Distribution of Diabetes among Patients with Myocardial Infraction

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2005-2-70-036

Date of submission:

August 2010

Department of Pharmacy

EAST WEST UNIVERSITY

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A Thesis paper is submitted in partial fulfillment of the requirements for the Degree of Bachelor of pharmacy on August, 2010.

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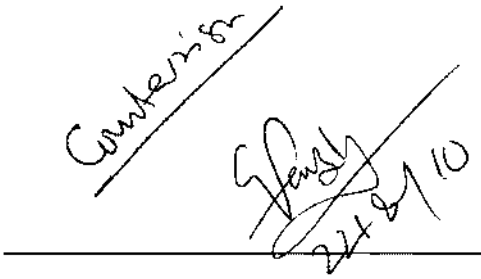
This thesis is dedicated to my parents

This thesis paper was submitted to the Department of Pharmacy, East West University on (....8..8..2010..)



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CERTIFICATE

This is to certify that, the thesis 'Diabetes associated with Hypertension and its effect on Myocardial Infarction' submitted to the Department of pharmacy, East West University, Mohakhali, Dhaka for the partial fulfill of the requirements for the degree of Bachelor of pharmacy (B.Pharm) was carried out by Arnob Sharif (ID: 2005-2-70-036) under our guidance and supervision and that no part of the thesis has been submitted for any other degree. We further certify that all the sources of information and other facilities available of this connection are duly acknowledged.

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

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. I am grateful to the doctors and nurses of NICVD who have helped me a lot during data collection from patients. I am grateful to all the patients from whom I collected necessary information.

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

Arnob Sharif

June, 2010.

CHAPTER 1

INTRODUCTION

1.1Communicable Disease

Communicable diseases can be passed from one person to another. Such diseases may be passed through common contact like colds or the flu. Other communicable diseases like AIDS and hepatitis may only be passed through sexual contact or sharing of infected hypodermic needles.

Non-communicable diseases are not usually passed from one person to another. Such diseases however may be genetic or environmentally caused, or in some cases caused by lifestyle choices like smoking.

Over 90 per cent of the world's disease burden occurs in developing countries and most is due to communicable diseases. While chronic diseases, such as heart disease and diabetes, are on the rise, communicable disease remains the major challenge. HIV and AIDS, malaria, and tuberculosis are important communicable diseases targeted by global control programs. However, the emphasis on these three epidemics now threatens to undermine efforts of proven efficacy for the control of other diseases that affect developing countries, by diverting attention and resources away from them. **(Michael Duty, 2009)**

A review of communicable diseases in developing countries is a study in inequity. Children bear the greatest burden. Many of the child survival gains achieved in the 1980s have either stagnated or reversed. Most of those affected come from impoverished settings where they are most likely to be malnourished and least likely to know about life-saving interventions, where to find them, or have the means to obtain them. Traditional values and behaviors untempered by adequate information or meaningful dialogue with the public health community conspire to compound these inequalities. Two diseases - pneumonia and diarrhea - cause most of the mortality.
(G. Pirozzi, 2008)

1.1.2 Common Communicable Diseases

The more common types of communicable diseases like colds or the common flu or any of the influenza variations can be passed from one person to another by common contact. This usual happens when an infected person coughs or sneezes, expelling the microbes into the air around them. These microbes can travel for several feet and land on people or objects. A person who touches these microbes may become infected if he inhales or ingests them, or if they penetrate an opening in the skin. The No. 1 way to protect from communicable disease is to wash hands thoroughly and regularly. **(Michael Duty, 2009)**

1.2.1 Non Communicable disease

Non-communicable diseases are not usually passed from one person to another. Such diseases however may be genetic or environmentally caused, or in some cases caused by lifestyle choices like smoking. **(Michael Duty, 2009)**

1.2.2 Causes of non communicable disease

The burden of non-communicable disease results from past and cumulative risks; the future burden will be determined by current population exposures to risk factors. Although the major risk factors for non communicable disease epidemics are more complex than those for infectious disease, they are well known and account for almost all such events; many are common to the main categories of non-communicable diseases and most are modifiable and operate in the same manner in all regions of the world, with some quantitative differences.

The ageing of populations, mainly due to falling fertility rates and increasing child survival, are an underlying determinant of non-communicable disease epidemics. Additionally, global trade and marketing developments are driving the nutrition transition towards diets with a high proportion of saturated fat and sugars. This diet, in combination with tobacco use and little physical activity, leads to population-wide atherosclerosis and the widespread distribution of non-communicable disease.

Table-1 shows the contribution of the major non communicable disease risk factors to the burden of disease. In developed countries, seven of the ten leading risk factors contributing to the burden of disease are for non communicable disease, compared with six and three of ten in developing countries with low and high rates of mortality, respectively. In most developing countries, non communicable disease risk factor levels have increased during the past decade, portending an increase in the rate of non-communicable diseases in the next two decades. **(R Beaglehole DSc, 2003.)**

1.2.3 Global burden of non communicable disease

This year there will be an estimated 56 million deaths globally, of which 60% will be due to non-communicable diseases:6 16 million deaths will result from cardiovascular disease (CVD), especially coronary heart disease (CHD) and stroke; 7 million from cancer; 3·5 million from chronic respiratory disease; and almost 1 million from diabetes. Mental health problems are leading contributors to the burden of disease in many countries and contribute substantially to the incidence and severity of many non communicable diseases including CVD and cancer.

		Developed countries			Developing countries		
Cause	% of total deaths	Rank	Cause	% of total deaths	Rank	Cause	% of total deaths
Ischaemic heart disease	12·4%	1	Ischaemic heart disease	22·6%	1	Ischaemic heart disease	9·1%
Cerebrovascular disease	9·2%	2	Cerebrovascular disease	13·7%	2	Cerebrovascular disease	8·0%
Lower respiratory infections	6·9%	3	Trachea, bronchus, lung cancers	4·5%	3	Lower respiratory infections	7·7%
HIV/AIDS	5·3%	4	Lower respiratory infections	3·7%	4	HIV/AIDS	6·9%
COPD	4·5%	5	COPD	3·1%	5	Perinatal conditions	5·6%
Perinatal conditions	4·4%	6	Colon and rectum cancers	2·6%	6	COPD	5·0%
Diarrhoeal diseases	3·8%	7	Stomach cancer	1·9%	7	Diarrhoeal diseases	4·9%
Tuberculosis	3·0%	8	Self-inflicted injuries	1·9%	8	Tuberculosis	3·7%
Road traffic accidents	2·3%	9	Diabetes	1·7%	9	Malaria	2·6%
Trachea, bronchus, lung cancers	2·2%	10	Breast cancer	1·6%	10	Road traffic accidents	2·5%

=chronic obstructive pulmonary disease. Developed countries include European countries, former Soviet countries, Canada, USA, Japan, Australia, and New Zealand.

Figure-1: Estimates of the ten leading causes of death in 20006

Figur-1 shows that non-communicable diseases are leading causes of death in developing and developed countries. Only in Africa do communicable diseases cause more deaths than non-communicable diseases; this year 2·8 million CVD deaths will occur in China and 2·6 million in India. Non-communicable diseases contribute substantially to adult mortality with the highest rates being in central and eastern European countries (figure).7 They add to health inequalities within and between countries, mainly affecting poor populations largely because of inequalities in the distribution of major risk factors.8—10 The global pattern of death will increasingly be dominated by non-communicable diseases; by 2020, CHD and stroke are expected to be the leading causes of death and loss of disability-adjusted life years. **(R Beaglehole DSc, 2003)**

1.2.4 Chronic non-communicable diseases

Chronic non-communicable diseases (CNCDs) are reaching epidemic proportions worldwide. These diseases — which include cardiovascular conditions (mainly heart disease and stroke), some cancers, chronic respiratory conditions and type 2 diabetes — affect people of all ages, nationalities and classes.

The conditions cause the greatest global share of death and disability, accounting for around 60% of all deaths worldwide. Some 80% of chronic-disease deaths occur in low- and middle-income countries. They account for 44% of premature deaths worldwide. The number of deaths from these diseases is double the number of deaths that result from a combination of infectious diseases (including HIV/AIDS, tuberculosis and malaria), maternal and prenatal conditions, and nutritional deficiencies. **(Abdallah S. Daar, 2007).**

1.3.1 Cardiovascular disease

Cardiovascular disease, also known as heart and circulatory disease, covers all diseases that affect the heart and circulation. This includes conditions such as coronary heart disease (angina and heart attack) and stroke. **(British heart foundation, 2010)**

1.3.2 The risk Factors of cardiovascular Disease

- Smoking
- High blood pressure
- High blood cholesterol
- Physical inactivity
- Being overweight or obese
- Diabetes
- A family history of heart disease
- Age – as patient get older, his/her risk increases

Ethnic group – some ethnic groups have a higher risk of heart disease. For example, South Asian people living in the UK have a higher risk than the rest of the UK population. **(British heart foundation, 2010)**

1.3.3 Types of Cardiovascular Disease

1.3.3.1 Rheumatic heart disease

Rheumatic heart disease is caused by one or more attacks of rheumatic fever, which then do damage to the heart, particularly the heart valves. Rheumatic fever usually occurs in childhood, and may follow a streptococcal infection. In some cases, the infection affects the heart and may result in scarring the valves, weakening the heart muscle, or damaging the sac enclosing the heart. The valves are sometimes scarred so they do not open and close normally. **(American heart association, 2009)**

1.3.3.2 Hypertensive heart disease

High blood pressure of unknown origin (primary hypertension) or caused by (secondary hypertension) certain specific diseases or infections, such as tumor in the adrenal glands, damage to or disease of the kidneys or their blood vessels. High blood pressure may overburden the heart and blood vessels and cause disease. Uncontrolled and prolonged elevation of blood pressure (BP) can lead to a variety of changes in the myocardial structure, coronary vasculature, and conduction system of the heart. These changes in turn can lead to the development of left ventricular hypertrophy (LVH), coronary artery disease, various conduction system diseases, and systolic and diastolic dysfunction of the myocardium, which manifest clinically as angina or myocardial infarction, cardiac arrhythmias (especially atrial fibrillation), and congestive heart failure (CHF). Thus, hypertensive heart disease is a term applied generally to heart diseases, such as LVH, coronary artery disease, cardiac arrhythmias, and CHF that are caused by direct or indirect effects of elevated BP. Although these diseases generally develop in response to chronically elevated BP, marked and acute elevation of BP can also lead to accentuation of an underlying predisposition to any of the symptoms traditionally associated with chronic hypertension. **(Kamran Riaz, 2010)**

1.3.3.2.1 Aneurysm

An aneurysm is a bulge or weakness in the wall of a blood vessel. Aneurysms can enlarge over time and may be life threatening if they rupture. They can occur because of high blood pressure or a weak spot in a blood vessel wall. Aneurysms can occur in arteries in any location in your body. The most common sites include the abdominal aorta and the arteries at the base of the brain.

1.3.3.2.2 Atherosclerosis

In atherosclerosis the walls of arteries become thick and stiff because of the build up fatty deposits. The fatty deposits are called plaques. When this happens, the flow of blood is restricted. Atherosclerosis can happen throughout the body. In the arteries of the heart it is known as coronary artery disease, in the legs, peripheral arterial disease. Atherosclerosis happens over a period of time and its consequences can be grave and include heart attack and stroke.

1.3.3.2.3 High blood pressure (hypertension)

High blood pressure is the excessive force of blood pumping through blood vessels. High blood pressure causes many types of cardiovascular disease, such as stroke and heart failure, and renal disease.

1.3.3.2.4 Peripheral arterial disease

Peripheral arterial disease (PAD) is caused by atherosclerosis, which is the narrowing and / or blockage of the blood vessels in the legs. PAD manifests as pain in the legs when walking, which is relieved by rest. If patients have PAD they are at greater risk of developing gangrene in legs. **(American heart association, 2009)**

1.3.3.3 Ischemic heart disease

Heart ailments caused by narrowing of the coronary arteries and therefore a decreased blood supply to the heart. Angina pectoris, commonly known as angina, is severe chest pain due to ischemia (a lack of blood and hence oxygen supply) of the heart muscle,

generally due to obstruction or spasm of the coronary arteries. Coronary artery disease, the main cause of angina, is due to atherosclerosis of the cardiac arteries.
Ischaemic heart disease may be present with any of the following problems:

1.3.3.3.1 Angina

Angina manifests as pain in the chest that result from reduced blood supply to the heart (ischemia). Blood carries oxygen around the body and depriving the heart of oxygen has serious consequences.

Angina is caused by atherosclerosis, that is the narrowing and / or blockage of the blood vessels that supply the heart. The typical pain of angina is in the chest but it can often radiate to the left arm, shoulder or jaw, the pain is related to exertion and is relieved by rest. An angina attack is also associated with shortness of breath and sweating. Woman may experience angina slightly differently. Women appear to have more pain in their shoulder and middle back area, and more throat, neck, and jaw pain than men. If angina symptoms rapidly worsen and occur at rest this may presage an impending heart attack (myocardial infarction) then patient should seek medical help immediately. Angina pectoris is the medical term for chest pain or discomfort due to coronary heart disease. Angina is a symptom of a condition called myocardial ischemia. It occurs
when the heart muscle (myocardium) doesn't get as much blood (hence as much oxygen) as it needs. This usually happens because one or more of the heart's arteries (blood vessels that supply blood to the heart muscle) is narrowed or blocked. Insufficient blood supply is called ischemia. Angina also can occur in people with valvular heart disease, hypertrophic cardiomyopathy (this is an enlarged heart due to disease) or uncontrolled high blood pressure. These cases are rare, though.
Typical angina is uncomfortable pressure, fullness, squeezing or pain in the center of the chest. The discomfort also may be felt in the neck, jaw, shoulder, back or arm. Many types of chest discomfort aren't related to angina. Acid reflux (heartburn) and lung infection or inflammation are examples.

1.3.3.3.1.2 Causes of Angina Pectoris

Angina often occurs when the heart needs more blood. For example, running to catch a bus could trigger an attack of angina while walking might not. Angina may happen during exercise, strong emotions or extreme temperatures. Some people, such as those with a coronary artery spasm, may have angina when they're resting. Angina is a sign that someone is at increased risk of heart attack, cardiac arrest and sudden cardiac death. **(American Heart Association, 2009)**

1.3.3.3.2 Atherosclerosis

In atherosclerosis the walls of arteries become thick and stiff because of the build up fatty deposits. The fatty deposits are called plaques. When this happens, the flow of blood is restricted. Atherosclerosis can happen throughout the body. In the arteries of the heart it is known as coronary artery disease, in the legs, peripheral arterial disease (PAD). Atherosclerosis happens over a period of time and its consequences can be grave and include heart attack and stroke.

1.3.3.3.3 Coronary artery disease

Coronary artery disease is also known as ischemic heart disease. It is caused by atherosclerosis, that is the narrowing and / or blockage of the blood vessels that supply the heart. It is one of the most common forms of heart disease and the leading cause of heart attacks and angina.

1.3.3.3.4 Coronary heart disease

Coronary heart disease refers to the disease of the arteries to the heart and their resulting complications, such as angina, heart attacks and heart failure.

1.3.3.3.5 Heart attack

A heart attack (myocardial infarction) occurs when the heart's supply of blood is stopped. A heart attack need not be fatal, especially if patient receive medical attention and treatment to deal with the blockage soon after patient has heart attack. But patient is

likely to be left with a damaged heart post heart attack. A heart attack manifests as severe central chest pain, which may also radiate to the left arm, shoulder or jaw. Severe shortness of breath, sweating and feeling faint are common additional symptoms. For a woman, however, the experience of heart attack may differ. Rather than acute chest pain she may have difficulty breathing, be very, very tired and have pain in her shoulder, jaw, or upper back pain.

