

Risk Factors of Cardiovascular Diseases: Study of Lipid Profile in Patients With Myocardial Infarction



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Risk factors of cardiovascular diseases: Study of lipid profile in patients with myocardial infarction

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Dedicated to My Parents and Teachers

CERTIFICATE

This is to certify that, the thesis Risk factors of cardiovascular disease: study of lipid profile in patients with myocardial infarction submitted to the Department of pharmacy, East West University, Mohakhali, Dhaka for the partial fulfill of the requirements for the degree of Bachelor of pharmacy (B.Pharm) was carried out by Abu Sayeed Khan (ID: 2006-1-70-041) under our guidance and supervision and that no part of the thesis has been submitted for any other degree. We further certify that all the sources of information and other facilities available of this connection are duly acknowledged.



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Contents



Abstract

1

CHAPTER: 1	<i>Introduction</i>	
1	Disease	1
1.1	Communicable disease	1
1.2	Non- Communicable disease	1
1.3	Cardiovascular disease or CVD	2
1.3.1	Magnitude of problem of CVD	2
1.3.2	Epidemiology of CVD	3
1.4	Types of CVD	3
1.4.1	Congenital heart disease	3
1.4.2	Acquired heart disease	4
1.5	The six types of cardiovascular disease	4
1.6	Global burden of CVD	6
1.7	Global trends in mortality of CVD	6
1.8	Global Trends in Mortality Of CHD	7
1.9	Risk Factors	10
1.10	Stroke	10
1.10.1	Global Trends in Mortality of Stroke	11
1.11	Congestive Heart Failure	11
1.12	Atrial Fibrillation	12

1.12.1	Global Burden Atrial Fibrillation	12
1.13	Relations to Stroke and Congestive Heart Failure	12
1.14	Hypertension	13
1.14.1	Global Burden of Hypertension	14
1.14.2	Global Awareness, Treatment, and Control of Hypertension	14
1.15	Smoking	15
1.15.1	Global Burden of smoking	16
1.15.2	Global Trends in Tobacco Consumption	16
1.15.3	Health Risks and Future Trends	16
1.15.4	Risks Associated with Environmental Tobacco Exposure	17
1.16	Physical Inactivity	18
1.16.1	Global Burden and Trends of Physical Inactivity	18
1.16.2	Risks of Physical Inactivity	18
1.17	Obesity	19
1.17.1	Global Burden of Obesity	19
1.17.2	Burden in the United States	19
1.17.3	Lifetime Risk of Developing Overweight or Obesity	20
1.18	Dyslipidemia	20
1.18.1	Global Burden and Trends of Dyslipidemia	20
1.18.2	Burden and Trends in the United States	20
1.18.3	Awareness, Treatment, and Control in the United States	22
1.19	Diabetes Mellitus	22

1.19.1	Global Trends of Diabetes Mellitus	22
1.19.2	Health Risks of Diabetes Mellitus	23
1.20	Metabolic Syndrome	23
1.20.1	Global Burden Of Metabolic syndrome	24
1.20.2	Burden in the United States	24
1.20.3	Risks of Metabolic syndrome	25
1.21	Inflammation	25
1.21.1	Distribution and Determinants in the United States	25
1.21.2	Cardiovascular Disease Risks of inflammation	25
1.22	Homocysteine	26
1.22.1	Cardiovascular Disease Risks	26
1.23	Drug used by Patient of CVD	27
1.24	Myocardial Infarction	28
1.24.1	Symptoms Myocardial Infarction	29
1.24.2	Heart Attack Causes	30
1.24.3	Risk factors of CVD and MI	31
1.25	Lipid profile	33
1.25.1	LDL and MI	33
1.26	Diagnostic of Myocardial infarction	34
1.26.1	Electrocardiogram	34
1.26.2	Blood tests	34
1.26.3	Chest X-ray	35

1.27	Treatment of myocardial infarction	35
CHAPTER:2	<i>Aim and Significance</i>	
2.1	Aim and objective of the study	37
2.2	Significance of the study	37
CHAPTER:3	<i>Materials and Method</i>	
3.1	Type of the study	39
3.2	Place of the study	39
3.3	Study Population	39
3.3.1	Inclusion criteria of the cases	39
3.3.2	Exclusion criteria of the cases	39
3.4	Sample size	40
3.5	Sampling technique	40
3.6	Research Approach	40
3.7	Research Equipment	40
3.8	Data collection method	40
3.8.1	Blood pressure measurement	41
3.9	Diagnosis of MI patients	41
3.9.1	Treatment	41
3.10	Study period	41
3.10.1	Data processing	42
3.10.2	Data analysis	42

CHAPTER: 4	RESULTS	
4.1	Distribution of patient's having myocardial infarction according to the gender.	43
4.2	Distribution Myocardial Infarction among patient Depending on Age	44
4.3	Distribution of myocardial infarction among different religions.	45
4.4	Distribution Myocardial Infarction among Smoker & Non Smoker	46
4.5	Distribution of MI patient among RBS	47
4.6	Distribution of MI patient among Hypertensive a non-hypertensive patient	48
4.7	Distribution of MI patient among patient percentage taking salt	49
4.8	Distribution of MI patient among patient percentage taking betel nut	50
4.9	Distribution Myocardial Infarction among Percentage of different type of MI	51
4.10	Distribution Myocardial Infarction among patient depending on creatinine.	52
4.11	Distribution Myocardial Infarction among patient depending Depending on Taking Tea or Cofee	53
4.12	Distribution Myocardial Infarction among patient depending Depending on Concentration of K+:	54
4.13	Distribution Myocardial Infarction among patient depending Depending on Concentration of Na+:	55
4.14	Distribution Myocardial Infarction among patient Depending on Occupation	56

4.15	Distribution Myocardial Infarction among patient Depending on Income	57
4.16	Distribution Myocardial Infarction among patient Depending upon oil use	58
4.17	Distribution Myocardial Infarction among patient Depending upon BP systolic	59
5.1.1	Distribution of Cholesterol level in MI patient	60
5.1.2	Distribution of HDL level among the MI Patients	60
5.1.3	Distribution of LDL among the MI Patients	61
5.1.4	Distribution of Triglyceride level among the MI Patients	61
Discussion		62
Conclusion		66
References		
Appendix		1-10



List of Figures

Fig 1.1: Myocardial infarction in heart.....	31
Fig 1.2. Angioplasty.....	36
Fig 1.3: Distribution (%) of myocardial infarction among male and female patients.....	43
Fig 1.4.: Distribution Myocardial Infarction among patient Depending on Age.....	44
Fig 1.5: Distribution (%) of myocardial infarction depending on religion.....	45
Fig 1.6: Distribution (%) of myocardial infarction in patients with smoking.....	46
Fig 1.7: Distribution Myocardial Infarction among Random Blood Sugar level (RBS).....	47
Fig 1.8 Distribution MI among Hypertensive and non Hypertensive patient.....	48
Fig 1.9 Distribution Myocardial Infarction among Percentage of MI Patient Taking Salt....	49
Fig 1.10 Distribution MI among Percentage of MI Patient taking betel nut.....	50
Fig 1.11: Distribution (%) of types of myocardial infarction	51
Fig 1.12 Distribution Myocardial Infarction among patient depending on creatinine.....	52
Fig 1.13: Distribution MI among patient depending on Taking Tea or Cofee	53
Fig 1.14: Distribution MI among patient depending Concentration of K+.....	54
Fig 1.15: Distribution MI among patient depending on Concentration of Na + level.....	55
Fig 1.16: Distribution Myocardial Infarction among patient Depending on Occupation.....	56
Fig 1.17 Distribution Myocardial Infarction among patient Depending on Income	57
Fig 1.18: Distribution (%) of myocardial infarction depending upon Oil use.....	58
Fig 1.19: Distribution (%) of myocardial infarction depending upon BP (systolic).....	59

List of Table

Table 1.1: Global Burden of CVD by World Health Organization Region.....	7
Table 1.2: Global Incidence and Prevalence of Cardiovascular Disease.....	8
Table 1.3. Global Burden of Cardiovascular Disease: Projected Future Burden.....	9
Table 1.4. Types of risk factors.....	10
Table 1.5. Distribution of Total Cholesterol among 135 MI Patients.....	60
Table 1.6. Distribution of HDL among the MI Patients.....	60
Table 1.7. Distribution of LDL among the MI Patients.....	61
Table 1.8. Distribution of Triglyceride among the MI Patients.....	61

List of Abbreviation

ACC: American College of Cardiology

AHA: American Heart Association

AMI: Acute Myocardial Infarction

BMI: Body Mass Index

CCU: Coronary Care Unit

CHD: Coronary Heart Disease

CHF: Congestive Heart Failure

CK: Creatinine Kinase

CKD: Chronic Kidney Disease

CPK: Creatinine Phospho Kinase

CT: Computed Tomography

CVD: Cardiovascular Disease

ECG: Electrocardiogram

ESC: European Society of Cardiology

ETT: Exercise Tolerance Testing

GI: Gastro intestinal

HDL: High Density Lipoprotein

HANES: Hispanic Health and Nutrition Examination Survey

HR: Heart Rate

LDL: Low Density Lipoprotein

LV: Left Ventricle

MI: Myocardial Infarction

MRI: Magnetic Resonance Imaging

NCHS: National Center for Health Statistics

NHES: National Health Examination Survey

NHIS: National Health Interview Survey

NICVD: National Institute of Cardiovascular disease

QMI: Old Myocardial Infarction

PAD: Peripheral artery disease

SCD: Sudden Cardiac Disease

SHS: Second Hand Smoking

WHO: World Health Organization



ABSTRACT

A myocardial infarction (MI) may be the first demonstration of coronary artery disease, or it may occur, repeatedly, in patients with established disease like high lipid profile, which is one of the major risk factors of MI. The excess percentage of Cholesterol and Low-density lipoprotein (LDL) and inadequate amount of High-density lipoprotein (HDL) cause the blockage of the coronary artery. This result lack of proper blood supply to the coronary arteries which ultimately cause insufficient oxygen transport to the heart muscle leading to MI. To find out the relationship between lipid profile and myocardial infarction we conducted a study with 135 MI patients at National Institute of Cardiovascular Disease (NICVD), Dhaka. We evaluated all medical history, diagnosis and treatment of the patients having MI. The other risk factors like smoking, hypertension, diabetes etc. were also investigated in MI patients to get an overall idea about the modifiable and non-modifiable risk factors of MI. Out of 135 patients 40 MI patients had high cholesterol levels (>240 mg/dl). Sixty seven patients had the cholesterol levels between 200-239 mg/dl. About 105 patients had HDL levels of 40 mg/dl or less. Seventy three patients had the LDL levels between 130 to 159 mg/dl, thirty six patients had the high LDL levels (162 to 189 mg/dl), and five MI patients had very high LDL levels (>190 mg/dl). It can be concluded that high lipid profile is one of the major risk factors of patients having MI admitted at NICVD.



Chapter 1

Introduction

1. Disease

A disease or medical condition is an abnormal condition of an organism that impairs bodily functions, associated with specific symptoms and signs. **(Disease at Dorland's Medical Dictionary 26 ed; 2001)**

1.1. Communicable disease

An infectious disease is a clinically evident illness resulting from the presence of pathogenic microbial agents, including pathogenic viruses, pathogenic bacteria, fungi, protozoa, multicellular parasites, and aberrant proteins known as prions. These pathogens are able to cause disease in animals and/or plants. Infectious pathologies are also called communicable diseases or transmissible diseases due to their potential of transmission from one person or species to another by a replicating agent (as opposed to a toxin). **(Dorland's Medical Dictionary for Healthcare Consumers)**

1.2. Non-communicable disease

Non-communicable disease is a disease which is not infectious. Such diseases may result from genetic or lifestyle factors. A non-communicable disease is an illness that is caused by something other than a pathogen. It might result from hereditary factors, improper diet, smoking, or other factors. Those resulting from lifestyle factors are sometimes called affluence. Examples include hypertension, diabetes, cardiovascular disease, cancer, and mental health problems, asthma, atherosclerosis, allergy etc. The non-communicable diseases are spread by: heredity, surroundings and behavior **(WHO, 2009)**. For example Obesity related non-communicable disease is considerably associated with high accumulation of calorie and uneven or less expenditure of calorie due to inadequate physical activity. Obesity could be related with many non-communicable diseases -Hypertension, Coronary Heart Disease (CHD), and NIDDM **(Kamla Raj, 2009)**.

1.3. Cardiovascular disease or CVD:

Heart disease or cardiovascular diseases is the class of diseases that involve the heart or blood vessels (**Maton & Anthea, 1993**). Most countries face high and increasing rates of cardiovascular disease. Each year, heart disease kills more Americans than cancer. (**US CDO,1999**). While the term technically refers to any disease that affects the cardiovascular system it is usually used to refer to those related to atherosclerosis (arterial disease). The broad term, CVD, includes coronary heart disease (CHD; myocardial infarction (MI), angina, coronary insufficiency, and coronary death), cerebrovascular disease (stroke and transient ischemic attacks), peripheral vascular disease, congestive heart failure (CHF), hypertension, and vascular and congenital heart disease.

1.3.1. Magnitude of problem of CVD:

Cardiovascular disease (CVD) is the number one cause of death worldwide (**Mathers, 2006; Murray and Lopez 1996; WHO 2002**). CVD covers a wide array of disorders, including diseases of the cardiac muscle and of the vascular system supplying the heart, brain, and other vital organs. It is widely acknowledged that heart disease and stroke are the leading causes of death and disability in the United States and other developed countries. What is less appreciated is that this holds true for the developing countries as well (**Chockalingam A, et al., 1999**). We are in the midst of a true global cardiovascular disease (CVD) epidemic. (**Bonow RO, et al., 2002**) CVD is responsible for approximately 30 percent of all deaths worldwide each year. (**WHO HR Report, 2002**) Nearly 80 percent of these deaths occur in low and middle income countries, and half occur in women CVD is the leading cause of mortality in every region of the world with the sole exception of sub-Saharan Africa where infectious diseases are still the leading cause .Furthermore, the projected trends in the global burden of CVD over the next two decades are elucidated, and ongoing efforts by the world community (including the World Health Organization (WHO) to combat and contain the current epidemic are outlined.



1.3.2. Epidemiology of CVD

At the beginning of the 20th century, CVD was responsible for less than 10 percent of all deaths worldwide, but by 2001 that figure was 30 percent. It predicted that CVD will be the leading cause of death and disability worldwide by 2020 mainly because it will increase in low- and middle-income countries. By 2001, CVD had become the leading cause of death in the developing world, as it has been in the developed world since the mid 1900s (**Mathers et al., 2006; WHO 2002**). Nearly 50 percent of all deaths in high-income countries and about 28 percent of deaths in low and middle-income countries are the result of CVD (**Mathers et al., 2006**). Other causes of death, such as injuries, respiratory infections, nutritional deficiencies, and HIV/AIDS, collectively still play a predominant role in certain regions, but even in those areas CVD is now a significant cause of mortality.

1.4. Types of CVD

CVD can be classifying into 2 types

- Congenital heart disease
- Acquired heart disease

1.4.1. Congenital heart disease:

Any abnormality of the heart that is present at birth. Cardiac abnormalities are generally caused by abnormal development of the heart and circulatory system before birth. Abnormal development can be caused by a variety of factors, including infection and use of certain drugs by the mother during pregnancy. Some congenital cardiac abnormalities are inherited and may be transmitted as autosomal or sex-linked traits. However, for some of the more common abnormalities, there is no obvious heritable relationship, nor is the origin of the disease readily explained. (**Encyclopedia Britannica Online,2010**)

1.4.2. Acquired heart disease

Acquired heart diseases are conditions affecting the heart and its associated blood vessels that develop during a person's lifetime, in contrast to congenital heart diseases, which are present at birth. Acquired heart diseases include coronary artery disease, coronary heart disease, rheumatic heart disease, diseases of the pulmonary vessels and the aorta, diseases of the tissues of the heart, and diseases of the heart valves. (**Encyclopedia Britannica , 2010**)

Acquired heart diseases include:

- Coronary artery disease or Ischemic heart disease
- Coronary heart disease,
- Rheumatic heart disease,
- Diseases of the pulmonary vessels and the aorta,
- Diseases of the tissues of the heart, and
- Diseases of the heart valves. (**Encyclopedia Britannica -2010**)

1.5. The six types of cardiovascular disease

i). **Ischemic heart disease or MI:** Is the most common type of cardiovascular disease in Canada and other industrialized countries around the world. It is the most common cause of death in most Western countries, and a major cause of hospital admissions (**WHO Health statistic, 2004**). It refers to problems with the circulation of blood to the heart muscle. A partial blockage of one or more of the coronary arteries can result in a lack of enough oxygenated blood (ischemia) thus causing symptoms such as angina (chest pain) and dyspnea (shortness of breath). A complete blockage of an artery causes necrosis (damage to the tissues) or a myocardial infarction, commonly known as a heart attack (**Public health agency of Canada**).

ii). **Cerebrovascular disease:** Stroke refers to a problem with the circulation of blood in the blood vessels of the brain. A blockage with effects lasting less than 24 hours is referred to as a transient ischemic attack. A complete blockage with long-term effects is referred to as a cerebrovascular thrombosis (clot) or accident or a stroke. Sometimes, a blood vessel in the brain can burst resulting in long term effects (**Public health agency of Canada**).

iii). **Peripheral vascular disease:** It affects the circulation primarily in the legs. Patients with this disease typically complain of pain in their calves especially when walking (**Public health agency of Canada**).

iv). **Heart failure:** Heart failure occurs when the pumping action of the heart cannot provide enough blood to the rest of the body as it is needed. This can happen as a result of damage to the heart muscle, for example from a heart attack, or from excessive consumption of alcohol, or because of a heart muscle disease also called a cardiomyopathy. Patients with heart failure usually suffer from shortness of breath and swelling of the legs (**Public health agency of Canada**).

v). **Rheumatic heart disease:** once common in Canada is a major problem in many poor countries. This disease begins with a bacterial infection in childhood, affecting joints and heart valves. The heart problems appear many years later. Often the valves have to be replaced by an operation. Other infections can occur attacking the inner tissues of the heart including the valves (endocarditis) and the outer tissue overlying the heart or pericarditis (**Public health agency of Canada**).

vi) **Congenital heart disease:** is a problem with the structure of the heart arising because of a birth defect. These anatomical defects can be as simple as a small hole in one of the inside walls of the heart or they can be very complex, affecting the way blood flows through the heart and lungs. Some congenital heart problems result in death unless immediately corrected by surgical intervention. Others cause disability to varying degrees and are treated by surgery later in life with correction of the problem sometimes requiring more than a single operation (**Public health agency of Canada**).

viii) Coronary Heart disease:

Coronary disease (or coronary heart disease) refers to the failure of coronary circulation to supply adequate circulation to cardiac muscle and surrounding tissue. It is already the most common form of disease affecting the heart and an important cause of premature death in Europe, the Baltic States, Russia, North and South America, Australia and New Zealand. It has been predicted that all regions of the world will be affected by 2020. (Boon NA et al, 2006) It is most commonly equated with atherosclerotic coronary artery disease, but coronary disease can be due to other causes, such as coronary vasospasm. It is possible for the stenosis to be caused by the spasm.

