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Original Research Article

Electrocardiography changes in localizing the culprit vessel in acute myocardial infarction with angiographic correlations

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ABSTRACT

Background: The electrocardiogram is the primary diagnostic tool to evaluate a patient with chest pain and suspected myocardial infarction. Anterior wall infarction due to occlusion of proximal LAD has worse prognosis compared to distal or branch vessel lesions. Diagnosis of anterior/inferior/posterior or lateral MI is based on patterns of ST deviation and risk assessment based on absolute magnitude of ST segment deviation or the width of QRS complexes. Although coronary angiography is the gold standard for determining the infarct related artery in acute myocardial infarction, ECG can be useful tool in identifying the culprit artery involved at the primary care.
Methods: A prospective cross-sectional study was conducted in Aarupadai Veedu Medical College and Hospital. Standard 12 lead electrocardiograph were recorded for 50 patients at a speed of 25 mm/s and voltage of 10mm/mv. Patients who had acute inferior wall MI additional right pericardial leads were recorded (V3R and V4R). The recorded ECG was interpreted using the electrocardiographic algorithms of Zimet-baum PJ et al. An ST elevation or depression was considered significant only if it was >1mm.
Results: The study result showed maximum specificity for LCx (100%) followed by RCA (92.67) and LAD (89.91). The sensitivity for identifying the culprit artery by ECG in acute myocardial infarction was 100% for both LAD and RCA coronary artery but 0% for LCx coronary artery. In case of LAD occlusion, the sensitivity is 100% for proximal LAD occlusion and 92.86%for distal LAD occlusion. The sensitivity and specificity for proximal and distal RCA is 100% and 80.43% respectively.
Conclusions: ECG is an easily, widely available and non-invasive tool to localize the site of culprit artery in acute myocardial infarction.

Keywords: Electrocardiogram, Electrocardiographic algorithm, Myocardial infarction, Occlusion, ST deviation

INTRODUCTION

The electrocardiogram is the primary diagnostic tool to evaluate a patient with chest pain and suspected myocardial infarction in emergency department.[1] The importance of electrocardiographic changes and their relationship with the infarct related artery was recognized in the 1980s. Electrocardiography reflects the physiology of myocardium during acute ischemia whereas the coronary angiography identifies the vessel anatomy. The 12-lead electrocardiogram is an effective tool for the rapid diagnosis of acute myocardial infarction.[2] Anterior wall infarction due to occlusion of proximal LAD has worse prognosis compared to distal or branch vessel lesions.[3-5]

Patients are diagnosed as having anterior/inferior/ posterior or lateral MI based on patterns of ST deviation and risk assessment based on absolute magnitude of ST segment deviation or the width of QRS complexes.[6] Predicting the site of occlusion is important to assess the prognosis and infarct related complications.[7] ST

depression also further gives information on the infarct related artery and the site of occlusion (proximal or distal).[8,9] The ECG may help in identifying the size of the myocardial ischemic area at risk. It also determines sub endocardial and transmural ischemia and presence of previous ischemia.[10] Left anterior descending obstruction usually causes ST elevation in the precordial leads V1-V4.[11] Isolated ST elevation in leads V4-V6 without ST elevation in leads V1-V3 is usually due to occlusion of left circumflex artery or distal branch of LAD.[12]

The incidence of mortality and complications are high in patients of acute inferior wall myocardial infarction with right ventricular involvement. In acute inferior wall myocardial infarction, the infarct related artery is usually the right coronary artery (RCA) and less often left circumflex (LCX).[13] In the presence of complications like hypotension bradycardia, shock and arrhythmias RCA is generally the culprit artery.[14] Specific ECG patterns for the site of occlusion in culprit artery have been well recognized.[15]

Several studies have linked ECG changes to specific infarct related arteries and site of occlusion.[4-15] Zimat-baum PJ et al, has made algorithms based on these findings. Early prediction of infarct related artery can recognize the amount of myocardium at risk and provide decisions regarding urgency of revascularization and prevent complication. Although coronary angiography is the gold standard for determining the infarct related artery in acute myocardial infarction, ECG can be useful tool in identifying the culprit artery involved at the primary care. This study determines the ECG changes in acute myocardial infarction for localization of culprit artery occlusion and its diagnostic accuracy with follow up coronary angiogram.[2,3]

METHODS

This is a prospective cross-sectional study conducted in Aarupadai Veedu Medical College and Hospital, Pondicherry among 50 patients admitted in ICCU with acute myocardial infarction. The study population of 50 patients includes 35 male and 15 female patients. The study was conducted for a period of one year from July 2017 to June 2018. This study was started after obtaining clearance from the institutional human ethical committee of AVMC and H. Acute myocardial infarction was diagnosed by history of chest pain with elevated cardiac enzymes CK-MB twice more than the upper limit and characteristic ECG changes (New ST elevation ≥0.2mV in leads V1, V2 OR V3 and ≥ 0.1Mv in lead I,II,III, aVF, aVR).

