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Dynamic Thiol/Disulfide Homeostasis as a Complementary Oxidative Stress Marker Across Albuminuria Stages in Type 2 Diabetes Mellitus

Atila ALTUNTAŞ[1], Bünyamin AYDIN[2], Sıddıka ERSOY[3], Özcan EREL[4], Hasret DEMİREL[5], Şeniz TUTUM[1]

[1] Suleyman Demirel University, Faculty of Medicine, Department of Internal Medicine, Division of Nephrology, Isparta, Türkiye
[2] Kütahya Evliya Çelebi Training and Research Hospital, Department of Internal Medicine, Division of Endocrinology and Metabolism, Kütahya, Türkiye
[3] Suleyman Demirel University, Faculty of Health Science, Department of Nursing, Isparta, Türkiye
[4] Yıldırım Beyazıt University, Faculty of Medicine, Atatürk Training and Research Hospital, Department of Biochemistry, Ankara, Türkiye
[5] Suleyman Demirel University, Faculty of Medicine, Department of Internal Medicine, Division of Geriatrics, Isparta, Türkiye

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Abstract

Objective
Diabetic nephropathy (DN) remains one of the most serious complications of type 2 diabetes mellitus (T2DM). Traditionally, disease progression has been evaluated through albuminuria, yet increasing evidence highlights the role of oxidative stress and redox imbalance in DN pathogenesis. The present study was designed to evaluate dynamic thiol/disulfide homeostasis across albuminuria stages and to explore its potential role as a complementary systemic oxidative stress marker alongside albuminuria in T2DM.

Material and Method
A total of 122 individuals were included and categorized into four study groups: 30 healthy participants (Group 1), 31 normoalbuminuric diabetic patients (Group 2), 31 patients with microalbuminuria (Group 3), and 30 patients with macroalbuminuria (Group 4). Plasma thiol/disulfide parameters were measured using the automated method developed by Erel and Neselioglu, while urinary albumin concentrations were determined by immunoturbidimetric assay.

Results
Progressive decreases in both native and total thiol levels were observed with advancing severity of albuminuria ($p<0.001$). Disulfide concentrations showed a downward trend in macroalbuminuric patients ($p=0.019$), although disulfide-related ratios remained largely unchanged across the groups. Correlation analyses identified significant negative associations between thiol concentrations and diabetes duration, HbA1c, as well as CRP levels ($p<0.001$).

Conclusion
Dynamic thiol/disulfide balance reflects systemic oxidative stress but cannot fully substitute for albuminuria as a marker of DN. Instead, it appears to provide complementary insight into oxidative mechanisms associated with DN severity in T2DM patients.

Keywords: Albuminuria, Diabetes mellitus, Diabetic nephropathy, Oxidative stress, Thiol/disulfide homeostasis

Correspondence: H.D. / hasretkacemer@gmail.com
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ORCID IDs of the Authors: A.A: 0000-0001-9196-3133; B.A: 0000-0003-2059-1074; S.E: 0000-0001-8094-8042; Ö.E: 0000-0002-2996-3236; H.D: 0000-0002-5548-3797; Ş.T: 0009-0000-5989-7340

Introduction

Diabetic nephropathy (DN) is among the most frequent and burdensome microvascular complications of type 2 diabetes mellitus (T2DM), substantially contributing to global rates of end-stage renal disease (1, 2). While albuminuria has long been used as the standard marker of DN, reflecting glomerular injury and endothelial dysfunction, its reliability is limited by external factors such as hydration, physical activity, and concurrent inflammatory processes (3, 4). These shortcomings highlight the need for additional biomarkers that more accurately capture underlying pathophysiological mechanisms and improve risk stratification.

Oxidative stress, a well-recognized hallmark of diabetes and its complications, arises from an imbalance between excessive production of reactive oxygen species (ROS) and insufficient antioxidant defenses (5, 6). Thiols, integral components of the antioxidant system, neutralize ROS and undergo oxidation to form disulfide bonds. The dynamic equilibrium between thiols and disulfides is critical for redox homeostasis, regulating processes such as apoptosis, detoxification, and signal transduction (7, 8). Disturbances in thiol/disulfide homeostasis have been linked to the development of various chronic conditions, including cardiovascular and kidney diseases as well as T2DM (9-12).

