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Which Is More Reliable In Polycystic Ovarian Syndrome: Body Mass Index or Waist Hip Ratio?

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Abstract

This was a prospective study, to compare between body mass index (BMI) and waist hip ratio (WHR) in polycystic ovarian syndrome (PCOS). 120 PCOS patients, infertile and anovulatory, have been chosen. The mean age of the participating patients was ± 25 years ± 5.64 SD. Measurements of WHR were taken by one person. Body mass index was calculated in each patient using the formula BMI = weight (kg) / height (m^2). On the second day of menstrual cycle, vaginal ultrasound was done for ovarian volume and microcysts numbers, and the following hormones were assayed: LH, FSH, oestradiol, prolactin, androstenedione, total testosterone, free testosterone and insulin. 72.5% of the studied PCOS patients their BMI were > 28 kg/m^2, and 82.5% their WHR were > 0.8. WHR correlated significantly with androstenedione (r = 0.271, P = 0.002), ovarian cyst number (r = 0.311, P = 0.000) and with the duration of infertility (r = 0.159, P = 0.044). On the other hand, it was negatively correlated significantly with FSH (r = - 0.236, P = 0.005) and prolactin (r = - 182, P = 0.034).). BMI was positively correlated with prolactin level (r = - 0.195, p = 0.023). There was no significant correlation between either BMI or WHR and serum levels of oestradiol (r = - 111, P = 0.205 and r = - 0.041, P = 0.641, respectively), insulin (r = 0.095, P = 0.275 and r = 0.035, P = 0.691, respectively), LH (r = 0.074, P = 0.382 and r = 0.82, P = 0.328, respectively) and total testosterone (r = - 0.045, P = 0.61 and r = 0.055, P 0.536, respectively) or free testosterone (r = - 0.021, P = 0.812 and r = 0.163, P = 0.061, respectively. Neither BMI nor WHR had significant correlation with the ovarian volume. But, the difference in WHR was significant between patients with ovarian volumes ≤ 9 ml and > 9 ml.

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In addition, total testosterone level increased significantly in patients with WHR > 0.8. This study indicated that WHR is a good mirror to the androgen levels and ovarian morphologies in PCOS patients. So, it is more reliable in reflecting the clinical features, endocrine profiles, ovarian characteristics and metabolic status of PCOS that are independent of BMI.

Keywords: Anovulation; hyperandrogenism; hyperinsulinaemia; obesity; polycystic ovarian syndrome.

1. Introduction

Polycystic ovary syndrome (PCOS) is defined by the National Institutes of Health (NIH) in April 1990, as a disorder having: i) hyperandrogenism and/or hyperandrogenemia, ii) oligo-ovulation, and iii) exclusion of known disorders. Alternatively, in Rotterdam [1] PCOS is defined, after the exclusion of related disorders, by two of the following three features: i) oligo- or anovulation, ii) clinical and/or biochemical signs of hyperandrogenism, or iii) polycystic ovaries. In essence, the Rotterdam 2003 expanded the NIH 1990 definition creating two new phenotypes: a) ovulatory women with polycystic ovaries and hyperandrogenism, and b) oligo-anovulatory women with polycystic ovaries, but without hyperandrogenism.

To date, PCOS remains a syndrome and, as such, no single diagnostic criterion (such as hyperandrogenism or PCO) is sufficient for clinical diagnosis. Known disorders which mimic the PCOS phenotype should be excluded.

Patients with PCOS have increased proportion of central to peripheral fat ratio; android pattern fat distribution. There is a positive correlation among trunkal fat and insulin [2]. Moreover, obesity impairs insulin resistance and exacerbates reproductive and metabolic features of PCOS. The distribution of body fat has significant effects on different clinical, hormonal and metabolic features that are independent of body weight. Increased WHRs are association with increased high levels of triglycerides and very low density lipoproteins, and lower levels of high density lipoproteins [3]. Obesity accentuates the metabolic alternations in PCOS, and is frequently an associated condition, either being an intrinsic component or a predisposing factor for the insulin resistance [4].

