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SAFETY AND EFFICACY OF FINERENONE IN CHRONIC KIDNEY DISEASE (CKD) PATIENTS

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ABSTRACT

Background: Finerenone is a selective non-steroidal mineralocorticoid receptor antagonist with established kidney and cardiovascular benefits in chronic kidney disease (CKD) associated with type 2 diabetes. Short-term real-world data from broader CKD populations, including non-diabetic CKD, remain less extensive. We evaluated renal responses and safety during routine nephrology care.

Methods: We conducted a prospective, observational, single-centre study in adults aged 18–75 years with CKD stages 1–4 and significant albuminuria/proteinuria. Twenty-eight eligible participants receiving or initiated on finerenone were followed at baseline and at 1, 3 and 6 months. Finerenone was used at 10–20 mg once daily with dose adjustment according to eGFR and serum potassium. Primary outcomes were longitudinal changes in eGFR and urine albumin-to-creatinine ratio (uACR); treatment-emergent adverse events were the principal safety outcome. Descriptive subgroup analyses compared diabetic and non-diabetic CKD.

Results: All 28 participants had recorded renal measurements at 1, 3 and 6 months. Mean eGFR increased from 45.5 ± 25.1 mL/min/1.73 m² at baseline to 54.8 ± 24.2 at 1 month, 55.9 ± 24.2 at 3 months and 55.4 ± 24.6 at 6 months; the mean 6-month change was 9.9 mL/min/1.73 m² (95% CI 5.7–14.2; $p < 0.001$). Median uACR decreased from 1040.0 mg/g (IQR 619.8–1485.8) to 313.5 mg/g (IQR 137.0–387.2), a 69.9% reduction in cohort medians. At 6 months, 25 participants (89.3%) had an increase in eGFR from baseline, and 19 (67.9%) had a patient-level uACR reduction of at least 30% in an exploratory analysis. Treatment-emergent adverse events were recorded in 18 participants (64.3%); hyperkalemia was recorded in 7 (25.0%), and 5 (17.9%) had maximum follow-up potassium ≥ 5.5 mmol/L. Hypotension occurred in 4 (14.3%) and renal deterioration/mild eGFR decline in 2 (7.1%). No cardiovascular composite event or hospitalization was documented.

Conclusions: In this small, uncontrolled observational cohort, finerenone exposure was associated with progressive reduction in albuminuria and an observed increase in mean eGFR over 6 months. Hyperkalemia and haemodynamic events remained clinically relevant and support structured monitoring. The findings are hypothesis-generating and cannot establish causality or long-term cardiorenal benefit.

Keywords: Finerenone; chronic kidney disease; albuminuria; uACR; eGFR; hyperkalemia; mineralocorticoid receptor antagonist; diabetic CKD; non-diabetic CKD

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INTRODUCTION

Chronic kidney disease is defined by abnormalities of kidney structure or function that persist for at least 3

months and has a major burden of kidney failure, cardiovascular disease and premature mortality. Risk is determined jointly by estimated glomerular filtration rate (eGFR) and albuminuria, making reduction of albuminuria an important therapeutic target in addition to preservation of filtration. Contemporary CKD care therefore combines renin–angiotensin system (RAS) blockade, sodium–glucose cotransporter-2 (SGLT2) inhibition when indicated, blood-pressure control and metabolic risk reduction [1]. Mineralocorticoid receptor (MR) activation contributes to renal inflammation, oxidative stress, endothelial dysfunction and fibrosis. These effects extend beyond sodium

