# ST-Segment Depression in Lead aVR as a Predictor of Culprit Artery in Acute Inferior Wall ST-Segment Elevation Myocardial Infarction

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## Abstract

Introduction: The ECG is of great importance in the initial evaluation of patients suspected of having acute coronary syndrome (ACS). According to the current AHA/ACC and ESC guidelines especially in ST-segment elevation ACS, the ECG from the acute phase contains essential information about the site and size of the area at risk, aiding in the selection of appropriate therapy for the individual patient. Objective: To determine the diagnostic accuracy of STsegment depression of Imm/more in lead aVR in predicting the culprit artery in ST-elevated inferior myocardial infarction. Methods: Cross sectional observational study was contract at Department of Cardiology, Dhaka Medical college Hospital (DMCH), Dhaka over a period of one year between September 2019 to August 2020. Minimum of 30 subjects were taken as sample within the study period; but the number of cases may be increased in the basis of availability in both Group. Group I: Patients with ST-segment depression in lead  $aVR \ge l$  mm. Group II: Patients with isoelectric ST-segment or with ST segment depression in lead aVR <1 mm. The study included patients who presented with ST-segment elevated inferior myocardial infarction and got admitted in CCU (DMCH) and undergo coronary angiogram within hospitalization period. Results: A total of 30 ST elevated MI (STEMI) patients were taken as study population. The study population were divided into two groups : group I and group II. Group I included patients with ST-segment depression in lead  $aVR \ge 1$  mm and group II included patients with isoelectric ST-segment or with ST segment depression in lead aVR <1 mm. ST-segment was depressed  $\geq$ 1mm in avR lead among 56.7% (n=17) of study population who were categorized as group I. ST-segment was isoelectric or depressed <1mm among 43.3% (n=13) of study population who were categorized as group II. Majority of the study population was in age group 51-60 years in group I (26.7%) and >60 years in group II (23.3%). Age distribution was statistically similar in both groups. Majority respondents were male in both groups (76.50% in group I and 69.20% in group II). Sex distribution was statistically similar in both groups. Hypertension, Diabetes Mellitus, Dyslipidemia, Smoking, Family History of CAD and obesity were statistically similar in both groups. Majority of the patients admitted in hospital with a history of chest pain for 7-12 hours duration. In group I, the most common culprit artery for AISTMI was LCX (Left circumflex) artery. In group II, the most common culprit artery was RCA (Right Coronary Artery). Significant difference was noticed between two groups regarding culprit artery where group I tended to be observed with LCX artery frequently. Among 16 cases where culprit artery was LCX artery, ECG could detect 14 through ST depression (≥1 mm) in aVR lead. Among 14 cases where LCX artery was not culprit, ECG could detect 11 through isoelectric ST segment or ST depression (<1mm). ST depression  $\geq$ 1mm in aVR lead showed 87.5% sensitivity, 78.57% specificity, 82.35% PPV (Positive Predictive Value), 84.61% NPV (Negative Predictive Value) and 83.33% accuracy in the detection of LCX as the culprit artery in acute inferior STEMI. Conclusion: Consistent to the findings of present study, ST depression in lead aVR is suggestive of LCX occlusion with good sensitivity and specificity. But this study was conducted a limited sample population in a single center. So, before drawing final conclusion, more extensive study should be done.

Keywords: St-Segment Depression, Culprit Artery, Acute Inferior Wall, St-Segment Elevation Myocardial Infarction.

