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The Clinical, Metabolic and Endocrine Parameters of Obese and Non-obese Polycystic Ovarian Syndrome women. A Prospective Comparative Study

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Abstract

Background: Frequent co-occurrence of obesity and polycystic ovarian syndrome (PCOS) results in a synergistic negative impact on the clinical, biochemical, and metabolic status.

Objective: The study aimed to compare the clinical, metabolic, and endocrine parameters of the obese (BMI≥30 Kg/m2) to that of non-obese PCOS women (BMI<30).

Materials and Methods: Prospective observational comparative study conducted on two hundred and ten PCOS women between May 2023 and May 2024. The study aimed to compare the clinical, metabolic, and endocrine parameters of the obese (BMI≥30 Kg/m2) to that of non-obese PCOS women (BMI<30), Homeostatic Model Assessment of Insulin Resistance (HOMA-IR) used to estimate insulin resistance.

Results: 97 (46.2%) were obese and 113 (53.8 %) were non-obese PCOS women. No significant difference was found in the prevalence of menstrual irregularity or clinical evidence of hyperandrogenism between the two groups of PCOS women. Whereas, the obese PCOS women had a significantly higher prevalence of insulin resistance (Fisher' Exact = 0.01) and fasting insulin level (p=0.028). The insulin resistance also was significantly higher in the obese PCOS women (HOMA-IR= 3& = 2.4 respectively and p= 0.01). Both groups were comparable with regard to FSH, estradiol, testosterone, DHEA, and AMH. Non-obese women have a significantly higher level of LH (p=0.04) than obese PCOS women. BMI had a significant positive correlation with HOMA-IR (p= 0.01) and a significant negative correlation with LH (p= 0.04).

Conclusion: Obese PCOS have a higher LH, insulin level, and higher insulin resistance than non-obese PCOS women and this adversely affects the reproductive and metabolic status. Therefore, targeting obesity in PCOS women will help to minimize these adverse outcomes.

Keywords: PCOS; menstrual irregularity; hyperandrogenism; BMI; insulin resistance.

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Original article

Introduction

Polycystic ovary syndrome (PCOS) is the most common endocrinopathy affecting women of reproductive age with a prevalence of 6–10% using Rotterdam's criteria (1, 2). PCOS has a wide spectrum of clinical and endocrinological manifestations and adversely affects metabolic and reproductive health (1, 3-5).

Infertility is the main presenting complaint as it affects about 40% of PCOS women (6). Even, if pregnancy is achieved, those women are at increased risk of reproductive problems; such as miscarriage, reported in up to 30% of PCOS pregnant women (7, 8), gestational diabetes mellitus; affects about 40–46% of pregnant PCOS women (9) and gestational hypertensive disorders affects up to 28.5% of PCOS patients (10, 11). Furthermore, unopposed hyper-estrogenemia can cause endometrial hyperplasia or endometrial cancer (12, 13). A causal association between PCOS and an increased risk of breast cancer was also mentioned (14).

PCOS affects both obese and non-obese women (15). 30–75% of PCOS women were reported to be obese (4, 16). Obesity and raised BMI have an adverse effect on fertility outcomes and are considered the key basis of metabolic complications such as metabolic syndrome, diabetes, hypertension, and cardiovascular diseases (16, 17). However, a meta-analysis conducted on 22 full-text researches reported that non-obese PCOS women are at increased risk of metabolic disorders as well as long-term metabolic complications than non-obese non-PCOS women (18). This report could confirm the theory of an endogenous metabolic dysfunction of PCOS independent of obesity.

Both PCOS and obesity adversely affect glycaemic control and endocrine status; PCOS women with raised BMI have an increased total testosterone, free androgen index, increased insulin resistance, and decreased sex hormone-binding globulin (19, 20). Furthermore, BMI was found to be significantly associated with all manifestations of PCOS (21). The metabolic and reproductive outcomes in PCOS women were found to be significantly worsened by obesity (19). In support of this, it was found that even a 5% weight loss in PCOS women causes a significant clinical improvement in androgenic, metabolic, and reproductive disturbances (22, 23).

