RESEARCH PAPER

Modulation of the PON1–Oxidized LDL Interaction by Demographic Factors in Chronic Kidney Disease

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ABSTRACT

Background & Aims: Chronic kidney disease (CKD) is associated with heightened cardiovascular risk driven by oxidative stress, inflammation, and dyslipidaemia. Paraoxonase 1 (PON1), an HDL-bound antioxidant enzyme, protects against lipid peroxidation, while oxidized LDL (Ox-LDL) reflects oxidative vascular injury. Altered PON1 activity and elevated Ox-LDL are increasingly recognized as key contributors to CKD-related cardiometabolic complications. This study aimed to assess the relationship between serum PON1 activity and Ox-LDL levels in patients with CKD stages 1–3b and age-matched healthy controls, and to determine the impact of dyslipidaemia on oxidative stress in CKD.

Methods: A case–control study was conducted among patients with CKD stages 1–3b aged 30–60 years and age-matched healthy controls at Saveetha Medical College and Hospital. Serum renal function markers and lipid profiles were measured using the Vitros 5600 analyzer. Serum PON1 activity and Ox-LDL levels were quantified using sandwich ELISA. Statistical evaluation included one-way ANOVA and correlation analysis using SPSS v15.0, with a significance threshold of $p < 0.05$.

Results: CKD patients with dyslipidaemia exhibited significantly lower PON1 activity and markedly elevated Ox-LDL levels compared to non-dyslipidaemic CKD patients and healthy controls ($p < 0.001$).PON1 levels declined progressively across CKD stages, from 1.76 ng/ml in stage 1 to 0.46 ng/ml in stage 3b, while Ox-LDL levels increased from 1.72 mg/dl to 4.82 mg/dl over the same stages.A strong negative correlation between PON1 and Ox-LDL was found in advanced CKD ($r = –0.256$, $p < 0.00001$), indicating worsening oxidative imbalance. In contrast, a positive association between these markers was observed among healthy individuals.

Conclusions: Dyslipidaemia significantly exacerbates oxidative stress in CKD by lowering antioxidant PON1 activity and elevating lipid peroxidation, reflected by increased Ox-LDL levels. The progressive reduction in PON1 and escalation of Ox-LDL across CKD stages demonstrate their potential as sensitive biomarkers of oxidative imbalance. Assessing the PON1 and Ox-LDL may support early cardiovascular risk detection, optimize therapeutic strategies, and potentially slow CKD progression.

Keywords: Chronic Kidney Disease, Paraoxonase 1 (PON1), Oxidized Low-Density Lipoprotein (Ox-LDL), Oxidative Stress, Lipid Peroxidation, Cardiovascular disease

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INTRODUCTION

Chronic kidney disease (CKD) is a multifactorial and progressive condition characterized by gradual deterioration of renal function, affecting millions worldwide and posing a significant burden due to heightened cardiovascular morbidity and mortality risks. The pathophysiology of CKD involves complex interactions between systemic inflammation, oxidative stress, lipid metabolism alterations, and antioxidant enzyme dysfunction, which collectively accelerate atherosclerosis and cardiovascular complications.[1]

Among the numerous biomarkers implicated in CKD-associated cardiovascular risk, Serum Paraoxonase 1 (PON1) and Serum Oxidised LDL (Ox-LDL) have garnered particular attention for their pivotal roles in modulating oxidative stress and atherogenesis.[2]

Serum Paraoxonase 1 (PON1) is an HDL-associated enzyme with potent antioxidant and antiatherogenic properties. It hydrolyzes oxidized lipids in both HDL and LDL, thereby preventing lipid peroxidation and attenuating vascular inflammation.[3] Multiple studies report markedly reduced PON1 activity in CKD patients, with further impairment correlating with advancing disease stages.[4,5] The decline in PON1 activity is attributed to chronic uremia, persistent inflammation, dysfunctional HDL metabolism, and accumulation of uremic toxins. Reduced PON1 activity is also linked to increased cardiovascular events, renal

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fibrosis, and accelerated CKD progression. Furthermore, genetic polymorphisms affecting PON1 enzymatic efficiency contribute to variability in CKD severity and susceptibility to atherosclerotic complications.[6]

Serum Oxidised LDL (Ox-LDL), in contrast, is a well-established marker of lipid peroxidation and oxidative injury. Ox-LDL levels rise progressively with worsening renal function and are consistently elevated in CKD cohorts. Functionally, Ox-LDL drives atherosclerosis by promoting foam cell formation, impairing endothelial function, and initiating proinflammatory cytokine cascades. In the context of CKD, excessive Ox-LDL not only augments cardiovascular risk but also directly damages renal tubular structures, induces extracellular matrix remodeling, and accelerates nephron loss.[7]

