APPENDIX

PROTOCOL

DEMOGRAPHIC DATA
Name :
Age :
Sex :
Education : Low/Intermediate/High
Region :
Caste :
Occupation :
Marital Status : Single/Married/Divorced/Widow
Type of family :
No. of sibling :
Address :

HISTORICAL DATA:
Chest pain : Yes/No
Shortness of breath : Yes/No
Sweating : Yes/No
Giddiness : Yes/No
Diabetes mellitus : Yes/No
Hypertension : Yes/No
Smoking : Yes/No
Alcohol : Yes/No
Exercise : Yes/No
If yes then Type of exercises and Duration:
Food frequency : No. of Meals
Eating Habits : Vegetarian/Non-Vegetarian
Type of food preferred:
Ischaemic Heart : Yes/No
Disease in the past
Family IHD : Yes/No

DRUG THERAPY:
Intake of any hypolipidaemic agents : Yes/No
Antihypertensive treatment : Yes/No
Antithyroid drug therapy : Yes/No
Any other : Yes/No

GENERAL PHYSICAL EXAMINATION:
Height :
Weight :
BMI :
Systolic BP :
Diastolic BP :
Pulse Rate :
ECG findings :

BIOCHEMICAL INVESTIGATIONS:
Lipid Profile
• TG’s
- Total Cholesterol
- HDL-Cholesterol
- LDL-Cholesterol
- VLDL-Cholesterol
- Apolipoprotein a
- Apolipoprotein b

ANTHROPOMETRIC MEASUREMENTS:

- Waist circumference :
- Hip circumference :
- Upper arm circumference :
- Skin folds :
- Sub scapular skin fold :
- Supra iliac skinfold :
- Medial calf :

Nutrition & Eating Habits Questionnaire

- Who prepares meals in your home?
- How many meals do you eat away from home on weekdays? BfSt: Lunch: Evening Meal
- How many meals do you eat away from home on weekends?
- Do you exercise now? No Yes If yes, what do you do and how often do you do it?
- Is there any reason you cannot or should not exercise?
- Has your weight changed in the last year? No Gained Lost____
- What do you think is a realistic weight for you?
- How long has it been since you were at that (realistic) weight?
- What kinds of diets and/or surgeries have you tried to lose weight?
- Do you currently take vitamins or minerals? If yes, list the names and amounts you take:
- Do you use any other dietary supplements on a regular basis? This would include things like fiber tablets or power, garlic pills, herbs, DHEA, etc. Please list the supplements and amounts:
- Do you use any meal replacement products (liquids, bars, etc); which ones & how often?
- What kinds of beverages and how much of them do you drink on most days?
- Coffee Tea Juice Regular soda Diet soda
- Milk (cups & what kind) Alcohol Water Other
- Circle the vegetables you eat; note # of servings eaten for e Day

252
ach group.

| Non-starchy group | \(,\) green beans, beets, broccoli, Brussels sprouts, cabbage, carrots, cauliflower, cucumber, mushrooms, onions, peppers, yellow or zucchini squash, tomatoes, turnips, wax beans |
| Leafy group | salad greens, kale, spinach, sprouts, turnip or mustard greens, |
| Starchy group | potato, corn, green peas, dried beans or peas (pinto, kidney, white, black, brown, lentils, black-eyed, split, etc), mixed vegetables with corn, peas, or pasta, lima beans, sweet potatoes, yams |
| Fresh group | apple, apricot, banana, blackberries, blueberries or other berries, cherries, grapefruit, grapes, honeydew, kiwi, mango, orange, papaya, peach, pear, pineapple, plums, strawberries, watermelon, other |
| Canned group | applesauce, apricot, fruit cocktail, grapefruit sections, mandarin orange, peach, pear, pineapple, other |
| Dried group | apple, apricot, raisins (cranberries), dates, figs, peaches, prunes, raisins, other dried fruits |
| Juice group | apple, cranberry, grape, grapefruit, mixed fruit, orange, pineapple, prune, other fruit juice |
| Other Foods and frequency of intake | Junk Food | Fatty foods |

253
ABSTRACT

Background: Studies have suggested that an elevated plasma concentration of apolipoprotein (apo) B coupled with obesity may be considered as an important risk factor for coronary heart disease (CHD) than the traditional lipid factors. Coronary artery disease (CAD) is a multifactorial disease resulting from interaction among various hereditary, cultural and environment factors. Population specific studies are rare.

