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Original Research Article

Assessment of serum prolactin, thyroid and cortisol levels among infertile females in Bangladesh: a cross-sectional study

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ABSTRACT

Background: Female infertility may involve endocrine disturbances affecting reproductive function. This study compared serum prolactin, thyroid hormones, cortisol, and related metabolic-reproductive parameters among healthy controls and women with primary or secondary infertility in Bangladesh.
Methods: This comparative cross-sectional study was conducted at the Department of Physiology, Ibrahim Medical College, Dhaka, from January to December 2024. It included 150 legally adult married women: 50 healthy controls, 50 with primary infertility, and 50 with secondary infertility, recruited from the infertility unit of Bangabandhu Sheikh Mujib Medical University (BSMMU), Dhaka. Fasting venous blood was collected on day 2 of the menstrual cycle; morning cortisol was measured from samples collected at 9:00 AM. Serum prolactin, fasting and 2-hour blood glucose, FSH, LH, TSH, T3, T4, and cortisol were measured by chemiluminescent immunoassay. Data were analyzed using SPSS version 26.0.
Results: BMI was significantly higher in primary infertility (27.99±4.17 kg/m²) and secondary infertility (27.62±3.68 kg/m²) than controls (25.04±3.91 kg/m²; $p<0.001$). Prolactin was higher in primary (19.59±9.11) and secondary infertility (18.79±8.10) than controls (10.58±5.01; $p<0.001$). Cortisol was also higher in primary (23.24±4.98) and secondary infertility (21.95±4.85) than controls (9.38±2.94; $p<0.001$). TSH differed significantly, highest in secondary infertility (3.24±2.28; $p<0.001$), whereas T3 and T4 did not differ significantly. Cortisol strongly correlated with prolactin among infertile women ($r=0.765$, $p<0.001$).
Conclusions: Infertile women had higher prolactin, cortisol, BMI, and selectively elevated TSH. Combined endocrine and metabolic assessment may aid infertility evaluation.

Keywords: Female infertility, Prolactin, Thyroid function, Cortisol

INTRODUCTION

Infertility is a clinically important reproductive health problem, commonly defined as failure to achieve pregnancy after at least 12 months of regular unprotected intercourse.[1,2] Although infertility may arise from male, female, combined, or unexplained factors, endocrine disturbance remains a major and potentially correctable contributor to female infertility. The hypothalamic-pituitary-ovarian axis regulates follicular development, ovulation, luteal function, and endometrial preparation through coordinated interactions among gonadotropins, ovarian steroids, thyroid hormones, prolactin, and stress-related neuroendocrine pathways.[3,4] Disruption at any level of this axis may present as anovulation, menstrual irregularity, luteal phase defect, impaired implantation, or

