

## STUDY OF THYROID AND LIPID PROFILE IN CHRONIC KIDNEY DISEASE

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## Abstract

**Background:** The development of chronic kidney disease (CKD) is associated with various comorbidities such as thyroid dysfunction, dyslipidaemia, and diabetes. This study aimed to assess the thyroid and serum lipid profiles of patients with CKD and establish a correlation between the severity of renal disease and these two metabolic parameters. **Materials and Methods:** This prospective study was conducted over one year among 100 patients with CKD admitted to the Department of General Medicine, Government Medical College & Hospital, Pudukottai. We categorised moderate CKD (stage 3) as having an eGFR between 30-60 ml/min, whereas severe CKD (stages 4 and 5) had an eGFR below 30 ml/min. Blood samples were collected and centrifuged, and biochemical assays were conducted within 24 h. We also examined the lipid profiles, including total cholesterol, triglycerides, and HDL cholesterol. **Result:** Among the 100 patients included in the study, 69% were male and 31% were female, with an average age of  $51.24 \pm 11.28$  years. Of these patients, 34% had diabetes mellitus, and 68% had hypertension. The distribution of CKD stages among the patients was as follows: 21% were grade 1, while 25%, 32%, 11%, and 11% were grades 2, 3, 4, and 5, respectively. Significant differences in thyroid (T3, T4, and TSH) and lipid profiles (total cholesterol, triglycerides, high-density lipoprotein, and low-density lipoprotein) were observed across CKD grades 2-5, with ( $p < 0.0001$ ). **Conclusion:** Significant variations in both thyroid and lipid profiles were observed across different CKD stages, suggesting the need for tailored management strategies in these patients.

## INTRODUCTION

Chronic kidney disease (CKD) involves various pathophysiological processes that lead to abnormal kidney function and a progressively decreasing glomerular filtration rate (GFR). These pathological changes in CKD result in the loss of renal metabolic, excretory, endocrine, and synthetic functions due to the accumulation of protein nitrogenous substances.<sup>[1,2]</sup> In the early stages of CKD, primary care physicians play a crucial role in managing the disease and slowing its progression to end-stage renal disease (ESRD) by addressing associated comorbidities. Lipid and thyroid dysfunction are two significant comorbidities in patients with CKD.<sup>[3,4]</sup> Hyperlipidaemia, characterised by abnormally high levels of lipids in the blood, is a well-known risk factor for atherosclerosis and cardiovascular diseases and is frequently observed in patients with CKD. Indian studies on the pathophysiological relationship between CKD and lipid profile have reported varying

findings, from no significant lipid profile abnormalities to notable alterations such as high triglycerides and low HDL levels.<sup>[2,5]</sup>

The spectrum of dyslipidaemia in CKD and dialysis patients differs significantly from that of the general population, affecting all lipoprotein classes and varying considerably depending on the CKD stage. Additionally, accumulating clinical evidence suggests that lipids play a crucial role in the development and progression of chronic renal diseases. Potentially harmful lipid abnormalities are consistently present in these patients, increasing their likelihood of progressing to ESRD.<sup>[5]</sup> There is also evidence of thyroid hormone dysfunction in patients with CKD. CKD affects the synthesis, secretion, metabolism, and elimination of thyroid hormones. Under normal conditions, iodine, which is essential for thyroid hormone synthesis, is removed from circulation by glomerular filtration.

In CKD, decreasing GFR leads to iodine accumulation in the blood, reducing thyroid hormone

synthesis via the 'Wolff-Chaikoff effect'.<sup>[2,6]</sup> This results in subnormal serum total and free T3 concentrations, normal reverse T3, and free T4 levels, with TSH levels typically remaining unchanged. Patients with CKD may exhibit symptoms of hypothyroidism.<sup>[2]</sup> Thus, analysing lipoprotein subclasses and thyroid hormone dysfunction in patients with CKD is essential for assessing clinical outcomes. This study aimed to determine the thyroid and serum lipid profiles of patients with CKD and establish a correlation between the severity of renal disease and these two metabolic parameters.

## MATERIALS AND METHODS

This prospective study was conducted among 100 patients with CKD admitted to the Department of General Medicine for one year at Government Medical College & Hospital, Pudukottai.

### Inclusion criteria:

Patients aged 40-60 years, of both sexes, and diagnosed with moderate-to-severe CKD were included.

### Exclusion criteria:

Patients with a history of hyperthyroidism or hypothyroidism; CKD patients currently undergoing dialysis; those with obesity; nephrotic syndrome; patients taking oestrogens, corticosteroids, anti-thyroid drugs, or dietary supplements; and pregnant or lactating women were excluded.

