Cardiovascular Risk Factors in Elderly Normolipidaemic Acute Myocardial Infarct Patients

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1. Introduction

Cardiovascular diseases (CVD) are a group of disorders of the heart and blood vessels, and include coronary heart disease (CHD), cerebrovascular disease (stroke), raised blood pressure (hypertension), peripheral artery disease, rheumatic heart disease, congenital heart disease and heart failure. According to the World Health Organization, CVD are the number one cause of death globally and claim 17 million lives each year. By 2030, almost 24 million people will die from CVD, mainly from heart disease and stroke. These are projected to remain the single leading causes of death (World Health Organization. Cardiovascular diseases (CVDs). Available from: http://www.who.int/mediacentre/factsheets/fs317/en/index.html). In the United States, CVD account for more than one-third (34.3%) of deaths annually, and responsible for nearly 3 million Americans reporting disability. The costs of CVD are also staggering. In 2010, the total cost including health care services, medications and lost productivity, is estimated to be over $503 billion in the United States (Centers for Disease Control and Prevention. Heart Disease and Stroke Prevention. Addressing the nation’s leading killers: at a glance 2010. Available from: http://www.cdc.gov/chronicdisease/resources/publications/AAG/dhsp.htm). Similarly, the National Heart Foundation of Australia reported that CVD are the leading cause of mortality and morbidity in Australia, killing one person nearly every 10 minutes (National Heart Foundation of Australia. Data and statistics. Available from: http://www.heartfoundation.org.au/information-for-professionals/data-and-statistics). Despite improvements over the last few decades, CVD remain as the second largest disease burden to our society after cancers. As the population ages, the economic impact of CVD on the health care system will become even greater. Tobacco smoking, an unhealthy diet, physical inactivity and high alcohol consumption increase the risk of CVD. Indeed, behavioral and dietary risk factors are responsible for about 80% of coronary heart disease and cerebrovascular disease (World Health Organization. Cardiovascular diseases (CVDs). Available from: http://www.who.int/mediacentre/factsheets/fs317/en/index.html).

Interestingly, both the incidence and mortality rate of CVD are much lower in Japan than other countries (Mozaffarian D) which may be attributed to the high consumption of...
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fish/seafood by the Japanese population (Meyer BJ). The fact that fish is abundant in omega-3 polyunsaturated fatty acids has opened an effective venue to the prevention and treatment of this disease by either dietary modifications or pharmacological supplementation.

Cardiovascular disease is multi-factorial which is associated with factors like hereditary, hyperlipidemia, obesity, hypertension, environmental and lifestyle variables like stress, smoking, alcohol consumption, etc (Chopra and Wasir 1998). Lipoprotein profile has been investigated extensively in recent years, which is deranged in large proportion of coronary artery disease (CAD) patients; especially Asians showing a mixed picture of dyslipidemia (Vasisht et al, 1990). Literature survey reveals dyslipidaemic subjects are more prone to myocardial infarction, due to increased free radical generation and ischemia as it is a conventional risk factor. (Malhotra et al, 2003; Mishra et al, 2005; Ghosh et al, 2006; Patil et al, 2007; Rajasekhar et al, 2004; Rani et al, 2005; Gomez et al, 1996). Lowering of high density lipoprotein-cholesterol (HDL-C) is a common phenomenon observed in MI patients supported by previous studies (Malhotra et al, 2003; Mishra et al, 2005; Ghosh et al, 2006; Patil et al, 2007; Rajasekhar et al, 2004; Rani et al, 2005). High density lipoprotein-cholesterol (HDL-c) is the most important independent protective factor for arteriosclerosis which underlies coronary heart disease (CHD). High density lipoprotein-cholesterol (HDL-c) associated paraoxonase-1 (PON1) enzyme is protective against lipid peroxidation (Singh et al, 2007). Numerous cohort studies and clinical trials have confirmed the association between a low high density lipoprotein-cholesterol (HDL-c) and increased risk of coronary heart disease (CHD). Low density lipoprotein-cholesterol (LDL-C) is considered as the most important risk factor of coronary artery disease (CAD). Its oxidized form promotes foam cells formation which initiates the process of atherosclerosis by accumulating in sub-endothelium cells leading to fatty streaks and complex fibro fatty or atheromatous plaques formation (Berliner et al, 1995). The oxidation of low-density lipoprotein (LDL) can be limited by antioxidant enzyme system, including superoxide dismutase, catalase, glutathione peroxidase and antioxidant vitamins C, A, E and other carotenoids. Among the endogenous antioxidant system, includes albumin, uric acid, and total bilirubin. Imbalance of this reaction either due to excess free radical formation or insufficient removal by antioxidants leads to oxidative stress (Frei et al, 1998; Shrinivas et al, 2000; Maritim et al, 2003).

Various other risk factors have been identified apart from dyslipidemia are caeruloplasmin, C-reactive proteins, Lipoprotein (a), plasma fibrinogen, etc. Since we have encountered myocardial infarct patients with normal serum lipid concentration, we conducted a prospective case-control study to evaluate the concentration of antioxidant enzymes, degree of lipid peroxidation and other risk factors associated with acute myocardial infarction.

2. Materials and methods

The prospective case-control study consisted of 165 patients (123 men and 42 women) with AMI, admitted to the Intensive Cardiac Care Unit (ICCU), Sharda Hospital, India. The diagnosis of AMI was established according to diagnostic criteria: chest pain lasting for ≤3 hours, electrocardiographic (ECG) changes (ST elevation ≥ 2 mm in at least two leads) and elevation in enzymatic activities of serum creatine phosphokinase (CPK) and aspartate aminotransferase (AST). The control group consisted of 165 age/sex-matched healthy volunteers (123 men and 42 women). The design of this study was pre-approved by the
institutional ethical committee board and informed consent was obtained from the patients and controls. Inclusion criteria were patients with a diagnosis of acute myocardial infarction (AMI) with normal lipid profile. Patients with diabetes mellitus, renal insufficiency, current and past smokers, hepatic disease or taking lipid lowering drugs or antioxidant vitamin supplements were excluded from the study. Normolipidemic status was judged by the following criteria: LDL≤160 mg/dl; HDL, ≥35 mg/dl; total cholesterol (TC), <200 mg/dl; and triglycerides (TG), <150 mg/dl (NCEP, ATP-III, 2001). Ten milliliters of blood was collected after overnight fasting for lipid profile assay. For ischemia-modified albumin (IscMA) analysis, 2 ml of blood was collected from the patients immediately after admission to intensive care unit.

