Hypertension with history of smoking: Effect on Myocardial Infarction

A thesis project submitted to the department of pharmacy, East West University, Bangladesh, in partial fulfillment of the requirements for the degree of Bachelor of Pharmacy.

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Submission Date: JUNE, 2010

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ACKNOWLEDGEMENTS

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

like to express heart-felt gratitude to my supervisor Mrs. Nishat Nasrin, Lecturer, Pharmacy, East West University (EWU). She has helped me a lot by giving me her time and directed me properly through a guideline to clear my confusion about my

am grateful to co- supervisor Mrs. Momena Shirin M. Phil (PSM). superintendent, Institute of Public Health, Mohakhali, Dhaka for her guidance, criticism and during the preparation of this study. Dr Sufia Islam (Associate Professor, Pharmacy, EWU), and Mrs Farhana Rizwan and Mr. Muhammad Assaduzzaman Lecturer, Dept. of Pharmacy, EWU) also helped me a lot through their invaluable gratefully acknowledge to Pro-vice chancellor Professor Muniruddin Ahmed Professor Dr. Chowdhury Faiz Hossain for their inspirations in my study.

sincere admiration to my caring parents for guiding me all through my life, my research project. I am very grateful to my brother, sisters and friends, me enormously. Finally, I am thankful to all of my friends who were research enthusiastically.

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List of Abbreviation

ACC: American College of Cardiology

AHA: American Heart Association

AMI: Acute Myocardial Infarction

BMI: Body Mass Index

CCU: Coronary Care Unit

CHD: Coronary Heart Disease

CHF: Congestive Heart Failure

CK: Creatinine Kinase

CKD: Chronic Kidney Disease

CPK: Creatinine Phospho Kinase

CT: Computed Tomography

CVD: Cardiovascular Disease

ECG: Electrocardiogram

ESC: European Society of Cardiology

ETT: Exercise Tolerance Testing

GI: Gastro intestinal

HDL: High Density Lipoprotein

HHANES: Hispanic Health and Nutrition Examination Survey

HR: Heart Rate

LDL: Low Density Lipoprotein

LV: Left Ventricle

MI: Myocardial Infarction

MRI: Magnetic Resonance Imaging

NCHS: National Center for Health Statistics

NHES: National Health Examination Survey

NHIS: National Health Interview Survey

NICVD: National Institute of Cardiovascular disease

OMI: Old Myocardial Infarction

PAD: Peripheral artery disease

SCD: Sudden Cardiac Disease

SHS: Second Hand Smoking

WHO: World Health Organization

Abstract

Myocardial infarction (MI) is the rapid development of myocardial necrosis caused by a [illegible] imbalance between oxygen supply and demand of the myocardium. This usually [illegible] from plaque rupture with thrombus formation in a coronary vessel, resulting in an [illegible] reduction of blood supply to a portion of the myocardium. There are several risk factors [illegible] among them, hypertension and smoking is the most important. To find out the [illegible]ship between smoking habits and hypertension with myocardial infarction the present [illegible] was conducted incorporating 135 MI patients from the National Institute of [illegible]vascular Disease (NICVD). In this survey type study data were collected using a [illegible]naire which includes question regarding name, address, sex, age, education, marital [illegible] occupation, food habit, smoking, alcohol, betel nut, cooking oil, table salt etc. The [illegible] risk was assessed for smokers, nonsmokers (persons with no history of smoking), [illegible] smokers (persons who had stopped smoking before infarction) and hypertensive [illegible] All data were evaluated and compared with each patient. The results of the study [illegible] that 96% patients were male and only 4% patient were female. Among the patients, [illegible] people were attacked by AMI and 44% with OMI. The distribution of MI in different [illegible] showed that from 36-55 years, people are more prone to suffering from MI. It was [illegible] that 59% of total population was suffering from hypertension and 73.3% [illegible] had smoking habit. The results of the study suggested that myocardial infarction is [illegible] prominent in hypertensive MI patients with the history of smoking. MI is a common [illegible] disorder where smoking and hypertension is giving synergistic effect on patients. Since [illegible] and hypertension is modifiable risk factor, a person can remain safe from this [illegible] by changing their lifestyle. Physical activity and nutritious food are essential for [illegible]. Moreover, smoking should be abandoned and blood pressure should be in [illegible]

CHAPTER 1

Introduction

Overview

Myocardial infarction (MI) occurs when blood vessels that supply blood to the heart are [illegible] preventing enough oxygen from getting to the heart. The heart muscle dies or [illegible] permanently damaged. It is a serious result of coronary artery disease. Coronary [illegible]-ease occurs from atherosclerosis, when arteries become narrow or hardened due to [illegible] plaque build-up. Further narrowing may occur from thrombi (blood clots) that [illegible] surfaces of plaques.

The most common cause of MI is narrowing of the epicardial blood vessels due to [illegible] plaques. Plaques rupture with subsequent exposure of the basement membrane [illegible] in platelet aggregation, thrombus formation, fibrin accumulation, hemorrhage into [illegible] and varying degrees of vasospasm. This can result in partial or complete [illegible] the vessel and subsequent myocardial ischemia. The risk factors that have been [illegible] with a higher incidence of myocardial infarction are age, gender, family [illegible] smoking, high blood pressure (hypertension), high blood cholesterol, obesity, [illegible] lack of physical activity, stress etc. Some of these risk factors are controllable [illegible] smoking and physical activity) while others are uncontrollable (such as age, [illegible] and family history).

The most common symptom of myocardial infarction is angina (chest pain). Other [illegible] myocardial infarction may include sweating, jaw pain, heartburn or indigestion, [illegible] pain, general feeling of illness. The pathophysiology of acute myocardial [illegible] complex. Loss of viable myocardium impairs cardiac function, which can lead to [illegible] cardiac output, and if damage is severe, to cardiogenic shock. Systolic and diastolic [illegible] are associated with ischemic myocardium. If left ventricular function is [illegible] impaired, pulmonary congestion and edema can occur. Ischemia can also [illegible] abnormal cardiac rhythms and conduction blocks that can further impair function [illegible] life-threatening in some cases.

[illegible] complications of acute M.I. are directly related to the coronary artery blood supply. [illegible] complications include arrhythmias and heart block, and hypotension, cardiogenic [illegible] cardial rupture, pericarditis, ventricular aneurism & congestive heart failure.

[illegible] number of laboratory biomarkers for myocardial injury are available. None is [illegible] sensitive and specific for myocardial infarction, particularly in the hours [illegible] of symptoms. The following biomarkers have been described in association

acute myocardial infarction: blood tests, echocardiogram, exercise stress testing, [illegible] procedures, and magnetic resonance imaging (MRI).

Tobacco use is the most common cause of avoidable cardiovascular mortality worldwide. The immediate noxious effects of smoking are related to sympathetic nervous activity, which increases myocardial oxygen consumption through a rise in blood pressure, heart rate, and myocardial contractility. Chronically, cigarette smoking induces arterial stiffness which may persist for a decade after smoking cessation. Besides, smoking should be avoided in any hypertensive patient because it can markedly increase the risk of cardiovascular complications like myocardial infarction.

This study was accomplished on myocardial infarction patients to find out the relation smoking and hypertension with them. The result of this study is consistent with the other studies regarding to myocardial infarction. This study will be help to increase the awareness of people with myocardial infarction.

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1.2. Communicable diseases

Communicable diseases are one of the categories where illnesses caused by microorganisms and transmitted from an infected person or animal to another person or animal **(Communicable diseases, n.d)**. Some diseases are passed on by direct or indirect contact with infected persons or with their excretions. Most diseases are spread through contact or close proximity because the causative bacteria or viruses are airborne; i.e., they can be expelled from the nose and mouth of the infected person and inhaled by anyone in the vicinity. Such diseases include diphtheria, scarlet fever, measles, mumps, whooping cough, influenza, and smallpox. Some infectious diseases can be spread only indirectly, usually through contaminated food or water, e.g., typhoid, cholera, dysentery. Still other infections are introduced into the body by animal or insect carriers, e.g., rabies, malaria, encephalitis, Rocky Mountain spotted fever. The human disease carriers, i.e., the healthy persons who may be immune to the organisms they harbor, are also a source of transmission. Some infective organisms require specific circumstances for their transmission, e.g., sexual contact in syphilis and gonorrhea, injury in the presence of infected soil or dirt in tetanus, infected transfusion blood or medical instruments in serum hepatitis and sometimes in malaria. In the case of AIDS, while a number of different circumstances will transmit the disease, each requires the introduction of a contaminant into the bloodstream. A disease such as tuberculosis may be transmitted in several ways-by contact (human or animal), through food or eating utensils, and by the air. Control of communicable disease depends upon recognition of the many ways transmission takes place. It must include isolation or even quarantine of persons with certain diseases. Proper antisepsis should be observed in illness and in health. Immunologic measures should be utilized fully. Some sexually transmitted infections are associated with cancer (cervical or penile). Education of the population in rules of public health is of great importance both in the matter of personal responsibility (disposal of secretions, preventing contact with the blood of others, proper handling and preparation of food, personal hygiene) and community responsibility (safe water and food supply, sterile blood supply, garbage and waste disposal). Animal and insect carriers must be controlled, and the activities of human carriers must be limited.

1.3. Noncommunicable diseases

Noncommunicable diseases are usually thought of as chronic conditions that do not result from an acute infectious process **(Harlan W and Harlan L, 2002)**. These conditions cause

death, dysfunction, or impairment in the quality of life, and they usually develop over relatively long periods—at first without causing symptoms; but after disease manifestations develop, there may be a protracted period of impaired health. Generally, these conditions or diseases result from prolonged exposure to causative agents, many associated with personal behaviors and environmental factors. Noncommunicable diseases also include injuries, which have an acute onset, but may be followed by prolonged convalescence and impaired function, as well as chronic mental diseases.

Noncommunicable diseases are the leading cause of functionary impairment and death worldwide. These conditions have been the leading cause of death in the United States and other high-income countries over the last fifty years, and they are emerging as a leading cause of death in low-to middle-income countries.

1.4. Cardiovascular disease

Cardiovascular disease is the class of diseases that involve the heart or blood vessels. While the term technically refers to any disease that affects the cardiovascular system, it is usually used to refer to those related to atherosclerosis. These conditions have similar causes, mechanisms, and treatments. Heart disease is the leading cause of death overall; stroke is the third leading cause.

1.5. Types of CVD: CVD can be classified into 2 types.

1.5.1. Congenital heart disease:

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

Congenital heart disease is the most common type of birth defect, with six out of every 1,000 babies being born with the condition **(Congenital heart disease, n.d.)**. Half of all babies born with congenital heart disease will require immediate surgery after birth, while the other 50% will probably require surgery or medication at some point during their childhood. Congenital heart disease can sometimes develop as the result of certain genetic

conditions, such as Down's syndrome. An infection during pregnancy, such as rubella, can also cause congenital heart disease.

As of 2002, the prevalence of congenital cardiovascular defects in the United States varied between 4 and 10 per 1000 live births and was estimated to range from 650 000 to 1.3 million people (**Moller JH, 2004**). From 1940 to 2002, about 2 million patients with congenital cardiovascular defects were born in the United States. Of these, several studies suggest that 9,200, or 2.3 per 1,000 live births, require invasive treatment or result in death in the first year of life. Some studies suggest that as many as 5 percent of newborns, or 200,000 per year, are born with tiny muscular ventricular septal defects, almost all of which close spontaneously (**Roguin N, 1995**).

1.5.2. Acquired heart disease:

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

1.5.2.1. Types of acquired heart disease:

The four main types of acquired heart disorder are:

Kawasaki disease: an illness that occurs mainly in young children and may leave the heart muscle or coronary arteries damaged.

Myocarditis: the heart muscle becomes inflamed and may be damaged after a viral infection.

Cardiomyopathy: a disease of the heart muscle, caused by a genetic disorder or after an infection. It leads to poor heart function.

Rheumatic heart disease: caused by rheumatic fever, this disease leads to heart muscle and valve damage (**Heart disorders acquired in children, n.d.**).

Cardiovascular Disease (CVD) includes dysfunctional conditions of the heart, arteries and veins that supply oxygen to vital areas of the body like the brain, the heart itself, and other organs. If oxygen doesn't arrive the tissue or organ will die. Ischemic Heart Disease is the technical term for obstruction of blood flow to the heart. In general this results because excess fat or plaque deposits are narrowing the veins that supply oxygenated blood to the heart. Excess buildups of fat or plaque are respectively termed arteriosclerosis and

atherosclerosis. Equally significant would be inadequate oxygen flow to the brain, which causes a stroke.

High Blood Pressure (hypertension) often results from this excess fat or plaque buildup because of the extra effort it takes to circulate blood. Even though the heart works harder, blockages still shortchange the needed blood supply to all areas of the body. The body's amazing survival systems will mask the subtle damage that is occurring from this extra wear and tear, but not forever. High blood pressure is called "The Silent Killer" because the first warning sign is an angina attack or a deadly heart attack or a stroke (**Medicinenet, 2003**).

Kidney disorders (which leave extra fluids, sodium, and toxins in the body), obesity, diabetes, birth control pills, pregnancy, smoking, excess alcohol, stress, and thyroid and adrenal gland problems can also cause and exacerbate a high blood pressure condition.

Damage to the heart tissues from CVD or from heart surgery will disrupt the natural electrical impulses of the heart and result in cardiac arrhythmia (an abnormally high or abnormally low heart rate). Individuals often don't realize the aftermath and side effects that invasive surgical procedures leave. Sudden fluctuations in heart rate can cause noticeable palpitations, with an associated faintness, or dizziness, and if severely abnormal could interfere with blood flow and even initiate a heart attack. Proper ranges of cholesterol are also important in the prevention of heart attack or stroke. Total blood cholesterol above 200 mg/dl, LDL cholesterol above 130 mg/dl, HDL cholesterol below 35 mg/dl; and lipoprotein (a) level greater than 30 mg/dl are indicators of problematic cholesterol. Cholesterol is not actually a damage mechanism but is more an indicator of compromised liver function, and increased risk of heart attack **(Cardiovascular Disease Facts and Statistics, n.d.)**.

1.6. Myocardial infarction:

A myocardial infarction (also known as a heart attack) is the death of heart muscle from the sudden blockage of a coronary artery by a blood clot. Coronary arteries are blood vessels that supply the heart muscle with blood and oxygen. Blockage of a coronary artery deprives the heart muscle of blood and oxygen causing injury to the heart muscle. Injury to the heart muscle causes chest pain and chest pressure sensation. If blood flow is not restored to the heart muscle within 20 to 40 minutes, irreversible death of the heart muscle will begin to occur. Muscle continues to die for six to eight hours at which time the heart attack usually is "complete." The dead heart muscle is eventually replaced by scar tissue.

