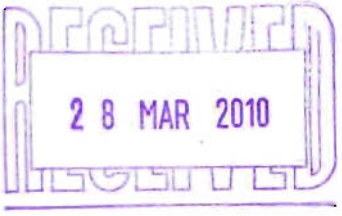
THE LIFE STYLE RISK FACTORS OF MYOCARDIAL INFARCTION AND EFFECT OF ANTILIPODIAL DRUG INTAKE FOR THE PREVENTION OF MYOCARDIAL INFARCTION

Submitted by:

AMATUNNUR
ID-2005-2-70-029

Department of Pharmacy
East West University

December-2009



THE LIFE STYLE RISK FACTORS OF MYOCARDIAL AND EFFETS OF ANTILIPODIAL DRUG INTAKE FOR THE PREVENTION OF MYOCARDIAL INFARCTION

A Thesis paper submitted to the Department of Pharmacy, East West University in conformity with the requirements for the Degree of Bachelor of Pharmacy.

Place of study was National Institute of Cardiovascular Disease (NICVD).

This thesis paper is dedicated to my parents

CERTIFICATE

This is to certify that, the thesis 'Life Style Risk Factors of Myocardial Infarction and the drugs that are commonly used to prevent MI' submitted to the Department of Pharmacy, East West University Mohakhali, Dhaka for the partial fulfillment of the requirements for the degree of Bachelor of Pharmacy (B.Pharm) was carried out by Amatunnur (ID: 2005-2-70-029) 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 laboratory facilities availed of this connection is duly acknowledged.

Sufia Islam 24.12.09

Dr. SUFIA ISLAM, PhD
Supervisor
Associate professor
Department of Pharmacy
East West University
Mohakhali, Dhaka

24.12.09

MOMENA SHIRIN
Co-Supervisor
Superintendent
Intravenous Fluid Production Unit
Institute of Public Health (IPH)
Mohakhali, Dhaka

Dr. CHOWDURY FAIZ HOSSAIN
Chairperson
Department of Pharmacy
East West University
Mohakhali, Dhaka

LIST OF CONTENTS

ACKNOWLEDGEMENTS

First and foremost I would like to express my thanks to Dr. Sufia Islam. She has helped me a lot by giving me her precious time and directed me properly through a guideline to clear my confusion about my study. I have got to know many new terms, ideas related my study.

I am grateful to co- supervisor Mrs. Momena Shirin M. Phil (PSM) superintendent, I.V. fluid, Institute of Public Health, Mohakhali, Dhaka for her guidance, criticism and encouragement during the preparation of this study.

I am gratefully acknowledged to Pro-vice chancellor Professor Muniruddin Ahmed and Chairperson Professor Dr. Chowdhury Faiz Hossain for their inspiration in my study

Moreover, I am grateful to my administration as they provide the facilities to use the computer lab with printer. May be it would have been impossible to finish the report without these facilities.

I express my sincere gratitude to my caring parents for guiding me all through my life, including that for my research project. I am very grateful to my brother, sisters and friends, who encouraged me enormously.

Finally, all extol and commendation should go to almighty "ALLAH" the most merciful who enabled me in completing this work soundly and orderly.

AMATUNNUR

December, 2009.

ABSTRACT

Myocardial infarction may be the first manifestation of coronary artery disease, or it may occur, repeatedly, in patients with established disease like High blood serum cholesterol which is the major risk factor of myocardial infarction.

High cholesterol (hypercholesterolemia) can cause the formation and accumulation of plaque deposits in the arteries. Atherosclerosis can lead to plaque ruptures and blockages in the arteries, which increase the risk for heart attack, stroke, circulation problems, and death. Long term duration of uncontrolled blood serum cholesterol may cause serious disorders. High blood serum cholesterol accelerates and exacerbates the occurrence of arteriosclerosis, increasing the risk of myocardial infarction. High blood cholesterol is associated with markedly increased mortality in the presence of abnormal high density lipoprotein (HDL), low density lipoprotein (LDL), triglycerides (TG), total cholesterol (TC) level after acute myocardial infarction and support for aggressive treatment of coronary risk factors among hypercholesterolemia patients.

The study was conducted in the National Institute of Cardiovascular Diseases and Hospital (NICVD). The data of 60 patients who were suffering from Myocardial Infarction were included in the study.

To find out the relationship between high blood cholesterol and myocardial infarction, and control of high blood cholesterol with in normal range by antilipodial drug, we conducted a study with 60 MI patients and then evaluate their all medical history, diagnosis and treatment. Collected all information then evaluated and compared with each patient.

The results of the study showed that patients of age ranged from 56 to 60 years were more prevalent to MI. Among 60 MI patients, 88% male and 12% female patients had been suffering myocardial infarction. The results showed that 92% patients were married, 37% were not educated, and 13% were unemployed. About 25% patients were service holder, 18% were businessman and 12% were housewives. There were MI patients who suffered from other diseases such as Diabetes mellitus (DM) (12%);

hypertension (11%); both asthma and Parkinson (8%); DM and hypertension (5%); both asthma & hypertension (5%); both DM & hypertension (8%).

The result of this study also showed that 41.66% MI patients have optimal range HDL, 43.33% have borderline HDL, 6.66% have HDL, 3.33% have very high HDL, 20% have optimal LDL, 46.66% have borderline LDL, 30% have high LDL, 20% have very high LDL, 26.66% have optimal TC, 41.66% have borderline TC, 25% have high TC, and 6.66% have very high TC with myocardial infarction. The results of the study suggest that myocardial infarction is more prominent in patients with hypercholesterolemia.

The result of this study also showed that the mean concentration ± SD of triglycerides (TG) at 1st day 192.85 ± 54 and after 15 days treatment with antilipodial drug TG was decreased to136.85 ± 14 mg/dl and its P=<0.01 , total cholesterol (TC) at 1st day 198.78 ± 42.9 mg/dl after 15 days treatment with antilipodial drug TC decreased to 186.25 ± 29.3 mg/dl and its P= <0.01, high density lipoprotein (HDL) at 1st day 36.13 ± 74 mg/dl after 15 days treatment with antilipodial drug which was increased to 41.76 ± 83 mg/dl and its P =<0.01, and low density lipoprotein (LDL) at 1st day 135.17 ± 33.5 mg/dl after 15 days treatment with antilipodial drug LDL decreased to 126.67 ± 55.8 mg/dl and its P =<0.02. It can be concluded that after 15 day HDL was increased and LDL, TG, TC were decreased with the anti lipid medication such as statin, niacin, Fibrates, resins prescribed by the doctor.

CHAPTER 1

INTRODUCTION

1.1Communicable Disease:

A communicable disease is carried by microorganisms and transmitted through people, animals, surfaces, foods, or air. Communicable diseases rely on fluid exchange, contaminated substances, or close contact to travel from an infected carrier to a healthy individual.

The disease might need a blood exchange via an injection, float along a sneeze in a movie theater, or get transmitted through childbirth. We understand their origin. Examples of communicable diseases include herpes, malaria, mumps, HIV/AIDS, influenza, chicken pox, ringworm, and whooping cough. Cancer, on the other hand, is not a communicable disease.

Parasites, bacteria, and viruses all qualify as pathogens, nicknamed "germs," and can cause a communicable disease. Their method of transmission, period of dormancy, ease of contagiousness, and relative danger can differ drastically from one disease to the next. Governmental health agencies spend a great deal of time and money studying the risk or spread of various contagious diseases in order to identify outbreaks, prevents reoccurrences, or develops treatments. They compile statistics such as incidence, which measures how many new cases are diagnosed per year, and prevalence, which identifies how many cases exist at any one time **(S.Mithra22 August 2009)**

Different types of infectious diseases in the first half of the twentieth century and cause increase death of the people that called communicable disease. But now a day in the developed countries were able to decrease mortality and morbidity from infectious diseases in the first half of the twentieth century because of the development of vaccines and other public health measures. They shared successful approaches and resources with developing countries through international assistance programs after World War II **(Christopher P. Howson, K. Srinath Reddy, Thomas J. Ryan, and Judith R. Bale, 1998).**

1.2Non-communicable disease:

A **non-communicable disease** or **NCD** 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 diseases of affluence.

Examples include hypertension, diabetes, cardiovascular disease, cancer, and mental health problems, asthma, atherosclerosis, allergy etc(**The European Strategy for the Prevention and Control of Noncommunicable Diseases, document from the World Health Organization 2005 Nov 5).**

Globally, non-communicable diseases (NCDs) are increasingly recognized as a major cause of morbidity and mortality. The global burden of disease study assessed and ranked the individual disease/condition contributing to burden of disease in the world in 1990.they contrasted their ranking with the projection global burdens in the year 2020, where heart disease and cerebro vascular disease, are projected to occupy the 1st and 4th positions in 2020 which was the 5th and 6th positions respectively in 1990 **(World Health Organization 2002).**

Coronary heart disease is an important cause of morbidity and mortality in most of the industrialized nations and is gaining an importance as a major disease in developing countries. Approximately 1.5 million myocardial infarction cases and 520,000 deaths related to this occur per year in the USA. Although cardiovascular disease is considered an acquired problem of the industrialized western world, the Improvement in the socioeconomic industrial base in developing countries has brought this problem to the doorstep of the third world country **(WHO, 1988).**

1.2.1. Coronary heart disease (CHD):

Coronary heart disease (CAD) is now a global health problem and an important cause of mortality in the affluent. With the improvement of socioeconomic status, urbanization and of dietary habit and life style the incidence of ischemic heart disease (IHD) is also increasing in the developing countries including Bangladesh.

Coronary heart disease (CHD) is the term for atherosclerosis affecting the coronary arteries. It is the single most common cause of death in Europe, responsible for nearly two million deaths a year. **(British Heart Foundation. European Cardiovascular Disease Statistics, 2008)**

CHD is caused by atherosclerosis, a chronic process in which lipid-rich plaque forms in the arterial wall. Factors that increase a person's risk of coronary heart disease include a family history of the condition, high cholesterol, high blood pressure, diabetes, and smoking **(National Institutes of Health, Heart, Lung, and Blood Institute September 2002. Fuster V, Moreno PR 2005).**

Coronary heart disease (CHD) is a narrowing of the small blood vessels that supply blood and oxygen to the heart. CHD is also called coronary artery disease (**Mosca L, Banka CL, Benjamin EJ, et al. February 19, 2007.**)

Coronary heart disease is usually caused by a condition called atherosclerosis, which occurs when fatty material and a substance called plaque build up on the walls of your arteries. This causes them to get narrow. As the coronary arteries narrow, blood flow to the heart can slow down or stop. This can cause chest pain (stable angina), shortness of breath, heart attack, and other symptoms (**Smith SC Jr, Allen J, Blair SN, et al.2006 may 26)**
Coronary heart disease (CHD) is the leading cause of death in the United States for men and women **(Mosca L, Banka CL, Benjamin EJ, et al 2007, 19 February).**

MI is a leading cause of morbidity and mortality in the United States. Approximately 1.3 million cases of nonfatal MI are reported each year, for an annual incidence rate of approximately 600 cases per 100,000 people. The proportion of patients diagnosed with NSTEMI compared with STEMI has progressively increased **(WHO 1988).**

International Cardiovascular diseases account for 12 million deaths annually worldwide. MI continues to be a significant problem in industrialized countries and is becoming an increasingly significant problem in developing countries Mortality/Morbidity Approximately 500,000-700,000 deaths are caused by ischemic heart disease annually in the United States.

Myocardial infarction is becoming a serious public health problem in Bangladesh **(Abul Faize, 1988).**

Coronary heart disease 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. And by 2020, it is very likely that all regions of the world will be affected **(Boon NA, College NR, Walker BR and Hunter JAA, 2006).**

The burden of non-communicable diseases in developing countries:

By the dawn of the third millennium, non communicable disease are sweeping the entire globe, with an increasing trend in developing countries where, the transition imposes more constraints to deal with the double burden of infective non-infective diseases in a poor environment characterized by ill-health systems. By2020, it is predicted that these diseases will be causing seven out of every 10 deaths in developing countries **(Today's challenges Geneva and diet, Nutrition and the prevention of chronic diseases, World health Organization,2003).**

The world health report 2001 has indicated that NCDs account for almost 60% deaths and 46% of the global burden of disease. 75% of total deaths due to NCDs occur in developing countries. Coronary heart disease (CHD) rank no 1 among the ten leading cause of mortality estimated for 1999(WHO REPORT). In UK one in four men and one in five women die from this disease, an estimated 300,000 people have a myocardial Infarction each year and approximately 1.7 million people have angina **(Haslet C, chilverE.R UK, 1999).**

Regionally, the countries of the south East Asia region are thus facing a double burden, with a heavy load of infectious diseases and an added burden of NCDs. Cardiovascular diseases ranked third as a cause of death in South East Asia. In the year 2000, 16.7 million people died from cardio vascular diseases, accounting for 30.3% of all deaths world wide; more than half of these deaths taking place in the developing countries **(WHO Non communicable Diseases prevention and management, 2001).**

South Asia (Pakistan, India, Bangladesh, Nepal and Sri Lanka) represents more than a quarter of the developing world, and is likely to be strongly affected by the increase in the cardiovascular disease.

Table 1: Percentage of Risk Factor MI of patients of different countries in South Asia

	Pakistan **(Gentller MM, White PD, 1994)**	India **(Nefzger MD, Hrubee, 1969)**	Bangladesh **(Denbrough MA, 1962)**	Nepal **(Meshalkin EN, 1981)**	Sri Lanka **(WHO, 1988)**
Hypertension*					
Men	17%	36.4%	9.8%	....	17%
Woman	....	37.5%	15.6%	....	....
Cigarette Smoking	....	....	....	73.7%	....
Men	34%	36.5%	50.3%		57.9%
Woman				57.95%	
Oral Smokeless Tobacco	10%	22%	....	....	....
Men	....	....	16.3%	....	....
Woman	....	....	21.4%	....	....
Diabetes#	5%	....	....	....	....
Men	....	13.1%	2.9%	....	5.8%
Woman	....	11.3%	0.7%	....	....

*Classified according to WHO criteria. #defined as random blood suger of > 140 mg/dl (7.8 mmol/L) apat from the study from India. Where a fasting cutoff of 126 mg/dl (7.0 mmol/L) was used.

In indigenous population of South Asia, high prevalence of rates for CHD risk factors are also apparent (table-1.1.2). in India, prevalence of coronary artery disease has been reported as being 11% in 2001;prevalence of coronary artery disease and its relationship

to lipid in a selected population in South India; the Chennai Urban population Study **(Mohan V, Deepa R, Rani ss, PremalthaG. 2001)**.

