STUDY OF CLINICAL PROFILE OF 50 PATIENTS WITH ACUTE INFERIOR WALL MYOCARDIAL INFARCTION

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CERTIFICATE

This is to certify that the dissertation titled **"STUDY OF CLINICAL PROFILE OF 50 PATIENTS WITH ACUTE INFERIOR WALL MYOCARDIAL INFARCTION"** is the bonafide original work of **DR. K.BABU,** in partial fulfillment of the requirements for M.D. Branch – I (General Medicine) Examination of the Tamilnadu DR. M.G.R Medical University to be held in MARCH 2008. The Period of study was from December 2006 to July 2007.

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DECLARATION

I, **DR. K. BABU,** solemnly declare that dissertation titled **"STUDY OF CLINICAL PROFILE OF 50 PATIENTS WITH ACUTE INFERIOR WALL MYOCARDIAL INFARCTION"** is a bonafide record of work done by me in the Department of Internal Medicine, Government Stanley Medical College and Hospital during December 2006 to July 2007 under the guidance of **Prof. A. K. GEETHADEVI, M.D.,** Professor of Medicine, Government Stanley Medical College and Hospital, Chennai.

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CONTENTS

INTRODUCTION

Myocardial infarction is the term used when the myocardium is necrosed due to ischemia. It may be transmural or subendocardial.

Patients with ischaemic heart disease fall into two large groups. Patients with stable angina and patients with acute coronary syndromes (ACS). ACS group in turn is composed of patients with acute myocardial infarction with ST elevation (STEMI), those with unstable angina (UA) and non ST segment elevation MI (NSTEMI).

Inferior wall infarction has got some special features like association with right ventricular infarction and bradyarrhythmias especially sinus bradycardia and second degree AV block.

Right Ventricular infarction is different from that of the left ventricle in the acute presentation, therapy and long term prognosis.

The early recognition of RVI is important, because the time of onset of its haemodynamic consequence is unpredictable and these may be prevented by the administration of intravenous fluid load.

The description of RVI appeared more than 60 years ago. But it was considered unimportant until Cohn and Co-workers in 1974 published their classic report on RVI as a distinct clinical entity. The reported incidence is between 25 – 50% of IWMI.

Involvement of RV is related to atherosclerotic occlusion of the right coronary artery and is associated with involvement of postero-inferior wall and posterior portion of the septum.

Clinically RVI can be suspected when a patient with IWMI presents with elevated JVP, positive Kussmaul's sign, hypotension, right sided third or fourth heart sounds, tender hepatomegaly, oliguria, rarely tricuspid regurgitation and clear chest.

Electrocardiogram was generally believed to be unhelpful in identifying RVI, until Erhardt and co-workers described the value of a right precordial lead in patients with autopsy proved RVI. A 1 mm ST elevation in this lead is 93% sensitive and 95% specific. The change is transient. In one series, 48% of the patients had resolution of ECG changes within 10 hours of the onset of symptoms.

AIM OF THE STUDY

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- To study the clinical profile of 50 serial cases of Acute Inferior Wall Myocardial Infarction with Right Ventricular Infarction and to analyse the age and sex distribution, symptomatology, clinical features, complications and outcome.

REVIEW OF THE LITERATURE

REVIEW OF THE LITERATURE

William Harvey, in 1667, was perhaps the first to report postmortem findings correlated with clinical description in a patient with ischemic heart disease. His subject, a middle aged man, had numerous episodes of "distressing pain in the chest" and died during one of them. The autopsy revealed rupture of left ventricle. A somewhat similar case was reported by Thomas Wills in 1674. Morgagni, in his de sidibus et causes morburum, published in 1761 cited an autopsy performed and described the left coronary artery of a patient who had experienced a speedy death, appeared to have changed into a bony canal.

Heberden's description of angina pectoris in 1772, may be considered as the beginning of modern period in the history of Coronary Artery Diseases.

In 1842, Marshall Hall emphasized that experimental obstruction of a coronary artery was soon followed by death. James B. Herrick in 1912, described the clinical picture of obstruction of coronary arteries.

Initially the diagnosis of right ventricular infarction was at biopsy. In 1948, Wartman and Hallerstein, described 22 instances of RVI out of 164 cases of myocardial infarction they autopsied. In 1959, Wade described 19 patients with right ventricular infarction.

Even though the description of right ventricular infarction appeared more than 60 years ago, it was not considered as an important entity, in large part because of studies in animals in which experimentally induced isolated right ventricular damage caused no substantial change in systemic venous pressure, pulmonary pressure or cardiac output.

In 1974, Cohn[1] and co-workers published their classic report on right ventricular infarction as a distinct clinical entity. Since then RVMI has been recognized more frequently and continues to be a diagnostic and therapeutic challenge.

Typically right ventricular infarction occurs with occlusion of RCA proximal to acute marginal branches[2,3,4]. Occasionally, occlusion of a dominant left circumflex artery may also produce RVMI[5]. LAD occlusion may result in the infarction of right ventricle.

Isolated right ventricular infarction accounts for less than 2% of all cases of infarction, but may result in considerable morbidity. It is associated with:

1. Isolated occlusion of right ventricular branches.
2. Occlusion of non-dominant right coronary artery[6] and
3. Occlusion of dominant right coronary artery with good collateral flow to posterior descending artery. But it may occur in the absence of coronary artery disease, when substantial right ventricular hypertrophy is present (e.g. COPD, PS, Pulmonary Hypertension, etc.,)[7,8]

Recognition of the syndrome of RVMI is important as it identified a significant clinical entity, which is associated with considerable morbidity and mortality.

Ischemia or infarction of right ventricle results in decreased right ventricular compliance, reduced filling and decreased RV stroke volume, in turn leading to diminished left ventricular filling and drop in cardiac output that results in systemic hypotension and shock[9,10].

Ischemic injury can produce conduction block at any level of the AV or intraventricular conducting system[11,12].

First degree AV block occurs in less than 15 percent of patients with acute myocardial infarction admitted to Coronary Care Units.

Mobitz type I block occurs in up to 10% of acute myocardial infarction. It occurs more commonly in inferior than anterior myocardial infarction. Mobitz type II block is rare following acute myocardial infarction accounting for 10% of all cases of second-degree heart block. Complete heart block may occur in 5 – 15 percent of patients with either anterior or inferior wall myocardial infarction. The incidence is higher in patients with right ventricular infarction. Bundle branch blocks are reported in 5 – 10 percent of patients with acute myocardial infarction. The usual setting will be, therefore, the patient that presents with an acute IWMI. 30 – 50% of these patients may have some RV involvement although it will only be hemodynamically significant in a minority. Anatomic evidence of RVMI is more common than expected haemodynamic pattern.

ANATOMY OF CORONARY CIRCULATION

The coronary arteries, the right and the left are the first branches of the aorta[13]. The left coronary artery divided into the left anterior descending and left circumflex arteries. Branches of the left anterior descending artery supplies the free wall of the left ventricle adjacent to interventricular septum. The left circumflex artery supplies the left atrium and parts of posterior and lateral wall of the left ventricle. In 35% of cases this artery supplies the S.A. node and in 20% of cases the A.V. node as well.

The right coronary artery supplies the whole of right ventricle, variable parts of diaphragmatic aspect of left ventricle, the posterio-inferior third of the interventricular septum, the right atrium and the conducting systems as far as proximal parts of right and left bundle branches. Right coronary artery supplies AV node in 80% and SA node in 65% of cases. In 50% of cases right atrium is supplied by right coronary artery and the remaining have dual supply. The left atrium is supplied by the left coronary artery in 62% and right coronary artery in 27% of cases, and in 11% equal contribution from both.

The venous blood from heart is drained into right atrium by the coronary sinus, thebesian veins and the anterior cardiac veins.

Atheromatous occlusion of right coronary artery is as common as that of left coronary artery.

ANATOMICO-PATHOLOGICAL CORRELATION

RVMI occurs due to proximal occlusion of the dominant right coronary artery.

Rarely isolated occlusion of right ventricular branches of right coronary artery can produce infarction of anterolateral aspects of right ventricle alone.

Occlusion of posterior descending artery produces infarction of posterior wall of right ventricle associated with infarction of posteroinferior wall of left ventricle and posterior part of interventricular septum.

In 90% of cases posterior descending artery is a branch of right coronary artery. Therefore, if the collateral to posterior descending artery is poor and right coronary artery is the dominant artery, occlusion of main stem of right coronary artery produces infarction of both anterolateral and inferior aspects of right ventricle and if those territories supplied by posterior descending artery are good, its territory may escape. In 10% of cases posterior descending artery is an extension of left circumflex artery also called left dominance. In this situation, infarction of posterior wall of right ventricle can occur with a normal right coronary artery.

Thus, right ventricular infarction is usually associated with infarction of one or the other wall of left ventricle. Right ventricle infarction can occur alone in the following situations.

1. Isolated occlusion of right ventricular branches.
2. Occlusion of Non-dominant right coronary artery and
3. Occlusion of dominant right coronary artery with good collateral flow to posterior descending artery.

In nearly 65% of population the sino atrial node artery arises from right coronary artery and supplies sino atrial node and right atrium. Occlusion of right coronary artery before origin of this branch is likely to cause ischemic damage to right atrium and SA node. The symptoms of RVMI may be more pronounced in the presence of combined right atrial infarction with associated rate and rhythm disturbances[14]. Atrioventricular node artery is a branch of posterior descending artery. Ischemic dysfunction of this node therefore, occurs only when blood flow in posterior descending artery is compromised. In cases of occlusion of right coronary artery. A.V. node can escape if there is good collateral flow to the posterior descending artery or if this vessel is arising from circumflex artery.

PATHOPHYSIOLOGY OF RVMI

- The right ventricle has the same cardiac output as the left
- RV is anatomically and physiologically designed to serve the low pressure pulmonary circulation
- Muscle mass of RV is only 15% of LV
- RV stroke work is 25% of LV

- Pulmonary vascular resistance is 10% of systemic vascular resistance
- Coronary blood flow occurs in both systole and diastole in the right ventricle in the absence of right ventricular hypertrophy.
- The main blood vessel is the right coronary artery (serves the lateral wall, the posterior wall and posterior interventricular septum by the posterior descending artery).
- The anterior wall is supplied by the conus artery and the LAD
- The RV is like a pocket, wrapped around the LV, sharing the interventricular septum and pericardium.
- The lower afterload and myocardial oxygen demand of the right ventricle (as compared with the left) explains its lower oxygen extraction at rest and its relative resistance to irreversible ischemic damage during right coronary occlusion.
- The increased incidence of right ventricular infarction in patients without a history of preinfarction angina may result from a lack of adequately formed collateral vessels.
- Right ventricular infarction complicates 30 – 50% of inferior wall myocardial infarction.
- The most reliable ECG findings is ST segment elevation in the right precordial leads, particularly V_4R with associated ST segment elevation in II,III and aV_F.