1.6. Global Burden of CVD

CVD is the leading cause of mortality worldwide; responsible for one-third of all deaths. According to WHO estimates, 17.5 million people died of CVD in 2005 (Grundy SM, 1997). Developing countries contributed to 80 percent of CVD deaths. There is considerable variation in CVD mortality rates across WHO regions (Table-1.1) and across countries. Potential reasons for such variation include differing stages of epidemiologic transition in various countries, varying environmental effects caused by dissimilar burden of CVD risk factors, inherent genetic differences, and distinct early childhood programming influences. (Reddy KS, 1998)

1.7. Global Trends in Mortality of CVD

The WHO projections indicate that a pattern of premature CVD mortality is likely to persist and can accentuate further in developing countries. In 2006, CVD is more prevalent in China and India than in all developed countries combined. By 2010, CVD is projected to be the leading cause of death in developing countries. By 2020, WHO estimates there will be nearly 20 million CVD deaths worldwide every year, and the number will increase to 24 million by 2030. Developing countries will account for 70 percent of deaths caused by coronary heart disease and 75 percent of deaths caused by stroke (Table 1.3) (Lamarche B et al., 1996).

Table 1.1 Global Burden of CVD by World Health Organization Region (2002 Data)

CVD	AFR	AMR	EUR	SEAR	WPR	EMR	World
Mortality(thousands)							
CHD	332	921	2373	2039	993	537	7208
Cerebrovascular	360	452	1447	1059	1958	226	5509
HTN heart disease	60	135	180	153	284	97	911
Rheumatic	19	10	30	133	109	24	327
Inflammatory	42	67	101	76	80	37	404
Other CVD	224	342	794	451	400	158	2374
All CVDs	1036	1928	4926	3911	3825	1079	16733
Total burden, DALYs (millions)							
CHD	3.03	6.22	15.75	20.73	7.50	5.32	58.64
Cerebrovascular	3.67	4.48	10.79	10.40	17.28	2.53	49.20
HTN heart disease	0.59	1.03	1.22	1.66	2.24	0.89	7.65
Rheumatic	0.51	0.16	0.38	2.62	1.61	0.58	5.86
Inflammatory	0.87	0.85	1.31	1.51	0.75	0.56	5.85
Other CVD	2.26	2.45	4.96	6.07	3.03	2.18	20.98
All CVDs	10.91	15.17	34.42	42.99	32.41	12.06	148.19

SOURCE: AFR, Africa; AMR, America; CHD, chronic heart disease; CVD, cardiovascular disease; DALYS, disability-adjusted life-years; EMR, Eastern Mediterranean Region; EUR, Europe; HTN, hypertension; SEAR, South East Asia Region; WPR, Western Pacific Region.

1.8. Global Trends in Mortality of CHD

The MONI study examined changes in risk factors, and coronary care in redefined populations in 31 countries over a 10-year period from the mid-1980s to the mid-1990s. On average, coronary event rates decreased from 23 (women) to 25 (men) percent, while CHD mortality rates reduced by 34 (women) to 42 (men) percent during

the observation period. The greatest decline in coronary event rates in men occurred in north European populations namely, Finland, which had the highest, levels at the beginning of the observation period, and northern Sweden. Populations experiencing notable increases in coronary event rates were predominantly from central and Eastern Europe and Asia, although the general pattern of increases and decreases appeared to be less consistent in women. In regions where coronary mortality rates were falling, it is estimated that improvements in survival contributed one-third, and change in heart attack rates accounted for two-thirds, on average, of the total change in survival rates. These data underscore the importance of both the prevention of heart disease and improved care of acute events in determining CHD mortality rates at the population level.

Table 1.2 Global Incidences and Prevalence of Cardiovascular Disease

CVD	AFR	AMR	EUR	SEAR	WPR	EMR	World
Annual Incidence in 2000 (thousands)							
CHD ^a	292	877	1932	1665	647	431	5844
Cerebrovascul lar ^b	858	1438	3901	2964	5468	622	15251
Cerebrovascul lar ^d	1637	5299	11669	5752	13706	1391	39,455

^aAcute MI, ^bFirst ever stroke, ^cIncludes angina, ^dFirst-ever stroke survivors AFR, Africa; AMR, America; CHD, chronic heart disease; CVD, cardiovascular disease; EMR, Eastern Mediterranean Region; EUR, Europe; HTN, hypertension; SEAR, South East Asia Region; WPR, Western Pacific Region. SOURCE: Available at: www3.who.int/whosis/menu.cfm?path=whosis_burden_burden_estimates_burden_estimates_2002N_burden_estimates_2002N_2002Rev_burden_estimates_2002N_2002Rev_Region&language=En Accessed on August 15, 2006

Table 1.3 Global Burden of Cardiovascular Disease: Projected Future Burden

Global Burden	2010	2020	2030
CVD Mortality	110	103	+7
Annual, million	18.1	20.5	24.2
All deaths (%)	30.8	31.5	32.5
CHD death, % of all	134	121	+13
Men	13.1	14.3	14.9
Women	13.6	13.0	13.1
Men	13.1	14.3	14.9
Stroke death, % of all			
Men	9.21	9.8	10.4
Women	11.5	11.5	11.8
CVD DALYs			
Annual, million	153	169	187
% of all DALYs	10.4	11.0	11.6
Global rank	3 rd : CHD	3 rd : CHD	3 rd : CHD
	5 th : Stroke	4 th : Stroke	4 th : Stroke
Rank in developing countries	4 th : CHD	3 rd : CHD	3 rd : CHD
	8 th : Stroke	6 th : Stroke	5 th : Stroke

The decline in CHD mortality in developed countries is in sharp contrast to future projections for the developing countries. Between 1990 and 2020, CHD mortality is expected to increase by 120 percent in women and by 137 percent in men in developing countries. It is estimated that the annual number of deaths caused by CHD in developing countries will rise to 11.1 million in 2020. CHD mortality will triple in Latin America, the Middle East and sub-Saharan Africa over the next two decades. By contrast, in developed countries CHD mortality is projected to increase by about 30 to 60 percent, largely because of the aging of the population (Yusuf S, 2001)



1.9 Risk Factors

Two type of risk factor in MI are modifiable and non modifiable. Modifiable which risk factor we can control by our self and non modifiable not able to control, it generally occurs increasing age and family or genetic fault. Overall global burden of all risk factor study are given below:

Table 1.4: Types of risk factors

Modifiable Factors	Non Modifiable Factors
High blood pressure	Increasing age
High cholesterol	Family history
Smoking	Sex
Diabetes	Genetic
Obesity	
Physical inactivity	

1.10. Stroke

Risk Factors: Age, elevated blood pressure, smoking, diabetes mellitus, electrocardiographic left ventricular hypertrophy, and atrial fibrillation are the major risk factors for stroke. A stroke risk score has been developed to estimate the risk of stroke. Global Burden: It is estimated that 15 million people suffer a stroke each year, and 5 million incur a permanent disability as a result. There are 5.5 million stroke deaths worldwide each year. Strokes accounted for the loss of 49.2 million DALYs worldwide in 2002 (see Table 1.1). Every year there are approximately 15.3 million new strokes and 39 million prevalent cases worldwide (see Table 1.1) (Mackay J, 2006) .

1.10.1. Global Trends in Mortality of Stroke

Stroke mortality has declined in the developed world over the last two decades. Data from the MONICA Study demonstrate a modest contribution of reduction in risk factors such as hypertension to the decline in stroke mortality in women, but not in men. **(Tolonen H et al., 2002).**

Global mortality because of cerebrovascular disease in the next two decades will parallel the CHD trends noted in an earlier section, with a 124 percent increase in women and a 107 percent increase in men in the developing countries, compared to increments of 56 percent in women and 28 percent in men in developed countries. **(Yusuf S, 2001)**

1.11. Congestive Heart Failure

Risk Factors: Advancing age, MI, hypertension, diabetes mellitus, valvular heart disease, and obesity are key risk factors for CHF. High blood pressure antedates more than 75 percent of heart failure **(Levy D,1996)**. A clinical risk score has been formulated to estimate the risk of developing CHF based on several of these risk factors **(Kannel WB, 1999)**

1.11.1. Global Burden of Congestive Heart Failure

CHF is clearly a major clinical and public health problem. The exact magnitude of the problem is difficult to assess because we lack broadly based population estimates of its prevalence, incidence, and mortality rates. It is estimated that there are nearly 23 million people with heart failure worldwide. Global Trends in Mortality: It is estimated that the burden of CHF will increase over the next two decades in developed countries **(Bonneux L, 1994)**. Despite a stable incidence rate, increasing prevalence can result because of a reduction in CHF mortality. **(Levy D, 2002)**



1.12. Atrial Fibrillation:

Risk Factors:

such as advancing age, male sex,

heart failure, MI, valvular heart disease, and increasing left atrial size. (Stewart S., 2001; Wang TJ., 2004). Miscellaneous factors associated with incident atrial fibrillation including alcohol consumption, hyperthyroidism, and reduced lung function are associated with an increased risk of atrial fibrillation (Mukamal KJ., 2005). Recent studies reported that elevated biomarkers such as C-reactive protein (CRP) and B-type natriuretic peptide concentrations also predict an increased risk of incident atrial fibrilla

are causally related to atrial fibrillation remains to be determined. Additionally, there is increasing evidence that there is a genetic predisposition to atrial fibrillation (Arnar DO., et al., 2006).

In contrast to industrialized countries, in developing countries valvular heart disease appears to be the most common predisposing condition (Arnar DO, et al, 2006). However, with the globalization of cardiovascular disease risk factors, hypertension and CHD are increasing in importance in developing countries.

1.12.1. Global Burden Atrial Fibrillation

The global burden of atrial fibrillation is unknown because most atrial fibrillation research has been conducted in North America and Western Europe (Ryder KM.,1999) Even within these geographic constraints, the reported studies have been from predominantly white cohorts.

1.13. Relations to Stroke and Congestive Heart Failure

Atrial fibrillation has been demonstrated to be an independent risk factor for stroke (Wolf PA., 1991) in virtually all settings and countries studied, with an annual stroke rate averaging approximately 5 percent in untreated patients. In Japan the adjusted risk ratio for stroke was 4.3 in women and 6.9 in men (Nakayama T.,1997) Canadian male air

force recruits with atrial fibrillation have an age-adjusted doubling of stroke risk in follow-up data from the Framingham Heart Study suggest that whereas the relative risk of atrial fibrillation for stroke does not change substantively with advancing age (rate ratio ranging from 3 to 5), the percentage of strokes attributable to atrial fibrillation increases markedly from 1.5 percent in subjects in their 50s to 24 percent in subjects 80 to 89 years of age, reflecting the higher prevalence of atrial fibrillation with advancing age (Wolf PA., 1991).

The relations between atrial fibrillation and heart failure are complex because they share common risk factors and can each predispose to the other's development. Atrial fibrillation doubles to triples the risk of developing congestive heart failure adjusting for the coexistent risk factors (Stewart S., 2002). In addition, in individuals with either atrial fibrillation or congestive heart failure, development of the other condition increases mortality. Risk prediction models for stroke and stroke and death (Wang T.J., 2003); have been developed to help clinicians assess the prognosis of patients with atrial fibrillation. Given the relation to heart failure and stroke it is not surprising that atrial fibrillation is a costly illness. In a study from the United Kingdom it was noted to increase from 1995 to 2000, rising from about 244 to 459 million pounds, or about 0.62 percent to 0.97 percent of the total National Health Service spent on atrial fibrillation.

1.14. Hypertension

Definition: Numerous epidemiologic investigations have demonstrated that blood pressure is related to vascular mortality in continuous fashion. (MacMahon S *et al.*, 1990; 2002) Given the continuous relations of blood pressure to vascular risk, any definition of hypertension is somewhat arbitrary, and largely based on thresholds for which there is evidence that the benefits of lowering blood pressure outweigh potential risks of treatment. It is not surprising, therefore, that the definition of *high* blood pressure (hypertension) has been lowered in successive blood pressure guidelines over the past 35 years. Guidelines of the seventh Joint National Committee on prevention, detection, evaluation, and treatment of high blood pressure in the United States (JNC VII) and the WHO-International Society for Hypertension define hypertension as a systolic blood

pressure of 140 mmHg or greater, or a diastolic blood pressure of 90 mmHg or greater, or the use of antihypertensive medication (**WHO hypertens, 1999**). The JNC VII also has categorized blood pressure readings in the range of a systolic pressure of 120 to 139 mmHg or a diastolic pressure of 80 to 89 mmHg as *prehypertension*. (**Chobanian AV., Bakris GL**) An important reason for this change is to simplify the classification system of blood pressure and to emphasize the continuous risk of relations of blood pressure to vascular disease.

Risk Factors: Advancing age, sedentary lifestyle, excess weight, increased dietary salt consumption, and reduced intake of potassium and increased alcohol consumption have been identified as risk factors for developing high blood pressure (**Chobanian AV, Bakris GL**). Family history of hypertension and African American ancestry has also been observed to elevate the risk of developing high blood pressure. Prehypertension is associated with increased risk of progression to hypertension, relative to those with optimal levels of blood pressure (**Vasan RS., 2001**).

1.14.1. Global Burden of Hypertension

Hypertension is the most common CVD disorder, affecting approximately 20 percent of the adult population. It is considered both as a disease condition and as one of the major risk factors for heart disease, stroke, and kidney disease. Worldwide an estimated 600 million people have high blood pressure. About 15 to 37 percent of the adult population worldwide is afflicted with hypertension, in those older than 60 years of age, as many as one-half are hypertensive in some populations (**Kearney PM., 2005**). In general, hypertension prevalence is higher in urban settings as compared with rural settings. It is estimated that the global prevalence of high blood pressure will increase to 1.56 billion by 2025.

1.14.2. Global Awareness, Treatment, and Control of Hypertension:

The detection and control of hypertension remains a challenge even in developed countries. The detection rates in most developed countries vary from 32 to 64 percent, whereas in many developing countries the reported detection rates are substantially

lower. The control rates in those already being treated for hypertension varies from 13 to 29 percent. However, in African countries, control rates were reported to be as low as 2 percent (**Fuentes R, Ilmanemi N**). Data from the MONICA Study demonstrated small decreases in mean systolic blood pressures in most countries evaluated and in both sexes during the time period 1979 to 1996. Acute rheumatic fever and subsequent rheumatic heart disease remain important cardiovascular problems in the tropical and subtropical developing countries of the Middle East, South America, Africa, and Asia, and there have been outbreaks in the United States in recent years. The incidence, however, remains higher in subgroups such as Polynesians, Australian aborigines, Maoris in New Zealand, and within the United States among blacks, Puerto Ricans, Mexican Americans, and Native Americans. Overall, incidence rates vary widely from less than 1 per 100,000 in developed countries to as high as 150 per 100,000 in China. Rheumatic fever is rare before 3 years of age, occurring most frequently between 5 and 15 years of age, when streptococcal infections are most frequent. During epidemics of streptococcal pharyngitis, the rheumatic fever attack rate can be 3 percent, whereas in endemic situations it is usually only 0.3 percent. In developing countries, rheumatic fever is the most frequent cause of heart disease in the pediatric age group, accounting for 25 to 40 percent of all CVD and 33 to 50 percent of all hospital admissions. The prevalence is high in the African continent, where it can reach 15:1000 school children. It is estimated that 12 million patients with rheumatic heart disease require further treatments to prevent disability and death because of rheumatic heart disease; of these, two-thirds are children of school age. More than 2 million require repeated hospital admissions and 300,000 die because of the illness every year. Another 1 million will need heart surgery in the next 5 to 20 years.

1.15. Smoking

Compare risk of myocardial infarction associated with smoking in men and women, taking into consideration differences in smoking behavior and a number of potential confounding variables. Ischemic heart disease is responsible for about 40% of deaths in Western countries, with smoking as a major modifiable risk factor (**National Institutes of Health., 1994**).

1.15.1. Global Burden of smoking

It is estimated that there are approximately 1.3 billion smokers (250 million women) in the world today, and these individuals consume an average of 14 cigarettes each per day. Of these, 300 million live in developed countries, whereas more than 900 million reside in developing countries. Overall, 47 percent of men and 12 percent of women in the world are current smokers. In developing countries, it is estimated that 48 percent of men and 7 percent of women smoke, whereas in developed countries, 42 percent of men and 24 percent of women are smokers. East-Asian countries account for a disproportionately high percentage (38 percent) of world smokers (**Yang G., 1916**). More than 60 percent of men in China are present smokers, as are more than 40 percent of men in India.

1.15.2. Global Trends in Tobacco Consumption

Tobacco consumption fell between 1981 and 1991 in most developed countries. In developed countries the decrease in smoking prevalence has been lowest among the least educated. By contrast, consumption is increasing in developing countries by about 3.4 percent per annum, having risen dramatically in some countries in recent years. Data from the MONICA Study suggest that whereas smoking rates are declining in most of the male populations, rates in women are increasing (**Evans A., 2001**). Smoking is increasing at an alarming rate among young women, especially in Eastern Europe. It is estimated that the number of individuals who smoke will increase to 1.7 billion throughout the world by 2025.

1.15.3. Health Risks and Future Trends

Smokers of all ages have a two- to threefold elevated risk of dying prematurely compared to nonsmokers. (Niu SR, 1998) Between the ages of 35 to 69 years, smokers lose approximately 20 years of life expectancy relative to nonsmokers. At older than 70 years of age, smokers lose approximately 8 years of life relative to nonsmokers. Smoking is an important CVD risk factor in both men and women, being particularly harmful in the latter after menopause and in those who use oral contraceptives. Smoking increases the risk of stroke and CHD by 100 percent, that for peripheral arterial disease by 300 percent,

and the risk of developing an aortic aneurysm by 400 percent (Peto R et al., 1996). Prospective studies show that cigarette smoking causes approximately 30 percent of CVD deaths worldwide. This is especially evident in populations with the clustering of CVD risk factors (i.e., those with diets that are high in saturated fat with subsequent high blood cholesterol and high blood pressure).

Smoking is responsible for 90 percent of all lung cancers and for 75 percent of chronic obstructive pulmonary disease (Peto R et al., 1996). WHO estimates that tobacco was responsible for 10 percent of the total global mortality and caused about 4.9 million deaths worldwide in 2000 and more than 59 million DALYs (4.1 percent of total) were lost because of smoking in 2000. In the United States, from 1995 to 1999, an average of 442,400 individuals died each year from smoking-related illnesses. One-third of these were CVD related.

Based on current smoking patterns and trends, smoking is expected to kill 10 million people annually worldwide by 2020, this is more than the total of deaths from malaria, maternal

is that more than 70 percent of these deaths will be occurring in developing countries. By 2020, smoking will cause about one in three of all adult deaths. Half of these deaths will occur in middle-age and at younger than 70 years of age.

Health risks diminish with smoking cessation. According to WHO, 1 year after quitting, the risk of CHD decreases by 50 percent, and within 15 years, the relative risk of dying from CHD for an ex smoker approaches that of a long-time (lifetime) nonsmoker.

1.15.4. Risks Associated with Environmental Tobacco Exposure

The risk of death from CHD increases by up to 30 percent among those exposed to environmental tobacco smoke at home or work. It is estimated that about 35,000 nonsmokers die from CHD each year as a result of exposure to environmental tobacco smoke (US Chronic Disease Overview., 1999).



1.16. Physical Inactivity

Significance

It is widely accepted that daily moderate-intensity physical activity helps lower blood pressure, reduce body fat, and improve glucose metabolism. Indeed, physical activity is essential to maintain overall good health and is important in maintaining a healthy weight. Physical activity also reduces the risk of diabetes mellitus, hypertension, CVD, and all-cause mortality (WHO., 2006).

