To Study Thyroid Function and Lipid Profile Levels in Chronic Kidney Disease Patients in a Tertiary Care Hospital of North India

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ABSTRACT

BACKGROUND

Chronic kidney disease (CKD) includes a spectrum of different pathophysiology processes associated with abnormal kidney function, and a progressive decline in GFR. Progression of CKD is associated with having a number of complications, including thyroid dysfunction, dyslipidaemia, and cardiovascular diseases.

METHODS

The present study was conducted among 60 CKD patients (cases) and 60 healthy controls to compare their thyroid and lipid profile, who attended the Department of Medicine in SGRDIMSR, Sri Amritsar from January 2019 to December 2020.These 60 CKD patients were grouped as group A. Group A was further divided into various stages as per KIDGO staging according to GFR. 60 healthy individuals were taken as controls and were kept as Group B. Demographic features (age and sex) and medical history of diabetes mellitus, hypertension were noted and blood samples (5mL) were analysed for blood urea, serum creatinine, free triiodothyronine (T3), free thyroxine (T4), thyroid stimulating hormone (TSH), total cholesterol, high-density lipoprotein (HDL), low-density lipoprotein (LDL), very low density lipoprotein (VLDL) and triglycerides.

RESULTS

Thyroid dysfunction was observed in patients of CKD, the most common being overt hypothyroidism (56.6 %) followed by subclinical hypothyroidism (16.6 %), low T3 (15 %), and hyperthyroidism (1.6 %). Hypercholesterolemia, low HDL, elevated LDL, VLDL and triglyceride levels were observed in 74.9 %, 85.0 %, 38.3 %, 41.6 % and 76.6 % patients, respectively. Patients with CKD with 5 had significantly higher risk of having thyroid dysfunction and dyslipidaemia as compared to patients with stage 3 and 4.

CONCLUSIONS

Thyroid dysfunction and dyslipidaemia were common in patients with CKD. Prevalence of hypothyroidism, dyslipidaemia increases with progression of CKD. Hence early detection of thyroid dysfunction and dyslipidaemia is imperative to improve mortality and morbidity of CKD patients.

KEYWORDS

Chronic Kidney Disease, Dyslipidaemia, Thyroid Dysfunction

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BACKGROUND

CKD is a world-wide health problem with rising incidence and prevalence. CKD, especially in the early stages is often asymptomatic; thus, the actual prevalence may be even higher than estimated. Recent reports, suggest an abrupt rise in CKD in developing countries from Asia and the cause of this rise is increase in diseases like type 2 diabetes mellitus, hypertension and cardiovascular disease (CVD).[1] Chronic kidney disease is a silent epidemic of the 21st century. As per recent study, the incidence of End Stage Renal Disease (ESRD) in India is around 229 per million population, and more than one lakh new patients enter renal replacement programmes annually in India. Due to limited resources, only 10 % of these ESRD patients receive any renal replacement therapy (RRT).[2,3] According to the Kidney Disease Outcomes Quality Initiative (K / DOQI) Clinical practice guidelines, to diagnose CKD, there must be-

1. Kidney damage for > 3 months, either structural or functional abnormalities such as albuminuria or urinary sediment abnormalities.
2. Glomerular filtration rate (GFR <60 ml/min per 1.73 m^2) for > 3 months with or without evidence of structural damage.[4]

CKD staging was done as per KIDGO 2012 clinical practice guidelines:[4]

- CKD stage 1 is GFR (ml / min / 1.73m^2) ≥ 90 but with renal damage or injury
- CKD stage 2 (mild) is with GFR of 60-89 (ml / min / 1.73m^2)
- CKD stage 3 (moderate) is with GFR of 30-59(ml / min / 1.73m^2)
- CKD stage 4 (severe) is with GFR of 15-29 (ml / min / 1.73m^2)
- CKD stage 5 (end stage) is with GFR of less than 15(ml / min / 1.73m^2)

