wjpmr, 2020,6(12), 325-327

SJIF Impact Factor: 5.922

WORLD JOURNAL OF PHARMACEUTICAL AND MEDICAL RESEARCH

www.wjpmr.com

Research Article

ISSN 2455-3301

WJPMR

STUDY OF CORRELATION BETWEEN BODY MASS INDEX AND BLOOD GLUCOSE LEVEL AMONG MEDICAL STUDENTS

Sana Bahadur*, Ammar Abdul Rahman and Muhammad Ramzan

Pakistan.

***Corresponding Author: Sana Bahadur**
Pakistan.
DOI: https://doi.org/10.17605/OSF.IO/4USCF

Article Received on 11/09/2020 **Article Revised on 01/10/2020** **Article Accepted on 21/10/2020**

ABSTRACT

Objective: To study the correlation between serum glucose and BMI in undergraduate students. **Material and methods:** This cross sectional study was conducted at Department of Medicine, Sheikh Zayed Hospital Rahim Yar Khan in month of December 2019. Total 166 students either male or female having age from 18 years to 24 years were selected. Serum blood glucose was measured and BMI was calculated. **Results:** Minimum age of students was 17 years and maximum age was 23 years. Mean age of the students was 19.17 ± 1.5 years, mean BMI was 24.65 ± 6.196 and mean serum glucose level was 126.23 ± 25.56mg/dl. The Pearson correlation test showed that the level of level of serum glucose increased with increasing BMI. This positive correlation was statistically significant (r= 0.625, P= 0.000). **Conclusion:** Findings of current study showed a positive correlation between BMI and serum glucose levels. Significant different between mean serum glucose levels and different age groups was detected. Results of this study also showed that there is insignificant difference between serum glucose levels of male and female students.

KEYWORDS: BMI, correlation, serum glucose, obesity.

INTRODUCTION

In general population, adiposity is measured by body mass index (BMI). BMI is calculated as weight in kg, divided by square of height in meters.[1] An individual can be divided into different categorizes according to their BMI i.e. under weight (if BMI ≤18.5), normal weight (if BMI ranges from 18.5–24.9), over weight (if BMI ranges from 25 to 29.9) and obese (if BMI is ≥30).[2]

Increased BMI can be a risk for carcinomas, stroke and heart diseases.[3]

In patients of type-II diabetics, obesity is a modifiable risk factor. The mechanism by which obesity induces insulin resistance is poorly understood. Several biological products (free fatty acids, leptin, adiponectin, TNF-alfa and resistin) secreted by adipocytes, which modulated the insulin secretions. Weight of body and insulin action may responsible for insulin resistance.[4] It is assumed that there is a positive correlation between serum sugar levels and BMI. Obesity is one the major public health problems globally. In different parts of the world the average BMI is rising few percent/decade, thus fuelling the concern about the effects of increased adiposity on health.[5]

So, a study was planned to detect the correlation between BMI and serum glucose among the undergraduates.

MATERIAL AND METHODS

This cross sectional study was conducted at Department of Medicine, Sheikh Zayed Hospital, Rahim Yar Khan in month of December 2019. Total 166 students either male or female having age from 18 years to 24 years were selected. Students with any systemic disease were excluded from the study. History was taken from all the students regarding family history of diabetes mellitus.

Weight and height of all the patients was measured by weighing machine and measuring tape to calculate BMI. Random blood sample was drawn and sent to laboratory for serum glucose levels. Findings of the laboratory test and BMI was entered on pre-designed proforma along with demographic profile of all the students. All the collected data was entered in SPSS version 17. Mean and SD was calculated for age, BMI, serum glucose level. Frequencies was calculated for gender and family history of DM. Pearson correlation test was applied to check the correlation between BMI and serum glucose levels. Stratification was done for age, gender and family history of DM. Post stratification student t test was applied to detect the difference of serum glucose levels

for these variables. P value ≤ 0.05 was considered statistically significant.

