International Journal of Research in Pharmacy and Pharmaceutical Sciences
www.pharmacyjournal.in
ISSN: 2455-698X
Received: 09-04-2026, Accepted: 10-05-2026, Published: 25-05-2026
Volume 11, Issue 2, 2026, Page No. 45-50

Diagnostic performance of Myeloperoxidase and oxidative stress biomarkers in Chronic Kidney Disease: A comparative study of anemic and non-anemic patients

Muzahim A Khalaf*, Israa G Zainal
Department of Chemistry, College of Science, University of Kirkuk, Kirkuk, Iraq
Corresponding Author: Muzahim A Khalaf

Abstract
Background: Chronic kidney disease [CKD] is characterized by reduced kidney function, accumulation of toxins, metabolic dysregulations, chronic oxidative stress and inflammation. This study evaluated the efficacy of some biomarkers related to oxidative stress and inflammation in CKD patients with and without anemia and assessed their diagnostic performance.
Methodes: A case-control study was involving 120 serum samples divided into: 50 apparently healthy subjects as control, 35 CKD patients without anemia, and 35 CKD patients with anemia. Serum myeloperoxidase [MPO], ischemia-modified albumin [IMA], total antioxidant capacity [TAC], malondialdehyde [MDA], glutathione [GSH], nitric oxide [NO], thiol parameters, iron indices, vitamin D3, parathyroid hormone [PTH], and liver enzymes were quantified. ROC analysis evaluated the diagnostic utility of the biomarkers.
Results: Patients with CKD exhibited significantly [$p \leq 0.05$] lower activity of MPO, ALT, AST, lower levels of IMA, TAC, NO, iron, and vitamin D3 and significantly [$p \leq 0.05$] higher MDA, GSH, AST/ALT ratio, UIBC, and TIBC compared with controls. The levels of TAC, MDA, and GSH differed significantly [$p \leq 0.05$] between anemic and non-anemic CKD groups. Myeloperoxidase demonstrated good diagnostic accuracy in differentiating CKD from controls [AUC ≈ 0.996]. The only marker capable of distinguishing CKD patients with anemia from those without anemia was TAC.
Conclusion: the results indicated that MPO, MDA, GSH, IMA, TAC, and vitamin D_3, have significant diagnostic value in CKD, and TAC provides discriminatory utility between anemic and non-anemic CKD patients, supporting its integration into clinical evaluation.

Keywords: Chronic kidney disease, myeloperoxidase, oxidative stress, antioxidant biomarkers and ROC analysis

Introduction

Chronic kidney disease [CKD] is a widespread and increasing health issue worldwide, marked by a slow decline in kidneys function, leading to the accumulation of toxins and metabolites in the blood [1]. Studies have shown that CKD is not only a condition related to kidney function but also associated with disturbances in metabolic balance, increased oxidative stress, and chronic health issues such as inflammation [2]. Oxidative stress and chronic inflammation are key factors involved in the development of CKD complications. Increased reactive oxygen species [ROS] cause damage to vital cellular components, such as proteins, lipids, and DNA [3]. Myeloperoxidase [MPO] was selected as a biomarker due to its prominent role in the inflammatory and oxidative processes associated with chronic kidney disease [CKD] [4]. Myeloperoxidase is an enzyme secreted by granulocytes, such as neutrophils, and is involved in the production of free radicals and peroxides, such as hypochlorite [HOCl], which contribute to the destruction of cellular tissue and increase oxidative damage [5]. This effect makes MPO a key factor in the development and progression of tissue inflammation in CKD [6]. Regarding kidney complications, MPO is believed to indirectly weaken the immune system and facilitate the deterioration of kidney function by continuously activating oxidative inflammation [5]. Research shows that MPO activity in patients with CKD were lower in patients than healthy individuals, which may be because of decreased neutrophil function in these patients due to toxins resulting from kidney failure, or even the effects of hemodialysis treatment. Low MPO activity is considered as an indicator of a weakened immune response and increased susceptibility to infection and tissue inflammation [7].Additionally, a study showed that MPO correlates negatively with the body's ability to combat oxidative stress [such as total antioxidant capacity [TAC], suggesting that increased MPO activity leads to decreased antioxidant defenses [8]. From the above relationship, this study aimed to highlight the importance of MPO as a potential diagnostic tool for assessing oxidative stress in patients with CKD, enhancing its role in monitoring disease progression and guiding treatment more accurately and to evaluate oxidative and inflammatory biomarkers in CKD patients with and without anemia and to assess their diagnostic performance using receiver operating characteristic [ROC] analysis.