- Clinical signs are: high central venous pressure, clear lung fields and systemic hypotension.
- With a large infarction, the RV may, essentially, become a conduit from the systemic veins to the pulmonary circulation.
- Elevation of right atrial pressure secondary to right ventricular infarction has been noted to serve as a stimulus for secretion of atrial natriuretic factor. Increased ANF can be detrimental to normal LV filling pressure by virtue of its potent vasodilating, natriuretic, diuretic and aldosterone inhibiting properties[15].
- Management is centered on volume loading and the early use of inotropes (dobutamine) to maintain blood flow to the LV (preload) and to maximize cardiac output.
- Right ventricular infarction occurs when there is an occlusion of the right coronary artery proximal to the acute marginal branches, but it may also occur with an occlusion of the left circumflex artery in patients who have left dominant coronary circulation.
- RVI less commonly may occur as a result of occlusion of the LAD.
- Haemodynamic insufficiency in the presence of inferior wall myocardial infarction suggests additional right ventricular infarction.
- Isolated RVI is extremely rare – 2% of autopsies.

HAEMODYNAMICS

- Ischemia or infarction of the right ventricle results in decreased right ventricular compliance, reduced filling and decreased right ventricular stroke volume; raised right atrial pressure.
- These changes lead to diminished left ventricular filling and drop in cardiac output.
- In addition, acute right ventricular dilatation causes a leftward shift of interventricular septum, increasing left ventricular end diastolic pressure with a decrease in left ventricular compliance and cardiac output[16,17].
- Brookes et al[18] demonstrated that the geometric changes in the left ventricle, caused by right ventricular dilatation due to RVMI, resulted in significant impairment of left ventricular contractile function in addition to the diastolic filling abnormalities and changes in compliance.
- Therefore, although the patient has clinical signs of increased right-sided pressure, the left ventricular filling and systolic function may be below normal.
- Because of the pathophysiology of right ventricular infarction, its management differs substantially from the routine management of LV infarction.

- Since haemodynamically important right ventricular infarction typically occurs in patients with an acute inferior wall MI, suspicion is warranted in any patient presenting with such an infarction.

Clinical Diagnosis

- The clinical triad[19,20] of hypotension, clear lung fields and elevated jugular venous pressure in a patient with an inferior wall myocardial infarction is virtually pathognomonic for right ventricular infarction.
- Caution must be exercised in relying on such findings, since they are readily masked by volume depletion and because the physical and haemodynamic signs of right ventricular infarction often emerge only after volume loading.
- Pulses paradoxus and Kussmaul's sign[21] (inspiratory increase in JVP) have been reported in RVMI.
- The presence of elevated JVP and Kussmaul's sign in the setting of acute inferior wall MI indicate a haemodynamically significant RVMI (sensitivity 88% and specificity 100%)
- Patients with intact right atrial perfusion manifest augmented atrial contraction resulting in enhanced 'a' wave and 'y' descent.
- Finding of diminished 'a' wave in patients with haemodynamically important RVMI, depressed right atrial function, right atrial infarction has been proved to be a bad prognostic indicator.

- Auscultation may reveal a right sided S_3 and S_4.
- Tricuspid regurgitation[22], secondary to right ventricular chamber dilatation, may be severe when related to papillary muscle dysfunction.

INVESTIGATIONS

ECG changes of Inferior wall infarction with Right Ventricular infarction

It has been shown that right ventricular infarction can be diagnosed accurately from surface ECG. The electrocardiographic diagnosis of right ventricular infarction is usually based on the manifestation of an elevated ST segment in the extreme right leads V_3R and V_4R.

Right ventricular infarction should be strongly suspected if, in the setting of acute inferior wall infarction, there is ST segment elevation of 1 mm or more in lead V1, lead V_4R or any one of the extra right precordial leads, V_4R-V_6R.

According to Braat et al[23,24] an ST segment elevation of more than or equal to 1 mm in lead V_4R in patients with inferior wall infarction, had a sensitivity of 93%, specificity of 95% in the diagnosis of right ventricular infarction.

The ST segment which is higher in lead V_4R than in lead V1 and V_3R offer the highest specificity and efficiency in diagnosis of RVMI. The ST segment elevation in right precordial lead is transient and changes disappeared within 10 – 18 hours after the onset of chest pain in 50% of the patients.

ST segment elevation in V_4R is a strong independent predictor[25,26,27] of major complications and in hospital mortality.

Reports of RVMI presenting as ST segment elevation in leads $V_{1,}V_2$, mimicking anterior wall MI[28,29] had been explained by Hurst. The two infarcts can be separated when vector concepts are used.

In RVMI, ST segment vector is directed anteriorly and is more than $+90^o$ to the right (producing a downward displacement of ST segment in lead I) while in case of anteroseptal left ventricular infarction, the vector is directed anteriorly but is usually locked from -30^o to -90^o to the left in frontal plane (producing ST segment elevation in lead I).

In one series 48% of the patients had resolution of ECG changes within 10 hrs of the onset of symptoms. Thus, it is imperative to record the ECG through the accessory right precordial leads as early as possible.

It is important to recognize the transient nature of ST segment elevation. A right precordial lead V_4R is the investigation of choice. ST segment elevation in lead V_4R remains the most predictive ECG finding for RVMI.

Right bundle branch block and complete heart block are the most frequent conduction abnormalities associated with RVMI. Rhythm disturbances such as atrial fibrillation[30], sinus bradycardia are also noted in ECG.

Use of 18 lead ECG in diagnosing RVMI and posterior wall MI in patients with acute inferior wall MI

Detection of RV and posterior wall involvement is important in patients with acute IWMI.

RV involvement is studied with right-sided leads V_4R, V_5R, V_6R and posterior wall is studied with $V_7 - V_9$ leads (V_7 left posterior axillary line, V_8 – inferior angle of left scapula V_9 – Left para vertebral area).

Posterior wall MI is also studied with mirror image changes found in V_1 V_2 – Tall 'R', with ST segment depression with upright 'T'.

Anderson[31] found that ST elevation in lead III greater than lead II had high sensitivity (88%) and specificity (91%) of diagnosing RVMI; using the principle that lead III was more oriented towards RV than LII..

Enzyme Study

Measurement of enzymes released to circulation by irreversibly damaged myocardial cells helps to detect AMI. Serum Creatine kinase (CK) level is the earliest to rise, detectable within4 – 8 hours after onset of AMI, reaches a peak in 24 hrs and declines to normal within 3– 4 days after the onset of pain. Of the three isoenzymes CK-MB remains the most useful test for myocardial necrosis.

The MB isoenzyme of CK has the advantage over total CK that it is not present in significant concentrations in extra cardiac tissue and therefore is more specific. However, cardiac surgery, myocarditis and electrical cardioversion often

result in elevated serum levels of MB isoenzyme. "CK – MB mass index" (which is a ratio of CK – MB mass: CK activity) greater than or equal to 2.5 is suggestive of myocardial damage.

Serum Markers for Myocardial Infarction

Cardiac Troponin – T (cTnT) and Cardiac Troponin I (cTnI). This is released to circulation when there is loss of integrity of myocardial cell membrane along with other proteins of cardiac contractile apparatus. The presence of cTnT in the serum has been found to be 100% sensitive for the diagnosis of AMI. It has a long diagnostic window (it can be detected up to 12 days post infarction). So, it has a role for the late diagnosis of AMI. It is also useful monitoring response to reperfusion therapy.

Myoglobin

This is the first serum marker to increase in serum after AMI. But it is less specific for myocardium and increased levels can be present in muscular injury or renal disease.

It is rapidly excreted in the urine, so blood levels return to normal range within 24 hrs of onset of infarction.

Echocardiography

Two-dimensional echocardiography: Abnormal findings include right ventricular dilatation, right ventricular wall asynergy and abnormal interventricular septal motion caused by a reversal of the transseptal pressure gradient due to the increased right ventricular end-diastolic pressure.

The short axis view has been shown to have the highest sensitivity (82%), with a specificity ranging from 62 percent to 93 percent for haemodynamically important right ventricular infarction.

Interatrial septal bowing[32] toward the left atrium, indicative of an increased right atrial - left atrial pressure gradient, is an important prognostic marker in right ventricular infarction. Patients with this finding have more hypotension, more heart block and higher mortality than patients without it.

Doppler echocardiography is particularly helpful in detecting such complications of right ventricular infarction as tricuspid regurgitation, ventricular septal defect and premature opening of the pulmonary valve (which indicates a noncompliant right ventricle).

Other Imaging Modalities

Nuclear Imaging

Myocardial perfusion imaging with TL^{201} or ^{99m}TC – sestamibi, which are distributed in proportion to myocardial blood flow and concentrated by viable myocardium, reveal a defect (cold spot) in most patients during the first few hours after development of a transmural infarct.

- ❖ Although perfusion scanning is extremely sensitive, it cannot distinguish acute infarcts from chronic scars and thus not specific for diagnosis of acute MI.
- ❖ Radionuclide ventriculography carried out with ^{99m}TC labeled red blood cells, frequently demonstrates wall motion disorders and reduction in the ventricular ejection fraction in patients with ST elevation myocardial infarction. While of value in assessing the haemodynamic consequences of infarction and in aiding the diagnosis of RV infarction when the RV ejection fraction is depressed, this technique is nonspecific as many cardiac abnormalities other than MI alter the radionuclide ventriculogram.
- ❖ Radionuclide angiography, perfusion imaging, scintigraphy, positron emission tomography have been used to evaluate patients with ST elevation myocardial infarction.

Nuclear Imaging

- ❖ Assess infarct size
- ❖ Assess collateral flow
- ❖ Assess jeopardized myocardium
- ❖ Determines effect of infarct on ventricular function
- ❖ Establishes prognosis
- ❖ However the necessity of moving a critically ill patient from coronary care unit to the nuclear medicine department limits their practical application.

- ❖ Cardiac radionuclide imaging for diagnosing myocardial infarction should be restricted to special limited situations where history, ECG findings and serum cardiac markers are equivocal.

64 slice CT scanning:

It can do a full scan in 10 seconds and produce high resolution images. This allows non-invasive and early diagnosis of coronary artery disease. It allows direct visualization of not only the lumen of coronary arteries but also plaque within the artery.

Magnetic resonance imaging

- ➢ MRI helps in
    - ♦ Locating and sizing the area of infarction.
    - ♦ Assess severity of ischaemic insult
    - ♦ Assess perfusion of infarcted and non infarcted tissue as well as of reperfused myocardium.
    - ♦ To identify myocardial edema, fibrosis, wall thinning and hypertrophy.
    - ♦ Assess ventricular chamber size and segmental wall motion

Both MRI, CT scanning has limited practical application, because of the need to transport patient with myocardial infarction to radiological department.

MANAGEMENT

The mortality from AMI halved after the introduction of Coronary Care Units and about 40% of the fall in mortality is caused by such interventions as coronary care units, pre-hospital resuscitation and new mechanical and medical treatments of coronary artery disease.

Pain relief

Alleviation of pain is a critical factor in the case of patients with AMI and morphine is the drug of choice. Morphine should be used cautiously in RVMI.

Patients with inferior wall MI with RVMI usually may present with systemic hypotension. Volume loading is the mainstay of treatment.

Some patients, with severe right ventricular dilatation may not improve with volume loading.

Generally volume infusion should be done with saline or colloids in 200 ml increments in 5 – 10 minutes.

Volume expansion is continued until Hypotension is corrected or capillary wedge pressure reaches 15 – 20 mm of Hg or right atrial pressure exceeds 15 mm of Hg.

If cardiac output is not improved with carefully administered volume infusion, then dobutamine[33,34] should be administered to augment right ventricular systolic performance and improve forward cardiac output.

Left ventricular output is also improved with dobutamine. Inotropic agents can increase the oxygen demand of an already ischemic myocardium. But an

increase in stroke volume and cardiac index may offset any increase in myocardial oxygen demand, by an augmented myocardial blood flow.

