Natural Resources for Human Health
eISSN: 2583-1194

Original Research

Received 18 May 2026
Revised 24 June 2026
Accepted 15 July 2026

Natural Resources for Human Health 2026, Vol. 6 (7s): 01–08

KEYWORDS:
Chronic kidney disease, Hemodialysis, Oxidative stress, Malondialdehyde, Total antioxidant capacity, Ferric reducing antioxidant power.

Association of Malondialdehyde and Total Antioxidant Capacity with Renal Function in Patients Undergoing Maintenance Hemodialysis: A Case–Control Study

Oxidative Stress and Antioxidant Capacity in Hemodialysis Patients

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ABSTRACT:

Background: Chronic kidney disease (CKD) is a progressive disorder characterized by irreversible loss of renal function and is associated with persistent oxidative stress and impaired antioxidant defense. Maintenance hemodialysis, although indispensable for patients with end-stage kidney disease, may further exacerbate oxidative stress by enhancing reactive oxygen species (ROS) generation and reducing circulating antioxidant levels, thereby contributing to cardiovascular complications and disease progression.

Objective: To evaluate oxidative stress biomarkers and total antioxidant capacity (TAC) in patients with chronic kidney disease undergoing maintenance hemodialysis and compare the findings with healthy controls.

Materials and Methods: This hospital-based case–control study included 40 CKD patients receiving maintenance hemodialysis and 40 healthy controls. Venous blood samples were collected after obtaining written informed consent and Institutional Ethics Committee approval. Serum urea, blood urea nitrogen (BUN), creatinine, uric acid, sodium, potassium, chloride, malondialdehyde (MDA), and TAC were estimated using standard biochemical methods. MDA was measured by the thiobarbituric acid reactive substances (TBARS) assay, while TAC was assessed using the Ferric Reducing Antioxidant Power (FRAP) assay. Statistical analysis was performed using the independent-samples Student's *t*-test and Pearson's correlation coefficient. A two-tailed p-value < 0.05 was considered statistically significant.

Results: CKD patients undergoing hemodialysis showed significantly higher serum urea (90.04±6.4 vs. 27.8±3.9 mg/dL), BUN (42.0±3.0 vs. 13.0±1.8 mg/dL), creatinine (9.5±1.4 vs. 0.8±0.1 mg/dL), uric acid (10.2±1.3 vs. 5.1±0.7 mg/dL), potassium (4.8±0.7 vs. 4.2±0.3 mEq/L), chloride (102.6±3.9 vs. 100.5±1.6 mEq/L), and MDA (8.6±2.0 vs. 2.9±0.6; $p<0.001$), whereas serum sodium (136.0±3.1 vs. 139.5±1.9 mEq/L) and TAC (0.7±0.2 vs. 1.7±0.09; $p<0.001$) were significantly reduced compared with controls. MDA showed a strong positive correlation with dialysis duration ($r = 0.87$) and significant

positive correlations with creatinine (r = 0.507) and uric acid (r = 0.37). TAC demonstrated a significant positive correlation with estimated glomerular filtration rate (r = 0.64).

Conclusion: CKD patients undergoing maintenance hemodialysis exhibit marked oxidative stress, evidenced by increased lipid peroxidation and diminished antioxidant capacity, along with significant renal dysfunction and electrolyte disturbances. Assessment of MDA and TAC may serve as useful biomarkers for monitoring oxidative stress and disease progression in patients undergoing maintenance hemodialysis.

INTRODUCTION

A gradual and irreversible loss of kidney function over months or years due to a decrease in nephron number and function, which results in a lower glomerular filtration rate (GFR), is the hallmark of chronic kidney disease (CKD), a progressive illness. CKD is defined by the National Kidney Foundation's (NKF) Kidney Disease Outcomes Quality Initiative (KDOQI) as kidney damage or a GFR of less than 60 mL/min/1.73 m² that has persisted for at least three months, regardless of the underlying cause. [1, 2] chronic kidney disease (CKD) is linked to endothelial dysfunction, oxidative stress, and persistent inflammation. These factors all play a major role in the development of cardiovascular problems, which are the main cause of morbidity and mortality in patients with chronic renal failure. [3] By encouraging endothelial damage, vascular inflammation, and lipid peroxidation, oxidative stress is a key factor in the development and advancement of atherosclerosis. Furthermore, it has been demonstrated that the length of dialysis treatment increases the severity of oxidative stress, indicating that extended exposure to oxidative damage may hasten renal impairment through cytotoxic processes. [4] An imbalance between the body's antioxidant defence system and the excessive production of reactive oxygen species (ROS) and reactive nitrogen species (RNS) leads to oxidative stress. By scavenging free radicals, endogenous antioxidant enzymes and non-enzymatic antioxidants preserve redox equilibrium under physiological settings. However, oxidative damage to lipids, proteins, nucleic acids, and cellular membranes occurs in CKD due to weakened antioxidant defences and elevated ROS and RNS production. [5]