reduced fecundability. In resource-limited clinical settings, including Bangladesh, endocrine evaluation is particularly relevant because several abnormalities, especially thyroid dysfunction and hyperprolactinemia, are detectable through relatively accessible laboratory tests and may be medically manageable when identified early. Prolactin has a recognized inhibitory effect on reproductive function when present in excess. Hyperprolactinemia can suppress pulsatile gonadotropin-releasing hormone secretion, alter follicle-stimulating hormone and luteinizing hormone dynamics, and contribute to ovulatory dysfunction. Thyroid dysfunction is closely related to this pathway because hypothyroidism may increase thyrotropin-releasing hormone activity, which can stimulate both thyroid-stimulating hormone and prolactin secretion. Clinical studies from South Asia and other settings have repeatedly observed higher prolactin and TSH levels among infertile women than among fertile controls. In Bangladesh, Bari et al. studied women with primary infertility, secondary infertility, and fertile controls, and reported significantly higher TSH and prolactin levels in the infertility groups, with positive correlations between TSH and prolactin in both primary and secondary infertility.[5] A more recent Bangladeshi study by Chowdhury et al. also reported higher frequencies of thyroid dysfunction and hyperprolactinemia among subfertile women than fertile controls.[6] Similar observations have been reported in Nepal by Bhandari et al., where primary infertility cases had higher prolactin and TSH levels than controls, and in India by Sharma et al., who observed thyroid and prolactin abnormalities across primary and secondary infertility groups.[7,8] Keerthanaa and Hiremath further reported hypothyroidism and hyperprolactinemia among infertile women with menstrual irregularities, supporting the clinical relevance of combined thyroid-prolactin assessment.[9] The relationship between thyroid function and prolactin is supported by broader evidence. Ramadras et al, in a systematic review and meta-analysis, found a positive moderate correlation between serum prolactin and TSH among infertile women.[10] Bassey et al also reported altered thyroid hormone and prolactin profiles among infertile women in Southern Nigeria, indicating that this endocrine pattern is not restricted to South Asian populations.[11] However, not all prolactin abnormalities occur in the presence of thyroid dysfunction. Badesara and Jakhar, in infertile euthyroid women, showed that hyperprolactinemia may still occur despite normal thyroid status.[12] These findings suggest that thyroid function and prolactin should be assessed together, while also being interpreted as distinct but interacting endocrine domains. Cortisol adds another clinically relevant dimension to endocrine assessment in infertility. As the principal glucocorticoid of the hypothalamic-pituitary-adrenal axis, cortisol reflects biological stress response and may interact with reproductive neuroendocrine regulation. Wdowiak et al observed interactions of cortisol and prolactin with other menstrual-cycle hormones among infertile women, indicating that stress-related endocrine pathways may influence conception probability.[13] Broader reviews of thyroid disease and reproduction also emphasize the reproductive relevance of thyroid dysfunction and thyroid autoimmunity, particularly in women seeking fertility care or assisted conception.[14,15] Despite this evidence, combined evaluation of prolactin, thyroid profile, cortisol, and related metabolic-reproductive hormones remains limited in Bangladeshi infertility research. The present study was therefore designed to compare serum prolactin, thyroid hormones, cortisol, and available metabolic-reproductive parameters among healthy controls, women with primary infertility, and women with secondary infertility in Bangladesh.

METHODS

This comparative cross-sectional study was conducted over a period of 12 months, from January 2024 to December 2024. All the study participants were selected from women attending the infertility unit of Bangabandhu Sheikh Mujib Medical University (BSMMU), Dhaka. A total of 150 legally adult married women were included and divided into three equal groups: healthy control women, women with primary infertility, and women with secondary infertility, with 50 participants in each group.

Women with infertility were enrolled if they had been unable to conceive after at least one year of regular unprotected intercourse. Primary infertility was defined as inability to conceive despite at least one year of unprotected intercourse with no previous conception. Secondary infertility was defined as inability to conceive after at least one previous conception. The control group consisted of apparently healthy female volunteers who visited the hospital for other non-relevant reasons.

Women with a previous history of endocrine disorders, current hormonal therapy, pregnancy, lactation, steroid use, severe systemic illness, or other conditions likely to interfere with reproductive or endocrine function were excluded. After obtaining written informed consent, relevant demographic and clinical information was recorded using a structured data collection form.

Fasting venous blood samples were collected from all participants on day 2 of the menstrual cycle. Morning serum cortisol was measured from samples collected at 9:00 AM. Serum prolactin, fasting blood glucose, 2-hour blood glucose, follicle-stimulating hormone, luteinizing hormone, thyroid-stimulating hormone, triiodothyronine, thyroxine, and cortisol levels were measured at the laboratory of Ibrahim Medical College using chemiluminescent immunoassay and standard laboratory procedures.

Data were entered, cleaned, and analyzed using SPSS version 26.0. Quantitative variables were expressed as mean±standard deviation, while categorical variables were summarized as frequency and percentage. Group-wise comparisons among controls, primary infertility, and secondary infertility groups were performed using one-

way analysis of variance. Post-hoc analysis was applied where appropriate. Correlations between hormonal parameters were assessed using correlation analysis. A p-value <0.05 was considered statistically significant. Ethical approval was obtained from the institutional ethics review committee.

RESULTS

The mean age was comparable across the control, primary infertility, and secondary infertility groups: 27.82±4.65 years, 27.08±4.15 years, and 28.98±4.81 years, respectively, with no significant difference between groups (p=0.113). Mean weight and BMI were significantly higher in both infertility groups compared with controls; BMI was 25.04±3.91 kg/m² in controls, 27.99±4.17 kg/m² in primary infertility, and 27.62±3.68 kg/m² in secondary infertility (p<0.001) (Table 1).