retention and may persist despite RAS inhibition because of incomplete suppression or “escape” of aldosterone signalling [2,3]. Steroidal MR antagonists such as spironolactone and eplerenone can reduce albuminuria and cardiovascular events but are limited in CKD by hyperkalemia, rises in creatinine and, for spironolactone, endocrine adverse effects. Finerenone is a selective non-steroidal MR antagonist developed to preserve MR-mediated anti-inflammatory and anti-fibrotic effects with a different tissue distribution and fewer endocrine off-target effects [3–5]. The clinical evidence base for finerenone initially focused on CKD with type 2 diabetes. In FIDELIO-DKD, finerenone reduced the composite kidney outcome of kidney failure, sustained eGFR decline or renal death, while FIGARO-DKD demonstrated a reduction in a cardiovascular composite driven in part by fewer heart-failure hospitalizations [6,7]. The prespecified FIDELITY pooled analysis confirmed reductions in both cardiovascular and kidney outcomes across the combined programme [8]. Hyperkalemia remains the principal treatment-limiting safety concern and is best managed with baseline and serial potassium assessment and dose adjustment [9]. The evidence base has expanded further. CONFIDENCE showed that simultaneous initiation of finerenone and empagliflozin in CKD with type 2 diabetes produced greater albuminuria lowering than either agent alone at 180 days, supporting complementary mechanisms [10]. Importantly for non-diabetic CKD, the FIND-CKD randomized trial published in 2026 enrolled 1,584 adults without diabetes and showed a slower annual eGFR decline with finerenone than placebo over 32 months (difference 0.7 mL/min/1.73 m² per year; 95% CI 0.3–1.1; $p < 0.001$), with hyperkalemia the most common adverse event [11]. These developments make characterization of short-term renal and safety responses in local routine practice clinically relevant, while also emphasizing the distinction between observational treatment-associated changes and randomized evidence of long-term benefit. Against this background, we evaluated short-term renal outcomes and safety among adults with proteinuric CKD treated with finerenone at a tertiary nephrology centre, with additional descriptive assessment of diabetic versus non-diabetic CKD and cardiovascular outcomes.

STUDY OBJECTIVES

The primary objectives were to evaluate changes in eGFR and uACR at 1, 3 and 6 months and to characterize the incidence and severity of treatment-emergent adverse events, particularly hyperkalemia. Secondary objectives were to describe cardiovascular events and hospitalizations and to compare renal responses between diabetic and non-diabetic CKD.

METHODS

Study design and setting

This was a prospective, observational, single-centre cohort study conducted in the Department of Nephrology, Geetanjali Medical College and Hospital, Udaipur, Rajasthan, India. Recruitment occurred over 6 months, with follow-up scheduled at 1, 3 and 6 months after finerenone exposure. All eligible CKD patients presenting during the recruitment period were considered; the final analysed cohort comprised 28 participants.

Participants

Adults aged 18–75 years with CKD stages 1–4 were eligible if they had significant albuminuria/proteinuria, defined as uACR >300 mg/g, urine protein-to-creatinine ratio (uPCR) >500 mg/g, or urine routine microscopy showing protein ≥2+ when quantitative measurements were unavailable. Participants were required to be receiving stable standard CKD care, including an ACE inhibitor/ARB and/or SGLT2 inhibitor unless there was a documented clinical reason for non-use. Both diabetic and non-diabetic CKD were included. Participants already taking finerenone and newly initiated participants were eligible.

Key exclusion criteria were significant liver disease, hyperkalemia at screening, severe or recent cardiovascular disease, conditions affecting gastrointestinal drug absorption, pregnancy or lactation, concomitant potassium-sparing diuretics or interacting drugs, known finerenone intolerance/contraindication, symptomatic low blood pressure in the absence of antihypertensive therapy, unwillingness to discontinue another MRA, and prior kidney transplantation.

Exposure and follow-up

Finerenone was administered at 10–20 mg once daily, with the dose selected and adjusted according to eGFR and serum potassium. At enrolment, demographic characteristics, body mass index, comorbidities and concomitant medications were recorded. Baseline laboratory assessment included serum creatinine, eGFR, potassium, glycated haemoglobin where relevant, uACR, uPCR and urine routine microscopy. At each follow-up visit, renal function, uACR, urine protein category, serum potassium and treatment-emergent adverse events were reassessed. Adverse events were documented with available information on severity and management.

Outcomes

Primary efficacy outcomes were changes from baseline in eGFR and uACR at 1, 3 and 6 months. Semiquantitative urine protein categories were treated as supportive proteinuria measures. The principal safety outcome was the occurrence of treatment-emergent adverse events, with particular attention to hyperkalemia and events associated with dose adjustment or interruption. Secondary outcomes were a cardiovascular composite defined in the protocol as myocardial infarction, stroke or hospitalization for

heart failure, overall hospitalization, and descriptive renal responses according to diabetes status.

