Original Article

Correlation Between Anthropometric And Serum Glycemic Parameters In A Sample Of Healthy Male Pakistani Population

Uzma Zafar, Hamid Javaid Qureshi

Objective: To find out the correlation between anthropometric and serum glycemic parameters in a sample of healthy male Pakistan population.

Methods: Alt was a correlational study. Eighty male subjects fulfilling the inclusion criteria were included in this study. Anthropometric parameters including BMI, waist to hip ratio and waist circumference, were measured. Fasting serum glucose and fasting serum insulin were estimated. Insulin resistance was determined quantitatively by HOMA-IR. Serum glycemic parameters were correlated with anthropometric parameters.

Results: BA significant positive correlation was observed between fasting serum glucose and BMI, waist to hip ratio and waist circumference. A significant positive correlation was observed between fasting serum insulin and waist circumference but there was no significant correlation between serum insulin and other anthropometric parameters. A significant positive correlation was observed between insulin resistance and waist to hip ratio and waist circumference but there was no significant correlation between insulin resistance and BMI.

Conclusions: These results provide evidence for linkage between anthropometric and glycemic parameters in apparently healthy, non obese adults having anthropometric parameters within normal range. Abdominal obesity as measured by waist circumference correlates not only with insulin resistance but also with fasting serum insulin and fasting serum glucose even in healthy subjects.

Keywords: Glycemic parameters, anthropometric parameters, insulin resistance syndrome.

Introduction

Insulin resistance is a well established risk factor for cardiovascular disease (CVD) and type 2 diabetes mellitus (T2DM)[1]. Insulin resistance means that there is decreased ability of target organs like liver, adipose tissue and skeletal muscles to respond to normal circulating concentration of insulin. Insulin resistance is compensated by hyperinsulinemia, which further aggravates the insulin resistance by down-regulating the insulin receptors[2,3]. Some risk factors that commonly cluster together like dyslipidemia, hypertension and hyperglycemia have been termed the insulin resistance syndrome or the metabolic syndrome. The National Cholesterol Education Program's Adult Treatment Panel III report (ATPIII) defined criteria used to identify patients with insulin resistance syndrome. Criteria for ATP III are shown in **Table 1**. When a subject has three of the five listed criteria, a diagnosis of insulin resistance syndrome can be made. The World Health Organization (WHO) guidelines **(Table-2)** also viewed CVD and T2DM as primary outcomes of the insulin resistance syndrome[4]. From these guidelines, one can assess insulin resistance by certain simple parameters like systolic and diastolic blood pressure. Overweight and obesity are also associated with insulin resistance and metabolic syndrome. Therefore, simple measurements of waist circumference, waist to hip ratio or body mass index (BMI) are recommended to identify the body weight component of metabolic syndrome[4]. Insulin resistance can also be calculated quantitatively from fasting serum glucose and fasting serum insulin by Homeostatic model assessment (HOMA-IR)[5]. It has been experimentally proved that increased intake of carbohydrates is associated with increased release of insulin, increased synthesis of fat and increased insulin resistance resulting in insulin resistance syndrome4. The present study was designed to see the correlation between anthropometric parameters (including BMI, waist to hip ratio and waist circumference) insulin resistance syndrome and serum glycemic indices (including fasting serum glucose, fasting serum insulin and insulin resistance calculated by HOMA-IR) in healthy subjects.

Study Design

It was a correlational study. This study was conducted in the Department of Physiology and Cell Biology, University of Health Sciences Lahore. Eighty healthy male subjects between the ages of 20-40 years were

selected fulfilling the inclusion criterion. They had no previous history of hypertension and diabetes mellitus. Their BMI and waist circumference were within the normal range given by ATPIII and WHO criteria.

