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Original Research Article

Obese and non-obese polycystic ovarian syndrome: comparison of clinical, metabolic, hormonal parameter and their differential response to oral ovulation induction drugs

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ABSTRACT

Background: Polycystic ovarian syndrome (PCOS) is a complex disorder characterized by hyperandrogenism, menstrual irregularities, and polycystic ovaries. The present study compared obese and non-obese PCOS patients in terms of clinical, metabolic, and hormonal parameters, as well as their response to oral ovulation induction drugs.
Methods: This study was conducted in the Department of Obstetrics and Gynaecology, AIMSR Bathinda, over one year after approval from the Institutional Ethics Committee. Women with PCOS presenting with infertility were enrolled. Patients were categorized into obese and non-obese groups, and their clinical, metabolic, and hormonal profiles were compared. Data were recorded in Microsoft Excel and analysed using SPSS software.
Results: The mean age was 25.3 years in the obese group and 26.1 years in the non-obese group. Mean fasting insulin levels were higher in obese patients (25.1 µIU/ml) compared to non-obese (15.9 µIU/ml). Similarly, insulin resistance and HOMA-IR values were significantly greater in the obese group. Mean testosterone levels were slightly higher in obese patients (58.3 ng/dl vs. 55.4 ng/dl). Conception rates following oral ovulation induction were significantly lower in obese women (30.6%) than in non-obese women (76.3%).
Conclusions: Obese women with PCOS are at higher risk of metabolic derangements and exhibit poorer responsiveness to ovulation induction therapy. Early identification and targeted weight management strategies may not only prevent adverse outcomes but also improve fertility outcomes in this population.

Keywords: Infertility, Polycystic ovarian syndrome, Obesity, Ovulation induction

INTRODUCTION

Polycystic ovarian syndrome (PCOS) is one of the most common endocrine disorders affecting women of reproductive age, yet it remains one of the most challenging to fully understand. Characterized by excess androgen levels, irregular menstrual cycles, and the presence of multiple small ovarian cysts, PCOS is not a single disease but a spectrum of interconnected reproductive and metabolic disturbances.[1,2] Symptoms often appear during adolescence and can present as acne, anovulation, or irregular periods, creating not only clinical challenges but also a significant impact on quality of life.[3]

Obesity adds another layer of complexity to this disorder. Women with higher body mass index (BMI) are more likely to experience menstrual disturbances and infertility related to anovulation compared to their lean counterparts. Even a BMI above 24 kg/m² has been associated with a measurable rise in reproductive dysfunction, and this risk escalates with increasing weight. Encouragingly, lifestyle

modifications and weight reduction have been shown to restore ovulatory cycles in many women.[4]

Pharmacological strategies remain central to treatment, with agents such as clomiphene citrate and tamoxifen widely used to induce ovulation. These anti-estrogenic drugs act by lifting the hypothalamic-pituitary axis from estrogen's inhibitory feedback, thereby promoting follicular development and ovulation.[5-7] However, the response to such agents may not be uniform, particularly when obesity is a contributing factor.

Against this background, the present study aims to compare the clinical, metabolic, and hormonal parameters of obese and non-obese women with PCOS, while also evaluating their differential response to oral ovulation induction therapy.

METHODS

The study was carried out in the department of obstetrics and gynecology, AIMSR Bathinda, after getting approved from the research committee, AIMSR, and the Ethics Committee for Biomedical and Health Research, Adesh University, for a period of 1 year from September 2022 to September 2023. This was a cohort study involving 320 women with PCOS, including 160 obese PCOS and 160 non-obese PCOS women presenting with infertility attending OPD at AIMSR, Bathinda (a tertiary health care center), who constituted the study population. Comparison of clinical, metabolic, and hormonal parameters among obese and non-obese groups: menstrual irregularities, metabolic syndrome, and blood sugar abnormalities may be high in the obese PCOS group. All the results were recorded in a Microsoft Excel sheet and were subjected to statistical analysis using SPSS software.

