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Research Article

Visceral adiposity index correlation with Rotterdam criteria in patients with polycystic ovary syndrome

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ABSTRACT

Background: The present research explores the correlation of visceral obesity index with Rotterdam criteria (hyperandrogenism and/or hyperandrogenemia, oligomenorrhea and Ultrasound polycystic ovarian morphology) among Egyptian polycystic ovary syndrome patients.
Methods: We enrolled one hundred female patients with polycystic ovary syndrome with age ranged 18-44 years (mean age 26.83±6.092 years).
Results: VAI very strongly correlated with waist circumference, TG and HDL-c, also moderately correlated with systolic B.P, BMI, HOMA-IR and insulin levels, also we found only insulin, menstrual cycles per year number and FGS were significant predictors of hyperandrogenemia in PCOS patients. Only Ultrasound polycystic ovarian morphology was a significant risk factor for oligomenorrhea in PCOS patients in logistic regression analysis. VAI, TT and Farman gallawy score can significantly predict number of the menstrual cycles per year number ($p < 0.05$). In logistic regression analysis, only oligomenorrhea was a significant independent risk factor for PCOM ($p < 0.05$). In addition, only VAI was a significant independent ($p < 0.05$) risk factor for metabolic syndrome.
Conclusions: VAI was an independent significant predictor for metabolic syndrome in patients with PCOS and a good marker of cardiometabolic risk in PCOS patients. In addition, VAI was significant predictors of annual menusteral cycle but not testosterone levels or polycystic ovarian morphology in PCOS. This study confirms the value of VAI in identification of patient with risk for metabolic syndrome and cardiometabolic risk in PCOS patients, but not found a role for VAI in diagnosis of PCOS.

Keywords: Hyperandrogenism, Oligomenorrhea, PCOS, VAI

INTRODUCTION

Polycystic ovary syndrome (PCOS) is one of the most common endocrine disorders of women at reproductive age and the major cause of anovulatory infertility.[1] Chereau first described it as the change of ovarian morphology in 1844.[2] PCOS is a disease with high heterogeneity, and its clinical features mainly include menstrual disorder, secondary amenorrhea, serum hormone abnormality, hairiness, acne, obesity, and infertility.[3]

Recently, the use of visceral adiposity index (VAI), a sex-specific mathematical model that uses both anthropometric [body mass index (BMI) and waist circumference (WC)] and functional [triglycerides (TG) and high-density lipoprotein cholesterol (HDL)] parameters, has been suggested as an index of applicable index for the evaluation of visceral fat dysfunction.[4]

In the last years, the VAI has been reported to express cardiometabolic risk and possible adipose tissue dysfunction (ATD) was identified in populations with

metabolic risks such as the general population with obesity, post-menopausal women with non-alcoholic fatty liver disease in patients with acromegaly, the VAI appears to be associated with disease activity, adiponectin levels, insulin sensitivity and secretion, and women with PCOS.[10]

Recent studies evaluated VAI in women with PCOS and reported that VAI correlated with insulin resistance, differentiate the "metabolically healthy polycystic ovary syndrome" from the "metabolically unhealthy polycystic ovary syndrome" Also VAI replace visceral CT scan as a marker for visceral adiposity.[11-13] VAI is considered a predictor of clinical severity and therapeutic outcome of PCOS.[14] And a predictor of diabetes risk in women with polycystic ovary syndrome.[15]

PCOS is characterized by hyperandrogenemia, Insulin resistance, chronic anovulation, and polycystic ovary morphology.[16] Hyperandrogenemia is the biochemical hallmark of PCOS.[17] Previous studies hypothesized that hyperinsulinemia causes hyperandrogenemia and anovulation in PCOS.[18] On the other hand other studies, since many normal-weight PCOS patients are not insulin resistant, the hypothesis that hyperandrogenism per se leads to abdominal visceral adiposity, insulin resistance and hyperinsulinemia has been postulated by many authors.[17]

Visceral adiposity index is a marker of ATD and correlated with insulin resistance in PCOS.[13] Relation between VAI with hyperandrogenism and anovulation in PCOS is unclear. So the evaluation of this relationship will be valuable in order to evaluate of VAI as marker of the main criteria of polycystic ovary syndrome, not ATD alone. The aim of this study was to evaluate the correlation of visceral obesity index with Rotterdam criteria (hyperandrogenism and/or hyperandrogenemia, oligomenorrhea and ultrasound polycystic ovarian morphology) among Egyptian polycystic ovary syndrome patients.