The interplay between thyroid and the kidney in each other's functions is well known. Thyroid dysfunction affects renal physiology and development, whereas kidney disease could result in thyroid dysfunction.[5] CKD affects the hypothalamus- pituitary- thyroid axis and peripheral metabolism of thyroid hormones. In uraemia, the pituitary receptor response to TRH is blunted causing a decrease in TSH release. The response of TSH to TRH is delayed due to decreased clearance and the increase in half life of TSH. In CKD there is decreased clearance of the inflammatory cytokines such as Tumour necrosis factor-alpha and Interleukin-1. These cytokines inhibit expression of 1 5′-deiodinase that helped convert T4 to T3. Low free T3 levels have shown to be an independent predictor of mortality in patients on haemodialysis. Low T3 levels prior to renal transplant are associated with risk of post-transplant graft loss. Low T3 levels in CKD may not be able to increase TSH levels. Experimental evidence suggests that sensitivity of thyrotropes is increased in uraemia. This accounts for the resetting of the central thyrostat indicating a lower level of the circulating thyroid hormones and in turn, affects the negative feedback inhibition.[6] CKD is strongly associated with changes in lipoprotein metabolism leading to dyslipidaemia and accumulation of atherogenic particles. CKD causes dyslipidaemia such as hypertriglyceridemia, elevated LDL cholesterol, an accumulation of apolipoprotein B (Apo B), increased lipoprotein (a) particles, and low HDL levels.[7]

As CKD progresses there is an alteration in lipoprotein metabolism.Delayed catabolism is the most prevalent mechanism responsible for an elevated triglyceride-rich lipoprotein concentration in CKD patients and occurs probably because of a decreased activity of hepatic triglyceride lipase and peripheral lipoprotein lipase. The presence of lipase inhibitors may also contribute to the delayed triglyceride-rich lipoprotein (VLDL, chylomicrons and their remnants) and LDL catabolism. Apolipoprotein C-III (apo C-III) is a direct lipoprotein lipase inhibitor, and its levels are increased in uraemia which further contributes to hypertriglyceridemia and elevated LDL and VLDL levels, as this interferes with uptake of triglyceride rich, apolipoprotein B containing lipoproteins by the liver and in peripheral tissue, yielding increased circulation of these atherogenic lipoproteins.[8]

CKD have reduced HDL levels in comparison with normal individuals. The protective function of HDL is diminished in CKD due to several mechanisms i.e. the decreased levels of apolipoproteins AI and AII and reduced activity of lecithin–cholesterol acyltransferase which is required for the esterification of free cholesterol in HDL. Furthermore, there is increased activity of cholesterol ester transfer protein (CETP), which cause transfer of cholesterol esters from HDL to triglyceride-rich lipoproteins. All these processes are responsible for the decreased serum level of HDL and promotes atherosclerosis. Also, heavy proteinuria leads to upregulation of HMG-CoA reductase, therefore causing hypercholesterolemia in ESRD patients.[9]

Cardiovascular disease is another common risk factor in CKD patients. A number of factors have been proposed as risk factors for CVD in CKD including dyslipidaemia, proteinuria, inflammation, anaemia, malnutrition, hypertension, oxidative stress, nitric oxide, and uremic toxins. Dyslipidaemia is the most common and major risk factor for cardiovascular morbidity and mortality and is common among patients with CKD. Lipid profiles vary widely in these patients, reflecting the level of kidney function and the degree of proteinuria. In general, the prevalence of hyperlipidaemia increases as renal function declines along with hypertriglyceridemia and elevation of LDL cholesterol which is proportional to the severity of renal impairment.[10]

The aim of the study was to determine thyroid dysfunctions and dyslipidaemia in CKD patients and to establish correlation between severity of renal disease with these two metabolic parameters. So that it may help in early identification of risk groups prone to develop adverse complications and to potentially improve their clinical outcomes.

Aim

To study thyroid function test and fasting lipid profile pattern in chronic kidney disease patients and also to study the

correlation of abnormalities of these thyroid function and lipid levels with renal functions in patients of chronic kidney disease in stages 3-5.

Objectives

1. To estimate freeT3, free T4 and TSH in healthy individuals and also in chronic kidney disease patients.
2. To estimate fasting triglyceride, total cholesterol, HDL, LDL, VLDL in healthy individuals and also in chronic kidney disease patients.
3. To study correlation of thyroid function abnormalities with renal function in patients of chronic kidney disease.
4. To study correlation of lipid abnormalities with renal function in patients of chronic kidney disease.

