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A Study of Thyroid Profile In Patients with Chronic Kidney Disease

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Abstract

Chronic kidney disease (CKD) is a non-communicable state characterised by a variety of physiological disorders, aberrant renal function that results in excretory, metabolic, and synthetic failure, and the accumulation of nitrogenous waste products, as well as a gradual decrease in glomerular filtration rate (GFR).1-3 CKD is defined as the presence of renal dysfunction, characterised by inappropriate albumin excretion or reduced kidney function, determined by measured or estimated glomerular filtration rate (GFR) 60 mL/min/1.73 m2 for 3 months or more 4-5. End-stage renal disease (ESRD) is a clinical condition in which the endogenous renal function has been irreversibly lost to the extent that the patient is permanently dependent on renal replacement treatment to prevent life-threatening uremia. Patients who are in stages 3 or 4 of chronic kidney disease are at a substantially higher risk of developing end-stage renal disease (ESRD) or death even before ESRD arises.9-10 The thyroid hormone regulates a variety of body processes, including growth and metabolism. The hypothalamic-pituitary-thyroid axis, which includes the thyroid gland, the anterior pituitary gland, and the hypothalamus, is a self-regulatory circuit. Tetraiodothyronine (T4) and triiodothyronine (T3) are the two primary hormones that the thyroid gland produces. To keep the right feedback mechanism and homeostasis, thyroid-stimulating hormone (TSH) from the anterior pituitary gland, thyroid-releasing hormone (TRH) from the hypothalamus, and T4 collaborate in perfect synchrony 11

1. The present study “A Study of Thyroid profile in patients of chronic kidney diseases” was carried out in the Department of Biochemistry, Muzaffarnagar Medical College, Muzaffarnagar in collaboration with Department of Medicine, on 100 subjects among them 50 cases and 50 controls, above the age group of 18 years (>18 years). 3.
2. Serum T3 and T4 levels were lowest, and TSH levels in cases as compared to controls, and the difference was statistically highly significant. And TSH levels were also higher in cases as compared to controls, and the difference was statistically highly significant.
3. Serum urea and creatinine levels were higher in cases as compared to controls, and creatinine clearance (CCL) levels were lower in cases as compared to controls, and the difference was statistically highly significant.
5. Serum levels of T3 and urea were weakly negatively correlated. And the difference was statistically significant.
6. Serum levels of T3 and creatinine were weakly negatively correlated, and the difference was statistically significant.
7. Serum levels of T3 and Creatinine clearance (CCL) were positively correlated, and the difference was statistically significant.

Keywords: NIL

Introduction

The kidney plays a vital role in the metabolism, degradation and excretion of thyroid hormones.13-14 The synthesis, secretion, metabolism, and degradation of thyroid hormones are disrupted by long-term and gradual deterioration of renal structure and function, such as in chronic kidney disease (CKD), which then manifests with various clinical syndromes of thyroid dysfunction.15-18 The kidney is the only other organ that competes with the thyroid for the removal of iodine from the body through glomerular filtration, making them closely related organs.

In the small intestine, dietary iodine is converted to iodine and absorbed. The kidney (80%) and thyroid (20%) are the primary organs responsible for removing circulating iodine from the blood.20 A higher plasma inorganic iodide content and an initial increase in thyroidal iodide uptake follow as a result of decreased iodide excretion in advanced renal failure.21 This rise in total body inorganic iodide can inhibit thyroid hormone production, which is known as the Wolff-Chaikoff effect.22-23 According to various research, the prevalence of thyroid dysfunction is found to range from 13% in early CKD to 70% in end-stage renal disease. 26. Prevalence of increased thyroid enlargement (goitre) has also been observed in end-stage renal disease (ESRD). .19

The present study was conducted in the Department of Biochemistry, Muzaffarnagar Medical College, Muzaffarnagar in collaboration with Department of Medicine, on 100 subjects among them 50 cases and 50 controls, above the age group of 18 years (>18 years). Each group was divided based on inclusion and exclusion criteria. Kidney disease cases were taken from the OPD of the Department of Medicine, and a random control was taken from the healthy subjects visiting, medical college, Muzaffarnagar.

Aims And Objectives

1. To evaluate the renal function and thyroid profile in patients with chronic kidney disease and compare the findings with normal healthy controls.
2. To assess the correlation between thyroid profile and chronic kidney disease patients.

Inclusion Criteria

1. Chronic kidney disease patients aged above 18 years (>18 years).
2. Kidney disease of 3 months or more than 3 months duration.
3. Written consent was obtained from each study participant.

