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Abdominal obesity in relation to inflammatory markers and insulin resistance in obese Egyptian Females

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Abdominal fat is known to release various inflammatory and anti inflammatory cytokines leading to development of insulin resistance (IR). However, the relationship between them has scarcely been reported in Egyptian obese women. To determine associations between abdominal obesity, measures of obesity, inflammatory markers and IR among obese women. A cross-sectional study comprising 200 obese women between ages of 18 to 35 years were classified according to waist circumference (WC) into two groups: abdominal obese group with WC ≥ 85 cm and non-abdominal obese group WC < 85 cm. Serum IL-1β, CRP and adiponectin were measured using a commercially available ELISA kit. Insulin resistance has been estimated by the Homeostasis Model Insulin Resistance (HOMA-IR). Abdominal obese group showed significant higher levels of HOMA-IR, fasting glucose, hs-CRP, IL-1β, BP levels, total cholesterol, triglyceride and lower adiponectin levels than non-abdominal obese group. In addition, adiposity indices including body mass index, waist to height and waist to hip ratios as well as body fat% and abdominal skin fold thickness were significantly higher in abdominal obese group. IL-1β and CRP showed significant positive correlations with adiposity indices; while adiponectin had inverse relations. Abdominal obesity is associated with high levels of pro inflammatory cytokines, metabolic abnormalities and low adiponectin levels. Results emphasize the role of certain pro- and anti-inflammatory cytokines in metabolic dysfunction in obese women with abdominal obesity.

Keywords: Abdominal obesity, inflammatory cytokines, metabolic dysfunction, IR

INTRODUCTION

Obesity is a significant universal health problem in which the numbers of obese just keep rising. It caused increasing risk of metabolic disorders such as type 2 diabetes, and development of cardiovascular diseases. It is reported that obesity corresponds to a sub-clinical inflammatory condition, which can lead to development of insulin resistance (Alexandraki et al., 2006; Carter et al., 2016; García et al. 2006; Qatanani and Lazar 2007; Thalmann and Meier 2007). There is a positive relationship between IR, abdominal obesity and serum lipid levels. This relationship is owing to the metabolic effects of the visceral adipose storage area which secretes a number of factors that are known to influence insulin function and lipid metabolism. In particular, visceral fat releases free fatty acids (FFAs) into the portal vein and this may cause increased hepatic lipid and glucose output and reduced insulin clearance insulin resistance (Crowther and Ojwang, 2006).

Individuals with abdominal obesity have an excess of adipose tissue, often accumulated in the visceral area (Taubes, 2009). Adipose tissue secrets biologically active cytokines (adipocytes)

able to modulate immunological responses, and plays a role in metabolism via regulation of auto crine and paracrine signaling (Charles et al., 2018; Juge-Aubry et al., 2005). It is considered as endocrine organ which is a source of adipokines like adiponectin and inflammatory cytokines such as interleukin (IL-1), (IL-6) and tumor necrosis factor (TNF) which contribute to the insulin resistant, pro inflammatory, thrombotic, and hypertensive state of visceral obesity (Despre et al., 2008).

Obesity or its related disorders leads to increase interleukin-1β (Mojtaba et al., 2011). IL-1β is a pro inflammatory cytokine continually elevated in the presence of obesity (Charles et al., 2018; Fain 2006). It is a major pro inflammatory cytokine which is produced mostly by macrophages and biologically active. IL-1β is formed through cleavage of pro-IL-1β by caspase-1 activated via the NLRP3 inflammasome(Sims and Smith 2010; Stienstra et al., 2010). IL-1β has a big role in the translation of obesity-associated inflammation into insulin resistance in rodent models (Tack et al., 2012; Wen et al., 2011). However, in human IL-1β is also released from non-fat cells by adipose tissue in case of obesity(Fain 2006; Koenen et al., 2011). High dose of IL-1β (20 ng/ml) has a big role in decreasing protein expression of IRS-1 and GLUT4 mRNA in murine 3T3-L1 adipocytes (Bing et al., 2015; Lagathu et al., 2006). Central obesity has been recognized as an independent risk factor for metabolic diseases.

