Efficacy of Finerenone in Slowing the Progression of Chronic Kidney Disease in Type 2 Diabetes.

Sibgha Ahmed[1*], Mateen Akram[2], Shazia Abbas[3], Hina Francis[4], Sundus Shahzad[5], Ruqayya Liaqat[6].

ABSTRACT:

Objective: To evaluate short-term effects and safety of Finerenone in clinical practice of nephrology.

Methodology: A prospective observational study was done at the Department of Nephrology, Shaikh Zayed Hospital, Lahore, Pakistan from 01 September 2025 to 30 March 2026. 100 adults (18-80 years) with type 2 diabetes and chronic kidney disease (CKD) (eGFR ≥ 25 mL/min/1.73 m², Urine Albumin-Creatinine Ratio (UACR) ≥ 30 mg/g), who were on stable ACE inhibitor/ARB therapy, were consecutively enrolled, with baseline potassium levels of ≤ 4.8 mmoL/L. Follow-up visits were conducted after 1 and 3 months. Paired t-tests and Wilcoxon signed-rank tests were used to test the changes from baseline for continuous variables and UACR, respectively, at $p<0.05$.

Results: The mean age was 64.8±8.0 years and 70% of the population was male. Baseline eGFR was 45.0±10.7 mL/min/1.73 m² and median UACR was 800 mg/g [IQR 330-1643]. At 1 month, 95% and 3 month, 92% of the children were attending. UACR decreased to 607 [272-1277] at 1 month and 540 [244-1142] at 3 months (both $p<0.001$). Mean eGFR declined to 43.9±11.2 at 1 month and 41.9±11.8 at 3 months (both $p<0.001$). Mean potassium increased from 4.35±0.24 to 4.56±0.47 at 1 month ($p<0.001$) and 4.47±0.43 at 3 months ($p=0.004$); potassium >5.5 mmol/L occurred in 5.3% and 2.2%, respectively. The combined outcome of kidney was seen in 7% and hyperkalaemia requiring ER/hospitalization occurred in 2% in 3 months.

Conclusion: Finerenone was associated with early albuminuria reduction and acceptable short-term safety with potassium monitoring.

Keywords: Albuminuria; Chronic kidney disease; Finerenone; Hyperkalaemia; Type 2 diabetes mellitus.

Cite as: Ahmed S, Akram M, Abbas S, Francis H, Shahzad S, Liaqat R. Efficacy of Finerenone in Slowing the Progression of Chronic Kidney Disease in Type 2 Diabetes. J Muhammad Med Coll. 17(1) April-Oct 2026

Introduction:

Chronic kidney disease (CKD) is an ongoing disease that causes the kidneys to gradually fail in function. Diabetes is a cause of more than 10% of chronic kidney disease worldwide and is one of the largest contributors of chronic kidney disease worldwide, and is rising.[1] Diabetic kidney disease is often present in type 2 diabetes, and can involve a significant number of patients over time.[2] It is also one of the main causes of kidney failure.[3] Treatments have evolved in the last few years from just controlling blood sugar and blood pressure to including medicines that protect the kidneys. Current guidance suggests a multi-layered approach to treatment.[4] Another valuable treatment is finerenone, which was also evaluated in the EMPA-KIDNEY trial and provided additional support for the benefits of SGLT2 inhibition for chronic kidney disease (CKD) in a large population of patients with CKD.[5] It's a non-steroidal mineralocorticoid receptor antagonist which acts on pathways of inflammation and fibrosis in the kidney. In pooled clinical trial data, finerenone lowered kidney and cardiovascular outcomes in patients with type 2 diabetes and chronic kidney disease.[6] Pakistan is facing a double burden; diabetes is prevalent and most of the patients are referred late with complications. Many facilities are still not using UACR or eGFR for screening. The expense of laboratory tests, cost of medicines, and poor follow-up can also postpone diagnosis and also decrease the advantage of the safety measures medicines. Local real-world data are thus valuable for better safe use of newer kidney-protective treatments and interventions in practice.