It has been reported that in obese PCOS patients, losing as little as 5 % of the initial body weight, improves spontaneous ovulation rates and spontaneous pregnancy. Weight reduction remains a worthwhile approach in obese women with PCOS [5, 6]. In this research, a comparison was made between body mass index and waist hip ratio in patients with PCOS.

2. Patients and Methods

2.1. Patients

This study was done in OB/GYN department of Benha Teaching Hospital. A total of 120 PCOS patients were selected. The mean age of the participating patients was ± 25 years ± 5.64 SD. Most of the patients (76.5%) their ages were between 20 and 30 years**.** All the patients were seen because of infertility and diagnosed to have

PCOS. They had spontaneous onset of puberty and normal sexual development. 74 patients (70%) had primary infertility and 36 patients (30%) had secondary infertility. The mean duration of infertility was 3.8 years ± 2.7 SD.

The diagnosis of PCOS was based on the clinical diagnosis of PCOS and ultrasound evidences of polycystic ovary (PCO). The clinical diagnosis was based on the presence of at least two of the following criteria: oligo-amenorrhoea, obesity (BMI ≥ 28), and clinical signs of hyperandrogenism or elevated either LH or LH/FSH ratio. Ultrasound diagnosis of PCO was based on the presence of ≥ 5 microcysts distributed peripherally and the hyperechoic stromal density of both ovaries.

2.2. Clinical Examination

Each patient underwent a screening history and physical examination. Measurements of waist to hip ratio were taken by one person. While standing up and relaxed, the smallest area around the waist using a measuring tape was taken (usually just above or around the umbilicus) and the circumference of hips (the widest diameter of the hips, usually at the levels of bony prominences or the level of greater trochanters of both femurs). Body mass index (BMI) was calculated in each patient using the formula BMI = weight (kg) / height (m^2).

The menstrual cycle was described as being either regular, oligomenorrhoeic (a cycle interval of longer than 35 days but less than 6 months) or amenorrhoeic (no menstruation for 6 months or more). mean

2.3. Ultrasound Examination

All women had undergone transvaginal ultrasound, Medison, SonoaceR5. Ultrasound was done in the early follicular phase between the 2^{nd} and 3^{rd} day of the menstrual cycle. In amenorrhoeic women, the investigations were performed after progestogen induced withdrawal bleeding.

The ovary was defined as being polycystic if there were multiple (more than four), small (2-8 mm) cysts arranged peripherally with increased echogenicity of the stroma. In addition, the ovary was measured in three planes, then the ovarian volume was calculated according to the formula for a prolate ellipsoid: (d1) x (d2) x (d3) x 0.5233, where the d1, d2, d3 are the three maximal longitudinal, anteroposterior and transverse diameters. Ovaries were considered enlarged when the ovarian volume exceeded 9 ml. Ovaries containing cysts (a dominant follicle, simple cyst, haemorrhagic cyst, endometrioma, and corpus luteum) > 14 mm in diameter were excluded. The number of small follicles in each ovary and the maximal diameter of follicle were also calculated carefully. The patients were divided into three groups ultrasonographically; group (I) (n = 36), in which less than 10 cysts were identified; group (II) (n = 48), in which there were between 10 and 14 cysts located under the ovarian capsule; group (III) (n = 36), in which more than 14 cysts were found under the ovarian capsule.

2.4. Hormonal Measurements

The following hormones were assayed on the second day of menses: LH, FSH, oestradiol, prolactin,

androstenedione, total testosterone, free testosterone and insulin.

Serum FSH and LH were measured using an immunoradiometric assay (IRMA CT cat. KP6CT) on solid phase (coated tube) (RADIM S.p.A Via del Mare, Pomezia "Roma"- Italia). The sensitivity was 0.18 and 0.20 mIU/ml, respectively. Serum 17β oestradiol was measured using radioimmunoassay (RIA) cat. KS25CT (RADIM S.p.A. Via del Mare, Pomezia "Roma"- Italia). The sensitivity was 10 pg/ml.