For further explain the role of abdominal obesity on inflammatory, anti inflammatory cytokines and IR the present study aimed to determine association between abdominal obesity, inflammatory markers and metabolic parameters among obese women with and without central obesity.

MATERIALS AND METHODS

A cross-sectional study comprising 200 obese women between ages of 18 to 35 years were recruited from the obesity clinic, National Research Centre.

Two hundred obese women between ages of 18 and 35 years were recruited from the obesity clinic, National Research Centre. None of the study participants had diabetes according to the criteria of American Diabetes Association (Puavilai et al., 1999) . One hundred obese women with abdominal obesity WC ≥ 85 cm and 100 non-abdominal obese women with WC < 85 cm were examined. We excluded women who smoked cigarettes, taking medication that affect lipids or hormones, with cardiovascular disease or chronic kidney or liver disease, pregnant or breastfeeding, or had any other major illness. Written informed consent was gained from each woman after a complete description of the study. The research has been authorized by the Ethical Committee of National Research Centre, Egypt (number = 16361), in accordance with the World Medical Association's Declaration of Helsinki.

WC at the umbilical level was measured in the late exhalation phase in standing position. Abdominal obesity was diagnosed by WC ≥ 85 cm. according to National Cholesterol Education Program (NCEP)-Adult Treatment Panel (ATP) III(Expert Panel on Detection, 2001)

Statistical analysis

We performed the statistical analyses using SPSS16.0 for Windows (SPSS Inc). The distribution of the variables was examined using Kolmogorov–Smirnov test of normality to verify whether it is followed a Gaussian pattern.
Means ± SDs were used in order to express the normally distributed data.

Clinical and biochemical assessment

All patients were subjected to full medical history and clinical examination. BMI has been determined as weight in kilograms divided by height in meters square (kg/m2). WC and hip circumference (HC) have been measured in cm using a plastic, non-stretchable tailor's tape. Subsequently, the waist-to-hip-ratio (WHR) was calculated as WC divided by HC. Skin fold thickness was measured in all subjects at the biceps, triceps, subscapular, suprailiac, and abdominal areas using Holtain caliper then, the sum of skin folds were calculated as well as waist to height ratio.

A full description of the anthropometric measurements has been reported elsewhere (Zaki et al., 2018). All measurements were obtained according to standardized equipment and following the recommendations of the International Biological Program (Weiner and Lourie, 1969). Body fat % was assessed by Tanita Body Composition Analyzer (SC-330). Blood pressure was measured according to a standardized operating procedure using calibrated sphygmomanometer and brachial inflation cuff (HEM-7200 M3, Omron Healthcare, Kyoto, Japan). Systolic and diastolic blood pressures (SBP and DBP) were measured twice in the right

arm in a sitting position after a 10-min rest period, then the average is used for analysis.

Laboratory measurements

Venous blood samples were collected by direct venipuncture after an overnight fast(minimum 12 h). Fasting plasma glucose and serum lipids (total cholesterol, high-density lipoprotein cholesterol (HDL-C), triglycerides (TG) were measured by enzymatic colorimetric methods using a Hitachi auto-analyzer 704 (Roche Diagnostics, Switzerland) (Hirschler et al., 2010). Low density lipoprotein cholesterol (LDL-C) was calculated according to certain equation (LDL-C= Total cholesterol –Triglycerides/5+HDL-C). Serum insulin concentration was analyzed by chemiluminescent immunoassay (Immulite 2000, Siemens, Germany)(Zaki et al., 2015). Insulin resistance has been estimated by the Homeostasis Model Insulin Resistance (HOMA-IR);as the outcome of fasting plasma insulin level (IU/mL) and fasting plasma glucose level (mmol/L) divided by 22.5 (Matthews et al., 1985). Serum IL-1β was measured using a commercially available Quantikine ELISA kit (R&D System, Inc., Minneapolis, MN).

RESULTS

Table 1 shows that abdominal obese group had significantly higher values of serum IL-1β, CRP, HOMA-IR and fasting glucose ,TC, TG, fat%, BP levels and lower adiponectin level than non-abdominal obese group. Moreover, WC, WHt, WHR, and abdominal skin fold thickness were significantly higher in abdominal obese group as compared to non-abdominal obese group.

