International Journal of Research in Medical Sciences
Pendse M et al. Int J Res Med Sci. 2016 Aug;4(8):3164-3171
www.msjonline.org
pISSN 2320-6071 | eISSN 2320-6012

DOI: http://dx.doi.org/10.18203/2320-6012.ijrms20162240

Research Article

Study of electrocardiographic differentiation between RCA and LCx occlusion in isolated inferior wall myocardial infarction

Manish Pendse*, Gayatri Pendse, Dhaval Dave, Prashant Kashyap

Department of Medicine, D.Y. Patil Hospital and Research Centre, Nerul, Navi-Mumbai, Maharashtra, India

Received: 07 July 2016
Accepted: 13 July 2016

***Correspondence:**
Dr. Manish Pendse,
E-mail: dhavald9488@yahoo.com

ABSTRACT

Background: Inferior myocardial infarctions account for 40 to 50% of all acute myocardial infarctions and are generally viewed as having a more favorable prognosis than anterior wall infarctions. The management, and in some instances, prevention of these complications, may be facilitated by early differentiation between AMI caused by RCA versus left circumflex coronary artery occlusion. These can be diagnosed from the electrocardiography (ECG) which remains a valuable and most widely used rational modality to diagnose and risk stratifying in an acute setting. The present study helps in Electrocardiographic differentiation between right coronary and the left circumflex coronary arterial occlusion in isolated inferior wall myocardial infarction.

Methods: The present study entitled "Electrocardiographic differentiation between right coronary and the left circumflex coronary arterial occlusion in isolated inferior wail myocardial infarction" was conducted from June 2007 to November 2009 at the Department of Medicine and Cardiology, Dr. D.Y. Patil Hospital and Research Center, Nerul, Navi Mumbai, Maharashtra, India.

Results: Out of 52 patients of acute inferior wall myocardial infraction, 41 were males and 11 were females. Thus the male to female ratio is 3.72:1. In the above table, the ST segment elevation in lead III was more than lead ii in42 patients. All these 42 patients were found to have RCA as the culprit vessel. The St Segment elevation in lead II was more than lead III in 9 patients. All these 9 patients were found to have LCx as the culprit vessel.

Conclusions: The incidence of acute inferior wall myocardial infarction is highest in age group of 50 to 59 years. The ST segment elevation in acute isolated inferior wall myocardial infarction was greater in lead III than in lead II when right coronary artery was the culprit vessel and vice versa when the left circumflex coronary artery was the culprit vessel. ST segment depression in lead I was common when the right coronary artery was the culprit vessel and not seen with left circumflex coronary artery occlusion. An upright T wave in lead V4R in acute isolated inferior wall myocardial infarction was common when the right coronary artery was the culprit vessel and not seen with left circumflex coronary artery occlusion.

Keywords: Inferior wall myocardial infarction, Electrocardiography, Right coronary artery, Left circumflex coronary artery

INTRODUCTION

Inferior myocardial infarctions account for 40 to 50 % of all acute myocardial infarctions and are generally viewed as having a more favourable prognosis than anterior wall infarctions.[1-3] Nearly 50% of patients suffering from inferior wall myocardial infarction, usually experience hemodynamic and bradycardiac complications.[1] Anterior wall myocardial infarction invariably occurred by occlusion of the left anterior descending coronary artery.

However inferior wall myocardial infarction can result from occlusion of either the right coronary artery or left circumflex coronary artery.[4] Right ventricular myocardial infarction and atrioventricular blocks are seen in 10-15 % patients with inferior MI due to RCA occlusion.[1,5,6] 'The management, and in some instances, prevention of these complications, may be facilitated by early differentiation between AMI caused by RCA versus left circumflex coronary artery occlusion.[7]

These can be diagnosed from the electrocardiography (ECG) which remains a valuable and most widely used rational modality to diagnose and risk stratifying in an acute setting.[4]

Previous theories have shown that the recording of lead V4R in the acute phase of an inferior wall MI can distinguish those patients with a proximal occlusion of the right coronary artery from those with a occlusion of the distal right or left circumflex coronary artery showing ST-T segment elevation >1mm in lead V4R. However, it is not possible to differentiate between occlusion of a distal right coronary artery and a left circumflex coronary artery using this criterion.[8]

In these patients (possible candidates for intracoronary thrombolytic therapy) coronary angiography might start with wrong coronary artery leading to a delay in reperfusion.[8] With the advent of intervention techniques such as angioplasty for acute MI, identification of culprit vessel is very important knowing the culprit vessel before angiography can help in risk stratifying and planning the procedure.