Statistical Analysis

Continuous variables are presented as mean $\pm$ standard deviation when approximately symmetric and as median (interquartile range [IQR]) when skewed. Categorical variables are presented as counts and percentages. Between-group baseline comparisons used an unpaired t-test for normally distributed continuous variables and the Mann–Whitney test when normality was not met; categorical comparisons used chi-square testing with continuity correction or Fisher exact testing for 2x2 tables, as appropriate. For longitudinal renal outcomes, repeated observations were assessed using the Friedman test for global change across visits and paired comparisons of each follow-up visit with baseline. Because uACR was markedly right-skewed, non-parametric longitudinal interpretation was emphasized. Two-sided p values <0.05 were considered statistically significant. Statistical analyses were performed in IBM SPSS Statistics version 26.0; Microsoft Excel was used for graphical representation.

Exploratory Analyses

Additional descriptive analyses were derived from the patient-level Master Chart after the primary analysis. These included CKD etiologic distribution; the proportion of participants with an increase or decrease in eGFR at 6 months; the proportion achieving patient-level uACR reduction thresholds of at least 30%, 50%, 75% and 90%; maximum follow-up potassium categories; and descriptive safety patterns by baseline CKD category. These analyses were prespecified for manuscript expansion as exploratory and were not intended to provide confirmatory subgroup or predictive inference.

Ethics and Consent

The study protocol was reviewed and approved by the Institutional Ethics Committee of Geetanjali Medical College and Hospital. Written informed consent was obtained from all participants. Study data were anonymized and securely stored with access restricted to study personnel.

RESULTS

Participant disposition and baseline characteristics

Twenty-eight participants were analysed. All 28 had baseline data and recorded renal follow-up measurements at 1, 3 and 6 months, corresponding to 100% completion of scheduled renal assessments. No cardiovascular composite event or hospitalization was documented during the six-month observation period.

Table 01: Baseline demographic, renal and clinical characteristics

Characteristic	Overall cohort (N=28)
Age, years	59.5 $\pm$ 12.1; median 60.5 (IQR 53.0–69.2)
Male sex	16 (57.1%)

Female sex	12 (42.9%)
BMI, kg/m ²	25.1 $\pm$ 6.5
eGFR, mL/min/1.73 m ²	45.5 $\pm$ 25.1
uACR, mg/g	1040.0 (619.8–1485.8)
Serum creatinine, mg/dL	2.0 $\pm$ 1.0
Serum potassium, mmol/L	4.3 $\pm$ 0.3
SBP/DBP, mmHg	133.1 $\pm$ 11.1 / 80.6 $\pm$ 6.8
Diabetes	23 (82.1%)
Non-diabetes	5 (17.9%)
G1A3	6 (21.4%)
G2A3	8 (28.6%)
G3aA3	5 (17.9%)
G3bA3	3 (10.7%)
G4A3	6 (21.4%)

The cohort therefore represented a broad proteinuric CKD spectrum, with 50.0% carrying G1/G2 labels and 50.0% carrying G3/G4 labels. CKD etiology was heterogeneous: chronic glomerulopathy 8 (28.6%), diabetic CKD 7 (25.0%), hypertensive nephrosclerosis 6 (21.4%), CKD of uncertain etiology 6 (21.4%), and type 1 diabetes-associated disease 1 (3.6%). Hypertension was documented in 22 participants (78.6%), dyslipidemia in 22 (78.6%), and previous CVD history in 14 (50.0%).

Background treatment was extensive: an ACE inhibitor/ARB was documented in 22 participants (78.6%), an SGLT2 inhibitor in 24 (85.7%), and statin therapy in 22 (78.6%). These concomitant treatments are important because the study evaluates finerenone in routine care rather than as isolated monotherapy.

Renal efficacy: estimated glomerular filtration rate

Mean eGFR increased from 45.5 $\pm$ 25.1 mL/min/1.73 m² at baseline to 54.8 $\pm$ 24.2 at 1 month, 55.9 $\pm$ 24.2 at 3 months and 55.4 $\pm$ 24.6 at 6 months. The mean paired changes from baseline were 9.3 mL/min/1.73 m² at 1 month (95% CI 5.5–13.0), 10.3 at 3 months (95% CI 6.8–13.9) and 9.9 at 6 months (95% CI 5.7–14.2), with $p < 0.001$ for each comparison. The global Friedman test was statistically significant (34.72; $p < 0.001$). The trajectory showed an early increase followed by relative stability between months 1 and 6.