Lipid Profile Total cholesterol (TC), triglyceride (TG) and high density lipoprotein-cholesterol (HDL-c) were analyzed enzymatically using kit obtained from Randox Laboratories Limited, Crumlin, UK. Plasma low density lipoprotein -cholesterol was determined from the values of total cholesterol and high density lipoprotein-cholesterol using the following formula:

\[ \text{LDL-cholesterol} = \text{TC} - \frac{\text{TG}}{5} - \text{HDL-cholesterol (mg/dl)} \]

Other assays - Serum albumin was measured by Bromocresol green binding method (Perry et al, 1979). Serum uric acid was estimated by the method of Brown based on the development of a blue color due to tungsten blue as phosphotungstic acid is reduced by uric acid in alkaline medium (Brown, 1945). Serum total bilirubin was estimated by the method of Jendrassik and Grof (Jendrassik and Grof, 1938).

The gluthathione peroxidase (GPx) activity was determined by the procedure of Paglia and Valentine (Paglia and Valentine,1967). Superoxide dismutase (SOD) enzyme activity was measured by SOD assay kit using rate of inhibition of 2-(4-indophenyl)-(4-Nitrophenol)-5-phenyltetrazolium chloride (I.N.T) reduction method modified by Sun et al (Sun et al, 1988). Catalase activity was measured spectrophotometrically as described by Beutler (Beutler, 1984). MDA levels were estimated by thiobarbituric acid (TBA) reaction (Bernheim et al, 1948).Conjugated diene (CD) levels were measured by Recknagel and Glende method (Recknagel and Glende, 1984) with little modification. Caeruloplasmin assay was done by p-phenylene diamine method (Ravin, 1961). Ischemia-modified albumin (IscMA) concentration was determined by addition of a known amount of cobalt (II) to a serum sample and measurement of the unbound cobalt (II) by the intensity of colored complex formed after reacting with dithiothreitol (DTT) by colorimeter (Libby, 2003). Lipoprotein (a), levels were determined by Latex- Enhanced turbidimetric method. Serum paraoxonase was estimated using Zeptometrux Assay Kit obtained from Zeptometrux Corp, New York, 14202 based on the cleavage of phenyl acetate resulting in phenol formation. The rate of formation of phenol is measured by monitoring the increase in absorbance at 270 nm at 25°C.

Estimation of ascorbic acid was carried out by Roe and Kuether method (Roe and Kuether, 1943). The C-reactive protein were determined using high sensitivity enzyme Immunoassay kit manufactured by Life Diagnostics,inc., Catalog Number: 2210. The principle of the assay was based on a solid phase enzyme-linked immunosorbent assay (Kumar and Sivakanesan,
The plasma fibrinogen was determined using kit which was obtained from TECloT Fib Kit 10 Catalog No: 050-500, manufactured by TECO GmbH, Dieselstr. 1, 84088 Neufahrn NB Germany (Kumar and Sivakanesan, 2008).

All chemicals of analytical grade were obtained from Sigma-Aldrich Company, New Delhi.

### 3. Results

Anthropometric parameters in acute myocardial infarction (AMI) patients and control are shown in Table 1. Total cholesterol, its ratio to high density lipoprotein -cholesterol (TC/HDL-C) and triglyceride were significantly higher in both sexes of patients compared with control (Table 2 and 3). The low density lipoprotein –cholesterol (LDL-c) and its ratio to high density lipoprotein –cholesterol (LDL-C/HDL-C) were higher in acute myocardial infarction (AMI) subjects than in control (Table-3). The behavioral pattern and familial history of cardiovascular disease is presented in Table 4. The distribution of risk factors and relative risk according to potential risk factors among cases and controls are presented in Table 5 and Table 6. The status of antioxidants and lipid peroxidation are shown in Tables 7. All antioxidants were significantly decreased in patients compared with controls. In agreement with this serum malondialdehyde (MDA) and conjugated diene (CD) were more abundant in patients compared with controls. Ischemia-modified albumin (IscMA) levels were also greater in both male and female patients compared with control (Table 7).

|                      | Control (n=165) | MI patients (n=165) | P-value (95%CI) |
|----------------------|-----------------|---------------------|-----------------|
| **Age (years)**      | 60.5 ± 3.4      | 61.8 ± 3.8          | 0.0037 (61.26-62.33) |
| **Height (m)**       | 1.63 ± 0.04     | 1.64 ± 0.59         | 0.2919 (1.55-1.72) |
| **Weight (kg)**      | 68.34 ± 3.97    | 72.01 ± 5.37        | <0.01 (71.25-72.76) |
| **BMI (kg/m²)**      | 25.40 ± 1.20    | 26.16 ± 1.45        | <0.01 (25.95-26.36) |
| **Waist Circumference (cm)** | 93.70 ± 3.63 | 100.77 ± 6.06   | <0.01 (99.91-101.62) |
| **Hip Circumference (cm)** | 100.01 ± 3.16 | 105.72 ± 5.23 | <0.01 (104.82-106.45) |
| **Waist-Hip ratio**  | 0.93 ± 0.01     | 0.95 ± 0.01         | <0.001 (0.94-0.95)  |
| **Mid Arm Circumference (cm)** | 29.70 ± 1.47 | 30.63 ± 1.87   | <0.01 (30.36-30.89) |
| **Biceps skin fold thickness (mm)** | 6.95 ± 1.05 | 7.5 ± 1.38     | <0.001 (7.30-7.69) |
| **Triceps skin fold thickness (mm)** | 11.97 ± 1.27 | 12.89 ± 1.69 | <0.001 (12.65-13.12) |
| **Systolic blood pressure (mmHg)** | 121.06 ± 4.19 | 134.32 ± 11.65 | <0.05 (132.67-135.96) |
| **Diastolic blood pressure (mmHg)** | 79.90 ± 3.64 | 86.04 ± 4.25 | <0.05 (85.44-86.63) |

