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The Role of Inflammatory Biomarkers in Assessing Chronic Kidney Disease Progression

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HIGHLIGHTS

1. Inflammatory biomarkers indicate kidney disease progression severity.
2. Biomarkers help predict chronic kidney disease complications.
3. Elevated biomarkers correlate with kidney function decline.
4. Biomarkers guide early intervertion strategies in CKD.
5. Monitoring inflammation aids CKD managemant and treatment.

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ABSTRACT

Background : Chronic Kidney Disease (CKD) is a progressive condition characterized by declining renal function and increased inflammation. Inflammatory markers, such as Adenosine Deaminase (ADA), C-Reactive Protein (CRP), and Erythrocyte Sedimentation Rate (ESR), play a significant role in monitoring disease progression. This study evaluates the relationship between these markers and kidney function across different stages of CKD **Methods :** This cross-sectional study involved 160 participants, including 80 CKD patients and 80 healthy controls. CKD patients were classified into stages 3, 4 and 5 based on eGFR levels. Blood samples were collected to measure ADA, CRP and ESR levels. EGFR was calculated using the MDRD equation. Statistical analysis, including ANOVA and Pearson correlation was performed to compare inflammatory markers between stages and assess their relationship with eGFR **Results:** ADA, CRP and ESR levels were significantly higher in CKD patients compared to controls. ADA and CRP levels showed a moderate inverse correlation with EGFR while ESR had the strongest inverse correlation ($r = -0.7$, $p < 0.001$). Multiple comparisons revealed significant differences in ADA and CRP between stages 3 and 5 ($p = 0.045$ and $p = 0.018$, respectively), while ESR showed significant differences across all stages. **Conclusion :** Inflammatory markers, particularly ESR, are closely associated with declining kidney function in CKD patients. These markers could serve as useful indicators of CKD progression, aiding in early detection and monitoring of disease severity.

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INTRODUCTION

Chronic Kidney Disease (CKD) is a progressive disorder marked by a sustained reduction in Glomerular Filtration Rate (GFR), which often leads to end-stage renal disease (ESRD) and other systemic complications, such as cardiovascular disease. Inflammation plays a significant role in CKD progression, with several biomarkers being linked to both the early detection and progression of the disease. Biomarkers such as C-Reactive Protein (CRP), Interleukin-6 (IL-6), and Tumor Necrosis Factor alpha (TNF-α) are central to assessing inflammation in CKD patients [1, 2]. Elevated levels of these markers correlate with a more rapid decline in kidney function, particularly in high-risk populations such as those with diabetes. Studies have demonstrated that biomarkers like TNFR1, TNFR2, and KIM-1 not only reflect glomerular damage but also non-glomerular injury, providing more comprehensive insight into CKD's trajectory [3]. Other research has highlighted that inflammatory biomarker, including NLR PLR and SII are significantly elevated in more advanced stages of CKD, further supporting their use in predicting disease progression. Ultimately, these biomarkers hold promise not only for tracking CKD progression but also for the development of targeted therapies to slow its course[4].

Chronic Kidney Disease (CKD) is a prevalent condition, affecting approximately 10-15% of the global population, and is frequently associated with multiple comorbidities that exacerbate its progression and patient outcomes. Hypertension diabetes and cardiovascular disease are the most common comorbidities observed in CKD patients, further complicating treatment and increasing the risk of adverse events such as kidney failure and cardiovascular incidents. Research indicates that over 98% of CKD patients have at least one comorbid condition, with many presenting with multiple conditions simultaneously, such as diabetes and heart failure, which significantly raise mortality risks [5]. Furthermore, in a Hungarian cohort study, the prevalence of CKD was found to be 14%, with cardiometabolic comorbidities such as hypertension (70.2%) and diabetes (41.5%) being particularly common [6]. Comorbidities like atrial fibrillation also play a crucial role in worsening kidney function, with 27% of patients with new-onset atrial fibrillation also diagnosed with CKD stages 3-5 [7]. These data emphasize the importance of managing CKD in conjunction with its comorbidities to mitigate the risks of progression and improve overall patient care.