RESULTS

Total 166 students were selected were for this study. Minimum age of students was 18 years and maximum age was 24 years. Mean age of the students was 19.17 ± 1.5 years, mean BMI was 24.65 ± 6.196 and mean serum glucose level was 126.23 ± 25.56mg/dl. The Pearson correlation test showed that the level of level of serum glucose increased with increasing BMI. This positive correlation was statistically significant (r= 0.625, P= 0.000). (Table 1) Students were divided into two age groups i.e. age group 18-20 years and 21-24 years. Total 160 students belonged to age group 18-20 years and only 6 students belonged to age group 21-24 years. In age group 18-20 years, mean serum glucose was 126.96 ± 25.68 mg/dl. In age group 21-24 years, mean serum glucose level was 106.33 ± 9.24 mg/dl. Statistically significant (P = 0.025) difference of mean serum glucose between both age groups was noted. (Table 2) Male patients were 77 and female patients were 89. Mean serum glucose level of maple students was 128.64 ± 26.24 and mean serum glucose level of female students was 124.15 ± 24.92. The difference of mean serum glucose levels between male and female patients was statistically insignificant with p value 0.714. (Table 3) Total 91 students found with family history of DM and 75 students found without family history of DM. Mean serum glucose level was 124.23 ± 25.50 mg/dl in students with family history of DM and 128.65 ± 25.59 mg/dl without family history of DM. The difference between mean serum glucose levels between both groups was statistically insignificant with p value 0.813. (Table 4)

Table 1: Correlation of BMI with serum glucose.

	Serum glucose (mg/dl)	
	Pearson correlation (r)	P-value
BMI	0.254	0.009

Table 2: Comparison of mean serum glucose level between the both age groups.

Age Group	n	Mean	Std. Deviation	P Value
18-20 years	160	126.96	25.68	0.025
21-24 years	6	106.33	9.24	

Table 3: Comparison of mean serum glucose level for gender.

Gender	n	Mean	Std. Deviation	P Value
Male	77	128.64	26.24	0.714
Female	89	124.15	24.92	

Table 4: Comparison of mean serum glucose level for gender.

Family history of DM	n	Mean	Std. Deviation	P Value
Yes	91	124.23	25.50	0.813
No	75	128.65	25.59	

DISCUSSION

In current study a positive correlation (r = 0.254) between serum glucose and BMI was detected. In different studies of the world, positive correlation was also detected between serum glucose and BMI.[6,7] Ethnicity also affects the association between diabetes mellitus and obesity which may explains the various levels of association between the serum glucose levels and obesity reported by different studies.[8] In age group 18-20 years, mean serum glucose was 126.96 ± 25.68 mg/dl. In age group 21-24 years, mean serum glucose level was 106.33 ± 9.24 mg/dl. Statistically significant (P = 0.025) difference of mean serum glucose between both age groups was noted. Increasing incidence of obesity globally is attributed to dietary habits and changing in life styles.[9] The mechanism by which obesity induces insulin resistance is poorly understood. Obesity causes peripheral resistance to insulin-mediated glucose uptake and may also decrease the sensitivity of the beta-cells to glucose.[10] These changes are largely reversed by weight loss, leading to a fall in blood glucose concentrations towards normal levels. Weight gain precedes the onset of diabetes; conversely, weight loss is associated with a decreased risk of type 2 diabetes.[11,12]

The administration of resistin, an adipocyte derived hormone, decreases while the neutralization of resistin increases insulin mediated glucose uptake by the adipocytes. Thus, resistin may be a hormone that links obesity to diabetes.[4] Leptin is produced by adipocytes and is secreted in proportion to the adipocyte mass. It signals the hypothalamus about the quantity of stored fat. Studies in humans and animals have shown that leptin is associated with obesity and insulin resistance.[13] The deficiency of adiponectin, an adipocyte-derived hormone, plays a role in the development of insulin

resistance and subsequently, type 2 diabetes.[14] Retinol-binding protein 4, free fatty acids, tumor necrosis factor-alpha, plasminogen activator inhibitor 1, interleukin-1 beta, uncoupling protein 2 and obestatin are also implicated in the adipose tissue induced pathogenesis of type 2 diabetes.[15]