Androstenedione was measured by radioimmunoassay using Coat-A-Count Direct Androstenedione procedure (Diagnostic Products Corporation, Los Angeles, CA, USA). The sensitivity was approximately 0.04 ng/ml (0.14 nmol/L). Serum total testosterone was measured by radioimmunoassay (RIA) cat. KS24CT (RADIM S.p.A. Via del Mare, Pomezia "Roma"-Italia). The sensitivity was 0.017 ng/ml. Free testosterone was measured by the DSL-4900 ACTIVE^{TM} Free Testosterone Coated-Tube Radioimmunoassay Kit (Corporate Headquarters, Webster, Texas, USA). Sensitivity was 0.18 pg/ml.

Serum insulin was measured by the DSL-1600 Insulin Radioimmunoassay Kit (Corporate Headquarters, Webster, Texas, USA). Sensitivity was 1.3 µIU/ml.

Serum prolactin was measured by immunoradiometric assay (IRMA CT cat. KP8CT) on solid phase (coated tube) (RADIM S.p.A. Via del Mare, Pomezia "Roma"-Italia). The sensitivity was 1 ng/ml (30 µIU/ml). All the results are expressed as mean.

2.5. Statistics

Analysis of data was performed using the Statistical Package for Social Sciences computer program (SPSS/PC+). The data were analyzed using the analysis of variance (ANOVA). Pearson's product-moment coefficient *r* was also used to test the correlation between the endocrine and the biophysical data after log transformation. Analysis of variance after log transformation and chi-square was used in addition to t-Test and Z-Test, when appropriate, to test the differences between groups.

3. Results

3.1. Figures and Tables

Most of the selected patients (87/120) (72.5%), their BMI were > 28 kg/m^2, and (99/120) (82.5%) their WHR were > 0.8.

There was no significant difference in the BMI between the groups of ovarian cysts or the sub-groups of the ovarian volume. However, the difference in WHR was significant between the two subgroups of ovarian volume (t-test). The WHR in the groups of the ovarian cysts, unlike BMI, was apparently increasing from group (I) to group (III) **(Table 1)**.

Table 1: Differences between BMI and WHR patients depending on the ultrasound findings:

Variable	Ovarian cysts			Ovarian volume	
	Gr.I (<10) (n=36)	Gr.II (10-14) (n=48)	Gr.III (>14) (n=36)	Subgr. ≤ 9ml (n=42)	Subgr. > 9ml (n=78)
BMI	31.9	30.9	31.4	30.6	31.7
WHR	**0.84**	**0.86**	**0.89**	0.851	0.861 +

Gr. = Group (according to the number of microcysts in the ovary). Subgr. = Subgroup (according to the ovarian volume). (+) $P < 0.05$ between subgroups of the ovarian volume (t-test).

Table 2: Hormonal differences between the BMI and WHR in the studied PCOS patients:

Hormone	WHR		BMI	
	≤ 0.8 (n=21)	> 0.8 (n=99)	≤ 28 (n=33)	>28 (n=87)
LH	**10.9**	**7.68**	8.69	8.07
FSH	5.53	4.89	4.59	5.16
Estradiol	100.7	80.96	71.08	90.11
Total testosterone	**0.96**	**1.1 (+)**	1.15	1.05
Free testosterone	**2.82**	**4.04 (a)**	3.69	3.71
Androstenedione	**2.9**	**3.95**	3.44	3.43
Prolactin	443.6	314.5	446.6	295.6
Insulin	**7.8**	**13.5**	**10.5**	**12.9**

N.B.: (+) $P<0.05$, (a) $P = 0.081$ Comparing values between corresponding subgroup (t-Test).

There were no significant differences in the hormonal levels between the patients of BMI ≤ 28 and > 28. On the other hand, the differences were obvious between the patients depending on WHR **(Table 2)**. Total testosterone was significantly higher in patients with WHR > 0.8. The LH level was higher, and the androgens levels and insulin levels are lower in patients with WHR ≤ 0.8 than in patients with WHR > 0.8.

There was no significant correlation between the menstrual patterns of PCOS patients and WHR or BMI. However, BMI were apparently less in the patients with normal menses than in patients with amenorrhoea or oligomenorrhoea **(Table 3)**.

