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Investigating the Impact of Sex Hormones on Obesity-Related Secondary Infertility in Women

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Abstract

General Background: Secondary infertility remains a significant reproductive health concern worldwide, and obesity has emerged as an important factor associated with endocrine and metabolic disturbances that may compromise female fertility. **Specific Background:** Obesity has been linked to alterations in gonadotropin secretion, thyroid function, prolactin levels, and lipid metabolism; however, evidence regarding these associations among women with secondary infertility remains limited. **Knowledge Gap:** Data evaluating hormonal and metabolic differences between obese and non-obese women with secondary infertility, particularly in Iraqi populations, are scarce. **Aims:** This study aimed to compare reproductive hormones, thyroid function, prolactin levels, and lipid profiles between obese and non-obese women diagnosed with secondary infertility. **Results:** Obese women demonstrated significantly lower luteinizing hormone and thyroid-stimulating hormone levels, together with significantly higher total cholesterol and triglyceride concentrations than non-obese women. Prolactin levels were elevated in both groups without significant between-group differences. Age-adjusted analyses confirmed the persistence of the luteinizing hormone and lipid profile differences. Body mass index correlated with triglycerides in obese women and with cholesterol in non-obese women. **Novelty:** The study provides evidence from a carefully selected secondary infertility cohort while excluding major confounding reproductive and endocrine conditions. **Implications:** The findings support the incorporation of early body mass index assessment, metabolic screening, and weight-management strategies into the evaluation and management of secondary infertility.

Keywords: Secondary Infertility, Obesity, Luteinizing Hormone, Lipid Profile, Reproductive Endocrinology

Key Findings Highlights

Reduced gonadotrophin concentrations were identified among participants with elevated body mass index.
Metabolic abnormalities were characterized by increased cholesterol and triglyceride concentrations.
Age-adjusted analyses confirmed the robustness of the endocrine and biochemical differences.

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Introduction

Infertility is defined as the failure to achieve clinical pregnancy after twelve months or more of regular, unprotected intercourse, and is conventionally separated into a primary form, in which conception has never occurred, and a secondary form, in which a couple has previously conceived but is currently unable to do so [1]. The most recent WHO synthesis estimates that around 17.5% of adults experience infertility at some point in life, with comparable burdens across high- and low-income settings [1]. Female factors account for roughly one third of cases, and ovulatory dysfunction remains the single largest contributor [2].

Adiposity is now a well-established modifiable determinant of subfertility. Excess fat tissue disturbs the hypothalamic–pituitary–ovarian (HPO) axis at several levels: pulsatile GnRH release is altered, the LH/FSH ratio is shifted, insulin resistance promotes ovarian androgen production, and peripheral aromatisation of androgens increases tonic oestrogen exposure [3,4]. Dysregulated adipokine signalling, in particular hyperleptinaemia and low adiponectin, further blunts follicular development and endometrial receptivity [5]. The clinical consequence is anovulation, oligomenorrhoea and reduced cycle fecundity; each unit increase in BMI above 29 kg/m² has been associated with approximately a 4% decline in spontaneous pregnancy rates [3].

obesity in the reproductive years rarely occurs alone. it clusters with polycystic ovary syndrome (pcos), subclinical thyroid dysfunction and atherogenic dyslipidaemia, each of which can independently impair fertility [6,7]. high-normal tsh values within the euthyroid range have been linked to hyperandrogenism and to poorer outcomes in assisted reproduction [8]. hyperprolactinaemia, classically attributed to pituitary adenoma, is also reported in women with metabolic disturbance and is a recognised cause of luteal-phase defect and secondary amenorrhoea [9].

in routine iraqi reproductive-medicine practice body weight is still often considered only after several unsuccessful cycles of empirical ovulation induction. to address this gap, the present study compared circulating gonadotrophins, prolactin, testosterone, tsH and a basic lipid profile between obese and non obese women presenting with secondary infertility, and examined how these variables relate to BMI within each stratum.

2. Materials and Methods

2.1 Study design and participants

A single-centre cross-sectional comparative study was conducted between September 2023 and April 2024 at the outpatient infertility clinic of Kamal Al-Samarrai Hospital for Fertility and IVF, Baghdad. Eligible participants were married women aged 18–42 years with a clinical diagnosis of secondary infertility, defined as the inability to conceive for at least twelve months following a previous pregnancy (live birth or miscarriage). the minimum interval since the last pregnancy was 18 months. exclusion criteria were: current pregnancy or lactation; known pituitary or adrenal disease; macroprolactinoma or prolactin > 100 ng/ml; overt thyroid disease on treatment; type 1 or type 2 diabetes; PCOS diagnosed by the 2003 Rotterdam criteria; current use of hormonal contraception, GnRH analogues, metformin or ovulation-induction agents; and a male-factor cause of infertility documented by semen analysis. After screening, 118 women were enrolled consecutively.

