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Stage-wise comparison of demographic characteristics and serum biochemical parameters in chronic kidney disease; a cross-sectional study

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ABSTRACT

Introduction: Chronic kidney disease (CKD) is a progressive condition marked by declining glomerular filtration and disturbances in biochemical and electrolyte profiles; identifying stage-specific changes in demographics and serum markers may improve early detection and guide stage-appropriate management.

Objectives: This study compared demographic characteristics and serum biochemical parameters across stages 1–3 in patients with CKD to identify stage-related differences associated with disease progression.

Patients and Methods: This cross-sectional study enrolled 75 patients with early CKD (stage 1: n=24, stage 2: n=25, stage 3: n=26) attending specialist clinics at Al-Fayhaa teaching hospital, Basra, Iraq (Feb–May 2025). Demographic data (age, sex) and fasting venous blood samples were collected for routine renal tests (creatinine, urea, estimated glomerular filtration rate [eGFR]), uric acid, total protein, albumin, total cholesterol, electrolytes (sodium [Na], potassium [K], chloride [Cl], magnesium [Mg], phosphate), and biomarkers such as neutrophil gelatinase-associated lipocalin (NGAL) and Cathepsin D. Group comparisons across CKD stages plus correlation analyses were performed to assess associations with disease progression.

Results: The results indicated that CKD progression was associated with male gender and older age, across with changes in serum electrolytes, kidney function tests, and biochemical parameters, including increasing creatinine, urea, uric acid, sodium, K, cholesterol, NGAL, and cathepsin D. The CKD progression also decreased eGFR, Mg, and total protein ($P < 0.05$), with no significant impact on albumin, phosphate, and Cl ($P > 0.05$).

Conclusion: CKD progression is associated with male gender, older age, and biochemical changes, including increased renal markers, lipids, sodium, K, NGAL, and cathepsin D, as well as decreased eGFR, Mg, and total protein. Albumin, phosphate, and Cl remained stable, demonstrating the complexity of CKD and the potential of new biomarkers.

Implication for health policy/practice/research/medical education:
In this study we found that chronic kidney disease (CKD) progression is closely linked to demographic factors such as male gender and older age, and is characterized by significant biochemical disturbances, including elevations in conventional renal markers, electrolytes, lipids, and emerging biomarkers like neutrophil gelatinase-associated lipocalin (NGAL) and cathepsin D, alongside reductions in estimated glomerular filtration rate (eGFR), magnesium, and total protein, while some parameters such as albumin, phosphate, and chloride remain unaffected, highlighting both the complexity of CKD-related metabolic changes and the potential value of novel biomarkers in disease monitoring.

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Original Article

Introduction

Kidney diseases embody a spectrum of disorders targeting the kidneys, organs entrusted with the critical role of purifying the blood by filtering waste products, excess nutrients, and surplus fluids. In light of the pervasive and variegated nature of kidney diseases, a foundational understanding becomes imperative, forming the cornerstone for strategizing effective preventative and management measures (1). This understanding, thus, establishes the grounding premise of this study, which

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ventures to unravel the intricate interplays between age, gender, and renal health parameters across diverse disease stages. Through these endeavours, we aspire to enrich the existing comprehension, paving the way for diagnostic and therapeutic advancements in the future (2). In pursuing advancements in the medical sphere, delineating the intricate web of connections between demographic determinants and health parameters emerges as a cornerstone. Especially, age and gender stand as pivotal demographic elements that have a bearing on the onset and trajectory of kidney diseases. In this segment, we probe deeply into the necessity of exploring the nexus between age, gender, and renal health parameters (3).