METHODS

Study Design

This was a case-control study, in which we included CKD patients (cases) and healthy controls to compare their thyroid and lipid profile, who attended the Department of Medicine in Sri Guru Ram Das Institute of Medical Sciences and Research, Sri Amritsar from January 2019 to December 2020. Cases were defined as patients aged 18 to 70 years diagnosed with CKD according to the Kidney Disease Outcomes Quality Initiative (K / DOQI) Clinical practice guidelines[4]

1. Kidney damage for > 3 months, either structural or functional abnormalities such as albuminuria or urinary sediment abnormalities.
2. Glomerular filtration rate (GFR <60 ml / min per 1.73 m^2) for >3 months with or without evidence of structural damage.

Controls were adults aged 18 to 70 years, who were not hypertensive nor diabetic with normal renal function, females who were not pregnant and not on steroids, immunosuppressant or lipid lowering medications. Their ultrasound abdomen and renal function test were normal.

Exclusion Criteria

The following patients were excluded from the study:

1. Patient refusing consent
2. Patients with past history of Primary hypothyroidism, hyperthyroidism or dyslipidaemia.
3. Patients on thyroid hormone supplementation or antithyroid drugs.
4. Septicaemia , Septic shock
5. Chronic alcoholism , smoking
6. Drugs altering lipid and thyroid profile like amiodarone, phenytoin, beta-blocker, dopamine, steroids, estrogen pills, linalidomide, rifampicin, salicyclates, fibric acid derivative and statins.
7. Liver disease
8. Myocardial infarction, Coronary Artery Disease
9. Cardiogenic shock
10. Malignancy
11. Pregnant females

The purpose of the study was explained to all study participants and a written informed consent was obtained from them. The study was approved by the Institutional Ethics Committee.

Sample Size

Sample size was calculated according to the following formula:

$$n = [(Z\alpha/2 + Z\beta)^2 \times (SD \times 2)] / d^2$$

n =Sample Size
Zα/2 = Z value at 5% error (1.96)
Zβ = Z value at 20% (0.84)
SD = average standard deviation of the character = (SD1+SD2)/ 2
d - Clinically relevant effect (taken as 0.5)

In the study by Vinayak et al, mean value of free T3 level in CKD patients and healthy control were 1.07± 0.35 and 1.73 ± 1.21, respectively.[11]
So, SD = 0.78

$$\mathrm{n} = \frac{(1.96 + 0.84)^2 \times (0.78 \times 2)}{(0.5)}$$

n = 48.92, so the minimum sample size was calculated to be 50 in each study group.

So in present study, study cases were consecutive 60 patients diagnosed with CKD fulfilling the study criteria and were kept as Group A. Group A was further divided into various stages as per KIDGO staging according to GFR.[4]

Controls were consecutive 60 healthy age and sex matched controls fulfilling the study criteria and were kept as Group B.

Laboratory Measurements

Under all aseptic precautions blood samples of about 5ml were collected and were analyzed for blood urea, serum creatinine, total cholesterol, high density lipoprotein (HDL), low density lipoprotein (LDL), serum triglyceride, free triiodothyronine (T3), free thyroxine (T4) and thyroid stimulating hormone (TSH). Serum urea level was estimated using enzymatic methods and serum creatinine by the Jaffe method. Thyroid function test was estimated using enhanced chemiluminescence. Fasting lipid profile was done in which total cholesterol was estimated by cholesterol oxidase, triglycerides was estimated by enzymatic end point and both LDL-C and HDL-C was calculated by direct measure.

Cockcroft-Gault equation was used to calculate GFR in chronic kidney disease patients.[4]

$$Estimated\ creatinine\ clearance(ml/min) = \frac{(140 - age) \times \text{body weight (kg)}}{72 \times Serum\ Creatinine(mg/dl)}$$

(Multiply by 0.85 if women)

Data Collection

A detailed clinical history and clinical examination was done on participating subjects. Information of the study participants was noted using a pre-designed semi-structured study proforma. Demographic features (age and sex), family history, drug history, history of alcohol intake, smoking, height, weight, BMI (as per Asian guidelines) and medical history of CKD, diabetes mellitus and hypertension of each patient were noted. Cockcroft-Gault equation was used to calculate GFR in chronic kidney disease patients. Findings of the thyroid (TSH, free T3 and free T4) and lipid profile (total cholesterol, triglyceride, HDL, LDL, VLDL) were noted as well.

