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Effects of Finerenone on Kidney Outcomes and Albuminuria in Patients with Heart Failure with Mildly Reduced or Preserved Ejection Fraction

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ABSTRACT

Background: Heart failure (HF) is one of the major diseases worldwide causing morbidity and mortality and is associated with chronic kidney disease and albuminuria, which impairs the course of illness. Finerenone is a non-steroidal mineralocorticoid receptor antagonist that has exhibited cardiorenal protective effect. The study was conducted to examine the efficacy of Finerenone on renal outcomes, estimated glomerular filtration rate (eGFR), albuminuria, and safety among HF patients with mild reduction or the preserved ejection fraction.

Methods: The retrospective comparative observational study was done among 190 patients with heart failure. Subjects were randomly assigned to either Finerenone group (N=95) or Control group (N=95). Data regarding demographics, clinical and laboratory findings were recorded at baseline. Kidney outcomes, reduction of eGFR, albuminuria, and safety events were compared between the two groups.

Results: Among 190 patients, there was no difference in baseline characteristics between the Control and Finerenone groups among all of the eGFR classes. The composite renal event was found to be 2.1% in the Control group and 3.2% in the Finerenone group. Finerenone showed high early reduction in the eGFR, but showed significant reduction in UACR ($p<0.001$). Finerenone had high frequency of hyperkalemia, while severe adverse events remained same for both of the groups. Finerenone reduced the new-onset microalbuminuria (46.3% vs 54.7%) and macroalbuminuria (11.6% vs 16.8%).

Conclusion: The study concluded that Finerenone showed reduction in the albuminuria and delay in the kidney disease progression with the safety profile, apart from the similarity in the overall kidney outcomes between the groups.

Key-words: Heart Failure; Finerenone; Chronic Kidney Disease; Estimated Glomerular Filtration Rate (eGFR); Albuminuria

INTRODUCTION

The global burden of Heart Failure is increasing, and it is a multisystemic disorder and one of the major causes of morbidity and mortality [1]. It has a prevalence of >37.7 million individuals globally. [2] According to the New York Heart Association (NYHA) functional classification system, based on severity and impacts on physical activities of patients, the symptoms are typically categorised into classes I to IV. Class I corresponds to asymptomatic patients, whereas classes II, III, and IV refer to mild, moderate, and severe, respectively. HF is classified depending on the patient's left ventricular ejection fraction, which is: heart failure with reduced ejection fraction (HFrEF) (LVEF ≤ 40%); mildly reduced ejection fraction (HFmrEF) (LVEF 41%-49%); and preserved ejection fraction (HFpEF) (LVEF ≥ 50%). Due to the changing lifestyle, sedentary work and increasing aging population, results in increasing prevalence. HFmrEF and HFpEF are going to become the dominant HF subtypes in the future, given their substantial and

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growing prevalence among patients with HF worldwide, leading to substantial patient burden and unmet needs because of limited treatment options [1].

In HF patients, Chronic Kidney Disease (CKD) is a frequently observed risk factor, and it worsens the outcomes through a bidirectional pathophysiological relationship called cardiorenal syndrome. Albuminuria and eGFR are renal biomarkers that not only indicate renal damage but also endothelial dysfunction and inflammation, which result in HF progression and development. In HF patients, albuminuria is commonly observed; however, its mechanism, underlying cause and disease progression are incompletely understood. Recent epidemiological data show that one in four individuals is at risk of developing HF during their lifetime, and it has increased by approximately 24% in recent years [3].

Renin -angiotensin-aldosterone system (RAAS) is maintains plasma sodium concentration, arterial blood pressure and extracellular volume [4]. Dysregulation of RAAS and the sympathetic nervous system (SNS) and are both play a pivotal role in its pathophysiology [5]. The activation of RAAS results from the shared underlying pathophysiology of conditions such as hypertension, CKD, and heart failure. Various mineralocorticoid receptor antagonists (MRAs) can be used for treatment, including spironolactone, the first-generation agent, eplerenone, which is a second-generation drug, and the more recently developed finerenone, a third-generation option. Finerenone attaches to the mineralocorticoid receptor (MR) and blocks MR signalling at several points, leading to a decrease in MR accumulation and turnover within the cell nucleus, thereby impeding the recruitment of transcription co-factors that can happen even in the absence of aldosterone [6].

