

Comparison of Fat Distribution Parameters between Individuals with and without Metabolic Syndrome

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Abstract

Objective: The aim of this study was to compare the body fat parameters in individuals with and without metabolic syndrome using an objective method.

Materials and methods: This case-control study was performed in Tehran and consisted of 105 individuals with age range of 20 to 55 years. All participants were overweight and obese. NCEP ATP III criteria were used for identifying subjects with metabolic syndrome. Waist circumference, weight and height were measured by an expert person and with non-elastic tape measure, balanced beam scale and standing stadiometer, respectively. Fat mass was measured using BIA (model: Tanita BC 418).

Results: Mean age of total participants, case group, and control group were 35 ± 14 , 35.85 ± 6.9 and 34.98 ± 8.1 years, respectively. Mean BMI in case group was higher than control group ($P<0.01$). There was no significant difference between case and control groups for total body fat, right hand fat, as well as right and left leg fats. But there was significant difference between the two groups for trunk and left right hand fats, before controlling of confounding factors. After controlling confounding factors this observed difference was disappeared. One unit increase in BMI raised the development odds of metabolic syndrome by 1.14% (95%CI: 1.02-1.27).

Conclusion: We found that BMI is a more accurate predictor of MetS rather than body fat mass. However, other predisposing factors especially at molecular levels are needed to clarify underlying mechanisms of this syndrome.

Keywords: Metabolic syndrome, Body fat distribution, Body mass index

Introduction

For the first time in 1988, a syndrome considered to be insulin resistance, called "Syndrome X" was introduced. This syndrome was called Metabolic Syndrome (MetS) later (1-3). In 1998, World Health Organization proposed criteria for diagnosis of MetS. In 2001, NCEP ATP III

expressed other criteria included five components. Then in 2005, a new global definition of the MetS was provided by International Diabetes Federation (IDF) (1). Despite aforementioned international organizations, other research groups also have presented different definitions (2). Therefore, a

gold standard method has never been established so far (1). Main components of MetS are dyslipidemia, elevated arterial blood pressure, high fasting blood sugar, central obesity, and insulin resistance (2).

MetS is a complex disorder that causes abundant socio-economic cost. MetS is a global epidemic health problem, resulting in increased risk of coronary artery disease, cardiovascular atherosclerotic disease and type 2 diabetes (2). The prevalence of MetS is high and it is increasing rapidly in all western population, probably due to epidemic of obesity (2,4). Prevalence of MetS is high in sub-Saharan African and Middle East countries including Morocco, Oman, Turkey and Iran. It was reported that the prevalence of MetS in Iran is 33.7%. Also, the prevalence of MetS is increasing in other regions of Asia including East Asia and China (4). The prevalence of MetS is increasing not only among adults, but also in children and adolescents (2,4) throughout the world.

Waist circumference (WC) is one of the criteria for diagnosis of MetS (2) and in some cases, it is the core diagnosis criterion (1). But not all individuals with an increased WC show the features of MetS. There are some individuals with lower WC who develop MetS as well. To terminate this discussion, emergency need of identifying amount of body fat mass was proposed (5). Studies have shown that visceral obesity is related to insulin resistance, hyperinsulinemia, dyslipidemia, and hypertension (3,6-9). Although WC is one of the components of MetS, it does not show the amount of body fat mass in different sites of the body (10). There are studies introducing WC as a simple measure of adiposity, especially abdominal obesity which is highly correlated with visceral fat assessed by computed tomography (11,12). Measurement of WC may be biased by holding breath and place of tape measure. Therefore, it would be better to use an objective way to assess body fat mass.

There are several reference methods to estimate body composition including

underwater weighing, air displacement, plethysmography, labeled water techniques, and dual-energy x-ray absorptiometry (DEXA). But these methods are not suitable in studies on large populations. Bioelectrical impedance is the only method with its own benefits such as being inexpensive, noninvasive and quick which has been used in studies with high population (13).

The aim of this study is to compare the body fat parameters in individuals with and without MetS using an objective method. To our knowledge this is the first study that has been ever conducted in Iran and worldwide as well.