METHODS

The present study entitled "Electrocardiographic differentiation between right coronary and the left circumflex coronary arterial occlusion in isolated inferior wail myocardial infarction" was conducted from June 2007 to November 2009 at the Department of Medicine and Cardiology, Dr. D.Y. Patil Hospital and Research Center, Nerul, Navi Mumbai, Maharashtra, India.

Study design

This is a hospital based prospective observational study. Diagnosis of acute inferior wall myocardial infarction was based on the following criteria:

- Chest pain lasting for >30 minutes.
- ST elevation of $\geq$1 mm in at least two of the three inferior leads (II, III, aVE).
- Elevation of plasma total and MB isoenzyme creatinine kinase levels.
- All the patients included in the present study were thrombolysed confirming to the accepted criteria.

Sample size

Total of 52 patients out of 64 cases of acute inferior wall Myocardial Infarction were considered for the project.

Inclusion criteria

- Availability of at least two 13 lead ECGs during the first 12 hours of myocardial infarction, including the ECG on admission.
- Coronary angiography performed within 10 days of infarction.

Exclusion criteria

- Presence of ST elevation in V4R indicative of RVML
- Previous myocardial infarction.
- Previous coronary artery bypass surgery.
- Bundle branch block.
- Inability to diagnose the culprit vessel when both RCA and LCx showed complete occlusion on electrocardiography and angiography.
- Patients do not satisfying the inclusion criteria.
- Uninterpretable ECGs.

All the patients satisfying the inclusion criteria were included in the present study.

Following ECG criteria were analysed:

- Magnitude of ST segment elevation in leads II and III, measured 80 msec after the J point. V
- The ST segment in lead I, measured 80 msec after the J point, and was denoted as isoelectric, depressed or elevated, using the TP segment as the baseline; and
- T wave polarity in V4R was deemed upright, flat, inverted, or biphasic.

RCA occlusion was diagnosed when one or more of the following criteria were present during the acute stage.

St Segment elevation in lead III higher than in lead II

This was based on the logic that in RCA occlusion the infract area is centered at the posterobasal and diaphragmatic segments of the left ventricle (LV). Thus the maximum ST segment elevation would be reflected in lead III, which is directly oriented towards this region.

ST segment depression in lead I

Since lead I is oriented more than 900 away from the infarction. ST segment depression would be expected in this lead.

Upright T wave in lead V4R

LCx coronary artery occlusion was diagnosed when one or more of the following criteria were present in ECG during acute infarction: ST segment elevation in lead II higher than in lead III. The LCx and obtuse marginal branches, supplies the inferolateral and posterolateral areas of LV.

Thus an occlusion would result in infarction in the region, which would subtend the maximal ST segment elevation in the direction of lead II. ST segment elevation in lead I. lead I is directed within 90° of the maximal segment elevation vector. Hence ST segment elevation would be expected in this lead.

Coronary angiography analysis

All the patients in the study were subjected to coronary angiography with an average period of 7 days of myocardial infarction.

The angiogram is to be analyzed for infarct location, degree of stenosis, and number of diseased vessels.

Obstructive coronary arterial lesions are graded:

- Mild: <50% diameter stenosis
- Moderate: 50-99% diameter stenosis
- Total occlusion angiogram was taken as the gold standard to determine culprit vessel.

When the angiogram showed lesions in both RCA and LCx, the following criteria were used to identify the culprit vessel:

- Total occlusion
- Presence of thrombus
- Irregular ulcerated plaque

A culprit artery is determined from the correlation between the electrocardiograms and the characteristics of occlusion. (Occlusion due to thrombosis formation and / or ulceration with the diseased contrast density.) Patients were classified into two groups, those with occlusion of the RCA and those with occlusion of LCx.

Statistical analysis

Categorical variables are to be presented in percentages. The chi-square test is used to examine the significance' of the difference between the ECG findings. For small numbers, Fisher's Exact test is used.

A p value < 0.05 considered statistically significant. The sensitivity and specificity will be assessed for all criteria. Accuracy of different criteria is calculated by (true positives and true negatives)/total patients. Data is analyzed on statistical software STATA version 8.0.

