The Research of Medical Science Review
ISSN: 3007-1208 & 3007-1216 Volume 3, Issue 4, 2025

COMPARISON OF ESTIMATED GLOMERULAR FILTRATION RATE (EGFR) AND URINE ACR IN DIABETIC CKD PATIENTS TAKING SGLT-2 INHIBITORS VS PATIENTS NOT TAKING SGLT-2 INHIBITORS

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DOI: https://doi.org/10.5281/zenodo.15285985

Keywords
Chronic kidney disease (CKD), Type 2 diabetes mellitus (T2DM), SGLT-2 inhibitors, Urine albumin-to-creatinine ratio (ACR), Diabetic nephropathy

Article History
Received on 15 March 2025
Accepted on 15 April 2025
Published on 26 April 2025

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Abstract

The current study compared the effects of sodium-glucose cotransporter-2 (SGLT-2) inhibitors on estimated glomerular filtration rate (eGFR) and urine albumin-to-creatinine ratio (ACR) in patients with diabetic chronic kidney disease (CKD) versus those not receiving SGLT-2 inhibitors.

Study Design: *Cohort Study*

Study Place*: CMH Rawalpindi*

Materials and Methods: *Patients with type 2 diabetes mellitus and chronic kidney disease (stages 2-4, eGFR ≥ 30 mL/min/1.73 m²) were divided into two groups: those treated with SGLT-2 inhibitors (mainly dapagliflozin) and those not treated with these agents. Data was collected retrospectively from hospital records, encompassing ACR and eGFR at baseline and after 24 weeks of follow-up. Independent t-tests and chi-square tests were utilized for statistical analyses, with significance established at $p < 0.05$.*

Results

A total of 162 patients were included in the study, with 54 receiving SGLT-2 inhibitors and 108 not receiving them. At baseline, both groups exhibited similar eGFR and ACR levels. Following 24 weeks, patients receiving SGLT-2 inhibitors demonstrated a more significant decrease in eGFR, declining from 67.5 to 65.2 mL/min/1.73 m², in contrast to the non-SGLT-2 group, which decreased from 68.9 to 68.2 mL/min/1.73 m². The reduction in ACR was more significant in the SGLT-2 inhibitor group than the non-SGLT-2 group.

Conclusion

SGLT-2 inhibitors significantly slowed ACR progression in diabetic CKD patients compared to other glucose-lowering therapies, suggesting their potential as nephroprotective agents in managing CKD.

INTRODUCTION

Chronic kidney disease (CKD) is a significant and advancing ailment frequently observed in people with type 2 diabetes mellitus (T2DM) [1]. It significantly contributes to the global occurrence of kidney failure [2]. Even with advancements in diabetes treatment, there are still few effective long-term medications

explicitly designed to prevent nephropathy in type 2 diabetes mellitus. ACE inhibitors and ARBs have demonstrated moderate effectiveness in reducing kidney decline, particularly in diabetic patients who already have proteinuria. Nonetheless, the alleged nephroprotective benefits of these drugs in individuals without proteinuria are not well-supported, leading to considerable doubts regarding their general effectiveness in treating chronic kidney disease among diabetic patients [4]. In recent years, sodium-glucose cotransporter-2 (SGLT2) inhibitors have emerged as a novel treatment option, designed initially to enhance glucose control in individuals with type 2 diabetes mellitus (T2DM). Numerous randomized controlled trials (RCTs) have demonstrated that these medications effectively preserve kidney function and reduce cardiovascular risk, alongside their ability to lower glucose levels. The interest in the possibilities of these studies for nephrology has significantly increased, particularly concerning T2DM patients who have CKD [5,6]. However, significant gaps exist in our understanding of their significance for various patient groups, especially for those who do not exhibit albuminuria or proteinuria. Research, such as the Comparative Effectiveness of Cardiovascular Outcomes in New Users of SGLT-2 Inhibitors (CVD-REAL 3), has shown significant real-world evidence supporting the kidney-protective advantages of SGLT2 inhibitors [7,8]. The findings from these trials show a more gradual decline in eGFR compared to other methods for controlling glucose levels. A team from Sweden, Denmark, and Norway supported the conclusion that the occurrence of renal events was reduced with SGLT2 inhibitors compared to dipeptidyl peptidase 4 (DPP-4) inhibitors [9]. However, the outcomes of both studies were constrained by the lack of thorough assessments of albuminuria or proteinuria, which restricted their relevance to CKD patients. The current circumstances highlight the crucial need to examine whether SGLT2 inhibitors offer unique protective benefits for renal function based on the pattern of eGFR reduction before starting treatment. This holds clinical significance as individuals experiencing a rapid decline in eGFR are at a heightened risk of swiftly progressing to renal failure [10]. Therefore, understanding the ability of SGLT2 inhibitors to influence the progression of renal decline in a broader CKD population, regardless of proteinuria levels, is crucial for creating more comprehensive and effective treatment strategies. This study aimed to assess the impact of sodium-glucose cotransporter-2 (SGLT-2) inhibitors on kidney function, mainly focussing on the urinary albumin-to-creatinine ratio (ACR) and estimated glomerular filtration rate (eGFR), about diabetic chronic kidney disease (CKD).

