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Original Research Article

Role of Lipid Profile, Thyroid Hormones and hsCRP in Patients with Chronic Renal Failure

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Abstract:

Background: Significant metabolic and endocrine abnormalities, especially in the metabolism of lipids and thyroid hormones, are linked to chronic renal failure (CRF), and these abnormalities collectively raise the risk of cardiovascular disease.

Objective: To access the connection between thyroid hormone levels, serum total cholesterol (TC), and systemic inflammation in patients with CRF.

Methods: 110 CRF patients between the ages of 18 and 70 participated in a cross-sectional study. Serum TC was estimated by the CHOD-PAP method, thyroid profile (T3, T4, TSH) was assessed using CLIA, and systemic inflammation was evaluated using hsCRP. Correlation analyses were performed between lipid profile, thyroid function, and hsCRP.

Results: The study population was primarily male (65.5%) and had a mean age of 52.3 years. Dyslipidemia was prevalent, with elevated triglycerides (188.2 ± 82.6 mg/dL) and reduced HDL cholesterol (38.5 ± 7.7 mg/dL), while mean TC remained near normal (175.5 ± 34.1 mg/dL). Thyroid analysis revealed reduced T3 levels (70.8 ± 13.4 ng/dL), suggesting "low T3 syndrome," while T4 and TSH remained within reference ranges. hsCRP was elevated (5.6 ± 4.1 mg/L), indicating systemic inflammation. Correlation analysis showed triglycerides and LDL were positively associated with hsCRP, whereas HDL correlated positively with T3 and inversely with hsCRP.

Conclusion: CRF patients exhibit an atherogenic lipid profile, low T3 syndrome, and persistent inflammation, highlighting a multifactorial risk for cardiovascular complications. Monitoring lipid and thyroid function along with inflammatory markers may provide a more comprehensive risk assessment and guide management strategies in CRF.

Keywords: Chronic Renal Failure (CRF), Lipid Profile, High-sensitivity C-reactive Protein (hsCRP), Serum Total Cholesterol.

Introduction

The persistent and permanent deterioration in kidney function that causes chronic renal failure (CRF), often referred to as chronic kidney disease (CKD), makes it impossible for the body to maintain proper metabolic and electrolyte balance. As the kidneys deteriorate, metabolic waste products accumulate, resulting in uremia, acidosis, anemia, and various endocrine and electrolyte disturbances. Polycystic kidney disease, glomerulonephritis, DM, and HTN are the main causes of CRF [1]. The functional status of the kidneys is typically assessed through the glomerular filtration rate (GFR), a vital indicator of renal efficiency. According to the kidney disease: Improving Global Outcomes (KDIGO) criteria, a persistent GFR below 60 mL/min/1.73 m² for three months or longer indicates chronic kidney disease [2]. This corresponds approximately to serum creatinine levels exceeding 137 µmol/L in men and 104 µmol/L in women [3]. The progression of the disease varies among individuals, depending on the cause and clinical management. Histologically, chronic renal failure is characterized by glomerulosclerosis, interstitial fibrosis, tubular atrophy, and infiltration by inflammatory cells such as monocytes and macrophages [2]. These structural alterations reduce the number of functioning nephrons, further worsening renal damage through compensatory hyperfiltration.

The causes of chronic renal failure differ across regions due to variations in genetics, environment, and healthcare accessibility. Some etiologies are preventable, such as infection-related or obstructive renal diseases, while others—like diabetic

nephropathy—require early detection and medical intervention to delay progression [4]. The thyroid gland plays a vital role in metabolic regulation, influencing cardiovascular function, growth, and electrolyte balance [5]. Likewise, the kidneys are essential for the metabolism and clearance of thyroid hormones and iodine [6]. This interrelationship means that disorders in one organ can significantly affect the other. In patients with CRF, thyroid hormone metabolism is frequently disturbed, leading to reduced triiodothyronine (T3) and thyroxine (T4) levels, altered hormone binding, and increased iodide retention due to impaired renal excretion [7]. As a result, thyroidal iodine stores rise, often causing enlargement of the gland. These biochemical and hormonal disturbances can further complicate the clinical course of renal failure and contribute to metabolic instability.