Materials and Methods

This case-control study was performed in Tehran and consisted of 105 individuals with age range of 20 to 55 years who had referred to Endocrine Center of Imam Khomeini Hospital from August 2011 to February 2012. All participants were overweight and obese. NCEP ATP III criteria were used for identifying subjects with MetS. MetS was diagnosed when an individual had at least three of the following criteria: central obesity (waist circumference ≥ 102 cm in men and ≥ 88 cm in women), high systolic blood pressure and/or high diastolic blood pressure (blood pressure $\geq 130/85$ mmHg) or taking medication for high blood pressure, fasting blood sugar ≥ 100 mg/dl, serum triglyceride ≥ 150 mg/dl, and HDL cholesterol < 40 mg/dl in men and < 50 mg/dl in women. (14) The control group was consisted of 51 overweight/obese subjects (without MetS) selected from those attending our center for routine medical care matched to cases by age and gender.

Exclusion criteria included pregnant, lactating and menopausal women, athletes, smokers, individuals with uncontrolled thyroid diseases, individuals with special or vegetarian diet, those taking nutritional supplement, fat and blood sugar reduction drugs, sedatives and medications for sleep disorders, and individuals with cancer and renal disease.

Weight was measured to nearest 0.1 kg by balanced beam scale (Seca Corp. Scale,

Germany) with light indoor clothing. Height was measured to nearest 0.5 cm using standard stadiometer. After calculating BMI, individuals were categorized to overweight and obese using WHO cut-off point (1997) (15). WC was measured using non-elastic tape measure, by an expert person. Demographic data (marital status, education level, occupation, and family history of obesity) were asked by a trained interviewer.

Fat mass was measured using BIA (model: Tanita BC 418 body composition analyzer) 2-3 hours after the last meal. Individuals must stand barefooted on the analyzer and hold a pair of handgrips. There is a strong correlation between Tanita measurements and DEXA (16).

Statistical Analysis

Data analysis was performed using SPSS software (Ver. 18). Independent T test was used to compare age, weight, height, BMI and fat distribution parameters. Chi square test was used to compare qualitative confounders. To control confounding factors, ANCOVA and logistic regression were used. $P < 0.05$ was considered significant.

Results

Total number of participants was 105 (66 men and 39 women). There was no significant difference between the two groups for demographic factors (Table 1), after controlling confounding factors.

Mean age of total participants, case group, and control group were 35 ± 14 , 35.85 ± 6.9 and 34.98 ± 8.1 years, respectively. There was no significant difference between the mean age of two groups ($P = 0.5$). The Mean weight in case group was higher than the control group ($P = 0.02$). Mean BMI in the case group was higher than the control group ($P = 0.01$).

There was no significant difference between the case and control groups for total body fat, right hand fat, as well as right and left leg fats. There was significant difference between the groups for trunk and left right hand fats. Since there was significant difference between the groups for BMI, a multivariate analysis was performed and the participants were categorized into obese ($BMI \geq 30 \text{ kg/m}^2$) and overweight ($BMI 25-29.99 \text{ kg/m}^2$) groups. There was significant difference between overweight and obese groups for fat

Table 1. Comparison of demographic factors between the study groups

Variables		Case n (%)	Control n (%)	P-value*
Gender	Male	34 (65.38)	32 (62.74)	0.7
	Female	18 (34.62)	19 (37.26)	
Marital status	Single	11 (21.15)	8 (15.68)	0.5
	Married	41 (78.85)	43 (84.32)	
Education level	School education	9 (17.30)	10 (19.60)	0.3
	Diploma	27 (51.92)	27 (52.94)	
	Bachelor or higher	16 (30.78)	14 (27.46)	
Occupation	housekeeper	12 (23.07)	7 (13.72)	0.7
	Employee	18 (34.61)	19 (37.25)	
	Worker	19 (36.53)	23 (45.09)	
	Others	3 (5.79)	2 (3.94)	
Family history of obesity	Yes	40 (76.92)	38 (74.50)	0.7
	No	12 (23.07)	13 (25.50)	

Chi-squared test*

Table 2. Comparison of age, weight, height and BMI between the study groups

Variables	Case (n=52)	Control (n=53)	Total	P-value*
Age	$35.85 \pm (6.9)$	$34.98 \pm (8.1)$	35.14 ± 7.5	0.5
Weight	$93.85 \pm (13.7)$	$87.26 \pm (15.6)$	$90.56 \pm (14.7)$	0.02
Height	$169.7 \pm (8.8)$	$167.9 \pm (8.3)$	$168.8 \pm (8.6)$	0.2
BMI	$32.5 \pm (4.2)$	$30.6 \pm (3.7)$	$31.61 \pm (4.02)$	0.01

*Independent T test

Data are presented as Mean±SD

distribution parameters. Logistic regression was performed to identify confounding effect of BMI on fat distribution parameters. According to Table 5, in final model, all of the fat distribution parameters were excluded, except for BMI. One unit increase in BMI, raised the development odds of MetS by 1.14% (95%CI: 1.02-1.27).

