CLINICAL PROFILE OF ACUTE MYOCARDIAL INFARCTION IN ELDERLY - A COMPARATIVE ANALYSIS WITH YOUNG

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CERTIFICATE

This is to certify that the Dissertation entitled **"CLINICAL PROFILE OF ACUTE MYOCARDIAL INFARCTION IN ELDERLY – A COMPARATIVE ANALYSIS WITH YOUNG",** herewith submitted by **Dr.MURUGAPANDIAN.*N*,** Post Graduate in General Medicine, Coimbatore Medical College to the Tamil Nadu Dr. *M.G.R.* Medical University is a record of a bonafide research work carried out by him under my guidance and supervision from Jan 2006 to Jun 2007.

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DECLARATION

I solemnly declare that the Dissertation titled "**CLINICAL PROFILE OF ACUTE MYOCARDIAL INFARCTION IN ELDERLY – A COMPARATIVE ANALYSIS WITH YOUNG",** was done by me at Coimbatore Medical College & Hospital during the period from Jan 2006 to Jun 2007 under the guidance and supervision of Head of Department &Prof. of medicine **DR.UMAKANTHAN, M.D.**

This dissertation is submitted to the Tamil Nadu Dr. *M.G.R.* Medical University towards the partial fulfillment of the requirement for the award of M.D. Degree (Branch *I*) in General Medicine.

Place: Coimbatore **Dr.MURUGAPANDIAN.N.**

Date:

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SL.NO	CONTENTS	PAGE

1.INTRODUCTION

Ageing is a natural process. In the words of Seneca: “Old age is an incurable disease”, but more recently, Sir James Sterling Ross commented: “You do not heal old age. You protect it; you promote it; you extent it”[1]. In the coming decades, as has been occurring in developed countries, India will experience the process of population ageing. As people age, a trend towards a change in the pattern of morbidity and mortality occurs.

Heart disease is the leading cause of hospitalization and death in elderly patients. The role of conventional cardiovascular risk factors in older persons is incompletely understood because only fragmentary and inadequate data are available in most instances. The manifestations of acute myocardial infarction are generally believed to be atypical in the elderly. Although the typical onset of crushing substernal or epigastric pain is still fairly common in the aged, other modes of presentation (such as acute confusion, syncope, atypical chest pain, congestive heart failure, vomiting or weakness) are frequently encountered. Since the presentation is variable, the diagnosis of myocardial infarction is often overlooked. The elderly are also believed to have a higher rate of complications and higher mortality.

Apart from diagnostic difficulty of AMI in elderly due o atypical clinical presentation, management of such cases is also challenging. They may be more refractory to medical therapy possibly because of advanced atherosclerotic disease and ventricular dysfunction particularly diastolic dysfunction. Even, they are more intolerant to therapy with multiple anti ischemic agents. Management decisions during the first 24 hrs have the greatest effect on survival in the elderly as is true with all age group. The major goals of treating elderly patients are to improve survival and reduce symptoms. Since elderly patients with AMI differ in clinical presentation than young patients with AMI, this issue needs perfect understanding. It will help us to reduce mortality and morbidity.

Knowing the differences between the elderly and young acute myocardial infarction patients in our local population will help identify aspects which may need further evaluation to formulate strategies to improve outcomes in elderly acute myocardial infarction patients.

2. AIM OF THE STUDY

The objective of the study is to analyze and compare the following events of acute myocardial infarction in elderly with acute myocardial infarction in young:

1) Age and sex distribution
2) Symptomatology and clinical presentation
3) Risk factors
4) Management aspects(with special emphasis on beta blockers and thrombolytic therapy)
5) Complications (from the day of admission to 30^{th} day)
6) Outcomes

3.MATERIALS AND METHODS

In this, observational cross sectional analytical study total 107 cases of acute myocardial infarction admitted in Intensive Coronary Care Unit of Coimbatore Medical College Hospital during 1st January 2006 to 30th June 2007were enrolled. One-month follow up of all the cases was done. The subjects were categorized in two groups:

a) Group I- All subjects of acute myocardial infarction having age < 60 years,

b) Group II- All subjects of acute myocardial infarction with age ≥ 60 years (≥60 yrs of age was considered as elderly, as per ICMR survey on Indian rural geriatric population[1] and Acute Myocardial Infarction study in Chinese population[2]).

The subjects fulfilling any of the following two criteria out of three were included in study[3].

1) Typical symptoms (Chest discomfort).

2) Typical pattern of ECG - ST segment elevation of ≥ 0.1mv in at least two consecutive leads (ST segment elevation at the J-point with cut-off points ≥0.2mV in V1 through V3 and ≥0.1mV in other leads) or abnormal Q waves (>40ms) in at least two contiguous leads or fresh left bundle branch block.

3) Elevated enzyme levels (Serum CPK-MB two times the upper limit of normal level).

Subjects of stable and unstable angina were excluded.

Subjects fulfilling the inclusion criteria were analyzed. A proforma was used to record the patient variables including name, age, sex, and address. A detailed history was recorded giving special emphasis to atypical symptoms and risk factors. History of hypertension, diabetes, smoking, dyslipidemia, similar illness in the family and others were noted.

A detailed clinical examination was carried out. Investigations carried out included urine analysis, complete hemogram, fasting and post meal blood sugar estimations, blood urea, sr.creatine, sr. electrolytes, lipid profile, SGOT level, CPK-MBlevel, chest x-ray and 2-D echo and Doppler study done for Left Ventricular Ejection Fraction and complications of myocardial infarction. Cases were graded as per Killip's classification[4] (Table 3.1).

Table 3.1: Killips' classification for severity of myocardial infarction

Killip class	Features
Class I	No signs of pulmonary or venous congestion.
Class II	Moderate heart failure – rales at lung bases, S_3gallop, tachypnea or signs of failure of right side of the heart.
Class III	Severe heart failure, pulmonary edema.
Class IV	Shock with systolic pressure <90 mmHg and evidence of peripheral vasoconstriction, peripheral cyanosis, mental confusion and oliguria.

Hypertension is defined as a previous record of at least two blood pressure recording of ≥140/90 or the requirement of regular intake of antihypertensive drug(s) [5]. Diabetes is defined as fasting plasma glucose of ≥126mg/dl, a random glucose level of≥200mg/dl with symptoms of diabetes, or the requirement of regular hypoglycemic drug(s)[6].

Patients who required regular use of antihypertensive agent(s) and hypoglycemic agent(s) on discharge from hospital (excluding the acute phase

changes and low doses of beta blockers and ACE inhibitors which are used to reduce the post myocardial infarction mortality and morbidity) were also regarded as hypertensives and diabetics respectively.

Obesity is defined as a body mass index of ≥30. All the patients were classified according to their body mass index[1](Table 3.2).

Table 3.2: WHO classification of body mass index:

Sr.No.	Classification	BMI
1.	Underweight	<18.50
2.	Normal	18.50-24.99
3.	Overweight	≥25.00
	✓ Pre-obese	25.00-29.99
	✓ Obese class I	30.00-34.99
	✓ Obese class II	35.00-39.99
	✓ Obese class III	≥40.00

Family history is recorded as a risk factor if there is a history of coronary heart disease in male first-degree relative of <55 years or in female first-degree relative of <65 years[4].

Dyslipidemia is defined as LDL Cholesterol level of ≥100mg/dl or HDL Cholesterol <40mg/dl or non-HDL Cholesterol ≥130mg/dl (non-HDL Cholesterol=Total Cholesterol – HDL Cholesterol) [7].

Patients were recognized as non-smoker if they never smoked or they gave-up smoking ≥15years back[4], as an ex-smoker if they stopped smoking for >3 months, and as a current smoker if they still smoked within last 3 months.

The complications like cardiogenic shock, heart blocks, arrhythmias, left ventricular failure at the time of admission were recorded. Ventricular premature contractions were graded as per Lown's grading system[8]. For all cases, the treatment received during in-hospital stay and one month follow-up period was noted in the proforma. The indications and contraindications for thrombolytic therapy (Inj. Streptokinase) and beta blockers were noted in particular.

All the cases were followed during stay in hospital (up to 7 days or discharge whichever was later) and one month after discharge from the hospital for various complications.

Table 3.3: Lown's grading of Ventricular Premature Contractions

Grade	Description of VPC
0	None
1	<30/hr
2	>30/hr
3	Multiform VPC
4A 4B	2 consecutive 3 or more consecutive
5	R-on-T Phenomenon

4. REVIEW OF LITERATURE

EPIDEMIOLOGY

No universal definition of "elderly" and no accurate biomarkers for ageing exist. The World Health Organization uses 60 years of age to define "Elderly" and most United States classification use the age of 65 years[9]. For the year 2003 the Sample Registration System of India estimates are 7.2 percent of total population were above the age of 60 years(around 7,77,38,4000 individuals)[1] . Clinically recognized or unrecognized myocardial infarction occurs in 35% of elderly persons; 60% of hospitalizations due to acute myocardial infarction occur in persons ≥65[10].

ATHEROSCLEROSIS[4, 11, 12]

Atherosclerosis is a disease of large and medium-sized muscular arteries and is characterized by endothelial dysfunction, vascular inflammation, and the buildup of lipids, cholesterol, calcium, and cellular debris within the intima of the vessel wall. This buildup results in plaque formation, vascular remodeling, acute and chronic luminal obstruction, abnormalities of blood flow and diminished oxygen supply to target organs.

A complex and incompletely understood interaction exists between the critical cellular elements of the atherosclerotic lesion (Figure.4.1).

These cellular elements are endothelial cells, smooth muscle cells, platelets, and leucocytes. Vasomotor function, the thrombogenicity of the blood vessel wall, the state of activation of the coagulation cascade, the fibrinolytic system, smooth muscle cell migration and proliferation, and cellular inflammation are complex and interrelated biological processes that contribute to atherogenesis and the clinical manifestations of atherosclerosis. Oxidized LDL may be involved as an initiator of atherosclerosis (Figure.4.2).

Atherosclerotic plaque may require 10-15 years for full development. Further growth is determined by the local activity of regulatory substances (i.e., interleukin (IL)–1, IL-6, transforming growth factor-beta) and by thrombin, leukotriene, prostaglandin, fibrin, and fibrinogen. The mechanisms of atherogenesis remain uncertain. The "response-to-injury" theory is most widely accepted. Endothelial injury causes vascular inflammation and a fibroproliferative response ensues. Probable causes of endothelial injury include oxidized LDL- C, infectious agents, toxins, including the byproducts of cigarette smoking, hyperglycemia, and hyperhomocystinemia.