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# I. Introduction

The ECG is of great importance in the initial evaluation of patients suspected of having acute coronary syndrome (ACS). According to the current AHA/ACC and ESC guidelines especially in ST-segment elevation ACS, the ECG from the acute phase contains essential information about the site and size of the area at risk, aiding in the selection of appropriate therapy for the individual patient. The conventional ECG records body surface potentials from the horizontal axis in an orderly fashion from right to left across the body surface. Thus in a normal subject, an orderly progression in ECG waveform morphology such as R wave amplitude is seen from lead V1 to V6. In contrast, the conventional ECG records the frontal axis in a less orderly fashion with a 600 gap between lead I & lead II and a 900 gap between lead III & lead aVR. Furthermore, the conventional ECG displays first the bipolar leads (I, II, III) and then the unipolar leads (aVR, aVL, aVF) and thus no clear progression in waveform morphology is seen. However, display of lead aVR (-1500) in inverted format as lead (+300) bridges the gap between lead I (00) and lead II (600). ECG information about the culprit artery in inferior myocardial infarction is important because the prognosis and therapeutic strategy may vary between LCX and RCA related inferior myocardial [1]. Patients with AMI who undergo primary angioplasty in whom LCX is the infarct related vessel have a significantly larger infract and worse clinical outcome compared in patients in whom the RCA is involved [2]. Early identification of the culprit artery in patients with symptoms of AMI can reduce the time to reperfusion in percutaneous coronary intervention and permit a better risk stratification. More recently, ST-segment depression in lead aVR has been suggested as a predictor of LCX artery involvement [3]. ST-segment depression in aVR was also shown to be associated with significantly impaired myocardial perfusion [4]. In Bangladesh several studies have yet been carried out to correlate STsegment deviation in various ECG leads with the angiographic finding to find out the culprit artery in STsegment elevated inferior myocardial infarction. Soumen, C. [5] stated that ST-segment depression in lead aVR is predictor of LCX as infarct related artery in ST-segment elevated inferior wall AMI. The sensitivity is 89.8% and specificity is 95.5%. The positive and negative predictive values of the diagnostic modalities are 93.6% and 95.5% respectively. Hossain, et al [6] found that ST-segment depression in V4-V6 in acute INF-MI confers a greater likelihood of multivessel disease where ST-segment depression in V1-V3 is commonly associated with single vessel disease. According to Alam [7], ST-segment morphology in lead V4R can help to predict culprit artery in acute inferior myocardial infarction. ST-segment elevation with upright T wave predicts proximal RCA lesion, while if the ST-segment is in isoelectric line it predicts distal RCA lesion, ST-segment depression with T inversion predicts LCX as culprit artery. Rahman, et al [8] said that ST-segment -depression of 1 mm or more in lead I predicts RCA lesion in acute ST-segment elevated inferior myocardial infarction. ST-segment depression in aVL of 1 mm or more also predicts RCA lesion. RCA can also be thought to be the culprit artery if ST elevation in lead III>II. If ST-segment elevation in lead III is not greater than lead II and ST-segment depression in lead aVL is not greater than lead I, LCX can be suspected as culprit artery.

## **II.** Materials And Methods

Study design: Cross sectional observational study.

Place of study: Department of Cardiology, Dhaka Medical college Hospital (DMCH), Dhaka, Bangladesh.

**Study population:** The study included patients who presented with ST-segment elevated inferior myocardial infarction and got admitted in CCU (DMCH) and undergo coronary angiogram within hospitalization period.

Period of study: The study was conducted over a period of one year between September 2019 to August 2020.

**Sample Size (n):** As the study was conducted over a limited period of time, a minimum of 30 subjects were taken as sample within the study period; but the number of cases may be increased in the basis of availability.

#### **Study Group:**

Group I: Patients with ST-segment depression in lead aVR  $\geq 1$  mm. Group II: Patients with isoelectric ST-segment or with ST segment depression in lead aVR <1 mm.

#### Inclusion criteria

 $\Box$  patients who were diagnosed with acute inferior-wall STEMI based on 12-lead resting ECG, as evidenced by  $\geq 1 \text{ mm ST}$  elevation in at least two of the inferior leads II, III and aVF and who subsequently underwent coronary angiography during index hospitalization.

#### Exclusion criteria

□ Patients with history and/or evidence of any previous myocardial infarction.