1.3.3.3.6 Sudden death

Sudden death occurs when there is an abrupt loss of the heart's ability to pump blood. This may be because of heart attack or serious abnormality of the heart's rhythm.

1.3.3.4 Cerebrovascular disease

Disease pertaining to the blood vessels in the brain. A cerebrovascular accident or stroke is the result of an impeded blood supply to some part of the brain. Cerebral vascular disease is caused by atherosclerosis, that is the narrowing and / or blockage of the blood vessels that flow to the brain. If the flow of blood is cut off this can lead to strokes and transient ischemic attacks.

1.3.3.4.1 Atherosclerosis

In atherosclerosis the walls of arteries become thick and stiff because of the build up fatty deposits. The fatty deposits are called plaques. When this happens, the flow of blood is restricted. Atherosclerosis can happen throughout the body. In the arteries of the heart it is known as coronary artery disease, in the legs, peripheral arterial disease. Atherosclerosis happens over a period of time and its consequences can be grave and include heart attack and stroke.

1.3.3.4.2 Stroke

A stroke occurs when the blood supply to the brain is interrupted. This can happen either when a blood vessel in the brain or neck is blocked or bursts. If this happens, brain is deprived of oxygen and parts of your brain may be permanently damaged.

The consequences of a stroke can include problems with speech or vision, weakness or paralysis.

1.3.3.4.3 Transient ischemic attacks

Just as stroke occurs when the flow of blood is blocked, TIAs happen when there is a brief blockage. The temporary loss of blood to the brain causes a brief, sudden change in brain function. This may manifest as temporary numbness or weakness on one side of the body, loss of balance, confusion, and blindness in one or both eyes, double vision, difficulty speaking, or a severe headache. But these will disappear quickly and permanent damage is unlikely. If patients have ever experienced symptoms like this you should seek medical advice. A TIA can be a warning that you are at risk of stroke sometime in the future.

1.3.3.5 Inflammatory heart disease

Inflammation of the heart muscle (myocarditis), the membrane sac (pericarditis) which surrounds the heart, the inner lining of the heart (endocarditis) or the myocardium (heart muscle). Inflammation may be caused by known toxic or infectious agents or by an unknown origin.

1.3.3.5.1 Cardiomyopathy

Cardiomyopathy refers to diseases of the heart muscle. Some types of cardiomyopathy are genetic, while others occur because of infection or other reasons that are less well understood. One of the most common types of cardiomyopathy is idiopathic dilated cardiomyopathy, where the heart is enlarged. Other types include ischemic, loss of heart muscle; dilated, heart enlarged; hypertrophic, heart muscle is thickened.

1.3.3.5.2 Pericardial disease

The sac that encases the heart is called the pericardium and it can be affected by a variety of conditions such as inflammation (pericarditis), fluid accumulation (pericardial effusion) and stiffness (constrictive pericarditis). The etiology of these conditions varies.

1.3.3.5.3 Valvular heart disease

The heart's valves keep blood flowing through the heart in the right direction. But a variety of conditions can lead to valvular damage. Valves may narrow (stenosis), leak (regurgitation or insufficiency) or not close properly (prolapse). Patient may be born with valvular disease, or the valves may be damaged by such conditions as rheumatic fever, infections connective tissue disorders, and certain medications or radiation treatments for cancer.

1.3.3.6 Congenital heart disease

Congenital heart disease is when people are born with malformations of the heart's structures. This may be the result of the genes that patients inherited from their parents or adverse exposure to certain elements while still in the womb, such as some medicines or too much alcohol. Congenital heart disease is a broad term and examples are holes in the heart, abnormal valves, and abnormal heart chambers.

1.3.3.7 Heart failure

Heart failure is a chronic condition that happens when the heart's muscle becomes too damaged to adequately pump the blood around body. If patients have heart failure, then their heart still works but because it is less effective patient's organs do not get enough blood and oxygen. Heart failure tends to affect older people more often and manifests as

shortness of breath, reduced exercise tolerance and swelling of the ankles. It results if the heart is damaged and weakened. **(World heart foundation, 2007)**

1.3.3.8 Coronary Heart disease

Coronary heart disease is caused by a gradual build up of fatty deposits in the walls of your coronary arteries, which can then cause them to narrow. The medical term for this condition is atherosclerosis and the fatty material is known as atheroma.

Over time, the artery may become so narrow that it can't deliver enough oxygen to your heart, especially when you're exerting yourself. This can lead to angina – a pain or discomfort in your chest.

If a piece of this fatty material breaks away from the artery wall it can cause a clot to form, which will then starve your heart of blood and oxygen. This is known as a heart attack. **(British heart foundation, 2010)**

1.3.3.8.1 Prevention of coronary heart disease

The report of the World Health Organization Expert Committee on Prevention of Coronary Heart Disease considered that a comprehensive action for coronary heart disease (CHD) prevention has to include three components:

1. A population strategy – for altering, in the entire population, those life-style and environmental factors, and their social and economic determinants, that are the underlying causes of the mass occurrence of coronary heart disease
2. A high risk strategy – identification of high risk individuals, and action to reduce their risk factor levels
3. Prevention of recurrent coronary heart disease events and progression of the disease in patients with clinically established coronary heart disease .

Prevention targeted at patients with established coronary disease and the high risk strategy targeted at healthy individuals at high risk are an integral part of clinical practice. The clinical approaches and the population approaches for coronary heart disease prevention are complimentary, but the population strategy is fundamental to reducing the burden of cardiovascular disease. **(David Wood. 2001)**

1.3.4 Myocardial Infraction

Myocardial infarction (MI) means that part of the heart muscle suddenly loses its blood supply. Without prompt treatment, this can lead to damage to the affected part of the heart. An MI is sometimes called a heart attack or a coronary thrombosis. An MI is part of a range or disorders called acute coronary syndromes.

There are different types of MI which are based on what is seen on ECG (heart tracing). The two main types are called ST elevation MI (STEMI) and non-ST elevation MI (NSTEMI). Patient's treatment will depend upon the type of MI he/she have.

MI, 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.

In a STEMI, the artery supplying an area of the heart muscle is completely blocked. However, in a NSTEMI, the artery is only partly blocked, so only part of the heart muscle being supplied by the affected artery is affected. **(Tim Kenny, 2010)**

1.3.4.1 Causes of Myocardial Infraction

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. Each plaque has an outer firm shell with a soft inner fatty core.

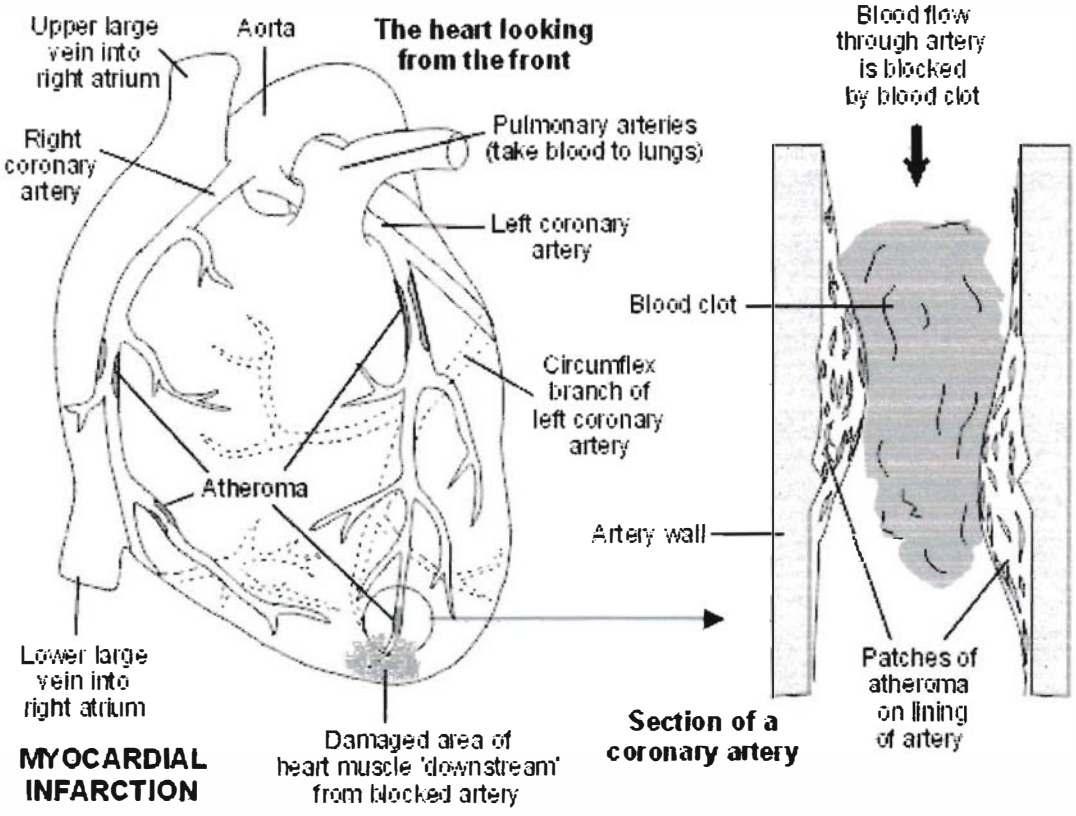

Figure 2. Pathogenesis of Myocardial Infraction

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 below 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.

1.3.4.2 Uncommon causes

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 travelling to a coronary artery where it gets stuck; taking cocaine which can cause a coronary artery to go into spasm; complications from heart surgery and some other rare heart problems. These are not dealt with further in this leaflet.

The rest of this leaflet deals only with the common cause - thrombosis over an atheroma plaque. **(Tim Kenny, 2010)**

1.3.4.3 Site of infarction

The ECG has been used to localize the site of ischemia and infarction. Some leads depict certain areas; the location of the infarct can be detected fairly accurately from analysis of the 12-lead ECG. Leads that best detect changes in commonly described locations are classified as follows:

- Inferior (or diaphragmatic) wall: II, II and aVF
- Septal: V1 and V2

- Anteroseptal: V1, V2, Vf3 and sometimes V4
- Anterior: V3, V4 and sometimes V2
- Apical: V3, V4 or both
- Lateral: I, aVL, V5 and V6

(American Heart Association, 2008).

1.3.4.4 Symptoms of Myocardial Infraction

The most common symptom of an MI is severe chest pain, which often feels like a heavy pressure feeling on your chest. The pain may also travel up into your jaw and down your left arm or down both arms. You may also sweat, feel sick and feel faint. You may also feel short of breath. The pain may be similar to angina, but it is usually more severe and lasts longer. (Angina usually goes off after a few minutes. MI pain usually lasts more than 15 minutes - sometimes several hours.) However, some people have only a mild discomfort in their chest when they have an MI. The pain can sometimes feel like indigestion or heartburn. A small MI occasionally happens without causing any pain (a 'silent MI'). This is usually diagnosed when you have an ECG (heart tracing) at a later stage. Some people collapse and die suddenly if they have a large or severe MI. This is not very common.

1.3.4.5 Diagnosis & Assessment of MI

Many people develop chest pains that are not due to an MI.

MI can be conformed by the following Tests; These are:

- A heart tracing called an ECG (electrocardiograph). There are typical changes to the normal pattern of the heart tracing if you have an MI. Patterns that occur with an MI include things called pathological Q waves and ST elevation. However, it is possible to have a normal ECG even if you have had an MI.

- Blood tests. A blood test that measures a chemical called troponin is the usual test that confirms an MI. This chemical is present in heart muscle cells. Damage to heart muscle cells releases troponin into the bloodstream. With an MI, the blood level of troponin increases within 3-12 hours from the onset of chest pain, peaks at 24-48 hours, and returns to a normal level over 5-14 days.

A rough idea as to the severity of the MI (the amount of heart muscle that is damaged) can be gauged by the degree of abnormality of the ECG and the level of troponin in the blood. Another chemical that may be measured in a blood test is called creatine kinase. This too is released from heart muscle cells during an MI.**(Dr Tim Kenny, 2010).**

1.3.4.6 Treatment of Myocardial infraction

The following is a typical situation and mentions the common treatments offered. Each case is different and treatments may vary depending on patient's situation. Treatments for STEMI and NSTEMI can also differ.

1.3.4.6.1 Aspirin and other anti platelet drugs

As soon as possible after an MI is suspected you will be given a dose of aspirin. Aspirin reduces the 'stickiness' of platelets. Platelets are tiny particles in the blood that trigger the blood to clot. It is the platelets that become stuck onto a patch of atheroma inside an artery that go on to form the clot (thrombosis) of an MI.

Another anti platelet drug called clopidogrel is also given. This works in a different way to aspirin and adds to the action of reducing platelet stickiness.

1.3.4.6.2 Injections of heparin or a similar drug

These are usually given for a few days to help prevent further blood clots forming.

1.3.4.6.3 Pain relief

A strong pain killer given by injection into a vein is given to ease the pain.

1.3.4.6.4 Treatment to restore blood flow in the blocked coronary artery

The part of the heart muscle starved of blood does not die (infarct) immediately. If blood flow is restored within a few hours, much of the heart muscle that would have been damaged will survive. This is why an MI is a medical emergency, and treatment is given urgently. The quicker the blood flow is restored, the better the outlook.

There are two treatments that can be done to restore blood flow back through the blocked artery:

- Emergency angioplasty is, ideally, the best treatment if it is available and can be done within a few hours of symptoms starting. In this procedure a tiny wire with a balloon at the end is put into a large artery in the groin or arm. It is then passed up to the heart and into the blocked section of a coronary artery using special X-ray guidance. The balloon is blown up inside the blocked part of the artery to open it wide again. A stent may be left in the widened section of the artery. A stent is like a wire mesh tube which gives support to the artery and helps to keep the artery widened. See separate leaflet called 'Angioplasty' for details.
- An injection of a 'clot busting' drug is an alternative to emergency angioplasty. In reality, this is the more common treatment as it can be given easily and quickly in most situations. Some ambulance crews are trained to give this treatment. Note: a common 'clot buster' drug used in the UK is called streptokinase. If you are given this drug you should not be given it again if you have another MI in the future. This is because antibodies develop to it and it will not work so well a second time. An alternative 'clot buster' drug should be given if you have another MI in the future.

Both the above treatments usually work well to restore blood flow and greatly improve the outlook. The most crucial factor is the quickness in which one or other treatment is given after symptoms have developed.

1.3.4.6.5 A beta-blocker drug

Beta-blocker drugs block the action of certain hormones such as adrenaline. These hormones increase the rate and force of the heartbeat. Beta-blocker drugs have some protective effect on the heart muscle and they also help to prevent abnormal heart rhythms from developing.

Beta-blocker drugs will also help to prevent having either another myocardial infarction.

1.3.4.6.6 Insulin

Some people have a raised blood sugar level when they have a myocardial infarction, even if they do not have diabetes. If this occurs, then patient's blood sugar levels may need to be controlled with insulin. If patient have diabetes then it is also likely that patient will need to be treated with insulin to control your blood glucose levels when you are in hospital.

1.3.4.6.7 Oxygen

Patient may be given oxygen which works to reduce the risk of damage to patient's heart muscle.

