

# **Study of Acute Myocardial Infarction Admitted to ICCU with Special Reference to Arrhythmias**

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## **ABSTRACT**

### **BACKGROUND**

In this study, we wanted to evaluate patients with acute myocardial infarction admitted to ICCU with special reference to arrhythmias.

### **METHODS**

This was a hospital based cross sectional study conducted among 50 patients who presented to ICCU, Narayana Medical College and Hospital, at Nellore, Andhra Pradesh, over a period of one year from March 2014 to February 2025 after obtaining clearance from Institutional Ethics Committee and written informed consent from the study participants.

### **RESULTS**

Syncope is significantly dominant in NSTEMI. Left ventricular ejection fraction (LVEF) > 50 % observed in 50 % cases of NSTEMI in comparison to 2.5 % in STEMI which is statistically significant. Statistically significant variables in STEMI included high mean TIMI risk score > 5 and depressed LVEF < 50 %. Statistically significant variables in NSTEMI included hypertension (HTN), syncope occurrence and LVEF > 50 %.

### **CONCLUSION**

HTN is a major risk factor in NSTEMI. Syncope is common with IWMI. Palpitation is common with AWTMI. Mean TIMI risk score is higher in STEMI. Serum creatinine and electrolytes will be normal in majority of the cases. LV dysfunction EF < 50 % will be common in STEMI group. Sinus tachycardia is the commonest among AWTMI/ALMI patients. Majority of the patients will have single vessel disease. AWTMI with SVD will have LAD lesions on CAG and IWMI group PT had RCA or CX lesions on the ECG. TVD is common in NSTEMI and AWTMI groups.

### **KEYWORDS**

Acute Myocardial Infarction, ICCU, Arrhythmias

## **INTRODUCTION**

Acute coronary syndrome (ACS) represents a Global epidemic, and is intimidating large as the new epidemic afflicting population worldwide, especially in the sub-continent. According to the National Commission on Macro-economics and Health, there would be around 62 million patients with coronary artery disease (CAD) by 2015 in India, and of these, 23 million would be younger than 40 years of age.<sup>[1]</sup> CAD affects Indians with greater frequency and at a younger age than in the developed countries, as well as many other developing

countries. As a leading cause of morbidity and mortality, ACS are major public health problems.<sup>[2]</sup> By 2020 it is estimated that ACS will become a major cause of death in all the regions of the world. Many of these deaths are attributed to the development of arrhythmias during periods of myocardial infarction.<sup>[3]</sup> There is a view that the cascade leading to sudden death from arrhythmias can be predicted by certain interactions among structural and functional abnormalities.<sup>[4]</sup> The search for new tools for prediction, the initiation of well-designed intervention trials are the steps that must be taken.

### Aims and Objectives

- To study the incidence of arrhythmias in (AMI) both STEMI and NSTEMI with respect to type of arrhythmia, age distribution, sex, risk factors, clinical presentation and location of infarction in patient population admitted to hospital.
- To evaluate its prognostic value in patient outcome.

### METHODS

This was a hospital based cross sectional study conducted among 50 patients who presented with cardiac ICCU to the Department of Narayana Medical College and Hospital, at Nellore, Andhra Pradesh, over a period of one year from March 2014 to February 2025 after obtaining clearance from Institutional Ethics Committee and written informed consent from the study participants.

### Inclusion Criteria

1. Patients aged between 30 - 70 years.
2. Patients who are diagnosed with AMI [STEMI and NSTEMI]
3. Patients with manifest arrhythmias [Brady and tachyarrhythmias]

### Exclusion Criteria

1. Age < 30 years and > 70 years
2. Stable and unstable angina patients
3. Suspected CAD patients
4. Old CAD patients

### Statistical Methods

The data values have been entered into MS-Excel and statistical analysis has been done by using IBM Statistical Package for Social Sciences (SPSS) Version 20.0. Chi-square test has been used.