METHODS

We enrolled one hundred female patients with polycystic ovary syndrome with age ranged 18-44 years (mean age 26.83±6.092 years). This study was conducted at specialized medical hospital and Mansoura university hospital, in Egypt, from July 2012 to December 2015.

Informed consent obtained from all participants. Inclusion criteria were as follows: 18 to 45 female patients with polycystic ovary syndrome (diagnosed according to the Rotterdam criteria which require the presence of two of three: oligomenorrhea (less than 6-9 menses per year), clinical hyperandrogenism (hirsutism defined as a Ferriman-Gallwey (FG) score ≥ 8, acne) or biochemical hyperandrogenism (serum total testosterone [TT] >0.8 ng/mL upper limit of the present laboratory) and polycystic ovary appearance on ultrasonography (≥12 follicles measuring 2-9 mm in at least one ovary and/or increased ovarian volume, >10 mL), with adequate hepatic and renal function, and absence of malignancies, metabolic or any osseous diseases.[19,20]

Exclusion criteria involved hepatic or renal disease, abnormal albumin levels, patients with autoimmune disease and endocrine disorder including type 2 diabetes (abnormal thyroid function, hypercrtolism, hyperprolactinemia, abnormal calcium}, hypertensive patient and cardiac disease.

BMI was calculated from body weight and height by the standard formula: BMI = (weight in kg/height in m^2). Waist circumference was estimated as the smallest circumference at the level of the umbilicus. VAI was calculated using the proposed formula: VAI = WC/36.58 + (one.89 × BMI) × TG/0.81 × 1.52/HDL.[4]

Laboratory evaluation included; laboratory evaluation included; basal prolactin (ng/ml), total testosterone (ng/ml), FBS (mg/dl), insulin (mIu/ml), Hb (g/dl), Hct%, WBC (X109/L), PLT (X109/L), uric acid, SGOT, SGPT, HDL-c, LDL-c (mg/dl) and TG (mg/dl) during the follicular phase. All blood samples were obtained at 09:00 hours in the morning after overnight fasting. Homeostasis model assessment for insulin resistance (HOMA-IR) was calculated using the standard formula: fasting glucose (mm)/fasting insulin (mIU/ml)/22.5. Insulin sensitivity was estimated indirectly using basal insulin and glucose values to calculate the HOMA-IR [glycemia (mg/dl)×insulinemia (mU/ml)/405.[22]

All data were analyzed using the SPSS statistical version 22.0. Pearson correlation was used to assess the correlation between VAI, total testosterone and number of menstrual cycle per year with other parameters, significant correlation was entered in multiple regression analysis for determination of independent predictors. An independent t test was used for comparison of continuous variables. Categorical data were analyzed by the Pearson Chi-square. A multivariate analyses model (enter method) was used to examine the relationship between polycystic ovarian morphology and metabolic syndrome with other significant parameters. P values less than 0.05 were considered significant.

RESULTS

We enrolled one hundred female patients with polycystic ovary syndrome from July 2012 to December 2015 with mean age 26.83±6.09 years (ranged from 18 year to 44). Details of patient's characteristics were presented in Table 1.

VAI correlation and independent predictors

In this study we found visceral adiposity index had statistically significant correlation ($p < 0.05$) with anthropometric parameter (waist circumference, BM and

Weight), clinical parameter (systolic B.P, diastolic B.P, menstrual cycles per years and Farman Gallwey score), biochemical (HDL-c and triglyceride), and hormonal parameter total testosterone, insulin and HOMA IR.

Table 1: patient's characteristics.

	Mean	Standard deviation
Age	26.83	6.092
Weight	82.8300	15.01787
Height	1.5978	0.03963
BMI	32.5057	6.27400
Waist circumference	115.44	16.468
VAI	3.7736	0.99776
Systolic B.P	132.7500	14.07726
Diastolic B.P	84.2500	8.62739
Menstrual cycles per years	7.55	1.956
Farman Gallawy score	13.94	3.598
Total cholesterol	229.0500	30.18775
LDL-c	142.9900	27.61458
HDL-c	48.7900	7.71630
Triglyceride	186.3500	39.88959
FBS	94.76	9.322
Total testosterone	99.3000	14.62632
Prolactin	17.6670	3.08620
TSH	2.4444	0.85217
Insulin	12.4593	5.78178
HOMA	2.9290	1.44578
Metabolic syndrome	59 (59%)	
Oligomenorrhoea	66 (66%)	
Ultrasound polycystic ovarian morphology	87 (87%)	

