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The role of ECG in localizing culprit vessel occlusion in acute ST segment elevation myocardial infarction with echocardiographic correlation
ECG and echo in conjunction for better management of STEMI

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ABSTRACT

Objective: Characteristic changes on electrocardiogram (ECG) along with left ventricular ejection fraction (LVEF) and regional wall motion abnormality (RWMA) detected by 2D-Echo helps in diagnosis and prognostication of myocardial infarction (MI). Hence, this study was undertaken to localize the site of infarction in acute ST segment elevation myocardial infarction (STEMI) by ECG and correlate it with RWMA on 2D-Echo.

Methods: This was a prospective study on 100 consecutive selected patients and detailed clinical history was taken. On the basis of admission ECG, patients were broadly classified into three subgroups i.e. anterior wall myocardial infarction (AWMI), inferior wall myocardial infarction (IWMI), anterior plus inferior wall MI. 2D-Echo was done and the culprit vessel localized on the ECG was thereafter correlated with the RWMA on 2D-Echo.

Results: The localization of culprit vessel territory on ECG broadly correlated with RWMA on 2D-Echo. Echo could elaborate the territory of infarction in greater detail than ECG. The Sensitivity, Specificity, Positive Predictive Value and Negative Predictive Value (PPV and NPV) of ECG compared to 2D-Echo in LAD territory were 96.6%, 85.3%, 90.5% and 94.6% and in RCA territory it was 93.3%, 92.8%, 84.8% and 97% and for LCx it was 50%, 98.9%, 75% and 96.8% respectively.

Conclusions: ECG proved to be relatively sensitive and specific investigation for diagnosis of acute STEMI. 2D-Echo supplements and supports ECG by elaborating the regions involved in MI. Therefore both ECG and Echo should be used in conjunction for better assessment of prognosis and management of the patient.

1. Introduction

Over the past decade, cardiovascular disease (CVD) has emerged as the single most important cause of death worldwide. In 2010, CVD caused an estimated 16 million deaths and led to 293 million disability-adjusted life-years (DALYs) lost—accounting for approximately 30% of all deaths and 11% of all DALYs lost that year.

Between 1990 and 2010, deaths from CVD increased from 26% to 29.5% of all deaths globally—a reflection of the rapidity of the epidemiologic transition—particularly in low- and middle-income regions.[1]

Ischemic heart disease (IHD) is growing among low-income groups, but primary prevention has delayed the disease to later in life in all socioeconomic groups. IHD is likely to become the most common cause of death worldwide by 2020.[2]

The fundamental pathological alteration underlying left ventricular dysfunction in acute MI is loss of functional myocardium.

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Depression of cardiac function in MI is directly related to the extent of left ventricular (LV) damage. Myocardial ischemia and necrosis set in within about 20–40 min. This occurs as a wavefront starting from the sub-endocardial region and progressing to the sub-epicardial region. The entire process usually takes 6 h to complete. Therefore any intervention for limiting infarct size should be initiated in this time window of 6 h.[3]

The initial ECG is diagnostic of acute MI in slightly more than half of the patients. Serial tracings increase the sensitivity to near 95%. A single ECG may never be diagnostic. However a pattern of ST segment displacement, especially with associated Q waves and T wave changes, and a clinical history of ischemic heart disease is highly suggestive of acute MI.[4]

Characteristic changes on ECG along with LVEF and RWMA detected by 2D-Echocardiography help in diagnosis and prognostication of MI. These investigations are non-invasive and can be done at less advanced centers.

Hence, this study was undertaken to localize the site of infarction in acute STEMI by ECG and correlate it with RWMA on 2D-Echocardiography and also to assess the prognosis of MI.

2. Methods

This prospective study was carried out in the ICCU of department of Medicine, SS Medical College & SGM Hospital, Rewa from June 2014 to Oct. 2015 on 100 consecutively selected patients fulfilling the following criteria:

All patients

- with acute MI with typical chest pain lasting >30 min.
- with ECG criteria - ST elevation ≥1 mm in at least two contiguous limb leads & ≥2 mm in chest leads.
- raised Cardiac Enzyme-Troponin T.
- and who underwent echocardiography.

The following patients were not considered for the study:
Patients with history of

- ECG and clinical features of pericarditis and those diagnosed as non-STEMI.
- ECG features suggestive of early repolarisation.
- Patients with previous MI.
- Patients with LBBB on baseline ECG.
- who could not undergo echocardiography.

