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Assessment of Thyroid and Parathyroid Dysfunction in Hemodialysis Patients

Safaa Abdulhussein Majeed [1],

Abdulkareem Mohammed Jewad [2],

Mohammed Younus Naji Al Atbee [3]

[1,2] College of Medical and Healthy Techniques, Southern Technical University- Basrah, Iraq. [3]College of Medicine, Consultant Nephrologist, University of Basra
Corresponding author: Safaa A. Majeed**,** College of Medical and Healthy Techniques, Southern Technical University- Basrah, Iraq, Email , safaaalsalih@gmail.com , Phone No.+9647728414442

Abstract

Background; Thyroid and parathyroid dysfunction in hemodialysis patients is a critical medical concern that arises due to the intricate relationship between kidney function and these endocrine glands. Hemodialysis, a life-saving procedure for individuals with kidney failure, often disrupts the delicate balance of hormones in the body. Thyroid dysfunction, characterized by alterations in thyroid hormone levels, is common and can lead to fatigue, weight changes, and mood swings. On the other hand, parathyroid dysfunction involves imbalances in calcium and phosphorus metabolism, resulting in bone and cardiovascular complications. Monitoring and managing thyroid and parathyroid health in hemodialysis patients is crucial to improving their overall well-being and quality of life.

Methodology:
Case-control the study was conducted on 75 patients undergoing hemodialysis and 75 age-matched healthy controls. Thyroid function tests and serum electrolytes were measured for each group.

Results;
Our results show a significant difference between patients and the control group in each oxidative stress marker and serum electrolyte level.

Conclusions:
From the results, we could conclude that thyroid and parathyroid dysfunction play a role in hemodialysis and can affect the management of end-stage renal failure patients.

Keywords; Hemodialysis, thyroid dysfunction, parathyroid dysfunction, electrolytes

Introduction

Hemodialysis patients (HD) is defined as a decline in renal function over three months, as expressed through the assessment of a glomerular filtration rate (GFR) falling below the threshold of 60 mL/min per 1.73 m^2 or a kidney damage marker (Gosmanova *et al.*, 2021). Chronic kidney disease indirectly impacts worldwide disorders and mortality by increasing the odds of at least five other leading causes of death. DM, malaria, HIV, hypertension, and cardiovascular disease (CVD). Using the Global Burden of Disease (GBD) as an illustration, it is estimated that there will be 1.2 million deaths each year, 19 million DALYs, and about the same number of years lost to reduced glomerular filtration rates. In addition, renal failure was the cause of death for 1.2 million people in 2015, up 32% from 2005. An estimated 2.3–7.1 million people with ESRD died in 2010 due to a lack of access to chronic dialysis (Covella *et al.*, 2019). Also, 1.7 million people are thought to die annually as a result of acute renal impairment (Luyckx *et al.*, 2018). It's estimated that about five to ten million individuals are dying annually because of renal disease (Bikbov *et al.*, 2020). Renal hemodialysis is a treatment option for people with end-stage renal disease (Palsson & Waikar, 2018). Renal failure that has persisted for a long time (hemodialysis patients'), a progressive ailment, is characterized by functional and structural kidney abnormalities from a variety of causes. A decrease in kidney function is indicated by a higher estimated glomerular filtration rate (eGFR) (Luyckx & Brenner, 2020). Oxidative stress has a significant role in the development of DN and its progression to ESRD (Pisoschi *et al.*, 2021).

Thyroid dysfunction

Thyroid hormones affect renal function directly and indirectly. Cardiovascular and renal blood flow mediate indirect effects. Direct impacts are on glomerular filtration rate, tubular secretion and absorption, and hormones. Thyroid hormone promotes sodium reabsorption and proximal convoluted tubule Na/K/ATPase activity. Thyroid hormones affect tubular calcium and potassium reabsorption. Thyroid hormones also release renin via adrenergic regulation. (Shrivastava & Khare, 2023).

