



Article

Predictors of Renal Structural Alterations in Hypothyroid Patients The Role of Physical Inactivity and Clinical Parameters

Jakhongirbek Kadirov¹, Mavluda Soliyeva²

1. Assistant, Department of Anatomy and Clinical Anatomy, Andijan State Medical Institute
2. Assistant, Department of Pediatrics and Neonatology, Andijan State Medical Institute

*corresponder: -

Abstract: Background: Hypothyroidism may influence renal physiology through alterations in cardiovascular performance, renal hemodynamics, and glomerular filtration. Identifying clinical characteristics potentially associated with renal structural alterations may contribute to a more comprehensive understanding of kidney involvement in patients with hypothyroidism.

To investigate potential clinical predictors of renal structural alterations in patients with hypothyroidism, with particular attention to physical inactivity and available clinical parameters. The available information establishes the study population and the renal assessment methods. However, patient-level data and statistical estimates required to identify predictors of renal structural alterations have not yet been provided. Therefore, no predictor effects, associations, or significance levels are reported in this draft.

The study provides a basis for examining potential relationships between clinical characteristics and renal morphology in patients with hypothyroidism. Identification of independent predictors requires verified clinical and ultrasound data, a clearly defined physical-activity measure, and an appropriate multivariable statistical analysis.

Keywords: hypothyroidism; renal structural alterations; renal ultrasonography; physical inactivity; clinical predictors; serum creatinine; renal morphometry.

INTRODUCTION

The thyroid gland and kidneys are closely interconnected through endocrine, cardiovascular, and renal physiological mechanisms. Thyroid hormones influence renal development, renal blood flow, glomerular filtration, tubular transport, and the regulation of water and electrolyte balance. Consequently, thyroid dysfunction may be accompanied by changes in renal physiology and biochemical indicators of kidney function. [1]

Hypothyroidism has been associated with changes in renal hemodynamics, including reduced renal blood flow and glomerular filtration. These changes may occur through systemic cardiovascular effects and alterations in intrarenal vascular regulation. Nevertheless, the presence and clinical significance of renal abnormalities can vary according to thyroid status, comorbidities, treatment, and other patient characteristics.

Renal ultrasonography is a non-invasive method for examining kidney dimensions and structural characteristics. It can provide morphometric information that complements biochemical evaluation. Serum creatinine is also commonly used in clinical practice to assess renal function, although it does not independently provide a complete assessment of kidney structure or establish chronic kidney disease stage.[2]

The identification of clinical factors associated with renal structural alterations is important for understanding variation among patients with hypothyroidism. Potentially relevant characteristics may include age, sex, body mass index, blood pressure, diabetes, thyroid disease severity, treatment status, and physical activity. Their relevance in a particular sample, however, must be demonstrated through analysis rather than assumed from general physiological considerations.

Physical inactivity is of particular interest because it may coexist with endocrine and metabolic conditions. To investigate its relationship with renal morphology, physical inactivity must be operationally defined and assessed using documented information. Without a specified instrument or criterion, it cannot be reliably classified as an exposure variable or evaluated as a predictor.[3]

The present study was conducted at Andijan City Central Hospital, Uzbekistan, between 1 March and 1 September 2026. Of the 245 patients examined, 156 were diagnosed with hypothyroidism. Renal ultrasonographic data and serum creatinine measurements were available for these patients. The other 89 patients were not diagnosed with hypothyroidism but were not designated as a control group.

The objective of this study was to investigate potential predictors of renal structural alterations in patients with hypothyroidism, focusing on physical inactivity and clinical parameters documented in the study.

Materials and Methods. This clinical and diagnostic study was conducted at Andijan City Central Hospital, Andijan, Uzbekistan, from 1 March to 1 September 2026.

Study Population. A total of 245 patients were examined during the study period. Hypothyroidism was diagnosed in 156 patients (63.7%). In the remaining 89 patients (36.3%), hypothyroidism was not diagnosed. These patients were not classified as a control group.

Renal ultrasonographic data and serum creatinine measurements were available for the 156 patients diagnosed with hypothyroidism. The proportion of patients with hypothyroidism refers to the examined clinical sample and should not be interpreted as a population prevalence estimate.

Classification of Hypothyroidism. Patients were classified according to the documented diagnosis of hypothyroidism. The diagnostic criteria, laboratory thresholds, and classification into overt or subclinical hypothyroidism should be reported in accordance with the original clinical records.

Assessment of Physical Inactivity. Physical inactivity was considered a potentially relevant clinical characteristic. However, the specific method used to assess physical activity and the threshold used to classify patients as sedentary have not been provided.

The final analysis should specify whether physical activity was evaluated through a validated questionnaire, structured interview, clinical documentation, or another method. If no formal assessment was conducted, physical inactivity should not be treated as a measured predictor.

