Prevalence of abdominal obesity and its associated comorbid condition in adult Yemeni people of Sana'a City

Mohammed Ahmed Bamashmos

Correspondence:
Mohammed Ahmed Bamashmos, MD
Associate Professor of Internal Medicine,
Faculty of Medicine, Sana'a University,
Yemen
Email: mohbamashmoos @yahoo.com

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Abstract

Objective: Abdominal obesity is a metabolic problem that has become increasingly common worldwide over the past several decades. Its prevalence is increased in both advanced and developing countries including Yemen. The aim of this cross sectional study was to investigate the prevalence of Abdominal obesity in a sample of Yemeni adult individuals and its association with other comorbid conditions namely, hypertension, diabetes, dyslipidemia (high triglyceride, low high density lipoprotein) and metabolic syndrome (MS)..

Methodology: A sample of 1118 adult Yemeni people equal to or over 18 years was randomly chosen to represent the population living in Sana'a City during a period of two years from April 2016 to April 2018. All the study group underwent full clinical history and examination which included measurement of BP and waist circumference and the following laboratory investigations (FBS, serum TG, HDL, and LDL).

Results: The prevalence of abdominal obesity in this study was 24.5% (7.9% male and 44.2% female). Central obesity in this study was significantly correlated with age and sex. The highest prevalent comorbidity in patients with abdominal obesity was high BP (41.3%), followed by high serum TG (40 %), higher prevalence of MS (40%), low serum HDL (37.8%) high LDL (20.1%) raised fasting blood glucose (22.1%) than those without abdominal obesity (5.5%, 31.3%, 16.6%, 8.5%, 12.5% and increased FBS 10% respectively).

Conclusion: Hypertension, diabetes, dyslipidemia and MS are strongly correlated with abdominal obesity.

Key words : central obesity, hypertension, dyslipidemia, MS

Introduction

Obesity is a worldwide epidemic. Beyond the fat mass per se, the pattern of fat distribution has a profound influence on cardiometabolic risk. Visceral abdominal fat (VAF) is metabolically active and pro-inflammatory and presents a higher cardiometabolic risk association and calcification of the coronary arteries than the body mass index (BMI) and has more impact on health than subcutaneous fat, presenting a risk factor for increased incidence of metabolic syndrome (1, 2). Abdominal obesity (AO) is directly associated with increased VAF, and it is also associated with endothelial dysfunction, inflammation, insulin resistance, diabetes mellitus, hypercholesterolemia, metabolic syndrome [MetS], and cancer (1, 3). There are several methods available to measure AO. Waist circumference (WC) provides an indicator of central adiposity that is the most practical and easiest method used in large-scale epidemiological studies (4). It is a good predictor of cardiometabolic morbidity and mortality, and it has also a positive association with visceral abdominal fat. However, WC does not allow us to differentiate between visceral fat and subcutaneous fat; methods such as absorptiometry by dual energy X-ray (DEXA), impedance, or densitometry can be used to handle this differentiation (5–7). WC measurement requires correct and standardized procedures, which depend mainly on training and adequate equipment. A standardized technique requires that the person being measured removes bulky or tight garments, as well as shoes with heels, empties their bladder then stands in the upright position, with arms loosely positioned to the side. The tape is passed around the body and positioned mid-way between the iliac crest and costal margin of the lower rib, ensuring that it is Population Organization (References) Recommended waist circumference threshold for abdominal obesity Men Women Europid IDF ≥94 cm ≥80 cm Caucasian WHO ≥94 cm [increased risk]≥102 cm [still higher risk] ≥80 cm [increased risk]≥88 cm [still higher risk] United States AHA/NHLBI [ATP III] ≥102 cm ≥88 cm Canada Health Canada ≥102 cm ≥88 cm European European cardiovascular societies ≥102 cm ≥88 cm Asian [including Japanese] IDF ≥90 cm ≥80 cm Asian WHO ≥90 cm ≥80 cm Japanese Japanese obesity society ≥85 cm ≥90 cm China Cooperative task force ≥85 cm ≥80 cm Middle East, Mediterranean IDF ≥94 cm ≥80 cm Sub-Saharan African IDF ≥94 cm ≥80 cm Ethnic Central and South American IDF ≥90 cm ≥80 cm. Waist circumference cutoffs are recommended for the diagnosis of abdominal obesity according to ethnicity and gender (8,9). In 2009, a method to standardize the diagnosis of metabolic syndrome was established, upon discussions held by the International Diabetes Federation (IDF) and the American Heart Association/National Heart, Lung, and Blood Institute. In this context, it was suggested that ethnicity and gender should be considered for the diagnosis of AO (9)

