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Circulating IL-6 and CRP Levels as Inflammatory Biomarkers in Obese Men with Hormonal Infertility

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Abstract

Background: Hormonal infertility is closely linked to obesity in males and is most likely to result due to chronic low-grade inflammation mediated by adipose tissue-derived cytokines. The presence of circulating IL-6 and CRP as the main inflammatory biomarkers that disorganize the hypothalamic-pituitary-gonadal axis and cause dysfunction of testosterone production and disturbed reproductive activity. **Aims of the study;** This study seeks to examine the levels of the inflammatory biomarkers of interleukin-6 (IL-6) and C-reactive protein (CRP) in the obese men with hormonal infertility and analyze their relations with the hormonal profiles and their body mass index (BMI) relative to the healthy control. **Methodology:** The case-control study was carried out between February and September 2025 in two hospitals in Nasiriyah, Iraq where 100 obese men with hormonal infertility and 50 healthy controls participated in the study. The eligible patients were aged between 25 and 45 years and had a BMI of 30kg/m 2 and the controls were normal in weight and had no infertility. Blood samples had been gathered, processed and stored to be analyzed. Hormonal evaluation was carried out by evaluating testosterone, LH, FSH and prolactin by ELISA, IL-6 and CRP was done by ELISA and immunoturbidimetric assays. **Result:** Of the 100 obese men in the study, the hormonal infertility of the men was compared to the control of 50 healthy men. There were notable differences in BMI of patients (32.4 vs. 24.1, $p<0.001$), but no difference was found in age, smoking, education and alcohol consumption. The levels of IL-6 and CRP were significantly higher in patients (8.6 pg/mL and 6.8 mg/L) compared to controls (2.1 pg/mL and 1.2mg/l, $p<0.001$). The analysis of hormones showed that patients had low testosterone and high LH, FSH and prolactin ($p<0.01$). Correlations revealed that there was inflammation that was attributed to obesity and hormonal imbalance, and that the categories of obesity that differed significantly across groups. **Conclusions**: This paper concludes that the obese men with hormonal infertility have high levels of IL-6 and CRP, low testosterone, and disturbed gonadotropins. These results indicate that obesity-induced chronic inflammation interferes with the hypothalamic–pituitary-gonadal axis, which is the reason why obesity leads to infertility by disrupting hormonal and immunological regulation.

Keywords: Obesity; Hormonal infertility; Interleukin-6 (IL-6); C-reactive protein (CRP); Inflammation; Hypothalamic–pituitary–gonadal axis

Introduction:

The issue of infertility is a worldwide health issue with an approximation of 10 to 15 percent of couples having infertility with a male factor prevailing in almost half the cases (1). Hormonal dysregulation is one of the key and most frequently used etiologies of male infertility that is commonly characterized by a lack of balance in the respective levels of gonadotropins and androgens, including luteinizing hormone (LH), follicle-stimulating hormone (FSH), and testosterone (21). Obesity is a recent parameter that has been found to have significant influence in the male reproductive health because excessive adipose tissue impairs hormonal balance, spermatogenic arrests as well as reduced potential reproductive fitness (16). Obesity has become widely prevalent throughout the world, this happens in men who are presumably hormonally infertile (23).

Obesity is associated with several male infertility mechanisms, such as hormonal imbalance, higher scrotal temperature, and sperm parameters (4). Abstract—Excess adipose tissue has a role of an active endocrine organ, secreting a variety of adipokines and pro-inflammatory cytokines that disrupt the hypothalamic-pituitary-gonadal (HPG) axis and inhibit Leydig cell function, resulting in decreased testosterone synthesis and abnormal gonadotropin regulation (22). IL-6 and C-reactive protein (CRP) are two important inflammatory mediators involved in low-grade systemic inflammation, which is typical for obese individuals. (15) High levels of IL-6 and CRP are linked with not only metabolic derangement but also with adverse effects on male reproduction such as decreased sperm parameters and increased testosterone, estradiol and luteinizing hormone (7).