Patients and Methods

Study Population and Clinical Definitions

This case-control study included 120 participants. CKD was defined according to the KDIGO guidelines as a decreased GFR [< 60 mL/min/1.73 m²] for more than 3 months. All patients were in stages 4-5 and recruited from the Al-Amal Dialysis Centre. Anemia was defined according to WHO criteria: Hemoglobin [Hb] < 13 g/dL in men and < 12 g/dL in women. The study protocol was approved by the local ethics committee, and informed consent was obtained.

Sample Collection and Pre-analytical Handling

Venous blood samples were collected, centrifuged at 3000 rpm for 10 minutes, and serum was stored at -80°C [instead of -20°C for better stability]. To ensure enzyme stability, samples underwent only one freeze-thaw cycle. Quality control was maintained using standard reference materials for each assay.

Biochemical and Hematological Analysis

Serum MPO activity was estimated using a spectrophotometric method with O–O-dianisidine dihydrochloride as the substrate [9]. Nitric oxide [NO] levels were measured using the Griess Reagent Assay [10]. The TAC was measured by method based on 2,2'-azinobis [3-ethylbenzothiazoline-6-sulfonate] [ABTS] [11]. The MDA was measured using a method based on the TBARS [Thiobarbituric Acid Reactive Substances] assay, as described by Buege and Aust [12]. Glutathione, total thiol, native thiol, and disulfide group levels were conducted using the Ellman method [13]. Dervisoglu *et al* [14] method use to estimate ischemia-modified albumin [IMA]. Colorimetric Iron Assay with Ferene [Ferene-S Method] [15], and Unsaturated Iron-Binding Capacity [UIBC] Using the Ferrozine Method [16], product kit: Kinetic UV Method for Alanine Aminotransferase [ALT] [17], and Alanine Aminotransferase [AST] [17].Serum 25 OH Vitamin D levels were measured using a VIDAS 25 OH Vitamin D Total testing kits [Biomerieux, Marcy I'Etoile, France] on mini VIDAS automated immunoassay platform [18]

Statistical Analysis

Statistical analyses were performed using IBM SPSS Statistics [version 26] and GraphPad Prism [version 9.0]. Data normality was assessed using the Shapiro-Wilk test. Receiver Operating Characteristic [ROC] curve analysis was employed to evaluate the diagnostic performance. The Area Under the Curve [AUC] was reported with 95% Confidence Intervals [95% CI]. A p-value of ≤ 0.05 was considered statistically significant.

Results

Table 1 presents the serum activity of MPO, and the levels of all studied parameters expressed as the mean ± SD, in all studied groups.

Table 1 : The activity of MPO and levels of all studied parameters in all studied groups

Parameters	Group I mean ±SD n=50	Group II mean ±SD n=35	Group III mean ±SD n=35
MPO U/L	34.66 ± 5.615	10.17 ± 3.992[a]	11.96 ± 4.479[a]
IMA	1.140 ± 0.132	0.5197 ± 0.158[a]	0.609± 0.141[a]
TAC µM	189.8 ± 12.29	89.66 ± 8.750[a]	28.51 ± 5.046[ab]
MDA nmol/L	44.92± 8.545	120.4± 10.53[a]	76.37 ± 9.727[ab]
GSH µmol/L	1.046 ± 0.609	10.22 ± 2.965[a]	8.148± 2.253[ab]
NO µM	16.96 ± 4.932	13.31 ± 4.542[a]	12.44± 2.972[a]
Total thiol µmole/L	0.086 ± 0.015	0.087 ± 0.013	0.092± 0.007
Natie thiol µmole/L	0.040 ± 0.005	0.041 ± 0.012	0.046 ± 0.012
Di sulphide µmole/L	0.024± 0.009	0.023 ± 0.006	0.023 ± 0.006
ALT U/L	18.18±7.721	10.47±2.042[a]	12.28±5.993[a]
AST U/L	19.98± 6.847	17.62±4.087	16.72±3.272[a]
AST/ALT	1.297±0.588	1.726±0.369[a]	1.608±0.341[a]
UIBC µmol/L	47.38± 12.39	63.55±6.784[a]	64.59 ± 8.183[a]
IRON µmol/L	20.72± 7.273	11.82±2.975[a]	9.072±2.066 [a]
TIBC µmol/L	60.24± 12.52	75.07±7.625[a]	76.79±8.633[a]
Vit D3 ng/ml	69.66 ±12.09	16.64±2.825[a]	12.47±2.287[a]

