Metabolic syndrome in obese versus non-obese persons In basrah

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Abstract

Background: The prevalence of obesity is continuously rising world-wide. Obesity is an important risk factor of cardiovascular disease (CVD), metabolic syndrome (MS), and type 2 diabetes (T2D).

Objective: To estimate the frequency of MS in obese versus non-obese subjects in Basrah, Iraq .

Methods: This is a prospective clinical study performed in Al-Sadr Teaching Hospital, Basrah, and included 86 obese subjects (with a BMI ≥ 30), 39 males and 47 females, and 132 non-obese subjects (with a BMI < 30), 60 males and 73 females as a control group. Measurement of height, weight, waist circumference (WC), blood pressure (BP), fasting blood glucose (FBG), total cholesterol (TC), triglycerides (TG) and high density lipoprotein-cholesterol (HDL-C) levels were carried out. The updated US National Cholesterol Education Program Adult Treatment Panel III (updated NCEP ATP III) definition was used for the diagnosis of MS.

Results: The frequencies of MS were significantly higher among obese male and female persons (38.5% and 42.6% respectively) compared to non-obese male and female persons (15.0% and 19.4% respectively), (P<0.01). On the other hand, the frequencies of MS were non-significantly higher among over weight male and female subjects (19.2% and 24.3% respectively) compared to normal weight male and female subjects (11.8% and 14.3% respectively), (P>0.05). The major determinants for MS in obese persons in either sex were WC, BP and TG (P<0.001, P<0.001 and P<0.05 respectively).

Conclusions: Obese individuals are at a substantial risk for MS, and thereby, at a high risk of atherosclerotic CVD and T2D, and their complications.

Key Words: Metabolic syndrome, obesity, cardiovascular disease, type 2 diabetes.

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Introduction

Obesity is a chronic metabolic disorder associated with cardiovascular disease (CVD) and increased morbidity and mortality. Obesity may be described as an excess body fat, and is defined as a body mass index (BMI= kg/m^2) of equal or more than 30.[1-4] When BMI exceeds 30, the mortality rates from all causes, CVD in particular, are increased by 50-100%.[1]

It has been suggested earlier that regional adipose tissue distribution could serve as an important correlate of glucose intolerance , hyperinsulinaemia and hypertriglyceridaemia as these abnormalities were more frequently observed in the presence of upper than lower body obesity.[5] In addition, some workers stressed on the advantage of anthropometric variables such as waist and hip circumferences to develop a simple index of body fat distribution, the waist to hip ratio.[6] Furthermore, a waist circumference, a measure of abdominal or central obesity, of >102 cm in men and > 88 cm in women implies an increased metabolic risk as well as CVD risk.[2,3]

Over weight and obesity is continuously rising in an epidemic rate all over the world , and became a significant world-wide health problem. Beside it is almost invariable in the developed world, however, also affects many developing countries.[3,4,7-11] The pandemic pattern of obesity reflects changes in both energy intake and energy expenditure.[4] Central obesity is an important risk factor of CVD, metabolic syndrome (MS), and type 2 diabetes (T2D). [2,12] Obesity is the predominant factor leading to insulin resistance (IR),[2,13] which is the major pathogenetic mechanism in the development of MS.[2,14,15]

The MS (syndrome X) , also called "insulin resistance syndrome" is a cluster of disorders including abdominal obesity, atherogenic dyslipidaemia, hypertension, impaired glucose tolerance as well as proinflammatory and thrombotic states.[2,12,16,17] That combination of diseases is associated with an increased risk of development of atherosclerosis, T2D, CVD, thrombotic events and all – cause mortality.[18-20]

The aim of this study was to determine the frequency of MS among obese versus non-obese persons in Basrah.