2.2 Ethics statement

The protocol was reviewed and approved by the Research Ethics Committee of the College of Science, Al-Nahrain University (approval no. CoS/REC/2023/14) and by the institutional review board of Kamal Al-Samarrai Hospital. The study conformed to the principles of the Declaration of Helsinki. Written informed consent was obtained from every participant before any procedure.

2.3 Sample size

Sample size was estimated for the primary comparison of serum LH between the two groups. Assuming a between-group difference of 1.0 IU/L with a pooled SD of 1.8 IU/L (effect size d ≈ 0.55), an α of 0.05 (two-sided) and a power of 0.80, a minimum of 53 participants per group was required. Recruitment was continued to 60 obese and 58 non-obese women to allow for a small loss to incomplete biochemistry.

2.4 Anthropometric measurements

Standing height was measured to the nearest 0.5 cm using a wall-mounted stadiometer, and body weight to the nearest 0.1 kg in light clothing without shoes. BMI was calculated as weight in kilograms divided by the square of height in metres. Participants with BMI ≥ 30 kg/m² were classified as obese (n = 60); those with BMI < 30 kg/m² as non-obese (n = 58).

2.5 Blood sampling and biochemical analyses

Venous blood (5 mL) was drawn between 08:00 and 10:00 after an overnight fast of at least ten hours, during the early follicular phase (cycle days 2–5). in women with oligomenorrhoea, sampling was performed after withdrawal bleeding induced by a short course of medroxyprogesterone acetate. samples were allowed to clot for thirty minutes, centrifuged at 3000 rpm for five minutes, and the serum aliquots stored at −20 °c until analysis. fsh, lh, prolactin, total testosterone and tsh were measured by enzyme-linked fluorescent assay on the mini-vidas analyser (biomérieux, france) using the manufacturer's calibrated kits. total cholesterol and triglycerides were determined enzymatically using commercial reagents (bioMérieux). All samples were run in duplicate; intra-assay and inter-assay coefficients of variation were below 6% and 8% respectively.

2.6 Statistical analysis

Data were analysed with SPSS version 26.0 (IBM, USA). Continuous variables are expressed as mean ± SD; skewed variables (prolactin,

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triglycerides, testosterone) are additionally summarised as median (interquartile range). Normality was tested with the Shapiro–Wilk test. Normally distributed variables were compared between groups using the independent-samples student t-test, and skewed variables either by mann–whitney u or after natural-log transformation; in this dataset both approaches gave concordant results, and parametric p-values are reported for ease of presentation. the 95% confidence intervals for mean differences were obtained using the welch formula. to adjust for the small age difference between groups, multivariable linear regression was used with each biochemical parameter as the dependent variable and group (obese vs. non-obese), age and duration since the last pregnancy as covariates. associations within each stratum were assessed using pearson's correlation coefficient (spearman's where skewness persisted). a two-tailed p-value below 0.05 was considered significant; $p < 0.01$ was considered highly significant.

3. Results

3.1 Cohort characteristics

Of 134 women initially screened, 118 met the inclusion criteria and completed biochemical assessment. The two groups were broadly comparable in stature, but the obese group was on average older and heavier (Table 1). Mean BMI was 31.2 ± 3.4 kg/m² in obese women compared with 22.4 ± 1.6 kg/m² in non-obese women ($p < 0.001$). The age difference, though modest, reached statistical significance ($p = 0.018$). The median interval since the last pregnancy was 4.2 years (IQR 2.5–6.8) in the obese group and 3.7 years (IQR 2.1–5.9) in the non-obese group ($p = 0.21$).

Table 1. Demographic and anthropometric characteristics of the study population.

Variable	Obese (n = 60)	Non-obese (n = 58)	p-value
Age (years)	33.2 ± 6.9	27.5 ± 6.7	0.018*
Height (cm)	159.1 ± 5.3	160.0 ± 7.1	0.448
Weight (kg)	78.4 ± 9.1	59.6 ± 6.4	<0.001**
BMI (kg/m²)	**31.2 ± 3.4**	**22.4 ± 1.6**	**<0.001****
Years since last pregnancy, median (IQR)	4.2 (2.5–6.8)	3.7 (2.1–5.9)	0.21

*Values are mean ± SD unless stated. * p < 0.05; ** p < 0.001. Comparison by independent-samples t-test (continuous) or Mann–Whitney U (median).*

