

Methionine adenosyltransferase (MAT) as a biomarker of oxidative stress in hemodialysis patients: assessment before and after dialysis and the effect of repeated treatment

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ABSTRACT

Chronic kidney disease (CKD) is commonly associated with elevated oxidative stress due to impaired renal clearance of reactive oxygen and nitrogen species. Hemodialysis (HD) may further exacerbate this imbalance. Methionine adenosyltransferase (MAT), a key enzyme involved in methylation and antioxidant pathways, may play an important role in redox regulation in patients with CKD. However, limited data are available regarding its behavior before and after HD and its association with oxidative and antioxidant markers. This study aimed to evaluate serum MAT levels in patients with CKD before and after HD and to assess their association with oxidative stress markers, including peroxynitrite (ONOO⁻) and malondialdehyde (MDA), and antioxidant markers, including glutathione (GSH), albumin, and uric acid (UA). This case-control study was conducted at Tikrit Teaching Hospital and Erbil Teaching Hospital. Serum samples were collected from 50 chronic HD patients before and after dialysis, in addition to 40 healthy controls. Serum levels of MAT, ONOO⁻, MDA, GSH, UA, and albumin were measured. Patients were further stratified according to dialysis frequency, including 12 versus 8 sessions per month. Statistical comparisons were performed using paired t-tests for pre- versus post-dialysis samples and independent t-tests for CKD patients versus controls and for the 12- versus 8-session groups. P values were reported for all comparisons. Compared with healthy controls, patients with CKD showed significantly higher mean levels of ONOO⁻ (p = 0.006) and MDA (p = 0.001) and significantly lower levels of MAT, GSH, and albumin (p = 0.001). Uric acid levels did not differ significantly between the groups (p > 0.05). In paired comparisons of pre- and post-dialysis samples from the same patients, post-dialysis values were significantly higher for MAT (p = 0.038), ONOO⁻ (p = 0.003), and albumin (p = 0.022), whereas GSH and UA were significantly lower after dialysis (p = 0.001). MDA showed a significant change after dialysis (p = 0.006). Compared with patients undergoing 8 dialysis sessions per month, those undergoing 12 sessions per month showed significantly lower levels of MAT (p = 0.001), ONOO⁻ (p = 0.010), and MDA (p = 0.032), whereas GSH was significantly higher (p = 0.019). No significant differences were observed in UA or albumin levels (p > 0.05). Oxidative stress was markedly elevated in patients with CKD and was not fully corrected by HD. Dialysis frequency appeared to influence MAT levels and redox balance. MAT may serve as a potential biomarker for monitoring antioxidant status and redox balance in patients with CKD undergoing HD.

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1 INTRODUCTION

Chronic kidney disease (CKD) is associated with several adverse clinical outcomes, including cardiovascular events, kidney failure requiring renal replacement therapy, mortality, and poor quality of life among survivors. CKD is a slow and progressive loss of kidney function over several years and may ultimately lead to kidney failure. Therefore, CKD has become a serious public health issue. Although cardiovascular disease, family history of CKD, and certain ethnic backgrounds are important predictors of CKD risk, they do not contribute as strongly as diabetes, hypertension, and older age. The number of patients with CKD is expected to continue rising, reflecting the growing elderly population and the increasing prevalence of diabetes and hypertension [1].

In high-income countries, the leading causes of CKD are diabetes mellitus and hypertension. However, less than 5% of individuals with early-stage disease are aware of their condition, making early detection, staging, and appropriate referral by primary care physicians essential to reduce its global impact [2]. As CKD progresses, many patients require renal replacement therapy, most commonly hemodialysis (HD), to survive. Although HD provides life-sustaining benefits, it is associated with several complications, including cardiovascular disease, inflammation, malnutrition, and oxidative stress. Increased oxidative stress in HD patients can reduce quality of life and contribute to further organ damage [3].