Administration of crystalloids and/or colloids will result in maximizing myocardial muscle fibre stretch through volume expansion, up to certain physiologic limits, creates a greater contractile force, stronger systolic contraction and improved stroke volume result. This provides oxygenated blood to LV.

Management of arrhythmias improves survival rate. Inferior wall MI associated with RVMI are prone for bradyarrhythmias.

Bradyarrhythmias resulting in atrio ventricular dyssynchrony and loss of right atrial contribution may lead to severe haemodynamic consequences.

High degree AV blocks necessitate atrio ventricular sequential pacing[35,36] to maintain cardiac output.

In contrast to the conventional treatment of left ventricular infarction, inferior wall MI with RVMI, necessitate the avoidance of vasodilators, nitrates, diuretics.

Early reperfusion[37,38] of right coronary artery significantly improves right ventricular mechanical function and lowers in-hospital mortality in patient with inferior wall MI with RVMI.

Reperfusion has been successfully undertaken with angioplasty or thrombolytic agents.

Strategies in Treatment of Right ventricular Infarction

1. **Maintain Right Ventricular Preload**
    - ❖ Volume load e.g. Iv Saline / Colloids
    - ❖ Although volume loading increases RAP and PCWP, it does increase cardiac output.
    - ❖ Avoid nitrates, diuretics, morphine boluses (these decrease preload)
    - ❖ Maintain atrioventricular synchrony:
        - AV sequential pacing for complete heart block
        - Prompt cardioversion for atrial fibrillation
2. **Inotropic Support**
    - ❖ Dobutamine is the agent of choice, then Noradrenaline, dopamine
    - ❖ Dobutamine increases cardiac output, stroke volume index and RV ejection fraction, consequently unloading the right ventricle.
3. **Reducing Right ventricular after load**
    - ❖ Intraaortic balloon counterpulsation
    - ❖ Vasodilators (Sodium nitroprusside)
    - ❖ Caution: these may also decrease LV preload and thus cardiac output
4. **Reperfusion**
    - ❖ Thrombolytic agents
    - ❖ Direct angioplasty

5. Temporary pacing

Temporary pacing should be performed in patients with any of the following:

a. Sinus bradycardia unresponsive to drug therapy
b. Mobitz type II second degree atrioventricular block
c. Third – degree heart block
d. Bilateral bundle branch block
e. Newly acquired bundle branch block
f. Right or left bundle branch block in conjunction with first – degree atrioventricular block.

Immediate surgical intervention is often required when PTCA is unsuccessful and patients have persistent chest pain or haemodynamic instability. Patients known to have mechanical abnormality leading to severe pulmonary congestion or hypotension, such as papillary muscle rupture (with resultant mitral regurgitation) or ventricular septal defects are candidates for immediate emergency surgery.

AV block is a common complication of right ventricular infarction and usually requires temporary pacing. AV sequential pacing should be employed in patients with AV block or junctional rhythms.

COMPLICATIONS

1. Shock
2. 2^{nd} or 3^{rd} degree heart block – indicates a poor prognosis and occurs in as many as 48% of RVMI.
3. Atrial fibrillation
4. Ventricular arrythmias
5. Ventricular septal rupture – in patients with RVMI plus transmural posterior septal infarction.
6. Right ventricular thrombus formation and subsequent pulmonary embolism.
7. Tricuspid regurgitation
8. Pericarditis (due to the frequent transmural injury of the relatively thin walled right ventricle)
9. Right to left shunt through a patent foramen ovale should be suspected in patients who have hypoxemia that is not responsive to the administration of oxygen.

Various degrees of AV block is a common complication of right ventricular infarction and usually require temporary pacing. AV sequential pacing should be employed in patients with AV block or junctional rhythms.

PROGNOSIS

RVMI occurs in 25 – 53% patients with inferior wall infarction. The bedside diagnosis of RVI is based upon a combination of findings of right sided heart failure including a right ventricular third or fourth sounds, elevated jugular venous pressure with a steep 'y' descent, a positive Kussmaul's sign, arterial hypotension and clear lung by X-ray and physical examination. ST segment elevation of the right sided precordial electrocardiographic leads, particularly lead V_4R are present in the majority of patients.

Rarely RVI can present as tricuspid regurgitation and rupture of interventricular septum. Shock is observed only in cases with transmural infarction of right ventricle with additional involvement of two or more walls of left ventricle.

Right ventricular infarction with inferior wall infarction is more susceptible to AV block. Pulmonary embolism is said to be more common in RVMI. Exact evidence of this complication is not clear.

In patients with right ventricular infarction, complete reperfusion of the right coronary artery by angioplasty results in the dramatic recovery of right ventricular performance and an excellent clinical outcome. In contrast unsuccessful reperfusion is associated with impaired recovery of right ventricular function, persistent haemodynamic compromise, and a high mortality rate.

- When inferior wall myocardial infarction is complicated by right ventricular infarction, however, the in-hospital mortality may be as high as 31 percent, as compared with 6 percent of patients with inferior wall myocardial infarction and no right ventricular involvement.
- Several studies have found that right ventricular dysfunction after a myocardial infarction is an independent risk factor for higher long-term mortality.

In the vast majority of survivors of right ventricular infarction, manifestations of right ventricular dysfunction return to normal. Clinical and haemodynamic recovery eventually occurs even in patients whose right ventricular function was depressed for weeks or months. This returns to normal due to the amelioration of concomitant left ventricular dysfunction, resulting in a reduction in right ventricular afterload or to a gradual stretching of the pericardium with amelioration of its restraining effect.

POSTERIOR WALL MYOCARDIAL INFARCTION

Posterior wall myocardial infarction is also called dorsal wall myocardial infarction.

True posterior wall infarction is infarction of posterior wall of left ventricle, usually caused by occlusion of left circumflex coronary artery, but also may be caused by right coronary artery occlusion.

There is no conventional electrode which is oriented directly to the posterior wall of the heart. This is because an electrode so oriented would have too much tissue (bone and muscle) between it and the heart, thereby attenuating and even distorting the electrocardiographic deflections.

Percentage of true posterior wall myocardial infarction is difficult to assess, as criteria for diagnosis are less well recognized.

Reported incidence of posterior wall myocardial infarction was 15 – 21%, often accompanied with inferior and lateral wall myocardial infarction. Isolated dorsal wall myocardial infarction is very rare and the incidence varies between 1–3%.

Risk factors and clinical presentation are similar to other myocardial infarction. Patients with inferolateral myocardial infarction who also have posterior wall myocardial infarction have a large size infarct with increased risk of complications.

The right precordial leads V_1 – V_3 and especially V_2 are oriented to the anterior wall and reflect the inverse change or mirror image of the classical presentation of posterior wall myocardial infarction.

The mirror image of QS complex in posterior wall is reflected by tall and slightly widened ‘R’ wave.

The mirror image of coved and elevated ST is theoretically reflected by a depressed concave upwards ST segment. This is usually less seen.

Mirror image of inverted symmetrical ‘T’ wave is reflected by an upright, widened and usually tall ‘T’ wave in right precordial leads. This upright ‘T’ wave is an essential characteristic of PWMI and diagnosis should not be entertained without this change.

T_2 to T_6 index which is the difference between the voltages of T waves in leads V_2 and V_6, if $\geq$ 0.38 mV, then the probability of posterior myocardial infarction is more likely.

Dorsal leads $V_7 – V_9$ significantly increase the detection of PWMI compared to standard 12 lead ECG.

V_7 – Level of V_6 at left posterior axillary line

V_8 – Left side, tip of scapula

V_9 – Left Para spinal space

Including dorsal leads $V_7 – V_9$ increased the sensitivity and specificity of detecting dorsal myocardial infarction to 58% and 98% respectively.

Schimitt[39] et al found the sensitivity of the ECG diagnosis of acute posterior wall myocardial infarction was increased by 11% (50 – 61%) by using extended leads $V_7 – V_9$ after comparing the angiographic findings. They found a trend towards extended infarct size in those patients with concomitant ST segment elevation in additional ECG leads.

Kulkarni et al, in his studies had found patients with ST segment elevation in the inferior and posterior leads were likely to have left circumflex occlusion (85%) rather than right coronary artery.

Rich et al[40], in his studies had found the combination of T_2 to T_6 index and extended leads, increased the specificity of detecting posterior wall myocardial infarction to 94%.

Coronary reperfusion based on symptoms, ECG changes, lead to decrease in mortality and morbidity.

Brady et al[41] ECG criteria:

1. ST segment depression, concavity upwards in $V_1 – V_2$.
2. Prominent 'R' wave in V_1 V_2
3. R/S ratio > 1.0 in V_2.
4. Prominent upright 'T' wave in V_1, V_2
5. Posterior leads $V_7 – V_9$ showing ST segment elevation of $\geq$ 1 mm
6. T_2 to T_6 index $\geq$ 0.38 mV

MATERIALS AND METHODS

MATERIALS AND METHODS

This study was conducted during December 2006 to July 2007 period. 50 consecutive patients admitted to the coronary care unit with a diagnosis of acute inferior wall infarction were included in the study.

All patients included in the study were subjected to ECG examination of V_3R and V_4R in addition to the conventional 12 leads. In addition, all patients were subjected to ECG examination of extended leads V_7 to V_9. Rhythm strips were taken in patients with arrhythmias. ECGs were examined at the time of admission, second day and on the day of discharge.

Only those cases with hyperacute inferior wall infarction were included in the study. Patients with slope elevation of ST segment in leads, II, III and aV_F were taken as having hyperacute inferior wall infarction. Right ventricular infarction was diagnosed if there was ST elevation equal to or more than 1 mm in V_4R. Posterior wall myocardial infarction was diagnosed if there was ST segment elevation equal to or more than 1 mm in extended leads V_7 to V_9,with tall 'R', ST segment depression, upright 'T' in leads V_1,V_2.

All patients were assessed clinically and electrocardiographically with special emphasis on presenting complaints, risk factors, vital signs, arrhythmias and mortality. Patients were followed up till discharge.

Patients who presented after 24 hrs of onset of chest pain were excluded, as the ST changes in right ventricular infarction may be transient. Patients with history of chronic lung disease, previous MI, rheumatic heart disease, pericardial disease or LBBB were excluded because diagnosis of right ventricular infarction is not possible in these cases when ECG is used as the criteria.

RESULTS

OBSERVATIONS

The observations in 50 patients with acute inferior wall myocardial infarction is presented in this section in descriptive and tabular form.

AGE

Cases were divided in to groups of 5 years difference for comparing age and sex incidence.

Peak incidence was found in 2 groups, one group with mean age 42 and another with mean age 62.

