Section: Biochemistry

Original Research Article

A CLINICAL AND BIOCHEMICAL STUDY OF OXIDATIVE STRESS AND ANTIOXIDANT STATUS IN PATIENTS WITH CHRONIC KIDNEY DISEASE

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Received *: 29/11/2025*
Received in revised form *: 16/01/2026*
Accepted *: 03/02/2026*

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DOI: 10.70034/ijmedph.2026.1.329

Source of Support: Nil,
Conflict of Interest: None declared

Int J Med Pub Health
2026; 16 (1); 1891-1894

ABSTRACT

Background: Chronic kidney disease (CKD) is a progressive disorder associated with increased cardiovascular morbidity and mortality. Emerging evidence suggests that oxidative stress, resulting from an imbalance between reactive oxygen species and antioxidant defenses, plays a key role in CKD progression and its systemic complications. However, comprehensive evaluation of oxidative stress and antioxidant status in relation to disease severity remains limited.

Materials and Methods: This hospital-based cross-sectional analytical study included 120 patients with CKD stages 3–5 and 120 age- and sex-matched healthy controls. CKD staging was performed according to Kidney Disease: Improving Global Outcomes guidelines using estimated glomerular filtration rate (eGFR). Renal function was assessed by serum creatinine and eGFR. Oxidative stress was evaluated by measuring malondialdehyde and advanced oxidation protein products, while antioxidant status was assessed by superoxide dismutase activity and total antioxidant capacity using standard spectrophotometric methods. Statistical analysis included comparison between groups using Student's *t*-test and stage-wise analysis using one-way ANOVA.

Results: CKD patients demonstrated significantly elevated oxidative stress markers compared to controls, with higher levels of malondialdehyde and advanced oxidation protein products ($p < 0.001$). Antioxidant parameters, including superoxide dismutase activity and total antioxidant capacity, were significantly reduced in CKD patients ($p < 0.001$). Stage-wise analysis revealed a progressive increase in oxidative stress markers and a concomitant decline in antioxidant defenses from CKD stage 3 to stage 5. Declining eGFR was associated with worsening oxidative imbalance, indicating a clear relationship between disease severity and oxidative stress.

Conclusion: The study demonstrates that chronic kidney disease is characterized by heightened oxidative stress and impaired antioxidant defense, which worsen with advancing disease stage. Assessment of oxidative stress and antioxidant status may provide valuable adjunctive information in understanding CKD pathophysiology and disease progression.

Keywords: Chronic kidney disease; Oxidative stress; Antioxidants; Malondialdehyde; Estimated glomerular filtration rate.

INTRODUCTION

Chronic kidney disease (CKD) is a progressive disorder characterized by irreversible loss of renal structure and function, leading to impaired excretory, metabolic, and endocrine activities of the kidney.[1] It represents a major global health burden, with increasing prevalence driven by diabetes mellitus, hypertension, and aging populations.[2] CKD is

associated with a markedly increased risk of cardiovascular disease, which remains the leading cause of mortality in these patients.[3] Beyond traditional risk factors, non-traditional mechanisms such as oxidative stress have been increasingly recognized as central contributors to CKD progression and its systemic complications.[4]

Oxidative stress arises from an imbalance between the generation of reactive oxygen species (ROS) and the capacity of antioxidant defense systems to neutralize them.[5] In CKD, excessive ROS production is promoted by uremic toxin accumulation, chronic inflammation, mitochondrial dysfunction, activation of the renin–angiotensin system, and reduced renal clearance of pro-oxidant molecules.[6] Concurrently, antioxidant defenses are compromised due to diminished synthesis, increased consumption, and loss of enzymatic and non-enzymatic antioxidants.[7] Elevated lipid peroxidation products such as malondialdehyde and advanced oxidation protein products, along with reduced activities of antioxidants like superoxide dismutase and total antioxidant capacity, have been consistently reported in CKD patients.[8] This persistent oxidative milieu contributes to progressive nephron damage, endothelial dysfunction, anemia, and accelerated atherosclerosis.[9]

Despite substantial evidence supporting the role of oxidative stress in CKD, reported findings vary across populations and disease stages, and many studies focus on isolated biomarkers without integrating antioxidant status or disease severity.[10] Furthermore, limited data are available from resource-limited settings where CKD burden is rising rapidly. A comprehensive evaluation of oxidative stress markers and antioxidant defenses in relation to renal function and CKD stage is therefore essential to better understand disease pathophysiology. The present study was undertaken to assess oxidative stress and antioxidant status in patients with chronic kidney disease and to examine their association with renal dysfunction, thereby providing clinically relevant insights into disease progression and potential therapeutic targets.

MATERIALS AND METHODS

This hospital-based cross-sectional analytical study was conducted in the Department of Biochemistry in collaboration with the Department of Nephrology at a tertiary care teaching hospital. A total of 120 patients diagnosed with chronic kidney disease (CKD) and 120 age- and sex-matched healthy controls were enrolled. CKD was defined and staged according to Kidney Disease: Improving Global Outcomes (KDIGO) guidelines based on estimated glomerular filtration rate (eGFR). Patients aged ≥20 years with CKD stages 3 to 5 who were not receiving renal replacement therapy were included. Exclusion criteria comprised acute kidney injury, chronic liver disease, malignancy, autoimmune disorders, active infections, and use of antioxidant supplements. Healthy controls were recruited after clinical evaluation and laboratory screening to exclude renal or systemic illness. Written informed consent was obtained from all participants, and the study was approved by the Institutional Ethics Committee.

