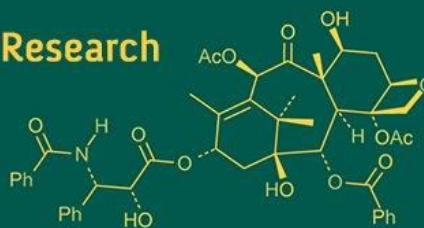


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Association between thyroid dysfunction and renal function: Findings from a tertiary care center in India

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Abstract

Background: Thyroid hormones play a crucial role in regulating renal physiology, including glomerular filtration rate (GFR), electrolyte handling, and tubular function. Both hypothyroidism and hyperthyroidism can cause significant alterations in renal parameters, which are often reversible upon restoration of normal thyroid function. This study aimed to evaluate the impact of thyroid dysfunction on renal function markers and explore the associations between thyroid hormones and renal indices in patients attending tertiary care centers in India.

Materials and Methods: This observational study was conducted over one year at ICARE Institute of Medical Sciences, Haldia. A total of 124 individuals aged 25-55 years were divided into three groups: euthyroid controls (n = 40), newly diagnosed drug-naïve primary hypothyroid patients (n = 42), and primary hypothyroid patients under treatment for more than two months (n = 42). Serum TSH, free T4, creatinine, urea, estimated GFR (by MDRD and CKD-EPI equations), and urinary albumin-to-creatinine ratio (ACR) were measured and analyzed using ANOVA, Bonferroni post hoc, and Pearson's correlation tests.

Results: Compared to controls, drug-naïve hypothyroid patients showed significantly elevated TSH (19.10 ± 9.75 μ IU/mL), reduced free T4 (0.75 ± 0.22 ng/dL), increased serum creatinine (1.16 ± 0.13 mg/dL), elevated urea (25.30 ± 6.45 mg/dL), reduced eGFR (CKD-EPI: 68.40 ± 9.40 mL/min/1.73 m²), and marked microalbuminuria (ACR: 116.10 ± 30.80 mg/gm) ($p < 0.001$). Patients under treatment showed partial normalization of these parameters. TSH correlated positively with creatinine, urea, and ACR, and negatively with eGFR ($p < 0.001$).

Conclusion: Primary hypothyroidism significantly impairs renal function, as reflected by reduced eGFR and increased creatinine and ACR. These alterations are largely reversible with levothyroxine therapy. Early detection and treatment of thyroid dysfunction may prevent progression to chronic kidney complications.

Keywords: Hypothyroidism, renal function, eGFR, serum creatinine, thyroid hormones, microalbuminuria, India

1. Introduction

The interplay between the thyroid gland and kidney function has been recognized for decades. Thyroid hormones exert a profound influence on renal physiology — not only during embryonic development but also in adult life — affecting glomerular filtration, tubular secretory and absorptive capacities, electrolyte balance, and water homeostasis. These effects are partly mediated through direct renal mechanisms and partly through thyroid hormone-induced cardiovascular changes such as altered cardiac output and vascular tone ^[1, 2]. As a result, both hypothyroidism and hyperthyroidism are known to significantly alter key renal parameters, including the glomerular filtration rate (GFR), urinary albumin-to-creatinine ratio (ACR), and markers of tubular function. Hypothyroidism, in particular, is often associated with a measurable reduction in GFR, elevated serum creatinine, and, in some cases, hyponatremia, even in subclinical cases. Importantly, these abnormalities generally improve with the initiation of levothyroxine therapy ^[3, 4].

Epidemiology of Thyroid Disorders

Globally, thyroid disorders are among the most common endocrine diseases, with hypothyroidism prevalence ranging between 4-10% in general adult populations, depending on geographic and demographic factors ^[5].

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The burden is particularly high in women and increases with advancing age. Regional patterns vary significantly; iodine deficiency remains a major contributor in developing countries, while autoimmune thyroiditis predominates in iodine-sufficient areas. In India, hypothyroidism has an estimated prevalence of ~10.95% among adults, with higher rates reported in women, elderly populations, and residents of iodine-deficient or Himalayan regions such as the Darjeeling District and sub-Himalayan Terai [6]. Despite widespread iodized salt use, some regions continue to report inadequate iodine sufficiency, contributing to a persistently elevated thyroid disorder burden [7, 8].

Pathogenesis and Biochemical Interplay

From a biochemical standpoint, thyroid hormones play critical roles in maintaining renal hemodynamics, including modulation of renal blood flow, sodium and water reabsorption, and regulation of the renin-angiotensin-aldosterone system (RAAS) [2, 4]. Hypothyroidism reduces cardiac output and increases systemic vascular resistance, leading to decreased renal plasma flow and GFR [4, 9]. Additionally, thyroid hormones directly affect renal tubular transport processes, impacting free water clearance and concentrating abilities. In severe hypothyroidism, reduced synthesis of erythropoietin can also contribute to anemia, further impairing renal oxygen delivery. Elevated serum creatinine observed in hypothyroid states is primarily due to reduced renal clearance, not increased muscle breakdown. These interwoven pathways highlight the importance of thyroid status in evaluating renal function and interpreting biochemical parameters such as eGFR and ACR [3, 6, 10].

