THE ROLE OF ECG IN LOCALIZING THE CULPRIT VESSEL OCCLUSION IN ACUTE ST SEGMENT ELEVATION MYOCARDICAL INFARCTION WITH ANGIOGRAPHIC CORRELATION

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ABSTRACT

BACKGROUND & OBJECTIVES

The Electrocardiogram remains a crucial tool in the identification and management of acute myocardial infarction. A detailed analysis of patterns of ST-segment elevation may influence decisions regarding the perfusion therapy. This study was undertaken to identify the culprit vessel from ECG in patients with acute ST elevation myocardial infarction and correlate with coronary angiogram.

MATERIALS & METHODS

This is a prospective study, conducted on 126 patients in Osmania General Hospital, Hyderabad. Patients with ST segment elevation from ECG was evaluated to identify culprit vessel and later correlated with coronary angiogram.

RESULTS

Amongst 126 patients in this study, 70 patients had anterior wall and 56 patients had inferior wall myocardial infarction. ST↑> 1mm in V4R, ST ↓ V3/ST ↑ LIII <0.5 were equally sensitive in proximal RCA occlusion. While in patients with distal RCA occlusion the ratio of ST depression in Lead V3/ST elevation in Lead III between 0.5-1.2 had maximum sensitivity. In LCx occlusion ST elevation in Lead lll> Lead II was the most sensitive and ratio of ST↓V3/ST↑LIII >1.2 was the most specific criteria. ST↓ in inferior leads > 1mm had maximum sensitivity in localizing occlusion in proximal D1 occlusion proximal to S1 as well. Absence of ST i in inferior leads is the most sensitive criteria in occlusion distal to S1 as well as in distal D1 in AWMI.

CONCLUSION

The admission ECG in patients with ST elevation AMI is valuable not only for determining early reperfusion treatment, but also provides important information to guide clinical decision-making.

KEYWORDS

ECG, AMI, ST Segment Elevation.

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INTRODUCTION: It is widely acknowledged that heart disease and stroke are the leading causes of death and disability worldwide in developed as well as in developing countries.

We are in the midst of a true global cardiovascular disease (CVD) epidemic. CVD is responsible for about 30 percent of all deaths worldwide each year. Nearly 80 percent of these deaths occur in developing countries. Indeed, CVD is the leading cause of mortality in almost every region of the world. The early (30-day) mortality rate from AMI is 30%, with more than half of these deaths occurring before the stricken individual reaches the hospital. Although the mortality rate after admission for AMI has declined by ~30% over the past two decades, approximately 1 of every 25 patients who survives the initial hospitalization dies in the first year after AMI. Mortality is approximately fourfold higher in elderly patients (over age 75) compared with younger patients.

The electrocardiogram remains a crucial tool in the identification and management of acute myocardial infarction. Acute risk stratification in myocardial infarction is still based on simple clinical parameters, laboratory markers and 12 lead electrocardiography. The electrocardiogram has been a preliminary screening and one of the most useful diagnostic investigations in myocardial infarction. Patients are diagnosed as having anterior, inferior or lateral myocardial infarction based on patterns and magnitude of ST deviation.

A detailed analysis of patterns of ST-segment elevation may influence decisions regarding the perfusion therapy. The early and accurate identification of the infarct related artery can help predict the area of myocardium at risk and guide decisions regarding the urgency of revascularization. Electrocardiographic signs of reperfusion represent an important marker of micro vascular blood flow and

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consequent prognosis. Electrocardiography reflects the electrophysiology of myocardium during acute ischemia whereas the coronary angiography identifies the vessel anatomy. This study is limited to the usefulness of identifying the culprit vessel in acute ST elevation pattern in myocardial infarction from Electrocardiography and correlates it with coronary angiography. Present study has been undertaken to observe patterns of ECG changes in patient with ST elevation acute myocardial infarction. Then it will be correlated with the angiography findings. In particular, a comparison study between various patterns ECG changes of acute phase of myocardial infarction with coronary angiogram to predict the infract related artery.

AIMS: This study is aimed at validating the usefulness of electrocardiography in localizing the culprit vessel in acute ST elevation myocardial infarction and correlating the findings with coronary angiogram in order to plan early, rapid intervention and revascularization.

METHODOLOGY:

Inclusion Criteria: All patients with acute myocardial infarction with chest pain lasting >30 minutes, with ECG criteria-ST elevation >1 mm in atleast two contiguous leads in limb leads & >2 mm in chest leads who underwent coronary angiogram.

Exclusion Criteria: Patients with history of Previous myocardial infarction Prior CABG.

Congenital heart disease ECG showing features of LVH Left BBB in baseline ECG.

MATERIALS AND METHODS:

Prospective observational study.
Study Period: November 2011 up to October 2013.
Set-Up: Osmania General Hospital, Hyderabad.

