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Original Article

Internal Medicine Section

Angiographic Localisation of Culprit Vessel in Non ST Elevated Acute Coronary Syndrome

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ABSTRACT

Introduction: Coronary Artery Disease (CAD) is a major cause of mortality and morbidity. Among Acute Coronary Syndrome (ACS), Non ST Elevated Acute Coronary Syndrome (NSTE-ACS) continues to increase. Unlike ST Elevated Myocardial Infarction (STEMI), association of ischaemic changes in Electrocardiogram (ECG) with culprit lesion localisation in NSTE-ACS has not been well reported.

Aim: To investigate the association between ECG abnormalities and angiographic localisation of culprit vessel in patients of NSTE-ACS.

Materials and Methods: This observational, prospective study was conducted in SCB Medical college and Hospital, Cuttack, Odisha, India, from December 2019 to November 2020. A total of 200 eligible patients of newly diagnosed NSTE-ACS were included. Demographic and risk factor assessment, clinical examination and routine blood investigations were done. All patients had an admission Electrocardiogram (ECG), Echocardiography (Echo) and Coronary Angiography (CAG) done within 72 hours of admission. Admission ECG was associated with CAG to assess predictive value in localisation of culprit vessel. Sensitivity, Specificity, Positive Predictive Value (PPV), Negative Predictive Value (NPV), Likelihood Ratio (LR), pre and post-test odds of individual ECG findings were assessed. Statistical Package for the Social Sciences (SPSS) version 24.0 was used for statistical analysis.

Results: Sensitivity, specificity, PPV, NPV, LR+, pretest odds and post-test odds of anterior wall, inferior wall, lateral wall and augmented Vector Right (aVR) group ECG changes in predicting Left Anterior Descending (LAD), Right Coronary Artery (RCA), Left Circumflex Artery (LCX) and Left main or Triple Vessel Disease (LM/TVD) as culprit artery were 75.9%, 90.5%, 91.7%, 73.1%, 7.96, 1.38 and 10.98; 69.1%, 91.5%, 87.8%, 76.9%, 8.14, 0.89 and 7.21; 64%, 93%, 87.3%, 77.4%, 9.10, 0.75 and 6.86 and 69.2%, 96.3%, 90%, 86.7%, 18.66, 0.48 and 8.98, respectively. Sensitivity, specificity, PPV and NPV of anterior and lateral, inferior and lateral and anterior and inferior wall ECG changes in predicting LAD and LCX, RCA and LCX and LAD and RCA as culprit vessels were 75.0%, 82.6%, 27.27% and 97.43%; 47.0%, 88.5%, 27.5% and 94.73% and 53.3%, 83.2%, 20.51% and 95.65%, respectively. The ECG was normal in 31% of which Myocardial Infarction with Non Obstructive Coronary Artery (MINOCA) (34%) and Single Vessel Disease (SVD) (30.6%) were prevalent. The MINOCA were mostly seen in normal ECG pattern.

Conclusion: An ECG is a moderately sensitive but highly specific parameter in predicting LAD, LCX, RCA and LM/TVD as culprit vessels in Non ST Segment Elevation Myocardial Infarction-Acute Coronary Syndromes (NSTE-ACS). Double territory ECG changes have a poor association in predicting culprit vessel. However, a good association was noted for (anterior and lateral) wall ECG changes in predicting LAD and LCX as culprit arteries.

Keywords: Angiography, Echocardiography, Electrocardiogram, Myocardial infarction, Unstable angina

INTRODUCTION

The Coronary Artery Disease (CAD) is a major cause of mortality and morbidity across the world. In 2015, nearly 17.9 million people died of Cardiovascular Disease (CVD) representing 31% of global deaths of which an estimated 7.2 million were due to CAD [1]. Coronary artery disease prevalence continues to rise in India with rapid epidemiologic transition with a projected rise in mortality in 117% and 105% men and women respectively between 1990-2020 [2].