Figure 4.1: Postulated steps in the pathogenesis of atherosclerosis

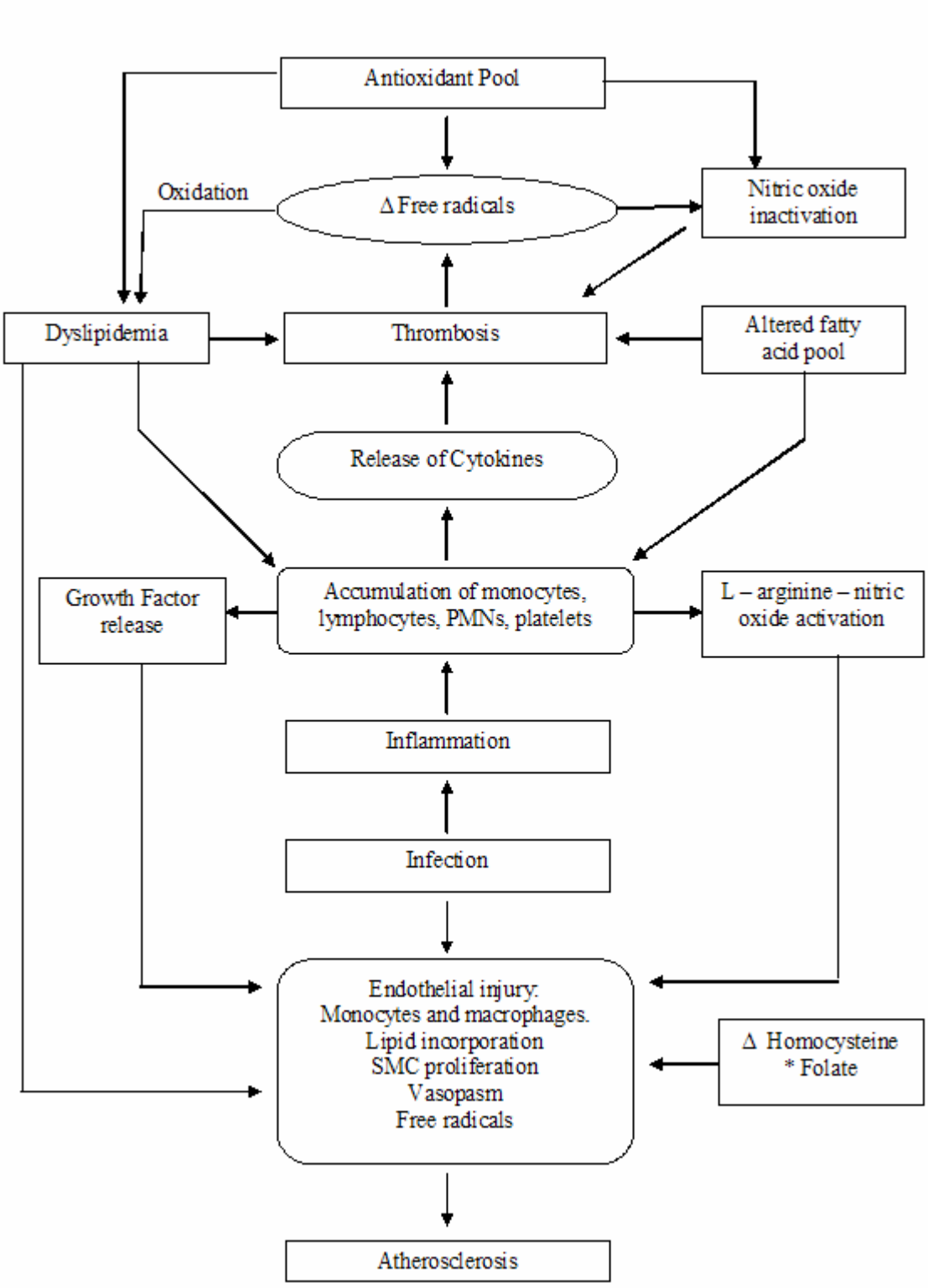
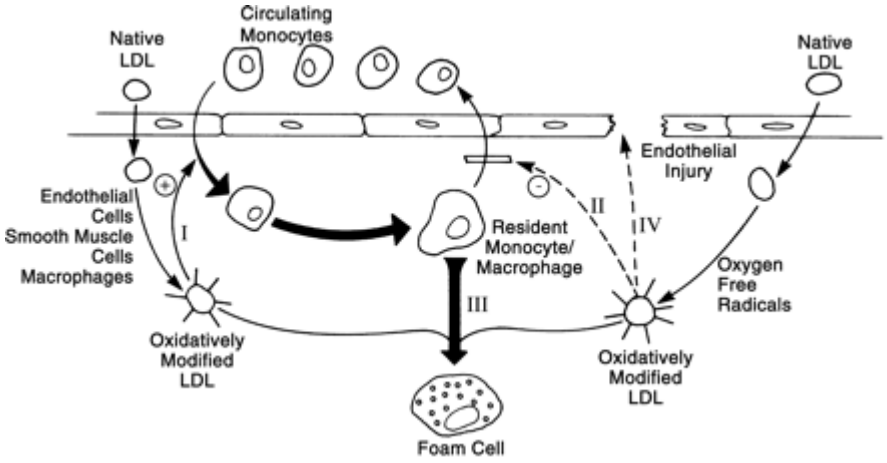


Figure: 4.2. Schema showing how oxidized LDL might be involved as an initiator of atherosclerosis



Although a logical conclusion is that the most severely stenotic lesions are the ones at the greatest risk of sudden occlusion, this is not the case. As previously described, acute coronary syndrome has been shown to more often develop because of rupture and thrombosis of mild (<60%) coronary stenoses. This occurs because of the relatively higher lipid content of the lipid core, the thinner fibrous cap, and the increased leukocyte activity at the shoulder regions of the plaque. These characteristics make such plaques, called the vulnerable plaques, much more prone to rupture.

Histopathology of atherosclerotic lesions

- Stary I lesion: The endothelium also expresses surface adhesion molecules E selectin and P selectin, attracting more polymorphonuclear cells and monocytes in the subendothelial space.
- Stary II lesion: Macrophages begin to take up large amounts of LDL (Fatty streak-Figure.4.3).
- Stary III lesion: As the process continues, macrophages eventually become foam cells.
- Stary IV lesion: Lipid exudes into the extracellular space and begins to coalesce to form the lipid core.
- Stary V lesion: Smooth muscle cells and fibroblasts move in, forming fibroatheromas with soft inner lipid cores and outer fibrous caps.
- Stary VI lesion: Rupture of the fibrous cap with resultant thrombosis causes acute coronary syndrome.
- Stary VII and VIII lesions: As lesions stabilize, they become fibrocalcific (Stary VII lesion) and, ultimately, fibrotic with extensive collagen content (Stary VIII lesion).

RISK FACTORS

The etiology of ischemic heart disease is multifactorial. Apart from the obvious ones such as increasing age and male sex, studies have identified several important risk factors (i.e., factors that make the occurrence of the disease more probable). Some of the factors are modifiable, others immutable (Table.4.1).

Table 4.1: Risk factors for atherosclerosis[1]

Not modifiable	Modifiable	Newer
Age	Cigarette smoking	C-reactive protein
Sex	Systemic hypertension	Homocysteine
Family history	Diabetes	Fibrinogen
Genetic factors	Dyslipidemia	Lipoprotein (a)
Personality (?)	Obesity	
	Sedentary habits	
	Stress	
	Infection (?)	

1. **AGE**: Age is the strongest risk factor for the development of coronary artery disease (≥ 45 for men; ≥ 55 for women). Elderly persons experience higher mortality and morbidity rates from coronary artery disease. Complication

rates of multiple therapeutic interventions tend to be higher in elderly; however, the magnitude of benefit from the same interventions is greater because these patients form the high-risk subgroup.

2. **SEX**: Atherosclerosis is more common among men than women. The higher prevalence of atherosclerosis in men is thought to be due to the protective effects of the female sex hormones. Nevertheless, the value of estrogen supplementation has been discredited by the Heart and Estrogen/Progestin Replacement Study (HERS). The presence of diabetes eliminates the protection associated with female sex. The incidence of coronary heart disease among women parallels that of men, but women demonstrate an approximately 10-year chronological delay in the onset of clinical manifestations.
3. **GENETIC FACTORS**: Family History of premature atherosclerosis (in male relatives before age 55 or in female relatives before age 65) is known to increase the risk of premature death. Genetic factors are probably the most important determinants of a given individuals Total Cholesterol and LDL level. However the importance of genetic factors in the majority of cases is largely unknown.
4. **HYPERTENSION** is strongly correlated with risk of atherosclerosis, particularly for the elderly. For men and women of all ages, the overall risk

of atherosclerosis is 2 to 3 times higher in persons with moderate to severe hypertension than in those with normal blood pressure; risk is intermediate for those with mild hypertension[13]. In the past, emphasis was placed on the importance of diastolic blood pressure. Many investigators feel that systolic blood pressure is a better predictor of ischemic heart disease than is the diastolic.

5. **DYSLIPIDEMIA**--i.e., a high level of total or low-density lipoprotein (LDL) cholesterol and a low level of high-density lipoprotein (HDL) cholesterol--are strongly correlated with risk of coronary artery disease in younger men and women; however, whether the correlation is as strong in the elderly is controversial. A high triglyceride level is a marker for obesity, glucose intolerance, and a low HDL level, all of which are risk factors for coronary artery disease[14]. The 14-years experience of the seven countries study showed that serum cholesterol concentrations is an important risk factor for the incidence of ischemic heart disease at levels perhaps 220mg/dl or more[15]. Lipid tetrad index can be considered as a risk factor if it is more than 20,000mg/dl[16].
6. **CIGARETTE SMOKING:** Some people commit suicide by drowning, but many by smoking. Smoking can exacerbate ischemia because the carbon monoxide derived from cigarette smoke reduces the oxygen-carrying

capacity of hemoglobin. Nicotine and other substances derived from cigarette smoke affect vascular smooth muscles and platelets, possibly initiating thrombotic events in persons whose circulation has been compromised by atherosclerosis[17]. Smoking may trigger ventricular arrhythmias, causing sudden cardiac death in vulnerable persons, presumably by enhancing sympathetic tone and reducing the threshold for ventricular fibrillation. The prevalence of cigarette smoking appears to decrease with age, primarily because smokers tend to die before reaching old age[18].

7. **DIABETES MELLITUS**: An important risk factor for hyperlipidemia and atherosclerosis and commonly associated with hypertension, abnormalities of coagulation, platelet adhesion and aggregation, increased oxidative stress, and functional and anatomic abnormalities of the endothelium and endothelial vasomotion.
8. **OBESITY**, even at an advanced age, is a significant risk factor for atherosclerosis. Progressive increases in body weight correlate with several other risk factors for atherosclerosis: increases in blood pressure and in cholesterol, triglyceride, and blood glucose levels and a decrease in HDL cholesterol level.

9. **INFECTION AND INFLAMMATION** may be involved in the atherosclerotic process. Several microorganisms, such as herpes viruses (e.g., cytomegalovirus), Chlamydia pneumoniae, and Helicobacter pylori, have been identified in atherosclerotic plaques in coronary arteries and other tissues. Elevated titers of antibodies to these microorganisms have been used to predict the recurrence of coronary events after an acute myocardial infarction.
10. **LEFT VENTRICULAR HYPERTROPHY** detected by ECG or echocardiography increases the likelihood of atherosclerosis-related events in elderly persons.

MYOCARDIAL INFARCTION

Ischemic myocardial necrosis usually resulting from abrupt reduction in coronary blood flow to a segment of myocardium.

History

1. Acute myocardial infarction (AMI) may present in a variety of ways in older people. The clinical presentation may be classical, atypical or silent (Table 4.2). Of elderly patients with documented acute myocardial infarction ,19 to 66% present with chest pain, 20 to 59% with dyspnoea, 15 to 33% with

neurologic symptoms, and 0 to 19% with gastrointestinal symptoms (e.g., epigastric distress, vomiting, nausea, heartburn, indigestion)[19,20].

Table 4.2: Clinical presentation of acute myocardial infarction in elderly[21]

Classical	Atypical	Silent
Pain Breathlessness	Acute confusion Giddiness Syncope Stroke Palpitation Abdominal Pain Vomiting	No symptoms

2. Elderly patients with acute myocardial infarction tend to delay longer than younger patients in seeking medical assistance after the onset of chest pain or other presenting symptoms of myocardial infarction[22].

3. Atypical presentations of acute myocardial infarction are often encountered in older people and the incidence of painless infarction increases with advancing age[23]. It is not clear why elderly infarct patients appear to report

less chest pain although a number of explanatory hypotheses have been proposed

a. age associated increased threshold of pain

b. overriding symptoms of breathlessness or other medical conditions

c. confusion

d. denial of symptoms

e. defective anginal warning system

f. autonomic system dysfunction with sensory nerve damage[24]

Physical[4, 12, 25]

1. Pulse volume, rate, and regularity: Tachycardia is common in persons with acute coronary syndrome and acute myocardial infarction. Heart rate irregularity may signal the presence of atrial fibrillation or frequent supraventricular or ventricular ectopic beats. Ventricular tachycardia is the most common cause of death for persons with acute myocardial infarction.

