"GENDER DIFFERENCE IN OUT COME OF ACUTE MYOCARDIAL INFARCTION"

Submitted By

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MD

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GENERAL MEDICINE

Under the guidance of

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2012

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I hereby declare that this dissertation entitled **"GENDER DIFFERENCE IN OUT COME OF ACUTE MYOCARDIAL INFARCTION"** is a bonafide and genuine research work carried out by me under the guidance of **DR.S.S.DEVARMANI** **M.D.** Professor Dept of Medicine.

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LIST OF ABRREVATIONS USED

AMI	Acute myocardial infarction
CAD	Coronary artery disease
CHD	Coronary heart disease
CHB	Complete heart block
HDL	High density lipoprotein
IWMI	Inferior wall myocardial infarction
LAHB	Left anterior hemi block
LCA	Left coronary artery
LCX	Left circumflex artery
LVEF	Left ventricular ejection fraction
MR	Mitral regurgitation
NSMI	Non-ST-segment myocardial infarction
OCP’s	Oral contraceptives
PDA	Posterior descending artery
RBBB	Right bundle branch block
RCA	Right coronary artery
V.F.	Ventricular fibrillation
V.T.	Ventricular tachycardia
VPC’s	Ventricular premature complexes
UA	Unstable angina

ABSTRACT

Background: for decade, acute coronary syndrome was thought primarily a disease of men, in whom most research was conducted. But acute coronary syndrome afflicted a diverse patient population and major subset of those are women .women present with special diagnostic and therapeutic challenge to primary physician

MATERIALS AND METHODS

1. SOURCE OF DATA:

The material for the present study was collected from patients who were admitted in ICU of BLDEU'S Shri B.M. Patil Medical College Hospital and Research Centre, Bijapur who were diagnosed with acute Myocardial infarction according to standard WHO criteria and were admitted within 24 hours of onset of chest pain.

The patients were informed about study in all respects and informed written consent was obtained.

Period of study was from November 2009 to March 2011.

2. METHOD OF COLLECTION OF DATA:

A total of 179 patients were taken for the study, every case was included after detail history clinical examination and relevant investigations which included ECG and CPK-MB.

Patient were followed up for a period of 30 days from the date of admission and were contacted after discharge by means of telephone or post card & were called up for follow up on 30^{th} day.

Analysis of all patients were done regarding morbidity and mortality first without adjustment for any base line characteristics, then with adjustment for age alone and finally

with adjustment for age and other base line characteristics recorded at study entry were considered in the model.

Period of study was from November 2009 to March 2011.

3. INCLUSION CRITERIA:

Patients who were admitted in ICU of BLDEU'S Shri. B.M. Patil's Medical College, Bijapur with symptoms of suspected acute myocardial infarction were included in the study. Patients were selected on the basis of history and clinical examination.

4. EXCLUSION CRITERIA:

Patients presenting beyond 24 hours of the onset of symptoms of suspected acute Myocardial Infarction.

RESULTS: chest pain was the main complaint in majority of male (83.1%) and female (82%) patients. Syncope, palpitation, abdomen pain occurred in less number of patients. In male hypertension(28%) was the significant risk factor followed by, smoking (21%),hypercholesterolemia (20.3%), diabetes mellitus (16.9%). In females, post menopausal state (90%), hypertension (54%), hypercholesterolemia (50%).In hospital complication are more common in women (49%) compared to men (38%).there is a significant mortality in women (24.5%) compared to men (10.2%)

CONCLUSION: Even though clinical symptoms are same between men and women with acute coronary syndrome have different risk factors, higher complication and higher in-hospital mortality compared to men.

CONTENTS

INTRODUCTION

Acute coronary syndromes are the commonest and most important cause for premature death in both developed and developing countries. It is the cause for 25% of deaths in female and 30% of deaths in male, and cuts short the life expectancy ranging from 3.4 to 9.4 years [1] .

Recently, it has reached an endemic proportion. The Global Burden of Diseases (GBD) study reported the estimated mortality from coronary artery disease (CHD) in India as 1.6 million in the year 2000.Extrapolatation of these numbers estimates the burden of CHD in India to be more than 32 million patients. Hospital statistics reveal that 20-25% of all medical admissions are due to CHD [2].

Acute coronary syndromes indicate myocardial impairment due to many factors. However most common cause is atherosclerotic disease of coronary arteries impairing the delicate balance of "supply and demand."[3].

Experiments have shown that, when there is reduction of 75% of cross sectional area of lumen, full range of increase in flow to maintain myocardial demand is not possible, and when luminal area is reduced by 80%, blood flow at rest is reduced. Further, minor decrease in the orifice can lead to dramatic reduction of blood flow and produce myocardial ischemia. Hemorrhage, thrombosis, fissuring of atheromatous plaque can worsen the obstruction and lead to clinical manifestations [4.]

The pattern of CAD in India has been reported to be as follows:[5]

1. CAD appears a decade earlier compared to age incidence in developed countries. The peak period is between 51-60 years.

2. Males are affected more than females.

3. Hypertension and diabetes mellitus account for about 40% of all cases.

4. Heavy smoking is responsible etiologically in a good number of cases. Men and

women with acute coronary syndromes demonstrate clinically important differences in epidemiology, diagnosis, prognosis, treatment and prevention. Studies conducted to know the profile of acute coronary syndromes are mainly on the male population and general conclusions are drawn. The same may not be applicable to women. The coronary risk factors of Indian women differ slightly from their western counterpart. There are very few studies conducted on Indian women. This study attempts to highlight the clinical profile of acute coronary syndromes, with particular reference to the Indian women.

OBJECTIVE OF THE STUDY:

1. To study the morbidity and mortality of myocardial infarction patients in women and men.
2. To find out the difference between both the sex.

REVIEW OF LITERATURE

Bonetas (1700) made the earliest correlation of the clinical picture of coronary artery disease. William Herberden described angina pectoris and later published his paper in 1772. John Hunter described the clinical description and clinical features in 1773.

In 1799, Caleb Parry proposed that angina may be due to inefficient delivery of blood to heart muscles, particularly during exercise[6].

In 1845, Leipzig, Vogel discovered cholesterol as major constituent in atheromatous plaque[7].

It was James. B. Herrick in 1912, who described the symptomatology, diagnosis and treatment of obstruction of the coronary arteries. The experimental study of blood lipids and atherosclerosis started in 1913 by Anitschkow[8].

In 1920, Pardee first recognized the electrocardiographic curve of acute stage of myocardial infarction. William Enthoven invented ECG Ladue Wrobleuski and Karmen introduced estimation of the SGOT enzyme levels in 1954. The value of LDH estimation was introduced in 1955 and CPK by Dreyfuss in 1960.

On the therapeutic side, Kobnson used streptokinase for thrombolysis in 1959. In 1977, Mc-curtly Gruntzing introduced the technique of PTCA. Surgical reperfusion by CABG has been undertaken with variable success since early 1970's.

Numerous articles have been published about the risk factors in IHD[9]. In 1938, cigarette smokers were shown to die at an early age. It was estimated that the smoking

directly contributes to 3, 25,000 premature deaths in America[10]. In 1981, William.B.Kannel showed the influence of cigarette smoking was more pronounced in men than in women. Only 30% of cardio vascular disease can be contributed to smoking in women compared to 20% in men[11].

An increased risk of vascular diseases, such as venous thromboembolism, stroke, myocardial infarction, has been linked with the current and past[12] history of oral contraceptives and steroids have been shown to increase serum cholesterol and triglyceride concentrations[13,14].

Patricia Wahl reported increased incidence of stroke and myocardial infarction in women of childbearing age who take oral contraceptives[15].

In 1983, Barret-Connor reported diabetic men had almost twice the ischemic heart disease death rate as that of diabetic women[16]. In 1981, Robert.I.Hamely 16 reported that 20% female siblings developed coronary artery disease when both father and mother had IHD[17].

T.H.Dave et al in1991 studied various risk factors specific to Indian women in development of coronary artery disease[18]. Sanjay.P.Zondey in 1994 reported that socio-economic status and physical activity were significantly associated with ischemic heart disease[19].

DEFINITIONS

Acute coronary syndromes is composed of patients with acute myocardial infarction (MI) with ST segment elevation on their presenting electrocardiogram (STEMI) and those with unstable angina (UA) and non-ST-segment elevation MI (NSTEMI)[20]. STSEMI is due to the formation of occlusive thrombosis at the site of rupture of an atheromatous plaque in a coronary artery. UA is defined as angina

pectoris or equivalent ischemic discomfort with at least one of the three features-

1) It occurs at rest (or with minimal exertion); usually lasting > 10 min.

2) It is severe and of new onset (i.e., within prior 4 to 6 weeks), and/or

3) It occurs with a crescendo pattern (i.e., distinctly more severe, prolonged, or more frequent than previously). The diagnosis of NSTEMI is established if a patient with clinical features of UA develops evidence of myocardial necrosis, as reflected in elevated cardiac enzymes. UA/NSTEMI is usually associated with severe coronary obstruction but not total occlusion of the culprit artery. Among patients with UA/NSTEMI, between 40 to 60% have evidence of myocardial necrosis with elevated enzymes[21].

EPIDEMIOLOGY

Coronary artery disease is a world wide disease Cardio vascular diseases rank number one in the United States in causing morbidity and mortality. Although CAD is considered a disease of industrialized western world, now it has brought this problem to the door step of the third world countries[22].

Epidemics of CHD began at different times in different countries. In developed countries, where epidemic began earlier (1920's), started declining now[23]. The decline in CHD in various countries is due to changes in the life style and related risk factors – diet, diet related serum cholesterol, cigarette use, exercise habits and better control of hypertension[24] .

CORONARY CIRCULATION

Anatomy

There are 2 main coronary arteries, the Left main coronary artery (LCA) and Right coronary artery (RCA).The right and left coronary arteries encircle the

epicardium like the crown encircles the head, hence the name “coronaries”. They arise from the aortic bulb, which is made up of three aortic sinuses. Anatomically the three sinuses are disposed such that, one is anterior and two are posterior. The RCA arises from the anterior sinus and LCA from posterior sinus, the remaining right posterior sinus being non- coronary one. The RCA is dominant system in about 85% of individuals. It means in about 85% individuals, it supplies the posterior diaphragmatic portion of the interventricular septum and the diaphragmatic surface of the left ventricle

RIGHT CORONARY ARTERY

The RCA originates from the right aortic sinus at a point lower than the origin of LCA. It passes down the right atrioventricular groove towards crux. The first branch of RCA is considered the conus artery. In 50% of the hearts, this vessel arises from the right coronary ostium. In the other 50% of individuals, it arises from a separate ostium in the right aortic sinus. It serves as collateral in patients with LAD obstruction.

The second branch of RCA is the Sino-atrial node artery. It has been found that this artery originates from RCA in 59%, from left circumflex artery (LCX) in 38% and dual supply in the remaining 3%. When it originates from RCA, it passes obliquely backward through the upper portion of the atrial septum and the antero-medial wall of right atrium. It sends branches to the sinus node, also to the right atrium or both atria. When it arises from LCX, it passes backwards in atrial septum to reach the sinus node area.

The mid portion of the RCA usually gives rise to one or several medium sized acute marginal or right ventricular branches. These branches supply the

anterior wall of the right ventricle and serve as a source of collateral circulation in patients with LAD obstruction.

The important branch of RCA is the posterior descending artery (PDA) when RCA is the dominant one. The PDA usually originates at or shortly before the crux, and passes forward in the posterior interventricular groove. During its course along the groove, it gives rise to a number of small "Inferior Septal Branches", which pass upward to supply the lower portion of the interventricular septum. About 15% do not have RCA dominance; about half of these have LCA dominance, in the remaining half, they are co-dominant. When LCA is dominant, it is large and continues down the diaphragmatic surface of the left ventricle, where it gives rise to "Posterior Left Ventricular Branches" and then reaches the crux and turns forward to become PDA. In these cases, the RCA is very small and terminates before reaching the crux. At or near the crux, the dominant RCA in 90% of subjects gives rise to a small "Atrioventricular Node Artery", which supplies the node.

Branches:

Conus artery is the first branch, which ramifies on lowest part ofpulmonary conus and upper part of right ventricle and anastomoses with a similar branch of left coronary artery. Some consider that the conus artery is of significance in coronary artery disease. Right Anterior Ventricular rami, usually 2 or 3 ramify towards the apex. Right posterior ventricular rami, commonly two, arise from second segment of right coronary artery and supply the diaphragmatic aspect of right ventricle.

As right coronary artery approaches crux of heart, it produces 1-3 posterior inter ventricular rami which give few branches to right ventricle and to the left ventricle. Atrial rami of right Coronary Artery: Atrial rami of right coronary artery are

divided in to three groups

1. Anterior group

2. Lateral (Right or Marginal) group

3. Posterior groups.

Anterior and lateral branches supply mainly right atrium. Posteriors supply mainly to right and left atrium. The artery of the S.A.Node is an atrial branch, distributes largely to myocardium of both atria, mainly right. It passes back between aorta and right auricular appendage and branches around base of Superior Vena Cava. 'Ramus Cristae terminalis' is a large branch which traverses S.A. node and supplies

the atria. Septal branches: Septal braches of RCA are small, numerous and supply posterior inter ventricular septum, but do not reach apical septal part which is supplied by terminal septal branches of anterior inter ventricular artery. A large posterior septal branch of right coronary artery supplies A.V.Node.

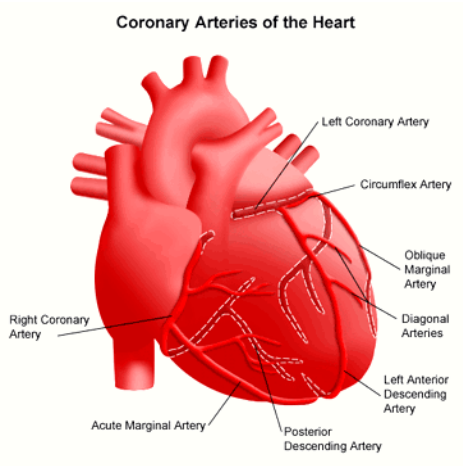


FIG. 1 LEFT MAIN CORONARY ARTERY

Left coronary artery supplies greater volume of myocardium almost all left ventricle and left atrium, most of inter ventricular septum. The main LCA arises from

the left aortic sinus. The initial segment of left coronary artery varies from few millimeters to few centimeters and lies between pulmonary trunk and left auricular appendage. After reaching atrioventricular sulcus, left coronary artery divides into two or three main rami and continues as anterior descending artery.

The LAD passes down the anterior interventricular groove towards cardiac apex. Its main branches are the septal and diagonal branches. The septal branches pass downwards into the inter-ventricular septum and interconnect with similar septal branches of PDA, to produce a network of collateral channels. The diagonal branch of the LAD passes over the anterolateral aspects of the heart and supplies the apex. In

37% of patients, LCA have a trifurcation instead of bifurcation, in these cases "Ramus Medianus" arises between LAD and LCX, supplying free wall along lateral aspect of left ventricle. In 22% of patients, LAD is larger and longer and supplies apex.

Branches:

It has two major branches namely left anterior descending artery (LAD) and left circumflex artery (LCX). Anterior descending artery produces right and left anterior ventricular rami and also anterior and posterior septal rami. Right anterior ventricular rami are one or two in number and small. Left anterior ventricular rami are two to nine in number, branch at angle from anterior descending artery, cross anterior aspect of left ventricle, larger terminal reaching the left border. One branch (diagonal artery) is often large (in 50% of cases) and it may arise separately from left coronary trunk. A small left conus artery may arise from anterior descending artery and anastomose with that of Right coronary artery. Anterior Septal Rami: It passes back and down in septum to which they supply about ventral 2/3 rd. Posterior Septal Rami: They are small and supply posterior third of septa.

Circumflex Artery (LCX)

LCX passes down the left ventricular groove, gives off obtuse marginal branches supplying free wall of left ventricle along its lateral aspect. Anterior and posterior rami of circumflex are small and supply left ventricle. Anterior ventricular branches – two to three in number, run parallel to diagonal artery. Posterior ventricular branches – are few and supply part of left ventricle. In addition, it gives one or two left atrial circumflex branches supplying left atrium.

REGULATION OF CORONARY BLOOD FLOW

Large coronary arteries that are capable of constriction and relaxation, serves as conduit. They are referred to as 'Conductance Vessels'. Small intra-myocardial arteries which show striking changes in tone are called 'Resistance Vessels'. Abnormal constriction or failure of dilation of these vessels results in myocardial ischemia.

Coronary circulation is controlled by requirement of oxygen by heart. Myocardium can extract high percentage or fixed percentage of oxygen from blood, but it is the coronary bed that alters the resistance and blood flow accordingly. Normally intra-myocardial resistance vessels have tremendous capacity to dilate. The coronary resistance vessels adopt themselves to physiological alteration in blood pressure to maintain blood flow level, appropriate to myocardial needs. This is 'Auto Regulation,' e.g. exercise, emotional stress (which can affect resistance and regulation of blood flow).

The average coronary blood flow is 60-90 ml per min/100 gr. of normal left ventricle at rest. Myocardial oxygen consumption (MVO2) is the main determinant of coronary flow. MVO2 has positive linear relation with coronary flow. Apart from MVO2, there are other factors which contribute to determinants of coronary flow:

- Coronary perfusion pressure,

- Systolic compression,
- Endothelial dependent relaxing factor EDRF,
- Adrenergic tone to coronary arteries (exogenous vaso-constriction)
- Exogenous vasodilators.

CORONARY ARTERY DISEASE

WOMEN VERSUS MEN

Incidence of coronary artery disease

CAD in the entire lifetime is more common as a cause of mortality in women than men. Not only common, CAD in women is a problem because of difficulty in diagnosis, increased morbidity associated with CAD events and treatment. In general, men have less favorable risk factors.

Analysis have indicated worse prognosis and increased death in women. The excess CAD death is attributed to the greater age of women, which is an extremely powerful risk factor in its own right. The delay in onset in women compared to men is presumably due to pre-menopausal exposure to endogenous ovarian estrogen. In females CAD is increased around the time of menopause when the estrogen in plasma begins to decline. It is observed in studies that atheromatous fatty streak and atheromatous plaque exist both in men and in women. The extent of fatty streak in both men and women is a function of plasma level of LDL cholesterol and in women, VLDL cholesterol, which is closely related to the triglyceride level. The lesions in female are more lipids filled, rich in macrophage and less densely fibrous.

Thus, lesion could be more unstable under certain circumstances. Likewise, coronary calcification is half as that of men on ultra fast computed tomography until age 60, when difference between age narrows.

A difference in female acute MI susceptibility by race is also suggested. Black women are more susceptible for acute MI. This prediction is borne out by national mortality data where black women have consistently had a 50% - 75% greater risk for death from diseases of heart. The reasons for this greater risk are unclear but could relate 30to the particular mix of CAD risk factors.