There is strong evidence that ROS play a role in the pathogenesis of uremic problems, especially in patients on maintenance haemodialysis. Renal damage and endothelial dysfunction are facilitated by increased RNS production and decreased nitric oxide (NO•) generation. Additionally, the higher frequency of cardiovascular illness seen in CKD patients has been linked to oxidative stress.. [6] It has been established that haemodialysis is a significant cause of oxidative stress. Polymorphonuclear leukocytes are activated during dialysis by the interaction between the dialysis membrane and circulating blood, which increases the generation of reactive oxygen species. Even a single haemodialysis session dramatically raises lipid peroxidation while concurrently lowering circulating antioxidant levels, according to multiple studies. Additionally, haemodialysis exacerbates rather than lessens oxidative stress by facilitating the loss of non-enzymatic antioxidants across the semipermeable dialysis membrane. Increased oxidative stress during haemodialysis is primarily caused by antioxidant depletion, inflammatory cell activation, chronic inflammation, and dialysis membrane biocompatibility. [6–7] Numerous chronic conditions, including as diabetes mellitus, atherosclerosis, chronic inflammation, and the development of chronic kidney disease (CKD) into end-stage renal disease (ESRD), have been linked to oxidative stress. Therefore, maintaining antioxidant defence mechanisms is crucial for delaying the progression of the disease and lowering the risk of cardiovascular disease and early death in people with chronic kidney disease. The cumulative activity of both enzymatic and non-enzymatic antioxidants is represented by total antioxidant capacity (TAC), which offers a comprehensive evaluation of the body's antioxidant defence system. Prior research has repeatedly shown that individuals receiving maintenance haemodialysis have much lower TAC, suggesting that their antioxidant defence is compromised. [6–7] Loss of circulating antioxidants during dialysis, together with persistent inflammation and continuous oxidative injury, may further enhance cardiovascular complications and mortality. Furthermore, blood cells undergo structural and functional changes when they come into contact with dialysis membranes of different biocompatibilities, which increases oxidative stress and inflammatory reactions during haemodialysis. [8–9] Thus, the purpose of this study is to assess total antioxidant level and oxidative stress indicators in haemodialysis patients with chronic renal disease.

MATERIALS AND METHODS

Study Design and Participants

This hospital-based case–control study was conducted at the Department of Biochemistry and Department of Nephrology MGM Medical College and Hospital, Navi Mumbai, Maharashtra, India. The study included 40 patients with chronic kidney disease (CKD) undergoing maintenance hemodialysis at the Dialysis Unit of MGM Medical College and Hospital and 40 healthy individuals who served as the control group.

Ethical Approval

The study protocol was reviewed and approved by the Institutional Ethics Committee of MGM Medical College and Hospital. Written informed consent was obtained from all participants prior to enrolment, and the study was conducted in accordance with the ethical principles of the Declaration of Helsinki.

Inclusion Criteria

- Adult patients diagnosed with chronic kidney disease (CKD).
- Patients receiving maintenance hemodialysis for at least three months.
- Patients willing to participate and provide written informed consent.

Exclusion Criteria

- Acute or chronic inflammatory diseases.
- Human Immunodeficiency Virus (HIV) infection.
- Hepatitis B surface antigen (HBsAg)-positive status.
- Patients unwilling to participate in the study.

Sample Collection

Approximately 5 mL of venous blood was collected aseptically from each participant under standard laboratory conditions. Blood samples were allowed to clot, and serum was separated by centrifugation. The separated serum was aliquoted and stored at −20°C until biochemical analysis.

Biochemical Analysis

Serum biochemical parameters, including urea, creatinine, uric acid, malondialdehyde (MDA), and total antioxidant capacity, were estimated in all study participants. Serum urea, creatinine, and uric acid were measured using commercially available diagnostic reagent kits according to the manufacturer's instructions. Malondialdehyde (MDA), a marker of lipid peroxidation and oxidative stress, was estimated using the Thiobarbituric Acid Reactive Substances (TBARS) method. Total antioxidant capacity (TAC) was determined by the Ferric Reducing Antioxidant Power (FRAP) assay, which measures the reducing potential of serum antioxidants by converting ferric (Fe^{3+})-tripyridyltriazine (TPTZ) complex to the ferrous (Fe^{2+})-TPTZ complex under acidic conditions.

