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Original Research Article.

Evaluation of Angiographic Profile in Patients with Non-ST-Elevation Myocardial Infarction (NSTEMI) and Normal ECG: A Prospective Study

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ABSTRACT

Background: In patients presenting with non-ST-elevation myocardial infarction (NSTEMI), coronary artery left anterior descending (LAD) and three-vessel disease are the most common complications in the presence of ST depression, while one-third of patients the infraction relative to the left artery circumflex (LCX) have normal ECG. We will compare the characteristics of coronary lesions of those with normal ECG compared with abnormalities, short-term and mid-term clinical endpoints, and evaluate coronary lesion predators using ECG, and clinical parameters.

Materials & Methods: A research study conducted on 350 patients approved of NSTEMI Diagnosis at the Department of Cardiology, SMS Medical College and Hospital Jaipur (Rajasthan) within 18 months. ECG induction was interpreted by an electrophysiologist who was blinded by the outcome of the coronary angiogram. Patients were given dichotomized into a normal or abnormal ECG group. The main end was the presence of a culprit lesion. The secondary endpoints included longer stays, hospitalizations within 60 days, and deaths in the hospital.

Results: Number of 350 patients diagnosed; 76 with normal and 274 have an abnormal ECG. At least one culprit lesion was found in 300 patients (85.72%), and especially in those with abnormal ECG (92.33% vs. 61.84%, P <0.05*). LAD was the most common lesion in both groups. There have been high incidences of two to three diseases in the abnormal ECG group (P <0.05*). On the other hand, there was a tendency for higher LCX involvement (P> 0.05) and normal coronary artery ECG group (p <0.05*).

Conclusions: Abnormal ECG was strongly associated with disease of two and three vessels, while normal ECG was strongly associated with LCX involvement or a common angiogram. Acceptance of ECG did not affect secondary outcomes.

Keywords: LAD, LCX, Non-ST-Elevation Myocardial Infarction (NSTEMI), Normal ECG, Culprit Lesion.

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INTRODUCTION

Cardiovascular disease is expected to be the leading cause of death worldwide due to the rapid increase in obesity, high blood pressure and diabetes. Atherosclerotic lesions and plaque rupture are the most common causes of myocardial infarction. Rest 12-lead ECG is the first diagnostic test for chest patients and should be performed and interpreted within the first 10 minutes of patient admission to the emergency department. Cardiac biomarkers are better, higher sensitivity to cardiac troponin, mandatory in all patients with suspected NSTEMI diagnosis, risk and treatment. Prompt, accurate diagnosis and risk classification of patients with asthma will help in the treatment of the appropriate medication as well as the timing of the invasive plan and revascularization options. NSTEMI is an acute ischemic event causing cardiomyocyte death by necrosis in a clinical setting consistent with acute myocardial ischemia.[1]

Non-ST-elevation myocardial infarction (NSTEMI) is the leading cause of illness and death.[2] Although electrocardiogram at admission is rare, the presence of a normal ECG does not exclude disease.[3]

Risk assessment is a cornerstone in screening NSTEMI patients. While ST depression is one of the seven leading causes of high mortality in NSTEMI patients (TIMI scores), there are few details with those who do not have ST depression and normal ECG typically present.

A left circumflex (LCX) coronary artery occlusion may occur with a normal ECG. A normal ECG does not exclude ACS, as previously

reported by McCarthy et al, as approximately 5% of patients discharged from an emergency room with a normal ECG have a serious ischemic event. According to a two-point risk analysis for non-ST elevation ACS, ECG flexibility was an important tool. In the TIMI (Thrombolysis In Myocardial Infarction) risk factors[4], binary variable ECG, the presence or absence of ST depression, were a risk factor for a combination of death or ischemic events, approximately 14 days after ACS, and in GRACE (Global Registry of Acute Coronary Events) scores, the ECG maintained six independent predictors of general mortality. We will compare the characteristics of coronary ulcers of those with normal ECG compared with abnormalities, short-term and mid-term clinical endpoints, and evaluate coronary lesions predators using ECG, and clinical parameters.