Materials and Methods

The current investigation was a cohort study that evaluated the impact of SGLT-2 inhibitors on ACR in diabetic CKD patients. Two patient cohorts were compared: those diabetic patients who were on SGLT-2 inhibitors and those who were not. Necessary kidney function tests involving eGFR and ACR were done at three routine follow-ups during the study period to assess the change of CKD in each cohort.

This study was conducted over six months in a public teaching hospital in Rawalpindi, Pakistan. In compliance with the schedule of routine clinical examinations that are conducted in the hospital, data was collected directly from patient records and the hospital laboratory. In addition, the regular hospital practices did not require further test procedures.

The study involved adult patients with diabetic chronic kidney disease, and the patients' information was retrieved from the hospital information system. Patients were categorised into two cohorts: one received SGLT-2 inhibitors, primarily dapagliflozin, while the other in the pair did not. To be included in the SGLT-2 inhibitor group, patients need to have an eGFR of 30 mL/min/1.73 m^2 and above — at the time of starting on dapagliflozin. Procox continuation was allowed if the patient's eGFR dropped below 30 mL/min/1.73m^2 but not if the patient was on dialysis [11]. For the current comparative study, patients in both groups were matched according to their age, eGFR, and other comparable clinical characteristics at baseline.

The patients included in the study fulfilled the inclusion criteria of type 2 diabetic mellitus patients with kidney disease stages 2 to 4 with an eGFR of 30 mL/min/1.73m2. To participate in the study, participants must have been over the age of 18 years and must have granted their consent to use their health records. Outpatients with T2DM who

embarked on dapagliflozin treatment at any point during the research phase were the only ones considered in the SGLT-2 inhibitor category.

No patient on SGLT-2 inhibitor was recruited in the study if the eGFR was less than 30 mL/min/1.73 m^2. Patients with acute kidney injury (AKI), patients on hemodialysis, patients with an eGFR ≤ 15 mL/min/1.73 m^2, pregnant or lactating women, and patients with non-diabetic kidney diseases that may influence renal outcomes independently were also excluded. Other criteria for excluding patients were the presence of malignant neoplastic diseases, as well as severe and systematic diseases influencing treatment prognosis.

The data was collected through a retrospective review of patient records. During routine visits, the hospital's laboratory conducted tests on blood samples, including serum creatinine and eGFR, while spot urine samples were used to measure the urinary albumin-to-creatinine ratio (ACR). Data collection was conducted at three checkpoints: data at baseline, three months, and after six months follow up. Acute kidney injury was calculated based on a serum creatinine of >1.5 mg/dL and from urine dip used at checkpoints with the proteinuria cutoff level set at ≥30 mg/g. The entire treatment status, together with dosage changes, was recorded properly throughout the study by extracting information on SGLT- 2 inhibitor prescription from the patient's files or prescription record.

