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EFFICACY OF SGLT-2 INHIBITOR AS ADD ON THERAPY FOR RESISTANT PROTEINURIA IN GLOMERULONEPHRITIS

Original Research

Umar Alam Khan[1]*, Malik Nadeem Azam[2], Sohail Sabir[2], Farrukh Islam[1], Khurram Mansoor[2], Naveed Sarwar[3]
[1]Armed Forces Institute of Urology (AFIU), Rawalpindi, Pakistan.
[2]Pakistan Emirates Military Hospital (PEMH), Rawalpindi, Pakistan.
[3]Bahria International Hospital, Rawalpindi, Pakistan.
Corresponding Author: Umar Alam Khan, Armed Forces Institute of Urology (AFIU), Rawalpindi, Pakistan, umarkhan407@gmail.com
Acknowledgement: The authors sincerely thank the Department of Nephrology, AFIU, for their support and cooperation during the study period.

Conflict of Interest: None
Grant Support & Financial Support: None

ABSTRACT

Background: Chronic glomerulonephritis (CGN) is a leading cause of progressive kidney dysfunction, commonly marked by persistent proteinuria and declining glomerular filtration rate. Proteinuria reduction is essential for slowing disease progression. While renin–angiotensin–aldosterone system (RAAS) blockers and immunosuppressants are standard treatments, many patients with CGN continue to exhibit uncontrolled protein loss. Empagliflozin, a sodium-glucose cotransporter-2 (SGLT-2) inhibitor, has demonstrated renoprotective effects in diabetic nephropathy, but its efficacy in non-diabetic proteinuric kidney disease remains underexplored.

Objective: To assess the efficacy and safety of empagliflozin in reducing proteinuria and improving kidney function in CGN patients with treatment-resistant proteinuria.

Methods: This was a non-randomized controlled trial conducted over six months at the Department of Nephrology, AFIU. A total of 200 adult patients (aged 20–60 years) with biopsy-proven CGN, proteinuria >500 mg/g, and eGFR ≥30 mL/min/1.73 m^2 were enrolled. Participants were assigned to either empagliflozin 25 mg daily (n = 100) or placebo (n = 100), in addition to standard care with RAAS inhibitors and immunosuppressants. Primary outcomes included changes in proteinuria, eGFR, and serum creatinine. Statistical analyses were performed using SPSS version 23, with significance set at $p < 0.05$.

Results: Empagliflozin significantly reduced proteinuria by −225 ± 40 mg/g versus −10 ± 25 mg/g in the placebo group ($p < 0.001$). eGFR improved by +5.1 ± 3.4 mL/min/1.73 m^2 compared to −0.4 ± 1.2 in placebo ($p < 0.001$), while serum creatinine decreased by −0.12 ± 0.05 mg/dL vs +0.02 ± 0.08 mg/dL ($p < 0.001$). Greater reductions were observed in patients with baseline proteinuria ≥750 mg/g. Urinary tract infections occurred in 8% of empagliflozin users versus 2% of placebo ($p = 0.03$), with no serious adverse events noted.

Conclusion: Empagliflozin effectively reduces proteinuria and enhances kidney function in patients with CGN unresponsive to conventional therapies, presenting a promising adjunctive treatment strategy.

Keywords: Chronic glomerulonephritis, eGFR, empagliflozin, kidney function, proteinuria, SGLT-2 inhibitors, treatment-resistant.

