Chapter - IV

Lipid profile and coronary artery disease
INTRODUCTION

Coronary Artery Disease (CAD) is an important cause of morbidity and mortality, increasing in the developed as well as in the developing countries. It was estimated in 2003, that 16.7 million or 29.2% of total global deaths resulted from various forms of Cardio Vascular Disease (CVD), in which coronary artery disease was the highest contributor. CAD is caused by atherosclerotic narrowing of the coronary arteries, leading to reduced blood supply to heart muscle, which can result in angina and/or myocardial infarction.

The prevalence of coronary artery disease is increasing dramatically, and it has been predicted that in the Indian subcontinent, in the next 20 years, due to rapid changes in demography and lifestyle consequent to economic development it is going to assume grievous proportions. The prevalence of CAD is four-times higher in urban India and two-times higher in rural India than that in the United States. Conventional risk factors like smoking, hypertension, and diabetes mellitus are not different among South Asians compared with the same among other ethnic groups. Lipid patterns, however, are known to vary with different food habits, climatic conditions, ethnic types, life style and geographical locations. Thus, South Asians are noted to have higher triglyceride (Tg) levels, low concentration of high-density lipoprotein cholesterol (HDL-C), increased visceral fat, and higher insulin resistance as more predominant risk factors. But total cholesterol (TC) levels have been observed to be lower in CAD patients of Indian origin when compared with their counterparts in the West. Most of such studies reported have been based on migrant Indians and a few reports are on people of South India and North India. Except for a few, there has been little study on lipid levels in patients with known CAD among Indians living in India, and especially in eastern India. It is, therefore planned to execute a broad-based study of serum lipid of eastern Indian people.

Hypercholesterolemia is one of the classic risk factors and it has positive association with CAD risk, as shown and confirmed repeatedly over the last five to six decades. Cholesterol was recognized as the lipid, present in atheromatous plaques soon after its discovery in the 19th century. The epidemiological association between serum
cholesterol, or more precisely, serum low-density lipoprotein cholesterol (LDL-C) and coronary artery disease (CAD) was well established in the 1960s and it came to be confirmed in the 1970s that familial hypercholesterolemia was a monogenic disorder due to mutations of the LDL receptor which raised circulating LDL without the collaboration of other CAD risk factors and could cause accelerated atherosclerosis.

Human body contains large quantities of cholesterol, which is found in brain and nervous tissues. Other tissues such as liver, kidney, spleen and skin also contain fairly good amounts of cholesterol. The total amount of cholesterol is about 140 grams in the body of man weighing 70 kg. The greater part of the cholesterol of the body is synthesized (about 1 gm/day) whereas about 0.3 gm/day is provided by the average diet. Plant synthesize phytosterol but not cholesterol. So dietary cholesterol is obtained only from animal sources like meat, liver, brain and egg yolk (a particularly rich source). Cholesterol is a white, waxy, solid found associated with fats but chemically different from them. It has a parent nucleus which is called cyclopentanoperhydrophenanthrene nucleus, with a hydroxyl group at C_3, an unsaturated bond at C_5-C_6, two methyl groups at C_10 and C_13, and 8 carbon paraffin side-chains attached to C_17. Cholesterol may occur free, or sometimes combined with fatty acids by ester linkage at the hydroxyl group (‘bound’ cholesterol esters).
Cholesterol is an essential constituent of cells. It aids in the permeability of the cells, controls the red cells from being easily hemolyzed and functions as defensive action. It assists formation of bile acids and bile salts, 7-dehydrocholesterol and vitamin D₃, corticosteroid hormones, androgens (male sex hormones), estrogens and progesterone (female sex hormones) and helps granulation of cell division; it also acts as an antagonist to phospholipid.

Atherosclerosis is characterized by the deposition of cholesteryl ester and other lipids in the connective tissue of the arterial walls. Diseases in which prolonged elevated levels of LDL-C and very low density lipoprotein cholesterol (VLDL-C) occur in the blood are accompanied by severe atherosclerosis. Rise in plasma free fatty acids also leads to increased VLDL-C secretion by the liver, causing extra triacylglycerol and cholesterol output into the circulation. Factors leading to higher levels of free fatty acids include emotional stress, nicotine from cigarette smoking, coffee drinking and partaking of few large meals rather than more continuous feeding. Atherosclerosis occurs due to hypercholesterolemia or more accurately hyper-β-lipoproteinemia. Recent
epidemiological data suggests a negative correlation between HDL cholesterol in human plasma and risks of premature heart disease. HDL deficiency is an independent risk factor for the incidence of early cardiovascular disease. The basic lesion is the formation of plaque in the smooth muscle cells of arterial wall consisting of cholesterol and lipids. These plaques later become fibrous, sclerosed and calcified, and eventually progressive thrombosis occurs at that site. It is due to this, the thrombosis of cerebral and coronary arteries develops.

The Low Density Lipoproteins (LDL) are heterogeneous population of spherical particles, with hydrophobic oily cores consisting of triglycerides (Tg) and cholesteryl ester. These particles are coated with a native surfactant of phospholipids, free cholesterol and apolipoproteins. On an average, LDL carries two-thirds of the total cholesterol (TC) in serum. Each LDL-C particle contains one molecule of apolipoprotein B-100 (apo B-100), which is the main protein component of LDL-C, and the other minor apolipoproteins are apo E and apo C II. By definition, LDL-C comprises the population of particles with hydrated density between 1.006 and 1.063 kg/L. As per definition to LDL-C is separated according to sequential density by ultra centrifugation, or by the so-called beta quantification method combining ultra centrifugation and chemical precipitation, which is the basis for measurement in most epidemiological studies. This widely variable density LDL-C population is heterogeneous, including remnant particles of intermediate density lipoproteins (IDL-C, 1.006-1.019 kg/L) and lipoprotein (a) [Lp (a), 1.050-1.080 kg/L]. The remaining LDL-C particles can be classified as light or heavy, the latter being considered as more atherogenic. In practice, all of these particles as a whole are atherogenic, because of wide density population of LDL (1.006 - 1.063 kg/L) as it is usually reported. Epidemiological and clinical studies have demonstrated a strong convincing correlation between low-density lipoprotein cholesterol (LDL-C) concentrations in serum and the incidence of coronary artery disease (CAD). Pathological studies have shown that increased LDL-C concentrations correlate highly with the extent of atherosclerotic lesions. On the other hand a reduction of LDL-C decreases the risk and ameliorates
the symptoms of CAD by causing a regression in the lesions. A comprehensive idea of the physical characters of five classes of lipoproteins discussed are tabulated here.

Table: 1

<table>
<thead>
<tr>
<th>Lipoprotein</th>
<th>Molecular mass (kDa)</th>
<th>Density kg/L</th>
<th>% Protein</th>
<th>Major lipids</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chylomicrons</td>
<td>&gt;400000</td>
<td>&lt;0.95</td>
<td>1.5 – 2.5</td>
<td>Tg</td>
</tr>
<tr>
<td>VLDLs</td>
<td>10000 – 80000</td>
<td>&lt;1.006</td>
<td>5 – 10</td>
<td>Tg, PL, CE</td>
</tr>
<tr>
<td>IDLs</td>
<td>5000 – 10000</td>
<td>1.006 – 1.019</td>
<td>15 – 20</td>
<td>CE, Tg, PL</td>
</tr>
<tr>
<td>LDLs</td>
<td>2300</td>
<td>1.019 – 1.210</td>
<td>20 – 25</td>
<td>CE, PL</td>
</tr>
<tr>
<td>HDLs</td>
<td>175 – 360</td>
<td>1.063 – 1.210</td>
<td>40 – 55</td>
<td>PL, CE</td>
</tr>
</tbody>
</table>

