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Evaluation of insulin resistance in young obese and its relation with smoking

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Abstract

Aim: To assess insulin resistance in young obese and its relation with smoking. **Methodology:** Ninety healthy subjects of either gender was divided into two groups of 45 each. Group I were smokers and group II were non-smokers. The mean weight (kgs), height (cm) and BMI (kg/ m2) was calculated. Serum insulin was estimated by sandwich ELISA technique. Insulin resistance was calculated by the formula- [HOMA-IR = FBG (mmol/L) 9 FPI (mIU/ml)/22.5. The results were compiled and subjected for statistical analysis using Mann Whitney U test. P value less than 0.05 was set significant. **Results:** Group I had 25 males and 20 females and group II had 22 males and 23 females. There were 35 normal in group I and 23 in group II, 6 overweight in group I and 12 in group II and 4 obese in group I and 10 in group II. The difference was significant (P< 0.05). The mean HOMA-IR Index in group I was 1.84 and in group II was 1.60. The difference was non- significant (P> 0.05). **Conclusion:** Young smokers with obesity were prone to develop insulin resistance. Smokers can be counselled that discontinuation of smoking especially in early stages could bring substantial reversal of the damage caused and hence this is a strong motivating factor for smokers to discontinue early.

Keywords: Insulin Resistance, Obesity, Diabetes.

INTRODUCTION

There is increasing evidence that prevalence of IR is increasing in childhood and adolescence.[1] Interestingly IR has also been reported in lean Asian population as well as normal body weight subjects, with greater whole body fat and higher levels of muscle lipids. However, the mechanism which contributes to the impaired insulin signalling in the absence of obesity is not much clear.[2]

Smoking is the major global cause of preventable death. The most popular form of smoking is tobacco smoking and is practiced by over one billion people in the majority of all human societies.[3] Smoking is one of the modifiable risk factors for many chronic diseases, such as cancer, chronic obstructive lung disease, asthma, atherosclerosis, coronary heart disease and peripheral vascular disorders. Smoking also increases inflammation and oxidative stress to directly damage β cell function and to impair endothelial function. Long-term smoking is reported to increase insulin resistance (IR), inflammation, lipid peroxidation and endothelial cell dysfunction.[4] Smokers have been found to be at high risk compared to non-smokers for development of diabetes and a strong relationship between tobacco smoke and metabolic syndrome among adolescents has been observed. Various studies show that long term smoking decreases the insulin sensitivity.

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Adverse effects due to short-term smoking have been also observed; however very few studies are available regarding the effect of short-term cigarette smoking on insulin resistance in human.[5,6,7]

Homeostatic model assessment of insulin resistance (HOMA-IR) has been found to be a convenient and reliable method for evaluating IR. It is considered as a better marker than plasma insulin levels and compares well with insulin resistance measured by euglycaemic clamp method.[8] Considering this, we assessed insulin resistance in young obese and its relation with smoking.

METHODS

A sum total of ninety healthy subjects of either gender was selected in this prospective, observational study. All were enrolled for the study once they gave written consent. Ethical approval was also obtained before starting the study.

Demographic profile such as name, age and gender etc. was recorded. Subjects were divided into two groups of 45 each. Group I were smokers and group II were non-smokers. The mean weight (kgs), height (cm) and BMI (kg/ m2) was calculated. 5 ml of venous blood was collected and assessment of serum insulin, fasting blood glucose, triglycerides, high-density lipoprotein cholesterol were assessed on fully automated biochemistry analyser. Serum insulin was estimated by sandwich ELISA technique. Insulin resistance was calculated by the formula- [HOMA-IR = FBG (mmol/L) 9 FPI (mIU/ml)/22.5. The results were compiled and subjected for statistical analysis using Mann Whitney U test. P value less than 0.05 was set significant.

RESULTS

Table 1: Distribution of subjects.

Groups	Group I	Group II
Male	25	22
Female	20	23

Group I had 25 males and 20 females and group II had 22 males and 23 females [Table 1].

Table 2: Assessment of BMI.

BMI	Group I	Group II	P value
Normal	35	23	0.05
Overweight	6	12	0.04
Obese	4	10	0.05

There were 35 normal in group I and 23 in group II, 6 overweight in group I and 12 in group II and 4 obese in group I and 10 in group II. The difference was significant (P< 0.05) [Table 2].

Table 3: Comparison of HOMA-IR Index.

Groups	HOMA-IR Index	P value
Group I	1.84	0.72
Group II	1.60	

The mean HOMA-IR Index in group I was 1.84 and in group II was 1.60. The difference was non- significant (P> 0.05) [Table 3].

DISCUSSION

Insulin resistance (IR) is regarded as primary initiating factor for obesity, diabetes mellitus, dyslipidemia and cardiovascular disease (CVD) in adults. It is known to be associated with abnormal lipid profiles in obese children, adolescents, and adults. Smoking causes molecular changes in lipid metabolism and glucose metabolism. In healthy young men, acute smoking showed an increased IR. IR is considered to be primary initiating factor for obesity, type 2 diabetes mellitus (T2DM or T2D), dyslipidemia and cardiovascular disease in adults. IR is known to be associated with abnormal lipid profile in obese children, adolescents and adults.[9] Considering this, we assessed insulin resistance in young obese and its relation with smoking.