Subclinical hypothyroidism is observed in nearly 8% of patients with CKD, with its prevalence increasing as renal function declines [8]. Thyroid dysfunction can influence renal hemodynamics and filtration capacity. In hypothyroid states, reduced cardiac output and renal plasma flow lead to decreased GFR, causing elevated serum creatinine levels even without structural kidney injury. Additionally, reduced tubular secretion of creatinine may exaggerate the apparent decline in renal function. Conversely, hyperthyroidism can temporarily enhance renal blood flow and filtration, potentially concealing underlying renal impairment. Both hypo- and hyperthyroidism can alter electrolyte handling, tubular transport, and kidney structure [9]. These effects are particularly relevant in advanced CKD, dialysis-dependent patients, or those with renal transplants, where thyroid abnormalities can intensify metabolic complications and impact treatment outcomes. Assessing thyroid hormone levels in such patients is therefore crucial for comprehensive disease management and prognostic evaluation.

Along with thyroid abnormalities, dyslipidemia represents another major metabolic disturbance in chronic renal failure. Patients with advanced CKD or end-stage renal disease (ESRD) typically show elevated triglycerides, reduced high-density lipoprotein (HDL) cholesterol, and normal or mildly elevated low-density lipoprotein (LDL) cholesterol [10]. This atherogenic lipid profile contributes to the heightened cardiovascular risk observed in this population [11]. Small, dense LDL particles that are prone to oxidation build up as a result of impaired hepatic lipase and lipoprotein lipase activity as well as poor clearance of triglyceride-rich lipoproteins. Moreover, HDL in CKD patients often loses its protective antioxidant and reverse cholesterol transport functions, further increasing cardiovascular susceptibility. In ESRD, low LDL cholesterol levels have paradoxically been linked to higher mortality, likely reflecting malnutrition and inflammation rather than improved lipid control [10]. Despite this paradox, dyslipidemia remains a critical modifiable risk factor in CKD. Understanding the interaction between thyroid function, lipid metabolism, and renal health is therefore essential. In order to shed light on their combined roles in disease progression, CVD, and general metabolic imbalance, the current study is to assess the link between blood TC and thyroid hormone levels in patients with Chronic Renal Failure (CRF).

Materials and Methodology

The Department of Medicine and the Department of Biochemistry collaborated to conduct this study at [Insert Name of Institution/Hospital] (please confirm the exact name). A total of 100 patients diagnosed with CRF were enrolled in the research. Participants were between 18 and 70 years of age and were included only after providing written informed consent. Individuals who were receiving lipid-lowering medications, as well as pregnant or lactating women, were excluded from participation. For all subjects, a thorough physical examination was performed, and a detailed clinical history was obtained. Information regarding age, gender, comorbid conditions such as DM and HTN, duration of kidney disease, and details of ongoing treatments was recorded in a structured format.

After fasting for the whole night, each participant had around 5 mL of venous blood drawn. Serum was separated from the samples by centrifugation and then kept at -20°C until analysis. The enzymatic CHOD-PAP technique was used to estimate the level of serum total cholesterol.

The Chemiluminescent Immunoassay (CLIA) technology was used to evaluate thyroid-stimulating hormone (TSH), thyroxine (T4), and triiodothyronine (T3), among other thyroid function assays. An immunoturbidimetric assay on a fully automated analyzer was used to measure the levels of hsCRP, which enables accurate identification of low-grade systemic inflammatory activity.

Statistical Analysis: Microsoft Excel was used to compile all of the data that was gathered, and the SPSS was used to analyze it. The mean ± SD was used to represent continuous variables. Pearson's correlation coefficient was used to assess the relationship between renal function measures, thyroid hormones, and serum total cholesterol. For every comparison, a p-value of less than 0.05 was deemed statistically significant.

Result:

110 patients with a mean age of 52.3 ± 14.6 years made up the study population. Males predominated, accounting for 65.5% (n=72), while females made

up 34.5% (n=38). Regarding renal replacement status, 54.5% (n=60) were non-dialysis patients, 40% (n=44) were undergoing hemodialysis, and 5.5% (n=6) were on peritoneal dialysis. The mean estimated glomerular filtration rate (eGFR) was markedly reduced at 20.1 ± 9.7 mL/min/1.73m², reflecting advanced renal impairment. Serum creatinine levels were elevated, averaging 3.8 ± 1.2 mg/dL. Collectively, these characteristics indicate a middle-aged population with chronic kidney disease, predominantly male, and with significant representation of dialysis-dependent individuals.