Statistical Analysis

Statistical analysis was performed using SPSS software. Continuous variables were expressed as mean ± standard deviation (SD). Statistical analysis was performed using the independent-samples Student's *t*-test and Pearson's correlation coefficient. A two-tailed p-value < 0.05 was considered statistically significant.

RESULTS

The demographic and anthropometric characteristics of the study participants are presented in Table 1. The mean age of patients undergoing maintenance hemodialysis was 47.6 ± 6.7 years**,** compared with 43.0 ± 8.6 years among healthy controls, with a statistically significant difference between the groups ($p = 0.0096$). The mean BMI was significantly lower in the hemodialysis group **(**23.14 ± 2.4 kg/m²**)** than in the control group **(**24.7 ± 3.2 kg/m²**,** $p = 0.019$). Hemodialysis patients

demonstrated significantly higher blood pressure compared with healthy controls. The mean systolic blood pressure was 144.8 ± 20.6 mmHg in the hemodialysis group compared with 119.6 ± 1.6 mmHg in controls ($p < 0.001$). Similarly, the mean diastolic blood pressure was significantly higher among hemodialysis patients **(**83.7 ± 7.1 mmHg**)** than controls **(**79.1 ± 2.6 mmHg**,** $p < 0.001$). Regarding sex distribution, the hemodialysis group comprised 26 males (65%) and 14 females (35%)**,** whereas the control group comprised 30 males (75%) and 10 females (25%)**.**

Table 1. Comparison of Demographic, Anthropometric, and Blood Pressure Characteristics between Hemodialysis Patients and Healthy Controls

Variable	**Hemodialysis**	**Control**	**p value**
Age (years)	47.6±6.7	43±8.6	0.0096
BMI	23.14±2.4	24.7 ±3.2	0.019
Systolic BP	144.8±20.6	119.6 ±1.6	< 0.001
Diastolic BP	83.7±7.1	79.1± 2.6	< 0.001
Male/Female	Male- 26(65%) Female n=14 (35%)	Male-30(75%) Female-10(25%)	-

The biochemical parameters of renal function and serum electrolytes in chronic kidney disease (CKD) patients undergoing maintenance hemodialysis and healthy controls are presented in Table 1. Patients undergoing hemodialysis exhibited significantly elevated serum urea levels compared with healthy controls (90.04 ± 6.4 mg/dL vs. 27.8 ± 3.9 mg/dL, $p < 0.001$), indicating severe impairment of renal excretory function. Similarly, blood urea nitrogen (BUN) levels were markedly higher in the hemodialysis group than in controls (42.0 ± 3.0 mg/dL vs. 13.0 ± 1.8 mg/dL, $p < 0.001$). A substantial increase in serum creatinine concentration was observed among CKD patients receiving haemodialysis (9.5 ± 1.4 mg/dL) compared to healthy individuals (0.8 ± 0.1 mg/dL), and this difference was highly statistically significant ($p < 0.001$). Elevated creatinine levels reflect a marked reduction in glomerular filtration rate and confirm advanced renal dysfunction in the study population. The mean serum uric acid concentration was also significantly higher in the hemodialysis group (10.2 ± 1.3 mg/dL) than in the control group (5.1 ± 0.7 mg/dL) ($p < 0.001$), suggesting impaired renal clearance of uric acid and altered purine metabolism associated with CKD. Evaluation of serum electrolyte levels demonstrated significant differences between the two groups. The mean serum sodium concentration was significantly lower in patients undergoing hemodialysis compared with healthy controls (136.0 ± 3.1 mEq/L vs. 139.5 ± 1.9 mEq/L, $p < 0.001$), indicating a tendency toward dilutional hyponatremia or altered sodium homeostasis commonly observed in CKD.

Conversely, serum potassium levels were significantly higher in the hemodialysis group (4.8 ± 0.7 mEq/L) than in controls (4.2 ± 0.3 mEq/L) ($p < 0.001$), reflecting reduced renal potassium excretion and the impaired ability of diseased kidneys to maintain potassium balance. Serum chloride concentrations were also significantly increased in CKD patients undergoing hemodialysis (102.6 ± 3.9 mEq/L) compared with healthy controls (100.5 ± 1.6 mEq/L), with the difference reaching statistical significance ($p = 0.0019$). Overall, these findings demonstrate profound impairment of renal function accompanied by significant disturbances in electrolyte homeostasis among patients with CKD undergoing maintenance hemodialysis when compared with healthy controls.

