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Original Research Article

Oxidative stress and antioxidant imbalance in chronic renal failure: relationship with serum creatinine and selenium levels

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ABSTRACT

Background: Chronic renal failure (CRF) is marked by progressive renal dysfunction and enhanced oxidative stress, contributing to cellular injury and disease progression. This study aimed to assess oxidative stress, antioxidant enzyme activities, and serum selenium levels in CRF patients and explore their relationship with renal function indices.
Methods: A cross-sectional case-control study was conducted on 46 patients with different degrees of CRF and 16 age- and sex-matched healthy controls. Renal function was assessed by serum creatinine, creatinine clearance, and estimated glomerular filtration rate (eGFR). Serum lipid peroxidation (MDA), superoxide dismutase (SOD), catalase, glutathione peroxidase, and selenium levels were estimated using standard biochemical methods. Data were analyzed using ANOVA and Pearson's correlation.
Results: Serum creatinine increased progressively from 0.93±0.21 mg/dl in controls to 7.82±1.51 mg/dl in severe CRF ($p<0.001$). Lipid peroxidation was significantly elevated in all CRF groups (14.2-15.9 nm MDA/ml vs. 5.1±1.1 nm MDA/ml, $p<0.01$). SOD (2.2-2.8 IU/mg protein) and catalase (2.5-3.0 IU/mg protein) activities were significantly higher than controls (1.5 IU/mg protein; $p<0.05$), indicating compensatory antioxidant upregulation. Glutathione peroxidase and selenium levels showed mild, nonsignificant reductions ($p>0.05$). No significant correlations were observed between serum creatinine and oxidative or antioxidant parameters.
Conclusions: CRF is associated with sustained oxidative stress and adaptive elevation of SOD and CAT, alongside declining selenium and glutathione peroxidase activity. Persistent redox imbalance likely contributes to renal injury, underscoring the need for antioxidant-focused management in CRF.

Keywords: Chronic renal failure, Glutathione peroxidase, Oxidative stress, Antioxidant enzymes, Superoxide dismutase, Selenium, Serum creatinine

INTRODUCTION

Chronic renal failure (CRF), also known as chronic kidney disease (CKD), is a major global public health concern characterized by a gradual and irreversible decline in renal function.[1,2] Progressive reductions in glomerular filtration rate (GFR) and corresponding elevations in serum creatinine are accompanied by metabolic disturbances, accumulation of uremic toxins, and a heightened risk of cardiovascular morbidity and mortality. In addition to established risk factors, such as hypertension, diabetes mellitus, and dyslipidemia, emerging evidence highlights the contributory roles of oxidative stress and impaired antioxidant defense mechanisms in the pathogenesis and progression of CRF.[2,3] Oxidative stress arises when the generation of reactive oxygen species (ROS) exceeds the capacity of endogenous antioxidant systems to neutralize them, resulting in damage to lipids, proteins, and nucleic acids, and promoting inflammation, fibrosis, and cellular apoptosis.[2-4] Multiple mechanisms contribute to this oxidative milieu in CKD, including mitochondrial dysfunction, NADPH oxidase activation, accumulation of

advanced glycation end-products (AGEs), endothelial nitric oxide deficiency, and persistent low-grade inflammation.[1,5,6] Retained uremic toxins, hypertension, and activation of the renin–angiotensin–aldosterone system further aggravate oxidative injury.[1,2] Studies have consistently reported elevated oxidative-damage biomarkers, such as malondialdehyde (MDA) and advanced oxidation protein products, even in moderate renal impairment, accompanied by diminished activities of key antioxidant enzymes, including superoxide dismutase (SOD), glutathione peroxidase (GPx), and catalase (CAT).[3,7,8]