The social difference is greater in women compared to men; women in social class V have nearly four times the standardized mortality ratio of women in class I.

Differences in CAD Presentations, Management and Outcome in Women vs. Men:

The tendency to overlook CAD in women is because of its lesser frequency in young and middle age and compounded by less classical presentations and more frequently it presents an angina in women and MI in men. In the Framingham study, the annual incidence of angina pectoris in women exceeds that of MI by ratio more than 2:1 in age 45 to 54 and 55 to 64, whereas in male MI incidence exceeds angina in all ages(31,32) . This gives the clinical impression that angina pectoris is an earlier and most common presentation in women.

TABLE-1 Annual incidence of angina, MI, and sudden death in men and women Per 10,000 population. Kannel WB.Patterns Am Heart J.1996; 111:383-390

	ANGINA		**MI**		**SUDDEN DEATH**	
Age (yrs)	**M**	**F**	**M**	**F**	**M**	**F**
45-54	30	21	54	9	11	3
55-64	74	54	91	25	21	6

65-74	52	51	119	51	31	13
75-84	25	94	168	90	43	32

Recognition of chest pain of cardio vascular etiology in women is difficult to diagnose because,

It is not expected.

Atypical, non-cardiac chest pains are more common in women compared to men.

True angina may not be typical.

True angina may not br typical

Because of atypical symptoms misdiagnosis is common. The reason for lack of classic anginal symptom in spite of having validated myocardial ischemia is unknown. The greater incidence of silent MI in women[27] may be related to atypicality of chest pain presentation.

Exercise tolerance test is to clarify atypical chest pain is not as useful in females as in males because of the susceptibility for false positive results[27]. Decision to perform exercise test is decided by the number of risk factors the patient has i.e. more the risk factors, the more likely the test will be a true positive.

There is possibility for a female patient having chest pain and positive exercise test and negative angiogram with normal lumen to have vaso-spastic disorder.

Clinical Presentation

Studies have clearly indicated that MI is more likely to be unrecognized among women. Nearly half of MI occurring in women is silent. Symptomatic women usually present late to the hospital after the onset of symptoms compared to men and all reports have analyzed that, admitted women exhibit adverse base line characteristics[33], they are older and have more adverse risk factors than men. Women show increased evidence of left ventricular failure.

Infarct size:

Occurrence of non-Q wave MI, otherwise termed subendocardial infarction, is reported to be higher in women[34].Infarct size is smaller but infarct expansion has been observed more frequently in women.

Cardiac rupture:

Relative frequency of cardiac rupture in patients over 70 years of age is higher in women than men with acute MI[35,36]. This is explained by lack of protective effect from collateral circulation due to the lesser extent of coronary artery disease and its presentation at a later age. Increased shear stress also plays a role in this.

Post MI mortality:

Frank MI in women has more unfavorable consequences than in men. Sudden deaths are more frequent. Early post MI mortality is greater in women compared to men

9% in female

in 6 Weeks mortality

4% in male

Excess mortality observed in studies, is concentrated within four weeks of post MI.

32% in female

after 1 Year

16% in male

Thus, post event mortality is about 50% greater in women than in men. This excess mortality is attributed to age and various risk predictors, either metabolic or those relating to severity of MI including congestive heart failure.

Thrombolysis:

Regarding outcome following thrombolysis, studies have revealed a number of features, which are detrimental to women like excess stroke, re-infarction, congestive cardiac failure, serious cerebral bleeding and shock. So residual morbidity is more in women treated with thrombolysis compared to men. The reason for the greater susceptibility of women to re-infraction and stroke and its remediation remains to be determined.

Operative procedure (PTCA&CABG):

PTCA and CABG show greater mortality in women than in men[37,38].

TABLE-2 Comparison of operative procedures like PTCA and CABG

MORTALITY	**MALE**	**FEMALE**
After successful PTCA	2.7%	4.2%
After CABG	2.6%	4.6%

This is accounted with later stage of diseases at the time of operation and greater inherent severity.

TABLE-3 Difference of CAD presentation and outcome in women vs. men[27]

PRESENTATIONS	W- WOMEN M- MALE	CONSEQUENCES	COMPARISON
Angina	W > M		
Atypical chest pain	W > M	MI morbidity	W > M
Silent MI	W > M	MI mortality	W = M
Death from MI	W > M		
Sudden death	W > M	Angioplasty mortality	W > M
Exercise test – false positive	W > M	CABG	W ≥ M
Angina leading to MI	W < M		

The important message from greater morbidity and mortality associated with CAD in women is that, very high priority should be given to early recognition of treatable risk factors. Early ischemic symptoms of atypical chest pain, fatigue should be aggressively worked up for potential coronary ischemia with exercise, particularly when risk factors of CAD are present in combination.

RISK FACTORS

Risk factor reduction is the primary clinical approach to prevent coronary artery disease (CAD) morbidity and mortality. The concept of risk factor identification and modification is based on the fact that exposure to certain host and environmental factors increases the statistical risk for developing a disease and that alteration of these conditions reduces the risk. Thus identifying risk factors may possibly retard the formation and growth of an atherosclerotic plaque. The Framingham study[39] was first of its kind to describe the primary and secondary risk factors like hypertension,

diabetes mellitus, hypercholesterolemia, cigarette smoking, obesity, race, family history of coronary artery disease, physical inactivity, personality type etc. Using Framingham data[39], Anderson et. al. concluded that the relative effects of serum cholesterol, hypertension and smoking were equal in both sexes. Women have the same risk factors like men. Although age, menopause, diabetes mellitus, low HDL cholesterol, and the use of oral contraceptives, may play an additional role in the development of coronary artery disease.

The Risk factors for CAD are divided into-

1. Modifiable risk factors

A) Major

1) Cigarette smoking.

2) Hypertension.

3) Diabetes mellitus.

4) Hyperlipidemia.

5) Obesity.

B) Minor risk factors

A) Oral contraceptives.

B) Physical activity.

C) Personality.

D) Socioeconomic status (SES) and psychosocial factors

E) Others–Alcohol, Hyperuricemia, Coffee consumption, Deficiency of trace

elements, Low circulating levels of antioxidants etc.

2. Non modifiable :(Other than gender)

1. Age
2. Race
3. Family history

1. **MAJOR MODIFIABLE RISK FACTORS:**

1). CIGARETTE SMOKING: Smoking is a well-documented risk factor for coronary artery diseases. Women, who smoke, are 3.6 times more likely to have a myocardial infarction than non-smokers. The incidence of sudden death is higher in smokers than in non-smokers. Cessation of smoking in high-risk individuals is followed by reduction in risk of coronary artery disease[39]. It may take 2-3 years for the risk to equal non-smokers, after cessation of smoking[40].

In the Framingham study, cardiovascular mortality increased by 18% in men and 31% in women for each 10 cigarette smoked per day[41]. Smoking has a synergistic effect on coronary artery disease mortality in individuals with other risk factors[40]. Passive smoking may also increase risk for CAD. Subjects who live with current or former smokers were prone to CAD by 9.6% in men and 6.1% in women**.**

Smoking predisposes to atherosclerosis by a variety of mechanisms:-

1. Smoking decreases HDL cholesterol. HDL was reduced by 12% in males and 7% in female smokers[40, 42].
2. Smoking has effect on coronary flow. It increases the risk of vasospasm[43].
3. Circulating carbon monoxide may damage the vascular endothelium[44].
4. Smoking leads to catecholamine release, which enhances platelet

aggregation[45, 46].

5. Smoking impairs oxygen transport and utilization by increasing the heart rate,

after load and contractibility by direct effects on myocardium, stimulation of autonomic ganglia and aortic and carotid body chemo receptors. This leads release of systemic hormones like catecholamine[47,48], growth hormone and cortisone. Thus, with increased demands, smoking decreases the exercise tolerance to angina and interferes with beta blockade[48].

6. Other chemicals in tobacco such as cyanides and oxides of nitrates may be harmful[44]**.**

2) HYPERTENSION

Numbers of studies have conclusively proved the role of hypertension in the development of coronary artery diseases. The risk of development of CAD is continuous and graded. According to data from Framingham study[39], hypertension is associated with two fold increases in risk for CAD. The same study also interpreted that the proportion of MI is not only twice that of normotensives but the incidence of infarction that go unrecognized significantly increases with the severity of hypertension and is twice as that of normotensives[49].

Kannel and Stammler, opined that hypertension, whether labile or fixed, systolic or diastolic, in males or females is atherogenic and thus predisposes to CAD[39, 50]. 50However, a systolic blood pressure is more potent risk factor for CAD[51].

The Medical Research Council Trial in Britain, which involved more than 85,000 patients, was the first to evaluate separately thiazide and beta-blockers against placebo

in persons with mild hypertension. The results suggest that treatment with either group, reduced cardio vascular mortality[52].

3) DIABETES MELLITUS

Women are particularly vulnerable to the cardiovascular squeal of diabetes[53]. Among patients evaluated for symptomatic coronary artery disease women are more likely than men to have diabetes mellitus.

Atherosclerosis occurs earlier in diabetics[54].Thus, diabetic women tend to have a 54excess of the major risk factors, predisposing them to CAD. These are often present even before diabetes mellitus manifests. All these atherogenic traits are further aggravated when diabetes is overtly manifested[49].

Diabetes increases the risk by 3 fold in women and puts them at the same risk as men of same age. Women with diabetes do not share the relative gender mediated pre-menopausal protection against CAD[54].

In Framingham study, diabetic women had three fold higher risks for recurrent MI and two fold higher risk for fatal re-infarction than non-diabetic women[33]. TIMI data reveals, that diabetic women treated with thrombolysis had twice in hospital mortality compared to diabetic men and 4 times the mortality of non-diabetic men. Diabetes increases the risk of heart failure by 8 fold in women as compared to 4 fold in men. Diabetes eliminates the protective effects of estrogens and removes the normal sex difference in the prevalence of CAD[55].

The mechanism how diabetes predisposes to CAD is uncertain. The following mechanisms may contribute:-

1. Diabetes frequently co-exists with other CAD risk factors like hypertension, obesity

and dyslipidemia[49].

2. Typical dyslipidemia in diabetes is increased triglyceride and decreased HDL. Resistance to the action of circulating insulin may play a role in dyslipidemia. Lipoproteins may be altered in glycation.

3. Atherosclerosis in diabetics may be complicated by procoagulant state caused by increased platelet adhesiveness and increased PAI-1[56].

4. Insulin may contribute to atherogenesis by promoting smooth muscle proliferation.Incidence of silent MI in diabetics was 42%, in one series by Bradley. The absence of pain may be due to autonomic neuropathy. Microangiopathy is an inevitable component of diabetes, which may involve the coronary arteries.

4) BLOOD LIPIDS

Total cholesterol (TC), High-density lipoprotein (HDL) and triglyceride levels predict CAD independently in men and women. However, the inverse relationship between HDL and CAD risk is stronger in women. Women tend to have lower serum cholesterol levels until 50 years and then they start to exceed men's level.

According to Framingham study[49], the ratio of TC to HDL cholesterol rises steadily with age from 3.4 at age 25-34 years to 4.7 at the age of 75-89 years. The net result is an increase in TC to HDL ratio as the age advances. However, virtually at all ages HDL values in women are 10 mg/dl higher than those in men. These changes may explain the rarity of CAD in young women and the gradual equalization of risk with advancing age. All atherogenic lipids rise with age till about 60 years and then decline[49, 53].

5) GENERALIZED OBESITY:

Obesity is associated with increased risk of hypertension, diabetes, dyslipidemia and CAD. Body Mass Index (BMI), which is defined as the weight in kilograms divided by height in squared meters (kg/m) is now accepted as the single best measure of obesity[57]. In the 16-year data from the Nurses' Health Study (NHS), CAD mortality was 4-fold lower in lean (BMI <21) than in obese women (58). For Asians, the optimum BMI is <23, whereas >23 is considered overweight and >25 obese[59]. Thus the BMI cut-off points for overweight are two units and obesity 5 units lower in Asians than in Whites.

MINOR RISK FACTORS

A) ORAL CONTRACEPTIVES (OCP's)

Oral contraceptive predisposes women to CAD at a much younger age. Patients on OCP's have been found to accelerate blood clotting and decreased blood concentration of some clotting factors[40].

Mann et. al was the first one to demonstrate the increased risk of MI with OCP's. The relative risk of MI was 4-5 times more when compared to non-user. Smoking along with OCP has increased the risk of CAD especially after 35 years. It thus has a synergistic effect leading to early menopause[40, 60],

Use of OCP's and smoking in pre-menopausal women, increases the incidence of MI 7-39 times more than non-smoking and non-pill users[60].

B) Physical activity

The Framingham study shows sedentary people had about 3 times more risk of developing CAD than a physically active person. Postulated mechanisms for favorable effects of physical activity include:

i. Exercise induced increase in HDL and Lipoprotein lipase levels.

ii. Increase in myocardial oxygen demand for any sub maximal task.

iii. Decrease in platelet adhesiveness and increased fibrinolytic activity.

iv. Improves myocardial perfusion.

Thus, exercise can be recommended as a strong adjuvant to dietary modification in prevention and treatment of obesity[40, 49].

C) Personality

Type A personality is associated with higher risk of CAD in women[61]. Depression is associated an increased risk of fatal CAD in women.

D) Socioeconomic status (SES) and psychosocial factors

CAD has now become a disease of the poor in rich countries and of the rich in poor countries. Women with less than a high school education have a 30%-50% higher CAD mortality than those with higher education. Depression, high hostility, low social support and low education level are associated with CAD, after controlling for adverse health behaviors. Indians with low literacy have a higher prevalence of CAD and risk factors such as smoking and hypertension. However, differences in SES failed to explain the excess burden of CAD among Indians in the UK. Despite having a lower level of TC, Indians had a three to four fold higher odds ratio for a high-risk lipid profile, after controlling for SES, age and sex[62]. The impact of psychosocial and behavioral factors on CAD in Indian women requires further investigation.

E) Others

1. Alcohol

In low to moderate quantity, it is protective due to increase in HDL and inhibition of platelet aggregation. In large doses, it is harmfull[63].

2. Diet

Certain dietary practices like high consumption of saturated fats, sucrose, animal fats and less of vegetable are associated with increased risk of MI. A high fiber diet is recommended.

2) Non- modifiable risk factors

Age:

Compared with the age group 34-44, CAD mortality among women increases 40-fold by the age of 80, when its incidence becomes identical in men and women. Women are about 10 years older than men at first manifestation of CAD, although they have a similar plaque burden[64]. Women lose this 10-year advantage if they[64]smoke, have diabetes, or had a premature menopause. The postmenopausal increase in the risk of CAD is related to a higher incidence of hypertension, diabetes, dyslipidemia and obesity. The steady increase in CAD mortality with age is in sharp contrast to that of breast cancer, which peaks between the ages of 40 and 50 years and declines steadily thereafter.

Asian Indian ethnicity:

The risk of CAD among Indians is double that of Americans and several-fold higher than other Asians. Indian ethnicity has now been demonstrated to be a risk factor by itself[64].

Family history:

Studies suggest that the familial aggregation of coronary artery disease may be influenced by genetic characteristics of risk factors and by common environmental factors (such as diet, smoking, exercise habits) encountered by family members[65].

The genetics of CAD appears to be a complex interaction of – genetics, molecular and cell biology and environmental issues. There are 4 main sources for evidence linking CAD with genetic issues.

Concentration of CAD in families.

Evidence in twins. Basic science of genetics.

Phenotypes linked by inherited patterns.

At present, it is estimated that about 40% of the risk of developing CAD is controlled by genetic factors and 60% by environmental factors.

Among women, a history of MI or sudden death before the age of 55 years in a sister is more strongly associated with risk of MI than that in a brother or parent. A

family history of premature CAD in a sister is associated with a 12-fold higher risk versus six fold for a brother and three fold for a parent[67]. Women with a family. history of premature CAD, especially in a sister, should follow a course of action similar to the one recommended for those who had survived an MI or had coronary revascularization at a young age.

MENOPAUSE

CAD is now a major cause of morbidity for both men and women. In women, death occurs at a later age than men, with CAD developing a decade later. This discrepancy in CAD between the sexes has been attributed to hormones and menopause.

CARDIOVASCULAR CONSEQUENCES OF MENOPAUSE[68, 69]

1) Haemodynamic changes- A gradual increase in peripheral vascular resistance is seen after menopause due to lack of estrogen effect on the receptors in the muscular layers of arterial wall. There is also a decrease in contractility, stroke volume and LV function.

2) There is an increase in Endothelin-1 after menopause, which is a powerful vasoconstrictor.

3) Estrogen has calcium antagonistic properties. After menopause, there is an increased calcium influx, leading to increased peripheral vascular resistance.

4) Haemostatic factors like Fibrinogen, PAI-1 and factor VII occurs are increased, thereby predisposing to atherogenesis.

5) Changes in lipid and carbohydrate metabolism – there is an increase in TC, LDL and a decrease in HDL levels.

6) There is an increase in insulin resistance, thereby leading to smooth muscle hyperplasia. This is due to hyperinsulinemia.

Framingham study demonstrated menopause doubled the risk of CAD. There was prompt loss of resistance to CAD in post-menopausal women compared to those of the same age who remained pre-menopausal. The type of menopause whether natural or surgical conferred the same risk[49].

Hormone replacement therapy (HRT) has gained importance as a primary prevention of CAD. (Various studies show that there is 15-25% reduction in mortality with HRT and morbidity from cardiovascular disease is reduced by 50-85%. Sullivan et.al. Showed mortality with HRT was 4% as compared to non-users where it was 35%[72].

CLINICAL FEATURES

It has been long assumed that the clinical expression of CAD is similar in men and women. The Framingham study suggests that gender differences do exist in clinical presentation. The most common clinical presentation of CAD in women was angina, where as MI was more common in men. MI and sudden death occurred rarely in women less than 75 years of age [70-76].

After 75 years, CAD occurred equally in men and women. Epicardial coronary artery disease occurred in more than 90% of men presenting with typical angina, compared to only 60-70% in women[77]**.** Women have greater prevalence of vasospastic angina and micro vascular angina or both. The prevalence of angiographic coronary disease in women varies drastically with age, nature of pain and presence of coronary risk factors[78].

Women more often than men report initial warning symptoms, including nausea, fatigue, dizziness, and shortness of breath[79]. Milner et al980 found that women reported a significantly greater number of symptoms compared with men. Further, women are usually 7 to 10 years older and more likely to have diabetes mellitus[81, 82]. Than men at the time they first experience an MI, suggesting that the symptoms may result from this illness rather than sex[81].

Vaccario et. al. found clinical abnormalities at presentation in acute coronary syndromes like higher Killip class, higher pulse rate and low systolic blood pressure in younger women compared to men. But there were no differences in elderly patients.