Exclusion Criteria

These include-

1. Patients with a history of thyroidectomy.
2. Family history of thyroid disorder.
3. history of any anti-thyroid drugs or medications for thyroid disease.
4. History of burns or trauma.
5. Children.
6. Pregnancy.

Aims And Objectives

1. To evaluate the renal function and thyroid profile in patients with chronic kidney disease and compare the findings with normal healthy controls.
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Review Of Literature

Galeti EH et al. (2022) concluded that according to the severity of the renal failure, the number of patients increases as T3 and T4 levels decrease and thyroid-stimulating hormone (TSH) levels increase. Peters J et al. (2021) concluded that patients with severe kidney disease were 2.5 times more likely to develop low-T3 syndrome than those with normal kidney function. Keunmoe P et al. (2020) demonstrated that the spectrum of TD is enormous. The most prevalent thyroid dysfunctions are low T3, low FT3, hypothyroidism, and low T3 combined with low FT3. Garbadi SF (2020) showed that in children with CKD, the low T3 syndrome is the most prevalent thyroid condition, followed by subclinical hypothyroidism (11.8%).90

Pan B et al. (2019) demonstrated that Patients with CKD frequently experience ESS, particularly low T3 syndrome. Avasthi G et al. (2001) demonstrated that patients with chronic renal insufficiency experience thyroid dysfunction on both a clinical & biochemical level 102

Study Tools

One pre-tested questionnaire was used to find out the presence of chronic kidney disease in patients

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attending the medicine OPD in Muzaffarnagar Medical College, Muzaffarnagar. A self-designed, pre-tested questionnaire was used for the study. It contains three sections (A, B and C).

SECTION A: Personal details.

SECTION B: History of patients.

SECTION C: Laboratory investigations.

Collection And Processing Of Blood Sample

The venipuncture site was cleaned with 70% methylated spirit and allowed to dry. In total, 5mL of whole blood was collected from the antecubital vein of each participant with a disposable 5cc syringe and needle.

Biochemical Investigations

1. Serum Urea: estimated by the Urease method.
2. Serum Creatinine: estimated by Jaffe's method.
3. GFR: estimated by the Cockcroft and Gault formula.
4. CCr = (140 − age) × body weight/serum creatinine × 72 (× 0.85 if female).
5. Thyroid Profile:

T3 – By Electrochemiluminescence Immunoassay method (on Roche Cobas e411).

T4 - By Electrochemiluminescence Immunoassay method (on Roche Cobas e411).

TSH - By Electrochemiluminescence Immunoassay method (on Roche Cobas e411).

				Albuminuria Categories		
				A1	A2	A3
				Normal	Moderately increased (microalbuminuria)	Severely increased (macroalbuminuria)
				< 30 mg/g	30 mg/g – 299 mg/g	≥ 300 mg/g
GFR Categories (mL/min/1.73m²)	G1	Normal or high	≥ 90			
	G2	Mildly decreased	60-90			
	G3a	Mildly to moderately decreased	45-59			
	G3b	Moderately decreased	30-44			
	G4	Severely decreased	15-29			
	G5	Kidney Failure	< 15			

Low risk | Intermediate risk | High Risk | Very High Risk

Signs and Symptoms

Patients with chronic kidney disease often have signs and symptoms. These include- dry skin, sallow complexion, low temperature, cold intolerance, decreased basal metabolic rate, lethargy, fatigue, oedema and hyporeflexia, muscle cramps, loss of appetite, nausea, vomiting, sleep problems, swelling of feet and ankles, and high blood pressure (hypertension). These signs and symptoms are often nonspecific.36

Complications

Chronic kidney disease affects almost every organ system in the body, and its common complications

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include: abnormally high levels of metabolic waste products like urea and creatinine, mineral bone diseases like hypocalcemia and hyperphosphatemia, dyslipidemia, thyroid dysfunction, anaemia, hyperlipidemia, nutrition, osteodystrophy, fluid and electrolyte abnormalities, hyperuricemia, metabolic acidosis, cognitive impairment and cardiovascular risk.37

Complications can develop at any phase, may result from adverse consequences of measures to prevent or treat the disease, and can result in mortality without progression to renal failure.

There are no specific kidney damage markers for hypertensive nephrosclerosis, although with the onset of decreased GFR, high-normal to high concentrations of albuminuria can develop.