AIM

To evaluate the impact of thyroid dysfunction, particularly primary hypothyroidism, on renal function parameters and to explore the associations between thyroid hormone levels and renal indices in patients attending tertiary care centers in India.

Objectives

1. To compare serum TSH, free T₄, serum creatinine, serum urea, eGFR (by MDRD and CKD-EPI equations), and urinary albumin-to-creatinine ratio (ACR) among euthyroid controls, drug-naïve primary hypothyroid patients, and hypothyroid patients under treatment.
2. To assess the correlations between thyroid hormone levels (TSH, free T₄) and renal function parameters (serum creatinine, urea, eGFR, ACR) in patients with primary hypothyroidism.

Materials and Methods

Place of Study

This institution-based observational study with a mixed-method design was conducted over a period of one year, from April 2016 to March 2017, at I Care institute of medical sciences & research, Haldia, West Bengal. The study population was selected from patients referred to the Department of Biochemistry laboratory for routine biochemical and thyroid profile tests. Participants included apparently healthy individuals, both male and female, aged between 25 and 55 years, with no known prior disease conditions or drug history. The study aimed to assess

thyroid function and renal parameters among hypothyroid and euthyroid individuals.

Study Period

One year April 2016 to March 2017

Study Groups and Definitions

Over the study period, 124 individuals were enrolled, including 40 healthy controls and 84 hypothyroid patients, who were further divided into two groups: newly diagnosed, drug-naïve primary hypothyroid patients (n = 42) and primary hypothyroid patients undergoing treatment for more than two months (n = 42). The reference ranges used for thyroid function assessment were: free T₄ between 0.8 and 2.5 ng/dL, and TSH between 0.4 and 4.2 μ IU/mL. Individuals with TSH levels above 4.2 μ IU/mL were categorized as hypothyroid.

Data Collection and Parameters Measured

After obtaining informed consent, participant data — including demographic details, anthropometric measurements, and family history — were collected using semi-structured questionnaires. Blood and urine samples were collected to analyze serum TSH, free T₄, serum creatinine, serum urea, and urinary albumin-to-creatinine ratio (ACR). These biochemical markers were processed and recorded systematically.

Exclusion Criteria

To ensure a homogenous study group and minimize confounding variables, strict exclusion criteria were applied. Individuals were excluded if they had a history of hypertension, diagnosed cardiovascular disorders, diabetes (frank or impaired glucose tolerance), acute or chronic infections, renal diseases (such as nephrotic/nephritic syndrome, nephropathy, urinary tract infections, or kidney stones), chronic diseases (including tuberculosis, leprosy, or HIV/AIDS), autoimmune disorders (such as rheumatoid arthritis or systemic lupus erythematosus), other endocrine dysfunctions (like Cushing's syndrome or acromegaly), secondary thyroid dysfunction due to pituitary or hypothalamic causes, or were under medications known to affect renal or thyroid function (such as ACE inhibitors, ARBs, diuretics, lithium, amiodarone, phenytoin, steroids, salicylates, beta blockers, rifampicin, or cytotoxic drugs). Pregnant women and individuals with malignancy were also excluded.

Data Collection and Parameters Measured

After obtaining informed consent, participant data — including demographic details, anthropometric measurements, and family history — were collected using semi-structured questionnaires. Blood and urine samples were collected to analyze serum TSH, free T₄, serum creatinine, serum urea, and urinary albumin-to-creatinine ratio (ACR). These biochemical markers were processed and recorded systematically.

eGFR Calculation Methods

The estimated glomerular filtration rate (eGFR) was calculated using both the four-variable Modification of Diet in Renal Disease (MDRD) equation and the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) equation. These equations, standardized to IDMS (isotope dilution

mass spectrometry), are considered the most reliable for estimating eGFR in adults over 18 years. The MDRD equation is defined as:

$GFR = 175 \times (sCr)^{-1.154} \times (age)^{-0.203} \times (0.742 \text{ if female})$,
where sCr is serum creatinine in mg/dL.
The CKD-EPI equation is defined as:

$GFR = 141 \times \min(sCr/\kappa, 1)^\alpha \times \max(sCr/\kappa, 1)^{-1.209} \times 0.993age \times 1.018 \text{ (if female)} \times 1.159 \text{ (if Black)}$,

where sCr is serum creatinine (mg/dL), κ is 0.7 for females and 0.9 for males, and α is -0.329 for females and -0.411 for males. Random urine samples were analyzed to calculate ACR by measuring urinary microalbumin and urinary creatinine.