Table 3: Differences between BMI and WHR depending on the menstrual presentation in PCOS patients:

Variable	PCOS patients		
	Amenorrhea (n=42)	Oligomenorrhoea (n=63)	Regular (n=15)
BMI	32.3	32	**28.1**
WHR	0.87	0.85	0.89

3.2. Correlations

Neither BMI nor WHR had significant correlation with ovarian volume. However, only WHR was significantly correlated with ovarian cyst number (r = 0.311, P = 0.000), androstenedione (r = 0.271, P = 0.002), and with the duration of infertility (r = 0.159, P = 0.044). On the other hand, WHR was negatively correlated significantly with FSH (r = - 0.236, P = 0.005) and prolactin (r = - 182, P = 0.034). There was no significant correlation between either BMI or WHR and serum levels of oestradiol (r = - 111, P = 0.205 and r = - 0.041, P = 0.641, respectively), insulin (r = 0.095, P = 0.275 and r = 0.035, P = 0.691, respectively), LH (r = 0.074, P = 0.382 and r = 0.82, P = 0.328, respectively) and total testosterone (r = - 0.045, P = 0.61 and r = 0.055, P 0.536, respectively) or free testosterone (r = - 0.021, P = 0.812 and r = 0.163, P = 0.061, respectively). Only, BMI was correlated with prolactin level (r = - 0.195, p = 0.023). FSH was negatively correlated significantly with the WHR (r = 0.236, P = 0.005).

4. Discussion

In this research, WHR was significantly correlated positively with ASD, ovarian cyst numbers, duration of infertility and negatively with FSH and prolactin. Also, the difference in WHR was significant between patients with ovarian volumes ≤ 9 ml and > 9 ml. In addition, total testosterone level was significantly higher in PCOS patients with WHR > 8. Moreover, there were obvious increases of free testosterone, ASD and insulin levels in PCOS patients with WHR >8. Our findings are supporting the android pattern of fat distributions in those patients with WHR > 0.8 and the reliability of WHR in reflecting the clinical, hormonal and metabolic features of PCOS that are independent of body weight.

On the other hand, and apart from grouping and subgrouping systems of the ultrasound findings, there was no significant correlation between WHR or BMI and ovarian volume, oestradiol, insulin, LH and total and free (T). The authors in [3] have reported that increased WHRs were associated with increased LH and ASD concentrations. Abdel Gadir and his collegues [7] did not find significant correlation between BMI and FSH, oestradiol, T, prolactin and ovarian volume. In another study done by the authors in [8], no relationship could be established between BMI and follicle number, ovarian volume and testosterone.

The authors in [9, 10] have another opinion that the number of follicles per ovary have been correlated

positively with BMI. In addition, BMI had significant correlation with the ovarian volume and testosterone concentration [11], free testosterone [12, 13] and with insulin levels [13]. In another research [9], the ovarian volume was positively correlated with BMI and WHR. However, increase BMI was not always associated with high glucose and insulin levels while high WHR tended to be so [14]. In this research, the insulin levels were higher in those patients with BMI >28 and WHR > 0.08 than in their counterparts. Although the relations were not significant, it may support the common view of the positive relationship between obesity and hyperinsulinaemia. Obese PCOS patents have significantly higher WHR than reference subjects with similar BMI, and are related positively with their insulin levels, whereas the WHR of non-obese PCOS and reference subjects are not different [14].

The authors in [7, 15] have shown a negative association between BMI and LH. In this research, in spite of the relation was not significant, the levels of LH in patients with WHR $\leq$ 0.8 were higher than in patient with WHR > 0.8. This may suggest possible different etiology and pathogenesis between the two sub-groups.

Our findings, supported by other literatures [7, 8, 14], may suggest the limitation of BMI in reflecting the metabolism of PCOS women, and the potential usefulness of the WHR.

5. Conclusion

It is better to include WHR in addition to the BMI as routine measurements in PCOS patients. WHR is a good mirror to the androgen levels and ovarian morphologies in PCOS patients. So, it is more reliable than BMI in reflecting the clinical features, endocrine profiles, ovarian characteristics and metabolic status in PCOS.

