DISSERTATION ON
PROGNOSTIC SIGNIFICANCE OF BUNDLE BRANCH BLOCKS IN ACUTE CORONARY SYNDROME

Submitted in partial fulfilment of
Requirements for

M.D. DEGREE BRANCH I GENERAL MEDICINE
OF
THE TAMILNADU DR.M.G.R. MEDICAL UNIVERSITY,
CHENNAI

MADRAS MEDICAL COLLEGE
CHENNAI - 600 003.
MARCH - 2010.

CERTIFICATE

This is to certify that this dissertation entitled **"PROGNOSTIC SIGNIFICANCE OF BUNDLE BRANCH BLOCKS IN ACUTE CORONARY SYNDROME"** Submitted by **Dr. MOORTHY .P** appearing for Part II M.D. Branch I General Medicine Degree examination in March 2010 is a bonafide record of work done by him under my direct guidance and supervision in partial fulfillment of regulations of the Tamil Nadu Dr.M.G.R. Medical University, Chennai. I forward this to the Tamil Nadu Dr.M.G.R. Medical University, Chennai, Tamil Nadu, India.

Prof. K. SIVA SUBRAMANIAN, MD.,
Professor of Medicine,
Institute of Internal Medicine,
Madras Medical College,
Government General Hospital,
Chennai - 600 003.

Prof.C.Rajendiran,MD.,
Director and Professor,
Institute of Internal Medicine,
Madras Medical College,
Government General Hospital,
Chennai - 600 003.

Prof.J.Mohanasundaram,MD.,Ph.D.DNB
Dean,
Madras Medical College,
Government General Hospital,
Chennai - 600 003.

DECLARATION

I solemnly declare that the dissertation titled is done by me at Madras Medical College & Govt. General Hospital, Chennai during 2007-2010 under the guidance and supervision of **Prof. K. Sivasubramanian.**

The dissertation is submitted to The Tamilnadu Dr.M.G.R. Medical University towards the partial fulfillment of requirements for the award of M.D. Degree (Branch I) in General Medicine.

Place :

Date;

Dr. Moorthy. P.
Postgraduate Student,
M.D. General Medicine,
Institute of Internal Medicine,
Madras Medical College,
Chennai - 600 003.

ACKNOWLEDGMENT

At the outset I would like to thank my beloved Dean, Madras Medical College, **Prof. J. Mohanasundram, M.D., Ph.D.DNB.,** for his kind permission to use the hospital resources for this study.

I would like to express my sincere gratitude to my beloved Professor and Director, Institute of Internal Medicine **Prof. C. Rajendiran, M.D.,** for his guidance and encouragement.

With extreme gratitude, I express my indebtedness to my beloved Chief **Prof. K. Sivasubramanian, M.D.,** for his motivation, advice and valuable criticism, which enabled me to complete this work.

I am extremely thankful to Assistant Professors of Medicine **Dr.G. Rajan, M.D., and Dr.S. Gopalakrishnan, M.D.,** for their kind co-operation and guidance.

I thank all Professors, Assistant Professors, and Post-graduates of the department of cardiology for their valuable support in helping in my study.

I would always remember with extreme sense of thankfulness for the co-operation and criticism shown by my Postgraduate Colleagues.

I am immensely grateful to the generosity shown by the patients who participated in this study. If at all, this study could contribute a little to relieve them from their suffering I feel that I have repaid a part of my debt.

CONTENTS

ABBREVIATIONS

ACS	-	Acute coronary syndrome
AMI	-	Acute myocardial Infarction
BBB	-	Bundle Branch Block
CAD	-	Coronary Artery disease
ECG	-	Electrocardiography
CK-MB	-	Creatine kinase - MB
UA	-	Unstable angina
NSTEMI	-	Non ST Segment Elevation Myocardial Infarction
STEMI	-	ST Segment Elevation Myocardial Infarction
ACC	-	American College of Cardiology.
AHA	-	American Heart Association
PCI	-	Percutaneous Coronary Intervention
CABG	-	Coronary Artery Bypass Graft
EF	-	Ejection Fraction
AV	-	Atrio Ventricular
LBBB	-	Left Bundle Branch Block
RBBB	-	Right Bundle Branch Block
HERO	-	Hirulog Early Reperfusion and Occlusion
ABBB	-	Acute Bundle Branch Block
CCU	-	Coronary Care Unit
CHF	-	Congestive Heart Failure
DM	-	Diabetes Mellitus

SHT	-	Systemic hypertension
MI	-	Myocardial Infarction
BP	-	Blood Pressure
HR	-	Heart Rate
AWMI	-	Anterior Wall Myocardial Infarction
IWMI	-	Inferior Wall Myocardial Infarction
PWMI	-	Posterior Wall Myocardial Infarction
RVMI	-	Right Ventricular Myocardial Infarction
VAT	-	Ventricular Activation Time
VSR	-	Ventricular Septum Rupture
P.(Value)	-	Probability of the Test
LV	-	Left Ventricular
PTCA	-	Percutaneous Transluminal Coronary Angioplasty
LAHB	-	Left Anterior Hemi Block
CHB	-	Complete Heart Block
SK	-	Streptokinase
DD	-	Diastolic Dysfunction
VPC	-	Ventricular Premature Contraction
VT	-	Ventricular Tachycardia
VF	-	Ventricular Fibrillation

INTRODUCTION

Presence of new onset bundle branch block is associated with increased mortality in patients with acute coronary syndrome (ACS). Development of new bundle branch block despite prompt fibrinolytic therapy may signify an extensive and ongoing AMI. It is associated with overall poor prognosis, and high risk for short term mortality.

Presence of complete Bundle Branch Block (BBB) Left or Right in AMI patients represents an independent and very important predictor of in-hospital complication and poor survival on long term.[8]

Earlier studies suggested that patients with BBB have move co-morbid conditions and are less likely to receive therapies such as thrombolytics, aspirin, β blockers and have an increased in hospital mortality rates.[5]

In our study we evaluated the prognostic significance of different types of BBB present during the course of AMI in the hospitalized patients and we followed up the patient at the end of one month.

AIM OF STUDY

1. To estimate the prevalence of BBB in patients with Acute coronary Syndrome.

2. To compare the clinical characteristics in patients with ACS with or without BBB.

3. To assess the prognostic significance of BBB in patients with ACS depending on its form of presentation.

4. Presence of BBB in ACS could be used for risk stratification and selection of treatment according to the risk pattern.

REVIEW OF LITERATURE

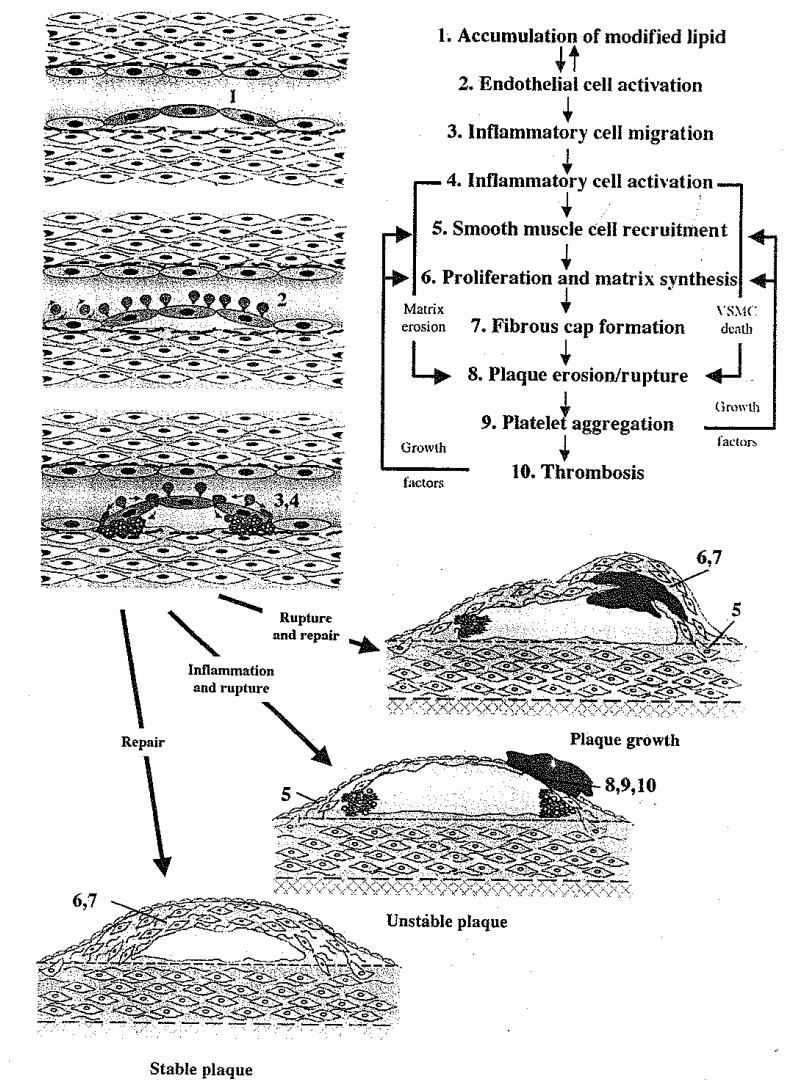
Coronary heart disease, or atherosclerotic CAD is the number one killer disease worldwide.

Most patients with coronary heart disease have some identifiable risk factor.

Table 1 Risk Factors and Interventions for Coronary Heart Disease[2]

Class	Risk Factors	Intervention
1.	Smoking Dyslipidemia High blood Pressure Preventive medications	Smoking cessation Lipid management Blood pressure management Aspirin, angiotensin- converting enzyme inhibitor, beta blocker
2.	Diabetes, prediabetes Physical inactivity overweight, obesity Unhealthy diet, alcohol	Diabetes management Activity management Weight management Improved diet
3.	Menopause, Micronutrients Psychological factors Novel biochemical and genetic markers	Hormone replacement therapy

Fig.1 Development and Progression of atherosclerosis[3]



Acute coronary syndromes comprise the spectrum of unstable cardiac ischemia from unstable angina to acute myocardial infarction. Acute coronary syndromes are now classified based on the presenting ECG as either "ST elevation" or "non-ST elevation".

Acute coronary syndromes without ST segment elevation[1]

- Non-ST-segment elevation acute coronary syndrome is further divided into non-ST-elevation myocardial Infarction (if CK-MB or troponin, or

both, may be elevated) on unstable angina if Cardiac biomarkers are normal.

- The symptoms consist of substernal chest pain or discomfort that may radiate to the jaw, left shoulder or arm. Dyspnea, nausea, diaphoresis or syncope may either accompany the chest discomfort or may be the only symptoms of acute coronary syndrome.

- About 1/3 of the patients with myocardial infarction have no chest pain per se- these patients tend to be older, female, have diabetes, and be at higher risk for subsequent mortality.

- In patients without ST segment elevation it is the presence of abnormal CK-MB or troponin values that are associated with myocyte necrosis and the diagnosis of myocardial infarction.

- Many patients with acute coronary syndrome will exhibit ECG changes during pain - either ST segment elevation, ST segment depression (or) T wave flattening or inversion. Dynamic ST segment shift is the most specific for acute coronary syndrome.

Table Braunwald Clinical Classification of UA/ NSTEMI[2]

Class	Definition	Death or MI to One year * (%)
Severity		
Class I	New onset of severe angina or accelerated angina; no rest pain	7.3
Class II	Angina at rest within past month but not within preceding 48 hr (angina at rest, subacute)	10.3
Class III	Angina at rest within 48 hr (angina at rest, subacute)	10.8†
Clinical Circumstances		
A. (Secondary Angina)	Develops in the presence of extra cardiac condition that intensifies myocardial ischemia	14.1
B. (Primary Angina)	Develops in the absence of extra cardiac condition	8.5
c. (Post Infarction Angina)	Develops within 2 wk after acute myocardial infarction	18.5‡
Intensity of treatment	Patients with unstable angina may also be divided into three groups depending on where unstable angina occurs (1) in the absence of treatment for chronic stable angina (2) during treatment for chronic stable angina. or (3) despite maximal antischemic drug therapy. There three groups that may be designated subscripts 1, 2, and 3, respectively	
ECG Changes	Patients with unstable angina may be further divided into those with or without transient ST -T wave change during pain	

* Date from TIMI III Registry: Scirica BM, et al: Am J Cardiol 90:821-826,2002.
† p = 0.057
‡p = < 0.001
UA/ NSTEMI - unstable angina/ non-ST elevation myocardial infraction.
From Braunwald E: Unstable angina: A Classification, circulation 80:410-A, 1989.

Table Short - Term Risk of Death or Nonfatal Myocardial Ischemia in Patients with Unstable Angina[2]

Feature	High Likelihood (Any of the Following)	Intermediate Likelihood (Absence of High-Likelihood Features and Presence of Any of the following	Low likelihood (Absence of High-or Intermediate - Likelihood Features but May have any of the following
History	• Accelerating tempo of ischemic symptoms in preceding 48 hours	• Prior MI, peripheral or cerebrovascular disease, or CABG; prior aspirin use	
Character of Pain	• Prolonged ongoing (>20 minutes) rest pain	• Prolonged (>20 min) rest angina, now resolved, with moderate or high likelihood of coronary artery disease • Rest angina (<20 min) or relieved with rest or sublingual NTG	• Now onset or progressive Canadian Cardiovascular system class III or IV angina the past 2 weeks without prolonged (>20 min) rest pain but with moderate or high likelihood of coronary artery disease
Clinical findings	• Pulmonary edema, most likely due to ischemia • New or worsening mitral regurgitation murmur • S_3 or new/ worsening rales • Hypotension, bradycardia, tachycardia • Age > 75 years	• Age > 70 years	
Electrocardiogram	• Angina at rest with transient ST segment changes >0.05 mV • Bundle branch block, new or presumed new • Sustained ventricular tachycardias	• T wave inversions >0.2 mV • Pathological Q waves	• Normal or unchanged ECG during an episode of chest discomfort
Cardiac markers	• Elevated (e.g., TnT or Tnl >0.1 ng/ml	• Slightly elevated (e.g., TnT>0.01 but < 0.1 ng/ml)	• Normal

Table American College of Cardiology/ American Heart Association Recommendations for Antischemic Therapy[2]

Class	Indication	Level of Evidence
Class I (Indicated)	Bed rest with continuous ECG monitoring for ischemia and arrhythmia detection in patients with ongoing rest pain	C
	NTG, sublingual tablet or spray, followed by intravenous administration, for the immediate relief of ischemia and associated symptoms	C
	Supplemental oxygen for patients with cyanosis or respiratory distress; finger pulse oximetry or arterial blood gas determination to confirm adequate arterial oxygen saturation (SaO_2, greater than 90%) and continued need for supplemental oxygen in the presence of hypoxemia	C
	Morphine sulfate intravenously when symptoms are not immediately relieved with NTG or when acute pulmonary congestion and/ or severe agitation is present	C
	A beta blocker, with the first dose administered intravenously if there is ongoing chest pain, followed by oral administration in the absence or contraindications	B
	In patients with continuing or frequently recurring ischemia when beta blockers are contraindicated, a nondihydropyridine calcium antagonist (e.g., verapamil or diltiazem) as initial therapy in the absence of severe LV dysfunction or other contraindications.	B
	An ACEI when hypertension persists despite treatment with NTG and a beta blocker in patients with LV systolic dysfunction or CHF and in ACS patients with diabetes.	B
Class IIa (good supportive evidence)	Oral long-acting calcium antagonists for recurrent ischemia in the absence of contraindications and when beta blockers and nitrates are fully used	C
	An ACEI for all post -ACS patients	B
	Intraaortic balloon pump (IABP) counter pulsation for severe ischemia that is continuing or recurs frequently despite intensive medical therapy or for hymodynamic instability in patients before or after coronary angiography	C
Class IIb (weak supportive evidence)	Extended - release form of nondihydropyridine calcium antagonists instead of a beta blocker Immediate - release dihydropyridine calcium antagonists in the presence of a beta blocker	B B
Class III (not indicated)	NTG or other nitrate with 24 hr of sildenafil (Viagra) use Immediate - release dihydropyridine calcium antagonists in the absence of a beta blocker	C A

ACEI = angiotensin -converting enzyme inhibitor; ACS =acute coronary syndrome; CHF = congestive heart failure; ECG - electrocardiographic; LV = left ventricular; NTG = nitroglycerin; SaO_2 = oxygen saturation in arterial blood.

Acute Myocardial Infarction with ST segment elevation[1]

STEMI results in most cases from an occlusive coronary thrombus at the site of preexisting athreosclerotic plaque.

STEMI presents as sudden but not instantaneous development of prolonged (>30 minutes) anterior chest discomfort (sometimes felt as gas or pressure).

Sometimes painless, masquerading as acute CHF, syncope, stroke, or shock.

ECG - ST segment elevation (or) left Bundle Branch Brock.

Immediate reperfusion treatment is warranted.

Primary PCI within 90 minutes to hospital presentation superior to thrombolysis.

Thrombolysis within 30 minutes of hospital presentation and 6-12 hours of onset of symptoms reduces mortality.

Table Hemodynamic subsets in acute myocardial infarction[1]

Category	CI or SWI	PCWP	Treatment	Comment
Normal	>2.2, < 30	<15	None	Mortality rate < 5%.
Hyperdynamic	>3.0, >40	<15	β-Blockers	Characterized by tachycardia; mortality rate < 5%
Hypovolemic	<2.5, <30	<10	Volume expansion	Hypotension, tachycardia, but preserved left ventricular function by echocardiography; mortality rate 4-8%.
Left ventricular failure	<2.2, <30	>15	Diuretics	Mild dyspnea, rales, normal blood pressure; mortality rate 10-20%.
Severe failure	<2.0, <20	>18	Diuretics, Vasodilators	Pulmonary edema, mild hypotension; inotropic agents, IABC may be required; mortality rate 20-40%
Shock	<1.8, <30	>20	Inotropic agents, IABC	IABC early unless rapid reversal occurs; mortality rate>60%

Cl, cardiac index (L/min/m^2); SWI, stroke work index (g-m/m^2, calculated as [mean arterial pressure-PCWP]. stroke volume index. 0.0136); PCWP, pulmonary capillary wedge pressure (in mm Hg; pulmonary artery diastolic pressure may be used instead); IABC, intra-aortic balloon counterpulsation.

TREATMENT OF STEMI[1]

ACC/AHA Guideline recommendations for selected medical treatments.

Medication	Acute Therapies ACS	Acute Therapies AMI	Discharge Therapies
Aspirin (ASA)	IA	IA	IA
Clopidogrel in ASA - allergic patients	IA	IC	IA
Clopidogrel, intended medical management	IA	-	IA
Clopidogrel or IIb/IIIa inhibitor, up front (prior to catheterization)	IA		
Clopidogrel, early catheterization/ percutaneous coronary intervention (catheterization/ percutaneous coronary intervention (cath/ PCI])	IA (prior to or at time of PCI)	IB	IA
Heparin (unfractionated or low - molecular - weight)	IA	IA[2]	-
β-Blockers	IB	IA	IB
Angiotensin - converting enzyme (ACE) inhibitors	IB[3]	IA/IIaB[4]	IA
GP IIb/IIIa inhibitors for intended early cath / PCI			
Eptifibatide/ tirofiban	IA	-	-
Abciximab	IA	IIaB[5]	-
GP IIb/IIIa inhibitors for high-risk patients without intended early cath/ PCI			
Eptifibatide/ tirofiban	IIaA	-	-
Abciximab	IIIA	-	-
Lipid-lowering agent[6]	-	-	IA
Smoking cessation counseling	-	-	IB

1 Class I indicates treatment is useful and effective, IIa indicates weight of evidence is in favor of usefulness/ efficacy, class IIb indicates weight of evidence is less well established, and class III indicates intervention is not useful/effective and may be harmful. Type A recommendations are derived from large-scale randomized trails, and B recommendations are derived from smaller randomized trials or carefully conducted observational analyses. ACC/AHA - American College of Cardiology/ American Heart Association.

2 As a class IIb, low-molecular-weight heparin (best studied is enoxaparin with tenecteplase) can be considered an acceptable alternative to unfractionated heparin for patients less than 75 years old who are receiving fibrinolytic therapy provided significant renal dysfunction is not present.

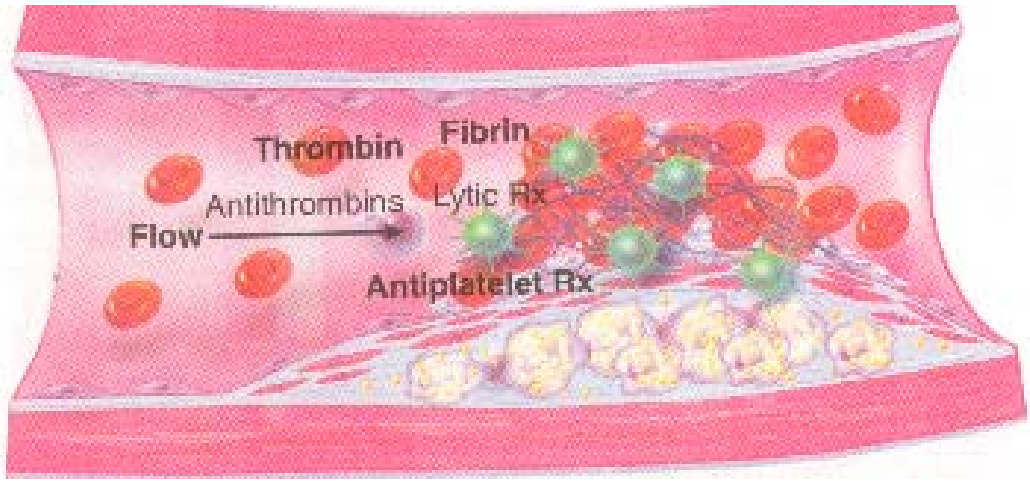
3 for patients with persistent hypertension despire treatment, diabetes mellitus, congestive heart failure, or any left ventricular dysfunction.

4 IA for patients with congestive heart failure or ejection fraction < 0.40, IIa for others, in absence of hypotension (systolic blood pressure < 100 mm Hg); angiotensin receptor blocker (valsartan or candesartan) for patients with ACE inhibitor intolerance.

5 As early as possible before primary PCI.

6 For patients with a low - density lipoprotein cholesterol level> 100 mg/ dL.

Pharmacological dissolution of thrombus in infarct-related artery[2]



This figure shows a schematic view of a longitudinal section of an infarct - related artery at the level of the obstructive thrombus. Following rupture of a vulnerable plaque (bottom center), the coagulation cascade is activated, ultimately leading to the deposition of fibrin strands and platelet aggregates obstructs flow (normally moving from left to right) in the infarct - related artery; this would correspond to TIMI grade 0 on angiography. Pharmacological reperfusion is a multipronged approach consisting of fibrinolytic agents that digest fibrin, antithrombins that prevent the formation of thrombin and inhibit the activity of thrombin that is formed, and antiplatelet therapy. STEMI = ST-elevation myocardial infarction; TIMI = Thrombosis in Myocardial Infarction. (Courtesy of Luke Wells, The Exeter Group).

Reperfusion Therapy[1]

The current recommendation is to treat patients with STEMI who seek medical attention within 12 hours of the onset of symptoms with reperfusion therapy either primary PCI (or) thrombolytic therapy.

Primary Percutaneous Coronary Intervention[1]

Immediate coronary angiography and primary PCI (including stenting) of the infarct - related artery have been shown to be superior to thrombolysis when done by experienced operators in high - volume centers with rapid time from first medial contact to intervention ("door to balloon").