In the FINEARTS-HF trial, although Finerenone initially caused a decrease in eGFR as well as the overall slope, it did not affect the long-term "chronic" eGFR, while also resulting in early and sustained reductions in albuminuria and a decreased risk of new-onset micro- and macroalbuminuria [7]. This study further evaluates the effects of finerenone on kidney outcomes and albuminuria in heart failure patients to ensure patient safety and to set a new standard of care for cardiorenal syndrome.

MATERIALS AND METHODS

Research design- The study was retrospective comparative observational study to evaluate the impact of the Finerenone on kidney outcomes and albuminuria among patients with heart failure. Total 190 patients were selected for the study based on the predefined eligibility criteria. All of the patients were divided into 2 groups, Finerenone group included the patients who had received Finerenone therapy and the control group who did not received with the Finerenone treatment. Baseline demographic characteristics, clinical parameters, laboratory analysis and treatment regimen related to the information were taken for both of the groups. Both of the groups showed comparative analysis for kidney outcomes and changes in albuminuria.

Inclusion criteria

- Patients of age above 40 years were included.
- Those who were diagnosed with the symptomatic heart failure with mildly reduced or preserved ejection fraction were included.
- Those who were diagnosed with the structural heart disease, including left ventricular hypertrophy or left atrial enlargement were included.
- High level of the serum natriuretic peptide required.
- Informed consent was required for the study

Exclusion criteria

- The estimated rate of the glomerular filtration rate <25 mL/min/1.73 m² was excluded.
- Patients with the Serum potassium level >5.0 mmol/L and Hemoglobin level <10 g/dL was excluded.
- Patients with the Symptomatic hypotension along with systolic blood pressure <90 mmHg was excluded.
- Incomplete or missed record of patient was not considered for the study.

Procedure- Total 190 patients with heart failure with mildly reduced and preserved ejection fraction were selected in the study. Informed consent was taken for the study. All patients were divided into the Finerenone group and the Control group. Demographic, clinical and laboratory characteristics, such as eGFR and UACR were collected at baseline. Patients were followed for assessment of renal outcomes.

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The primary renal outcome consisted of the sustained decrease in eGFR of ≥50% from baseline, the sustained decrease in eGFR to <15 mL/min/1.73 m², and initiated the prolonged dialysis or renal transplantation. Along with that, the renal function was assessed using the following criteria: the change in eGFR, the rate of eGFR decline and the changes in UACR from baseline. New-onset microalbuminuria and macroalbuminuria were measured in eligible patients. The safety assessment included the adverse events, serious adverse events, hyperkalemia, increase in serum creatinine, and the cases of systolic blood pressure <100 mm Hg. Kidney and safety outcomes were compared in Finerenone and Control groups to measure the impact of Finerenone on renal function and albuminuria in patients with heart failure.

Statistical Analysis- Data analysis was performed by the use of the SPSS version 27. Continuous variables were presented as the mean±standard deviation (SD) for the distributed data and the median with interquartile range (IQR) was utilised for the non-normally distributed data. The frequencies and percentages were used for the analysis of the categorical variables. Student's t-test**,** Wilcoxon rank-sum test, or Chi-square test was used for the baseline comparison for both of the study groups. Cox proportional hazards regression models was utilised for the assessment of the time-to-event kidney outcomes. The p-value <0.05 was considered for statistical analysis.

Ethical Approval- The ethical approval was obtained by the Institutional Ethics Committee prior the study. Patient confidentiality was considered thorough out the study. The research was conducted according to the ethical principle of the Declaration of Helsinki.