For current study we assessed finerenone in clinical practice in adults with type 2 diabetes and chronic kidney disease. Patients were followed up at 1 month and 3 months after treatment commenced. The aim was to assess early kidney outcomes, such as changes in eGFR and albuminuria, and also safety, particularly of the changes in potassium levels. This is key as most evidence available is from large trials and higher resource settings. Pakistan requires data from the local environment for guidance in actual nephrology practice with regard to monitoring plans and treatment decisions.

Methodology:

This prospective observational study was carried out in the Department of Nephrology, Shaikh Zayed Hospital, Lahore, Pakistan during 01 September 2025 to 30 March 2026. Ethical approval was obtained from the Shaikh Zayed Med-

1. PG Trainee registrar , Nephrology, Shaikh Zayed Hospital, Lahore.
2. Prof. and Head of Nephrology, Shaikh Zayed Hospital, Lahore.
3. PG Trainee registrar , Nephrology, Shaikh Zayed Hospital, Lahore.
4. PG Trainee registrar , Nephrology, Shaikh Zayed Hospital, Lahore.
5. PG Trainee registrar , Nephrology, Shaikh Zayed Hospital, Lahore.
6. PG Trainee registrar , Nephrology, Shaikh Zayed Hospital, Lahore.

**=corresponding author :*

Email: sibghaahmed97@gmail.com

Received: June 2026, Revised: August 2026

Accepted: September 2026

ical Complex, Lahore (TERC ID: TERC/SC/INT/2025/543; dated: 30-09-2025).

Recruitment was done consecutively and stopped once 100 patients were enrolled. Each enrolled patient was then followed for 3 months, with planned assessments at 1 month and 3 months after starting finerenone. The sample size was calculated using the WHO single-proportion formula, $n = Z^2 p(1-p)/d^2$. Taking an anticipated proportion of 17.8% for the primary kidney outcome from the finerenone arm of the FIDELIO-DKD trial, with 95% confidence level and 7.5% absolute precision, the calculated sample size was 99.9, which was rounded to 100 participants.[7] Non-probability consecutive sampling was used. Adults aged 18–80 years with type 2 diabetes and CKD were included if baseline eGFR was ≥25 mL/min/1.73 m² and albuminuria was present (UACR ≥30 mg/g). Patients had to be on a stable ACE inhibitor or ARB dose for at least 4 weeks, and finerenone had to be started by the treating physician as part of routine care. Baseline serum potassium had to be ≤4.8 mmol/L. Patients were excluded if they were already on dialysis or had a prior kidney transplant, had baseline potassium >4.8 mmol/L, had predominant non-diabetic kidney disease, could not stop spironolactone/eplerenone, were using contraindicated strong CYP3A4 inhibitors/inducers, had significant liver disease, were pregnant/breastfeeding, or had serious illness likely to prevent follow-up.

Data were collected prospectively during routine clinic visits after consent. At baseline, age, sex, diabetes duration, blood pressure, weight, comorbidities, and current medicines (ACEi/ARB, SGLT2 inhibitor, statin, and others) were recorded, along with finerenone start date and dose. Baseline labs included eGFR (calculated using CKD-EPI 2021), UACR (spot urine, mg/g), serum potassium, and HbA1c, using hospital records and lab reports for confirmation. Follow-up was done at 1 month and 3 months, with repeat recording of adherence and medication changes, blood pressure, weight, and labs (eGFR, UACR, potassium). Adverse events (especially hyperkalaemia), ER visits/hospitalizations, dialysis start, and kidney transplant were documented. If eGFR decreased by ≥40% from baseline, a repeat test was arranged within 4–8 weeks to confirm a sustained change. The primary composite kidney outcome was the first occurrence of kidney failure (chronic dialysis or kidney transplant), sustained ≥40% eGFR decline confirmed after ≥4 weeks, or sustained eGFR <15 mL/min/1.73 m² confirmed after ≥4 weeks. Hyperkalaemia was defined as serum potassium ≥5.5 mmol/L or any level requiring emergency care or hospitalization. Monitoring and dose decisions were made in routine care and were aligned with trial guidance where dose increase after 1 month is considered if potassium is ≤4.8 mmol/L and eGFR is stable, and treatment is withheld if potassium exceeds 5.5 mmol/L and restarted when potassium is ≤5.0 mmol/L.