Table 2 shows relation between serum inflammatory markers, adiponectin and adiposity indices. Inflammatory markers IL-1β and CRP showed significant positive correlations with adiposity indices including BMI, WHR, WHt and WC. On the other hand, adiponectin showed significant negative relations with these parameters.

Table 1. Clinical and biochemical characteristics of the study participants

	Non abdominal obese Group n=100	Abdominal obese Group n=100	P
	Mean ± SD	Mean ± SD	
IL-1β(pg/ml)	21.7±7.4	27.8±10.4	0.009
HOMA-IR	3.1±1.6	5.5 ± 2.5	0.006
TC (mg/dl)	185.3 ± 40.2	201.9 ± 39.9	0.04
TG(mg/dl)	82.2 ± 31.9	110.6 ± 36.2	0.001
HDL(mg/dl)	12.2± 48.5	50.1 ± 13.1	0.40
LDL(mg/dl)	122 ± 46.2	129 ± 41.5	0.30
SBP (mmHg)	100.5±14.5	110.2±15.7	0.001
DBP (mmHg)	67. 4 ± 8.9	72.4 ± 10.2	0.002
F. Glu (mg/dL)	83.7± 20.7	103.2 ± 24.7	0.001
Fat%	30.7 ± 7.9	42.2 ± 5.8	0.001
WHR	0.79 ± 0.04	0.85 ± 0.06	0.001
CRP (mg/L)	1.4 ± 0.5	4.9 ± 2.8	0.001
Adiponectin(µg/ml)	9.6 ± 2.1	7.9 ±1.7	0.05
WC (cm)	82.7± 4.4	104.4 ± 11.66	0.001
WHt	0.56 ± 0.06	0.61 ± 0.08	0.001
Abdominal. SF	24.74 ± 6.0	33.18 ± 6.9	0.001

IL-1β: interlukin-1β; HOMA-IR: homeostasis model assessment of Insulin Resistance; TC : total cholesterol; TG: triglyceride ; HDL: high-density lipoprotein cholesterol; LDL: low-density lipoprotein cholesterol; SBP: systolic blood pressure , DBP: Diastolic blood pressure , F.Glu: fasting glucose; WHR: waist to hip ratio; CRP:C-reactive protein; WC: waist circumference; WHt: waist to height ,Abdominal. SF: abdominal skin fold.

Table 2.Correlations between serum inflammatory markers, adiponectin and adiposity indices.

Adiposity indices	IL-1B(pg/ml)		CRP(mg/L)		adiponectin (µg/ml)	
	r	p	r	p	r	p
BMI (kg/m^2)	.298	0.01	.308	0.05	-.285	0.02
WHR	0.26	0.02	.378	0.03	-.34	0.03
WHt	0.31	0.005	0.65	0.01	-.37	0.04
WC (cm)	-0.1	0.3	0.6	0.001	-.298	.007

IL-1β:interlukin-1β; CRP:C-reactive protein; BMI: body mass index WHR: waist to hip ratio; WC: waist circumference; WHt: waist to height

DISCUSSION

To our knowledge, this is the first study investigate the relationship between inflammatory markers, IR and abdominal obesity among Egyptian obese premenopausal women.

This study provides evidence regarding associations between IL-1β, with abdominal obesity and IR. One of the most concerning complications of obesity is IR; it is also considered a precursor of type 2 diabetes mellitus and MS. It is clinically diagnosed by HOMA-IR (Alberti et al., 2009).

In the present study, abdominal obesity exhibits discrete association with inflammatory markers. In addition it showed a positive correlation with HOMA-IR. Abdominal obesity shares in the development of insulin resistance, hyperinsulinism and is associated with cardiovascular complications (Del-Rio-Navarro et al., 2008) . The result of the current study is in agreement with González-Jiménez et al., and Lin et al., who concluded that WC is highly sensitive and specific and correlates with insulin resistance in adults and it is regarded as a predictive risk factor for insulin resistance (González-Jiménez et al., 2016; Lin et al., 2015).

Measures of anthropometric indicators such as WC , WHR and WHTR are used to assess central fat distribution(Owolabi et al., 2017). Also Satpathy et al., who used WHR ratio to assess abdominal obesity (Satpathy et al., 2015).