Table 1. Anthropometric data of control and patients (mean ± SD)
### Variables

|               | Controls (n=165) | Patients (n=165) | P-value (95%CI) |
|---------------|------------------|------------------|-----------------|
| Age           | 60.55 ± 3.98     | 61.84 ± 3.80     | 0.0037 (61.26-62.42) |
| Total Cholesterol (mg/dl) | 168.58 ± 12.16 | 186.44 ± 13.95 | <0.001 (184.31-188.56) |
| HDL-Cholesterol (mg/dl) | 50.51 ± 6.78   | 41.27 ± 4.62     | <0.001 (40.56-41.97) |
| Triglycerides (mg/dl)   | 107.84 ± 11.51  | 128.96 ± 12.19   | <0.001 (127.10-130.82) |
| LDL-Cholesterol (mg/dl) | 83.59 ± 11.95   | 119.37 ± 14.05   | <0.001 (117.22-121.51) |
| TC: HDL-C       | 3.39 ± 0.36      | 4.57 ± 0.58      | <0.001 (4.48-4.65) |
| LDL: HDL-C      | 1.90 ± 0.31      | 2.93 ± 0.51      | <0.001 (2.85-3.00) |
| TG: HDL-C       | 2.17 ± 0.35      | 3.16 ± 0.49      | 0.3149 (3.086-3.234) |

**Table 2. Lipid profile in patients and healthy controls (mean ± SD)**

| Ratio | Controls (n=165) | Patients (n=165) |
|-------|------------------|------------------|
| TC/HDL-C |                  |                  |
| 2-3   | 2.90 ± 0.09      | -                |
|       | (n=28)           |                  |
| 3-4   | 3.44 ± 0.25      | 3.70 ± 0.20      |
|       | (n=129)          | (n=31)           |
| 4-5   | 4.19 ± 0.22      | 4.53 ± 0.27      |
|       | (n=8)            | (n=90)           |
| 5-6   | -                | 5.26 ± 0.23      |
|       |                  | (n=44)           |
| TG/HDL-C |                  |                  |
| 1-2   | 1.77 ± 0.13      | -                |
|       | (n=56)           |                  |
| 2-3   | 2.38 ± 0.23      | 2.65 ± 0.27      |
|       | (n=109)          | (n=59)           |
| 3-4   | -                | 3.42 ± 0.26      |
|       |                  | (n=99)           |
| 4-5   | -                | 4.22 ± 0.19      |
|       |                  | (n=7)            |
| LDL-C/HDL-C |                  |                  |
| 1-2   | 1.71 ± 0.17      | 1.86 ± 0.15      |
|       | (n=106)          | (n=5)            |
| 2-3   | 2.23 ± 0.21      | 2.57 ± 0.27      |
|       | (n=59)           | (n=81)           |
| 3-4   | -                | 3.32 ± 0.21      |
|       |                  | (n=74)           |
| 4-5   | -                | 4.11 ± 0.12      |
|       |                  | (n=5)            |

**Table 3. Distribution of Lipid ratios in patients and healthy controls (mean ± SD)**
### Table 4. Behavioral Pattern in AMI patients and control

| Behavioral Pattern        | AMI Cases (n=165) | Controls (n=165) |
|---------------------------|-------------------|------------------|
| Age (y)                   | 61.84 ± 3.80      | 60.55 ± 3.98     |
| BMI (kg/m²)               | 26.16 ± 1.45      | 25.40 ± 1.20     |
| Waist-to-hip ratio        | 0.95 ± 0.11       | 0.93 ± 0.08      |
| Alcohol intake (servings/d)| 0.36 ± 0.68      | 0.15 ± 0.34      |
| Physical activity (MET-min/d) | 56.23 ± 123.8  | 97.83 ± 174.8a   |
| Current cigarette smokers (%) | 14.45            | 3.6b             |
| Current bidi smokers (%)  | 23.67            | 12.31c           |
| Family history of MI (%)  | 37.57            | 8.48d            |
| Hypertension (%)          | 49.09            | 1.8e             |
| Alcoholics (%)            | 47.87            | 20.60f           |

Values are in Mean ± SD  

|                | No. of cases N | No. of controls N | Age- and sex-adjusted RR (95% CI) | Multivariate RR (95% CI) |
|----------------|----------------|-------------------|-----------------------------------|--------------------------|
| **Cigarette smoking**                |                |                   |                                   |                          |
| Never smoker                              | 120            | 136               | 1.0                               | 1.0                      |
| >10 cigarettes/d                          | 36             | 6                 | 7.8 (4.9, 13.5)                   | 7.4 (4.3, 15.2)          |
| **Bidi smoking**                         |                |                   |                                   |                          |
| Never smoker                              | 120            | 136               | 1.0                               | 1.0                      |
| > 10 bidis/d                              | 49             | 8                 | 8.2 (5.2, 14.2)                   | 6.5 (3.9, 12.9)          |

Numbers in parentheses are percent unless mentioned otherwise  
*Triffle thinker: subjects who thinks and worries on unnecessary small things

Table 5. Distribution of risk factors among AMI patients and control
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| BMI (kg/m²) | No  | Yes  | 1.0          | 1.0          |
|-------------|-----|------|--------------|--------------|
| 20-24.9     | 30  | 51   | 1.0          | 1.0          |
| ≥ 25        | 135 | 114  | 2.7 (1.8, 4.1) | 2.9 (1.6, 5.1) |
| Waist –to-hip ratio | | | | |
| ≤ 0.95      | 52  | 137  | 1.0          | 1.0          |
| > 1.0       | 113 | 28   | 3.9 (2.1, 6.3) | 2.8 (1.6, 5.7) |
| Family history of MI | | | | |
| No          | 97  | 151  | 1.0          | 1.0          |
| Yes         | 62  | 14   | 2.1 (1.6, 2.7) | 2.7 (1.8, 3.8) |
| History of Hypertension | | | | |
| No          | 136 | 142  | 1.0          | 1.0          |
| Yes         | 29  | 23   | 2.1 (1.7, 3.2) | 1.9 (1.4, 2.9) |
| Education level | | | | |
| Highest level of education | 25  | 27   | 1.0          | 1.0          |
| None        | 101 | 132  | 3.1 (1.3, 5.1) | 3.6 (1.0, 6.2) |
| Type of Family | | | | |
| Split       | 20  | 64   | 1.0          | 1.0          |
| Joint       | 145 | 101  | 4.5 (1.5, 2.9) | 3.9 (1.2, 2.6) |
| Civil Status | | | | |
| Lower Class | 10  | 19   | 1.0          | 1.0          |
| Middle Class| 119 | 131  | 3.4 (4.3, 6.7) | 2.8 (3.7, 5.9) |
| Higher Class| 36  | 15   | 4.7 (4.9, 7.2) | 3.8 (3.1, 4.7) |
| Leisure –time exercise | | | | |
| Non-exerciser | 82  | 58   | 1.0          | 1.0          |
| ≥ 145 MET-min/d | 83  | 107  | 0.76 (0.4, 0.8) | 0.68 (0.4, 0.7) |
| Household income | | | | |
| >10 000 rupees/month | 155 | 146  | 1.0          | 1.0          |
| <5000 rupees/month  | 10  | 19   | 1.8 (1.2, 2.7) | 1.7 (1.0, 3.1) |
| Hindu religion | | | | |
| No          | 33  | 12   | 1.0          | 1.0          |
| Yes         | 132 | 153  | 0.8 (0.6, 1.1) | 0.9 (0.7, 1.3) |

