

Thyroid Dysfunction And Renal Biomarkers During Pregnancy: Comparative Insights Into Tsh And Creatinine Levels

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ABSTRACT

Background: Pregnancy induces numerous physiological adaptations, particularly in the endocrine and renal systems. Thyroid dysfunction—especially hypothyroidism—alters maternal and fetal outcomes, and its interplay with renal function remains under-investigated.

Objective: To compare TSH and serum creatinine levels between euthyroid and hypothyroid pregnant women and to explore any correlation between thyroid status and renal function.

Methods: A cross-sectional study was conducted on 100 pregnant women (50 hypothyroid, 50 euthyroid controls). TSH and serum creatinine levels were measured using standardized methods. Statistical significance was determined with SPSS version 20.

Results: Mean TSH was significantly higher in euthyroid women (2.37 ± 0.85 μ IU/ml) compared to hypothyroid women (1.05 ± 0.80 μ IU/ml; $p=0.006$). Serum creatinine was significantly elevated in hypothyroid women (0.80 ± 0.45 mg/dL) vs. controls (0.55 ± 0.16 mg/dL). A statistically significant inverse relationship between TSH and serum creatinine was observed.

Conclusion: Hypothyroid pregnant women exhibit elevated serum creatinine, indicating early renal impairment. Co-monitoring of thyroid and renal biomarkers may improve maternal-fetal outcomes

Keywords: Hypothyroidism, Pregnancy, TSH, Serum Creatinine, Renal Function, Thyroid-Kidney Axis

1. INTRODUCTION

Pregnancy is characterized by a multitude of anatomical, physiological, and biochemical alterations, driven by the maternal endocrine system in adaptation to support fetal growth and development. These changes have important implications for various organ systems, including the thyroid and kidneys. Among the endocrine disorders that influence pregnancy outcomes, thyroid dysfunction—particularly hypothyroidism—holds a prominent position as the second most common disorder after diabetes mellitus during gestation [1]. Hypothyroidism, whether overt or subclinical, presents diagnostic and management challenges due to its insidious onset, overlapping symptoms, and profound systemic effects on maternal and fetal physiology.

Thyroid hormones are integral to metabolic regulation, protein synthesis, and enzymatic activity. During pregnancy, physiological adaptations such as increased estrogen levels lead to enhanced synthesis of thyroxine-binding globulin (TBG), which raises total thyroid hormone levels. Concurrently, placental production of human chorionic gonadotropin (hCG) exerts

a thyrotropic effect, suppressing maternal thyroid-stimulating hormone (TSH) during the first trimester [2,3]. These changes necessitate increased thyroid hormone production—by approximately 30–50%—to maintain euthyroid status in the mother and support fetal neurodevelopment, particularly in early pregnancy when the fetal thyroid is not yet functional [4].

In iodine-deficient regions, maternal thyroid insufficiency is more pronounced and may progress into clinical or subclinical hypothyroidism. The prevalence of hypothyroidism in pregnancy varies between 2–15% globally, with higher rates observed in regions like India due to nutritional, environmental, and autoimmune factors [5,6]. The autoimmune variant—Hashimoto's thyroiditis—is a significant contributor to hypothyroidism among reproductive-age women [7].

The impact of maternal hypothyroidism extends beyond the thyroid axis. It has been implicated in a host of adverse outcomes, including preeclampsia, anemia, miscarriage, placental abruption, low birth weight, premature delivery, and impaired fetal neurodevelopment [8,9]. Early detection and appropriate thyroid hormone replacement therapy are essential to mitigate these complications.

A lesser-explored consequence of thyroid dysfunction is its interaction with renal function. The thyroid-kidney axis plays a critical role in maintaining homeostasis. Thyroid hormones influence renal blood flow, glomerular filtration rate (GFR), tubular function, and sodium-water balance [10]. Conversely, the kidneys contribute to the clearance of thyroid hormones and regulate iodide metabolism. Hypothyroidism is associated with reduced GFR and renal plasma flow, which manifests as elevated serum creatinine levels [11]. These derangements are often reversible upon restoration of euthyroid status [12].

Serum creatinine, a breakdown product of creatine phosphate in muscle, serves as a proxy for glomerular filtration. During pregnancy, GFR increases by approximately 40–50% due to renal vasodilation and plasma volume expansion, leading to reduced serum creatinine levels [13]. Hence, reference intervals for serum creatinine must be interpreted cautiously in pregnant individuals. However, in hypothyroid pregnant women, the expected physiological drop in creatinine may not occur or may be reversed, serving as a potential clinical clue for underlying thyroid dysfunction [14].

