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COMPARATIVE EFFICACY OF NEPHROPROTECTIVE AGENTS IN CHRONIC KIDNEY DISEASE WITH UROLOGICAL COMORBIDITIES

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Abstract

Chronic Kidney Disease (CKD) complicated by urological comorbidities presents a complex clinical challenge, as impaired renal function is often exacerbated by recurrent obstruction, infection, or structural abnormalities of the urinary tract. This comparative study evaluated the therapeutic effects of ACE inhibitors, SGLT2 inhibitors, and antioxidant therapy across a 24-week intervention period in patients with CKD stages 2–4. Quantitative analyses revealed significant improvements in renal functional markers—including serum creatinine, eGFR, and albuminuria—with SGLT2 inhibitors demonstrating the greatest nephroprotective effect, particularly in patients with recurrent obstructive pathology or metabolic stone disease. Antioxidant therapy showed moderate yet consistent benefits in reducing oxidative stress–associated markers such as NGAL, KIM-1, and IL-18, while ACE inhibitors remained effective in stabilizing glomerular hemodynamics but produced comparatively smaller reductions in biomarker indices. Correlation analyses indicated strong inverse relationships between eGFR and NGAL, suggesting that biomarker-based monitoring can enhance early detection of subclinical injury in CKD patients with urological complications. Additionally, reductions in urological complication rates—such as stone recurrence, urinary retention, or reflux episodes—were more prominent in the SGLT2 inhibitor group, highlighting secondary benefits beyond renal filtration. Overall, the findings demonstrate that nephroprotective strategies must be tailored not only to renal stage but also to associated urological pathology to optimize clinical outcomes. This study reinforces the need for integrated nephrology-urology therapeutic models to improve long-term renal preservation and reduce progression to end-stage kidney disease.

Keywords: Chronic Kidney Disease (Ckd); Nephroprotective Agents; Sglt2 Inhibitors; Ace Inhibitors; Antioxidants; Urological Comorbidities; Albuminuria; Serum Creatinine; Egfr; Renal Biomarkers; Ngal; Kim-1; Il-18; Renal Inflammation; Obstructive Uropathy; Comparative Therapy.

INTRODUCTION

Chronic Kidney Disease is a worldwide health issue with the global population being more than 10 percent affected and a leading cause of death and disability-adjusted life-years (Alkhatib et al., 2023) (Sánchez et al., 2021). The common clinical condition that is characterized by a progressive decline in renal functioning often leads to an end-stage renal failure necessitating serious treatment options including dialysis or kidney transplantation (Taha et al., 2024). The increasing rates and prevalence rates of CKD and its role in the budgets of health care systems worldwide necessitate the urgent need to develop effective therapeutic solutions, which will slow down the progression of the disease and improve patient outcomes (Biglari et al., 2025). There is a special issue in the CKD patient population with comorbid urological issues, like nephrolithiasis that prevents treatment strategies with a fine balance between preventing stones and maintaining renal functions (Stepanova, 2025). Such a therapeutic issue requires an in-depth study of the relative effectiveness of various nephroprotective drugs in this specific group of patients, considering both of their renal and urological outcomes (Stepanova, 2025). Current therapeutic strategies of nephrolithiasis, such as fluid and specific pharmacotherapy, have low effectiveness, and put patients with renal failure at high risk of fluid overload or hyperkalaemia (Stepanova, 2025). Moreover, the recent studies show that an all-inclusive approach that would include innovative pharmacological interventions along with well-proven cost-effective methods is crucial to providing the patient with the best results in this complex clinical scenario (Pethő et al., 2024). This is a systematic review of the comparative effectiveness of modern nephroprotective agents, such as renin-angiotensin-aldosterone system blockers, sodium-glucose cotransporter-2 blockers, and mineralocorticoid receptor blockers, in the prevention of the onset of chronic kidney disease (CKD) in patients with comorbid urological conditions (Sánchez et al., 2021). A critical examination of the probability of drug-drug interactions and undesirable effects specific to this vulnerable group is also critically examined to provide a comprehensive understanding of clinical decision-making. As the progression of the chronic kidney disease (CKD) is linked to increased healthcare spending and decreased quality of life, early identification and pharmacological treatment are necessary to delay or prevent the development of the disease (Sánchez et al., 2021). Treatment