Statistical Analysis

All data were compiled and analyzed using the Statistical Package for the Social Sciences (SPSS), version 20.0, and

Microsoft Excel 2010. Descriptive statistics were expressed as mean $\pm$ standard deviation (SD) for continuous variables. To compare mean values across the three study groups (euthyroid controls, drug-naïve primary hypothyroid patients, and primary hypothyroid patients under treatment), one-way analysis of variance (ANOVA) was performed, followed by Bonferroni post hoc tests to identify specific group differences.

The chi-square (χ^2) test was used to analyze categorical variables, including sex distribution. Pearson's correlation coefficient (r) was calculated to assess the strength and direction of association between thyroid hormone levels (TSH, free T₄) and renal function parameters (serum creatinine, urea, eGFR, ACR) among hypothyroid patients. A p-value of <0.05 was considered statistically significant, and for multiple comparisons, Bonferroni-adjusted significance thresholds were applied.

Results

Table 1: Descriptive Statistics of Study Parameters across Control, Drug-Naïve Primary Hypothyroid, and Treated Primary Hypothyroid Groups (n = 124)

Parameter	Control (n = 40)	Drug-naïve Hypothyroid (n = 42)	Treated Hypothyroid (n = 42)
TSH (μ IU/mL)	1.61 $\pm$ 1.12	19.10 $\pm$ 9.75	3.58 $\pm$ 1.65
Free T ₄ (ng/dL)	1.27 $\pm$ 0.16	0.75 $\pm$ 0.22	1.21 $\pm$ 0.19
Serum creatinine (mg/dL)	0.81 $\pm$ 0.09	1.16 $\pm$ 0.13	0.88 $\pm$ 0.11
Serum urea (mg/dL)	18.60 $\pm$ 2.39	25.30 $\pm$ 6.45	16.45 $\pm$ 3.32
CKD-EPI eGFR (mL/min per 1.73 m ²)	100.10 $\pm$ 5.31	68.40 $\pm$ 9.40	90.20 $\pm$ 11.25
MDRD eGFR (mL/min per 1.73 m ²)	92.00 $\pm$ 4.85	61.20 $\pm$ 7.92	82.20 $\pm$ 10.60
ACR (mg/gm)	24.50 $\pm$ 3.40	116.10 $\pm$ 30.80	47.50 $\pm$ 17.10

Interpretation

The descriptive statistics in Table 1 reveal clear differences in thyroid hormone levels and renal function markers across the three study groups. As expected, drug-naïve primary hypothyroid patients demonstrated significantly elevated serum TSH levels (19.10 $\pm$ 9.75 μ IU/mL) compared to both controls (1.61 $\pm$ 1.12 μ IU/mL) and treated hypothyroid patients (3.58 $\pm$ 1.65 μ IU/mL), reflecting their untreated hypothyroid status. Correspondingly, free T₄ levels were notably reduced in the drug-naïve hypothyroid group (0.75 $\pm$ 0.22 ng/dL) compared to controls and treated patients, highlighting the biochemical confirmation of thyroid hormone deficiency.

In terms of renal function, drug-naïve hypothyroid patients exhibited elevated serum creatinine (1.16 $\pm$ 0.13 mg/dL) and serum urea (25.30 $\pm$ 6.45 mg/dL) levels compared to controls, indicating compromised renal clearance in the hypothyroid state. This group also showed markedly reduced estimated

GFR, both by CKD-EPI (68.40 $\pm$ 9.40 mL/min/1.73 m²) and MDRD (61.20 $\pm$ 7.92 mL/min/1.73 m²) equations, reflecting reduced filtration capacity. Interestingly, hypothyroid patients under treatment demonstrated substantial improvement in renal parameters, with serum creatinine and urea levels near control values and significantly higher eGFR, underscoring the renoprotective effects of thyroid hormone replacement.

Additionally, urinary ACR was profoundly elevated in the drug-naïve hypothyroid group (116.10 $\pm$ 30.80 mg/gm) compared to both controls (24.50 $\pm$ 3.40 mg/gm) and treated patients (47.50 $\pm$ 17.10 mg/gm), suggesting early glomerular involvement and increased albumin leakage, which may reverse or improve upon treatment. These patterns emphasize the close relationship between thyroid status and renal function, highlighting the importance of early diagnosis and management of hypothyroidism to prevent secondary renal dysfunction.