Mean serum prolactin was significantly higher in the primary and secondary infertility groups, 19.59±9.11 and 18.79±8.10, respectively, compared with controls, 10.58±5.01 (p<0.001). FSH, LH, TSH, and cortisol also differed significantly among the three groups, while T3 and T4 did not show statistically significant group differences (Table 2).

Table 1: Baseline characteristics of the study participants by group.

Characteristics	Control, n=50	Primary infertility, n=50	Secondary infertility, n=50	Test statistic	P value
Weight, kg	52.82±7.09	57.40±8.83	57.22±8.72	F=4.939	0.008
BMI, kg/m²	25.04±3.91	27.99±4.17	27.62±3.68	F=8.439	<0.001
Age category, N (%)					
≤25 years	16 (32.0)	21 (42.0)	13 (26.0)	χ^2=3.799	0.704
26 to 30 years	20 (40.0)	17 (34.0)	20 (40.0)		
31 to 35 years	10 (20.0)	10 (20.0)	12 (24.0)		
>35 years	4 (8.0)	2 (4.0)	5 (10.0)		
Mean±SD	27.82±4.65	27.08±4.15	28.98 ± 4.81	F=2.216	0.113

Values are presented as mean ± SD or n (%). p-values for continuous variables are from one-way ANOVA; p-value for age category is from chi-square test.

Table 2: Hormonal and metabolic profile of the study participants by group.

Parameters	Control, n=50	Primary infertility, n=50	Secondary infertility, n=50	F statistic	P value
Cortisol	9.38±2.94	23.24±4.98	21.95±4.85	154.460	<0.001
Prolactin	10.58±5.01	19.59±9.11	18.79±8.10	21.462	<0.001
TSH	1.72±0.84	2.21±1.71	3.24±2.28	10.147	<0.001
T3	4.00±1.50	4.33±2.72	4.93±2.31	2.226	0.112
T4	13.09±4.85	13.79±4.99	12.97±4.80	0.406	0.667
FSH	6.48±3.83	3.73±2.03	4.66±2.23	12.331	<0.001
LH	8.37±5.16	4.86±3.15	3.47±2.03	23.460	<0.001

Values are presented as mean ± SD. p-values are from one-way ANOVA.

Table 3: Thyroid status of the study participants by group.

Thyroid status	Control, n=50	Primary infertility, n=50	Secondary infertility, n=50	P value
Euthyroid	47 (94.0)	43 (86.0)	36 (72.0)	0.035
Hypothyroid	2 (4.0)	6 (12.0)	13 (26.0)	
Hyperthyroid	1 (2.0)	1 (2.0)	1 (2.0)	

Euthyroid status was most frequent in all groups, observed in 94.0% of controls, 86.0% of primary infertility participants, and 72.0% of secondary infertility participants. Hypothyroidism was more frequent in secondary infertility, 26.0%, than in primary infertility, 12.0%, and controls, 4.0%, while hyperthyroidism was uncommon and equally distributed across the three groups, 2.0% each. The overall thyroid-status distribution differed significantly across groups (p=0.035) (Table 3).

When examining the prevalence of elevated hormone levels among participants with primary and secondary infertility (n=100), elevated serum cortisol (>20 µg/dl) was present in 21 participants (24.0% of primary infertility, 18.0% of secondary infertility). Elevated prolactin (>28 ng/ml) was detected in 12 participants (14.0% of primary infertility, 10.0% of secondary infertility). Elevated TSH (>4.0 mIU/l) was observed in 19 participants, more frequently in secondary infertility

(26.0%) than primary infertility (12.0%). The overall distribution of elevated hormone levels did not differ significantly between the two infertility groups (χ^2=3.269, p=0.195) (Table 4).