Understanding the relations between age and gender with renal health parameters sets the stage for individualized treatment strategies. It empowers clinicians to tailor treatments cognizant of these relationships, thereby fine-tuning therapeutic approaches for every patient (4). Moreover, it paves the way for risk stratification, wherein a deep comprehension of these correlations assists in pinpointing individuals at a higher risk, thereby laying grounds for pre-emptive measures aimed at forestalling the emergence or exacerbation of kidney ailments (5). Venturing into the correlations of age, gender, and renal health attributes grants a lens to discern the patterns dictating disease prevalence and incidence among varied demographic cohorts, thus enriching epidemiological explorations. It becomes an invaluable tool for healthcare planning, offering policymakers and healthcare dispensers a knowledge base to steer resource allocation wisely and to carve out healthcare agendas attuned to specific needs. A meticulous scrutiny of the ties interlinking age, gender, and renal health facets could unearth the underlying pathophysiological dynamics at play in kidney diseases, fostering a richer scientific understanding and potentially unveiling targets for novel therapeutic strategies (6).

Furthermore, it could spearhead the identification of novel biomarkers for renal diseases, thereby enhancing the precision in diagnostics and prognostics. A discerning understanding of how age and gender modulate renal health parameters can catalyze strategies for early detection, thus opening avenues for timely interventions capable of arresting or even reversing the disease progression. It fortifies the clinician's arsenal (6), enabling more accurate prognostic assessments, which in turn aid in framing realistic therapeutic objectives and expectations. In steering this research, we aspire to craft a meticulous analysis that does not just shed light on the existing correlations but also fosters a groundwork for ensuing studies. An endeavor directed towards nurturing a deeper comprehension and facilitating strides in refining patient care in the renal health landscape (7). The landscape of kidney disease is markedly varied, with patients experiencing different symptoms and outcomes based largely on the stage of their disease. Early-stage kidney diseases might present minor symptoms and can often be managed with lifestyle adjustments and medication (8). In contrast, later stages can involve serious complications, necessitating more intensive interventions, including dialysis or kidney transplantation. Focusing on different disease stages in this study offers a granular perspective on the trajectory of kidney diseases, facilitating a nuanced understanding of the alterations in renal health parameters as the disease progresses (9). It allows for the identification of critical intervention points where therapeutic actions could be most beneficial. Furthermore, delineating the disease into distinct stages offers the potential to uncover stage-specific markers, which could be pivotal in the early diagnosis and monitoring of disease progression (10).

Objectives

This study aimed to compare demographic characteristics (age, sex) and a panel of serum biochemical parameters including renal function tests (creatinine, urea, estimated glomerular filtration rate [eGFR]), uric acid, total protein, albumin, total cholesterol, electrolytes (sodium [Na], potassium [K], chloride [Cl], magnesium [Mg], phosphate) and biomarkers (neutrophil gelatinase-associated lipocalin [NGAL], Cathepsin D) across stages 1–3 of early chronic kidney disease (CKD), and to evaluate stage-related trends and associations using correlation and regression analyses to determine which measures best discriminate disease stage and predict progression.

Materials and Methods

Study design and participants

This observational, cross-sectional comparative study was conducted on 75 CKD patients with different stages of early CKD, including stage 1 (n = 24), stage 2 (n = 25), and stage 3 (n = 26), referred to the specialist clinics at Basra city, Al-Fayhaa teaching hospital, Iraq, between February and May 2025.

Inclusion and exclusion criteria

Eligible participants were adults aged 18 – 80 years with a clinical diagnosis of early CKD defined as stages 1–3 by eGFR and/or albuminuria, stable renal function over the preceding three months, and the ability to provide informed consent. Exclusion criteria comprised participants with incomplete data for laboratory analysis and those who were unwilling to continue the study.

Group classification

The GFR was estimated using the modification of the diet in renal disease (MDRD) equation. Patients were categorized according to kidney disease improving global

outcomes (KDIGO) staging system for CKD: stage 1 (S1) denoted kidney damage with normal or increased eGFR (≥90 mL/min/1.73 m²), stage 2 (S2) denoted mild reduction in eGFR (60–89 mL/min/1.73 m²), and stage 3 (S3) denoted moderate decrease in eGFR (30–59 mL/min/1.73 m²) (11).

