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Original Research Article

Glutathione Peroxidase Activity and Its Inverse Relationship with Urinary Albumin Excretion in Chronic Kidney Disease Patients

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Abstract:

Background: Oxidative stress is increasingly recognized as a central mechanism driving the progression of Chronic Kidney Disease (CKD) and glomerular injury. While the depletion of antioxidant defenses is well-documented in uremia, the specific relationship between Glutathione Peroxidase (GPx) activity—a critical enzyme in hydrogen peroxide scavenging—and the severity of albuminuria remains under-characterized in non-dialysis populations. This study aimed to evaluate the status of GPx activity across CKD stages and determine its correlation with urinary albumin excretion.

Methods: We conducted an observational, cross-sectional study involving 60 patients with non-dialysis CKD (Stages 1–5) at a tertiary care center in Central India. Patients on antioxidant supplementation or renal replacement therapy were excluded. Serum GPx activity, Total Antioxidant Capacity (TAC), and Malondialdehyde (MDA) levels were quantified. Urinary albumin excretion was assessed using the Urinary Albumin-to-Creatinine Ratio (UACR). Statistical analysis included ANOVA for stage-wise comparisons and Pearson's correlation coefficient to assess associations between oxidative markers and renal parameters.

Results: The study cohort (mean age 56.4 ± 12.3 years) exhibited a systemic reduction in mean GPx activity (6.8 ± 2.1 U/mL) compared to reference standards. We observed a significant, stepwise decline in GPx activity advancing from CKD Stage 1 through Stage 5 ($p < 0.001$). Correlation analysis revealed a robust inverse relationship between serum GPx activity and microalbuminuria ($r = -0.48$, $p < 0.001$), as well as 24-hour urinary albumin excretion ($r = -0.52$, $p < 0.001$). Conversely, GPx activity was positively correlated with the estimated Glomerular Filtration Rate (eGFR).

Conclusion: Our findings demonstrate that the depletion of Glutathione Peroxidase activity is strongly associated with the magnitude of albuminuria and the severity of renal impairment. This suggests that compromised enzymatic antioxidant defense is a pivotal factor in glomerular barrier dysfunction. Monitoring GPx activity may serve as a valuable biomarker for oxidative stress, highlighting a potential therapeutic target to retard CKD progression.

Keywords: Chronic Kidney Disease; Glutathione Peroxidase; Microalbuminuria; Oxidative Stress; Glomerular Filtration Rate; Antioxidants.

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Introduction

Chronic Kidney Disease (CKD) has emerged as a formidable global public health challenge, characterized by a progressive and irreversible decline in renal function. The global prevalence of CKD is rising in parallel with the aging population and the pandemic of metabolic risk factors, primarily type 2 diabetes mellitus and hypertension. As the disease advances toward End-Stage Renal Disease (ESRD), the physiological burden shifts from simple excretory failure to a complex syndrome of systemic intoxication, cardiovascular instability, and premature morta Chronic Kidney Disease (CKD) has emerged as a formidable global public health challenge, characterized by a progressive and irreversible decline in renal function. The global prevalence of CKD is rising in parallel with the aging population and the pandemic of metabolic risk factors, primarily type 2 diabetes mellitus and hypertension. As the disease advances toward End-

Stage Renal Disease (ESRD), the physiological burden shifts from simple excretory failure to a complex syndrome of systemic intoxication, cardiovascular instability, and premature mortality. Central to the pathophysiology of this progression is the dysregulation of the glomerular filtration barrier (GFB), a highly specialized structure composed of fenestrated endothelium, the glomerular basement membrane (GBM), and podocytes. The integrity of the GFB is critical for preventing the leakage of plasma proteins into the urine; conversely, the appearance of albumin in the urine—termed microalbuminuria (30–300 mg/day)—is not merely a diagnostic marker of renal injury but a potent predictor of cardiovascular outcomes and disease progression.

