

# A Study of Arrhythmias in First 48 Hours of Acute Myocardial Infarction in a Tertiary Care Hospital

MOHINI SINGH<sup>1</sup>, MONIKA GHAI<sup>2</sup>, SR RAMAKRISHNAN<sup>3</sup>

## ABSTRACT

**Introduction:** Myocardial infarction is defined as a clinical or pathologic event caused by myocardial ischemia in which there is evidence of myocardial injury or necrosis. The diagnosis of myocardial infarction is established by typical symptoms, cardiac biomarkers, electrocardiographic changes, or imaging evidence of new regional wall motion abnormality or new loss of viable myocardium. Arrhythmias are well-recognised complications of Acute Myocardial Infarction (AMI) and are important risk factors for mortality in both men and women, across a wide age range.

**Aim:** To analyse the incidence of arrhythmias in acute myocardial infarction with respect to the type, age, gender distribution, location and extension of AMI and to interpret effect of arrhythmias on mortality and morbidity in patients in the first 48 hours of AMI.

**Materials and Methods:** This observational study included 120 patients admitted within one hour of chest pain in Cardiac care unit at Sri Ramachandra Institute of Higher Education and Research Chennai, Tamil Nadu, India from February 2017 to April 2018 after fulfilling the inclusion criteria. Diagnosis of AMI with arrhythmia was made on clinical features, electrocardiography, cardiac biomarkers and echocardiogram. For statistical analysis of data, Statistical Package for Social Sciences (SPSS 16.0 version) was used.

**Results:** In this study of 120 patients, 74 (61.7%) were males and 46 (38.3%) were females (post-menopause) and highest

incidence of arrhythmia was noted in the age group of 61-70 years (32.5%). A total of 71 patients (59.2%) presented with chest pain ( $p < 0.001$ ) and 59.2% had type 2 diabetes. Anterior Wall Myocardial Infarction (AWMI) was observed in 29.16%, followed by Inferoposterior Wall Myocardial Infarction (IPWMI) which was observed in 21.6%. A total of 40.8% had arrhythmias within first hour of hospitalisation. In this study, ventricular premature complex was the most common arrhythmia, observed in 29.2%, followed by sinus tachycardia (20%). Tachyarrhythmias were more frequently observed in anterior infarction whereas bradyarrhythmias were more frequently observed in inferior infarction. 30.8% of patients had ejection fraction of  $< 40\%$ . Overall, mortality was 10% {5.8% in AWMI and 1.7% in IPWMI}. Cardiac biomarkers and left ventricular function were good predictors of extent of infarction ( $p < 0.001$ ). Ten deaths were due to arrhythmias and two secondarily to cardiogenic shock.

**Conclusion:** In this study, it was observed that incidence of myocardial infarction increases with age and was noted more in males than females and in females it was noted more in post-menopausal group. Most common type of arrhythmias observed were ventricular premature complexes and sinus tachycardia and significant number of patients had arrhythmias during the first hour of hospitalisation. Overall, mortality rate was 10% and cardiac biomarkers and left ventricular function are good predictors of infarct size.

**Keywords:** Acute coronary syndrome, Bradyarrhythmias, Cardiac biomarkers, Tachyarrhythmia

## INTRODUCTION

Acute Myocardial Infarction (AMI) is a clinical event in the setting of myocardial ischemia with the evidence of myocardial injury [1,2]. Cardiovascular Disease (CVD) in India has become the leading cause of mortality [3] and in all parts of India (including the rural areas and poorer states) [4]. CVD affects Indians a decade earlier than the western population [5-7]. It is predicted that deaths due to CVD in India by 2020 will increase by 111% compared to the year 1990 and this is higher than mortality rate predicted in any other region of the world [8]. In AMI, cardiac arrhythmias are well-recognised, frequent complications and important predictors of mortality, which can be due to an imbalance of autonomic nervous system and electrolytes and also due to ischemia which causes conduction blockade in the infarcted zone. Conduction abnormalities especially high grade atrio-ventricular block is a common complication of ST-Elevation Myocardial Infarction (STEMI) and although in the era of primary percutaneous coronary intervention, the rates of post-myocardial infarction the incidence of conduction abnormalities is low and decreasing, but it continues to be associated with high risk for in hospital deaths [9-11]. Ventricular arrhythmias are common within the first 48 hours of infarction and continue to have a negative impact on the patient's outcome [12-14]. The magnitude of risk of arrhythmias in AMI varies

from patient to patient with infarct size and left ventricular function being the most important risk stratifier. With limited data available from southern India, this study was undertaken to evaluate cardiac arrhythmias in the first 48 hours of AMI with respect to the incidence, location and extent of myocardial injury.

## MATERIALS AND METHODS

The present study was a prospective observational study conducted from February 2017 to April 2018. This study included 120 patients who were admitted within one hour of chest pain in Cardiac care unit after fulfilling the inclusion criteria. Patient's written consent was obtained and design of the work was approved by the institutional ethics committee (REF:CSP-MED/16/JAN/27/15).

**Inclusion Criteria:** All patients with ST-Elevation myocardial infarction who developed arrhythmia within first 48 hours of AMI. Patients undergoing percutaneous intervention (angioplasty and pacemaker insertion) as part of the treatment at our hospital and patients above the age of 19 years.

**Exclusion Criteria:** Patients without myocardial infarction but having arrhythmias. Patients with previous history of myocardial infarction, previous cardiac surgeries, valvular heart diseases, and those who received treatment outside our institution.