2. High or low blood pressure: Hypotension often reflects hemodynamic compromise and is a predictor of poor outcome in the setting of acute myocardial infarction.
3. Diaphoresis: This is a common finding.
4. Tachypnoea: Patients often have rapid breathing.
5. Shock
6. Syncope
7. Leg edema
8. Congestive heart failure: Signs and symptoms of heart failure may indicate cardiogenic shock or a mechanical complication of acute myocardial infarction such as ischemic mitral valve regurgitation.
9. Heart sounds and gallop: An S_4 gallop is a common early finding. The presence of an S_3 is an indication of reduced left ventricular function.
10. Heart murmurs: These, particularly those of mitral regurgitation and ventricular septal defect, may be found after the initial presentation; their presence indicates a grave prognosis. The murmur of aortic insufficiency may signal the presence of aortic dissection as a primary etiology, with or without the complication of acute myocardial infarction.
11. Pulmonary congestion, rales

12. Stigmata of risk factors: Patients may develop xanthelasmas, livedo reticularis, or both.
13. Body habitus: Central obesity is often seen.
14. Diagonal ear crease, short stature, baldness, thoracic hairiness
15. Findings consistent with previous coronary artery disease: These patients may have scarring from coronary artery bypass graft or similar surgeries

INVESTIGATIONS

1. Routine investigations
    a. Urine analysis
    b. CBC count
    c. Chemistry panel(fasting and post prandial blood sugar, blood urea, serum creatinine, serum electrolytes, serum magnesium if needed)
    d. Thyroid function tests - To exclude thyroid disorders
2. Fasting lipid profile
    a. Total cholesterol level
    b. LDL-C level

c. HDL cholesterol (HDL-C) level

d. Triglyceride level

3. ECG [26]

   a. T waves: Peaked upright T waves may be the first ECG manifestation of myocardial infarction

   b. ST segment changes:

      i. Convex ST segment elevation ≥1mm in two consecutive leads with peaked or inverted T waves usually is indicative of myocardial injury

      ii. Posterior wall infarction is recognized by ST segment depression in leads V1toV3

      iii. Right Ventricular infarction is diagnosed with ST segment elevation in V4R

   c. Q waves: Development of new pathological Q waves (>40ms)

   d. New Left Bundle Branch Block

4. Serum markers[26]

   a. Creatine kinase with MB isozymes: Its level increases within 3-12 hours of chest pain, peak at 24 hours, and return to baseline after

48-72 hours. It has >95% sensitivity and specificity for myocardial injury.

b. Troponins (I or T): It is the preferred biomarker for the diagnosis of ST segment elevation myocardial infarction. Their level increases 3-12 hours of chest pain, peat at 24-48 hours, and return to baseline after 5-14 days.

c. Lactate dehydrogenase and lactate dehydrogenase isozymes

d. Serum aspartate aminotransferase

5. Chest X-ray: When rales are detected, a chest x-ray may be indicated to document left-sided heart failure. When pulmonary hypertension is suspected, a chest x-ray can confirm the diagnosis by showing large central pulmonary arteries with oligemic lung fields. Aortic knob calcification is detected in about 30% of the elderly but has no pathologic significance. In contrast, intracardiac calcification, most commonly on the aortic valve and the mitral annulus, is almost always pathologic; it signifies valvular stenosis. In the elderly, coronary artery calcification, best seen with fluoroscopy, does not necessarily signify major stenosis, as it does in younger patients. Calcification of the pericardium (due to constrictive pericarditis) or the left ventricular wall (due to an old myocardial infarction) is occasionally also seen.

6. Special tests
    a. Specific lipid studies (if necessary)
        i. Small, dense LDL-C level
        ii. Apoprotein profile
    b. Miscellaneous tests
        i. Homocysteine level
        ii. Inflammatory markers (e.g., CRP)
7. Imaging Studies:
    a. Echocardiography
        i. Transthoracic echocardiography helps assess left ventricular function, wall motion abnormalities in the setting of acute coronary syndrome or acute myocardial infarction, and mechanical complications of acute myocardial infarction.
        ii. Transesophageal echocardiography is most often used for assessing possible aortic dissection in the setting of acute myocardial infarction.
        iii. Stress echocardiography can be used to evaluate hemodynamically significant stenoses in stable patients who are thought to have coronary artery disease.

iv. Treadmill echocardiography stress testing and dobutamine echocardiography stress testing provide equivalent predictive values.

b. Nuclear imaging studies (myocardial perfusion imaging): These studies are also useful in assessing patients for hemodynamically significant coronary artery stenoses.

c. Electron beam CT scanning: Electron beam computed tomography (EBCT) scanning is a noninvasive method of evaluating calcium content in the coronary arteries. Healthy coronary arteries lack calcium. As atherosclerotic plaques grow, calcium accumulates because of a perpetuating inflammatory process or the healing and scarring induced by this process.

d. Magnetic resonance angiography

8. Procedures:

a. Coronary angiography: Coronary arterial luminography remains the criterion standard for defining significant flow-limiting stenoses that must be revascularized through percutaneous or surgical intervention to improve prognosis. Quantitative coronary angiography (QCA) is used to perform computerized quantitative analysis of the entire coronary tree. It introduces a correction factor for the presence of

diffuse disease. QCA has been widely used in many trials of atherosclerotic progression and regression.

b. Coronary blood flow determinations: Because of the inherent limitations of coronary angiography, attention has been directed to using physiological approaches for determining the severity of coronary stenoses. The 5 methods of measuring human coronary blood flow in the cardiac catheterization laboratory are (1) thermodilution, (2) digital subtraction angiography, (3) electromagnetic flow meters, (4) Doppler velocity probes (for measuring CFR), and (5) pressure wires (for measuring fractional flow reserve [FFR]).

c. Intravascular ultrasound (IVUS) demonstrates the luminal dimensions and, more importantly, the tissue composition of the vascular wall in tomographic sub segments that can be summated to create a 3-dimensional picture showing arterial remodeling and the diffuseness of atherosclerosis with clarity unobtainable by angiography (luminography).

TREATMENT

1. Unless contraindicated, aspirin (or if contraindicated, ticlopidine or clopidogrel) should be given. The role of glycoprotein IIb/IIIa inhibitors (e.g., tirofiban, abciximab) in the treatment of elderly patients with acute myocardial infarction is under study[28].

2. For chest pain associated with acute myocardial infarction, morphine sulfate 2 to 4 mg IV is the drug of choice, but nitrates and β-blockers may also be used[29].

3. Oxygen may be given but is unlikely to help if the oxygen saturation exceeds 94%. For heart failure, oxygen therapy, furosemide 20 to 80 mg IV, and nitrates may be used. For hypotension, IV fluids or dobutamine may be needed.

4. Subcutaneous heparin 7500 U q 12 h should be given to reduce the incidence of venous thromboembolism after treatment of acute myocardial infarction.

5. The role of intravenous heparin in treatment of these patients is controversial, and that of low-molecular-weight heparin is under study[27].

6. Unless contraindicated, a β-blocker should be given to patients at admission. It is given intravenous initially, then orally. Such therapy can reduce the mortality rate by 23% in elderly patients who have had a myocardial infarction. Risk of recurrent myocardial infarction is also reduced. Early intravenous β-blocker therapy may be given with or without thrombolytic therapy.

7. Nitrates probably do not reduce mortality in patients with acute myocardial infarction but may be used for treatment of chest pain and heart failure[30].

8. Early and continued use of an angiotensin-converting enzyme (ACE) inhibitor is recommended for patients with acute myocardial infarction who are hemodynamically stable (systolic blood pressure ≥100 mm Hg) and who have heart failure, a large anterior myocardial infarction, or a left ventricular ejection fraction of ≤ 40%. Such therapy can reduce the risk of death, severe heart failure, and severe left ventricular systolic dysfunction. The reduction in risk of death and severe heart failure is greater among elderly than among younger patients. When ACE inhibitor therapy is initiated during acute myocardial infarction, the patient's blood pressure, renal function, and serum potassium should be closely monitored.

9. Use of calcium channel blockers (except amlodipine) during acute myocardial infarction is not recommended, because these drugs do not benefit and may harm patients with acute myocardial infarction. Switching elderly patients who have been taking calcium channel blockers to β-blockers may be advisable[31].

10. The use of intravenous magnesium during acute myocardial infarction is controversial. Magnesium should not be routinely used in the treatment of elderly patients with acute myocardial infarction.

11. The prophylactic use of antiarrhythmic drugs other than β-blockers does not improve the clinical outcome in patients with acute myocardial infarction. In a meta-analysis, prophylactic lidocaine, once commonly used, did not improve survival and was associated with an increased incidence of asystolic cardiac arrest. Also, the elderly are at increased risk of lidocaine toxicity. Therefore, the use of lidocaine during acute myocardial infarction should be limited to treatment of patients with life-threatening ventricular arrhythmias. Patients with supraventricular tachyarrhythmias may be treated with β-blockers or direct-current cardioversion.

12. The need for a pacemaker may be temporary (during acute myocardial infarction by transvenous pacing) or permanent (after acute myocardial infarction).

13. Reperfusion therapy (thrombolytics or Percutaneous Coronary Angioplasty) during acute myocardial infarction can reduce the absolute and percent mortality rate more among elderly patients than among younger patients. In patients > 80 who had had a myocardial infarction, the mortality rate was 41% lower for patients given streptokinase than for those given placebo, and 14.1 lives per 100 treated patients were saved. Medical therapy alone is preferred for most elderly patients who have had a myocardial infarction, but if revascularization is indicated, Percutaneous Coronary Angioplasty is generally preferred to Coronary Artery Bypass Graft.

14. Unless contraindicated, reperfusion therapy should be considered for elderly patients who have ischemic symptoms that last ≥ 30 minutes and that occur within 6 to 12 hours of clinical presentation and who have an ST-segment elevation of at least 1 to 2 mm in ≥2 ECG leads or who have left bundle branch block. Elderly patients with persistent myocardial ischemia, hypotension, or cardiogenic shock occurring > 12 hours after the onset of symptoms may still benefit from Percutaneous Coronary Angioplasty.

Intracoronary stent placement decreases the risk of restenosis after Percutaneous Coronary Angioplasty. The potential benefit of reperfusion therapy is higher for elderly patients with a large anterior acute myocardial infarction.[32]

15. Streptokinase (1.5 million U IV over 1 hour) may be preferable to recombinant human tissue plasminogen activator (rt-PA) for the elderly because it causes fewer episodes of stroke and cerebral hemorrhage; it is also less expensive. However, the choice of thrombolytic drug is controversial. Minimization of delays in treatment is more important than drug choice[28, 33].

LONG-TERM MANAGEMENT[31, 34, 35]

1. Modifiable risk factors should be controlled. Long-term drug therapy may include antiplatelet drugs, anticoagulants, β-blockers, nitrates, and ACE inhibitors. Calcium channel blockers are generally avoided; use of a calcium channel blocker instead of a β-blocker after myocardial infarction doubled the risk of mortality in one study. Automatic implantable cardioverter-defibrillators and surgical treatment (revascularization with Percutaneous

Coronary Angioplasty or Coronary Artery Bypass Graft) are appropriate for some patients.

2. Long-term use of the β-blockers propranolol, timolol, and metoprolol reduces rates of recurrent myocardial infarction and sudden cardiac death more in elderly patients than in younger ones.

3. Class I antiarrhythmic drugs and d-sotalol (not clinically available) increase mortality rates after myocardial infarction and should not be used in elderly patients after myocardial infarction. Amiodarone and d, l-sotalol (which is available) do not significantly affect mortality rates after myocardial infarction. β-Blockers are the only antiarrhythmic drugs that reduce mortality rates in elderly patients with nonsustained ventricular tachycardia or complex ventricular arrhythmias after myocardial infarction; unless specifically contraindicated, β-blockers should be used to treat these patients after myocardial infarction.

4. Because estrogen replacement therapy does not appear to improve outcome after myocardial infarction and may increase the incidence of venous thromboembolic events and of gallbladder disease, it is not recommended for postmenopausal women who have had a myocardial infarction.

