



The relationship between thyroid functions and blood parameters in patients with renal failure

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Abstract

Renal function is significantly impacted by both hypothyroidism and hyperthyroidism. Despite this, there is a lack of clinical research examining the relationship between thyroid dysfunction and renal function. This study aimed to assess alterations in biochemical indicators of renal function in individuals with thyroid dysfunction and to establish a connection between these values and the patient's thyroid profile. Additionally, the impact of changes in thyroid function on renal function during treatment was explored. Various interactions exist between the functions of the thyroid and kidneys in the presence of disease. Thyroid hormones have a significant impact on the development and function of the kidneys, causing changes in renal blood flow and glomerular filtration rate (GFR) both before and within the kidneys. A decrease in GFR is seen in individuals with hypothyroidism, while those with hyperthyroidism experience an increase in GFR, as well as heightened activation of the renin-angiotensin-aldosterone system. In the context of chronic kidney disease (CKD), a medical phenomenon known as low T3 syndrome has been acknowledged as a distinct presentation of nonthyroidal illness. It has been observed that individuals with chronic kidney disease frequently exhibit a higher incidence of primary hypothyroidism and subclinical hypothyroidism. The potential advantages of a hypothyroid condition in chronic kidney disease, coupled with the increased risk of disease advancement with hyperthyroidism, underscore the importance of a cautious strategy when managing thyroid hormone irregularities in this patient group. Thyroid dysfunction is commonly associated with glomerulonephritis, a condition often attributed to an autoimmune etiology. Specific medications can affect the functions of both the thyroid and kidneys. Although interactions involving thyroid and renal malignancies have been reported minimally, it is imperative for nephrologists and endocrinologists to possess a thorough understanding of these connections in order to provide optimal care for patients.

Keywords: Kidney disease, renal function, thyroid dysfunction

Introduction

Thyroid dysfunction is known to disrupt the normal functions of various organ systems within the human body, including the heart, muscles, and brain. Additionally, the status of the thyroid gland can have significant effects on renal function. This impact on the kidney is attributed to the thyroid hormone's ability to induce systemic or localized hemodynamic changes, as well as directly influence renal function. Both hypothyroidism and hyperthyroidism can noticeably impact renal function. Furthermore, there have been documented cases linking different types of glomerulopathies with thyroid abnormalities [1-3]. However, there is a paucity of clinical research on the relationship between thyroid dysfunction and renal function, leaving much to be understood about how thyroid dysfunction influences kidney function in humans. Additionally, the effects of thyroid hormones on renal function in humans may be subtle, often going unnoticed in clinical practice due to changes in measured renal function parameters falling within normal limits. It is widely acknowledged that there is an intricate interplay between thyroid and renal function, and thyroid disorders can significantly impact kidney function, particularly by affecting glomerular filtration rate (GFR). Various tests are used in routine clinical practice to assess kidney function, with serum creatinine and estimated GFR (eGFR) being the most commonly used biomarkers. The study revealed significant variations in renal function in correlation with the severity of thyroid dysfunction. Whether assessed through creatinine levels or eGFR, these changes in renal function persist [4]. Furthermore, there was a notable decrease in the average serum creatinine level in hyperthyroid patients prior to treatment in comparison to

after treatment, while the mean eGFR also saw a significant decline post-treatment. It was observed that TSH levels had a clear positive connection with serum creatinine and a distinct negative relationship with eGFR across all patients with thyroid issues. In cases of hypothyroidism, there is an increase in serum creatinine levels alongside a decrease in eGFR, whereas in hyperthyroidism, the opposite is true. These findings align with findings from previous studies [5]. An elevation in serum creatinine levels has been associated with an elevation in TSH levels. Previous research has indicated a temporary increase in serum creatinine among individuals with hypothyroidism. Despite the use of less sensitive TSH and FT4 tests in past studies, their findings remain consistent with more recent research. Verhelst *et al.* demonstrated that individuals with hyperthyroidism had reduced serum creatinine levels, whereas those with hypothyroidism exhibited elevated levels [6]. The glomerular filtration rate (GFR) experiences a reversible decrease of approximately 40% in over 55% of individuals with hypothyroidism. The decrease in glomerular filtration rate (GFR) seen in patients with hypothyroidism is attributed to various complex mechanisms. Furthermore, structural abnormalities in the glomerulus, such as thickening of the glomerular basement membrane and expansion of the mesangial matrix, can also result in a decrease in renal blood flow in individuals with hypothyroidism. Decreased sensitivity to adrenergic stimulation, reduced release of renin, diminished levels of angiotensin II, and decreased activity of the renin-angiotensin system can all result in a loss of GFR in hypothyroid individuals [2]. Renal parenchymal growth retardation seen in hypothyroidism leads to a limitation in