RESULTS

Data was collected with the knowledge of patients and due consent. Those who were not fitting in the inclusion criteria were not taken in the study. This study was approved by the ethical committee of our institution. The analysis thus is made for the 52 patients who were included in the study. The following were the observations made in present study.

Table 1: Sex distribution.

Sex	Number of patients (n=52)
Male	41
Female	11
Male: Female ratio	3.72:1

Out of 52 patients of acute inferior wall myocardial infraction, 41 were males and 11 were females. Thus the male to female ratio is 3.72:1.

Table 2: Age distribution (n=52).

Age group in year	Males (n=41)	Female (n=11)
<40	1 (2.4%)	0 (0%)
40-49	11 (26.8%)	1 (9.1%)
50-59	14 (34.1%)	6 (54.5%)
60-69	12 (29.2%)	3 (27.3%)
>70	50.70±7.20	56.6±6.020

The table shows age wise distribution in male and females. The maximum number of patients were in group of 50-59 i.e.20 (40%), followed by 15 (30%) in age group of 60-69. The mean age of presentation in case of male was 50.70+7.20 years.

Table 3: ECG correlation with Culprit vessel.

ECG Findings ST segment elevation	Culprit Vessel		p value
	RCA(n=42)	LCx (n=10)	
III>II	42	0	<0.001
II>III	0	9	<0.001
II=III	0	1	NS

In the above table, the ST segment elevation in lead III was more than lead ii in42 patients. All these 42 patients were found to have RCA as the culprit vessel. The p value of ST segment elevation in group, III>II is statistically significant (p<0.001). The St segment elevation in lead II was more than lead III in 9 patients. All these 9 patients were found to have LCx as the culprit vessel. The p value of ST segment elevation in group, II>III is statistically significant (p<0.001). Out of all the patients the ST segment elevation in lead II was equal to in lead III in 1 patient and the culprit vessel was LCx. This value was not considered significant.

The Table 4 shows ST segment in lead I which was depressed in 34 patients, in which all were found to have RCA as the culprit vessel. The p value of ST segment depression in lead I is statistically significant (p<0.001). The ST segment in lead I was elevated in 7 patients, in which all were found to have LCx as the culprit vessel.

This data is statistically significant with a p value of <0.001. The ST segment was however isoelectric in 11 patients, out of which 8 had RCA and 3 had LCx as the culprit vessel. This difference is however statistically not significant with a p value of >0.068.

Table 4: ECG correlation with Culprit vessel in led I.

ECG Findings ST segment in led I	Culprit	Vessel	p value
	RCA (n=42)	LCx (n=10)	
Depressed	34 (80.9%)	0	<0.001
Elevated	0 (0)	7 (70%)	<0.001
Isoelectric	8 (19.04%)	3 (30%)	NS

Table 5: ECG correlation with Culprit Vessel (ECG Findings T wave in lead V4R).

ECG Findings T wave in lead V4R	Culprit	Vessel	p value
	RCA (n=42)	LCx (n=10)	
Upright	37 (88.1%)	0	<0.001
Inverted	0 (0)	8 (80%)	<0.001
Flat	5 (11.9%)	-	NS

The above table shows ECG correlation with culprit vessel with T wave changes in lead V4R. The T wave was upright in 37 patients, out of which all were found to have RCA as the culprit vessel. The p value of upright T wave in V4R is statistically significant (p<0.001).The T wave was inverted in 8 patients; all these 8 patients were found to have LCx as the culprit vessel. The p value is <0.001 which is statistically significant for inverted T wave in V4R. The T wave was flat in 7 patients in both RCA and LCx vessels. This difference is not statistically significant (p>0.068).

Table 6: Usefulness of different ECG signs in localizing the Culprit vessel.

ECG findings	Sensitivity	Specificity	Accuracy
RCA			
1. ST elevation lead III>II	95.5%	100%	98.5%
2. ST depression in lead I	82.2%	96.3%	86%
3. T wave upright in lead V4R	88.3%	100%	91.5%
LCx			
1. ST elevation lead III>II	92.6%	100	98%
2. ST depression in lead I	66.6%	100%	91%
3. T wave upright in lead V4R	77.8%	100%	94%

The above table shows sensitivity, specificity, and accuracy of ST segment elevation in lead III>II were 97.5%, 100% and 98.5% respectively in patients with RCA as the culprit vessel. The sensitivity, specificity and accuracy of ST depression in lead I were 82.2%, 96.3%, and 86% respectively. The sensitivity, specificity and accuracy of upright T wave in lead V4R in RCA patients were 88.3%, 100%, and 91.5% respectively.