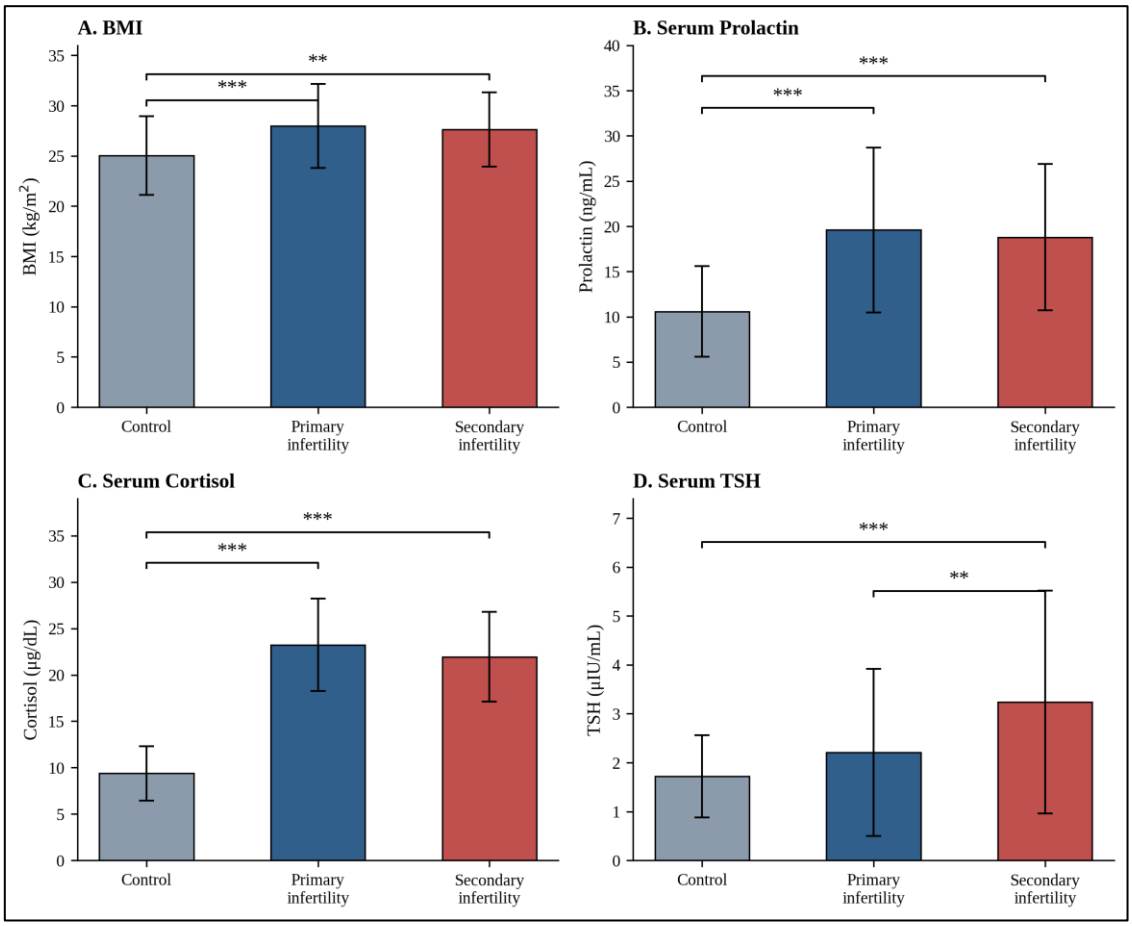


Figure 1: Comparison of BMI and key hormonal parameters across study groups. Mean (± SD) BMI (A), serum prolactin(B), serum cortisol (C), and serum TSH (D) among controls, women with primary infertility, and women with secondary infertility. Error bars represent standard deviation. **p<0.01, ***p<0.001 (Tukey post-hoc comparison vs. indicated group; see Table 5).

Table 4: Cortisol, prolactin and thyroid status of the study participants by group.

Hormone status	Primary infertility, n=50	Secondary infertility, n=50	P value
High level cortisol	12 (24.0)	9 (18.0)	
High level prolactin	7 (14.0)	5 (10.0)	0.195
High level of TSH	6 (12.0)	13 (26.0)	

Table 5: Pearson correlation of cortisol and selected endocrine parameters by group.

