Original Article

Acute ST-segment elevation myocardial infarction: The prognostic importance of lead augmented vector right and leads V_7–V_9

Veeresh Patil Hebbal, Huliyurdurga Srinivasasetty Natraj Setty, Cholenahalli Manjunath Sathvik, Vikram Patil, Sarthak Sahoo, Cholenahalli Nanjappa Manjunath

Department of Cardiology, Sri Jayadeva Institute of Cardiovascular Sciences and Research, Bengaluru, Karnataka, India

Address for correspondence:
Dr. H. S. Natraj Setty, #493, 4th Cross, 7th Main, J.P. Nagar 3rd Phase, Bengaluru - 560 069, Karnataka, India. E-mail: drnatrajsetty75@gmail.com

Abstract

Background: Acute myocardial infarction (MI) is associated with high mortality and among survivors have high morbidity. Electrocardiogram (ECG), a cost-effective and easily available, has traditionally been used not only just for diagnosis of MI but also for culprit vessel recognition and for prognostication. However, the role of lead augmented vector right (aVR) and leads V_7–V_9 in acute MI are often neglected in clinical practice. We studied the role of lead aVR and leads V_7–V_9 in ST-elevation MI (STEMI) patients. **Methods:** A total of 209 patients presenting with STEMI were enrolled in the study. History of comorbid conditions and habits was enquired. Routine blood tests were performed. Full spectrum ECG (including V7–9) and 2D-ECHO was performed on all patients. All the patients underwent revascularization by primary percutaneous coronary intervention. The role of lead aVR, lead V7, and leads V8–9 was analyzed in anterior wall MI (AWMI) and inferior wall MI. All the patients were followed up for 1 month for outcome assessment. **Results:** Of the 209 patients, 85.1% were males and 35.8% were diabetic, 60.2% were smokers, AWMI accounted for 55.5%. Lead aVR ST deviation was noted in 75.1% of patients (elevation in 17.7% and depression in 47.1%). V7 ST elevation occurred in 27.6% and V8–9 elevation occurred in 7.5% of the study population. Total death was 11.9% in the study (including the in-hospital mortality), all these patients had lead aVR ST segment deviation ($P < 0.001$). **Conclusion:** Lead aVR ST deviation and Lead V7 ST deviation helps to prognosticate the STEMI patients as high risk and those with aVR ST depression had higher mortality compared to aVR ST elevation because of larger myocardial involvement.

Key words: Lead augmented vector right, leads V7–9, outcome, primary percutaneous coronary intervention, ST deviation, ST-elevation myocardial infarction

INTRODUCTION

In the standard 12-lead electrocardiogram (ECG) some parts of the heart are under-represented. Present lead positions only represent the anterior surface and the inferior surface of heart but not the posterior and the apical segments. The vector of lead augmented vector right (aVR) is orientated approximately from the cardiac apex to the

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outflow tract of the right ventricle. Directionally, lead aVR is as much reciprocal to V_5 (facing the antero-apical region) as it is to V_7 (facing the inferior-apical region).[1] Transmural infarction in the basal interventricular septum may produce ST elevation in aVR when the injury current dipole has a component in the direction of the vector of lead aVR. This injury current dipole induces ST depression in left lateral chest leads V_5–V_7 and in leads I and II because these leads are reciprocal to aVR, in that their lead vectors have components in a direction opposite to that of aVR. Likewise, a transmural apical infarction can produce aVR ST depression. Since the lead V_7 records changes from the inferior cardiac apex, which is not available on the standard 12-lead ECG, ST segment changes in the often neglected lead aVR becomes particularly relevant.[2] The unique nonanterior and noninferior direction of lead aVR means that the prognostic implications of ST deviations (elevation or depression) could be different depending on the infarct location. In particular, aVR ST depression can be reflective of ST elevation in V_7 that the standard 12-lead ECG fails to capture.[3] The standard chest leads cover the anterior part of the chest only up to the midaxillary line (lead V_6), whereas V_7 lead placed at the posterior axillary line will also record ST elevation from a larger apical infarction due to an occlusion of a left anterior descending artery with a larger distribution to the inferior portion of the cardiac apex. Deeper aVR ST depression thus reflects higher STGJ elevation not only in leads V_5 and V_6 but also in lead V_7 and signifies a bigger apical infarction. However, V_7 is not a "standard" ECG lead and is not commonly recorded. The literature on its utility is also relatively nonuniform.[4] The major obstacle in the ECG diagnosis of posterior infarction lies in the absence of standard leads facing the posterior left ventricular wall.[5] The chest leads V_7–V_9 identifies those patients with concomitant posterior wall involvement,[6] as the ST depression in the precordial chest leads are neither specific nor sensitive for the diagnosis of posterior infarction.[7,8] However, these leads (V_7–V_9) are rarely used in clinical practice.