C = Cholesterol; CE = Cholesterol ester; Tg = Triglyceride; PL = Phospholipid

Gofmann et al. first reported the association between high level of triglyceride and their relation to coronary artery disease. In 1959, Albrink et al. reported that fasting triglyceride levels were higher in patients with CAD compared to controls. After that there are many studies collaboratively supported that plasma triglyceride levels are strongly associated with an increased risk of coronary disease. But in few studies, multivariate analysis did not identify triglyceride as an independent risk factor after controlling total cholesterol or HDL. Exact pathogenic role of triglyceride in coronary artery disease still remains nebulous. Association of several direct and indirect mechanisms, including effects on the metabolism of lipoproteins, transport proteins and enzymes, and the coagulation system conjointly playing an important role in the pathogenesis of CAD is proved. But surprisingly, the status of triglyceride, as an independent risk factor still remains controversial. In various metabolic and clinical conditions high level of triglyceride is increasingly recognized as a good marker, such as post prandial lipemia, insulin resistance, hyperinsulinemia, decreased HDL-C, elevated LDL-C, poorly controlled diabetes, and central obesity, all of which are
associated with an increased risk of atherosclerosis\textsuperscript{34}. Triglyceride (Tg) itself does not accumulate in the lumen of vessel wall but may be atherogenic due to the fact that Tg circulates in the plasma as part of the complex lipoproteins. The atherogenic role of Tg rich lipoproteins VLDL-C, LDL-C, chylomicrons and their remnants is established, and it is proved that VLDL-C and chylomicrons or their remnants enter the subendothelium and accumulate leading to atherosclerotic plaque formation. In hypertriglyceridemia, when VLDL-C production is over, it leads to form an abnormal lipoprotein profile and contributes to atherosclerosis in human atheromata. Very high Tg levels are associated with excessive alcohol consumption and genetic predisposition with inherited structural mutations in lipoprotein lipase (LPL) or apolipoprotein C II, a cofactor for LPL. A very high Tg level is less likely to be associated with atherogenesis because it is believed that the vascular endothelium is impermeable to the large uncatabolized Tg-rich lipoproteins\textsuperscript{35}.

However, there is some evidence for Tg as an independent risk factor in certain subgroups; for example, women 50–69 years of age\textsuperscript{36}, men with low total cholesterol levels\textsuperscript{37}, and patients with type 2 diabetes mellitus\textsuperscript{38}. These findings are supported by West\textsuperscript{39} and Fontbonne\textsuperscript{40}. A study conducted on migrant Indians has stressed the role of elevated serum Tg as a risk factor for CAD in this ethnic group\textsuperscript{41}. Also, there are studies, reporting contradictorily about the independent role of elevated serum Tg as a risk factor for CAD\textsuperscript{30,42-45}. So, in the present study there exists an excellent opportunity in an attempt to explore the prevailing contradictions.

An inverse association exists between serum concentrations of HDL-C and CAD risk\textsuperscript{16}. In 1975 Miller and Miller\textsuperscript{47} emphasized the inverse relationship between plasma high-density lipoprotein cholesterol (HDL-C) concentration and coronary artery disease (CAD) and since then prospective studies in several countries have confirmed this relationship and found HDL-C to be independent of other risk factors\textsuperscript{48-50}. It was reported that the Indians not only had the lowest serum levels of HDL-C but also the highest levels of lipoprotein (a) i.e. Lp (a) compared to the other ethnic groups\textsuperscript{51}. One of the mechanisms by which HDL-C protects against CAD is through reverse transport of
cholesterol whereby HDL-C promotes the efflux of cholesterol from cells, bringing it to the liver to be metabolized into bile acids and then excreted\textsuperscript{52}.

This was controversial to that demonstrated by the British Regional Heart Study which found that HDL-C was not a major risk factor in the etiology of ischemic heart disease (IHD) in British men\textsuperscript{53}. However, there are probably cross-cultural differences on the degree of influence of HDL-C on the pathogenesis of CAD. The present study is conducted with particular reference to the clinical value of serum HDL-C in the assessment of risk to CAD in the people of eastern India in view of the aforesaid conflicting reports on the index.

However, the relationship between coronary artery disease and various lipid parameters along with their normal ranges, the stage of progression or regression of the disease process still suggests to conduct further studies. The aim of the present study is to investigate and evaluate further relation of lipid profile levels their changing patterns under the influence of altered physiological condition in vivo and hence predisposing to various stages of CAD.
MATERIALS AND METHODS

Male and female participants aged between 20 and 80 years are invited to take part in the study. The exclusion criteria included for selection of participant are:

i) A self-reported history of heart disease;
ii) Under medication for hypertension, liver, heart or renal disease;
iii) Weight gain or weight loss regimes;
iv) Smoking;
v) Pregnancy;

Participants in this study are volunteers who are the patients of various outpatient and inpatients clinics like Calcutta Heart Clinic & Hospital, and IPGME&R. For all studies, verbal and written consent are obtained from all patients.

Fasting venous blood samples from healthy individuals, collected in tubes without anticoagulant, are allowed to clot in the dark at room temperature, and then centrifuged at 2500 rpm at 4°C for 20 min. The separated serum is frozen at -80°C for later analysis of lipid profile. The parameters investigated are detailed in material and method chapter (Chapter III).

i) Serum total cholesterol is estimated by CHOD-PAP method. The assay is performed using commercially available “LABKIT” reagent.

ii) HDL cholesterol is estimated by phosphotungstate method using commercially available reagent of Bayer Diagnostic Limited.

iii) Serum LDL cholesterol is estimated by a homogeneous method based on an innovative detergent technology. In the present study, commercially available kit of Daiichi Pure Chemicals Co., Ltd, Tokyo, Japan is used.

iv) The FASTER I (Fast Assessment of Thoracic Pain by nEuRal networks)-
This study is conducted between 2005 and 2007 at two investigational centers in Kolkata. The inclusion criterion is admittance to the CCU because of chest pain with duration of 15 minutes within the last 8 hours, susceptible of an ACS. This study received such patients, having an exclusive criteria of pathological ST-segment elevation on the admission 12-lead ECG or strong
suspicion of acute myocarditis. In case of any cTnI elevation 0.1 g/L within the first 12 hours, a last sample for analysis of cTnI and CK-MB is drawn. The Access AccuTnI assay is a sandwich immunoassay with a 99th percentile of 0.04 g/L among healthy subjects regardless of age and 0.021 g/L among subjects. The lowest concentration measurable with a 10% CV is 0.06 g/L. 54,55.
RESULTS

TABLE NO. IV.1

Serum lipid profile levels (Mean ± SD) of male

Age-20-40 years

<table>
<thead>
<tr>
<th></th>
<th>No.</th>
<th>Chol (mg/dl)</th>
<th>Tg (mg/dl)</th>
<th>HDL-C (mg/dl)</th>
<th>LDL-C (mg/dl)</th>
<th>Chol/HDL-C</th>
<th>LDL-C/HDL-C</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>55</td>
<td>185.0±40.0</td>
<td>163.0±72.0</td>
<td>39.4±8.4</td>
<td>112.4±39.0</td>
<td>4.8±1.0</td>
<td>2.97±1.2</td>
</tr>
<tr>
<td>Diabetic</td>
<td>50</td>
<td>192±38.6</td>
<td>155±80.5</td>
<td>44.8±10.6*</td>
<td>121±38.1*</td>
<td>4.7±2.2</td>
<td>3.0±1.9</td>
</tr>
<tr>
<td>Over weight</td>
<td>55</td>
<td>199±15.9</td>
<td>178±22.1</td>
<td>37±5.2</td>
<td>124±21</td>
<td>5.2±0.46</td>
<td>3.2±0.52</td>
</tr>
</tbody>
</table>

Level of significant *p< 0.05,  ** p< 0.01,  ***p< 0.001
No. = Total no. of participant  Chol = Cholesterol
TABLE NO. IV.2

Serum lipid profile levels (Mean ± SD) of female
Age-20-40 years

<table>
<thead>
<tr>
<th></th>
<th>No.</th>
<th>Chol (mg/dl)</th>
<th>Tg (mg/dl)</th>
<th>HDL-C (mg/dl)</th>
<th>LDL-C (mg/dl)</th>
<th>Chol/HDL-C</th>
<th>LDL-C/HDL-C</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>55</td>
<td>186±42.8</td>
<td>120±52.3</td>
<td>42.9±8.3</td>
<td>121±35.6</td>
<td>4.47±0.99</td>
<td>2.9±0.9</td>
</tr>
<tr>
<td>Diabetic</td>
<td>50</td>
<td>209±41</td>
<td>146±60*</td>
<td>51±8</td>
<td>129±33</td>
<td>4.0±0.6</td>
<td>2.5±0.5</td>
</tr>
<tr>
<td>Overweight</td>
<td>55</td>
<td>216±19*</td>
<td>254±44***</td>
<td>36.9±3.3</td>
<td>129±13</td>
<td>5.8±0.39*</td>
<td>3.5±0.34</td>
</tr>
</tbody>
</table>

