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# Body Fat Distribution and the Levels of Some Biochemical, Hormonal and Inflammatory Biomarkers in Obese Healthy Women

**Sanaa Jameel Thamer***
University of Basrah, College of Sciences, Department of Biology, Iraq
Corresponding author email: sanaa.thamer@uobasrah.edu.iq.

**ABSTRACT**

The metabolic alternations related to obesity are a serious risk factors that leads to many health problems. The aim of the study is to estimate some biochemical and hormonal parameters in the blood of obese females with different distribution of body fat.Methods:premenopausal healthy females (n=148) that have various body weight and aged (39-43 years), which were assessed their obesity by BMI and the regional body fat by the WC, WHR and WHtR parameters. The Navy seal formula were used to detect body composition. Blood biochemical, insulin, cortisol, thyroid hormones, total testosterone, visfatin, IL8 and CRP were tested in the serum. Results: according to WC and WHR, the obese females (n=83) revealed two types of obesity, the central and peripheral obesity. The Navy formula showed high significant fat ratio and fat mass particularly in central obesity females. The serum of obese category showed significant increasing in fasting glucose, insulin, HOMIR with dyslipidemia. The serum cortisol, total testosterone, visfatin, IL8 and CRPwere also elevated in obese females with significant differences in central obesity group than the peripheral obesity group. Compared to normal weight females, the obese females revealed significantincreasing in TSH and FT3 levels while the FT4 was insignificant. Conclusion: the obesity in healthy females especially the central obesity type was associated with alternations in serum profiles of somehormonal and inflammatory biomarkers.

**Keywords**Body Fat, metabolism, CRP.

## Introduction

Obesity is the most common metabolic disorders in the word, especially in developing countries [1]. The overweight or body fat depots consider the first marker of health problems including hypertension, diabetes and cardiovascular disease [2]. The body fat is classified into central (android shape) and peripheral fat (ganoid shape) [3]. The central fat is resultsfrom fat deposit in abdominal region, while the deposit around the hips and limbs results in peripheral adiposity [4].The abdominal fat is distributing into two lines, the subcutaneous and visceral fats, the visceral obesity plays important role as an endocrine gland that releasing many adipocytokines, those effecting on glucose and lipid metabolism and contributed to several metabolic dysfunctions, involving hyperinsulinmea, dyslipidemia[5]. The obesity in both women and men was observed to associate with many hormonal abnormalities, in women, the central (abdominal) obesity is correlated directly with diabetes and insulin resistance [6]. The ratio of urinary cortisone/ cortisol is increased in women with abdominal obesity [7] and the high cortisol level was correlated with central body fat [8]. The abnormalities in sex hormones are associated with obesity in female by decreasing in estradiol and follicle stimulating hormones [9]. The functions of thyroid gland in obesity is affecting on weight gain and on development of metabolic risk factors [10].
The obesity can estimated by body mass index (BMI) that widely used as anthropometric parameter for general adiposity but not identify the body composition[11]. The indirect measurement of visceral fat is using waist circumference WC and waist to hip ratio WHR[12], while the peripheral fat distribution measured by hip or thigh circumference [13]. The previous information can elicit that the BMI cannot expressed the body fat mass, also the endocrinal and metabolic alternations that associateor as resulted from obesity is a controversial matter, therefore the aim of the study is to estimate some blood biomarkers in obese healthy females with different body fat deposition, also to explore the correlation between the distribution type of body adiposity with the tested blood parameters.

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## Materials and methods

In this study the participants consist of premenopausal females (n=148) with aged (39-43 years) and various body weight. The participants were dividing according to body mass index (BMI) to normal body weight (BMI =18.5-24.9 kg/m$^2$, n=65) and obese (BMI ≥ 25 kg/m$^2$, n=83). The obese females were classified to central obesity (n=55) and peripheral obesity (n=28) according to WC and WHR. The study protocol was following according to the ethical committal of Biology department - Science-collage. Basing on questioners, physical examination and medical history, the participants consider with good health. The questioners included (social status, healthy status, diseases, diet type, allergy, smoking, drugs, hormonal therapy,polycystic ovary syndrome, life style,physical activity).The study was carried from May 2019 to January 2021.