1.16.1. Global Burden and Trends of Physical Inactivity

WHO estimates that 60 percent of the world population is insufficiently physically active, a situation that is particularly striking among women and that undoubtedly has contributed to the increased prevalence of obesity and diabetes. Physical inactivity is widespread in developed countries and is increasing in urban areas of developing countries, especially in poorer communities. This trend for physical inactivity is influenced by cultural patterns, local traditions, and the lack of civic organizations to promote the benefits of exercise. In developing countries that previously relied on walking or bicycling for transportation, there has been a progressive increase in the use of automobiles and motorized public transportation.

1.16.2. Risks of Physical Inactivity

Physical inactivity caused about 1.9 million deaths globally in 2000. It is estimated that about 20 percent of cases of CHD, 15 percent of diabetes and some cancers, and 10 percent of strokes are attributable to physical inactivity. The relative risk of CHD associated with physical inactivity ranges from 1.5 to 2.4, relative to people who do follow current minimum physical activity recommendations. This increase in risk associated with physical inactivity is comparable to that observed for high blood cholesterol, high blood pressure, or cigarette smoking. World Health Organization Physical Activity Initiative WHO has begun formulating a Global Strategy on Diet, Physical Activity and Health, under a May 2002 mandate from the World Health Assembly (WHO., 2006).

1.17. Obesity

Definition: Overweight and obesity are currently defined by body mass index (BMI; calculated as weight in kilograms per height in meters).² A BMI of 25.0 to 29.9 defines overweight, and a BMI 30.0 defines obesity.

1.17.1. Global Burden of Obesity

Obesity is a disease condition that is highly prevalent in both developing and developed countries (Fig. 2–5). According to WHO data, an estimated 1 billion people across the world are now overweight or obese. Between 50 and 75 percent of the adults studied in the MONICA study were overweight or obese, and low levels of education were associated with increased body mass index (Molarius A et al., 2000). In the European Union, an estimated 200 million of the 350 million adults are overweight or obese. The WHO estimates that about 18 million children younger than 5 years of age are overweight, and these children are at increased risk of developing adult obesity and related problems of dyslipidemia and hypertension in their teen years (Ebbeling CB., 2002).

Global Trends in Prevalence: Obesity rates have risen threefold or more in some parts of the Middle East, North America, Eastern Europe, the Pacific Islands, Australia, and China since 1980. BMI is increased in approximately half of the female populations and in two-thirds of the male population. It is estimated that the number of overweight people in the world will increase to 1.5 billion by 2015 if current trends continue unabated.

1.17.2. Burden in the United States

In the United States, 35 percent of United States adults were considered overweight (BMI 25.0 to 29.9 kg/m²), and another 30 percent were considered obese (BMI 30.0 kg/m²) in 2003 (see Table 2–7). Hispanic men and Hispanic and black women were more likely to be overweight or obese than were their white counterparts. Among non-Hispanic black women, approximately one-half of females older than 40 years of age were obese, and 77 percent were overweight. A socioeconomic gradient is evident, with excess weight being

more common in those in a low social class and with the least education. The estimated annual health care cost of overweight and obesity is \$117 billion. (Thom T et al., 2006)

1.17.3. Lifetime Risk of Developing Overweight or Obesity

Recent data from the Framingham Heart Study indicate that at 50 years of age the lifetime risk is 1 in 2 for developing "overweight or more," 1 in 4 for obesity, and 1 in 10 for stage II obesity (BMI 30).

1.18. Dyslipidemia

Dyslipidemia is a disruption in the amount of lipids in the blood. In societies of developed countries, most dyslipidemias are hyperlipidemias; that is, an elevation of lipids in the blood, often due to diet and lifestyle. The prolonged elevation of insulin levels can lead to dyslipidemia. Increased levels of O-GlcNAc transferase (OGT) are known to cause dyslipidaemia. (Fredrickson DS, 1965)

1.18.1. Global Burden and Trends of Dyslipidemia

It is estimated that more than 80 percent of the world population has suboptimal levels of serum cholesterol (i.e., in excess of 150 mg/dL). Excessive levels of serum cholesterol are estimated to cause 18 percent of global cerebrovascular disease (mostly nonfatal events) and 56 percent of global CHD. Overall this amounts to approximately 4.4 million deaths (7.9 percent of total; Fig. 2–6) and 40.4 million DALYs (2.8 percent of total). Data from the MONICA Study demonstrate small decreases in mean cholesterol levels of the populations studied between 1979 and 1996 (WHO MONICA Project, 2001).

1.18.2. Burden and Trends in the United States

In the United States, the age-adjusted mean cholesterol is 5.27 mmol/L (203 mg/dL) (Thom T, et al., 2006). An estimated 34.5 million Americans have serum cholesterol levels of 240 mg/dL or higher, an estimated 17 percent of the adult population (see Table 2–7). An estimated 99.9 million, or one-half of the adult population, have serum cholesterol levels in excess of 200 mg/dL. Of note, approximately 10 percent of

adolescents (ages 12 to 19 years) have total serum cholesterol levels exceeding 200 mg/dL. The prevalence of elevated However, beginning at 50 years of age a higher proportion of women than men have total blood cholesterol of 200 mg/dL or higher. The mean level of LDL cholesterol for American adult's age 20 years and older is 123 mg/dL. About 43 percent of men and 35.8 percent of adults have LDL cholesterol levels in excess of 130 mg/dL. Values of HDL cholesterol of less than 40 mg/dL are considered low. About 33.6 percent of men and 12.6 percent of women have values below this threshold. The prevalence of a low HDL cholesterol value is slightly lower in black women relative to white and Hispanic females. Trends in the United States Serial NHANES surveys I to III have demonstrated a sequential decrease in the percentage of individuals with elevated serum cholesterol levels. This observation is consistent across all ethnicities, both sexes, and in all educational strata. These data suggest a change in population determinants of serum cholesterol levels in the United States, such as the dietary content of saturated fats, despite the increase in the prevalence of overweight noted across the surveys. After NHANES III, decline in serum cholesterol levels has been limited. Between NHANES III and NHANES 1999 to 2002, the age-adjusted mean total cholesterol serum cholesterol level is similar in men and women and comparable across ethnicities. concentration decreased marginally from 5.31 mmol/L (206 mg/dL) in NHANES III to 5.27 mmol/L (203 mg/dL) in NHANES 1999 to 2000. Awareness, Treatment, and Control in the United States for hypercholesterolemia mirror the suboptimal patterns observed with hypertension. In the NHANES investigation between 1999 and 2002, among participants who had a total cholesterol concentration 5.2 mmol/L (200 mg/dL) or who reported using cholesterol-lowering medications, only 63.3 percent reported awareness that they had hypercholesterolemia. Women, blacks, and Mexican-Americans were less likely to be aware of hypercholesterolemia (Thom T., 2006).

1.18.3. Awareness, Treatment, and Control in the United States

In the United States awareness, treatment, and control levels for hypercholesterolemia mirror the suboptimal patterns observed with hypertension. In the NHANES investigation between 1999 and 2002, among participants who had a total cholesterol concentration 5.2 mmol/L (200 mg/dL) or who reported using cholesterol-lowering medications, only 63.3 percent reported awareness that they had hypercholesterolemia. Women, blacks, and Mexican-Americans were less likely to be aware of hypercholesterolemia. (Thom T., 2006).

1.19. Diabetes Mellitus

An estimated 170 million people are affected by diabetes—the majority by type II diabetes. (Roglic G,2005). Two-thirds of these individuals live in the developing world. The top 10 countries, in terms of absolute numbers of individuals with the condition, are India, China, United States, Indonesia, Japan, Pakistan, Russia, Brazil, Italy, and Bangladesh.

1.19.1. Global Trends of Diabetes Mellitus

The prevalence of diabetes worldwide is increasing at an alarming rate. The prevalence of diabetes in adults globally was estimated to be 4.0 percent in 1995 and was projected to rise to 5.4 percent by the year 2025. The number of adults with diabetes in the world was 30 million in 1985 and is projected to double from 135 million in 1995 to 360 million in 2030. The proportional rise projected by 2030 is much larger in developing countries where a 170 percent increase (from 84 million to 298 million) is estimated, compared to a 42 percent increase in developed countries (from 51 million to 72 million). The highest increase is projected to occur in India and China. Indeed, the prevalence of diabetes in these two countries combined will increase from 45 million in 1995 to an estimated 121 million in 2030. The vast majority of people with diabetes in developing countries are likely to be between the ages of 45 and 64 years, while those in developed countries will be age 65 years or older. The main reasons for the rising epidemic of diabetes are

population aging, unhealthy diets, increasing epidemics of obesity, and sedentary lifestyles.

1.19.2. Health Risks Diabetes Mellitus

The number of deaths attributed to diabetes was previously estimated at just over 800,000 worldwide. However, it has long been known that the number of deaths related to diabetes is considerably underestimated. A more plausible figure is likely to be approximately 4 million deaths per year related to the presence of the disorder. This is about 9 percent of the global total mortality. Most deaths caused by diabetes are premature. Approximately 75 percent of the mortality among diabetic men and 57 percent among diabetic women are attributable to CVD. Among people with diabetes, CVD is 2 to 4 times more common; the risk of stroke is 2 to 4 times higher; and more than 60 percent have high blood pressure (APC., 2003).

1.20. Metabolic Syndrome

The metabolic syndrome has been defined as the presence of three or more of the following abnormalities: waist circumference 102 cm (40 in) in men and 88 cm (36 in) in women; serum triglyceride level 150 mg/dL (1.7 mmol/L) or treatment for elevated triglycerides; HDL cholesterol level <40 mg/dL (1.03 mmol/L) in men and <50 mg/dL (1.3 mmol/L) in women or treatment for low HDL; blood pressure 130/85 mmHg or treatment for elevated blood pressure; or serum glucose level 100 mg/dL (5.6 mmol/L) or treatment for elevated blood sugar. WHO defines the *metabolic syndrome* as the presence of diabetes, impaired glucose tolerance, impaired fasting glucose, or insulin resistance plus two or more of the following abnormalities: (Alberti KG., 1998)

1. High blood pressure defined as a value 140/90 mmHg
2. Hyperlipidemia identified by a triglyceride concentration 150 mg/dL (1.695 mmol/L) and/or HDL cholesterol <35 mg/dL (0.9 mmol/L) in men and <39 mg/dL (1.0 mmol/L) in women
3. Central obesity characterized by a waist-to-hip ratio of >0.90 in men or >0.85 in women and/or BMI >30 kg/m²
4. Microalbuminuria denoted by a urinary albumin excretion rate 20 g/min or an albumin-to-creatinine ratio 30 mg/g

More recently, the International Diabetes Federation has defined the metabolic syndrome as: presence of central obesity (defined as waist circumference 94 cm for European men and 80 cm for European women, with ethnicity specific values for other groups) plus any two:

1. *Raised triglycerides*, 150 mg/dL (1.7 mmol/L) or specific treatment for this lipid abnormality
2. *Reduced HDL cholesterol*, <40 mg/dL (1.03 mmol/L) in men, <50 mg/dL (1.29 mmol/L) in women, or specific treatment for this lipid abnormality
3. *Raised blood pressure*, systolic 130 mmHg or diastolic 85 mmHg, or treatment of previously diagnosed hypertension
4. *Raised fasting plasma glucose*, 100 mg/dL (5.6 mmol/L)

1.20.1. Global Burden of Metabolic syndrome:

The prevalence of the metabolic syndrome varies from 10 to 25 percent based on the criteria used, the population investigated, and the age of the sample. Prevalence increases with age and is much higher in individuals with diabetes mellitus.

1.20.2 Burden in the United States

There are 47 million Americans with the metabolic syndrome, with an overall age-adjusted prevalence of 24 percent. Older age, postmenopausal status, higher BMI, high carbohydrate consumption, physical inactivity, and Mexican American ethnicity are key correlates of the metabolic syndrome. Prevalence increases with age exceeding 40 percent in individuals older than 65 years of age. Among different ethnicities, Mexican Americans have the highest age-adjusted prevalence of the metabolic syndrome (31.9 percent). The age-adjusted prevalence is similar for men (24.0 percent) and women (23.4 percent). However, among African Americans, women have about a 57 percent higher prevalence than do men, and among Mexican Americans, women have about a 26 percent higher prevalence than do men. (Lakka HM et al., 2002)

1.20.3. Risks of Metabolic syndrome

The presence of the metabolic syndrome is an ominous indicator of future CVD risk. (Grundy SM., 2005) .In a prospective study of Finnish men, the metabolic syndrome was associated with a threefold increased risk of CHD/CVD and twofold elevated mortality relative to individuals without the syndrome.

1.21. Inflammation

Inflammation is a fundamental component of atherosclerosis (Libby P., 2002). Plasma levels of several inflammatory markers have been used as a surrogate for vascular inflammation, including that in the atherosclerotic plaque. CRP has emerged as a premier inflammatory marker. There is a paucity of data in the published literature regarding the distribution of CRP levels in developing countries.

1.21.1. Distribution and Determinants in the United States

The distribution of CRP (using high-sensitivity C-reactive protein [hs-CRP] assays) has been investigated in men in the NHANES 1999 to 2000 survey. The median CRP concentrations were 1.6 mg/L for all men, 1.6 mg/L for white men, 1.7 mg/L for African American men, 1.5 mg/L for Mexican American men and 1.8 mg/L for other men. Age, BMI, and smoking are other positive correlates of CRP (Ford ES Survey 1999–2000) Ethnic and racial variation in CRP levels also has been observed in Canadian samples and is not fully explained by cardiovascular risk factors.

1.21.2. Cardiovascular Disease Risks of inflammation

Plasma levels of CRP are elevated in CHD patients (Libby P Circulation- 2002) .Plasma CRP predicts a wide variety of CVD endpoints including CHD, peripheral vascular disease, and stroke. There is an increased risk of CHD, even at levels below those indicating acute inflammation in clinical practice. Assays for hs-CRP to assess cardiovascular risk are not yet used in routine clinical practice. On the basis of the available evidence, Centers for Disease Control and AHA workshop recommended

against screening of the entire adult population for hs-CRP as a public health measure. The group recommended that hs-CRP measurement appears to have its best utility when performed to detect enhanced absolute risk in persons in whom multiple risk-factor scoring indicates an intermediate 10-year CHD risk (10 to 20 percent). However, the benefits of this strategy or any treatment based on this strategy remain uncertain (Smith SC., 2004).

1.22. Homocysteine

Plasma homocysteine levels show a strong inverse correlation both with dietary intake and with plasma levels of the vitamins folate, B₆, and B₁₂, all of which are essential cofactors in homocysteine metabolism. A common polymorphism in the gene for methylene-tetrahydrofolate reductase appears to influence the sensitivity of homocysteine levels to folic acid deficiency. In the NHANES III examination, plasma homocysteine levels varied according to age, sex, and ethnicity. The mean plasma homocysteine level was 21.5 percent higher in men than it was in women, 11.8 percent higher in non-Hispanic whites than it was in Mexican Americans, and 42 percent higher in persons 70 years or older as compared to individuals younger than the age of 30 years. Others have suggested that Asians may have higher plasma homocysteine levels compared to other ethnicities (Chandalia M., 2003)

1.22.1. Cardiovascular Disease Risks

Rare homozygous defects of the key enzyme cystathionine--synthase cause homocystinuria, which is associated with an up to 10-fold elevation of plasma homocysteine levels and with premature atherosclerosis, recurrent thromboses of coronary, cerebral, or peripheral arteries and venous thrombosis. A meta-analysis calculated that each 5 mol/L increase in the plasma homocysteine level increases the risk for CHD by approximately 15 to 25 percent. Recently, the results of two large, randomized, double-blind secondary prevention trials, comparing treatment with vitamin B₁₂ and/or folic acid for lowering plasma homocysteine with administration of a placebo were reported, HOPE2 and NORVIT (Bonaa KH., Njolstad I) . These trials cast doubt on the causal link between homocysteine and CVD because, in both, treatment lowered

homocysteine levels, but there was no reduction in morbidity or mortality. At present, there is insufficient evidence to recommend measuring homocysteine levels in the general population. Homocysteine levels should be measured in patients with a history of premature coronary artery disease and/or stroke who do not have classic risk factors. It should also be determined in individuals with a history of venous thromboembolism (Malinow MR., 1999).



1.23. Drugs used by patient of CVD

Antihypertensive:

- a) Diuretics : eg- Furosemide , Spironolactone
- b) Sympatholytic agents : a) β - blocker : eg. Atenolol, Pindolol, Acebutolol
- c) α_1 blocker : Prazosin, Tarazosin
- d) Vasodilator : Hydrazine ,Na nitroprusside, Minoxidil, Diazoxide
- e) Calcium Channel Ca^{++} Blocker: Amlodipine, Nifedipine , Nimodipine
- f) ACE inhibitors : Captopril ,Enalapril,Ramipril, Lisinopril, Quinapril, Ramipril
- g) Central acting agent : Methyl dopa or α Methyl dopa, Clonidine

Drug used for congestive Cardiac Failure CCF:

- a) Cardiac glycoside : e.g. Digoxin, Digitoxin
- b) Adreceptor agonists : Dopamine
- c) Vasodilator: Hydrazine ,Na nitroprusside, Minoxidil, Diazoxide
- d) ACE inhibitors : Captopril ,Enalapril,Ramipril, Lisinopril
- e) Diuretics : eg- Furosemide , Spironolactone

Anti Arrhythmic Drugs:

- a) Na^{++} channel blocker :Lignocaine , Procainamide
- b) Ca^{++} Blocker: Amlodipine, Nifedipine , Nimodipine
- c) Potassium Channel blocker: Amiodarone
- d) β - blocker : eg. Atenolol, Pindolol ,Acebutolol, Propranolol

Drug used for Myocardial infarction or Anti- ischemic or Antianginal drugs:

- a) Nitrates : Nitroglycerine ,Isosorbide dinitrate , Isosorbide mononitrate
- b) Ca⁺⁺ channel blocker: Varapamil, Nifedipine
- c) Other drugs: Trimetazidine HCL
- d) Platelets inhibitors(anticoagulant) : Aspirin, Clopidogrel ,
Heparin,Warfarin.
- e) Thrombolytic agent: streptokinase, *eurokinates*

Antihyperlipidemic Drug:

Atorvastatin , Simvastatin, Lovastatin, Pravastatin Fluvastatin, Pravastatin,
Rosuvastatin ,Cholestyramine (Howland , Mycek J.Lippincott's 3rd ed)

1.24. Myocardial Infarction:

Myocardial infarction (MI) or acute myocardial infarction (AMI), commonly known as a heart attack, is the interruption of blood supply to part of the heart, causing heart cells to die. This is most commonly due to occlusion (blockage) of a coronary artery following the rupture of a vulnerable atherosclerotic plaque, which is an unstable collection of lipids (fatty acids) and white blood cells (especially macrophages) in the wall of an artery. The resulting ischemia (restriction in blood supply) and oxygen shortage, if left untreated for a sufficient period of time, can cause damage or death (infarction) of heart muscle tissue (myocardium). Classical symptoms of acute myocardial infarction include sudden chest pain (typically radiating to the left arm or left side of the neck), shortness of breath, nausea, vomiting, palpitations, sweating, and anxiety (often described as a sense of impending doom). Women may experience fewer typical symptoms than men, most commonly shortness of breath, weakness, a feeling of indigestion, and fatigue. Clinical profiles and presentations differ between women and men with AMI; Women have less typical symptoms of AMI than men (Circ J., 2006).

