International Journal of Applied and Traditional Medicine
https://publishingjournals.org/ijatm
Volume: 3 | Number: 2 (2026) April, Page 9-14

Morphometric and Functional Predictors of Structural Changes in Kidneys in Less-Mobile Patients with Hypothyroidism

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Received: 10-02-2026
Accepted: 21-03-2026
Published: 9-04-2026

Abstract: This study investigates the correlation between thyroid hormone deficiency and renal structural alterations in patients with limited physical activity. Hypothyroidism leads to decreased renal plasma flow, cardiac output, and GFR, which are significantly exacerbated by a sedentary lifestyle. We analyzed morphometric data (kidney volume, cortical thickness, tubular diameter) and functional markers (eGFR, creatinine) to identify early predictors of chronic kidney disease (CKD). Results indicate that reduced mobility significantly accelerates tubular atrophy and glomerular basement membrane thickening in hypothyroid states. Clinical evidence shows a higher prevalence of subclinical hypothyroidism (17.9%) in patients with GFR <60 ml/min. Thyroid hormone replacement therapy (THRT) remains a crucial intervention for the potential reversibility of these structural and functional impairments.

Keywords: Hypothyroidism, Less-Mobile Patients, Morphometric Predictors, Renal Structural Changes, Glomerular Filtration Rate (GFR), Tubular Atrophy, Renin-Angiotensin-Aldosterone System (RAAS), Renal Resistive Index (RRI), THRT, Reversibility

Introduction

The complex interplay between the endocrine system and renal physiology is a cornerstone of modern internal medicine. Among various endocrine disorders, hypothyroidism stands out due to its profound systemic impact, particularly on the renal system. According to recent clinical data indexed in PubMed Central (PMC), thyroid hormones (T3/T4) are indispensable for maintaining renal hemodynamics, glomerular filtration, and electrolyte balance. The deficiency of these hormones triggers a cascade of pathophysiological changes that lead to both functional decline and structural damage in the kidneys [1, 2].

Pathophysiological Mechanisms. The primary functional impairment in hypothyroidism is characterized by a significant decrease in renal plasma flow (RPF) and cardiac output. This reduction is often mediated by the dysregulation of the Renin-Angiotensin-Aldosterone System (RAAS), where a lack of thyroid hormones leads to decreased renin gene expression and lowered plasma renin activity. Consequently, the Glomerular Filtration Rate (GFR) can decline by as much as 30-40%, presenting clinically as elevated serum creatinine and urea levels [3, 4].

Morphometric and Structural Alterations.Beyond functional decline, prolonged hypothyroidism induces specific morphometric changes in the renal parenchyma. Research indicates that thyroid hormone deficiency leads to:

Renal Atrophy: A measurable reduction in overall kidney mass and volume.

Tubular Degeneration: Atrophy of renal tubule cells, specifically a decrease in cell height

within the thick ascending limb of Henle's loop, and a general reduction in tubular diameters [5].

Histological Damage: Structural alterations often include the thickening of the glomerular and tubular basement membranes, which serves as a precursor to chronic interstitial fibrosis.

The Impact of Immobility (Hypokinesia).A critical, yet often overlooked, factor in this pathological process is the patient's mobility status. In less-mobile or sedentary patients, the negative effects of hypothyroidism are significantly exacerbated. Physical inactivity leads to peripheral venous stasis and reduced skeletal muscle pump activity, which increases renal venous pressure and worsens intrarenal hypoxia. Studies show that subclinical hypothyroidism has a high prevalence (17.9%) in patients with pre-existing renal impairment (GFR <60 ml/min), compared to only 7% in those with normal function. For elderly and less-mobile patients, especially those with comorbidities like diabetes (Diabetic Kidney Disease), these changes accelerate the progression toward irreversible renal failure [6].

Clinical Significance and Reversibility. One of the most vital aspects of managing this condition is the potential for reversibility. Clinical evidence suggests that Thyroid Hormone Replacement Therapy (THRT) with Levothyroxine (T4) can significantly improve mean eGFR and, in many cases, reverse early-stage structural changes. Therefore, identifying early morphometric predictors—such as changes in cortical thickness and the Renal Resistive Index (RRI)—is essential for preventing permanent kidney damage in less-mobile populations.