Among Acute Coronary Syndromes (ACS), the fraction of Non ST Segment Elevation Myocardial Infarction-Acute Coronary Syndromes (NSTE-ACS) continues to increase while that of STEMI is declining [3]. According to Kerala and Himachal Pradesh ACS registries, NSTE-ACS accounts for 63% and 54.5% of ACS cases, respectively [4,5].

The standard 12 lead ECG has long been a reliable clinical tool for diagnosis and treatment of Acute Myocardial Infarction (AMI). Unlike ST Elevated Myocardial Infarction (STEMI), association of ischaemic changes in Electrocardiogram (ECG) with culprit lesion localisation in NSTE-ACS has not been well reported [6]. Electrocardiogram can provide valuable information to improve clinical decision making and targeted early invasive therapy. Initial catheterisation of presumed culprit artery in NSTE-ACS would reduce the time to perfusion and can transform into mortality benefit for the patients [7].

Thus, the authors conducted, that, the present study to investigate the association between ECG abnormalities and angiographic localisation of culprit vessel in patients of NSTE-ACS.

MATERIALS AND METHODS

This prospective, observational study was conducted at the Department of Cardiology, SCB Medical College and Hospital, Cuttack, Odisha, India, from December 2019 to November 2020. Study was conducted as per the principles of Declaration of Helsinki and International Committee on Harmonization of Good Clinical Practice (ICH-GCP). All the research participant signed the informed consent. The study protocol was approved by the Institute Ethics Committee. Total 200 patients were included.

Inclusion criteria: All consecutive, newly diagnosed cases of NSTE-ACS admitted in the Department of Cardiology who gave written informed consent for being a participant in the study were included.

Exclusion criteria: Cases with age <18 or >80 years, previous history of ACS, angioplasty or Coronary Artery Bypass Graft (CABG), contraindications to Coronary Angiogram (CAG), CKD and allergy to iodinated compounds, lack of consent for CAG ST/T changes due to secondary causes were excluded.

Patients with presence of acute onset typical ischaemic symptoms with normal ECG or ST segment depression or T-wave inversion or flattening with rise/fall of troponin level with atleast one value above 99th percentile upper reference limit were diagnosed as Non ST Segment Elevation Myocardial Infarction- Acute Coronary Syndromes (NSTE-ACS) [3] and those with normal troponin level were classified as Unstable Angina (UA) [8].

ST segment depression ≥0.05 mV (0.5 mm) and T wave inversion ≥0.1 mV (1 mm) with prominent R wave or R/S ratio >1, in two contiguous leads, were considered significant ST/T changes.

Study Procedure

Aforementioned admission ECG changes in V1-6, I,aVL were taken as anterior wall; V5-6, I, aVL were taken as lateral wall; II, III, aVF were taken as inferior wall. When these ECG changes were noted in anterior, inferior and lateral wall, they correspond to LAD, RCA and LCX territories respectively and ST elevation in aVR with ST depression in other leads was considered as aVR group ECG changes correspond to LMCA or Triple Vessel Disease (TVD). The MINOCA was defined as patients with Acute Myocardial Infarction (AMI) with absence of obstructive disease (< 50% obstruction) on angiography in any major epicardial vessel.

A standard 12 lead ECG on admission along with echocardiography was undertaken for Regional Wall Motion Abnormality (RWMA) and LV function. Blood parameter including Troponin T (quantitative) and lipid profile were measured. Demographic variables, risk factors and ST/T changes in different wall territory on admission ECG were recorded. Detailed clinical examination and risk stratification was done. Accordingly Guideline Directed Medical Therapy (GDMT) was ensured to all.

Patients underwent diagnostic Coronary Angiography (CAG) within 72 hours of admission depending upon the risk stratification and subject to availability of cath lab. Coronary angiography was done with Siemens Artis Zee machine via the femoral or radial route for evaluation and detection of location and severity of lesions and accordingly treatment was advised by the treating physician. Optimal angiographic views were taken to assess the lesion characteristics and quantification of diameter stenosis by Quantitative Coronary Angiography (QCA). Obstructive lesions were observed in two orthogonal views and diametric stenosis of atleast 50% were considered significant.