METHODS

Study design

A prospective study conducted in Sri Jayadeva Institute of Cardiovascular Sciences and Research, Bengaluru, India.

Study population

Includes patients both male and female aged 18 years and above presenting with ischemic chest pain with ECG diagnosis of ST-elevation acute myocardial infarction (MI).

Inclusion criteria

- Patients both male and female age of 18 years and above
- Chest pain with ST elevation MI <12 h duration
- New onset Left bundle branch Block
- On-going chest pain with ST elevation MI >12 h duration
- Patients undergoing primary percutaneous coronary intervention (PCI).

Exclusion criteria

- Non-ST-elevation MI
- History of cerebrovascular accident (hemorrhagic within 3 months)
- Patients thrombolyzed elsewhere.

Study method

Patients baseline characteristics involving age, sex, body mass index, diabetes (on treatment or fasting blood sugar (FBS) >120), hypertension (on treatment/two readings >140/90 mm of Hg), smoking (current/ex-smoker), alcohol are taken into consideration. The onset of symptoms to first medical contact, first medical contact to referral to tertiary center for further management, door to balloon time was noted. All the patients underwent primary PCI.

INVESTIGATIONS AND PROCEDURES

Blood investigations

Blood investigations including a complete hemogram, renal function test, FBS and fasting lipid profile (12 h overnight fasting), serology were performed.

Electrocardiogram

All the patients will undergo 12 lead ECG with additional leads of V_7, V_8, V_9 as per the protocol for lead placement. The amount of ST-segment change was measured to the nearest 0.5 mm at 60 ms after the J-point on all 12 leads including aVR and V_7, V_8, V_9 (0.5 mm of ST deviation representing a change of 50 μV). Any aVR ST depression/elevation of 0.5 mm or more and V_7, V_8, V_9 ST elevation of 0.5 mm was considered as significant.

Echocardiogram

To assess for LV function, regional wall motion abnormality, chamber enlargement, pericardial effusion and any other structural abnormality.

Coronary angiogram and angioplasty

All patients who gave consent for primary PCI underwent coronary angiography followed by percutaneous transluminal coronary angioplasty/stenting of the culprit vessel. The stent used was either DES or BMS (as per the consent). Revascularization was considered successful if TIMI III flow is established, resolution of symptoms and resolution of ST segment by >50%.

Premedications

All the patients received aspirin 325 mg bolus dose and a P2Y12 inhibitor either 600 mg of clopidogrel or 60 mg of prasugrel or 180 mg of ticagrelor and 80 mg of atorvastatin as per the ACC/AHA guidelines.[9] Heparin and GP2b3a inhibitor (eptifibatide) as per the cardiologist's discretion.

Medications postprocedure

All the patients received maintenance dose of aspirin, clopidogrel/prasugrel/ticagrelor, 12 h infusion of GP2b3a inhibitor. Other medications such as angiotensin-converting enzyme inhibitor, angiotensin receptor blocker's, diuretics, and beta-blockers were added if there were no contraindications.

Follow-up

All the patients were followed up for 30 days for outcome assessment.

Outcome/end point

Primary endpoint: All cause 30 days mortality. Secondary endpoint: Rehospitalization within 30 days either for heart failure, reinfarction, or other morbidity.

RESULTS

The baseline demographic profile of the patients studied is represented in Table 1. The mean age was 55 years with the range of 28–80 years. Of the total 209 patients, 85.1% of patients were male. About 66% of patients had chest pain <12 h, diabetic accounted for 35.8% and hypertensive patients were 41.6% of the total study population. Anterior wall MI (AWMI) accounted for 55.5% and 45.5% were inferior wall MI (IWMI). LAD was the culprit vessel in 49.7% of patients.