EXAMINATION

General examination:

Patient appears anxious and restless. Cold perspiration and pallor may be evident. Patient in cardiogenic shock usually have cold clammy extremities.

Pulse may be normal. Patients with anterior wall infarction often have sympathetic hyperactivity and present with tachycardia, hypertension or both. While patients with inferior wall MI have increased Para sympathetic hyperactivity and present with bradycardia, hypotension or both.

Blood pressure may be normal in uncomplicated MI. In cardiogenic shock it may fall. Temperature – mild decease in temperature occurs in first 4-6 hours. A small spike occurs in next 24 hours of MI.

Respiratory rate may be elevated in left ventricular failure.

Cardiovascular system examination:-

Dyskinetic segment may be seen in 3-5th intercostal space.

Heart sounds may be soft; S_3 and S_4 may be present.

Systolic murmur after MI, either transient or permanent is the result of mitral regurgitation secondary to papillary muscle dysfunction or rupture of interventricular septum.

Pericardial rub may be heard in 20% of the cases. Dressler's syndrome occurs after 6 weeks of MI.

Respiratory system: - May be normal or patient may have creptations, if patient is in left ventricular failure.

Abdominal examination:-Tender hepatomegaly is present in cases of congestive cardiac failure.

Neurological examination: - In uncomplicated MI it is normal. There is increased incidence of cerebro- vascular accidents in cases of complicated MI.

Relevant investigations:

E.C.G

E.C.G findings in acute transmural MI are characteristic. The infarction processes evolves through 3 phases.

A) **Hyper acute phase** –Is characterized by 4 principle electrocardiographic manifestations in leads oriented to infarct surface.

1) Increased ventricular activation time.
2) Increased amplitude of R wave
3) Slope elevation of ST segment.
4) Tall and widened T waves.

B) Fully evolved phase –

1) Pathological Q wave or QS complexes are formed in this phase. The Q wave should be at least 0.04 seconds or at least 30% of amplitude of R wave.
2) Coved and elevated ST segment with convexity upwards.
3) Inverted symmetrical arrowhead T wave.

C) Phase of resolution

1) During this phase ST-segment returns to baseline.
2) T wave returns to normal upright configuration.
3) Only evidence of infarction may be Q or QS complexes.

SERUM ENZYMES

Irreversibly injured myocardial cells release a number of enzymes into the circulation, which can be measured.

These enzymes are-

1) Serum – glutamine oxaloacetic transaminase (SGOT).
2) Lactic Dehydrogenase (LDH)
3) Creatinine Kinase (CK)
4) Troponin I and T

TABLE-4 Activity of enzymes in a patient of AMI can be summarized as:

ENZYMES	EXCEEDS NORMAL VALUES	PEAK VALUES	RETURN TO NORMAL(IN DAYS)
CK	6-8 hrs	24 hrs	3-4 days
SGOT	8-12 hrs	18-36 hrs	3-5 days
LDH	24-48 hrs	3-6 days	8-14 days
Troponin I and T	12-16 hrs	24-36 hrs	10-14 days

The levels of isoenzyme CK-MB which present in heart muscle appears to be the most useful test for MI.

ECHOCARDIOGRAPH

It is helpful in prognostic information as well as characterization of ventricular function. It is helpful in assessing hypokinesia or dyskinesia, which may predispose to left ventricular mural thrombus. The other complications like Mitral regurgitation, ventricular septal defect, hemopericardium or ventricular aneurysms can be detected.

SCINTIGRAPHIC METHODS

Radionuclide studies like Hotspot imaging and cold spot imaging with Technetium 99 and Thallium 201 respectively helps in diagnosis and localization of infarction. Position emission tomography is used to assess metabolic function of infarcted regions.

CORONARY ANGIOGRAPHY

Coronary angiography is helpful in localization of obstruction in coronary artery and to assess extent of myocardial infarction.

TREATMENT[84]

Pre hospital phase

It is very important to recognize the acute myocardial infarction and shift the patient to the hospital as soon as possible. Mortality of acute MI is greatest during the first few hours after infarction. This is because of arrhythmias.

In coronary care unit

1. General measures
2. Pharmacological intervention
3. Treatment of haemodynamic disturbances.

1) General measures:

Control of pain: Alleviation of pain and anxiety reduces the sympathetic discharge and thus reduces myocardial oxygen consumption, cardiac output and arterial pressure. Sublingual nitroglycerine is given in the doses of 5 mg. Three doses can be repeated every 5 minutes if chest pain is not relived. . For prompt relief, 2-4 mg of Morphine can be given intravenously. It can be repeated every 5 minutes. As an alternative, Pethidine 75-100 mg intravenously can be along with Phenargan to avoid vomiting.

Oxygen: Oxygen inhalation reduces the area of ischemic injury. It should be given for up to 6 hours in uncomplicated MI. In left ventricular failure oxygen administration should be continued until left ventricular failure subsides.

Bed rest: In uncomplicated MI bed rest is advised for a minimum of 12 hours. In the absence of complications patients should be encouraged to resume an upright posture or sit on a chair by 24 hours. Patient can be made to ambulate in the room with in 4-5 days.

Diet: Because of the risk of vomiting and aspiration soon after the infarction, patients are advised not take orally for first 4-12hours of infarction.

Bowel: Aim is to avoid strain during evacuation. Bed side commode, diet rich in fiber and routine stool softeners are recommended.

Sedation: Diazepam (5mg) or Lorazepam (0.5 – 2mg) can be administered 3-4 times per day.

2) Pharmacological intervention

Treatment of Ml

Anti-thrombotic agents: The primary treatment is to establish and maintain patency of infarct related artery. It also reduces the patient tendency to thrombosis and thus reduces mural thrombus formation or DVT.

Aspirin in the dose of 325 mg sublingually initially and then 150 mg per day preferably life long. Clopidogrel 300 mg initially and then 75 mg for one month.

Thrombolysis: The principle goal is prompt restoration of coronary artery patency. It also limits infarct size, limits LV dysfunction, reduces incidence of serious

complications. Thrombolytic therapy reduces relative risk of in-hospital death up to 50%, when administered within first hour of onset of symptoms. Benefit of therapy is seen 3-6 hours after onset of infarction. It may benefit up to 12 hours, if there is ongoing chest pain. Intravenous Streptokinase, Urokinase and TPA are used in the setting of acute MI.

Beta-blockers: Beta-blockers when given, improves myocardial oxygen supply, reduce pain, limits infarct size and reduces incidence of serious arrhythmias. It is also used for secondary prevention on long-term basis.

ACE inhibitors: These drugs reduce mortality following MI. Maximum benefit is seen in elderly patients; in patients with anterior infarction, a prior infarction and globally depressed LV function.

Nitrates: Intravenous or oral nitrates are used to relive pain, limits infarct size.

Non-ST segment elevation MI and unstable angina are treated with anti-thrombotic agents, anti-platelet drugs (aspirin, clopidogrel), beta-blockers, ACE inhibitors and nitrates (whenever indicated). Patient should be watched carefully as the risk of development of ST segment elevation MI is high in first 24 hours.

3) Treatment and prophylaxis for arrhythmias. The incidence of arrhythmias is higher, soon after the onset of symptoms .The mechanisms responsible for infarct – related arrhythmias are autonomic system imbalance, electrolyte disturbances, ischemia and slowed conduction.

A) Tachyarrhythmias

1. Ventricular premature complexes This is the most common arrhythmias observed. Frequent multifocal VPC`s extra systole occurring in pairs and R-on T phenomenon require immediate treatment because they may be forerunners of fatal arrhythmias. Intraventricular ligocaine is the treatment of choice. A bolus dose of 1mg/kg iv; followed by 2-4mg/kg is administered.

2. Ventricular tachycardia

This needs immediate treatment with IV ligocaine. DC shock of 150-200 joules is given, in cases where the drug is ineffective. Repeated attacks can be prevented with Intravenous infusion of xylocaine, amiodrone or bretyleium.

3. Ventricular fibrillation

This can be life threatening. Immediate thump on the chest and external cardiac message should be promptly done. Defibrillation should be done DC shock (200-400)

4. Supra Ventricular arrhythmias

These require treatment with intravenous verapamil, beta-blockers or digoxin.

5. Accelerated Idio-ventricular rhythm

This rhythm is usually benign and does not require specific treatment. They are usually self-limiting. If haemodynamic compromise occurs, intravenous atropine suppresses it by increasing the sinus rate.

Brady arrhythmias:

Sinus node dysfunction may present as sinus bradycardia, sinus arrest or sino atrial arrest or sino atrial block usually due to vagal stimulation. They are common with inferior wall infarction. They respond to intravenous atropine. If they do not respond to atropine then temporary pacing may be required.

Treatment of haemodynamic disturbances:

In cases of hypotension, intravenous dopamine or dobutamine should be administered. In cases of right ventricular infarction, fluid replacement is needed.

Limitation of infarct size:

Infarct size is an important determinant of prognosis in acute myocardial infarction. Patients who succumb from cardiogenic shock generally exhibit massive infarct. Survivors with large infarcts frequently exhibit late impairment of ventricular function and their long-term mortality is higher than those with small infarct. The occurrence of complications is related to the infarct size. Limitation of infarct is done by accelerating reperfusion either pharmacologically or mechanically (angioplasty).

COMPLICATIONS

Many studies show that the complications, prognosis and mortality of acute myocardial infarction in women are different from male population. Women have a higher mortality rates than men after acute myocardial infarction.

A study by Nicholas et. al.[34] showed that in-hospital mortality rates were significantly[34] higher by 14% in women compared to 9% in men. 29% of women compared to 23% of men died during the three year follow up after infarction. This study concluded women who had a myocardial infarction were older; more often had hypertension, diabetes, previous heart failure cardiogenic shock and congestive cardiac failure. Complex arrhythmias occurred more frequently in men than women.

The following variables were defined as predictors of outcome for in hospital deaths. These are age, marital status, diabetes, cardiomegaly, Killip class, duration of chest

pain before admission, left ventricular ejection fraction and creatinine kinase levels.

The report from multi center investigation of the infarct size (MILIS) [86] showed a higher mortality rate after acute myocardial infarction in women, when compared to men after adjusting to baseline characteristics. This study was unlike the other studies where gender was not considered as an independent risk factor. This study was conducted mainly on black women, which had restricted eligibility. Many other studies showed that higher mortality rate has been attributed to older age of women and late presentation to the hospital. The other factors like diabetes, hypertension and obesity may contribute to a poorer prognosis[34].

With same base line characteristics, women with ST-segment elevation myocardial infarction had worse prognosis than men, but women with non ST-segment elevation myocardial infarction had similar outcomes compared to men. Women with unstable angina had better prognosis[87-88].

MATERIALS AND METHODS

1. SOURCE OF DATA:

The material for the present study was collected from patients who were admitted in ICU of BLDEU'S Shri B.M. Patil Medical College Hospital and Research Centre, Bijapur who were diagnosed with acute Myocardial infarction according to standard WHO criteria and were admitted within 24 hours of onset of chest pain.

The patients were informed about study in all respects and informed written consent was obtained.

Period of study was from November 2009 to March 2011.

2. METHOD OF COLLECTION OF DATA:

A total of 179 patients were taken for the study, every case was included after detail history clinical examination and relevant investigations which included ECG and CPK-MB.

Patient were followed up for a period of 30 days from the date of admission and were contacted after discharge by means of telephone or post card & were called up for follow up on 30^{th} day.

Analysis of all patients were done regarding morbidity and mortality first without adjustment for any base line characteristics, then with adjustment for age alone and finally with adjustment for age and other base line characteristics recorded at study entry were considered in the model.

3. INCLUSION CRITERIA:

Patients of any age who were admitted in ICU of BLDEU'S Shri. B.M. Patil's Medical College, Bijapur with symptoms of suspected acute myocardial infarction were included in the study.

Patients were selected on the basis of history and clinical examination.

4. EXCLUSION CRITERIA:

Patients presenting beyond 24 hours of the onset of symptoms of suspected acute Myocardial Infarction.

5. SAMPLING SIZE:

time period of study – November 2009-may 2011.

Incidence of the Myocardial Infarction is 7 % (8th edition A P I Text Book of Medicine)[7]. Margin of error is 4% at 95% of level of confidence the calculated sample size is 179 using the below statistical formula.

$$n = \frac{(1.96)^2(9)\,(1\text{-}P)}{d^2}$$

6. STATISTICAL ANALYSIS:

Data will be analyzed using following statistical methods

1. Diagrammatic presentation
2. Mean + SD percentage
3. Proper Statistical tests

a) Z test

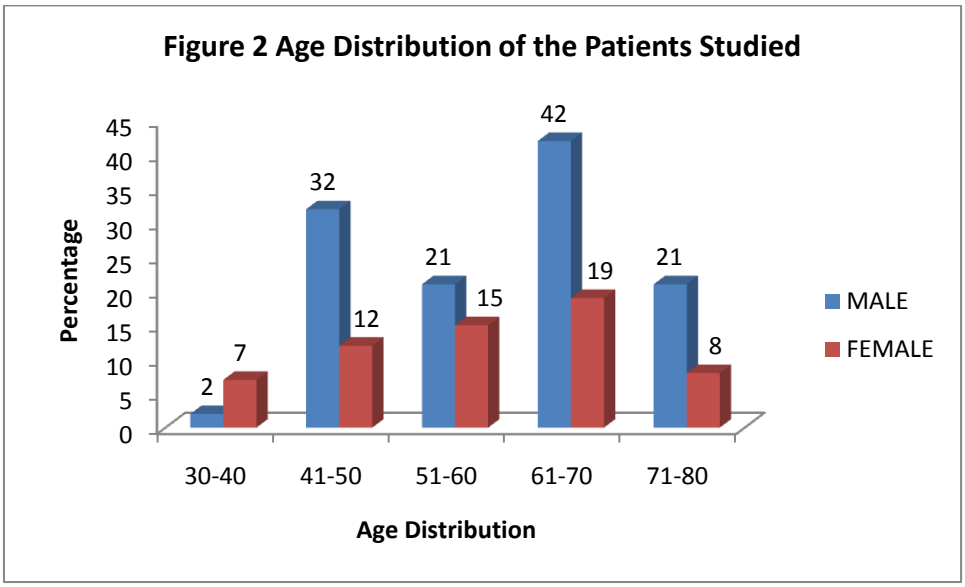
b) X^2 test (If necessary)

RESULTS

Study Design: Comparative prospective study of the gender difference in outcome of Acute Myocardial Infarction among 118 consecutive males and 61 consecutive females from November 2009 to May 2011 was undertaken to study the risk factors, clinical profile and immediate complications of acute coronary syndromes in women in comparison of men.

Table 5: Age distribution of the patients studied

Age in years	Male		Female		Combined	
	No	%	No	%	No	%
30-40	2	1.6	7	11.4	9	5.0
41-50	32	27.1	12	19.6	44	24.5
51-60	21	17.7	15	24.5	36	20.1
61-70	42	35.5	19	31.1	61	34.0
71-80	21	17.7	8	13.1	29	16.2
Total	118	100.0	61	100.0	100	100.0
Mean± SD	60.60±11.51		58.10±11.50		59.78±11.54	

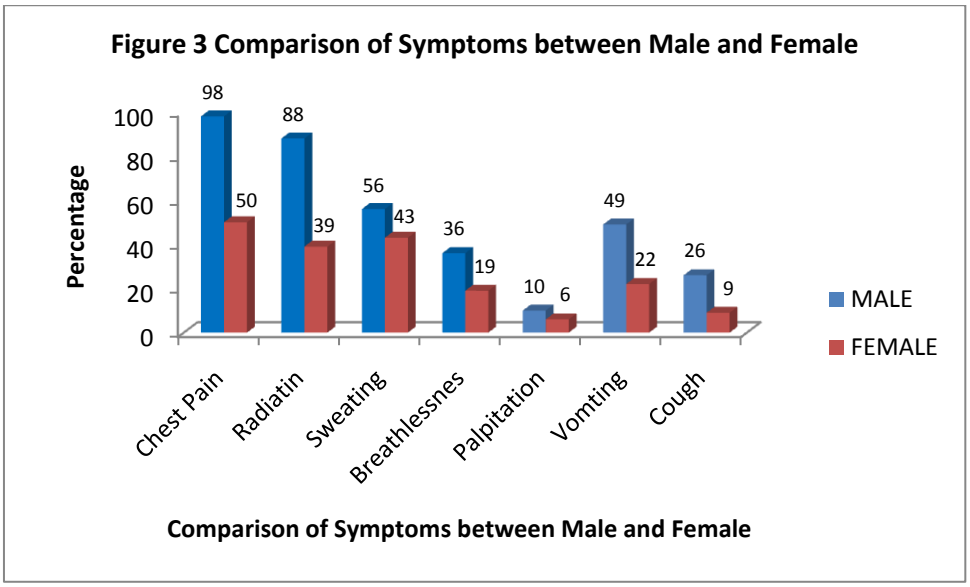


In this study, out of 118 male patients admitted, 34 patients were below the age of 50 years. The youngest patient was 35 years and the oldest patient was 80 years. The average age was 60 years.

In this study, out of 61 female patients admitted, 53 patients were below the age of 50 years. The youngest patient was 38 years and the oldest patient was 80 years. The average age was 58 years.

Table 6: Comparison of Symptoms between male and female

Symptoms	Male (n=118)		Female (n=61)		Combined (n=179)		P value
	No	%	No	%	No	%	
1. Chest pain	98	83.1	50	82.0	148	82.0	0.8559
2. Radiation	88	74.6	39	63.9	127	70.9	<0.1372
3. Sweating	56	47.5	43	70.5	99	55.3	0.0033**
4. Vomiting	49	41.5	22	36.1	71	39.7	0.4791
5. Breathlessness	36	30.5	19	31.1	55	30.7	0.93
7. Palpitation	10	8.47	6	9.84	16	8.94	0.7622
8. cough	26	22	9	14.8	45	19.6	0.2444



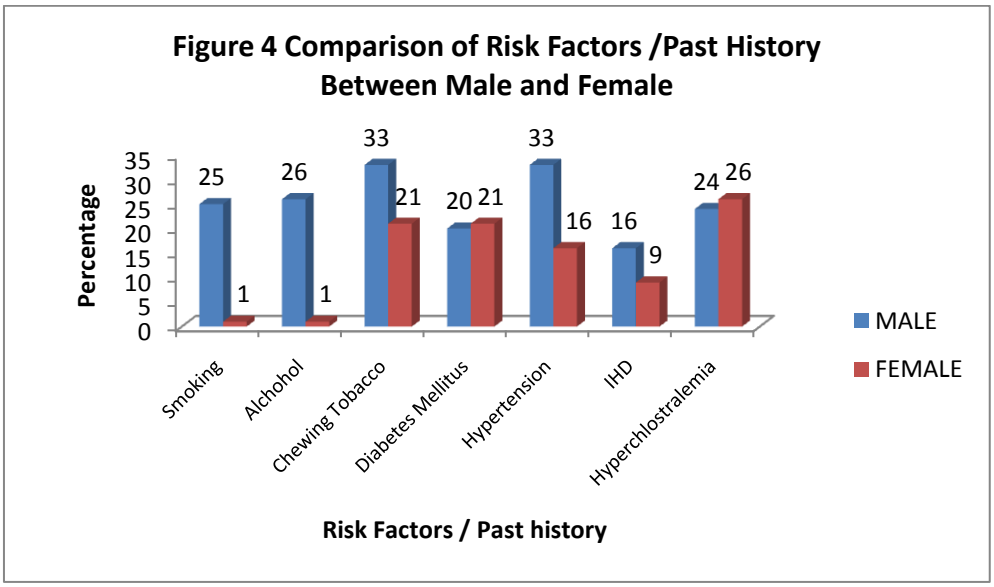
Symptomatology:

Chest pain was the main complaint in majority of male (83.0% and female (82.0%) patients. Vomiting was seen in 49(41.5%) male and 22(36.1%) female patients. sweating, which was next common symptom, seen in 43 (70.5%) female patients as compared to 56 (47.5%) patients in males, was significant finding.