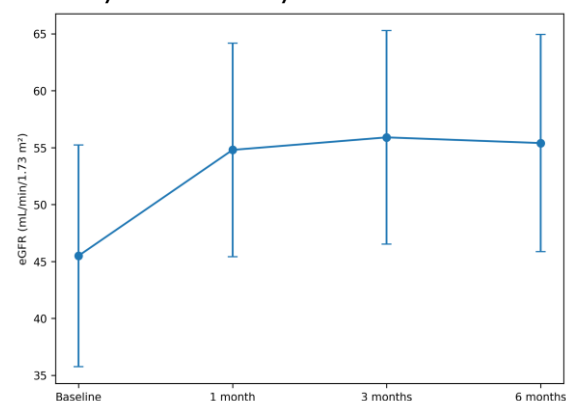


Figure 02: Mean eGFR trajectory over 6 months. Error bars represent 95% confidence intervals.

Table 02: Longitudinal renal outcomes

Outcome	Baseline	1 month	3 months	6 months
eGFR, mean $\pm$ SD (mL/min/1.73 m ²)	45.5 $\pm$ 25.1	54.8 $\pm$ 24.2	55.9 $\pm$ 24.2	55.4 $\pm$ 24.6
Change from baseline, mean (95% CI)	—	9.3 (5.5–13.0)	10.3 (6.8–13.9)	9.9 (5.7–14.2)
uACR, median (IQR) mg/g	1040.0 (619.8–1485.8)	856.5 (528.2–1189.2)	679.5 (412.5–965.8)	313.5 (137.0–387.2)
Mean paired uACR change, mg/g	—	–199.2	–437.8	–771.5

An exploratory patient-level analysis showed that 25 of 28 participants (89.3%) had a higher eGFR at 6 months than at baseline, whereas 3 (10.7%) had a lower value. This analysis describes direction of change only and does not distinguish treatment effect from biological variability or background care.

Renal Efficacy: Albuminuria and semiquantitative proteinuria

Median uACR decreased progressively from 1040.0 mg/g (IQR 619.8–1485.8) at baseline to 856.5 mg/g (IQR 528.2–1189.2) at 1 month, 679.5 mg/g (IQR 412.5–965.8) at 3 months and 313.5 mg/g (IQR 137.0–387.2) at 6 months. Relative to the baseline cohort median, this corresponds to approximate decreases of 17.6%, 34.7% and 69.9%, respectively. These percentages represent changes in cohort medians rather than the median of individual percentage responses. The Friedman test across visits was 78.56 ($p < 0.001$), and all paired comparisons with baseline were statistically significant ($p < 0.001$).

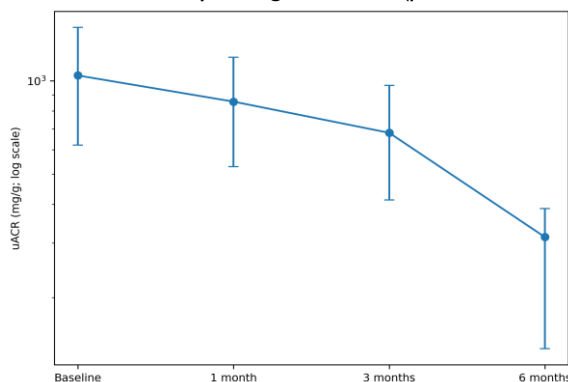


Figure 03: Median uACR trajectory over 6 months. Error bars represent the interquartile range; the y-axis is logarithmic.

Semiquantitative urine protein findings were concordant with the uACR trajectory. At baseline, all participants had either 2+ (15; 53.6%) or 3+ (13; 46.4%) protein on urine routine microscopy. At 6 months, 17 (60.7%) had 1+, 8 (28.6%) trace, 2 (7.1%) 2+ and 1 (3.6%) 3+. Thus, the proportion with 2+ or 3+ protein decreased from 100.0% at baseline to 10.7% at 6 months.

In a patient-level exploratory analysis, 19 of 28 participants (67.9%) had at least a 30% reduction in uACR by 6 months. Only one participant met each of the $\geq 50\%$, $\geq 75\%$ and $\geq 90\%$ reduction thresholds. These threshold analyses are descriptive and should not be interpreted as remission or as a validated responder analysis.

Safety Outcomes

At least one treatment-emergent adverse event was recorded in 18 of 28 participants (64.3%). Hyperkalemia was recorded as an adverse-event term in 7 participants (25.0%), and 5 (17.9%) had a maximum follow-up potassium ≥ 5.5 mmol/L. Hypotension occurred in 4 participants (14.3%), and renal deterioration/mild eGFR decline in 2 (7.1%). No gynecomastia was recorded.