Data collection and laboratory assessment

At enrollment, all participants provided written informed consent, and basic demographic data (age, sex) were recorded; fasting venous blood samples were then obtained from each patient and processed in the central laboratory for biochemical and biomarker analyses. Routine renal function tests included serum urea, creatinine, and eGFR, as well as uric acid, serum proteins, albumin, total cholesterol, and electrolytes (Na, K, Cl, and Mg). Specific biomarkers measured were NGAL and Cathepsin D. Laboratory results were entered into the study database and compared across the three CKD stage groups.

Outcome measurement

The primary outcome was the comparison across three CKD stage groups (stages 1, 2, and 3) of demographic variables (age, sex) and laboratory parameters, including routine renal function tests (serum urea, creatinine, and eGFR), uric acid, total protein and albumin, total cholesterol, electrolytes (Na, K, Cl, Mg), and the specific biomarkers NGAL and cathepsin D. The secondary outcome assessed the relationships between CKD stage progression and the aforementioned demographic characteristics, biochemical measures, and biomarker levels using appropriate correlation and regression analyses.

Statistical analysis

Data were analyzed in SPSS version 27 (IBM Corp., Armonk, NY, USA). Continuous variables are presented as mean ± standard deviation (SD) and categorical variables as frequency (%); normality was assessed with the Shapiro–Wilk and homogeneity of variances with Levene's test. Quantitative variables were compared across CKD stages by one-way analysis of Variance (ANOVA) with least significant difference (LSD) post hoc pairwise comparisons. Categorical comparisons used chi-square tests, and associations between gender and CKD stage were assessed with binary logistic regression to provide odds ratios (ORs) and 95% confidence intervals (CIs). Missing data were handled by listwise exclusion for each analysis, outliers were inspected by boxplots, and a two-sided $P < 0.05$ indicated statistical significance.

Results

The results indicated that early-stage CKD participants were predominantly female, while later-stage participants showed a shift toward male predominance; statistical modeling indicated that male gender was significantly more likely in the most advanced stage compared with the earlier stages, whereas the difference between the first two stages was not significant. Age also differed across stages, with participants in the middle and advanced stages being older on average than those in the earliest stage; overall group differences were statistically significant, and pairwise post hoc tests confirmed significant age increases for the middle and advanced stages compared with the earliest stage, while the age difference between the middle and advanced stages did not reach statistical significance (Table 1).

The results indicated that serum creatinine showed a progressive increase from CKD stage 1 to stage 3, with statistically significant differences across stages and clear pairwise separations between each stage from 1 to 3. Blood urea similarly rose with disease severity, with the most pronounced elevation in advanced disease, and significant differences when stage 3 and 2 were compared with stage 1, while the difference between stage 1 and 2 was not statistically significant. The eGFR declined markedly as the disease progressed, with highly significant differences across stages and significant pairwise declines between each successive stage. Serum uric acid was comparable between stages 1 and 2, but was substantially higher in advanced disease, producing significant overall and pairwise differences when stage 3 and 2 were compared with stage 1 (Table 2).

The comparative analysis of serum electrolyte concentrations across stages of CKD demonstrated that sodium levels were higher in more advanced stages, with significant differences between stage 1 and both stages 2 and 3, while the difference between the two more advanced stages, such as stages 2 and 3, was not significant. Potassium increased with disease progression, reaching notably higher concentrations in stage 3 and showing significant pairwise differences when compared with stages 1 and 2, but not between stages 1 and 2. Chloride values did not differ significantly across stages. Magnesium concentrations declined with advancing disease and were significantly lower in stage 3 compared with earlier stages, while differences between stages 1 and 2 were not significant. Serum phosphate showed no significant variation across disease stages (Table 3).

