment area is a good marker for patency. Early evaluation of Q and R waves has no value in predicting coronary artery patency.

C - An increase in the T wave magnitude:

The early hyperacute injury phase of MI is also characterized by an increase in the magnitude of T waves (22). This may be the earliest manifestation and may appear before ST segment changes, particularly in anterior wall MI (22). The classical pathological Q waves of necrosis do not appear until these large T waves have regressed (2).

RECIPROCAL CHANGES:

The ST segments appear reciprocally negative in ECG leads with their positive poles oriented away from the involved area of myocardium.(22).In a study by Quyyumi et al (23). ST segment depression remote from the region of acute myocardial infarction was investigated in