In Chronic Kidney Disease (CKD), the decline in kidney function is closely associated with elevated levels of inflammatory biomarkers, including Adenosine Deaminase (ADA) Activity, C-Reactive Protein (CRP), and Erythrocyte Sedimentation Rate (ESR). As kidney function deteriorates, these biomarkers tend to increase, reflecting heightened systemic inflammation [8]. ADA, an enzyme involved in purine metabolism and the immune response, has been shown to rise in correlation with decreased Glomerular Filtration Rate (GFR), indicating its role in immune activation as kidney damage progresses [9]. CRP, an acute-phase protein produced in the liver in response to inflammation, is another important marker that increases significantly in CKD patients, particularly in advanced stages. Elevated CRP levels are associated with an increased risk of cardiovascular events and faster progression of CKD. ESR, a nonspecific marker of inflammation, also tends to rise as kidney function worsens, signifying the persistent inflammatory state in CKD patients [10]. Together, these biomarkers provide valuable insights into the inflammatory mechanisms at play in CKD, and their levels may serve as indicators of disease progression, helping to identify patients at higher risk for adverse outcomes and guiding potential therapeutic interventions aimed at reducing inflammation and slowing kidney function decline[11].

AIMS & OBJECTIVES

To assess the level of Adenosine Deaminase (ADA) activity in patients with Chronic Kidney Disease (CKD) and to analyze the correlation between ADA activity, C-Reactive Protein (CRP), Erythrocyte Sedimentation Rate (ESR), and different stages of CKD, based on estimated Glomerular Filtration Rate (eGFR).

MATERIALS AND METHODS

This prospective, cross-sectional study was conducted on 160 participants, with 80 diagnosed CKD patients and 80 healthy controls. The CKD patients were further categorized into stages 3, 4 and 5 based on their eGFR levels, using the Kidney Disease Outcomes Quality Initiative (KDOQI) guidelines. The study population was recruited from the nephrology department of a tertiary care hospital, and written informed consent was obtained from all participants before inclusion.

Inclusion Criteria:

- Patients aged 18 years and above.
- Diagnosed with CKD stages 3, 4 or 5 based on eGFR levels (<60 ml/min/1.73 m^2 for at least 3 months).

Exclusion Criteria

- Patients with acute kidney injury.
- History of recent infection or autoimmune disease.
- Patients on immunosuppressive therapy or cortic-osteroids.
- Pregnant women and patients with active malignancies.

Sample Collection

Venous blood samples were collected from all participants after an overnight fast. Serum levels of ADA, CRP, and ESR were measured using standard biochemical methods. ADA levels were determined using the colorimetric method, CRP levels were measured via immunoturbidimetric assay, and ESR was measured using the Westergren method.

eGFR Calculation

The eGFR was calculated using the Modification of Diet in Renal Disease (MDRD) equation:

eGFR = 175 * (Serum Creatinine)-1.154 *(Age)-0.203 *(0.742 if female)

Statistical Analysis

Data were analyzed using SPSS version 25 Continuous variables were expressed as mean ± Standard Deviation (SD), while categorical variables were expressed as percentages. Comparisons between groups were made using the independent t-test or ANOVA for continuous variables and the chisquare test for categorical variables. Pearson correlation analysis was conducted to assess the relationship between ADA, CRP, ESR and eGFR .

Multiple comparisons between CKD stages were made using post hoc tests and a p-value of <0.05 was considered statistically significant. This methodology was designed to evaluate the relationship between inflammatory markers (ADA, CRP and ESR) and kidney function, as indicated by eGFR in CKD patients across various stages.

RESULTS

The majority of cases (51.25%) fall in the 51–60 age group, followed by 36.25% in the 41–50 age group. The male participants make up a significant portion of the cohort (83.75%), while females account for 16.25%. The age distribution highlights that kidney disease incidence increases with age, particularly in the older groups. The largest portion of controls (33.75%) is in the 51–60 age group, followed by 27.5% in the 18–30 age group. The controls are evenly split by gender, with 50% male and 50% female participants. This even gender distribution contrasts with the case group, and the age spread indicates a more balanced representation of younger and older individuals in the control group. Highest number of participants in both the case and control groups falls within the 51–60 age range. Overall, the figure emphasizes the age-related trend of chronic kidney disease being more prevalent in older age groups.