In decades past, many clinicians were taught in medical school that the adipose cell is a vehicle for energy storage and nothing more. Today, there is a different perception of the adipose cell; specifically, it is an active endocrine organ that communicates with gut hormones and a master regulator in the brain to control appetite and satiety. It also exerts pathologic effects on other organs and critical metabolic and immunologic processes .Four major hormones—ghrelin, insulin, peptide YY (PYY) from the gut, and leptin from fat tissue—participate in appetite and satiety regulation in communication with each other and the central control of energy balance, the arcuate nucleus in the hypothalamus. Ghrelin is a short-term appetite hormone, the “hunger hormone”, that brings on feelings of hunger at mealtimes. Secreted in the gastric fundus, ghrelin rises immediately before meals and falls as insulin levels rise in response to the meal(10,11). It also increases during weight loss, which may be a factor in making successful weight loss so difficult. However, ghrelin levels decrease in persons who have had gastric bypass surgery, which may aid in maintaining weight loss (10). Insulin, in addition to its multiple other metabolic functions, acts as a satiety signal to the brain, causing feelings of fullness. PYY, secreted in the distal small intestine in response to food, signals satiety to the hypothalamus to counteract the influence of ghrelin. Levels of PYY are significantly lower in obese versus normal-weight persons,(8) but are elevated after gastric bypass(11). Leptin was the original “satiety factor,” discovered to much excitement by Friedman and coworkers in 1994(12). This hormone is produced in adipose tissue in proportion to body fat; the more fat present, the more leptin secreted. Initially, it was hoped that exogenous leptin administration would be a “magic bullet” for curing obesity and establishing the condition as a metabolic disorder instead of a personal failing(9). Despite its effectiveness in mice with a genetic defect in the leptin molecule, exogenous leptin does not produce meaningful weight loss in obese humans, apparently because the brain becomes resistant to it. However, leptin is one of many neuro hormonal pathways that have evolved genetically to prevent starvation and ensure survival. In addition, the hypothalamus contains neurons that can either stimulate or inhibit food intake, and many of the gut hormones exert actions on both sides of the equation(13). The possibilities for routes to intervention among this wealth of pathways remain promising and rational in the search for weight control treatments. Current thinking now holds that fat tissue is an active participant in weight regulation. In one current theory, it is the basis of adiposopathy, defined by Bays and colleagues as pathogenic adipose tissue whose toxicity may be worsened by fat accumulation and a sedentary lifestyle in genetically susceptible individuals(14). This dysfunctional tissue releases increased amounts of free fatty acids (FFAs) and abnormal amounts of inflammatory factors such as cytokines from macrophages. Such changes can promote insulin resistance in skeletal muscle and the liver, increased insulin secretion, dyslipidemia, hypertension, and type 2 diabetes, all components of the metabolic syndrome, which increases the risk of atherosclerosis (14-16). Muscle biopsies in obese patients show that deposits of fat are stored in liver when fat can no longer be stored subcutaneously, with some cases resulting in steatohepatitis and eventually fibrosis. Excess fat may be associated with androgen elevations in women (or decreases in men);

increases in plasminogen-activator inhibitor 1, which encourages thrombosis; and asthma, as a consequence of pro inflammatory changes. Even osteoarthritis may be an outcome of increased inflammation, although it is also mediated by the mechanical load of excess weight on the joints(17,18). Not everyone who is obese has metabolic syndrome or type 2 diabetes—although these are perhaps the most prevalent complications of obesity. Genetic susceptibility is yet another confounding component of obesity. The ability to differentiate genetically susceptible humans is not yet within reach. Better predictors of who will and will not respond to adiposity with endocrine and other consequences are still needed. In the meantime, ongoing research on appetite and satiety regulation has made it clear that obesity is a disease largely beyond an individual's control(19) rather than a function of inadequate willpower. The development and availability of medications and procedures that address the underlying pathobiology of obesity herald a new approach to stemming the tide of the obesity epidemic.

