

Efficacy and Safety of Finerenone in Non-Diabetic Chronic Kidney Disease: A Real-world Retrospective Study

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Abstract

Background

Finerenone, a selective nonsteroidal mineralocorticoid receptor antagonist (MRAs), has demonstrated cardiorenal benefits in patients with diabetic kidney disease (DKD). However, evidence regarding its effectiveness and safety in non-diabetic chronic kidney disease (non-DKD) still remains limited. This study was designed to evaluate the real-world efficacy and safety of finerenone in non-DKD and compare its performance with other treatment strategies.

Methods

We conducted a single-center, retrospective cohort study including 281 patients with chronic kidney disease from August 2023 to June 2025. Patients were categorized into three groups: non-diabetic CKD treated with finerenone (nonDKDF, $n = 151$), DKD treated with finerenone (DKDF, $n = 65$), and non-DKD treated with renin–angiotensin system inhibitors plus SGLT-2 inhibitors (nonDKDAS, $n = 65$). Primary outcomes included changes in urinary albumin-to-creatinine ratio (UACR), estimated glomerular filtration rate (eGFR), serum potassium, and systolic blood pressure (SBP) over 12 months. Subgroup analyses were performed based on glomerular disease pathology.

Results

Patients in the nonDKDF group showed significantly greater reductions in UACR compared to both DKDF and nonDKDAS groups at all time points, particularly at 6 month time ($P < 0.01$). Renal function remained stable in the nonDKDF group, while eGFR declined in the comparator groups. Serum potassium levels remained within the safe range in all groups. In membranous nephropathy, finerenone exhibited the most pronounced antiproteinuric response, and multivariable regression analysis showed that membranous nephropathy is an independent risk factor for UACR decline ($\beta = -947.6$, $P = 0.019$). No serious adverse events related to hyperkalemia or hypotension were found.

Conclusion

Finerenone demonstrated favorable efficacy and safety in patients with non-diabetic CKD, particularly in those with membranous nephropathy. These findings support its potential role as an adjunctive therapy in glomerular diseases with persistent proteinuria beyond diabetic populations.

1. Introduction

Chronic kidney disease (CKD) is a leading global cause of mortality, affecting more than 10% of the population worldwide, or approximately 800 million individuals [1,2]. Non-diabetic CKD accounts for

nearly half of all cases of end-stage renal disease (ESRD) [1,3]. In clinical practice, a reduction in the urinary albumin-to-creatinine ratio (UACR) by at least 20–30% is a well-validated surrogate marker for delaying CKD progression in patients with moderate-to-severe albuminuria [4,5]. Current proteinuria-targeting therapies include renin–angiotensin system inhibitors (RASi), sodium–glucose cotransporter-2 inhibitors (SGLT-2i), and mineralocorticoid receptor antagonists (MRAs) [5–8].

Finerenone is a nonsteroidal MRA (NS-MRA) with greater selectivity ($K_d = 1.52$ nM) and higher binding affinity for mineralocorticoid receptors compared to steroidal MRAs [9]. Its anti-fibrotic and anti-inflammatory properties contribute to meaningful reductions in proteinuria and preservation of renal function, while posing a lower risk of hyperkalemia and sex hormone–related adverse events [10–12]. In large phase III trials, finerenone reduced the risk of CKD progression by 18% and cardiovascular events by 14% in patients with diabetic kidney disease (DKD), leading to FDA approval in July 2021 for DKD treatment [13,14].

Given that mineralocorticoid receptor overactivation drives inflammation, oxidative stress, and fibrosis across CKD etiologies [15], it is plausible that finerenone may offer similar renoprotective benefits in non-diabetic CKD. In this retrospective study, we evaluated the efficacy and safety of finerenone in patients with non-diabetic CKD, and compared its effectiveness with standard combination therapy using RASi plus SGLT-2i. We further assessed differential treatment responses between patients with DKD and non-diabetic CKD, focusing on finerenone’s albuminuria-lowering effects and renal safety profile.

2. Methods

2.1 Ethics

This study was approved by the Ethics Committee of Guangdong Provincial People's Hospital (Approval No. KY2024-760-02) and all procedures were conducted in accordance with the ethical standards of the institutional review board and the principles of the Declaration of Helsinki. Written informed consent was obtained from each patient and patient information is presented in supplementary Table S1.

2.2 Study Design and Population

This retrospective, single-center cohort study was conducted at Guangdong Provincial People's Hospital between August 2023 and June 2025. Adult patients aged 18 years or older with biopsy-proven or clinically diagnosed CKD were included. Participants were categorized into three groups: non-diabetic CKD patients treated with finerenone (nonDKDF), diabetic CKD patients treated with finerenone (DKDF), and non-diabetic CKD patients treated with a combination of RASi and SGLT2i (nonDKDAS). Inclusion criteria were as follows: (1) age ≥ 18 years; (2) patients with a clinical diagnosis of CKD, as defined by KDIGO 2024 criteria (abnormal kidney structure or function lasting ≥ 3 months); (3) patients receiving finerenone treatment should at least be on medication for at least six months; (4) had available baseline and at least one follow-up measurement of UACR, estimated glomerular filtration rate (eGFR), serum potassium (K^+), or systolic blood pressure (SBP). Exclusion criteria included baseline eGFR below 15

mL/min/1.73 m², active malignancy, prior kidney transplantation, or incomplete follow-up data. Clinical and laboratory data were extracted from electronic medical records, and all analyses were performed using anonymized datasets.

2.3 Data Collection and Variables

Clinical and laboratory data were retrospectively extracted from the hospital's electronic medical record system. Baseline demographic information included age, sex, and renal pathology, where available. Biochemical measurements included UACR, UPCR, serum creatinine, eGFR, serum potassium, and SBP. These parameters were recorded at baseline and at 3, 6, 9, and 12 months following treatment initiation. Medication use was documented for RASi, SGLT-2i, and immunosuppressive agents, both prior to and during treatment. eGFR was calculated using the CKD-EPI equation. Renal pathology diagnoses were based on biopsy results when available and were categorized into specific subtypes, including IgA nephropathy, membranous nephropathy, lupus nephritis, focal segmental glomerulosclerosis, minimal change disease, mesangial proliferative glomerulonephritis, and non-biopsied CKD. Data completeness at each time point was assessed prior to analysis, and all biochemical results were validated by trained nephrologists prior to final dataset lock.

2.4 Treatment Groups

Patients were classified into three treatment groups according to disease states and prescribed therapeutic regimen. The nonDKDF group included non-diabetic CKD patients who received finerenone at a fixed dose of 10 mg once daily for a minimum of six months. The DKDF group comprised diabetic CKD patients treated with the same finerenone regimen. The nonDKDAS group included non-diabetic CKD patients who received concurrent therapy with a RASi and an SGLT-2i, without finerenone. All medications were prescribed and adjusted by attending nephrologists based on clinical judgment. Concomitant use of immunosuppressive agents, diuretics, or other nephroprotective therapies was permitted and recorded. Group classification was based on treatment received at the time of finerenone initiation or, for the nonDKDAS group, the initiation of dual therapy. All patients were followed for 6 to 12 months.