## Athropometric measurements

The general obesity was measured using the common formula of body mass index BMI= body weight (kg) / height$^2$ (m) [14]. Waist circumference (WC) was measure at the umbilicus region (cm), the hip circumference (HC) was measure at iliac ridge over the buttocks (cm), the neck circumference at the neck place (cm). All measured by plastic strip [15]. The WHR ratio by WC/HC, the WHtR ratio by WC/height. The central obesity was determined according to WC≥ 80 cm, WHR ≥ 0.85 and a WHTR > 0.50; the peripheral obesity when WHR< 0.85[16]. The percentage of body fat according to formula of Amrican Navy:females= 163.205 × log10 (waist + hip - neck) - 97.684 ×log10 (height) - 78.387 [17]. Using digital scale to measure the weight. The blood pressure was taken in sitting position.

## Blood sample

The venous blood samples (5ml) of the participants were taken in the morning after fasting (12 hours, at 8-10 Am).Blood specimens were left at room temperature to clot and get serum after centrifugation (3000rpm, 10 minutes). Serum was stored at -70◦c in deep freeze for biochemical and hormonal test[18].

## Serum assays

Serum glucose and lipid profiles (triglycerides TG, total cholesterol T-ch, high density lipoprotein HDL and very low density lipoprotein VLDL) were measured by a commercial kits (Biolab, France). LDL according to formula: Tch-HDL- TG/5 [19].The insulin, thyroid stimulating hormone TSH, total testosterone and cortisol were measured by Elisa kits using COBAS 411e automated analyzer (Roche Diagnostics, Germany). Free triiodothyronine FT3 (Calbiotech, USA Elisa Kit), free thyroxine FT4 (Calbiotech, USA Elisa Kit), visfatin (abcam, USA Elisa Kit), interleukin 8 (Bio-Vender, UK Elisa Kit), C-reactive protein (Enzo, USA Elisa Kit). The homeostatic index of insulin resistance (HOM-IR) by the formula: [glucose (mmol/l) × insulin (pmol/l)] ÷ 155 [20].

## Statistical Analysis

The data were analyzed by ANOVA usingt- test. The correlation by bivariate analysis(SPSS version 22). The least significant difference ($p \leq 0.05$) was used to compare the means. The data expressed by means ± standard deviation SD.

## Results

A according to BMI (table 1), the females participants were dividing into obese group and normal weight group. The using of anthropometric parameters WC,WHR and WHtR showed significant ($p \leq 0.05$) increasing in these parameters in obesegroup 105.257, 0.858, 0.641respectively compared to normal levels in normal weight group 74.360, 0.739, 0.452 respectively.

The Navy formula revealed that obese group showed higher body fat percentage (%BF) 50.550 % and higher fat mass 47.5 kg with significant ($p \leq 0.05$) than the normal weight group 29.23%, 18.7 kg respectively (table 1). The lean mass between the two groups showed insignificant differences. The results of Navy formula showed that the normal body weight group had an average body fat.

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The obese group display a significant elevation in blood pressure130.119/85.268 mmHg compared to blood pressure in normal body weight group 118.5/78.901 mmHg.

The obese group showed two type of regional obesity,the central and peripheral obesity(table 2). The central obesity group characterized by significant ($p \leq 0.05$) increasing in WHR and WHtR 0.921, 0.673respectively, while in peripheral obesity group the values of WHR and WHtR were 0.795, 0.609 respectively.According to Navy formula the central obesity group have higher fat percentage 51.6% and fat mass 49.5 kg with significant ($p \leq 0.05$) than the peripheral obesity group49.5 % and 45.5 kg respectively. The lean body mass showed insignificant between the two groups 46.5 kg.

There was insignificant differences in systolic and diastolic pressure between the central and peripheral obesity.

The increasing of body weight in obese female group showed alternations in serum biochemical and hormonal parameters compared to normal weight female group(table 3).In obese group, there were significant ($p \leq 0.05$) elevation in serum glucose 9.375 mmo/l and lipid profile TG 134.421 mg/dl, T-ch 196.47mg/dl, LDL 128 mg/dl, VLDL 37.153 mg/dl, while there was a significant decreasing in HDL level 41.244 mg/dl compared to normal weight group.

Comparing to normal weight category, the obese females display significant ($p \leq 0.05$) increasing in serum level of TSH 2.231 µIU/ml and FT3 3.090 pg/ml, while showed insignificant differences in serum level of TF4 (1.807 ng/dl).

The obese group showed significant ($p \leq 0.05$) elevation in serum cortisol, insulin and total testosterone hormones 883.740 nmol/l, 189.257 pmol/l, 0.578 ng/mlrespectively than the normal weight female group. The mean values of HOMIR, vesfatin, IL8 and CRP were significantly higher in obese group when compared to normal weight group (obese: 11.451, 35.721 ng/ml, 4.100 pg/ml, and 180.501ng/ml respectively).