Material and Method

This was a cross sectional population based study conducted in Sana'a city for a period of 2 years between April 2017 and April 2018. A sample of 1,118 adult Yemeni people (508 male and 610 aged ≥18 years) was randomly selected. The data collection was from those attending Al-Kuwait University Hospital and Consultation Clinic. All the participants in this study underwent complete clinical history (regarding their age, occupation, habits, any history of hypertension, diabetes mellitus, dyslipidemia and medication). Anthropometric measurements included measurement of waist circumference and systolic and diastolic blood pressure. Waist circumference was manually measured on standing subjects with soft tape midway between the lowest rib and the iliac crest. WC was divided into abdominal overweight (85–95 cm in males and 80–90 cm in females) and abdominal obesity groups (WC ≥ 95 cm in males and ≥ 90 cm in females) (20,21). WC ≥ 85 cm in males and ≥ 80 cm in females were defined as elevated WC. Two blood pressure recordings were obtained from the right arm of patients with standard mercury sphygmomanometer in a sitting position after 10 minutes of rest; measurements were taken in 3-5 minute intervals and the mean values were calculated . Blood pressure was classified as normotensive (SBP < 120 mmHg and DBP < 80 mmHg), pre-hypertensive (SBP: 120–139 mmHg and/or DBP: 80–89 mmHg) and hypertensive (SBP ≥ 140 mmHg and/or DBP ≥ 90 mmHg) by the Seventh Report of the Joint National Committee on the Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (22,23); fasting blood glucose, total cholesterol, triglyceride, LDL and HDL cholesterol were measured.

The American Diabetes Association criteria was used to classify FBG as normal glucose (FBG < 5.6 mmol/L), impaired fasting glucose (IFG) (FBG ≥ 5.6 mmol/L ≤ FBG < 7.0 mmol/L), and diabetic (FBG ≥ 7.0 mmol/L).

Type 2 diabetes mellitus was defined according to the American Diabetes Association (24) A1C ≥ 6.5%. The test should be performed in a laboratory using a method that is NGSP certified and standardized to the DCCT assay or FPG ≥126 mg/dl (7.0 mmol/l). Fasting is defined as no caloric intake for at least 8 hours. In the absence of unequivocal hyperglycemia, result should be confirmed by repeat testing or 2-hour plasma glucose ≥200 mg/dl (11.1mmol/l) during an OGTT. The test should be performed as described by the World Health Organization, using a glucose load containing the equivalent of 75 g anhydrous glucose dissolved in water. In the absence of unequivocal hyperglycemia, result should be confirmed by repeat testing or in a patient with classic symptoms of hyperglycemia or hyperglycemic crisis, a random plasma glucose 200 mg/dl (11.1mmol | l).

Dyslipidemia was classified according to ATP III, TG: Normal < 1.69 mmol/L, Borderline high 1.69–2.26 mmol/L, High 2.26–5.65 mmol/L, Very high ≥ 5.65 mmol/L; TC: Desirable < 5.17 mmol/L, Borderline high 5.17–6.24 mmol/L, High ≥ 6.24 mmol/L; HDL-C: High 1.56 mmol/L, Optimal 1.03–1.56 mmol/L, Low <l.03 mmol/L; LDL-C: Optimal <2.59 mmol/L, Near optimal 2.59–3.38 mmol/L, Borderline high 3.38–4.16 mmol/L, High 4.16–4.94 mmol/L, Very high ≥4.94 mmol/L.(25).

Metabolic syndrome was diagnosed by the presence of three or more of the following criteria (26).