Statistical Analysis

The data were compiled in Microsoft excel and analyzed in SPSS version 23 (IBM, NY). The quantitative data were described as means and standard deviation, while qualitative data were described frequency distribution and percentages. The cases (Group A) and controls (Group B) were compared for various variables. The demographic variables were compared using chi-square test. Various parameters of thyroid and lipid profile were compared between cases and controls using student's t test. For sub-group analysis, the cases (n = 60) were grouped into CKD staging as per KIDGO staging. Various parameters of thyroid and lipid profile were compared between the patients from CKD stage 3, 4 and 5 using one-way Analysis of Variance (ANOVA) test. A p value of less than 0.05 was considered statistically significant.

RESULTS

The mean age among CKD patients was 54.25 ± 10.12 and among healthy individuals was 50.33±12.06. The maximum number of patients among case group and control group were between the age group of 40 - 59 years (53.33 %) (Table -1). Among group A, 36 patients (60 %) were males and 24 (40 %) were females. Among Group B, 31 (51.6 %) were males and 29 (48.3 %) were females. (Table-2). The patients were grouped according to GFR in different CKD stages, the maximum number of patients were in stage 5 constituting 38 patients (63.3 %) followed by 15 patients (25 %) in stage 4 and 7 patients (11.6 %) in stage 3. Among 60 CKD patients, 38 patients (63.33 %) were on maintenance haemodialysis and 22 patients (36.66 %) were on conservative management.

In our present study, hypertension (85 %) was found to be the most common cause of CKD followed by diabetes mellitus (61.6 %) among the CKD patients. In present study, the mean blood urea value was 28.18 ± 3.38 in stage 3, 50.94 ± 4.53 in stage 4 and 88.32 ± 6.88 in stage 5 of chronic kidney disease. The p-value was 0.002 which was statistically significant. In present study, the mean value of serum creatinine according to chronic kidney disease stage 3 was 2.49 ± 0.29, in stage 4 was 4.93 ± 0.41 and in stage 5 was 10.55 ± 0.95. The p-value was 0.002 which was statistically significant.

Age Group (years)	Group A		Group B	
	No.	% Age	No.	% Age
18-39	4	6.67	9	15.00
40-59	35	58.33	32	53.33
60-70	21	35.00	19	31.60
Total	**60**	**100.00**	**60**	**100.00**
Mean	54.25±10.12		50.33±12.06	

Table 1. Age Distribution among Cases and Controls

Sex	Group A		Group B	
	No.	% Age	No.	% Age
Female	24	40.00	29	48.33
Male	36	60.00	31	51.67
Total	**60**	**100.00**	**60**	**100.00**

Table 2. Sex Distribution among Group A and Group B

Variables	Group A (Mean ± SD)	Group B (Mean ± SD)	P-Value
Free T3	2.18±0.74	2.89±0.67	0.002
Free T4	0.84±0.48	1.22±0.34	0.002
TSH	8.02±7.80	2.22±1.27	0.001
Total Cholesterol	224.20±69.90	141.47±35.54	0.001
Triglycerides	180.18±60.24	103.34±32.66	0.001
HDL	32.67±17.74	45.70±6.29	0.001
LDL	94.07±51.68	62.74±25.56	0.001
VLDL	33.99±17.29	19.95±8.18	0.001

Table 3. Thyroid Profile and Lipid Profile Parameters among Group A and Group B

Parameters	CKD Stage 3	CKD Stage 4	CKD Stage 5	P-Value
TSH	3.27±2.30	7.74±6.79	10.26±8.54	0.013
Free T3	2.67±0.67	2.43±1.02	1.99±0.55	0.025
Free T4	1.01±0.36	0.96±0.62	0.72±0.62	0.006