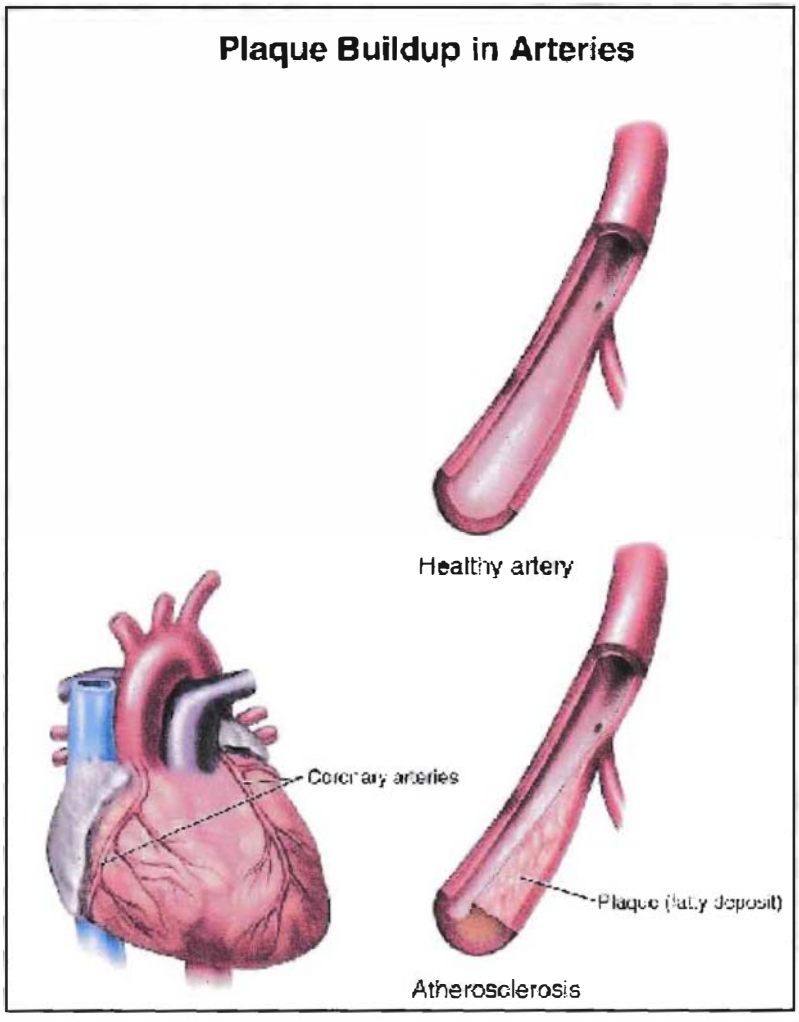

Fig 1.1: Myocardial infarction **(Summit medical group, 2009).**

1.6.1. New Definition for Myocardial Infarction:

Representatives of four major worldwide organizations dedicated to treating heart disease (the American College of Cardiology, the American Heart Association, the European Society of Cardiology, and the World Heart Federation), have agreed to new criteria for diagnosing myocardial infarctions. A myocardial infarction occurs when the blood supply to part of the heart muscle is blocked, usually by the sudden formation of a blood clot within a coronary artery, leading to death of part of the heart muscle. Classically, a "heart attack" is diagnosed when any two of these three things occur: typical symptoms (chest pain, shortness of breath), typical changes on the electrocardiogram (ECG), and elevation in the blood of certain biomarkers (usually, the enzyme known as MB-CK) that are released by damaged heart cells. The change in the new definition of myocardial infarction is that the preferred biomarker is troponin. Elevations in troponin levels above the 99th percentile of the upper reference limit,

when accompanied by either symptoms compatible with heart attack or by ECG changes, will now clinch the diagnosis **(Fogoros NR, 2007)**.

Troponin is far more specific for heart cell damage than older biomarkers, and any elevation in troponin levels is considered to indicate heart muscle damage. Troponin is also more "sensitive;" it is released earlier and more reliably than older biomarkers.

1.6.2. Classification: There are two basic types of acute myocardial infarction:

Transmural: associated with atherosclerosis involving major coronary artery. It can be subclassified into anterior, posterior, or inferior. Transmural infarcts extend through the whole thickness of the heart muscle and are usually a result of complete occlusion of the area's blood supply.

Subendocardial: involves small area in the subendocardial wall of the left ventricle, ventricular septum, or papillary muscles. Subendocardial infarcts are thought to be a result of locally decreased blood supply, possibly from a narrowing of the coronary arteries. The subendocardial area is farthest from the heart's blood supply and is more susceptible to this type of pathology **(Morphology of acute myocardial infarction, n.d.)**.

Clinically, myocardial infarction is further sub classified into ST elevation MI versus non ST elevation MI based on ECG changes

1.7. Causes of MI:

The most common cause of MI is narrowing of the epicardial blood vessels due to atheromatous plaques. Plaques rupture with subsequent exposure of the basement membrane that results in platelet aggregation, thrombus formation, fibrin accumulation, hemorrhage into the plaque, & varying degrees of vasospasm. This can result in partial or complete occlusion of the vessel & subsequent myocardial ischemia. Total occlusion of the vessel for more than 4-6 hours results in irreversible myocardial necrosis, but reperfusion within this period can salvage the myocardium and reduce morbidity and mortality.

Nonatherosclerotic causes of MI include coronary vasospasm as seen in variant angina and in patients using cocaine and amphetamines; coronary emboli from sources such as an infected heart valve; occlusion of the coronaries due to vasculitis; or other causes leading to mismatch of oxygen supply and demand, such as acute anemia from GI bleeding. MI induced by chest trauma has also been reported, usually following severe chest trauma such as motor vehicle accidents and sports injuries (**Medicinenet 2003**).

1.8. Symptoms of Myocardial Infarction:

Classical symptoms of acute myocardial infarction include sudden chest pain (typically radiating to the left arm or left side of the neck), shortness of breath, nausea, vomiting, palpitations, sweating, and anxiety (often described as a sense of impending doom). Women may experience fewer typical symptoms than men, most commonly shortness of breath, weakness, a feeling of indigestion, and fatigue. Approximately one quarter of all myocardial infarctions is silent, without chest pain or other symptoms (**Mallinson T, 2010**).

Pain: This is severe, usually retrosternal and may radiate into the jaw, shoulders and down the arms. It is described as a tight band around the chest and lasts for several hours.

Dyspnoea: This is due to either the pain or pulmonary congestion caused by pulmonary hypertension and pulmonary edema. There is also an increase in the myocardial oxygen demand. If the left ventricle is affected, the cardiac output will be reduced and a shock state may exist.

Extreme pallor: This is due to the decreased cardiac output and redirection of blood away from the skin to the major organs. The skin will also feel cool and clammy.

Nausea and vomiting: This is due to pain, redirection of blood away from the gastrointestinal system, release of adrenaline and other catecholamines into the blood and from side effects of medications used to treat the condition and symptoms.

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

Tachyrhythmias: This is due to the heart trying to compensate for the low cardiac output by increasing the rate (heart rate x stroke volume = cardiac output). The myocardium also becomes very irritable following infarction due to release of metabolites and electrolytes such as potassium and calcium from dying cells. This results in ventricular arrhythmias.

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

Pyrexia: The patient's temperature rises to around 39°C due to release of metabolites during the inflammatory process initiated by the necrotic tissue and widespread death of cells. This normally occurs over 24 – 48 hours and returns to normal within 7 days.

Sense of Impending Doom: This is due to the release of adrenaline and other catecholamines as part of the compensation mechanism. Also the real fear of death exists due to the nature of the disease and the information known by the general public. It may also be that the patient is normally an anxious / highly-strung individual who worries a lot.

1.9. Risk factors:

There are many risk factors associated with coronary heart disease and stroke. The major risk factors, tobacco use, alcohol use, high blood pressure (hypertension), high cholesterol, obesity, physical inactivity, unhealthy diets, have a high prevalence across the world. Of particular significance in developing countries is the fact that while they are grappling with increasing rates of cardiovascular disease, they still face the scourges of poor nutrition and infectious disease.

1.9.1. Nonmodifiable risk factors

Increasing age: Over 83 percent of people who die of coronary heart disease are 65 or older. At older ages, women who have heart attacks are more likely than men are to die from them within a few weeks **(Risk Factors and Coronary Heart Disease, n.d.)**. With the aging of human the efficiency of heart decreases. The heart's walls may thicken and arteries may stiffen and harden, making the heart less able to pump blood to the muscles of the body. Because of these changes, the risk of developing cardiovascular disease increases with age. Because of sex hormones, women are usually protected from heart disease until menopause, and then their risk increases. Women with 65 and older have about the same risk of cardiovascular disease as men of the same age **(Heart Disease Risk Factors, 2009)**.

Compared with non-SCD (Sudden cardiac disease) the risk of SCD is relatively highest in the younger age groups, but the absolute risk is much higher among the upper age groups than the younger. The risk of SCD was slightly lower in women but not enough to warrant a different treatment strategy **(Abildstrom SZ, *et al,* 2002).**

Increased age is associated with a greater proportion of patients with functional limitations, heart failure, prior coronary disease and renal insufficiency and a lower proportion of male and diabetic patients. The proportion of patients presenting with chest pain within 6 h of symptom onset, and with ST-segment elevation, was lower in each successive age group. The effect of age persisted but was attenuated after adjustment for differences in patient characteristics; similar trends were observed for one-year mortality **(Mehta RH, *et al,* 2001)**.

Gender: Males are at higher risk of myocardial infarction than women, and males are also more likely to suffer myocardial infarction earlier in life. However, heart disease kills more females each year than any other disease, including breast cancer. An alarming survey reported by the American Heart Association found that only 8% of women perceive heart

disease as the greatest threat to their health despite the fact that heart disease is the leading cause of death among both women and men (**Risk factors for Myocardial Infarction or Heart Attack, 2008).**

Women have a well-documented higher mortality after acute myocardial infarction. Much of this disparity has been attributed to differences in age and attendant co-morbidities. Female patients with coronary artery disease typically are older, have a higher prevalence of risk factors, and have a lower functional status than their male counterparts **(Anderson RD, *et al,* 2007).**

Heredity (including **Race**): Heart disease tends to run in families. For example, if parents or siblings had a heart or circulatory problem before age 55, then you are at greater risk for heart disease than someone who does not have that family history. Risk factors (including high blood pressure, diabetes, and obesity) may also be passed from one generation to another **(Heart Disease Risk Factors, 2009)**.

A much greater incidence of myocardial infarction was noted when one or more among parents and sibling had a history of hypertension, apoplexy, ischemic heart disease or sudden death than when neither parents nor sibling suffered from these diseases **(Noboru Y, 1973)**. Children of parents with heart disease are more likely to develop it themselves. African Americans have more severe high blood pressure than Caucasians and a higher risk of heart disease. Heart disease risk is also higher among Mexican Americans, American Indians, native Hawaiians and some Asian Americans. This is partly due to higher rates of obesity and diabetes. Studies looking at the incidence of pulmonary embolism in various races show that African American patients are the highest risk group, with a 50% higher incidence than American whites. Asian/Pacific Islanders/American Indian patients have a markedly lower risk of thromboembolism **(Sutherland SF, 2009)**.

Most people with a strong family history of heart disease have one or more other risk factors. Just as we can't control age, sex and race, can't control family history. Therefore, it's even more important to treat and control any other risk factors you have (**Risk Factors and Coronary Heart Disease, n.d.)**.

1.9.2. Modifiable risk factors

Diabetes: One of four patients with acute myocardial infarction had diabetes mellitus (**Tenerz Å, *et al*, 2000**). Diabetes seriously increases your risk of developing cardiovascular disease. Even when glucose levels are under control, diabetes increases the risk of heart disease and stroke, but the risks are even greater if blood sugar is not well controlled. About three-quarters of people with diabetes die of some form of heart or blood vessel disease.
During MI, hyperglycemia is associated with increased levels of inflammatory markers, enhanced expression of cytotoxic T-cells, and reduced expression of T-cells, which are implicated in limiting the immune process. An increased inflammatory immune process seems a likely mechanism linking acute hyperglycemia to poor cardiac outcome in MI patients **(Marfella R, *et al*, 2003).**

High blood pressure: High blood pressure increases the heart's workload, causing the heart to thicken & become stiffer. It also increases the risk of stroke, heart attack, kidney failure & congestive heart failure. If high blood pressure exists with obesity, smoking, high blood cholesterol levels or diabetes, then the risk of heart attack or stroke increases several times. Mechanical stress on blood vessels because of high blood pressure is an especially important factor in endothelial dysfunction, the progression of atherosclerosis, and plaque rupture. **(Rakugu H, 1995).**

According to the definitions used hypertension had been detected before or was detected after myocardial infarction in 36% of the patients. Two thirds had a history of hypertension known before infarction **(Wilhelmsson C, *et al*, 1978).**

Smoking: Smokers' risk of developing coronary heart disease is 2–4 times that of nonsmokers. Cigarette smoking is a powerful independent risk factor for sudden cardiac death in patients with coronary heart disease; smokers have about twice the risk of nonsmokers. Cigarette smoking also acts with other risk factors to greatly increase the risk for coronary heart disease. After incident myocardial infarction, smoking was associated with an elevated risk for recurrent coronary events. In persons who quit smoking after infarction, the risk declined to equal that of nonsmokers by 3 years after cessation (**Thomas D R, *et al,* 2002**).

People who smoke cigars or pipes seem to have a higher risk of death from coronary heart disease (and possibly stroke) but their risk isn't as great as cigarette smokers'. Exposure to other people's smoke increases the risk of heart disease even for nonsmokers. 96% of the

patients with premature MI and 55% of the controls reported current smoking habits ($p<0.001$) **(Panagiotakosa DB, *et al,* 2006)**. Tobacco use is one of the most important causes of AMI globally, especially in men. All forms of tobacco use, including different types of smoking and chewing tobacco and inhalation of SHS, should be discouraged to prevent cardiovascular diseases **(Teo KK, *et al,* 2006)**.

Blood Cholesterol: As blood cholesterol rises, so does risk of coronary heart disease. When other risk factors (such as high blood pressure and tobacco smoke) are present, this risk increases even more. A person's cholesterol level is also affected by age, sex, heredity and diet. Unhealthy cholesterol levels, especially high LDL cholesterol and low HDL cholesterol as effect on heart disease **(Risk Factors and Coronary Heart Disease, n.d.)**.
Patients who have recovered from a myocardial infarction and who have high cholesterol levels are at an increased long-term risk for reinfarction, death from coronary heart disease, and all-cause mortality **(Wong N D, *et al,* 1991)**.

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 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. In this population-based study, overweight and obese status is independently associated with the premature occurrence of AMI, but not with an increased incidence of in-hospital complications **(Suwaidi J A, *et al,* 2001)**.

The overweight category is associated with increased relative and population is attributable risk for hypertension and cardiovascular sequelae. Interventions to reduce adiposity and avoid excess weight may have large effects on the development of risk factors and cardiovascular disease at an individual and population level **(Peter W F. *et al,* 2002)**.

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 **(Risk factors for Myocardial Infarction or Heart Attack, 2008)**. Patients with acute

myocardial infarction report a higher subjective mental stress during 2 to 4 weeks preceding the acute coronary event **(Chockalingam A, *et al,* 2003)**.

The presence of mental stress—induced ischemia is associated with significantly higher rates of subsequent fatal and nonfatal cardiac events, independent of age and previous myocardial infarction, and predicted events over and above exercise-induced ischemia. These data suggest that the relationship between psychological stress and adverse cardiac events may be mediated by the occurrence of myocardial ischemia **(Jiang W, *et al,* 1996)**.

Chronic kidney disease: Chronic kidney disease (CKD) was found to be an independent risk factor for all-cause mortality as well as adverse cardiovascular disease (CVD) events in high-risk populations. It is also a risk factor for death in the year following myocardial infarction or coronary angioplasty. Whether the risk is similar after coronary stenting, whether impaired renal function is associated with an increased risk of cardiovascular death or myocardial infarction (MI) after coronary stenting and whether this risk is maintained beyond the first year are uncertain. Chronic Kidney Disease was strongly associated with an increased risk of incident MI and CVD mortality independent from common cardiovascular risk factors in men and women from the general population **(Meisinger C, *et al,* 2006)**. The presence of even mild chronic kidney disease is associated with a high risk of cardiovascular death and MI following coronary stenting. Further research to address the causes of the association and to define the best therapy for these patients is necessary **(Charytan DM, *et al,* 2006)**.

Sedentary lifestyle: 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 is, the greater the 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.

The effects of maintained physical activity have been related to an increase in para- could expect a lower Heart rate in the Young Group nonsedentary than in the young group sedentary. However, it should be pointed out that the simple measurement of HR is a labile parameter when measured in wakeful conditions. HR can be altered by the influence of the quality and duration of previous rest and also by anxiety or other psychological influences. To avoid these influences, for comparative purposes some authors considered HR during sleep even in athletes. Taking into account these considerations, we prefer to assess the

differences in sedentary life styles by means of a bicycle effort test **(Migliaro ER, *et al*, 2001)**.