The lipid profile of the study cohort (n=110) revealed dyslipidemia patterns commonly associated with chronic kidney disease. The mean total cholesterol level was 175.5 ± 34.1 mg/dL, remaining within near-normal limits. Triglycerides were notably elevated, averaging 188.2 ± 82.6 mg/dL, indicating hypertriglyceridemia in many patients. Low-density lipoprotein (LDL) cholesterol was measured at 104.8 ± 29.7 mg/dL, showing moderate levels. High-density lipoprotein (HDL) cholesterol averaged 38.5 ± 7.7 mg/dL, which is lower than recommended protective thresholds, suggesting reduced cardioprotective capacity. Overall, the lipid abnormalities point toward an atherogenic profile, characterized by high triglycerides and low HDL, consistent with cardiovascular risk in renal disease patients.

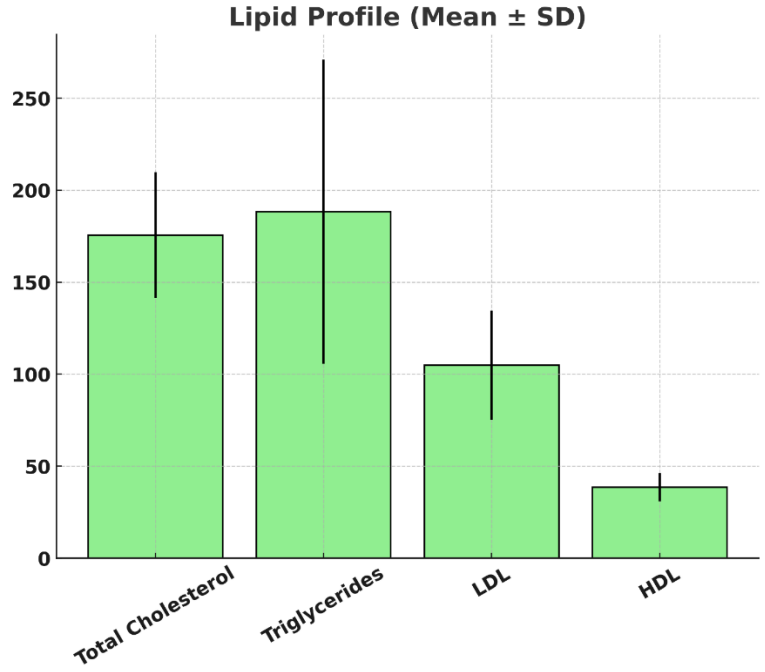

Figure 1: Lipid profile

Analysis of thyroid function and inflammation markers (n=110) demonstrated mean triiodothyronine (T3) levels of 70.8 ± 13.4 ng/dL (median 71), which were at the lower end of the reference range, reflecting possible low T3 syndrome in chronic illness. Thyroxine (T4) levels averaged 6.5 ± 1.1 µg/dL (median 6.6), suggesting near-normal thyroid hormone status. Thyroid-stimulating hormone (TSH) values were 3.1 ± 1.4 µIU/mL (median 3.0), indicating preserved pituitary-thyroid axis regulation. The systemic inflammatory marker hsCRP was increased, with a mean of 5.6 ± 4.1 mg/L (median 4.8), suggesting ongoing inflammatory burden in the study population. These findings highlight metabolic-inflammatory interplay.