Culprit Lesion Determination

Patients who had Single Vessel Disease (SVD), the culprit artery localisation was straight forward. In multi vessel disease, the lesion was taken as culprit if the Regional Wall Motion Abnormality (RWMA) on echocardiography matched myocardial segments supplied by the artery containing the significant lesion or if an obvious eccentric thrombus with scalloped or overhanging edges and a narrow neck was noted [7]. Thrombus was indicated by globular intraluminal mass with rounded or polypoid shape, or haziness of lesion. After locating the culprit artery, the patient's admission ECG was compared with CAG finding to study whether it has any predictive value for identifying culprit artery.

STATISTICAL ANALYSIS

All the data collected were entered into the Microsoft Excel 2007 software and further analyzed in Statistical Package for the Social Sciences (SPSS) version 24.0 (IBM Chicago). All the categorical variables were expressed in terms of number/frequency and percentages. Association between two categorical variables were obtained by using Chi-square test. All the continuous variables were expressed in terms of mean and standard deviation. Significance level in comparison of means between two groups were obtained by independent sample t-test test/Mann-Whitney U test while between more than two groups were obtained by using Analysis of Variance (ANOVA). A p-value <0.05 was taken as statistically significant. Sensitivity, Specificity, Positive Predictive Value (PPV), Negative Predictive Value (NPV), Likelihood Ratio (LR), pretest and post-test odds of individual ECG findings were assessed.

RESULTS

Out of 200 Non ST elevated acute coronary syndrome patients, Non ST Elevation Myocardial Infarction (NSTEMI) was 78% (n=156) and UA 22% (n=44). The mean age was 57.61±8.44 years with 69.5% (n=139) being male. Family history of CAD (44, 22%), smoking [38% (n=76)], diabetes mellitus [36.5% (n=73)], hypertension [(103, dyslipidemia [10% (n=20)] and tobacco chewing [30.5% (n=61)] were the risk factors identified. The mean BMI was 23.94±2.33 kg/m^2. The ST/T changes in anterior wall, inferior wall and lateral wall were 48% (n=96), 37% (n=74) and 31.5% (n=63) respectively. aVR group ECG changes was 25% (n=50) and normal ECG in 31% (n=62) [Table/Fig-1].

Parameter	n (%)
Age (years)	57.61±8.44
Sex (male)	139 (69.5)
Body mass index (Kg/m^2)	23.94±2.33
Non ST elevation acute coronary syndrome	
Non ST elevation myocardial infarction	156 (78.0)
Unstable angina	44 (22.0)
Risk factors	
Hypertension	103 (51.5)
Smoking	76 (38.0)
Diabetes mellitus	73 (36.5)
Tobacco chewing	61 (30.5)
Family history of CAD	44 (22.0)
Dyslipidemia	20 (10.0)
ECG changes	
Anterior wall territory	96 (48%)
Inferior wall territory	74 (37%)
Lateral wall territory	63 (31.5%)
aVR group	50 (25%)
Normal	62 (31%)
Culprit artery on CAG	
Left Anterior Descending (LAD) artery	116 (58%)
Right Coronary Artery (RCA)	94 (47%)
Left Circumflex Artery (LCX)	86 (43%)
Left Main (LMCA)/TVD	65 (32%)
MINOCA	24 (12%)

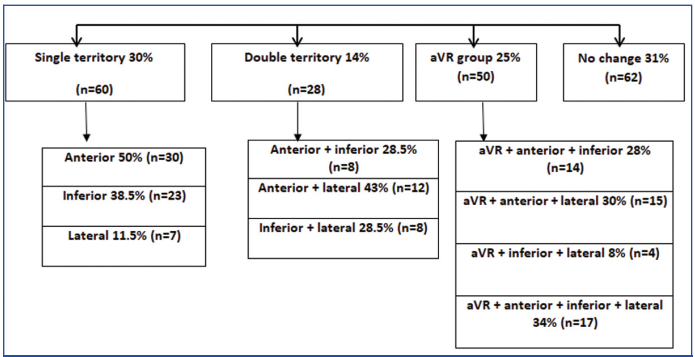
[Table/Fig-1]: Baseline characteristics (n=200).
MINOCA: Myocardial infarction in non obstructive coronary artery; TVD: Triple vessel disease