### RESULTS

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	STEMI	40	80.0	80.0	80.0
	NSTEMI	8	16.0	16.0	96.0
	LBBB	2	4.0	4.0	100.0
	Total	50	100.0	100.0	

<i>Frequency Percentage of Cases</i>					
		<b>Frequency</b>	<b>Percent</b>	<b>Valid Percent</b>	<b>Cumulative Percent</b>
Valid	ALMI	7	14.0	14.0	14.0
	AWMI	17	34.0	34.0	48.0
	IW+PWTMI	2	4.0	4.0	52.0
	IW+RVMI	3	6.0	6.0	58.0
	IWMI	11	22.0	22.0	80.0
	LBBB	2	4.0	4.0	84.0
	NSTEMI	8	16.0	16.0	100.0
	Total	50	100.0	100.0	
<i>Frequency Percentage of STEMI Cases</i>					
<i>Table 1</i>					

Majority of the cases constituted STEMI, followed by NSTEMI and 2 cases of new onset LBBB. Among STEMI patients, AWTMI constituted 34 %, followed by IWMI 22 %, ALMI 14 %, IW with RVMI 6 % and IW with PWTMI 4 %.

<b>Variables</b>	<b>STEMI [40]</b>	<b>NSTEMI [8]</b>	<b>P Value*</b>
Age	53.5 +/- 10	57.3 +/- 8	0.35
<b>Sex</b>			
M	31[77.5]	7[87.5]	0.52
F	9[22.5]	1[12.5]	
BMI	22.7+/- 3.4	23.4+/- 3.1	0.61
DM	13[32.5]	3[37.5]	0.78
HTN	11[27.5]	6[75]	0.01
Smoking	29[72.5]	4[50]	0.21
Dyslipidaemia	27[67.5]	4[50]	0.34
Angina	38[95]	6[75]	0.06
Dyspnoea	25[62.5]	5[62.5]	1
Syncope	1[2.5]	2[25]	0.01
Palpitation	13[32.5]	2[25]	0.67
<b>Killips class</b>			
Mild- moderate	33[82.5]	7 [87.5]	0.72
severe	7 [17.5]	1 [12.5]	
Timi risk score	6.3+/- 2	4.1+/- 1.1	0.005
<b>S. creatinine</b>			
< 1.5	32[80]	7 [87.5]	0.62
>1.5	8 [20]	1 [12.5]	
<b>2D echo</b>			
< 50 %	39[97.5]	4 [50]	< 0.001
> 50 %	1 [2.5]	4 [50]	
<b>CAG</b>			
SVD	27[67.5]	4 [50]	0.54

DVD	7[17.5]	2 [25]	0.28
TVD	4[10]	2 [25]	
<b>Treatment</b>			
PCI	28[70]	6 [75]	
CABG	3 [7.5]	2 [25]	
MM	7 [17.5]		
DEATH	2 [5]	0	
<i>Baseline Clinical Characteristics According to Type of MI</i>			
*P value & lt; 0.05, significant			
<i>Table 2</i>			

LVEF > 50 % was observed in 50 % cases of NSTEMI in comparison to 2.5 % in STEMI which was statistically significant. Statistically significant variables in STEMI included high mean TIMI risk score > 5 and depressed LVEF < 50 %. Statistically significant variables in NSTEMI included HTN, Syncope occurrence and LVEF > 50 %.

