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ASSOCIATION BETWEEN OBESITY AND INFERTILITY: A CLINICAL AND HORMONAL PERSPECTIVE

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ABSTRACT

Background: Infertility in both men and women is increasingly linked to obesity, which has become a major global public health concern. Overweight causes insulin resistance, chronic inflammation, hormonal imbalance, and disruption of the hypothalamic-pituitary-gonadal axis, all of which have a negative impact on reproductive function. Improving reproductive outcomes requires an understanding of the clinical and hormonal mechanisms connecting obesity and infertility.

Objective: To ascertain whether obesity and infertility are related from a clinical and hormonal standpoint in adults of reproductive age who visit the Department of Endocrinology and Diabetes, Ward-6, Jinnah Postgraduate Medical Centre (JPMC), Karachi.

Methodology: From January 2025 to January 2026, a comparative cross-sectional study was carried out at the Department of Endocrinology and Diabetes, Ward-6, Jinnah Postgraduate Medical Centre (JPMC), Karachi, Pakistan. Through consecutive sampling, 240 infertile men and women between the ages of 20 and 45 were recruited and divided into two groups: obese (BMI ≥30 kg/m²) and non-obese (BMI 18.5–24.9 kg/m²). Anthropometric measurements, reproductive history, follicle-stimulating hormone (FSH), luteinizing hormone (LH), prolactin, estradiol, progesterone, testosterone, anti-Müllerian hormone (AMH), thyroid-stimulating hormone (TSH), fasting insulin, and fasting glucose were all evaluated. SPSS version 26.0 was used to analyse the data. Independent sample t-tests and Chi-square tests were used to compare groups, and multivariable logistic regression was used to find independent predictors of infertility. Statistical significance was defined as a p-value of less than 0.05.

Result: Of the 240 participants, 110 (45.8%) were not obese and 130 (54.2%) were. Menstrual irregularities (61.5% vs. 27.3%), polycystic ovary syndrome (38.5% vs. 15.5%), insulin resistance (69.2% vs. 24.5%), and metabolic syndrome (35.4% vs. 12.7%) were all significantly more common in obese participants ($p<0.001$). Serum levels of LH, total testosterone, prolactin, fasting insulin, and HOMA-IR were significantly higher in obese female participants than in non-obese females, while AMH levels were significantly lower ($p<0.05$). Male obesity was associated with significantly lower serum testosterone levels and worse semen quality, including lower sperm motility and concentration ($p<0.05$). Obesity (Adjusted OR=2.84, 95% CI: 1.67–4.82), insulin resistance (Adjusted OR=2.41, 95% CI: 1.39–4.18), and elevated testosterone in women (Adjusted OR=1.96, 95% CI: 1.12–3.42) were found to be independent predictors of infertility using multivariable logistic regression.

Conclusion: Through a variety of clinical and hormonal mechanisms that impact both male and female reproductive function, obesity is closely linked to infertility. To improve reproductive outcomes, infertility management should include the early detection of endocrine abnormalities and the application of weight reduction techniques.

KEYWORDS: Polycystic Ovary Syndrome, Endocrinology, Insulin Resistance, Obesity, Infertility, Hormonal Profile, Reproductive Health, JPMC Karachi.

INTRODUCTION

About 10% to 15% of couples who are of reproductive age worldwide struggle with infertility, which has serious medical, psychological, social, and financial repercussions. Infertility is defined by the World Health Organisation (WHO) as the inability to conceive after 12 months or more of consistent, unprotected sexual activity. Obesity is one of the most significant modifiable risk factors contributing to impaired reproductive function in both women and men, despite the fact that infertility has multiple aetiologies. Obesity is a significant area of reproductive and endocrine research because its prevalence has increased in tandem with rising rates of infertility.

