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Original research article: Correlation between insulin resistance (by HOMA-IR) and lipid profile among obese adults

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Abstract
Background: Obesity is a major growing global health problem of recent times. It is a leading preventable cause of death worldwide, with increasing rates in adults and children. Obesity, diabetes and hyperlipidaemia have been traditionally considered as diseases of affluence. The accumulation of central fat and presence of insulin resistance have associated with the dyslipidaemia seen in metabolic syndrome
Objective: To determine the correlation between insulin resistance (by HOMA-IR) and lipid profile among obese adults.
Material and Methods: A total of 100 obese individuals (BMI>25 Kg/m^2, WHR >0.9 in men, >0.8 in women) between the age group 18-60 years were included in this study. Fasting venous blood samples were collected and analyzed in auto analyzer for fasting blood glucose (FBG), Total cholesterol (TC), triglycerides, HDLc by enzymatic method, LDLc by Friedewald formula and insulin by immunofluoroscent method. Insulin resistance was calculated by a homeostasis model assessment (HOMA-IR) index (Fasting plasma glucose [mmol/L]X fasting plasma Insulin[uU/ml] /22.5)
Results: There was significant correlation between BMI and Insulin Resistance. Lipid profile were positively correlated with HOMA-IR whereas HDL showed negative correlation and all associations were statistically significant ($p<0.001$).
Conclusion: Our study reveals that there is a strong relationship between insulin resistance and lipid profile in obese adults

Keywords: obesity, insulin resistance, lipid profile, homeostasis model assessment insulin resistance

Introduction

Obesity is a major growing global health problem of recent times. It is a leading preventable cause of death worldwide with increasing rates in adults and children [1, 2]. In 2015, 600 million adults (12%) and 100 million children were obese [3]. In India the prevalence of overweight increased from 9.7% near the turn of the century to nearly 20% in studies reported after 2010 [4]. (Thesis) Obesity contributes to various chronic disease that accounts for morbidity and mortality in middle-aged and older persons [5].

Obesity, diabetes and hyperlipidaemia have been traditionally considered as diseases of affluence. A wealth of data indicates that Asian Indian people abdominal obesity and insulin resistance and develop glucose intolerance more often [6].

In many studies obesity has been clearly demonstrated to be associated with insulin resistance**.** Insulin resistance in turn has been found to be linked with many of the conditions typically associated with obesity and other conditions that appear to be less typically associated with simple obesity. These effects have generally been observed across sex ethnic and glucose tolerance categories [7]. The accumulation of central fat and presence of insulin resistance have associated with the dyslipidaemia seen in metabolic syndrome [8].

Adipocytes and adipose tissues are the key players in the pathogenesis of insulin resistance associated with obesity. Hypertrophic dysfunctional adipocytes, mainly encountered in Visceral adipose tissue (VAT) and upper body Subcutaneous adipose tissue (SAT), are highly lipolytic resulting in enhanced free fatty acids (FFA) release as well as in impaired secretion of adipokines (increased leptin and resistin, decreased adiponectin etc.) into the circulation [9]. The accumulation of central fat and presence of insulin resistance have associated with the dyslipidaemia as seen in metabolic syndrome. Abnormalities of lipoproteins are most pronounced with central obesity. Centrally obese individuals have an increased proportion of visceral fat, and visceral adipose tissue promotes insulin resistance.

Abnormalities of lipoproteins are most pronounced with central obesity. Centrally obese individuals have an increased proportion of visceral fat, and visceral adipose tissue promotes insulin resistance [7]. Also the better ability of lipid ratios to predict cardiovascular disease compared with single lipid markers is of particular clinical relevance and can be possibly explained by association of lipid ratios with a cluster of cardiovascular risk [10]. The present study was undertaken to determine the relationship of insulin resistance, lipid profile and its ratio in obese adults.

Material and Methods

Study Design

This study was carried out in the SMS Medical College & Hospital Jaipur to determine the Correlation between Insulin Resistance (by HOMA-IR) And Lipid Profile among Obese Adults. A total of 100 obese individuals (BMI>25 Kg/m^2, WHR >0.9 in men, >0.8 in women) between the age group 18-60 years were included in this study.

Sample Collection and Methods

Fasting venous blood samples were collected and analyzed in auto analyzer for fasting blood glucose (FBG), Total cholesterol (TC), triglycerides, HDLc by enzymatic method, LDLc by Friedewald formula and insulin by immunofluoroscent method. HbA1c was analyzed in whole blood sample by turbidometric method. The estimate of insulin resistance was calculated by a homeostasis model assessment (HOMA-IR) index (Fasting plasma glucose [mmol/L]X fasting plasma Insulin[uU/ml] /22.5)

Statistical analysis

Data was analyzed using SPSS 19.0 statistical software. Continuous data were summarised in forms of Mean and SD; and the difference in means were analyzed using Student unpaired t-test. Correlation between insulin resistance (HOMA-IR) and variable lipid profiles were found out using Pearson's correlation coefficient.