A growing body of evidence underscores the deleterious interplay between reduced PON1 activity and elevated Ox-LDL levels in CKD—the PON1/Ox-LDL axis. This pathogenic interaction amplifies oxidative stress, fosters vascular calcification, and exacerbates end-organ damage. Evaluating the relationship between Serum PON1 and Ox-LDL across CKD stages, and comparing them with demographically matched control groups, provides critical insights into the progression of both renal and cardiovascular complications.[8] Such analysis also enables better risk stratification and may guide the development of targeted therapeutic interventions aimed at restoring antioxidant defenses and mitigating lipid peroxidation.

CKD patients enrolled in the assessment of PON1 and Ox-LDL interrelationships across various stages of kidney disease progression. By establishing clear demographic parameters, this analysis will facilitate better understanding of how patient characteristics may influence biomarker relationships and contribute to the overall interpretation of oxidative stress dynamics in CKD progression.

MATERIALS AND METHODS :

Participants:

Inclusion criteria: CKD stage 1-3b patients aged 30–60 years, with diagnosed dyslipidemia per NKF KDOQI guidelines and comorbid conditions like type 2 diabetes mellitus, obesity, or hypertension. Exclusion criteria: End-stage renal disease, acute infections, or inflammatory diseases.

Sample Collection:

Blood Samples will be collected from chronic kidney disease patients from Saveetha Medical College and Hospital.10 ml of blood will be collected from the anti-cubital vein under aseptic conditions. Serum is separated after centrifugation. Handle and store specimens in stoppered containers to avoid contamination and evaporation. Refrigerated at - 20 ˚ C (long term storage)

Biochemical Analysis:

Renal function tests (urea, creatinine) and lipid markers measured via Vitros 5600 dry chemistry analyzer. Lipid profile estimation included total cholesterol (CHOD–PAP method), triglycerides (GPO–PAP method), and HDL cholesterol (indirect precipitation method). LDL and VLDL cholesterol were calculated using the Friedewald formula, excluding samples with triglycerides >400 mg/dL. All assays were performed following the manufacturer's instructions with appropriate quality control, and reference ranges were as per standard clinical laboratory guidelines. Serum PON1 levels assessed using sandwich ELISA. The assay procedure involves running all standards and samples in duplicates or triplicates with a standard curve for each assay. First, 100 µL of standard diluent, standards, and samples are added to the respective wells and incubated for 80 minutes at 37 °C. After washing the plate four times, 100 µL of biotinylated antibody working solution is added and incubated for 50 minutes at 37 °C, followed by another wash. Then, 100 µL of Streptavidin-HRP conjugate is added, incubated for 50 minutes, and washed again. Next, 100 µL of TMB substrate is added and incubated for 10 minutes at 37 °C without shaking. The reaction is stopped with 100 µL of stop solution, changing the color from blue to yellow. Absorbance is read at 450 nm within 10–15 minutes. The mean absorbance values of standards are plotted against concentrations to generate a standard curve, from which sample concentrations are determined, adjusting for dilution if necessary. Serum ox-LDL levels assessed using sandwich ELISA. In this assay, standards and samples are run in duplicates or triplicates, and a standard curve is generated for each test. A 100 µL volume of standard diluent, standards, and samples is added to designated wells and incubated for 80 minutes at 37°C. After washing the plate four times, 100 µL of biotinylated antibody working solution is added and incubated for 50 minutes, followed by washing. Next, 100 µL of Streptavidin-HRP conjugate is added, incubated for 50 minutes, and washed again. Then, 100 µL of TMB substrate is added and incubated for 10 minutes at 37°C without shaking. The reaction is stopped with 100 µL of stop solution, changing the color from blue to yellow. Absorbance is measured at 450 nm within 10–15 minutes, and the analyte concentration is calculated from the standard curve, adjusting for any sample dilution.

Statistical Analysis:

One way ANOVA,Conducted using SPSS v15.0.P-values < 0.05 considered statistically significant.