Alcohol: Excessive alcohol use leads to an increase in blood pressure, and increases the risk for heart disease. It also increases blood levels of triglycerides which contribute to atherosclerosis **(Heart Disease Behavior, 2009)**. 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 **(Risk Factors and Coronary Heart Disease, n.d.)**.

Acute ingestion of large quantities of alcohol is known to be able to trigger acute myocardial infarction. Since consumption of alcohol is very common in the community, the triggering effect of binge-drinking and consumption of large amounts of alcohol on acute myocardial infarction should be considered as a crucial subject for public health so as to raise the consciousness of the population, especially young persons **(Biyik I and Ergene O, 2005)**. Many of these risk factors are modifiable; so many heart attacks can be prevented by maintaining a healthier lifestyle. Physical activity, for example, is associated with a lower risk profile. Non-modifiable risk factors include age, sex, and family history of an early heart attack (before the age of 60), which is thought of as reflecting a genetic predisposition.

1.10. Pathophysiology:

1.10.1. Mechanisms of Occlusion

Most MIs are caused by a disruption in the vascular endothelium associated with an unstable atherosclerotic plaque that stimulates the formation of an intracoronary thrombus, which results in coronary artery blood flow occlusion. If such an occlusion persists long enough (20 to 40 minutes), irreversible myocardial cell damage and cell death will occur **(Bolooki HM & Bajzer CT, 2009)**.

The development of atherosclerotic plaque occurs over a period of years to decades. The initial vascular lesion leading to the development of atherosclerotic plaque is not known

with certainty. The two primary characteristics of the clinically symptomatic atherosclerotic plaque are a fibromuscular cap and an underlying lipid-rich core. Plaque erosion may occur because of the actions of metalloproteases and the release of other collagenases and proteases in the plaque, which result in thinning of the overlying fibromuscular cap. The action of proteases, in addition to hemodynamic forces applied to the arterial segment, can lead to a disruption of the endothelium and fissuring or rupture of the fibromuscular cap. The degree of disruption of the overlying endothelium can range from minor erosion to extensive fissuring, which results in an ulceration of the plaque. The loss of structural stability of a plaque often occurs at the juncture of the fibromuscular cap and the vessel wall, a site otherwise known as the plaque's "shoulder region." Disruption of the endothelial surface can cause the formation of thrombus via platelet-mediated activation of the coagulation cascade. If a thrombus is large enough to occlude coronary blood flow completely for a sufficient period, MI can result.

1.10.2. Mechanisms of Myocardial Damage

The severity of an MI is dependent on three factors: (1) the level of the occlusion in the coronary artery; (2) the length of time of the occlusion; and (3) the presence or absence of collateral circulation. Generally, the more proximal the coronary occlusion is, the more extensive the amount of myocardium at risk of necrosis (**Bolooki HM & Bajzer CT, 2009**). The larger the MI is, the greater the chance of death because of mechanical complication or pump failure. The longer the period of vessel occlusion, the greater the chances of irreversible myocardial damage distal to the occlusion.

The death of myocardial cells first occurs in the area of myocardium most distal to the arterial blood supply—that is, the endocardium. As the duration of the occlusion increases, the area of myocardial cell death enlarges, extending from the endocardium to the myocardium and ultimately to the epicardium. The area of myocardial cell death then spreads laterally to areas of watershed or collateral perfusion. Generally, after a 6- to 8-hour period of coronary occlusion, most of the distal myocardium has died. The extent of myocardial cell death defines the magnitude of the MI. If blood flow can be restored to at-risk myocardium, more heart muscle can be saved from irreversible damage or death (**Bolooki HM & Bajzer CT, 2009**).

1.11. Complications

Complications may occur immediately following the heart attack (in the acute phase), or may need time to develop (a chronic problem). After an infraction, an obvious complication is a second infarction, which may occur in the domain of another atherosclerotic coronary artery or in the same zone if there are any live cells left in the infarct. Along with this complication there also following problems are occurring.

1.11.1. Arrhythmia: Since the electrical characteristics of the infarcted tissue change, arrhythmias are a frequent complication. The re-entry phenomenon may cause rapid heart rates, and ischemia in the electrical conduction system of the heart may cause a complete heart block. Sinus arrhythmia, defined by means of a calculation of variance of the R-R interval on admission to hospital, was present in 73 of 176 patients admitted to a coronary care unit with acute myocardial infarction. These patients had lower hospital mortality. They tended to have a higher incidence of inferior infarction, and a lower incidence of anterior infarction, and to have smaller infarcts as measured by the Norris index. The main difference between patients with sinus arrhythmia and without sinus arrhythmia related to heart rates on admission to hospital, the patients with the former having slower heart rates at that time **(Wolf MM, *et al*, 1978)**.

1.11.2. Congestive heart failure: Congestive heart failure (CHF) is an imbalance in pump function in which the heart fails to adequately maintain the circulation of blood. The most severe manifestation of CHF, pulmonary edema, develops when this imbalance causes an increase in lung fluid secondary to leakage from pulmonary capillaries into the interstitium and alveoli of the lung.

A myocardial infarction may compromise the function of the heart as a pump for the circulation, a state called heart failure. There are different types of heart failure; left- or right-sided (or bilateral) heart failure may occur depending on the affected part of the heart, and it is a low-output type of failure. If one of the heart valves is affected, this may cause dysfunction, such as mitral regurgitation in the case of left-sided coronary occlusion that disrupts the blood supply of the papillary muscles. The incidence of heart failure is particularly high in patients with diabetes and requires special management strategies.

1.11.3. Cardiogenic shock: A complication that may occur in the acute setting soon after a myocardial infarction or in the weeks following it is cardiogenic shock. Cardiogenic shock is

defined as a hemodynamic state in which the heart cannot produce enough of a cardiac output to supply an adequate amount of oxygenated blood to the tissues of the body.

While the data on performing interventions on individuals with cardiogenic shock is sparse, trial data suggests a long-term mortality benefit in undergoing revascularization if the individual is less than 75 years old and if the onset of the acute myocardial infarction is less than 36 hours and the onset of cardiogenic shock is less than 18 hours. If the patient with cardiogenic shock is not going to be revascularized, aggressive hemodynamic support is warranted, with insertion of an intra-aortic balloon pump if not contraindicated. If diagnostic coronary angiography does not reveal a culprit blockage that is the cause of the cardiogenic shock, the prognosis is poor **(Reynolds HR and Hochman J S, 2008)**.

1.11.4. Myocardial rupture: Laceration or tearing of the walls of the heart, the interatrial or interventricular septum of the papillary muscles or chordae tendineae, or of any of the valves of the heart results from myocardial rupture. Rupture may be due to a variety of pathological entities; however, the majorities are secondary to myocardial infarction. The clinical presentation of myocardial rupture depends on the mechanism and site of injury and the hemodynamic effects of the rupture. Mortality rates are extremely high unless early diagnosis and surgical intervention are provided rapidly **(Shirani J, 2008)**.

Myocardial rupture is most common three to five days after myocardial infarction, commonly of small degree, but may occur one day to three weeks later. In the modern era of early revascularization and intensive pharmacotherapy as treatment for MI, the incidence of myocardial rupture is about 1% of all MIs. (Yip HK, et al, 2003). This may occur in the free walls of the ventricles, the septum between them, the papillary muscles, or less commonly the atria. Rupture occurs because of increased pressure against the weakened walls of the heart chambers due to heart muscle that cannot pump blood out effectively. The weakness may also lead to ventricular aneurysm, a localized dilation or ballooning of the heart chamber.

1.11.5. Pericarditis: As a reaction to the damage of the heart muscle, inflammatory cells are attracted. The inflammation may reach out and affect the heart sac. This is called pericarditis. In Dressler's syndrome, this occurs several weeks after the initial event. Pericarditis that occurs shortly after a heart attack is caused by an overactive response by the body's immune system. When the body senses blood in the pericardial sac or dead or severely damaged heart tissue (as with a heart attack), it triggers an inflammatory response. Cells from the immune

system try to clean up the heart after injury, but, in some cases, the cells can attack healthy tissue by mistake **(LeWinter MM, n.d)**.

1.11.6. Thromboembolism: MI can quickly become fatal if emergency medical treatment is not received promptly. If diagnosed within 12 hours of the initial episode (attack) then thrombolytic therapy is initiated. When a blood vessel is injured, the body uses platelets and fibrin to form a blood clot, because the first step in repairing it (hemostasis) is to prevent loss of blood. If that mechanism causes too much clotting, and the clot breaks free, an embolus is formed **(Furie B and Furie BC, 2008)**.

1.11.7. Ventricular aneurism: An aneurysm is a section of defective wall that bulges outward. A ventricular aneurysm is a defect in the left (or right) ventricle of the heart, usually produced by transmural infarction. The 2 types of aneurysms are true and false. A true aneurysm is made of damaged myocardial wall. A false aneurysm is actually a rupture, whereby the wall of the aneurysm is not myocardium but rather an external containing boundary (eg, pericardium). A functional left ventricular (LV) aneurysm, a forme fruste variant of a true aneurysm, protrudes during ventricular systole but not during diastole; it consists of fibrous tissue, with or without myocardial fibers. True and false LV aneurysms frequently occur in the areas of a healed MI. LV aneurysms may develop after a blunt chest injury, in which case the aneurysm is attributed to myocardial contusion or direct vascular damage, resulting in myocardial necrosis.

1.12. Diagnosis:

The category of invasive tests includes:

1.12.1. Blood Tests:

Blood tests are performed to detect the death of myocardial cells. Living heart cells contain enzymes and proteins (e.g., creatine phosphokinase, troponin, myoglobin) within cell membranes associated with specialized cellular functions such as contraction. When a heart muscle dies, cellular membranes lose integrity and intracellular enzymes and proteins slowly leak into the bloodstream. These enzymes and proteins can be detected by a blood sample analysis. The concentration of the enzymes in a blood sample and more importantly, the changes in concentration found in samples taken over time correlate with the amount of heart muscle that has died **(Bolooki HM & Bajzer CT, 2009)**.

Table 1.1: Normal Values of Blood Tests to Detect Myocardial Infarction

Analyte	Normal Range
Total creatinine phosphokinase (CPK)	30-200 U/L
CK, MB fraction	0.0-8.8 ng/mL
CK, MB fraction (% of total CPK)	0-4%
CK, MB2 fraction	<1 U/L
Troponin I	0.0-0.4 ng/mL
Troponin T	ng/mL

1.12.2. Echocardiogram:

Echocardiogram is a non-invasive diagnostic test that can determine whether the heart muscle has suffered changes and detect blood clots. It is similar with an ultrasound producing visual images of the heart. This test can provide information regarding the heart's strength which is essential in determining the severity of the heart attack, which portion of the heart may have been affected, and what coronary artery is blocked. In normal conditions, approximately 60 percent of the blood in the left ventricle is pumped out each time the heart contracts. If echocardiogram reveals 40 to 45 percent of the blood is pumped out, the patient has suffered a minor heart attack. When 30 to 40 percent of the blood is pumped out, the patient has suffered a moderate to severe heart attack. When only 10 to 25 percent of the blood is pumped out, the patient has suffered a massive heart attack **(Heart Attack Medical Tests & Diagnosis, 2009)**.

1.12.3. Exercise Tolerance Testing (ETT):

Testing the heart during exercise can help identify coronary artery disease. In coronary artery disease, blood flow through the coronary arteries (which supply blood to the heart muscle) is partly or completely blocked. If the coronary arteries are only partly blocked, the heart may have an adequate blood supply when the person is resting but not when the person exercises. Thus, testing the heart during exercise can help identify coronary artery disease. Because exercise stress testing specifically monitors how the heart is functioning, the testing helps doctors distinguish between problems due to a heart disorder and those due to other problems that limit exercise, such as lung disorders, anemia, and poor general fitness.

Exercise testing has two components. Exercise or a drug is used to stress the heart, making it beat faster, and the person is tested for signs of inadequate blood flow to the heart.

The person is also monitored for symptoms that suggest coronary artery disease, such as low blood pressure, shortness of breath, and chest pain.

To stress the heart, most people walk on a treadmill or pedal an exercise bicycle. People who cannot use their legs can use an arm crank. Gradually, the pace of the exercise and the force required to do it (workload) are increased. The ECG is monitored continuously, and blood pressure is measured at intervals. Usually, the person being tested is asked to keep going until the heart rate reaches between 80% and 90% of the maximum for age and sex. If symptoms, such as shortness of breath or chest pain, become too uncomfortable or if significant abnormalities appear on the ECG or blood pressure recordings, the test is stopped sooner. Testing takes about 30 minutes. Exercise stress testing has a small risk; the chance of its causing a heart attack or death is 1 in 5,000. People who cannot exercise can be evaluated using pharmacologic stress testing. For this procedure, a drug, such as dipyridamole, dobutamine, or adenosine, is injected to simulate the effects of exercise on blood flow.

Sometimes, these tests show abnormalities in people who do not have coronary artery disease (a false-positive result), and sometimes tests do not show any abnormalities in people who have the disease (a false-negative result). In people without symptoms, especially younger people, the likelihood of coronary artery disease is low, despite an abnormal test result. In such cases, a positive result is usually more likely to be false than true. These false-positive results may cause considerable worry and medical expense. For these reasons, most experts discourage routine exercise stress testing (such as for screening purposes before an exercise program is begun or during an evaluation for life insurance) in people who do not have symptoms **(Diagnosis, 2006).**

1.12.4. Radiologic Procedures:

Anyone thought to have a heart disorder has chest x-rays taken from the front and the side. The x-rays show the shape and size of the heart and outline blood vessels in the lungs and chest. Abnormal heart shape or size and abnormalities such as calcium deposits within heart tissue are readily seen. Chest x-rays also can detect information about the condition of the lungs, particularly whether blood vessels in the lungs are abnormal and whether there is fluid in or around the lungs. X-rays can detect enlargement of the heart, which is often due to heart failure or a heart valve disorder. The heart does not enlarge when heart failure results from constrictive pericarditis, in which scar tissue forms throughout the sac that envelops the heart (pericardium).

The appearance of blood vessels in the lungs is often more useful in making a diagnosis than the appearance of the heart itself. For instance, enlargement of the pulmonary arteries (the arteries that carry blood from the heart to the lungs) and narrowing of the arteries within the lung tissue suggest high blood pressure in the pulmonary arteries, which may lead to thickening of the muscle of the right ventricle (the lower heart chamber that pumps blood through the pulmonary arteries to the lungs). X-rays of other parts of the body may be taken to detect blockages in other blood vessels **(Diagnosis, 2006)**.

1.12.5. Magnetic Resonance Imaging (MRI):

With magnetic resonance imaging (MRI), a powerful magnetic field and radio waves are used to produce detailed images of the heart and chest. This expensive and sophisticated procedure is used predominantly for the diagnosis of complex heart disorders that are present at birth (congenital) and to differentiate between normal and abnormal tissue. MRI has some disadvantages. It takes longer to produce MRI images than computed tomography (CT) images. Because of the movement of the heart, the images obtained with MRI are fuzzier than those obtained with CT. However, newer MRI scans that are timed to match specific parts of the ECG (called gated MRI) are much clearer than conventional MRI scans **(Diagnosis, 2006)**.

1.13. Global situation of Cardiovascular Disease (CVD)

According to World Health Organization (WHO), in 2005, 17.5 million people died of CVD. This is 30 percent of all deaths globally. Over 80 percent of chronic disease deaths occur in low and middle income countries and occur almost equally in men and women **(WHO, 2009)**.