Nephron Hypertrophy

Persistent increases in GFR (single-nephron) and filtration pressure (also known as glomerular hypertension) across the glomerular filtration barrier, which suggests glomerular hyperfiltration, cause residual nephron hypertrophy. Transforming growth factor and epithelial growth factor receptor55-56 are expressed as a result of glomerular hyperfiltration and glomerular hypertension.

Impaired Glomerular Filtration

Production of angiotensin II and mTOR signalling ultimately exacerbate podocyte loss and proteinuria by maintaining prolonged podocyte hypertrophy and glomerular hyperfiltration. The peptide hormone angiotensin II is a component of the renin-angiotensin system (RAS), which promotes aldosterone secretion and vasoconstriction (and, consequently, salt retention and an increase in blood pressure). In turn, aldosterone directly inhibits the glomerular barrier sieving function, possibly by repressing the expression of the podocyte protein nephrin.

Fibrosis

Interstitial fibrosis is one of the non-specific wound-healing reactions associated with nephron loss. Proximal tubular epithelial cells are activated by invading immune cells, albuminuria, and, in the case of diabetes, glucosuria.

Thyroid Hormone

The thyroid hormone regulates a variety of body processes, including growth and metabolism. The hypothalamic-pituitary-thyroid axis, which includes the thyroid gland, anterior pituitary gland, and hypothalamus, is a self-regulatory circuit. Tetraiodothyronine (T4) and triiodothyronine (T3) are the two primary hormones that the thyroid gland produces. To keep the right feedback mechanism and homeostasis, thyroid-stimulating hormone (TSH) from the anterior pituitary gland, thyroid-releasing hormone (TRH) from the hypothalamus, and T4 collaborate in perfect synchrony.11 Thyrotropin-releasing hormone (TRH) is produced in the hypothalamic paraventricular nucleus (PVN), where it stimulates the anterior pituitary gland's thyrotroph cells to produce and secrete thyroid-stimulating hormone (TSH). TSH then stimulates the synthesis and release of 3,5,3 5'-L-tetraiodothyronine (thyroxine, T4) and 3,5,3'-L-triiodothyronine (T4) by means of the TSH receptor (TSHR) on thyroid follicular cells. Only about 0.2% and 0.02% of the total T3 and T4 are present in plasma as free unbound hormones (fT3, fT4) because the majority of the circulating T4 and T3 is bound to carrier proteins such as thyroxine binding globulin, transthyretin, and albumin.12

Results

The present study was conducted in the Department of Biochemistry, Muzaffarnagar Medical College, Muzaffarnagar, in collaboration with the Department of Medicine, on 100 subjects, among them 50 cases and 50 controls, above the age of 18 years 18.

