Reversible Renal Impairment Due to Hypothyroidism: A Case Report on the Role of Thyroid Hormone Replacement Therapy

Srivalsa Bhaskaran [1], Kamaal Mohideen Khan [1], Karthik V [1], Mani Shanthini [1*]

Abstract

Background: Renal impairment is frequently linked to hypothyroidism, a condition characterized by insufficient thyroid hormone production. Thyroid hormones, particularly triiodothyronine (T3) and thyroxine (T4), are crucial in regulating renal function by influencing serum creatinine levels and glomerular filtration rate (GFR). The multifaceted interaction between hypothyroidism and renal function necessitates careful assessment and management to prevent or reverse renal impairment. Methods: A 60-year-old male presented with symptoms of lethargy and muscle aches. Laboratory tests revealed elevated serum creatinine levels (2.7 mg/dL), reduced GFR (27 mL/min/1.73 m²), and a thyroid-stimulating hormone (TSH) level of 400 mIU/L. A 99mTcDTPA renal scan showed compromised cortical function in both kidneys. The patient was diagnosed with hypothyroidism and initiated on levothyroxine therapy at 100 micrograms daily. Results: After two months of thyroid hormone replacement therapy (THRT), the patient's renal function improved significantly, with normalized serum creatinine (0.87 mg/dL) and increased GFR (87 mL/min/1.73 m²). By six months, the patient remained asymptomatic, with stable thyroid function and improved renal parameters. Conclusion: This case highlights the significant impact of hypothyroidism on renal function, demonstrating that appropriate THRT can lead to substantial recovery in renal impairment. Routine evaluation of thyroid function should be considered in patients with unexplained renal dysfunction to identify and manage potentially reversible causes.

Keywords: Hypothyroidism, Renal Impairment, Thyroid Hormone Replacement Therapy (THRT), Glomerular Filtration Rate (GFR), Serum Creatinine

Significance | This case showed the reversible nature of renal impairment in hypothyroidism, emphasizing the importance of timely thyroid hormone replacement therapy.

*Correspondence. Mani Shanthini, Department of Microbiology, Sree Balaji Medical College and Hospital, Bharath Institute of Higher Education and Research (BIHER), Chennai, Tamil Nadu, India. E-mail: mani.s@bharathuniv.ac.in

Editor Hussein Mahfoudh Saeed Baharetha, Ph.D., And accepted by the Editorial Board 20 December 2021 (received for review 29 November 2021)

Introduction

Renal impairment is a prevalent clinical concern, and its association with thyroid dysfunction, particularly hypothyroidism, is increasingly recognized. Thyroid hormones play a crucial role in regulating various physiological processes, including renal function. The hormones triiodothyronine (T3) and thyroxine (T4) are known to influence serum creatinine levels and glomerular filtration rate (GFR), thereby affecting renal function. Hypothyroidism, a common endocrine disorder characterized by insufficient thyroid hormone production, has been identified as a potential cause of renal impairment. Notably, the pathophysiology of renal dysfunction in hypothyroidism is multifaceted, involving both direct and indirect effects of thyroid hormones on renal and cardiovascular systems (Montenegro et al., 1996; Suher et al., 2005). In hypothyroidism, renal impairment can occur due to a direct effect of thyroid hormones on kidney function as well as through

Author Affiliation.
[1] Department of Microbiology, Sree Balaji Medical College and Hospital, Bharath Institute of Higher Education and Research (BIHER), Chennai, Tamil Nadu, India.

Please Cite This:
Srivalsa Bhaskaran, Kamaal Mohideen Khan et al. (2021). Reversible Renal Impairment Due to Hypothyroidism: A Case Report on the Role of Thyroid Hormone Replacement Therapy, Journal of Angiotherapy, 5(2), 1-5, 2161

hemodynamic changes. Thyroid hormones are involved in renal growth and development, renal hemodynamics, and the maintenance of sodium and water balance. A reduction in thyroid hormone levels can lead to decreased cardiac output and renal blood flow, resulting in diminished GFR and increased serum creatinine levels. This phenomenon is particularly notable as it can often be reversible with appropriate thyroid hormone replacement therapy (THRT) (Vikrant et al., 2013).

Despite the potential for reversible renal impairment, hypothyroidism often goes unrecognized as a cause of renal dysfunction. Clinical manifestations may be subtle or absent, even in the presence of significant thyroid hormone deficiency. Therefore, patients with elevated serum creatinine levels and unexplained renal impairment should be evaluated for thyroid function. Accurate assessment often requires additional diagnostic tools such as isotope GFR studies to differentiate between true renal impairment and increased creatinine production or tubular secretion.