Aim: The aim of this study was to evaluate the association of body mass index (BMI), blood lipids and apolipoproteins with the CAD among the Khatri caste, which is an indigenous population of Northwest India.

Materials and Methods: The study was carried on 150 CAD patients and 150 normal controls belonging to the Punjabi Khatri caste ranging in age from 35–45 years. Height and body weight was measured using standard techniques. Blood was drawn from each subject to analyze serum concentrations of lipids and apolipoproteins.

Results: The study demonstrated that CAD patients had elevated BMI in both males and females than normal controls. Apo B levels were an important predictor of CAD. ApoA/ApoB ratio among CAD patients was 0.74 compared with 1.53 in normal subjects; controls had 105.79% higher ApoA/ApoB ratio than CAD subjects. Total cholesterol, LDL-C, triglycerides, LDL-C/HDL-C ratio of the two groups also showed significant differences. Prevalence of obesity in CAD patients was 70.7% compared with 10% in normal controls.

Conclusions: Apo B levels were found to the best predictor of CAD, even though significant differences were also found between CAD and normal subjects for other lipoprotein traits. Obesity was high CAD patients than normal controls.

Keywords: CAD, Body Mass Index, lipid profile, apolipoproteins.
INTRODUCTION:
Asian populations have experienced a spurt in chronic diseases later than the western populations. South Asia, where infectious diseases are still highly prevalent, has suffered much more after this transition, as the prevalence, incidence and mortality from coronary artery disease (CAD) among them have been reported to be higher than among the western and other Asians, irrespective of whether they live in India or abroad (Reddy 2007, Gupta 2008, Murthy et al. 2012). The prevalence of CAD doubled to 3 to 4% in rural India and quadrupled to 9 to 11% in urban India over the past four decades (Reddy 2007, Gupta 2008). Heart diseases are rising in Asian Indians 5–10 years earlier than in other populations around the world with the mean age for first presentation of acute myocardial infarction in Indians is 53 years (Sharma and Ganguly 2005). This increasing burden of chronic diseases may be attributed to demographic transition coupled with unplanned urbanization, food habits, lifestyle changes and genetic factors.

Epidemiologic studies have shown that an increased risk of coronary artery disease exists with elevated level of total cholesterol, very low density lipoproteins (VLDL), low density lipoproteins (LDL) and lower levels of high density lipoproteins (HDL) (Arsenault et al. 2009). However, it is yet difficult to define the independent contribution of several interrelated abnormalities in lipoproteins to atherogenesis. In this context, Lamarche et al. (1997) argue that despite the association between LDL levels and CAD is well accepted but yet a relatively high proportion of cases with CHD have LDL in the normal range. There are several possible explanations for the absence of a positive association between total cholesterol and vascular mortality. For example, the total cholesterol level in the elderly people may not represent their lifetime exposure, because of many lifestyle factors and diseases that may modulate it with advancing age (Lindberg et al. 1995).

Several additional components of the lipoprotein system have been identified and are under evaluation. These components include apolipoprotein subclasses, HDL subclasses, small dense LDL particles, remnants of chylomicrons and VLDL, IDL, and Lp (a). Apolipoproteins bind lipids to form lipoproteins. Lipoproteins particles contribute to overall metabolic homeostasis by transporting hydrophobic lipids in the blood plasma to and from different tissues in the body:
very-low-density lipoprotein (VLDL) is the principal vehicle for the transport of endogenous triglyceride (TG), and its metabolic product, low-density lipoprotein (LDL) transports cholesterol (Mason 1998). The main structural protein of VLDL is called apolipoprotein (apo) B-100 (Apo B). Apo B formation and degradation are two major points of regulation of VLDL secretion (Mason 1998). Numerous epidemiological studies have shown that plasma LDL and high density lipoproteins (HDL) cholesterol are important risk factors for CAD. The indices such as total cholesterol/HDL and LDL/HDL cholesterol ratios; and apolipoprotein B, the protein moiety of LDL may also be relevant predictors of CAD (Reviewed in McGrowder et al. 2011, Bambauer et al. 2012). Apolipoprotein A-I (Apo-A1) has molecular mass of 28 kDa and it is the major structural HDL apolipoprotein and accounts for 70% of total HDL protein, whereas the second major HDL apolipoprotein, apoA-II, represents the 20% that constitutes approximately 70% of the protein in HDL (Superko 2009). ApoA contains domain that are very similar to plasminogen. The main function of plasminogen is to dissolve fibrin blood clots.