Parameters	Primary minus control	P value	Secondary minus control	P value	Secondary minus primary	P value
Weight, kg	4.58	0.017	4.40	0.023	-0.18	0.994
BMI, kg/m²	2.96	<0.001	2.59	0.004	-0.37	0.884
Cortisol	13.87	<0.001	12.57	<0.001	-1.29	0.303
Prolactin	9.01	<0.001	8.21	<0.001	-0.80	0.858
TSH	0.48	0.337	1.51	<0.001	1.03	0.009
FSH	-2.75	<0.001	-1.82	0.004	0.93	0.229
LH	-3.51	<0.001	-4.89	<0.001	-1.38	0.149

Mean differences are calculated as the second named group minus the first named group. p-values are Tukey-adjusted.

Table 6: Pearson correlation of cortisol and selected endocrine parameters by group.

Correlation pair	Control, n=50	Primary infertility, n=50	Secondary infertility, n=50	Infertility combined, n=100	Overall, n=150
Cortisol vs prolactin	r=-0.050; p=0.731	r=0.796; p<0.001	r=0.733; p<0.001	r=0.765; p<0.001	r=0.714; p<0.001
Cortisol vs TSH	r=0.100; p=0.488	r=0.113; p=0.434	r=0.033; p=0.822	r=0.031; p=0.757	r=0.234; p=0.004

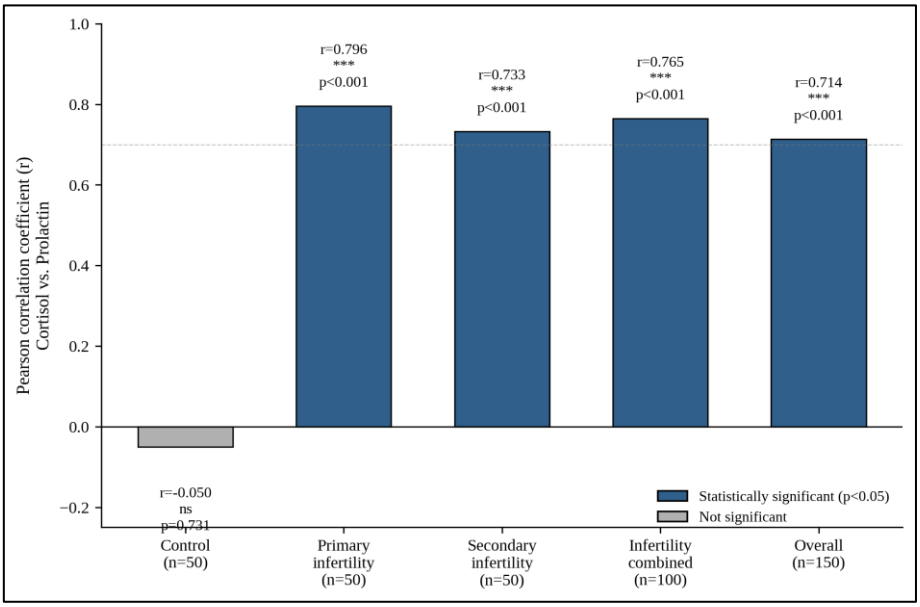


Figure 2: Correlation between serum cortisol and serum prolactin by study group. Pearson correlation coefficient (r) between serum cortisol and serum prolactin by study group. Bars in blue indicate a statistically significant correlation (p<0.05); the bar in gray (control group) indicates no significant correlation. ***p<0.001; ns = not significant (see Table 6).