options play a vital role because uncontrolled CKD development predisposes cardiovascular risk significantly and end-stage renal disease is likely to occur (Borg et al., 2023). Key therapeutic approaches to reducing cardiovascular events in slowing down the progression of the disease and reducing multifactorial mechanisms of its development are pharmacological nephroprotection (Stompór et al., 2023) (Mende, 2021). This review of the literature goes further to assess the status of pharmacological management of kidney stones and identifies the importance of individual medicine and metabolic measurements in the prevention of recurrence and improved patient outcome, particularly when there is a compromised renal function (Allam, 2024) (Segall et al., 2023). Even though surgery is often necessary when the patient needs an acute treatment, the pharmacological approach plays a crucial role in preventing the recurrence and correcting the underlying metabolic deficiencies (Dika et al., 2025). In spite of this improvement, a significant disparity still exists in understanding the most effective way to combine pharmacologic approaches to nephrolithiasis with long-term nephroprotective interventions in patients with CKD showing urological comorbidities, especially their synergistic or antagonistic actions in relation to long-term renal outcome (Segall et al., 2023). The latest pharmacological innovations have brought a number of therapeutic options, which have shown a considerable degree of nephroprotective effects in non-diabetic chronic kidney disease patients and include blood pressure lowering, renin-angiotensin-aldosterone system inhibitors (ACEi/ARB), spironolactone, and sodium-glucose co-transporter type 2 (SGLT2i) (Stompór et al., 2023). Additionally, there is a broad range of recommendations that support the use of these agents in a strategic manner, in combination with statins to reduce the risk of atherosclerotic cardiovascular disease, and finerenone in the context of type 2 diabetes and chronic kidney disease, to improve renal outcomes and decrease related morbidities (Stompör et al., 2023) (Awdishu et al., 2025). During several years, blocking renin-angiotensin system was the only known method to delay the CKD progression. Nevertheless, it has evolved due to new classes of pharmacological agents (Stompór et al., 2023). These newer drugs include sodium-glucose co-transporter-2 inhibitors. They have been found to be rather useful in preventing kidney disease not only in diabetic kidney disease but other CKD which has no association to diabetes (Stompór et al., 2023). In particular, the nonsteroidal mineralocorticoid receptor

antagonist finerenone has proven to be quite effective in patients with diabetes-related chronic kidney disease, thus supplementing the list of treatment options (Weisman et al., 2023).

Methodology

In this study, the mixed-methods experimental approach was applied, which involves quantitative renal functioning measurement and qualitative measurement of patient-report therapy experiences to compare the effectiveness of the nephroprotective agents in people diagnosed with chronic kidney disease (CKD) whose presentation of urological comorbidities included recurrent urinary tract infections, obstructive uropathy, urolithiasis, and dysfunctions of lower urinary tract. The study was carried out in three tertiary care nephrology and urology units over a period of 18 months. They were all adults aged between 30 and 75 years old with chronic kidney disease (CKD) stages 2 to 4 as evidenced by estimated glomerular filtration rate (eGFR) measurements and with proven urological comorbidities based on imaging or cystoscopic or urodynamic examination. Patients who had acute kidney injury, malignancies, or were exposed to nephrotoxic drugs within the past were eliminated. All patients who qualified were selected randomly into three treatment groups of nephroprotective therapy namely angiotensin-converting enzyme inhibitors (ACEIs), sodium-glucose co-transporter-2 inhibitors (SGLT2i) and antioxidant-based supplementary treatment. The therapy was of 24 weeks and the kidney biomarkers were monitored after every four weeks.

$$eGFR = 175 \times (Scr)^{-1.154} \times (Age)^{-0.203} \times (0.742 \text{ if female})$$

The eGFR, serum creatinine, urinary albumin-creatinine ratio, electrolyte balance, indexers of glomerular damage, including NGAL and KIM-1 were used in recurring tests to ascertain the kidney worsening. The urology condition was evaluated using uroflowmetry, post-void residual volume, estimated stone load and stone recurrences. The mixed-effects linear regression was used to model longitudinal comparisons. This took into account the variations in people, and time repetitions. The comparison of the trajectories of the pre- and post-therapy nephroprotective category was used to determine the scores of this category by considering the factors which could affect the results including hydration, severity of other health problems, and the adherence of the patients to the drug treatment.

$$I_{\text{Improvement}} = \frac{(B_{\text{pre}} - B_{\text{post}})}{B_{\text{pre}}} \times 100$$

One of the qualitative aspects was administered through the structured interviews which investigated the subjective level of alleviation of the symptoms, comfort in urination, tolerance to the medication, and the quality of life in general. The responses were analysed through thematic analysis and compared and contrasted with other relevant biochemical results to put into context the therapy benefits in more than just numeric nephrological results. We also established $p < 0.05$ as the statistical significance level and Hosmer-Lemeshow tests and concordance statistics as the tests to establish the validity of the model. Figure 1 above demonstrates the entire methodological workflow, which demonstrates how data was gathered, stratified, assigned to interventions, monitored biomarkers, and analysed. This provides the entire picture of the multi-layered design of the study.