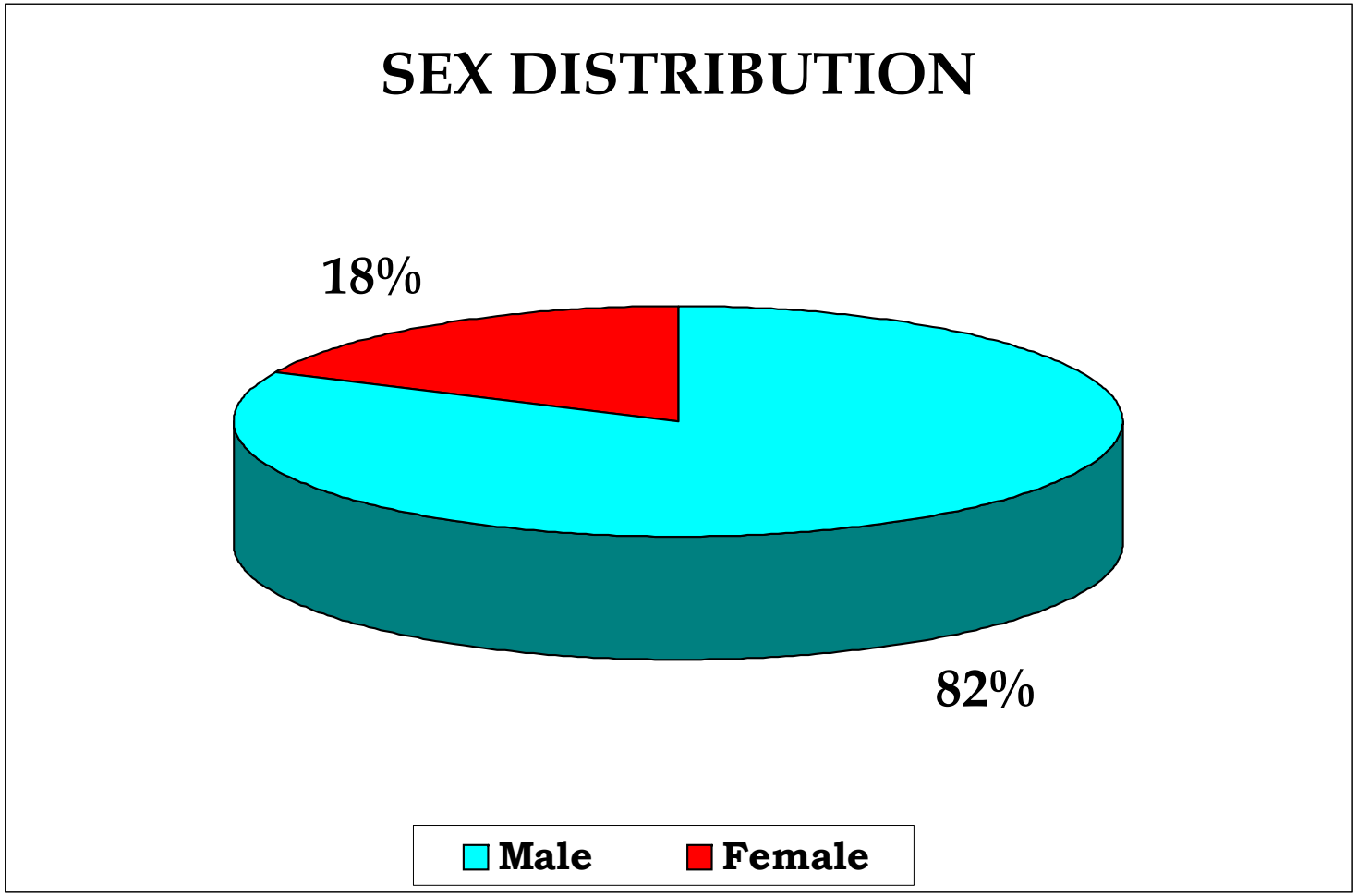
The lowest age was 31 years who was a male. The patient with highest age was a female of 82 years.

41 patients were male and 9 were female.

TABLE – 1

AGE AND SEX DISTRIBUTION OF PATIENTS

Age	Total No. of patients	Percentage	Male	Female
30 – 34	1	2%	1	0
35 – 39	1	2%	1	0
40 –44	10	20%	9	1
45 – 49	7	14%	7	0
50 – 54	3	6%	3	0
55 – 59	8	16%	7	1
60 – 64	10	20%	7	3
65 – 69	4	8%	3	1
70 –74	4	8%	2	2
75 – 79	1	2%	1	0
80 and above	1	2%	0	1
Total	50	100%	41	9


SEX DISTRIBUTION
18%
82%
Male
Female

PRESENTING SYMPTOMS (TABLE – 2)

Chest pain – 49 patients had complaints of retrosternal chest pain. The one patient who did not have chest pain as presentation had dyspnoea as presenting complaint.

TABLE – 2

PRESENTING SYMPTOMS

Sl.No	Symptoms	No.	Percentage
1.	Chestpain	49	98%
2.	Sweating	43	86%
3.	Vomiting	26	52%
4.	Palpitation	8	16%
5.	Syncope	7	14%
6.	Dyspnoea	4	8%

Radiation

Radiation was noticed in 29 patients (58%). Radiation to left upper limb was seen in 20 patients (40%), 3 patients (6%) had radiation to epigastrium, another 3 patients (6%) had radiation to right upper limb. Other sites of radiation were neck – 4%, Jaw – 2%.

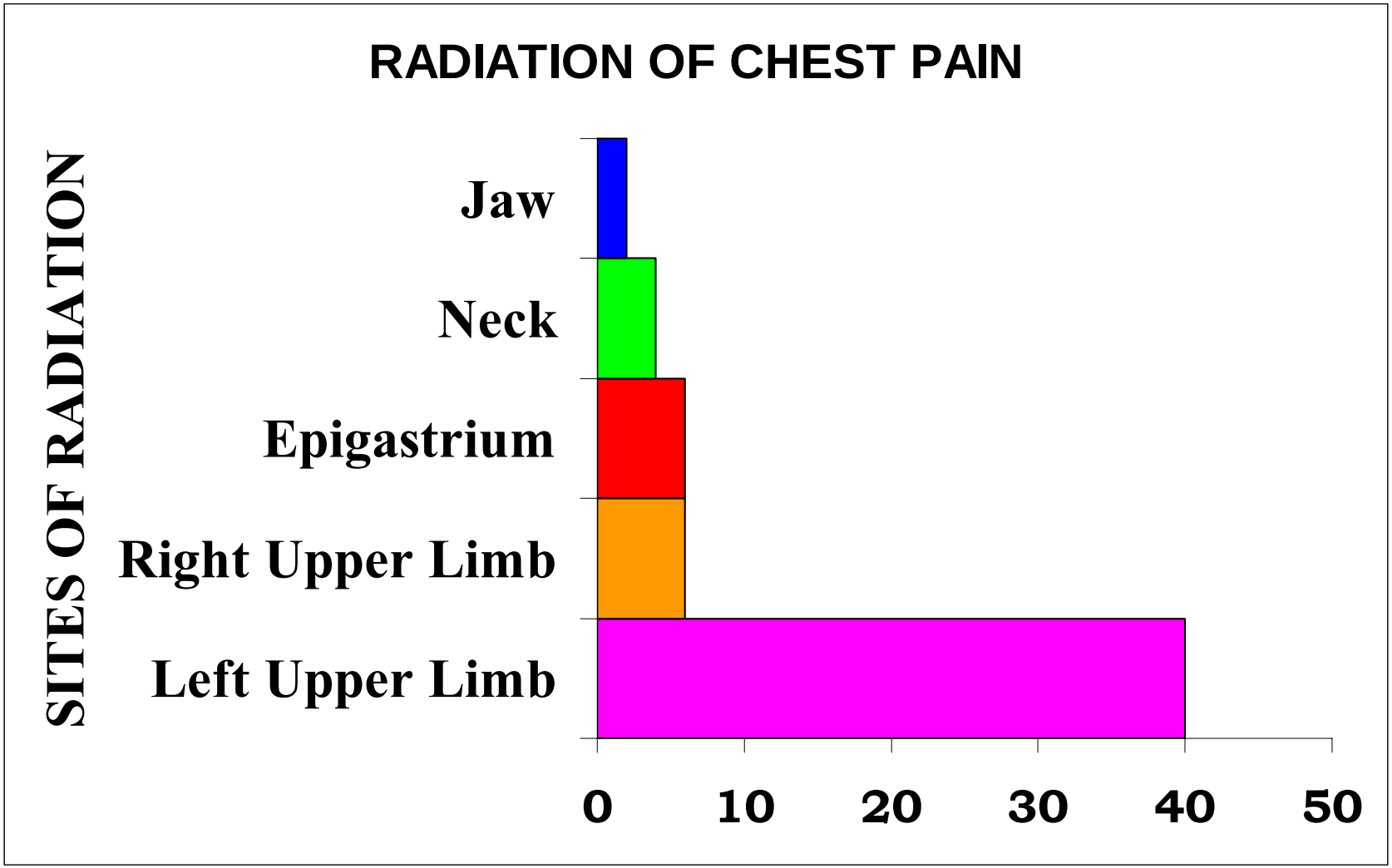
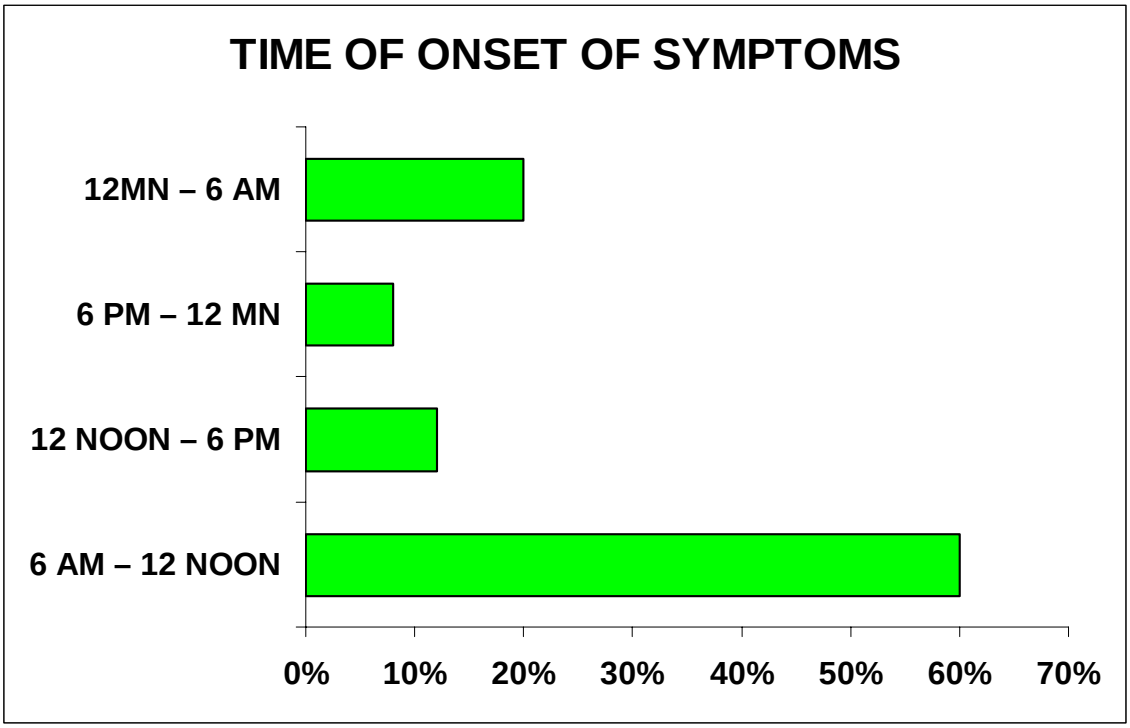

RADIATION OF CHEST PAIN
SITES OF RADIATION
Jaw
Neck
Epigastrium
Right Upper Limb
Left Upper Limb
0
10
20
30
40
50

TABLE – 3

TIME OF ONSET OF SYMPTOM

6 AM – 12 NOON	12 NOON – 6 PM	6 PM – 12 MN	12MN – 6 AM
60%	12%	8%	20%
30	6	4	10



Past History

CAD – unstable angina preceded acute myocardial infarction in 8 patients (16%). Most of these occurred within 2 weeks of acute myocardial infarction.