Other symptoms (Syncope, Palpitation, Abdomen pain) occurred in less number of patients

Table 7: Comparison of risk factors/past history between male and female

Age in years	Male		Female		Combined		P Value
	No	%	No	%	No	%	
1. IHD	16	13.6	9	1.64	25	13.96	0.827
2. DM	20	16.9	21	34.4	41	22.90	0.0084
3. HTN	33	28	16	54.1	49	27.37	0.8049
4. Smoking	25	21.2	1	1.64	26	14.5	0.0004
5. Alcohol	26	22.0	1	1.64	27	15.08	0.0003
6. Hyper cholesterolemia	24	20.3	26	39.3	50	27.93	0.0016
7. Post Menopause	--	--	45	90.0	--	--	--
8. Chewing tobacco	33	28	21	34.4	54	30.16	0.3721



Considering the risk factors, hypertension (20.3%) was the significant risk factor in males followed by alcohol and smoking.

Considering the risk factors hypercholesterolemia (39.3.0%) was the significant risk factor in females. Diabetes was the next significant risk factor. Most of the females were post menopausal (90%)

Table 8: Comparison of BMI and Vital parameters between male and female

Results are presented in Mean ± SC (Min – Max)

Parameters	Male	Female	Combined	P Value
BMI	23.89±5.10 (17-38)	31.37±5.80 (18-44)	26.4+6.41 (10-44)	0.689
PR	95.27±18.71 (66-200)	97.18±24.18 (58-2000)	95.9±20.7 (58-200)	0.546
SBP	124.86±26.8 (70-200)	133.08±33.6 (50-190)	132.78±31.71 (50-200)	0.100
DBP	78.95±13.9 (70-200)	80.5±15.1 (60-130)	86.45±17.61 (40-130)	0.5008
RR	23.91±7.8 (13-30)	21.85±4.52 (12-38)	18.57±3.97 (12-38)	0.237

Tab 9: Comparison of levels of Lipid parameters between male and female

Results are presented in Mean ± SD (Min-Max)

Parameters	Male	Female	Combined	P Value
Cholesterol	2O4.95±33.14 (155-320)	237.37±24.18 (68-340)	216.0±45.3 (25-345)	0.218
HDL	36.14±9.26 (56-12)	34.45±10.77 (56-12)	35.6±9.81 (12-56)	0.371
LDL	108.03±12.76 (90-134)	108.50±13.39 (90-143)	108.0±12.9 (90-143)	0.999+
TG	107.03±28.86 (60-182)	130.21±37.44 (70-200)	115.0±33.8 (60-200)	0.601

+ Mean LDL is statistically significantly (Female patients have high mean LDL)

Table 10: Comparison of Abnormal lipid parameters between Male and Female patients. Results are presented in Mean ± SD (Min-Max)

Parameters	Male (m=118)	Female (n=61)	Combined (n=179)	P Value
Total chol(>200 mg/dl)	52 (44.1%)	42 (68.8%)	94 (53.0%)	0.0016
HDL (M<40mg/dl;F :<50mg/dl)	28 (23.7%)	16 (23.7%)	44 (91.0%)	0.7127
LDL (>100mg/dl)	64 (54.2%)	36 (54.2%)	100 (83.0%)	0.5416
Triglycerides (>160 mg/dl)	9 (7.63%)	18 (7.63%)	27 (85.0%)	0.0001

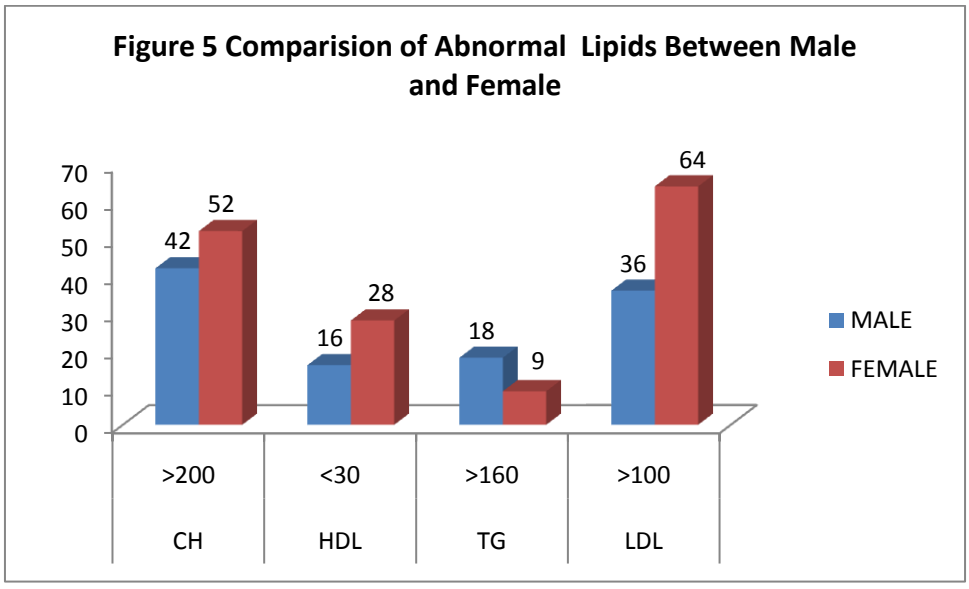
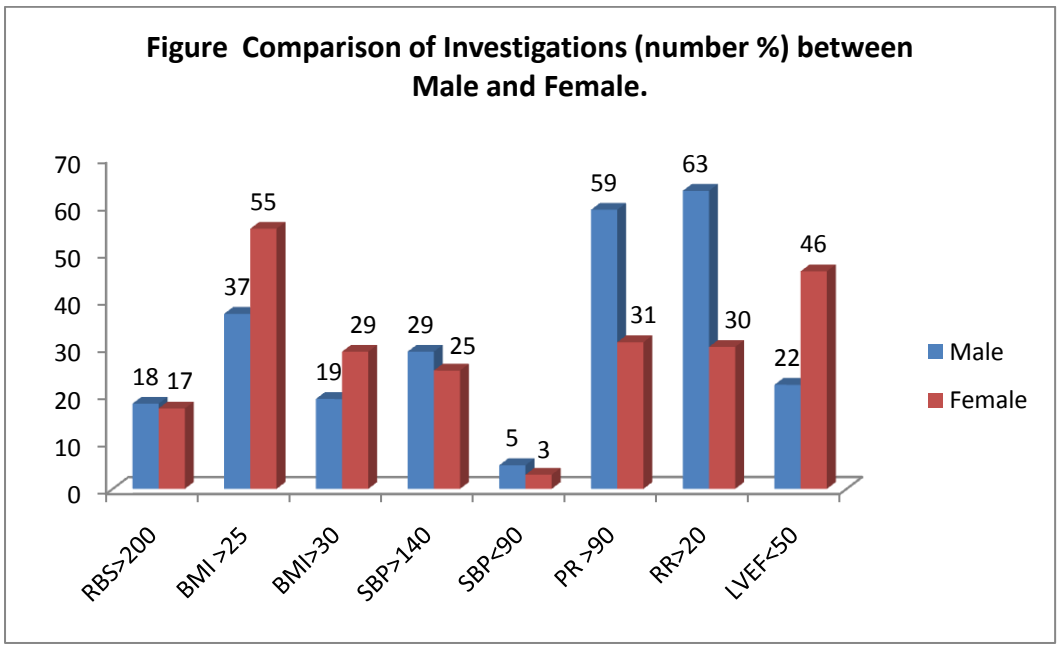


Table 11: Comparison of Investigations (number %) between male and female.

Investigations	Male (n=118)	Female (n=61)	Combined (n=179)	P Value
RBS # (>200mg/dl) (Over Weight)	18 (15.3%)	17 (27.90%)	35 (.0%)	0.0328
(BMI>25kg/m2) Obese (BMI>30 kg/m2)	37 (31.4%) 19(%)	55 (90.2%) 29(%)	92 (.0%) 38(%)	0.0145 0.0004
Hypertension (>140/90mm Hg) Hypotension	29 (24.6%)	25(.0%)	54 (41%)	0.3631
(<90 systolic mm Hg)	5(4.24%)	3(4.92%)	7(.0%)	0.4690
Pulse rate (>90 bpm)	63(53.4%)	30(49.2%)	93(.0%)	0.8292
Resp.rate (>20/min)	63(53.4%)	30(47.2%)	31(31.0%)	0.607

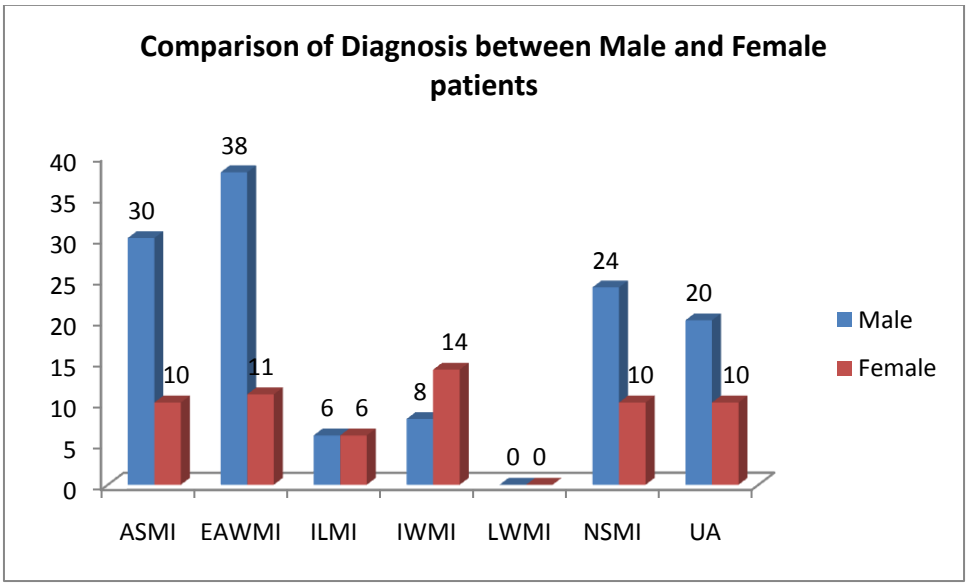


 # 11 male and nine female were non diabetic patients

12 males and eight females were non-hypertensive

Table 12: Comparison of Diagnosis between Male and Female patients.

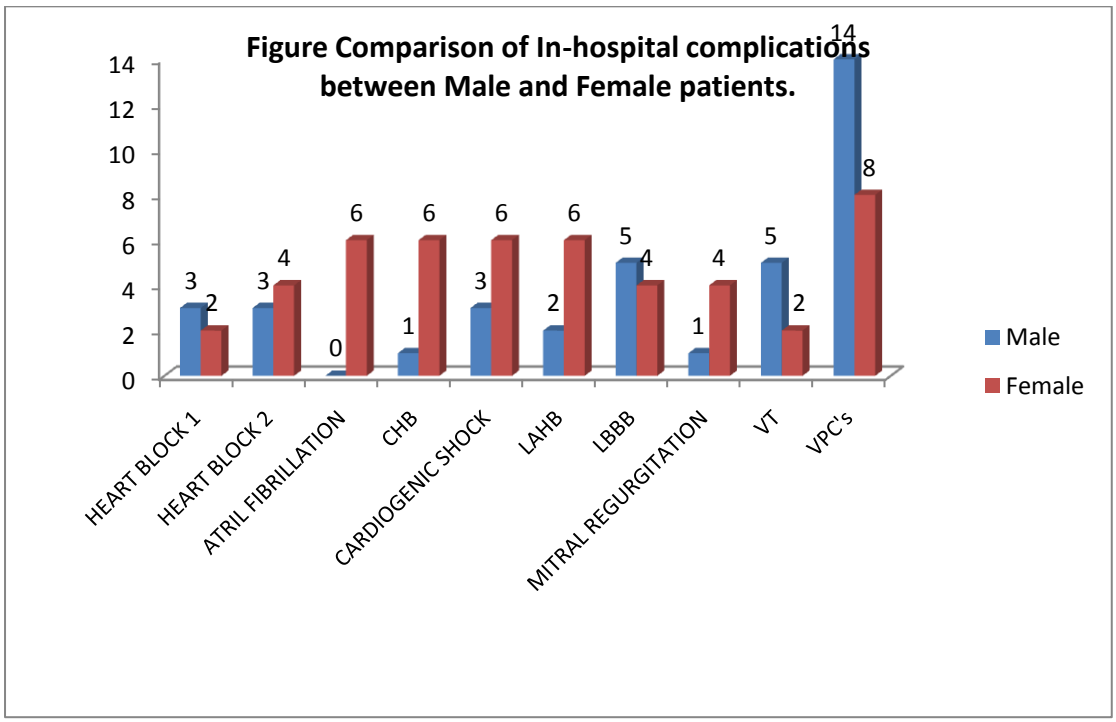
Parameters	Male (n=118)		Female (n=61)		P Value
	No	%	No	%	
ASMI	30	35.4	10	16.3	0.169
EAWMI	38	32.2	11	18.0	0.043
ILMI	6	5.08	6	9.8	0.228
IWMI	8	6.78	14	22.9	0.001
LWMI	--	--	0	--	---
NSMI	24	20.3	10	16.3	0.523
UA	20	16.9	10	16.3	0.924



Anterior wall infarction accounted for 68 (67%) in males and 21 (34%) in females. Next common is inferior wall infarction, which is 8 (6.7%) in males and 14 (22.9%) in females.

Table 13: Comparison of In-hospital complications between Male and Female patients.

Parameters	Male (n=118)		Female (n=61)	
	No	%	No	%
HEART BLOCK 1	3	2.54	2	3.27
HEART BLOCK 2	3	2.54	4	6.55
Atrial Fibrillation	-	-	6	9.83
CHB	1	0.84	6	9.83
Cardiogenic shock	3	2.54	6	9.83
LAHB	2	1.69	6	9.83
LBBB	5	4.23	4	6.55
Mitral regurgitation	1	0.84	4	6.55
VT	5	4.23	2	3.2
VPC s	14	11.8	8	11.86



In-hospital complications are more common in women 49 (%) compared to men 38 (%)

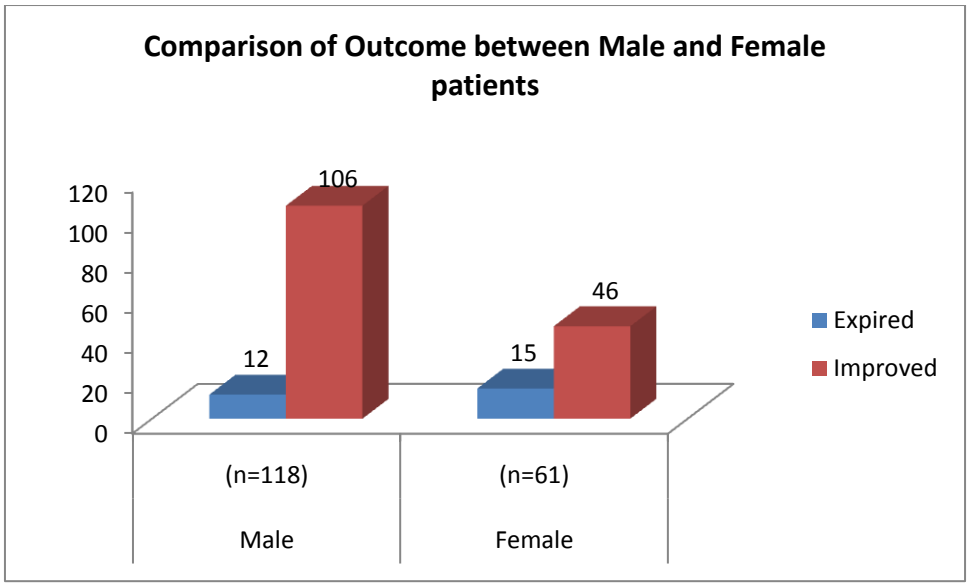
Table 14: Comparison of CKMB and LVEF between. Male and Female patients

Results are presented in Mean + SD (Min-Max)

Investigations	Male (n=118)	Female (n=61)	Combined (n=179)	P Value
CKMB	140.42±102.60 (12-453)	116±71.52 (12-400)	140±24.00 (12-435)	0.369
LVEF	35.54±6.32	40.32±7.45	00.0±00.0	0.628

Table 15: Comparison of Outcome between Male and Female patients

Out come	Male (n=118)	Female (n=61)	Combined (n=179)	P Value
E	12(10.2%)	15(24.59%)	27(15.08%)	0.0106
I	106(89.8%)	46(75.41%)	152(84.91%)	0.0106



There was significant mortality in women 15 (24.0%) compared to men 12(10.2%)

*** Moderately significant 0.01<P≤0.05**

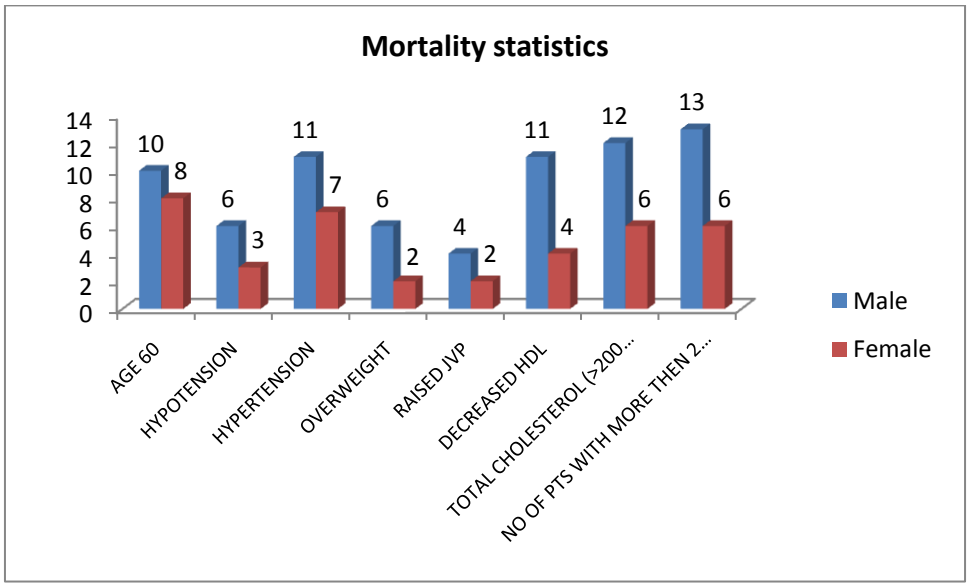
**** Strongly significant P≤0.01**

Table 16: Mortality statistics

CHARACTERISTICS	FEMALE DEATH PTS (OUT OF 15)	MALE DEATH PTS (OUT OF 12)
AGE > 60	10	8
HYPOTENSION	6	3
HYPERTENSION	9+2*	4+3*
OVERWEIGHT	6	2
RAISED JVP	4	2
DECREASED HDL	11	4
TOTAL CHOLESTEROL (>200 MG/DL)	12	6
NO OF PTS WITH MORE THEN 2 RISK FACTORS	13	6

* NEWLY DETECTED CASES

Most of the patients who died were above 60 years of age. Most of them had two or more risk factors.