significant differences [p≤0.05] between patient groups compared with the control. [b] significant difference [p≤0.05] between CKD without and with anemia patients.

The results indicated significantly [$P \leq 0.05$] decrease in the activity of MPO, ALT, AST, and the levels of IMA, TAC, NO, iron, vit D_3 and significantly [$P \leq 0.05$] increase in the levels of MDA, GSH, AST/ALT, UIBC, and TIBC in patients groups compared to control. Group III represented significantly [$P \leq 0.05$] decrease in TAC, MDA and GSH levels compared to group II.

Tables 2 and 3 showed the significant correlation analysis between MPO activity and the parameters studied in groups II and III respectively.

Table 2: significant correlation coefficient between MPO activity and the parameters studied in CKD without anemia patients +

Parameter	r/p
MPO vs TAC	-0.431/0.02

Table 3: Correlation coefficient between MPO activity and the parameters studied in CKD patients with anemia

Parameter	r/p
MPO vs NO	0.379/0.043
MPO vs native thiol	0.379/0.043
MPO vs disulfide	-0.402/0.031

Table 2 showed that MPO correlated negatively with TAC in group II, correlated negatively with disulfide levels, and positively with NO and native thiol levels in group III. The results in Table 4 showed the ROC curve for Group II compared to Group I, MPO at a cutoff of approximately 18.81 with 96.4% sensitivity and 100% specificity, and an AUC of roughly 0.996, the ROC curve for group III compared to Group I at various cutoff points: MPO at about 19.6, with 100% sensitivity and specificity [AUC ≈ 0.996]; and the ROC curve of group III compared to group II, MPO at cutoff ≈ 18.3, sensitivity 85%, specificity 40%, AUC ≈ 0.40; AST at cutoff ≈ 11.5, sensitivity 0%, specificity 100%, AUC ≈ 0.36;

Table 4: ROC in Group II and III compared to Group I

ROC in group I compared to control					
Cutt off	Specificity	Sensitivity	AUC	Parameters	NO.
18.8120	1.000	0.964	0.996	MPO	1
ROC in CKD with anemia patients compare to control groups					
19.6500	1.000	1.000	0.996	MPO	1
ROC in CKD with anemia patients compare to without anemia					
18.30	1.000	0.000	0.367	MPO	1

Figs. 1,2 and 3 represented the ROC analysis of MPO in Group II, Group III compared to Group I and group II compared to group III respectively.

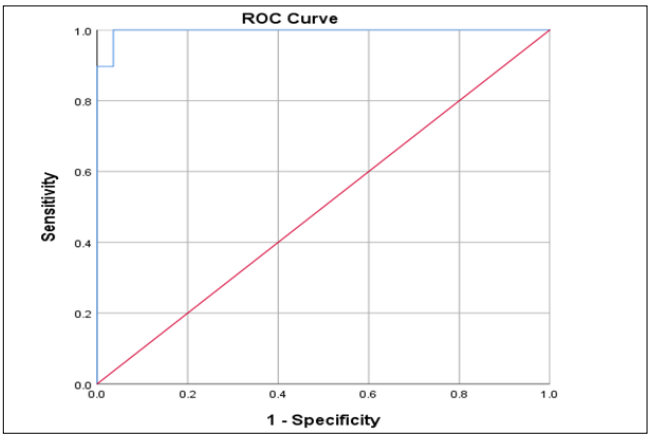

Fig 1: ROC of MPO in Group II compared to Group I.