Table Indications for catheterization and percutaneous coronary intervention[1]

Class I	Early invasive strategy for any of the following high-risk indicators
	Recurrent angina/ ischemia at rest or with low-level activity
	Elevated troponin
	ST-segment depression
	Recurrent ischemia with evidence of CHF
	High-risk stress test result
	EF < 0.40
	Hemodynamic instability
	Sustained ventricular tachycardia
	PCI within 6 months
	Prior CABG
	In the absence of these findings, either an early conservative or early invasive strategy

Class IIa	Early invasive strategy for patient with repeated presentations for ACS despite therapy
Class III	Extensive comorbidities in patients win whom benefits of revascularizatin are not likely to outweigh the risks Acute chest pain with low likelihood of ACS
	Acute MI after fibrinolytic therapy (2004 ACC/ AHA AMI Guideline)
Class I	Recurrent ischemia (Spontaneous or provoked) Recurrent MI Cardiogenic shock or hemodynamic instability
Class IIa	LV EF ≤ 0.40, CHF (even transient), serious ventricular arrhythmias
Class IIb	Routine PCI as part of invasive strategy after fibrinolytic therapy

MI, myocardial infarction; CHF, congestive heart failure; EF, ejection fraction, PCI, percutaneous coronary intervention, CABG, coronary artery bypass grafting; ACS, acute coronary syndrome; ACC/ AHA, American College of Cardiology/ American Heart Association, AMI, acute myocardial infarction; LV EF, left ventricular ejection fraction.

Stenting - generally in conjunction with the platelet glycoprotein IIb/IIa antagonist abciximab - is standard for patients with acute myocardial infarction. In the subgroup of patients with cardiogenic shock, early catheterization and percutaneous or surgical revascularization (CABG) are the preferred management and has been shown to reduce mortality.

Thrombolytic Therapy[1]

Table Thrombolytic therapy for acute myocardial infarction

	Streptokinase	Alteplase; Tisseu Plasminogen Activator (t-PA)	Reteplase	Tenecteplase (TNK-t-PA)
Source	Group c streptococcus	Recombinant DNA	Recombinant DNA	Recombinatn DNA
Half-life	20 minutes	5 minutes	15 minutes	20 minutes
Usual dose	1.5 million units	100 mg	20 units	40 mg
Administration	750,000 units over 20 minutes followed by 750,000 units over 40 minutes	Initial bolus of 15 mg, followed by 50 mg infused over the next 30 minutes and 35 mg over the following 60 minutes	10 unit as a bolus over 2 minutes, repeated after 30 minutes	Single eight adjusted bolus, 0.5 mg/ kg
Antticoagulation after infusion	Aspirin, 325 mg daily; there is no evidence that adjunctive heparin improves outcome following streptokinase	Aspirin, 325 mg daily heparin, 5000 units as bolus, followed by 1000 units per hour infusion, subsequently adjusted to maintain PTT 1.5-2 times control	Aspirin,325 mg; heparin as with t-PA	Aspirin, 325 mg daily
Clot selectivity	Low	High	High	High
+	+++	+	+	+
Bleeding	+	+	+	+
Hypotension	+++	+	+	+
Allergic reactions	++	0	0	+
Reocclusion	5-20%	10-30%	-	5-20%

Contraindications to thrombolytic therapy include previous hemorrhagic stroke, other strokes or cerebrovascular events within 1 year, known intracranial neoplasm, active internal bleeding (excluding menstruation), or suspected aortic dissections.

Post thrombolytic management[1]

- Cardiac care unit monitoring
- Asprin, clopidogrel
- Anticoagulation - Unfractionated heparin, Low molecular weight heparin
- Analgesia
- Morphine sulfate 4-8 mg, or meperidine, 50-75 mg
- β-Adrenergic Blocking Agents
- Nitrates
- ACE Inhibitors
- Angiotensin Receptor Blockers
- Aldosterone Antagonists

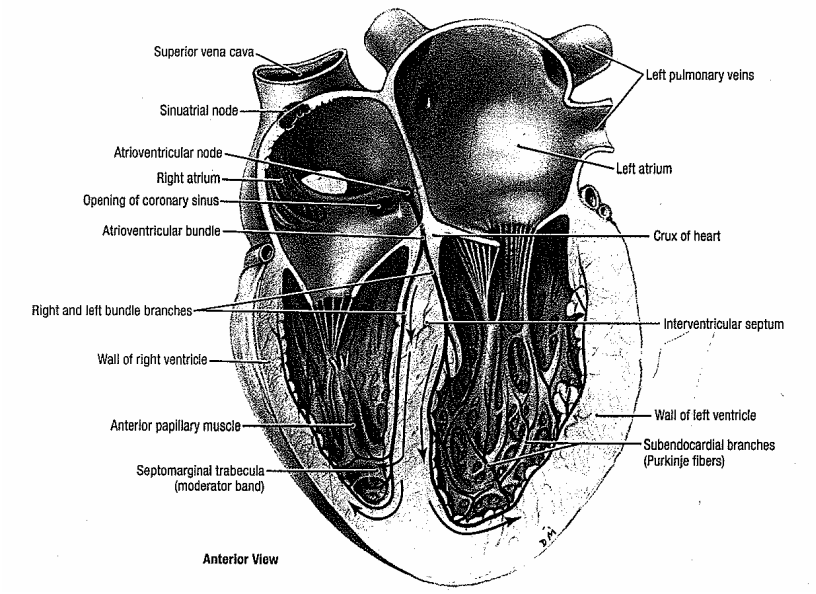
Complications[1]

- Postinfarction Ischemia
- Arrhythmias
- Myocardial Dysfunction
- Mechanical Defects
- LV Aneurysm
- Pericarditis
- Mural Thrombus

Secondary Prevention

1. Life-Style Modification
2. Nitrates
3. Anticoagulants
4. Beta Blockers,ACE Inhibitors
5. Hormone therapy

Conduction System of Heart, Coronal Section[13]



- The sinuatrial (SA) node in the wall of the right atrium near the superior end of the sulcus terminalis extends over the anterior aspect of the opening of the superior vena cava. The SA node is the "pacemaker" of the heart because it initiates muscle contraction and determines the heart rate. It is supplied by the sinuatrial nodal artery, usually a branch of the right coronary artery but it may e a branch of the left.

- Contraction spreads through the atrial wall (myogenic induction) until it reaches the atrioventricular (AV) node in the interatrial septum just superior to the opening of the coronary sinus. The AV node is supplied by the atrioventricular nodal artery, usually arising from the right coronary artery posterior at the inferior margin of the interatrial septum.

- The AV bundle, usually supplied by the right coronary artery, passes from the AV node in the membranous part of the interventricular septum, dividing into right and left bundle branches on either side of the muscular part of the interventricular septum.

- The right bundle branch travels inferiorly in the interventricular septum to the anterior wall of the ventricle, then though the septomarginal trabecula to the anterior wall of the ventricle, then through the septomarginal trabecula to the anterior papillary muscle; excitation spreads throughout the right ventricular wall through a network of subendocardial branches form the right bundle (Purkinje fibers)

- The left bundle branch lies beneath the endocardium on the left side of the interventricular septum and branches to enter the anterior and posterior papillary muscles and the wall of the left ventricle; further branching into a plexus of subendocardial branches (Purkinje fibers) allows the impulses to be conveyed throughout the left ventricular wall. The bundle branches are usually supplied by the left coronary, except the posterior limb of the left bundle branch, which is supplied by both coronary arteries.

- Damage to the cardiac conduction system (often by compromised blood supply as in coronary artery disease) leads to disturbances of muscle contraction. Damage to the AV node results in "heart block" because the atrial excitation wave does not reach the ventricles, which begin to contract independently at their own, slower rate. Damage to one of the branches results in "bundle branch block", in which excitation goes down the unaffected branch to cause systole of that ventricle; the impulse then spreads to the other ventricle, producing later, asynchronous contraction.

Arrhythmias in AMI[2]

A leading hypothesis for a major mechanism of arrhythmias in the acute phase of coronary occlusion is reentry caused by in homogenecity of the electrical characteristics of ischemic myocardium. Thus all forms of bradycardia and tachycardia can depress the cardiac output in patients with STEMI.

The role of arrhythemias in complicating the course of patients with STEMI and the prevention and treatment of these arrhythmias in this setting are discussed here and summarized in Table.

Table Cardiac Arrhythmias and their management during acute myocardial infarction[2]

Category	Arrhythmia	Objective of Treatment	Therapeutic Options
1. Electrical instability	Ventricular premature beats	Correction of electrolyte deficits and increased sympathetic tone	Potassium and magnesium solutions, beta blocker
	Ventricular tachycardia	Prophylaxis against ventricular fibrillation, restoration of hemodynamic stability	Antiarrhythmic agents; cardioversion/ defibrillation
	Accelerated idioventricular rythm	Observation unless hemodynamic function is compromised	Increased sinus rate (atropine, atrial pacing) antiarrhythmic agents
	Nonparoxysmal atrioventricular junction tachycardia	Search for precipitating causes (e.g., digitalis intoxication); suppress arrhythmia only if hemodynamic function its compromised	Atrial overdrive pacing; antiarrhythmic agents; cardioversion relatively contraindicated if digitalis intoxication present
2. Pump failure/ excessive sympathetic stimulation	Sinus tachycardia	Reduce heart rate to diminish myocardial oxygen demands	Antipyretics; analgesics; consider beta blocker unless congestive heart failure present; treat latter if present with anticongestive measures (diuretics, after load reduction)
	Atrial fibrillation and / or/ atrial flutter	Reduce ventricular rate; restore sinus rhythm	Verapamil, digitalis glycosides; anticongestive measures (diuretics, afterload, reduction); vardioversion; rapid atrial pacing (for atrial flutter)
	Paroxysmal supraventricular tachycardia	Reduced ventricular rate, restore sinus rhythm	Vagal maneuvers; verapamil, cardiac glycosides, beta-adrenergic blockers; cardioversion; rapid atrial pacing
3. Bradyarrhythimas and conduction disturbances	Sinus bradycardia	Acceleration of heart rate only if hemodynamic function is compromised	Atropine; atrial pacing
	Junctional escape rhythm	Acceleration of sinus rate only if loss of atrial "kick" causes	Atropine; atrial pacing
	Atrioventricular block and intraventricular block		Insertion of pacemaker

Ischemic injury can produce conduction block at any level of the atrioventicular (AV) or intraventricular conduction system. Such blocks can occur in the AV node and the bundle of his, producing, various grades of AV block, in either main bundle branch, producing right or left bundle branch block. and in the anterior and posterior divisions of the left bundle, producing left anterior or left posterior (fascicular) divisional blocks, Disturbances of conduction can, of course, occur in various combination. Clinical features of proximal and distal AV conduction disturbances in patients with STEMI are summarized in Table.

Table Atrioventricular (AV) Conduction Disturbances in Acute Myocardial Infarction[2]

	Proximal	Distal
Site of block	Intranodal	Infranodal
Site of infarction	Inferoposterior	Anteroseptal
Compromised arterial supply	RCA (90%), LCU (10%)	Septal perforators of LAD
Pathogenesis	Ischemia, necrosis, hydropic cell swelling excess parasympathetic activity	Ischemia, necrosis, hydropic cell swelling
Predominant type features of third-degree AV block	First – degree (PR > 200 msec) Third type I second – degree	Mobitz type II second-degree Third degree
Common premonitory features of third-degree-AV block	(a) First second degree AV block (b) Mobitz I pattern	(a) Intraventricular conduction block (b) Mobitz II pattern
Features of escape rhythm following third-degree block		
(a) Location (b) QRS width (c) Rate (d) Stability of escape rhythm	(a) Proximal conduction system (His bundle) (b) <0.12/sec (c) 45-60 min but may be as low as 30/min (d) Rate usually stable; asystole uncommon	(a) Distal conduction system (bundle branches) (b) 0.12/ sec (c) Often < 30 / min (d) Rate of often unstable with moderate to high risk of ventricular asystole
Duration of high-grave AV block	Usually transient (2-3 days)	Usually transient but some form of AV conduction disturbance and / or intraventricular defect may persist
Associated mortality rate	Low unless associated with hypotension and /or congestive heart failure	High because of extensive infarction associated with power failure or ventricular arrhythmias
Pacemaker therapy (a) Temporary (b) Permanent	(a) Rarely required, may be considered for bradycardia associated with left ventricular power failure, syncope, or angina (b) Almost never indicated because conduction defect is usually transient	a. Should be considered in patient with anteroseptal infarction and acute bifascicular b lock. b. Indicated for patients with high-grade AV block in His-Purkinje system and those with transient advanced AV block and associated bundle branch block

* Some studies suggest that a wide QRS escape rhythm (>0.12 sec) following high-grade AV block in inferiori infarction is associated with a worse prognosis.
LAD – left anterior descending coronary artery; LCX- left circumflex coronary artery; RCA – right coronary artery.
Modified from Antma EM. Rutherford JD: Coronary Care Medicine: A Practical Approach. Boston, Martinus Nijhoff, 1986; and Dreifus LS, Fisch, C, Griffin JC, et al. guidelines for implantation of cardiac pacemakers and antiarrhythmia devices. J Am. Coll Cordial. 18:1, 11991. Reprinted with permission from the American College of Cardiology.

Intraventricular Block[2]

The right bundle branch and the left posterior division have a dual blood supply from the left anterior descending and right coronary arteries, whereas the left anterior division is supplied by septal perforators originating from the left anterior descending coronary artery. Not all conduction blocks observed in patients with STEMI can be considered to be complications of infarcts because almost half are already present at the time the first ECG is recorded, and they may represent antecedent disease of the conduction system. Compared with patients without conduction defects, STEMI patients with bundle branch blocks have more comorbid conditions; are less likely to receive therapies such as thrombolytics, aspirin, and beta blockers; and have an increased in-hospital mortality rate. In the prefibrinolytic 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 bundled) had been reported to occur in 5 to 10 per cent of patient with STEMI. More recent series in the fibrinolytic era suggest that intraventricular blocks occur in about 2 to 5 per cent of patients with MI. (Investigators performing primary PCI for STEMI have reported an association between new-onset bundle branch block and abnormal myocardial perfusion even if epicardial flow is restored.

ISOLATED FASCICULAR BLOCKS. Isolated left anterior divisional block is unlikely to progress to complete AV block. Mortality is increased in these patients, although not as much as in patients with other forms of conduction block. The posterior fascicle is larger than the anterior fascicle,

and, in general, a larger infarct is required to block it. As a consequence, mortality is markedly increased. Complete AV block is not a frequent complication of either form of isolated divisional block.

RIGHT BUNDLE BRANCH BLOCK. This conduction defect alone can lead to AV block because it is often a new lesion, associated with anteroseptal infarction. Isolated right bundle branch block is associated with an increased mortality risk in patients with anterior STEMI even if complete AV block does not occur, but this appears to be the case only if its is accompanied by congestive heart failure.

BIFASCICULAR BLOCK. The combination of right bundle branch block with either left anterior or posterior divisional block or the combination of left anterior and posterior divisional blocks (i.e., left bundle branch block)C is known as bidivisional or bifascicular block. If new block occurs in two of the three divisions of the conduction system, the risk of developing complete AV block is quite high. Mortality is also high because of the occurrence of severe pump failure secondary to the extensive myocardial necrosis required to produce such an extensive intraventricular block. Patients with intraventricular fibrillation late in their hospital stay. However, the high rate of mortality in these patients occurs even in the absence of high-grade AV block and appears to be related to cardiac failure and massive infarction rather than to the conduction disturbance.

Preexisting bundle branch block or divisional block is less often associated with the development of complete heart block in patients with STEMI than are conduction defects acquired during the course of the infarct.

Bidivisional block in the presence of prolongation of the P-R interval (first-degree AV block) may indicate disease of the third subdivision rather than of the AV node and is associated with a greater risk of complete heart block than if first-degree AV block is absent.

Complete bundle branch block (either left or right), the combination of right bundle branch block and left anterior divisional (fascicular) block, and nay of the various forms of trifascicular block are all more often associated with anterior than with inferoposterior infarction. All these forms are more frequent with large infarcts and in older patients and have a higher incidence of other accompanying arrhythmias than is seen in patients without bundle branch block.

TEMPORARY PACING[2]. Just as is the case for complete AV block, transvenous ventricular pacing has not resulted in statistically demonstrable improvement in prognosis among patients with STEMI who develop intraventricular conductions defects. However, temporary pacing is advisable in some of these patients because of the high risk of developing complete AV block. This includes patients with new bilateral (bifascicular) bundle branch block (i.e., right bundle branch block with left anterior or posterior divisional block and alternating right and left bundle branch block); first-degree AV block adds to this risk. Isolated new block in only one of the three fascicles even with P-R prolongation and preexisting bifascicular block and normal P-R interval poses somewhat less risk; these patients should be monitored closely, with insertion of a temporary pacemaker deferred unless higher degree AV block occurs.

Noninvasive external temporary cardiac pacing is possible routinely in conscious patients and is acceptable to many but not all patients despite the discomfort. Used in a standby mode, it is virtually free of complications and contraindications and provides an important alternative to transvenous endocardial pacing. Once it is clinically evident that continuous pacing is required, external pacing, which is generally not well tolerated for more than minutes to hours, should be replaced by a temporary transvenous pacemaker.

ASYSTOLE. The presence of apparent ventricular asystole on monitor displays of continuously recorded ECGs may be misleading because the rhythm may actually be fine ventricular fibrillation. Because of the predominance of ventricular fibrillation as the cause of cardiac arrest in this setting, initial therapy should include electrical counter-shock, even if definitive electrocardiographic documentation of this arrhythmia is not available. In the rare instance in which asystole can be documented to be the responsible electrophysiological disturbance, immediate transcutaneous pacing (or stimulation with a transvenous pacemaker if one is already in place) is indicated.[1]

PERMANENT PACING. The question of the advisability of permanent pacemaker insertion is complicated because not all sudden deaths in STEMI patients with conduction defects are caused by high-grade AV block. A high incidence of late ventricular fibrillation occurs in CCU survivors with anterior STEMI complicated by either right or left bundle branch block. Therefore ventricular fibrillation rather than asystole caused by failure of AV

conduction and infranodal pacemakers could be responsible for late sudden death.

Long-term pacing is often helpful when complete heart block persists throughout the hospital phase in a patient with STEMI, when sinus node function is markedly impaired, or when type II second- or third-degree block occurs intermittently.[1] When high-grade AV block is associated with newly acquired bundle branch block or other criteria of impairment of conduction system function, prophylactic long-term pacing may be justified as well. Additional considerations that drive a decision to insert a permanent pacemaker include whether the patient is a candidate for an implantable cardioverter-defibrillator or has severe heart failure that might be improved with biventricular pacing.

1. Wong C.K. et al[5].

Insights form the Hirulog and Early Reperfusion or Occlusion (HERO)-2 trial. (European Heart Journal doi:10.1093eurheartj/ ehi622).

Patients with any type of BBB at randomization had worse baseline characteristics than those with normal intraventricular conduction. However, only patients with RBBB accompanying anterior AMI at randomization (and not patients with RBBB accompanying inferior AMI or patients with LBBB at randomization) had a higher mortality rate after adjustment for baseline characteristics. This finding persisted after further adjustment for the presenting features of AMI

Approximately 1% of patients who had ST-elevation AMI with normal intraventricular conduction at randomization developed new BBB (most commonly RBBB accompanying anterior AMI) within 60 min after commencing fibrinolytic therapy. New BBB was associated with higher 30-day mortality.

The higher mortality and the higher incidence of RBBB seen in patients with anterior AMI may be explained by septal ischaemia from a more proximal left anterior descending artery occlusion (before the large septal branch) and the course of the right bundle branch traversing the septum towards the apex. Higher peak enzyme levels were observed in these patients. In contrast, the left bundle has a more varied distribution from a true bifasclcular system to a network of fibres, and more extensive ischemia or necrosis is required to produce complete LBBB. Thus, new LBBB was far less likely to develop within 60 min than new RBBB, but when new LBBB did develop, the mortality rate was as high as that of patients with RBBB accompanying anterior AMI.

In the current American and European guidelines, new or presumed-new LBBB within 12 hours after the onset of symptoms suggestive of AMI is a Class I indication for fibrinolytic therapy. New or presumed-new LBBB was an inclusion criterion in the HERO-2 trial. The 300 HERO-2 patients with LBBB at randomization had worse pre-infarction characteristics (older age and previous AMI), worse presenting features (higher pulse rate and Killip class), and nearly twice the unadjusted 30-day mortality rate of patients with normal conduction. However, after adjustment for pre-infarction characteristics and presenting

features, their 30-day mortality rate was no higher than that of patients with normal intraventricular conduction[12]-an interesting observation which corraborates the findings of a GUSTO-1 substudy in 131 patients with LBBBJ.

2. Taporan Daniela et al. Study[8] says,

Patients with BBB and AMI are less likely to receive thrombolytic therapy, associate more frequently severe heart failure and have an increased risk for in-hospital death. No clinically significant differences in the development of recurrent ischemia, angina or mechanical complications were seen between patients with BBB and without BBB.

3. Acute Med Dkayama 2009 Feb 63(1):25-33 et al Study[14] Says

New permanent RBBB was a strong independent predictor for an adverse short term prognosis in patients with inferior MI as well as in patients with anterior MI. New permanent RBBB during inferior MI is a strong independent predictor for increased in hospital mortality, regardless of the infarction location.

4. Vojnosaint Preg et al., Study[15] 2009 Jan 66(1) : 74 says

The patients with ABBB in AMI are at risk group of patients that commonly exhibit both early and remote complications accompanies by high mortality . That is the reason why this sub-group of AMI patients should receive an urgent diagnostics followed by aggressive therapeutic treatments.

5. Barsheshet *et al* (Am. J. Cardiol: 2008 Aug 15:102(4) 507-8.)[16]

Study says RBBB rather than LBBB is an independent predictor of mortality in hospitalized patients with systolic heart failure. This prognostic marker could be used for risk stratification and selection of treatment.

6. Lerecouverex et al Arch Mal Coeur Vaies 2005 Dec 98(12) 1231-8 study says, RBBB is associated with worse prognosis.

7. Biagini et al[17] (J Am Coll Cardiology 2005 Sep 6: 46(5) 856-63) Study says

LAHB in AMI increased risk of death. Isolated LAHB should not be considered a benign electrocardiography abnormality in these patients.

8. Islam MN et al[18] Says (Bangladesh Med Res Counc Bul 2002 Apl 28(1) 26-35).

Specific in hospital complication were significantly higher in patients with RBBB than without RBBB.

9. Vrugada et al[19] (Circulation 2002 Jan 1:105 (1): 73-8) study Says

An ECG showing RBBB with STEMI in the right pericardial leads is a marker of malignant ventricular arrhythmias and sudden death. Recurrence of Malignant arrhythmias is high after the occurrence of symptoms.

10. Melgarejo Moreso. A et al[10]., (Chin Cardiol 2001 May:24 (5): 37-6) Study say

In the patients with AMI, the classification of BBB according not only to location but also to time of appearance is of practical interest. New BBB is an independent predictor of short and long term mortality.

11. Sergia Rocha et al[9]., (ESC congress 2007), Austria Study says.

RBBB in AMI - is associated with poor prognosis.

12. Gunnarson.G et al., Scand Cardiovasc Dec 2000 34(6) 875-9.

New onset BBB associated with poor programs.