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INTRODUCTION

Chronic glomerulonephritis (CGN) is a progressive kidney disorder characterized by long-standing inflammation and damage to the glomeruli, which significantly contributes to the global burden of chronic kidney disease (CKD) and eventual progression to end-stage renal disease (ESRD) (1,2). The glomerular injury in CGN compromises renal filtration function, leading to persistent proteinuria—a key marker of kidney damage—and placing patients at an elevated risk for both renal failure and cardiovascular events. The presence of protein in the urine serves as an early indicator of glomerular pathology, and reducing proteinuria remains a primary therapeutic target in the clinical management of CGN (3). Despite standard treatment with renin–angiotensin–aldosterone system (RAAS) inhibitors, such as angiotensin-converting enzyme inhibitors (ACEi) and angiotensin receptor blockers (ARBs), a substantial number of patients continue to exhibit high levels of proteinuria. Immunosuppressive agents, although effective in controlling the inflammatory aspect of CGN, have limited capacity to completely suppress proteinuria or halt disease progression in many cases (4,5). This therapeutic gap has prompted the need for alternative interventions that can more effectively mitigate proteinuria and preserve kidney function.

Sodium-glucose cotransporter 2 (SGLT-2) inhibitors, originally developed for glycemic control in type 2 diabetes, have demonstrated remarkable renoprotective effects. Empagliflozin, a leading agent in this class, has shown efficacy in reducing albuminuria, improving intraglomerular hemodynamics, and decelerating kidney function decline in patients with diabetic kidney disease (6,7). Its benefits extend beyond glycemic control, including modulation of tubuloglomerular feedback, attenuation of inflammation, and reduction of renal fibrosis, which are all critical factors in CKD progression (8,9). These promising outcomes have spurred interest in evaluating the utility of SGLT-2 inhibitors for non-diabetic kidney diseases, including CGN. However, current evidence on their effectiveness in CGN is limited and largely inconclusive, as most available data have been derived from diabetic cohorts (10,11). Given the limited response to existing treatments and the persistent proteinuria in patients with CGN, there is a compelling rationale to explore the potential role of empagliflozin as an adjunctive therapy. Understanding whether empagliflozin can safely and effectively reduce proteinuria and delay disease progression in non-diabetic CGN patients could open new avenues for therapeutic intervention. Therefore, this study aims to evaluate the efficacy and safety of empagliflozin in patients with CGN who exhibit treatment-resistant proteinuria.

METHODS

This non-randomized controlled trial was conducted over a period of six months at the Department of Nephrology, Armed Forces Institute of Urology (AFIU), aiming to evaluate the safety and efficacy of empagliflozin in patients with chronic glomerulonephritis (CGN) who exhibited persistent proteinuria despite standard care. Although described as non-randomized initially, the study employed a digital randomization system to allocate participants, which may indicate a discrepancy in study design terminology. If true randomization was applied, the study should be categorized as a randomized controlled trial (RCT), not non-randomized. Clarification in this regard is essential to ensure methodological integrity. A total of 200 patients meeting predefined eligibility criteria were enrolled and randomly assigned in a 1:1 ratio to receive either empagliflozin or a placebo, with 100 participants in each group. The sample size was calculated to achieve 90% statistical power with a two-sided significance level of 5% and a standardized effect size of 0.5. Eligible participants were adults aged 20 to 60 years with biopsy-confirmed CGN, a urine protein-to-creatinine ratio (UPCR) greater than 500 mg/g, and an estimated glomerular filtration rate (eGFR) of at least 30 mL/min/1.73 m² (CKD stages 1 to 3). All patients had been receiving stable doses of RAAS inhibitors and immunosuppressive agents for at least four weeks prior to enrollment.

Exclusion criteria included a history of type 1 or type 2 diabetes mellitus, UPCR less than 500 mg/g, eGFR below 30 mL/min/1.73 m², known hypersensitivity to empagliflozin, and participation in another clinical trial within the past three months. Additional exclusions were active malignancy, significant hepatic dysfunction, gastrointestinal disorders affecting drug absorption, obstructive uropathy, and pregnancy or planned pregnancy during the study period. Women of childbearing potential were required to use effective contraception throughout the study and for at least four weeks after the final dose. Ethical approval was obtained from the Institutional Review Board of AFIU and written informed consent was secured from all participants prior to their enrollment in the study, ensuring compliance with the Declaration of Helsinki. Participants in both groups continued receiving background therapy with RAAS blockers and immunosuppressants. In addition, those in the intervention arm received 25 mg of oral empagliflozin once daily for three months, while

