Republic of Iraq

Ministry of Higher Education& Scientific Research

University of Babylon – College of Science

Department of Biology

The Relation between Thyroid Stimulating Hormone (TSH) and Kidney Failure Disease

Research project

Submitted to the Department of Biology as a Partial Fulfillment of the Requirements for the Degree of B. Sc. in Biology/Microbiology

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2022 AD **1443 AH**

بِسْمِ اللَّـهِ الرَّحْمَـٰنِ الرَّحِيمِ

هُوَ الَّذِي جَعَلَ الشَّمْسَ ضِيَاءً وَالْقَمَرَ نُورًا وَقَدَّرَهُ مَنَازِلَ لِتَعْلَمُوا عَدَدَ السِّنِينَ وَالْحِسَابَ ۚ مَا خَلَقَ اللَّهُ ذَٰلِكَ إِلَّا بِالْحَقِّ ۚ يُفَصِّلُ الْآيَاتِ لِقَوْمٍ يَعْلَمُونَ ﴿٥﴾

صدق الله العلي العظيم
سورة يونس : الآية (5)

Dedication

I dedicate this research to our parents and teachers, who taught us to think, understand and express. I earnestly feel that without their inspiration, able guidance, and dedication, I would not be able to pass through the tiring process of this research.

Acknowledgments

First of all, I would like to express my gratitude to God for enabling me to complete this research I would like to thank my supervisor lecturer Rasha Kadhim Mahdi and also I would like express my grateful thanks to Dr. Alaa Tariq Al-Shamakhi for their support throughout my research their comments and suggestions have been invaluable in shaping my ideas and views contained in this research.

Finally, my mother and father have been a constant rock of support and encouragement without their influence none of this would have been possible.

Abstract

The current study was carried out at the University of Babylon - College of Science - Department of biology to determine the effect of kidney failure disease on blood creatinine and thyroid stimulating hormone levels.

In this study, 200 samples were used, including 150 people with kidney failure disease, known as the Patients group, and 50 healthy people, known as the Control group.

The statistical analysis of the creatinine test revealed significant differences between the groups, with the Patients group showing an increase compared to the control group, and the results of the thyroid stimulating hormone showing an increase in the patient's group compared to the control group. From our results, it can be concluded that TSH elevations are common in patients with chronic kidney disease, but they do not always indicate hypothyroidism, because there are various mechanisms of interaction between kidney and thyroid functions and these interactions have both functional and structural correlates.

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Chapter One

1-1: Introduction

Chronic kidney disease (CKD) is characterized as long-term kidney impairment accompanied by a decrease in glomerular filtration rate (GFR) and albuminuria (Trevidi *et al*., 2021). CKD is a progressive, irreversible condition that is the eighth leading cause of death in the United States. According to a population study, one in every ten American adults (more than 30 million people) has some form of CKD, and risk factors for CKD include diabetes, hypertension, hyperlipidemia, and thyroid disorders (Mohamedali *et al*., 2014).

Chronic kidney disease has a variety of endocrine and metabolic effects. One of the most prevalent affects bone metabolism, resulting in chronic kidney disease mineral bone disorder (CKD-MBD) and secondary hyperparathyroidism, hypothyroidism, and nodular goiter are two more significant endocrine pathologies seen in people with chronic renal disease (Cotoi *et al*., 2020).

The thyroid gland's function is one of the most crucial in the human body since it controls the majority of the body's physiological processes. Thyroid hormones (T3 and T4) have several functions, including metabolism, development, protein synthesis, and the control of numerous other essential hormones. Any thyroid malfunction can impact the synthesis of thyroid hormones (T3 and T4), which can be connected to a variety of diseases throughout the body, thyroid hormone levels and how they impact the course of CKD are one of the most significant disorders that have received little attention (Mariani and Berns, 2012).

Thyroid function has been linked to kidney function and CKD. Hypothyroidism has been associated with altered kidney function via effects

on cardiac output, intra-renal hemodynamics, and the renin-angiotensin-aldosterone system (RAAS), as well as structural changes such as decreased kidney-to-bodyweight ratio, truncated tubular mass, and altered glomerular architecture as well as Hypothyroidism, is more common in patients with CKD (Huang *et al*., 2020).