The sensitivity, specificity, and accuracy of ST elevation in lead II>III were 92.6%, 100% and 98% respectively in LCx patients. The sensitivity, specificity, and accuracy of ST segment depression in lead I in LCx patients were 66.6%, 100% and 91% respectively. The sensitivity, specificity, and accuracy of inverted T wave in lead V4R in LCx patients were 77.8%, 100% and 94% respectively.

Table 7: Coronary angiography profile of Culprit vessels.

Vessel	Mild stenosis	Severe stenosis	Complete occlusion	Total
RCA	1	7	34	42
LCx	0	2	8	10

The above table shows out of 42 patients of RCA; 1,7, and 34 were found to have mild stenosis, serve stenosis and complete occlusion respectively. Among 10 patients of LCx; 2 have severe stenosis and 8 had complete occlusion. The maximum number of patients i.e. 42 had severe stenosis in both RCA and LCx.

DISCUSSION

This study was conducted from June 2007 to October 2009 at department of Medicine and Cardiology, Dr. D.Y. Patil Hospital, Nerul, Navi Mumbai. A total of 64 patients of 'Acute Inferior Wall Myocardial Infraction' admitted in CCU and ICCU, out of which 52 were fulfilling the inclusion criteria and underwent coronary angiography with an average period of 7 days, were included in this study. The mean time to coronary angiography from acute myocardial infraction was 7 days in present study.

Thus in this study, the following outcome was achieved:

- Out of 52 patients of acute inferior wall myocardial infarction analyzed, 41 were males and 11 were females. The male: female ratio being 3.72:1.
- The mean age of presentation was 55.70±7.20 for males and 56.60±6.20 for females.
- The mean time to coronary angiography in this study was 7 days.
- Out of 52 patients studied, 42 had RCA as the culprit vessel and 10 LCx. The ratio being 4.2:1.

Despite major advances in diagnostic tools over past few decades, ECG remains the corner stone in diagnosis of myocardial infarction. In fact the discovery of newer ECG signs and their validation has been facilitated by coronary angiography as demonstrated in present study. With the advent of interventional techniques such as angioplasty in acute myocardial infarction, identification of culprit vessel is very important. Knowing the culprit vessel before angioplasty can help in risk stratifying and planning the procedure. In addition to statistical probabilities, several ECG criteria help to identify the culprit vessel.

Each of these criteria is based on one or two of the anatomical facts. First, the myocaç1ial distribution of the RCA is slightly rightward in frontal plane, and consequently the current of injury resulting from its occlusion will be reflected more in lead III than in II. Conversely the distribution of LCx is slightly leftward in the frontal plane and the current of injury from its closure will be reflected more in lead II than III.

In present study the ST elevation in lead III was more than in lead II in 42 patients. All these patients had RCA as the culprit vessel. The p value of ST segment elevation in group 'lead III > lead II' is statistically significant i.e. <0.001. The sensitivity, specificity and accuracy of ST segment elevation in lead III>II were 97.5%, 100% and 98.5% respectively. The ST segment elevation in lead II was more than III in 9 patients. All these 9 patients had LCx as the culprit vessel. Thus the p value for group 'lead II >lead III 'is <0.001 which is statistically significant. The sensitivity specificity and accuracy of ST elevation in lead II>III were 92.6%, 100% and 98% respectively. The St Segment elevation was equal in both lead II and lead III in 1 patient and this patient had LCx as the culprit vessel. This data is not statistically significant.

Gupta A et al reported ST segment elevation in leads II and III as the most important sign to differentiate between the RCA and LCx occlusion.[4] Zimetbaum et al also recently reported a sign and found it useful in diagnosis proximal and distal RCA occlusions.[8] They reported a 97% positive predictive value of ST segment in lead III exceeding that in lead II for an occlusion in proximal or mid RCA. This sign was seen in only 12.5% patients with distal RCA occlusion in their study. However, in their study 37 patients with ST segment elevation in lead V4R were not excluded. He also revealed that ST segment elevation in lead V1 predicts proximal RCA occlusion.

