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Original article

Hyperprolactinemia in Women with Delayed Conception and Elevated Thyroid-Stimulating Hormone: A Retrospective Case-Control Study from Libya

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Abstract
Thyroid dysfunction and hyperprolactinemia are clinically relevant endocrine abnormalities in women presenting with impaired fertility. This study aimed to compare serum prolactin concentrations and the frequency of recorded hyperprolactinemia between women with delayed conception and previously documented elevated thyroid-stimulating hormone (TSH) and fertile controls with normal TSH, and to explore pooled associations of prolactin with TSH, age, and body mass index (BMI). This retrospective case-control study used medical and laboratory records from Nour Al Hayat Fertility Clinic, Libya, and included 100 women aged 20–40 years: 50 cases and 50 controls. TSH and prolactin were measured using an automated electrochemiluminescence immunoassay platform. Cases had higher mean TSH (7.31 ± 4.32 vs. 1.58 ± 1.24 mIU/L) and prolactin (38.96 ± 28.25 vs. 13.52 ± 5.04 ng/mL) than controls. Because elevated TSH was an eligibility criterion for cases and normal TSH was required for controls, the between-group TSH contrast was inherent to the selection criteria and should be interpreted descriptively rather than as an independent finding. Hyperprolactinemia, according to the study-defined laboratory threshold, was recorded in 30/50 cases (60.0%) and none of the controls ($P < 0.001$). In the pooled sample, prolactin correlated positively with TSH (rho = 0.631), age (rho = 0.537), and BMI (rho = 0.737; all $P < 0.001$). These pooled correlations are exploratory and may partly reflect between-group differences rather than independent within-group relationships. FT4 measurements were unavailable, and elevated prolactin was not confirmed by repeat testing. Therefore, the study could not distinguish overt from subclinical hypothyroidism or establish persistent pathological hyperprolactinemia. The findings describe hormonal patterns in a selected clinical cohort but do not establish causality or independent relationships between the hormones.
Keywords. Female Infertility; Prolactin; Hyperprolactinemia; Thyroid-Stimulating Hormone; Case-Control Study.

Introduction

Infertility is a common reproductive health condition with substantial personal, social, and public health implications. The World Health Organization (WHO) estimates that approximately one in six people experience infertility during their lifetime [1]. Infertility is commonly defined as the failure to achieve pregnancy after at least 12 months of regular, unprotected sexual intercourse [2]. Clinical evaluation is generally recommended after 12 months in women younger than 35 years and after 6 months in women aged 35 years or older, with earlier assessment when relevant reproductive risk factors are present [3].

Female reproductive dysfunction may arise from several causes, including ovulatory disorders and endocrine abnormalities, some of which may be amenable to treatment. Accordingly, the investigation of potentially reversible endocrine disturbances remains an important component of reproductive assessment. Thyroid function contributes to the regulation of female reproductive physiology, including menstrual cyclicity, ovarian function, and processes involved in conception and early pregnancy [4–6]. Thyroid-stimulating hormone (TSH), secreted by the anterior pituitary gland, is widely used to assess the thyroid axis. Nevertheless, an elevated TSH concentration alone does not establish whether hypothyroidism is overt or subclinical, as this distinction requires assessment of circulating free thyroxine (FT4) [5,6]. The clinical significance of modest TSH elevations in women attempting conception remains an area of ongoing investigation, and thyroid results should be interpreted according to appropriate laboratory reference ranges and the individual clinical context [5,15]. These considerations are particularly relevant when evaluating women with delayed conception and previously documented elevated TSH levels.

Prolactin (PRL) is a peptide hormone secreted predominantly by lactotroph cells of the anterior pituitary gland and is regulated primarily through tonic inhibition by hypothalamic dopamine. Although its principal physiological role is lactation, prolactin also influences reproductive function. Persistent hyperprolactinemia may suppress hypothalamic gonadotropin-releasing hormone signaling, disrupt gonadotropin secretion, and impair ovulatory function, potentially contributing to menstrual irregularities and reduced fertility [7–9]. However, an elevated serum prolactin concentration does not invariably indicate persistent pathological hyperprolactinemia. Pregnancy, physiological stress, certain medications, primary hypothyroidism, renal dysfunction, and macroprolactinemia are among the factors that may affect prolactin measurements and their clinical interpretation [8,9]. Therefore, prolactin results should be interpreted with consideration of relevant physiological and clinical influences.