This study aims to analyze these specific predictors to provide a framework for early intervention and improved clinical outcomes in hypothyroid patients with restricted mobility.

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Predictors of Structural Progression. Recent literature identifies several key predictors for structural changes. Serum Cystatin C More sensitive than creatinine in detecting early GFR decline in hypothyroid patients.

Renal Resistive Index (RRI): An ultrasound-based morphometric marker that increases significantly in sedentary hypothyroid patients, indicating high intrarenal vascular resistance. Albuminuria often correlates with the severity of basement membrane thickening.

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Methodology

Study Design and Patient Selection. A prospective, comparative clinical study was conducted to evaluate the morphometric and functional predictors of renal changes. The study population consisted of 120 patients diagnosed with primary hypothyroidism (TSH > 10 mIU/L). The participants were divided into two main cohorts: Group A (Active Group, n=60). Patients maintain a moderate physical activity level (>7,000 steps per day). Group B (Less-Mobile Group, n=60).Patients with restricted mobility due to comorbid conditions or a sedentary lifestyle (<3,000 steps per day or bedridden for >6 hours/day during daylight). Inclusion Criteria. Confirmed primary hypothyroidism. Age range: 45–75 years. Willingness to undergo repeated ultrasound and biochemical screening. Exclusion Criteria. Pre-existing advanced Stage 4 or 5 Chronic Kidney Disease (CKD). Congenital renal anomalies. Recent use of nephrotoxic drugs. Morphometric Evaluation (Imaging Techniques). To identify structural changes, high-resolution B-mode Ultrasonography (USG) and Color Doppler Imaging were utilized. The following morphometric parameters were measured. Renal Volume and Dimensions: Length, width, and thickness of both kidneys. Cortical Thickness: Measured at the mid-kidney level to assess atrophy. Parenchymal Echogenicity: Graded on a scale of 0 to 3 to identify signs of fibrosis or inflammation. Renal Resistive Index (RRI): Measured via Doppler in the interlobar arteries to assess intracranial vascular resistance, a key predictor of structural damage.

Functional and Biochemical Assessment. Laboratory analyses were performed using

standardized kits in accordance with WHO laboratory manuals. Key functional indicators included. Glomerular Filtration Rate (eGFR): Calculated using the CKD-EPI formula, which is more accurate for assessing renal function in endocrine disorders. Serum Markers: Fasting levels of Thyroid-Stimulating Hormone (TSH), Free Thyroxine (fT4), Serum Creatinine, and Blood Urea Nitrogen (BUN). Urinary Analysis: 24-hour urinary protein excretion and microalbuminuria levels to detect early glomerular basement membrane permeability changes. Hypokinesia Monitoring. To objectively quantify the "less-mobile" status, patients utilized electronic pedometers or accelerometers over 14 days. For bedridden patients, daily activity logs maintained by caregivers were analyzed to calculate the immobility index.

Statistical Analysis. Data were processed using SPSS Statistics (Version 26.0).

Student's t-test was used for comparing means between the active and less-mobile groups. Pearson Correlation Analysis was applied to determine the strength of the relationship between TSH levels, RRI (Resistive Index), and cortical thickness. A p-value < 0.05 was considered statistically significant. CKD-EPI Formula. State-of-the-art formula for calculating kidney function. Renal Resistive Index (RRI). This index is the main predictor of vascular resistance in sedentary patients. Parenchymal Echogenicity: A method of measuring changes in the density of kidney tissue.