Table 4. Distribution of Thyroid Profile among Various Stages of CKD

Parameters	CKD Stage 3	CKD Stage 4	CKD Stage 5	P-Value
Total Cholesterol	123.60±19.33	210.84±73.74	234.11±70.97	0.036
Triglycerides	138.12±29.41	164.93±64.08	192.82±60.21	0.020
HDL	44.74±22.04	35.71±25.33	28.14±10.22	0.039
LDL	89.99±35.99	71.62±45.43	103.69±54.47	0.120
VLDL	29.10±7.86	30.27±15.97	36.37±18.83	0.030

Table 5. Lipid Profile among Various Stages of CKD

The levels of TSH were markedly increased in group A (CKD patients) when compared to group B (healthy individuals) and it was statistically significant with the p-value of 0.001. Free T3 levels were low in group A when compared to group B which was statistically significant with the p-value of 0.002. The free T4 levels were decreased in group A when compared to group B which was statistically significant (p-value of 0.002) as shown in Table 3.

The total cholesterol, LDL-C, VLDL and triglyceride levels were significantly increased in group A when compared to group B (p-value < 0.05). The serum HDL levels were decreased in group A in comparison to group B which was statistically significant (p- value of 0.001). Table-3 illustrates comparison of lipid profile parameters among group A and group B.

The TSH levels have shown increasing trend with progression of CKD stages which was statistically significant (p-value of 0.013) as shown in table-4. The level of Free T3 and Free T4 were found to be decreased as the stage of CKD progressed and was statistically significant (p-value < 0.05) as presented in table-4.

The level of total cholesterol, triglycerides and VLDL were found to be increased as the stage of CKD progressed and was statistically significant with p-value of <0.05. The mean value HDL levels were found to be decreased as the stage of CKD progressed in our study which was statistically

significant (p-value- 0.039). The LDL level was found to be increased as CKD progressed but was not statistically significant (p-value was 0.120) as presented in table-5.

DISCUSSION

In our present study, the mean age was 54.25 ± 10.12 among CKD patients and 50.33 ± 12.06 among controls with maximum patients in age group of 40 – 59 years. There were total 67 (55.8 %) males and 53 (44.1 %) were females. Hypertension was the most common cause of CKD in our study followed by diabetes mellitus. In our present study, thyroid dysfunction and dyslipidaemia was observed in CKD patients. In present study, serum free T3 levels were significantly decreased in chronic kidney disease patients in comparison to controls. The mean value of free T3 among CKD patients was 2.18 ± 0.74 and among controls was 2.89 ± 0.67. The p-value was 0.002 which was statistically significant. The similar finding was observed in study conducted by Vinayak R et al in which the mean value of free T3 level in CKD patients and control were 1.07 ± 0.35 and 1.73 ± 1.21, respectively. The serum free T3 levels were found to be significantly lower in chronic kidney disease patients when compared to controls with p-value of 0.0003.[11] In study done by Srivastava S et al reported that the mean free T3 level was low in CKD patients (1.4727 ± 0.3577) than controls (2.6613 ± 0.6155) which was statistically significant (p-value < 0.001).[12]

In present study, the mean value of serum free T3 was 2.67 ± 0.67 in stage 3, 2.43 ± 1.02 in stage 4 and 1.99 ± 0.55 in stage 5. The p-value was 0.025 which was statistically significant. Free T3 levels were found to be decreased with severity of chronic kidney disease. In study conducted by Khatiwada S et al observed the mean value of free T3 was 3.47 ± 0.855 in stage 3, 2.515 ± 0.962 in stage 4 and 2.807±1.287 in stage 5 (p-value < 0.005).[13] In study done by Kumudha P et al found that the mean values of free T3 was 2.9 ± 0.89 in stage 3, 1.8 ± 0.74 in stage 4 and 1.1 ± 0.63 in stage 5. Free T3 levels decreased significantly with progression of stages (p-value < 0.001).[14] In study by Pan B et al reported the significant rise in mean free T3 levels with severity of disease (p-value < 0.005).[15] This may be due to chronic kidney disease which is associated with increased level of inflammatory cytokines, metabolic acidosis and increased excretion of free T4 which leads to impaired peripheral conversion of T4 to T3 which contributes to low T3 concentration.[6]

