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Assessment of Serum Biomarkers of Oxidative Stress in Patients with Chronic Renal Failure: A Cross-Sectional Study

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ABSTRACT

Background: Chronic renal failure (CRF) is a progressive and irreversible decline in kidney function associated with increased oxidative stress, chronic inflammation, endothelial dysfunction, and a high risk of cardiovascular complications. Oxidative stress plays a crucial role in the progression of renal injury and contributes significantly to adverse clinical outcomes. This study was designed to evaluate renal function parameters, oxidative stress biomarkers, antioxidant status, inflammatory markers, and endothelial dysfunction indicators in patients with chronic renal failure and compare them with healthy controls.

Materials and Methods: A total of 128 participants, comprising 64 patients with chronic renal failure and 64 age and gender matched healthy controls were included. Renal function was assessed using serum urea, creatinine, uric acid, and estimated glomerular filtration rate (eGFR). Oxidative stress was evaluated by measuring malondialdehyde (MDA), reactive oxygen species (ROS), and advanced oxidation protein products (AOPP). Antioxidant status was assessed using superoxide dismutase (SOD), catalase, glutathione, and total antioxidant capacity. Inflammatory markers and endothelial dysfunction parameters were also analysed.

Results: CRF patients exhibited significantly elevated serum urea, creatinine, uric acid, MDA, ROS, AOPP, hs-CRP, interleukin-6, tumor necrosis factor-α, and homocysteine levels compared with controls ($p<0.001$). Antioxidant biomarkers including SOD, catalase, glutathione, total antioxidant capacity, and nitric oxide were significantly reduced ($p<0.001$). Oxidative stress markers showed a strong positive correlation with serum creatinine, whereas antioxidant parameters demonstrated significant negative correlations with disease severity.

Conclusion: Chronic renal failure is associated with enhanced oxidative stress, impaired antioxidant defense, chronic inflammation, and endothelial dysfunction. These biomarkers may serve as useful indicators of disease progression and cardiovascular risk, facilitating early intervention and improved clinical management in patients with chronic renal failure.

Keywords*: Chronic renal failure, Chronic kidney disease, Oxidative stress, Inflammation, Endothelial dysfunction, Antioxidants, eGFR.*

INTRODUCTION

Chronic kidney disease (CKD), previously referred to as chronic renal failure (CRF), is a progressive and irreversible deterioration of renal function characterized by structural or functional abnormalities of the kidneys persisting for more than three months. It represents a major global public health challenge due to its increasing prevalence, substantial healthcare burden, and association with premature morbidity and mortality. Recent Kidney Disease: Improving Global Outcomes (KDIGO) guidelines emphasize that CKD affects nearly 10–15% of the global population and significantly increases the risk of cardiovascular disease, hospitalization, and all-cause mortality (1, 2)

The progression of CKD is accompanied by the gradual loss of nephron function, resulting in impaired excretion of metabolic waste products, electrolyte imbalance, endocrine dysfunction, and accumulation of uremic toxins. These alterations contribute to a complex pathophysiological milieu involving chronic inflammation, endothelial dysfunction, oxidative stress, and accelerated atherosclerosis. Cardiovascular disease remains the leading cause of death among CKD patients, with risk increasing as renal function declines (3-5).

Among the non-traditional cardiovascular risk factors, oxidative stress has emerged as a critical mechanism contributing to both renal injury and cardiovascular complications. Oxidative stress develops when the production of reactive oxygen species (ROS) exceeds the capacity of endogenous antioxidant defense systems. Increased generation of free radicals, mitochondrial dysfunction, chronic inflammation, and reduced antioxidant activity collectively promote oxidative damage to lipids, proteins, and nucleic acids in CKD patients (6-9). This oxidative imbalance accelerates renal fibrosis, endothelial dysfunction, vascular calcification, and progression of kidney disease (10,11).

Several biomarkers have been investigated to assess oxidative stress and antioxidant status in CKD. Lipid peroxidation products such as malondialdehyde (MDA) reflect oxidative membrane damage, whereas antioxidant enzymes including superoxide dismutase (SOD) represent protective cellular defense mechanisms. Altered nitric oxide metabolism and elevated inflammatory mediators further contribute to endothelial dysfunction and increased cardiovascular risk (12-15). Previous studies have demonstrated significantly elevated oxidative stress markers and reduced antioxidant levels in patients with advanced CKD and those undergoing dialysis (16, 17).

Despite increasing evidence supporting the role of oxidative stress in CKD pathogenesis, data regarding the relationship between renal dysfunction, oxidative injury, antioxidant status, and endothelial biomarkers remain limited in the Indian population. Therefore, the present study was undertaken to evaluate serum biomarkers of oxidative stress, antioxidant defence, and endothelial dysfunction in patients with chronic renal failure and to examine their association with disease severity and renal function parameters.

MATERIALS DN METHODS

This hospital-based analytical cross-sectional study was conducted in the Department of Biochemistry at Arundathi Institute of Medical Sciences, Malkajgiri, Telangana from January 2025 to January 2026. A total of 128 participants who are clinically diagnosed with chronic renal failure referred for laboratory investigations and age and gender matched healthy volunteers with normal renal functional tests were recruited as control subjects.

