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EVALUATION OF THE EFFECTS OF INSULIN RESISTANCE ON OBESITY AND RELATED PARAMETERS

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Abstract: Obesity, especially abdominal obesity, is one of the most important underlying risk factors for metabolic syndrome and increases the risk of developing various pathological conditions like Type 2 diabetes mellitus, dyslipidaemia, hypertension and non alcoholic fatty liver disease. Insulin resistance means that the ability of insulin to stimulate glucose utilization is reduced, it is an integral feature of the metabolic syndrome and an important predictor of Type 2 Diabetes Mellitus development. It is well known that obesity is associated with diabetes mellitus, and the main basis of this link is thought to be the ability of obesity to stimulate insulin resistance. In this study, we aimed to evaluate the relationship between insulin resistance and obesity, related biochemical parameters, body analysis data. In this retrospective case control study, the insulin resistance status of 120 volunteers was determined by calculating the Homeostatic Model Assessment-Insulin Resistance formula. Biochemical values and body analysis results of individuals with and without insulin resistance were compared. Insulin resistance was detected in 56 (%46.7) of the subjects participating in the study. Insulin resistance was significantly related with weight, body mass index, body fat mass (p < 0.05). However, there was no correlation between insulin resistance and body fat percentage, abdominal fat percentage and abdominal fat mass (p>0.05). Fasting plasma glucose, insulin, triglyceride and alanine aminotransferase levels were found to be higher in individuals with insulin resistance (p<0.05). There was no significant relationship between insulin resistance and HbA1c percentages, cholesterol, AST and Vitamin B12 levels (p>0.05). In conclusion, considering the risks of diseases caused by insulin resistance, findings of this study emphasizes the importance of detecting insulin resistance. Determining the presence of insulin resistance can help prevent a variety of diseases by regulating nutrition. Keywords: insulin resistance, obesity



1. Introduction

It is known that in recent years, the worldwide prevalence of obesity and metabolic complications have increased significantly. World Health Organization data show that in 2016, 1.9 billion adults over the age of 18 are overweight; of which 650 million are obese [1]. Obesity, especially abdominal obesity, is one of the underlying risk factors for the metabolic syndrome (MetS) [2]. Obesity increases the risk of developing various pathological conditions such as insulin resistance, type 2 diabetes mellitus (DM), dyslipidaemia, hypertension and non alcoholic fatty liver disease (NAFLD). It is also associated with metabolic changes such as increased fat mass, hypertension, elevated triglyceride and blood glucose levels and low HDL-cholesterol levels [3].

Insulin resistance means that the ability of insulin to stimulate glucose utilization is reduced. While pancreatic β -cells increase insulin production and secretion as a compensatory mechanism (hyperinsulinemia), glucose tolerance remains normal [4]. At the same time, insulin resistance is an integral feature of the metabolic syndrome and is an important predictor of DM development [5]. It is well known that obesity is associated with DM, and the main basis of this link is the ability of obesity to stimulate insulin resistance [6].

It is known that insulin resistance is associated with many diseases like MetS, DM, and obesity and is an important factor that triggers many other metabolic changes. Within this information, we aimed to evaluate the association of insulin resistance with obesity and other possible metabolic abnormalities in adult subjects.

2. Material and Method

Study was planned as a retrospective case control study and 120 volunteers, who were 18 years old and did not have bariatric surgery, who applied to the Nutrition and Diet Policlinic of Samsun Buyuk Anadolu Hospital between 01.11.2016-01.04.2017 were included. At the beginning of the study, approval was obtained from Ondokuz Mayıs University Clinical Research Ethics Committee with approval number B.30.2.ODM.0.20.02/1432.

Body analysis data that was evaluated in the study were; weight, body mass index (BMI), body fat mass, body fat percentage, abdominal fat mass, abdominal fat percentage. BMI was calculated by dividing weight to height's square. According to World Health Organization (WHO) criteria, obesity was defined by BMI \geq 30 kg/m² and excessive weight defined by BMI \leq 30 kg/m² BMI \geq 25 kg/m² [7]. Body composition was obtained from bioelectrical impedance analysis (TARTI, TANITA BC-418) results. Clinical and metabolic data include fasting blood glucose (FBG), HbA1C, insulin, triglyceride, cholesterol, alanine aminotransferase (ALT), aspartate aminotransferase (AST) and Vitamin B12. Insulin resistance was defined by having HOMA-IR index over 2.5 which was calculated by the formula of fasting insulin (μ U/ml) × fasting glucose (mg/dL) / 405 [8].