glomerular surface area for filtration. In addition, there is a reduction in the absorption of sodium, chloride, and water in the proximal tubules. The expression of the chloride channel on the basolateral side of the kidney is also decreased. This leads to a decrease in chloride reabsorption and an increase in distal chloride supply, triggering the tubuloglomerular feedback mechanism mediated by the macula densa, which decreases renin-angiotensin system activity. Consequently, there is a decrease in the glomerular filtration rate. Myopathy and rhabdomyolysis are common factors contributing to the temporary rise in serum creatinine levels in cases of thyroid-related kidney abnormalities. Normalizing thyroid function through effective therapy typically leads to restoration of renal function [2, 3]. The present investigation observes that renal function in patients with hypothyroidism typically returns to normal levels following correction of thyroid dysfunction. These findings align with prior studies indicating that glomerular filtration rate (GFR) in individuals with myxedema is approximately one-third lower than in those with normal thyroid function but can be readily restored through thyroxine replacement therapy [1, 2].

Thyroid dysfunction and renal function

Hyperthyroidism, conversely, elicits a contrasting impact on serum creatinine and eGFR. The etiology of this condition is multifaceted and intricate. Initially, hyperthyroidism elevates cardiac output levels due to its advantageous chronotropic and inotropic effects on the heart. Moreover, the activation of the renin-angiotensin system prompts a rise in blood volume, consequently boosting renal blood flow. Excessive production of thyroid hormone diminishes resistance in afferent glomerular arterioles, resulting in an elevation in glomerular hydrostatic pressure and GFR. The initiation of tubuloglomerular feedback spawns a reduction in chloride load through heightened chloride absorption in the proximal tubule and Henle's loop segments, thereby leading to an increase in GFR [2]. Treating hyperthyroidism reverses these effects, leading to a normalization of the glomerular filtration rate (GFR). Furthermore, hyperthyroid patients experience an elevation in GFR along with a reduction in total muscle mass, resulting in significantly lower levels of serum creatinine, which is an indirect indicator of GFR [7]. In light of these findings, the impact of thyroid hormone on GFR can provide insights into the fluctuation of serum creatinine levels in various thyroid disorders. While subtle, these fluctuations can have significant implications for certain individuals. Changes in GFR in hypothyroidism may lead to heightened risk of toxicity in patients taking medications with narrow therapeutic windows, such as digoxin or metformin. It is also crucial to note that in cases where serum creatinine levels unexpectedly rise, screening for thyroid hormone is essential to detect underlying hypothyroidism.

Interactions between thyroid disorders and kidney disease

For a significant amount of time, the reciprocal influence between the thyroid and kidney in modulating their respective functions has been acknowledged. Dysfunction of the thyroid can impact the physiological processes and growth of the kidneys, while kidney diseases can lead to thyroid dysfunction. There are shared underlying causes for the coexistence of thyroid and kidney disorders [8]. Thyroid hormones play a significant role in protein synthesis and cellular growth. Research conducted on neonatal rats has

illustrated the stimulatory impact of thyroid hormones on the development of the kidneys. The status of thyroid hormones has an impact on the functional renal mass, as indicated by the ratio of kidney size to body mass, with decreased ratios observed in cases of hypothyroidism and increased ratios in cases of hyperthyroidism. Nevertheless, significant hyperthyroidism has the potential to result in the breakdown of proteins and eventual deterioration of the kidneys. In addition, infants with congenital hypothyroidism are often found to have a higher incidence of congenital kidney abnormalities. The function of the kidneys in newborns is also influenced by thyroid hormones. [9]

Impact of Thyroid Abnormalities on Renal Function

The functioning of the thyroid gland has an impact on renal blood flow, glomerular filtration rate, tubular function, electrolyte balance, and the integrity of the kidneys. An overview of the diverse consequences of both an underactive and overactive thyroid on kidney function is presented in Figure 1.