Table 2: Biochemical parameters of renal function and serum electrolytes in chronic kidney disease (CKD) patients undergoing maintenance hemodialysis and healthy controls.

Parameter	Haemodialysis	Controls	p value
Urea (mg/dl)	90.04 ±6.4	27.8±3.9	< 0.001
BUN (mg/dl)	42.0 ±3.0	13.0±1.8	< 0.001
Creatinine (mg/dl)	9.5±1.4	0.8±0.1	< 0.001

Uric acid (mg/dl)	10.2±1.3	5.1±0.7	< 0.001
Na (mEq/L)	136.0±3.1	139.5±1.9	< 0.001
K (mEq/L)	4.8±0.7	4.2±0.3	< 0.001
Cl (mEq/L)	102.6±3.9	100.5±1.6	0.0019

The comparison of serum total antioxidant capacity (TAC) and malondialdehyde (MDA) levels between chronic kidney diseases (CKD) patients undergoing maintenance hemodialysis and healthy controls is presented in Table 2. Conversely, serum malondialdehyde (MDA) levels, a well-established biomarker of lipid peroxidation and oxidative stress, were significantly elevated in the hemodialysis group compared with healthy controls. The mean MDA concentration was 8.6 ± 2.0 in CKD patients undergoing hemodialysis, while the corresponding value in the control group was 2.9 ± 0.6 ($p < 0.001$). The significantly higher MDA levels suggest enhanced oxidative damage to cellular membrane lipids in patients with chronic kidney disease undergoing maintenance hemodialysis. MDA showed a strong positive correlation with dialysis duration ($r = 0.87$), $p < 0.001$ and significant positive correlations with creatinine ($r = 0.507$), $p < 0.001$ and uric acid ($r = 0.37$), $p < 0.001$

A significant reduction in serum total antioxidant capacity (TAC) was observed in the hemodialysis group compared with healthy controls. The mean TAC level in CKD patients undergoing hemodialysis was 0.7 ± 0.2, whereas the control group exhibited a significantly higher mean TAC of 1.7 ± 0.09 ($p < 0.001$). This finding indicates a marked impairment of the antioxidant defense system in patients receiving maintenance hemodialysis. TAC demonstrated a significant positive correlation with estimated glomerular filtration rate ($r = 0.64$), $p < 0.001$.

Table 3: Comparison of Total Antioxidant Capacity and Malondialdehyde Levels between Chronic Kidney Disease Patients Undergoing Hemodialysis

Parameter	Haemodialysis	Controls	p value
TAC	0.7±0.2	1.7±0.09	< 0.001
MDA	8.6±2.0	2.9±0.6	< 0.001

DISCUSSION

Sustained decrease in glomerular filtration rate (GFR) to less than 60 mL/min/1.73 m² for at least three months is a hallmark of chronic kidney disease (CKD), which is defined by permanent anatomical and functional degeneration of the kidneys.[9] Renal function is evaluated by either calculating GFR (eGFR) from serum creatinine concentrations or directly measuring GFR (mGFR) using exogenous filtration indicators such inulin.[10] Despite increasing nephron loss, the kidneys have a significant functional reserve that preserves proper physiological balance. However, maintenance haemodialysis (HD) is often started when the eGFR falls to less than 15 mL/min/1.73 m², and biochemical problems typically only show up after more than 50% of renal function has been lost. [11]

Serum urea and creatinine levels were considerably higher in patients receiving maintenance haemodialysis than in healthy controls ($p < 0.001$), indicating serious impairment of renal excretory function. Amin et al. and Khasawnah et al. published similar results, showing markedly elevated serum urea and creatinine concentrations in CKD patients. [12,13] Skeletal muscle constantly produces creatinine by the non-enzymatic breakdown of creatine phosphate, which is mainly removed by glomerular filtration. As a result, serum creatinine is regarded as a trustworthy biochemical indicator of kidney health. As nephron function gradually declines, creatinine clearance is reduced, which causes it to build up in the blood. Similarly, the kidneys typically eliminate urea, the primary byproduct of protein metabolism produced in the liver via the urea cycle. Urea and creatinine accumulate over time in patients with chronic kidney disease (CKD) due to decreased renal function. [14]