Prior investigations have reported associations between thiol/disulfide disequilibrium and metabolic as well as inflammatory abnormalities in diabetes (13, 14). Furthermore, thiol/disulfide homeostasis has been proposed as a novel oxidative stress marker, particularly in conditions characterized by increased metabolic demand (13). Nevertheless, its diagnostic and prognostic value in DN remains to be fully clarified. Although thiol/disulfide homeostasis has been investigated in diabetes, data comparing these redox parameters across albuminuria-defined nephropathy stages remain limited. A stage-based evaluation may clarify whether thiol depletion shows a graded relationship with nephropathy severity and whether it parallels metabolic and inflammatory burden. Therefore, we investigated thiol/disulfide parameters in T2DM patients stratified into normo-, micro-, and macroalbuminuria groups.

The present study aimed to examine dynamic thiol/disulfide homeostasis in T2DM patients across different albuminuria categories. By exploring correlations with metabolic and inflammatory markers, we sought to determine whether these redox parameters could function as complementary indicators alongside albuminuria in evaluating DN-related risk in T2DM.

Material and Method

Study Population

The study included 122 individuals, divided into four groups: 30 healthy controls (Group 1), 31 T2DM patients with normoalbuminuria (Group 2), 31 T2DM patients with microalbuminuria (Group 3), and 30 T2DM patients with macroalbuminuria (Group 4).

Inclusion Criteria:

- Age ≥18 years.
- Confirmed diagnosis of T2DM (for patient groups).
- No systemic, infectious, or inflammatory conditions (for control group).
- No use of corticosteroids or immunosuppressive therapies within the last month.
- Non-smokers and non-alcohol consumers.

Exclusion Criteria:

Participants presenting with active infections, malignancies, or additional chronic conditions were excluded to ensure sample homogeneity.

Sample Collection and Laboratory Analysis

Venous blood samples (3 mL) were obtained after overnight fasting into EDTA-containing tubes. Plasma was isolated by centrifugation at 3000 rpm for 10 minutes and stored at -80°C until analysis.

Dynamic thiol/disulfide homeostasis was measured using the fully automated method developed by Erel and Neselioglu (15). This technique quantifies native thiol (N-thiol), total thiol (T-thiol), and disulfide levels, and calculates disulfide/native thiol and disulfide/total thiol ratios as indicators of redox balance.

Urinary albumin excretion was measured from first-morning urine samples using an immunoturbidimetric assay (Beckman Coulter System). Participants were classified based on urinary albumin levels as follows:

- Normoalbuminuria: <30 mg/day.
- Microalbuminuria: 30–300 mg/day.
- Macroalbuminuria: >300 mg/day (2).

Estimated glomerular filtration rate (eGFR) was not available for all participants in a standardized manner and was therefore not included in the analysis.

Statistical Analysis

All statistical analyses were performed using IBM SPSS Statistics for Windows, version 22.0 (SPSS Inc., Chicago, IL, USA). Data distribution was evaluated using the Kolmogorov-Smirnov test. For normally distributed data, one-way analysis of variance (ANOVA) was applied, followed by Bonferroni post-hoc comparisons. Non-normally distributed data were examined with the Kruskal-Wallis test. Categorical data were analyzed using the chi-square (χ^2) test. Correlations were evaluated with Pearson's test for parametric variables and Spearman's test for non-parametric variables. A p-value <0.05 was accepted as the threshold for statistical significance.

Results

A total of 122 participants were included in the study. The control and diabetic groups were comparable in terms of age and sex distribution, with no statistically significant differences observed (p>0.05). The mean age of the study population was 52.83 ± 9.54 years, consisting of 50 females (41%) and 72 males (59%). Demographic and biochemical parameters of the participants are summarized in Table 1.