- Fasting glucose ≥100 mg/dL (or receiving drug therapy for hyperglycemia)
- Blood pressure ≥130/85 mm Hg (or receiving drug therapy for hypertension)
- Triglycerides ≥150 mg/dL (or receiving drug therapy for hypertriglyceridemia)
- HDL-C < 40 mg/dL in men or < 50 mg/dL in women (or receiving drug therapy for reduced HDL-C)
- Waist circumference ≥102 cm (40 in) in men or ≥88 cm (35 in) in women; if Asian American, ≥90 cm (35 in) in men or ≥80 cm (32 in) in women (The international diabetes federation [IDF] criteria allow the use of a body mass index [BMI] >30 kg/m2 in lieu of the waist circumference criterion.)

Statistical analysis was undertaken using the Statistical Package for the Social Sciences (Windows version 13.0; SPSS, Chicago IL USA). Differences between groups were tested statistically using the Chi square test for categorical and T test for numerical variables. Data were considered statistically significant when the p-value was ≤ 0.05.

Results

A study sample included 1,118 persons aged between 18-83; of them 508 (45.4%) were male and 610 (54.6%) were female . The Mean age ± SD was 47.4 ±10.2 years, with an age range of 30–77 years with no significant age difference between men and women.

The prevalence of abdominal obesity according to WC was 24.5%. The physical and metabolic characteristic of the study population by gender are shown in Table 1. Women have significantly higher prevalence of abdominal obesity, systolic and diastolic BP, high serum TG, high LDL, low serum HDL as well as high prevalence of MS.

Table 1: The clinical and laboratory characteristics of the study group

Variable		Men=508 (45.4%)	Women =610 (54.7%)	Total =1118	p-value
WC					
-	Normal	468(92.1%)	240(55.8%)	708(75.5%)	0.000
-	Obese	40(7.9%)	190(44.2%)	230(24.5%)	
BP					
-	Normal	458(91.2%)	516(84.6%)	974(87.1%)	0.003
-	High	50(9.8%)	94(15.5%)	144(12.9%)	
FBS					
-	Normal	434(85.4%)	524(85.9%)	958(85.7%)	0.65
-	IFG	28(5.5%)	16(2..6%)	45(3.9%)	
-	Diabetes	26(9.1%)	70(11.5%)	95(10.4%)	
TG					
-	Normal	310(61%)	440(72.1%)	750(67.1%)	
	High	198(39%)	170(27.9%)	368(32.9%)	0.000
HDL					
-	Normal	412(81.1%)	544(89.2%)	956(85.5%)	0.000
-	High	96(18.9%)	66(10.5%)	162(14.5%)	
LDL					
-	Normal	304(59.8%)	296(48.5%)	600(53.7%)	0.000
-	High	204(40.2%)	310(51.5%)	518(46.3%)	
-	MS	85 (16.7%)	155 (25.4%)	240 (21.4%)	0.000

Table 2: The prevalence of both clinical and laboratory characteristics between obese and non-obese

Factors	Total =1118	With central obesity= 230	Without central obesity=888	P- value
Age	47.5±10.2	41.2±11.4	38.4±11.4	0.0321
Male gender	508 (45.4%)	40(17.3%)	468(52.7%)	0.0001
FBS mg/dl	140 (12.5%)	51(22.1%)	89(10%)	0.0001
BP mmHg	144(12.8%)	95(41.3%)	49 (5.5%)	0.0001
TG mg/dl	368(32.9%)	92(40%)	276(31.3%)	0.011
LDL	245 (21.9%)	134(20.1%)	111(12.5%)	0.0001
HDL	162 (14.4%)	87(37.8%)	75(8.5%)	0.0001
MS	240(21.4%)	92 (40%)	148(16.6%)	0.0001

The highest prevalent comorbidity in patients with abdominal obesity was high BP (41.3%), followed by high serum TG (40 %), high prevalence of MS (40%), low serum HDL (37.8%) high LDL (20.1%) raised fasting blood glucose (22.1%) than those without abdominal obesity (5.5%, 31.3%, 16.6%, 8.5%, 12.5% and increased FBS 10% respectively).