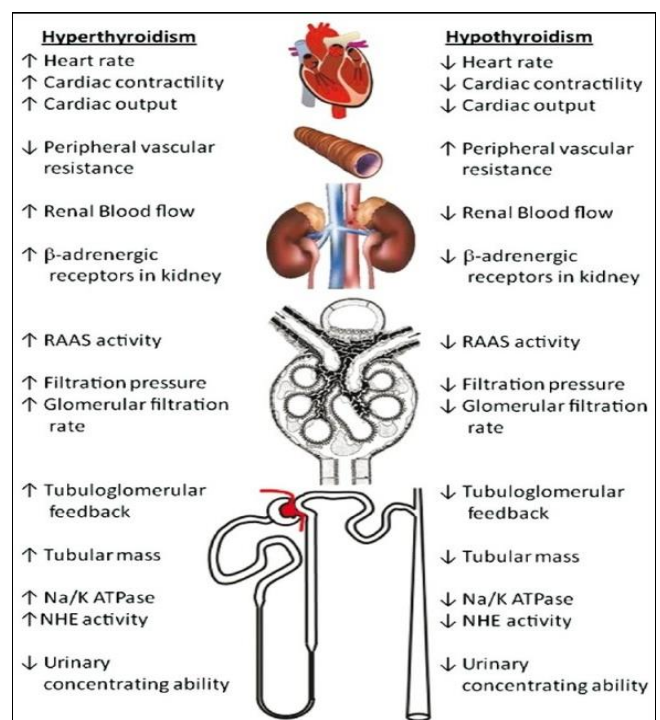


Fig 1: Illustrates the impact of both hyperthyroidism and hypothyroidism on the physiological processes and function of the kidney

Effects of an overactive thyroid on Kidney Function

Hyperthyroidism results in an increase in renal blood flow (RBF) and glomerular filtration rate (GFR). The influence of thyroid hormones on RBF and GFR occurs at multiple levels. Among the pre-renal factors, thyroid hormones enhance cardiac output through positive chronotropic and inotropic effects, while reducing systemic vascular resistance, ultimately resulting in an elevation in RBF. The synthesis of nitric oxide (NO) from endothelial cells in the renal cortex and medulla is enhanced due to the stimulation of nitric oxide synthase (NOS) by thyroid hormones and indirectly by endothelial shear stress caused by high arterial pressure. This results in a reduction of vasoconstrictor endothelin levels within the kidneys. Consequently, there is an increase in intrarenal vasodilation and a decrease in vasoconstriction, ultimately resulting in an overall improvement in renal blood flow (RBF) [10]. The glomerular