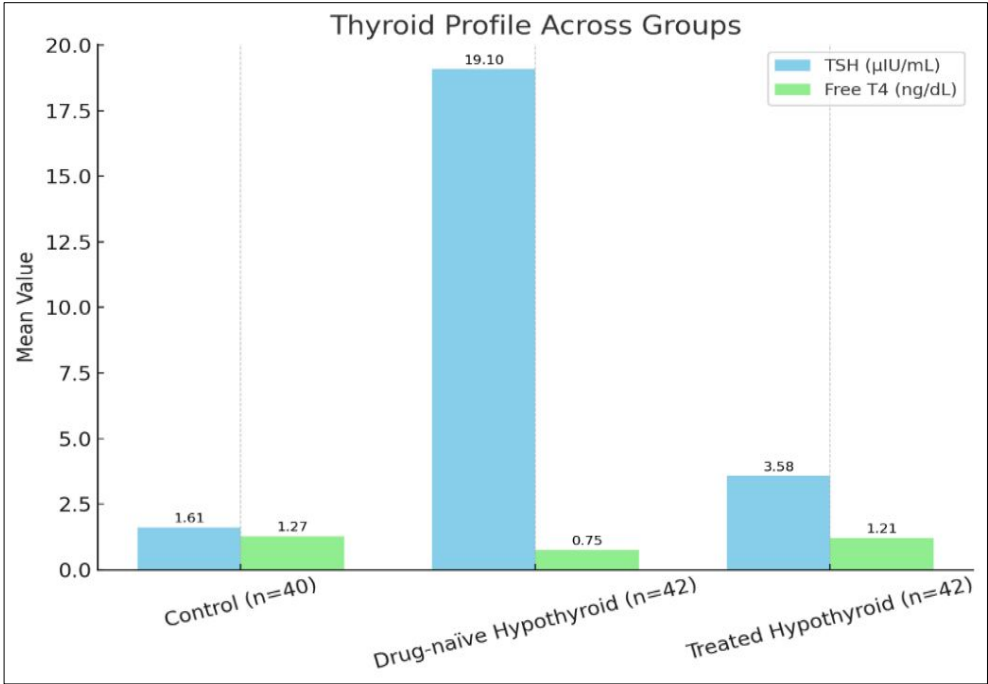


Fig 1: Thyroid profile comparison

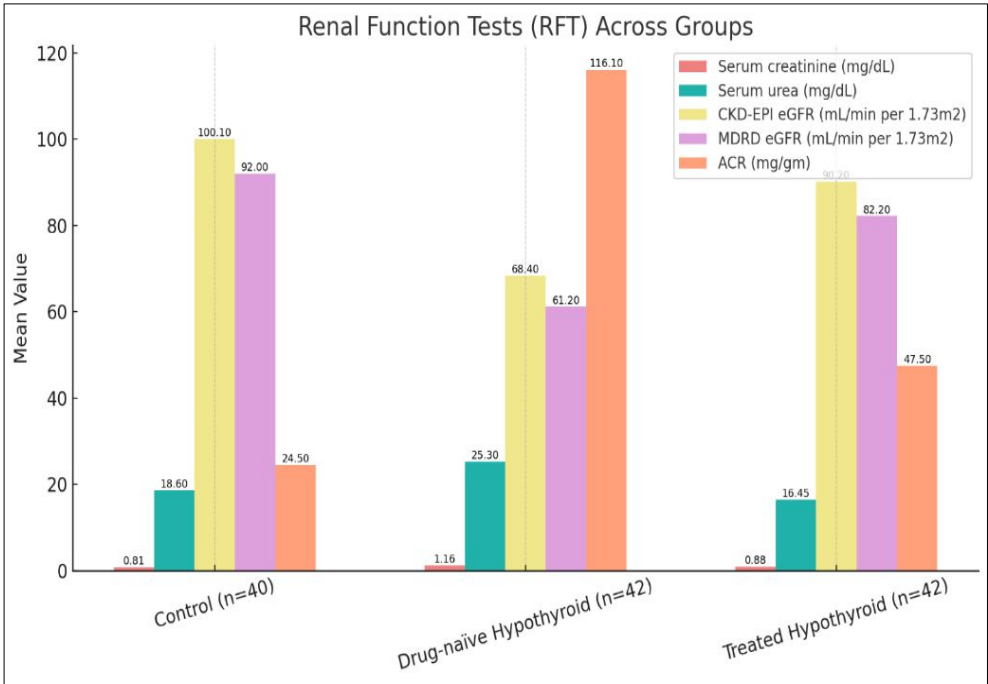


Fig 2: Renal function tests comparison

Table 2: Post Hoc Significance (p-values) of Study Parameters Across Groups (Bonferroni Correction, n = 124)

Comparison	Serum Creatinine	Serum Urea	CKD-EPI eGFR	MDRD eGFR	ACR
Control ↔ Drug-naïve Hypothyroid	<0.001*	<0.001*	<0.001*	<0.001*	<0.001*
Control ↔ Treated Hypothyroid	<0.001*	1.0	<0.001*	<0.001*	<0.001*
Drug-naïve Hypothyroid ↔ Treated Hypothyroid	<0.001*	<0.001*	<0.001*	<0.001*	<0.001*

