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SERUM ADVANCED OXIDATION PROTEIN PRODUCTS AS BIOMARKERS OF OXIDATIVE STRESS IN PATIENTS WITH CHRONIC KIDNEY DISEASE

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Abstract

Background: Chronic kidney disease (CKD) is characterized by progressive deterioration of renal function and is associated with increased oxidative stress, which contributes to inflammation, fibrosis, and cardiovascular complications. Advanced Oxidation Protein Products (AOPPs) are reliable biomarkers of oxidative protein damage and have emerged as potential indicators of CKD progression. This study aimed to evaluate serum AOPP levels and determine their association with CKD severity. **Methods:** An analytical cross-sectional study was conducted among 120 patients with CKD stages 2–5 and 30 age- and sex-matched healthy controls at a tertiary care hospital. Serum AOPP concentrations were measured using enzyme-linked immunosorbent assay (ELISA). Routine biochemical parameters, including serum creatinine, estimated glomerular filtration rate (eGFR), serum albumin, and phosphate, were analyzed using standard laboratory methods. CKD severity was classified according to KDIGO guidelines. Statistical analyses included independent sample t-test, one-way ANOVA, Pearson's correlation, and multiple linear regression. **Results:** CKD patients exhibited significantly higher serum AOPP levels than healthy controls (138.6 ± 39.8 vs. 56.3 ± 12.4 µmol/L, $p<0.001$). AOPP concentrations increased progressively across CKD stages, from 92.8 ± 18.6 µmol/L in stage 2 to 185.1 ± 30.2 µmol/L in stage 5 ($p<0.001$). Serum AOPPs demonstrated a strong positive correlation with serum creatinine ($r=0.694$, $p<0.001$) and serum phosphate ($r=0.516$, $p<0.001$), while showing a significant negative correlation with eGFR ($r=-0.731$, $p<0.001$) and serum albumin ($r=-0.408$, $p<0.001$). Multiple regression analysis identified serum creatinine and eGFR as independent predictors of circulating AOPP levels. **Conclusion:** Serum AOPP concentrations increase

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significantly with worsening CKD severity and are closely associated with declining renal function. AOPPs may serve as a valuable biomarker for assessing oxidative stress and monitoring disease progression in patients with chronic kidney disease.

Keywords: Advanced Oxidation Protein Products; Chronic Kidney Disease; Oxidative Stress; Biomarkers; Estimated Glomerular Filtration Rate; Renal Function; Protein Oxi.

INTRODUCTION

A growing body of evidence indicates that oxidative stress is a central mechanism in the initiation and progression of chronic kidney disease (CKD), contributing to renal dysfunction, inflammation, fibrosis, and cardiovascular complications. CKD is characterized by a persistent decline in kidney function lasting more than three months and affects approximately 10–15% of the global population, posing a substantial public health burden.[1,2] Despite advances in the management of hypertension, diabetes, and other traditional risk factors, many patients continue to experience progressive loss of renal function. Consequently, there is increasing interest in identifying novel biomarkers that not only reflect disease severity but also provide insight into the underlying pathogenic mechanisms. Among these emerging biomarkers, Advanced Oxidation Protein Products (AOPPs) have gained considerable attention because of their close association with oxidative stress and chronic inflammation in CKD.[3,4]

Advanced Oxidation Protein Products are dityrosine-containing oxidized plasma proteins formed primarily through the reaction of albumin and other plasma proteins with chlorinated oxidants such as hypochlorous acid and chloramines generated by activated neutrophils during oxidative stress.[4,5] Initially described as markers of oxidative protein damage in uremic patients, AOPPs are now recognized not only as biomarkers but also as biologically active mediators that amplify inflammatory and fibrotic pathways. Elevated circulating AOPP concentrations have been consistently observed in patients with CKD, particularly those with advanced disease and those receiving dialysis, reflecting the imbalance between excessive reactive oxygen species production and impaired antioxidant defense mechanisms.[5]