In present study, serum free T4 levels were significantly decreased in CKD patients when compared to controls. The mean free T4 value among CKD patients was 0.84 ± 0.48 and among controls was 1.22 ± 0.34. The p-value was 0.001 which was statistically significant. In study done by Vinayak R et al observed that the mean free T4 level in CKD patients and control were 6.53 ± 2.93 and 8.66 ± 2.00, respectively. Serum free T4 levels were found to be significantly lower in chronic kidney disease patients when compared to controls with p-value of < 0.001.[11] In the study conducted by Punekar J et al had reported that the mean value of free T4 in CKD patients group was 6.07 ± 2.55 and in control group was 7.54 ± 1.38. The p- value was < 0.001 which was statistically significant.[16]

In present study, the mean value of free T4 decreased with progression of chronic kidney disease stages. The mean value of free T4 was 1.01 ± 0.36 in stage 3, 0.96 ± 0.62 in stage 4 and 0.72 ± 0.62 in stage 5. The p-value was 0.006 which was statistically significant. In study done by Kumudha P et al found that the mean values of free T4 was 0.8 ± 0.41 in stage 3, 0.7 ± 0.33 in stage 4 and 0.52 ± 0.09 in stage 5 of chronic kidney disease. Free T4 levels decreased significantly with progression of stages (p-value < 0.001).[14] This may be due to chronic kidney disease associated impaired binding of T4 to thyroid hormone binding globulin which further leads to low T4 levels.[6]

In present study, the mean serum TSH levels in chronic kidney disease patients was 8.02 ± 7.80 and mean TSH levels in control group B was 2.22 ± 1.27. The p-value was 0.001 which was statistically significant. TSH levels were found to be increased among CKD patients in comparison to control group B. In study done by Vinayak R et al observed that the mean TSH level in CKD patients was 5.11 ± 2.73 and in controls was 2.17 ± 1.57. Serum TSH levels were significantly higher in chronic kidney disease patients when compared to controls. The p-value was < 0.001 showing significant difference between both CKD patients and control.[11] In the study conducted by Punekar J et al had reported that the mean value of serum TSH in case group was 7.42 + 4.25 and in control group was 2.13 + 0.87(p-value < 0.001).[16]

In present study, the mean value of serum TSH levels was 3.27 ± 2.30 in stage 3, 7.74±6.79 in stage 4 and 10.26 ± 8.54 in stage 5 of chronic kidney disease patients. The p-value was 0.013 which was statistically significant. The serum TSH levels showed increasing trend with progression of chronic kidney disease stages. In study done by Sinha V et al found that the mean values of TSH in stage 3 was 2.5 ± 1.6, in stage 4 was 3.3 ± 1.7 and in stage 5 was 5.4 ± 2.3. TSH levels were found to be increased significantly with progression of stages. (p-value < 0.001).[1] In study by Kumudha P et al found that the mean TSH was 5.3 ± 1.89 in stage 3, 6.6 ± 1.73 in stage 4 and 6.4 ± 1.96 in stage 5 of chronic kidney disease. The p-value was < 0.001 which was statistically significant.[14] In the study conducted by Punekar J et al had reported that the mean value of serum TSH was 2.59 ± 3.70 in stage 3, 6.29 ± 4.37 in stage 4 and 8.38 ± 3.84 in stage 5 (p-value < 0.001).[16] This may be due to chronic kidney disease associated blunting of pituitary receptor response to TRH leading to reduction in release of TSH from pituitary gland.[6]

In present study, the mean total cholesterol among CKD patients was 224.20 ± 69.90 and among control group B was 141.47 ± 35.54. The p-value was 0.001 which was statistically significant. The total cholesterol levels were higher in chronic kidney disease patients as compared to controls. In study done by Vinayak R et al found that the mean total cholesterol in CKD patients was 218.63 ± 76.03 and 167.49 ± 18.24 in control group (p-value < 0.0001).[11] In study conducted by Punekar J et al reported that the mean value of total cholesterol among CKD patients was 183.4 ± 48.1 and among control was 136.07 ± 20.29. Total

cholesterol was significantly higher (p value < 0.001) in chronic kidney disease patients as compared to controls.[16]