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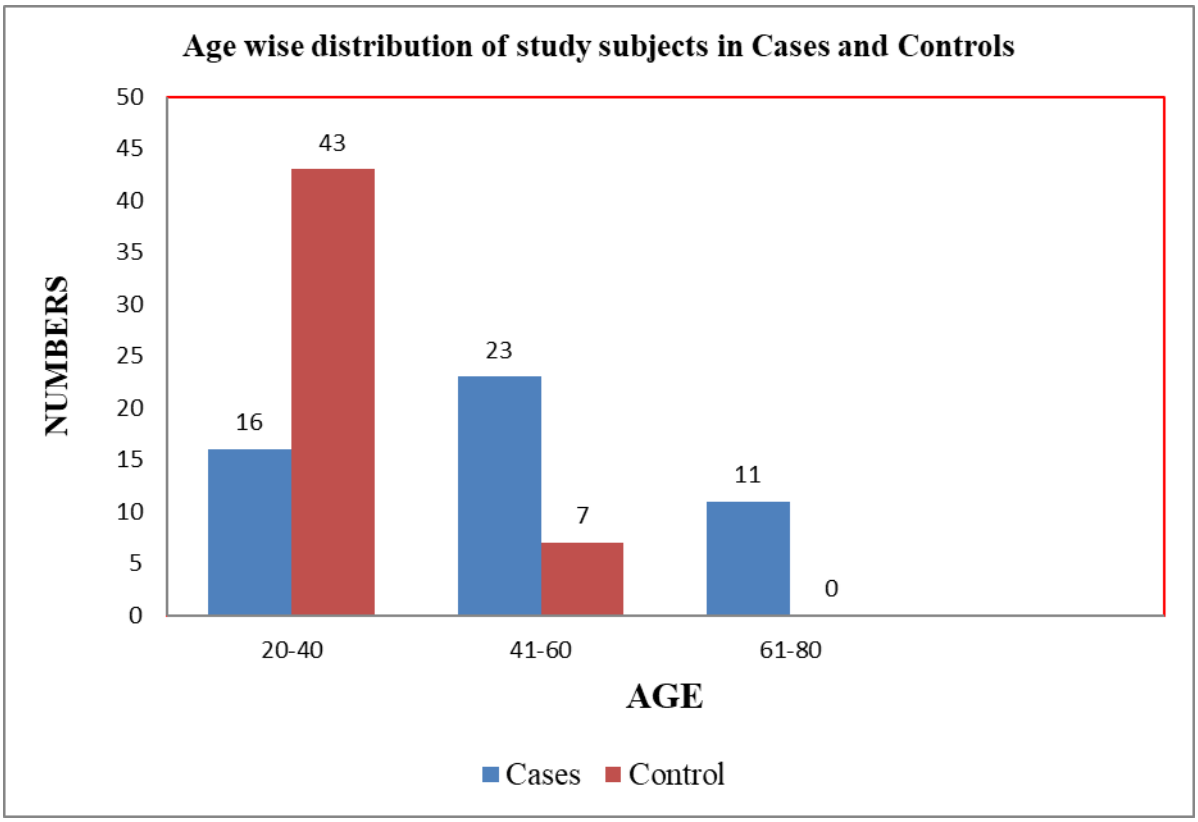
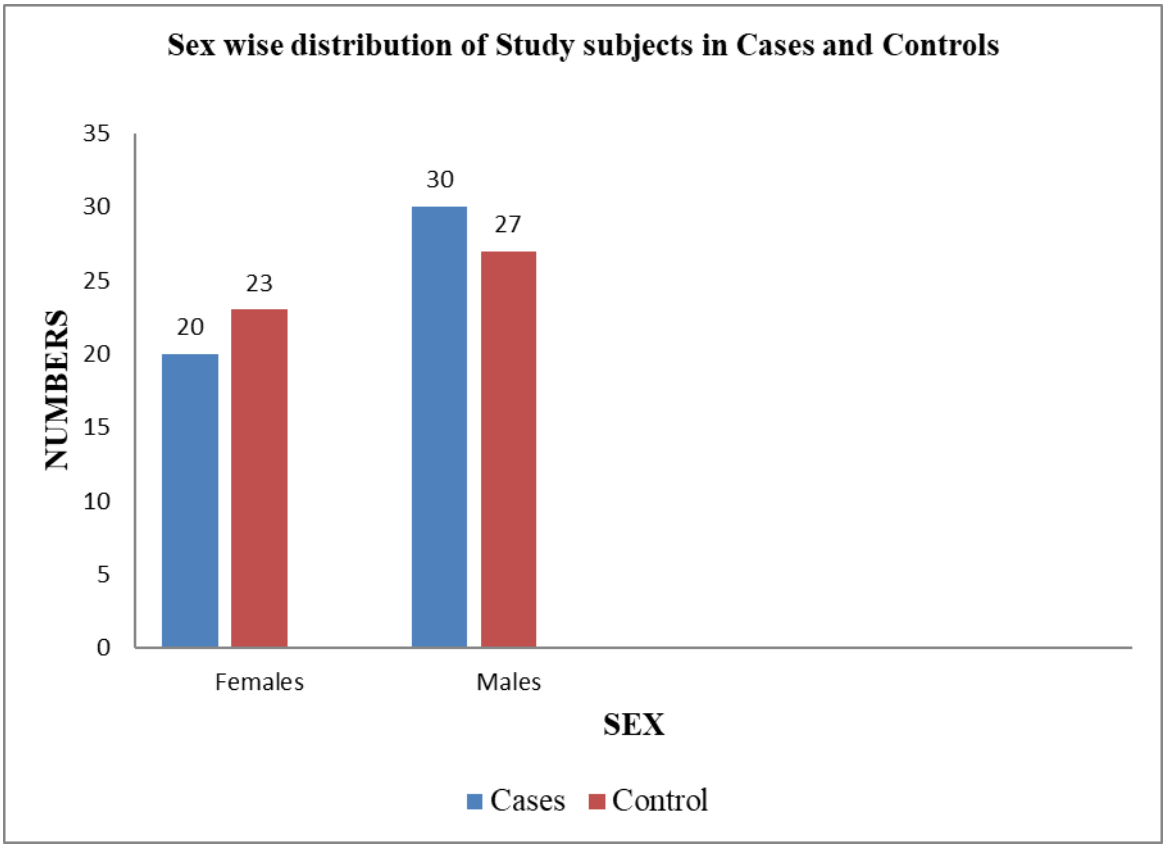


