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Thyroid Function and Renal Biomarker Correlation in Euthyroid and Hypothyroid Pregnant Women: A Comparative Analysis

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ABSTRACT

Introduction: Thyroid dysfunction, particularly hypothyroidism, is a prevalent endocrine disorder during pregnancy that significantly affects maternal and fetal health. Thyroid hormones also influence renal physiology, making the assessment of renal markers crucial in hypothyroid pregnancies.

Objectives: To estimate serum creatinine levels in euthyroid and hypothyroid pregnant women and assess their correlation with thyroid-stimulating hormone (TSH).

Methods: A cross-sectional comparative study was conducted among 200 pregnant women, comprising 100 newly diagnosed hypothyroid (cases) and 100 euthyroid (controls), aged 20–40 years. TSH was measured using electro-chemiluminescence (Cobas e411), and serum creatinine was assessed by the Modified Jaffe's method. Statistical analysis was done using SPSS v20.0.

Results: Mean serum TSH was significantly lower in hypothyroid cases (1.32 ± 0.89 µIU/ml) compared to controls (2.28 ± 0.91 µIU/ml, p = 0.002). Mean serum creatinine was significantly higher in cases (0.87 ± 0.41 mg/dl) versus controls (0.55 ± 0.17 mg/dl, p = 0.001). A significant correlation was found between TSH and serum creatinine levels in hypothyroid patients.

Conclusion: Hypothyroidism in pregnancy is associated with elevated serum creatinine, indicating renal function impairment. Regular monitoring of thyroid and renal parameters is essential for optimal maternal and fetal outcomes

Keywords: TSH (Thyroid Stimulating Hormone); Serum Creatinine; Hypothyroidism; Euthyroid; Pregnancy; Renal Function; Electro-chemiluminescence.

1. INTRODUCTION

Thyroid disorders are among the most common endocrine abnormalities in women of reproductive age, second only to diabetes mellitus during pregnancy [1]. Hypothyroidism, characterized by deficient production of thyroid hormones, can significantly influence the physiological adaptations of pregnancy. These hormonal alterations lead to systemic effects, including changes in renal function, metabolism, and cardiovascular dynamics. The demand for thyroid hormone rises during pregnancy, and failure to meet this demand may compromise both maternal health and fetal development [2,3].

Pregnancy is marked by dramatic changes in thyroid physiology. There is a 50% increase in thyroxine-binding globulin due

to elevated estrogen levels, enhanced renal iodine clearance, and increased demand for thyroid hormone synthesis to support maternal and fetal needs [4]. In iodine-sufficient areas, the thyroid gland enlarges by approximately 10%, while in iodine-deficient regions, the increase can range from 20–40% [5]. These changes, if not countered by physiological adaptations, may precipitate thyroid dysfunction, most commonly hypothyroidism. It is estimated that the global prevalence of hypothyroidism during pregnancy ranges from 2% to 15%, depending on the population and iodine status [6].

Renal function undergoes profound alterations during pregnancy to accommodate the increased metabolic load. Plasma volume expansion, increased cardiac output, and vasodilatation contribute to enhanced renal plasma flow and glomerular filtration rate (GFR), resulting in reduced serum concentrations of creatinine, urea, and uric acid in healthy pregnancies [7]. Structural adaptations such as dilation of the renal pelvis and calyces—often termed gestational hydronephrosis—are also observed, particularly in the third trimester. These changes are influenced by mechanical compression from the enlarging uterus and the relaxant effect of progesterone on smooth muscle [8].

Creatinine, a waste product of muscle metabolism, is primarily eliminated by the kidneys. Its concentration in serum is inversely related to GFR and serves as a reliable marker of renal function. During normal pregnancy, serum creatinine values are expected to be lower than in the non-pregnant state due to enhanced clearance [9]. However, in hypothyroid pregnant women, serum creatinine levels may rise owing to decreased renal perfusion, reduced GFR, and impaired tubular function—all consequences of altered thyroid hormone status [10]. Studies suggest that even subclinical hypothyroidism may be associated with reversible elevations in serum creatinine levels, emphasizing the importance of early diagnosis and monitoring [11].

Thyroid hormones are essential for normal renal development and function. They influence renal growth, GFR, sodium reabsorption, and expression of various transport proteins. Conversely, kidney diseases may affect thyroid hormone metabolism and function. Uremia can impair the hypothalamic-pituitary-thyroid axis and alter peripheral conversion of T4 to T3, resulting in “low T3 syndrome” or non-thyroidal illness syndrome [12]. This complex interrelationship between the thyroid and kidney justifies the concurrent evaluation of both organs, particularly in states of hormonal imbalance like hypothyroidism.

