

Thyroid Hormone Replacement in an Older Adult with Subclinical Hypothyroidism: A Clinical Case Study

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Abstract

Subclinical hypothyroidism is frequently identified in older adults and is characterized by an elevated serum thyroid-stimulating hormone (TSH) concentration with circulating free thyroxine (FT4) levels remaining within the reference range. Although often clinically subtle, thyroid dysfunction may influence renal blood flow, vascular resistance, cardiac output, and glomerular filtration. The clinical significance of treating subclinical hypothyroidism in elderly individuals with impaired renal function remains uncertain, particularly because of concerns regarding overtreatment and cardiovascular complications.

Following clinical assessment, low-dose levothyroxine therapy was initiated at [DOSE] µg/day and subsequently adjusted according to serum TSH concentrations, clinical response, and treatment tolerance. Serial evaluation demonstrated progressive normalization of thyroid function accompanied by stabilization/improvement of renal function. At [6] months, eGFR increased.

Keywords: Subclinical hypothyroidism; Levothyroxine; Estimated glomerular filtration rate; Chronic kidney disease; Elderly; Thyroid hormone replacement.

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Introduction

Subclinical hypothyroidism is a biochemical thyroid disorder characterized by persistent elevation of serum thyroid-stimulating hormone while circulating free thyroxine remains within the normal reference range. The condition becomes increasingly common with advancing age and is frequently identified during routine laboratory evaluation of older adults [1].

The clinical significance of subclinical hypothyroidism in elderly patients remains a subject of considerable discussion. Some individuals remain asymptomatic, whereas others present with nonspecific manifestations or coexistence of cardiovascular, metabolic, or renal disease. Decisions regarding thyroid hormone replacement therefore require consideration of biochemical abnormalities, age, symptoms, cardiovascular risk, comorbid

conditions, and the potential consequences of treatment [2].

An important but sometimes overlooked relationship exists between thyroid function and renal physiology. Thyroid hormones influence cardiac output, systemic vascular resistance, renal blood flow, sodium handling, and glomerular filtration. Reduced thyroid hormone activity may consequently contribute to decreased renal perfusion and alterations in serum creatinine and estimated glomerular filtration rate.

The interaction is bidirectional because impaired renal function may itself affect thyroid hormone metabolism, protein binding, clearance, and peripheral conversion. These physiological connections make interpretation of thyroid and renal abnormalities particularly challenging in older patients with multiple chronic disorders [3-5].

Previous investigations have demonstrated associations between hypothyroidism and reductions in renal function. Improvements in renal parameters after correction of thyroid dysfunction have also been reported [6]. However, the potential benefit of levothyroxine therapy in older patients with mild or subclinical thyroid dysfunction requires careful evaluation because excessive thyroid hormone replacement may increase the risk of tachyarrhythmias, myocardial ischemia, and other complications.

This case describes the clinical course of an older adult with subclinical hypothyroidism and reduced renal function who underwent carefully monitored levothyroxine replacement. The case highlights the temporal relationship between improvement in thyroid status and changes in renal function and discusses the clinical considerations surrounding treatment in elderly patients.

Case Presentation

This case demonstrates the clinical course of an older adult with subclinical hypothyroidism and impaired renal function following initiation of thyroid hormone replacement therapy. Improvement or stabilization of eGFR following normalization of thyroid status raises the possibility that thyroid dysfunction contributed, at least partly, to the patient's renal impairment [7].

Thyroid hormones exert several physiological effects relevant to kidney function. They influence cardiac output, systemic vascular resistance, renal vascular tone, intrarenal hemodynamics, tubular function, and sodium and water homeostasis. Reduced thyroid hormone activity may therefore decrease renal blood flow and glomerular filtration.

Hypothyroidism has also been associated with elevations in serum creatinine. In some patients, these abnormalities may be at least partially reversible following restoration of euthyroidism. Hollander et al. demonstrated an association between the severity of thyroid dysfunction and renal function, while Kreisman and Hennessey reported reversible increases in serum creatinine in patients with severe hypothyroidism.

The relationship may also be clinically relevant in patients with chronic kidney disease. Shin et al. reported that thyroid hormone replacement was associated with preservation of renal function among patients with chronic kidney disease and subclinical hypothyroidism. Subsequent investigation by the same group suggested that thyroid hormone replacement could attenuate the decline in renal function in this population [8].

The present case is clinically relevant because treatment decisions in elderly patients with subclinical hypothyroidism can be difficult. Older adults frequently have hypertension, diabetes mellitus, chronic kidney disease, ischemic heart disease, arrhythmias, and other conditions that may simultaneously influence renal function and increase the risks associated with thyroid hormone therapy (Figure 1).