In Bngladesh, acute myocadial infarction is the leading cause of mortality and mobidity and also emerging as a major health problem in develoing countries **(Rashid K.M. Khbiruddin Md. Hyder S.1999).**

With industrialization and developing of the country, now a day, like other developing countries, there are more cases on myocardial infarction in Bangladesh. It was reported that coronary heart disease ranks third among the cardiovascular disease in Bangladesh. Prevalence of the disease was found to be 3.38 and 14/1000 in two different studies carried out in 1976 and 1984 respectively **(Rhashid AKMH. 1997).**

According to one survey, it was 3.3/1000 **(Hayee MA, Chowdhury NA, Akhter N, and Ahsan S.1993).**

NATIONAL RELEVANCE

Worldwide immigration has created ethnic diversity in many countries such as the United States, Canada, Malaysia, and Singapore. Several studies from these countries indicate important differences in the incidence of CVD for recent immigrants now exposed to a new set of environmental risks. Some of these differences may be attributable to socioeconomic factors and culturally based patterns of diet, behavior, and activity, whereas others may be attributable to genetic factors. Studies of ethnically diverse groups in their original homeland and their newly adopted country continue to provide insights that improve understanding of the complexities and approaches for reducing the burden of CVD in the wider population **(Christopher P. Howson, K. Srinath Reddy, Thomas J. Ryan, and Judith R. Bale. 1998).**

1.2.2. CLASSIFICATION OF CORONARY HEATR DISEASE:

Coronary heart disease are two types, those are;

1.2.2.1. Angina:

Angina - or angina pectoris (Latin for squeezing of the chest) - is chest pain, discomfort, or tightness that occurs when an area of the heart muscle is receiving decreased blood oxygen supply. It is not a disease itself, but rather a symptom of coronary artery disease,

the most common type of heart disease. The lack of oxygen rich blood to the heart is usually a result of narrower coronary arteries due to plaque buildup, a condition called angina. Narrow arteries increase the risk of pain, coronary artery disease, heart attack, and death.

The actual angina attacks are the result of this reduced oxygen supply to the heart. Physical exertion is a common trigger for stable angina, as the heart demands more oxygen than it receives in order to work harder. In addition, severe emotional stress, a heavy meal, exposure to extreme temperatures, and smoking may trigger angina attacks **(National Heart LaBI, 2001).**

Pain is usually found in the upper chest and most sufferers say it feels like a heavy weight or squeezing sensation that eases off during rest.

This pain may also be felt in the neck, shoulders, arms or back and is frequently mistaken for indigestion, but the relationship with exertion is the key feature that points to a cardiac cause.

Angina can also come on when walking after a heavy meal or against a cold wind or when angry or stressed.

As the coronary arteries become more blocked, pain comes on with relatively little exertion, starting even at rest and taking longer to ease off – this is called crescendo or unstable angina and is serious because, unless treatment is given, one of the coronary arteries may become totally blocked **(Boden WE, O'rourke RA, Teo KK, et al, 2007 Mar 26)**

- **Stable (or chronic) angina** is brought on when the heart is working harder than usual, such as during exercise. It has a regular pattern and can be predicted to happen over months or even years. Symptoms are relieved by rest or medication.

- **Unstable angina** does not follow a regular pattern. It can occur when at rest and is considered less common and more serious as it is not relieved by rest or medicine. This version can signal a future heart attack within a short time - hours or weeks.

It's thought that nearly 7 million people in the United States suffer from angina. About 400,000 patients go to their doctors with new cases of angina every year **(national heart lung blood institute November 2007).**

1.2.2.2MYOCARDIAL INFARCTION:

The term "myocardial infarction" focuses on the heart muscle, which is called the myocardium, and the changes that occur in it due to the sudden deprivation of circulating blood. This is usually caused by arteriosclerosis with narrowing of the coronary arteries, the culminating event being a thrombosis (clot). The main change is death (necrosis) of myocardial tissue.

The word "infarction" comes from the Latin "infarcire" meaning "to plug up or cram." It refers to the clogging of the artery, which is frequently initiated by cholesterol piling up on the inner wall of the blood vessels that distribute blood to the heart muscle.

Myocardial infarction (MI or AMI for acute myocardial infarction), commonly known as a heart attack, occurs when the blood supply to part of the heart is interrupted causing some 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 (like cholesterol) 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 and / or death (infarction) of heart muscle tissue. (Myocardium) **Kosug Kimura K, Ishikawa T et al. (March 2006).**

The ideal definition of myocardial infraction is, immediately after an acute coronary occlusion, blood flow ceases in the coronary vessels beyond the occlusion except for small amounts of collateral flow from surrounding vessels. The area of muscle that has either zero flow or so little flow that it cannot sustain cardiac muscle function is said to be infracted. The overall process is called a myocardial infarction. **(C.Guyton, M.D., John E. Hall, Ph.D., tenth edition)**

1.2.2.3. Causes of Myocardial Infarction:

The most common cause of MI is narrowing of the epicardial blood vessels due to atheromanous plaques. Plaque rupture with subsequent exposure of the basement

membrane results in platelet aggregation, thrombus formation, fibrin accumulation, hemorrhage into the plaque, and varying degrees of vasospasm. This can result in partial or complete occlusion of the vessel and subsequent myocardial ischemia. Total occlusion of the vessel for more than 4-6 hours results in irreversible myocardial necrosis, but reperfusion within this period can salvage the myocardium and reduce morbidity and mortality.

No atherosclerotic causes of MI include coronary vasospasm as seen in variant (Prinzmetal) angina and in patients using cocaine and amphetamines; coronary emboli from sources such as an infected heart valve; occlusion of the coronaries due to vasculitis; or other causes leading to mismatch of oxygen supply and demand, such as acute anemia from GI bleeding. MI induced by chest trauma has also been reported, usually following severe chest trauma such as motor vehicle accidents and sports injuries (**Medscape's article "New Definition of 'MI' Poised for World Domination ").**

Thrombosis - the cause in most cases

The common cause of MI:

The common cause of an MI is a blood clot (thrombosis) that forms inside a coronary artery, or one of its branches. This blocks the blood flow to a part of the heart.

Blood clots do not usually form in normal arteries. However, a clot may form if there is some atheroma within the lining of the artery. Atheroma is like fatty patches or 'plaques' that develop within the inside lining of arteries. (This is similar to water pipes that get 'furred up'.) Plaques of atheroma may gradually form over a number of years in one or more places in the coronary arteries.

What happens is that a 'crack' develops in the outer shell of the atheroma plaque. This is called 'plaque rupture'. This exposes the softer inner core of the plaque to blood. This can trigger the clotting mechanism in the blood to form a blood clot. Therefore, a build up of atheroma is the root problem that leads to most cases of MI. (The diagram above shows four patches of atheroma as an example. However, atheroma may develop in any section of the coronary arteries.)

Treatment with 'clot busting' drugs or a procedure called angioplasty (see below) can break up the clot and restore blood flow through the artery. If treatment is given quickly enough this prevents damage to the heart muscle, or limits the extent of the damage.

Uncommon causes of MI:

Various other uncommon conditions can block a coronary artery and cause an MI. For example: inflammation of the coronary arteries (rare); a stab wound to the heart; a blood clot forming elsewhere in the body (for example, in a heart chamber) and traveling to a coronary artery where it gets stuck; cocaine abuse which can cause a coronary artery to go into spasm; complications from heart surgery; and some other rare heart problems. There are not dealt with further.

The rest of this leaflet deals only with the common cause - thrombosis over an atheroma plaque (**Stahmer S; Acute Coronary Syndrome. eMedicine, January 2007).**

Classified according to WHO criteria. #defined as random blood suger of > 140 mg/dl (7.8 mmol/L) apat from the study from India. Where a fasting cutoff of 126 mg/dl (7.0 mmol/L) was used.

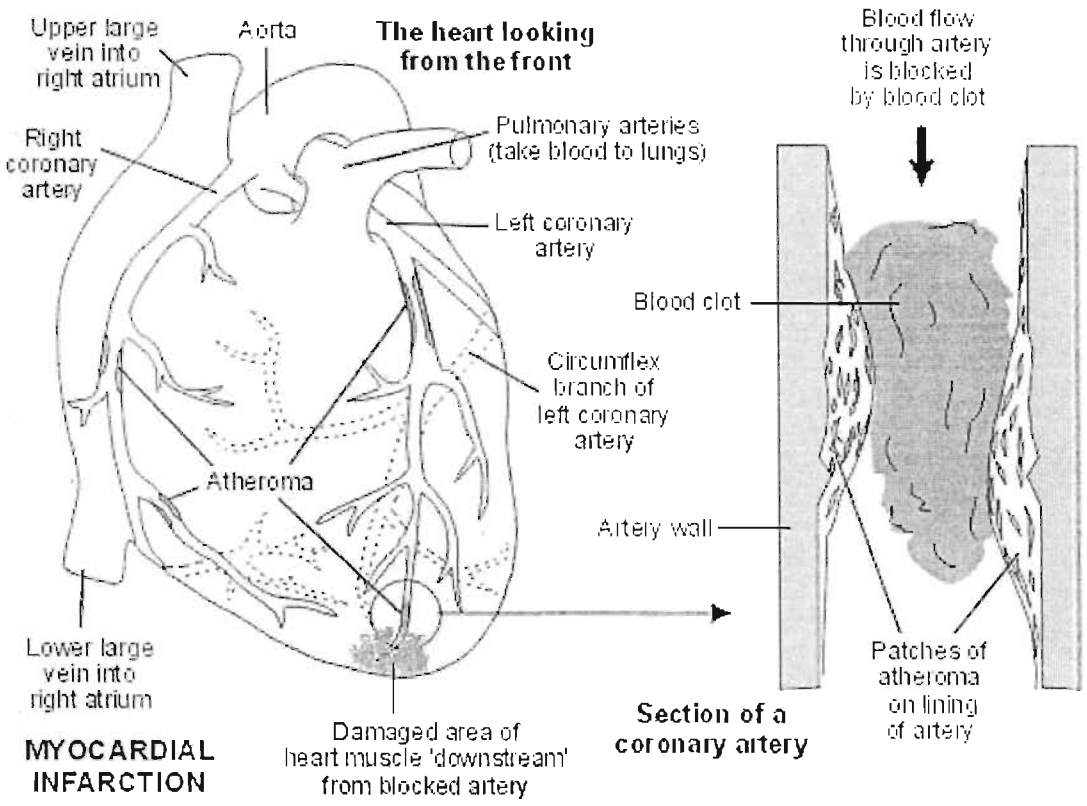
In indigenous population of South Asia, high prevalence of rates for CHD risk factors are also apparent (table-1.1.2). In India, prevalence of coronary artery disease has been reported as being 11% in 2001; prevalence of coronary artery disease and its relationship to lipid in a selected population in South India; the Chennai Urban population Study (**Mohan V, Deepa R, Rani ss, PremalthaG. 2001**).

In Bngladesh, acute myocadial infarction is the leading cause of mortality and mobidity and also emerging as a major health problem in develoing countries **(Rashid K.M. Khbiruddin Md. Hyder S.1999).**

With industrialization and developing of the country, now a day, like other developing countries, there are more cases on myocardial infarction in Bangladesh. It was reported that coronary heart disease ranks third among the cardiovascular disease in Bangladesh. Prevalence of the disease was found to be 3.38 and 14/1000 in two different studies carried out in 1976 and 1984 respectively **(Rhashid AKMH. 1997).**

According to one survey, it was 3.3/1000 **(Hayee MA, Chowdhury NA, Akhter N, and Ahsan S.1993).**

1.2.3. HOW DOSE MYOCARDIAL INFARCTION OCCUR?



If MI occurs, a coronary artery or one of its smaller branches is suddenly blocked. The part of the heart muscle supplied by this artery loses its blood (and oxygen) supply. This part of the heart muscle is at risk of dying unless the blockage is quickly undone. (The word 'infarction' means death of some tissue due to a blocked artery which stops blood from getting past.)

If one of the main coronary arteries is blocked, a large part of the heart muscle is affected. If a smaller branch artery is blocked, a smaller amount of heart muscle is affected. In people who survive an MI, the part of the heart muscle that dies ('infarcts') is replaced by scar tissue over the next few weeks **(Stahmer S; Acute Coronary Syndrome. eMedicine, January 2007) .**

1.2.4 Types of Myocardial Infarction:

New clinical classification of MI

Classification	Description
1	Spontaneous MI related to ischemia due to a primary coronary event, such as plaque erosion and/or rupture, fissuring, or dissection
2	MI secondary to ischemia due to an imbalance of O2 supply and demand, as from coronary spasm or embolism, anemia, arrhythmias, hypertension, or hypotension
3	Sudden unexpected cardiac death, including cardiac arrest, often with symptoms suggesting ischemia with new ST-segment elevation; new left bundle branch block; or pathologic or angiographic evidence of fresh coronary thrombus—in the absence of reliable biomarker findings
4a	MI associated with PCI
4b	MI associated with documented in-stent thrombosis
5	MI associated with CABG surgery

(Alpert JS, Thygesen K, Antman E, et al, 2000).

There are five main types of myocardial infarction: -

Anterior wall – This is due to an occlusion of the left anterior descending artery and affects the front wall of the left ventricle. The papillary muscles and intraventricular septum may also be affected (anteroseptal). This sort of infarction has disastrous consequences on the patient by reducing the cardiac output dramatically. Cardiogenic shock is a very real state following an anterior infarction.

Lateral wall – This is due to an occlusion of the lateral branch of the circumflex artery. If the left anterior descending artery is also occluded this will result in an Anterolateral Infarct.

Inferior wall – This is due to an occlusion of the right coronary artery and results in an infarction of the base of the left ventricle.

Posterior wall – This is due to an occlusion of the posterior branch of the right coronary or left circumflex artery. It affects the back wall of the left ventricle.

Right ventricular infarction – This is due to occlusion of the right coronary artery or sometimes the left circumflex artery. A right ventricular infarct may occur in conjunction with an inferior or posterior left ventricular infarction. A right ventricular infarction will affect the pulmonary circulation and oxygenation of the blood due to decreased cardiac output from the right ventricle.

(European Society of Cardiology, American College of Cardiology, 2000)

1.2.5 Symptoms of Myocardial Infarction:

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.

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.

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.

Nausea and vomiting – This is due to pain, redirection of blood away from the gastrointestinal system (splanchnic circulation), release of adrenaline and other catecholamines into the blood (fight / flight response) and from side effects of medications used to treat the condition and symptoms.

General fatigue – This is due to reduced cardiac output and generalised muscle ischaemia. The patient will be prostrated during the attack and this may also be a cause of this.

Tachyrhythmias – This is due to the heart trying to compensate for the low cardiac output by increasing the rate (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.

Hypotension – This is due to reduced cardiac output. Initially the patient may be hypertensive due to the aggravated compensation mechanisms.
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.