2.5 Outcome Measures

The primary outcome was the percentage change in UACR (Δ UACR%) from baseline to 12 months. Secondary outcomes included the percentage change in UPCR (Δ UPCR%), absolute change in eGFR rate (Δ eGFR), and changes in serum potassium concentration. Response to therapy was further categorized based on UACR reduction thresholds of $\geq 30\%$, $\geq 50\%$, and $\geq 75\%$, assessed at 3, 6, 9, and 12 months. The proportion of patients achieving each response level was compared across treatment groups and within pathological subtypes. Adverse renal outcomes, including progression to eGFR < 15 mL/min/1.73 m² or a decline of $\geq 40\%$ from baseline, were recorded but not modeled due to low event frequency. Hyperkalemia was defined as serum potassium > 5.5 mmol/L, and its occurrence was descriptively reported. All outcomes were evaluated using the last available value for each time point, and missing intermediate data points did not preclude inclusion in longitudinal analyses.

2.6 Statistical Analysis

All statistical analyses were performed using R (version 4.2.2) and SPSS (version 26.0). Continuous variables are expressed as mean $\pm$ standard deviation (SD) or median with interquartile range (IQR), depending on distribution normality, and compared using one-way ANOVA or the Kruskal–Wallis test, as appropriate. Categorical variables are presented as frequencies and percentages and were compared using the Chi-square or Fisher's exact test. For comparisons involving more than two groups, post hoc analyses were conducted using the Tukey's honestly significant difference (HSD) test to adjust for multiple pairwise comparisons following ANOVA.

To assess longitudinal trends in UACR, eGFR, and serum potassium, linear mixed-effects models were applied with treatment group, time point, and group-by-time interaction included as fixed effects, and patient ID as a random effect. This approach accommodated missing intermediate values under the assumption of data missing at random.

The primary outcome (Δ UACR%) and secondary outcomes (Δ UPCR%, Δ eGFR, and Δ K⁺) were analyzed using multivariate linear regression to identify independent predictors of response. Covariates included age, sex, baseline values of each respective outcome, medication exposure (RASi, SGLT2i, immunosuppressants), and pathological diagnosis. Variables were selected a priori based on clinical relevance and prior published literature [1]. Due to the low incidence of renal progression and hyperkalemia, time-to-event or logistic models were not performed.

3. Results

3.1 Baseline Characteristics

From August 2023 to June 2025, 413 CKD patients treated with either finerenone or a combination of RASi and SGLT-2i were screened, including 86 patients in the DKDF group, 239 in the nonDKDF group, and 88 in the nonDKDAS group. After excluding patients with a baseline eGFR below 15 mL/min/1.73 m², active malignancy, history of kidney transplantation, or incomplete follow-up data, a total of 281 patients were included in the final analysis, comprising 151 nonDKDF group, 65 DKDF group, and 65 nonDKDAS group (Fig. 1). In nonDKDF group, the underlying etiologies included 71 cases of IgA nephropathy (IgAN), 51 of membranous nephropathy (MN), 16 of non-biopsied CKD, 5 of lupus nephritis (LN), 3 of minimal change disease (MCD), 3 of mesangial proliferative glomerulonephritis (MGPN), and 2 of focal segmental glomerulosclerosis (FSGS) (Fig. 1). Baseline characteristics are detailed in Table 1.

Table 1
Baseline Clinical Characteristics of Study Participants

Baseline Characteristics	nonDKDF (N = 151)	DKDF (N = 65)	<i>P</i> value nonDKDF vs. DKDF	nonDKDAS (N = 65)	<i>P</i> value nonDKDF vs. nonDKDAS
Age (years)	47.54 ± 15.63	54.15 ± 13.36	< 0.001	46.02 ± 14.45	0.506
Male, n (%)	63 (42%)	33 (51%)	0.241	33 (51%)	0.241
Female, n (%)	88 (58%)	32 (49%)	0.241	32 (49%)	0.241
SBP (mmHg)	139 ± 14.8	144 ± 10.5	0.045	137 ± 8.9	0.263
UACR (mg/g)	727.4 (423.4, 1669.7)	685.7 (312.3, 1227.1)	0.272	458.9 (270.6, 980.4)	0.002
≤ 300 mg/g, n (%)	22 (15%)	20 (31%)		22 (34%)	
> 300 mg/g, n (%)	129 (85%)	45 (69%)		43 (66%)	
UPCR (mg/g)	1011.4 (534.5, 1951.2)	884.5 (492.3, 1745.0)	0.298	738.4 (426.7, 1483.9)	0.014
Scr (μmol/L)	105.88 ± 39.92	115.19 ± 46.17	< 0.001	96.31 ± 29.21	< 0.001
eGFR (ml/min/1.73m ²)	67.82 ± 28.06	62.34 ± 27.18	0.070	73.79 ± 25.69	0.097
< 60, n (%)	60 (40%)	33 (51%)		19 (29%)	
60–90, n (%)	58 (38%)	22 (34%)		29 (45%)	
> 90, n (%)	33 (22%)	10 (15%)		17 (26%)	
K ⁺ (mmol/L)	4.13 ± 0.43	4.26 ± 0.42	0.043	4.09 ± 0.39	0.318
RASi, n (%)	83 (55%)	37 (57%)	0.790	65 (100%)	< 0.001
SGLT-2i, n (%)	56 (37%)	30 (46%)	0.222	65 (100%)	< 0.001
Immunosuppressants, n (%)	51 (34%)	10 (15%)	0.007	4 (6%)	< 0.001
Values are expressed as mean ± standard deviation or median (interquartile range) for continuous variables, and number (percentage) for categorical variables.					

Compared with the DKDF group, nonDKDF patients were younger (47.5 ± 15.6 vs. 54.2 ± 13.4 years, *P* < 0.001), had lower baseline serum potassium (4.13 ± 0.43 vs. 4.26 ± 0.42 mmol/L, *P* = 0.043), lower baseline SBP (139 ± 14.8 vs. 144 ± 10.5 mmHg, *P* = 0.045), and more frequently received

immunosuppressive therapy (34% vs. 15%, $P = 0.007$). No significant differences were observed in baseline eGFR, UACR, or UPCR between these two groups.