13. EB Sgarbossa et al[12] J Am Coll Cardiol 1998: 31:105-12 study says

Bundle Branch block at hospital admission inpatient in patients with acute AMI predicts in hospital complications and poor shortterm survival.

14. Eugene Brancwold[2] 8th Edition page 1281

Bifasicular blocks ACS, is associated with risk of developing complete AV block is quiet high. Mortality is also high because of the occurrence of severe pump failure secondary to the extensive myocardial necrosis required to produce such an extensive intraventricular block.

Regarding prognosis significance of RBBB (Page 1281)

Patients with Intraventricular conduction defects particularly right bundle branch block account for the majority of patients who develop ventricular fibrillation late in their hospital stay. However the high rate of mortality in these patients occurs even in the absence of high grade AV block and appears to be related to cardiac failure and massive infarction rather than to the conduction disturbance.

MATERIALS AND METHODS

Study Design

This study is a single centre prospective analytical study carried out in the coronary care unit, Department of Cardiology, Madras Medial College during the period of January 2009 to December 2009.

Total number of patients were 150. All the patients in the study were hospitalized. No out patients were included. A detailed informed concent was obtained from the patients Our Institutional ethical committee clearance was obtained. Standard approved protocol were used for treating all the patients. The results were tabulated and analysed using chi-square test.

Inclusion and Exclusion Criteria

Patients presented with ACS in the coronary care unit were included and observed. Serial ECGs of all the patients admitted with ACS in CCU were studied. CK-MB was measured in some of the cases. Since facilities for measuring. Troponin was not available we were not able to measure it.

Patients demographics, clinical variables like prior MI, angina, CHF, Cardiac risk factors like DM, SHT, smoking dylipidemia chest pain on admission, Killip class, use of thrombolytic therapy, reasons for not using thrombolytic therapy were recorded.

Patients were followed until discharge from the hospital and at the end of one month. During the period of follow up, events like ventricular

dysfunction, arrhythmias, recurrent angina, CHF, 2°, 3° heart block, mechanical complications, cardiac arrest and death were recorded. These variables are compared between ACS patients with BBB and without BBB.

Patients were excluded if presented with

1. Pre existing BBB
2. Non Specific Intraventricular conduction defects.

Study Protocol

Preliminary history with detail questioning regarding.

- Time of onset of chest pain, time of admission.
- Patients clinical status on admission including BP, HR, Killip class were recorded.
- Serial ECGs were studied to record the AMI location such as AWMI, IWMI others PWMI, RVMI.
- Presence of new onset of BBB, arrythmias
- History of thrombolysis and treatment of complication during the course in the hospital.
- Previous history of MI, angina, CHF.
- Presence of cardiovascular risk factors like DM, HT, Smoking Dyslipidemia.

Definitions[4]

RBBB

ECG Changes in completed and Incomplete RBBB

- A tall R wave (rSR') or notched tall R wave (rR') in lead V_1 and V_2 and a deep, wide and prominent S wave in leads V_5, V_6 and standard leads 1 and a VL is seen in complete right bundle branch block. In incomplete bundle branch block, a wider rSr, or rSR' is common in V_1 - V_2.

- QRS (rSR') duration is > 0.12 sec. in complete RBBB but it remains in between 0.10 to 0.12 sec. in incomplete RBBB.

- VAT is prolonged in V_1 - V_2 (>0.03 sec.) in complete RBBB but is normal in complete RBBB.

- ST segment is slightly depressed and T is inverted in V_1 and V_2, while ST is slightly elevated with concavity upwards in leads V_5 - V_6 and T is upright in these leads. These changes are seen both in complete and incomplete RBBB.

LBBB

- ECG changes in complete and incomplete LBBB. Precordial leads V1 and V5 are best for diagnosis of LBBB.

- The leads V_5, V_6 and standard lead 1 show wide slurred R wave or RsR' to RR' complex with absent q wave. The initial small r wave in V_1

disappears. There is a slurring of S wave (if seen) in V_5-V_6 due to activation of posterobasal region.

- QRS interval is > 0.12 second resulting in QS pattern in V_1-V_2. In incomplete bundle branch block, QRS remains between 0.10 to 0.12 sec.

- VAT is > 0.09 second in complete LBBB. It is less increased in incomplete LBBB.

- ST segment depression with convexity upwards and T wave inversion occurs in leads I and V_5-V_6, while on the other hand, leads V_1-V_2 may show mild elevation of ST segment with concavity upwards and T is upright in these leads. This change is seen in both complete and incomplete LBBB.

- Horizontal heart position will reveal similar morphology of QRS in leads, I, aVL and V_5-V_6. Vertical heart position will show similar morphologies of QRS in leads II, aVF and V_5-V_6.

- In LBBB (complete or incomplete), there is always an absence of q wave in leads, I, V_5-V_6 due to septal activation from right to left. The presence of q wave in these leads suggests an associated myocardial infarction.

LAHB

a. Left anterior fascicular or hemiblock (LAH)

- Mean left QRS axis deviation is more than -30°.

- A rS complex in leads II, III and aVF. The S wave is deeper in lead III than lead II.

- A qR complex is present in leads I and aVL.

- A slight increase in QRS duration in these leads but less than 0.11 sec.

- VAT is slightly prolonged.

- ST segment depression and T wave inversion in leads registering positive QRS deflection. T wave is upright with slight elevation of ST segment in leads registering negative QRS deflection,

- Precordial leads will have RS complex with slurring of S wave in V_5-V_6.

B. Left posterior fascicular or left posterior hemiblock (LPH)

- Mean right QRS axis deviation (+120° clockwise to + 180°)

- Prominent S waves in leads I and aVL (rs complex), a tall R waves in leads II, III and aVF (qR or qRs complex); the R wave will be thaller in lead III than II and may be slurred.

- Procordial leads show rS complex in V_1 and V_2 and Rs in V_5 and V_6 with transition zone in V_3 through V_4.

- The ST segment depression and T wave inversion appears as a secondary change in leads registering positive QRS deflection. T is upright in leads with negative QRS deflection.

Bifascicular or Bilateral Bundle Branch Block

a. Right bundle branch block (RBBB) with left anterior fascicular or hemiblock (LAH)

- Pattern of right bundle branch block (rSR' or rR' or slurred R) in V_1 and V_2 with wide S in leads I, V_5, V_6. This is due to terminal QRS orientation to the right and interiorly.

- Left axis deviation is > - 30°. This is due to initial QRS vector being oriented superiorly and to the left on frontal plane.

B. Right bundle branch block with left posterior fasciular block

- Typical features of RBBB as discussed above

- Right axis deviation > + 120°.

C. Trifascicular block

1. Right bundle branch block, left anteriro fascicular block and AV conduction delay (commonest type).

   - Pattern of RBBB as described above.

   - Left axis deviation > - 30°.

   - Prolonged P-R interval > 0.20 sec.

2. Right bundle branch block, left posterior fascicular block and AV conduction delay (uncommon type).

- Pattern of RBBB as described above.

- Right axis deviation > + 120°.

- Prolonged PR interval > 0.20 sec.

3. Right bundle branch block plus alternating left anterior and left posterior fascicular block.

- Pattern of RBBB as described. It is fixed block.

- Sometimes, left anterior fascicular block and, left posterior fascicular block alternates with RBBB.

- Follow up

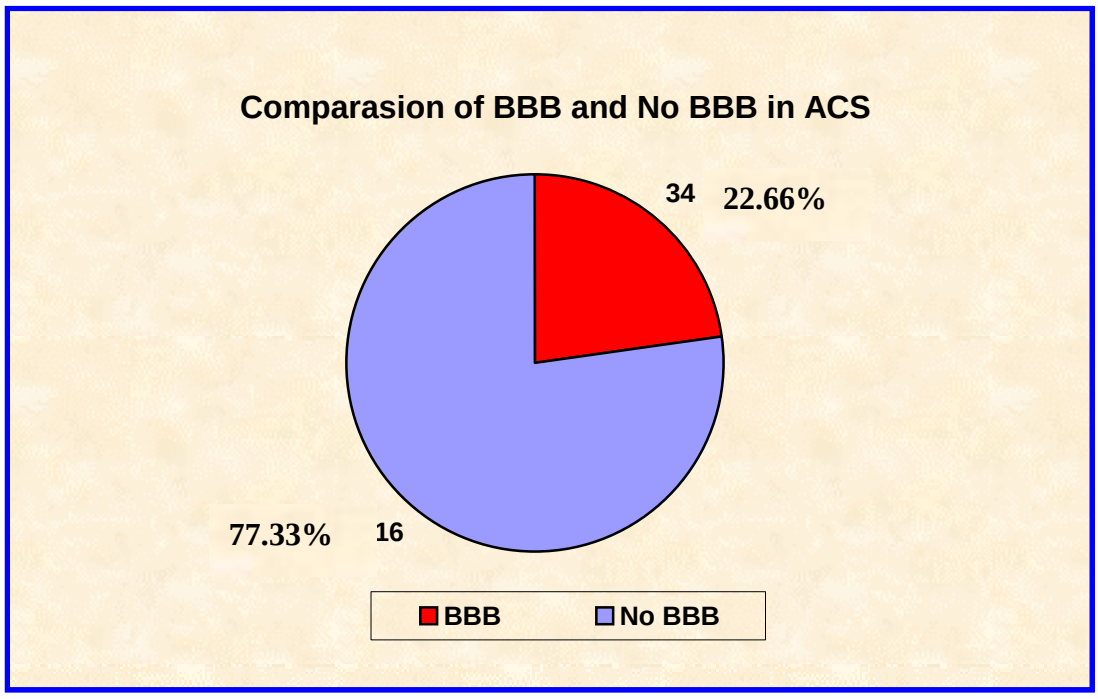
All the patients presented will new onset BBB were followed up till discharge from hospital and for 30 days after the onset of AMI. During this period complication like ventricular dysfunction, arrhythmias, recurrent angina. CHF, Heart block, Mechanical complications like VSR, cardiac arrest, and death were recorded.

Standard guidelines for the treatment of ACS by American heart Association were followed. The treatment modified according to the complications during the hospital stay.

OBSERVATIONS

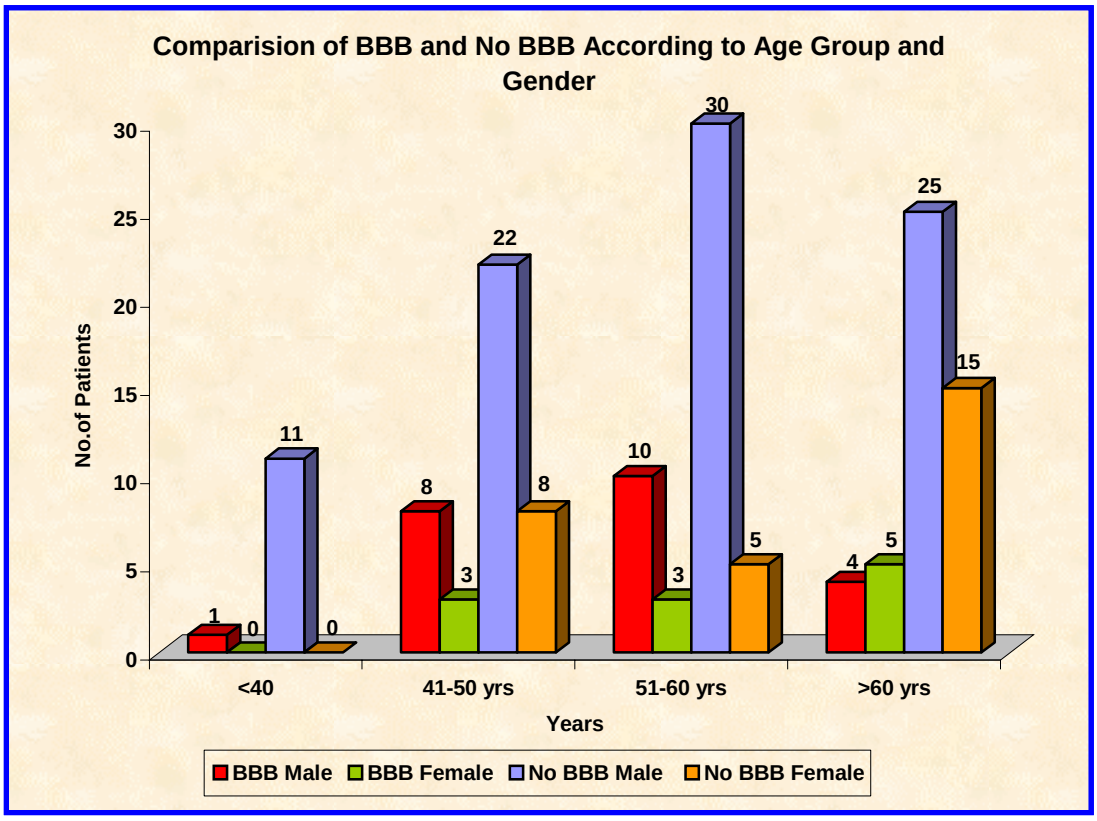
Gender and BBB

Total		BBB		No BBB		P. Value
Total	150	Total	34	Total	116	0.337
Male	111	Male	21	Male	88	
Female	39	Female	11	Female	28	



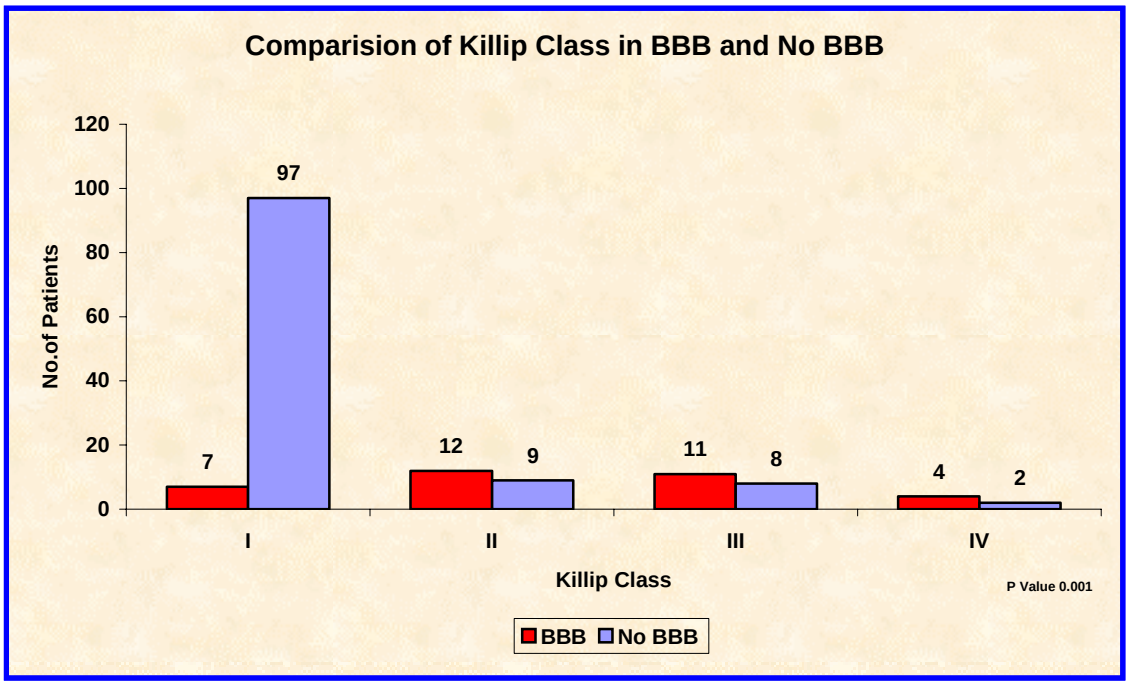
Age and BBB

AGE	Total		BBB		No BBB		P Value
	Male	Female	Male	Female	Male	Female	
<40 yrs	12	0	1	0	11	0	0.413
41-50 yrs	30	11	8	3	22	8	
51-60 yrs	40	8	10	3	30	5	
>60 yrs	29	20	4	5	25	15	



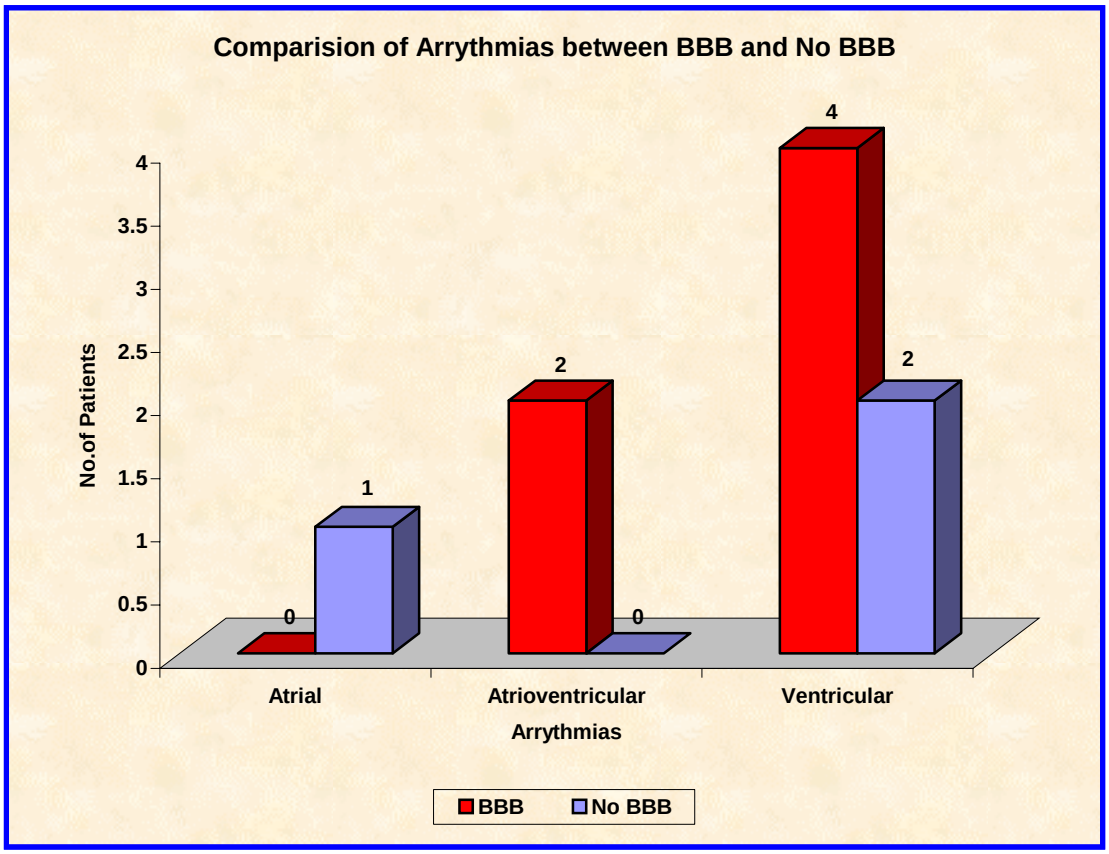
Killip class and BBB

Total	BBB (n=34)	No BBB (n=116)	P Value
I – 104	I – 7	I – 97	0.001 **
II – 21	II – 12	II – 9	
III – 19	III – 11	III – 8	
IV – 6	IV – 4	IV – 2	



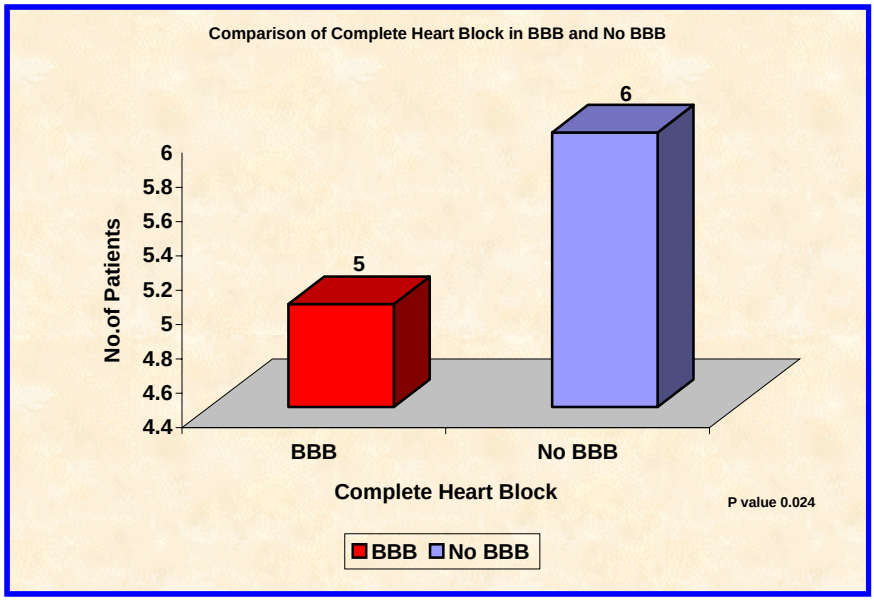
Arrythmias and BBB

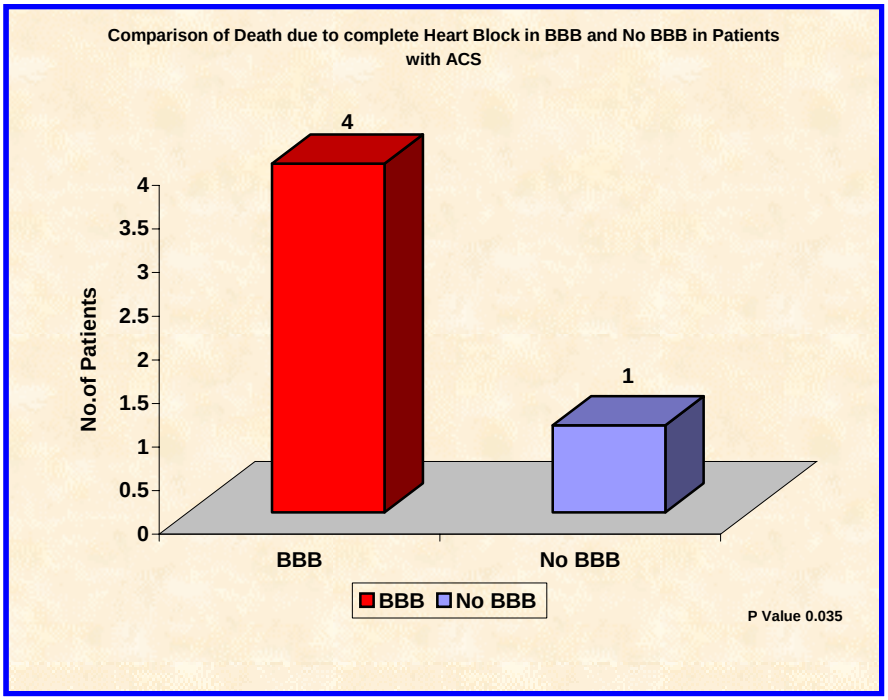
	Total		BBB		No BBB		P Value
	Brady arrhythmias	Taccy arrhythmias	Brady arrhythmias	Taccy arrhythmias	Brady arrhythmias	Taccy arrhythmias	
Atrial	–	1	–	–	–	1	0.008 **
Atrioventicular	2	-	2	–	–	–	
Ventricular	2	4	2	2	–	2	