It is estimated that over the next 10 years (2006–15), China will lose $558 billion in foregone national income due to the combination of heart disease, stroke and diabetes. The newly released Global Burden of Disease (2004 Update) stated that cardiovascular diseases are the leading cause of death in the world, particularly among women; such diseases caused almost 32 percent of deaths in women; and 27 percent in men in 2004. Infectious and parasitic diseases are the next leading cause, followed by cancers, but these groups show much smaller overall sex differentials **(WHO, 2009)**.

CVD accounted for almost 198,000 deaths in the United Kingdom (UK) in 2006. Thirty-five percent of deaths were from CVD; 30 percent of premature deaths in men and 22 percent in women were from CVD. Each year CVD causes over 4.3 million deaths in Europe

and over 2 million deaths in the European Union (EU). CVD causes 48 percent of all deaths in Europe and in the EU (42 percent) **(British Heart Foundation, 2008)**.

CVD accounts for more deaths than any other disease in Canada. 2004 CVD mortality was 72,338, accounting for 31 percent of male deaths and 33 percent of female deaths. CVD costs the Canadian economy about $18 billion annually **(Profiles of research, 2007)**. Coronary heart disease (CHD) is a major cause of death and disability in developed countries. Although CHD mortality rates have declined over the past four decades in the United States and elsewhere, CHD remains responsible for about one-third of all deaths in individuals over age 35.

1.14. Coronary Heart Disease (CHD), Angina Pectoris

It is estimated that about 1.4 million people older than age 35 in the United Kingdom have had a heart attack (MI), (970,000 men and 439,000 women). About 619,000 men and 336,000 women ages 55 to 75 in the United Kingdom have or have had angina. Overall, it is estimated that just over 1.5 million men and 1 million women who have CHD (either heart attack or angina) are living in the United Kingdom **(British Heart Foundation, 2008)**.

CHD is the single most common cause of death in Europe, accounting for 1.92 million deaths each year. It is also the single most common cause of death in the European Union, accounting for over 741,000 deaths each year. In 2003 there were just under 29,000 bypass procedures performed in the United Kingdom. In addition, 73,000 PCI procedures are performed annually **(British Heart Foundation, 2008)**.

In 2005, 445,687 people that died from coronary heart disease. . Worldwide, coronary heart disease killed more than 7.6 million people in 2005-6 **(WHO, 2009)**. CHD alone is the most common cause of death in the United Kingdom, causing around 94,000 deaths in 2006. One in five deaths of men and one in seven deaths of women are from CHD. Other forms of heart disease cause around 31,000 deaths. Total deaths from heart disease in the United Kingdom in 2006 were just over 126,000 **(British Heart Foundation, 2008)**.

1.15. Prevalence of CVD in USA

CVD is the number 1 cause of death in America. Up to 1 million Americans will die of CVD in 2002. According to statistics released by the American Heart Association (AHA) at least 58,800,000 million Americans (i.e. 1 person in 4) suffer from some form of heart disease, 50 million suffer from high blood pressure, 12 million suffer from coronary heart disease, 6.2 million suffer from angina pectoris, 7 million suffer from heart attack, 4.4 million suffer from

stroke, 1.8 million suffer from rheumatic heart disease/fever, 1 million suffer from congenital cardiovascular defects and 4.6 million suffer from congestive heart failure.

It is a myth that heart disease is a man's disease. In fact, cardiovascular diseases are the number one killer of women (and men). These diseases currently claim the lives of more than a half a million females every year - more than the next 16 causes of death put together. In 57 percent of men and 64 percent of women who died suddenly from CVD, there were no previous symptoms of the disease. Besides, stroke killed 159,942 people in 1996. On average, someone in the US suffers a stroke every 53 seconds. **(Heart Disease Facts, n.d.)**.

1.16. Prevalence of CVD in Europe & EU:

CVD kills 4 million Europeans each year. It's the No 1 cause of death. It causes 49% of all European deaths: 55% deaths (women); 43% (men). About half of all deaths from CVD are from heart disease and nearly a third is from stroke. CVD is the main cause of death for women in all 15 countries of the EU and it is the main cause of death for men in all these countries except France **(Heart Disease Facts, n.d.)**.

1.17. Prevalence of CVD and Associated Risk Factors in Asia:

Major Causes of Death for Asian/Pacific Islander Males and Females

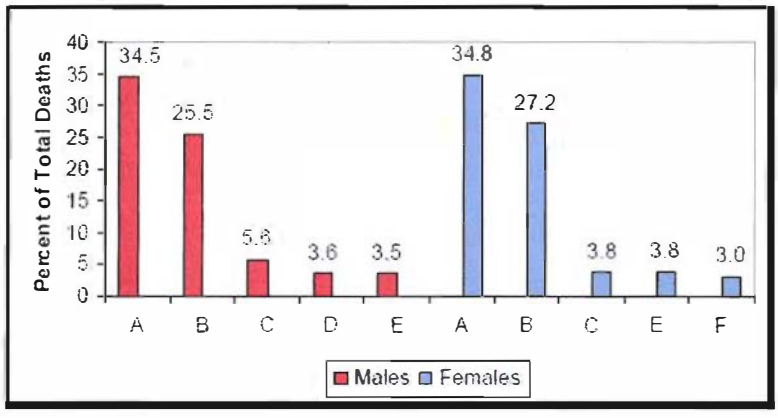

Figure 1.4: Death of Asian/Pacific Islander Males and Females by CVD **(British Heart Foundation, 2008)**.

Cardiovascular Disease (CVD): Among Asians, 5.2 percent have heart disease, 2.9 percent have coronary heart disease, 21.0 percent have hypertension, and 1.8 percent has had a stroke. In 2006, death rates for diseases of the heart in Asians/Pacific Islanders were 136.3 for males and 87.3 for females **(NHIS, 2008)**.

Hypertension: Data from the 2008 NHIS survey showed that 21.0 % of Asian adults age 18 & older had been told on two or more occasions that they had hypertension **(NHIS 2008).**

Smoking: Among Asians only age 18 and older, 9.9 percent are current smokers: (NHIS 2008, NCHS). Rates of use of any tobacco product in 2006, among persons age 12 and older, were 16.0 percent for non-Hispanic Asians only **(NHIS, 2008).** According to 2004–06 data, most Asian adults had never smoked, with rates ranging from 65 percent of Korean adults to 84 percent of Chinese adults. Korean adults (22%) were about two to three times as likely to be current smokers as were Japanese (12%). Asian Indian (7%), or Chinese (7%) adults **(Barnes PM, *et al,* 2008).**

High Blood Cholesterol and Other Lipids: In adults, total cholesterol levels of 240 mg/dL or higher are considered high risk. Levels from 200 to 239 mg/dL are considered borderline-high risk. The percentage of Asian/Pacific Islanders age 20 and older who've ever been told by a professional that they have high blood cholesterol is 29.2 percent **(Statistical fact sheets, 2008).**

Physical Inactivity: Among Asian only adults, age 18 and older, 39.8 percent report no leisure-time physical activity **(NHIS, 2008).**

Overweight and Obesity: According to the 2008 NHIS study of the NCHS, among Asian only adults age 18 and older, 38.1 percent are overweight or obese. (Overweight is BMI of 25 or higher but less than 30.0. Obese is BMI of 30.0 and higher.). Data from the 2008 NHIS study of the NCHS showed 9.4 percent of Asian only adults age 18 and older were obese.

Diabetes Mellitus: Data from the NHIS 2008 showed the prevalence of physician-diagnosed diabetes to be 8.0 percent among Asians. (National Health Interview Survey, 2008. National Center for Health Statistics.). The prevalence of diabetes was more than twice as high for Asian Indian adults (14%) compared with Chinese (6%) or Japanese adults (5%) **(Barnes PM, *et al,* 2008).**

1.18. Tobacco Use:

In Great Britain in 2006, 23 percent of men and 21 percent of women all ages smoked cigarettes. A 50-year cohort study of British doctors showed that mortality from coronary heart disease was 60 percent higher in smokers (and 80 percent higher in heavy smokers) than in nonsmokers. In 2000, smoking caused about 25,000 deaths from cardiovascular disease **(British Heart Foundation, 2008).**

The WHO estimates that by 2020, tobacco is expected to be the single greatest cause of death and disability worldwide, accounting for about 10 million deaths per year. The global tobacco epidemic is predicted to prematurely claim the lives of some 250 million children and adolescents, a third of whom are in developing countries **(World No-Tobacco Day, 2004)**. Tobacco use kills 5.4 million people a year and accounts for one in 10 adult deaths worldwide. Unchecked, tobacco-related deaths will increase to more than 8 million a year by 2030 and 80 percent of those deaths will occur in the developing world. **(WHO, Tobacco facts, n.d.)**.

A joint study by the CDC and the WHO stated that smoking results in a 100 percent increase in the risk of stroke and CHD; a 300 percent increase in the risk of death from undiagnosed CHD; more than a 300 percent increase in the risk of PAD; and a 400 percent increase in the risk of aortic aneurysm **(WHO, The Atlas of Heart Disease and Stroke, 2004)**. Each year, smoking kills over 1 million people in Europe (453,000 from CVD) and about 654,000 people in the EU (183,000 from CVD) **(European Cardiovascular Disease Statistics, 2008)**. A study by the Canadian Heart and Stroke Foundation, found that 19 percent of Canadians smoke, and 29 percent of all smoking-related deaths are heart disease and stroke-related. Almost 6,300 nonsmokers die each year from exposure to secondhand smoke in Canada, and 14.5 percent of heart disease and stroke deaths are attributed to smoking **(Statistics, n.d.)**.

1.19. High Blood Pressure in Children and Adolescents

Analysis of NHES, HHANES, and NHANES surveys of the NCHS (1963–2002), found the BP, pre-HBP and HBP trends in children and adolescents ages 8–17, were downward from 1963–88 and upward thereafter. Pre-HBP and HBP increased 2.3 percent and 1 percent respectively, between 1988 and 1999. Obesity increase partially explained the HBP and pre-HBP 1988–99 rise, more so abdominal than general obesity. Blood pressure and HBP reversed their downward trends 10 years after the increase in the prevalence of obesity. Also an ethnic and gender gap appeared in 1988 for pre-HBP and 1999 for HBP; non-Hispanic blacks and Mexican-Americans had a greater prevalence of HBP and pre-HBP than non-Hispanic whites, and males greater than females. **(Din-Dzietham R, *et al*, n.d)**.

1.20. Smoking Associated with Hypertension:

1.20.1. Smoking:

Other than advanced age, smoking is the single most important risk factor for coronary artery disease. Cigarette consumption is the leading preventable cause of death in the United States, where it accounts for more than 450,000 deaths annually **(Heart Disease Behavior, 2009)**. Ischemic heart disease causes 35 to 40 percent of all smoking-related deaths, with an additional 8 percent attributable to second-hand smoke exposure. Despite the relative stability in prevalence of current smokers in the United States, rates of tobacco use are increasing among adolescents, young adults, and women. Although increased recognition of the hazards of smoking might be hoped to slow these trends, almost 1 billion individuals now smoke worldwide. Smoking has a particularly large impact in the developing world and annually accounts for 1.17 million deaths worldwide **(Ezzati M, *et al*, 2005).** Even among nonsmokers, inhaled smoke, whether from passive exposure or from cigar or pipe consumption, increases coronary risk. Passive smoking exposure can cause endothelial dysfunction in the coronary circulation as well as increased bronchial responsiveness and concomitant pulmonary dysfunction.

Landmark studies in the early 1950s first reported strong positive associations between cigarette smoke exposure and coronary heart disease. Over the next 50 years, an exceptionally consistent series of prospective studies have documented the effects of smoking on coronary risk. These studies suggest that, compared with nonsmokers, persons who consume 20 or more cigarettes daily have a two- to threefold increase in total coronary heart disease. Moreover, these effects depend on dose; consumption of as few as one to four cigarettes daily increases coronary artery disease risk. Such "light" levels of smoking have a major impact on myocardial infarction and all-cause mortality, even among smokers who do not report inhalation. Smoking acts synergistically with oral contraceptive agents, placing younger women taking these agents at even higher relative risk. In addition to myocardial infarction, cigarette consumption directly relates to increased rates of sudden death, aortic aneurysm formation, symptomatic peripheral vascular disease, and ischemic stroke. As for coronary disease, risk directly increases with the number of cigarettes consumed. Prospective evidence has linked cigarette consumption to an elevated risk of hemorrhagic stroke, including intracranial hemorrhage and subarachnoid hemorrhage, again in a dose-response manner. Continued smoking is also a major risk factor for recurrent myocardial infarction.

Historically, cigarette consumption was prevalent in men before women and, at least in the United States, smoking prevalence remains lower in women than men. However, this gender gap has markedly narrowed, with overall consumption rates in women now in excess of 20 percent. Native Americans and those with less education have higher rates, whereas black and Hispanic women appear to smoke less than white women. Because of adverse synergy with oral contraceptives, young female smokers who take oral contraceptives have particularly elevated risks of premature coronary disease and stroke. Smoking is especially hazardous for women with diabetes **(Al-Delaimy WK, *et al*, 2002)**.

1.20.2. Hypertension

There is agreement that hypertension is a quantitative rather than a qualitative disease. The basis for this statement rests on a large number of epidemiologic studies showing that the distribution of blood pressure in the population is continuous, although the curve is skewed at the higher levels of blood pressure. This was not always accepted: In the 1950s there was a sharp debate between Sir George Pickering and Lord Robert Platt that played out in the correspondence section of the Lancet, and was subsequently assembled in a monograph **(Swales JD, 1985)**. Pickering took the view that the distribution of blood pressure in the population is continuous, and that there is no discernible separation between subjects with high blood pressure from those with normal blood pressure. Platt had data that indicated that there was a bimodal distribution, and that a hypertensive subpopulation could be distinguished from the normotensive majority, a dispute that could be summarized as "one hump or two" The general consensus was that Pickering won, and that the unimodal distribution holds sway. This has important implications, first because it suggests that hypertension is unlikely to be the result of a single physiologic process or gene, and second, because it also suggests that any blood pressure level used to define hypertension is arbitrary.

1.20.3. Classification of Hypertension:

Table 1.2: Classification of Hypertension **(Chobanian AV, *et al*, 2003)**.

Classification	Systolic pressure		Diastolic pressure	
	mmHg	kPa	mmHg	kPa
Normal	90–119	12–15.9	60–79	8.0–10.5
Prehypertension	120–139	16.0–18.5	80–89	10.7–11.9
Stage 1	140–159	18.7–21.2	90–99	12.0–13.2

Stage 2	≥160	≥21.3	≥100	≥13.3
Isolated systolic hypertension	≥140	≥18.7	<90	<12.0

1.20.4. Epidemiology of Hypertension:

Hypertension is a condition that can claim a number of firsts: It is the most common chronic condition in the United States; it is the number one reason for an office visit to a physician; it accounts for the most drug prescriptions; it is a major risk factor for heart disease and stroke, which are the first and third leading causes of death in the United States; and it is the number one attributable risk for death throughout the world **(Chobanian AV, *et al*, 2003)**. At the same time, it is both preventable and treatable in the majority of patients. Despite these impressive statistics, hypertension continues to be neglected. It is not recognized as a condition worthy of specialist care, and only about one-third of hypertensive patients in the United States have their blood pressure controlled to target levels that have been proven in numerous studies to reduce the rates of heart attacks and strokes. In addition, although the death rates from these two conditions have been decreasing over the past 20 years, the rates of two others that are also consequences of hypertension—heart failure and chronic kidney disease—have been increasing. It gets worse yet: A major public health concern in the United States today is the epidemic of obesity, which has resulted in a major increase in the prevalence of type II diabetes, and which increases the risk of cardiovascular events to the same degree as a prior myocardial infarction, for which the most effective treatment is the aggressive reduction of blood pressure. So perhaps the worst single statistic relating to hypertension is that less than 25 percent of patients with diabetes have their blood pressure adequately controlled.

