FACTORS INVOLVED IN FIRST MYOCARDIAL INFARCTION ITS COMPLICATIONS AND THROMBOLYTIC PATTERN IN SELECTED HOSPITALS OF NEPAL

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"FACTORS INVOLVED IN FIRST MYOCARDIAL INFARCTION, ITS COMPLICATIONS AND THROMBOLYTIC PATTERN IN SELECTED HOSPITALS OF NEPAL"

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INTRODUCTION

Atherosclerosis is a focal disease of the intima characterized by plaques, which consist mainly of lipid and connective tissue matrix proteins. Atherosclerosis is considered as an immune/inflammatory or healing response of the intima to injury. Premature Atherosclerosis is the term that may be reserved for atherosclerotic events occurring in individuals 45 years of age or younger. The incidence of heart attack in men below 45 years of age was as high as 18%. (Health Information India 1988). Comparable figures from other countries were 5% in USA (American Heart Association 1990), 6% in Australia, 9% in UK, 2% in Japan (Aoki etal 1986). Atherosclerosis is a multifactorial disease. Coronary heart disease (CHD) is the leading cause of death in the world.

World Health Organization [WHO] has predicted that by AD 2020 up to three-quarter of death in developing countries would result from non-communicable diseases (NCDs) and that CHD will top the list of killers. Data also indicate that epidemiological transition, which is characterized by aging and changing life style and culminates in epidemics of hypertension (HTN) and CHD, is rapidly occurring in India and other developing countries. [1,2]

THE EPIDEMIOLOGICAL TRANSITIONS

At the beginning of the 20th century, cardiovascular disease (CVD) accounted for less than 10 percent of all deaths worldwide. At its end, CVD accounted for nearly half of all deaths in the developed world and 25 percent in the developing world. [3,4] By 2020, CVD will claim 25 million deaths annually and CHD will surpass infectious disease as the world's number one cause of death and disability.

This global rise in CVD is the result of a dramatic shift in the health status of individuals around the world over the course of the 20th century. Equally important, there has been an unprecedented transformation in the dominant disease profile, or the distribution of diseases responsible for the majority of death and debility. Before 1900, infectious diseases and malnutrition were the most common causes of death. These have been gradually supplanted in some (mostly developed) countries by chronic diseases such as CVD and cancer thanks largely to improved nutrition and public health measures. As this trend spreads to and continues in developing countries. CVD will dominate as the major cause of death by 2020, accounting for at least one in every three deaths.[4]

This shift in the diseases that account for the lion's share of mortality and morbidity is known as the epidemiological transition.[5,6] The epidemiological transition never occurs ill isolation but is tightly intertwined with changes in personal and collective wealth (economic transition), social structure (social transition), and demographics (demographic trnsition

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CURRENT WORLDWIDE VARIATIONS IN THE GLOBAL BURDEN OF CVD

An epidemiological transition much like the one that occurred in the United States is occurring throughout the world. As in the United States, worldwide CVD rates have risen steadily throughout the 1900s. At the close of the 20th century, 28 percent of all deaths worldwide were due to CVD. Where as communicable diseases (CDs) accounted for 34 percent of the total. [4] With the ongoing global transition dominated by the transition in the developing world-CVD will be the number one cause of death by 2020, accounting for 36.3 percent of all deaths whereas CDs will account for barely half that, at 15.1 percent [4].

Looking behind the global transition reveals vast discrepancies in regional rates of change. These wide variations began to appear early in the 20th century. Although most of the world remained in the phase of pestilence and famine, economic circumstances in several relatively confined regions changed rapidly, accelerating the pace of their epidemiological transitions. Thus, the global burden of CVD is best understood by examining the differential rates of change in each economic region. In addition to variability in the rate of the transition, there are unique regional features that have modified aspects of the U.S.-style transition in various parts of the world.

For most populations, the last century has witnessed the most dramatic improvements in health in history. Life expectancy at birth has increased from a global average of 46 years in 1950 to 66 years in 1998 [7]. The health status and disease profiles of human societies have historically been linked to the level of their economic development and social organization. With industrialization, the major causes of death and disability, in the more advanced societies, have shifted from a predominance of nutritional deficiencies and infectious diseases, to those classified as degenerative [chronic diseases such as cardiovascular disease (CVD), cancer, and diabetes]. This shift has been termed "the epidemiologic transition" [8]. At any given time, different countries in the world or even different regions within a country are at different stages of the epidemiologic transition. This transition can occur not only between different disease categories (eg, deaths from childhood diarrhea and malnutrition giving way to adult chronic diseases), but also within a specific disease category (eg, rheumatic heart disease of the young giving way to chronic coronary artery diseases of middle age or valve calcification, degeneration, and heart failure of the elderly) [9]. (Table 1).

For countries in the earliest stage of development, the predominant circulatory diseases are Rheumatic heart disease (RHD), those due to other infections, and nutritional deficiency related disorders of the heart muscle. Geographic regions experiencing this phase include Sub-Saharan Africa (SSA) and the rural areas of South America and South Asia *(SA)*. During the second stage, as infectious disease burdens are reduced and nutrition improves, diseases related to hypertension, such as hemorrhagic stroke and hypertensive heart disease, become more common. Regions experiencing this phase include China and other Asian countries. During the third stage, as life expectancy continues to improve, high-fat diets, cigarette smoking, and sedentary lifestyles become more common. Noncommunicable diseases then predominate, with the highest mortality caused by atherosclerotic CVD, most frequently ischemic heart disease and atherothrombotic stroke, especially at ages below 50 years. This phase is found in urban India, [10]. Latin America, and the former socialist countries. For most developing and middle-income countries, the increased incidence of CVD adds to the continuing burden of infectious, nutritional, and perinatal diseases, which has been termed the "double-burden" (Table .2) [7]. During the fourth stage, increased efforts to prevent, diagnose, and treat ischemic heart disease and stroke are able to delay these diseases to more advanced ages. The regions that have reached this fourth stage include Western Europe, North America (excluding some parts of Mexico), Australia, and New Zealand.