Complete Heart Block and BBB

Total	BBB (n=34)	No BBB (n=116)	P Value
Total – 11	Total – 5	Total – 6	
AWMI – 3	AWMI – 2	AWMI – 1	0.024 *
IWMI & Others – 8	IWMI & Others – 3	IWMI & Others – 5	
Death – 5	Death – 4	Death – 1	0.035 *



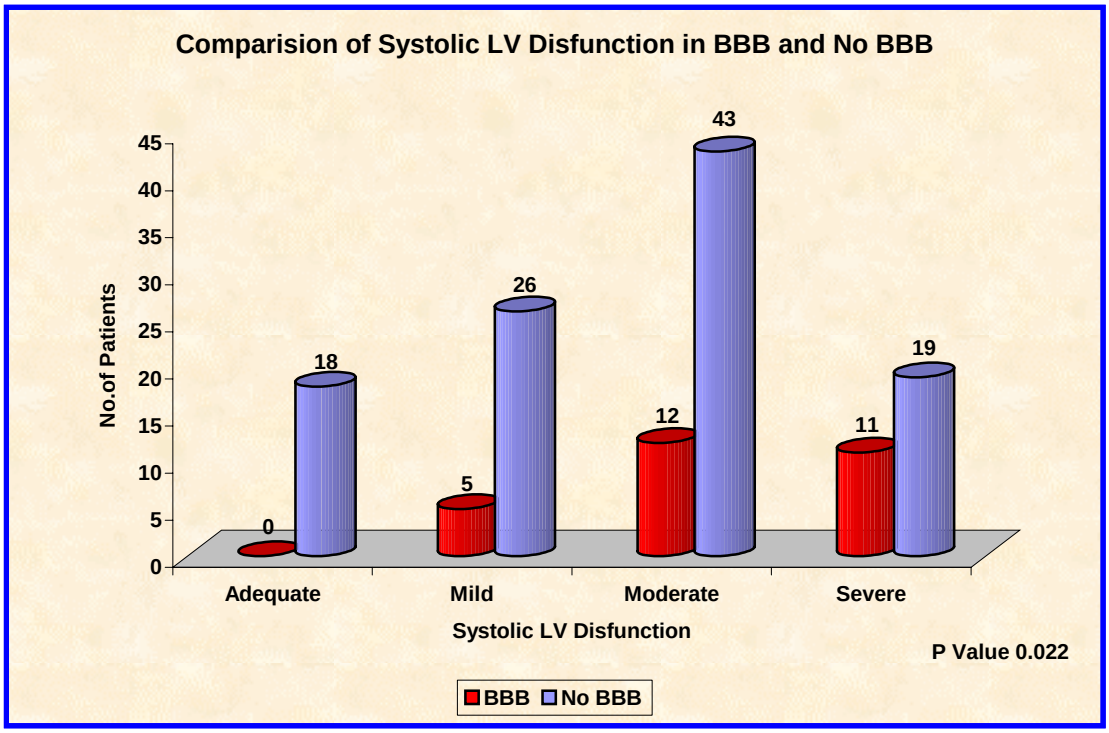


MI location and BBB

Total	BBB (n=34)	No BBB (n=116)	P Value
AWMI – 74	AWMI – 20	AWMI – 54	
IWMI & Others – 69	IWMI & Others – 13	IWMI & Others – 55	0.208
UA – 7	Both – 1	UA – 7	

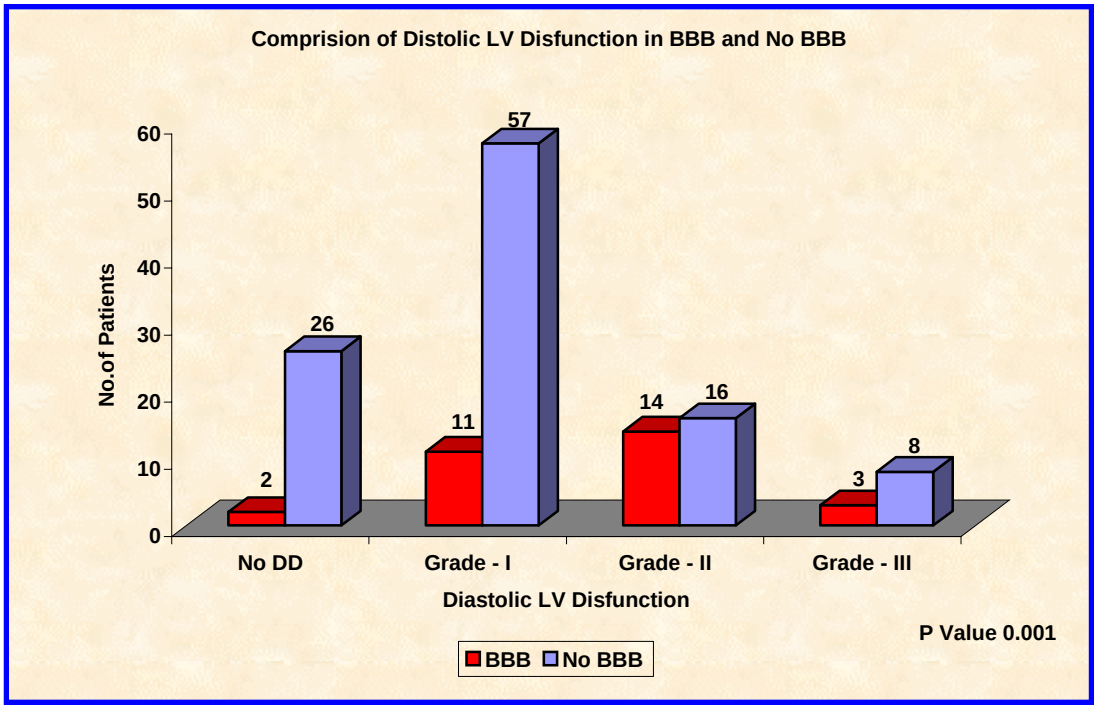
Comparison of Systolic LV dysfunction between BBB and No BBB

Total	BBB	No BBB	P Value
Adequate – 20	Adequate – 0	Adequate – 18	
Mild – 31	Mild – 5	Mild – 26	
Moderate – 55	Moderate – 12	Moderate – 43	0.022 *
Severe – 30	Severe – 11	Severe – 19	
	Death – 6	Death – 10	



Diastolic Dysfunction and BBB

Total	BBB (n=34)	No BBB (n=116)	P Value
No DD – 27	No DD – 2	No DD – 26	0.001 **
Grade - I – 68	Grade - I – 11	Grade - I – 57	
Grade - II – 30	Grade - II – 14	Grade - II – 16	
Grade - III – 11	Grade - III – 3	Grade - III – 8	



Recurrent Angina

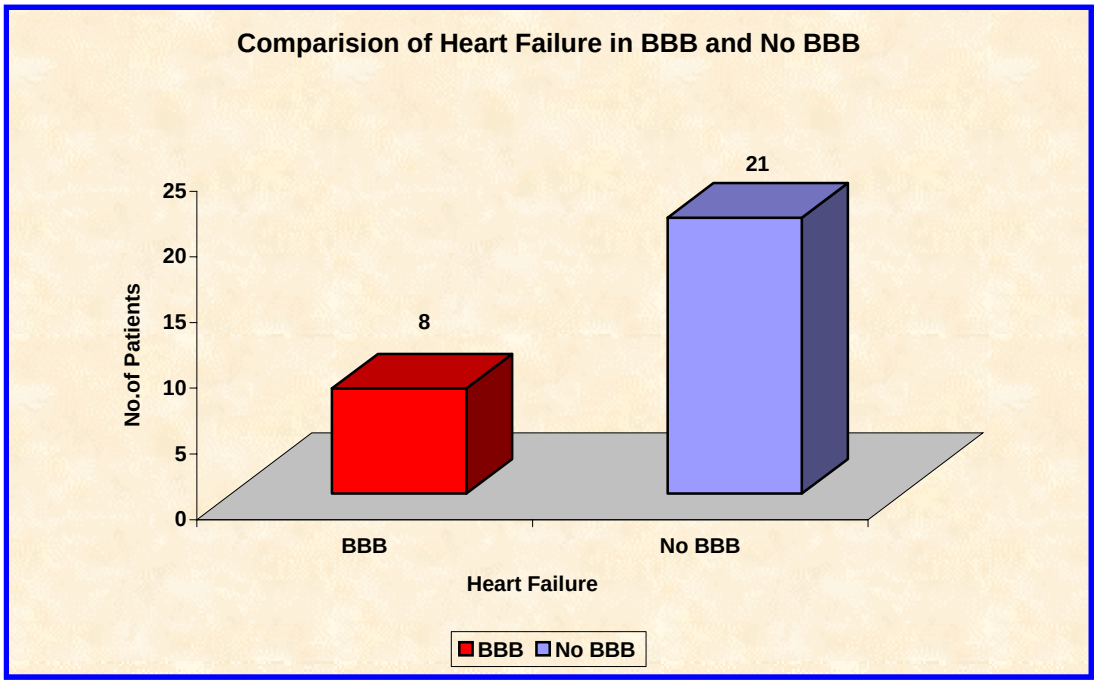
Total	BBB (n=34)	No BBB (n=116)	P Value
Total – 21	Total – 5	Total – 16	0.52
AWMI – 11	AWMI – 2	AWMI – 9	
IWMI – 10	IWMI – 3	IWMI – 7	

Thrombolysis

Total	BBB	No BBB	P Value
Total – 93	Yes – 22	Total – 116	< 0.001 **
0-3 hrs – 32	0 - 3 hrs – 5	0-3 hrs – 27	
3-6 hrs – 46	3 - 6 hrs – 11	3-6 hrs – 35	
>6 hrs – 15	>6 hrs – 11	>6 hrs – 35	
Not Thrombolysed-50	Not Thrombolised- 7	Not Thrombolysed-43	
UA – 7		UA – 7	

Heart Failure

Total	BBB	No BBB	P Value
Total – 27	Total – 8	Total – 21	0.004 **

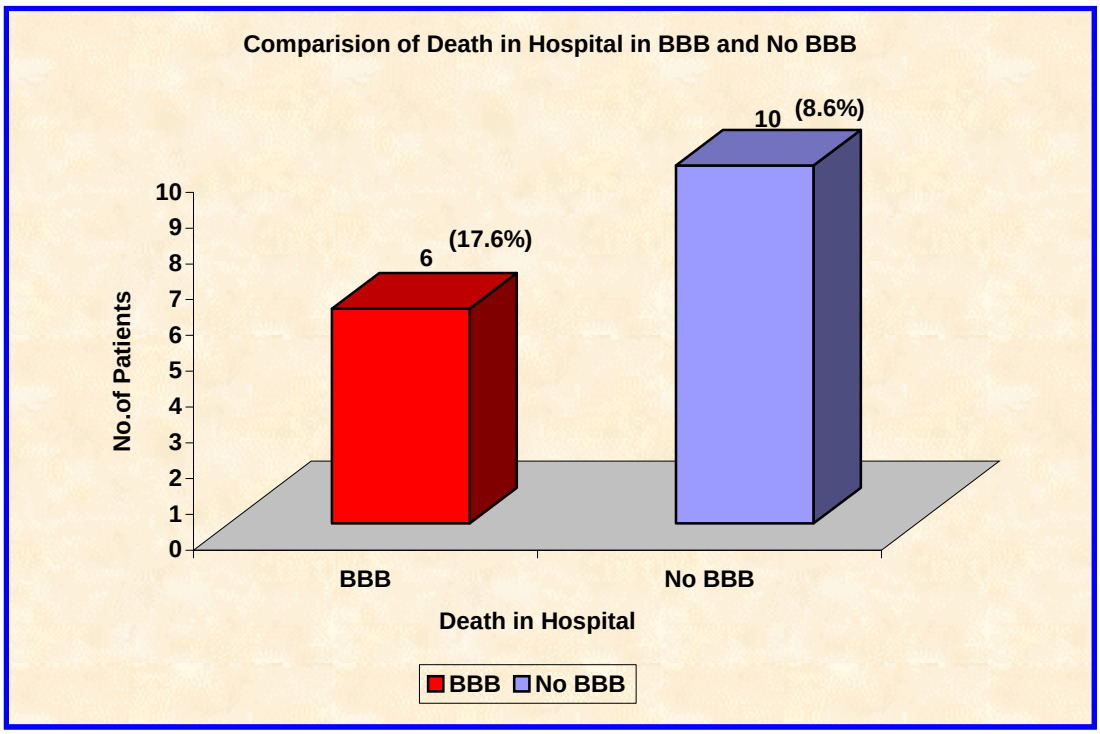


Cardiac Arrest

Total	BBB	No BBB
Total – 17	Total – 7 Revived – 1	Total – 10

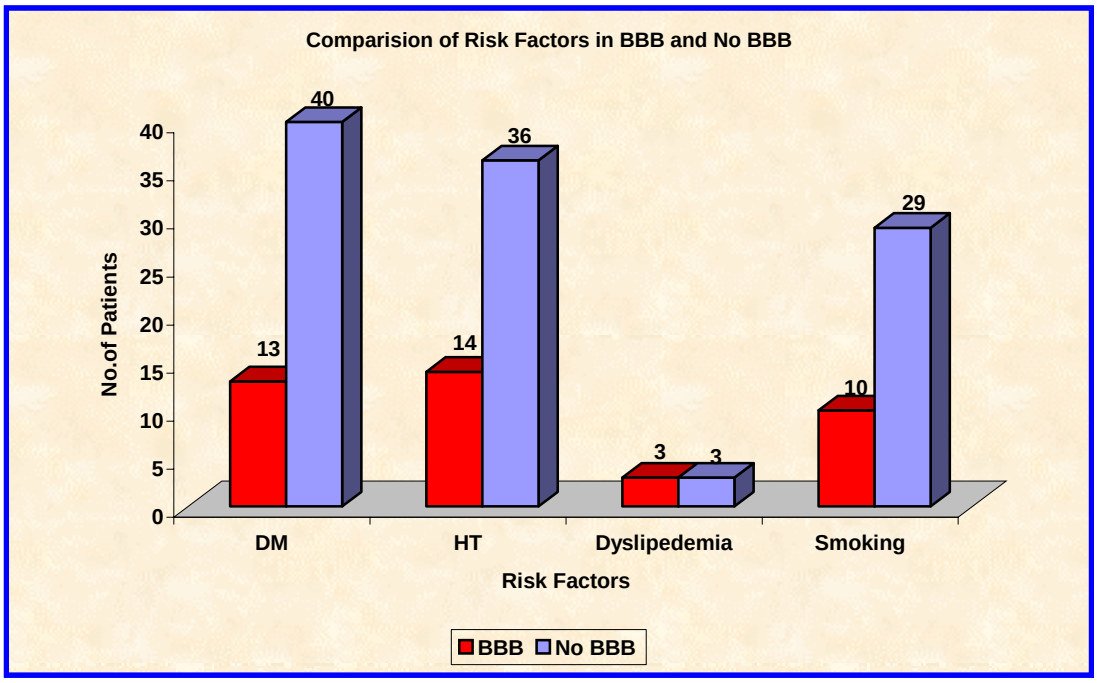
Death in Hospital

Total	BBB	No BBB	P Value
Total – 16	Total – 6 (17.6%)	Total – 10 (8.6%)	0.134



Risk Factors

Total	BBB	No BBB	P Value
DM – 53	DM – 13	DM – 40	0.607
HT – 50	HT – 14	HT – 36	
Dyslipidemia – 6	Dyslipidemia – 3	Dyslipidemia – 3	
Smoking – 39	Smoking – 10	Smoking – 29	

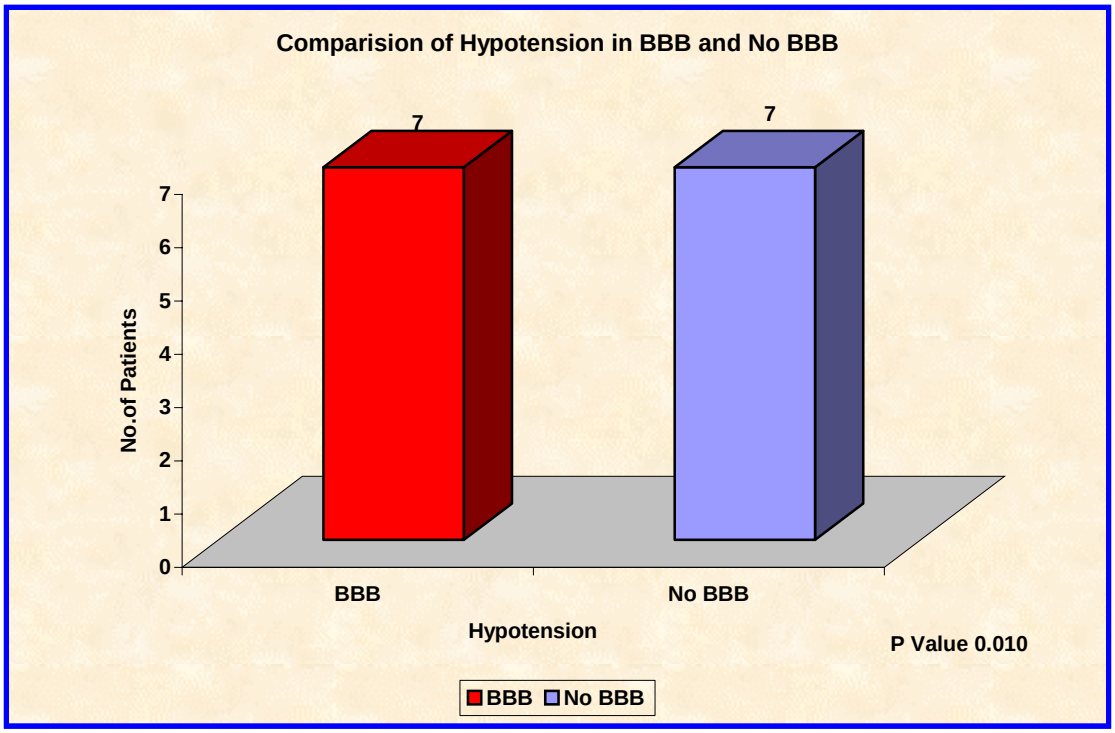


History of Previous MI

Total	BBB (n=34)	No BBB (n=116)	P Value
H/o. Previous MI- 18	H/o. Previous MI- 5	H/o. Previous MI- 13	0.589

BP

Total	BBB (n=34)	No BBB (n=116)	P Value
Hypotension – 10	Hypotension – 7	Hypotension – 7	0.010 **
Hypertension – 31 Normal BP – 109	Hypertension – 6 Normal BP – 25	Hypertension – 25 Normal BP – 84	0.620



Tachypnoea

Total	BBB	No BBB	P Value
Total – 21	Total – 7	Total – 14	0.126

History of Surgery

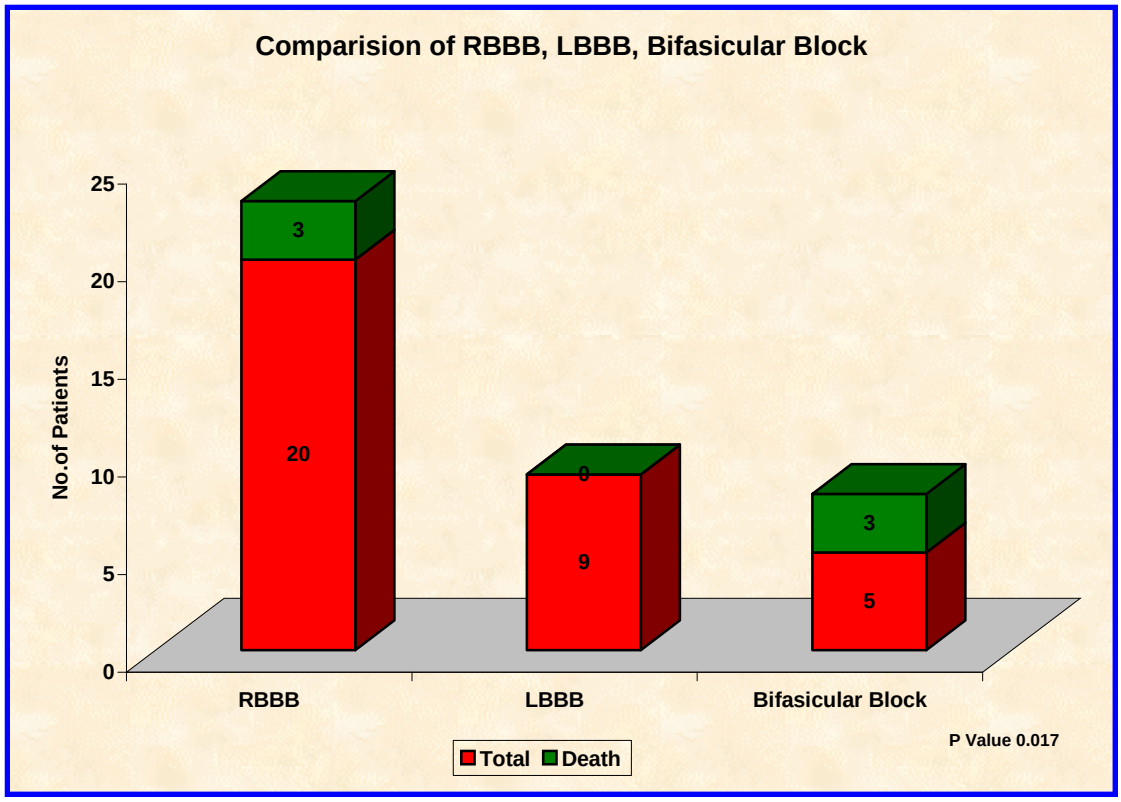
Total	BBB	No BBB
PTCA with stent – 1 CABG – 2	PTCA with stent– 0 CABG – 0	PTCA with stent– 1 CABG – 2