AMI is diagnosed according to fourth universal definition [15] as acute myocardial injury with clinical evidence of acute myocardial ischemia and with detection of a rise and/or fall of cardiac troponin (cTn) values with at least one above the 99<sup>th</sup> percentile of the Upper Reference Limit (URL) and at least one of the following:

- Symptoms of myocardial ischemia
- New ischemic electrocardiographic changes
- Development of pathological Q waves
- Imaging evidence of new loss of viable myocardium or new regional wall motion abnormality in a pattern consistent with an ischemic aetiology
- Identification of an intracoronary thrombus by angiography or autopsy

Diagnosis of AMI was made according to fourth universal definition [15]:

- Symptoms of myocardial ischemia- Chest pain: Classic ischemic chest pain is chest tightness or pressure, in the substernal area, with radiation to the left arm or jaw, also noted was the duration, character, similarity to possible previous episodes and provoking factors. Associated symptoms include shortness of breath, diaphoresis, weakness, and anxiety.
- Twelve-lead Electrocardiogram (ECG).
- ECG manifestations of acute myocardial ischemia (in the absence of left bundle branch block).
- ST-elevation- New ST-segment elevation at the J-point in two contiguous leads with the cut-points:  $\geq 0.1$  mV in all leads other than leads V2-V3; For leads V2-V3:  $\geq 2$  mm in men  $\geq 40$  years;  $\geq 2.5$  mm in men  $< 40$  years, or  $\geq 1.5$  mm in women regardless of age.
- Electrocardiographic manifestations of ischemia in the setting of left bundle branch block:
  - » ST segment elevation  $\geq 1$  mm and concordant with the QRS complex (points -5).
  - » ST segment depression  $\geq 1$  mm in lead V1, V2 or V3 (points 3).
  - » ST segment elevation  $\geq 5$  mm and discordant with the QRS complex (points 2).
  - » A score of  $\geq 3$  had a specificity of 98% for AMI.
  - » Types of arrhythmias were also noted in the ECG.
- Cardiac Biomarkers:
  - » Creatine PK-MB-values above 24 U/L was considered positive.
  - » Troponin T by card test assay was done in all patients and values above 30 ng/L was considered positive.
  - » The cardiac biomarkers were done at the time of admission, 6 hours, 24 hours after and 48 hours after admission.
- A 2-D echocardiography with Doppler flow study (ECHO): Echocardiogram was done in all patients to look for regional wall motion abnormalities and to rule out aortic dissection.
- Detailed physical examination was done for all the patients with special attention given to pulse, the measurement of blood pressure, auscultation of the heart and lungs, and assessment for heart failure or circulatory compromise. History also included risk factor analysis for myocardial ischemia.
- The diagnosis of arrhythmia was carried out as per AHA guidelines and treated accordingly.

### STATISTICAL ANALYSIS

The statistical analysis and interpretations were undertaken as follows. The continuous variables were analysed in terms of averages such as mean value and they have been interpreted by independent student t-test. The categorical variables were analysed

in terms of percentages and interpreted by  $\chi^2$  (Chi-square) test. The p-values  $\leq 0.05$  were considered significant. SPSS 16 was used to analyse the data.

### RESULTS

Total number of 120 patients were included in this study, of which 74 (61.7%) were male and 46 (38.3%) were female patients.

The age group varied from 21 to 90 years and highest incidence of arrhythmia was noted in the age group of 61-70 years (32.5%). Least number of arrhythmias was noted in the age group 21-30 years and 31-40 years (2.5% each) [Table/Fig-1]. Mean age of male was  $59.6 \pm 14.1$  and females was  $62.4 \pm 10.5$  ( $t=1.273$  and  $p>0.05$ ).

It was observed in this study that chest pain was the commonest

Age group	Gender		Total
	Female	Male	
21-30 y	0	3	3 (2.5%)
31-40 y	0	3	3 (2.5%)
41-50 y	7	11	18 (15%)
51-60 y	10	19	29 (24.2%)
61-70 y	19	20	39 (32.5%)
71-80 y	8	14	22 (18.3%)
81-90 y	2	4	6 (5%)
Total	46	74	120 (100%)

[Table/Fig-1]: Age wise gender distribution.

symptom noted in 71 patients (59.2%) and 20 patients (16.7%) presented with other symptoms such as epigastric pain, palpitations, syncope or a combination of symptoms [Table/Fig-2].

Clinical features	Number of patients	Chi square test
Chest pain	71 (59.2%)	$\chi^2=37.050$ $p<0.001$
Dyspnea	29 (24.2%)	
Other symptoms	20 (16.7%)	
Total	120 (100%)	

[Table/Fig-2]: Distribution based on clinical features.

Risk factors noted in this study have been summarised in [Table/Fig-3] with Type 2 diabetes noted as the commonest risk factor present in 59.2% subjects.

Risk factors	Percentage
Type 2 DM	59.2%
Hypertension	53.3%
Family history of Coronary Artery Disease (CAD)	20.8%
Chronic Kidney Disease (CKD)	11.7%
Cerebrovascular Accident (CVA)	5.8%
Chronic Obstructive Pulmonary Disease (COPD)	5%

[Table/Fig-3]: Distribution based on risk factors in 120 cases.

It was noted that both cardiac biomarkers (CK-MB and troponin T) showed serial rise with peak rise occurring by 12 hours in 82 (68.3%) patients for CK-MB and in 113 (94.1%) patients for troponin T.

AWMI was observed in 35 (29.16%) patients, followed by IPWMI which was observed in 26 (21.6%) patients. Inferior Wall with Right Ventricular Wall Myocardial Infarction (IW+RVMI) was observed in 15 (12.5%) patients, anterolateral wall (ALWMI) and Inferior Wall Myocardial Infarction (IWMI) was noted in 14 (11.6%) patients each, Anteroseptal Myocardial Infarction (ASWMI) was observed in 10 (8.3%) patients and Lateral Wall Myocardial Infarction (LWMI) was noted in 6 (5%) patients. Anterior infarction (including lateral wall)

was observed in 54.1% and inferior infarction (including posterior and right ventricle) was noted in 45.8%.

In this study it was observed that 49 (40.8%) patients had arrhythmias within first hour of hospitalisation, 39 (32.5%) patients had arrhythmias within 24 hours of hospitalisation and 32 (26.6%) patients demonstrated arrhythmias by 48 hours of hospitalisation with Ventricular Premature Complex (VPC) as the most common arrhythmia, observed in 35 (29.2%), and idioventricular rhythm, reperfusion arrhythmia and Supraventricular Tachycardia (SVT) observed only in 1 (0.8%) patient each.

[Table/Fig-4] shows distribution of various types of arrhythmias according to the location of infarction in 120 cases.

Out of 120 patients, 62.5% of patients had left ventricular Ejection Fraction (EF) >40% and 37.5% of patients had ejection fraction of <40% [Table/Fig-5].

Mortality according to location of the infarct was higher with anterior infarction (5.8% AWM) than inferior infarction (1.7% IPWMI and IWMI each) [Table/Fig-6].

