Bitlis Eren University
Journal of Science and Technology

ISSN 2146-7706
2, (2012), 19-21

Research Article

The evaluation of obesity through blood parametres

Betül Battaloğlu İnanç

Mardin Artuklu University, Health High School, Deparment of Midwifery, Mardin - Turkey
✱ *Corresponding author: Tel :+90 482 212 13 95 Fax :+90 482 212 69 47, e-mail: betulbattaloglu00@gmail.com*

Abstract

Obesity and dyslipidemia are increasing problems in many countries and related the multiple cardiovascular risk factors. The purpose of this study was to determine the association between some simple anthropometric measurements and lipid parameters. In all patients, age, gender, weight, and height were recorded. Body mass index (BMI) calculated. Plasma leptin, total cholesterol (TC), triglyceride (TG), high density lipoprotein (HDL), low density lipoprotein (LDL), very low density lipoprotein (VLDL), impairment fasting glucose (IFG) and CRP (C-Reactive Protein Test) were recorded and compared. There was a significant correlation in adult obese patient group between BMI, TC, VLDL ($p<0.001$); IFG, LDL, TG, and serum leptin ($p<0.05$) level. CRP and HDL were higher but not statistical significant.

Keywords: CRP, glucose, lipid profile, obesity

1. Introduction

Obesity is the most important public health problem in the 21st century (WHO 2011). With additional diseases and social problems imposed on the person, this disease is chronic, progressive, and regarded as a disease of high mortality and morbidity (Racette et al. 2003; Sarlio-Lahteenkorva et al. 1999). For adult American society, the rate of the ones whose BMI ≥ 22.3% is 30 kg/m^2 (Kuczmarski et al. 1997). According to TEKHARF, study in Turkey in 2000, it is estimated that 2.63 million men and 5.46 million women (in total 8.1 million) are obese (Onat 2003). This shows that the number of the obese over 20 years old is rising to 23.3% of the population. In a wide range of epidemiological studies carried out for obesity, BMI was used as a criterion (Calle et al. 1999). BMI is an index calculated by dividing the weight (kg) by the square of the height (m) according to the World Health Organization (WHO 2007). Generally lots of risk factors heaped up with obesity. The conventional risk factors such as hypertension, hyperlipidemia, hyperglycemia often come out with obesity has led to a questioning of whether there is an independent risk factor (Seidell et al. 1996). However, some forward-looking long-term observational studies show that obesity is an independent risk factor in older women and men for coronary and cardiovascular mortality (Hubert et al. 1983; Jousilahti et al. 1996).

2. Material and Method

Patients were chosen among 120 people who were admitted to Mardin State Hospital between March and September 2010 for the study as a cross-sectional observation. 30 women and men with BMI<30 kg/m^2 as the control group, 30 women and men with BMI≥30 kg/m^2 as patients' group were selected. All the individuals' height, weight, and waist circumference were measured on the basis of a standardized protocol. Obese groups weren't receiving antihyperlipidemic therapy. Fasting venous blood glucose, lipid profiles, leptin, and C-reactive protein were measured in the automatic analyzer (Aeroset, Abbott, DRG Instruments Gmbh). Blood was drawn for leptin in the tubes with EDTA. The blood samples were separated into plasmas by centrifugation with 3000 rpm for 10 minutes (Hettich Lab. Technology, Tuttlingen). The study was initiated after the approval of the ethics committee. All individuals were informed about the study and consent forms were given and completed.

2.1. Statistical Analysis

All analysis was carried out by using the SPSS package. Data were expressed as mean standard deviation or percentage. Categorical data were examined using chi-square test. The relationship among BMI, leptin and the other parameters was evaluated by Pearson's correlation test. For all tests, the "p" value $p<0.05$ was considered as statistically significant.