Discussion

The primary observation of this case control study is that independent of age, gender, marital status, education level, occupation, and family history of obesity, it can be concluded that BMI is a better predictor of MetS rather

than body fat mass. Our study shows that one unit increase in BMI, raises the development odds of MetS by 1.14%. However, the findings of the present study suggest that the combined use of BMI, WC and regional body fat values in statistical model substantially gives a better picture of predisposing factors of MetS. For example, although there was a significant difference between the two study groups for trunk fat before controlling the confounding factor, after controlling confounding factor, the difference did not remained.

In individuals with MetS, non-esterified fatty acid (NEFA) metabolism may be disrupted and contributed to insulin resistance. Although

Table 3. Comparison of body fat distribution parameters between the study groups

Variables	Case (n=52)	Control (n=53)	Total	P-value*
Total Body Fat	28.84±9.8	25.42±8.6		0.06
Trunk Fat	16.29±4.9	14.39±4.6		0.04
Right Hand Fat	1.69±0.8	1.44±0.5		0.07
Left Hand Fat	1.83±0.9	1.53±0.6		0.05
Right Leg Fat	4.69±2.3	4.05±1.6		0.1
Left Leg Fat	4.64±2.2	3.99±1.6		0.09

*Independent T test.

Measuring unit is kg for all the variables.

Data are presented as Mean±SD.

Table 4. Multivariate analysis of fat distribution parameters in the study groups according to BMI

Variable	Overweight	Obese	P-value*	
Case (with MetS)	Total Body Fat (n=13)	22.33±5.3 (n=13)	30.95±10.1 (n=40)	0.0001
	Trunk Fat (n=13)	12.6±2.5 (n=13)	17.48±4.9 (n=40)	0.0001
	Right Hand Fat (n=13)	1.23±0.3 (n=13)	1.8±0.8 (n=40)	0.01
	Left Hand Fat (n=13)	1.31±(0.3) (n=13)	2.005±0.9 (n=40)	0.01
	Right Leg Fat (n=13)	3.6±1.4 (n=13)	5.05±2.4 (n=40)	0.05
	Left Leg Fat (n=13)	3.58±1.4 (n=13)	4.98±2.38 (n=40)	0.051
	Control (without MetS)	Total Body Fat (n=28)	20.87±5.3 (n=28)	30.73±8.4 (n=24)
Trunk Fat (n=28)		11.93±2.5 (n=28)	17.27±5.03 (n=24)	0.0001
Right Hand Fat (n=28)		1.12±0.2 (n=28)	1.81±0.6 (n=24)	0.0001
Left Hand Fat (n=28)		1.18±(0.3) (n=28)	1.93±0.6 (n=24)	0.0001
Right Leg Fat (n=28)		3.35±1.3 (n=28)	4.86±1.7 (n=24)	0.001
Left Leg Fat (n=28)		3.27±1.2 (n=28)	4.82±1.7 (n=24)	0.0001

*ANCOVA

Measuring unit is kg for all the variables.

Data are presented as Mean±SD.

Table 5. Logistic regression analysis for identifying confounding effect of BMI*

Variable	$\alpha \pm SE$	$\beta \pm SE$	OR	P-value	Confidence interval	
					Higher	Lower
BMI	-4.1 \pm 0.05	-0.13 \pm 0.05	1.14	0.02	1.27	1.02

*Forward stepwise

*Binary logistic

there is a correlation between visceral fat accumulation and portal delivery of non-esterified fatty acids in human, majority of portal NEFAs originate from the systemic circulation. Adipose tissue acts as an endocrine organ and secretes cytokines including interleukin-6 (IL-6) and tumor necrosis factor- α (TNF- α). Evidences have shown that in obesity, macrophage penetrates the adipose tissue and it is involved in inflammation. In obese individuals, C-reactive protein (CRP) marker is also high but adiponectin levels are low. Adiponectin has been shown to improve insulin signaling and has potential protection for atherosclerosis (5).