A plausible physiological connection between thyroid function and prolactin secretion is mediated by hypothalamic thyrotropin-releasing hormone (TRH), which stimulates pituitary release of both TSH and prolactin. In primary hypothyroidism, reduced thyroid hormone availability may increase TRH secretion and consequently contribute to elevated circulating prolactin [4,8]. Increased prolactin may, in turn, interfere with the hypothalamic–pituitary–gonadal axis and reproductive function [7–9]. This mechanism provides a rationale for examining TSH and prolactin concurrently in

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women presenting with delayed conception. Nevertheless, because TSH elevation does not independently establish the biochemical severity or type of thyroid dysfunction, the coexistence of elevated TSH and prolactin should not be interpreted as definitive evidence of overt hypothyroidism or a causal relationship between the two hormones when FT4 measurements are unavailable.

Previous investigations have reported an association between serum TSH and prolactin, although the magnitude of this relationship varies across populations and study designs. A systematic review and meta-analysis of 11 studies involving infertile women reported a moderate positive pooled correlation between serum TSH and prolactin (r = 0.431, 95% confidence interval [CI]: 0.251–0.582), with substantial between-study heterogeneity (I^2 = 80%) [10]. In a study of 221 non-pregnant women, Sinha et al. also reported a positive correlation between TSH and prolactin (r = 0.239, P = 0.01) [11]. Conversely, Şahin and Önder found no statistically significant differences in TSH or prolactin concentrations between fertile and infertile women, whose hormone concentrations were generally within reference ranges [12]. These findings suggest that the observed relationship may vary according to participants' underlying endocrine status, selection criteria, and the distribution of hormone concentrations within the study population.

Evidence from Libya provides additional regional context. A study conducted in the Ubari region compared 50 infertile women with 30 fertile controls and reported higher mean TSH and prolactin concentrations in the infertile group, together with a positive correlation between the two hormones [13]. Although this study supports the relevance of investigating thyroid–prolactin relationships in the Libyan population, differences in recruitment settings and participant selection limit direct comparison with studies employing other eligibility criteria. Further examination of women with delayed conception who have specifically documented elevated TSH levels may therefore provide additional information on the distribution of prolactin concentrations in this clinical subgroup. Interpretation of such findings must, however, distinguish differences between predefined groups from evidence of an independent association between the two hormones.

Accordingly, the present study aimed to compare serum prolactin concentrations and the frequency of hyperprolactinemia between women with delayed conception and previously documented elevated TSH levels and fertile women with normal TSH levels attending a fertility clinic in Tripoli, Libya. A secondary objective was to explore the relationships of serum prolactin with TSH, age, and BMI.

Materials and Methods: Study design and setting

A retrospective case-control study was conducted at Nour Al Hayat Fertility Clinic, Libya. Medical records and laboratory reports were reviewed for a seven-month period from October 2025 through April 2026. The study as a case-control investigation in which women with delayed conception and elevated serum TSH constituted the case group, while healthy fertile women with normal TSH served as controls. Laboratory measurements had been completed as part of routine clinical care and were subsequently abstracted for analysis.

Study population and group definition

The final dataset comprised 100 women aged 20-40 years, including 50 cases and 50 controls. Cases were women with documented delayed conception, elevated serum TSH, irregular menstrual cycles, nulliparity, and complete TSH and prolactin results. Controls were healthy fertile women with normal TSH and a history of at least one successful pregnancy. Women with low TSH, known male-factor infertility, polycystic ovary syndrome, uterine or other reproductive-tract abnormalities, and incomplete medical or laboratory records were excluded.

Data collection

Data were extracted using a standardized collection form. Variables included age, body mass index (BMI), menstrual-cycle regularity, parity status, serum TSH concentration, serum prolactin concentration, and the duration of delayed conception (years).