Methodology

After subject selection, written informed consent was taken from the subjects. Demographic information was taken, history and physical examination was completed. Waist circumference (in centimeters) was measured in the horizontal plane midway between the costal margin and the iliac crest at the end of normal expiration. Hip circumference (in centimeters) was taken at the widest point of gluteal region. Waist to hip ratio was calculated by dividing waist circumference by hip circumference. Height (in meters) and weight (in kilogram) were measured in subjects wearing usual clothes, without shoes. Body mass index (BMI) was calculated as weight divided by the square of height in meters. Blood pressure was measured with a mercury sphygmomanometer on the right arm with the subjects in sitting position after a five minute period of rest6. After 8 10 hours of overnight fast, 5 ml of venous blood was drawn by aseptic techniques. Serum glucose was determined after enzymatic oxidation in the presence of glucose oxidase by enzymatic colorimetric test for Glucose (Human Gesellschaft for Biomedica and Diagnostica D-65205 Wiesbaden-Germany)7. Insulin was measured in human serum quantitatively by immunoenzymometric assay with an automated EIA analyzer CODA, Bio-Rad laboratories, Hercules, CA, USA with the kit (Monobind Inc. Lake Forest, CA 92630, USA)8. Insulin resistance was calculated from fasting serum glucose (mmol/l) and fasting serum insulin (μ IU/ml) by HOMA-IR (Homeostatic model assessment for insulin resistance) using following formula5. HOMA-IR = Fasting serum glucose x Fasting serum insulin / 22.5

Statistical Analysis

The data was entered and analyzed using SPSS version 17 (Statistical Package for Social Sciences). Mean ± SEM (Standard error of mean) were given for normally distributed quantitative variables and median with IQR (Interquartile range) were given for non-normally distributed quantitative variables. Pearson's correlation and Spearman Rho correlation were applied to correlate normally and non-normally distributed quantitative variables. A p - value of < 0.05 was considered as statistically significant.

Results

Table 3 shows anthropometric and serum glycemic parameters in 80 subjects. A significant positive correlation was observed between fasting serum glucose and BMI, waist to hip ratio and waist circumference. A significant positive correlation was observed between serum insulin and waist circumference but there was no significant correlation between serum insulin and BMI and waist to hip ratio. A significant positive correlation was observed between insulin resistance and waist to hip ratio and waist circumference but there was no significant correlation between insulin resistance and BMI. **(Table-4. Figures 1-5)**

Discussion

The present study determined correlations between serum glycemic parameters including fasting serum glucose, serum insulin and insulin resistance, and anthropometric parameters including BMI, waist to hip ratio and waist circumference, in a sample of healthy male population. Although the subjects selected in this study were not having insulin resistance syndrome, as their anthropometric measurements were below the cut off levels given by WHO and ATP111, but significant positive correlation was found between insulin resistance measured quantitatively by HOMA-IR and waist to hip ratio and waist circumference. Insulin resistance best correlated with waist circumference (*rho* = 0.401) than the other above mentioned anthropometric parameters. This finding differs from the previous work done on obese and overweight subjects in which

Table-1: ATP III clinical identification of the met abolic syndrome.

Risk factor	Defining Level
Aabdominal obesity given as waist circumference	
Men	>102cm
Women	> 88cm
Triglycerides	?1.7 mmol/L
HDL Cholesterol	
Men	<1.04 mmol/L
Women	< 1.30 mmol/L
Blood Pressure	?130/85
Fasting Glucose	? 6.1 mmol/L

Table-2: WHO Clinical Criteria for Metabolic Syndrome

Insulin resistance identified by one of the following:
Type 2 diabetes
Impaired fasting glucose
Impaired glucose tolerance
Plus any two of the following
Antihypertensive medication or blood pressure (≥ 140/90)
Plasma triglycerides ≥ 7.1mmol/L
HDL cholesterol < 0.9mmol/L in men, > 0.85 in women
BMI > 30 Kg/m2 and waist to hip ratio > 0.9 in men, > 0.85 in women
Urinary albumin excretion rate ≥ 20µg/min or albumin to creatine ratio ≥ 3.4 mg/mmol

Table-3: Anthropometric and serum glycemic parameters of the study population

Anthropometric and other parameters	
Age (years)	
BMI (Kg/M^2)	29.01 ± 0.48
Waist circumference (cm)	23.2±0.19
Waist to hip ration	84.68±0.91
Systolic blood pressure (mm of Hg)	111.51±0.008
Diastolic blood pressure (mmof Hg)	60.72±0.73
Serum Glycemic Parameters	
Serum glucose (mmol/L)	4.8±0.041
Serum insuline* (u IU/ML)	13.0 (9.7-27)
Insulin resistance *	206 (1.8-5.5)