Study Design: The study population consisted 126 cases of acute myocardial infarction attending the medical intensive care unit of upgraded department of medicine in Osmania general hospital, Hyderabad., with elevation of ST segment in ECG who subsequently underwent coronary angiography.

The patients convening to the inclusion criteria were enrolled into the study after being explained the proceedings of the study and after they signed the consent form.

The patients presenting with chest pain were evaluated with standard 12 lead ECG, and cardiac enzymes (CK and CK-MB or Troponins).

A detailed history was taken about the chest pain, presence of risk factors and duration of risk factors as appropriate.

Acute myocardial infarction was differentiated into inferior wall and anterior wall infarction. Based on the various ECG criteria culprit vessel was identified.

Coronary angiography reports were collected. The infarct related artery (IRA) was identified from total occlusion or significant stenosis (>70%).

Statistical Analysis: All data obtained were recorded and presented as Mean±Standard Deviation. The ECG findings in the patients with anterior and inferior wall were compared using the X2 test, as appropriate by using SPSS 16 software. A P-value <0.05 was considered statistically significant. The sensitivity, specificity, positive predictive value and negative predictive value of individual parameters were calculated.

RESULTS: Out of 126 patients included in the study, 70 had AWMI and 56 had IWMI.

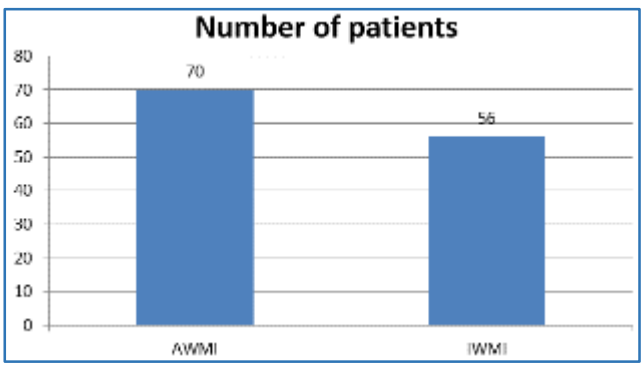

Fig. 1: Number of patients with AWMI and IWMI

Out of 70 patients with AWMI, 25 patients had block proximal to S1,32 had block proximal to D1,7 had distal to S1 and 6 had distal to D1.

ANTERIOR WALL MYOCARDIAL INFARCTION:

ST ↑ in V1 >2.5 mm	Proximal to S1
Complete RBBB	Proximal to S1
ST↑ in aVR	Proximal to S1
ST↓ in V5	Proximal to S1
Q wave in aVL	Proximal to D1
Q wave V4-V6	Distal to S1
ST↓ in aVL	Distal to D1
Inferior ST ↓> 1.0mm	Proximal to S1/D1
Absence of inferior ST↓	Distal to S1/D1

Table 1: ECG predictors of LAD occlusion site (Engelen's criteria)

Site of Occlusion	Number of patients
Proximal to S1	25
Distal to S1	7
Proximal to D1	32
Distal to D1	6

Table 2: Showing sites of occlusion in patients with AWMI

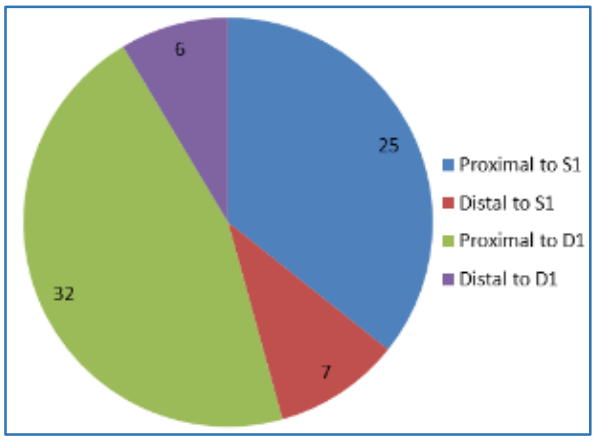

Fig. 2: Sites of occlusion in AWMI

OCCLUSION PROXIMAL TO S1:

1. **ST Elevation in Lead V1 >2.5mm:** Seen in 14 patients out of 25 with occlusion proximal to S1 and in 4 of the patients with other sites of occlusion. Sensitivity-56%, specificity-91.1%.