Significant group differences were observed for fasting blood glucose (FBG), glycated hemoglobin (HbA1c), diabetes duration, high-density lipoprotein (HDL), serum creatinine, albumin/creatinine ratio (Alb/Cr-R), and erythrocyte sedimentation rate (ESR) (p<0.05). Post-hoc Bonferroni analysis revealed that the macroalbuminuric group had a markedly longer duration of diabetes compared to both the normoalbuminuric and microalbuminuric groups (p=0.001). Additionally, the control group exhibited significantly lower FBG levels than all diabetic groups (p≤0.001).

Serum creatinine concentrations were significantly elevated in the macroalbuminuric group compared with controls (p=0.014). HDL cholesterol levels differed across groups, with the microalbuminuric group

Table 1 Demographic, Clinical, and Laboratory Characteristics of Study Groups

	Control (N = 30)	Normo-albuminuria (N = 31)	Micro-albuminuria (N = 31)	Macro-albuminuria (N = 30)	P value
Age (years)	51,63±7,26	53,09±9,26	54,68±11,47	51,83±9,79	0,581
Male n(%)	%43,30 (13)	%35,50 (11)	%48,40 (15)	%36,70 (11)	0,708
BMI (kg/m²)	27,49±3,79	28,79±4,74	30,77±5,96	27,83±5,38	0,054
DM duration (years)		9,13±6,73^{a}	11,35±7,84^{b}	15,87±6,01^{a,b}	0,001*
Glucose (mg/dL)	92,00±7,77^{a,b,c}	161,13±92,56^{a,b,c}	223,84±107,52^{b}	267,63±194,51^{c}	<0.001*
Creatinine (mg/dL)	0,83±0,13^{a}	1,45±2,39	1,01±0,24^{b}	1,92±1,24^{a,b}	0,011 *
Triglyceride (mg/dL)	199,86±217,01	179,16±92,26	168,03±70,19	117,83±40,28	0,074
HDL (mg/dL)	50,20±11,13^{a}	52,41±12,07^{b}	42,42±10,37^{a,b,c}	51,27±15,06^{c}	0,007*
LDL (mg/dL)	115,83±38,37	106,48±39,82	96,42±36,73	113,33±39,48	0,206
HbA1C (%)	5,21±0,28^{a,b,c}	7,76±2,71^{a}	8,81±2,12^{b}	8,58±0,04^{c}	<0.001*
Alb/Cr-R (mcg/L / mg/L)		7,54±6,48^{a,b}	107,35±72,22^{a,c}	356,62±390,30^{b,c}	<0.001*
CRP (mg/L)	6,65±5,16	6,38±10,85	9,47±13,26	8,25±6,77	0,557
ESR (mm/h)	17,86±13,36^{a}	23,64±16,53^{b}	27,93±27,53^{c}	41,27±28,27^{a,b,c}	0,001*

Parameters were expressed as mean ± SD. Abbreviations: BMI: Body mass index; DM: Diabetes mellitus; HDL: High density lipoprotein; LDL: Low density lipoprotein; HbA1c: Glycated hemoglobin; Alb/Cr-R: Albumin/creatine ratio; CRP: C-reactive protein; ESR: Erythrocyte sedimentation rate ; * P < 0.05 is considered significant for statistical analyses. ^{a,b,c,d} There is a significant difference between the parameters marked with the same letter (p < 0.05).

showing significantly lower HDL compared to other diabetic subgroups (p=0.007). Furthermore, ESR values were notably higher in the macroalbuminuric group relative to all others (p=0.001). HbA1c values also differed significantly among groups, with the control group showing the lowest levels (p<0.001).

Plasma thiol/disulfide homeostasis parameters varied significantly among the groups (p<0.05, Table 2). Native thiol and total thiol concentrations were highest in the control group and lowest in the macroalbuminuric group, with statistically significant differences between these categories (p<0.001). Disulfide levels were reduced in the macroalbuminuric group compared with the controls (p=0.019).