Patients with ECG finding suggestive of single territory, double territory, aVR group and normal ECG were 30% (n=60), 14% (n=28), 25% (n=50) and 31% (n=62) respectively. Among single territory, anterior wall ECG changes (50%) were most common followed by inferior wall (38.5%) and lateral wall (11.5%) [Table/Fig-2].

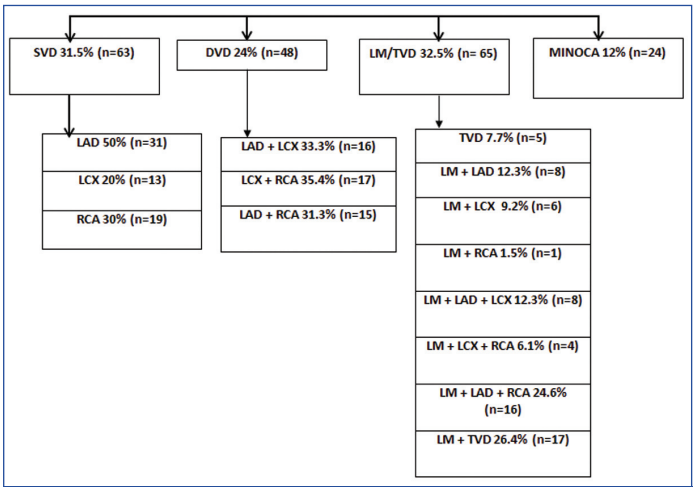
Patients with Single Vessel Disease (SVD), Double Vessel Disease (DVD), Left Main (LM) or Triple Vessel Disease (TVD) and Myocardial Infarction with Non Obstructive Coronary Arteries (MINOCA) were 31.5% (n=63), 24% (n=48), 32.5% (n=65) and 12% (n=24) respectively. In SVD, LAD was the most common culprit artery (50%) followed by RCA (30%) and LCX (20%) respectively. In patients with DVD, lesions in LAD and LCX, LCX and RCA and LAD and RCA were almost equally distributed in 33.3%, 35.4% and 31.3% respectively [Table/Fig-3].

In patients with normal ECG, SVD, DVD, TVD and MINOCA were 30.6% (n=19), 14.5% (n=9), 20.9% (n=13) and 34% (n=21) respectively. Among SVD, LAD was the most common culprit artery

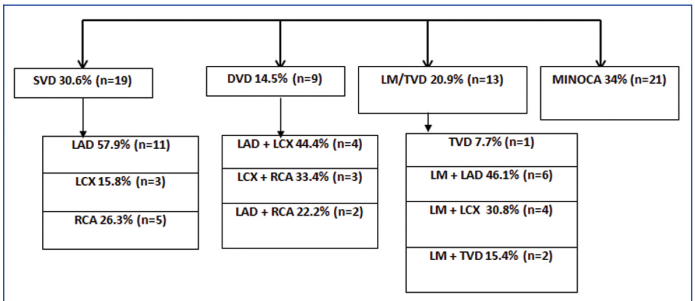
57.9% (n=11). MINOCA were mostly seen with normal ECG pattern in 87.5% (21 out of 24 patients) [Table/Fig-4].



[Table/Fig-2]: ECG profile of Non ST elevated acute coronary syndrome (N=200).