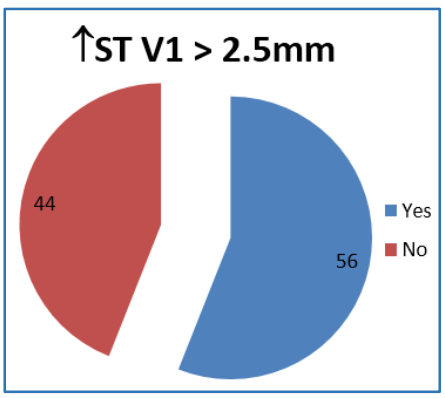

Fig. 3: Patients with ST 1 Vi>2.5 mm in occlusion proximal to S1 in AWMI

2. **Complete RBBB:** Seen in 4 out of 25 patients with occlusion proximal to S1 and 3 patients with other sites of occlusion. Sensitivity-16%, Specificity-93.33%.

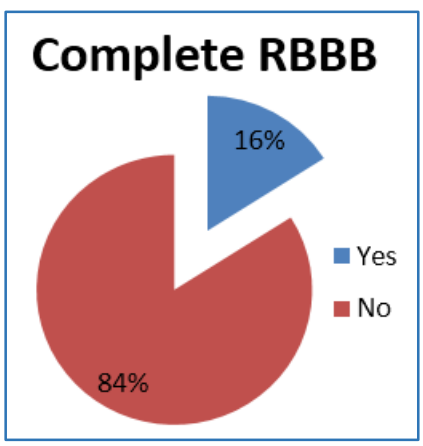

Fig. 4: Patients with complete RBBB in occlusion proximal to S1 in AWMI

3. **ST Elevation in Lead VR:** Seen in 8 out of 25 patients with occlusion proximal to S1 and 2 of those with other sites of occlusion. Sensitivity-32%, Specificity-95.55%.

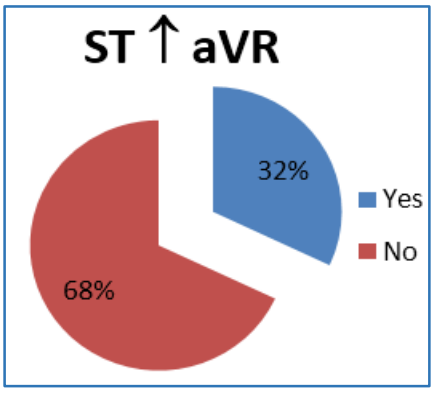

Fig. 5: Patients with ST taVR in occlusion proximal to S1 in AWMI

4. **ST Depression in Lead V5:** Seen in 4 out of 25 patients with occlusion proximal to S1 and 2 of those with other sites of occlusion. Sensitivity-16%. Specificity-95.5%.

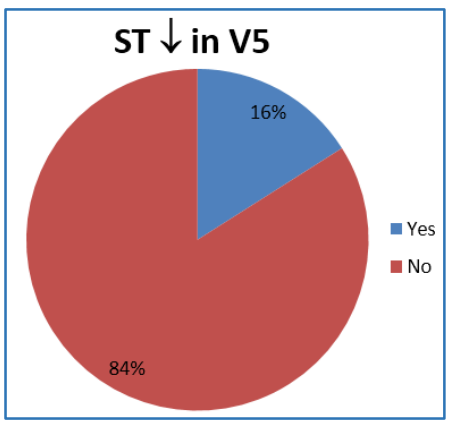

Fig. 6: patients with ST ↓ V5 in occlusion proximal to S1 in AWMI

5. **ST Depression in Inferior Leads >1.0mm (LII, III, aVF):** Seen in 19 out of 25 patients with occlusion proximal to S1 and 6 of those with other sites of occlusion. Sensitivity–76%, Specificity–86.7%.

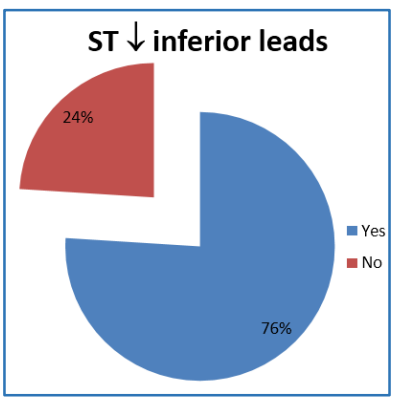

Fig. 7: Patients with ST ↓ inferior leads in occlusion proximal to S1in AWMI

Criteria	Number of patients fulfilling the criteria
↑ ST V1 > 2.5mm	14
Complete RBBB	4
ST ↑ aVR	8
ST ↓ in V5	4
ST ↓ in inferior leads	19

Table 3: Comparing various criteria for sites of occlusion proximal to S1 in AWMI

