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Original Research Article

RELATIONSHIP BETWEEN CKD STAGES AND MALONDIALDEHYDE AND SUPEROXIDE DISMUTASE LEVELS

Vivek Chourasiya[1], Shreya Nigoskar[2],Himani M Jheetay[3]

[1] *PhD Scholar, Department of Biochemistry, Malwanchal University, Indore, Madhya Pradesh, India.*
[2]*Professor and Head of the Department (HOD), Department of Biochemistry, Index Medical College, Malwanchal University, Indore, Madhya Pradesh, India.*
[3]*Assistant Professor, Department of Biochemistry, GEIMS, Dehradun, India.*

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Corresponding Author:
Vivek Chourasiya,
PhD Scholar, Department of Biochemistry, Malwanchal University, Indore, Madhya Pradesh, India.
Email: vivekpcps02@gmail.com

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ABSTRACT

Background: Chronic kidney disease (CKD) shows stage-dependent clinical risk. Oxidative stress is considered a key non-traditional pathway in CKD progression but stage-wise deterioration of oxidative burden with parallel antioxidant depletion needs clearer description using simple biomarkers.
Objectives: To compare malondialdehyde (MDA) and superoxide dismutase (SOD) across CKD stages G2–G5 and to examine whether renal indices (serum creatinine, blood urea, eGFR, urine ACR) showed a consistent stage-wise trend as biological validation.
Materials and Methods: This hospital-based cross-sectional analytical study was conducted in the Department of Biochemistry, Index Medical College, Indore (12 months). Adult CKD patients (≥18 years) were staged by KDIGO 2012 eGFR categories into G2, G3a, G3b, G4 and G5 (n=20 per stage). MDA was estimated by TBARS spectrophotometry and SOD activity by NBT inhibition method in erythrocyte lysate. Non-normal variables were analysed using Kruskal–Wallis test. Dunn's post-hoc test with Bonferroni correction was applied for pairwise stage comparisons of MDA and SOD. Categorical variables were compared using Chi-square/Fisher's exact.
Results: Baseline variables were comparable across stages (age p=0.748, BMI p=0.772, sex p=0.436, diabetes p=0.639, hypertension p=0.443). Renal indices worsened progressively from G2 to G5: creatinine median 1.51 to 6.82 mg/dL (H=92.114, p<0.001), urea 41.70 to 171.00 mg/dL (H=90.261, p<0.001), eGFR 76.40 to 9.85 mL/min/1.73 m² (H=95.055, p<0.001), urine ACR 99.95 to 1066.15 mg/g (H=76.675, p<0.001). MDA rose stage-wise from 4.23 to 11.12 nmol/mL (H=88.980, p<0.001) and SOD declined from 6.85 to 2.26 U/mL (H=87.436, p<0.001). Post-hoc testing showed significant differences mainly between early vs advanced stages (MDA: G2 vs G3b/G4/G5, G3a vs G4/G5, G3b vs G5; SOD: G2 vs G3b/G4/G5, G3a vs G4/G5, G3b vs G5; all adjusted p<0.001).
Conclusion: Oxidative burden (MDA) increased and antioxidant defence (SOD) decreased in a clear severity-linked gradient across CKD G2–G5, paralleling progressive impairment of renal indices. This dual-marker framework describes stage-dependent redox imbalance and supports further longitudinal validation for monitoring and risk profiling.
Keywords: chronic kidney disease, oxidative stress, malondialdehyde, superoxide dismutase, CKD staging, albumin–creatinine ratio.