Table 1:Distribution of Cases According to Age and Gender

Age (Years)	Male (n, %)	Female (n, %)	Total (n, %)
18-30	8 (10%)	4 (5%)	12 (15%)
31-40	8 (10%)	2 (2.5%)	10 (12.5%)
41-50	21 (26.25%)	8 (10%)	29 (36.25%)
51-60	30 (37.5%)	11 (13.75%)	41 (51.25%)
Total	67 (83.75%)	25 (16.25%)	80 (100%)

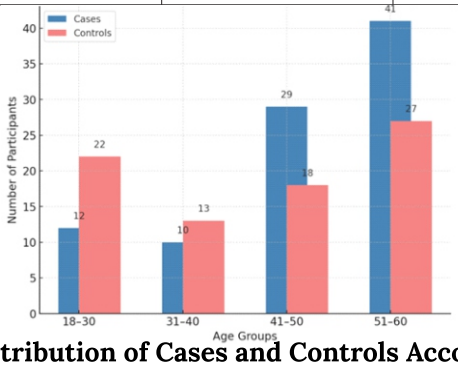

Figure 1: Distribution of Cases and Controls According to Age

Estimation of inflammatory markers in CKD patients

The study results reveal significant differences between cases and controls across key biochemical markers, highlighting the impact of Chronic Kidney Disease (CKD) on inflammation and kidney function. Creatinine levels were markedly higher in the case group (5.5 ± 4.3 mg/dl) compared to controls (0.95 ± 0.32 mg/dl), with a p-value of <0.001, indicating severe impairment in renal function among CKD patients. Similarly, Adenosine Deaminase (ADA) Activity, an inflammatory marker, was significantly (P<0.001) elevated in the case group (19.2 ± 9.0 U/L) compared to controls (7.8 ± 2.4 U/L)

Table 2: Distribution of Control According to Age and Gender

Age (Years)	Male (n, %)	Female (n, %)	Total (n, %)
18-30	10(12.5%)	12 (15%)	22 (27.5%)
31-40	5 (6.25%)	8 (10%)	13 (16.25%)
41-50	10 (12.5%)	8 (10%)	18 (22.5%)
51-60	15 (18.75%)	12 (15%)	27 (33.75%)

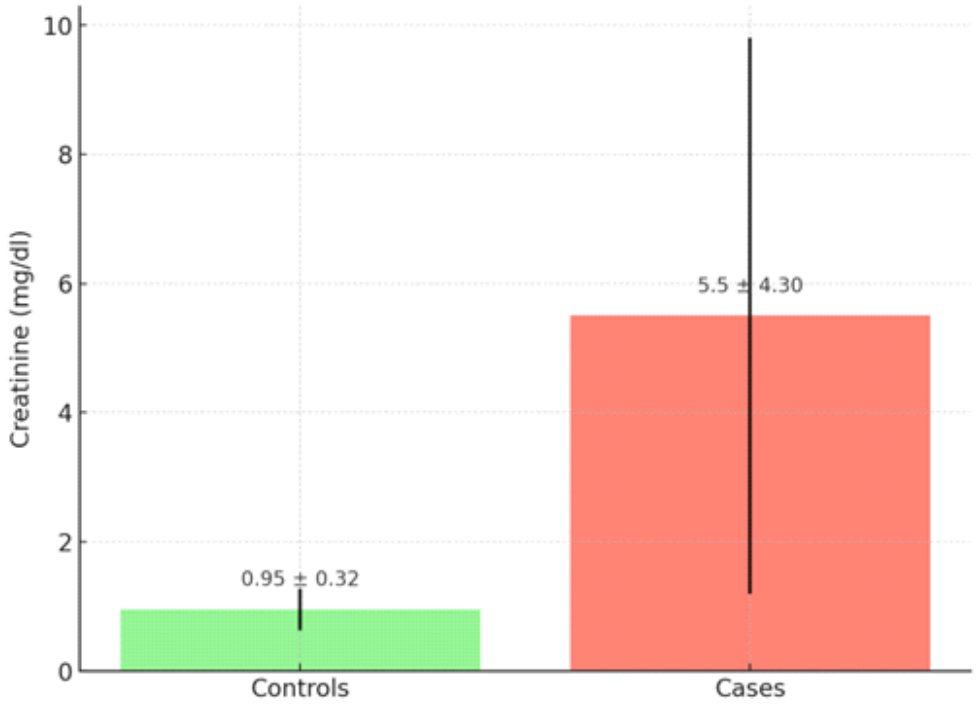

Figure 2: Comparison of Creatinine Between Cases and Controls

Table 3: Comparison of Biochemical Parameter

Biochemical Parameters	Controls Mean ± SD	Cases Mean ± SD	P-value
ADA (U/L)	7.8 ± 2.4	19.2 ± 9.0	<0.001
Creatinine (mg/dl)	0.95 ± 0.32	5.5 ± 4.3	<0.001