The diagnosis of CKD was based on clinical profiles and renal function test results. The eGFR was calculated using the MDRD formula. An eGFR

between 30-60 ml/min was classified as moderate CKD (stage 3), while an eGFR below 30 ml/min was classified as severe CKD (stages 4 and 5).

**Specimen Collection:** A fasting venous blood sample (3 ml) was obtained from each subject. The samples were transferred into clean sterile centrifuge tubes and allowed to clot. Each clotted sample was then centrifuged at 3000 rpm for 3 min at room temperature to obtain the serum. The serum was carefully removed using a micropipette and transferred to Eppendorf tubes. Biochemical assays were conducted within 24 hours of collection.

**Assay parameters:** Total T3, T4, and TSH levels were analysed using the ... assay. The reference ranges were as follows: T3:0.7-2.0 ng/ml, T4:4.5-12.5 ng/ml, and TSH: 0.4-4.0  $\mu$ U/ml. Lipid profiles, including total cholesterol, triglycerides, and HDL cholesterol, were analysed.

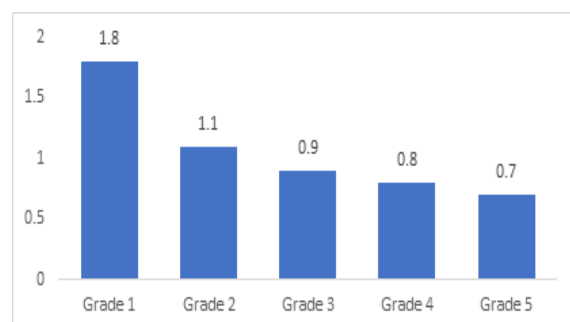
**Statistical analysis:** Data were statistically analysed using SPSS version 22. For continuous variables, data are presented as mean  $\pm$  SD, and means were compared using one-way analysis of variance (ANOVA). For categorical variables, data are presented as counts and percentages. The level of significance was set at  $p < 0.05$ .

## RESULTS

The demographic data indicated an average age of  $51.24 \pm 11.28$  years among the participants, with 31% female and 69% male participants. The distribution of CKD stages among the patients was as follows: 21% were grade 1, while 25%, 32%, 11%, and 11% were grades 2, 3, 4, and 5, respectively [Table 1].

**Table 1: Demographic data, comorbidities, and different grades of chronic kidney disease (CKD) among study participants.**

Variables		Frequency	Percentage
Age (Mean $\pm$ SD)		51.24	11.28
Gender	Male	69	69%
	Female	31	31%
Comorbidities	DM	34	34%
	HTN	68	68%
CKD stage	Grade 1	21	21%
	Grade 2	25	25%
	Grade 3	32	32%
	Grade 4	11	11%
	Grade 5	11	11%



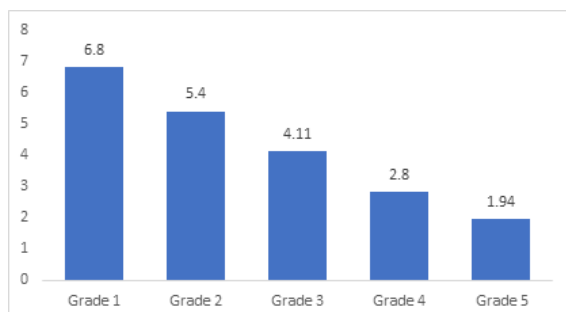
**Figure 1: Different grades of CKD stage-T3 among study participants**

The distribution of comorbidities showed that 34% of the participants had diabetes mellitus, whereas a significantly higher proportion (68%) had hypertension.

A comprehensive analysis of the thyroid and lipid profiles across different CKD stages and grades is presented in Figure 1–7. In CKD stage-T3, the results showed a significant decline in the mean values from Grade 1 to Grade 5. Grade 1 had a mean of  $1.8 \pm 0.11$  ( $p < 0.0001$ ). As the grade increased, the mean values progressively decreased, reaching  $0.7 \pm 0.21$  in Grade 5 [Figure 1].

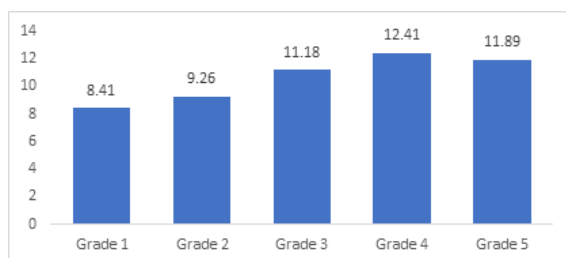
For CKD stage T4, a similar trend was observed. Grade 1 reported a mean of  $6.8 \pm 0.91$  ( $p < 0.0001$ ).