1.3.4.7 Patterns & complication of Myocardial Infraction

This often depends on the amount of heart muscle that is damaged. In many cases only a small part of the heart muscle is damaged (infarcts or dies) which heals as a small patch of scar tissue. The heart can usually function normally with a small patch of scar tissue. A larger MI is more likely to be life-threatening or cause complications.

Even before treatments became available to restore blood flow such as 'clot busting' drugs and angioplasty, many people make a full recovery as many MIs are small. With the help of modern treatment, particularly if patients are given treatment within a few hours to

restore blood flow, a higher percentage of people now make a full recovery.

Some possible complications that may occur after an MI include the following:

- **Heart failure.** If a large area of the heart muscle is damaged, then the pumping ability of the heart may be reduced. Less blood than usual is then pumped around the body, especially when extra blood is needed when patients exercise. Symptoms such as breathlessness, tiredness, and swollen ankles may develop. Mild heart failure can often be treated with medication. Severe heart failure can be serious and even life-threatening.
- **Abnormal heart rhythms** may occur if the electrical activity of the heart is affected. The main risk of this happening is within the first few hours after an MI. Sudden, chaotic, fast heart beats may occur. This is called ventricular fibrillation and is the common cause of cardiac arrest. This needs immediate treatment with an electrical shock given by a defibrillator. Otherwise, collapse and sudden death is likely. Other less serious abnormal heart rhythms can also occur following an MI which can often be treated with drugs.
- **A further MI** may occur sometime in the future. This is more likely if the coronary arteries are badly affected with atheroma, or further build up of atheroma continues. If the risk of this is thought to be high then surgery may be advised to bypass or widen severely narrowed coronary arteries.

The most crucial time is during the first day or so. If no complications arise, and you are well after a couple weeks, then you have a good chance of making a full recovery. A main objective then is to get back into normal life, and to minimise the risk of a further MI.

1.3.4.8 Risk factors

MI is common. About 146,000 people in the UK have an MI each year. Most MIs occur in people over 50 and become more common with increasing age. Sometimes younger people are affected. An MI is three times more common in young men than young women. However, after the menopause, the female hormones no longer protect the heart

so the risk of having a MI is then the same for men and women.

An MI may occur in people known to have heart disease such as people with angina. It can also happen 'out of the blue' in people with no previous symptoms of heart disease. (Atheroma often develops without any symptoms at first.) Certain risk factors increase the risk of more atheroma forming which can lead to an MI occurring. **(Tim Kenny, 2010)**

Six primary risk factors have been identified with the development of atherosclerotic coronary artery disease and MI—hyperlipidemia, diabetes mellitus, hypertension, smoking, male gender, and family history of atherosclerotic arterial disease. The presence of any risk factor is associated with doubling the relative risk of developing atherosclerotic coronary artery disease. **(H. Michael Bolloki, 2009)**

1.3.4.8.1 Modifiable risk factors

High Blood Cholesterol Level.

An elevated level of total cholesterol is associated with an increased risk of coronary atherosclerosis and MI. Laboratory testing provides a measure of certain types of circulating fat particles. Elevated levels of low-density lipoprotein (LDL) cholesterol are associated with an increased incidence of atherosclerosis and MI. A full summary of the National Heart, Lung, and Blood Institute's cholesterol guidelines is available online and includes a free Palm OS software download for point of care use.

Diabetes Mellitus.

Diabetics have a substantially greater risk of atherosclerotic vascular disease in the heart as well as in other areas of their vasculature. Diabetes increases the risk of MI because it increases the rate of atherosclerotic progression and adversely affects blood cholesterol levels. This accelerated form of atherosclerosis occurs regardless of whether a patient has insulin- or noninsulin-dependent diabetes.

Hypertension.

High blood pressure (BP) has consistently been associated with an increased risk of MI. This risk is associated with systolic and diastolic hypertension. The control of hypertension with appropriate medication has been shown to reduce the risk of MI significantly. A full summary of the National Heart, Lung, and Blood Institute's JNC VI guidelines is available online.

Tobacco Use.

Certain components of tobacco and tobacco combustion gases are known to damage blood vessel walls. The body's response to this type of injury elicits the formation of atherosclerosis and its progression, thereby increasing the risk of MI. The American Lung Association maintains a website with updates on the public health initiative to reduce tobacco use and is a resource for smoking cessation strategies for patients and health care providers. Other public and private sources of smoking cessation information are also available online. **(H. Michael Bolloki, 2009)**

Obesity and overweight: People who have excess body fat — especially if a lot of it is at the waist — are more likely to develop heart disease and stroke even if they have no other risk factors. Excess weight increases the heart's work. It also raises blood pressure and blood cholesterol and triglyceride levels, and lowers HDL ("good") cholesterol levels. It can also make diabetes more likely to develop. Many obese and overweight people may have difficulty losing weight. But by losing even as few as 10 pounds, you can lower your heart disease risk.

Mental Stress: Individual response to stress may be a contributing factor. Some scientists have noted a relationship between coronary heart disease risk and stress in a person's life, their health behaviors and socioeconomic status. These factors may affect established risk factors. For example, people under stress may overeat, start smoking or smoke more than they otherwise would.

Physical inactivity: An inactive lifestyle is a risk factor for coronary heart disease. Regular, moderate-to-vigorous physical activity helps prevent heart and blood vessel disease. The more vigorous the activity, the greater your benefits. However, even moderate-intensity activities help if done regularly and long term. Physical activity can help control blood cholesterol, diabetes and obesity, as well as help lower blood pressure in some people.

Alcohol: Drinking too much alcohol can raise blood pressure, cause heart failure and lead to stroke. It can contribute to high triglycerides, cancer and other diseases, and produce irregular heartbeats. It contributes to obesity, alcoholism, suicide and accidents. The risk of heart disease in people who drink moderate amounts of alcohol (an average of one drink for women or two drinks for men per day) is lower than in nondrinkers. One drink is defined as 1-1/2 fluid ounces (fl oz) of 80-proof spirits (such as bourbon, Scotch, vodka, gin, etc.), 1 fl oz of 100-proof spirits, 4 fl oz of wine or 12 fl oz of beer. It's not recommended that nondrinkers start using alcohol or that drinker's increase the amount they drink.

Diet and Nutrition: A healthy diet is one of the best weapons you have to fight cardiovascular disease. The food people eat (and the amount) can affect other controllable risk factors: cholesterol, blood pressure, diabetes and overweight. Choose nutrient-rich foods — which have vitamins, minerals, fiber and other nutrients but are lower in calories — over nutrient-poor foods. A diet rich in vegetables, fruits, whole-grain and high-fiber foods, fish, lean protein and fat-free or low-fat dairy products is the key. **(American Heart Association, 2009).**

1.3.4.8.2 Non modifiable risk factors

- Family history. Individual's risk is increased if there is a family history of heart disease or a stroke that occurred in patient's father or brother aged below 55, or in mother or sister aged below 65.
- Ethnic group. Certain ethnic groups, for example British Asians, have a higher risk of developing cardiovascular diseases.

Figure3. Comparison of population attributable risks between women & woman

		Odds ratio (95% CI)	PAR (95% CI)	*P* interaction
A1	♀	3.30 (2.85,3.82)	43.1 (38.2,48.1)	0.20
	♂	2.87 (2.63.3.13)	46.6 (43.3,50.0)	
king	♀	2.86 (2.47,3.32)	15.5 (13.5,17.5)	0.06
	♂	3.04 (2.84,3.26)	46.3 (44.1,48.4)	
king	♀	1.04 (0.88.1.22)	2.5 (0.6,4.5)	<0.0001
	♂	1.61 (1.49,1.74)	18.1 (15.2,21.1)	
on	♀	2.95 (2.66,3.28)	35.8 (33.0,38.6)	0.0001
	♂	2.32 (2.16,2.48)	19.5 (18.1,21.0)	
	♀	4.26 (3.68,4.94)	19.1 (17.2,21.0)	<0.0001
	♂	2.67 (2.43,2.94)	10.1 (9.2,11.0)	
al	♀	2.26 (1.98,2.57)	35.9 (30.3.41.5)	0.03
	♂	2.24 (2.08,2.42)	32.1 (28.8,35.4)	
cial	♀	3.49 (2.40.5.09)	25.7 (18.4,33.1)	0.02
	♂	2.58 (2.11.3.15)	21.7 (17.0,26.4)	
	♀	2.07 (1.77,2.43)	37.3 (28.0,46.6)	<0.0001
	♂	1.30 (1.20,1.41)	22.9 (17.8,27.9)	
phol	♀	2.42 (2.00,2.93)	46.9 (36.9.56.9)	<0.0001
	♂	1.13 (1.06,121)	10.5 (6.3,14.8)	
et	♀	1.78 (1.54,2.04)	25.5 (18.4.32.6)	0.20
	♂	1.68 (1.56,1.82)	26.8 (22.6,31.1)	

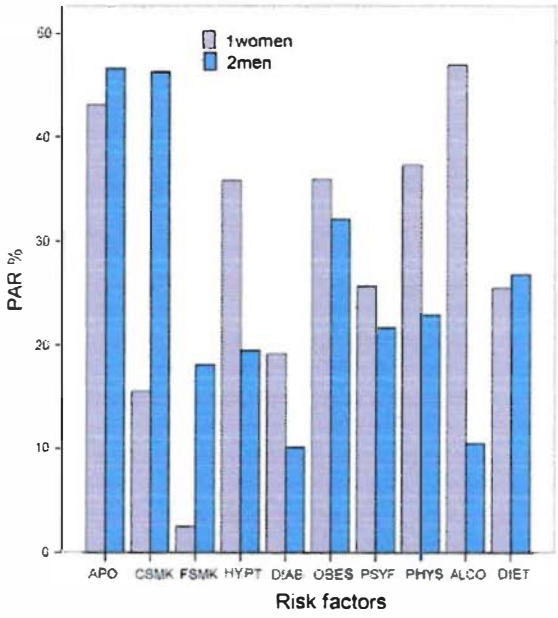

Figure3. shows Comparison of population attributable risks between women and men. APO, ApoB/A ratio; CSMK, current smoking; FSMK, former smoking; HYPT, hypertension; DIAB, diabetes; OBES, abdominal obesity; PSYF, psychosocial; PHYS, physical Inactivity; ALCO, lack of alcohol intake; DIET, high risk diet **(Daniel G Hackam, 2003)**

1.3.4.9 Magnitude of Myocardial Infraction

1.3.4.9.1 Magnitude of myocardial Infraction in World Wide

The importance of cardiovascular disease to the health of the world can be easily stated (Figure-4). Approximately one-third of the total deaths worldwide are cardiovascular in origin. Of those,

43% arise from coronary heart disease and 32% from stroke. The cardiovascular deaths amount to 16.6 million compared with 5.7 million from the major infectious diseases (AIDS, tuberculosis

and malaria) (Figure 3 and 4).22 Crucially, 78% of these cardiovascular deaths are not in the high income countries of the world but in the lower and middle income countries. There is variation across the world: for example, in some countries such as China stroke is more common than coronary heart disease whereas the reverse is true in other countries. In China, urbanization is accompanied by an increase in coronary heart disease relative to cerebrovascular disease. In higher income countries both are falling on an age-adjusted basis but, since demographics are changing (more elderly persons), the total number of persons with these afflictions will be increasing over the next few decades.

The higher number of people dying of cardiovascular disease in the developing world is a fact usually not appreciated and widely ignored. It is certainly true that cardiovascular disease tends to occur in older patients and infections in younger patients, but ischaemic heart disease and cerebrovascular disease still remain important causes of death (Fig 4) even in those under the age of 60. It is projected that the two entities will be the first and second causes of death by 2020.1 Even in sub-Saharan Africa cardiovascular disease is a major cause of death in the age range 15–60 years, killing more persons than infectious diseases, and the probability of death from a non-communicable disease is higher than in established market economies.

Furthermore, these deaths take out of the population people at an age when they are contributing to the economic, social and political stability of their country.

(Philip Poole-Wilson, 2005)

Figure 4. Global causes of death

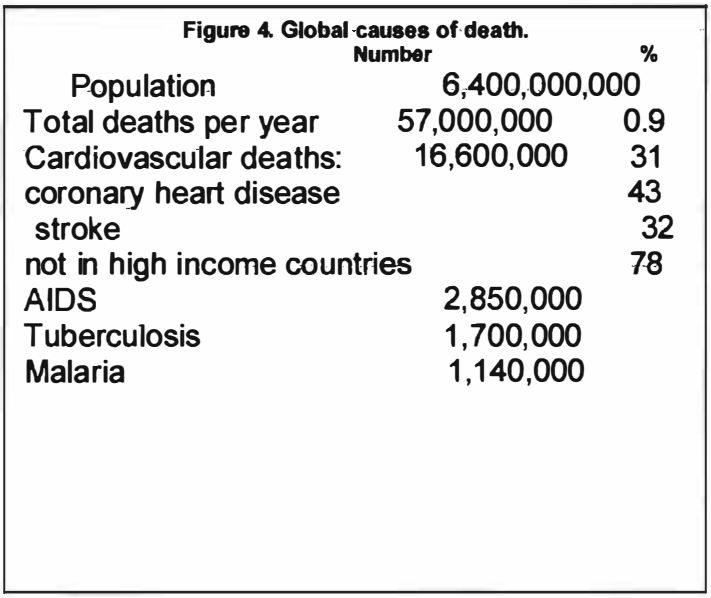

Figure 4. Global causes of death.

	Number	%
Population	6,400,000,000	
Total deaths per year	57,000,000	0.9
Cardiovascular deaths:	16,600,000	31
coronary heart disease		43
stroke		32
not in high income countries		78
AIDS	2,850,000	
Tuberculosis	1,700,000	
Malaria	1,140,000	

(Philip Poole-Wilson, 2005)

1.3.4.9.2 Magnitude of myocardial Infraction in Asian Countries

The mean (SD) age for first AMI was lower in South Asian countries (53.0 [11.4] years) than in other countries (58.8 [12.2] years<.001). Protective factors were lower in South Asian controls than in controls from other countries (moderate- or high-intensity exercise, 6.1% vs. 21.6%; daily intake of fruits and vegetables, 26.5% vs. 45.2%; alcohol consumption ≥once/wk, 10.7% vs. 26.9%). However, some harmful factors were more common in native South Asians than in individuals from other countries (elevated apolipoprotein B_{100} /apolipoprotein A-I ratio, 43.8% vs 31.8%; history of diabetes, 9.5% vs. 7.2%). Similar relative associations were found in South Asians compared with individuals from other countries for the risk factors of current and former smoking, apolipoprotein B_{100} /apolipoprotein A-I ratio for the top vs. lowest tertile, waist-to-hip ratio for the top vs. lowest tertile, history of hypertension, history of diabetes, psychosocial factors such as depression and stress at work or home, regular moderate- or high-intensity exercise, and daily intake of fruits and vegetables. Alcohol consumption was not found to be a risk factor for AMI in South Asians. The combined odds ratio for all 9 risk factors was similar in South Asians (123.3; 95% confidence

interval [CI], 38.7-400.2] and in individuals from other countries (125.7; 95% CI, 88.5-178.4). The similarities in the odds ratios for the risk factors explained a high and similar degree of population attributable risk in both groups (85.8% [95% CI, 78.0%-93.7%] vs 88.2% [95% CI, 86.3%-89.9%], respectively). When stratified by age, South Asians had more risk factors at ages younger than 60 years. After adjusting for all 9 risk factors, the predictive probability of classifying an AMI case as being younger than 40 years was similar in individuals from South Asian countries and those from other countries.