The enzymatic antioxidant defense network, SOD, CAT, and GPx, forms the primary line of protection against ROS. SOD catalyzes the dismutation of superoxide anions into hydrogen peroxide; CAT converts hydrogen peroxide into water and oxygen; and GPx reduces hydrogen peroxide and lipid hydroperoxides using reduced glutathione. In CRF, these systems are compromised due to decreased enzyme expression, impaired glutathione availability, and depletion of essential trace elements.[8,9] Among these, selenium (Se) plays a critical role as a structural and functional component of GPx. Selenium deficiency is well-documented in CKD, attributed to dietary insufficiency, urinary and dialysis-related losses, and altered metabolism.[9] Plasma Se and GPx activity decline with worsening renal function, and although Se supplementation modestly increases GPx activity, its clinical benefits on renal outcomes remain unproven.[9-12]

Despite the extensive evidence linking oxidative stress with CKD progression, most studies have focused on end-stage or dialysis populations. Consequently, the relationship between renal function parameters and oxidative or antioxidant markers during earlier stages of CRF remains insufficiently characterized. Limited data exist on the correlations between oxidative stress biomarkers and key indices of renal function, such as serum creatinine and creatinine clearance. Understanding these associations may provide deeper insight into the development of redox imbalance in CRF and its potential clinical significance. The present study was therefore designed to determine the role of oxidative stress and involvement of the antioxidant enzyme system across varying stages of renal failure. We hypothesized that worsening renal function would be associated with heightened oxidative stress, reduced antioxidant enzyme activities, and lower selenium levels.

METHODS

Study design and population

This was a hospital-based, cross-sectional, case-control study conducted to assess oxidative stress and antioxidant status in patients with CRF. The study was carried out from 1998 to 2001 at the Department of Nephrology, Seth G.S. Medical College and KEM Hospital, Mumbai. The study enrolled forty-six (n=46) patients diagnosed with CRF attending the nephrology outpatient and inpatient hospital settings. An equal number of age and sex matched healthy individuals were recruited as controls.

Ethical considerations

The data used in this study were retrospective and fully anonymized prior to analysis. No additional procedures, samples, or interventions were performed for research purposes. Written informed consent had been obtained from all participants at the time of data collection for the use of their clinical information. Formal Ethical Committee approval was not required at the time the study was conducted, in accordance with the institutional Ethical Committee policies then in effect. The study adhered to the principles of the Declaration of Helsinki.

Inclusion and exclusion criteria

Patients aged between 20 and 65 years with established CRF, defined as persistently elevated serum creatinine and/or reduced GFR for at least three months, were included. Subjects with acute renal failure, active infection, hepatic dysfunction, malignancy, diabetes mellitus with poor glycemic control, or those receiving corticosteroid therapy, antioxidant supplementation, or on long-term alcohol or tobacco use were excluded. Pregnant or lactating women were also excluded from the study.

Clinical and biochemical evaluation

A detailed clinical history, including duration and etiology of renal disease, was recorded for each patient. Routine hematological and biochemical investigations were carried out, including blood urea, serum creatinine, electrolytes, fasting blood glucose, and urinalysis. Serum creatinine was measured using the modified Jaffe's kinetic method. Creatinine clearance was calculated using a 24-hour urine collection and corrected for body surface area. For female participants, values were adjusted using a factor of 0.85.

Assessment of renal function by radionuclide scintigraphy

Renal functional assessment was further performed using a radionuclide scan with technetium^{99m} diethylene triamine pentaacetic acid (^{99m}Tc-DTPA). A rapid intravenous bolus of ^{99m}Tc-DTPA (5 mCi) was administered, and sequential dynamic images were acquired at two-second intervals for the first minute and then integrated at thirty-second intervals for a total of twenty minutes using a gamma camera. Glomerular filtration rate was calculated by the Gates' method, providing an objective evaluation of renal function.

Estimation of oxidative stress and antioxidant parameters

Morning venous blood samples were collected after a ≥10-hour fast to assess oxidative stress and antioxidant

parameters. Lipid peroxidation was measured as serum MDA using the TBARS assay. Serum SOD activity was determined by the pyrogallol autoxidation inhibition method, and catalase activity was assessed by monitoring the decomposition of hydrogen peroxide at 240 nm. GPx activity was quantified using hydrogen peroxide and reduced glutathione, with NADPH consumption at 340 nm used to calculate activity. Serum selenium concentration was measured fluorometrically with diaminonaphthalene after wet digestion and expressed in µg/dl.