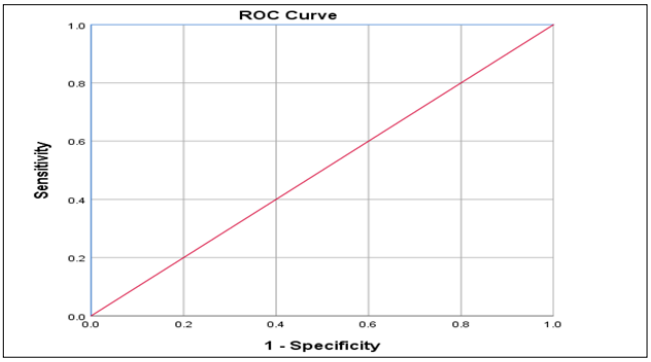

Fig 2: ROC of MPO in Group III compared to Group I.

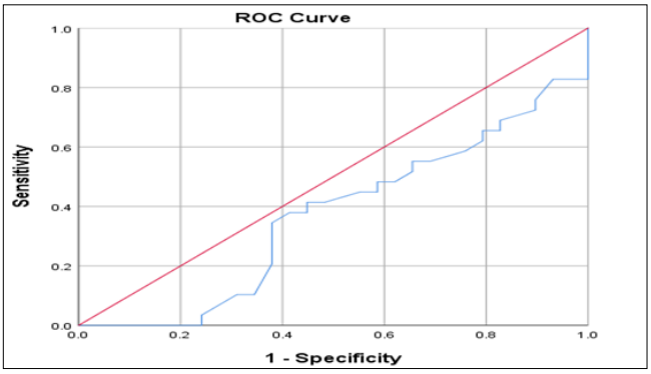

Fig 3: ROC of MPO in group II compared to group III.

Discussion

Myeloperoxidase is a neutrophil enzyme and a primary source of ROS, especially HOCl, which is one of the most powerful oxidants in the body [19]. In patients with kidney failure, MPO is rapidly depleted because of ongoing oxidative stress [20]. Our results demonstrated a significant decrease in MPO activity in CKD patients compared to controls [AUC = 0.996, 95% CI: 0.991–1.000]. While MPO showed exceptional diagnostic accuracy in identifying CKD, it exhibited poor discriminatory power between anemic and non-anemic CKD subgroups [AUC = 0.40]. This suggests that MPO exhaustion in neutrophils occurs early in the progression of renal failure, likely due to chronic exposure to uremic toxins, and is not directly exacerbated by the presence of anemia." This may explain why IMA levels are not significantly elevated compared to acute disease [6]. Abdelrahman *et al* [21], confirms that the disruption of albumin and protein synthesis caused by kidney failure impacts the production and accumulation of IMA in the blood. Elmukhtar Habas Sr. *et al* [22], shows that anemia increases oxidative stress and directly decreases antioxidant capacity, including the use of TAC as a supportive measure agent, and Abod K. S., *et al* [23], measured TAC and found a significantly decrease in patients with CKD compared to control.The relative decline in MDA among anemic renal failure patients could result from compensatory mechanisms or treatments like antioxidants, iron supplements, or erythropoietin [24]. Zhang *et al* [25] found, significant increase in MDA level in CKD patients compared to control, also suggested that patients receiving regular anemia treatment might have lower MDA. Patients with kidney failure consistently experience high levels of ROS [26], this stimulates the production of GSH as part of the antioxidant defense mechanism. The buildup of uremic toxins stimulates the production of GSH as a protective response to safeguard cells from damage caused by these toxins [27], Interestingly, elevated GSH here does not necessarily indicate improvement; instead, it might signal chronic oxidative stress and the body's attempt to compensate. Al Za'abi *et al* [28], showed that patients with early-stage CKD have a compensatory increase in GSH, but this increase decreases in advanced stages due to excessive consumption, and Nasri *et al* [29], discovered that elevated GSH is linked to increased activity of defense pathways, such as NCKD2, supporting the hypothesis of a compensatory rise in GSH during the early stages of the disease. Reduced NO production in CKD patients occurs due to inhibition of the eNOS enzyme activity in endothelial cells, which decreases NO synthesis [30].Chronic Inflammation and Endothelial Dysfunction in CKD leads to and decreases NO production [30].and with the study by Morsy *et al* [31], they indicated that the buildup of urea and uremic toxins reduces the activity of the enzyme NOS, which lowers NO levels. In the advanced stages of kidney failure, the body reaches a new balance between free radicals and antioxidants because of the continued activation of defense pathways such as thiol-disulfide homeostasis [32]. Cakirca *et al* [33], found that both total and native thiol levels were significantly lower in patients, while disulfide levels remained similar and did not differ significantly from control after adjusting for albumin levels. Global AST activity less influenced by kidney failure than ALT and the AST/ALT ratio [34]. A recent study indicated that AST and ALT activity decrease as the disease progresses, but the ratio between them remains a more reliable indicator of kidney function risk [35]. Ethiopian study confirmed that AST activity is directly proportional to kidney function [measured by eGFR], but they remain less sensitive to changes than ALT as CKD progresses [36].Although ALT is more affected by kidney failure and ischemic attack; AST remains at a more stable level. Oliveira *et al* [37], showed that AST and ALT decrease proportionally as CKD worsens. In contrast, the association between ALT and another factor was stronger, while AST remained relatively stable and Abdulkadir *et al* [38], they observed that AST gradually declined as the disease advanced, but the decrease was very modest compared to ALT, confirming that AST remains within the normal reference range even in the later stages of CKD. In patients with renal failure, renal metabolic damage causes decreased levels of pyridoxal phosphate-binding enzymes, such as ALT, which impairs their transport outward or slows their function production [39]. Italian and Hungarian study, showing that ALT/AST levels gradually decrease as CKD worsens [before and