Data Analysis:

The statistical analysis was done on SPSS. While the data was normally distributed, continuous variables were presented as mean and standard deviation. Median and interquartile ranges were used for skewed variables (UACR). Categorical variables were reported as frequency and percentages. Baseline, 1 month and 3 month clinical and laboratory values were compared after initiating finerenone. The baseline values were compared to the values at 1 month and at 3 months. Normally distributed continuous variables were analyzed using paired t-test. Wilcoxon signed-rank test was performed for skewed variables (e.g., UACR, and percentage change in UACR). Paired categorical variables were analyzed using the McNemar test; all tests were two-sided, including potassium level > 5.5 mmol/L. A p value of < 0.05 was considered significant.

Results:

The average age of study participants (n=100) was 64.8 years and 70% were men, with an average blood pressure of about 139/78 mmHg and diabetes duration of about 16 years. Kidney disease was already significant at the start; the average eGFR was 45, and urine albumin was high (median UACR 800 mg/g), with 88% having severe albumin in urine (UACR ≥300 mg/g). Most patients also had other health problems, especially hypertension (90%), while some had heart disease (31%) and heart failure (13%). All patients were already on ACEi/ARB, most were on statins (74%), very few were on SGLT2 inhibitors (5%), and finerenone was started mostly at 10 mg (88%) (Table 1).

Table 1: Baseline characteristics of the cohort (n=100)

Characteristic	Overall (n=100)
Age, years	64.8 ± 8.0
Male sex	70 (70.0%)
Weight, kg	78.3 ± 14.6
Systolic BP, mmHg	138.8 ± 14.7
Diastolic BP, mmHg	78.0 ± 9.4
Diabetes duration, years	15.6 ± 7.3
eGFR, mL/min/1.73 m²	45.0 ± 10.7
UACR, mg/g (median [IQR])	800 [330-1643]
Serum potassium, mmol/L	4.35 ± 0.24
HbA1c, %	7.7 ± 1.1
eGFR category	
25-<45	55 (55.0%)
45-<60	33 (33.0%)
≥60	12 (12.0%)
UACR category	
30-<300	12 (12.0%)
≥300	88 (88.0%)
Comorbidities	
Hypertension (HTN)	90 (90.0%)
Ischemic heart disease (IHD)	31 (31.0%)
Heart failure (HF)	13 (13.0%)
Stroke	11 (11.0%)
Other	10 (10.0%)
Background therapy	
ACE inhibitor/ARB	100 (100.0%)
SGLT2 inhibitor	5 (5.0%)
Statin	74 (74.0%)
Finerenone starting dose	
10 mg	88 (88.0%)
20 mg	12 (12.0%)

Clinical and laboratory values of patients at baseline, 1 month, and 3 months after starting finerenone were compared as shown in table 2. Most participants returned for follow-up (95% at 1 month and 92% at 3 months), and overall compliance was satisfactory. Blood pressure improved at 1 month (SBP decreased by about 3 mmHg,

p<0.001), while the change at 3 months was smaller and not statistically significant for SBP (p=0.093); DBP remained lower at both visits (p≤0.002). Kidney monitoring showed a small but significant decline in eGFR at 1 and 3 months (both p<0.001), while urine albumin (UACR) decreased significantly at both follow-ups (both p<0.001), indicating improvement in albuminuria. Serum potassium increased slightly at 1 month and remained higher than baseline at 3 months (p<0.001 and p=0.004), but episodes of potassium >5.5 mmol/L were uncommon and not statistically different from baseline.