DISCUSSION

Cardiovascular disease is now a major cause of morbidity and mortality for both men and women, with acute coronary syndromes developing about a decade later in women. A number of studies have highlighted the differences between men and women in the risk factors, clinical profile and immediate complications of acute coronary syndromes. Several studies from abroad and from our own country have brought out the differences in the clinical profile between the sexes.

The observations made in 61 female and 118 male patients with acute coronary syndromes who are admitted in hospital attached to SHRI B M PATIL MEDICAL COLLEGE are included in this study.

MEN AGE IN DIFFERENT STUDIES

Table No. 17

Shows the mean age of females in various studies.

Various studies	Men age
Gupta et. al [90]	27.6+6.6 yrs
Myeong et.al[55]	57.3+11.9 yrs
Emingel et al [91]	53.8±9.5 yrs
Present study (F)	58.10±11.51 yrs
Present study (M)	60.60±11.51 yrs

The mean age of occurrence of acute coronary syndromes in females in this study is 58.10±11.51. This is comparable with the other studies. The youngest patient was 40 yrs. and the oldest was 80 yrs.

The mean age of occurrence of acute coronary syndromes in males in this study is 60.60±11.51 yrs. The youngest patient was 35 yrs. and the oldest was 80 yrs.

Comparison of risk factors in females in different studies

Table No. 18 Shows risk factors in various studies.

Risk factors	**Seyoum al [92] (%)**	**Heish et al[93] (%)**	**Kata yama al [18] (%)**	**Zhan g et al [94] (%)**	**Grun dtvig a et al [95] (%)**	**Present study (F) (%)**	**Present Study (M) (%)**
Post-menopausal	30	97.7	84	--	--	90	--
Diabetes mellitus	32.8	23.7	44	10	40.80	34*	16*
Hypertension	37.2	55	53	54	48	28**	49**
Family history of IHD	-	7.0	18	30	37.20	18	4
Hypercholesterolemia	20	18.2	81	51	56.6	5O	24
Overweight	45	4	58	-	8.8	22.0	26
Tobacco Chewing	-	-	--	15	--	34	33
Cigarette smoking oral contraceptives	17 Nil	11.5 Nil	4 Nil	33 -	3.6 Nil	1.64 --	25 --

- **11 males and 9 females were non-diabetic newly detected patients**

**** 12 males and 8 females were non-hypertensive newly detected patients.**

Table 19: Comparison of risk factors

CHARACTER ISTICS	Acute coronary syndromes (Augusto et al [89])			Acute coronary syndromes (PRESENT STUDY)		
	MEN	WOMEN	P VALUE	(Male) (n=118)	Female (n=61)	P VALUE
AGE	59±3.3	58±3.8	0.67	60.60±11.51	58.10±11.50	0.232
Hypertension %	57.7	40.7	0.12	33 (28%)	16 (54%)	0.8049
Diabetes %	9.3	9.2	0.99	2O (16%)	21(54%)	0.0084
Current of former smoker	74.7	63	0.67	25(21%)	1(1,64)	<0.001

1. Age

Mean age in Augusto et al [89] studies was 59±3.3 yrs in males and 58±3.8 yrs in females. Mean age in the present study was 60.60±11.51 yrs in males and 58.10±11.50 yrs in females.

2. Menopause

The incidence of acute myocardial infarction increased in the postmenopausal women. In this study, 90% belonged to this group. Heish et al [93] observed 97% of their cases were post-menopausal. There were 3 patients in the pre-menopausal period in this study.

The discrepancy in cardiovascular disease between the sexes has been attributed to hormones and menopause. Post menopausal hormone replacement

therapy has proved to reduce the relative risk of coronary artery disease to 0.3-0.79 and improved survival in women. Thus, menopause is a strong risk factor for acute myocardial infarction.

3. Hypertension.

The present study revealed hypertension as the next important risk factor. The incidence in various studies varied between 30 to 54% [18, 92, 93-95]. The present study showed an incidence of 33% in females and 16% in males. 12 males and 8 females were non-hypertensive newly detected patients. Zhang et.al[64] noted hypertension in 54% cases. 12 patients were both diabetic and hypertensive (40%).

4. Diabetes Mellitus.

Diabetes mellitus is the next important risk factor. The incidence in various studies varied from 26 to 56%[18, 92, 93-95]. The present study 21 (54%) females and 20 (16%) males were diabetics. 16 male and 9 female were non diabetic newly detected patients. The remaining patients were known cases of diabetes. Thus, diabetes mellitus is a strong risk factor for CAD.

5. Family history of Ischemic heart disease.

The incidence in various studies varied from 7-37% [18, 92, 93-95]. The incidence in this study was 18% (F) and 15% (M), which was comparable to the incidence in Katayama et al[18] Our study is comparable to these studies.

6. Hypercholesterolemia

The incidence of hypercholesterolemia in this study was 50% (F) and 24% (M), which was similar to the Zhang et.al [94] study (51%).Grung et al [95], study showed an incidence of 56.6% our study is comparable to these studies.

8. Tobacco Chewing

The incidence of tobacco chewing in this study was 33% (F). Seyoum et al [92] and Heish et al [93] noted an incidence of 10% and 11.5% respectively in females.

9. Smoking

Smoking (25.0%) was the significant risk factor in males.

In the present study, only one woman was smoker and none of them used oral contraceptives. This was comparable to the Indian studies by Seyoum et al[92] and Hsin et al [93] while the incidence of smoking in western population was 49.3%.

Table No-20. CLINICAL PRESENTATION COMPARISION IN FEMALES.

Symptoms	Seyoum et al [96] (%)	Charles et al [67] (%)	Culic et al [98]	Thuresson et al [79] (%)	Present Study (%) (F)	Present study (%) (m)
Chest pain	73.8	86.7	79.9	80	82.0	83.0
Sweating	64.20	41.8	48.1	66	70.5	47.5
Vomiting	44	56	34	44	36.1	41.5
Breathlessness cough	23.8	52	48.4	33	55.0	30.5
	18	9.3	7.8	30	22	45
Palpitation	Nil	12.3	13.3	14	16	8

1. Chest pain –

Chest pain was the most common presenting symptom in this study (82% in females and 83% in males). Pain was associated with sweating in 70% (F) and 47% (M) of cases. In most studies, [79, 96-98] chest pain was seen in 73-86% of cases and sweating was seen in 41-66% of cases, which are comparable to our study.

2. Breathlessness –

Breathlessness was observed in 55% (F) and 30% (M) of the cases. The incidence in other studies varied from 23-52% [79, 96-98] of the cases.

3. Vomiting – Vomiting was seen in 36% in female and 46% in male. The incidence in difference studies varied from 18% to 64.20%[79, 96-98.]

4. Syncope –

Syncope as a presenting feature was seen in 2% in males and none of in females in this study. The incidence in various studies varied from 7.8% to 30% [79, 96-98].

5. Pain in abdomen

6% of females and 2% males presented with pain in abdomen. The incidence in different studies varied from 0-14% [79, 96-98.]

6. Palpitation

Palpitation occurred in 2% of males 4% of females.

Table 21: Physical signs and other characteristics of acute coronary syndromes comparison.

CHARECTERISTICS	**Acute coronary syndromes (Augusto et al [89])**			**Acute coronary syndromes (PRESENT STUDY)**		
	MEN	**WOMEN**	**P VALUE**	**Male (n=118)**	**Female (n=61)**	**P VALUE**
Heart rate beats/min	74	76	0.008	95.27+18.71	97.18+24.18	0.592
Systolic BP in mm hg	130	132	0.03	124.86+26.6	133.08+33.6	0.1004
Elevated total cholesterol %	34	43	<0.001	52(44.1%)	42(68%)	0.316

Physical signs –

1) Heart rate: Mean heart rate in August et al [89] study was 74 beats/min in males and 76 beats/min in females. Present study shows mean heart rate of 95.27+18.71 beats/min in males and 97.18+24.18 beats/min in females.

2) Blood Pressure

Augusto et al[89] study shows mean Systolic BP (in mm hg) of 130 in males and 132 in females Present study mean Systolic BP (in mm hg) of124.86+26.6 in males and 133.08+33.6 in females.

Hypertension was seen in 33(28.0%) cases in males and 16(54.0%) in females. 12 males and 8 females were newly detected.

3) Heart sounds

Third heart sound was present in 3 (6%) of male 8 (16%) of females. These patients had left ventricular failure.

4) Murmurs: Systolic murmur was found in 3(6%) of females and none of males.

5) Cerebrovascular disease: Occurred in 2 patients (one male and one female)

6) Left ventricular clot: Seen in 2 female patients and one male patient.

LABORATORY INVESTIGATION

1) CKMB

Present study shows mean CKMB of 140.42+103 u/dl in males and 116.88+71.5 u/dl in females. Culic et al [98] noticed mean CKMB of 105+78 u/dl in males and 94+69 u/dl in females.

2) Hypercholesterolemia:

Hypercholesterolemia was seen in 24 (20.3%) of males 26 (39%) of females. Augusto et al [89] study showed elevated total cholesterol in 34% of males and 43% of females.

SITE OF INFARCTION

Table No. 22 Shows the different sites of infarction in various studies.

Site of infarction	Seyom et al [92] (%)	Grung et al [95] (%)	Bhat et.al [96] (%)	Howard et.al[91] (%)	Present Study (%) M	Present study (F) (%)
Anterior wall infraction	51	46.6	41.7	37	67	22
Inferior wall infarction	20	9.8	29.8	35	6.7	22.9

In this study anterior wall infarction was observed in 67% of males 22% of females. This was similar to the incidence observed by Seyom et.al[92]. (41.7%) Grung et al[95]. (37%) and Inferior wall infarction was the next commonest site, observed in 6.7% of males 22.9% of females. This was similar to the incidence observed by Bhat et.al[96] (29.8%) when compared to female unstable angina is seen in 20(16.9%) male patients where 10 as in female it is seen in10 (16.3%)patients.

COMPLICATONS

Table No.-23 shows the incidence of various complications in different studies.

Complications	Seyoum et al [92] (%)	Bhat et al [96] (%)	Augusto etl al [89] (%) M	F	Present Study (%) (M)	Present study (%) (f)
Left ventricular failure	30	14.2	--	11.9	16.0	26.0
Cardiogenic shock	10	7.1	4.3	--	2.54	9.83
VPC's	10	25	--		11.8	11.86
Atrioventricular block	5	25	5.4	5.4	0.0	0.0
Ventricular tachycardia	5	25	4.3	4.2	4.23	3.2
Post infarct VSD	Nil	Nil	--	-	Nil	Nil
Left anterior hemiblocks	Nil	25	--	--	- Nil	Nil
Ventricular fibrillation	Nil	25	1.3	2.9	Nil	Nil
Percarditis	Nilf	3	-	-	Nil	Nil
Atrial fibrillation	-	-	8.6	11.0	Nil	9.83
Acute mitral regurgitation	-	-	0.6	1.6	0.84	6.55

Left ventricular failure was the most common complication in this study accounting to 26% of the females and 16% of males. Bhat etal[96] observed left ventricular failure in 14.2% cases.

The incidence of cardiogenic shock was 9.83% in females and 2.54% in males. Seyoum et al [92] 10%.

VPC's occurred in 6% of females and 4% of males. Bhat et.al [96] noticed VPC's in 25% of the cases.

Ventricular tachycardia occurred in 3.2% of females and 4.23% of males. The incidence in various studies varied between 4 to 25% of the cases. [89, 92, 96.]

Left anterior hemi block was seen in 1.69% of males.

MORTALITY

Among 179 patients (118 male and 61 female), 12 (10.2%) deaths occurred in males and 15 (24.59%) deaths occurred in females.

Table No – 24 Shows mortality among males and females in various studies.

Sex	Howard Et al [91] (%)	Stone et.al [94] (%)	Culic et al [98] (%)	Present study (%)
Female	17.5	9.3	21.4	24.59
Male	12.3	2.3	12.8	10.2
Ration	1.42 :1	3.32:1	1.76:1	2.8:1

Table No. 25 Show the comparative study of the cause of the death.

Cause of death	Gupta et al 90 (%)	Present study (F) %	Present study (M) %
Cardiogenic shock	13.04	9.83%	2.54%
Left ventricular failure	39	12%	10%
Ventricular tachycardia	Nil	4%	3%
Mitral regurgitation	30	0.84%	6.55%

Among 15 deaths in females, 13 patients died during first 5 days and the due to ventricular tachycardia in 2 patients and cardiogenic shock in 6 patients. 4 patients died due to complete heart block and the remaining 3 patients due to mitral regurgitation.

Among 12 deaths in males, 10 patients died during first 5 days following Ventricular tachycardia in 4 patients and Ventricular fibrillation in 4 patients. 2 (2%) deaths were due to cardiogenic shock and the remaining 2 patients following mitral regurgitation.

DISCUSSION

The Ischemic heart disease is leading cause of morbidity and mortality in present day world. Although mortality has declined significantly but rate is still high.

Age is a powerful predictor of short term outcome in acute myocardial infarction. In this study, a total of 179 consecutive cases with acute myocardial infarction admitted from October 2009 to may 2011 were studied. Out of 179 patients 116 were males (average age is 60 yrs) and 61 were female (average age is 59yrs) which was similar to Emingel et al[91] & myeong et al [55].

Clinical presentation: Chest pain was the most common presenting symptom in this study (82% in females and 83% in males). Pain was associated with sweating in 70% female and 47% male of cases. In most studies [79, 96-98] chest pain was seen in 73-86% of cases and sweating was seen in 41-66% of cases, which are comparable to our study. Breathlessness was observed in 55% female and 30% male of the cases. The incidence in other studies varied from 23-52% [79, 96-98] of the cases. Vomiting was seen in 36% in female and 46% in male. The incidence in different studies varied from 18% to 64.20%[79, 96-98.] Other symptoms (Syncope, Palpitation, Abdomen pain) occurred in less number of patients. Myeong *et al*[55] showed that female patients were more likely to present late in the case acute myocardial infarction and has a number of reasons, i.e., in our social setup people are less compliance for the treatment of females, and females are more likely to present with atypical chest pain .

Modifiable risk factors account for a large (over 90%) proportion for the risk of an initial acute myocardial infarction. The effect of these risk factors is consistent in men and women, the effect of the risk factors is particularly striking in young men and women indicating that most premature myocardial infarction are preventable. Two most important risk factors are smoking and abnormal lipids (together they

account for about two-thirds of an acute myocardial infarction). The incidence of hypercholesterolemia in this study was 50% female and 24% male which was similar to the Zhang et.al [94] study (51%) and Grung et al [95].

In the present study, one woman was smoker and none of them used oral contraceptives. This was comparable to the Indian studies by Seyoum et al[92] and Hsin et al [93] while the incidence of smoking in western population was 49.3%.

Hypertension, diabetes, and abdominal obesity were the next most important risk factors in men and women the present study revealed hypertension as the next important risk factor. The incidence in various studies varied between 30 to 54% [18, 92, 93-95]. The present study showed an incidence of 33% in females and 16% in males. 12 males and 8 females were non-hypertensive and newly detected patients. Zhang et.al[64] noted hypertension in 54% cases. 12 patients were both diabetic and hypertensive (40%).

Diabetes mellitus is the next important risk factor. The incidence in various studies varied from 26 to 56%[18, 92, 93-95], the present study 21 (54%) females and 20 (16%) males were diabetics. 16 male and 9 female were newly detected patients

We found that the coexistence of HT and DM was common (12.6%) among patients with MI Considerable experiment have shown that elevated blood pressure is critically important in the pathogenesis of diabetic heart disease. Coronary artery disease is much more common in patients with both DM and HT than in patients with DM or HT alone, and the development of atherosclerosis was found to be accelerated, with more plaque fissuring and a lower coronary perfusion reserve index. Patients with combined DM and HT also tend to have impaired systolic and diastolic ventricular function with more left ventricular hypertrophy and congestive

heart failure than counterparts with DM or HT alone. Around 6 males and 5 females are both tp2 dm and hypertension.

The importance of modifying risk factors is supported by data from randomized trials—eg, blood-pressure lowering, lipid lowering, dietary modification—or persuasive evidence of causality from observational studies (eg, smoking cessation). Some studies have suggested that a pill that combines a statin, antihypertensive drugs, and aspirin, together with avoidance of smoking, could potentially reduce the risk of myocardial infarction by more than 80% to 90%.

Studies suggest that one of the major emphases in research should be to understand why currently known risk factors develop in some individuals and populations, and to identify approaches to prevent their development or reduce them. For example, understanding the mechanisms by which social factors affect development of risk factors (urbanization , food and tobacco policies, shifts occupation from energy expending jobs to sedentary ones, and urban structure, etc) could lead to new approaches to prevent development of risk factors (primordial prevention), which in turn could reduce coronary heart disease.

In this study anterior wall infarction was observed in 67% of males 22% of females. This was similar to the incidence observed by Seyom et.al[92]. (41.7%) Grung et al[95]. (37%) and Inferior wall infarction was the next commonest site, observed in 6.7% of males 22.9% of females. This was similar to the incidence observed by Bhat et.al[96] (29.8%) when compared to female unstable angina is seen in 20(16.9%) male patients where as in female it is seen in10 (16.3%)patients.