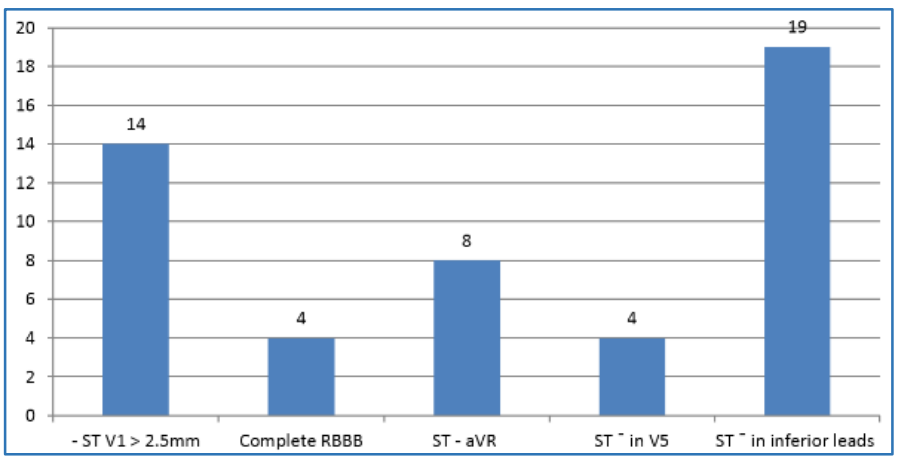

Fig. 8: Comparison of different criteria in patients with AWMI occlusion proximal to S1

OCCLUSION PROXIMAL TO D1:

1. **Q Wave in Lead aVL** seen in 15 out of 32 patients with occlusion proximal to D1 and 6 of those with other sites of occlusion. Sensitivity–46.8%, Specificity–84.2%.

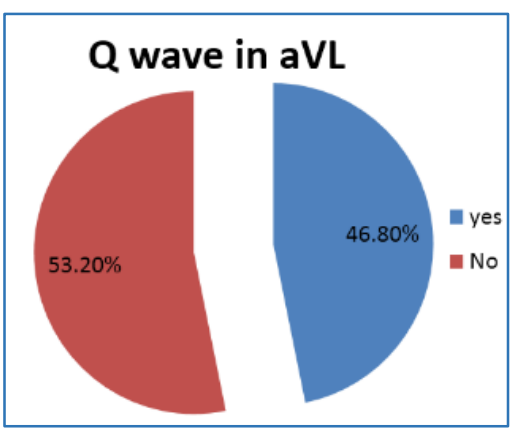

Fig. 9: Patients with Q wave in lead aVL in occlusion proximal to D1 in AWMI

2. **ST Depression in Inferior Leads (LII, III AVF):** Seen in 11 out of 32 patients with occlusion proximal to D1 and 3 of those with other sites of occlusion. Sensitivity–34.3%, Specificity–92.1%.

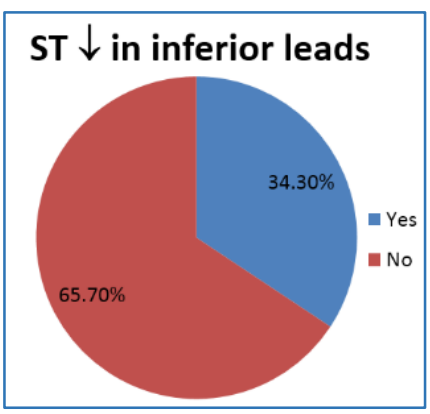

Fig. 10: Patients showing ST ↓ in inferior leads with occlusion proximal to D1 in AWMI

OCCLUSION DISTAL TO S1

1. **Q Wave in Lead V4 to V6:** Seen in 2 out of 7 patients with occlusion distal to S1 and 1 of those with other sites of occlusion, Sensitivity–28.5%, Specificity–98.4%.

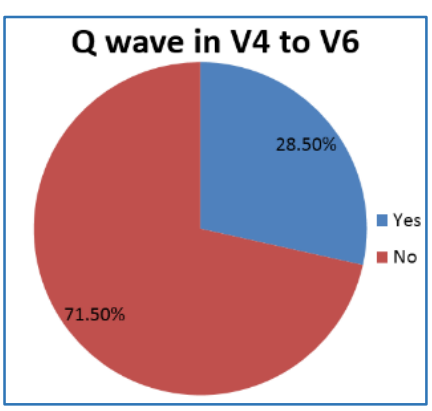

Fig. 11: patients showing Q wave in leads V4 to V6 with occlusion Distal to S1 in AWMI

2. **Absence of ST Depression in Inferior Leads (LII, III, aVF):** Seen in 6 out of 7 patients with occlusion distal to S1 and 32 of those with other sites of occlusion, Sensitivity–85.7%, Specificity–49.2%.