methods

This is a prospective clinical study , conducted from January 2006 throughout December 2007 , and included 86 obese subjects (with a BMI ≥ 30) , 39 males, 23-69 years of age and 47 females, 21-74 years of age. They consulted the Outpatient Medical Department in Al-Sadr Teaching Hospital in Basrah, Iraq. Regarding medical and drug history, 6 (15.4%) of obese males were hypertensive (4 of them on anti – hypertensive medication specifically angiotensin converting enzyme inhibitors), 5 (12.8%) with T2D (4 on sulphonyl ureas drugs and the fifth was on insulin therapy) and 4 (10.3%) with hypercholesterolaemia (on statins). Among obese females, 8 (17.0%) were hypertensive (5 of them on anti – hypertensive medications, 3 on angiotensin converting enzyme inhibitors, 1 on thiazide diuretic and the fifth was on calcium channel blocker), 4 (8.5%) with T2D (on sulphonyl ureas drugs) and 3 (6.4%) with hypercholesterolaemia (2 of them on statins). In addition, 132 apparently healthy non-obese subjects (with a BMI < 30), 60 males, 21-72 years of age and 72 females, 21-71 years of age were included as a control group. They have no history of systemic diseases such as hypertension or T2D.

For all participant, height, weight, and waist circumference (WC) were measured. Systolic blood pressure (SBP) and diastolic blood pressure (DBP) were also measured.

The updated US National Cholesterol Education Program Adult Treatment Panel III (updated NCEP ATP III) definition was used for the diagnosis of the MS in this study. The diagnosis of MS require the presence of at least three of the following:[12,21]

1. Increased WC: Men ≥ 102 cm
   Women ≥ 88 cm.
2. Elevated TG: ≥ 150 mg/dl
3. Decreased HDL-C: Men < 40 mg/dl.
   Women < 50 mg/dl.
4. Elevated BP: ≥ 130/85 mm Hg or the use of medication for hypertension.
5. Elevated FBG: ≥ 100 mg/dl or the use of medication for hyperglycemia.

Blood samples were collected in a fasting state and used for the estimation of fasting blood glucose (FBG) level and lipid profile. FBG, total cholesterol (TC), triglycerides (TG) and high density lipoprotein- cholesterol (HDL-C) levels were estimated by enzymatic methods using kits from bioMerieux, France .

Statistical analysis was performed using Chi-square (X^2) and t-tests. $P<0.05$ was considered statistically significant.

Results

Table 1 presents the physiological and biochemical parameters in the studied subjects. Among males, WC ($P<0.001$), SBP,DBP, FBG, TG ($P<0.01$) were significantly higher among obese subjects compared to non-obese subjects, while HDL-C was significantly lower among obese in comparison to non-obese males ($P<0.001$). In females, similarly, WC , SBP, DBP , FBG, TG were significantly higher among obese subjects compared to non-obese subjects ($P<0.001$ for all parameters apart from $p<0.01$ for TG). On the other hand, HDL-C was significantly lower among obese than non-obese females ($P<0.001$).

As presented in Table 2, The frequencies of MS were significantly higher among obese male and female people (38.5% and 42.6% respectively) in comparison to non-obese male and female subjects (15.0% and 19.4% respectively), ($P<0.01$).

Although the frequencies of MS were higher among over weight male and female subjects (19.2% and 24.3% respectively) in comparison to male and female subjects with normal weight (11.8% and 14.3% respectively), however, the differences were statistically not significant ($P>0.05$), Table 3 .

Table 4 presents variables of MS in men. BP≥130/85mm.Hg was found among 59.0% of obese subjects compared to 16.7% of non-obese men ($P < 0.001$) . Also, WC ≥ 102 cm was observed among 64.1% of obese in comparison to 23.3% of non-obese subjects ($P < 0.001$). FBG of ≥ 100mg /dl was found among 30.8% of obese subjects compared to 15.0% of non-obese males, however this difference was statistically not significant ($P > 0.05$). In addition, TG level of ≥ 150 mg/dl and HDL-C level of
< 40mg/dl were reported among 53.8% and 43.6% of obese men respectively , in comparison to 30.0% and

18.3% of non-obese men ($P < 0.05$, $P < 0.01$ respectively) .

MS variables in women are presented in Table 5 . BP≥ 130/85mm.Hg was reported among 70.2% of obese females compared to 13.9% of non - obese females
($P < 0.001$) . In addition, WC ≥ 88 cm was found among 78.7% of obese subjects in comparison to 38.9% of non-obese subjects ($P < 0.001$) . In addition, FBG level of ≥ 100mg/dl and TG level of ≥ 150mg/dl were observed among 25.5% and 55.3% of obese women respectively , compared to 11.1% and 31.9% of non-obese women respectively ($P < 0.05$). HDL-C < 50mg /dl was observed among 27.7% of obese females compared to 18.1% of non-obese females, however this difference was statistically not significant ($P > 0.05$).