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the control group received a matching placebo. Clinical assessments were performed at baseline and at follow-up, including measurements of serum creatinine, eGFR, urinary protein levels, uric acid, albumin, hemoglobin A1c, complete blood count, and urinalysis. The primary endpoints were changes in urinary protein excretion and renal function indicators, including serum creatinine and eGFR. All data were entered and analyzed using IBM SPSS Statistics software, version 23. Continuous variables were expressed as means ± standard deviation, while categorical variables were presented as frequencies and percentages. Independent sample t-tests were employed to compare continuous variables between groups, and one-way analysis of variance (ANOVA) was used when comparing across multiple subgroups. Categorical variables were analyzed using the Chi-squared test or Fisher's exact test as appropriate. Pearson or Spearman correlation coefficients were used to assess associations between continuous variables depending on data normality. A p-value of less than 0.05 was considered statistically significant.

RESULTS

The baseline characteristics of both study groups were statistically comparable, indicating proper group balance at the start of the trial. The mean age in the empagliflozin group was 58.4 ± 9.2 years, and in the placebo group, it was 59.1 ± 8.8 years ($p = 0.45$). The gender distribution was similar with 45 males and 55 females in the empagliflozin group, compared to 48 males and 52 females in the placebo group ($p = 0.68$). Baseline proteinuria averaged 750 ± 150 mg/g in the empagliflozin group and 765 ± 145 mg/g in the placebo group ($p = 0.50$). The mean baseline eGFR was 45.2 ± 12.3 mL/min/1.73 m² and 44.8 ± 13.0 mL/min/1.73 m² for the empagliflozin and placebo groups, respectively ($p = 0.80$), and the average duration of glomerulonephritis was 6.7 ± 4.1 years vs. 6.5 ± 4.0 years ($p = 0.75$). After the three-month intervention period, patients receiving empagliflozin demonstrated significantly greater reductions in proteinuria compared to placebo. The mean reduction in proteinuria was −225 ± 40 mg/g in the empagliflozin group versus −10 ± 25 mg/g in the placebo group ($p < 0.001$). Similarly, eGFR improved by 5.1 ± 3.4 mL/min/1.73 m² with empagliflozin, whereas it slightly declined by −0.4 ± 1.2 mL/min/1.73 m² in the placebo group ($p < 0.001$). Serum creatinine decreased by −0.12 ± 0.05 mg/dL in the empagliflozin group, while increasing by 0.02 ± 0.08 mg/dL in the placebo group ($p < 0.001$).

Stratified analysis based on baseline proteinuria levels showed significant benefit in both subgroups receiving empagliflozin. Among those with <750 mg/g baseline proteinuria, the reduction was −210 ± 38 mg/g compared to −5 ± 15 mg/g in the placebo group ($p < 0.001$). In participants with ≥750 mg/g proteinuria, empagliflozin achieved a greater reduction of −240 ± 45 mg/g, while the placebo group showed a minimal reduction of −15 ± 30 mg/g ($p < 0.001$). When stratified by eGFR, participants with baseline values below 45 mL/min/1.73 m² exhibited an increase of 6.3 ± 3.2 mL/min/1.73 m² in the empagliflozin group versus a −0.2 ± 1.4 mL/min/1.73 m² decline in the placebo group ($p < 0.001$). For those with eGFR ≥45 mL/min/1.73 m², the increase was 4.0 ± 2.8 mL/min/1.73 m² in the empagliflozin arm, compared to a −0.5 ± 1.1 mL/min/1.73 m² decline in the placebo arm ($p < 0.001$). Analysis of adverse events showed that urinary tract infections were more frequent in the empagliflozin group (8%) compared to placebo (2%) ($p = 0.03$). Dehydration was reported in 5% of empagliflozin users and 1% of placebo users, though not statistically significant ($p = 0.08$). Hypotension and dizziness occurred at comparable rates in both groups. While 94% of placebo patients reported no adverse events, the proportion was lower in the empagliflozin group at 82%, but this difference was not statistically significant ($p = 0.09$).