Across CKD stages, serum cholesterol was significantly elevated in the advanced stages of 2 and 3 compared to stage 1; however, no significant difference was observed between them. Total serum protein was significantly lower in stages 2 and 3 compared to stage 1, while remaining similar between them. Serum albumin did not differ significantly across CKD stages. The NGAL

Table 1. Comparative analysis of demographic characteristics among patients with different CKD stages

CKD stage		Frequency	Percent	*P* value*
Gender				
Stage 1 (n = 24)	Female	18	75	0.002
	Male	6	25	
Stage 2 (n = 26)	Female	21	80.8	
	Male	5	19.2	
Stage 3 (n = 25)	Female	9	36	
	Male	16	64	
Between CKD stage comparison		**OR (95% CI)**		***P* value****
Male gender vs female	Stage 2 vs 1	0.71 (0.18–2.73)		0.624
	Stage 3 vs 1	5.33 (1.55–18.30)		0.008
	Stage 3 vs 2	7.46 (2.09–26.64)		0.002
CKD stage		**Mean**	**SD**	***P* value*****
Age (year)				
Stage 1		37.21	14.87	<0.001
Stage 2		63.19	14.60	
Stage 3		56.68	13.28	
Between CKD stage comparison		**Mean difference (95% CI)**		***P* value******
Stage 2 vs 1		25.98 (17.93–34.03)		<0.001
Stage 3 vs 1		19.47 (11.34–27.60)		<0.001
Stage 3 vs 2		6.51 (-1.45–14.48)		0.108

CKD: Chronic kidney disease; OR: Odds ratio; CI: Confidence interval; SD: Standard deviation.
*Chi-square, **Binary logistic regression, ***One-way ANOVA, ****Post hoc LSD.

Table 2. Comparative analysis of kidney function tests and uric acid level among patients with different CKD stages

	CKD stage	Mean	SD	*P* value*
Creatinine (mg/dL)	Stage 1	0.56	0.10	<0.001
	Stage 2	0.73	0.20	
	Stage 3	1.73	0.45	
	Between CKD stage comparison	**Mean difference (95% CI)**		***P* value****
	Stage 2 vs 1	0.17 (0.01–0.034)		0.039
	Stage 3 vs 1	1.17 (1.00–1.33)		<0.001
	Stage 3 vs 2	0.99 (0.82–1.15)		<0.001
Urea (mg/dL)	**CKD stage**	**Mean**	**SD**	***P* value***
	Stage 1	22.95	5.54	<0.001
	Stage 2	31.57	11.10	
	Stage 3	78.49	25.57	
	Between CKD stage comparison	**Mean difference (95% CI)**		***P* value****
	Stage 2 vs 1	8.62 (-0.66–17.90)		0.068
	Stage 3 vs 1	55.54 (46.16–64.90)		<0.001
	Stage 3 vs 2	49.92 (37.73–56.10)		<0.001
eGFR (mL/ min/1.73 m²)	**CKD stage**	**Mean**	**SD**	***P* value***
	Stage 1	161.58	45.71	<0.001
	Stage 2	74.88	8.90	
	Stage 3	46.16	5.30	
	Between CKD stage comparison	**Mean difference (95% CI)**		***P* value****
	Stage 2 vs 1	-86.69 (-101.67 – -71.72)		<0.001
	Stage 3 vs 1	-115.42 (-130.54–[-100.30])		<0.001
	Stage 3 vs 2	-28.72 (-43.54–[-13.90])		<0.001
UA (mg/dL)	**CKD stage**	**Mean**	**SD**	***P* value***
	Stage 1	4.46	0.67	<0.001
	Stage 2	4.16	0.76	
	Stage 3	6.14	0.59	
	Between CKD stage comparison	**Mean difference (95% CI)**		***P* value****
	Stage 2 vs 1	-0.30 (-0.69–0.08)		0.119
	Stage 3 vs 1	1.67 (1.28–2.06)		<0.001
	Stage 3 vs 2	1.98 (1.59–2.36)		<0.001

CKD: Chronic kidney disease; eGFR: estimated glomerular filtration rate; UA: uric acid; SD: Standard deviation; CI: Confidence interval. * One-way ANOVA, **Post hoc LSD.