3.2 Hormonal and lipid profile

the full hormonal and lipid comparison, together with mean differences and 95% confidence intervals, is summarised in table 2. mean serum lh was lower in obese than in non-obese women (2.71 ± 1.42 vs. 3.72 ± 1.93 iu/l; mean difference 1.01 iu/l, 95% ci 0.39–1.63; $p = 0.002$), and the lh/fsh ratio was correspondingly lower in obese women (0.40 vs. 0.56; $p = 0.012$). tsh was modestly but significantly lower in the obese group (2.41 ± 1.18 vs. 2.89 ± 1.24 miu/l; $p = 0.033$). mean prolactin was above the upper reference limit of 20 ng/ml in both groups, with no significant difference between strata. fsh and total testosterone did not differ between the groups. both total cholesterol and triglycerides were significantly higher in obese women ($p < 0.001$ and $p = 0.005$ respectively), consistent with the atherogenic lipid pattern of central adiposity.

after multivariable adjustment for age and duration since the last pregnancy, the between-group differences in lh (adjusted $\beta = -0.86$ iu/l, 95% ci −1.51 to −0.21; $p = 0.010$), cholesterol (adjusted $\beta = 32.4$ mg/dl, 95% ci 20.1–44.7; $p < 0.001$) and triglycerides (adjusted $\beta = 28.7$ mg/dL, 95% CI 6.9–50.5; $p = 0.011$) remained significant. The TSH difference was attenuated but retained borderline significance (adjusted $p = 0.058$).

Table 2. Biochemical parameters in obese and non-obese women with secondary infertility.

Parameter	Obese (n = 60)	Non-obese (n = 58)	p-value	95% CI for difference
Reproductive hormones				
FSH (IU/L)	6.84 ± 2.97	6.62 ± 2.48	0.671	−0.78 to 1.22
LH (IU/L)	2.71 ± 1.42	3.72 ± 1.93	0.002**	0.39 to 1.63
LH / FSH ratio	0.40 ± 0.18	0.56 ± 0.25	0.012*	0.04 to 0.28
Prolactin (ng/mL) †	26.4 ± 13.7	24.8 ± 16.9	0.572	−4.0 to 7.2
Total testosterone (ng/mL)	0.33 ± 0.16	0.27 ± 0.17	0.143	−0.02 to 0.14
Thyroid axis				
TSH (mIU/L)	2.41 ± 1.18	2.89 ± 1.24	0.033*	0.04 to 0.92
Lipid profile				

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Parameter	Obese (n = 60)	Non-obese (n = 58)	p-value	95% CI for difference
Total cholesterol (mg/dL)	215.8 ± 36.4	180.6 ± 31.3	<0.001**	22.8 to 47.6
Triglycerides (mg/dL) †	142.3 ± 62.8	110.5 ± 58.4	0.005**	9.7 to 54.0

*Values are mean ± SD. * p < 0.05; ** p < 0.01. † Log-transformed before parametric testing; results were concordant with Mann–Whitney U. CI direction expressed for higher-minus-lower group means.*

3.3 Correlation analyses

Within the obese subgroup, BMI correlated positively with triglycerides (r = 0.41, p = 0.008), and showed a weaker positive trend with prolactin (r = 0.22, p = 0.092). TSH showed a moderate positive correlation with FSH (r = 0.34, p = 0.031). In the non-obese subgroup the pattern was different: BMI correlated positively with cholesterol (r = 0.42, p = 0.039); TSH was positively associated with both cholesterol (r = 0.59, p = 0.002) and triglycerides (r = 0.48, p = 0.015), and negatively with weight (r = −0.39, p = 0.046). the principal coefficients are presented in Table 3.

Table 3. Significant Pearson correlations involving BMI and biochemical parameters in each group.

Variable pair	Obese group, r (p)	Non-obese group, r (p)
BMI – Weight	0.82 (<0.001)**	0.58 (0.003)**
BMI – Triglycerides	0.41 (0.008)**	−0.28 (0.175)
BMI – Cholesterol	0.17 (0.310)	0.42 (0.039)*
BMI – Prolactin	0.22 (0.092)	0.18 (0.391)
TSH – FSH	0.34 (0.031)*	0.32 (0.116)
TSH – Cholesterol	0.18 (0.281)	0.59 (0.002)**
TSH – Triglycerides	0.07 (0.658)	0.48 (0.015)*
TSH – Weight	−0.10 (0.539)	−0.39 (0.046)*
LH – Prolactin	0.18 (0.269)	0.06 (0.790)