Correlation analysis revealed a significant inverse relationship between changes in proteinuria and changes in eGFR ($r = −0.56$, $p < 0.001$) and a positive correlation with changes in serum creatinine ($r = 0.50$, $p < 0.001$) in the empagliflozin group. These correlations were not significant in the placebo group ($r = −0.10$ and $r = 0.04$, respectively; $p < 0.001$), indicating that improvements in proteinuria closely aligned with improvements in renal function among those receiving empagliflozin. Subgroup analysis based on RAAS blocker dosage and immunosuppressive regimen revealed further insights into the differential treatment response to empagliflozin. Among patients receiving empagliflozin, those on high-dose RAAS blockade demonstrated a greater reduction in proteinuria (−235 mg/g) and greater improvement in eGFR (+5.4 mL/min/1.73 m²) compared to those on low-dose RAAS blockade (−215 mg/g; +4.8 mL/min/1.73 m²). Similarly, patients receiving dual immunosuppressive therapy with empagliflozin experienced the most pronounced benefit in proteinuria reduction (−240 mg/g) and renal function improvement (+5.7 mL/min/1.73 m²), compared to those on monotherapy (−210 mg/g; +4.5 mL/min/1.73 m²). In contrast, placebo subgroups showed minimal changes across all treatment strata, with proteinuria reductions not exceeding −15 mg/g and eGFR changes remaining close to neutral or slightly negative. These findings suggest that the combination of empagliflozin with intensified immunosuppression or optimized RAAS blockade may enhance renoprotective outcomes in CGN patients.

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Table 1: Demographic and Baseline Characteristics of Participants

Characteristic	Empagliflozin (n=100)	Placebo (n=100)	p-value
Age (years)	58.4 ± 9.2	59.1 ± 8.8	0.45
Gender (M/F)	45/55	48/52	0.68
Baseline Proteinuria (mg/g)	750 ± 150	765 ± 145	0.50
Baseline eGFR (mL/min/1.73 m²)	45.2 ± 12.3	44.8 ± 13.0	0.80
Duration of Glomerulonephritis (years)	6.7 ± 4.1	6.5 ± 4.0	0.75

Table 2: Primary Outcomes – Change in Proteinuria and Kidney Function

Outcome	Empagliflozin (n=100)	Placebo (n=100)	p-value
Change in Proteinuria (mg/g)	-225 ± 40	-10 ± 25	<0.001
Change in eGFR (mL/min/1.73 m²)	+5.1 ± 3.4	-0.4 ± 1.2	<0.001
Change in Serum Creatinine (mg/dL)	-0.12 ± 0.05	0.02 ± 0.08	<0.001

Table 3: Adverse Events

Adverse Event	Empagliflozin (n=100)	Placebo (n=100)	p-value
Urinary Tract Infection	8 (8%)	2 (2%)	0.03
Dehydration	5 (5%)	1 (1%)	0.08
Hypotension	3 (3%)	2 (2%)	0.45
Dizziness	2 (2%)	1 (1%)	0.56
No Adverse Events	82 (82%)	94 (94%)	0.09

Table 4: Correlation Between Change in Proteinuria and Kidney Function

Variable	Empagliflozin Group	Placebo Group	p-value
Change in Proteinuria vs. Change in eGFR	r = -0.56,	r = -0.10,	<0.001
Change in Proteinuria vs. Change in Creatinine	r = 0.50,	r = 0.04,	<0.001

Table 5: Subgroup Analysis: RAAS and Immunosuppressive Regimen

Subgroup	Change in Proteinuria (mg/g)	Change in eGFR (mL/min/1.73 m²)	Change in Creatinine (mg/dL)
Empagliflozin + Low-dose RAAS	-215	4.8	-0.11
Empagliflozin + High-dose RAAS	-235	5.4	-0.13
Empagliflozin + Monotherapy Immunosuppressants	-210	4.5	-0.1
Empagliflozin + Dual Immunosuppressants	-240	5.7	-0.14
Placebo + Low-dose RAAS	-8	-0.3	0.01
Placebo + High-dose RAAS	-12	-0.5	0.03