filtration rate (GFR) typically rises by 18–25% in individuals with hyperthyroidism. This enhancement in GFR cannot be attributed solely to increased renal blood flow. The activation of the renin – angiotensin – aldosterone system (RAAS) is also implicated in the increased glomerular filtration rate. Thyroid hormones play a crucial role in stimulating the Renin-Angiotensin-Aldosterone System (RAAS) through a complex and interwoven mechanism. In individuals with hyperthyroidism, there is an increase in β -adrenergic activity, leading to a higher number of β -adrenergic receptors in the renal cortex, thereby enhancing the activation of the RAAS. Specifically, T3 promotes the expression of the renin gene, resulting in elevated levels of plasma renin, angiotensin II, and serum angiotensin converting enzyme. Furthermore, there is an increase in angiotensinogen synthesis by the liver and a rise in the density of angiotensin receptors^[11]. Therefore, there is a resultant rise in the activity of the Renin-Angiotensin-Aldosterone System (RAAS). This leads to dilation of the afferent arteriole and constriction of the efferent arteriole, causing an increase in filtration pressure. This amplifies the elevation in Glomerular Filtration Rate (GFR) beyond that caused by an increase in Renal Blood Flow (RBF). This contributes to an enhancement in proximal sodium reabsorption. Moreover, hyperthyroidism leads to an increase in the size of tubules, renal tissue, and tubular reabsorptive functions. An increase in basolateral sodium levels triggers the basolateral sodium calcium exchanger, along with efficient chloride reabsorption and movement through the basolateral chloride channel, thereby improving calcium reabsorption, particularly at the loop of Henle. As a consequence, there is a reduction in chloride transportation to the distal nephron, which is recognized by the macula densa, ultimately resulting in heightened RAAS activity. In hyperthyroidism, heightened sensitivity of the macula densa further stimulates the RAA system.^[12] Upon successful treatment of hyperthyroidism, the adverse effects are reversed, leading to a restoration of the glomerular filtration rate (GFR) to its normal levels. In hyperthyroid patients, the levels of serum creatinine, which is an indicator inversely related to GFR, exhibit a significant reduction. The decline in serum creatinine levels can be linked to both the rise in glomerular filtration rate (GFR) and the decrease in muscle mass among these individuals. The introduction of cystatin C, which is a naturally secreted inhibitor of cysteine protease found in all nucleated cells, has emerged as a unique indicator of renal function and a predictor of potential cardiovascular risks. The relationship between serum cystatin C levels and GFR in hyperthyroid individuals is not highly correlated. Treatment for hyperthyroidism often leads to a rise in serum creatinine levels and a decline in serum cystatin C levels. Urinary neutrophil gelatinase-associated lipocalin (NGAL), a novel biomarker used to assess renal function, appears to remain stable regardless of changes in thyroid function.^[13] The elevation in levels of 24-hour urine protein seen in individuals with hyperthyroidism is likely a result of glomerular hyperfiltration, a condition that typically resolves with appropriate treatment of hyperthyroidism. The higher levels of Urinary N-acetyl- β -D-glucosaminidase (NAG) in hyperthyroidism are thought to be caused by disruptions in the glomerular basement membrane and tubular damage due to hyperfiltration, hypertrophy, and hyperplasia. The reduced ability of the kidneys to concentrate urine in hyperthyroidism is believed to be caused by an increase in renal blood flow and osmotic

diuresis, rather than insensitivity to vasopressin. Hyperthyroidism is associated with decreases in total body water and interchangeable potassium levels, although levels of sodium in the blood typically remain normal^[14].

Chronic Kidney Disease and Thyroid Dysfunction

Hyperthyroidism possesses the ability to initiate or accelerate the advancement of chronic kidney disease (CKD) through multiple pathways. In addition, hyperthyroidism heightens the probability of proteinuria, a condition that is recognized for its detrimental effects on renal function. Thirdly, the heightened mitochondrial energy metabolism induced by hyperthyroidism, coupled with decreased superoxide dismutase expression, contributes to an elevated production of free radicals and subsequent renal damage. Oxidative stress also plays a role in the development of hypertension in individuals with hyperthyroidism, which further exacerbates the advancement of CKD. Increased activity of the renin-angiotensin-aldosterone system (RAAS) can expedite the progression of renal fibrosis in this context. Hypothyroidism is not considered a significant contributor to the worsening of CKD, except in cases where there is a mild to moderate decline in glomerular filtration rate (GFR). Treating hypothyroidism has the potential to enhance GFR in CKD patients.^[15] Specifically, the incidence of subclinical hypothyroidism tends to rise as the glomerular filtration rate decreases. Among patients with chronic kidney disease, the primary and most prevalent anomaly in thyroid function is a decline in T3 levels, specifically total T3 as opposed to free T3. Referred to as the "low T3 syndrome" in CKD, this occurrence can be ascribed to various factors including fasting, persistent metabolic acidosis, and chronic protein deficiency, all of which affect the metabolism and protein bonding of T3. Consequently, there is a decrease in the conversion of T4 to T3 and its association with proteins. Additionally, research has demonstrated that inflammatory cytokines like tumor necrosis factor (TNF)- α and interleukin (IL)-1 can inhibit the function of type 1 5'-deiodinase, which plays a role in converting T4 to T3 outside of the thyroid gland. Moreover, impairment in the regulation of iodine by the kidneys can result in elevated levels of iodine in the bloodstream, leading to a prolonged Wolff-Chaikoff effect. The medical significance of the low T3 syndrome is presently a topic of debate. Studies show that reduced T3 levels, specifically total T3 instead of free T3, in patients with chronic kidney disease are associated with elevated inflammatory markers like highly sensitive C-reactive protein (hsCRP) and IL-6, signs of malnutrition (low prealbumin, IGF-1), heightened endothelial dysfunction, impaired cardiac function, decreased survival rates, and increased overall and cardiovascular mortality as observed in specific research^[16]. Further studies have also demonstrated a reduction in T4 levels among a considerable portion of individuals with chronic kidney disease (CKD). However, patients with CKD may encounter variations in their free T4 levels, fluctuating between low and normal, as a result of the compromised binding of T4 to proteins. The thyroid function in individuals with chronic kidney disease closely resembles that seen in a range of non-thyroidal conditions, including severe infections, heart failure, malignancies, and in patients without kidney disease who are hospitalized. The resemblance between chronic kidney disease and non-thyroidal illness has given rise to the concept of a "sick euthyroid state," now more commonly referred to as "non-thyroidal illness." Unlike other non-

