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Validity of dual-energy X-ray absorptiometry scan in evaluation of trunk fat in non-obese patients with polycystic ovary syndrome

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Abstract

Objective
To detect trunk obesity, which was claimed to be a major cause of insulin resistance, in nonobese patients with polycystic ovary syndrome (PCOS) by using dual-energy X-ray absorptiometry (DEXA) scan.

Patients and methods
A total of 60 normal weight, with BMI less than 25 kg/m^2, patients with PCOS were included in the study. DEXA scan was used to assess the body composition of all patients. Metabolic and hormone measurements and cardiovascular risk were determined. All data were collected and compared with 60 healthy controls.

Results
Normal-weight patients with PCOS had a higher ratio of total and trunk fat than non-PCOS controls. Moreover, they have higher insulin resistance and β-cell function values. No differences were observed in other cardiovascular or metabolic risk factors.

Conclusion
Nonobese women with PCOS had a more significant total and trunk fat compared with non-PCOS women. DEXA scan is a fast and easy method to analyze body composition.

Keywords: Dual-energy X-ray absorptiometry, polycystic ovary, trunk fat

INTRODUCTION

A polycystic ovary syndrome (PCOS) is a complex endocrinal disease affecting 7% of women in their reproductive age. Approximately 80% of patients with PCOS develop hyperandrogenism, which is highly related to insulin resistance (IR) and obesity [1].

Abdominal obesity that appears to dominate in these cases is a major underlying factor. Women with PCOS were documented to have a higher waist circumference (WC) than BMI-matched controls, and 50–60% of cases have abdominal obesity [2]. Approximately 27% reduction in peripheral sensitivity to insulin was noticed in women with PCOS, independent of BMI [3]. Normal weight PCOS patients who had increased quantity of trunk fat are affected by abdominal obesity risks as overweight PCOS patients [4].

The β-cell dysfunction is associated with diminishing pancreatic insulin secretion, which in turn is associated with IR. Early detection and treatment of IR in the subgroup of patients with PCOS with normal weight could ultimately decrease the frequency or seriousness of dyslipidemia, hypertension, diabetes, and cardiovascular risks and is exceptionally critical

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not only for their follow-up but also for possible therapeutic modalities utilizing insulin sensitizers [5]. The homeostasis model assessment (HOMA) was used to measure IR. Pancreatic β-cell function and insulin sensitivity were calculated using HOMA calculator software [6].

Dumesic *et al.* [7] described a high percentage of fat in the abdomen in patients with PCOS with normal weight compared with normal weight patients without PCOS via dual-energy X-ray absorptiometry (DEXA). Ciala *et al.* [8] demonstrated that fat mass increase, especially in abdomen region, was associated with insulin resistance. DEXA can evaluate both total and regional fat mass with negligible radiation exposure [9]. In our study, DEXA was used, which is a fast and simple method for detection of both of total and regional fat.

Our study aimed to estimate body composition using DEXA in women with PCOS who have normal BMI.

PATIENTS AND METHODS

The study population consisted of 60 women with PCOS with the mean age of 28 ± 5.3 years, attending gynecology clinic between April 2015 and August 2017. Patients were eligible for inclusion if they had an ideal normal weight (BMI ≥18 but <25 kg/m²). Patients were also excluded if they had neoplastic, kidney or liver illness, diabetes, Cushing syndrome and endocrine disorder, or if they were on steroids, antiandrogenic drugs, statins, or insulin resistant-modifying drugs. Sixty healthy weight-matched women without PCOS were incorporated into the study as a control group. PCOS was diagnosed according to the current Rotterdam criteria [10]. The ethical committee approved the study, and informed consent was obtained from all the participants.

Evaluation included full examination with emphasis on medicinal history and use of medications. WC was estimated at the midpoint between the inferior border of the ribs and the iliac crest close to the umbilicus.