Grouping of study subjects

Based on serum creatinine concentrations and creatinine clearance values, the study participants were categorized into four groups representing progressively increasing severity of renal impairment. Group I included patients with serum creatinine levels ranging from 1.5 to 3.0 mg/dl, Group II comprised those with levels between 3.1 and 6.0 mg/dl, Group III included patients with serum creatinine concentrations of 6.1 to 9.0 mg/dl, and Group IV consisted of patients with serum creatinine levels exceeding 9.0 mg/dl. In addition, a control group was established, consisting of subjects with normal renal function, defined as serum creatinine levels below 1.2 mg/dl and a GFR greater than 90 ml/min.

Statistical analysis

All data were expressed as mean±standard deviation (SD). Statistical analysis was performed using SPSS software version (insert version). Differences between groups were analyzed using Student's *t*-test or one-way analysis of variance (ANOVA) as appropriate. Correlation between serum creatinine and oxidative stress parameters was assessed using Pearson's correlation coefficient (*r*). A p value <0.05 was considered statistically significant.

RESULTS

A total of 46 patients with chronic renal failure (CRF) of varying severity and etiology, who satisfied the inclusion criteria, were included in the study along with 16 normal healthy controls (Group I).

Table 1: Patient demographic data.

Group	Mean age (years)	Males (N)	Females (N)
I (n=16)	42.37±11.41	10	6
II (n=16)	42±9.38	12	4
III (n=20)	38.50±12.01	8	12
IV (n=10)	43.40±12.85	7	3

Data presented as Mean±SD.

The renal failure groups comprised 27 males and 19 females, while the control group included 10 males and six females. All subjects were between 18 and 65 years of age (Table 1).

Renal function parameters

Serum creatinine, creatinine clearance, and GFR measured by ^{99m}Tc DTPA showed progressive deterioration with increasing severity of renal failure (Table 2). The mean serum creatinine increased from 0.925±0.211 mg/dl in controls (Group I) to 7.82±1.51 mg/dl in Group IV. Correspondingly, creatinine clearance declined from 85.36±21.29 ml/min in controls to 8.74±4.74 ml/min in Group IV. The 99mTc DTPA-measured GFR also decreased from 90.24±30.05 ml/min in Group I to 14.63±5.88 ml/min in Group IV.

Oxidative stress and antioxidant parameters

Serum oxidative stress parameters, antioxidant enzyme activities, and selenium levels are presented in Table 3. Data presented as Mean±SD. p values compared to the control group (Group I), ns: nonsignificant. GPx: Glutathione peroxidase; SOD: Superoxide dismutase.

Lipid peroxidation levels were significantly elevated in all CRF groups (Groups II–IV) compared with controls ($p<0.01$ for all), indicating increased oxidative stress in CRF. However, no significant differences in lipid peroxidation were observed between the CRF subgroups.

Serum glutathione peroxidase levels showed a mild, nonsignificant reduction in all CRF groups compared to controls ($p>0.05$). Serum catalase activity was significantly increased in all CRF groups ($p<0.01$), though the rise did not differ significantly across the stages of renal failure. Similarly, serum superoxide dismutase (SOD) levels were significantly higher in all CRF groups compared to controls ($p<0.05$), but no progressive trend with disease severity was evident. Serum selenium concentrations were lower in all CRF groups relative to controls, but these reductions were not statistically significant ($p>0.05$).

Overall, these findings indicate that oxidative stress increases and antioxidant enzyme activities are upregulated in patients with chronic renal failure, irrespective of disease severity, while selenium levels tend to decrease without statistical significance.