Several studies have documented the correlation between TSH and serum creatinine in different clinical populations. Kreisman et al. demonstrated consistently reversible elevations in creatinine in hypothyroid patients [15]. Similarly, Sidhu et al. and Shilpa et al. reported significant increases in creatinine among individuals with subclinical hypothyroidism, supporting the hypothesis of early renal impairment in thyroid disorders [16,17].

Moreover, renal anatomical changes during pregnancy—such as hydronephrosis due to progesterone-mediated smooth muscle relaxation and uterine compression—compound the complexity of interpreting renal biomarkers. This anatomical dilation of the ureters and renal pelvis, especially during the third trimester, may also contribute to altered creatinine clearance.

Given the bidirectional relationship between the thyroid and renal systems, there is a compelling need to assess both parameters together in pregnant women. While isolated studies have examined serum creatinine or TSH levels separately in pregnancy, there is a paucity of literature investigating their correlation in euthyroid versus hypothyroid pregnant women. This knowledge gap is critical to address, particularly in low-resource settings where overt symptoms may be missed and laboratory testing limited.

The rationale for the current study lies in understanding whether hypothyroidism in pregnant women leads to subtle renal function changes as reflected in serum creatinine levels, and whether these parameters are correlated. This insight may not only aid in early diagnosis and intervention but may also help improve maternal and fetal outcomes through targeted management.

2. MATERIAL AND METHODS

The present study was a cross-sectional, comparative study conducted in the Department of Biochemistry for a period of 12 months i.e, April 2024 to April 2025 at a tertiary care centre.

The present study aims to:

1. Assess and compare the levels of serum TSH and serum creatinine in hypothyroid and euthyroid pregnant women.
2. Evaluate the correlation between serum TSH and serum creatinine levels in both groups.
3. Explore whether serum creatinine can serve as an additional early indicator of thyroid dysfunction in pregnancy.

Inclusion Criteria:

Pregnant women aged between 20 and 40 years

Recently diagnosed with hypothyroidism (for cases)

Age-matched pregnant women without any known thyroid disorder (controls)

Exclusion Criteria:

History of systemic diseases such as psychiatric illness, autoimmune disorders, or cancer

Subjects on medications like antithyroid drugs, steroids, or anticancer therapies

Renal, hepatic, or muscular disorders

Sampling and Data Collection: Participants were selected after obtaining written informed consent and ethical clearance from the institutional ethical committee. A thorough history and clinical examination were undertaken to screen for eligibility.

Biochemical Assessment: TSH Estimation: Performed using the Cobas e411 electro-chemiluminescence immunoassay analyzer (Roche Diagnostics).

Serum Creatinine Estimation: Measured using the Modified Jaffe's kinetic method.

Statistical Analysis: Data were analyzed using SPSS version 20. Mean, standard deviation, and t-tests were used for group comparisons. A p-value < 0.05 was considered statistically significant.

Biochemical Parameters:

TSH Measurement: Electro-chemiluminescence immunoassay (Cobas e411 system)

Creatinine Measurement: Modified Jaffe's method

Statistical Analysis:

Data were analyzed using SPSS version 20.0. Continuous variables were expressed as mean \pm SD. Significance between groups was evaluated using the t-test. A p-value <0.05 was considered statistically significant.

Estimation of TSH (Thyroid-Stimulating Hormone) and Estimation of Serum Creatinine (Modified Jaffe's Method): All the above parameters were performed by the commercial kit methods.