When compared to the nonDKDAS group, nonDKDF patients had significantly higher baseline levels of UACR (727.4 vs. 458.9 mg/g, $P = 0.002$), UPCR (1011.4 vs. 738.4 mg/g, $P = 0.014$), and serum creatinine (105.9 ± 39.9 vs. 96.3 ± 29.2 $\mu\text{mol/L}$, $P < 0.001$). No significant differences were observed in baseline SBP. Immunosuppressant use was also more frequent in the nonDKDF group (34% vs. 6%, $P < 0.001$). RASi and SGLT-2i were administered to all patients in the nonDKDAS group, in contrast to significantly lower usage rates observed in the nonDKDF group (both $P < 0.001$).

These differences underscore the need for adjusted analyses in subsequent outcome comparisons across treatment groups.

3.2 Longitudinal Renal and Safety Outcomes

Figure 2 illustrates the changes in UACR, eGFR, serum potassium and SBP over 12 months across three treatment groups. All groups showed a significant reduction in UACR over time ($P < 0.01$ by Friedman test). Notably, patients in the nonDKDF group exhibited the most pronounced decrease, with significant group-by-time interaction effects when compared with both the DKDF group ($\beta = -49.69$, $P = 0.008$) and the nonDKDAS group ($\beta = -96.76$, $P < 0.001$). These results underscore the superior antiproteinuric efficacy of finerenone in non-diabetic CKD compared to other treatment regimens.

Renal function, as measured by eGFR, declined significantly over time in the DKDF ($\chi^2 = 30.12$, $P < 0.001$) and nonDKDAS ($\chi^2 = 16.39$, $P = 0.003$) groups. These changes may reflect expected hemodynamic effects of RAAS blockade. In contrast, the eGFR remained stable in the nonDKDF group throughout the 12-month follow-up period ($P = 0.539$), and no significant interaction was detected between this group and either comparator ($P > 0.20$), indicating a potential renoprotective effect of finerenone in non-diabetic CKD patients.

Serum potassium levels remained within a stable range across all groups during follow-up. No significant temporal changes were observed (Friedman $P > 0.45$), and group-by-time interaction effects were non-significant between the nonDKDF and DKDF groups ($P = 0.678$) and marginal between the nonDKDF and nonDKDAS groups ($\beta = 0.04$, $P = 0.065$). These findings suggest that potassium homeostasis was preserved, and clinically relevant hyperkalemia was uncommon across all treatment groups.

SBP declined over time in all cohorts, with the DKDF group demonstrating the most pronounced reduction ($\chi^2 = 18.40$, $P = 0.0010$), followed by the nonDKDAS group ($\chi^2 = 9.12$, $P = 0.028$). A marginal, non-significant trend toward SBP reduction was observed in the nonDKDF group ($\chi^2 = 7.20$, $P = 0.0661$). Group-by-time interaction analyses revealed significantly attenuated SBP reductions in both nonDKDF ($\beta = -0.26$, $P = 0.041$) and nonDKDAS ($\beta = -0.31$, $P = 0.019$) compared with the DKDF cohort.

3.3 Treatment Response by Subgroup

The proportion of patients achieving $\geq 30\%$ reduction in urinary UACR at 3, 6, 9, and 12 months is shown in Table 2, stratified by treatment group and pathological subtype.

Table 2 UACR Response Rate ($\geq 30\%$ Reduction) at 3, 6, 9, and 12 Months Across Treatment and Pathological Subgroups				
Group	Month 3	Month 6	Month 9	Month 12
DKDF	28/57 (49.1%)	40/61 (65.6%)	31/43 (72.1%)	20/30 (66.7%)
nonDKDAS	26/62 (41.9%)	31/58 (53.4%)	27/52 (51.9%)	13/28 (46.4%)
nonDKDF	82/137 (59.9%)	108/138 (78.3%)	61/85 (71.8%)	34/43 (79.1%)
IgA nephropathy	38/67 (56.7%)	50/67 (74.6%)	31/45 (68.9%)	17/21 (81.0%)
Membranous nephropathy	31/44 (70.5%)	38/44 (86.4%)	20/28 (71.4%)	12/15 (80.0%)
Lupus nephritis	1/5 (20.0%)	2/4 (50.0%)	2/4 (50.0%)	1/1 (100.0%)
Minimal change disease	1/2 (50.0%)	3/3 (100.0%)	1/1 (100.0%)	1/1 (100.0%)
FSGS	0/2 (0.0%)	2/2 (100.0%)		
Mesangial proliferative GN	1/2 (50.0%)	2/3 (66.7%)	1/1 (100.0%)	1/1 (100.0%)
CKD without renal biopsy	9/14 (64.3%)	10/14 (71.4%)	5/5 (100.0%)	2/4 (50.0%)
This table summarizes the proportion of patients achieving a $\geq 30\%$ reduction in urinary albumin-to-creatinine ratio (UACR) from baseline at 3, 6, 9, and 12 months. The response rate is presented as both frequency and percentage. Groups include treatment-based classifications (DKDF, nonDKDAS, nonDKDF) and pathological subgroups among nonDKDF patients (IgA nephropathy, membranous nephropathy, lupus nephritis, minimal change disease, FSGS, mesangial proliferative glomerulonephritis, and CKD without renal biopsy).				

Across treatment groups, the nonDKDF group consistently demonstrated the highest response rates, with 59.9%, 78.3%, 71.8%, and 79.1% of patients achieving $\geq 30\%$ UACR reduction at 3, 6, 9, and 12 months, respectively. By contrast, response rates in the DKDF and nonDKDAS groups were lower, particularly at month 12 (66.7% and 46.4%, respectively). These findings suggest that finerenone may exert a more sustained antiproteinuric effect in non-diabetic CKD patients compared to diabetic CKD or conventional RASi+SGLT2i therapy.

Subgroup analysis within the nonDKDF cohort revealed variability in treatment response by pathological subtype. MN patients exhibited consistently high response rates exceeding 70% at all time points, with 80.0% responding by month 12. IgAN patients also responded favorably, with response rates increasing from 56.7% at month 3 to 81.0% at month 12. Other subtypes, including lupus nephritis, minimal change

disease, mesangial proliferative glomerulonephritis (MPGN), and CKD without biopsy, showed variable but generally positive trends.

Although the number of patients was limited in rare subtypes (e.g., FSGS, MCD), preliminary patterns suggest potential responsiveness to finerenone, warranting further evaluation in expanded multi-center cohort studies.

3.4 Response Magnitude and Temporal Trends

Temporal trends in the proportion of patients achieving $\geq 30\%$, $\geq 50\%$, and $\geq 75\%$ reductions in UACR across the three treatment groups are shown in Fig. 3.

The nonDKDF group consistently exhibited the highest response rates across all thresholds and time points, particularly at month 6. At this time point, significant between-group differences were observed: 76.8% of nonDKDF patients achieved $\geq 30\%$ UACR reduction (vs. 63.1% in DKDF and 41.5% in nonDKDAS, $P = 0.0035$), 57.6% achieved $\geq 50\%$ reduction (vs. 38.5% and 24.5%, $P = 0.0019$), and 31.1% achieved $\geq 75\%$ reduction (vs. 15.4% and 7.5%, $P = 0.0166$). These results suggest that finerenone elicits a more rapid and substantial antiproteinuric effect in non-diabetic CKD compared to either diabetic CKD or dual RASi+SGLT2i therapy.