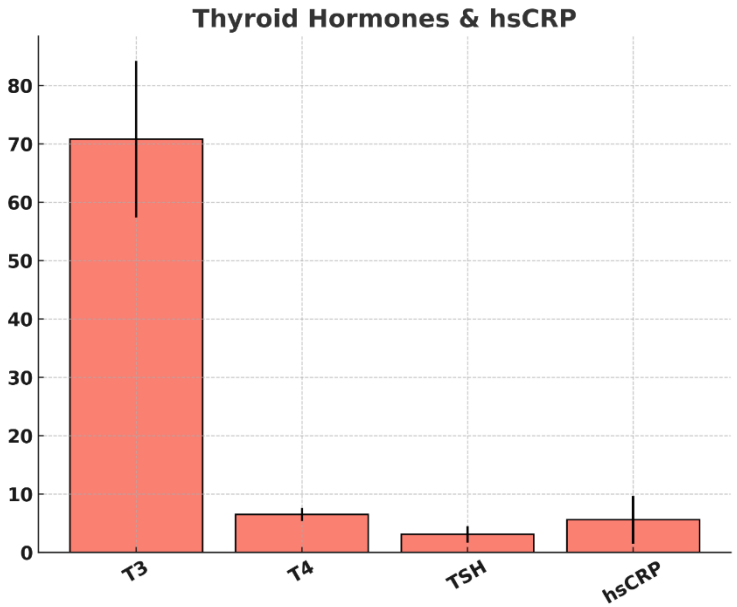

Figure 2: Thyroid Hormones & hsCRP

Spearman's correlation analysis revealed complex associations between lipid parameters, thyroid hormones, and hsCRP. Total cholesterol showed weak positive correlations with T3 (ρ=+0.12), T4 (+0.05), TSH (+0.18), and hsCRP (+0.21). Triglycerides exhibited an inverse relationship with T3 (–0.25) and T4 (–0.08), but positive correlations with TSH (+0.14) and hsCRP (+0.31). LDL cholesterol correlated positively with T3 (+0.10), T4 (+0.02), TSH (+0.22), and hsCRP (+0.19). Conversely, HDL cholesterol displayed favorable associations, correlating positively with T3 (+0.27) and T4 (+0.11), but negatively with TSH (–0.20) and hsCRP (–0.24). These findings suggest inflammatory and thyroid influences on lipid metabolism.

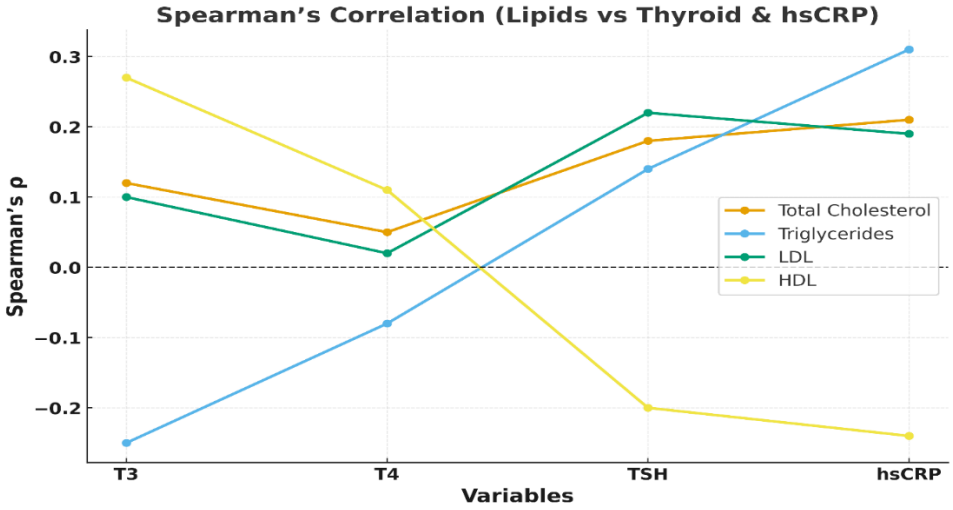

Figure 3: Spearman's Correlation (Lipids vs Thyroid & hsCRP)

The association between lipid parameters and hsCRP demonstrated that systemic inflammation significantly impacts lipid metabolism. Total cholesterol showed a weak positive correlation with hsCRP (ρ=+0.21). Triglycerides exhibited a stronger positive correlation (ρ=+0.31), indicating that higher inflammatory states may drive hypertriglyceridemia. LDL cholesterol had a modest positive correlation with hsCRP (ρ=+0.19), suggesting inflammatory contributions to atherogenic lipid patterns. Conversely, HDL cholesterol showed a negative correlation with hsCRP (ρ=–0.24), reflecting loss of its anti-inflammatory, protective role in the presence of elevated systemic inflammation. Overall, the results highlight inflammation-linked dyslipidemia as a critical risk factor in this cohort.

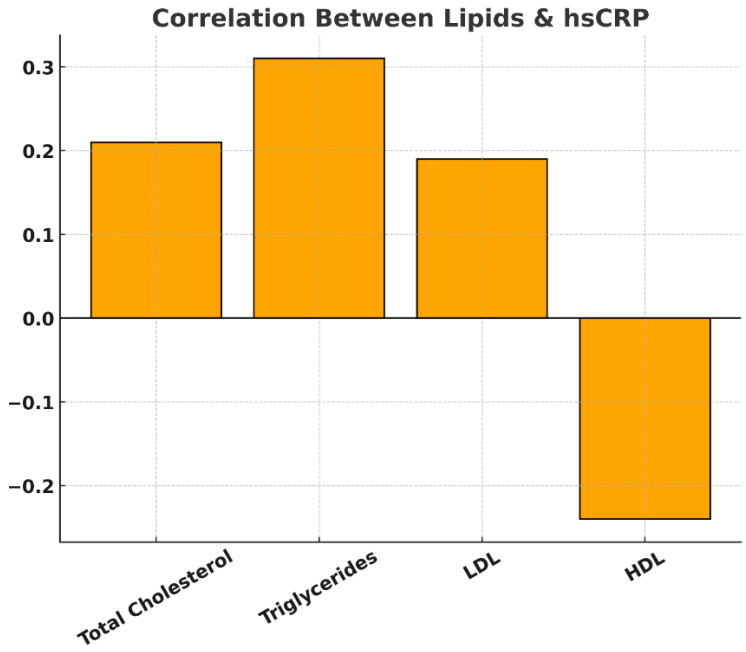

Figure 4: Correlation Between Lipid Profile and hsCRP

Discussion:

In the current investigation, we assessed thyroid hormone levels and serum lipid profiles in CRF patients and investigated their connections to systemic inflammation. The study cohort comprised predominantly middle-aged patients with a mean age of 52.3 years, with males representing a higher proportion. More than 40% of the patients were undergoing hemodialysis, indicating advanced renal dysfunction in the study population. The mean eGFR of 20.1 mL/min/1.73m² confirmed that most patients were in the late stages of CKD. In the study conducted by Cobo et al (2019) the epidemiological data consistently show that males represent a higher proportion of CRF cases than females (13). According to Zheng et al (2016) testosterone has been associated with accelerated renal injury and progression of CKD (14).

Our findings on lipid profile revealed that although mean TC levels remained within near-normal range (175.5 mg/dL), patients displayed an atherogenic dyslipidemia pattern characterized by elevated triglycerides (188.2 mg/dL) and low HDL cholesterol (38.5 mg/dL). The presence of this lipid pattern in our study highlights the enhanced cardiovascular risk faced by CRF patients even in the absence of markedly elevated total cholesterol levels. As per Attman et al (2009) CRF is characterized by dyslipidemia involving elevated triglycerides, accumulation of remnant lipoproteins, and reduced HDL, all of which accelerate atherogenesis (15). Moradi et al also found that Small dense LDL particles, common in CRF, are more susceptible to oxidation and endothelial damage (16). With respect to thyroid function, we observed reduced T3 levels (mean 70.8 ng/dL), while T4 and TSH values were largely within the normal range. This pattern is compatible with the so-called "low T3 syndrome," a common non-thyroidal illness seen in CKD. Our results support this association and reaffirm the importance of routine thyroid function monitoring in CRF patients. As per Fan et al (2016) Low T3 syndrome is prevalent in CKD patients, affecting 47% of those studied. Its incidence increases with CKD progression (17).

Elevated hsCRP levels in our cohort underscore the chronic inflammatory state inherent to CRF. Our correlation analyses revealed that inflammation influenced lipid and thyroid parameters: triglycerides and LDL were positively correlated with hsCRP, while HDL demonstrated an inverse relationship, reflecting its loss of anti-inflammatory function. Koley et al (2018) found a significant positive correlation between triglycerides and hsCRP in patients with dyslipidemia, but it does not specifically address patients with CRF (18). Song et al (2022) also found that there was significantly positively associated with elevated triglycerides (TG) and hs-CRP in hemodialysis patients, indicating a correlation between higher hs-CRP levels and increased TG, which is a component of metabolic syndrome (19).

The interplay of thyroid hormones, dyslipidemia, and inflammation in CKD patients highlights the complex metabolic disturbances in this population. We found that HDL correlated positively with T3 and T4, whereas triglycerides correlated negatively with T3. Pucci et al (2000) found that elevated thyroid hormones enhance cholesterol efflux and upregulate hepatic LDL receptors, indirectly

promoting higher HDL concentrations (20). Conversely, triglycerides (TG) display a negative association with T3, as hyperthyroid states are generally accompanied by reduced TG levels due to increased lipolysis and hepatic clearance (21).These findings emphasize that monitoring both lipid profile and thyroid hormones, alongside inflammatory markers, can provide a more comprehensive risk assessment in CRF patients.

Conclusion

The present study demonstrates that patients with CRF exhibit significant disturbances in lipid and thyroid metabolism, compounded by systemic inflammation. Despite near-normal serum total cholesterol levels, the presence of elevated triglycerides, reduced HDL cholesterol, and low T3 syndrome indicates an atherogenic and pro-inflammatory state, predisposing patients to heightened cardiovascular risk. The positive correlations of triglycerides and LDL with hsCRP and the inverse relationship of HDL with inflammation underline the importance of evaluating both metabolic and endocrine parameters in CRF. Routine monitoring of lipid profile, thyroid hormones, and hsCRP may therefore be beneficial for early detection of cardiovascular risk and improved clinical management of CRF patients.

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