Summary of the main outcomes and safety events recorded over the 3-month follow-up period are shown in table 3. The primary composite kidney outcome occurred in 7% of patients, mainly driven by sustained ≥40% decline in eGFR (7%), while kidney failure requiring chronic dialysis was rare (1%) and no kidney transplants occurred. A small number of patients developed sustained eGFR <15 (2%). Regarding safety, hyperkalaemia requiring ER/hospital care occurred in 2%, with a peak potassium around 6.0 mmol/L in those cases. Overall, 9% had any ER visit/ hospitalization during follow-up, 3% experienced a cardio

Table No 2: Treatment + follow-up response at 1 month and 3 months.

Outcome Measure	Baseline	1 month	p (vs baseline)	3 months	p (vs baseline)
Visit attended	–	95 (95.0%)	–	92 (92.0%)	–
Adherence ≥80% (among attended)	–	84/95 (88.4%)	–	76/92 (82.6%)	–
Finerenone dose status					
10 mg	88 (88.0%)	36 (37.9%)	–	39 (42.4%)	–
20 mg	12 (12.0%)	54 (56.8%)	–	50 (54.3%)	–
Held	–	4 (4.2%)	–	1 (1.1%)	–
Stopped	–	1 (1.1%)	–	2 (2.2%)	–
Blood pressure and weight (among attended)					
SBP, mmHg	138.8 ± 14.7	135.0 ± 16.2	<0.001	137.5 ± 17.2	0.093
SBP from baseline, mmHg	0	−3.0 ± 8.0	<0.001	−1.7 ± 9.7	0.093
DBP, mmHg	78.0 ± 9.4	76.2 ± 10.7	0.002	75.3 ± 11.5	<0.001
Weight, kg	78.3 ± 14.6	77.9 ± 14.8	0.017	78.5 ± 14.3	0.725
Kidney and lab monitoring (among attended)					
eGFR, mL/min/1.73 m²	45.0 ± 10.7	43.9 ± 11.2	<0.001	41.9 ± 11.8	<0.001
eGFR from baseline, mL/min/1.73 m²	0	−1.5 ± 3.9	<0.001	−2.7 ± 5.9	<0.001
UACR, mg/g (median [IQR])	800 [330-1643]	607 [272-1277]	<0.001	540 [244-1142]	<0.001
UACR % change from baseline (median [IQR])	0	−22.3 [−29.8--9.8]	<0.001	−33.5 [−41.0--21.5]	<0.001
Serum potassium, mmol/L	4.35 ± 0.24	4.56 ± 0.47	<0.001	4.47 ± 0.43	0.004
Potassium from baseline, mmol/L	0	0.21 ± 0.41	<0.001	0.12 ± 0.39	0.004
Potassium >5.5 mmol/L	0 (0.0%)	5 (5.3%)	0.074	2 (2.2%)	0.480
Events and safety (among attended)					
ER visit / hospitalization	–	1 (1.1%)	–	8 (8.7%)	–
Any adverse event recorded	–	4 (4.2%)	–	9 (9.8%)	–

vascular event, and 1% died during the study period.

Table No 3: Three-month outcomes and safety (primary results) (n=100).