Figure 5: Age-wise distribution of study subjects in Cases and Controls



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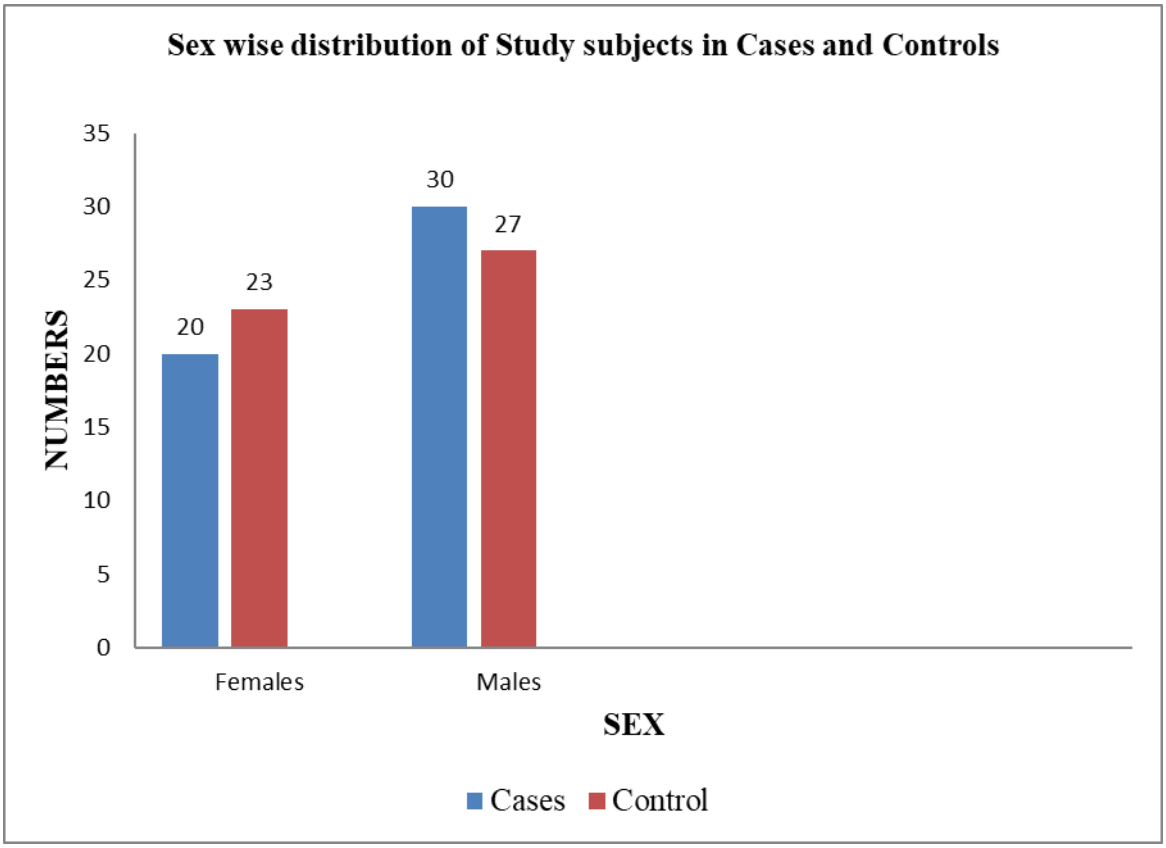

Table 1: Age and sex-wise distribution of study subjects in case and controls

Age in Years	Cases						Controls					
	Females		Males		Total		Females		Males		Total	
	No.	%age	No.	%age	No.	%age	No.	%age	No.	%age	No.	%age
20-40	4	20.0%	12	40.0%	16	32.0%	19	82.7%	24	88.9%	43	86.0%
41-60	8	40.0%	15	50.0%	23	46.0%	4	17.3%	3	11.1%	7	14.0%
61-80	8	40.0%	3	10.0%	11	22.0%	-	-	-	-	-	-
Total	20	100.0%	30	100.0%	50	100.0%	23	100.0%	27	100.0%	50	100.0%