Thyroid hormone levels and how they impact the progress of CKD are one of the most significant disorders that have received little attention. Renal dysfunction has been seen to coexist with particular amounts of thyroid hormone. This research is being conducted to simplify the significance of relationships between thyroid function and renal illness. This research is critical because it demonstrates a relationship between two distinct disorders. The information obtained from this paper will assist to expand clinical understanding and enable physicians to give better therapy to their patients with thyroid or renal problems.

Chapter Two

Literature Review

2-1: Chronic Kidney Disease (CKD)

Chronic kidney disease is a general term for a variety of disorders affecting the structure and function of the kidney; variation in disease expression is related to cause and pathology, severity, and rate of progression (Levey and Coresh, 2012). The best available indicator of overall kidney function is glomerular filtration rate (GFR), which equals the total amount of fluid filtered through all of the functioning nephrons per unit of time, the diagnosis of CKD is based on establishing a chronic reduction in kidney function and structural kidney damage (Webster *et al*., 2016).

CKD is distinguished by a consistently abnormal glomerular filtration rate (GFR). It represents an evolving process that is initiated by various courses, all of which result in persistent and usually progressive kidney damage of varying severity (Sharma and Prasad, 2005).

An adult patient is diagnosed with CKD in their glomerular filtration rate (GFR) is less than 60 ml/min/1.73 m2 for three months or over, or if their GFR is greater than 60 ml/min/1.73 m2, but there is evidence of renal structure injury, Albuminuria, changes in renal imaging, hematuria/leukocyturia, persistent hydro electrolytic disorders, histological changes in kidney biopsy, and a history of kidney transplantation are all indicators of renal injury (Ammirati, 2020).

Many featured are common to the progression of renal failure of various causes, and the final histologic appearance is one of glomerulosclerosis, interstitial fibrosis, and loss of native renal cell,

nevertheless, the causes of renal failure are heterogeneous, and the mechanisms and locations of the initial injury may vary (Henry, 2003).

Atkins, (2005) described five epidemiologic aspects of chronic kidney disease. First, early kidney damage is prevalent; second, there is the prevalence and incidence of end-stage kidney failure; third, there is the changing pattern of chronic kidney disease; fourth, there is diabetes and cardiovascular pandemic; and finally, there is what can be done to reduce the impending disease burden with all of its medical and financial ramifications.

The GFR is considered to be the best marker for kidney function, the early stages of renal function impairment are clinically silent and can only be diagnosed by measuring GFR using external filtration markers (measured GFR, mGFR) and once GFR has decreased to 60, functional impairments can be detected by determining internal filtration markers and calculating the estimated GFR (eGFR), the complications of CKD increase with decreasing GFR and may progress from gradual redistribution. The goal of GFR determination is to detect CRD early so that it can be slowed (Thomas and Thomas, 2009).

2-2: Creatinine

Serum creatinine is derived almost completely from the breakdown of creatine in muscle tissue as energy in the phosphocreatine form. The existence of this metabolite in the circulation is a physiological factor because this is one of the catabolite protein metabolisms, the excretion of creatinine occurs through the kidney and the levels of plasma creatinine reflect the renal filtration rate (Saraiva *et al*., 2014). Under stable case and steady kidney function, creatinine is usually produced at a proximately constant rate by the body independent of the absolute amount

of the muscle mass, through the glomeruli, the creatinine is filtered out of the blood (and is eliminated to a small extent in the proximal tubules of the kidney) (Patel *et al*., 2012).

As serum creatinine (SCr) is derived from muscle creatinine, different factors affect SCr levels such as muscle turnover (recovery from starvation and muscle disorder), dietary protein, muscle mass, medication, and creatine levels in addition to glomerular filtration and tubular transport (Isaacs *et al*., 2014).

During a steady state of renal function, serum creatinine (Cr) is typically used to estimate glomerular filtration rate (GFR), even though serum Cr is derived from creatine, 95 percent of which is found in muscle, serum Cr level may be used as a surrogate of muscle mass in end-stage renal disease (ESRD) patients with a balanced distribution of low GFRs (Park *et al*., 2013). A study was done by Kreisman *et al*., (1999) mentioned that creatinine affected the hypothyroid state in the physiology of the kidney through changes in water and electrolytes metabolism, hyponatremia, and reliable alterations of renal hemodynamics, including decrements in renal blood flow, renal plasma flow, glomerular filtration rate (GFR), and single nephron GFR.