Inclusion Criteria: Adults aged 18-70 years, clinically diagnosed with chronic renal failure, with documented renal dysfunction for ≥3 months, and willing to provide informed consent.

Exclusion Criteria: Acute kidney injury, acute inflammatory conditions, chronic liver disease, malignancy, autoimmune disorders, pregnancy, active infection, recent surgery, under antioxidant supplementation, current smokers and alcohol dependence, and patients on immunosuppressive therapy.

The Written informed consent was obtained from all the study participants and study protocol was approved by the institutional ethics committee.

A structured proforma was used to collect demographic information, clinical variables, details of clinical examination. After overnight fasting, approximately 10mL of venous blood was collected under aseptic precautions by the experienced phlebotomist. Blood samples were centrifuged at 3000 rpm for 10 minutes and separated serum was stored at −80°C until analysis.

The following laboratory investigations were performed including renal Function test such as blood urea, serum creatinine, Blood Urea Nitrogen (BUN), uric Acid, estimated GFR, serum albumin and total protein. Electrolyte Profile including serum sodium, potassium, chloride, calcium, magnesium and bicarbonate. Glycaemic Parameters including FBS, PBS and HbA1c. Lipid Profile including total Cholesterol, triglycerides, HDL, LDL, VLDL, Non-HDL and Atherogenic Index of Plasma. Inflammatory markers such as hs-CRP, ESR, IL-6 and TNF- α, oxidative Stress Biomarkers including MDA, ROS, AOPP, Lipid peroxidation products, antioxidant Biomarkers like SOD, Catalase, GSH, GPx and TAC and endothelial dysfunction markers including Nitric Oxide (NO), Homocysteine and Paraoxonase-1.

Data were entered into Microsoft Excel sheet and analysed using SPSS v.26.0. Continuous variables were expressed as Mean ± Standard Deviation. Categorical variables were expressed as frequencies and percentages. A p-value <0.05 was considered statistically significant.

RESULTS

Table 1: Demographic profile of study subjects

Variable	Controls (n=64)	CRF Patients (n=64)	p-value
Age (years)	52.17 ± 10.84	54.28 ± 11.36	0.287
Male, n (%)	38 (59.4)	40 (62.5)	0.718
Female, n (%)	26 (40.6)	24 (37.5)	0.718

Table 2: Clinical characteristics of CRF patients

Variable	Value
Disease duration (In years)	4.72 ± 2.14
Hypertension (%)	70.3%
Diabetes Mellitus (%)	42.2%
Haemodialysis (%)	34.4%
BMI (kg/m²)	24.71 ± 3.86
Systolic BP (mmHg)	148.3 ± 18.5
Diastolic BP (mmHg)	91.4 ± 11.7

Table 3: Comparison of renal function parameters

Parameter	Controls	CRF Patients	p-value
Urea (mg/dL)	27.48 ± 7.21	102.84 ± 39.15	<0.001
Creatinine (mg/dL)	0.96 ± 0.22	5.92 ± 3.11	<0.001
Uric Acid (mg/dL)	4.32 ± 1.04	8.76 ± 2.51	<0.001
eGFR (mL/min/1.73m²)	112.4 ± 15.8	32.7 ± 18.9	<0.001

Table 4: Comparison of serum electrolyte levels

Parameter	Controls	CRF Patients	p-value
Sodium (mEq/L)	139.8 ± 3.1	134.2 ± 4.8	<0.001
Potassium (mEq/L)	4.1 ± 0.4	5.8 ± 0.9	<0.001
Calcium (mg/dL)	9.2 ± 0.6	8.1 ± 0.7	<0.001
Phosphorus (mg/dL)	3.6 ± 0.8	5.7 ± 1.2	<0.001

Table 5: Comparison of lipid profile.

Parameter	Controls	CRF Patients	p-value
Total Cholesterol (mg/dL)	171.8 ± 28.4	203.6 ± 36.8	<0.001
Triglycerides (mg/dL)	118.4 ± 27.1	184.9 ± 49.3	<0.001
LDL-C (mg/dL)	104.6 ± 22.3	138.7 ± 31.5	<0.001
HDL-C (mg/dL)	46.8 ± 8.2	33.4 ± 6.5	<0.001

Table 6: Comparison of inflammatory markers

Parameter	Controls	CRF Patients	p-value
hs-CRP (mg/L)	0.72 ± 0.26	4.92 ± 1.87	<0.001
ESR (mm/hr)	11.2 ± 4.1	39.6 ± 11.3	<0.001
IL-6 (pg/mL)	4.3 ± 1.7	16.2 ± 5.4	<0.001
TNF-α (pg/mL)	8.4 ± 2.1	21.7 ± 6.8	<0.001

Table 7: Comparison of oxidative stress markers

Parameter	Controls	CRF Patients	p-value
MDA (nmol/mL)	2.71 ± 0.81	7.18 ± 1.94	<0.001
ROS (AU/mL)	1.26 ± 0.41	4.82 ± 1.37	<0.001
AOPP (µmol/L)	45.6 ± 13.8	132.5 ± 34.6	<0.001