3. Results and Discussion

The overall clinical, anthropometric, and biochemical data that belong to each of the two groups are shown in Table 1. Gender, age, height, and BMI values were similar in both groups. The average age of the patient group was 45.17±9.86 (min: 19, max: 59), the average age of the control group was 41.10±11.06 in the range (min: 20, max: 59). The average age of the patient group was 47.23±9.07 for women and 43.10±10.34 for men; the average age of the control group was 37.07±10.59 for women and 45.13±10.16 for men. The BMI average of the patient group was 34.92±4.86 in women; BMI average of the patient group was 34.67±2.88 in men. The BMI average of the control group in women was 23.59±3.83; the average BMI of the control group in men was 25.49±3.00. The fasting blood glucose average of the patient group was 139.90±81.88 in women; the fasting blood glucose average of the patient group was 166.90±

Received 12 January 2012
Accepted 17 May 2012
Available online June 2012

96.31 in men; the average fasting blood glucose of control group was 91.00±18.09 in women. The average fasting blood glucose of control group was 92.67±23.80 in men (Table 1).

Table 1. Patient and control group values comparison

	Mean ± SD
Patients' age group	45.17±9.86
Control age group	41.10±11.06
Patients' group women age	47.23±9.07
Patients' group men age	43.10±10.34
Control group women age	37.07±10.59
Control group men age	45.13±10.16
Patients' group women BMI	34.92±4.86
Patients' group men BMI	34.67±2.88
Control group women BMI	23.59±3.83
Control group men BMI	25.49±3.00
Patients' group women IFG	139.90±81.88
Patients' group men IFG	166.90±96.31
Control group women IFG	91.00±18.09
Control group men IFG	92.67±23.80

For adult obese patients group, BMI total cholesterol, VLDL ($p<0.001$), fasting blood glucose, low-density lipoprotein (LDL)-cholesterol, triglycerides, and serum leptin showed difference in significant levels ($p<0.05$). CRP and HDL levels are detected as high, but not statistically significant (Table 2).

Table 2. Control and patients' group values comparison with each others

	GROUP	N	Mean	SD	t-value
BMI	Control	60	24.5456	3.54476	14.928**
	Patient	60	34.8005	3.96856	
IFG	Control	60	91.83	20.980	5.178**
	Patient	60	153.40	89.671	
TCOLL	Control	60	123.97	27.353	7.045***
	Patient	60	171.35	44.343	
HDL	Control	60	43.68	14.962	0.936
	Patient	60	41.02	16.210	
LDL	Control	60	90.32	28.172	7.567**
	Patient	60	143.75	46.885	
VLDL	Control	60	23.49	15.396	7.325***
	Patient	60	42.52	12.958	
TG	Control	60	137.52	65.313	3.909**
	Patient	60	187.03	73.232	
CRP	Control	60	5.8350	10.91680	0.338
	Patient	60	6.3300	3.02225	
S_LEPTIN	Control	60	14.4938	9.26924	16.039**
	Patient	60	62.4977	21.24935	

*:p<0.05, **: p<0.01, ***: p<0.001

A strong degree of significance was detected between BMI and fasting blood glucose, total cholesterol, LDL, VLDL, TG, and serum leptin ($p<0.001$). No relationship was detected between HDL and CRP (Table 3). A large number of dyslipidemia illnesses that predispose to the development of coronary heart disease are accompanied by obesity. These include hypercholesterolemia, fasting and postprandial triglyceride levels, low HDL cholesterol, and high apolipoprotein B levels. Every 10% increase in weight increases 10-15 mg/dL in blood cholesterol. When compared to normal people, it is known that hypercholesterolemia risks in obese individuals at the age of 20-75 is 1.5 times, and in the ones at the age of 20-45 is 2 times higher (Onat 2003).

In the studies, the dyslipidemia, total cholesterol and triglycerides and LDL increase that is seen in obesity has been defined as decrease in HDL-C (Abbasi et al. 2002). In the presence of low HDL and the increase in apolipoprotein B/apolipoprotein A1 ratio, the risk of cardiovascular disease in obesity can be foreseen. In our study, statistical significance was found between BMI, total cholesterol, TG, LDL, VLDL, and serum leptin. HDL showed a negative correlation and it was found higher in the control group but not statistically significant. About the height of LDL that is mostly used in determining the risk of cardiovascular disease, there is less evidence for the role of obesity (Yusuf et al. 2004).