Sense of Impending Doom – This is due to the release of adrenaline and other catecholamines 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. The onset of symptoms in myocardial infarction (MI) is usually gradual, over several minutes, and rarely instantaneous (**National Heart, Lung and Blood Institute. Heart Attack Warning Signs. Retrieved November 22, 2006).**

Chest pain is the most common symptom of acute myocardial infarction and is often described as a sensation of tightness, pressure, or squeezing. Chest pain due to ischemia (a lack of blood and hence oxygen supply) of the heart muscle is termed angina pectoris. Pain radiates most often to the left arm, but may also radiate to the lower jaw, neck, right arm, back, and epigastrium, where it may mimic heartburn. Levine's sign, in which the patient localizes the chest pain by clenching their fist over the sternum, has classically been thought to be predictive of cardiac chest pain, although a prospective observational study showed that it had a poor positive predictive value(**Marcus GM, Cohen J, Varosy PD, et al. 2007).**

Shortness of breath (dyspnea) occurs when the damage to the heart limits the output of the left ventricle, causing left ventricular failure and consequent pulmonary edema. Other symptoms include diaphoresis (an excessive form of sweating), weakness, light-headedness, nausea, vomiting, and palpitations. These symptoms are likely induced by a massive surge of catecholamines from the sympathetic nervous system. This occurs in response to pain and the hemodynamic abnormalities that result from cardiac dysfunction. Loss of consciousness (due to inadequate cerebral perfusion and cardiogenic shock) and even sudden death (frequently due to the development of ventricular fibrillation) can occur in myocardial infarctions. Women and older patients experience atypical symptoms more frequently than their male and younger counterparts (Little **RA, Frayn KN, Randall PE, et al. (1986), Canto JG, Goldberg RJ, Hand MM, et al. December 2007).**

Women also have more symptoms compared to men (2.6 on average vs. 1.8 symptoms in men). **Canto JG, Goldberg RJ, Hand MM, et al. (December 2007).** The most common symptoms of MI in women include dyspnea, weakness, and fatigue. Fatigue, sleep disturbances, and dyspnea have been reported as frequently occurring symptoms which may manifest as long as one month before the actual clinically manifested ischemic event. In women, chest pain may be less predictive of coronary ischemia than in men (**Canto JG, Goldberg RJ, Hand MM, et al. December 2007,McSweeney JC, Cody M, O'Sullivan P, Elberson K, Moser DK, Garvin BJ 2003).**

Approximately half of all MI patients have experienced warning symptoms such as chest pain prior to the infarction **(D Lee, D Kulick, J Marks. Heart Attack (Myocardial Infarction) by MedicineNet.com. Retrieved November 28, 2006, Canto JG, Goldberg RJ, Hand MM, et al. (December 2007).**

Approximately one fourth of all myocardial infarctions are silent, without chest pain or other symptoms. These cases can be discovered later on electrocardiograms or at autopsy without a prior history of related complaints. A silent course is more common in the elderly, in patients with diabetes mellitus. And after heart transplantation, probably because the donor heart is not connected to nerves of the host. It may be difficult for a layman to self diagnose a heart attack (**Davis TM, Fortun P, Mulder J, Davis WA,**

Bruce DG (2004). Rubin's Pathology - Clinicopathological Foundations of Medicine. Maryland: Lippincott Williams & Wilkins. 2001 In diabetics, differences in pain threshold, autonomic neuropathy, and psychological factors have been cited as possible explanations for the lack of symptoms, Davis TM, Fortun P, Mulder J, Davis WA, Bruce DG 2004).

Any group of symptoms compatible with a sudden interruption of the blood flow to the heart is called an Acute Coronary Syndrome **(American Heart Association Retrieved November 25, 2006).**

The differential diagnosis includes other catastrophic causes of chest pain, such as pulmonary embolism, aortic dissection, pericardial effusion causing cardiac tamponade, tension pneumothorax, and esophageal rupture(**Boie ET 2005).**

Acute MI may have unique manifestations in individual patients. The degree of symptoms ranges from none at all to sudden cardiac death. An asymptomatic MI is not necessarily less severe than a symptomatic event but patients who experience asymptomatic MIs are more likely to be diabetic. Despite the diversity of manifesting symptoms of MI, there are some characteristic symptoms.

- Chest pain described as a pressure sensation, fullness, or squeezing in the midportion of the thorax
- Radiation of chest pain into the jaw or teeth, shoulder, arm, and/or back
- Associated dyspnea or shortness of breath
- Associated epigastric discomfort with or without nausea and vomiting
- Associated diaphoresis or sweating
- Syncope or near-syncope without other cause
- Impairment of cognitive function without other cause

An MI may occur at any time of the day, but most appear to be clustered around the early hours of the morning, are associated with demanding physical activity, or both. Approximately 50% of patients have some warning symptoms (angina pectoris or an anginal equivalent) before the infarct **(Quitnet http://www.quitnet.com/).**

1.2.6 Diagnosis of myocardial infraction:

Diagnosis of myocardial infarction is most important; we can diagnosis by several ways. In some patients, coronary artery disease is first diagnosed when they present with the severe pain and haemodynamic disturbance of acute infarction, usually as a result of sudden thrombotic osculation of an atherosclerotic coronary artery. On close questioning, there may be a history of some chest discomfort prior to the infract, but in some cases no warning at all **(Henderson AH, weatherall DJ, 1996)**

We can diagnosis the MI by-

Resting electrocardiogram:

The ECG is often normal, pathological Q waves almost always indicate myocardial infarction and other causes are uncommon. Finding Q waves, therefore, has considerable specificity but it is insensitive for the diagnosis of coronary artery disease. The ECG may also show changes suggestive of other diagnosis including pericarditis, LV hypertrophy right heart strain, and arterial fibrillation.

Chest X-ray:

This is usually normal, but if there is cardiac failure there may be increased cardiothoracic ratio and pulmonary venous congestion. The aortic contour may give a clue to aortic dissection or thoracic aortic aneurysm, but is not reliable in this regard. Other causes of chest pain may be suggested by pleural shadowing, radiographic features pulmonary hypertension, masses in the lung fields or mediastinum, and the fluid level of a hiatus hernia behind the heart.

Blood test:

Full blood count to detect any anemia, thyroid function tests to detect thyrotoxicosis (high output state, angina) or hypothyroidism (hyper –lipidaemia and coronary disease), and urea and electrolytes to asses renal function are usually performed. A lipid profile including HDL, LDL triglycerides, and glucose measurement are also routine as they may require management in their own right. Additionally, very high cholesterol or a new finding of impaired glucose tolerance would way the diagnostic probabilities towards coronary artery disease **(HofgreenC, Karlson BW, HerlitzJ, .1995).**

1.3. Risk factors of MI:

Risk factors for atherosclerosis are generally risk factors for myocardial infarction:

Males are more at risk than females **(Wilson PW, D'Agostino RB, 1998; 97).**

Socioeconomic factors such as a shorter education and lower income (particularly in women), and unmarried cohabitation may also contribute to the risk of MI. To understand epidemiological study results, it's important to note that many factors associated with MI mediate their risk via other factors. For example, the effect of education is partially based on its effect on income and marital status. Women who use combined oral contraceptive pills have a modestly increased risk of myocardial infarction, especially in the presence of other risk factors, such as smoking (**Nyobe, Jensen G, Appleyard M, 1991, Khader YS, Rice J, 2003).**

The following risk factors have been associated with a higher incidence of myocardial infarction. Some of these risk factors are controllable (such as smoking and physical activity) while others are uncontrollable (such as age, genetics, and family history).

Age: Four out of five patients with coronary artery disease are 65 years of age or older. After menopause, females are more likely to die within the first year of having a myocardial infarction than males.

Gender: Males are at higher risk of myocardial infarction than women, and males are also more likely to suffer myocardial infarction earlier in life. However, heart disease kills more females each year than any other disease, including breast cancer. An alarming survey reported by the American Heart Association found that only 8% of women perceive heart disease as the greatest threat to their health despite the fact that heart disease is the leading cause of death among both women and men. Over 500,000 American women die from cardiovascular disease each year--twice the number of deaths from all cancers combined. Also, women are more likely to die within the first year of a heart attack than men.

Family history/race: A family history of heart disease increases the risk of coronary artery disease and myocardial infarction. In the United States, African Americans tend to

have more severe high blood pressure than Caucasians, increasing coronary artery disease/myocardial infarction risk. The incidence of heart disease is also higher among certain population groups such as Mexican Americans, American Indians, native Hawaiians and some Asian Americans.

Smoking: Cigarette smokers are twice as likely to experience myocardial infarction compared to non-smokers. Smokers also have a two to four time higher risk of sudden cardiac death (within an hour of a heart attack).

High blood pressure (hypertension): Alone or in association with obesity, smoking, high blood cholesterol levels or diabetes, high blood pressure increases the risk of myocardial infarction and stroke.

High blood cholesterol: High total and low-density lipoprotein (LDL cholesterol) levels and low HDL cholesterol levels increase the risk of myocardial infarction Cholesterol levels can be lowered with dietary/lifestyle modifications such as exercise or medications.

Obesity: Obesity is a major risk factor for cardiovascular disease; Obesity increases coronary artery disease, myocardial infarction, and stroke risk. Obesity increases strain on the heart, raises blood pressure and cholesterol, and increases diabetes risk. Weight reduction can be achieved with modifications to diet and increased physical activity.

Diabetes: Approximately two-thirds of patients with diabetes die from heart or blood vessel disease. Adults with diabetes are three to seven times more likely to develop heart disease. A recent recommendation from the U.S. government advocates aggressive treatment of high cholesterol in people with diabetes.

Lack of physical activity: Regular exercise reduces the risk of coronary artery disease and myocardial infarction by controlling blood cholesterol levels, decreasing the risk of obesity or diabetes, and lowering blood pressure levels in some patients.

Stress: Research indicates a possible relationship between stress and coronary artery disease, which may lead to myocardial infarction Hypertension (high blood pressure) and

high cholesterol are associated with stress, as are increased tendencies to smoke, gain weight and/or decrease physical activity **(the women's health resources on the web science 1999).**

Modifiable risk factors of mi:

Everybody has a risk of developing atheroma which can lead to an MI. However, certain 'risk factors' increase the risk and include:

Preventable or treatable risk factors:

- Smoking
- Hypertension (high blood pressure)
- High cholesterol level
- Lack of exercise
- A poor diet
- Obesity
- Excess alcohol

Having diabetes. But if you have diabetes, the increased risk of heart disease is minimized by good control of the blood sugar level, and reducing blood pressure if it is high.

Tobacco smoke: smoker's risk of MI is more than twice that of nonsmokers. Cigarette smoking is the biggest risk factor for sudden cardiac death. Smokers have two to four times the risk of nonsmokers. Cigarette smoking also acts with other risk to greatly increase the risk for coronary heart disease. It is responsible for 25%of CHD deaths under 65 years of age in men **(WHO, technical Report, 1979).**

Cigarette smoke oxidizes LDL and unmasks its binding to scavenger cell receptors. Adaptation to smoking decreases HDL, but increases on cessation of smoking **(Gershilick AH and Davis SW, 2001; Allen RA. Kluft C, 1985).**

Among all the risk factors, smoking cigarette showed evidence to increase the risk of MI. **(Savotham SG Berry JN, 1968).** Cigarette smoking may be an important risk factors associated with myocardial infarction among the people residing in rural area **(Guptal R, Gupta VP, Ahluwalia NS, 1994).**

Diabetes mellitus: Diabetes seriously increases the risk of developing MI. Even when glucose levels are under control, diabetes greatly increases the risk of heart disease and stroke. The overall prevalence of MI is as high as 55% among the patient with diabetes compared with 2-4% for general population without diabetes. About two thirds of the people with diabetes die of some form of heart **(MaCconel RB, 1996).**

About diabetes in different countries: More than seven million diabetes cases in the 46 countries of the WHO African Region in 2000 resulted in a total economic loss of US$25.51 billion. Diabetes and the presence of prior coronary disease are important risk factors for initial and recurrent coronary heart disease in persons infected with HIV. Given the increased life expectancy of HIV-infected persons after the use of effective antiretroviral therapy, prevalence of diabetes and CHD risk are likely to increase. South Asians with type 2 diabetes are significantly more at risk of losing their eyesight and this may occur up to seven years earlier than their white European counterparts. Only one-third of 39.8 million diabetics in China are aware of their condition, according to the Chinese Ministry of Health. The number of diabetics in urban areas has increased by 39% in the past six years. Diabetes cases in the UK increased by 74% between 1997 and 2003, an increase linked to growing obesity rates. Diabetes prevalence increased from 2.8% in 1996 to 4.3% in 2005 **(Procor, 2009).**

High blood pressure: Hypertension has been established unequivocally as a risk factor in that individual with accelerated atherosclerogenosis, an increased incidence of coronary heart disease (CHD). Hypertension is probably directly or indirectly responsible for 10-20% of all deaths **(Thomas J, Leaverton PE, 1992).**

High blood pressure increases the heart's workload, causing the heart to enlarge and weaken. It also increases the risk of atherosclerosis; both the systolic and diastolic hypertension is associated risk of MI. the risk is same for men and woman. While reduction of blood pressure reduces the risk of cerebrovascular events. It does not appear to affect the risk of MI **(Islam N, 1983. Gershilick AH, 2001).**

Elevated blood pressure or hypertension emerges as a major cause of stroke, hypertensive heart disease, and hypertensive kidney failure even before coronary heart disease and atherothrombotic stroke become major causes of mortality **(Whelton et al., 1995).**

Hypercholesterolemia: High serum cholesterol, especially when associated with a low value of high-density lipoprotein (HDL), is strongly associated with coronary atheroma. There is increasing evidence that high serum triglyceride is also independently linked with coronary atheroma. Familial hypercholesterima, combined with hypertriglyceridemia and remnant hyperlipidemia are also associated with MI **(Phillips WR, 1993).**

Although some investigators have shown a strong association of total cholesterol and LDL, with coronary artery disease **(Ramachandran A, Sathyamurthy I, 2001).** Other findings suggest that this disease arises at lower lipid concentration in people from south Asia then in those from other regions **(Krishnaswarmi S, Prasad N K, 1989).** On the other hand characteristics lipid abnormalities, such as high triglycerides and low HDL with normal LDL values are common in American recommendation of the use of statins as first-line agents may not be entirely applicable to all population. Only data from heart protection study clearly show benefit with LDL reduction from 2.5 mmol/L, where 24% reduction in even was observed. This suggest that clinical endpoint trials will be required in the South Asian setting to define the best therapeutic strategy for treatment of CHD. Cost of coronary prevent might not be prohibitive, if generic drugs are used. Rural communities have lower risk factors than urban westernized populations, this difference may be attributable to a low fat (15-20 g per day) diet based on whole grain (400g per day) combined with physically demanding occupations. With the rapid rural to urban migration in South Asia. Risk reduction strategies should therefore focus on protection of health life style **(Bhatragar D, Anand I S, 1995) (Singh R B, Sharma J P, 1997).**

The factor most important in causing atherosclerosis is a high blood plasma concentration of cholesterol in the form of low-density lipoproteins. As explained, the plasma concentration of these high-cholesterol low-density lipoproteins is directly increased by eating highly saturated fat in the daily diet. To a lesser extent, it is also increased by eating increased cholesterol in any form. Therefore, both or either of these dietary indiscretions can contribute to the development of atherosclerosis. An interesting

example of this occurs in rabbits that normally have low plasma cholesterol concentration because of their vegetarian diet. Simply feeding these animals large quantities of cholesterol as part of their daily nutrition will lead to serious atherosclerotic plaques all through their arterial system **(C.Guyton, M.D., John E. Hall, Ph.D., and tenth edition).**

Obesity: Obesity is a major risk factor for cardiovascular disease; Obesity is important in-patient with cardiac disease of any type because the demands on the heart are increased. Overweight and obesity have become increasingly common; worldwide, at least 1.1 billion adults are overweight and 312 million are obese, when overweight and obesity are defined conventionally as having a body mass index (BMI) of >25 kg/m^2 and >30 kg/m^2, respectively. In the general population, overweight and obesity are associated with increased risk of developing cardiovascular disease, and thus it is not surprising that in cohorts of patients with prevalent ischemic heart disease or acute coronary events, well over 50% are overweight or obese. Obesity is associated with increased prevalence of cardiovascular risk factors, such as hypertension, diabetes mellitus. It is an important risk factor of coronary heart disease **(Islam N, Khan M, Latif ZA, et Al, 1983, Haslam DW, James WPT. 2005; World Health Organization. Obesity and overweight. May 30, 2008, Yusuf S, Hawken S, Ôunpuu S, Bautista L, 2005; Calle EE, Thun MJ, Petrelli JM, 1999; Mehta L, Devlin W, McCullough PA, 2007; Steinberg BA, Cannon CP, 2007).**

Obese adolescents have the same risk of premature death in adulthood as people who smoke more than 10 cigarettes a day, while those who are overweight have the same risk as less heavy smokers.