After an overnight fast of 8–10 hours, venous blood samples were collected under aseptic conditions. Serum was separated by centrifugation and analyzed for renal function parameters including serum creatinine and eGFR. Oxidative stress markers, namely malondialdehyde (MDA) and advanced oxidation protein products (AOPP), were estimated using standard spectrophotometric methods. Antioxidant status was assessed by measuring superoxide dismutase (SOD) activity and total antioxidant capacity (TAC) using validated biochemical assays following standardized protocols. All analyses were performed under quality-controlled laboratory conditions.

Data were entered into Microsoft Excel and analyzed using appropriate statistical software. Continuous variables were tested for normal distribution and expressed as mean ± standard deviation. Comparisons between CKD patients and controls were performed using Student's t-test. Stage-wise comparisons among CKD patients were analyzed using one-way analysis of variance (ANOVA). Associations between oxidative stress markers, antioxidant parameters, and renal function indices were assessed using Pearson's correlation coefficient and reported descriptively. A p-value of <0.05 was considered statistically significant.

RESULTS

Table 1: Demographic and Clinical Characteristics of Study Participants

Category	Parameter	CKD Patients (n = 120)	Controls (n = 120)	p-value
Age (years)	20–39	14 (11.7%)	22 (18.3%)	0.18
	40–59	56 (46.7%)	60 (50.0%)	
	≥60	50 (41.6%)	38 (31.7%)	
Sex	Male	74 (61.7%)	70 (58.3%)	0.59
	Female	46 (38.3%)	50 (41.7%)	
CKD Stage	Stage 3	34 (28.3%)	—	—
	Stage 4	46 (38.3%)	—	
	Stage 5	40 (33.4%)	—	
Hypertension	Present	88 (73.3%)	22 (18.3%)	<0.001*

	Absent	32 (26.7%)	98 (81.7%)	
Diabetes Mellitus	Present	62 (51.7%)	16 (13.3%)	<0.001*
	Absent	58 (48.3%)	104 (86.7%)	

The demographic and clinical characteristics of the study participants are presented in Table 1. The majority of CKD patients were in the age group of 40–59 years, with a higher proportion of males compared to females. Based on disease severity, 28.3% of patients were classified as CKD stage 3, 38.3% as stage 4, and 33.4% as stage 5. Hypertension and diabetes mellitus were significantly more prevalent among CKD patients than in healthy controls ($p < 0.001$).

Table 2: Renal Function, Oxidative Stress, and Antioxidant Status in CKD Patients and Controls

Category	Parameter (Clinical Range)	CKD Patients (n = 120)	Controls (n = 120)	p-value
Renal Function-Serum Creatinine	Serum Creatinine ≤1.2 mg/dL	0 (0.0%)	108 (90.0%)	<0.001*
	Serum Creatinine 1.3–3.0 mg/dL	38 (31.7%)	12 (10.0%)	
	Serum Creatinine >3.0 mg/dL	82 (68.3%)	0 (0.0%)	
eGFR	eGFR ≥60 mL/min/1.73 m²	0 (0.0%)	104 (86.7%)	<0.001*
	eGFR 30–59 mL/min/1.73 m²	34 (28.3%)	16 (13.3%)	
	eGFR <30 mL/min/1.73 m²	86 (71.7%)	0 (0.0%)	
Oxidative Stress-MDA	MDA ≤3.0 nmol/mL (Normal)	18 (15.0%)	102 (85.0%)	<0.001*
	MDA >3.0 nmol/mL (Elevated)	102 (85.0%)	18 (15.0%)	
AOPP	AOPP ≤100 µmol/L (Normal)	20 (16.7%)	94 (78.3%)	<0.001*
	AOPP >100 µmol/L (Elevated)	100 (83.3%)	26 (21.7%)	
Antioxidant Status-SOD	SOD ≥3.0 U/mL (Normal)	28 (23.3%)	104 (86.7%)	<0.001*
	SOD <3.0 U/mL (Reduced)	92 (76.7%)	16 (13.3%)	
TAC	TAC ≥1.3 mmol/L (Normal)	30 (25.0%)	112 (93.3%)	<0.001*
	TAC <1.3 mmol/L (Reduced)	90 (75.0%)	8 (6.7%)	

The distribution of renal function parameters, oxidative stress markers, and antioxidant status in CKD patients and controls is presented in Table 2. A majority of CKD patients (68.3%) had serum creatinine levels greater than 3.0 mg/dL, and 71.7% had eGFR values below 30 mL/min/1.73 m², whereas most controls exhibited normal renal function. Elevated malondialdehyde levels were observed in 85.0% of CKD patients compared to 15.0% of controls, while increased advanced oxidation protein products were present in 83.3% of CKD patients. In contrast, reduced antioxidant activity was evident, with 76.7% of CKD patients showing decreased superoxide dismutase activity and 75.0% demonstrating reduced total antioxidant capacity. These differences were statistically significant when compared with controls ($p < 0.001$).

