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The Level of Thyroid Stimulating Hormone, Thyroxine, Free Thyroxine, Triiodothyronine, Free Triiodothyronine, and Prolactin among Breastfeeding Women from Baghdad city: A Case–Control Study

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ABSTRACT:

Background: The postpartum period involves a multitude of adaptations, both endocrine and metabolic, allowing the mother to lactate successfully while recovering from parturition. Changes in the action of thyroid hormones during lactation might have a substantial effect on physiology for mothers as well as on lactational performance. Despite this growing awareness of postpartum thyroid disease, the relationship between lactation and thyroid hormone homeostasis has not yet been fully elucidated.

Objective: To examine serum levels of thyroid stimulating hormone (TSH), total thyroxine (T4), free thyroxine (FT4), total triiodothyronine(T3), free triiodothyronine (FT3) and prolactin in breastfeeding women compared to healthy controls.

Methods: In this case–control study, we conducted a survey in Baghdad, Iraq between February 2025 and May 2026. Fifty breastfeeding women aged 25–43 years were recruited along with forty healthy females matched for age and body mass index to act as controls. Exclusion criteria included history of diabetes mellitus (28) or renal disorder. Fasting venous blood samples were taken. Hormonal analyses were done using the vendor-specified Enzyme-linked immunosorbent assay (ELISA) methods. Statistical analyses used were Student's t-test and Pearson correlation analysis.

Results: Serum TSH and prolactin levels in breastfeeding women were significantly higher than those of controls ($p < 0.01$). In contrast, the lactating participants showed significant reductions of total and free thyroid hormone concentrations. Positive correlations were seen between prolactin and TSH concentrations, while inverse correlations were found with the parameters of prolactin with T3/T4 using correlation analysis.

Conclusion: the assessment of thyroid function during lactation is very important to optimize maternal healthcare and breastfeeding outcomes.

1. INTRODUCTION

Lactation represents among the most complex physiological events and is regulated through an orchestrated interaction of endocrine, metabolic, and neurohormonal systems. The maintenance of successful breastfeeding thus reflects an interplay of prolactin and oxytocin, as well as glucocorticoids, insulin and thyroid hormones to achieve mammary gland differentiation, lactogenesis and metabolism-homeostasis in the mother. Thyroid hormones in particular represent an important class of

endocrine mediators due to their widespread influence on cellular metabolism, protein synthesis, thermogenesis and neuroendocrine regulation [1].

The main Circulating thyroid hormones modulate oxygen consumption, mitochondrial activity, lipid metabolism and glucose utilization in virtually all tissues. hormones produced by the thyroid gland are thyroxine (T₄) and triiodothyronine (T₃), which are primarily regulated through hypothalamic secretion of thyrotropin-releasing hormone (TRH) along with pituitary release of thyroid stimulating hormone (TSH). Thyroid hormones also affect ovulatory function, implantation, placental development and fetal neurodevelopment as well as lactational physiology during reproductive life [2]

Pregnancy causes considerable changes in the hypothalamic–pituitary–thyroid axis. In addition, excess estrogen levels trigger the liver to produce thyroxine-binding globulin (TBG) that leads to high levels of total T₃ and T₄. Additionally, the chain has structural similarities with TSH and as such hCG also possesses thyrotropic activity, often resulting in transient suppression of circulating TSH concentrations during the early stages of pregnancy. These events guarantee sufficient maternal thyroid hormone production to meet both fetal developmental demands and maternal metabolic needs.[2]

Endocrine homeostasis is dramatically re-calibrated after parturition as maternal physiology transitions from pregnancy to lactation. During lactation, prolactin release is stimulated by suckling (by infant) and promotes milk production within mammary epithelial cells. They are required for maintenance of mammary gland responsiveness to prolactin and support the heightened metabolic demands of lactation. Thus, thyroid dysfunctions in the early puerperium might negatively affect milk supply, breastfeeding time and maternal health.[3]

Post partum thyroid dysfunctions is one of the most common endocrine disorders that can occur after pregnancy. Postpartum thyroiditis is a common condition, with an estimated worldwide prevalence of 5–10% in women, although this rate varies according to geographic region, iodine status and autoimmune susceptibility. This disorder usually presents as transient thyrotoxicosis then hypothyroidism, however, isolated hypothyroid or hyperthyroid presentations exist. Clinical manifestations consist of fatigue, depression, cold intolerance, impaired concentration, constipation and generalised weakness; symptoms which often overlap with the normal postpartum experience and thus go underdiagnosed.[4]