Previously the fourth stage was considered to be the "final" stage of the epidemiologic transition. However, we propose that a fifth stage be added, where social upheaval or war breaks down existing social and health structures, leading to a resurgence of conditions seen in the first two stages. Diseases of the third and fourth stages persist. This regressive stage is associated with increased deaths due to both cardiovascular (CV) and non-CV causes such as infectious diseases, violence, and consequently a decrease in life expectancy. It is likely that Russia represents such a situation, where in the last 10 years, life expectancy has shortened with a marked increase in deaths from CV diseases, infectious diseases, accidents, and violence [11].

Subsets of the population in a country may be at different stages of the CVD epidemic. An "early-adopter" subset of a community (either part or whole), such as one with rapid social and economic development (such as Mexico), may experience an early increase in CVD [12] and thus have a higher level of CVD than other parts of the population. The decline in CVD burden may also occur earlier for this community. The transition of CVD from being a disease of the wealthy to one of the poor has been documented in the United Kingdom and the United States (US) [13,14]. For example, CVD was relatively rare in the African-American community in the 1960s, but now its incidence equals or exceeds that in the white population of the United States [15]. A similar pattern is appearing in some parts of India [16]. Therefore, the pattern of CVD continues to be in transition in most countries; indeed, it may vary within a country by geography or socioeconomic status and is potentially bi-directional.

TABLE 1. Modified Model of the Stages of Epidemiologic Transition as it Pertains to Cardiovascular Diseases

Stage of Development	Deaths From CVD, % of total deaths	Pridominant CVDs and Risk Factors	Regional Examples
1. Age of pestilence and famine	5-10	Rheumatic heart disease, Infections, and nutritional cardiomyopathies	Sub-Saharan Africa, rural India, South America
2. Age of receding pandemics	10-35	As above + hypertensive heart disease and hemorrhagic strokes	China
3. Age of degenerative and man-made diseases	35-65	All forms of strokes, ischemic heart disease at young ages, increasing obesity, and diabetes	Urban India, former socialist economies, aboriginal communities
4. Age of delayed degenerative diseases	<50	Stroke and ischemic heart disease at old age	Western Europe, North America, Australia, New Zealand
5. Age of health regression and social upheaval	35-55	Re-emergence of deaths from rheumatic heart disease, infections, increased alcoholism, and violence; increase in ischemic and hypertensive diseases in the young	Russia

Global Burden of Cardiovascular Disease

The high current burdens of NCDs are highlighted by the estimates provided by the Global Burden of Disease Study (Table 2) [4] and in the World Health Report 1999 (Table 3), [3] which indicate that these disorders together contributed to 59% of global mortality (31.7 million deaths) and 43% of the global burden of disease in 1998. Several NCDs such as cardiovascular diseases (CVD), cancers, diabetes, and chronic obstructive pulmonary disease are linked by common lifestyle determinants such as diet, physical activity, and tobacco consumption. These four disorders together contribute to about 50% of global mortality. Because these conditions tend to affect individuals in middle and old age, they account for a smaller proportion (19%) of the global burden of disease. It is estimated that 30.9% of all deaths in 1998, as well as 10.3% of the total disease related burden, in terms of disability adjusted life year loss (DALY loss) were attributable to CVD (Table 3). [3]

TABLE 2. Deaths (In Thousands) Due to Cardiovascular Disease and to Infectious and Parasitic Diseases in 30- to 69- Year-Olds by Sex and Region in 1990

	Men		Women	
Region	CVD	IPD	CVD	IPD
EME	483	42	227	12
FSE	416	20	253	6
India	611	429	481	240
China	576	158	439	89
OAI	289	147	226	140
SSA	183	215	211	228
LAC	186	62	147	48
MEC	285	56	215	35
Worldwide	3,028	1, 128	2,201	798

EME indicates established market economics; FSE, formerly socialist economies; OAI, other Asian and Pacific Island countries; SSA, Sub-Saharan Africa; LAC, Latin American/Caribbean; and MEC, Middle East Crescent.

The Growing Burden of CVD in the Developing (Low and Middle Income) Countries

The World Health Report 1999 estimates that in 1998, 78% of the burden of NCDs and 85% of the CV burden arose from the low and middle income countries (Table 3). The CVD burden afflicts both men and women, with CV deaths accounting for 34% of all deaths in women and 28% in men in 1998.[3] As the epidemics advance, the social gradient also reverses with the poor becoming the most vulnerable victims in both developed and developing countries. [17]