Renal Structural Assessment. Renal ultrasonographic data were available for the 156 patients diagnosed with hypothyroidism. The recorded measurements should be used to define the renal structural outcomes investigated in the study.

Depending on the parameters actually collected, these may include kidney length, width, anteroposterior diameter, parenchymal thickness, cortical thickness, renal volume, or other documented ultrasound characteristics. Only parameters present in the original dataset should be included.

The ultrasound equipment, examination protocol, measurement units, and criteria used to classify structural alterations should be specified from the study documentation.

Biochemical Assessment. Serum creatinine was the available biochemical indicator of renal function in the hypothyroid group. The laboratory method, measurement units, reference interval, and numerical results should be reported from the clinical records. Estimated glomerular filtration rate (eGFR) was not included because no eGFR data were provided.

Candidate Clinical Predictors. Potential predictors should be selected according to the variables actually recorded. Candidate characteristics may include demographic, clinical, thyroid-related, and lifestyle variables, provided that they are available and sufficiently complete in the dataset. The final manuscript should distinguish between variables selected in advance and variables explored after reviewing the data. No predictor should be included merely

Statistical Analysis. The analysis should begin with descriptive statistics for the study population and renal ultrasound outcomes. Associations between candidate predictors and renal structural parameters may then be evaluated using statistical methods appropriate to the outcome type and data distribution.

If the sample size and data quality permit, multivariable regression may be considered to estimate adjusted associations. The model should be specified according to the actual outcome, the number of observations, the distribution of the measurements, and the availability of covariates. Potential collinearity and missing data should also be assessed. The statistical software, tests, model specifications, effect estimates, confidence intervals, and significance threshold must be reported after the analysis has been completed. No statistical results are claimed in this draft.[4]

Ethical Considerations. The final manuscript should identify the approving ethics committee, approval number and date, informed-consent procedure, and participant confidentiality measures, using the original study documentation.

Materials and Methods: This clinical and diagnostic study was conducted at Andijan City Central Hospital, Andijan, Uzbekistan, from 1 March to 1 September 2026. A total of 245 patients were examined, and hypothyroidism was diagnosed in 156 (63.7%). The remaining 89 patients had no documented diagnosis of hypothyroidism and were not designated as a control group. Renal ultrasonographic data and serum creatinine measurements were available for the 156 patients diagnosed with hypothyroidism. The operational definition of physical inactivity, the complete set of clinical covariates, and the numerical renal measurements require confirmation from the original study records.[5]

RESULTS

Characteristics of the Examined Population. A total of 245 patients were examined between 1 March and 1 September 2026. Hypothyroidism was diagnosed in 156 patients (63.7%), while 89 patients (36.3%) had no documented diagnosis of hypothyroidism. The 89 patients without a diagnosis of hypothyroidism were not designated as a control group. Accordingly, the available information does not establish a healthy comparison group.

Figure 1. Renal Ultrasound Findings. Renal ultrasonographic data were available for the 156 patients diagnosed with hypothyroidism. The specific measurements and the number of patients meeting any predefined criteria for structural alterations have not been provided. The results should report the actual measurements and their distributions. Any classification of structural alterations must follow the study's documented ultrasound criteria.[6]