Children who watch too much television, drink sweet beverages, and skip breakfast are more likely to be overweight or obese. Eliminating TV advisement for unhealthy foods could drastically reduce prevalence of childhood obesity; from 17.8% to 15.2% for boys and from 15.9% to 13.5% for girls **(Professor Joseph O.M. Pobee, 1974).**

Non-Modifiable risk factors of MI:

Risk factors that are fixed and we cannot change:

Age: About four out of five people who die of coronary heart disease are 65 or older. At older ages, woman who have heart attacks are more likely than men to die from them within a few weeks. MI rates increases with age. Atherosclerosis is rare in childern, except in familial hyperlipidemia but is often detectable in young men between 20-30 years of age. It is almost universal in the elderly in the west **(Am, Heart J, 1986).** African Americans are 20 times more likely to develop heart failure before age 50 years than whites the same age, and their risk is linked to risk factors earlier in adulthood **(Procor, 2009).** Age is important because women's risk of heart disease starts to rise during middle age, in part because of the drop in estrogen levels that comes with menopause. But middle age also is when many women develop heart disease risk factors **(Robert W. Griffith, MD, November 28, 2002).**

Male sex (gender): Men have a greater risk of MI than woman do, and they have attacks earlier in life. Eevn after menopause, it's not as great as men's. The Framingham Heart Study presented prospective population data showing possible sex based differences in initial clinical menifestation of CHD. Pattern of coronary heart disease morbidity and mortality in hearrt disease in the sexes **(Am Heart J, 1986).** Males are more at risk than females **(Wilson PW, D'Agostino RB, Levy D, Belanger AM, Silbershatz H, Kannel WB. 1998)**

Female sex (gender): Women who use combined oral contraceptive pills have a modestly increased risk of myocardial infarction, especially in the presence of other risk factors, such as smoking. ***(Khader YS, Rice J, John L, Abueita O, 2003)***. About two-thirds of the women who have a heart attack do not make a full recovery. Heart disease is the #1 killer of American women. One of every three deaths for American women is from heart disease - only one in every thirty death is from breast cancer. Women's heart disease risk starts to rise in middle age. One in eight American women aged 45-64, and one in three women over 65, have some form of heart disease. Nearly two-thirds of American women who die suddenly of a heart attack have no prior symptoms. About two-thirds of American woman women who have a heart attack do not make a full recovery **(Robert W. Griffith, MD, November 28, 2002).**

Socioeconomic factors: such as a shorter education and lower income (particularly in women), and unmarried cohabitation may also contribute to the risk of MI. To understand epidemiological study results, it's important to note that many factors associated with MI mediate their risk via other factors. For example, the effect of education is partially based on its effect on income and marital status ***(Nyboe J, Jensen G, Appleyard M, Schnohr P.1989).*** An unhealthy dietary intake, assessed by a simple dietary risk score, increases the risk of AMI globally and accounts for 30% of the population-attributable risk **(Romaina Iqbal, PhD; Sonia Anand, MD; Stephanie Ounpuu, PhD; Shofiqul Islam, MSc; Xiaohe Zhang, MSc; Sumathy Rangarajan, MSc; Jephat Chifamba, DPhil; Ali Al-Hinai, MD; Matyas Keltai, MD; Salim Yusuf, DPhil, November 21, 2007).**

Family history: Family history of MI is positively associated with the risk of early MI in women. While the association with parental history of MI is mediated through the clustering of other common risk factors, the association of sibling history of MI with early-onset MI in young women is only partially explained by the clustering of established and newly identified risk factors **(Robert J Bryg, MD on March 07, 2009).**

a family history of heart disease or a stroke that occurred in a father or brother aged below 55, or in a mother or sister aged below 65being male. Ethnic group (for example, British Asians have an increased risk).

Risk factors are discussed more fully in another leaflet called 'Preventing Heart Disease'. Briefly, if you can reduce any risk factors, it reduces your risk of having an MI (or of having a further MI if you have already had one). Some risk factors are fixed and you cannot change them. However, if you have a fixed risk factor, you may want to make extra effort to reduce preventable risk factors such as smoking or lack of exercise. No authors listed; **(Reperfusion in acute myocardial infarction. Drug Ther Bull. 2005 July).**

1.4. Prevention of Myocardial infarction:

Priorities of CDC's Heart Disease and Stroke Prevention Program

- **Control high blood pressure**
- **Control high cholesterol**
- **Know signs and symptoms,**
- **Improve Emergency Response**
- **Improve quality of care (prevent first and second events; control risk factors and the diseases)**
- **Eliminate disparities**

Prevention and disease control can reduce heart attacks and strokes, deaths from these diseases, and the disability suffered by heart disease and stroke survivors.

In 2002, age–adjusted death rates for heart disease were 30% higher for African Americans than for whites, and stroke death rates were 41% higher **(Division for Heart Disease and Stroke Prevention, National Center for Chronic Disease Prevention and Health Promotion)**

1.5. TREATMENT OF MI:

The risk of a recurrent myocardial infarction decreases with strict blood pressure management and lifestyle changes, chiefly smoking cessation, regular exercise, a sensible diet for patients with heart disease, and limitation of alcohol intake.

Patients are usually commenced on several long-term medications post-MI, with the aim of preventing secondary cardiovascular events such as further myocardial infarctions, congestive heart failure or cerebrovascular accident (CVA). Unless contraindicated, such medications may include: [**Rossi S, editor. Australian Medicines Handbook 2006, Smith A, Aylward P, Campbell T, 2003)**

- **Antiplatelet drug therapy** such as aspirin and/or clopidogrel should be continued to reduce the risk of plaque rupture and recurrent myocardial infarction. Aspirin is first-line, owing to its low cost and comparable efficacy, with clopidogrel reserved for patients intolerant of aspirin. The combination of

clopidogrel and aspirin may further reduce risk of cardiovascular events; however the risk of hemorrhage is increased **(Peters RJ, Mehta SR, Fox KA, Zhao F, 2003).**

- **Beta blocker therapy** such as metoprolol or carvedilol should be commenced. These have been particularly beneficial in high-risk patients such as those with left ventricular dysfunction and/or continuing cardiac ischemia. β-Blockers decrease mortality and morbidity. They also improve symptoms of cardiac ischemia in NSTEMI(**Yusuf S, Peto R, Lewis J, Collins R, Sleight P 1985, Dargie HJ 2001).**

- **ACE inhibitor therapy** should be commenced 24–48 hours post-MI in hemodynamically-stable patients, particularly in patients with a history of MI, diabetes mellitus, hypertension, anterior location of infarct (as assessed by ECG), and/or evidence of left ventricular dysfunction. ACE inhibitors reduce mortality, the development of heart failure, and decrease ventricular remodelling post-MI (**Pfeffer MA, Braunwald E, Moye LA, Basta L, 1992)**

- **Stain therapy** has been shown to reduce mortality and morbidity post-MI. The effects of statins may be more than their LDL lowering effects. The general consensus is that statins have plaque stabilization and multiple other ("pleiotropic") effects that may prevent myocardial infarction in addition to their effects on blood lipids (**Ray KK, Cannon CP 2005).**

- **The aldosterone antagonist** agent eplerenone has been shown to further reduce risk of cardiovascular death post-MI in patients with heart failure and left ventricular dysfunction, when used in conjunction with standard therapies above **(Keating G, Plosker G 2004).**

- **Omega-3 fatty acids**, commonly found in fish, have been shown to reduce mortality post-MI. While the mechanism by which these fatty acids decrease mortality is unknown, it has been postulated that the survival benefit is due to electrical stabilization and the prevention of ventricular fibrillation. However, further studies in a high-risk subset have not shown a clear-cut decrease in potentially fatal arrhythmias due to omega-3 fatty acids(**Brouwer IA, Zock PL, Camm AJ, Bocker D,2006, Raitt MH, Connor WE, Morris C, Kron J,2005).**

1.6. High cholesterol:

Cholesterol is a soft, waxy fat particle (lipid) that circulates in the blood. It is produced in the liver and is the most common steroid in the body. Cholesterol is a building block for cell membranes and it is essential in the formation of bile (which aids in the digestion of fats), vitamin D, and other steroids and hormones (e.g., progesterone, estrogen, testosterone).

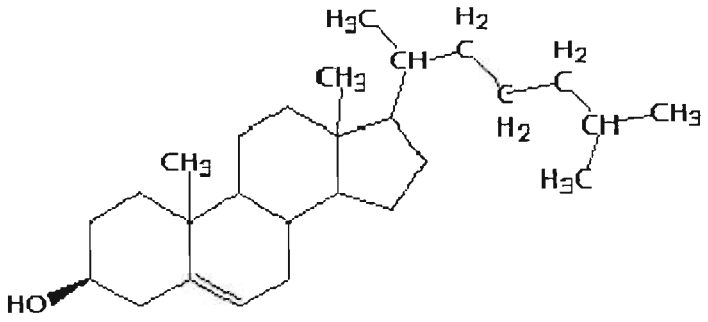


Fig: cholesterol

The liver produces most of the cholesterol the body needs; however, many popular foods contain cholesterol and substances used by the liver to produce cholesterol. A high intake of these foods can increase the level of cholesterol in the blood. **(Stanley J. Swierzewski, 01 Jul 2000).**

Lipids, such as cholesterol and triglycerides, are fats that are an integral part of cells, and that may dissolve in alcohol, but are insoluble in water. They are thus insoluble in the blood. In order for lipids to be transported in blood, they are packaged as lipoprotein. Lipoproteins have a shell of phospholipids and proteins that allow them to dissolve in blood. The lipids, transported as lipoprotein, are transported to various tissues for energy utilization, lipid deposition, steroid hormone production, and bile acid formation.

High cholesterol is a major modifiable risk factor for cardiovascular diseases, and it is essential that the informed consumer understand the importance of an elevated cholesterol, as a risk factor for cardiovascular diseases; target levels of cholesterol that may necessitate treatment of high cholesterol levels; and treatments available to lower blood cholesterol levels **(Libby, P. Atherosclerosis The new view. Sci Am 286; 5:47.)**

High cholesterol (**hypercholesterolemia**) can cause the formation and accumulation of plaque deposits in the arteries. **Plaque** is composed of cholesterol, other fatty substances, fibrous tissue, and calcium. When it builds up in the arteries, it results in atherosclerosis, or coronary heart disease (CHD). **Atherosclerosis** can lead to plaque ruptures and blockages in the arteries, which increase the risk for heart attack, stroke, circulation problems, and death.

The development of plaques and blockages in the arteries involves several steps.

1. When the innermost lining of the arteries (**endothelium**) is damaged, cholesterol particles deposit into the damaged wall and form plaques (see figure below).
2. More cholesterol and other substances incorporate into the plaque and the plaque grows, narrowing the artery (Step 2).
3. Plaque deposits can grow large enough to interfere with blood flow through the artery (called a **blockage**) (Step 3-4). When the arteries supplying the heart with blood (coronary arteries) are blocked, chest pain (angina) may occur; when arteries in the legs are blocked, leg pain or cramping may occur; when arteries supplying the brain with blood are blocked, stroke may occur.
4. If a plaque ruptures or tears, a blood clot may develop on top of it (Step 5). If a blood clot completely blocks blood flow through a coronary artery, heart attack (myocardial infarction) occurs; if an artery supplying blood to the brain is completely blocked, stroke occurs **(Stanley J. Swierzewski, III, M.D. 01 Jul 2000).**

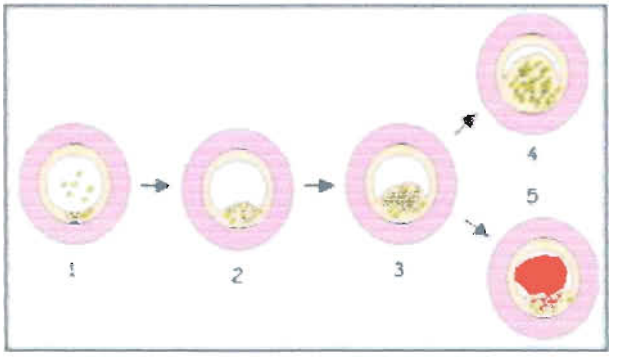


Fig: development of plaques and blockages

1.6.1. Types of Cholesterol:

Cholesterol is measured in milligrams per deciliter of blood (mg/dL). Several different types of blood cholesterol can be measured, and high levels of some types are worse or better than high levels of other types. Types include the following:

- Total blood cholesterol
- HDL (high-density lipoprotein) cholesterol ("good" cholesterol)
- LDL (low-density lipoprotein) cholesterol ("bad" cholesterol)
- Triglycerides ("backbone" of many types of fat)

Total blood cholesterol: is the most common cholesterol measurement. It measures the concentration of fat (lipid) in the bloodstream, including cholesterol and triglyceride molecules contained in LDL, HDL, and other lipid particles.

Total blood cholesterol levels can be used to help determine if LDL and triglyceride levels are likely to be normal or elevated. If total cholesterol levels are elevated, a **lipid profile** is used to determine which lipid level is too high.

HDL ("good") cholesterol: may help protect against atherosclerosis by preventing cholesterol from depositing on arterial walls as it circulates in the bloodstream. Low HDL levels may be caused by a genetic predisposition, lack of exercise, smoking, and/or obesity.

Some physicians believe it is important to assess the **ratio** between total blood cholesterol and HDL cholesterol. The ratio is calculated by dividing the HDL number into the total cholesterol number. For example, total cholesterol of 200 mg/dL and an HDL of 40 mg/dL would yield a ratio of 5:1 (200/40). A ratio below 5:1 is desirable and the optimum ratio is about 3.5:1. **(Stanley J. Swierzewski, III, M.D. 01 Jul 2000).**

Low density lipoprotein — LDL particles contain a core of cholesterol esters, lesser amounts of triglyceride, and are enriched in apolipoprotein B-100, which is the ligand for binding to the apolipoprotein B/E (LDL) receptor. LDL can be internalized by hepatic and nonhepatic tissues. Hepatic LDL cholesterol can be converted to bile acids and secreted into the intestinal lumen. LDL cholesterol internalized by nonhepatic tissues can

be used for hormone production, cell membrane synthesis, or stored in the esterified form.