The high burdens of CVD in the developing countries are attributable to the increasing incidence of atherosclerotic diseases, perhaps due to urbanization and higher risk factor levels (such as obesity, diabetes, dyslipidemia, hypertension, etc), the relatively early age at which they manifest, the large sizes of the population, and the high proportion of individuals who are young adults or middle-aged in these countries. For example, about half of the deaths attributable to CVD in the developing countries in 1990 occurred below the age of 70 years, in contrast to about a quarter in the developed countries [4]. Such a pattern of premature CVD mortality is likely to haunt the developing countries even more in the future. Between 1990 and 2020, the increase in ischemic heart disease (IHD) mortality (120% in women and a 137% in men) in the developing countries is expected to be much greater than among developed countries (29% and 48%, respectively) (Table 4) [4]. A similar pattern for increases in cerebrovascular disease mortality is predicted (124% and 107%, increases among men and women in developing countries versus 78% and 56% increases, respectively, in the developed countries). These projections are largely based on the expected changes in the demographics of the population and do not account for potential increases in risk factor levels [10,18]. However, with urbanization and changing lifestyles the number of people with diabetes, obesity, dyslipidemia, or high blood pressure may increase, suggesting that the increases in CVD based purely on demographic shifts are likely underestimates.

TABLE 3. Contribution of Noncommunicable Diseases and Various Individual Diseases to the Global Mortality and Global Burden of Disease* in 1998, Subdivided by Low Income Countries and Middle Income Countries

Disease Category	Contribution of NCD to Total Global Mortality, %	Contributions of LIC+MIC to Global NCD Mortality, %	Contribution of NCD to Total Burden of Disease, %	Contributions of LIC+MIC to NCD Burden of Disease, %
Total NCD	58.8	77.8	43.1	85.3
Total CVD	30.9	78.5	10.3	86.3
Total cancers	13.4	72.1	5.8	79.9
Diabetes	1.1	73.2	0.8	73.2
COPD	4.2	87.5	2.1	91.4

LIC indicates low income country; MIC, middle income country; NCD, non-communicable disease; and COPD, chronic obstructive pulmonary disease.
Data are derived from estimates provided in the World Health Report 1999 [3].
*Burden of disease calculated as Disability-Adjusted Life-Years. .

Risk Factors for Atherosclerotic Cardiovascular Disease

A large body of epidemiologic studies has clearly demon started a link between certain risk markers and CVD. These can be classified into two categories: (I) those that have been proven to be causal (risk factors), and (2) those that show associations with CVD but for whom a cause and effect association is yet to be proven (risk markers). These markers could be classified as predisposing (eg, obesity which may work through raising blood pressure, glucose, and lipids) or direct (e.g., smoking).

Ecological, case-control, and cohort studies in many populations have identified a number of markers, which are associated with either an increased, or a decreased risk of CVD. Whether or not these associations are causal is decided by applying several criteria. These include the strength and consistency of association, temporal relationship, dose response relationship, biologic plausibility, experimental evidence, and very importantly, concordant evidence from randomized human trials when available. It is the coherence of information from several different types of studies, which has led to our body of knowledge and provides persuasive evidence of the causal link of several risk factors with CVD.

It has been suggested that conventional risk factors only explain about half of the variance in CHD [18]. However, there may be several reasons why the role of conventional risk factors like tobacco, cholesterol, and high blood pressure may have been substantially underestimated.

TABLE 4. Estimate of Ischemic Heart Disease Mortality (Thousands) by Region and Sex and Projected Changes between 1990 and 2020

	Women			Men		
Region	1990	2020	% Increase	1990	2020	% Increase
EME	838	1107	32	829	1209	46
FSE	559	702	26	468	712	52
Total developed countries	1397	1809	29	1297	1921	48
India	556	1197	115	619	1405	127
China	377	684	81	386	811	110
OAI	227	552	143	233	581	149
SSA	117	263	125	92	222	141
Latin America	169	412	144	179	444	148
Middle East	291	717	146	319	874	114
Total developing countries	1737	3825	120	1828	4337	137
World	3134	5634	80	3125	6258	100

Abbreviations as in Table 2.

Proven and Putative Risk Markers for Cardiovascular Diseases

Risk factors that are causally linked:

1. Tobacco consumption
2. Elevated LDL
3. Low HDL
4. High blood pressure
5. Elevated glucose
6. Physical inactivity*
7. Obesity*
8. Diet*

Risk markers that show associations.

1. Low socioeconomic status*
2. Elevated prothrombotic factors. fibrinogen, PAI-1
3. Markers of infection or inflammation
4. Elevated homocysteine
5. Elevated lipoproteln(a)
6. Psychologicai factors (depression, anger proneness, hostility, stress, acute life-events) and breakdown in social structures (loss of social support and cohesion)*

*Predisposing risk factors: A predisposing risk factor is presumed to work, at least in part, through an impact on other risk factors that act directly. For example, obesity raises blood pressure, causes dyslipidemia, and increases blood glucose. It is likely that some of the predisposing risk factors also have direct effects.

PAI indicates plasminogen activator inhibitor.

South Asians

South Asian (SA) refers to people who originate from India, Sri Lanka, Bangladesh, Nepal, and Pakistan.

Disease Burden

There are relatively few mortality studies from India, as there is no uniform completion of death certificates and no centralized death registry for CVD [16]. However, the WHO and the World Bank estimate that deaths attributable to CVD have intreased in parallel with the expanding population in India, and that CVD now accQunts for a large proportion of disability adjusted life years (DAL Y) lost [4]. Of all deaths in 1990, approximately 25% were attributable to CVD, com- pared with 10% from diarrheal diseases, 13% from respiratory infections, and 8% from tuberculosis [4]. SA migrants to the United Kingdom, South Africa, Singapore, and North America experience 1.5 to 4.0 times higher CHD mortality compared with indigenous populations [20].