Although response rates declined slightly or plateaued after month 6 in all groups, the early treatment window appeared to be the most discriminative. By month 12, the response advantage in the nonDKDF group remained apparent, though between-group differences were no longer statistically significant.

These findings support the notion that early initiation of finerenone may offer distinct renal benefits in non-diabetic CKD, particularly in the first 6 months of therapy.

3.5 Renal Response by Pathological Subtype

Among nonDKDF patients with IgAN and MN, longitudinal changes in UACR, eGFR, and serum potassium were assessed over the 12-month follow-up (Fig. 4). Both subgroups demonstrated significant reductions in UACR over time ($P < 0.001$ for both), with MN patients exhibiting a more pronounced decrease compared to those with IgAN. A significant group-by-time interaction was observed ($\beta = -96.77$, $P = 0.002$), suggesting that finerenone may exert a stronger antiproteinuric effect in MN, potentially reflecting differing patterns of glomerular injury.

Renal function remained stable in both subgroups throughout the study period. The eGFR trajectories did not show statistically significant within-group decline (IgAN: $\chi^2 = 1.31$, $P = 0.860$; MN: $\chi^2 = 4.46$, $P = 0.347$), and no significant interaction between groups was found ($\beta = 0.956$, $P = 0.195$). These findings further support the renal safety of finerenone in both pathological types of glomerular diseases.

Serum potassium levels also remained stable throughout the 12-month period in both groups. There were no significant temporal changes (IgAN: $\chi^2 = 1.10$, $P = 0.894$; MN: $\chi^2 = 2.93$, $P = 0.569$), and no significant interaction was observed between the subtypes ($\beta = -0.006$, $P = 0.587$).

SBP decreased over time in IgAN patients ($\chi^2 = 16.88$, $P = 0.002$), while remained stable in MN patients ($\chi^2 = 8.44$, $P = 0.076$). The group-by-time interaction was statistically significant ($\beta = -0.41$, $P = 0.038$). Notably, no episodes of hypotension were documented in either subgroup throughout the treatment period. Overall, these results suggest that finerenone is both effective and has tolerable side effects in patients with IgAN and MN, with MN patients potentially experiencing a greater reduction in albuminuria.

3.6 Comparison of Response Distribution Between Pathological Subtypes

The distribution of urinary UACR response thresholds at 3, 6, 9, and 12 months was compared between patients with IgAN and MN within the nonDKDF group, as illustrated in Fig. 5. No statistically significant differences were observed between the two groups at any time point for the predefined thresholds of $\geq 30\%$, $\geq 50\%$, and $\geq 75\%$ UACR reduction, with all P -values greater than 0.05 by chi-square test.

Numerically, MN patients demonstrated higher response rates across most time points and thresholds. At 6month time, 74% of MN patients achieved at least 30% UACR reduction compared to 67% of patients with IgAN. For the $\geq 50\%$ threshold, the response rates were 57% in the MN group and 49% in the IgAN group. At the $\geq 75\%$ threshold, 33% of MN patients responded compared to 21% in the IgAN group. Although these differences did not reach statistical significance, the consistent trend toward higher response rates in MN suggests a potentially stronger antiproteinuric effect in this subgroup.

Overall, the results indicate that both IgAN and MN patients benefit from finerenone treatment, with broadly similar response distributions. These findings support the applicability of finerenone in major glomerular disease subtypes within non-diabetic chronic kidney disease.

3.7 Multivariate Predictors of Renal and Safety Outcomes

Multivariate linear regression was conducted to identify independent predictors of changes in albuminuria (Δ UACR), kidney function (Δ eGFR), and serum potassium (Δ K⁺) after treatment. Variables included into the models included sex, age, baseline UACR, baseline eGFR, baseline potassium, and the use of RASi, SGLT-2i, and immunosuppressants, as well as renal pathological diagnoses.

As shown in Fig. 6, membranous nephropathy was significantly associated with a greater reduction in albuminuria ($\beta = -947.6$, 95% CI: -1694.3 to -200.8 , $P = 0.019$), indicating a more favorable antiproteinuric response. Baseline potassium level was the only significant predictor of Δ K⁺ ($\beta = 0.41$, 95% CI: 0.11 – 1.13 , $P = 0.028$), suggesting a modest impact of pretreatment potassium on treatment-related hyperkalemia. No significant predictors were identified for Δ eGFR. These findings highlight potential subgroup-specific efficacy and safety profiles of finerenone.

4. Discussion

This retrospective study adds valuable real-world data to the application of finerenone in patients with non-diabetic CKD. The present study shows that in a cohort encompassing multiple glomerular

pathologies, finerenone was associated with a significant reduction in albuminuria, stable kidney function, and minimal changes in serum potassium over the 12-month follow up period. These findings suggest that finerenone is effective in several glomerular diseases aside from diabetic kidney disease.

Compared with earlier single-disease studies, this study incorporated a wider range of pathological subtypes and introduced a dual-comparator framework [16]. In patients with MN, response rates remained consistently high at all time points. Multivariate models identified MN as an independent predictor of albuminuria reduction, pointing to a possible disease-specific sensitivity to MRAs. This aligns with pathophysiological insights, as subepithelial immune complex deposition in MN may augment MR activation and podocyte injury, rendering such patients more responsive to MR blockade [17,18].

Our present findings are consistent with previous studies focusing on specific glomerular diseases in terms of reproducible reductions in proteinuria, maintenance of eGFR, and a low incidence of clinically significant hyperkalemia. Additionally, the present study also offers insights to the clinical therapeutic effects of finerenone in a cohort with 12 months' follow-up data. In our previous study, we found that finerenone exerts significant antiproteinuric effects on MN in a small sample of 20, with 69% (9/13) patients showed UACR reduction. A similarly pronounced antiproteinuric effect of finerenone was observed in another small IgAN cohort (N = 49), in which the median UACR declined by 41.86% at 6 months. In this present study, 51 MN patients demonstrated the highest response rates, which persisted throughout the follow-up period, and MN emerged as an independent predictor of Δ UACR in multivariate regression analysis. Seventy-one IgAN patients also demonstrated significant reductions in UACR over time in this present study. These results strengthen the hypothesis that finerenone may have superior efficacy in proteinuric CKD characterized by subepithelial immune complex deposition, possibly due to enhanced podocyte injury and mineralocorticoid receptor activation [17,18]. These findings are further supported by the study's larger cohort (n = 151), longer follow-up period (12 months), inclusion of both non-diabetic CKD and DKD control groups, and the use of refined statistical modeling, including time-group interaction analyses, which together enhance the interpretive strength of the results.