Experimental studies have demonstrated that AOPPs actively participate in renal injury rather than merely reflecting oxidative stress.[5,6] They stimulate oxidative stress through activation of the CD36 receptor and nicotinamide adenine dinucleotide phosphate (NADPH) oxidase, resulting in increased production of reactive oxygen species. Furthermore, AOPPs activate inflammatory signaling pathways including nuclear factor-kappa B (NF-κB), promote the release of pro-inflammatory cytokines such as tumor necrosis factor-α and interleukin-6, and enhance transforming growth factor-β-mediated fibrosis. These molecular events contribute to mesangial cell proliferation, podocyte injury, tubular epithelial cell apoptosis, extracellular matrix deposition, and progressive glomerulosclerosis, ultimately accelerating deterioration of renal function.[6-8]

Clinical investigations have shown a significant positive correlation between serum AOPP concentrations and CKD severity. Patients with declining estimated glomerular filtration

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rate (eGFR), increasing albuminuria, and advanced CKD stages consistently exhibit higher circulating AOPP levels than healthy individuals.[6-8] Moreover, AOPP concentrations are markedly elevated in patients undergoing maintenance hemodialysis because of persistent oxidative stress and reduced renal clearance. Several studies have further demonstrated associations between elevated AOPPs and endothelial dysfunction, vascular calcification, cardiovascular disease, muscle wasting, and increased mortality among CKD patients, suggesting that AOPPs may provide prognostic information beyond conventional renal biomarkers.[8]

Recent research has expanded the understanding of AOPPs beyond renal injury by highlighting their systemic effects. AOPPs have been implicated in CKD-associated skeletal muscle atrophy through activation of oxidative stress pathways and in adipose tissue inflammation through macrophage activation, emphasizing their role as circulating uremic toxins with widespread biological consequences.[8,9] These findings support the concept that oxidative protein modifications contribute not only to renal disease progression but also to many of the metabolic and cardiovascular complications responsible for increased morbidity and mortality in CKD patients.[9,10]

Although oxidative stress biomarkers such as malondialdehyde, advanced glycation end products, and 8-hydroxy-2′-deoxyguanosine have been investigated extensively, AOPPs possess unique advantages because they are relatively stable in circulation, can be measured using simple spectrophotometric assays, and closely reflect cumulative oxidative protein damage. Nevertheless, their clinical utility in predicting CKD severity and progression remains incompletely established, particularly in diverse populations where data remain limited. Further evaluation of the relationship between serum AOPP levels and CKD severity may improve risk stratification, facilitate earlier identification of patients at higher risk of progression, and support the development of targeted antioxidant therapeutic strategies. Therefore, the present study aims to evaluate serum Advanced Oxidation Protein Products as biomarkers of oxidative stress and to determine their association with the severity of chronic kidney disease.

MATERIALS AND METHODS

This analytical cross-sectional study was conducted at the Department of Biochemistry in collaboration with the Department of Nephrology of a tertiary care teaching hospital in Karachi, Pakistan. The study was carried out over a period of six months following approval from the Institutional Review Board (IRB). Written informed consent was obtained from all participants prior to enrollment, and all study procedures were performed in accordance with the ethical principles outlined in the Declaration of Helsinki.

Adult patients diagnosed with chronic kidney disease (CKD) attending the nephrology outpatient clinics or admitted to the nephrology wards were consecutively recruited. Chronic kidney disease was defined according to the Kidney Disease: Improving Global Outcomes (KDIGO) guidelines as abnormalities of kidney structure or function persisting for more than three months. Age- and sex-matched healthy volunteers with normal renal

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function and no history of chronic systemic illness were enrolled as controls. The sample size was calculated using the formula for comparison of means with a 95% confidence level and 80% study power. A non-probability consecutive sampling technique was employed to recruit eligible participants during the study period.

Patients aged 18–70 years with established CKD stages 2–5 were included in the study. CKD staging was based on estimated glomerular filtration rate (eGFR) calculated using the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) equation in accordance with KDIGO recommendations.

Patients with acute kidney injury, active infections, chronic inflammatory or autoimmune disorders, malignancy, chronic liver disease, pregnancy, recent surgery or trauma, and those receiving antioxidant supplementation or immunosuppressive therapy were excluded. Individuals with conditions known to influence oxidative stress independently of CKD were also excluded to minimize confounding.