Discussion

MS is a collection of comorbidities that considerably increases the risk of development atherosclerotic CVD and T2D and peripheral artery disease.[22-25] The prevalence of MS is dramatically increasing worldwide as a consequence of the continued obesity epidemic,[8,26] and as a result will have a substantial impact on the global incidence of CVD and T2D.[26] Currently, it is widely recognized that abdominal obesity, hyperglycaemia, dyslipidaemia and elevated BP are common metabolic derangements that characterize the distinctive MS.[17] In addition, MS refers to a clustering of conventional or major and emerging CVD risk factors within a single individual. The established risk factors, include obesity, diabetes, dyslipidaemia, hypertension, cigarette smoking, family history of coronary heart disease (CHD) and aging. The other emerging risk factors are closely related to central obesity and include hypertriglyceridaemia, small dense LDL particles, IR, and proinflammatory as well as prothrombotic states.[11,27]

The exact aetiological factors of MS is still under considerable matter of debate, and seem to be multifactorial in nature. IR and its consequent hyperinsulinaemia, is a well recognized as an important or even central factor in the pathogenesis of MS.[14,28] The other implicated factors for the development of MS are lifestyle factors including obesity, sedentary lifestyle and diet.[29-32] Additional aetiological factors for MS are atherogenic dyslipidaemia , pro-inflammatory and pro-thrombotic states.[21,31,33] Importantly to mention that, IR itself seems to be due to a combination of genetic factors, physical inactivity, obesity or pro-inflammatory state.[14,33-35]

Beside the fact that, patients with MS are at a considerable risk for atherosclerotic CVD and T2D ,and the increased susceptibility for to cardiovascular (CV) events including myocardial infarction , stroke and peripheral artery disease.[20,36] Also, MS is associated with other clinical conditions including chronic renal disease, polycyctic ovary syndrome, depression, fatty liver, lipodystrophies and other diseases.[31,37-40]

The present study clearly demonstrated a significant increase in the frequency of MS in obese people of either sex. This observation was similar to that of other reports.[22,41] The relationship between MS and obesity is based on the identification of several substances released from the adipose tissues including non – esterified fatty acids (NEFAs), inflammatory cytokines, plasminogen activator inhibitor-1 (PAI-1), adiponectin, leptin and resistin. Obese people have elevated circulating NEFAs and cytokines such as tumour necrosis factor-α, and interleukin-6. These adipocyte products enhance IR in the muscle. Additionally, obese individuals have reduced levels of adiponectin which proposed to have both anti-atherogenic and anti-inflammatory properties.[2] Furthermore, it has been proposed that ectopic fat accumulation, visceral and hepatic, with the pro-inflammatory state are crucial in the pathogenesis of MS.[7,26]. Moreover, It has been suggested that environmental factors during periods earlier in life may influence the susceptibility to the occurrence of MS later in life, with a particular emphasis on the role of unbalanced maternal nutrition.[42] The co-existence of MS and obesity substantially increases the risk of CHD and acute CV events more than the presence of obesity alone.[43]

The epidemic of excess weight world-wide results from imbalance between physical activity and energy intake. Physical inactivity together with unhealthy diet consequently lead to an increase in the incidence of over weight and obesity, and hence, increases the risk of CVD.[2,44] Central or visceral obesity is a predictor of atherosclerotic CVD, T2D and MS.[7] It is associated with multiple atherosclerotic CVD risk factors.[2] It has been emphasized that MS is a multiplex CV risk factors and develops through interaction of obesity and metabolic

susceptibility.[45] Some CVD risk factors may act in a multiplicative and not in an additive fashion.[46]

The present study also revealed that the major determinants for MS in obese persons were WC, BP and hypertriglyceridaemia respectively. It has been found that, among the five components of the ATP III criteria, WC was the single most common parameter to likely meet the MS criteria.[47] In addition, Obese people tend to have an elevated BP compared with lean people,[2] and definitely high BP is a well established CV risk factor. Furthermore, hypertriglyceridaemia is a recognized risk factor for CVD[48-50], and had been reported among patients with CHD.[51-53]

Definitely, combating and dealing with MS will substantially reduces the risk of T2D, CVD and CV events, with lifestyle intervention is the key step in MS intervention strategy. Low energy expenditure and physical inactivity increases the risk of T2D and CVD.[54-56] On the other hand, Practicing regular moderate physical activity reduce IR, improve glucose tolerance and lipid profile. Also, it prevent weight gain, obesity, MS, T2D and CVD.[1,7,17,44,55,57,58]

In conclusion, obese persons are at a considerable risk of the development of MS. Thus, they are at a much greater risk of atherosclerotic CVD and T2D, and their adverse clinical consequences.