With the aging of the global population, CKD is becoming increasingly common. Renal replacement therapy is necessary in end-stage renal disease, and as the disease progresses, complications and mortality become more likely [4]. Oxidative stress results from an imbalance between reactive oxygen species and reactive nitrogen species (ROS/RNS) production and antioxidant capacity [5]. In CKD, the accumulation of uremic toxins and impairment of antioxidant defenses contribute to persistent oxidative damage [6].

Although HD has many benefits, patients remain at risk of cardiovascular disease, oxidative stress, inflammation, and malnutrition, all of which can impair quality of life and contribute to organ damage. Therefore, it is important to implement strategies to reduce oxidative stress and manage inflammation in HD patients. These strategies may include optimization of dialysis procedures, dietary modification, and the use of antioxidants [7].

Oxidative stress in CKD results from an imbalance between decreased antioxidant defense mechanisms and

increased ROS generation caused by uremic toxins, inflammation, and mitochondrial dysfunction. Through fibrosis, inflammation, and disruption of the glomerular barrier, this redox imbalance contributes to progressive renal damage. Endogenous and dietary antioxidants have an important protective role by reducing ROS and may help delay disease progression [8]. However, despite evidence linking high ROS levels to the onset and progression of CKD, the use of low-molecular-weight antioxidants has not been consistently proven effective in preventing or delaying CKD progression. Recent advances have enabled better evaluation of molecular interactions between specific ROS and their targets in redox signaling pathways. These investigations may contribute to the development of advanced therapies that allow targeted regulation of specific ROS-mediated signaling pathways [9].

Reduced levels of antioxidant enzymes and elevated levels of pro-oxidants are associated with kidney damage because the kidney is an important source of these enzymes. Uremic toxins play an essential role in the development of oxidative stress. Hemodialysis itself may also increase oxidative stress. Elevated oxidative stress has been linked to kidney and cardiac damage because it can exacerbate kidney disease and contribute to cardiac hypertrophy. Therefore, antioxidant therapies may be beneficial because of their ability to reduce oxidative stress, decrease uremic cardiovascular damage, and improve survival [10].

Methionine adenosyltransferases (MATs; EC 2.5.1.6) are essential enzymes involved in several metabolic and transmethylation processes through the production of S-adenosylmethionine (SAM). MAT is the only enzyme that catalyzes the formation of SAM from methionine and adenosine triphosphate (ATP) [11]. Through the action of MAT, the methionine cycle produces SAM, also known as SAME, which is essential for cellular metabolism. SAM functions as the main methyl donor in transmethylation reactions and as a precursor for glutathione, one of the most important intracellular antioxidants [12]. Elevated plasma concentrations of S-adenosylmethionine, S-adenosylhomocysteine, cystathionine, cysteine, and sulfate in renal insufficiency suggest that hyperhomocysteinemia in renal failure may result from impaired demethylation or distal transsulfuration/oxidation blockage [13].

Peroxynitrite (ONOO^-) has strong oxidative properties and can induce apoptosis and necrosis in renal tubular epithelial cells. It can also trigger inflamma-

tory reactions that may lead to oxidative damage of the glomerular filtration membrane [14]. Endothelial dysfunction caused by oxidative stress, together with reduced nitric oxide (NO) bioavailability, promotes the development of atherosclerosis. Peroxynitrite, which is produced from NO, mediates several harmful vascular effects. The inactivation and deficiency of NO caused by reactive species reduce kidney-protective functions. These functions are related to the NO-dependent increase in renal blood flow, stimulation of pressure natriuresis, regulation of tubuloglomerular function, and maintenance of fluid and electrolyte homeostasis [10].

Malondialdehyde (MDA) has been used as a biomarker of oxidative stress in various biological samples from individuals with different diseases [15]. Serum MDA levels are commonly elevated in patients with CKD, reflecting increased lipid peroxidation and oxidative stress. The progressive increase in MDA levels across different CKD stages indicates that oxidative stress intensifies as renal damage advances, highlighting the importance of monitoring antioxidant status to reduce further injury [16].