2-3: Thyroid gland

The thyroid gland, like other endocrine glands, is regulated by a feedback system that includes the hypothalamus, pituitary, and target gland (the thyroid), this relationship between them is referred to as the HPT axis, and the hypothalamus produces thyrotropin-releasing hormone (TRH), a tripeptide that is secreted into the venous system that drains to the pituitary gland, and TRH binds to receptors in thyrotropic cells, causing the production and secretion of thyroid stimulating hormone (TSH), also known as thy Thyroid stimulating hormone bind to TSH

receptors in the thyroid gland's follicular cells, causing the production and secretion of thyroid hormones, T3 and T4(Chiasera, 2013).

Thyrotropin-releasing hormone (TRH) from the hypothalamus, thyroid-stimulating hormone (TSH) from the anterior pituitary gland, and T4 all work together to keep the feedback mechanism and homeostasis (Shahid *et al*., 2022).

Mishra, (2012) described the importance of thyroid hormones to the human body which is linked to the physical, mental, memory and sexual functioning of our bodies, in addition, it is linked to many other important functions of our bodies.

The interactions between kidney and thyroid functions are known for years, Thyroid hormones (TH) are necessary for the growth and development of the kidney and the maintenance of water and electrolyte homeostasis, On the other hand, the kidney is involved in the metabolism and elimination of TH, in addition to clinically both hypothyroidism and hyperthyroidism are accompanied by remarkable alterations in the metabolism of water and electrolyte, as well as in cardiovascular function. All these effects generate changes in water and electrolyte kidney management, Moreover, the decline of kidney function is accompanied by changes in the synthesis, secretion, metabolism, and elimination of TH (Iglesias and Dı´ez, 2009).

Thyroid hormones affect renal function by both pre-renal and direct renal effects

1- Pre-renal effects are mediated by the influence of thyroid hormones on the cardiovascular system and renal blood flow (RBF).

2- The direct renal effects are mediated by the effect of thyroid hormones on

A- glomerular filtration rate (GFR).

B- tubular secretory and re-absorptive processes, as well as

C- hormonal influences on renal tubular physiology.

Thyroid hormones affect the renal clearance of water load by their effects on the GFR (Basu and Mohapatra, 2012).

Kim *et al*., (2020) demonstrated the effects of TSH and fT4 on kidney function simultaneously, In fact; the metabolism of T4 is altered in CKD patients; deiodination of T4 and T4-binding plasma proteins is reduced and inhibitors of T4 binding to plasma proteins and metabolic acidosis are presented.

It is well known that the kidney can have an impact on how the thyroid functions, the kidney is in control of thyroid hormone metabolism by degrading and excreting thyroid hormones through renal function, thus in the case of kidney diseases, thyroid disorders may be encountered negatively; however, this process is not straight forward, and thyroid abnormalities primarily occur when CKD is present (Aljabri *et al*., 2019).

Chronic kidney diseases (CKDs) may affect thyroid function both have common etiological factors And are commonly observed in CKD patients with nonimmune primary hypothyroidism especially, subclinical hypothyroidism prevalence increases with a decline in glomerular filtration rate (GFR) Usually low T3 level (total more than free T3) is the first thyroid manifestation in that patients, thyroid disorders are discovered to be a risk factor for CKD progression, and vice versa (Kamal *et al*., 2019).

2-4: Thyroid Stimulating Hormone (TSH)

Thyroid-stimulating hormone, also known as TSH, is a glycoprotein hormone produced by the anterior pituitary. It is the primary stimulus for thyroid hormone production by the thyroid gland. It also exerts growth effects on thyroid follicular cells leading to enlargement of the thyroid. The hypothalamic-pituitary axis regulates TSH release (Pirahanchi *et al*., 2022). TSH is a 28 to 30 kDa glycoprotein subset of the cystine-knot growth factor superfamily, It is produced from the basophilic cells of the anterior pituitary gland and after being released from the anterior pituitary, TSH travels to the thyroid gland and binds with TSH receptors on thyroid cells while this physiological action activates the second messenger pathway resulting in thyroid gene expression and the release of T3/T4 (Mohamedali *et al*., 2014). TSH consists of an α and a β subunit, the α subunit is common to both TSH and gonadotropins while the β subunit is a prerequisite for the bioactivity of TSH, both subunits are glycosylated post transnationally, which is controlled by the thyrotropin-releasing hormone (TRH) and is essential for exerting sufficient hormonal bioactivity (Sugimoto and Mori, 2012).