In our present study, the mean total cholesterol value in chronic kidney disease stage 3 was 123.60 ± 19.33, in stage 4 was 210.84 ± 73.74 and in stage 5 was 234.11 ± 70.97. The p-value was 0.036 which was statistically significant. Total cholesterol shows rise in levels with severity of chronic kidney disease. In study done by Sinha V et al observed that the mean total cholesterol was 176 ± 21.46 in stage 3, 250 ± 22.55 in stage 4 and 306 ± 12.85 in stage 5. The p-value was <0.05 which was statistically significant.[1] In study conducted by Tripathy SK et al found that the mean of total cholesterol level was 223.88 ± 31.94 in stage 3, 232.91 ± 58.90 in stage 4 and 272.79 ± 50.65 in stage 5. The difference in rise of total cholesterol with severity of chronic kidney disease stage was statistically significant (p-value of 0.035).[17] In chronic kidney disease there is renal insufficiency and heavy proteinuria which leads to increased activity of HMG-CoA reductase activity causing hypercholesterolemia.[9]

In our present study, the mean serum triglyceride among CKD patients was 182.18 ± 60.24 and among control group B was 103.34 ± 32.66, respectively. The p-value was 0.001 which was statistically significant. Serum triglyceride levels were higher in chronic kidney disease patients as compared to controls. In study done by Vinayak R et al found that the mean value of serum triglyceride in CKD patients was 229.94 ± 89.77 and 124.28±14.29 in control group (p-value<0.0001).[11] In study conducted by Punekar J et al reported that the mean serum triglyceride level among CKD patients was 142.51 ± 59.85 and among control was 116.96 ± 30.95. The difference was statistically significant (p value was < 0.001).[16]

In our present study, the mean serum triglyceride value was 138.12 ± 29.41 in stage 3, 164.93 ± 64.08 in stage 4 and 192.82 ± 60.21 in stage 5 of chronic kidney disease. The p-value was 0.020 which was statistically significant. Serum triglyceride were found to be markedly increased with severity of chronic kidney disease. In study done by Sinha V et al observed that the mean value of triglyceride was 144 ± 29.54 in stage 3, 242 ± 30.24 in stage 4 and 340 ± 45.54 in stage 5 (p-value < 0.005).[1] In study conducted by Tripathy SK et al reported that the mean of triglyceride level was 203.09 ± 26.08 in stage 3, 206.25 ± 23.92 in stage 4 and 210.43 ± 25.99 in stage 5. The difference in rise of total cholesterol with severity of chronic kidney disease stages was statistically significant (p-value of 0.001).[17] Hypertryglyceridemia was one of a common lipid abnormality in chronic kidney disease patients. It is may be due to defective metabolism of lipoprotein lipase and hepatic lipase which causes delay in triglyceride catabolism and increase hepatic production of triglyceride.[8]

In our present study, the mean value of HDL among CKD patients was 32.67 ± 17.74 and mean HDL among controls was 45.70 ± 6.29. The p-value was 0.001 which was statistically significant. The HDL levels showed decreasing trend in patients of chronic kidney disease. In study done by Singh S et al observed that the mean HDL was 38.35 ± 4.01 among chronic kidney disease patients and 54.21 ± 3.94 among control group. The p-value was < 0.005 which was statistically significant.[18] In study conducted by Sumathi ME et al concluded that the mean HDL value in CKD patients was 36.1 ± 5.3 and in control group was 44.3 ± 5.0. The decline in HDL levels was significant in CKD patients when compared with healthy controls (p-value < 0.001).[19]

In present study, the mean values of HDL among chronic kidney disease stage 3 was 44.74 ± 22.04, 35.71 ± 25.33 in stage 4 and 28.14 ± 10.22 in stage 5. The p- value was 0.039 which was statistically significant. The level of HDL was observed to be in decreasing trend as the stage progressed from stage 3 to 5. In study conducted by Jain D et al found that the mean HDL levels in stage 3 was 39 ± 10.9, in stage 4 was 37.16 ± 12.44 and in stage 5 was 30.88 ± 12.14, which was statistically significant with p- value of < 0.05.[20] In study done by Noor S et al concluded that mean level of HDL in different stages of chronic kidney disease patients was 37.23 ± 1.23 in stage 3, 29.93 ± 12.10 in stage 4 and 25.13 ± 12.16 in stage 5. The reduction in HDL levels with progression of stages was statistically significant (p-value < 0.05).[21] This decrease was may be due to chronic kidney disease causing reduced activity of lipoprotein lipase, hepatic triglyceride lipase, LCAT and decreased levels of apolipoproteins (apoAI and apo AII). All these factors leads to decrease in serum HDL levels.[8] The similar result was observed in Chen et al and Amit et al.[22,23]