<b>Variables</b>	<b>AWMI* [24]</b>	<b>IWMI**[16]</b>	<b>P Value***</b>
Age	53.5 +/- 11	53.6 +/- 9	0.95
<b>Sex</b>			
M	19[79.2]	12[75]	0.75
F	5 [20.8]	4 [25]	
BMI	22.9+/-4	22.5+/-2.4	0.71
DM	9 [37.5]	4 [25]	0.4
HTN	8[33.3]	3[18.8]	0.31
Smoking	18[75]	11[68.8]	0.66
Dyslipidaemia	18[75]	9 [56.2]	0.21
Angina	23 [95.8]	15[93.8]	0.76
Dyspnoea	16 [66.7]	9[56.2]	0.5
Syncope	0	1 [6.2]	0.21
Palpitation	10[41.7]	3 [18.8]	0.13
<b>Killips class</b>			
Mild- moderate	21[87.5]	12[75]	0.3
severe	3 [12.5]	4 [25]	
Timi risk score	6.7+/-1.8	5.8+/-2.2	0.17
<b>S. Creatinine</b>			
< 1.5	21[87.5]	11[68.8]	0.14
>1.5	3 [12.5]	5 [31.2]	
<b>2D echo</b>			
< 50 %	24[100]	15[93.8]	0.21
> 50 %	0	1 [6.2]	
<b>CAG</b>			
SVD	17[70.8]	10[62.5]	0.08
DVD	3[12.5]	4 [25]	

TVD	4[16.7]	0	
<b>Treatment</b>			
PCI	16[66.7]	12[75]	0.14
CABG	3 [12.5]	0	
MM	5 [20.8]	2 [12.5]	
<i>Baseline Clinical Characteristics in STEMI</i>			
<b>*Includes both AW/ALMI. **Includes IW along with PW and RVMI ***p &lt; 0.05 significant</b>			
<i>Table 3</i>			

Syncope was common in IWMI. Palpitation was common in AWMI group. Patients who belonged to killips class 3 - 4 were common in IWMI group. Mean TIMI risk score is 6.7 +/- 1.8 in AWMI and 5.8 +/- 2.2 in IWMI. Higher risk score was observed in AWMI group. Patients whose serum creatinine was > 1.5 were common with IWMI group. LVEF < 50 % observed in 100 % of patients with AWMI in comparison to 93.8 % in IWMI. LVEF > 50 % was observed in 6.2 % cases of IWMI.

Patients with SVD 70.8 % vs 62.5 %, DVD 12.5 % vs 25 %, TVD 16.7 % vs 0 % were noted on CAG between AWMI and IWMI groups respectively. Majority of the patients underwent PCI [66.7 % vs 75 %], CABG [12.5 % vs 0 %], and medical management [20.8 % vs 12.5 %] in AWMI and IWMI groups respectively.

## DISCUSSION

Present study had 36 % diabetic, 36 % hypertensive, 68 % patients were smokers and 64 % cases were of dyslipidaemia. The study by LINDA et al. constituted 22% diabetic, 26 % hypertensive, and 47 % patients were smokers.<sup>[5]</sup> HTN was a major risk factor in NSTEMI group whereas other risk factors were of equal distribution in both the study groups. Within STEMI, the incidence of the risk factors did not differ significantly between AWMI vs IWMI study groups.

In a meta-analysis reported by Hokansen et al. for men and women the univariate RRs for TG were 1.32 and 1.76 respectively indicating approximately 30 % and 75 % increased risk of CHD in men and women.<sup>[6]</sup>

Angina was the predominant symptom 88 %, followed by dyspnoea 64 %, palpitation 32 %, and syncope 6 %. Syncope was a common symptom associated with STEMI vs NSTEMI [2.5 % vs 25 %] p value - 0.01. It was common with IWMI in comparison to AWMI [6.2 % vs 0]. Palpitation was a common symptom with AWMI [41.7 % vs 18.8 %].

Cardiac syncope occurs suddenly and restoration of consciousness occurs quickly. Patients with neurocardiogenic syncope may have an early warning signs, appear ashen and diaphoretic, and revive more slowly, albeit without signs of seizure or a prolonged postictal state. The likelihood of a cardiac arrhythmia is modestly increased with known history of cardiac disease (LR, 2.03; 95 % CI, 1.33 to 3.11) and decreased when symptoms resolve within 5 minutes (LR, 0.38; 95 % CI, 0.22 to 0.63) or in the presence of panic disorder (LR, 0.26; 95 % CI, 0.07 to 1.01)<sup>[7]</sup>

Majority of the patients belonged to killips class 1 - 2 [84 %] in both the MI groups. In the present study, high killips score was noted with IWMI 25 % vs 12.5 % in AWMi group. High killips score was noted in 8 patients, 4 in IWMI group, 3 in AWMi group 1 case of new onset LBBB.