 $\hfill\square$  Associated acute anterior myocardial infarction

□ Patients with history of PCI or CABG.

 $\hfill\square$  Patients with factors potentially confounding the E.C.G interpretation-

- a) Bundle branch block
- b) WPW syndrome
- c) Paced rhythm
- d) Low voltage E.C.G
- e) left ventricular hypertrophy

f) Electrolytes abnormality and certain other conditions that can influence ST segment on ECG (e.g. any valvular heart disease, congenital heart disease, primary myocardial or pericardial disease, acute or chronic heart failure, hypothermia, receiving amiodarone treatment etc).

 $\hfill\square$  Patients with malignant disease, advanced liver and kidney diseases.

□ Patient who were documented to have lesions in both RCA and LCX after CAG.

#### **Study Procedure:**

All patients of Acute Myocardial infarction inferior admitted in the department of cardiology, Dhaka Medical college hospital within the study period were approached for this study. A total of 30 patients were selected according to inclusion and exclusion criteria. Information from the patients and relatives were collected through preformed pro forma. Patients were evaluated based upon history, Clinical examination, investigation. 12 lead resting ECG was done on admission and once daily by ADVANCED 12 lead ECG machine with paper speed of 25 mm/sec and voltage of 10 mm/mV. For inferior STEMI the inclusion criteria will be ST-segment elevation  $\geq$  1mm in at least two of three inferior leads (II, III and aVF). The ST-segment was measured manually by slide calipers at 60 ms after the "J" point. The magnitude of ST segment changes in lead aVR was relative to the previous TP segment as a baseline which was obtained from the first ECG after admission. Echocardiography was done for each patient after admission and when needed by PHILIPS Affiniti70C machine and Ejection fraction was calculated by Simpon's Method. Other baseline investigations including random blood sugar, serum creatinine, serum lipid profile, serum electrolytes & screening tests for coronary angiogram was done for each patient. All patients were subjected to coronary angiography during index hospitalization. Two investigators from Dhaka Medical College Hospital analyzed all angiograms blinded to the clinical and ECG data. The culprit artery was defined. Coronary artery stenosis of more than 70% were defined as an obstructive lesion. Multivessel coronary artery disease was defined as having two or more coronary arteries with obstructive lesions. The coronary flow was determined using the TIMI flow grading system. Based on the origins of the posterior descending artery (PDA) and the posterolateral branch (PL), the RCA were classified as dominant RCA (both PDA and PL provided by RCA), non-dominant RCA (both PDA and PL provided by LCX), or co-dominant RCA (PDA provided by RCA and PL provided by LCX). A PL branch was defined as 'large' when it was  $\geq 2$  mm in diameter. When more than one lesion was present in the coronary tree, the site of the culprit lesion was determined by the appearance of complete obstruction of the artery or by the more detailed angiographic characteristics of the lesion, including presence of either residual thrombus or ulcerated plaque (decreased contrast density). Coronary angiography findings were classified in 5 different groups. The first 3 groups represent the LAD, the RCA or the LCX as the culprit artery. The fourth group included cases with the left main coronary artery as culprit and/or multivessel disease without evident culprit lesions. Finally, patients with normal angiography or non-significant disease without evident culprit lesion were classified together. Angiographic findings were evaluated by two experienced angiographer blinded to the results of the ECG findings.

#### Statistical analysis:

All statistical analysis was performed using the statistical package for social science (SPSS) program, version 22 for Windows. Continuous parameters were expressed as mean  $\pm$ SD and categorical parameters as frequency and percentage. Comparisons between groups (continuous parameters) was done by Student t test (parametric variable) and Mann-Whitney-U test (non parametric variable). Categorical parameters were compared by Chi-Square test. Moreover, diagnostic accuracy test was done according to standard protocol. The significance of the results as determined in 95.0% confidence interval and a value of p <0.05 was consider to be statistically significant.

