SENSITIVITY OF ECG SIGNS OF MYOCARDIAL SCARING – COMPARISON WITH MPI SPECT

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Abstract: Aim of the study was to compare the sensitivity and specificity of the ECG sign of myocardial necrosis – sequel Q, by using MPI SPECT study findings as a gold standard. All patients included in the study had fixed perfusion defect on the MPI SPECT study. We analyzed patients characteristics: gender, age, presence of significant Q wave on ECG (determined as >40 msec length and amplitude of >1/3 of accompanying R wave), present in at least two consecutive ECG leads, presence of fixed perfusion defect on MPI SPECT study, corresponding to the vascular territory. Data collection was made from patients medical records, ECG recording, and MPI SPECT study performed as one day rest/stress ECG gated study with Tc99m sestamibi, using Dipyridamole as vasodilating stressor. Significant Q-wave was recorded in 13 (41.9%) subjects, 5 in ECG leads corresponding to the inferior wall, 4 to the anterior wall, 3 both the inferior and the anterior wall, and 1 in the inferolateral wall. Only 41.9% of patients with myocardial infarction (detected with MPI SPECT study), had ECG sign of sequela, which indicates that ECG signs of myocardial scaring as a result of myocardial infarction are quite insensitive, as proven with concomitant performing of MPI SPECT study.

1. INTRODUCTION

Heart Failure affects nearly 6 million Americans [6]. Roughly 670,000 people are diagnosed with heart failure each year [2]. It is caused by many conditions that damage the heart muscle, including: coronary artery disease, heart attack, and cardiomyopathy.

Coronary artery disease is the leading cause of death worldwide. It is on the rise and has become a true pandemic that respects no borders. 3, 8 million men and 3.4 million women worldwide die each year from CAD [8]. CAD happens when the arteries that supply blood to the heart muscle become hardened and narrowed. This is due to the buildup of cholesterol and other material, called plaque, on their inner walls. This buildup is called atherosclerosis. As it grows, less blood can flow through the arteries. As a result, the heart muscle can't get the blood or oxygen it needs. This can lead to chest pain (angina) or a heart attack. Most heart attacks happen when a blood clot suddenly cuts off the hearts' blood supply, causing permanent heart damage.

ACUTE MYOCARDIAL INFARCTIONS IS DIAGNOSED WITH

- **ECG :** Acute myocardial infarction (MI) affects both ventricular depolarization (appearance of pathological Q waves) and repolarization (ST-T wave changes). Specific manifestations depend on whether the lesion is subendocardial or transmural in location. The ECG sign of subendocardial ischemia is ST segment depression (A). Depression is reversible if ischemia is only transient but depression persists if ischemia is severe enough to produce infarction. T wave inversion with or without ST segment depression (B) is sometimes seen but not ST segment elevation or Q wave. That is why subendocardial infarction is also called non-ST-elevation myocardial infarction (NSTEMI) and less commonly non-Q wave myocardial infarction.
- **Blood tests:** Blood is drawn to measure levels of cardiac enzymes that indicate heart muscle damage. Levels of troponins (Three subunits have been identified: troponin I (TnI), troponin T (TnT), and troponin C (TnC). The genes that encode for the skeletal and cardiac isoforms of TnC are identical; thus, no structural difference exists between them. However, the skeletal and cardiac subforms for TnI and troponin TnT are distinct, and immunoassays have been designed to differentiate between them.), CK-MB(The criterion most commonly used for the diagnosis of acute MI was 2 serial elevations above the diagnostic cutoff level or a single result more than twice the upper limit of normal. Although CK-MB is more concentrated in the myocardium, it also exists in skeletal muscle and false-positive elevations occur in a number of clinical settings, including trauma, heavy exertion, and myopathy) and LDH are measured.
- **Echocardiogram** is a standard tool in the management of patients with acute myocardial infarction (MI). The role of echocardiography in establishing the diagnosis, location, and extent of MI, in diagnosing

mechanical complications of infarction, and in providing prognostic information that is important for risk stratification will be reviewed.

- **Cardiac catheterization:** Cardiac catheterization, also called cardiac cath, may be used during the first hours of a heart attack if medications are not relieving the ischemia or symptoms. The cardiac cath can be used to directly visualize the blocked artery and help determine which procedure is needed to treat the blockage.

ECG has remained the most widely used laboratory technique for the evaluation of the patient with known or suspected heart disease. This position is explained by its wide applicability and its role as a safe and cheap tool for initial screening. ECG criteria are frequently insensitive and/or nonspecific.

DIAGNOSIS OF MYOCARDIAL SCARING

ECG- Pathologic Q waves are a sign of previous myocardial infarction.

They are the result of absence of electrical activity. A myocardial infarction can be thought of as an elecrical 'hole' as scar tissue is electrically dead and therefore results in pathologic Q waves. Pathologic Q waves are not an early sign of myocardial infarction, but generally take several hours to days to develop. Once pathologic Q waves have developed they rarely go away. However, if the myocardial infarction is reperfused early (e.g. as a result of percutaneous coronary intervention) stunned myocardial tissue can recover and pathologic Q waves disappear. In all other situations they usually persist indefinitely.