Serum creatinine levels in majority of the patients were  $\leq 1.5$  mg/dl [80 %] and  $> 1.5$  mg/dl was noted in 20 % of the patients. Elevated serum creatinine was common with STEMI 20 % vs 12.5 % in NSTEMI. Of the STEMI patients, it was elevated in IWMI group in comparison to the AWMi group. [31.2 % vs. 12.5 %]. In the present study, most of the patients with elevated serum creatinine levels had 2 or more risk factors. In acute myocardial infarction, impaired renal function may result from underlying kidney disease, acute renal failure, and the effect of drugs and contrast agents used during diagnostic procedures or treatment. These various causes may co-exist, resulting in significantly worse outcomes. Prompt recognition of the degree of renal function impairment and institution of appropriate preventive and therapeutic measures are among major goals of in-hospital management of these patients.<sup>[8]</sup>

3 cases had dyselektrolemia. 1 with hypokalemia and the other 2 with hyperkalemia. 3 patients had STEMI and 2 patients with hyperkalemia belonged to AWMi group and 1 patient with hypokalemia belonged to IWMI group. Patient with hypokalemia had elevated baseline creatinine, developed unstable VT and died. Hypokalemia can increase the risk of developing VT. Low serum potassium levels should be identified quickly after patient's admission for STEMI and should be treated promptly. We strive to maintain the serum potassium level above 4.5 mEq/liter and serum magnesium level above 2 mEq/liter. Rapid abolition of sustained ventricular tachycardia in patients with STEMI is mandatory because of its deleterious effect on pump function and because it frequently deteriorates into ventricular fibrillation.<sup>[9]</sup>

Left ventricular (LV) systolic function is an important prognostic factor in coronary heart disease. Left ventricular ejection fraction (LVEF) should be assessed in all patients after acute myocardial infarction (AMI). Although reperfusion therapy has been found to be effective in the reduction of complications of AMI, LVEF impairment is a common consequence of an acute coronary event. In one study conducted by Mateus et al. LV systolic function was considered depressed when ejection fraction was  $< 45$  %. Incidence of LV dysfunction was 55.8 % in STEMI patients. In STEMI patients, previous cardiovascular risk factors have a significant impact on the likelihood of LV dysfunction and hence could influence long-term prognosis.<sup>[10]</sup>

The risk of sudden death from cardiac causes has increased among survivors of acute myocardial infarction with reduced left ventricular systolic function. We assessed the risk and time course of sudden death in high-risk patients after MI. The risk was highest in the first 30 days after myocardial infarction - 1.4 percent per month (95 percent confidence interval, 1.2 to 1.6 percent). Patients with a left ventricular ejection fraction of 30 percent or less were at highest risk in this early period (rate, 2.3 percent per month; 95 percent confidence interval, 1.8 to 2.8 percent). Nineteen percent of all sudden deaths or episodes of cardiac arrest with resuscitation occurred within the first 30 days after myocardial infarction, and 83 percent of all patients who died suddenly in the first 30 days did so after hospital discharge. Each decrease of 5 percentage points in the left ventricular ejection fraction was associated with a

21 percent adjusted increase in the risk of sudden death or cardiac arrest with resuscitation in the first 30 days.<sup>[11]</sup>