The data were analyzed using the 24.0 version of the Statistical Package for Social Sciences (SPSS) package program. After determining the number of people who applied to the Nutrition and Diet Polyclinic between the given dates, the sample size was calculated as 120 with %95 power and %5 error margin. The results are presented as the mean; unpaired Student-t test and chi-



square test were used to compare the continuous and categorical variables in insulin resistant and non-insulin subjects. Pearson correlation was used for correlation analysis. A value of p<0.05 was considered statistically significant.

3. Results

Table 1 summarizes the main characteristics of the individuals evaluated in the study.

	Minimum	Maximum	Mean ± SD
Age	18	78	38.7 ± 12.7
Weight (kg)	43.3	159.6	87.4 ± 19.4
Height (m)	1.4	1.8	$1.64 \pm .09$
BMI (kg/m ²)	17.6	63.9	32.6 ± 7.7
Body Fat Percentage(%)	13.7	59.5	35.2 ± 9.4
Body Fat Mass (kg)	7.1	91.8	31.8 ± 13.5
Abdominal Fat Percentage (%)	6.7	61.3	33.4 ±8.6
Abdominal Fat Mass (kg)	1.5	34.1	15.9 ± 6.1

Table 1. Body analysis data of individuals participating in the study

82 women (%68.3) and 38 men (%31.7) individuals aged between 18 and 78 years (38.7 \pm 12.7) were evaluated in the study. Mean BMI values were found in the obese range (32.6 kg/m²). When assessed in detail, it was determined that 16 individuals (13.3) were normal weight (BMI \leq 24.99 kg/m²), 32 (%26.7) were overweight (BMI 25-29.99 kg/m²) and 72 (%60) were obese (BMI \geq 30 kg/m²).

The data on the biochemical parameters of the individuals evaluated in the study are summarized in Table 2.

Table 2. Conclusions on biochemical parameters of individuals participating in the study

	Unit	Reference Range	Mean ± SD	
Fasting Blood Glucose	mg/dL	74-109	107.2 ± 22.2	
Insulin	µU/mL	2.6-24.9	11.1 ± 7.0	
Tryglycerides	mg/dL	0-200	138.4 ± 97.1	
Cholesterol	mg/dL	0-200	186.2 ± 58.1	
AST	U/L	0-32	21.5 ± 13.1	
ALT	U/L	0-33	25.2 ± 18.3	
Vitamin B12	pg/mL	191-663	368.4 ± 190.7	



Insulin resistance was detected in 56 (%46.7) of the subjects participating in the study. Thirtyfive of these individuals were female (%62.5) and 21 were male (%37.5). When evaluated in detail, it is seen that all of the biochemical findings are among the reference values.

In Table 3, evaluation of body-analysis data of individuals with and without insulin resistance is given.

	Insulin Resistance (+)	Non-Insulin	p-value
	Group (HOMA-	Resistance Group	
	IR>2.5, n=56)	(HOMA-IR<2.5,	
		n=56)	
Age	37.8 ± 14.6	39.5 ± 10.7	.380
Weight (kg)	93.5 ± 6.7	82.0 ± 9.9	.003*
Height (m)	1.6 ± 0.1	1.6 ± 0.0	.458
BMI (kg/m ²)	34.4 ± 7.0	31.0 ± 8.0	.002*
Body Fat Percentage(%)	36.3 ± 8.2	34.3 ± 10.4	,263
Body Fat Mass (kg)	34.4 ± 12.9	29.4 ± 13.7	.017*
Abdominal Fat Percentage (%)	34.6 ± 6.7	32.3 ± 9.9	.144
Abdominal Fat Mass (kg)	17.1 ± 5.6	14.9 ± 6.4	.055

Table 3. Evaluation of individual's insulin resistance status and body analysis values

*p <0.05

Body weight and body fat mass averages were found significantly higher in those with insulin resistance. Individuals with insulin resistance were found to have a higher BMI (p<0.05). No significant correlation was found between insulin resistance and body fat percentage, abdominal fat mass and abdominal fat percentage (p>0.05).

Table 4 provides an assessment of the biochemical characteristics of individuals with and without insulin resistance.

