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Original Research Article

A STUDY OF THYROID PROFILE IN CHRONIC KIDNEY DISEASE PATIENTS AT A TERTIARY CARE HOSPITAL IN KUMAON REGION OF UTTARAKHAND

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Abstract

Background: To study thyroid profile in chronic kidney disease patients at a tertiary care hospital in kumaon region, Uttarakhand. **Materials and Methods:** This cross-sectional study was carried out in the department of General Medicine, Government Medical College and associated Dr Susheela Tiwari Government hospital Haldwani, Nainital. All patients underwent the investigations for Urine (Albumin by dipstick method and Microscopy) and Biochemical investigations (Hemoglobin, Blood urea, Serum creatinine, Serum electrolytes, Serum proteins, Serum albumin, Serum calcium, Serum phosphorus, Serum ALP and Blood sugar levels. **Result:** The incidence of thyroid dysfunction in chronic kidney disease in this study was 70%. Majority of patients had subclinical hypothyroidism in the age group of 31-40 years (28.7%) and maximum patients of overt hypothyroidism were in 51-60 years (30.76%). Patients of all Stages (I to V) of chronic kidney disease were included with maximum cases lying in Stage II (34%). Significant alteration in thyroid parameters was seen in chronic kidney disease in all the stages. **Conclusion:** Chronic kidney disease and hypothyroidism may exhibit overlapping symptom complexes. Hence it is suggested to assess the renal status of the patient at the time of diagnosis of Hypothyroidism and to have a strict observation of these parameters in follow-up phase.

INTRODUCTION

Chronic kidney disease (CKD) is defined as persistent kidney damage accompanied by a reduction in the glomerular filtration rate (GFR) and the presence of albuminuria.[1] Chronic kidney disease is a worldwide health problem with increasing incidence and prevalence, poor outcome, and high cost of investigation and treatment. The kidney normally plays an important role in the metabolism, degradation, and excretion of thyroid hormones.[2]

All levels of the hypothalamic-pituitary-thyroid axis may be involved, including alterations in hormone production, distribution, and excretion. As a result, abnormalities in thyroid function tests are frequently encountered in uremia. End- stage renal disease (ESRD) alters the hypothalamic-pituitary-thyroid hormone axis in addition to the peripheral thyroid hormone metabolism. Among thyroid hormones, triiodothyronine (T3) is the most metabolically active thyroid hormone and can be reduced in ESRD patients even with a normal TSH level.[3]

In general, reduced T3 levels in ESRD patients are due to the decreased peripheral tissue conversion of T4 into T3, while thyroid gland production of T3 is normal and T3 clearance rates are normal or decreased, as in other non-thyroidal illnesses.[4] Most patients with end-stage renal disease have decreased plasma levels of free triiodothyronine (FT3), which reflect diminished conversion of thyroxine (T4) to T3 in the periphery.[5] This abnormality is not associated with increased conversion of T4 to the metabolically inactive reverse T3 (rT3), since plasma rT3 levels are typically normal. This finding differentiates the uremic patient from patients with chronic illness in which the conversion of T4 to T3 is similarly reduced, but the generation of rT3 from T4 is enhanced. In addition to decreased production, low levels of total T3 may also reflect reduced protein binding.[6]

Thyroid hormones are necessary for growth and development of the kidney and for maintenance of water and electrolyte homeostasis.[7] The kidney is closely related to the thyroid as it is the only other organ that competes with base iodine clearance.8

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The kidney normally contributes to the clearance of iodine, primarily by glomerular filtration. Thus iodide excretion is diminished in advanced renal failure, leading sequentially to an elevated plasma inorganic iodide concentration and an initial increment in thyroidal iodide uptake. Increased total body inorganic iodide can potentially block thyroid hormone production by affecting the pituitary-thyroid axis and peripheral metabolism of thyroid hormones. It has been shown that in chronic kidney disease (CKD), as the glomerular filtration rate (GFR) falls, there is a higher possibility of developing clinical and subclinical hypothyroidism (SCH).[8]

Hyperthyroidism can result in/accelerate chronic kidney disease (CKD) by several mechanisms. Firstly, hyperthyroidism results in intra-glomerular hypertension (increased filtration pressure) and consequent hyperfiltration. Secondly, hyperthyroidism predisposes to proteinuria, which is known to cause direct renal injury. Thirdly, hyperthyroidism-induced increased mitochondrial energy metabolism along with down-regulation of superoxide dismutase contributes to the increased free radical generation and consequent renal injury.[9] Oxidative stress also contributes to hypertension in hyperthyroidism, which contributes to CKD progression.[10]