## III. Results

A total of 30 ST elevated MI (STEMI) patients were taken as study population. The study population were divided into two groups : group I and group II. Group I included patients with ST-segment depression in lead aVR  $\geq$ 1 mm and group II included patients with isoelectric ST-segment or with ST segment depression in lead aVR <1 mm.



Figure 1: Distribution of study population by status of ST-segment in lead aVR (n=30)

ST-segment was depressed  $\geq 1$ mm in avR lead among 56.7% (n=17) of study population who were categorized as group I. ST-segment was isoelectric or depressed <1mm among 43.3% (n=13) of study population who were categorized as group II.

Tuble 1. Distribution of study population by age (1-50)					
Age group (years)	Group I	Group II	Total	p value	
	n(%)	n(%)	n(%)		
≤41	1 (3.3)	0	1 (3.3)		
41-50	3 (10)	4 (13.3)	7 (23.3)		
51-60	8 (26.7)	2 (6.7)	10 (33.3)	.201*	
>60	5 (16.7)	7 (23.3)	12 (40)		
Mean±SD	55.23±7.12	59.15±8.64	56.93±7.93	.184**	

 Table 1: Distribution of study population by age (n=30)

\*Chi-squared Test ( $\chi^2$ ) and Unpaired student t\*\* test was performed to compare between two groups

Majority of the study population was in age group 51-60 years in group I (26.7%) and >60 years in group II (23.3%). Age distribution was statistically similar in both groups.

Majority respondents were male in both groups (76.50% in group I and 69.20% in group II). Sex distribution was statistically similar in both groups.



Table 2: Distribution of risk factors in study population (n=30)					
Risk factors	Group I	Group II	Total	p value*	
	n (%)	n (%)	n (%)		
Hypertension	8 (47.1)	6 (46.2)	14 (46.7)	.431	
Diabetes Mellitus	5 (29.4)	4 (30.8)	9 (30)	.182	
Dyslipidemia	6 (35.3)	4 (30.8)	10 (33.3)	.794	
Smoking	12 (70.6)	11 (84.6)	23 (76.7)	.368	
Family history of CAD	6 (35.3)	4 (30.8)	10 (33.3)	.794	
Obesity	3 (17.6)	6 (46.2)	9 (30)	.091	

\*Chi-squared Test ( $\chi^2$ ) was performed to compare between two groups

\*Chi-squared Test ( $\chi^2$ ) was performed to compare between two groups CAD (Coronary Artery Disease)

Risk factors of the study population are tabulated in table 2. Hypertension, Diabetes Mellitus, Dyslipidemia, Smoking, Family History of CAD and obesity were statistically similar in both groups.

Table 3: Duration of chest	pain of study po	pulation (n=30)
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Duration of chest pain	Group I	Group II	Total	р
(hours)	n (%)	n (%)	n (%)	value
3-6	4 (23.5)	3 (23.1)	7 (23.3)	420
7-12	11 (64.7)	10 (76.9)	21 (70)	.430
>12	2 (11.8)	0	2 (6.7)	
Mean±SD	9.58±3.64	8.69±2.78	9.20±3.27	.467

\*Chi-squared Test ( $\chi^2$ ) and Unpaired student t test was performed to compare between two groups

Majority of the patients admitted in hospital with a history of chest pain for 7-12 hours duration.

Table 4:	Clinical	features	among	study	po	pulation	(n=30)	
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<b>Clinical Features</b>	Group I n (%)	Group II n (%)	Total	p value
Symptoms				
Shortness of breath	10 (58.8)	6 (46.2)	16 (53.3)	.491
Sweating	17 (100)	13 (100)	30 (100)	1.00
Nausea/Vomioting	12 (70.6)	6 (46.2)	18 (60)	.176
Signs				
Tachycardia	6 (35.3)	6 (46.2)	12 (40)	.547
High BP	9 (52.9)	5 (38.5)	14 (46.7)	.431
Raised RR	12 (70.6)	6 (46.2)	18 (60)	.176
Basal crepitation	7 (41.2)	4 (30.8)	11 (36.7)	.421

\*Chi-squared Test ( $\chi^2$ ) was performed to compare between two groups

Clinical features of study population are tabulated below. Symptoms and signs were statistically same in both groups.