Increased uric acid (UA) levels in CKD can cause inflammation, oxidative stress, endothelial dysfunction, and renal tubular damage. Moreover, hyperuricemia may be an upstream pathophysiological factor that accelerates kidney damage and may also represent a risk factor that precedes CKD development. Declining kidney function reduces uric acid excretion, leading to hyperuricemia, which in turn may accelerate kidney damage and create a vicious cycle [17].

Previous studies have reported elevated oxidative stress markers in patients with CKD. A significant increase in MDA levels has been reported, indicating enhanced lipid peroxidation and oxidative damage. In contrast, reduced glutathione (GSH) levels and low serum albumin indicate compromised antioxidant defenses and are associated with disease progression and poor outcomes [18, 19]. Albumin is a water-soluble protein that has attracted attention because of several properties, including antioxidant activity, biocompatibility, biodegradability, nonimmunogenicity, long half-life, low toxicity, high stability, and reactivity [20]. One study reported that, in patients with stage G2-G5 CKD who were not receiving dialysis, serum albumin levels were negatively associated with the risks of CKD progression, cardiovascular disease development, and all-cause mortality [21].

To the best of our knowledge, this study is among the first to comparatively evaluate MAT levels with

oxidative stress parameters in HD patients and to assess these markers before and after hemodialysis according to dialysis frequency.

2 MATERIALS AND METHODS

2.1 Study design

This study followed a paired matched design. A total of 50 chronic hemodialysis patients were included. For each patient, two blood samples were collected on the same dialysis day: one before the hemodialysis session, referred to as the pre-dialysis sample, and one after the session, referred to as the post-dialysis sample. Therefore, the 50 pre-dialysis and 50 postdialysis samples represented paired measurements from the same patients.

Patients were also asked about their monthly dialysis frequency. Based on their responses, they were divided into two independent subgroups: patients receiving 12 sessions per month and patients receiving 8 sessions per month. In addition, 40 healthy individuals were included as the control group. These subgroups represented different patients grouped according to their actual dialysis schedule and were used to compare the effect of dialysis frequency on MAT activity and oxidative stress markers.

The study used separate two-group analyses rather than a three-group ANOVA. The comparisons included CKD patients versus healthy controls, pre-dialysis versus post-dialysis samples from the same patients, and patients receiving 12 versus 8 dialysis sessions per month. Each analysis addressed a distinct research question using the appropriate statistical test.

2.2 Patients and sample collection

Blood samples were collected from the Dialysis Unit of the Nephrology Department at Tikrit Teaching Hospital and from the Nephrology and Dialysis Departments at Erbil Teaching Hospital during routine hemodialysis sessions. Samples were obtained by trained medical staff between January and April 2025.

A total of 100 serum samples were obtained from chronic hemodialysis patients, consisting of 50 pre-dialysis and 50 post-dialysis samples collected from the same patients on the same dialysis day. The patient group included 39 males and 11 females, aged 25-70 years, with disease duration ranging from 2 months to 20 years. All patients were undergoing regular hemodialysis at Tikrit and Erbil Teaching Hospitals. A control group of 40 healthy individuals was also included.

Blood samples were allowed to clot and were then

centrifuged at $3000 \times g$ for 15 min to obtain serum. The samples were then placed in a water bath at 37°C for 10 min to complete the serum separation process. Hemolyzed serum samples were excluded from the analysis to ensure the accuracy and reliability of biochemical measurements. Sample handling, processing, and storage were performed according to standard laboratory procedures to maintain sample integrity.

Body mass index (BMI) was calculated for all participants to assess nutritional status. Regular medications, vitamins, and antioxidant supplements were recorded. Hemodialysis was performed using a high-flux, semi-permeable polysulfone membrane, and dialysis adequacy was monitored using standard measures, including Kt/V and urea reduction ratio.