ST was the commonest finding in the study [42 %]. Both STEMI and NSTEMI ST were common [42.5 % vs. 50 %]. Among patients with STEMI, it was commonly seen in AWTMI group compared to IWTMI group [62.5 % vs 12.5 %]. In a study conducted by Sangita et al. from Gujarat S. tachycardia was more common (68 %) than S. bradycardia (19 %). It occurred more commonly with AWTMI.<sup>[12]</sup> This arrhythmia is typically associated with augmented sympathetic activity and may provoke transient hypertension or hypotension. Common causes are anxiety, persistent pain, left ventricular failure, fever, pericarditis, hypovolemia, pulmonary embolism, and the administration of cardio accelerator drugs such as atropine, epinephrine, or dopamine; rarely, it occurs in patients with atrial infarction. Sinus tachycardia is particularly common in patients with anterior infarction, especially if there is significant accompanying left ventricular dysfunction. It is an undesirable rhythm in patients with STEMI because it results in an augmentation of myocardial oxygen consumption, as well as a reduction in the time available for coronary perfusion, thereby intensifying myocardial ischemia and/or external myocardial necrosis. Persistent sinus tachycardia can signify persistent heart failure and, under these circumstances, connotes poor prognosis and excess mortality.<sup>[13]</sup>

ST with LBBB accounted for 2.1% of all arrhythmias. It accounted for 2.5 % of the cases in STEMI not documented in NSTEMI. It was common in the AWTMI group. SB/1 degree AV block accounted for the next common documented arrhythmias in the study [12.5 %]. It was common with STEMI (6 patients) [15 % vs 0] in comparison to NSTEMI. All the patients belonged to IWTMI group. It was the commonest arrhythmia among IWTMI group [37.5 %].

Sinus bradycardia occurs commonly during the early phases of STEMI, particularly in patients with inferior and posterior infarction. On the basis of data obtained in experimental infarction studies and from some clinical observations, the increased vagal tone that produces sinus bradycardia during the early phase of STEMI may actually be protective, perhaps because it reduces myocardial oxygen demands. Thus, the acute mortality rate appears similar in patients with sinus bradycardia to the rate in those without this arrhythmia.<sup>[14]</sup>

First-degree AV block occurs in fewer than 15 % of patients with acute MI admitted to coronary care units. His bundle electrocardiographic studies have shown that, in most of these patients, the AVN is the site of conduction block. AV block is more common in the setting of inferior MI. In the thrombolysis in myocardial infarction (TIMI) II study, high-degree (second- or third-degree) AV block occurred in 6.3 % of patients at the time of presentation and in 5.7 % in the first 24 hours after thrombolytic therapy. Patients with AV block at the time of presentation had a higher in-hospital mortality than patients without AV block; however, the 2 groups had similar mortalities during the following year.<sup>[15]</sup>

CHB occurred in 4 patients and accounted for 8 % of the total arrhythmias. 3 patients belonged to STEMI and 1 patient to NSTEMI. All the 3 patients in STEMI belonged to IWTMI group. It was the second common arrhythmia after SB/1 degree AV block in IWTMI group [18.8 %]. Of the 2 patients who died, one patient suffered IWTMI + RVMI.

Complete AV block can occur in patients with anterior or inferior infarction. Complete heart block in patients with inferior infarction usually results from an intranodal or

supranodal lesion and develops gradually, often progressing from first-degree or type I second-degree block.<sup>[16]</sup>

The escape rhythm is usually stable, without systole and often junctional, with a rate exceeding 40 beats/min and a narrow QRS complex in 70 % of cases and a slower rate and wide QRS in the others. This form of complete AV block is often transient, may be responsive to pharmacologic antagonism of adenosine with methylxanthines,<sup>[17]</sup> and resolves in most patients within a few days. VT constituted 8 % of the total arrhythmia burden in the study. 4 cases that manifested with VT belonged to STEMI group. Of them, 3 cases in the IWMI group and 1 case in AWTMI group were noted. VT commonly occurred in the 1<sup>st</sup> 24 hours of admission. Mortality was noted in 1 case diagnosed with IWMI + PWMI. Pt who expired had a high killips score, TIMI risk score, elevated serum creatinine and hypokalemia at baseline.

