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Research Article

A Study on Prevalence of Dyslipidemia in Obese Patients in a teaching hospital in Kerala

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Abstract: Obesity is an independent risk factor for dyslipidemia and Type2 Diabetes, both of which are inturn risk factors for coronary artery disease and stroke,and is rising in prevalence throughout the developed and developing countries This study aims to find the prevalence of Dyslipidemia in Obese Patients compared to non-obese patients who attended Genaral Medicine department of Travancore Medical College, Kollam, Kerala during the period June 2011 to May 2012 The total participant population comprised of 140 patients who were 30 to 65 years old at the time of examination All participants underwent routine physical examinations that included measurement of Body mass Index and waist circumference. About 5 ml of blood sample was collected and fasting Lipid Profile estimated Among the study group 82 patients were obese (40 males and 42 females).The mean Cholestrol was higher in obese (223.02mg/dl) compared to non obese (189.17mg/dl), The study showed that total Cholesterol, LDL Cholestrol and TG were significantly higher in obese compared to non obese ($\chi^2(1,N=140)=7.824$, P=0.005). It was seen that the mean TG was higher in obese (165.6) compared to non obese (115.5). By analyzing the results of the study conducted it was concluded that there was an increased risk of Dyslipidemia among obese compared to non-obese people. Although not all overweight or obese individuals are metabolically unhealthy, the majority are at risk of developing atherosclerotic diseases due to dyslipidemia especially when combined with other risk factors like hypertension, diabetes, smoking, physical inactivity, and consumption of an atherogenic diet. Hence a community based education in this regards is of utmost important.
Keywords: Obesity, Dyslipidemia, Risk factors.

INTRODUCTION

People are predisposed to various diseases based on their way of living and occupational habits. They are preventable, and can be lowered with changes in diet, lifestyle, and environment. Lifestyle diseases characterize those diseases whose occurrence is primarily based on daily habits of people. The onset of these lifestyle diseases is insidious, they take years to develop, and once encountered do not lend themselves easily to cure. The main factors contributing to lifestyle diseases include bad food habits including consumption of diets rich in highly saturated fats, sugars and salt, typified by fast foods; lack of regular physical activity etc and are the priority areas for further research [1, 2]. This lifestyle results in higher levels of risk factors such as hypertension, diabetes, dyslipidemia and obesity that act independently and synergistically [3]. According to the report, jointly prepared by the World Health Organization and the World Economic Forum [4] , 60% of all deaths worldwide in 2005 (35 million) resulted from non communicable diseases and accounted for 44% of premature deaths. Almost half of those who die from chronic diseases will be in their productive years. The report also points to the fact that countries like Brazil, China, Russia and India currently lose more than 20 million productive life-years annually to chronic diseases and the number is expected to grow by 65% by 2030.The report, also says India will incur an accumulated loss of $236.6 billion by 2015 on account of unhealthy lifestyles and faulty diet. According to a survey conducted by the Associated Chamber of Commerce and Industry (ASSOCHAM) [5], 68% of working women in the age bracket of 21-52 years were found to be afflicted with lifestyle ailments such as obesity, depression, chronic backache, diabetes, hypertension and dyslipidemia In many countries, peoples' diet changed substantially in the second half of the twentieth century with increase in consumption of meat, dairy products, vegetable oils, fruit juice, and alcoholic beverages, and decrease in consumption of starchy staple foods such as bread, potatoes, rice, and maize flour. Other aspects of lifestyle also changed,

notably, large reductions in physical activity and prevalence of obesity. Physical inactivity as indicated by a sedentary lifestyle is associated with the occurrence of obesity and metabolic syndrome. Regular physical activity along with low saturated fat in diet would help curb the growing menace of obesity and its co morbidities [6].