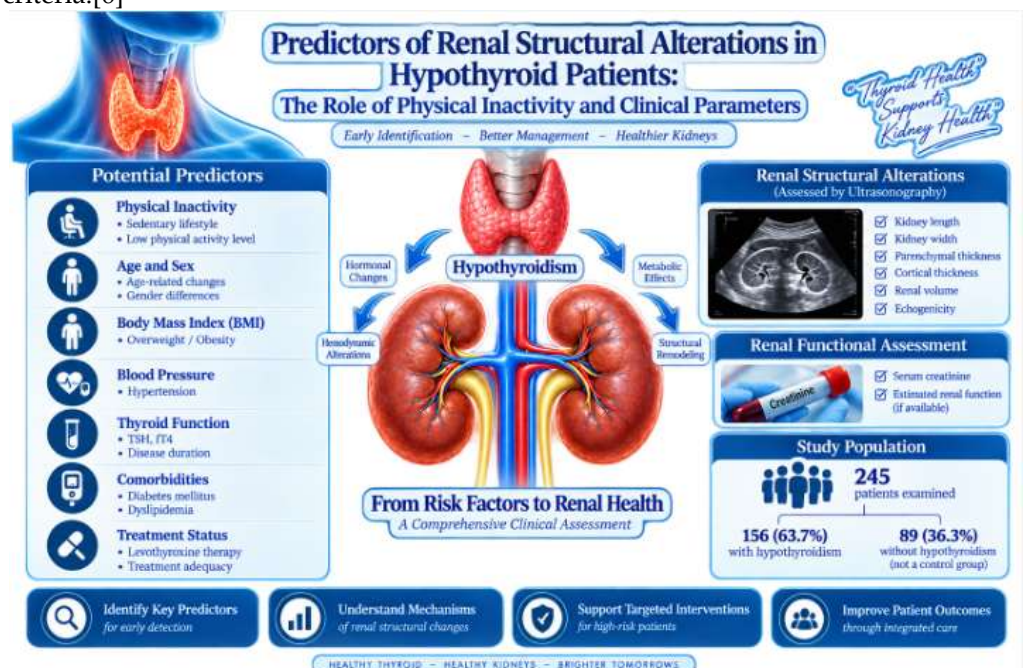


Figure 1. Conceptual Framework of Potential Predictors of Renal Structural Alterations in Hypothyroid Patients[7]

This figure presents a conceptual framework illustrating the potential relationships between hypothyroidism, physical inactivity, and renal structural alterations. It identifies relevant clinical and demographic parameters, including age, sex, body mass index, blood pressure, thyroid function, comorbidities, and treatment status, that may be

associated with renal changes. Renal structure is assessed using ultrasonographic parameters, while renal function is evaluated using serum creatinine and estimated glomerular filtration rate, when available. The framework is intended to guide the clinical assessment of hypothyroid patients and the investigation of factors potentially associated with renal structural alterations. The relationships shown are conceptual and do not represent statistically confirmed predictors or causal effects.[8]

Serum Creatinine. Serum creatinine measurements were available for the hypothyroid group. The numerical values, units, reference interval, and distribution of results have not been provided. Consequently, no conclusion can currently be drawn about the frequency or magnitude of elevated serum creatinine in this group.

Analysis of Potential Predictors. The available information does not include the patient-level dataset or statistical estimates needed to evaluate relationships between clinical characteristics, physical inactivity, and renal structural outcomes.[9]

Therefore, independent predictors, adjusted associations, confidence intervals, and p-values cannot be reported in this draft. These results should be inserted only after analysis of the verified data.[10]

DISCUSSION

This study focuses on potential clinical predictors of renal structural alterations in patients diagnosed with hypothyroidism. The available information establishes a clinical sample of 156 hypothyroid patients with renal ultrasonographic data and serum creatinine measurements.[11]

The investigation is supported by the established physiological relationship between thyroid hormones and renal function. Published reviews describe the effects of thyroid dysfunction on renal hemodynamics, glomerular filtration, tubular processes, and other aspects of kidney physiology. These mechanisms provide a rationale for investigating renal characteristics in hypothyroidism, but they do not establish the presence of specific structural abnormalities in the present sample.

Renal ultrasonography offers a means of describing kidney morphology, but the interpretation of any abnormality depends on the measurements obtained, the criteria applied, and relevant clinical characteristics. Without numerical ultrasound findings, it is not possible to determine whether structural alterations were common, uncommon, or associated with particular patient characteristics in this study.[12]

Serum creatinine provides a biochemical measure relevant to renal function. However, serum creatinine alone is insufficient to characterize all aspects of renal function or to establish chronic kidney disease stage. Its interpretation should be restricted to the actual values and laboratory information available.

The potential role of physical inactivity requires particular methodological care. A sedentary lifestyle should be treated as a predictor only if it was assessed using a defined and documented method. If such information is unavailable, the relationship between physical inactivity and renal structural alterations cannot be tested reliably.

The identification of independent predictors also requires consideration of confounding. Demographic characteristics, comorbidities, thyroid disease severity, and treatment status may influence the observed relationships, if these variables were recorded. A multivariable model should therefore be guided by the study design, clinical rationale, data completeness, and sample size rather than by an arbitrary list of variables.[13]

An additional limitation is that the 89 patients without a diagnosis of hypothyroidism were not classified as a control group. Their clinical status should not be assumed to represent a healthy population, and they should not be used as healthy controls without evidence supporting that classification.[14]

The principal limitation of the present manuscript draft is the absence of numerical renal ultrasound measurements, serum creatinine values, and patient-level covariate data. The definition and measurement of physical inactivity, statistical procedures, and ethics documentation also require confirmation. These limitations prevent the identification of predictors or the drawing of conclusions about the strength and direction of associations.[15]

CONCLUSION

This clinical and diagnostic study examined 245 patients at Andijan City Central Hospital between 1 March and 1 September 2026. Hypothyroidism was diagnosed in 156 patients (63.7%). Renal ultrasonographic data and serum creatinine measurements were available for the hypothyroid group. The remaining 89 patients were not diagnosed with hypothyroidism but were not designated as a control group.

The available information establishes the study population and the principal renal assessment methods. However, identification of predictors of renal structural alterations requires the actual ultrasound measurements, serum creatinine results, documented clinical covariates, and a clearly defined assessment of physical inactivity.

