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Twelve-Month Albuminuria Response to Finerenone in Patients With Chronic Kidney Disease and Type 2 Diabetes Mellitus: A Real-World Study

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Abstract

Introduction

Finerenone, a selective non-steroidal mineralocorticoid receptor antagonist, reduces albuminuria and cardiorenal risk in patients with chronic kidney disease (CKD) and type 2 diabetes mellitus (T2DM). However, real-world data on its longer-term effects across different levels of baseline albuminuria remain limited. This study evaluated longitudinal changes in urinary albumin concentration, renal function, and serum potassium during 12 months of finerenone treatment in routine nephrology practice.

Materials and methods

This retrospective, observational, single-center study included 68 patients with CKD and T2DM receiving finerenone in addition to optimized renin-angiotensin system blockade. Patients receiving sodium-glucose cotransporter-2 inhibitors, glucagon-like peptide-1 receptor agonists, other mineralocorticoid receptor antagonists, or calcium dobesilate were excluded. Urinary albumin concentration was measured in spot urine samples and expressed in mg/L. Patients were stratified according to baseline albuminuria into Group 1 (30-300 mg/L; n=28), Group 2 (>300 to ≤1000 mg/L; n=33), and Group 3 (>1000 mg/L; n=7). Urinary albumin concentration, estimated glomerular filtration rate (eGFR), serum albumin, and serum potassium were evaluated at baseline and at three, six, and 12 months.

Results

Mean urinary albumin concentration decreased progressively from 383.4±328.7 mg/L at baseline to 230.0±240.1 mg/L at three months, 115.6±120.1 mg/L at six months, and 48.5±50.1 mg/L at 12 months (p<0.001), corresponding to a mean 12-month relative reduction of 86.8±6.1%. The relative reduction did not differ significantly among baseline albuminuria groups (85.2%, 88.4%, and 85.8%, respectively; p=0.258). Mean eGFR remained stable at three months and was higher than baseline at six and 12 months, reaching 65.3±17.7 mL/min/1.73 m² at month 12 (adjusted p<0.001). Serum potassium increased significantly during follow-up (p<0.001). Sixteen patients (23.5%) developed at least one potassium concentration >5.5 mmol/L; all such episodes occurred among patients receiving finerenone 20 mg/day and were managed by temporary treatment interruption for two weeks, followed by reinitiation at 10 mg/day after normalization of serum potassium.

Discussion

The observed albuminuria reduction was substantially greater than that reported in randomized finerenone trials. However, urinary albumin concentration in spot urine samples is influenced by urine dilution and is not directly comparable with urinary albumin-to-creatinine ratio measurements. The absence of a control group also limits causal interpretation of the observed albuminuria and eGFR changes.

Conclusions

In this real-world cohort of patients with CKD and T2DM, finerenone treatment was associated with a progressive reduction in urinary albumin concentration over 12 months, with comparable proportional reductions across baseline albuminuria groups. Serum potassium elevations required temporary treatment interruption followed by dose reduction in a subset of patients. Larger prospective controlled studies using standardized albuminuria measurements are needed to confirm these findings.

Categories: Internal Medicine, Nephrology

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Introduction

Chronic kidney disease (CKD) is a major complication of type 2 diabetes mellitus (T2DM) and is associated with progressive loss of kidney function, increased cardiovascular morbidity and mortality, and a substantial healthcare burden. Patients with diabetes and CKD remain at high residual risk of kidney failure, atherosclerotic cardiovascular disease, heart failure, and premature mortality despite advances in glycemic and blood pressure management [1,2]. Albuminuria is a particularly important marker in this population, reflecting kidney damage and potentially systemic endothelial injury, and identifying patients at increased risk of adverse kidney and cardiovascular outcomes. Moreover, treatment-induced reductions in albuminuria have been associated with a lower subsequent risk of CKD progression [3].

Blockade of the renin-angiotensin system (RAS) with an angiotensin-converting enzyme inhibitor (ACEi) or an angiotensin receptor blocker (ARB) has long represented a cornerstone of treatment for patients with diabetic CKD and albuminuria. However, substantial residual cardiorenal risk persists despite optimized RAS blockade, highlighting the need for additional therapeutic strategies targeting complementary pathways involved in the progression of diabetic kidney disease [1,2].

Mineralocorticoid receptor overactivation has emerged as an important contributor to the pathophysiology of CKD associated with T2DM. Excessive mineralocorticoid receptor signaling promotes inflammation, oxidative stress, vascular dysfunction, and fibrosis within the kidney and cardiovascular system [4,5]. Although steroidal mineralocorticoid receptor antagonists can reduce albuminuria, their broader use in patients with CKD has been limited by the risk of hyperkalemia, particularly in patients with impaired kidney function and concomitant RAS blockade [4,5].

Finerenone is a selective, non-steroidal mineralocorticoid receptor antagonist with pharmacological and tissue-distribution characteristics that distinguish it from traditional steroidal mineralocorticoid receptor antagonists [5]. The pivotal Finerenone in Reducing Kidney Failure and Disease Progression in Diabetic Kidney Disease (FIDELIO-DKD) trial demonstrated that, when added to optimized RAS blockade, finerenone reduced the risk of CKD progression and cardiovascular events in patients with CKD and T2DM [6]. The complementary Finerenone in Reducing Cardiovascular Mortality and Morbidity in Diabetic Kidney Disease (FIGARO-DKD) trial demonstrated a significant reduction in cardiovascular events across a broader spectrum of CKD severity [7]. Subsequently, the prespecified pooled analysis, Finerenone in chronic kidney disease and Type 2 diabetes: Combined FIDELIO-DKD and FIGARO-DKD Trial Programme Analysis (FIDELITY), involving more than 13,000 participants, confirmed significant cardiovascular and kidney benefits of finerenone across a broad range of CKD severity [8].

Reduction in albuminuria represents an early and clinically relevant pharmacodynamic response to finerenone. In FIDELIO-DKD, finerenone produced an approximately 31% greater reduction in urinary albumin-to-creatinine ratio (UACR) than placebo at month 4 [6]. Subsequent mediation analyses of the FIDELIO-DKD and FIGARO-DKD programs demonstrated that early reduction in albuminuria accounted for a substantial proportion of the observed long-term kidney benefit of finerenone [9]. These findings further support albuminuria as an important intermediate marker of therapeutic response.

Although randomized controlled trials provide robust evidence regarding the efficacy and safety of finerenone, real-world evidence remains comparatively limited. Patients encountered in routine nephrology practice may differ from clinical trial populations with respect to comorbidities, baseline kidney function, albuminuria severity, concomitant therapies, adherence, and laboratory monitoring. Recent observational studies have confirmed reductions in albuminuria with finerenone in routine clinical practice, but available cohorts remain relatively small, and follow-up has generally been shorter than 12 months [10,11].