Type of arrhythmias and number of patients	Location of the infarction and number of patients							Total	Chi-square test
	AWMI	ASWMI	ALWMI	IWMI	IPWMI	IW+RVMI	LWMI		
Ventricular Premature Complexes (VPC)	12	4	6	2	4	6	1	35	$\chi^2=5.074$ df=3 p>0.05
Sinus Tachycardia (ST)	9	3	4	2	4	1	1	24	
Ventricular Tachycardia (VT)	3				3	2	1	9	
Atrial Fibrillation (AF)	4	2	1		1			8	
Supraventricular Tachycardia (SVT)	1							1	
1 & 2 Atrioventricular Block (AV)				2	3			5	
Complete Heart Block (CHB)			1	2	3	2		8	
Left Bundle Branch Block (LBBB)	6							6	
Right Bundle Branch Block (RBBB)		1	1	2	3	2	1	10	
Sinus Bradycardia (SB)				3	5	2	2	12	
Idioventricular rhythm			1					1	
Reperfusion arrhythmia				1				1	
Total	35	10	14	14	26	15	6	120	

[Table/Fig-4]: Distribution of arrhythmias according to the location of the infarction.

AWMI: Anterior wall myocardial infarction; ASWMI: Anteroseptal wall myocardial infarction; ALWMI: Anterolateral wall myocardial infarction; IWMI: Inferior wall myocardial infarction; IPWMI: Inferior posterior wall myocardial infarction; IW+RVMI: Inferior wall and right ventricular myocardial infarction; LWMI: Lateral wall myocardial infarction

Correlation between the mortality, type of arrhythmia and ejection fraction are depicted in [Table/Fig-7].

Out of 120 patients included in this study, 108 (90%) recovered and 12 (10%) died. Out of 12 patients who died, 10 died of cardiac arrhythmias and two died of cardiogenic shock and out of these 10 patients who died secondarily to arrhythmias, four patient had

Type of arrhythmias	Ejection fraction		Total	Chi-square
	<40%	>40%		
Sinus tachycardia	6 (25%)	18 (75%)	24 (20%)	$\chi^2=13.048$ df=10 p>0.05
Sinus bradycardia	1 (8.3%)	11 (91.6%)	12 (10%)	
Atrial fibrillation	1 (12.5%)	7 (87.5%)	8 (6.7%)	
Ventricular tachycardia	5 (55.5%)	4 (44.4%)	9 (7.5%)	
Ventricular premature complexes	12 (34.28%)	23 (65.7%)	35 (29.2%)	
Complete heart block	4 (50%)	4 (50%)	8 (6.6%)	
1 <sup>st</sup> and 2 <sup>nd</sup> degree Atrioventricular block	3 (60%)	2 (40%)	5 (4.1%)	
Left bundle branch block and Right bundle branch block	11 (68.7%)	5 (31.25%)	16 (13.3%)	
Reperfusion arrhythmias	1 (100%)	0	1 (0.8%)	
Supraventricular tachycardia	0	1 (100%)	1 (0.8%)	
Idioventricular rhythm	1 (100%)	0	1 (0.8%)	
Total	45 (37.5%)	75 (62.5%)	120 (100%)	

[Table/Fig-5]: Distribution of arrhythmias in relation to Ejection fraction.

Ventricular Tachycardia (three patients from AWM) and one patient from IPWMI), four patients had complete heart block (two patients from IWMI, one patient each from IPWMI and ALWMI), one patient had persistent sinus tachycardia (AWMI) in the first one hour of Myocardial Infarction, which after six hours degenerated into Ventricular Fibrillation and could not be reverted. One patient had haemodynamic unstable Atrial Fibrillation (AWMI). All the patients who died had ejection fraction <40%.

To know the extent of the infarction, the parameters which were looked into were ST segment elevation and appearance of Q waves on ECG, peak levels of cardiac biomarkers, Regional Wall Motion Abnormality (RWMA) and EF on 2D ECHO. The results are depicted in [Table/Fig-8].

In this study, all 120 patients received antiplatelets, nitrates, ACE inhibitors or Angiotensin Receptor Blockers (ARBs), beta adrenergic blockers. A total of 95 (79.2%) received thrombolytic therapy in the form of Unfractionated Heparin (UFH), Low Molecular Weight Heparin (LMWH) or fibrinolytic agents like Streptokinase and only

Location of infarct	Survived		Mortality		Total		Significance
	No	%	No	%	No	%	
Anterior Wall Myocardial Infarction (AWMI)	28	23.3	7	5.8	35	29.2	$\chi^2=3.299$ df=1 p>0.05
Anteroseptal Wall Myocardial Infarction (ASWMI)	10	8.3	0	0.0	10	8.3	
Anterolateral Wall Myocardial Infarction (ALWMI)	13	10.8	1	0.8	14	11.7	
Inferior Wall Myocardial Infarction (IWMI)	12	10.0	2	1.7	14	11.7	
Inferior Posterior Wall Myocardial Infarction (IPWMI)	24	20.0	2	1.7	26	21.7	
Inferior Wall And Right Ventricular Myocardial Infarction (IW+RVMI)	15	12.5	0	0.0	15	12.5	
Lateral Wall Myocardial Infarction (LWMI)	6	5.0	0	0.0	6	5.0	
Total	108	90.0	12	10.0	120	100.0	

[Table/Fig-6]: Mortality according to location of the infarct.

Type of arrhythmias	Total no.	EF <40%	Mortality	Chi-square test
Ventricular tachycardia	9	5	4	$\chi^2=1.640$ p>0.05
Sinus tachycardia	24	6	1	
Complete heart block	8	4	4	
Atrial fibrillation	8	1	1	

[Table/Fig-7]: Correlation between mortality, EF and type of arrhythmias.