Inclusion criteria

- Patients with chest pain <12 hours,
- Age between 20 and 75 years,
- ECG evidence of acute myocardial infarction.

Exclusion criteria

- History of previous myocardial infarction,
- Previous revascularization,
- Valvular heart disease,
- Patients on pace maker rhythm,
- ECG evidence of left bundle branch block.

Standard 12 lead electrocardiograph were recorded for patients who met the following inclusion criteria at a speed of 25mm/s and voltage of 10mm/mv. Patients who had acute inferior wall MI additional right pericardial leads were recorded (V3R and V4R). The recorded ECG was interpreted using the electrocardiographic algorithms of Zimet-baum PJ et al. An ST elevation or depression was considered significant only if it was >1mm.

Coronary angiogram was performed within 1 week for patients with electrocardiographic evidence of acute myocardial infarction. The patients were divided into three groups according to the site of occluded artery as (1). LAD, (2). RCA, (3) LCx. Group 1 was further subdivided into 1(a) and 1(b) according to occlusion in LAD is proximal or distal. Group 2 also subdivided into 2(a) and 2(b) depending upon whether RCA occlusion is proximal or distal.

Statistical analysis

The chi-square test was used to compare the electrocardiographically identified infarct related artery with the angiographically documented site of occlusion of the culprit artery.

RESULTS

The study population had 50 patients out of which 32 patients had anterior wall myocardial infarction and remaining 18 patients had inferior wall myocardial infarction according to Zumet-baum PJ et al, algorithym.

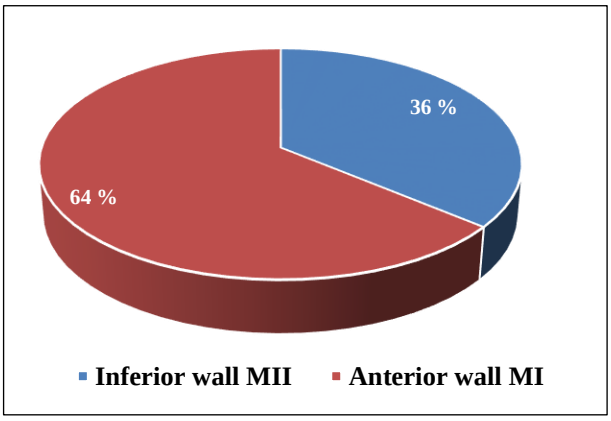

Figure 1: Distribution of myocardial infarction.

Figure 1 shows 32 patients (64%) had anterior wall myocardial infarction and 18 patients (36%) had inferior

wall myocardial infarction according to Zimet-Baum PJ et al, ECG criteria following patients were further subdivided according to the involved artery.

Table 1: Infarct related artery in inferior wall myocardial infarction.

Infarct related artery	Number of patients (N=18)	Percentage
Left circumflex	2	11.11
Proximal RCA	11	61.11
Distal RCA	5	27.78

Table 1 shows culprit artery in Inferior wall myocardial infarction.11 patients had involvement of proximal right coronary artery, 5 patients had lesion in the left circumflex artery and remaining 2 patients had lesion in the distal right coronary artery according to ECG criteria.

Table 2: Infarct related artery in anterior wall myocardial infarction.

Infaret related artery	Number of patients (N = 32)	(%)
Proximal LAD	24	75
Distal LAD	8	25

Table 2 shows infarct related artery in anterior wall myocardial infarction.24 patients (75%) had lesion in the proximal left anterior descending artery and remaining 8 patients (25%) had involvement of distal Left anterior descending artery. ECG criteria were correlated with follow-up coronary angiogram findings. 48 patients (96%) ECG criteria correlated fully with that of coronary angiogram findings and remaining 2 patients (4%) coronary angiogram site of occlusion was not fulfilled by ECG criteria. The site of culprit artery in undiagnosed by ECG criteria was LCx coronary artery.

Table 3: Correlation of electrocardiogram criteria with coronary angiogram.

Groups by ECG criteria	Sensitivity (%)	Specificity (%)
1 (LAD)	100	89.91
2 (RCA)	100	92.67
3 (LCx)	0	100
1(a) (Proximal LAD)	100	77.78
1(b) (Distal LAD)	92.86	71.43
2(a) (Proximal RCA)	100	90.67
2(b) (Distal RCA)	80.43	92.86

Table 3 shows correlation of ECG criteria for identifying culprit artery with follow up coronary angiogram. The study result showed maximum specificity for LCx (100%) followed by RCA (92.67) and LAD (89.91).The sensitivity for identifying the culprit artery by ECG in acute myocardial infarction was 100% for both LAD and RCA coronary artery but 0% for LCx coronary artery.In case of LAD occlusion the sensitivity is 100% for proximal LAD occlusion and 92.86% for distal LAD occlusion. The sensitivity and specificity for proximal and distal RCA is 100% and 80.43% respectively.