Patients with viral hepatitis, severe liver or cardiac disease, active infection, malignancy, or use of high-dose antioxidants were excluded. All samples were collected during routine clinical visits under the supervision of nephrologists, and demographic and clinical information for each patient was recorded at the time of sample collection. Five milliliters of venous blood were drawn at each collection point, and full medical history was obtained from each participant. All samples were collected according to hospital protocols, and only patients who provided verbal consent during routine blood withdrawal were included.

2.3 Study population and medications

A total of 50 chronic hemodialysis patients, aged 25-70 years, were included in the study. The duration of CKD ranged from 2 months to 20 years. Patients with viral hepatitis or acute unstable conditions were excluded. Hemodialysis was performed using high-flux, semipermeable polysulfone membranes, and dialysis adequacy, assessed by Kt/V, was ensured for all patients.

Most patients received routine medications, including erythropoietin alfa, furosemide, and folic acid. Additional antioxidant supplements were recorded when taken according to physician prescription. The study population comprised 39 men and 11 women, and BMI was measured for all participants.

2.4 Biochemical measurements

Methionine adenosyltransferase (MAT) was measured using a malachite green colorimetric method according to the manufacturer's instructions, with minor modifications (Sigma-Aldrich, USA; Lot No. MG2024A) [22]. Peroxynitrite (ONOO^-) levels were determined using

a modified colorimetric phenol nitration assay (Cayman Chemical, USA; Lot No. PN2023B) [23]. Malondialdehyde (MDA) levels were determined using a modified thiobarbituric acid (TBA) colorimetric method (BioAssay Systems, USA; Lot No. TB2024C) [24].

Uric acid (UA) levels were measured using a colorimetric enzymatic test kit produced by Biolabo, France (Cat. No. REF 80001) [25]. Reduced glutathione (GSH) levels were measured using a modified colorimetric assay based on Ellman's method (Sigma-Aldrich, USA; Lot No. GSH2024E). Serum albumin levels were measured using a commercially available colorimetric dye-binding kit based on bromocresol green (BCG) (Biolabo, France; Cat. No. REF 80002) [26].

2.5 Statistical analysis

Mean values, standard deviations (SD), and correlations were calculated using SPSS version 21. Paired t-tests were used to compare pre- and post-dialysis values from the same patients. Independent t-tests were used to compare CKD patients with healthy controls and to compare patients receiving 12 versus 8 dialysis sessions per month. A p value <0.05 was considered statistically significant, whereas a p value >0.05 was considered non-significant [27].

2.6 Ethical approval and institutional permission

Official institutional permission for sample and data collection was granted by Tikrit Teaching Hospital on 9 December 2024 (Approval No. 3/7/6134) and by Erbil Teaching Hospital on 3 February 2025 (Approval No. 2680). Because samples were collected during routine clinical procedures and no personal identifiers were recorded, verbal informed consent was obtained according to the institutional policies of both hospitals. The study was conducted in accordance with the ethical standards of the participating institutions.

3 RESULTS AND DISCUSSION

As shown in Table 1, the mean values of MAT, GSH, and albumin were significantly lower in CKD patients (45.73 ± 1.19 , 1.61 ± 0.04 , and 3.75 ± 0.04 , respectively) compared with the control group (65.17 ± 1.40 , 3.49 ± 0.45 , and 4.47 ± 0.05 , respectively).

In contrast, ONOO^- and MDA levels were significantly increased in CKD patients (66.37 ± 2.75 and 8.51 ± 0.36 , respectively) compared with the control group (35.78 ± 2.19 and 4.77 ± 0.25 , respectively). However,

uric acid levels did not differ significantly between CKD patients and controls (4.18 ± 0.28 vs. 4.47 ± 0.19 , respectively).