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References

1. Poirier P, Despres JP. Exercise in weight management of obesity. Cardiol Clin 2001; 19: 459-470.
2. Grundy SM. Obesity. Metabolic syndrome, and cardiovascular disease. J Clin Endocrinol Metab 2004; 89: 2595-2600.
3. Elia M. Nutrition. In: Kumar P, Clark M, eds. Kumar & Clark Clinical Medicine. 7th ed, Edinburgh: Saunders; 2009: 207-240.
4. Hanlon P, Byres M, Walker BR, Macdonald HM. Environmental and nutritional factors in disease. In: Colledge NR, Walker BR, Ralston SH, eds. Davidson's Principles and Practice of Medicine. 21st ed, Edinburgh: Churchill Livingstone; 2010: 95-129.
5. Kissebah AH, Vydelingum N, Murray R , Evans DF, Hartz AJ, Kalkhoff RK, et al . Relation of body fat distribution to metabolic complications of obesity. J Clin Endocrinol Metab 1982; 54: 254-260 .
6. Krotkiewski M, Björntorp P, Sjöström L, Smith U. Impact of obesity on metabolism in men and women . Importance of regional adipose tissue distribution . J Clin Invest 1983; 72:1150-1162.
7. Zoeller RF. Physical activity and obesity: The interaction and implications for disease risk and the role of physical activity in weight management. Am J Lifestyle Med 2007; 1: 437-446.
8. Misra A, Khurana L. Obesity and the metabolic syndrome in developing countries.
J Clin Endocrinol Metab 2008 ;93(Suppl 1):S9-S30.
9. Ucan O, Ovayolu N. Relationship between diabetes mellitus, hypertension and obesity, and health-related quality of life in Gaziantep, a central south-eastern city in Turkey. J Clin Nurs 2010 ;19: 2511-2519.
10. Ozcirpici B, Coskun F, Sahınoz S, Ozgur S, Bozkurt AI. Obesity prevalence in Gaziantep, Turkey. Indian J Community Med 2009 ;34: 29-34.
11. de Onis M, Blössner M, Borghi E. Global prevalence and trends of overweight and obesity among preschool children. Am J Clin Nutr 2010; 92: 1257-1264.
12. Grundy SM, Brewer Jr HB, Cleeman JI, Smith Jr SC, Lenfant C. 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: 433-438.
13. Abbasi F, Brown BW Jr, Lamendola C, McLaughlin T, Reaven GM. Relationship between obesity , insulin resistance, and coronary heart disease risk. J Am Coll Cardiol 2002; 40: 937-943.
14. Reaven GM. The metabolic syndrome: is this diagnosis necessary? Am J Clin Nutr 2006; 83: 1237-1247.
15. McLaughlin T, Abbasi F, Cheal K, Chu J, Lamendola C, Reaven G. Use of metabolic markers to identify overweight individuals who are insulin resistant . Ann Intern Med 2003; 139: 802-809.
16. Banaszewska B, Duleba AJ, Spaczynski RZ, Pawelczyk L. Lipids in polycystic ovary syndrome ;

