The Anthropometric and Biochemical Parameters in Normal Weight-Central Obesity Females

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Abstract

Central obesity is abdominal fat that accumulates and affects the health status. The present study aims to estimate some blood biomarkers among normal-weight females with central obesity and determine the prevalence of central obesity. The method: healthy females with normal body weight and age group of (33-44 y) have participated and their body anthropometric parameters were determined. The Navy formula detected the body composition. Serum glucose, lipid profile, insulin, and cortisol hormone were tested. The results: a high prevalence has been found of the central obesity among females with normal weight (71.098%). Central obesity is characterized by increasing WC, WHR, and WHtR with high-fat percentage and fat mass (40.55%, 25.6 kg) than females without central obesity (31.5%, 19.4 kg, respectively). The central obesity females showed significant elevation in serum glucose 8.937 mmol/L, insulin 177.884 pmol/L, and HOMI 10.216 with dyslipidemia than the ones with the non-central obesity. A high significant cortisol level (29.126 µg/dl) was observed in the normal weight central obesity group. Conclusions: the incidence of central obesity among normal-weight females has been associated with changes in some blood profiles, which may contribute to increasing adiposity-related risk factors and health outcomes and the effect on body shape.

Keywords: *Biochemical parameters , anthropometric, females , obesity*

Introduction

The local body fat distribution revealed an important role in initiating health problems [1]. The distribution of body fat is identified as the android shape (central) and ganoid shape (peripheral) [2]. Central obesity is one of the regional fat accumulations that contribute mainly to metabolic abnormalities like hypertension, diabetes type2, and cardiovascular diseases [3] by promoting mediating factors including inflammations, insulin resistance, and dyslipidemia [4].

Central (abdominal) obesity was observed in subjects with normal body weight (BMI < 25 Kg/m²) [5], which resulted from many factors, mainly diet type and lifestyle modifications [6]. The central fat is characterized by excessive abdominal fat deposit even among the subjects with normal weight [7]. The abdominal fat has been considered as the optimal criterion for the prediction of the fitness and for the metabolic syndrome incidence, especially visceral fat type [8, 9].

Abdominal obesity is gender-specific obesity [10] that is affected by the age [11]; in the females, abdominal obesity showed a specific android pattern (apple shape) with increasing WC [11, 12].

The modern direct measurement for abdominal obesity is magnetic resonance imaging or computer tomography scan [13]. Since they are expensive, not available, and limited in use, the simple alternative is using the anthropometric indicators including WC, WHR, and WHTR [7, 14]. These indicators were

suggested by many epidemiological studies and health organizations [15, 16].

The most widely used obesity index measurement is the BMI method. Still, with some limitations, represented in the fact that it does not provide the pattern of body fat distribution, therefore the combination between abdominal measurements with BMI was recommended [17]. In general, the regional fat depots were assessed among overweight and obese people and not considered in normal-weight subjects, which is an important indicator for the health condition. This study aims to determine the prevalence of central obesity among females with normal body weight, assess the risk factors associated with abdominal fat deposits through blood biomarkers, and detect the correlation between WC and some blood parameters.

Materials and Methods

The population sample of the study consists of healthy females (n=173) with normal body weight (BMI≥25 kg/m^2) and age (33-44) years. The subjects were divided according to age into two age groups, the first age group: (33- 38 years) and the second age group (39- 44 years). The data has been collected from the participants according to the protocol of the ethical committee of Biology Department-Science College-Basrah University. The data has been collected firstly by questioner forms and then the anthropometric parameters and blood samples have been taken. Questioner forms asked all participants about social status, diet type, lifestyle, physical activity, smoking, drugs, hormonal therapy, allergy condition, and polycystic ovaries syndrome. The exclusion criteria were pregnancy, athletic females, hormonal therapy, females with diseases, and medications. The study was carried out from July 2019 to February 2021.