Laboratory analysis and reference intervals

Serum TSH and prolactin values were obtained from laboratory records. Measurements were performed using the Cobas e411 automated immunoassay analyzer (Roche Diagnostics, Mannheim, Germany) based on electrochemiluminescence immunoassay (ECLIA) technology, following the manufacturer's recommended procedures and laboratory quality-control standards. The reference intervals were 0.35-4.5 mIU/L for TSH and 5.18-26.53 ng/mL for prolactin. Elevated TSH was defined as a value >4.5 mIU/L, and hyperprolactinemia as a prolactin concentration >26.53 ng/mL.

Statistical analysis

Statistical analyses were performed with SPSS version 26. Continuous variables were summarized as mean ± standard deviation (SD), and categorical variables as frequencies and percentages. Between-group differences in continuous variables were assessed using independent-samples t-tests, except for TSH, which was summarized descriptively because TSH status determined group assignment. Categorical variables were compared using chi-square tests. Mean differences for inferential comparisons are presented with two-sided 95% confidence intervals (CIs), and standardized between-group differences are expressed as Cohen's d. For categorical comparisons, Cramer's V was calculated where appropriate. The absolute between-group difference in hyperprolactinemia frequency is reported with a 95% CI.

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Associations between prolactin and TSH, age, and BMI were evaluated using Spearman's rank correlation. Approximate 95% CIs for Spearman's rho were estimated from the reported coefficients using Fisher's z transformation. Statistical significance was set at $P < 0.05$.

Ethical considerations

Ethical approval was reported as having been obtained from the Department of Medical Laboratory Sciences, Faculty of Medical Technology, University of Tripoli (Ethical Approval No. 17/2026). Participant confidentiality and anonymity were maintained, and personal identifiers were removed from records before statistical analysis.

Results

Participant characteristics

The study included 100 women, 50 in each group. As shown in (Table 1), the mean age was higher among cases than controls (32.26 ± 4.63 vs. 29.86 ± 4.34 years; mean difference 2.40 years; 95% CI: 0.62 to 4.18; P = 0.009; Cohen's d = 0.53, 95% CI: 0.13 to 0.93). However, the age-category distribution did not differ significantly between groups (chi-square = 5.342, P = 0.148). BMI was also higher among cases (29.77 ± 3.83 vs. 24.88 ± 2.16 kg/m^2; mean difference 4.89 kg/m^2; 95% CI: 3.65 to 6.13; P < 0.001; Cohen's d = 1.57, 95% CI: 1.11 to 2.01). BMI-category distributions differed significantly (chi-square = 39.455, P < 0.001): obesity was recorded in 25 cases (50.0%) and no controls, while normal BMI was recorded in 6 cases (12.0%) and 28 controls (56.0%).

Table 1. Demographic and clinical characteristics of study participants, with standardized between-group effect sizes.

Variable	Cases (n=50)	Controls (n=50)	P value	Effect size	95% CI
Age, years; mean ± SD	32.26 ± 4.63	29.86 ± 4.34	0.009	Cohen's d = 0.53	0.13 to 0.93
Age ≤24 years, n (%)	2 (4.0)	6 (12.0)	0.148*	Cramer's V = 0.23	—
Age 25–29 years, n (%)	13 (26.0)	19 (38.0)	—	—	—
Age 30–34 years, n (%)	19 (38.0)	16 (32.0)	—	—	—
Age ≥35 years, n (%)	16 (32.0)	9 (18.0)	—	—	—
BMI, kg/m^2; mean ± SD	29.77 ± 3.83	24.88 ± 2.16	<0.001	Cohen's d = 1.57	1.11 to 2.01
BMI: normal, n (%)	6 (12.0)	28 (56.0)	<0.001†	Cramer's V = 0.63	—
BMI: overweight, n (%)	19 (38.0)	22 (44.0)	—	—	—
BMI: obese, n (%)	25 (50.0)	0 (0.0)	—	—	—

Note: P values for age-category and BMI-category distributions are overall chi-square tests. Cramer's V is reported for categorical effect sizes.