Discussion

The prevalence of abdominal obesity is increasing dramatically worldwide(27,28). In the United States, the overall age-adjusted prevalence of abdominal obesity increased significantly from 46.4% (95% confidence interval [CI], 42.1%–50.8%) in 1999–2000 to 54.2% (95% CI, 51.3%–57.0%) in 2011–2012(27).

Obesity is a major risk factor for hypertension, type 2 diabetes, coronary heart disease, and certain types of cancer(29,30,31,32,33). Obesity is classified as general obesity (defined as body mass index ≥30 kg/m2) and abdominal obesity (defined as waist circumference [WC] ≥90 cm for men and WC ≥80 cm for women), based on World Health Organization (WHO) recommendations for Asians(34,35). In particular, abdominal obesity has a close relationship with central fat localization and cardiovascular disease, independently of general obesity(36,37,38). The overall prevalence of abdominal obesity among the study population in the present study was found to be 24.5% which is comparable to that found in Nigeria (21.8%) [39] , Tanzania (24.8%)(40) and Brazil (30%) (41), lower than that was found in South Asia (68.9%) (42), USA (56%)(43) and Mexico (74%) (44). The prevalence of abdominal obesity in population-based studies from the Arab world was found to be variable and was in the range of 23 % to 46.5% (45,46).

In our study, there was significant gender difference which is consistent with the findings from Saudi Arabia (46) and Taiwan (23) .

The highest prevalent abdominal obesity co morbidity in our study population was hypertension (41.3%)(47) followed by atherogenic dyslipidemia as high TG (40%) low serum HDL (37.8%) and high serum LDL (20.1%) (48), followed by higher prevalence of MS (41.3%) (49.50) and raised fasting blood glucose (22.1%) (51) than those without central obesity (5.5%, 31.3%, 16.6%, 8.5%, 12.5% and increased FBS 10%) respectively .

This finding was in keeping with the existing knowledge that obesity is clearly linked to essential hypertension. Hypertension was also the most common comorbid condition in abdominal obese subjects (47, 48). The mechanism linking abdominal obesity with hypertension might be explained by the activation of the renin–angiotensin–aldosterone system which primarily leads to the activation of the sympathetic activity, promotion of the leptin resistance by increased pro-coagulatory activity. The cumulative effect of this cascade is endothelial dysfunction and inflammatory changes. Additional mechanisms include the enhanced renal sodium reabsorption with a resultant increase in volume expansion usually observed in abdominally obese patients (52). In our study 40% of the obese patients had dyslipidemia (51). This fact can be explained by the accumulation of adipose tissue and the release of free fatty acids which are easily directed to the liver for higher production of TG and very low density lipoprotein (51,52). Raised fasting blood glucose was the lowest comorbidity among abdominal obese subjects (53.54).

Conclusion

Our study demonstrates an alarming high prevalence of abdominal obesity among Yemeni patients that increases the burden on an overstrained Yemeni health system with uprising CVDs and other AO related health problems e.g. hypertension, dyslipidemia, DM. As obesity is the main modifiable risk factor for these comorbid conditions, raising community awareness and promotion of healthy lifestyle together with organizing training courses for health educators are highly recommended. There is also an urgent need to develop strategies for prevention, detection, and treatment of AO that could contribute to decreasing the incidence of grave consequences such as cardiovascular disease and diabetes.

Abbreviations: World Health Organization (WHO), waist circumference (WC), abdominal obesity (AO) , Metabolic Syndrome (MS), Adult Treatment Panel (ATP) serum triglyceride (TG),High Density Lipoprotein (HDL), Fasting Blood Sugar (FBS), National Cholesterol Education Program Adult Treatment Panel (NCEP ATP),

References

1- Shah RV, Murthy VL, Abbasi AS, Blankstein R, Kwong RY, Goldfine AB, et al. Visceral adiposity and the risk of metabolic syndrome across body mass index: the MESA study. JACC Cardiovasc Imaging. 2014;7(12):1221–1235. doi:10.1016/j.jcmg.2014.07.017

2- Carmienke S, Freitag MH, Pischon T, Schlattmann P, Fankhaenel T, Goebel H, et al. General and abdominal obesity parameters and their combination in relation to Epidemiology of Abdominal Obesity http://dx.doi.org/10.5772/65342 85 mortality: a systematic review and meta-regression analysis. Eur J Clin Nutr. 2013; 67 (6):573–585. doi:10.1038/ejcn.2013.61

3- Martins IS, Marino SP. The potential of central obesity antropometric indicators as diagnostic tools. Rev Saúde Pública. 2003;37(6):760–767.