Obesity is an independent risk factor for dyslipidemia, Type2 Diabetes and Coronary artery disease and is rising in prevalence throughout the developed and developing countries. It has been proposed that hyperinsulinaemia stimulates 11β-hydroxysteroid dehydrogenase in omental adipose tissue, generating cortisol and promoting a cushingoid fat distribution. Adipose tissue is now recognized to be a source of a number of inflammatory cytokines(interleukin IL-6), tumor necrosis factor-α (TNF-α), growth factors (heparin binding epidermal growth factor HB-EGF) and hormone-like substances (leptin, adiponectin, resistin)Weight reduction is associated with an improvement in a number of coronary risk factors including BP and fasting blood glucose and lipid levels. Rapid weight gain in childhood (between 2 and 11years) appears to predict various risk factors for coronary disease in adulthood. Both diabetes mellitus and dyslipidemia are important risk factors for CAD. Body Mass Index is promulgated by the World Health Organization(WHO) as the most useful epidemiological measure of obesity. A BMI between 25 and 30kg/m^2 is defined as overweight and above 30kg/m^2 is defined as obesity. Persons with normal weight has a BMI of 18.5-24.9 It is nevertheless a crude index that does not take into account the distribution of body fat, resulting in variability in different individuals and populations. An increase in visceral fat reflects central obesity and increases health risks. The Waist Circumference (WC) is used to assess the amount of visceral obesity. Waist-hip circumference ratio (WHR), Waist-height ratio (WHR) and Waist Circumference (WC) are commonly used to predict the risk of obesity related morbidity and mortality as they account for regional abdominal obesity [5]. The term metabolic syndrome is applied to a constellation of obesity, poor glucose tolerance and an atherogenic lipid profile [4, 6]. Obesity is considered to be the link between insulin resistance and metabolic abnormalities inclusive of diabetes, hypertension and dyslipidaemia, all of which are risk factors for coronary artery disease [7].The present study was, therefore done to assess the association between obesity and dyslipidemia and to find out its prevalence in obese people and to compare it with non obese people.

The aim of the study was to study the prevalence of Dyslipidemia in Obese Patients and to compare this with non-obese people.

MATERIALS AND METHODS

The study was conducted on patients who attended Genaral Medicine Department of Travancore Medical College Kollam. Kerala during the period June 2011 to May 2012.Only those patients who satisfied the inclusion criteria and showed willingness to participate in study were included. The total participants population comprised of 140 patients who were 30 to 65 years old at the time of examination. Self answered questionnaires were used for collecting participants information including age, occupation, medical history, the use of antidiabetic, antihypertensive or lipid lowering medications, smoking status and alcohol consumption habits.

All participants underwent routine physical examinations that included measurement of waist circumference and resting blood pressure. Waist circumference was measured midway between the inferior margin of last rib and the iliac crest at the end of expiration with a heavy duty inelastic plastic fiber measuring tape to the nearest 0.5cm while the subject stood balanced on both feet.BMI was calculated in all patients. Central obesity was measured by waist circumference. The cut points were greater than or equal to 40inches for men and greater than or equal to 35 inches for women. About 5 ml of blood sample was collected from anterior cubital vein under aseptic conditions after an overnight fast from all participants. It was collected in a plain dry tube and was allowed to clot. After retraction of the clot, serum was separated by centrifugation at 3000 rpm for 5minute and fasting Lipid Profile was estimated. Total cholesterol by cholesterol oxidase method, HDL cholesterol by direct non-immunological homogenous enzymatic colorimetric method, LDL cholesterol by direct enzymatic colorimetric assay, Triglycerides by Lip/GK colorimetric assay. All these estimations were carried on Roche Cobas Integra 400 plus fully automated clinical chemistry analyzer. Results were recorded as per proforma, interpreted and analysed.

RESULTS

140 patients in the age group of 30 to 65 years who attended medical OP were included in the study after informed consent. Those with thyroid or renal diseases, or those who where on steroid therapy were excluded since these are causes of secondary dyslipidemia. Smokers, alcoholics and postmenopausal women were also excluded.

The results obtained were summarized and Statistical analysis of the results were done.