Increased body weight has been associated with an increased risk of morbidity and mortality from coronary artery disease in several populations (Ford et al. 2007, Beaglehole and Bonita 2008, WHO 2009). Body mass index (BMI), expressed as body weight in kilograms divided by height in square meters (kg/m²), is highly correlated with body weight and poorly correlated with height (Willett 1990) and is frequently used as a measure of obesity in large epidemiological studies. Lamon-Fava et al. (1996) studied the distribution of BMI in men (mean age, 49 ±10 years) and women (mean age, 49 ± 10 years) and the association of BMI with known CHD risk factors. They found in men, BMI increased with age until age 50 years, when it reached a plateau. In women, there was a trend toward an increase in BMI with age up to the seventh decade of life. Seventy-two percent of men and 42% of women had a BMI ≥25.00, the cutoff point for the definition of overweight. They further found in age-adjusted analyses that BMI was significantly and linearly associated with systolic blood pressure, fasting glucose levels, plasma total cholesterol, VLDL cholesterol, and LDL cholesterol levels and was inversely and linearly associated with HDL cholesterol levels (P < .001) in nonsmoking men and women. The association between BMI and apolipoprotein B and A-I was similar to that of LDL and HDL cholesterol, respectively. LDL size was also linearly associated with BMI: subjects with higher
BMI had smaller LDL particles. However they did not find association of Lipoprotein (a) levels with BMI in this population.

The present study was undertaken to compare blood lipid profile, apolipoproteins and BMI of coronary artery disease patients with normal controls drawn from an endogamous caste group of Khatris of Northwest India and to analyse whether apoA/ apo B ratio be used as a more accurate lipid risk factor.

**MATERIALS AND METHODS**

This cross sectional study was carried out on 150 diagnosed cases of CAD (75 males and 75 females) and 150 normal controls CAD (75 males and 75 females) belonging to Khatri caste of Punjab. The study sample was drawn from the department of Biochemistry and department of Cardiology, Christian Medical College and Hospital, Ludhiana and G. B. Pant Hospital, New Delhi. The controls were drawn from those who had visited for routine and normal checkup and had no history of CAD. Each subject was administered a health and lifestyle questionnaire schedule, which included questions on weight, height, socio-demographic details, medical history, and lifestyle habits. All participants gave their written consent voluntarily to take part in the study. The inclusive criterion was that the subjects should be belonging to Khatri caste and between 35 -45 years of age. The subjects younger than 35 and older than 45 years and not belonging to Khatri caste, and those who refused to participate in the study were excluded. Apart from these the exclusion was made if the patient suffered from any of the following conditions: congenital heart disease, valvular heart disease, dilated cardiomyopathy, recent myocardial infarction of less than 6 weeks duration prior to admission. Levels of educational attainments and yearly family income were comparable among CAD patients and controls and there were no statistically significant differences.

Their height was measured in centimeters and weight in kilograms. BMI was calculated by the following formula: \( \text{BMI} = \frac{\text{Body Weight in Kilograms}}{\text{Height}^2 \text{ (square meters)}} \). Five milliliter of blood was drawn from each subject, after a 12- to 14-hour fast, in 0.1% EDTA tubes. Lipid Profile estimation was done on automated analyzer — Hitachi – 902. Total cholesterol was estimated by colorimetric analysis. The other components of lipid profile were estimated as follows:
TRIGLYCERIDES: Triglycerides levels were estimated using GPO-PAP enzymatic colorimetric analysis in human serum on Roche autoanalyzer (Hitachi – 902).