Table 4. Evaluation of individual's insulin resistance status and biochemical properties

	Unit	Insulin	Insulin	p-value
		Resistance (+)	Resistance (-)	
		(n=56)	(n=64)	
Fasting Blood Glucose	mg/dL	113.1	102.1	.039*
HbA1c	%	6.0	5.7	.259
İnsulin	µU/mL	16.6	6.2	.001*
Triglycerides	mg/dL	163.5	116.5	.039*
Cholesterol	mg/dL	191.5	181.6	.367



AST	U/L	23.6	19.6	.584
ALT	U/L	30.7	20.4	.026*
Vitamin B12	pg/mL	377.8	360.1	.837

*p <0.05

When biochemical findings were examined, fasting plasma glucose, HbA1c, insulin, triglyceride and ALT were significantly higher in insulin resistance group (p<0.05). There was no significant difference between the two groups in terms of cholesterol, AST, B12 vitamin (p>0.05).

4. Discussion

It is emphasized that patients diagnosed with insulin resistance have a higher propensity risk of development of MetS, DM and cardiovascular diseases. In the light of this information, considering the health effects of insulin resistance, the aim of this study was to evaluate the relationship between insulin resistance and obesity, related biochemical parameters and body analysis data.

The main cause of insulin resistance, which plays an important role in the pathogenesis of DM, is obesity [9]. World Health Organization data reveals that %66.8 of Turkey's population is overweight or obese[1]. The prevalence of insulin resistance in Turkey was reported to be %26.2 [10]. Bilge et al found that the incidence of insulin resistance in the obese population was significantly higher than in the non-obese population, which is consistent with our results [11]. A study on women with polycystic ovarian syndrome suggests that BMI is higher in women with insulin resistance [12]. The relationship between adiposity and insulin resistance is also shown in a study by Carmina et al. [13]. In a study with adolescents and adults, the HOMA-IR score correlated positively with BMI, total fat mass, and abdominal fat mass [14]. While %60 of the participants in our study were obese, insulin resistance was detected in %56.9 of these individuals. Insulin resistance was associated with BMI, body fat mass and abdominal fat mass (p<0.05).

Intracellular lipid metabolism in pancreatic beta cells is involved in regulation of insulin secretion. Changes in intracellular lipid homeostasis may account for insulin resistance-related conditions [15]. In a study where participants were divided into seven groups according to their triglyceride, HDL-cholesterol levels, the HOMA-IR value was found to be higher in groups with high triglyceride levels. [16]. In a study of healthy adults, it has been shown that insulin resistance is associated with hypertriglyceridemia [17]. However, the results of studies on triglyceride levels and insulin resistance are contradictory. Our study results show that triglyceride levels are associated with insulin resistance (p<0.05).

ALT level; independent of the direct criteria of adiposity, insulin sensitivity and secretion, may be effective in predicting future diabetes (prediabetes) [18]. In a study by Maximos et al., NAFLD patients with elevated ALT levels have been shown to have elevated serum insulin levels [19]. Moreover, in another study with young adults, ALT levels were associated with insulin resistance [20]. The results obtained according to our study showed that ALT is associated with insulin resistance (p<0.05) and is consistent with the literature.

The results of studies investigating vitamin B12 and insulin resistance vary. In a study of adolescents showing prediabetic and insulin resistance characteristics, it was reported that one third of participants had low or borderline B12 Vitamin levels [21]. In a study conducted in women with polycystic over syndrome, low B12 Vitamin levels have been shown to be associated with insulin



resistance [22]. In a study conducted by Baltaci et al., It was demonstrated that B12 Vitamin levels were not associated with insulin resistance [23]. The results of our study showed that vitamin B12 levels were not associated with insulin resistance (p>0.05)

5. Conclusions

Lifestyle changes (such as healthy diet, weight loss, exercise) are the main treatment modality for treating both insulin resistance and obesity. Medical treatment options may be considered when lifestyle modification is not available. The fact that insulin resistance is seen at all ages in our study is an important result when considering the risks of disease and emphasizes the importance of detecting insulin resistance. Determining the presence of insulin resistance can help prevent a variety of diseases by regulating nutrition. In connection with the subject, studies conducted to establish relationships with wider populations and change patient groups can be suggested.