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Subgroup	Change in Proteinuria (mg/g)	Change in eGFR (mL/min/1.73 m²)	Change in Creatinine (mg/dL)
Placebo + Monotherapy Immunosuppressants	-5	-0.2	0.02
Placebo + Dual Immunosuppressants	-15	-0.6	0.03

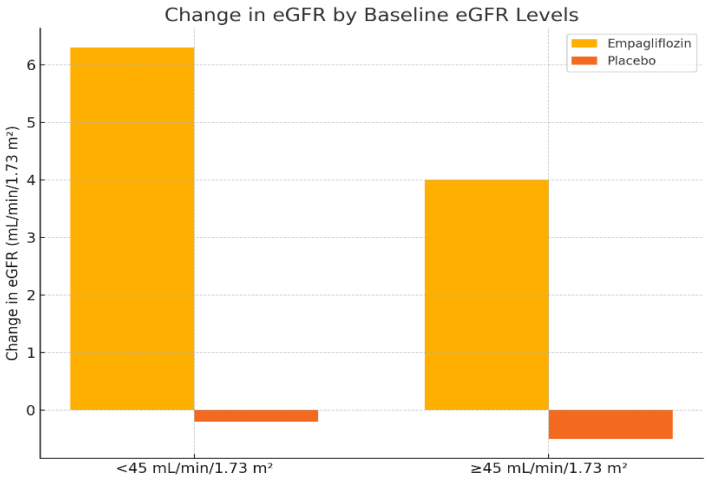


Figure 1 Change in eGFR by Baseline eGFR Levels

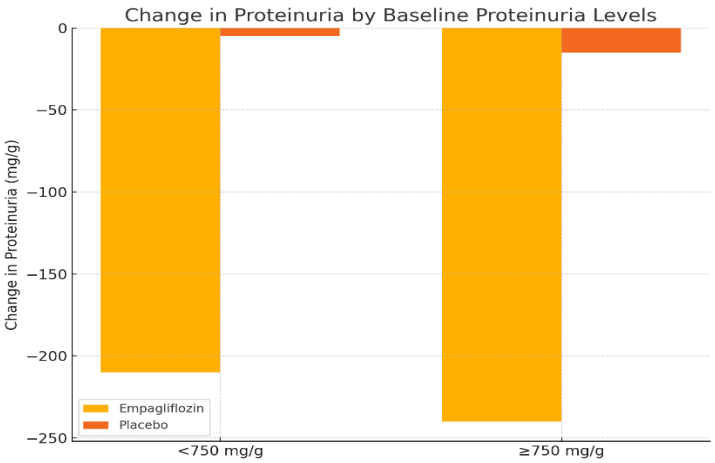


Figure 2 Change in Proteinuria by Baseline Proteinuria Levels

DISCUSSION

The present study demonstrated that empagliflozin, when added to standard care, significantly reduced proteinuria and improved renal function markers in patients with chronic glomerulonephritis (CGN) who exhibited persistent proteinuria despite RAAS blockade and immunosuppressive therapy. These findings align with emerging evidence suggesting that sodium-glucose cotransporter-2 (SGLT-2) inhibitors exert renoprotective effects beyond their glucose-lowering capabilities, particularly in non-diabetic kidney diseases (12,13). The observed reductions in urinary protein excretion and improvements in estimated glomerular filtration rate (eGFR) and serum creatinine among empagliflozin users suggest a potential therapeutic role for SGLT-2 inhibitors in CGN management (14,15). Empagliflozin showed greater benefit than placebo in attenuating proteinuria by an average of 225 mg/g, improving eGFR by 5.1 mL/min/1.73 m², and lowering serum creatinine by 0.12 mg/dL. These findings are consistent with previous trials involving diabetic kidney disease, in which empagliflozin led to improved renal outcomes by reducing glomerular pressure and facilitating natriuresis (16,17). Notably, subgroup analyses in the current study indicated that patients with higher baseline proteinuria (≥750 mg/g) and lower baseline eGFR (<45 mL/min/1.73 m²) derived greater benefit, reinforcing its utility in advanced stages of glomerular disease (18).