We believe that this approach could be further simplified by using the TC/HDL-C ratio. Because there is more cholesterol in the very LDL (VLDL) fraction in individuals with elevated TG concentrations, the LDL-C/HDL-C ratio may underestimate the magnitude of the dyslipidemic state in these patients. On that basis, we propose that the high

prevalence of moderate hypertriglyceridemia among patients with CHD explains why the TC/ HDL-C ratio was the best predictor of ischemic heart disease (IHD) risk in several observational prospective studies, including the Quebec Cardiovascular Study.(Lamarche B, 1996)However, reduction of this ratio and of the LDL-C/ HDL-C ratio in patients initially free of IHD who were treated with a lipid-lowering drug (lovastatin) was found to predict a decreased risk of a first IHD event (Grundy SM., 1997)

1.24.1. Symptoms of Myocardial Infarction:

Classical symptoms of acute myocardial infarction include sudden chest pain (typically radiating to the left arm or left side of the neck), shortness of breath, nausea, vomiting, palpitations, sweating, and anxiety (often described as a sense of impending doom). Women may experience fewer typical symptoms than men, most commonly shortness of breath, weakness, a feeling of indigestion, and fatigue. Approximately one quarter of all myocardial infarctions is silent, without chest pain or other symptoms.

- i. **Pain:** This is severe, usually retrosternal and may radiate into the jaw, shoulders and down the arms. It is described as a tight band around the chest and lasts for several hours. It is unrelieved by GTN.
- ii. **Dyspnoea:** This is due to either the pain or pulmonary congestion caused by pulmonary hypertension and pulmonary oedema. There is also an increase in the myocardial oxygen demand. If the left ventricle is affected, the cardiac output will be reduced and a shock state may exist.
- iii. **Extreme pallor:** This is due to the decreased cardiac output and redirection of blood away from the skin to the major organs. The skin will also feel cool and clammy.
- iv. **Nausea and vomiting:** This is due to pain, redirection of blood away from the gastrointestinal system, release of adrenaline and other catecholamine's into the blood and from side effects of medications used to treat the condition and symptoms.

- v. **General fatigue:** This is due to reduced cardiac output and generalized muscle ischemia. The patient will be prostrated during the attack and this may also be a cause of this.
- vi. **Tachyarrhythmia's:** This is due to the heart trying to compensate for the low cardiac output by increasing the rate (heart rate x stroke volume = cardiac output). The myocardium also becomes very irritable following infarction due to release of metabolites and electrolytes such as potassium and calcium from dying cells. This results in ventricular arrhythmias such as VT, VF and SVT.
- vii. **Hypotension:** This is due to reduced cardiac output. Initially the patient may be hypertensive due to the aggravated compensation mechanisms.
- viii. **Pyrexia:** The patient's temperature rises to around 39°C due to release of metabolites during the inflammatory process initiated by the necrotic tissue and widespread death of cells. This normally occurs over 24 – 48 hours and returns to normal within 7 days.
- ix. **Sense of Impending Doom:** This is due to the release of adrenaline and other catecholamine's as part of the compensation mechanism. Also the real fear of death exists due to the nature of the disease and the information known by the general public. It may also be that the patient is normally an anxious / highly-strung individual who worries a lot.

1.24.2. Heart Attack Causes

Over time, cholesterol buildup can occur in these blood vessels in the form of plaque. This narrows the artery and can restrict the amount of blood that can flow through it. If the artery becomes too narrow, it cannot supply enough blood to the heart muscle when it becomes stressed. Just like arm muscles that begin to hurt if you lift too much, or legs that ache when you run too fast; the heart muscle will ache if it doesn't get adequate blood supply.

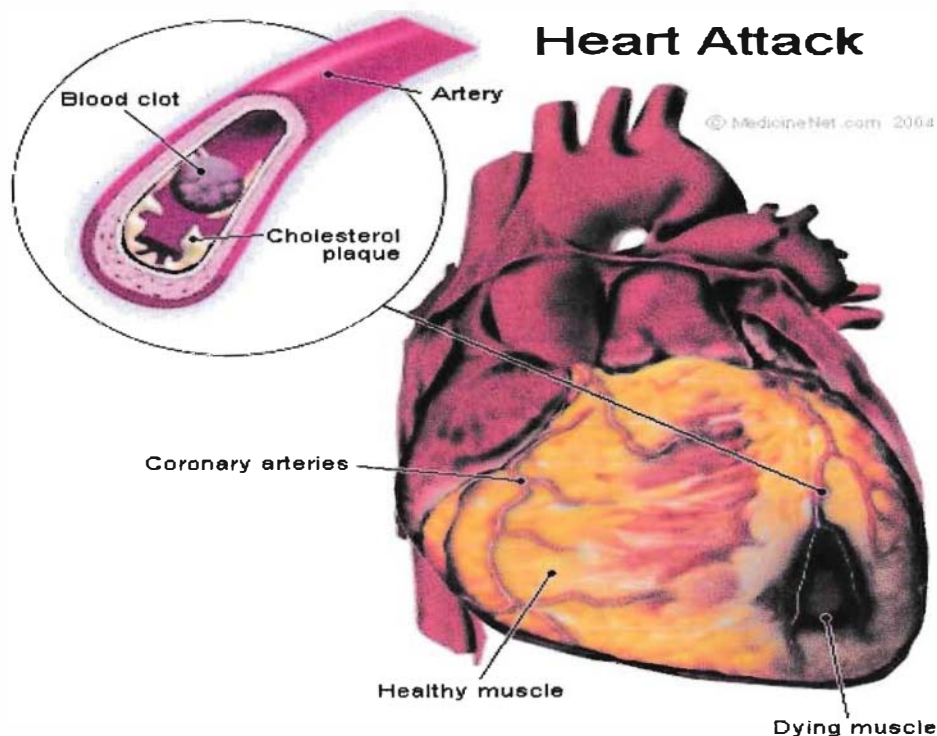


Fig 1.1: Myocardial infarction in heart.

This ache is called angina. If the plaque ruptures, a small blood clot can form within the blood vessel and acutely block the blood flow. When that part of the heart loses its blood supply completely, the muscle dies. This is called a heart attack, or an MI - a myocardial infarction (myo=muscle +cardial=heart; infarction=death due to lack of oxygen).

1.24.3. Risk factor of CVD and MI:

Heart attack is most often caused by narrowing of the arteries by cholesterol plaque and their subsequent rupture. This is known as atherosclerotic heart disease (ASHD) or coronary artery disease (CAD). The risk factors for ASHD are the same as those for stroke (cerebrovascular disease) or peripheral vascular disease:

Heart Attack Risk Factors

- Family history or heredity
- Smoking
- High blood pressure
- High cholesterol
- Diabetes



While heredity is beyond a patient's control, all other risk factors can be addressed to minimize the risk of developing coronary artery disease or decreasing its progression if already present.

Non-coronary artery disease causes of heart attack may also occur, these include:

Cocaine use: This drug can cause the coronary arteries to go into enough spasm to cause a heart attack. As well, because of the irritant effect on the heart's electrical system, cocaine can also cause fatal heart rhythms.

Prinzmetal angina or coronary artery vasospasm: Coronary arteries can go into spasm and cause angina without specific cause known as Prinzmetal angina. There can be EKG changes associated with this situation, and the diagnosis is made by heart catheterization showing normal coronary arteries that go into spasm when challenged with a medication injected in the cath lab. Approximately 2%-3% of patients with heart disease have coronary artery vasospasm.

Anomalous coronary artery: In their normal position, the coronary arteries lie on the surface of the heart. On occasion, the course of part the artery can dive into the heart muscle itself. When the heart muscle contracts, it can temporarily kink the artery and cause angina. Again, diagnosis is made by heart catheterization.

Inadequate oxygenation: Just like any other muscle, heart muscle requires adequate oxygen supply for it to work. If there isn't adequate oxygen delivery, angina and heart attack can occur. This means that an adequate number of red blood cells and normal lung function are required to deliver oxygen to the cells of the heart. Profound anemia from

bleeding or bone marrow failure can lead to lack of red blood cells. Lack of oxygen in the bloodstream can occur due to a variety of causes including respiratory failure or carbon monoxide poisoning.

1.25. Lipid profile

The lipid profile is a group of tests that are often ordered together to determine risk of coronary heart disease. They are tests that have been shown to be good indicators of whether someone is likely to have a heart attack or stroke caused by blockage of blood vessels or hardening of the arteries (atherosclerosis). (Lipid Profile January 4, 2009) . The lipid profile typically includes:

- Total cholesterol
- High density lipoprotein cholesterol (HDL-C) — often called good cholesterol
- Low density lipoprotein cholesterol (LDL-C) — often called bad cholesterol
- Triglycerides

1.25.1 LDL and MI:

Total cholesterol (TC)/high-density lipoprotein cholesterol (HDL-C) and low-density lipoprotein cholesterol (LDL-C)/HDL-C ratios are used to predict ischemic heart disease risk. Variation in the TC/HDL-C ratio may be associated with more substantial alterations in metabolic indices predictive of ischemic heart disease risk and related to the insulin resistance syndrome than variation in the LDL-C/HDL-C ratio. DESPITE considerable advances during the past 40 years, there is increasing awareness among scientists, epidemiologists, and clinicians that current approaches to evaluation of coronary heart disease (CHD) risk in asymptomatic individuals remain suboptimal. (Superko HR Curr). LDL-C/HDL-C ratio combined with elevated TG is associated with High CHDrisk. This dyslipidemic state (lipid triad) has been described as atherogenic dyslipidemia (Grundy S., 1997).

1.26. Diagnostic of Myocardial infraction:

EKGs, blood tests, and chest x-ray are other tests that are likely to be performed to assist with the diagnosis.

1.26.1. Electrocardiogram

The electrocardiogram (ECG or EKG) will help direct what happens acutely in the ER. The EKG measures electrical activity and conduction in heart muscle. In a heart attack in which the full thickness of the heart muscle is involved, the EKG shows characteristic changes that establish the diagnosis of a myocardial infarction. Some heart attacks only involve small parts of the heart muscle; in these cases, the EKG can look relatively normal.

1.26.2. Blood tests

If the EKG does not diagnose a heart attack, blood testing may be required to further look for heart damage. An EKG can be normal even in the presence of a heart attack. This is done with blood tests that can measure chemicals that leak out of irritated heart muscle cells and can be measured in the blood. Levels of the cardiac enzymes myoglobin, CPK, and troponin are often measured, alone or in combination, to assess whether heart muscle damage has occurred. Unfortunately, it takes time for these chemicals to accumulate in the blood stream after the heart muscle has been insulted. Blood samples need to be drawn at the appropriate time so that the results can be usefully interpreted. For example, the recommendation for the troponin blood test is to draw a first sample at the time the patient presents to the ER and then a second sample 6-12 hours later. Usually it requires two negative samples to confirm that no heart muscle damage has occurred.

1.26.3. Chest X-ray

A chest x-ray may be taken to look for a variety of findings including the shape of the heart, the width of the aorta, and the clarity of the lung fields.

If a heart attack has been proven not to have occurred, further evaluation of the heart may be undertaken using stress tests, echocardiography, or heart catheterization.

1.27. Treatment of myocardial infarction

If the EKG shows that there is an acute heart attack (myocardial infarction), then the goal is to open the blocked artery as soon as possible and restore blood supply to the heart muscle. When a heart attack strikes, the key thing to remember is that time equals muscle. The longer the delay in seeking medical care, the more heart muscle will be damaged. There is a window of opportunity to restore blood supply to the heart muscle by unblocking the affected heart artery. Treatments must be done in a hospital and include administration of clot-busting drugs to dissolve the clot at the site of the ruptured plaque and heart catheterization and angioplasty (in which the blood vessel is opened by balloon, often with adjunctive placement of a stent), or both. Sometimes special procedures, such as bypass surgery and angioplasty, were used to treat heart disease. Heart disease and its risk factors must be controlled to prevent MI (**American Heart association., 2000**).

Heart catheterization

The favored treatment is heart catheterization. Tubes are threaded through the femoral artery in the groin or through the brachial artery in the elbow, into the coronary arteries, and the area of blockage is identified.

Angioplasty

Angioplasty (angio= artery + plasty=repair) is then considered if possible. A balloon is placed at the blockage site and as it opens, it squashes the plaque into the blood vessel wall. Afterwards, a stent or a mesh cage is placed across the angioplasty site to keep it from closing down. Guidelines recommend that the time from the time the patient presents to the hospital to having the blood vessel open be less than 90 minutes.

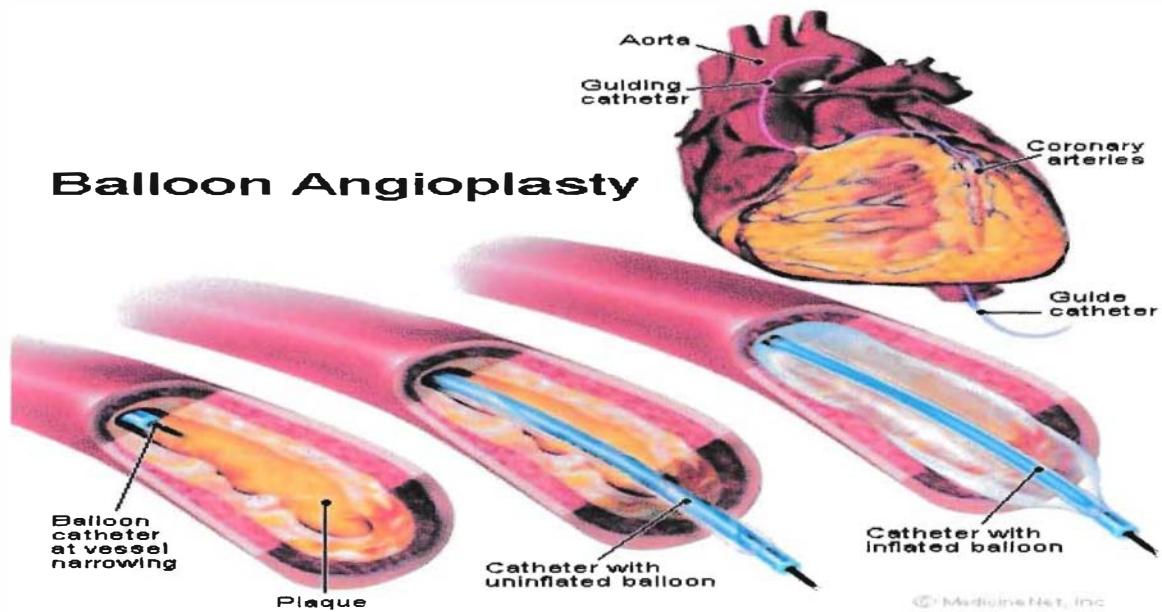


Fig 1.2. : Angioplasty

Not all hospitals have the capabilities of doing heart catheterizations 24 hours a day, and may transfer the patient with an acute heart attack to a hospital that has that technology available. If the transfer time will delay angioplasty treatment beyond the 90 minute window recommendation, clot-busting drugs may be considered to dissolve the blood clot that has obstructed the coronary artery. Tissue plasminogen activator (TPA or TNK) can be used intravenously. After TPA infusion, the patient may still be transferred for heart catheterization and further care. If the EKG is normal but the history is suggestive of an heart attack or angina, the evaluation will continue with the blood tests described above. However, the patient will likely be treated as if the heart attack was happening with aspirin, oxygen, nitroglycerin and blood thinning medications until the presence of heart damage is proven not to be present.



Chapter 2

Aim and Significance

2.1 Aim and objective of the study

To give overall idea about cardiovascular disease and global burden of CVD and especially over myocardium infraction and its risk factor specially focus on lipid profile relationship with myocardium infraction patient. High lipid profile characteristics are one of the major risks of myocardial infarction that can modify, treat or control by changing the lifestyle or taking medicine. The risks of occurring myocardial infarction are greater if blood lipid profile is not well controlled. All the factors keeping in mind the present was conducted to find out the lipid profile and other risk factors related to MI.

2.2. Significance of the study

Plasma lipids consist mostly of lipoprotein-spherical macromolecular complexes of lipids and specific protein (Apolipoprotein). The clinically important lipoproteins, listed in decreasing order of atherogenicity, are LDL, Very Low Density Lipoprotein (VLDL) and chylomicrons and HDL. The occurrence of CHD is positively associated with high total cholesterol and even more strongly with elevated LDL cholesterol in the blood. The prevalence of myocardial infarction is increasing day by day. Hyperlipidemic patients have a higher prevalence of myocardial infarction compared to the normal population. Long term duration of uncontrolled blood lipid may cause various disorders, mainly involving small vessels. The disorders include retinopathy, nephropathy and neuropathy which ultimately lead to visual disturbance, renal failure and gangrene. High blood lipid accelerates and exacerbates the occurrence of arteriosclerosis, increasing the risk of myocardial infarction, cerebral infarction and occlusive artery disease of the lower extremities. The morbidity and mortality of these patients are due to these complications. Globally, non-communicable diseases (NCDs) are increasingly recognized as a major cause of morbidity and mortality. Coronary Heart Disease is one of the non-communicable diseases. Myocardial Infarction is one kind of coronary heart disease. hyperlipidemic is the major risk factor of Myocardial Infarction among other risk factors which can modify, treat or control by changing lifestyle or taking medicine. This study will be help to increase the awareness between people health by taking immediate treatment, by taking drug, or by controlling blood sugar level, food habit, physical activity to avoid the harmful effect of myocardial infarction. This study is expected to

Study of lipid profile in patients with myocardial infarction

provide important information to better understand the relationship between the hyperlipidemic and Myocardial Infarction. Thus, the result of the study is expected to improve management of Myocardial Infarction in patients with hyperlipidemic which ultimately will help to improve the disease management process.



Chapter 3

Material and Method

3.1. Type of study:

It is a case control study. It was attempted to establish relationship between Myocardial Infarction and lipid profile. In addition to this, the study examined for other risk factors and presence of MI.

3.2. Place of study:

The study was be conducted in National Institute of Cardiovascular Diseases & Hospital (NICVD). This hospital is the largest and the pioneer cardiac hospital in Bangladesh .It was established in 1981, situated at the heart of the Dhaka city composed of 400 beds, offering 24 hours of services. This institute comprises of Outdoor, Emergency, highly specialized Coronary care unite, Post coronary care unit Intensive care unite and has a full fledged indoor. A good number of Doctors and medical specialists and other supporting staffs are providing cardiac medical and surgical care services to all categories of patients from different parts of the country- including referred patients from other medical college hospitals & district hospitals.

3.3. Study population:

All admitted patient of MI diagnosed by the hospital physicians.

3.3.1 Inclusion Criteria of the cases:

- i). Patient of diagnosed MI ages 25-85yers
- ii) Both sexes irrespective of religion and occupation.

3.3.2 Exclusion Criteria of the cases:

- i) Patients of cardiac disease other than Myocardial Infarction.
- ii) Post operative patient
- iii) Any other chronic diseases.

3.4. Sample size:

The objective of the study is to find out the relationship between lipid profile and myocardial infarction. The sample sizes were 135 patients.

3.5. Sampling Technique:

In this study, purposive sampling technique was followed. All the cases were fulfilled above the selection criteria were incorporated, 135 cases were achieved.

3.6. Research Approach:

After getting the approval of the research proposal from the honorable faculty members, formal permission was obtained from the competent authorities of NICVD. The data were collected from the wards 3, 4, 5, 6, and 7, (Medicine Ward).

3.7. Research Equipments:

The subsequent equipments were used in this stud

- I). Interview schedule
- II). Measuring Tape.
- III). Weighing machine (Bathroom Scale)
- IV) Sphygmomanometer. (Aneroid type)
- V). Stethoscope.