Results

Out of 100 obese adults; 50 were male and 50 were female. The mean age was 37.85 ± 9.06, the mean weight was 83.96± 10.78; the mean height was 164.5± 7.39 and the mean BMI were 30.97±3.63. In this study there is statistically significant correlation between HOMA-IR and with various lipid profile parameters. (Table 1)

Similarly there is statistically significant correlation between BCF and with various lipid profile parameters. (Table 2)

Table 1: Comparison of mean of HOMA-IR with other parameters

Parameters	Mean	SD	P value
HOMA – IR	2.33	0.74	0.001 S
BMI	30.97	3.63	
TC	209.74	26.72	0.001 S
TG	156.24	41.01	0.001 S
HDL	41.94	3.37	0.001 S
LDL	136.37	23.35	0.001 S
VLDL	31.26	8.25	0.001 S

Table 2: Comparison of mean of BCF with other parameters

Parameters	Mean	SD	P value
BCF	173.39	44.34	0.001 S
BMI	30.97	3.63	
TC	209.74	26.72	0.001 S
TG	156.24	41.01	0.001 S
HDL	41.94	3.37	0.001 S
LDL	136.37	23.35	0.001 S
VLDL	31.26	8.25	0.001 S

Discussion

Obesity is a cause of insulin resistance, but is also strongly associated with hypertension, dyslipidemia and glucose intolerance. Thus insulin resistance and lipid profile levels were studied in obese adults. Increases in BMI are associated with increases in total cholesterol (TC), triglyceride, total low density lipoprotein and small dense low density lipoprotein and with decreases in high density lipoprotein [11].

In our study mean BMI of the study population was 30.97. Mean HOMA-IR score was 2.33. HOMA-IR score> 2 was taken as cut-off for insulin resistance in our study. There was significant correlation between BMI and Insulin Resistance. So, more the obesity more is the insulin resistance. Our findings are consistent with the study conducted by Chizumi Yamada *et al*. [12] Saghafi-Asl *et al.*, [13] who also reported there is a significant correlation between HOMA-IR and BMI. The difference in HOMA-IR value in different studies are due to the fact that HOMA-IR value is dependent on the ethnicity of the population, dietary habits and different methods used for calculation of HOMA-IR score.

In our study we estimated total cholesterol (TC), triglycerides (TG), low density lipoprotein (LDL), high density lipoprotein (HDL), very low density lipoprotein (VLDL) Mean value of TC, TG, LDL, HDL, VLDL, TC/HDL, TG/HDL were 209.74 mg/dl, 156.24 mg/dl,136.37 mg/dl, 41.94mg/dl, 31.26 mg/dl, 5.05, 4.26 respectively. These are above the cut-off vales for Indian population [4]. TC, TG, LDL, VLDL, TC/HDL, TG/HDL were positively correlated with HOMA-IR whereas HDL showed negative correlation and all associations were statistically significant ($p<0.001$).

Arti Gurung *et al.*, [14] studied 50 obese adults and compared with 50 controls and reported that mean values for TC, TG, HDL, LDL, VLDL, TC/HDL, TG/HDL and HOMA-IR were 198.02, 201.66, 36.8, 118.6, 40.36, 5.47, 5.67, 5.54 respectively. Their data showed TC, TG, LDL, VLDL, TC/HDL, TG/HDL, LDL/HDL levels were statistically significant at $p<0.05$ level in obese than controls, although on the contrary to our study, they did not find any significant correlation of HDL with insulin resistance in obese subjects.

In another study conducted by Liying Zhang *et al.*, [15] reported that when HOMA-IR was used as a continuous variable in the adjusted regression models, TG, TG/HDL-C and TC/HDL-C ratio were significantly association with HOMA-IR in both overweight/obese men and women ($P < 0.05$). The Pearson correlation coefficient between TG, TG/HDL and TC/HDL with HOMA-IR were 0.32–0.37, 0.33–0.41 and 0.24–0.33respectively. The optimal cut-offs for TG/HDL-C were 1.51 mmol/L in men and 0.84mmol/L in women. The optimal cut-offs for TG were 1.78 mmol/L in men and 1.49 mmol/L in women, respectively.

So, from our study it is quite evident that lipid parameters are associated with insulin resistance in overweight and obese Indian adults. Therefore TC, TG, LDL and VLDL can be used as surrogate markers for insulin resistance in Indian obese adults.

In this study beta cell function was calculated on the basis of fasting serum insulin level and fasting serum glucose level using same computer based HOMA 2 calculator. Mean value of BCF was 173.39%. Statistic analysis showed that BCF has significant correlation with BMI. As with increase in BMI, there is increase in insulin resistance, thus increases BCF value. Our results are consistent with various authors namely Gang Chen *et al*, [16], Lui J *et al*, [17] and Chen X *et al* [18] who also found positive significant correlation between BMI and BCF (HOMA-b) in obese non-diabetic patients. Although BCF increases in response to increased insulin resistance in obese adults initially; with further progression to T2DM, the beta cell function (HOMA-b) decreases as there is exhaustion of beta cells [19].

In this study we found that BCF value had shown significant positive linear correlation with TG, TC, LDL, VLDL and negative correlation with HDL. Similarly Hyun Yoon *et al.*,

[20] in their study found that mean BCF was 138.03% for dyslipidemic group and reported that the variables showing a significant difference in the mean of HOMA-b were age, BMI, TG, HDL-C and FBS (for all $p<0.001$)

In another study done by Fumiaki Imamura *et al.*, [21] found that age, BMI, HDL-C, and triglycerides(TG) were associated with both IR and β-cell dysfunction ($p< 0.02$ for both), but in opposite directions. Higher BMI was independently associated with more β-cell function or high HOMA-b value in initial stage of obesity.

Conclusion

BMI can be used as a marker to identify individuals with insulin resistance syndrome. Increase in BMI are associated with increase in TC, triglyceride, LDL and VLDL and with decrease in HDL. HOMA-IR showed significant positive association with obese adults. Therefore these lipid parameters can be used as surrogate markers for insulin resistance in obese adults

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