Table 2: Pearson correlations between VAI with laboratory and clinical parameters and regression analysis (stepwise method) with significant correlated parameters for determination of VAI predictors PCOS patients.

	r	P value	B	β	P value
Age	0.034	0.737			
Waist circumference	0.417	<0.001*	0.032	0.534	<0.001*
BMI	0.274	0.006*	-0.072	-0.453	<0.001*
Weight	0.275	0.003*	Excluded in stepwise regression		
Height	-0.091	0.366			
Systolic BP	0.204	0.046*	Excluded in stepwise regression		
Diastolic BP	0.135	0.179			
Menstrual cycles per years	-0.313	0.002*	-0.024	-0.047	0.013*
Farman Gallawy score	0.240	0.013*	Excluded in stepwise regression		
Total cholesterol	**-0.001**	**0.994**			
HDL-c	-0.561	<0.001*	2.736	-0.547	<0.001*
LDL-c	-0.056	0.583			
Triglyceride	0.723	<0.001*	1.649	0.745	<0.001*
Total testosterone	**0.248**	**0.016***	**0.003**	**0.041**	**0.035***
Prolactin	0.101	0.315			
TSH	-0.016	0.873			
FBS	-0.103	0.310			
Insulin	0.240	0.016 *	Excluded in stepwise regression		
HOMA	0.214	0.033 *	Excluded in stepwise regression		

*Correlation is significant at the 0.05 level (2-tailed).

However, in regression analysis by stepwise method to eliminate effect of collinearity, we found only total testosterone, menstrual cycles per year number and the lipid parameters of the index (HDL-c and Triglyceride) and anthropometric parameters Waist circumference, BMI as shown in Table 2.

Hyperandrogenemia

In present study, we found total testosterone had statistically significant correlation (P <0.05) with anthropometric parameter (waist circumference, BM and weight), clinical parameter (systolic B.P, diastolic B.P, menstrual cycles per years and Farman Gallwey score), VAI, HDL-c, insulin and HOMA IR.

However, in regression analysis, we found only insulin, menstrual cycles per year and FGS were significant predictors of hyperandrogenemia in polycystic ovarian syndrome as shown in Table 3.

Oligomenorrhea

Only Ultrasound polycystic ovarian morphology was a significant risk factor for Oligomenorrhea in PCOS patients in logistic regression analysis (p <0.05) as presented in Table 4.

Table 3: Pearson correlation and multiple regression (enter method) analysis between total testosterone levels with other statistically significant correlated factors.

	r	P value	B	β	P value
Waist circumference	0.274	0.006*	0.111	0.125	0.374
BMI	0.258	0.005*	Excluded because of collinearity with weight		
Weigh	0.298	0.04*	0.016	0.016	0.902
VAI	0.248	0.013*	-1.560	-0.106	0.339
Systolic B.P	0.232	0.019*	0.038	0.036	0.699
Diastolic B.P	0.207	0.039*	Excluded because of collinearity with systolic B.P		
Cycles number/year	-0.413	0.001*	-1.934	-0.259	0.005*
Farman Gallawy score	0.473	<0.001*	1.581	0.389	<0.001*
HDL-c	-0.231	0.021*	-8.046	-0.110	0.267
Insulin	0.392	<0.001*	0.575	0.227	0.023*
HOMA	0.380	0.001*	Excluded because of collinearity with Insulin		

*significant at the 0.05 level.

Table 4: Logistic regression analyses (enter method) for predictors of oligo-ovulation in PCOS patients.

	B	S.E.	Wald	Signature	Odd ratio	95% C.I .for OR	
						Lower	Upper
Metabolic syndrome	-0.863	0.594	2.110	0.146	0.422	0.132	1.352
VAI	-0.026	0.317	0.007	0.935	0.974	0.523	1.815
HOMA	0.116	0.232	0.251	0.616	1.123	0.713	1.771
TT	0.015	0.019	0.641	0.424	1.015	0.978	1.054
PCOM	-2.757	0.775	12.651	<0.001*	0.063	0.014	0.290

*Significant at the 0.05 level.

Table 5: Pearson correlation and multiple regression (enter method) analysis between number of menusteral cycle per year with other statistically significant correlated factors.

	r	P value	B	β
VAI	0.313	0.002*	-0.387	-0.197
Systolic B.P	0.248	0.013*	-0.017	-0.123
Diastolic B.P	0.212	0.034*	Excluded because of Collinearity with Systolic B.P	
TT	-0.413	0.001*	-0.042	-0.313
Farman Gallawy score	0.219	-0.005*	-0.009	0.929
Insulin	0.246	0.013*	-0.015	-0.046
HOMA	0.225	0.025*	Excluded because of Collinearity with Insulin	

*significant at the 0.05 level.