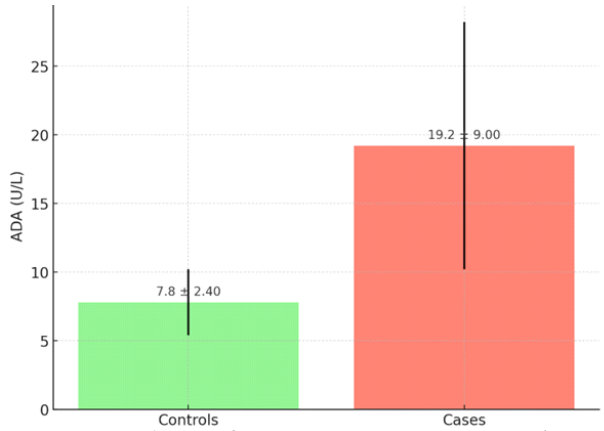

Figure 3: Comparison of ADA Between Cases and Controls

Role of ADA, ESR and CRP in CKD

Compares the levels of Adenosine Deaminase (ADA), C-Reactive Protein (CRP), and Erythrocyte Sedimentation Rate (ESR) across different stages of Chronic Kidney Disease (CKD). As CKD progresses from stage 3 to stage 5, there is a clear increase in the mean levels of these inflammatory markers. ADA levels rise from 17.03 ± 4.15 U/L in stage 3 to 21.08 ± 13.74 U/L in stage 5. CRP increases from 30.05 ± 7.12 mg/L in stage 3 to 45.28 ± 23.83 mg/L in stage 5, while ESR climbs from 28.12 ± 4.03 mm/h in stage 3 to 54.02 ± 14.42 mm/h in stage 5. The progression of CKD is associated with a statistically significant increase in ESR ($p = 0.003$), while the increases in ADA ($p = 0.286$) and CRP ($p = 0.702$) are not statistically significant. This indicates that ESR is a more reliable marker for tracking inflammation in advancing CKD.

Table 4: Comparison of ADA, CRP and ESR Between Different Stages of CKD

Biochemical Parameters	Stage 3 (Mean ± SD)	Stage 4 (Mean ± SD)	Stage 5 (Mean ± SD)	P-value
ADA	17.03 ± 4.15	19.48± 6.07	21.08 ± 13.74	0.286
CRP	30.05 ± 7.12	33.15 ± 12.95	45.28 ± 23.83	0.702
ESR	28.12 ± 4.03	35.05 ± 8.87	54.02 ± 14.42	0.003

ADA: Adenosine deaminase (mg/dl), CRP: C-reactive protein (mg/dl), ESR: Erythrocyte sedimentation rate (mm/hour) (N- 0-22 mm/h for men and 0-29 mm/h for women)

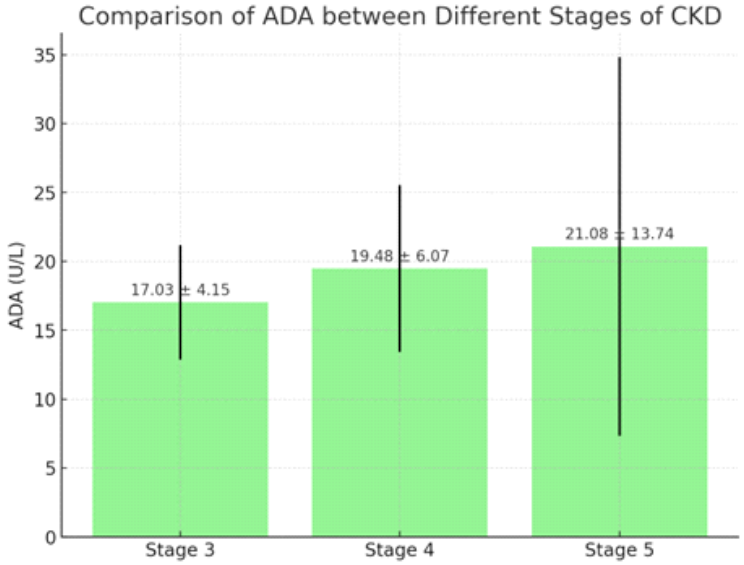

Figure 4: Comparison of ADA Between Different Stages of CKD

Correlation of ADA and eGFR biochemical markers

The correlation of ADA with AGE ,Weight, Creatinine & ESR. The correlation analysis between ADA levels and various biochemical parameters indicate significant and non-significant associations. Age and weight both show weak negative corre-lations with ADA, with r-values of -0.078 and -0.05, respectively, and their p-values (0.57 for age and 0.71 for weight) indicate no statistical significance.