The study incorporated three checkpoints or follow-ups, which were used to capture data. The first was done at the onset of the study, that is, after the participants had enrolled. The second intervention was done when participants reached the three-month follow-up, while the third was done when the six-month follow-up was done through routine clinical assessments. This allowed attenuation of the two groups' kidney function variation over time. Urine ACR was the primary endpoint considered in the study to compare patients on SGLT-2 inhibitors and those not on these drugs. Secondary outcomes of frailty were a 6-month decline in the estimated glomerular filtration rate (eGFR) and more severe renal events, which include the development of ESKD or the initiation of dialysis.

All statistical analysis was carried out under IBM SPSS Statistics 26.0. Age, hemoglobin A1c, eGFR, and ACR were expressed by mean ± SD; the gender, eGFR categories, and proteinuria prevalence were expressed in frequency and percentage, respectively. To assess differences between those variables in the SGLT-2 inhibitor group compared to the non-SGLT-2 inhibitor group, mean differences on continuous variables and proportions for categorical variables were calculated and compared using independent samples t-tests and chi-squared tests, respectively. To compare the crude efficacy of the two techniques, standardized mean differences (SMD) were obtained. An SMD of less than 10% was used as a cut point to determine the extent of equivalence between the two groups. The within-group and between-group differences in eGFR and ACR at baseline and 12- and 24-week follow-up visits were evaluated by repeated-measure ANOVA. The difference in eGFR/ACR at 24 weeks from baseline was compared by the two groups using an independent sample t-test. Comparison was also made on the chi-square test of percentage change from the baseline to 24 weeks. Likewise, all tests concerned two-tailed, and $p<0.05$ was adopted as the measure of significance. The findings are reported with a 95% confidence interval where value is available.

The study protocol was reviewed and approved by the institutional review board (IRB) of the hospital CMH Rawalpindi. All participants willingly signed consent forms before the data collection exercise; patients' information was not disclosed throughout the study.

Results

This study analyzed the impact of SGLT-2 inhibitors in comparison to alternative glucose-lowering medications on kidney function, utilizing age-matched patient cohorts. The average age of patients in the SGLT-2 inhibitor group (n = 54) was 63.5 years, comparable to the non-SGLT-2 group (n = 108), with a mean age of 64.3 years. The proportion of women was nearly identical in both groups, with 37.0% in the SGLT-2 group compared to 36.1% in the non-SGLT-2 group. In the SGLT-2 group, hemoglobin A1c levels exhibited minimal variation, recorded at 7.9% (63.3 mmol/mol), whereas the non-SGLT-2 group presented levels of 7.8% (61.7 mmol/mol), indicating a standardized mean difference of 5.1%.

The average kidney function, measured by eGFR, was similar in both groups, with the SGLT-2 group at 67.5

mL/min/1.73 m² and the non-SGLT-2 group at 68.9 mL/min/1.73 m². The eGFR ≥60 mL/min/1.73 m² range encompassed 72.2% of participants in the SGLT-2 group and 73.1% of those not receiving SGLT-2 therapy. The rate of eGFR decline before treatment was higher in the SGLT-2 group (-1.5 mL/min/1.73 m²/year) than the non-SGLT-2 group (-1.2 mL/min/1.73 m²/year). The prevalence of proteinuria was similar in both groups, with 29.6% in the SGLT-2 group and 28.7% in the non-SGLT-2 group.

Table 1. Demographic and clinical characteristics of the study sample

Characteristics	SGLT-2 Inhibitor Group (n = 54)	Non-SGLT-2 Inhibitor Group (n = 108)	Standardized Mean Difference (%)
Age, years	63.5 ± 10.8	64.3 ± 11.2	5.3
Women, n (%)	20 (37.0%)	39 (36.1%)	0.9
Hemoglobin A1c, %	7.9 ± 1.3	7.8 ± 1.4	5.1
Hemoglobin A1c, mmol/mol	63.3 ± 14.2	61.7 ± 15.6	4.8
eGFR, mL/min/1.73 m²	67.5 ± 18.0	68.9 ± 17.6	2.8
eGFR ≥60 mL/min/1.73 m², n (%)	39 (72.2%)	79 (73.1%)	2.5
eGFR <60 mL/min/1.73 m², n (%)	15 (27.8%)	29 (26.9%)	1.3
eGFR 45–59 mL/min/1.73 m², n (%)	12 (22.2%)	22 (20.4%)	3.1
eGFR <45 mL/min/1.73 m², n (%)	3 (5.6%)	7 (6.5%)	2.7
Proteinuria, n (%)	16 (29.6%)	31 (28.7%)	2.4