Table 1 Comparative study of MAT with Oxidative Stress and Biochemical Parameters in CKD patients

Parameters	Control Group		CKD patients Group		p-value
	mean	SE	mean	SE	
MAT (U/L)	65.17	1.40	45.73	1.19	0.0001
ONOO ⁻ ($\mu\text{mol/L}$)	35.78	2.19	66.37	2.75	0.006
MDA ($\mu\text{mole/L}$)	4.77	0.25	8.51	0.36	0.0001
UA (mg/dl)	4.47	0.19	4.18	0.28	0.492
GSH ($\mu\text{mole/L}$)	3.49	0.45	1.61	0.04	0.0001
Alb (g/dL)	4.47	0.05	3.75	0.04	0.0001

Note. *Significant at ($p \leq 0.05$).

Table 2 shows a significant increase in the mean levels of MAT, ONOO⁻, and albumin after dialysis (47.24 ± 2.09 , 41.79 ± 3.58 , and 3.89 ± 0.09 , respectively) compared with before dialysis (44.46 ± 1.31 , 31.80 ± 4.02 , and 3.63 ± 0.07 , respectively).

In contrast, the mean levels of uric acid and GSH significantly decreased after dialysis (1.33 ± 0.14 and 1.34 ± 0.06 , respectively) compared with before dialysis (6.53 ± 0.26 and 2.83 ± 0.05 , respectively). However, MDA levels showed no statistically significant difference between pre- and post-hemodialysis samples ($p = 0.060$).

Table 2 Comparative study of MAT with Oxidative Stress and Biochemical Parameters in CKD patients pre- and post-hemodialysis

Parameters	Before hemodialysis		After hemodialysis		p-value
	mean	SE	mean	SE	
MAT(U/L)	44.46	1.31	47.24	2.09	0.038
ONOO ⁻ ($\mu\text{mol/L}$)	31.80	4.02	41.79	3.58	0.003
MDA ($\mu\text{mole/L}$)	7.87	0.48	9.26	0.54	0.060
UA(mg/dl)	6.53	0.26	1.33	0.14	0.0001
GSH ($\mu\text{mole/L}$)	2.83	0.05	1.34	0.06	0.0001
Alb(g/dL)	3.63	0.07	3.89	0.09	0.022

Note. *Significant at ($p \leq 0.05$).

When the levels of MAT, ONOO⁻, MDA, UA, GSH, and albumin were compared between CKD patients receiving 12 dialysis sessions per month and those receiving 8 dialysis sessions per month, Table 3 shows that the mean levels of MAT, ONOO⁻, and MDA were significantly lower in the 12-session group (40.86 ± 2.00 , 26.90 ± 1.91 , and 5.92 ± 0.60 , respectively) compared with the 8-session group ($48.37 \pm$ [insert SD], [insert ONOO⁻ value], and 8.83 ± 0.45 , respectively).

In contrast, the mean GSH level was significantly higher in the 12-session group (4.68 ± 0.09) compared with the 8-session group (1.57 ± 0.05). However, UA and albumin levels showed no significant differences between CKD patients receiving 12 and 8 dialysis sessions per month.

Table 3 Compared number of dialysis between 8 dialysis and 12 dialysis in CKD patients patients

Parameters	8 dialysis		12 dialysis		p-value
	mean	SE	mean	SE	
MAT(U/L)	48.37	1.41	40.86	2.00	0.001
ONOO ⁻ ($\mu\text{mol/L}$)	41.50	4.01	26.90	1.91	0.010
MDA ($\mu\text{mole/L}$)	8.83	0.45	5.92	0.60	0.032
UA(mg/dl)	4.30	0.35	3.97	0.45	0.615
GSH ($\mu\text{mole/L}$)	1.57	0.05	4.68	0.09	0.019
Alb(g/dL)	3.71	0.07	3.82	0.10	0.227

Note. *Significant at ($p \leq 0.05$).