Contrary to our study, several studies such as You et al (17) and Brochu et al (18) studies, have shown that the amount of visceral adipose tissue and subcutaneous fat is higher in individuals with MetS. These two cross-sectional studies were performed on postmenopausal women. Koster et al concluded that body fat distribution is an important factor in development of MetS in obese elderly, despite having the same amount of fat mass (19). Their result is not consistent with the present study, probably due to the age difference of the participants.

Although high BMI in young adults is associated with metabolic disorder including cardiovascular disease (CVD) (20), evidence indicates that the objective methods are better predictor for metabolic disorders than BMI (21). It seems that the results of studies have been inconsistent. Some studies showed that body fat is more strongly related to the risk of MetS than BMI (22-24); but Kim et al prospective cohort study showed that BMI is a strong predictor for fatal coronary heart disease in both men and women (25). Freedman et al study also showed that BMI is strongly related to levels of lipid profile

parameters, fasting insulin, and blood pressure among young and middle-aged individuals (26).

Although same as body fat mass, BMI might be a predictor for metabolic disorders, but it is not a good one. The U-shape relationship between BMI and body fat mass can be affected by age and gender (26,27). Finally, it is suggested to use both body fat mass and BMI together, rather than using BMI alone, to assess metabolic disorders (28).

Our study had several limitations including small sample size, not using more accurate method for measuring body fat mass and not matching the groups in terms of weight and BMI. The strengths of this study were performing the whole study procedures by a trained person. Secondly, to our knowledge, this is the first study that compares the total and regional body composition using objective method between patient with MetS and healthy subjects in Iran.

According to the cohort study performed by Kuk and Arden during 8.7 years in 6011 men and women, a rare phenotype of obese individuals who are metabolically healthy, need to improve their obesity. Lack of nutritional intervention is contrary to U.S obesity treatment algorithm, so it is recommended that all overweight and obese individuals with and without MetS follow a nutrition therapy program (29).

Conclusion

BMI is a better predictor of MetS rather than body fat mass. Although many factors such as physical inactivity, western diet, BMI and genetic has been shown to be inducing factors for developing MetS, probably other factors involved in molecular mechanism of the disorders are also important.