Values are expressed as mean ± SEM and median (IQR)*

Values are expressed as mean ± SEM and median* (IQR)

Table-4: Correlation between anthropometric and serum glycemic parameters of the study population

Anthropometric parameters	**Serum Glyceimic Parameters**		
	Fasting serum glucose	**Fasting serum insulin**	**Insulin resistance**
BMI	r=0.292	rho=0.135	rho=0.143
	p= 0.005*	p= 0.20	p= 0.174
Waist to hip ratio	r=0.281	Rho=0.207	rho=0.221
	p=0.007*	p=0.05*	p=0.046*
Waist cricumference	r=0.336	rho=0.25	Ho=0.401
	p=0.002*	p=0.00.23*	p=0.00*

** $p < 0.05$ is considered statistically significant.*

BMI best correlated with HOMA-IR of more than 2.5[9]. BMI best correlated with HOMA-IR of more than 2.5.[9] We also found significant positive correlation of fasting serum glucose with waist circumference, BMI and waist to hip ratio. There was also significant positive correlation of fasting serum insulin with waist circumference (*rho*=0.25). All the three glycemic parameters showed positive correlation with waist circumference. Waist circumference is a recognized anthropometric parameter of central obesity and is more highly correlated with metabolic risk factor

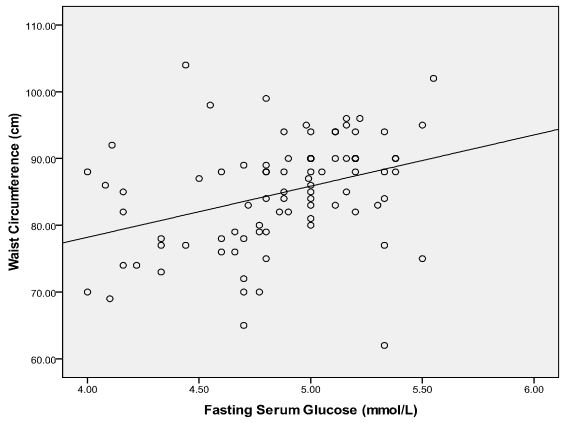

Fig-1: Scatter-plot showing significant correlation (p = 0.002) between serum glucose and waist circumference

Fig-2: Scatter-plot showing significant correlation (p = 0.007) between serum glucose and waist to hip ratio

Fig-3: Scatter-plot showing significant correlation (p = 0.023) between fasting serum insulin and waist circumference

Fig-4: Scatter-plot showing significant correlation (p = 0.000) between insulin resistance and waist circumference

Fig-5: Scatter-plot showing significant correlation (p = 0.046) between insulin resistance and waist to hip ratio

than is an elevated BMI.[10-12] Excessive intake of carbohydrates increases the synthesis of triglycerides by the liver and adipose tissues and raised levels of triglycerides increases central fat. Central fat or visceral fat is responsible for the release of various adipokines (like visfatin, resistin and tumor necrosis factor alpha) which correlate positively with insulin resistance and negatively with insulin sensitivity. These findings are in concordance with the results of the other studies, which also showed waist circumference as the best predictor of insulin resistance.[13-18] Waist to hip ratio also showed positive correlation with fasting serum glucose and insulin resistance. These results are also supported by the WHO and ATPIII criteria of insulin resistance syndrome. In summary, the predominant

fasting serum glucose and insulin resistance were waist circumference and waist to hip ratio (depicting central adiposity)

Conclusion

Our findings suggest that higher anthropometric measurements although within normal range, are associated with insulin resistance in a sample of seemingly healthy, non obese adults. Moreover, abdominal obesity as measured by waist circumference correlates not only with insulin resistance but is also correlated with fasting serum insulin and fasting serum glucose even in healthy subjects. Hence by life style modification (including modification of eating habits and increased physical activity), insulin resistance can be controlled and future threats of insulin resistance like atherosclerosis, hypertension and diabetes can be postponed. In this way, expected dreadful complications due to chronic inflammation induced by insulin resistance may be delayed.

Acknowledgements

We wish to thank Vice Chancellor and the Director Administration, of the University of Health Sciences, Lahore for providing the opportunity and necessary facilities, to carry out this research.