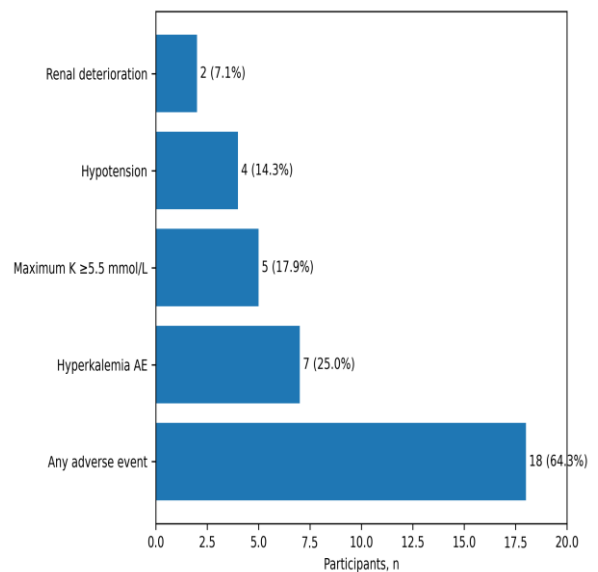


Figure 04: Participant-based safety outcomes during follow-up. Counts represent participants rather than repeated event terms.

Table 03: Safety outcomes during follow-up

Safety outcome	n	%
Any adverse event	18	64.3
Hyperkalemia recorded as AE	7	25.0
Maximum potassium ≥ 5.5 mmol/L	5	17.9
Hypotension	4	14.3
Renal deterioration / mild eGFR decline	2	7.1
Gynecomastia	0	0.0

The Master Chart also contained 12 hyperkalemia event terms and 6 hyponatremia event terms across follow-up. These are event-level observations and may include repeated episodes; they were therefore not interpreted as 12 and 6 distinct affected participants. No cardiovascular composite event or hospitalization was recorded during the observation period.

Maximum follow-up potassium was <5.0 mmol/L in 22 participants (78.6%), 5.0 to <5.5 mmol/L in 1 (3.6%), and ≥ 5.5 mmol/L in 5 (17.9%). Exploratory baseline-stage tabulation showed documented hyperkalemia in 0/6 G1A3, 0/8 G2A3, 1/5 G3aA3, 0/3 G3bA3 and 4/6 G4A3 participants; these very small cells preclude inferential comparison.

Dose patterns and exploratory subgroup analyses

At 1 and 3 months, 14 participants (50.0%) received 10 mg daily and 14 (50.0%) received 20 mg. At 6 months, dose documentation was available for 27 participants: 13 (48.1%) received 10 mg and 14 (51.9%) received 20 mg. The differing denominator at 6 months was not interpreted as treatment discontinuation because the remaining participant continued to have renal follow-up data without a recorded dose in the dose summary.

Table 04: Diabetes-stratified renal trajectory and selected exploratory findings

Parameter	Diabetic (n=23)	Non-diabetic (n=5)
Baseline eGFR, mean $\pm$ SD	46.1 $\pm$ 25.6	42.9 $\pm$ 25.0
1-month eGFR	55.5 $\pm$ 25.0	51.2 $\pm$ 22.1
3-month eGFR	56.7 $\pm$ 25.0	51.9 $\pm$ 22.6
6-month eGFR	56.3 $\pm$ 25.2	51.4 $\pm$ 23.7
Baseline uACR, median (IQR), mg/g	955.0 (590.5–1494.5)	1201.0 (1011.0–1256.0)
Baseline creatinine, mean $\pm$ SD, mg/dL	2.0 $\pm$ 1.1	2.0 $\pm$ 0.9
Mean patient-level uACR reduction at 6 months, exploratory	38.6%	28.4%

Mean eGFR increased in both diabetes subgroups. The diabetic group changed from 46.1 ± 25.6 to 56.3 ± 25.2 mL/min/1.73 m², whereas the non-diabetic group changed from 42.9 ± 25.0 to 51.4 ± 23.7 . The difference between groups was not tested as a treatment interaction, and the non-diabetic sample of five participants was too small for reliable comparative efficacy or safety inference.