Over one billion people worldwide suffer from obesity, and the prevalence of the condition is rising in both developed and developing nations, according to the World Obesity Federation. Rapid urbanisation, sedentary lifestyles, poor eating habits, and rising rates of metabolic disorders have made obesity a major public health concern in Pakistan. Because excessive adiposity negatively impacts reproductive physiology through intricate endocrine, metabolic, and inflammatory mechanisms, the coexistence of obesity and infertility has become a major concern.

Excessive adipose tissue accumulation that serves as an active endocrine organ rather than just a place to store energy is what defines obesity. Leptin, adiponectin, resistin, tumour necrosis factor-alpha (TNF-α), interleukin-6 (IL-6), and other biologically active substances that control metabolism, inflammation, insulin sensitivity, and reproductive function are all secreted by adipose tissue. Dysregulation of these adipokines in obesity causes endocrine dysfunction and chronic low-grade inflammation, both of which have a detrimental effect on fertility.

Disruption of the hypothalamic-pituitary-gonadal (HPG) axis is one of the main mechanisms connecting obesity to infertility. The pulsatile secretion of gonadotropin-releasing hormone (GnRH) is altered by excess adiposity, which results in aberrant secretion of luteinizing hormone (LH) and follicle-stimulating hormone (FSH). Follicular development, ovulation, steroid hormone production, and gametogenesis are all hampered by these hormonal disruptions. As a result, compared to people of normal body weight, obese people have a lower capacity for reproduction.

Ovarian physiology in women is significantly impacted by obesity. Insulin resistance and compensatory hyperinsulinemia, which promote ovarian androgen production while inhibiting hepatic synthesis of sex hormone-binding globulin (SHBG), are linked to increased adiposity. Particularly in women with polycystic ovary syndrome (PCOS), elevated circulating free testosterone encourages hyperandrogenism, follicular arrest, and chronic anovulation. About 70–80% of cases of anovulatory infertility are caused by PCOS, one of the most prevalent endocrine disorders affecting women of reproductive age. PCOS's metabolic and reproductive symptoms are made worse by obesity, which results in irregular menstruation, decreased fertility, and unfavourable pregnancy outcomes. Additionally, ovarian reserve and oocyte quality are adversely affected by obesity. Research has shown that obese women often have lower concentrations of anti-Müllerian hormone (AMH), reduced implantation rates after assisted reproductive technologies (ART), altered follicular fluid composition, and impaired mitochondrial function within oocytes. Together, these variables lead to decreased rates of conception, a higher risk of miscarriage, and worse outcomes after in vitro fertilisation (IVF).

Numerous maternal and foetal complications, such as gestational diabetes mellitus, hypertensive disorders of pregnancy, preeclampsia, caesarean delivery, preterm birth, foetal macrosomia, congenital anomalies, and stillbirth, are also linked to pregnancy among obese women. As a result, obesity has a negative impact on neonatal health and pregnancy maintenance in addition to influencing conception.

Another effect of obesity that is becoming more widely acknowledged is male infertility. Increased aromatization of testosterone into estradiol is facilitated by excess adipose tissue, which suppresses pituitary and hypothalamic gonadotropin secretion. Hypogonadotropic hypogonadism results from decreased Leydig cell testosterone production due to decreased LH stimulation. Men who are obese frequently have lower levels of total and free testosterone, higher levels of oestrogen, impaired spermatogenesis, erectile dysfunction, decreased libido, and poorer semen quality, which is characterised by decreased sperm concentration, motility, morphology, and increased sperm DNA fragmentation.

Reproductive function is further compromised by metabolic abnormalities linked to obesity. Gonadal function is compromised in both sexes by insulin resistance, dyslipidemia, oxidative stress, chronic systemic inflammation, endothelial dysfunction, and mitochondrial damage. Increased oxidative stress in reproductive tissues due to elevated inflammatory cytokines causes DNA damage, germ cell apoptosis, impaired embryo development, and decreased implantation potential.