Triiodothyronine T3

Controlling metabolism, development, and protein synthesis requires the hormones triiodothyronine (T3) and thyroxine (T4). Furthermore, renal disease may be significantly impacted by these hormones. T4 production is limited to the thyroid gland. Furthermore, most of T3 and reverse T3 (rT3) are produced by the peripheral enzymatic deiodination of T4 in the liver, kidney, skeletal muscle, heart, and brain. Kidneys are required for thyroid hormone metabolism, breakdown, and removal. Thyroid dysfunction takes on distinct characteristics in patients with severe kidney disease. (Mohamedali *et al.*, 2014). Hypothyroidism refers to a state in which there are lower-than-normal levels of circulating thyroid hormones. Due to the fact that the hypothalamic and pituitary systems control the thyroid, hypothyroidism may develop if any of

these systems malfunction. Primary hypothyroidism can be identified by a concentration of thyroid-stimulating hormone that is above the reference level but below the recommended ranges and a thyroxine-free level that is below the reference level. When the malfunction occurs on hypothalamic neurons or in the pituitary gland, the condition is known as central hypothyroidism (Badiu, 2018) However, the kidneys are crucial in the breakdown and elimination of thyroid hormones. The term "chronic kidney disease" (CKD) refers to a group of conditions characterized by abnormal kidney function and a gradual decrease in glomerular filtration rate (GFR)(Tippannavar & Shekhanawar, 2022). According to the findings of a number of studies, serum levels of thyroid hormones are frequently abnormal in patients who are receiving regular maintenance hemodialysis.(Kopple et al., 2005). Decreases in glomerular filtration rate are associated with an increase in the prevalence of primary hypothyroidism, most often in the form of CKD(Iglesias & Díez, 2009). The kidney's glomerular filtration normally removes iodine. Thus, advanced renal failure decreases iodide excretion, which increases plasma inorganic iodide and thyroidal uptake (Tippannavar & Shekhanawar, 2022). Chronic kidney disease (CKD) interferes with thyroid function in a number of ways. These include lowering blood levels of thyroid hormone, decreasing its ability to bind to proteins, and upsetting the thyroid's ability to store iodine. The hypothalamic-pituitary-thyroid axis is also disrupted in chronic kidney disease. Thyroid hormones are thus altered in CKD(Khatri *et al*., 2019). The kidney is primarily responsible for controlling the metabolic processes, breakdown, and elimination of thyroid hormones (Tippannavar & Shekhanawar, 2022). Renal failure-related hyperthyroidism is defined by an elevated T4 level and a decreased TSH level (Bichari et al., 2020)

Thyroxin T4

Under strict brain control, the thyroid gland produces and secretes triiodothyronine (T3) and thyroxine (T4), which account for 85–90% for T3 and 10-15% for T4 of the circulating thyroid hormone. The hypothalamic-pituitary-thymic axis and thyroid hormone regulate the body's rate of energy (calorie) consumption, affecting every cell and organ in the body. This is known as the metabolic rate, and it influences weight growth or loss via varying the heart rate. Thyroxine (T4) is converted to triiodothyronine (T3) by de-iodination once it has been released into the bloodstream by the thyroid. This is because T3 is more efficiently used by cells with thyroid hormone receptors than T4. As a result, T4 is commonly regarded as the inactive version of thyroid hormone, while T3 is regarded as the active type (Rana *et al.*, 2022).

Thyroid-stimulating hormone TSH

Thyroid-stimulating hormone is producing by anterior pituitary gland produces. It is a glycoprotein hormone and single most critical stimulus for thyroid hormone production by the thyroid gland.

This leads to thyroid hypertrophy due to the proliferation of thyroid follicular cells. Regulation of TSH production occurs at the hypothalamic-pituitary axis level.
Thyroid-stimulating hormone (TSH) stimulates thyroid follicular cells to produce T3 and T4. The active ingredient is a thyroid hormone called T3. Only 20% of the body's hormones are actually used to produce T3, with the vast majority coming from peripheral T4 to T3 conversion. More than 80% of hormone secretion is due to T4 (tetraiodothyronine), generally known as thyroxine. Discharge into circulation causes de-iodination, leading to the production of T3. TSH secretion is upregulated when T3/T4 levels are low and downregulated when they are high in the anterior pituitary due to a negative feedback loop involving T4 and T3. Parathyroid hormone After being released into circulation, it undergoes de-iodination and gives rise to T3. When T3/T4 levels are low, the anterior pituitary produces more TSH, and when T3/T4 levels are high, the pituitary produces less TSH.