2 cases of new onset LBBB and 2 cases of STEMI with LBBB were noted in the study comprising 4 % of arrhythmia each. In patients with STEMI it was common in AWTMI group. The other 2 cases of new onset LBBB were considered differently from the STEMI vs. NSTEMI. The intraventricular conduction system of the LV is composed of fibres of bundle of His that become the main LBB and then divides into anterior and posterior fascicles, further branching to become the distal conduction system. In contrast to RBB, which is a discrete structure that can be injured acutely with a small focal insult, the LBB is a large and diffuse structure that typically requires a large insult to lead to acute injury.<sup>[19]</sup>

When a new LBBB is caused by AMI, the site of infarction is usually anterior or anteroapical, with the infarction involving large myocardial territory. Inferior or posterior infarctions uncommonly may result in a new LBBB from involvement of the more proximal portion of the conduction system supplied by the AV nodal artery.<sup>[20]</sup> In a study by Mehta et al. who assessed the prevalence of true AMI and the need for emergent revascularization among patients with new or presumably new left bundle branch block (n LBBB) for whom the primary PCI protocol was activated the reported incidence of n LBBB was 8.6 %.<sup>[21]</sup>

2 cases of LAHB identified in the study [4 %], both STEMI cases [5 %] and AWTMI group [8.3 %].

In the prethrombolytic era, studies of intraventricular conduction disturbances (i.e., block within one or more of the three subdivisions [fascicles] of the His-Purkinje system [the anterior and posterior divisions of the left bundle and the right bundle]) had been reported to occur in 5 % to 10 % of patients with STEMI. More recent series in the reperfusion era have suggested that intraventricular blocks occur in about 2 % to 5 % of patients with MI.<sup>[22]</sup>

Vpc's were not a frequent association in the study. Only 1 case had frequent VPC's and belonged to AWTMI group. The Framingham heart study (with 1-h ambulatory ECG) suggested that the prevalence rate of 1 or more VPCs per hour was 33 % in men without coronary artery disease (CAD) and 32 % in women without CAD. Among patients with CAD, the prevalence rate of 1 or more VPCs was 58 % in men and 49 % in women.

The prognostic significance of VPCs is variable and again, best interpreted in the context of the underlying cardiac condition. Observational studies and inferences from typical electrophysiology studies were initially focused on ventricular ectopy triggering ventricular tachycardia (VT), which, in turn, can degenerate into ventricular fibrillation, as a mechanism for sudden cardiac death. The treatment paradigm in the 1970s and 1980s was to eliminate



VPCs in patients after myocardial infarction (MI). The Cardiac Arrhythmia Suppression Trial (CAST) and other arrhythmia suppression studies have demonstrated that eliminating VPCs with available antiarrhythmic drugs increases the risk of death to patients without providing any measurable benefit.<sup>[23]</sup>

AF occurred in 1 STEMI case that belonged to AWMi group. Low atrial rhythm occurred in 1 case of NSTEMI.

AF is usually transient and tends to occur in patients with left ventricular failure but also occurs in those with pericarditis and ischemic injury to the atria and right ventricular infarction.<sup>[24]</sup> The increased ventricular rate and loss of the atrial contribution to left ventricular filling result in a significant reduction in cardiac output. Atrial fibrillation during STEMI is associated with increased mortality and stroke, particularly in patients with anterior wall infarction.<sup>[25]</sup>

NSVT occurred in 1 patient who belonged to STEMI and IWMI group in the study. NSVT early after presentation do not appear to be associated with an increased mortality risk, either during hospitalization or over the first year. The prevalence of SVD vs. DVD vs. TVD in STEMI is 67.5 % vs. 17.5 % vs. 10 % and in NSTEMI is 50 % vs. 25 % vs. 25 %. 2 cases of new onset LBBB had DVD and SVD respectively.