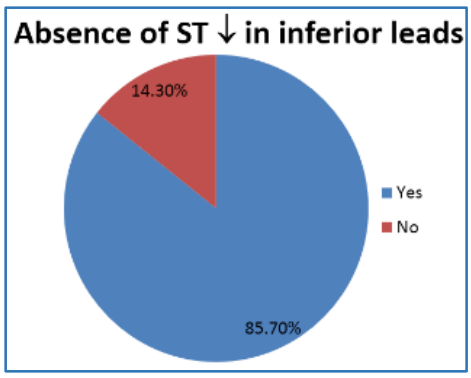

Fig. 12: Patients showing absence of ST ↓ in inferior leads with occlusion

Distal to S1 in AWMI

Occlusion Distal to D1:

1. **ST Depression in Lead aVL:** Seen in 1 out of 6 patients with occlusion distal to D1 and none of those with other site of occlusion, Sensitivity–16.66%, Specificity–100%.

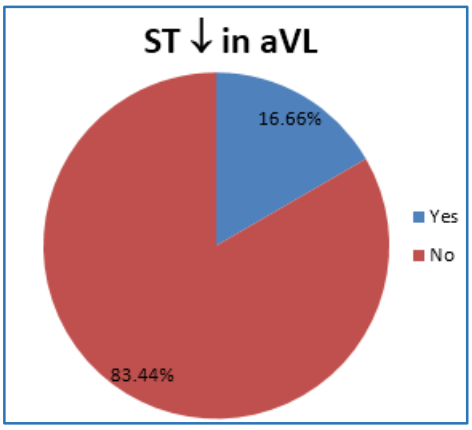

Fig. 13: Patients showing absence of ST ↓ in lead aVL with occlusion Distal to D1 in AWMI

2. **Absence of ST Depression in Inferior Leads (Lead II, III, aVF)** Seen in 4 out of 6 patients with occlusion distal to D1 and 32 of those other sites of occlusion. Sensitivity–6.6%, specificity–50%.

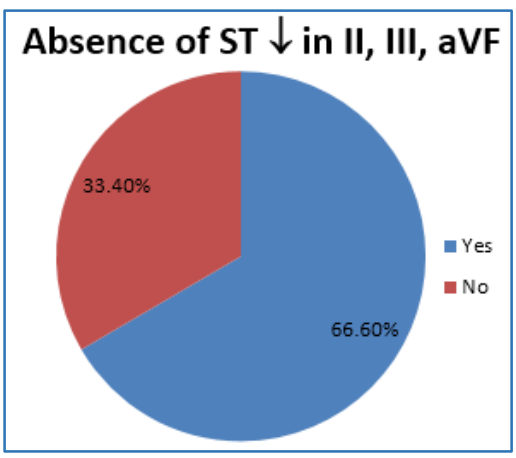

Fig. 14: Patients showing absence of ST↓ in inferior leads with occlusion distal to D1 in AWMI

	Present Study		Manjunath et al		Engelen et al[1]	
	Sensitivity	**Specificity**	**Sensitivity**	**Specificity**	**Sensitivity**	**Specificity**
Proximal to S1						
ST ↑ V1>2.5mm	56	91	71	66	12	100
ST ↑ aVR	32	96	50	100	43	95
Complete RBBB	16	93	-	-	-	-
ST ↓ V5	16	96	8	100	17	98
Inferior ST↓ >1.0mm	76	87	90	85	49	85
Proximal to D1						
Q aVL	47	84	66	90	44	85
Inferior ST↓ >1.0mm	34	92	82	90	51	86
Distal to S1						
Q wave V4-V6	28	98	25	88	24	93
Absence of inferior ST↓	86	49	93	79	48	83
Distal to D1						
ST↓aVL	17	100	10	100	22	95
Absence of Inferior ST↓	67	50	82	89	50	86

Table 6: Comparison of various criteria to identify culprit vessel in AWMI with present study

Inferior Wall Myocardial Infarction: Out of 56 patients with IWMI, 21 patients had culprit lesions localized to proximal RCA, 24 in distal RCA and Left circumflex artery in 11 patients by CAG.

Site of occlusion	Number of patients
Proximal RCA	21
Distal RCA	24
LCx	11

Table 7: Sites of occlusion in inferior wall MI

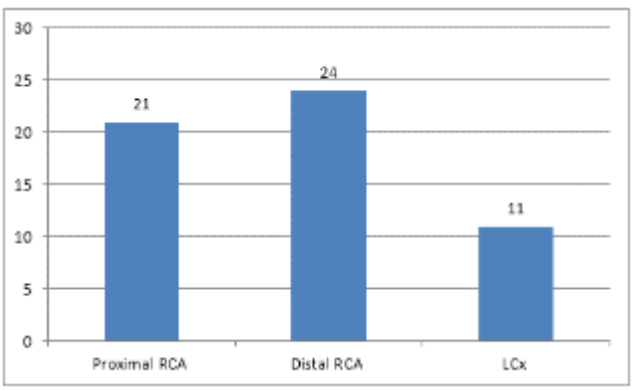

Fig. 15: Showing different sites of occlusion in inferior wall myocardial infarction

RCA Occlusion

1. **ST elevation Lead III > lead II:** Seen in 39 out of 45 patients with RCA occlusion and only 3 out of 11 with LCX occlusion. Sensitivity–86.6%, Specificity–72.72%.