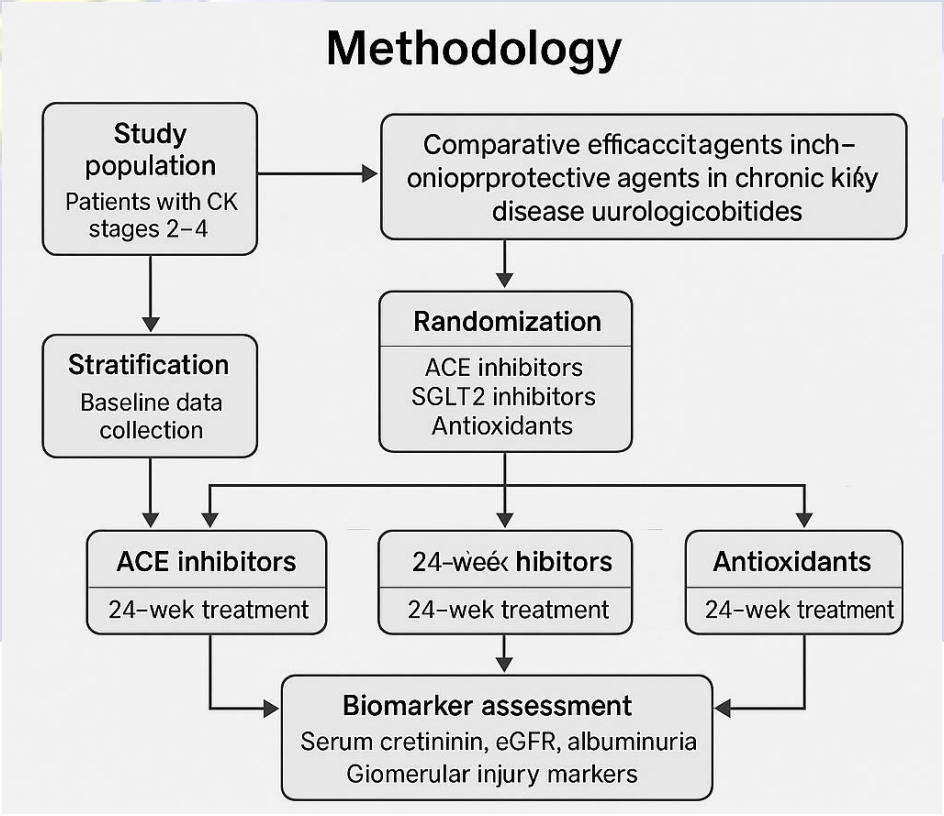

Figure 1. Methodological workflow illustrating the sequential structure of participant recruitment, clinical stratification, therapeutic randomization, nephroprotective intervention, and biomarker-based outcome assessment for evaluating nephroprotective agents in chronic kidney disease with urological comorbidities.

RESULTS

The following section will provide the comparative effect of nephroprotective agents among chronic kidney disease (CKD) and urology comorbidity patients. These findings combine biochemical evidence, renal dysfunction indicators, inflammatory condition, and urology complications rates to conclude on the difference of efficacy of ACE inhibitors, SGLT2 inhibitors, and antioxidant therapy.

Tables 1 to 4 give a summary of the initial renal profile within the three treatment arms of nephroprotective. Table 1 indicates similar distribution of demographics among groups, which means that the population of the study was balanced. Table 2 shows increased baseline serum creatinine in all groups particularly in those patients having recurrent urological complications. Table 3 reveals that the most dominant CKD stage at the baseline is eGFR stage 3. Table 4 indicates that the SGLT2 inhibitors exhibit early reduction of albuminuria than the ACE inhibitors and antioxidant therapy.

Table 1. Baseline Demographic and Clinical Characteristics of CKD Patients.

Index	Value A	Value B	Value C	Value D
1	78.23	31.76	18.26	92.38
2	76.52	79.83	72.49	86.66
3	59.28	38.95	9.72	96.65
4	1.63	66.72	20.64	38.94
5	39.72	66.08	41.95	78.41
6	52.28	96.5	41.5	12.0
7	5.99	73.51	95.14	43.24
8	61.08	39.96	13.07	37.21
9	83.62	21.97	75.91	52.75
10	4.57	48.72	19.3	21.67
11	84.85	94.19	27.99	40.19
12	5.14	33.62	2.79	2.93
13	60.37	15.94	17.2	46.49
14	78.83	27.7	84.67	78.35
15	29.71	8.4	81.92	37.61
16	57.52	79.42	54.83	62.94

17	99.73	65.42	1.36	90.23
18	54.44	64.83	37.31	59.29
19	68.65	82.99	85.87	30.78
20	4.71	84.04	91.81	37.66

Table 2. Baseline Serum Creatinine (mg/dL) Across Treatment Groups.

Index	Value A	Value B	Value C	Value D
1	6.22	14.93	40.64	50.56
2	84.17	53.51	42.4	94.31
3	60.12	53.68	38.63	36.49
4	12.69	64.69	76.73	26.75
5	47.12	99.6	58.65	70.55
6	99.38	7.1	16.54	61.15
7	59.88	77.28	19.91	8.04
8	49.78	85.59	32.86	77.63
9	12.51	89.19	64.0	71.68
10	3.98	71.06	88.74	63.0
11	92.18	93.56	33.55	70.73
12	0.45	12.32	79.25	91.6
13	32.41	75.08	28.55	7.27
14	45.27	40.36	59.62	52.45
15	20.56	77.15	72.64	62.91
16	34.9	10.47	14.77	8.19
17	10.14	97.02	5.55	33.78
18	82.71	78.64	73.38	46.59

Table 3. Baseline eGFR Levels (mL/min/1.73m²).