INTRODUCTION

Chronic kidney disease (CKD) is a progressive condition where loss of nephron mass leads to a sustained fall in glomerular filtration and rising uremic milieu. Clinical risk is strongly stage-dependent. Large population data have shown that lower eGFR tracks with higher rates of death and cardiovascular events, even before kidney failure, so CKD staging is not just descriptive but prognostic too.[1]

Oxidative stress is considered a major non-traditional pathway linking CKD progression with vascular and systemic injury. In moderate to severe CKD, an oxidant–inflammatory phenotype is often present, with elevated oxidative injury markers and inflammatory mediators suggesting persistent redox imbalance even in pre-dialysis stages.[2] This is biologically plausible because uremic toxins, chronic inflammation, RAAS activation, endothelial activation and mitochondrial dysfunction can increase reactive oxygen species generation, while antioxidant reserves may fall due to reduced enzyme activity and substrate availability. Clinical studies also showed that endothelial dysfunction is related to oxidative stress burden and it worsens with increasing renal impairment, supporting oxidative injury as a mechanism for the cardio-renal continuum.[3-4]

A key question for clinical translation is whether oxidative imbalance shows a graded deterioration across KDIGO G-stages and whether the trend parallels conventional renal indices. Several stage-focused studies reported that oxidative stress increased progressively with advancing CKD. Dounousi et al. demonstrated that oxidative stress was progressively enhanced across CKD stages, indicating a true severity gradient rather than a binary "CKD vs non-CKD" phenomenon.[5] Similar observations were reported by Karamouzis et al., where oxidative stress rose with advancing CKD stage, reinforcing that redox injury accumulates as filtration declines.[6] Studies evaluating broader oxidative panels across CKD stages also reported increasing oxidative damage markers with declining renal function and altered antioxidant enzyme activity, strengthening the stage-wise hypothesis.[7]

Among available biomarkers, malondialdehyde (MDA) remains widely used as a lipid peroxidation readout, while superoxide dismutase (SOD) reflects a primary enzymatic antioxidant defense. Prior clinical work in chronic renal insufficiency and dialysis cohorts has shown elevated circulating MDA fractions in renal disease, consistent with increased lipid peroxidation load.[8] Antioxidant systems are also disturbed in CKD. Earlier mechanistic clinical studies highlighted derangements in glutathione pathways and protein oxidation products in chronic renal failure, showing that oxidative injury involves multiple molecular targets and is not limited to lipids alone.[9-10] Additional human studies assessing antioxidant enzyme activity in CKD reported reduced erythrocyte antioxidant defenses and worsening oxidant–antioxidant imbalance as renal dysfunction advanced.[11-12]

Despite this evidence, many published datasets either mix stages unevenly, focus on dialysis populations, or use heterogeneous oxidative panels that make stage-wise comparison difficult. Also, fewer studies have examined a simple dual-marker framework where oxidative burden (MDA) and antioxidant reserve (SOD) are evaluated together across CKD G2–G5 with parallel reporting of renal indices for biological validation. Therefore, this study was designed to evaluate stage-wise worsening of oxidative stress and antioxidant depletion across CKD G2–G5 by comparing MDA and SOD across stages, while also examining stage trends in serum creatinine, blood urea, eGFR and urine ACR to confirm internal biological consistency.

MATERIALS AND METHODS

This hospital based cross-sectional analytical study was conducted in the Department of Biochemistry, Index Medical College, Indore over 12 months. Adult patients (≥18 years) with established CKD were enrolled as cases and CKD staging was done as per KDIGO 2012 eGFR categories. Patients were classified into five groups G2, G3a, G3b, G4 and G5, with equal stage distribution (n=20 per stage). Age and sex matched healthy individuals were recruited separately as controls for the overall study, but the present paper focused on the stage-wise comparisons within CKD (G2–G5).

Inclusion criteria included adults with stable CKD (G2–G5) and willingness to provide written informed consent. Exclusion criteria included acute kidney injury or rapidly progressive renal failure, active systemic infection, autoimmune flare, malignancy, recent antioxidant supplementation (vitamin C/E) or corticosteroid exposure in the preceding 4 weeks, chronic liver disease, uncontrolled thyroid disorder and pregnancy.

Clinical and demographic variables were recorded using a structured proforma, including age, sex, BMI, systolic and diastolic blood pressure, diabetes status, hypertension status and medication history. Blood pressure was recorded using standard sphygmomanometry methods and BMI was calculated using height and weight measurements.