Mechanical Complications

Total	BBB	No BBB
Total VSR – 1	Total VSR – 0	Total VSR – 1

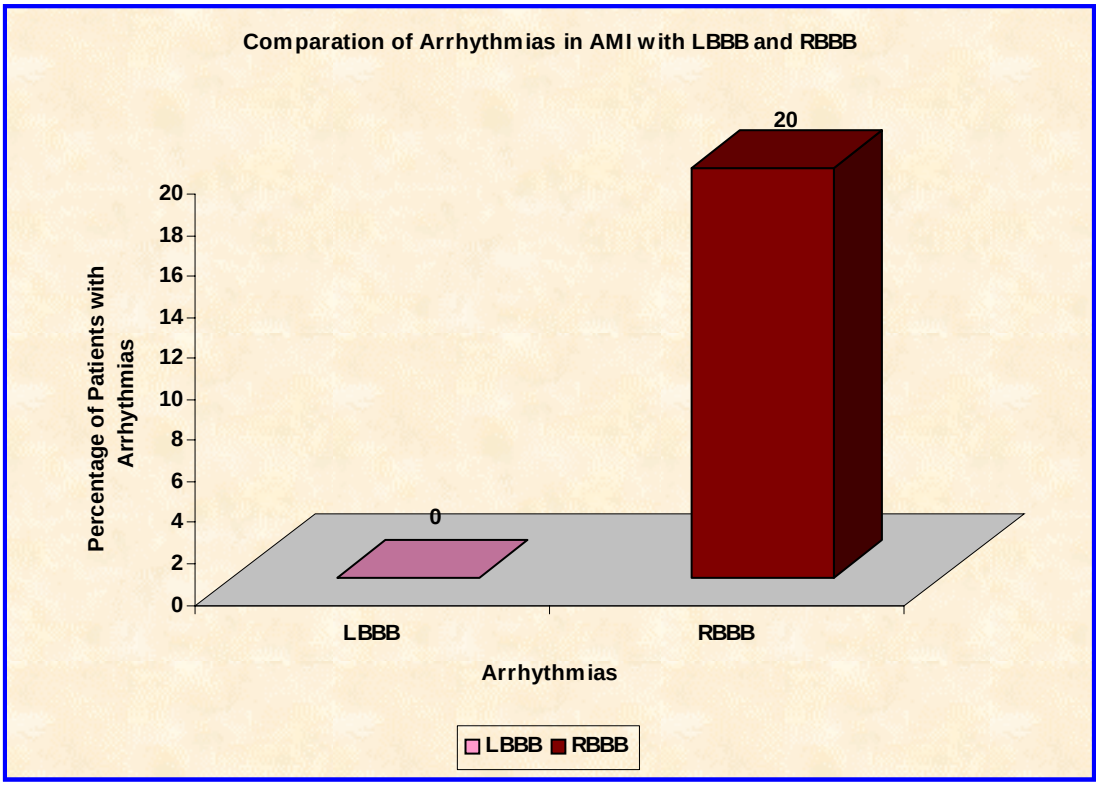
Mortality in various type of BBB

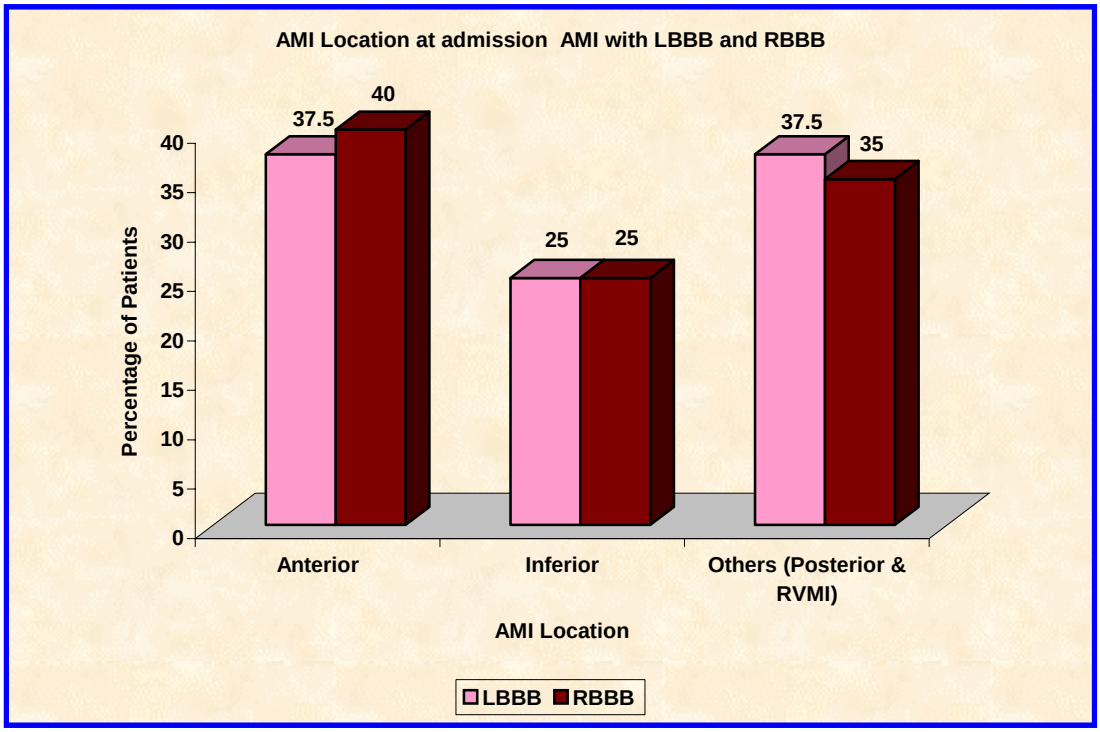
RBBB	LBBB	Bifasicular Block	P Value
Total – 20 Death – 3	Total – 9 Death – 0	Total – 5 Death – 3	0.017 *

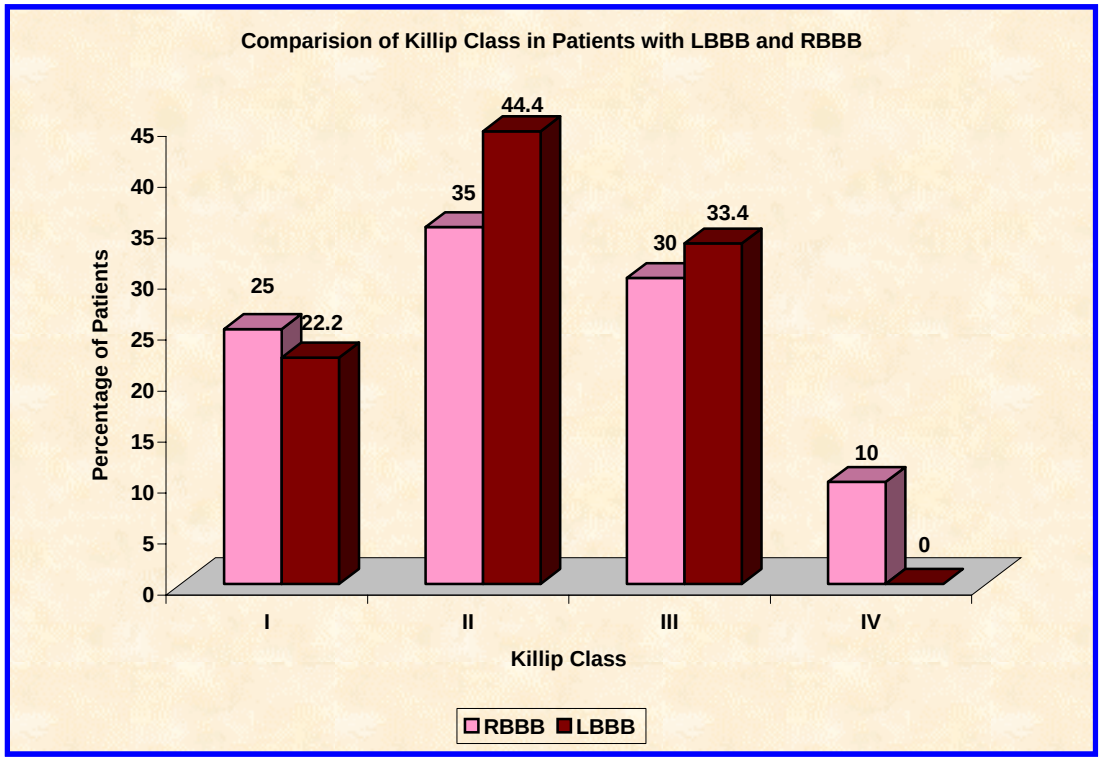


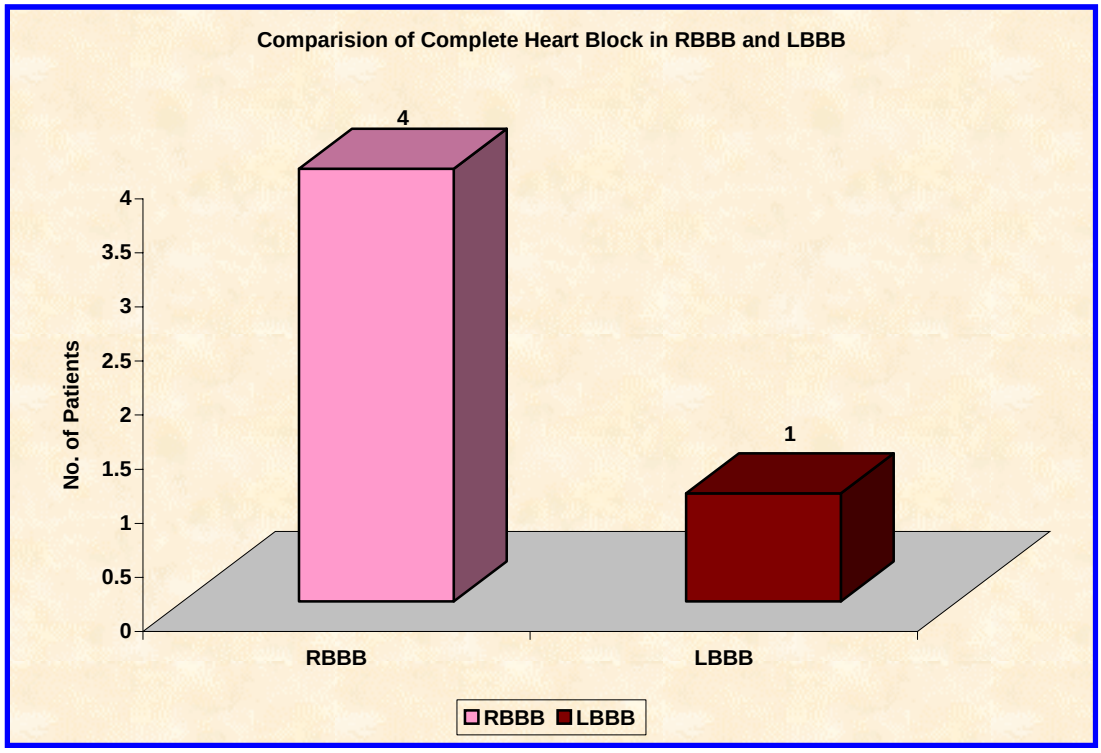
Comparison of LBB and RBBB

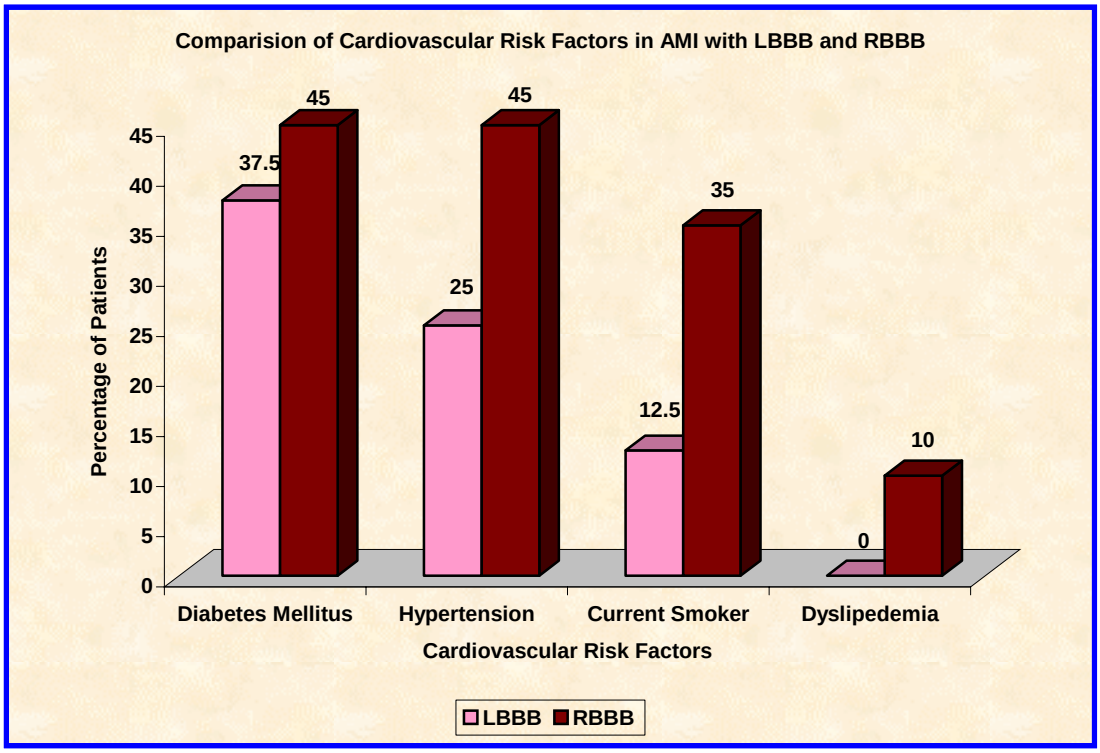
Characteristics	AMI With LBBB	AMI with RBBB
Mean Age (Years)	55.25	65.3
Male Gender (%)	77.8%	65%
Female Gender (%)	22.2%	35%
Cardiovascular history (%)		
• Previous Myocardia infarction	37.5%	20%
• Angina	50%	10%
• Congestive Heart Failure	40%	26%
Cardiovascular Risk Factors (%)		
• Diabetes Mellitus	37.5%	45%
• Hypertension	25%	45%
• Current Smoker	12.5%	35%
• Dyslipedemia	0	10%
Clinical Status on Admission		
Killip Class (%)		
• I (No congestive heart failure)	22.2%	25%
• II (Rales, Jugular venous distension)	44.4%	35%
• III (Pulmonary edema)	33.4%	30%
• IV (Cardiogenic shock)	0%	10%
AMI Location at admission (%)		
• Anterior	37.5%	40%
• Inferior	25%	25%
• Others (Posterior & RVMI)	37.5%	35%
Ejection fraction (%) (mean)	42%	44%
Arrhythmias	0%	20%
Mortality	0%	15%


Comparation of Arrhythmias in AMI with LBBB and RBBB
Percentage of Patients with Arrhythmias
20
18
16
14
12
10
8
6
4
2
0
0
20
LBBB
RBBB
Arrhythmias
LBBB
RBBB


AMI Location at admission AMI with LBBB and RBBB
Percentage of Patients
40
35
30
25
20
15
10
5
0
37.5
40
25
25
37.5
35
Anterior
Inferior
Others (Posterior & RVMI)
AMI Location
LBBB
RBBB

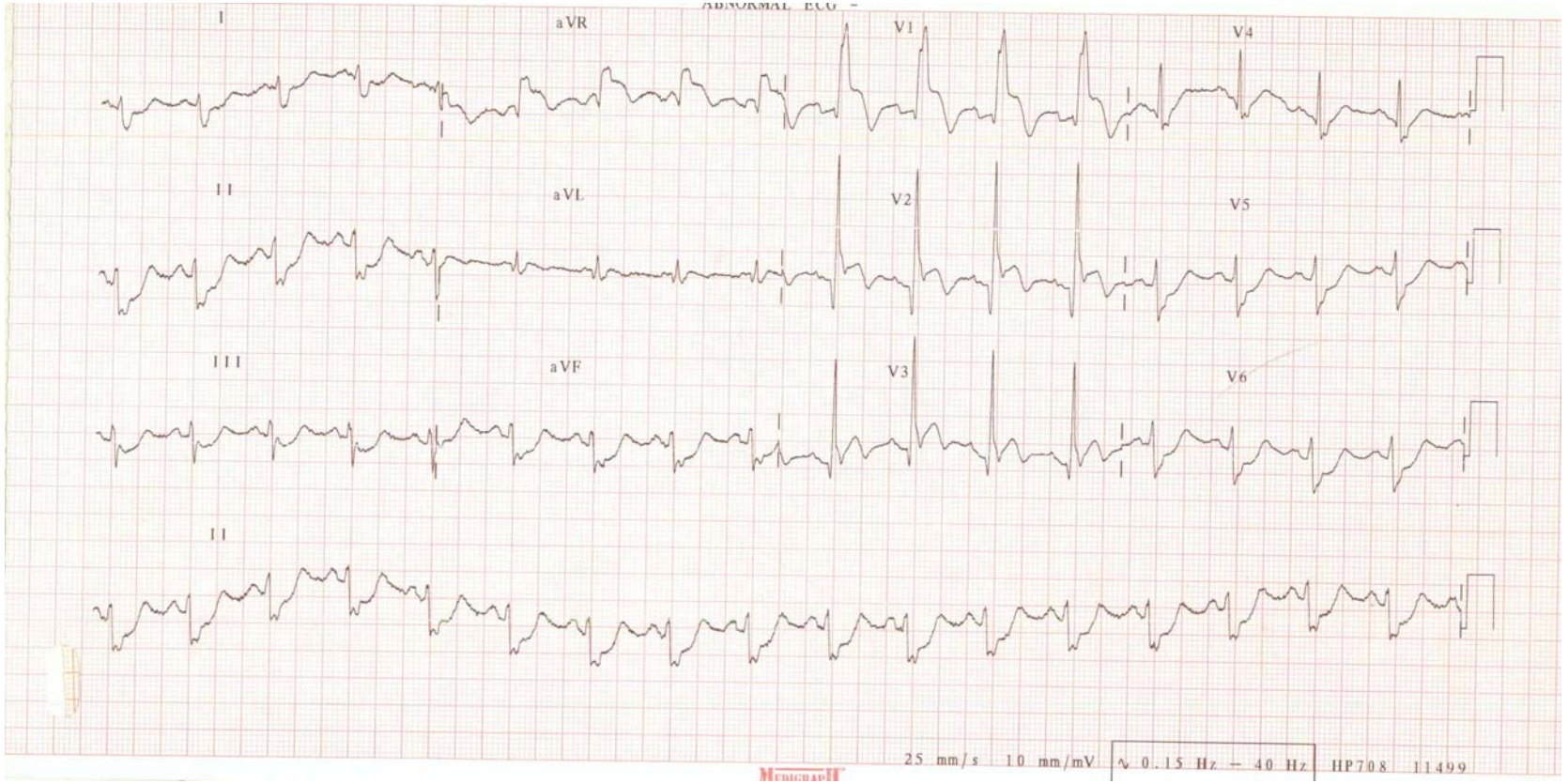

Comparision of Killip Class in Patients with LBBB and RBBB
Percentage of Patients
45
40
35
30
25
20
15
10
5
0
25
22.2
35
44.4
30
33.4
10
0
I
II
III
IV
Killip Class
RBBB
LBBB


Comparision of Complete Heart Block in RBBB and LBBB
No. of Patients
4
3.5
3
2.5
2
1.5
1
0.5
0
4
1
RBBB
LBBB
RBBB
LBBB

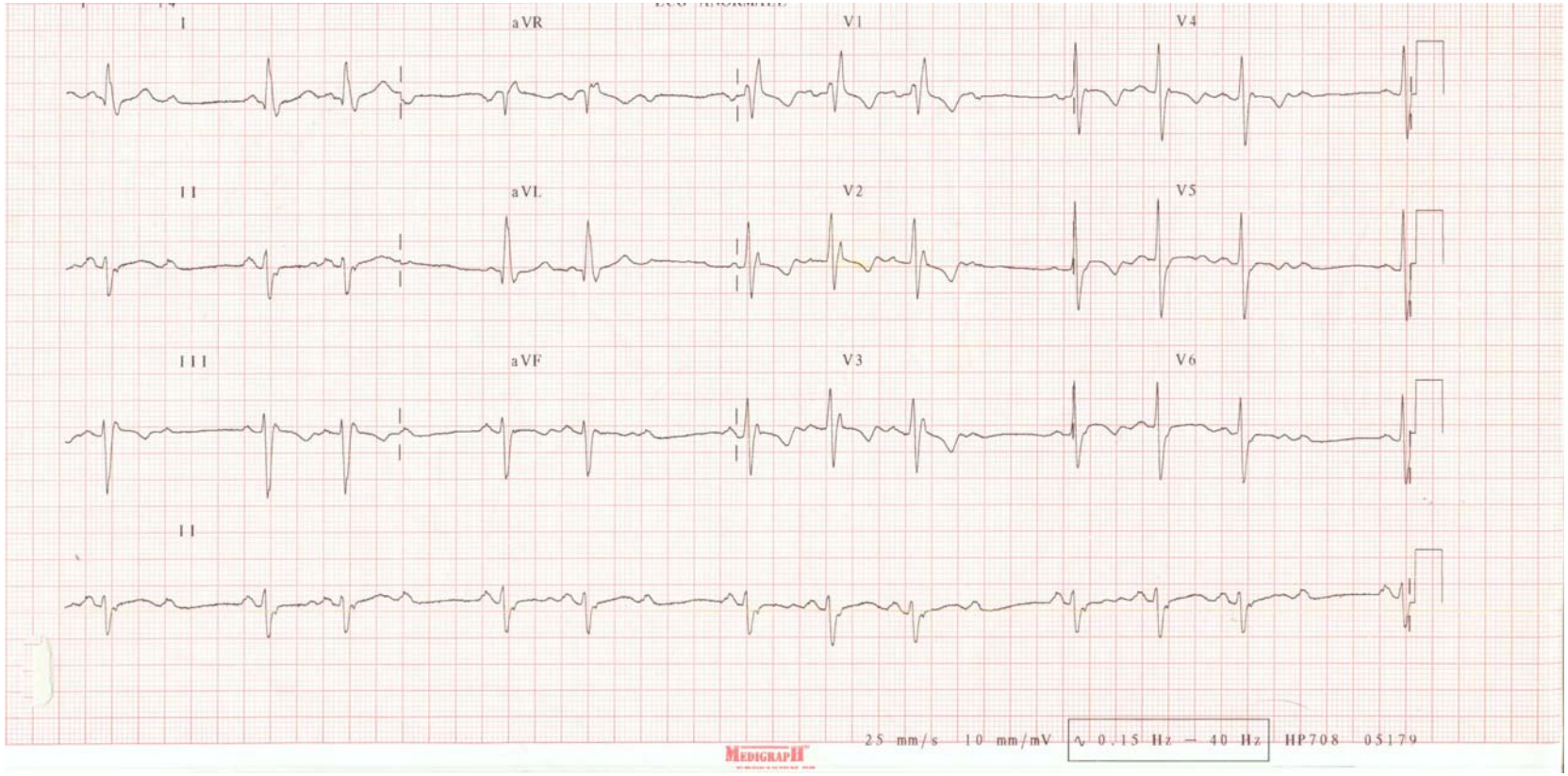

Comparision of Cardiovascular Risk Factors in AMI with LBBB and RBBB
Percentage of Patients
0
5
10
15
20
25
30
35
40
45
37.5
45
25
45
12.5
35
0
10
Diabetes Mellitus
Hypertension
Current Smoker
Dyslipedemia
Cardiovascular Risk Factors
LBBB
RBBB