Creatinine demonstrates a moderate positive correlation with ADA (r = 0.24), but this relationship is not statistically significant (p = 0.068). However, ESR shows a stronger positive correlation with ADA ® = 0.35) and reaches statistical significance with a p-value of 0.006. This suggests that ADA levels are significantly associated with ESR, an important marker of inflammation, in chronic kidney disease patients.

Table 5: Pearson Correlation Coefficient (r-value) of ADA with Various Biochemical Parameters (age, weight, Creatinine & ESR)

Biochemical Parameters	Correlation with ADA (r-value)	P-value
AGE (years)	-0.078	0.57
WEIGHT (kg)	-0.05	0.71
CREATININE (mg/dl)	0.24	0.068
ESR (mm/hour)	0.35	0.006

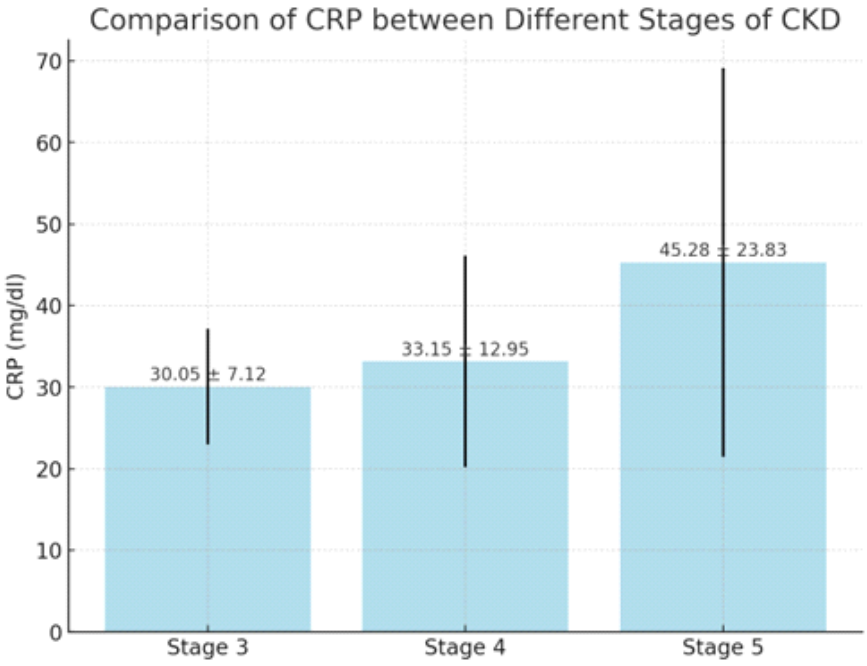

Figure 5: Comparison of CRP Between Different Stages of CKD

the correlation of eGFR with ADA, CRP and ESR. ADA demonstrates a weak negative correlation with eGFR (r = -0.21) that is not statistically sign-ificant (p = 0.11). CRP also shows a moderate negative correlation with eGFR (r = -0.32), and this relat-ionship is statistically significant (p = 0.012), indic-ating that lower eGFR levels are associated with higher CRP reflecting inc-reased inflammation. ESR exhibits the strongest negative correlation with eGFR (r = -0.70), with high statistical significance (p = 0.001). This suggests that ESR is highly predictive of decreased kidney function, as lower eGFR is associated with sign-ificantly elevated ESR, a key inflammatory marker in chronic kidney disease.

Table 6: Pearson Correlation Coefficient (r-value) of eGFR with Various Common Parameters (ADA, CRP, ESR)

Biochemical Prameters parameters	Correlation with eGFR (r-value)	P-value
ADA	-0.21	0.11
CRP	-0.32	0.012
ESR	-0.7	0.001