The 4 main risk factors, which showed consistently significant associations across all South Asian countries in both sexes were current and former smoking, high $ApoB_{100}$ /Apo-I ratio, history of hypertension, and history of diabetes. Alcohol consumption did not appear to be protective in native South Asians and this may be related to lower prevalence or differences in patterns of drinking (binge drinking in South Asians vs regular drinking in other countries).

The higher PAR due to low daily consumption of fruits and vegetables, lack of regular exercise, and high WHR observed among native South Asians compared with individuals from other countries contributes to the higher rates of CHD observed in South Asians. The 9 risk factors collectively explained 86.0% of the risk in South Asians and suggests that modifying behavior related to known risk factors could lead to a substantial impact. The role of other novel risk factors such as lipoprotein(a) or homocysteine, which are elevated in South Asians, in causing CHD is unclear. Recent randomized trials of homocysteine lowering have not demonstrated a reduction in CHD.[23-25] Thus, the role of novel risk factors as an important cause for CHD in South Asians is likely to be small. **(Prashant Joshi, 2007)**

Figure 5. Distribution of AMI between South Asian people and other countries:

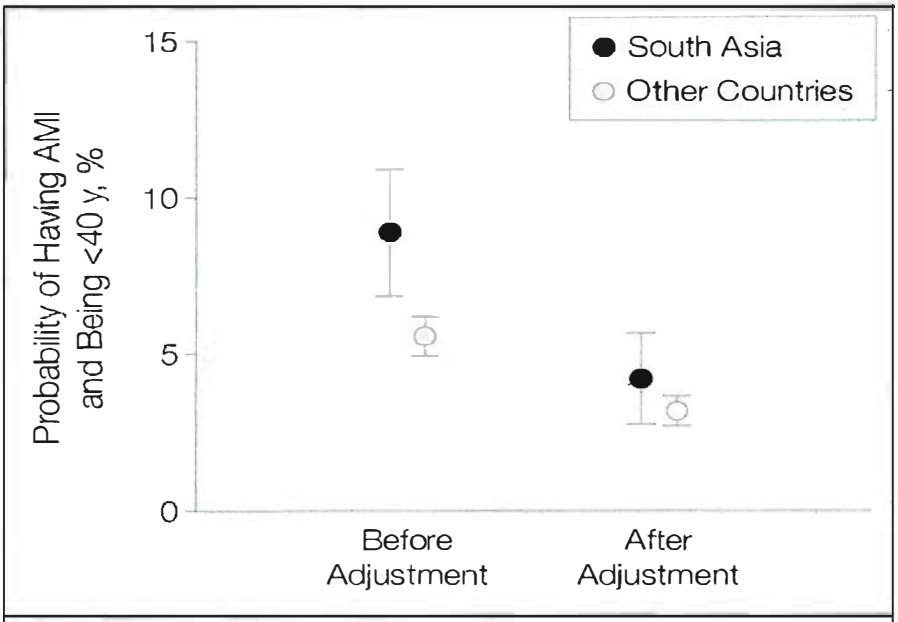

Figure5. This analysis was performed among acute myocardial infarction (AMI) cases only. Before adjustment for the 9 risk factors, there was a higher probability of cases who were younger than 40 years in the South Asian group compared with cases from other countries ($P = .001$). However, after adjustment for the 9 risk factors, the difference in probabilities of predicted cases of AMI in younger persons was attenuated and not statistically significant ($P = .27$). Error bars indicate 95% confidence intervals.

(Prashant Joshi, 2007)

1.3.4.9.3 Magnitude of myocardial Infraction in SAARC countries:

South Asia, housing more than half of the worlds poor and illiterate and constituting 23% of the world's population is a notable reality in the face of the cardiovascular disease epidemic. The meager per capita average budgetary expenditure on health ranging between US $ 11-20i for different countries of the region further highlights this challenge. The prevention and control of cardiovascular diseases is therefore one of the major health care issues faced by South Asia; the fact that this remains largely unrecognized adds to the gravity of the situation. Cardiovascular diseases are low on the

list of priorities in these countries where reproductive and communicable disease issues dominate health care; the economic impact of these diseases and its implications for disadvantaged groups also remains largely unrecognized. A significant lack of awareness about the magnitude of the problem at all levels and a consequent lack of will to address this issue is therefore likely to have serious implications.

Same ethnic group, sharing cultural similarities, geographic proximity, economic priorities, and similar epidemiological characteristics of cardiovascular disease.vii This need was emphasized upon at the executive council meeting of the SAARC Cardiac Society in July 2000, where it was proposed that a series of practice guidelines be formulated for the region through consensus amongst member countries. The first in this series are guidelines for the prevention of CHD in South Asia. In addition to clinical guidelines, this document also addresses key public health and policy issues specific to South Asia and summarizes public health and policy recommendations that have been identified as being crucial to the preventive approach to CHD in the region. Prevention of CHD has been categorized under primordial, primary and secondary, employing both behavioral and drug interventions with recommended goals. Tertiary prevention has not been dealt with. Recommendations highlight the public health and clinical approaches, with considerable overlap. Key policy issues relating to heart disease prevention have been alluded to in an attempt to sensitize clinicians to the crucial relevance of policy changes in the larger perspective of Cardiovascular disease prevention

Figure 6. Cardiovascular health services in South Asia

Figure 6. Cardiovascular health services in South Asia

	Pakistan	India	Bangladesh	Nepal	Sri Lanka
Population (million)	141.55	1014	129.19	24.7	19.24
Cardiologists	384	2,500	110	44	14
Cardiac surgeons	19	15,00	25	7	9
Cardiology Trainees	12	250	200	4	10
CV nurses	102	N/a	200	67	65
CV technicians	276	N/a	400	28	15
Cardiology departments	18	20	18	4	4
Cardiac surgery units	12	80	2	2	2
Catheter laboratories	13	160	2	1	2
Yearly catheter interventions	532	10,000	100	N/a	N/a
Open heart surgeries	3650	20,000	700	150	N/a

(Sania Nishtar, 2002)

1.3.4.9.4 Magnitude of myocardial Infraction in Bangladesh:

In Bangladesh, Healthy life expectancy at birth was 54.3 years in 2002 with 55.3 years for male and 53.3 years for female **(The World Health Report, 2004).**

There were no significant differences between Bangladeshi and white patients in the time from pain onset to hospital arrival (arrival time 64.5 (117.5) minutes *v* 63.0 (140.3) minutes, p = 0.63), but once in hospital it took almost twice as long for Bangladeshi as for white patients to receive thrombolysis (median (interquartile range) door to needle time 42.5 (78.0) minutes *v* 26.0 (47.7) minutes, p = 0.012). Bangladeshis were significantly less likely than whites to complain of central chest pain (odds ratio (OR) 0.11, 95% confidence interval (CI) 0.03 to 0.38; p = 0.0006) or to offer classic descriptions of the character of the pain (OR 0.25, 95% CI 0.09 to 0.74; p = 0.0118). These differences persisted after adjustment for age, sex, and risk factor profile differences including diabetes. Proportions of Bangladeshi and whites interpreting their

symptoms as "heart attack" were similar (45.2% *v*46.9%; p = 0.99). Bangladeshi patients with AMI often present with atypical symptoms, which may lead to slower triage in the casualty department and delay in essential treatment. This needs recognition by emergency staff if mortality rates in this high risk group are to be reduced.

Bangladeshis were significantly less likely than whites to complain of central chest pain (OR 0.11, 95% CI 0.03 to 0.38; p = 0.0006 (tables 2 and 3). These differences persisted after adjustment for differences in age, sex, and risk factor profiles including diabetes and for fluency in English. Similarly, Bangladeshis were less likely than whites to offer classic descriptions of the character of the pain (heaviness, tightness, weight, pressure, band-like, gripping) and were more likely to offer non-classic descriptions (sharp, stabbing, pinching, burning) (OR 0.25, 95% CI 0.09 to 0.74; p = 0.0118)

(K Barakat, 2002)

Figure 7. Ten most common causes of morbidity in hospitals in Bangladesh during 1997.

Diseases	% of all cases
Diarrhoea	15.90
Intestinal worm	7.38
Skin diseases	9.3
Anaemia	9.92
Acute Respiratory Infections	6.1
Deficiency diseases	6.63
Eye diseases	4.36
Injuries	4.35
Ear diseases	3.28
Asthma	2.31

(WHO. Country Health System Profile,Bangladesh, 2004)

1.4 Diabetes Mellitus:

The term diabetes mellitus describes a metabolic disorder of multiple aetiology characterized by chronic hyperglycemia with disturbance of carbohydrate, fat and protein metabolism resulting from defects in insulin secretion, insulin action or both. The effect of diabetes mellitus include long – term damage, dysfunction and failure of various organs. Diabetes mellitus may present with characteristic symptoms such as thirst, polyuria, blurring of vision, and weight loss. In its most severe forms, ketoacidosis or a non-ketotic hyperosmolar state may develop and lead to stupor, coma and, in absence of effective treatment, death. **(WHO, 2009)**

1.4.1 Types of diabetes Mellitus: There are two types of diabetes mellitus: type-1 and type-2.

1.4.1.1 Type-1 Diabetes Mellitus:

Type 1 diabetes is usually diagnosed in children and young adults, and was previously known as juvenile diabetes. In type 1 diabetes, the body does not produce insulin. Insulin is a hormone that is needed to convert sugar, starches and other food into energy needed for daily life. Only 5-10% of people with diabetes have this form of the disease. With the help of insulin therapyand other treatments, even young children with type 1 diabetes can learn to manage their condition and live long, healthy, happy lives. (**WHO, 2009**)

1.4.1.2 Type-2 Diabetes Mellitus:

Most diabetics are type-2 diabetics. The disease is influenced by genetic factors, aging, obesity, and peripheral insulin resistance rather than by autoimmune processes or viruses. The metabolic alterations observed are milder than those described for type-1, but the long term clinical consequence can be just as devastating. **(Lippincott Williams & wilkins, 3rd edition)**

1.4.2 Symptoms

Diabetes often goes undiagnosed because many of its symptoms seem so harmless. Recent studies indicate that the early detection of diabetes symptoms and treatment can decrease the chance of developing the complications of diabetes.

1.4.2.1 Symptoms for Type 1 Diabetes

- Frequent urination
- Unusual thirst
- Extreme hunger
- Unusual weight loss
- Extreme fatigue and Irritability

1.4.2.2 Symptoms for Type 2 Diabetes

- Any of the type 1 symptoms
- Frequent infections
- Blurred vision
- Cuts/bruises that are slow to heal
- Tingling/numbness in the hands/feet
- Recurring skin, gum, or bladder infection. **(American Diabetes Association, 2009)**

1.4.3 Complication:

The complications of diabetes mellitus include retinopathy, nephropathy, neuropathy, and increased risk for atherosclerotic vascular disease. Diabetes mellitus is the leading cause of blindness in young people and is comparable with macular degeneration as a cause of blindness in older adults. Diabetes mellitus is the leading cause of end-stage renal disease requiring renal replacement therapy, dialysis, or transplantation. DM is the leading cause

of no traumatic amputations of the lower extremity, a result of peripheral neuropathy and peripheral vascular disease. DM is associated with a two- to fivefold increased risk for CHD.

Two large trials—the Diabetes Control and Complications Trial (DCCT) and the United Kingdom Prospective Diabetes Study (UKPDS)—have demonstrated that there is a clear relation between glucose control and the risks for retinopathy (onset and progression), nephropathy (as measured by albuminuria), and neuropathy (both clinical and electromyographic measures. The relation between the degree of hyperglycemia and CHD risk has also been established, but this increased CHD risk begins well below the glycemic threshold for the diagnosis of diabetes.

Screening

Screening should be done for diabetic complications (Table-5).

Figure 8. Microvascular Complications in Diabetes Mellitus: Screening and Interventions:

Complication	Detection	Primary Prevention	Secondary Prevention
Retinopathy	Dilated eye examination (fundus photography); IV intravenous fluorescein angiography (IVFA); optical coherence imaging (OCT)	Glycemic control; BP control; lipid-lowering therapy	Glycemic control; BP control; laser therapy; lipid-lowering therapy ; corticosteroid injections ; anti-VEGF injections ; PKC β therapy
Nephropathy	Urine micoalbumin	Glycemic control BP control; ACEI-ARB therapy; lipid lowering therapy	Glycemic control; BP control; ACEI-ARB therapy; lipid-lowering therapy
Neuropathy	Monofilament testing	Daily foot inspection	Proper footwear; podiatry management—foot calluses, ulcers, deformities *

(H. Michael Bolloki, 2009)

1.4.4 Diagnostic criteria

The diagnosis of diabetes is based on several findings. The following criteria have been established by the American Diabetes Association:

1. Fasting glucose level higher than 126 mg/dL on two occasions. This fasting glucose value is based on data showing that blood glucose values above this fasting level are consistently associated with the risk for retinopathy, the diabetes complication essentially unique to diabetes. This cut point value will miss a number of patients who have diabetes based on oral glucose tolerance testing results. Because of simplicity, fasting blood glucose (BG) concentrations are one of the most common ways to diagnose DM. There are recent observational data suggesting that this threshold for the diagnosis DM may be too high, because patients with impaired glucose tolerance develop retinopathy.
2. Random or "casual" glucose higher than 200 mg/dL, with symptoms of DM. This is a common way to diagnose DM. Many patients may not have obvious symptoms, but that should not alter the fact that a random BG level in this range generally establishes the diagnosis of DM. This criterion is not affected by the time of the last meal.
3. Oral glucose tolerance test result after a 75-g oral glucose load, 2-hour value higher than 200 mg/dL.

The oral glucose tolerance test (OGTT) is not generally recommended in clinical practice. Such testing requires 3 days of high carbohydrate intake. False-positive test results may occur in the presence of recent fasting. Oral glucose tolerance testing is not always reproducible. Consequently, OGTT is generally considered a research tool, and often two OGTTs are necessary to establish a diagnosis of DM.

Hemoglobin A_{1c} ($HgbA_{1c}$) values are too insensitive to be used as a screening test for DM. Elevated values (e.g. higher than 6.2%) are usually associated with a diagnosis of DM, but patients may have a diagnosis of DM with values below this range. Thus,

elevated $HgbA_{1c}$ values are a somewhat specific test for the diagnosis of DM, but are not highly sensitive. **(Byron J. Hoogwerf, 2009)**

1.4.5 Prevention & Cure:

Guidelines for medical nutrition therapy have been established by the American Diabetes Association (ADA). The primary focus of these guidelines is targeted to outcomes including glycemic control, weight reduction (as appropriate), blood pressure control, and a favorable lipid profile. There is clear evidence that excess saturated fat in the diet has a detrimental effect on lipid profiles and therefore saturated fat restriction is recommended. The data supporting absolute restriction of carbohydrates are not robust, so the ADA guidelines allow flexibility in carbohydrate and non–saturated fat intake. Separate guidelines have been published about the carbohydrate content and composition of the diet.[8] The most important variable in prandial glycemic excursion is total carbohydrate intake. Low glycemic index foods consumed alone result in lower prandial glucose excursion than high glycemic index foods. However, in the context of a mixed meal, differences between low and high glycemic index foods are attenuated. The guidelines support the concept that glycemic index may be a consideration in nutrition therapy. Both the amount and source of carbohydrates are important determinants of postprandial glucose. The relative effects of each have been recently studied. Brand-Miller and colleagues have reported that they analyzed the relative impact of the glycemic index and total carbohydrate content of individual foods on glycemic load—the product of glycemic index and total grams of carbohydrate—using linear regression analysis. Carbohydrate content (total grams) alone explained 68% of the variation in glycemic load, and the glycemic index of the food explained 49%. When total carbohydrate and glycemic index were both included in the regression analysis, the glycemic index accounted for 32% of the variation

1.5 Diabetes Associated with MI:

Diabetic subjects are more likely to experience a myocardial infraction and have worse outcomes compared to non-diabetic subjects.**(Ian L Williams, Brian Noronha, Azfar G Zaman, November 13, 2003).** Diabetes is associated with a marked increase in the risk of coronary heart disease. It has been debated whether patients with diabetes who have not had myocardial infarctions should be treated as aggressively for cardiovascular risk factors as patients who have had myocardial infarctions.