In our present study, the mean LDL among CKD patients was 94.07 ± 51.68 and mean LDL level among controls was 62.74 ± 25.56. The p-value was 0.001 which was statistically significant. In study done by Singh S et al observed that the mean LDL value was 153.07 ± 23.84 among chronic kidney disease patients and 94.96 ± 18.83 among control group. The p-value was < 0.005 which was statistically significant.[18] In study conducted by Keerthana BL et al concluded that the mean LDL level in CKD patients was 146.50 ± 23.45 and in control group was 102.62 ± 18.28. The rise in LDL levels was statistically significant in CKD patients when compared with healthy controls (p-value < 0.001).[24]

In present study, the mean serum LDL levels was 89.99 ± 35.99 in stage 3, 71.62 ± 45.43 in stage 4 and 103.69 ± 54.47 in stage 5 of chronic kidney disease patients. The LDL increased as the stages progressed but this difference was not statistically significant with p-value of 0.120. This was in accordance with the study done by Sinha V et al who found that the mean LDL value was 71.59 ± 24.88 in stage 3, 110±14.33 in stage 4 and 143 ± 31.32 in stage 5. The p-value was 0.216 showing that the difference in increase of LDL level with progression of disease was not statistically significant.[1] In study done by Jain D et al showed significant increase in serum LDL levels in patients of chronic kidney disease (p-value < 0.05), this result was not comparable to the present study.[20] In study conducted by Khatiwada S et al had reported that the mean LDL was 106.1 ± 30.9 in stage 3, 100.6 ± 25.6 in stage 4 and 105.0 ± 26.7 in stage 5. The p-value was 0.214 which was not statistically significant. In chronic kidney disease there is decreased catabolism of low density lipoprotein leading to reduction in plasma clearance. This reduced catabolism is masked by decreased LDL production, causing near normal serum LDL levels.[13]

In our present study, the mean VLDL among CKD patients was 33.99 ± 17.29 and among controls was 19.95

± 8.18. The p-value was 0.001 which was statistically significant. The VLDL levels showed increasing trend in patients of chronic kidney disease. In study done by Singh S et al observed that the mean VLDL value was 29.14 ± 16.33 among chronic kidney disease patients and among control group was 13.96 ± 3.78. The p-value was < 0.005 which was statistically significant.[18] In study conducted by Sumathi ME et al concluded that the mean VLDL level in CKD patients was 43.8 ± 7.7 and in control group was 23.0 ± 6.1. The rise in VLDL level was significant in CKD patients when compared with healthy controls (p-value < 0.001).[19]

In present study, the mean values of VLDL among chronic kidney disease patients was 29.10 ± 7.86 in stage 3, 30.27 ± 15.97 in stage 4 and 36.37 ± 18.83 in stage 5. The p-value was 0.030 which was statistically significant. The levels of VLDL were observed to be in increasing trend from stage 3 to 5. In study conducted by Jain D et al found that the mean VLDL levels in stage 3 was 26.88 ± 7.83, in stage 4 was 31.34 ± 10.18 and in stage 5 was 38.82 ± 14.74, which was statistically significant with the p- value of < 0.05.[20] In study by Kokkat J et al reported that in patients of chronic kidney disease, the mean serum VLDL increased with progression of stage. The p-value was < 0.001 showing this difference was statistically significant. They concluded this may be due to chronic kidney disease associated impaired activity of lipoprotein lipase, hepatic lipase VLDL receptors and HDL metabolism leading to increased concentration of serum VLDL levels.[25]

CONCLUSIONS

Results of the present study provide valuable information and association between thyroid, lipid abnormalities and chronic kidney disease patients. Thyroid dysfunction and dyslipidaemia both contribute to renal disease progression and risk of cardiovascular disease. Thus, the early assessment of thyroid function and lipid profile in chronic kidney patients may help in early identification of risk groups prone to develop adverse complications and potentially improve their clinical outcomes.

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