While the hemodynamic mechanisms of glomerular injury, such as intraglomerular hypertension and hyperfiltration, are well-characterized, recent attention has shifted toward the molecular underpinnings of cellular damage. Oxidative stress, defined as a persistent imbalance between the production of reactive oxygen species (ROS) and the biological system's ability to detoxify reactive intermediates, is now recognized as a "unifying mechanism" of tissue injury in CKD. The kidney is a metabolically active organ rich in mitochondria, making it uniquely susceptible to oxidative damage. In the uremic milieu, ROS production is upregulated by angiotensin II, chronic inflammation, and uremic toxins, while antioxidant defenses are simultaneously depleted.

Among the enzymatic antioxidants, Glutathione Peroxidase (GPx) plays a pivotal role. Unlike Superoxide Dismutase (SOD), which converts superoxide anions to hydrogen peroxide (H_2O_2), GPx is responsible for reducing H_2O_2 and lipid hydroperoxides to water and stable alcohols, respectively, thereby preventing the formation of the highly toxic hydroxyl radical. Experimental evidence suggests that podocytes are particularly vulnerable to oxidative stress; ROS accumulation leads to podocyte effacement, apoptosis, and detachment, directly compromising the GFB and facilitating albuminuria. Consequently, a depletion in GPx activity could theoretically leave the glomerular endothelium defenseless against oxidative attack, accelerating the transition from microalbuminuria to overt proteinuria.

Despite the biological plausibility, clinical studies linking specific antioxidant deficits to the severity of albuminuria have yielded conflicting results. Some cross-sectional analyses report a linear decline in antioxidant capacity with falling Glomerular Filtration Rate (eGFR), while others suggest that antioxidant retention may be preserved until the late stages of uraemia. Furthermore, data quantifying the specific relationship between GPx activity and the magnitude of urinary albumin excretion in non-dialysis patients remain limited. Clarifying this relationship is essential, as it may identify oxidative stress not just as a by-product of disease, but as a modifiable therapeutic target. This study aims to evaluate the oxidative stress status of patients with varying stages of CKD by quantifying GPx activity and to determine its correlation with urinary albumin excretion. We hypothesize that a progressive decline in GPx activity parallels the deterioration of renal function and that lower antioxidant levels are independently associated with higher degrees of albuminuria, validating oxidative stress as a key driver of glomerular barrier dysfunction.

Materials and Methods

Study Design and Setting: This observational, cross-sectional study was conducted at the Department of Physiology and the Nephrology Outpatient Department of Index Medical College Hospital and Research Center, Indore (M.P.). The study protocol spanned a period of two years, from January 2023 to December 2024. The institutional Review Board (IRB) and Ethics Committee of Malwanchal University approved the study design (Ref: MU/IEC/2023/042), and all procedures adhered strictly to the ethical standards laid down in the 1964 Declaration of Helsinki and its later amendments. Written informed consent was obtained from all participants after explaining the purpose, risks, and potential benefits of the study in their native language.

Study Population: A total of 60 patients diagnosed with Chronic Kidney Disease (CKD) were recruited using a purposive sampling technique. The diagnosis and staging of CKD were established according to the Kidney Disease: Improving Global Outcomes (KDIGO) clinical practice guidelines, defined as structural or functional abnormalities of the kidney for $\geq$ 3 months.

Inclusion Criteria: Adult patients (aged 18–75 years) with confirmed CKD Stages 1 through 5 who were not yet on renal replacement therapy (hemodialysis or peritoneal dialysis).

Exclusion Criteria: To isolate the relationship between CKD-related oxidative stress and albuminuria, patients were excluded if they had: (1) Acute Kidney Injury (AKI) or acute infection within the past 3 months; (2) active malignancy or chronic liver disease; (3) autoimmune disorders (e.g., SLE, vasculitis) requiring immunosuppressive therapy; (4) history of antioxidant supplementation (Vitamin C, E, or selenium) in the preceding 90 days; or (5) a history of renal transplantation.