Diabetes is associated with a marked increase (by a factor of two to four) in the risk of coronary heart disease. Clinically established coronary heart disease itself is associated with an increase in mortality from coronary heart disease by a factor of three to seven, depending on the mode of presentation. The plasma cholesterol level is a strong predictor of the risk of cardiovascular events both in patients with diabetes and in patients with coronary heart disease. The high-risk status of these groups of patients and their need for more aggressive lipid-lowering therapy has been recognized by both the National Cholesterol Education Program and the American Diabetes Association.

The reduction in plasma lipids recommended by the National Cholesterol Education Program is greater for patients with coronary heart disease than for patients with diabetes. Rates of myocardial infarction in nondiabetic subjects with and without prior myocardial infarction at base line were 18.8 percent and 3.5 percent, respectively ($P<0.001$).**(Steven M. Haffner, 1998)**

In a recent, large, prospective cohort study that included 12 550 adults, the development of type II diabetes was almost 2.5 times as likely in persons with hypertension than in their normotensive counterparts. This, in conjunction with considerable evidence of the increased prevalence of hypertension in diabetic persons, suggests that these 2 common chronic diseases frequently coexist. Moreover, each pathophysiological disease entity, although independent in its own natural history, serves to exacerbate the other. In a recent report of Gress et al, hypertensive patients who were taking ß-blockers had a 28% higher risk of diabetes than did those taking no medication. In contrast, patients with

hypertension who received thiazide diuretics, ACE inhibitors, or Ca^{2+} antagonists were found not to be at greater risk for subsequent diabetes than were patients who were not receiving any antihypertensive medications. However, that study was not prospective or randomized, and other randomized prospective trials have not shown an increase in the development of diabetes with ß-blocker or low-dose diuretic treatment of hypertension. Recent studies have reported that ACE inhibitor therapy reduced the propensity of hypertensive patients to develop type-2 diabetes by 11% and 34% in trials extending for 6 and 4 years, respectively, suggesting that antihypertensive treatment may have a significant impact on the propensity for the development of diabetes in this population. These observations are in contrast to a recent report in which no reduction in progression to diabetes on ACE inhibition therapy was observed. Thus, more controlled randomized prospective trials are required to address the potential for ACE inhibitor therapy to reduce the rate of development of diabetes in hypertensive patients.

There is an increasing body of data from controlled clinical trials indicating that rigorous control of arterial pressure to levels <140/90 mm Hg markedly reduces cardiovascular disease morbidity and mortality and the development of end-stage renal disease in persons with type 2 diabetes. In the Systolic Hypertension in the Elderly Program study, elderly persons with type 2 diabetes derived more benefit from aggressive systolic blood pressure lowering in reduction of CVD than did those without diabetes. Baseline therapy in the SHEP study used a low-dose diuretic, which is often a necessary component of the antihypertensive regimen because of the sodium sensitivity and expanded plasma volume that is often present in diabetic patients. Data from the subset analysis of type II diabetes in the Hypertension Optimal Treatment trial suggest that reduction in diastolic pressures from <90 mm Hg to values <85 mm Hg is beneficial in reducing CVD events. The initial drug therapy in HOT was with a dihydropyridine Ca^{2+} antagonist, but >70% of diabetic patients required at least 3 drugs to control the diastolic pressure to levels <85 mm Hg. Special benefits of aggressive blood pressure lowering in the diabetic population was observed in a subanalysis of this cohort in the Systolic Hypertension in Europe Trial. In that trial, although systolic pressure was reduced by a comparable amount in each group the risk reduction in mortality from CVD was 13% in nondiabetic patients versus 76% for

the diabetic patients. Again, diabetic patients required more antihypertensive treatment to achieve goal blood pressures, with up to two thirds requiring ≥2 medications as previously observed. Moreover, the benefit confirmed per mm Hg blood pressure reduction was greater in diabetic patients than in those patients with hypertension but without concomitant diabetes mellitus, providing further evidence for rigorous reduction of arterial pressure in diabetic patients. **(James R. Sowers, 2001.)**

However, there were differing opinions among members of the National Cholesterol Education Program panel, with some suggesting that diabetic patients should have the same intensity of cholesterol-lowering therapy as patients with coronary heart disease. Thus, there is controversy about how aggressively to treat cardiovascular risk factors in patients with diabetes. It has been suggested that such patients should be treated as if they had established coronary heart disease. **(Steven M. Haffner, 1998)**

CHAPTER 2

AIM & SIGNIFICANCE OF THE STUDY

2.1. Aim of the study

Diabetes is one of the major risks of myocardial infarction. It can modify, treat or control by changing the lifestyle or taking medicine. The risks of occurring myocardial infarction are greater if blood sugar is not well controlled.

All the factors keeping in mind the present study was designed to assess the distribution of diabetes among patients with myocardial infraction.

2.2. Significance of the study

Diabetes mellitus is a heterogeneous group of metabolic disorders which is characterized by hyperglycemia. Any kind of defects in insulin secretion, insulin action, or both are the causes of hyperglycemia. Diabetes mellitus is a complex disease where multiple levels of abnormalities are present in various tissues.

The prevalence of diabetes is increasing day by day. Diabetes patients have a higher prevalence of myocardial infarction compared to the normal population.

Long term duration of uncontrolled diabetes may cause various disorders, mainly involving small vessels. The disorders include retinopathy, nephropathy and neuropathy which ultimately lead to visual disturbance, renal failure and gangrene. Diabetes accelerates and exacerbates the occurrence of arteriosclerosis, increasing the risk of myocardial infarction, cerebral infarction and occlusive artery disease of the lower extremities. The morbidity and mortality of these patients are due to these complications.

Globally, non- communicable diseases (NCDs) are increasingly recognized as a major cause of morbidity and mortality. Coronary Heart Disease is one of the non communicable diseases. Myocardial Infarction is one kind of coronary heart disease. Diabetic Mellitus is the major risk factor of Myocardial Infarction among other risk factors which can modify, treat or control by changing lifestyle or taking medicine.

This study will be help to increase the awareness between people health by taking immediate treatment, by taking drug, or by controlling blood sugar level, food habit 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 diabetes and Myocardial Infarction. Thus, the result of the study is expected to improve management of Myocardial Infarction in patients with diabetes which ultimately will help to improve the disease management process.

CHAPTER 3

MATERIALS AND METHODS

3.1 Type of study

This study was attempted to find out the distribution of diabetes in patients with Myocardial Infraction. In addition to this, the study examined for other risk factors and presence of MI.

3.2 Place of study

The study was 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

One thirty five (135) patients diagnosed to have myocardial infarction 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 Sampling Technique

In this study, purposive sampling technique was followed.

3.5 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).

3.6 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.

3.7 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, clinical examination and laboratory tests.

3.7.1 Blood pressure Measurement

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

3.8 Study period

Study period was 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.

3.9 Statistical analysis

All the data were checked after collection. Then data was entered into personal computer using Excel programming. 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 Infraction among male and female patients:

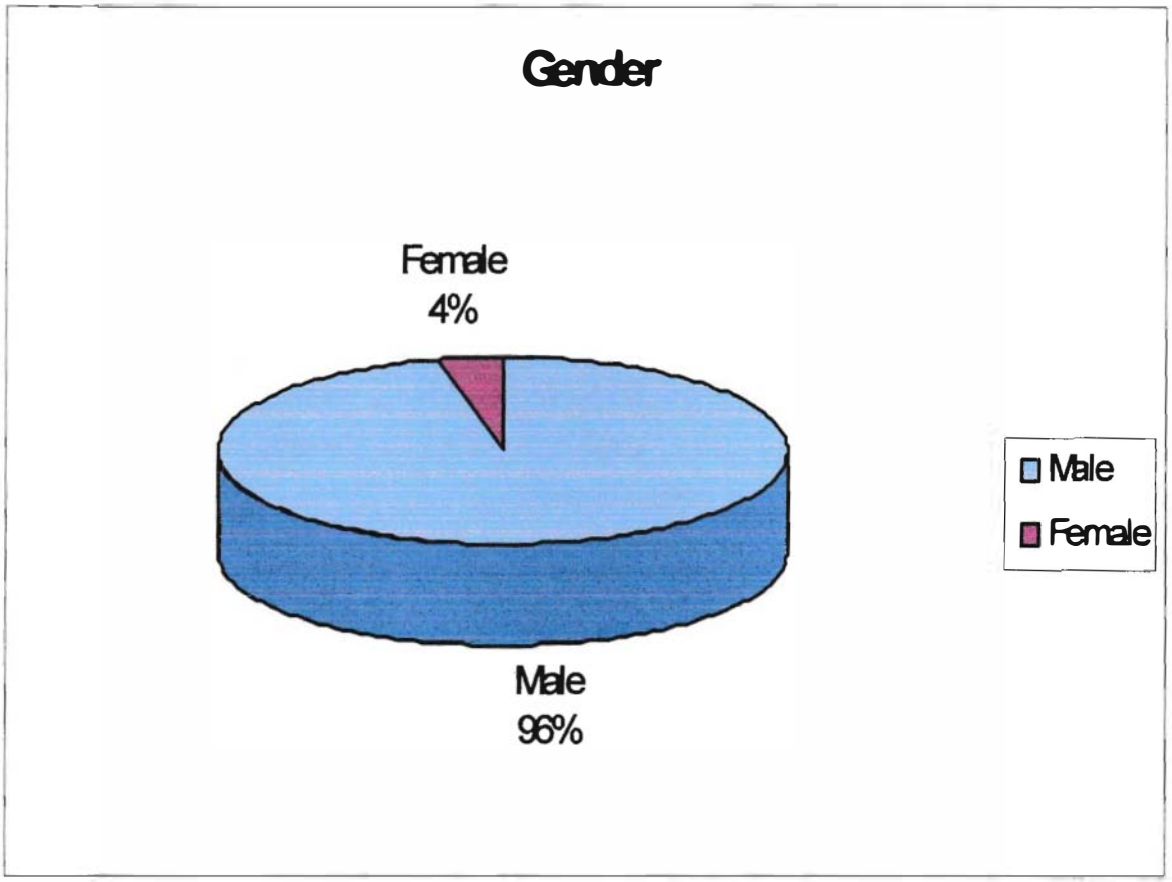

Fig 9: Distribution of Myocardial Infraction among male and female patients.

This figure shows 96% male and 4% female patients have Myocardial Infraction.

4.2 Distribution of Myocardial infraction among different religions:

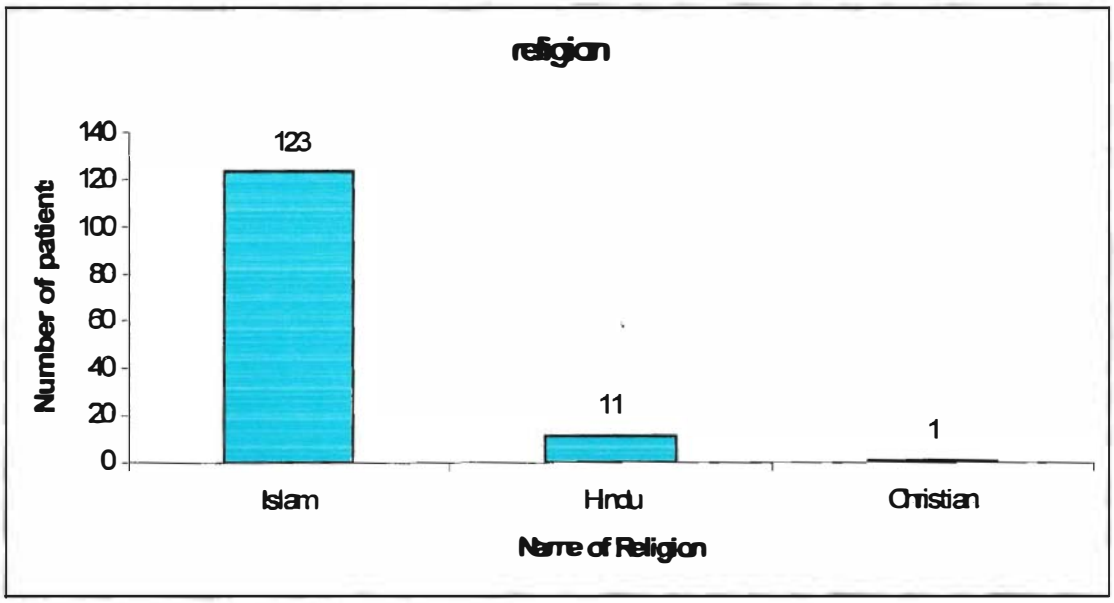

Fig 10: Distribution of Myocardial infraction among different religions.

This figure shows that 123 MI patients (91.11%) were Muslim, 11 patients (8.15%) were Hindus and 1 patient (0.74%) was Christian.

4.3 Distribution of Myocardial Infraction among patients according to age variation:

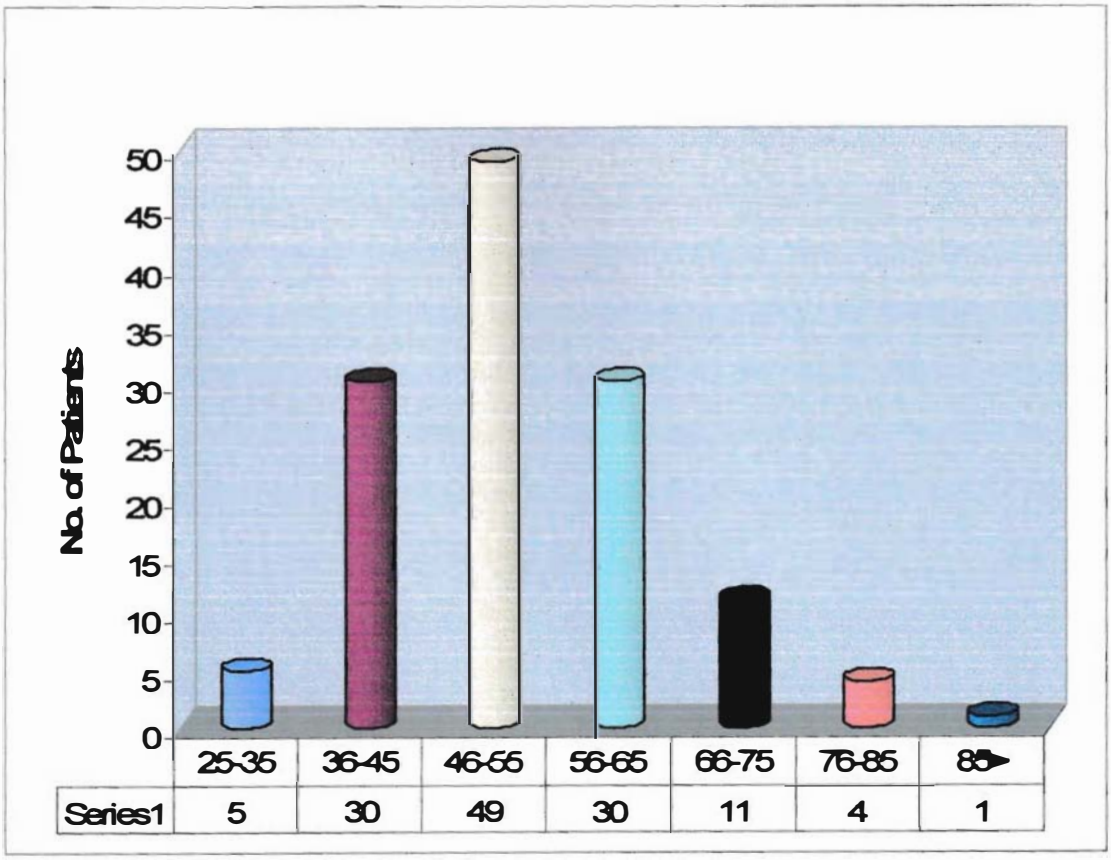

Fig 11: Distribution of Myocardial Infraction among patients according to age variation.