Given these physiological interconnections, it is critical to evaluate thyroid and renal function parameters in pregnant women, particularly those diagnosed with hypothyroidism. Most available literature emphasizes the isolated assessment of thyroid function or renal markers. However, few studies have explored their combined assessment during pregnancy, especially in resource-limited or rural settings. In India, where iodine deficiency and delayed diagnosis of thyroid disorders remain challenges, such studies are of particular relevance [13].

The current study was undertaken to assess and compare serum creatinine and thyroid-stimulating hormone (TSH) levels in euthyroid and hypothyroid pregnant women attending a tertiary care center. By establishing the correlation between these two parameters, the study aims to provide insights into the pathophysiological interplay between thyroid dysfunction and renal function during pregnancy. Understanding these associations is crucial for optimizing clinical monitoring and preventing adverse maternal and fetal outcomes.

2. MATERIAL AND METHODS

A comparative cross-sectional study was conducted at the Department of Biochemistry and Department of Physiology for a period of 12 months i.e, February 2024 to February 2025 at a tertiary care centre. The study included 200 pregnant women, divided into two groups:

Cases (n=100): Recently diagnosed hypothyroid pregnant women (age 20–40).

Controls (n=100): Age- and sex-matched euthyroid pregnant women.

Investigations:

TSH: Measured using Cobas e411 electro-chemiluminescence.

Creatinine: Assessed using the Modified Jaffe’s method.

Statistical Analysis: Data were analyzed using SPSS version 20.0. Results were expressed as mean ± SD. A p-value < 0.05 was considered statistically significant.

Inclusion Criteria

Pregnant women aged 20–40 years.

Recently diagnosed with hypothyroidism or confirmed euthyroid status.

No history of systemic diseases (cardiac, diabetes, liver, muscular disorders).

No current medication affecting thyroid or renal function.

Exclusion Criteria

Known cases of psychiatric or autoimmune connective tissue diseases.

Individuals on medications like antithyroid drugs, steroids, or chemotherapy.

History of chronic kidney disease or cancer.

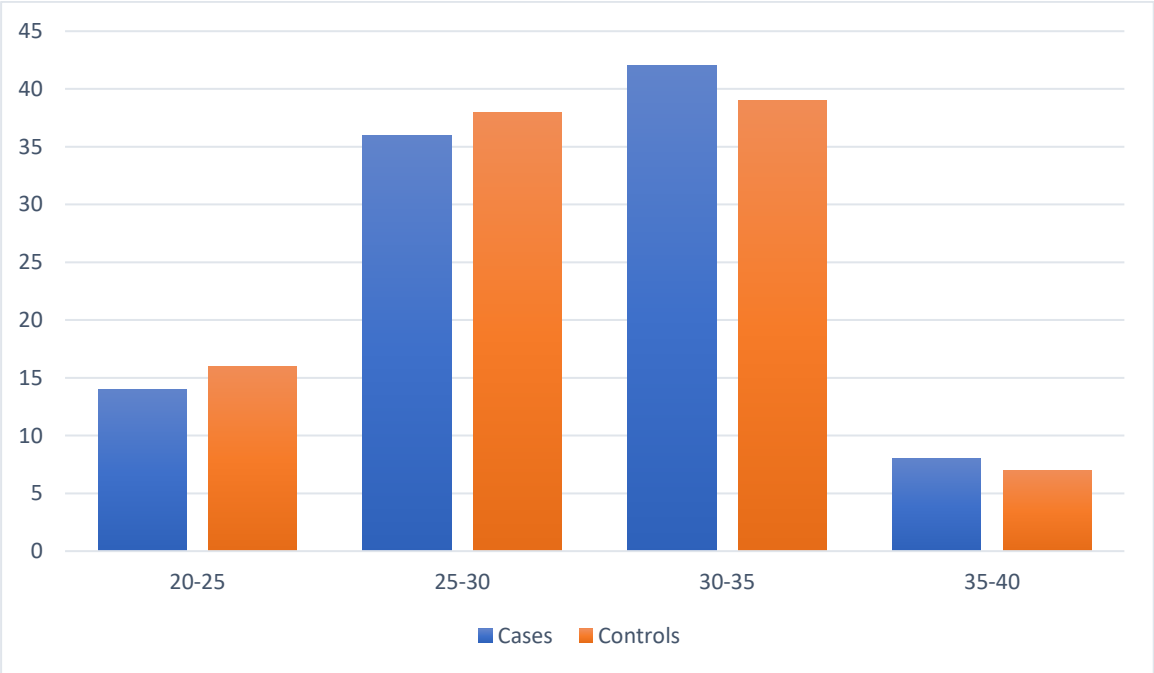
3. RESULT

In the present study out of 200 participants, including 100 hypothyroid patients and 100 controls, demographic characteristics were recorded. The demographic characteristics, serum TSH, and serum creatinine levels were analyzed accordingly.