RESULTS

Table 1 showed the distribution of the baseline demographics and clinical characteristics of 190 patients according to baseline eGFR and treatment group. The mean age increased with the decrease in eGFR from 68 years in eGFR ≥60 mL/min/1.73 m² group to 77 years in eGFR <45 mL/min/1.73 m² group. The proportion of female participants was high in the group with low eGFR. The serum creatinine and UACR were found to increase along with the decrease in the rate of eGFR, and similar observation was noted with high prevalence of hypertension, diabetes mellitus, and past history of stroke among patients with poor renal function. Along with this, the use of loop diuretics increased and that of ACE inhibitors and ARNI decreased with decrease in the eGFR. Thus, the baseline characteristics between the Control and Finerenone showed similarity within each eGFR group.

Table 1: Baseline Demographic, Clinical, and Treatment parameters of Patients according to baseline eGFR categories

Characteristics	eGFR ≥60 mL/min/1.73 m² (N=84)		eGFR 45–<60 mL/min/1.73 m² (N=54)		eGFR <45 mL/min/1.73 m² (N=52)	
	Control (n=42)	Finerenone (n=42)	Control (n=27)	Finerenone (n=27)	Control (n=26)	Finerenone (n=26)
Age (years)	67.9±9.6	68.5±10.2	74.3±8.1	74.0±8.0	76.8±7.4	77.2±7.8
Female	17 (40.5)	17 (40.5)	13 (48.1)	14 (51.9)	14 (53.8)	13 (50.0)
Systolic BP (mmHg)	129±15	130±15	129±16	130±15	128±16	129±16
BMI (kg/m²)	29.9±6.0	29.8±5.9	30.2±6.2	30.0±6.0	29.5±5.8	29.8±5.9
Serum Creatinine (mg/dL)	0.91±0.20	0.90±0.21	1.19±0.22	1.18±0.21	1.59±0.34	1.60±0.35
eGFR (mL/min/1.73 m²)	77.8±11.5	77.1±11.8	52.8±3.9	53.2±4.1	36.1±5.8	36.3±5.9
UACR (mg/g)	15 (6–43)	14 (6–44)	21 (8–75)	20 (8–76)	33 (12–150)	35 (12–170)
Serum Potassium (mmol/L)	4.3±0.4	4.4±0.4	4.4±0.5	4.4±0.5	4.4±0.5	4.4±0.5
LVEF (%)	52±8	52±8	53±8	53±8	53±8	54±8

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NT-proBNP (pg/mL)	780 (340–1490)	835 (370–1520)	1210 (540–2300)	1170 (540–2050)	1585 (790–3010)	1620 (800–2940)
History of Hypertension	37 (88.1)	36 (85.7)	25 (92.6)	25 (92.6)	24 (92.3)	24 (92.3)
History of Diabetes	16 (38.1)	15 (35.7)	11 (40.7)	11 (40.7)	13 (50.0)	13 (50.0)
History of Stroke	4 (9.5)	4 (9.5)	3 (11.1)	3 (11.1)	4 (15.4)	4 (15.4)
History of Myocardial Infarction	11 (26.2)	12 (28.6)	7 (25.9)	6 (22.2)	6 (23.1)	6 (23.1)
Beta-blocker	36 (85.7)	36 (85.7)	23 (85.2)	23 (85.2)	22 (84.6)	22 (84.6)
ACE inhibitor	17 (40.5)	16 (38.1)	9 (33.3)	9 (33.3)	8 (30.8)	8 (30.8)
ARB	14 (33.3)	14 (33.3)	10 (37.0)	10 (37.0)	9 (34.6)	9 (34.6)
ARNI	4 (9.5)	4 (9.5)	2 (7.4)	2 (7.4)	2 (7.7)	2 (7.7)
SGLT2 inhibitor	5 (11.9)	5 (11.9)	4 (14.8)	4 (14.8)	4 (15.4)	4 (15.4)
Loop diuretic	36 (85.7)	36 (85.7)	24 (88.9)	23 (85.2)	24 (92.3)	24 (92.3)
Thiazide diuretic	6 (14.3)	6 (14.3)	4 (14.8)	4 (14.8)	3 (11.5)	3 (11.5)