Chronic kidney disease affects the synthesis of thyroid hormones by regulating the hypothalamus-pituitary-thyroid axis, also Previous studies have shown that thyroid hormones can interfere with the accumulation of collagen in the cortical interstitium and glomeruli; thyroid-stimulating hormone (TSH) influences kidney disease progression; and low triiodothyronine (T3) levels may play a role in worsening renal function (Yang *et al*., 2022).

A study done by Asif *et al*., (2013) demonstrated there was a significant inverse association between estimated GFR and TSH levels

throughout the normal and high TSH from this correlation and concluded that chronic kidney disease is associated with biochemical thyroid dysfunctions causing most commonly subclinical hypothyroidism.

Chapter Three

Material and Methods

3-1: Subjects

The current study included the collection of 200 samples, 150 of which were patients who had already been diagnosed with kidney failure disease and ranged in age from 10 years and up, and 50 samples were collected from healthy people as a control group. Method of data collection after obtaining informed consent from all samples and personal information was recorded.

3-2: Blood collection

Blood was collected by venipuncture. Separated into 3 ml untreated gel tube for serum. The sera were frozen at -20C in two replicates until thawed for serum creatinine and TSH estimation.

3-3: Measurement of Creatinine

The estimation of creatinine in the serum and plasma was performed by using a commercially kit (BIOLABO , France), Kit procedure included-:

-Assay Procedure	Blank	Standard	Sample
Working reagent	1mL	1mL	1mL
Demineralized water	100µL		
Standard		100 µL	
Sample			100µL

Mix well, and let stand for 30 seconds at room temperature. Record absorbance (A1) at 490 nm by using (spectrophotometer Liv-1100, EMCLAB, Germany) and then record the second absorbance (A2) after 2

minutes and calculate the concentration of creatinine from the equation in the kit (mg/dl)=(Au2- A1)Assay/(A2-A1) Standard concentration.

3-4: Measurements of Thyroid stimulating hormone (TSH) ELISA

Thyroid stimulating hormone (TSH) was determined by using Enzyme Linked Immunosorbent assay techniques. ELISA instrument was used (Reader and Washer, Biotek, U.S.A). The quantitative competitive enzyme technique was used (ELISA) kit (Calbiotech, Life Science Company). The assay procedure is as follow :-

1- Pipette 50 microliter of control or specimen into the assigned well.

2-100 microliters of ready to use enzyme conjugate reagent were added to all wells.

3-The microplate swirled gently for 20-30 seconds to mix and covered.

4-Plate Incubated for 60 minutes at room temperature.

5- The contents of the microplate were discarded by aspiration and the microplate was washed by washing buffer 4 times.

6- 100 microliter of TMB substrate solution were added to all well

7- Plate Incubated at room temperature for 15 minutes.

8- 50 microliter of stop solution were added to each well and gently mix for 15-20 seconds. The color in the well should change from blue to yellow.

9- The optical density was measured by using a microplate reader at 450 nm and then calculating the concentration of TSH (µIU/ml) from the equation in the kite = optical density of sample / optical density of standard * standard concentration.

3-5: Statistical Analysis

The Sigma-plot software program (12.5) was used to perform statistical analysis. Normality was checked before analyzing data for this purpose. Mann-Whitney test was used for inconsistency with this distribution. The T-Student test was used for comparisons between groups. Variations were considered significant when P-value ≤ 0.001.