4 patients (8%) had effort angina before onset of present infarction. All had the complaint within last 1 year.

TABLE – 4

RISK FACTORS

Variables	No. of patients	Percentage
Diabetes Mellitus	11	22%
Hypertension	17	34%
Dyslipidemia	5	10%
Obesity	6	12%
Smoking	28	56%
Alcohol	14	28%

Diabetes Mellitus

11 patients (22%) had Diabetes and all of them were on oral hypoglycemic agents. An attempt was made to go through the earlier medical records wherever available.

Systemic Hypertension

Patients were considered to be hypertensive if they had been told to have hypertension earlier by a doctor and put on anti-hypertensive drugs or serial BP recordings in hospital were consistently high. 17 patients (34%) had hypertension.

Smoking

Out of 41 male, 28 were smokers. None of the female patients were smokers. All the males were smoking 10 – 20 beedies/cigarettes per day for atleast 15 – 30 years. 85% of them were current smokers.

Alcohol

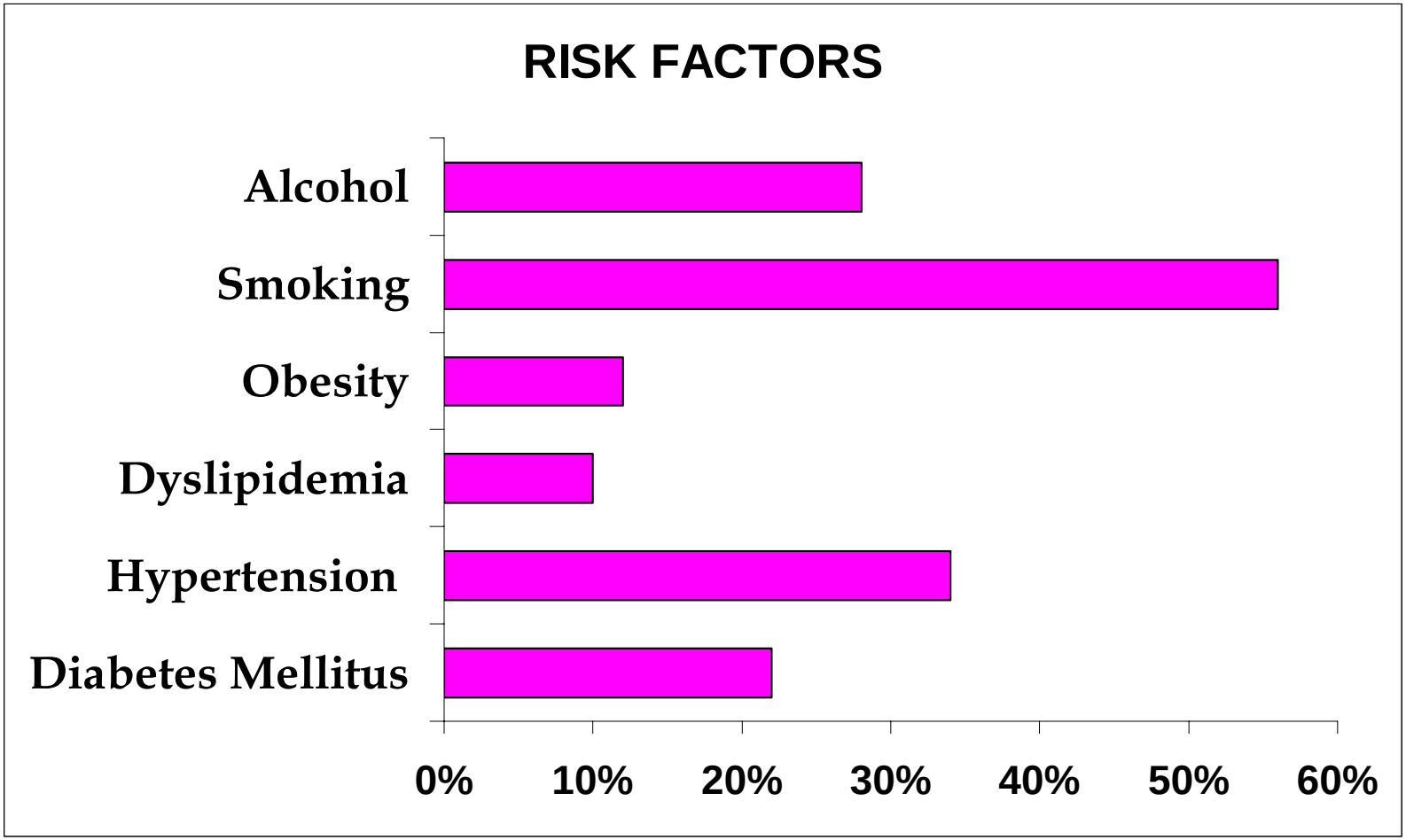
Out of 41 males, 14 were in habit of consuming alcohol. None of the female patients were in habit of consuming alcohol. All the male were in a habit of consuming alcohol for atleast 10 – 18 years, most of the days.

Obesity

6 patients (12%) were found to be obese. Body mass index was calculated for all patients.

Dyslipidemia

5 patients (10%) were found to have dyslipidemia, detected on routine check up elsewhere. None of them were on lipid lowering agents.


RISK FACTORS
Alcohol
Smoking
Obesity
Dyslipidemia
Hypertension
Diabetes Mellitus
0%
10%
20%
30%
40%
50%
60%

Family History of

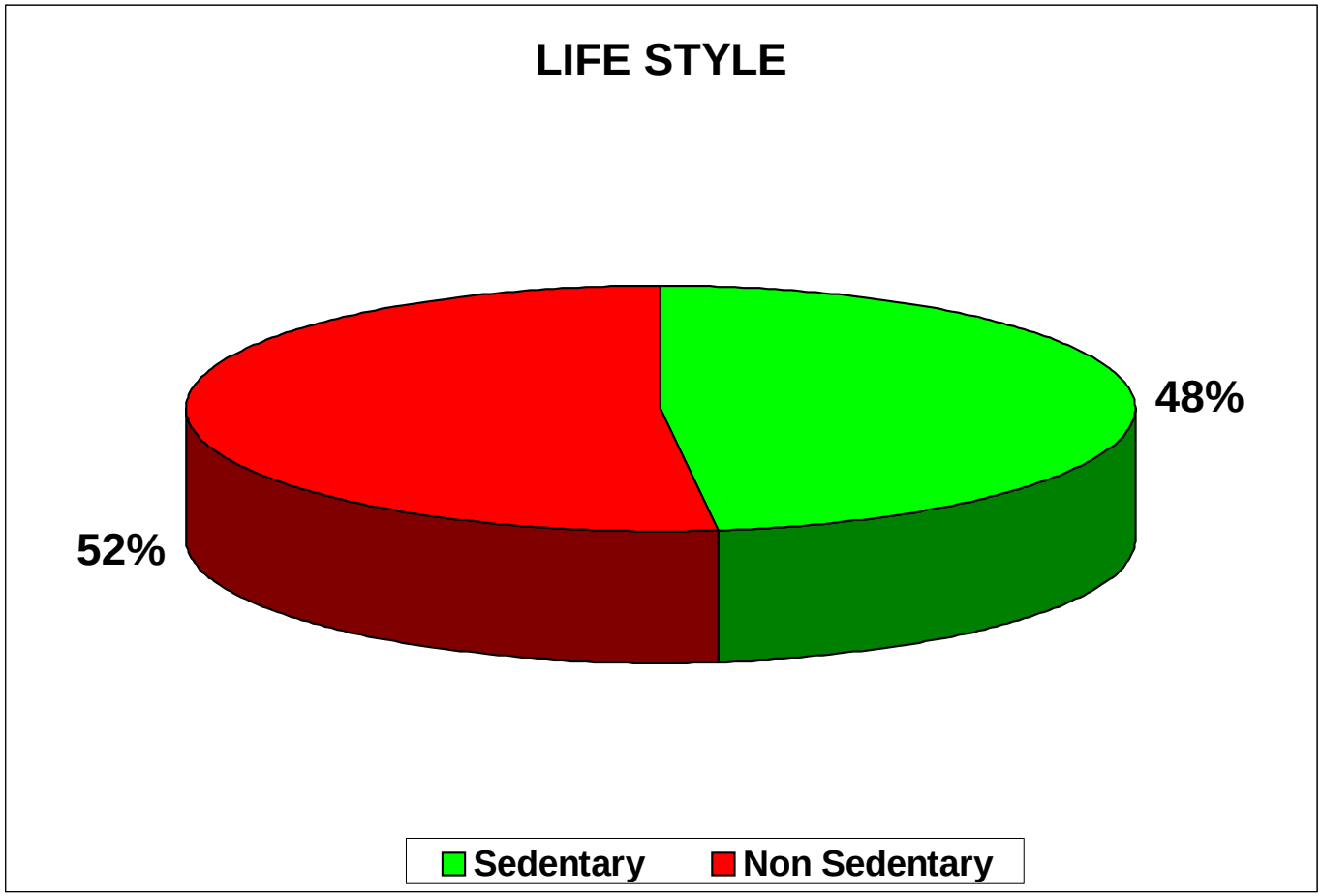
Family History of Diabetes Mellitus was noted in 10 patients (20%), hypertension was noted in 7 patients (14%) and coronary artery disease was noted in 9 patients (18%).

Occupation

26 patients (52%) were manual workers and the remaining doing sedentary jobs.

Treatment History

Out of 11 diabetic patients, 7 were on regular oral hypoglycemic agents, one was on oral hypoglycemic agents and insulin, 3 were on irregular treatment. 3 patients with angina were on aspirin, nitrates, ACE inhibitors and beta blockers. Out of 17 hypertensive patients, 8 were on regular anti hypertensive treatment, mostly combination therapy comprising of calcium channel blockers, ACE inhibitors and beta blockers.


LIFE STYLE
48%
52%
Sedentary
Non Sedentary

Clinical Examination

Pallor was noticed in 9 patients (18%). 14 patients (28%) had bradycardia. Tachycardia was seen in 6 patients (12%). Hypotension was noticed in 15 patients (30%). 16 patients (32%) had raised JVP. Right sided third or fourth sounds were detected in 10 patients (20%). All the patients had evidence of right ventricular infarction in the ECG. Tender hepatomegaly was seen in2 patients (4%).

TABLE – 5

CLINICAL SIGNS AT THE TIME OF PRESENTATION

Clinical sign	No. of patients	Percentage
Pallor	**9**	**18%**
Pulse < 60 / min	**14**	**28%**
Pulse > 100 / min	**6**	**12%**
BP < 100 mm Hg systolic	**15**	**30%**
↑ JVP	**16**	**32%**
Right sided S_3 / S_4	**10**	**20%**
Tender hepatomegaly	**2**	**4%**

ECG

ECG evidence of right ventricular infarction was present in 19 patients (38%). True posterior wall infarction, as evidenced by tall 'R' in V_1 with ST depression and upright 'T' wave, was observed in 8 patients (16%). The typical change was evident only after 24 hours.