Table 1: Total Cholesterol In The Study Groups

Total Cholesterol (TC)	Obese	Non-obese	Total
<200mg/dl	26	38	64
>200mg/dl	56	20	76
Total	82	58	140

Table 2: LDL-Cholesterol in the Study Groups

LDL-Cholesterol	Obese	Non-obese	Total
<130mg/dl	20	32	52
>130mg/dl	62	26	88
Total	82	58	140

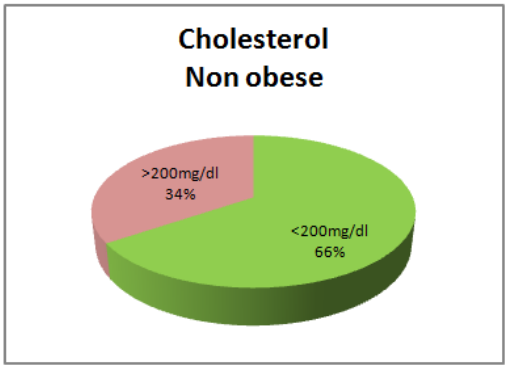
Table 3: HDL-Cholesterol in the Study Groups

HDL-Cholesterol	Obese	Non-obese	Total
Low	42	30	70
Normal or high	40	28	70
Total	82	58	140

*HDL values were considered low if the values are <40mg/dl in men and <50mg/dl in women

Table 4: Triglyceride (Tg) in the Study Groups

Triglyceride (TG)	Obese	Non-obese	Total
<150mg/dl	20	44	64
>150mg/dl	62	14	76
Total	82	58	140



(A)

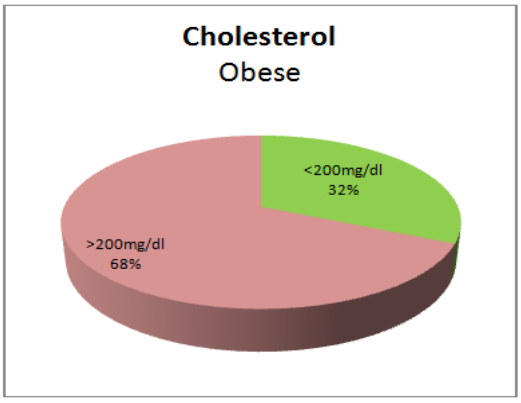


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Fig. 1: Total Cholesterol in the Study Groups (A-Non Obese, B-Obese)