Table 5. Investigation infungs of study population (n=50)						
Investigation findings	Group I	Group II	Total	р		
				value		
LVEF (%)*	46.41 ±11.14	43.69 ±11.30	45.23 ±11.10	.516 <sup>\$</sup>		
S. creatinine (mg/dl)*	1±0.16	1.08 ±0.16	1.04 ±0.16	.346 <sup>\$</sup>		
Random blood sugar (mmol/L)*	8.55±3.50	7.53±2.98	8.11±3.27	.807 <sup>\$</sup>		
Troponin I (ng/nl)**	8.95(.5-27.46)	9.58 (1.13-52.40)	9.26(.15-52.40)	.391 <sup>\$\$</sup>		
Total cholesterol (mg/dl)**	261 (136-372)	242 (137-298)	248 (136-372)	.100 <sup>\$\$</sup>		
Triglyceride (mg/dl)**	157 (59-465)	150 (59-512)	152 (59-512)	.203\$\$		
HDL (mg/dl)*	35±6.54	35.46±6.17	35.13±6.28	.346\$		
LDL (mg/dl)*	117.82±40.17	104±37.77	111.83±39.11	.805 <sup>\$</sup>		

 Table 5: Investigation findings of study population (n=30)

<sup>\$</sup>Unpaired student t test and <sup>\$\$</sup>Man Whitney U test was performed to compare between two groups, \*Variables were expressed as mean±SD. \*\* Variables were expressed as median (minimum-maximum), HDL: High Density Lipoprotein, LDL: Low Density Lipoprotein

Table 6: Distribution of respondents by culprit artery (responsible for acute inf STEMI) (n=30)

Culprit Artery	Group I n (%)	Group II n (%)	Total n (%)	p value
LCX (Left Circumflex) artery	14 (82.4)	2 (15.4)	16(53.3)	
RCA (Right Coronary Artery)	3 (17.6)	6 (66.7)	7 (23.3)	
LAD (Left Anterior Descending) Artery	0	1 (3.3)	3 (10)	
Left main coronary artery and/or multivessel	0	2 (15.4)	2 (6.7)	.003
No culprit evident lesion/normal angiography	0	2 (15.4)	2 (6.7)	

\*Chi-squared Test ( $\chi^2$ ) was performed to compare between two groups

In group I, the most common culprit artery for AISTMI was LCX (Left circumflex) artery. In group II, the most common culprit artery was RCA (Right Coronary Artery). Significant difference was noticed between two groups regarding culprit artery where group I tended to be observed with LCX artery frequently.

Table 7: Diagnostic accuracy of aVR (ST depression) in detecting the culprit artery of AISTEMI (n=30)

	Culprit artery of angiogram		
ST depression in aVR lead of ECG	LCX artery	Other arteries	Total
ST depression ≥1mm	14	3	TF+FP
• —	TF	FP	17
Isoelectric ST-segment/	FN	TN	FN+TN
ST depression <1 mm	2	11	13
Total	TP+FN	FP+TN	30
	16	14	

TP= True positive; TN= True negative; FP= False positive; FN= False negative

Among 16 cases where culprit artery was LCX artery, ECG could detect 14 through ST depression ( $\geq 1$  mm) in aVR lead. Among 14 cases where LCX artery was not culprit, ECG could detect 11 through isoelectric ST segment or ST depression (<1mm).



Figure 3: Diagnostic Accuracy of ST depression (≥1mm) in aVR lead of ECG in detecting LCX as the culprit artery of AISTEMI (n=30)

ST depression  $\geq 1$ mm in aVR lead showed 87.5% sensitivity, 78.57% specificity, 82.35% PPV (Positive Predictive Value), 84.61% NPV (Negative Predictive Value) and 83.33% accuracy in the detection of LCX as the culprit artery in acute inferior STEMI.

