Impact of Obesity on Castelli’s Risk Index I and II, in Young Adult Females

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Abstract

Background: Obesity is one of today’s most blatantly visible, yet most neglected, public health problems. In 2016, 39% of adults worldwide were overweight. Fueled by economic growth, urbanization, an increasingly sedentary lifestyle, and a nutritional transition to processed foods and high calorie diets over the last 30 years, many countries have witnessed the prevalence of obesity in its citizens double, and even quadruple. Obesity especially visceral obesity causes insulin resistance and is associated with dyslipidemia, impaired glucose metabolism, and hypertension all of which exacerbate atherosclerosis, and are risk factors for developing cardiovascular diseases (CVD). The primary dyslipidemia related to obesity is characterized by increased total cholesterol (TC), decreased high density lipoprotein (HDL) levels and abnormal low density lipoprotein (LDL) composition. Lipoprotein ratios are becoming increasingly popular as a way to predict atherosclerosis and CVD.

Aims and Objectives: The present study was undertaken to assess the impact of overweight/obesity on lipid profile parameters and lipoprotein ratios- Castelli’s Risk Index I and II, in young adult females.

Materials and Method: The present study was conducted in KIMS, Hubli, the study and its conduct was cleared by the Ethical committee. Sixty apparently healthy young females were selected for the study. Health status and other personal data were obtained via comprehensive questionnaire. The subjects were divided into two groups based on BMI; Healthy (BMI 18.5-24.99) and Overweight (BMI > 25). Lipid profile was evaluated and lipoprotein ratios calculated. Comparison between the two groups was done using students’ t-test.

Results: Values for Castelli’s Risk Index I & II were found to be significantly higher in the overweight group compared to the control group.

Conclusion: Obesity leads to an unfavorable lipid pattern and raises values of both Castelli’s Risk Index I & II.

Keywords: Obesity, Castelli’s Risk Index I & II, Lipid profile, Lipoprotein ratios.

Introduction

Noncommunicable diseases (NCDs), such as heart disease, stroke, cancer, chronic respiratory diseases and diabetes, are the leading cause of mortality in the world. This invisible epidemic is an under-appreciated cause of poverty and hinders the economic development of many countries. Common, modifiable risk factors underlie the major NCDs. They include tobacco, harmful use of alcohol, unhealthy diet, insufficient physical activity, overweight/obesity, raised blood pressure, raised blood sugar and raised cholesterol.

Obesity, which broadly refers to excess body fat, has become an important public health problem, and has
reached to epidemic proportions. Large, high-quality longitudinal or prospective studies have confirmed that obesity is a significant risk factor for and contributor to increased morbidity and mortality, primarily from cardiovascular disease (CVD) and diabetes, but also from cancer and other acute and chronic diseases, including osteoarthritis, liver and kidney disease, sleep apnea, and depression. For the majority of these co morbid conditions, weight loss can result in a significant reduction in risk.

The primary dyslipidemia related to obesity is characterized by increased triglycerides, decreased HDL levels, and abnormal LDL composition. The pathophysiology of the typical dyslipidemia observed in obesity is multifactorial and includes hepatic overproduction of VLDL, decreased circulating TG lipolysis and impaired peripheral free fatty acid (FFA) trapping, increased FFA fluxes from adipocytes to the liver and other tissues and the formation of small dense LDL.

Dyslipidemia is recognized as a prominent risk factor for cardiovascular (CV) disease. Lipid abnormalities, including high levels of low-density lipoprotein cholesterol (LDL-C), elevated triglycerides and low levels of high-density lipoprotein cholesterol (HDL-C), are associated with an increased risk of CVD, thereby serving as contributors to this process.

There is now overwhelming experimental and clinical evidence that atherosclerosis is a chronic inflammatory disease. The atherogenic process starts with the accumulation of several plasma lipoproteins in the subendothelial space at sites of flow perturbation and endothelial dysfunction. In the intima, LDL undergoes oxidative modifications by reactive oxygen species, which promote the uptake of ox-LDL (oxidized LDL) into macrophages.