4- Olinto MTA, Nácul LC, Dias-da-Costa JS, Gigante DP, Menezes AMB, Macedo S. Intervention levels for abdominal obesity: prevalence and associated factors. Cad Saúde Pública. 2006;22(6):1207–1215. doi:10.1590/S0102-311X2006000600010

5- Lee K, Lee S, Kim YJ, Kim YJ. Waist circumference, dual-energy X-ray absortiometrically measured abdominal adiposity, and computed tomographically derived intra-abdominal fat area on detecting metabolic risk factors in obese women. Nutrition. 2008;24:625–631. doi:10.1016/j.nut.2008.03.004

6- Lean ME, Han TS, Deurenberg P. Predicting body composition by densitometry from simple anthropometric measurements. Am J Clin Nutr. 1996;63:4–14.

7- Menezes TN, Rocha FL, Belém PLO, Pedraza DF. Abdominal obesity: a critical review of the measurement techniques and cutoff points of anthropometric indicators adopted in Brazil. Ciência & Saúde

8- World Health Organization (WHO). Waist Circumference and Waist-Hip Ratio: Report of a WHO Expert Consultation, Geneva, 8–11 December 2008. Geneva: WHO; 2011.
9- Alberti KG, Eckel RH, Grundy SM, Zimmet PZ, Cleeman JI, Donato KA, et al. Harmonizing the metabolic syndrome: a joint interim statement of the International Diabetes Federation task force on Epidemiology and Prevention; National Heart, Lung, and Blood Institute; American Heart Association; World Heart Federation; International Atherosclerosis Society; and International Association for the Study of Obesity. Circulation. 2009;120:1640–1645. doi:10.1161/CIRCULATIONAHA.109.192644
10- Batterham RL, Cohen MA, Ellis SM, et al. Inhibition of food intake in obese subjects by peptide YY3-36. N Engl J Med. 2003;349:941-948.
11- Investigating Minds. March 30, 2009. http://im-promise-of-leptin_30.html. 0tb.blogspot.com/2009/03/obesityreviving-
12 - Kashyap SR, Gatmaitan P, Brethauer S, Schauer P. Bariatric surgery for type 2 diabetes: weighing the impact for obese patients. Cleve Clin J Med. 2010;77:468-476.
13- Suzuki K, Simpson KA, Minnion JS, ShillitoJC, Bloom SR. The role of gut hormones and the hypothalamus in appetite regulation. Endocr J. 2010;57:359-372.
14- Bays H. Adiposopathy—defining, diagnosing, and establishing indications to treat 'sick fat': what are the regulatory considerations? US Endocrine Dis. 2006;12-14.
15- Bays H, Ballantyne C. Adiposopathy: why do adiposity and obesity cause metabolic disease? Future Lipidol. 2006;1:389-420.
16- Guilherme A, Virbasius JV, Puri V, Czech MP. Adipocyte dysfunctions linking obesity to insulin resistance and type 2 diabetes. Nat Rev Mol Cell Biol. 2008;9:367-377.
17- Klein S. Obesity as a chronic disease: epidemiology, morbidity, and mortality. http://cme. medscape.com/viewarticle/567413. Accessed November 1, 2012.
18- Aronne LJ, Nelinson DS, Lillo JL. Obesity as a disease state: a new paradigm for diagnosis and treatment. Clin Cornerstone. 2009;9:9-25.
19- Allison DB, Downey M, Atkinson RL, et al. Obesity as a disease: a white paper on evidence and arguments commissioned by the Council of the Obesity Society. Obesity (Silver Spring). 2008;16:1161-1177.
20- Hu L, Huang X, You C, Li J, Hong K, Li P, et al. Prevalence and Risk Factors of Prehypertension and Hypertension in Southern China. PLoS One. 2017; 12(1): e170238 doi: 10.1371/journal.pone.0170238 .