Parathyroid dysfunction

PTH regulates calcium and phosphate homeostasis, making it a key regulator of bone and mineral metabolism. PTH is both synthesized and cleaved in the parathyroid gland. Polypeptide synthesis for PTH occurs in the endoplasmic reticulum, where a 115-amino-acid precursor is first cleaved in two to yield a 90-amino-acid pro-PTH. Secretory granules in the parathyroid gland store the mature, full-length PTH, which consists of 84 amino acids and is activated after being cleaved from pro-PTH. It has been estimated that the entire PTH synthesis, cleavage, and storage process takes less than an hour. PTH is secreted into the extracellular space when storage granules fuse with the outer membrane. Rapidity and regulation by [Ca2+]e in the extracellular environment have been demonstrated for this mechanism in vitro(Jacquillet & Unwin, 2019).

When extracellular [Ca2+] rises from 0.5 to 2.0 mM, PTH secretion is 50% suppressed. When PTH enters the bloodstream, the liver and kidneys quickly flush it out of the body. Due to its short half-life and rapid degradation, mature PTH is largely regulated at the gene expression level (Jacquillet & Unwin, 2019).

Parathyroid hormone regulates cellular and molecular processes that have far-reaching effects on skeletal homeostasis. Despite an increase in bone formation by osteoblasts, persistent hyperparathyroidism results in a net loss of bone mass. The only method of treatment for bone anabolic osteoporosis is intermittent PTH analog therapy. From primitive skeletal stem cells to mature, matrix-embedded osteocytes, the osteoblast lineage is replete with functional PTH receptors. Furthermore, osteoclast-mediated bone remodeling releases dormant growth factors within the bone matrix (Wein & Kronenberg, 2018).

Objective:

The aim of this study was to evaluate the activities of Triiodothyronine Hormone (T3), Thyroxine (T4) Hormone (T4) and Thyroid stimulating hormone (TSH), and serum Parathyroid Hormone (PTH) levels with thyroid dysfunction in hemodialysis patients and compare them with control groups.

Prevalence:

the number of patients suffering from Hemodialysis patients (HD) has been on the rise, affecting an estimated 843.6 million people over the world in 2017.1 (Jager *et al.*, 2019).

Methodology:

This is a case-control study that included 75 healthy people as controls and 75 patients who attended a dialysis center in Basrah Teaching Hospital, Basrah City, southern Iraq, through the period of November 2022 to May 2023. All patients were diagnosed and confirmed by a specialist physician. Other diseases that can cause a similar clinical picture to chronic kidney disease, like sepsis-infected double immune, catheter-critical, chest infection, hepatitis C, B infectious disease, hepatitis, and chronic liver disease, were excluded. Patients and controls in this study ranged in age from 17 to 67 years. Participants' demographic data, including age, sex, and clinical findings, were collected using a standardized questionnaire. After collecting 5 ml of venous blood from each of the 150 participants (75 patients and 75 controls), the blood was centrifuged to obtain serum for the measurement of thyroid function tests (T3, T4, and TSH) by VIDS ,Serum electrolytes and kidney function tests (urea, Cr, Ca+, and PO4) were measured using Cobas C111, and K+ was measured using ABL800 Flex.

Statistical Analysis

The statistically significant differences were determined using SPSS (version 26).

Results:

The total number of samples is 150, divided mainly into 75 patients and 75 healthy people. In terms of sex distribution, the patient group study included 40 males (53.3%) and 35 females (46.7%). The control group included 75 (53.3%) males and 35 females (46.7%). as shown in **Figure 1**.