during dialysis], emphasizing the need to establish new reference ranges for kidney disease patients [40]. Elevated UIBC and TIBC in the patient groups indicate active iron deficiency because the available binding capacity increases when free iron is low [41]. This pattern often indicates functional iron deficiency, where iron stores are full or high, but the body cannot utilize them due to chronic inflammation and increased heparin [42]. Aljuraisy *et al* [43], reported an assessment of kidney failure cases, found significant increases in UIBC and TIBC levels, along with lower iron levels in patient groups compared to controls, without a direct link between vitamin D and iron indicators. Ho *et al* [44], demonstrated that low TIBC in CKD patients is linked to malnutrition and inflammation, consistent with the observation of high UIBC/TIBC and low iron, and Batchelor *et al* [45], showed that the mechanisms of iron deficiency in CKD were examined, emphasizing how inflammation and increased heparin impair iron utilization, resulting in higher TIBC and UIBC and consistently low actual iron levels.. The kidneys convert vitamin D into their active form, calcitriol, in CKD patients, the kidneys' ability is impaired, leading to a functional deficit impairment [46].Association in cases of anemia: Vitamin D stimulates the production of erythropoietin, the hormone responsible for red blood cell production [47]. Zhang *et al* [48], they found that vitamin D3 deficiency is associated with an increased risk of kidney decline and anemia. The observed deficiency in Vitamin D3 [25 [OH]D] in our CKD cohort may contribute to anemia through the modulation of the Hepcidin-Ferroportin axis [49]. Recent studies [Zhang *et al*., 2024] suggest that Vitamin D acts as a negative regulator of Hepcidin; thus, its deficiency leads to iron sequestration and functional iron deficiency, consistent with the elevated UIBC and TIBC levels found in our results [50]."The results of this study showed that there were moderate and statistically significant inverse relationships between MPO activity and TAC level in patients with CKD, where the correlation coefficient was r = -0.4312 with a p value of 0.0195, indicating that increased MPO activity is associated with a decrease in the body's overall ability to resist oxidation. The greater MPO activity, the higher the amount of ROS, and the lower the TAC, which indicates antioxidant consumption or the body's inability to replenish them [51]. Also results showed that MPO activity was negatively correlated with disulfide [r = –0.4015] and was positively correlated with NO [r = 0.3792] and native thiol [r = 0.3788] respectively. Positive correlations with NO and native thiol may indicate that MPO's oxidative and inflammatory activity leads to the production of ineffective NO or NOS-related NO, which depletes native thiols and decreases the oxidized form [disulfide] as MPO levels increase [52]. The negative correlations with disulfide suggest that MPO is linked to a high oxidation state that affects protein conditions and antioxidant system [53]. The results show that the best diagnostic indicators for CKD compared to the control group [AUC > 0.90] MPO, these results are considered excellent indicators for diagnosing CKD compared to the control and the best diagnostic indicators for CKD with anemia compared to controls [AUC > 0.90]: MPO, these are considered excellent indicators for diagnosing CKD with anemia compared to controls. Finally, the results of this study show that weak indicators, with AUC below 0.6, include MPO- which are considered poor indicators for differentiating CKD without anemia from CKD with anemia and are unreliable as sole diagnostic tools.