Table 3. Comparative analysis of serum electrolytes among patients with different CKD stages

Na (mEq/L)	**CKD stage**	**Mean**	**SD**	***P* value***
	Stage 1	139.58	4.49	
	Stage 2	145.73	6.27	0.002
	Stage 3	146.20	5.83	
	Between CKD stage comparison	**Mean difference (95% CI)**		***P* value****
	Stage 2 vs 1	6.15 (2.98 – 9.31)		<0.001
	Stage 3 vs 1	6.61 (3.42 – 9.81)		<0.001
	Stage 3 vs 2	0.46 (-2.66 – 3.59)		0.766
K (mEq/L)	**CKD stage**	**Mean**	**SD**	***P* value***
	Stage 1	3.87	0.33	
	Stage 2	3.98	0.47	<0.001
	Stage 3	4.90	0.61	
	Between CKD stage comparison	**Mean difference (95% CI)**		***P* value****
	Stage 2 vs 1	0.10 (-17 – 0.37)		0.460
	Stage 3 vs 1	1.03 (0.75 – 1.30)		<0.001
	Stage 3 vs 2	0.93 (0.65 – 1.20)		<0.001
Cl (mmol/L)	**CKD stage**	**Mean**	**SD**	***P* value***
	Stage 1	102.87	3.94	
	Stage 2	105.76	10.50	0.267
	Stage 3	103.45	5.96	
Mg (mg/dL)	**CKD stage**	**Mean**	**SD**	***P* value***
	Stage 1	3.05	0.92	
	Stage 2	3.01	0.66	0.027
	Stage 3	2.53	0.64	
	Between CKD stage comparison	**Mean difference (95% CI)**		***P* value****
	Stage 2 vs 1	-0.04 (-0.46 – 0.38)		0.855
	Stage 3 vs 1	-0.52 (0.09 – 0.95)		0.017
	Stage 3 vs 2	-48 (-90 – [-0.06])		0.024
Phosphate (mg/dL)	**CKD stage**	**Mean**	**SD**	***P* value***
	Stage 1	1.05	0.17	
	Stage 2	0.98	0.20	0.297
	Stage 3	1.06	0.23	

CKD: Chronic kidney disease; Na: Sodium; K: Potassium; Cl: Chloride; Mg: Magnesium; SD: Standard deviation; CI: Confidence interval.
*One-way ANOVA, **Post hoc LSD.

and Cathepsin D exhibited a progressive and statistically significant increase with advancing stage, with each higher stage showing greater concentrations than the preceding stage (Table 4).

Discussion

The study found that CKD progression is correlated with male gender and older age. The CKD progression has consistently been demonstrated to be associated with male gender and advanced age in previous studies. The findings from cohort study by Grams et al, encompassing 3,939 adults with diverse racial and ethnic backgrounds, revealed that women had significantly lower risk of incident end-stage renal disease compared with men (hazard ratio 0.72; 95% confidence interval 0.59 to 0.87), with males demonstrating a mean unadjusted eGFR slope of −1.43 mL/min per 1.73 m² per year compared to −1.09 mL/min per 1.73 m² per year in women (12). The acceleration of CKD progression in older patients has been extensively documented in population-based prospective cohorts, with older age independently associated with increased mortality risk despite lower rates of progression to renal replacement therapy in advanced stages (13). Similarly, gender differences in age-related glomerular filtration rate decline have been attributed to biological mechanisms including differential renal hemodynamics, hormone metabolism, and vasodilatory responses; a Chinese population-based screening study demonstrated that men in the CKD group exhibited significantly faster eGFR decline (0.44 mL/min per 1.73 m² per year adjusted difference from healthy individuals) compared with women (0.15 mL/min per 1.73 m² per year) (14). In older patients with CKD stages 4 and 5 enrolled in the European study by Chesnaye et al on treatment in advanced CKD, renal function declined 16.2% annually in men versus 9.6% in women, with this sex disparity remaining robust after adjustment for cardiovascular risk factors, comorbidities, and informative censoring from death and dialysis initiation (15). The increased vulnerability of males to faster CKD

Table 4. Comparative analysis of serum protein, cholesterol, NGAL, and Cathepsin D levels among patients with different CKD stages