Numerous clinical studies have shown that reproductive outcomes are greatly improved by even modest weight loss. Restoring ovulatory cycles, improving insulin sensitivity, normalising reproductive hormone profiles, improving semen quality, and increasing spontaneous pregnancy rates are all possible with lifestyle changes that include calorie restriction, increased physical activity, and behavioural interventions. In certain obese infertile patients, pharmacological treatments like glucagon-like peptide-1 (GLP-1) receptor agonists and bariatric surgery have also demonstrated encouraging results. As a result, weight optimisation is emphasised in current reproductive medicine guidelines as a crucial part of managing infertility.

Data assessing the clinical and hormonal relationship between obesity and infertility are still scarce in Pakistan, despite growing evidence from around the world. Rather than offering a thorough evaluation of endocrine and reproductive dysfunction in obese infertile individuals, the majority of local studies have concentrated mainly on PCOS or isolated hormonal abnormalities. Developing effective preventive and therapeutic strategies requires an understanding of the impact of obesity on reproductive health, given the rapidly rising prevalence of obesity in Pakistan.

In order to assess the relationship between obesity and infertility from both clinical and hormonal viewpoints, the current study was carried out at the Department of Endocrinology and Diabetes, Ward-6, Jinnah Postgraduate Medical Centre (JPMC), Karachi. The study sought to determine endocrine factors independently linked to infertility and to

compare reproductive hormone profiles, metabolic parameters, and clinical manifestations between obese and non-obese infertile adults. By highlighting the significance of early endocrine evaluation, weight management, and multidisciplinary care in improving fertility outcomes among obese patients, the findings are anticipated to contribute to evidence-based clinical practice.

METHODOLOGY

From January 2025 to January 2026, a comparative cross-sectional study was carried out in the Department of Endocrinology and Diabetes, Ward-6, Jinnah Postgraduate Medical Centre (JPMC), Karachi, Pakistan. The department is a suitable setting for assessing infertility and hormonal abnormalities associated with obesity because it offers specialised endocrine and reproductive endocrine services.

Non-probability consecutive sampling was used to select 240 individuals of reproductive age (20–45 years) who came to the infertility clinic. According to the World Health Organization's body mass index (BMI) classification, participants were split into two groups: non-obese (BMI 18.5–24.9 kg/m²) and obese (BMI ≥30 kg/m²). Patients with primary or secondary infertility lasting at least a year, both male and female, were included. Those with known chromosomal disorders, cancer, pregnancy, congenital reproductive tract abnormalities, or those undergoing hormonal therapy within the last three months were not included.

A structured proforma was used to record demographic information, medical history, lifestyle factors, reproductive history, menstrual characteristics (in women), and anthropometric measurements, such as height, weight, waist circumference, and BMI, following approval from the Institutional Review Board and written informed consent from each participant. Standardised chemiluminescent immunoassays were used to measure fasting blood glucose, fasting insulin, thyroid-stimulating hormone (TSH), follicle-stimulating hormone (FSH), luteinizing hormone (LH), prolactin, estradiol, progesterone, anti-Müllerian hormone (AMH), and total testosterone in venous blood samples obtained after an overnight fast. To evaluate insulin resistance, the Homeostatic Model Assessment for Insulin Resistance (HOMA-IR) was computed. The World Health Organization's laboratory guidelines were followed when analysing the semen of male participants.

SPSS version 26.0 was used for data entry and analysis. Whereas categorical variables were expressed as percentages and frequencies, continuous variables were shown as mean ± standard deviation. Clinical and hormonal variables were compared between the obese and non-obese groups using independent sample t-tests and Chi-square tests. To find independent predictors of infertility, multivariable logistic regression analysis was used. Statistical significance was defined as a p-value of less than 0.05.

RESULTS

A total of 240 infertile participants were included in the study. Of these, 130 (54.2%) were classified as obese (BMI ≥30 kg/m²), while 110 (45.8%) had a normal BMI (18.5–24.9 kg/m²). The mean age of the study population was 31.8 ± 5.6 years, with no statistically significant difference between the two groups (p=0.284). Females constituted 60.0% (n=144) of the participants, whereas males accounted for 40.0% (n=96). The mean BMI was significantly higher in the obese group (33.9 ± 3.2 kg/m²) compared to the non-obese group (22.8 ± 1.6 kg/m², p<0.001).