Accordingly, no independent predictor or causal relationship can be concluded from the currently available information. Further interpretation should be based on verified clinical data and an appropriate statistical analysis.

REFERENCES

- [1] P. Iglesias, M. A. Bajo, R. Selgas, and J. J. Díez, "Thyroid dysfunction and kidney disease: An update," *Reviews in Endocrine and Metabolic Disorders*, vol. 18, no. 1, pp. 131–144, 2017, doi: 10.1007/s11154-016-9395-7.
- [2] A. I. Katz, D. S. Emmanouel, and M. D. Lindheimer, "Thyroid hormone and the kidney," *Nephron*, vol. 15, nos. 3–5, pp. 223–249, 1975, doi: 10.1159/000180514.
- [3] C. M. Rhee, "The interaction between thyroid and kidney disease: An overview of the evidence," *Current Opinion in Endocrinology, Diabetes and Obesity*, vol. 23, no. 5, pp. 407–415, 2016, doi: 10.1097/MED.0000000000000275.
- [4] X. Wang, X. Shang, Y. Li, C. Lu, Y. Shao, D. Yang, W. Teng, and X. Shi, "Association between hypothyroidism, levothyroxine replacement and kidney function outcomes: A systematic review and meta-analysis," *Frontiers in Endocrinology*, vol. 17, Art. no. 1841255, 2026, doi: 10.3389/fendo.2026.1841255.
- [5] C. M. Rhee and Y. Narasaki, "Hormones and nephrons: Disentangling the thyroid-kidney connection," *Clinical Journal of the American Society of Nephrology*, vol. 20, no. 7, pp. 905–908, 2025, doi: 10.2215/CJN.00000000761.
- [6] P. Dousdampanis, K. Trigka, G. A. Vagenakis, and C. Fourtounas, "The thyroid and the kidney: A complex interplay in health and disease," *International Journal of Artificial Organs*, vol. 37, no. 1, pp. 1–12, 2014, doi: 10.5301/ijao.5000300.
- [7] C. Ellervik, S. Mora, P. M. Ridker, D. I. Chasman, et al., "Hypothyroidism and kidney function: A Mendelian randomization study," *Thyroid*, vol. 30, no. 3, pp. 365–379, 2020, doi: 10.1089/thy.2019.0167.
- [8] Kidney Disease: Improving Global Outcomes KDIGO CKD Work Group, "KDIGO 2024 clinical practice guideline for the evaluation and management of chronic kidney disease," *Kidney International*, vol. 105, no. 4S, pp. S117–S314, 2024, doi: 10.1016/j.kint.2023.10.018.
- [9] B. B. Rabiev, "Women's migration and childhood illness social determinants of health," *Journal of Family Medicine and Health Care*, vol. 12, no. 2, pp. 27–32, 2026, doi: 10.11648/j.jfmhc.20261202.11.
- [10] C. P. Kovesdy, "Epidemiology of chronic kidney disease: An update 2022," *Kidney International Supplements*, vol. 12, no. 1, pp. 7–11, 2022, doi: 10.1016/j.kisu.2021.11.003.
- [11] L. H. Mariani and J. S. Berns, "The renal manifestations of thyroid disease," *Journal of the American Society of Nephrology*, vol. 23, no. 1, pp. 22–26, 2012, doi: 10.1681/ASN.2010070766.
- [12] U. T. Schultheiss, N. Daya, M. E. Grams, J. Seufert, M. Steffes, J. Coresh, E. Selvin, and A. Köttgen, "Thyroid function, reduced kidney function and incident chronic kidney disease in a community-based population: The Atherosclerosis Risk in Communities study," *Nephrology Dialysis Transplantation*, vol. 32, no. 11, pp. 1874–1881, 2017, doi: 10.1093/ndt/gfw301.
- [13] I. Al Fahdi, I. Al Salmi, F. Al Rahbi, F. Shaheen, and S. Hannawi, "Thyroid dysfunction and kidney dysfunction," *Oman Medical Journal*, vol. 37, no. 3, Art. no. e377, 2022, doi: 10.5001/omj.2022.55.
- [14] A. Levin, S. B. Ahmed, J. J. Carrero, et al., "Executive summary of the KDIGO 2024 Clinical Practice Guideline for the Evaluation and Management of Chronic Kidney Disease: Known knowns and known unknowns," *Kidney International*, vol. 105, no. 4, pp. 684–701, 2024, doi: 10.1016/j.kint.2023.10.016.
- [15] C. M. Rhee, "The interaction between thyroid and kidney disease: An overview of the evidence," *Current Opinion in Endocrinology, Diabetes and Obesity*, vol. 23, no. 5, pp. 407–415, 2016, doi: 10.1097/MED.0000000000000275.