A structured data collection form was used to record demographic and clinical characteristics, including age, sex, body mass index, smoking status, duration of CKD, presence of diabetes mellitus and hypertension, medication history, and dialysis status where applicable. Blood pressure measurements were obtained using a calibrated sphygmomanometer after participants had rested for at least five minutes in the seated position. Following an overnight fast of 8–10 hours, approximately 5 mL of venous blood was collected from each participant under aseptic conditions. Blood samples were allowed to clot at room temperature and subsequently centrifuged at 3000 rpm for 10 minutes to separate serum.

Serum aliquots intended for Advanced Oxidation Protein Products (AOPPs) estimation were stored at −80°C until analysis to prevent oxidative degradation. Routine biochemical investigations, including serum creatinine, blood urea nitrogen, albumin, calcium, phosphate, uric acid, and lipid profile, were measured using an automated clinical chemistry analyzer according to the manufacturer's instructions. Estimated glomerular filtration rate was calculated using the CKD-EPI equation, and patients were categorized into CKD stages based on KDIGO criteria.

Serum Advanced Oxidation Protein Products were quantified using a commercially available enzyme-linked immunosorbent assay (ELISA) kit according to the manufacturer's protocol. Briefly, serum samples and standards were added to pre-coated microplate wells, followed by incubation with enzyme-conjugated antibodies. After sequential washing steps, chromogenic substrate was added, and the reaction was terminated using stop solution. Optical density was measured at 450 nm using a microplate reader, and AOPP concentrations were calculated from the standard calibration curve. All samples were analyzed in duplicate, and both internal quality control sera and manufacturer-provided calibrators were included in each analytical run to ensure assay precision and reproducibility.

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The severity of chronic kidney disease was determined using the estimated glomerular filtration rate. Participants were classified into CKD stage 2 (eGFR 60–89 mL/min/1.73 m^2), stage 3 (30–59 mL/min/1.73 m^2), stage 4 (15–29 mL/min/1.73 m^2), and stage 5 (<15 mL/min/1.73 m^2). The relationship between serum AOPP concentrations and CKD severity was evaluated across these stages. Data were analyzed using the Statistical Package for the Social Sciences (SPSS) version 26.0 (IBM Corp., Armonk, NY, USA). Continuous variables were tested for normality using the Shapiro–Wilk test. Normally distributed variables were expressed as mean ± standard deviation (SD), whereas non-normally distributed variables were presented as median and interquartile range (IQR). Categorical variables were summarized as frequencies and percentages.

Comparisons of continuous variables among CKD stages were performed using one-way analysis of variance (ANOVA) followed by Tukey's post hoc test for normally distributed data, or the Kruskal–Wallis test with Dunn's post hoc analysis for non-parametric data. Differences between CKD patients and healthy controls were assessed using the independent-samples t-test or Mann–Whitney U test, as appropriate.

Associations between serum AOPP levels and renal function parameters, including serum creatinine and eGFR, were determined using Pearson's or Spearman's correlation coefficients depending on data distribution. Multiple linear regression analysis was performed to identify independent predictors of serum AOPP concentrations after adjustment for potential confounding variables, including age, sex, diabetes mellitus, hypertension, and body mass index. A two-tailed *p*-value <0.05 was considered statistically significant throughout the analysis.

RESULTS

A total of 150 participants were included in the study, comprising 120 patients with chronic kidney disease and 30 healthy controls. There were no significant differences between the groups regarding age, gender distribution, or body mass index ($p>0.05$). However, the prevalence of hypertension and diabetes mellitus was significantly higher among CKD patients than controls ($p<0.001$ and $p=0.002$, respectively).

Patients with CKD had significantly elevated serum creatinine and lower estimated glomerular filtration rate (eGFR) and serum albumin compared with healthy controls (all $p<0.001$). Serum Advanced Oxidation Protein Product (AOPP) levels were markedly higher in CKD patients than in controls (138.6 ± 39.8 vs. 56.3 ± 12.4 µmol/L, $p<0.001$).