These PCOS women were divided into two groups: obese and non-obese PCOS. Women with BMI ≥23 kg/m² were included in the obese PCOS group, and the other group included those women with BMI <23 kg/m² (normal and underweight women) designated as the non-obese PCOS group. All women with a history of oligo/amenorrhea and clinical signs of hyperandrogenism, like acne or hirsutism, were enrolled in the study from the outpatient clinic. Patients fulfilling at least two out of three Rotterdam Criteria were recruited, and records were maintained on predesigned forms after obtaining written informed consent.

Various clinical, metabolic, and hormonal parameters were compared between the two groups. Clinical parameters included signs of androgen excess, such as excessive hair growth, acne, and alopecia. Excessive hair growth was evaluated by the modified Ferriman and Gallwey score.

The patients enrolled in the study were called on day 2 of their cycle for the investigations [follicle-stimulating hormone (FSH), luteinizing hormone (LH), testosterone, androstenedione, fasting insulin, triglycerides, high-density lipoprotein (HDL), low-density lipoprotein (LDL), and cholesterol levels]. The results were compared in the obese and non-obese PCOS groups.

The Homeostasis Model Assessment of Insulin Resistance (HOMA-IR), a marker of IR, was used in this study. Patients with HOMA-IR >2 were defined as having IR. IR was compared between obese and non-obese PCOS groups.

All these patients were treated with an oral ovulation induction drug starting on Day 2-5 of their cycle for 5 days. In case of failure of ovulation, the dose will be increased in subsequent cycles. Response to oral ovulation induction drug was assessed by ovulation. A transvaginal scan (TVS) was done, and patients were called when periods were missed and a urine pregnancy test was done.

RESULTS

The mean age of participants was comparable between the obese group (25.3 years) and the non-obese group (26.1 years). As expected, mean BMI was significantly higher in the obese group (27.3 kg/m²) compared to the non-obese group (22.9 kg/m²).

Obese patients demonstrated higher mean systolic (122.3 mmHg) and diastolic (82.9 mmHg) blood pressure values than their non-obese counterparts (82.9 mmHg and 81.15 mmHg, respectively). The Ferriman-Gallwey score, reflecting clinical hyperandrogenism, was significantly higher in the obese group (8.24) than in the non-obese group (5.13; p<0.001) (Table 1).

Table 1: Ferriman-Gallwey score.

Ferriman-Gallwey score	Obese group	Non-obese group
Mean	8.24	5.13
SD	1.86	1.91
P value	0.000 (Significant)	

Acanthosis nigricans was also more prevalent among obese patients (56.88%) compared to non-obese patients (31.88%; p<0.05). Similarly, a positive family history of diabetes was reported more frequently in the obese group (57.5%) than in the non-obese group (31.88%; p<0.05).

With respect to metabolic parameters, obese women exhibited significantly higher mean total cholesterol (154.83 mg/dl vs. 121.38 mg/dl, p<0.001) and triglyceride levels (189.48 mg/dl vs. 170.09 mg/dl, p<0.001), whereas LDL and HDL values were comparable between the two groups (Table 2).

Hormonal evaluation revealed that obese women had markedly elevated fasting insulin levels (25.1 µIU/ml vs. 15.9 µIU/ml, p<0.001) and higher HOMA-IR scores (3.05

vs. 2.16, p<0.001). Insulin resistance was observed in 79.38% of obese patients, compared to 51.88% of non-obese patients (p<0.001). Testosterone, FSH, and estradiol levels did not differ significantly between groups (Table 3).

Table 2: Lipid profile.

Lipid profile	Obese group		Non-obese group		P value
	Mean	SD	Mean	SD	
Total cholesterol (mg/dl)	154.83	49.33	121.38	49.48	0.001 (Significant)
LDL (mg/dl)	38.81	6.45	39.61	7.32	0.520
HDL (mg/dl)	45.43	9.86	43.76	9.81	0.64
Triglycerides (mg/dl)	189.48	32.01	170.09	31.64	0.000 (Significant)

Table 3: Hormonal profile.