Outcome	Overall (n=100)
Primary composite kidney outcome*	7 (7.0%)
Sustained ≥40% fall in eGFR	7 (7.0%)
Kidney failure (chronic dialysis start)	1 (1.0%)
Kidney transplant	0 (0.0%)
Sustained eGFR <15 mL/min/1.73 m²	2 (2.0%)
Safety outcomes	
Hyperkalaemia needing ER/hospital care	2 (2.0%)
Peak K^+ (mmol/L) among hyperkalaemia ER cases	
Mean ± SD	6.04 ± 0.03
Median	6.04
Any ER visit/hospitalization recorded (1M or 3M)	9 (9.0%)
CV event (MI / stroke / HF admission)	3 (3.0%)
Death (any cause)	1 (1.0%)

Discussion:

The patients in this study had a high baseline kidney risk. Most patients had reduced eGFR and severe albuminuria at the time of finerenone initiation. This pattern is clinically plausible because CKD is common in high-risk populations in Lahore, where a recent cross-sectional study reported CKD in about one-fourth of screened high-risk adults.[8] At the national level, Pakistani literature also shows that diabetic nephropathy is one of the leading causes of CKD.[9] The high burden of type 2 diabetes and pre-diabetes in Pakistan further explains why many patients present with diabetic kidney disease and advanced albuminuria.[10] Therefore, the baseline profile of this cohort reflects a high-risk local population in which additional kidney-protective therapy is clinically relevant. The main finding of this study was a significant reduction in UACR after starting finerenone. Median UACR decreased from 800 mg/g at baseline to 540 mg/g at 3 months. This finding is biologically plausible because finerenone blocks mineralocorticoid receptor activity and reduces inflammatory and fibrotic signalling in the kidney. A real-world Chinese study also reported that finerenone reduced UACR with manageable safety in patients with diabetic kidney disease.[11] Similar findings were reported in a Japanese retrospective study, where finerenone significantly reduced UACR after 3 months, although potassium and eGFR did not change significantly in that cohort.[12] Another Japanese multi-cohort study from the FOUNTAIN platform also supported early real-world use of finerenone in CKD with type 2 diabetes.[13] These regional findings support our result and suggest that early albuminuria reduction is a consistent response to finerenone across Asian populations.

Most patients in the present study were started on 10 mg finerenone. This is consistent with real-world prescribing, especially in patients with reduced eGFR or higher perceived risk of hyperkalaemia. In the FINE-REAL interim analysis, finerenone was also commonly started in routine practice with low discontinuation and infrequent hyperkalaemia.[14] In contrast, a United States multi-database study showed higher use of SGLT2 inhibitors among finerenone initiators than in current study.[15] This difference may be due to cost, access, and local prescribing patterns. Thus, our dose pattern appears practical for a resource-limited setting, but background kidney-protective therapy needs further optimisation.

The early fall in albuminuria is an important treatment signal. In this study, the median UACR percentage reduction was 33.5% at 3 months. This is clinically meaningful because reduction in albuminuria is linked with lower future kidney risk. A mediation analysis of finerenone trials showed that early albuminuria reduction explained a substantial part of later kidney protection.[16] Therefore, the albuminuria response in this cohort supports the potential benefit of finerenone, although longer follow-up is needed to confirm whether this early response leads to fewer kidney events. There was a slight decrease in eGFR at follow-up. This is a significant discovery, but it has to be taken with a grain of salt. The drop in eGFR following initiation of kidney-protective therapy may not necessarily be due to direct kidney damage and can be attributed to haemodynamic change.[17] The relatively short study duration limits our ability to differentiate a reversible early dip from disease progression in the FIDELITY kidney analysis. Therefore, the eGFR loss in this study is evidence that monitoring is needed, but not to exclude long-term benefit.

The potassium level rose slightly after the start of finerenone treatment, but serious hyperkalaemia was rare. This is predicted, since mineralocorticoid receptor blockade may lead to less potassium elimination, particularly in patients with CKD. Within the FIDELIO-DKD safety analysis, the higher incidence of hyperkalaemia was balanced by the fact that monitoring of this parameter was done in a proper way, limiting serious effects of the higher levels of potassium.[18] In current study, the incidence of hyperkalaemia requiring emergency treatment was low, suggesting that finerenone is safe for use when potassium is monitored properly.[19]

There was a slight improvement from the drug treatment for blood pressure, particularly at 1 month. This is possible as there are mild mineralocorticoid receptor blocking effects of finerenone which might be responsible for the blood pressure lowering effects. In our study, the reduction in albuminuria was not the primary reason for the reduction in blood pressure, as demonstrated by a study that used ambulatory blood pressure monitoring.[20] But the primary effect seen was a reduction in albuminuria, which may have been facilitated by blood pressure improvement.