Result

General Characteristics of the Study Population. The study included 120 patients (Mean age: 58.4 ± 7.2 years). Baseline analysis showed that both Group A (Active) and Group B (Less-Mobile) had significantly elevated TSH levels (Mean: 14.2 ± 3.8 mIU/L). However, despite similar hormonal profiles at the start, the renal response varied drastically between the two groups due to the mobility factor. Comparative Functional Renal Markers [11]. Functional analysis through the CKD-EPI formula revealed a marked disparity in the Estimated Glomerular Filtration Rate (eGFR). Group A (Active): Mean eGFR was 74.5 ± 5.2 ml/min/1.73m², indicating mild renal impairment. Group B (Less-Mobile): Mean eGFR dropped to 58.2 ± 6.4 ml/min/1.73m² ($p < 0.01$). Observation: Less-mobile patients showed a 21.8% faster decline in filtration capacity compared to active patients with the same degree of hypothyroidism. Serum creatinine levels in Group B were significantly higher (118.4 ± 12.5 μmol/L vs. 94.2 ± 8.6 μmol/L in Group A). Morphometric and Ultrasonographic Predictors. The structural integrity of the kidneys was assessed using high-definition morphometry. The following significant predictors of structural damage were identified. Renal Cortical Thickness (RCT) [12].

In the less-mobile group, the average cortical thickness was 1.21 ± 0.14 cm, compared to 1.58 ± 0.12 cm in active patients. Thinning of the cortex is a strong predictor of irreversible tubular atrophy [13].

Renal Resistive Index (RRI). Doppler imaging showed that the RRI in Group B was 0.74 ± 0.04, while in Group A it remained within the near-normal range of 0.64 ± 0.03. An RRI > 0.70 in less-mobile hypothyroid patients serves as a definitive predictor of high intrarenal vascular resistance and impending fibrosis. Parenchymal Echogenicity. 72% of patients in Group B exhibited Grade II or III echogenicity, suggesting interstitial edema and early-stage fibrosis. In Group A, only 28% showed similar changes. Correlation Analysis: Hypokinesia and Renal Atrophy. A strong negative correlation ($r = -0.68$, $p < 0.001$) was found between the "Daily Step Count" and "Renal Cortical Thickness." This confirms that physical inactivity (hypokinesia) synergistically interacts with thyroid hormone deficiency to accelerate renal tissue degeneration [14].

Discussion

The results demonstrate that hypothyroidism alone induces functional renal changes, but the addition of a less-mobile state triggers structural (morphometric) damage. The increase in RRI suggests that venous congestion and reduced skeletal muscle pump activity lead to intrarenal hypertension, which, combined with low T3/T4 levels, causes the "double-hit" effect on the renal parenchyma. This validates Resistive Index (RRI) and Cortical Thickness as the most reliable morphometric predictors for this specific patient category [15].

Conclusion

The study provides compelling evidence that hypothyroidism, when combined with a less-mobile lifestyle, acts as a significant catalyst for both functional and structural renal deterioration. The primary conclusions are as follows. Synergistic Damage: While hypothyroidism induces functional declines (low eGFR), physical inactivity (hypokinesia) triggers irreversible morphometric changes, such as cortical thinning and tubular atrophy.

Key Predictors identified: The Renal Resistive Index (RRI > 0.70) and Renal Cortical Thickness (RCT < 1.3 cm) have been identified as the most reliable ultrasonographic predictors for early structural kidney damage in less-mobile hypothyroid patients. Hemodynamic Impact: The reduction in the Renin-Angiotensin-Aldosterone System (RAAS) activity, coupled with venous congestion from lack of mobility, creates a "double-hit" effect, accelerating the transition from subclinical renal impairment to overt Chronic Kidney Disease (CKD). Reversibility: Functional changes remain largely reversible with early hormone replacement therapy; however, structural morphometric changes in sedentary patients require longer recovery and targeted physical interventions. Recommendations for Clinical Practice. Based on the results, the following protocol is recommended for managing hypothyroid patients with limited mobility. Mandatory Renal Screening. All hypothyroid patients with a sedentary lifestyle or restricted mobility should undergo annual Renal Doppler Ultrasonography to monitor RRI and cortical thickness. Early Hormone Optimization: Achieving euthyroid status (TSH < 4.0 mIU/L) should be prioritized to restore renal hemodynamics and prevent the onset of fibrosis.

Mobility Interventions: For "less-mobile" patients, even passive physical therapy or "seated exercises" should be integrated into the treatment plan to reduce renal venous congestion and improve intrarenal blood flow.

Biochemical Monitoring: Beyond serum creatinine, the use of Cystatin C is recommended for this group as a more sensitive marker for early filtration decline.

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