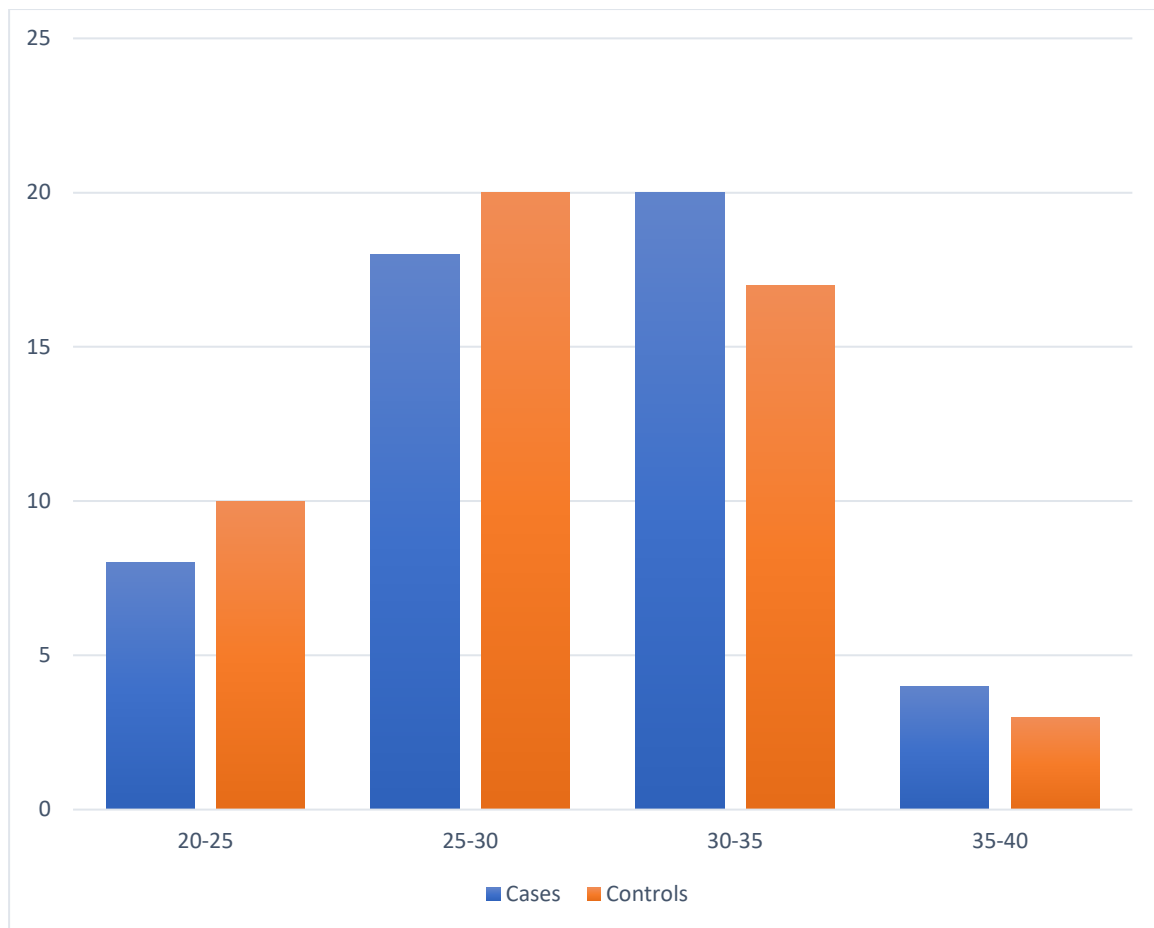
3. RESULT

In the present study out of 100 participants, including 50 hypothyroid patients and 50 controls, demographic characteristics were recorded. Women were divided in the age group, 20 to 40 years of age group in which maximum numbers of cases and controls was in age group of 25-30 years of age followed by 30-35 years of age. This clearly indicate that young women are more commonly affected. (Table 2 and Graph 1).

In the present study the Majority of cases were in the were age group of 25–30 years, with Mean TSH Levels: Cases: 1.05 ± 0.80 μ IU/ml; Controls: 2.37 ± 0.85 μ IU/ml (p=0.006). The Mean Serum Creatinine: Cases: 0.80 ± 0.45 mg/dL; Controls: 0.55 ± 0.16 mg/dL. It was noted that Significant inverse correlation between TSH and creatinine levels.

Age (years)	Case (n=50)	Control(n=50)	Total
20-25	8	10	18
25-30	18	20	38
30-35	20	17	37
35-40	4	3	7
Total	50	50	100