COMPLICATIONS

The majority of elderly patients with proven acute myocardial infarction have arrhythmias or conduction disorders but only a minority of these is of any clinical significance. Complications such as heart failure, heart block and shock picture are more prevalent with advancing age (Table 4.3)[36] but no significant age difference was noted in the incidence of pericarditis, stroke disease or pulmonary thromboembolism in one large CCU study[36, 37].

When the left ventricular free wall, papillary muscle, or interventricular septum ruptures after acute myocardial infarction, mortality risk is very high unless surgical repair is promptly performed.

Table 4.3: Complications of acute myocardial infarction in younger (<60years) and elderly (≥60years)

Complication	<60 years	≥60 years
Atrial fibrillation or flutter	5%	14%
Heart block or conduction defects	20%	35%
Left ventricular failure	47%	74%
Right heart failure	6%	22%
Shock	14%	25%

PROGNOSIS

1. Clearly the reported severity and mortality of acute myocardial infarction in older patients depends upon the selection of the patients in the studies concerned. Since 1969 an increasing proportion of patients admitted to coronary care units have been over 65 years of age and their mortality rates have been falling [38] (Table4.4).

2. Most deaths are due to shock or cardiac rupture. Adverse prognostic factors include age, previous myocardial infarction, hypertension, diabetes mellitus, cardiomegaly and heart failure[39].

3. Elderly patients are more liable to cardiovascular complications associated with respiratory problems, renal failure or metabolic upsets.

4. In elderly survivors of acute myocardial infarction, the expected mortality in the first year is 20-30% and thereafter 5-10% each year in the ensuing nine years[40, 41, 42].

Table. 4.4: Hospital Mortality of AMI over 70 years of age

Study	Number of patients	Mortality
Nonis et al. 1970	212	42%
Williams et a. 1976	104	46%
Latting and Silverman.1980	175	32%
MacDonald et al. 1983	317	16%
Hopper et al. 1989	269	24%

5.OBSERVATION

1. Age and sex

The present study comprised of total 107 cases of acute myocardial infarction of which 52(49%) belonged to group I (age <60 years) and 55(51%) belonged to group II (age ≥60 years). Mean age of the cases was 54.3 and 65.6 in group I and II respectively. The male and female ratio was 4:1 in group I and 2.3:1 in group II indicating occurrence of myocardial infarction is more in elderly female population (Figure 5.1&5.2)

FIGURE 5.1: AGE INCIDENCE

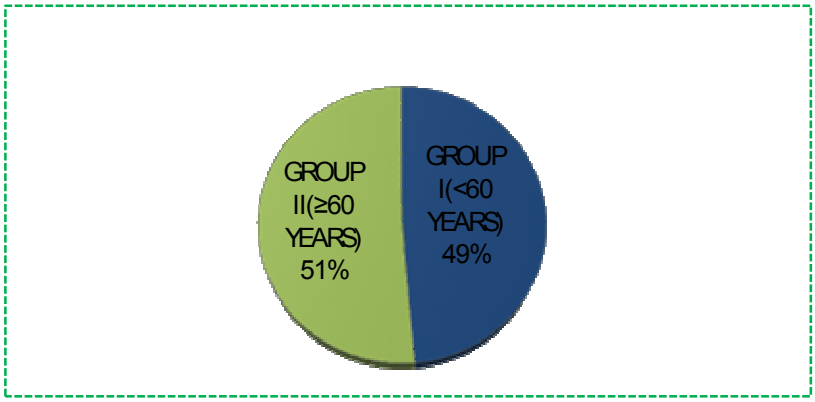
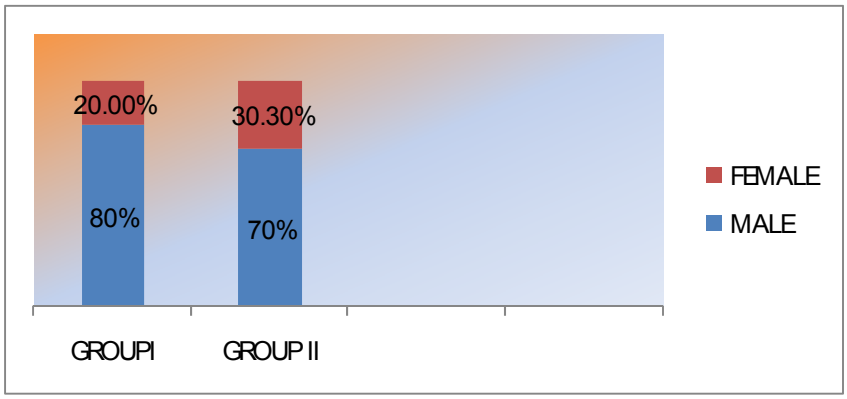


FIGURE 5.2: SEX INCIDENCE



2. Symptoms

In the analysis of symptomatology atypical chest pain, sweating, dyspnoea and giddiness were observed predominantly in the elderly group with acute myocardial infarction than group I (Table 5.1 and Figure 5.3). The number of elderly subjects arriving within 6 hours of chest pain was significantly less as compared to young subjects (43/52 i.e. 82.7% vs. 25/55 i.e. 45.4%).

FIGURE 5.3: SYMPTOMATOLOGY

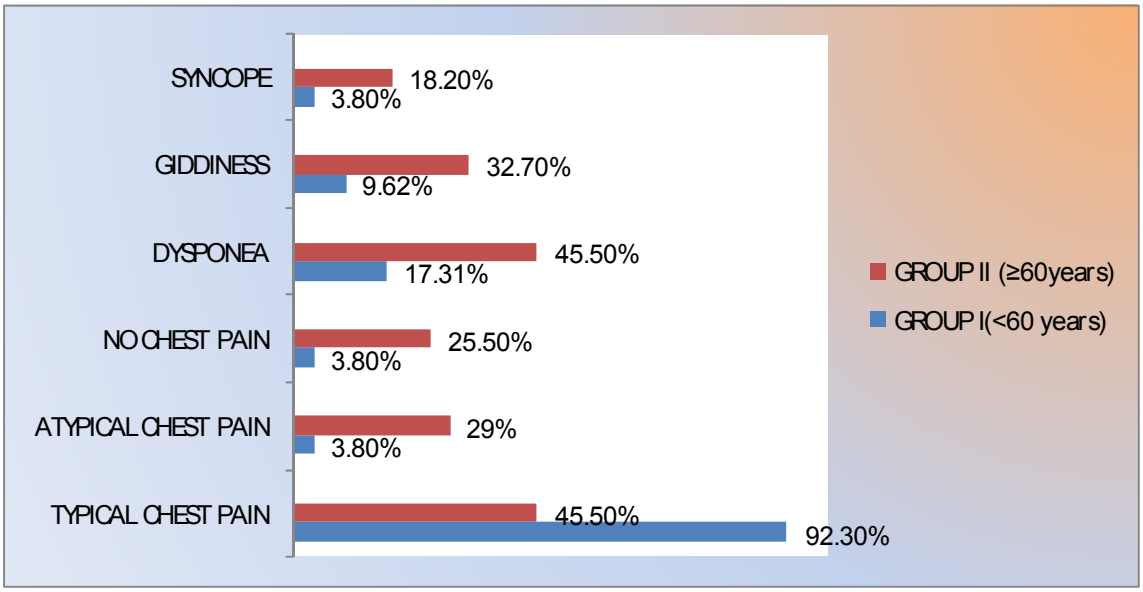


Table 5.1: Incidence of frequency of presenting symptoms

Sr.no	Symptoms	Group I (<60years) (n=52)	Group II (≥60years) (n=55)
1.	Typical chest pain	48(92.3%)	25(45.5%)
2.	Atypical chest pain	02(3.8%)	16(29.1%)
3.	No chest pain	02(3.8%)	14(25.5%)
4.	Sweating	31(59.6%)	30(54.5%)
5.	Nausea and/or vomiting	19(36.5%)	21(38.2%)
6.	Dysponea	09(17.3%)	25(45.5%)
7.	Giddiness	05(9.6%)	18(32.7%)
8.	Syncope	02(3.8%)	10(18.2%)
9.	Palpitation	03(5.8%)	10(18.2%)
10.	Altered sensorium	03(5.8%)	04(7.3%)
11.	Abdominal pain	01(1.9%)	03(5.5%)
12.	Focal neurological deficit	00	01(1.8%)

3. Risk factors

Among the risk factors, hypertension was commonly seen in elderly (group II) and in 20 (36.4%) elderly subjects no risk factor was found (Table 5.2).

Table 5.2: Comparison of risk factors

Sr.no	Risk factors	Group I (<60years) (n=52)	Group II (≥60years) (n=55)
1.	Hypertension	19(36.5%)	28(50.9%)
2.	Diabetes mellitus	10(19.2%)	10(18.2%)
3.	Smoking	30(57.7%)	16(29.1%)
4.	Dyslipidemia	08(15.4%)	07(12.7%)
5.	Obesity	13(25.0%)	05(9.1%)
6.	Family history	13(25.0%)	03(5.5%)
7.	No risk factors	09(17.3%)	20(36.4%)

4. Treatment

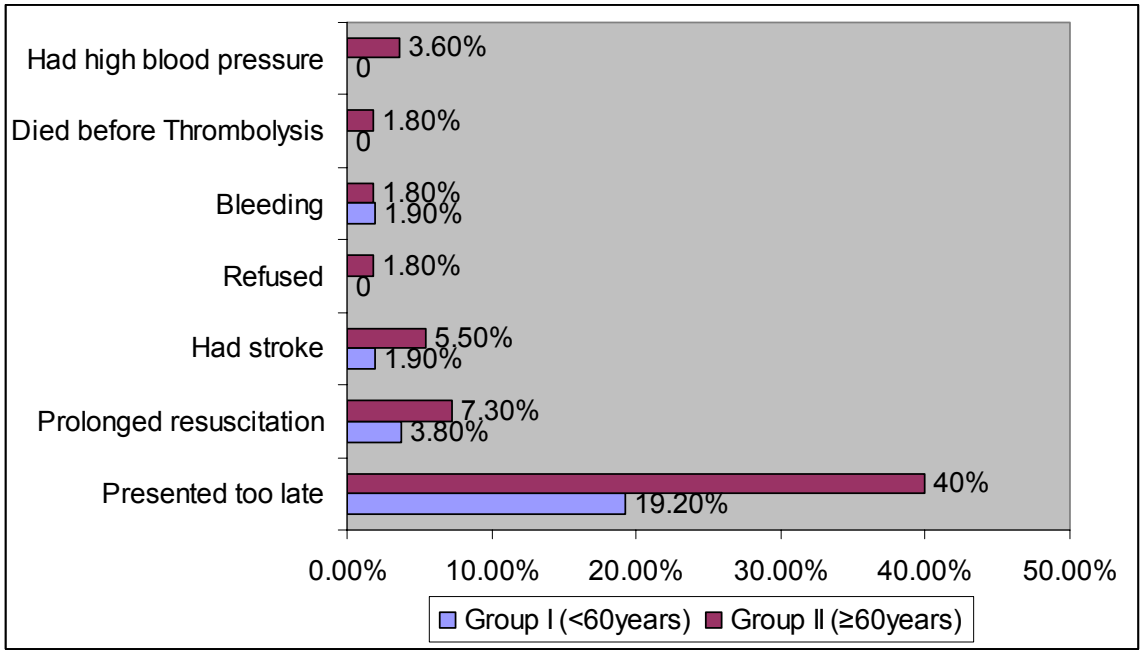
Assessment of treatment received by cases during in hospital stay revealed that thrombolytic therapy was under used in elderly group (II) only in 21 (38.2%) cases as compared to 38 (73.1%) in group (I). Among the other drugs used in the treatment of acute myocardial infarction and its complications (Aspirin, Nitroglycerin, Heparin, Beta-blockers, ACE-inhibitors, Calcium channel blockers, Lipid lowering agents, and Diuretics), only 10 (18.2%) cases

from elderly group (II) received beta-blockers as compared to 44 (84.6%) cases from group (I) (Table 5.3 and Figure 5.4).