HDL-CHOLESTEROL: HDL - C was measured directly by homogeneous enzymatic colorimetric test in human serum on Roche autoanalyzer (Hitachi-902).

LDL-CHOLESTEROL: LDL-Cholesterol was estimated by using Homogeneous Enzymatic Colorimetric assay for its direct quantitative determination in human serum on automated analyzer-Roche (Hitachi 902).

APOLIPOPROTEIN B: It was quantitatively estimated by Immunoturbidimetric immunoassay with Randox kit in serum on semiautomated analyzer — Clima Plus. (Cat. No. LP 2117) (Labeur et al, 1990)

APOLIPOPROTEIN A-1: It was quantitatively estimated by Immunotrubiditnetric Immunoassay with Randox kit in serum on semiautomated analyzer Clima Plus. (Cat. No. LP 2117) (Labeur et al, 1990).

VLDL-Cholesterol was calculated by TG/5.

Statistical Analysis

Data was recorded on a predesigned proforma and managed in a Microsoft Excel spreadsheet. All the entries were double checked for any possible keyboard error. The data was subjected to statistical analysis using Stata software. The descriptive data is presented as mean and standard deviation for the continuous variables and as absolute quantities and percentages for the discrete parameters. The correlation coefficient was worked out to find out the degree of association between anthropometric parameters on the one-side and lipid fractions on the other.

RESULTS

Table 1 summarizes the results of descriptive analysis t-tests comparing means of body mass index and various characteristics of the of plasma lipid and apolipoprotein levels (mg/dl) of men and women CAD subjects and controls. CAD subjects (both males and females) had significantly higher body mass index (BMI), blood levels of total cholesterol, triglycerides, low density lipoproteins (LDL), very dense lipoproteins (VLDL) and Apo B levels than controls. CAD subjects had 27% triglycerides, 24% total cholesterol, 25% VLDL, 47.5% LDL and 68.4% Apo B than normal controls. On the contrary, normal subjects had 27% higher blood levels of high density cholesterol (HDL) and 23% of Apo A than CAD patients. ApoA/ApoB ratio among
CAD patients was 0.74 compared with 1.53 in normal subjects; normal controls had 105.79% higher ApoA/ApoB ratio than CAD subjects.

The frequency distribution of the subjects according to different categories of BMI is shown in Table 2. The proportion of the normal controls having normal BMI value (ranging 18.0-22.99 kg/m²) was much greater (20%) than that of CAD patients (1.3%). As expected, the prevalence of obesity in CAD patients was much higher (70.7%) compared with 10% in normal controls. The differences were statistically significant. Prevalence of obesity grade II was low (0.7%) in CAD patients, and zero in normal controls. Among Asian Indian subjects, escalating population-wide generalized obesity correlates strongly with increasing cardiovascular risk factors (Gupta and Gupta 2008).

**DISCUSSION**

The present study finds significant differences between CAD subjects and normal controls for various lipoprotein traits and BMI. ApoB level was significantly higher in CAD subjects than normal and so also ApoA/ApoB ratio. It has now been widely argued that Apo B plays important roles for the hepatic secretion of VLDL and metabolism and clearance of triglycerides from the circulation (Young 1990). Thus, for a given cholesterol concentration, a high number of apo B-containing lipoproteins will result in the presence of an elevated number of small, dense LDL particles, which have been associated with an enhanced risk of IHD (Austin et al. 1990). Patients with CAD may be characterized by increased transport rates of LDL apo B (Kesaniemi 1985). Our results are consistent with other such case-control reports that supported the role of apo B as an important risk factor for CAD (Genest et al. 1992, Olofson et al. 2007, Philip et al. 2011).