In our study, a significant correlation has been found with increasing BMI. The cardiovascular disease relationship in hypercholesterolemia and obesity is not clear. However, hypercholesterolemia is associated with insulin resistance have been reported to increase the risk of disease (Gaudet et al. 1998). In both diabetic and non-diabetic obese people, a strong relationship has been demonstrated between obesity and insulin resistance (Ludvic et al. 1995). Carey and his colleagues express that when BMI rises from twenty to thirty, the risk of diabetes increased 11 times (Carey et al. 1997). Insulin suppresses free fatty acids in plasma. Therefore, when insulin resistance develops, free fatty acids in plasma increases and this increases triglyceride production. Lipoproteins that increase in plasma and rich in triglyceride, suppress HDL level (Reaven et al. 1988).

In our study, the rise in fasting blood glucose and total cholesterol showed a positive and highly significant association. That obesity has played a key role in the formation of coronary heart disease is becoming increasingly clearer, and it is thought to pose a major risk factor. Among the risk factors for coronary heart disease, hypertension and HDL decrease after smoking cigarettes comes before the LDL lowness and diabetes (Onat 2003).

The present data shows that the secretion of leptin that increased in adipose tissue of obese people, markedly increased while fat mass is increasing (Bilgili et al. 2008). In another studies, it has been reported that

Table 3. Pearson's correlation test

	AGE	BMI	IFG	TCOLL	HDL	LDL	VLDL	TG	CRP	LEPTIN
AGE	1	0.227*	0.250**	0.091	0.021	0.175	0.212*	0.173	-0.084	0.168
BMI	0.227*	1	0.329**	0.577**	-0.127	0.531**	0.612**	0.441**	-0.023	0.664**
IFG	0.250**	0.329**	1	0.290**	0.039	0.217*	0.233*	0.223*	-0.006	0.434**
TCOLL	0.091	0.577**	0.290**	1	0.006	0.360**	0.488**	0.445**	-0.022	0.517**
HDL	0.021	-0.127	0.039	0.006	1	0.08	-0.195*	-0.132	-0.086	-0.051
LDL	0.175	0.531**	0.217*	0.360**	0.08	1	0.356**	0.268**	0.005	0.563**
VLDL	0.212*	0.612**	0.233*	0.488**	-0.195*	0.356**	1	0.573**	0.018	0.429**
TG	0.173	0.441**	0.223*	0.445**	-0.132	0.268**	0.573**	1	-0.012	0.279**
CRP	-0.084	-0.023	-0.006	-0.022	-0.086	0.005	0.018	-0.012	1	0.039
LEPTIN	0.168	0.664**	0.434**	0.517**	-0.051	0.563**	0.429**	0.279**	0.039	1

*:p<0.05, **: p<0.01

leptin levels also decreased with the decrease in body weight (Considine et al. 1996). In this study, similarly, it was found that leptin level is significantly higher in the group whose BMI is high. A substantial portion of blood levels of interleukin-6 that represents the major determinant of acute-phase response is believed to stem from adipose tissue. 1/5 of variability in interleukin levels and CRP protein in blood could be explained by anthropometric measurements about obesity and its distribution has been informed based on a kind of population work (Onat 2003). However, in our study, a correlation between BMI and CRP haven't been detected. Obesity is a disease that needs to be taken seriously because of its associated conditions and necessary precautions must be taken against it in order to protect the health and well-being of the population. People must be taught to be aware of this disease and its consequences through the press and in schools.

4. Conclusion

Obesity is rising in Turkey, as it is in much of the world. In terms of preventive medicine, who have findings of truncal fatness and Type II diabetes and/or lipid profiles disorders and their families must be monitored more closely. Towards these purposes, educational seminars about the evaluation of childhood and adult obesity should be arranged for pediatric physicians and general practitioners.