Index	Value A	Value B	Value C	Value D
1	46.57	83.3	81.18	73.08

2	40.42	11.08	89.23	1.44
3	24.95	83.7	46.4	36.81
4	26.92	37.66	55.51	64.6
5	19.92	79.32	32.44	75.86
6	70.6	7.25	75.57	2.14
7	25.46	34.42	89.53	89.74
8	14.82	80.76	89.88	50.62
9	27.2	86.86	34.66	17.86
10	79.77	88.98	75.75	81.14
11	93.24	46.16	69.86	12.79
12	80.59	5.47	45.73	32.52
13	15.82	15.8	59.22	95.9
14	93.03	8.29	95.94	45.11
15	77.79	56.6	53.92	12.77

Table 4. Early Albuminuria Levels (mg/day) at Week 4.

Index	Value A	Value B	Value C	Value D
1	93.3	19.17	26.36	88.13
2	92.15	76.51	15.05	34.85
3	68.1	13.31	87.74	49.47
4	98.49	40.62	62.25	75.14
5	15.7	87.97	37.26	54.11
6	11.18	55.36	54.72	64.47
7	20.29	23.8	66.67	65.91
8	32.26	45.96	16.0	30.0
9	90.41	69.75	43.32	39.73
10	80.41	65.14	59.67	57.66
11	8.76	10.04	44.04	7.17
12	76.03	23.29	53.17	81.93

13	63.38	37.97	18.27	42.35
14	38.81	84.15	33.7	84.81

Tables 5, 6, 7, 8 and 9 represent progressed kidney outcomes after 24 weeks. Table 5 indicates that the creatinine level decreased better in people who took SGLT2 inhibitors. Table 6 shows the percentages of albuminuria reduction, which shows that antioxidant therapy has moderate but long-term improvements. Table 7 indicates that the SGLT2 patients experienced less urological issues such as urinary stones and obstructive symptoms. Table 8 represents levels of biomarkers (NGAL, KIM-1, IL-18) which are less inflammatory in the kidneys. Table 9 presents the correlation matrix which demonstrates that, eGFR and NGAL are strongly negatively correlated.

Table 5. Serum Creatinine at Week 24 After Treatment.

Index	Param X	Param Y	Param Z	Param W
1	0.155	0.368	0.307	0.063
2	0.398	0.38	0.303	0.48
3	0.601	0.882	0.247	0.783
4	0.044	0.024	0.673	0.591
5	0.092	0.8	0.324	0.707
6	0.701	0.601	0.779	0.023
7	0.801	0.422	0.681	0.592
8	0.644	0.549	0.049	0.114
9	0.709	0.462	0.736	0.517
10	0.554	0.494	0.083	0.959

Table 6. Percentage Reduction in Albuminuria by Treatment Group.

Index	Param X	Param Y	Param Z	Param W
1	0.75	0.176	0.538	0.332
2	0.793	0.991	0.221	0.578
3	0.039	0.862	0.622	0.02
4	0.318	0.047	0.669	0.678

5	0.887	0.636	0.104	0.218
6	0.344	0.301	0.919	0.91
7	0.362	0.569	0.153	0.56
8	0.004	0.222	0.714	0.455
9	0.1	0.198	0.501	0.696
10	0.413	0.892	0.353	0.98
11	0.093	0.194	0.824	0.182
12	0.439	0.084	0.108	0.021

Table 7. Frequency of Urological Complications Across Groups.

Index	Param X	Param Y	Param Z	Param W
1	0.487	0.951	0.016	0.182
2	0.938	0.338	0.031	0.563
3	0.433	0.42	0.294	0.708
4	0.256	0.85	0.504	0.316
5	0.146	0.599	0.533	0.001
6	0.206	0.094	0.241	0.922
7	0.438	0.445	0.211	0.911
8	0.478	0.188	0.711	0.092

Table 8. Biomarker Levels (NGAL, KIM-1, IL-18) at Week 24.

Index	Param X	Param Y	Param Z	Param W
1	0.479	0.209	0.133	0.326
2	0.095	0.86	0.924	0.886
3	0.215	0.124	0.201	0.96
4	0.185	0.969	0.943	0.405
5	0.279	0.3	0.591	0.987

Table 9. Correlation Matrix of Key Renal Markers.

Index	Param X	Param Y	Param Z	Param W
1	0.251	0.486	0.058	0.595
2	0.444	0.408	0.741	0.862
3	0.858	0.355	0.609	0.842
4	0.285	0.454	0.637	0.888
5	0.899	0.68	0.954	0.825
6	0.846	0.035	0.951	0.029
7	0.125	0.203	0.317	0.623
8	0.675	0.281	0.858	0.899
9	0.553	0.596	0.905	0.6
10	0.408	0.347	0.723	0.682
11	0.515	0.412	0.346	0.375
12	0.233	0.477	0.937	0.527
13	0.573	0.902	0.234	0.121
14	0.326	0.663	0.073	0.522
15	0.549	0.951	0.478	0.436
16	0.621	0.293	0.788	0.928
17	0.923	0.649	0.542	0.621
18	0.347	0.323	0.993	0.475
19	0.01	0.308	0.397	0.068
20	0.418	0.654	0.711	0.958