Role of hyperinsulinemia and effects of metformin ; Am J Obstet Gynaecol 2006; 194: 1266-1272 .
17. Carroll S, Dudfield M. What is the relationship between exercise and metabolic abnormalities? A review of the metabolic syndrome. Sport Med 2004; 34: 371-418.
18. Lakka HM, Laaksonen DE, Lakka TA, Niskanen LK, Kumpusalo E,Tuomilehto J, et al. The metabolic syndrome and total and cardiovascular disease mortality in middle-aged men. JAMA 2002;288: 2709-2716.
19. Meigs JB. Metabolic syndrome: In search of a clinical role. Diabetes Care 2004; 27: 2761-2763.
20. Hoerger TJ, Ahmann AJ. The impact of diabetes and associated cardiometabolic risk factors on members: strategies for optimizing outcomes. J Manag Care Pharm 2008 ; 14(Suppl C): S2-S14.
21. Third Report of The National Cholesterol Education Program (NCEP) Expert Panel on detection, evaluation, and treatment of high blood cholesterol in adults (Adult Treatment Panel III) final report. Circulation 2002;106:3143-3421.
22. Ford ES, Giles WH, Dietz WH. Prevalence of metabolic syndrome among US adults: findings from the Third National Health and Nutrition Survey. JAMA 2002; 287: 356-359.
23. Greenland P. Critical questions about the metabolic syndrome. Circulation 2005; 112: 3675-3676.
24.Antonini-Canterin F, La Carrubba S, Gullace G, Zito C, Di Bello V, Di Salvo G, et al. Association between carotid atherosclerosis and metabolic syndrome: Results from the ISMIR study. Angiology 2010; 61: 443-448.
25. Zerati AE, Wolosker N, Rosoky RMA, Saes GF, Ragazzo L, Puech-Leao P. Prevalence of metabolic syndrome in patients with intermittent claudication and its correlation with the segment of arterial obstruction. Angiology 2010; 61: 784-788.
26. Bruce KD, Byrne CD. The metabolic syndrome : common origins of a multifactorial disorder. Postgrad Med J 2009; 85: 614-621.
27. Matfin G. Developing therapies for the metabolic syndrome: challenges, opportunities, and the unknown. Therapeutic Advances in Endocrinology and Metabolism 2010; 1: 89-94.
28. Lebovitz HE, Banerji MA. Point: visceral adiposity is causally related to insulin resistance. Diabetes Care 2005; 28:2322–2325.
29. Mokdad AH, Bowman BA, Ford ES, Vinicar F, Marks JS, Koplan JP . The continuing epidemics of obesity and diabetes in the United States. JAMA 2001; 286: 1195-1200.
30. Grundy SM , Hansen B, Smith Jr SC Jr, Cleeman JI, Kahn RA. Clinical management of metabolic syndrome: report of the American Heart Association / National Heart, Lung, and Blood Institute / American Diabetes Association conference on scientific issues related to management. Circulation 2004; 109: 551-556.
31. Grundy SM, Cleeman JI , Daniels SR, Donato KA, Eckel RH, Franklin BA, et al; Diagnosis and management of the metabolic syndrome: an American Heart Association/National Heart, Lung, and Blood Institute Scientific Statement. Circulation 2005;112:2735-2752.
32. Prentice AM. The emerging epidemic of obesity in developing countries. Int J Epidemiol 2006 ;35:93-99.
33.Dandona P , Aljada A, Chaudhuri A , Mohanty P, Garg R. Metabolic syndrome: a comprehensive perspective based on interactions between obesity, diabetes, and inflammation. Circulation 2005 ;111:1448-1454.
34. Bouzakri K, Ribaux P, Halban PA. Silencing mitogen-activated protein 4 kinase 4 (MAP4K4) protects beta cells from tumor necrosis factor-α-induced decrease of IRS-2 and inhibition of glucose - stimulated insulin secretion . J Biol Chem 2009; 284: 27892-27898.
35. Pollex RL, Hegele RA. Genetic determinants of the metabolic syndrome. Nat Clin Pract Cardiovasc Med 2006; 3: 482–489.
36. Janghorbani M, Amini M. Metabolic syndrome in type 2 diabetes mellitus in Isfahan, Iran: Prevalence and risk factors. Metabolic Syndrome and Related Disorders 2007; 5: 243-254.
37. Vaccarino V, McClure C, Johnson BD, Sheps DS, Bittner V, Rutledge T, et al. Depression , the metabolic syndrome and cardiovascular risk. Psychosomatic Medicine 2008; 70: 40-48.
38. Locatelli F , Pozzoni P , Del Vecchio L. Renal manifestations in the metabolic syndrome. J Am Soc Nephrol 2006 17(Suppl 2):S81-S85.
39. Khunti K, Davies M. Metabolic syndrome. BMJ 2005; 331: 1153-1154 .
40. Glueck CJ, Papanna R, Wang P, Goldenberg N, Sieve-Smith L. Incidence and treatment of metabolic syndrome in newly referred women with confirmed polycystic ovarian syndrome . Metabolism 2003; 52: 908-915 .
41. Goodpaster BH, Krishnaswami S, Harris TB,