Lipoprotein levels are highly correlated with the severity of atherosclerosis in various vascular beds. Lipoprotein levels not only modulate the risk of coronary artery disease and its clinical events in patients with hypercholesterolemia but also determine the outcome following percutaneous coronary angioplasty. More recently in patients with hypertension lipoprotein levels were found to be predictive of target organ damage including angina, myocardial infarction, left ventricular hypertrophy and heart failure (**Rimmm EB,Stmpfer MJ,1997; SosanM, Czajkowshi,1998; Bruce B. Duncan,1999)**

Circulating LDL can also enter macrophages and some other tissues through the unregulated scavenger receptor. This pathway can result in excess accumulation of intracellular cholesterol and the formation of cholesterol-enriched cells (called foam cells) that contribute to the formation of fatty deposits, inflammatory cells and smooth muscle cells in the lining of the arteries, called atheromatous plaques **(The Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on detection, evaluation, and treatment of high blood cholesterol in adults (Adult Treatment Panel III). Circulation 2002)**

Triglycerides:

Triglycerides, as major components of very low density lipoprotein (VLDL) and chylomicrons, play an important role in metabolism as energy sources and transporters of dietary fat. They contain more than twice as much energy (9 kcal/g) as carbohydrates and proteins.

Triglycerides cannot pass through cell membranes freely. Special enzymes on the walls of blood vessels called lipoprotein lipases must break down triglycerides into free fatty acids and glycerol. Fatty acids can then be taken up by cells via the fatty acid transporter (FAT).

In the human body, high levels of triglycerides in the bloodstream have been linked to atherosclerosis, and, by extension, the risk of heart disease and stroke. However, the relative negative impact of raised levels of triglycerides compared to that of LDL: HDL

ratios is as yet unknown. The risk can be partly accounted for by a strong inverse relationship between triglyceride level and HDL-cholesterol level(**American Heart Association.. Retrieved 2009-05-22).**

The national heart, lung and blood institute issued the third report of the national cholesterol education program (NCEP) expert panel on, evaluation, and treatment of high blood cholesterol in adults (adult treatment panel-3) in may 2001.The ATP3 report presented the NCEP'S updated clinical guidelines for cholesterol testing and management and described the following classifications for cholesterol and triglyceride testing.

Adult lipid classification:

Table: 1

Test	optimal	Near optimal/above optimal	Borderline high	High	Very high
Total cholesterol (mg/dl)	<200	---------	200-239	>240	---------
HDL-cholesterol(mg/dl)	<40-low ≥60-high	---------	----------	---------	---------
LDL-cholesterol(mg/dl)	<100	100-129	130-159	160-189	≥190
Triglycerides(fasting) (mg/dl)	<150	--------	150-199	200-499	≥500

1.6.2. Abnormalities occur due to high blood cholesterol:

When our body has too much cholesterol, it builds up on the walls of our arteries. Over time this buildup will cause "hardening of the arteries". This hardening means that our arteries become narrowed and blood flow to the heart becomes limited.

Our blood carries oxygen to our heart. If enough oxygen and blood cannot reach our heart, we may suffer chest pains. If the blood supply to certain parts of our heart is cut off, by a blockage, the result is a heart attack.

The higher blood cholesterol level, the higher chances of getting heart disease or having a stroke. This would make high blood cholesterol level a "risk factor". A "Risk Factor" is a condition that elevates or raises chances of getting a disease.

High blood cholesterol levels by themselves have no visible symptoms. This is why so many people are unaware they have high cholesterol levels. This is also the reason why it is so important for people (of all ages) to determine their cholesterol levels.

An excess of either total or LDL cholesterol in the blood is a risk for heart disease and atherosclerosis. People can have an excess of cholesterol because of diet and because of the rate at which cholesterol is processed in the body. Most of the excess cholesterol comes from diet. Cholesterol can build up on the artery walls of your body. This buildup is called plaque. Over time, plaque can cause the arteries to become narrow, which is called atherosclerosis. As a result, less oxygen–rich blood can pass through. When the arteries that carry blood to the heart are affected, coronary artery disease can result. A heart attack occurs when a coronary artery becomes completely blocked. A coronary artery can become blocked either by plaque buildup or by a plaque that ruptures or bursts which causes a clot. Angina can also develop because of plaque buildup. Angina happens when the heart does not receive enough oxygen–rich blood **(Cholesterol Prevention and Treatment Strategies,Cardiovascular Disease, Self Management Program)**

Researchers have found that changes in LDL are mainly a result of liver dysfunction caused by the following:

- Liver dysfunction is genetically inherited, more probable in early onset of problem with no disease or aging indicators.
- Liver dysfunction occurs as part of the aging process.
- Liver dysfunction occurs from disease, diabetes being the most common.

A defect in the receptor protein for LDL in the liver has been found to be a cause for high LDL. This defect is considered to be genetic, although dietary environment and an unhealthy lifestyle could precipitate liver dysfunction, and often does with the aging process and with disease, diabetes being the most common. The LDL receptor from the liver is responsible for removing cholesterol from the blood; and once the LDL is bound to the receptor a signal is sent for the body to cease producing LDL.

Damaged receptors do not send the "Stop Production" signal, and result in excessive LDL levels. Also, a diet high in saturated fat decreases the number of LDL receptors, and thereby reduces "Stop Production" feedback. Another association to LDL levels is Low Thyroid Function. Although not as common, individuals with thyroid problems need to realize this association.

1.6.3. Prevention of high blood cholesterol:

High blood cholesterol is a major risk factor for heart disease. There are a number of things that can be done to maintain normal cholesterol levels and reduce the risk of developing heart disease. All people at any age can take steps to keep normal cholesterol levels. People with high total cholesterol, high LDL cholesterol, or low HDL cholesterol should talk with their doctor about the best way to control or improve their cholesterol.

What affects cholesterol levels?

A number of things can affect the cholesterol levels in blood. These include the following:

- **Diet.** Certain foods have types of fat that raise your cholesterol level. These types of fats include saturated fat, trans fatty acids or trans fats, and dietary cholesterol. Saturated fats come largely from animal fat in the diet, but also some vegetable oils such as palm oil. Trans fats are made when vegetable oil is hydrogenated to harden it. Research suggests that trans fatty acids can raise cholesterol levels. Dietary cholesterol is found in foods that come from animal sources such as egg yolks, meat, and dairy products.

- **Weight.** Being overweight tends to increase LDL levels, lowers HDL levels, and increases total cholesterol level.

- **Physical Inactivity.** Lack of regular physical activity can lead to weight gain, which could raise your LDL cholesterol level.

- **Heredity.** High blood cholesterol can run in families. An inherited genetic condition results in very high LDL cholesterol levels. This condition is called familial hypercholesterolemia.

- **Age and Sex.** As people get older, their LDL cholesterol levels tend to rise. Men tend to have lower HDL levels than women. Younger women tend to have lower LDL levels than men, but higher levels at older ages (after age 55 years).

What can we do?

Have our cholesterol checked. There are usually no signs or symptoms of high blood cholesterol, so it is important to have your blood cholesterol checked. A simple blood test can be done by your doctor to check your blood cholesterol level. A lipoprotein profile can be done to measure several different kinds of cholesterol as well as triglycerides (another kind of fat found in the blood).

Desirable or optimal levels for adults with or without existing heart disease are

- Total cholesterol: Less than 200 mg/dL.
- Low Density Lipoprotein (LDL) cholesterol ("bad" cholesterol): Less than 100 mg/dL.
- High Density Lipoprotein (HDL) cholesterol ("good" cholesterol): 40 mg/dL or higher.
- Triglycerides: Less than 150 mg/dL.

Maintain a Healthy Diet. An overall healthy diet can help to maintain normal blood cholesterol levels. Saturated fat, trans fats, and dietary cholesterol tend to raise blood cholesterol levels. Other types of fats, such as monounsaturated and polyunsaturated fats can help to lower blood cholesterol levels. Getting enough soluble fiber in the diet can also help to lower cholesterol. For some people, a diet that has too many carbohydrates can lower HDL (the good cholesterol) and raise triglycerides. Alcohol can also raise triglycerides, and excessive alcohol use can lead to high blood pressure, another risk factor for heart disease and stroke (**healthy diet and nutrition, see CDC's Nutrition and Physical Activity Program Web site**).

Maintain a Healthy Weight. Being overweight or obese can raise our bad cholesterol levels. Losing weight can help you lower your blood cholesterol levels. Healthy weight status in adults is usually assessed by using weight and height to compute a number called the "body mass index" (BMI). BMI is used because it relates to the amount of body fat for most people. An adult who has a BMI of 30 or higher is considered to be obese. Overweight is a BMI between 25 and 29.9. Normal weight is a BMI of 18.5 to 24.9. Proper diet and regular physical activity can help to maintain a healthy weight. Other measures of excess body fat may include waist measurements or waist and hip measurements. **(BMI at the CDC's Nutrition and Physical Activity Program Web site).**

Be Active. Physical activity can help to maintain a healthy weight and lower blood cholesterol levels. The Surgeon General recommends that adults should engage in moderate–level physical activities for at least 30 minutes on most days of the week. **(CDC's Nutrition and Physical Activity Program Web site).**

No Tobacco. Smoking injures blood vessels and speeds up the process of hardening of the arteries. Further, smoking is a major risk for heart disease and stroke. If you don't smoke, don't start. Quitting smoking lowers one's risk of heart attack and stroke **(CDC's Tobacco Intervention and Prevention Source Web site).**

Genetic Factors. Genes can play a role in high blood cholesterol. Very high blood cholesterol levels can be related to a condition known as familial hypercholesterolemia. It is also possible that high blood cholesterol levels within a family are due to factors such as common diet **(CDC's National Office of Public Health Genomics Web site).**

1.6.4. Medications of Hyperlipidimia:

If found high blood cholesterol, doctor may prescribe medications, in addition to lifestyle changes, to help bring it under control. The primary focus of treatment is to get LDL cholesterol under control. Treatment plan and goal will depend on LDL level and level of risk for heart disease and stroke. Risk for heart disease and stroke will be based on whether also have other risk factors and may include blood pressure level or high blood pressure treatment, smoking status, age, and HDL level, family history of early heart disease, and existing cardiovascular disease or diabetes. People with existing

cardiovascular disease or diabetes are considered high risk. heart disease by using the 10-year **(The National Cholesterol Education Program at the National Heart, Lung, and Blood Institute's Web site.)**

Various medications can lower blood cholesterol level. They may be prescribed individually or in combination with other drugs.

- **Statin drugs** lower LDL cholesterol by slowing down the production of cholesterol and by increasing the liver's ability to remove the LDL–cholesterol already in the blood. Among the most commonly prescribed medication for lowering cholesterol.

Statins currently available in the U.S.

-Atrovastatin (Lipitor), Fluvastatin (Lescol), Lovastatin (Altroprev), Paravastati (Paravachol), Rosuvastatin (Crestor), Simvastatin (Zoocor)

- **Bile acid sequestrants** help to lower LDL cholesterol by binding with cholesterol-containing bile acids in the intestines, and are then eliminated in the stool. The medications are cholestyramine (Prevalite, Questran), Colesevelam (Welchol) and Colestepo (Colestid).

- **Niacin**, or nicotinic acid, is a B vitamin that can improve all lipoproteins. Nicotinic acid lowers total cholesterol, LDL-cholesterol, and triglyceride levels, while raising HDL-cholesterol levels. Because the levels needed are well above recommended dietary intake levels, niacin treatment for cholesterol should only be done only under medical supervision because of possible adverse side effects.

- **Fibrates** are used mainly to lower triglycerides and, to a lesser extent, to increase HDL levels. Available fibrates drugs are Gemfibrozil (Lopid), Finofibrate (Antara, Tricor), and Clofibrate (Atromid).

All drugs may have adverse side effects, so their use needs to be checked by doctor on a usual basis.

1.6.5. Intervention:

Lipids, such as cholesterol and triglycerides, are fats that are an integral part of cells, and that may dissolve in alcohol, but are insoluble in water. They are thus insoluble in the blood. In order for lipids to be transported in blood, they are packaged as lipoprotein. Lipoproteins have a shell of phospholipids and proteins that allow them to dissolve in blood. The lipids, transported as lipoprotein, are transported to various tissues for energy utilization, lipid deposition, and steroid hormone.

By changing life style we can change our lipid profile from higher to lower in an acceptable range.

Intensive drug intervention with diet suggests that drug can lower serum cholesterol concentration, and lipid lowering medicine have the direct rule on reducing the serum concentration of lipid to an acceptable level. Niacin, statins, resins, fibrates may after substantial benefit for person with high serum cholesterol concentration. Nutrition guidelines proposed by the national advisory committee for nutrition, education (NACNE) in 1983 in collaborations with committee of medical aspects of food policy (CMOKAO) in 1984. Both committees recommended that total fat and energy reduction by 30% lowers the risk of heart disease **(Blankenhorn DH. Two new diet heart studies N.Engj. Med .1986; 312:851-52).**

Present study is to investigate the effect of lipid lowering drugs on blood lipids on Ml patients. WE did this study in National Institute of Cardiovascular Diseases & Hospital (NICVD).Here we work with 60 patients which show the positive result that lipid lowering drug reducing the serum cholesterol concentration of lipid to an acceptable level. Life style change such as exercising and eating a healthy diet are the first line of against high cholesterol.

Serum cholesterol concentration of lipid can also be lowering by following factor-

Dietary modification:

Rigorous dieting can improve cholesterol level, though generally only of the order of 0.3-0.6 mmol/l. In the dietary component, however, only about 10% of total calories were made up of fat. This intake is very markedly below most dietary levels in the western world. A study incorporating diet in the management of coronary artery disease was the St Thomas' Atherosclerosis regression study (STARS).Although an examination

of cholestetyramine added to dietary treatment, the diet alone group in whom the primary aim was to reduce fat intake to 27% of dietary of energy also showed significant falls in LDL cholesterol compared with a control group over 39 months, triglycerides level also reduce **(Department of cardiology, Royal Brmptom, London, UK).**

Physical exercise:

Physical inactivity continues to increase cholesterol level and is associated with increasing levels of obesity. Physical inactivity is itself a risk factor for the development of coronary artery disease. A more rec3ent systematic review suggests that exercise trials peruse have favorably influenced mortality. Recent data from the British Regional Heart Study also suggest that exercise in subjects with coronary artery disease is associated with reduced cardiovascular disease mortality. Physical exercise can aid symptom management after myocardial infarction **(Wannamethee SG, Shaper AG; 2000, GanzP, Braunwald E; 1997).**

Exercise is important life long, but has particular and attractive benefits for the elderly. Longitudinal studies of the age related decline in maximal aerobic performance, as assessed by maximal oxygen uptake, indicate that between 60-90 years minimal oxygen aerobic requirement for survival is reached. Exercise and its contribution to manage lipid, although a substantial amount of debate was engendered by the papers discussed by Curfaman, the essential message is 2-fold; 1.exercise can act as a trigger of myocardial infarction; and 2. Paradoxically, perhaps, exercise taken regularly is nonetheless proactive

Drug:

Various medications can lower blood cholesterol level. They may be prescribed individually or in combination with other drugs.

The specific choices of medication or combination of medications are

- Statins
- Bile-acid-binding resins
- Cholesterol absorption inhibitors
- Combination cholesterol adsorption inhibitor and statin
- Fibrates
- Niacin
- Combination niacin and statin.