Renal function parameters were assessed using routine biochemistry methods. Serum creatinine was estimated using an enzymatic IDMS-traceable method on an automated analyser. Blood urea was measured by the urease–GLDH method. Estimated GFR was calculated using the CKD-EPI 2021 equation. Urine albumin–creatinine ratio (ACR) was measured from a spot urine sample using immunoturbidimetry for urine albumin and an enzymatic/Jaffe-based creatinine method.

Oxidative stress markers were evaluated using established biochemical assays. Plasma MDA was estimated by the thiobarbituric acid reactive substances (TBARS) spectrophotometric method. Erythrocyte SOD activity was estimated using the nitroblue tetrazolium (NBT) inhibition based method (riboflavin photoreduction system). All samples were collected after an overnight fast and assays were performed in duplicate. Hemolysed samples were excluded for oxidative assays and internal quality control procedures were followed for routine biochemistry.

Ethical approval was obtained from the Institutional Ethics Committee and written informed consent was obtained from all participants. Data confidentiality was maintained.

Statistical Analysis

Statistical analysis was carried out using SPSS version 26.0. Normality was assessed using Kolmogorov–Smirnov and Shapiro–Wilk tests. Since most biochemical variables showed non-normal distribution, stage-wise comparisons across CKD G2–G5 were analysed using the Kruskal–Wallis test. Post-hoc pairwise comparisons were planned using Dunn's test with Bonferroni correction for multiple testing, particularly for MDA and SOD. Categorical variables were compared across CKD stages using Chi-square test or Fisher's exact test as appropriate. A two-sided p value <0.05 was considered statistically significant.

RESULTS

Continuous data shown as median (IQR); categorical as n (%). P values: Kruskal–Wallis for continuous, Chi-square for categorical.

Table 1: Baseline comparability across CKD stages (G2–G5)

Variable	G2 (n=20)	G3a (n=20)	G3b (n=20)	G4 (n=20)	G5 (n=20)	p-value
Age (years)	42.5 (32.8–60.8)	54.5 (34.0–61.5)	54.5 (42.5–63.2)	51.5 (35.5–72.2)	50.5 (34.8–59.0)	0.748
BMI (kg/m²)	26.1 (23.3–27.7)	25.8 (22.4–26.9)	26.1 (24.2–27.7)	25.2 (23.5–28.0)	24.6 (23.0–27.6)	0.772
Sex: Male	14 (70.0%)	11 (55.0%)	11 (55.0%)	8 (40.0%)	10 (50.0%)	0.436
Sex: Female	6 (30.0%)	9 (45.0%)	9 (45.0%)	12 (60.0%)	10 (50.0%)	
Diabetes: Yes	5 (25.0%)	5 (25.0%)	7 (35.0%)	7 (35.0%)	9 (45.0%)	0.639
Diabetes: No	15 (75.0%)	15 (75.0%)	13 (65.0%)	13 (65.0%)	11 (55.0%)	
Hypertension: Yes	13 (65.0%)	14 (70.0%)	17 (85.0%)	14 (70.0%)	17 (85.0%)	0.443
Hypertension: No	7 (35.0%)	6 (30.0%)	3 (15.0%)	6 (30.0%)	3 (15.0%)	

Baseline variables were well balanced across CKD stages (G2–G5). Age and BMI showed no significant stage-wise difference (Kruskal–Wallis p>0.05). Sex distribution, diabetes and hypertension proportions were also comparable across stages (Chi-square p>0.05).

Values are median (IQR). Overall stage comparison by Kruskal–Wallis test.