Arrhythmias

First degree AV block was observed in 3 patients (6%). Second degree – mobitz type I was seen in 4 patients (8%). Second degree – Mobitz type II was seen in 3 patients (6%). Complete heart block was noted in 7 patients (14%). Transient complete RBBB was noted in one case which progressed to complete heart block. Left anterior hemi block was present in 1 case (2%). Most of the arrhythmias were transient, requiring no specific treatment. 3 patients who had right ventricular myocardial infarction, hypotension and complete heart block, expired during hospital stay. Atrial fibrillation was present in 2 patients (4%) and in both patients it had developed within first 24 hours. Papillary muscle dysfunction and mitral regurgitation was noted in 2 patients (4%).

Mortality

3 patients expired in the hospital, 2 of them had right ventricular infarction, profound hypotension and complete heart block expired within 24 hours of admission.

The other patient with right ventricular infarction, hypotension, and complete heart block developed CVA, right sided hemiplegia. CT brain showed infarct in left MCA territory.

TABLE – 6

COMPLICATION DEVELOPED DURING FOLLOW UP

Complications	No. of patients	Percentage
Atrial Fibrillation	2	4%
First Degree heart block	3	6%
Second degree heart block – Mobitz Type I	4	8%
Second Degree heart block – Mobitz Type II	3	6%
Complete Heart Block	7	14%
RBBB	1	2%
LAHB	1	2%
Mortality	3	6%

DISCUSSION

DISCUSSION

Fifty proven cases of Acute Inferior Wall Myocardial Infarction were selected for analyzing the various risk factors, clinical features and incidence of complications. All patients were admitted within 24 hours of onset of symptoms. All patients were subjected to ECG examination of V_3R and V_4R, and extended leads V_7 V_8 V_9 and rhythm strip wherever indicated.

On analyzing the age and the incidence, it was found that acute inferior wall infarction was most common in male above 40 years. 82% were male and 96% of patients were above the age of 40.

98% of patients presented with chest pain. All of them had typical retrosternal chest pain. The pain lasted for less than an hour in 22% of patients and more than an hour in the rest. All the patients had chest pain lasting for > 30 minutes.

When analyzing symptoms other than chest pain, the most common was sweating, present in 86% patients associated with cold extremities.

52% patients had vomiting and in all, it was associated with nausea.

14% of patients had history of syncope or pre syncope. Of these 7 cases, 6 of them had evidence of right ventricular infarction. High frequency of syncopal attacks in right ventricular infarction is consistent with high frequency of hypotension and bradyarrhythmias in these patients.

History of palpitation was observed in 16% patients. Of the 8 patients presented with palpitations, 2 had atrial fibrillation and 5 had right ventricular infarction.

A strong circadian periodicity was observed in the time of onset of symptom. This increased incidence in the morning hours has been reported in earlier studies.

Thomson et al, in his studies had revealed increased incidence of chest pain in morning hours[42,43].

22% of the patients reported physical exertion as a precipitating factor for symptoms. Rest of them developed the symptoms at rest. Most of the western studies have shown high incidence of attacks occurring at rest.

On analyzing the risk factors for coronary artery disease, smoking was the most frequent risk factor. 56% were smokers. All of them were male. 28% of patients were alcoholic. 34% of patients were Hypertensive; 22% were diabetic. Obesity (based on Body Mass Index) was noted in 6 patients (12%) and Dyslipidemia was noted in 10% (5 patients).

Hector Bueno[44] et al, in his studies had analyzed the incidence of risk factors. He had found hypertension in 45%, diabetes mellitus in 23%, smoking in 63%, Obesity in 22%, Dyslipidemia in 33%.

Previous history of coronary artery disease was noted in 12 patients. 8 of them had history of unstable angina, and 4 had effort angina. Out of 12 patients, 3 patients were on regular anti anginal medication.

Family history of coronary artery disease was noted in 18% of patients.

On analyzing the clinical signs, pallor was noted in 18% of patients. Bradycardia was noticed on 28% of patients, of these 60% were having conduction disturbances and the remaining RVI.

Compared to previous studies, 30% of patients had systolic BP < 100 mm Hg at the time of admission and of these 11 patients (22%) had RVMI. Chockalingam et al[45], in his study had revealed that though RVMI was seen in a third of acute IWMI, Hypotension was seen in only 10%.

38% (19) of patients showed evidence of RVMI. All of them showed ST elevation of more than 1 mm in V_3R and V_4R. The reported incidence of RVMI in patients with inferior wall MI ranges from 25 – 53%. All the patients who had ECG evidence of RVMI had clinical features of the same. The ECG changes in RVMI were transient and the changes reverted to normal or near normal within 24 hours of symptoms.

Saleem Sheriff et al[46], in his study had detected 34% RVMI in patients with IWMI. Chockalingam et al in his study had revealed RVMI was seen in one third cases of IWMI.

Of the 19 patients with ECG evidence of RVMI, 16 were showing elevated JVP with positive Kussmaul's sign. Raised JVP in these cases were exclusively due to RV failure, as there were no signs of LV failure in these patients. Previous reported incidence of positive Kussmaul's sign in RVMI varied from 20 –90%.

Morgan et al[47], in his study of RVMI had revealed that Kussmaul's sign was seen in 72% of patients with RVMI.

Of the 19 patients with ECG evidence of RVMI, 10 patients had right sided S_3 and S_4 and two patients had tender hepatomegaly. All patients with RVMI and hypotension were treated with IV fluids in addition to usual measures. Most patients had to be treated with inotropic support.

True posterior wall infarction, detected by the occurrence of tall 'R' in V_1 V_2 with ST depression associated with upright 'T', with ST elevation in $V_{7,}V_{8,}V_9$ was seen in 9 patients. 3 of these patients had associated RVMI. Incidence of posterior wall MI in patients with IWMI has been reported to be 15 – 20%. Gorselen[48] et al in his studies had detected an incidence of 15% of posterior wall MI in patients with acute IWMI.

5 patients out of 9 cases of true posterior wall MI had conduction disturbance. One had LAHB, one had complete heart block, one patient had mobitz type II, 2^{nd} degree block and two patients had mobitz type I 2^{nd} degree heart block.

Some form of conduction disturbance were noticed in 42% of patients. Complete heart block was seen in 7 cases (14%). All of them were associated with RVMI and hypotension and three of them died during hospital stay. All the rest improved with conservative management.

Atrial fibrillation was transient and was noticed in 4% of patients. This might be due to atrial ischaemia. Reported incidence[49] of AF in acute MI is 10 – 15% but it is only 1/3 in those with IWMI as compared to AWMI.

One patient (2%) developed complete RBBB and one patient developed left anterior hemiblock (2%). The reported incidence of right bundle branch block in acute IWMI is 2 – 4%.Peter Berger et al[50], in his studies of complications of acute myocardial infarction, found atrial fibrillation in 12% of patients with IWMI, heart blocks in 50%, with high degree heart blocks in 14%. Philip Podrid et al[51], in his study had detected atrial fibrillation in 10%, first degree atrio ventricular block in 4%, Mobitz type I second degree atrioventricular block in 10%, type II Mobitz second degree atrio ventricular block in 2%, complete heart block in 8% of patients with IWMI.

Various studies[52-57] have shown conduction disturbance was more common in IWMI with RVMI than without RVMI.

Pericardial friction rub was noticed in three patients (6%) and this is almost equal to what is reported in literature. It occurred between 2nd and 4th day and gradually disappeared within a week. Papillary muscle dysfunction with pansystolic murmur was seen in two patients (4%) and this was not associated with major haemodynamic instability. Reported incidence[58] of papillary muscle dysfunction is 1 – 5% in-patient with IWMI.

60% of patients (30) received thrombolytic therapy and the rest of them were treated conservatively.

Three patients expired while on treatment. Two of them had severe RVMI, complete heart block and persistent hypotension. The other patient developed right-sided hemiplegia due to infarct in left middle cerebral artery territory. Various studies have shown high incidence of mortality[59-63] in patients with IWMI with RVMI.

The rest of the patients left the hospital without major complications.

CONCLUSION

CONCLUSION

1. The incidence of Acute Inferior Wall Myocardial Infarction is much higher in males than in females, the difference being less as age advances.
2. In males, there is a distinct increase in the incidence after age of 40 years in this study.
3. Typical retrosternal chest pain lasting for more than 30 minutes associated with sweating was seen in almost all patients.
4. Smoking was the most prevalent risk factor (56%).
5. The onset of symptoms in majority of the patients (60%) was between 6 am – 12 noon.
6. The incidence of Right ventricular infarction in this study was 38%.
7. Syncope or presyncope was a prominent symptom in patients with right ventricular infarction.
8. Triad of raised JVP, Hypotension and clear lung fields were seen in 15 patients (30%).
9. All cases of inferior wall infarction should have right-sided leads during ECG examination and this should be done as early as possible.

10. If diagnosis of RVMI is correctly made and treated early, the prognosis is usually good.
11. Incidence of true posterior wall myocardial infarction in this study was 16%.
12. Mortality is higher in patients with right ventricular infarction when compared with those without this complication.

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BIBLIOGRAPHY

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ANNEXURE

PROFORMA

Name : Age : Sex:

I.P. No : Date of Admission:

Complaints of:

Chest pain	:	Y/N
Radiation	:	Y/N
Sweating	:	Y/N
Dyspnoea	:	Y/N
Vomiting	:	Y/N
Palpitation	:	Y/N
Syncope	:	Y/N

Past History of:

Diabetes Mellitus	:	Y/N
Hypertension	:	Y/N
Coronary Artery Disease	:	Y/N
Dyslipidemia	:	Y/N

Family History of:

Diabetes Mellitus	:	Y/N
Hypertension	:	Y/N
Ischemic Heart Disease	:	Y/N

Personal History of:

Smoking	:	Y/N
Alcohol	:	Y/N

Occupation

Sedentary / Non Sedentary :

On Examination

BP	:	PR	:
Pallor	:	CVS	:
JVP	:	RS	:
		Abdomen	:

Investigation

RBS	:
Urea	:
Creatinine	:
CPK	:
CPK MB	:
Sr.Cholesterol	:
Hb%	:
ECG	:
Chest X ray – PA view	:

Treatment

Conservative

Thrombolysis

Anticoagulants

Complications

Shock	:
Rhythm disturbance	:
Death	:
Discharge	:

Ref.No. /ME1/2007

Stanley Medical College,
Chennai-1 Dt. -9-2007

Sub:Medical Education—Stanley Medical College, Chennai—
Ethical Committee constituted for approval of Dissertation/
Thesis submitted—regarding.

The Ethical Committee meeting was held on 3-9-2007 and 7-9-2007 to discuss the paper submitted for Dissertation / Thesis.

The following Members of the Ethical Committee were present and discuss in detail for the approval of the papers presented by the individual by means of power point presentation.

Dr.A.Sundaram, Dean incharge,
Dr.S.Madhavan, Prof. of Pharmacology,
Dr.Thenmozhivalli, Prof. of Microbiology,
Dr.S.Natarajan, Prof. of Medicine,
Dr.K.Balasubramanian, Prof. of Physiology
Dr.M.L.Shyamala, Prof. of Surgery,
Thiru M.Panneerselvam, Junior Administrative Officer.