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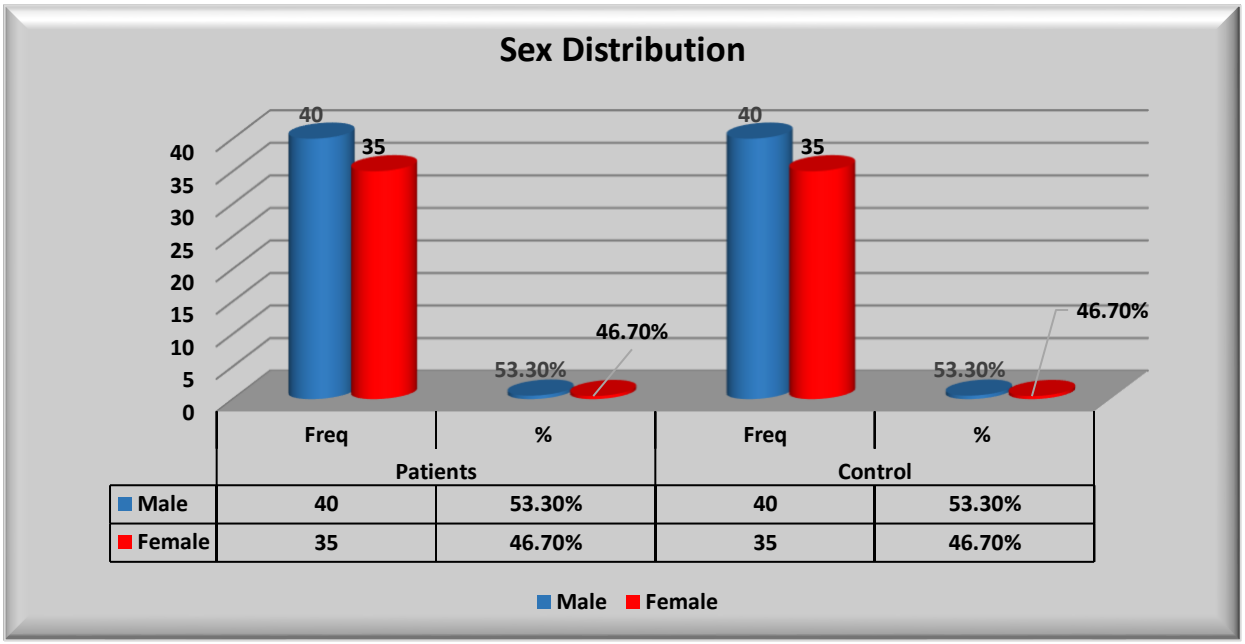
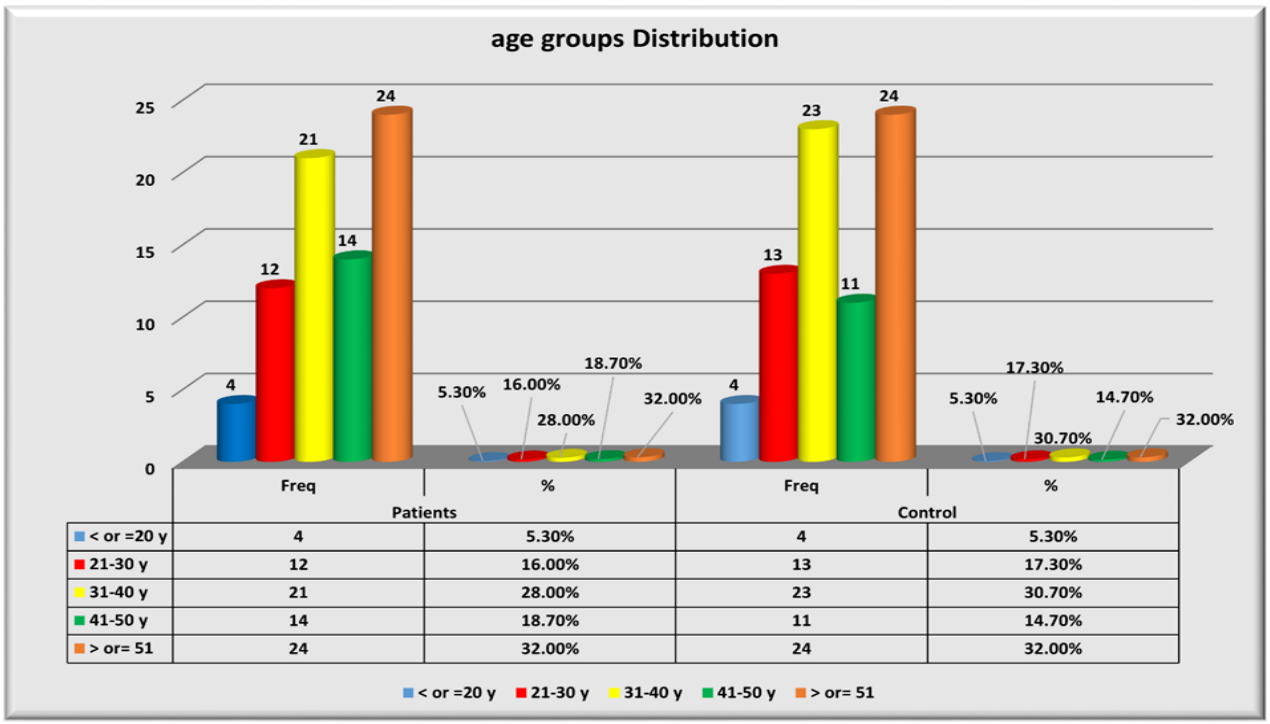