Temporal Trends

In India, the CHD rate is expected to rise in parallel with the increase in life expectancy secondary to increases in per capita income and declining infant mortality. The average life expectancy has increased from 41 years in the years 1951 to 1961, to 61.4 years in the years 1991 to 1996 and is projected to reach 72 years by 2030, which could lead to large increases in CVD prevalence [10]. By contrast, in the UK and Canada, although the CHD mortality rate of SAs compared with other populations remains high, a decline in CHD rates has been observed over the past 10 years. [21,19]. These data indicate that the high rates of CHD with economic changes are reversible and perhaps even avoidable. Therefore, lessons learnt from migrant SAs may be helpful in developing prevention strategies for the Indian subcontinent.

Risk Factors

Compared with Europeans, SAs (in the UK and Canada) do not display high rates of smoking, HTN, or elevated cholesterol but still have higher rates of CHD. [19,22,23]. However, smoking, HTN, and diabetes mellitus (DM) are strongly associated with CHD among SAs [24]. SAs in the UK and Canada suffer a high prevalence of impaired glucose tolerance (IGT), central obesity, elevated triglycerides, and low HDL cholesterol, [22,25]. and NIDDM at rates 4 to 5 times higher than in Europeans (19% versus 4% by age 55 years) [23,25]. High rates of diabetes has been reported among SAs in the UK (10% to 19%), Trinidad (21%), Fiji (25%), South Africa (22%), Mauritius (20%), and Canada (10%) [26,22]. By contrast, the prevalence of diabetes in rural India is 2% to 3% and approximately 8% in urban area [27]. In addition, there is increasing evidence that elevations in blood glucose even in the nondiabetic range increases CHD risk among Sas [24]. SAs have elevated levels of Lp(a), a lipoprotein which is genetically mediated and associated with increased atherosclerosis, thrombogenesis, and clinical events [22,28]. Recent studies have confirmed that SAs also have higher levels of homocysteine, fibrinogen, and plasminogen activator inhibitor (PAI-l), [22]. All of which could increase the risk for thrombosis. Although the degree of subclinical atherosclerosis is related to clinical events, it appears that SAs have a higher propensity for clinical events compared with Europeans or Chinese, even after adjusting for all known risk factors and the degree of atherosclerosis [22]. This probably suggests the potential for greater plaque rupture and thrombotic events among SAs.

Geographic Variations

Marked increases in both CHD prevalence and risk factors are observed in urban India compared with rural setting [1]. A recent overview of prevalence surveys conducted over 2 decades in India reported a 9-fold increase of CHD in urban centers, compared with a 2-fold increase in CHD rates among rural populations [1]. This increase in CHD rates in urban areas is associated with an increase in the prevalence of lipid and glucose abnormalities as well as HTN and obesity. By contrast, the rates of tobacco smoking are higher in rural compared with urban populations. (Although these studies used somewhat different methods of sampling and varying definitions for CHD, collectively, they suggest that there is likely a real increase in CHD; however, the magnitude of the increase remains uncertain).

Migration Patterns

SAs in the UK have higher risk factor levels compared with their siblings living in India (BMI, 27 versus 23 kg/m2; systolic BP, 144 versus 137 mm Hg; total cholesterol, 6.3 versus 5.0 mmol/L; lower HDL cholesterol, 1.14 versus 1.27 mmol/L; and higher fasting glucose, 5.4 versus 4.6 mmol/L) [29]. Therefore, adverse changes in CVD risk factors and disease rates are observed when South Asians adopt an urban lifestyle whether they live in India or abroad.

Materials and Methods

This is a multi centric prospective observational study with five centers in the Kathmandu valley. The present study consists of 213 cases of first acute myocardial infarction (MI) seen in the National Academy of Medical Sciences (NAMS) Bir Hospital, Tribhuvan University Teaching Hospital (TUTH), Medicare National Hospital and Research Center (Medicare), Norvic Escorts International Hospital (Norvic) and in The Sahid Gangalal National Heart Center (SGNHC) during the period of 1 year (1 October 2001 to 30 September 2002). The patients are mainly from the lower and middle classes of society in the Bir Hospital, TUTH and in SGNHC While patients coming to the Medicare Hospital and NORVIC Hospital are mainly from the middle and upper classes. Peoples are of various regions and multi ethnic in the Kathmandu valley. So this group should form a representative sample of first acute MI in Nepal.

Inclusion Criteria

Both sexes and all age group patients of suspected first acute MI were included in this study.

Exclusion Criteria

Patients of unstable angina, Old MI and second and subsequent MI were not included in this study.

MI Diagnosis

Cases of STelevation myocardial infarction (STEMI) and Non STelevation myocardial infraction (NSTEMI) were diagnosed on the basis of The World Health Organization (WHO) criteria. According to WHO definition, the diagnosis of MI is based on the presence of at least two of the following three criteria.

- A clinical history of ischemic type chest discomfort.
- Changes on serially obtained electrocardiographic tracings and
- A rise and fall in serum cardiac markers [30,31].

Diabetes mellitus diagnosed on the presence of at least one of the followings.