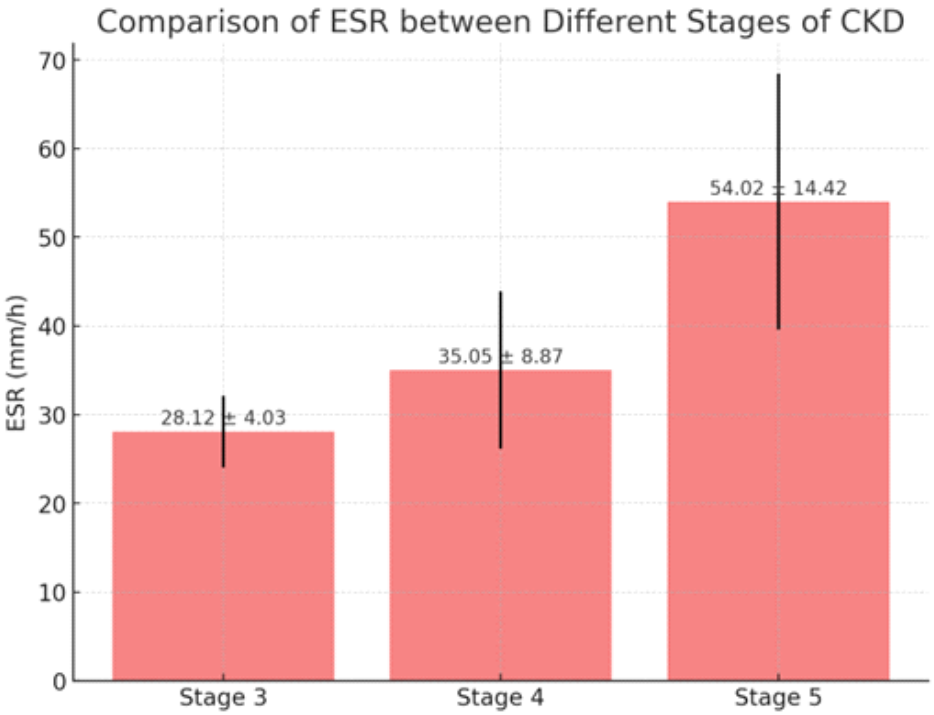

Figure 6: Comparison of ESR between Different Stages of CKD

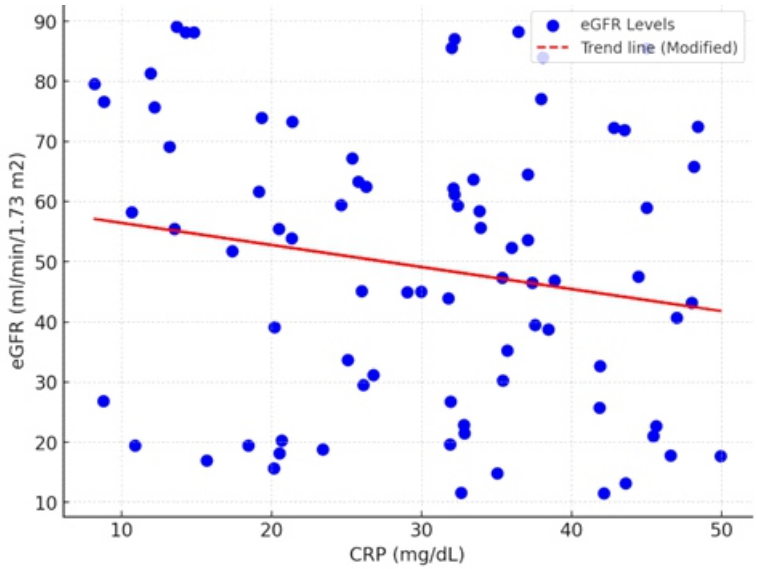

Figure 7: Shows an Inverse Relationship Between CRP and eGFR, where Higher CRP Levels are Associated with Worsening Kidney Function.

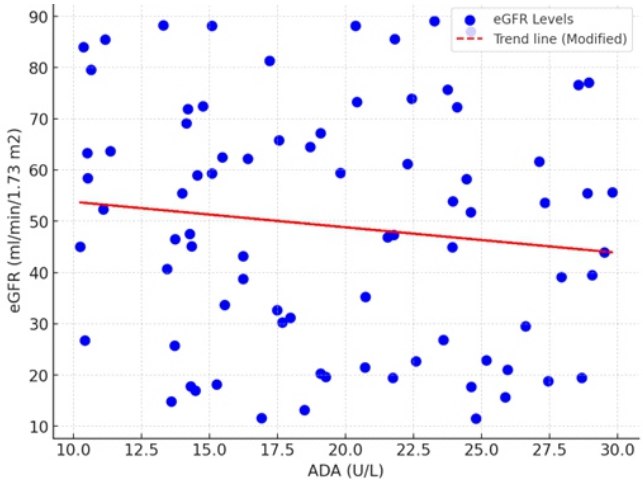

Figure 8 : Correlation of ADA with eGFR. ADA: Adenosine Deaminase, eGFR: Estimated Glomerular filtrationrate

Presents the strongest correlation, with an r-value of -0.7 between ESR and eGFR. As ESR increases, eGFR sharply decreases, highlighting ESR as a significant realism. These figures collectively demonstrate how inflammation is closely tied to the progression of chronic kidney disease.