Table 8: Comparison of antioxidant markers

Parameter	Controls	CRF Patients	p-value
SOD (U/mL)	5.87 ± 1.13	3.12 ± 0.96	<0.001
Catalase (kU/L)	61.4 ± 9.3	41.8 ± 8.1	<0.001
GSH (mg/dL)	41.7 ± 8.5	24.6 ± 6.3	<0.001
TAC (mmol/L)	1.68 ± 0.29	0.89 ± 0.24	<0.001

Table 9: Comparison of endothelial dysfunction markers

Parameter	Controls	CRF Patients	p-value
Nitric Oxide (µmol/L)	39.7 ± 7.1	21.6 ± 5.2	<0.001
Homocysteine (µmol/L)	8.4 ± 2.6	24.7 ± 7.5	<0.001
Paraoxonase-1 (U/mL)	95.4 ± 18.7	58.1 ± 16.3	<0.001

Table 10: Correlation of oxidative stress markers with serum creatinine

Parameter	Correlation coefficient (r)	p-value
MDA	+0.742	<0.001
ROS	+0.681	<0.001
AOPP	+0.709	<0.001
SOD	−0.654	<0.001
Nitric Oxide	−0.621	<0.001

DISCUSSION

The present study evaluated renal function parameters, oxidative stress biomarkers, antioxidant status, inflammatory markers, and endothelial dysfunction indicators among patients with chronic renal failure (CRF). The findings demonstrated significant alterations in biochemical parameters, characterized by elevated serum urea, creatinine, lipid peroxidation products, inflammatory markers, and homocysteine levels, accompanied by reduced antioxidant enzyme activity and nitric oxide bioavailability. These observations support the growing evidence that oxidative stress and inflammation play critical roles in the progression of chronic kidney disease (CKD) and its cardiovascular complications.
As expected, serum urea and creatinine concentrations were significantly higher in CRF patients compared with healthy controls, reflecting impaired glomerular filtration and progressive nephron loss. Similar findings have been reported by KDIGO guidelines and several epidemiological studies, which identified declining estimated glomerular filtration rate (eGFR) as a major determinant of adverse renal and cardiovascular outcomes (1,18). The reduction in eGFR observed in the present study confirms progressive renal dysfunction and supports its utility as a reliable marker of disease severity.

A major finding of this study was the significant increase in malondialdehyde (MDA) and other oxidative stress biomarkers among CRF patients. MDA is a well-recognized product of lipid peroxidation and serves as an indicator of oxidative damage to cellular membranes. Elevated MDA levels in CKD have been consistently documented and are attributed to excessive production of reactive oxygen species (ROS), mitochondrial dysfunction, chronic inflammation, and accumulation of uremic toxins (7, 12). Podkowińska and Formanowicz reported that oxidative stress contributes directly to renal fibrosis, endothelial injury, and progression of kidney disease (6). The strong positive correlation observed between serum creatinine and MDA in the present study further supports the association between worsening renal function and oxidative damage.

The study also demonstrated a significant reduction in antioxidant defence mechanisms, including superoxide dismutase (SOD), catalase, glutathione, and total antioxidant capacity. These findings are in agreement with previous investigations showing depletion of endogenous antioxidant systems in CKD patients (8, 17). Reduced antioxidant activity may result from increased utilization of antioxidant molecules in response to persistent oxidative stress. Consequently, the imbalance between oxidant generation and antioxidant defence promotes cellular injury and accelerates disease progression.

Inflammatory biomarkers, including high-sensitivity C-reactive protein (hs-CRP), interleukin-6, and tumor necrosis factor-alpha, were significantly elevated among CRF patients. Chronic low-grade inflammation is increasingly recognized as a hallmark of CKD and contributes to endothelial dysfunction, vascular calcification, and accelerated atherosclerosis (5, 10). Elevated hs-CRP levels observed in the present study are consistent with reports indicating that inflammation is an independent predictor of cardiovascular morbidity and mortality in CKD populations (15).

Another important observation was the significant reduction in nitric oxide levels and elevation of homocysteine concentrations among CRF patients. Nitric oxide plays a critical role in maintaining vascular homeostasis through regulation of vasodilation, platelet aggregation, and endothelial integrity. Reduced nitric oxide bioavailability, together with hyperhomocysteinemia, has been implicated in endothelial dysfunction and cardiovascular disease among CKD patients (14). The findings of the present study support the concept that endothelial dysfunction develops early in renal impairment and worsens with disease progression.

CONCLUSION

The present study demonstrated that chronic renal failure is associated with significant alterations in renal function, oxidative stress, antioxidant defense mechanisms, inflammatory status, and endothelial function. Patients with chronic renal failure exhibited elevated levels of serum urea, creatinine, lipid peroxidation products, inflammatory biomarkers, and homocysteine, along with reduced levels of superoxide dismutase, nitric oxide, and other antioxidant parameters.

These findings suggest that oxidative stress and chronic inflammation play important roles in the progression of renal dysfunction and the development of cardiovascular complications. Assessment of these biomarkers may aid in disease monitoring, risk stratification, and identification of potential therapeutic targets in chronic renal failure patients.

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