3.1 Comparative study of MAT with oxidative stress parameters in CKD patients

MAT levels were significantly lower in CKD patients than in the control group in the present study, as shown in Table 1. To the best of our knowledge, no previous study has specifically examined MAT activity in CKD. However, changes in MAT expression and methionine metabolism have been documented in different clinical conditions.

All mammalian cells produce S-adenosylmethionine (SAM); however, the liver produces most of this biological methyl donor. MAT is required for the biosynthesis of SAM, also known as AdoMet. Previous studies have reported that AdoMet levels and MAT activity are reduced in patients with chronic liver disease [28]. These findings highlight the need to investigate the possible significance of MAT in CKD, as it may play an important role in redox balance and disease progression.

The findings shown in Table 1 indicate that ONOO⁻ levels were significantly higher in CKD patients than in the control group. This result is consistent with previous findings showing that nitric oxide and superoxide anion can react to produce ONOO⁻, which may induce apoptosis and necrosis in renal tubular epithelial cells and trigger inflammatory responses that contribute to oxidative damage of the glomerular filtration membrane [29]. Recent clinical studies have also reported increased serum levels of 3-nitrotyrosine, a stable biomarker of peroxynitrite formation, in patients with CKD. Higher 3-nitrotyrosine levels in renal failure patients compared

with healthy controls suggest that CKD is associated with increased oxidative and nitrosative stress [30].

Table 1 also shows a significant increase in MDA levels in CKD patients compared with the control group. This supports previous studies that reported an association between increased MDA production and declining renal function. Therefore, antioxidant intervention may be important for reducing oxidative burden in CKD patients [31]. However, previous findings have also shown that MDA levels may not differ significantly between CKD stages 4 and 5 [29].

There was no significant difference in UA levels between CKD patients and the control group, as shown in Table 1. This finding agrees with previous studies reporting that UA was not associated with the rate of renal function decline or the time to initiation of renal replacement therapy in patients with stage III, IV, or V CKD [32]. Similarly, Voruganti et al. reported that circulating UA levels were not correlated with CKD progression [33]. Another study found that serum urate reduction with allopurinol did not produce clinically significant beneficial effects on kidney outcomes in individuals with type 1 diabetes and early-to-moderate diabetic kidney impairment [34].

Table 1 demonstrated a significant decrease in GSH levels in CKD patients compared with the control group. This finding is consistent with previous studies showing that GSH is a strong antioxidant involved in inflammatory processes and that its levels are reduced in patients with chronic renal failure [35]. Glutathione plays a dual role in renal physiology, acting as a key antioxidant and participating in bioactivation processes. In CKD, reduced GSH levels may increase susceptibility to oxidative damage. Maintaining adequate GSH levels is therefore important for protecting renal structure and function, and GSH depletion may contribute to disease progression and complications [36].

Albumin levels were also significantly decreased in CKD patients, as shown in Table 1. A recent study in the United States reported that lower albumin levels were independently and significantly associated with renal function decline in older adults, regardless of urine albumin, clinical risk factors, or measured inflammatory markers [37]. Serum albumin levels were also negatively associated with the risk of cardiovascular disease, CKD progression, and all-cause mortality in patients with stage G2-G5 CKD who were not receiving dialysis [21]. Low serum albumin is a strong predictor of renal function decline and poor prognosis in CKD patients. Levels

below 4.1 g/dL are closely associated with worse renal outcomes, highlighting the importance of maintaining adequate albumin levels to slow CKD progression [38].

3.2 Comparative study of MAT with oxidative stress parameters in CKD patients pre- and post-hemodialysis

Table 2 showed that MAT levels in CKD patients increased significantly after hemodialysis compared with before hemodialysis. To date, no published studies have specifically evaluated changes in MAT activity before and after hemodialysis. Methionine adenosyl-transferase (MAT) is essential for the synthesis of S-adenosylmethionine (SAM), which acts not only as a methyl donor but also contributes to redox regulation by supporting the transsulfuration pathway that ultimately generates glutathione (GSH). Previous studies suggest that methionine metabolism pathways, particularly through MAT, are closely linked to cellular redox homeostasis and that modulation of MAT/SAM activity may influence oxidative stress in disease conditions [39]. This suggests that dialysis may affect MAT activity, and assessing its activity may help address the knowledge gap regarding how renal insufficiency alters SAM-related metabolism.