The anthropometric measurements:

The body mass index (BMI) is measured by weight kg/height m^2 [18]. Waist circumference (WC) was measured at the umbilicus region (cm), hip circumference (HC) was measured at iliac ridge over the buttocks (cm), the neck circumference at the neck place. All measurements were done by plastic strip [19]. The WHR ratio by WC/HC, the WHtR by WC/height. The central obesity was determined according to WC, WHR and WHtR: WC ≥ 80 cm, WHR ≥ 0.85, and WHTR > 0.50 [20]. Basing on questioners and medical history, all participants have been considered in good health. The participants were classified to normal body weight with central obesity and normal body weight without central obesity. The percentage of body fat (%BF) has been measured according to the formula of American Navy: females= 163.205 × log10 (waist + hip - neck) - 97.684 ×log10 (height) - 78.387 [21]. Using a digital scale to measure the weight. The blood pressure was taken in a sitting position.

The Blood Sample

The venous blood sample (5ml) from all participants was drawn after overnight fasting (at 8-10 Am). Serum specimens were collected in a gel tube after blood clotting for 15 minutes at room temperature and then centrifuged at 3000 rpm for 10 minutes. The serum was stored at deep freeze -70 °C [22].

The serum assays:

Serum glucose and lipid profiles (triglycerides TG, total cholesterol T-ch, and high-density lipoprotein HDL) were measured by using the commercial kits (Biolab, France). According to the formula, low-density lipoprotein (LDL) is Tch- HDL- TG/5 [23]. The insulin has been measured by Elisa kit (Abcam, USA) and cortisol by Elisa Kit (Crystal Chem, USA). The homeostatic index of insulin resistance (HOMI) by the formula: [glucose (mmol/L) × insulin (pmol/L)] ÷ 155 [24].

The Statistical Analysis

The data were analyzed by ANOVA test by SPSS version 22. T-Test was used for the two groups, Chi-square was used for obtaining values of percentage, and a general linear model was used for groups with many factors. The correlation by bivariate analysis.

The data has been expressed by mean ± standard deviation SD.

Results

The female participants of this study had a normal body mass index (23-24 kg/m^2) (Table 1). A group of females revealed a significant ($p \leq 0.05$) increase in WC (94.189 cm), WHR (0.908), and WHtR (0.579) than other female participants, therefore, they have been divided to normal BMI with central obesity and normal BMI without central obesity. The female group without central obesity showed normal ranges in WC (78.419 cm), WHR (0.778), and WHtR (0.479) values.

According to anthropometric parameters, the healthy females with normal body weight showed a high prevalence of central obesity 123 (71.098 %), while the females without central obesity reveal less occurrence 50 (28.901%), table (2).

The Navy formula (table 1) revealed that the females with normal weight and central obesity are considered obese as they showed higher body fat percentage (% BF) and higher fat mass 40.55%, 25.6 kg, respectively with significantly ($p \leq 0.05$) compared to the non-central obesity females group (31.5%, 19.4 kg respectively). The lean body mass that has been displayed by this formula showed that the non-central obesity females had more lean mass, 42.1 kg, than the females with central obesity 37.4 kg.

The systolic and diastolic pressure were significantly ($p \leq 0.05$) elevated in normal weight-central obesity group 123.927 mmHg and 80.893 mmHg respectively compared to normal weight without central obesity 119.371 mmHg and 77.440 mmHg.

According to age groups (table 3), in the central obesity group, the second age group, 39-44y of central obesity displayed a higher WC (95.588), WHR (0.917), WHtR (0.585) with significantly ($p \leq 0.05$) than the first age group (33-38 y). The Navy formula also displays insignificant differences in the percentage of body fat, fat and lean mass between the two age groups. There were significant differences ($p \leq 0.05$) between the systolic and diastolic pressure between the two age groups.

In normal weight without central obesity, the second age group showed higher significance ($p \leq 0.05$) values in the ratio of WC, WHR, and WHtR than the first age group. According to the Navy formula, the second age group considers obese by high significant body fat percentage and fat mass values than the first age group, which have been considered with average body fat. No significance was observed in lean mass between the two age groups. The systolic and diastolic pressure showed insignificant differences.