In PCOS genetically predisposed women, weight gain and obesity are important risk factors for the clinical and biochemical manifestations of this syndrome (24). In support of this, data from the Northern Finland Birth Cohort (NFBC) reported that body mass index (BMI) was significantly associated with manifestations of PCOS at all ages (21). The frequent co-occurrence of both obesity and PCOS (21, 25) results in a synergistic negative impact on the clinical, biochemical, and metabolic status. Therefore, this study was conducted to compare the clinical, metabolic, and endocrine characteristics of obese and non-obese PCOS women.

Materials and Methods

This prospective comparative study was conducted on two hundred and ten PCOS women who attended the out-patient clinic at Albayda Fertility Teaching Governmental Centre and Al-Tafual Private Centre in Albadya/Libya between May 2023 and May 2024.

Ethical approval was obtained from Al-Mukhtar Committee for Bio-safety and Bioethics (MCBB) reference number (NBC: 007. H. 23. 10) and verbal consent for participation in the study was obtained from all the participants before the commencement of the study.

Two hundred and ten women were diagnosed as PCOS patients using Rotterdam criteria [2]; the diagnosis was established when the participant fulfilled at least two out of the three following criteria: (1) oligo-/anovulation, (2) clinical and/ or biochemical hyperandrogenism (3) polycystic ovary on ultrasound. Those with medical or endocrinological problems likely to affect glycaemic control or those using insulin sensitizers in the two months preceding the commencement of the study were excluded.
Detailed history and physical examination were carried out and recorded on prepared proforma. Variables including age, menstrual irregularity, and clinical features of hyperandrogenism (hirsutism, acne, etc.) were recorded.

Weight and height were measured and body mass index was calculated using the following formula [Weight (Kg) / Height(m)2. In the current study; the participants were divided into two groups; obese (BMI ≥ 30) and non-obese (normal BMI: 18.5-24.9

and overweight with BMI between 25 and 29.9). Ovaries were examined by transvaginal ultrasound and defined as polycystic if there were≥ 12 follicles measuring < 10 mm in diameter per ovary.

Measurements and laboratory analyses

Serum FSH, LH, and estradiol were measured on the second day of the spontaneous or induced menstrual cycle. Serum testosterone, DHEA, AMH, vitamin D, TSH, and prolactin were analyzed for the subjects. Over-night fasting serum insulin and glucose were measured and insulin resistance was calculated using the Homeostatic Model Assessment of Insulin Resistance (HOMA-IR).
HOMA-IR= fasting insulin (mIU/L) × fasting plasma glucose (mg/dL)/405 (26).
A cutoff value of 2.6 was used to identify individuals at risk of insulin resistance (27).

Statistical Analysis

Statistical Package for the Social Sciences (SPSS) software, version-26 was used for data collection and analysis. The number and percentages were computed for qualitative variables; symptoms attributed to PCOS (oligomenorrhea, acne, and hirsutism), unilateral or bilateral PCO, and normal and abnormal HOMA-IR. Shapiro-Wilk test is used to test the distribution of quantitative data. The quantitative variables including; age, body mass index (BMI), basal FSH, LH and estradiol levels, prolactin, TSH and testosterone levels, vitamin D, fasting insulin, glucose, and HOMA-IR were presented by mean and standard deviation or medium and range for normally distributed and skewed data respectively. A t-test used to compare mean values of normally distributed data and the Mann-Whitney U test was used to compare the skewed data among the two study groups. Fisher's exact test was used to compare the qualitative data. Pearson's and Spearman's tests were used to study the correlation between normal and skewed data respectively. P values < 0.05 were considered as significant.

Results

A total of two hundred and ten women with PCOS visited Albayda Teaching Fertility Governmental Centre and Al-Tafual Private Centre in Albadya/Libya during the study period; from May 2023 to May 2024. Of whom 97 (46.2%) were obese PCOS women and 113 (53.8 %) were non-obese. The mean age and standard deviation of the included women were 27.5 (5.5) years and their BMI ranged between 19 and 62%.