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References

[1] The Rotterdam ESHRE/ASRM-Sponsored PCOS Consensus workshop group (2004). "Revised 2003 consensus on diagnostic criteria and long-term health risks related to polycystic ovary syndrome." Fertil Steril., 81:19–25.

[2] Godoy-Matos AF[1], Vaisman F, Pedrosa AP, Farias ML, Mendonça LM, Pinheiro MF. (2009). "Central-to-peripheral fat ratio, but not peripheral body fat, is related to insulin resistance and androgen markers in polycystic ovary syndrome." Gynecol Endocrinol., Dec; 25(12):793-8.

[3] Pasquali R., Casimirri F., Venturoli S., Antonio M., Morselli L., Reho S., Pezzoli A. and Paradisi R. (1994). "Body fat distribution has weight-independent effects on clinical, hormonal, and metabolic features of women with polycystic ovary syndrome." Metabolism, 43:706-713.

[4] Carmina E., Wong L., Paulson R,J., Dsauer M.V., Stanczyk F.Z. and Lobo R.A. (1997). "Endocrine abnormalities in ovulatory women with polycystic ovaries on ultrasound." H.Reprod., 12:905-909.

[5] Motta AB. "The role of obesity in the development of polycystic ovary syndrome. (2012). "Curr Pharm Des., 18(17):2482-91.

[6] Saravelos H., Li T.C., Bontis J., Basil C., Tarlatzis and Cooke I.D. (1997). "Laparoscopic management of polycystic ovarian syndrome." Gynaecol. Endoscopy, 6:331-340.

[7] Abdel Gadir A., Khatim M.S., Mowafi R.S., Alnaser H.M., Muharib N. S. and Show W.R. (1992). "Implications of ultrasonically diagnosed polycystic ovaries." I. Correlations with basal hormonal profiles. Hum. Reprod., 7:453-457.

[8] Pache T.D., De-Jong F.H., Hop W.C. and Fauser B.C. (1993). "Association between ovarian changes assessed by transvaginal sonography and clinical and endocrine signs of the polycystic ovary syndrome." Fertil. Steril., 59:544-549.

[9] Sikka P, Gainder S, Dhaliwal LK, Bagga R, Sialy R, Sahdev S. (2007). "Ultrasonography of the ovaries and its correlation with clinical and endocrine parameters in infertile women with PCOS." Int J Fertil Womens Med. Jan-Feb; 52(1):41-7.

[10] Takahashi K., Eda Y., Okada S., Abu-Musa A., Yoshino K. and Kitao M. (1993). "Morphological assessment of polycystic ovary using transvaginal ultrasound." Hum. Reprod., 8: 844-849.

[11] Balen A.H., Conway G.S., Kaltsas G., Techatraisak K., Manning P.J., West C. and Jacobs H.S. (1995). "Polycystic ovary syndrome: the spectrum of the disorder in 1741 patients." Hum. Reprod., 10:2107-2111.

[12] Gjönnaess H. (1994). "Ovarian electrocautery in the treatment of women with polycystic ovary syndrome." Acta Obstet. Gynaecol., Scand., 73:407-412.

[13] Yoshino K., Takahashi K., Eda Y., Okada S. and kitao M. (1993). "Plasma androgens and sex hormone binding globulin in women with polycystic ovaries diagnosed by transvaginal ultrasound." J. Reprod. Med., 38: 858-862.

[14] Norman R.J., Masters S.C., Hague W.M., Beng C.,Pannal P. and Wang X.J. (1995). "Metabolic approaches to subclassification of polycystic ovary syndrome." Fertil. Steril., 63: 329-335.

[15] Insler V., Shoham Z., Barash Z., Koistinen R., Seppala M., Hen M., Lunenfeld B. and Zadik Z. (1993). "Polycystic ovaries in non-obese and obese patients: possible pathophysiological mechanism based on new interpretation of facts and findings." Hum. Reprod., 8: 379-384.