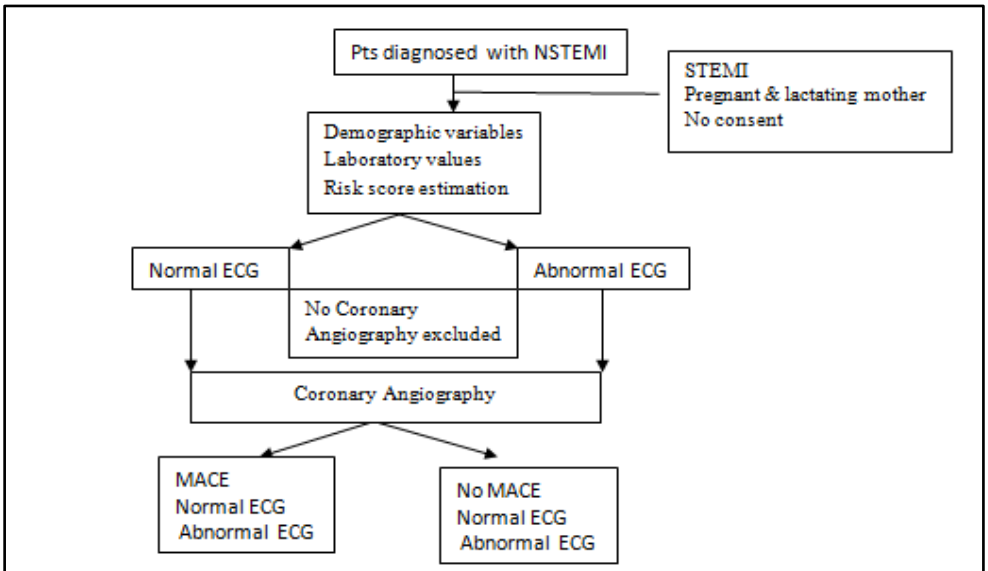

Figure: 1- Flow diagram of distribution of patients according to echocardiography

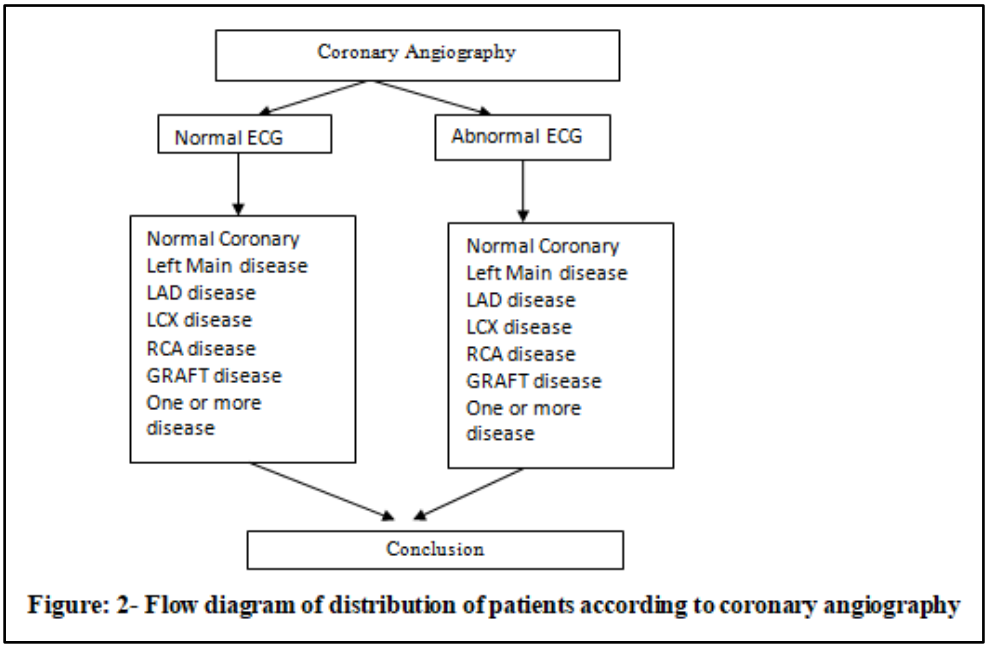

Figure: 2- Flow diagram of distribution of patients according to coronary angiography

MATERIALS & METHODS

A prospective observational study done on 350 patients admitted with Diagnosis of NSTEMI in Department of cardiology, SMS Medical college & Hospital Jaipur (Rajasthan) for 18 months period.

Inclusion Criteria

- Diagnosed case of NSTEMI included in study.

Exclusion Criteria

- STEMI
- Pregnant and Lactating mother
- Not giving consent.
- Complete or Incomplete Bundle Branch Block.
- Major comorbidity like pulmonary or renal failure.
- Pacemaker implantation
- Cardiac glycosides.
- Congenital heart disease
- Dilated cardiomyopathy

Methods

NSTEMI was defined according to the ACC/AHA guidelines[6] and the universal definition of myocardial infarction[7] as the presence of

positive cardiac troponin with levels greater than 99th percentile upper reference limit AND either symptoms suggestive of ischemia, new ST/TW changes (ST elevation was excluded), new Q waves, or new wall motion abnormalities on echocardiogram.