DISCUSSION

Acute MI is associated with high mortality and among the survivors have high morbidity. These events have been related to the time duration from symptom onset to recanalization of the occluded vessel. Higher the time elapsed, worse the outcome. Delay in revascularization may lead to greater myocardial loss and more significant ventricular dysfunction and recurrent hospitalizations for heart failure. Because of varying symptoms of acute MI ranging from classical symptoms of MI to atypical symptoms to silent MI, clinical recognition of the acute event is important and challenging. Misdiagnosis of the clinical symptoms or neglecting the subtle complaints without, proper investigation can be fatal to the patients. Myocardial survival depends on the timely recognition and effective management. One of the simplest easily available and cost effective investigations is an electrocardiogram. ECG has traditionally been used not just for the diagnosis for the acute MI but also for the recognition of the culprit vessel involved as well as, for the prognosis (e.g., presence of QRBBB pattern, the presence of complete heart block in AWMI, etc., are associated with higher mortality). However, the technical difficulty is the recognition of the ECG changes by the primary health care providers and timely initiation of treatment and early referral to tertiary care hospital for further management. There are certain pitfalls in the 12 lead ECG such as the role of lead aVR ST-segment deviation without ST elevation in precordial leads and role of ST depression in V1 and V2 leads with tall R wave and upright T wave (indicative of posterior wall MI). Lead V1 and V2 represents mirror image of the posterior leads (V_7–V_9) which do not represent in the regular 12 lead ECG and are infrequently ordered. This may result in failure to recognize an isolated posterior wall MI. Prior studies have evaluated the role of Lead aVR and ST deviation in patient presenting with acute coronary syndrome (ACS). There are also studies which have evaluated the role of V_7–V_9 leads ST deviation in patients presenting with ACS. In some of these studies, patient received only thrombolytic therapy and few received primary PCI. In this study, we combined both aVR lead and lead V_7–V_9 ST segment deviation in patient presenting with acute MI [Tables 2a-c]. The study population who underwent primary PCI were included

Table 1: Basic demographic data

Demographic data	Total 209
Age in years	
Mean (range)	55 (28-80)
Males (%)	178 (85.1)
Females (%)	31 (14.9)
Chest pain <12 h (%)	138 (66)
Chest pain >12 h (%)	71 (34)
Diabetic patients (%)	75 (35.8)
K/c/o hypertension (%)	87 (41.6)
BMI (%)	
Mild obesity*	81 (38.7)
Moderate obesity*	77 (36.8)
Severe obesity*	24 (11.4)
Smokers (%)	126 (60.2)
LVEF	
Mean (range)	48 (35-55)
Culprit vessel (%)	
LAD*	104 (49.7)
LCX*	23 (11)
RCA*	82 (39.3
AWMI (%)	116 (55.5)
IWMI (%)	93 (44.5)
Systolic BP (mmHg)	
Mean (range)	130 (60-220)
Diastolic BP (mmHg)	
Mean (range)	78 (30-110)
Symptom onset to referral to tertiary care (h)	
Mean (range)	13 (1-74)
Door to balloon (h)	
Mean (range)	0.5 (0.2-0.5)

BP: Blood pressure, BMI: Body mass index, LVEF: Left ventricular ejection fraction, LAD: Left anterior descending, LCX: Left circumflex artery, RCA: Right coronary artery, AWMI: Anterior wall myocardial infarction, IWMI: Inferior wall myocardial infarction

and evaluated. In this study, we found that lead aVR and V_7 correlate with each other more often. Lead V_8 and V_9 correlated more with the posterior wall involvement rather than lead V_7.We first evaluated the association of aVR ST deviation with ST-elevation MI (STEMI) [Table 3] and found that aVR ST deviation was associated with higher mortality [Table 4] compared with normal aVR in STEMI, which was statistically significant. We further evaluated aVR ST elevation versus aVR ST depression and found that aVR ST depression was associated with higher mortality than aVR ST elevation. We then assessed the importance of ECG leads V_7–V_9 in the study population mainly patients with IWMI. There was a statistically significant correlation of V_7–V_9 leads with STEMI. V_7 is more associated with AWMI [Tables 5 and 6], whereas V_8–V_9 showed more significance with IWMI [Tables 7 and 8]. Two patients who had V_8–V_9 ST elevation had mortality. Of total 209 patients recruited for the study, the overall mortality was 25 (11.96%). 17 (68%) were in-hospital mortality (IHM) and 8 (32%) occurred within 30 days (reinfarction-3, LVF-2, VT/VF-3). Among the 17 patients who had IHM [Table 9], it was seen that.