By the predefined group criteria, all 50 cases had elevated TSH (>4.5 mIU/L), whereas all 50 controls had TSH within the reported reference interval. This separation reflects the study's selection criteria and should not be interpreted as an independent association. Mean TSH and prolactin concentrations and prolactin categories are summarized in (Table 2).
Mean TSH was 7.31 ± 4.32 mIU/L in cases and 1.58 ± 1.24 mIU/L in controls; this contrast was expected because TSH status defined group assignment. Mean prolactin was higher in cases than controls (38.96 ± 28.25 vs. 13.52 ± 5.04 ng/mL; P < 0.001; Cohen's d = 1.25, 95% CI: 0.82 to 1.67). Prolactin-category distribution differed significantly (chi-square = 42.886, P < 0.001). Hyperprolactinemia was recorded in 30 cases (60.0%) and no controls; normal prolactin in 19 cases (38.0%) and 48 controls (96.0%); and low prolactin in 1 case (2.0%) and 2 controls (4.0%). The absolute
difference in hyperprolactinemia frequency was 60.0 percentage points (95% CI: 44.4 to 72.4). (Figure 1) illustrates the difference in mean prolactin.

Table 2. Hormonal findings and selected between-group comparisons

Measure	Cases (n=50)	Controls (n=50)	Difference (95% CI)	Effect size (CI where available)	P value
TSH, mIU/L	7.31 ± 4.32	1.58 ± 1.24	—	—	—
Prolactin, ng/mL	38.96 ± 28.25	13.52 ± 5.04	25.44 (17.30 to 33.58)	Cohen's d = 1.25 (0.82 to 1.67)	<0.001
Hyperprolactinemia, n (%)	30 (60.0)	0 (0.0)	60.0 percentage points (44.4 to 72.4)	Cramer's V = 0.65	<0.001*
Normal prolactin, n (%)	19 (38.0)	48 (96.0)	—	—	—
Low prolactin, n (%)	1 (2.0)	2 (4.0)	—	—	—

** The P value shown for hyperprolactinemia refers to the overall three-category prolactin classification comparison. Mean differences are calculated as cases minus controls*

Note: TSH was an eligibility criterion for group assignment; therefore, TSH values are presented descriptively, and no between-group inferential statistics are reported for this measure. —, not reported.

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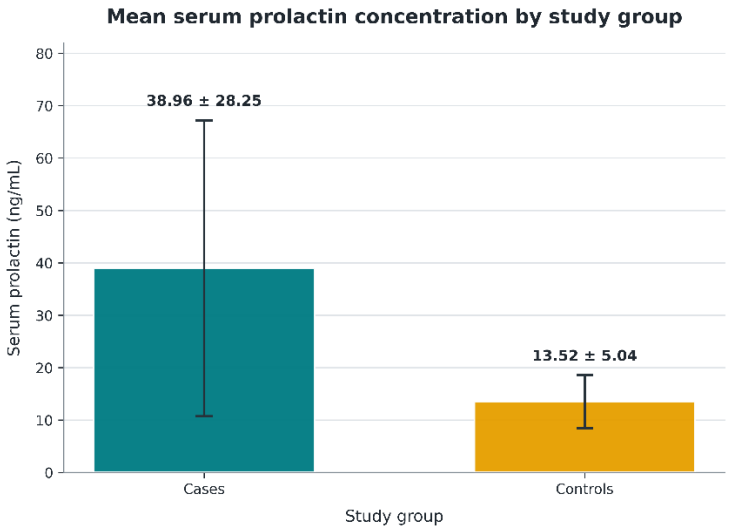


Figure 1. Mean serum prolactin concentration in cases and controls. Bars represent mean values; error bars indicate standard deviation. The figure visually summarizes the between-group difference reported in (Table 2).