ECG Interpretation

- ECG will be recorded as the presence or absence of ST segment depression, T wave changes, Q waves.
- The electrocardiographic results will be then dichotomized as either normal (none of the above changes are present) or abnormal (one or more of the above findings are observed).

Echocardiography

Baseline echocardiography was performed on admission or after the coronary angiogram in the lateral decubitus position, in a standard fashion using commercially available systems. Echocardiographic parameters such as left ventricular (LV) end-diastolic and systolic dimensions, LV mass, ejection fraction (EF), left atrial volume index, and diastolic function were extracted from the echocardiography report.

Coronary Angiography

The coronary angiography films and reports were reviewed. The presence or absence of culprit lesion, number of lesions, location and segment involved, degree of initial stenosis, type of intervention, and residual stenosis were extracted. The location and segment involved were coded according to the SCCT coronary segments diagram.[8]Diameter stenosis $\geq$50% is considered significant in Lt main coronary artery and diameter stenosis $\geq$ 70% is considered significant in other coronary arteries.

Statistical Analysis

Continuous variables will be expressed as means ± SD and compared by use of the unpaired Student t test. Categorical variables will be expressed as percentages and compared by use of the Pearson Chi-square test as appropriate.

Table 1: Baseline characteristics stratified by normal versus abnormal ECG on admission

Variables	All patients (N=350)	Normal ECG (N=76)	Abnormal ECG (N=274)	p-value
Age (yrs)	62.8±7.34	61.72±8.11	63.08±7.90	0.67
Male	260	53	207	>0.05
Female	90	23	67	>0.05
BMI	28.9±6.33	29.3±7.12	28.7±7.89	>0.05
Smokers	198	53	145	<0.05
Non-smokers	152	23	129	<0.05
Diabetes	130	43	87	<0.05
Hypertension	251	34	217	<0.05
Dyslipidemia	217	26	191	<0.05
CAD	128	19	109	<0.05
NYHA Class ≥2	28	3	25	<0.05
TIMI Score	3.12±0.67	2.87±0.98	3.19±1.2	<0.05
GRACE Score	137.5±15.6	129.5±12.7	156.9±20.2	<0.05
Prior PCI	57	13	48	<0.05
Prior CABG	8	3	6	>0.05
PAD	20	4	16	<0.05

Table 2: Laboratory findings by normal versus abnormal ECG on admission

Laboratory values	All patients	Normal ECG	Abnormal ECG	P-value
Troponin T (microg/L)	0.77±0.12	0.090±0.0056	0.29±0.08	<0.05*
Hb (g/dl)	10.8±1.2	10.5±1.34	10.89±1.06	>0.05

Table 3: Baseline ECG findings

Admission ECG	All Patients (N=350)
Normal ECG	76 (21.71%)
ST depression	68 (19.42%)
T Wave Changes	101 (28.85%)
Q Waves	38 (10.85%)
1 Abnormality	145 (41.42%)
More than 1 abnormality	82 (23.42%)

Table 4: Echocardiographic parameters stratified by ECG on admission

ECHOCARDIOGRAPHY	All patients (N=350)	Normal ECG (N=76)	Abnormal ECG (N=274)	p-value
Mean ejection fraction (%)	176 (50.28%)	42 (55.26%)	134 (48.90%)	<0.05*
Ejection fraction ≤40%	70 (20.00%)	16 (21.05%)	54 (19.70%)	>0.05
Wall motion abnormality	190 (54.28%)	30 (39.47%)	160 (58.39%)	<0.01**
Significant Valvular disease	57 (16.28%)	13 (17.10%)	44 (16.05%)	>0.05
Diastolic function				
Normal	74 (21.14%)	26 (34.21%)	48 (17.51%)	<0.05*
Grade 1 Diastolic disfunction	195 (55.71%)	41 (53.94%)	154 (56.20%)	
Grade 2 Diastolic disfunction	60 (17.14%)	6 (7.90%)	54 (19.70%)	
Grade 3 Diastolic disfunction	21 (6.00%)	3 (3.94%)	18 (6.56%)	