- MET, metabolic equivalent. RR estimates were obtained by using conditional logistic regression analysis controlled for the matching factors (age, sex, and hospital) and then additional potential risk factors.
- Also adjusted for hospital.
- Covariates controlled for in the multivariate model were as follows: age; sex; hospital; cigarette smoking never, current (≤10 cigarettes/d, >10 cigarettes/d); bidi smoking [never, current (≤10 bidis/d, >10 bidis/d)]; BMI, in kg/m² (20-24.9, ≥25); waist-to-hip ratio (≤0.95, >1.0); leisure time physical exercise (none, ≥145 MET-min/d, ≤145 MET-min/d); history of hypertension (no, yes); history of diabetes (no, yes); history of high cholesterol (no, yes); family history of IHD (no, yes); education (none, primary school, middle, secondary, higher secondary, college, graduate or professional); household income (<5000, 5000-10000, 10000-15000, >10000 rupees/mo); and Hindu religion (no, yes).
- Bidis (pronounced bee-dees) are small hand-rolled cigarettes manufactured in India and other southeast Asian countries. They are exported to as many as 122 countries, according to one bidi manufacturer. Bidi cigarettes are made of tobacco wrapped in tendu or temburni leaf (Diospyros melanxylon).

Table 6. Relative risk (RR) of Acute Myocardial Infarction (AMI) according to potential risk factors.
Table 7. Antioxidant status and Lipid Peroxidation in Control and AMI patients (mean ± SD)

| Parameter                                      | Control (n=165) | AMI patients (n=165) | P value (95% CI) |
|------------------------------------------------|-----------------|----------------------|------------------|
| Serum albumin (mg/dl)                          | 4.4 ± 0.3       | 4.2 ± 0.3            | <0.001(4.17-4.28) |
| Serum uric acid (mg/dl)                        | 5.8 ± 1.2       | 4.3 ± 0.9            | <0.01(4.18-4.45)  |
| Serum ascorbic acid (mg/dl)                    | 5.3 ± 1.2       | 2.8 ± 0.7            | <0.0001(2.70-2.89) |
| Serum Total bilirubin (mg/dl)                  | 0.8 ± 0.2       | 0.7 ± 0.2            | <0.001(0.62-0.69)  |
| Serum superoxide dismutase (U/gHb)            | 1826.5 ± 31.9   | 813.9 ± 208.9        | <0.02 (784.42-843.37) |
| Serum glutathione peroxidase (U/gHb)          | 61.3 ± 3.9      | 42.6 ± 6.3           | <0.001(41.71-43.48) |
| Serum catalase (k/gHb)                        | 256.2 ± 26.7    | 193.1 ± 35.9         | <0.001(188.03-198.16) |
| Serum Lipoprotein (a) (mg/dl)                 | 3.0 ± 1.1       | 10.9 ± 2.2           | <0.0001 (10.58-11.21) |
| Serum malondialdehyde (nmol/L)                | 5.7 ± 1.0       | 14.8 ± 1.7           | <0.02 (11.55-15.06)  |
| Serum conjugated dienes (µmol/L)              | 31.0 ± 2.7      | 48.3 ± 5.5           | <0.001 (47.44-49.11)|

Table 8. Other Biochemical parameters in Control and AMI patients (mean ± SD)

| Parameter                                      | Control (n=165) | AMI patients (n=165) | P value (95% CI) |
|------------------------------------------------|-----------------|----------------------|------------------|
| Plasma fibrinogen (mg/dl)                      | 237.5 ± 17.4    | 357.8 ± 23.2         | <0.0001 (354.52-361.07) |
| Serum caeruloplasmin (mg/dl)                   | 20.4 ± 2.3      | 51.5 ± 2.4           | <0.0001 (51.16-51.83) |
| Serum Arylesterase activity (kU/L)             | 98.4 ± 6.2      | 69.7 ± 10.0          | <0.0001 (68.28-71.11) |
| Serum Ischemia modified albumin (U/ml)         | 81.9 ± 3.9      | 97.5 ± 11.7          | <0.001 (95.84-99.15) |
| Serum C-reactive protein (mg/dl)               | 1.1 ± 0.3       | 3.0 ± 1.1            | <0.0001 (2.84-3.15)  |

4. Discussion

Coronary artery disease (CAD) remains the major cause of morbidity and mortality in all developed and developing countries in the world including India (Reddy and Yusuf, 1998). Dyslipidemia is one of the major modifiable risk factors for CAD (Chopra et al, 1998; Vasisht et al, 2000; Malhotra et al, 2003).

The coronary artery disease (CAD) risk factors do not predict the occurrence of acute myocardial infarction (AMI) as variation in risk factors is observed in South Asian population due to varied dietary habits and life style (Mishra et al, 2005). The search for various conventional risk factors among Asians could be helpful in recognizing the future events of stroke. These curiosities prompted us to identify the newer risk factors, with respect to Indian population.
The search for the newer risk factors continues and researchers are investigating the role of inflammatory markers and other potential risks factors which could link with acute myocardial infarction (AMI).

In this prospective case-control study in India, only normolipidaemic acute myocardial infarction (AMI) patients were selected. The study was designed to identify and evaluate potential risk factors in normolipidaemic acute myocardial infarction (AMI) patients. The subjects selected for the study comprised of 165 controls, 48-69 y and 165 acute MI patients, 48-69 y.

Anthropometric variables in acute myocardial infarction (AMI) patients showed highly significant differences in waist/hip ratio and biceps skin fold thickness. Study reported (Heitman et al, 2004) that waist /hip ratio is a dominant, independent and predictive variable of cardiovascular disease and coronary heart disease deaths in Australian men and women. Megnien et al, 1999 also reported high hip circumference relative to weight and waist circumference is a better predictor of low incidence of cardiovascular disease and coronary heart disease. The present study is in good agreement with the observations of the above studies. Among Indians the cardiovascular risk is high even the prevalence of obesity is minimal (Megnien et al, 1999). In the present study the mean body mass index and waist /hip ratio in all subjects was 26.56 and 0.96 respectively, showing a significantly higher body mass index and weight /hip ratio in patients compared with control.