All this drugs lowering the serum cholesterol level **(By myoclinic staff 2000)**

1.6.6. Research Questions

- Is there any relationship between high blood serum cholesterol and myocardial infarction?
- What is the relationship between the life style risk factors and myocardial infarction?
- How to control high blood serum cholesterol after 15 days treatment in hospital?

1.6.7. Objective:

The objectives of this study are 1.to find out the relationship between high blood cholesterol and some life style risk factors as smoking habit, food habit, consumption of extra salt, age, education, income, marital status which is related with myocardial infarction. 2. The effect of antilipodial drug for lowering lipid profile within normal range.

So the overall objective of this study are-

- The relationship between high blood cholesterol and myocardial infarction.
- The relationship between life style risk factors and myocardial infarction.
- Effect of antilipodial drug for control of lipid profile with in normal range.

CHAPTER 2

OBJECTIVE OF MI

2.1. Aim of the study:

High blood serum cholesterol (hypercholesterolemia) is a major modifiable risk factor for cardiovascular diseases, and it is essential that the informed patients to understand the importance of an elevated cholesterol, as a risk factor for cardiovascular diseases; target levels of cholesterol that may necessitate treatment of high cholesterol levels; and treatments available to lower blood cholesterol levels. The risks of occurring myocardial infarction are greater if blood cholesterol level is not well controlled.

The aim of our study to evaluate the control of lipid profile of MI patients by antilipodial drug.

2.2. Significance of the study:

High cholesterol (hypercholesterolemia) can cause the formation and accumulation of plaque deposits in the arteries. Plaque is composed of cholesterol, other fatty substances, fibrous tissue, and calcium. When it builds up in the arteries, it results in atherosclerosis, or coronary heart disease (CHD). Atherosclerosis can lead to plaque ruptures and blockages in the arteries, which increase the risk for heart attack, stroke, circulation problems, and death.

The prevalence of high blood cholesterol is increasing day by day. People who have high blood cholesterol have a higher prevalence of myocardial infarction compared to the normal population.

Long term duration of uncontrolled high blood cholesterol may cause various disorders. There are a variety of different lipid disorders (dyslipidemias) that can occur as either a primary event or secondary to some underlying disease. The primary dyslipidemias are associated with overproduction and/or impaired removal of lipoproteins. The later defect can be induced by an abnormality in either the lipoprotein itself or in the lipoprotein receptor. Other disorders are Obesity, physical inactivity, hypertriglyceridemia.

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. Hypercholesterolemia 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 cholesterol level, food habit, and physical activity to avoid the harmful effect of myocardial infarction.

This study is expected to provide important information to better understand the relationship between the blood serum cholesterol and Myocardial Infarction, and utilization of drug and its control of lipid profile with in normal range. Thus, the result of the study is expected to control the lipid profile of Myocardial Infarction in patients which ultimately will help to improve the disease management process.

CHAPTER 3

MATERIALS AND METHOD

3.1 Type of study:

It is a descriptive study. It was attempted to establish relationship between Myocardial Infarction and high blood cholesterol and its control. In addition to this, the study examined for other risk factors and presence of MI.

3.2 Place of study:

The study was being 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:

Sample size was 60

3.5. Sampling Technique:

In this study, purposive sampling technique was followed.

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 following equipments were used in this study,

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, recording blood pressure and anthropometrical measurements examination by checklist. And most important lipid profile examination which was performed by blood has to be taken at the first date of admission and estimation of total cholesterol (TC), serum triglycerides (TG), high density lipoprotein cholesterol (HDL-C) and low density lipoprotein cholesterol (LDL-C) was performed on the sample drawn after 12 hour overnight fast, Than the process was done after 15days later. We show the mean concentration and standard deviation of HDL, LDL, TC, and TG in mg/dl in result at first day than after 15 days of medication while under treatment in NICVD.

3.8.1Blood pressure Measurement:

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

3.8.2. Estimation of lipid profile:

Estimation of total cholesterol (TC), serum triglycerides (TG), high density lipoprotein cholesterol (HDL-C) and low density lipoprotein cholesterol (LDL-C) was performed on the sample drawn after 12 hour overnight fast. TC was estimated with ferric chloride method. Method described by Rosendirg and Gottrified was used for determination of TG. After precipitation of LDL-C from serum by phosphotungstic acid and magnesium chloride, the supernatant was taken and HDL-C estimation done by the method describe for TC **(Rosenberg, B. and Gotttrfried, S.p 1993)**

3.9. Diagnosis of Myocardial Infarction patients:

This study was performed on 60 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. **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

3.10. Study period:

Study period will be one year commencing from February 2008 to May 2009. To complete the study in time a work schedule is prepared depending on different task 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.

3.11. Data analysis:

All the data were checked after collection. Then data was entered into computer, with the help of software SPSS windows programmed version 12.0. The result was shown in bar, pie chart and calculate the percentage the different risk factors of MI patients.

CHAPTER 4

RESULTS

4.1: % Distribution (%) of myocardial infarction among male and female patients.

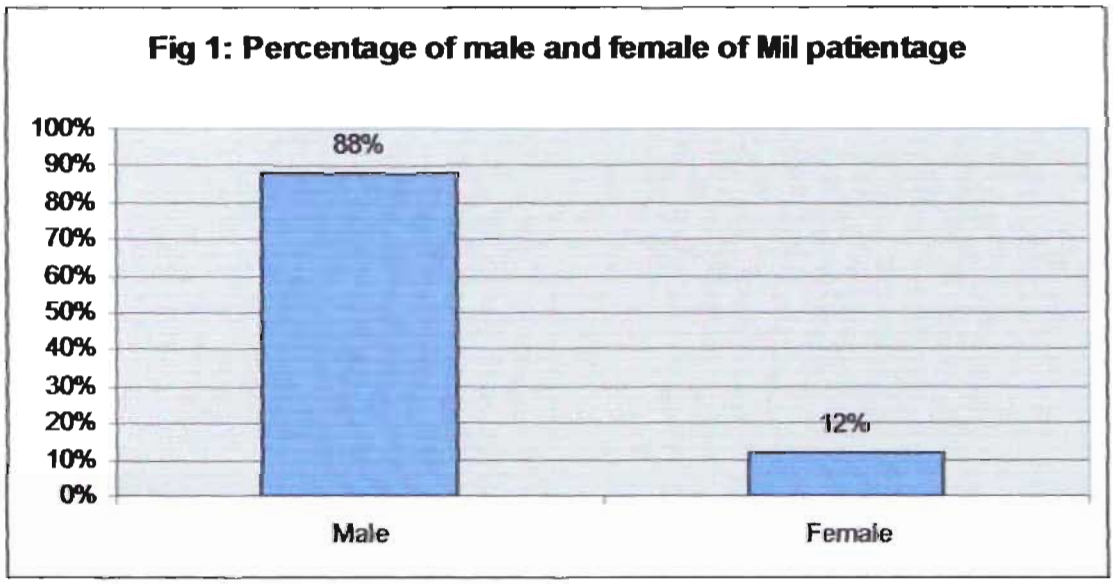


Fig.1. Shows 88% male and 12% female patients have myocardial infarction disease.

4.2. The age distribution (%) of total patients with myocardial infarction.

The distribution of patients between 25- 80. But myocardial infarction is more prevalent in the age range of 56-60.

Age Range	Percentage of patients
25-30	3
31-35	0
36-40	5
41-45	7
46-50	9
51-55	4
56-60	18
61-65	5
66-70	2
71-75	4
76-80	2
81-85	1

Table 2: Shows different age of patients with myocardial infarction.

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

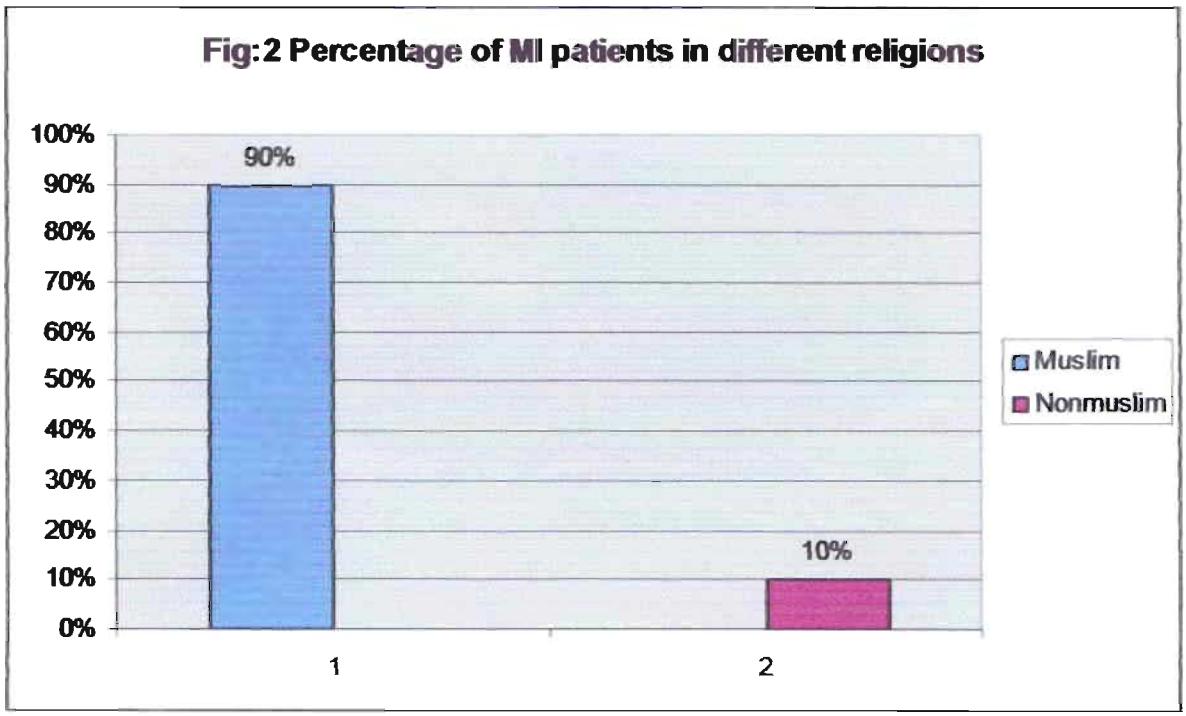


Fig 2. Shows 90% Muslim and 10% Non-Muslim patients with myocardial infarction.

4.4. Distribution (%) of myocardial infarction among different marital status.

The result of the study showed that myocardial infarction is more common in married patients.

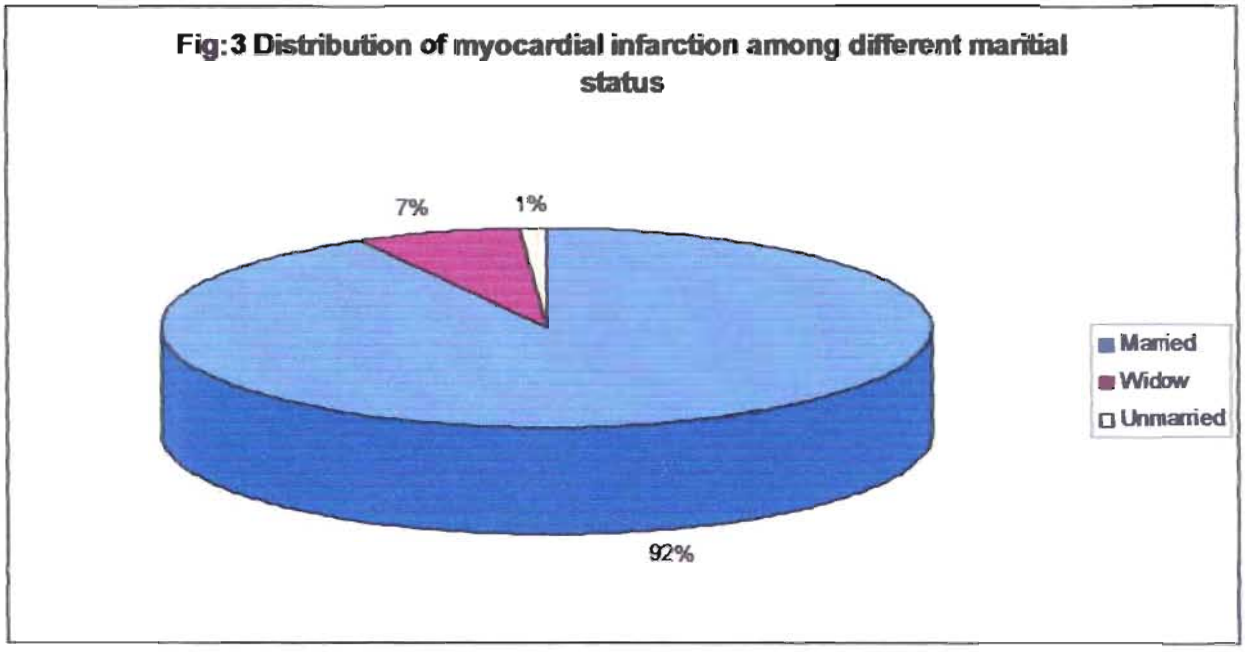


Fig3. Shows 92% married, 7% widow and 1% unmarried patients with myocardial infarction.

4.5. Distribution (%) of myocardial infarction among different types of patients with different education level.

The result of the study showed that myocardial infarction is more common in illiterate patients.

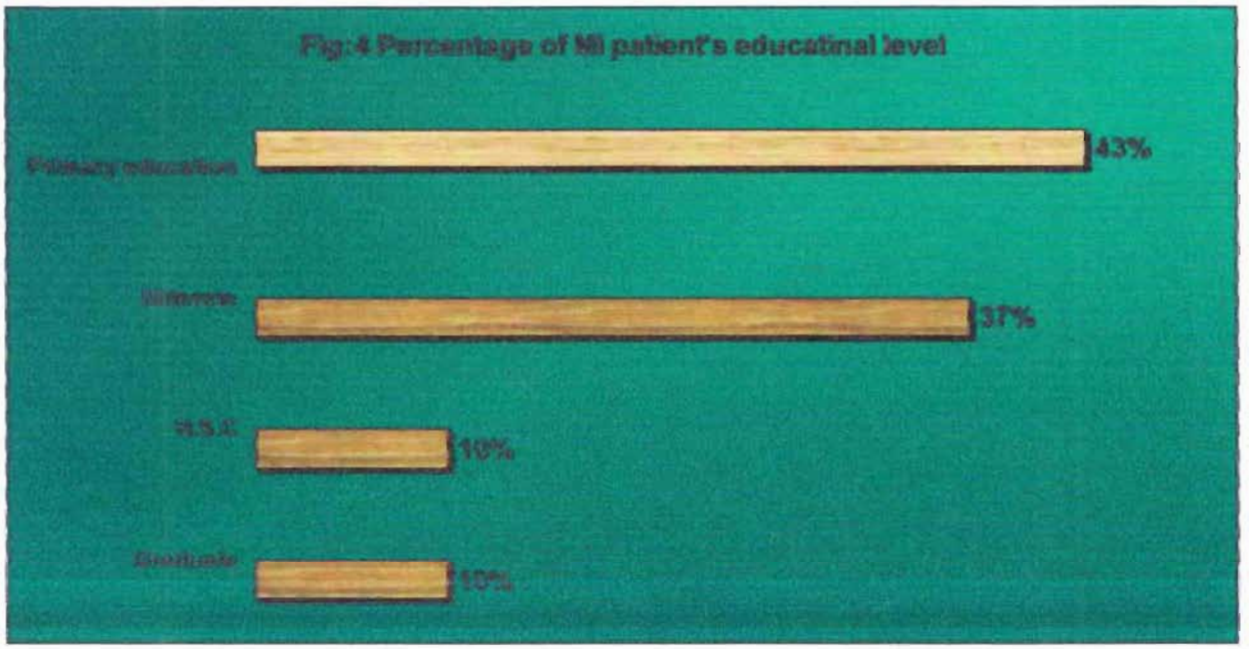


Fig4. Shows 37% illiterate, 43% primary education 10% H.S.C., and 10% graduate patients with myocardial infarction.