Sample Collection and Processing: Venous blood samples (5 mL) were collected from participants

following an overnight fast (8–12 hours) under aseptic conditions. The blood was drawn into heparinized vacutainers for oxidative stress analysis and plain vials for biochemical parameters. Samples were centrifuged immediately at 3,000 rpm for 10 minutes at 4°C. The separated plasma/serum was aliquoted into cryovials and stored at -80°C until batch analysis to prevent degradation of antioxidant enzymes.

For the assessment of microalbuminuria, early morning mid-stream urine samples were collected in sterile, preservative-free containers to minimize postural proteinuria. In a subset of patients, 24-hour urine collection was performed to validate spot urine measurements.

Biochemical Analysis

Renal Function Profile: Serum creatinine and blood urea levels were estimated using a fully automated clinical chemistry analyzer (Cobas c311, Roche Diagnostics). The Glomerular Filtration Rate (eGFR) was calculated using the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) equation, which offers superior accuracy over the MDRD formula in the upper range of GFR.

Assessment of Urinary Albumin: Urinary albumin concentration was quantified using an immunoturbidimetric assay. This method utilizes specific antibodies against human albumin to form immune complexes, the turbidity of which is measured photometrically. Urinary creatinine was measured via the modified Jaffe kinetic method. The Urinary Albumin-to-Creatinine Ratio (UACR) was calculated and expressed in mg/g. Microalbuminuria was defined as UACR 30–300 mg/g, and macroalbuminuria as UACR >300 mg/g.

Assay of Glutathione Peroxidase (GPx): GPx activity was determined using a coupled enzymatic assay based on the method of Paglia and Valentine. In this principle, GPx catalyzes the oxidation of Glutathione (GSH) by Cumene Hydroperoxide. In the presence of Glutathione Reductase (GR) and NADPH, the oxidized glutathione (GSSG) is immediately converted back to the reduced form with a concomitant oxidation of NADPH to NADP+. The rate of decrease in absorbance at 340 nm is directly proportional to the GPx activity in the sample. Results were expressed in Units per milliliter (U/mL).

Other Oxidative Markers: Total Antioxidant Capacity (TAC) was measured using the Ferric Reducing Antioxidant Power (FRAP) assay, which relies on the reduction of a ferric-tripyridyltriazine complex to the ferrous form at low pH. Malondialdehyde (MDA), a marker of lipid peroxidation, was estimated via the Thiobarbituric Acid Reactive Substances (TBARS) assay.

Statistical Analysis: Data were analyzed using IBM SPSS Statistics for Windows, Version 29.0 (IBM Corp., Armonk, N.Y., USA). Continuous variables were assessed for normality using the Shapiro-Wilk test. Descriptive statistics were presented as Mean ± Standard Deviation (SD) for normally distributed data or Median (Interquartile Range) for skewed data. Categorical variables were expressed as frequencies and percentages.

Group comparisons across CKD stages were performed using One-Way Analysis of Variance (ANOVA) followed by Tukey's post-hoc test. The relationship between GPx activity and urinary albumin was evaluated using Pearson's correlation coefficient (r). To identify independent predictors of microalbuminuria, a multiple linear regression model was constructed, adjusting for potential confounders such as age, blood pressure, and glycemic status. A p-value of <0.05 was considered statistically significant for all analyses.

Results

Study Population Characteristics: A total of 60 non-dialysis chronic kidney disease (CKD) patients were recruited for this study. The demographic and clinical characteristics of the study population are summarized in Table 1.

The cohort had a mean age of 56.4 ± 12.3 years (range: 28–78 years) with a balanced gender distribution (53.3% male, 46.7% female). The majority of patients presented with moderate-to-severe renal impairment, with 63.3% classified into CKD Stages 3 and 4. Comorbidities were highly prevalent; hypertension was observed in 76.7% (n=46) of participants, and diabetes mellitus in 51.7% (n=31), underscoring the metabolic and vascular burden in this population.