Low-density lipoprotein (LDL) cholesterol concentration has been the primary index of cardiovascular disease risk and the main target for therapy. However, several lipoprotein ratios have been defined in an attempt to optimize the predictive capacity of the lipid profile. Castelli’s Risk Index I (TC/HDL) and II (LDL/HDL) are two ratios which have shown promise in predicting atherogenicity and CVD. Castelli Risk Index I (CRI-I) has been particularly shown to reflect coronary plaques formation and the thickness of intima-media in the carotid arteries of young adults.

The lipoprotein ratios take into consideration both the proatherogenic and antiatherogenic factors, hence they could prove to be a better index for predicting atherogenicity and CVD.

The present study was undertaken to assess the impact of overweight/obesity on lipid profile parameters and the lipoprotein ratios; Castelli’s Risk Index I and II (CRI- I and CRI- II), in young adult females.

Materials and Method

This study was conducted in the department of Physiology, KIMS Hubli, with the assistance of Biochemistry lab, KIMS Hubli. The study and its conduct were cleared by the ethical committee KIMS Hubli.

Inclusion criteria

1) Healthy young females with the age ranging between 18-35 years

Exclusion criteria

1) Women on lipid lowering drugs, oral contraceptives, or any medications which may influence lipid profile.
2) Pregnant and lactating women.

Methods of collection of data

Health status of the volunteers was assessed by comprehensive questionnaire and clinical examination. The collection of the samples, which is an invasive procedure to be performed in the study, was explained to the subjects. The volunteers were asked to do overnight fasting, and the blood sample was collected next day morning, an informed consent for the procedure was taken. They were advised to continue their normal daily diet and working routine.

Anthropometric data: Measurements were taken while subjects were relaxed, standing erect and had their arms at their sides and feet together.

- **Body height** was measured by wall mounted Stadiometer.
- **Body weight** was recorded by clinical weighing machine, with subjects dressed in light clothes and no shoes.
- **BMI** was calculated as per formula: Weight
Castelli’s Risk Index I (CRI-I) was calculated by the formula: CRI-I = TC/HDL.

Castelli’s Risk Index II (CRI-II) was calculated by the formula: CRI-II = LDL/HDL.

The subjects were divided into two groups based on BMI:

- Those with BMI in the range of 18.5-24.99, were placed in the Control group/Controls.
- Those with BMI > 25 were placed in the Study group/Subjects.

Vital parameters like pulse rate, BP were recorded. After selecting the subjects, appointment was scheduled in prior and they were requested to do an overnight fast prior to the day of the test to get fasting blood sample for lipid profile analysis. Between 7am to 10am, 2ml of venous blood was collected, in a plain bulb by venepuncture under aseptic precaution. Serum lipid profile was analyzed in Biochemistry clinical Lab, in KIMS, Hubli, with clinical chemistry Analyzer (Type Model: XL-300 ERBA).

Statistical Analysis

Comparison between the two groups was done by students’ t-test. All the analysis was done by using SPSS-20 software.

Results

The present study had a total of sixty participants, who were divided into two groups based on their BMI. The mean values of serum total cholesterol (STC), serum triglycerides (STG), HDL, LDL, CRI-I and CRI-II is shown in Table 1.

| Table 1: Mean values of lipid profile parameters and Castelli’s Risk Index I and II in subjects and controls. |
|-------------------------------------------------|-------------------------------------------------|-----------------|-----------------|-----------------|
| Parameter                                       | Subjects (n=30)                                 | Controls (n=30) | ‘t’ value       | P value         |
| Serum total Cholesterol(mg/dl)                  | 165.97 ± 26.4                                  | 141.67 ± 29.16 | 3.383           | 0.001           |
| Serum Triglycerides(mg/dl)                      | 128.27 ± 48.5                                  | 126.43 ± 48.3  | 0.147           | 0.884           |
| High density Lipoprotein(mg/dl)                 | 31.47 ± 7.42                                   | 33.4 ± 7.85    | -0.981          | 0.331           |
| Low density Lipoprotein(mg/dl)                  | 108.7 ± 25.86                                  | 82.95 ± 21.6   | 4.198           | <0.0001         |
| CRI-I (TC/HDL)                                  | 5.43 ± 1.02                                    | 4.28 ± 0.5     | 5.5             | <0.0001         |
| CRI-II (LDL/HDL)                                | 3.59 ± 0.95                                    | 2.54 ± 0.54    | 5.5             | <0.0001         |
In the present study we noted that the levels of TC, STG, and LDL were higher in the study group, however the rise in the levels of TC (P value < 0.001) and LDL (P value < 0.0001) was statistically significant. Though the mean value of HDL was slightly lower in the subject group, it was not statistically significant (P value= 0.331).