4.6. Distribution (%) of myocardial infarction among different occupational MI patients.

The result of the study showed that business and service holder persons are more prone to myocardial infarction.

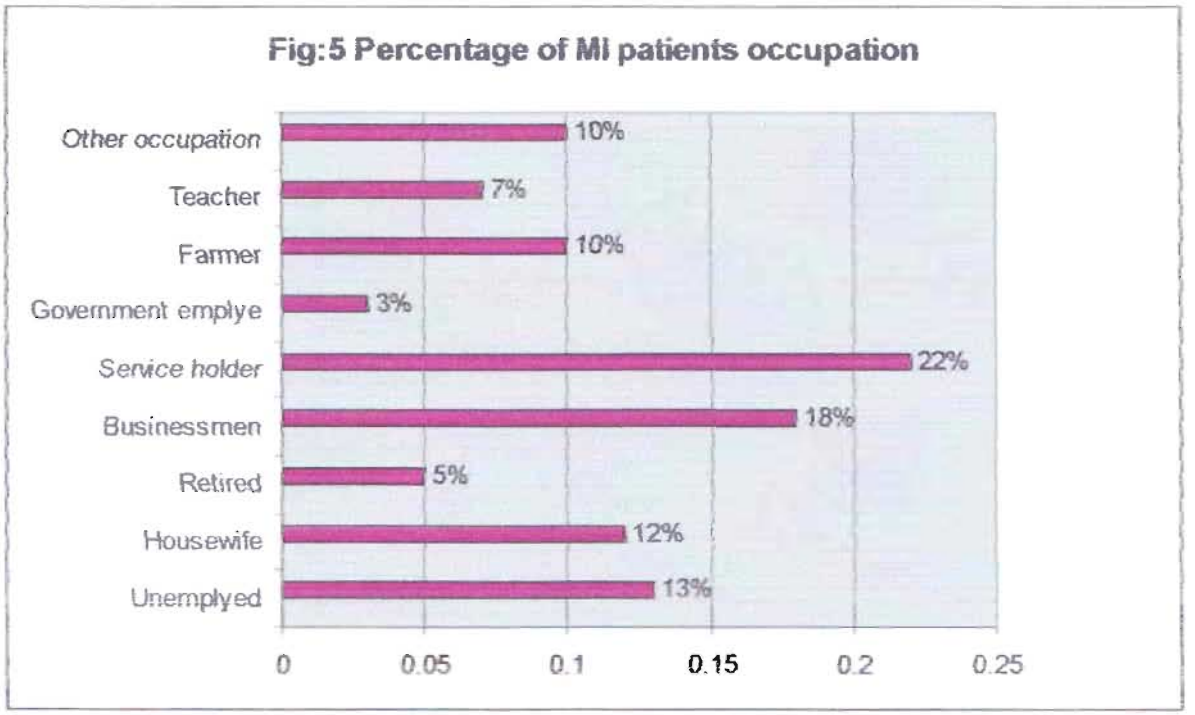


Fig5. Shows 13% unemployed, 12% housewife, 5% retired, 18% business, 22% service holder, 3% government employee, 10% farmer, 7% teacher, and 10% other occupational patients with myocardial infarction.

4.7. Distribution (%) of myocardial infarction among patients with different family history.

Table no: 3

Disease name	No of patients
Diabetes	6
Hypertension	2
Asthma	2
Coronary heart disease	7
Both asthma and hypertension	3
Both diabetes and hypertension	2
Both coronary heart disease and hypertension	2

Table Shows 60% no family history, 29% diabetes, 26% hypertension, 5% asthma, 11% coronary heart disease, 17% both asthma and hypertension, 23% both diabetes and hypertension, and 19% both coronary heart disease and hypertension patients with myocardial infarction.

4.8. Distribution (%) of myocardial infarction in patients with other diseases.

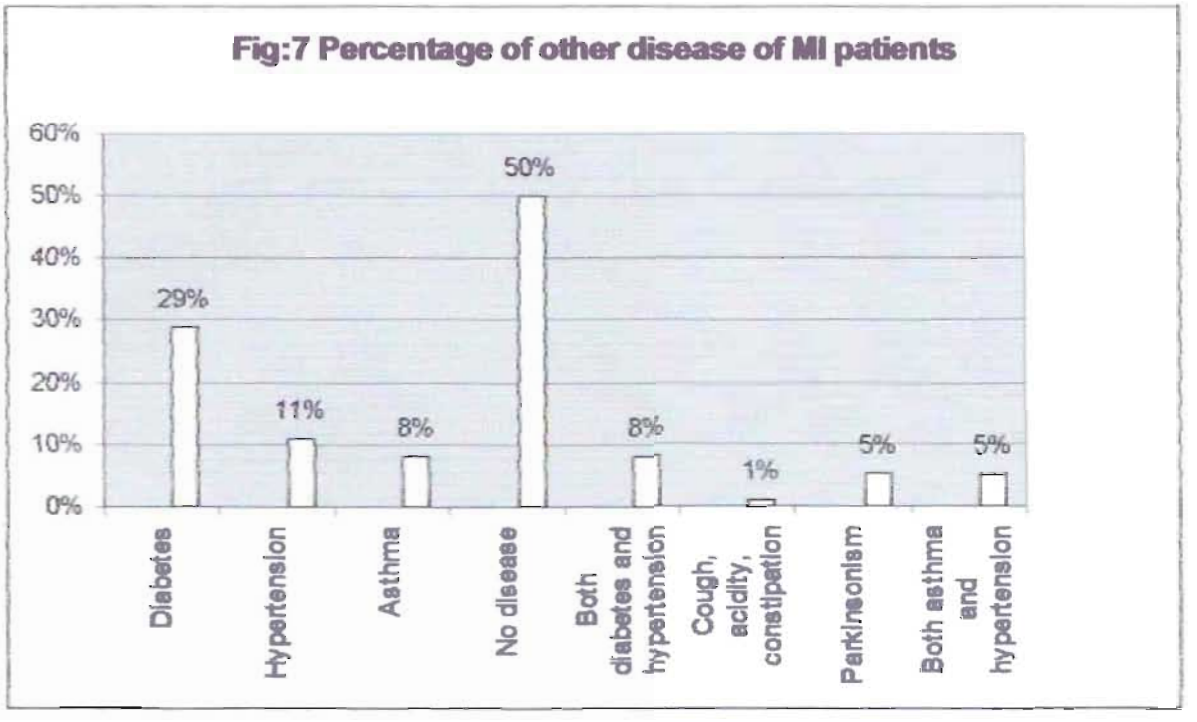


Fig.7. Shows 29% diabetes, 11% hypertension, 8% asthma, 50% no disease, 8% both diabetes and hypertension, 1 % cough, acidity, constipation, 5% parkinsonism and 5% both asthma and hypertension patients with myocardial infarction.

4.9. Distribution of lipid profile among the MI patients.

Table no: 4

Range	HDL mg/dl	No of patients	LDL mg/dl	No of patients	TC mg/dl	No of patients
optimal	<38	25	<100	12	<200	16
borderline	39-40	26	<100-129	28	201-219	25
high	41-42	4	160-189	18	220-239	15
Very high	42≥	2	≥190	12	≥240	4

Table shows 25 patients have optimal range high density lipoprotein (HDL), 26 patients have borderline HDL, 4 have high HDL, and 2 have very high HDL. 12 patients have optimal low density lipoprotein (LDL), 28 have borderline LDL, 18have high LDL, and 12 have very high LDL. And 16 patients have optimal total cholesterol (TC), 28 have borderline TC, 15 have high TC, 4 have very high TC.

4.9.1. Distribution of serum lipid in patients with Myocardial infarction of arrival after 15 days.

Table no: 5

Name of the lipid parameter/ variables	Mean concentration ± SD of serum lipid profile		P values (95% C.I)
	1st day	After 15 days	
Triglycerides (TG) mg/dl	192.3 ± 54	136.85 ± 14	P< 0.01
Total Cholesterol (TG) mg/d	198.78 ± 42.9	186.25 ± 29.3	P<0.01
High density Lipoprotein (HDL) mg/dl	36.13 ± 74	41.76 ± 83	P<0.01
Low density Lipoprotein (LDL) mg/dl	135.17 ± 33.5	126.67 ± 55.8	P<0.02

Values are mean ± standard deviation where P< 0.01 (P= significance 95% C.I), n=60 (n= no of sample size).

Table shows that mean concentration ± SD of triglycerides (TG) at 1st day 192.85 ± 54 and after 15 days treatment with antilipodial drug TG was decreased to (136.85 ± 14) mg/dl, total cholesterol (TC) at 1st day (198.78 ± 42.9) mg/dl after 15 days treatment with antilipodial drug TC was decreased to (186.25 ± 29.3) mg/dl, high density lipoprotein (HDL) at 1st day (36.13 ± 74) mg/dl after 15 days treatment with antilipodial drug which was increased to(41.76 ± 83) mg/dl, and low density lipoprotein (LDL) at 1st day (135.17 ± 33.5) mg/dl after 15 days treatment with antilipodial drug LDL was decreased to (126.67 ± 55.8) mg/dl.

CHAPTER 5

DISCUSSION

Coronary heart disease (CHD) is now global health problem and important cause of mortality worldwide. Coronary heart disease (CHD) is the major cause of death in middle aged and older people in most of the developed and many of the developing countries. Myocardial infarction is by far the most important from of Ischemic heart disease IHD) and alone is the leading cause of death worldwide.

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 may, 2007).**

In our observation age group 55-60 years had the heist incidence of myocardial infarction 30%, Followed by age group 45-50 years (15%). In the study conducted by khondaker et at 1986 found, heist incidence of AMI in age group 41-50 years (32.30%), and in the study Conducted by Abul Faiz et al2, found heist incidence of AMI in age group 51-60 years, (34.4%) **(Abul Faiz et al.1998; 3:1)**

In the study among 60 patients 54 of the patients were Muslim (90%) and (10%) 6 of them were non Muslim who are suffering from MI. MI was more prevalent among the people whose education level was up to the mark, that means those who were illiterate 35%.

In our study mostly service holder 22% and people involved in business 18% were suffering from MI. That means sedentary lifestyle and less involvement in physical activity is responsible for MI.

Among 60 patients 34 (57%) has the habit of betel nut chewing and (68%) had the habit of smoking were nonsmoker. Most of the patients (60%) have no other diseases which may have contributed to MI.

In the study of MA Muqueet shows the change in blood lipid of high fibre biscuits group after 1st 2nd and 3rd visit of trial. Total cholesterol, LDL and TG were gradually declined, but HDL cholesterol and sugar gradually increased. And after one and three month trial, three are gradual decline of total cholesterol, LDL and TG but HDL cholesterol and blood glucose level remained almost the same **(MA Muqueet, KMHS Sirajul Haque; 2003)**

In our study age group 30% patients have high LDL and 20% patients have high LDL. IN case of TC 15% patients have high level of cholesterol 6.6% have very high, 6.6% have high level of HDL which is acceptable and only 3.33% patients have HDL and satisfactory level.

Serum lipids and lipoprotein were estimated in 60 patients with acute MI during acute phase at the day of admission and after 15th day predischarge. Serum total lipids, total cholesterol (TC), and Low density lipoprotein cholesterol (IDLC) showed a significant changes during the hospital stay. High density lipoprotein (HDLC) however started increases from the 1st day statistically significant enhancement on predischarge day, as compare to first day.

Table 5 shows mean concentration ± SD of serum lipid profile of 60 patients. mean concentration ± SD of triglycerides (TG) at 1st day 192.85 ± 54 and after 15 days treatment with antilipodial drug TG was decreased to 136.85 ± 14 mg/dl and its (P=<0.01) , total cholesterol (TC) at 1st day 198.78 ± 42.9 mg/dl after 15 days treatment with antilipodial drug TC decreased to 186.25 ± 29.3 mg/dl and its (P =<0.01), high density lipoprotein (HDL) at 1st day 36.13 ± 74 mg/dl after 15 days treatment with antilipodial drug which was increased to 41.76 ± 83 mg/dl and its(P =<0.01), and low density lipoprotein (LDL) at 1st day 135.17 ± 33.5 mg/dl after 15 days treatment with antilipodial drug LDL decreased to 126.67 ± 55.8 mg/dl and its(P =<0.02). It can be concluded that after 15 day HDL was increased and LDL, TG, TC were decreased with the anti lipid medication such as statin, niacin, Fibrates, resins prescribed by the doctor.

Many scientists have showed in their study a reduction of total cholesterol, HDL cholesterol and LDL cholesterol after acute MI. Other have, however reported no change

in serum total cholesterol and HDL cholesterol. Similar variations have also been noted in serum triglycerides levels **(Biorch, G 1957; Bjorntrop 1960; Tibblin, G 1963; Avogaro, A 1970; Heldenburg D 1980; Ryder, R.e.J 1984).**

Due to irregular administer of medicine and patients unawareness, patients do not get the proper effect of anti lipid drugs. Sometimes it also happens that patients are incapable of buying costly drugs. So this also hampers the achievement of benefits from intervention of medicines. Along with the intervention of drugs and medicines, people can also achieve success being conveying lipid profile in to normal range and increase HDL by changing dietary control, physical exercise and living sedentary world. Reduction of weight is also helpful.

It is difficult to generalize the result of the study, which was done with small sample size. A large sample size could help to get a more generalize information for study.

CONCLUSION

High cholesterol (hypercholesterolemia) can cause the formation and accumulation of plaque deposits in the arteries. And the plaque is the main reason of myocardial infarction.

The main objective of the study was to find out the relationship between MI and high blood cholesterol, and Utilization of drug and its control of lipid profile with in normal range. In this sociodemographic study it was found that age, educational status, occupations were significantly associated with coronary heart disease. Among life style risk factors smoking, type of smoking, betel nut chewing, and tobacco consumption all were highly significant associated with development of MI.

In our study we found that the high blood lipid parameters more prone to having myocardial infarction. Many epidemiological studies found a relation between serum triglycerides concentration incidence of mortality of coronary heart disease.

In our study we can conclude that regular administration of proper medicine such as statin, niacin, Fibrates, resins and changing the diet, have satisfactory result in lowering lipid profile.

Outcome of this study may provide important information for future study.