On a transvaginal ultrasound scan (TVS), the polycystic appearance of the ovaries was found in 97% of the whole population. 92.6% of PCOS women had a bilateral polycystic ovary and 7.4% had unilateral PCO-like ovary. The mean and standard deviation of FSH and LH of the whole study group were 6.4 (1.9) and 9.7 (18) mIU/ml respectively. The mean serum prolactin was normal 22.9 (17) mIU/ml and also the level of TSH was within normal with a mean of 2 (1.2) Pmol/L. The total testosterone ranged between 0.03 and 5 mIU/ml with a mean of 0.6 (0.6) mIU/ml and the mean level of DHEA was 269 (169) mIU/ml for all the included participants. The AMH ranged between 0.9 and 12.3 ng/ml and the mean level of vitamin D was 20 (11) ng/ml. The mean fasting blood glucose was 85 (14) mg/dl and the mean fasting insulin and HOMA-IR were 12.4 (7.2) and 2.7 (1.7) respectively. The age of the two groups was comparable; 27.5 (5.2) years for the non-obese group and 27.5 (5.9) years for the obese PCOS women with a p-value of 0.9. The mean BMI of the obese group was 35.4 (5.2) Kg/m2 and that for the non-obese PCOS was 25 (3.4) kg/m2 and the difference was statistically significant (P value < 0.001).

Almost all the included women were infertile. Regarding the frequency of menstrual irregularity and clinical symptoms of hyperandrogenism, seven women were asymptomatic, and six out of the seven non-symptomatic women were non-obese PCOS. 57.3% of the patients were presented with both menstrual irregularity and a clinical picture of hyperandrogenism, about 22% of the women presented with amenorrhea-oligomenorrhea alone, and 17.5% of PCOS patients were presented only with clinical picture of hyperandrogenism. However, there was no significant difference between the two studied groups in the frequency of the presenting symptoms (Fisher's Exact 5.299 and p-value= 0.146) Figure 1.

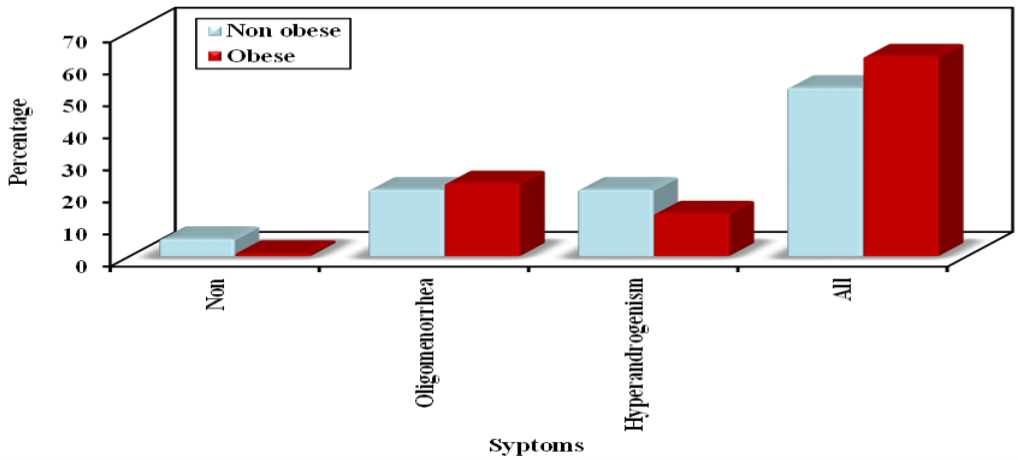

Figure 1. Comparison between the two studied groups according to symptoms

As shown in Table 1; there was no significant difference between the two study groups with regard to the analyzed hormones (FSH, E2, TSH, prolactin). The difference in the androgens (total testosterone and DHEA) between the two groups also did not reach a significant level. There was no significant difference between the two groups in the level of vitamin D and AMH whereas the non-obese PCOS women had a significantly higher level of LH level (P-value of 0.04).

The prevalence of insulin resistance (IR) in the whole study group was 40% and IR prevalence was significantly
higher among the obese PCOS than non-obese PCOS (49.5% and 31.9% respectively with Fisher's Exact test= 0.01). Table 2 demonstrates the difference in the glycemic indices between the two groups.