Our results showed that group I had 25 males and 20 females and group II had 22 males and 23 females. Acar et al.[10] aimed to evaluate the relationship between the insulin resistance and smoking in individuals without advanced age, obesity, high blood glucose levels and hypertension. In this study 100 patients (50 non-smokers, 50 smokers) were included. Between both groups, body mass index, systolic and diastolic blood pressure, triglyceride, LDL cholesterol, fasting blood glucose, insulin, HbA1c and HOMA values were similar (P>0.05). HDL cholesterol levels in smokers compared to non-smokers was statistically significant (P<0.05). In stepwise multiple linear regression analysis, triglyceride and waist/hip ratio were identified as an important independent predictor of an increase in HOMA in smokers.

Our results showed that there were 35 normal in group I and 23 in group II, 6 overweight in group I and 12 in group II and 4 obese in group I and 10 in group II. Targher et al.[11] reported a link between cigarette smoking and insulin resistance in T2DM and associated dyslipidemia. These proatherogenic changes finally lead to metabolic syndrome and CAD.

Our results showed that the mean HOMA-IR Index in group I was 1.84 and in group II was 1.60. Mashrani et al.[12] reported insulin resistance to be associated with activation of the JNK pathway in non- obese subjects. Gupta et al.[13] examined the effect of short-term cigarette smoking on insulin resistance and lipid profile in asymptomatic healthy adults. This study comprised of 44 healthy male subjects in the age group of 18-40 years having BMI 25+3 and WHR < 1.0. Of these 22 smokers were included in the study group and 22 non-smokers in the control group. Subject selection was done such that one smoker and one non-smoker sibling or first- degree male relative were selected from the same family. They compared fasting plasma glucose, insulin, lipid profile, and homeostatic model assessment index (HOMA Index) as a measure of insulin resistance between both the groups. Our observation showed that significantly higher values of serum glucose (133.36 +/- 23.45 mg/dl; P < 0.001), serum insulin (32.04 +/- 6.0 2 microU/ml; P < 0.001) and HOMA index (3.62 +/- 0.21; P < 0.001) were found in smokers as compared to non-smokers (serum glucose 86.95 +/- 19.32 mg/dl, insulin 20.09 +/- 4.8 microU/ml, HOMA index 3.29 +/- 0.30). No significant difference was observed for number of subjects having insulin resistance (HI > 3.8) and lipid profile in both the groups in this study.

Ikeda et al.[14] examined the clinical significance of the insulin resistance index as determined by homeostasis model assessment (HOMA-IR), we investigated the relationship between HOMA-IR and the insulin resistance estimated by the euglycemic-hyperinsulinemic clamp method in various subgroups and compared the significance of HOMA-IR with that of fasting plasma insulin levels (FIRI). HOMA-IR was significantly correlated to the inverse of the glucose infusion rate (1/GIR) in both diabetic and non-diabetic subjects. Giovanni et al.[15]evaluated the effects of chronic cigarette smoking on insulin sensitivity in patients with noninsulin-dependent diabetes mellitus (NIDDM). They examined 28 smokers and 12 nonsmokers with NIDDM, of similar sex, age, body mass index, waist/hip ratio, alcohol consumption, physical activity level, glycometabolic control, diabetes duration, and treatment. Insulin and C-peptide responses to oral glucose load were significantly higher in smokers than nonsmokers, whereas glucose levels were not substantially different. During insulin clamp (20 mU/min.m2), carried out in combination with tritiated glucose infusion and indirect calorimetry, total glucose disposal was markedly reduced in smokers vs. nonsmokers [19 +/- 1.2 vs. 33 +/- 5 mumol/min.kg fat-free mass (FFM); P < 0.001], in a dose-dependent fashion (F = 6.8, P < 0.001 by ANOVA when subjects were categorized for number of cigarettes smoked per day). Oxidative and nonoxidative pathways of insulin-mediated intracellular glucose metabolism were similarly reduced in smokers vs. nonsmokers. Plasma free fatty acid

levels (240 +/- 33 vs. 130 +/- 23 microEq/L; P < 0.05) and lipid oxidation rate (1.39 +/- 0.1 vs. 0.95 +/- 0.2 mumol/ min.kg FFM; P < 0.05) were less suppressed by hyperinsulinemia in smokers than nonsmokers. Chronic cigarette smoking seems to markedly aggravate insulin resistance in patients with NIDDM.

Fov et al.[16] determined the association between smoking and incident diabetes among U.S. adults. They examined the relationship between smoking status categories (never, former, and current) and incident 5-year type 2 diabetes among 906 participants free of diabetes at baseline. We also considered the effect of pack-year categories (never, former <20 pack-years, former > or = 20 pack-years, current <20 pack-years, and current > or = 20 pack-years) upon diabetes incidence. Of current smokers, 96 (25%) developed diabetes at 5 years, compared with 60 (14%) never smokers. After multivariable adjustment, current smokers exhibited increased incidence of diabetes compared with never smokers (odds ratio [OR] 2.66, P = 0.001). Similar results were found among current smokers with > or = 20 pack-years with normal glucose tolerance (5.66, P = 0.001).

CONCLUSION

Authors found that young smokers with obesity were prone to develop insulin resistance. Smokers can be counselled that discontinuation of smoking especially in early stages could bring substantial reversal of the damage caused and hence this is a strong motivating factor for smokers to discontinue early.

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