Level of significant *p< 0.05, ** p< 0.01, ***p< 0.001

No. = Total no. of participant  Chol = Cholesterol
TABLE NO. IV.3

Serum lipid profile levels (Mean ± SD) of male
Age-41-60 years

<table>
<thead>
<tr>
<th>No.</th>
<th>Chol (mg/dl)</th>
<th>Tg (mg/dl)</th>
<th>HDL-C (mg/dl)</th>
<th>LDL-C (mg/dl)</th>
<th>Chol/HDL-C</th>
<th>LDL-C/HDL-C</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>55</td>
<td>181±41.6</td>
<td>160±45</td>
<td>40±9.9</td>
<td>111±35.8</td>
<td>4.8±1.6</td>
</tr>
<tr>
<td>Diabetic</td>
<td>50</td>
<td>184±42.8</td>
<td>151±50.9</td>
<td>41.1±11</td>
<td>115±31</td>
<td>4.7±1.2</td>
</tr>
<tr>
<td>Over weight</td>
<td>55</td>
<td>177±37.9</td>
<td>135±49</td>
<td>35±4.9*</td>
<td>131±24*</td>
<td>4.6±1.6</td>
</tr>
<tr>
<td>Hypertensive</td>
<td>45</td>
<td>190±20</td>
<td>158±34</td>
<td>39±5.3</td>
<td>110.5±25</td>
<td>4.8±0.4</td>
</tr>
</tbody>
</table>

Level of significant *p< 0.05,  **p< 0.01,

No. = Total no. of participant  Chol = Cholesterol
TABLE NO. IV.4

Serum lipid profile levels (Mean ± SD) of female

Age-41-60 years

<table>
<thead>
<tr>
<th>No.</th>
<th>Chol (mg/dl)</th>
<th>Tg (mg/dl)</th>
<th>HDL-C (mg/dl)</th>
<th>LDL-C (mg/dl)</th>
<th>Chol/HDL-C</th>
<th>LDL-C/HDL-C</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>80</td>
<td>165±52.1</td>
<td>108±52.1</td>
<td>43.3±13.7</td>
<td>100.6±43.7</td>
<td>4.1±1.1</td>
</tr>
<tr>
<td>Diabetic</td>
<td>75</td>
<td>189±45</td>
<td>144±60</td>
<td>50±8.6</td>
<td>118±38</td>
<td>4.0±1.0</td>
</tr>
<tr>
<td>Over weight</td>
<td>80</td>
<td>218±51.3**</td>
<td>205±45***</td>
<td>42.2±7.4</td>
<td>130±43</td>
<td>5.5±1.2</td>
</tr>
<tr>
<td>Hypertensive</td>
<td>75</td>
<td>223±10.9***</td>
<td>140±40.7</td>
<td>37±4.4**</td>
<td>134±25**</td>
<td>5.6±1.3</td>
</tr>
</tbody>
</table>

Level of significant *p<0.05, ** p<0.01, ***p<0.001
No. = Total no. of participant  Chol = Cholesterol
TABLE NO. IV.5

Serum lipid profile levels (Mean ± SD) of male

Age-61-80 years

<table>
<thead>
<tr>
<th></th>
<th>No.</th>
<th>Chol (mg/dl)</th>
<th>Tg (mg/dl)</th>
<th>HDL-C (mg/dl)</th>
<th>LDL-C (mg/dl)</th>
<th>Chol/HDL-C</th>
<th>LDL-C/HDL-C</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>198</td>
<td>165±52.1</td>
<td>108±52.1</td>
<td>43.3±13.7</td>
<td>100.6±43.7</td>
<td>4.1±1.1</td>
<td>2.5±1.2</td>
</tr>
<tr>
<td>Diabetic</td>
<td>80</td>
<td>175.3±45</td>
<td>118±55</td>
<td>39±10**</td>
<td>95±42.7</td>
<td>3.5±1.4</td>
<td>2.4±1.2</td>
</tr>
<tr>
<td>Over weight</td>
<td>80</td>
<td>183±53</td>
<td>152±42</td>
<td>41±8.0</td>
<td>120±44</td>
<td>4.7±1.5</td>
<td>3.1±1.2</td>
</tr>
<tr>
<td>Hypertensive</td>
<td>75</td>
<td>172±43.6</td>
<td>123±37.5</td>
<td>39.8±8.2**</td>
<td>107.1±37*</td>
<td>4.0±1.0</td>
<td>2.4±1.1</td>
</tr>
</tbody>
</table>

Level of significant *p< 0.05,  **p< 0.01,
No. = Total no. of participant  Chol = Cholesterol
TABLE NO. IV.6

Serum lipid profile levels (Mean ± SD) of female

Age-61-80 years

<table>
<thead>
<tr>
<th></th>
<th>No.</th>
<th>Chol (mg/dl)</th>
<th>Tg (mg/dl)</th>
<th>HDL-C (mg/dl)</th>
<th>LDL-C (mg/dl)</th>
<th>Chol/HDL-C</th>
<th>LDL-C/HDL-C</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>135</td>
<td>179 ±45.3</td>
<td>111 ±50</td>
<td>47±11.7</td>
<td>109.8±30.1</td>
<td>3.9±1.1</td>
<td>2.4±0.98</td>
</tr>
<tr>
<td>Diabetic</td>
<td>80</td>
<td>189 ±39</td>
<td>128 ±44**</td>
<td>39±8.6**</td>
<td>121±15**</td>
<td>4.2±1.2</td>
<td>2.8±1.0</td>
</tr>
<tr>
<td>Over weight</td>
<td>80</td>
<td>183 ±45</td>
<td>129 ±52</td>
<td>51±14</td>
<td>107±40</td>
<td>3.8±1.3</td>
<td>2.3±1.2</td>
</tr>
<tr>
<td>Hypertensive</td>
<td>85</td>
<td>194.2 ±15.9**</td>
<td>130 ±16.9**</td>
<td>53±8.5</td>
<td>129±24.8</td>
<td>3.9±0.9</td>
<td>2.4±0.6</td>
</tr>
</tbody>
</table>

Level of significant *p< 0.05, ** p< 0.01,
No. = Total no. of participant  Chol = Cholesterol
On the basis of experimental results of this study incorporated in tables IV.1 to IV.6, a consolidated data profile is appended here with an intention to have a comprehensive idea of the levels of lipid profile i.e., cholesterol, Tg, HDL-C, LDL-C, chol/HDL-C, LDL-C/HDL-C in different age groups of male and female.

In respect of lipid profile in male and female age group of 20-40 years:
- Cholesterol levels are 185.0±40.0 and 186±42.8
- Tg levels are 163.0±72.0 and 120±52.3
- HDL-C levels are 39.4±8.4 and 42.9±8.3
- LDL-C levels are 112.4±39.0 and 121±35.6
- Chol/HDL-C levels are 4.8±1.0 and 4.47±0.99
- LDL-C/HDL-C levels are 2.97±1.2 and 2.9±0.9

In respect of lipid profile in male and female age group of 41-60 years:
- Cholesterol levels are 181±41.6 and 201±47.3
- Tg levels are 160±45 and 141±50.1
- HDL-C levels are 40±9.9 and 45±9.5
- LDL-C levels are 111±35.8 and 128±42.6
- Chol/HDL-C levels are 4.8±1.6 and 4.7±1.5
- LDL-C/HDL-C levels are 2.96±1.3 and 2.5±1.2

In respect of lipid profile in male and female age group of 61-80 years:
- Cholesterol levels are 165.3±52.1 and 179±45.3
- Tg levels are 108±52.1 and 111±50
- HDL-C levels are 43.3±13.7 and 47±11.7
- LDL-C levels are 100.6±43.7 and 109.8±30.1
- Chol/HDL-C levels are 4.1±1.1 and 3.9±1.1
- LDL-C/HDL-C levels are 2.5±1.2 and 2.4±0.98

In view of the above comparative findings in respect of same age group of male and female, reveal no significant changes.
Fig. IV.1: Levels of lipid profile in diabetic groups

Fig. IV.2: Cholesterol and lipoprotein ratio in diabetic groups

Fig. IV.1 and Fig. IV.2 are showing the levels of lipid profile in diabetic groups
The findings incorporated in tables IV.1 to IV.6 and (Fig. IV.1 and IV.2), a consolidated information of the levels of lipid profile i.e., cholesterol, Tg, HDL-C, LDL-C, chol/HDL-C, LDL-C/HDL-C in different age groups of male and female diabetic.