Correlation analyses indicated a strong negative correlation between BMI, HbA1c, diabetes duration, creatinine, CRP, and sedimentation with total and native thiol levels (p<0.001, Table 3). Specifically, HbA1c showed a significant negative correlation with native thiol (r=-0.287, p<0.01) and total thiol levels (r=-0.330, p<0.01).

To further explore independent predictors of thiol-disulfide homeostasis, multiple regression analyses were performed. HbA1c (β=-0.42, p<0.001) and diabetes duration (β=-0.38, p<0.001) emerged as the strongest predictors of decreased thiol levels, independent of other variables.

These associations indicate a relationship between glycemic burden and thiol depletion; however, the magnitude of correlations suggests that thiol parameters are influenced by multiple clinical factors.

Table 2 Plasma Thiol/Disulfide Homeostasis Parameters in the Study Groups

	Control (N = 30)	Normo-albuminuria (N = 31)	Micro-albuminuria (N = 31)	Macro-albuminuria (N = 30)	P value
N-Thiol (µmol/L)	299,44±46,88[a]	291,65±60,51[b]	254,59±47,55[a,b]	201,07±40,54[a,b]	<0.001*
T-Thiol (µmol/L)	333,58±44,59[a]	325,29±54,21[b]	287,02±50,93[a,b]	229,01±38,42[a,b]	<0.001*
Disulfide (µmol/L)	19,14±4,14[a]	16,82±7,02	16,22±9,57	13,90±6,19[a]	0,019
Disulfide/N-Thiol (%)	0,07±0,04	0,07±0,04	0,07±0,04	0,07±0,04	0,890
Disulfide/T-Thiol (%)	0,06±0,02	0,06±0,03	0,06±0,03	0,06±0,03	0,749
N-Thiol/T-Thiol (%)	0,9±0,05	0,89±0,06	0,89±0,06	0,87±0,06	0,628

Parameters were expressed as mean ± SD. Abbreviations: N-Thiol: Native Thiol; T-Thiol: Total Thiol; *P < 0.05 is considered significant for statistical analyses. [a,b,c,d] There is a significant difference between the parameters marked with the same letter (p < 0.05).

Table 3 Correlation Analysis of Clinical and Biochemical Parameters with Thiol/Disulfide Homeostasis Markers

	BMI	Glucose	Creatinine	HDL	HbA1c	Alb/Cr-R	CRP	ESR
N-thiol	0,054	-0,229*	-0,345**	0,202*	-0,287**	-0,315**	-0,184*	-0,392**
T-thiol	0,030	-0,305**	-0,301**	0,215*	-0,330**	-0,348**	-0,225*	-0,413**
Disulfide	-0,034	-0,360**	0,169	0,048	-0,222*	-0,142	-0,167	-0,052

Abbreviations: BMI: Body mass index; HDL: High density lipoprotein; HbA1c: Glycated hemoglobin; Alb/Cr-R: Albumin/creatine ratio; CRP: C-reactive protein; ESR: Erythrocyte sedimentation rate; N-Thiol: NativeThiol; T-Thiol: Total Thiol.
* Correlation level is low-medium strength (p<0,05)
** Correlation level is very good - excellent (p <0.01)

Discussion

Diabetic nephropathy (DN), a key microvascular complication of type 2 diabetes mellitus (T2DM), continues to be a major cause of end-stage renal disease worldwide. Albuminuria has traditionally been used as a marker for DN, reflecting glomerular damage and also serving as a predictor of cardiovascular outcomes (15, 16). Nevertheless, the diagnostic specificity of albuminuria is compromised by its susceptibility to external factors such as hydration status, exercise, and infection. As a result, increasing emphasis has been placed on complementary biomarkers to strengthen risk stratification and improve the longitudinal monitoring of disease progression.