Cholesterol (mg/dL)	**CKD stage**	**Mean**	**SD**	***P* value***
	Stage 1	171.75	31.69	
	Stage 2	199.23	59.26	0.049
	Stage 3	201.44	42.17	
	Between CKD stage comparison	**Mean difference (95% CI)**		***P* value****
	Stage 2 vs 1	27.48 (1.41 – 53.54)		0.039
	Stage 3 vs 1	29.69 (3.37 – 56.00)		0.028
	Stage 3 vs 2	2.20 (-23.58 – 28.00)		0.865
Protein (g/dL)	**CKD stage**	**Mean**	**SD**	***P* value***
	Stage 1	6.87	1.32	
	Stage 2	4.96	1.75	0.001
	Stage 3	4.84	0.74	
	Between CKD stage comparison	**Mean difference (95% CI)**		***P* value****
	Stage 2 vs 1	-1.90 (-2.66 – [-1.44])		<0.001
	Stage 3 vs 1	-2.03 (-2.79 – [-1.26])		<0.001
	Stage 3 vs 2	-0.12 (-0.87 – 0.62)		0.741
Albumin (g/dL)	**CKD stage**	**Mean**	**SD**	***P* value***
	Stage 1	3.26	0.31	
	Stage 2	2.94	0.59	0.076
	Stage 3	3.23	0.65	
NGAL (ng/mL)	**CKD stage**	**Mean**	**SD**	***P* value***
	Stage 1	38.95	12.34	
	Stage 2	84.69	15.50	<0.001
	Stage 3	141.48	14.58	
	Between CKD stage comparison	**Mean difference (95% CI)**		***P* value****
	Stage 2 vs 1	45.73 (37.69 – 53.77)		<0.001
	Stage 3 vs 1	102.52 (94.40 – 110.64)		<0.001
	Stage 3 vs 2	56.79 (48.82 – 64.74)		<0.001
Cathepsin D (ng/mL)	**CKD stage**	**Mean**	**SD**	***P* value***
	Stage 1	103.95	38.74	
	Stage 2	218.80	15.49	<0.001
	Stage 3	252.36	31.93	
	Between CKD stage comparison	**Mean difference (95% CI)**		***P* value****
	Stage 2 vs 1	114.84 (97.89 – 131.80)		<0.001
	Stage 3 vs 1	148.40 (131.28 – 165.52)		<0.001
	Stage 3 vs 2	33.55 (16.77 – 50.33)		<0.001

CKD: Chronic kidney disease; NGA: Neutrophil gelatinase-associated lipocalin; SD: Standard deviation; CI: Confidence interval.
*One-way ANOVA, **Post hoc LSD.

progression has been attributed to hormonal influences, including potentially deleterious effects of testosterone and protective effects of endogenous estrogens, as well as sex-related differences in cardiovascular comorbidities and hypertension prevalence. Notably, diabetes demonstrated a disproportionately adverse impact on renal decline, specifically in women, resulting in comparable decline rates between diabetic men and women despite slower progression in non-diabetic females (16). Current KDIGO clinical practice guidelines recommend early detection and intensive management of modifiable risk factors such as blood pressure control, proteinuria reduction through renin-angiotensin system blockade, and cardiovascular risk modification, particularly in high-risk populations, including older males with CKD (17,18). In conclusion, this study corroborates substantial evidence that male gender and older age independently predict accelerated CKD progression through pathophysiological mechanisms involving sex hormones, renal hemodynamics, and differential cardiovascular risk profiles, and emphasizes the critical importance of implementing age-specific and sex-specific intervention strategies to mitigate adverse renal and cardiovascular outcomes in vulnerable CKD populations.