Age (years)	*Case (n=100)*	*Control(n=100)*	*Total*
20-25	14	16	30
25-30	36	38	74
30-35	42	39	81
35-40	8	7	-
Total	100	100	200

Table 2: Age wise distribution

In the present study the maximum number of participants were in the 25-35 years of age group, indicating that young to middle- aged pregnant womeb were predominantly affected.



Graph 1. Showing Age wise distribution of case and controls.

Mean serum creatinine of cases was 0.87 ± 0.41, and in controls it was 0.55 ± 0.17, there being less mean serum creatinine in controls as compared to cases and the difference between both the groups being significant. Mean serum TSH in cases was 1.32 ± 0.89, and in controls it was observed to be 2.28 ± 0.91.

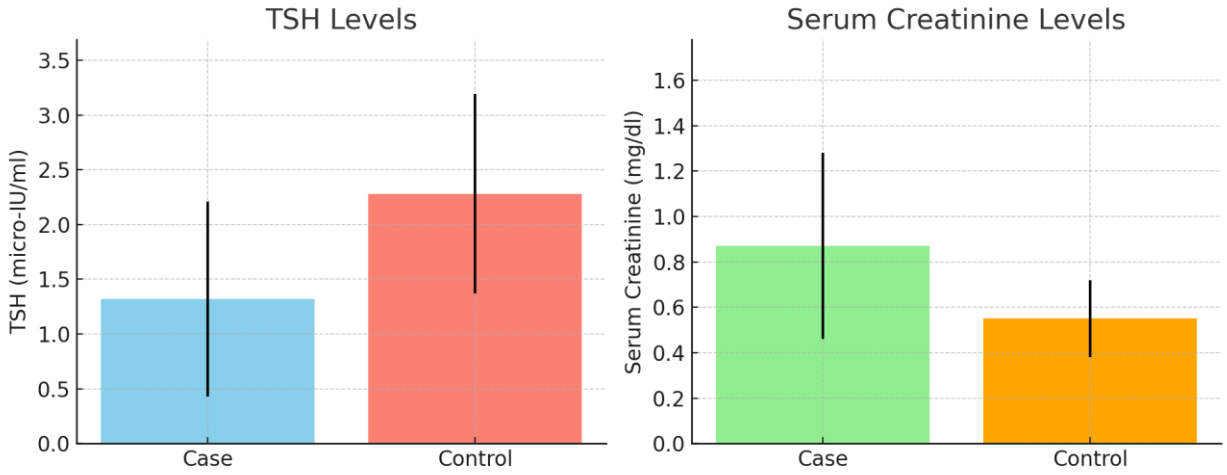
Parameters	**Case (n=100)**	**Control(n=100)**	**P value**
TSH (micro-IU/ml)	1.32 ± 0.89	2.28 ± 0.91	0.002
Serum creatinine (mg/dl)	0.87 ± 0.41	0.55 ± 0.17	0.001

Table 3. Comparison of level of TSH and creatinine

Comparison of TSH and Serum Creatinine Levels

The following statistical comparison was conducted between two groups: cases (n=100) and controls (n=100). The parameters compared include Thyroid Stimulating Hormone (TSH) and serum creatinine levels.

The mean TSH level in cases was 1.32 ± 0.89 micro-IU/ml, while in controls it was significantly higher at 2.28 ± 0.91 micro-IU/ml (P = 0.002). On the other hand, the mean serum creatinine level was higher in cases (0.87 ± 0.41 mg/dl) as compared to controls (0.55 ± 0.17 mg/dl), with this difference also being statistically significant (P = 0.001).



Graph 2: Bar graphs comparing TSH and serum creatinine levels between cases and controls.

The study findings demonstrate significant differences in both TSH and serum creatinine levels between cases and controls. Lower TSH and higher serum creatinine levels were observed in the case group compared to the control group. These results suggest potential physiological alterations in thyroid and renal function markers in the study population, warranting further investigation to understand the clinical implications.

4. DISCUSSION

The present study demonstrated a statistically significant difference in serum creatinine and TSH levels between cases and controls. Mean serum creatinine in cases was 0.87 ± 0.41 mg/dl, significantly higher than that in controls (0.55 ± 0.17 mg/dl, p = 0.001). Similarly, mean TSH levels were significantly lower in cases (1.32 ± 0.89 µIU/ml) compared to controls (2.28 ± 0.91 µIU/ml, p = 0.002).