Criteria	Category	Components	Survived		Died		Total		Significance
			No	%	No	%	No	%	
ECG	ST (MM) segment elevation	2-5	79	65.8	8	6.7	87	72.5	$\chi^2=0.019$ $p>0.05$
		>5	29	24.2	4	3.3	33	27.5	
		Total	108	90.0	12	10.0	120	100.0	
	New Q waves	Yes	42	35.0	7	5.8	49	40.8	$\chi^2=1.690$ $p>0.05$
		No	66	55.0	5	4.2	71	59.2	
		Total	108	90.0	12	10.0	120	100.0	
Cardiac biomarkers	CPK-MB >100 U/L	Yes	39	32.5	12	10.0	51	42.5	$\chi^2=20.748$ $p<0.001$
		No	69	57.5	0	0.0	69	57.5	
		Total	108	90.0	12	10.0	120	100.0	
	Troponin T >500 ng/L	Yes	37	30.8	12	10.0	49	40.8	$\chi^2=22.137$ $p<0.001$
		No	71	59.2	0	0.0	71	59.2	
		Total	108	90.0	12	10.0	120	100.0	
2D ECHO	Global hypokinesia	Yes	26	21.7	12	10.0	38	31.7	$\chi^2=32.388$ $p<0.001$
		No	82	68.3	0	0.0	82	68.3	
		Total	108	90.0	12	10.0	120	100.0	
	EF <40%	Yes	33	27.5	12	10.0	45	37.5	$\chi^2=30.341$ $p<0.001$
		No	75	62.5	0	0.0	75	62.5	
		Total	108	90.0	12	10.0	120	100.0	

[Table/Fig-8]: Extension of infarction (n=120).

25 (20.8%) cases underwent primary Percutaneous Intervention (PCI) due to consent related issue. It was observed that the mode of termination of arrhythmias was pharmacological in 52 (43.3%) cases, by DC shock in 14 (11.6%) cases, by temporary electrical pacing in 4 (3.3%) cases and spontaneous recovery was observed in 39 (32.5%) cases. Eleven (9.1%) cases had persistent arrhythmias.

## DISCUSSION

### Demographics of Patients with Myocardial Infarction and Arrhythmias

In this study of 120 patients, 61.7% were males and 38.3% were females. Highest incidence of AMI was observed in age group of 61-70 years both for males and females followed by age group of 51-60 years. Meta-analysis of various studies by National Cholesterol Education Programme [16] (Bethesda, MD, National Heart, Lung, Blood Institute, NIH, 2001) have clearly indicated that there is higher incidence of myocardial infarction and arrhythmias in males compared to pre-menopausal women. However, after menopause the coronary risk and risk of arrhythmias equals to that of men. In a study by Kumar V et al., out of 50 cases enrolled in study 38 were males and 12 were females and 66.7% females were between age group 50-59 years and 16.6% females were between the age group 60-69 years, and only 8.3% were females were below 50 years [17]. In a study by Patil PR et al., it was observed that maximum number of patients were in the age group 50-59 years for both male (37.9%) and females (65.22%) and females were above 40 years of age [18]. This is clearly demonstrated by present study as well. In the age group 21 to 30 years and 31 to 40 years, there were three patients in each group and all were males. On the other hand, in the age group 61 to 70 years there were equal number of males and females.

### Risk Factor Analysis

In this study 59.2% of patients were diabetic and 53.3% of patients had hypertension. A 20.8% had family history of coronary artery disease. In a study by Stone PH et al., where 85 diabetic patients with AMI were compared to 415 non-diabetic patients, it was observed that diabetic patients had complicated course due to heart failure and complications like arrhythmias [19]. In a study by the National Registry of Myocardial Infarction that enrolled more than 540,000 patients between 1994 and 2006 who presented

with a first myocardial infarction with no previous history of CVD, 86 percent had one of five major risk factors (hypertension, smoking, dyslipidemia, diabetes mellitus, or family history of CAD) [20]. In a study by Patil PR et al., 52.9% were smokers, 38.2% and 36.30% were hypertensives and diabetic respectively [18]. In a study by Marangmei L et al., 22% were diabetic and 26% were hypertensives [21].

### Time of Onset of Arrhythmia

The present study shows that highest incidence of arrhythmias occurred during the first hour of hospitalisation {49 (40.8%) patients} and also with the first 24 hours {39 (32.5%) patients} of myocardial infarction. In the study by Aufderheide TP it was observed that around 90% of patients with AMI had some cardiac rhythm disturbances and approximately 25% within 24 hours following infarct onset had cardiac conduction disturbance [22]. In a study by Kumar V et al., 70% of arrhythmias occurred during the first hour of presentation [17].

### Locations of Infarction and Arrhythmias

In this study it was observed that 65 (54.1%) of patients had anterior infarction (including lateral wall) and 55 (45.8%) patients had inferior infarction (including posterior and right ventricle) and was observed that amongst tachyarrhythmias the commonest arrhythmias noted in this study was VPCs and ST observed in 23 patients and 17 patients respectively in anterior infarction (including lateral wall) and in inferior infarction (including posterior and right ventricular) only 12 patients and 7 patients had VPCs and ST. Amongst bradyarrhythmias, sinus bradycardia was observed in two patients with anterior infarction and in 10 patients with inferior infarction. Complete Heart Block (CHB) was observed in seven patients with inferior infarction and one patient with anterior infarction. According to a study conducted by Hreybe H et al., [23] Cardiology Department of the Medical College of Georgia, Augusta, Georgia, USA- "Location of AMI and associated arrhythmias" patients with inferior wall myocardial infarction were more likely to develop bradyarrhythmias in contrast to patients with anterior wall myocardial infarction who showed a tendency to develop tachyarrhythmias. In inferior wall myocardial infarction- Conduction disturbance may occur either at presentation or after hours or days. Sinus bradycardia, Mobitz type I (Wenckebach), and complete heart block are commonly seen, since the SinoAtrial node, AtrioVentricular node, and bundle of HIS are primarily supplied by the right coronary artery [24]. In the sub study

from the TRACE trial, CHB incidence was significantly higher among patients with an inferior wall myocardial infarction than among those with anterior wall myocardial infarction (9.4 versus 2.5%) [25]. In a study by Marangmei L et al., VPCs and Sinus Tachycardia were the commonest arrhythmias noted in 23% and 21%, respectively and in IWMI sinus bradycardia along with heart block were the commonest arrhythmias observed in 28.95% against 6.55% in AWWMI. AWWMI had sinus tachycardia observed in 31% compared to 7.8% in IWMI. [21]. In a study by Patil PR et al., it was observed that VPCs and ST was noted in 13.75% and 10%, respectively in AWWMI and Sinus Bradycardia in 17.5% and AV blocks in 15% in IWMI [18].