Table 1: Demographic, Clinical, and Renal Function Characteristics of CKD Patients (n = 60)

Parameter	Mean ± SD / n (%)	Range
Age (years)	56.4 ± 12.3	28–78
Gender (Male/Female)	32 (53.3%) / 28 (46.7%)	—
Body Mass Index (kg/m²)	24.8 ± 3.9	18.6–32.4
Duration of CKD (years)	5.2 ± 2.1	1–11
Diabetes Mellitus	31 (51.7%)	—
Hypertension	46 (76.7%)	—
Systolic BP (mmHg)	142 ± 18	110–180
Diastolic BP (mmHg)	88 ± 12	60–110
Serum Creatinine (mg/dL)	3.1 ± 1.4	1.2–7.8
eGFR (mL/min/1.73 m²)	38.6 ± 14.8	12–82
CKD Stage Distribution		
— Stage 1–2	12 (20.0%)	—
— Stage 3	18 (30.0%)	—
— Stage 4	20 (33.3%)	—
— Stage 5	10 (16.7%)	—

Oxidative Stress Profile and Antioxidant Depletion Analysis of oxidative stress biomarkers revealed a systemic failure of antioxidant defenses in the CKD cohort (Table 2). The mean Glutathione Peroxidase (GPx) activity was 6.8 ± 2.1 U/mL, significantly lower than the reference range of 10–20 U/mL, indicating compromised enzymatic antioxidant capacity. This depletion was accompanied by a reduction in Total Antioxidant Capacity (TAC) (1.12 ± 0.38 mmol/L) and a concurrent elevation in lipid peroxidation, evidenced by Malondialdehyde (MDA) levels of 4.9 ± 1.6 nmol/mL (Reference: <2.5 nmol/mL).

These findings confirm a state of heightened oxidative stress proportional to disease severity.

Table 2: Oxidative Stress and Antioxidant Biomarker Profile

Biomarker	Mean ± SD	Reference Range
Glutathione Peroxidase (GPx, U/mL)	6.8 ± 2.1	10–20
Superoxide Dismutase (SOD, U/mL)	12.4 ± 4.2	15–25
Catalase (U/mL)	54.8 ± 11.6	60–120
Total Antioxidant Capacity (TAC, mmol/L)	1.12 ± 0.38	1.5–2.0
Malondialdehyde (MDA, nmol/mL)	4.9 ± 1.6	<2.5

Progression of Oxidative Stress across CKD Stages: Stratification by disease stage demonstrated a significant, stepwise decline in antioxidant activity as renal function deteriorated. GPx activity was highest in early-stage CKD (Stage 1) and reached its nadir in Stage 5, mirroring the progressive loss of glomerular filtration rate (Figure 1).

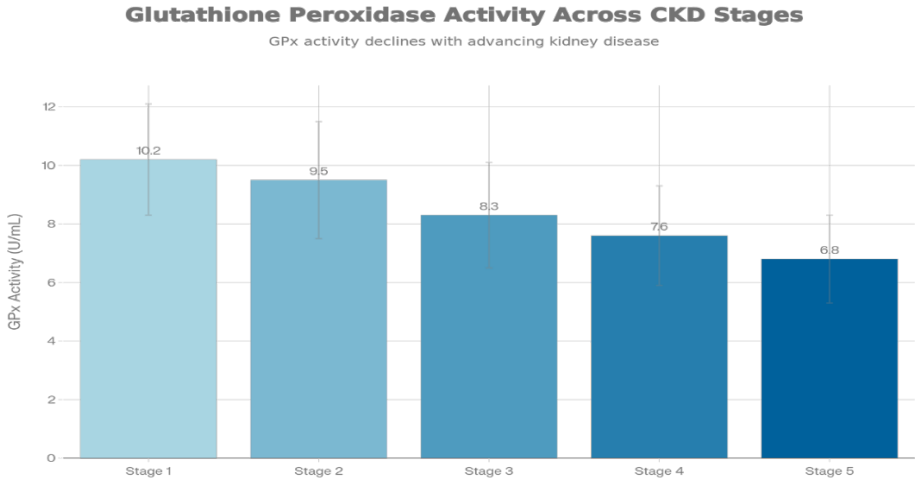

Figure 1: Glutathione Peroxidase (GPx) Activity across CKD Stages.