thyroidal illness conditions, chronic kidney disease does not result in elevated total rT3 levels.^[17] The reason for this occurrence is an enhanced movement of rT3 into areas outside of blood vessels and within cells. Some individuals may exhibit slightly elevated levels of free rT3 due to reduced clearance by the kidneys. A distinguishing factor in CKD cases is the elevated presence in individuals with chronic kidney disease (CKD), thyroid stimulating hormone (TSH) is produced in response to thyrotropin releasing hormone (TRH), indicating potential dysfunction of the pituitary gland due to uremia.^[16] Furthermore, the circadian pattern of thyroid-stimulating hormone (TSH) and its glycosylation is disrupted in chronic kidney disease (CKD), leading to a decline in its effectiveness. As a result, individuals with CKD often exhibit decreased levels of triiodothyronine (T3) and normal to decreased levels of thyroxine (T4), accompanied by elevated TSH and an associated growth in the thyroid gland.^[18] Chronic Kidney Disease (CKD) leads to a decrease in the excretion of iodide, causing an elevation in the level of inorganic iodide in the blood as well as an increase in the iodine content of the thyroid gland, ultimately resulting in thyroid gland enlargement. Among CKD patients, there is a higher incidence of structural abnormalities in the thyroid gland such as an elevated incidence of goiter, especially among females, thyroid nodules, and thyroid carcinoma has been observed when compared to the general populace^[19]. Patients with chronic kidney disease (CKD) do not show increased prevalence of autoimmune thyroid disease. Within CKD patients, the occurrence of positive thyroglobulin and thyroid microsomal antibodies is actually minimal. However, autoimmune thyroid disease may coincide with other autoimmune disorders commonly linked to CKD, including lupus nephritis and type 1 diabetes mellitus. Screening for antithyroid antibodies is essential when a high level of thyroid-stimulating hormone (TSH) is identified alongside another autoimmune disorder. The treatment strategy for autoimmune thyroid disease remains steady irrespective of the existence of chronic kidney disease (CKD). The effects of CKD on thyroid function are depicted in Figure 2.