Table 2: Stage-wise distribution of renal indices and oxidative markers (main table)

Variable	G2	G3a	G3b	G4	G5	Kruskal–Wallis H	p value
Serum creatinine (mg/dL)	1.51 (1.38–1.69)	2.21 (1.96–2.44)	3.03 (2.71–3.21)	5.04 (4.03–5.16)	6.82 (6.47–8.40)	92.114	<0.001
Blood urea (mg/dL)	41.70 (37.72–50.25)	69.25 (59.48–72.93)	88.35 (76.22–99.38)	139.55 (117.45–146.75)	171.00 (156.12–191.90)	90.261	<0.001
eGFR (mL/min/1.73 m²)	76.40 (72.05–83.73)	50.25 (48.25–54.88)	36.65 (35.58–40.62)	21.40 (20.02–26.23)	9.85 (7.28–12.62)	95.055	<0.001
Urine ACR (mg/g)	99.95 (54.33–117.88)	157.60 (111.33–214.57)	225.85 (199.65–306.15)	445.45 (256.25–576.15)	1066.15 (461.43–1201.55)	76.675	<0.001
MDA (nmol/mL)	4.23 (3.92–4.59)	5.58 (5.15–6.52)	6.99 (6.11–7.38)	8.47 (7.93–9.81)	11.12 (10.55–11.85)	88.980	<0.001
SOD (U/mL)	6.85 (6.29–7.62)	6.09 (5.18–6.56)	4.47 (4.19–4.97)	3.69 (3.44–3.99)	2.26 (2.05–2.78)	87.436	<0.001

A significant stage-wise gradient was observed for clinical and biochemical parameters. SBP and DBP increased with advancing CKD stage (p<0.05). Renal dysfunction markers showed strong monotonic worsening, with serum creatinine, blood urea and urine ACR rising and eGFR falling progressively from G2 to G5 (all p<0.001). Oxidative stress burden increased across stages with a significant rise in MDA, while antioxidant defence showed a significant decline in SOD from G2 to G5 (both p<0.001).

(A) MDA pairwise (Dunn–Bonferroni)

Table 3: Post-hoc pairwise comparisons

Comparison	Z	Adjusted p	Significant (Bonferroni)
G2 vs G3a	-2.213	0.269	No
G2 vs G3b	-3.927	<0.001	Yes
G2 vs G4	-6.434	<0.001	Yes
G2 vs G5	-8.423	<0.001	Yes
G3a vs G3b	-1.714	0.865	No
G3a vs G4	-4.221	<0.001	Yes
G3a vs G5	-6.210	<0.001	Yes
G3b vs G4	-2.507	0.122	No
G3b vs G5	-4.496	<0.001	Yes
G4 vs G5	-1.989	0.467	No

Pairwise post-hoc testing showed that MDA differed mainly between early and more advanced CKD stages. Significant increases were seen for G2 vs G3b/G4/G5, G3a vs G4/G5 and G3b vs G5 (adjusted p<0.001). Comparisons between adjacent stages (G2 vs G3a, G3a vs G3b, G3b vs G4 and G4 vs G5) were not significant after Bonferroni correction, indicating that the rise in MDA became clearly distinguishable only beyond early stage transitions.

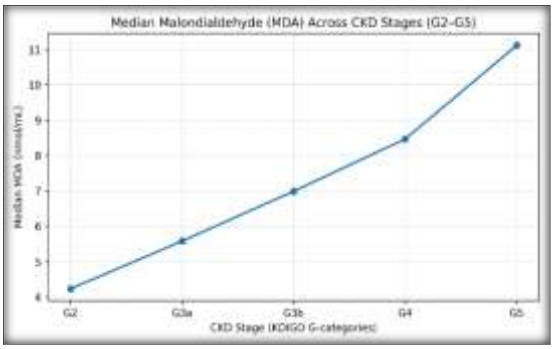

Figure 1

(B) SOD pairwise (Dunn–Bonferroni)

Table 4: ?