[Table/Fig-3]: Angiographic profile of NSTE-ACS (n=200).
SVD: Single vessel disease; DVD: Double vessel disease; LM: Left main; TVD: Triple vessel disease; MINOCA: Myocardial Infarction with non-obstructive coronary arteries; LAD: Left anterior descending; RCS: Right coronary artery; LCX: Left circumflex artery



[Table/Fig-4]: Angiographic profile of NSTE-ACS with normal ECG (n=62).
SVD: Single vessel disease; DVD: Double vessel disease; LM: Left main; TVD: Triple vessel disease; MINOCA: Myocardial Infarction with non-obstructive coronary arteries; LAD: Left anterior descending; RCS: Right coronary artery; LCX: Left circumflex artery

The sensitivity, specificity, PPV, NPV, LR+, pretest odds and post-test odds of anterior wall, inferior wall, lateral wall and aVR group ECG changes in predicting LAD, as culprit artery were 75.9%, 90.5%, 91.7%, 73.1%, 7.96, 1.38 and 10.98; RCA as culprit artery were 69.1%, 91.5%, 87.8%, 76.9%, 8.14, 0.89 and 7.21; LCX as a culprit artery were 64%, 93%, 87.3%, 77.4%, 9.10, 0.75 and 6.86 and LM/TVD as culprit artery were 69.2%, 96.3%,90%, 86.7%, 18.66, 0.48 and 8.98 respectively. Sensitivity, specificity, PPV and NPV of anterior and lateral, inferior and lateral and anterior and inferior wall ECG changes in predicting LAD and LCX, as culprit vessels were 75.0%, 82.6%, 27.27% and 97.43%; RCA and LCX as culprit vessels were 47.0%, 88.5%, 27.5% and 94.73% and LAD and RCA as culprit vessels were 53.3%, 83.2%, 20.51% and 95.65% respectively [Table/Fig-5].

DISCUSSION

In the present study, authors assessed the validity of ECG changes in predicting the culprit artery compared to the gold standard i.e. coronary angiography in patients with NSTE-ACS.

The mean age was 57.62±8.45 years and was nearly 10 years earlier than that reported in Western studies [9-11]. A male preponderance was observed that is probably due to the gender bias and atypical presentation in females, as shown in INTERHEART study and its South Asian cohort 76% and 85% respectively [12,13]. Risk factor profiles of our NSTE-ACS patients was similar to the CREATE registry [14] and NE registry [15]. Tobacco chewing as a risk factor in 1/3rd of present study cases should be noted with a caution considering the large prevalence of tobacco chewing in our part of the country. The NSTE-ACS was more prevalent compared to unstable angina (78% vs 22%) similar to previous studies [15,16]. It could be due to the increased reliance on highly sensitive biomarkers of myocardial necrosis.

In the present study, anterior wall (48%) ECG changes were most common followed by inferior wall (37%), lateral wall (31.5%) and aVR group ECG changes (25%) similar to 3D-EINSTEIN study [16] and Sanaani A et al., [6]. The ECG was normal in 31% similar to 3D-EINSTEIN study [16]. In these patients, MINOCA (34%) and SVD (30.4%) were most prevalent, which is similar to Hiremath RG et al., [17] (25% and 37.5%), Teixeira R et al., [18] (26.2% and 32.7%) and Moustafa A et al., [19] (23.4% and 53.2%). LAD (57.9%) was the most common culprit vessel in normal ECG group similar to Moustafa A et al., [19] (55.6%).

On the basis of coronary angiogram, LM/TVD (32.5%) was most prevalent followed by SVD (31.5%), DVD (24%) and MINOCA (12%), as seen in Sidhu NS et al., [20] study in 37.7%, 32.2%, 16.7% and 18.9% patients respectively. However, contrasting