Consequently, thyroid hormone replacement should not be initiated solely on the assumption that it will improve kidney function. Treatment decisions should incorporate the degree and

persistence of TSH elevation, thyroid hormone concentrations, clinical manifestations, cardiovascular status, renal function, concurrent medications, and overall clinical condition [9].

Another important consideration is the interpretation of serum creatinine-derived eGFR in patients with thyroid dysfunction. Changes in thyroid status may influence renal hemodynamics as well as creatinine production and metabolism. An apparent improvement in creatinine-based eGFR after treatment therefore needs to be interpreted within the broader clinical context rather than regarded as definitive evidence of structural renal recovery.

In the current case, the temporal relationship between normalization of thyroid function and improvement or stabilization of renal parameters suggests a potential beneficial association. Nevertheless, causality cannot be established from an individual case. Other factors, including blood pressure control, diabetes management, medication changes, hydration, dietary factors, and the natural variability of serum creatinine, may contribute to observed changes in eGFR [10,11].

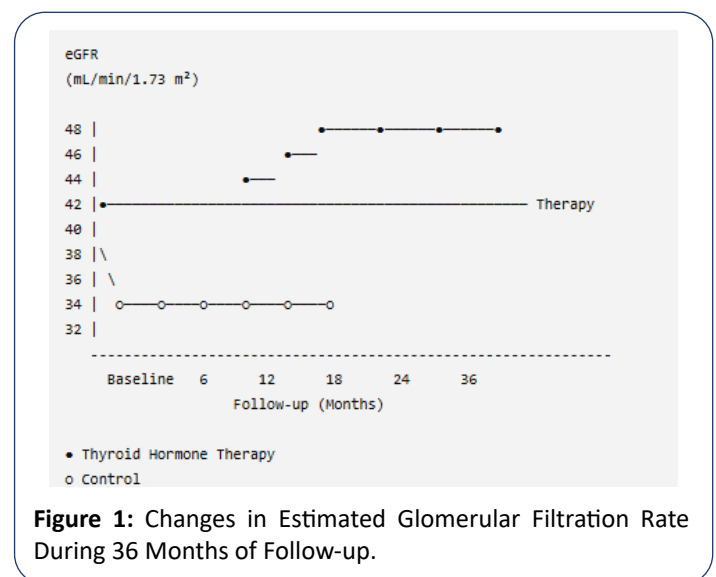


Figure 1: Changes in Estimated Glomerular Filtration Rate During 36 Months of Follow-up.

The case also emphasizes the importance of conservative dosing in elderly patients. Excess thyroid hormone exposure may increase the risk of palpitations, tachyarrhythmias, myocardial ischemia, and loss of bone mineral density. Treatment should therefore begin cautiously, particularly in individuals with cardiovascular disease, and dosage adjustments should be guided by serial biochemical evaluation and clinical response.

Long-term monitoring remains essential. Serial assessment of TSH and FT4 should be accompanied by evaluation of renal function, cardiovascular status, medication tolerance, and relevant comorbid conditions [12].

Clinical Significance

The coexistence of subclinical hypothyroidism and renal impairment in an older patient presents a diagnostic and therapeutic challenge. Abnormal renal parameters may not necessarily represent irreversible progression of chronic kidney

disease, because thyroid dysfunction can influence both renal hemodynamics and biochemical markers used to estimate kidney function.

Recognition and appropriate management of thyroid dysfunction may therefore be clinically relevant when unexplained or progressive reductions in eGFR occur in an elderly patient.

This case further demonstrates why treatment decisions should remain individualized. Levothyroxine therapy may be considered when clinical circumstances support treatment, but careful dosing and monitoring are particularly important in older individuals.

Interpretation of this case is limited by its single-patient nature. A temporal association between thyroid hormone replacement and improvement in renal function cannot establish a direct causal relationship.

Serum creatinine-derived eGFR is also influenced by age, muscle mass, nutritional status, medication use, and other physiological factors. Additional renal biomarkers or directly measured GFR would provide stronger evidence regarding true changes in filtration capacity.

Furthermore, multiple comorbidities commonly present in elderly individuals may independently influence kidney function.

Accordingly, the findings from this case should not be generalized to all older adults with subclinical hypothyroidism.

Conclusion

This clinical case highlights a potentially important relationship between thyroid function and renal physiology in an older adult with subclinical hypothyroidism and impaired kidney function. Following carefully monitored levothyroxine replacement, normalization of thyroid status was accompanied by stabilization or improvement in estimated glomerular filtration rate.

The observation suggests that thyroid dysfunction should be considered as a potentially modifiable contributor when renal impairment is identified in older patients with persistent subclinical hypothyroidism.

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