These observations were also supported by Nair et al, who found ST segment elevation in lead III exceeded that in lead II in 26 patients and 23 of them had RCA as the culprit vessel.[9] In 3 patients, the LCx contained the culprit lesion.

Saw J et al showed St elevation in lead III> lead II is more sensitive than V4R in diagnosing RVMI.[10] It is an excellent screening tool for BVMI, given its universal availability on all electrocardiograms. Moreover III>II is significant predictor of in hospital mortality.

Chia BL et al reported an ST II/III ratio <1 (ST segment elevation in lead III>II) or ST depression in lead I are sensitive and specific markers of RCA occlusion, whereas an ST II/III ratio of 1 or more are sensitive and specific markers of LCx occlusion.[11]

Recently Fiol M et al reported ST segment elevation in lead II equal to or greater than in lead III, LCx is the likely culprit vessel because the injury vector is directed leftward.[12] Conversely if ST segment elevation is higher in lead III than II, RCA is likely culprit. This is also observer by Herz et al who found higher ST segment elevation in lead III than in lead II in 80% patients with occlusion of the RCA versus that of the LCx.[13] According to these investigators, the criterion of higher ST segment elevation in lead III than in lead II and a lower ST segment depression in lead aVL than in lead I is a sensitive and specific marker for RCA related AMI.

The criteria of higher ST elevation in lead III than in lead II was used not only to predict the culprit vessel but also to precisely locate the place of occlusion, proximal occlusion of RCA is usually associated with right ventricular involvement. Kusgue et a1 also reported the degree of ST segment elevation in inferior leads II, III, aVF particularly lead III was significantly greater in RCA occlusion than in the LCx.[14] These findings are in consistent with the results of previous studies.

The outcome of patients with acute inferior wall MI depends in large part on the occluded artery (RCA or LCx). Patients with acute myocardial infarction due to occlusion in the RCA usually have poorer outcome than do those with occlusion of the LCx, mainly to associated complications (hemodynamic changes due to right ventricular involvement) but true right ventricular infarction may be ascertained by assessment of hemodynamic parameters and confirmed by imaging techniques.

Therefore, it is important from a clinical point of view to predict the culprit vessel during evolving acut myocardial infarction. The RCA supplies blood mainly to the posterior part of the septum and the inferior part of the inferoposterior wall of the myocardium whereas the LCx supplies blood mainly to the posterior part of the inferoposterior wall and the posterior part of the lateral wall.

Because ST segment elevation in leads II, III and aVE is a key in diagnosing an evolving AMI that affects the inferoposterior wall of the heart, some characteristic ST segment changes in these leads, the reciprocal leads (I, aVL and V1 to V3) and the right precoedial leads (V3R to V5R) may provide information that identifies the culprit artery (RCA and LCx).

An injury vector in the presence of ST segment elevation in leads II, III, and aVF and a ST segment depression in lead I identifies the RCA as the culprit vessel. In this case, the injury vector is directed to the right and provokes a ST segment depression in lead I. A St Segment elevation in lead I suggests occlusion of the LCx, because the injury vector is directed leftward, within 900 of the maximum ST segment elevation vector. Hence ST segment elevation would be expected in this lead.

An isoelectric ST segment in lead I may be found with occlusion of the RCA or the LCx. The importance of ST segment depression in leads I and aVL (ST segment depression is usually greater in lead aVL than in lead I) as a marker of occlusion that the criterion of ST depression in lead I strongly favors' AMI caused by occlusion of the RCA.

In present study, the ST segment in lead I was depressed in 34 patients of which all had RCA as the culprit vessel. The p value of ST segment depression in lead I is statistically significant ($p<0.001$). The sensitivity specificity and accuracy of ST depression in lead I were 82.2%, 96.3% and 86% respectively.

The T segment in lead I was elevated in 7 patients out of which all had LCx as the culprit vessel. The p value of ST segment depression in lead I is statistically significant ($p<0.001$).The sensitivity, specificity and accuracy of ST depression in lead I were 66.6%, 100%, and 91% respectively.