Another clinically relevant question is whether the proportional albuminuria response to finerenone differs according to baseline albuminuria severity. Patients with greater albuminuria are at particularly high risk of CKD progression [1,3], making characterization of treatment response across different levels of baseline albuminuria clinically relevant. Assessment of renal function and serum potassium alongside albuminuria is also essential, because preservation of kidney function represents a major therapeutic objective, whereas hyperkalemia remains the principal treatment-related safety concern associated with mineralocorticoid receptor blockade [12].

Therefore, the primary aim of the present study was to evaluate the 12-month change in urinary albumin concentration during finerenone treatment in patients with CKD and T2DM receiving background RAS blockade in a real-world outpatient nephrology setting. A further objective was to determine whether the magnitude of the relative albuminuria reduction differed according to baseline albuminuria severity. Secondary objectives were to evaluate longitudinal changes in estimated glomerular filtration rate (eGFR), serum albumin, and serum potassium, and to assess hyperkalemia requiring dose modification.

Materials And Methods

Study design and setting

This retrospective, observational, single-center study evaluated the 12-month real-world clinical and laboratory outcomes of finerenone treatment in patients with CKD and T2DM. The study included consecutive eligible patients followed in an outpatient nephrology practice at Medical Center “St. Anna”, Ltd., Varna, Bulgaria, between 1 August 2025 and 1 August 2026. During this period, the medical records and routine laboratory tests of 68 patients who met the study criteria were reviewed.

The study was designed to evaluate changes in urinary albumin concentration during finerenone therapy, with particular emphasis on whether the magnitude of the albuminuria response differed according to the severity of baseline albuminuria. Changes in eGFR, serum albumin, and serum potassium were assessed as secondary renal, biochemical, and safety outcomes.

The study was conducted in accordance with the principles of the Declaration of Helsinki. The study was approved by the Commission on Ethics of Research at Medical Center “St. Anna”, Ltd., Varna, Bulgaria (Protocol No. 2/17.09.2026). Due to the retrospective nature of the study and the use of anonymized clinical data, the requirement for individual informed consent was waived by the ethics committee.

Study population and patient selection

The source population consisted of adult patients with T2DM and CKD who were routinely followed by a nephrologist. Medical records of 109 patients with documented CKD and T2DM were retrospectively reviewed for eligibility. Patient selection was performed according to predefined inclusion and exclusion criteria.

The inclusion criteria were age ≥ 18 years; a diagnosis of T2DM and CKD with persistent albuminuria; baseline eGFR ≥ 25 mL/min/1.73 m²; baseline serum potassium ≤ 4.8 mmol/L; stable ACEi or ARB therapy at an optimized or maximum tolerated dose; initiation of finerenone at an eGFR-adjusted dose; and availability of baseline and 12-month follow-up clinical and laboratory data. The exclusion criteria were age < 18 years; type 1 diabetes mellitus; spironolactone or another mineralocorticoid receptor antagonist; baseline serum potassium > 4.8 mmol/L; concomitant sodium-glucose cotransporter-2 (SGLT2) inhibitor treatment; concomitant glucagon-like peptide-1 (GLP-1) receptor agonist treatment; calcium dobesilate treatment; nephrolithiasis; recurrent urinary tract infections; and chronic glomerulonephritis or another renal disease potentially contributing to albuminuria.

Of the 109 patient records screened, 36 patients were excluded based on the predefined baseline eligibility criteria: 10 because of baseline serum potassium > 4.8 mmol/L, six because of concomitant spironolactone therapy, six because of GLP-1 receptor agonist treatment, and 14 because of SGLT2 inhibitor treatment.

The remaining 73 patients initiated finerenone treatment and underwent serum potassium reassessment approximately four weeks after treatment initiation. At the four-week potassium assessment, five patients (6.8%) had serum potassium concentrations > 5.5 mmol/L and were excluded from the final longitudinal cohort. The remaining 68 patients had normal serum potassium concentrations at this assessment and were included in the 12-month analysis.

Patients receiving SGLT2 inhibitors were excluded to minimize the potential influence of their established albuminuria-lowering and renoprotective effects on the observed changes during finerenone treatment. Similarly, patients receiving GLP-1 receptor agonists or calcium dobesilate were excluded because these therapies could potentially influence albuminuria and/or renal outcomes and thereby confound the assessment of changes observed during finerenone treatment.

Complete medical documentation, including available laboratory measurements at baseline and at months 3, 6, and 12, was required for study inclusion. All 68 patients included in the final cohort had complete data for urinary albumin concentration, eGFR, serum albumin, and serum potassium at all four assessment points.

Finerenone treatment and background therapy

Finerenone was administered in addition to pre-existing RAS blockade with either an ACEi or an ARB at the optimized or maximum tolerated dose. The initial dose of finerenone was selected according to baseline renal function. In accordance with the recommended dosing regimen, patients with an eGFR ≥ 60 mL/min/1.73 m² initiated finerenone at a dose of 20 mg once daily, whereas patients with an eGFR ≥ 25 to < 60 mL/min/1.73 m² initiated treatment at a dose of 10 mg once daily.

Serum potassium was monitored throughout the 12-month follow-up period as the principal treatment-related safety parameter. Treatment modifications related to changes in serum potassium concentration

were recorded from the patients' medical records. Serum potassium was reassessed approximately four weeks after finerenone initiation. Only patients with serum potassium concentrations within the normal laboratory reference range at this assessment who continued finerenone treatment were included in the final study cohort. Subsequent serum potassium measurements were evaluated at months 3, 6, and 12 as part of the longitudinal safety analysis.

All patients continued background RAS blockade during follow-up. Patients receiving concomitant SGLT2 inhibitors, GLP-1 receptor agonists, spironolactone or other mineralocorticoid receptor antagonists, or calcium dobesilate were excluded from the study to minimize the potential influence of concomitant therapies on albuminuria and renal outcomes.

Data collection and clinical variables

Clinical and laboratory information was obtained retrospectively from the patients' medical records. Baseline was defined as the assessment performed at or immediately before initiation of finerenone treatment.

Baseline demographic and clinical variables included age, sex, current smoking status, duration of T2DM, glycated hemoglobin (HbA1c), systolic blood pressure (SBP), use of an ACE inhibitor or ARB, eGFR, urinary albumin concentration, serum albumin, and serum potassium.

Sex was analyzed as a binary variable. Smoking status represented current smoking at baseline. Duration of diabetes was recorded in years. Blood pressure status was categorized according to the presence or absence of SBP > 140 mmHg.

Renal function was assessed using eGFR, expressed as mL/min/1.73 m². Serum creatinine was not included as a separate study outcome because eGFR was selected as the principal measure of renal function.

Urinary albumin concentration was expressed in mg/L. The albuminuria strata used in the present study were predefined study-specific categories based on urinary albumin concentration.