Chapter Four

Results and Discussion

4-1: Results

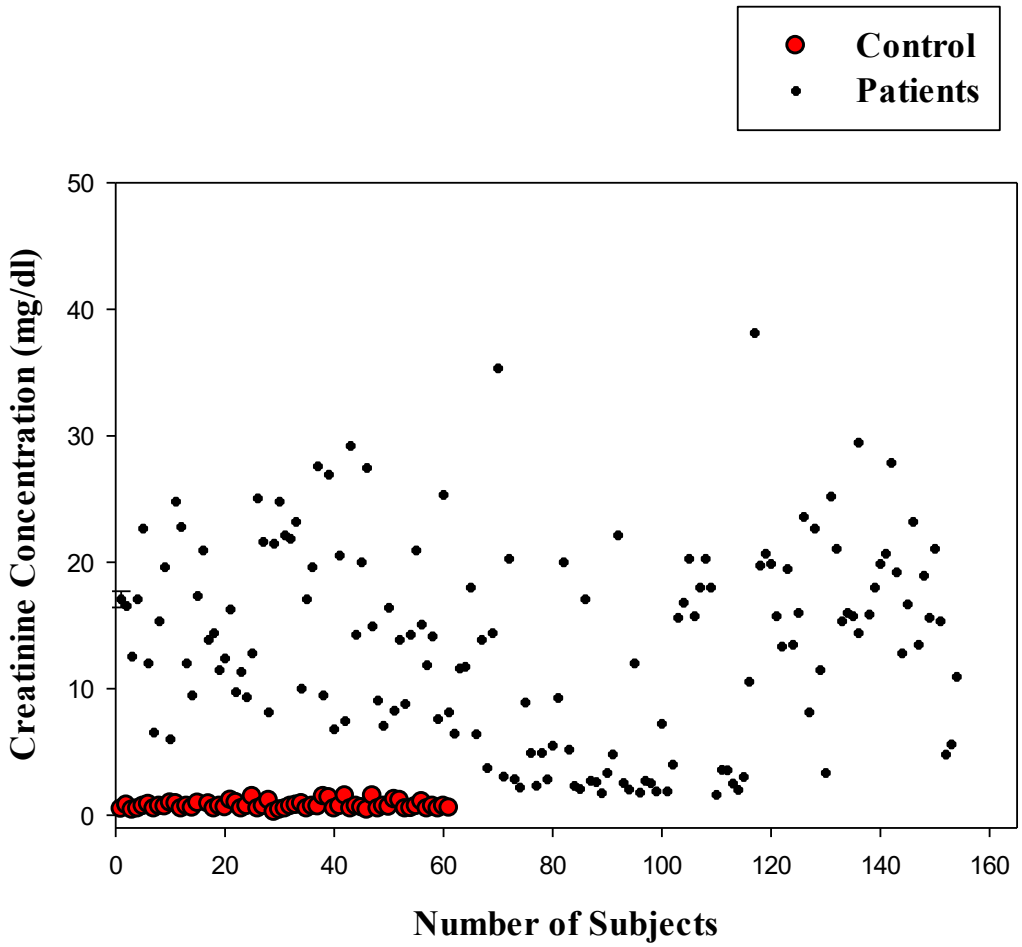

Figure (1): Creatinine Levels (Mean±SD) in Control and Patients with Chronic Kidney Disease (mg/dl).

*** Significant (≤0.001)**

Statistical analysis showed a significant increase in creatinine levels in patients with chronic kidney disease (13.571±7.875) in comparison with the control group (0.748± 0.307) as shown in figure (1).