Table 2a: Of the total 209 patients, 17.7% patients had aVR ST elevation and 47.4% of patients had aVR ST depression while 34.9% had no aVR ST deviation. In AWMI group, 25.8% patients had ST elevation and 66.3% patients has ST depression in lead aVR

	aVR ST E	aVR ST D	aVR N	Total
AWMI (%)	30 (25.8)	77 (66.3)	9 (7.3)	116
IWMI (%)	7 (7.5)	22 (23.6)	64 (68.8)	93
Total (%)	37 (17.7)	99 (47.15)	73 (34.9)	209

AWMI: Anterior wall myocardial infarction, IWMI: Inferior wall myocardial infarction, aVR: Augmented vector right

Table 2b: V_7 elevation has seen in 26.7% of the study population. In AWMI group, 33.6% of patients had V_7 elevation

	V_7 E	V_7 N	Total
AWMI (%)	39 (33.6)	77 (66.4)	116
IWMI (%)	17 (18.2)	76 (81.8)	93
Total (%)	56 (26.7)	153 (73.3)	209

AWMI: Anterior wall myocardial infarction, IWMI: Inferior wall myocardial infarction, aVR: Augmented vector right

Table 2c: 9.5% of the study population had V_8-V_9 ST elevation. In IWMI group, 13.9% of patients had associated V_8-V_9 ST elevation

	V_8-V_9 E	V_8-V_9 N	Total
AWMI (%)	7 (6.0)	100 (94.0)	116
IWMI (%)	13 (13.9)	89 (86.1)	93
Total (%)	20 (9.5)	189 (90.5)	209

AWMI: Anterior wall myocardial infarction, IWMI: Inferior wall myocardial infarction, aVR: Augmented vector right

Table 3: Correlation of lead augmented vector right changes (elevation or depression) with myocardial infarction

	Anterior wall MI	Inferior wall MI	aVR change (EL/D)
Anterior wall MI			
Pearson correlation	1	−0.971**	0.636**
Significant (two-tailed)		<0.001	<0.001
n	209	209	209
Inferior wall MI			
Pearson correlation	−0.971**	1	−0.629**
Significant (two-tailed)	<0.001		<0.001
n	209	209	209
aVR change (EL/D)			
Pearson correlation	0.636**	−0.629**	1
Significant (two-tailed)	<0.001	<0.001	
n	209	209	209

**Correlation is significant at the 0.01 level (two-tailed). There is a correlation between the aVR changes and the occurrence of MI both in the anterior wall and inferior wall. This correlation is found to be statistically significant (P<0.001). MI: Myocardial infarction, EL/D: Elevation or depression, aVR: Augmented vector right

Table 4: Association between augmented vector right changes and the outcome

Status aVR changes	Alive (%)	Dead (%)	Total
Abnormal	111 (81.6)	25 (18.4)	136
Normal	73 (100.0)	0	73
Total	184 (87.1)	25 (11.9)	209

χ^2: 15.24, df: 1, P<0.01. The association is statistically significant (P<0.001). Total death was 11.9% in the study (includes IHM and 30 days mortality), all the patients who died had Lead aVR ST deviation. IHM: In-hospital mortality, aVR: Augmented vector right

Table 5: Relation between V_7 changes and anterior wall myocardial infarction

V_7 deviation	Anterior wall MI (%)		Total
	Yes	No	
Elevated	38 (70.4)	16 (29.6)	54 (100.0)
Normal	78 (50.3)	77 (49.7)	155 (100.0)
Total	116 (55.5)	93 (44.5)	209 (100.0)

χ^2: 6.517, df: 1, P<0.011. MI: Myocardial infarction

Table 6: Relation between V_7 changes and inferior wall myocardial infarction

V_7 deviation	Inferior wall MI (%)		Total
	No	Yes	
Elevated	38 (68.5)	16 (31.5)	54 (100.0)
Normal	78 (50.3)	77 (49.7)	155 (100.0)
Total	116 (55.5)	93 (44.5)	209 (100.0)

χ^2: 5.358, df: 1, P<0.021. MI: Myocardial infarction

Table 7: Relation between V_8 and V_9 changes and anterior wall myocardial infarction

V_8-V_9 deviation	Anterior wall MI (%)		Total
	Yes	No	
Elevated	7 (35.0)	13 (65.0)	20 (100.0)
Normal	109 (57.7)	80 (42.3)	189 (100.0)
Total	116 (55.5)	93 (44.5)	209 (100.0)

χ^2: 3.764, df : 1, P<0.052. MI: Myocardial infarction

Lead augmented vector right and in hospital mortality

- 6 (35.3%) had aVR ST elevation
- 11 (64.7%) had aVR ST depression.