Comparison	Z	Adjusted p	Significant (Bonferroni)
G2 vs G3a	1.469	1.000	No
G2 vs G3b	4.030	<0.001	Yes
G2 vs G4	5.894	<0.001	Yes
G2 vs G5	8.213	<0.001	Yes
G3a vs G3b	2.562	0.104	No
G3a vs G4	4.426	<0.001	Yes
G3a vs G5	6.745	<0.001	Yes
G3b vs G4	1.864	0.623	No
G3b vs G5	4.183	<0.001	Yes
G4 vs G5	2.319	0.204	No

Post-hoc analysis confirmed significant antioxidant depletion across more widely separated stages. SOD was significantly lower in G3b/G4/G5 compared with G2 and significantly lower in G4/G5 compared with G3a (all adjusted p<0.001). SOD was also significantly lower in G5 compared with G3b (adjusted p<0.001). Adjacent-stage differences (G2 vs G3a, G3a vs G3b, G3b vs G4 and G4 vs G5) were not significant after correction, suggesting that the decline in SOD was most evident when comparing early CKD with advanced CKD.

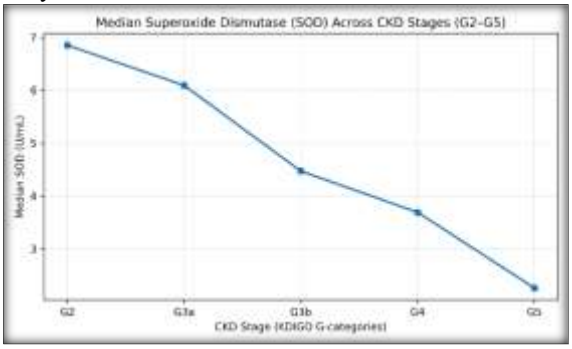

Figure 2

DISCUSSION

This study was undertaken to determine whether oxidative imbalance demonstrates a true stage-wise gradient across pre-dialysis CKD. Adult CKD patients from a tertiary care setting in Indore were classified into KDIGO G2–G5 with equal distribution per stage and lipid peroxidation burden (MDA) and antioxidant defence (SOD) were compared across stages. Parallel assessment of serum creatinine, blood urea, eGFR and urine ACR was included to verify internal biological consistency of CKD staging.

Table 1 showed that CKD stage groups were broadly comparable at baseline. Age and BMI did not differ significantly across G2–G5 and sex distribution, diabetes prevalence and hypertension prevalence were also statistically comparable. This baseline balance reduced the likelihood that the observed stage-wise differences in oxidative markers were primarily driven by demographic differences rather than CKD severity. Equal sampling across stages also limited common biases from uneven stage

representation, which has affected interpretability in several earlier stage-based CKD datasets.

Table 2 demonstrated strong internal validation of stage classification. Renal dysfunction markers worsened monotonically from G2 to G5, with serum creatinine and blood urea increasing progressively, eGFR declining stepwise and urine ACR rising markedly, all with strong overall significance. This pattern aligned with established CKD stage biology and supported that the cohort reflected a true severity spectrum rather than misclassified or acute renal dysfunction. Large population studies have shown that lower eGFR is independently associated with higher mortality and cardiovascular event rates, confirming that CKD staging is prognostic and clinically meaningful.[13]

In the same table, oxidative stress and antioxidant defence showed an opposite directional stage gradient. MDA rose steadily from G2 through G5 while SOD declined progressively, both with strong Kruskal–Wallis significance. Similar graded increases in oxidative stress across CKD stages have been reported. Dounousi et al. demonstrated that oxidative stress was progressively enhanced with advancing CKD stages, supporting a dose–response relationship rather than a binary "CKD vs non-CKD" phenomenon.[5] Karamouzis et al. reported increasing oxidative stress with advancing CKD stage, reinforcing that oxidant burden accumulates as renal function declines.[6] Kuchta et al. also described stage-linked differences in oxidative stress markers in CKD, supporting a progressive redox disturbance across severity categories.[7] Oberg et al. described a combined oxidant stress and inflammatory phenotype in moderate to severe CKD, supporting that redox imbalance is present before dialysis and tends to intensify with worsening renal dysfunction.[2]

The stage-wise rise in MDA is biologically plausible because declining filtration increases uremic toxin load and is accompanied by sustained inflammation, RAAS activation, endothelial activation and mitochondrial dysfunction. These drivers promote reactive oxygen species formation and lipid peroxidation, so MDA increases as a cumulative marker of membrane lipid injury. De Vecchi et al. quantified free and total plasma MDA in chronic renal insufficiency and dialysis patients and reported higher MDA fractions in renal disease, supporting lipid peroxidation as a consistent biochemical footprint in CKD.[8] The marked rise in urine ACR across stages also supports a renal mechanism for escalating oxidative injury, as proteinuria increases tubular stress and inflammatory signalling which can amplify oxidative pathways and systemic redox imbalance.