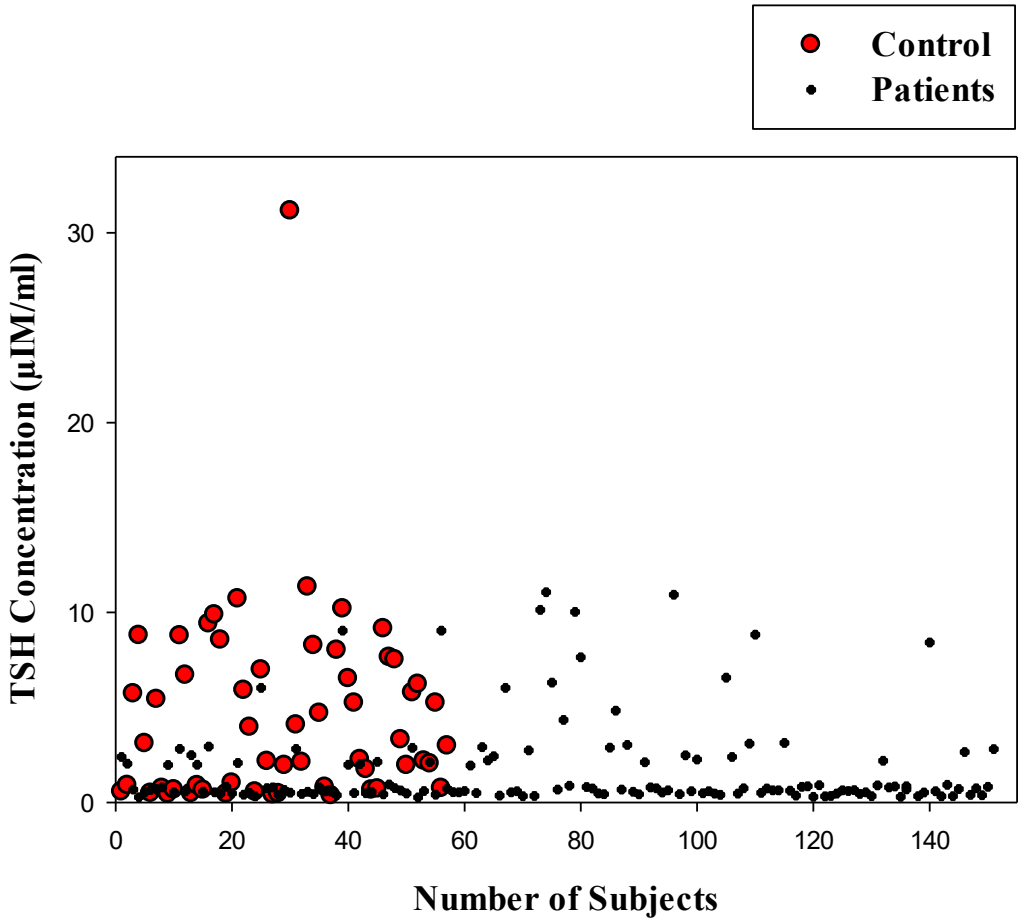

Figure (2): TSH Levels (Mean±SD) in Control and Patients with Chronic Kidney Disease (µIM/ ml).

*** Significant (≤0.001)**

Our results of TSH hormone revealed a significant decrease in TSH hormone (1.680 ± 0.189) in patients with chronic kidney disease as compared with the control group (4.556± 0.645) as shown in figure (2).

4-2: Discussion

CKD is a progressive loss of renal function disease characterized by an increase in serum nitrogen compounds and other toxins that can cause endocrine and metabolic changes. In general, some of the signs and symptoms of CKD are similar to those of hypothyroidism, such as

asthenia, cold intolerance, dry, brittle hair, somnolence, growth delay, lethargy, and hypercholesterolemia (Garrido-Magaña *et al*., 2009).

Our data showed a significant increase in serum creatinine and thyroid stimulating hormone (TSH) in patient groups as compared with the control group. This is consistent with a study done by Claus *et al*., (2005) that studied the effects of hypothyroidism on changes in cystatin C and serum creatinine in 17 patients and discovered that had a mean serum creatinine of 91 mmol/l, and mentioned that hypothyroidism is associated with renal failure, and described that this increase in the creatinine levels, which this elevation do to creatine kinase that is regularly reported in hypothyroid patients as a result of hypothyroid myopathy which associated with proximal weakness, myalgias and muscle enlargement that occur through different mechanisms are most probably related to impaired glycogenolysis and impaired mitochondrial oxidative metabolism impairment.

Further, similar results found by Mariani and Berns, (2012) Serum creatinine levels over 6 mg/dl have been associated with hypothyroidism, with a few patients even described as having the end-stage renal disease (ESRD), even though creatinine levels in most reports have been in the 1.5–2.5 mg/dl range. Another study looked at the relationship between serum creatinine and hypothyroidism and discovered that serum creatinine levels above 6 mg/dl have been linked to hypothyroidism, with a few patients even being described as having ESRD, even though most reports show creatinine levels in the 1.5–2.5 mg/dl range (Mamatha *et al*.,2016).