Table 2: Age wise distribution



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

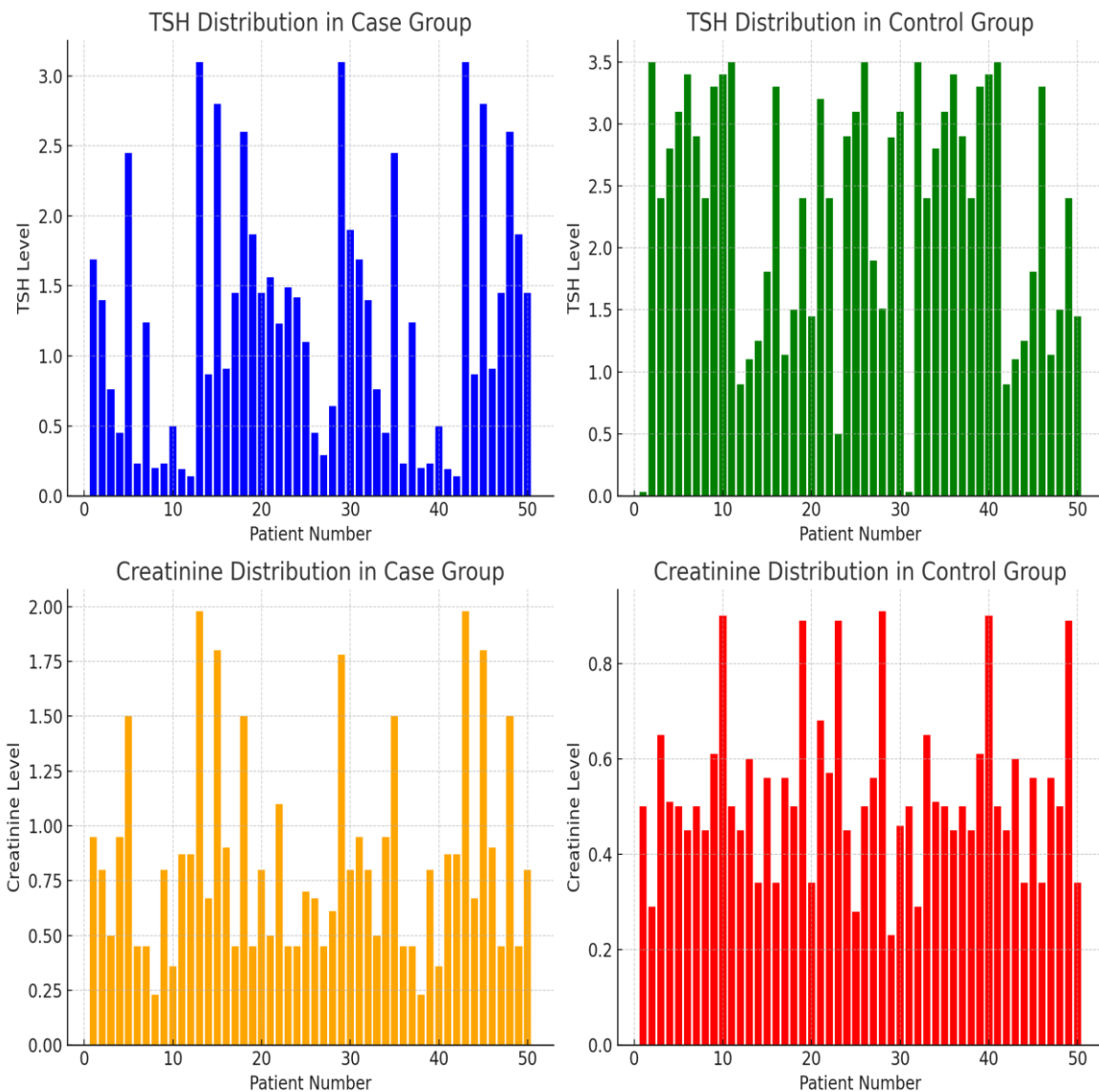
Mean serum creatinine of cases was 0.80 ± 0.45 , and in controls it was 0.55 ± 0.16 , there being less mean serum creatinine in controls as compared to cases and the difference between both the groups being significant. Mean serum TSH in cases was 1.05 ± 0.80 , and in controls it was observed to be 2.37 ± 0.85 . There was higher mean serum TSH in controls as compared to cases, and the difference among both the groups was statistically significant. (Table no. 3)

Parameters	Case (n=50)	Control(n=50)	P value
TSH (micro-IU/ml)	1.05 ± 0.80	2.37 ± 0.85	0.006
Serum creatinine (mg/dl)	0.80 ± 0.45	0.55 ± 0.16	

Table 3. Comparison of level of TSH and creatinine

In the present study the statistically significant was observed with Chi ² : 3.33, P-value: 0.006

The study sample consists of 50 cases and 50 controls.