In respect of lipid profile in male and female diabetics of 20-40 years age group:

- Cholesterol levels are 192±38.6 and 209±41
- Tg levels are 155±80.5 and 146±60
- HDL-C levels are 44.8±10.6 and 51±8
- LDL-C levels are 121±38.1 and 129±33
- Chol/HDL-C levels are 4.7±2.2 and 4.0±0.6
- LDL-C/HDL-C levels are 3.0±1.9 and 2.5±0.5

In respect of lipid profile in male and female diabetics of 41-60 years age group:

- Cholesterol levels are 184±42.8 and 189±45
- Tg levels are 151±50.9 and 144±60
- HDL-C levels are 41.1±11 and 50±8.6
- LDL-C levels are 115±31 and 118±38
- Chol/HDL-C levels are 4.7±1.2 and 4.0±1.0
- LDL-C/HDL-C levels are 3.0±1.2 and 2.4±1.0

In respect of lipid profile in male and female diabetics of 61-80 years age group:

- Cholesterol levels are 175.3±45 and 189±39
- Tg levels are 118±55 and 128±44
- HDL-C levels are 39±10 and 39±8.6
- LDL-C levels are 95±42.7 and 121±15
- Chol/HDL-C levels are 3.5±1.4 and 4.2±1.2
- LDL-C/HDL-C levels are 2.4±1.2 and 2.8±1.0
In view of the above findings a comparison is made in respect of male and female diabetic of same age groups of 20-40 years and no significant change (p>0.05) is observed. But when compared with same age groups of control it is observed that HDL-C is found significantly (p<0.05) lower and LDL-C is significantly higher (p<0.05) in diabetic males and the level of Tg is significantly (p<0.05) higher in diabetic females.

When a comparison is made in respect of male and female diabetic of same age groups (41-60 years), no significant change is observed.

Another comparison is made between male and female diabetics, in respect of age groups of 61-80 years, only the level of LDL-C is significantly (p<0.01) higher in females than males. The level of HDL is significantly lower (p<0.01), LDL-C and Tg are significantly higher (p<0.01) in female diabetics than the control group. No other significant change is observed in any other parameter in this group.
Fig. IV.3 and Fig. IV.4 are showing the lipid profile level of 41-60 years of female hypertensive and control group.
The experimental results of lipid profile i.e., cholesterol, Tg, HDL-C, LDL-C, chol/HDL-C, LDL-C/HDL-C in different age groups of hypertensive male and female are incorporated in the tables IV.1 to IV.6.

In respect of lipid profile in hypertensive male and female 41-60 years age group:

- Cholesterol levels are 190±20 and 209±41
- Tg levels are 158±34 and 140±40.7
- HDL-C levels are 39±5.3 and 37±4.4
- LDL-C levels are 110.5±25 and 134±25
- Chol/HDL-C levels are 4.8±0.4 and 5.6±1.3
- LDL-C/HDL-C levels are 2.8±.63 and 3.8±.78

In respect of lipid profile in hypertensive male and female 61-80 years age group:

- Cholesterol levels are 172±43.6 and 194.25±15.9
- Tg levels are 123±37.5 and 130±16.9
- HDL-C levels are 39.8±8.2 and 53±8.5
- LDL-C levels are 107.1±37 and 129±24.8
- Chol/HDL-C levels are 4.0±1.0 and 3.9±0.9
- LDL-C/HDL-C levels are 2.4±1.1 and 2.4±0.6

A comparison is made from the above findings in respect to male and female hypertensive groups of 41-60 years. It is observed that the levels of cholesterol and LDL-C are significantly higher in females (p<0.05) than the males. Along with it cholesterol/HDL-C and LDL-C/ HDL-C are higher in females compared to the males but no significant correlation could be drawn between them. When hypertensive males and females are compared with control groups the level of cholesterol is significantly (p<0.001) higher and HDL-C is significantly (p<0.01) lower in female hypertensive group compared to control group.

It is observed from the above findings, in respect of male and female hypertensive of same age groups of 61-80 years that the level of cholesterol and LDL-C are significantly (p<0.05) higher in female groups than the male groups and HDL-C is significantly (p<0.01) lower in male groups than the female groups. Again when a
comparison is made in male and female hypertensive groups with same age group of control the levels of cholesterol and Tg are significantly (p<0.01) higher in hypertensive female groups than the control groups but no significant correlation is observed in hypertensive male groups.
Fig. IV.5 and Fig. IV.6 are showing lipid profile levels of overweight female of 20-40 years age group against control group.
From the tables IV.1 to IV.6 a consolidated findings are appended here to derive a comprehensive idea of the levels of lipid profile i.e., cholesterol, Tg, HDL-C, LDL-C, chol/HDL-C, LDL-C/HDL-C in different age groups of overweight male and female.

In overweight male and female 20-40 years of age groups:

- Cholesterol levels are 199±15.9 and 216±19
- Tg levels are 178±22.1 and 254±44
- HDL-C levels are 37±5.2 and 36.9±3.3
- LDL-C levels are 124±21 and 129±33
- Chol/HDL-C levels are 5.2±0.46 and 5.8±0.39
- LDL-C/HDL-C levels are 3.2± 0.52 and 3.5± 0.34
- And their BMI levels are 28.9±1.5 and 29.8±2.4

In overweight male and female 41-60 years age of groups:

- Cholesterol levels are 177±37.9 and 218±51.3
- Tg levels are 135±49 and 205±45
- HDL-C levels are 35±4.9 and 42.2±7.4
- LDL-C levels are 131±24 and 130±43
- Chol/HDL-C levels are 4.6±1.6 and 5.5±1.2
- LDL-C/HDL-C levels are 2.9± 1.4 and 4.5± 0.94
- And their BMI levels are 28±1.6 and 29±2.2

In overweight male and female 61-80 years age of groups:

- Cholesterol levels are 183±53 and 183±45
- Tg levels are 152±42 and 129±52
- HDL-C levels are 41±8.0 and 51±14
- LDL-C levels are 120±44 and 107±40
- Chol/HDL-C levels are 4.7±1.5 and 3.8±1.3
- LDL-C/HDL-C levels are 3.1± 1.2 and 2.3± 1.2
- And their BMI levels are 27.9±2.4 and 28±3.0
In respect of the above observation of lipid profile in contrast of male and female overweight groups of same age, the level of cholesterol (p<0.05) and Tg (p<0.001) are significantly higher in female than in male of 20 – 40 years of age group. Again the same age group of overweight male is compared with the control group where the level of cholesterol is significantly (p<0.01) higher and when female are compared with the control group, cholesterol and Tg levels are significantly (p<0.01) higher and HDL-C is significantly (p<0.01) lower.

In overweight male 41-60 years of age group, significantly (p<0.05) higher level of LDL-C and lower level (p<0.05) of HDL-C are observed in comparison to control group. The level of Tg is found to be significantly higher (p<0.05) and (p<0.01) in male and female age group of 61-80 years against the control groups. No other significant changes are found in other parameters.