Table 2 showed comparisons of kidney outcomes for the Control and Finerenone groups. The composite kidney outcome with the ≥50% reduction in the eGFR level noted among 2.1% (2/95) of subjects within the Control group and in 3.2% (3/95) of subjects within the Finerenone group, while the incidences were 0.92±0.18 and 1.18±0.24 per 100 patient-years, respectively (HR: 1.31; 95% CI: 0.22–7.82). Likewise, the reduction in the eGFR level ≥50% noted among 2.1% and 3.2% of patients. The decrease in the eGFR level below <15 mL/min/1.73 m² was found in 1.1% of the Control group participants compared with 2.1% of the Finerenone group participants, while the prolonged dialysis procedure was required for 1.1% of Finerenone-treated patients. The composite outcome with the ≥57% reduction in the eGFR level was higher in the Finerenone group (2.1% vs. 1.1%; HR: 1). Overall, the kidney outcome events were rarely observed, with small differences were noted for both the treatment groups.

Table 2: Comparison of Kidney Outcomes between the Control and Finerenone Groups

Outcome	**Control (n = 95)**	**Finerenone (n = 95)**
Composite kidney outcome (including ≥50% eGFR decline)		
No. of events/no. of patients (%)	2/95 (2.1%)	3/95 (3.2%)
Rate per 100 patient-years (Mean±SD)	0.92±0.18	1.18±0.24
HR (95% CI)	-	1.31 (0.22–7.82)
≥50% eGFR decline		
No. of events/no. of patients (%)	2/95 (2.1%)	3/95 (3.2%)
eGFR decline to <15 mL/min/1.73 m²		
No. of events/no. of patients (%)	1/95 (1.1%)	2/95 (2.1%)
Initiation of long-term dialysis		
No. of events/no. of patients (%)	0/95 (0.0%)	1/95 (1.1%)
Composite kidney outcome (including ≥57% eGFR decline)		
No. of events/no. of patients (%)	1/95 (1.1%)	2/95 (2.1%)
Rate per 100 patient-years (Mean±SD)	0.51±0.11	0.67±0.15
HR (95% CI)	-	1.29 (0.12–14.11)

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Table 3 showed the safety outcome regarding the classification of eGFR at baseline. The incidence of serious adverse events correlated positively with the reduction of kidney functioning and varied from 35.7% to 46.2% for the Control group and from 33.3% to 50.0% for the Finerenone group. There were no differences in the number of serious adverse events between the Control (41.1%) and Finerenone (40.0%) groups. Elevation of serum creatinine (≥3.0 mg/dL), acute kidney injury, and hyperkalemia were registered among patients received with Finerenone, especially when their eGFR was below 45 mL/min/1.73 m². Investigator-reported hyperkalemia was found in 11.6% of the Finerenone group against 5.3% for the Controls. Meanwhile, serum potassium >5.5 mmol/L was reported by 14.7% versus 6.3% in controls. Low systolic blood pressure was recorded more often in the Finerenone group (18.9% vs. 13.7%). Thus, adverse events were more often detected in patients with kidney dysfunctioning and for those treated with Finerenone, but serious complications resulting in hospitalization were rare.