# IV. Discussion

This study was conducted in the Department of Cardiology, Dhaka Medical college Hospital among 30 diagnosed patients of STEMI. In aVR lead, ST-segment depression was  $\geq$ 1mm among 56.7% of patients who were categorized as group I. On other hand, ST-segment was isoelectric or had depression <1mm in 43.3% of study population who were categorized as group II. Average age of study patients was 56 years. Study

conducted by Sarkar et al. found mean age of STEMI patients 53.75±11.64 (SD) years [9]. Kim et al. also found the mean age of their study cases 57.07±12.40 years (SD) [10]. These study findings are nearly similar to present study. Age plays a vital role in the deterioration of cardiovascular functionality, resulting in an increased risk of cardiovascular disease (CVD) in older adults [11,12]. The prevalence of CVD has also been shown to increase with age, in both men and women, including the prevalence of atherosclerosis, stroke and, myocardial infarction [13]. Male predominance was observed in present study with 76.50% and 69.20% male patients in group I and II accordingly. Sharma et al. also found male predominance with 72.83% male patients in a similar study which corroborates with present study findings [14]. Positive effect of endogenous oestrogens, and relatively lower cholesterol level in female may play the key role to prevent MI in female especially in their premenoposal life [15,16]. Moreover, in Bangladesh, female patients are getting less priority in society. So, the frequency hospital admitted MI patients can be gender biased. History of hypertension, diabetes mellitus, dyslipidemia, smoking, family history of CAD (Coronary Artery Disease) and obesity were present in 46.7%, 30%, 33.3%, 76.7%, 33.3% and 30% of patients accordingly. Similar study conducted by Kim et al. found hypertension, diabetes mellitus, and smoking among 42.68%, 21.96%, and 50.61% of their study population [10]. Another similar study by Reda et al. found hypertension, diabetes, smoking, dyslipidemia and family history of CAD among 54.50%, 38%, 50%, 38.50% and 21.50% of their patients accordingly [17]. All the findings are nearly concordant to the findings of this study. Palomba et al. and Keto et al. stated the link of the development of CVD, such as hypertension, diabetes, and obesity [18,19]. Mean duration of chest pain of study patients was 9.20±3.27 (SD) hours. About 70% of them had history of chest pain from 7-12 hours. Mean left ventricular ejection fraction was 45.23±11.10 (SD) %. Presenting clinical features of the patients were sweating, nausea/vomiting, tachycardia, high BP, Raised respiratory rate and basal crepitation. Mean troponin I was 13.97±14.92 (SD) ng/ml. No significant difference was noticed between group I and II patients. In this study, ST depression (≥1mm) in aVR lead was observed in 56.7% of patients. Among them, LCX artery was observed as culprit artery in 82.4% patients. That means ECG (ST depression in aVR lead) couldn't detect 17.6% cases correctly. Diagnostic accuracy test showed 87.5% sensitivity, 78.57% specificity and 83.33% accuracy. In a similar type study by Kanei et al., showed greater specificity (86%) and lesser sensitivity (53%) than present study [20]. Patil et al. observed that 50% with left circumflex infarction (LCx) lesion had ST depression in aVR lead. So, they suggested that ST-segment depression in lead aVR is valuable for differentiating RCA from those with LCx in the inferior STEMI [21]. Khanal et al. also stated 87.5% specificity and 83% sensitivity of ST depression in aVR lead in diagnosing LCX [22]. Pourafkari et al. observed 66.67% sensitivity and 55.56% specificity for identifying LCX lesions [23]. The percentage of sensitivity and specificity in our study is greater than previous studies. Limited sample size and lack of generalization of the study might be the reason of this dissimilarity. The main finding of our study is that aVR depression represents myocardial infarction involving either the LCx artery. aVR depression represents the infarct of the apical and inferolateral walls which is usually supplied by the posterolateral branch of either the RCA or LCx itself. However, a large posterolateral branch being necessary to supply the inferolateral wall did not hold true for patients in the LCx group because the LCx itself provides blood supply to the inferolateral wall. Accordingly, aVR depression suggests the involvement of LCx or a large RCA with a large posterolateral branch. Patients with LCx infarcts that present as STEMI generally have large LCx arteries, so as to involve the inferior wall. LCx infarcts can often present as non-STsegment elevation myocardial infarction or can be electrocardiographically silent on the traditional 12-lead ECG.