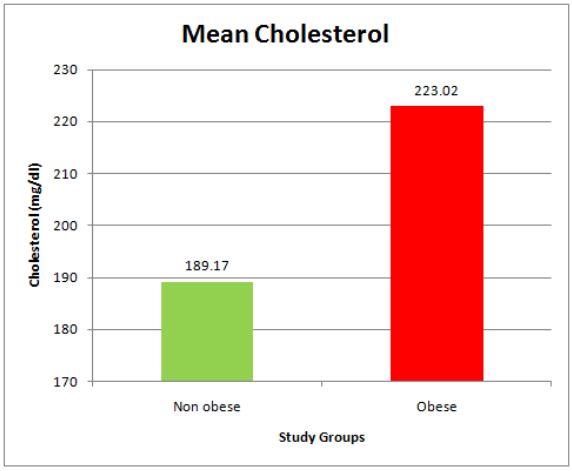


Fig. 2: Total Cholesterol: Comparison of Mean

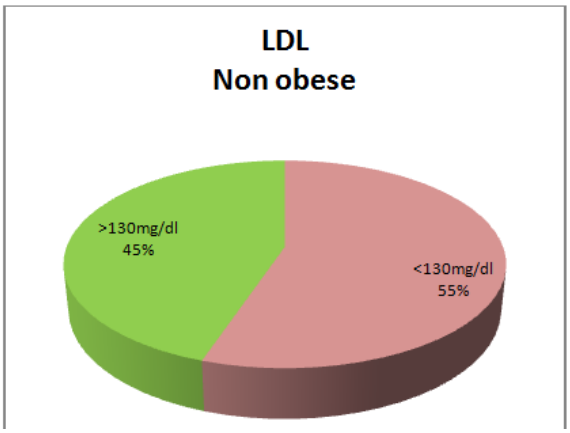


Fig. 3: LDL Cholesterol in the Study Groups

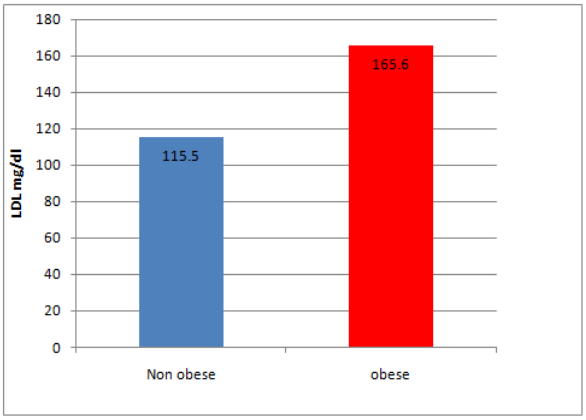


Fig. 4: LDL Cholesterol: Comparison of Mean

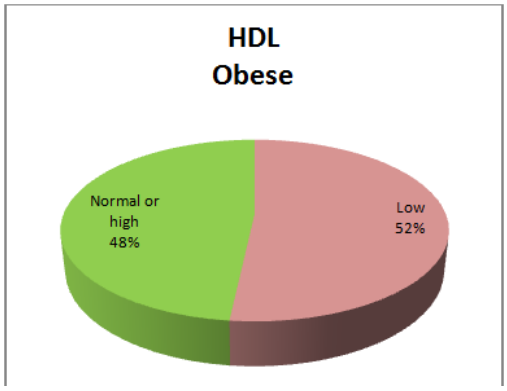


Fig. 5: HDL Cholesterol in the Study Groups

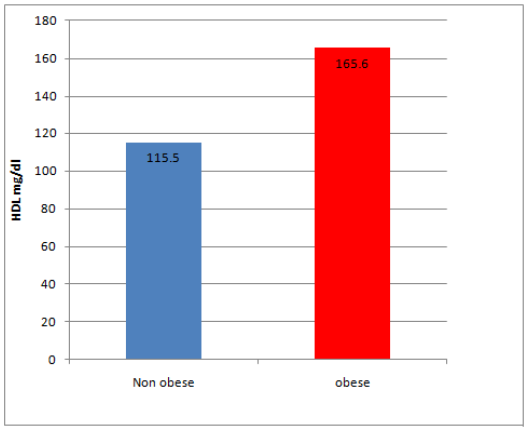
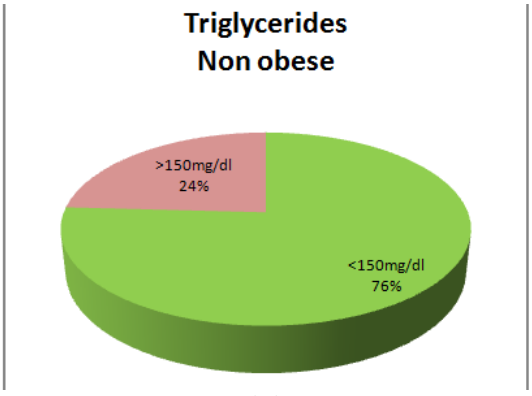
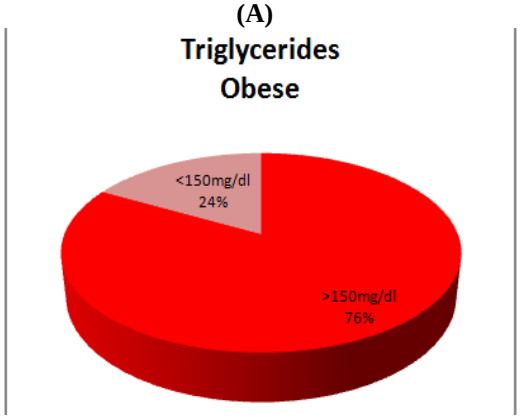