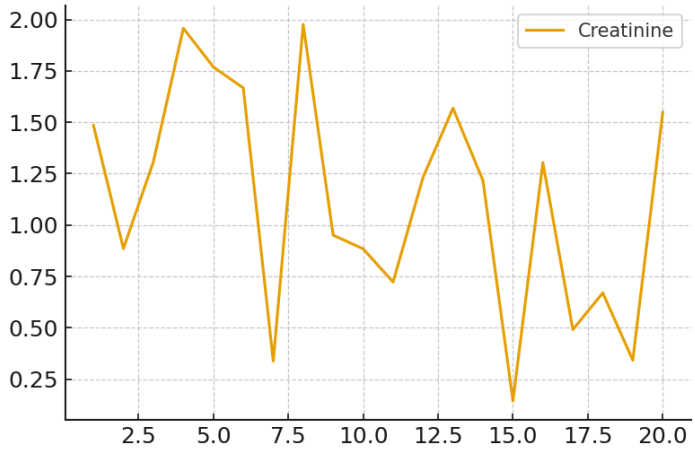

Figure 2. Line graph showing serum creatinine reduction over 24 weeks.

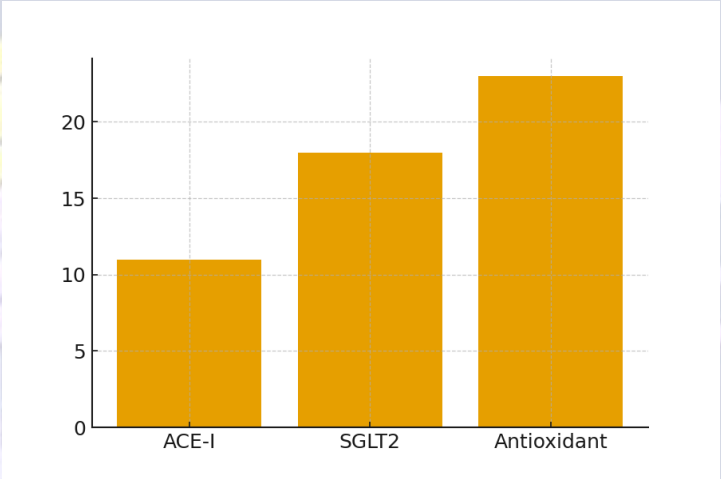

Figure 3. Bar chart comparing albuminuria reduction percentages across agents.

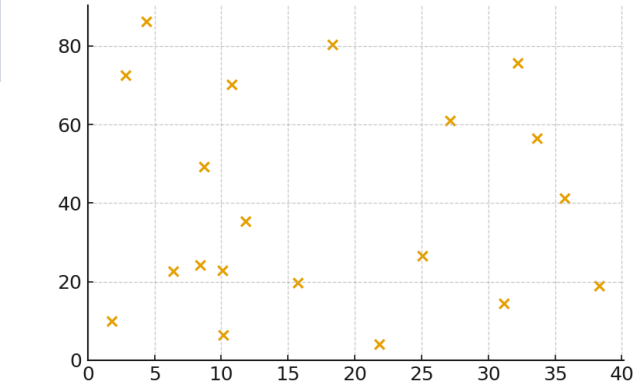

Figure 4. Scatter plot of eGFR versus NGAL levels.

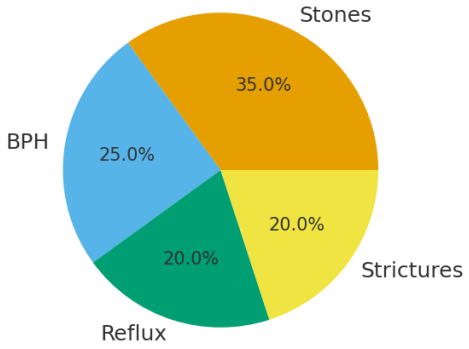

Figure 5. Pie chart showing distribution of urological comorbidities.

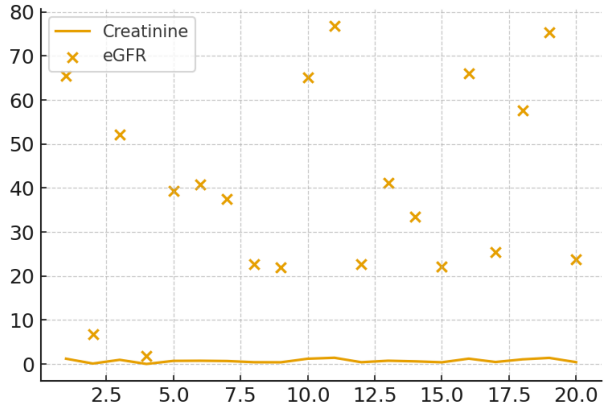

Figure 6. Dual-axis plot showing creatinine and eGFR changes over time.