Laboratory methods

All laboratory investigations included in the study were performed as part of routine clinical monitoring in the outpatient nephrology practice at the Medical Center "St. Anna", Ltd., Varna, Bulgaria. Blood and urine samples were obtained during scheduled outpatient visits, and routine biochemical analyses were performed in the clinical laboratory of the Medical Center "St. Anna", Ltd., Varna, Bulgaria, using a Cobas 6000 analyzer (Roche Diagnostics, Mannheim, Germany), according to the manufacturer's instructions and the laboratory's standard operating procedures and internal quality-control practices.

The laboratory parameters evaluated in the present study included HbA1c, serum creatinine for calculation of eGFR, serum albumin, serum potassium, and urinary albumin concentration. Laboratory results were retrieved retrospectively from the patients' medical records. Longitudinal measurements of urinary albumin concentration, eGFR, serum albumin, and serum potassium were recorded at baseline and at approximately three, six, and 12 months after initiation of finerenone.

HbA1c was determined using an immunoinhibition assay standardized according to the Diabetes Control and Complications Trial/National Glycohemoglobin Standardization Program (DCCT/NGSP). Results were expressed as percentages (%), calculated as the ratio of HbA1c to total hemoglobin (THb). THb was measured using a colorimetric method. An HbA1c level < 6.5% was considered indicative of good glycemic control.

Serum creatinine was measured using a kinetic colorimetric Jaffé method and expressed in μmol/L. Renal function was assessed using the eGFR, calculated from serum creatinine using the Modification of Diet in Renal Disease (MDRD) equation and expressed as mL/min/1.73 m². Although serum creatinine was used for calculation of eGFR, eGFR was selected as the principal measure of renal function for the longitudinal analyses, and serum creatinine was not analyzed as a separate study outcome.

Serum albumin was determined spectrophotometrically and expressed in g/L, with a laboratory reference interval of 35–52 g/L. Serum potassium was measured using a potentiometric ion-selective electrode (ISE) method and expressed in mmol/L, with a laboratory reference interval of 3.5–5.5 mmol/L. Serum potassium was assessed as the principal laboratory safety parameter during finerenone treatment.

Urinary albumin concentration was measured in a single first-morning spot urine sample at each predefined assessment point (baseline and months 3, 6, and 12). This standardized sampling approach was intended to reduce variability related to the timing of urine collection. In routine clinical practice at our center, the UACR was not routinely determined during the study period; therefore, albuminuria monitoring was based on urinary albumin concentration, conventionally reported as microalbuminuria (MAU), and expressed in mg/L. Accordingly, the predefined albuminuria strata (30–300 mg/L, > 300 to < 1000 mg/L, and > 1000 mg/L)

were study-specific categories based on urinary albumin concentration and should not be interpreted as guideline-defined UACR-based albuminuria categories.

All laboratory measurements included in the analysis were obtained as part of routine clinical care rather than specifically for research purposes. The use of the same clinical laboratory and analytical platform throughout the observation period minimized potential inter-laboratory and inter-method analytical variability.

Follow-up assessments

Patients were followed for 12 months after initiation of finerenone. Laboratory measurements were evaluated at four predefined time points: baseline and approximately three, six, and 12 months after treatment initiation.

Urinary albumin concentration, eGFR, serum albumin, and serum potassium were recorded at each of these time points. The same follow-up intervals were used for the longitudinal analyses presented in the study.

For each patient, relative reduction in urinary albumin concentration was calculated at months 3, 6, and 12 in relation to the baseline measurement using the following formula:

$$\text{Relative albuminuria reduction (\%)} = \frac{\text{Baseline albuminuria} - \text{Follow-up albuminuria}}{\text{Baseline albuminuria}} \times 100$$

The absolute change in eGFR from baseline to month 12 was calculated as the 12-month eGFR minus baseline eGFR.

Baseline albuminuria groups

To investigate whether baseline albuminuria severity was associated with the subsequent response to finerenone, patients were stratified into three predefined groups according to baseline urinary albumin concentration.

Group 1 consisted of patients with baseline urinary albumin concentrations of 30–300 mg/L. Group 2 included patients with concentrations >300 to ≤1000 mg/L, and Group 3 included patients with concentrations >1000 mg/L.

These study-specific thresholds were selected to distinguish patients with lower, intermediate, and markedly elevated baseline urinary albumin concentrations and to facilitate comparison of longitudinal treatment responses across different levels of albuminuria severity. They were not intended to correspond to guideline-defined UACR-based albuminuria categories.

Study outcomes

The primary outcome was the relative change in urinary albumin concentration from baseline to 12 months after initiation of finerenone.

Additional albuminuria-related outcomes included changes in urinary albumin concentration at months 3 and 6, the longitudinal change in albuminuria across the complete 12-month observation period, and comparison of the magnitude of the 12-month relative albuminuria reduction among the three baseline albuminuria groups.

Secondary renal and biochemical outcomes included longitudinal changes in eGFR and serum albumin from baseline through months 3, 6, and 12 and the absolute change in eGFR between baseline and month 12.

Safety assessment focused primarily on serum potassium. The longitudinal change in serum potassium was evaluated, and the occurrence of serum potassium >5.5 mmol/L during follow-up was recorded. The proportion of patients requiring finerenone dose reduction from 20 mg/day to 10 mg/day because of serum potassium >5.5 mmol/L was also assessed. The frequency of serum potassium >5.5 mmol/L was compared among the three baseline albuminuria groups using the Fisher-Freeman-Halton exact test.

Statistical analysis

Continuous variables were summarized as mean ± standard deviation (SD), while categorical variables were presented as absolute numbers and percentages. Distributional characteristics of continuous variables were evaluated before statistical testing.

Baseline demographic, clinical, and laboratory characteristics were compared among the three predefined albuminuria groups. Continuous variables were compared using the Kruskal-Wallis test. Because of the small

number of patients in Group 3 and the presence of expected cell frequencies below five, categorical variables were compared using the Fisher-Freeman-Halton exact test, an extension of Fisher's exact test for contingency tables larger than 2×2.

Longitudinal changes in urinary albumin concentration, eGFR, serum albumin, and serum potassium across baseline and the three-, six-, and 12-month assessments were evaluated using the Friedman test for repeated measurements. When the Friedman test demonstrated a statistically significant overall longitudinal difference, post-hoc pairwise comparisons between baseline and each follow-up assessment were performed using the Wilcoxon signed-rank test. Adjustment for multiple comparisons was performed using the Holm method.