Table 5: Culprit lesions and interventions

Variables	All patients (N=350)	Normal ECG (N=76)	Abnormal ECG (N=274)	p-value
Normal epicardial coronaries	50 (14.28%)	29 (38.15%)	21 (7.66%)	<0.05*
1 culprit lesion	187 (53.42%)	36 (47.36%)	151 (55.10%)	
2 culprit lesions	71 (20.28%)	6 (7.89%)	65 (23.72%)	
3 culprit lesions	42 (12%)	6 (7.89%)	36 (13.13%)	
Characteristic of culprit lesion (N=300)				
Native vessels	285 (81.42%)	72 (94.73%)	213 (77.73%)	>0.05
Graft	7 (2.00%	1 (1.31%)	6 (2.18%)	
Native & graft	8 (2.28%)	3 (3.94%)	5(1.82%)	
Coronary artery involvement (N=300)				
Left Main	14 (4.66%)	3 (3.94%)	11 (4.01%)	.0.05
LAD	182 (60.66%)	19 (25%)	163 (59.48%)	
LCX	53 (17.66%)	21 (27.63%)	42 (15.32%)	
RCA	45 (15%)	13 (17.10%)	32 (11.67%)	
Graft	6 (2%)	1 (1.31	5 (1.82%)	
Intervention				
No intervention	65 (21.71)	16 (21.05%)	649 (17.88%)	>0.05
CABG	20 (6.85%)	4 (5.26%)	16 (5.83%)	
DES				
1 vessel	134 (44.57%)	39 (51.31%)	95 (34.67%)	
2 vessels	56 (18.85%)	4 (5.26%)	52 (19.98%)	
3 vessels	25 (8.33%)	3 (3.94%)	22 (8.02%)	

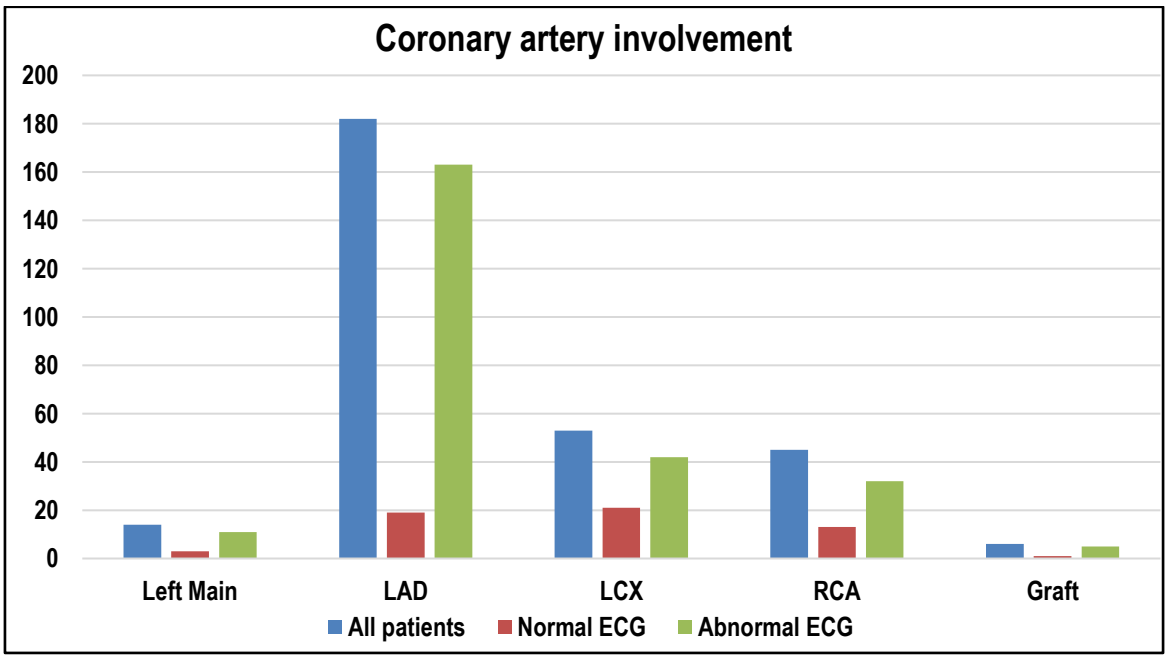

Figure 1: Percentage of detected culprit lesions stratified by coronary artery territory. LAD, left anterior descending; LCX, left circumflex; RCA, right coronary artery.

Table 6: Clinical endpoint by normal versus abnormal ECG on admission

Clinical endpoint	All patients	Normal ECG	Abnormal ECG
In hospital mortality	24 (6.85%)	3 (3.94%)	21 (7.66%)
Mace-14 days	10 (2.85%)	0	10 (3.64%)
Mace-30 days	4 (1.14%)	0	4 (1.45%)
Mace 6 months	10 (2.85%)	0	10 (3.64%)

RESULTS

A total of 350 patients (mean age 62.8±7.34 years, 74% male) were identified and analyzed. There was significant prevalence of cardiovascular risk factors including diabetes mellitus (37.14%), hypertension (71.71%), and smoking history (56.57%), with more than one third of patients having known prior coronary artery disease (CAD) (Table 1).