The cardiovascular risk factors were assessed by physical examination: resting blood pressure (diastolic and systolic). Participants were classified as having hypertension if they had a systolic blood pressure of at least 140 mmHg, diastolic blood pressure of at least 90 mmHg, or were taking medication for either high blood pressure or hypertension. Metabolic evaluation included total cholesterol, high-density lipoproteins (HDL), and low-density lipoproteins (LDL) and triglycerides. Samples of fasting blood insulin and glucose concentrations were collected. Insulin resistance was estimated by the homeostasis model assessment (HOMA-IR) method: [fasting insulin (μIU/ml)×fasting glucose (mg/dl)]/405. Using HOMA calculator software, β-cell function and insulin sensitivity were calculated [11]. Hormone estimation was performed between days 2 and 6 of a menstrual cycle in the control subjects or during a spontaneous bleeding episode or progestin-induced menstrual cycle in the patients with

PCOS. It includes serum follicle-stimulating hormone and prolactin, luteinizing hormone, serum progesterone, and total testosterone.

Body composition estimation was done using DEXA: lean mass and fat tissue content were estimated using total body scanning (Lunar Prodigy Advance; GE Medical Systems, Madison Wistosin, United States). The DEXA was calibrated daily before any scans utilizing a phantom as indicated by the standard methodology per manufacturer's guidelines. Total and regional fat contents including trunk and legs were evaluated. Analysis of data was done by using the manufacturer's validated software en CORE_V13.5_EN.

RESULTS

Means and standard division were calculated for all of the variables studied. The paired Student *t*-test was used to identify the significance of differences in variables with normal distribution between patients and controls. The probability level up to 0.05 was considered statistically significant whereas those up to 0.01 was considered highly significant. The analyses were performed using SPSS version 16.0 (SPSS Inc., Chicago, Illinois, USA).

Table 1 shows the anthropometric characteristics of patients with PCOS and controls. Patients with PCOS were older than controls (median range: 28.3 ± 5.3 versus 27.1 ± 2.3 years, $P = 0.737$) but had similar BMI (23.3 ± 3 vs. 23.2 ± 3.1 kg/m²) and weight (60.2 ± 6.4 vs. 60.1 ± 6.1 kg) (Table 1).

Table 2 shows the hormone levels between the two groups. There were significant increases in prolactin (11.06 ± 0.055 vs. 9.6 ± 0.1, respectively) and serum progesterone (2.4 ± 0.3 vs. 1.2 ± 0.5, respectively) in patients than control.

HOMA-IR in patients with PCOS (1.8 ± 0.025) was significantly higher than control (1.6 ± 0.02), and there was

Table 1. Anthropometric parameters in patients with polycystic ovary syndrome and controls

Anthropometric measure	Patients	Controls	*P*
Age	28.3±5.3	27.1±2.3	0.737
Weight (kg)	60.2±6.4	60.1±6.1	0.985
BMI (kg/m²)	23.3±3	23.2±3.1	0.97
Waist circumference (cm)	81.5±3.1	80.4±3.2	0.691

Table 2. Hormone parameters in patients with polycystic ovary syndrome and controls

Hormone parameters	Patients	Controls	*P*
FSH (IU/ml)	5.5±1.5	6.1±1.1	0.606
LH (IU/ml)	8.8±4.4	4.6±1.7	0.198
Prolactin (ng/ml)	11.06±0.055	9.6±0.1	<0.0001***
Serum progesterone (ng/ml)	2.4±0.3	1.2±0.5	0.023*
Testosterone (nmol/l)	2.3±0.2	2.1±0.1	0.196

***Correlation is significant at the 0.001 level; *Correlation is significant at the 0.05 level; FSH, follicle stimulating hormone; LH, luteinizing hormone

significantly lower β-cell level function in patients with PCOS (90.5 ± 0.2) than controls (97.3 ± 0.3) (Table 3).

The body composition analysis revealed greater total and trunk fat in patients with PCOS (32.4 ± 1.8) versus in controls (23.5 ± 1.7). Lean mass and leg fat show no difference between patients with PCOS and weight-matched controls (Table 4). Total body fat, trunk fat, leg fat, and lean mass were most closely associated with HOMA-IR (Table 5).