	Patients		Control	
	Freq	%	Freq	%
Male	40	53.30%	40	53.30%
Female	35	46.70%	35	46.70%

Figure (1): Sex distribution of the study groups

The classification of both study groups according to age shows the most patients with an age of more than 51 years, and another detail is shown in **Figure 2**



	Patients		Control	
	Freq	%	Freq	%
< or =20 y	4	5.30%	4	5.30%
21-30 y	12	16.00%	13	17.30%
31-40 y	21	28.00%	23	30.70%
41-50 y	14	18.70%	11	14.70%
> or= 51	24	32.00%	24	32.00%

Figure (2): Study group distribution according to the age group

According to **Table (1)** There is no statistically significant difference observed between the healthy and patient groups in relation to age and sex (P = 0.778, P = 1.0), respectively.

Table (1) Statistical distribution of study groups by their age and sex duration of study.

			Sex		Total	Age (Years)
			Male	Female		Mean ±SD
Study Groups	Patients	Freq	40	35	75	41.84±12.86
		%	53.3%	46.7%	100.0%	
	Control	Freq	40	35	75	41.24±13.1
		%	53.3%	46.7%	100.0%	
P .Value			1.000			0.778

- Chi-square and independent t-test was used

Table 2 included the study of thyroid function tests (T3, T4, and PTH) between the two groups, which showed a statistically significant increase in TSH and PTH in patients compared with the control (=0.000). The test of T3 and T4 showed a decrease in hemodialysis patients (p = 0.000).

Table (2): Comparison of thyroid markers among the control and hemodialysis patients' groups

Variables	Study Groups	Mean ±SD	P.Value
T3	Patients	0.4585±0.33	0.000
	Control	1.8396±0.41	
T4	Patients	8.8451±1.077	0.000
	Control	11.4057±1.79	
TSH	Patients	3.02761.008	0.000
	Control	1.1091±.64	
PTH	Patients	204.275±46.05	0.000
	Control	172.680±40.15	

- independent t test or Mann-Whitney U test were used

Table (3) include the study of electrolytes (phosphate, potassium, and calcium) and renal function tests (urea and creatinine). The study showed a statistically significant increase in phosphate and potassium in patients (p = 0.000), while decreasing calcium in patients. Creatinine and urea also showed a significant increase in patients compared to control (p = 0.000).

Table (3): Comparison of electrolyte, urea, and creatinine among the control and hemodialysis patients' groups

Variables	Study Groups	Mean ±SD	P.Value
Phosphate	**Patients**	**6.1252±1.37**	**0.000**
	Control	**3.9321±0.45**	
Potassium	**Patients**	**4.8493±1.11**	**0.000**
	Control	**3.8284±0.29**	
Calcium	**Patients**	**6.8672±0.77**	**0.000**
	Control	**8.8013±0.57**	
Urea	**Patients**	**97.4959±15.48**	**0.000**
	Control	**25.9880±3.68**	
Creatinine	**Patients**	**8.1877±1.03**	**0.000**
	Control	**0.8039±0.09**	

Independent t test or Mann-Whitney U test were used

Discussion

The results of this study concur with previously reported results of several studies, as in the following: (Cachat *et al*., 2015;Fedak *et al*., 2016; Pasala & Carmody, 2017 ;Vanholder *et al*., 2018; Vega *et al*., 2020a; Levey *et al*., 2020). Results from our study agree with those from other studies (Kashif *et al*., 2023;(Kachhawa & Sinha, 2019) There is a difference of opinion among researchers (Disease, 2023). This may be because models were collected for various kidney failure stages, while our study used CKD's final stages.