21- Hou X, Lu J, Weng J, Ji L, Shan Z, Liu J, et al. Impact of waist circumference and body mass index on risk of cardiometabolic disorder and cardiovascular disease in Chinese adults: a national diabetes and metabolic disorders survey. PLoS One. 2013; 8(3):e57319
22- Canadian Organ Replacement (CORR) 2001 Annual Report. Ottawa, ON, Canada: Canadian Institute for Health Information; 2001.
23- European Society of Hypertension-European Society of Cardiology Guidelines Committee: Guidelines for the management of arterial hypertension. J Hypertens 2003, 21:1011-1053.
24- American Diabetes Association. Standards of Medical Care in Diabetes 2011.
DIABETES CARE, VOLUME 34, SUPPLEMENT 1, JANUARY 2011
25- Executive summary of the 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). JAMA2001;285(19):2486-97. A.y
26- Ford E.S., Maynard L.M., Li C. Trends in mean waist circumference and abdominal obesity among US adults, 1999–2012. JAMA. 2014;312:1151–1153.
27- Yoo S., Cho H.J., Khang Y.H. General and abdominal obesity in South Korea, 1998-2007: gender and socioeconomic differences. Prev Med. 2010;51:460–465.
28- Lilja M., Eliasson M., Stegmayr B., Olsson T., Soderberg S. Trends in obesity and its distribution: data from the Northern Sweden MONICA Survey, 1986–2004. Obesity (Silver Spring) 2008;16:1120–1128.
29- Welty T.K. Health implications of obesity in American Indians and Alaska Natives. Am J Clin Nutr. 1991;53:1616S–1620S.
30- Must A., Spadano J., Coakley E.H., Field A.E., Colditz G., Dietz W.H. The disease burden associated with overweight and obesity. JAMA. 1999;282:1523–1529.
31- Lee Z.S., Critchley J.A., Chan J.C. Obesity is the key determinant of cardiovascular risk factors in the Hong Kong Chinese population: cross-sectional clinic-based study. Hong Kong Med J. 2000;6:13–23.
32- Hu F.B., Wang B., Chen C. Body mass index and cardiovascular risk factors in a rural Chinese population. Am J Epidemiol. 2000;151:88–97.
33- Ahn S., Zhao H., Smith M.L., Ory M.G., Phillips C.D. BMI and lifestyle changes as correlates to changes in self-reported diagnosis of hypertension among older Chinese adults. J Am Soc Hypertens. 2011;5:21–30.
34- WHO . WHO Expert Committee; 1995. Physical Status: The Use of and Interpretation of Anthropometry.
35- WHO, IASO, IOTF . International Diabetes Institute; Melbourne: 2000. The Asia-Pacific Perspective: Redefining Obesity and its Treatment.
36- Pouliot M.C., Despres J.P., Lemieux S. Waist circumference and abdominal sagittal diameter: best simple anthropometric indexes of abdominal visceral adipose tissue accumulation and related cardiovascular risk in men and women. Am J Cardiol. 1994;73:460–468.
37- Yusuf S., Hawken S., Ounpuu S. Obesity and the risk of myocardial infarction in 27,000 participants from 52 countries: a case-control study. Lancet. 2005;366:1640–1649.
38- Reis J.P., Macera C.A., Araneta M.R., Lindsay S.P., Marshall S.J., Wingard D.L. Comparison of overall obesity and body fat distribution in predicting risk of mortality. Obesity (Silver Spring) 2009;17:1232–1239.
39- Chukwuonye II, Chuku A, Onyeonoro UU, Okpechi IG, Madukwe OO, Umeizudike TI, et al. Prevalence of abdominal obesity in Abia State, Nigeria: results of a population based house-to-house survey. Diabetes Metab Syndr Obes Targets Ther. 2013;6:285–291. doi:10.2147/DMSO.S43545