ECGs

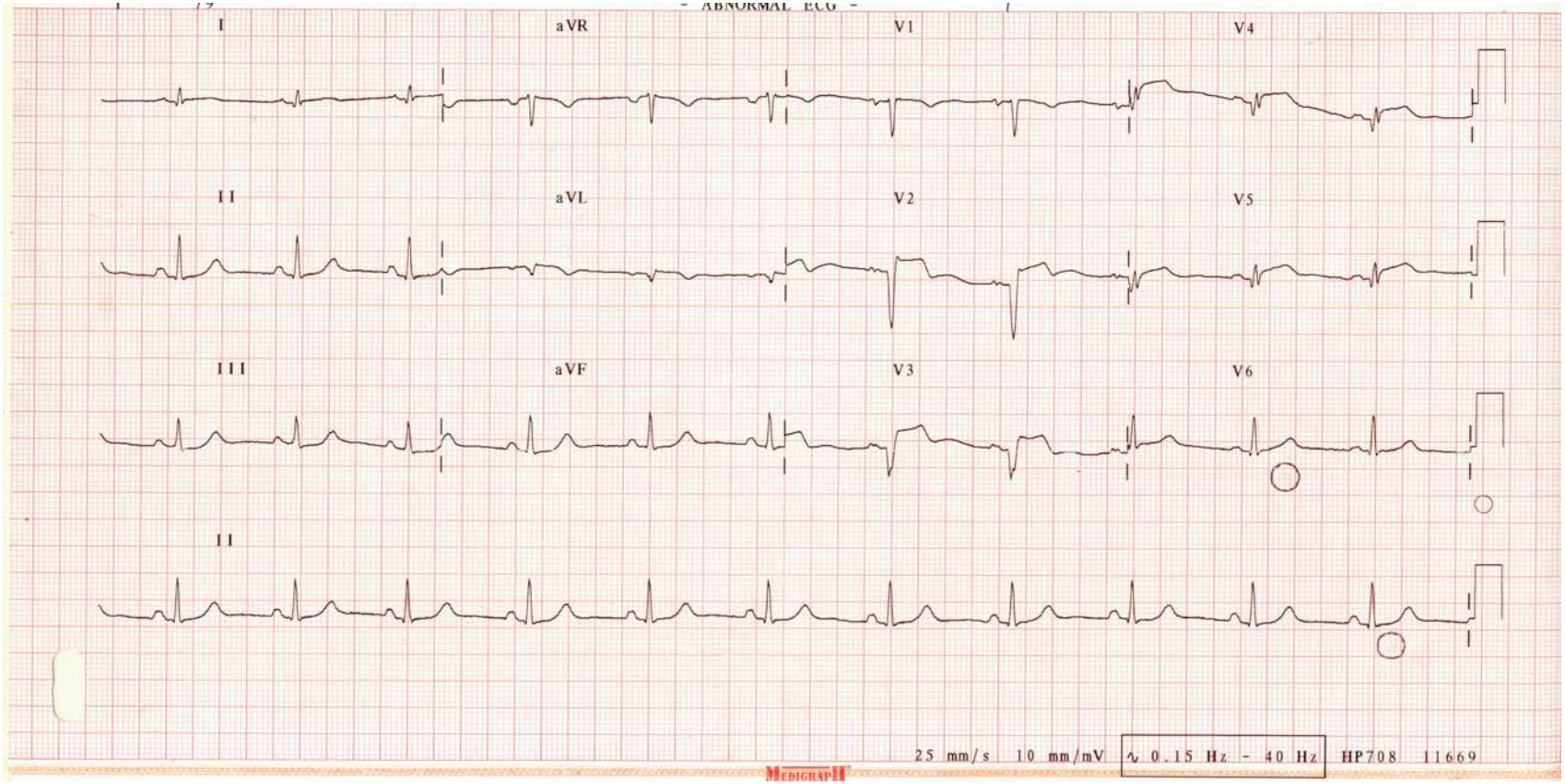
AWMI with Bifasicular Block (RBBB + LAHB)



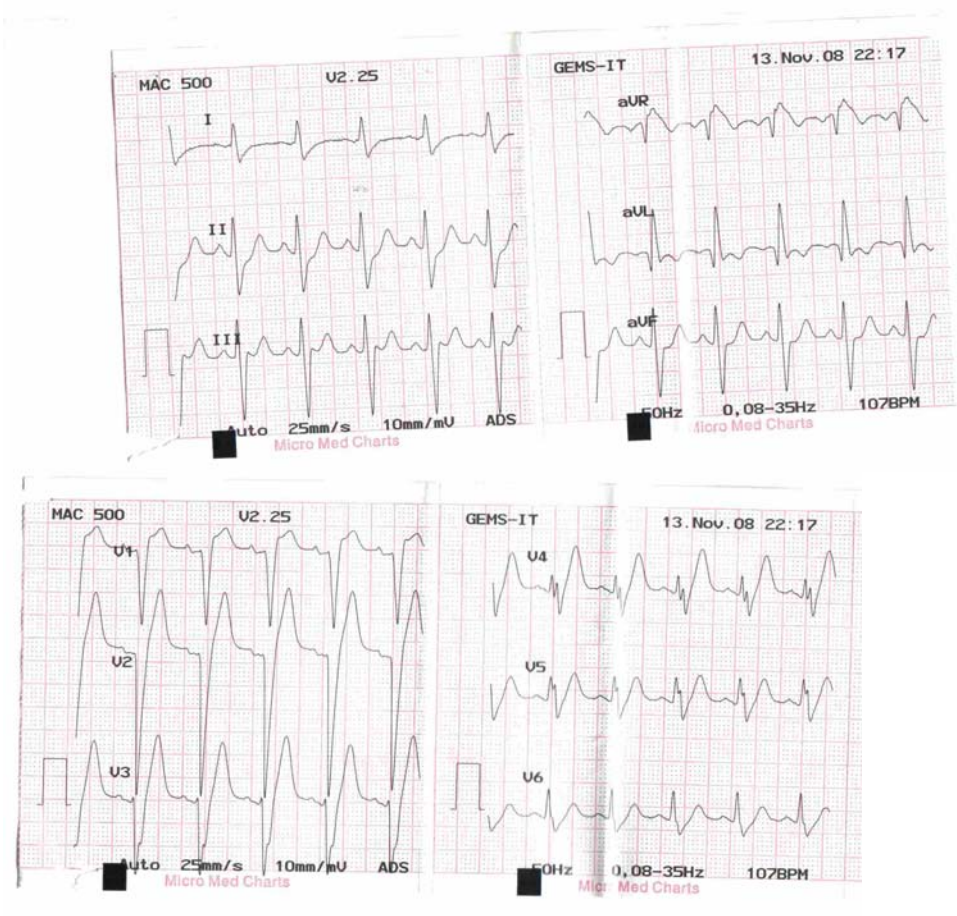
AWMI with Bifasicular Block (RBBB + LAHB) with 2^0 AV Block



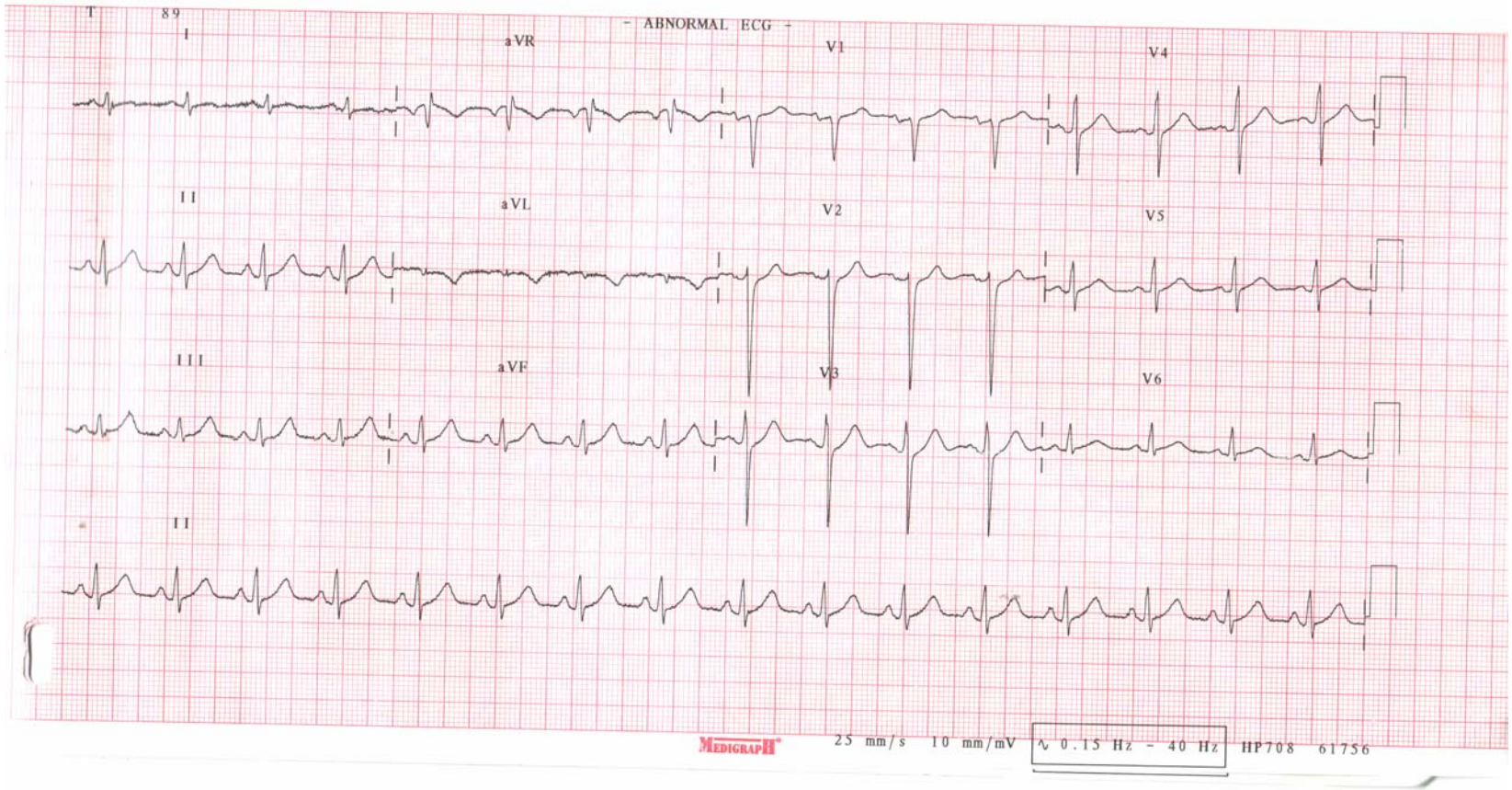
AWMI


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II
25 mm/s 10 mm/mV ∿ 0.15 Hz - 40 Hz HP708 11669

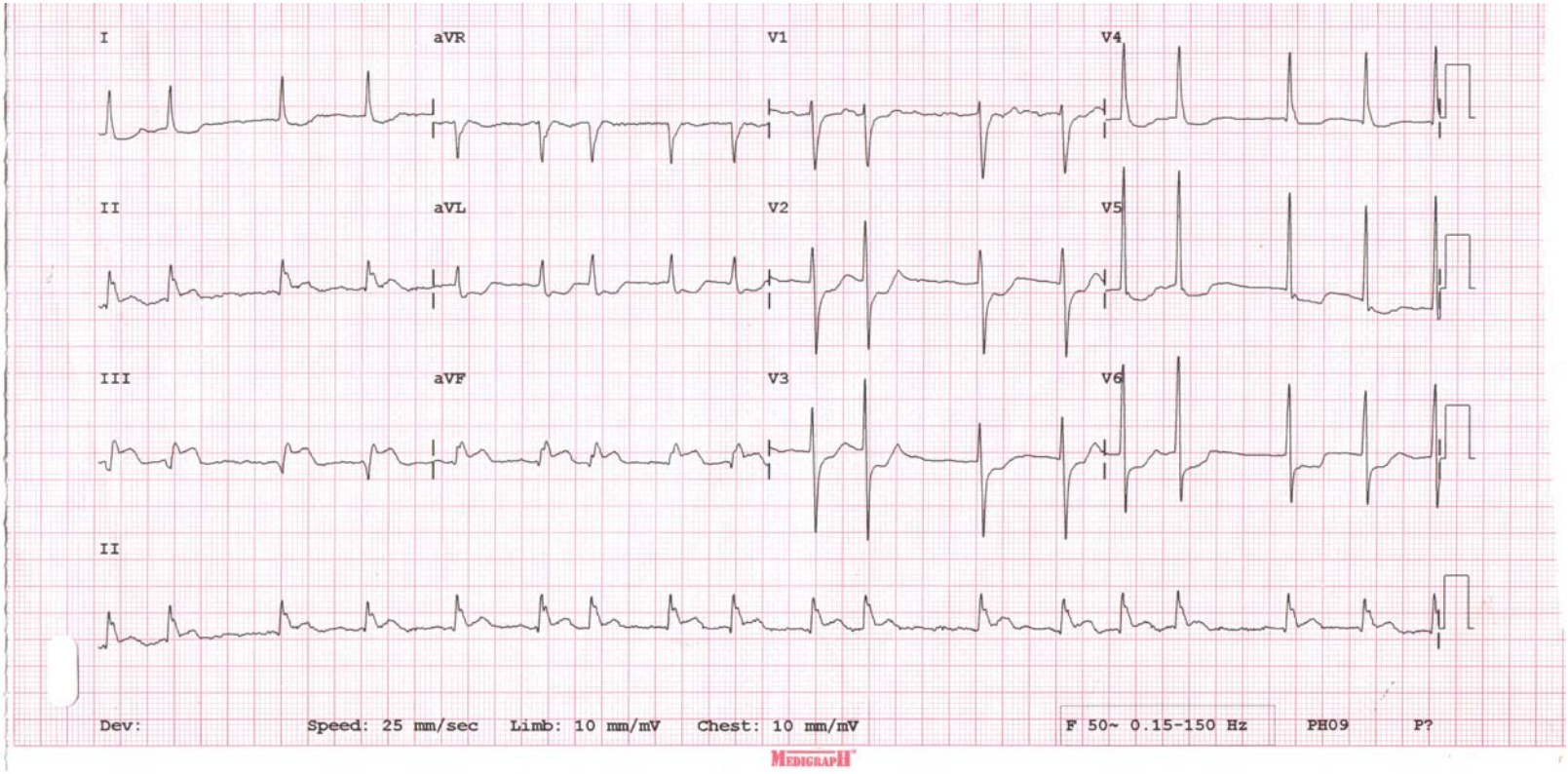
AWMI with LBBB before SK



AWMI with LBBB after SK



IWMI with Atrial Fibrillation



DISCUSSION

Total number of patient included in the study were 150. Variables that were analysed in the study were gender, age, Killip class arrhythmias, complete heart block, MI location, systolic LV dysfunction, diastolic LV dysfunction, recurrent angina thrombolysis, Heart failure, cardiac arrest, death in hospital and death at the end of one month. Risk factors like (DM, HT, Dyslipidemia, smoking, previous history of MI). Blood pressure, previous history of PTCA, CABG, Mechanical complication like VSR., These variables were compared with patients having ACS with new onset BBB and without BBB.

In this study the in-hospital mortality in no BBB group was 8.6% in BBB group was 17.6% at the end of one month there was no increase in mortality between the two groups.

Clinical patterns and mortality in LBBB, RBBB were also compared.

Gender

In this study total number of male patients were - 111 of which 23 (20.72%) had BBB.

Total female patients were 39 of which 11 (28.20%) had BBB.

P value 0.337.

Female patients had higher incidence of BBB.

Age group

In this study high incidence of BBB was noticed in 51-60 yrs age group.

P value - 0.413

Age was not a significant risk factor.

Killip Class

This variable was compared with NO BBB group patients.

P value - 0.001

Killip class II, III, IV were seen more in patients with BBB.

This study concurs with Toporan Daniela et al[8]., study. It showed BBB patients more often associated with heart failure, cardiogenic shock and complete heart block and poor thrombolytic therapy.

Arrythmias

Presence of arrythmias in both groups were compared.

P value - 0.008

BBB group patients had more incidence of arrythmias. It is a significance prognostic indicator. This study concurs with study done by Wong et al [5](Hero trial), Vrugada et al[19].

Complete Heart Block

Incidence of heart block between. BBB and no BBB group were compared.

P value - 0.024

Higher incidence of complete heart block was found in BBB group.

Mortality due to CHB between BBB and no BBB were compared.

P value - 0.035 It is a significant prognostic indicator.

High incidence of death was seen in BBB group.

This study concurs with Melagarejo - Moreno A et al[10]. study it showed new BBB in AMI is an independent predictor of short and long term mortality. Heart failure and complete AV block was more often associated with mortality.

Among the survivors one patients was in need of permanent pacemaker implantation.

MI location

MI location like AWMI, IWMI, others compared between BBB and no BBB group.

P value - 0.208

MI location was not a significant risk factor for BBB.

Systolic LV dysfunction

This variable was compared between BBB and no BBB.

P value - 0.022

High incidence of systolic dysfunction was found in BBB group. It is significant prognostic indicator.

Diastolic LV dysfunction

Compared between BBB and no BBB

P value - 0.001

High incidence of diastolic dysfunction was found in BBB group. It is a significant prognostic indicator.

Recurrent Angina

History of recurrent angina was compared with BBB and no BBB group.

P value - 0.52

This was not a significant variable.

Thrombolysis

Thrombolysis was compared between BBB and no BBB group.

P value - < 0.001.

Increased incidence of late thrombolysis was found in BBB.

This study concurs with study done by Wong CK et al[5]., It showed STEMI patients with BBB have more comorbid conditions, are less likely to receive therapies such as thrombnolytics and had increased in hospital mortality rate.

Heart failure

Incidence of Heart failure between BBB and no BBB were compared.

P. Value - 0.004.

Increased incidence of HF was seen in BBB groups. It is significant prognostic Indicator. The study concurs with study done by Antonio D, Chiara et al[6]., Wong CK et al[5]. and Bharsheshet A et al[16].

Death in Hospital

Compared between BBB and no BBB group.

Percentage of death in BBB group - 17.6%.

Percentage of death in no BBB group - 8.6%.

BBB patients had high percentage of in-hospital mortality.

Mortality at the end of one month was same in both groups.

This study concurred with study done by Toporan Daniela[8] who showed presence of BBB on AMI is an independent strong predictor of poor outcome and was associated with high risk of in-hospital death.

This study concurs with study done by CK Wong et al[5]., Melgarejo - Moreno et al[10]., Gunnarson, G. et al[11]., EB Sgarbossa et al[12]., Dobri-C et al[15]., Barsheshet .A. et al[16].

Risk Factors

Risk factors like DM, HT, Dyslipidemia, smoking were compared between both group.

P value - 0.607.

These risk factors were not a significant for BBB.

History of MI

Previous history of MI was compared between two groups.

P value - 0.589.

This was not a significant variable.

Blood Pressure

Blood pressure of patients on both groups compared. Hypotension was found commonly in BBB groups.

P value - 0.010.

High incidence of Hypotension was present in BBB group. It is a significant prognostic indicator.

History of Surgery and Mechanical complications were not a significant variable.

Mortality among various types of BBB

- Out of 34 patients 20 (58.8%) patients had RBBB, out of these 3 patients died (15%).
- 9 (26.4%) patients had LBBB. No death recorded.
- 5 (14.7%) patients had bifasicular block of which 3 patients died (60%).
- Mortality between these groups were compared.

Bifasicular block (RBBB + LAHB) was associated with increased incidence of mortality. (60%) This study concurs with study done by CK Wong et al[5]., it showed the high mortality and higher incidence of RBBB in patients with AWMI may be explained by Septal ischemia from a more proximal left anterior descending artery occlusion (before the large Septal branch) and the course of the RBB traversing the Septum towards the apex.

Engene Branwald[2] says bifasicular block is associated with high mortality because of chances of complete AV block and occurrence of severe pump failure secondary to extensive myocardial necrosis.

When LBBB, RBBB group were compared. RBBB group had higher incidence (58.8%) and mortality (15%) than LBBB group at the end of 1 month this study concurs with study done by Petrina et al[20]., CK Wong et al[5]., Antonio D Chira et al[6], Sergia Rocha et al[9], Iwasaki et al[14]., Islam MN et al[18]., Vrugada et al[19].,

Mortan F et al study showed presence of bifasicular, or trifasicular block in ACS progress to complete heart block and associated with high mortality.

SUMMARY

Prognostic significance of BBB in ACS was studied in coronary care unit, Department, of cardiology. Madras Medical College, following observations were made.

- Of 150 patients studied 34 (22.66%) patients had new onset BBB.
- BBB patients had higher killip class (II, III, IV)/.
- BBB patients had increased incidence of arrhythmias.
- Higher incidence of complete heart block.
- Higher incidence of death due to complete heart block was observed.
- Higher incidence of systolic and diastolic LV dysfunction.
- BBB was associated with higher incidence of hypotension, heart failure.
- Higher percentage of in hospital death (17.6%).
- Among BBB group higher incidence of Mortality was seen in bifasicular block.

No difference was observed in mortality at the end of 1 month between the two groups.

CONCLUSION

When physicians are called to see a patient with ACS with BBB, major diagnostic and prognostic issues should be addressed. Many studies have dealt with the aspects of this problem.

Patients having new onset BBB accompanying ACS early after fibrinolytic therapy independently have higher in-hospital mortality than patient without these conducting abnormalities. Patients with RBBB are more prone to arrhythmias and heart failure. Patients with LBBB are more prone to systolic LV dysfunction. Patients with bifasicular block are more prone to complete heart block, heart failure and cardiogenic shock.

Among BBB, bifasicular block is associated with higher incidence of mortality.

Bunde Branch block with ACS patients had worse clinical pattern such as

- Higher Killip class
- Arrhythmias
- Complete heart block
- Systolic, diastolic LV dysfunction
- Hypotension
- Heart failure

Emergency physicians and cardiologists should be familiar with the mechanisms related to BBBs and with prognostic implication of BBBs in the setting of ACS. Such knowledge constitutes an immediate available clinical tool for the management of patients with ACS, especially nowadays when the pathways to the optimal reperfusion strategy are available.

BIBLIOGRAPHY

1. Current medical diagnosis and treatment 2009. Chapter 10: Page 308 - 331.

2. Braunwald's heart disease. A textbook of cardiovascular medicine -Eight Edition. Page 1127, 1251, 1277, 1280, 1281, 1289.

3. Text book of cardiovascular Medicine - Topal Cellular Interactions in the development and progression of atherosclerosis. (Source: Weibsberg DL. Atherogenesis: current understanding of the causes of atheroma. Heart 2000: 83:247-252, with permission)

4. Textbook of clinical Electrocardiography. 2nd Edition by SN Chugh. page. 137, 151, 161, 165.

5. CK Wong et al., Prognostic difference between different types of bundle branch block during the early phase of Acute myocardial Infarction insights from the Hirulog and Early reperfusion or occlusion (HERO)-2 trial. EHJ doi:10,1093/ Eurheartj/eh:622.

6. Antonio Di Chiara et al: Right bundle branch block during the acute phase of myocardial Infarction. EHJ (2006) 27, 1-2doi10, 1093/ eu heart/ cli 522.

7. L. Julian Haywood et al; Left bundle branch block in Acute myocardial Infarction. benign (or) Malignant? J.Am.Coll. Cardiol 2005:46:39-41.

8. Toparan daniela et al; Clinical characteristics and Prognosis significance of Bundle Branch Block (BBB) associated with Acuts myocardial Infarction - clinic of Internal medicine and cardiology, Emergency hospital "Saint pantelimon" Bucharest, Romania.

9. Sergia Rocha et al., Prognostic significance of Right bundle Branch Block in Acute Coronary syndrome. ESC congress 2007 - Vienna - Austria.

10. Melgarejo - Moreno et al: Prognostic significance of bundle branch block in acute myocardial Infarction the Importance of location and time of appearance. Clin cardiol-01-May 2001; 24(5): 371-6.

11. Gunnarson.G et al., Bundle Branch Block and acute myocardial Infarction. Treatment and outcome. Scand cardiovasc 01-Dec.2000; 34(6): 575-9.

12. EB sgarbossa et al., Acute myocardial Infarction and complete bundle branch block at hospital admission. Clinical characteristics and outcome in the thrombolytic era. Gusto-1 investigations. Global utilization of streptokinase and t-PA (tissue plasminogen activator) for occluded coronary arteries. J.Am.Coll. Cariol 1998; 31:105-110.

13. Grantz atlas of Anatomy. Page - 62. Conducting system of the heart.

14. Iwasaki et al., Prognostic significance of right bundle branch block in pateitns with acute inferior myocardial infarction. Acta Med Okayama. 2009 Feb:63(1): 25-33.