Thyroid dysfunction

There are a variety of disorders that can affect the thyroid gland and cause either excessive or insufficient thyroid hormone production (hyperthyroidism). Thyroid disorders have the potential to impact various bodily functions, including heart rate, mood, energy level, metabolism, bone health, and pregnancy, among others (Balwan & Kour, 2022). Epidemiological studies indicate a significantly elevated prevalence of thyroid functional disease, specifically hypothyroidism, among individuals with CKD and ESRD in comparison to the general population(Rhee *et al*., 2015).

Iodine plays a crucial role in the production of thyroid hormones. Inadequate or excessive consumption of iodine can potentially lead to thyroid disorders. Insufficient consumption of iodine can lead to a decrease in thyroid function, resulting in a range of conditions collectively known as iodine deficiency disorders (IDDs). These disorders include goiter, cognitive impairment, and

congenital abnormalities (Scherbaum, 2023). Thyroid illness can result from poor or high iodine intake. Insufficient iodine intakes diminish thyroid function and cause goiter, cognitive impairment, and congenital abnormalities, known as IDDs (Bertinato, 2021).

Triiodothyronine Hormone

The thyroid gland produces the thyroid hormone, which is a molecular structure with three iodine molecules attached. The significance of this factor lies in its impact on essential physiological functions within the body, including but not limited to temperature regulation, growth, and heart rate (Balwan & Kour, 2022).

This study agrees with several previous studies (Mehsen *et al*., 2020) Research has demonstrated that inflammation can lead to changes in the metabolism of thyroid hormones in both peripheral and central systems, specifically the hypothalamic-pituitary-thyroid axis (Abdel *et al*., 2023). Deiodination shows a decrease in individuals with uremia, nonthyroidal diseases, malnutrition, inflammation, and the usage of specific medications such as glucocorticoids (Rashighi & Harris, 2017).

The low T3 levels can be attributed to several factors, including decreased conversion of T4 to T3 in the peripheral tissues due to conditions like uremia, malnutrition, inflammation, and mild illness. Furthermore, T3 binding to thyroid hormone nuclear receptors and T3-induced transcriptional activation may be reduced (Rhee *et al*., 2015).

Thyroxine (T4) Hormone

This hormone, which the gland primarily produces, is essential for controlling basic metabolic processes. This hormone is detected in the bloodstream and is associated with a protein known as thyroxine-binding globulin (TBG). Free thyroxine, also known as thyroid hormone or FT4 is the sole hormone that can be entered.

This study agree with several previous studies (Kachhawa & Sinha, 2019) (Kashif et al., 2023) The low levels of T4 are altered as a result of impaired binding between hormones and proteins, which can be associated with conditions such as uremia, low protein states, and the use of certain medications. The cellular uptake of T4 is impaired. Reduced T4 due to hypoalbuminemia or other low-protein conditions (Rhee *et al*., 2015).

Thyroid stimulating hormone

(TSH) is a hormone produced by the pituitary gland; its function is to stimulate the thyroid gland, which sits at the base of the neck, to create its own hormones, such as T3 and thyroxine(Disease, 2023).

This study agree with several precious studies (Khatiwada *et al*., 2015). With a high TSH level, clearance is reduced, although the levels are usually within the normal range. The response to TRH (thyrotropin-releasing hormone) is lessened. There is a decrease in plasticity. The half-life is increased, and the process of glycosylation is impaired. The high TSH level clearance is reduced, although the levels are usually within the normal range. The response to TRH (thyrotropin-releasing hormone) is lessened. There is a decrease in plasticity. The half-life is increased, and the process of glycosylation is impaired (Rhee *et al*., 2015). TSH levels in hemodialysis may be reduced because clearance is reduced, although levels generally remain within the normal range. The response to TRH (thyrotropin-releasing hormone) is diminished, pulsatility is decreased, the half-life is increased, and glycosylation is impaired (Rhee *et al*., 2015). On the other hand, according to points of view(Disease, 2023), He disagrees with us; however, the difference in measurements between what we found and what they found may be attributable to the fact that they chose patients who were in varying stages of renal failure, whereas in our study, patients who were on dialysis were chosen to agree with the study (Kachhawa & Sinha, 2019).