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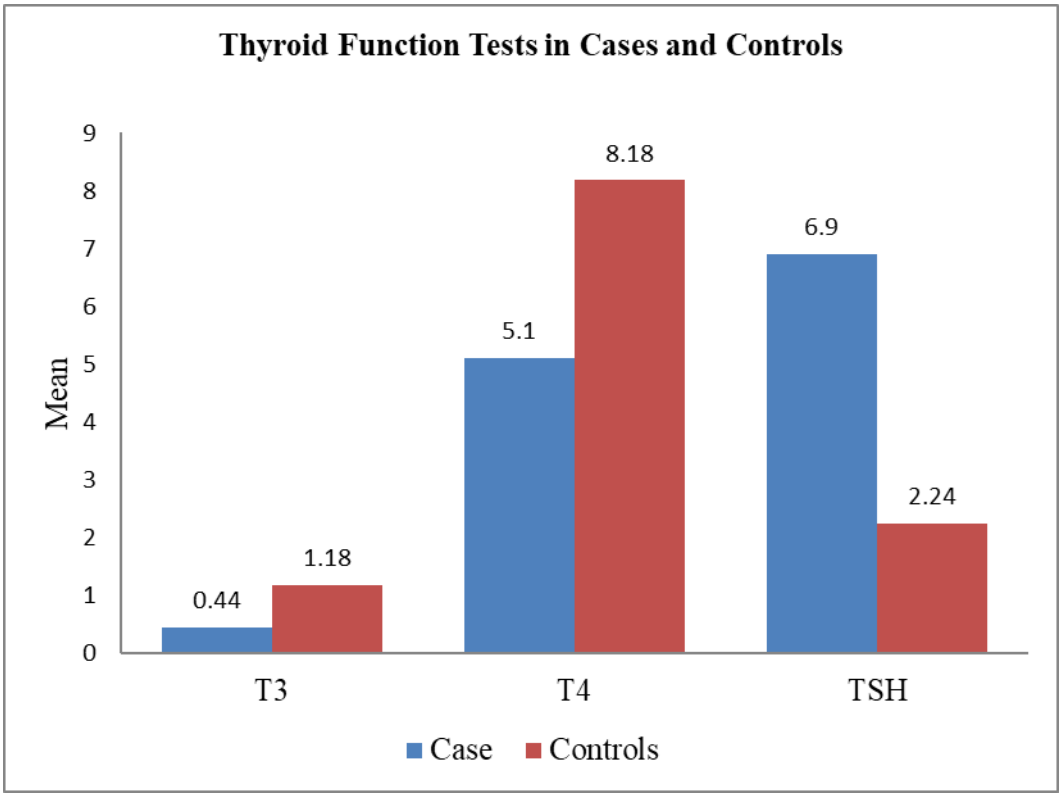

Table 3: Urea, Creatinine, Creatinine Clearance (CCL) in Cases and Controls

	Cases (Mean ± SD)	Controls (Mean ± SD)	P Value
Urea	111.04 ± 43.962	27.0 ± 5.907	0.001***
Creatinine	6.60 ± 2.857	1.00 ± 0.0000	0.001***
Creatinine Clearance (CCL)	13.20 ±5.729	110.84 ± 9.447	0.001***

Analysis of the above table 6 depicts:

1. Urea levels were significantly higher in cases as compared to controls.
2. Creatinine levels were significantly higher in cases as compared to controls.
3. Creatinine Clearance (CCL) levels were significantly higher in controls as compared to cases.

(***= Highly Significant)

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Table 2 Thyroid Function Tests in Cases and Controls

Thyroid Function	Cases (Mean ± SD)	Controls (Mean ± SD)	P Value
T3 (ng/ml)	0.44 ± 0.760	1.18 ± 0.388	0.001***
T4(µg/dl)	5.10 ± 2.150	8.18 ± 1.480	0.001***
TSH (µIU/ml)	6.90 ± 8.678	2.24 ± 1.001	0.001***

Discussion

The present study was conducted in the Department of Biochemistry, Muzaffarnagar Medical College, Muzaffarnagar in collaboration with Department of Medicine, on 100 subjects among them 50 cases and 50 controls, above the age group of 18 years (>18 years). Each group was divided on the basis of inclusion and exclusion criteria. Chronic kidney disease cases were taken from the OPD of the Department of Medicine, and a random control was taken from the healthy subjects visiting MMC.

Table 3 depicts that males more in number than compared to females. In the present study, 57% total population were males and 43 % were females.

Table 4 showed that males are more in between age groups of 20-40 and 41-60 years, i.e. males are 40% and 50% respectively. Females were more in between age group of 61-80 years. i.e, females were 40% in cases. In a study by Dr J. Punekar et al., the male-to-female ratio in the case group was 1.08:1.97. In a study conducted by Upendra Nath Gupta et al., there were 100 patients with CKD, 60 of which were men and 40 of which were women.93 and in a study by Md. Mosharruf Hossain et al. found that 63% of patients were men and 37% were women, resulting in a male: female ratio of over 1.7:1.104.