Significant difference was observed between the sex in the occurrence of complications. after acute myocardial infarction, females have been reported to have

left ventricular failure (26%), cardiogenic shock(9.83%),atrial fibrillation(9.83%),,mitral regurgitation (6.55%) and vpc,s (11.6%) more frequently than males .

Males have a higher incidence of left ventricular failure (16.1%), vpc's (11.8%), and cardiogenic shock (2.54%). Over all incidence of complications are more in case of female than in males which is similar to studies done by seyom et al [92] and bhat etal[96]

Presence of certain risk factors, laboratory investigations and other co morbid non-cardiac conditions can affect the outcome in patients with acute myocardial infarction. This may be due both modifiable and non modifiable factors

Female gender was another feature which has higher mortality in patients with Acute Myocardial

Infarction.6–10 this may be due to the fact that females are usually older than males at presentation among 179 patients (118 male and 61 female), 12 (10.2%) deaths occurred in males and 15 (24.59%) deaths occurred in females. Among 15 deaths in females, 13 patients died during first 5 days and the cause is ventricular tachycardia in 2 patients and cardiogenic shock in 6 patients, 4 patients died due to complete heart block and the remaining 3 patients due to mitral regurgitation.

Among 12 deaths in males, 10 patients died during first 5 days following Ventricular tachycardia in 4 patients and Ventricular fibrillation in 4 patients. 2 (2%) deaths were due to cardiogenic shock and the remaining 2 patients following mitral regurgitation.

Mean stay in the hospital was 5.94±3.40 days. In a study by Gurwitz *et al*, [99] showed that

Although in-hospital death after acute myocardial infarction has recently declined for patients less than 65 years of age but improvements have not been realized for old age groups in our study it was observed that there is increase in mortality after myocardial infarction with increase in age

In a retrospective survey done in USA where 384 878 patients who were enrolled in the National Registry of Myocardial Infarction between 1994 and 1998. Kenfield *et al.*[10] found that the overall mortality rate for women for the period within 1 week of infarction was 16·7% compared with 11·5% for men. The ages of the patients were 30–89 years. The gender difference was age-related: in patients less than 50 years, the mortality for women was more than twice that of men — with increasing age, the differences between the sex disappeared and there was no significance beyond 74 years. This concludes that coronary artery disease in pre-menopausal women may have a different path physiology.

Women had more complications than men during hospitalization Women may not anticipate the possibility of coronary ischemia in the same way as men so that they underestimate the gravity of chest pain when it occurs.

On the other hand, in our study, a small number of deaths were observed, 15 females and 12 males.

In terms of percentage, a higher mortality occurred in the female sex than in the male sex

CONCLUSION

Women with acute coronary syndromes had similar clinical symptoms compared to men.

Postmenopausal state, hypertension, hypercholesterolemia and diabetes were the commonest risk factors in women. Smoking, hypercholesterolemia was the risk factors in men.

The morbility & mortality in women following acute coronary syndrome increases with age

SUMMARY

The observation was made in a total of 179 patients of which 61 are women and 118 patients men with acute coronary syndromes studied over a period between Oct-2009 to May-2011 who were admitted ICU of BLDEU'S SHRI B M PATIL MEDICAL COLLEGE.

- Mean age in the present study was 60.60±11.51 yrs in males and 58.10±11.50 yrs in females.
- Chest pain was the main complaint in majority of male and female patients (82%in females and 83% in males). Others symptoms (syncope, palpitation, pain abdomen) occurred in less number of patients (10% of males and 10% of females)
- 90% of females belonged to postmenopausal group.
- In males, hypertension 33 (28%) was the significant risk factor followed by smoking 25 (21%), hypercholesterolemia 24 (20.3%), diabetes mellitus 20 (16.9%)& Overweight 19(16.1 %,).
- In females, hypercholesterolemia 26(39%) was the significant risk factor followed by diabetes mellitus 21 (34.4%), hypertension 16 (54%) overweight 29(47.5%) family history 18% and tobacco chewing 34%.
- Mean heart rate of 95.27±18.71 beats/min in males and 97.18±24.18beats/min in females. Mean systolic BP of124.86±26.8 mm hg in males and 133.08±33.6 mmhg in females.

- Hypertension was seen in 33 (28.0%) cases in males and 16(54.0%) in females 12 males and 8 females were newly detected hypertensives.
- RBS more than 200 mg/dl was seen in 18(15.3%) male patients and 17(27.9%) females' patients. 11 male and 9 female were newly detected diabetic patients.
- Third heart sound was present in 3(6%) of males and 8(16%) of females. These patients had left ventricular failure.
- Systolic murmur was found in 3 (6%) of females and none of males.
- Cerebrovascular disease occurred in two patients (one male and one female)
- Left ventricular clot was seen in 2 female patients and none of males.
- Present study shows mean CKMB of 140.42+103 u/dl in males and 116.88+71.5 u/dl in females.
- Anterior wall infarction was the next commonest site, observed in 68 (67.6%) males 21(32%) females. Inferior wall infarction is seen in 8(6.78%) male and 6 (9.8%) female.
- NSTMI was seen in 24(20.3%) males & 10 (16.3%) females; UA was seen in 20(16.9%) males and 10(16.3%) females.
- In females, the most common complication was Left ventricular failure 26% followed by ventricular premature beats 11.6%, cardiogenic shock 9.83% LBBB 9.8%, and Acute mitral regurgitation 6.55% and Ventricular tachycardia 3.2%.

- In males, the most common complication was Left ventricular failure 16% followed by VPC's 11%, Ventricular tachycardia 4.2%, LBBB 4.23%, cardiogenic shock 2.54% and Left anterior hemi block 1.69%.

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PROFORMA

NAME DOA

AGE DOE

INCOME I.P.NO

OCCUPATION

ADDRESS

CHIEF COMPLAINTS

H/O PRESENTING COMPLAINTS

1)	**CHEST PAIN**	**YES/NO**
	1) Duration	Hours/day/months
	2) Onset	Sudden/gradual
	3) Site	Precordia l/retrostemal
	4) Nature aching	Squeezing /crushing /dull
	5) Sweating	Mild/moderate/severe
	6) Radiation neck/abd/jaw	Left arm/right arm
	7) Any other related factors.	
2)	**BREATHLESSNESS**	**YES/No**
	1) Duration	Hours/days/months
	2) Onset	sudden/gradual

3) Severity — Grade 1

Grad 2

Grade 3

Grade 4

4) Orthopnea

5) PND

3) PALPITATIONS — **YES/No**

a. Duration — continuous / paroxysmal

b. Onset — abrupt/gradual

c. Present at — rest/exertion/emotional onset

d. Any other symptoms ass.

4) SWEATING — **YES/No**

Duration

Onset associated with pain

5) SYNCOPE — **YES/NO**

6) PAIN ABDOMEN — **YES/NO**

7) OTHER SYMPTOMS

PAST HISTORY

a. H/O angina pain/M.I Yes/no

b. H/o Diabetes mellitus Yes/no

c. H/o Hypertension Yes/No

d. H/o CVA Yes/No

e. H/o Similar complaints

FAMILY HISTORY

1) IHD/DM/Hypertension

2) Similar complaints in the family Yes/no

PERSONAL HISTORY

1) Tobacco chewing/smoking

   Panparg / guttka/alcohol.

2) Oral contraceptive use Yes/no

3) Personality Type A/B

4) Diet Veg / non-veg

5) Appetite normal/disturbed

6) Sleep Normal/disturbed

7) Bowel and bladder normal/disturbed

MENSTRUAL HISTORY

1) Age of menarche
2) Married life/no of children
3) Age of menopause

GENERAL PHYSICAL EXAMINATION

1)	Appearance	Normal/ill looking
2)	Pallor	Present/absent
3)	Icterus	Present/absent
4)	Cyanosis	Present/absent
5)	Clubbing	Present/absent
6)	Signs of hyperlipidemia senilies	xanthoma/xanthalesma/arcus
7)	Pedal edema	Present/absent
8)	Significant lymphadenopathy	Present/absent
9)	BMI	

VITAL SIGNS

1) Pulse
2) BP
3) Respiratory rate
4) Temp

SYSTEMIC EXAMINATION

CARDIOVASCULAR SYSTEM

INSPECTION

JVP

1) Shape of chest
2) Precordial fullness
3) Apical impulse
4) Pulsations (epigastric, parasternal)

PALPATION

1) Apical impulse
2) Parasternal heave
3) Epigastric pulsations
4) Trill
5) Other palpable sounds

PERCUSION

ASCULTATION

1) Heart sounds — normal/faint/accentuated
2) Added sounds — S3/S4/S3S4
3) Murmur — Yes/no

1) Site

2) Grade

3) Position best heard

4) Relation to respiration

5) With bell of stethoscope

6) Special maneuvers

4) Pericardial rub

RESPIRATORY SYSTEM

ABDOMINAL EXAMINATION

NERVOUS SYSTEM EXAMINATION

INVESTIGATION

1)	Haemogram	TC	DC-N	LM	ESR
2)	Urine	Alb	sugar	ketone bodies	Microscopy
3)	Blood sugar	RBS	FBS	PPBS	
4)	Blood urea		Sr. Creatinine		
5)	Serum electrolytes		Sr. Sodium	Sr. potassium	
6)	Lipid profile		Sr. cholesterol,	Triglycerides	
7)	CPK – MB				
8)	ECG				
9)	ECHOCARDIOGRAM				

10) OTHERS

FINAL DIAGNOSIS

TREATMENT GIVEN

OUT COME

DISCUSSION

SIGNATURE OF GUIDE **SIGNTURE OF CANDIDATE**

KEY TO MASTER CHART

Occ	occupation
Farm	farmer
Hswf	house wife
Chp	chest pain
Rad	radiation
Pal	palpitation
Brt	breathlessness
Swe	sweating
V	vomting
C	cough
DM	diabetes mellitus
HTN	hypertension
IHD	ishcaemic heart disease
MPU	menopause
DT	Diet
S	Smoking
A	Alcohol
CT	Chewing tobacco