According to World Health Organization (1998) and National Institutes of Health (1998) have set normal range of BMI at 19–24.9 kg/m², overweight at 25–29.9 kg/m², and obese at ≥ 30 kg/m². Japan Society for the Study of Obesity (JASSO) decided to define BMI ≥ 25 as obesity (Kanazawa et al. 2002). The findings of the present study are on the expected lines. Prevalence of obesity was higher in CAD patients than controls among middle aged adult Khadris of Punjab. When compared with other Indian studies, prevalence of obesity was lower in the present sample of normal controls (10%) than that reported among urban females (25.3%) by Sidhu and Tatla.
(2002), but prevalence of overweight and obesity put together was higher in the present sample (80%) than urban females (45.3%). Gopalan (1998) found the prevalence of overweight and obesity as 50% and 14%, respectively. Among Asian Indian subjects, generalized obesity correlates strongly with increasing cardiovascular risk factors (Gupta and Gupta 2008).

**Conclusion**

Apolipoproteins are the significant predictors of the coronary artery disease. The predictive ability of ApoA/ApoB ratio was also better than ApoB, apoA values. Thus ApoA/ApoB ratio is a better marker of cardiovascular risk and their inclusion in further clinical guidelines should not be discarded.

**REFERENCES**


331


Superko HR. 2009. Advanced lipoprotein testing and subfractionation are clinically useful. Circulation 19, 2383-2395.


Table 1: Mean standard deviation of plasma lipid and apolipoprotein levels (mg/dl) of men and women CAD patients and controls

<table>
<thead>
<tr>
<th></th>
<th>FEMALES</th>
<th>t-test</th>
<th>MALES</th>
<th>t-test</th>
<th>OVERALL</th>
<th>t-test</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N</td>
<td>Mean</td>
<td>σ</td>
<td>p</td>
<td>N</td>
<td>Mean</td>
</tr>
<tr>
<td>Triglycerides (TG) (mg/dl)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CAD</td>
<td>75</td>
<td>144.940</td>
<td>57.553</td>
<td></td>
<td>75</td>
<td>178.027</td>
</tr>
<tr>
<td>CONTROL</td>
<td>75</td>
<td>113.040</td>
<td>25.233</td>
<td>0.000</td>
<td>75</td>
<td>141.013</td>
</tr>
<tr>
<td>Total</td>
<td>150</td>
<td>128.993</td>
<td>47.091</td>
<td></td>
<td>150</td>
<td>159.520</td>
</tr>
<tr>
<td>Total cholesterol (TC) (mg/dl)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CAD</td>
<td>75</td>
<td>194.560</td>
<td>43.507</td>
<td></td>
<td>75</td>
<td>199.813</td>
</tr>
<tr>
<td>CONTROL</td>
<td>75</td>
<td>157.760</td>
<td>22.664</td>
<td>0.000</td>
<td>75</td>
<td>159.173</td>
</tr>
<tr>
<td>Total</td>
<td>150</td>
<td>176.160</td>
<td>39.192</td>
<td></td>
<td>150</td>
<td>179.493</td>
</tr>
<tr>
<td>High-density lipoproteins (HDL) (mg/dl)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CAD</td>
<td>75</td>
<td>40.200</td>
<td>15.848</td>
<td>1.000</td>
<td>75</td>
<td>34.160</td>
</tr>
<tr>
<td>CONTROL</td>
<td>75</td>
<td>48.770</td>
<td>7.404</td>
<td></td>
<td>75</td>
<td>45.520</td>
</tr>
<tr>
<td>Total</td>
<td>150</td>
<td>44.480</td>
<td>13.056</td>
<td></td>
<td>150</td>
<td>39.840</td>
</tr>
<tr>
<td>Low-density lipoproteins (LDL) (mg/dl)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CAD</td>
<td>75</td>
<td>125.596</td>
<td>42.134</td>
<td></td>
<td>75</td>
<td>131.240</td>
</tr>
<tr>
<td>CONTROL</td>
<td>75</td>
<td>88.236</td>
<td>22.165</td>
<td>0.000</td>
<td>75</td>
<td>85.849</td>
</tr>
<tr>
<td>Total</td>
<td>150</td>
<td>106.916</td>
<td>38.431</td>
<td></td>
<td>150</td>
<td>108.545</td>
</tr>
<tr>
<td>Very-Low-density lipoproteins (VLDL) (mg/dl)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CAD</td>
<td>75</td>
<td>28.989</td>
<td>11.511</td>
<td>0.000</td>
<td>75</td>
<td>39.963</td>
</tr>
<tr>
<td>CONTROL</td>
<td>75</td>
<td>22.608</td>
<td>5.046</td>
<td></td>
<td>75</td>
<td>32.448</td>
</tr>
</tbody>
</table>