IL-6 is a pleiotropic cytokine that takes part in immunoregulation, inflammation, and metabolism. This molecule is secreted by adipocytes, macrophages, and other cells in response to tissue stress or inflammatory stimuli (9). Higher serum concentrations of IL-6 in obesity promote a state of low-grade chronic inflammation, which has the potential to alter endocrine function. C-reactive protein (CRP), an acute-phase protein mainly produced by the liver in response to stimulation by IL-6, is a systemic marker of inflammation. High serum levels of C-reactive protein (CRP) have been associated with obesity, insulin resistance and cardiovascular disease in a manner consistent with common pathways of metabolic derangement and inflammation (13). Notably, IL-6 and CRP have been reported to impair reproductive hormone synthesis, so inflammatory processes may increase hormonal infertility in obese men (18).

There are multiple studies that have shown the effects of systemic inflammation on male reproductive hormones. As an example, persistent inflammation may inhibit the pulsatile secretion of hypothalamic gonadotropin-releasing hormone (GnRH), leading to decreased LH and FSH secretion and a consequent reduction in testosterone production by Leydig cells (6). Obesity also promotes more aromatization of testosterone to estradiol, which further amplifies negative feedback on the HPG axis, reducing bioavailable testosterone (11). Thus, these mechanisms all suggest that obesity related inflammation may represent a central mediator which connects obesity to hormonal infertility in men.

While the link between obesity, inflammation and reproductive dysfunction is well established, few studies have objectively measured circulating IL-6 and CRP levels in hormone deficient obese males. Understanding the relationship between these inflammatory biomarkers and hormonal parameters can help with understanding the pathophysiology of male infertility and potential therapeutic targets (2). This could be particularly useful following assessments of IL-6 and CRP levels as alterations in the circulating levels of either could indicate the perturbation of endocrine systems and prompt measures, such as lifestyle change, anti-inflammatory treatments or assisted reproductive approaches to manage consequences (1).

Abundant evidence suggests a role of inflammation in the pathophysiology of male reproductive health and we therefore, intend in the present study to assess circulating IL-6 and CRP levels, as inflammatory biomarkers, their correlation with hormonal deficiency and reproductive hormones in obese men with hormonal infertility. Methods: This study aims to determine the role of systemic inflammation in male hormonal infertility by comparing these parameters with healthy controls in addition to discussing possible clinical implications in diagnosis and treatment of this condition.

Methodology:

The present study was case-control observational study conducted at girly age between February 1, 2025 and September 10, 2025 in Al-Habboubi Teaching Hospital, and Nasiriyah General Hospital, Nasiriyah, Iraq. We enrolled a sample of 150 males: 100 men with hormonal infertility and obesity, and 50 healthy age-matched controls, normal BMI. Patient group was included men between the age of 25–45 years with hormone proven infertility and BMI $\geq$ 30 kg/m², while exclusion criteria were genetic disorder, chronic systemic disease, infection, smoking and alcohol abuse, current use of hormone and antiinflammatory drugs. Healthy controls were selected based on normal BMI (18.5–24.9 kg/m²), absence of infertility, and no history of chronic or acute illnesses. Venous blood samples (10 mL) were collected from all participants after overnight fasting, with 5 mL in EDTA tubes for plasma separation and 5 mL in plain tubes for serum isolation. Samples were centrifuged at 3000 rpm for 10 minutes at 4°C, and plasma and serum aliquots were stored at −80°C until analysis. Circulating interleukin-6 (IL-6) and C-reactive protein (CRP) levels were measured as inflammatory biomarkers using commercially available enzyme-linked immunosorbent assay (ELISA) kits and high-sensitivity immunoturbidimetric assays, respectively, following the manufacturer's instructions, with all measurements performed in duplicate to ensure accuracy. Serum levels of total testosterone, free testosterone, luteinizing hormone (LH), follicle-stimulating hormone (FSH), and prolactin were assessed using ELISA, calibrated with certified reference standards and expressed in SI units.

Statistical analysis:

Data analysis was done through the SPSS version 25.0 where continuous variables were presented as the mean and standard deviation (SD) and frequencies and percentages respectively in the categorical variables. Independent t-tests and chi-square tests were used to compare patients and controls, Pearson correlation coefficient was used to identify relationships between inflammatory biomarkers, hormonal parameters, and BMI. The p-value of less than 0.05 was taken as a significant value that gave the results a reflection on the significant relationships that existed between obesity, inflammation, and hormonal dysregulation in men with hormonal infertility.