This figure shows 5 patients are in 25-35 age range, 30 patients are in 36-45 age range, 49 patients are in 46-55 age range, 30 patients are in 56-66 age range, 11 patients are in 66-75 age range, 4% patients are in 76-85 age range, 1 patient has age over 85 years.

4.4 Distribution of MI according to patients' occupation:

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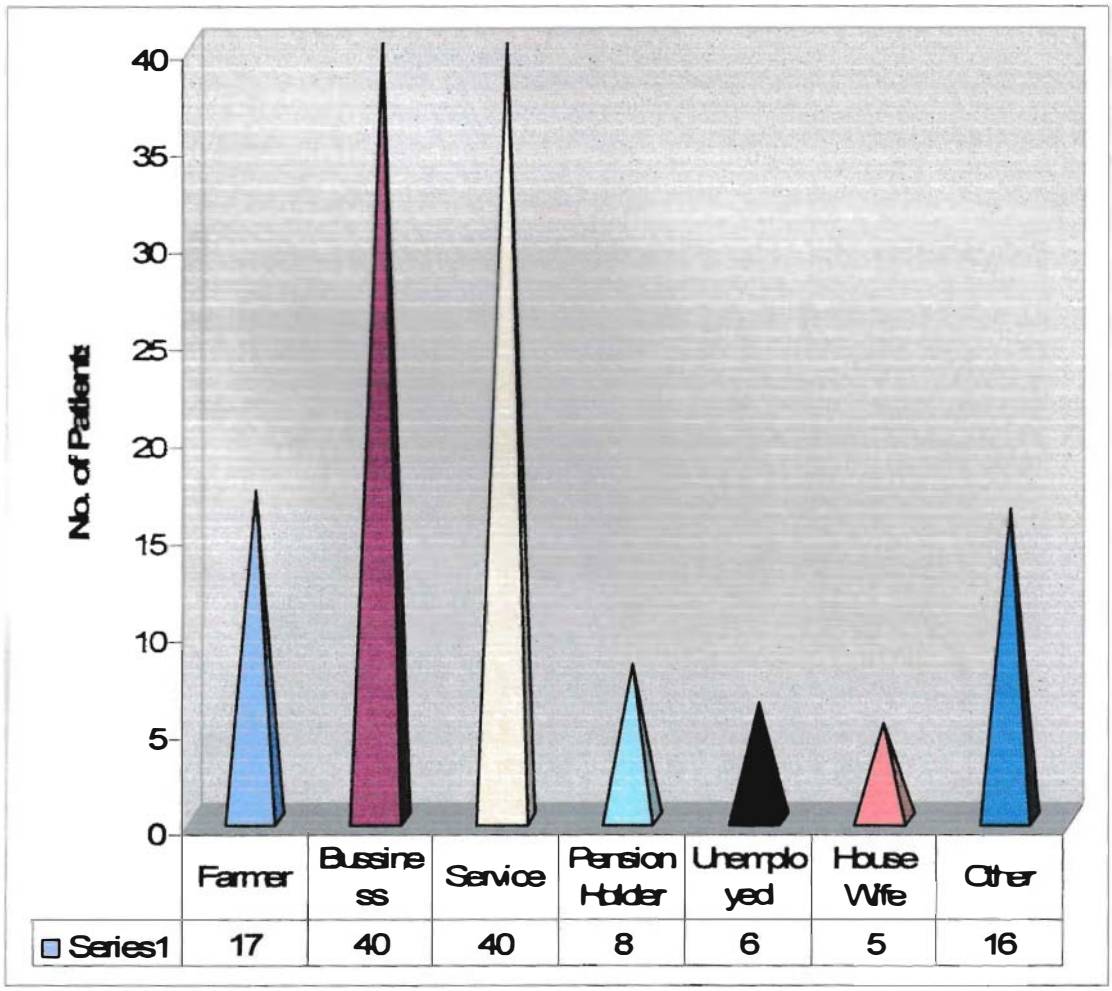

Fig 12: Distribution of MI according to patients' occupation.

This figure shows 29.63% MI patients are business man, 29..63% are service holder, 12.59% are farmer, 5.93% are pension holder, unemployed 4.44%, House Wife 3.70%. 11.85% MI patients are involved in other occupations.

4.5 Distribution of Myocardial Infraction according to the family income of patients:

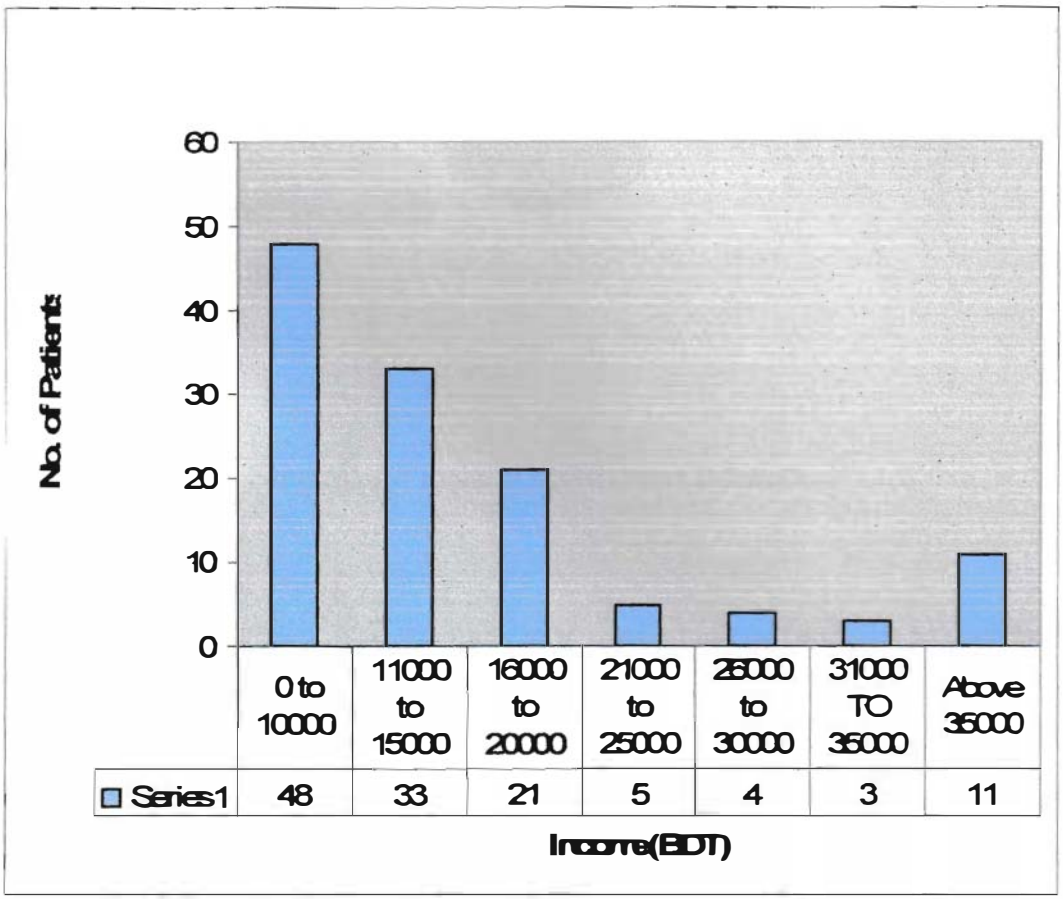

Fig 13: Distribution of Myocardial Infraction according to the family income of patients.

This figure shows 38.40% patients have income less than 10000 BDT per month, 26.40% have 11000-15000, 16.80% have 16000-20000, 4% have 21000-25000, 3.20% have 26000-30000, 2.40% have 31000-35000, 8.80% have above 35000 BDT per month.

.6 Percentage of different types of Myocardial Infraction:

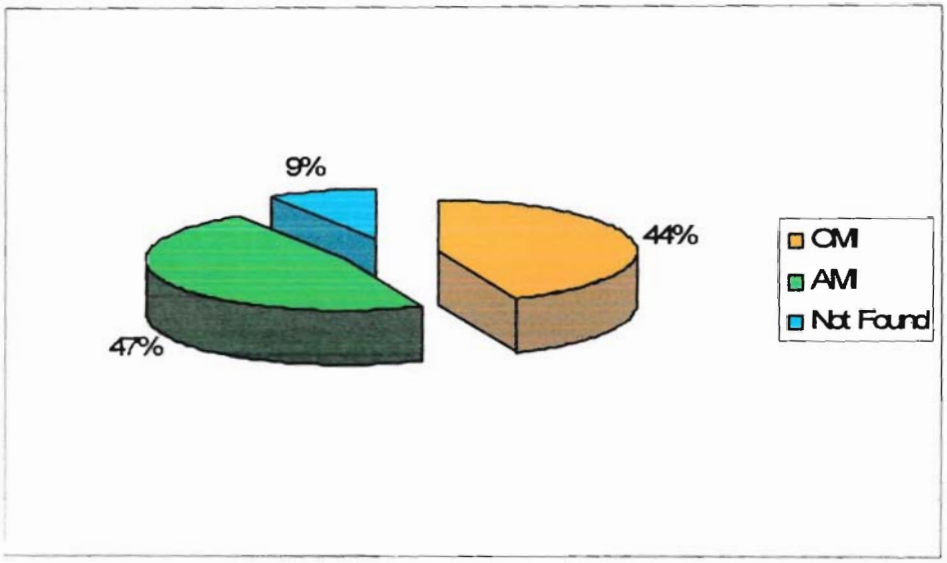

Fig 14: Percentage of different types of Myocardial Infraction.

This figure shows 47% patient has AMI, 44% patient has OMI and data has not found from 9% patient.

4.7 Percentage of MI patients with a history of smoking:

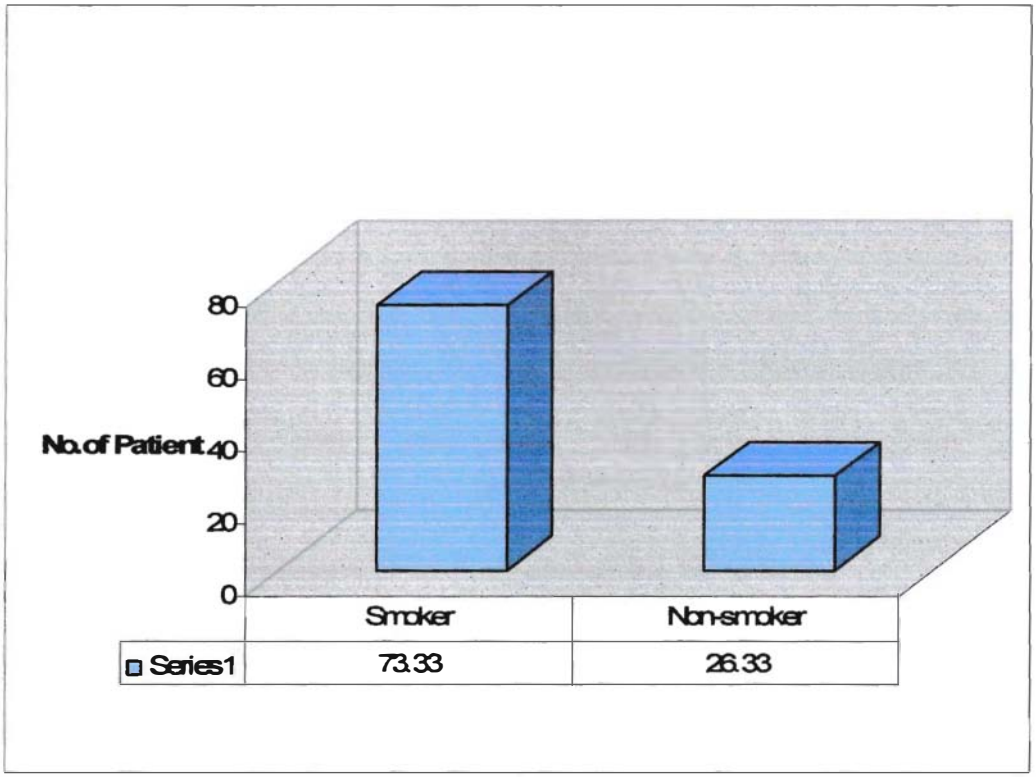

Fig 15: Percentage of MI patients with a history of smoking.

This figure shows that 73.33% MI patient are smoker and 26.33% MI patients are non-smoker.

4.8 Length of exposure to smoking among the MI patients:

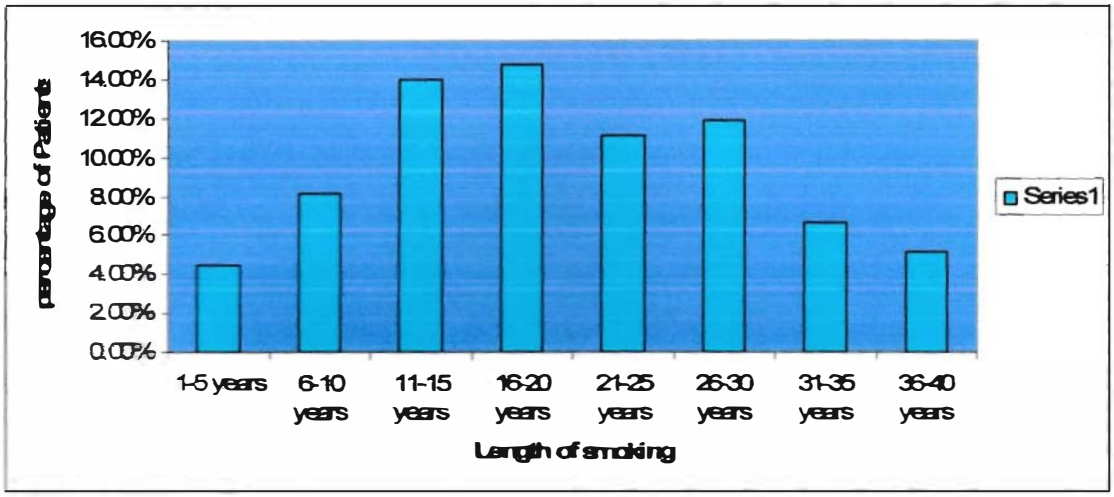

Fig 16: Length of exposure to smoking among the MI patients.

This figure shows duration of smoking among MI patients who are smoker. Here, 4.44% patients are smoking for 1-5 years, 8.1% are smoking for 6-10 years, 14.81%are smoking for16-20 years, 11.1% are smoking for 21-25 years, 11.86% are smoking for 26-30 years, 6.66% are smoking for 31-35 years, 5.2% are smoking for 36-40 years.