Table 5 depicts: Serum T3 and T4 levels are lower in cases as compared to controls, and the difference is statistically highly significant (p = 0.001***). Serum TSH levels were also higher in cases as compared to controls, and the difference was statistically highly significant. One study conducted by Shamsuddin M et al., Rajeev G et al., Kumar R, Lim VS found that on comparing cases (CKD patients) to controls (normal healthy individuals), there was a substantial decrease in serum T3 level, and T4 level, and a rise in TSH level, which is the same as in our study.106-107, 98,108 In our study mean serum T3 levels were 0.44 ± 0.760 in cases and 1.18 ± 0.388 in controls and the difference was highly significant p = 0.001***. Mean serum T4 levels were 5.10 ± 2.150 in cases, and 8.18 ± 1.480 in controls, and the difference was highly significant, p = 0.001***. Mean serum TSH levels were 6.90 ± 8.678 in cases, and 2.24 ± 1.001 in controls, and the difference was highly significant (p = 0.001***).

Table 6 showed that serum urea and creatinine levels were higher in cases as compared to controls, and creatinine clearance (CCL) levels were lower in cases as compared to controls, and the difference was statistically highly significant. Athab A (2016) observed that the hemodialysis group had higher blood urea levels than the controls. This is because urea, a nitrogenous basic metabolic waste product that is primarily produced by the liver and expelled from the body through urine, has a high concentration in the blood of chronic dialysis patients, and because kidney failure causes inadequate kidney function, which prevents urea from being excreted from the body.109 According to Alain and colleagues (2010), other factors besides renal function can affect the level of urea in blood serum, such as patients consuming more protein, a rise in the protein catabolic rate, dehydration, muscle damage from starvation, and, in some cases, chronic liver disease. Urea does not represent renal function in any of the pre-renal conditions mentioned above because the serum creatinine concentration is normal.

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In the present study, Table 7 (A) depicts the mean serum T3 level was 0.44±0.760 in cases, and the mean serum urea level was 111.04 ± 43.962 in cases; hence, Karl Pearson's correlation coefficient (r) was -0.287, so serum T3 and serum urea were weakly negatively correlated, but the difference was statistically significant (p=0.043**).

Table 7 (B) showed that the mean serum T3 levels was 0.44±0.760 in cases and the mean serum creatinine levels in cases was 6.60 ± 2.857, hence Karl Pearson's correlation coefficient (r) was -0.358, so serum T3 and serum creatinine were weakly negatively correlated and the difference was statistically significant (p=0.011**).

Table 7 (C) depicts mean serum T3 levels was 0.44±0.760 in cases and the mean serum creatinine clearance (CCL) levels in cases was 13.20 ± 5.729, hence Karl Pearson's correlation coefficient (r) was 0.525, so serum T3 and creatinine clearance (CCL) were positively correlated but the difference was statistically significant (p=0.00009****) which is consistent with Manickam K study which shows positive correlation between Total T3 and creatinine clearance and it is statistically significant p <0.005 which shows serum T3 levels were associated with the severity of CKD even in the normal TSH levels.95

Table 7 (D) depicts that the mean serum T4 levels were 5.10 ± 2.150 in cases, and the mean serum urea levels in cases were 111.04 ± 43.962; hence, Karl Pearson's correlation coefficient (r) was 0.123, so serum T4 and serum urea were weakly correlated, and the difference was statistically non-significant (p=0.394).

Table 7 (E) showed that the mean serum T4 levels were 5.10 ± 2.150 in cases, and the mean serum creatinine levels in cases were 6.60 ± 2.857; hence, Karl Pearson's correlation coefficient (r) was -0.172, so serum T4 and creatinine were weakly negatively correlated. And the difference was statistically not significant (p=0.232).

In the present study Table 7 (F) depicts the mean serum T4 levels was 5.10 ± 2.150 in cases and the mean creatinine clearance (CCL) levels in cases was 13.20 ± 5.729, hence Karl Pearson's correlation coefficient (r) was 0.114, so T4 and creatinine clearance (CCL) were weakly correlated. So the difference was statistically not significant (p=0.429), which is consistent with the study done by Kaptein EM et al. (1988). The study shows that T4 and creatinine clearance (CCL) were positively correlated, but it is not statistically significant.113 Another study done by Manickam K also shows a positive correlation between T4 and creatinine clearance (CCL) values, but the difference was statistically significant, p <0.05.96

Table 7 (G) in the present study showed that mean serum TSH levels in cases were 6.90 ± 8.678, and the mean serum urea levels in cases were 111.04 ± 43.962; hence, Karl Pearson's correlation coefficient (r) was 0.045, so TSH and serum urea showed negligible correlation, but the difference was statistically not significant (p=0.75624).