Parathyroid Hormone

Is a hormone that plays an important part in regulating calcium and phosphate levels in the body. Chief cells found in the parathyroid glands are the main source of it. PTH plays an essential role in bone and mineral metabolism by regulating calcium and Pi balance (Jacquillet & Unwin, 2019).

This study agrees with several previous studies (Dhillon-Jhattu et al., 2023) (Yamada & Nakano, 2023). Increased PTH secretion is observed when there is a decrease in serum Ca levels, an increase in serum phosphorus levels, or a decrease in serum vitamin D levels. Conversely, elevated levels of Ca or calcitriol in the blood suppress PTH secretion. The decrease in serum calcium levels that occurs, as a result, stimulates the secretion of PTH and had a role in the development of secondary hyperparathyroidism. The factors involved in the reduced response of the target organs to PTH include downregulation (Kritmetapak & Pongchaiyakul, 2019) .

Serum electrolytes

A hemodialysis machine filters the blood of wastes, salts, and fluids. Hemodialysis. When the kidneys fail, you may be able to live an active life with advanced kidney failure. If the kidneys can't filter waste and fluid from the blood, you need dialysis. This usually occurs when renal function is 10–15%. involves the supplementation of Ca and bicarbonate, as well as the removal of K, magnesium, urea, and other toxins through the process of diffusion. Ultrafiltration is a process that effectively eliminates water and Na (Atif, 2016).

Blood Urea

It is the major nitrogenous component of human and animal urine and the end product of protein metabolism. measures blood urea nitrogen. Blood waste, urea, and nitrogen are eliminated by the

kidneys (Ephraim & Jewell, 2021). Blood urea levels were found to be significantly higher in the CKD group compared to the control group (P<0.000).

In the hemodialysis patients' group, the levels of blood urea are significantly higher (P< 0.000) than the control group. This study agrees with several previous studies (N & Gb, 2023). This could be connected to generalized impairment in the proximal convoluted tubules' functions and impaired transport activities of the collecting tubules' epithelial cells. The non-protein waste product of creatine phosphate metabolism in muscles is creatinine, which is filtered by the glomeruli in the kidneys. Conversely, if there is a deficit in the renal filtration process as a consequence of decreased renal function, the amount of creatinine in the blood may rise. This rise in creatinine concentration is also due to the fact that creatinine is one of the metabolic by-products that is usually eliminated with urine, and in the event of renal failure, there is a reduction in GFR, which results in an increase in serum (Vega *et al*., 2020a).

Urea levels are markedly increased in hemodialysis patients', reaching pre-dialysis concentrations that can reach 10 times or more the upper limit of the normal range in patients with ESRD. It is used as a marker of uremic retention in hemodialysis patients and the adequacy of intradialytic solute removal (Vanholder *et al*., 2018) . The extracellular and intracellular fluids in the blood distribute urea, a byproduct of the liver's catabolism of proteins and amino acids, before the glomeruli filter it. A high blood urea nitrogen level is an indicator of renal function impairment because urea produced by the liver is filtered by the kidney (Mehan *et al*., 2017). Our study is consistent with the findings of the researchers (Kachhawa & Sinha, 2019;Laibi *et al*., 2023).

Creatinine

Creatinine is a byproduct of creatine metabolism and is considered a chemical waste product. The body naturally produces the chemical creatine, which primarily serves to give muscles energy. The purpose of this test is to evaluate the functionality of the kidneys. High levels may indicate kidney problems, while low levels may indicate muscular mass. Low levels may indicate liver or muscle dysfunction (Laibi *et al*., 2023). The current study found that creatinine levels in CKD patients are significantly higher (P > 0.000) than control when compared to the control group. This was one of the findings of the study. An elevated level beyond the normal range may be attributed to factors such as kidney damage or failure, infection, or diminished blood flow. Dehydration, which refers to the reduction of bodily fluids Muscle-related issues, such as the degeneration of muscle fibers, Our study aligns with the findings of the researchers (Ataei *et al*., 2014; Dwi Payana e*t al*., 2020; Hasani *et al*., 2022; Levey *et al*., 2020; Pasala & Carmody, 2017; Vega *et al*., 2020b, 2020a; Laibi *et al*., 2023)