Based on the observations of the aforementioned studies and further supported by the present study it could be concluded that weight/hip ratio is a better predictor of cardiovascular disease (CVD) than body mass index. So it is better tool for indentifying the future risk of acute myocardial infarction (AMI) in subjects by non-invasive procedures.

Observations of lipid profile

The mean total cholesterol level of the controls compared with acute myocardial infarction patients (186.44 ± 13.95 mg/dl) was significantly (p<0.001) higher compared with controls (168.58 ± 12.16 mg/dl). The mean high density lipoprotein-cholesterol level in the patients was significantly lower (p<0.001) compared with controls. Triglyceride (TG) values observed in acute myocardial infarction (AMI) patients was (129mg/dl) significantly higher than controls (107.8mg/dl). The mean low density lipoprotein-cholesterol (LDL-c) levels in patients was (119.4mg/dl) significantly higher than controls (83.6 mg/dl). The total cholesterol / high density lipoprotein – cholesterol ratio in acute myocardial infarct patients (4.6) was significantly (p<0.001) higher compared with controls (3.4). The present study observed significantly higher ratio (2.9) in acute myocardial infarction patients compared with control (1.9).

Earlier studies in lipid profile analysis conducted on acute myocardial infarction patients (Mishra et al, 2001; Das et al, 2002; Goswami et al, 2003; Kharb et al, 2003; Malhotra et al, 2003; Burman et al, 2004; Rajashekhar et al, 2004; Sivaraman et al, 2004; Rani et al, 2005; Shindhe, et al, 2005; Yadav et al, 2006; Patil et al, 2007) observed higher total cholesterol, triglyceride, low-density lipoprotein –cholesterol and lower levels of high-density lipoprotein-cholesterol in patients compared to controls.

Also higher ratio of total cholesterol to high density lipoprotein-cholesterol, low-density lipoprotein-cholesterol to high-density cholesterol-lipoprotein and higher triglyceride to
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High-density cholesterol-lipoprotein was observed in the present study. The present study concludes the importance of assessing the lipid ratios even in normolipidaemic subjects as it is one of the atherogenic factors for development of myocardial infarction and other coronary complications. The practice of computing the ratio should be implemented even in a normal health check-up packages. In the final analysis it appears that myocardial infarction and coronary artery disease are not always associated with an elevated serum total cholesterol concentration. The major concern of this observation is that subjects who maintain desirable total cholesterol concentration also are targets for myocardial infarction (MI) and coronary artery disease (CAD) and therefore analysis of other risk factors that are non-conventional and newly emerging will be of immense important in the eventual assessment of the risk status. The existing literature and the results of the present study all point out that acute myocardial infarction and coronary artery disease patients have significantly higher total cholesterol concentration whether the values are in the desirable range or elevated.

Antioxidant status

The serum endogenous antioxidants were decreased in acute myocardial infarction compared to controls. Similarly the enzyme antioxidants were also significantly lowered in patients.

Study conducted (Olusi et al, 1999; Djousse et al 2003) in acute myocardial infarction patients, reported significantly lower (p<0.0001) albumin and bilirubin (p<0.0001), where as lower levels of uric acid (Jing et al, 2000; Brand et al, 1985; Niskanen et al, 2004) and ascorbic acid (Nyossen et al, 1997; Bhakuni et al, 2006; Das et al, 2002; Kurl et al, 2002) in acute myocardial infarct patients were reported.

The aforementioned studies suggested the expected risk of acute myocardial infarction is increased where these endogenous antioxidants are lowered due to enhanced utilization during oxidative stress in patients. Though, uric acid is well established antioxidant, but at times it can also act as a pro-oxidant, which might increase the risk of myocardial infarction. Aulinskas et al, (1983) established the role of ascorbic acid as up regulator of low density -lipoprotein (LDL) receptors, facilitating the clearance of low density -lipoprotein (LDL). The low levels of ascorbic acid in acute myocardial infarction (AMI) patients in the present study might be due to enhanced utilization of ascorbic acid during oxidative stress in patients.

The enzymatic antioxidants namely superoxide dismutase, catalase and glutathione peroxidase are also lowered in patients compared with controls. The findings of the present study concurs to earlier studies (Senthil et al, 2004; Bhakuni et al, 2006; Jain et al, 2000; Rajashekhar et al, 2004; Das et al, 2002; Gupta et al, 2006; Patil et al, 2007) where lower activities of superoxide dismutase, catalase and glutathione peroxidase. Studies conducted (Senthil et al, 2004; Shindhe et al, 2005; Rajasekhar et al, 2004; El-Badry et al, 1995; Gupta et al, 2006 and Kharb 2003) also reported reduced activities of glutathione peroxidase in patients compared with controls. These studies are based on the hypothesis of decreased antioxidants due to oxidative insult in myocardial infarct patients. Thus it is indicative that low levels of both endogenous and enzyme antioxidants in circulation may be due to its increased utilization to scavenge toxic lipid peroxides.
Lipoprotein (a) and lipid peroxidation

The mean serum Lipoprotein (a) malondialdehyde (MDA) and conjugated diene (CD) levels in MI patients were higher compared with controls. Earlier studies conducted (Burman et al, 2004; Guha et al, 2001; Bal et al, 2001; Rajashekhari et al, 2004) also observed higher Lipoprotein (a) in AMI patients where as Nascetti et al, (1996) did not observed any change in Lipoprotein (a) levels in cardiovascular disease (CVD) patients and concluded lipoprotein (a) not to be considered as an independent risk factor in cardiovascular disease (CVD) patients.

Studies conducted (Senthil et al, 2004; Das et al, 2002; Kharb 2003; Bhakuni et al, 2006; Shindhe et al, 2005; Gupta et al, 2006) reported higher levels of malondialdehyde (MDA) in myocardial infarct patients.