When stratified according to CKD stage, serum AOPP concentrations increased progressively with disease severity, rising from 92.8 ± 18.6 µmol/L in stage 2 to 185.1 ± 30.2 µmol/L in stage 5 ($p<0.001$). Concurrently, serum creatinine and phosphate levels increased significantly, whereas eGFR and serum albumin declined across advancing CKD stages. Correlation analysis demonstrated that serum AOPP levels were positively associated with serum creatinine ($r=0.694$, $p<0.001$) and serum phosphate ($r=0.516$, $p<0.001$), while significant negative correlations were observed with eGFR ($r=-0.731$,

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p<0.001) and serum albumin (r=−0.408, p<0.001). Multiple linear regression analysis revealed that serum creatinine and eGFR were independent predictors of serum AOPP concentrations after adjustment for potential confounding variables (Adjusted R^2=0.60, p<0.001).

Table 1: Baseline Demographic and Clinical Characteristics of the Study Participants

Variable	Controls (n=30)	CKD Patients (n=120)	p-value
Age (years), Mean ± SD	49.8 ± 10.2	52.4 ± 11.6	0.281
Male, n (%)	18 (60.0)	74 (61.7)	0.862
BMI (kg/m²), Mean ± SD	24.8 ± 3.1	25.9 ± 4.0	0.176
Hypertension, n (%)	6 (20.0)	86 (71.7)	<0.001
Diabetes mellitus, n (%)	5 (16.7)	59 (49.2)	0.002
Serum Creatinine (mg/dL), Mean ± SD	0.89 ± 0.18	3.84 ± 1.92	<0.001
eGFR (mL/min/1.73 m²), Mean ± SD	98.6 ± 12.5	33.8 ± 18.9	<0.001
Serum Albumin (g/dL), Mean ± SD	4.28 ± 0.32	3.54 ± 0.51	<0.001
AOPPs (µmol/L), Mean ± SD	56.3 ± 12.4	138.6 ± 39.8	<0.001

Table 2: Comparison of Biochemical Parameters According to CKD Stage

Variable	Stage 2 (n=30)	Stage 3 (n=30)	Stage 4 (n=30)	Stage 5 (n=30)	p-value
Serum Creatinine (mg/dL)	1.42 ± 0.31	2.31 ± 0.52	4.08 ± 0.86	7.55 ± 1.64	<0.001
eGFR (mL/min/1.73 m²)	70.4 ± 8.2	43.5 ± 6.3	22.8 ± 3.8	11.2 ± 2.5	<0.001
Serum Albumin (g/dL)	3.92 ± 0.41	3.61 ± 0.39	3.38 ± 0.46	3.11 ± 0.53	<0.001
Serum Phosphate (mg/dL)	3.8 ± 0.6	4.4 ± 0.7	5.2 ± 0.8	6.1 ± 0.9	<0.001
AOPPs (µmol/L)	92.8 ± 18.6	124.6 ± 21.8	151.9 ± 27.4	185.1 ± 30.2	<0.001

Table 3: Correlation of Serum AOPPS with Renal Function Parameters

Variable	Correlation coefficient (r)	p-value
Age	0.112	0.174
Serum Creatinine	0.694	<0.001
eGFR	−0.731	<0.001
Serum Albumin	−0.408	<0.001
Serum Phosphate	0.516	<0.001
BMI	0.084	0.309

Table 4: Multiple Linear Regression Analysis for Predictors of Serum AOPP Levels

Variable	β Coefficient	Standard Error	Standardized β	p-value
Age	0.18	0.12	0.08	0.143
Male gender	2.14	3.68	0.04	0.561
Diabetes mellitus	6.72	3.14	0.15	0.034
Hypertension	3.58	3.47	0.07	0.302
Serum Creatinine	9.84	1.82	0.41	<0.001
eGFR	−1.28	0.24	−0.46	<0.001

Model statistics: R^2 = 0.62; Adjusted R^2 = 0.60; F = 38.4; $p < 0.001$.