### Mortality in Relation to Location, Extent of Infarction and Arrhythmias

**Location of infarction:** Anterior infarction compared to inferior infarction is associated with poor outcome, mainly due to large infarct size [26-30]. In a study by Stone PH et al., it was observed that anterior infarction had evidence of large infarct size than inferior infarction (21.2 versus 14.9 Eq/m<sup>2</sup> CK -MB fraction  $p < 0.001$ ) and was accompanied by high incidence of heart failure (40.7 versus 14.7  $p < 0.001$ ) and in hospital death (11.9 versus 2.8  $p < 0.001$ ) [26]. In this study, it was observed that mortality was higher with anterior infarction (AWMI 5.8% and ALWMI 0.8%) than inferior infarction (IPWMI and IWMI 1.7% each)

**Extent of infarction:** Infarct size in patients with STEMI is an important determinant of the prognosis. In this study it was observed that out of 12 patients who died, seven patients developed new Q waves on ECG. Large infarct and high mortality rates can be predicted by the appearance of new Q waves in the setting of STEMI. In a study by Huey BL et al., 150 patients with STEMI were included in the study, 115 subjects developed Q waves and this group exhibited high ST segment elevation and peak creatine kinase level and low left ventricular ejection fraction (reflective of large infarction) [31].

In a study by Mauri F et al., it was observed that ST segment elevation on admission ECG correlated with the extent of myocardial injury and survival rate [32]. In a study by Hathaway WR et al., concluded that the sum of ST segment deviation, QRS duration and ECG evidence of prior infarction for new inferior infarction were strong predictors of extent of myocardial injury and mortality [33]. Present study included 120 patients with STEMI of which 87 had ST elevation of 2-5 mm. Thirty-three had ST elevation  $> 5$  mm and out of which four patients died.

Cardiac biomarkers are released into the circulation in response to myocardial injury and thus correlate with the infarct size. In this study, it was observed that all the 12 patients who died had high levels of CPK-MB and troponin T ( $p < 0.001$ ). Giannitsis E et al., in his study noted that except for admission values, all single point troponin T levels on any of the first four days correlated well with infarct size and perform as well as the peak troponin T levels and were significantly higher in STEMI ( $p < 0.01$ ) [34]. In a study by Arruda-Olson AM et al., included 121 subjects with AMI and concluded that the independent predictor of measurable infarct size on SPECT-MPI included troponin T on day 1, 2 and 3 and peak value but not at presentation or values less than 12 hours [35]. In a study by Mayr A et al., it was observed that there was significant correlation between the acute and midterm infarct size ( $p < 0.001$ ) and single point and peak levels of Creatine Kinase (CK) and Cardiac troponin T (CtnT) and cardiac biomarkers also showed significant correlation with the left ventricular ejection fraction ( $p < 0.002$ ) [36]. Thus in AMI, cardiac biomarkers within the first four days are good predictors for accurate infarct size and LV function.

Location and extent of infarct can be identified by echocardiography (regional wall motion abnormality and left ventricular ejection fraction) and it correlates with the outcome of the patients with AMI [37]. Assessment of infarct size by ECHO has been recommended by 2003 ACC/AHA/ASE task force [38]. As, studies have shown

that assessment of infarct size only by electrocardiogram can be underestimated in 95% of cases, especially in posterior, apical and right ventricular wall infarction [39-45]. This was in concordance with present study as all the 12 patients who died had global hypokinesia and EF  $< 40\%$  ( $p < 0.001$ ).

**Arrhythmias:** Out of 120 patients included in this study, 108 (90%) recovered with the treatment and 12 (10%) died. Out of 12 patients who died, 10 died of cardiac arrhythmias and two died of cardiogenic shock. All the patients who died had ejection fraction  $< 40\%$ .

Out of four patients with ventricular tachycardia who died three had AWWMI and one patient had IPWMI. It was observed from GUSTO I trial of 40,895 patients with acute STEMI, 10.2% had VT or VF and higher incidence of 30 day and in hospital mortality rate and 80 to 85% of these arrhythmias occurred during the first 48 hours [46]. In a study by Al-Khatib SM et al., (GUSTO-III trial), it was observed that the 30 day mortality rate was 44% with Ventricular Tachycardia/Ventricular Fibrillation [47]. In a study by Stone PH et al., where it was observed that patients with anterior infarction had poor outcome due to presence of large infarct size, serious ventricular arrhythmias and higher rate of heart failure [26].

In this study, it was noted that eight patients had CHB and out of which four patients died and of which two patients had IWMI, one patient had IPWMI and only one patient had ALWMI. In anterior infarction conduction disturbance are less frequently observed but serious and this may be related to the infarction size. Whereas conduction disturbance in inferior infarction are commonly observed, as the primary arterial supply of SA node, bundle of HIS and AV node is Right coronary artery. High degree AtrioVentricular block-Advanced (second or third degree) AtrioVentricular block is associated with an increase in mortality in patients with an inferior or anterior wall myocardial infarction [10,11,25,48-52].

In this study, one mortality with sinus tachycardia with AWWMI was observed and which degenerated into ventricular fibrillation. Persistent sinus tachycardia is usually associated with large infarct (anterior), with high 30 day mortality and marked impairment of LV function [53]. According to Braunwald textbook of cardiovascular medicine, the presence of persistent sinus tachycardia is an indirect evidence of significant left ventricular dysfunction and predicts higher mortality rates; either patient develops dangerous ventricular arrhythmias or heart failure [54].

Atrial fibrillation in AMI has prognostic significance. In this study, it was observed that one subject with anterior wall myocardial infarction with atrial fibrillation had died during this study. According to Braunwalds textbook of cardiovascular medicine, atrial fibrillation in myocardial infarction is associated with increased mortality particularly in patients with anterior wall infarction [55]. In patients with a recent myocardial infarction, the development of atrial fibrillation increases mortality [56,57]. This effect is primarily due to associated risk factors, such as heart failure and cardiogenic shock, not atrial fibrillation itself [57,58]. It is widely accepted that the likelihood of death after myocardial infarction correlates inversely with left ventricular performance. The degree of left ventricular dysfunction is determined by the extent of myocardial necrosis or infarct size. Thus, pump failure and increased mortality result when the infarct is large and ventricular compromise is severe [59,60]. In this study, it was observed that 12 patients who died of which two patients had cardiogenic shock and 10 patients had arrhythmias, all these patients had Ejection Fraction  $< 40\%$  and global hypokinesia ( $p < 0.001$ ), thus it is observed that mortality is influenced by the type of arrhythmias, LV function, infarct location and size of infarction.