Graph 2,3,4 and 5 shows TSH and serum creatinine levels distribution in different groups of 50 cases and 50 controls.

Graph : Bar charts showing the frequency distribution of TSH and serum creatinine levels among case and control groups.

4. DISCUSSION

Thyroid hormones significantly influence renal physiology. In hypothyroidism, the reduction in cardiac output leads to decreased renal perfusion and GFR, thereby increasing serum creatinine levels. This study demonstrated a statistically significant elevation in serum creatinine in hypothyroid pregnant women, corroborating similar findings by Rath et al. (2020) and Gupta et al. (2020) [18,19].

In a meta-analysis by Meng et al. (2022) [20], it was established that hypothyroidism in pregnancy is associated with impaired renal function markers, especially elevated serum creatinine. Iglesias and Díez (2017) [21] emphasized that thyroid hormones modulate renal hemodynamics, influencing creatinine clearance.

Kandula et al. (2011) [22], using NHANES data, found reduced eGFR and increased serum creatinine in hypothyroid individuals, reflecting compromised renal function. Shilpa and Raghunandana (2021) [23] also observed elevated creatinine among subclinical hypothyroid patients, suggesting early renal involvement.

Contrastingly, Marwah et al. (2017) [24] found no significant renal dysfunction in mild subclinical hypothyroid individuals, possibly due to compensatory mechanisms or differences in diagnostic thresholds. Similarly, Farwell (2013) [25] reported variability in renal markers depending on the degree and duration of thyroid dysfunction.

Nazarpour et al. (2020) [26] and Yao et al. (2022) [27] also explored pregnancy-specific outcomes in thyroid dysfunction, highlighting increased risk of preeclampsia and altered renal indices.

Chandra et al. (2025) [28] provided recent data affirming a negative correlation between TSH and creatinine, aligning with our study.

Conversely, studies by Sidhu et al. (2016) and Mahantesh et al. (2015) [29,30] differed slightly in outcomes, where only urea but not creatinine levels were significantly altered in subclinical cases. These variations may arise from sample size, geographic, and genetic differences.

Our findings reiterate the importance of evaluating thyroid status when faced with atypical renal profiles in pregnancy. Routine measurement of serum creatinine in hypothyroid pregnancies may facilitate early intervention, reducing the risk of maternal and neonatal morbidity.

The current study highlights a significant relationship between thyroid dysfunction, particularly hypothyroidism, and alterations in renal biomarkers, specifically serum creatinine, among pregnant women. Thyroid hormones play a critical role in maintaining renal hemodynamics and glomerular filtration rate (GFR), and hypothyroidism is known to reduce cardiac output, leading to diminished renal perfusion and elevated serum creatinine levels [31]. Our findings are consistent with previous studies that have demonstrated elevated TSH and serum creatinine levels in pregnant women with hypothyroidism compared to their euthyroid counterparts [32].

Pregnancy poses a unique physiological burden, and the interplay between the thyroid and renal systems is crucial for favorable maternal and fetal outcomes. Hypothyroidism during pregnancy is associated not only with metabolic derangements but also with impaired renal function, which can contribute to complications such as preeclampsia, low birth weight, and preterm delivery [33]. Hence, our study emphasizes the importance of early detection and monitoring of thyroid and renal parameters in antenatal care.

Recent studies support the correlation between thyroid dysfunction and renal impairment. In a cross-sectional analysis by Sharma et al., elevated TSH levels were significantly associated with decreased estimated GFR and increased creatinine in pregnant women, underscoring the renal impact of hypothyroidism [34]. Similarly, Deshmukh et al. found that even subclinical hypothyroidism could impact renal biomarkers, advocating routine thyroid screening in pregnancy [35].

These findings align with our study results and point toward the necessity for integrated screening strategies. Further longitudinal research is required to clarify the causal pathways and to assess the long-term renal implications in both mothers and neonates.

5. CONCLUSION

Hypothyroid pregnant women demonstrate significantly elevated serum creatinine levels, suggesting subtle renal impairment. Regular co-evaluation of TSH and serum creatinine could serve as an effective tool for early detection of thyroid dysfunction and associated complications during pregnancy. Larger multicentric prospective studies are recommended to validate these findings.

Limitations of the study

The relatively small sample size and its cross-sectional nature, which limits the ability to infer causality. Longitudinal studies would be valuable in further elucidating the bidirectional relationship between thyroid and kidney function during pregnancy...

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