The biochemical parameter between the two main groups (table 4) showed significant ($P \leq 0.005$) elevation in serum lipid profiles of central obese females TG, T-ch, LDL: 163.420 mg/dl, 177.656 mg/dl, 112.7 mg/dl, and a decrease in the HDL 47.233 mg/dl compared to non-central obesity.

There was a significant ($p \leq 0.05$) increase in serum glucose and insulin levels in females with normal weight and central obesity group 8.937 mmol/L, 177.884 pmol/L respectively compared to normal weight without central obesity 8.079, 171.464. Therefore, the homeostatic index of insulin resistance was significantly higher in the central obesity group 10.216 than the non-central obesity group 8.937.

The results observed an increased level of serum cortisol in normal weight-central obesity females 29.126 µg/dl with significant difference ($p \leq 0.05$) than the non-central obesity females 21.621 µg/dl.

From table (5), according to age groups, in normal weight-central obesity females, it was observed that the second age group (39-44 y) showed significant ($p \leq 0.05$) elevation in glucose, TG, T-ch, and LDL levels compared with the second age group. Also, there was an increase in the insulin level 179.629 pmol/L and HOMI (10.316) with significant differences compared to the first age group 176.140 pmol/L and 10.116,

respectively. The serum cortisol level displayed significant elevation in the second age group 30.531 µg/dl compared to the first age group 27.721 µg/dl.

There were significant differences in normal weight without central obesity ($p \leq 0.05$) in the levels of glucose, TG, insulin, and HOMIR between the two age groups. A significant elevation in these parameters characterizes the second age group. No significant differences were observed in the levels of T-ch, HDL, LDL, and cortisol between the two age groups.

The results of the bivariate correlation analysis (table 6) showed that BMI had a positive correlation with fat mass percentage. The WC displayed a positive correlation with WHR and serum cortisol level. There was a positive correlation between WHR and HOMIR and between serum fasting glucose and TG level.

Table 1: The Anthropometric parameters in normal weight females with and without central obesity.

Parameter	Normal BMI with central obesity (n=123)	Normal BMI without central obesity (50)	P value (Significant at $p \leq 0.05$)
Weight	63.367±1.501	61.838±1.643	0.532
Height	162.478±1.731	163.325±1.309	0.231
BMI	24.004±1.567	23.183±1.537	0.819
WC	94.189±2.773	78.419±1.842	0.003
HC	103.680±5.171	100.737±3.192	0.420
WHR	0.908±0.058	0.778±0.087	0.001
WHtR	0.579±0.093	0.479±0.097	0.001
NC	33.810±1.218	33.362±1.063	0.530
% BF	40.55±1.040	31.5±1.624	0.000
Fat mass (kg)	25.6±1.537	19.4±1.857	0.000
Lean mass (kg)	37.4±1.978	42.1±1.607	0.001
Body category	obese	average	-
SBP mmHg.	123.927±3.863	119.371±1.118	0.032
DBP mmHg.	80.893±2.674	77.440±1.311	0.044

Table 2: the prevalence of central obesity among normal weight females in the study population.

	Normal BMI with central obesity	Normal BMI without central obesity
The prevalence	123 (71.098 %)	50 (28.901 %)

Chi-Square=17.640, Sig= 0.000

Table 3: the anthropometric parameters in normal weight females with and without central obesity according to each groups.