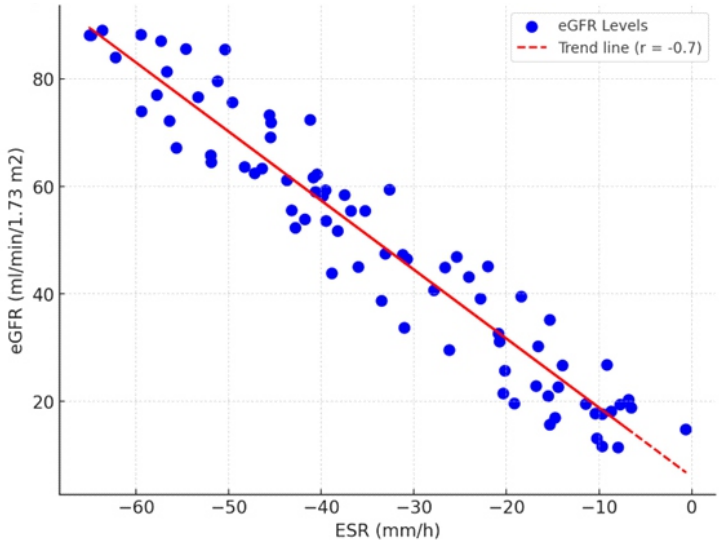

Figure 9: Correlation of ESR with eGFR ESR Erythrocyte Sedimentation Rate eGFR Estimated Glomerular Filtration rate

Multiple comparisons of ADA, CRP and ESR between different stages of CKD

Presents the multiple comparisons of ADA levels between different stages of CKD. The analysis shows a significant difference in ADA levels between Stage 3 and Stage 5 (p = 0.045), suggesting a marked increase in ADA as CKD progresses to later stages. However, the differences between Stage 3 and Stage 4 (p = 0.286) and between Stage 4 and Stage 5 (p = 0.532) are not statistically significant, indicating that ADA levels show more pronounced changes only between the early and later stages of CKD.

Table 7: Multiple Comparisons of ADA Between Different Stages of CKD

Stage Comparisons	Mean Difference	P-value
Stage 3 vs Stage 4	-2.45	0.286
Stage 3 vs Stage 5	-4.05	0.045
Stage 4 vs Stage 5	-1.6	0.532

highlights the multiple comparisons of CRP levels between different CKD stages. The results show a significant increase in CRP between Stage 3 and Stage 5 (p = 0.018), while the comparisons between Stage 3 and Stage 4 (p = 0.540) and Stage 4 and Stage 5 (p = 0.089) do not reach statistical significance. This suggests that CRP levels escalate significantly only between the earlier and later stages of CKD.

Table 8: Multiple comparison of CRP between different stages of CKD

Stage Comparisons	Mean Difference	P-value
Stage 3 vs Stage 4	-3.1	0.54
Stage 3 vs Stage 5	-15.23	0.018
Stage 4 vs Stage 5	-12.13	0.089

Presents the multiple comparisons of ESR levels across different CKD stages, with all comparisons showing statistically significant differences. The greatest mean difference is between Stage 3 and Stage 5 (p = 0.001), followed by Stage 4 and Stage 5 (p 0.015) and Stage 3 and Stage 4 (p = 0.034). This indicates that ESR is a highly sensitive marker, with levels increasing progressively as CKD severity worsens, making it a valuable indicator of disease progression.

Table 9: Multiple Comparison of ESR Between Different Stages of CKD

Stage Comparisons	Mean Difference	P-value
Stage 3 vs Stage 4	6.93	0.034
Stage 3 vs Stage 5	25.9	0.001
Stage 4 vs Stage 5	18.97	0.015

DISSCUSSION

Chronic Kidney Disease (CKD) is increasingly recognized as a systemic disorder marked by persistent inflammation, contributing to renal function decline and heightened cardiovascular risk. The present stu-dy provides critical insights into the role of inflam-matory biomarkers Aden-osine Deaminase (ADA), C-Reactive Protein (CRP), and Erythrocyte Sedimentation Rate (ESR) as indicators of CKD progression. The findings align with current research, high lighting how immune activation and systemic inflamma-tion exacerbate kidney damage and impact overall patient outcomes[12,13].