Our findings are in concordance with the study by Abdelrahman et al., who reported significantly lower TSH levels in pregnant women with hypothyroidism compared to euthyroid controls, suggesting an altered thyroid profile in pathological pregnancies [14]. Kumar et al. also observed elevated creatinine levels in hypothyroid pregnancies, attributing this to reduced renal plasma flow and glomerular filtration rate (GFR) [15]. This supports the hypothesis that thyroid dysfunction adversely affects renal physiology during pregnancy.

Further supporting our data, Rai et al. highlighted a significant inverse correlation between TSH and renal function markers in pregnant women, affirming that thyroid hormones modulate renal hemodynamics and creatinine clearance [16]. Additionally, Dillmann et al. explained that hypothyroidism can decrease renal blood flow and GFR, thereby elevating serum creatinine levels [17].

In line with our study, Bhattacharjee et al. observed significantly altered TSH levels in pregnancy and emphasized the importance of trimester-specific reference ranges for accurate interpretation and clinical decision-making [18]. Saki et al. also reported lower TSH levels in complicated pregnancies and recommended routine screening for thyroid dysfunction [19].

Contrastingly, Casey et al. found no significant association between maternal TSH levels and adverse pregnancy outcomes in a large cohort study, challenging the need for universal thyroid screening during pregnancy [20]. Similarly, Cleary-Goldman et al. did not observe a consistent link between mild TSH alterations and renal function markers, suggesting that observed variations might fall within physiological adaptation during pregnancy [21].

Soldin et al. emphasized that pregnancy is a state of increased GFR and reduced serum creatinine due to hemodilution and physiological changes in renal function, which may mask subtle renal impairments if not interpreted against pregnancy-

specific norms [22].

Overall, the current findings underscore the interplay between thyroid function and renal physiology during pregnancy. The observed alterations in serum creatinine and TSH levels between cases and controls highlight the potential role of thyroid dysfunction in modifying renal parameters and vice versa. It is crucial to interpret these biochemical markers in the context of gestational changes to avoid misdiagnosis and ensure optimal maternal-fetal outcomes.

In the present study, we observed a statistically significant difference in both TSH and serum creatinine levels between hypothyroid (cases) and euthyroid (controls) pregnant women. The mean TSH level was significantly lower in cases (1.32 ± 0.89 µIU/ml) compared to controls (2.28 ± 0.91 µIU/ml), with a p-value of 0.002. Additionally, the mean serum creatinine level was notably higher in cases (0.87 ± 0.41 mg/dl) than in controls (0.55 ± 0.17 mg/dl), with a p-value of 0.001.

Our findings align with the study conducted by Saki et al., [23] who found that thyroid dysfunction, particularly hypothyroidism during pregnancy, can significantly affect renal function by reducing glomerular filtration rate (GFR), leading to elevated serum creatinine levels. Similarly, Kumar et al. reported that maternal hypothyroidism is associated with increased serum creatinine due to compromised renal plasma flow and metabolic clearance [24].

The altered TSH values observed in our study are consistent with the findings of Bhattacharjee et al., who emphasized the variation of thyroid hormone levels during pregnancy and the importance of interpreting these values with pregnancy-specific reference intervals [25]. They also observed that thyroid dysfunction, even in its subclinical form, may impact renal and metabolic parameters.

Rai et al. conducted a similar study on pregnant women and observed a significant inverse correlation between TSH levels and renal function markers, supporting the hypothesis that thyroid hormones influence renal hemodynamics [26]. Our results also align with the observations of Dillmann, who highlighted that hypothyroid states reduce renal perfusion and filtration due to decreased cardiac output and systemic vascular resistance, thereby elevating serum creatinine [27].

Contrary to our findings, Cleary-Goldman et al. did not report a significant association between mild thyroid dysfunction and pregnancy outcomes, suggesting that variations in TSH and creatinine might reflect normal physiological adaptation during gestation in some populations [28]. However, such discrepancies could be attributed to differences in sample size, population characteristics, or diagnostic thresholds.

In conclusion, our study reinforces the interrelationship between thyroid status and renal function during pregnancy. Elevated serum creatinine in hypothyroid women highlights the importance of early screening and monitoring of thyroid function to prevent renal and metabolic complications. Further longitudinal studies are recommended to validate these findings across larger and more diverse populations.

5. CONCLUSION

This study highlights a statistically significant alteration in renal function among hypothyroid pregnant women, demonstrated by elevated serum creatinine levels. The correlation between TSH and creatinine underscores the need for integrated monitoring. Timely detection and management of thyroid dysfunction can prevent complications by addressing underlying renal changes. Despite limitations such as sample size and study duration, the findings reinforce the clinical value of dual biochemical screening in antenatal care protocols.

DECLARATIONS:

Conflicts of interest: There is no any conflict of interest associated with this study

Consent to participate: There is consent to participate.

Consent for publication: There is consent for the publication of this paper.

Authors' contributions: Author equally contributed the work.

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