*Pearson coefficient (r) with two-tailed p-value. * p < 0.05; ** p < 0.01.*

4. Discussion

The principal finding of this study is that women with secondary infertility and BMI in the obese range show a measurably lower LH and lower TSH together with an atherogenic lipid pattern compared with their non-obese counterparts of the same clinical phenotype. the difference in lh (mean 1.01 iu/l, 95% ci 0.39–1.63) persisted after adjustment for age and duration since last pregnancy, which argues against confounding by reproductive senescence. the magnitude of the lh difference is in keeping with the report of dardar et al. (mean lh 2.9 vs. 4.1 iu/l in obese vs. lean infertile libyan women) [11] and with the indian cross-sectional study of goswami et al., who found a similar downward shift of pituitary gonadotrophins in obese women with secondary infertility [10].

the depressed lh and lh/fsh ratio in the obese group deserves a comment. classical pcos is associated with an elevated lh/fsh ratio, and at first reading our finding seems to contradict that. the discrepancy reflects the case-mix: pcos was an exclusion criterion, and the obese women in this cohort represent simple obesity rather than the polycystic ovary phenotype. in simple obesity without pcos, several authors have described a relative reduction in lh pulse amplitude attributed to hyperinsulinaemia, increased opioidergic tone in the hypothalamus, and increased peripheral oestrone exposure from aromatisation in adipose tissue [5,7]. the present data is compatible with that mechanism.

mean prolactin was above the upper reference limit of 20 ng/ml in both groups, with no significant between-group difference. hyperprolactinaemia is a recognised cause of luteal-phase defect and secondary amenorrhoea, and the elevation observed in our cohort may itself contribute to the infertile phenotype independent of bmi [9,11]. mild hyperprolactinaemia in the absence of a pituitary lesion is well described in women with stress, insulin resistance and subclinical thyroid dysfunction [8,12]; the modestly lower tsh and high prolactin observed in the obese group are therefore not necessarily independent findings, but the loss of statistical significance for tsh after age adjustment urges caution in interpreting the thyroid signal.

the lipid findings are unsurprising in direction but informative in magnitude. triglycerides scaled with bmi within the obese stratum, whereas in the non-obese stratum it was cholesterol that scaled with bmi. a plausible interpretation is that the two strata occupy different points on the dyslipidaemic trajectory: in lean and overweight women, modest bmi increments primarily push total cholesterol upwards, while once obesity is established the triglyceride-rich vldl fraction becomes the dominant abnormality. The strong positive correlation of TSH with both cholesterol and triglycerides in the non-obese stratum agrees with recent work showing that even high-normal TSH values are associated with an atherogenic lipid pattern and with poorer reproductive outcomes [8,13].

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4.1 Strengths and limitations

The main strengths of this work are the use of a clearly defined secondary-infertility population, the exclusion of PCOS, overt thyroid disease and male-factor cases that would otherwise confound the hormonal comparison, sampling restricted to the early follicular phase, and the use of both non-parametric and age-adjusted analyses to test the robustness of the findings. Limitations should also be acknowledged. The study is single-centre and modest in size; insulin, HOMA-IR, oestradiol, anti-Müllerian hormone and waist-to-hip ratio were not measured, which would have allowed a fuller mechanistic interpretation and an assessment of central adiposity. some women with oligomenorrhoea required progestin-induced bleeding to define cycle day, which may have introduced minor heterogeneity in baseline hormone levels. The cross-sectional design does not allow inference about causality between obesity and the observed endocrine differences.

4.2 Clinical implications

The data argue for the integration of bmi assessment and basic lipid and thyroid screening early in the workup of secondary infertility, rather than after several rounds of empirical ovulation induction. a meaningful proportion of women in the obese stratum can be expected to resume ovulatory cycles with even modest weight reduction [3,5,14], offering a low-cost first-line intervention before assisted reproductive technologies are considered. where weight loss is not achievable by lifestyle measures alone, recent evidence on glp-1 receptor agonists in PCOS and obesity-associated subfertility may broaden the therapeutic options [7].

5. Conclusion

In this cohort of Iraqi women with secondary infertility, obesity was accompanied by significantly lower LH and TSH and by significantly higher total cholesterol and triglycerides; prolactin was elevated regardless of bmi. the lh and lipid differences persisted after adjustment for age. bmi tracked with triglycerides in the obese stratum and with cholesterol in the non-obese stratum. together, these findings support a measurable influence of adiposity on the endocrine and metabolic environment of secondary infertility, and indicate that weight management warrants earlier consideration in the diagnostic and therapeutic pathway.

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Author contributions

A.H.J. conceptualised the study and supervised the project. S.A.I. and D.M.H. carried out patient recruitment, anthropometric measurements and sample collection. Z.H.J. performed the biochemical assays and contributed to data curation. A.H.J. and Z.H.J. carried out the statistical analysis. A.H.J. drafted the manuscript; all authors critically revised the manuscript and approved the final version.

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Conflict of interest

The authors declare no conflict of interest.

Data availability

The anonymised dataset supporting the findings of this study is available from the corresponding author upon reasonable request.

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