This value declined through the grades, culminating in a mean of  $1.94 \pm 0.62$  in Grade 5. Examining the thyroid-stimulating hormone (TSH) levels in CKD stage-TSH, the study found that Grade 1 had a mean of  $8.41 \pm 1.64$  ( $p < 0.0001$ ), while Grade 5 showed an increased mean of  $11.89 \pm 1.26$ , indicating an increase in TSH levels as the CKD grade advanced [Figure 2].



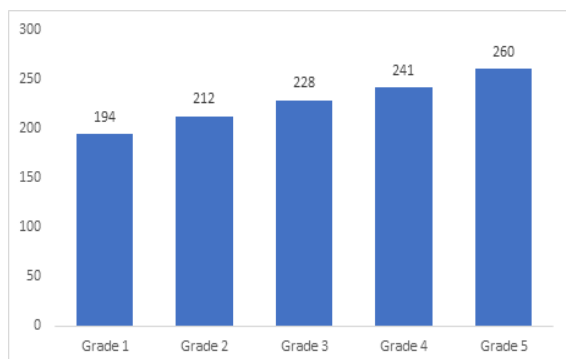
**Figure 2: Different grades of CKD stage-T4 among study participants**

For CKD stage TSH, Grade 1 patients reported a mean of  $8.41 \pm 1.64$  with a p-value of  $<0.0001$ . This value increased from grade to grade 4, culminating in a mean of  $12.41 \pm 3.06$  in Grade 4. However, Grade 5 showed a mean of  $11.89 \pm 1.26$  [Figure 3].



**Figure 3: Different grades of CKD stage TSH among study participants**

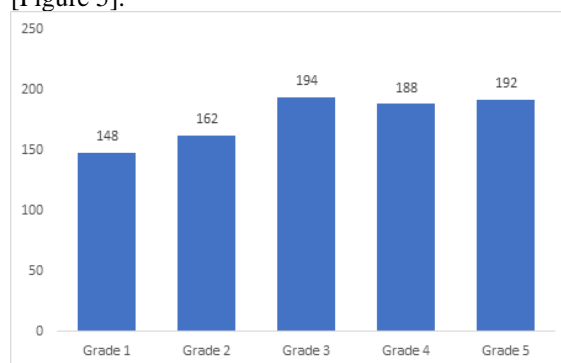
The total cholesterol (TC) levels in CKD stage TC also followed a notable pattern. Grade 1 started with a mean of  $194 \pm 19.27$  and a significant difference ( $p < 0.0001$ ). By Grade 5, the mean increased to  $260 \pm 28.45$ , demonstrating a clear upward trend [Figure 4].



**Figure 4: Different grades of CKD stage-TC among study participants**

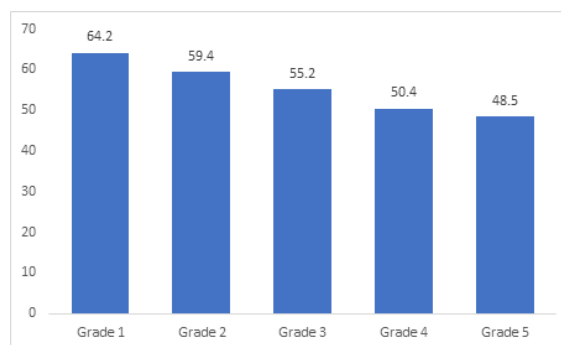
For triglycerides (TGL) in CKD stage TGL, the mean values began at  $148 \pm 11.24$  for Grade 1 with a P value

of  $<0.0001$  and increased to  $192 \pm 17.36$  by Grade 5, reflecting a consistent increase in triglyceride levels [Figure 5].



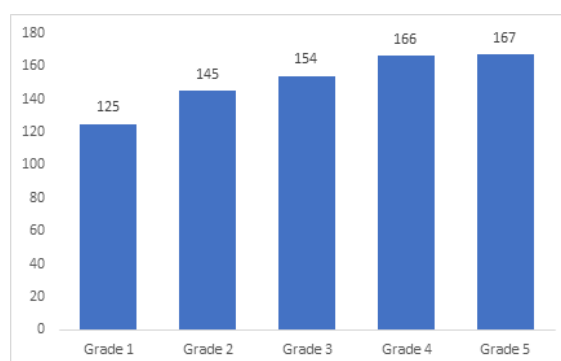
**Figure 5: Different grades of CKD stage-TGL among study participants**

High-density lipoprotein (HDL) levels in CKD stage HDL showed a decreasing trend from grades 1 to 5. The mean HDL level was  $64.2 \pm 2.18$  in Grade 1 with a P value of  $<0.0001$ , declining to  $48.5 \pm 4.2$  in Grade 5 [Figure 6].



**Figure 6: Different grades of CKD stage-HDL among study participants**

Lastly, low-density lipoprotein (LDL) levels in CKD stage LDL showed an upward trajectory. Grade 1 had a mean LDL of  $125 \pm 10.12$  with a p-value of  $<0.0001$ , which increased to  $167 \pm 16.4$  by Grade 5 [Figure 7].