The association of breastfeeding with altered thyroid hormone metabolism has been linked to several proposed mechanisms. At the same time, lactation raises maternal iodine status in that significant amounts of iodine are excreted into breast milk to meet the demand for thyroid hormone production and neurological maturation. Deficiency of iodine when lactating can thus put lactating mothers at risk of relative hypothyroxinemia. In addition, the immunological rebound which follows on from pregnancy-associated immune suppression may provoke postpartum thyroid inflammation as an autoimmune response.[5]

The connection between prolactin and thyroid hormones is physiologically also important. Increased TRH secretion promote the release of both TSH and prolactin from the anterior pituitary. Thus, thyroid dysfunction may modify prolactin dynamics and in turn the mechanism of lactation. Hypothyroidism is associated with delayed lactogenesis, low milk supply, menstrual abnormalities and infertility. On the other hand, hyperthyroidism might affect maternal nutritional status and metabolic stability so that breastfeeding may not be successful. Although the importance of postpartum endocrine disturbances is gaining recognition, there are limited regional studies on thyroid hormone profiles in breastfeeding women populations in Iraq. Familiarizing with the biochemical changes occurring during lactation can help us to diagnose postpartum thyroid dysfunction early and fashion maternal healthcare gaining better insights. Thus, the objective of this study was to investigate serum concentrations of TSH, T₄, FT₄, T₃ and FT₃ as well as prolactin among lactating women in comparison with healthy controls who were not breastfeeding.[6]

Objective: To examine serum levels of thyroid stimulating hormone (TSH), total thyroxine (T₄), free thyroxine (FT₄), total triiodothyronine (T₃), free triiodothyronine (FT₃) and prolactin in breastfeeding women compared to healthy controls.

2. MATERIALS AND METHODS

Materials and Methods

Study Design

Methods: This study was a recorded case–control study in Baghdad, Iraq from February 2025 to May 2026. The study protocol complied with ethical principles for biomedical research involving human subjects.

Study Population

The study involved fifty breastfeeding women aged between 25 and 43 years. As controls, forty either healthy woman matched for age and body mass index (BMI) were recruited. Participants were recruited from outpatient health care facility and community health center. Women were excluded if they had diabetes mellitus, dubbed chronic renal disease, hepatic disorders, autoimmune conditions or pollen endocrine abnormality other than thyroid dysfunction since the time of diagnosis. To minimize confounding variables, we excluded individuals receiving hormonal medications, corticosteroids and pharmacotherapy for thyroid disorders.

Blood Sample Collection

Ten-hour fasting blood samples (approximately 5 ml) were drawn aseptically from a forearm vein for each subject between the hours of 8 AM and 9AM. Blood samples were subsequently aliquoted into plain non-heparinized tubes and allowed to clot at room temperature. Then, the samples were blended at 4000 rpm for 5 minutes to provide serum. In the case of serum, the separated solution was compiled in sterile Eppendorf tubes and stored at -20°C until biochemical analysis.

Hormonal and Biochemical Analysis

Serum hormones of TSH, total T4, FT4, total T3, FT3 and prolactin were measured with enzyme-linked immunosorbent assay (ELISA) kits from United States Biological Company. You also have serum urea and creatinine concentrations along with to the exclusion of renal dysfunction.

Statistical Analysis

Statistical analysis: SPSS 26. Data were represented as mean $\pm$ SD. Differences in study groups were assessed with a Student's independent t-test on the data sets. Pearson correlation coefficients were also calculated to measure relationships between prolactin concentrations and thyroid hormone parameters. Statistical significance was defined at $p < 0.05$.

3. RESULTS AND DISCUSSION

Results

Table 1. Comparison of Hormonal Parameters Between Groups

Parameter	Control	Breastfeeding Women	p-value
TSH ($\mu\text{IU/mL}$)	3.1 ± 1.36	5.1 ± 1.2	<0.01
FT4 (ng/dL)	1.5 ± 0.99	0.5 ± 0.02	<0.01
Total T4 ($\mu\text{g/dL}$)	10.20 ± 2.51	3.6 ± 1.7	<0.01
Total T3 (ng/dL)	100 ± 23.25	50 ± 20.36	<0.01
FT3 (pg/mL)	2.99 ± 1.35	1.3 ± 0.15	<0.01
Prolactin (ng/mL)	26.3 ± 1.8	350 ± 23.84	<0.01