Furthermore, patients with IgAN exhibited greater variability in albuminuria response [19]. This heterogeneity likely reflects differences in disease activity, chronicity, and the use of immunosuppressive therapy at baseline. Although response rates were less uniform, reductions in proteinuria were still observed without deterioration in eGFR, supporting the renal safety of finerenone across multiple glomerular disorders.

A notable strength of this study lies in the inclusion of two relevant comparator groups: DKD patients receiving finerenone treatment, and non-diabetic CKD patients treated with RASi combined with SGLT-2i. While both RASi and SGLT-2i are widely accepted antiproteinuric drugs regardless of patients' disease status, finerenone appeared to elicit a more rapid and potentially greater reduction in albuminuria in selected non-diabetic CKD patients [20,21]. This may offer an alternative for those who cannot tolerate the hemodynamic or glycemic effects of current first-line drugs.

Mechanistically, finerenone exerts antifibrotic, anti-inflammatory, and podocyte-protective effects, distinguishing it from other agents whose antiproteinuric benefits are partly mediated by hemodynamic mechanisms [22,23]. RASi and SGLT-2i typically induce transient eGFR declines due to efferent arteriolar dilation or tubuloglomerular feedback, respectively [24–26]. While similar early changes have been reported with finerenone in prior trials, we did not observe this effect in our cohort [27–29]. This discrepancy may be explained by the timing of eGFR assessment, which began at month three, potentially missing short-term fluctuations. Moreover, the lower rate of concurrent RASi or SGLT-2i use in our non-diabetic subgroup may have attenuated compound hemodynamic effects. These findings suggest that finerenone may have a more hemodynamically stable effect on the kidney, especially relevant for patients with limited renal reserve or borderline blood pressure.

In this study, SBP declined over time across all treatment groups, with the most pronounced reduction observed in DKDF patients, followed by those receiving RASi and SGLT-2i. A marginal and non-significant decline was noted in the nonDKDF group, suggesting variable antihypertensive responsiveness to finerenone depending on CKD etiology. While baseline SBP was similar between nonDKDF and nonDKDAS groups, it was modestly higher in DKDF patients. Among pathological subtypes, SBP decreased significantly in the IgAN subgroup but not in MN, reflecting potential differences in vascular reactivity or RAAS involvement. Younger age and greater hemodynamic sensitivity in IgAN may partly explain this disparity. Importantly, no hypotensive events occurred in either subgroup, underscoring the favorable safety profile of finerenone.

Overall, these findings indicate that finerenone exerts a modest and well-tolerated BP-lowering effect in non-diabetic CKD, with the magnitude of response likely influenced by patient phenotype, including age, proteinuria, and underlying pathology.

Despite the promising results, several limitations still warrant attention. The single-center, retrospective design may introduce the potential for selection bias and unmeasured confounding, although efforts have been made to adjust this discrepancy using multivariable models. The sample size for some pathological subtypes was modest, limiting subgroup comparisons. Follow-up data were occasionally incomplete, though handled with imputation and mixed-effects modeling.

In summary, this study provides preliminary but clinically valuable evidence supporting the use of finerenone in non-diabetic CKD, particularly among patients with membranous or IgA nephropathy. Further prospective trials are warranted to confirm these findings, explore long-term renal outcomes, and refine patient selection strategies across glomerular disease phenotypes.

Conclusion

In this real-world retrospective study, finerenone demonstrated clinically meaningful antiproteinuric effects and preserved kidney function in patients with non-diabetic chronic kidney disease, with a favorable safety profile regarding serum potassium. Notably, membranous nephropathy patients exhibited the most pronounced response, suggesting potential disease-specific benefits. Compared to

both diabetic patients and non-diabetic patients treated with RASi plus SGLT-2 inhibitors, finerenone appeared more effective in reducing albuminuria without compromising renal safety. These findings support the potential role of finerenone as an adjunctive therapy in non-diabetic CKD, particularly for glomerular diseases with persistent proteinuria. Prospective trials are needed to confirm these observations and guide patient selection strategies for mineralocorticoid receptor antagonists in this population.

Declarations

Conflicts of Interest

The authors declare no competing interests.

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Author Contribution

Research idea and study design: S.X. Liu, X.Y. Bai; data acquisition: J.Q. Li, D.F. Wu, M.H. Huang; data analysis/interpretation: J.Q. Li, D.F. Wu, R.B. Liu; statistical analysis: J.Q. Li, N. Fan, H. Jiang, P.M. Liu; supervision or mentorship: H.S. Xu. S.X. Liu, X.Y. Bai take responsibility that this study has been reported honestly, accurately and transparently, and accepts accountability for the overall work by ensuring that questions pertaining to the accuracy or integrity of any portion of the work are appropriately investigated and resolved.

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Data Availability Statement

The data that support the findings of this study are available in the supplementary material of this article.

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Figures

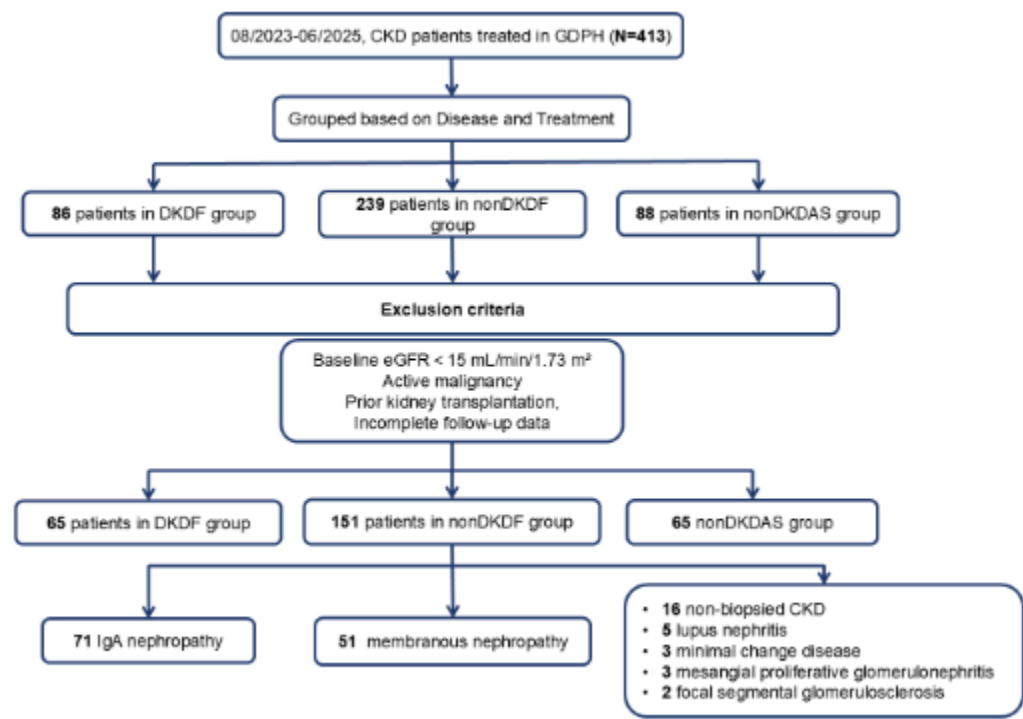


Figure 1

Flowchart of patient selection and cohort classification.