DISCUSSION

Our study used DEXA scans to examine the body composition of patients with PCOS who were not obese in comparison with a control group of healthy patients. Measurements, such as BMI and WC, are essential for large-scale surveys, and accurate and precise determinations of trunk fat may offer crucial additional information during clinical assessments. In the current study, total fat and regional fat in patients with PCOS were measured using the DEXA method. Previous studies demonstrated that

DEXA scan is a safe, accurate, and fast method [4]. The total fat and free-fat masses were measured in the trunk and upper and lower extremities [12]. The trunk fat was identified as the region under the chin, the area outlined by the vertical lines within the left and right glenoid fossae and bordering laterally to the ribs. Moreover, the part outlined by oblique lines that cross the femoral necks and converge below the pubic symphysis. The lower body fat included all fat under these oblique lines. In our study, we found that the trunk fat was higher (38.7 ± 2.1 vs. 31.1 ± 1.8, $P < 0.009$) in PCOS with normal weight than in control individuals without PCOS, which is a high value similar to those reported in other studies [13].

In the present study, anthropometric data showed no difference in BMI or WC in normal-weight PCOS and non-PCOS patients, although we observed an increase in their trunk fat in relation with controls as measured by DEXA. A similar result was obtained from Carmina *et al.* [4].

This study examined normal BMI women with PCOS and excluded obese and overweight individuals with increased BMI to avoid confusing factor of obesity. Regardless of total obesity status, trunk fat contributes to IR development with total resultant endocrine disorders such as PCOS, metabolic illnesses, and cardiovascular risks [13]. IR plays a significant and independent role in the pathogenesis of PCOS and the metabolic consequences of the disease [14,15]. One of the mechanisms that could contribute to IR is decreased insulin secretion owing to hypoactive or inactive β cells in the pancreas and ineffective signaling pathways of the insulin. Therefore, diagnosis of IR could significantly help the physicians to prevent many of its consequences. Although insulin resistance is not a diagnostic criterion for PCOS, it plays a vital role in its clinical characteristics. The prevalence of insulin resistance in patients with PCOS is 50–70% [16]. A total of 30–40% of patients with PCOS have impaired glucose tolerance test [17]. Hyperinsulinism along with insulin resistance was observed in 65% of obese women with PCOS, whereas insulin resistance is found in 20% of thin women with PCOS [18]. In our study, normal-weight women with PCOS showed differences in HOMA-IR, β-cell function, and insulin sensitivity compared with controls. These findings direct that trunk fat is one of the predisposing factors in inducing IR and disrupting insulin sensitivity in normal-weight women with PCOS and highlight the need to focus on end points other than body weight in diagnosis and treatment of women with PCOS.

Table 3. Metabolic parameters and body composition in patients with polycystic ovary syndrome and controls

Metabolic parameters	Patients	Controls	*P*
Fasting glucose (mg/dl)	85.9±11.5	85.5±10.6	0.967
Fasting insulin (mIU/l)	6.9±3.8	7.1±2.4	0.942
HOMA-IR	1.8±0.025	1.6±0.02	<0.0001***
β-Cell function%	90.5±0.2	97.3±0.3	<0.0001***
Insulin sensitivity%	114.0±2.3	109.2±2.1	0.056
Total cholesterol (mg/dl)	187±4.7	184±4.2	0.456
Triglycerides (mg/dl)	105±4.2	103±2.1	0.502
LDL (mg/dl)	121±2.5	120±1.5	0.584
HDL (mg/dl)	48±5.9	47.3±5.4	0.887

HDL, high-density lipoprotein; HOMA-IR, homeostasis model analysis insulin resistance; LDL, low-density lipoprotein.

Table 4. Body composition in patients with polycystic ovary syndrome and controls

Body composition	Patients	Controls	*P*
Total body fat%	32.4±1.8	23.5±1.7	0.003**
Trunk fat%	38.7±2.1	31.1±1.8	0.009**
Leg fat%	36.2±2.4	33.4±1.5	0.162
Lean mass%	46.8±3.1	58±10.1	0.14

**Correlation is significant at the 0.01 level

Table 5. Correlation analysis between body composition parameters and homeostasis model analysis insulin resistance, β cell function% and insulin sensitivity%