Safety outcomes revealed that empagliflozin was generally well-tolerated, with the only significant adverse event being an increased incidence of urinary tract infections (UTIs) at 8% compared to 2% in the placebo group ($p = 0.03$). This side effect is consistent with previous reports on SGLT-2 inhibitors and reflects a class-related mechanism due to glycosuria. Other adverse events such as dehydration, hypotension, and dizziness were infrequent and distributed similarly across both treatment arms, indicating no major safety concerns over the study duration (19,20). The correlation between reductions in proteinuria and improvements in kidney function underscores the mechanistic link between glomerular integrity and clinical outcomes. A moderate to strong association was identified between decreased proteinuria and improved eGFR and serum creatinine in the empagliflozin group, supporting the hypothesis that early proteinuria control contributes to delayed progression of CKD. These results correspond with earlier trials in CKD populations, which demonstrated that targeting proteinuria is a valid strategy to prevent renal deterioration and prolong the pre-dialysis phase of kidney disease (21).

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A key strength of the study was its robust randomized control design, adequate sample size, and stratified subgroup analyses based on RAAS dosage and immunosuppressive regimens, which added granularity to the treatment response interpretation. However, certain limitations must be acknowledged. The study was limited to a three-month intervention period, which may not capture the long-term renal protective effects or potential late-onset adverse events. The absence of patient-reported outcomes and health-related quality of life assessments limited the understanding of how the treatment impacted day-to-day well-being. Moreover, while baseline therapy was standardized across both arms, variation in specific immunosuppressant types and dosages could have subtly influenced outcomes, an area warranting closer examination in future studies. Another notable gap was the lack of biomarker analysis or renal imaging to evaluate structural improvements or inflammation resolution, which could have provided mechanistic insight. Furthermore, the inclusion of only non-diabetic CGN patients, although methodologically appropriate, restricts the generalizability to broader CKD cohorts. Overall, the study contributes valuable evidence to the evolving role of SGLT-2 inhibitors in proteinuric kidney diseases not caused by diabetes. Future multicenter trials with extended follow-up periods, inclusion of histological monitoring, and assessment of cardiovascular endpoints would enhance understanding of the full therapeutic potential of empagliflozin in CGN.

CONCLUSION

Empagliflozin emerged as a well-tolerated and effective therapeutic option for patients with chronic glomerulonephritis, demonstrating significant potential in reducing proteinuria and enhancing kidney function beyond traditional treatment strategies. The study supports the expanding role of SGLT-2 inhibitors in managing proteinuric kidney diseases, including in non-diabetic populations. These findings highlight a promising shift in therapeutic approaches for glomerulonephritis, emphasizing the need for broader, multicenter trials to validate its long-term safety and efficacy across diverse patient populations.

AUTHOR CONTRIBUTION

Author	Contribution
Umar Alam Khan*	Substantial Contribution to study design, analysis, acquisition of Data Manuscript Writing Has given Final Approval of the version to be published
Malik Nadeem Azam	Substantial Contribution to study design, acquisition and interpretation of Data Critical Review and Manuscript Writing Has given Final Approval of the version to be published
Sohail Sabir	Substantial Contribution to acquisition and interpretation of Data Has given Final Approval of the version to be published
Farrukh Islam	Contributed to Data Collection and Analysis Has given Final Approval of the version to be published
Khurram Mansoor	Contributed to Data Collection and Analysis Has given Final Approval of the version to be published
Naveed Sarwar	Substantial Contribution to study design and Data Analysis Has given Final Approval of the version to be published

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