Conclusion

In conclusion, the present study showed that MPO and TAC could serve as useful biomarkers for monitoring oxidative burden and disease severity. The consistent MPO activity between anemic and non-anemic CKD patients indicates that anemia has a limited effect on neutrophil-driven oxidative responses. However, the observed reduction in TAC, GSH, and MDA among anemic patients underscores the potential benefit of antioxidant-targeted therapies in this subgroup.

Reference

1. Hasegawa S, Jao TM, Inagi R. Dietary metabolites and chronic kidney disease. Nutrients.,2017:9(4):358.
2. Verma S, Singh P, Khurana S, Ganguly NK, Kukreti R, Saso L, *et al*. Implications of oxidative stress in chronic kidney disease: a review on current concepts and therapies. Kidney Res. Clin. Pract.,2021:40(2):183.
3. Al-Hweish A, Sultan SS, Mogazi K, Elsammak MY. Plasma myeloperoxidase, NT-proBNP, and troponin-I in patients on CAPD compared with those on regular hemodialysis. Hemodial. Int.,2010:14(3):308-15.
4. Chen HH, Huang YL, Wu CY, Chen MC, Shiue HS, Hsu SL, *et al*. Plasma myeloperoxidase interactions with cadmium, lead, arsenic, and selenium and their impact on chronic kidney disease. Ecotoxicol. Environ. Saf.,2025:290:117726.
5. Lin W, Chen H, Chen X, Guo C. The roles of neutrophil-derived myeloperoxidase [MPO] in diseases: the new progress. Antioxidants.,2024:13(1):132.
6. Kadatane SP, Satariano M, Massey M, Mongan K, Raina R. The role of inflammation in CKD. Cells.,2023:12(12):1581.
7. Aratani Y. Myeloperoxidase: Its role for host defense, inflammation, and neutrophil function. Arch. Biochem. Biophys.,2018:640:47-52.
8. Garça MF, Aslan M, Tuna B, Kozan A, Cankaya H. Serum myeloperoxidase activity, total antioxidant capacity and nitric oxide levels in patients with chronic otitis media. J. Membr. Biol.,2013:246:519-24.
9. Bradley PP, Priebat DA, Christensen RD, Rothstein G. Measurement of cutaneous inflammation: estimation of neutrophil content with an enzyme marker. J. Invest. Dermatol.,1982:78(3):206-9.
10. Jankar JS, Harley KN, Mohod KM. Study of serum nitric oxide [NO] in type 2 diabetes mellitus patients having normoalbuminuria and microalbuminuria in Central India. Hypertension.,2018:13:14.
11. Erel O. A novel automated direct measurement method for total antioxidant capacity using a new generation, more stable ABTS radical cation. Clin. Biochem.,2004:37(4):277-85.
12. Buege JA, Aust SD. [30] Microsomal lipid peroxidation. Methods Enzymol.,1978:52:302-10.
13. Ellman GL. Tissue sulfhydryl groups. Arch. Biochem. Biophys.,1959:82(1):70-7.
14. Dervisoglu P, Oner T. Usefulness of ischemia-modified albumin for assessment of the effects of small ventricular septal defects on the pulmonary vascular bed. Cardiol. Young.,2021:31(7):1207-12.