Gupta A et al observed the ST segment in lead I was depressed in 65 patients of whom 64 patients were found to have RCA as the culprit vessel.[4] The sensitivity, specificity and accuracy were 85%, 96%, and 88% repectively. The ST segment in lead I was elevated in 14 patients and all these patients had LCx as the culprit vessel. The sensitivity, specificity and accuracy were 50%, 1009'o, and 87% respectively. Our findings were similar to Gupta A. These observations were supported by Chia et al, who found that occlusion of the RCA never presents ST segment elevation in lead I, whereas ST segment depression was present in most of the patients in whom the RCA was the culprit vessel.[11]

Baiery CN et al also observed that elevation or isoelctric ST segments in lead I in 100% of patients who had AMI of the inferior wall with occlusion of LCx and only 28% of patients who had occlusion of the RCA.[15] He revealed the presence of both ST segment elevation in two or more inferior leads and ST segment elevation in one or more lateral leads with an isoelectric or elevated ST segment in lead I identified circumflex coronary artery occlusion with sensitivity, specificity positive predictive value, and negative predictive value of 83%, 96%, 91%, and 93% respectively.

When these criteria were prospectively applied by him to an additional cohort of 19 secutive patients with inferior wall MI (5 with LCx and 14 with RCA occlusion), presence of LCx occlusion was predicted with a sensitivity of 80%, specificity of 93%, positive predictive value of 100% and negative predictive value of 93%. Kontus et al reported this sign with a figure of 23% for LCx patients and 58% for their RCA patients.[16]

Berry C et al showed ST segment depression in lead aVL and I only during occlusion of LCx.[17] Huey et al reported that ST segment depression in lead I was significantly less common in LCx in AMI.[18] Nair et al found ST segment elevation in lead Tin only three patients, each of whom had the culprit lesion in LCx.[9] Conversely, of 27 patients with an isoelectric (3 RCA) or depressed (22 RCA, 2 LCx) ST segment in lead I, 25 patients had the culprit lesion in RCA. Similar results were reported by Hasdai et al''.[19]

In lead V4R, which overlies the RV, the T wave is normally inverted. With ischemia in this region the T wave polarity is expected to change. In our study, the T wave was upright in 37 patients in lead V4R. All these patients had RA as the culprit vessel. The p value of upright T wave in V4R is statistically significant ($p<0.001$). The sensitivity specificity and accuracy of upright T wave in lead V4R in RCA patients were 88.3%, 100%, and 91.5% respectively. The T wave was inverted in lead V4R in 8 patients out of which all had LCx as the culprit artery 'The p value of this was statistically very significant (p<0.001). The sensitivity specificity and accuracy of inverted T wave in lead V4R in LCx patients were 77.8%, 100% and 94% respectively. Importantly though 'ST segment depression in lead I' and 'T wave polarity in lead V4R' were less sensitive all had excellent specificity.

These findings are supported by Gupta A et al.[4] He observed the T wave was upright in 68 patients in lead V4R. All these 68 patients were found to have culprit lesion in RCA. The sensitivity, specificity and accuracy of this sign were 89%, 100%, and 92% respectively. He found T wave was inverted in 22 patients in lead V4R who have LCx as the culprit vessel. The sensitivity, specificity and accuracy of this sign were 79%, 100% and 98% respectively.

CONCLUSION

Finally at the end of the study we have reached the following conclusions:

- 'The incidence of acute inferior wall myocardial infarction is highest in age group of 50 to 59 years. The average incidence being 55.70±720 for males and 56.60±6.20 for females.
- The ST segment elevation in acute isolated inferior wall myocardial infarction was greater in lead III than in lead II when right coronary artery was the culprit vessel and vice versa when the left circumflex coronary artery was the culprit vessel.
- ST segment depression in lead I was common when the right coronary artery was the culprit vessel and not seen with left circumflex coronary artery occlusion.
- An upright T wave in lead V4R in acute isolated inferior wall myocardial infarction was common when the right coronary artery was the culprit vessel and not seen with left circumflex coronary artery occlusion.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: The study was approved by the Institutional Ethics Committee