The relative reduction in urinary albumin concentration from baseline to month 12 was calculated individually for each patient using the formula

$$\left(\frac{\text{Baseline value} - \text{Month 12 value}}{\text{Baseline value}} \right) \times 100.$$

The mean and SD of the individual

percentage reductions were calculated for the overall cohort. The proportion of patients achieving a reduction of at least 50% was determined from the individual patient-level results. Differences in the 12-month relative albuminuria reduction among the three baseline albuminuria groups were assessed using the Kruskal-Wallis test. Within-group longitudinal changes in albuminuria were assessed using the Friedman test.

The occurrence of serum potassium >5.5 mmol/L requiring finerenone dose reduction was summarized as n (%) for the overall cohort and according to baseline albuminuria group. Between-group differences in hyperkalemia frequency were assessed using the Fisher-Freeman-Halton exact test.

All statistical analyses were performed using IBM SPSS Statistics for Windows, Version 20 (Released 2011; IBM Corp., Armonk, New York, United States). All statistical tests were two-sided, and a p-value <0.05 was considered statistically significant. All longitudinal analyses were performed using complete patient-level data from the 68 included patients. No laboratory measurements were missing at the predefined assessment points; therefore, no missing-data imputation was required.

Results

Patient characteristics

A total of 68 patients with CKD and T2DM who initiated treatment with finerenone were included in the final analysis. All patients received finerenone in addition to background RAS blockade with either an ACEi or an ARB. The initial finerenone dose was determined according to baseline renal function, with patients with an eGFR ≥60 mL/min/1.73 m² receiving 20 mg once daily and those with an eGFR ≥25 to <60 mL/min/1.73 m² receiving 10 mg once daily.

According to baseline urinary albumin concentration, patients were stratified into three predefined groups: Group 1 included 28 patients (41.2%) with albuminuria of 30-300 mg/L, Group 2 included 33 patients (48.5%) with albuminuria >300 to ≤1000 mg/L, and Group 3 included seven patients (10.3%) with albuminuria >1000 mg/L.

The mean age of the study population was 57.7±7.1 years. Thirty-one patients (45.6%) were male, and 37 (54.4%) were female. The mean duration of T2DM was 8.2±2.7 years, and the mean baseline HbA1c was 8.03±1.51%. Twenty-four patients (35.3%) were current smokers, while SBP >140 mmHg was documented in 31 patients (45.6%). Thirty-six patients (52.9%) were receiving an ACE inhibitor and 32 (47.1%) an ARB.

Age, sex distribution, duration of diabetes, smoking status, and the proportion of patients with SBP >140 mmHg were broadly comparable among the three albuminuria groups. Significant between-group differences were observed for baseline HbA1c (p<0.001), serum albumin (p<0.001), and serum potassium (p=0.003). HbA1c was highest and serum albumin lowest among patients with the most pronounced baseline albuminuria. Baseline demographic and clinical characteristics according to albuminuria group are summarized in Table 1.

Characteristic	Overall (n=68)	Group 1 (n=28)	Group 2 (n=33)	Group 3 (n=7)	p-value
Age, years	57.7±7.1	57.8±8.8	57.7±6.0	57.0±4.5	0.990
Male sex, n (%)	31 (45.6)	12 (42.9)	15 (45.5)	4 (57.1)	0.829
Current smoking, n (%)	24 (35.3)	10 (35.7)	12 (36.4)	2 (28.6)	1.000
Diabetes duration, years	8.2±2.7	8.3±2.9	8.1±2.8	8.3±2.1	0.991
HbA1c, %	8.03±1.51	7.30±1.31	8.08±1.09	10.68±0.84	<0.001
SBP >140 mmHg, n (%)	31 (45.6)	14 (50.0)	14 (42.4)	3 (42.9)	0.881
eGFR, mL/min/1.73 m ²	57.0±18.1	58.0±19.4	52.9±17.1	72.5±5.5	0.067
Serum albumin, g/L	43.7±4.7	42.5±3.6	46.9±3.5	33.8±2.8	<0.001
Serum K ⁺ , mmol/L	4.28±0.24	4.16±0.13	4.33±0.26	4.57±0.13	0.003
ACE inhibitor, n (%)	36 (52.9)	16 (57.1)	17 (51.5)	3 (42.9)	0.829
ARB, n (%)	32 (47.1)	12 (42.9)	16 (48.5)	4 (57.1)	0.829

TABLE 1: Baseline characteristics of the study population according to baseline albuminuria.

Values are presented as mean ± SD for continuous variables and number (percentage) for categorical variables. The study population comprised 68 patients with chronic kidney disease and type 2 diabetes mellitus receiving finerenone. Patients were stratified according to baseline urinary albumin concentration into Group 1 (30-300 mg/L; n=28), Group 2 (>300 to ≤1000 mg/L; n=33), and Group 3 (>1000 mg/L; n=7). Between-group comparisons were performed using the Kruskal-Wallis test for continuous variables and the Fisher-Freeman-Halton exact test for categorical variables because of small expected cell frequencies. A two-sided p-value <0.05 was considered statistically significant.

HbA1c: glycated hemoglobin; SBP: systolic blood pressure; eGFR: estimated glomerular filtration rate; ACE: angiotensin-converting enzyme; ARB: angiotensin receptor blocker; SD: standard deviation; n: number of patients

Changes in albuminuria during finerenone treatment

A marked and progressive reduction in urinary albumin concentration was observed throughout the 12-month follow-up period. In the overall cohort, mean albuminuria decreased from 383.4±328.7 mg/L at baseline to 230.0±240.1 mg/L at month 3, 115.6±120.1 mg/L at month 6, and 48.5±50.1 mg/L at month 12.

The longitudinal reduction in albuminuria was highly statistically significant (Friedman $\chi^2=199.48$, $p<0.001$). Pairwise comparisons demonstrated significantly lower albuminuria at months 3, 6, and 12 compared with baseline, with all comparisons remaining significant after correction for multiple testing (adjusted $p<0.001$). The longitudinal changes in albuminuria and other laboratory parameters are summarized in Table 2.

Parameter	Baseline	Month 3	Month 6	Month 12	Overall p-value
Albuminuria, mg/L	383.4±328.7	230.0±240.1	115.6±120.1	48.5±50.1	<0.001
Relative albuminuria reduction, %	Reference	42.4±13.6	71.7±10.7	86.8±6.1	not applicable
eGFR, mL/min/1.73 m ²	57.0±18.1	57.1±18.0	60.7±17.8	65.3±17.7	<0.001
Serum albumin, g/L	43.7±4.7	45.1±4.4	45.5±3.5	45.6±1.8	0.091
Serum K ⁺ , mmol/L	4.28±0.24	4.71±0.25	5.04±0.39	5.05±0.31	<0.001

TABLE 2: Longitudinal changes in renal and laboratory parameters during 12 months of finerenone treatment.