3.8. Data collection method:

After explaining the purpose of the study to the respondents and obtaining their verbal consent, the researcher interviewed all the respondents by asking question in Bengali and using a thoroughly pre –tested questionnaires the questionnaires was be consists of three parts. Part -1 was consists of the respondents general information, part-2 behavioral characteristics and Part-3 was consists of Physical examination e.g. recording blood pressure and anthropometrical measurements examination by checklist, clinical examination and laboratory tests.

3.8.1. Blood pressure Measurement:

Measurement of blood pressure was made on each study participant with an aneroid type of sphygmomanometer using a standardized technique.

3.9. Diagnosis of Myocardial Infarction patients:

This study was performed on 135 consecutive patients of acute Myocardial Infarction (AMI) admitted to the Department of cardiology, NICVD, for treatment and irrespective of age and Sex. All patients of acute anterior, inferior both anterior and inferior, and right ventricular infarction with inferior were included in the study. Patients were diagnosed on the basis of following criteria:

1. Chest pains that characteristic of AMI and OMI.
2. Increased level of cardiac enzymes in serum. Creatine kinase (CK)

3.9.1. Treatment

1. Bed rest
2. Sedative
3. Beta- blocker
4. Anti coagulant drug
5. Anti ulcer drug
6. Inhalers
7. Injections
8. Pain killer or anesthetic

3.10. Study period

Study period was one year instigation from July 2009 to May 2010. To complete the study in time a work schedule is prepared depending on different tasks of the study. The four months were spent on board meeting for literature review, selection of topic, development of the protocol. Subsequent months spent on official correspondence, data collection, data analysis, report writing and submission of report.

3.10.1 Data Processing:

Appropriate template was prepared basing on the variable and data were entered. After the entry was being made data were analyzed by using Microsoft office excel version 2003 and 2010. The result was shown in bar, pie chart and calculate the percentage the different risk factors of MI patients.

3.10.2 Data analysis:

All the data were checked after collection. Then data was entered into excel, and count the number of common risk factor people and percentage are calculated help of software Microsoft excel windows programmed version 2003, 2010. The result was shown in bar, pie chart and calculate the percentage the different risk factors of MI patients.



Chapter 4

RESULT

Result: All result was shown by bar and pie, chart calculate the percentage the different risk factors of MI patients.

4.1. Distribution (%) of patient's myocardial infarction according to the gender. Patient patient were studied 130 male 96% and 5 female 4% patient having myocardial infarctio

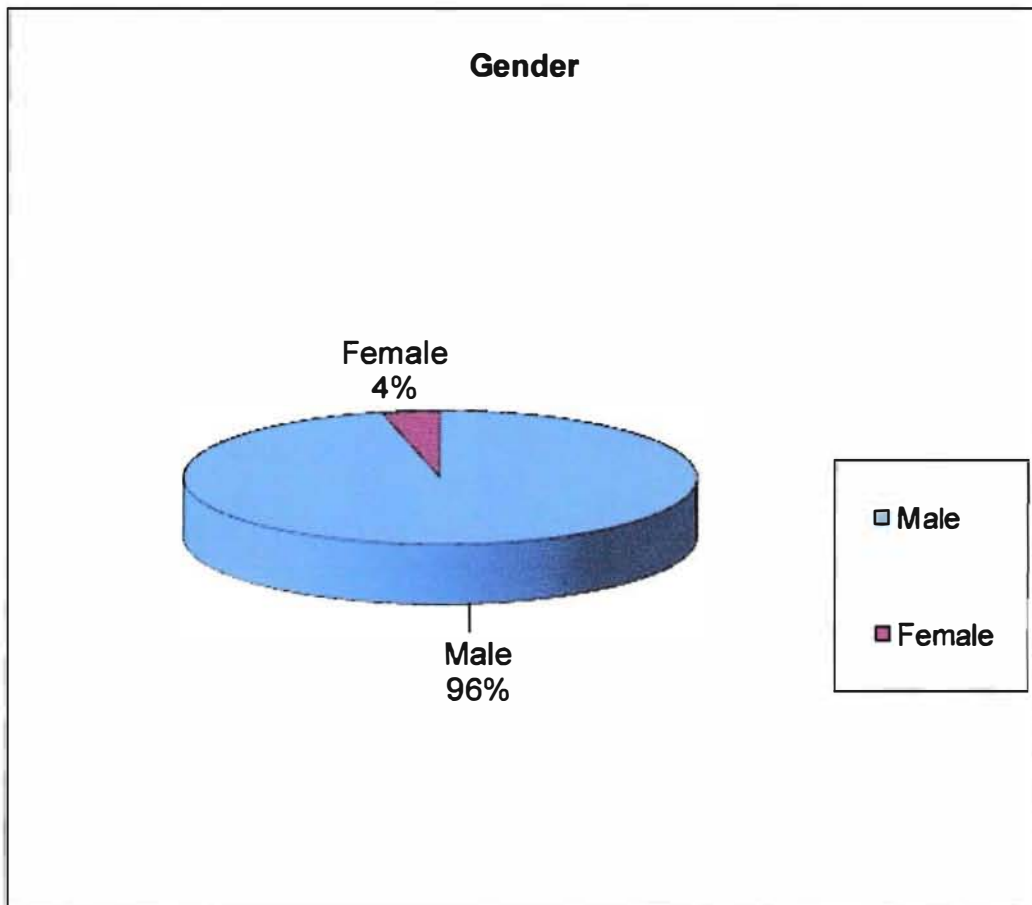


Fig.1.3 Distribution (%) of myocardial infarction among male and female patients

4.2. Distribution Myocardial Infarction among patient Depending on Age :

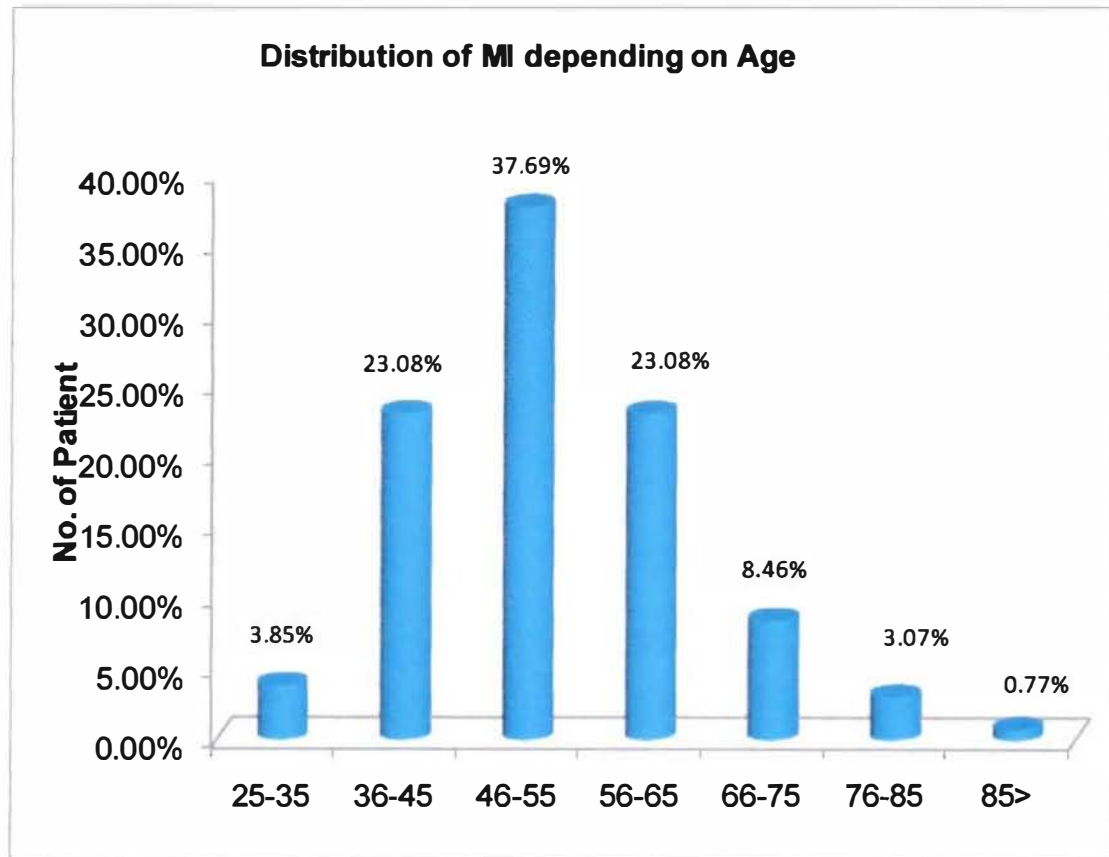


Fig.1.4. Distribution Myocardial Infarction among patient Depending on Age

The distribution of patients aged from 25-95 years old is shown in chart. But myocardial infarction is more prevalent in the age range from 46-55 years old patients.

4.3. Distribution (%) of myocardial infarction among different religions

Result Showed that Muslim and Non-Muslim patients with myocardial infarction. Islam 123 (91.11%) Hindu 11 (8.15%) Christian 1 (0.74 %).

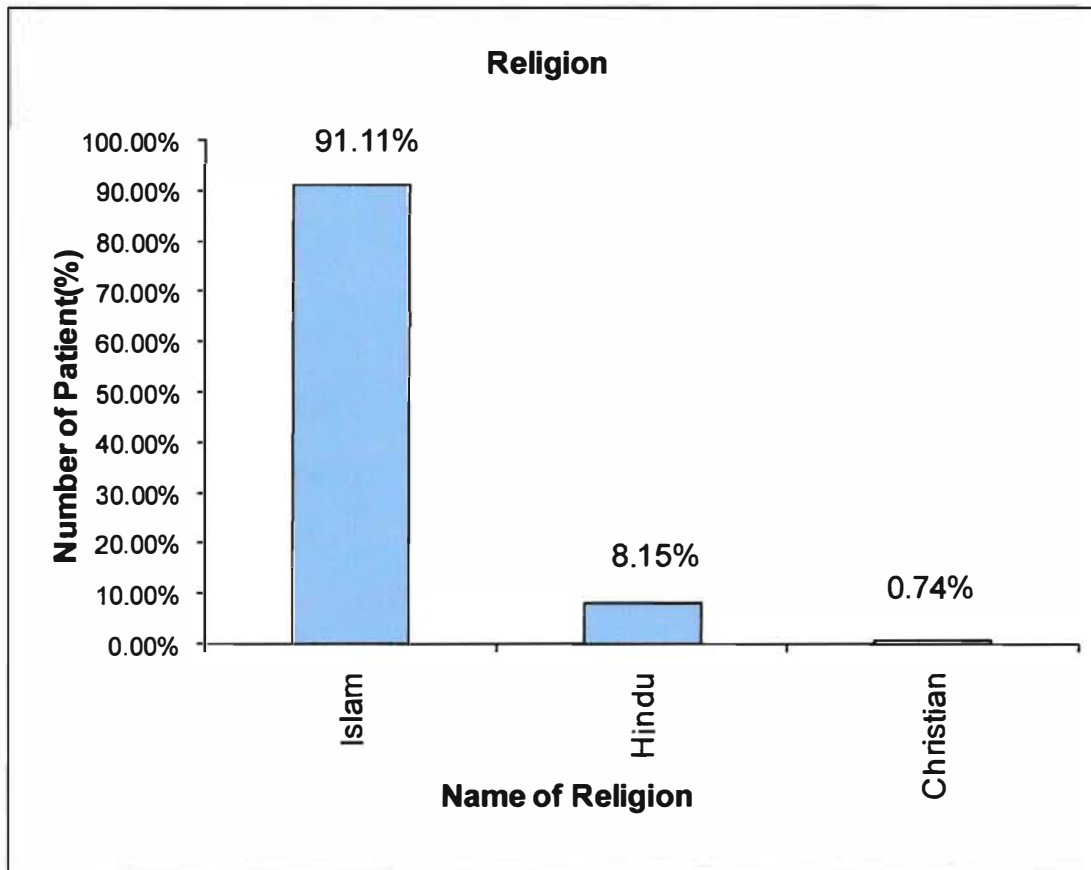


Fig.1.5 Shows different religion of patients with myocardial infarction.

4.4. Distribution Myocardial Infarction among Smoker & Non Smoker

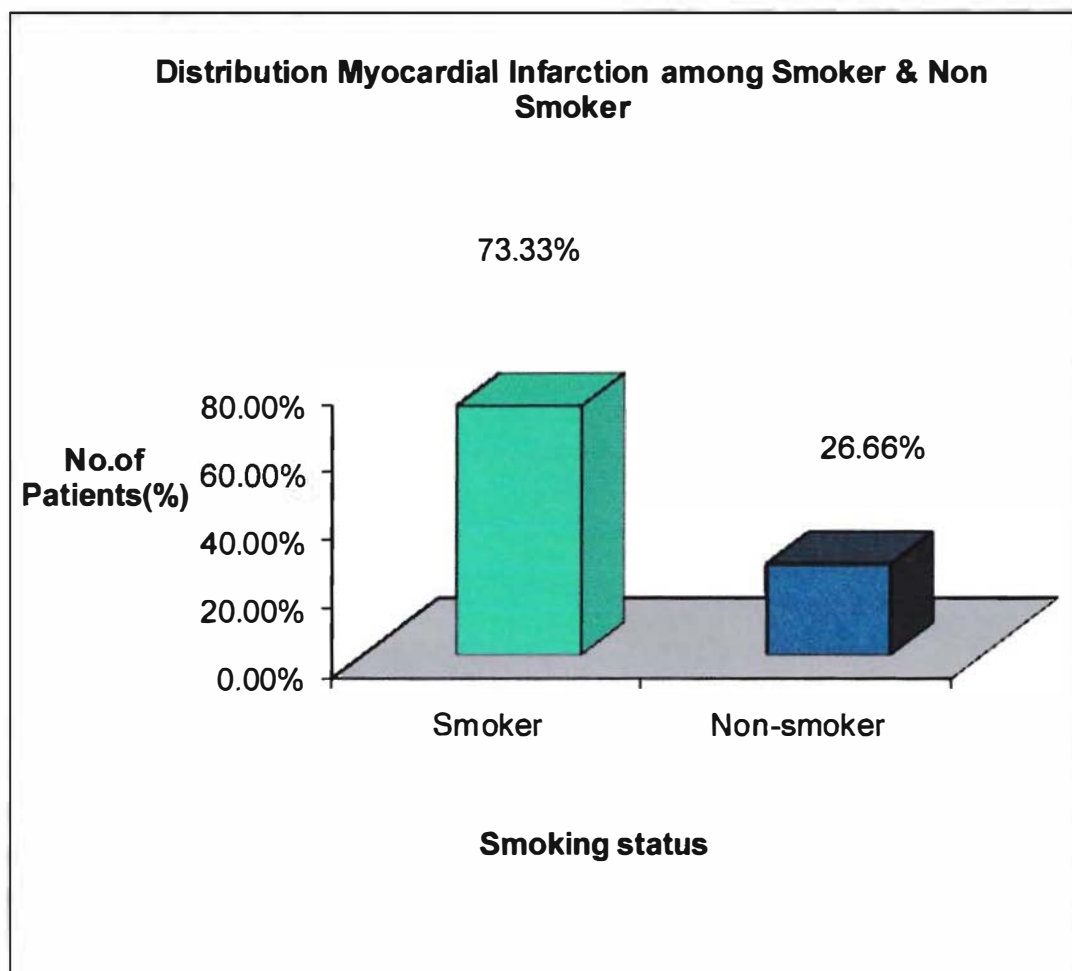


Fig 1.6. Distribution (%) of myocardial infarction in patients with smoking

4.5. Distribution Myocardial Infarction among Random Blood Sugar level (RBS) :35.55 % of patient are abnormal and 11.11 % are normal value of RBS.

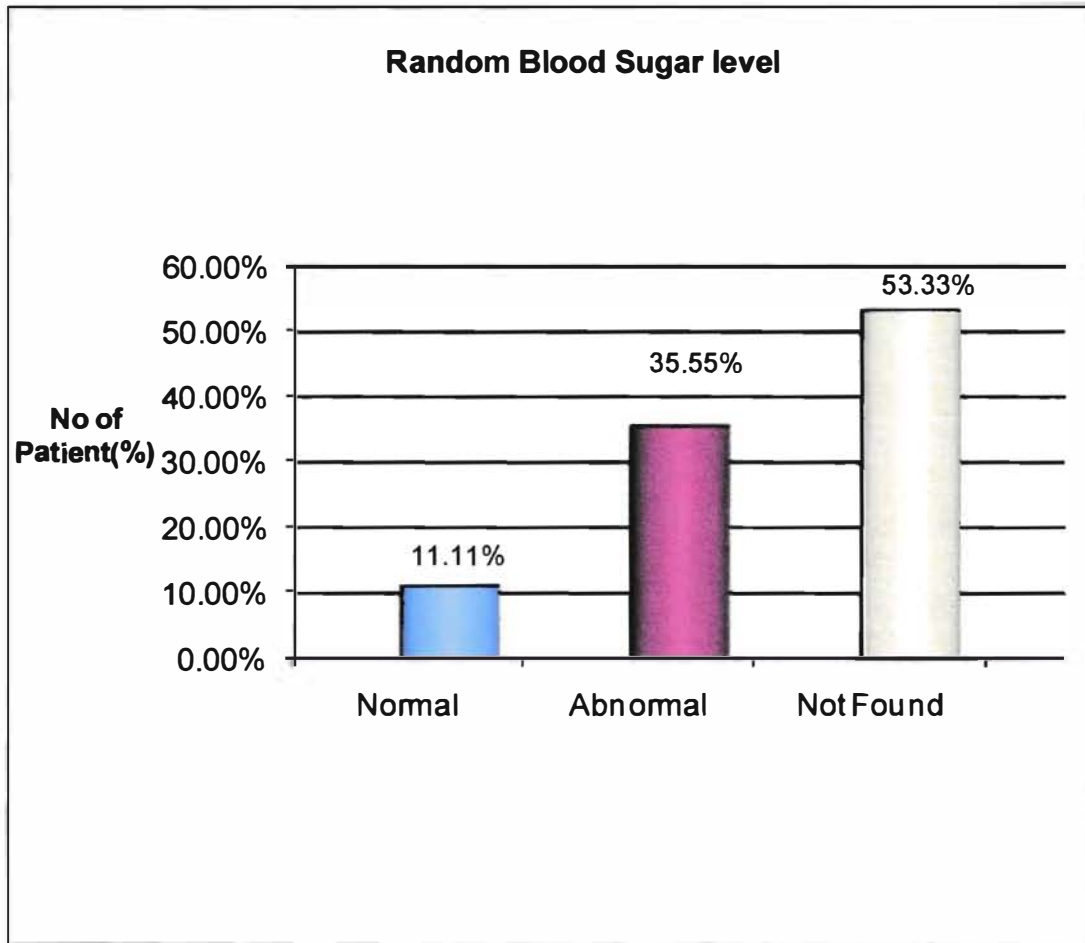


Fig1.7. Distribution Myocardial Infarction among Random Blood Sugar level (RBS)

4.6. Distribution Myocardial Infarction among Hypertensive and non

Hypertensive patient: 80 patient has High BP and 54 patient Non High BP.