15. Artiss JD, Vinogradov S, Zak B. Spectrophotometric study of several sensitive reagents for serum iron. Clin. Biochem.,1981:14(6):311-5.
16. Clement DB, Asmundson RC. Nutritional intake and hematological parameters in endurance runners. Phys. Sportsmed.,1982:10(3):37-43.
17. Burtis CA, Ashwood ER, Aldrich JE, Tietz NW. Tietz fundamentals of clinical chemistry. [No Title]., 1996.
18. Moreau E, Bächer S, Mery S, Le Goff C, Piga N, Vogeser M, *et al*. Performance characteristics of the VIDAS® 25-OH Vitamin D Total assay–comparison with four immunoassays and two liquid chromatography-tandem mass spectrometry methods in a multicentric study. Clin. Chem. Lab. Med.,2016:54(1):45-53.
19. Ulfig A, Leichert LI. The effects of neutrophil-generated hypochlorous acid and other hypohalous acids on host and pathogens. Cell. Mol. Life Sci.,2021:78(2):385-414.
20. Kishi S, Nagasu H, Kidokoro K, Kashihara N. Oxidative stress and the role of redox signalling in chronic kidney disease. Nat. Rev. Nephrol.,2024:20(2):101-19.
21. Abdelrahman G, Wang Q, Nunes B. Knowledge tracing: A survey. ACM Comput. Surv.,2023:55(11):1-37.
22. Habas Sr E, Al Adab A, Arryes M, Alfitori G, Farfar K, Habas AM, *et al*. Anemia and hypoxia impact on chronic kidney disease onset and progression: review and updates. Cureus., 2023, 15(10).
23. Abod KS, Mohammed MT, Taay YM. Evaluation of total oxidant status and antioxidant capacity in sera of acute-and chronic-renal failure patients. J. Phys. Conf. Ser.,2021:1853:12038.
24. Xie T, Yao L, Li X. Advance in iron metabolism, oxidative stress and cellular dysfunction in experimental and human kidney diseases. Antioxidants.,2024:13(6):659.
25. Sun Y, Jin D, Zhang Z, Zhang Y, Zhang Y, Kang X, *et al*. Effects of antioxidants on diabetic kidney diseases: mechanistic interpretations and clinical assessment. Chin. Med.,2023:18(1):3.
26. Gwozdzinski K, Pieniazek A, Gwozdzinski L. Reactive oxygen species and their involvement in red blood cell damage in chronic kidney disease. Oxid. Med. Cell. Longev.,2021:2021(1):6639199.
27. Pieniazek A, Bernasinska-Slomczewska J, Gwozdzinski L. Uremic toxins and their relation with oxidative stress induced in patients with CKD. Int. J. Mol. Sci.,2021:22(12):6196.
28. Al Za'abi M, Ali H, Ali BH. Effect of flaxseed on systemic inflammation and oxidative stress in diabetic rats with or without chronic kidney disease. PLoS One.,2021:16(10):e0258800.
29. Elfaleh I, Abbassi F, Habibi M, Ahmad F, Guedri M, Nasri M, *et al*. A comprehensive review of natural fibers and their composites: An eco-friendly alternative to conventional materials. Results Eng.,2023:19:101271.
30. Baaten CC, Vondenhoff S, Noels H. Endothelial cell dysfunction and increased cardiovascular risk in patients with chronic kidney disease. Circ. Res.,2023:132(8):970-92.
31. El Morsy M, Currie T, Bovie D, Kuzuhara M, Lacy B, Li Y, *et al*. Dynamical and Atmospheric Characterization of the Substellar Companion HD 33632 Ab from Direct Imaging, Astrometry, and Radial-Velocity Data. Astrophys. J.,2025:981(1):20.
32. Ebert T, Neytchev O, Witasp A, Kublickiene K, Stenvinkel P, Shiels PG. Inflammation and oxidative stress in chronic kidney disease and dialysis patients. Antioxid. Redox Signal.,2021:35(17):1426-48.
33. Çakırca G, Damar Çakırca T, Üstünel M, Torun A, Koyuncu I. Thiol level and total oxidant/antioxidant status in patients with COVID-19 infection. Ir. J. Med. Sci.,2021:191:1925-30.
34. Agidew MM, Mulu AT, Teklemariam AB, Mengstie MA, Melake A, Zewde EA, *et al*. Impact of aging on alanine aminotransferase levels and frailty in chronic kidney disease patients: laboratory-based cross-sectional study in Northwest Ethiopia. Front. Med.,2025:12:1524459.
35. Das SS, Hossain MS, Sultana A, Rana FA, Hossen A, Maowla MS, *et al*. The influence of chronic kidney disease on hepatocellular carcinoma. J. Primeasia,2025:6(1):1-8.
36. Mallik R, Biswas B, Islam N, Rahman MM, Elora EJ, Hossain MA, *et al*. The Public Health Burden of Chronic Kidney Disease-Associated Hepatocellular Carcinoma. [No Journal].,2022:5(1):1-9.
37. Reis G, Dos Santos Moreira-Silva EA, Silva DCM, Thabane L, Milagres AC, Ferreira TS, *et al*. Effect of early treatment with fluvoxamine on risk of emergency care and hospitalisation among patients with COVID-19: the TOGETHER randomised, platform clinical trial. Lancet Glob. Health,2022:10(1):e42-51.
38. Abdulkadir A, Abdullahi NO, Waheed SA, Umar MB, Madaki F. Inhibitory effects of *Moringa oleifera* seed extracts on crude xanthine oxidase. [No Journal]., 2023.
39. Martinusen D, Marin JG, Cheng E, Lau W. Chronic kidney disease and end stage renal disease. Renal Med. Clin. Pharm., 2020, 45-115.
40. Okawa Y, Mitsuhashi T, Suzuki E. Aspartate/alanine aminotransferase ratio and development of chronic kidney disease in non-diabetic men and women: a population-based longitudinal study in Kagawa, Japan. Clin. Exp. Nephrol.,2024:28:909-21.
41. Faruqi A, Zubair M, Mukkamalla SKR. Iron-Binding Capacity. StatPearls [Internet]., 2024.
42. Pasricha SR, Tye-Din J, Muckenthaler MU, Swinkels DW. Iron deficiency. Lancet.,2021:397(10270):233-48.
43. Alkaralosy SM, Aljuraisy YHZ, Zidan ZK. Evaluation of the Antioxidant Activity of Fig Leaf Extract and Olive Leaf Extrac. J. Educ. Sci. Stud., 2023, 1(22).
44. Yu PH, Lin MY, Chiu YW, Lee JJ, Hwang SJ, Hung CC, *et al*. Low serum iron is associated with anemia in CKD stage 1–4 patients with normal transferrin saturations. Sci. Rep.,2021:11(1):8343.
45. Batchelor EK, Kapitsinou P, Pergola PE, Kovesdy CP, Jalal DI. Iron deficiency in chronic kidney disease: updates on pathophysiology, diagnosis, and treatment. J. Am. Soc. Nephrol.,2020:31(3):456-68.
46. Annamalai C, Viswanathan P. Vitamin D and Acute Kidney Injury: A Reciprocal Relationship. Biomolecules,2025:15(4):586.
47. Zouine N, Lhilali I, Godderis L, El Midaoui A, El Jaafari S, Filali-Zegzouti Y. The Interplay Between