Increased ADA levels, which reflect immune system activation, were significantly higher in CKD patients compared to healthy controls, supporting its role in chronic inflammation. This result mirrors studies showing that ADA is not only a marker of immune activation but is also linked to worse renal out comes particularly in CKD stages 4 and 5 [14]. Similarly, CRP a well established acute-phase protein produced by the liver in response to inflammation showed a storng inverse correlation with eGFR, corroborating the understanding that elevated CRP levels predict worse CKD outcomes and are associated with cardiovascular risks . These findings are crucial because cardiovascular disease is the leading cause of death in CKD patients, and CRP has been shown to predict cardiov-ascular mortality even at early stages of renal impairment [7].

The study's finding that ESR had the strongest inverse correlation with eGFR (r = -0.7, p < 0.001) is consistent with other research that positions ESR as a robust marker for systemic inflammation in CKD [18]. Elevated ESR levels are particularly relevant in chronic diseases as it is a nonspecific marker of inflammation, reflecting the ongoing inflammatory processes that accelerate CKD progression. This highlights the importance of early intervention to mitigate inflammation and reduce the risk of complications like cardiovascular disease, which are prevalent among CKD patients[19,21].

These inflammatory markers are not only useful for tracking disease progression but also for identifying patients at high risk for adverse outcomes. Elevated-levels of ADA, CRP and ESR indicate heightened imm-

-une and inflammatory activity, which is central to the pathophysiology of CKD. For example, ADA is involved in purine metabolism and immune modulation, and its elevated levels in CKD patients suggest increased immune system activation as kidney function declines. Similar patterns have been observed in studies examining CKD progression in patients with comorbidities like diabetes, where increased ADA, CRP, and ESR were linked to accelerated renal deterioration.

Furthermore the association between CRP levels and declining kidney function in this study is consistent with other. Given that CKD patients are more likely to die from cardiovascular events than progress to end-stage renal disease, the relationship between elevated CRP levels and cardiovascular complications is clinically significant. This underscores the importance of regular monitoring of CRP levels to identify high-risk patients who may benefit from aggressive cardiovascular prevention strategies.

In summary, the present study affirms the utility of ADA, CRP, and ESR as key biomarkers for assessing CKD progression and highlights the potential of these markers to inform clinical decision-making. The significant correlations between these markers and eGFR suggest their value in both predicting CKD progression and guiding treatment interventions aimed at reducing inflammation and mitigating cardiovascular risk. Further studies should focus on the longitudinal use of these markers in CKD management particularly in the context of comorbid conditions such as diabetes and hypertension, which complicate CKD outcomes [24]

CONCLUSION

Based on the findings of this study, it is evident that inflammatory biomarkers, particularly Adenosine Deaminase (ADA), C-Reactive Protein (CRP) and Erythrocyte Sedimentation Rate (ESR) are significantly elevated in patients with Chronic Kidney Disease (CKD) compared to healthy controls. These markers exhibit a clear relationship with the progression of CKD as their levels tend to increase with advancing disease stages. Among these, ESR showed the strongest correlation with the decline in kidney function, as measured by the estimated glomerular filtration rate (eGFR). This highlights ESR as a particularly valuable marker for assessing systemic inflammation and CKD progression.

Furthermore, the study demonstrated that while ADA and CRP levels also increased with disease severity, their correlation with CKD stages was less pronounced than that of ESR. ADA, which is involved in purine metabolism and immune modulation, had a moderate inverse correlation with eGFR, suggesting its role in immune activation in response to kidney damage. CRP a well known marker of inflammation also showed a significant inverse correlation with eGFR underlining its importance in monitoring systemic inflammation in CKD patients. The results confirm that these inflammatory markers can serve as useful tools for identifying patients at risk for rapid disease progression and for guiding therapeutic interventions.In conclusion, this study reinforces the critical role of inflammation in CKD progression and suggests that monitoring ADA, CRP and especially ESR could provide valuable insights into disease severity. Early detection and consistent monitoring of these biomarkers could improve the management of CKD by enabling timely interventions aimed at reducing inflammation and slowing the deterioration of kidney function. Future research should explore the longitudinal application of these markers and evaluate their potential in guiding personalized treatment strategies for patients with CKD, particularly those with comorbid conditions like diabetes and cardiovascular disease.

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