Values are presented as mean ± SD for the overall study cohort (n=68). Urinary albumin concentration, eGFR, serum albumin, and serum potassium were assessed at baseline and at three, six, and 12 months after initiation of finerenone. Overall longitudinal differences were evaluated using the Friedman test for repeated measurements. When a significant overall difference was identified, post-hoc comparisons between baseline and each follow-up assessment were performed using the Wilcoxon signed-rank test with Holm correction for multiple comparisons. Urinary albumin concentration was significantly lower at all follow-up assessments compared with baseline (all adjusted p<0.001). For eGFR, the difference at month 3 was not statistically significant (p=0.173), whereas differences at months 6 and 12 remained significant after adjustment (both adjusted p<0.001). Relative albuminuria reduction was calculated individually as the percentage change from baseline; therefore, the baseline entry is designated as "Reference," and no separate overall longitudinal p-value is applicable to this derived variable. A p-value <0.05 was considered statistically significant.

eGFR: estimated glomerular filtration rate; K⁺: potassium; SD: standard deviation; mg/L: milligrams per liter; g/L: grams per liter; mmol/L: millimoles per liter

Compared with baseline, albuminuria was significantly lower at months 3, 6, and 12 (all Holm-adjusted p<0.001). For eGFR, the difference at month 3 was not significant (p=0.173), whereas the differences at months 6 and 12 were significant after correction for multiple comparisons (both adjusted p<0.001). Relative albuminuria reduction was calculated in relation to the baseline value; therefore, no baseline value or overall longitudinal p-value is applicable.

The mean relative reduction from baseline was 42.4% at month 3 and 71.7% at month 6, reaching 86.8±6.1% at month 12. The median relative reduction at 12 months was 87.6%. All 68 patients demonstrated a reduction of at least 50% in urinary albumin concentration by month 12.

The temporal pattern of albuminuria reduction in the overall study population is illustrated in Figure 1.

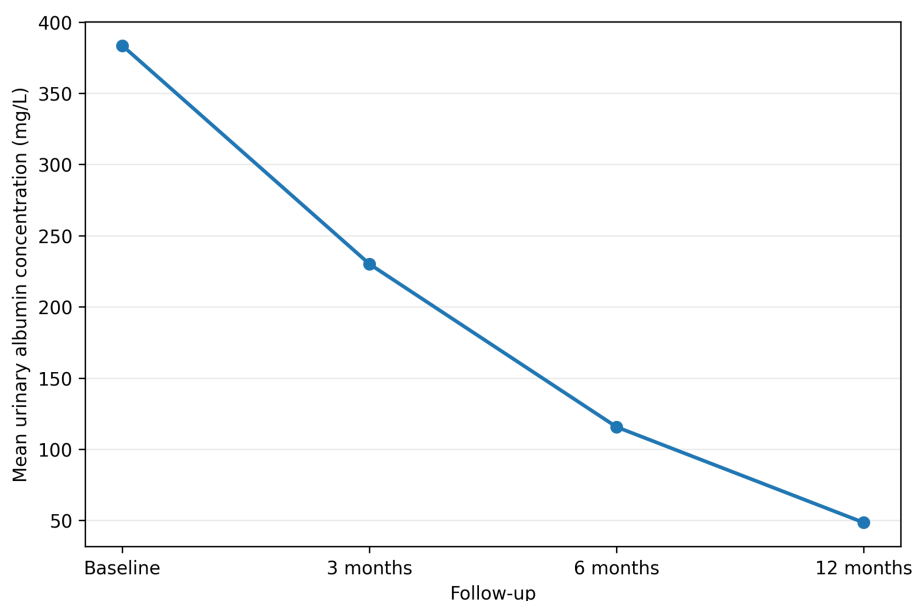


FIGURE 1: Longitudinal changes in mean urinary albumin concentration during 12 months of finerenone treatment.

The figure shows mean urinary albumin concentration in the overall study cohort (n=68) at baseline and at three, six, and 12 months after finerenone initiation. Urinary albumin was measured in spot urine samples and expressed in mg/L. A significant longitudinal reduction was observed (Friedman test, $p<0.001$), with all follow-up values significantly lower than baseline after Holm correction (all adjusted $p<0.001$).

mg/L: milligrams per liter

When analyzed according to baseline albuminuria severity, a significant longitudinal reduction was observed in all three groups (all $p<0.001$). In Group 1, mean urinary albumin concentration decreased from 62.7 ± 40.2 mg/L at baseline to 37.1 ± 24.0 mg/L at month 3, 15.2 ± 9.3 mg/L at month 6, and 7.3 ± 3.4 mg/L at month 12. In Group 2, the corresponding values decreased from 514.0 ± 137.5 mg/L to 274.4 ± 120.4 , 143.4 ± 59.1 , and 62.5 ± 33.2 mg/L, respectively. In Group 3, mean albuminuria decreased from 1050.1 ± 75.6 mg/L at baseline to 792.7 ± 98.7 mg/L at month 3, 386.1 ± 73.2 mg/L at month 6, and 147.2 ± 41.8 mg/L at month 12.

Despite pronounced differences in absolute urinary albumin concentrations, the relative magnitude of the 12-month response was similar among the groups. Mean relative reductions were $85.2\pm8.0\%$ in Group 1, $88.4\pm3.9\%$ in Group 2, and $85.8\pm4.5\%$ in Group 3. The difference in relative albuminuria reduction among the three groups was not statistically significant ($p=0.258$). Group-specific changes in albuminuria are presented in Table 3 and illustrated in Figure 2.

Group	Baseline, mg/L	Month 3, mg/L	Month 6, mg/L	Month 12, mg/L	12-month reduction, %	Within-group p
Group 1 (n=28)	62.7±40.2	37.1±24.0	15.2±9.3	7.3±3.4	85.2±8.0	<0.001
Group 2 (n=33)	514.0±137.5	274.4±120.4	143.4±59.1	62.5±33.2	88.4±3.9	<0.001
Group 3 (n=7)	1050.1±75.6	792.7±98.7	386.1±73.2	147.2±41.8	85.8±4.5	<0.001

TABLE 3: Changes in albuminuria according to baseline albuminuria category.

Values are presented as mean ± SD. Patients were stratified into three predefined groups according to baseline urinary albumin concentration: Group 1 (30-300 mg/L; n=28), Group 2 (>300 to ≤1000 mg/L; n=33), and Group 3 (>1000 mg/L; n=7). Urinary albumin concentration was measured in spot urine samples at baseline and at three, six, and 12 months after initiation of finerenone. Within-group longitudinal changes were evaluated using the Friedman test, demonstrating statistically significant reductions in all three groups (all p<0.001). The 12-month relative albuminuria reduction was calculated individually as the percentage reduction from baseline. Between-group differences in relative reduction were evaluated using the Kruskal-Wallis test and were not statistically significant (p=0.258). The groups represent study-specific urinary albumin concentration categories rather than guideline-defined urinary albumin-to-creatinine ratio categories.