After 24 weeks, the SGLT-2 inhibitor group showed a more significant fall in eGFR than the group taking other glucose-lowering agents. At the outset, the mean eGFR for the SGLT-2 group stood at 67.5 mL/min/1.73 m², falling to 65.2 mL/min/1.73 m² by 24 weeks, illustrating a 3.4% total drop. The non-SGLT-2 group showed a less significant drop, from 68.9 mL/min/1.73 m² at baseline to 68.2 mL/min/1.73 m² at 24 weeks, with a total change of just 1.0%. The standardized mean difference between groups has slightly risen over time, suggesting a more significant effect of SGLT-2 inhibitors on reducing eGFR.

Table 2. eGFR at baseline and checkpoints

Period	Cases: SGLT2 Inhibitor Group (n = 54)	Cohort: Other Glucose-Lowering Drugs Group (n = 108)	Standardized Mean Difference (%)
eGFR (mL/min/1.73 m²) at Baseline	67.5 ± 18.0	68.9 ± 17.6	2.8
eGFR at 12 Weeks	66.3 ± 17.7	68.5 ± 17.5	3.5
eGFR at 24 Weeks	65.2 ± 17.5	68.2 ± 17.3	4.4
Total Change in eGFR (24 weeks)	-3.4%	-1.0%	2.9

The group using SGLT-2 inhibitors showed a more significant decrease in urinary albumin-to-creatinine ratio (ACR) than the group on other types of glucose-lowering medications. At the outset, the SGLT-2 group showed an ACR of 300 mg/g, which fell by 20% to 230 mg/g by the 24th week. On the other hand, the non-SGLT-2 group reported a minimal reduction of 10%, from 305 mg/g at baseline to 275 mg/g. The standardized mean difference between the groups escalated from 1.7% at baseline to 8.9% in 24 weeks, illustrating a more significant effect of SGLT-2 inhibitors on reducing ACR and reflecting improved kidney function.

Table 3: Change in Urinary Albumin-to-Creatinine Ratio (ACR) and eGFR Over Time

Period	Cases: SGLT2 Inhibitor Group (n = 54)	Cohort: Other Glucose-Lowering Drugs Group (n = 108)	Standardized Mean Difference (%)
ACR at baseline (mg/g)	300 ± 95	305 ± 98	1.7
ACR at 12 Weeks (mg/g)	240 ± 87	285 ± 90	6.5
ACR at 24 Weeks (mg/g)	230 ± 85	275 ± 89	8.9
Total Change in ACR (%)	-20%	-10%	5.3

Discussions

Our findings highlight the therapeutic utility of SGLT2 inhibitors for preventing the worsening of chronic kidney disease in patients with T2DM. The results are similar to clinical trials by Perkovic et al. [12] and Siddiqui et al. [13], showing that the benefits seen in controlled settings are possible in the field. Previous RCTs of SGLT2 inhibitors have gradually included patients with varying stages of renal dysfunction, which our study adds to [14]. Several processes, such as the correction of glomerular hyperfiltration, have been put forward to explain the kidney-sparing effects of these inhibitors. One of the findings in our analysis that is in harmony with clinical trials is that patients experience an initial decline in eGFR but are later stabilized [15]. To the best of our knowledge, our study is one of the very few that have used laboratory measurements of renal function. However, large-scale epidemiological trials have compared SGLT2 inhibitors with other antihyperglycaemic drugs. What made it possible to observe changes in eGFR trajectories and to match the patients according to their rate of kidney function decline before treatment was the availability of multiple eGFR measurements before and after the initiation of SGLT2 inhibitors. This methodological approach did help in attaining equality in the primary risk of development of chronic renal disease in the groups under the study, which is the factor often overlooked in randomized clinical trials and observational studies. Our results, therefore, supported our hypothesis that the rate of eGFR decline was similar before starting either treatment. However, the decline significantly lessened once SGLT2 inhibitors were started, proving that these drugs are useful for the preservation of kidneys.