Table 6: Logistic regression analyses (enter method) for predictors of ultrasound polycystic ovarian morphology (PCOM).

	B	S.E.	Wald	Signature	Odd ratio	95% C.I .for OR	
						Lower	Upper
Total testosterone	0.029	0.029	1.040	0.308	1.030	0.973	1.090
Oligo-ovulation	-2.507	0.704	12.693	<0.001*	0.082	0.021	0.324
Diastolic B.P	0.007	0.064	0.012	0.913	1.007	0.889	1.141
Systolic B.P	0.030	0.043	0.483	0.487	1.030	0.947	1.121

*Significant at the 0.05 level.

However, by linear regression analysis VAI, TT and Farman Gallwey score can significantly predict number of the menstrual cycles per year number ($p < 0.05$). Also in Pearson correlation, we found menstrual cycles per year number had statistically significant correlation with VAI, systolic and Diastolic B.P, Insulin, HOMA IR, TT and Farman Gallawy score as described in Table 5.

Ultrasound polycystic ovarian morphology (PCOM)

In logistic regression analysis, only oligomenorrhea was a significant independent risk factor for PCOM ($p < 0.05$) as described in Table 6.

Metabolic syndrome

In logistic regression analysis for determination of risk factor of metabolic syndrome in PCOS patients, only VAI was a significant independent ($p < 0.05$) risk factor for metabolic syndrome (Table 7).

Table 7: Logistic regression analyses (enter method) for metabolic syndrome risk factors in PCOS patients.

	B	S.E.	Wald	Signature	Odd ratio	95% C.I .for OR	
						Lower	Upper
Waist circumference	0.011	0.027	0.166	0.684	1.011	0.959	1.066
VAI	1.395	0.376	13.737	<0.001*	4.033	1.929	8.433
Systolic B.P	0.029	0.034	0.728	0.394	1.029	0.963	1.100
Diastolic B.P	0.003	0.057	0.003	0.954	1.003	0.897	1.123
Farman Gallawy score	0.103	0.085	1.464	0.226	1.109	0.938	1.311
Total testosterone	0.001	0.023	0.002	0.964	1.001	0.957	1.047
HOMA	0.119	0.226	0.279	0.598	1.127	0.723	1.755
BMI $\geq$ 30 Kg/m^2	0.634	0.944	0.451	0.502	1.886	0.296	12.005
oligoovulation	-0.526	0.545	0.933	0.334	0.591	0.203	1.718

*Significant at the 0.05 level.

DISCUSSION

In the present study, we investigated the relation of the component of PCOS according Rotterdam criteria oligo-amenorrhea, clinical hyperandrogenism or hyperandrogenemia and US polycystic ovarian morphology with visceral adiposity index (VAI) as a marker metabolic dysfunction of visceral obesity.

In this study we found total testosterone correlated with VAI with statistically significance in agreement Andreoulakis et al results, but previous study depend only on Pearson correlation only for their conclusion which not enough to conclude a role for VAI in determination of TT levels in PCOS patients.[17] In regression analysis, we found VAI was non-significant predictor of total testosterone levels in polycystic ovarian disease patients.

By contrast, we found TT level was a significant predictor for VAI in PCOS which in agreement with Oh et al results.[13] These finding indicated that hyperandrogenemia leads to visceral obesity and adipose tissue dysfunction cystic ovarian disease patients.

Hyperandrogenemia is the biochemical hallmark of PCOS.[21] The mechanisms explained the association between visceral obesity and hyperandrogenism was controversial. Some studies suggested insulin resistance, studies suggested that insulin also contributes to hyperandrogenemia and anovulation in affected women through a number of distinct molecular mechanisms involved insulin receptor and post receptor defect.[22,23] Other studies suggested that inappropriate gonadotropin secretion and exaggerated ovarian response to gonadotropin, which one of the most characteristic biochemical abnormalities in PCOS is involved in hyperandrogenemia and insulin resistance pathogenesis.[24] In current study insulin was a significant predictor for TT levels which indicated a pivotal role for insulin resistance in hyperandrogenemia pathogenesis. Unfortunately pituitary and hypothalamic role not evaluated in our study.