- Diagnosed case of diabetes mellitus on diet control and / or taking anti diabetic drugs.
- Abnormal blood sugar during hospital stay based on WHO criteria

Diagnostic criteria of diabetes mellitus using an oral glucose (75 g.) tolerance test (WHO 1985)

TABLE 5. Venous Plasma (mmol/L)

	Normal	Diabetic
Fasting	<6.1	>=7.8
2 hours after glucose	<8.9	>=11.1

Hypertension was diagnosed on presence of at least one of the followings

- Known case of hypertension on anti hypertensive drugs.
- When systolic blood pressure was more than 140 mm of Hg and / or diastolic blood pressure more than 90 mm of Hg (JNC V) [32].

Smoker

Those patients who were smoking at the time of presentation labeled as smokers.

Positive Family History

CHD in a male relative with onset at age 55 or less or a female relative with onset at age 65 or less is defined as a positive family history [33].

All the cases were closely interrogated examined and investigated. Serial electrocardiogram (ECG) and cardiac enzymes were done. Blood sugar done in all cases and lipid profile in 148 cases.

Result

Age distribution:

Total Patients included in the study were 213. Ages of two patients could not be determined. The youngest patient was 29 years while oldest patient aged 84 years. Mean age of first MI was 57 ± 11 years. Younger patients (<=45 years) were 42(19.7%)

TABLE 6.

Age (years)	Frequency	Percent
21-30	2	0.9
31-40	13	6.2
41-50	50	23.5
51-60	74	34.7
61-71	45	21.1
71-80	26	12.2
81-90	1	0.5
Total (N)	211	99.1
Missing System	2	0.9
Total	213	100.0

TABLE 7. Age distribution of the Patients aged <=45 years.

Sex	Frequency	Percent
Male	33	15.5
Female	9	4.2
Total	42	19.7
Male: Female	33:9	3.7:1

Sex Incidence

Sex analysis done in 213 patients. Male patients were 157 (73.7%) and female patients were 56(26.3%). Male and female ratio was 2.8:1.0

Age and Sex correlation

TABLE 8. Grouped age (Male)

Age (Years)	Frequency	Percent
21-30	2	1.3
31-40	7	4.5
41-50	39	24.8
51-60	51	32.5
61-71	36	22.9
71-80	19	12.1
81-90	1	0.6
Total (N)	155	98.7
Missing System	2	1.3
Total	157	100.0

TABLE 9. Grouped age (Female)

Age (Years)	Frequency	Percent
31-40	6	10.7
41-50	11	19.6
51-60	23	41.1
61-71	9	16.1
71-80	7	12.5
Total (N)	56	100.0

TABLE 10. Risk Factors analysis.

Risk factors	Frequency	Percent
1. Smoking	154	72.3
2. Hypertension (HTN)	94	44.1
3. Diabetes Mellitus (DM)	62	29.1
4. Smoking + HTN	64	30.10
5. Smoking + DM	40	18.8
6. HTN +DM	34	16.00
7. Smoking + DM + HTN	20	9.4
8. Positive family history	44	20.7

Lipid profile

TABLE 11. TOTAL SERUM CHOLESTEROL (mg %)

Mg %	N	Minimum	Maximum	Mean	Std. Deviation
Total serum cholesterol	148	89.01	368.00	202.3552	43.1197

Mg % Total serum cholesterol	Frequency	Percent
<=150	14	9.46
151-200	51	34.46
>=201	83	56.08
Total (N)	148	100.00

TABLE 12. SERUM TRIGLYCERIDE (mg %)

Mg %	N	Minimum	Maximum	Mean	Std. Deviation
Serum triglyceride	148	56.00	517.08	226.4842	98.9458

Mg % Serum triglyceride	Frequency	Percent
<=150	37	25.0
151-200	40	27.0
>=201	71	48.0
Total (N)	148	100.0

TABLE 13. SERUM LOW DENSITY LIPOPROTEIN (mg %) (LDL)

Mg %	N	Minimum	Maximum	Mean	Std. Deviation
serum low density lipoprotein	148	38.00	268.00	123.9792	35.8367

Mg % serum low density lipoprotein	Frequency	Percent
<=150	30	20.27
151-200	65	43.92
>=201	53	35.81
Total (N)	148	100.0

TABLE 14. SERUM HIGH DENSITY LIPOPROTEIN (mg %) (HDL)

Mg %	N	Minimum	Maximum	Mean	Std. Deviation
Serum high density lipoprotein	148	19.35	168.00	42.9928	14.4518

Mg % Serum high density lipoprotein	Frequency	Percent
<=35	41	27.70
36- 40	33	22.30
41- 50	44	29.73
>=51	30	20.27
Total (N)	148	100.00

Types of MI

Out of 213 cases STEMI were 192 (90.14 %), NSTEMI were 19 (8.92%) and New LBBB were 2 (0.93%). Types of STEMI are shown in Table 15.

TABLE 15. STEMI distribution 192 = (90.14 %)

STEMI types	Frequency	Percent
Anterior	96	45.07
Inferior	71	33.33
Inferior +Right ventricle	7	3.28
Inferior + Anterior	5	2.34
Lateral	5	2.34
Inferior + Lateral	4	1.87
Inferior + Lateral + Posterior	2	0.94
Inferior + Posterior	2	0.94

Time between symptom onset and Presentation to emergency room

Patients came to hospitals after onset of chest pain at different time intervals.

TABLE 16.

Home to center time (Hours)	Frequency	Percent
Within 2 hours	36	16.9
Within 6 hours	90	42.3
Within 12 hours	117	54.9

	Minimum	Maximum	Mean	Std. Deviation
Home to center time (Hours)	0.50	528.00	35.0948	63.2432

Thrombolytic pattern

Total number of STEMI and new LBBB cases were 194 (91.07 %) but thrombolysis done in 95 (44.60%) cases only.