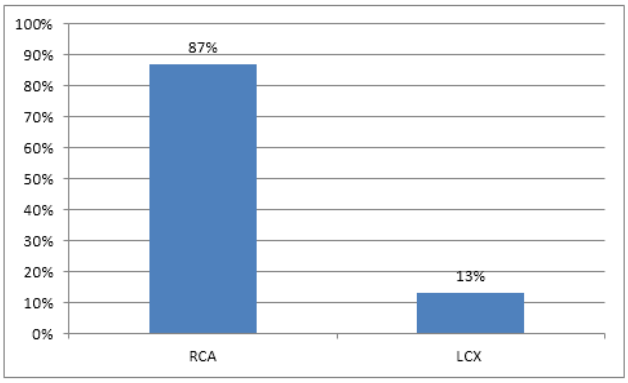

Fig. 16: Showing criteria ST ↑ lead III > II in identifying culprit artery in inferior wall MI

2. ST Depression >1mm in Leads I, aVL: Seen in 37 out of 45 patients with RCA occlusion and only 3 out of 11 with LCX occlusion. Sensitivity–82.22%, Specificity–72.72%.

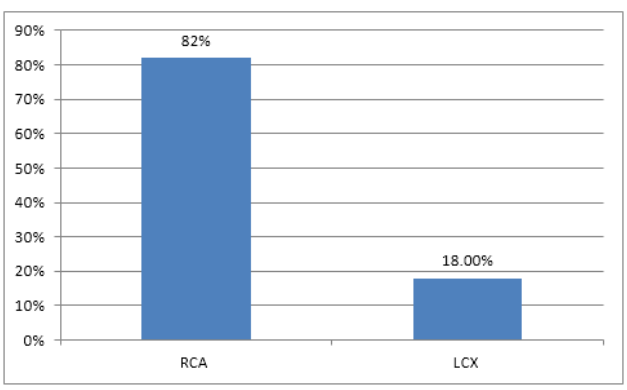

Fig. 17: Showing criteria ST↓ > 1mm in lead I, aVL identifying culprit artery in inferior wall MI

	Present Study		Glancy et al [2]	
ECG criteria for RCA occlusion	**Sensitivity**	**Specificity**	**Sensitivity**	**Specificity**
ST ↑ LIII > ST ↑ L II	87	73	96	40
ST ↓ LI, aVL	82	73	100	60
ST ↑ V4R > 1mm	71	66	86	100
ECG criteria for LCx occlusion				
ST ↑ LII > LIII	73	89	40	96
Isoelectric or ST↑ I, aVL	55	84	60	100

Table 8: Comparison of various ECG criteria in IWMI with present study

DISCUSSION: In present study we compared various ECG criteria for diagnosis of infarct related artery in acute myocardial infarction with angiographic findings as gold standard. Study included 126 patients. Among the study cases 92(73.01%) were male and 34(26.98%) were female. In this study incidence of CAD were about four times high in male than that of female. A study in Washington State concluded that Coronary Artery Disease (CAD) is equal opportunity killer in men and women over their lifetimes. In Washington State in 1991, the incidence of CAD death was 42% in female and 39% in male.[3] In 21 year follow-up of 12000 men and women from The Copenhagen City Heart Study found incidence of CAD in male approximately twice that found in female.[4] In the Framingham Heart Study, 26-year follow-up of men and women aged 35 to 84 years indicated that CAD morbidity was twice as high in men as in women, and 60 per cent of coronary events occurred in men.[5] Similarly, in Hadjadj S, Coisne D, Mauco G, et al incidence of CAD in male (119) was more twice that found in female (27).[6]

In our study, most prevalent age group of patients with ACS was between 50-59 years of age 46(36.50%), followed by 60-69 years age group 40(31.74%). Mean age of patients with ACS was 57.35+/-10.47 years. The INTERHEART Study observed Southeast Asia and Japan (57), North America (59) that were almost same mean age

(years) as in our study observed.[7] The mean age of patients was 58.6 years found in The CREATE-ECLA Randomized Controlled Trial which were as nearly same to this study.[8] Age is the most powerful independent risk factor for atherosclerosis. In this study, incidence of CAD were less on age more than 70 years as compared to age lesser than 70 years.