Our study showed that laboratory parameters such as troponin c was statistically significant in between normal ECG and abnormal ECG patients (table 2).

There were 76 (21.72%) patients with normal and 274 (78.28%) with abnormal ECG. The most common ECG abnormalities were T wave changes, followed by ST depression (Table 3). Patients with normal ECG were younger, had lower prevalence of known

CAD, heart failure, intake of cardiovascular medications, lower creatinine and peak CKMB (Table 1).

Baseline echocardiography was performed on all patients. The mean LVEF was (50±11)% with 20% having EF <40% and almost half having some wall motion abnormalities. The majority (78.85%) had diastolic dysfunction. Patients with normal admission ECG had smaller LV size, higher EF, lower incidence of wall motion abnormalities, systolic and diastolic dysfunction (Table 4).

Coronary Angiography

A total of 300 patients (85.72%) had abnormal coronary angiogram; 32.28% had more than one culprit lesion, and 60.66% had a culprit lesion in the left anterior descending (LAD) coronary artery (Table 4). Patients with normal ECG had significantly more normal coronary angiogram (38.15% vs. 7.66%, P<0.05), but similar distribution of culprit lesion, interventions, and degree of stenosis (Table 5).

The admission and discharge cardiovascular medications, stratified by patients with and without culprit lesions, are summarized in Table 6. A significantly higher percentage of patients with culprit lesions was discharged on cardiovascular medications as compared to those with normal coronaries, although significantly higher than at admission for both groups.

DISCUSSION

We found that 85.71% of patients had a culprit lesion, usually LAD, those with a normal ECG had normal coronary arteries or LCX involvement and there was no difference in hospital stay or hospitalization and no hospital deaths between groups.

A normal ECG in patients presenting with NSTEMI does not indicate a specific culprit. Indeed, LAD was a culprit lesionthat is most common in those with normal and abnormal ECG, and without finding nearby, mid or distal lesions. However, those with normal ECG had a higher tendency to have LCX involvement, emphasizing therefore the importance of leading ECG (V7-9) [9-11] which became standard care for patients with their first thoughtless and internal ECG / risk severe acute coronary syndrome (stage IIa, Level of evidence B).[12] It is estimated that 4% of all myocardial acute infarction indicates the presence of a distinct ST elevation in the posterior chest leads.[13]

Patients with normal ECG have a very common coronary angiogram (38.15% vs. 7.66%, P <0.05), but the same distribution of culprit lesion, intervention, and stenosis rate. There was no significant difference in length of stay, hospital rehabilitation or hospital mortality among those with normal and abnormal ECG, or between those with or without a culprit lesion. This may be due to prompt medical management and coronary intervention of all NSTEMI patients regardless of the initial outcome of the ECG, as well as the small sample size. On the other hand, and not surprisingly, patients with NSTEMI and ECG abnormalities had abnormal wall movements, low LVEF, severe diastolic function, and a very high incidence of two or three vessels. Abnormal ECG was associated with more than three times the risk of finding a culprit lesion and a positive prediction of 92% and this effect could be explained by understanding the pathophysiology of ischemic changes in the ECG. When sufficient myocardial perfusion is limited to the subendocardial layer as in NSTEMI, affected myocytes may lose their long-term activation capacity resulting in common similarities in the QRS complex and T wave spatial directions. The resulting ischemic T waves are converted from positive to negative in most ECG leads because the axis of the ischemic T waves is directed away from the affected region of the left ventricle.[14] On the other hand, a change in the base of the ST phase occurs when absorption occurs. insufficiency causes the myocardial-cell membrane to have abnormal fillings in ion flow. The resulting energy difference between the damaged and injured myocardium creates a continuous flow of the injured current. When injury is limited to the subendocardial layer of the left ventricle, the effect of “subendocardial injury” shifts the axis of the ST segment usually far from that ventricle, but not too far from the affected region. When insufficient lubrication passes through all layers of the myocardial (transmissions), the effect of epicardial ischemia shifts the T-wave axis to the affected region of the left ventricle.[14]

CONCLUSIONS

Abnormal ECG was strongly associated with disease of two and three vessels, while normal ECG was strongly associated with LCX involvement or a common angiogram. Acceptance of ECG did not affect secondary outcomes.

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