15. Dobri-c et al., Prognostic significance of acute bundle branch block in patients with acute myocardial Infarction. Vojnosanit Pregl.2009 Jan; 66(1): 74.

16. Barsheshet A et al., Effect of bundle branch block patterns on mortality in hospitalised patients with heart failure. Am J Cardiol. 2008 May 1:101(9): 1303-8.

17. Biagini E et al., Prognostic significance of leftanterior hemiblock in patients with suspected coronary artery disease. J.Am. Coll cardiol.2005 Sept 6:46(5): 858 - 63.

18. Islam MN et al., Incidence and Prognostic significance of right bundle branch block complicating acute myocardial Infarction. Bangladesh med Res Counc Bull. 2002. Apr:28(1): 26-35.

19. Vrugada J et al., Long term follow-up of individuals with the electrocardiographic pattern of right Bundle branch block and ST segment elevation in Precordial leads V_1-V_3. Circulation 2002 Jan 1:105(1):73-8.

20. Petrina M et al., The 12-lead electrocardiogram as a Predictive tool of mortality after acute myocardial Infarction: current status in an era of revasularization and reperfusion. Am.Heart.J.152:11-18, 2006.

21. Compilation by Mortan F et al., Course and treatment of chronic bifasicular and trifasicular block. Sept.12, 1998. EHJ.

MASTER CHART

S.No.	Name	Sex	Age	IP No	Date of onset of ACS	Time of Admission	Clinical status on Admission			BP	HR	RR	BBB		Bifasicular block	Complete heart block	MI Location	CVS History	CVS Risk Factors	Investigations		Thrombolysis	Ventricular Dysfunction	Arrythmias	Recurrent ischemia/ Angina	CHF	Heart Block	Mechanical Complications	Cardiac Arrest	At the time of discharge	At the end of 1 month	Death
							Chest pain	Killip Class	Arrythmias				RBBB	LBBB						CK-MB	ECHO											
1	Elumalai	M	49	50842	26.06.09	30.06.09 11.40 am	+	I	No	160/120	70	18	-	-	-	-	IW MI	Previous AWMI	DM/HT	-	Hypokinesia Infero Posterior Wall EF 30% Gr.II DD	No	EF 30% severe	No	Yes	No	No	No	No	No	Stable	Stable	-
2	Mangali	M	55	50189	28.06.09	28.06.09 5.45 pm	+	III	3rd degree heart block	100/60	50	18	+	-	-	Yes III°	IW MI RV MI	-	DM/HT Dyslipidemia	564	Severe LVDF Gr.II DD Hypokinesia & Infero Posterior Wall	Yes	EF 45% moderate	Yes	No	-	Recovered	No	No	Stable	Stable	-
3	Abirami	F	65	50210	30.06.09	01.07.09 3 pm	-	II	No	100/80	90	16	+	-	-	-	AW MI	-	HT	-	Severe LVDF Gr.II DD	Delayed Presentation	EF 30% severe	-	-	-	-	-	-	Stable	Stable	-
4	Srinivasan	M	70	50167	28.06.09	28.06.09 2.12 pm	+	II	-	110/60	60	16	-	+	-	-	AW MI	-	-	584	EF 41% Gr.I DD	Yes	EF 41% moderate	-	-	-	-	-	-	LBBB	Stable	-
5	Anjammal	F	42	51218	TW 8 hrs	01.07.09 4.05 pm	+	I	-	120/80	80	N	-	-	-	-	IW MI PW MI	-	-	-	EF 48% Gr.I DD	Yes	Mild	-	-	-	-	-	-	Stable	Stable	-
6	Ahmed	M	65	51028	01.07.09	01.07.09 2.02 am	-	I	-	140/80	100	N	-	-	-	-	AW MI	-	DM	-	EF 47% No DD	Yes	Mild	-	-	-	-	-	-	Stable	Stable	-
7	Gopal	M	50	51107	24.06.09	01.07.09	+	I	-	130/90	80	N	-	-	-	-	AW MI	-	HT	-	EF 40% Gr.I DD	Late presentation	Moderate	-	-	-	-	-	-	Stable	Stable	-
8	soundarajan	M	41	50621	28.06.09	29.06.09	+	I	-	120/80	70	N	-	-	-	-	AW MI	-	-	-	EF 45% Gr.I DD	No	Moderate	-	-	-	-	-	-	Stable	Stable	-
9	Senthamarai	M	70	50071	TW 10 hrs	27.06.09	+	I	1° AV block	130/80	80	N	-	-	-	-	IW MI RV MI	-	-	130	EF 42% Gr.I DD	Yes	Moderate	-	-	-	-	-	-	Stable	Stable	-
10	Nagappan	M	55	52570	TW 7½ hrs	07.07.09	+	I	-	100/70	80	N	-	-	-	-	AW MI	-	-	-	EF 42% Gr.I DD	Yes	Moderate	-	-	-	-	-	-	Stable	Stable	-
11	Neelamegam	M	43	59376	TM 2½ hrs	02.08.09	+	I	-	120/90	90	N	+	-	-	-	AW MI	Previous AWMI	Smoker Alcoholic	-	EF 43% No DD	Yes	-	-	Yes	-	-	-	-	Stable	Stable	-

S.No.	Name	Sex	Age	IP No	Date of onset of ACS	Time of Admission	Clinical status on Admission			BP	HR	RR	BBB		Bifasicular block	Complete heart block	MI Location	CVS History	CVS Risk Factors	Investigations		Thrombolysis	Ventricular Dysfunction	Arrythmias	Recurrent ischemia/ Angina	CHF	Heart Block	Mechanical Complications	Cardiac Arrest	At the time of discharge	At the end of 1 month	Death
							Chest pain	Killip Class	Arrythmias				RBBB	LBBB						CK-MB	ECHO											
12	Mary Vanitha	F	65	51214	TW 4½ hr	01.07.09		II	-	130/70	55	N	-	+	-	Yes on permanent pace	IW MI RV MI	-	DM/HT	-	EF 32% Gr.II DD	Yes	Severe	-	-	-	3° HB	-	-	Stable	Stable	-
13	Palaniammal	F	45	50120	TW 5 hrs	04.07.09	+	I	-	200/100	80	N	-	-	-	-	IW MI	-	HT	-	EF 47% Grade I	Yes	Mild	-	-	-	-	-	-	Stable	Stable	
14	Somasundaram	M	65	51715	TW 9 hrs	03.07.09	+	I	-	130/80	80	N	-	-	-	-	AW MI	-	-	-	EF 43% GradeI	No	Moderate	-	-	-	-	-	-	Stable	Stable	
15	Mani	M	60	51712	TW 6 hrs	02.07.09	+	I	-	90/70	78	N	-	-	-	-	UA	Previous AWMI 2002	DM/HT	-	-	No	Severe	-	Yes	+	-	-	Yes in Hospital			Yes
16	Gunasundari	F	47	52026	2 days	05.07.09	+	I	-	130/90	108	N	-	-	-	-	IW MI	-	DM/HT	-	EF 40% Grade DF	No Delayed	Moderate	-	-	-	-	-	-	Stable	Stable	
17	Jambulingam	M	60	51956	TW >12 h	04.07.09	-	II	-	150/90	80	N	+	-	-	-	IW MI	Previous CVA	DM/HT	-	EF 38% Grade DI	No	Moderate	-	-	-	-	-	-	Stable	Stable	
18	Anirunisha	F	40	52552	TW 6 hrs	07.07.09	+	II	-	150/90	80	N	-	-	-	-	IW MI	-	DM/HT	-	EF 47% Grade II	No	Mild	-	-	-	-	-	-	Stable	Stable	
19	Annammal	F	68	52127	TW 2 days	06.07.09	-	II	-	130/80	50	N	+	-	-	-	IW MI	-	DM/HT	-	EF 32% Grade III	No	Severe	-	-	-	Complete+ Resolved	-	-	Stable	Stable	
20	Roosevelt	M	60	52343	TW 6 hrs	07.07.09	+	III	-	160/80	82	N	+	-	-	-	AW MI	-	HT/DM/ Smoker	-	EF 34% Grade II D	Yes	Moderate	-	-	+	-	-	-	Stable	Stable	
21	Puspavalli	F	56	52386	TW 4½ hr	07.07.09	+	I	-	130/80	80	N	-	-	-	-	IW MI	Old AWMI	DM/HT	-	EF 32% Grade III	Yes	Severe	-	-	-	-	-	-	Stable	Stable	
22	Veerapathran	M	44	52479	TW 1 hr	07.07.09	+	I	-	110/80	70	N	-	-	-	-	IW MI	-	HT/DM	-	EF 47% Grade I	Yes	Mild	-	-	-	-	-	-	Stable	Stable	
23	Srinivasan	M	81	52484	TW <6 hrs	11.07.09	+	I	-	110/80	72	N	-	-	-	-	IW MI PW MI	-	HT	-	EF 40% Grade DI	Yes	Moderate	-	-	-	-	-	-	Stable	Stable	

S.No.	Name	Sex	Age	IP No	Date of onset of ACS	Time of Admission	Clinical status on Admission			BP	HR	RR	BBB		Bifasicular block	Complete heart block	MI Location	CVS History	CVS Risk Factors	Investigations		Thrombolysis	Ventricular Dysfunction	Arrythmias	Recurrent ischemia/ Angina	CHF	Heart Block	Mechanical Complications	Cardiac Arrest	At the time of discharge	At the end of 1 month	Death
							Chest pain	Killip Class	Arrythmias				RBBB	LBBB						CK-MB	ECHO											
24	Mohan	M	55	54178	TW >24 h	14.07.09	+	I	-	130/80	78	N	-	-	-	-	AW MI	-	Smoker	-	EF 40% Grade DI	No	Moderate	-	-	-	-	-	-	Stable	Stable	
25	Palani	M	64	54184	12 hrs	14.07.09	+	I	-	160/90	80	N	-	-	-	-	UA	Previous IWMI	DM/HT	-	EF 42% Grade I DD	No	Moderate	-	Yes	-	-	-	-	Stable	Stable	
26	Shanker	M	41	54180	TW <6 hrs	14.07.09	+	I	-	130/80	80	N	-	-	-	-	AW MI	-	-	-	EF 47% No DD	Yes	Mild	-	-	-	-	-	-	Stable	Stable	
27	Murthi	M	45	54785	TW >6 hrs	17.07.09	+	I	-	110/80	80	N	-	-	-	-	IW MI	-	-	-	EF 42% Gr.I DD	No	Moderate	-	-	-	-	-	-	Stable	Stable	
28	Munisamy	M	55	55014	TW >24 h	17.07.09	+	I	-	160/100	80	N	-	-	-	-	IW MI	-	DM/HT	-	EF 41% Gr.I DD	No	Moderate	-	-	-	-	-	-	Stable	Stable	
29	Nagamani	F	45	54787	TW 1 day	17.07.09	+	III	-	110/80	90	N	-	-	-	-	UA	Previous IWMI ASMI	DM/HT	-	EF 25% Gr.III DD	No	Severe	-	-	+	-	-	+			yes
30	Kuttiammal	F	73	54782	TW >12 h	17.07.09	-	III	-	100/70	50		-	-	-	+	AW MI	-	-	-	-	No	-	-	-	+	+	-	+			Yes
31	Annadurai	M	54	55191	TW 12 hrs	18.07.09	+	II	-	140/100 80/70	80	N	-	-	-	-	AW MI	-	HT/Smoker	-	-	No	-	-	-	+	-	-	+			Yes
32	Dayalan	M	42	55438	TW <6 hrs	19.07.09	+	I	-	130/80	80	N	-	-	-	-	IW MI	-	Smoker/	-	EF 46% Gr.I DD	Yes	Mild	-	-	-	-	-	-	Stable	Stable	
33	Subramaniyan	M	76	55795	TW >12 h	20.07.09	+	I	-	180/100	90	N	-	-	-	-	IW MI	-	HT,CKD	-	EF 42% Gr.II DD	No	Moderate	-	-	-	-	-	-	Stable	Stable	
34	Gunasekar	M	74	55884	TW >6 hrs	21.07.09	+	IV	AF	70/?	80		-	-	-	-	IW MI PW MI	-	-	-	-	No	-	AF	-	+	-	-	+			yes
35	Devaraj	M	45	56436	TW 10 hrs	27.07.09	+	I	-	110/80	80		-	-	-	-	RV MI PW MI	-	-	-	EF 41% Gr.I DD	No	Moderate	-	-	-	-	-	-	Stable	Stable	

S.No.	Name	Sex	Age	IP No	Date of onset of ACS	Time of Admission	Clinical status on Admission			BP	HR	RR	BBB		Bifasicular block	Complete heart block	MI Location	CVS History	CVS Risk Factors	Investigations		Thrombolysis	Ventricular Dysfunction	Arrythmias	Recurrent ischemia/ Angina	CHF	Heart Block	Mechanical Complications	Cardiac Arrest	At the time of discharge	At the end of 1 month	Death
							Chest pain	Killip Class	Arrythmias				RBBB	LBBB						CK-MB	ECHO											
36	Deeplai Rai	F	62	56193	10 hrs	21.07.09	+	I	-	110/70	60		-	-	-	-	UA	-	DM/HT	-	EF 46% Gr.I DD	No	Mild	-	-	-	-	-	-	Stable	Stable	
37	Thiyalnayagi	F	63	55883	TW UA	21.07.09	+	I	-	90/70	90	N	-	-	-	-	UA	Previous AWMI	DM/HT		EF 40% Grade I DD	No	Moderate	-	-	-	-	-	-	Stable	Stable	
38	Chennareddy	M	75	56429	TW 6 hrs	22.07.09	+	I	-	130/80	100		-	-	-	+	RV MI PW MI	-	-		EF 32% Grade II DD	Yes	Severe	-	-	-	-	-	-	Stable	Stable	
39	Bastin	M	87	56448	TW 6 hrs	22.07.09	+	IV	-	120/80	50	N	+	-	LAHB	-	AW MI	-	Smoker Alcoholic		-	Yes	-	-	-	-	+	-	-	+	-	-	yes
40	Fathima	F	45	56887	TW 2 days	24.07.09	+	I	-	120/80	70	N	-	-	-	-	AW MI	Previous MIIW	DM/HT	###	EF 30% DD Grade II	No	Severe	-	-	-	-	-	-	Stable	Stable	
41	Bakthavatsalu	M	52	57415	TW 9 hrs	26.07.09	-	I	-	100/70	80	N	-	-	-	-	IW MI PW MI	-	Smoker Alcoholic		EF 42% Grade I DD	Yes	Moderate	-	-	-	-	-	-	Stable	Stable	
42	Vasanthammal	F	60	57332	TW >3 day	25.07.09	+	III	-	160/90	86	N	-	+	-	-	AW MI	-	HT		EF 30%	Yes	Severe	-	-	-	-	-	-	Stable	Stable	
43	Krishnan	M	80	57330	TW 2 days	25.07.09	-	I	-	190/110	80	N	-	-	-	-	AW MI	-	Smoker Alcoholic		EF 42% Grade I DD	No	Moderate	-	-	-	-	-	-	Stable	Stable	
44	Tajuddin	M	52	57990	TW 6 hrs	26.07.09	+	I	-	160/100	90		-	-	-	-	AW MI	-	HT		EF 42% Grade I DD	Yes	Moderate	-	-	-	-	-	-	Stable	Stable	
45	Dayanithi	M	52	57883	TW >11 h	27.07.09	+	II	-	110/80	70	N	+	-	LAHB	-	AW MI	-	Smoker		EF 35% GradeI	Yes	Moderate	-	-	-	-	-	-	Stable	Stable	
46	Indira	F	49	57992	TW 6 hrs	27.07.09	+	I	-	130/80	80	N	-	-	-	-	PW, IW, MI	Previous MIIW	-		EF 30% Grade I DD	Yes	Severe	-	-	-	-	-	-	Stable	Stable	
47	Munisamy	M	60	57042	TW 6 hrs	24.07.09	+	IV	+	110/80	60	N	+	-	-	-	IW MI PW MI	Previous MI	DM/HT Smoker/ Alcoholic	213	EF 30% Grade II DD	Yes	Severe	VT	-	-	-	-	+	-	-	yes

S.No.	Name	Sex	Age	IP No	Date of onset of ACS	Time of Admission	Clinical status on Admission			BP	HR	RR	BBB		Bifasicular block	Complete heart block	MI Location	CVS History	CVS Risk Factors	Investigations		Thrombolysis	Ventricular Dysfunction	Arrythmias	Recurrent ischemia/ Angina	CHF	Heart Block	Mechanical Complications	Cardiac Arrest	At the time of discharge	At the end of 1 month	Death
							Chest pain	Killip Class	Arrythmias				RBBB	LBBB						CK-MB	ECHO											
48	Manoharan	M	43	57324	TW 3 hrs	25.07.09	+	I	-	130/80	80	N	-	-	-	-	AW MI	-	Smoker Alcoholic		EF 47% No DD	Yes	Mild	-	-	-	-	-	-	Stable	Stable	
49	Ellammal	F	76	57882	TM >24 hr	24.07.09	-	I	-	100/70	90	N	-	-	-	-	AW MI	-	DM/HT	810	EF 30% Grade I DD	No	Severe	-	-	-	-	-	-	Stable	Stable	
50	Avediammal	F	48	57842	TW 6 hrs	24.07.09	+	I	-	140/90	78		-	-	-	-	IW MI	-	-		EF 47% Grade I DD	Yes	Mild	-	-	-	-	-	-	Stable	Stable	
51	Gandhi	M	50	58480	TW 7 hrs	29.07.09	+	I	-	110/80	70	N	+	-	-	-	AW MI	-	DM/HT/Smoker Alcoholic		EF 42% Grade I DD	Yes	Moderate	-	-	-	-	-	-	Stable	Stable	
52	Neethi	M	36	58494	TW 6 hrs	29.07.09	+	I	-	110/80	80	N	-	-	-	-	IW MI	-	Smoker Alcoholic		EF 47% Grade I DD	Yes	Mild	-	-	-	-	-	-	Stable	Stable	
53	Arumugam	M	60	58260	TW 6 hrs	29.07.09	+	III	-	130/80	86	N	-	-	-	-	AW MI	-	DM/Smoker/ Alcoholic		EF 41% Gr.I DD	Yes	Moderate	-	-	-	-	-	-	Stable	Stable	
54	Subramani	M	67	58502	TW 4 hrs	29.07.09	+	I	1° AV block	130/90	70	N	-	-	-	-	IW MI	Angina	DM		EF 46% No DD	Yes	Mild	-	-	-	° AV bloc	-	-	Stable	Stable	
55	Annamalai	M	60	58508	TW 8 hrs	29.07.09	+	II	-	110/80	80	N	-	-	-	-	AW MI	-	-		EF 42% Grade I DD	Yes	Moderate	-	-	-	-	-	-	Stable	Stable	
56	Chandrasekar	M	50	58862	TW 6 hrs	30.07.09	+	I	-	140/90	80	N	-	-	-	-	RV MI PW MI	-	Smoker/ Alcoholic	546	EF 43% Grade I DD	Yes	Moderate	-	-	-	-	-	-	Stable	Stable	
57	Prabahar	M	43	58799	TW >12 hr	30.07.09	-	I	-	160/70	80	N	-	-	-	-	AW MI	-	DM	###	EF 43% Grade I DD	No	Moderate	-	-	-	-	-	-	Stable	Stable	
58	Venkatesh	M	38	58563	TW 12 hrs	30.07.09	+	I	-	130/80	82	N	-	-	-	-	IW MI	-	-		EF 30% DD Grade II	Delayed	Severe	-	-	-	-	-	-	Stable	Stable	
59	Kesavan	M	55	58672	TW 8 hrs	31.07.09	+	I	-	110/80	80	N	-	-	-	-	AW MI	-	-		EF 48% Gr.I DD	Yes	Mild	-	-	-	-	-	-	Stable	Stable	

S.No.	Name	Sex	Age	IP No	Date of onset of ACS	Time of Admission	Clinical status on Admission			BP	HR	RR	BBB		Bifasicular block	Complete heart block	MI Location	CVS History	CVS Risk Factors	Investigations		Thrombolysis	Ventricular Dysfunction	Arrythmias	Recurrent ischemia/ Angina	CHF	Heart Block	Mechanical Complications	Cardiac Arrest	At the time of discharge	At the end of 1 month	Death
							Chest pain	Killip Class	Arrythmias				RBBB	LBBB						CK-MB	ECHO											
60	Chinnathai	F	85	60112	TW >30 m	04.08.09	+	I	-	110/70	90	N	-	-	-	+	IW MI	-	-		EF 40% Grade I DD	-	Moderate	-	-	-	Complete Heart Block Reverted	On temporary pacemaker	-	Stable	Stable	
61	Gopalakrishnan	M	50	59896	TW 2 hrs	04.08.09	+	I	-	160/90	70		+	-	-	-	IW MI	Previous AW MI	Dislipidemia	153	EF 44% No DD	Yes	Mild	-	-	-	-	-	-	Stable	Stable	
62	Mothibasha	M	40	59894	TW 24 hrs	04.08.09	+	I	-	110/80	80		-	-	-	-	IW MI	-	-		EF 48% No DD	Yes	Mild	-	-	-	-	-	-	Stable	Stable	
63	Mumtaz	F	60	59833	TW >12 h	03.08.09	+	I	-	140/80	80	N	-	-	-	-	IW MI RV MI	-	DM/HT		EF 56% Normal	-	-	-	-	-	-	-	-	Stable	Stable	
64	Vijaya	F	49	60685	TW >12 h	06.08.09	-	II	-	90/60	100	N	-	-	LAHB	-	AW MI	Previous MI PTCA done	DM		EF 39% Grade I DD	-	Moderate	-	-	-	-	-	-	Stable	Stable	
65	Mariyan	M	55	60658	TW 6 hrs	06.08.09	+	I	-	110/80	90		-	-	-	-	AW MI	DM/ Smoker Alcoholic			EF 42% Grade I DD	Yes	Moderate	-	-	-	-	-	-	Stable	Stable	
66	Saroja	F	48	60748	TW 5 hrs	06.08.09	+	II	-	80/60	90	N	+	-	-	+	IW, MI, RV MI	-	DM		EF 30% Grade III	Yes	Severe	-	-	-	-	-	-	Stable	Stable	
67	Paruna	F	57	60444	TW >12 h	05.08.09	+	III	-	120/90	100	N	-	-	-	-	AW MI	-	HT		EF 32% Grade II DD	No	Severe	-	-	+	-	-	-	Stable	Stable	
68	Noorkhan	M	50	61873	TW >3 hrs	11.08.09	+	I	-	150/90	80	N	-	-	-	-	AW MI	-	HT/DM/Smoker Alcoholic		EF 42% Grade I DD	-	Moderate	-	-	-	-	-	-	Stable	Stable	
69	Lakshmipathy	M	58	61024	TW 12 hrs	07.08.09	+	I	-	130/80	78	N	-	-	-	+	IW MI	-	DM		EF 33% Grade II DD	Yes	Severe	-	-	+	Complete on transvers	Pacemaker	+			yes
70	Victor	M	68	61789	TW >12 h	11.08.09	-	II	-	180/90	80	N	-	-	-	-	AW MI	-	DM/HT	527	EF 43% Grade I DD	No	Moderate	-	-		-	-	-	Stable	Stable	
71	Sekar	M	38	62616	TW 6 hrs	13.08.09	+	I	-	200/110	65	N	-	-	-	-	IW MI	-	HT/Smoker		EF 40% Grade IIDD	Yes	Moderate	-	-		-	-	-	Stable	Stable	