Tg is significantly higher (p<0.05) in 61-80 years of male and female groups in comparison to the control group. The level of HDL-C is significantly (p<0.05) lower and LDL-C is significantly higher in the same age group of males when compared with the controls. No other significant changes are found.
Relation between cholesterol and lipid fractions in different male experimental groups

Fig. IV-6a: showing the relation between cholesterol and lipid fraction in different male experimental groups
Fig. IV-6b: showing the relation between cholesterol and lipid fraction in different female experimental groups.
### TABLE NO. IV.7

**Serum lipid profile levels (Mean ± SD) of myocardial infarcted against control group**

<table>
<thead>
<tr>
<th></th>
<th>No.</th>
<th>Chol (mg/dl)</th>
<th>Tg (mg/dl)</th>
<th>HDL-C (mg/dl)</th>
<th>LDL-C (mg/dl)</th>
<th>Chol/HDL-C</th>
<th>LDL-C/HDL-C</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Myocardial infarcted</strong></td>
<td>80</td>
<td>235 ±39.9**</td>
<td>205 ±40**</td>
<td>34 ±6.1***</td>
<td>145 ±36.8**</td>
<td>6.1 ±1.6**</td>
<td>4.7±1.4**</td>
</tr>
<tr>
<td><strong>Control</strong></td>
<td>60</td>
<td>158.5 ±22.5</td>
<td>123 ±58</td>
<td>42.8 ±8.76</td>
<td>90.7 ±20.8</td>
<td>3.8 ±0.8</td>
<td>2.5 ±0.9</td>
</tr>
</tbody>
</table>

**Level of significant**  *p<0.05,  **p<0.01,

From the experimental results of this study incorporated in table IV.7, a consolidated findings are appended here to derive a comprehensive idea of the levels of lipid profile i.e., cholesterol, Tg, HDL-C, LDL-C, chol/HDL-C, LDL-C/HDL-C in myocardial infarcted group in relation to control group.

Cholesterol levels are 235±39.9 and 158.5±22.5
Tg levels are 205±40 and 123±58
HDL-C levels are 34±6.1 and 42.8±8.76
LDL-C levels are 145±36.8 and 90.7±20.8
Chol/HDL-C levels are 6.1±1.6 and 3.8±0.8
LDL-C/HDL-C levels are 4.7±1.4 and 2.5±0.9
Over and above their BMI levels are 26.1±3.1 and 23.1±2.3
And ages are 60±11.2 years and 55±17 years
A comparative discussion in respect of myocardial infarcted group and control group, the levels of cholesterol, Tg, LDL-C, cholesterol/HDL-C and LDL-C/HDL-C are significantly higher (p<0.01) than the control group. Level of HDL-C level is significantly lower (p<0.001) in myocardial infarcted group than control group.
Fig.IV.7 and Fig.IV.8 are showing lipid profile level of myocardial infarcted against control group.
TABLE NO. IV.8

Serum lipid profile levels (Mean ± SD) of male group with / without hypertension and diabetes

<table>
<thead>
<tr>
<th></th>
<th>No.</th>
<th>Chol (mg/dl)</th>
<th>Tg (mg/dl)</th>
<th>HDL-C (mg/dl)</th>
<th>LDL-C (mg/dl)</th>
<th>Chol/HDL-C</th>
<th>LDL-C/HDL-C</th>
</tr>
</thead>
<tbody>
<tr>
<td>With hypertension</td>
<td>80</td>
<td>217.6 ±28.9*</td>
<td>116±16.6</td>
<td>46.2±15.2</td>
<td>131.5±13.3**</td>
<td>4.3±1.3</td>
<td>3.2±1.6</td>
</tr>
<tr>
<td>and diabetes</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Without hypertension</td>
<td>65</td>
<td>150±25</td>
<td>135±32</td>
<td>36±6.0</td>
<td>73±20</td>
<td>4.4±2.2</td>
<td>2.6±1.9</td>
</tr>
<tr>
<td>and diabetes</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Level of significant *p<0.05, ** p< 0.01, *** p< 0.001,

TABLE NO. IV.9

Serum lipid profile levels (Mean ± SD) of female group with / without hypertension and diabetes

<table>
<thead>
<tr>
<th></th>
<th>No.</th>
<th>Chol (mg/dl)</th>
<th>Tg (mg/dl)</th>
<th>HDL-C (mg/dl)</th>
<th>LDL-C (mg/dl)</th>
<th>Chol/HDL-C</th>
<th>LDL-C/HDL-C</th>
</tr>
</thead>
<tbody>
<tr>
<td>With hypertension</td>
<td>80</td>
<td>228.6 ±19.9*</td>
<td>158±57</td>
<td>41.2±8.3</td>
<td>142.7±15.6***</td>
<td>5.0±1.4</td>
<td>3.0±1.4</td>
</tr>
<tr>
<td>and diabetes</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Without hypertension</td>
<td>65</td>
<td>150±25</td>
<td>135±32</td>
<td>36±6.0</td>
<td>73±20</td>
<td>4.4±2.2</td>
<td>2.6±1.9</td>
</tr>
<tr>
<td>and diabetes</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Level of significant *p<0.05, ** p< 0.01, *** p< 0.001,
The experimental results of this study incorporated in tables IV.8 to IV.9 depict the levels of lipid profile i.e., cholesterol, Tg, HDL-C, LDL-C, chol/HDL-C, LDL-C/HDL-C comparing individual group of male and female with hypertension and diabetes in relation to control groups without hypertension and diabetes respectively. These findings are consolidated hereunder for discussion.

In respect of lipid profile in male with hypertensive diabetic group (52.4±8.6 years) against without hypertension diabetes group (54±10.4 years):

- Cholesterol levels are 217.6±28.9 and 150±25
- Tg levels are 116±16.6 and 135±32
- HDL-C levels are 46.2±15.2 and 36±6.0
- LDL-C levels are 131.5±13.3 and 73±20
- Chol/HDL-C levels are 4.3±1.3 and 4.4±2.2
- LDL-C/HDL-C levels are 3.2±1.6 and 2.6±1.9

In respect of lipid profile in female with hypertensive diabetic group (50.5±5.9 years) against control (55±11 years) without hypertensive diabetic group:

- Cholesterol levels are 228.6±19.9 and 168.6±28.9
- Tg levels are 158±57 and 116±16.6
- HDL-C levels are 41.2±8.3 and 38±6.0
- LDL-C levels are 142.7±15.6 and 105±25
- Chol/HDL-C levels are 5.0±1.4 and 4.3±1.3
- LDL-C/HDL-C levels are 3.0±1.4 and 3.2±1.6

In view of the above a comparative discussion in respect of male with hypertension and diabetes, cholesterol and LDL-C are significantly high (p<0.05) and (p<0.001) respectively in comparison to the control group. It is evident that the ratio of HDL-C levels decreased (p<0.05) in reference to the above context.
Similar observations for all these parameters hold good in respect of female with hypertension and diabetes also except HDL-C.

**TABLE NO. IV.10**

Serum lipid profile levels (Mean ± SD) of male hypertensive, diabetic smoking against normotensive, non-diabetic, non-smoker

<table>
<thead>
<tr>
<th></th>
<th>No.</th>
<th>Chol (mg/dl)</th>
<th>Tg (mg/dl)</th>
<th>HDL-C (mg/dl)</th>
<th>LDL-C (mg/dl)</th>
<th>Chol/HDL-C</th>
<th>LDL-C/HDL-C</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypertensive diabetic, with smoking habit</td>
<td>80</td>
<td>225.8±56.2***</td>
<td>221.6±59.6***</td>
<td>30.1±5.3***</td>
<td>136.1±12.6***</td>
<td>7.5±1.6***</td>
<td>4.4±1.6***</td>
</tr>
<tr>
<td>Without hypertensive diabetes or smoking habit</td>
<td>80</td>
<td>158.5±22.5</td>
<td>123±58.9</td>
<td>42.8±8.76</td>
<td>90.7±20.8</td>
<td>3.8±0.8</td>
<td>2.5±0.9</td>
</tr>
</tbody>
</table>

Level of significant *** p< 0.001,
Fig. IV.11 and Fig. IV.12 showing the lipid profile levels of hypertensive diabetic smoker against control group.
From the experimental results of this study incorporated in table IV.10. The levels of lipid profile i.e., cholesterol, Tg, HDL-C, LDL-C, chol/HDL-C, LDL-C/HDL-C comparing male hypertensive diabetic smoker group (59.6±12.5 years) in relation to control group (59.6±12.5 years). The findings are consolidated hereunder for discussion.