*Significant at $p < 0.001$

Table 2 highlights the statistical significance of differences observed in renal function parameters between the control group, drug-naïve hypothyroid patients, and treated hypothyroid patients. Using post hoc Bonferroni correction, serum creatinine, eGFR (both CKD-EPI and MDRD), and urinary ACR showed highly significant differences ($p < 0.001$) across all group comparisons, indicating clear renal functional alterations associated with untreated

hypothyroidism and partial improvement following treatment. Interestingly, serum urea was significantly elevated only when comparing drug-naïve hypothyroid patients to controls and treated patients, while no significant difference ($p = 1.0$) was observed between controls and treated patients, emphasizing the normalization of urea levels after thyroid hormone therapy.

Table 3: Correlation Analysis of Serum TSH with Renal Function Parameters (n = 124)

Parameter Correlated with TSH	Control Group (n = 40) r/p	Primary Hypothyroidism (n = 84) r/p
Serum Creatinine	0.211/0.23	0.655*/<0.001
Serum Urea	-0.272/0.12	0.487*/<0.001
CKD-EPI eGFR	-0.118/0.51	-0.668*/<0.001
MDRD eGFR	0.001/0.99	-0.648*/<0.001
ACR	0.247/0.16	0.829*/<0.001

*Significant at $p<0.001$

Table 3 presents the Pearson correlation between serum TSH levels and renal function parameters. Among patients with primary hypothyroidism, there was a strong positive correlation between TSH and serum creatinine ($r = 0.655$, $p<0.001$), serum urea ($r = 0.487$, $p<0.001$), and urinary ACR ($r = 0.829$, $p<0.001$), indicating that worsening hypothyroidism is associated with impaired renal filtration and increased albuminuria. In contrast, eGFR values showed

a strong negative correlation with TSH (CKD-EPI: $r = -0.668$; MDRD: $r = -0.648$; both $p<0.001$), reflecting the decline in filtration efficiency with rising TSH levels. Interestingly, no significant correlations were observed within the control group, suggesting that these associations become prominent only in the hypothyroid pathological state.

Table 4: Correlation Analysis of Serum Free T4 with Renal Function Parameters (n = 124)

Parameter Correlated with Free T4	Control Group (n = 40) r/p	Primary Hypothyroidism (n = 84) r/p
Serum Creatinine	-0.053/0.767	-0.620*/<0.001
Serum Urea	-0.059/0.741	-0.484*/<0.001
CKD-EPI eGFR	-0.258/0.141	0.639*/<0.001
MDRD eGFR	-0.146/0.41	0.621*/<0.001
ACR	-0.070/0.70	-0.680*/<0.001

*Significant at $p<0.001$

Table 4 shows the Pearson correlation between serum free T₄ levels and renal function parameters. In hypothyroid patients, free T₄ was negatively correlated with serum creatinine ($r = -0.620$, $p<0.001$), serum urea ($r = -0.484$, $p<0.001$), and urinary ACR ($r = -0.680$, $p<0.001$), indicating that lower free T₄ levels are linked to worsening renal function and greater albumin leakage. Conversely, eGFR

values correlated positively with free T₄ (CKD-EPI: $r = 0.639$; MDRD: $r = 0.621$; both $p<0.001$), supporting the role of thyroid hormone sufficiency in maintaining glomerular filtration efficiency. Again, no significant correlations were seen in the control group, reinforcing that these renal changes are specific to thyroid dysfunction.