Correlation analysis

Correlation analysis between serum creatinine and oxidative stress parameters, antioxidant enzyme activities, and selenium levels is shown in Table 4. In the control group (Group I), serum creatinine showed a positive but nonsignificant correlation with lipid peroxidation, glutathione peroxidase, catalase, SOD, and selenium. In CRF groups (II-IV), serum creatinine exhibited weak and nonsignificant negative correlations with most parameters, particularly lipid peroxidation, catalase, and selenium. None of the observed correlations reached statistical significance.

Table 2: Serum creatinine, creatine clearance, and 99MTC DTPAR GFR.

Group	Serum creatinine (mg/dl)	Creatinine clearance (ml/min)	99MTC DTPAR GFR (ml/min)
I (n=16)	0.925±0.211	85.36±21.29	90.24±30.05
II (n=16)	2.33±0.522	34.00±8.57	32.56±11.77
III (n=20)	4.43±0.685	16.49±4.91	26.67±11.50
IV (n=10)	7.82±1.51	8.74±4.74	14.63±5.88

Data presented as Mean±SD. GFR: Glomerular filtration rate.

Table 3: Serum oxidative stress parameters, antioxidant parameters, and selenium levels.

Group	Serum lipid peroxidation (nmMDA/ml)	Serum GPX (IU/mgPRC)	Serum catalase (IU/mgPRC)	Serum SOD (IU/mgPRC)	Selenuim (ng/dl)
I (n=16)	5.14±1.11	1.45±0.43	1.53±0.27	1.49±0.24	75.21±9.92
II (n=16)	15.90±3.10 (p=0.007)	0.69±0.52 (p=ns)	2.95±0.64 (p=0.003)	2.77±1.30 (p=0.001)	59.53±7.02 (p=ns)
III (n=20)	14.23±3.10 (p=0.000)	0.80±0.69 (p=ns)	2.49±0.64 (p=0.008)	2.69±0.88 (p=0.012)	55.57±11.80 (p=ns)
IV (n=10)	14.38±3.65 (p=0.002)	0.66±0.53 (p=ns)	2.65±0.76 (p=0.009)	2.23±0.85 (p=0.024)	64.25±17.16 (p=ns)

Data presented as Mean±SD. p values compared to the control group (Group I), ns: nonsignificant. GPx: Glutathione peroxidase; SOD: Superoxide dismutase.

Table 4: Correlation coefficients for the correlation of serum creatinine with oxidative stress parameters, antioxidant parameters, and selenium levels.

Group	Serum lipid peroxidation (nmMDA/ml)	Serum GPX (IU/mgPRc)	Serum catalase (IU/mgPRC)	Serum SOD (IU/mgPRC)	Selenuim (ng/dl)
I (n=16)	0.2006 (p=0.4563)	0.3242 (p=0.2205)	0.2388 (p=0.3731)	0.0428 (p=0.8749)	0.3231 (p=0.2223)
II (n=16)	-0.2034 (p = 0.4498)	0.1394 (p=0.6067)	-0.1296 (p=0.6325)	0.3199 (p=0.2271)	0.0687 (p=0.8005)
III (n=20)	-0.0362 (p = 0.8795)	0.1475 (p=0.5350)	-0.2944 (p=0.2076)	0.2870 (p=0.2198)	-0.1421 (p=0.5500)
IV (n=10)	-0.0232 (p = 0.9493)	0.5881 (p=0.0737)	0.340 (p=0.3361)	0.2342 (p=0.5148)	0.1447 (p=0.6901)

GPx: Glutathione peroxidase; SOD: Superoxide dismutase.

