Journal of Advanced Medical and Dental Sciences Research

@Society of Scientific Research and Studies NLM ID: 101716117

Journal home page: www.jamdsr.com

doi: 10.21276/jamdsr

Index Copernicus value = 85.10

(e) ISSN Online: 2321-9599;

(p) ISSN Print: 2348-6805

Original Article

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

¹Ranjeet Kumar, ²Ankur Arvind Shah

¹Assistant Professor, Dept of Biochemistry, Major SD Singh Medical College and Hospital, Farrukhabad, UP., India;

²Assistant Professor, Dept of Biochemistry, KM Medical College and Hospital, Sonkh Road, Mathura, U.P., India

ABSTRACT:

Background: Insulin resistance (IR) is regarded as primary initiating factor for obesity, diabetes mellitus, dyslipidemia and cardiovascular disease (CVD) in adults. The present study was conducted to assess insulin resistance in young obese and its relation with smoking. **Materials & Methods:** 82 healthy subjects in the age group of 20–30 years of either gender were enrolled. Subjects were divided into two groups. Group I were smokers and group II were non-smokers. The subjects were divided according to their BMI (kg/ m2), as per ICMR guidelines. 5 ml of venous blood was collected in the fasting state. 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. **Results:** Normal subjects were 70% in group I and 48% in group II, overweight 20% in group I and 20% in group II and obese in 10% in group I and 32% in group II. The mean HOMA-IR Index in group I as 1.78 and in group II was 1.62. The difference was non- significant (P> 0.05). **Conclusion:** Young smokers with obesity were prone to develop insulin resistance.

Key words: insulin resistance, Obesity, diabetes

Received: 14 November 2017

Accepted: 18 December 2017

Corresponding author: Ankur Arvind Shah, Assistant Professor, Dept of Biochemistry, KM Medical College and Hospital, Sonkh Road, Mathura, U.P., India

This article may be cited as: Kumar R, Shah AA. Assessment of insulin resistance in young obese and its relation with smoking. J Adv Med Dent Scie Res 2018;6(1):154-157.

INTRODUCTION

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.¹ There is increasing evidence that prevalence of IR is increasing in childhood and adolescence. 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 (Beta) 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 The present study was conducted to assess insulin resistance in young obese and its relation with smoking.

MATERIALS & METHODS

The present study consisted of 82 healthy subjects in the age group of 20–30 years of either gender. All were enrolled for the study once they gave written consent.

Data such as name, age and gender etc. was recorded. Subjects were divided into two groups. Group I were smokers and group II were non-smokers. The subjects were divided according to their BMI (kg/ m2), as per ICMR guidelines. 5 ml of venous blood was collected in the fasting state. Serum insulin, fasting blood glucose (FBG), triglycerides (TG), high-density lipoprotein cholesterol (HDL-C) 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. Results thus obtained were subjected to statistical analysis. P value less than 0.05 was considered significant.

RESULTS

Table I Distribution of subjects as per BMI

| BMI | Group I | Group II | P value |
|------------|---------|----------|---------|
| Normal | 70% | 48% | 0.02 |
| Overweight | 20% | 20% | 1 |
| Obese | 10% | 32% | 0.05 |

Table I, graph I shows that normal subjects were 70% in group I and 48% in group II, overweight 20% in group I and 20% in group II and obese in 10% in group I and 32% in group II. The difference was significant (P < 0.05).





Table II Comparison of HOMA-IR Index

| Groups | HOMA-IR Index | P value |
|----------|---------------|---------|
| Group I | 1.78 | 0.91 |
| Group II | 1.62 | |

Table II, graph II shows that mean HOMA-IR Index in group I as 1.78 and in group II was 1.62. The difference was non-significant (P> 0.05).

Graph II Comparison of HOMA-IR Index



DISCUSSION

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.⁵ IR is known to be associated with abnormal lipid profile in obese children, adolescents and adults. There is increasing evidence that prevalence of IR is increasing in childhood and adolescence.⁶ 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 signaling in the absence of obesity is not much clear.⁷ Although smoking is known to decreases body weight, it is associated with central obesity. Few studies have demonstrated that smoking cessation is associated with improvement in insulin sensitivity.8,9 The present study was conducted to assess insulin resistance in young obese and its relation with smoking.

In present study, normal subjects were 70% in group I and 48% in group II, overweight 20% in group I and 20% in group II and obese in 10% in group I and 32% in group II. Mashrani et al¹⁰ reported insulin resistance to be associated with activation of the JNK pathway in non- obese subjects.

We observed that mean HOMA-IR Index in group I as 1.78 and in group II was 1.62. Juneja et al¹¹ evaluated the prevalence of insulin resistance in young obese subjects and its relation to smoking is not well established. This study comprising seventy-five healthy young adults was undertaken to find insulin resistance in obese smokers and non- smokers both. Present study showed an overall prevalence of raised homeostatic model assessment of insulin resistance in 14.7 % otherwise healthy young subjects (20-30 years age group). Non-smokers did not show any significant correlation between insulin resistance and body mass index at either stage (normal, pre-obese as well as obese). Smokers also did not show any significant difference of insulin resistance in normal and prestages. However, marked increase obese in homeostatic model assessment of insulin resistance was observed in obese smokers. Homeostatic model assessment of insulin resistance showed a linear trend in relation to body mass index and its values were found to be higher in smokers.

Solanki et al¹² assessed the effects of smoking on IR. This study comprised 40 young adults who smoked minimum 6 beedis/cigarettes daily and 25 age matched control subjects. Blood samples were collected in plain bulb and biochemical analyses like fasting blood glucose (FBG), lipid profile, fasting insulin were done and then Homeostatic Model Assessment of Insulin Resistance was used to find out IR. Smokers has significantly higher level of fasting blood glucose, fasting insulin, total cholesterol, triglycerides, LDL-C, HOMA-IR index and lower level of HDL-C as compared to non-smokers. A significant association was noted between the smoking status, including both the numbers of cigarettes/beedis smoke per day and fasting insulin level as well as for HOMA-IR index. Smokers have a high risk of developing an insulin resistance and hyperinsulinemia as compared with a matched group of non-smokers. This may help to explain the high risk of cardiovascular disease in smokers.

Bajaj¹³ postulated that nicotine triggers insulin resistance in smokers. Raised HOMA IR and TG in smokers can be attributed to nicotine content of cigarette/ bidis. Similarly, glucogenic response can also be attributed to nicotine induced increase in plasma cortisol levels. It is postulated that smoking induced biochemical changes in subjects with obesity makes them vulnerable to insulin resistance and T2DM. Targher et al¹⁴ reported a link between cigarette smoking and insulin resistance in T2DM and associated dyslipidemia. These proatherogenic changes finally lead to metabolic syndrome and CAD.

CONCLUSION

Authors found that young smokers with obesity were prone to develop insulin resistance.

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