Lead V_7 and coronary angiography

- 9 (52.94%) had V_7 elevation
- 8 (47.05%) had V_7 depression.

Lead V_8–V_9 and coronary angiography

- 2 (11.76%) had V_8–V_9 elevation
- 15 (88.23%) had V_8–V_9 depression.

Our study showed that patients with aVR ST depression had higher mortality 11 (64.7%) than patients with aVR STelevation6 (35.3%). This is also true with lead V_7 which is facing reciprocally to lead aVR.

Table 8: Relation between V_8 and V_9 changes and inferior wall myocardial infarction

V_8-V_9 deviation	Inferior wall MI (%)		Total (%)
	No	Yes	
Elevated	7 (35.0)	13 (65.0)	20 (100.0)
Normal	109 (57.1)	80 (42.9)	189 (100.0)
Total	116 (55.5)	93 (45.5)	209 (100.0)

χ^2: 3.583, df: 1, P<0.058. MI: Myocardial infarction

Table 9: Relation between lead changes and outcome

Outcome of patients with aVR ST Deviation			
Mortality	aVR ST E	aVR ST D	Total
IHM	6	11	17
Outcome of patients with V7 ST Deviation			
	V_7 E	V_7 N	
IHM	9	8	17
Outcome of patients with V_8-$_9$ ST deviation			
	V_8-V_9 E	V_8-V_9 N	
IHM	2	15	17

IHM: In-hospital mortality, aVR: Augmented vector right, E: Elevation, D: Depression, N: Neutral

Table 10: Comparison of our study with create registry

	Create	Our study
Mean age (years)	56.3	55
Diabetic patients (%)	30.4	35.8
Hypertensive patients (%)	37.7	41.6%
Smokers (%)	40.2	60.2
Thrombolytics (%)	58.5	0
Time from onset to hospital (h)	6	13
Overweight	<1/3rd	0
Body mass index >30 (%)	7	85
Severe obesity (%)	5.6	11.4
In-hospital mortality (%)	5.6	8.1
30 days outcome		
Death* (%)	8.6	11.9

Our study correlated with the study conducted by Wong *et al.*,[1] supports the above showing aVR ST depression was associated with higher 30 days mortality for anterior but not for inferior acute MI.

The create registry by Dewis Xvier *et al.*[10] which was a multicenter study carried out in Indian patients during 2001–2005. We correlated our study results with that of create registry [Table 10] and is shown below:

We found that in our study the IHM was higher compared to create registry. This may be due to various reasons such as higher diabetic patients, hypertensive patients, and higher number of smokers. Gross increases in obesity with 11.1% severe obesity and also added to these is the delay in arrival to the hospital. These may be attributed to the changing lifestyle and food habits of the population over the past one decade since the create registry to our study.

With acute AWMI, different mechanisms may explain the bad prognosis associated with aVR ST elevation (basal septal infraction from a culprit left coronary descending artery stenosis before the first septal artery potentially involving the left main artery) and that associated with aVR ST depression (large apical area involvement) when compared with patient with a neutral aVR ST level.

Limitations of the study

- Small, single-center study population
- Patients undergoing primary PCI only were evaluated
- Postprocedure ECG and 30 days ECG for the resolution of aVR ST elevations and its correlation with outcome was not evaluated
- Different/varying levels of ST elevation and its correlation/impact on mortality was not evaluated.

CONCLUSION

Acute MI is an emergency condition associated with high mortality and morbidity. Early identification and timely intervention can be lifesaving. Lead aVR ST deviation and also Lead V7 (reciprocal to lead aVR) helps to prognosticate the patients as high risk (aVR ST depression had higher risk compared to aVR ST elevation) due to the involvement of large area of myocardium. Posterior leads (V_7–V_9) should be taken in all ACS patients as to not miss the isolated posterior wall MI. Further larger studies evaluating the role of these posterior leads and its association with the IRA outcome is needed as in aVR lead with lead V_7.

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Conflicts of interest

There are no conflicts of interest.

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