Table 1. Baseline Demographic and Clinical Characteristics of the Study Participants

Variable	Obese (n=130)	Non-obese (n=110)	p-value
Age (years), Mean ± SD	32.2 ± 5.7	31.4 ± 5.5	0.284
Female, n (%)	82 (63.1)	62 (56.4)	0.293
Male, n (%)	48 (36.9)	48 (43.6)	0.293
BMI (kg/m²), Mean ± SD	33.9 ± 3.2	22.8 ± 1.6	<0.001
Duration of infertility (years)	4.1 ± 1.8	3.2 ± 1.5	0.001
Waist circumference (cm)	104.8 ± 9.3	83.7 ± 7.4	<0.001

Obese participants had a significantly longer duration of infertility and greater waist circumference than non-obese participants.

Table 2. Clinical Characteristics According to Obesity Status

Clinical Variable	Obese n (%)	Non-obese n (%)	p-value
Menstrual irregularities*	50 (61.0)	17 (27.4)	<0.001
Polycystic ovary syndrome (PCOS)*	32 (39.0)	10 (16.1)	0.002
Insulin resistance	90 (69.2)	27 (24.5)	<0.001

Metabolic syndrome	46 (35.4)	14 (12.7)	<0.001
Hypertension	31 (23.8)	10 (9.1)	0.003
Erectile dysfunction†	19 (39.6)	8 (16.7)	0.013

Obese women had significantly higher frequencies of menstrual irregularities and PCOS compared with non-obese women. Obese participants also demonstrated significantly higher rates of insulin resistance, metabolic syndrome, hypertension, and erectile dysfunction (all $p<0.05$).

Table 3. Female Hormonal Profile

Hormonal Parameter	Obese Females (n=82) Mean ± SD	Non-obese Females (n=62) Mean ± SD	p-value
FSH (mIU/mL)	6.8 ± 1.9	7.2 ± 1.8	0.181
LH (mIU/mL)	11.5 ± 3.4	7.9 ± 2.5	<0.001
LH/FSH ratio	1.72 ± 0.58	1.09 ± 0.36	<0.001
Estradiol (pg/mL)	63.8 ± 19.4	69.1 ± 18.5	0.087
Progesterone (ng/mL)	7.2 ± 2.6	10.1 ± 2.9	<0.001
Total Testosterone (ng/dL)	59.6 ± 18.2	36.5 ± 10.8	<0.001
Prolactin (ng/mL)	23.9 ± 8.4	17.6 ± 6.1	<0.001
AMH (ng/mL)	2.4 ± 1.1	3.5 ± 1.3	<0.001
TSH (µIU/mL)	3.2 ± 1.2	2.5 ± 0.9	0.001

Obese female participants had significantly higher LH, LH/FSH ratio, testosterone, prolactin, and TSH levels, while progesterone and AMH levels were significantly lower than those in non-obese females.

Table 4. Male Hormonal Profile and Semen Parameters

Variable	Obese Males (n=48)	Non-obese Males (n=48)	p-value
Testosterone (ng/dL)	305 ± 72	452 ± 81	<0.001
FSH (mIU/mL)	5.9 ± 1.5	6.4 ± 1.6	0.108
LH (mIU/mL)	6.4 ± 1.8	5.9 ± 1.6	0.126
Sperm concentration (million/mL)	27.3 ± 10.8	43.9 ± 12.5	<0.001
Progressive motility (%)	35.6 ± 9.8	51.8 ± 11.2	<0.001
Normal morphology (%)	3.6 ± 1.5	5.4 ± 1.8	<0.001

Obese men exhibited significantly lower serum testosterone concentrations and significantly poorer semen quality than non-obese men.