RECOMMENDATIONS

In the light of study findings the following recommendation has been made:

1. Cardiovascular disease prevention should be integrated with health promotion initiatives.
2. Hypercholesterolemia should be lowered by quick treatment of taking lipid lowering drugs.
3. Also should be maintaining by healthy diet, by healthy weight, and by far away from smoking.
4. High blood cholesterol should be control by physical activity since physical activity is also a contributing factor to control high blood cholesterol.
5. Physical activity should be encouraged for everybody especially to Muslim.
6. Those who have positive family history of non-communicable diseases should be aware of the risk of myocardial infarction.

REFERANCES:

Abul Faiz al., Myocardial Infarction in a district Hospital. Bang Heart. J .1998; 3:1

Allen RA, Kluft C, Bromer EJP: Effect of chronic smoking on fibrinolysis Atherosclerosis: 1985; 443-450.

Alpert JS, Thygesen K, Antman E, et al. Myocardial infarction redefined-a consensus document of the joint European Society of Cardiology/ American College of Cardiology Committee for the redefinition of myocardial infarction. J am Coll Cardiol 2000; 36:959-969.

Avogaro, A., Bitilin Bon, G., Cazzatato, C., Quinci, G.B., Seanson, A., Sparla, H. and zagtti, G.C. Varations in apolipoprotiens B and A during the course of myocardial infarction. Eur. J. Clin. Invest. 2007; 121-129.

Bhatragar D, Anand I S, "An evaluation of technologies for identifying acute cardiac ischemia in the emergency department: executive summary of a National Heart Attack Alert Program Working Group Report". *Ann Emerg Med* 29(1)1995: 1–12. doi:10.1016/S0196-0644(97)70297-X. PMID 8998085.

Biorch, G., Bioquist, G. and sievers, J. (1957) cholesterol values in patients with Myocardial infarction and normal control group. Acta. Med Second,1957; 156, 493-497.

Bjorntrop, P. and Malmcrone, R. (1960) Serum cholesterol in patient with Myocardial infarction in youger ages. Acta. Med. Second. 168, 151-155.

Blankenhorn DH. Two new diet heart studies N. EngJ. Med. 1986; 312:851-52

Boden WE, O'rourke RA, Teo KK, et al, Pace B. Angina. Journal of American Medical Association 284:2007; 916-7.

Boon NA, College NR, Walker BR and Hunter JAA, Myocardial infarction - secondary prevention, Clinical Knowledge Summaries 2006.

British Heart Foundation.European Cardiovascular Disease Statistics, 2008. http://www.thrombosisadviser.com /scripts/pages/en/understanding-thrombosis/how-thrombosis-developes/thrombosis-and-acute-coronary-syndrome/index.php#ref36#ref36.

Brouwer IA, Zock PL, Camm AJ, Bocker D, Hauer RN, Wever EF, Dullemeijer C, Ronden JE, Katan MB, Lubinski A, Buschler H, Schouten EG; SOFA Study Group. "Effect of fish oil on ventricular tachyarrhythmia and death in patients with implantable cardioverter defibrillators: the Study on Omega-3 Fatty Acids and Ventricular Arrhythmia (SOFA) randomized trial". JAMA,2006; **295** (22): 2613–9. doi:10.1001/jama.295.22.2613. PMID 16772624.

Bruce B. Duncan, M I Shmidt, S Offenbacher, S, Kenneth K P Savage, G Hesis, Factor V111 and Other Hemostesis Variables Are Related to Incident Diabetes in Communinities (ARIC) Study. Diabetes Care 1999, 22(5).

C, Josephson M, Steinhaus D, Kluger J, Kang J, Cox B, Zhang H, Schoenfeld D ."Prevention of fatal arrhythmias in high-risk subjects by fish oil n-3 fatty acid intake".2005 (18): 2762–8. doi:10.1161/CIRCULATIONAHA.105.549527PMID16267249.http://circ.ahajournals.org/cgi/content/full/112/18/2762.

Canto JG, Goldberg RJ, Hand MM, *et al.* "Symptom presentation of women with acute coronary syndromes: myth vs reality". *Arch. Intern. Med.* **167** (22) 2007: 2405–13. doi:10.1001/archinte.167.22.2405.PMID18071161.http://archinte.amaassn.org/cgi/pmidlookup?view=long&pmid=18071161

Christopher P. Howson, K. Srinath Reddy, Thomas J. Ryan, and Judith R. Bale, 1998.

D Lee, D Kulick, J Marks. Heart Attack (Myocardial Infarction) by MedicineNet.com . Retrieved November 28, 2006.

Davis TM, Fortun P, Mulder J, Davis WA, Bruce DG . "Silent myocardial infarction and its prognosis in a community-based cohort of Type 2 diabetic patients: the Fremantle

Diabetes Study". Diabetologia, 2004; **47** (3): 395–9. doi:10.1007/s00125-004-1344-4. PMID 14963648.

Division for Heart Disease and Stroke Prevention, National Center for Chronic Disease Prevention and Health Promotion

Dr S W Davies, Depertment of cardiology, Royal Brompton Hospital, Sydney Steet, London Sw3 6NP, UK.

European Society of Cardiology, American College of Cardiology, 2000.

Ganaz P, Braunwald E. Coronary blood flow and myocardial ischaemia. In: Braunwald E. (ed) Heart Disease. A Textbook of cardiovascular Medicine, 5th end. Philadelphia, PA: WB Saunders, 1997; 1161-3

Gershilick AH and Davis SW. Ischaemic Heart Disease: therapeutic issues; Brit. Med. Bull: 2001; 59; 1-123.

Gordon, T., Castelli, W.P., Hjortland, Mckannel, W.B. and Dawber, T.K High density lipoprotein as protective factor against coronary heart disease: The Farmington study, J. Am. Med, 1997.

Guptal R, Gupta VP, Ahluwalia NS, 1994

Guyton AC, Hall JE. Textbook of Medical Physiology. 11th edition, 2006.
Haslet C, Chilver E.R, Hunter J.A.A, Davidsons principles and Practice of medicine. 13th edi, H. brace UK, 1999 245, 247.

Hayee MA, Chowdhury NA, Akhter N, and Ahsan S. Atypical presentation of acute myocardial infarction (AMI). TAS 1993; 3(3): 84-86.

Health Situation in the South-East Asia region, 1998-2000.

Heldenburg D., Rebenstein, A., Levtov, O., Berns, L., Werbin, B. and Tamir, L. Serum lipids and lipoprotein concentrations during the acute phase of myocardial infarction. Atherosclerosis, 1980; 35, 433-437.

Henderson AH, weatherall DJ, Myocardial infarction: diagnosis and investigations1996.

HofgreenC, Karlson BW, HerlitzJ, .1995 International diagnostic criteria for acute myocardial infarction and acute stroke. *Am Heart J* 1984;108:150-8. PMID 6731265

Islam N, Khan M , Latif ZA. Et. Al. Hypertention in Bangladesh- A preliminary survey: Bangladesh med. Res. Counc. Bull. 1983; 9; 11-14

Keating G, Plosker G . "Eplerenone: a review of its use in left ventricular systolic dysfunction and heart failure after acute myocardial infarction". *Drugs* **64** (23) 2004: 2689–707.

Kosug , M; Kimura K, Ishikawa T et al. ''Defferences between men and women in terms of clinical features of ST-segment elevation acute myocardial infarction". Circulation Journal 70 (3) 2006:22-226.dio:10.1253/circj.70.222.Pmid16501283.

Http://www. Jstage.jst.go/jp/articale/circj/70/3/222/pdf. Retrieved on 2008-05-31

Krishnaswarmi S, Prasad N K, "Chronic Chlamydia pneumoniae infection as a risk factor for coronary heart disease in the Helsinki Heart Study". 1989.

Little RA, Frayn KN, Randall PE, *et al.* . "Plasma catecholamines in the acute phase of the response to myocardial infarction". *Arch Emerg Med* **3** (1) 2004: 20–7. PMID 3524599.

Marcus GM, Cohen J, Varosy PD, *et al.* "The utility of gestures in patients with chest discomfort". Am. J. Med.2007; **120** (1): 83–9. doi:10.1016/j.amjmed.2006.05.045. PMID 17208083. http://linkinghub.elsevier.com/retrieve/pii/S0002-9343(06)00668-1

Mc Connel RB, Pyke DA and Robert JAF. Blood group in diabetes Mellitus.Br Med. J: 1967; 272-77.

McSweeney JC, Cody M, O'Sullivan P, Elberson K, Moser DK, Garvin BJ . "Women's early warning symptoms of acute myocardial infarction". *Circulation* ,2003; **108** (21): 2619–23. doi:10.1161/01.CIR.0000097116.29625.7C. PMID 14597589.

Medscape's article "New Definition of 'MI' Poised for World Domination"

Mitra .S.What is communicable disease? (Availale at http://www. Wise geek.com/ what is a communicable disease. Htm, acsssedon 22 august.

Mohan V, Deepa R, Rani ss, PremalthaG. Prevalalence G of Coronary artery disease and its relationship to lipids in the selected Population in South India: the Chennai urban Population Study (Cups NO. 5) J Am Coll cardiol 2001; 38; 683-704.

Mosca L, Banka CL, Benjamin EJ, et al. Evidence-Based Guidelines for Cardiovascular Disease Prevention in Women: 2007 Update. *Circulation.* 2007; Published online before print February 19, 2007.

National Heart Laung, U.S.Department of Health and Human Services. Facts About Angina. National Institute of Health. Ref Type: Pamphlet . 2001;

National Heart, Lung and Blood Institute Heart Attack Warning Signs Retrieved November 22, 2006.

National Heart, Lung and Blood Institute. Heart Attack Warning Signs: 2007Nov

Nyboe J, Jensen G, Appleyard M, Schnohr P, Risk factors for acute myocardial infarction in Copenhagen. I: Hereditary, educational and socioeconomic factors. Copenhagen City Heart Study; Eur Heart J 10 (10): 1989; 910–6.

Pfeffer MA, Braunwald E, Moye LA, Basta L, Brown EJ Jr, Cuddy TE, Davis BR, Geltman EM, Goldman S, Flaker GC, et al. "Effect of captopril on mortality and morbidity in patients with left ventricular dysfunction after myocardial infarction. Results of the survival and ventricular enlargement trial. The SAVE Investigators". *N Engl J Med.* 1999;**327** (10): 669–77. PMID 1386652.

Phillips WR.Watyis D, Havel RJ, Plasma lipo protein and prognosis of coronary artery disease- evaluation by angiography and clinical events: Circulation, 1993 ; 88; 2762-70.

Procor, United States: High HF risk in blacks under 50; 2009

Professor Joseph O.M. Pobee, Reperfusion in acute myocardial infarction. Drug Ther Bull;1974.

Quitnet http://www. Quintet.com/

Ramachandran A, Sathyamurthy I, "Coronary angioplasty with or without stent implantation for acute myocardial infarction. Stent Primary Angioplasty in Myocardial Infarction Study Group". *N Engl J Med* 341 (26);2001.

Rashid K.M., Khbiruddin Md., Hyder S . Text book of Community Medicine and public health, 3rd edi RKH publishers Dhaka. 1999; 264,265,

Ray KK, Cannon CP. "The potential relevance of the multiple lipid-independent (pleiotropic) effects of statins in the management of acute coronary syndromes". *J. Am.* Coll. Cardiol.20005; **46**(8):1425–33.

http://linkinghub.elsevier.com/retrieve/pii/S0735-1097(05)01773-0.

Reperfusion in acute myocardial infarction. Drug Ther Bull. 2005 July
Rhashid AKMH. Influence of life style on coronary heart disease (Dissertation), Dhaka: NIPSOM; 1997: 40-60.

Rimmm EB, bStmpfer MJ .The role of antioxidants in Preventive cardiology. Curropion cardiol.1997; 12:188-194

Robert W. Griffith, MD, the US National Heart, Lung, and Blood Institute. "National Campaign Helps Women Learn the Heart Truth: November 28, 2002;

Rossi S, editor. Australian Medicines Handbook 2006. Adelaide: Australian Medicines Handbook; 2006. ISBN 0-9757919-2-3.

Rubin's Pathology - Clinicopathological Foundations of Medicine. Maryland: Lippincott Williams & Wilkins. 2001. pp. 549. ISBN 0-7817-4733-3.

Ryder, R.e.J., Hayes, T.m., Molligan, I.P., Kingswood, J.C., Williams, S. and Owens , D.R. (1984) how soon after myocardial infarction should plasma lipid values be assessed? Br. Med. J. 289. 1651-1653.

Savotham SG Berry JN, Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries ;1968.

Singh R B, Sharma J P, "Coronary artery bypass grafting within 30 days of an acute myocardial infarction". *Ann. Thorac. Surg.* 59 (5);1997: 1169–76.

Smith A, Aylward P, Campbell T, *et al.* Therapeutic Guidelines: Cardiovascular, 4th edition. North Melbourne: Therapeutic Guidelines; 2003. ISSN 1327-9513

Smith SC Jr, Allen J, Blair SN, et al. AHA/ACC guidelines for secondary prevention for patients with coronary and other atherosclerotic vascular disease: 2006 update: endorsed by the National Heart, Lung, and Blood Institute. *Circulation.* 2006 May 16;113(19):2363-72. Erratum in: Circulation. 2006 Jun 6;113(22):e847.

SosanM, Czajkowshi,, New CAD Risk Factors: Interesting, but how Useful? Patient Care. June 1998;32

Stahmer S; Acute Coronary Syndrome. EMedicine, January 2007

The European Strategy for the Prevention and Control of Noncommunicable Diseases, document from the World Health Organization 2005 Nov .

The women's health resources on the web science 1999.

The World health Organization Monica Project: Geografic Variations in the major risk factors risk of coronary heart disease in men and women aged 35-65 years. W.H.O. Health Stat Q 41:115, 1988.

Thomson S. G. Kienast J. PykeS. D. et. al. Gemastatic factors and the risk of MI of sudden death in patients with angina pectoris. European Concerted Action all thrombosis and disabilities Angina Pectoris Study Group: N. Engl. J. Med. 1995; 332, 635-41.

Tibblin, G. and cramer, K. Serum lipids during the course of acute myocardial infarction and one year after wards Acta. Med.Second,1960; 174, 451-455.

Today's challenges Geneva and diet, Nutrition and the prevention of chronic diseases. In Technical report Series 916. Geneva, World health Organization; 2003.

Wannamethee SG, Shaper AG, Walker M. Physical activiy and mortality in older men with diagnosed coronary heart disease. Circulation 2000; 102: 1358-63

Whelton et al., Guidelines for the Management of Patients with Acute Myocardial Infarction 1995.

WHO Non communicable Diseases prevention and management, 2001; 1, 15.
Wilson PW, D'Agostino RB, Comparison of case fatality in south Asian and white patients after acute myocardial infarction: observational study, 1998; 97.
Wilson PW, D'Agostino RB, Levy D, Belanger AM, Silbershatz H, Kannel WB . 1998

Yusuf S, Peto R, Lewis J, Collins R, Sleight P . "Beta blockade during and after myocardial infarction: an overview of the randomized trials". *Prog Cardiovasc Dis* **27** (5) 1985 : 335–71. doi:10.1016/S0033-0620(85)80003-7. PMID 2858114.