Ethical clearance:005/04/2024/IEC/SMCH

RESULT

The data collectively demonstrate a strong association between dyslipidaemia and oxidative stress parameters in chronic kidney disease (CKD) patients across stages 1 to 3b. Table 1 shows that among patients with dyslipidaemia, 100% had low serum Paraoxonase 1 (PON1) levels across all CKD stages, with none exhibiting normal levels, whereas in non-dyslipidaemic patients, most maintained normal PON1 activity (ranging from 91.11% to 97.78%). Similarly, Oxidised LDL (Ox-LDL) was significantly elevated in all dyslipidaemic patients (rising from 80% in stage 1 to 100% in stages 2 to 3b), whereas non-dyslipidaemic

patients had uniformly low Ox-LDL levels across all stages, indicating a clear oxidative imbalance associated with lipid abnormalities. Table 2 further reinforces this by showing that dyslipidaemic patients had markedly reduced PON1 levels (mean values declining from 1.764 ng/ml in stage 1 to 0.462 ng/ml in stage 3b) compared to significantly higher levels in non-dyslipidaemic patients (ranging from 5.333 to 5.674 ng/ml), with p-values < 0.001, confirming statistical significance. Table 3 In contrast, Ox-LDL levels in dyslipidaemic patients were substantially elevated (1.723 to 4.824 mg/dl across stages), whereas non-dyslipidaemic patients showed negligible Ox-LDL concentrations (approximately 0.101–0.105 mg/dl). These biochemical alterations support the hypothesis that dyslipidaemia exacerbates oxidative stress in CKD. Moreover, Table 4 presents a correlation analysis between PON1 and Ox-LDL, revealing a stepwise shift from non-significant or weak correlations in early stages to strong negative correlations in advanced CKD, particularly among dyslipidaemic patients (e.g., r = -0.25607 in stage 3b, $p < 0.00001$). Interestingly, in the control group, a positive and highly significant correlation was found (r = 0.258271, $p < 0.00001$), suggesting a normal physiological relationship between antioxidant enzyme and oxidized lipids that is disrupted in CKD, especially when compounded by dyslipidaemia. Overall, these results strongly indicate that dyslipidaemia in CKD patients is closely associated with reduced antioxidant defense (low PON1) and increased oxidative stress (high Ox-LDL), both of which deteriorate further with disease progression, underscoring the importance of early lipid management to mitigate oxidative injury and cardiovascular risk in CKD.

Table 1: PON1–Ox-LDL Comparison in CKD Stages 1–3b

CKD Stages	↓ PON1- Dyslipidaemic		↑ Ox-LDL- Dyslipidaemic	
	With	Without	With	Without
Stage 1	100%	4%	80%	0%
Stage 2	100%	8%	100%	0%
Stage 3a	100%	3 %	100%	0%
Stage 3b	100%	2%	100%	0%

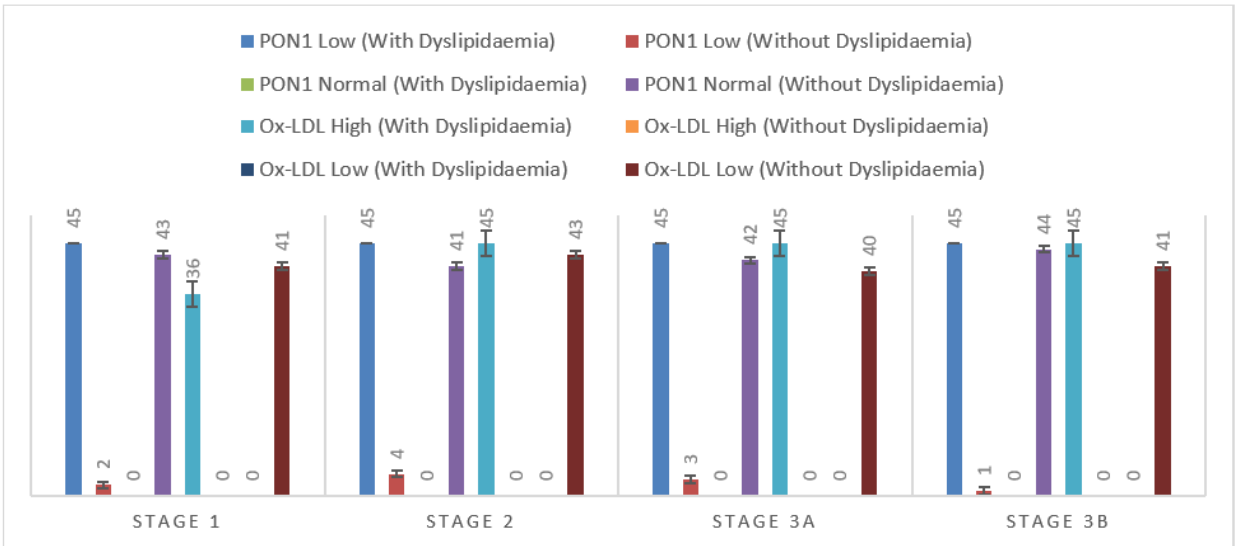

Figure 1: PON1 and Ox-LDL Levels Across CKD Stages 1–3b by Dyslipidaemia Status.The bar chart compares PON1 activity and Ox-LDL levels across CKD stages 1 to 3b, separating patients with and without dyslipidaemia. PON1 levels are consistently lower in the dyslipidaemia group across all CKD stages. Ox-LDL levels are consistently higher in the dyslipidaemia group compared to those without dyslipidaemia.