References

- [1] Obesity and Overweight, World Health Organization, http://www.who.int/ mediacentre/factsheets/fs311/en/
- [2] Carr, D. B. Et al., Intra-abdominal fat is a major determinant of the National Cholesterol Education Program Adult Treatment Panel III criteria for the metabolic syndrome. *Diabetes*, 53(8), , pp. 2087-2094, 2004.
- [3] Weiss, R. et al., Obesity and the metabolic syndrome in children and adolescents, *N Engl J Med*, *350(23)*, pp. 2362-2374, 2004.
- [4] Oliveira, C.L. et al., Obesity and metabolic syndrome in infancy and adolescence. *Rev Nutr*, *17*(2), pp. 237-245, 2004.
- [5] Lillioja, S. et al., Insulin resistance and insulin secretory dysfunction as precursors of noninsulin-dependent diabetes mellitus, *N. Engl. J. Med*, 329(27), pp. 1988–1992, 1993.
- [6] Kahn, B.B., Flier, J.S., Obesity and insulin resistance, J. Clin. Investig, 106(4), 473–481, 2000.
- [7] WHO Technical Counsultation, Obesity: preventing and managing the global epidemic. Report of a WHO consultation, Report No: 0512- 3054, 2000.
- [8] Matthews, D.R. et al., Homeostasis model assessment: insulin resistance and beta-cell function from fasting plasma glucose and insulin concentrations in man, *Diabetologia*, 28(7), pp. 412– 419, 1985.
- [9] Gupta, D. et al., Over-nutrition, obesity and insulin resistance in the development of β-cell dysfunction, *Curr Diabetes Rev*, 8(2), pp. 76-83, 2012.
- [10]Kaya, A. et al., The prevalence of insulin resistance in the Turkish population, Endocrine *Abstracts*, *41*, (2016).
- [11]Bilge, U. et al., Obesity and Insulin Resistance Rates in a Population Under 40 Years Old Who Admitted to a Family Medicine Outpatient Clinic, *Turk Aile Hek Derg*, 20(2), pp. 50-53, 2016.
- [12]Tosi, F. et al., Total body fat and central fat mass independently predict insulin resistance but not hyperandrogenemia in women with polycystic ovary syndrome, *J Clin Endocrinol Metab*, 100(2), pp. 661-669, 2015



- [13]Carmina, E., Abdominal fat quantity and distribution in women with polycystic ovary syndrome and extent of its relation to insulin resistance, *J Clin Endocrinol Metab*, 92(7), pp. 2500-2505, 2007.
- [14]Linder, K. et al., Relationships of body composition and liver fat content with insulin resistance in obesity-matched adolescents and adults, *Obesity (Silver Spring)*, 22(5), pp. 1325-1331, 2014.
- [15]Boden, G., Shulman, GI., Free fatty acids in obesity and type 2 diabetes: defining their role in the development of insulin resistance and beta cell dysfunction, *Eur J Clin Invest*, 32(3), pp. 14-23, 2002.
- [16]Bao, Y. et al., Dyslipidaemia and insulin resistance in Chinese in Shanghai. *Zhonghua nei ke za zhi*, 40(5), pp. 299-302, 2001.
- [17]Simental-Mendía, L.E. Et al., Insulin secretion is increased in non-diabetic subjects with fasting hypertriglyceridaemia. *Diabetes/metabolism research and reviews*, 29(3), pp. 214-219, 2013.
- [18]Vozarova, B. et al., High alanine aminotransferase is associated with decreased hepatic insülin sensitivity and predicts the development of type 2 diabetes, *Diabetes*, *51*(6), pp. 1889-1895, 2002.
- [19]Maximos, M. et al., The role of liver fat and insulin resistance as determinants of plasma aminotransferase elevation in nonalcoholic fatty liver disease, *Hepatology*, 61(1), pp. 153-160, 2015.
- [20]Simental-Mendia, L.E. et al., Insulin resistance is associated with elevated transaminases and low aspartate aminotransferase/alanine aminotransferase ratio in young adul ts with normal weight, *Eur J Gastroenterol Hepatol*, *29*(*4*), pp. 435-440, 2017.
- [21]Ho, M. et al., Vitamin B12 in obese adolescents with clinical features of insulin resistance, *Nutrients*, 6(12), pp. 5611-5618, 2014.
- [22]Kaya, C. et al., Obesity and insulin resistance associated with lower plasma vitamin B12 in PCOS, *Reprod Biomed Online*, *19*(*5*), pp. 721-726, 2009.
- [23]Baltaci, D et al., Association of vitamin B12 with obesity, overweight, insulin resistance and metabolic syndrome, and body fat composition; primary care-based study, *Med Glas (Zenica)*, 10(2), pp. 203-210, 2013.