Our study also demonstrated that CKD progression was accompanied by declining renal function, dyslipidemia, electrolyte imbalances, including hypernatremia, hyperkalemia, and hypomagnesaemia, systemic inflammation, and reduced total protein, while albumin, phosphate, and chloride levels remained unaffected. The demonstration of progressive renal functional deterioration accompanied by dyslipidemia, systemic inflammation, and electrolyte disturbances in CKD aligns with established pathophysiological mechanisms documented in the literature. These findings corroborate extensive evidence indicating that CKD progression is characterized by a cascade of interconnected metabolic derangements; elevated inflammatory markers, particularly systemic immune-inflammation indices and cytokines, have been causally associated with both the development and progression of CKD, creating a bidirectional relationship where systemic inflammation simultaneously precipitates and results from declining renal function (19,20). The elevated levels of hyperkalemia, hypernatremia, and hypomagnesaemia documented in this study reflect impaired renal electrolyte handling mechanisms inherent to progressive nephron loss, with prior research demonstrating that dyskalemia, particularly hyperkalemia, constitutes one of the most prevalent electrolyte disturbances in CKD and is associated with increased cardiovascular morbidity and accelerated disease progression (21,22). Dyslipidemia manifested in the form of abnormal lipoprotein metabolism and elevated triglyceride-rich lipoproteins has been independently associated with rapid renal progression and advancement to end-stage renal disease, with both lower and higher total cholesterol levels increasing risk for renal replacement therapy in stages 3-5 CKD through mechanisms involving glomerular lipid deposition, macrophage infiltration, and pro-inflammatory cytokine stimulation (23). Notably, the finding that phosphate, albumin, and chloride levels remained unaffected contrasts with typical CKD-mineral bone disease patterns; serum phosphate usually remains normal until advanced CKD stages due to compensatory mechanisms via fibroblast growth factor-23 and parathyroid hormone-mediated phosphaturia, and albumin preservation may reflect adequate nutritional status or absence of significant proteinuria-related losses in this cohort (24). Overall, this study reinforces the complex multisystem nature of CKD progression, wherein simultaneous dysregulation of lipid metabolism, inflammatory homeostasis, and specific electrolyte handling mechanisms, coupled with selective preservation of certain biochemical parameters, collectively characterize the evolution toward end-stage renal disease and warrant comprehensive metabolic assessment to identify high-risk patients amenable to targeted therapeutic intervention.

Conclusion

The findings demonstrate that CKD progression is strongly influenced by demographic factors such as male gender and advancing age, and is accompanied by distinct biochemical alterations. Progressive decline in renal function was reflected by rising levels of traditional kidney function markers and electrolytes, alongside elevations in cholesterol and novel biomarkers such as NGAL and Cathepsin D, while reductions in eGFR, magnesium, and total protein further highlighted the metabolic disturbances associated with disease advancement. In contrast, albumin, phosphate, and chloride remained relatively stable across stages, suggesting that not all serum parameters are equally sensitive to disease progression. Collectively, these results underscore the multifactorial biochemical changes in CKD and highlight the potential utility of emerging biomarkers in complementing conventional measures for disease monitoring and risk stratification.

Limitations of the study

The study has several limitations, including a small overall sample and unequal stage groups limiting statistical power; a cross-sectional, single-center design that prevents causal or temporal inferences and reduces generalizability; incomplete adjustment for key confounders (diabetes, hypertension control, BMI, medications, smoking, socioeconomic status); restriction to CKD stages 1–3 and lack of quantitative urine protein measures or longitudinal biomarker sampling; and multiple comparisons without clear adjustment increasing the risk of false-positive findings.

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Conflicts of interest

The author declares no conflict of interest.

Data availability statement

The datasets generated during and/or analyzed during the current study are available from the corresponding author on reasonable request.

Declaration of generative artificial intelligence (AI) and AI-assisted technologies in the writing process

While preparing this work, the author utilized AI tools (Perplexity.ai, Copilot and Grammarly.com) to refine grammar points and language style in writing.

Subsequently, the author thoroughly reviewed and edited the content as necessary, assuming full responsibility for the publication's content.

Ethical issues

The study was conducted in accordance with principles of the Declaration of Helsinki. Informed written consent was obtained from all participants. This study resulted from a research project and was approved by the Medical Ethics Committee of Southern Technical University, Basra, Iraq (Reference No. MEC: 63; Approval Date: 10 January 2025). Besides, the author has ultimately observed ethical issues (including plagiarism, data fabrication, and double publication).

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