The free T4 levels vary from being low to normal in CKD. This is primarily because of an impaired protein binding of T4 in CKD. The thyroid profile is similar to that observed in several non-thyroidal illnesses (NTIs) such as severe infections, heart failure, malignancies, and in several hospitalized patients without renal disease. This led to the consideration of a ―sick euthyroid state‖ in CKD, which is now called ―non- thyroidal illness.‖ However, unlike other NTI states, there is no increase in total rT3 levels in CKD.[11] This is due to an increased redistribution of rT3 into extravascular and intracellular spaces. In some patients, due to an impaired renal clearance, free rT3 levels may be mildly elevated.

Another difference from other NTIs is that the thyroid stimulating hormone (TSH) levels are elevated in CKD. However, TSH is released in response to thyrotropin releasing hormone (TRH) in CKD patients, indicating pituitary disturbances in uremia.[12] In addition, the circadian rhythm of TSH and its glycosylation is altered in CKD, compromising its activity. Thus, CKD patients have low T3 and normal or reduced T4 levels, and consequently elevated TSH and attendant increase in thyroid gland volume.[13]

These mechanisms are probably reflective of the physiological adaptation of the body to CKD to reduce the protein nitrogen turnover, reduce the protein catabolism and nitrogenous waste load. CKD results in reduced iodide excretion, which results in increased serum inorganic iodide level and the thyroid gland iodine content and consequent thyroid gland enlargement. Structural changes in thyroid among CKD patients include an increased prevalence of goiter (especially among women), thyroid nodules, and thyroid carcinoma, compared to general population.[14] Prevalence of Thyroid dysfunction in CKD is found to be ranging from 13% in early CKD to 70% in ESRD according to various studies.[7]

It is well known that CKD can induce a number of complications and comorbidities as here is a dearth of data in Indian scenario about the prevalence of thyroid dysfunction among CKD population we conducted the present investigation to study thyroid profile in chronic kidney disease patients at a tertiary care hospital in kumaon region, Uttarakhand.

MATERIALS AND METHODS

This cross-sectional study was carried out in the department of General Medicine, Government Medical College and associated Dr Susheela Tiwari Government hospital Haldwani, Nainital. Subjects were recruited from patients admitted in Medicine ward and OPD patients after obtaining written consent. The study was carried out from January 2021 to September 2022.

Study Population

The study included subjects aged more than or equal to 18 years and CKD was diagnosed on the basis of history, examination and on NKF (National Kidney Foundation) criteria, kidney disease of 3 or more than 3 months duration.

The study excluded Pregnant women, Severe cognitive, speech or hearing defects, Patients with acute kidney injury, Patients not willing to participate in the study and Known case of thyroid disorder.

Study tools

A written consent was taken from all potentially eligible subjects and subjects were excluded fromthe study if they were not matched with inclusion criteria of the study A structured questionnaire was used to collect information from each patient. The information obtained involved demographic data, age, occupation, mental status educational level, socioeconomic status (modified kuppuswamy‘s scale). Relevant history of duration, onset, initiation of disease, manifestation of disease including past, family and personal history was recorded The patients were subjected to lab and imaging investigation like serum creatinine, urea, Na, K, and Ca Proforma was prepared in English and Hindi to make it convenient for the population to communicate.

All patients underwent the investigations for Urine (Albumin by dipstick method and Microscopy) and Biochemical investigations (Hemoglobin, Blood urea, Serum creatinine, Serum electrolytes, Serum proteins, Serum albumin, Serum calcium, Serum phosphorus, Serum ALP and Blood sugar levels.

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Statistical analysis

SPSS version 25.0 analyzed the Excel data when it was loaded. Frequency and percentage analysis was used for categorical variables and mean and SD were used for continuous variables. To find the significance in categorical data ANOVA test was used. P value of < 0.05 was considered statistically significant.

RESULTS

In the present study amongst 150 patients with chronic kidney disease 66 had Subclinical Hypothyroidism, 39 patients had Overt Hypothyroidism and 45 patients had normal thyroid profile. Incidence of thyroid dysfunction in chronic kidney disease in this study was 70 %.