Variables	Anterior wall	Inferior wall	Lateral wall	AVR group	Anterior and lateral	Inferior and lateral	Anterior and inferior
Territory ECG changes	96 (48%)	74 (37%)	63 (31.5%)	50 (25%)	44 (22%)	29 (14.5%)	39 (19.5%)
Culprit artery from respective territory ECG changes	88	65	55	45	12	8	8
Total culprit artery on CAG, n (%)	LAD 116 (58%)	RCA 94 (47%)	LCX 86 (43%)	LMCA/TVD 65 (32.5%)	LAD and LCX 16 (8%)	RCA and LCX 17 (8.5%)	LAD and RCA 15 (7.5%)
Sensitivity	75.9%	69.1%	64%	69.2%	75%	47%	53.3%
Specificity	90.5%	91.5%	93%	96.3%	82.6%	88.5%	83.2%
PPV	91.7%	87.8%	87.3%	90%	27.2%	27.5%	20.5%
NPV	73.1%	76.9%	77.4%	86.7%	97.4%	94.7%	95.6%
LR+	7.96	8.14	9.1	18.66	4.31	3.82	3.17
Pretest odds	1.38	0.89	0.75	0.48	0.086	0.093	0.081
Post-test odds	10.98	7.21	6.86	8.98	0.344	0.355	0.256

[Table/Fig-5]: Validity of ECG changes in prediction culprit Artery (n=200).
*LAD: Left anterior descending; RCA: Right coronary artery; LCX: Left circumflex; LMCA: Left main coronary artey; TVD: Triple vessel disease; PPV: Positive predictive value; NPV: Negative predictive value; LR: Likelihood ratio

to the present observation, Desai AP and Bhagarhatta R, [21], Iqbal F and Barkataki JC, [15] and Moustafa A et al., [19] reported, SVD as most common culprit vessel affecting 47.6%, 35.3% and 53.4% patients respectively. Overall, LAD (58%) was the most common culprit vessel followed by RCA (47%), LCX (43%) and LMCA/TVD (32.5%) similar to the observations reported by Sharma R et al., [13], Moustafa A et al., [19] and Deora S et al., [22]. Among SVD, LAD (50%) was the most common culprit vessel similar to Desai AP and Bhagarhatta R, [21] and Sanaani A et al., [6] study.

In patients with LAD as culprit artery (n=116), anterior wall ECG changes were seen in 75.9% (n=88). Sensitivity, specificity, PPV and NPV of anterior wall ECG changes predicting LAD as culprit artery were 75.9%, 90.5%, 91.7% and 73.1% respectively. This was similar to that reported by Desai AP and Bhagarhatta R, [21], who observed 73.60%, 93.50%, 90%, 81.80%; Khanra D et al., [16] who observed 72.41%, 90%, 89.36%, 73.77% and Sanaani A et al., [6] who found 90.9%, 98.5%, 96.77%, 95.77% respectively.

In patients with RCA as culprit artery (n=94), inferior wall ECG changes were present in 69.1% (n=65). Sensitivity, specificity, PPV and NPV of inferior wall ECG changes predicting significant RCA as culprit artery were 69.1%, 91.5%, 87.8% and 76.9% respectively, similar to that reported by Desai AP and Bhagarhatta R, [21] was 63.10%, 93.70%, 90.50%, 72.70% and Khanra D et al., [16] was 51.42%, 84.93%, 62.06%, 78.48% and Sanaani A et al., [6] was 93.33%, 90.80%, 63.63%, 98.75% respectively.

In patients with LCX as culprit artery (n=86), lateral wall ECG changes were present in 64% (n=55). Sensitivity, specificity, PPV and NPV of lateral wall ECG changes predicting significant LCX as culprit artery were 64.0%, 93.0%, 87.3% and 77.4% respectively similar to that reported by Desai AP and Bhagarhatta R, [21] was 63.10%, 93.70%, 90.50%, 72.70% and by Khanra D et al., [16] was 28.57%, 91.78%, 62.5%, 72.80% respectively [21].

In patients with LMCA/TVD as the culprit artery (n=65), aVR group ECG changes were present in 69.2% (n=45). Sensitivity, specificity, PPV and NPV of aVR group ECG changes predicting significant LMCA/TVD lesion were 69.2%, 96.3%, 90.0% and 86.7% respectively similar to that reported by Desai AP and Bhagarhatta R, which was 66.60%, 98.50%, 91.40% and 92.50% respectively [21].