### Limitation(s)

The limitation of the study was the small study sample and the limited use of reperfusion therapy due to consent related issues which can influence the outcome.

## CONCLUSION(S)

In this study it was observed that incidence of myocardial infarction increases with age, in diabetic and hypertensive's and was noted more in males than females (postmenopausal). Significant number of patients had arrhythmias in the first hour of hospitalisation. VPCs and ST were the commonest arrhythmias noted. Tachyarrhythmias were more common in anterior infarction and bradyarrhythmias were more common in inferior infarction. Mortality rate was 10% and correlated with LV function, infarct size and infarct location. Arrhythmias are well-recognised complications of AMI, hence early diagnosis and prompt treatment may improve the outcome. More studies designed for short and long term outcomes of specific management strategies specially well designed intervention trials and refinement of the existing tools is needed for more efficient prevention of premature deaths from arrhythmias.

## REFERENCES

- Anderson JL, Morrow DA. Acute myocardial infarction. *N Engl J Med*. 2017;376:2053.
- Reddy K, Khaliq A, Henning RJ. Recent advances in the diagnosis and treatment of acute myocardial infarction. *World J Cardiol*. 2015;7(5):243-76. doi:10.4330/wjc.v7.i5.243.
- Srinath Reddy K, Shah B, Varghese C, Ramadoss A. Responding to the threat of chronic diseases in India. *Lancet*. 2005;366:1744-49.
- Report on Causes of Death in India 2001-2003. New Delhi, India: Office of the Registrar General of India; 2009.
- Joshi P, Islam S, Pais P, Reddy S, Dorairaj P, Kazmi K, et al. Risk factors for early myocardial infarction in South Asians compared with individuals in other countries. *JAMA*. 2007;297:286-94.
- Xavier D, Pais P, Devereaux PJ, Xie C, Prabhakaran D, Reddy KS, et al. Treatment and outcomes of acute coronary syndromes in India (CREATE): A prospective analysis of registry data. *Lancet*. 2008;371:1435-42.
- Harikrishnan S, Leeder S, Huffman M, Jeemon P, Prabhakaran D. A Race against Time: The Challenge of CVD in Developing Economies. 2<sup>nd</sup> ed. New Delhi, India: New Delhi Centre for Chronic Disease Control; 2014.
- Grover G, Dutta R, Gadpayle AK, Saha S. Changing patterns of cardiovascular risk factors and heart related sickness in relation with acute myocardial infarction in Delhi, India. *J Ind Med Assoc*. 2009;107(9):636-38.
- Auffret V, Loirat A, Leurent G, Martins RP, Filippi E, Coudert I, et al. High-degree atrioventricular block complicating ST segment elevation myocardial infarction in the contemporary era. *Heart*. 2016;102:40-49.
- Harikrishnan P, Gupta T, Palaniswamy C, Kolte D, Khera S, MD, Mujib M, et al. Complete heart block complicating ST-segment elevation myocardial infarction. *JACC*. 2015;1:529-38.
- Singh SM, FitzGerald G, Yan AT, Brieger D, Fox KA, López-Sendón J, et al. High-grade atrioventricular block in acute coronary syndromes: Insights from the Global Registry of Acute Coronary Events. *Eur Heart J*. 2015;36:976-83.
- Bigger JT Jr, Dresdale FJ, Heissenbuttel RH, Weld FM, Wit AL. Ventricular arrhythmias in ischemic heart disease: Mechanism, prevalence, significance, and management. *Prog Cardiovasc Dis*. 1977;19:255-300.
- O'Doherty M, Tayler DI, Quinn E, Vincent R, Chamberlain DA. Five hundred patients with myocardial infarction monitored within one hour of symptoms. *Br Med J (Clin Res Ed)*. 1983;286:1405-08.
- Tran HV, Ash AS, Gore JM, Darling CE, Kiefe CI, Goldberg RJ. Twenty-five year trends (1986-2011) in hospital incidence and case-fatality rates of ventricular tachycardia and ventricular fibrillation complicating acute myocardial infarction. *Am Heart J*. 2019;208:01-10.
- Thygesen K, Alpert JS, Jaffe AS, Chaitman BR, Bax JJ, Morrow DA, et al. Fourth Universal definition of myocardial infarction (2018). *J Am Coll Cardiol*. 2018;72(18):2231-64.
- National cholesterol education programme (Bethesda, MD, National Heart, Lung and Blood institute, NIH, 2001, Harrison 18<sup>th</sup> edition.
- Kumar V, Singh MP, Agrawal PK, Kumar A, Chauhan S. Study of arrhythmias in acute myocardial infarction within 48 hours. *Int J Adv Med*. 2017;4:103-07.
- Patil PR, Khatana P, Patil DR. Incidence of cardiac arrhythmias in patients with acute myocardial infarction during the first 48 hours of the onset of chest pain. *Int J Adv Med*. 2017;4:1144-49.
- Stone PH, Muller JE, Hartwell T, York BJ, Rutherford JD, Parker CB, et al. The effect of diabetes mellitus on prognosis and serial left ventricular function after acute myocardial infarction: Contribution of both coronary disease and diastolic left ventricular dysfunction to the adverse prognosis. The MILIS Study Group. *J Am Coll Cardiol*. 1989;14(1):49-57.
- Canto JG, Kiefe CI, Rogers WJ, Peterson ED, Frederick PD, French WJ, et al. Number of coronary heart disease risk factors and mortality in patients with first myocardial infarction. *JAMA*. 2011;306(19):2120-27.
- Marangmei L, Singh SK, Devi KB, Raut SS, Chongtham DS, Singh KB. Profile of cardiac arrhythmia in acute myocardial infarction patients within 48 hours of admission: A hospital based study at RIMS Imphal. *J Med Soc*. 2014;28:175-79.
- Aufderheide TP. Arrhythmias associated with Acute myocardial infarction and thrombolysis. *Emergency Medicine Clinics of North America*. 1998;16(3):583-600.
- Hreybe H, Saba S. Location of acute myocardial infarction and associated arrhythmias and outcome. *Clin Cardiol*. 2009;32(5):274-77.
- Feigl D, Ashkenazy J, Kishon Y. Early and late atrioventricular block in acute inferior myocardial infarction. *J Am Coll Cardiol*. 1984;4:35-38.