Fig.6: HDL Cholesterol: Comparison of Mean



(A)



(B)

Fig. 7: Triglycerides in the Study Groups (A-No-Obese, B-Obese)

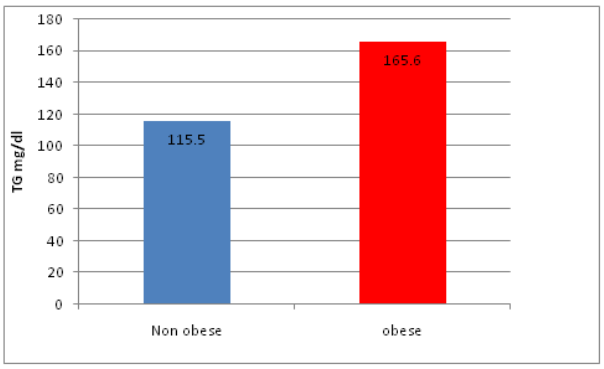


Fig. 8: Triglycerides: Comparison of Mean

DISCUSSION

In the present study, 140 subjects were included. They were grouped into two- obese and non obese- based on their Body Mass Index (BMI). BMI above 30 was taken as obese and BMI < 25 was as normal weight subjects. BMI between 25 and 30 was taken as overweight. Since there were only 5 people with obesity in the study group which could be statistically insignificant, they were also grouped among obese.82 subjects were obese and 58 were non obese.Total Cholesterol was estimated in all subjects and the results are summarised in table 1 and charts 1 and 2. A chi-square test was conducted to find out the relationship between total cholesterol in obese and non obese people. Result were obtained as follows; $\chi^2(1,N=140)=7.824$, P=0.005. The study showed that the total Cholesterol was significantly higher in obese people compared to non obese. These findings correlate well with the findings of Philip et al [8]. In this study, Cholesterol levels in conjunction with hypertension increase with the rising trend in overweight and obesity while amplifying the burden of cardiovascular disease.

LDL Cholesterol was estimated in all subjects and the results are summarised in table 2 and charts 3 and 4. A chi-square test ($\chi^2(1,N=140)=6.894$, P=0.009) was conducted to find out the relationship between LDL-Cholesterol in obese and non obese people. Results show that the LDL Cholesterol was significantly higher in obese people compared to non obese. HDL Cholesterol was estimated in all subjects and the results are summarised in table 3 and charts 5 and 6. A chi-square test χ^2 (2, N=140) =1.695, P=0.429 was conducted to find out the relationship between HDL-Cholesterol in obese and non obese. The result shows that it is insignificant because p value is greater than 0.05. These findings correlate well with the studies of Grundy SM and Barnett JP [9] except in the case of HDL cholesterol. One of the most common consequences of obesity is dyslipidemia, that is, elevations of very low-density lipoprotein (VLDL) triglycerides and low-density lipoprotein (LDL) cholesterol and low concentrations of high-density lipoprotein (HDL) cholesterol.

Triglyceride was estimated in all subjects and the results are summarised in table 4 and charts 7 and 8. A chi-square test showed $\chi^2(1,N=140)$ showed P=0.005).From the graphs it is seen that the mean TG is higher in obese compared to non obese. These findings correlate well with the findings of Lemieux *et al*. [10]. Hypertriglyceridemia is directly associated with elevated waist circumference and this hypertriglyceridemic waist is a high risk clinical phenotype associated with atherogenesis.

CONCLUSION

By analyzing the results of the study conducted it was concluded that there was an increased risk of Dyslipidemia among obese compared to non-obese people. Although not all overweight or obese individuals are metabolically unhealthy, the majority are at risk of developing atherosclerotic diseases due to dyslipidemia especially when combined with other risk factors like hypertension,diabetes and smoking, physical inactivity, and consumption of an atherogenic diet. Hence a community based education in this regards is of utmost important.

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