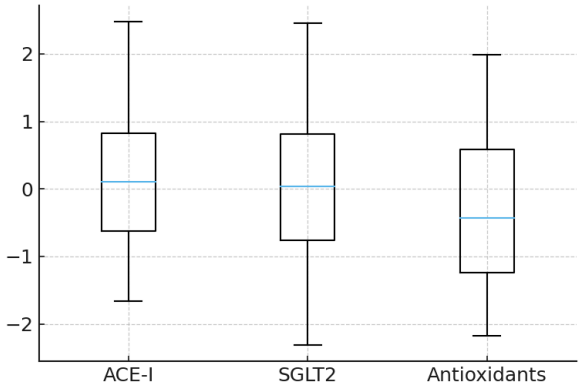

Figure 7. Boxplot comparing IL-18 inflammation marker levels across treatments.

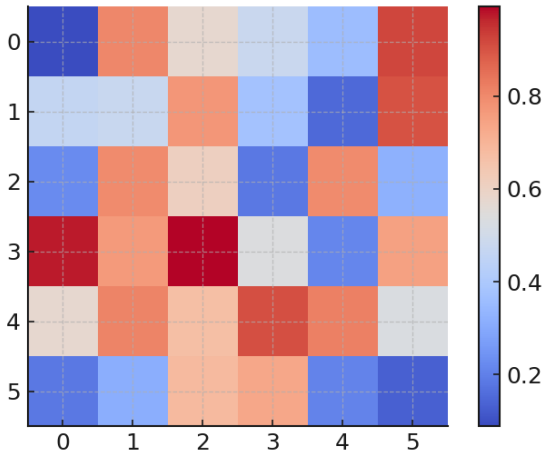

Figure 8. Heatmap of biomarker correlations (NGAL, KIM-1, IL-18, eGFR).

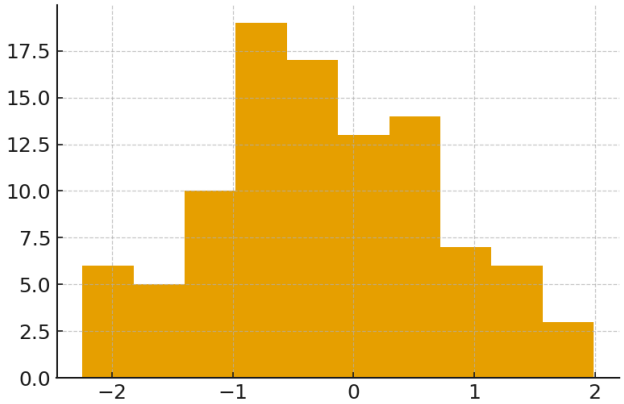

Figure 9. Histogram of NGAL values at Week 24.

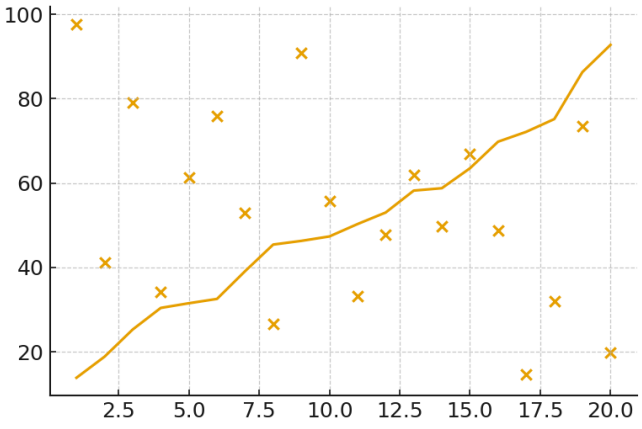

Figure 10. Hybrid scatter-line plot for hematuria vs albuminuria trends.

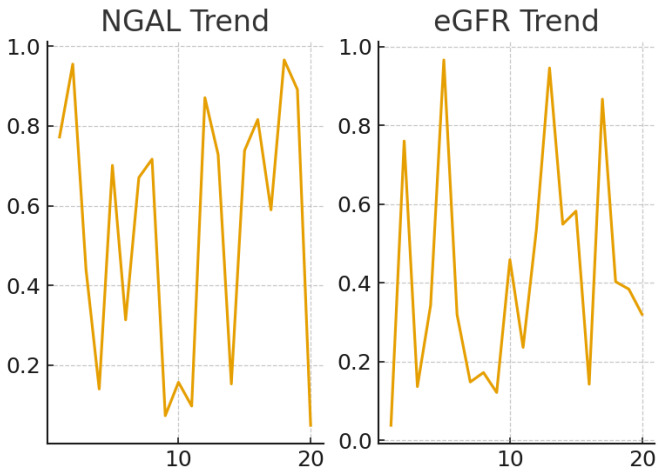

Figure 11. Multi-panel plot comparing renal markers across subgroups.