AWMI Anterior wall myocardial infarction

IWMI Inferior wall myocardial infarction

LWMI Lateral wall myocardial infarction

SNMI Non ST elevation myocardial infarction

EAWMI Extensive Anterior Wall Myocardial infrction

UA Unstable Anigina

HB1 First Degree Heart Block

HB2 Second Degree Heart Block

MR Mitral regurgitation

CHB Complete Heart Block

LAHB Left anterior hemi block

RBBB Right bundle branch block

LBBB Left bundle branch block

VT Ventricular tachycardia

VPC'S Ventricular premature complex

I Improved

D Death

S.NO	NAME	AGE	M/F	OCCU	CHP	RAD	PAL	BRT	SWE	OTH		PASTHISTORY				MPU	ERSONS HiSTOR				FAMILY HISTORY				BMI	PR	RY	BP	RR	JVP	RBS	CH	HDL	TG	LDL	VLDL	CPKMB	TROP-T	LVEE	WMA	D/A	LVF	OUT
										V	C	DM	HTN	IHD	HC		DT	HABITS			DM	HTN	IHD	HCL																		
																		S	A	CT																						-	
1	BHIMARAYA	58	M	FARM	+	-	-	-	+	-	-	-	-	-	-	NA	V	+	-	+	-	-	-	-	26	98	R	111/80	30	N	102	200	30	100	100	20	110	+	45	+	AWMI	-	I
2	RAMESH	54	M	FARM	+	+	+	+	+	-	+	-	+	-	-	NA	NV	-	-	+	-	-	-	-	38	90	IR	140/90	28	R	184	201	33	160	120	22	100	ND	ND	ND	AWMI	LVF	D
3	ARJUN CHANDAR	40	M	FARM	+	-	-	-	+	-	-	+	-	-	-	NA	V	-	-	-	-	-	-	-	25	110	R	126/78	27	N	210	231	32	134	119	32	96	+	46	+	IWMI	-	I
4	MAHADEVAPPA	64	M	FARM	+	+	-	-	+	-	-	-	-	-	+	NA	NV	+	+	+	+	-	-	-	35	180	IR	100/64	26	N	126	330	44	170	130	33	210	+	ND	ND	IWMI	VF	D
5	REVAPPA RATHOD	64	M	FARM	+	+	-	-	+	-	-	-	-	-	+	NA	NV	-	-	-	-	+	-	-	30	80	R	116/84	26	N	108	300	15	200	112	40	148	+	40	+	AWMI	-	I
6	BASHEER	60	M	TAIL	+	+	-	+	+	-	-	-	-	-	+	NA	NV	-	-	-	-	-	-	-	32	90	R	124/68	32	N	94	259	20	160	122	40	80	+	35	+	IWMI	VPC'S	I
7	BAGAWWA	52	F	HSWF	+	+	-	-	+	-	-	-	-	-	-	Y	V	-	-	-	-	-	-	-	38	94	R	144/92	30	N	76	150	55	78	90	40	111	+	34	+	AWMI	-	I
8	NARSAPPA	57	M	SHKR	+	+	-	-	+	-	-	-	+	-	-	NA	V	-	-	-	-	+	-	+	36	100	R	160/98	30	N	123	168	12	90	98	23	98	+	45	+	ASMI	LBBB	I
9	SHANTABAI	70	F	HSWF	+	+	-	-	+	-	-	-	-	-	-	Y	V	-	-	-	-	-	-	-	32	98	R	142/88	25	N	110	190	23	100	90	16	104	+	ND	ND	ASMI	VPC'S	I
10	MADHUKANNA	40	M	BUSI	+	+	-	-	+	-	-	-	-	-	+	NA	NV	+	+	+	-	-	-	-	35	112	R	126/84	26	N	121	290	14	160	100	18	116	+	58	+	ASMI	-	I
11	MEERABAI	60	F	HSWF	+	+	-	+	+	-	-	-	+	-	+	Y	NV	-	-	-	-	-	-	-	34	86	R	168/98	27	N	126	260	24	165	113	44	68	+	45	ND	ASMI	-	I
12	LAKAPPA	65	M	FARM	+	+	+	+	+	-	-	+	+	-	+	NA	NV	+	+	+	-	-	-	-	30	200	R	156/90	24	N	260	301	32	177	124	34	90	+	ND	ND	IWMI	VT	D
13	SHANTABAI	72	F	HSWF	-	-	+	+	+	-	-	+	-	+	+	Y	V	-	-	-	-	-	-	-	28	188	R	144/78	23	N	156	288	33	165	132	50	68	+	ND	ND	IWMI	VT	D
14	SANGAWWA	58	F	HSWF	+	-	-	-	+	+	-	+	-	+	-	Y	V	-	-	-	-	-	-	-	33	90	R	124/68	25	N	301	190	44	100	91	19	86	+	45	+	AWMI	-	I
15	BHIMAPPA PATI	55	M	FARM	+	+	-	-	+	+	-	-	+	-	+	NA	NV	-	-	-	-	-	-	-	38	68	R	160/98	26	N	160	255	30	177	100	20	102	+	42	+	AWMI	-	I
16	SHARANAPPA KSRY	65	M	FARM	+	+	-	+	-	+	-	-	-	-	+	NA	NV	+	-	-	+	-	-	-	30	84	R	60/44	27	R	140	317	44	165	132	43	45	+	25	+	IWMI	CSK	D
17	NEELAWA	54	F	HSWF	+	-	-	+	+	+	-	-	+	-	+	Y	V	-	-	-	-	+	-	-	37	87	R	178/98	29	N	120	301	33	156	124	34	58	+	ND	ND	AWMI	-	I
18	RAFIQ AHMED	62	M	RETD	+	-	-	+	-	+	-	-	-	+	-	NA	NV	+	-	-	-	-	+	-	22	100	R	124/58	31	R	80	200	41	80	109	30	48	+	25	+	AWMI	VPC'S	I
19	GURAPA	80	M	FARM	-	-	-	+	-	+	-	-	-	-	-	NA	V	+	-	-	-	-	-	-	25	68	R	132/78	33	R	100	230	32	90	102	33	124	+	35	+	AWMI	-	I
20	AMEENSAB	60	M	BUSI	+	-	-	-	+	+	-	-	-	-	-	NA	NV	-	-	-	-	-	-	-	30	78	R	112/68	30	N	146	25	33	120	120	24	24	+	34	+	UA	-	I
21	NAZEER AHMED	55	M	BUSI	+	-	-	-	-	-	-	-	-	-	-	NA	NV	-	-	-	-	-	-	-	28	90	R	110/78	23	N	102	234	21	132	109	41	12	ND	ND	ND	UA	-	I
22	LAXAPPA	60	M	FARM	+	-	-	-	+	-	-	+	-	-	-	NA	V	-	+	-	-	-	-	-	32	78	R	144/90	24	N	243	178	23	144	102	20	54	ND	ND	ND	LWMI	LBBB	I
23	GODAVARI	80	F	HSWF	+	+	+	+	+	+	-	-	-	-	-	Y	V	-	-	-	-	-	-	-	38	110	IR	70/40	25	N	115	209	25	112	90	33	324	ND	ND	ND	IWMI	CSK	D
24	NINGANA GOUDA	80	M	FARM	+	+	+	+	+	+	-	+	-	+	-	NA	V	-	-	-	-	-	-	-	28	120	IR	66/40	26	N	324	250	28	90	98	21	120	ND	ND	ND	AWMI	CSK	D
25	BASSANA GOUDA	40	M	FARM	-	+	-	-	-	+	-	+	-	-	+	NA	NV	-	-	-	-	-	-	-	30	102	R	110/78	27	N	202	250	30	100	98	20	132	+	45	+	LWMI	-	I
26	LALABAI	65	F	HSWF	-	-	-	+	+	+	+	+	-	-	-	Y	NV	-	-	-	-	-	-	-	30	102	R	100/70	28	R	280	266	29	88	100	12	90	+	38	+	IWMI	LVF	D
27	ABDUL AHMED	57	M	BUSI	-	+	-	-	+	-	-	+	-	-	-	NA	NV	-	-	-	-	-	-	-	26	100	R	134/68	30	R	340	254	30	90	109	14	120	+	45	+	UA	-	I
28	LALABAI	65	F	HSWF	-	-	-	+	+	+	-	-	-	-	-	Y	NV	-	-	-	-	-	-	-	30	68	R	124/86	32	R	128	213	40	98	103	16	104	+	50	+	UA	VPC'S	
29	SHREESHAIL PATIL	52	M	BUSI	+	+	-	-	-	-	-	-	+	-	+	NA	NV	-	-	+	-	-	+	-	40	58	R	186/98	34	R	89	255	55	177	111	15	86	ND	ND	ND	LWMI	LBBB	I
30	LAYAWWA	70	F	HSWF	+	+	-	-	+	+	+	-	-	-	+	Y	V	-	-	-	-	-	-	-	38	100	R	60/40	33	R	230	265	42	166	123	23	140	+	ND	ND	IWMI	CSK	D
31	BASU	38	M	BUSI	+	+	-	-	-	+	-	+	+	-	+	NA	NV	+	+	+	-	-	-	-	30	98	R	154/92	31	R	200	277	54	189	132	35	240	+	38	+	IWMI	-	I
32	MAHADEVAPPA	64	M	FARM	+	+	-	+	+	+	-	-	+	+	+	NA	V	+	+	+	-	-	-	-	35	100	R	180/90	32	R	230	289	46	190	120	43	120	+	ND	ND	IWMI	LVF	D
33	MAYAPPA	65	M	WCM	+	+	+	+	+	+	-	-	-	+	+	NA	NV	+	-	-	-	-	+	+	38	104	IR	60/40	30	N	120	301	48	166	100	32	111	+	ND	ND	AWMI	CSK	D
34	BHIMARAYA	45	M	FARM	+	-	-	-	+	-	-	-	-	-	-	NA	NV	+	+	+	+	+	+	-	36	100	R	144/90	30	N	98	170	23	100	100	13	104	+	43	+	LWMI	-	I
35	VITTAL SHRNAPPA	38	M	FARM	+	+	-	+	+	+	-	-	-	-	-	NA	NV	+	+	+	-	-	+	+	34	102	R	132/90	30	N	105	167	19	98	120	20	156	+	39	+	LWMI	-	I
36	SHANTWWA	50	F	HSWF	-	-	-	+	-	+	-	+	+	-	+	Y	NV	-	-	-	-	-	-	-	34	100	R	160/90	32	N	200	256	32	166	105	25	111	+	28	+	IWMI	-	I
37	NINDAPPA	65	M	BUSI	+	+	-	-	-	-	+	+	-	-	-	NA	V	-	-	+	-	-	-	-	53	89	R	140/90	34	N	90	188	22	78	101	32	176	ND	ND	ND	UA	-	I
38	MALLAM	45	F	HSWF	+	-	-	-	+	-	+	+	+	-	-	NO	V	-	-	-	+	-	-	-	40	90	R	160/90	36	N	100	198	32	99	120	34	302	+	40	+	UA	LBBB	I
39	BASVANTARAYA	65	M	FARM	+	-	-	-	+	-	+	-	-	-	-	NA	V	-	-	-	-	-	-	-	40	98	R	124/80	32	N	88	212	44	89	97	45	105	+	44	+	UA	-	I
40	MAHADEVAPPA	70	M	FARM	+	+	-	+	+	-	+	+	+	-	-	NA	V	-	+	+	-	-	+	+	36	100	IR	188/98	31	R	244	213	45	90	90	44	100	ND	ND	ND	AWMI	LVF	D
41	VITTAPA	48	M	FARM	-	-	-	-	+	-	-	+	-	-	-	NA	V	-	-	-	+	-	-	-	24	90	R	112/82	30	R	210	222	43	123	96	42	400	+	44	+	IWMI	-	I
42	UMADEVI	64	F	HSWF	+	+	-	-	+	+	-	-	+	-	-	Y	V	-	-	-	-	-	-	-	30	100	R	98/64	23	N	99	231	32	111	99	43	110	+	24	+	AWMI	-	I
43	SAHARA	69	F	HSWF	+	+	-	-	+	+	-	-	-	+	-	Y	NV	-	-	-	-	-	-	-	28	110	R	100/88	24	N	102	198	44	100	91	12	88	+	42	+	AWMI	-	I
44	ASHRAFBEE	64	F	HSWF	+	+	-	-	+	+	-	-	-	+	-	Y	NV	-	-	-	-	-	-	-	22	88	R	112/66	25	N	142	189	40	109	103	10	60	+	34	+	LWMI	-	I
45	RAJASAB	45	M	BUSI	+	+	-	-	+	-	-	+	-	-	-	NA	NV	+	+	+	+	-	-	-	34	88	R	124/88	26	N	200	170	40	120	108	19	98	+	44	+	AWMI	-	I
46	BASVAARJ	47	M	BUSI	+	+	-	-	+	-	-	+	-	-	-	NA	NV	+	+	+	+	-	-	-	34	90	R	120/80	27	N	260	166	43	105	100	18	102	+	42	+	IWMI	VPC'S	I
47	ANIL BIRADA	48	M	BUSI	+	+	-	-	+	-	-	-	-	-	+	NA	NV	+	+	+	-	-	-	+	28	88	R	110/80	21	N	110	280	30	170	120	45	90	+	46	+	AWMI	-	I
48	SHAKUNTALA	71	F	HSWF	+	+	-	-	-	+	-	-	-	-	+	Y	NV	-	-	-	-	-	-	-	29	100	R	112/78	24	N	100	290	44	178	133	44	100	+	44	+	IWMI	-	I
49	SHARANAPPA	71	M	FARM	+	+	-	-	-	+	-	-	-	-	+	NA	V	-	-	-	-	-	-	-	24	88	R	94/60	15	N	98	345	45	189	134	56	22	+	34	+	UA	-	I
50	SHIVAMMA	54	F	HSWF	+	+	-	-	-	-	-	+	-	-	+	Y	V	-	-	-	-	-	-	-	26	86	R	40/20	16	N	201	300	55	177	143	51	245	+	32	+	IWMI	CSK	D
51	RAJASAB	74	M	FARM	+	+	-	-	-	-	-	-	-	-	-	NA	V	+	+	+	-	-	-	-	30	88	R	112/80	17	N	100	212	34	100	99	55	112	+	ND	ND	AWMI	-	I
52	SAIRABEE	78	F	HSWF	-	-	-	-	-	+	-	-	+	-	-	Y	NV	-	-	-	-	+	-	-	21	68	R	190/98	19	R	68	200	32	90	98	18	88	+	ND	ND	LWMI	VPC'S	I
53	SHIVANAND	40	M	BUSI	+	+	-	-	+	-	-	-	-	-	+	NA	NV	+	+	+	-	-	-	+	32	100	R	112/68	14	N	116	302	32	145	120	43	26	+	ND	ND	UA	LBBB	I
54	ABDUL KAREEM	50	M	BUSI	+	+	-	-	+	-	-	-	-	-	+	NA	NV	+	+	+	-	-	-	+	30	86	R	116/82	16	N	121	288	23	155	100	44	31	+	ND	ND	UA	VT	I
55	SURABEE	65	F	HSWF	-	-	-	+	-	+	+	+	+	-	-	Y	V	-	-	-	-				32	90	R	164/90	18	R	301	213	31	120	98	18	119	+	ND	ND	IWMI	LVF	D
56	HUSENBEE	70	F	HSWF	+	-	-	-	-	-	-	-	+	-	+	Y	NV	-	-	-	-	-	-	-	18	100	R	188/98	19	R	107	290	19	150	109	40	68	+	32	+	IWMI	-	I
57	CHANDRAKALA	48	F	HSWF	-	-	-	-	+	-	-	+	+	-	-	Y	V	-	-	-	+	-	-	-	30	100	R	134/76	17	R	290	243	33	97	90	19	100	+	52	+	AWMI	-	I
58	SHIVARAYA	50	M	FARM	+	+	-	-	+	-	-	-	-	-	+	NA	V	+	+	+	-	-	-	+	28	80	R	98/60	14	R	108	320	44	160	109	50	124	+	46	+	IWMI	-	I
59	AMANSIDDA	40	M	FARM	+	+	-	-	+	-	-	-	-	-	+	NA	V	+	+	+	-	-	-	+	26	90	R	100/80	20	R	111	312	56	177	111	51	200	+	44	+	IWMI	VPC'S	I
60	ANASUYA	45	F	HSWF	+	-	-	-	-	-	-	+	-	-	-	Y	V	-	-	-	+	-	-	-	32	88	R	128/76	21	R	301	198	30	70	102	20	230	+	48	+	AWMI	-	I
61	APPASAB	45	M	FARM	+	+	-	-	+	+	+	-	-	-	-	NA	V	+	+	+	-	-	-	-	26	100	R	100/84	22	N	99	180	40	88	100	33	124	+	41		UA	-	I
62	MUCHAPPA	52	M	FARM	+	+	-	+	+	+	-	-	-	-	-	NA	V	+	+	+	-	-	-	-	22	98	R	76/34	20	N	143	200	43	90	99	44	100	+	ND	ND	IWMI	CSK	D
63	DHANUSAAB	40	M	BUSI	+	+	-	-	+	+	-	-	-	-	-	NA	V	+	+	+	-	-	-	-	27	84	R	98/60	16	N	109	213	42	100	98	32	111	+	ND	ND	LWMI	VPC'S	I
64	DEVUDU	45	M	BUSI	+	+	-	-	+	+	-	-	-	-	-	NA	V	+	+	+	-	-	-	-	19	78	R	126/86	18	N	89	200	41	104	109	43	160	+	ND	ND	LWMI	-	I
65	YALLAWAA	50	F	HSWF	+	-	-	-	-	-	-	+	-	-	+	Y	V	-	-	-	+	-	-	+	30	100	R	144/90	19	R	289	156	32	120	120	20	139	+	+	ND	IWMI	LVF	D
66	REKHA	48	F	HSWF	+	-	-	-	-	-	-	-	-	-	-	Y	V	-	-	-	-	-	-	-	19	112	R	110/82	20	N	98	178	32	96	130	22	16	+	+	+	UA	-	I
67	SHIV SHANKAR	48	M	BUSI	+	+	-	-	+	-	-	-	-	-	+	NA	V	-	-	-	+	-	-	-	32	86	R	110/68	21	R	110	180	34	79	129	23	100	+	+	+	AWMI	-	I
68	HAJISAB	65	M	FARM	+	+	-	+	-	+	-	-	-	+	-	NA	NV	-	-	+	-	-	-	-	31	86	R	114/76	20	R	100	197	19	88	116	24	112	+	+	+	AWMI	-	I
69	KANTAMMA	55	F	HSWF	+	+	-	-	-	+	-	-	-	-	-	Y	V	-	-	-	-	-	-	-	20	66	R	110/78	21	R	107	159	40	120	117	12	23	ND	ND	ND	UA	VPC'S	I
70	BASAMMA	48	F	HSWF	+	+	-	-	+	-	-	-	-	-	-	Y	V	-	-	-	-	-	-	-	21	88	R	98/76	20	R	122	201	45	103	109	14	22	ND	ND	ND	UA	VT	I
71	BASALINGAPPA	50	M	FARM	+	+	-	-	+	-	-	-	-	-	+	NA	V	-	-	-	+	-	-	-	32	98	R	124/80	20	R	100	231	46	122	98	23	32	+	+	50	UA	-	I
72	BABULAL	42	M	BUSI	+	+	-	-	+	-	-	-	-	-	+	NA	NV	-	-	-	+	-	-	-	33	78	R	120/78	18	R	98	244	19	88	99	25	98	+	+	48	AWMI	-	I
73	SHIVAPPA	42	M	BUSI	+	+	-	-	+	-	-	-	+	-	-	NA	NV	-	-	-	-	-	-	-	19	98	R	166/90	19	R	100	198	20	90	90	34	210	+	+	32	AWMI	-	I
74	SHANTIWWA	51	F	HSWF	+	+	-	-	-	-	-	-	+	-	-	Y	V	-	-	-	-	-	-	-	21	100	R	156/88	20	R	99	200	32	78	100	35	109	+	+	34	IWMI	-	I
75	VEERESH	32	M	BUSI	+	+	-	-	+	-	-	+	+	-	-	NA	NV	-	-	-	-	-	-	-	20	108	R	170/98	20	R	230	212	20	76	109	32	222	+	+	42	IWMI		I
76	KASHIRAYA	45	M	BUSI	+	+	-	+	+	-	+	-	-	-	+	NA	V	-	-	+	+	-	-	-	36	190	R	132/90	22	R	110	231	30	100	125	33	300	+	ND	ND	AWMI	VT	D
77	SOMAPPA	60	M	FARM	+	+	-	-	+	+	-	-	-	+		NA	V	-	-	-	-	-	-	-	21	100	R	112/80	21	N	98	231	40	109	117	32	100	+	44	+	AWMI	-	I