Table 5.3: Comparison of treatment

Sr.no	Treatment	Group I (<60years) (n=52)	Group II (≥60years) (n=55)
1.	Thrombolytic therapy	38(73.1%)	21(38.2%)
2.	Beta blockers	44(84.6%)	10(18.2%)
3.	Reasons for non thrombolysis		
	✓ Presented too late	10(19.2%)	22(40%)
	✓ Prolonged resuscitation	02(3.8%)	04(7.3%)
	✓ Had stroke	01(1.9%)	03(5.5%)
	✓ Refused	0	01(1.8%)
	✓ Bleeding	01(1.9%)	01(1.8%)
	✓ Died before thrombolysis	0	01(1.8%)
	✓ Had high blood pressure	0	02(3.6%)

FIGURE 5.4: REASONS FOR NON THROMBOLYSIS



5. Early complications(0 to 7 days)

Assessment of early complications of acute myocardial infarction during the time of hospitalization revealed that 35 (63.6%) cases from group II presented with heart failure at the time of admission in ICCU as compared to only 21 (40.4%) cases in-group I (Table 5.5). 28 (50.9%) cases from group II had arrhythmias during in hospital stay compared to only 14 (26.9%) from group I. AV block was seen in 15 (27.3%) cases from elderly group as compared to 4 (7. 7%) cases from group I (Table 5.4).

Table 5.4: Various arrhythmias observed during 7 days follow up

Sr.no	Arrhythmias	Group I (<60years) (n=52)	Group II (≥60years) (n=55)
1.	Ventricular premature contractions (Lown's grading) I II III IV V	05(9.6%) 02 01 02 00 00	14(25.5%) 05 04 03 02 00
2.	Atrial fibrillation	00	02(3.6%)
3.	Atrio ventricular block 1st degree 2nd degree 3rd degree	04(7.7%) 02 02 00	15(27.3%) 06 04 05
4.	RBBB	02(3.8%)	01(1.8%)
5.	LBBB	02(3.8%)	01(1.8%)
6.	VT/VF	01(1.9%)	02(3.6%)

Table 5.5: Complication of acute myocardial infarction on 7 days follow up

Sr.no	Complications	Group I (<60years) (n=52)	Group II (≥60years) (n=55)
1.	Heart failure	21(40.4%)	35(63.6%)
2.	Cardiogenic shock	01(1.9%)	09(16.4%)
3.	Arrhythmias	14(26.9%)	28(50.9%)
4.	Reinfarction	00	01(1.8%)
5.	Cardiac arrest	07(13.7%)	06(10.9%)
6.	Cerebro vascular accidents	00	05(9.1%)
7.	Bleeding complications	01(1.9%)	03(5.5%)
8.	Death	05(9.6%)	18(32.7%)

6. Late complications (8 to 30 days)

When one month follow up of all the cases of acute myocardial infarction was done, mortality, heart failure, post myocardial infarction angina were the common complications observed in elderly group (Table 5.6).

Table 5.6: Late complications

Sr.no	Symptoms	Group I (<60years) (n=52)	Group II (≥60years) (n=55)
1.	Death	00	04(7.3%)
2.	Post myocardial infarction angina	02(3.8%)	04(7.3%)
3.	Heart failure	01(1.9%)	05(9.1%)
4.	Cardiogenic shock	00	02(3.6%)
5.	Fresh myocardial infarction	00	02(3.6%)
6.	Cerebro vascular accidents	00	01(1.8%)

7. Mortality

Mortality was found highly significant in elderly population group (II) than young population with acute myocardial infarction 22(3940%) vs. 0 5 (9.6%) (Table 5.7).

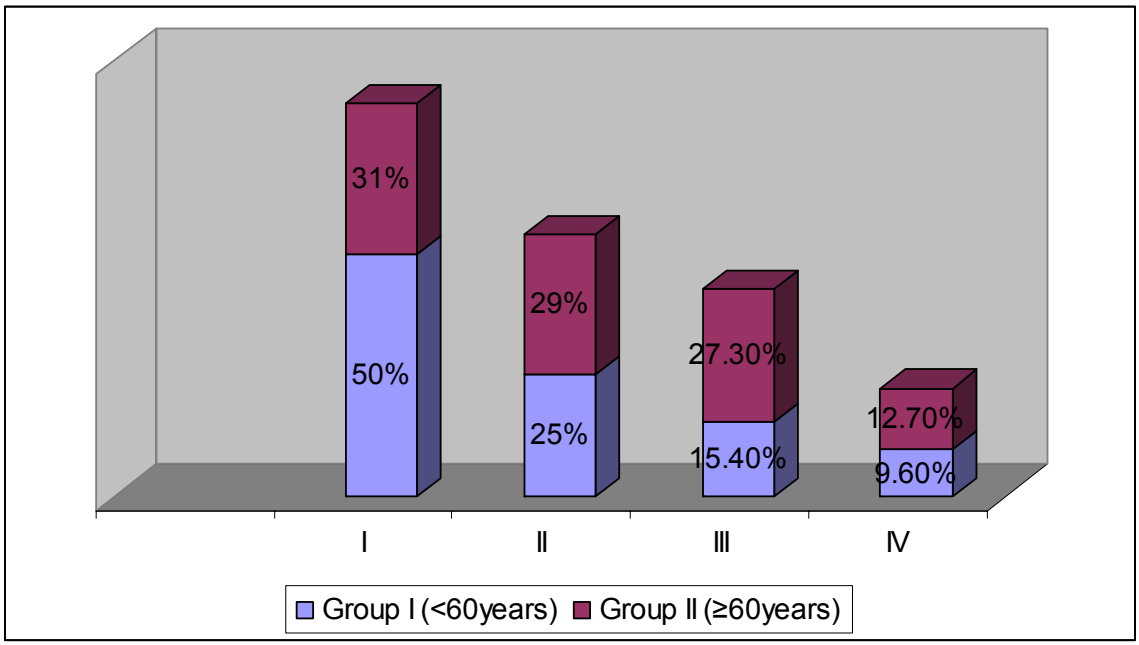
Table 5.7: Distribution of Mortality as per different time intervals

Sr.no	Time of death	Group I (<60years) (n=52)	Group II (≥60years) (n=55)
1.	<24 hours	04(7.7%)	12(21.8%)
2.	≥24hours to 7 days	01(1.9%)	06(10.9%)
3.	8th day to 30th day	00	04(7.3%)

8. Killip class:

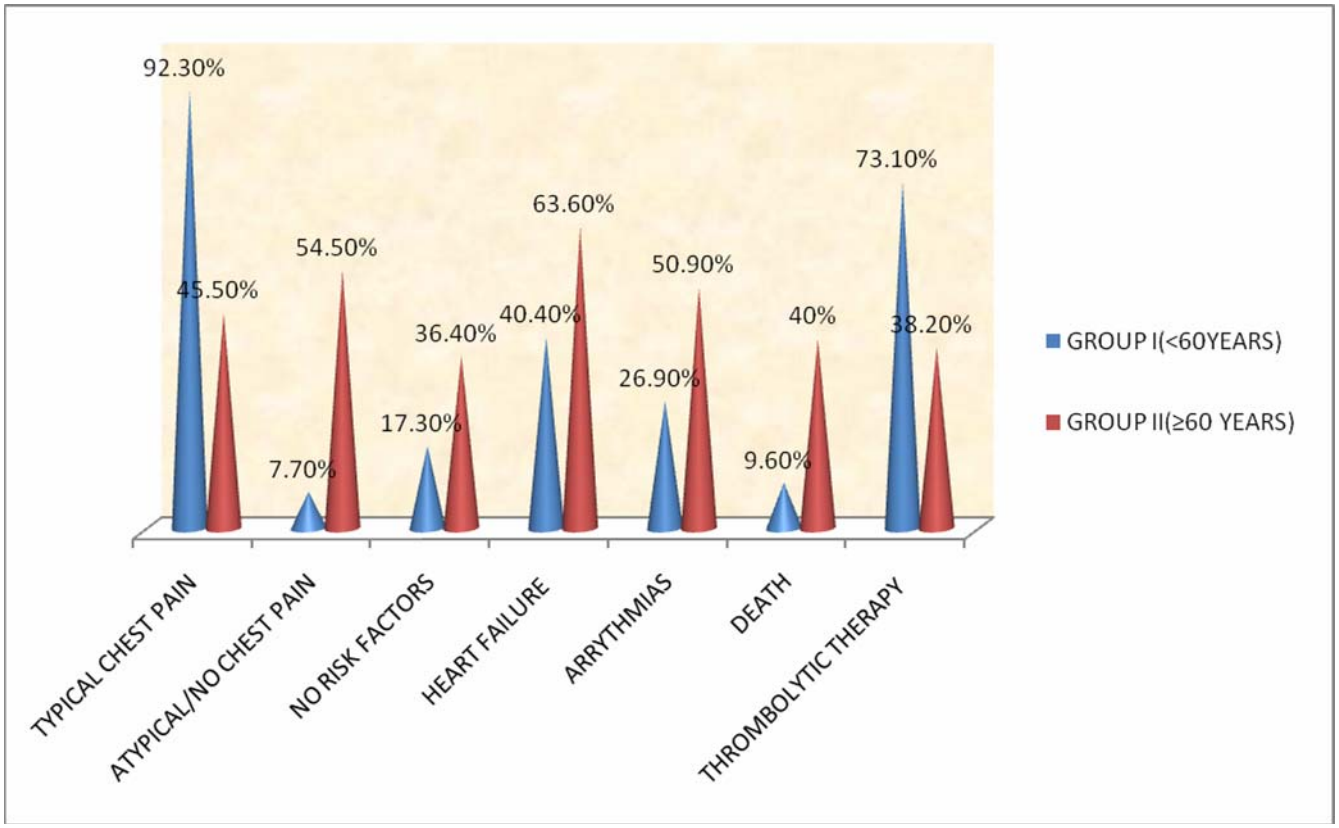
High Killip class was found in elderly population than young population (Figure 5.5)

FIGURE 5.5: KILLIP CLASS



Important features of my study are presented in Figure 5.6

FIGURE.5.6: SALIENT FEATURES BETWEEN TWO GROUPS



Interesting X-rays and ECGs are presented in Figures 5.7 to 5.13

6.DISCUSSION

1. Age and Sex

The present study shows that with increasing age the preponderance of male among patients with acute myocardial infarction admitted to the hospital decreases and sex ratio becomes smaller. This possibly reflects a higher percentage of females in an elderly population and also a very likely a more equal distribution of risk factors for acute myocardial infarction between both genders at high age[2, 43]. However, the role of hormone replacement therapy to reduce the risk of Coronary Artery Disease in postmenopausal women is still controversial. More than 50% of the patients with acute myocardial infarction belong to elderly group.

2. Symptomatology

Various authors have previously emphasized the variability in the clinical presentation of acute myocardial infarction (AMI) in the elderly[2, 44]. In the present study more cases among the elderly population had atypical chest pain (29.1%), no chest pain (25.5%), on admission to hospital as compared to young population (Group I) (3.8% and 3.8%). Dyspnoea and other nonspecific symptoms like giddiness, syncope and abdominal pain are frequently observed

in elderly patients as compared to young myocardial infarction in the present study. Such high incidence of nonspecific symptoms in elderly could be because of preoccupied non-cardiac problems, or it could be noticed that these patients are sometimes not able to describe their symptoms accurately or do not remember their complaints and possibly have increased pain threshold[45].

Knowing of the common local atypical presentations would increase our awareness in considering an acute cardiac event when the elderly present atypically. By detecting acute myocardial infarction earlier, the outcome may be improved with early interventions.

3. Risk factors

Among the risk factors evaluation, hypertension was the commonest risk factor seen in elderly than young with acute myocardial infarction in the present study while the incidence of smoking was less in group II (elderly) as compared to group I. Similar type of observations are made by various authors in their study[45, 46]. The low incidence of smoking in elderly is well explained as most of the elderly quit smoking as age advances and also number of females (postmenopausal) increases in elderly group with acute myocardial infarction

who are usually non-smokers. It has also been pointed out that cigarette smoking may be less of a risk for cardio-vascular disease in old age[47].