On the other hand, other studies reported that hyperandrogenemia per se can leads to abdominal visceral adiposity, insulin resistance and hyperinsulinemia, which had been, reported previously.[25]

In the same veins, we found number of menstrual cycle per year significantly correlated with VAI. In addition, in regression analysis we found VAI was significant independent predictors for number of menstrual cycles per year in women with polycystic ovarian syndrome. These findings were in agreement with other studies [10.17] reported that higher VAI associated with Oligomenorrhea in PCO women, By contrast another

study reported obesity does not appear to have an important effect on menstrual cycle pattern in PCOS.[26]

Multiple studies have found associations between overweight or obesity and irregular menstruation in women without PCOS.[27,28] The risks for oligomenorrhea and amenorrhea increase 2-fold with each increase in obesity grade.[29] Adipose tissue appears to exert its greatest effects on reproductive cyclicity via complex mechanisms involving hyperandrogenism and hyperinsulinemia.[30]

This controversy may explain by multiple reasons, firstly the subjective nature of cycle irregularity, which can easily over or underestimated. Secondly genetic and ethnicity differences between studies. Thirdly diagnosis of PCOS vary according cafeteria used for diagnosis. The Rotterdam criteria did not require irregular menses or ovulatory dysfunction for diagnosis citing that women with regular menstrual cycles could be considered to have PCOS in the presence of PCO and hyperandrogenemia or hyperandrogenism.[19] While androgen excess society (Aes) excludes the diagnosis of PCOS in women with regular menses and subclinical ovulatory dysfunction.[31] Subclinical ovulatory dysfunction can occur in women with regular menstrual bleeding.[32] Fourthly, high rates of amenorrhea and menstrual irregularity and the prevalence may be associated with certain stresses, which not included in most of studies.[33]

In the same veins, we found women with polycystic ovarian US morphology (PCOM) had statistically significant higher VAI than those without PCOM appearance by US. These findings were similar with other studies in PCOS women, but in logistic regression analysis we found visceral obesity was none a significant risk factor for US polycystic ovarian morphology.[34]

The morphological features of the ovaries in women with polycystic ovarian syndrome include enlarged ovary size, multiple small follicles of similar size, increased ovarian stromal volume, and echogenicity, peripheral distribution of the follicles, and higher stromal blood flow. Ultrasound polycystic ovarian morphology (PCOM) has been recognized as a component of PCOS diagnosis. The inclusion of PCOM sparked a controversy as it broadens the population of women who meet the criteria for PCOS and allows for the creation of two phenotypically different patient populations who previously would have been excluded.[35] gonadotropin secretion and action as important primary defects in disease pathogenesis and some gene regions regulating FSH and LH secretion dysfunction, suggesting genetic susceptibility in PCOS pathogenesis and morphology.[36]

Also In this study we found VAI was an independent significant predictors for metabolic syndrome in patients with PCOS. These findings is contestant with other recent studies reported VAI as a predictor for metabolic syndrome in PCOS patients.[34,37] In the present study, VAI was very strongly correlated with waist circumference, , TG and HDL-c , also moderately correlated with systolic B.P ,BMI, HOMA-IR and insulin levels. Therefore, in the present study it is not surprising that VAI had a good value in predicting Met Syndrome in PCOS women and a good marker of cardiometabolic risk in PCOS patients.

The relation between metabolic syndrome and PCOS phenotype was controversial, some studies in turkey and Iran reported high prevalence in all phenotypes while studies in Latin population found incidence of Met Syndrome in all phenotypes except type A was not different from that in non-PCOS women.[34,38] The difference in age and ethnicity between these studies may explain this controversy.

The present study limitation had some limitation, Firstly total testosterone was the only androgen that was measured in our study and other androgens such as androstenedione and dehydroepiandrosterone sulfate or free testosterone do not be analyzed because of the limitation of research funding. Secondly absence of assay of pituitary gondotrophins which have central role in pathogenesis of the disease. Thirdly exclusion of post-menopausal syndrome and congenital adrenal hyperplasia on clinical base only.

CONCLUSION

In conclusion VAI was an independent significant predictor for metabolic syndrome in patients with PCOS and a good marker of cardiometabolic risk in PCOS patients. In addition, VAI was a significant predictor of annual menusteral cycle but not testosterone levels or polycystic ovarian morphology in PCOS.

This study confirms the value of VAI in identification of patient with risk for metabolic syndrome and cardiometabolic risk in PCOS patients, but not found a role for VAI in diagnosis of PCOS.

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