Other biochemical parameters

The levels of caeruloplasmin, C-reactive protein, fibrinogen, ischemia-modified albumin were higher and arylesterase activities were lowered in patients. Studies conducted (Grobusch et al, 1999; El-Badry et al, 1995; Giurgie, 2005; Awadallah et al, 2006) observed significantly higher (p<0.001) levels of caeruloplasmin where as (Berton et al, 2003; Bhagat et al, 2003; Sivaraman et al, 2004; Kulsoom et al, 2006; Boncler et al, 2006) observed higher levels of C-reactive protein in patients. Shukla et al, (2006) stated elevated levels of caeruloplasmin as a risk factor for acute myocardial infarct patients. The reactive oxygen species disrupts copper binding to caeruloplasmin thus impairing its antioxidant property and further promoting oxidative pathology. Studies conducted on plasma fibrinogen levels in acute myocardial infarct patients (Harkut et al, 2004; Coppola et al, 2005; Beg et al, 2007; Sivaraman et al, 2004) reported rise in plasma fibrinogen as the present study. Earlier study conducted (Chawla et al, 2006; Auxter, 2003; Bar-Or et al, 2001) in acute myocardial infarct patients also reported higher levels in patients as observed by the present study. Studies on arylesterase activities in acute myocardial infarct patients (Aviram et al, 1999; Ayub et al, 1999; Richard et al, 2000; Jarvik et al, 2002; Azizi et al, 2002; Singh et al, 2007; Sarkar et al, 2006) also observed lower activities as concurrent to the current study. Increased C-reactive protein (CRP) concentrations in patients with unstable angina and acute myocardial infarct might induce the production by the monocytes of the tissue factor which initiates the coagulation process. C-reactive protein together with fibrinogen acts as a chemotactic factor. Fibrinogen is responsible for the adhesion of macrophages to the endothelial surface for their migration into the intima. The elevated c-reactive protein levels have been found to be related to the occurrence of cardiovascular complications such as sudden cardiac death or AMI (Pepys and Hirschfield, 2003).

Our study concluded apart from lipid profile, other variables which could be a probable risk for the future myocardial events have to be equally monitored. It is also recommended to increase dietary antioxidant intake in persons who already have known risk factors so that to some extent the myocardial infarction could be delayed. It is also important to check inflammatory markers like c-reactive protein and ischemia-modified albumin in a regular period of time after stepping early forties as they could be a cost effective mode of diagnosis and the subjects can be efficiently monitored and complications of myocardial infarction can be prolonged.
5. References

Auxter S. Cardiac Ischemia testing: a new era in chest pain evaluation. Clin Lab News 2003; 29, 1-3.

Aulinskas TH, Vander Westhyzen DR. Coetzee GA. Ascorbate increases the number of low density lipoprotein receptors in cultured arterial smooth muscle cells. Atherosclerosis 1983; 47: 159-71.

Aviram M, Rosenblat M, Billecke S, Eroul J, Soverson R, Bisaier CL. Human serum paraoxonase (PON1) is in activated by oxidized low density lipoprotein and preserved by antioxidants. Free Radic Biol Med 1999; 26:892-904.

Awadallah SM, Hamad M, Jbarah I, Salem NM, Mubarak MS. Autoantibodies against oxidized LDL correlate with serum concentrations of caeruloplasmin in patients with cardiovascular disease. Clin Chim Acta 2006; 365: 330-336.

Ayub A, Mackness MI, Sharon A, Mackness B, Patel J, Durrington PN. Serum Paraoxonase After Myocardial Infarction. Arteriosclerosis, Thrombosis, and Vascular Biology 1999; 19:330-335.

Azizi F, Rahmani M, Raiszadeh F, Solati M, Navab M. Associations of lipids, lipoproteins, apolipoproteins and paraoxonase enzyme activity with premature coronary artery disease. Coronary Artery Dis 2002; 13(1): 9-16.

Balarajan R. Ethnicity and variations in mortality from coronary heart disease. Health Trends 1996; 28:45–51.

Bar-Or,D, Lau E, Rao N, Bampos N, Winkler JV, Curtis CG. Reduction in the cobalt binding capacity of human albumin with myocardial ischemia. Ann. Emerg. Med 1999; 34: 556.

Berliner JA, Navab M, Fogelman AM, Frank JS, Demer LL, Edwards PA, et al. Atherosclerosis: basic mechanisms. Oxidation. Inflammation, and genetics. Circulation 1995; 91:2488-96.

Bernheim S, Bernheim MLC, Wilbur KM. The reaction between thiobarbituric acid and the oxidant product of certain lipids. J Biol Chem 1948; 174: 257-264.

Berton G, Cordiano R, Palmieri R, Pianca S, Pagliara V, Palatini P. C-Reactive Protein in Acute Myocardial Infarction: Association With Heart Failure. Am Heart J 2003; 145(6):1094-1101.

Beutler E. Red Cell Metabolism: A Manual of Biochemical Methods, 3rd edition. New York, Grune and Stratton 1984; 105.

Beg M, Nizami A, Singhal KC, Mohammed J, Gupta A, Azfar SF. Role of serum fibrinogen in patients of ischemic cerebrovascular disease. Nepal Med Coll J 2007; 9: 88-92.

Bhagat S, Gaiha M, Sharma VK, Anuradha S A. Comparative Evaluation of C - reactive protein as a Short-Term Prognostic Marker in Severe Unstable Angina- A Preliminary Study. Journal of Assoc Physicians 2003; 51:349-354.

Bhakuni P, Chandra M, Misra MK. Levels of free radical scavengers and antioxidants in post perfused patients of myocardial infarction. Current Science 2005; 89: 168-170.

Bhakuni P, Chandra M, Misra MK. Oxidative stress parameters in erythrocytes of post-reperfused patients with myocardial infarction. J Enzyme Inhib Med Chem 2005; 20(4):337-81.

Boncler M, Luzak B, Watala C. Role of C-reactive protein in atherogenesis. Postepy Hig Med Dosw 2006; 60:538-46.
Cardiovascular Risk Factors in Elderly Normolipidaemic Acute Myocardial Infarct Patients

Brand FN, Mcgee DL, Kannel WB, Stokes J, Castelli W P. Hyperuricemia as a risk factor of coronary heart disease: The Framingham Study. *American Journal of Epidemiology* 1985; 121: 11-18.

Brown H. *J Clin Chem* 1945; 158:601.

Bulatao RAO, Stephens PW. Demographic estimates and projections, by region, 1970-2015. In: Jamison DT, Mosley WH, eds. Disease control priorities in developing countries. *Washington, DC: World Bank*, 1990. (Health sector priorities review no. 13.)