References

1. Reaven GM. The metabolic syndrome: time to get off the merry-go-round? *Journal of internal medicine* 2010;269:127-36.
2. Kassi E, Pervanidou P, Kaltsas G, Chrousos G. Metabolic syndrome: definitions and controversies. *Biomed Central Medicine* 2011;9:48.
3. Reaven GM. Role of insulin resistance in human disease. *Diabetes* 1988;37:1595-607.
4. Mirsa A, Khurana L. Obesity and the Metabolic Syndrome in Developing Countries. *The Journal of Clinical Endocrinology & Metabolism* 2008; 93:S9-S30.
5. Després JP, Lemieux I. Abdominal obesity and metabolic syndrome. *Nature* 2006;444:881-7.
6. Kahn BB, Filier JS. Obesity and insulin resistance. *Journal of clinical investigation* 2000;106:473-81.
7. Kissebah AH. Central obesity: measurement and metabolic effects. *Diabetes Rev* 1997;5:8-20.
8. Wajchenberg BL. Subcutaneous and visceral adipose tissue: their relation to the metabolic syndrome. *Endocr Rev* 2000;21:697-738.
9. Goodpaster B, Krishnaswami S, Resnick H, et al. Association between adipose tissue distribution and both type 2 diabetes and impaired glucose tolerance in elderly men and women. *Diabetes Care*. 2003;26:372-9.
10. Goodpaster B, Krishnaswami S, Harris T, Katsiaras A, Kritchevsky S, Simonsick E, et al. Obesity, Regional Body Fat Distribution, and the Metabolic Syndrome in older Men and Women. *Arch intern Med* 2005;165:777-83.
11. Chan D.C, Watts G.F, Barrett P.H.R, Burke V. Waist circumference, waist-to-hip ratio and body mass index as predictors of adipose tissue compartments in men. *Q J Med* 2003;96:441-7
12. Leitzmann M.F, Moore S.C, Koster A, Harris T.B, Park Y, Hollenbeck A, Schatzkin A. Waist circumference as compared with Body-Mass Index in predicting Mortality from Specific Causes. *Plos one* 2011; 6(4):18582
13. Rodríguez G, Moreno LA, Blay MG, Garagorri JM, Sarrí A, Bueno M. Body composition in adolescents: measurements and metabolic aspects. *International Journal of Obesity* 2004;28:554-8.
14. Panagiotakos DB, Pitsavos C, Skoumas Y, Stefanidis C. The Association between Food Patterns and the Metabolic Syndrome Using Principle Component Analysis: The ATTICA Study. *Journal of the American Dietetic Association* 2007;107:979-87.
15. Kagawa M, Kerr D, Uchida H, Binns CW. Differences in the relationship between BMI and percentage body fat between Japanese and Australian-Caucasian young men. *British Journal of Nutrition* 2006;95:1002-7.
16. Toshihide T, Suzuki K, Mimura Y, Furujo M, Shiraga H, Kanadani T, et al. Evaluation of partial body composition using bioelectrical impedance in Japanese children. *Asia Pac journal of Clinical Nutrition* 2010;19(4):594-601.
17. You T, Ryan AS, Nicklas BJ. The Metabolic Syndrome in Obese Postmenopausal Women: Relationship to Body Composition, Visceral Fat, and Inflammation. *J Clin Endocrinol Metab* 2004;89(11):5517-22.
18. Brochu M, Tchernof A, Dionne IJ, Sites CK, Eltabbakh GH, Sims EA, et al. What are the physical characteristics associated with a normal metabolic profile despite a high level of obesity in postmenopausal women? *Journal of clinical endocrinol metabolic* 2001;86:1020-5.
19. Koster A, Stenholm S, Alley DE, Kin LJ, Simonsick EM, Knaya AM, et al. Body fat distribution and inflammation among obese older adults with and without metabolic syndrome. *Obesity* 2010;18:2354-61.
20. Berenson GS, McMahan CA, Voors AW, Webber LS, Srinivasan SR, Frank GC, et al. 1980. Cardiovascular risk factors in children: The early natural history of atherosclerosis and essential hypertension. New York: Oxford University Press.
21. Adams TD, Heath EM, LaMonte MJ, Gress RE, Pendleton R, Strong M, et al. 2007. The relationship between body mass index and per cent body fat in the severely obese. *Diabetes Obes Metab* 9:498-505.
22. De Lorenzo F, Mukherjee M, Kadziola Z, Suleiman S, Kakkar VV. 1998. Association of overall adiposity rather than body mass index with lipids and procoagulant factors. *Thromb Haemost* 80:603-06.
23. Nagaya T, Yoshida H, Takahashi H, Matsuda Y, Kawai M. 1999. Body mass index (weight/height²) or percentage body fat by bioelectrical impedance analysis: Which variable better reflects serum lipid profile? *Int J Obes Relat Metab Disord* 23:771-4.
24. Choi JW, Choe HW, Pai SH. 2003. Serum lipid concentrations correlate more strongly with total body fat than with body mass index in obese humans. *Clinica Chimica Acta* 329:83-7.
25. Kim J, Meade T, Haines A. 2006. Skin fold thickness, body mass index, and fatal coronary heart disease: 30 year follow up of the Northwick Park heart study. *J Epidemiol Community Health* 60:275-9.
26. Freedman DS, Katzmarzyk PT, Dietz WH, Srinivasan SR, Berenson GS. The relation of BMI and skinfold thicknesses to factors among young and middle-aged adults: the Bogalusa Heart Study. *Annals of Human Biology* 2010;37(6):726-37.
27. Meeuwse S, Horgan GW, Elia M. The relationship between BMI and percent body fat, measured by bioelectrical impedance, in a large adult sample is curvilinear and influenced by age and sex. *Clinical nutrition* 2010;29:560-6.

28. Phillips CM, Tierney AC, Perez-Martinez P, Defoort C, Blaak EE, Gjelstad IM, et al. Obesity and Body Fat Classification in the Metabolic Syndrome: Impact on Cardiometabolic Risk Metabotype. *Obesity* 2012;10:188
29. Kuk JL, Ardern CI. Are metabolically normal but obese individuals at lower risk for all cause mortality? *Diabetes care* 2009;32:2297-9.