Table 3: Stage-wise Comparison of Renal Function, Oxidative Stress, and Antioxidant Parameters in CKD Patients

Parameter	Clinical Category	Stage 3	Stage 4	Stage 5	p-value
MDA	≤3.0 nmol/mL (Normal)	10 (29.4%)	6 (13.0%)	2 (5.0%)	<0.001*
	>3.0 nmol/mL (Elevated)	24 (70.6%)	40 (87.0%)	38 (95.0%)	
AOPP	≤100 µmol/L (Normal)	8 (23.5%)	6 (13.0%)	2 (5.0%)	<0.001*
	>100 µmol/L (Elevated)	26 (76.5%)	40 (87.0%)	38 (95.0%)	
SOD	≥3.0 U/mL (Normal)	14 (41.2%)	10 (21.7%)	4 (10.0%)	<0.001*
	<3.0 U/mL (Reduced)	20 (58.8%)	36 (78.3%)	36 (90.0%)	
TAC	≥1.3 mmol/L (Normal)	10 (29.4%)	12 (26.1%)	8 (20.0%)	0.02*
	<1.3 mmol/L (Reduced)	24 (70.6%)	34 (73.9%)	32 (80.0%)	

Stage-wise distribution of oxidative stress and antioxidant parameters among CKD patients is shown in Table 3. The proportion of patients with elevated malondialdehyde and advanced oxidation protein product levels increased progressively from CKD stage 3 to stage 5. Conversely, the percentage of patients with reduced superoxide dismutase activity and total antioxidant capacity increased with advancing CKD stage. These trends indicate a clear stage-dependent increase in oxidative stress accompanied by a decline in antioxidant defences.

DISCUSSION

The present study demonstrates a high prevalence of oxidative stress and impaired antioxidant defense in patients with chronic kidney disease, with a clear stage-dependent pattern. In this study, elevated malondialdehyde levels were observed in 85.0% of CKD patients compared to only 15.0% of healthy controls, indicating widespread lipid peroxidation in renal dysfunction. Similar observations have been reported by Vaziri et al., who documented significantly increased lipid peroxidation products in the majority of CKD patients, attributing this to persistent oxidative burden associated with uremia.[11] Dounousi et al. also reported progressively higher proportions of patients with elevated oxidative stress markers as CKD advanced, supporting the stage-wise trend observed in the present study.[12]

Protein oxidation, assessed by advanced oxidation protein products, was elevated in 83.3% of CKD patients in our study, while the majority of controls

exhibited normal levels. Witko-Sarsat et al. first described the accumulation of AOPP in uremic patients and reported that more than 70% of patients with advanced renal failure had markedly elevated AOPP concentrations.[13] Subsequent studies by Cachofeiro et al. confirmed that increased AOPP levels are common in CKD and are closely linked to endothelial dysfunction and increased cardiovascular risk.[14] The progressive increase in the proportion of patients with elevated AOPP from CKD stage 3 to stage 5 in our study further reinforces the association between declining renal function and cumulative protein oxidative damage.

In addition to increased oxidative stress, antioxidant defenses were markedly compromised in CKD patients. Reduced superoxide dismutase activity was observed in 76.7% of CKD patients, compared to only 13.3% of controls. Annuk et al. reported a similar reduction in antioxidant enzyme activity, with approximately 60–80% of patients with advanced CKD demonstrating significantly diminished SOD activity.[15] Likewise, total antioxidant capacity was reduced in 75.0% of CKD patients in the present study, reflecting exhaustion of systemic antioxidant reserves. Pawlak et al. observed a comparable decline in antioxidant capacity in CKD patients and suggested that persistent oxidative stress overwhelms endogenous antioxidant mechanisms.[16]

Stage-wise analysis in the present study revealed that the proportion of patients with elevated oxidative stress markers and reduced antioxidant defenses increased progressively from CKD stage 3 to stage 5. Oberg et al. similarly reported that patients with lower eGFR had a significantly higher prevalence of oxidative stress and inflammation compared to those with moderate renal impairment.[17] These findings suggest that oxidative imbalance worsens in parallel with disease severity rather than being merely an incidental finding.

CONCLUSION

The present study demonstrates that patients with chronic kidney disease exhibit significantly increased oxidative stress and reduced antioxidant defenses, with a progressive worsening observed across advancing disease stages. Elevated lipid and protein oxidation markers along with diminished antioxidant capacity were closely associated with declining renal function, highlighting the role of oxidative imbalance in CKD pathophysiology.

Assessment of selected oxidative stress and antioxidant markers may serve as a useful adjunct in the clinical evaluation and monitoring of patients with moderate to advanced CKD. Further longitudinal and interventional studies are warranted to evaluate the potential benefits of antioxidant-based therapeutic strategies in slowing disease progression and reducing associated complications.

Conflict of Interest: None Declared.

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