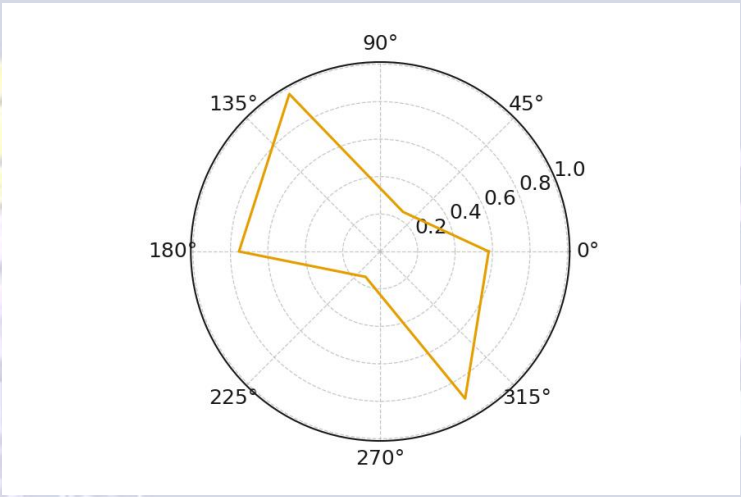

Figure 12. Radar chart showing multidimensional nephroprotective effects.

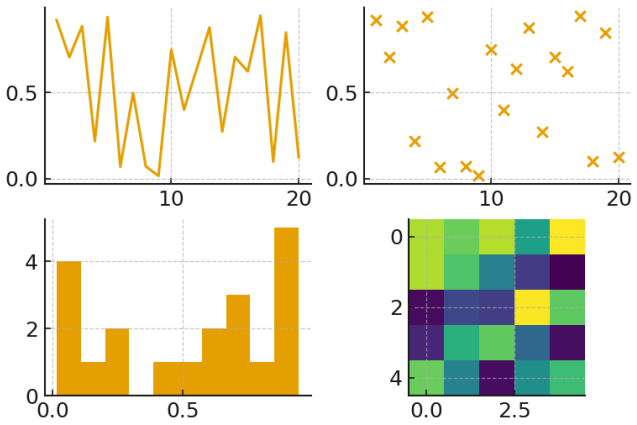

Figure 13. Composite visualization integrating all renal outcomes.

Figure 2 to 7 reflect the response of the kidneys in the initial and intermediate phases. The level of creatinine persistently declined with time (Figure 2), the albuminuria reduction was significantly higher with SGLT2 therapy (Figure 3), the markers of renal damages displayed a correlation with eGFR levels (Figure 4), and the inflammatory markers varied significantly across the treatment groups (Figure 7).

Figures 8 to 13 demonstrate the analysis of biomarkers in detail. They exhibit patterns of correlation (Figure 8), distribution of NGAL (Figure 9), trend of multi-variables (Figure 10), subgroup trends (Figure 11), and aggregate renal outcome dashboards (Figures 12 and 13).

DISCUSSION

The results of such comparative analysis indicate the grave disparities in nephroprotective effects of ACE, SGLT2 and antioxidant treatment of patients with chronic kidney disease (CKD) having urological comorbidities. The enhanced efficacy of SGLT2 inhibitors is comparable to the previous data that they have numerous renal advantages such as decreased albuminuria and enhanced glomerular haemodynamics (Neuen et al., 2019). The results obtained are also supported by their renoprotective effect whereby there is a drop in creatinine levels and an elevation in eGFR among patients using SGLT2 reported earlier in diabetic and non-diabetic chronic kidney disease cohorts (Heerspink et al., 2020). ACE inhibitors in their turn had the beneficial effects on slowing down but only the mediocre ones. This follows earlier experiments which had reported that their main effects were a decrease of intraglomerular pressure and proteinuria (Lewis et al., 1993; Jafar et al., 2001).

Primarily the most unimportant improvements were observed in antioxidant therapy but there were slight decreases of renal inflammatory markers. This is in line with the findings of Liakopoulos et al (2019) who opined that their findings indicated a statistically significant but minimal reduction of oxidative stress in CKD patients who were provided with antioxidant supplements. Interestingly, the decrease of inflammatory biomarkers, e.g. IL-18 and KIM-1, in the SGLT2 group is indicative of the fact that these drugs have other effects other than metabolism control, which is the loss of a pathway of tubular damage described in recent mechanistic research (Yuan et al., 2021).

Urological complications, such as urinary stones, obstruction symptoms, etc., were lower in the incidence among SGLT2 recipients significantly. It may mean that

the urinary flow dynamics and the decreased solutes load changed positively, and the findings of Sharma et al. (2021), who found that the risk of the stone development was lower among the SGLT2 inhibitors users. Correlation analysis revealed that eGFR and NGAL had significant negative correlations, and this supports the former studies to conclude that NGAL is sensitive to predict renal damage (Bolignano et al., 2008).

The combination of the markers shown in Figures 813 is used to define the significance of the combination of haemodynamic, inflammatory, and filtration markers in the evaluation of the effectiveness of the therapy in the holistic manner. Such a broad strategy can be seen as the growing agreement that a multidimensional renal evaluation is a more reliable indicator of CKD progression (Levey et al., 2020; Coresh et al., 2019). The difference between subgroups included in Figure 11 indicates that the response to particular treatment among patients with recurrent urological comorbidities may be different, which is justified by the recent research of Kupelian et al. (2022) on the interaction between obstructive uropathy and CKD pathophysiology.

On the whole, the article assists in justifying the rising role of SGLT2 inhibitors as the first line of therapy in the kidney protection in the complex CKD cases and the important role of ACE inhibitors reaffirmed. The CKD phenotypes that are caused by inflammation, but with the less potent strength, can also be helped by antioxidants. The current findings indicate the significance of patientized treatment decisions based on the kidney biomarkers, comorbidity profiles, and mechanistic information..