The stage-wise fall in SOD in Table 2 is consistent with progressive depletion or dysfunction of antioxidant systems in CKD. SOD detoxifies superoxide and reduced activity permits longer persistence of superoxide with downstream formation of reactive intermediates and nitric oxide quenching, contributing to endothelial dysfunction and vascular remodelling. Endothelial studies in CKD have linked oxidative stress burden with impaired endothelial function and have shown worsening vascular dysfunction with increasing renal impairment, supporting this mechanistic bridge between CKD progression and cardiovascular risk.[14,15] Broader antioxidant pathway disruption has also been described in chronic renal failure. Ceballos-Picot et al. reported glutathione system disturbances in chronic renal failure, indicating that antioxidant impairment in CKD involves multiple pathways beyond lipid peroxidation alone.[9] Stepniewska et al. reported impairment in erythrocyte antioxidant defence systems in chronic renal failure, supporting the plausibility of reduced erythrocyte-based antioxidant enzyme activity with advancing disease.[11] Oxidative injury involving protein oxidation is also relevant. Witko-Sarsat et al. described advanced oxidation protein products as biologically active mediators elevated in chronic renal failure, supporting that oxidative stress in CKD is multi-target and linked with inflammation and immune activation.[10] Human studies from diverse settings have also reported altered oxidative markers in CKD cohorts, supporting the generalisability of oxidant–antioxidant imbalance in renal dysfunction.[12]

Table 3A and 3B clarified that the most robust pairwise differences for MDA and SOD occurred between early and more advanced CKD stages, while several adjacent-stage comparisons did not remain significant after Bonferroni correction. For MDA, significant differences were mainly observed for comparisons involving wider stage separation, particularly G2 versus G3b/G4/G5 and G3a versus G4/G5, while adjacent comparisons such as G2 versus G3a or G4 versus G5 were not significant after correction. For SOD, significant depletion was again most evident when comparing G2 with G3b/G4/G5 and comparing G3a with G4/G5, with additional significance for G3b versus G5. This pattern is expected when changes occur gradually across stages and when correction for multiple testing is conservative, particularly with modest per-stage sample size. The post-hoc findings therefore supported a clinically useful interpretation: oxidative burden and antioxidant depletion were most clearly distinguishable when comparing early CKD with advanced CKD rather than between every adjacent KDIGO category. Similar stage-based studies have reported progressive trends across CKD, though pairwise separations between neighbouring stages can be inconsistent depending on sample size, marker variability and multiplicity handling.[16-18]

A few limitations should be acknowledged. This was a single-centre hospital-based study so findings may not generalise to all CKD populations. The cross-sectional design captured association with stage but could not prove temporal change or causality. Oxidative markers were assessed using TBARS-based MDA and an erythrocyte SOD activity assay,

both of which can be influenced by pre-analytical factors and inter-assay variability.

CONCLUSION

In conclusion, this study showed a clear stage-wise oxidative stress gradient across CKD G2–G5. Lipid peroxidation burden increased progressively as renal function worsened, while antioxidant defence declined in parallel. The same monotonic trends in creatinine, urea, eGFR and albuminuria supported biological validity of staging and strengthened the interpretation that oxidative imbalance tracks CKD severity. These findings support using a simple dual-marker framework (MDA for oxidative burden and SOD for antioxidant reserve) to describe redox status across CKD stages and to guide future work on risk stratification and monitoring, ideally through larger multicentre longitudinal studies with adjustment for inflammation and treatment-related confounders.

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