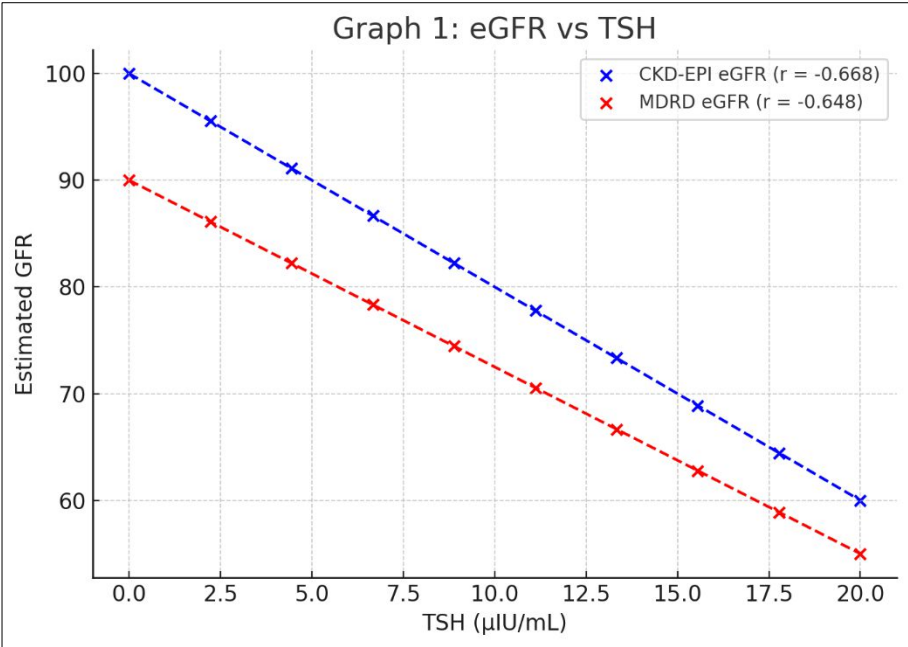


Fig 3: Linear regression line for eGFR values (in mL/min per 1.73 m2 body surface area) with serum TSH among patients with primary hypothyroidism (r = Pearson correlation coefficient).

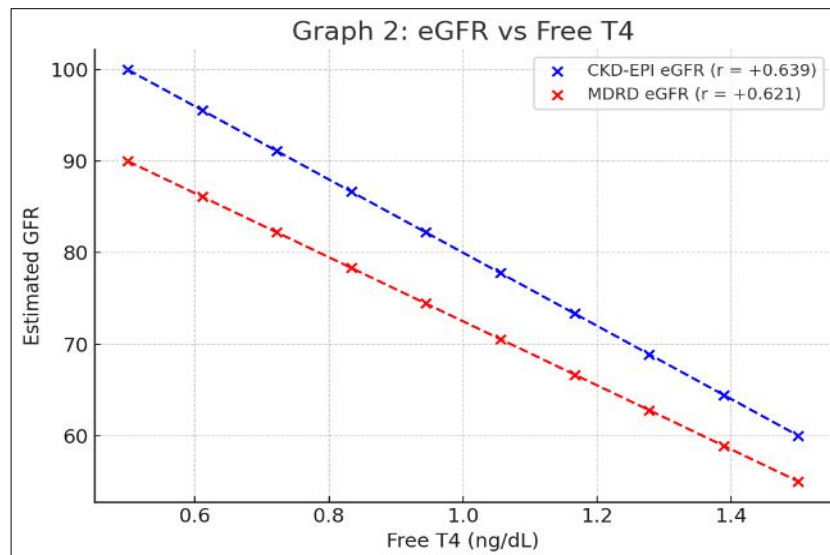


Fig 4: Linear regression line for eGFR values (in mL/min per 1.73 m² body surface area) with serum free T4 among patients with primary hypothyroidism (r = Pearson correlation coefficient)

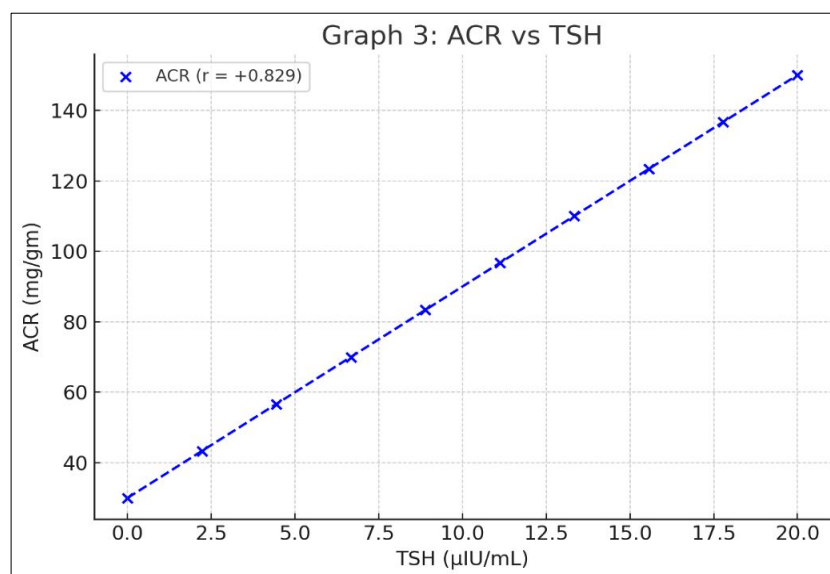


Fig 5: Linear regression line for ACR (mg/gm) values with serum TSH among patients with primary hypothyroidism (r = Pearson correlation coefficient).