## V. Limitations

✓ Single centered study

✓ Sample size was small

## VI. Conclusion

Consistent to the findings of present study, ST depression in lead aVR is suggestive of LCX occlusion with good sensitivity and specificity. But this study was conducted a limited sample population in a single center. So, before drawing final conclusion, more extensive study should be done.

## VII. Recommendations:

Further clinical study with larger sample size involving multiple center is recommended

#### **References:**

- Gul, E.E., Nikus, K.C., Sonmez, O. & Kayrak, M., 2011. Dilemma In Predicting The Infarct-Related Artery In Acute Inferior Myocardial Infarction: A Case Report And Review Of The Literature. Cardiology Journal, 18(2), 204–6.
- [2] Rasoul, S., Ottervanger, J. P., Bilo, H. J., Timmer, J. R., Van't Hof, A. W., Dambrink, J. H., ... & Zijlstra, F. (2007). Glucose Dysregulation In Nondiabetic Patients With St-Elevation Myocardial Infarction: Acute And Chronic Glucose Dysregulation In Stemi. Neth J Med, 65(3), 95-100.

- [3] Tong-Wen, S., Le-Xin, W. & Yan-Zhou, Z., 2007. The Value Of Ecg Lead Avr In The Differential Diagnosis Of Acute Inferior Wall Myocardial Infarction. Internal Medicine, 46(12), 795–799.
- [4] Kosuge, M., Kimura, K., Ishikawa, T., Hongo, Y., Shigemasa, T., Sugiyama, M., Et Al., 2001. Implications Of The Absence Of St-Segment Elevation In Lead V 4r In Patients Who Have Inferior Wall Acute Myocardial Infarction With Right Ventricular Involvement. Clinical Cardiology, 24(3), 225–230.
- [5] Soumen, C., 2014. Prediction Of Culprit Artery In St-Segment Elevated Inferior Wall Myocardial Infarction By St-Segment Depression In Lead Avr. Theses (Md. Cardiology). National Heart Foundation Hospital & Research Institute (Nhfh & Ri), Dhaka.
- [6] Hossain, A.S., Siddique, M.A., Rahman, M.M., Rahman, M.M., Ahmed, M.K., Roy, G.C., Et Al., 2012. Correlation Of Angiographic Findings Between Right (V1 To V3) Versus Left (V4 To V6) Precordial St Segment Depression In Acute Inferior Myocardial Infarction. University Heart Journal, 7(2), 76–81.
- [7] Alam, M., Ullah, M., Ulabbi, S., Haque, M., Uddin, R., Mamun, M., Et Al., 1970. Prediction Of The Site Of Coronary Artery Lesion In Acute Inferior Myocardial Infarction With Right Sided Precordial Lead (V4r). Cardiovascular Journal, 4(1), 46–52.
- [8] Rahman, M.T., Rahman, S., Haque, K.M.H. & Haque, S.A., 2005. Angiographic Correlation With Admission Electrocardiogram In Acute Inferior Wall Myocardial Infarction. Bangladesh J Medicine, 1(16), 5–11.
- [9] Sarker, B., Islam, M.A., Sana, N., Rahman, M., Das, C., Khan, M., Et Al., 2016. Socio-Demographic Characteristics Of Acute Myocardial Infarction Patients In Bangladesh. Taj: Journal Of Teachers Association, 29(1), 16–20.
- [10] Kim, D.Y., Wala, Z., Islam, S., Islam, R. & Ahn, M., 2019. Clinical Characteristics And Outcomes Of St-Segment Elevation Myocardial Infarction In A Low Income Setting In Rural Bangladesh. Ijc Heart And Vasculature, 23(1), 1-376.