ECG criteria correlated 100 percent in 35 patients and partially in 13 patients. Coronary angiogram revealed occlusion in LCx for two patients which was not identified by ECG criteria. The sensitivity of ECG criteria to identify the culprit vessel is 100% for both LAD and RCA occlusion and 0% for LCx occlusion.

DISCUSSION

There are many studies that correlate electrocardiographic changes with that of infarct related artery.[1-15] In case of anterior wall myocardial infarction, the occlusion is in the LAD coronary artery. With inferior wall myocardial infarction, the occlusion is in either RCA or LCX coronary artery. According to Zimet-baum PJ et al, ECG criteria for LCX coronary artery is ST elevation in I, Avl, V5, V6and ST depression in V1, V2 and V3.[8]

Herz I et al, and Bairey CN et al, demonstrated that an ST depression in V1,V2 and V3 does not differentiate between a lesion in the RCA or the LCX.[16,17] Therefore authors modified the algorithm and stated that if the infarct related artery was not the RCA no ST elevations in III>II and no ST depressions in I and a VL it would be the LCX.

Kosuge M et al, studied the criteria of ratio of ST depression in V3/ST elevation in III in patients with acute IWMI and found the sensitivity, specificity, PPA and NPA for RCA occlusion proximal to RV branch to be 91%, 91%, 88% and 93% respectively.[18] In case of ST depression in lead I and a VL right coronary artery is the major culprit artery by electrocardiogram. Furthermore, ST increases in V3R signifies lesion in the proximal RCA. ST elevation of more than 1mm in lead V4R with upright T wave in that lead is the most sensitive ECG sign of RVMI.[19,20] This sign is rarely seen more than 12 hours after the infarction.

Occlusion of RCA causes a right ventricular infarction, which can lead to cardiogenic shock, atrioventricular block and is an independent predictor for bad outcome.[21,22] In case of anterior wall myocardial infarction ST elevation in V1>2.5MM OR st depression >1mm in II III AVF indicates lesion in the proximal LAD. ST elevation in V1 without ST depression in lead II II AVF indicates lesionn in the distal LAD.

LAD occlusion may lead to extensive anterior wall myocardial infarction or only apical, apical-anterior or mid-anterior MI depending on the site of occlusion.[23] Aldrich et al, showed the frequency of ST elevation in patients with acute myocardial infarction due to LAD occlusion to be in descending order;V2,V3,V4,V5,aVL,V1 and V6.[24]

Identification of lesion in the proximal LAD coronary artery is important because more aggressive treatment is warranted. These groups of patients have increased risk of complete heart block due to necrosis of the interventricular septum below the atrioventricular node. Several studies have established statistically convincing correlations between ST segment changes and infarct related arteries. However, the predictive values of the electrocardiographically determined infarct related artery will never become 100%, because of variations in coronary anatomy. Klarich KW et al, demonstrated that there is a great variability of myocardium at risk in an acute myocardial infarction for a given angiographically documented occluded coronary artery.[25]

CONCLUSION

This study determined the diagnostic value of electrocardiographic criteria for the prediction of infarct related artery. ECG is an easily, widely available and noninvasive tool to localize the site of culprit artery in acute myocardial infarction. Early identification of the culprit artery by ECG criteria helps to determine the best therapeutic option for a given patient with acute myocardial infarction and prevent further complications.