REFERENCES

1. Berger PB, Ryan TJ. Inferior myocardial infarction: high risk group. Circulation. 1990;81:401-11.
2. Effectiveness of intravenous thrombolytic treatment in acute myocardial infarction. Lancet. 1986;1:397-402.
3. ISIS-2 [second international study of infarct survival] collaborative group : Randomised trial of intravenous streptokinase, oral aspirin, both or either among 17187 cases of suspected acute myocardial infarction. Lancet. 1988;2:349-60.
4. Gupta A, Lokhandwala YY, Kerkar PG, Vora AM. Electrocardiographic Differentiation between right coronary and left circumflex coronary arterial occlusion in isolated inferior wall myocardial infarction. Indian Heart Journal. 1999;51:281-4.
5. Berger PB, Ruocco NA Jr, Ryan TJ, Jacobs AK, Zaret BL, Wackers FJ, et al Frequency and significance of right ventricular dysfunction during inferior wall left ventricular myocardial infarction treated with thrombolytic therapy [results from thrombolysis in myocardial infarction [TIMI] II trial] Am J Cardiol. 1993;71:1148-52.
6. Kinch JW, Ryan TJ. Right ventricular infarction, NEJM 1994;330:1211-7.
7. Assali AR, Herz I, Vaturi M, Adler Y, Solodky A, Birnbaum Y, et al, Electrocardiographic criteria for predicting the culprit artery in inferior wall myocardial infarction, Am J Cardiol. 1999;84:87-8.
8. Zimetbaum PJ, Krishnan S, Gold A, Carrozza JP, Josephson ME. Usefulness of ST segment elevation in lead III exceeding that in lead II for identifying the culprit vessel in inferior wall myocardial infarction. Am J Cardiol. 1988;81:918-9.
9. Nair R, Glancy D. ECG discrimination between right coronary and left circumflex coronary arterial occlusion in patients with acute inferior wall myocardial infarction. Value of old criteria and use of lead V4R. Chest. 2002;122:134-9.
10. Saw J, Davies C, Fung A. Value of ST segment elevation in lead III greater than lead II in inferior wall myocardial infarction for predicting the site of lesion. Bayes de Luna A, clinical electrocardiography: A textbook, 2nd Edition, Armonk NY. 1998:3-26.
11. Chia BL, Yip JW, Tan HC, Usefulness of ST segment III/II ratio and ST segment deviation in lead I for identifying the culprit vessel in acute inferior wall myocardial infarction. Am J Cardiol 2000;86:341-3.
12. Fiol M, Carrillo A, Cygankiewicz I, Ayestarán J, Caldés O, Peral V,et al, New criteria based on ST changes in 12 lead surface ECG to detect proximal vs distal right coronary artery occlusion in cases of acute inferior wall myocardial infarction. Ann Noninvasive Electrocardiol. 2004;9(4):383-8.
13. Herz I, Assali AR, Adler Y, Solodky A, Sclarovsky S. New electrocardiographic criteria for predicting either the right or left circumflex artery as the culprit coronary artery in inferior wall acute myocardial infarction. Am J Cardiol. 1997;80:1343-5.
14. Kosuge M, Kimura K, Ishikawa T, Hongo Y, Mochida Y, Sugiyama M, et al, New electrocardiographic criteria for predicting the site of lesion in acute inferior wall myocardial infarction. Am J Cardiol. 1998;82:1318-22.
15. Bairey CN, Shah PK, Lew AS, Hulse S. Electrocardiographic differentiation between

occlusion of left circumflex coronary artery and right coronary artery as a cause of inferior acute myocardial infarction. Am J Cardiol. 1987;60:456-9.

16. Kontos MC, Desai PV, Jesse RL. Usefulness of admission electrocardiogram for identifying the infarct related artery in inferior wall myocardial infarction. Am J Cardiol. 1997;79:182-4.
17. Berry C, Zaleucki A, Vovach R, Savage M, Goldbert S. Surface electrocardiogram ischaemia during coronary artery occlusion. Am J Cardiol. 1989;63:21-6.
18. Huey BL, Beller GA, Kaiser DL, Gibson RS. A comprehensive analysis of myocardial infarction due to left circumflex coronary occlusion: Comparison with infarction due to right coronary artery and left descending artery occlusion. J Am coll Cardiol. 1988;12:1156-66.
19. Hasdai D, Birnbaum Y, Herz I, Sclarovsky S, Mazur A, Solodky A. ST segment depression in lateral limb leads in inferior wall acute myocardial infarction. Implications regarding the culprit artery and site of obstruction. Eur Heart J. 1995;16:1549-53.

Cite this article as: Pendse M, Pendse G, Dave D, Kashyap P. Study of electrocardiographic differentiation between RCA and LCx occlusion in isolated inferior wall myocardial infarction. Int J Res Med Sci 2016;4:3164-71.