Calcium

Calcium, a mineral, is commonly linked to promoting bone and dental health. However, it also serves crucial functions such as aiding in blood clotting, facilitating muscle contractions, and

regulating heart rhythms and nerve activities (Onose *et al*., 2023). Recent research found a substantial decrease (P<0.000) in blood Ca levels in CKD patients compared to the control group. The present study's findings also indicate that the levels of blood calcium in CKD patients are significantly lower (P <0.000). This finding agrees with (Wan et al., 2019). who found that serum Ca concentrations are lower in renal failure patients and that this decrease in serum Ca could be due to an increase in serum Pi because serum Ca and Pi concentrations have an inverse relationship and any increase in one will result in a decrease in the other (Perry & Salusky, 2011). Another possible cause for the decrease in serum calcium is a disturbance in vitamin D synthesis due to renal failure, which is caused by the kidney's failure to synthesize the active form of vitamin D (1,25-dihydroxycholecalciferol), which is very important for calcium absorption in the patient's intestine (Jameson *et al*., 2010).

Potassium

Potassium is an essential mineral required by all tissues in the body for proper functioning. The term "electrolyte" is used to describe this substance due to its ability to carry a minor electrical charge, which plays a crucial role in activating various cellular and neural processes (Akram e*t al.*, 2020). The study found a significant rise (P<0.000) in blood potassium levels in hemodialysis patients compared to the control group.

Total body K+ levels are mostly maintained by the kidney and vary from 3,000 to 4,000 mEq in a person weighing 70 kg. When tubular necrosis occurs in chronic kidney disease, it can seriously harm the collecting duct and late distal tubule. This harm directly affects the cells responsible for K+ secretion, which keeps K+ in the blood. Hyperkalemia is uncommon in CKD patients until GFR drops to 15–20 mL/min. It is possible to keep the plasma K+ concentration somewhat normal even in the face of a significant reduction in kidney mass because of an adaptively enhanced rate of K+ secretion in the remaining nephrons. This adaptation is thought to be similar to what happens to healthy people who consume a lot of K+ in their diet. Long-term K+ loading increases the secretory capacity of the distal nephron in animal models, resulting in increased kidney K+ excretion independent of plasma K+ concentration (Palmer & Clegg, 2019).

Phosphorus

The current research also discovered that CKD patients had a very significant rise (P<0.000) in blood phosphorus levels when compared to the control group. This finding is consistent with that of (Suki & Moore, 2016), who found that phosphorus is required for a variety of biological and metabolic processes essential to life, including the storage of energy for usage by all cells, including skeletal and heart muscles. There are many valid factors for chronic hyperphosphatemia in dialysis patients, including the following: Phosphate elimination during a single hemodialysis session is only 800 - 1000 mg. As a result, dialysis three times a week is insufficient to eliminate the required daily intake of phosphorus (1,000 mg/d) for dialysis patients. To preserve freshness,

many retail food items include highly accessible inorganic phosphate as a preservative. These, as previously stated, add to the patient's phosphate load. Patients undergoing dialysis must take a variety of medicines, and phosphate binders must be added to the patient's pill load. Phosphate binders are often big tablets that are difficult to swallow or may alter the perception of taste if chewed during a meal, making adherence difficult. Furthermore, these drugs often induce gastrointestinal problems and have a varied phosphate-binding capacity. Finally, two more significant variables have been identified, both of which are linked to the methods employed to treat these individuals. One is the use of large dosages of calcitriol or analogs, which have been shown to improve active phosphate absorption from the small intestine.

The mean blood phosphorus, also known as inorganic phosphate, or iP, remains relatively constant at 3.8 mg/dL in people with normal renal function, according to extensive cross-sectional population studies. When a patient's GFR drops below 30 mL/min/1.73 m2, which denotes Stage 4 CKD, it functions similarly in people with compromised kidney function. At this time, the patients' serum phosphorus levels start to rise and keep rising as they approach end-stage renal failure. (Suki & Moore, 2016)

Conclusion

In conclusion, thyroid and parathyroid dysfunction in hemodialysis patients is common and can have a significant impact on patient well-being and clinical outcomes. Timely diagnosis, regular monitoring, and a personalized treatment approach are essential for managing these conditions effectively in this patient population

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