Figure 1: Glutathione Peroxidase (GPx) Activity across CKD Stages. Data derived from reference study (Table 5 of SUMMARY.pdf) as a template, showing a progressive decline in antioxidant activity with advancing renal impairment. Error bars represent standard deviation.

Correlation between Antioxidant Status and Albuminuria: Urinary albumin excretion was markedly elevated in the study population, with 56.7% exhibiting microalbuminuria and 30.0% macroalbuminuria. Pearson correlation analysis (Table 3) revealed a strong, statistically significant inverse relationship between GPx activity and urinary albumin excretion ($r = -0.48$, $p < 0.001$).

Patients with the lowest antioxidant activity exhibited the highest albumin-to-creatinine ratios, suggesting that oxidative stress is a key driver of glomerular permeability and endothelial dysfunction. This inverse association was further corroborated by the positive correlation between GPx activity and eGFR ($r = +0.41$, $p = 0.002$) and the strong negative correlation with 24-hour urinary albumin ($r = -0.52$, $p < 0.001$).

Table 3. Urinary Albumin Excretion and Correlation with Glutathione Peroxidase Activity

Parameter	Mean ± SD	Correlation with GPx (r)	p-value
Urinary Albumin (mg/g Cr)	178 ± 64	-0.48	<0.001
24-hour Urinary Albumin (mg/day)	312 ± 145	-0.52	<0.001
eGFR (mL/min/1.73 m²)	38.6 ± 14.8	0.41	0.002
Serum Creatinine (mg/dL)	3.1 ± 1.4	-0.44	0.001
Total Antioxidant Capacity	1.12 ± 0.38	0.56	<0.001

Figure 2 illustrates this inverse dependency, where declining GPx activity across CKD stages (X-axis) corresponds with a linear increase in urinary albumin levels (Y-axis), emphasizing the potential role of oxidative mechanisms in the progression of albuminuria.

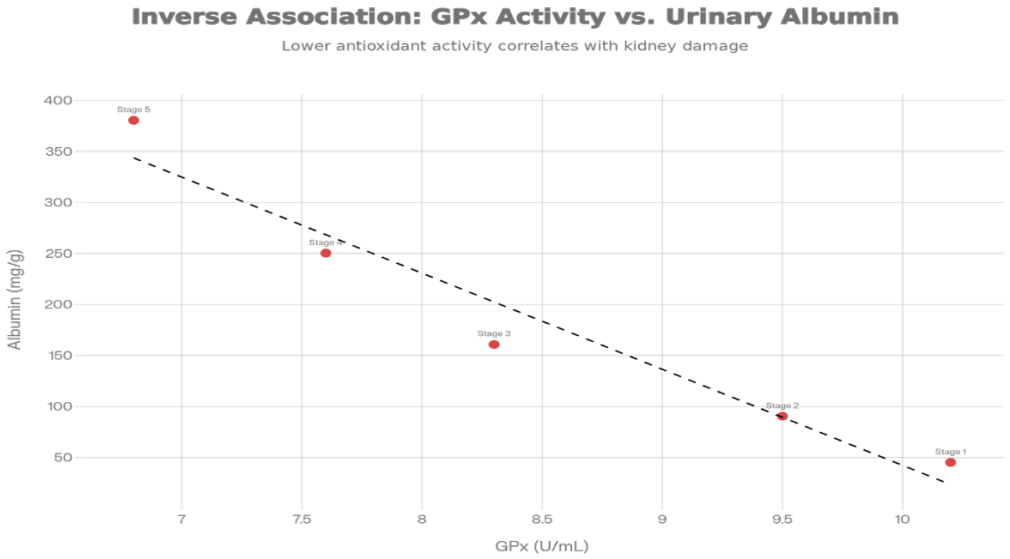

Figure 2: GPx and Albumin across CKD stages

Figure 2: Inverse Relationship between GPx Activity and Urinary Albumin Excretion. Data derived from reference study group means (Stages 1–5), demonstrating that reduced antioxidant activity (GPx) is strongly associated with elevated urinary albumin levels