The present study established ECG as a moderately sensitive but highly specific parameter in predicting LAD, LCX, RCA and LM/TVD as culprit vessels in NSTE-ACS. ECG changes predicting LAD (75.9%) as culprit artery were most sensitive followed by LM/TVD (69.2%), RCA (69.1%) and LCX (64%) whereas prediction of LM/TVD (96.3%) as culprit artery was most specific followed by LCX (93%), RCA (91.5%) and LAD (90.5%). Double territory ECG changes have a poor association in predicting culprit vessel in view of poor positive predictive value. However, a good association was noted for anterior and lateral wall ECG changes in predicting LAD and LCX as culprit arteries with a sensitivity, specificity and NPV of 75%, 82.6% and 97.43% respectively. The moderate sensitivity of ECG for localisation of culprit arteries in present study could be due to individual variation in coronary anatomy, presence of collateral circulation, left ventricular hypertrophy and inadequate representation of posterior wall.

Limitation(s)

Some of the limitations of present study were small sample size, single centre study, inter-observer variation in analysis of ECG, Echo and CAG. The observer performing the CAG was not being blinded to the ECG and Echo findings which could have resulted in some bias in reporting.

CONCLUSION(S)

Admission ECG as a modality in predicting culprit artery in Non ST Elevated Acute Coronary Syndrome has acceptable validity. Compared to double territory lesion, single coronary involvement can be predicted in a better way using ECG in NSTE-ACS. Larger scale studies on a more diverse population could further establish its role in efficient management of NSTE-ACS.

REFERENCES

[1] Roth GA, Johnson C, Abajobir A, Abd-Allah F, Abera SF, Abyu G et al. Global, regional, and national burden of cardiovascular diseases for 10 causes, 1990 to 2015. J Am Coll Cardiol. 2017;70(1):01-25.

[2] Guha S, Sethi R, Ray S, Bahl VK, Shanmugasundaram S, Kerkar P et al. Cardiological Society of India: Position statement for the management of ST elevation myocardial infarction in India. Indian heart journal. Indian Heart J. 2017;69 Suppl 1(Suppl 1):S63-97.

[3] Giugliano R.P., Braunwald E. Non ST Elevation Acute Coronary Syndromes. Braunwald's Heart Disease: A textbook of cardiovascular medicine, 11th Edition, 2018; 60:1181-1208.

[4] Padinhare PM, Mathew R, Harikrishnan S, Krishnan MN, Zachariah G, Joseph H et al. Presentation, management and outcomes of 25748 acute coronary syndrome admissions in Kerala, India: Results from Kerala ACS Registry. European Heart J. 2013;34:121-29. Doi: 10.1093/eurheart/ehs219.

[5] Negi PC, Merwaha R, Panday D, Chauhan V, Guleri R. Multicenter HP ACS Registry. Indian Heart J. 2015;68:118-127. Doi: 10.1016/j.ihj.2015.07.027.

[6] Sanaani A, Yandrapalli S, Jolly G, Paudel R, Cooper HA, Aronw WS. Correlation between electrocardiographic changes and coronary findings in patients with acute myocardial infarction and single vessel disease. Ann Transl Med. 2017;5(17):347. Doi: 10.21037/atm.2017.06.33.

[7] Kamal AM, Soltan GM, Ali MAA. Electrocardiographic prediction of culprit artery in acute ST-segment elevation myocardial infarction. Memoufia Med J. 2018;31(4):1463-69. Doi: 10.4103/mmj.mmj_51_16.

[8] Moliterno DJ, Januzzi JL. Evaluation and management of Non ST segment elevation myocardial infarction. Hurst's The Heart. 14e 2017;39: 995-1016.

[9] Basit H, Malik A, Huecker MR. Non ST Segment Elevation Myocardial Infarction. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2020 Jan. [Updated 2020 Oct 15]. Available from: https://www.ncbi.nlm.nih.gov/books/NBK513228/.