The present study observed no risk factor in 40.5% cases in elderly with myocardial infarction. Similar observation has also been reported by others suggesting "age" itself is a major risk factor for myocardial infarction[48].

Knowing the prevalence of various modifiable risk factors among the age groups may help in planning appropriate secondary preventive programmes to target the different age groups. Emphasis for the elderly population should be more targeted at better control of hypertension and diabetes mellitus, while for the younger population, in addition to hypertension and diabetes mellitus, smoking habits and hyperlipidemia should be emphasized.

4. At admission

A striking finding on admission during and after the ICCU stay was the high occurrence rate of heart failure and cardiogenic shock in the group II as compared to group I. This high incidence is probably related to pre existing heart disease as reflected by more frequent occurrence of hypertension, cardiomegaly or a decline in myocardial reserve[46].

It is known that heart failure is an important predictor of poor outcome after acute myocardial infarction. It is also recognized that even with "best practice" interventions, the prognosis for the established heart failure in the elderly remains poor. Also, the management of heart failure in elderly patients is often complicated by multiple co-morbid conditions, polypharmacy and the difficulty in tolerating recommended target doses of drugs.

Therefore further research should be aimed at developing more effective strategies for the prevention of heart failure in elderly patients. In the present study, the next common complications observed in elderly with myocardial infarction were ventricular premature contractions and AV blocks as compared to young with myocardial infarction. This is not related to differences in location and extent of myocardial necrosis and ischemia. In ageing persons, the atrio-ventricular conduction system is subject to spontaneous fibrosis and more vulnerable to ischemia and necrosis[49].

5. Treatment

The evaluation of in hospital treatment of acute myocardial infarction depicts that elderly patient with acute myocardial infarction were under thrombolysed and there was restricted use of b Blockers as compared to

young[50]. History of peptic ulcer, delayed arrival at hospital > 6-10 hours and H/O recurrent TIA were identified in 61.8% of elderly thrombolytic ineligible patients compared with 26.9% young with myocardial infarction. Advanced age, presence of obstructive airway disease, diabetes, heart failure has limited the use of ß blockers in the present study. Literature also supports the above observation[51, 52, and 53].

6. Early complications

During 7 days hospital follow up elderly patients with acute myocardial infarction had more complications like mortality, heart failure, arrhythmias, cardiogenic shock and stroke as compared to young. Similar data is reported in literature by other authors[54, 55].

7. Late complications

During one month follow up of all the cases; complications like post myocardial infarction angina, congestive cardiac failure, and re-infarction are observed more in elderly as compared to young. Complications like cardiogenic shock, re-infarction, cerebrovascular episode and ventricular aneurysm were observed only in elderly group. In the aged, the adaptations of cardiovascular system to stress is impaired as a consequence of anatomical,

functional and metabolic changes in the heart itself and also increase in impedance to ventricular ejection due to anatomical changes in the arterial bed and insufficient vasodilatory capacity of the peripheral vessels. These ages' related changes hamper normal ventricular functions and its adaptive mechanisms to the hemodynamic burden elicited by myocardial necrosis. This explains why ventricular dysfunction occurred more frequently in the very elderly patients before and during an acute myocardial infarction.[56, 57, 58.]

8. Mortality

In the present study, the overall mortality in elderly with myocardial infarction was found to be higher than young. Structural changes of the heart related to the process of ageing contribute to a great extent to the high early and late mortality of acute myocardial infarction in the aged[59, 60]. However age related changes in other organs and deterioration of their adaptive mechanisms to ventricular failure also play a role.

7. CONCLUSION

From the study I conclude that,

1. More than 50% of patients of acute myocardial infarction belong to elderly age group.
2. With increasing age the preponderance of male among patients with acute myocardial infarction admitted to the hospital decreases and sex ratio becomes smaller.
3. The manifestations of acute myocardial infarction are more subtle in the elderly.
4. Elderly patients with acute myocardial infarction have different risk factors.
5. The elderly subjects are under thrombolysed.
6. Beta blockers are underutilized in elderly with acute myocardial infarction.
7. The elderly patients have higher complication and mortality rate.

8. SUMMARY

From the study and literature it is well known that the incidence of coronary heart disease increases dramatically with age. Acute myocardial infarction may present in a variety of ways in old age. Chest pain is the most common presenting symptom but atypical or silent presentations are well recognized and the incidence of painless infarction increases with advancing age. Most elderly patients with acute myocardial infarction have arrhythmias or conduction defects. The severity and mortality of AMI increase in old age. The elderly benefit as much as their younger counterparts from admission to coronary care units and early treatment with streptokinase, aspirin, beta blockers and other measures. The longer term treatment in survivors should include low dose aspirin, warfarin, beta blockers, lipid lowering agents, life style modifications and other measures.

KEY WORDS:

1. Acute myocardial infarction in elderly
2. Ischemic heart disease
3. Ageing and heart
4. Atherosclerosis
5. Coronary artery disease

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Figure 4.3: Fatty-streak formation in atherosclerosis

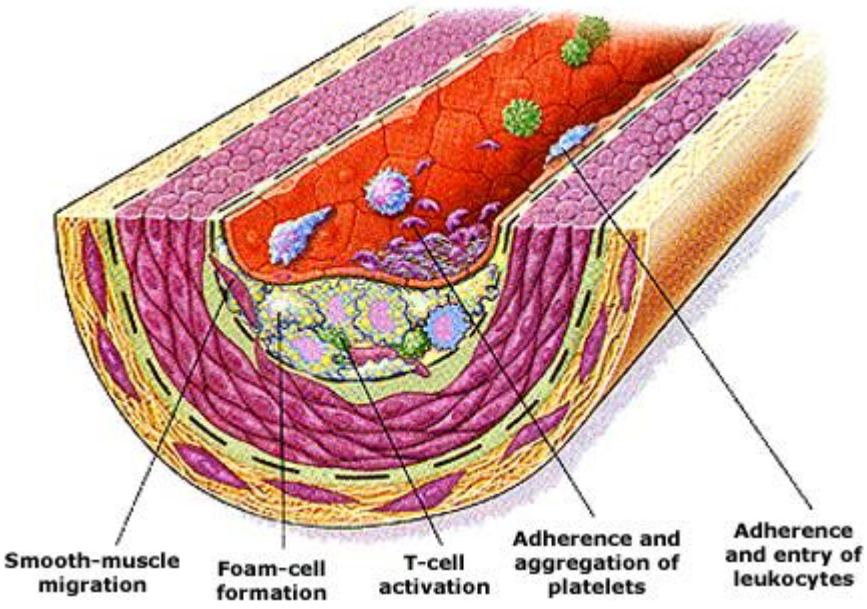


Figure 5.7: 70 years old male patient with acute myocardial infarction and heart failure

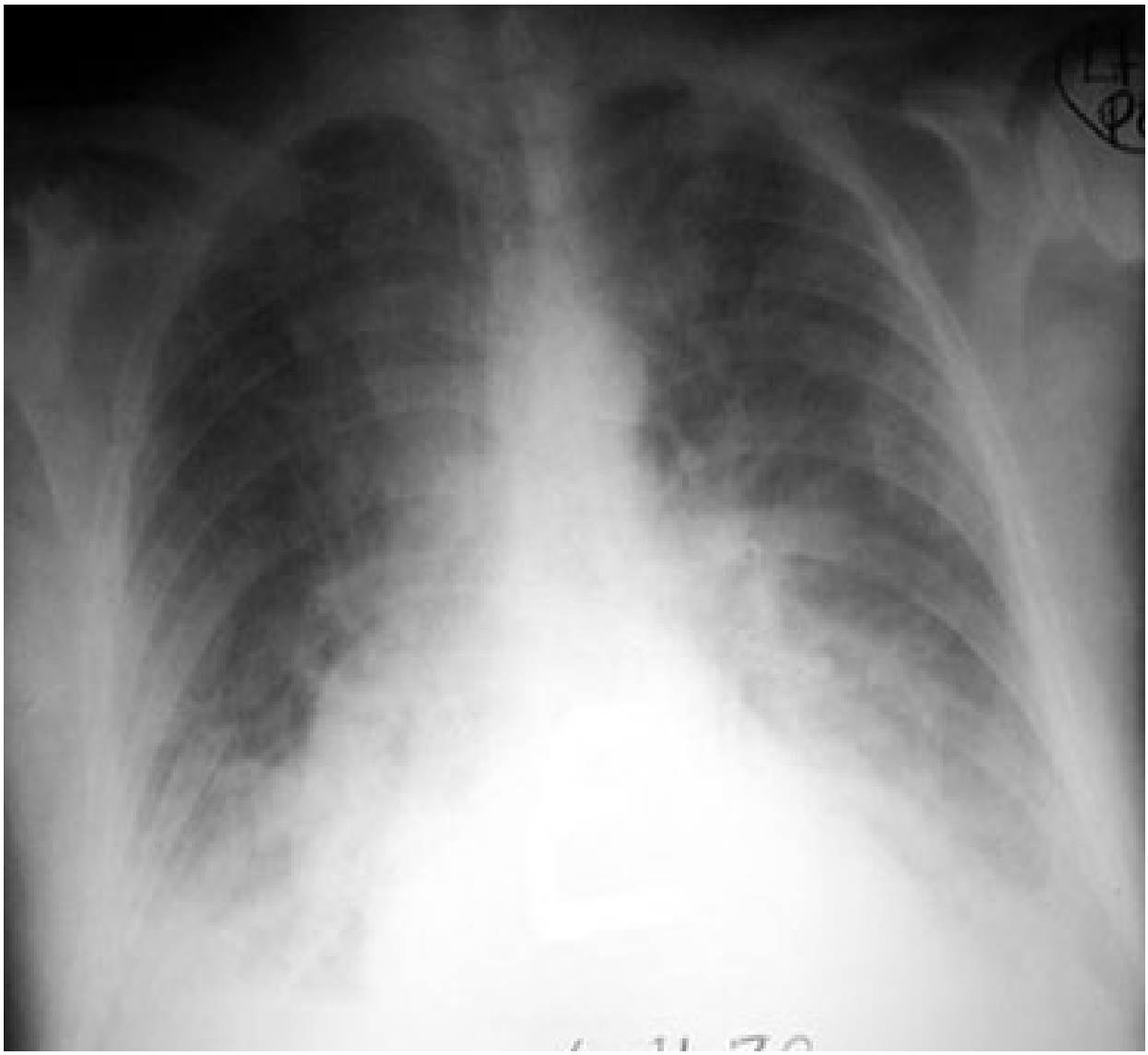
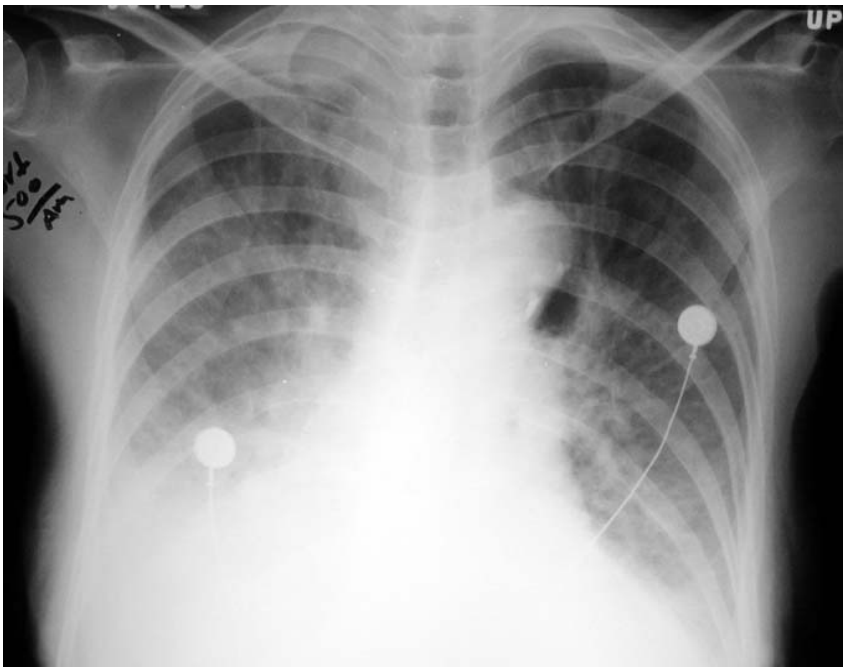