SD: standard deviation; mg/L: milligrams per liter; n: number of patients

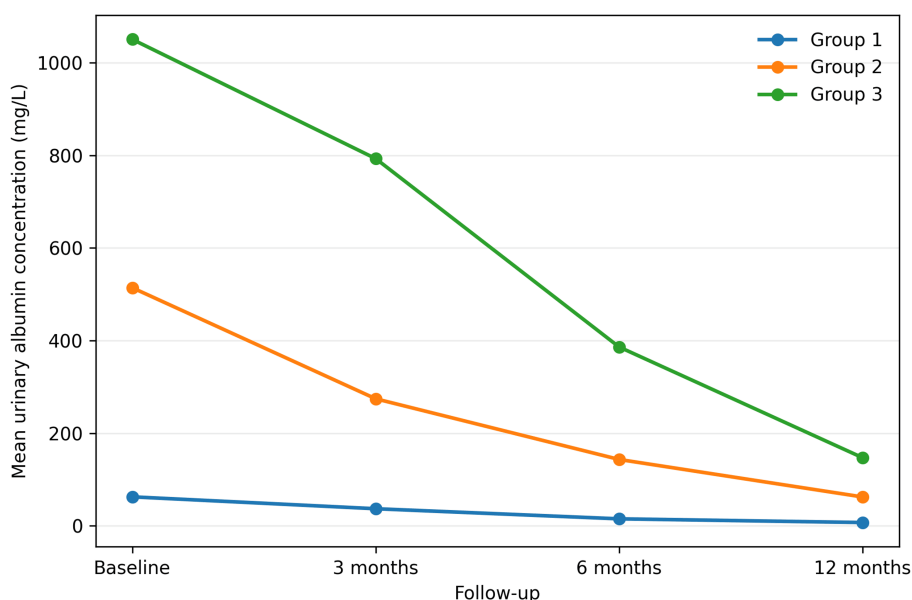


FIGURE 2: Longitudinal changes in mean urinary albumin concentration according to baseline albuminuria category during 12 months of finerenone treatment.

The figure illustrates changes in mean urinary albumin concentration at baseline and at three, six, and 12 months in Group 1 (30-300 mg/L; n=28), Group 2 (>300 to ≤1000 mg/L; n=33), and Group 3 (>1000 mg/L; n=7). Significant longitudinal reductions were observed within all three groups (Friedman test, all p<0.001). The relative 12-month reduction did not differ significantly among groups (Kruskal-Wallis test, p=0.258). The groups were defined according to urinary albumin concentration in spot urine samples rather than urinary albumin-to-creatinine ratio.

mg/L: milligrams per liter

Changes in renal function

Mean eGFR was 57.0±18.1 mL/min/1.73 m² at baseline and remained essentially unchanged at month 3 (57.1±18.0 mL/min/1.73 m²; p=0.173).

At month 6, mean eGFR was 60.7 ± 17.8 mL/min/1.73 m² and increased to 65.3±17.7 mL/min/1.73 m² at month 12. Both the 6- and 12-month measurements differed significantly from baseline (adjusted p<0.001). The mean absolute change in eGFR from baseline to month 12 was +8.25 mL/min/1.73 m².

The mean 12-month changes in eGFR were +5.90 mL/min/1.73 m² in Group 1, +10.31 mL/min/1.73 m² in Group 2, and +7.94 mL/min/1.73 m² in Group 3. The magnitude of eGFR change differed significantly among the three groups ($p=0.016$).

Changes in serum albumin

Mean serum albumin concentration increased numerically from 43.7±4.7 g/L at baseline to 45.1±4.4 g/L at month 3, 45.5±3.5 g/L at month 6, and 45.6±1.8 g/L at month 12; however, the overall longitudinal change did not reach statistical significance ($p=0.091$).

The most pronounced numerical increase was observed among patients with the highest baseline albuminuria. In Group 3, mean serum albumin increased from 33.8 g/L at baseline to 34.6 g/L at month 3, 37.2 g/L at month 6, and 42.1 g/L at month 12, occurring in parallel with the reduction in urinary albumin concentration.

Serum potassium and safety outcomes

Serum potassium increased during follow-up. Mean potassium concentration was 4.28±0.24 mmol/L at baseline, increasing to 4.71±0.25 mmol/L at month 3, 5.04±0.39 mmol/L at month 6, and 5.05±0.31 mmol/L at month 12. The overall longitudinal change was statistically significant ($p<0.001$).

Five additional cases of hyperkalemia (serum potassium >5.5 mmol/L) were identified at the four-week assessment among the 73 patients who initiated finerenone treatment. These patients were excluded from the final longitudinal cohort; therefore, the subsequent safety analysis pertains to the remaining 68 patients.

During the 12-month follow-up, 16 of 68 patients (23.5%) developed at least one serum potassium concentration >5.5 mmol/L. All episodes of serum potassium >5.5 mmol/L occurred among patients receiving finerenone at a dose of 20 mg/day; no such episodes were observed among patients receiving 10 mg/day. No patient exceeded the 5.5 mmol/L threshold at month 3, whereas 15 patients had serum potassium >5.5 mmol/L at month 6 and one patient at month 12. The highest recorded serum potassium concentration was 6.1 mmol/L.

In all 16 patients who developed serum potassium concentrations >5.5 mmol/L, finerenone was temporarily discontinued for two weeks. Following normalization of serum potassium, treatment was reinitiated at a reduced dose of 10 mg once daily. Hyperkalemia requiring temporary treatment interruption and subsequent dose reduction occurred in one of 28 patients (3.6%) in Group 1, 12 of 33 patients (36.4%) in Group 2, and three of seven patients (42.9%) in Group 3. The frequency of hyperkalemia requiring treatment interruption and subsequent dose reduction differed significantly among the three baseline albuminuria groups (Fisher-Freeman-Halton exact test, $p=0.002$).

Discussion

The present real-world study evaluated the 12-month renal and biochemical response to finerenone in patients with CKD and T2DM receiving background RAS blockade. Several clinically relevant findings emerged. First, finerenone treatment was associated with a marked and progressive reduction in urinary albumin concentration throughout the 12-month observation period. Second, although absolute albuminuria differed substantially according to baseline severity, the relative magnitude of albuminuria reduction at 12 months was comparable across the three predefined albuminuria strata. Third, mean eGFR remained essentially unchanged during the first three months and was higher than baseline at months 6 and 12. Finally, serum potassium increased during treatment, with concentrations >5.5 mmol/L occurring exclusively among patients receiving finerenone 20 mg/day and being managed by temporary treatment interruption for two weeks, followed by reinitiation at 10 mg/day after normalization of serum potassium.