Table 1: Comparison between the two studied groups according to endocrine status

	Non obese	Obese	U	p
FSH	2.36 – 13.40 6.10 (5.0 – 7.40)	1.63 – 13.40 6.10 (5.14 – 7.30)	4853.0	0.764
LH	2.16 – 27.50 7.88 (5.65 – 11.15)	1.50 – 255.0 6.90 (5.50 – 8.70)	4191.50*	.043*
E2	8.0 – 191.0 40.0 (30.0 – 68.0)	0.95 – 361.0 42.0 (31.0 – 69.0)	2613.500	0.940
Prolactin	0.78 – 84.0 20.0 (11.25 – 30.30)	1.0 – 134.0 17.35 (12.44 – 25.50)	4909.0	0.467
TSH	0.39 – 5.50 1.70 (1.10 – 2.76)	0.23 – 8.20 1.87 (1.26 – 2.61)	4825.50	0.742
Testosterone	0.03 – 5.0 0.45 (.28 – .60)	0.10 – 2.96 0.49 (.33 – .70)	4489.0	0.355
DHEA	35.55 – 941.0 245.0 (179.0 – 302.0)	42.04 – 760.0 228.5 (149.0 – 300.0)	4231.50	0.394
AMH	0.88 – 14.40 4.05 (2.94 – 6.75)	1.10 – 14.40 3.90 (2.84 – 6.36)	4551.0	0.741
Vit. D	3.0 – 45.0 15.20 (10.84 – 25.0)	5.0 – 81.0 21.0 (13.30 – 28.0)	3953.50	0.060

U: Mann Whitney test.
p: p-value for comparing between the two studied groups. *: Statistically significant at p ≤ 0.05.

Table 2: **Comparison between obese and non-obese PCOS women in glycemic indices and IR**

	Non-obese	Obese	Test of Sig.	p-value
Fasting glucose	84.79 ± 13.94	86.03 ± 14.29	t= 0.636	0.525
Fasting insulin	2.0 – 32.40 10.0 (8.0 – 12.90)	2.10 – 45.0 12.50(7.60 – 18.90)	U= 4517.50	0.028*
HOMA-IR	2.40 ± 1.37	3.02 ± 2.16	t= 2.2	0.01*

U: Mann Whitney test
t: Student t-test
p: p-value for comparing between the two studied groups *: Statistically significant at p ≤ 0.05

The two groups had similar fasting glucose levels, while the obese PCOS women had a significantly higher fasting insulin level (p= 0.03) and higher insulin resistance (p=0.01) than the non-obese ones.

The correlation between BMI and the studied biochemical variables was non-significant (Table 3) with the exception of a significant positive correlation between BMI and HOMA- IR as shown in Figure 2, whereas, the BMI shows a negative significant correlation with LH.

Table 3: **Correlation between BMI and reproductive-related hormones and IR in PCOS women.**