Figure 5.8: 73 years old female patient with acute myocardial infarction with pulmonary edema (before and after treatment)



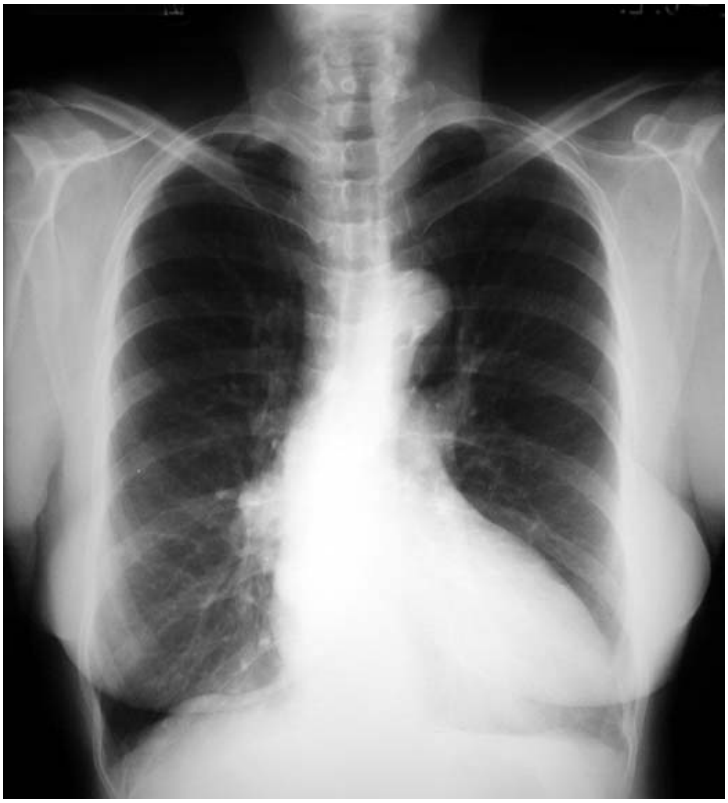


Figure 5.9: 58 years old female patient with acute anterior wall myocardial infarction

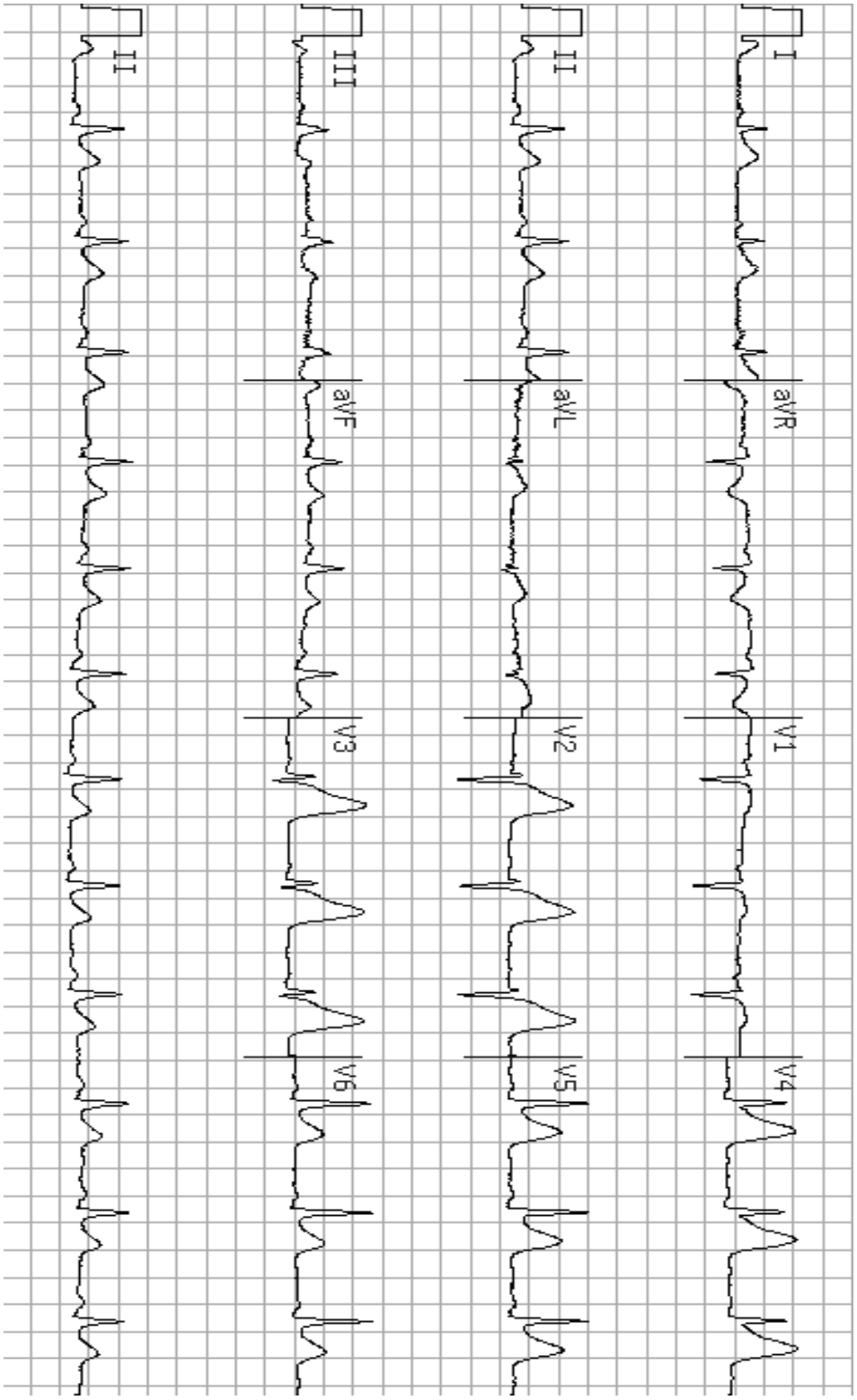


Figure 5.10: 66 years old female patient with antero-septal myocardial infarction and RBBB

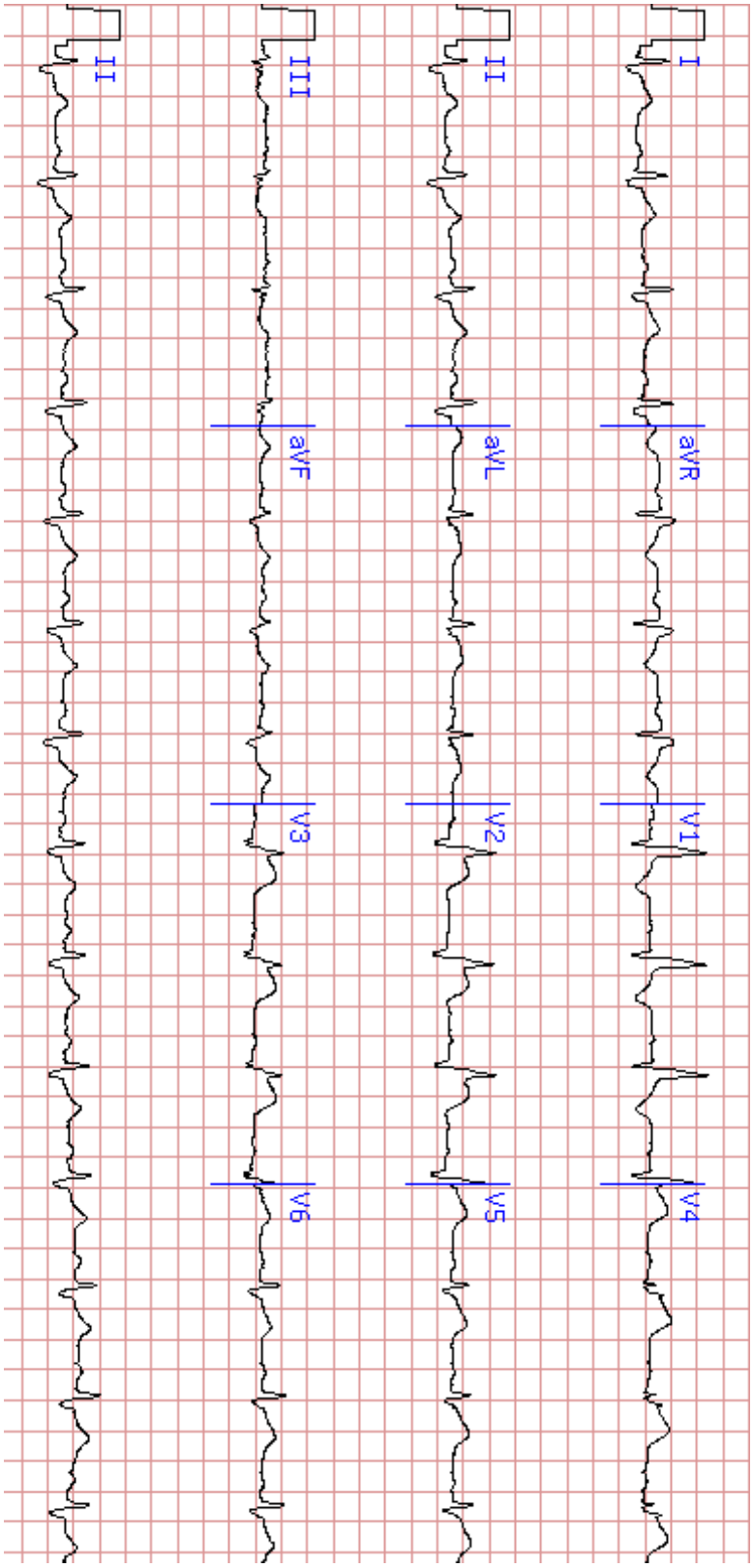


Figure 5.11: 74 years old female patient with acute infero-lateral and probably posterior myocardial infarction (MI) with second degree AV block (Type 1) with 2:1 block initially and then 3:2 AV Wenkebach's with underlying sinus rate of about 85/min. Left axis deviation is present.