Department of physiology
SIMS/Services Hospital, Lahore
theesculapio@hotmail.com

References

1. Leonardo J, Tamariz J, Hunter Y, James S, Yeh H, Schmidt M, Astor B, Brancati F. Blood viscosity and hematocrit as a risk factor for type 2 diabetes mellitus. The atherosclerosis risk in communities (ARIC) study. Am J Epidemiol 2008;168:1153-60
2. Diagnosis and classification of diabetes mellitus. American Diabetes Association. Diabetes Care 2008; 31: 55-60
3. Buse JB, Polonsky KS, Burant CF. Type 2 Diabetes mellitus. In: Larsen PR, Krokenberg HM, Melmed S, Polonsky KS. Williams Text Book of Endocrinology. Philadelphia: Saunders; 2003: 1427-42
4. Beilby J. Definition of metabolic syndrome: Report of the national heart, lung and blood institute/American heart association conference on scientific issues related to definition. Circulation 2004; 109: 433-8
5. Mathews DR, Hosker JP, Turner RC. Homeostasis model assessment. Insulin resistance and beta cell dysfunction from fasting plasma glucose and insulin concentration in man. Diabeto- logia 1985; 28: 419-21
6. Hashimoto N, Suzuki Y, Haruki AT, Sakuma Y, Iwai R, Takahashi H. The association between the ferritin concentration and the serum insulin resistance markers, and follow up analysis for three years in non diabetic Japanese men. Diabetes Care 2008; 31: 55-60.
7. Braham D, Trinder P. An improved colour reagent for the determination of blood glucose by the oxidase system. Analyst 1972; 40: 1232-7
8. Clark PM. Assay for insulin, proinsulin and C peptide. Ann Clin Biochem 1999; 36: 541-64
9. Gomez-Garcia A, Nieto Alcanter E, Gomez-Alonso C, Figuero-Nunez B, Alvarez-Augelar C. Anthropometric parameters as predictors of insulin resistance in overweight and obese adults. Aten Primaria 2010;42:364-71
10. Henderson DC, Fan X, Sharma B, Copeland PM, Borbac P, Freudenreicho O, Cather C, Goff DC. Waist circumference is the best anthropometric predictor in non diabetic patients With schizophrenia treated with clozapine but not olanzapine. J Psychiatr Pract 2009;15:251-67
11. Bosy-Westphal A, Geisler C, Onur S, Korth O, Selberg O, Schrezenmer J. Value of body fat mass vs anthropometric obesity indices in the assessment of metabolic risk factors. Int J Obes 2006;30: 475-83
12. Allemand-Jander D. Clinical diagnosis of metabolic and cardiovascular risks in overweight children: early development of chronic diseases in the obese child. Int J Obes 2010;34: 32-6
13. Misra A, Khurana L. The metabolic syndrome in South Asians: epidemiology, determinants, and prevention. Metab Syndr Relat Disorder 2009;7: 497-514
14. Moebus S, Hanisch JU, Aidelsburger P, Bramlage P, Wasem J, Joeckel KH. Impact of 4 different definitions used for the assessment of the prevalence of the metabolic syndrome in primary healthcare: the German metabolic and cardiovascular risk project. Cardiovasc Diabetol 2007;6:22- 6.
15. Łopatyński J, Mardarowicz G, Szcześniak G. A comparative evaluation of waist circumference, waist-to-hip ratio, waist-to-height ratio and body mass index as indicators of impaired glucose tolerance and as risk factors for type-2 diabetes mellitus. Ann Univ Mariae Curie Sklodowska [Med] 2003; 58:413-9.
16. Waris JS, Misra A, Vikram NK, Pandey RM, Gupta R.

Comparison of definitions of the metabolic syndrome in adult Asian Indians. J Assoc Physicians India 2008;56: 158-64
17. Khan FA, Khan SH, Ijaz A, Sattar A, Dilawar M. Common Anthropometric Indices and Insulin Resistance. Pak J Med Res 2009; 4:433-8
18. Fukuchi S, Hamaguchi K, Seiki M, Himeno K, Sakata T, Yoshimatsu H. Role of fatty acid composition in the development of metabolic disorders in sucrose induced obese rats. Expel Biol Med 2004;6:486-93