Table 2. Correlation Between Prolactin and Thyroid Hormone Parameters

Parameter	Correlation Coefficient (r)
TSH	0.360
FT4	-0.535
Total T4	-0.501
Total T3	-0.301
FT3	0.325

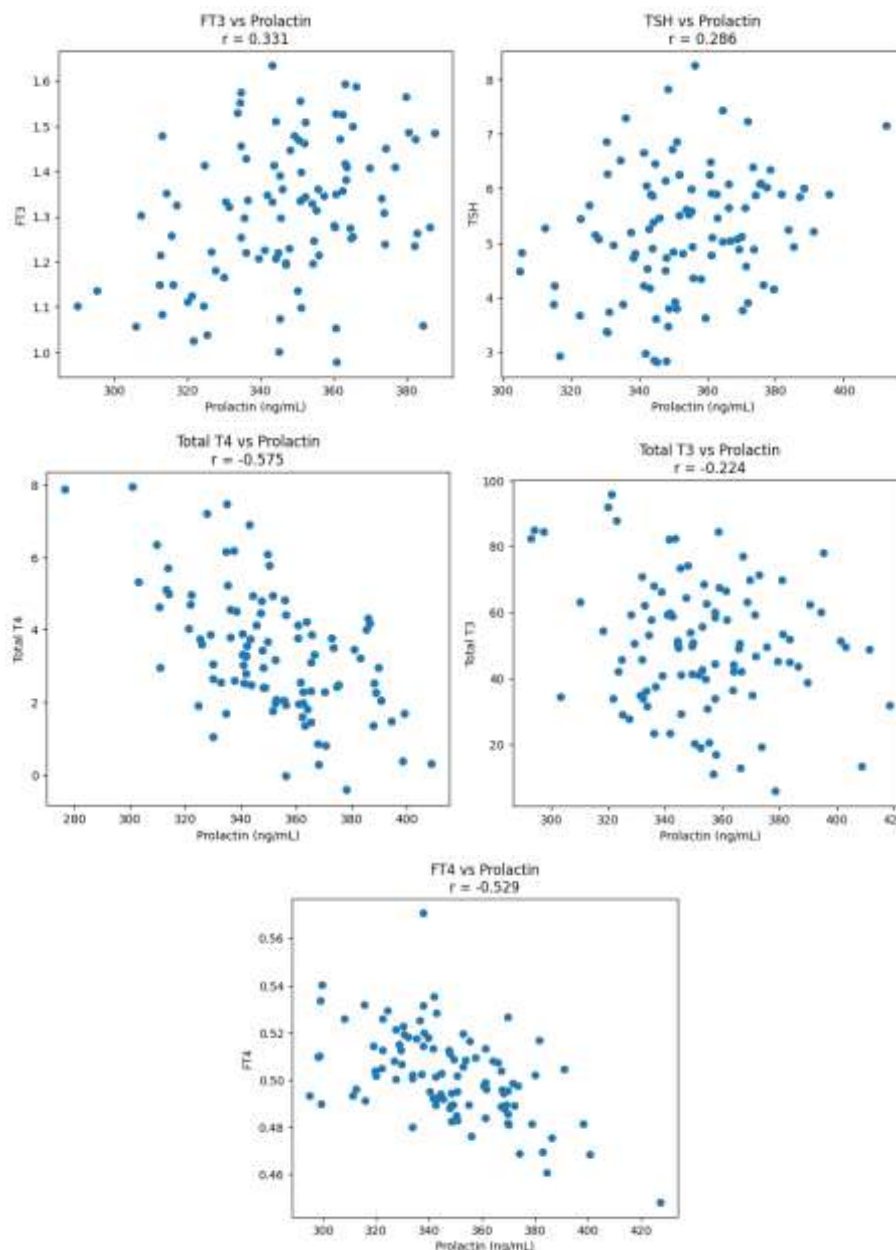


Figure 1 Correlation analysis of serum prolactin levels with different thyroid hormone parameters in breastfeeding women

DISCUSSION

The postpartum period is marked by massive changes in maternal endocrine patterns, important to sustain lactation and to return the maternal physiology back toward homeostasis. The current study discovered marked changes in thyroid hormone metabolism in lactating women as evidenced by increased TSH and prolactin levels, linked with low serum T3 and T4 concentrations. These results highlight the close physiological connection between lactation and thyroid gland activity. The significantly increased circulating concentration of prolactin in breastfeeding women aligns with the known physiological role of this peptide hormone during lactogenesis. Suckling induces hypothalamic suppression of dopaminergic secretion that promotes the release of prolactin by anterior pituitary lactotrophs. [7] Persistently elevated levels of PRL are necessary to maintain lactation by sustaining mammary epithelial differentiation and continued milk synthesis. The large rise in prolactin concentrations seen in the current study thus represents an expected endocrine adjustment to active nursing.[8]