S.No.	Name	Sex	Age	IP No	Date of onset of ACS	Time of Admission	Clinical status on Admission			BP	HR	RR	BBB		Bifasicular block	Complete heart block	MI Location	CVS History	CVS Risk Factors	Investigations		Thrombolysis	Ventricular Dysfunction	Arrythmias	Recurrent ischemia/ Angina	CHF	Heart Block	Mechanical Complications	Cardiac Arrest	At the time of discharge	At the end of 1 month	Death
							Chest pain	Killip Class	Arrythmias				RBBB	LBBB						CK-MB	ECHO											
72	Palani	M	56	62659	TW 10 hrs	13.08.09	+	II	-	70/50	70	N	+	-	-	-	IW MI	-	Smoker		EF 30% Grade III DD	No	Severe	-	-	-	-	-	-	Stable	Stable	
73	Namachivayam	M	76	62454	TW 2 hrs	12.08.09	+	I	-	160/90	80	N	-	-	-	-	IW MI	Previous AW MI	DM/ Dyslipidemia		EF 30% Grade I	Yes	Severe	-	+	-	-	-	-	Stable	Stable	
74	Govindan	M	58	62488	1 hr	12.08.09	+	I	-	140/90	80	N	-	-	-	-	IW MI	-	Smoker Alcoholic		EF 48% No DD	Yes	Mild	-	-	-	-	-	-	Stable	Stable	
75	Dhanasekar	M	44	62451	11 hrs	12.08.09	+	II	-	140/90	90	N	+	-	-	-	AW MI	-	Smoker/ Alcoholic		EF 40% Grade	Yes	Moderate	-	-	-	-	-	-	Stable	Stable	
76	Raja Mohamed	M	31	62780	>12 hrs	14.08.09	+	I	-	130/80	70	N	-	-	-	-	IW MI	-	Smoker/ Alcoholic		EF 42% Grade	No	Moderate	-	-	-	-	-	-	Stable	Stable	
77	Raja	M	47	62866	<6 hrs	14.08.09	+	I	-	130/80	80	N	-	-	-	-	IW MI	-	Smoker		EF 47% Grade	Yes	Mild	-	-	-	-	-	-	Stable	Stable	
78	Gopal	M	65	62730	5 hrs	14.08.09	+	I	-	100/70	80		-	-	-	-	IW MI RV MI	Previous IW MI AW MI	DM		EF 32% Grade	Yes	Severe	-	+	-	-	-	-	Stable	Stable	
79	Janardhanan	M	71	62872	>12 hrs	14.08.09	-	III	-	130/80	90	N	-	+	-	-	IW MI	Previous IW MI	HT/COPD		EF 30% Grade	No	Severe	-	+	+	-	-	-	Stable	Stable	
80	Faruk Sahib	M	52	62857	12 hrs	14.08.09	-	III	VPC+	130/80	78	N	-	+	-	-	IW MI	Previous IW MI	DM		EF 30% Grade	No	Severe	-	+	+		-	-	Stable	Stable	
81	Doss	M	54	62961	<6 hrs	14.08.09	+	I	VPC+	110/80	80	N	-	+	-	-	AW MI	-	-		EF 40% Grade	Yes	Mild	-	-	-	-	-	-	Stable	Stable	
82	Dhanabakiam	F	55	62063	2 days	15.08.09	+	IV	-	120/80	78		-	-	+	+	AW MI	-	-		EF-	No	-	-	-	+	+	-	+			yes
83	Devagi	F	52	63145	12 hrs	15.08.09	+	I	-	130/80	72		-	-	-	-	IW MI	-	-		EF 46%	Yes	Mild	-	-	-	-	-	-	Stable	Stable	

S.No.	Name	Sex	Age	IP No	Date of onset of ACS	Time of Admission	Clinical status on Admission			BP	HR	RR	BBB		Bifasicular block	Complete heart block	MI Location	CVS History	CVS Risk Factors	Investigations		Thrombolysis	Ventricular Dysfunction	Arrythmias	Recurrent ischemia/ Angina	CHF	Heart Block	Mechanical Complications	Cardiac Arrest	At the time of discharge	At the end of 1 month	Death
							Chest pain	Killip Class	Arrythmias				RBBB	LBBB						CK-MB	ECHO											
84	Ganesan	M	53	62990	6 hrs	15.08.09	+	I	-	120/80	72		-	+	-	-	AW MI	Previous IW MI			EF 32%	Yes	Severe	-	+	-	-	-	-	Stable	Stable	
85	Kandasamy	M	51	63720	2 hrs	17.08.09	+	II	-	160/90	80	N	-	-	-	-	AW MI	-	DM/Smoker/ Alcoholic		EF 45% Gr.I DD	Yes	Moderate	-	-	-	-	-	-	Stable	Stable	
86	Ellammal	F	75	63344	4 hrs	17.08.09	+	III	-	110/80	100		-	-	-	-	AW MI	-	HT		EF 37% Grade I DD	Yes	Moderate	-	-	-	-	-	-	Stable	Stable	
87	Ramamurthy	M	60	63513	UA	17.08.09	+	I	-	160/100	74		-	-	-	-	UA	-	HT		EF 47% Grade I DD	Yes	Mild	-	-	-	-	-	-	Stable	Stable	
88	Jeyamani	M	50	64015	8 hrs	18.08.09	+	I	-	130/80	81		-	-	-	-	AW MI	-	Smoker/ Alcoholic		EF 44% Grade I	Yes	Moderate	-	-	-	-	-	-	Stable	Stable	
89	Murugesan	M	64	63976	3 hrs	18.08.09	-	I	-	130/80	45		+	-	-	-	IW MI RV MI	-	-		EF 50% adequate	Yes	Mild	-	-	-	-	-	-	Stable	Stable	
90	Manimaran	M	47	64001	2 days	18.08.09	-	I	-	110/80	100		-	-	-	-	AW MI	-	DM/Smoker		EF 40% Grade I	No	Moderate	-	-	-	-	-	-	Stable	Stable	
91	Sankar	M	38	63812	6 hrs	18.08.09	+	I	-	140/100	84	N	-	-	-	-	AW MI	-	Smoker		EF 47%	Yes	Mild	-	-	-	-	-	-	Stable	Stable	
92	Rappai	M	58	64189	3 hrs	18.08.09	+	I	-	110/70	68	N	-	-	-	-	IW MI	-	HT/DM		EF 46% Gr.I DD	Yes	Mild	-	-	-	-	-	-	Stable	Stable	
93	Deraj	M	52	62085	4 hrs	19.08.09	+	I	-	130/70	60	N	-	-	-	-	AW MI	-	-		EF 47% No DD	Yes	Mild	-	-	-	-	-	-	Stable	Stable	
94	Govindaraj	M	85	64345	6 hrs	19.08.09	+	I	-	100/70	72	N	-	-	-	-	IW MI RV MI	-	-		EF 42% Grade I	Yes	Moderate	+	-	-	-	-	+	Stable	Stable	
95	Varadarajan	M	45	65196	2 days	20.08.09	+	I	-	100/70	70	N	-	-	-	-	IW MI	-	-	558	EF 33% Grade II	No	Severe	-	-	-	1 2nd degr	-	-	Stable	Stable	

S.No.	Name	Sex	Age	IP No	Date of onset of ACS	Time of Admission	Clinical status on Admission			BP	HR	RR	BBB		Bifasicular block	Complete heart block	MI Location	CVS History	CVS Risk Factors	Investigations		Thrombolysis	Ventricular Dysfunction	Arrythmias	Recurrent ischemia/ Angina	CHF	Heart Block	Mechanical Complications	Cardiac Arrest	At the time of discharge	At the end of 1 month	Death
							Chest pain	Killip Class	Arrythmias				RBBB	LBBB						CK-MB	ECHO											
96	Damodaran	M	58	65149	8 hrs	23.08.09	+	I	-	100/70	42	N	-	-	-	+	AW MI	-	HT	644	EF 30% Grade II	Yes	Severe	-	-	-	-	-	-	Stable	Stable	
97	Arumugam	M	62	65667	4 hrs	24.08.09	-	II	-	140/80	100	N	-	-	-	-	AW MI PW MI	-	DM/HT		EF 38% Grade I DD	Yes	Moderate	-	-	+	-	-	-	Stable	Stable	
98	Iruthayaraj	M	48	65636	24 hrs	24.08.09	-	II	-	150/100	80		+	-	-	-	IW,PW,M	-	Smoker/ Alcoholic		EF 35% Grade I DD	No	Moderate	-	-	-	-	-	-	Stable	Stable	
99	Periyasamy	M	50	65609	>12 hrs	24.08.09	+	I	-	100/70	100	N	-	-	-	-	AW MI	-	Smoker		EF 43% Grade I DD	No	Moderate	-	-	-	-	-	-	Stable	Stable	
100	Subramani	M	58	65969	<6 hrs	25.08.09	+	I	-	130/90	80	N	-	-	-	-	IW,RV,PV	-	-		EF 42% Gr.I DD	Yes	Moderate	-	-	-	-	-	-	Stable	Stable	
101	Mohandass	M	52	65902	<6 hrs	25.08.09	+	I	-	110/70	70	N	-	-	-	-	IW MI	-	HT		EF 44%Gr.I DD	Yes	Moderate	-	-	-	-	-	-	Stable	Stable	
102	Deenadayalan	M	42	65765	10 hrs	25.08.09	+	III	-	110/70	90	N	-	+	-	-	AW MI	Previous IW MI	DM/Smoker		EF 32% Gr.III DD	Yes	Severe	-	+	+	-	-	-	Stable	Stable	
103	Babu	M	65	65959	<6 hrs	25.08.09	+	I	-	120/70	60	N	-	-	-	-	IW, PW	Previous IW MI	DM		EF 38% Grade I DD	Yes	Moderate	-	+	-	-	-	-	Stable	Stable	
104	Munirathinam	M	60	64918	<6 hrs	21.08.09	+	III	-	90/70	60	N	-	-	+	-	AW MI	-	-		-	Yes	-	-	-	-	+	-	+			yes
105	Babu Raj	M	65	65926	6 hrs	25.08.09	+	I	-	120/70	80	N	-	-	-	+	IW, PW	Previous IF	DM	25	EF 32% Gr.II DD	Yes	Severe	-	+	-	Yes	-	-	Stable	Stable	
106	soundarajan	M	55	65965	>12 hrs	25.08.09	+	III	-	120/90	110	N	-	-	-	-	AW MI	-	-		EF 35% Gr.II DD	No	Severe	-	-	+	-	-	-	Stable	Stable	
107	Kuppa padayatc	M	51	66604	UA	27.08.09	+	I	-	110/70	70	N	-	-	-	-	AW MI	PTCA +	-		EF 38% Grade I DD	No	Moderate	-	+	-	-	-	-	Stable	Stable	

S.No.	Name	Sex	Age	IP No	Date of onset of ACS	Time of Admission	Clinical status on Admission			BP	HR	RR	BBB		Bifasicular block	Complete heart block	MI Location	CVS History	CVS Risk Factors	Investigations		Thrombolysis	Ventricular Dysfunction	Arrythmias	Recurrent ischemia/ Angina	CHF	Heart Block	Mechanical Complications	Cardiac Arrest	At the time of discharge	At the end of 1 month	Death
							Chest pain	Killip Class	Arrythmias				RBBB	LBBB						CK-MB	ECHO											
108	Thamimkhan	M	35	66387	2 hrs	27.08.09	+	I	-	150/100	80	N	-	-	-	-	IW MI	-	Smoker		EF 52% No DD	Yes	Mild	-	-	-	-	-	-	Stable	Stable	
109	Pari	M	52	66284	<6 hrs	26.08.09	+	I	-	130/80	80	N	-	-	-	-	AW	-	-		EF 47% No DD	Yes	Mild	-	-	-	-	-	-	Stable	Stable	
110	Parthasarathy	M	53	66232	6 hrs	28.08.09	+	I	-	130/90	70	N	-	-	-	-	IW	-	Dyslipidemia		EF 47% Gr.I DD	Yes	Mild	-	-	-	2° AV	-	-	Stable	Stable	
111	Datchanamurthy	M	55	66757	5 hrs	28.08.09	+	I	-	170/100	80	N	-	-	-	-	AW	-	HT/DM	70	EF 42% Gr.I DD	Yes	Moderate	-	-	-	-	-	-	Stable	Stable	
112	Jeyakumar	M	53	56819	UA	28.08.09	+	I	-	120/90	70	N	-	-	-	-	UA	-	Smoker		EF 48% No DD	No	Adequate	-	-	-	-	-	-	Stable	Stable	
113	Joyce	F	75	66893	12 hrs	28.08.09	-	III	-	110/70	100	N	+	-	-	-	AW	-	DM/HT		EF 38% Gr.II DD	Yes	Moderate	-	-	+	-	-	-	Stable	Stable	
114	Sulthan	M	46	66933	<6 hrs	28.08.09	+	II	-	120/80	84	N	-	+	-	-	IW	-	-		EF 35% Gr.II DD	Yes	Moderate	-	-	-	-	-	-	Stable	Stable	
115	Jeyaraman	M	50	66986	6 hrs	29.08.09	+	I	-	100/70	76	N	-	-	-	-	IW, RV	-	HT/Smoker		EF 40% Gr.I DD	No	Mild	-	-	-	-	-	-	Stable	Stable	
116	Ruckmani	F	65	67092	4 hrs	29.08.09	+	I	-	100/70	90	N	-	-	-	-	AW	-	DM/HT		EF 45% Gr.I DD	Yes	Moderate	-	-	-	-	-	-	Stable	Stable	
117	Rathinam	M	70	66976	2 - 3 Days	29.08.09	+	IV	-	120/80	110	N	+	-	-	-	AW, IW	-	-		-	No	-	-	-	+	-	-	+	died		yes
118	Loganathan	M	48	67118	24 hrs	29.08.09	+	I	-	100/70	86	N	-	-	-	-	AW	-	DM/Smoker		EF 44% Gr.I DD	Yes	Moderate	-	-	-	-	-	-	Stable	Stable	
119	Jagadesan	M	30	67670	<6 hrs	31.08.09	+	I	-	150/100	74	N	-	-	-	-	AW	-	-		EF 47% No DD	Yes	Mild	-	-	-	-	-	-	Stable	Stable	

S.No.	Name	Sex	Age	IP No	Date of onset of ACS	Time of Admission	Clinical status on Admission			BP	HR	RR	BBB		Bifasicular block	Complete heart block	MI Location	CVS History	CVS Risk Factors	Investigations		Thrombolysis	Ventricular Dysfunction	Arrythmias	Recurrent ischemia/ Angina	CHF	Heart Block	Mechanical Complications	Cardiac Arrest	At the time of discharge	At the end of 1 month	Death
							Chest pain	Killip Class	Arrythmias				RBBB	LBBB						CK-MB	ECHO											
120	Elizabeth	F	55	67674	18 hrs	31.08.09	-	III	-	80/60	36		+	-	-	+	AW	-	DM/HT		-	No	-	-	-	-	-	-	+	Death in Hospital		
121	Saraswathy	F	65	67525	5 hrs	31.08.09	+	I	-	140/90	80	N	-	-	-	-	AW	-	-		EF 47%Gr.I DD	Yes	Mild	-	-	-	-	-	-	Stable	Stable	
122	Rajendiran	M	43	67612	2½ hrs	31.08.09	+	I	-	150/100	80	N	-	-	-	-	AW	Previous MI	DM/Smoker Alcoholic		EF 45% Gr.I DD	Yes	Moderate	-	+	-	-	-	-	Stable	Stable	
123	Mariyal	F	65	68280	6 hrs	02.09.09	+	I	-	140/90	80	N	-	-	-	-	IW,RV,PV	revious CV	-		EF 46% Gr.I DD	Yes	Mild	-	-	-	-	-	-	Stable	Stable	
124	Rani	F	49	68302	3 hrs	02.09.09	+	II	-	110/80	60	N	+	-	-	-	IW,RV,PV	-	-		EF 40% Gr.I DD	Yes	Moderate	-	-	-	-	-	-	Stable	Stable	
125	Sathya	M	43	68305	UA	31.08.09	+	I	-	110/70	77	N	-	-	-	-	UA	-	DM/Smoker		EF 47% No DD	No	Mild	-	-	-	-	-	-	Stable	Stable	
126	Rosi Nardes	M	75	68608	3 days	03.09.09	-	I	-	100/70	46	N	-	-	-	+	IW	-	-		EF 32% Gr.III DD	No	Severe	-	-	-	-	-	-	Stable	Stable	
127	Janakiammal	F	85	68690	UA	10.09.09	+	I	-	100/70	80	N	-	-	-	-	UA	-	DM		EF 47% Gr.I DD	No	Mild	-	-	+	-	-	-	Stable	Stable	
128	Munusamy	M	37	70097	6 hrs	09.09.09	+	I	Junctional Rhythms	130/70	76	N	-	-	-	-	AW	-	-		EF 47% Gr.I DD	Yes	Mild	-	-	-	-	-	-	Stable	Stable	
129	Shanmugam	M	60	70591	2½ hrs	10.09.09	+	I	-	140/70	72	N	-	-	-	-	IW,PW, RV, MI	-	-		EF 52% No DD	Yes	Adequate	-	-	-	-	-	-	Stable	Stable	
130	Ramadoss	M	52	70694	1 day	10.09.09	+	I	-	140/100	80	N	-	-	-	-	AW	-	Smoker/ Alcoholic		EF 45% Gr.I DD	No	Moderate	-	-	-	-	-	-	Stable	Stable	
131	Subramani	M	73	70386	6 hrs	09.09.09	-	I	-	120/80	70	N	-	-	-	-	AW	-	-		EF 48% No DD	Yes	Mild	-	-	-	-	-	-	Stable	Stable	

S.No.	Name	Sex	Age	IP No	Date of onset of ACS	Time of Admission	Clinical status on Admission			BP	HR	RR	BBB		Bifasicular block	Complete heart block	MI Location	CVS History	CVS Risk Factors	Investigations		Thrombolysis	Ventricular Dysfunction	Arrythmias	Recurrent ischemia/ Angina	CHF	Heart Block	Mechanical Complications	Cardiac Arrest	At the time of discharge	At the end of 1 month	Death
							Chest pain	Killip Class	Arrythmias				RBBB	LBBB						CK-MB	ECHO											
132	Arumugam	M	82	70708	7 hrs	10.09.09	+	I	-	120/70	80	N	-	-	-	-	IW, RV	-	Smoker/ Alcoholic		EF 42% Gr.I DD	Yes	Moderate	-	-	-	-	-	-	Stable	Stable	
133	Safiabegam	F	65	71067	6 hrs	11.09.09	+	I	-	140/80	70	N	-	-	-	-	AW	-	DM/HT		EF 46% Gr.I DD	Yes	Mild	-	-	-	-	-	-	Stable	Stable	
134	Ismail	M	33	71942	34 hrs	14.09.09	+	I	-	110/70	70	N	-	-	+	-	AW	-	-		EF 34% Gr.II DD	No	Severe	-	-	-	-	-	-	Stable	Stable	
135	Sarogammal	F	75	72541	7 hrs	16.09.09	+	II	-	110/80	90	N	+	-	-	-	AW	-	DM/HT		EF 42% Gr.I DD	Yes	Moderate	-	-	-	-	-	-	Stable	Stable	
136	Mukram	M	65	72400	UA	16.09.09	+	I	-	130/70	80	N	-	-	-	-	AW	-	-		EF 55% No DD	No	Adequate	-	-	-	-	-	-	Stable	Stable	
137	Gunalan	M	54	72291	4 hrs	16.09.09	+	I	-	150/80	80	N	-	-	-	-	IW, PW	-	Dyslipidemia		EF 47% Gr.I DD	Yes	Mild	-	-	-	-	-	-	Stable	Stable	
138	Suresh	M	49	72226	3 hrs	15.09.09	+	III	-	100/70	130	N	+	-	-	-	AW	-	-		-	Yes	-	VT/VF	-	+	-	-	+	Death in Hospital		
139	Manjula	F	48	72621	3 hrs	17.09.09	+	I	-	120/80	70	N	-	-	-	-	AW	-	DM/ Hypothyroid		EF 47% Gr.I DD	Yes	Mild	-	-	-	-	-	-	Stable	Stable	
140	Kuppamuthu	M	40	72681	4 hrs	17.09.09	+	I	VPC+	130/70	100	N	-	-	-	-	AW	-	DM/HT/ Smoker Alcoholic		EF 42% Gr.I DD	Yes	Mild	-	-	-	-	-	-	Stable	Stable	
141	Valliammal	F	70	73040	UA	18.09.09	+	II	-	150/70	80	N	-	-	-	-	IW	-	-		EF 43% Gr.I DD	No	Moderate	-	-	+	-	-	-	Stable	Stable	
142	Munnasingh	M	29	73053	2 hrs	18.09.09	+	I	-	130/80	56	N	-	-	-	-	IW	-	Smoker/ Alcoholic		EF 50% No DD	Yes	Mild	-	-	-	-	-	-	Stable	Stable	
143	Chandran	M	42	74184	8 hrs	21.09.09	+	II	-	160/80	100	N	-	-	-	-	AW	-	-		-	Yes	Moderate	-	-	+	-	-	-	Stable	Stable	

S.No.	Name	Sex	Age	IP No	Date of onset of ACS	Time of Admission	Clinical status on Admission			BP	HR	RR	BBB		Bifasicular block	Complete heart block	MI Location	CVS History	CVS Risk Factors	Investigations		Thrombolysis	Ventricular Dysfunction	Arrythmias	Recurrent ischemia/ Angina	CHF	Heart Block	Mechanical Complications	Cardiac Arrest	At the time of discharge	At the end of 1 month	Death
							Chest pain	Killip Class	Arrythmias				RBBB	LBBB						CK-MB	ECHO											
144	Devagiammal	F	80	73458	8 hrs	20.09.09	-	I	-	100/80	40		-	-	-	+	IW, RV	-	HT/DM		-	Yes	-	-	-	-	+	-	-	Death in Hospital		
145	Saraswathy	F	70	73440	12 hrs	20.09.09	-	III	-	80/60	110	N	-	-	-	-	AW	-	-		-	No	-	-	-	-	-	+	+	Death in Hospital		
146	Lakshmikanthar	F	60	74044	10 hrs	22.09.09	+	IV	AF	60/?	100	N	-	-	-	-	IW	revious CV	HT		-	Yes	-	-	-	-	-	-	+	Death in Hospital		
147	Valihussain	M	70	73808	7 hrs	21.09.09	+	I	-	130/90	60	N	-	-	-	-	IW	-	-		EF 42% Gr.I DD	Yes	Moderate	-	-	-	-	-	-	Stable	Stable	
148	Ramamurthy	M	54	74143	UA	21.09.09	+	I	-	160/90	80	N	-	-	-	-	AW	-	HT		EF 47% No DD	No	Mild	-	-	-	-	-	-	Stable	Stable	
149	Raj Mohan	M	48	74033	2 hrs	22.09.09	+	I	-	150/100	100	N	-	-	-	-	AW	-	-		EF 54% No DD	Yes	Adequate	-	-	-	-	-	-	Stable	Stable	
150	Chandran	M	42	74184	12 hrs	22.09.09	+	II	-	160/90	100	N	-	-	-	-	AW	-	-		EF 40% Gr.I DD	Yes	Moderate	-	-	-	-	-	-	Stable	Stable	