DISCUSSION

The present study investigated the oxidative stress profile and antioxidant enzyme status in patients with CRF and their relationship with renal function parameters, including serum creatinine and GFR. The findings demonstrated significantly elevated serum lipid peroxidation, indicative of increased oxidative stress, accompanied by enhanced activities of antioxidant enzymes, SOD and catalase, in CRF patients compared to healthy controls. Although serum glutathione peroxidase and selenium levels showed a decreasing trend with worsening renal function, these changes were not statistically significant. Furthermore, correlations between serum creatinine and oxidative/antioxidant parameters were weak and nonsignificant. These results are consistent with prior reports indicating an imbalance between pro-oxidant and antioxidant systems in CKD. A growing body of literature identifies oxidative stress, an imbalance between the ROS generation and antioxidant defenses, as a key contributor to the pathogenesis and progression of CKD.[3] Numerous studies have documented that lipid peroxidation, as reflected by elevated MDA levels, is significantly higher in patients with CKD compared with healthy individuals, irrespective of dialysis status.[3,13] The increased oxidative burden in CRF can be attributed to multiple mechanisms, including the accumulation of uremic toxins, mitochondrial dysfunction, activation of NADPH oxidases, and chronic inflammation.[2,3] In our study cohort, we observed a significantly increased level of lipid peroxidation compared to healthy controls, indicating the presence of oxidative damage in renal impairment. Our observation of heightened MDA levels across all CRF stages supports the hypothesis that oxidative stress is an early and persistent feature of renal dysfunction through the disease spectrum. Though we did not find a strict linear increase of lipid peroxidation with worsening creatinine levels, the sustained increase we observed is in line with

the literature that suggests redox stress is a persistent phenomenon in CKD rather than exclusively a late-stage event.[14]

Antioxidant enzyme behavior: SOD, catalase, and adaptation

It is well established that reduced activity of antioxidant enzymes, together with decreased levels of low–low-molecular-weight antioxidants such as reduced glutathione, contributes significantly to the heightened oxidative burden observed in patients with CKD.[3] Antioxidant enzyme systems constitute the primary defense against excessive ROS. SOD catalyzes the dismutation of superoxide radicals into hydrogen peroxide, which is subsequently decomposed into water and oxygen by catalase; both are complemented by other systems like glutathione peroxidases.

Multiple studies have demonstrated that patients with CKD often exhibit reduced activity of these enzymes, potentially resulting from the accumulation of uremic toxins, nonenzymatic glycation, or direct oxidative inactivation.[2,3,15] Conversely, some studies have reported preserved or even elevated antioxidant enzyme activity in moderate stages of CKD, suggesting a compensatory upregulation in response to increased oxidative stress.[7,15,16] Such adaptive enhancement has been proposed as a transient protective mechanism in uremic conditions; however, it appears insufficient to restore redox equilibrium as oxidative stress continues to accumulate. In the present study, we observed significant upregulation of both SOD and catalase activities in the renal failure groups compared with controls. This finding supports the adaptive-response hypothesis, whereby increased ROS generation stimulates compensatory enhancement of antioxidant enzyme activity to mitigate oxidative stress. Notably, however, no further incremental rise in SOD or CAT activity was detected in groups with higher serum creatinine levels, suggesting a plateau or potential exhaustion of this adaptive mechanism. These results indicate that beyond a certain threshold of renal impairment, the antioxidant defense system may fail to mount a proportional response, consistent with previous reports describing the transient nature and eventual attenuation of compensatory upregulation in CKD.[2,15]

Selenium, glutathione peroxidase, and trace-element dynamics

Studies have reported that plasma glutathione peroxidase activity is significantly lower in CKD patients and decreases progressively with disease stage.[9,16] Selenium is an essential trace element, critical for the biosynthesis of selenoproteins, among which the glutathione peroxidases are important for the reduction of hydrogen peroxide and organic hydroperoxides.[9,17] Previous studies have shown that CKD and dialysis patients exhibit markedly reduced plasma selenium and glutathione activity, likely due to urinary and dialysis-related losses, inadequate dietary intake, and altered metabolism.[9,17] Animal models have also demonstrated that selenium deficiency enhances oxidative stress and renal fibrosis via TGF-β1 upregulation.[18] Clinical trials have reported that selenium supplementation (e.g., 200 μg/day for three months) can increase plasma selenium and glutathione peroxidase activity in CKD patients.[10] On the other hand, a study in hemodialysis patients reported that, despite an increase in plasma selenium concentrations, glutathione peroxidase protein levels remained unchanged, suggesting that in advanced renal failure, the ability of kidney to synthesize glutathione peroxidases may be irreversibly compromised.[19] Other literature also suggests mixed results on clinical outcomes with selenium supplementation in CKD patients.[3,20] In the present study, we observed a mild, non-significant reduction in serum selenium levels and a similar trend toward decreased glutathione peroxidase activity in the renal failure groups compared with controls. Although these differences did not reach statistical significance, the direction of change aligns with previous reports demonstrating reduced selenium concentrations and glutathione peroxidase activity in patients with CKD.