1.21. Review of existing literature in this field:

It was found that Tobacco use, ghee intake, raised fasting glucose, high cholesterol, paternal history of cardiovascular disease, low income, and low level of education are associated with premature acute myocardial infarction in South Asians. The association of parental consanguinity with acute myocardial infarction is reported for the first time **(Ismail J, 2004)**. Fatality rates in South Asian (Bangladeshi, Indian and Pakistani) patients with acute myocardial infarction have shown similar declines to those reported for white patients during the past 15 years **(Liew R, *et al*, 2006)**. Research carried out on 2640 patients where 29%

South Asian were admitted with acute myocardial infarction between January 1988 and December 2002. In the past 15 years, death from acute myocardial infarction among South Asians has declined at a rate similar to that seen in white patients. This is largely caused by reductions in indices of infarct severity.

A research was done to identify entry characteristics associated with subsequent myocardial infarction in treated hypertensive patients. Univariate analysis indicated that myocardial infarction cases had higher cholesterol level and were more likely to have a previous history of diabetes than controls. The initial systolic blood pressure and pulse pressure of cases were significantly higher than in controls. A logistic regression model indicated that initial pulse pressure, either as a continuous or as a categorical variable, was the only measure of blood pressure independently associated with myocardial infarction after adjustment for other risk factors. **(Fang J, 1995).**

From that study following result is obtain that a large pulse pressure difference appears to be the most powerful measure available of initial blood pressure to identify, in advance, those hypertensive patients at greatest risk for a subsequent myocardial infarction. The clinical features of 304 patients with acute myocardial infarction with and without hypertension were studied retrospectively. Typical ischemic chest pain was the most common presenting symptom and occurred with a similar frequency in patients with and without hypertension. However, the group with hypertension consisted of proportionately more females than males, more frequently had previously diagnosed hypertension and congestive heart failure, and more often presented with shortness of breath and pulmonary edema. These findings suggest that the higher mortality rate observed in hypertensives during follow-up after myocardial infarction may be due, at least in part, to more severe underlying left ventricular dysfunction **(Clark L T, *et al*, 1990).**

The Tobacco use is one of the most important causes of AMI globally, especially in men. Tobacco use is one of the major avoidable causes of cardiovascular diseases. It aimed to assess the risks associated with tobacco use (both smoking and non-smoking) and second hand tobacco smoke (SHS) worldwide. Research carried on subjects of acute myocardial infarction (AMI) with 27,089 participants in 52 countries (12,461 cases, 14,637 controls) **(Teo KK, *et al*, 2006).** The findings was that all forms of tobacco use, including different types of smoking and chewing tobacco and inhalation of SHS, should be discouraged to prevent cardiovascular diseases.

Although myocardial infarction (MI) is strongly related to smoking, few have studied why some smokers are more vulnerable than others. A study was explored how the risk of MI in current and former smokers is modified by other cardiovascular risk factors and incidence of MI (fatal and nonfatal) amongst 10619 women **(Janzon E, *et al*, 2004)**. Of the 3738 smokers, one-third had at least one major biological risk factor besides smoking; 228 women had MI during follow-up. Smoking and hypertension showed a synergistic effect on incidence of MI. Although smoking is a major risk factor for MI, the risk varies widely between women with similar tobacco consumption. The results illustrate the need of a global risk factor assessment in female smokers and suggest that female smokers should be targets both for intensified risk factor management and programmes to stop smoking.

Scientists investigated the effects of 1-week of smoking cessation on ambulatory blood pressure, heart rate, and heart rate variability in 39 normotensive male habitual smokers. The ambulatory blood pressure, heart rate, and ECG R-R intervals were measured during a 24-hour period with a portable recorder (TM-2425) on the last day of 1-week smoking and nonsmoking periods. The order of the 2 periods was randomized. In the smoking period, the subjects were instructed to smoke cigarettes according to their usual smoking patterns **(Minami J, *et al*, 1999)**. The plasma norepinephrine and epinephrine concentrations were significantly lower in the nonsmoking period than in the smoking period ($P<0.05$ for each). These results demonstrate the substantial and immediate benefits of smoking cessation on these cardiovascular indices.

The smoking habits of 48 patients with malignant hypertension were compared with those of 92 consecutive patients with non-malignant hypertension. Thirty-three of the patients with malignant and 34 of the patients with non-malignant hypertension were smokers when first diagnosed **(Bloxham CA, *et al*, 1979)**. This difference was significant, and remained so when only men or black and white patients were considered separately. Results suggest that malignant hypertension is yet another disease related to cigarette smoking.

The prevalence of smoking, hypertension and diabetes mellitus was assessed in 221 patients suffering from internal carotid stenosis and compared with the prevalences in two sex- and age-matched control groups. This index was lowest in the normotentive non-smokers **(Müller HR and Buser MW, 1990)**. It was only insignificantly higher in the hypertensive non-smokers but significantly so in the normotensive smokers. The index was highest in the hypertensive non-smokers. It was concluded that cigarette smoking, especially if associated with hypertension, is a determinant risk factor for carotid stenosis and occlusion.

A research was done to assess the role of blood pressure in the association between cigarette smoking and left ventricular mass in male and female subjects with essential hypertension. A case-control study with matching ratio of 1:4. In patients with essential hypertension, heavy cigarette smoking (>20 cigarettes/day) is associated with a definite increase in left ventricular mass through a rise in whole-day blood pressure. A pressor mechanism of that type may not be detected by the standard measurement of blood pressure in the clinic, which would make ambulatory blood pressure monitoring a valuable diagnostic tool in this setting. **(Verdecchia P, *et al*, 1995)**.

Myocardial rupture is one of the important complications found with myocardial infarction. Fifty-three of 4,369 patients with acute myocardial infarction died of myocardial rupture. The incidence of rupture varied directly, among men, with the systolic blood pressure on admission to the coronary care unit (CCU). Rupture occurred in 0.3% of the men with systolic pressures **(Christensen DJ, *et al*, 1977)**. Diastolic blood pressure, past history of hypertension, and sustained hypertension after infarctions were not related to the occurrence of rupture. Hypertension appears to be one of several variables interacting to influence the occurrence of myocardial rupture.

To explore risk factors for acute myocardial infarction (AMI) mortality in hypertensive patients treated in primary care. Cardiovascular disease risk factors remain strong predictors of AMI mortality in hypertensive patients but with a different pattern in the two genders. Markers of organ damage are more important predictors in men, whereas markers of impaired glucose metabolism are more important predictors in women. **(Bøg-Hansen E, *et al*, 2007)**.

Pathologic hemorheological parameters and increased platelet aggregation in association with other risk factors significantly increase the possibility of the development of myocardial ischemia. The effects of cigarette smoking and hypertension on hemorheological variables (blood viscosity over a wide range of shear rates, plasma viscosity, microhematocrit, and plasma protein concentration) and on arterial stiffness (pulse wave velocity) were investigated in 33 normotensive men and 81 mild to moderately hypertensive men. Cigarette smoking and hypertension were independently associated with higher blood viscosity at all studied shear rates as well as with higher plasma viscosity, hematocrit, and pulse wave velocity. At constant hematocrit levels, hypertension remained associated with a higher blood viscosity, while the association with cigarette smoking disappeared **(Levenson J, *et al*, 1987)**. Normotensive smokers had the same increase of blood and plasma viscosity

and pulse wave velocity as hypertensive nonsmokers. No interactive effects of hypertension or cigarette smoking on blood or arterial variables were observed, suggesting that the effect of these two factors on blood and vascular rheology are cumulative. Smoking and hypertension may change the flow properties of the blood and the behavior of the arterial wall and this may explain the arterial damage observed in cigarette smokers and hypertensive patients. Study shows that in most patients with heart failure due to left ventricular systolic dysfunction, the underlying cause is coronary heart disease. To reduce progression to heart failure in a patient with acute myocardial infarction, it is important to achieve the earliest possible reperfusion, whether by thrombolysis or primary percutaneous coronary intervention **(Dargie H, 2005)**.

To achieve success to proper treatment is the effective integration of antithrombotic therapy combined with timely reperfusion, either primary percutaneous coronary intervention or fibrinolysis for ST-elevation myocardial infarction, and invasive investigation and revascularisation for non-ST-elevation myocardial infarction, underpinned by risk stratification and optimized systems of care **(Harvey D and White HD, 2008).** After the development of troponin assays for the detection of myonecrosis, the universal definition and classification of myocardial infarction now indicates the underlying pathophysiology.

Aims and objectives of the study

The present study was designed to assess:

- the relationship between myocardial infarction and hypertension.
- the prevalence of smoking associated with hypertension.
- the relationship between life style risk factors and myocardial infarction.

Significance of the study

Hypertension results from an increased amount of blood pumped by the heart or from increased resistance to the flow of blood through the arterial blood vessels. Hypertension is known as the "silent killer," and it also often produces few explicit symptoms like it results in damage to the heart, sometimes myocardial infarction. And a patient presenting with acute myocardial infarction and severe or uncontrolled hypertension represents an emergency situation requiring prompt and accurate evaluation of both conditions.

The prevalence of hypertension is getting higher day by day. This group of patients is very susceptible to myocardial infarction. Moreover patients along with history of smoking are in serious risk of develop MI. Smoking is addiction as well as it contain nicotine which cause serious damage to cardiac function. Unfortunately smoking is a common practice in our society as well as in the whole world.

If hypertension persists, various problems could arise out. People with hypertension can contract heart attack at anytime. Hypertension can also cause defective vision. Hypertension can also damage the kidneys as the blood vessels of the kidney can thicken with high blood pressure. Smoking causes permanent destruction of the alveoli and narrowing of small blood vessels in the lungs. It results decreased oxygen supply, which require a higher blood pressure, thus causing extensive circulatory problems and premature heart attacks. Smokers face difficulty in running and exercising. Both factors are responsible for increasing the risk of myocardial infarction. The morbidity and mortality of these patients are due to such complications.

Hypertension and smoking are the most important risk factors of developing myocardial infarction. Since, these factors are modifiable a person can gain control over these factors by changing lifestyle, exercise or taking medicine. This study will be helpful to increase the awareness among people. By taking immediate treatment, or by controlling blood pressure, food habit and physical activity a person can avoid the harmful effect of myocardial infarction. This study is also relates these risk factors to myocardial infarction. Thus, the result of the study is expected to improve management of Myocardial Infarction in hypertensive patients with smoking habits which ultimately will help to improve the disease management process.

CHAPTER 2

Materials and Methods

2.1 Type of study:

It was a descriptive study. It was attempted to establish a relationship in myocardial infarction patients with history of smoking and hypertension. In addition to this, the study examined for other risk factors.

2.2 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 unit, Post coronary care unit Intensive care unit and has a fully 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.

2.3 Study population: All admitted patients of MI diagnosed by the hospital physicians.

2.3.1 Inclusion Criteria of the cases:

i). Patient of diagnosed MI ages 25-85yers

ii) Both sexes irrespective of religion and occupation.

2.3.2 Exclusion Criteria of the cases

i) Patients of cardiac disease other than Myocardial Infarction.

ii) Post operative patient

iii) Any other chronic diseases.

2.4 Sample size: Sample size was 135.

2.5 Sampling Technique: In this study, purposive sampling technique was followed.

2.6 Research Approach:

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

2.7. Research Equipments:

The following equipments were used in this study,

1. Interview schedule
2. Measuring Tape.
3. Weighing machine (Bathroom Scale)
4. Sphygmomanometer. (Aneroid type)
5. Stethoscope.

2.8. Data collection method:

After explaining the purpose of the study to the respondents and obtaining their verbal consent, the researcher interviewed all the respondents by asking question in Bengali and using a thoroughly pre–tested questionnaire. The questionnaire consisted of three parts: Part-1 consisted of the respondents' general information; part-2 was about behavioral characteristics whereas Part-3 consisted of physical examination, recording blood pressure and anthropometrical measurements examination by checklist, clinical examination and laboratory tests.

2.9. Diagnosis of Myocardial Infarction patients:

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

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

2.9.1. Treatment

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

2.10. Study period:

Study period was one year commencing from July 2009 to May 2010. To complete the study in time, a work schedule was 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 were spent on official correspondence, data collection, data analysis, report writing and submission of report.

2.11. Data analysis:

All the data were checked after collection. Then data were entered into broad sheet. The result was shown in bar, pie chart and calculate the percentage the different risk factors of MI patients.

CHAPTER 3

Results

Results

3.1. Distribution (%) of myocardial infarction in hypertensive patients.

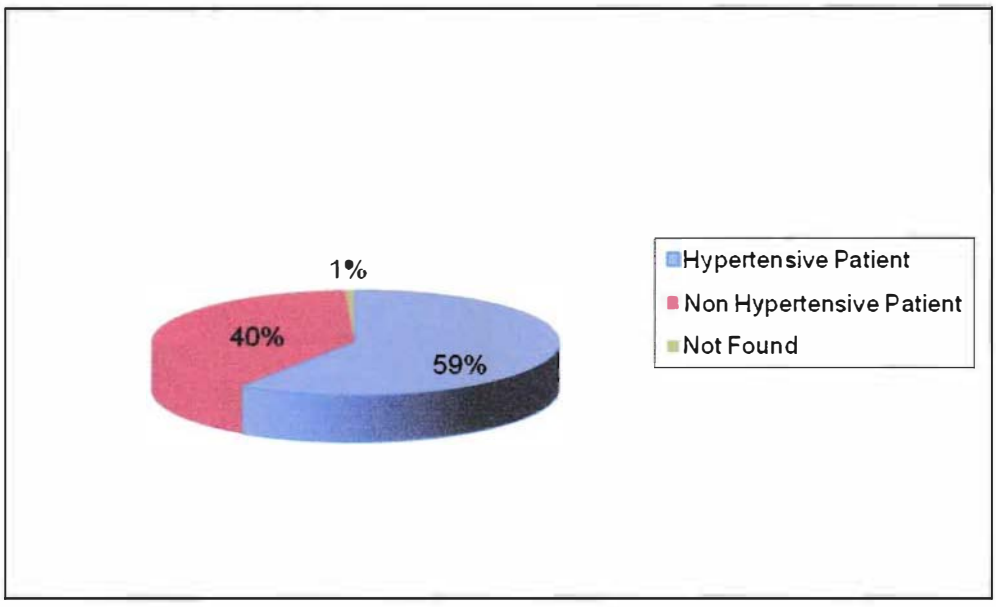

Fig. 3.1: Distribution (%) of myocardial infarction in hypertensive patients

The study shows that among 135 patients 59% were hypertensive and 40% were non-hypertensive patients with myocardial infarction. Among them data of 1% patients were not found (Fig 3.1).