LIST OF PAPERS SUBMITTED FOR ETHICAL COMMITTEE APPROVAL
ETHICAL MEETING

Dr. Kiruba Mohan, Prof. of Dermatology

1."N.O.C. for PMS study of pregabalin" - Dr.Parimalam Kumar
2. " A Phase IIb/III trial of LLL-3348 of lupin ltd in plaque psoriasis - Dr.A.Ramesh

Dr.M.Thirunavkarasu, M.D.(Psy)D.PM , Prof. of Psychiatry
"Prevalence, socio-demographic variables and method of suicide among various causes of death."

(2)Psychological autopsy of suicide.

V.Rohit
Effect of chewing gums (XYLITOL)

K.Chinthnidhi
Mycotic infections in immuno compromised and cancer patients.

Malavika Prasad
Profile of Hypertensive emergencies - A study of 100 cases from Dept. of medicine, GSH.

3. Sandhya Rani.C Final MBBS,
Assessment of coverage and quality of maternal and child health services at Minjur Primary Health Centre; Block level
4.C.Muralidharan, Final year.
The implications of mobile phones on hearing loss.
5.V.Sarath Chander, 3rd MBBS
Prevalence of Deafness in children.
6.B.Madhusoothanan, 3rd year
(1) Lung functions in type 2 diabetes.
(2)Hyponatremia in intensive medical care patients in GSH.
7.S.Sathyapriya – II MBBS.,
"A study about screening tests for cases of urinary tract infections (UTIs)Using Urine samples."
8.S.Moogaambiga,
"Estended spectrum beta lactamase producing microbes.

POST GRADUATES
1.Dr.R.Arunprakas –M1. P.G.
Analysis of clinical profile of systemic lupus erythematosus
2.Dr.S.Murugananth – M.2 P.G.
Clinical Profile of infectious fevers
3.Dr.N.Loganathan – M2 P.G.
Clinical and Epidemiological profile of Human Leptospirosis in North Chennai.
4.Dr. K. Babu – M3 – P.G.
Study of Clinical Profile of patients with acute inferior wall myocardial infarction.
5.Dr. S.P.Maharajan – M3 – P.G.
Analytical study of atrial fibrillation in Govt. Stanley Medical College Hospital.
6.Dr.P.R.Sowmini – M3 - P.G.
Clinical profile of arrhythmias complicating acute anterior wall myocardial infarction.
7.Dr.E.Uma Maheswari – M4 – PG
Clinical Radiological analysis of Focal seizures with CT Scan.
8.Dr.S.Sudha Selvi, M4 - PG
Clinical profile of chronic obstructive pulmonary disease.

9.Dr.N.Jayanthi. M6- PG
Prevalence of B2 glycoprotein 1 Dependent anticardiolipin antibodies in acute ischemic stroke.
10.Dr.Lavanya. S. – MD PG
Comparative study of fasting lipid profile in chronic renal failure patients on conservative management, on dialysis and after renal transplant.
11.Dr.R.Geetha – Pharmacology

Evaluation of the sedative effects produced by antihistamines inhealthy volunteers by new techniques.

12.Dr.K.G.Devibala, Pharmacology

To evaluate the efficacy of rupatadine in controlling pruritis in lichen planus.

13.Dr.B.Anitha, Physiology

Visual Evoked potentials in hypothyroid patients.

14.Dr.M.Thirumaran, Physiology

Heart rate variability analysis in alcohol dependant individuals.

15.Dr.K.Vinod, Anaesthesia

Real time ultra sound guided catheterization of IJV – A prospective comparison with land mark guided technique.

16.Dr.Rajesh. C.P. – M6 – PG

Cardiac conduction abnormalities and asymptomatic myocardial infarction in NIDDM patients.

The papers presented to the Committee members by the Profs./Asst. Prof./Post Graduates/Under graduates were discussed across the table while their presentation.

The above papers discussed in detail with its supportive documents submitted by them and approved the above papers submitted for Ethical Committee.

Name of the Members	Signature
Dr.A.Sundaram, Dean incharge,	
Dr.S.Madhavan, Prof. of Pharmacology,	8/10/07
Dr.Thenmozhivalli, Prof. of Microbiology,	
Dr.S.Natarajan, Prof. of Medicine,	9.10.2007
Dr.K.Balasubramanian, Prof. of Physiology,	
Dr.M.L.Shyamala, Prof. of Surgery,	08/10/07
Thiru M.Panneerselvam, Junior Administrative Officer.	

ABBREVIATIONS

ACS	**:**	**ACUTE CORONARY SYNDROME**
RVI	**:**	**RIGHT VENTRICULAR INFARCTION**
IWMI	**:**	**INFERIOR WALL MYOCARDIAL INFARCTION**
AWMI	**:**	**ANTERIOR WALL MYOCARDIAL INFARCTION**
TR	**:**	**TRICUSPID REGURGITATION**
RCA	**:**	**RIGHT CORONARY ARTERY**
LAD	**:**	**LEFT ANTERIOR DESCENDING**
COPD	**:**	**CHRONIC OBSTRUCTIVE PULMONARY DISEASE**
PS	**:**	**PULMONARY STENOSIS**
AV	**:**	**ATRIO VENTRICULAR**
SA	**:**	**SINO ATRIAL**
RV	**:**	**RIGHT VENTRICLE**
LV	**:**	**LEFT VENTRICLE**

AMI	**:**	**ACUTE MYOCARDIAL INFARCTION**
PTCA	**:**	**PERCUTANEOUS TRANSLUMINAL CORONARY ANGIOPLASTY**
CVA	**:**	**CEREBRO VASCULAR ACCIDENT**
MCA	**:**	**MIDDLE CEREBRAL ARTERY**
CK	**:**	**CREATINE KINASE**
RAP	**:**	**RIGHT ATRIAL PRESSURE**
PCWP	**:**	**PULMONARY CAPILLARY WEDGE PRESSURE**
JVP	**:**	**JUGULAR VENOUS PRESSURE**
RBBB	**:**	**RIGHT BUNDLE BRANCH BLOCK**
LAHB	**:**	**LEFT ANTERIOR HEMI BLOCK**
ANF	**:**	**ATRIAL NATRIURETIC FACTOR**

MASTER CHART

Sl.No.	Name	Age	Sex	DOA	TOA	Occupation	IP No	SYMPTOMATOLOGY						
								Angina	Radiation	Sweating	Palpitations	Breathlessness	Vomiting	Syncope
1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
1	SENGENI	45	M	6.12.06	7.00 a.m.	NS	44091	+	+	+	-	-	+	-
2	DURAIRAJ	66	M	8.12.06	9.00 a.m.	NS	46116	+	-	+	-	-	+	+
3	CHANDRAN	60	M	15.12.06	10.00 a.m.	NS	46626	+	-	+	+	-	-	-
4	ESTHER	65	F	25.12.06	7.00 a.m.	S	46979	+	+	+	-	-	-	-
5	BALAKRISHNAN	64	M	10.1.07	10.00 a.m.	S	567	+	-	-	-	-	+	-
6	KUPPULAXMI	72	F	3.2.07	10.05 p.m.	S	3721	+	-	+	+	+	-	+
7	MUTHURATNAM	63	M	6.2.07	7.40 a.m.	NS	4097	+	+	+	-	-	+	-
8	RAMASAMY	68	M	10.2.07	12.20 p.m.	S	4397	+	-	-	+	+	+	+
9	MURUGESAN	60	M	13.2.07	3.30 a.m.	NS	4722	+	-	+	-	-	-	-
10	JOSEPH	74	M	18.2.07	3.00 a.m.	S	5406	+	+	-	-	-	-	-
11	SETHURAM	60	M	18.2.07	3.50 a.m.	S	5407	+	-	-	+	-	+	-
12	SELVARAJ	53	M	23.2.07	9.30 a.m.	S	6050	+	-	+	-	-	-	-
13	ANANDRAJ	55	M	25.2.07	6.35 a.m.	NS	6250	+	-	+	-	-	+	-
14	SELVAM	45	M	27.2.07	7.40 a.m.	NS	6508	+	+	+	+	-	-	-
15	SAMPATH	48	M	1.3.07	2.30 p.m.	NS	6778	+	+	+	-	-	+	-
16	BALAKRISHNAN	41	M	7.3.07	10.15 a.m.	NS	7469	+	+	+	-	-	-	-
17	MANI	38	M	10.3.07	11.25 a.m.	NS	7813	+	+	+	-	-	+	-

Sl.No.	Name	Age	Sex	DOA	TOA	Occupation	IP No	SYMPTOMATOLOGY						
								Angina	Radiation	Sweating	Palpitations	Breathlessness	Vomiting	Syncope
1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
18	RAJENDRAN	47	M	13.3.07	7.00 a.m.	NS	7922	+	+	+	-	-	+	-
19	SYED RAHUTHULA	48	M	14.3.07	3.00 a.m.	NS	8244	+	+	+	-	-	+	-
20	KATHYEBEE	82	F	15.3.07	11.20 a.m.	S	8463	-	-	-	-	+	+	+
21	THANGAPPAN	55	M	18.3.07	1.40 p.m.	S	8767	+	+	+	-	-	-	-
22	ARUMUGAM	64	M	21.3.07	3.30 p.m.	S	9191	+	-	+	-	-	+	-
23	HABEEB	55	M	25.3.07	12.30 a.m.	S	9617	+	+	+	-	-	-	-
24	SELVARANI	55	F	1.4.07	12.35 a.m.	S	10447	+	+	+	-	-	+	-
25	KAMALA	60	F	4.4.07	11.30 a.m.	S	10842	+	-	+	+	-	-	+
26	AHMED BASHA	58	M	8.4.07	1.50 p.m.	S	11262	+	-	+	-	-	-	-
27	VINCENT	49	M	8.4.07	3.45 a.m.	NS	11271	+	-	+	-	-	-	-
28	JEYAKUMAR	50	M	12.4.07	10.20 a.m.	S	11820	+	+	+	-	-	+	-
29	PALANI	54	M	17.4.07	9.00 p.m.	S	12343	+	+	+	-	-	-	-
30	BEGUM	43	F	25.4.07	4.00 a.m.	NS	13243	+	+	+	-	-	-	-
31	BABU	42	M	25.4.07	9.30 a.m.	NS	13267	+	-	+	-	-	+	-
32	MANI	42	M	28.4.07	11.15 p.m.	NS	13682	+	+	+	-	-	+	-
33	MUNIVEL	58	M	6.5.07	1.20 a.m.	NS	14586	+	+	+	+	-	-	-
34	KOTHANDAPANI	40	M	12.5.07	9.10 a.m.	NS	15416	+	+	+	-	-	+	-