GDPH: Guangdong Provincial People's Hospital

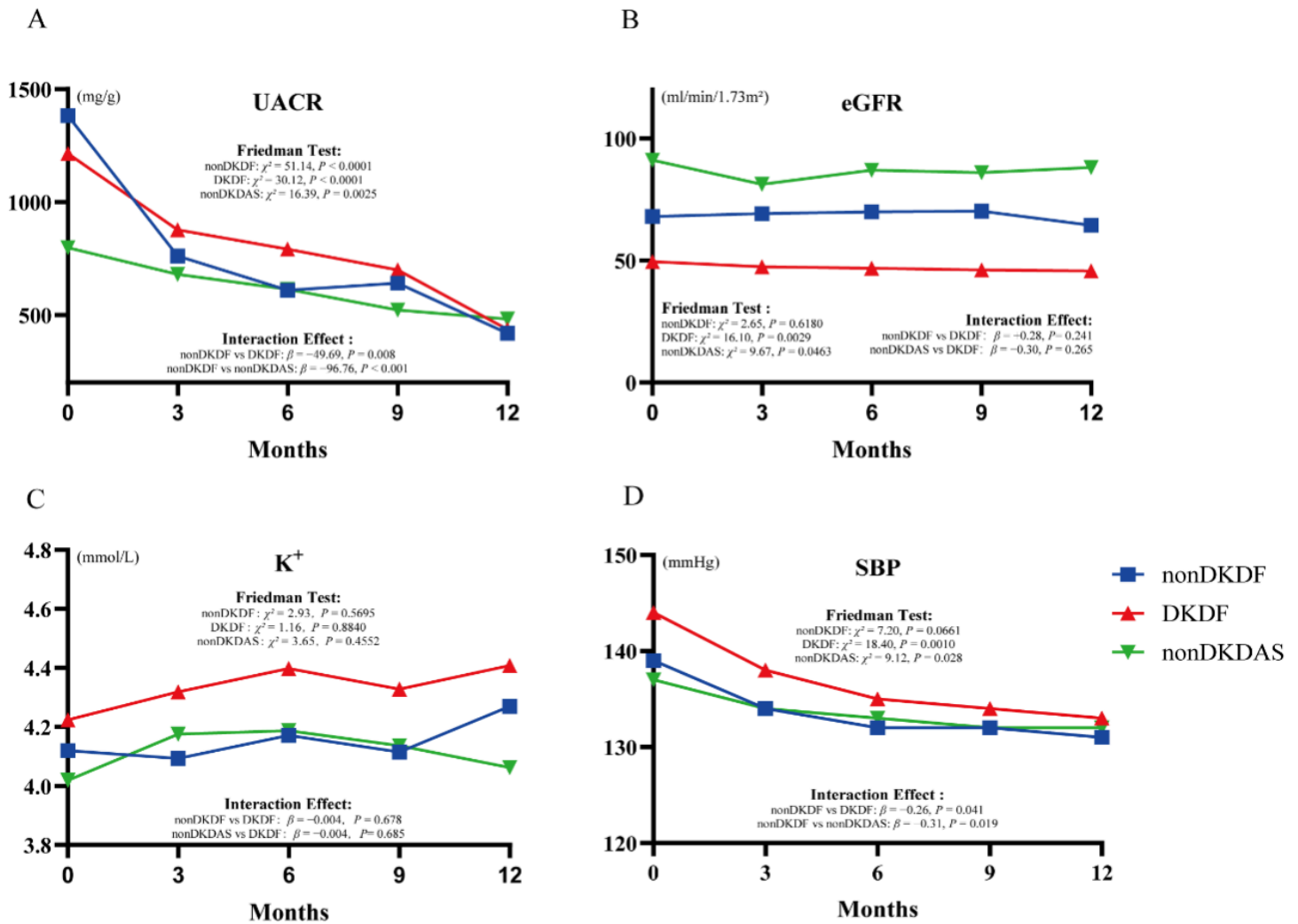


Figure 2

Longitudinal changes in UACR, eGFR, serum potassium and SBP across DKDF, nonDKDF, and nonDKDAS groups during 12 months of follow-up.

Each curve represents mean $\pm$ SEM values at baseline, 3, 6, 9, and 12 months. Friedman's test was applied to assess intra-group temporal trends, and group $\times$ time interactions were tested using linear mixed-effects models. **A**, All groups demonstrated significant albuminuria reduction over time ($P < 0.01$), with nonDKDF showing the greatest reduction compared to DKDF ($\beta = -49.69$, $P = 0.008$) and nonDKDAS ($\beta = -96.76$, $P < 0.001$). **B**, eGFR declined significantly in DKDF and nonDKDAS groups ($\chi^2 = 30.12$ and 16.39 , respectively). No significant eGFR change was observed in nonDKDF ($P = 0.539$). **C**, No significant changes in potassium levels were observed in any group ($P > 0.45$). **D**, SBP declined significantly in DKDF ($\chi^2 = 18.40$, $P = 0.001$) and nonDKDAS ($\chi^2 = 9.12$, $P = 0.028$), with a non-significant trend observed in nonDKDF ($P = 0.066$).