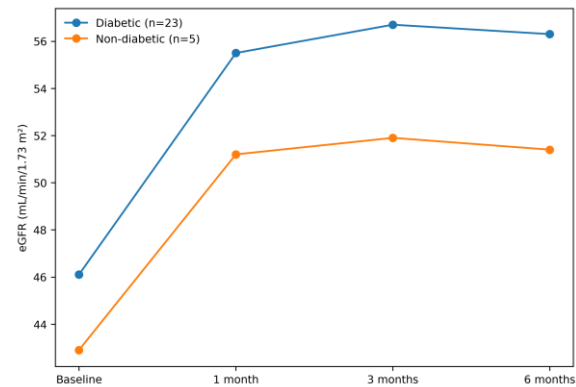


Figure 06: Mean eGFR trajectory stratified by diabetes status. The figure is descriptive; no treatment-by-diabetes interaction was estimated.

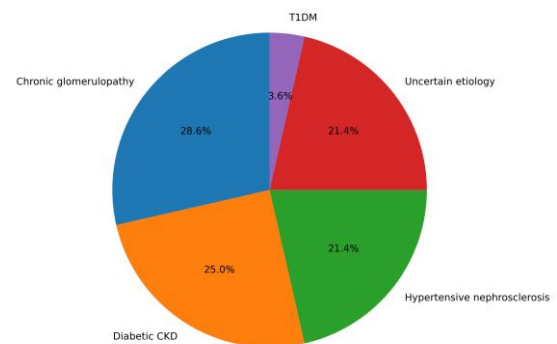


Figure 05: Baseline CKD etiologic spectrum.

DISCUSSION

Principal Findings

This prospective single-centre cohort provides a detailed description of renal and safety observations during finerenone use in routine nephrology practice. The most prominent findings were a progressive reduction in albuminuria, an early increase and subsequent maintenance of mean eGFR above baseline, improvement in semiquantitative urine protein categories, and a clinically relevant frequency of hyperkalemia and haemodynamic adverse events. Because the study had no comparator group and included only 28 participants, these findings should be interpreted as treatment-associated observations rather than proof of causal efficacy.

The baseline population was heterogeneous. Half of the participants had G1/G2 stage labels and half had G3/G4 labels, but all had marked albuminuria/proteinuria. This combination created a cohort spanning relatively preserved filtration to advanced predialysis CKD. The median uACR of 1040 mg/g was substantially elevated, suggesting a population in whom reduction of albuminuria would be clinically meaningful. Background therapy was also intensive, with 85.7% receiving SGLT2 inhibition and 78.6% receiving an ACE inhibitor or ARB. Consequently, any renal change observed during follow-up reflects a treatment context in which finerenone was added to

other kidney-protective strategies rather than substituted for them.

The current findings are consistent with the biological rationale for MR blockade. Preclinical work supports effects on oxidative stress, macrophage recruitment and profibrotic pathways, while clinical trials have established reductions in albuminuria and hard cardiorenal outcomes in selected populations [3–8]. The practical implication is that finerenone should be considered a component of multifactorial CKD risk reduction, with the magnitude of albuminuria response interpreted alongside background RAS and SGLT2 therapy.

Interpretation of eGFR change

Mean eGFR increased by approximately 9.9 mL/min/1.73 m² at 6 months, with the largest change during the first month. The persistence of the higher mean through months 3 and 6 is notable, but an observational rise in creatinine-based eGFR should not be equated with recovery of nephron mass or reversal of structural fibrosis. Hydration status, intercurrent illness, changes in glycemic control, creatinine generation, concomitant treatments and biological variation can influence eGFR. In the absence of an untreated comparator, the contribution of finerenone cannot be isolated.

The long-term randomized evidence addresses a different question. FIDELIO-DKD demonstrated a reduction in a kidney failure/sustained eGFR decline composite over a median 2.6 years rather than an increase in short-term eGFR [6]. The present study therefore adds information about the short-term trajectory observed in practice but cannot substitute for outcome evidence. The 2026 FIND-CKD trial provides particularly relevant context for the five non-diabetic participants: among 1,584 adults without diabetes, finerenone slowed annual eGFR decline by 0.7 mL/min/1.73 m² compared with placebo over 32 months [11]. This supports a kidney-protective effect in a randomized population but remains conceptually distinct from the short-term eGFR increase observed in our uncontrolled cohort.