	HOMA-IR		β-Cell function%		Insulin sensitivity%	
	r	*P*	*R*	*P*	*r*	*P*
Total body fat%	1.00	<0.0001***	−1.00	0.005**	0.999	0.028*
Trunk fat%	1.00	<0.0001***	−1.00	0.005**	0.999	0.028*
Leg fat%	1.00	<0.0001***	−1.00	0.005**	0.999	0.028*
Lean mass%	−1.00	<0.0001***	1.00	0.005**	−0.999	0.028*

HOMA-IR, homeostasis model analysis insulin resistance. ***Correlation is significant at the 0.001 level. **Correlation is significant at the 0.01 level. *Correlation is significant at the 0.05 level.

Carmina *et al.* [4], reported that insulin resistance is associated with PCOS, with an estimated occurrence of 50–90% among people with PCOS. In the present study, there was no difference in HOMA-IR between patients with normal BMI with or without PCOS; however, an increase in trunk fat and a risk factor for insulin resistance was observed.

However, HOMA is commonly known as an indicator of IR and its value increases with an increase in resistance to insulin [19]. Results from the present study showed a significant ($P < 0.001$) correlation between HOMA-IR, total body fat, and trunk fat ratio. β-Cell dysfunction is related to decreases in pancreatic insulin secretion [19]. There is strong evidence that when excess insulin cannot compensate for the degree of IR, accordingly hyperglycemia becomes clinically significant, resulting in rushing in the decline of β-cell reserves [19]. β-Cell dysfunction affects patients with PCOS who are obese and those who are not, although this is not associated with glucose intolerance in most women [20]. Our findings support this concept; β-cell function% was 90.5 ± 0.2 in relation to 97.3 ± 0.3 in non-PCOS controls.

Regarding metabolic parameters, we could not detect a difference between HDL, LDL, and total cholesterol in normal-weight lean PCOS and controls without PCOS. Han *et al.* [21] study revealed that compared with BMI-matched and age-matched controls, lean women with PCOS have lower levels of HDL and higher levels of LDL than controls who were lean and were without PCOS. Lean women with PCOS have a 3–10% incidence of undiagnosed diabetes, with higher risk in obese patients [22].

Conclusion

Nonobese women with PCOS had a more significant total and trunk fat compared with non-PCOS women. DEXA scan is a fast and easy diagnostic tool to analyze body composition to follow those patients with high insulin resistance although they have normal BMI.

Recommendations

Further studies are needed to investigate such patients after decreasing their trunk fat and improving their insulin resistance through different interventions (medications, behavior modification, diet, and exercises).

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Conflicts of interest
There are no conflicts of interest.