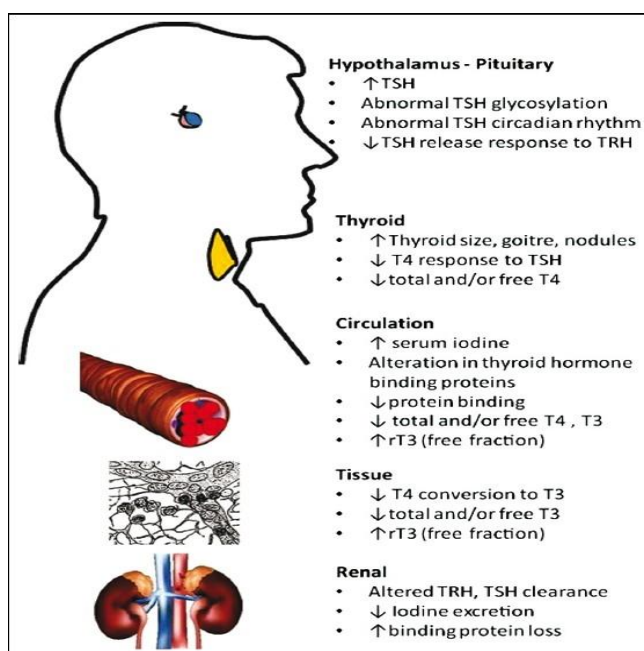


Fig 2: Demonstrates the influence of prolonged kidney disease on the functionality of the thyroid gland

Pharmaceutical interventions for conditions affecting the thyroid and kidneys Medications administered for thyroid or kidney disorders can potentially impact the functioning of other organs. Thionamides like methimazole, carbimazole, and propylthiouracil can induce hypothyroidism and renal impairment through immune-mediated processes, leading to the development of diverse glomerular diseases^[20] Another drug, Lenalidomide, recognized for its antitumor and antiangiogenic properties in renal cell carcinoma, may lead to subacute thyroiditis and temporary thyrotoxicosis. On the other hand, Sunitinib, a novel treatment for renal cell carcinoma, has been associated with hypothyroidism, which some researchers suggest may be connected to a more favorable prognosis^[21]. The utilization of lithium has been found to result in hypothyroidism, nephrogenic diabetes insipidus, and chronic kidney disease. The administration of amiodarone has been associated with the development of hypothyroidism and hyperthyroidism, along with cases of acute renal dysfunction. Rifampicin has been recognized as a potential cause of tubulointerstitial nephritis and hyperthyroidism. In the management of hyperthyroidism in patients with chronic kidney disease, it is essential to modify the treatment regimen as necessary. Patients with hyperthyroidism who are receiving hemodialysis need to be administered the typical therapeutic dosage of 131I for treatment due to the clearance of the substance through dialysis. On the other hand, individuals undergoing peritoneal dialysis require a notable decrease in the amount of 131I when treating thyroid cancer in order to mitigate the risk of excessive radiation exposure^[22].

Thyroid dysfunction and dyslipidemia in chronic kidney disease patients

Chronic kidney disease (CKD) is emerging as a significant public health concern, with a growing number of individuals experiencing diminished kidney function, particularly in developed nations. Recent data indicates a sudden surge in CKD cases in developing nations in Asia attributed to the rise in coexisting conditions. This increase in CKD cases is also leading to a substantial rise in healthcare costs for the management of advanced-stage CKD^[23]. Numerous studies indicate that the advancement of chronic kidney disease (CKD) correlates with the onset of various complications, including thyroid dysfunction, dyslipidemia, and cardiovascular illness. The kidney plays a crucial role in the metabolism, breakdown, and elimination of thyroid hormones. CKD disrupts the hypothalamus-pituitary-thyroid axis, impacting thyroid function through various mechanisms such as lowered levels of circulating thyroid hormones, modifications in peripheral hormone metabolism, inadequate binding to carrier proteins, reduced tissue levels of thyroid hormones, and alterations in iodine storage within the thyroid gland. As a result, there is an impairment in the metabolism of thyroid hormones in patients with Chronic Kidney Disease (CKD). CKD is associated with an increased occurrence of primary hypothyroidism, both in symptomatic and asymptomatic presentations, while hyperthyroidism is not as prevalent. The frequency of primary hypothyroidism, especially in its asymptomatic form, grows as the glomerular filtration rate (GFR) decreases^[24]. Dyslipidemia has been widely recognized as a conventional risk factor for cardiovascular disease in patients suffering from chronic kidney disease (CKD). Numerous observational studies have highlighted the