Table 5. Metabolic Parameters

Parameter	Obese (n=130)	Non-obese (n=110)	p-value
Fasting glucose (mg/dL)	109.8 ± 18.4	91.5 ± 12.7	<0.001
Fasting insulin (µIU/mL)	22.9 ± 8.5	11.3 ± 4.6	<0.001
HOMA-IR	5.8 ± 2.1	2.4 ± 1.1	<0.001
Total cholesterol (mg/dL)	214.5 ± 34.8	181.7 ± 29.2	<0.001
Triglycerides (mg/dL)	192.6 ± 41.9	139.2 ± 33.7	<0.001

Obese participants demonstrated significantly higher fasting glucose, fasting insulin, HOMA-IR, cholesterol, and triglyceride levels than non-obese participants, indicating marked metabolic dysfunction.

Table 6. Multivariable Logistic Regression Analysis for Factors Associated with Infertility Severity

Variable	Adjusted Odds Ratio (AOR)	95% Confidence Interval	p-value
Obesity (BMI ≥30 kg/m²)	2.84	1.67–4.82	<0.001
Insulin resistance	2.41	1.39–4.18	0.002

Elevated female testosterone	1.96	1.12–3.42	0.018
Reduced AMH	2.27	1.30–3.95	0.004
Low male testosterone	2.18	1.16–4.08	0.015

Multivariable logistic regression identified obesity, insulin resistance, elevated testosterone in women, reduced AMH levels, and low testosterone in men as independent predictors of infertility severity.

DISCUSSION

The current study assessed the relationship between obesity and infertility in reproductive-age adults who visited the Department of Endocrinology and Diabetes, Ward-6, Jinnah Postgraduate Medical Centre (JPMC), Karachi, from a clinical and hormonal standpoint. The results showed that obesity was substantially linked to longer periods of infertility, metabolic problems, abnormal reproductive hormones, irregular menstruation, polycystic ovarian syndrome (PCOS), poor semen quality, and changed gonadal hormone levels. These findings demonstrate the significant impact of obesity as a modifiable endocrine factor on both male and female reproductive health.

There is a high prevalence of obesity among people seeking infertility treatment, as evidenced by the fact that 54.2% of the infertile participants in the current study were obese. Compared to non-obese individuals, obese participants had significantly higher waist circumferences, BMIs, and longer periods of infertility. Similar results have been documented in earlier research demonstrating that obesity has a detrimental impact on fertility by changing the regulation of reproductive hormones and decreasing the likelihood of natural conception. Adipokines and inflammatory mediators are produced by excess body fat, which functions as an endocrine organ and disrupts normal reproductive physiology.

The substantial correlation between obesity and insulin resistance was one of the study's key findings. HOMA-IR values and fasting insulin levels were significantly higher in obese individuals than in non-obese individuals. One of the main mechanisms connecting obesity and infertility is thought to be insulin resistance. Elevated levels of circulating free testosterone are caused by hyperinsulinemia, which increases the production of androgens by ovarian theca cells and decreases the production of sex hormone-binding globulin (SHBG) in the liver. Particularly in women with PCOS, these hormonal alterations lead to follicular dysfunction, anovulation, and decreased fertility.

In comparison to non-obese women, the current study found that obese women had a noticeably higher prevalence of PCOS. It is well known that obesity exacerbates PCOS's metabolic and reproductive symptoms. Insulin resistance, hyperandrogenism, and aberrant follicular development are all facilitated by increased visceral adiposity. Obesity-induced disruption of the hypothalamic-pituitary-ovarian axis is further supported by the higher LH levels and increased LH/FSH ratio found in obese women in this study. Similar findings from earlier studies have shown that, in comparison to women of normal weight, obese women with PCOS experience more severe menstrual irregularities, fewer ovulatory cycles, and worse pregnancy outcomes.