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REFERENCES

1. Myocardial infarction redefined-a consensus document of the joint European society of cardiology/American college of cardiology committee for the redefinition of myocardial infarction. Eur Heart J. 2000;21:1502-3.
2. Schweitzer P. The electrocardiographic diagnosis of acute myocardial infarction in the thrombolytic era. Am Heart J. 1990;119:642-54.
3. Iwasaki K, Kusachi S, Kita T, Taniguschi G. Prediction of isolated first diagonal branch occlusion by 12-lead electrocardiography: ST segment shift in leads I and avL. J Am Coll Carrfiol. 1994;23:1557-61.
4. Schuster EH, Grifith LS, Bulkley BH. Preponderance of acute proximal left anterior descending coronary arterial lesions in fatal myocardial infarction: A clinicopathologic study. Am J Cardiol. 1981;47:1189-96.
5. Klein LW, Weintraub WS, Agarwal JB, Schneider RM, Seelaus PA, Katz RI, Helfant RH: Prognostic sigdicance of severe narrowing of the proximal portion of the left anterior descending coronary artery. Am J Cardiol. 1986;5842-46.
6. Arnold AE, Simoons ML. Expected infarct size without thrombolysis', a concept that predicts immediate and long-term benefit from thrombolysis for evolving myocardial infarction. European Heart J. 1997;18(11):1736-48.
7. Karha J, Murphy SA, Kirtane AJ, de Lemos JA, Aroesty JM, Cannon CP, Antman EM, et al. Evaluation of the association of proximal coronary culprit artery lesion location with clinical outcomes in acute myocardial infarction. Am J Cardiol. 2003;92:913-8.
8. Zimet-baum PJ, Josephson EM. Use of the electrocardiogram in acute myocardial infarction. N Eng J Med. 2003;348:933-40.
9. Fiol M, Cygankiewicz I, Guindo J, Flotats A, Genis AB, Carreras F, et al. Evolving myocardial infarction with ST elevation: ups and downs of ST in different leads identifies the culprit artery and location of the occlusion. Annals Noninvasive Electrocardiol. 2004;9(2):180-6.
10. Moshkovitz Y, Sclarovsky S, Behar S, Reicher-Reiss H, Kaplinsky E, Goldbourt U, Sprint Study Group. Infarct site-related mortality in patients with recurrent myocardial infarction. Am J Med. 1993;94(4):388-94.
11. Blanke H, Cohen M, Schlueter GU, Karsch KR, Rentrop KP. Electrocardiographic and coronary arteriographic correlations during acute myocardial infarction. Am J Cardiol. 1984;54(3):249-55.
12. Aldrich HR, Hindman NB, Hinohara T, Jones MG, Boswick J, Lee KL, et al. Identification of the optimal electrocardiographic leads for detecting acute epicardial injury in acute myocardial infarction. Am J Cardiol. 1987;59(1):20-3.
13. Sapin PM, Musselman DR, Dehmer GJ, Cascio WE. Implications of inferior ST-segment elevation accompanying anterior wall acute myocardial infarction for the angiographic morphology of the left anterior descending coronary artery morphology and site of occlusion. Am J Cardiol. 1992;69(9):860-5.
14. Berger PB, Ryan TJ. Inferior myocardial infarction: high- risk subgroups. Circulat. 1990;81:401-11.
15. Gorgels AP, Engelen DJ, Wellens HJ. In: The electrocardiogram in acute myocardial infarction. Fuster V, Alexander RW, O'Rourke RA, eds. Hurst's the Heart. 11th ed. McGraw-Hill: New York. 2004:1351-60.
16. 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(10):1343-5.
17. Bairey CN, Shah PK, Lew AS, Hulse S. Electrocardiographic differentiation of occlusion of the left circumflex versus the right coronary artery as a cause of inferior acute myocardial infarction. Am J Cardiol. 1987;60(7):456-9.
18. Kosuge M, Kimura K, Ishigawa T, Hongo Y, Mochida Y, Sugiyama M, et al. New electrocardiographic criteria for predicting the site of coronary artery occlusion in inferior wall acute

myocardial infarction. Am J Cardiol. 1998;82:1318-22.
19. Braat SH, Brugada P, den Dulk K, van Ommen V, Wellens HJ. Value of lead V4R for recognition of the infarct coronary artery in acute inferior myocardial infarction. Am J Cardiol. 1984;53:1538-41.
20. Klein HO, Tordjman T, Ninio R, Sareli P, Oren V, Lang R, et al. The early recognition of right ventricular infarction: diagnostic accuracy of the electrocardiographic V4R lead. Circulat. 1983;67:558-65.
21. Berger PB, Ryan TJ. Inferior myocardial infarction: high risk subgroups. Circulat. 1990;81:401.
22. Wellens HJJ. The value of the right precordial leads of the electrocardiogram. N Engl J Med. 1999;340: 381.
23. Bayés de Luna A, Wagner G, Birnbaum Y, Nikus K, Fiol M, Gorgels A, et al. International society for holter and noninvasive electrocardiography. A new terminology for the left ventricular walls and for the locations of Qwave and Qwave equivalent myocardial infarction based on the standard of cardiac magnetic resonance imaging. A statement for healthcare professionals from a committee appointment by the international society for holter and Noninvasive electrocardiography. Circulat. 2006;114:1755-60.
24. Aldrich HR, Hindman NB, Hinohara T, Jones MG, Boswick J, Lee KL, et al. Identification of the optimal electrocardiographic leads for detecting acute epicardial injury in acute myocardial infarction. Am J Cardiol. 1987;59:20-3.
25. Klarich KW, Christian TF, Higano ST, Gibbons RJ. Variability of myocardium at risk for acute myocardial infarction. Am J Cardiol. 1999;83(8):1191-5.

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