BMI is a good measure of adiposity; however, the relationship between actual body fat and BMI differs between ethnic groups, and as a consequence, the cut off points for the overweight status and obesity based on BMI, will have to be ethnicity specific.[16]

CONCLUSION

Findings of current study showed a positive correlation between BMI and serum glucose levels. Significant difference between mean serum glucose levels of different age groups was detected. Results of this study also showed that there is insignificant difference between male and female students.

REFERENCES

1. Hu F. Obesity epidemiology. Oxford: Oxford university press, 2008; 87-97.
2. World Health Organization Obesity: Preventing and Managing the Global Epidemic. World Health Organization Geneva, Switzerland, 1997; 786-987.
3. Prospective Studies Collaboration, Whitlock G, Lexington S, She liker P, Clarke R, Emerson J, Halsey J, Qizilbash N, Collins R, Pete R. Body-mass index and cause-specific mortality in 900 000 adults: collaborative analyses of 57 prospective studies. The Lancet, 2009; 373(9669): 1083–96.
4. Stephan CM, Bailey ST, Bhatt S, Brown EJ, Banerjee RR, Wright CM, Patel HR, Anima RS, Lazar MA. The hormone resistin links obesity to diabetes. Nature, 2001; 409(6818): 307-12.
5. WHO Global InfoBase team. Surveillance of chronic diseases and risk factors: Country level data and comparable estimates. Geneva: World Health Organization, 2005.
6. Adame GB, Geoffrey CO, Bale GS, Ibrahim SA, Sane SH, Tambala MA. Relationship between random blood sugar and body mass index in an African population. Int J Diabetes & Metabolism 2006; 14: 144-5.
7. Jhanghorbani M, Hedley AJ, Jones RB, Gilmour WH. Is the association between glucose level and "all causes" and cardiovascular mortality risk dependent on body mass index? Med. J. Islamic Republic Iran, 1992; 6: 205-12.
8. Diaz VA, Mucinous AG, Baker R, Carniola M, Majid A. How does ethnicity affect the association between obesity and diabetes? Diabet Med., 2007; 24(11): 1199-204.
9. Pelletier DL, Rahm M. Trends in body mass index in developing countries. Food and Nutrition Bulletin, 1998; 19(3): 223-39.
10. DeFrantz RA, Ferrannini E. Insulin resistance. A multifaceted syndrome responsible for NIDDM, obesity, hypertension, dyslipidemia, and atherosclerotic cardiovascular disease. Diabetes Care, 1991; 14(3): 173-94.
11. Feebler JP. From obesity to diabetes. Pathophysiological considerations. Int J Obes Relat Metab Disord, 1992; 16(12): 937-52.
12. Knower WC, Pettit DJ, Saad MF, Charles MA, Nelson RG, Howard BV, Boards C, Bennett PH. Obesity in the Pima Indians: its magnitude and relationship with diabetes. Am J Clint, 1991; 53(6): 1543S-51S.
13. Niswender KD, Magnuson MA. Obesity and the beta cell: lessons from leptin. J Clin Invest, 2007; 117(10): 2753–6.
14. Kadowaki T, Yamauchi T, Kubota N, Hara K, Ueki K, Tobe K. Adiponectin and adiponectin receptors in insulin resistance, diabetes, and the metabolic syndrome. J Clin Invest, 2006; 116(7): 1784–92.
15. Kahn SE, Hull RL, Utzschneider KM. Mechanisms linking obesity to insulin resistance and type 2 diabetes. Nature, 2006; 444(7121): 840-6.
16. Deurenberg P, Yap M. The assessment of obesity: methods for measuring body fat and global prevalence of obesity. Best Pract Res Clin Endocrinol Metab, 1999; 13(1): 1-11.