- Aplin M, Engström T, Vejstrup NG, Clemmensen P, Torp-Pedersen C, Køber L, et al. Prognostic importance of complete atrioventricular block complicating acute myocardial infarction. *Am J Cardiol*. 2003;92:853-56.
- Stone PH, Raabe DS, Jaffe AS, Gustafson N, Muller JE, Turi ZG, et al. Prognostic significance of location and type of myocardial infarction: Independent adverse outcome associated with anterior location. *J Am Coll Cardiol*. 1988;11(3):453-63.
- Haim M, Hod H, Reisin L, Kornowski R, Reicher-Reiss H, Goldbourt U, et al. Comparison of short- and long-term prognosis in patients with anterior wall versus inferior or lateral wall non-Q-wave acute myocardial infarction. Secondary Prevention Reinfarction Israeli Nifedipine Trial (SPRINT) Study Group. *Am J Cardiol*. 1997;79(6):717-21.
- Becker RC, Burns M, Gore JM, Spencer FA, Ball SP, French W, et al. Early assessment and in-hospital management of patients with acute myocardial infarction at increased risk for adverse outcomes: A nationwide perspective of current clinical practice. The National Registry of Myocardial Infarction (NRM-2) Participants. *Am Heart J*. 1998;135:786-96.
- Califf RM, Pieper KS, Lee KL, Van De Werf F, Simes RJ, Armstrong PW, et al. Prediction of 1-year survival after thrombolysis for acute myocardial infarction in the global utilization of streptokinase and TPA for occluded coronary arteries trial. *Circulation*. 2000;101(19):2231-38.
- Fresco C, Carinci F, Maggioni AP, Ciampi A, Nicolucci A, Santoro E, et al. Very early assessment of risk for in-hospital death among 11,483 patients with acute myocardial infarction. GISSI investigators. *Am Heart J*. 1999;138:1058-64.
- Huey BL, Gheorghide M, Crampton RS, Beller GA, Kaiser DL, Watson DD, et al. Acute non-Q wave myocardial infarction associated with early ST segment elevation: Evidence for spontaneous coronary reperfusion and implications for thrombolytic trials. *J Am Coll Cardiol*. 1987;9:18-25.
- Mauri F, Franzosi MG, Maggioni AP, Santoro E, Santoro L. Clinical value of 12-lead electrocardiography to predict the long-term prognosis of GISSI-1 patients. *J Am Coll Cardiol*. 2002;39:1594-600.
- Hathaway WR, Peterson ED, Wagner GS, Granger CB, Zabel KM, Pieper KS, et al. Prognostic significance of the initial electrocardiogram in patients with acute myocardial infarction. GUSTO-I Investigators. Global Utilization of Streptokinase and t-PA for Occluded Coronary Arteries. *JAMA*. 1998;279:387-91.
- Giannitsis E, Steen H, Kurz K, Ivancic B, Simon AC, Futterer S, et al. Cardiac magnetic resonance imaging study for quantification of infarct size comparing directly serial versus single time-point measurements of cardiac troponin T. *J Am Coll Cardiol*. 2008;51:307-14.
- Arruda-Olson AM, Roger VL, Jaffe AS, Hodge DO, Gibbons RJ, Miller TD. Troponin T levels and infarct size by SPECT myocardial perfusion imaging. *JACC Cardiovasc Imaging*. 2011;4:523-33.
- Mayr A, Mair J, Klug G, Schocke M, Pedernig K, Trieb T, et al. Cardiac troponin T and creatine kinase predict mid-term infarct size and left ventricular function after acute myocardial infarction: A cardiac MR study. *J Magn Reson Imaging*. 2011;33:847-54.
- Douglas PS, Garcia MJ, Haines DE, Lai WW, Manning WJ, Patel AR, et al: ACCF/AHA/ASA/ASNC/HFSA/HRS/SCAI/SCCM/SCCT/SCMR 2011 appropriate use criteria for echocardiography. A report of the American college of cardiology foundation appropriate use criteria task force, American society of echocardiography, American heart association, American society of nuclear cardiology, heart failure society of America, heart rhythm society, society for cardiovascular angiography and interventions, society of critical care medicine, society of cardiovascular computed tomography, and society for cardiovascular magnetic resonance endorsed by the American college of chest physicians. *J Am Coll Cardiol*. 2011;57(9):1126-66.
- Cheitlin MD, Armstrong WF, Aurigemma GP, Beller GA, Bierman FZ, Davis JL, et al. ACC/AHA/ASE 2003 guideline update for the clinical application of echocardiography: summary article: A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (ACC/AHA/ASE Committee to Update the 1997 Guidelines for the Clinical Application of Echocardiography). *Circulation*. 2003;108:1146-62.
- Stamm RB, Gibson RS, Bishop HL, Carabello BA, Beller GA, Martin RP. Echocardiographic detection of infarct-localized asynergy and remote asynergy during acute myocardial infarction: Correlation with the extent of angiographic coronary disease. *Circulation*. 1983;67:233-44.
- Heger JJ, Weyman AE, Wann LS, Dillon JC, Feigenbaum H. Cross-sectional echocardiography in acute myocardial infarction: Detection and localization of regional left ventricular asynergy. *Circulation*. 1979;60:531-38.
- Andersen HR, Falk E, Nielsen D. Right ventricular infarction: Frequency, size and topography in coronary heart disease: A prospective study comprising 107 consecutive autopsies from a coronary care unit. *J Am Coll Cardiol*. 1987;10:1223-32.
- Lorell B, Leinbach RC, Pohost GM, Hutter AM, Leinbach RC, Desanctis RW, et al. Right ventricular infarction. Clinical diagnosis and differentiation from cardiac tamponade and pericardial constriction. *Am J Cardiol*. 1979;43:465-71.
- Arditti A, Lewin RF, Hellman C, Sclarovsky S, Strasberg B, Agmon J. Right ventricular dysfunction in acute inferoposterior myocardial infarction. An echocardiographic and isotopic study. *Chest*. 1985;87:307-14.
- Young E, Cohn PF, Gorlin R, Levine HD, Herman MV. Vectorcardiographic diagnosis and electrocardiographic correlation in left ventricular asynergy due to coronary artery disease. I. Severe asynergy of the anterior and apical segments. *Circulation*. 1975;51:467-76.
- Bogaty P, Boyer L, Rousseau L, Arsenault M. Is anteroseptal myocardial infarction an appropriate term? *Am J Med*. 2002;113:37-41.