The mean value for CRI-I in subjects was 5.43 ± 1.02 which was significantly higher compared to the control group value of 4.28 ± 0.5. The difference was highly significant (P value < 0.0001).

The mean value for CRI-II in subjects was 3.59 ± 0.95 which was significantly higher compared to the control group value of 2.54 ± 0.54. The difference was highly significant (P value < 0.0001).

**Discussion**

In the present study, the pattern of dyslipidemia associated with the overweight/obese group was an elevation in the level of STC, STG and LDL, and a decrease in the level of HDL. The findings concur with similar studies in the past (10, 11, 12 and 13).

The striking observation in the present study was the significantly higher values of CRI-I and II in overweight/obese group, which was similar to a study done by Myat Su Bo et al. (14).

In the evaluation of dyslipidemia, triglycerides (TGs), LDL, HDL, and total cholesterol (TC) are the lipid profiles that are commonly considered, with emphasis majorly on LDL as “bad lipoprotein” (15). However using either LDL or HDL alone to predict the risk of atherosclerosis and CVD alone is inadequate, especially in individuals with intermediate risk (16).

Studies have, however, demonstrated that in times when the conventional lipid parameters (TG, HDL-C, LDL-C, and TC) remain apparently normal, lipid ratios such as the Castelli’s risk index I and II are the diagnostic alternatives that have been shown in predicting the risk of developing cardiovascular events (17).

Moreover isolated elevation in triglyceride increases the CHD risk but these effects can be balanced by cardio protective lipoprotein of HDL cholesterol (18). There is evidence suggesting that lipid ratios, such as TC/HDL, and TG/HDL, which take account of the proportion between the pro-atherogenic and anti-atherogenic fractions, are more effective than single measures of lipids in detecting atherosclerosis, CVD, and Insulin resistance (19).

LDL is considered to be proatherogenic. Oxidation of LDL within the arterial wall may be an important early step in atherogenesis. The uptake of oxidized LDL by macrophages is a likely explanation for the formation of macrophage foam cells in early atherosclerotic lesions. In addition, oxidized LDL has many other potentially proatherogenic properties (20).

High density lipoproteins (HDL) play an important role in reverse cholesterol transport from peripheral tissues to the liver, which is one potential mechanism by which HDL may be anti-atherogenic. In addition, HDL particles have anti-oxidant, anti-inflammatory, anti-thrombotic, and anti-apoptotic properties, which may also contribute to their ability to inhibit atherosclerosis (21).

**Conclusion**

Overweight/obesity leads to an unfavorable lipid pattern characterized by high TC, LDL levels, and low HDL levels. Though High LDL levels are traditionally used to predict the risk of CVD, it’s the delicate
balance between pro-atherogenic and anti-atherogenic fractions in blood that play a key role in determining atherogenicity, hence we conclude that using lipoprotein ratios like CRI-I and II may be a better way of predicting CVD risk.

In the present study overweight/obesity was associated with high CRI-I and II values, suggesting that such individuals may have a higher risk of developing CVD in the future, unless drastic steps were taken to reduce the weight. These lipoprotein ratios can be used for screening and primary prevention of CVD in overweight/obese individuals.

**Limitations:** Since it was a cross sectional study, no casual relationship can be established. The study is limited as such because it didn’t consider the impact of type of diet and level of level of physical activity on lipid profile. Future studies can overcome these limitations by incorporating physical activity, dietary intake pattern, and the influences of social and environmental factors.

**Conflict of Interest:** The authors declare that there are no conflicts of interest regarding the publication of this paper.

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