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

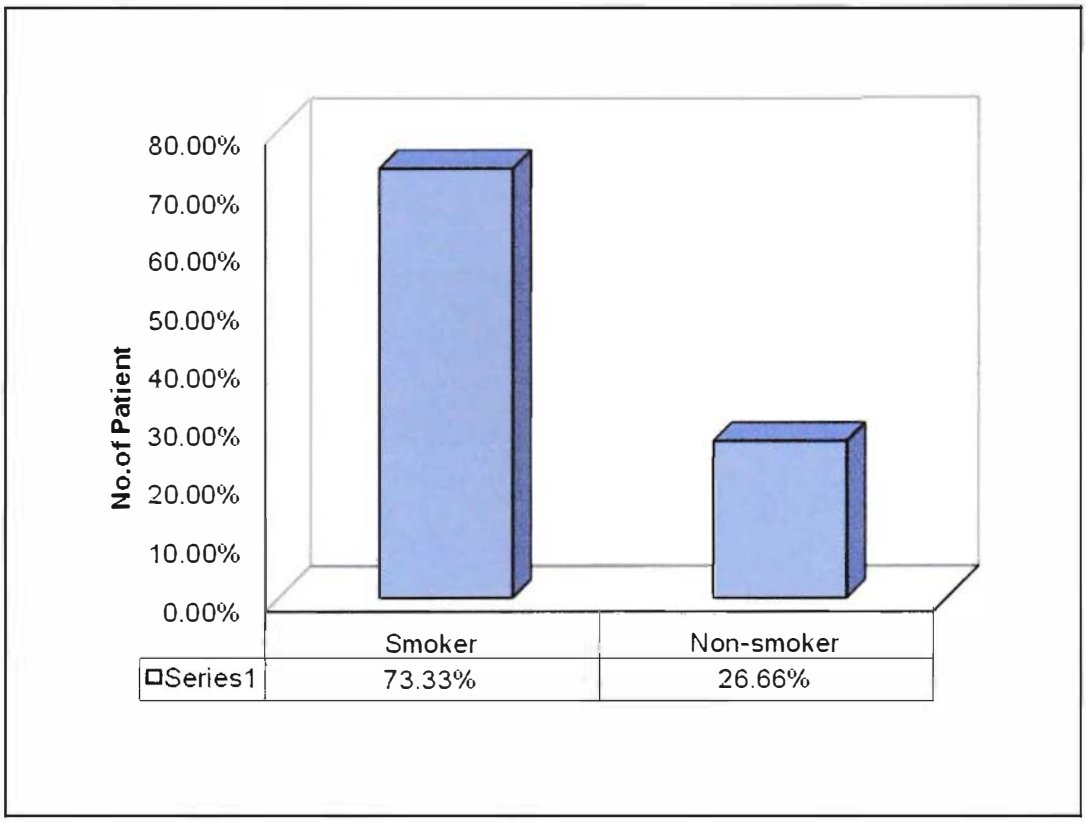

Fig 3.2: Distribution (%) of myocardial infarction in patients with smoking.

The study shows that among 135 patients 73.33% patients were smoker and 26.66% were non-smoker with myocardial infarction (Fig 3.2).

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

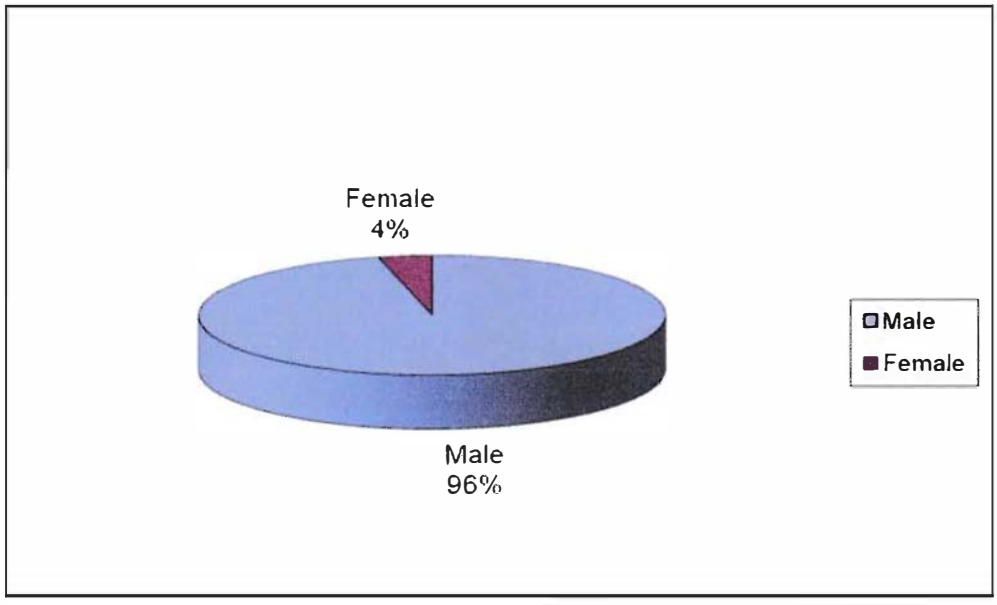

Fig 3.3: Distribution (%) of myocardial infarction among male and female patients.

The study shows that among 135 patients 96% were male and 4% were female patients having myocardial infarction disease (Fig 3.3).

3.4: Distribution (%) of types of myocardial infarction

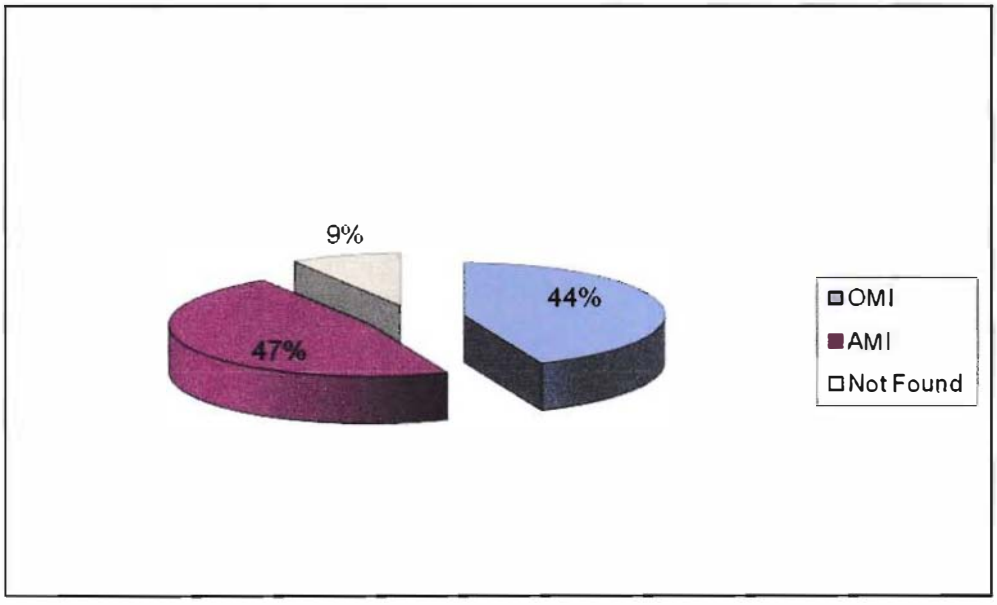

Fig 3.4: Distribution (%) of types of myocardial infarction

The study shows that among 135 patients 47% were suffered from AMI and 44% were suffered from OMI patients (Fig 3.4).

3.5: Distribution (%) of myocardial infarction depending on occupation.

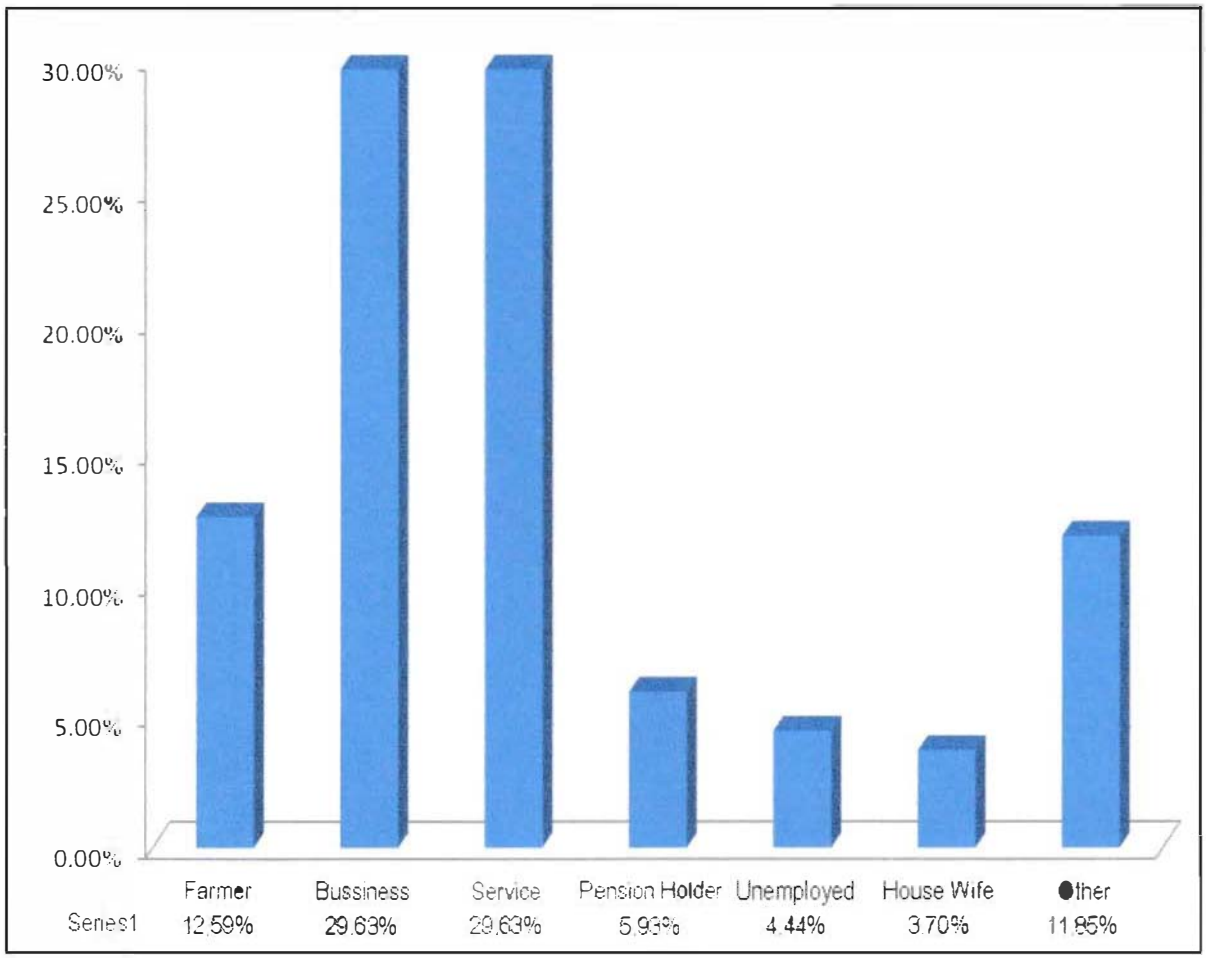

Fig 3.5: Distribution (%) of myocardial infarction depending on occupation.

The study shows that among 135 patients 12.59% farmer, 29.63% business, 29.63% service, 5.93% pension holder, 4.44% unemployed, 3.70% housewife and 11.85% other occupational patients with myocardial infarction (Fig 3.5).

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

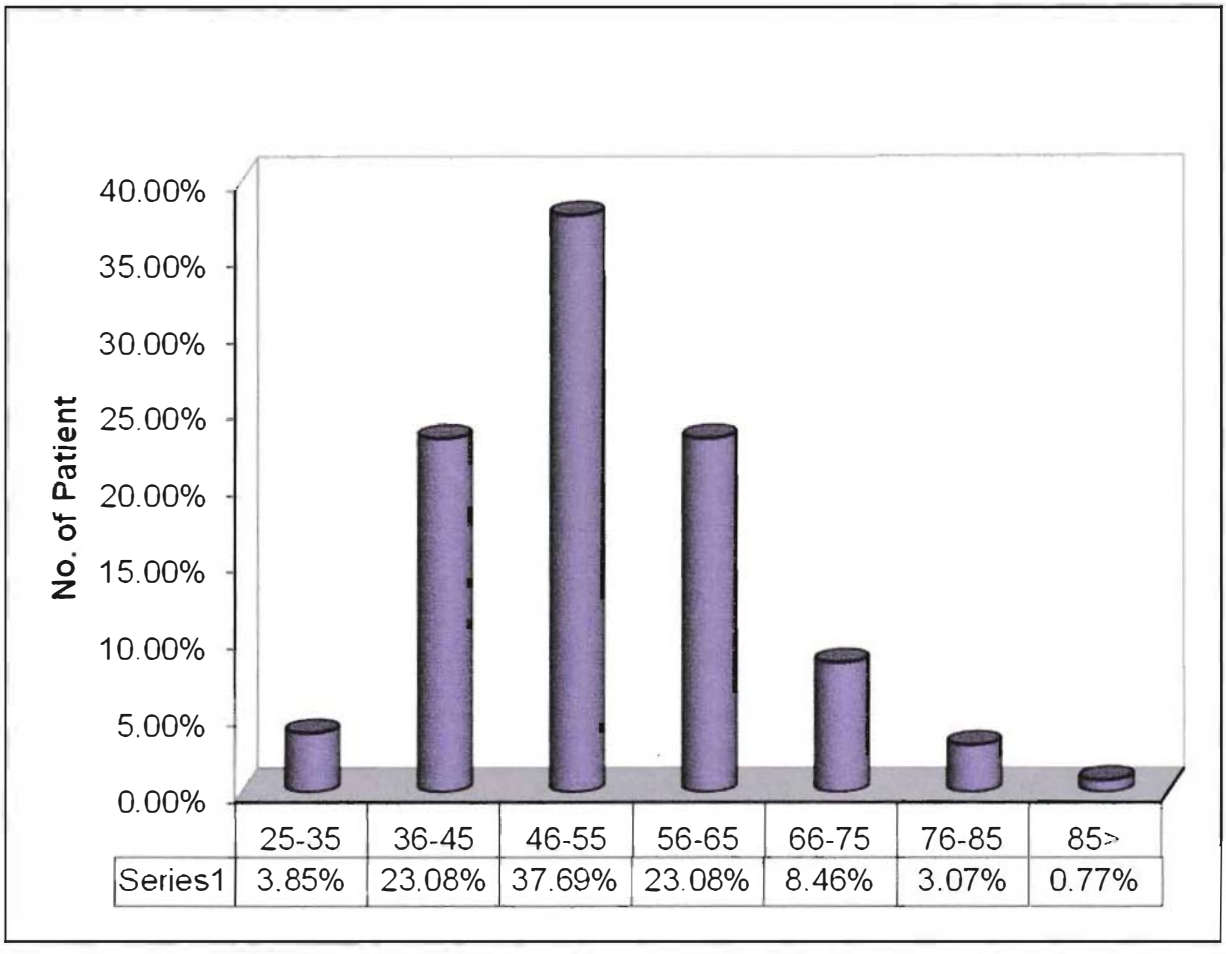

Fig 3.6: The age distribution (%) of total patients with myocardial infarction.

The study shows that among 135 patients of different age with myocardial infarction where 3.85% patients were in 25-35 years, 23.08% patients were in 36-45 years, 37.69% patients were in 46-55 years, 23.08% patients were in 56-65 years, 8.46% patients were in 66-75 years, 3.07% patients were in 76-85 years and 0.77% patients were in more than 85 years old (Fig 3.6).