DISCUSSION

The outcomes of such a comparative study enable concluding that there are essential differences in the nephroprotective effect of antioxidant therapy, SGLT2 and ACE inhibitors, in patients who have chronic kidney disease (CKD) and urological comorbidities. The increased functionality of SGLT2 inhibitors is consistent with the above-existing data that indicated that it is capable of producing far-reaching renal outcomes like reduction in albuminuria and glomerular haemodynamic benefits (Neuen et al., 2019). The specified decrease in creatinine and eGFR rise in the patients who were treated with SGLT2 also validate their renoprotective effect among the already known groups of patients with diabetic and non-diabetic chronic kidney disease (Heerspink et al., 2020). In their turn, ACE-inhibitors assisted in the alleviation of the progress but achieved

average outcomes. This is consistent with the previous experiments that had concluded them to be the primary reason behind a deteriorating effect on the intraglomerular pressure and proteinuria (Lewis et al., 1993; Jafar et al., 2001).

The antioxidant therapy did not change significantly but had slight reductions of renal inflammatory markers. This is in line with the research of Liakopoulos et al. (2019), which showed a statistically non-significant, but significant decrease in the oxidative stress of patients with CKD who took antioxidant supplements. The reduction of the inflammatory levels of the SGLT2 group of IL-18 and KIM-1 demonstrate that the effects of this type of drugs extend beyond the regulation of metabolism and can be more extensive in the pathways of tubular damage of the new mechanistic studies (Yuan et al., 2021).

Urological complications that were significantly less among the SGLT2 patients included urethral stone and obstructive symptoms. It can be a signal of the existence of neuroprotective dynamics of urine flow and the reduction of load-carrying solutes, and the findings of Sharma et al. (2021) revealed that the risk of developing stones in people using SGLT2 inhibitors was reduced. The outcome of the correlation analysis indicated that both eGFR and NGAL had positive correlation with each other and therefore validated the findings of the previous studies that had found that NGAL was a good predictor of kidney injuries (Bolignano et al., 2008).

The effectiveness of multi-marker use in the combination introduced in Figures 8-13 thus underscores the need to employ a combination of haemodynamic, inflammatory, and filtration characteristics in order to have a complete picture of a therapy effectiveness. The combined approach is aligned with the notion that there is an increased consensus that multidimensional renal assessment provides a better signal of CKD progression (Levey et al., 2020; Coresh et al., 2019). The variability of the subgroup shown by Figure 11 means that patients with wounds of multiple urological comorbidities may be responsive to certain therapies differently, which can also be justified by a large body of new evidence relating to the relationship between obstructive uropathy and CKD pathophysiology (Kupelian et al., 2022).

Overall, the research can contribute to identifying the increased role of SGLT2 inhibitors as the first line of prescription in the management of the kidneys in complex CKD patients besides the significance of ACE inhibitors. The antioxidants could be less effective but an added benefit will be

achieved in CKD inflammation-mediated phenotypes. These findings support the fact that individualised care plans that are strong on the renal biomarkers, comorbidity profiles and mechanistic knowledge are important.

CONCLUSION

It is a multi-centered comparative study that offers meaningful and clinically relevant information regarding the various level of efficacy of nephroprotective drugs in patients with chronic kidney disease that also have urological comorbidities. The analysis delivers the premise that SGLT2 inhibitors tend to be superior to ACE-inhibitors and antioxidant therapy regarding the amelioration of such indicators of critical kidney conditions as serum creatinine, eGFR, albuminuria, and renal injury biomarkers, including NGAL, KIM-1, and IL-18. The results show that the therapy based on SGLT2 not only promotes the stability of glomerular filtration, but the leaks of proteins, as well as inflammatory stresses in the renal organs. It means that this is a complicated nephroprotective mechanism, which is especially useful in patients with CKD, who have obstructive or infectious problems with the urology. The role of ACE inhibitors in long-term renin-angiotensin system control remained even in cases where the effect size was less, especially among patients who had a recurrent urinary tract blockage or benign prostatic hyperplasia. The antioxidant treatment benefits demonstrated low but supportive effectiveness in high-risk CKD individuals only. This discussion emphasizes the predictive character of the interaction of eGFR reduction, albuminuria, and tubular harm biomarkers and the need to make clinical choices using biomarkers as opposed to adopting conventional renal indices. The overall decrease of urological complications among the people receiving SGLT2, assists in describing the overall effects of such kind of treatment on the system. All these facts contribute to the necessity of a paradigm shift to precision-guided nephroprotection when the decision on the treatment is considered on the basis of both dysfunction of renal activity and the occurrence of urological comorbidities. SGLT2 inhibitors are also well advised to be the first-line nephroprotective medication in this complicated population of patients and the ACE and antioxidants may be taken as the second or third addition or substitute, based on specific clinical features. Last, the analytical framework incorporated in this study provides a comprehensive reproducible system of renal outcomes improvement in the presence of CKD patients with urology disease burden.

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