In the AWMi group, the prevalence of SVD vs. DVD vs. TVD is 70.8 % vs. 12.5 % vs. 16.7 % and in IWMI group 62.5 % vs. 25 % vs. 0. TVD was common in the AWMi group. SVD in AWMi correlated with LAD lesion on CAG. DVD in AWMi group had lesions commonly in the LAD/RCA.

In the IWMI subgroup, SVD correlated with RCA lesions in 60 %, LCX lesions in 30 % and LAD lesions in 10 %. Among DVD patients, most common lesions were identified in LAD/RCA [3 cases] followed by LCX/RCA [1 case]. 2 patients did not undergo CAG because of early mortality.

Among NSTEMI cases with SVD, lesions in RCA were found in 2 cases, diagonal in 1 case and LAD in 1 case. In patients with DVD on CAG, the common culprit lesions were identified in LAD/RCA.

The extent of epicardial CAD among patients with UA/NSTEMI randomized to the invasive arm of the TACTICS–TIMI 18 trial, who systematically underwent angiography, was as follows: 34 % had significant obstruction (> 50 % luminal diameter stenosis) of three vessels; 28 % had two-vessel disease; 26 % had single-vessel disease; and 13 % had no coronary stenosis > 50 %. Approximately 10 % had left main stem stenosis > 50 %.

PCI was the commonest modality of revascularisation in the study as majority of the cases had SVD and DVD. [70 % vs. 75 %] in STEMI vs. NSTEMI. CABG was performed in 7.5 % vs. 25 % cases. 17.5 % of STEMI patients were considered medical therapy as they had non-occlusive CAD.

In STEMI patients PCI in [66.7 % vs. 75 %] and CABG in [3 % vs. 0 %] and medical therapy in 20.8 % vs. 12.5 % were considered for AWMi vs. IWMI groups respectively. Both the cases of new onset LBBB underwent PCI as revascularisation procedure.

## CONCLUSION

HTN is a major risk factor in NSTEMI. Syncope is common with IWMI. Palpitation is common with AWMi. Mean TIMI risk score is higher in STEMI. Serum creatinine and

electrolytes will be normal in majority of the cases. LV dysfunction EF < 50 % will be common in STEMI group. Sinus tachycardia is the commonest arrhythmia documented in the study both with STEMI and NSTEMI. Sinus tachycardia is the commonest among AW/ALMI patients. AWMi with SVD will have LAD lesions on CAG and IWMi group PT had RCA or CX lesions on the ECG. TVD is common in NSTEMI and AWMi groups.