Sl.No.	Name	Age	Sex	DOA	TOA	Occupation	IP No	SYMPTOMATOLOGY						
								Angina	Radiation	Sweating	Palpitations	Breathlessness	Vomiting	Syncope
1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
35	ANTHONY	40	M	13.5.07	2.30 a.m.	NS	15534	+	+	+	-	-	-	-
36	MUTHIAH	63	M	14.5.07	8.45 a.m	S	15790	+	-	+	+	-	+	-
37	ALEXANDER	74	M	15.5.07	9.00 a.m.	S	15820	+	+	-	-	+	+	+
38	ARTHUR	55	M	17.5.07	5.45 p.m.	S	16188	+	+	+	-	-	-	-
39	CHITRARAMAN	31	M	17.5.07	7.35 p.m.	NS	16195	+	+	+	-	-	+	-
40	ETHIRAJ	56	M	17.5.07	9.00 a.m.	NS	16200	+	+	-	-	-	-	-
41	SARADA	60	F	22.5.07	7.00 a.m.	S	16803	+	-	+	-	-	-	-
42	MOHD. FAROOQ	48	M	6.6.07	9.00 a.m.	NS	18705	+	+	+	-	-	-	-
43	RAJ	40	M	10.6.07	7.50 a.m.	NS	19154	+	-	+	-	-	+	-
44	RAJ NAIDU	40	M	25.6.07	8.20 a.m.	NS	21054	+	+	+	-	-	-	-
45	SHANKAR	42	M	1.7.07	6.30 a.m.	NS	21728	+	-	+	-	-	+	-
46	SELVARAJ	44	M	7.7.07	10.50 a.m.	NS	22681	+	+	+	-	-	-	-
47	ABDULKADAR	66	M	8.7.07	8.25 a.m.	S	22716	+	-	+	-	-	+	+
48	MANONMANI	63	F	14.7.07	7.30 a.m.	S	23437	+	+	+	-	-	+	-
49	JAGAN	78	M	29.7.07	8.45 a.m.	S	25434	+	+	+	-	-	+	-
50	SIVAGAMI	71	F	30.7.07	8.00 a.m.	S	25465	+	-	+	-	-	-	-

Sl.No	RISK FACTORS								FAMILY HISTORY			ON EXAMINATION								
	DM	HT	CAD	Dyslipedemia	Obesity	Smoking	Alcoholism	Menopausal	DM	HT	CAD	PR	BP	Anaemia	JVP	RV/S3/S4	Murmur	RUB	Breath Sound	Hepatomegaly
	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35
1	+	+	+	-	-	+	+	-	+	+	+	50	80/60	+	↑	+	-	-	N	-
2	-	-	-	-	-	+	+	-	-	-	-	80	170/100	-	N	-	-	-	N	-
3	-	+	-	-	-	+	+	-	-	-	-	82	90/50	-	↑	+	-	+	N	+
4	-	-	-	-	-	-	-	+	-	-	-	54	130/80	-	N	-	-	-	N	-
5	-	-	-	-	-	+	-	-	-	-	-	78	180/90	-	N	-	-	-	N	-
6	-	-	-	-	-	-	-	+	-	-	-	50	70/50	+	↑	+	-	-	N	-
7	-	+	+	-	-	+	-	-	-	+	-	90	160/80	-	N	-	PSM	-	N	-
8	-	-	-	-	-	+	-	-	-	-	-	110	80/60	-	↑	-	-	-	N	-
9	+	+	+	+	-	+	-	-	+	-	+	84	150/70	-	N	-	-	-	N	-
10	-	-	-	-	+	-	-	-	-	-	-	52	80/50	-	↑	+	-	-	N	-
11	-	+	+	-	-	+	-	-	-	+	-	121	120/70	-	N	-	-	+	N	-
12	-	-	-	-	-	+	-	-	-	-	-	84	120/80	-	N	-	-	-	N	-
13	-	-	-	-	-	+	-	-	-	-	-	86	130/80	-	N	-	-	-	N	-
14	+	+		-	-	+	-	-	-	-	-	128	110/80	-	N	-	-	-	N	-
15	-	+	+	-	-	+	+	-	-	-	-	82	190/110	-	N	-	-	-	N	-
16	-	-	-	-	-	-	-	-	-	-	-	80	130/80	-	N	-	-	-	N	-
17	+	+	+	+	+	-	-	-	+	-	+	54	90/50	+	↑	-	-	-	N	-

Sl.No	RISK FACTORS								FAMILY HISTORY			ON EXAMINATION								
	DM	HT	CAD	Dyslipedemia	Obesity	Smoking	Alcoholism	Menopausal	DM	HT	CAD	PR	BP	Anaemia	JVP	RV/S3/S4	Murmur	RUB	Breath Sound	Hepatomegaly
	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35
18	-	-	-	-	-	+	+	-	-	-	-	42	80/60	-	↑	+	-	-	N	+
19	-	-	-	-	-	+	+	-	-	-	-	81	116/80	-	N	-	PSM	-	N	-
20	-	-	-	-	-	-	-	+	-	-	-	68	80/60	+	↑	+	-	+	N	-
21	-	-	-	-	-	-	-	-	-	-	-	91	120/80	-	N	-	-	-	N	-
22	-	-	-	-	-	+	-	-	-	-	-	93	110/70	-	N	-	-	-	N	-
23	-	+	+	+	+	+	+	-	+	+	+	41	80/60	-	↑	+	-	-	N	-
24	-	-	-	-	-	-	-	+	-	-	-	84	120/70	-	N	-	-	-	N	-
25	+	+	+	-	-	-	-	+	+		+	40	80/50	+	↑	-	-	-	N	-
26	-	-	-	-	-	+	+	-	-	-	-	83	140/80	-	N	-	-	-	N	-
27	-	-	-	-	-	+	+	-	-	-	-	82	140/90	-	N	-	-	-	N	-
28	-	-	-	-	-	+	-	-	-	-	-	41	70/50	-	↑	-	-	-	N	-
29	+	-	-	-	-	-	-	-	+	-	-	53	80/60	+	↑	+	-	-	N	-
30	-	+	-	-	-	-	-	-	-	-	-	123	170/100	-	N	-	-	-	N	-
31	+	-	+	-	-	+	+	-	+	-	+	74	110/80	-	N	-	-	-	N	-
32	-	-	-	-	-	+	-	-	-	-	-	41	80/50	+	↑	-	-	-	N	-
33	-	-	-	-	-	+	+	-	-	-	-	76	120/80	-	N	-	-	-	N	-
34	-	-	-	-	-	+	-	-	-	-	-	84	130/80	-	N	-	-	-	N	-

Sl.No	RISK FACTORS								FAMILY HISTORY			ON EXAMINATION								
	DM	HT	CAD	Dyslipedemia	Obesity	Smoking	Alcoholism	Menopausal	DM	HT	CAD	PR	BP	Anaemia	JVP	RV/S3/S4	Murmur	RUB	Breath Sound	Hepatomegaly
	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35
35	+	+	+	+	+	-	-	-	+	+	+	88	170/120	-	N	-	-	-	N	-
36	-	+	-	-	-	-	-	-	-	-	-	120	106/70	-	N	-	-	-	N	-
37	-	-	-	-	-	-	-	-	-	-	-	81	130/80	-	N	+	-	-	N	-
38	-	-	-	-	-	-	-	-	-	-	-	56	110/70	-	N	-	-	-	N	-
39	-	+	-	-	-	-	-	-	-	-	-	95	200/130	-	N	-	-	-	N	-
40	+	-	-	-	+	+	+	-	-	-	-	94	190/100	-	N	-	-	-	N	-
41	-	-	-	-	-	-	-	+	-	-	-	90	130/80	-	N	-	-	-	N	-
42	+	+	+	+	-	+	+	-	-	-	-	42	80/50	+	↑	-	-	-	N	-
43	-	+	+	-	+	-	-	-	+	+	+	86	120/80	-	↑	-	-	-	N	-
44	-	-	-	-	-	-	-	-	-	-	-	51	100/70	-	N	-	-	-	N	-
45	-	-	-	-	-	+	-	-	-	-	-	88	110/70	-	N	-	-	-	N	-
46	-	-	-	-	-	+	+	-	-	-	-	85	140/90	-	N	-	-	-	N	-
47	+	+	-	-	-	+	-	-	+	+	+	130	110/70	-	N	-	-	-	N	-
48	-	-	-	-	-	-	-	+	-	-	-	82	160/100	+	N	-	-	-	N	-
49	-	-	-	-	-	-	-	-	-	-	-	83	80/60	-	↑	+	-	-	N	-
50	-	-	-	-	-	-	-	+	-	-	-	84	150/80	-	N	-	-	-	N	-

Sl.No	ECG	Conservative	Thrombolysed	COMPLICATIONS		
				Shock	Arrhythmia	Death
	36	37	38	39	40	41
1	IWMI + RVI	+	-	+	Second degree AVB Type 1	-
2	IWMI	-	+	-	_	-
3	IWMI + RVI	-	+	+	_	-
4	IWMI	-	+	-	_	-
5	IWMI	-	+	-	_	-
6	IWMI + RVI	+	-	+	CHB	+
7	IWMI + DMI	-	+	-	_	-
8	IWMI + RVI	-	+	+	Second degree AVB Type 1	-
9	IWMI	-	+	-	_	-
10	IWMI + RVI + DMI	+	-	+	Second degree AVB Type 2	-
11	IWMI	-	+	-	AF	-
12	IWMI + DMI	+	-	-	Second degree AVB Type 1	-
13	IWMI	-	+	-	_	-
14	IWMI	+	-	-	AF	-
15	IWMI	+	-	-	Second degree AVB Type 2	-
16	IWMI	-	+	-	_	-
17	IWMI + RVI + DMI	+	-	+	Second degree AVB Type 1	-

Sl.No	ECG			COMPLICATIONS		
		Conservative	Thrombolysed	Shock	Arrhythmia	Death
	36	37	38	39	40	41
18	IWMI + RVI	+	-	+	CHB	-
19	IWMI	-	+	-	_	-
20	IWMI + RVI	+	-	+	CHB	+
21	IWMI	-	+	-	_	-
22	IWMI	-	+	-	_	-
23	IWMI + RVI + DMI	+	-	+	First Degree AVB	-
24	IWMI	-	+	-	_	-
25	IWMI + RVI	+	-	+	CHB	-
26	IWMI	-	+	-	_	-
27	IWMI	-	+	-	_	-
28	IWMI + RVI	+	-	+	CHB	-
29	IWMI + RVI	+	-	+	Second Degree AVB Type 2	-
30	IWMI + DMI	+	-	-	_	-
31	IWMI	-	+	-	_	-
32	IWMI + RVI	+	-	+	CHB	-
33	IWMI	-	+	-	_	-
34	IWMI + DMI	-	+	-	_	-

Sl.No	ECG			COMPLICATIONS		
		Conservative	Thrombolysed	Shock	Arrhythmia	Death
	36	37	38	39	40	41
35	IWMI	+	-	-	_	-
36	IWMI + RVI	-	+	-	_	-
37	IWMI + RVI	+	-	-	First Degree AVB	-
38	IWMI	-	+	-	_	-
39	IWMI + DMI	+	-	-	_	-
40	IWMI	-	+	-	_	-
41	IWMI	-	+	-	_	-
42	IWMI + RVI	+	-	+	CHB	+
43	IWMI +RVI	-	+	-	First Degree AVB	-
44	IWMI	-	+	-	_	-
45	IWMI + DMI	-	+	-	LAHB	-
46	IWMI	-	+	-	_	-
47	IWMI + RVI	-	+	-	_	-
48	IWMI	-	+	-	_	-
49	IWMI + RVI	+	-	+	RBBB	-
50	IWMI	-	+	-	_	-

CHB : Complete Heart Block
AVB : Atrio Ventricular Block
LAHB : Left Anterior Hemi Block
RBBB : Right Bundle Branch Block
AF : Atrial Fibrillation
PSM : Pan Systolic Murmur
IWMI : Inferior Wall Myocardial Infarction
RVI : Right Ventricular Infarct
DMI : Dorsal Wall Myocardial Infarction
NS : Non Sedentary
S : Sedentary