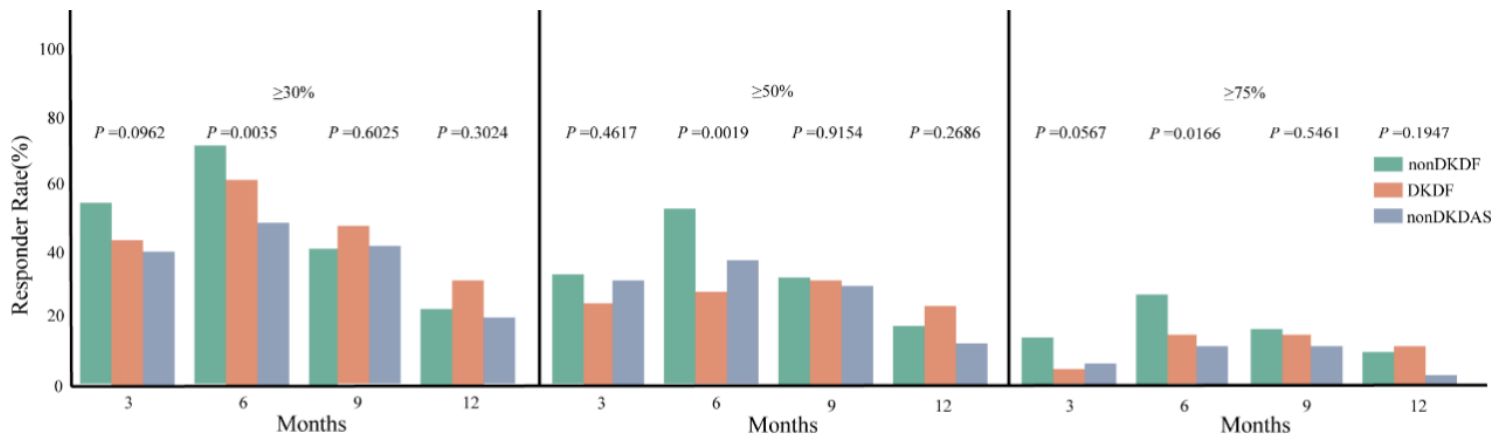


Figure 3

Temporal trends in proportion of patients achieving UACR reduction thresholds.

Proportions of patients achieving UACR reduction from baseline across three treatment groups at 3, 6, 9, and 12 months. Response thresholds were defined as $\geq 30\%$, $\geq 50\%$, and $\geq 75\%$, as shown in three separate panels. Group differences in response rates were statistically significant only at month 6 across all thresholds ($\geq 30\%$ response at month 6: $P = 0.0035$, $\geq 50\%$ response at month 6: $P = 0.0019$, $\geq 75\%$ response at month 6: $P = 0.0166$). No statistically significant differences were found at months 3, 9, or 12 ($P > 0.05$).

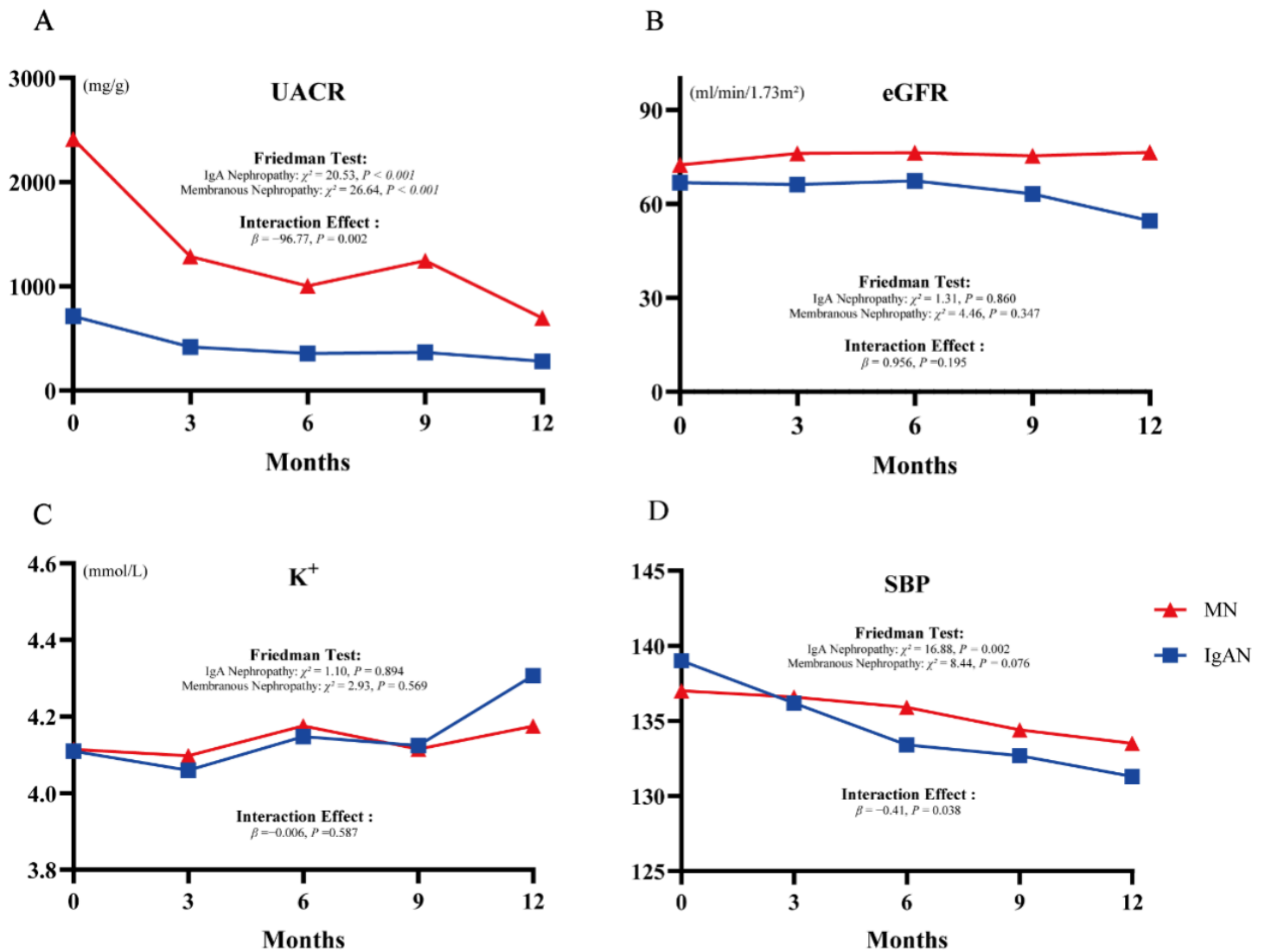


Figure 4

Longitudinal trends of renal biomarkers in IgA nephropathy and membranous nephropathy patients treated with finerenone.

This composite figure shows the mean values of UACR, eGFR and serum potassium in IgA nephropathy and membranous nephropathy patients at baseline and at 3, 6, 9, and 12 months of finerenone treatment. Error bars represent standard error (SE). **A**, UACR significantly decreased over time in both groups (IgAN: $\chi^2 = 20.53, P < 0.001$; MN: $\chi^2 = 26.64, P < 0.001$), with a significant group $\times$ time interaction ($\beta = -96.77, P = 0.002$). **B**, eGFR remained stable in both groups (IgAN: $\chi^2 = 1.31, P = 0.860$; MN: $\chi^2 = 4.46, P = 0.347$), with no significant interaction ($\beta = 0.956, P = 0.195$). **C**, Serum potassium levels did not change significantly (IgAN: $\chi^2 = 1.10, p = 0.894$; MN: $\chi^2 = 2.93, P = 0.569$), and the interaction term was non-significant ($\beta = -0.006, P = 0.587$). **D**, SBP declined significantly over time in IgAN patients ($\chi^2 = 16.88, P = 0.002$), but not in those with MN ($\chi^2 = 8.44, P = 0.076$), and a significant interaction was observed ($\beta = -0.41, P = 0.038$).