Table 1: showing the baseline characteristics of the study population

		Subclinical Hypothyroidism (66)		Overt Hypothyroidism (39)		Normal patients (45)	
		N	%	N	%	N	%
Age (in years)	≤30	6	9.1%	4	10.3%	6	13.3%
	31-40	19	28.8%	8	20.5%	2	4.4%
	41-50	18	27.3%	9	23.1%	7	15.6%
	51-60	17	25.8%	12	30.8%	24	53.3%
	>61	6	9.1%	6	15.4%	6	13.3%
	Mean age in years	45.98±12.54		48.51±13.41		50.48±12.03	
Gender	Males	38	57.6%	24	61.5%	29	64.4%
	Females	28	42.4%	15	3846.0%	16	35.6%
	Male: Female:	1.3:1		1.6:1		1.8:1	
Comorbidities	Hypertension	51	77.3%	30	76.9%	33	73.3%
	Diabetes mellitus	18	27.3%	18	46.2%	24	53.3%
	ADPKD	6	9.1%	0	0.0%	0	0.0%
	Chronic glomerulo nephritis	2	3.0%	4	10.3%	0	0.0%
	Obstructive uropathy	1	1.2%	4	10.3%	0	0.0%
	Multiple myeloma	0	0.0%	2	5.1%	0	0.0%
	Connective tissue orders	4	6.1%	8	20.5%	0	0.0%
	Unspecified	2	3.0%	2	5.1%	2	4.4%

In the present study 9% of the patients had subclinical hypothyroidism below the age of 30, 28.7% had subclinical hypothyroidism among 31-40 years, 27.2% had subclinical hypothyroidism among 41-50 years, 25.7% had subclinical hypothyroidism among 51-60 years and 9% had subclinical hypothyroidism among > 61 years.

In the present study subclinical hypothyroidism was seen in 57.57% of males and 42.42% in females. Overt hypothyroidism was seen in 61.5% of males 38.46% of females. 64.4% of males were normal whereas 35.55% females were seen to be normal.

Patients with Subclinical Hypothyroidism patients had Comorbidities like hypertension (77.27%), diabetes mellitus (22.27%), ADPKD (9%), Chronic glomerulo nephritis (3.03%), obstructive uropathy (1.15%), connective tissue disorders (6.06%) & unspecified conditions (3.03%).

Table 2: showing the laboratory parameters among study population

Parameter	Subclinical Hypothyroidism (66)	Overt Hypothyroidism (39)	Normal patients(45)	P value (ANOVA)
	Mean ±S.D	Mean ±S.D	Mean ±S.D	
Hb	8.18±1.71	7.9±1.56	8.36±1.46	0.410
B. Urea	156.14± 57.74	148.70±64.98	145.94±55.91	0.640
S. Creatinine	11.61±3.32	10.27±3.05	9.16±3.89	0.001
Creatinine clearance (ml/min)	6.05±1.75	6.84±2.17	7.57±3.12	0.004

The mean S. Creatinine was significantly more among patients with Subclinical hypothyroidism. The mean Creatinine clearance (ml/min) was significantly lesser among patients with Subclinical hypothyroidism.

Table 3: showing Laboratory parameters, Lipid Profile and Thyroid Profile among different stages of Kidney disease

		Stage I	Stage II	Stage III	Stage IV	Stage V	P value (ANOVA)
		Mean ±S.D	Mean ±S.D	Mean ±S.D	Mean ±S.D	Mean ±S.D	
Laboratory parameters	Hb	8.36±1.46	8.17±1.72	7.9±1.95	8.1±1.21	7.9±1.51	0.810
	B. Urea	145.94±55.91	160.75±59.97	158.40±63.56	140.65±57.12	110.5±49.02	0.260
	S. Creatinine	9.16±3.89	11.62±3.37	10.56±2.83	9.94±2.95	14.2±3.65	0.001*
	Creatinine clearance (ml/min)	8.00±3.19	6.13±1.82	6.15±2.18	7.39±1.64	4.8±1.48	0.001*
Lipid Profile	Total cholesterol	232.37±38.99	226.25±40.40	227.69±37.36	225.91±29.07	246.6±25.96	0.750