Cholesterol levels are 225.8±56.2 and 158.5±22.5
Tg levels are 221.6±59.6 and 123±58.9
HDL-C levels are 30.1±5.3 and 42.8±8.76
LDL-C levels are 136.1±12.6 and 90.7±20.8
Chol/HDL-C levels are 7.5±1.6 and 3.8±0.8
LDL-C/HDL-C levels are 4.4±1.6 and 2.5±0.9

A comparative discussion in respect of male hypertensive diabetic smoker group reveals that cholesterol, Tg, LDL-C, cholesterol/HDL-C and LDL-C/HDL-C are significantly higher (p<0.001) compared to the control group. It is evident that the level of HDL-C is significantly lower (p<0.05) in reference to the above context.
**TABLE NO. IV.11**

Serum lipid profile levels (Mean ± SD) of hypercholesterimic and normocholesterimic male groups

| No.
| Chol (mg/dl) | Tg (mg/dl) | HDL-C (mg/dl) | LDL-C (mg/dl) | Chol/HDL-C  | LDL-C/HDL-C |
|----------|-------------|-------------|--------------|--------------|------------|------------|
| Hypercholesterimic group | 80 | 277±36.2*** | 229±35.1*** | 42.5±11.6 | 190±45 | 7.0±2.5*** | 4.9±2.0*** |
| Normocholesterimic group | 80 | 138±18 | 107±58 | 41±13 | 77±19 | 3.6±0.97 | 2.0±0.8 |

Level of significant *** p< 0.001,
Fig. IV.13

Fig. IV.13A

Fig. IV.13 and IV.13 A are showing lipid profile level of hypercholesterimic male and normocholesterimic male groups.
### TABLE NO. IV.12

Serum lipid profile levels (Mean ± SD) of hypercholesterimic and normocholesterimic female groups

<table>
<thead>
<tr>
<th></th>
<th>No.</th>
<th>Chol (mg/dl)</th>
<th>Tg (mg/dl)</th>
<th>HDL-C (mg/dl)</th>
<th>LDL-C (mg/dl)</th>
<th>Chol/HDL-C</th>
<th>LDL-C/HDL-C</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Hypercholesterimic group</strong></td>
<td>80</td>
<td>286.2±21***</td>
<td>163±65.1**</td>
<td>48 ± 9.7</td>
<td>188 ± 26.3</td>
<td>5.7±1.3***</td>
<td>4.1±1.2***</td>
</tr>
<tr>
<td><strong>Normocholesterimic group</strong></td>
<td>80</td>
<td>140±25</td>
<td>91±33</td>
<td>48 ± 11</td>
<td>79 ± 17</td>
<td>3.8±1.3</td>
<td>2.5±1.1</td>
</tr>
</tbody>
</table>

Level of significant **p< 0.01, *** p< 0.001,
Fig. IV.14 showing lipid profile level of hypercholesterimic female against normocholesterimic female

Fig. IV.15 Lipid profile status of hypercholesteremic male and female

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Fig.IV.15 and Fig.IV.16 are showing the lipid profile levels of hypercholesterimic male and female groups.
From the experimental results of this study incorporated in tables IV.11 and IV.12 the levels of lipid profile i.e., cholesterol, Tg, HDL-C, LDL-C, chol/HDL-C, LDL-C/HDL-C in male and female hypercholesteremic groups in relation to control groups are consolidated hereunder for discussion.

In respect of lipid profile in male and female hypercholesterimic groups:

- Cholesterol levels are 277±36.2 and 286.2±21
- Tg levels are 229±35.1 and 163±65.1
- HDL-C levels are 42.5±11.6 and 48±9.7
- LDL-C levels are 190±45 and 188±26.3
- Chol/HDL-C levels are 7.0±2.5 and 5.7±1.3
- LDL-C/HDL-C levels are 4.9±2.0 and 4.1±1.2

In view of the above findings a comparative discussion in respect of male and female groups apparently indicate no significant difference (>0.35) except in the males, significantly high (<0.05) levels of Tg and Chol/HDL-C are observed.

But when hypercholesterimic male and female groups are compared with normcholesterimic control groups, significantly (p<0.001) high levels of Tg, LDL-C, chol/HDL-C and LDL-C/HDL-C are found. No significant correlation is observed in case of HDL-C.
### TABLE NO. IV.13

Serum lipid profile levels (Mean ± SD) of male hypertriglyceridemic groups against normo triglyceridemic groups

<table>
<thead>
<tr>
<th></th>
<th>No.</th>
<th>Chol (mg/dl)</th>
<th>Tg (mg/dl)</th>
<th>HDL-C (mg/dl)</th>
<th>LDL-C (mg/dl)</th>
<th>Chol/HDL-C</th>
<th>LDL-C/HDL-C</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Hypertriglyceridemic</strong></td>
<td>280</td>
<td>203 ± 47***</td>
<td>228± 15***</td>
<td>37±8.7</td>
<td>117±46</td>
<td>5.4±1.7</td>
<td>3.2±1.5</td>
</tr>
<tr>
<td><strong>Normotriglyceridemic</strong></td>
<td>245</td>
<td>169±42</td>
<td>106±37</td>
<td>41±13.1</td>
<td>105±39</td>
<td>4.1±1.3</td>
<td>2.6±1.2</td>
</tr>
</tbody>
</table>

**Level of significant**  **p< 0.01, *** p< 0.001,**

### TABLE NO. IV.14

Serum lipid profile levels (Mean ± SD) of female

<table>
<thead>
<tr>
<th></th>
<th>No.</th>
<th>Chol (mg/dl)</th>
<th>Tg (mg/dl)</th>
<th>HDL-C (mg/dl)</th>
<th>LDL-C (mg/dl)</th>
<th>Chol/HDL-C</th>
<th>LDL-C/HDL-C</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Hypertriglyceridemic</strong></td>
<td>245</td>
<td>209±49</td>
<td>210±39</td>
<td>41±11</td>
<td>131±43</td>
<td>5.3±1.5</td>
<td>3.3±1.2</td>
</tr>
<tr>
<td><strong>Normotriglyceridemic</strong></td>
<td>250</td>
<td>128±20</td>
<td>112±29</td>
<td>39.5±10</td>
<td>72±21</td>
<td>3.7±1.1</td>
<td>2.0±1.0</td>
</tr>
</tbody>
</table>

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From the experimental results of this study incorporated in tables IV.13 to IV.14 the levels of lipid profile i.e., cholesterol, Tg, HDL-C, LDL-C, chol/HDL-C, LDL-C/HDL-C comparing male and female hypertriglyceridimic group in relation to normo triglyceridimic group are consolidated here under.

In respect of lipid profile in male hypertriglyceridimic group and normo triglyceridimic group:

- Cholesterol levels are 203 ±47 and 169±42
- Tg levels are 228±15 and 106±37
- HDL-C levels are 37±8.7 and 41±13.1
- LDL-C levels are 117±46 and 105±39
- Chol/HDL-C levels are 5.4±1.7 and 4.1±1.3
- LDL-C/HDL-C levels are 3.2±1.5 and 2.6±1.2

In respect of lipid profile in female hypertriglyceridimic group against normo triglyceridimic group:

- Cholesterol levels are 209±49 and 128±20
- Tg levels are 210±39 and 112±29
- HDL-C levels are 41±11 and 39.5±10
- LDL-C levels are 131±43 and 72±21
- Chol/HDL-C levels are 5.3±1.5 and 3.7±1.1
- LDL-C/HDL-C levels are 3.3±1.2 and 2.0±1.0

In view of the above findings in respect of male and female hypertriglyceridimic groups, cholesterol and LDL-C are significantly higher (p<0.001) in comparison to the normo triglyceridimic group. No other significant change is found in respect of other parameters in all groups.
### TABLE NO. IV.15

Serum lipid profile levels (Mean ± SD) of male high level of HDL-C groups against low level of HDL-C groups

<table>
<thead>
<tr>
<th></th>
<th>No.</th>
<th>Chol (mg/dl)</th>
<th>Tg (mg/dl)</th>
<th>HDL-C (mg/dl)</th>
<th>LDL-C (mg/dl)</th>
<th>Chol/HDL-C</th>
<th>LDL-C/HDL-C</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>High level of HDL-C group</strong></td>
<td>245</td>
<td>178±40</td>
<td>117±50</td>
<td>48±6.8</td>
<td>105±35</td>
<td>3.4±0.8</td>
<td>2.0±0.7</td>
</tr>
<tr>
<td><strong>Low level of HDL-C group</strong></td>
<td>285</td>
<td>171±51</td>
<td>172±45**</td>
<td>30±11*</td>
<td>110±44</td>
<td>5.7±1.8*</td>
<td>3.6±1.5*</td>
</tr>
</tbody>
</table>