In our study, patients with acute myocardial infarction, Hypertension was found to be most frequent accounting risk factor 70 out of 126(55.55%) followed by smoking54 out of 126(42.85%), 25.39%(32 out of 126) were with dyslipidaemia. In this study 70 patients (55%) were hypertensive. According to JNC–VII guide lines 35.4% had SBP>140mmHg and 41.6% had DBP>90mmHg. Smoking is an important modifiable risk factor. The prevalence of smoking is increasing in India. This study showed that smoking (79%) was one of the most important risk factors associated with CAD. Smoking has been established as an independent risked factor for coronary artery disease. In Framingham Study 60% of patients with coronary artery diseases were smokers. Smoking interacts with other risk factors synergistically and increases the risk factor of coronary artery disease.[9]

Dyslipidaemia is widely accepted risk factor for CAD. Studies on South Asian population have reported that they generally do not have higher Total Cholesterol or LDL-Cholesterol concentration than white or Afro-Caribbean population; however, they have lower HDL-cholesterol and higher triglyceride concentration.[10] In this study 6 out of 48(12.5%) had Dyslipidaemia.

Various ECG criteria where compared in both groups for identification of culprit artery later correlated with coronary angiography.

Among the 126 patients included in the study 70 patients had anterior wall myocardial infarction and 56 had inferior wall myocardial infarction.

In patients with anterior wall myocardial infarction different criteria were analyzed to identify the culprit artery. Most of our results were consistent with study done by Glancy et al. In localizing culprit vessel proximal to S1, five different criteria were compared. Our study shows that ST depression in inferior leads >1mm had maximum sensitivity in localizing occlusion proximal to S1 as well as D1. ST depression in V_5 has maximum specificity in occlusion proximal to S1 and presence of Q wave in aVL in patients with occlusion proximal to D1 respectively. Absence of ST depression in the inferior leads is the most sensitive criteria in occlusion distal to S1 as well as in D1 in anterior wall myocardial infarction. While presence of Q waves lead V4-V6 has the maximum specificity in occlusion distal S1. And ST depression in lead aVL has maximum specificity in occlusion distal to D1.

In the 56 patients with acute inferior MI, the culprit lesion was in the RCA in 45 patients and in the LCX in 11 patients, a ratio of 34.1:1. Other studies of patients with acute inferior MI have found RCA to LCX ratios ranging from 2.2:1 to 7.0:1, and averaging 3.9:1.[11,12,13,14,] Thus, the RCA is much more likely than the LCX to contain the culprit lesion in patients with acute inferior wall myocardial infarction.

In the present study it was found that ST segment elevation in lead III exceeding that in lead II, that is a ratio of elevation in lead III/elevation in lead II >1, had a high specificity and sensitivity for RCA occlusion. 39(97.6%) of patients with RCA lesion were positive for this criteria, compared with only 3(2.4%) of the patient with LCX lesion, giving a sensitivity of 87%, a specificity of 73%, a positive predictive value of 93.2% and a negative predictive value of 75% for RCA involvement. These result is consistent with research done by Radhakrishnan Nair, D. Luke Glancy shows a sensitivity of 96%, a specificity of 40%, a positive predictive value of 89% and a negative predictive value of 67% for RCA involvement.[15]

These results suggest that a ratio of ST elevation in lead III/ST elevation in lead II >1 is an important predictor of RCA occlusion, a finding consistent with that of previous studies as Zimetbaum et al, who had showed that a higher ST segment elevation in lead III than in lead II was only seen in RCA occlusion, and Chia et al. reported that ST segment elevation in lead III greater than that in lead II was valuable in predicting RCA occlusion.

In addition, it was found that ST depression ≥1 mm in lead aVL was of value in predicting RCA occlusion. This criterion was also positive in 37(69%) of patients with RCA lesion but in only 3 with LCX lesion, giving a sensitivity of 82.2% which was same the previous criteria, but specificity was only 72.7%, a positive predictive value of 93.5% and a negative predictive value of 23.5% for RCA involvement. This finding is in agreement with previous studies. Bailey et al,[16] reported that ST segment depression in lead I and aVL was only observed during RCA occlusion. Birnbaum et al,[17] reported that ST segment depression in lead aVL was a sensitive early ECG sign of RCA occlusion. Similarly, Huey et al[18] demonstrated ST depression in lead I in 22% of their LCX patients and 59% of their RCA patients, whereas Kontos et al[19] reported this finding in 28% of their LCX patients and 58% of their RCA patients.

Among the 45 patients with RCA occlusion 21 patients had proximal RCA occlusion and 24 patients had distal RCA occlusion. Different criteria analysed to determine proximal site of occlusion in RCA were ST↑>1mm in V_4R, ST↑V_1 and ST↓V_3/ST ↑LIII <0.5. ST↑>1mm in V_4R was present in 15 patients and in 6 patients with distal RCA occlusion, also in 3 patients with LCx occlusion. ST↓V_3/ST ↑LIII<0.5 was also present in 15 patients. Both these criteria have highest sensitivity (71.4%) in diagnosing proximal RCA occlusion whereas the ST elevation in V_1had maximum specificity.