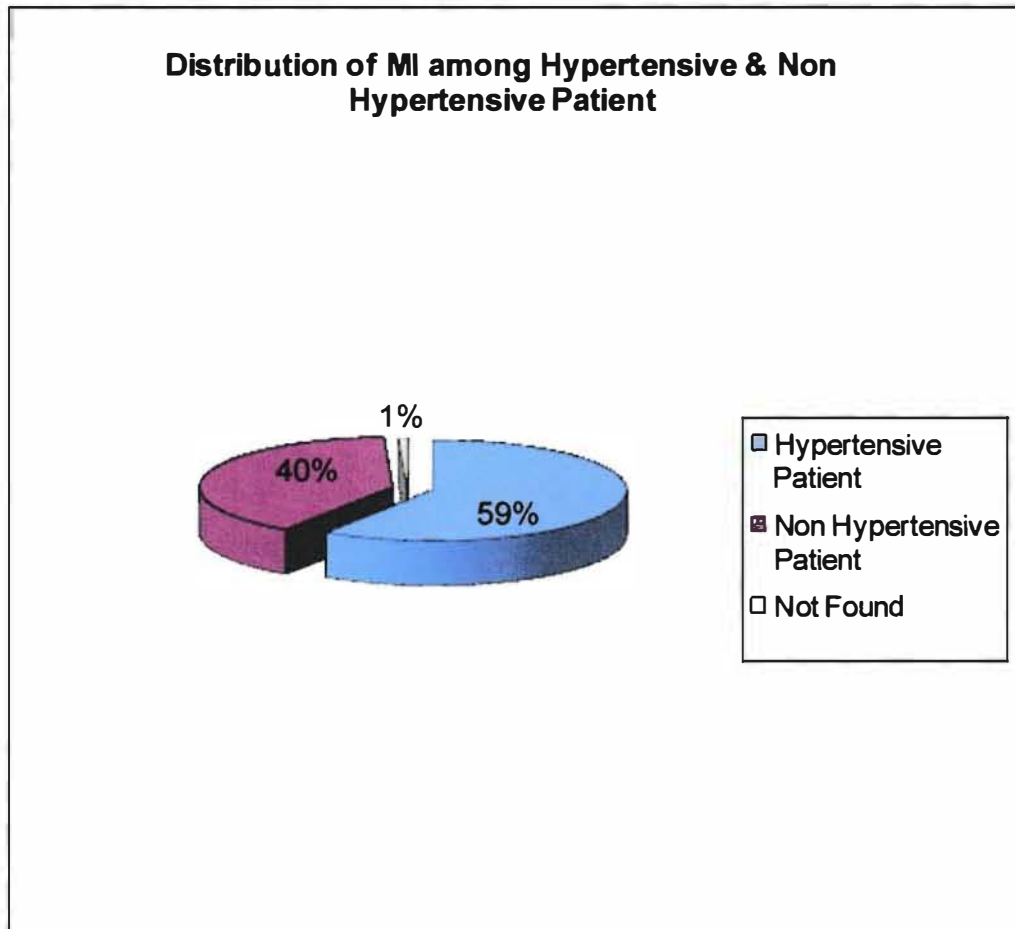


Fig1.8. Distribution(%) Myocardial Infarction among Hypertensive and non Hypertensive patient

4. 7. Distribution Myocardial Infarction among Percentage of MI Patient Taking Salt : 90 (67 %) patient taking extra table salt ant 45 (33%) patient are not .

Percentage of MI Patient Taking Salt

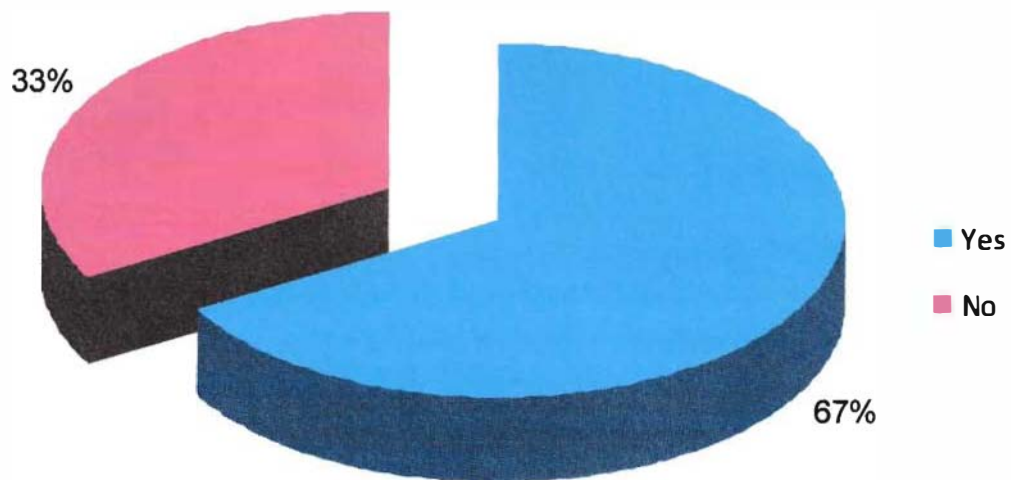


Fig1.9. Distribution Myocardial Infarction among Percentage of MI Patient Taking Salt

4.8. Distribution Myocardial Infarction among Percentage of MI Patient taking betel nut : 59 (43.70%) patient chew betel nut salt ant 76 (56.30%) patient are not.

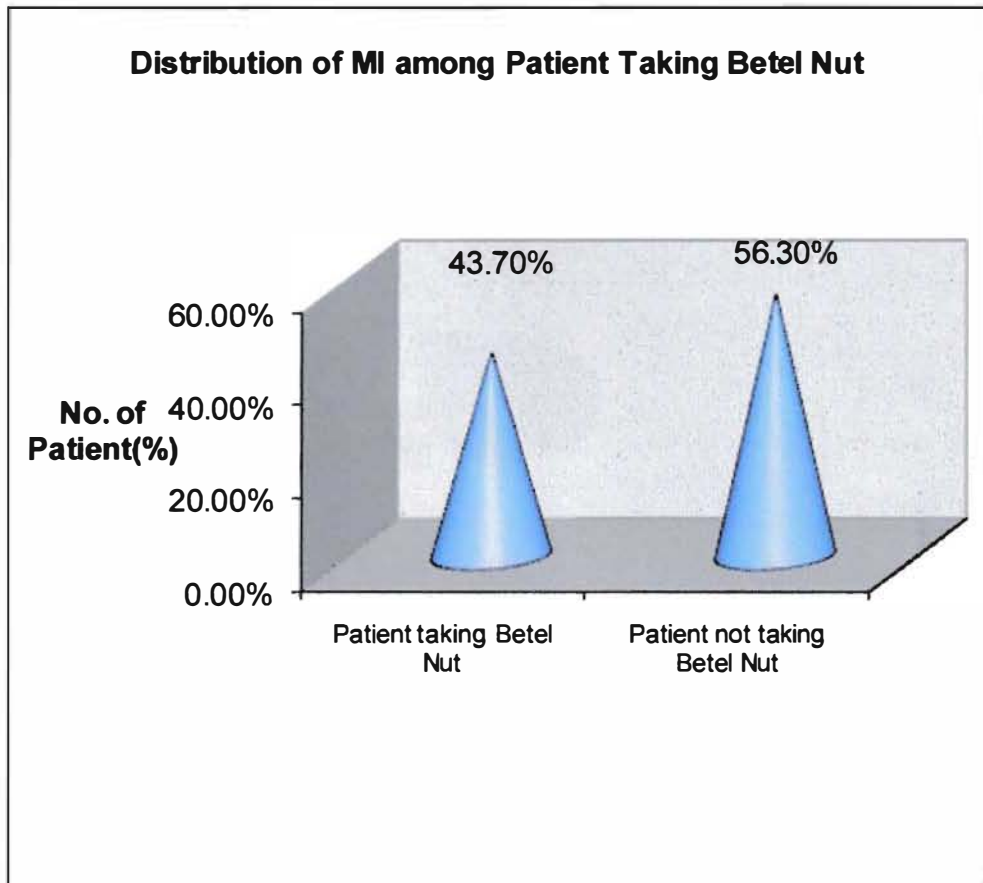


Fig1.10. Distribution Myocardial Infarction among Percentage of MI Patient taking betel nut.

4.9 Distribution Myocardial Infarction among Percentage of different type of MI: 64 patient (47 %) patient AMI, 59 patient (44 %) OMI and 12 patient patient data are not found

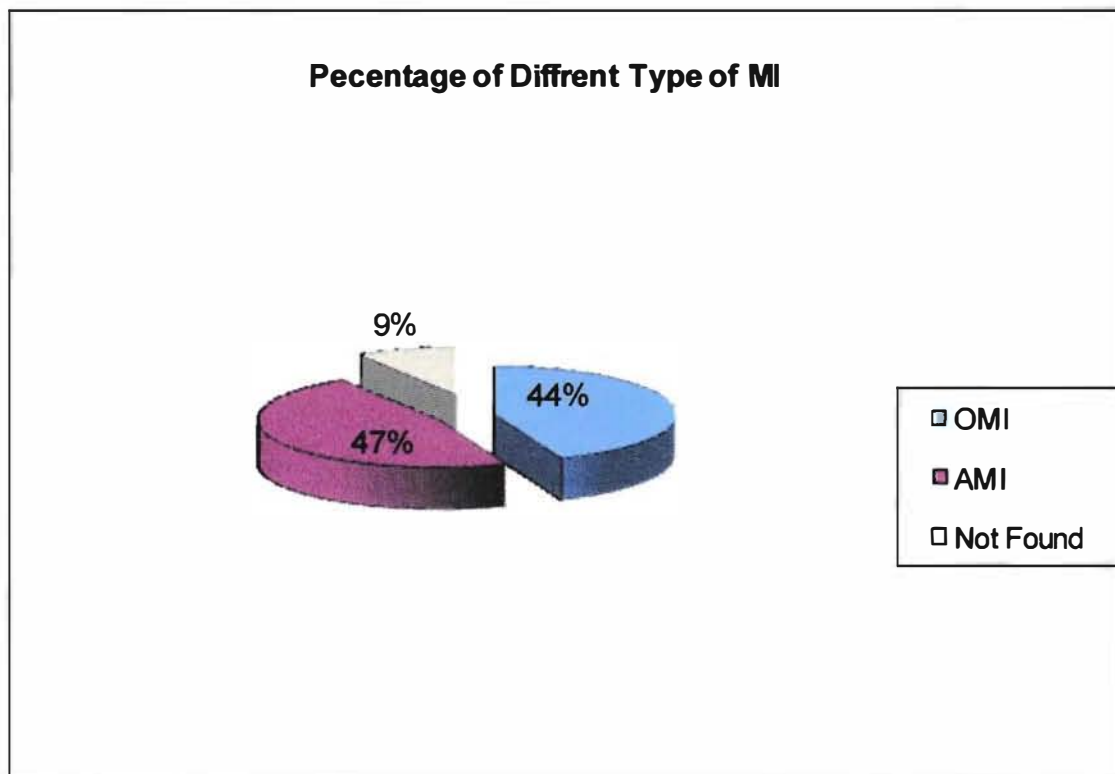


Fig 1.11 Distribution (%) of types of myocardial infarction

4.10. Distribution Myocardial Infarction among patient depending on creatinine: Normal 43 patient (31.85) Abnormal : 6 patient (4.44 %) patient ,87 patient (64.44%) patient data were not found .

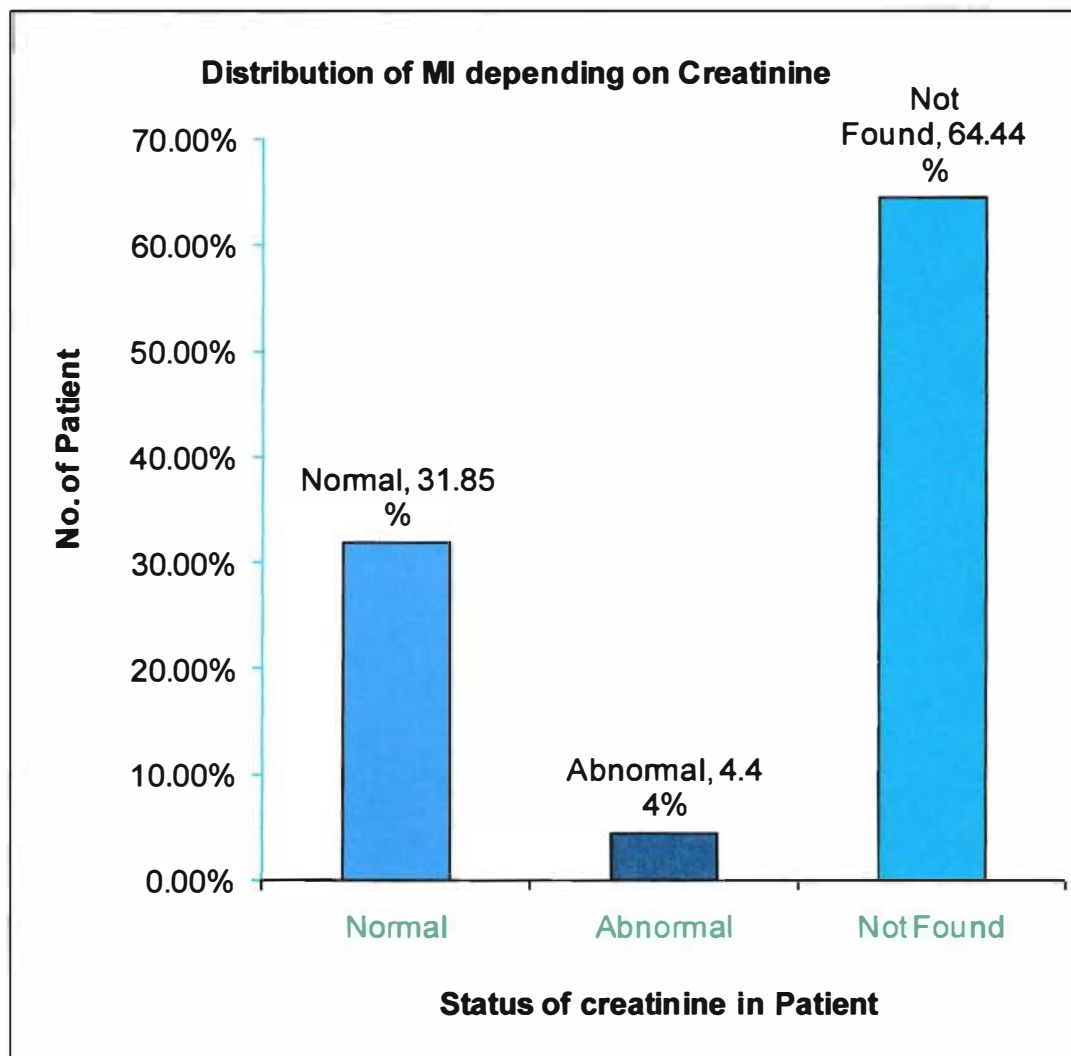


Fig1.12. Distribution Myocardial Infarction among patient depending on creatinine

4.11. Distribution Myocardial Infarction among patient deepending Depending on Taking Tea or Cofee:

Taking tea or cofee 79 patient (58.52 %) and 56 patient (41.48 %) patient not taking tea or cofee .

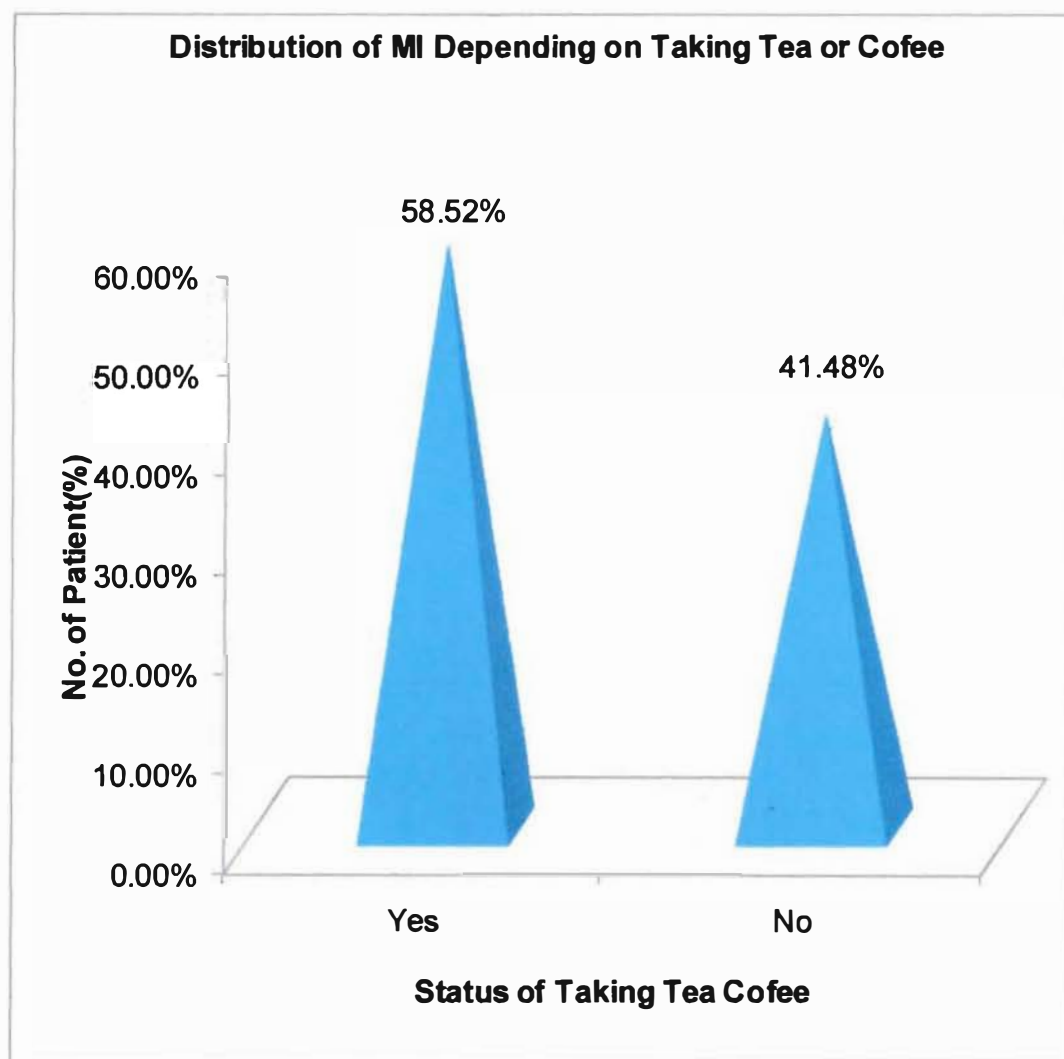


Fig 1.13. Distribution Myocardial Infarction among patient deepending onTaking Tea or Cofee

4.12. Distribution Myocardial Infarction among patient depending on Concentration of K⁺:

K⁺ level were normal range 47 patient (34.81 %) , 14 patient (10.37%) were abnormal and 74 no of patient data not found .