Changes in albuminuria during finerenone treatment

The most prominent finding of the present study was the progressive reduction in urinary albumin concentration. Mean albuminuria decreased from 383.4±328.7 mg/L at baseline to 230.0±240.1 mg/L at three months, 115.6±120.1 mg/L at six months, and 48.5±50.1 mg/L at 12 months. This corresponded to mean relative reductions of 42.4%, 71.7%, and 86.8%, respectively. Moreover, all patients demonstrated at least a 50% reduction in urinary albumin concentration by month 12.

These findings are directionally consistent with the established antiproteinuric effect of finerenone demonstrated in the pivotal FIDELIO-DKD and FIGARO-DKD clinical development program [6,7]. In FIDELIO-DKD, finerenone produced an approximately 31% greater reduction in UACR than placebo at month 4, and this effect was maintained during follow-up [6]. The pooled FIDELITY analysis similarly demonstrated an early reduction in UACR of approximately 32% with finerenone compared with placebo [8]. Importantly, mediation analysis of the FIDELIO-DKD and FIGARO-DKD trials demonstrated that the early reduction in albuminuria mediated a substantial proportion of the subsequent kidney benefit of finerenone [9]. These

observations are consistent with broader evidence supporting treatment-induced albuminuria reduction as a clinically relevant surrogate marker for CKD progression [3].

The magnitude of albuminuria reduction observed in our cohort was considerably greater than that reported in the pivotal randomized trials and should therefore be interpreted cautiously. Several methodological and population-related factors may contribute to this difference. Most importantly, the present study assessed urinary albumin concentration in spot urine samples, expressed in mg/L, whereas the pivotal randomized trials assessed albuminuria using UACR. Urinary albumin concentration may be influenced by variations in urine dilution and concentration. Consequently, direct numerical comparison between the percentage changes observed in the present study and UACR-based reductions reported in randomized clinical trials is not appropriate.

In addition, the retrospective observational design may introduce selection effects and regression to the mean, particularly when treatment is initiated in patients selected because of persistently elevated albuminuria. The absence of a parallel untreated control group also prevents separation of the pharmacological effect of finerenone from temporal variability in urinary albumin excretion or other changes in clinical management during follow-up. Nevertheless, the progressive nature of the reduction across multiple time points and its observation across all three baseline albuminuria strata support the presence of a consistent longitudinal signal.

Recent real-world studies provide additional context. In a European real-world cohort of 60 patients with diabetes and CKD, UACR decreased by 37.1% after six months of finerenone treatment [10]. Similarly, a Chinese observational study involving 105 patients reported a 39.55% reduction in UACR after 12 weeks of treatment [11]. These findings support the antiproteinuric effectiveness of finerenone outside randomized clinical trial settings while simultaneously emphasizing that the substantially greater 12-month reduction observed in our cohort requires cautious interpretation.

Additional evidence from routine clinical practice has been provided by MEDFINE-RWD (Real-World Effectiveness and Safety of Finerenone in People With Type 2 Diabetes and Chronic Kidney Disease), a Spanish multicenter retrospective observational study involving 844 patients with CKD and T2DM treated with finerenone. This larger observational cohort provides further evidence on albuminuria response and treatment safety in routine clinical practice [13].

Response according to baseline albuminuria

An important finding of the present analysis was that the relative response to finerenone appeared broadly consistent across the spectrum of baseline albuminuria. Despite substantial differences in absolute urinary albumin concentration, the mean 12-month relative reductions were 85.2% in Group 1, 88.4% in Group 2, and 85.8% in Group 3, with no statistically significant difference among groups.

This observation suggests that, within the limitations of the present cohort, baseline severity of albuminuria did not substantially modify the proportional albuminuria response during finerenone treatment. Patients with markedly elevated baseline albuminuria experienced larger absolute reductions, whereas the proportional response remained similar. This finding may be clinically relevant because higher albuminuria identifies patients at increased risk of progressive kidney disease and adverse cardiorenal outcomes [1,3].

Changes in renal function

Mean eGFR remained essentially unchanged during the initial three months of therapy but subsequently increased from 57.0 ± 18.1 mL/min/1.73 m² at baseline to 60.7 ± 17.8 mL/min/1.73 m² at six months and 65.3 ± 17.7 mL/min/1.73 m² at 12 months. The mean absolute difference between baseline and month 12 was +8.25 mL/min/1.73 m².

This pattern differs from that generally observed in randomized finerenone trials. In FIDELIO-DKD, finerenone was associated with a modest early decline in eGFR followed by a slower chronic rate of kidney function decline compared with placebo [6]. A similar early hemodynamic pattern followed by longer-term preservation of kidney function has been described across the finerenone clinical development program [8].

Accordingly, the increase in mean eGFR observed in the present cohort should not be interpreted as evidence that finerenone directly increased glomerular filtration. Several factors may have contributed, including biological variability in eGFR, changes in volume status, metabolic or blood pressure control, regression to the mean, and other unmeasured changes in clinical management. Furthermore, because this was an uncontrolled retrospective study, the counterfactual eGFR trajectory in the absence of finerenone is unknown. The present finding is therefore best interpreted as preservation of renal function accompanied by a higher observed mean eGFR during follow-up rather than as proof of a treatment-induced improvement in filtration.

The magnitude of eGFR change differed among the baseline albuminuria groups, with the largest mean

increase observed in Group 2. However, given the small number of patients in Group 3 and the absence of randomization or adjustment specifically designed to compare eGFR trajectories among these strata, this between-group difference should be interpreted cautiously.

Serum albumin

Serum albumin increased numerically during follow-up, from 43.7 ± 4.7 g/L at baseline to 45.6 ± 1.8 g/L at month 12, although the overall longitudinal difference did not reach statistical significance. The most pronounced numerical increase was observed among patients with the highest baseline albuminuria, in whom mean serum albumin increased from 33.8 g/L to 42.1 g/L.

The parallel reduction in urinary albumin concentration and numerical increase in serum albumin among patients with the greatest baseline albuminuria is biologically plausible. Nevertheless, because the overall serum albumin change was not statistically significant and Group 3 contained only seven patients, no firm conclusion regarding an effect of finerenone on serum albumin can be drawn from these observations. This finding should therefore be regarded as descriptive and hypothesis-generating.

Serum potassium and safety

Hyperkalemia represents the principal clinically relevant safety concern associated with mineralocorticoid receptor antagonism [12]. In the present study, 16 patients (23.5%) developed at least one serum potassium concentration >5.5 mmol/L during follow-up.

This finding can be directly compared with the laboratory-defined hyperkalemia analysis from FIDELIO-DKD, in which serum potassium >5.5 mmol/L occurred in 21.4% of finerenone-treated patients compared with 9.2% receiving placebo [12]. More severe hyperkalemia, defined as serum potassium >6.0 mmol/L, occurred in 4.5% and 1.4%, respectively [12]. These data demonstrate that potassium elevation represents an expected pharmacological consequence of finerenone treatment but can generally be managed through structured biochemical monitoring and treatment modification.