Level of significant: *p<0.05, **p<0.01, ***p<0.001,

### TABLE NO. IV.16

Serum lipid profile levels (Mean ± SD) of female high level of HDL-C groups against low level of HDL-C groups

<table>
<thead>
<tr>
<th></th>
<th>No.</th>
<th>Chol (mg/dl)</th>
<th>Tg (mg/dl)</th>
<th>HDL-C (mg/dl)</th>
<th>LDL-C (mg/dl)</th>
<th>Chol/HDL-C</th>
<th>LDL-C/HDL-C</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>High level of HDL-C group</strong></td>
<td>285</td>
<td>197±42.7**</td>
<td>115±52</td>
<td>54±7.0**</td>
<td>121±39</td>
<td>3.7±0.8*</td>
<td>2.2±0.8*</td>
</tr>
<tr>
<td><strong>Low level of HDL-C group</strong></td>
<td>155</td>
<td>164±50</td>
<td>154±58</td>
<td>30±4.5</td>
<td>107±42</td>
<td>5.6±1.6</td>
<td>3.6±1.3</td>
</tr>
</tbody>
</table>

Level of significant: *p<0.05, **p<0.01,
From the experimental results of this study incorporated in tables IV.15 and IV.16 the levels of lipid profile i.e., cholesterol, Tg, HDL-C, LDL-C, chol/HDL-C, LDL-C/HDL-C comparing male and female with high level of HDL-C in relation to low level of HDL-C group the findings are consolidated hereunder.

In respect of lipid profile in male with low level of HDL-C against high level of HDL-C:

Cholesterol levels are 171±51 and 178±40  
Tg levels are 172±45 and 117±50  
HDL-C levels are 30±11 and 48±6.8  
LDL-C levels are 110±44 and 105±35  
Chol/HDL-C levels are 5.7±1.8 and 3.4±0.8  
LDL-C/HDL-C levels are 3.6±1.5 and 2.0±0.7

In respect of lipid profile in female with low level of HDL-C against high level of HDL-C:

Cholesterol levels are 164±50 and 197±42.7  
Tg levels are 154±58 and 115±52  
HDL-C levels are 30±4.5 and 54±7.0  
LDL-C levels are 107±42 and 121±39  
Chol/HDL-C levels are 5.6±1.6 and 3.7±0.8  
LDL-C/HDL-C levels are 3.6±1.3 and 2.2±0.8

It is evident from a comparative discussion that Tg, Chol/HDL-C and LDL-C/HDL-C are significantly high (p<0.05) with low level of HDL-C of male when compared to those of high level of HDL-C of male.

Also the levels of cholesterol, Tg, Chol/HDL-C and LDL-C/HDL-C are significantly higher (p<0.01) in the group of low level of HDL-C of female when compared to those of high level of HDL-C of female. No other significant correlation is observed in other cases.
DISCUSSION

Most of the epidemiological studies available on lipid levels and conventional risk factors of CAD on Indians are based on the data, relating to investigations performed on migrant Asian Indians. There have been few studies on Indians living in India (Native Indians) and those are available mostly from the studies conducted in South India and few in North India\textsuperscript{56-58}.

Epidemiological studies revealed a higher incidence of cardiovascular diseases in populations with high levels of total cholesterol (TC) and LDL-C, and low levels of HDL-C. In the present study, it is certainly proved to be correlated with the said proatherogenic lipids status which is also reasonably upheld by Tarchalski et al\textsuperscript{59}.

In various disease conditions it is observed that the Cholesterol/HDL-C ratio and LDL-C/HDL-C are more sensitive and more specific indicators than the total cholesterol, Tg and HDL-C level considered in isolation\textsuperscript{60,61}.

The present study is undertaken to elicit the significant and cogent risk factors of pathophysiological conditions and their intimate relation with coronary artery disease. Diabetes is a major risk factor in emigrant Indians with CHD\textsuperscript{62} and the present study highlights its importance. The most characteristic lipid abnormality in diabetics is hypertriglyceridaemia, with or without association with the increased levels of total cholesterol, LDL-C and decreased level of HDL cholesterol\textsuperscript{63,64}.

In tables at consecutive serial Nos. IV.1 to IV.6, it is observed in case of diabetics, an increase of LDL-C with simultaneous elevation of total cholesterol. In female age group of 41-60 years, male age group of 41-60 and 61-80 years, Tg level increases with increase of total cholesterol level. In case of male diabetics of 20-40 years of age group, it is observed that Tg level is positively correlated with total cholesterol level. And when comparison is made between the same age group of male and female diabetics, it is observed in the group of male that the level of HDL-C is significantly lower and LDL-C is significantly higher in diabetic males than in the same age groups of females and control male. Tg in female (20-40 years) is significantly higher in diabetics than in non-diabetics. In female diabetics (age group of 41-60 years), the level of HDL-C is seen to be significantly higher than the male group.
A high prevalence of diabetes in cases with CHD explains the diabetic dyslipidemia characterized by the lower level of HDL-C and higher level of triglycerides\textsuperscript{65}. Our present findings also corroborate the same observation. Fontbonne \textit{et al.}\textsuperscript{66} in a prospective cohort study also supports our observation that elevated plasma levels of Tg in diabetics are positively and significantly correlated with CAD events and CAD mortality\textsuperscript{67}. It has been suggested that the increased level of triglyceride may be due to insulin deficiency which results in faulty glucose utilization, causing hyperglycemia and mobilization of fatty acids from adipose tissue. In diabetes, blood glucose is not utilized by tissue resulting in hyperglycemia. The fatty acid from adipose tissue is mobilized for energy purpose and excess fatty acids are accumulated in the liver, which are converted into triglyceride\textsuperscript{68}. The results of this study are in tune with the above assertion.

Total cholesterol is increased in all groups of diabetics when compared with the control groups. Some of the plausible reasons of higher concentration of serum total cholesterol in diabetes may be attributed to decrease muscular exercise or inhibition of cholesterol catabolism. In the present study LDL-C is increased in diabetic groups. The most possible reason may be that insulin increases the number of LDL receptors, so chronic insulin deficiency might be associated with a diminished number of LDL receptor. This in turn causes increase in LDL-C particles and as a result there is an increase in LDL-C value in diabetes mellitus.

There are many more reasons for the higher risk of CAD from diabetes in women. These include differences in coagulation\textsuperscript{69}, the pattern of obesity and possible role for hyperinsulinemia\textsuperscript{70}. Diabetes may also alter estrogen related protective mechanisms\textsuperscript{71}. Furthermore, low-grade inflammation may have a greater role in perturbing insulin action in women, or inflammatory factors may interact with female sex hormones, resulting in a decrease of protective effects of estrogens on body fat distribution and insulin action\textsuperscript{72}. In stead of elaboration on this account it is suffice it to say that the underlying basis for the sex difference in associated risk from diabetes remains, for the most part, speculative.

Measurement of blood pressure is affected in the short term by many variables, including cigarette smoking, physical exercise, mental and physical stress and ambient temperature\textsuperscript{73} and is also viewed to be a subject of considerable variation. In spite of all
these, hypertension is already recognized as one of the major risk factors in the development of coronary atherosclerosis\textsuperscript{74}. It frequently coexists with other risk factors\textsuperscript{75-77} especially dyslipidemia, which may act synergistically in the pathogenesis of atherosclerotic disease\textsuperscript{78}. According to the present results, the levels of total cholesterol is significantly higher and HDL-C is lower in 41-60 year age group of hypertensive male group than in the normotensive male group, and the similar phenomenon is observed in the same age group of females also. But when compared, between the same age group (41-60 year) of males and females, total cholesterol, Tg, cholesterol/HDL-C, LDL-C/HDL-C are significantly higher in female group than in the male group. This type of phenomenon happens, because probably 80\% of the female hypertensive of this group is postmenopausal.

A comparative study of the lipid profile levels between hypertensive and normotensive groups and search for optimal cutoff points of lipid profiles (TC, Tg, LDL-C and HDL-C) as indicators of hypertension risk factors are conducted for the first time on the population of eastern India. According to the expert panel of a National Cholesterol Education Program (NCEP), the practical action plan for considering an association between lipid profile levels connected with the risk of developing CAD are calculated, with TC >200 mg/dl, Tg >150 mg/dl, LDL-C >130 mg/dl, and HDL-C <40 mg/dl as risk factors for CAD\textsuperscript{79}. In the present study the level of TC of 165 mg/dl (which has equal sensitivity and specificity) is the lowest value for ruling out hypertension, which is found even lower than the cutoff value (200 mg/dl) for TC in subject with CAD\textsuperscript{80}.