Ethical approval:

The Human Ethics Committee of the Al-Habboubi Teaching Hospital approved the study. Each of the participants was well informed about the study and he was requested to sign a written consent form. Individuals were also assured that their personal information will not leak out to other people.

Results

Sociodemographic and Clinical Characteristics of Patients with Hormonal Infertility

The data in the table indicate a comparison of sociodemographic and clinical features of patients with hormonal infertility (n=100) and healthy controls (n=50). The age of the patients was 34.5 years (±6.8) and the control group was 33.2 years (±7.1) and was not statistically significant (p=0.18). The average body mass index (BMI) of the patients was 32.4 (3.5) as opposed to 24.1 (2.9) in healthy controls, and this was statistically significant (p<0.001). In terms of smoking status, there was no significant difference in the current smokers where 28% of patients were current smokers and 24% of controls were current smokers (p=0.59). On the education aspect, 46 percent of the patients received a college education and above as opposed to 52 percent of controls (p=0.48). It was also demonstrated that 5% of patients took alcohol on a regular basis as opposed to 6% of controls, which were not statistically significant (p=0.78). Lastly there was a period of 18 months (IQR: 1230) of infertility in the patients, and there was no control data given.

Table 1: Comparison Between Obese Men with Hormonal Infertility and Healthy Controls

Variable	Patients (n = 100)	Controls (n = 50)	p-value (test)
Age, years (mean ± SD)	34.5 ± 6.8	33.2 ± 7.1	0.18 (t-test)
BMI, kg/m² (mean ± SD)	32.4 ± 3.5	24.1 ± 2.9	<0.001 (t-test)
Smoking status current, n (%)	28 (28.0%)	12 (24.0%)	0.59 (χ^2)
Education ≥college, n (%)	46 (46.0%)	26 (52.0%)	0.48 (χ^2)
Alcohol use (regular), n (%)	5 (5.0%)	3 (6.0%)	0.78 (χ^2)

Duration of infertility (months), median (IQR)	18 (12–30)	—	—

Circulating Inflammatory Biomarkers in Obese Men with Hormonal Infertility

Results The mean serum interleukin-6 (IL-6) level in the patients group was 8.6 ± 3.2 pg/ml, compare to 2.1 ± 0.9 pg/ml in the healthy group with a statistically significant difference ($p < 0.001$). Conversely, the average C-reactive protein (CRP) level was significantly higher in the patients (6.8 ± 2.9 mg/L vs 1.2 ± 0.6 mg/L in the healthy group, $p < 0.001$). Conclusions: The present results demonstrate significantly higher levels of circulating inflammatory markers in hypogonadal men with obesity relative to a healthy comparison cohort.

Table 2: Comparison of IL-6 and CRP Levels between Patients and Healthy Controls

Biomarker (unit)	Patients (n = 100) mean ± SD	Controls (n = 50) mean ± SD	p-value (t-test)
IL-6 (pg/mL)	8.6 ± 3.2	2.1 ± 0.9	<0.001
CRP (mg/L)	6.8 ± 2.9	1.2 ± 0.6	<0.001

Hormonal Profile in Obese Men with Hormonal Infertility

The results showed statistically significant differences in hormone levels between obese and hormonally infertile men and healthy controls. The mean total testosterone level in patients was 10.2 ± 3.1 nmol/L, compared to 18.4 ± 4.2 nmol/L in the healthy control group ($p < 0.001$), while the mean free testosterone level was 220 ± 65 pmol/L in patients versus 410 ± 88 pmol/L in healthy controls ($p < 0.001$). The mean luteinizing hormone (LH) level in patients was 7.6 ± 2.8 mIU/mL compared to 5.2 ± 1.9 mIU/mL in the healthy group ($p < 0.001$), and the mean follicle-stimulating hormone (FSH) level was 6.9 ± 2.5 mIU/mL compared to 4.8 ± 1.7 mIU/mL in healthy controls ($p < 0.001$). It was found that prolactin in the patients was 12.1 + 4.7 ng/mL versus 9.6 + 3.2 ng/ml in healthy controls and this was statistically significant ($p = 0.002$). These findings show that there is a great amount of hormonal imbalance relating to obesity and male hormonal infertility.