4.9 Distribution of Patients suffering from Myocardial Infraction with a habit of taking excess tea or coffee:

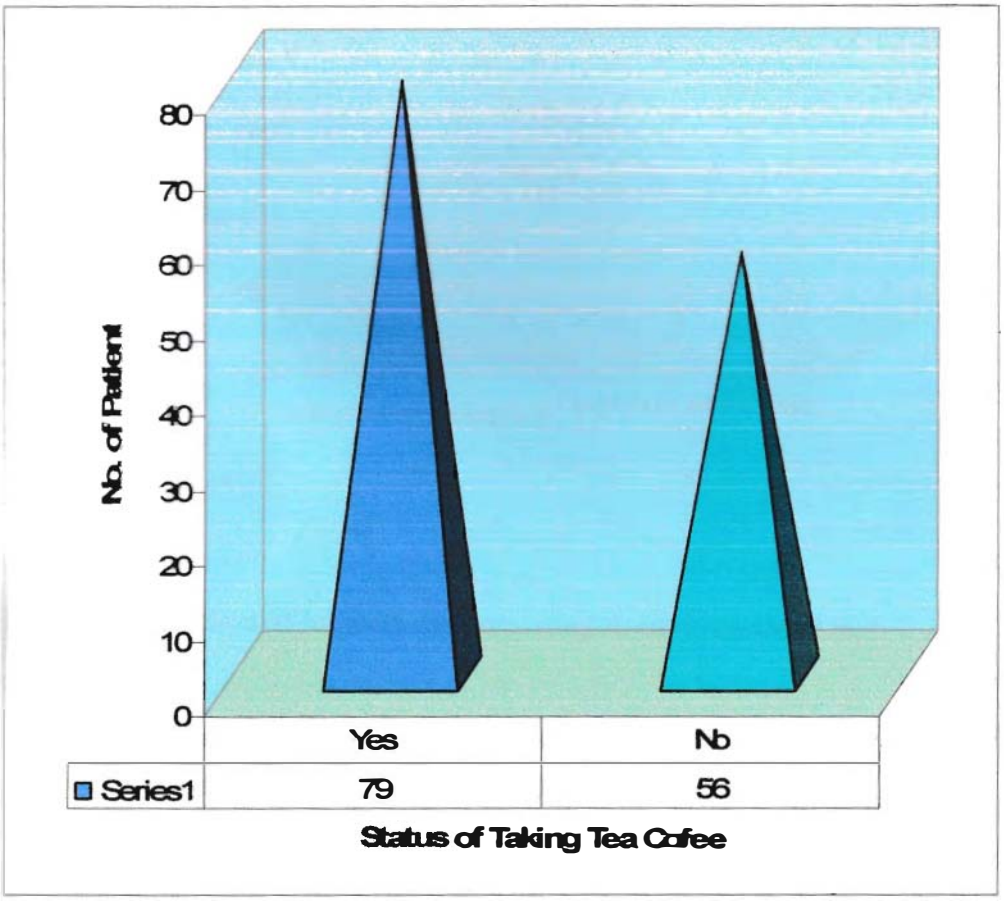

Fig 17: Distribution of Patients suffering from Myocardial Infraction with a habit of taking excess tea or coffee.

This shows that 58.52% MI patient taking excess tea or coffee and 41.48% are not.

4.10 Distribution of MI patients according to chewing Betel Nut

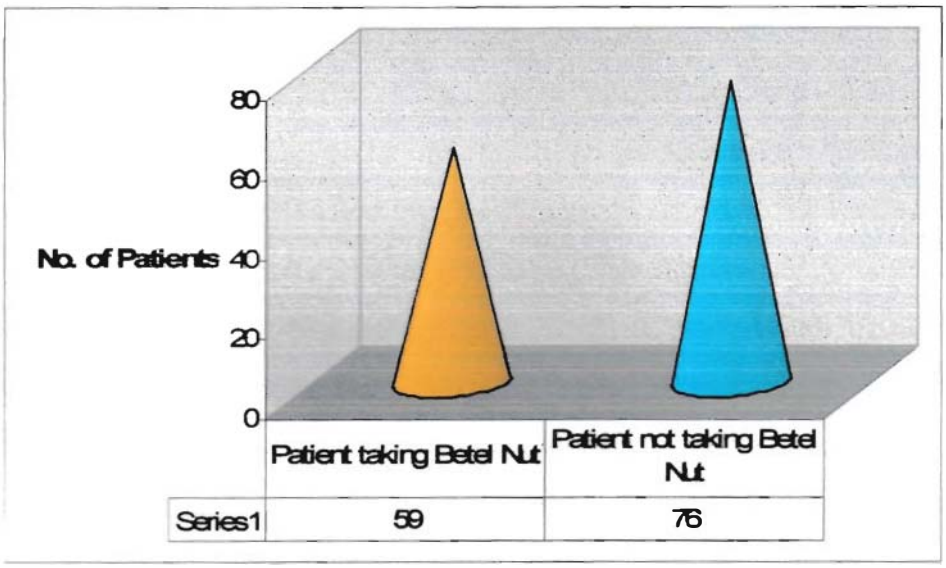

Fig18: Distribution of MI patients according to chewing Betel Nut.

This figure shows that 43.70% patients take Betel Nut and 56.30% patients don't take Betel Nut.

4.11 **Type of oil consumed by the MI patients:**

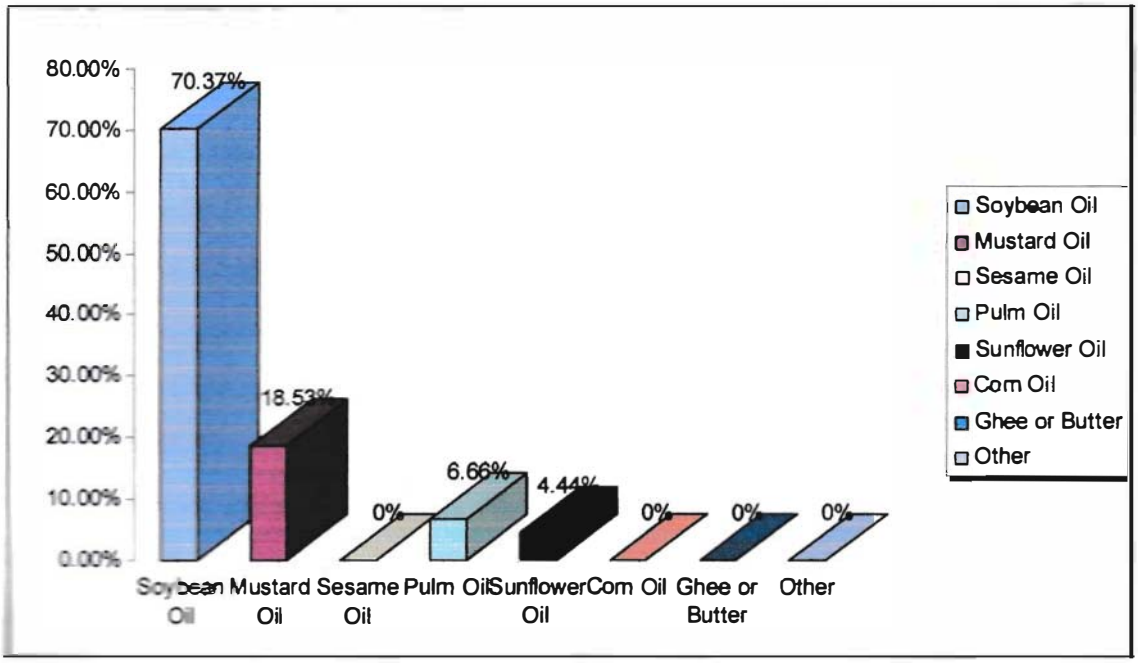

Fig 19: Distribution of MI depending on type of oil

This figure shows 70.37% patient take soybean oil, 18.53% take mustered oil, 6.66% take palm oil, 4.44% take sunflower oil.

4.12 Distribution of MI patients depending on salt intake:

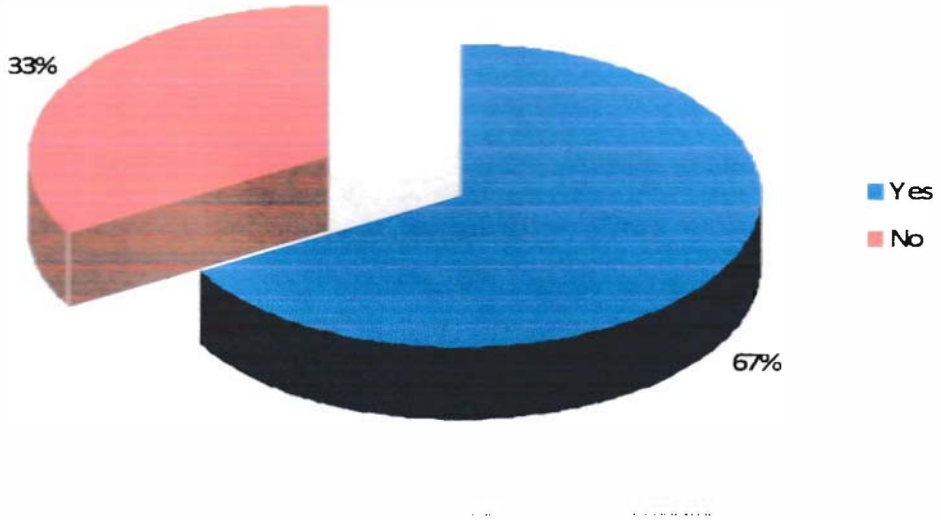

Fig 20: Distribution of MI depending on salt intake.

This figure shows 67% patients takes table salt and 33% patients don't take table salt.

4.14 Distribution of Myocardial Infraction depending on Random Blood Sugar of Patients.

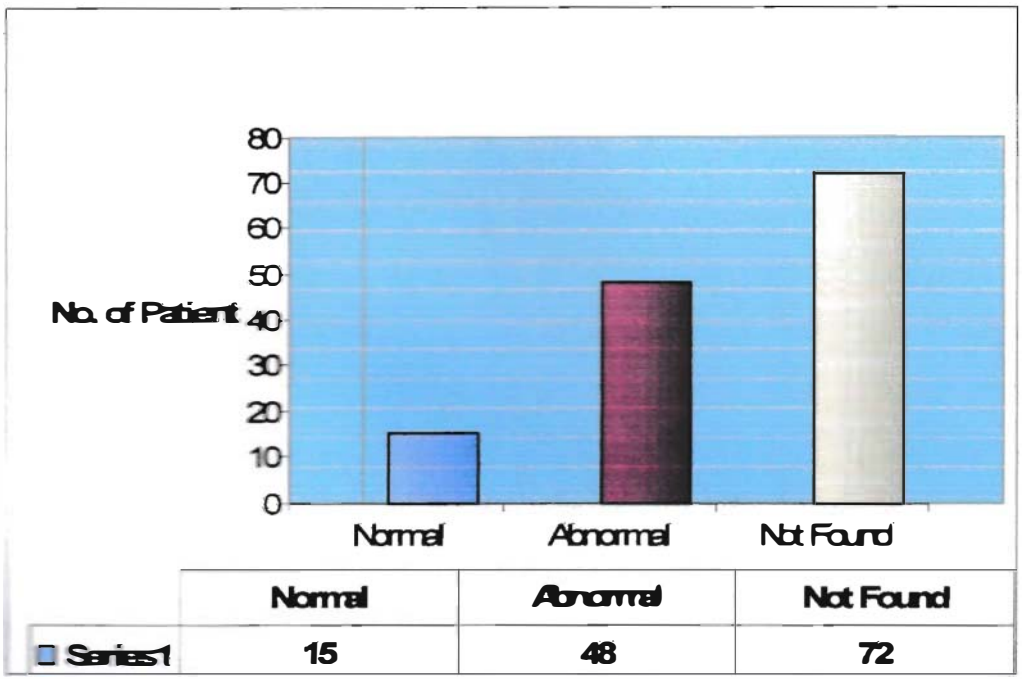

Fig 22: Distribution of Myocardial Infraction depending on Random Blood Sugar of Patients.

This figure shows 11.11% MI patients have normal RBS, 35.55% MI patients have abnormal RBS and for 53.33% patients the RBS data didn't found.

4.15 Distribution of Systolic BP among MI patients.

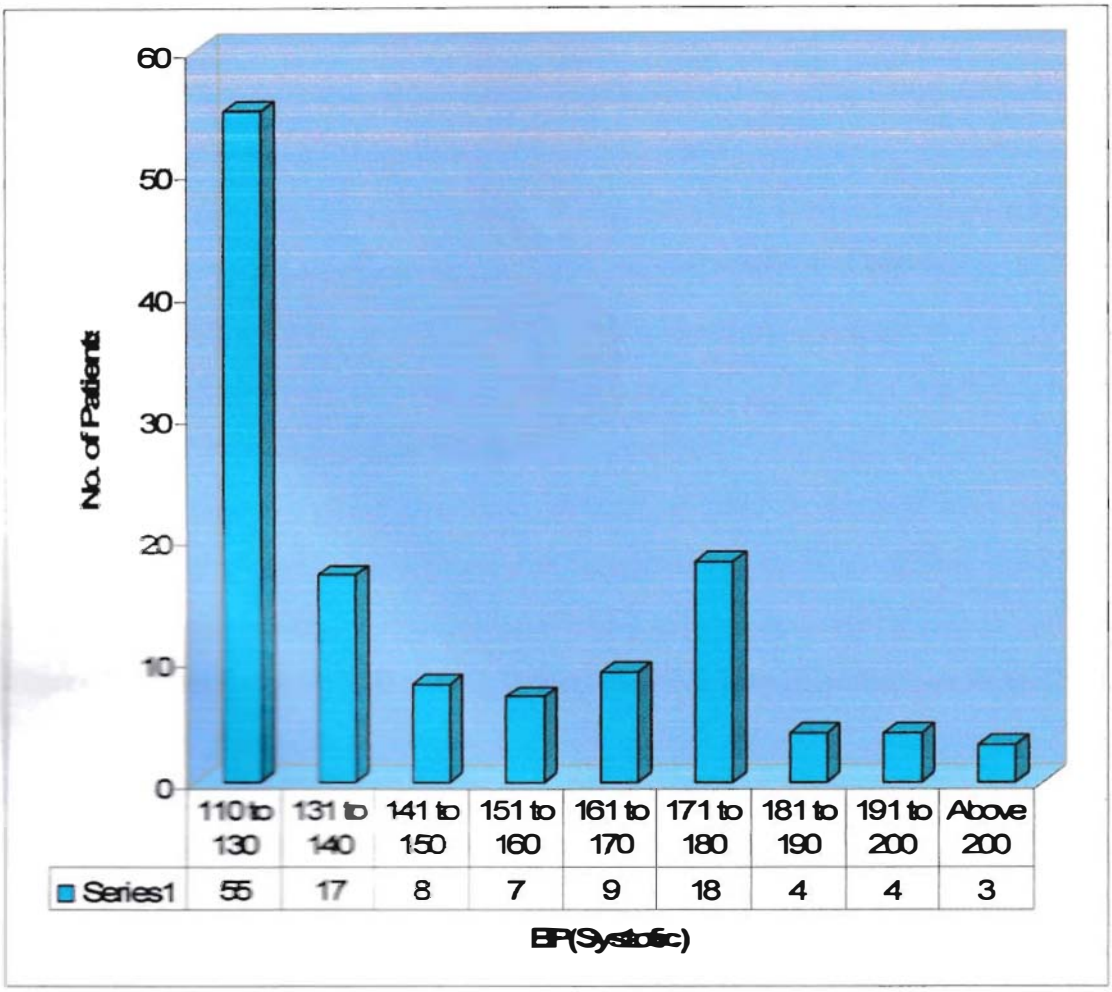

Fig 23: Distribution of Systolic BP among MI patients.