Increased TSH concentrations in lactating women suggest compensatory stimulation of the thyroid gland due to decreased peripheral availability of thyroid hormone. Thyroid hormones have a major role in energy metabolism, mitochondrial respiration and protein secretion in mammary tissue. The increased metabolic demand of lactation can accelerate the utilization of thyroid hormone and stimulate the pituitary to secrete more TSH. These results are in accordance with prior studies reporting higher concentrations of TSH postpartum. Multiple physiological mechanisms could be proposed to explain this entity.[9] Maternal requirements for iodine during lactation are markedly increased because iodine is actively secreted into breast milk to support neonatal thyroid hormone synthesis. Thus, the depletion of iodine in pregnant women can not only hinder thyroid function but also cause compensatory mechanisms to increase TSH secretion.[10]

The largest decreases in T3, FT3, T4 and FT4 concentrations (85%±83%, 38% and Tmax percentage reductions) values amongst breastfeeding women also provide further support for the hypothesis of modulation of thyroid hormone metabolism during lactation. Lower circulating concentrations of thyroid hormone may indicate increased peripheral use, iodine deficiency or postpartum autoimmune thyroiditis.[11] Such endocrine patterns have been shown among women with postpartum thyroid dysfunction, especially in the hypothyroid phases of postpartum thyroiditis. Pregnancy instigates temporary immune tolerance to avert maternal immunological expropriation of foetal tissues.[12] After delivery restoration of immune tolerance may incite auto reactivity to thyroid tissue in susceptible women. This immune rebound phenomenon is thought to significantly play a role in postpartum thyroiditis, and might at least partly contribute to the hormonal changes found in the present study.[13]

The inverse correlations between prolactin and thyroid hormone concentrations are physiologically significant. Increased metabolism of the thyroid hormones in some tissues and during lactation at peak milk production may potentially use higher amounts of prolactin. Furthermore, hypoprolactinemia through activation of TRH hypothalamic resistance can also occur due to reduced availability of thyroid hormone.[14] TRH provokes secretion of anterior pituitary TSH and prolactin; thus, there is functional interrelationship between thyroid status and lactational physiology.[15]

Clinical Implications of Hypothyroidism upon Lactation. Maternal hypothyroidism may impair lactation mainly through reduced responsiveness to prolactin and metabolic activity of the mammary gland. [16] Untreated hypothyroidism is associated with delayed lactogenesis, low milk production, fatigue and depression and possible adverse effects on the well-being of the mother. It is therefore important to detect thyroid dysfunction as soon as possible during the postpartum period in order to ensure successful breastfeeding.[17]

This recent study provides relevant regional information on endocrine alterations related to breast feeding in Iraqi women. However, a few limitations should be highlighted. Findings may not be generalizable, due to the relatively small sample size. Furthermore, you were not investigated for iodine status, thyroglobulin autoantibodies, duration of breastfeeding and postpartum intervals that could affect the interpretation of hormonal changes. Future longitudinal studies with auto-immune marker testing and nutrition assessment are advocated.[18]

Notwithstanding those limitations, the study highlights the significance of evaluating thyroid function during lactation.[19] Routine endocrine screening of postpartum women, especially those presenting with fatigue and/or depression and/or breastfeeding difficulties may lead to early thyroid diagnosis and management.[20]

CONCLUSION

Adequate availability of thyroid hormone has been suggested to be crucial for normal lactational physiology and maternal metabolic stability. Postpartum thyroid function monitoring may help to screen for endocrine disorders resulting in improved maternal-infant outcomes.

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AUTHOR CONTRIBUTIONS

ammal esmaeel ibrahim: Conceptualization, Investigation.Methodology, Laboratory Analysis.

Data Curation, Formal Analysis.Supervision, Writing—Review & Editing.

CONFLICT OF INTEREST

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

ETHICS STATEMENT

the study approval by al-nahrain/ college of pharmacy with number (nah.co.pha.12)

DATA AVAILABILITY STATEMENT

Data supporting the findings of this study are available from the corresponding author upon reasonable request.

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