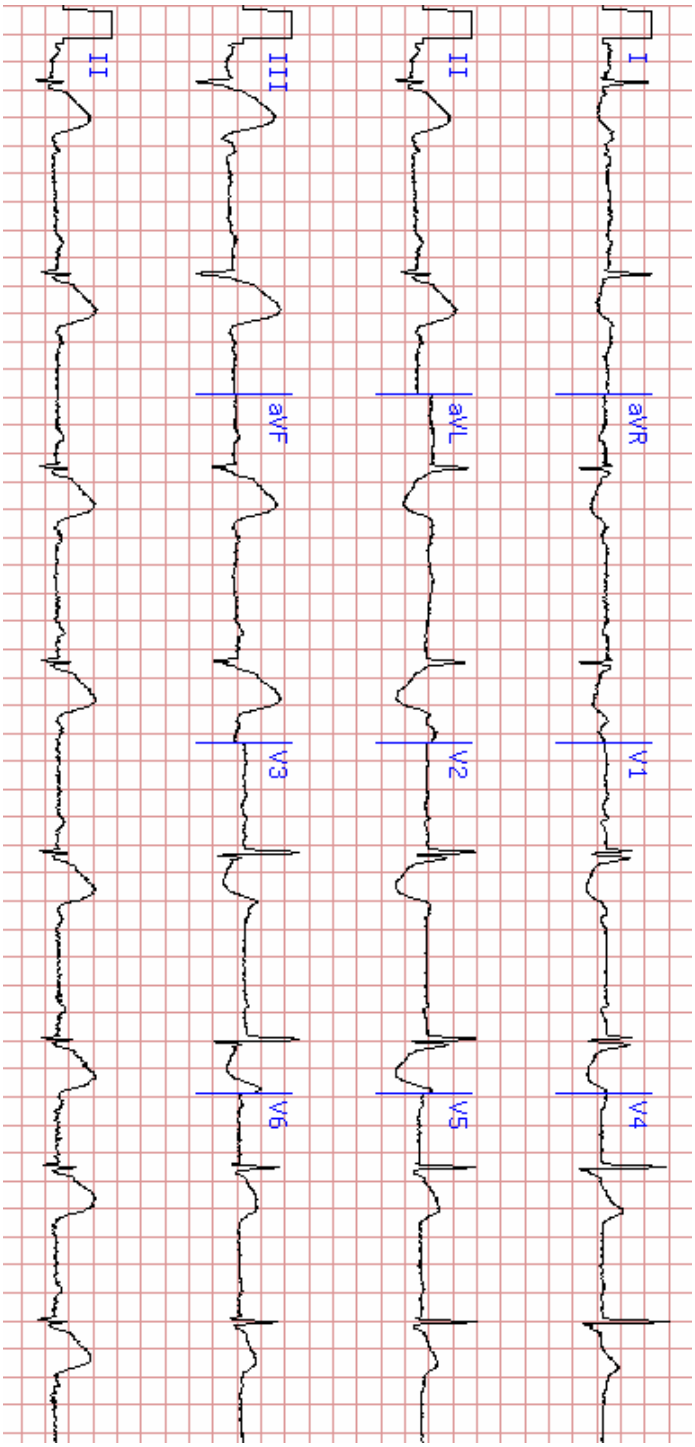


Figure 5.12: 69 years old male patient with large infero-(postero)-lateral myocardial infarction

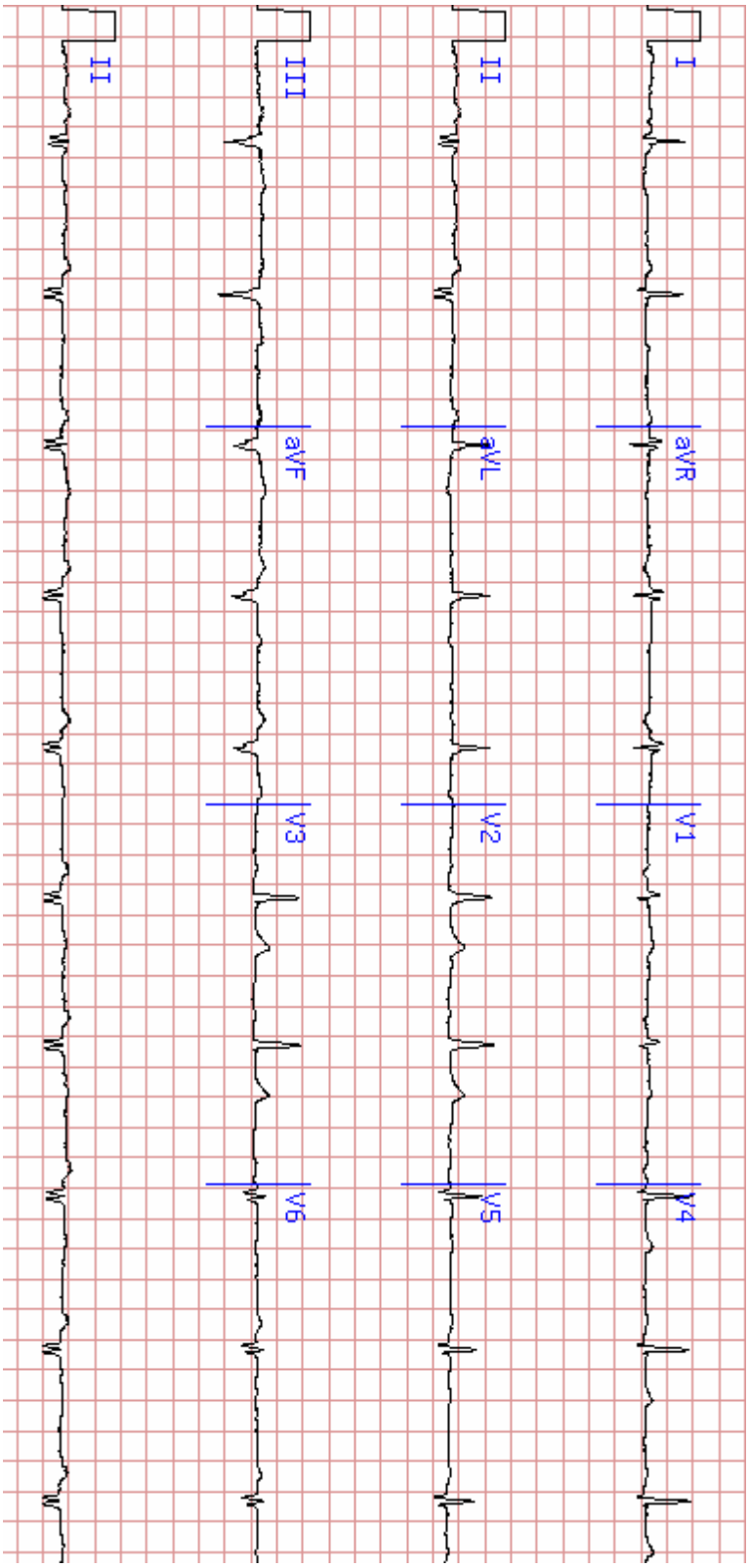
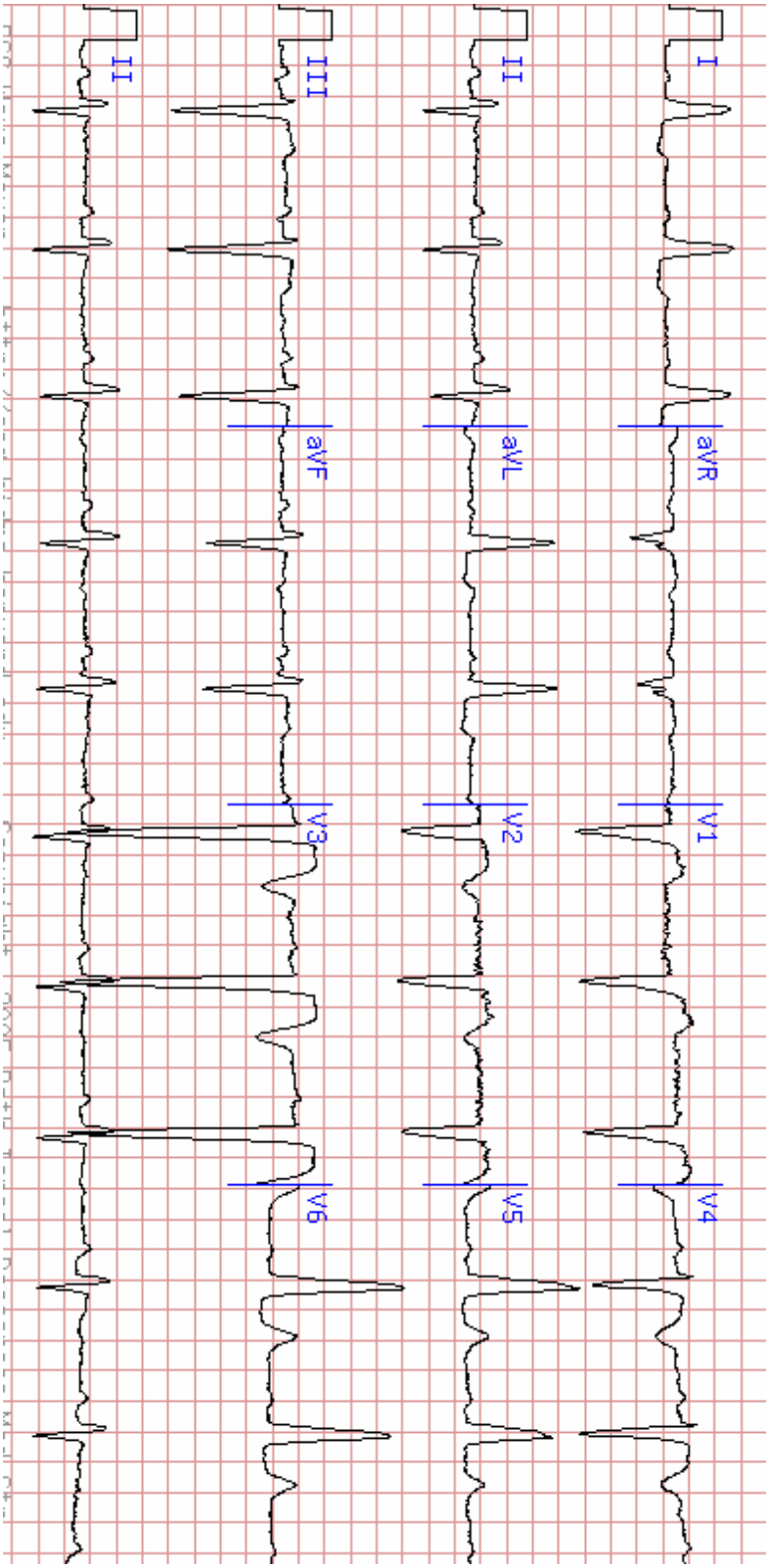


Figure 5.13: 68-year-old male with acute anterior wall myocardial infarction and left bundle branch block



ABBREVIATIONS USED IN MASTER CHART AND PROFORMA

AS	-	Altered sensorium
FND	-	Focal Neurological Deficit
Abd	-	Abdomen
HT	-	Systemic Hypertension
DM	-	Diabetes Mellitus
RBBB	-	Right Bundle Branch Block
LBBB	-	Left Bundle Branch Block
AVB 1	-	Atrio Ventricular Block Type-I
VT	-	Ventricular Tachycardia
VPC 1	-	Ventricular Premature Contraction – Lown's Grade 1
HF	-	Heart Failure
CA	-	Cardiogenic Arrest
CS	-	Cardiogenic Shock
PMIA	-	Post Myocardial Infarction Angina

PROFORMA

Name:	Age:	Sex:	Address:
IP No:	Occupation:	D.O.A:	
		D. O.D:	

Diagnosis:

Chief complaints:

Chest pain-typical, atypical, no chest pain

Sweating

Palpitation

Syncope

Dyspnoea

Nausea/or Vomiting

Giddiness

Altered sensorium

Abdominal pain

Focal neurological deficit

History of Present Illness:

Past history:

Diabetes

Hypertension

Dyslipidemia

Stoke (TIA or CVA)

IHD

Family history:

IHD/Obesity/Diabetes/Hypertension/Hyperlipidemia

Personal history:

Smoking / Alcohols / Other

GENERAL EXAMINATION:

Conscious

Oriented

Obesity (BMI)

Dyspnoea/Tachypnea

Cyanosis

Clubbing

Pitting pedal edema

Xanthelasmas

Tendon xanthomas

Arcus senilis

Anemia

Jaundice

Lymphadenopathy

Vital signs:

Pulse rate

Blood Pressure

Respiratory rate

Temperature

Examination of the cardiovascular system

JVP

Inspection

Palpation

Auscultation

Examination of other systems:

Respiratory system

Abdomen

CNS

Investigations:

1) Urine analysis

2) Complete hemogram
3) Blood sugar-fasting and post prandial
4) Bl.urea, sr.creatinine, sr.electrolytes & sr.lipid profile
5) CPK-MB level, SGOT level
6) Chest X-ray
7) ECG
8) Echo and Doppler

Complications:

Heart Failure **/** Cardiogenic shock / Cardiac arrest / Arrhythmias / Reinfarct /CVA / Bleeding / Death- <24hrs, 1-7days, 8-30days

Treatment given:

Thrombolytic therapy given or not – if not given, the reason

Beta blockers – given or not - if not given, the reason

Others

30 DAYS FOLLOW UP SHEET

NAME:

AGE: SEX: M/F I.P.NO:

DIAGNOSIS:

SYMPTOMS

SIGN

THERAPY

COMPLICARTIONS

DEATH