Table 3: Comparison of Testosterone, LH, FSH, and Prolactin Levels between Patients and Healthy Controls

Hormone (unit)	Patients (n = 100) mean ± SD	Controls (n = 50) mean ± SD	p-value (t-test)

Total Testosterone (nmol/L)	10.2 ± 3.1	18.4 ± 4.2	<0.001
Free Testosterone (pmol/L)	220 ± 65	410 ± 88	<0.001
LH (mIU/mL)	7.6 ± 2.8	5.2 ± 1.9	<0.001
FSH (mIU/mL)	6.9 ± 2.5	4.8 ± 1.7	<0.001
Prolactin (ng/mL)	12.1 ± 4.7	9.6 ± 3.2	0.002

Correlation Analysis between Inflammatory Biomarkers, Hormones, and BMI in Obese Men with Hormonal Infertility

Correlational analyses showed significant relationships between inflammatory markers, some hormonal markers, and body mass index (BMI) in men with obesity and hormonal infertility. IL-6 showed a strong positive correlation with BMI ($r = 0.62$, $p < 0.001$), while CRP had a moderate positive correlation with BMI ($r = 0.58$, $p < 0.001$). On the other hand, there was moderate negative correlation between IL-6 and total testosterone ($r = -0.45$, $p = 0.001$) and Association between CRP and total testosterone was also negative ($r = -0.40$, $p = 0.001$). There was also weak positive relationship between the IL-6 and luteinizing hormone (LH) ($r = 0.31$, $p = 0.001$), CRP and follicle-stimulating hormone (FSH) ($r = 0.28$, $p = 0.004$). These results indicate the role of chronic inflammation on hormonal equilibrium and how it is linked with obesity in this group of patients.

Table 4: Pearson Correlation Coefficients and p-values for IL-6, CRP, BMI, and Hormonal Parameters

Variable pair	Pearson r	p-value
IL-6 vs BMI	0.62	<0.001
CRP vs BMI	0.58	<0.001
IL-6 vs Total Testosterone	−0.45	<0.001

CRP vs Total Testosterone	−0.40	<0.001
IL-6 vs LH	0.31	0.001
CRP vs FSH	0.28	0.004

Distribution of BMI Categories among Obese Men with Hormonal Infertility and Healthy Controls

Body mass index (BMI) classification results showed a significant difference between men with hormonal infertility and obesity and the healthy control group. The percentage of patients whose BMI was normal (18.524.9) was lower than the 82.0 of the controls ($p < 0.001$). In the overweight classification (2529.9) 24 patients (24.0) occurred as opposed to the control group of only 7 persons (14.0). Obesity class I (38.0%), Obesity class II (22.0%), and Obesity class III (10.0%), had the highest percentage of obesity. The control group did not record any case of severe obesity (≥35). These findings suggest that patients with hormonal infertility are more likely to have obesity of different levels than healthy controls with high level of statistical significance.

Table 5: Comparison of BMI Classification between Patients and Controls

BMI category (kg/m²)	Patients n (%) (n=100)	Controls n (%) (n=50)	p-value (χ^2)
Normal (18.5–24.9)	6 (6.0%)	41 (82.0%)	<0.001
Overweight (25–29.9)	24 (24.0%)	7 (14.0%)	
Obesity class I (30–34.9)	38 (38.0%)	2 (4.0%)	
Obesity class II (35–39.9)	22 (22.0%)	0 (0.0%)	
Obesity class III (≥40)	10 (10.0%)	0 (0.0%)	

Discussion:

The current research examined the following sociodemographic, clinical, inflammatory, hormonal, and anthropometric difference between obese men with hormonal infertility, and a healthy control group. Sociodemographic and clinical parameter analysis showed that there were no significant difference in age, smoking status, education level, or alcohol consumption of the patients and control groups, therefore suggesting that the sociodemographic and clinical parameters were in comparative balance across the population of the study. The mean age of patients was 34.5 ± 6.8 years compared to 33.2 ± 7.1 years in controls ($p = 0.18$), suggesting that age did not confound the associations observed in subsequent analyses. This is consistent with earlier research suggesting that, in isolation, age does not differentially classify hormonal infertility men from men from the general population (17, 5). In contrast, there was a striking difference in BMI, patients having a mean BMI of 32.4 ± 3.5 kg/m² compared to 24.1 ± 2.9 kg/m² in controls ($p < 0.001$). This reinforces the established link of obesity with the hormonal type of infertility, in line with earlier reports highlighting the negative impact of adiposity excess on males reproduction heath (20). Confirming this, the distribution of BMI categories showed an even fewer (6%) normal BMI patients in relation to control BMI (82%), whereas overweight and class I obesity were around double that of the control group, and class II obesity and class III obesity categories were virtually exclusively patients currently unable to conceive.