That is ST elevation in V_1present only in 1 patient with proximal RCA occlusion and none of the patients with other site of occlusion. These findings were also consistent with Glancy et al.

And in patients with localizing culprit lesion in distal RCA, the ratio of ST depression in V3/ST elevation in lead III between 0.5–1.2 had maximum specificity and ST coving V_4R without ST elevation had the maximum specificity.

In patients with LCx occlusion ST elevation in Lead II> Lead III was the most sensitive and ratio of ST↓V_3/ST ↑LIII >1.2 was the most specific criteria.

Any health personal involved in emergency care should have in depth knowledge of assessing ECG in ST elevation MI. ECG assessment that is relevant to emergency primary care physicians are:

1. Proximal LAD coronary artery occlusion in anterior myocardial infarction.
2. Patients at higher risk, grade III of ischemia or ST depression in V4–V6, indicating multivessel disease in inferior acute myocardial infarction.
3. Right ventricular infarction accompanying acute inferior myocardial infarction.

It is important for emergency physicians to identify a very proximal LAD coronary artery occlusion in acute anterior myocardial infarction. If the infarct site is proximal to the first diagonal branch of the LAD artery, a large portion of the left ventricle is at risk for infarction including the anteroseptal, anterosuperior, anterolateral, and apical regions. Such high risk patients may require urgent transfer to the cardiac catheterisation laboratory for primary percutaneous coronary intervention or immediate treatment in the emergency department with a thrombolytic agent.

In patients with acute inferior myocardial infarction a second ECG recorded with right ventricular leads, as it is not unusual to see isoelectric ST segments in the right precordial leads. If ST segment elevation of 1 mm is observed in lead V4R, the diagnosis of right ventricular infarction can be made and no further right precordial.

ECGs need to be recorded. It is important to identify patients with right ventricular infarction because hypotension in these patients is usually caused by inadequate filling of the left ventricle and by the poorly contracting right ventricle. Therefore, treatment should be aimed at augmenting ventricular filling by volume expansion and avoiding diuretics and nitrates. Such treatment is contrary to the treatment of cardiogenic shock due to pump failure, as occurs with large infarctions of the left ventricle.

SUMMARY: Our study consists of 126 patients who has presented to our emergency department with acute chest pain, ECG showing ST segment elevation and culprit vessel was identified from ECG based on the various criteria.

Among 126 patients, 70 patients were diagnosed to have anterior wall MI and 56 patients with inferior wall MI.

In patients with anterior wall MI culprit vessel occlusion were identified as proximal to S1, proximal to D1, Distal to S1 and Distal to D1 based on different ECG criteria.

Among the five criteria compared in lesion proximal to S1, ST depression in inferior leads >1mm had maximum sensitivity in localizing occlusion proximal to S1.

ST depression in inferior leads >1mm had maximum sensitivity in localizing occlusion in proximal D1 as well.

ST depression in V_5 has maximum specificity in occlusion proximal to S1 and presence of Q wave in aVL in patients with occlusion proximal to D1 respectively.

Absence of ST depression in the inferior leads is the most sensitive criteria in occlusion distal to S1 as well as in D1 in anterior wall myocardial infarction.

While presence of Q waves lead V4-V6 has the maximum specificity in occlusion distal S1 and ST depression in lead aVL has maximum specificity in occlusion distal to D1.

In 56 patients with Inferior wall MI 45 patients had occlusion in RCA and 11 patients in LCx In RCA occlusion 21 patients had proximal RCA occlusion and 24 had distal RCA occlusion.

Patients with proximal RCA occlusion criteria ST↑>1mm in V_4R, ST↓V_3/ST↑LIII<0.5 were equally sensitive. The ST elevation in V_1 had maximum specificity.

While in patients with distal RCA occlusion the ratio of ST depression in V3/ST elevation in lead III between 0.5–1.2 had maximum specificity and ST coving V_4R without ST elevation had the maximum specificity.

In patients with LCx occlusion ST elevation in Lead III> Lead II was the most sensitive and ratio of ST↓V_3/ST ↑LIII >1.2 was the most specific criteria.

CONCLUSION: The admission ECG in patients with ST elevation acute myocardial infarction is valuable not only for determining who should and should not receive early reperfusion treatment, but also for providing information regarding the location and extent of acute myocardial injury.

By reflecting the pathophysiology of the myocardium during acute ischemia important information to guide management and determine prognosis can be derived from the electrocardiogram. Electrocardiographic markers of proximal coronary-artery occlusion identify relatively large myocardial infarctions that benefit most of the early and complete revascularization strategies such as angioplasty.

These criteria although are not the substitute of invasive procedure for differentiating the culprit artery in acute myocardial infarction, but provides an economical, reliable and quick method of differentiating infarct related artery in acute inferior myocardial infarction.

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