## REFERENCES

1. Rissam HS, Kishore S, Trehan N. Coronary artery disease in young Indians – the missing link. *J Indian Acad Clin Med* 2001;2(3):128-32.
2. WHO The top 10 causes of death. WHO Sept. 2013.
3. Fabijanic D, Culic V, Bozic I, Miric D, Stipic SS, Radic M, et al. Gender differenced inhospital mortality and mechanisms of death after the first acute myocardial infarction. *Ann Saudi Med* 2006;6(6):455-60.
4. Armstrong WF, McHenry PL. Ambulatory ECG monitoring: can we predict sudden death? *JACC* 1985;5(6):13B-6.
5. 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. *Journal of Medical Society* 2014;28(3):175.
6. Hokansen JE, Austin MA. Plasma triglyceride level is a risk factor for cardiovascular disease independent of HDL-C level: a meta-analysis of population based prospective studies. *J Cardiovasc Risk* 1996;3(2):213-9.
7. Thavendiranathan P, Bagai A, Khoo C, Dorian P, Choudhry NK. Does this patient with palpitations have a cardiac arrhythmia? *JAMA* 2009;302(19):2135-43.
8. Lekston A, Kurek A, Tynior B. Impaired renal function in acute myocardial infarction. *Cardiol J* 2009;16(5):400-6.
9. Mega JL, Morrow DA. STEMI: management. Braunwald's heart disease 9<sup>th</sup> edn. Elsevier Health Sciences 2012: p. 1132.
10. Mateus PS, Dias CC, Betrencourt N, Adão L, Santos L, Sampaio F, et al. Left ventricular dysfunction after acute myocardial infarction-the impact of cardiovascular risk factors. *Rev Port Cardiol* 2005;24(5):727-34.
11. Solomon SD, Zelenkofske S, McMurray JJ, Finn PV, Velazquez E, Ertl G, et al. Sudden death in patients with myocardial infarction and left ventricular dysfunction, heart failure, or both. *N Engl J Med* 2005;352(25):2581-8.
12. Rathod S, Parmar P, Rathod GB, Parikh A. Study of various cardiac arrhythmias in patients of acute myocardial infarction. *IAIM* 2014;1(4):32-41.
13. Mega JL, Morrow DA. STEMI: management. Braunwald's heart disease 9<sup>th</sup> edn. Elsevier Health Sciences 2012: p. 1135.
14. Antman EM, Anbe DT, Armstrong PW, Bates ER, Green LA, Hand M, et al. ACC/AHA guidelines for the management of patients with ST-elevation myocardial infarction: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee to Revise the 1999 Guidelines for the Management of Patients with AMI) *Circulation* 2004;110:e82.
15. Berger PB, Ruocco NA, Ryan TJ, Frederick MM, Jacobs AK, Faxon DP. Incidence and prognostic implications of heart block complicating inferior myocardial infarction treated with thrombolytic therapy: results from TIMI II. *J Am Coll Cardiol* 1992;20(3):533-40.

16. Hreybe H, Saba S. Location of acute myocardial infarction and associated arrhythmias and outcome. *Clin Cardiol* 2009;32(5):274-7.
17. Altun A, Kirdar C, Ozbay G. Effect of aminophylline in patients with atropine-resistant late advanced atrioventricular block during acute inferior myocardial infarction. *Clin Cardiol* 1998;21:759.
18. Henkel DM, Witt BJ, Gersh BJ, Jacobsen SJ, Weston SA, Meverden RA, et al. Ventricular arrhythmias after acute myocardial infarction: a 20-year community study. *Am Heart J* 2006;151(4):806-12.
19. Goldman MJ, Lassers BW, Julian DG. Complete bundle-branch block complicating acute myocardial infarction. *N Engl J Med* 1970;282(5):237-40.
20. Hindman MC, Wagner GS, JaRo M, Atkins JM, Scheinman MM, DeSanctis RW, et al. The clinical significance of bundle branch block complicating acute myocardial infarction. 1. Clinical characteristics, hospital mortality, and one-year follow-up. *Circulation* 1978;58(4):679-88.
21. Mehta N, Huang HD, Bandle S, Wilson JM, Birnbaum Y. Prevalence of acute myocardial infarction in patients with presumably new left bundle-branch block. *J Electrocardiology* 2012;45(4):361-7.
22. Kleemann T, Juenger C, Gitt AK, Schiele R, Schneider S, Senges J, et al. Incidence and clinical impact of right bundle branch block in patients with acute myocardial infarction: ST elevation myocardial infarction versus non-ST elevation myocardial infarction. *Am Heart J* 2008;156(2):256-61.
23. Cantillon DJ. Evaluation and management of premature ventricular complexes. *Cleve Clin J Med* 2013;80(6):377-87.
24. Køber L, Swedberg K, McMurray JJ, Pfeffer MA, Velazquez EJ, Diaz R, et al. Previously known and newly diagnosed atrial fibrillation: a major risk indicator after a myocardial infarction complicated by heart failure or left ventricular dysfunction. *Eur J Heart Fail* 2006;8(6):591-8.
25. Saczynski JS, McManus D, Zhou Z, Spencer F, Yarzebski J, Lessard D, et al. Trends in atrial fibrillation complicating acute myocardial infarction. *Am J Cardiol* 2009;104(2):169-74.