According to our findings, serum creatinine levels were significantly higher in another study on 80 hypothyroid patients by Khan

et al., (2013), and mentioned that these changes may result in physiological effects such as changes in renal hemodynamics, a decrease in GFR, and thus reduced creatinine clearance. Changes in the synthesis, secretion and elimination of thyroid hormones may occur if kidney functions are impaired while Increased serum creatinine levels, decreased glomerular filtration rate (GFR) and renal blood flow, impaired free water excretion, and hyponatremia are the most common renal dysfunctions in hypothyroid patients (Akagunduz, and Akcakaya, 2021; Huang *et al*., 2020; El Ters *et al*., 2014).

Some observational studies provided a correlation between TSH levels, even when they were within the normal range and excretory kidney function. A study was done by Schairer *et al*., (2020) described that higher TSH levels were associated with lower GFR, In addition, to directly influencing thyroid hormone on renin-angiotensin system activity and kidney ion channels and transporters, which leads to increased Na+-K+-2Cl co-transporter and sodium uptake, the main impact on GFR appears to be of a more indirect and functional nature. Thyroid hormones reduce systemic vascular resistance by increasing nitric oxide synthase activity, increase cardiac output by increasing inotropy and chronotrophy, and increase blood volume, and thus blood pressure. These combined effects result in increased renal plasma flow, reduced renal vascular resistance, and as a result higher GFR (Miranda *et al*., 2017; Stan and Drake, 2018; Saini *et al*., 2012). Another study done by Basu and Mohapatra (2012) attributed these raises in TSH hormone and serum creatinine in chronic renal disease patients to the renal blood flow (RFB) which is reduced in hypothyroidism by decreased cardiac output (negative chronotropic and inotropic effects), increased peripheral vascular resistance, intrarenal vasoconstriction, decreased renal response

to vasodilators, and a decreased expression of renal vasodilators such as vascular (IGF-1).

Conclusions

In the disease states of each organ, there are various mechanisms of interaction between kidney and thyroid functions. These interactions have both functional and structural correlates. TSH elevations are common in patients with chronic kidney disease, but they do not always indicate hypothyroidism.

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الخلاصة

اجريت الدراسة الحالية في جامعة بابل –كلية العلوم-قسم علوم الحياة لكشف تأثير امراض الكلية المزمنة (الفشل الكلوي) على مستوى كرياتنين الدم و مستوى الهرمون المحفز للغدة الدرقية .

استخدمت في هذه الدراسة 200 عينة منها 150 شخص مصاب بأمراض الكلية المزمن (الفشل الكلوي) وسميت بمجموعة المرضى Patients ومجموعة السيطرة مكونة من 50 شخص سليم سميت ب Control.

التحليل الاحصائي لفحص الكرياتنين اظهر وجود فروقات معنوية بين المجاميع حيث اظهرت مجموعة Patients زيادة مقارنة مع مجموعة السيطرة Control، بينما اظهرت نتائج الهرمون المحفز للغدة الدرقية زيادة في مستوى الهرمون في مجموعة المرضة Patients مقارنة مع مجموعة السيطرة Control.

من نتائجنا يمكن أن نستنتج أن ارتفاعات TSH شائعة في مرضى الكلى المزمن ، لكنها لا تشير دائمًا إلى قصور الغدة الدرقية ، لأن هناك آليات مختلفة للتفاعل بين وظائف الكلى والغدة الدرقية وهذه التفاعلات لها ارتباطات وظيفية وهيكلية.

جمهورية العراق

وزارة التعليم العالي والبحث العلمي

جامعة بابل – كلية العلوم

قسم علوم الحياة

العلاقة بين الهرمون المحفز للغدة الدرقية ومرض الفشل الكلوي

مشروع بحث

مقدمة الى مجلس قسم علوم الحياة وهي جزء من متطلبات نيل درجة البكالوريوس
في علوم الحياة / الاحياء المجهرية

من قبـــــــــــــــــــــــــــل

الطالب

سلام مسلم محمد

بأشـــــــــــــــــــــــــــراف

م. رشا كاظم مهدي

1443 هـ 2022 م