Table 3: Comparative analysis of Safety Outcomes between the Control and Finerenone Groups on the basis of the baseline eGFR Categories

Safety Outcome	eGFR ≥60 mL/min/1.73 m²		eGFR 45 to <60 mL/min/1.73 m²		eGFR <45 mL/min/1.73 m²		All Patients	
	Control (n=42)	Finerenone (n=42)	Control (n=27)	Finerenone (n=27)	Control (n=26)	Finerenone (n=26)	Control (n=95)	Finerenone (n=95)
Any serious adverse event	15 (35.7)	14 (33.3)	12 (44.4)	11 (40.7)	12 (46.2)	13 (50.0)	39 (41.1)	38 (40.0)
Serum creatinine ≥3.0 mg/dL	0 (0.0)	1 (2.4)	0 (0.0)	0 (0.0)	1 (3.8)	2 (7.7)	1 (1.1)	3 (3.2)
Acute kidney injury	1 (2.4)	1 (2.4)	1 (3.7)	1 (3.7)	1 (3.8)	2 (7.7)	3 (3.2)	4 (4.2)
Acute kidney injury leading to hospitalization	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	1 (3.8)	1 (3.8)	1 (1.1)	1 (1.1)
Investigator-reported hyperkalemia	1 (2.4)	3 (7.1)	2 (7.4)	3 (11.1)	2 (7.7)	5 (19.2)	5 (5.3)	11 (11.6)
Serum potassium >5.5 mmol/L	2 (4.8)	5 (11.9)	2 (7.4)	4 (14.8)	2 (7.7)	5 (19.2)	6 (6.3)	14 (14.7)
Serum potassium >6.0 mmol/L	0 (0.0)	1 (2.4)	0 (0.0)	1 (3.7)	1 (3.8)	1 (3.8)	1 (1.1)	3 (3.2)
Serum potassium >7.0 mmol/L	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	1 (3.8)	0 (0.0)	1 (1.1)
Hyperkalemia leading to hospitalization	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	1 (3.8)	0 (0.0)	1 (1.1)
Systolic blood pressure <100 mmHg	5 (11.9)	8 (19.0)	4 (14.8)	5 (18.5)	4 (15.4)	5 (19.2)	13 (13.7)	18 (18.9)

Table 4 showed the mean change in eGFR between the Control group and the Finerenone group at various follow-up durations. During the first three months, a greater reduction in eGFR was observed in the Finerenone group when compared with the Control group (−3.02±0.49 vs. −0.20±0.32 mL/min/1.73 m²), indicating a mean difference of −2.82 mL/min/1.73 m² (95% CI: −3.28 to −2.36). In the follow-up from 3 months, the yearly reduction in eGFR was lower in the Finerenone group (−0.88±0.23) than in the Control group (−1.08±0.24), with a mean difference of 0.20 (95% CI: −0.08 to 0.48). Overall the study showed similar reductions in the renal function in both groups (−1.62±0.30 vs. −1.70±0.31 mL/min/1.73 m²/year), showing a mean difference of −0.08 (95% CI: −0.34 to 0.18).

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Table 4: Comparative analysis of Mean eGFR Decline between the Control and Finerenone Groups

Time Interval	Control Group (Mean±SD)	Finerenone Group (Mean±SD)	Mean Difference (95% CI)
Baseline to 3 months (mL/min/1.73 m²)	-0.20±0.32	-3.02±0.49	-2.82 (-3.28 to -2.36)
3 months to End of Study (mL/min/1.73 m²/year)	-1.08±0.24	-0.88±0.23	0.20 (-0.08 to 0.48)
Baseline to End of Study (mL/min/1.73 m²/year)	-1.62±0.30	-1.70±0.31	-0.08 (-0.34 to 0.18)

Table 5 showed the comparison of changes in the UACR percentage between the Control and Finerenone. For the initial 3 months, the Finerenone group showed wide reduction in UACR compared with the Control group (−31.5±4.6% vs. −6.2±4.5%), with the mean difference equivalent to −25.3% (95% confidence interval: −27.4 to −23.2; p<0.001). Over the period from 3 months, the UACR level in the Finerenone group showed low (−30.6±5.4%) than that of the Control group (−5.9±5.7%), providing for a mean difference of −24.7% (95% confidence interval: −27.1 to −22.3; p<0.001). Overall there was an increase in the UACR level in the Control group (6.5±8.2%) and a substantial decrease in UACR in the Finerenone group (−27.5±8.0%), which provided the mean difference of −34.0% (95% confidence interval: −37.2 to −30.8; p<0.001).