Time between presentation to emergency room and start of thrombolysis (Door to Needle time)

Thrombolysis done in 95 (44.60%) patients but 40 (18.78%) cases only Thrombolysed within 30 minutes.

TABLE 17.

	Minimum	Maximum	Mean	Std. Deviation
Door to Needle time (Houurs)	0.10	11.00	1.1210	1.2761

Killip classes at Presentation

TABLE 18.

S.N.	Killip classes	Frequency	Percent
I	No signs of Congestive Heart failure (CHF)	117	54.9
II	S3 gallop and bibasillar rales	53	24.9
III	Acute pulmonary oedema	23	10.8
IV	Cardiogenic shock	20	9.4
	Total	213	100.0

Complications of first MI

TABLE 19.

Arrhythmias		Frequency	Percent
	Ventricular		
	Extra systole (VPCs)	87	40.84
	Ventricular tachycardia (VT)	16	7.51
	Ventricular fibrillatron (VF)	8	3.75
	1. Atrial		
	Extrasystole (APCs)	25	11.74
	Atrial fibrillation (AF)	5	2.35
	Paroxysmal supraventricular tachycardia (PSVT)	2	0.94
	conduction defects		
	First degree atrioventricular block	18	8.45
	Second degree atrioventricular block	11	5.16
	Complete heart block (CHB)	14	6.57
	Right bundle branch block (RBBB)	2	0.94
	Left bundle branch block (LBBB)	2	0.94
	Trifascicular block	3	1.4
Left ventricular thrombus		1	0.47
Cerebrovascular accident (CVA)		2	0.94
Percarditis		3	1.4
Ventricular septal defect (VSD)		2	0.94

In Hospital Mortality

Total numbers of death were 16 (7.5%). Male cases were 11 (5.16%) and female cases were 5 (2.35%).

TABLE 20. Grouped age (Death)

Age Group (Years)	Frequency	Percent
31-40	2	12.5
36- 40	2	12.5
41- 50	5	31.3
61-70	5	31.3
71 - 80	2	12.5
Total	16	100.0

TABLE 21. Mortality And Sex Correlation (Death)

Age Group (Years)	Frequency	Percent
Male	11	68.8
Female	5	31.3
Total	16	100.0

Total Hospital Stay

TABLE 22.

Days	Frequency	Percent
1	2	1.4
2	11	7.6
3	14	9.7
4	22	15.3
5	28	19.4
6	21	14.6
7	12	8.3
8	8	5.6
9	4	2.8
10	4	2.8
11	9	6.3
12	3	2.1
14	2	1.4
15	1	0.7
16	2	1.4
21	1	0.7
Total	144	100.0

Days	N	Minimum	Maximum	Mean	Std. Deviation
Hospital Stay	144	1	21	6.28	3.32

TABLE 23. CCU Stay

Days	Frequency	Percent
1	2	1.4
2	12	8.3
3	22	15.3
4	31	21.5
5	33	22.9
6	20	13.9
7	9	6.3
8	5	3.5
9	1	0.7
10	2	1.4
11	3	2.1
12	1	0.7
15	1	0.7
16	1	0.7
21	1	0.7
Total	144	100.0

Days	N	Minimum	Maximum	Mean	Std. Deviation
CCU	144	1	21	5.07	2.72

TABLE 24. Ward / Cabin stay

Days	N	Minimum	Maximum	Mean	Std. Deviation
Ward Cabin	30	1	10	4.83	2.56

Discussion

Age distribution

The majority of cases 124 (58.2%) belonged to the age group 41-60 years. The number of cases in sixth decade 74 (34.7%) being higher than in the fifth decade 50 (23.5%). Similar maximum incidence in the sixth decade has been reported by Pandey in Nepal, Banerjea, Padmavati and Subramanian, in India [34,35,36,37]. Younger patients had suffered MI more (19.7%) than what seen in other parts of the world.

Sex incidence

All the reports published related to MI there has been a striking preponderance of males. But Male and female ratio has decreased significantly in this study (2.8:1.0) than in past (6.5:1.0) due to increased number of female cases. Male 48 (30.6%) and female 17 (30.4%) are equally affected below the age of 50 years. In the past study male affected more than female below the age 50.[34]

Smoking

Commonest risk factor for CHD was smoking 154(72.3%) in this study. While Pandey (1970) found 81% smoker smoking habit has decreased by 9%. This trend can be taken positively to decrease present burden of CHD. But still much have to be done to decrease smoking habit significantly.

Hypertension:

94 (44.1%) found to be hypertensive. Incidence of HTN has increased by 16%. Pandey, found the incidence of HTN 28 %, Vakil 33.1 % and white & bland 25% [34,38,39].

Diabetes Mellitus

It was found in 62 (29.1%). DM has increased by 15%. Other workers have found the incidence of DM as follows in their series: Pandey 14.3%, Banerjee 18.3%, Lal 25.9%, and Subramanian 7%. [34,35,40,37].

Family History

Family history positive for CHD was found in 44 (20.7%) while in past it was only 16% (Pandey 1970). This data also reflect increasing burden of CHD

Lipid Profile

148 cases of first MI were analyzed for lipid profile. Total serum cholesterol was found abnormal (>200mg %) in 83 (55.7%) triglyceride was found to be above 150 mg % in 111(75%) 52(35.1%) had LDL above 130mg % while 73 (64.2%) patients had HDL less than 40 mg %.