Additional real-world safety data have been reported in the prospective FINE-REAL (Non-interventional Study to Collect Real-World Data and Provide Insights Into the Use of Finerenone in Routine Clinical Practice) study, which included 1,916 patients with CKD and T2DM receiving finerenone in routine clinical practice. Hyperkalemia was reported as an adverse event in 8% of participants. However, differences in study design, monitoring, and hyperkalemia definitions preclude direct comparison with the laboratory-defined potassium elevations observed in our cohort [14].

In the present cohort, hyperkalemia was managed by temporary discontinuation of finerenone for two weeks, followed by reinitiation at 10 mg/day after normalization of serum potassium. This approach highlights the importance of biochemical monitoring and treatment adjustment in patients developing hyperkalemia during finerenone therapy.

The observation that all episodes in our cohort occurred among patients receiving 20 mg/day is noteworthy but should not be interpreted as demonstrating a causal dose-dependent risk. Initial finerenone dose was determined by baseline eGFR rather than randomly assigned, creating important differences between the 10-mg and 20-mg groups. In FIDELIO-DKD, higher baseline serum potassium, lower eGFR, and greater UACR were among the factors associated with an increased risk of hyperkalemia [12]. This is particularly relevant to the present cohort because baseline serum potassium differed significantly among the three albuminuria strata.

Clinical implications

The present findings add real-world evidence supporting the antiproteinuric effect of finerenone in patients with CKD and T2DM receiving RAS blockade. The observation of a progressive reduction in urinary albumin concentration across all baseline albuminuria strata suggests that a proportional treatment response may occur across a broad range of albuminuria severity. At the same time, the potassium findings reinforce the importance of biochemical monitoring and individualized dose management.

An important feature of the present cohort is the exclusion of patients receiving SGLT2 inhibitors or GLP-1 receptor agonists. This design reduced the potential confounding effect of concomitant therapies capable of influencing albuminuria and renal outcomes and therefore allowed a more isolated assessment of longitudinal changes during finerenone treatment. However, this feature also limits the generalizability of our findings to contemporary clinical practice, in which finerenone may be used as part of multidrug cardiorenal protective therapy [1,2]. This approach is also reflected in the 2026 American Diabetes Association Standards of Care, which emphasize individualized, comprehensive kidney and cardiovascular risk reduction in patients with type 2 diabetes and CKD [15].

A prespecified FIDELITY analysis demonstrated that the cardiovascular and kidney benefits of finerenone were observed irrespective of baseline SGLT2 inhibitor use, while concomitant SGLT2 inhibitor therapy was

associated with fewer hyperkalemia events [16]. More recently, the randomized CONFIDENCE trial directly evaluated simultaneous initiation of finerenone and empagliflozin in patients with CKD and T2DM. At 180 days, combination therapy produced a 29% greater reduction in UACR than finerenone alone and a 32% greater reduction than empagliflozin alone [17]. These findings support an increasingly integrated approach to cardiorenal risk reduction and are particularly relevant when interpreting the generalizability of the present cohort, in which SGLT2 inhibitor use was intentionally excluded.

Strengths and limitations

Several strengths of this study should be acknowledged. The 12-month follow-up provides information beyond the early treatment period evaluated in several available real-world studies. Repeated measurements at three, six, and 12 months allowed assessment of the temporal pattern of albuminuria, eGFR, serum albumin, and potassium changes. The exclusion of patients receiving SGLT2 inhibitors, GLP-1 receptor agonists, other mineralocorticoid receptor antagonists, and calcium dobesilate reduced several important sources of pharmacological confounding. Furthermore, stratification according to baseline albuminuria allowed assessment of whether proportional response differed according to initial disease severity.

The study also has important limitations. First, its retrospective, single-center observational design limits causal inference and may introduce selection bias and residual confounding. Furthermore, the exclusion of patients receiving SGLT2 inhibitors or GLP-1 receptor agonists, although intended to reduce confounding from concomitant kidney-protective therapies, may have introduced selection bias. This limits the generalizability of our findings to patients receiving contemporary combination treatment regimens. Second, the relatively small overall sample size ($n=68$), particularly the limited number of patients in the highest-albuminuria group ($n=7$), reduced statistical power and limited the precision and generalizability of subgroup analyses. Third, no untreated or placebo control group was available; consequently, changes over time cannot be attributed exclusively to finerenone.

Fourth, albuminuria was assessed as urinary albumin concentration in first-morning spot urine samples and expressed in mg/L rather than as the UACR. Although standardized first-morning sampling was intended to reduce variability related to collection timing, urinary albumin concentration remains influenced by urine dilution and hydration status. Consequently, the study-specific albuminuria strata should not be directly equated with guideline-defined UACR-based albuminuria categories, and the observed percentage reductions should not be interpreted as equivalent to changes in UACR or urinary albumin excretion. Direct quantitative comparisons with studies using UACR should therefore be interpreted cautiously. This limitation is consistent with previous recommendations emphasizing the importance of standardizing urinary albumin measurements and accounting for variations in urine concentration [18].

Fifth, the magnitude of the observed albuminuria reduction was substantially larger than that reported in randomized trials and most available real-world studies. Regression to the mean, selection of patients with elevated baseline albuminuria, biological variability, changes in urine concentration, adherence to background therapy, and unmeasured changes in clinical management may have contributed to this finding.

Sixth, five patients who developed serum potassium concentrations >5.5 mmol/L at the four-week assessment after finerenone initiation were excluded from the final longitudinal cohort. Consequently, the safety analysis was restricted to patients who had normal serum potassium concentrations at this early assessment. This selection may underestimate the frequency of hyperkalemia among all patients initiating finerenone and limits the generalizability of the safety findings.

Despite these limitations, the present study provides clinically relevant real-world observations regarding the longitudinal albuminuria response, renal function trajectory, and potassium-related safety of finerenone over 12 months in patients with CKD and T2DM treated with background RAS blockade.

Conclusions

In this retrospective, single-center study of patients with CKD and T2DM receiving background RAS blockade, finerenone treatment was associated with a progressive reduction in urinary albumin concentration over 12 months. Comparable proportional reductions were observed across the three predefined baseline albuminuria groups. Mean eGFR was higher at six and 12 months than at baseline, although the uncontrolled observational design does not permit attribution of this change to finerenone.

Serum potassium increased during follow-up, and 23.5% of patients developed potassium concentrations >5.5 mmol/L, requiring temporary treatment interruption followed by dose reduction. These findings highlight the importance of potassium monitoring during finerenone treatment. Because albuminuria was assessed using urinary albumin concentration in spot samples rather than UACR, and no control group was available, the magnitude and causality of the observed response require confirmation in larger prospective controlled studies.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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Disclosures

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