3.7: Distribution (%) of myocardial infarction depending upon Oil use

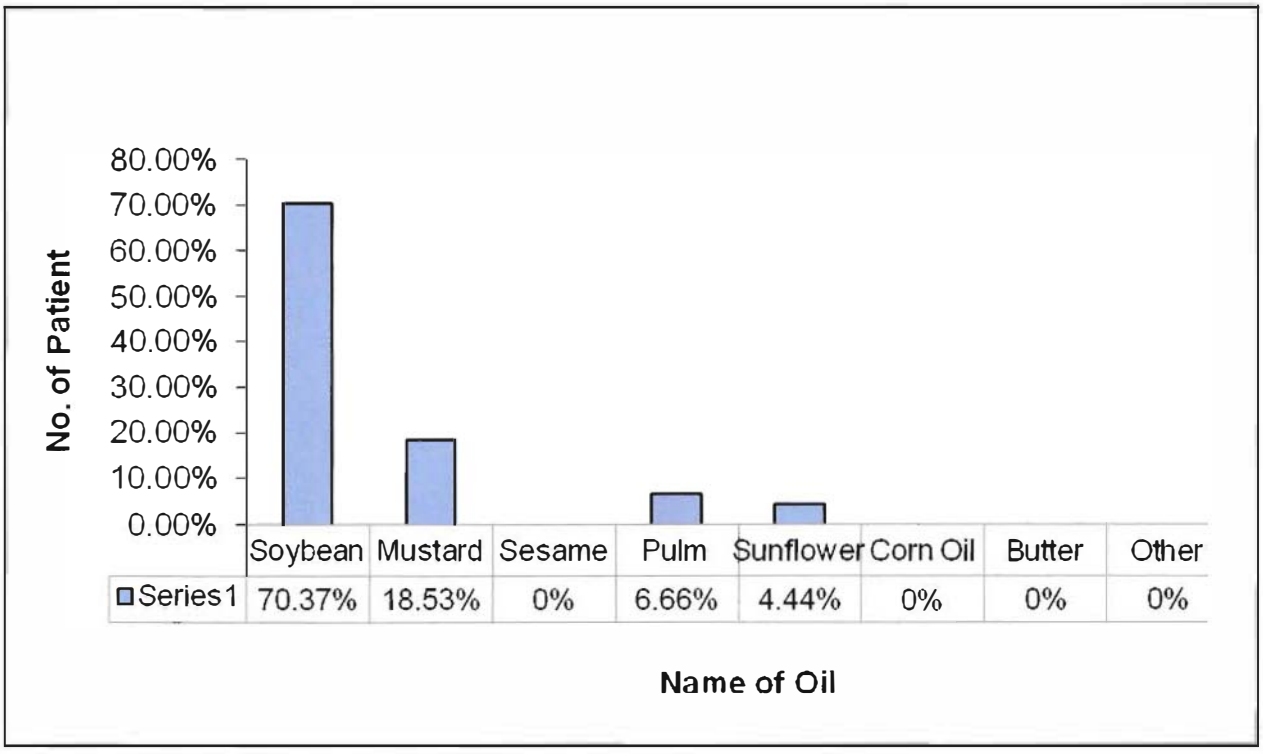

Fig 3.7: Distribution (%) of myocardial infarction depending upon Oil use

The study shows that among 135 patients 70.37% patients were used Soybean oil, 18.53% were used Mustard oil, 6.66% were used Pulm oil, 4.44% were used Sunflower oil (Fig 3.7).

3.8: Distribution (%) of myocardial infarction depending upon BP (systolic).

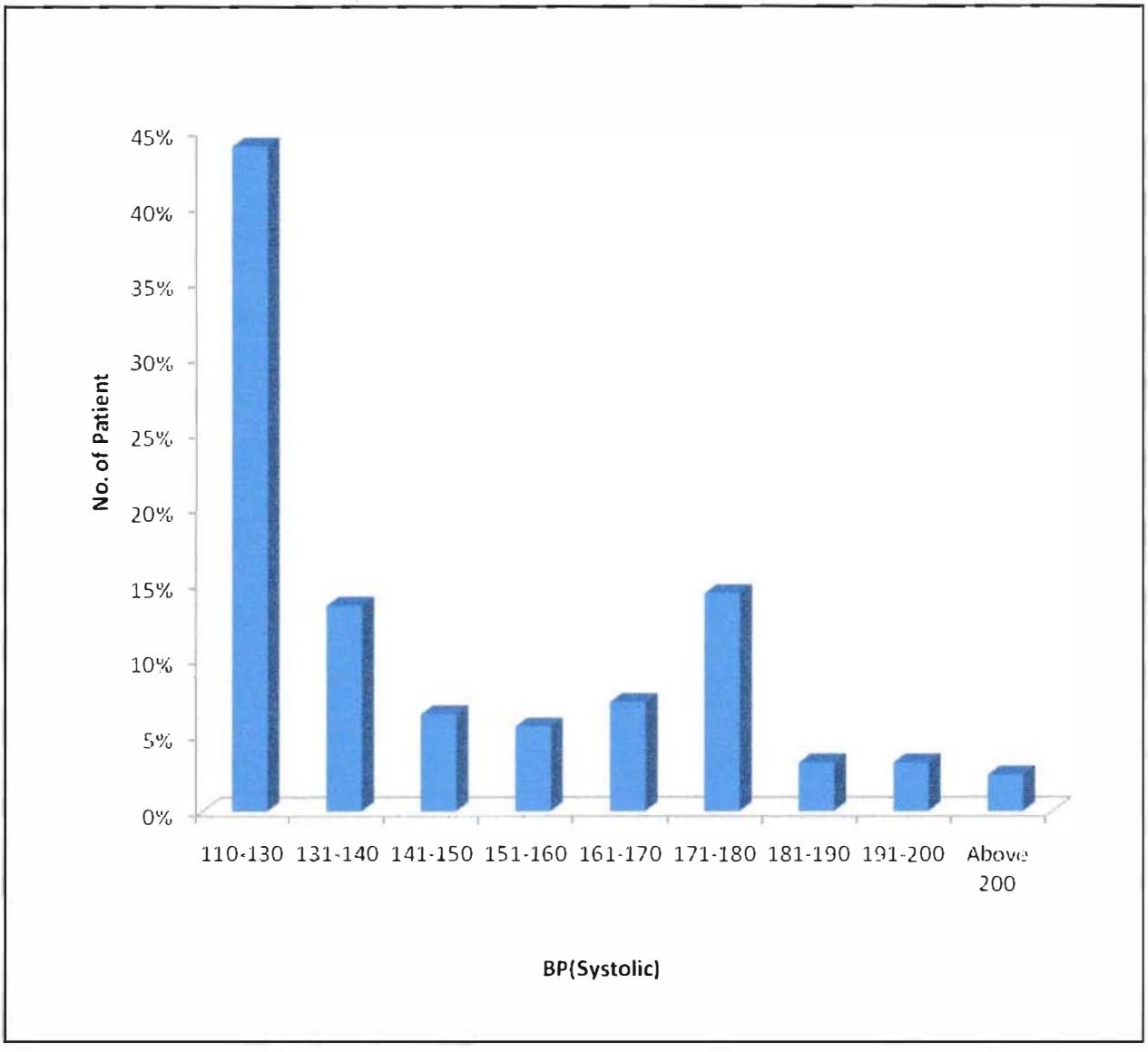

Fig 3.8: Distribution (%) of myocardial infarction depending upon BP (systolic).

The study shows that among 135 patients has systolic blood pressure where 44% patients had 110-130 mmHg, 13.60% had 131-140 mmHg, 6.40% had 141-150 mmHg, 5.60% had 151-160 mmHg, 7.20% had 161-170 mmHg, 14.20% had 171-180. mmHg, 3.20% had 181-190 mmHg, 3.20% had 191-200 and 2.40% had more than 200 mmHg (Fig 3.8).

3.9: Distribution (%) of myocardial infarction according to smoking duration.

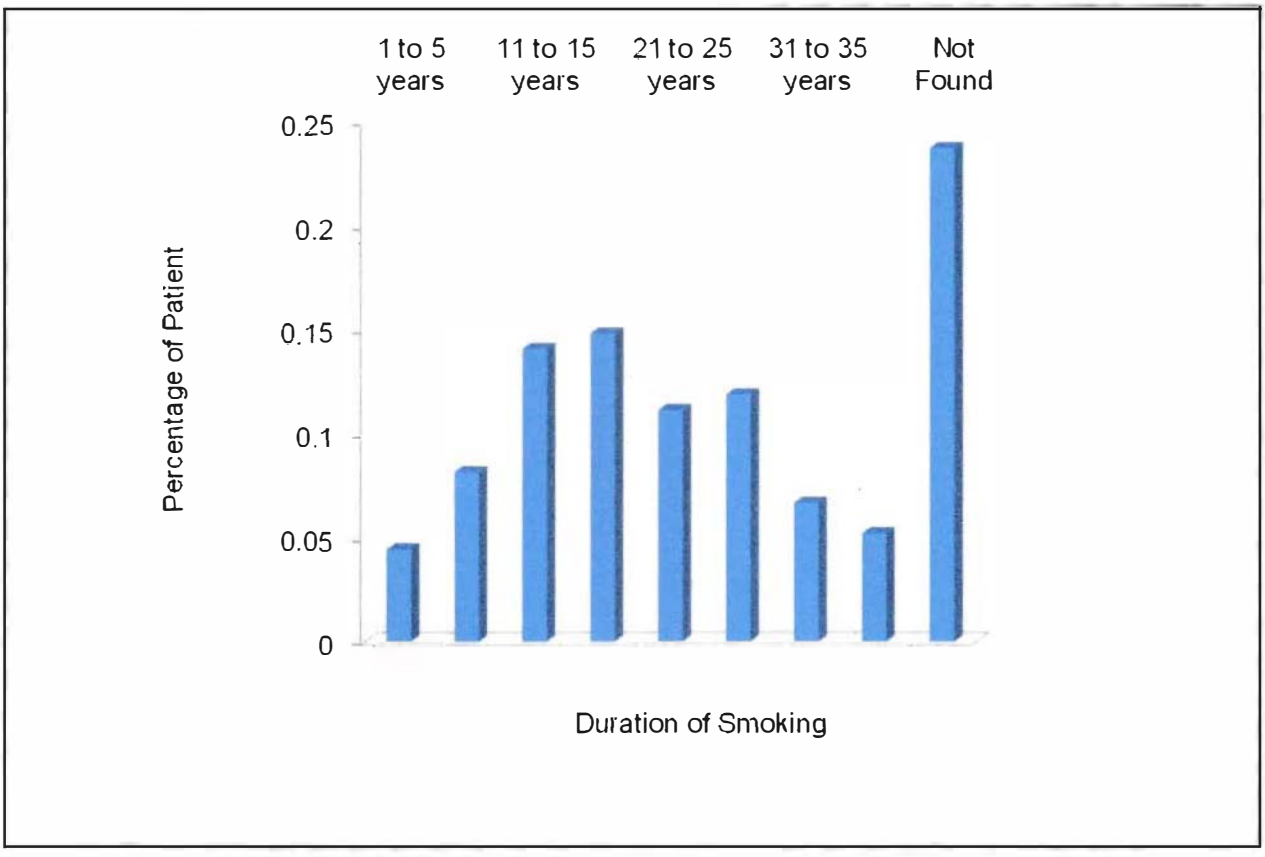

Fig 3.9: Distribution (%) of myocardial infarction according to smoking duration.

The study shows that among 135 patients 4.44% were smoking for 1-5 years, 8.15% were smoking for 6-10 years, 14.07% were smoking for 11-15 years, 14.81% were smoking for 16-20 years, 11.11% were smoking for 21-25 years, 11.86% were smoking for 26-30 years, 6.66% were smoking for 31-35 years, and 5.20% were smoking for 36-40 years. Information of 23.70% were not found (Fig 3.9).

CHAPTER 4

Discussion and Conclusion

This is a survey based study that has collected data on Myocardial Infarction over a period of 1 year. Data were obtained from numerous sources; there was no age limit, and the cardiology departments coordinated their efforts. Using data from this study, we demonstrated that Myocardial infarction was more prevalent in smokers than non-smokers. Besides hypertensive patients were more prone to have myocardial infarction than non-hypertensive patients. In addition, the distribution of risk factors like smoking and hypertension was compared among MI patients. Such data are of interest in order to design and implement effective secondary prevention strategies.

In this study, 135 events were recorded. Among these, 130 (96%) men and 5 (4%) women were suffering from MI. Here 47% people were attacked by AMI and 44% people with OMI. The distribution of MI in Random Blood Sugar was found 11% normal and 35.5% abnormal. Rest of the data was not found. 67% of the total population takes table salt with their meal. Betel nuts were taken by 43.7% of population. 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%. People of 58.52% took excessive tea or coffee. Distribution of MI was more prevalent in businessman and service holder when it was categorized based on occupation.

Hypertension is clearly a major public health problem. It is the one of the most important risk factors in Myocardial infarction. And smoking is another risk factor. Tobacco use is the most common cause of avoidable cardiovascular mortality worldwide. In our study we found 59% of total population was suffering from hypertension and 73.3% population were from smoking. Chronically, cigarette smoking induces arterial stiffness which may persist for a decade after smoking cessation. The incidence of hypertension is increased among those who smoke 15 or more cigarettes per day and the coexistence of hypertension and smoking is common in myocardial infarction patients.

According to Ismail J, tobacco use, ghee intake, raised fasting glucose, increased cholesterol; low income and low level of education are associated with premature acute myocardial infarction in South Asia **(Ismail J, 2004)**. In this study we assessed the above mentioned risk factors as well as some other factors. And the obtained result that we found is consistent with that study.

Systolic blood pressure and pulse pressure difference are appeared to be the most important parameter found in MI patients. It indicates that hypertension patients are at

greatest risk for developing MI. And this finding suggests that the high mortality rate is observed in hypertension patients.

Tobacco is the one of the most important cause of MI. From the research of Teo KK, its confirmed that both smokers and second hand smokers are susceptible to develop MI (**Teo KK, *et al*, 2006**). Teo KK concluded that all forms of tobacco use including different types of smoking and chewing tobacco or inhalation of second hand smoking should be discouraged to prevent cardiovascular disease. Our study also showed that about 74% of patients were habituated with tobacco.

Janzon E proposed that smoking and hypertension are the two risk factors which give synergistic effect on incidence of MI **(Janzon E, *et al*, 2204)**. The risk varies widely between women with similar tobacco consumption. Although, in our study, there were only 5 women and each of them have no smoking habit, but the result is consistent in case of male patients.

Muller HR and Buser MW carried out a research to find out the prevalence of smoking, hypertension and diabetes mellitus and they compared it with sex and age matched control **(Müller H R and Buser M W, 1990)**. The index was lowest in normotensive patients and its highest in hypertensive non-smokers. It can be concluded that smoking associated with hypertension is a determinant risk factor for occlusion of blood vessels and resulting myocardial infarction.

Conclusion

The main objective of the study was to find out the relationship among MI patients with hypertension and smoking habits. In this study it was found that age, educational status, occupation was significantly associated with coronary heart disease. Among life style risk factors diabetes, smoking, type of smoking, betel nut chewing, and tobacco consumption all were highly significant associated with development of MI. Family history of asthma, hypertension and CHD shows a significant relationship with MI.

The finding reports here, for myocardial infarction, are consistent with there of other investigations. A significant association of smoking and hypertension with myocardial infarction was noted. From the result it was obtained that among 135 MI patients 59% patients had hypertension and 73.3% had smoking habit. This study will be help to increase the awareness between people health by taking immediate treatment, by taking drug, or by controlling blood pressure, food habit and physical activity to avoid the harmful effect of myocardial infarction.

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 smoking and hypertension responsible for development of cardiovascular diseases.

CHAPTER 5

References

Abildstrom SZ, Rask-Madsen C, Ottesen M M, Andersen P K, Rosthøj S, Torp-Pedersen C, Køber L, 2002. *Impact of age and sex on sudden cardiovascular death following myocardial infarction.* Heart 2002; 88:573-578

Al-Delaimy WK, Manson JE, Solomon CG, 2002.*Smoking and risk of coronary heart disease among women with type 2 diabetes mellitus.* Arch Intern Med 2002; 162:273.

Anderson RD, Pepine C J, 2007. *Gender Differences in the Treatment for Acute Myocardial* Infarction. *Circulation* 2007; 115:823-826.

Barnes PM, Adams PF, Powell-Griner E. *Health Characteristics of the Asian Adults Population: United States*, 2004-2006: Advance Data from Vital and Health Statistics; 394.

Biyik I and Ergene O, 2005. *Acute myocardial infarction associated with heavy alcohol intake in an adolescent with normal coronary arterie.* Cardiology in the young 2006; 16:2:190-192

Bloxham CA, et al, 1979. *Malignant Hypertension and Cigarette Smoking.* British Medical journal, 1979:1:581-583.