The data was collected by taking a detailed history from the patients particularly keeping the following points in view:

a) Time of onset of typical chest pain, nature of pain, radiating, increasing with exertion, not relieved by rest and associated symptoms like excessive sweating, vomiting, nausea, breathlessness, giddiness, fatigue and others. A history of smoking, alcohol consumption, hypertension, diabetes mellitus, dyslipidemia and family history of IHD was taken.
b) A thorough clinical examination was carried out in each case with special reference to pulse, blood pressure, cardiovascular and respiratory examination for the presence of any cardiac enlargement, S3 gallop, rub, murmur and basal crepitations in the lungs.
c) Investigations like complete blood count, liver and renal function tests, random blood sugar, ECG, 2D-Echocardiography & enzymes like Troponin T and SGOT were done.
d) ECG was taken at the time of admission for the diagnosis of myocardial infarction, the criteria consisting of ST segment elevation of ≥2 mm, 0.08 second from J point in ≥2 contiguous chest leads or ≥1 mm in contiguous limb leads and with typical evolutionary changes or presence of new pathological Q waves.

Further patients were classified into 3 broad subgroups.
i) Anterior wall myocardial infarction (AWMI),
ii) Inferior wall myocardial infarction (IWMI),
iii) Anterior+ Inferior myocardial infarction.

Continuous cardiac monitoring was done and patients were treated with generally accepted methods of coronary care unit.
e) As soon as feasible, a 2D-Echocardiogram was performed. With the patient in left lateral decubitus position, multiple parasternal long axis views, short axis and apical views were taken to study RWMA and for estimation of LVEF in patients presenting with acute MI.
f) ECG was recorded on a standard ECG machine at a paper speed of 25 mm/s.

The culprit vessel localized on the ECG was thereafter correlated with the RWMA on 2D-Echocardiography.

3. Observations and results

Out of 100 patients with acute MI who were enrolled in this study 79 were male and 21 were female (M:F = 3.8:1).

In present study, maximum patients (61%) had AWMI followed by IWMI (36%) followed by Anterior+Inferior Wall MI (3%) of patients.

In present study out of 61 AWMI patients, 29 patients (29%) had extensive AWMI, 17 patients (17%) had anteroseptal and 15 patients (15%) had anterolateral Wall MI.

Out of 36 IWMI patients, 28 patients (28%) had isolated IWMI, 7 patients (7%) had inferoposterior and 1 patient (1%) had inferolateral MI. Three patients (3%) had Antero–Inferior Wall MI (Table 1).

Maximum RWMA was found to be present in Anterior/IVS/Apical Region 44%, followed by Inferior Wall in 36%, followed by lateral Wall in 8%, Global Hypokinesia was present in 4% while there was no RWMA in 8% of the patients.

The culprit vessel was found to be LAD in 63 patients, RCA in 33 patients and LCx in 4 patients on ECG.

The Culprit vessel was found to be LAD in 59 patients, RCA in 30 patients, LCx in 06 patients and there was no RWMA in 8 patients on Echocardiography.

In present study, Tachyarrhythmias including Atrial Fibrillation, Ventricular Bigeminy and Ventricular Tachycardia were found to be higher in AWMI while Bradyarrhythmias including second degree AV Block, CHB and SA Node Dysfunction were found to be higher in patients with IWMI. 7 patients developed tachyarrhythmias out of which 6 had AWMI and 1 had IWMI, 10 patients

Table 1
Site of Infarction determined by Localization of changes in ECG leads.

Site of infarction	Type of MI according to localization in ECG leads (n = 100)	Number (%)
Anterior (n = 61)	Extensive Anterior (ST↑ in V1-V6, I, aVL)	29
	AnteroSeptal (ST↑ V1-V3,qRBBB)	17
	AnteroLateral (ST↑ V5,V6,I,aVL)	15
Inferior (n = 36)	Inferior (ST↑II, III, aVF)	28
	InferoPosterior (ST↑II, III, aVF, V1, V3R, V4R; ST↓ I, aVL, V2, V3 with R in V1)	7
	InferoLateral (ST↑ II, III, aVF, I, aVL, V5, V6)	1
ANT + Inferior (n = 3)	Anterior + Inferior (ST↑ in all Leads)	
	Total	**100**

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