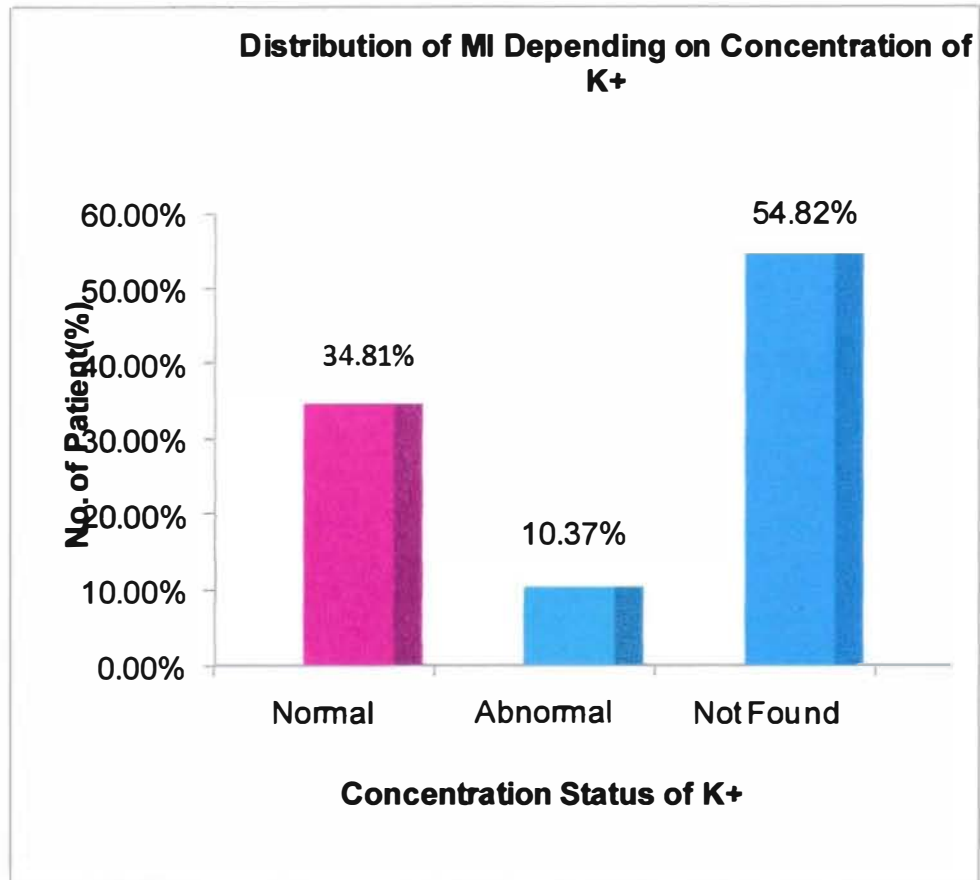


Fig 1.14. Distribution Myocardial Infarction among patient depending Concentration of K⁺

**4.13. Distribution Myocardial Infarction among patient depending
Depending on Concentration of Na^+ level :**

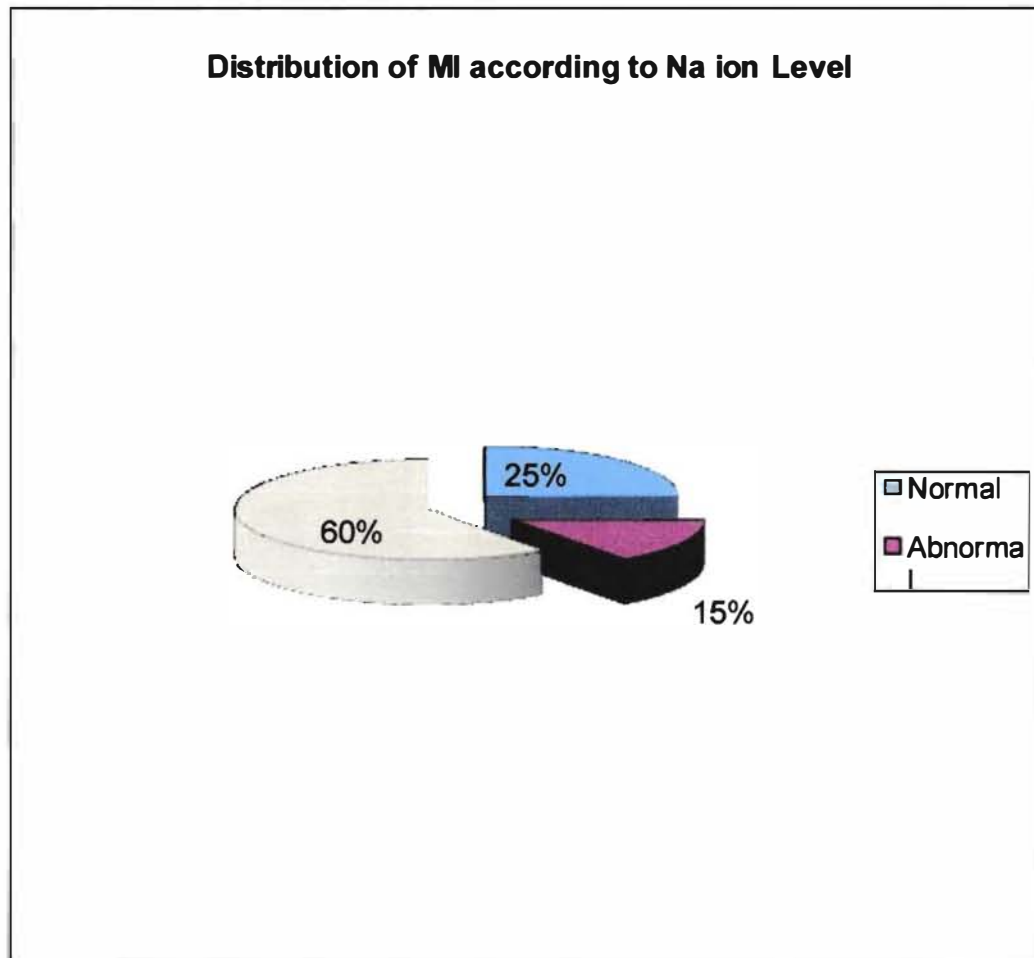


Fig 1.15. Distribution Myocardial Infarction among patient depending on Concentration of Na^+ level

4.14. Distribution Myocardial Infarction among patient Depending on Occupation: 12.59 % farmer , 29.63 % bussnessmen , 29.63 % service 5.93 %pension holder , 4.44% unemployee ,3.70 %house wife and others 11.85%

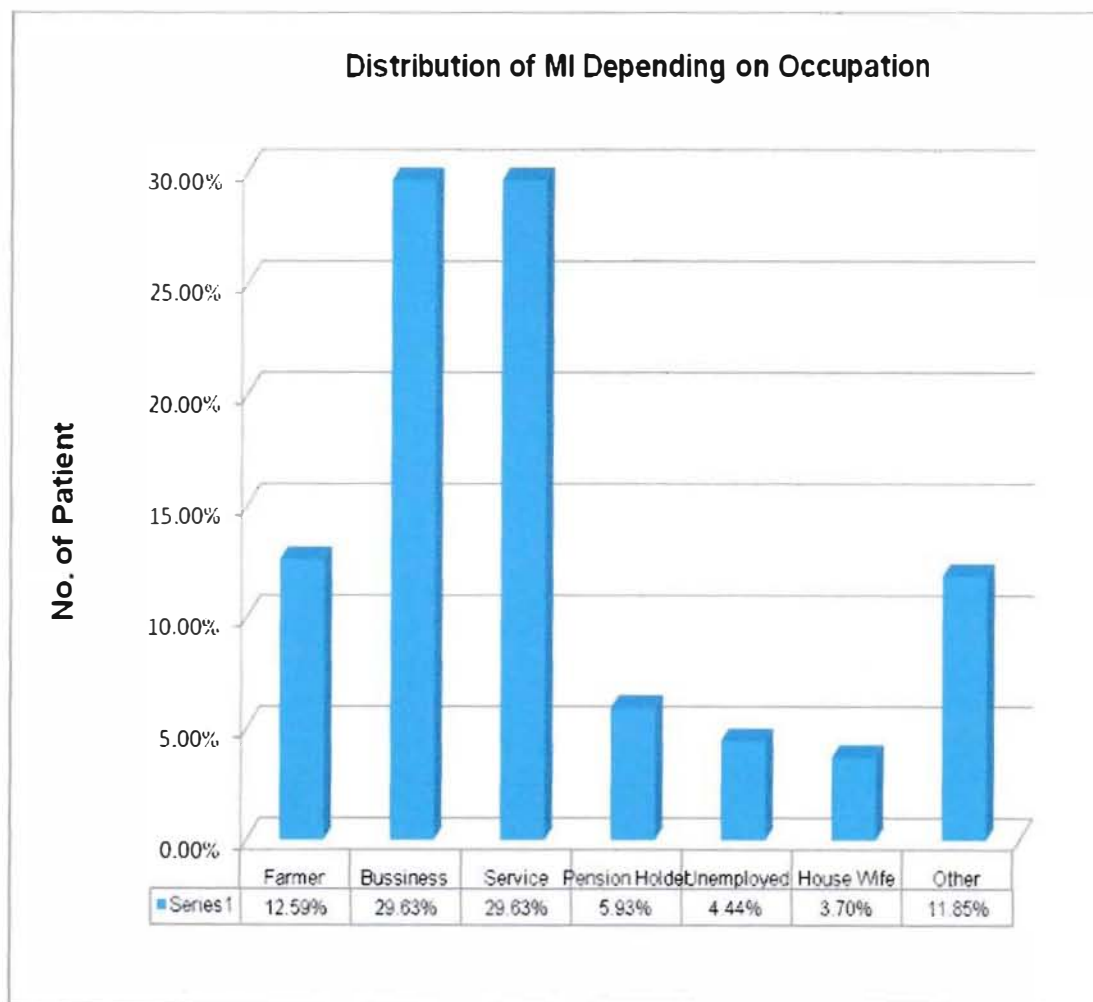


Fig 1.16. Distribution Myocardial Infarction among patient Depending on Occupation

4.15. Result : Distribution Myocardial Infarction among patient Depending on Income :

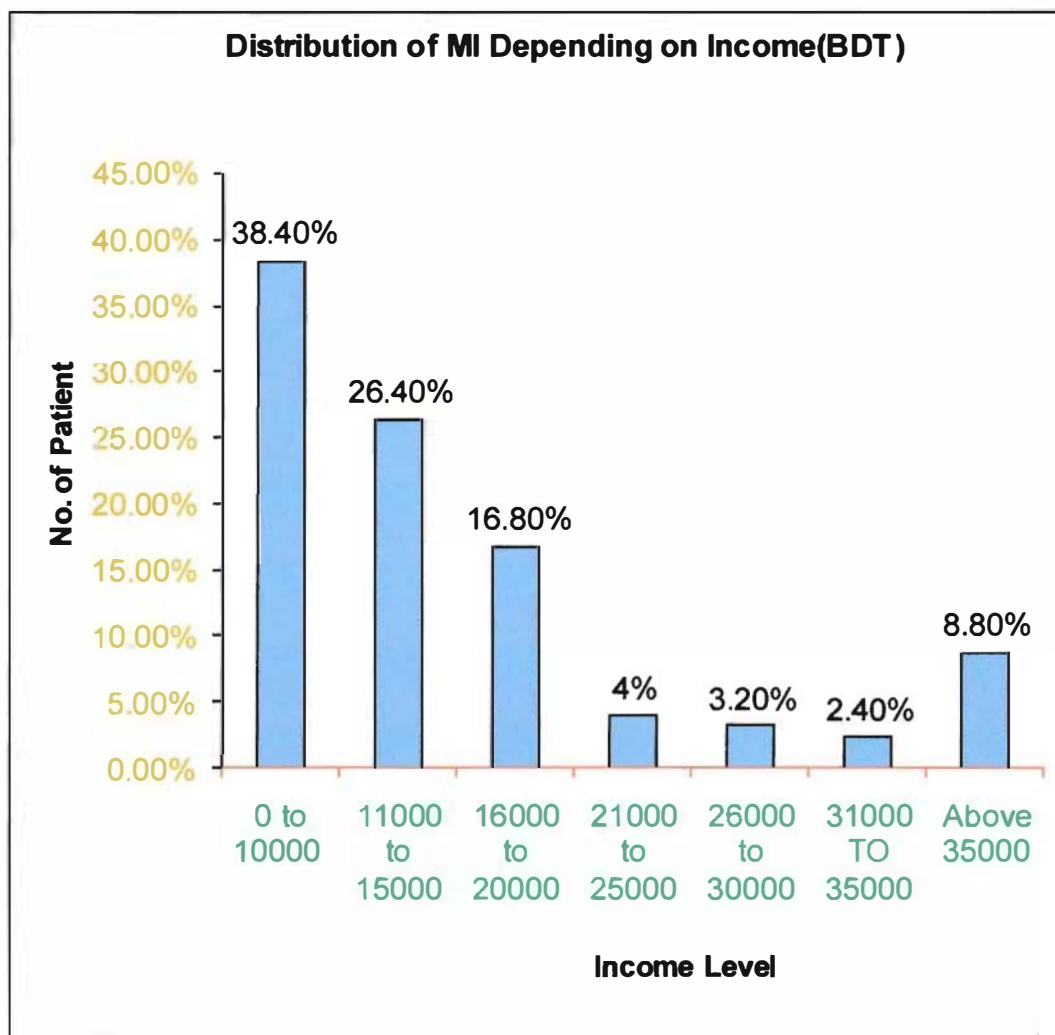


Fig 1.17 Distribution Myocardial Infarction among patient Depending on Income

4.16 . Distribution Myocardial Infarction among patient Depending upon oil use :

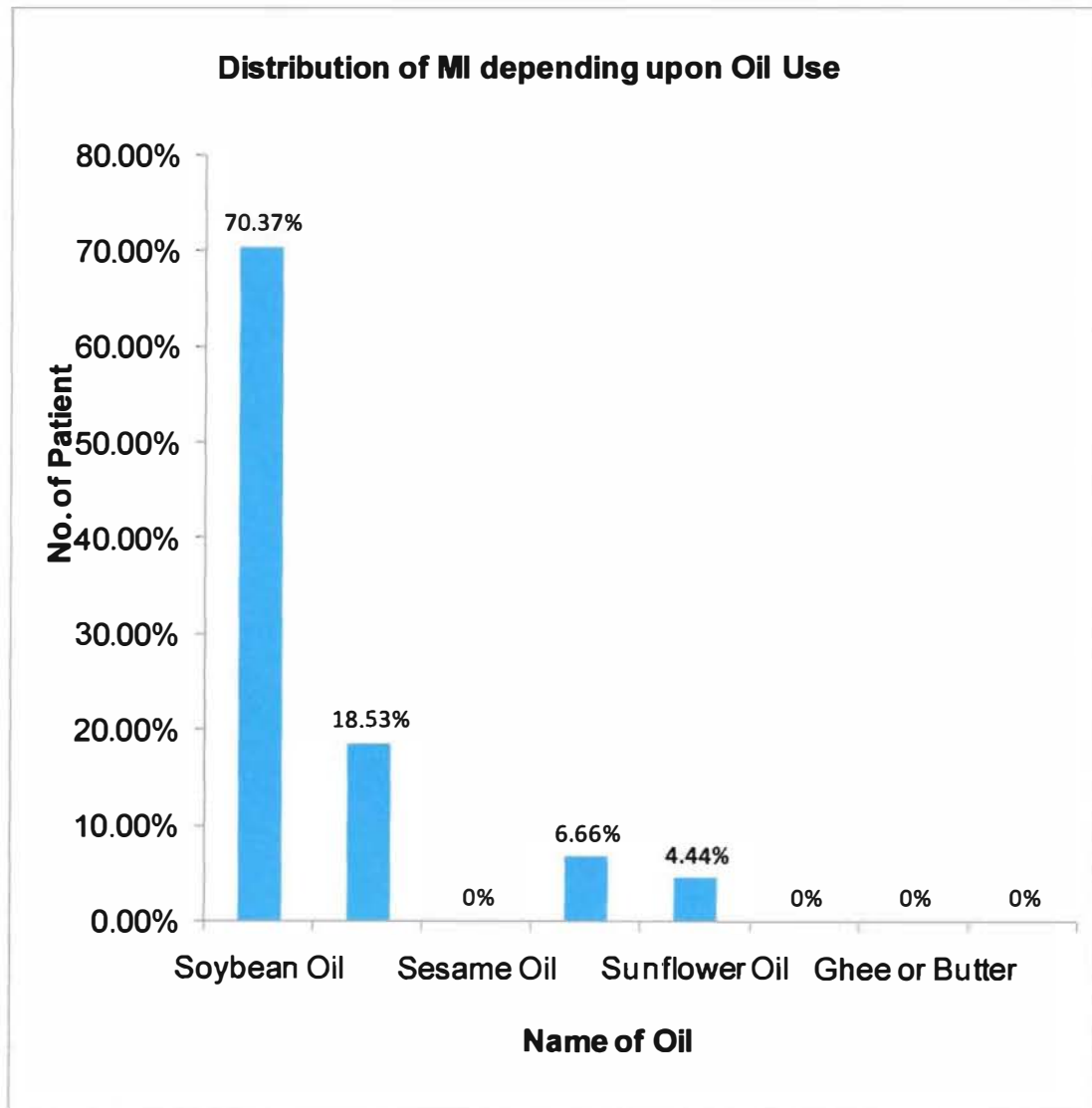


Fig 1.18: Distribution (%) of myocardial infarction depending upon Oil use.