Table 2: Stage-wise Serum PON1 in CKD (With/Without Dyslipidaemia)

CKD Stages	Status of dyslipidaemia	Serum Peroxonase 1 ng/ml			p-value
		Mean	S. D	Variance	
Stage 1	With	1.764	0.216	0.047	< 0.00001*
	Without	5.397	1.632	2.662	
Stage 2	With	0.848	0.081	0.007	< 0.00001*

	Without	5.333	1.694	2.870	
Stage 3a	With	0.601	0.063	0.004	< 0.00001*
	Without	5.508	1.692	2.862	
Stage 3b	With	0.462	0.168	0.028	< 0.00001*
	Without	5.674	1.589	2.526	
Control group		4.054047	0.784814	0.615933	-
Values are expressed as mean (SD). All mass values are in ng/ml).CKD, chronic kidney disease; SD, standard deviation*P < .05 was considered statistically significant.					

Table 3: Stage-wise Serum Ox-LDL in CKD (With/Without Dyslipidaemia)

CKD Stages	Status of dyslipidaemia	Serum Ox-LDL mg/dl			p-value
		Mean	S. D	Variance	
Stage 1	With	1.723	0.229	0.052	< 0.00001*
	Without	0.103	0.012	0.00014	
Stage 2	With	2.207	0.163	0.027	< 0.00001*
	Without	0.101	0.011	0.00012	
Stage 3a	With	3.171	0.345	0.119	< 0.00001*
	Without	0.105	0.012	0.00016	
Stage 3b	With	4.824	0.520	0.270	< 0.00001*
	Without	0.104	0.012	0.00015	
Control group		0.061456	0.031245	0.000976	-
Values are expressed as mean (SD). All mass values are in mg/dl).CKD, chronic kidney disease; SD, standard deviation*P < .05 was considered statistically significant.					

Table 4: Correlation of PON1 and Ox-LDL in CKD (Stages 1–3b) With/Without Dyslipidaemia

CKD Stages	Status of dyslipidaemia	Correlation between serum PON 1 & Ox-LDL		
		Correlation value	Remarks	p value
Stage 1	With	0.238584	Positively correlated	< 0.00001*
	Without	-0.052540	Negatively correlated	0.39811
Stage 2	With	0.045582	Positively correlated	< 0.00001*
	Without	-0.06574	Negatively correlated	< 0.00001*
Stage 3a	With	-0.20301	Negatively correlated	< 0.00001*
	Without	-0.0456	Negatively correlated	< 0.00001*
Stage 3b	With	-0.25607	Negatively correlated	< 0.00001*
	Without	-0.20273	Negatively correlated	< 0.00001*
Control group		0.258271	Positively correlated	1.8033
Values are expressed as correlation (r). chronic kidney disease; SD, standard deviation*P < .05 was considered statistically significant.				

DISCUSSION

The present study demonstrates a striking decline in serum paraoxonase 1 (PON1) activity and a concomitant rise in oxidized LDL (Ox-LDL) levels across progressive stages of chronic kidney disease (CKD), with the most profound alterations observed in stage 3b patients with dyslipidaemia. Among dyslipidaemic participants, PON1 activity was uniformly low in all CKD stages, whereas non-dyslipidaemic patients largely maintained normal PON1 values. In parallel, Ox-LDL concentrations were consistently elevated in the dyslipidaemic group, underscoring a significant oxidative burden in this population.

These results are consistent with recent findings that CKD is characterized by diminished PON1 activity, which is further aggravated by lipid abnormalities, thereby amplifying oxidative stress. The high

prevalence of low PON1 activity in our dyslipidaemic cohort echoes previous reviews describing HDL dysfunction and reduced antioxidative defense in CKD.[9] Meta-analytic evidence linking low PON1 to renal impairment and increased cardiovascular risk. Moreover, our observations align with mechanistic work showing that PON1 deficiency exacerbates lipid peroxidation and accelerates cardiovascular pathology in CKD models.