Parameter	Normal BMI with central obesity (n=123)			Normal BMI without central obesity (50)		
	Age group1 33-38 y, n=53	Age group 2 39-44 y, n=70	P value (Significant at $p \leq 0.05$)	Age group1 33-38 y, n=32	Age group 2 39-44 y, n=18	P value (Significant at $p \leq 0.05$)
Weight	62.554±1.499	64.181±1.545	0.840	60.498±1.500	63.179±1.533	0.790
Height	161.751±1.520	163.205±1.698	0.477	162.293±1.714	164.358±1.703	0.500
BMI	23.912±1.614	24.096±1.102	0.693	22.976±1.679	23.390±1.100	0.661
WC	92.791±2.527	95.588±3.502	0.033	77.377±3.505	79.462±3.548	0.050
HC	103.202±1.528	104.159±2.485	0.525	99.872±.3108	101.603±3.503	0.417
WHR	0.899±0.010	0.917±0.010	0.010	0.774±0.050	0.782±0.064	0.039
WHtR	0.573±0.068	0.585±0.076	0.040	0.476±0.072	0.483±0.051	0.041
NC	32.901±0.551	34.720±0.863	0.700	33.451±0.742	33.274±0.670	0.653
% BF	40.5±0.400	40.6±0.503	0.680	30.8±0.115	32.2±0.251	0.039
FM (kg)	25.1±0.100	26.1±0.129	0.511	18.5±0.151	20.3±0.208	0.045
LM (kg)	36.9±0.476	37.9±0.208	0.583	41.5±0.264	42.7±0.378	0.733
Body category	obese	obese	-	average	obese	-
SBP mmHg	121.424±5.532	126.430±5.389	0.033	118.390±6.190	120.352±6.144	0.740
DBP mmHg	79.113±3.706	82.673±3.487	0.046	78.128±2.594	76.752±2.731	0.668

Table 4: the serum assays in normal weight females with and without central obesity.

Parameter	Normal BMI with central obesity (n=123)	Normal BMI without central obesity (50)	P value (Significant at p ≤ 0.05)
Glucose mmol/L	8.937±1.837	8.079±1.508	0.000
TG mg/dl	163.420±2.780	145.933±1.678	0.001
T-ch mg/dl	177.656±1.247	173.830±1.841	0.000
HDL mg/dl	47.233±1.231	51.44±1.088	0.003
LDL mg/dl	112.7±1.830	106.25±1.390	0.000
Insulin poml/L	177.884±1.982	171.464±1.369	0.040
HOMI	10.216±0.215	8.937±0.151	0.001
Cortisol µg/dl	29.126±0.818	21.621±0.872	0.000

Table 5: the serum assays in normal weight females with and without central obesity according to age groups.

Parameter	Normal BMI with central obesity (n=123)			Normal BMI without central obesity (50)		
	Age group1 33-38 y, n=53	Age group 2 39-44 y, n=70	P value (Significant at p ≤ 0.05)	Age group1 33-38 y, n=32	Age group 2 39-44 y, n=18	P value (Significant at p ≤ 0.05)
Glucose mmol/L	8.902±1.115	8.972±1.321	0.011	8.019±1.141	8.139±1.307	0.007
TG mg/dl	160.310±3.615	166.530±4.586	0.012	141.651±2.154	150.215±2.577	0.031
T-ch mg/dl	176.802±3.527	178.510±3.577	0.051	173.230±1.577	174.431±1.386	0.341
HDL mg/dl	48.768±2.358	45.699±1.732	0.030	50.560±2.886	52.320±2.886	0.602
LDL mg/dl	110.4±2.577	115±2.309	0.001	106.6±1.970	105.9±2.081	0.719
Insulin pmol/L	176.140±2.732	179.629±5.196	0.032	170.301±3.114	172.628±2.154	0.042
HOMI	10.116±0.166	10.316±0.329	0.045	8.810±0.240	9.064±0.121	0.002
Cortisol µg/dl	27.721±1.577	30.531±1.100	0.003	20.881±1.154	22.362±1.732	0.320

Table 6: the observed significant correlations among the variables in the study.

Variables	Pearson Correlation Sig. (p ≤ 0.05)
BMI-Fat mass	.917(*) .028
WC-WHR	.958(*) .010
WC-Cortisol	.906(*) .034
WHR-HOMIR	.907(*) .034
Glucose -TG	.942(*) .017

Discussion

The general anthropometric parameter to diagnose obesity in the population uses the body mass index BMI [18]. In this study, we found that the healthy females with normal body weight showed central obesity by increasing WC, WHR, and WHtR despite lowering their BMI (18.5-24.9 kg/m^2). The high prevalence of central obesity among the study participants suggests the importance of detecting the WC, WHR, and WHtR in normal-weight subjects to determine regional obesity as critical measurements for body fat distributions [25].