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	Triglyceride	177.37±20.55	171.05±24.02	175.26±32.91	216.95±113.99	188.6±26.14	0.007*
	HDL	36.2±5.75	37.58±7.11	37.57±5.26	36.43±7.97	37.6±3.91	0.820
	LDL	147.17±22.63	145.43±22.25	153.80±34.30	152.73±26.99	154.2±32.01	0.590
	VLDL	47.68±15.11	44.78±14.43	45.26±14.88	43.13±22.03	47±21.23	0.830
Thyroid Profile	T3	1.77±2.47	1.02±0.29	4.28±3.97	3.42±3.02	8.58±3.19	0.001*
	T4	6.92±2.47	8.46±2.41	5.41±2.40	4.61±1.44	3.82±0.41	0.001*
	TSH	6.98±17.15	7.30±0.8	51.25±40.07	48.96±18.84	79.76±9.02	0.001*

The mean S. Creatinine increased significantly from stage I to stage V. The mean Creatinine clearance (ml/min) decreased significantly from stage I to stage V.

No significant p value was seen in lipid parameters apart from triglycerides which had p value of 0.0073. The significant alteration in thyroid parameters was seen in chronic kidney disease in all the stages where p value<0.001.

Table 4: showing

	STAGE I	STAGE II	STAGE III	STAGE IV	STAGE V
Subclinical Hypothyroidism	0	51	12	3	0
	0.0%	100.0%	46.2%	13.0%	0.0%
Overt Hypothyroidism	0	0	14	20	5
	0.0%	0.0%	53.8%	87.0%	100.0%
Normal thyroid	45	0	0	0	0
	100.0%	0.0%	0.0%	0.0%	0.0%
p-value	0.001*				

Thyroid dysfunction showed no significance according to the stage of chronic kidney disease. It was observed that 92.66% of CKD patients of II, III, IV and V underwent hemodialysis whereas 7.33% were not hemodialysed. 26.66% of the patients showed altered echotexture with normal kidney on USG, 11.33% showed features of obstruction, 16.66% had scarred kidney and 45.33% had small kidneys.

DISCUSSION

In the present study, 66 had Subclinical Hypothyroidism, 39 patients had Overt Hypothyroidism and 45 patients had normal thyroid profile. Incidence of thyroid dysfunction in chronic kidney disease in this study was 70%.

Hossain M et al,[3] reported that 61% patients of CKD were suffering from different thyroid abnormalities. Among total patients of thyroid illness 11% were suffering from primary hypothyroidism, 5% were subclinical hypothyroidism and 45% were of low T3 syndrome.

In a study by Chandra A,[15] subclinical and clinically apparent hypothyroidism have been reported to occur in 40% and 16% of patients, respectively, with CKD not requiring renal replacement therapy. A study done by Gupta UN et al,[16] demonstrated that there is 53% of prevalence of SCH and clinical hypothyroidism in chronic renal disease patients. It has been estimated that the prevalence of subclinical primary hypothyroidism ranges between 4% and 10% in the general population.[1,17]

The incidence of thyroid dysfunction in CKD patients is greater than that found among the general population.[18] High rate of thyroid dysfunction in CKD patients as observed in our study may also be due to high prevalence of thyroid autoimmunity in study population, excess iodine nutrition or iodine deficiency, and the inclusion of subjects with non-thyroidal illness.[19] CKD is associated with a higher prevalence of primary hypothyroidism, both overt and subclinical, but not with hyperthyroidism.[20]

In the present study, majority of patients had subclinical hypothyroidism in the age group of 31-40 years (28.7%) and maximum cases of overt hypothyroidism among patients were reported in the age group of 51-60 years (30.76%). Gupta UN et al,[16] reported that age varied from 22 to 72 years, the number of patients aged 30 or below were 27, between 31 and 60 years were 59, and that of 60 years and above were 14. Higher TSH levels are seen with increasing age. Hossain M et al.3 reported that 11% of patients were between the age of 20-29 yrs, 20% were between 30-39 yrs, 33% were between 40-49 yrs and 36% were between 50-60 yrs.

The mean age of patients enrolled in our study with subclinical hypothyroidism was 45.98 years, with overt hypothyroidism was 48.51 years and normal patients was 50.48 years. Chandra A,[15] found that the mean age was 55.9 years in case of subclinical hypothyroidism, 55.2 years for Primary hypothyroidism and 55.0 years for Non-hypothyroid. Hossain M et al,[3] stated that the mean age of the patients was 44.42 yrs.