In comparison between the central and peripheral obesitygroups(table 4), the central obesity group revealed significant ($p \leq 0.05$) elevation in serum levels of glucose, lipid profile compared to the peripheral obesity group.

The female with central obesity showed significant ($p \leq 0.05$) increasing in the levels of the tested hormones compared to females with peripheral obesity. The levels of FT3,cortisol and insulin were significantly high in central obesity (3.367 pg/ml, 947.192 nmol/l, 192.412 pmol/l respectively).

The concentrations of visfatin, IL8 and CRP were increased significantly ($p \leq 0.05$) in central obesity (41.959 ng/ml, 4.768 pg/ml, 197.773 ng/ml respectively) compared to peripheral obesity (29.483 ng/ml, 3.432 pg/ml, 163.229 ng/ml respectively).

The elevation in serum glucose and insulin in central obesity group reflected in high level of HOMIR(11.855) with significantly ($p \leq 0.05$) than the peripheral obesity group (11.047).

The serum level of TSH was elevated in both central and peripheral obesitywith significant differences ($p \leq 0.05$) between them (2.446µIU/ml, 2.016µIU/ml respectively). There were insignificant differences in serum levels of HDL, FT4and total testosterone between the two groups.

The bivariate correlation analysis (table 5) display the significant ($p \leq 0.05$) correlations among the tested variables. In central obesitygroup, the BMI correlated positively with VLDL and HOMIR. The WC have positive correlation with serum glucose level, while the WHR correlated positively with serum level of IL8. The serum level of HDL revealed negative correlation with cortisol and CRP, while it showed positive correlation

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with total testosterone level. There was a positive correlation between TSH and FT3. The serum visfatin level correlated with the level of CRP in positive correlation.

In peripheral obesitygroup (table 5), there was a positive correlation between WC and serum glucose level. The WHR correlated negatively with serum TSH and positively with HOMIR. There was a negative correlation between level of HDL and level of IL8. The serum level of TSH correlated positively with both FT4 and HOMIR which they showed also a positive correlation between them. There was a positive correlation between serum total testosterone and CRP level.

**Table 1.** Anthropometric parameters in normal weight and obese females.

| Parameters | Control group Normal weight (n=65) Mean± SD | Obese group (n=83) Mean± SD | P value Sig. $p \leq 0.05$ |
|---|---|---|---|
| Weight kg | 64.962±12.890 | 94.359±18.321 | 0.000 |
| Height cm | 164.311±11.965 | 163.966±14.510 | 0.670 |
| BMI kg/m$^2$ | 23.75±2.890 | 35.169±1.582 | 0.000 |
| WC cm | 74.360±13.533 | 105.257±19.859 | 0.003 |
| HC cm | 100.523±8.561 | 122.802±11.303 | 0.025 |
| WHR | 0.739±0.011 | 0.858±0.051 | 0.012 |
| WHtR | 0.452±0.005 | 0.641±0.004 | 0.043 |
| TC cm | 54.392±4.011 | 91.272±6.783 | 0.001 |
| NC cm | 33.524±3.920 | 39.401±1.895 | 0.032 |
| % BF | 29.23±1.128 | 50.550±3.502 | 0.013 |
| Body Fat category | average | obese | - |
| Fat mass kg | 18.7±1.468 | 47.5±1.930 | 0.000 |
| Lean mass kg | 45.3±1.966 | 46.5±1.825 | 0.241 |
| SBP mmHg. | 118.500±6.713 | 130.119±9.545 | 0.010 |
| DBP mmHg. | 78.901±7.111 | 85.268±8.601 | 0.000 |

**Table 2**. Anthropometric parameters in obese females with central and peripheral obesity.

| Parameters | Central obesity (n=55) Mean± SD | peripheral obesity (n=28) Mean± SD | P value Sig. $p \leq 0.05$ |
|---|---|---|---|
| Weight kg | 96.703±4.098 | 92.015±2.560 | 0.033 |
| Height cm | 164.521±0.219 | 163.411±0.303 | 0.621 |
| BMI kg/m$^2$ | 35.701±.587 | 34.638±.733 | 0.372 |
| WC cm | 110.853±1.025 | 99.661±1.002 | 0.033 |
| HC cm | 120.252±11.511 | 125.351±7.701 | 0.048 |
| WHR | 0.921±.005 | 0.795±.008 | 0.022 |
| WHtR | 0.673±.009 | 0.609±.007 | 0.027 |
| TC cm | 82.024±1.515 | 100.520±1.616 | 0.034 |
| NC cm | 39.271±0.658 | 39.532±0.721 | 0.541 |
| % BF | 51.6±.622 | 49.5±.756 | 0.046 |
| Body Fat category | obese | obese | - |
| Fat mass kg | 49.5±0.128 | 45.5±0.321 | 0.0301 |
| Lean mass kg | 46.5±0.011 | 46.5±0.021 | 0.830 |
| SBP mmHg. | 130.298±0.199 | 129.940±0.138 | 0.590 |