**Figure 7: Different grades of CKD stage-LDL among study participants**

## DISCUSSION

In the present study, a significant proportion of participants had comorbid conditions, with 34%

diagnosed with diabetes mellitus and a considerably higher proportion (68%) with hypertension. These findings align with the well-documented high prevalence of these conditions among patients with chronic kidney disease (CKD), as both diabetes and hypertension are major risk factors for the development and progression of CKD. Similarly, Adem et al. observed that approximately 39% of respondents were female and 48.1% were hypertensive. In their study, the overall prevalence of CKD was 31.5%, whereas in our study most of the participants were from CKD grade 3 stage.<sup>[7]</sup> Galeti et al. observed that the majority of their participants (66%) were in CKD grade 4.<sup>[2]</sup>

CKD occurs when a disease process impairs the structure or function of the kidney, leading to chronic kidney failure. Cardiovascular disease is a major cause of death in patients with mild-to-moderate CKD and end-stage renal disease (ESRD). Dyslipidaemia and thyroid dysfunction are well-known risk factors for cardiovascular diseases and are common in patients with CKD. We observed a significant difference in the thyroid (T3, T4, and TSH) and lipid profiles (total cholesterol, triglycerides, high-density lipoprotein, and low-density lipoprotein) across CKD grades 2-5 ( $p < 0.0001$ ).

In our study, thyroid dysfunction was observed to increase with the CKD grade. Studies by Aminul et al., Bhutal et al., and Marwah et al. also pointed toward thyroid dysfunction among patients with CKD.<sup>[8-10]</sup> Various aetiologies have been proposed for the pathophysiology of renal dysfunction in hypothyroidism. In the hypothyroid state, hypovolaemia occurs due to decreased cardiac output, resulting in a reduction in renal blood flow. Thyroxine leads to increased systemic and renal vasoconstriction, further decreasing renal blood flow.<sup>[11]</sup> Thyroxine has been suggested to increase creatinine reabsorption in the blood by affecting urinary excretion. Additionally, thyroxine may influence transcription in the sarcoplasmic reticulum, affecting the  $\text{Na}^+/\text{Ca}^{2+}$  exchanger and  $\text{Na}^+/\text{K}^+$ -ATPase activity in the kidneys. These changes are associated with an increase in creatinine levels.<sup>[12]</sup>

Another possible mechanism of action of thyroid hormones on renal function could be explained by their influence on the maturation of the renin-angiotensin system (RAAS). Plasma renin activity and plasma levels of angiotensinogen, angiotensin II, and aldosterone are directly associated with plasma levels of thyroid hormones.<sup>[13]</sup> Patil et al. demonstrated that dyslipidaemia is prevalent in patients with chronic renal failure (CRF). Although total cholesterol levels were higher in CRF patients than in controls, the difference was not statistically significant. Triglycerides showed a statistically significant increase in CRF. HDL-C levels showed a statistically significant decrease compared to the controls. These lipid abnormalities, including the significant rise in triglycerides and the drop in HDL-

C, may contribute to the high cardiovascular mortality in CRF patients.<sup>[5]</sup>

Triglyceride levels were elevated in the CKD stage in our study. The present study demonstrated that CKD progression is commonly accompanied by lipid dysfunction, manifesting as hypertriglyceridaemia. This aligns with observations from recent studies by Gupta et al., Galeti et al., and Patil et al.<sup>[1,2,5]</sup> Sammaiah et al. observed that the most common lipid abnormalities were low HDL levels (50%) and hypertriglyceridemia (48%).<sup>[14]</sup> Elevated triglyceride levels are attributed to impaired lipoprotein lipase activity and the direct inhibitory action of various uremic toxins on enzymes involved in lipid metabolism. These mechanisms are the key contributors to the development of hypertriglyceridaemia in patients with renal failure. This highlights the critical need for routine screening for hypothyroidism and dyslipidaemia among patients with CKD. Importantly, thyroid hormone levels and their effects on CKD progression have not yet been thoroughly studied.

## CONCLUSION

This study emphasises the significant association between dyslipidaemia and thyroid dysfunction in the progression of CKD. The findings underscore the critical importance of routine screening for hypothyroidism and dyslipidaemia in patients with CKD. Future research should prioritise larger, more diverse populations with longitudinal data to better understand these relationships and their implications for managing CKD.

**Limitations:** This study included a relatively small number of patients with CKD, which may limit the applicability of the findings to larger populations. The study was conducted at a single centre in India, which may not represent the range of patients with CKD across different regions or healthcare settings. Additionally, the study lacked longitudinal follow-up data, which could have provided insights into the progression of dyslipidaemia and cardiovascular morbidity over time in patients with CKD.

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