Types of MI

Out of 213 cases 192 (90.14%) were STEMI. Among STEMI Anterior wall MI was most common 96(45.0%), inferior wall with right ventricular infarction 7(3.28 %) and inferior MI with posterior wall infarction 2 (0.94 %). In past study also anterior wall MI was commonest (Pandey 1970)

Home to center time

Only 36 (16.9 %) of patients reached center within 2 hours after onset of chest pain and 117 (54.9%) reached within 12 hours.

Door to needle time

Thrombolysis had done in 95(44.6 %) patients. Only 40 (18.78 %) cases were thrombolysed within 30 minutes.

Killip Classes

Most of the cases presented with killip classes I and II. 20 % cases presented with class III and IV.

Complications

Commonest complication was ventricular arrhythmia 111(52.1%) and conduction abnormalities found in 50 (23.47 %), complete heart block in 14(6.57%) and VSD found in 2 (0.94%) cases.

In hospital mortality

Combined in hospital mortality were 16(7.5%). Most of death 10 (4.69%) occurred in 51-70 age group. Increased death rate was seen in male patients 11(5.168%). Death occurred in younger age group (<=45 years) was only one.

Hospital Stay

Average CCU stay was 5 days while maximum stay was 21 days. Average stay was usually longer in CCU. That increased the cost of hospital stay. Uncomplicated inferior wall MI can be transferred from CCU to ward after 48 hours and anterior wall after 72 hours without affecting hospital mortality rate. Average ward stay was 4.8 days.

Conclusion

- Mean age of first MI was 57 years.
- 58.2 % of cases occurred in the age group 41-60 years. The highest incidence being in the Sixth decade.
- Younger (<=45yrs) population had suffered MI more (19.7%) than what seen in other parts of the world.
- The disease was found to affect males 2.8 times, more than females. The numbers of females are more than in the past study.
- Still commonest CHD risk factor was smoking (72.3%).
- HTN (44% versus 28%) and DM (29 % versus 14%) have increased.
- 83 (38.97%) cases had total serum cholesterol more than 200mg.%, while abnormal serum Triglyceride (>150mg) was found in 111 (52.11%) .
- Anterior wall MI is 1.4 times commoner than inferior wall.
- Only 17% of MI patients came within 2 hours to hospital after chest pain.
- Only 95 (44.6%) of STEMI were thrombolysed and 40 (18.78%) were thrombolysed within 30 minutes after coming to emergency.
- Most common complication was ventricular arrhythmia, CHB was found in 14 (6.57%) and VSD in 2 (0.94%)
- In Hospital mortality was 7.5%.
- Incidence of MI had increased at least 8 times over the past four decades.

Framework Recommendations

Policy recommendations

- CHD should be considered as one of the major health problem now and in near future.
- CHD prevention should be seen as being synergistic with poverty reduction strategies, and addressed in development initiatives.
- Policy changes should integrate heart health with communicable disease, reproductive health and population control programmes in an attempt to create cost and time effective opportunities for prevention.
- Polices relation to food and nutrition should ensure availability, favorable pricing, and labeling of heart healthy food.

Programme recommendations

Public health

- As the target population is at the prime of his or her life (5^{th} and 6th decades), MI has huge impact on health and economy of the country. Therefore Cardiovascular disease prevention should be integrated with primary health care.
- Patients have poor knowledge regarding prevention of CHD and should be educated about CHD risk factors through mass media like radio, newspaper and television. Health personnel at primary and tertiary health service level should also educate people regarding prevention of CHD
- Patient should be informed about symptoms and signs of MI as they can come to hospital immediately after onset of chest pain
- The public health approach should target population-wide lifestyle interventions, population-wide screening for high blood pressure and screening of the high-risk group for diabetes and hypercholestrolaemia.
- Lifestyle advice should center on tobacco use cessation, weight control, a heart healthy diet, physical activity and stress management.
- Cardiovascular health should be addressed in school based health education and/or as part of the science curriculum.
- Infrastructure support and local capacity building for research should be prioritized.

Clinical

- There should be provision for thrombolysis at district and zonal hospital level also. Doctors should know the importance and technique of thrombolytic therapy.
- "Cardiology update" programme should be conducted regularly at central level for physicians working at periphery.
- The availability of effective and affordable drugs, devices and procedures should be ensured.
- Referral chains should be established which should provide effective links between primary, secondary and tertiary health care centers whenever required.
- If possible coronary angiography (CAG) and percutaneous coronary intervention (PCI) services should be extended at least to regional level for managing complicated MI.