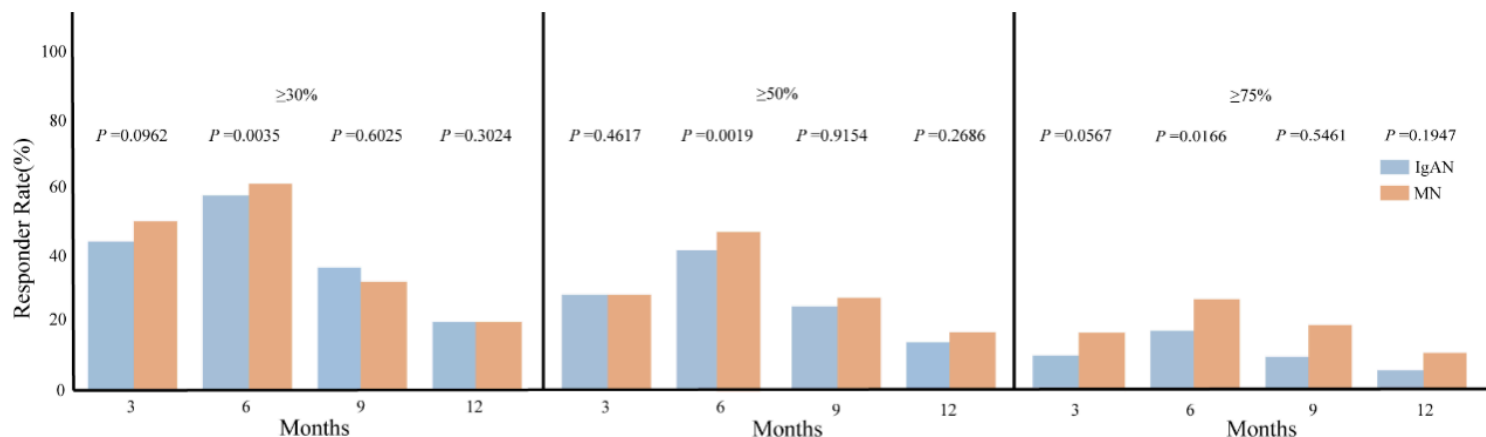


Figure 5

UACR response rates ($\geq 30\%$, $\geq 50\%$, $\geq 75\%$) over time in patients with IgA nephropathy and membranous nephropathy.

This figure shows the proportion of responders (patients with a $\geq 30\%$, $\geq 50\%$, or $\geq 75\%$ reduction in UACR from baseline) at 3, 6, 9, and 12 months among nonDKDF patients with IgA nephropathy and membranous nephropathy. P-values indicate group differences at each time point (Chi-square test), with no statistically significant differences observed (all $P > 0.05$), suggesting a comparable albuminuria-lowering response to finerenone in both pathological subtypes.

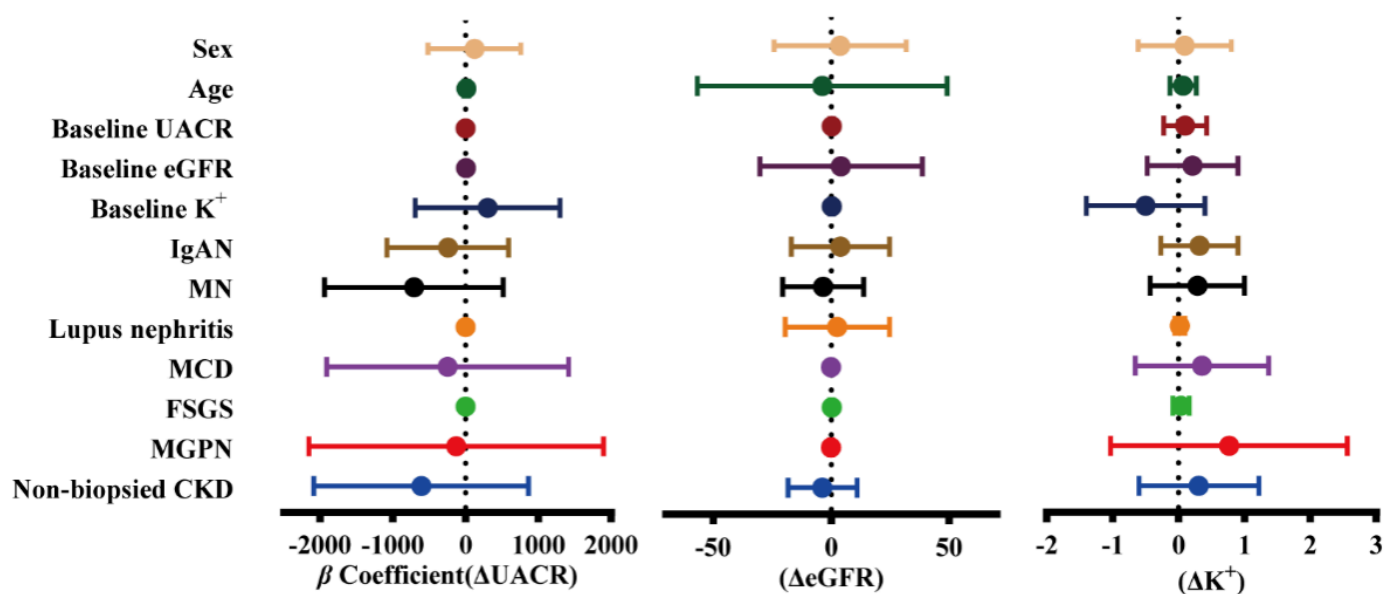


Figure 6

Multivariate predictors of changes in albuminuria, kidney function, and serum potassium following treatment.

Forest plots display the β coefficients and 95% confidence intervals from multivariate linear regression models for changes in UACR (Δ UACR, left panel), eGFR (Δ eGFR, middle panel), and serum potassium (Δ K⁺, right panel) after treatment. Variables included in the model were sex, age, baseline UACR, baseline eGFR, baseline potassium, use of RAS inhibitors, SGLT-2 inhibitors, immunosuppressants, and underlying pathological diagnoses (including IgA nephropathy, membranous nephropathy, lupus nephritis, minimal change disease, FSGS, MPGN, and non-biopsied CKD). In the multivariate linear regression analysis, membranous nephropathy was the only significant predictor of Δ UACR ($\beta = -947.6$, 95% CI: -1694.3 to -200.8 , $P = 0.019$), while baseline potassium level was significantly associated with Δ K⁺ ($\beta = 0.41$, 95% CI: $0.11-1.13$, $P = 0.028$). No significant predictors were found for Δ eGFR.