Correlation analyses: In the pooled study population (n = 100), prolactin showed positive correlations with TSH (Spearman's rho = 0.631, approximate 95% CI: 0.496 to 0.736), age (rho = 0.537, approximate 95% CI: 0.381 to 0.663), and BMI (rho = 0.737, approximate 95% CI: 0.632 to 0.815); all P < 0.001. As shown in (Table 3), the strongest correlation was with BMI, followed by TSH and age. These pooled associations are exploratory: the cases and controls were selected according to TSH status and differed in age and BMI, so the correlations may partly reflect between-group differences rather than independent within-group relationships.

Table 3. Exploratory Spearman correlations with serum prolactin in the pooled study sample (n = 100).

Variable correlated with prolactin	Spearman rho	Approximate 95% CI	P value	Interpretation
TSH	0.631	0.496 to 0.736	<0.001	Positive association; exploratory
Age	0.537	0.381 to 0.663	<0.001	Positive association; exploratory
BMI	0.737	0.632 to 0.815	<0.001	Positive association; exploratory

(Figure 2) provides a visual exploration of the pooled TSH–prolactin association. The scatter plot complements the coefficient reported in (Table 3) but does not establish an independent or causal relationship.

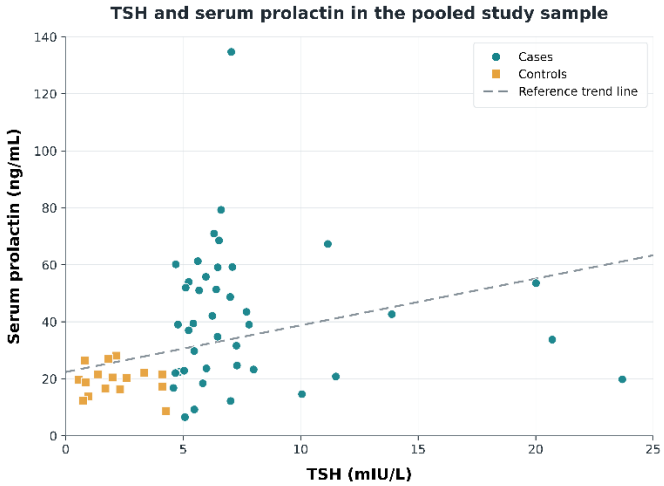


Figure 2. Exploratory scatter plot showing the reported positive relationship between serum TSH and prolactin across the pooled study population. The association is descriptive and should be interpreted cautiously because elevated TSH defined the case group

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Discussion

The principal finding of this study was the high frequency of elevated serum prolactin among women with delayed conception and previously documented elevated thyroid-stimulating hormone (TSH). Hyperprolactinemia, defined according to the laboratory reference threshold used in this study, was observed in 30 of 50 cases (60.0%), compared with none of the 50 fertile controls. Furthermore, mean serum prolactin was substantially higher among cases than controls (38.96 ± 28.25 vs. 13.52 ± 5.04 ng/mL; $p < 0.001$), with a large standardized between-group difference (Cohen's d = 1.25; 95% CI: 0.82–1.67). These findings indicate that elevated prolactin concentrations frequently coexisted with elevated TSH among the selected cases. Nevertheless, the retrospective case–control design does not establish that hyperprolactinemia caused delayed conception or that the observed hormonal abnormalities were independently responsible for reproductive dysfunction.

The observed findings are biologically plausible in light of the interaction between thyroid function and prolactin regulation. Hypothalamic thyrotropin-releasing hormone (TRH) stimulates the secretion of both TSH and prolactin by the anterior pituitary gland. In primary hypothyroidism, reduced thyroid hormone availability may increase TRH secretion and consequently contribute to elevated circulating prolactin. Primary hypothyroidism is recognized as a potential secondary cause of hyperprolactinemia [4,8]. Elevated prolactin may subsequently interfere with hypothalamic gonadotropin-releasing hormone signaling, impair gonadotropin secretion, and disrupt ovulatory function, potentially contributing to menstrual irregularities and reduced fertility [7–9]. However, FT4 was not measured, and the available data did not establish a formal diagnosis of overt or subclinical hypothyroidism. Elevated TSH alone therefore cannot confirm the type or severity of thyroid dysfunction in these participants.