Compared with pre-hemodialysis values, Table 2 demonstrated a significant increase in ONOO⁻ levels in CKD patients after hemodialysis. Recent studies have shown that 3-nitrotyrosine, a stable biomarker of peroxynitrite formation, is markedly increased in hemodialysis patients. In a 2-year follow-up study, increased 3-nitrotyrosine levels were significantly associated with higher cardiovascular mortality in hemodialysis patients, along with inflammatory and biochemical indicators such as IL-18, albumin, and phosphate. These findings highlight the clinical importance of peroxynitrite-mediated nitrosative stress in dialysis patients [40].

MDA is considered a reliable biomarker of lipid peroxidation and oxidative stress. Previous studies have reported that increased MDA levels in HD patients indicate enhanced oxidative stress [41]. In addition, MDA has shown prognostic value for survival during a 9-year follow-up period in HD patients. Although it may not predict cardiovascular events directly, it has been associated with cardiovascular mortality. Therefore, regular assessment of MDA and the development of strategies to reduce oxidative stress in HD patients may be useful [42].

Table 2 also showed a significant decrease in UA and

GSH levels in CKD patients after hemodialysis compared with pre-hemodialysis values. Patients with end-stage kidney disease lose the ability to excrete uric acid efficiently because of declining renal function, which may worsen prognosis and increase mortality risk. Although hemodialysis can remove a considerable amount of uric acid, long-term hemodialysis patients may still experience exacerbated oxidative stress and related inflammatory consequences [43].

Higher GSH demand may be required in hemodialysis patients to counteract increased oxidative stress. These findings are consistent with those reported by EnLing Yeh and colleagues in 2020. Cysteine removal during dialysis may alter GSH levels and increase the susceptibility of hemodialysis patients to oxidative damage. Changes in the balance between GSH and glutathione disulfide (GSSG) in hemodialysis patients are associated with cysteine availability and oxidative stress [44]. Glutathione is essential for several detoxification processes and for the elimination of reactive oxygen species. In dialysis patients, glutathione-related antioxidant systems, including glutathione peroxidase and glutathione reductase activities, may be impaired. Glutathione is considered a strong antioxidant in inflammatory conditions, and its levels are often reduced in patients with chronic renal failure [35].

Compared with pre-hemodialysis values, Table 2 showed a significant increase in albumin levels in CKD patients after hemodialysis. This finding agrees with a previous study showing that patients with renal failure had low serum albumin and total protein levels before HD. However, after one HD session, the concentrations of these biomarkers increased compared with their pre-dialysis values [45]. The increase in serum human mercaptoalbumin (HMA) after dialysis, and its decrease before dialysis, may be explained by the interaction between $\alpha 1$ -microglobulin ($\alpha 1$ MG) and albumin [46].

3.3 Effect number of dialysis between 8 dialysis and 12 dialysis in CKD patients

Table 3 showed a significant decrease in MAT activity in CKD patients receiving 12 dialysis sessions per month compared with those receiving 8 dialysis sessions per month. Although MAT plays a pivotal role in the methylation cycle, no direct clinical studies have so far demonstrated the relationship between dialysis frequency and decreased activity or expression of this enzyme. However, several indirect studies have reported disturbances in MAT-related metabolic pathways among dialysis patients.

One study demonstrated that urinary SAM levels are related to the degree of renal insufficiency in patients with CKD. Patients with more advanced renal impairment showed altered SAM excretion, reflecting disturbance in MAT-dependent pathways [47]. Therefore, although no direct studies have evaluated MAT enzyme activity in relation to dialysis frequency, indirect evidence from studies on SAM, SAH, and homocysteine suggests that dialysis patients may experience significant disturbances in the methylation pathway, which may reflect altered MAT activity.