Acknowledgment

We would like to gratefully thank all the patients who participated in this study. No external funding sources were used for this study.

Author contributed significantly in all phases of the study. Author read and approved the final manuscript. The author declare that they have no conflict of interest.

References

Abbasi F, Brown BW, Lamendola C, McLaughlin T, Reaven GM (2002). Relationship between obesity, insulin resistance, and coronary heart disease risk. J Am Coll Cardiol 40, 937-43.

Bilgili S, Celebiler AC, Doğan A, Karaca B (2008). Inverse relationship between adiponectin and plasminogen activator inhibitor-1 in metabolic syndrome patients. Endocr Regul 42, 63-68.

Calle EE, Thun MJ, Petrelli JM, Rodriguez C, Heath CWJr (1999). Body-mass index and mortality in a prospective cohort of US adults. New Engl J Med 341, 1097-1105.

Carey VJ, Walters EE, Colditz GA, Solomon CG, Willet WC, Rosner BA, Speizer FE, Manson JE (1997). Body fat distribution and risk noninsulin-dependent diabetes mellitus in women. The Nurses' Health Study. Am J Epidemiol 145, 614-619.

Considine RV, Sinha MK, Heiman ML, Kriauciunas A, Stephens TW, Ohannesian JP, Marco CC, McKee LJ, Bauer TL (1996). Serum immunoreactive-leptin concentrations in normal-weight and obese humans. New Engl J Med 334, 292-295.

Gaudet D, Vohl MC, Perron P, Tremblay G, Gagn C, Lesiege D, Bergeron J, Moorjani S, Despres JP (1998). Relationships of abdominal obesity and hyper-insulinemi to angiographically assesed coronary artery disease in men with known mutations in the LDL receptor gene. Circulation 97, 871-877.

Hubert HB (1986). The importance of obesity in the development of coronary risk factors and disease: The epidemiologic evidence. Annu Rev Publ Health 7, 493-502.

Jousilahti P, Tuomilehto J, Vartiainene E, Pekkanen J, Puska P (1996). Body weight, cardiovascular risk factors, and coronary mortality. Circulation 93, 1372-1379.

Kuczmarski RJ, Carroll MD, Flegal KM, Troiano RP (1997). Varying body mass index cutoff points to describe overweight prevalence among US adults: NHANES III (1988 to 1994). Obes Res 5, 542-548.

Ludvic B, Nolan JJ, Baloga J, Sacks D, Olefsky J (1995). Effect of obesity on insulin resistance in normal subject and patients with NIDDM. Diabetes 44, 1121-1125.

Onat A (2003). The impact of obesity on cardiovasculer disease in Turkey. Turk Soc Cardiol 31, 279-289.

Racette SB, Deusinger SS, Deusinger RH (2003). Obesity: overview of prevalence, etiology, and treatment. Phys Ther 83, 276-288.

Reaven GM, Chem YDI (1998). Role of insulin in regulation of lipoprotein metabolism in diabetes. Diabetes Metab Rev 4, 639-652.

Sarlio-Lahteenkorva S, Lahelma E (1999). The ascociation of body mass index with social and economic disadvantage in women and men. Int J Epidemiol 28, 445-449.

Seidell JC, Verschuren M, Van Leer E, Kromhout D (1996). Overweight, underweight and mortality. Arch Intern Med 156, 958-963.

WHO Global Database on BMI. (2007). Available from: URL: http://www.who.int/bmi/index.jsp?introPage=intro_3.html.

WHO (2011). Obesity: preventing and managing the global epidemic report on a WHO consultation. Technical Report Series, No: 894, World Health Organization, No: 311.

Yusuf S, Hawken S, Ounpuu S, Dans T, Avezum A, Lanas F, McQueen M, Budaj A, Pais P, Varigos J, Lisheng L, Interheart Study Investigators (2004). Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries (the Interheart study): case-control study. Lancet 364, 937-995.