40- Munyogwa MJ and Mtumwa AH
The Prevalence of Abdominal Obesity and Its Correlates among the Adults in Dodoma Region, Tanzania: A Community-Based Cross-Sectional Study
Adv Med. 2018; 2018: 6123156.
41- Linhares RS, Horta BL, Gigante DP, Dias-da-Costa JS, Olinto MTA. Distribution of general and abdominal obesity in adults in a city in southern Brazil. Cad Saúde Pública. 2012;28(3):438–447. doi:10.1590/S0102-311X2012000300004
42- Misra A, Shivastava U. Obesity and dyslipidemia in South Asians. Nutrients. 2013;5:2708– 2733. doi:10.3390/nu5072708
43- Beltrán-Sánchez H, Harhay MO, Harhay MM, McElligott S. Prevalence and trends of metabolic syndrome in the adult US population, 1999–2010. J Am Coll Cardiol. 2013;62 (8):697–703. doi:10.1016/j.jacc.2013.05.064
44- Barquera S, Campos-Nonato I, Hernández-Barrera L, Pedroza A, Rivera-Dommarco JA. Prevalence of obesity in Mexican adults, 2000–2012. Salud Pública Méx. 2013;55(2):S151–S160
45- Badran M and Laher I Obesity in Arab speaking countries.
Journal of Obesity Volume 2011, Article ID 686430, 9 pages doi:10.1155/2011/686430
46- Hazzaa M. Al-Hazzaa, Nada A. Abahussain, Hana I. Al-Sobayel, Dina M. Qahwaji, Nouf A. Alsulaiman, and Abdulrahman O. Musaiger.
Prevalence of Overweight, Obesity, and Abdominal Obesity among Urban Saudi Adolescents: Gender and Regional Variations.
47- Amole OI, Adesina AS. The prevalence of abdominal obesity and hypertension among adults in Ogbomoso, Nigeria. African journal of primary health care and family medicine 2011, 188
48- Khairun Nisa Berawi, Sutopo Hadi, Nur Indrawati Lipoeto, Irza Wahid and Jamsari; Dyslipedemia Incidents Between General Obesity and Central Obesity of Employees with Obesity at Universities. Lampung biomedical and pharmacological J
Vol. 11(1), 201-207 (2018)
49- Barroso, AT, Marins BL, Alves R. Association of central obesity with the incidence of cardiovascular diseases and risk factors. International journal of cardiovascular science s 2017,30 (5) 416-424
50- Iloh GUP, Ikwudinma AO. Abdominal obesity in adult Nigerian Africans: Prevalence and co-occurrence with cardio-metabolic risk factors in a resource poor setting of a rural hospital in EasternNigeria. AmJHealth Res. 2013;1(3):73–80.
51- LEE Wei-Jei ; CHEN Hsin-Hung ; WEU WANG ; WEI Po-Li ; LIN Ching-Mei ; HUANG Ming-Te. Metabolic syndrome in obese person referred from weight reduction surgery in Taiwan. J Formos Med Assoc 2003; 102; 459-464
52- Xavier HT, Izar MC, Faria Neto JR, Assad MH, Rocha VZ, Sposito AC, et al; Sociedade Brasileira de Cardiologia. [V Brazilian Guidelines on Dyslipidemias and Prevention of Atherosclerosis]. Arq Bras Cardiol. 2013;101(4 Suppl 1):1-20. doi: 105935/abc20135010. [
53- Pimenta AM, Kac Gilberto, Gazzinelli A, Corrêa-Oliveira R, Velásquez- Meléndez G. Association between central obesity, triglycerides and hypertension in a rural area in Brazil. Arq Bras Cardiol. 2008;90(6):386-92. doi: 10.1590/S0066-782X2008000600006
54- Termizy H M, Mafauzy M. Metabolic syndrome and its characteristics among obese patients attending an obesity clinic. 2009. Singapore Med J 2009; 50: 390- 394