4.17. Distribution Myocardial Infarction among patient Depending upon BP systolic :

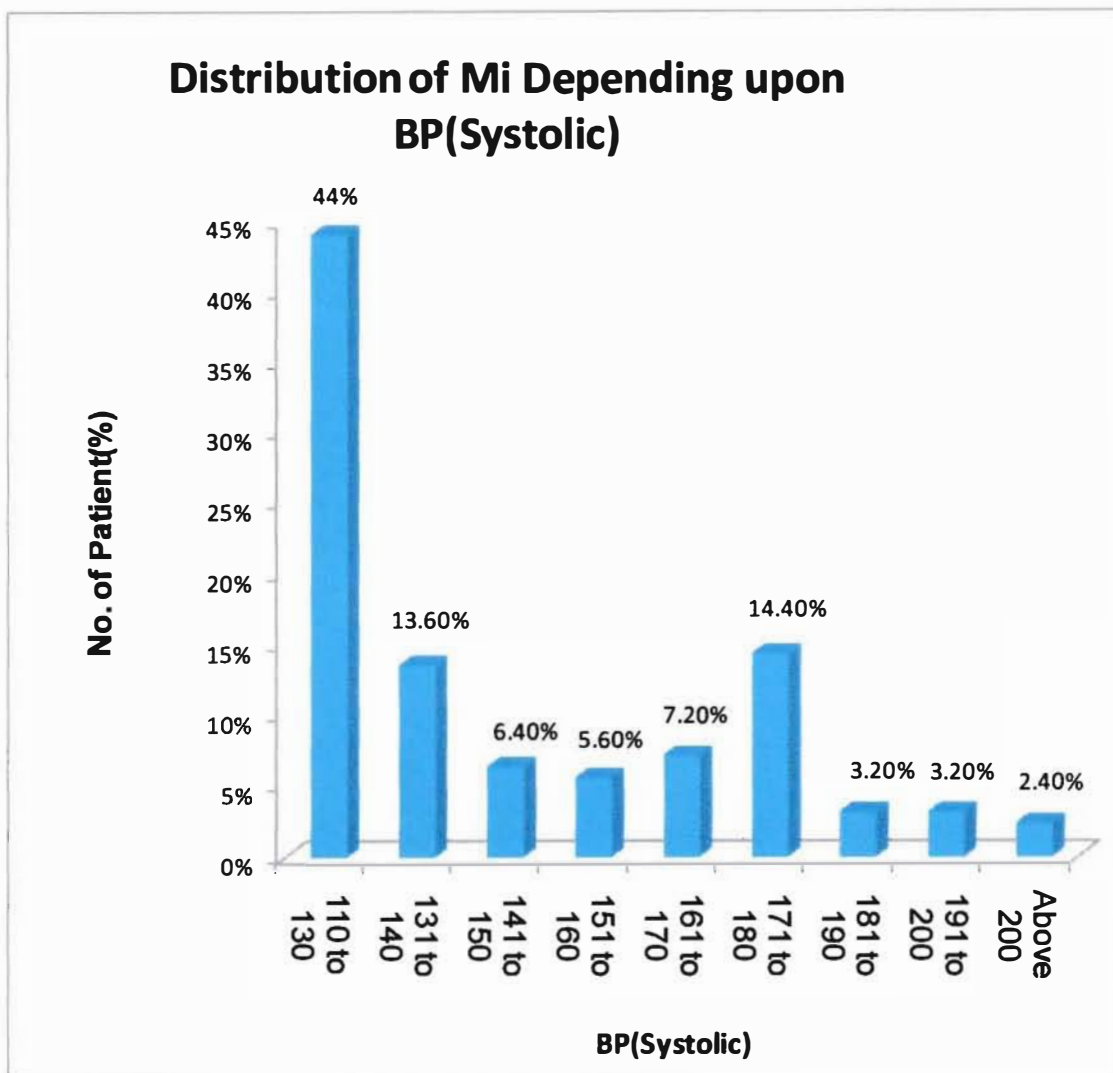


Fig 1.19 Distribution (%) of myocardial infarction depending upon BP (systolic)

5.1.1 Distribution of Cholesterol level in MI patient

Table 1.5. Distribution of Total Cholesterol among 135 MI Patients:

Total Cholesterol mg/dl			
Test	Range in mg/dl	Number of Patient	%
Optimal	<200	28	21.4815
Near Optimal/Above Optimal	---		0
Borderline to High	200-239	67	50.3704
High	>240	40	29.6296
Very High			0
Total		135	

5.1.2. Distribution of HDL level among the MI Patients

Table 1.6. Distribution of HDL among the MI Patients

Total HDL mg/dl			
Test	Range in mg/dl	Number of Patient	%
Optimal	> 36	9	6.667
Near Optimal/Above Optimal	38-37	45	33.33
Borderline to High	39-40	51	11.3333
High	41-42	29	21.4815
Very High	>42	--	0
Total		135	



5.1.3 . Distribution of LDL among the MI Patients

Table 1.7. Distribution of LDL among the MI Patients

Total LDL mg/dl			
Test	Range in mg/dl	Number of Patient	%
Optimal	<100	3	2.222
Near Optimal/Above Optimal	100--129	17	0.12592
Borderline to High	130--159	73	54.0741
High	160--189	36	26.6667
Very High	>190	5	3.7037
Total		135	

5.1.4. Distribution of Triglyceride level among the MI Patients

Table 1.8. Distribution of Triglyceride among the MI Patients

Total Triglyceride mg/dl			
Test	Range in mg/dl	Number of Patient	%
Optimal	<150	4	3
Near Optimal/Above Optimal	150--159	10	7
Borderline to High	160—199	49	36
High	200--499	53	39
Very High	200--499	19	14
Total		135	100%

DISCUSSION

The result of the study showed that there is 96 % male and 4% female patient associate with MI among 135 patient so there are more risk of MI than female patient and most of them (91%) are Muslim , Hindu (8 %) and rarely Christian patient(1%) were found associated with MI .There were different type of MI were suffering by the patient among them 47 % patient had AMI , 44% patient had OMI .A good number people has Normal value of Creatinine 31.85% , abnormal are 4.44% but 64.44% data were not found . Majority of the patient were found smoking (73.33%) and patient were non-smoker (26.66%) so smoker is higher risk for MI. Patient who chew betel nut were 43.70% and not chew 56.30 % . The RBS of the patient found abnormal 35.55% and normal 11.11 % so number of diabetics patient were more than patient has normal value of RBS. Most of studied patient of MI had hypertension 59% and non hypertensive patient were low 40% and the majority of them taking table salt 67% and 33% were not taking extra salt. A good number of people taking tea or coffee 58.52 % suffering from MI and 41.48 % not taking Tea or Coffee. K⁺ level were normal range 47 patient (34.81 %) , 14 patient (10.37%) were abnormal and 74 no of patient data not found Concentration of Na⁺ level of the patient 25 % normal and 15 % abnormal and 60 % patient no data found. 12.59 % farmer , 29.63 % bussinessmen , 29.63 % service 5.53 %pension holder , 4.44% unemployee ,3.70 %house wife and others 11.85% . The study showed that age is vital factor for MI and most of the Patient experienced with MI age 45-55 (37.69 %) and socond priority to patient age beteen 56-65 and 36-45 (23.08%) .Most of MI patient had low income 38.40 % had income < 10000 so income also gratly responsible for MI .Most of the patient use Soybean oil (70.37%) , mustard oil 18.53% and sunflower oil 4.44% 44% of the patient had normal BP systolic 44% and 14.40 % had higher blood presure.In caase of Lipid profile patient who had Optimal value were 21.48% Borderline to high cholesterol level was 50.37 % , and High were 29.62% . HDL level (mg/dl) among the MI Patients Optimal 6.66% , Near Optimal/Above Optimal 33.33% , Borderline to High 11.633 % , High 21.48 % . LDL level (mg/dl) among the MI Patients Optimal 2.22% , Near Optimal/Above Optimal 0.15 % , Borderline to High 54.07 % , High 26.66 % and very high 3.70 % . Total Triglyceride mg/dl was Optimal 3% Near

Study of lipid profile in patients with myocardial infarction

Optimal/Above Optimal 7%, Borderline to High 36%. High 39 % Very High 14% .Result of the study showed that 29% Hyperlipidemic patient had myocardial infarction. Hyperlipidemia is the major risk factor of myocardial infarction which can be modified, treated or controlled by changing lifestyle or taking medicine.

The American Heart Association is the nation's oldest and largest voluntary health organization dedicated to reducing disability and death from diseases of the heart and stroke. Myocardial Infarction, America's No. 1 and No. 3 killers and all other cardiovascular diseases in America claim over 870,000 lives a year. In fiscal year 2005–06 the association invested over \$543 million in research, professional and public education, advocacy and community service programs to help all Americans live longer, healthier lives (**American heart association, 2007**).

For individual with hyperlipidemia, CHD risk is greatly increased with the both type I and type II diabetics patient . Over 70% of patients with diabetes die from macrovascular disease, mainly coronary heart disease.. Hypertension is very common in Hyperlipidemic patient, is strongly related to obesity and highly protective of cardiovascular complications. Many patients with high profile lipid level are overweight and have high cardiovascular risk. They need intensive and sustained advice on life style and appropriate treatment to achieve other risk factor targets as well as glycaemic control. Hyperlipidemic subjects are more likely to experience a myocardial infarction and have worse outcomes compared to non-Hyperlipidemic subjects. The underlying pathophysiology of the atherosclerotic process is not significantly different in Hyperlipidemic patient subjects, but the prothrombotic and procoagulant state with which hyperlipidemia is associated is thought to contribute to the higher incidence of and worse prediction after myocardial infarction (**Ian L Williams, 2003**). The prevalence of Hyperlipidemia among patients with acute myocardial infarction in a geographically defined population in the developing world is high especially in America with a trend for poor outcomes.

The National Cholesterol Education Program report from the United States and guidelines from Europe consider Hyperlipidemia to be a CHD equivalent, thereby

elevating it to the highest risk category (**national cholesterol education program (NCEP) expert panel on 2002, De Backer G., Ambrosioni, 2003**).

According to World Health Organization (WHO) estimates, in 2003, 16.7 million people around the globe die of CVD each year. This is over 29 percent of all deaths globally. (*www.who.int*). Eighty percent of chronic disease deaths occur in low and middle income countries and half are women. Cardiovascular disease alone will kill five times as many people as HIV/AIDS in these countries. (*Chronic Diseases and Their Common Risk Factors, WHO, Oct. 2005*) According to WHO estimates, 16.6 million people around the globe die of cardiovascular diseases (CVD) each year. In 2001 there were 7.2 million deaths from heart disease and 5.5 million from stroke. Another 15 million each year survive minor strokes. 600 million people with high blood pressure are at risk of heart attack, stroke and cardiac failure. In the WHO Report 2002 – Preventing Risks, Promoting Healthy Life, the WHO predicts that unless action is taken, by the year 2020 there will be 9 million deaths caused by tobacco, compared to almost 5 million now. 5 million deaths attributable to overweight and obesity, compared to 3 million now. At least 20 million people survive heart attacks and strokes every year; many require continuing costly clinical care (WHO). Cardiovascular Disease: Prevention and Control. 2006) CVD accounted for more than 216,000 deaths in the United Kingdom (UK) in 2004. Thirty-seven percent of deaths are from CVD, and 32 percent of premature deaths in men and 24 percent in women are from CVD. (*British Heart Foundation, 2006 Edition.*) CVD on average cost every EU citizen 230 Euro's in healthcare, but it led to 268.8 million lost working days, 2 million deaths and 4.4 million whose daily lives were affected. Some 1.4 million people were involved in providing unpaid care to sufferers of CHD and stroke alone, which together account for 47 percent of costs and 2/3 of deaths. (*European Heart Journal. Jose Leal, Oxford University, 2006*) About 231,000 heart attacks (myocardial infarctions) occur annually in the UK (128,000 in men and 103,000 in women in 2004). It is estimated that almost 1.3 million people living in the UK have had a heart attack (MI), (870,000 men and 419,000 women). About 760,000 men and 428,000 women under 75 living in the UK who have or have had angina. Overall, it is estimated that just over 1.5 million men and 1.1 million women who have had CHD (either heart attack or angina) are living in the UK. (*British Heart Foundation., 2006*)

Study of lipid profile in patients with myocardial infarction

According to the WHO, in 2002 there were 7.22 million deaths from CHD globally. (*Atlas of Heart Disease and stroke, WHO., 2004*) *know* it today, mortality among Hyperlipidemic patients in MI was reported to be as high as 40% (**Partamian J., 1965**). The results of the study suggested that myocardial infarction is more prominent in patients with higher lipid profile rather in patients with other risk factors According to WHO estimates, 15 million people each year suffer strokes and 5 million are left permanently disabled. (**Atlas of Heart Disease and Stroke, WHO, September 2004**) In England, 34 percent of men and 30 percent of women have HBP (140/90 mmHg or higher) or are being treated for hypertension. About 78 percent of men and 67 percent of women with HBP are not being treated. Of those being treated, just under 60 percent remain hypertensive. (**British Heart Foundation, Coronary Heart Disease Statistics, 2006**). In developed countries; lower socioeconomic groups have a greater prevalence of risk factors, higher incidence of disease and higher mortality. In developing countries, as the CVD epidemic matures, the burden will shift to the lower socioeconomic groups. (**WHO.**) A blood cholesterol level of less than 5.0 mill moles per liter (mmol/L) is suggested for both primary and secondary prevention of CHD. About 66 percent of men and women in the UK have blood cholesterol levels of 5.0 mmol/L and above. (*British Heart Foundation. Coronary Heart Disease Statistics, 2006 Edition.*) HDL -cholesterol level for men age 16 and above in England is 1.4 mmol/L and for women 1.6 mmol/L. Overall, about 6 percent of men and 2 percent of women have HDL-cholesterol levels of less than 1.0 mmol/L. (*British Heart Foundation Statistics, 2006*)

Conclusion

CVD has no geographic, gender, or socioeconomic boundaries. The global burden of disease as a result of CVD is rising, principally because of a sharp rise in the developing countries that are experiencing rapid health transition. Contributory causes include the aging of the world population, lifestyle changes caused by urbanization, progressive industrialization, and growing globalization, probable effects of fetal undernutrition on adult susceptibility to vascular disease, and possible gene-environment interactions influencing ethnic disparities. Altered diets and diminished physical activity are critical factors contributing to the acceleration of CVD epidemics, along with abnormal value of lipid profile. The prevalence of risk factors for CVD, however, varies across developing regions with consequent variations in the burden of CVD. The CVD epidemic in developing countries like Bangladesh differs from that observed in developed countries in the last century by virtue of its rapidity, occurrence in a background of limited health care infrastructure, widespread poverty, and low levels of community education. A global public health response must integrate policies and programs that effectively impact the multiple determinants of these diseases in a resource-sensitive and context-specific manner and provide protection over the life span through ancient, primary, and secondary prevention. Taking proper food and regular exercise help us to reduce the risk of MI and also help to control abnormal level of lipid profile data. The Study also shows that in government cardiovascular hospital patient having MI, majority of them are poor and they don't have sufficient money for lipid profile test. For this reason proper treatment is hampered to poor people. Government should come forward to keep eye on this fact for MI patient in Bangladesh.

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Appendix

NATIONAL INSTITUTE OF CARDIOVASCULAR DISEASES HOSPITAL (NICVD) DHAKA.

Questionnaire

1. Name of the respondent

.....

2. Address.....

3. Mobile.....

Block no.	House No.	Village/word	Thana	District

3. Sex: 1 = Male

2= Female

4. What is your age (in complete years)?

5. What is your religion?

1 = Islam

4=Buddhism

2 = Hinduism

5. Others (Specify).....

3 = Christianity

6. How far have you studied?

1 = Illiterate 5

6.HSC or its equivalent

2 = Non-formal education 6

7.Graduate +

3 = Class I-V

8.Others (Specify).....

4 = Class VI-IX

7. What is your marital status?

1 = Married and spouse live together

5.= Divorced

2 = Spouse live separately

6. = Never married

3 = Widow

4 = Widower

8. What is your occupation?

1 = Farmer

5. = Unemployed

2 = Businessman

6. = House Wife

3 = Service

7. = Others (specify).....

4 = Pension holder7

9. How many members are there in your family?

10. What is your average monthly family income (Taka)

11. Do you have your own income (Taka)?

1. Yes 2. No

12. If yes, how much (Taka)?

13. Did you ever smoke any form of tobacco?

1 = Yes

2 = No (go to no. 19)

14. Do you currently smoke?

1 = Yes

2 = No (go to no. 19)

15. How much do you smoke?

1 = Everyday

2 = occasionally (go to no. 19)

16. What type of cigar do you smoke?

1 = Cigarette

2 = Biri

3 = Cigarette & Biri both

4 = Others (specify).....

17. For how long you are smoking daily?

Year Month...

18. Do you currently chew betel nut?

1 = Yes

2 = No (go to no. 23)

19. How much do you chew betel nut

1 = Everyday

2 = occasionally (go to no. 23)



20. Do you use smokeless tobacco with betel?

1 = Yes

2 = No (go to no. 23)

21. What do you take with betel?

1 = Tobacco leaf

2 = Jorda (preparation of tobacco leaf)

3 = both

21. What do you take with betel?

1 = Tobacco leaf

2 = Jorda (preparation of tobacco leaf)

3 = both

22. For *how* long do you chew betel?

23. Do you take excess tea or coffee?

1 = Yes

2 = No

24. Please do not mind, have you ever consumed a drink that contains alcohol ?

1 = Yes

2 = No

25. Do you currently drink alcohol?

1 = Yes

2 = No

26. For how long do you drink alcohol?

Year Month...

27. What types of oil or fat are used most often for meal preparation in your household?

1. Soybean oil

5. Sunflower oil

2. Mustard oil

6. Corn oil

3. Sesame oil

7. Gheef Butter

8. Other (specify)

4. Palm oil

28. Do you take table salt?

1 = Yes

2 = No

29. Did you come here with the following complaints?

1 = Yes 3. Don't know

2 = No

1. Chest pain,

2. left arm pain

3. right arm pain,

4. jaw pain

5. neck pain

6. back pain

7. shortness of breath

8. heart burn

9. pulmonary edema

10. nausea, vomiting,

11. loss of consciousness

12. sweating

13. Other (specify).....

30. Do any member of your family have the following diseases ?

1 = Yes

2 = No

3 = Don't know

- 1. Hypertension
- 2. Diabetes Mellitus
- 3. Asthma
- 4. Coronary heart disease

31. If yes-----

What is the relation between you & them?

- 1 = Father 3 =parent
- 2 = Mother 4 =siblings

32. God forbid, are you suffering from any chronic disease?

- 1 = Yes
- 2 = No
- 3 = don't know

- 1. Hypertension
- 2. Bronchitis
- 3. Diabetes Mellitus
- 4. Arthritis
- 5. Cancer
- 6. Other (specify).....

33. For female only-

Did you take birth control pill any time in your life?

- 1 = Yes
- 2 = No

34. If yes-

Do you take pills now?

1 = Yes

2 = No

36. If yes

For how long are you taking?

- Yrs. Month

37. Currently use drugs:

(1) ACE Inhibitor:

Benzepiril, Captopril, Enalapril, Fosinopril, Lisinopril, Moexipril, Quinapril, Ramipril

(2) β blocker:

Atenolol, Labetalol, Metoprolol, Nadolol, propranolol.

3) Diuretics:

Bumetanoid, Furosemide, Hydrochlorothiazide, Spironolactone, Triamterene.

(4) Antiplatelets:

Aspirin, Abciximab, Clopidogrel, Dipyridamole, Eptifibatide, Ticlopidine, Tirofiban.

(5) Anticoagulents:

Danaparoid, Enoxaprin, Heparin, Lepirudin, Warfarin.

(6) Organic Nitrate:

Isosorbide di nitrate, Isosorbide mononitrate, Nitroglycerin.

(7) Lipid lowering agent:

Atorvastatin, Fluvastatin, Lovastatin, Pravastatin, Rosuvastatin Calcium, Simvastatin, Cholestyramine (Questran®, Questran® Light, Prevalite®, Locholest®, Locholest® Light), Colestipol, Colesevelam Hcl .

(8) Ca^{+} Channel Blocker:

Amlodipine, Diltiazem, Felodipine, Nicardipine, Nitredipine, Nifedipine, Verapamil.

(9) Na^{+} Channel Blocker:

Disopyramide, Flecainide, Lidocaine, Mexiletine, Procainamide, Propafenone, Quinidine, Tocainide.



(10) K^+ Channel Blocker: Amiodarone, Dofetilide, Sotalol.

(11) Aldosteron Antagonist agent: Eplerenone.

(12) Angiotensin Receptor Antagonist: Losartan.

(13) Other Drugs: Omega-3 fatty acid, Adenosine, Digoxin, Benzodiazepines.

38. Do you know your blood group?

1= Yes, I know

2= No, I don't know

If Yes,

L =A

2 = B

3 = O

4=AB

39. Do you think that it is essential to know blood group of any individual?

1=Yes.

2=No.

From record

- 1. Height (cm)
- 2. Weight (kg)
- 3. Waist girth (cm)
- 4. Hip girth (cm)
- 5. Systolic Blood Pressure (rnm/Hg).....
- 6. Diastolic Blood Pressure (rnm/Hg)
- 7. Blood group.....
- 8. S. Lipid Profile.....
 - Total Cholesterol..... mg/dl.
 - Triglyceridemg/dl.....
 - HDL-Cholesterol mg/dl .
 - LDL-Cholesterol..... mg/dl.....
- 9. Blood urea.....
- 10. Serum electrolyte.
 - 1=Na..... m.eq/L
 - 11.2=Km.eq/L
- 12. SGPT.....
- 13. SGOT.....
- 14. CKMB.....
- 15. Troponine-I
- 16. RBS
- 17. Echocardiography
- 18. X-ray chest
-
- 20. ECG Report
 - 1= Yes, 2= No
 - Site of MI ,
 - 1. Anterior MI
 - 2. Extensive anterior



Prescription:

Used Drug (Brand Name)	Type/Mechanism	Generic or Chemical Name	Dose/Frequency

Rx