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| | | | |
|---|---|---|---|
| DBP mmHg. | 85.407±0.311 | 85.129±0.660 | 0.637 |

**Table 3.** Serum biomarkers in normal weight and obese females.

| Parameters | Control group (n=65) Normal weight Mean± SD | Obese group (n=83) Mean± SD | P value Sig. $p \leq 0.05$ |
|---|---|---|---|
| Glucose mmol/l | 8.20±0.387 | 9.375±0.715 | 0.014 |
| Triglycerides mg/dl | 81.021±7.124 | 134.421±7.333 | 0.004 |
| Total cholesterol mg/dl | 174.311±10.238 | 196.47±9.131 | 0.011 |
| HDL mg/dl | 49.560±3.751 | 41.244±6.620 | 0.039 |
| LDL mg/dl | 109.0±5.140 | 128±7.894 | 0.021 |
| VLDL mg/dl | 26.590±0.330 | 37.153±1.822 | 0.005 |
| TSH µIU/ml | 1.679±0.065 | 2.231±0.061 | 0.044 |
| FT3 pg/ml | 3.010±0.007 | 3.090±0.008 | 0.048 |
| FT4 ng/dl | 1.810±0.011 | 1.807±0.007 | 0.101 |
| Cortisol nmol/l | 623.311±1.011 | 883.740±0.912 | 0.040 |
| Insulin pmol/l | 172.550±10.204 | 189.257±13.716 | 0.030 |
| HOMI-IR | 9.128±0.086 | 11.451±0.086 | 0.048 |
| Total testosterone ng/ml | 0.355±0.018 | 0.578±0.083 | 0.037 |
| Visfatin ng/ml | 14.906±0.036 | 35.721±0.187 | 0.028 |
| IL8 pg/ml | 1.874±0.015 | 4.100±0.516 | 0.034 |
| CRP ng/ml | 120.791±0.011 | 180.501±0.007 | 0.029 |

**Table 4.** Serum biomarkers in obese females with central and peripheral obesity.

| Parameters | Central obesity (n=55) Mean± SD | Peripheral obesity (n=28) Mean± SD | P value Sig. $p \leq 0.05$ |
|---|---|---|---|
| Glucose mmol/l | 9.550±0.040 | 9.201±0.228 | 0.010 |
| Triglycerides mg/dl | 149.321±14.756 | 119.521±11.166 | 0.044 |
| Total cholesterol mg/dl | 212.011±17.908 | 180.929±18.304 | 0.035 |
| HDL mg/dl | 40.172±4.906 | 42.317±5.422 | 0.257 |
| LDL mg/dl | 142.0±4.707 | 114.0±4.483 | 0.021 |
| VLDL mg/dl | 39.395±1.873 | 34.911±1.974 | 0.011 |
| TSH µIU/ml | 2.446±0.008 | 2.016±0.004 | 0.029 |
| FT3 pg/ml | 3.367±0.039 | 2.813±0.015 | 0.048 |
| FT4 ng/dl | 2.131±0.037 | 1.483±0.051 | 0.767 |
| Cortisol nmol/l | 947.192±0.015 | 820.288±0.444 | 0.035 |
| Insulin pmol/l | 192.412±9.017 | 186.102±10.273 | 0.019 |
| HOMI-IR | 11.855±0.020 | 11.047±0.022 | 0.009 |
| Total testosterone ng/ml | 0.606±0.037 | 0.550±0.356 | 0.452 |
| Visfatin ng/ml | 41.959±0.317 | 29.483±0.044 | 0.020 |
| IL8 pg/ml | 4.768±0.012 | 3.432±0.058 | 0.012 |
| CRP ng/ml | 197.773±0.002 | 163.229±0.008 | 0.025 |

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**Table 5.** Observed significant correlations among variables in females with central and peripheral obesity.