Vitamin D Deficiency, Iron Status, and Anemia Risk in Moroccan Women of Reproductive Age: A Cross-Sectional Analysis. Epidemiologia.,2024:5(4):805-27.
48. Zhang J, Zhang A. Association between serum 25-hydroxyvitamin D3 level and cognitive impairment in older chronic kidney disease patients. Sci. Rep.,2024:14(1):12403.
49. Pistis KD, Westerberg PA, Qureshi AR, Beshara S, Sterner G, Bárány P, *et al*. The effect of high-dose vitamin D supplementation on hepcidin-25 and erythropoiesis in patients with chronic kidney disease. BMC Nephrol.,2023:24(1):20.
50. Ramos CF, Doulidis PG, Polakova N, Burgener IA, Jensen-Jarolim E, Cimarelli G, *et al*. Iron deficiency in dogs suffering from atopic dermatitis. BMC Vet. Res.,2024:20(1):506.
51. Cozza G, Rubin M, Artusi I. Mapping the oxidative landscape in cystic fibrosis: methodological frontiers and application. Front. Pharmacol.,2025:16:1632924.
52. Aranda-Rivera AK, Cruz-Gregorio A, Arancibia-Hernández YL, Hernández-Cruz EY, Pedraza-Chaverri J. RONS and oxidative stress: an overview of basic concepts. Oxygen.,2022:2(4):437-78.
53. Tejaswi G, Suchitra MM, Rajasekhar D, Kiranmayi VS, Rao PS. Myeloperoxidase, protein carbonyls and oxidative stress in coronary artery disease. J. Indian Coll. Cardiol.,2017:7(4):149-52.