Table 7 (H) depicts that mean serum TSH levels in cases were 6.90 ± 8.678 and the mean serum creatinine levels in cases were 6.60 ± 2.857, hence Karl Pearson's correlation coefficient (r) was 0.251, so TSH and creatinine were weakly correlated, but the difference was statistically not significant (p=0.079).

In the present study, Table 7 (I) showed that the mean serum TSH levels in cases were 6.90 ± 8.678, and the mean creatinine clearance (CCL) levels in cases were 13.20 ± 5.729; hence, Karl Pearson's correlation coefficient (r) was -0.181, so TSH and creatinine clearance (CCL) were weakly and negatively correlated. And the difference was statistically not significant (p=0.209).

Summary

The present study "A Study of Thyroid profile in patients of chronic kidney diseases" was carried out in the Department of Biochemistry, MMIMSR in collaboration with Department of Medicine, M.M Institute of Medical Sciences and research, Mullana, Ambala on 100subjects among them 50 diagnosed CKD patients along with 50 healthy controls, above the age group of 18 years (>18 years). Each group was divided based on inclusion and exclusion criteria. Chronic kidney disease cases were taken from the OPD of the Department of Medicine, and random controls were taken from the healthy subjects visiting MMU. Both cases and controls were subjected to estimation of Thyroid profile, serum urea, serum creatinine and creatinine clearance (CCL), besides routine general physical examination and investigation. The results thus obtained were

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statistically analysed, compared and correlated. THE FINDINGS ARE SUMMARIZED AS UNDER.

1. Subjects ranged above 18 years (>18 years) of age among both cases and controls.
2. The majority of subjects in both cases and controls were males, 57 (57%) and the majority of subjects were in between age group of 20-40 years, 59 (59%), in both cases and controls.
3. Serum T3 and T4 levels were lower in cases as compared to controls, and the difference was statistically highly significant. And TSH levels were also higher in cases as compared to controls, and the difference was statistically highly significant.
4. Serum urea and creatinine levels were higher in cases as compared to controls, and creatinine clearance (CCL) levels were lower in cases as compared to controls, and the difference was statistically highly significant.
5. Serum levels of T3 and urea were weakly negatively correlated. And the difference was statistically significant.
6. Serum levels of T3 and creatinine were weakly negatively correlated, and the difference was statistically significant.
7. Serum levels of T3 and Creatinine clearance (CCL) were positively correlated, and the difference was statistically significant.
8. Serum levels of T4 and urea were weakly correlated, and the difference was statistically nonsignificant.
9. Serum levels of T4 and creatinine were weakly negatively correlated. And the difference was statistically not significant.
10. Serum levels of T4 and creatinine clearance (CCL) were weakly correlated. And the difference was statistically not significant.
11. Serum levels of TSH and urea show negligible correlation. And the difference was statistically not significant.
12. Serum levels of TSH and creatinine were weakly correlated. And the difference was statistically not significant.
13. Serum levels of TSH and creatinine clearance (CCL) were weakly negatively correlated. And the difference was statistically not significant.

Conclusion

This study was conducted among the CKD patients to show that thyroid dysfunction is an additional risk factor in CKD patients who fulfilled all the inclusion and exclusion criteria, and reveals the significant association between CKD progression and thyroid dysfunction and mean serum levels of T3, T4, decrease and TSH increase significantly in cases as compared to controls. In patients with low GFR, the levels of serum T3, T4 were found to be low. This shows a direct linear relationship between GFR and serum levels of T3, T4. Our data highlights that serum levels of T3 and T4 are positively correlated and weakly correlated, respectively, with the creatinine clearance (CCL) and T3 and T4 are directly proportional to the creatinine clearance (CCL), and TSH is weakly negatively correlated with creatinine clearance (CCL). Hence, a routine thyroid function status should be evaluated in each and every patient of CKD to reduce the morbidity and mortality rate of CKD patients, as well as reduce the social burden and health expenditure. Further studies for improving the clinical and biochemical criteria to diagnose thyroid dysfunction in CKD patients are needed.

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