Significant alterations in serum electrolyte concentrations were also observed in the present study. Serum sodium levels were significantly lower in patients undergoing maintenance hemodialysis than in healthy controls (136.0 ± 3.1 vs. 139.5 ± 1.9 mEq/L, $p < 0.001$), suggesting disturbances in sodium homeostasis commonly associated with advanced CKD. Conversely,

serum potassium levels were significantly higher in the hemodialysis group (4.8 ± 0.7 vs. 4.2 ± 0.3 mEq/L, $p < 0.001$), reflecting impaired renal potassium excretion secondary to reduced nephron function. Electrolyte abnormalities are frequently encountered in CKD and are associated with increased cardiovascular morbidity and mortality. Therefore, regular monitoring of serum electrolytes is essential for timely detection and correction of electrolyte disturbances in patients receiving maintenance hemodialysis. [15] In contrast, AL-Hisnawi RAAA and Salih H. [16] reported a non-significant reduction in serum sodium levels among CKD patients compared with healthy controls, indicating that electrolyte abnormalities may vary depending on patient characteristics, dialysis adequacy, and disease severity.

It is becoming more well acknowledged that oxidative stress is a key factor in the development of cardiovascular problems and the advancement of chronic kidney disease. Serum malondialdehyde (MDA), a well-known indicator of lipid peroxidation, was considerably greater in CKD patients receiving maintenance haemodialysis than in healthy controls in the current study (8.6±2.0 vs. 2.9 ± 0.6, $p < 0.001$). Increased oxidative damage to membrane lipids due to an overabundance of reactive oxygen species (ROS) is indicated by elevated MDA levels. The significant increase in MDA observed in the present study suggests that oxidative stress is markedly enhanced in patients undergoing maintenance hemodialysis. Although dialysis facilitates the removal of several circulating toxins, the hemodialysis procedure itself promotes ROS generation through activation of polymorphonuclear leukocytes, complement activation, and interaction of blood with the dialysis membrane. Consequently, the increased production of free radicals exceeds the capacity of dialysis to eliminate oxidative products, resulting in persistently elevated MDA concentrations.

Our findings are consistent with those of Neetha Kundoor and Shruti Mohanty et al., [17] who reported a significant increase in post-dialysis MDA levels compared with pre-dialysis values in patients with chronic kidney disease, indicating enhanced oxidative stress during the hemodialysis session. Similarly, De Vecchi, Amedeo F. et al. [18] demonstrated significantly higher plasma MDA concentrations in dialysis patients than in healthy individuals, further supporting the role of lipid peroxidation in CKD.

The present study also demonstrated a significant reduction in total antioxidant capacity (TAC) among patients undergoing maintenance hemodialysis compared with healthy controls (0.7 ± 0.2 vs. 1.7 ± 0.09, $p < 0.001$). TAC represents the cumulative antioxidant activity of enzymatic and non-enzymatic defense systems and reflects the overall ability of biological fluids to neutralize reactive oxygen species. The observed reduction in TAC indicates depletion of endogenous antioxidant reserves resulting from persistent oxidative stress and chronic inflammation associated with CKD and maintenance hemodialysis.

Our findings agree with those reported by Ulver et al. [19], who observed significantly elevated MDA levels accompanied by reduced antioxidant capacity in patients with chronic renal failure receiving dialysis therapy. The simultaneous increase in MDA and decrease in TAC observed in the present study clearly demonstrate an imbalance between oxidant generation and antioxidant defense mechanisms in patients undergoing maintenance hemodialysis. This redox imbalance may contribute to accelerated progression of renal dysfunction, endothelial injury, chronic inflammation, and the increased cardiovascular risk associated with advanced CKD.

Conclusion: Compared with healthy individuals, patients with chronic kidney disease (CKD) undergoing maintenance hemodialysis exhibit marked deterioration in renal function, significant electrolyte disturbances, increased lipid peroxidation, and compromised antioxidant defense mechanisms. The observed elevation in malondialdehyde (MDA) levels, together with a reduction in total antioxidant capacity (TAC), highlights the presence of substantial oxidative stress in this patient population. These findings suggest that the evaluation of oxidative stress biomarkers and antioxidant status may serve as valuable adjunctive tools for assessing disease severity, monitoring disease progression, and evaluating therapeutic responses in CKD patients receiving maintenance hemodialysis. Furthermore, well-designed prospective and randomized clinical studies are warranted to determine whether antioxidant-based therapeutic interventions can effectively reduce oxidative stress, improve clinical outcomes, and enhance the overall quality of life in these patients.

AUTHOR CONTRIBUTIONS

Sonal Tiwari- Investigation, Methodology, Laboratory Analysis

Dr. Parineeta Samant- Conceptualization, , Formal Analysis

FINANCIAL SUPPORT AND SPONSORSHIP

Nil.

CONFLICTS OF INTEREST

There are no conflicts of interest.

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