Katsiaras A, Kritchevsky SB, Simonsick EM, et al. Obesity, regional body fat distribution, and the metabolic syndrome in older men and women. Arch Intern Med 2005; 165; 777-783.
42. Bruce KD, Hanson MA. The Developmental origins, mechanisms, and implications of metabolic syndrome. J Nutr 2010; 140: 648-652.
43. Van Guilder GP, Hoetzer GL, Greiner JJ, Stauffer BL, DeSouza CA. Metabolic syndrome and endothelial fibrinolytic capacity in obese adults. Am J Physiol Regul Integr Comp Physiol 2008; 294: 39-44.
44. Lakka TA, Bouchard C. Physical activity, obesity and cardiovascular diseases.
Handb Exp Pharmacol 2005; 170: 137-163.
45. Grundy SM. Metabolic syndrome: A multiplex cardiovascular risk factor. J Clin Endocrinol Metab 2007; 92: 399-404.
46. Ferrannini E. Metabolic Syndrome: A solution in search of a problem. J Clin Endocrinol Metab 2007; 92: 396-398.
47. Meis SB, Schuster D, Gaillard T, Osei K. Metabolic syndrome in nondiabetic, obese, first-degree relatives of African American patients with type 2 diabetes: African American triglycerides-HDL-C and insulin resistance paradox. Ethn Dis 2006 16:830-836.
48. Watts GF, Karpe F. Triglycerides and atherogenic dyslipidaemia: extending treatment beyond statins in the high - risk cardiovascular patient. Heart 2011; 97: 350-356.
49. Watts GF, Karpe F. Why, when and how should hypertriglyceridaemia be treated in the high - risk cardiovascular patient? Expert Rev Cardiovasc Ther 2011; 9: 987- 997.

50. Nikkila M, Koivula T, Niemela K, Sisto T. High density lipoprotein cholesterol and triglycerides as markers of angiographically assessed coronary artery disease. Br Heart J 1990; 63: 78-81.
51. Ajlan SK, Baqir YA. Changes of liver enzymes in coronary heart disease. Karbala J Med, 2011; 4: 993-1000.
52. Baqir YA, Hamadi SS, Ajlan SK. Plasma fibrinogen and coronary heart disease: The relationship. Journal Faculty Medicine Baghdad, 2003; 45: 135-140.
53. A1-Naama LM, Ajlan SK, AI-Hamdi AT, Al-Naama MM. Lipid profile in coronary heart disease. J Bahrain Medical Society 2001;13:171-175.
54. Hamilton MT, Hamilton DG, Zderic TW. Role of low energy expenditure and sitting in obesity, metabolic syndrome, type 2 diabetes, and cardiovascular disease. Diabetes 2007; 56: 2655-2667.
55. Mora S, Lee IM, Buring JE, Ridker PM. Association of physical activity and body mass index with novel and traditional cardiovascular biomarkers in women. JAMA 2006; 295:1412-1419.
56. Blair SN, Kampert JB, Kohl HW 3rd, Barlow CE, Macera CA, Paffenbarger RS Jr, et al. Influences of cardiorespiratory fitness and other precursors of cardiovascular disease and all-cause mortality in men and women. JAMA 1996; 276: 205-210.
57. Lakka TA, Laaksonen DE. Physical activity in prevention and treatment of the metabolic syndrome. Appl Physiol Nutr Metab 2007; 32: 76-88.
58. Mora S, Cook N, Buring JE, Ridker PM, Lee IM. Physical activity and reduced risk of cardiovascular events. Circulation 2007; 116: 211
0-2118