Mean IL-6 levels were 8.6 ± 3.2 pg/mL in patients compared to 2.1 ± 0.9 pg/mL in controls, and CRP levels were 6.8 ± 2.9 mg/L versus 1.2 ± 0.6 mg/L in controls, both with $p < 0.001$. These results demonstrate a significant systemic pro-inflammatory state in this patient population, which is consistent with previous studies reporting that obesity is associated with chronic low-grade inflammation and elevated circulating cytokines, including IL-6 and CRP (12, 8). The elevated inflammatory markers are hypothesized to impair hypothalamic-pituitary-gonadal axis function, contributing to hormonal dysregulation and reduced spermatogenesis, as observed in obese infertile men (3).

Hormonal analysis revealed significant disturbances in reproductive hormones among patients. Total and free testosterone levels were significantly lower in patients compared to controls (10.2 ± 3.1 nmol/L vs. 18.4 ± 4.2 nmol/L; 220 ± 65 pmol/L vs. 410 ± 88 pmol/L, $p < 0.001$), while LH and FSH levels were elevated in patients (7.6 ± 2.8 mIU/mL and 6.9 ± 2.5 mIU/mL, respectively) compared to controls, suggesting a compensatory gonadotropin response. Prolactin was also elevated in patients (12.1 ± 4.7 ng/mL vs. 9.6 ± 3.2 ng/mL, $p = 0.002$). These findings are consistent with studies showing hypogonadotropic hypogonadism in obese men due to aromatization of testosterone to estradiol in adipose tissue and subsequent negative feedback on the hypothalamus and pituitary (24, 14). The increased LH and FSH will be pointing to a disruption in the feedback mechanisms which can reflect partial primary testicular dysfunction.

The interaction between obesity, inflammation, and hormonal imbalance was also explained through correlation analyses. There was a strong positive correlation between IL-6 and CRP and BMI ($r = 0.62$ and 0.58, respectively, $p < 0.001$) and a weak negative correlation between the markers and total testosterone ($r = 0.45$ and 0.40, $p < 0.001$). Also, the IL-6 was positively correlated with LH ($r = 0.31$, $p = 0.001$) and the CRP with FSH ($r = 0.28$, $p = 0.004$). The findings are in line with the hypothesis that chronic inflammation among obese men leads to hormonal dysregulations, which is also consistent with

previous studies that show an inverse correlation between inflammatory cytokines and the levels of testosterone in obese men (10, 18). The mechanistic underlying is most probably due to inhibited steroidogenesis in Leydig cells by cytokines and modulated hypothalamic secretion of GnRH.

All in all, the current results emphasize the multifactorial nature of hormonal infertility in obese men and cite combined effects of high BMI, chronic systemic inflammation, and impaired endocrine function. The findings largely correspond to the literature, but certain studies have found less significant correlations between inflammatory markers and reproductive hormones in non-obese cohorts and that obesity is a major modifier of the association (25). The clinical implications of these findings are evident, as it may be proposed that hormonal profiles and reproductive outcomes may be enhanced in the case of obese and inflamed men by the means of their management.

Conclusion:

The present study concludes that IL-6 and CRP are good inflammatory biomarkers and are observed to be significantly high in the obese men having hormonal infertility than the healthy controls. The results indicate that chronic inflammation of low grade in obesity interferes with the hypothalamic-pituitary-gonadal axis, resulting in poor production of testosterone and hormonal imbalance. These findings underscore the significance of observing IL-6 and CRP as diagnostic and prognostic indicators, which provide information on the pathophysiology of obesity-inflammation-male infertility relationships.

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