The significance of this connection underscores the necessity of considering thyroid function tests in the diagnostic workup of renal impairment, particularly when the etiology remains unclear. This case report aims to illustrate the impact of hypothyroidism on renal function, highlighting the potential for recovery with appropriate THRT. By documenting a case of reversible renal impairment secondary to hypothyroidism, this report emphasizes the importance of addressing thyroid dysfunction in patients with renal issues and the effectiveness of levothyroxine therapy in restoring normal renal function.

Case Report

A 60-year-old male presented with symptoms of lethargy and muscle aches but did not report any urinary complaints. Physical examination revealed bradycardia with a heart rate of 56 beats per minute, normal blood pressure at 130/80 mmHg, pallor, dry skin, periorbital puffiness, non-pitting edema, and slow-relaxing ankle reflexes. Notably, the thyroid gland was not palpable, and the abdominal examination did not reveal any significant findings.

Laboratory investigations showed the following abnormalities: hemoglobin was 8 g/dL, indicative of microcytic hypochromic anemia; serum urea was elevated at 65 mg/dL; creatinine levels were 2.7 mg/dL; and the estimated Glomerular Filtration Rate (eGFR), calculated using the Cockcroft–Gault equation, was 27 mL/min/1.73 m². Electrolyte levels included sodium at 137 mEq/L, potassium at 4.1 mEq/L, and chloride at 101 mEq/L. Calcium was 8.7 mg/dL, phosphorus was 3.1 mg/dL, and uric acid was 6.2 mg/dL. Other relevant values included total protein at 7.1 g/dL, albumin at 4.2 g/dL, bilirubin at 0.7 mg/dL, aspartate aminotransferase (AST) at 80 IU/L, alanine aminotransferase (ALT) at 30 IU/L, alkaline phosphatase at 77 U/L, random blood sugar at 90 mg/dL, cholesterol at 422 mg/dL, triglycerides at 757 mg/dL, and serum creatine phosphokinase at 271 U/L.

Urinalysis showed 6–8 pus cells with a sterile culture. Urinary myoglobin was absent, and 24-hour urine protein levels were 80 mg. Ultrasound evaluation revealed normal-sized kidneys (left kidney: 9.7 x 3.7 cm; right kidney: 9 x 3.6 cm) with patent renal arteries on both sides.

A 99mTcDTPA renal scan demonstrated severely compromised cortical function in both kidneys, though clearance was adequate. The patient was diagnosed with hypothyroidism based on the following thyroid profile: thyroid-stimulating hormone (TSH) was elevated at 400 mIU/L, free T3 was 1.8 pg/mL, free T4 (FT4) was 0.87 ng/dL, and anti-thyroid peroxidase antibodies were markedly elevated at 4550 IU/mL. The patient was initiated on levothyroxine at a dose of 100 micrograms daily and advised to return for follow-up.

Follow-Up and Outcome

At the 2-month follow-up, the patient's serum creatinine levels had normalized. By the 6-month follow-up, the patient was asymptomatic and showed significant improvements: hemoglobin increased to 12 g/dL, serum urea decreased to 24 mg/dL, creatinine reduced to 0.87 mg/dL, and eGFR improved to 87 mL/min/1.73 m², as calculated by the Cockcroft–Gault equation. Serum cholesterol levels were reduced to 165 mg/dL, triglycerides decreased to 154 mg/dL, and thyroid function normalized with TSH at 3 mIU/L and FT4 at 1.0 ng/dL. A repeat 99mTcDTPA renal scan indicated a mild compromise in cortical function but showed adequate clearance in both kidneys, demonstrating a remarkable recovery of renal function following thyroid hormone replacement therapy (THRT).

Discussion

The case presented highlights a significant interaction between hypothyroidism and renal function. Initial renal impairment prompted a thorough investigation, which ultimately revealed hypothyroidism as the underlying cause. Thyroid hormone replacement therapy (THRT) led to a remarkable recovery of renal function, underscoring the importance of considering thyroid dysfunction in patients with renal abnormalities.

In primary hypothyroidism, elevations in serum creatinine are often observed, and this elevation is usually reversible upon appropriate treatment (Davis et al., 1983; Steiger et al., 1991). Hypothyroidism affects renal function through several mechanisms, including impaired renal growth and development, altered renal hemodynamics, and disturbances in sodium and water homeostasis. Thyroid hormones play a crucial role in regulating glomerular filtration rate (GFR) and renal blood flow (RBF). A decline in thyroid hormone levels, rather than thyroid

autoimmunity itself, is more commonly associated with kidney dysfunction (Kreisman & Hennessey, 1999).