	FSH	LH	Testosterone	DHEA	AMH	HOMA-IR
BMI	0.7	0.04*	0.58	0.79	0.48	0.01*

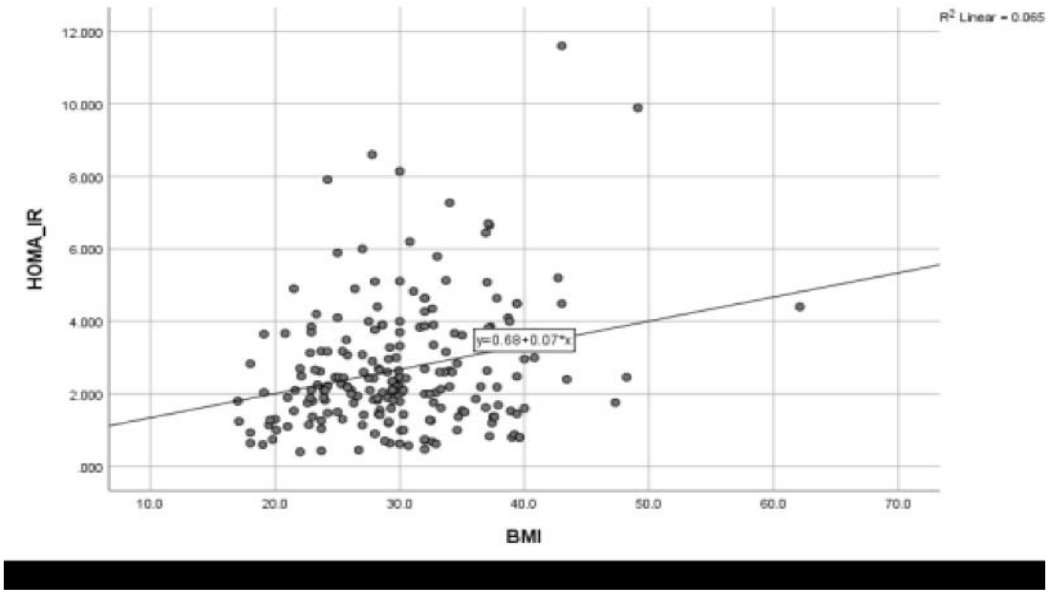

Figure 2. **Relationship between BMI and HOMA-IR**

Discussion

PCOS is a common heterogeneous disorder affecting women of reproductive age with a wide range of clinical, endocrinological, and metabolic manifestations (28). For the diagnosis of PCOS, two out of three following manifestations should be found; irregular or anovulation, clinical and/or biochemical hyperandrogenism, and ultrasound polycystic appearance of the ovaries (2). Ultrasound evidence of PCO in the current study was found in 97% and this result was higher than what (93.71%) was reported before (29) whereas, another study found a much lower incidence of polycystic appearance of the ovaries in PCOS women (60.93%) (30). So, the diagnosis of PCOS does not always require the presence of a PCO picture on ultrasound scan.

Both obesity and PCOS worsen each other in a vicus cycle (31). Obesity is a risk factor for PCOS and greatly modifies the clinical course and increases the co-morbidities of PCOS (19). It was mentioned in the literature that 30-70% of PCOS women were obese, however, this disorder also affects the normal weight women but less frequently (32, 33). The prevalence of obesity in the current study was 46.2% and this prevalence lies in the range recorded before in the literature. However, the prevalence of obesity in PCOS women was higher (75.61%) in a previous study (34) and this could be explained by the used cutoff values of BMI in (34); as BMI>23 kg/m2 was considered obese.

This study was conducted to compare the clinical, hormonal, and metabolic manifestations of obese women with PCOS to those of non-obese PCOS women. No significant difference was recorded in the clinical manifestations of PCOS between obese and non-obese PCOS women in the present study and this was similar to (4). In contrast, Sachdeva et al (34) used the Ferriman-Gallwey score and reported that the symptoms of clinical hyperandrogenism and the prevalence of menstrual irregularity were significantly higher in obese PCOS as compared to non-obese PCOS women.

There was no significant difference between the two studied groups in the circulating levels of the reproductive-related hormones; FSH, E2, prolactin, and TSH. Surprisingly, LH level was significantly higher in the non-obese PCOS women. Previous studies (34, 35) reported that the difference in FSH and LH between the obese and non-obese PCOS did not reach a significant level. The overall prevalence of hyperprolactinemia in this study was 36.7%, the same result reported before (36).

There was no significant difference in the biochemical hyperandrogenism (total testosterone and DHEA) between the two study groups and the same result was reported before (34, 35). On the contrary, (37) reported a higher level of androgens in obese PCOS than the none obese ones. The overall prevalence of insulin resistance (IR) in our PCOS participants, HOMA-IR ≥ 2.6 was 40%, this was lower than what was reported before (34, 35). The higher prevalence of IR in these two studies could be explained by the lower BMI cutoff value (BMI >23 and >25 respectively were used as BMI cutoff values) whereas, the cutoff BMI value in the current study was ≥ 30. The prevalence of IR among obese PCOS women was statistically higher than the non-obese PCOS women in the present study and also in Sachdeva et al study (34).

Similar to Sachdeva et al (34), the current study reported that the obese PCOS women had a significantly higher level of fasting insulin and insulin resistance than the non-obese whereas the fasting glucose level was similar in both studies. In accordance with our result, the difference in HOMA-IR levels between the various PCOS women phenotypes was not significantly different (35).

This study established a direct significant correlation between BMI and HOMA-IR, and (4, 29, 35) also reported the same correlation. This suggested the need for early screening, lifestyle modification, and weight reduction in PCOS women. The current study found a significant negative correlation between BMI and LH.

Conclusion

Obese PCOS women have higher LH, insulin levels, and higher insulin resistance than non-obese PCOS women and this adversely affects the reproductive and metabolic status. Therefore, targeting obesity in PCOS women will help to minimize these adverse outcomes.

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