The prevalence of hyperprolactinemia and the difference in mean prolactin concentrations observed in the present study are broadly consistent with previous investigations reporting higher prolactin concentrations among women with infertility or thyroid dysfunction. Saxena et al. reported a relationship between serum TSH and prolactin in women investigated for infertility [14]. Similarly, Sinha et al. reported a positive correlation between TSH and prolactin among 221 non-pregnant women, although their reported correlation was weaker than that observed in the present pooled sample ($r = 0.239$, $P = 0.01$) [11]. These findings support further investigation of the coexistence of thyroid-axis disturbance and elevated prolactin in women presenting with reproductive dysfunction.

The present findings also need to be considered in relation to the systematic review and meta-analysis by Ramadras et al., which included 11 studies involving infertile women and reported a moderate positive pooled correlation between serum TSH and prolactin ($r = 0.431$, 95% CI: 0.251–0.582) [10]. The correlation observed in the current dataset was numerically stronger (Spearman's rho = 0.631, approximate 95% CI: 0.496–0.736; Table 3). However, this difference should not be interpreted as evidence of a stronger underlying physiological relationship. The studies differed in their populations and selection criteria, and the present analysis included predefined groups with distinctly different TSH distributions. Therefore, the estimates are not directly interchangeable, and the design of the present study provides an important alternative explanation for the stronger observed correlation.

The case–control selection strategy imposes an important limitation on the interpretation of the hormonal findings. All cases were selected on the basis of previously documented elevated TSH, whereas controls were required to have TSH concentrations within the normal reference range. Consequently, the observed between-group difference in TSH was largely predetermined by the eligibility criteria and should be regarded as a descriptive feature of the selected groups rather than an independent study finding. Furthermore, because the two groups were deliberately selected to have different TSH distributions, the positive correlation between TSH and prolactin in the pooled sample may have been substantially influenced by this between-group separation. Combining groups with higher TSH and prolactin concentrations among cases and lower concentrations among controls can produce or magnify a positive pooled correlation, even when the within-group relationship is weak or absent. Accordingly, the observed Spearman correlation coefficient (rho = 0.631) should be interpreted strictly as an exploratory association within this selected sample, not as evidence of an independent relationship or a causal effect of TSH on prolactin secretion. Separate within-group analyses and appropriately adjusted statistical models would be necessary to investigate these relationships more rigorously.

Differences in demographic and clinical characteristics provide further context for interpreting the hormonal findings. As summarized in (Table 1), cases were older than controls (32.26 ± 4.63 vs. 29.86 ± 4.34 years; $p = 0.009$) and had a higher mean body mass index (BMI; 29.77 ± 3.83 vs. 24.88 ± 2.16 kg/m^2; $p < 0.001$). Obesity was present in 25 cases (50.0%), whereas no control was classified as obese. These differences indicate that the groups were not fully comparable beyond their predefined thyroid status. Prolactin was positively correlated with age (rho = 0.537, $p < 0.001$) and BMI (rho = 0.737, $p < 0.001$). Although these associations may indicate that prolactin variation occurred alongside differences in demographic and metabolic characteristics, they do not establish independent relationships. In the absence of multivariable adjustment, the respective contributions of TSH, age, BMI, and other potentially relevant factors cannot be separated.

The findings also differ from those of Şahin and Önder, who reported no statistically significant differences in TSH or prolactin concentrations between fertile and infertile women, with hormone concentrations generally within reference ranges [12]. This apparent discrepancy may reflect differences in study populations, eligibility criteria, endocrine phenotypes, and hormone distributions. Unlike studies enrolling women with infertility irrespective of thyroid status, the present study specifically selected cases with previously documented elevated TSH. The resulting group therefore

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represents a selected endocrine phenotype rather than the full spectrum of women experiencing delayed conception. Accordingly, the current findings should not be generalized to all infertile women or interpreted as evidence that elevated TSH and hyperprolactinemia are universal features of infertility.