| The central obesity | | The peripheral obesity | |
|---|---|---|---|
| Variables | Pearson Correlation Sig. $p \leq 0.05$ | Variables | Pearson Correlation Sig. $p \leq 0.05$ |
| BMI-VLDL | .909(*) .033 | WC-Glucose | .963(**) .008 |
| BMI-HOMIR | .918(*) .028 | WHR-TSH | -.902(*) .037 |
| WC-Glucose | 981(**) .003 | WHR-HOMIR | .956(*) .011 |
| WHR-IL8 | .923(*) .025 | HDL-IL8 | -.880(*) .049 |
| HDL-Cortisol | -.969(**) .006 | TSH-FT4 | .925(*) .024 |
| HDL-Testosterone | .985(**) .002 | TSH-HOMIR | .947(*) .014 |
| HDL-CRP | -.913(*) .030 | FT4-HOMIR | .903(*) .036 |
| TSH-FT3 | .967(**) .007 | Testosterone-CRP | .883(*) .047 |
| Visfatin-CRP | .973(**) .005 | - | - |

## Discussion

The identifying obesity in population is an important matter to estimate the metabolic syndrome associated with fat deposit. The indirect method to evaluate the total body fat isusing different anthropometric parameters [21]. Some digital or radial techniques were developed to determine the body composition, but characterized with high cost and show limitations in availability and usage [22]. According to these restrictions, we used in this study various anthropometric parameters in addition to BMI to classify obesity in health females with aged 39-43 years. The general diagnosis of obesity in individual is using the BMI, thisparameter is establish the popular obesity but it consider useless to reveal the body fat distribution ,therefore adding another parameters including WC,WHR and WHtR is useful to determine the regional obesity. In the current study, the group of obese participant showed a central obesity (WHR≥ 0.85) that characterized by excessive fat depots in the abdominal area, while the other group of obese participants showed peripheral obesity (WHR< 0.85) that distinguish by excessive fat accumulation around the hip and the thighs. The measuring of WC consider a good index for central obesity detection, while the WHR and WHtR are essential measurements with BMI [15]. The premenopausal women in middle age showed higher visceral fat deposition compared to young women [23]. The small muscle mass in the legs was associated with high ratio of WHR [24].

In this study we determined the percentage of body fat by using a formula of American Navy.This formula includesex, age and neck circumference in addition to all other anthropometric parameters for measurement.In this study, the formula results show that the obese healthy females group have a high fat percentage, but their lean mass was the same to the lean massof normal weight females, therefore this formula indicate that the obesity in obese group related to high percentage of fat and the weight again is result from fat deposit. In normal body weight group in spite of normal levels of all anthropometric measurement, the group showed mild percentage of body fat that classified under average fat.From these results we can give an impression of the importance of this formula to detect body fat percentage and body fitness degree even so in normal body weight category. The Navy formula is simple, suitable, certain and inexpensive method for measure body composition [25, 26] and used as one of prevention or treatment programs in obesity monitoring and its related

complications [27]. The formula can be use any time of the day regardless of immediate food intake and physical activity without fluctuation in results [28]. The Navy formula give similarities in results with the results of using bioelectrical analysis for body composition measurement in obese and normal adults for both gender and recommended to use as screening tools [29].

The elevation of blood pressure in obese females may related to structural alternations in kidney that effect on nephron activity by renal fat deposition [30] or related to alternations in aldosterone secretion and angiotensin II [31].

The study results showed that the obese females particularly with central obesity had dyslipidemia compared to normal weight females. Theenlargement of abdominal fatcause disturbing in lipid profile that increased lipolytic activity of adipose tissue which in turn elevate TG production and transfer it to the liver. This will enhance VLDL synthesis in the liver and increase the circulating TG. In the same time, theinhibition of lipoprotein lipase in adipose tissue and skeletal muscles also cause hypertriglyceridemia. These events lead to exchange cholesteryl esters from VLDL to HDL and LDL, and hydrolyze TG by hepatic lipase result in producing a small dense of LDL and a low HDL concentration [32].

In the current study, the increasing inglucose and insulin levels were observed in both central and peripheral obesity females while the HOMI was higher in central obese females than peripheral, this suggested that the centralobesity contributed to accelerate developing of insulin resistance by disturbing in lipid profiles and as shown from the positive correlation between the WC and the serum glucose level. The high circulating free fatty acids resulted from lipolytic activity will enhance gluconeogenesis that caused insulin resistance [33]. The metabolites of fatty acids like acyl- coenzyme, diacylglycerol and ceramides impair insulin signaling [34]. The high flux of free fatty acids to the liver during hyperglycemiawill increased TG synthesis and decreased insulin clearance, that lead to hyperinsulinemia and insulin resistance [35].