Adiponectin is an adipokine constitutively produced at high levels by fat tissue. Though, few prior studies have been able to determine whether adiponectin is associated with obesity-related diseases such as IR and to assess the relation between adiponectin and other cytokines. Adipocytes produced adiponectin that regulate metabolic processes and improves insulin sensitivity and involved in multisystem diseases including obesity, diabetes and dyslipidemia.

In the current study, serum adiponectin was lower in the subjects with abdominal obesity than those without. However, adiponectin showed significant negative relations with adiposity indices (BMI, WHR, WHt and WC). Compatible with previous reports, low level of serum adiponectin is a strong risk for abdominal obesity. Adiponectin concentrations have been found to be associated conversely with systemic inflammation and increased concentrations of high-sensitive CRP in patients with metabolic syndrome (Zaki et al., 2015). Moreover, previous studies reported that adiponectin level was significantly lower in abdominal obese cases that associated with negative correlations with WC,WHR and WHtR, (Montazerifar et al., 2018; Ochiai et al., 2014)

Elevation of serum hs-CRP and IL-1β concentration enhances the incidence of IR in the subjects with abdominal obesity. A significant increase in CRP values was found in women with abdominal obese compared with non abdominal obese women.

Also, the current study found a significant positive correlation between WC which is considered measure of abdominal obesity and hs-CRP level. This finding is consistent with the previous study reported that hs-CRP level was positively correlated with WC among ninety-one overweight to obese (BMI > 25 kg/m²) women from Kelantan, Malaysia (Sanip et al., 2013). Overall, this study suggests that higher WC in obese women promote inflammation. Thus, measuring the hs-CRP level as an indicator of inflammation might be valuable to expect the cardiovascular risk of the obese adult population. According to Taverne et al., having an hs-CRP level tested is beneficial to predict the future CVD events in the absence of serum low-density lipoprotein test result (Taverne et al., 2013). Our results showed TC, TG--- Level of hs-CRP might be the key which reduce the prevalence of abdominal obesity and endocrine, nutritional and metabolic diseases as well as cardiovascular

diseases (Shahadan et al., 2018).

IL-1β, is one of the most important cytokine produced largely by macrophages, is involved in the development of obesity associated insulin resistance (Bing et al., 2015). The primary source of IL-1 is macrophage, but epidermal, epithelial, lymphoid and vascular tissues also synthesize IL-1β production and secretion has as well been reported from pancreatic islets (Maedler et al., 2009). It was reported that increased secretion of IL-1β have been linked not only to various autoimmune and auto-inflammatory diseases, but also to metabolic dysregulation (Mojtaba et al., 2011). IL-1β has a big role in macrophage-adipocyte crosstalk in obesity. In this study IL-1β shows higher levels in abdominal obesity group than non abdominal obesity group with significant difference. And also there is a positive correlation between IL-1β and BMI, WHR and WHt which they considered as abdominal obesity indices. These results were in agreement with other study concluded that changes in serum interleukin-1β are influenced by abdominal obesity (Satpathy et al., 2015). In addition, significant correlation was also seen between CRP and WC and WHR in obese group (Satpathy et al., 2015).

IL-1β damages insulin sensitivity in adipose tissue by inhibition of insulin signal transduction by IL-1β leads to damage insulin sensitivity in adipose tissue. The receptor binding or production of IL-1β improves insulin signaling and action in human adipocytes. This is compatible with a reduction in macrophage stimulated pro-inflammatory profile and lipolysis. Targeting IL-1β may be beneficial for defending against obesity-related insulin resistance at the tissue and systemic level (Bing et al., 2015).

CONCLUSION

Aabdominal obesity showed significant effect on inflammatory and anti-inflammatory proteins that might be linked with metabolic dysfunction and IR in obese women

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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AUTHOR CONTRIBUTIONS

Moushira Zaki: Conceived the idea, planned for the study, did the statistical analysis and writing the article.

Shams Kholoussi: Did the immunological and biochemical analyses and reviewed the manuscript.

Walaa Yousef: Did anthropometric measurements, reviewed the manuscript and collect data.

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