It is observed that elevated fasting plasma triglyceride level is found to have a strong correlation with the degree of end-organ involvement in hypertension\textsuperscript{81}. In the present study, Tg level of 150 mg/dl also has the highest specificity, but the lowest sensitivity, though the false negative (due to food habit) at this level is high. For LDL-C, the optimal level is found to be 100 mg/dl. Therefore, it leaves a scope for screening the proposed LDL-C >130 mg/dl for CAD, but it is intended a higher level of LDL-C for case findings. This study obtained the highest specificity at the level of LDL-C 145 mg/dl. For HDL-C, it may be seen that since the observed level of 41 mg/dl, is close to
the level of the HDL-C cutoff point in CAD so it is in agreement as per National Cholesterol Education Program.

Hyperlipidemia and hypertension have been associated in several cross sectional studies. Hypertension and serum cholesterol are strongly correlated among hypertensive, which leads to early recommendations for treating elevated cholesterol in hypertensive.

The correlation of lipids and hypertension has also been linked via a mechanism of angiogenesis and expression. Lipid abnormalities and insulin resistance have been associated with sympathetic hyperfunction, which may play a role in the development of hypertension.

Mean blood pressure level increases consistently with each body mass index category, the magnitude of this relationship being comparable to that seen with age. This association is recognized early last century and has been repeatedly demonstrated. In this study, it is included the observed relation between BMI, hypertension and age. Not only that but also, like these results, the positive association between body mass index and total cholesterol, LDL-C, plasma glucose and Tg, and the inverse association with HDL-C, have been observed to be consistent in other cross-sectional studies in both women and men.

Obesity is an independent risk factor for CAD mortality in association with diabetes, hyperlipidemia and hypertension. The role of obesity in cardiovascular risk has been documented from the Framingham Heart Study and, in the Nurses' Health Study and is recognized as an independent risk factor for myocardial infarction. Several studies, including some cited above, have suggested that plasma cholesterol concentrations are not related to BMI, but in the present study cholesterol is very significantly correlated in obese diabetics compared to normal weight diabetics, which is an observation consistent to findings reported in other studies. But it has been observed that the mean serum Tg levels are higher in obese diabetics in comparison to obese control group, which is substantiated by other studies also. High levels of cholesterol, triglyceride, LDL-C and low HDL-C may be due to the obesity, increased calorie intake and lack of muscular exercise in this group.
There is a noxious effect of hypercholesterolemia in the overweight group, especially in male subjects, but this has no relevance in the young women because, hormonal effects found in this stage, may act as a protective factor against adverse changes in lipid profile. Before menopause, changes in lipid profile are sensitive to the influence of sex hormones, especially estrogen, which has a favorable effect on lipoproteins by increasing HDL-C and reducing LDL-C levels; as a matter of fact females are at an advantage before post menopausal stage. The positive association between body mass index and total cholesterol, LDL-C, fasting blood glucose and triglycerides, and the inverse association with HDL-C, have been observed consistent as in other cross-sectional studies in both women and men.

Although the exact biochemical mechanisms responsible for the association between obesity and the above diseases are yet to be completely and convincingly elucidated, it is known that increase in triglyceride stores is associated with a linear increase in the production of cholesterol which in turn is associated with increased cholesterol secretion. Similarly, increased levels of circulating triglyceride in obesity are associated with decreased concentrations of high-density lipoprotein, which may account for the increased risks for cardiovascular disease and heart attack in obese persons.

Waist or hip circumferences have not been measured in the population under study and cannot therefore derive specific comment on the role of body fat distribution in relation to the other coronary risk factors measured including fasting insulin concentrations. However, it is interesting to note that the observations are in typical association and are very similar to those described in syndromes of insulin resistance, with hypertension, hypertriglyceridaemia, and hyperglycaemia all associated with increasing of BMI in the present study.

This study has demonstrated that Tg level is high in diabetic subject, especially in females along with diminished level of HDL-C, which is also supported by some authors and they have described that Tg levels as a risk factor for CAD independent of HDL-C level and despite glycemic control and few authors have reported that Type 2 DM increases the risk of CHD more markedly in women than in men. Adverse changes induced by type 2 DM in some cardiovascular risk factors, such as
HDL-C, total cholesterol, Tg, LDL-C particle size and blood pressure have been found to be more pronounced in women than in men\textsuperscript{111,112}. This result of the study also have been strongly upheld by Juutilainen et al. who in their study of 1059 Type 2 diabetic subjects aged 45-64 years found a considerably higher diabetes related relative risk for a major CHD event in diabetic women than in men\textsuperscript{113}. They found that the burden of obesity, elevated blood pressure, and atherogenic dyslipidemia (low HDL-C and high Tg) are present in the diabetics, greater in women than in men. In analysis of prospective studies of coronary death among women with DM, Warren et al. corroborated that the relative risk of coronary death from diabetes was greater for women than men\textsuperscript{114}.

There is a deficiency in the present study that high triglyceride levels seem to increase the risk of coronary artery disease in women, but this association is not alienated from the combined effects of obesity and diabetes due to limited study material and methodology adopted. Hypertriglyceridemia plays central role in the pathogenesis and clinical course about which are often referred to as the diseases like Type 2 DM, hypertension, and CAD\textsuperscript{115}. High Tg and low HDL-C are important risk factors of CAD because these lipids are closely associated with a series of atherogenic and thrombogenic changes all believed to play a central role in the pathogenesis of IHD. Higher level of Tg and lower level of HDL-C are closely linked to a change in LDL-C particle size, density distribution, and composition leading to a smaller and denser LDL-C particle that is more prone to oxidation\textsuperscript{116} and thus more atherogenic\textsuperscript{117}.

LDL-C status is abundantly clear in this study. LDL-C and total cholesterol levels increased significantly in the age group of 41 – 60 years of women than that of 61-80 years of women. A meta-analysis of pooled observational data supports our views and demonstrates an increase risk of coronary artery disease in women who had hyperlipidemia in the age group 55-65 years\textsuperscript{118}.

In the present study it is observed that overweight, diabetic and hypertensive groups are associated with low plasma levels of HDL-C. It has been well documented that reduced HDL-C level is associated with an increased risk of coronary heart disease\textsuperscript{119}. It may be due to a number of HDL-C particle functions that possibly will contribute to direct cardioprotective effects, including promotion of cellular cholesterol efflux and anti-
oxidative and anti-inflammatory properties\textsuperscript{120}. In the present findings low HDL-C levels are often accompanied by elevated Tg, cholesterol and LDL-C, and these combinations have been strongly associated with an increased risk of CHD.

Again when hypertensive or overweight groups are compared with normal control groups, HDL-C is significantly (p<0.05) low in these groups than control group. Nearly identical findings are also reported in the Pawtucket Heart Study\textsuperscript{121}. Similarly, a pooled analysis among three European Cohorts consisting of a total of 402 elderly men demonstrated a significant relation of physical activity with HDL-C\textsuperscript{122}. The same findings are observed in several studies that include only women\textsuperscript{123,124}. Our limited findings regarding HDL cholesterol could be the answer to the stated hypothesis. Moreover, patients with higher LDL-C with typically lower HDL-C and elevated Tg blood levels, may further increase risk of atherosclerosis\textsuperscript{125}.

When comparison is made with men, women have slightly higher HDL-C levels, and these levels tend to remain steady throughout life, even in various pathophysiological conditions, a trend as observed in this study. Several studies have demonstrated the protective effects of high HDL-C levels, even in groups with high total cholesterol levels. An HDL-C level higher than 60 mg per dl (1.55 mmol per L) is considered as "negative risk factor" (i.e., it offsets one of the other coronary risk factors).

As expected, a sex effect is observed for HDL-C levels in both subjects and controls for all the three ethnic age groups studied, with HDL-C values significantly higher in females than males. This observation accounts largely for the natural protection against CHD that females enjoy compared to their male counterparts.
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