References

1. Azziz R, Carmina E, Dewailly D, Diamanti-Kandarakis E, Escobar-Morreale HF, Futterweit W, *et al*. Force on the phenotype of the polycystic ovary syndrome of the androgen excess and PCOS society. Fertil Steril 2009;91:456–88.
2. Lim SS, Davies MJ, Norman RJ. Overweight, obesity and central obesity in women with polycystic ovary syndrome: a systematic review and meta-analysis. Hum Reprod Update 2012; 18:618–637.
3. Cassar S, Misso ML, Hopkines WG, Shaw CS, Teede HJ, Stepto NK. Insulin resistance in polycystic ovary syndrome: A systemic review and meta-analysis of euglycaemic–hyperinsulinaemic clamp studies. Hum Reprod 2016;31:2619–3.
4. Carmina E, Bucchieri S, Esposito A, Del PuentePasquale A, Mansueto P, Orio F *et al*., Abdominal fat quantity and distribution in women with polycystic ovary syndrome and extent of its relation to insulin resistance. J Clin Endocrinol Metab 2007;92:2500–5.
5. Matthews DR, Hosker JP, Rudenski AS, Naylor BA, Treacher DF, Turner RC. Homeostasis model assessment: insulin resistance and beta-cell function from fasting plasma glucose and insulin concentrations in man. Diabetologia 1985; 28:412–419.
6. Cersoosimo E, Solis-Herrera C, Trautmann ME, Malloy J, Trilitt CL. Assessment of pancreatic b-cell function: review of methods and clinical applications. Curr Diabetes Rev 2014; 10:2–42.
7. Dumesic DA, Akopians AL, Madrigal VK, Ramirez E, Margolis DJ, Sarma MK, *et al*. Hyperandrogenism accompanies increased intra-abdominal fat storage in normal weight polycystic ovary syndrome women. J Clin Endocrinol Metab 2016; 101:4178–4188.
8. Kozakowski J, Zgliczynski W. Body composition, glucose metabolism markers and serum androgen-association in women with polycystic ovary syndrome, Endokrynologia Polska 2013;64:94–100.
9. Frossing S, Nylander MC, Chabanova E. Quantification of visceral adipose tissue in polycystic ovary syndrome: dual energy X-ray absorptiometry versus magnetic resonance imaging: background polycystic ovary syndrome (PCOS) is associated with frequent overweight and abdominal obesity. Acta radiol 2017; 59:13–17.
10. Rotterdam ESHRE/ASRM-Sponsered PCOS Consensus Workshop Group. Revised 2003 consensus on diagnostic criteria and long-term health risks related to polycystic ovary syndrome. Fertil Steril 2004; 81:19–25.
11. University of Oxford, Oxford (UK). HOMA Calculator. Diabetes Trials Unit- The Oxford Centre for Diabetes, Endocrinology, and Metabolism. Available at: *https://www.dtu.ox.ac.uk/homacalculator*. [Accessed 12 October 2015].
12. Vega GL, Adams-Huet B, Peshock R, Shah B, Grundy SM. Influence of body content and fat distribution on variation in metabolic risk. J Clin Endocrinol Metab 2006; 91:4459–4466.
13. Macruz CF, Lima SM, Salles JE, da Silva GM, Scalissi NM. Assessment of the body composition of patients with polycystic ovary syndrome using dual-energy X-ray absorptiometry. Int J Gynaecol Obstet 2017; 136:285–9.
14. RamezaniTehrani F, Montazeri SA, Hosseinpanah F, Cheraghi L, Erfani H, Tohidi M, *et al*. Trend of cardio-metabolic risk factors in polycystic ovary syndrome: a population-based prospective cohort study. PLoS One 2015; 10:e0137609.
15. Stepto NK, Cassar S, Joham AE, Hutchison SK, Harrison CL, Goldstein RF, *et al*. Women with polycystic ovary syndrome have intrinsic insulin resistance on the euglycaemic-hyperinsulinaemic clamp. Hum Reprod 2013; 28:777–784.
16. Dunaif A. Insulin resistance and the polycystic ovary syndrome: mechanism and implications for pathogenesis. Endocr Rev 1997; 18:774–800.
17. Legro RS, Kunselman AR, Dodson WC, Dunaif A. Prevalence and predictors of risk for type 2 diabetes mellitus and impaired glucose tolerance in polycystic ovary syndrome: a prospective, controlled study in 254 affected women. J Clin Endocrinol Metab 1999; 84:165–169.
18. Chang RJ, Nakamora RM, Judd HL, Kaplan SA. Insulin resistance in non-obese patients with polycystic ovary syndrome. J Clin Endocrinol Metab 1983; 57:356–359.
19. Cersosimo E, Solis-Herrera C, Trautmann ME, Malloy J, Triplitt CL. Assessment of pancreatic b-cell function: review of methods and clinical applications. Curr Diabetes Rev 2014; 10:2–42.
20. Diamanti-Kandarakis E, Dunaif A. Insulin resistance, and the polycystic ovary syndrome revisited an update on mechanisms and implications. Endocr Rev 2012; 33:981–1030.
21. Han Y, Kim HS, Lee HJ, Oh JY, Sung YA. Metabolic effects of polycystic ovary syndrome in adolescents. Ann Pediatr Endocrinol Metab 2015; 20:136–142.
22. Melmed S, Jameson JL. Disorders of the anterior pituitary and hypothalamus. In: Kasper DL, Braunwald E, Fauci AS, Hauser SL, Longo DL, Jameson JL, editors. *Harrison's principles of internal medicine*. 16th ed. New York, NY: McGraw Hill; 2008. pp. 2076–2097.