Burman A, Jain K, Gulati R, Chopra V, Agrawal DP, Vaisisht S. Lipoprotein (a) as a marker of Coronary Artery Disease and its Association with Dietary Fat. *J Assoc Physicians India* 2004; 52:99-102.

Centers for Disease Control and Prevention. Heart Disease and Stroke Prevention. Addressing the nation’s leading killers: at a glance 2010. Available from: http://www.cdc.gov/chronicdisease/resources/publications/AAG/dhdsp.htm

Chawla R, Goyal N, Calton R, Goyal S. Ischemia modified albumin: A novel marker for acute coronary syndrome. *Indian Journal of Clinical Biochemistry* 2006; 21(1):77-82.

Chopra V, Wasir H. Implications of lipoprotein abnormalities in Indian patients. *Journal Assoc Physicians of India* 1998; 46:814-8.

Coppola G, Rizzo M, Maurizio GA, Corrado E, Alberto DG, Braschi A, Braschi G, Novo S. Fibrinogen as a predictor of mortality after acute myocardial infarction: a forty-two-month follow up study. *Ital Heart J* 2005; 6:315-322.

Das S, Yadav D, Narang R, Das N. Interrelationship between lipid peroxidation, ascorbic acid and superoxide dismutase in coronary artery disease. *Current Science* 2002; 83:488-491.

Djousse L, Rothman KJ, Cupples LA, Levy D, Ellison RC. Effect of serum albumin and bilirubin as a risk factor for Myocardial infarction. *Am J Cardiol* 2003; 91: 485-488.

El- Badry I, Abon El N, Yehia T K, Zakhari MM. Free radicals activity in Acute Myocardial Infarction. *The Egyptian Heart Journal* 1995; 47: 71-78.

Executive Summary of The Third Report of The National Cholesterol Education Program (NCEP) Expert panel on Detection, Evaluation, and treatment of high Blood Cholesterol in Adults (Adult Treatment Panel III). Expert Panel of Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults. *JAMA* 2001; 285(19):2486-97.

Frei B, Stocker R, Ames BN. Antioxidant defenses and lipid peroxidation in human blood plasma. *Proc Natl Acad Sci USA* 1988; 85: 9748-9752.

Friedewalds, WT, Levy RI, Fredrickson DS. Estimation of the concentration of low density lipoprotein cholesterol in plasma without the use of preparative ultracentrifuge. *Clin. Chem* 1972; 18, 499-502.

Ghosh J, Mishra TK, Rao YN, Aggarwal SK. Oxidised LDL, HDL Cholesterol, LDL Cholesterol levels in patients of Coronary Artery Disease. *Indian Journal of Clinical Biochemistry* 2006; 21(1):181-184.

Giurgea N, Constantinescu MI, Stanciu R, Suciu S, Muresan A. Caeruloplasmin- acute – phase reactant or endogenous antioxidant? The case of cardiovascular disease. *Med Sci Monit* 2005; 11: RA 48-51.

Gomez MA, Anderson JL, Karagounis LA, Muhlestein JB, Mooers FB. An emergency medicine based protocol for rapidly ruling out myocardial ischemia reduces
hospital time and expense. Results of randomized study (ROMO). J. Am. coll. Cardiol 1996; 28:25-33.

Goswami K Bandopadhyay. Lipid profile in middle class Bengali population of Kolkata. Ind J of Clin Biochem 2003; 18:127-130.

Gupta M, Chari S. Proxidant and Antioxidant status in patients of type II Diabetes Mellitus with IHD. Indian Journal of Clinical Biochemistry 2006; 21(2):118-122.

Harkut PV, Sahashrabhojney VS, Salkar RG. Plasma fibrinogen as a marker of major adverse cardiac events in patients of type 2 Diabetes with unstable angina. Int J Diab Dev Countries 2004; 24: 69-74.

Heitman BL, Frederickson P, Lissner L. Hip Circumference and Cardiovascular Morbidity and Mortality in Men and Women. Obesity Research 2004; 12:482-487.

Jarvik GP, Tsai NT, Mckinstry LA, Wani R, Victoria HB, Richter RJ, Schellenberg GD, Heagerty PJ, Hatsuikami TS, Furlong CE. Vitamin C and E Intake Is Associated With Increased Paraoxonase Activity. Arteriosclerosis, Thrombosis, and Vascular Biology 2002; 22:1329.

Jendrassik L, Grof B. Biochem Zeit 1938; 297:81 9.

Jing F, Alderman M H. Serum uric acid and cardiovascular mortality. JAMA 2000; 283: 2404-2410.

Kharb S. Low Glutathione levels in acute myocardial infarction. Ind J Med Sci 2003; 57; Issue8: 335-7.

Kurl S, Tuomainen TP, Laukkanen JA, Nyyssonen K, Lakka T, Sivenius J, Salonen JT. Plasma Vitamin C Modifies the Association Between Hypertension and Risk of Stroke. Stroke 2002; 33:1568.

Kulsoom B, Nazrul SH. Association of serum C - reactive protein and LDL: HDL with myocardial infarction. J Pak Med Assoc 2006; 56 (7):318-22.

Kumar A, Sivakanesan R. Does plasma fibrinogens and C-reactive protein predict the incidence of myocardial infarction in patients with normal lipids profile? Pak J Med Sci 2008; 24:336-339.

Libby P. Vascular biology of atherosclerosis: Overview and state of art. Am J Cardiol 2003; 91(suppl): 3A-6A.

Malhotra P, Kumari S, Singh S, Verma S. Isolated Lipid Abnormalities in Rural and Urban Normotensive and Hypertensive North-West Indians. Journal of Assoc Physicians of India 2003; 51:459-463.

Maritim AC, Sanders RA, Watkins JB. Diabetes, oxidative stress, and antioxidants: a review. J Biochem Mol Toxicol 2003; 17: 24-38.

Megnien JL, Denarie N, Cocal M. Predictive value of Waist-to-hip ratio on Cardiovascular Risk Events. Int J Obes Relat Metab Disord 1999; 23:90-97.

Meyer BJ. Are we consuming enough long chain omega-3 polyunsaturated fatty acids for optimal health? Prostaglandins Leukot Essent Fatty Acids. 2011;DOI: 10.1016/j.plefa.2011.04.010

Mishra A, Luthra K, Vikram NK. Dyslipidemia in Asian Indians: Determinants and Significance. Journal Assoc Physicians India 2005; 52:137-142.