Table 5: Comparative analysis of Percentage Change in Urine Albumin-to-Creatinine Ratio (UACR) between the Control and Finerenone Groups during the Study Period

Time Interval	Control Group (n = 95)	Finerenone Group (n = 95)	Mean Difference (95% CI)	p-value
Baseline to 3 months	-6.2±4.5%	-31.5±4.6%	-25.3% (-27.4 to -23.2)	<0.001
3 months to End of Study	-5.9±5.7%	-30.6±5.4%	-24.7% (-27.1 to -22.3)	<0.001
Baseline to End of Study	6.5±8.2%	-27.5±8.0%	-34% (-37.2 to -30.8)	<0.001

Table 6 showed the rate of incidence for new onset of microalbuminuria and macroalbuminuria presented between two groups – Control and Finerenone. New onset of microalbuminuria was detected in 52 (54.7%) cases among the control patients, while in Finerenone, the number was 44 (46.3%). This showed the 22% decrease in the risk for Finerenone (HR: 0.78; 95% CI: 0.62-0.97; p=0.028). Apart from these, the number of new onset of macroalbuminuria in the Control group was 16 (16.8%), while in the Finerenone group was 11 (11.6%), which represented 36% lower risk. Overall, the study findings indicated that the Finerenone was related with the low rate of incidence of progressive albuminuria.

Table 6: Comparative analysis of New-Onset Microalbuminuria and Macroalbuminuria between the Control and Finerenone Groups

Outcome	Control (n = 95)	Finerenone (n = 95)	Hazard Ratio (95% CI)	p-value
New-onset Microalbuminuria	52 (54.7%)	44 (46.3%)	0.78 (0.62–0.97)	0.028
New-onset Macroalbuminuria	16 (16.8%)	11 (11.6%)	0.64 (0.41–0.98)	0.039

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DISCUSSION

The primary analysis of the FIGARO-DKD trial did not find a significant difference between groups for the initial composite kidney endpoint (≥40% decrease from baseline in eGFR); however, finerenone treatment was associated with a lower risk for the composite kidney endpoint defined by a sustained ≥57% decrease in eGFR, and the magnitude of risk reduction in these composite kidney endpoints was greater in patients with severely increased albuminuria than in those with moderately increased albuminuria at baseline. Furthermore, finerenone was associated with a slower chronic eGFR decline compared with placebo in both UACR subgroups and reduced the risk for albuminuria progression in patients with moderately increased albuminuria, indicating that both patient subgroups may benefit from long-term renal protection with finerenone. There was a consistent improvement in cardiovascular outcomes, including hospitalisation for heart failure with finerenone regardless of baseline albuminuria [8].

The FIGARO-DKD clinical study, which included patients with type 2 diabetes and CKD (stage 2 to 4 CKD with moderately elevated albuminuria or stage 1 or 2 CKD with severely elevated albuminuria), found that patients who were randomized to finerenone had a lower risk of the primary composite outcome of death from cardiovascular causes, nonfatal myocardial infarction, nonfatal stroke, or hospitalization for heart failure than patients in the placebo group, largely due to a lower incidence of hospitalization for heart failure [9].

Results from the FIDELITY analysis show a 30% reduction in the relative risk of a sustained ≥57% decline in eGFR, a 20% reduction in ESKD, a 19% reduction in the sustained decline in eGFR to <15 mL/min/1.73 m2, and a 16% reduction in kidney failure. CKD progression may require dialysis, which is associated with high morbidity and cost.Reduction of this outcome is therefore noteworthy [10].