78	HANAMATH	80	M	FARM	-	-	-	+	-	-	+	+	+	-	-	NA	V	-	-	+	-	-	-	-	32	90	R	180/90	24	R	300	156	32	99	129	31	290	+	ND	ND	AWMI	LVF	D
79	RUDRAGOUDA	50	M	FARM	+	+	-	-	+	+	-	-	-	-	+	NA	V	+	+	+	-	-	-	-	19	98	R	110/80	18	N	120	165	41	78	118	35	34	+	34	+	UA	-	I
80	RUPANNA	55	M	FARM	+	+	-	-	+	-	-	-	-	-	+	NA	V	+	+	+	-	-	-	-	22	88	R	112/80	17	N	109	175	26	76	100	43	44	+	36	+	UA	-	I
81	SUMITHA	53	F	HSWF	+	-	-	-	-	-	-	-	-	-	+	Y	V	-	-	-	-	-	-	-	23	86	R	110/84	19	N	111	189	38	100	98	4240	124	+	44	+	IWMI	VPC'S	I
82	MADANI	64	M	FARM	+	+	-	-	+	+	-	-	-	+	-	NA	V	-	-	-	-	-	-	-	22	68	R	116/68	21	N	108	198	39	69	99	19	421	+	28	+	IWMI	VPC'S	I
83	NABISAB	60	M	FARM	+	+	-	+	-	+	-	-	-	+	-	NA	NV	-	-	-	-	-	-	-	25	84	R	112/66	22	N	98	189	30	100	97	18	324	+	32	+	AWMI	-	I
84	SHANTAMMA	71	F	HSWF	-	-	-	-	+	+	-	-	-	+	-	Y	V	-	-	-	-	-	-	-	21	66	R	120/78	20	N	97	179	31	105	92	19	189	+	ND	ND	LWMI	-	I
85	SUREKHA	51	F	HSWF	+	+	-	-	-	-	-	-	-	+	-	Y	NV	-	-	-	-	-	-	-	23	108	R	100/68	24	N	123	165	24	125	109	12	198	+	ND	ND	IWMI	-	I
86	SALEEM	45	M	BUSI	+	+	-	-	+	-	-	+	-	-	+	NA	NV	-	-	-	-	-	-	-	21	120	R	132/80	25	N	209	190	25	135	100	13	56	+	41	+	UA	-	I
87	SUKRASAB	68	M	FARM	+	+	-	-	+	+	-	-	+	-	-	NA	V	-	-	-	-	-	-	-	19	112	R	160/90	209	N	124	211	29	128	98	15	76	+	45	+	UA	-	I
88	APPANAA	70	M	FARM	-	-	-	+	-	+	-	-	+	-	-	NA	V	-	-	-	-	-	-	-	18	112	R	156/98	19	N	132	210	31	101	90	19	66	+	46	+	UA	-	I
89	KALAPPA	48	M	BUSI	+	+	-	-	+	-	-	-	-	-	-	NA	NV	+	+	+	-	-	-	-	20	102	R	98/60	21	N	142	208	35	109	92	23	100	+	47	+	IWMI	-	I
90	ANSUYA	62	F	HSWF	+	+	-	-	-	-	-	-	-	+	-	Y	V	-	-	-	-	-	-	-	30	100	R	100/80	20	N	108	241	34	121	100	25	231	+	35	+	AWMI	VT	I
91	SUKANYA	64	F	HSWF	+	+	-	-	-	+	+	-	-	-	-	Y	V	-	-	-	-	-	-	-	21	98	R	120/88	18	N	301	233	43	141	109	29	200	+	32	+	LWMI	-	I
92	BABAJI CHAVAN	75	M	FARM	+	+	-	+	-	+	-	+	+	-	-	NA	V	-	-	-	-	-	-	-	30	74	R	160/98	19	R	256	214	56	162	122	32	200	+	ND	ND	IWMI	LVF	D
93	CHARU SUKANYA	58	F	HSWF	+	-	-	-	+	-	-	-	-	-	+	Y	V	-	-	-	-	-	-	-	20	100	R	110/76	21	N	111	231	53	85	103	31	209	+	34	+	LWMI	-	I
94	SHILPA PAWAR	64	F	HSWF	+	+	-	-	+	-	-	-	+	-	-	Y	V	-	-	-	-	-	-	-	19	98	R	180/98	20	N	109	200	42	78	100	23	100	+	44	+	IWMI	-	I
95	SHEKAWWA	70	F	HSWF	-	-	-	-	-	+	-	-	+	-	-	Y	V	-	-	-	-	-	-	-	18	88	R	156/90	20	N	132	200	17	100	109	25	34	+	45	+	UA	-	I
96	JINABEE	68	F	HSWF	+	+	-	-	-	+	-	-	+	-	-	Y	V	-	-	-	-	-	-	-	23	86	R	156/98	19	N	123	212	33	123	102	22	65	+	34	+	UA	LBBB	I
97	RAJA	45	M	BUSI	+	+	-	-	+	-	-	+	-	-	+	NA	NV	+	+	+	-	-	-	-	24	66	R	132/84	21	N	400	209	22	123	120	23	54	+	41	+	UA	-	I
98	CHANAMMA	68	F	HSWF	+	+	-	-	-	+	-	-	-	-	-	Y	V	-	-	-	-	-	-	-	22	80	R	112/68	20	N	89	166	20	150	129	32	12	+	40	+	UA	VPC'S	I
99	RANI BIRADAR	74	F	HSWF	+	+	-	+	+	-	-	-	+	-	-	Y	V	-	-	-	-	-	-	-	21	90	R	102/68	24	N	100	188	31	98	133	34	100	+	44	+	IWMI	-	I
100	NANDABASAPPA	55	M	FARM	+	+	-	-	-	-	-	-	-	-	-	NA	V	-	-	-	-	-	-	-	17	82	R	116/78	23	N	400	190	29	99	122	35	231	+	29	+	IWMI	-	I
102	RADHABAI	62	F	HSWF	+	+	-	+	-	-	+	+	-	+	-	Y	V	-	-	-	-	-	-	-	28	86	R	170/90	25	N	301	199	44	100	109	10	124	+	ND	ND	IWMI	LVF	D
103	SANGABASSAVA	55	F	HSWF	+	+	-	+	-	-	+	+	-	-	-	Y	V	-	-	-	-	-	-	-	30	84	R	168/96	26	N	456	187	40	99	115	19	144	+	ND	ND	IWMI	LVF	D
104	YALAWWA	80	F	HSWF	-	-	-	-	-	+	+	-	-	-	-	Y	V	-	-	-	-	-	-	-	22	100	r	112/76	21	N	10	155	43	87	110	18	32	+	ND	ND	UA	-	I
105	GURUSIDDAPA	68	M	FARM	+	+	-	-	-	+	+	+	-	-	-	NA	V	-	-	-	-	-	-	-	19	98	r	110/80	19	N	112	167	41	67	102	21	44	+	ND	ND	UA	-	I
106	SUVARNA	60	F	HSWF	+	+	-	-	-	-	-	-	+	-	-	Y	V	-	-	-	-	-	-	-	30	90	R	180/90	18	R	124	188	32	69	120	20	110	+	44	+	AWMI	-	I
107	FATIMA BEE	70	F	HSWF	+	+	-	-	-	+	-	-	+	-	-	Y	NV	-	-	-	-	-	-	-	34	88	R	190/98	22	R	142	190	21	69	98	21	144	+	38	+	AWMI	-	I
108	SUNDARAWA	80	F	HSWF	+	+	-	+	-	-	-	-	+	-	-	Y	V	-	-	-	-	-	+	-	32	98	R	166/88	20	R	98	200	24	121	90	23	176	+	25	+	LWMI	-	I
109	GURUSIDAWWA	79	M	FARM	-	-	-	-	+	+	+	+	-	-	-	NA	V	-	-	-	-	-	-	-	18	88	r	100/82	21	N	102	209	44	132	97	22	23	+	ND	ND	UA	-	I
110	APPASAB	72	M	FARM	+	+	-	-	+	+	+	+	-	-	-	NA	V	-	-	-	-	-	-	-	18	90	r	98/80	20	N	122	212	32	140	92	33	14	+	ND	ND	UA	-	I
111	RAMANNA	45	M	BUSI	+	+	-	-	+	-	+	-	-	-	+	NA	V	+	+	+	-	-	-	+	26	100	R	120/80	19	N	124	222	30	165	90	14	156	+	42	+	AWMI	VPC'S	I
112	KANTAMMA	63	F	HSWF	+	+	-	-	-	+	+	-	-	-	-	Y	V	-	-	-	-	-	-	-	19	90	R	112/80	18	N	111	203	39	170	100	19	23	ND	ND	ND	UA	VT	I
113	BASAPPA	80	M	FARM	-	-	-	+	-	+	-	-	-	-	-	NA	V	+	+	+	-	-	-	+	30	100	R	130/88	20	N	100	188	29	92	119	20	121	+	34	+	IWMI	-	I
114	PARASAPPA	74	M	FARM	+	+	-	-	-	+	-	-	-	-	-	NA	V	-	-	-	-	-	-	-	20	98	R	110/80	21	N	100	190	28	90	121	25	100	+	32	+	AWMI	-	I
115	CHANDAN	62	M	FARM	+	+	-	-	+	+	+	-	-	-	-	NA	V	-	-	-	-	-	-	-	20	90	R	120/78	20	N	90	155	39	81	130	44	32	+	28	+	UA	-	I
116	CHANDRAMMA	80	F	HSWF	-	-	-	+	-	+	-	-	-	-	-	Y	V	-	-	-	-	-	-	-	19	88	R	112/68	17	N	80	189	43	79	100	41	132	+	33	+	LWMI	VPC'S	I
117	DARAPPA	58	M	FARM	+	+	-	-	-	-	-	-	-	-	-	NA	V	+	+	+	-	-	-	-	18	100	R	110/80	20	N	70	200	48	87	98	40	141	+	32	+	LWMI	-	I
118	BASAMMA	66	F	HSWF	+	+	-	-	-	+	+	-	-	-	-	Y	V	-	-	-	-	-	-	-	20	104	R	116/78	16	N	78	209	49	60	90	18	33	+	34	+	UA	-	I
119	PARVATI	61	F	HSWF	+	+	-	-	+	-	-	-	-	-	-	Y	V	-	-	-	-	-	-	-	22	106	R	134/80	18	N	112	211	40	69	97	19	106	+	42	+	IWMI	LBBB	I
120	SHAKAWWA	61	F	HSWF	+	+	-	+	+	-	+	-	-	+	+	Y	V	-	-	-	-	-	-	-	38	100	R	140/78	21	R	100	189	42	121	120	15	201	+	ND	ND	IWMI	LVF	D
121	MALLIKURJAN	45	M	BUSI	+	+	-	-	+	-	+	-	-	-	-	NA	V	+	+	+	-	-	-	-	22	78	R	134/80	20	N	98	198	44	131	113	32	42	+	30	+	UA	-	I
122	HUSAIN SAB	60	M	FARM	+	+	-	-	-	+	-	-	-	-	-	NA	NV	-	-	-	-	-	-	-	23	88	R	120/80	20	N	88	165	38	120	114	37	100	+	29	+	AWMI	VPC'S	I
123	LAKSHMI	72	F	HSWF	-	-	-	+	-	+	-	-	-	+	-	Y	V	-	-	-	-	-	-	-	30	90	R	116/70	17	R	68	167	43	94	109	29	121	+	28	+	IWMI	-	I
124	GUJUBAI PAWAR	71	F	HSWF	+	+	-	+	-	-	+	-	-	-	+	Y	V	-	-	-	-	-	-	-	28	89	R	124/70	19	R	87	170	45	79	99	20	320	+	ND	ND	IWMI	LVF	D
125	BHIMAPPA RAO	70	M	FARM	+	+	-	+	+	-	-	-	+	-	-	NA	V	-	-	-	-	-	-	-	22	100	R	190/98	21	N	100	199	41	77	100	18	24	+	33	+	UA	-	I
126	BEENA BAI	70	F	HSWF	+	+	-	+	-	+	-	-	+	-	-	Y	NV	-	-	-	-	-	-	-	19	108	R	160/90	20	N	111	200	20	120	109	21	53	+	34	+	UA	-	I
127	MD MOINUDDIN	57	M	FARM	+	+	-	-	+	-	-	-	+	-	-	NA	V	+	+	+	-	-	-	-	18	88	R	166/88	22	N	123	210	21	111	108	27	123	+	41	+	LWMI	-	I
128	LAKSHMAN	59	M	FARM	+	+	-	-	+	-	-	-	+	-	-	NA	V	+	+	+	-	-	-	-	22	98	R	148/90	24	N	141	211	28	105	121	33	144	+	27	+	AWMI	-	I
129	MALLAWWA	64	F	HSWF	+	+	-	-	-	+	-	-	+	-	-	Y	V	-	-	-	-	-	-	-	20	122	R	170/98	25	N	107	190	27	81	115	22	231	+	31	+	LWMI	-	I
130	AKBAR MAHOOB	45	M	BUSI	+	+	-	-	+	-	-	-	-	-	+	NA	NV	-	-	-	-	-	-	-	25	110	R	110/80	26	N	99	266	45	156	110	40	100	+	30	+	IWMI	-	I
131	FARDINA	70	F	HSWF	-	-	-	+	-	-	+	-	-	-	+	Y	NV	+	+	+	-	-	-	-	34	200	R	124/67	21	N	143	278	44	176	132	46	100	+	ND	ND	IWM I	VT	D
132	RAVI	50	M	BUSI	+	+	-	-	+	-	-	+	+	-	-	NA	V	-	-	-	-	-	-	-	18	100	R	166/90	22	R	213	198	45	99	110	18	123	+	34	+	IWMI	-	I
133	PARASURAM	45	M	BUSI	+	+	-	-	+	-	-	+	+	-	-	NA	V	-	-	-	-	-	-	-	19	88	R	188/90	20	R	198	167	43	70	98	22	345	+	33	+	IWMI	-	I
134	SHANTA	67	F	HSWF	-	-	-	+	-	+	-	+	+	-	-	Y	V	-	-	-	-	-	-	-	20	98	R	156/88	19	N	321	155	42	85	110	24	412	+	31	+	IWMI	-	I
135	TARA BAI	61	F	HSWF	+	-	-	+	-	-	+	+	+	-	-	Y	V	-	-	-	-	-	-	-	32	88	R	156/98	16	R	302	200	23	67	98	33	204	+	ND	ND	IWMI	LVF	D
136	BHEEMARAYA	68	M	FARM	+	+	-	-	-	+	-	-	-	-		NA	V	+	-	-	-	-	-	-	21	98	R	120/80	18	N	98	210	12	67	99	41	100	+	32		AWMI	-	I
137	SHIVAMMA	64	F	HSWF	+	+	-	-	-	-	-	-	-	-	-	Y	V	-	-	-	-	-	-	-	23	100	R	112/68	22	N	100	222	26	90	90	20	34	+	43		UA	-	I
138	CHANDRASHEKAR	59	M	FARM	+	+	-	-	+	-	-	-	-	-	-	NA	V	+	+	+	-	-	-	-	25	112	R	100/82	20	N	111	188	20	92	95	29	154	+	27		LWMI	VPC'S	I
139	MARUTI	60	M	FARM	+	+	-	-	-	-	-	-	-	-	-	NA	V	+	+	+	-	-	-	-	25	140	R	98/60	20	N	121	156	28	69	121	19	162	+	ND	ND	LWMI	-	I
140	VIJAY KUMAR	41	M	BUSI	+	+	-	-	+	-		-	-	-	+	NA	NV	+	+	+	-	-	-	+	31	120	R	124/84	22	N	100	289	44	155	134	42	243	+	33		AWMI	-	I
141	CHANDRAKALA	70	F	HSWF	-	-	-	-	-	-	-	-	-	+	-	Y	V	-	-	-	-	-		-	32	98	R	100/76	24	N	99	178	44	100	110	18	312	+	44		LWMI	-	I
142	PANCHAYYAA	70	M	FARM	-	-	-	+	-	-	+	-	-	+	-	NA	V	-	-	-	-	-	-	-	22	88	R	112/76	20	N	108	178	43	120	98	32	32	+	31		UA	VT	I
143	SHARANAPPA	75	M	FARM	-	-	-	+	-	-	-	-	-	+	-	NA	V	-	-	-	-	-	-	-	19	86	R	112/72	19	N	132	200	41	143	99	33	109	+	28		IWMI	-	I
144	BASALINGAPPA	72	M	FARM	+	+	-	+	-	-	-	-	+	-	-	NA	V	-	-	-	-	-	-	-	18	80	R	160/98	16	N	141	211	27	120	90	35	184	+	45		IWMI	-	I
145	SUCHITRA	66	F	HSWF	+	+	-	+	+	+	-	-	+	-	+	Y	V	-	-	-	-	+	-	-	18	100	R	112/80	15	N	100	320	35	150	130	34	109	+	34	+	AWMI	LBBB	I
146	SAVITRA	65	F	HSWF	+	+	-	+	+	+	+	-	+	-	+	Y	V	-	-	-	-	+	-	-	23	102	R	98/80	18	N	123	313	34	152	133	44	246	+	36	+	AWMI	-	I
147	IBRAHIM	70	M	FARM	-	-	-	+	-	+	+	-	-	-	-	NA	NV	-	-	-	-	-	-	-	22	120	R	124/84	19	N	122	199	44	144	123	43	143	+	27	+	LWMI	-	I
148	KALAPPA	41	M	BUSI	+	+	-	-	+	-	-	-	-	-	-	NA	V	+	+	+	-	-	-	-	21	98	R	120/80	22	N	89	187	41	156	132	40	245	+	ND	ND	LWMI	-	I
149	CHANDRA	45	M	BUSI	+	+	-	-	+	-	-	-	-	-	-	NA	V	+	+	+	-	-	-	-	25	90	R	100/60	20	N	98	190	30	99	121	12	321	+	ND	ND	AWMI	-	I
150	DUNDAPPA	61	M	FARM	+	+	-	-	-	+	-	-	-	-	-	NA	V	-	-	-	-	-	-	-	19	94	R	140/90	23	N	108	200	39	90	109	18	453	+	26	+	IWMI	VPC'S	I
151	ALISAB	50	M	FARM	+	+	-	-	+	+	-	-	-	-	-	NA	NV	-	-	-	-	-	-	-	21	88	R	112/68	24	N	108	213	44	89	117	24	421	+	34	+	IWMI	-	I
152	RAMANA GOUDE	72	M	FARM	-	-	-	+	-	+	-	-	-	-	-	NA	V	-	-	-	-	-	-	-	24	100	R	100/84	22	N	129	223	31	83	118	21	123	+	43	+	AWMI	-	I
153	SATU	80	M	FARM	-	-	-	+	-	+	+	-	-	-	-	NA	V	-	-	-	-	-	-	-	17	92	R	132/86	19	N	132	241	44	109	98	18	342	+	41	+	AWMI	-	I
154	SAKET	80	M	FARM	-	-	-	+	-	+	+	-	-	-	-	NA	V	-	-	-	-	-	-	-	19	88	R	100/68	18	N	116	213	46	108	100	42	24	+	27	+	UA	-	I
155	NIJALANGAPPA	62	M	FARM	+	+	-	-	-	-	-	-	-	-	-	NA	V	-	-	-	-	-	-	-	23	106	R	140/80	20	N	129	212	48	121	112	30	28	+	44	+	UA	-	I
156	RAMALINGAPPA	60	M	FARM	+	-	-	-	+	-	-	-	-	-	-	NA	V	-	-	-	-	-	-	-	26	100	R	120/84	22	N	101	190	49	143	113	31	101	+	45	+	LWMI	VPC'S	I
157	KASHIMSHAB	70	M	FARM	+	-	-	+	-	+	-	-	-	-	-	NA	NV	-	-	-	-	-	-	-	31	80	R	102/78	24	N	120	179	50	109	109	34	222	+	36	+	LWMI	VT	I
158	BHIMAPPA	80	M	FARM	+	-	-	+	-	-	-	-	-	-	-	NA	V	-	-	-	-	-	-	-	32	86	R	120/84	22	N	99	180	52	100	121	33	312	+	37	+	AWMI	-	I

159	SHIVANAND	49	M	FARM	+	+	-	-	+	-	-	-	-	-	-	NA	V	-	-	-	-	-	-	-	33	82	R	120/80	20	N	100	199	43	98	100	18	109	+	30	+	AWMI	-	I
160	SUKRANA	50	M	FARM	+	+	-	-	+	-	-	-	-	-	-	NA	V	-	-	-	-	-	-	-	19	80	R	116/82	19	N	109	167	42	90	90	21	44	+	33	+	UA	-	I
161	MD KHAN	50	M	FARM	+	-	-	-	+	-	-	-	-	-	-	NA	NV	-	-	-	-	-	-	-	21	100	R	108/68	16	N	111	197	44	93	99	20	100	+	44	+	IWMI	-	I
162	LAKSHMAN	50	M	FARM	+	-	-	-	+	-	-	-	-	-	-	NA	V	-	-	-	-	-	-	-	20	126	R	112/68	18	N	98	210	49	100	98	39	98	+	28	+	IWMI	-	I
163	LAKKU	47	M	FARM	+	+	-	-	-	-	-	+	-	-	-	NA	NV	-	-	+	+	-	-	-	28	88	R	144/80	19	N	300	201	50	120	97	32	100	+	44	+	IWMI	-	I
164	SURENDER	48	M	FARM	+	+	-	-	+	-	-	+	-	-	-	NA	V	-	-	+	+	-	-	-	24	78	R	122/84	19	N	280	213	51	100	96	31	109	+	38	+	AWMI	VPC'S	I
165	SIDDNA GOUDA	42	M	FARM	+	+	-	-	+	+	+	-	-	-	+	NA	V	+	+	+	-	-	+	-	33	90	R	116/80	21	N	110	300	33	166	100	41	34	+	ND	ND	UA	-	I
166	GARUDAPAA	48	M	FARM	+	+	-	-	+	+	-	-	-	-	+	NA	V	+	+	+	-	-	+	-	31	68	R	110/78	22	N	98	287	44	182	130	42	26	+	ND	ND	UA	-	I
167	RAJA SHARMA	48	M	FARM	+	+	-	-	+	-	-	-	-	-	+	NA	V	+	+	+	-	-	+	-	32	68	R	106/82	20	N	99	267	31	180	133	44	44	+	ND	ND	UA	-	I
168	SRIDHAR BABU	50	M	FARM	+	+	-	-	+	+	-	-	-	-	+	NA	V	+	+	+	-	-	-	+	28	90	R	110/68	22	N	123	273	25	170	132	34	201	+	36	+	AWMI	-	I
169	RAVINDER REDDY	60	M	FARM	-	-	-	+	-	+	-	-	+	+	-	NA	V	-	-	-	-	-		-	22	78	R	180/90	20	R	98	200	32	100	100	33	201	+	29	+	AWMI	LBBB	I
170	YALLAPPA	80	M	FARM	+	+	-	-	-	-	-	-	+	+	-	NA	V	-	-	-	-	-	-	-	23	78	R	168/98	16	R	120	210	36	120	98	23	234	+	35	+	AWMI	VPC'S	I
171	SHIVRAYA	77	M	FARM	+	+	-	-	-	-	-	-	+	-	-	NA	V	-	-	-	-	-	-	-	26	108	R	166/88	19	N	112	201	46	90	99	21	400	+	29	+	IWMI	-	I
172	SWAMY	67	M	FARM	+	+	-	+	+	+	+	-	-	-	-	NA	V	-	-	-	-	-	-	-	21	112	R	104/76	18	N	136	198	43	99	90	25	205	+	32	+	LWMI	-	I
173	MUTKAPPA	62	M	FARM	+	+	-	+	-	-	-	-	+	-	-	NA	V	-	-	-	-	-	-	-	19	120	R	164/88	20	R	132	188	31	96	98	20	186	+	33	+	IWMI	-	I
174	SHIVRAYA	70	M	FARM	+	+	-	-	-	-	-		-	+	-	NA	V	-	-	-	-	-	-	-	20	98	R	108/64	17	N	109	209	32	65	100	18	94	+	32	+	LWMI	-	I
175	SHANTABAI	70	F	HSWF	-	-	-	-	+	-	-	+	-	-	+	Y	V	-	-	-	-	-	-	-	28	100	R	112/68	19	N	402	312	26	166	133	19	41	+	32	+	IWMI	CSK	D
176	SHIVAJI	68	M	FARM	+	+	-	-	+	-	-	+	-	-	-	NA	V	-	-	+	-	-	-	-	30	80	R	128/88	22	N	222	211	33	78	98	32	32	+	44	+	UA	-	I
177	S K RAO	68	M	FARM	+	+	-	-	-	-	-	+	-	-	-	NA	V	-	-	+	-	-	-	-	26	90	R	60/40	20	N	301	222	37	90	99	27	30	+	45	+	UA	VPC'S	I
178	RAYAPPA	70	M	FARM	+	+	-	-	+	-	-	-	+	-	-	NA	V	-	+	+	-	-	-	-	24	78	R	158/90	20	N	144	231	39	92	121	29	100	+	44	+	IWMI	LVF	I
179	K.HANJAGI	68	M	FARM	+	+	-	-	-	-	-	-	+	-	-	NA	V	-	+	+	-	-	-	-	23	88	R	148/90	19	N	124	198	42	100	113	18	140	+	28	+	AWMI	LVF	I
180	SHIVANNA	60	M	FARM	+	+	-	+	-	+	+	-	+	-	-	NA	V	+	+	+	-	-	-	-	24	68	R	168/98	24	N	119	212	31	89	109	20	112	+	26	+	LWMI	LVF	I