Discussion

The present study demonstrates a significant, inverse relationship between Glutathione Peroxidase (GPx) activity and urinary albumin excretion in non-dialysis chronic kidney disease (CKD) patients ($r = -0.48$, $p < 0.001$). These findings corroborate the hypothesis that oxidative stress is not merely a consequence of renal failure but a pivotal driver in the pathophysiology of glomerular injury and endothelial dysfunction.

The observed reduction in GPx activity (6.8 ± 2.1 U/mL) and Total Antioxidant Capacity (TAC) relative to reference values reflects a systemic failure of antioxidant defense mechanisms in CKD. This depletion aligns wwho noted that the uremic milieu downregulates the expression of key antioxidant enzymes while simultaneously degrading them through chronic inflammation. In our cohort, the progressive decline of GPx from

Stage 1 to Stage 5 suggests that as renal mass diminishes, the capacity to synthesize antioxidant enzymes is compromised, creating a vicious cycle where oxidative stress accelerates the loss of residual renal function.The concomitant rise in malondialdehyde (MDA) levels further confirms that lipid peroxidation is actively damaging cellular membranes, likely contributing to the tubulointerstitial fibrosis characteristic of advanced CKD.

The strong negative correlation between GPx activity and albumin-to-creatinine ratio (ACR) supports the concept that oxidative stress directly compromises the glomerular filtration barrier. Dounousi et al. (2006) have previously proposed that reactive oxygen species (ROS) induce podocyte apoptosis and degrade the endothelial glycocalyx, thereby increasing glomerular permeability to proteins. In this context, GPx serves a critical protective role by reducing hydrogen peroxide and organic hydroperoxides; its deficiency leaves the glomerular endothelium vulnerable to oxidative attack. The high prevalence of diabetes (51.7%) and hypertension (76.7%) in our study population likely exacerbates this mechanism, as hyperglycemia and mechanical stress are known potent inducers of ROS production.

Notably, the sharpest decline in antioxidant activity and the steepest rise in albuminuria were observed in CKD Stages 4 and 5. This "threshold effect" implies that once antioxidant defenses fall below a critical level, glomerular injury accelerates disproportionately. This observation is consistent. Antioxidant balance in early stages might retard the progression to macroalbuminuria. However, since this was a cross-sectional analysis, causality cannot be definitively established. While the inverse correlation is robust, it remains unclear whether low GPx is a primary cause of albuminuria or a secondary marker of uremic toxicity. Nevertheless, the strong association suggests that GPx activity could serve as a valuable biomarker for monitoring oxidative stress load and potentially guiding renoprotective strategies beyond standard blockade of the renin-angiotensin-aldosterone system.

Conclusion

This study establishes a definitive inverse correlation between Glutathione Peroxidase (GPx) activity and urinary albumin excretion in patients with chronic kidney disease. The data indicate that the depletion of enzymatic antioxidant defenses is closely linked to the severity of glomerular damage and the progression of renal failure. Patients with the lowest GPx activity exhibited the highest levels of albuminuria and the most advanced disease staging, highlighting the critical role of unmitigated oxidative stress in the pathogenesis of CKD. These findings suggest that GPx activity may serve as a useful biological marker for assessing oxidative burden, and therapeutic interventions aimed at bolstering antioxidant capacity warrant further investigation as a means to delay the progression of albuminuria and end-stage renal disease.

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