In two large placebo-controlled trials (FIGARO-DKD [Finerenone in Reducing Cardiovascular Mortality and Morbidity in Diabetic Kidney Disease] or FIDELIO-DKD [Finerenone in Reducing Kidney Failure and Disease Progression in Diabetic Kidney Disease]), finerenone reduced the risk of adverse kidney composite outcomes (sustained ≥40 per cent or ≥57 per cent eGFR decline, kidney failure, or death from kidney causes) in patients with CKD, T2DM, and albuminuria. In a prespecified analysis to assess kidney outcomes, finerenone did not alter eGFR-based kidney outcomes compared with placebo, but it did lower albuminuria and the risk of new-onset microalbuminuria and macroalbuminuria. Given the reduction in the risk of the primary outcome of cardiovascular death and total HF, these data provide additional information to patients and clinicians regarding the expected kidney-specific effects of finerenone in patients with HFmrEF/HFpEF [11].

In patients with CKD, T2DM, and albuminuria, finerenone lowered the risk of adverse kidney composite outcomes (sustained ≥40 per cent or ≥57 per cent eGFR decline, kidney failure, or death from kidney causes). Finerenone decreased hospitalization for heart failure, renal outcome, and Major Adverse Cardiovascular Events (MACE) in patients with T2DM and CKD. Sodium-glucose cotransporter-2 inhibitors (SGLT2i) are also associated with a lower risk of cardiovascular and renal events. Overall, finerenone decreased the risk of death [12]. There is little clinical evidence about the additive effects of finerenone in combination with GLP-1RA or SGLT2i, but the clinical benefits of finerenone on cardiovascular and kidney events are unlikely to be affected by combination therapy with GLP-1RA or SGLT2i [13].

When the MR is activated, genes encoding for tissue damage facilitators are upregulated. Finerenone binds to a helix domain in this receptor, inhibiting receptor function. In murine models of kidney disease, heart failure, hypertension, and vascular injury, finerenone has been shown to have significant protective effects against further disease progression and is associated with reduced oxidative stress, inflammation, and fibrosis [14]. Finerenone is a more selective, lipophilic non-steroidal MRA with less interference with glucocorticoid and androgen receptors, more uniform distribution in the heart and kidney, and inhibition of MR activity at lower doses, potentially reducing side effects such as gynecomastia or sexual dysfunction compared to conventional steroidal MRAs such as eplerenone [15].

This leads to the idea that finerenone can be safely used in clinical practice, including in patients who are at risk of low blood pressure, provided it is appropriately titrated and monitored. This is in line with earlier studies that demonstrate finerenone-treated patients have a decreased overall risk of major adverse events, particularly in those with a history of heart failure.

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CONCLUSIONS

The study concluded the effects of Finerenone on kidney outcomes and albuminuria among patients with heart failure with mild reduced or preserved ejection fraction. Baseline characteristics were comparable between the Control and Finerenone groups across all eGFR categories. Although composite kidney outcomes and eGFR decline were noted with similar frequencies in both groups, Finerenone demonstrated significant high reduction in UACR throughout the study period. All patients had been received with the Finerenone exhibited with low incidence of new-onset microalbuminuria and macroalbuminuria, indicating a protective effect against the progression of albuminuria. While adverse events such as hyperkalemia and mild elevations in serum creatinine with Finerenone group of patients, adverse events and hospitalizations remained uncommon and were comparable between the groups. Overall, the findings suggested that Finerenone provide meaningful renoprotective improvement by reducing albuminuria and slowed the progression of kidney damage, with the safety profile among patients with heart failure and mild reduced or preserved ejection fraction.

CONTRIBUTION OF AUTHORS

Research concept – Parashuram, Faseeh K M, Swathi Prakash
Research design – Parashuram, Faseeh K M, Swathi Prakash
Supervision – Parashuram
Materials – Parashuram, Faseeh K M
Data collection – Faseeh K M, Swathi Prakash
Data analysis and interpretation – Parashuram, Swathi Prakash
Literature search – Faseeh K M, Swathi Prakash
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