Mishra TK, Routray SN, Patnaik UK, Padhi PK, Satapathy C, Behera M. Lipoprotein (a) and Lipid Profile in Young Patients with Angiographically Proven Coronary Artery Disease. Indian Heart Journal 2001; 53 : (5) Article No. 60.

Mozaffarian D. JELIS, fish oil and cardiac events. Lancet. 2007; 369(9567):1062-1063.
Cardiovascular Risk Factors in Elderly Normolipidaemic Acute Myocardial Infarct Patients

Nascetti S, D’Addato S, Pascarelli N, Sangiorgi Z, Grippo MC, Gaddi A. Cardiovascular disease and Lp(a) in the adult population and in the elderly: the Brisighella study. *Riv Eur Sci Med Farmacol* 1996; 18(5-6):205-12.

National Heart Foundation of Australia. Data and statistics. Available from: http://www.heartfoundation.org.au/information-for-professionals/data-and-statistics

Niskanen LK, Laaksonen DE, Nyyssonen K, Alfthan G, Lakka H M, Lakka TA, Salonene JT. Uric acid level as a risk factor for cardiovascular and all-cause mortality in middle-aged men: a prospective study. *Arch Intern Med* 2004; 164:1546-51.

Nyyssonen K, Markku TP, Salonen R, Tuomilehto J, Salonen JT. Vitamin C deficiency and risk of myocardial infarction: prospective population study of men from eastern Finland. *BMJ* 1997; 314:634.

Olusi SO, Prabha K, Sugathan TN. Biochemical Risk factors for Myocardial Infarction Among South Asian Immigrants and Arabs. *Annals of Saudi Medicine* 1999; 19: 147-149.

Paglia DE, Valentine WN. Studies on quantitative and qualitative characterization of erythrocyte glutathione peroxidase. *J Lab Clin Med* 1967; 70: 158-69.

Patil N, Chavan V, Karnik ND. Antioxidant Status in Patients with Acute Myocardial Infarction. *Indian Journal of Clinical Biochemistry* 2007; 22(1):45-51.

Pepys MB and Hirschfield G M. C-reactive protein: a critical update. *J Clin Invest* 2003; 111(12): 1805-1812. doi: 10.1172/JCI200318921.

Perry BW, Doumas BT. Effect of heparin on albumin determination by use of bromocresol green and bromocresol purple. *Clin Chem* 1979; 25:1520-1522.

Rajasekhar D, Srinivasa Rao PV, Latheef SA, Saibaba KS, Subramanyam G. Association of serum antioxidants and risk of coronary heart disease in South Indian population. *Indian J Med Sci* 2004; 58(11):465-71.

Rani SH, Madhavi G, Ramachandra RV, Sahay BK, Jyothy A. Risk factors for coronary heart disease in type II diabetes. *Indian Journal of Clinical Biochemistry* 2005; 20(2):75-80.

Ravin HA. An improved colorimetric enzymatic assay of caeruloplasmin. *J. Lab. Med* 1961; 58, 161-168.

Recknagel RO, Glende EA. Spectrophotometric detection of lipid conjugated dienes. *Methods Enzymol* 1984; 105:331-337.

Reddy KS. Cardiovascular disease in India. *World Health Stat Q* 1993; 46:101-7.

Reddy KS, Yusuf S. Emerging epidemic of cardiovascular disease in developing countries. *Circulation* 1998; 97:596–601.

Richard JW, Leview I, Righetti A. Smoking Is Associated With Reduced Serum Paraoxonase Activity and Concentration in Patients With Coronary Artery Disease. *Circulation* 2000; 101:2252.

Roe JH, Kuether CA. *J. Biol Chem* 1943; 147:399.

Sarkar PD, TMS Madhusudhan B. Association between paraoxonase activity and lipid levels in patients with premature coronary artery disease. *Clin Chim Acta* 2006; 373:77-81.

Senthil S, Veerappan RM, Ramakrishna RM, Pugalendi KV. Oxidative stress and antioxidants in patients with cardiogenic shock complicating acute myocardial infarction. *Clin Chim Acta* 2004; 348 (1-2):131-7.
Shrinivas K, Vijaya Bhaskar M, Aruna Kumari M, Nagaraj K, Reddy KK. Antioxidants, lipid peroxidation and lipoproteins in primary hypertension. *Indian Heart J* 2000; 52:285-88.

Shinde S, Kumar P, Patil N. Decreased Levels Of Erythrocyte Glutathione In Patients With Myocardial Infarction. *The Internet Journal of Alternative Medicine* 2005; 2:1.

Singh S, Venketish S, Verma JS, Verma M, Lellamma CO, Goel RC. Paraoxonase (PON1) activity in northwest Indian Punjabis with coronary artery disease & type II diabetes mellitus. *Indian J Med Res* 2007; 125:783-7.

Sivaraman S K, Zachariah G, Annamalai PT. Evaluation of C - reactive protein and other Inflammatory Markers in Acute Coronary Syndromes. *Kuwait Medical Journal* 2004; 36(1):35-37.

Sun Y, Oberly LW, Li Y. A simple method for clinical assay of superoxide dismutase. *Clin Chem* 1988; 34: 497-500.

Vasisht S, Narula J, Awtade A, Tandon R, Srivastava LM. Lipids and lipoproteins in normal controls and clinically documented coronary heart disease patients. *Ann Natl Acad Med Sci (India)* 1990; 26:57-66.

World Health Organization. Cardiovascular diseases (CVDs). Available from: [http://www.who.int/mediacentre/factsheets/fs317/en/index.html](http://www.who.int/mediacentre/factsheets/fs317/en/index.html)

Yadhav AS, Bhagwat VR, Rathod IM. Relationship of Plasma homocysteine with lipid profile parameters in Ischemic Heart disease. *Indian Journal of Clinical Biochemistry* 2006; 21(1):106-110.
Among the non-communicable diseases, cardiovascular disorders are the leading cause of morbidity and mortality in both the developed and the developing countries. The spectrum of risk factors is wide and their understanding is imperative to prevent the first and recurrent episodes of myocardial infarction, stroke or peripheral vascular disease which may prove fatal or disabling. This book has tried to present an update on risk factors incorporating new research which has thrown more light on the existing knowledge. It has also tried to highlight regional diversity addressing such issues. It will hopefully be resourceful to the cardiologists, general practitioners, family physicians, researchers, graduate students committed to cardiovascular risk prevention.

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