Definition of a pathologic Q wave

1. Any Q-wave in leads V2–V3 ≥ 0.02 s or QS complex in leads V2 and V3
2. Q-wave ≥ 0.03 s and > 0.1 mV deep or QS complex in leads I, II, aVL, aVF, or V4–V6 in any two leads of a contiguous lead grouping (I, aVL,V6; V4–V6; II, III, and aVF)
3. R-wave ≥ 0.04 s in V1–V2 and R/S ≥ 1 with a concordant positive T-wave in the absence of a conduction defect.

- **MPI SPECT**- Assessment of viable myocardium in a specific coronary artery

distribution after a heart attack: Stress images in SPECT may help determine the degree of inducible ischemia or viable myocardium that are amenable to revascularization. The value of SPECT in the diagnosis of CAD was confirmed in a meta-analysis [5], which demonstrated it to be highly effective in assessing myocardial perfusion with a quoted sensitivity of 86%, specificity of 74%, and a normalcy rate of 89%. The rate of normal perfusion scans in patients with a low likelihood of CAD.

- **Echocardiography**- Areas of abnormal regional wall motion are almost

universally observed in patients with MI; the degree of wall-motion abnormality may be categorized by use of a semiquantitative wall-motion score. Of note, infarcts may be missed during echocardiography when the infarction is small or when it involves just the apex.

- **MRI** enables direct visualization of the myocardium with excellent

delineation of the epicardial and endocardial interfaces [10]. As a consequence, it may be used to accurately define segmental wall thinning indicative of previous MI. In some patients with a clinical history of transmural infarction, residual myocardium may be demonstrated at the site of the infarction. In other patients, MRI shows virtually complete replacement of muscle by scar.

Pathological Q wave on a 12-lead ECG is a well-known marker for detecting previous MI with certain limitations: Firstly, Q-waves do not always appear on post-MI ECG and can regress or even disappear over time in 25–60% of patients [4]. Secondly, the Q-wave to non-Q-wave MI ratio drastically declines in response to immediate and aggressive therapeutic interventions [4]. Finally, MI, particularly in the posterolateral segment, does not always manifest as Q wave [9]. Q waves can occur in other clinical situations besides MI. Therefore, although pathological Q waves on 12-lead ECG are indicative of abnormal cardiac electrophysiology, they cannot be specifically inferred as irreversible myocardial damage. Indeed, certain reports have not found Q waves to be of any practical use in MI detection in two thirds of cases [7, 1].

Recent studies suggested that MPI SPECT diagnosis is the gold standard for diagnosing MI.

2. AIM OF THE STUDY

To compare the sensitivity and specificity of the ECG sign of myocardial necrosis- sequel Q, by using MPI SPECT study findings as a gold standard.

3. MATERIALS AND METHODS

Retrospective study conducted from March to May 2016 that consecutively included patients with old myocardial infarction and fixed perfusion defect on SPECT MPI.

Following a thorough medical history, MPI SPECT study was performed as one day rest/stress ECG gated study with Tc99m sestamibi, using Dipyridamole as vasodilatating stressor. For the rest portion patients were injected with Inject with 10 mCi of radiopharmaceutical 60–90 minutes prior to imaging. Upon completion of rest imaging and review for quality approval, the patients were prepared for stress testing.

After 2-4 hours patient underwent pharmacologic stress with dipyridamole. The radiopharmaceutical 25-30mCi was injected at peak stress and imaging began after 45 minutes of application.

In the next step, the patients' 12-lead resting ECGs were evaluated for the of significant Q wave on ECG (determined as >40 msec length and amplitude of >1/3 of accompanying R wave), present in at least two consecutive ECG leads.

Also patients' characteristic such as gender and age were analyzed.

Statistical analysis:

Descriptive and comparative statistics, by using the Students t-test and Chi-square test (for continuous and categorical variables). Significance was determined at level of 0.05.

4. RESULTS

31 patients were included in the study, 11 (35,5%) women and 20 (64,5%) men, at mean age of 60.9±10.1 years, mean age for men 59.9 years, mean age for women 62.5 years (p=ns). Significant Q-wave was recorded in 13 (41.9%) subjects, 5 in ECG leads corresponding to the inferior wall, 4 to the anterior wall, 3 both the inferior and the anterior wall, and 1 in the inferolateral wall.

All patients included in the study had fixed perfusion defect on the MPI SPECT study. In 12 patients the scaring was in the inferior wall (38.6%), in 6 (19.4%) on the infero-lateral wall, in 5 (16.1%) on inferior wall, 3 (9.7%) patients had apical scaring, 3 (9, 7%) in anterior and inferior wall, and in the anterolateral wall in 2 (6.5%) patients.

Only 41.9% of patients with myocardial infarction (detected with MPI SPECT study), had ECG sign of myocardial scaring.

5. CONCLUSION

ECG signs of myocardial scaring as a result of myocardial infarction are quite insensitive, as proven with concomitant performing of MPI SPECT study.

This being the first study with such a subject in the Republic of Macedonia has inspired us as authors to continue to work and take a deeper look in this matter and broaden the study so it can be published with applicable number of patients.

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