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Table 5: Comparison of Serum AOPP Levels between Controls and CKD Stages

Group	AOPPs (µmol/L), Mean ± SD	Post hoc comparison
Controls	56.3 ± 12.4	Reference
CKD Stage 2	92.8 ± 18.6	$p < 0.001$ vs Controls
CKD Stage 3	124.6 ± 21.8	$p < 0.001$ vs Stage 2
CKD Stage 4	151.9 ± 27.4	$p = 0.002$ vs Stage 3
CKD Stage 5	185.1 ± 30.2	$p < 0.001$ vs Stage 4

DISCUSSION

The present study evaluated the relationship between serum Advanced Oxidation Protein Products (AOPPs) and the severity of chronic kidney disease (CKD). The findings demonstrated that serum AOPP concentrations were significantly higher in patients with CKD than in healthy controls and increased progressively with advancing CKD stage. Furthermore, AOPP levels showed a strong positive correlation with serum creatinine and serum phosphate, whereas a significant inverse correlation was observed with estimated glomerular filtration rate (eGFR). Multiple regression analysis further identified serum creatinine and eGFR as independent predictors of circulating AOPP levels, suggesting that oxidative protein damage intensifies as renal function deteriorates.

Oxidative stress is a well-established contributor to CKD progression through persistent generation of reactive oxygen species and impairment of endogenous antioxidant defense systems. Reduced renal clearance together with chronic inflammation promotes the accumulation of oxidatively modified proteins, including AOPPs, which act not only as biomarkers of oxidative stress but also as mediators of renal injury. Experimental studies have demonstrated that AOPPs activate NADPH oxidase, receptor for advanced glycation end products (RAGE), CD36, and nuclear factor-κB (NF-κB) signaling pathways, leading to increased production of inflammatory cytokines, tubular apoptosis, glomerulosclerosis, and interstitial fibrosis, thereby accelerating CKD progression.

In the present study, serum AOPP concentrations increased significantly from CKD stage 2 to stage 5, indicating a close association between oxidative stress and worsening renal impairment. These findings are consistent with those reported by Toualbi et al., who observed a gradual rise in AOPP levels with advancing CKD stage, with the highest concentrations among hemodialysis patients. Similarly, recent evidence demonstrated that AOPP concentrations exceeding 200 µmol/L accurately identified advanced CKD (stages 4–5), supporting their potential utility as a marker of disease severity.

A strong inverse relationship between serum AOPPs and eGFR was observed in this study, suggesting that oxidative protein modification increases as renal filtration declines. This observation agrees with previous clinical investigations reporting that declining kidney function results in both enhanced oxidative stress and reduced elimination of oxidized proteins. Consequently, accumulation of AOPPs further aggravates renal injury by promoting endothelial dysfunction, inflammatory cell recruitment, and activation of

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profibrotic signaling pathways. These findings reinforce the concept that oxidative stress is both a consequence and a driver of CKD progression.

Our regression analysis demonstrated that serum creatinine and eGFR remained independent determinants of AOPP concentrations after adjustment for demographic and clinical variables. This finding suggests that renal dysfunction itself is the principal factor influencing circulating AOPP levels rather than age or sex. Although diabetes mellitus showed a modest independent association with AOPPs, hypertension did not remain statistically significant after multivariable adjustment. These observations are biologically plausible because hyperglycemia enhances oxidative stress and protein oxidation, whereas declining renal clearance contributes substantially to AOPP accumulation. Recent reviews have similarly emphasized that oxidative biomarkers are closely linked with renal function and may improve risk stratification beyond conventional biochemical indices.

The progressive increase in serum phosphate accompanied by rising AOPP concentrations observed in the present study may also reflect worsening disturbances in mineral metabolism associated with advanced CKD. Hyperphosphatemia contributes to oxidative stress, endothelial dysfunction, and vascular calcification, while oxidative protein products further amplify inflammatory pathways involved in cardiovascular complications. Since cardiovascular disease remains the leading cause of mortality among CKD patients, the combined assessment of renal function and oxidative stress biomarkers such as AOPPs may provide additional prognostic information.

The findings of this study have important clinical implications. Measurement of serum AOPPs is relatively inexpensive, technically straightforward, and reflects cumulative oxidative protein damage. Therefore, AOPPs may serve as a useful adjunct biomarker for assessing CKD severity and identifying patients at increased risk of disease progression. Future prospective multicenter studies with larger sample sizes are warranted to establish standardized reference ranges, determine diagnostic cut-off values, and evaluate the prognostic value of AOPPs in predicting renal outcomes and cardiovascular events. In addition, interventional studies investigating antioxidant therapies targeting AOPP-mediated pathways may provide novel strategies to slow CKD progression and improve patient outcomes.

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