333
<p>| | | | | | | | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>APO A (mg/dl)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CONTROL</td>
<td>75</td>
<td>125.691</td>
<td>17.151</td>
<td>1.000</td>
<td>75</td>
<td>120.317</td>
<td>18.818</td>
<td>1.000</td>
</tr>
<tr>
<td>Total</td>
<td>150</td>
<td>113.769</td>
<td>19.872</td>
<td>150</td>
<td>109.157</td>
<td>18.564</td>
<td>300</td>
<td>111.463</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>APO B (mg/dl)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CAD</td>
<td>75</td>
<td>131.622</td>
<td>26.635</td>
<td>75</td>
<td>149.299</td>
<td>17.209</td>
<td>150</td>
<td>140.461</td>
</tr>
<tr>
<td>CONTROL</td>
<td>75</td>
<td>84.977</td>
<td>16.538</td>
<td>0.000</td>
<td>75</td>
<td>81.809</td>
<td>15.934</td>
<td>0.000</td>
</tr>
<tr>
<td>Total</td>
<td>150</td>
<td>108.300</td>
<td>32.183</td>
<td>150</td>
<td>115.554</td>
<td>37.677</td>
<td>300</td>
<td>111.927</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>APO A/B ratio</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CAD</td>
<td>75</td>
<td>0.822</td>
<td>0.291</td>
<td>75</td>
<td>0.665</td>
<td>0.102</td>
<td>150</td>
<td>0.743</td>
</tr>
<tr>
<td>CONTROL</td>
<td>75</td>
<td>1.547</td>
<td>0.408</td>
<td>0.000</td>
<td>75</td>
<td>1.512</td>
<td>0.296</td>
<td>0.000</td>
</tr>
<tr>
<td>Total</td>
<td>150</td>
<td>1.18</td>
<td>0.507</td>
<td>150</td>
<td>1.089</td>
<td>0.391</td>
<td>300</td>
<td>0.028</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CAD</td>
<td>75</td>
<td>31.145</td>
<td>2.126</td>
<td>75</td>
<td>30.262</td>
<td>1.516</td>
<td>150</td>
<td>30.703</td>
</tr>
<tr>
<td>CONTROL</td>
<td>75</td>
<td>26.431</td>
<td>2.667</td>
<td>75</td>
<td>27.707</td>
<td>1.624</td>
<td>150</td>
<td>27.069</td>
</tr>
<tr>
<td>Total</td>
<td>150</td>
<td>28.788</td>
<td>3.372</td>
<td>0.000</td>
<td>150</td>
<td>28.984</td>
<td>2.024</td>
<td>0</td>
</tr>
</tbody>
</table>
Table 2 Comparison of BMI categories between CAD patients and controls

<table>
<thead>
<tr>
<th>BMI category</th>
<th>CAD</th>
<th>Controls</th>
<th>X^2 value</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N</td>
<td>(%)</td>
<td>N</td>
<td>(%)</td>
</tr>
<tr>
<td>18.5-24.9 BMI Normal</td>
<td>2</td>
<td>1.3</td>
<td>30</td>
<td>20</td>
</tr>
<tr>
<td>25.0-30.0 Overweight</td>
<td>42</td>
<td>28</td>
<td>105</td>
<td>70</td>
</tr>
<tr>
<td>30.0-35.0 Obesity (Grade I)</td>
<td>105</td>
<td>70</td>
<td>15</td>
<td>10</td>
</tr>
<tr>
<td>35.0-40.0 Obesity II (Grade II)</td>
<td>1</td>
<td>0.7</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Total</td>
<td>150</td>
<td>100</td>
<td>150</td>
<td>100</td>
</tr>
</tbody>
</table>