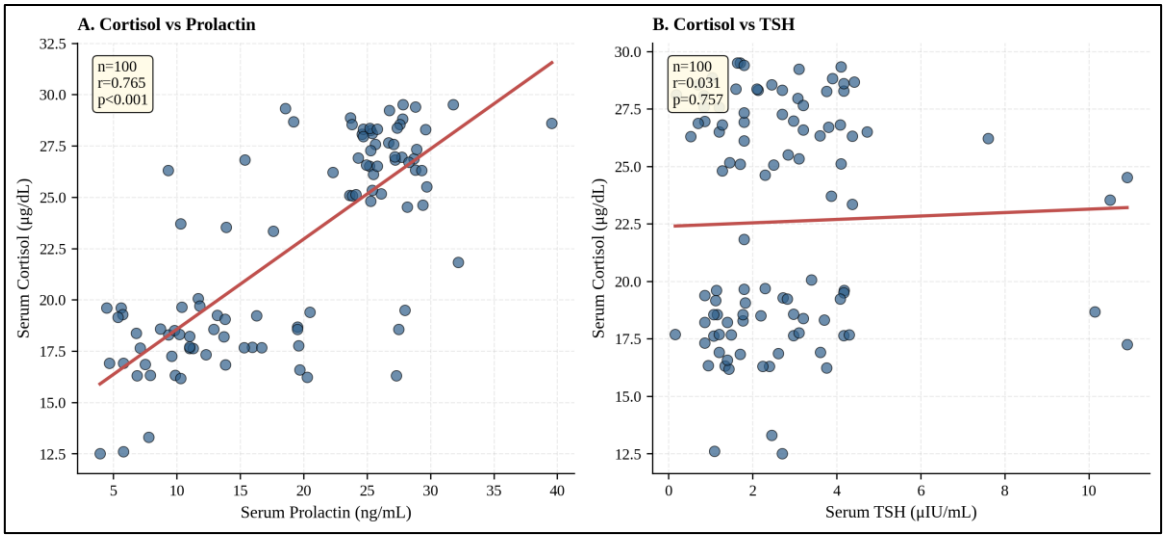


Figure 3: Scatter plots with fitted linear regression lines for serum cortisol against serum prolactin (Panel A) and serum TSH (Panel B) among women with infertility (primary and secondary groups combined, n=100). Cortisol was strongly and positively correlated with prolactin (r=0.765, p<0.001), whereas no significant correlation was observed between cortisol and TSH (r=0.031, p=0.757). Correlation coefficients correspond to the "Infertility combined" column of Table 6.

Post-hoc analysis showed that, compared with controls, the primary infertility group had significantly higher weight by 4.58 kg (p=0.017), BMI by 2.96 kg/m² (p<0.001), prolactin by 9.01 (p<0.001), and cortisol by 13.87 (p<0.001). The secondary infertility group also had significantly higher weight by 4.40 kg (p=0.023), BMI by 2.59 kg/m² (p=0.004), prolactin by 8.21 (p<0.001), TSH by 1.51 (p<0.001), and cortisol by 12.57 (p<0.001) compared with controls. Compared with primary infertility, the secondary infertility group had significantly higher TSH by 1.03 (p=0.009), while no significant differences were observed for weight, BMI, prolactin, FSH, LH, or cortisol (Table 5).

Cells show Pearson correlation coefficient and p value. "Infertility combined" includes primary and secondary infertility groups only.

Among infertile participants combined, serum cortisol showed a strong positive correlation with prolactin (r=0.765, p<0.001). Cortisol did not show a significant correlation with TSH within the combined infertility group, while prolactin and TSH showed only a weak overall correlation when all 150 participants were analyzed together (r=0.163, p=0.047) (Table 6).

DISCUSSION

The present comparative cross-sectional study observed distinct endocrine and metabolic differences among controls, women with primary infertility, and women with secondary infertility. Age was comparable across groups, which reduces age-related imbalance as an explanation for the hormonal differences. However, both infertility groups had significantly higher BMI than controls, with mean BMI of 27.99±4.17 kg/m² in primary infertility and 27.62±3.68 kg/m² in secondary infertility, compared with 25.04±3.91 kg/m² in controls. This finding is consistent with evidence that excess adiposity and insulin resistance may influence reproductive function through altered ovarian steroidogenesis, ovulatory dysfunction, and metabolic-endocrine disruption.[16]