The pathophysiology of renal impairment in hypothyroidism is multifaceted. Thyroid hormones directly influence the cardiovascular system, which in turn affects cardiac output and renal blood flow. Reduced cardiac output leads to decreased GFR and contributes to hyperlipidemia. Indirect effects through paracrine signaling further exacerbate renal dysfunction. Restoration of thyroid hormone levels through levothyroxine therapy often results in the normalization of GFR and RBF. For patients with chronic kidney disease (CKD), THRT has been shown to improve eGFR significantly (Nakahama et al., 2001).

Although hypothyroid myopathy, which can lead to myalgia and rhabdomyolysis, occasionally causes acute kidney injury, it is a rare complication (Leonetti et al., 1992; Sekine et al., 1993). Overall, THRT for primary hypothyroidism has proven effective in enhancing renal function in CKD patients.

Conclusion

This case underscores the necessity of considering thyroid function in patients with unexplained renal impairment. Routine thyroid function tests should be included in the diagnostic workup for renal dysfunction of unknown origin. For patients with known chronic kidney disease, evaluating thyroid function and administering appropriate THRT can correct reversible renal impairment caused by hypothyroidism. Recognizing and treating hypothyroidism in such cases can lead to significant improvements in renal function and overall patient health.

Ethics

The study adhered to ethical guidelines outlined by the Declaration of Helsinki and received full approval from the institutional review board (IRB) of Bharath Institute of Higher Education and Research prior to commencement. Written informed consent was obtained from the patient for the collection, use, and analysis of his medical data, and his confidentiality was strictly maintained throughout the study. Identifiable information was anonymized to ensure privacy. The study involved no experimental interventions; all treatments followed standard medical care guidelines. The patient voluntarily participated in the study and was informed of his right to withdraw at any point without impacting his medical care.

Author contributions

S.B., K.M.K. K.V., M.S. played integral roles in the development of this manuscript. Each author actively participated in the discussion of results and contributed significantly to the preparation and refinement of the final manuscript.

Acknowledgment

Author was grateful to their department.

Competing financial interests

The authors have no conflict of interest.

References

Davis, R. G., Madsen, K. M., & Fregly, M. J. (1983). Kidney structure in hypothyroidism. American Journal of Pathology, 113(1), 41–49. https://doi.org/10.1016/S0002-9440(19)80083-7

Derubertis, F. R., Michelis, M. F., Bloom, M. E., & et al. (1971). Impaired water excretion in myxedema. American Journal of Medicine, 51(1), 41–53.

Kreisman, S. H., & Hennessey, J. (1999). Consistent reversible elevations of serum creatinine levels in severe hypothyroidism. Archives of Internal Medicine, 159(1), 79–82. https://doi.org/10.1001/archinte.159.1.79

Leonetti, F., Dussol, B., & Berland, Y. (1992). Rhabdomyolysis and renal failure in hypothyroidism. Presse Médicale, 21(1), 31–32. https://doi.org/10.1016/S0755-4982(05)80409-5

Montenegro, J., González, O., Saracho, R., Aguirre, R., González, Ó., & Martínez, I. (1996). Changes in renal function in primary hypothyroidism. American Journal of kidney diseases, 27(2), 195-198. https://doi.org/10.1016/S0272-6386(96)90539-9

Nakahama, H., Sakaguchi, K., Horita, Y., & et al. (2001). Treatment of severe hypothyroidism reduced serum creatinine levels in two chronic renal failure patients. Nephron, 88(3), 264–267. https://doi.org/10.1159/000046283

Sekine, N., Yamamoto, M., Michikawa, M., & et al. (1993). Rhabdomyolysis and acute renal failure in a patient with hypothyroidism. Internal Medicine, 32(3), 269–271. https://doi.org/10.2169/internalmedicine.32.269

Steiger, M., Watson, A., & Morgan, A. (1991). Hypothyroidism and renal impairment. Journal of the Royal Society of Medicine, 84(11), 688–689. https://doi.org/10.1177/014107689108401121

Suher, M., Koc, E., Ata, N., & Ensari, C. (2005). Relation of thyroid disfunction, thyroid autoantibodies, and renal function. Renal failure, 27(6), 739-742. https://doi.org/10.1080/08860220500243338

Vikrant, S., Chander, S., Kumar, S., & Gupta, D. (2013). Hypothyroidism presenting as reversible renal impairment: an interesting case report. Renal failure, 35(9), 1292-1294. https://doi.org/10.3109/0886022X.2013.824381