This figure shows that 45% have BP 110-130, 13% have 131-140, 6% have 141-150, 5% have 151-160, 7% have 167-170, 40% have 171-180, 3% have 181-190, 3% have 191-200, and 2% have above 200.

4.16: Distribution of MI among Hypertensive & Non Hypertensive Patients:

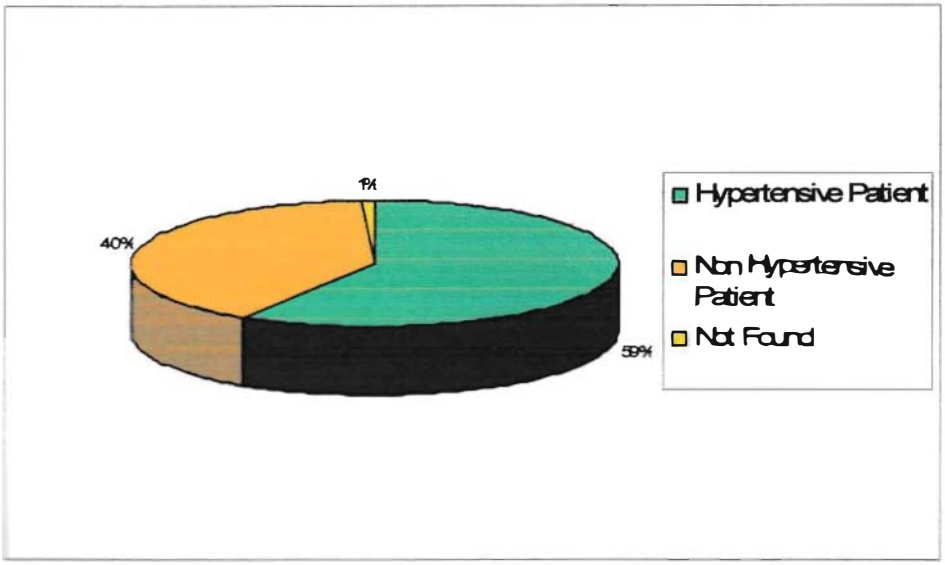

Figure 24: Distribution of MI among Hypertensive & Non Hypertensive Patients.

This figure shows that 59% patients are suffering from Hypertension, 40% are non hypertensive patient & 1% patient doesn't have relevant data.

4.17 Distribution of Diabetes among MI patients:

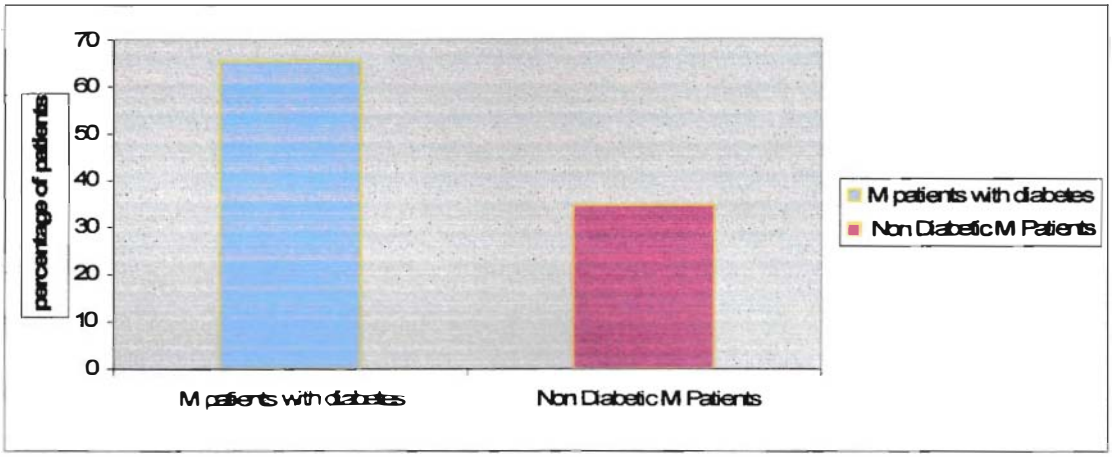

Fig 25: Distribution of Diabetes among MI patients.

This Figure shows that 65.19% MI patients are suffering from Diabetes whereas 34.82% MI patients are not suffering from Diabetes.

CHAPTER 5

DISCUSSION & CONCLUSION

The world is faced with the emergence of cardiovascular disease as a major health problem. Neither the solution nor the prevention of the disease will be achieved by any single organization or simple policy but by: 1. coordination and unification of effort at a political level. 2. The creation of demonstrably effective health systems 3. The activities and efforts of health workers – nurses, managers, physicians, cardiovascular specialists and cardiologists. **(Philip Poole-Wilson, July/August 2005)**

For individual with diabetic mellitus, CHD risk is greatly increased with the both type I and type II. Over 70% of patients with diabetes die from macro vascular disease, mainly coronary heart disease. Thus, implicit in the long term management of diabetic patients is the requirement for multiple risk factor modification for coronary prevention. Hypertension is very common in type II diabetes. is strongly related to obesity and highly protective of cardiovascular complications. Many patients with type II diabetes are overweight and have high cardiovascular risk. They need intensive and sustained advise in life style and appropriate treatment to achieve other risk factor targets as well as glycaemic control **(David Wood, 2001).**

Diabetic subjects are more likely to experience a myocardial infarction and have worse outcomes compared to non-diabetic subjects. The underlying pathophysiology of the atherosclerotic process is not significantly different in diabetic subjects, but the prothrombotic and procoagulant state with which diabetes is associated is thought to contribute to the higher incidence of and worse prognosis after myocardial infarction **(Ian L Williams, 2003).**

The prevalence of diabetes mellitus among patients with acute myocardial infarction in a geographically defined population in the developing world is high with a trend for poor outcomes. However, mortality was not significantly higher in diabetes mellitus than non-diabetes mellitus patients **(Ayman El-Menyar, 2008).**

Our study was a population based studies where data was collected on Myocardial Infraction over a period of 1 year. Data was collected from the patients of National institute of Cardio vascular disease, there was no age limit, only the patients of MI were

chosen and cardiology department coordinated their efforts. Using data from this study, we demonstrated that Myocardial Infraction was more frequent in hypertensive patients than non-hypertensive patients.

The result of the study showed that patients of age range 46 to 55 years were more prone to Myocardial Infraction. In the study the ratio of male & female MI patient is 96: 4. 91.11% Muslim and 8.15% Hindu & 0.74% Christian patients were with Myocardial Infraction, here 47% people were attached by AMI and 44% people with OMI. In our study we found that 73.33% MI patient are smoker and 28.88% patient are smoking for 11-20 years and 11.86% people are smoking for 26-30 years. 67% of the total population takes table salt with their meal. Betel nuts were taken by 43.7% of patient. The distribution of MI in different age range were collected where 36-55 years people are more in danger of suffering from MI. The percentage was 58.51%. In our study we also found 28.15% of total population was suffering from only hypertension and diabetes. The result of the study suggested that myocardial infraction is more prominent in patients with diabetic mellitus and hypertension rather in patients with other risk factors.

Cardiovascular disease (CVD) accounts for more than 75% of total mortality among patients with type 2 diabetes. Several recent studies have compared the magnitude of risk associated with a history of type-2 diabetes with a history of MI on subsequent CHD and CVD mortality. One Finnish prospective study, for example, found that the risk of CHD death in patients with diabetes but no previous MI was similar to that of nondiabetic subjects who had had an MI, but the finding was challenged in several later studies.**(Susan Jeffrey, May 10, 2005)**

The main objective of the study was to find out the relationship between MI and diabetes. In this socio demographic study it was found that age, educational status, occupation were significantly associated with coronary heart disease. Among life style risk factors smoking, Length of smoking, betel nut chewing, and tobacco consumption all were significantly associated with development of MI.

Hypertension and CHD shows a significant relation with MI. Type of occupation also observed in this study. Elevated blood pressure and blood sugar level also an indicator of development of MI.

The finding reports here, for myocardial infarction, are consistent with there of other investigations and yet definite. A significant association of diabetes with myocardial infarction was noted. From the result it was obtained that among 60 patients 29% patients with diabetic mellitus had myocardial infarction.

Several fixed and modifiable life style factors which include smoking habit, social class, table salt intake etc has effect on MI.

Including the above-mentioned factors the study has been designed to explore the relationship between diabetes and MI.

This study will help to increase the awareness among people health by taking immediate treatment, by taking drug, or by controlling blood sugar level, food habit, and physical activity to avoid the harmful effect of myocardial infarction.

Prevention targeted at patients with established coronary disease and the high risk strategy targeted at healthy individuals at high risk are an integral part of clinical practice. The clinical approaches and the population approaches for coronary heart disease prevention are complimentary, but the population strategy is fundamental to reduce the burden of cardiovascular disease. **(David Wood, 2001)**

The outcome of this study may provide important information for future in depth study as well as may help in providing ideas for policy maker to formulate proper investigation strategies among the people regarding diabetes and risk factors responsible for development of cardiovascular diseases.

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ANNEXURE

NATIONAL INSTITUTE OF CARDIOVASCULAR DISEASES

HOSPITAL

(NICVD) DHAKA .

Questionnaire

1. Name of the respondent

.....

2. Address:.....

3.

Mobile.....

..

Block No.	House No.	Village/word	Thana	District

3. Sex: 1 = Male

2= Female

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

5. What is your religion?

1 = Islam

2 = Hinduism

3 = Christianity

4=Buddhism

5. Others (Specify).....

6. How far have you studied ?

1 = Illiterate 5

2 = Non-formal education 6

3 = Class I-V

4 = Class VI-IX

6.HSC or its equivalent

7.Graduate +

8.Others (Specify).....

7. What is your marital status?

1 = Married and spouse live together

2 = Spouse live separately

3 = Widow

4.= Divorced

5. = Never married

6 = Widower

8. What is your occupation?

1 = Farmer

2 = Businessman

5. = Unemployed

6. = House Wife

3 = Service 7. = Others (specify).....

4 = Pension holder7

9. How many members are there in your family? [ ]

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

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

1. Yes 2. No

12. If yes, how much (Taka)? [ ]

13. Did you ever smoke any time in your life?

1 = Yes 2 = No (go to no, 19)

14. Do you currently smoke?

1 = Yes 2 = No (go to no. 19)

15. How much do you smoke?

1 = Everyday 2 = occasionally (go to no. 19

16. What type of cigar do you smoke?

1 = Cigarette 2 = Biri

3 = Cigarette & Biri both 4 = others (specify).....

17. For how long you are smoking daily?

Year ..... Month...

18. Do you currently chew betel nut?

1 = Yes 2= ' No (go to no.23)

19. How much do you chew betel nut?

1 = Everyday 2 = occasionally (go to no. 23)

20. Do you use smokeless tobacco with betel?

1 = Yes 2 = No (go to no. 23)

21. What do you take with betel?

1 = Tobacco leaf 2 = Jorda (preparation of tobacco leaf) 3 = both

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

Year ..... Month...

23.Do you take excess tea or coffee?

1 = Yes 2. = No

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

1 = Yes 2 = No

25. Do you currently drink alcohol?

1 = Yes 2 = No

26. For how long do you drink alcohol?

Year ..... Month...

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

1. Soybean oil 5.Sunflower oil
2 .Mustard oil 6.Corn oil
3. Sesame oil 7.GheefButter 8.Other (specify) .....
4. Pulm oil

28. Do you take table salt?

1 Yes 2 = No

29. Did you come here with the following complaints?

1 = Yes 2 = No 3. Don't know

1. Chest pain,
2. left arm pain
3. right arm pain,
4. jaw pain
5. neck pain
6. back pain
7. shortness of breath
8. heart burn
9. pulmonary edema
10. nausea, vomiting,
11. loss of conciousness
12. sweating
13. Other (specify).....

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

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

1. Hypertension
2. Diabetes Mellitus

3. Asthma

4. Coronary heart disease

31. If yes-

What is the relation between you & them?

1 = Father 3 =parent

2= Mother 4 =siblings

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

1 = Yes 2 = No 3 = Don't know

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

33. for female only-

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

1 = Yes 2 = No

34. If yes-

Do you take pills now?

1 = Yes 2 = No

36. If yes

For how long are you taking? Year ..... Month...

37. Currently use drugs:

(1) ACE Inhibitor: Benzepril, Captopril, Enalaprl, Fosinopril, Lisinopril, Moexipril, Quinapril, Ramipril
(2) β blocker: Atenolol, Labetalol, Metoprolol, Nadolol, propranolol.
3) Diuretics: Bumetanoid, Furosemide, Hydrochlorothiazide, Spironolactone, Triamterene.

(4) Antiplatelets: Aspirin, Abciximab, Clopidogrel, Dipyridamole, Eptifibatide, Ticlopidine, Tirofiban.
(5) Anticoagulents: Danaporoid, Enoxaprin, Heparin, Lepirudin, Warfarin.
(6) Organic Nitrate: Isosorbide di nitrate, Isosorbide mononitrate, Nitroglycerin.
(7)Lipid lowering agent: Atorvastatin, Fluvastatin, Lovastatin, Pravastatin, Rosuvastatin Calcium,Simvastatin, Cholestyramine (Questran®, Questran® Light, Prevalite®, Locholest®, Locholest® Light), Colestipol, Colesevelam Hcl .
(8) Ca^+ Channel Blocker: Amlodipine, Diltiazem, Felodipine, Nicardipine, Nitredipine, Nifedipine, Verapamil.
(9) Na^+ Channel Blocker: Disopyramide, Flecainide, Lidocaine, Mexiletine, Procainamide, Propafenone, Quinidine, Tocainide.
(10) k^+ Channel Blocker: Amiodarone, Dofetilide, Sotalol.
(11) Aldosteron Antagonist agent: Eplerenone.
(12) Angiotensin Receptor Antagonist: Losartan.
(13) Other Drugs: Omega-3 fatty acid, Adenosine, Digoxin, Benzodiazepines.

38. Do you know your blood group?

1= Yes, I know 2= No, I don't know

If Yes,

1 = A 2 = B

3 = O 4=AB

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

1=Yes. 2=No.

From record

1. Height (cm)
2. Weight (kg)
3. Waist girth (cm)
4. Hip girth (cm)
5. Systolic Blood Pressure (mm/Hg).....
6. Diastolic Blood Pressure (mm/Hg) .....
7. Blood group.....
8. S. Lipid Profile.....

Total Cholesterol..... mg/dl. .....

Triglyceride .....mg/dl.....

HDL-Cholesterol mg/dl .

LDL-Cholesterol..... mg/dl.....

9. Blood urea.....

10. Serum electrolyte. .....

l=Na..... m.eq/L .....

11.2=K .....m.eq/L . .....

12. SGPT.....

13. SGOT.....

14. CKMB.....

15. Troponine-1

16. RBS

17. Echocardiography.....

18. X-ray chest.....

20. ECG Report

1= Yes, 2= No

Site of MI ,

1. Anterior MI
2. Extensive anterior

Prescription:

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