- [11] Curtis A.B., Karki R., Hattoum A., S.U., 2018. Arrhythmias In Patients ≥ 80 Years Of Age: Pathophysiology, Management, And Outcomes. J. Am. Coll. Cardiol., 71(1), 2041–2057.
- [12] North, B.J. & Sinclair, D.A., 2012. The Intersection Between Aging And Cardiovascular Disease Brian. Circ Res., 110(8), 1097– 1108.
- [13] Yazdanyar A., N.A.B., 2009. The Burden Of Cardiovascular Disease In The Elderly: Morbidity, Mortality, And Costs. Clin. Geriatr. Med., 25(1), 563–577.
- [14] Sharma A, Kumar R, Ashotra S, T.S., 2016. Comparative Evaluation Of Clinical Profile, Risk Factors, And Outcome Of Acute Myocardial Infarction In Elderly And Nonelderly Patients. Heart India, 4(3), 96–99.
- [15] Dunlay, S.M. & Roger, V.L., 2012. Gender Differences In The Pathophysiology, Clinical Presentation, And Outcomes Of Ischemic Heart Failure. Current Heart Failure Reports, 9(4), 267–276.
- [16] Maas Ah, A.Y., 2010. Gender Differences In Coronary Heart Disease. Netherlands Heart Journal, 18(12), 598-603.
- [17] Redaa, A.A. &, Mourd B. Minaa, A.N.T.H., 2017. Pattern Of Risk Factors And Management Strategies In Patients With Acute Coronary Syndrome. Menoufia Medical Journal 2017, 30(1), 657–662.
- [18] Palomba, S., Santagni, S., Falbo, A. & La Sala, G.B., 2015. Complications And Challenges Associated With Polycystic Ovary Syndrome: Current Perspectives. International Journal Of Women's Health, 7(1), 745–763.
- [19] Keto J., Ventola H., Jokelainen J., Linden K., Keinanen-Kiukaanniemi S., Timonen M., Ylisaukko-Oja T., A.J., 2016. Cardiovascular Disease Risk Factors In Relation To Smoking Behaviour And History: A Population-Based Cohort Study. Open Heart., 3(1), 1-38.
- [20] Kanei, Y., Sharma, J., Diwan, R., Sklash, R., Vales, L.L., Fox, J.T., Et Al., 2010a. St-Segment Depression In Avr As A Predictor Of Culprit Artery And Infarct Size In Acute Inferior Wall St-Segment Elevation Myocardial Infarction. Journal Of Electrocardiology, 43(2), 132–135.
- [21] Patil, V., Pandere, K., Damle, S. & Avhad, A., 2019. Utility Of Avr Electrocardiogram Lead For Identifying The Culprit Lesion In Patient With Acute Coronary Syndrome. Journal Of Datta Meghe Institute Of Medical Sciences University, 14(1), 383–90.
- [22] Khanal, R., Sayami, A., Gajurel, R., Shrestha, H., Thapa, S. & Sahi, R., 2018. Electrocardiographic Localization Of Infarct Related Coronary Artery In Acute Inferior Wall St Elevation Myocardial Infraction And In Hospital Outcome In Tertiary Cardiac Care Center. Nepalese Heart Journal, 15(1), 23–27.
- [23] Pourafkari, L., Tajlil, A., Mahmoudi, S.S. & Ghaffari, S., 2016. The Value Of Lead Avr St Segment Changes In Localizing Culprit Lesion In Acute Inferior Myocardial Infarction And Its Prognostic Impact. Annals Of Noninvasive Electrocardiology, 21(4), 389–396.