The observed frequency of elevated prolactin also has implications for laboratory interpretation. Consensus recommendations emphasize that an increased prolactin result should be interpreted in its clinical context and that secondary causes, including medication exposure, pregnancy, primary hypothyroidism, renal dysfunction, and other relevant conditions, should be considered. Repeat measurement may be appropriate when a modest elevation could reflect transient physiological or sampling-related influences, and macroprolactinemia may complicate interpretation [8,9].

From a clinical perspective, the findings support considering prolactin assessment when women with delayed conception present with menstrual irregularity and evidence of thyroid-axis disturbance. Identifying a potentially reversible endocrine abnormality may contribute to a comprehensive reproductive evaluation. However, the results do not justify attributing infertility to a single hormonal measurement. Current infertility guidance emphasizes assessment of ovulatory function, reproductive anatomy, and male-factor infertility as appropriate to the clinical presentation [3]. Similarly, elevated TSH requires interpretation alongside FT4 and the broader clinical context, particularly when the elevation is modest, and its reproductive significance is uncertain [5]. The present findings should therefore be viewed as supporting further investigation of coexisting thyroid-axis abnormalities and elevated prolactin, rather than as establishing a diagnostic or therapeutic pathway.

Several features strengthen the descriptive value of this study. First, the use of the same laboratory platform for TSH and prolactin measurements reduces potential variability attributable to differences between assay platforms. Second, the study assessed both continuous hormone concentrations and categorical prolactin status, providing complementary perspectives on the between-group differences. Third, the inclusion of age, BMI, and the recorded duration of delayed conception enabled the exploration of potentially relevant clinical and demographic characteristics. Finally, the study contributes data from a fertility-care setting in Tripoli, Libya, adding to the regional evidence concerning the coexistence of elevated TSH and prolactin in women presenting with delayed conception.

Study limitations

Several limitations should be considered when interpreting the findings of this study. First, FT4 concentrations were not available in the reviewed laboratory records. Consequently, although elevated TSH was documented among cases, it was not possible to distinguish overt from subclinical hypothyroidism or to establish the biochemical severity of thyroid dysfunction. Second, prolactin concentrations were based on a single measurement extracted from routine laboratory records, without documented repeat testing under standardized sampling conditions. Therefore, the recorded elevations cannot be considered definitive evidence of persistent pathological hyperprolactinemia. Furthermore, information regarding sampling conditions, medication exposure, pregnancy testing, and macroprolactin assessment was not consistently available, limiting the ability to exclude all potential physiological and secondary causes of elevated prolactin. Finally, the predefined selection of cases and controls according to TSH status, together with differences in age and BMI between the groups, may have influenced the pooled correlations. In the absence of within-group analyses and multivariable adjustment, the independent contributions of these variables cannot be determined. These limitations support interpreting the results as descriptive and exploratory rather than as evidence of independent hormonal or causal relationships.

Conclusion

Women with delayed conception and elevated TSH in this Libyan cohort had substantially higher serum prolactin concentrations than fertile women with normal TSH, and 60% had prolactin concentrations above the study-defined laboratory threshold for hyperprolactinemia. In the pooled sample, prolactin was positively correlated with TSH, age, and BMI. However, these associations should be interpreted as exploratory, particularly because case and control selection was based on TSH status and the groups differed in age and BMI. The findings describe the coexistence of elevated prolactin and TSH in this selected clinical population but do not establish causality or an independent relationship between the hormones. Although prolactin assessment may be considered as part of a comprehensive fertility evaluation, the findings should not be generalized to all women with delayed conception. Future prospective studies with larger samples, direct assessment of FT4 and thyroid autoimmunity, standardized prolactin sampling and repeat testing, medication review, and multivariable analysis are needed to clarify the relationships between thyroid function, prolactin concentrations, and reproductive outcomes.

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Conflict of interest

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Data availability

The datasets generated and/or analyzed during the current study are available from the corresponding author on reasonable request, subject to appropriate ethical and confidentiality considerations.

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Author contributions

Dr. Eida Elmansorry was responsible for the study conception and design, methodology, and development of the research plan. Data collection was performed by all authors. Dr. Elmansorry conducted the data analysis, drafted the manuscript, and critically revised all sections of the paper. All authors read and approved the final version of the manuscript.

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