[10] Canto JG, Rogers WJ, Goldberg RJ, Peterson ED, Wenger NK, Vaccarino V, et al. NRMI Investigators. Association of age and sex with myocardial infarction symptom presentation and in-hospital mortality. JAMA. 2012;307(8):813-22.

[11] Nourjah P. National Hospital Ambulatory Medical Care Survey: 1997 emergency department summary. Adv Data. 1999;(304):01-24.

[12] Yusuf S, Hawken S, Ounpuu S, Dans T, Avezum A, Lanas F, et al. Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries (the INTERHEART study): Case-control study. Lancet. 2004;364(9438):937-52. Doi: 10.1016/S0140-6736(04)17018-9. PMID: 15364185.

[13] Sharma R, Bhairappa S, Prasad S R, Manjunath CN. Clinical characteristics, angiographic profile and in hospital mortality in acute coronary syndrome patients in south indian population. Heart India. 2014;2:65-69.

[14] Xavier D, Pais P, Devereaux PJ, Xie C, Prabhakaran D, Reddy KS, et al. Treatment and outcomes of acute coronary syndromes in India (CREATE): A prospective analysis of registry data. Lancet. 2008;371 (9622):1435-42.

[15] Iqbal F, Barkataki JC. Spectrum of acute coronary syndrome in North Eastern India- a study from a major center. Indian Heart J. 2016;68:128-131. Doi: 10.1016/j.ihj.2015.07.040. Epub 2016 Jan 18. PMID: 27133318; PMCID: PMC4867010.

[16] Khanra D, Sinha SK, Tiwari P, Razi MM, Aggrawal P, Soni S, et al. Three dimensional echocardiography in non ST elevation acute coronary syndrome in North India (3D-EINSTEIN)- A single centre prospective study. J Pract Cardiovasc Sci. 2019;5:94-101.

[17] Hiremath RG, Kabade DM, Malkiwodeyar PK, Khatawkar AV, Hubballi VB, Majigoudra G, et al. A study of correlation between electrocardiographic findings and coronary angiogram in patients of acute coronary syndrome. J. Evid. Based Med. Healthc. 2018;5(51):3514-19. Doi: 10.18410/jebmh/2018/717.

[18] Teixeira R, Lourenço C, António N, Monteiro S, Baptista R, Jorge E, et al. The importance of a normal ECG in non-ST elevation acute coronary syndromes [A importância de um EGC normal em síndromes coronarianas agudas sem supradesnivelamento do segmento ST]. Arq Bras Cardiol. 2010;94(1):25-33. Translated from Portuguese. Doi: 10.1590/s0066-782x2010000100006. PMID: 20414523.

[19] Moustafa A, Abi-Saleh B, El-Baba M, Hamoui O, AlJaroudi W. Anatomic distribution of culprit lesions in patients with non-STsegment elevation myocardial infarction and normal ECG: Cardiovasc Diagn Ther. 2016;6(1):25-33. Doi: 10.3978/j.issn.2223-3652.2015.10.05.

[20] Sidhu NS, Rangaiah SKK, Ramesh D, Veerappa K, Manjunath CN. Clinical Characteristics, Management Strategies, and In-Hospital Outcomes of Acute Coronary Syndrome in a Low Socioeconomic Status Cohort: An Observational Study From Urban India. Clin Med Insights Cardiol. 2020;14:1179546820918897.

[21] Desai AP, Bhagarhatta R. Localization of Angina Related Artery by Admission ECG in Unstable Angina and NSTE-ACS Patients. Journal of Hypertension and Cardiology 2016; 2(1):10-16. Doi: 10.14302/issn.2329-9487.jhc-13-335.

[22] Deora S, Kumar T, Ramalingam R, NanjappaManjunath C. Demographic and angiographic profile in premature cases of acute coronary syndrome: Analysis of 820 young patients from South India. Cardiovasc Diagn Ther. 2016;6(3):193-98.

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