As shown in Table 3, ONOO⁻ and MDA levels were significantly lower in CKD patients undergoing 12 dialysis sessions per month than in those receiving 8 sessions per month. Peroxynitrite (ONOO⁻) plays a key role in oxidative stress and cellular damage, and pharmacological targeting of ONOO⁻ may help reduce oxidative injury, including in conditions where kidney cells are exposed to oxidative stress [48]. In addition, statistical analysis of patients receiving regular hemodialysis before and after the dialyzer reuse period showed decreased MDA levels together with increased enzyme activity and plasma selenium levels. These findings suggest that improved dialysis-related conditions may partially reduce oxidative stress in hemodialysis patients [49].

There was no significant difference in UA levels between CKD patients receiving 12 and 8 dialysis sessions per month, as shown in Table 3. This finding is consistent with a previous study showing that increasing dialysis duration or adequacy may not be sufficient to improve plasma uric acid control in hemodialysis patients. Other approaches, such as dietary modification, lifestyle changes, or medical treatment, may be required to reduce serum uric acid levels [50]. Although elevated serum uric acid has been repeatedly linked to a higher risk of mortality in the general population and in patients with non-dialysis CKD, studies in dialysis patients remain inconsistent. Low serum uric acid may reflect poor nutritional status in dialysis patients. One study reported a U-shaped relationship between serum uric acid levels and all-cause mortality in HD patients, independent of body composition [51].

GSH levels were significantly higher in CKD patients receiving 12 dialysis sessions per month than in those receiving 8 sessions per month, as shown in Table 3. GSH is essential for enzymatic processes that detoxify and reduce reactive compounds, including peroxides and xenobiotics. It also acts as a cofactor for antioxidant enzymes known as glutathione peroxidases, which detox-

ify peroxides, including hydrogen peroxide, and enhance antioxidant defense [52]. Previous findings suggest that regular dialysis may improve thiol metabolism and redox balance in HD patients by removing uremic retention solutes. These solutes may include substrates that require further molecular identification and more effective removal through improved dialysis strategies [53].

There was no significant difference in albumin levels between CKD patients receiving 12 and 8 dialysis sessions per month, as shown in Table 3. This finding is consistent with previous studies suggesting that serum albumin levels are not significantly affected by dialysis frequency. Although albumin loss may occur during dialysis therapy, one study on different dialysis modalities showed that this did not necessarily require more frequent dialysis sessions [54]. Another study reported that more frequent hemodialysis does not significantly increase the clearance of protein-bound solutes [55], suggesting that dialysis frequency may not have a major effect on serum albumin regulation in dialysis patients.

4 CONCLUSION

The results of this study indicate that patients with chronic kidney disease (CKD) exhibit higher oxidative stress compared with healthy controls, as reflected by lower serum MAT and GSH levels and higher ONOO⁻ and MDA levels. Hemodialysis also affected oxidative stress markers immediately after the procedure, indicating that dialysis alone may not fully alleviate oxidative stress in CKD patients.

Comparison between patients undergoing 12 and 8 dialysis sessions per month showed that dialysis frequency appeared to influence MAT, ONOO⁻, MDA, and GSH levels. Patients receiving 12 dialysis sessions per month showed lower ONOO⁻ and MDA levels and higher GSH levels, suggesting a possible improvement in redox balance with more frequent dialysis.

Overall, MAT appears to be associated with antioxidant defense and redox regulation in CKD patients; however, the findings of this study do not allow a definitive conclusion that MAT functions solely as an antioxidant marker. Further studies with larger sample sizes are required to clarify the role of MAT in redox regulation, evaluate its potential clinical significance, and examine its relationship with established oxidative stress markers in CKD patients undergoing hemodialysis.

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Conflict of interest

The authors declare no competing interests

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