- [46] Newby KH, Thompson T, Stebbins A, Topol EJ, Califf RM, Natale A. Sustained ventricular arrhythmias in patients receiving thrombolytic therapy: Incidence and outcomes. The GUSTO Investigators. *Circulation*. 1998;98:2567-73.
- [47] AL-Khatib SM, Granger CB, Huang Y, Lee KL, Califf RM, Simoons ML, et al. Sustained ventricular arrhythmias among people with acute coronary syndrome with no ST-segment elevation: Incidences, predictors, and outcomes. *Circulation*. 2002;106:309-12.
- [48] Solodky A, Assali A, Herz I, Hasdai D, Kusniec J, Sulkes J, et al. Early Development of high-degree atrioventricular block in inferior acute myocardial infarction is predicted by a J-Point/R-Wave ratio above 0.5 on admission. *Cardiology*. 1998;90:274-79.
- [49] Meine TJ, Al-Khatib SM, Alexander JH, Granger CB, White HD, Kilaru R, et al. Incidence, predictors, and outcomes of high-degree atrioventricular block complicating acute myocardial infarction treated with thrombolytic therapy. *Am Heart J*. 2005;149:670-74.
- [50] Gang UJ, Hvelplund A, Pedersen S, Iversen A, Jøns C, Abildstrøm SZ, et al. High-degree atrioventricular block complicating ST-segment elevation myocardial infarction in the era of primary percutaneous coronary intervention. *Europace*. 2012;14(11):1639-45.
- [51] Kim HL, Kim SH, Seo JB, Chung WY, Zo JH, Kim MA, et al. Influence of second- and third-degree heart block on 30-day outcome following acute myocardial infarction in the drug-eluting stent era. *Am J Cardiol*. 2014;114(11):1658-62.
- [52] Goldberg RJ, Zevallos JC, Yarzebski J, Alpert JS, Gore JM, Chen Z, et al. Prognosis of acute myocardial infarction complicated by complete heart block (the Worcester Heart Attack Study). *Am J Cardiol*. 1992;69(14):1135-41.
- [53] Crimm A, Severance HW Jr, Coffey K, Mckinnis R, Wagner GS, Califf RM. Prognostic significance of isolated sinus tachycardia during first three days of acute myocardial infarction. *Am J Med*. 1984;76:983-88.
- [54] Braunwalds Heart disease textbook of Cardiovascular Medicine, Eleventh edition, Chapter 37, 706-10.
- [55] Braunwalds Heart disease textbook of Cardiovascular Medicine, Eleventh edition, Chapter 59, 1123-72.
- [56] Crenshaw BS, Ward SR, Granger CB, Stebbins AL, Topol EJ, Califf RM. Atrial fibrillation in the setting of acute myocardial infarction: the GUSTO-I experience. Global Utilization of Streptokinase and TPA for Occluded Coronary Arteries. *J Am Coll Cardiol*. 1997;30:406-13.
- [57] Eldar M, Canetti M, Rotstein Z, Boyko V, Gottlieb S, Kaplinsky E, et al. Significance of paroxysmal atrial fibrillation complicating acute myocardial infarction in the thrombolytic era. SPRINT and Thrombolytic Survey Groups. *Circulation*. 1998;97:965-70.
- [58] Goldberg RJ, Seeley D, Becker RC, Brady P, Chen ZY, Osganian V, et al. Impact of atrial fibrillation on the in-hospital and long-term survival of patients with acute myocardial infarction: A community-wide perspective. *Am Heart J*. 1990;119:996-1001.
- [59] DeWood MA, Spores J, Notske R, Mouser LT, Burroughs R, Golden MS, et al. Prevalence of total coronary occlusion during the early hours of transmural myocardial infarction. *N Engl J Med*. 1980;303:897-902.
- [60] Reiner JS, Lundergan CF, van den Brand M, Boland J, Thompson MA, Machecourt J, et al. Early angiography cannot predict postthrombolytic coronary reocclusion: Observations from the GUSTO angiographic study. Global Utilization of Streptokinase and t-PA for Occluded Coronary Arteries. *J Am Coll Cardiol*. 1994;24:1439-44.

**PARTICULARS OF CONTRIBUTORS:**

1. Associate Professor, Department of General Medicine, Sri Ramachandra Institute of Higher Education and Research, Chennai, Tamil Nadu, India.
2. Postgraduate, Department of General Medicine, Sri Ramachandra Institute of Higher Education and Research, Chennai, Tamil Nadu, India.
3. Professor, Department of General Medicine, Sri Ramachandra Institute of Higher Education and Research, Chennai, Tamil Nadu, India.

**NAME, ADDRESS, E-MAIL ID OF THE CORRESPONDING AUTHOR:**

Dr. SR Ramakrishnan,  
1/16, 8 Street, Lakshmi Nagar, Nanganallur, Chennai-600061, Tamil Nadu, India.  
E-mail: drsrk\_71@yahoo.com

**PLAGIARISM CHECKING METHODS:** [Jain H et al.]

- Plagiarism X-checker: Dec 10, 2019
- Manual Googling: Jan 09, 2020
- iThenticate Software: Apr 15, 2020 (13%)

**ETYMOLOGY:** Author Origin**AUTHOR DECLARATION:**

- Financial or Other Competing Interests: None
- Was Ethics Committee Approval obtained for this study? Yes
- Was informed consent obtained from the subjects involved in the study? Yes
- For any images presented appropriate consent has been obtained from the subjects. NA

Date of Submission: **Dec 09, 2019**Date of Peer Review: **Feb 01, 2020**Date of Acceptance: **Mar 28, 2020**Date of Publishing: **May 01, 2020**