Correlations between oxidative/antioxidant markers and renal function metrics

An important consideration is whether oxidative stress parameters and antioxidant enzyme activities correlate quantitatively with indices of renal function, such as serum creatinine or eGFR. Several studies have reported inverse associations between glutathione peroxidase activity and serum creatinine levels or eGFR decline, while others have identified significant links between antioxidant enzyme gene polymorphisms, such as single-nucleotide polymorphisms (SNPs) in SOD or glutathione peroxidase, and accelerated deterioration of renal function.[15,16] In contrast, our findings revealed only weak, non-significant correlations between serum creatinine and the oxidative or antioxidant markers assessed. This suggests that although redox imbalance and compensatory antioxidant responses are evident in renal dysfunction, they may not exhibit a simple linear relationship with conventional renal function indices. Instead, these changes likely reflect multifactorial influences, including nutritional status, inflammatory activity, and comorbid conditions, that are not fully captured by creatinine-based measures alone.

The coexistence of persistent oxidative stress, limited antioxidant up-regulation, and suboptimal selenium/glutathione peroxidase status indicates a chronic redox disequilibrium in renal failure.[7] This imbalance likely contributes to glomerular and tubular injury, interstitial fibrosis, endothelial dysfunction, and cardiovascular comorbidity in CKD.[14] Clinically, oxidative stress biomarkers and antioxidant enzyme profiles may help identify patients at higher risk of progression, while evidence-based antioxidant or trace-element interventions may offer therapeutic benefit. Integrating redox assessment with conventional renal

function markers may therefore provide a more comprehensive understanding of disease trajectory and enhance strategies for risk stratification and management in CKD.

Strengths and limitations

The strength of this study lies in its integrated evaluation of multiple redox parameters, including lipid peroxidation, SOD, catalase, glutathione peroxidase, and selenium, providing a comprehensive assessment of oxidative balance in renal failure. However, several limitations should also be acknowledged.

The cross-sectional design limits the ability to infer causality between oxidative stress and renal function decline, and serum-based measurements may not fully capture intrarenal oxidative dynamics. The modest sample size reduces statistical power, particularly for subgroup analyses stratified by CKD stage, underlying etiology, or comorbid conditions. Additionally, incomplete characterization of dietary selenium intake, other trace elements (e.g., zinc, copper), inflammatory status, and concurrent medications may have introduced residual confounding.

Future studies should adopt longitudinal designs to elucidate temporal relationships, conduct randomized trials assessing targeted antioxidant or selenium supplementation, expand biomarker panels, and incorporate redox profiling into precision approaches for chronic kidney disease management.

CONCLUSION

In conclusion, our findings support the established concept that renal dysfunction is characterized by heightened oxidative stress, accompanied by compensatory upregulation of key antioxidant enzymes such as SOD and catalase, with trends toward reduced selenium levels and diminished glutathione peroxidase activity. Although these changes did not show strong correlations with conventional renal function indices, such as serum creatinine, they nonetheless reflect underlying redox disturbances that may contribute to the progression and complications of CRF. Incorporating redox biology into renal disease management, through the use of oxidative stress biomarkers and targeted interventional strategies, represents a promising avenue for advancing precision-based nephrology care.

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