References:

1. Gupta R., Gupta v.p., Meta-analysis of coronary heat disease prevelence in Inida. Indian Heart J., 1996;48:241-5.
2. Gupta R. Singhal s., Coronary heart disease in India. Circulation, 1997;96:3785.
3. World Health Report 1999: Making a difference. Genera. world Health Organization, 1999
4. Murray CJL, Lopez AD, The Global Burden of Disease. cambridge, MA. Harvard School of public Health, 1996
5. Omran AR. The epidemiologic transition. A theory of the epidemiology of population change: Milbank mem fund Q 49:509-538,1974
6. olshansky SJ, Auit AB. The fourth stage of the epidemiologic transition: The age of delayed degenerative diseases. Milbank Q 64: 355-391, 1986.
7. Sen K, Bonita R. Global health status: two steps forward, one step back. Lancet. 2000;356:577-582.
8. Omran AR. The epidemiologic transition: a theory of the epidemiology of population change. Millbank Memorial Fund Q. 1971;49:509-538.
9. Pearson TA, Jarnison DT, Trejo-Gutierrez H. Cardiovascular disease. In:Jamison DT, ed. Disease Control Priorities in Developing Countries. New York, NY: Oxford University Press; 1993:577-599.
10. Reddy KS, Yusuf S. Emerging epidemic of cardiovascular disease in developing countries. Circulation. 1998;97:596-601.
11. Leon D, Chenet L, Shkolnikov VM, et al. Huge variation in Russian mortality rates 1984-1994: artefact, alcohol, or what? Lancet. 1997:350. 383-388.
12. Bobadilla JL, Frenk J, Lozano R, et al. The epidemiologic transition and health priorities. In: Jamison DT, ed. Disease Control Priorities in Developing Countries. New York, NY: Oxford University Press: 1993:351-356.
13. Kaplan GA, Keil JE. Socioeconomic factors and cardiovascular disease: a review of the literature. Circulation. 1993;88: 1973-1998.
14. Marmot MG, Kogevinas M, Elston MA. Socioeconomic status and disease. WHO Reg Publ Eur Ser. 1991;37:113-146.
15. National Heart. Lung. and Blood Institute Fact Book. Fiscal Year 1999 USA. Bethesda, Md: National Institutes of Health; 1999.
16. Reddy KS. Cardiovascular disease in India. World Health Stat Q. 1993; 46:101-107.
17. Popkin BM. The nutrition transition and its health implication in lowerincome countries. Public Health Nutr. 1998;1:5-21.
18. Chockalingam A. Balaguer-Vinto I, eds. Impending Global Pandemic of Cardiovascular Diseases: Challenges and Opportunities for the Prevention and Control of Cardiovascular Diseases in Developing Countries and Economies in Transition. World Heart Federation. Barcelona: Prous Science; 1999.
19. Sheth T, Nair C, Nargundkar M, et al. Cardiovascular and cancer mortality among Canadians of european, south Asian and Chinese origin from 1979 to 1993: an analysis of 1.2 million deaths. Can Med Asoc J.1999;161:132-138.
20. Enas EA. Yusuf S. Mehta I. Prevalence of coronary artery disease in Asian Indians. Am J Cilrdiol. 1992;70:945-949.
21. Balarajan R. Bthnicity and variations in mortality from coronary heart disease. Health Trends,. 1996;28:45-51.
22. Anand SS. Yusuf S. Vuksan V. et al. Differences in risk factors, atherosclerosis, and cardiovascular disease between ethnic groups in Canada: the Study of Health Assessment and Risk in Ethnic groups. Lancet 2000;356:279-284.

23. McKeigue PM, Ferrie JE. Pierpoint T, et al. Association of early-onset coronary heart disease in south Asian men with glucose intolerance and hyperinsulinemia. Circulation. 1993;87: 152-161.
24. Pais P. Pogue J, Gerstein H. et at. Risk factors for acute myocardial infatttion in Indians: a case-control study. Lancet. 1996;348:358-363.
25. McKeigue PM, Shah B, Marmot MG. Relation of central obesity and insulin resistance with high diabetes prevalence and cardiovascular risk in south Asians. Lancet. 1991;337:382-386.
26. McKeigue PM, Miller GI, Mam1Ot MO. Coronary heart disease in South Asians overseas: a review. J Clin Epidemiol. 1989;42:597-609
27. Ramachandran A, Dharmaraj D, Snehalatha C, et al. Prevalence of glucose intolerance in Asian Indians. Diabete, Care 1992;15:1348-1355.
28. Anand S. Enas. E. Pogue J. et al. Elevated Lipoprotein (a) Levels in South Asians in North America. Metabolism. 1998;47:182-184.
29. Bhatnagar D. Anand IS, Durrington PN, et al. Coronary risk factors in people from the Indian subcontinent living in west London and their siblings in India. Lancet. 1995;345:405-409.
30. Gillum RF, Fortmann SP, Prineas RJ, Kottke TE. International diagnostic criteria for acute myocardial ifrarction and acute stoke , AM Heart J. 1984;108:150-158.
31. Tunstall-pedoe h, Kuulasmaa k, Amouyel p, et al. Myocardial infarction and coronary deaths in the world Health organization MONICA project Circulation. 1994;318:797-803.
32. Joint National committee on detection evaluation and treatment of High blood pressure. The fifth report of the joint National committee on detection evaluation and treatment of High blood pressure (JNC V). Arch intern med 1993 153:154-183
33. Expert panel on detection, evaluation and treatment of high blood cholesterol in Adult (Adult treatment panel I) Circulation 1994; 89: 1329-1445.
34. Pandey MR, Indian Heart J., 73-82, 1970.
35. BANERJEA. J. C.--Indian Heart J., 5: 9t, 1953.
36. PADMAVATI, S. et al-Am. Heart J., 19: 849, 1959.
37. SUBRAMANIAM, R. R. and PADMANABHAN, c.-Indian Heart (J., 16: 21O, 1964.
38. VAKIL, R. J. - Indian Heart J., 1: 201, 1949.
39. WHITE, P. D.-Heart Disease. Edition 4th, The Macmillan Co., New York, 1951.
40. LAL et al-Indian Heart J. 19: 12,1967.