Recent investigations have underscored the importance of thiol/disulfide homeostasis as a novel oxidative stress marker. This balance plays a central role in maintaining redox regulation, antioxidant defenses, and multiple cellular processes (10). In line with this, our findings showed a stepwise decline in native thiol and total thiol levels with increasing albuminuria severity. These results support the view that oxidative stress contributes significantly to DN progression (9, 17). The observed inverse relationship between thiol status, disease chronicity, and HbA1c provides additional evidence that prolonged hyperglycemia precipitates oxidative damage and redox dysregulation (12, 18).

Further research highlights the broader relevance of thiol/disulfide imbalance in a variety of chronic disorders. For example, its diagnostic and prognostic utility has been emphasized in metabolic syndrome, diabetes, and cardiovascular disease (2). In autoimmune conditions such as Graves' disease, abnormal thiol/disulfide regulation has been linked to elevated oxidative stress (19). Similarly, findings in acute urticaria patients—where decreased thiol levels coincide with elevated oxidative stress—reinforce the concept of thiol/disulfide imbalance as a marker of systemic oxidative dysregulation (4).

Our correlation analyses confirmed significant inverse associations between HbA1c, Alb/Cr-R, and thiol levels, echoing previous findings that chronic hyperglycemia and albuminuria severity are tied to oxidative stress markers. Elevated HbA1c levels promote the generation of advanced glycation end-products (AGEs), which contribute to increased oxidative burden and thiol depletion (9). In parallel, Alb/Cr-R showed significant inverse correlations with native thiol ($r=-0.315$, $p<0.01$) and total thiol ($r=-0.348$, $p<0.01$), indicating that albuminuria may reflect systemic oxidative stress rather than representing a kidney-specific marker alone. Moreover, previous studies have associated elevated Alb/Cr-R with endothelial dysfunction and increased inflammation, underscoring its role as a systemic indicator of redox imbalance. (9). Accordingly, thiol/disulfide homeostasis should be interpreted as a systemic redox marker, whereas albuminuria primarily reflects renal microvascular injury; therefore, substitution is not implied.

Mechanistically, persistent hyperglycemia promotes oxidative stress through mitochondrial ROS generation, activation of the AGE–RAGE axis, and redox-sensitive inflammatory signaling, all of which can deplete circulating thiol pools. Thiol groups act as major extracellular antioxidants and redox buffers; thus, reduced native/total thiol levels may reflect diminished systemic antioxidant capacity accompanying endothelial dysfunction and microvascular injury. This provides biological plausibility for the graded decrease observed across albuminuria stages.

Importantly, the impact of thiol/disulfide imbalance extends beyond nephropathy to other diabetes-associated complications such as cardiovascular disease. Studies have shown its role in endothelial dysfunction, a precursor of atherosclerosis and vascular pathology in diabetes (10). For instance, Rodrigues et al. demonstrated that diminished thiol levels were linked to vascular impairment in chronic kidney disease, supporting the idea of systemic consequences of thiol dysregulation (9). Similarly, thiol depletion has been implicated in increased arterial stiffness and subclinical atherosclerosis in T2DM patients, emphasizing its broader relevance in cardiovascular morbidity (19).

Although disulfide levels were reduced in the macroalbuminuria group compared with controls ($p=0.019$), the stability of disulfide-to-thiol ratios indicates that thiol depletion, rather than disulfide accumulation, is more indicative of oxidative imbalance. Prior reports similarly suggest that native and total thiol levels are more sensitive markers of redox disturbances in DN (14). In contrast, disulfide ratios tend to remain relatively stable, reflecting their limited role in acute oxidative shifts (10).

In a study evaluating patients with metabolic syndrome, total and native thiol levels were found to be significantly lower compared to healthy controls; although a decrease in disulfide levels was also observed, it did not reach statistical significance, and no differences were noted in the disulfide/total thiol and disulfide/native thiol ratios (20). In a similar study evaluating patients with heart failure with preserved ejection fraction, total thiol, native thiol, and disulfide

levels were found to be lower ($p < 0.001$, $p = 0.001$, $p = 0.041$ respectively) compared to the control group; however, no significant differences were observed in the disulfide/total thiol and disulfide/native thiol ratios (21). These results underscore the utility of thiol levels as dynamic markers of redox regulation, particularly in the context of chronic diseases like diabetes.