Interpretation of Albuminuria Response

The reduction in median uACR from 1040.0 to 313.5 mg/g was progressive and statistically robust. The approximately 69.9% reduction in cohort medians is numerically large, but it should not be confused with a median individual percentage reduction. The patient-level exploratory analysis showed that 67.9% achieved at least a 30% uACR reduction, providing a complementary responder perspective. Even after improvement, the median remained above 300 mg/g, indicating persistent albuminuria and residual kidney risk.

These findings are directionally consistent with other finerenone datasets. In the 2025 CONFIDENCE trial, simultaneous initiation of finerenone and empagliflozin reduced UACR more than either monotherapy at day 180, supporting additive albuminuria lowering when

the two mechanisms are combined [10]. An observational multicentre IgA nephropathy cohort also reported lower proteinuria with finerenone-containing treatment [12]. Direct numerical comparison with the present cohort is inappropriate because populations, background therapies, endpoints and analytical approaches differ.

Safety and Monitoring

Hyperkalemia was the principal safety concern. Seven participants had hyperkalemia recorded as an adverse-event term and five had a maximum potassium ≥ 5.5 mmol/L. The distinction between participant-level incidence and repeated event terms is important: 12 hyperkalemia event entries in the Master Chart do not represent 12 unique affected participants. Prior FIDELIO-DKD safety analyses similarly identify lower eGFR and higher baseline potassium as important risk factors, supporting the use of structured potassium monitoring and protocolized dose management [9]. Hypotension and renal deterioration were also observed in a minority of participants. These events reinforce that mean renal improvement can coexist with clinically important individual-level deterioration. No gynecomastia was recorded, which is compatible with the non-steroidal receptor selectivity of finerenone, although a sample of 28 participants cannot exclude uncommon endocrine events [3–5].

Diabetic versus non-diabetic CKD

Both diabetic and non-diabetic participants showed an early increase in mean eGFR. However, only five participants were classified as non-diabetic, limiting precision and making comparative interpretation especially sensitive to single-patient changes. The current findings should therefore be presented as descriptive subgroup data rather than evidence that finerenone works similarly or differently according to diabetes status. The emergence of FIND-CKD as a large randomized trial in non-diabetic CKD is important because it provides direct evidence in a population that was previously under-represented in outcomes trials [11].

STRENGTHS AND LIMITATIONS

Strengths include prospective follow-up, complete recorded renal follow-up at all scheduled visits, systematic serial uACR/eGFR assessment, and inclusion of both diabetic and non-diabetic CKD. The study also captures routine-care background therapy, which improves clinical realism.

Limitations are substantial. The cohort was small and single-centre, the non-diabetic subgroup comprised only five participants, follow-up was limited to six months, and there was no concurrent control group. Background therapies and intercurrent changes were not randomised or fully quantified, preventing attribution of observed renal changes specifically to finerenone. The cardiovascular composite had zero observed events, so the study had no meaningful power to evaluate clinical cardiovascular protection.

Exploratory responder and stage-specific analyses were not prespecified for confirmatory inference.

CONCLUSION

In this 28-participant prospective observational cohort of proteinuric CKD, finerenone use was accompanied by a progressive reduction in albuminuria, improvement in semiquantitative urine protein categories and an observed increase in mean eGFR over six months. Most participants had higher eGFR at six months than at baseline, and two-thirds had at least a 30% patient-level reduction in uACR in an exploratory analysis. Hyperkalemia remained the most important safety signal, and hypotension and renal deterioration were also observed. No cardiovascular events or hospitalizations were documented during the short follow-up.

These findings support continued evaluation of finerenone as part of comprehensive CKD care but should not be interpreted as evidence of causal short-term eGFR recovery or long-term outcome benefit. The appropriate clinical message is one of potential antiproteinuric benefit together with the need for careful potassium, renal-function and blood-pressure monitoring. Larger controlled studies and longer follow-up are required to define durability and clinical outcome effects.

ETHICS APPROVAL AND CONSENT

The study protocol was reviewed and approved by the Institutional Ethics Committee of Geetanjali Medical College and Hospital. Written informed consent was obtained from all participants.

FUNDING

None

CONFLICT OF INTEREST

The authors declare no conflict of interest.

AUTHOR CONTRIBUTIONS

Dr G K Mukhiya provided overall academic supervision and critical guidance throughout the study and manuscript preparation. Dr Nilanjan Mukherjee was involved in patient evaluation, data collection, analysis, and manuscript preparation. Dr Nilanjan Mukherjee is the corresponding author.

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