Bøg-Hansen E, Larsson CA, Gullberg B, Melander A, Boström K, Råstam L, Lindblad U, 2007. *Predictors of acute myocardial infarction mortality in hypertensive patients treated in primary care.* Scandinavian Journal of Primary Health Care, 2007: 25(4):237 - 243

Bolooki HM & Bajzer CT, 2009. *Acute Myocardial Infarction*, Retrieved on 1st April from http://www.clevelandclinicmeded.com/medicalpubs/diseasemanagement/cardiology/acute-myocardial-infarction/

British Heart Foundation, 2008. *Coronary Heart Disease Statistics.* Retrieved on 2nd April 2010 from http://www.heartstats.org/documents/download.asp?nodeid=8030&libraryversionid=2940

Cardiovascular Disease Facts and Statistics, (n.d.). Retrieved on 2nd April 2010 from http://www.healingwithnutrition.com/cdisease/cardiovascular/cardiovascular.html

Centers for Disease Control and Prevention (CDC): *Annual smoking-attributable mortality, years of potential life lost, and economic costs—United States*, 1995-1999. *MMWR Morb Mortal Wkly Rep* 2002; 51:300.

Charytan DM, Kuntz RE, Chhabra A, Cutlip DE., 2006. *Relationship of chronic kidney*

disease to cardiovascular death and myocardial infarction following coronary stenting. J Nephrol. 2006;19(6):764-70.

Chobanian AV, Bakris GL, Black HR. *The seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure*: the JNC report. JAMA 2003; 289(19):2560–2572.

Chockalingam A, Venkatesan S, Dorairajan S, Moorthy C, Chockalingam V, Subramaniam T, 2003. *Estimation of Subjective Stress in Acute Myocardial Infarction.* Journal of postgraduate medicine. 2003 :(49) 3: 207-210.

Christensen D J, Ford M, Reading J, Castle JH, 1977. *Effect of hypertension on myocardial rupture after acute myocardial infarction.* CHEST 1977 ;(72) 5: 618-622

Clark L T, Maw M, Bridges D, Kline D, 1990. *Atypical myocardial infarction and hypertension: an inner city experience.* Journal of human hypertension 1990;(4)

Collinson PO, Boa FG, Gaze DC. *Measurement of cardiac troponins.* Ann Clin Biochem 2001; 38:423–49.

Communicable diseases, n.d. *Communicable diseases.* Retrieved on 2nd April 2010 from http://encyclopedia2.thefreedictionary.com/communicable+diseases

Congenital heart disease, n.d. In Kidshealth. *Congenital Heart Defects.* Retrieved on 30th March 2010 from http://kidshealth.org/parent/medical/heart/congenital_heart_defects.html?tracking=P_

Dargie H, 2005. *Heart failure post-myocardial infarction: a review of the issues.* Heart 2005; 91:ii3-ii6

Diagnosis, 2006. *Medical History and Physical Examination.* Retrieved on 3rd April from http://www.merck.com/mmhe/sec03/ch021/ch021c.html

Din-Dzietham R, Liu Y, Bielo M-V,Shamsa F. *High Blood pressure trended in children and adolescents in national surveys, 1963-2002.* Circulation

European Cardiovascular Disease Statistics, 2008. Retrieved on 17th April from http://www.heartstats.org/temp/ESspweb08spchapter.1.pdf

Ezzati M, Henley SJ, Thun MJ, Lopez AD, 2005. *Role of smoking in global and regional cardiovascular mortality. Circulation* 2005; 112:489.

Fang J, Madhavan S, Cohen H, Alderman M. H, 1995. *Measures of blood pressure and myocardial infarction in treated hypertensive patients.* Journal of hypertension . 1995, (13): 413-419

Fogoros NR, 2007. *Heart Disease Blog.* Retrieved on 1st April 2010 from

http://heartdisease.about.com/b/2007/10/25/new-definition-for-myocardial-infarction.htm

French JK & White HD, 2004. *Clinical implications of the new definition of myocardial infarction.* Heart. 2004; 90(1): 99–106.

Furie B and Furie BC 2008. "Mechanisms of thrombus formation". *New England Journal of Medicine* 359 (9): 938–949.

Handbook of disease, 2003. *Myocardial infarction.* Retrieved on 19th April from http://www.wrongdiagnosis.com/symptoms/chest_pain/book-causes-12a.htm

Harlan W and Harlan L, 2002. In healthline. *Noncommunicable Disease Ccontrol.* Retrieved on 2nd April 2010 from http://www.healthline.com/galecontent/noncommunicable-disease-control

Harvey D White HD, 2008. *Acute myocardial infarction. 2008*;118;219-222. Circulation. Heart Attack Medical Tests & Diagnosis, 2009. In Omnimedical, *Cardiovascular System.* Retrieved on 5th April from http://www.omnimedicalsearch.com/conditions-diseases/heart-attack-tests-diagnosis.html

Heart Disease Behavior, 2009. In *Centers for Disease control and prevention.* Retrieved on 5th April from http://www.cdc.gov/heartdisease/behavior.htm

Heart Disease Risk Factors, 2009. In *Texas Heart institute centre,* Retrieved on 13th April From http://www.texasheartinstitute.org/hic/topics/hsmart/riskfact.cfm

Heart disorders, acquired, in children, n.d. In *Better Health Channel.* Retrieved on 5th April http://www.betterhealth.vic.gov.au/bhcv2/bhcarticles.nsf/pages/Heart_disorders_acquired_children

Heart Disease Facts, (n.d.). Retrieved on 5th April from http://www.annecollins.com/nutrition/heart-facts.htm

Heart resource. n.d. Retrieved on 5th April from http://www.health-res.com/coronary-heart-disease-statistics/

Hoffman JE, et al. *Prevalence of congenital heart disease.* Am Heart J. 2004;147:425-439.

Ismail J, 2004. *Risk factors for non-fatal myocardial infarction in young South Asian adults.* Heart 2004; 90:259-263

Janzon E, Hedblad B, Berglund G, Engström G , 2004. *Tobacco and myocardial infarction in*

middle-aged women: a study of factors modifying the risk. J Inter Med. 2004; 256(2):111-8.

Jiang W, *et al,* 1996. *Mental stress--induced myocardial ischemia and cardiac events.* JAMA. 1996; 275(21):1651-6.

Kurth T, Kase CS, Berger K, 2003. *Smoking and the risk of hemorrhagic stroke in men.* Stroke 2003; 34:1151.

Liew R, 2006. *Declining case fatality rates for acute myocardial infarction in South Asian and white patients in the past 15 years.* Heart 2006; 92:1030-1034

Levenson J, Simon AC, Cambien FA, Beretti C, 1987. *Cigarette smoking and hypertension: Factors independently associated with blood hyperviscosity and arterial rigidity.* Arteriosclerosis, 1987;(7): 572-577,

LeWinter MM, n.d. Pericardial Diseases. In: Libby P, Bonow RO, Mann DL, Zipes DP, eds. Braunwald's Heart Disease: A Textbook of Cardiovascular Medicine. 8th ed. Philadelphia, Pa; Saunders Elsevier; 2007: chap 70.

Mallinson T, 2010. *"Myocardial Infarction"*. Focus on First Aid (15): 15. http://www.focusonfirstaid.co.uk/Magazine/issue15/index.aspx.

Marfella R, Siniscalchi M, Esposito K, Sellitto A, Fanis UD, Romano C, Portoghese M, Siciliano S, Nappo F, Sasso FC, Mininni N, Cacciapuoti F, Lucivero G, Giunta R, Verza M, Giugliano D, 2003. *Effects of Stress Hyperglycemia on Acute Myocardial Infarction.* Diabetes Care 2003; 26: (11): 3129-3135

Medicinenet 2003. *High Blood Pressure - A Silent Killer.* Retrieved on 3rd April 2010 from http://www.medicinenet.com/script/main/art.asp?articlekey=13118

Mehta RH, Rathore SS, Radford MJ, Wang Y, Krumholz HM, 2001. *Acute myocardial infarction in the elderly: differences by age.* J Am Coll Cardiol, 2001; 38:736-741

Meisinger C, Döring A, Löwel H, 2006. *Chronic kidney disease and risk of incident myocardial infarction and all-cause and cardiovascular disease mortality in middle-aged men and women from the general population.* European Heart Journal, 2006; 27 :(10)1245-1250.

Migliaro ER, 2001. *Relative influence of age, resting heart rate and sedentary life style in short-term analysis of heart rate variability.* Braz J Med Biol Res 2001; (34):493-500.

Minami J, Ishimitsu T, Matsuoka H, 1999. *Effects of Smoking Cessation on Blood Pressure and Heart Rate Variability in Habitual Smokers.* Hypertension. 1999; 33:586-590.

Moller JH. *Prevalence and incidence of cardiac malformation.* In: Perspectives in Pediatric Cardiology. 1998; 6:19–26;

Morphology of acute myocardial infarction at prenecrotic stage" (in Russian). *Kardiologiia* 2010; 50 (1): 4–8.

Müller H R and Buser M W, 1990. *Smoking and hypertension: risk factors for carotid stenosis.* Journal of Neurology, 1991; 4(2):238

NHIS 2008. In *Centers for disease control and prevention.* Retrieved on 2nd April from http://www.cdc.gov/nchs/nhis/nhis_2008_data_release.htm

Noboru Y, 1973. *Effects of Hereditary and Environmental Factors on Development of Myocardial Infarction.* Japanese circulation journal 1973;37(1), 69-76,

Panagiotakosa DB, Rallidis LS, Pitsavos C, Stefanadis C, Kremastinos D, 2006. *Cigarette smoking and myocardial infarction in young men and women: A case-control study.* International journal of Cardiology 2007; 116(3):371-375

Peter W F, D'Agostino RB, Sullivan L, Parise H, Kannel W, 2002. *Overweight and Obesity as Determinants of Cardiovascular Risk. Archive of Internal Medicine.* 2002; 162:1867-1872.

Pleis JR, Lucus JW, Ward BW, *2008. Summary health statistics for U.S. adults: National Health Interview Survey.* Vital Health Stat. No. 2009; 240(242);

Profiles of research, 2007. *Heart and Stroke Foundation of Canada.* Retrieved on 2nd April http://www.heartandstroke.com/site/c.ikIQLcMWJtE/b.4194855/k.6388/Profiles_of_research.htm

Rakugu H, Yu H, Kamitani A, Nakamura Y, Ohishi M, Kamide K, Nakata Y, Takami S, Higaki J, Ogihara T, 1995. *Links between hypertension and myocardial infarction.* The American heart journal 1996; 132 (2):(42),(67) 213-221.

Reynolds H R, Hochman J S, 2008. *Post-Myocardial Infarction Cardiogenic Shock. Circulation.* 2008; 117:686-697

Risk Factors and Coronary Heart Disease, n.d. Retrieved on 1st April 2010 from http://www.americanheart.org/presenter.jhtml?identifier=4726

Risk factors for Myocardial Infarction or Heart Attack, 2008. Retrieved on 1st April 2010 http://www.imaginis.com/heart-disease/heartattack.asp

Roguin N, et al. *High prevalence of muscular ventricular septal defect in neonates.* JACC 1995; 26:1545-8;

Sands AJ, et al. *Incidence and risk factors for ventricular septal defect in "low-risk" neonates.* Arch Dis Child Fetal Neonatal Ed 1999; 81(1):F61–F63

SCORE project. *Systematic Coronary Risk Evaluation, a project between 1998 and 2001* initiated by the ESC and European Union BIOMED-2 programmed.

Shirani J, 2008. *Myocardial Rupture.* Retrieved on 2nd April from http://emedicine.medscape.com/article/156455-overview

Statistics, n.d. *Heart and Stroke Foundation of Canada Statistics.* Retrieved on 15th April 2010 from http://www.heartandstroke.com/site/c.ikIQLcMWJtE/b.3483991/k.34A8/Statistics.htm; Accessed 10/10/08)

Statistical fact sheets, 2008. *Trends in cholesterol screening and awareness of high blood cholesterol: United States,* 1991–2003. MMWR Morb Mortal Wkly Rep. 2005; 54: 865–870

Summit medical group, 2009. In *Myocardial Infarction (Heart Attack).* Retrieved on 2nd April from http://media.summitmedicalgroup.com/media/db/

Sutherland SF, 2009. *Pulmonary Embolism.* Retrieved on 1st April 2010 from http://emedicine.medscape.com/article/759765-overview

Swales JD. *Platt versus Pickering. An Episode* in *Recent Medical History*. Cambridge: Keynes Press. British Medical Association, 1985.

Suwaidi JA, Wright RS, Grill JP, Hensrud DD, Murphy JG, Squires RW, Kopecky SL, 2001. *Obesity is associated with premature occurrence of acute myocardial infarction.* Retrieved on 2nd April from Clin Cardiol. 2001; 24(8):542-7.

Tenerz Å, Lönnberg I, Berne C, Nilsson G, Leppert J, 2000. *Myocardial infarction and prevalence of diabetes mellitus. Is increased casual blood glucose at admission a reliable criterion for the diagnosis of diabetes?* European Heart Journal, 2000; 22 :(13)1102-1110.

Teo KK, Ounpuu S, Hawken S, Pandey MR, Valentin V, Hunt D, Diaz R, Rashed W, Freeman R, Jiang L, Zhang X, Yusuf S, 2006. *Tobacco use and risk of myocardial infarction* in 52 countries in the INTERHEART study: a case-control study. Lancet. 2006 Aug 19; 368(9536):647-58.

Thomas DR, Susan R. Heckbert, Robert CK, Nicholas LS, Lemaitre RN, Psaty BM, 2002. *Smoking Status and Risk for Recurrent Coronary Events after Myocardial Infarction.* Annals of Internal Medicine 2002; 137 (6) 494-500

Verdecchia P, Giuseppe S, Claudia B, Antonella A, Ivano Z, Massimo B, Roberto G, Nicola N, Carlo P, 1995. *Cigarette smoking, ambulatory blood pressure and cardiac hypertrophy in essential hypertension.* Journal of Hypertension 1995; 13(10)

WHO, 2009. *Cardiovascular diseases (CVDs).* Retrieved on 2nd April from http://www.who.int/mediacentre/factsheets/fs317/en/print.html

WHO, Tobacco facts, n.d. Retrieved on 7th April 2010 from www.who.int/tobacco/mpower/tobacco_facts/en/print.html

WHO, 2004. *The Atlas of Heart Disease and Stroke.* Retrieved on 7th April 2010 from http://www.who.int/cardiovascular_diseases/resources/atlas/en/

WHO Working Group. The establishment of ischaemic heart disease registers (report of fifth working group). Copenhagen: World Health Organization. *WHO Reg Publ Eur Ser* 1972; 821.

Wilhelmsson C, Vedin JA, Elmfeldt D, Tibblin G, Wilhelmsen L, 1978. *Hypertension and myocardial infarction.* J Chro Dis. 1978; 31(3):157-164.

Wolf MM, Varigos GA, Hunt D, Sloman JG, 1978. *Sinus arrhythmia in acute myocardial infarction.* Med J Aust. 1978; 2(2):52-3

Wong ND, Wilson PF, Kannel WB, 1991. *Serum Cholesterol as a Prognostic Factor after Myocardial Infarction: The Framingham Study.* . Journal of Hypertension, 1991:115(9) 687-693

World No-Tobacco Day, WHO Web site, January 2004. Retrieved on 7th April 2010 from www.who.int/tobacco/communications/events/en/wntd_outcomes_2004.pdf

Yip HK, 2003. "*Cardiac rupture complicating acute myocardial infarction in the direct percutaneous coronary intervention reperfusion era*" (PDF). Chest 124 (2): 565–71.

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
3 = Service
4 = Pension holder7
5. = Unemployed
6. = House Wife
7. = Others (specify).....

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.GheeButter 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