Despite the recognized involvement of disulfides in oxidative stress, our study did not find significant differences in thiol/disulfide ratios across groups. While other studies have reported elevated disulfide concentrations in advanced diabetic complications (22), our results suggest that thiol depletion may be a more consistent indicator of systemic oxidative imbalance in DN. Variability in findings may be due to methodological differences, sample diversity, or confounding factors such as inflammation and metabolic control.

Overall, our study confirms that thiol concentrations are reliable indicators of systemic oxidative stress. However, unlike albuminuria which directly reflects renal damage, thiol/disulfide balance provides a broader view of systemic oxidative changes. This supports the idea that it should not be considered a replacement for albuminuria, but rather a complementary marker. This wider relevance is further supported by evidence linking thiol depletion to both inflammation and endothelial dysfunction, underscoring its integrative role in the context of chronic disease management (14).

Comparable findings have been documented in other oxidative stress-related conditions. Matteucci et al. (17) reported that impaired thiol metabolism accelerates diabetes complications, while Jeevan et al. (23) demonstrated reduced thiol levels in chronic kidney disease patients. Similarly, Yazici et al. (18) observed diminished thiol levels in diabetes patients, linking them to enhanced oxidative stress, highlighting the systemic implications of thiol dysregulation.

This study has limitations. First, the cross-sectional design precludes causal inference; therefore, it cannot be determined whether thiol depletion contributes to nephropathy progression or represents a consequence of cumulative oxidative and inflammatory burden. Longitudinal studies with repeated thiol/disulfide assessments are warranted to clarify temporal relationships and prognostic value. Second, albuminuria was used for nephropathy staging, but eGFR was not consistently available across all participants. Inclusion of eGFR (and combined KDIGO risk categories) would enable more comprehensive characterization of renal involvement and should be incorporated in future studies.

Clinical Implications and Future Directions
Although albuminuria remains the reference standard for DN diagnosis, our findings suggest that thiol/disulfide homeostasis may serve as an adjunctive oxidative stress biomarker. As these markers reflect systemic redox status rather than kidney-specific damage, their role in clinical practice should be considered within a multimarker approach that incorporates both established and emerging parameters (24, 25). Large-scale multicenter longitudinal studies are needed to validate the prognostic potential of thiol markers in DN progression.

Additionally, therapeutic strategies that target thiol homeostasis could offer new opportunities to mitigate oxidative stress-related complications in T2DM. Supplementation with agents such as N-acetylcysteine or glutathione precursors has been shown to restore redox balance (14). Evaluating these interventions in conjunction with thiol-based monitoring could yield new insights into optimizing the management of diabetes and its complications.

Conclusion

Dynamic thiol/disulfide homeostasis provides meaningful information regarding oxidative mechanisms in DN. While it cannot replace albuminuria as a diagnostic tool, it may serve as a complementary marker, enhancing risk prediction and disease monitoring in T2DM. Further research, particularly longitudinal and interventional studies, will be essential to fully establish the clinical value of thiol/disulfide metrics in the management of diabetes-associated renal and cardiovascular complications.

Conflict of Interest Statement
The authors have no conflicts of interest to declare.

Ethical Approval
This study was approved by the Suleyman Demirel University Clinical Research Ethics Committee (Date: 15.04.2020, No: 72867572.050.01.04). The study was conducted in accordance with the principles of the Declaration of Helsinki.

Consent to Participate and Publish
Written informed consent to participate and publish was obtained from all individual participants or legal guardians included in the study.

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Data available on request from the authors.

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Authors Contributions
A.A: Methodology; Supervision; Writing- review & editing

B.A: Formal analysis; Methodology; Visualization

S.E: Conceptualization; Project administration; Supervision

Ö.E: Data curation; Formal analysis; Validation

H.D: Data curation; Investigation; Writing-original draft

Ş.T: Investigation; Validation; Visualization

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