significant role of total and low-density lipoprotein (LDL) cholesterol as independent markers of cardiovascular morbidity and mortality in this population. Several factors contribute to the development of dyslipidemia in individuals with CKD, including a decrease in the activity of lipoprotein lipase and hepatic triglyceride lipase. This diminished activity impairs the clearance of triglyceride-rich, apolipoprotein B-containing lipoproteins by the liver and peripheral tissues, leading to elevated levels of these atherogenic lipoproteins circulating in the bloodstream. The progression of CKD is accompanied by unique changes in lipoprotein metabolism. Studies have revealed that mortality from cardiovascular disease is significantly higher, ranging from 10 to 30 times, in dialysis patients compared to the general population [25]. Recent studies indicate that abnormalities in lipid metabolism could potentially impact the development of renal disease. It has been observed that individuals with chronic kidney disease (CKD) who also experience thyroid dysfunction and dyslipidemia may face a higher likelihood of cardiovascular complications, leading to increased rates of morbidity and mortality. Hence, timely identification of thyroid and lipid irregularities through regular screenings, coupled with effective management strategies for CKD patients, may offer benefits in delaying the progression of CKD and reducing cardiovascular risks. A survey conducted in eastern Nepal revealed a prevalence of CKD in 10.6% of the general population, with age and diabetes mellitus identified as significant factors in predicting CKD. Additionally, a separate study found dyslipidemia to be a common occurrence among Nepalese individuals with CKD. Given the observed decline in thyroid function and prevalence of dyslipidemia in CKD patients, coupled with limited data on thyroid function and dyslipidemia in the Nepalese CKD population, our current research endeavors to address this gap in knowledge through an investigation within this specific demographic [26].

Thyroid Parameters and Kidney Disorder

Diabetes mellitus (DM) and its associated complications have become significantly widespread and are receiving heightened awareness, particularly in developing nations. The recorded prevalence of diabetes within a representative subset of Chinese adults was measured at 11.6%, underscoring the significance of diabetes as a pressing public health concern in China. Chronic kidney disease (CKD) is characterized by the persistent existence of diminished kidney function or impairment, frequently stemming from diabetes mellitus (DM) and hypertension. Roughly 13% of individuals in the overall US populace are affected by CKD, with a noticeable uptick in global incidence rates [27]. Diabetic kidney disease (DKD) is a prevalent microvascular complication of diabetes mellitus and a primary contributor to end-stage renal disease (ESRD) globally. Between 20% and 40% of diabetic patients develop DKD, with 40% progressing to ESRD. Reduced estimated glomerular filtration rate (eGFR) and elevated urinary albumin to creatinine ratio (UACR) are key indicators of diabetic kidney disease, reflecting renal function and the extent of renal damage. The thyroid gland plays a critical role in regulating various physiological functions in the body. Thyroid hormone influences renal tubular function, the renin-angiotensin system, and is linked to hemodynamic and cardiovascular changes that impact

renal blood flow. On the other hand, the kidney not only metabolizes and eliminates thyroid hormone but also serves as a target for specific actions of iodothyronines [28]. Disruptions in hormone levels within the hypothalamic-pituitary axis frequently coincide with deteriorating kidney function, with emerging research indicating a potential role of these hormonal imbalances in the development of CKD. Thyroid dysfunction leads to notable alterations in both glomerular and tubular functions as well as in the regulation of electrolytes and water balance [29].

Conclusions

Thyroid dysfunction has been found to be linked with impaired kidney function. Following treatment, hypothyroid patients experienced a statistically significant decrease in average serum creatinine levels and a notable improvement in mean eGFR. Conversely, hyperthyroid patients saw a significant increase in average serum creatinine levels post-treatment, along with a decrease in mean eGFR. TSH levels were discovered to have a notable positive correlation with serum creatinine and a substantial negative correlation with eGFR in patients exhibiting thyroid dysfunction. Clinicians must recognize the correlation between thyroid disorders and abnormal kidney function markers, prompting consideration of thyroid function testing for patients with only slightly elevated renal function markers. These results underscore the importance of monitoring creatinine levels in patients with thyroid dysfunction, while also highlighting the necessity for further investigation into the potential adverse effects of thyroid dysfunction on kidney function. There are multiple pathways through which the functions of the kidney and thyroid interact in different diseased states of each organ. This interplay encompasses alterations in function as well as structural linkages. Elevated levels of TSH are frequently observed in cases of chronic kidney disease, although this does not always signify hypothyroidism.

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