Hormonal profile	Obese group		Non-obese group		P value
	Mean	SD	Mean	SD	
Fasting insulin (µIU/ml)	25.1	2.71	15.9	2.6	0.001 (Significant)
Testosterone (ng/dl)	58.3	9.04	55.4	9.17	0.62
HOMA-IR	3.05	2.11	2.16	1.81	0.000 (Significant)
Insulin resistance present, N (%)	127	79.38	83	51.88	0.000 (Significant)
Follicular stimulating hormone (IU/l)	5.35	0.73	5.03	1.03	0.79
Estradiol (pg/ml)	165.4	20.91	162.1	21.19	0.45

Table 4: Response to oral ovulation drug.

Response to oral ovulation drug	Obese group		Non-obese group		P value
	Number	Percentage	Number	Percentage	
Not-conceived	111	69.38	38	23.75	0.001 (Significant)
Conceived	49	30.62	122	76.25	
Total	160	100	160	100	

The response to ovulation induction therapy differed substantially between groups. Conception was achieved in 76.25% of non-obese patients, compared with only 30.62% of obese patients (p<0.001), highlighting the impact of obesity on treatment outcomes (Table 4).

DISCUSSION

In the present study, the mean age of patients in both the obese and non-obese PCOS groups was comparable (25.3 vs. 26.1 years), which is consistent with findings from Sachdeva et al, who also reported similar mean ages across groups.[8]

Obese women demonstrated a higher prevalence of acanthosis nigricans (56.88%) compared to non-obese women (31.88%), a finding supported by previous studies. Akshaya et al observed significantly greater rates of acanthosis in obese women (14.3% vs. 9.1%), while Makhija et al also reported a higher prevalence among obese PCOS patients (28.7% vs. 3.33%).[9,10] These dermatological manifestations reflect underlying hyperinsulinemia and hyperandrogenism, both of which are characteristic of PCOS.[11]

Our study further highlights the metabolic burden associated with obesity in PCOS. Obese women had significantly higher fasting insulin levels, HOMA-IR, and prevalence of insulin resistance compared to their non-obese counterparts. These findings align with Sachdeva et al, who reported significantly altered insulin resistance indices in obese women with PCOS.[9] The interplay of obesity, hyperinsulinemia, and decreased sex hormone-binding globulin (SHBG) contributes to elevated free androgen levels, thereby exacerbating hyperandrogenic features. This mechanism also explains the higher Ferriman–Gallwey scores and androgenic trends seen in our obese group. Similar associations between BMI and hyperandrogenism have been reported by Kim et al.[12]

Importantly, our results demonstrate that obesity negatively influences fertility outcomes in PCOS. Only 30.62% of obese women conceived following ovulation induction compared with 76.25% of non-obese women, indicating a significantly reduced response to therapy. This is in agreement with Sachdeva et al, who also found higher resistance to oral ovulation induction drugs among obese patients (58.87% vs. 37.5%).[8] Obesity, therefore, not only worsens the clinical and metabolic phenotype of PCOS but also impairs treatment success.

Taken together, these findings emphasize that obesity amplifies the clinical severity, metabolic dysfunction, and therapeutic resistance in PCOS. Addressing weight reduction through lifestyle interventions should therefore be considered an essential component of management before or alongside pharmacological ovulation induction.

CONCLUSION

This study demonstrates that obesity significantly worsens the clinical, metabolic, and hormonal profile of women with PCOS. Obese women exhibited higher rates of acanthosis nigricans, insulin resistance, and dyslipidemia, along with higher Ferriman-Gallwey scores, compared to non-obese women. Most importantly, obese women had a markedly lower conception rate following oral ovulation induction therapy with clomiphene citrate, tamoxifen, and letrozole. These findings highlight obesity as a critical factor influencing both disease severity and therapeutic response. Incorporating weight reduction strategies alongside pharmacological therapy may therefore enhance reproductive outcomes in this population.

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Ethical approval: The study was approved by the Institutional Ethics Committee of Adesh Institute of Medical Sciences and Research, Bathinda

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