The WHR is associated with increased visceral fat depsit that expressed by central obesity which is caused alternations in metabolic profiles [36]. The visceral fat is accompanied with insulin resistance, dyslipidemia and inflammation [37]. The lipolytic activity in adipocytes in obesity condition increased macrophage accumulation in adipose tissue that increased production of pro- inflammatory proteins like IL8, IL6 and visfatin [38, 39], and lowering the angiognetic ability and cause hypoxia which in turn stimulated metabolic alternations and diseases occurrence [40]. Also the risk factors of metabolic diseases is increased with accumulation of abdominal visceral fat and subcutaneous fat, independently of total body fat [41]. The metabolic alternations in obese females with central obesity is related to incidence of insulin resistance [15].

In this study the high level of thyroid stimulating hormone in obese females may effect on body composition by affecting on thermogenesis and energy expenditure [42, 43].The inflammatory cytokines like IL6 and 8 is resulted from the fat accumulation in obesity caused morphological and functional changes in thyroid gland by varying the permeability of gland blood vessels [44, 45], impact on iodide uptake by thyroid cells, or inhibit the mRNA expression of symporter sodium/iodide [45]. In adult women and men there is a positive correlation between BMI, WC and serum TSH, FT3and FT4 [46]. The overweight person have high level of TSH and low level of FT4, while obese person have high level of TSH and low ratio FT3/FT4, suggest the thyroid effect on body weight [10].The hypothyroidism cause insulin resistance even if the thyroid stimulating hormone TSH is at the normal level range [47]. It found that the visceral body fat reduction enhance the thyroid parameters than the reduction in subcutaneous body fat [48].

The study results display that the obese females had higher cortisol level than the normal weight females, this may explained by alternations in glucocorticoids activities in abdominal obesity [49], or the elevation of serum cortisol in central obesity group of this study may explained by the increasing in blood cortisol lead to visceral fat accumulation and increased central obesity [50]. The high expression of glucocorticoids receptor in visceral fat made it hypersensitive to cortisol that cause fat accumulation[51].The visceral adipose tissue contain 11β-hydroxy steroid dehydrogenase I that activate the cortisone (inactive) to cortisol (active) that may contribute to regulate cortisol level in blood circulation[52].

From the study results, the high level of total testosterone in obese females, may related to the obese females with abdominal obesity showed hyper-androgenism , as the adipose tissue act as intracrine site for androgen synthesis (androstenedione convert to testosterone)[53]. The hyperinsulimia caused changes in levels of sex hormones include androgen, estrogen and sex hormone binding globin [54]. The premenopausal overweight women showed high total and free testosterone level [55]. The obese women with insulin resistance display low levels of estradiol and sex hormone binding globin [56].

From the study results, the increasing in visfatin level in obese females with central obesity may related that the visfatin level is associated with visceral fat not subcutaneous fat in obese subjects [57]. The obese women showed high serum of visfatin level compared to normal weight women [58]. Visfatin secretion increased with increased adiposity [59] and its level correlate positively with insulin resistance, WC and IL6 in obesity [60, 61]. The obese women with insulin resistance showed elevated level of hormone visfatin [56].

The elevation of inflammatory biomarkers including IL8 and CRP in obese females especially with central obesity possibly related that the excessive size of adipocytes caused decreased in anti-inflammatory adipokines secretion from adipose tissue and enhance inflammatory process in addition to hyperglycemia caused elevation in IL8 [62]. The WHR is associated with increased visceral fat depots that expressed by central obesity which is caused alternation in metabolic profiles[36], these association was shown in our results in which the WHR correlated positively with IL8. The free fatty acids intermediates activate the pro-inflammation pathway include serine kinase that stimulate the IL8 secretion that promote secretion of C-reactive protein [63]. The process of chronic-low grade inflammation in obesity play important role in metabolic syndrome development, central obesity, insulin resistance, dyslipidemia, and increased oxidative stress by production of reactive oxygen species [64, 65].

## Conclusion

Monitoring the metabolic changes associated with obesity increase demand to use reliable and low cost measurement tool, therefore, in this study the using of Navy formula was useful to detect differences in fat ratio and fat mass between the regional obesitygroups. In obese healthy females, the alternations in biochemical profiles was associated with changes in some hormonal and inflammation biomarkers which were characterizedin central obesity group.

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