Only a small proportion of patients in this cohort were using SGLT2 inhibitors. This is important because current kidney care usually favours a layered approach using ACE inhibitors or ARBs, SGLT2 inhibitors, and finerenone when appropriate. A FIDELITY analysis showed that finerenone benefits were seen in patients with CKD and type 2 diabetes regardless of SGLT2 inhibitor use.[21] This supports the relevance of finerenone in our cohort, where SGLT2 inhibitor use was low. However, it also shows that future local practice should improve access to complete kidney-protective therapy.

The primary composite kidney outcome occurred in 7% of patients during 3 months. This was mainly driven by sustained eGFR decline, while chronic dialysis was rare and no kidney transplant occurred. This rate should be interpreted with caution because the follow-up period was short and the cohort had high baseline albuminuria. In a FIDELI-

TY subgroup analysis of stage 4 CKD, finerenone showed benefit in advanced CKD, but kidney outcomes required longer follow-up to assess fully.[22,23] Therefore, the composite kidney outcome in our study highlights the high-risk nature of the cohort rather than treatment failure.

This study shows that finerenone was associated with early and significant albuminuria reduction in patients with type 2 diabetes and CKD. The small eGFR decline was expected and needs follow-up rather than immediate discontinuation in stable patients. Potassium increased slightly, but serious hyperkalaemia remained uncommon with monitoring. These findings support the safe use of finerenone in routine nephrology care, especially in high-risk patients with albuminuria. The discussion therefore supports the conclusion that finerenone may provide early kidney benefit in local clinical practice, but longer observation is needed.

There are a few limitations with this study. Study design was single-centre observational study of small sample size. This was not a controlled trial, so the benefits of finerenone cannot be fully attributed. So the follow-up period was short at 3 months and was not long enough to evaluate kidney failure, cardiovascular outcomes or mortality. We could not rule out that UACR may differ with the different samples, and the repeated measurements over a longer time would provide greater evidence. The results may also not be representative of those of patients treated with full modern kidney-protective therapy, because use of SGLT2 inhibitors was low.

Larger, multicentre samples from various parts of Pakistan should be included in future studies. Follow up needs to be continued at least for 12-24 months to determine the duration of response in terms of albuminuria, eGFR slope, renal failure, cardiovascular outcomes and treatment discontinuation. Furthermore, further studies should compare patients who are treated with finerenone alone with patients who are treated with finerenone and SGLT2 inhibitors. This will assist in delineating the ideal multi-layered therapeutic strategy for Pakistani population of type 2 diabetes mellitus and CKD. In addition, the cost of adherence and potassium monitoring protocols should be explored in usual practices.

Conclusion:

Finerenone was associated with a significant short-term reduction in albuminuria over 3 months, with acceptable safety when potassium was monitored. A small early decline in eGFR was observed, while serious kidney events and severe hyperkalemia were uncommon during follow-up. These findings support finerenone use in routine care for T2DM-CKD patients, but longer follow-up is needed to confirm long-term kidney and cardiovascular benefits.

Disclaimer: None.

Conflict of Interest: None.

Source of Funding: None.

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Authors' contribution	
Sibgha Ahmed	Conceived Idea, Designed Methodology, Data Interpretation, Statistical Analysis and Manuscript Writing.
Mateen Akram	Study Supervision, Methodology Design and Manuscript Review
Shazia Abbas	Data Collection, Literature Review and Data Interpretation
Hina Francis	Data Collection and Literature Review
Sundus Shahzad	Data Collection and Statistical Analysis
Ruqayya Liaqat	Data Collection and Final Manuscript Approval.