Table 1. Physiological and biochemical Parameters among the studied subjects

Parameter	Obese males n=39	Non-obese males n=60	Obese females n=47	Non-obese females n=72
Age (year)	45.4 (9.9)	48.8 (12.4)	42.2(11.9)	39.8(13.8)
BMI (kg/m^2)	33.8 (2.4)***	24.1 (3.1)	35.2(2.9)***	24.6(3.4)
WC (cm)	107.6 (7.4)***	98.0 (3.8)	96.2(8.9)***	87.1(2.4)
SBP (mm.Hg)	133.1 (12.9)**	125.7 (12.2)	131.8(9.6)***	123.2(11.3)
DBP (mm.Hg)	90.1 (9.2)**	84.3 (8.1)	89.4(9.4)***	82.1(7.6)
FBG (mg/dl)	102.6 (19.2)**	94.1 (10.6)	104.4(23.2)***	92.3(8.5)
TG (mg/dl)	158.8 (35.9)**	139.9 (31.3)	156.0(30.1)**	138.3(28.9)
HDL-C (mg/dl)	38.5 (6.1)***	45.9 (6.4)	48.3(7.7)***	53.2(5.5)

Values are given in mean(SD) .
****: P < 0.01, ***: P < 0.001 (obese males vs non-obese males, and obese females vs non-obese females)**

Table 2. The frequency of MS among the study groups

MS presence		Obese		Non-obese	
		No.	%	No.	%
Males*	Present	15	38.5	9	15.0
	Absent	24	61.5	51	85.0
	Total	39	100.0	60	100.0
Females**	Present	20	42.6	14	19.4
	Absent	27	57.4	58	80.6
	Total	47	100.0	72	100.0

*** : X^2= 7.734 , P < 0.01 (obese males vs non-obese males)**
**** : X^2= 7.509 , P < 0.01 (obese females vs non-obese females)**

Table 3. The frequency of MS among non-obese subjects

MS presence		Normal weight		Over weight	
		No.	%	No.	%
Males*	Present	4	11.8	5	19.2
	Absent	30	88.2	21	80.8
	Total	34	100.0	26	100.0
Females**	Present	5	14.3	9	24.3
	Absent	30	85.7	28	75.7
	Total	35	100.0	37	100.0

*** : X^2= 0.644 , P > 0.05 (normal weight males vs over weight males)**
**** : X^2= 1.149 , P > 0.05 (normal weight females vs over weight females)**

Table 4. Criteria of MS among males

Group		BP ≥ 130/85 mm.Hg		WC ≥102cm		FBG ≥ 100mg/dl		TG ≥150mg/dl		HDL < 40mg/dl	
		No.	%	No.	%	No.	%	No.	%	No.	%
Obese	Present	23	59.0	25	64.1	12	30.8	21	53.8	17	43.6
	Absent	16	41.0	14	35.9	27	69.2	18	46.2	22	56.4
	Total	39	100.0	39	100.0	39	100.0	39	100.0	39	100.0
Non-obese	Present	10	16.7	14	23.3	9	15.0	18	30.0	11	18.3
	Absent	50	83.3	46	76.7	51	85.0	42	70.0	49	81.7
	Total	60	100.0	60	100.0	60	100.0	60	100.0	60	100.0
Level of significance		X^2 =19.038 P < 0.001		X^2=16.326 P < 0.001		X^2 = 3.462 P > 0.05		X^2 = 5.556 P < 0.05		X^2= 7.423 P < 0.01	

Table 5. Criteria of MS among females

Group		BP ≥ 130/85 mm.Hg		WC ≥88cm		FBG ≥ 100mg/dl		TG ≥150mg/dl		HDL < 50mg/dl	
		No.	%	No.	%	No.	%	No.	%	No.	%
Obese	Present	33	70.2	37	78.7	12	25.5	26	55.3	13	27.7
	Absent	14	29.8	10	21.3	35	74.5	21	44.7	34	72.3
	Total	47	100.0	47	100.0	47	100.0	47	100.0	47	100.0
Non-obese	Present	10	13.9	28	38.9	8	11.1	23	31.9	13	18.1
	Absent	62	86.1	44	61.1	64	88.9	49	68.1	59	81.9
	Total	72	100.0	72	100.0	72	100.0	72	100.0	72	100.0
Level of Significance		$X^2 = 39.108$ $P < 0.001$		$X^2 = 18.117$ $P < 0.001$		$X^2 = 4.227$ $P < 0.05$		$X^2 = 6.322$ $P < 0.05$		$X^2 = 1.50$ $P > 0.05$	

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