As mentioned in previous CVD-REAL studies, SGLT2 inhibitors have been associated with a reduced risk of cardiovascular events, heart failure, and mortality [16,17]. The initiation of SGLT2 inhibitors was always found to be associated with a reduced risk of significant kidney outcomes, although event rates for kidney outcomes differed across countries [18]. Thus, our study is critical because it demonstrates the experience of using SGLT2 inhibitors in a more diverse population of patients. However, many trials recruit highly selected patients whose characteristics may not fully mimic those of true-world patients. This gap between clinical trials and regular practice has been acknowledged before, where most individuals with diabetes receiving usual care are not eligible for large SGLT2 inhibitor trials.

This cohort study also shows that the use of real-world registry data, together with the results of clinical trials, can improve the understanding of the drug's effectiveness and applicability to everyday clinical

practice. Even though the presented results are promising, several limitations must be mentioned. However, we appreciate that because of the use of strict statistical matching methods to match patients based on eGFR and other baseline covariates, the risk of unmeasured confounding cannot be entirely ruled out. Further, the lack of socioeconomic information about all the participants, including income or educational level, could have influenced the results. Furthermore, it is necessary to acknowledge that there is a possibility of bias in creatinine measurements, performed during both acute infection and consecutive follow-ups, in our study. However, it is an observational study reflecting real-life clinical practice. Moreover, our analysis only ranked treatments based on their effectiveness with having consideration of safety information, though previous studies have shown that SGLT2 inhibitors may have renal protective effects against AKI [19,20]. Finally, our ability to examine this important marker of kidney disease was limited by the dearth of albuminuria information in our sample population. Our study differs from previous work in that it looks at the impact of SGLT2 inhibitors in a real-world population, strengthening the argument for their use in managing chronic renal disease. Prior studies have shown that SGLT2 inhibitors slow down eGFR reduction trials. It was especially significant that CREDENCE included patients with chronic renal disease and type 2 diabetes, as it showed that SGLT2 inhibitors significantly reduce cardiovascular and kidney events in patients with moderately to severely reduced kidney function.

Conclusions

The present research findings highlight the growing literature on the potential of SGLT2 inhibitors for the prevention of CKD progression. Although these medications are typically not recommended for individuals with an eGFR under 45 mL/min per 1.73 m^2 due to reduced glucose-lowering efficacy, the results indicate that the advantages of these drugs may still be seen in patients with lower eGFR values. SGLT2 inhibitors could potentially play a more significant role in managing diabetic kidney disease among a broader group as clinical guidelines evolve.

Acknowledgements: ***None***
Conflict of Interest: ***None***

Authors Contribution:

Author	*Contribution*
MS	*1. Substantial Contribution to study design, analysis, acquisition of Data* *2. Manuscript Writing* *3. Has given Final Approval of the version to be published*
FI	*1. Substantial Contribution to study design, acquisition and interpretation of Data* *2. Critical Review and Manuscript Writing* *3. Has given Final Approval of the version to be published*
AS	*1. Substantial Contribution to acquisition and interpretation of Data* *2. Manuscript Writing* *3. Has given Final Approval of the version to be published*
SS	*1. Contributed to Data Collection and Analysis* *2. Critically reviewed the article* *3. Has given Final Approval of the version to be published*
NK	*1. Substantial Contribution to study design and Data Analysis* *2. Manuscript Writing and Critical review of the article* *3. Has given Final Approval of the version to be published*
MFH	*1. Contributed to study concept and Data collection* *2. Critical review of the manuscript* *3. Has given Final Approval of the version to be published*

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