In this study, we compared the results of the Navy formula with the WC, WHR, and WHtR and found that the normal weight females with central obesity showed more fat percentage in both age groups. Simultaneously, the normal weight females without central obesity also showed a fat ratio and were considered obese in older age (39-43y). These results indicate that the Navy formula was adequate to determine body fitness by including all body anthropometric parameters in addition to sex and age. Also, this formula was effective in expressing fat accumulation in the normal body weight. The Navy formula is an adequate and certain formula for the detection of the body composition with similar results as using the bioelectrical analysis [26, 27], and suitable for use any time even after the physical activity [28], it has also been considered as a screening tool for body fitness and obesity complications [29].

The normal-weight females without central obesity showed high-fat percentage, especially in the second age, this may related that the Navy formula reflect the fitness degree of these participants. In our study, all participants showed a sedentary lifestyle. In the central obesity females, the formula showed a higher fat percentage as expected with increasing the abdominal fat deposit in this group.

The total body mass, including the fat and non-fat mass, was expressed by BMI. In contrast, the WC expressed only the fat accumulation in the abdominal region and HC revealed the fat tissue in peripheral body parts [20]. In normal BMI with central obesity, the increase in WC and decrease in the HC may decrease fat deposits in hips and legs [6].

In this study, the females with central obesity showed elevation in lipid profile, glucose, insulin, and HOMI, indicating that the central fat accumulation causes a disturbance in lipid and glucose metabolism and developing the risk factors leading to glucose intolerance, dyslipidemia, hypertriglyceridemia, insulin resistance, and then systematic inflammation [7].

The lipolytic activity resulting from lipids disturbance, causing high TG production and transferring to the liver, which in turn increases VLDL synthesis and TG elevation in the blood circulation, enhances the exchange of the cholesterol esters and TG hydrolyzing, leading to lower HDL levels [30]. High TG production in liver during hyperglycemia decreases the insulin clearance and causes hyperinsulinemia [31].

Some previous studies revealed the importance of determining the central obesity among people with normal weight [7, 32] as it is pointed to developed morbidities like that in obese people with central obesity [7, 33]. These because of a strong association between the central obesity and CVD, regardless of BMI [34]; also, the WC and WHR are more correlated with the risk factors of metabolic problems beyond the BMI [34, 35].

The excessive visceral fat depots in the abdominal region may cause metabolic alternations, including dyslipidemia and hyperinsulimia that elevated risk factors of cardiovascular diseases CVD [36, 37, 38]. The presence of hypertension and diabetes type 2 in normal-weight central obese individuals leading to develop cardiovascular diseases (CVD) was more than the individuals without the central obesity [39, 40].

In this study, the elevated systolic and diastolic blood pressure in normal–weight–central obesity females may be explained by the fact that the ratio of body adiposity was an independent risk factor for hypertension [41]. The risk factor of hypertension was 63% in normal-weight Malaysian women [42]. The normal weight subjects with high-fat percentages showed elevated blood pressure and were more expected to develop CVD [43].

The study results showed that the normal BMI with central obesity has high serum cortisol compared to female without central obesity, this may be related to increased visceral fat accumulation [44], or the elevated cortisol in blood circulation that caused abdominal obesity [45]. The visceral adipose tissues are supplied with more blood vessels than the peripheral adipose tissues and with more glucocorticoid receptors; therefore, the visceral fat was more sensitive to TG and glucocorticoids. The glucocorticoids, like cortisol stimulate proliferation and differentiation of human adipose tissue that causes a redistribution of fat cells from peripheral to central accumulation [46].

Conclusions

Inclusion of central adiposity measurements and body fat percentage with BMI are important parameters for

detecting body fat deposition and body fitness even in the absence of whole-body obesity. It is important to consider the metabolic alternations associated with central obesity in normal BMI to avoid the risk factors of metabolic complications. Considering normal weight subjects as a control in clinical studies based only on BMI and regardless the abdominal obesity will lead to misclassification and underestimation of blood metabolites like lipid profile and hormones that will affect the study results.

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Conflict of Interest: None to declare.

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