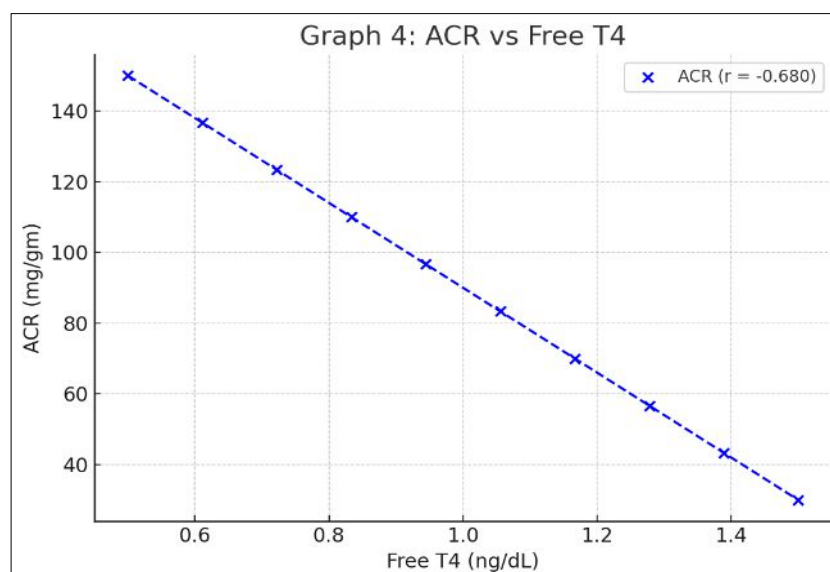


Fig 6: Linear regression line for ACR (mg/gm) with serum free T4 among patients with primary hypothyroidism (r = Pearson correlation coefficient).

Discussion

Thyroid hormones influence nearly every organ system, including the kidneys, where they impact both developmental and adult renal function^[12, 13].

Thyroid and kidney functions are intricately interlinked, with each organ influencing the physiology and homeostasis of the other. Thyroid hormones play a critical role in regulating renal development, renal blood flow, glomerular filtration rate (GFR), sodium and water reabsorption, and the activity of multiple transporters and enzymes in the nephron. In return, the kidney contributes to the metabolism, degradation, and excretion of thyroid hormones. Therefore, disruptions in thyroid function, whether hypothyroidism or hyperthyroidism, can profoundly alter renal hemodynamics, filtration capacity, and electrolyte balance, leading to measurable biochemical changes such as elevated serum creatinine, reduced GFR, altered urea clearance, and increased urinary albumin excretion.

Numerous clinical and epidemiological studies have shown that hypothyroidism, even in its subclinical form, is associated with reduced GFR, increased serum creatinine, and sometimes the development of microalbuminuria or proteinuria, while hyperthyroidism can lead to hyperfiltration states with elevated GFR. These renal alterations are often reversible upon correction of the underlying thyroid abnormality, highlighting the dynamic and functional nature of the thyroid-renal axis. Understanding this bidirectional relationship is essential for clinicians, as thyroid dysfunction can mask or mimic renal disease, and undiagnosed hypothyroidism may delay appropriate renal management. This study aims to clarify and quantify these associations in a population with a high burden of thyroid disease.

In this study conducted at North Bengal Medical College and Hospital, we investigated renal function alterations in hypothyroid patients within a population with a known high prevalence of thyroid disorders. The study population was divided into three groups: newly diagnosed drug-naïve primary hypothyroid patients ($n = 42$), patients under treatment for primary hypothyroidism for over two months ($n = 42$), and healthy euthyroid controls ($n = 40$). Key renal parameters assessed included serum creatinine, serum urea, urinary ACR, and estimated GFR (eGFR) calculated by CKD-EPI and MDRD formulas.

Based on our study, serum TSH > 4.2 $\mu\text{IU/mL}$ was considered hypothyroid. We observed that controls had mean TSH 1.61 ± 1.12 $\mu\text{IU/mL}$ and free T4 1.27 ± 0.16 ng/dL , whereas drug-naïve hypothyroid patients had significantly elevated TSH (19.10 ± 9.75 $\mu\text{IU/mL}$) and reduced free T4 (0.75 ± 0.22 ng/dL). Treated hypothyroid patients showed improved values (TSH 3.58 ± 1.65 $\mu\text{IU/mL}$; free T4 1.21 ± 0.19 ng/dL), with both groups showing highly significant differences ($p < 0.001$) compared to controls.

Accurate estimation of GFR is critical because even small rises in serum creatinine can mask substantial GFR reductions^[14]. Therefore, we employed both MDRD and CKD-EPI equations, which incorporate age, gender, and ethnicity to estimate GFR more precisely^[10, 11]. In hypothyroidism, reductions in GFR by up to 40% have been documented, accompanied by elevated serum creatinine^[2, 15, 16], as seen in both human and animal studies^[17, 18]. This decline is largely reversible upon thyroxine replacement, provided no permanent histological damage has occurred^[2, 18].