Significant changes in female reproductive hormones were also shown by the study. Compared to non-obese females, obese females had lower levels of progesterone and AMH and higher levels of LH, testosterone, prolactin, and TSH. While decreased AMH indicates decreased ovarian reserve or changed follicular dynamics, decreased progesterone indicates impaired ovulation and luteal phase dysfunction. Oxidative stress and inflammation associated with obesity may have a detrimental effect on ovarian follicles and oocyte competence, which may lower the potential for fertility and the success rates of assisted reproductive methods.

Significant drops in serum testosterone levels and a decline in semen parameters were linked to obesity in male participants. Compared to non-obese males, obese males displayed aberrant morphology, decreased progressive motility, and lower sperm concentration. These results are in line with earlier research showing that increased aromatase activity in adipose tissue, which changes testosterone into estradiol, is a contributing factor to male infertility. High levels of oestrogen hinder spermatogenesis, reduce Leydig cell testosterone production, and suppress gonadotropin secretion. Furthermore, oxidative stress brought on by obesity may worsen sperm function and increase sperm DNA fragmentation.

Additionally, the current study found a strong correlation between obesity and elements of the metabolic syndrome, such as dyslipidemia, hypertension, and impaired glucose metabolism. By encouraging endothelial dysfunction, inflammation, and hormonal imbalance, these metabolic abnormalities may independently contribute to infertility. Obesity-related chronic inflammation raises circulating cytokines like interleukin-6 (IL-6) and tumour necrosis factor-alpha (TNF-α), which may affect testicular and ovarian function.

Obesity, insulin resistance, high testosterone levels in women, low testosterone levels in men, and decreased AMH levels were found to be independent predictors of the severity of infertility using multivariable logistic regression analysis. These results highlight the fact that obesity directly contributes to reproductive dysfunction through a number of endocrine pathways, rather than just being a related condition.

The study's findings have significant clinical ramifications. In addition to reproductive interventions, comprehensive metabolic and endocrine assessments should be part of infertility management. Reducing weight through diet,

exercise, and lifestyle changes has been demonstrated to improve insulin sensitivity, normalise reproductive hormone profiles, restore ovulation, and improve fertility outcomes. For those who are extremely obese, pharmacological weight control or bariatric surgery may offer extra advantages in specific circumstances.

This study sheds important light on the connection between obesity and infertility in a clinical context in Pakistan. It is important to recognise some limitations, though. The cross-sectional design makes it more difficult to determine whether obesity and infertility are causally related. Furthermore, variables like food habits, degree of physical activity, genetic predisposition, and psychological stress were not thoroughly examined. It is advised that future longitudinal studies with larger sample sizes investigate the long-term reproductive effects of obesity and the effects of weight loss interventions on fertility outcomes.

Overall, the results show that complex hormonal, metabolic, and reproductive mechanisms strongly link obesity to infertility. Implementing multidisciplinary weight management techniques and early detection of obesity-related endocrine abnormalities may greatly enhance fertility outcomes and lessen the burden of infertility.

CONCLUSION

Through a variety of clinical, hormonal, and metabolic mechanisms, the current study showed a strong correlation between obesity and infertility. Menstrual irregularities, polycystic ovary syndrome, insulin resistance, altered reproductive hormone profiles, impaired ovarian function, lower testosterone levels in males, and poor semen quality were all more common in obese people.

The hypothalamic-pituitary-gonadal axis, hyperinsulinemia, inflammation, and aberrant sex hormone metabolism are among the endocrine disorders caused by obesity that significantly reduce fertility in both men and women. The results highlight the significance of evaluating metabolic and hormonal status in infertile patients and show that elevated BMI is an independent risk factor for the severity of infertility.

Reproductive outcomes may be improved by early detection of obesity-related reproductive abnormalities and the application of efficient weight management techniques, such as dietary counselling, lifestyle modification, and suitable endocrine interventions. Infertility management protocols should include a multidisciplinary approach involving endocrinologists, gynaecologists, and fertility specialists, especially for obese patients.

To assess the long-term impacts of weight loss interventions on hormonal normalisation, fertility restoration, and pregnancy outcomes, more long-term research is needed.

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