Serum prolactin was significantly higher in both infertility groups compared with controls. Mean prolactin was 19.59±9.11 in primary infertility and 18.79±8.10 in secondary infertility, compared with 10.58±5.01 in controls. Post-hoc analysis confirmed significant differences between controls and both primary and secondary infertility groups, while the difference between the two infertility groups was not significant. This agrees closely with Bari et al, who reported higher prolactin levels among women with primary and secondary infertility compared with fertile controls in Bangladesh.[5] Similar findings were also reported by Sharma et al in India and Bhandari et al in Nepal, supporting the relevance of hyperprolactinaemic tendency across South Asian infertility populations.[7,8] Since elevated prolactin may suppress gonadotropin-releasing hormone pulsatility and alter ovulatory function, the present finding supports the clinical value of prolactin assessment in routine female infertility evaluation. The thyroid profile showed a more selective pattern. TSH differed significantly across groups, with the highest mean value in secondary infertility, 3.24±2.28, compared with 2.21±1.71 in primary infertility and 1.72±0.84 in controls. Post-hoc analysis showed that secondary infertility had significantly higher TSH than both controls and primary infertility, while the primary infertility-control difference was not significant. This is consistent with Ruman et al, who reported higher TSH among women with secondary infertility in Bangladesh.[17] Bari et al, and Chowdhury et al, also reported thyroid dysfunction more frequently among infertile or subfertile women than controls.[5,6] In the present study, euthyroid status remained the most frequent category in all groups, but hypothyroidism was more common in secondary infertility, 26.0%, than in primary infertility, 12.0%, and controls, 4.0%, whereas hyperthyroidism was uncommon and equally distributed, 2.0% in each group. The overall thyroid-status distribution differed significantly across groups (p=0.035), indicating that the excess of hypothyroidism in secondary infertility represents a categorical difference rather than a chance finding. T3 and T4 did not differ significantly across groups. This finding suggests that thyroid involvement in the present sample was mainly reflected through TSH variation rather than overt alteration in circulating thyroid hormone levels. This pattern is compatible with subclinical thyroid dysfunction, where TSH changes may precede measurable abnormalities in T3 or T4. The finding is also consistent with the predominance of euthyroid and hypothyroid categories in the current data. Cortisol was markedly higher in both infertility groups, with mean values of 23.24±4.98 in primary infertility and 21.95±4.85 in secondary infertility, compared with 9.38±2.94 in controls. The difference was significant for both infertility groups, while primary and secondary infertility did not differ significantly. Wdowiak et al similarly observed interactions between cortisol, prolactin, and other menstrual-cycle hormones among infertile women, suggesting that hypothalamic-pituitary-adrenal axis activity may interact with reproductive endocrine regulation.[13] The strong positive correlation between cortisol and prolactin among infertile women in the present study, r=0.765, p<0.001, further supports this endocrine interaction. However, Karunyam et al, in a systematic review, noted heterogeneity in cortisol findings across infertility studies.[18] Therefore, the present result supports an association between infertility and higher morning cortisol, but it should not be interpreted as proof that cortisol elevation is a universal or causal feature of infertility.

An important contrast with previous literature was observed in the prolactin-TSH relationship. Although Ramadras et al reported a moderate positive correlation between prolactin and TSH in infertile women, the present study showed no significant correlation within the combined infertility group.[10] This may reflect sample composition, exclusion criteria, the predominance of euthyroid participants, or the influence of cortisol and metabolic variables. Overall, the findings indicate that infertile women in this sample had higher prolactin,

cortisol, BMI, and selectively higher TSH, particularly in secondary infertility, while overt thyroid hormone differences were not observed.

Limitations

This was a single-center cross-sectional study with a modest sample size, so causal relationships between endocrine parameters and infertility could not be established. The control group consisted of healthy hospital visitors rather than community-based fertile controls, and thyroid-status classification was based on biochemical cutoffs rather than long-term clinical follow-up.

CONCLUSION

This study observed significantly higher serum prolactin and cortisol levels among women with primary and secondary infertility compared with controls. TSH was also significantly higher, particularly among women with secondary infertility, while T3 and T4 did not differ significantly across groups. The strong positive correlation between cortisol and prolactin among infertile women suggests a possible interaction between stress-related and reproductive endocrine pathways. These findings support the clinical relevance of combined endocrine assessment, including prolactin, thyroid profile, cortisol, and metabolic parameters, in the evaluation of female infertility.

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Ethical approval: The study was approved by the Institutional Ethics Committee

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