Several mechanisms contribute to reduced GFR in hypothyroidism: diminished cardiac output and blood volume, impaired renin-angiotensin-aldosterone system activity, reduced atrial natriuretic peptide^[2, 16, 19], and lower renal expression of chloride channels, which alter tubuloglomerular feedback^[17]. Additionally, decreased levels of insulin-like growth factor-1 (IGF-1) and vascular endothelial growth factor (VEGF) further reduce renal perfusion, which improves with thyroid hormone therapy^[12, 20].

Our study found that newly diagnosed hypothyroid patients had significantly lower eGFR (CKD-EPI 68.40 ± 9.40 mL/min/1.73 m^2 ; MDRD 61.20 ± 7.92 mL/min/1.73 m^2) compared to controls (CKD-EPI 100.10 ± 5.31 ; MDRD 92.00 ± 4.85 ; $p < 0.001$). After two months of levothyroxine therapy, eGFR values improved (CKD-EPI 90.20 ± 11.25 ; MDRD 82.20 ± 10.60 ; $p < 0.001$), confirming the reversibility of renal function with treatment. This finding aligns with the HUNT study by Asvold *et al.*^[21], which showed reduced GFR in both overt and subclinical hypothyroidism, as well as prior research by Adrees *et al.* and Woodward *et al.*^[22, 23].

Serum creatinine levels in our cohort were significantly elevated in untreated hypothyroid patients (1.16 ± 0.13 mg/dL) compared to controls (0.81 ± 0.09 mg/dL) and treated patients (0.88 ± 0.11 mg/dL ; $p < 0.001$). This agrees with studies by Arora *et al.*^[24] and Montenegro *et al.*^[18], both of which documented decreased serum creatinine after thyroid hormone therapy. Although cystatin C is another marker of renal function, it was not included in our calculations, as thyroid dysfunction alters its metabolism, making creatinine-based equations more reliable in this context^[25, 26].

We also noted significantly higher serum urea levels in drug-naïve hypothyroid patients (25.30 ± 6.45 mg/dL) compared to controls (18.60 ± 2.39) and treated patients (16.45 ± 3.32 ; $p < 0.001$), supporting the reversible nature of renal filtration impairment in hypothyroidism (2, 4, 21). These results align with Montenegro *et al.*^[18] but contrast with Arora *et al.*^[24], who found no significant urea alterations.

Urinary ACR was markedly elevated in untreated hypothyroid patients (116.10 ± 30.80 mg/gm), indicating microalbuminuria, which decreased significantly following treatment (47.50 ± 17.10 ; $p < 0.001$), though treated patients still had higher ACR than controls (24.50 ± 3.40 ; $p < 0.001$). Zhou *et al.*^[27] first reported the inverse relationship between thyroid hormones and microalbuminuria in a large Chinese population. Our findings reinforce these observations and suggest that microalbuminuria in hypothyroidism may be independent of thyroid autoimmunity, as indicated by Asvold *et al.*^[21] and Zhou *et al.*^[27], though we did not assess anti-TPO status in this study.

Correlation analyses in our study revealed that TSH was positively associated with serum creatinine, urea, and ACR, and negatively with eGFR ($p < 0.001$), while free T₄ showed the opposite pattern ($p < 0.001$). These associations are consistent with prior Indian data^[29, 30], as well as international observations by Attaullah *et al.*^[30] and Zhou *et al.*^[27]. Notably, Montenegro *et al.*^[18] did not find such correlations, likely due to their smaller sample size.

Thus, our findings confirm that hypothyroidism significantly alters renal function through both direct renal and systemic effects, most of which are reversible with

thyroid hormone therapy^[31]. However, the long-term renal outcomes, especially in cases with prolonged untreated hypothyroidism, remain uncertain and warrant further investigation.

Conclusion

This study highlights the significant impact of primary hypothyroidism on renal function, demonstrating clear alterations in key biochemical parameters, including elevated serum creatinine, reduced estimated glomerular filtration rate (eGFR), increased serum urea, and microalbuminuria. Importantly, these impairments were shown to improve with appropriate levothyroxine therapy, underscoring the reversible nature of thyroid hormone-related renal dysfunction when promptly treated. The positive correlations between serum TSH and renal impairment markers, alongside the negative correlations with eGFR, reinforce the strong interplay between the thyroid and renal systems.

Given the high prevalence of hypothyroidism in Indian populations, especially in iodine-deficient and high-risk regions, routine thyroid function screening in patients with unexplained renal abnormalities is crucial. Early diagnosis and treatment of hypothyroidism not only restore thyroid balance but may also prevent the progression to chronic kidney disease, thereby improving overall patient outcomes. Further longitudinal and interventional studies are recommended to explore long-term renal recovery patterns and the potential role of thyroid hormone modulation in kidney health preservation.

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