The biochemical profile identified in our study suggests that CKD, particularly when compounded by dyslipidaemia, creates a pro-oxidative milieu in which HDL's protective role is compromised. Dysfunctional HDL in CKD has been shown to possess altered composition and reduced capacity to prevent LDL oxidation, thereby facilitating vascular injury. In this context, Ox-LDL not only reflects oxidative stress but may actively contribute to renal injury. Indeed, prior research has shown that oxidized lipoproteins can intensify proteinuria-related damage and accelerate renal function decline.

The correlation analysis in our cohort revealed that in advanced CKD stages, particularly stage 3b, PON1 and Ox-LDL exhibit a strong inverse relationship among dyslipidaemic patients. This finding supports the concept that reduced PON1 enzymatic activity directly permits unchecked LDL oxidation, contributing to a feed-forward loop of oxidative stress and inflammation. Conversely, the positive correlation seen in the control group likely represents the physiological balance between PON1 and lipid oxidation, which is disrupted in CKD.

From a pathophysiological standpoint, reduced PON1 activity in CKD is not solely attributable to decreased protein concentration but may also involve functional impairments such as reduced lactonase activity and post-translational modifications. Experimental data suggest that enhancing PON1 expression or activity could ameliorate renal lipotoxicity through mechanisms such as activation of lipophagy and suppression of pyroptosis.[10]

Advancing age is associated with both progression of CKD and a rise in oxidative stress; many cohorts studying PON1 and ox-LDL show older mean ages in advanced CKD and dialysis groups, which confounds the attribution of lower PON1 activity to kidney dysfunction alone. Several reports therefore adjust for age (and sex) when testing associations between PON1 and ox-LDL. Overall, lower PON1 activity with higher ox-LDL is more pronounced in older patients and in male-predominant cohorts, though sex effects vary among studies.Uremic retention solutes, chronic inflammation, and increased reactive oxygen species in advanced CKD and in older patients can reduce hepatic PON1 synthesis and/or inactivate the enzyme, while simultaneously promoting LDL oxidation.[11] demographic structure across CKD stages shapes the observed relationship between serum PON1 activity and oxLDL. The balance of evidence indicates lower PON1 activity and higher oxidative lipoprotein burden with advancing CKD, modified by comorbidities, lipoprotein composition and treatment modality; integrating standardized biochemical assays, genotyping, and longitudinal designs will strengthen causal inference and guide targeted interventions.[12] The healthcare system is heavily burdened by the increasing number of patients, the high stakes of the end-stage renal disease movement, and the poor visibility of persistent kidney infection (CKD). By causing vascular inflammatory responses, LOX-1 plays a crucial part in the development and advancement of atherosclerotic injuries. One of the main risk factors for atherosclerosis is having high levels of LDLC in the blood.[13] The ratio of triglycerides (TG) to high-density lipoprotein (HDL) cholesterol is a measure of small/dense low-density lipoprotein particles, which are closely associated with certain metabolic and vascular problems.[14] When the body's antioxidant capacity is exceeded by ROS, oxidative stress leads to abnormal apoptosis and inflammation. Dyslipidemia and metabolic syndrome raise ROS.[15]

Taken together, our findings reinforce the utility of simultaneous PON1 and Ox-LDL measurement as complementary biomarkers for risk stratification in CKD. They also highlight the potential benefit of early lipid management strategies aimed at reducing oxidative injury, which may help slow CKD progression and lower cardiovascular risk. Targeted therapeutic interventions such as pharmacologic upregulation of PON1 or lifestyle modifications that improve HDL quality merit further clinical investigation.

CONCLUSION

The present evidence highlights the pivotal role of Paraoxonase 1 (PON1) in protecting renal tissues from lipid-induced damage. By enhancing lipophagy and inhibiting pyroptosis, PON1 mitigates renal lipotoxicity, thereby potentially slowing the progression of chronic kidney disease. These findings open avenues for targeted therapeutic interventions aimed at modulating PON1 activity to improve renal outcomes, particularly in patients with metabolic dysregulation and lipid accumulation.

CONFLICT OF INTEREST:

No conflicts of interest regarding this investigation**.**

AUTHOR CONTRIBUTION

The conception and design of the study, data collection, and laboratory experiments. Prepared the initial draft of the manuscript, analyzed and interpreted the results, reviewed relevant literature, and were involved in the statistical analysis, data interpretation, and subsequent revision and finalization of the manuscript.

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