In our study, Subclinical hypothyroidism was seen in 57.57% of males and 42.42% of females and 61.5% of males and 38.46% of females were having overt hypothyroidism. The Similar results were reported by Hossain M et al,[3] with male to female ratio of 1.7:1.

In current study, 77.27% patients with Subclinical Hypothyroidism and 76.92% patients with Overt Hypothyroidism also had hypertension. Subclinical primary hypothyroidism is most commonly caused by chronic autoimmune thyroiditis, which is typically characterized by a mild asymptomatic

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goiter with diffuse hypoechogenicity on thyroid ultrasound and by the presence of a high titer of serum thyroid autoantibodies.[24]

We observed that 22.27% patients with Subclinical Hypothyroidism and 46.15% patients with Overt Hypothyroidism also had diabetes mellitus. Hypothyroidism (Hashimoto's thyroiditis) has been investigated to be associated with diabetes mellitus. A meta- analysis reported a frequency of 11% in thyroid dysfunction in the patients of diabetes mellitus.[25] Autoimmunity has been implicated to be the major cause of thyroid-dysfunction associated diabetes mellitus.[26]

In present study, patients with Subclinical Hypothyroidism patients had co-morbidities like, ADPKD (9%), Chronic glomerulo nephritis (3.03%), obstructive uropathy (1.15%), connective tissue disorders (6.06%) & unspecified conditions (3.03%).

There is an increasing prevalence of diseases that predispose individuals to CKD, such as hypertension, diabetes, obesity and other, rendering the prevention and early detection of CKD a health-care priority in both developed and developing countries.[27]

In the present study, the mean S. Creatinine was significantly more among patients with Subclinical hypothyroidism. The mean Creatinine clearance (ml/min) was significantly lesser among patients with Subclinical hypothyroidism. We also had observation that the mean S. Creatinine increased significantly from stage I to stage V. The mean Creatinine clearance (ml/min) decreased significantly from stage I to stage V.

Studies by Kreisman SH and Hennessey JV et al,[28] Khan AH and Majumder I,[29] and Vaneet Kaur et al,[30] also point toward mean S. Creatinine level being significantly higher in hypothyroid cases. The serum Creatinine concentration increases in hypothyroid patients due to reduction of glomerular filtration rate because of hemodynamic changes in severe hypothyroidism. Serum Creatinine level may also be increased due to hypothyroid myopathy.[28]

In the present study, no significant difference was seen in lipid parameters apart from triglycerides (p-value = 0.007). In general, the prevalence of hyperlipidemia increases as renal function declines, with the degree of hypertriglyceridemia and elevation of LDL cholesterol being proportional to the severity of renal impairment.[31] CKD affects lipoprotein metabolism, leading to hypercholesterolemia, hypertriglyceridemia and excess LDL cholesterol.[32]

Many studies have reported rise in level of lipid parameters and dyslipidemia prevalence in CKD patients, which may further assist in renal disease progression.[33] Raju et al,[34] found that there was a significant increase of serum triglycerides and very low density lipoprotein (VLDL) with a decrease in serum HDL cholesterol in both non dialysis and hemodialysis groups of CKD patients when compared with control. But there was no alteration in serum total cholesterol and LDL cholesterol in both groups.

In the present study, significant alteration in thyroid parameters was seen in chronic kidney disease in all the stages where p value<0.001.

In the present study, 26.66% of the patients showed altered echotexture with normal kidney on USG, 11.33% showed features of obstruction, 16.66% had scarred kidney & 45.33% had small kidneys. Gowda AS,[35] reported that ultrasound abdomen showed evidence of CKD in all patients, contracted kidney was present in 90% of the patients, remaining patients had poor corticomedullary differentiation. Al Miraj AK et al,[36] reported that Ultrasound abdomen showed features of contracted kidney in 40 patients accounting for 80% and the remaining 10 patients had loss of corticomedullary differentiation, accounting for 20%.

CONCLUSION

Chronic kidney disease and hypothyroidism may exhibit overlapping symptom complexes. Hence it is suggested to assess the renal status of the patient at the time of diagnosis of Hypothyroidism and to have a strict observation of these parameters in follow up phase especially in person having risk factors for developing kidney disease eg-hypertension, diabetes mellitus.

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